THE PATHOLOGY OF
Food and Pasture Legumes

Edited by

D.J. ALLEN

Formerly of the Centro Internacional de Agricultura Tropical Regional Bean Program Tanzania

and

J.M. LENNÉ

International Crops Research Institute for the Semi-Arid Tropics India

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Preface

Sometime in 1988, Professor Gordon E. Russell took on the huge task of editing a replacement book for Butler and Jones' *Plant Pathology* which had been published by Macmillan in 1949. A large number of eminent plant pathologists were invited to contribute to the production of what Russell foresaw as 'one of the most important and prestigious books on plant pathology, which should become a standard work of reference for many years to come'. Sadly, this ambitious project, which was to have covered the world's major crop plants, never came to fruition: Russell died suddenly and Macmillan effectively went out of scientific publication. This left a number of authors in search of a book, reminiscent perhaps of Pirandello's *Six Characters in Search of an Author*.

The present volume retains the original structure for treatment of individual diseases of a given crop but its focus is much sharper; we address the legume crops alone. The plant family *Leguminosae* is second in economic importance only to *Gramineae*, which includes the world's cereals and pasture grasses; they clearly deserve similar treatment in a separate volume. Our new book is unique. It brings together for the first time recent information on the pathology, not only of the pulses, or grain legumes, but also of the leguminous oil-seed crops and pasture legumes from both temperate and tropical environments. Thus, *The Pathology of Food and Pasture Legumes* has coverage yet broader than our two earlier books, *The Pathology of Tropical Food Legumes* (J. Wiley & Sons, 1983) and *Diseases of Tropical Pasture Plants* (CAB International, 1994), to which the new book becomes a sequel.

About a quarter of the total output of crop protein in the world as a whole comes from legumes which are consequently of great importance in human diets, and it is estimated that, to at least 700 million people, pulses are an essential component of their diet. Furthermore, about 1000 million hectares of land, in the tropics and subtropics alone, are suitable for pastures, and 25% of the land surface of the earth is under permanent pasture that supports a large part of our domestic livestock. In developed countries, grain legumes are a

valuable protein-rich supplement to animal feed, and legumes as a whole are agriculturally important since their symbiosis with *Rhizobium* enables them to yield with less assistance from purchased nitrogen fertilizer than other crops.

Legumes, and perhaps particularly tropical legumes, were a relatively neglected group of crops until about 30 years ago, and concerted attention to pasture species is yet more recent. Both food and pasture legumes now receive considerable emphasis by research teams, both at the national and international levels. The result is a fast-moving field generating information that needs regular, critical review. With research being conducted most often by multidisciplinary teams with a commodity focus, progress in crop improvement has been rapid. However, commodity research sometimes runs the risk of myopia, and synthesis of information across crops can help to share common problems and to give insight to principles applicable to related crop plants, fostering an improved communication between plant scientists in different commodity teams. At a time when resources allocated to commodity crop teams are ever harder to secure, the pertinence of a wider, cross-commodity perspective may be keenly appreciated. This book has been written principally for legume scientists in the hope of kindling closer communication, and thus a greater efficiency, among them. The book's extensive bibliography should extend its use as a source of reference beyond legume science alone, perhaps including those concerned with policy-making in crop improvement among its readership.

The authors are to be congratulated, we feel, on the coverage and quality of their chapters, and it is a pleasure to record our thanks to all contributors, some of whom have undertaken their large tasks at short notice despite the heavy pressure of senior positions, and all have gracefully accepted the ravages of our red pens. Leonora Duque de Allen and I. Nageshwara Rao have given us very considerable secretarial support and David Wood has given us valuable assistance with editing. The inconsistencies and inaccuracies that remain must be borne in large measure by the editors themselves.

Finally, the editors, authors and publishers would like to thank the UK Department For International Development (DFID) for assistance towards the cost of printing the colour plates.

David J. Allen
Jillian M. Lenné

Vernacular and Scientific Names of Some Legume Crops

Adzuki bean	*Vigna angularis* (Willd.) Ohwi & Ohashi
Alfalfa	*see* Lucerne
Alpestrine clover	*Trifolium alpestre* L.
Alsike clover	*Trifolium hybridum* L.
Arrowleaf clover	*Trifolium vesiculosum* Savi
Asparagus bean	*see* Yardlong bean
Bambarra groundnut	*Vigna subterranea* (L.) Verdc.
Bean	*see* Common bean
Berseem clover	*Trifolium alexandrinum* L.
Black gram	*Vigna mungo* (L.) Hepper
Blackeye pea	*Vigna unguiculata* (L.) Walp., cvs with white seed and black hilum
Bonavist bean	*see* Hyacinth bean
Broad bean	*Vicia faba* L., large-seeded types
Butter bean	*see* Lima bean
Butterfly pea	*Clitoria ternatea* L.
Catjang	*Vigna unguiculata* (L.) Walp. cv. gr. *biflora*
Caucasian clover	*Trifolium ambiguum* Bieb.
Centro	*Centrosema pubescens* Benth.
Chickpea	*Cicer arietinum* L.
Cluster bean	*Cyamopsis tetragonoloba* (L.) Taub.
Common bean	*Phaseolus vulgaris* L.
Common stylo	*Stylosanthes guianensis* (Aublet) Sw.
Cowpea	*Vigna unquiculata* (L.) Walp.
Crimson clover	*Trifolium incarnatum* L.
Faba bean	*Vicia faba* L.
French bean	*see* Common bean
Garbanzo	*see* Chickpea
Goa bean	*see* Winged bean
Gram	*see* Chickpea
Green gram	*see* Mung bean
Greenleaf	*Desmodium intortum* (Miller) Urb.

Ground bean	*Macrotyloma geocarpum* (Harms) Marechal & Baudet
Groundnut	*Arachis hypogaea* L.
Guar	*see* Cluster bean
Haricot bean	*see* Common bean
Horse bean	*Mucuna sloanei* Fawcett & Rendle
Horse gram	*Macrotyloma uniflorum* (Lam.) Verdc.
Hyacinth bean	*Lablab purpureus* (L.) Sweet
Indigo	*Indigofera* spp.
Jack bean	*Canavalia ensiformis* (L.) DC.
Jungli bean	*Vigna trilobata* (L.) Verdc.
Kersting's groundnut	*see* Ground bean
Kidney bean	*see* Common bean
Lentil	*Lens culinaris* Medik.
Leucaena	*Leucaena leucocephala* (Lam.) de Wit
Lima bean	*Phaseolus lunatus* L.
Lucerne	*Medicago sativa* L.
Mat bean	*see* Moth bean
Monkey nut	*see* Groundnut
Moth bean	*Vigna aconitifolia* (Jacq.) Marechal
Mung bean	*Vigna radiata* (L.) Wilcz.
Narrow-leaved lupin	*Lupinus angustifolius* L.
Navy bean	*Phaseolus vulgaris* L., cvs with small white seed
Pea	*Pisum sativum* L.
Peanut	*see* Groundnut
Pearl lupin	*Lupinus mutabilis* Sweet
Perennial glycine	*Neonotonia wightii* (Wight. & Arn.) Lackey
Persian clover	*Trifolium resupinatum* L.
Phasey bean	*Macroptilium lathyroides* (L.) Urb.
Pigeonpea	*Cajanus cajan* (L.) Millsp.
Red clover	*Trifolium pratense* L.
Red gram	*see* Pigeonpea
Red hot poker tree	*Erythrina abyssinica* Lam. ex DC.
Rice bean	*Vigna umbellata* (Thunb.) Ohwi & Ohashi
Russell lupin	? *Lupinus polyphyllus* Lindl. × *L. arboreus* Sims
Sand plain lupin	*Lupinus cosentinii* Guss.
Scarlet runner bean	*Phaseolus coccineus* L.
Shrubby stylo	*Stylosanthes scabra* Vog.
Sieva bean	*Phaseolus lunatus* L., small-seeded types
Silverleaf	*Desmodium uncinatum* (Jacq.) DC.
Siratro	*Macroptilium atropurpureum* (DC.) Urb.
Small hop clover	*see* Yellow suckling clover
Snake bean	*see* Yardlong bean
Snap bean	*Phaseolus vulgaris* L., cvs grown for their green pods
Soyabean	*Glycine max* (L.) Merr.
Subterranean clover	*Trifolium subterraneum* L.
Sunn-hemp	*Crotalaria juncea* L.
Sweet clover	*Melilotus* spp.

Sword bean	*Canavalia gladiata* (Jacq.) DC.
Tepary bean	*Phaseolus acutifolius* A. Gray
Tick bean	*Vicia faba* L., small-seeded types
Tree lupin	*Lupinus arboreus* Sims
Tropical kudzu	*Pueraria phaseoloides* (Roxb.) Benth.
Urd bean	*see* Black gram
Velvet bean	*Mucuna pruriens* (L.) DC. var. *utilis* (Wall. ex Wight) Baker ex Burck.
White clover	*Trifolium repens* L.
White lupin	*Lupinus albus* L.
Winged bean	*Psophocarpus tetragonolobus* (L.) DC.
Yam bean: African	*Sphenostylis stenocarpa* (Hochst. ex Rich.) Harms
Mexican	*Pachyrrhizus erosus* (L.) Urban and *P. tuberosus* (Lam.) Spreng.
Yardlong bean	*Vigna unquiculata* (L.) Walp. cv. gr. *sesquipedalis*
Yellow lupin	*Lupinus luteus* L.
Yellow suckling clover	*Trifolium dubium* Sibth.
Zigzag clover	*Trifolium medium* L.

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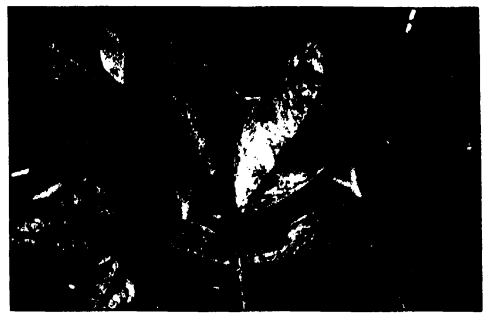

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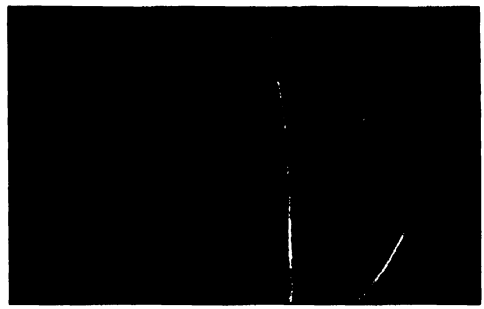

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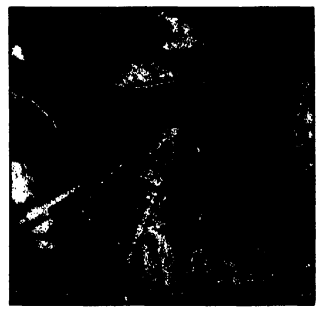

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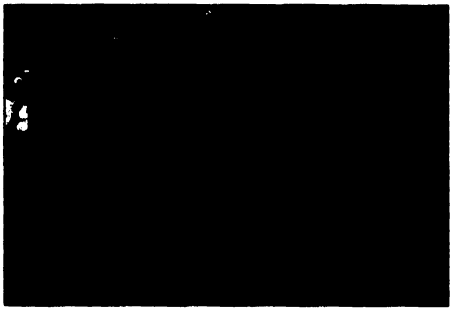
Plate 1. Reddish-brown lesions of early leaf spot (top) and black lesions of late leaf spot (bottom) on abaxial surfaces of groundnut. Photo courtesy of ICRISAT.

Plate 2. Web blotch of groundnut caused by *Phoma arachidicola*. Photo courtesy of P. Subrahmanyam.

Plate 3. Small, curled, distorted, pale yellow leaves caused by chlorotic rosette (top) and dark green leaves with outward rolled margins typical of green rosette (bottom) of groundnut. Photos courtesy of P. Subrahmanyam.

Plate 4. Angular leaf spot lesions on bean leaf. Adaxial surface on left, and on the right, the abaxial surface on which synnemata are conspicuous Photo courtesy of CIAT.

Plate 5. Common bacterial blight lesions on bean leaves. Photo courtesy of CIAT.



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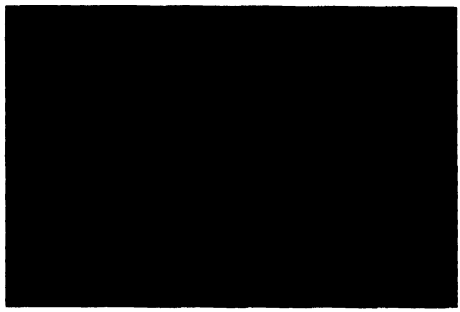

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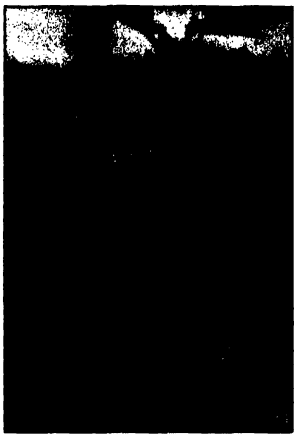

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Plate 6. Halo blight lesions on bean leaves. Photo courtesy of D.J. Allen.

Plate 7. Green vein-banding symptoms induced by blackeye cowpea mosaic virus in Tanzania. Photo courtesy of D.J. Allen and John Wiley & Sons.

Plate 8. The witchweed of cowpea bears a loose spike of dull mauve to pale purple flowers (northern Nigeria). Photo courtesy of J.B. Smithson and John Wiley & Sons.

Plate 9. Interveinal chlorosis is a typical symptom of the root parasite *Striga gesnerioides* (northern Nigeria). Photo courtesy of D.J. Allen.

Plate 10. Water-soaked lesion on middle pea seedling caused by *Aphanomyces euteiches*. This pathogen can attack the plant at all stages of maturity and is not limited to root tips or seedling rot, as are *Pythium* spp. Photo courtesy of J.M. Kraft.

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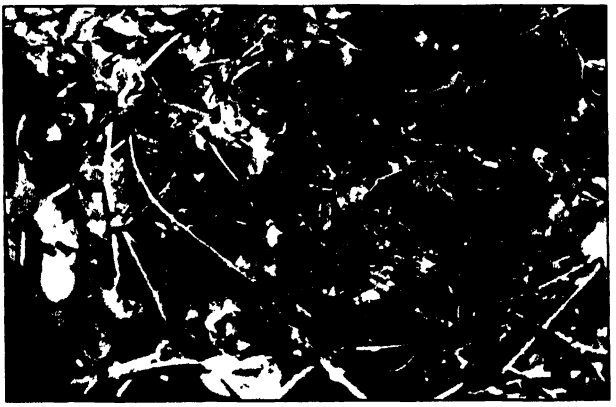

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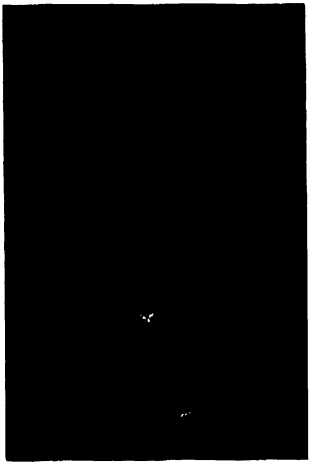

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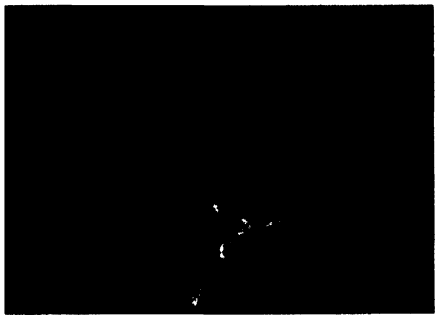
Plate 11. Vascular discoloration of above-ground pea stem from plant infected with *Fusarium oxysporum* f. sp. *pisi* race 2. Photo courtesy of J.M. Kraft.

Plate 12. Vine rot and fluffy white mycelial mass on pea vines on soil surface infected with *Sclerotinia sclerotiorum*. Photo courtesy of J.M. Kraft.

Plate 13. Symptoms of alfalfa mosaic virus in a semi-leafless pea cultivar. Note vascular discoloration and chlorosis. Photo courtesy of J.M. Kraft.

Plate 14. Pod symptoms of pea streak. Note brown necrotic lesions with associated sunken areas. Photo courtesy of J.M. Kraft.

Plate 15. *Ascochyta fabae* on faba bean showing pycnidia in centre of lesion and possible effect of toxin along one midrib. Photo courtesy of G.J. Jellis.



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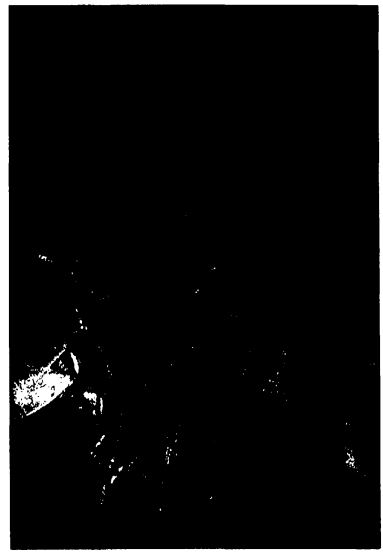

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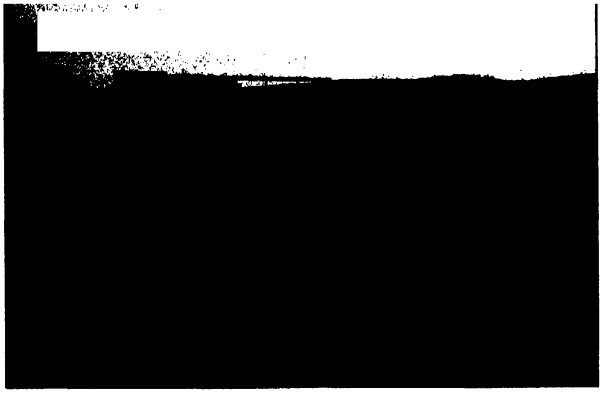

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Plate 16. Downy mildew (*Peronospora viciae*) sporulating on underside of faba bean leaf. Photo courtesy of G.J. Jellis.

Plate 17. Uredosori of lentil rust on leaves and stem. The heavy attack has caused pod abortion. Photo courtesy of CODIS, ICARDA.

Plate 18. Foliar blight from stem girdling by the ascochyta blight pathogen. Photo courtesy of B. Bayaa.

Plate 19. Screening lentil for resistance to vascular wilt using a 'sick-plot' with rows of a repeated susceptible check. Photo courtesy of B. Bayaa.

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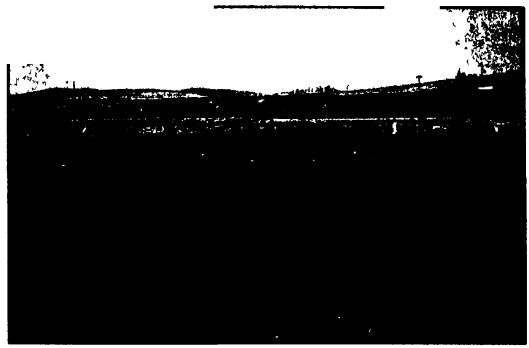

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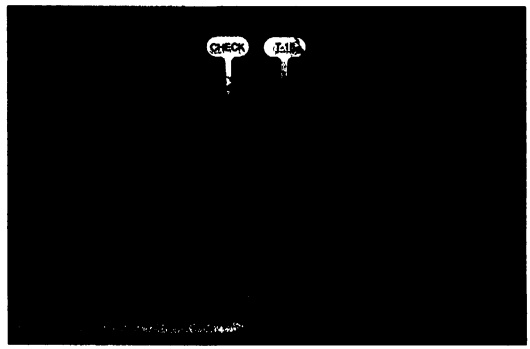

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Plate 20. Broomrape attack in lentil: *Orobanche crenata* (unbranched inflorescence of white flowers) and *O. aegyptiaca* (branched inflorescence of blue flowers). Photo courtesy of CODIS, ICARDA.

Plate 21. Effective field screening for resistance to fusarium wilt in the chickpea wilt-sick plot at ICRISAT Centre, Patancheru, India. Photo courtesy of M.P. Haware.

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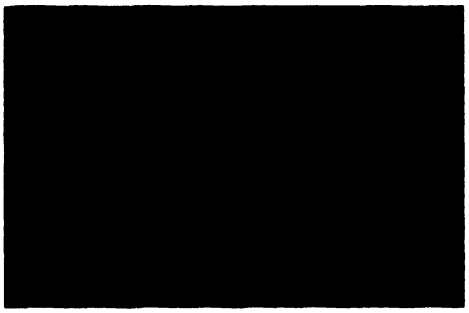
Plate 23. Leaf reddening on *desi* chickpea caused by stunt virus. Photo courtesy of ICRISAT.



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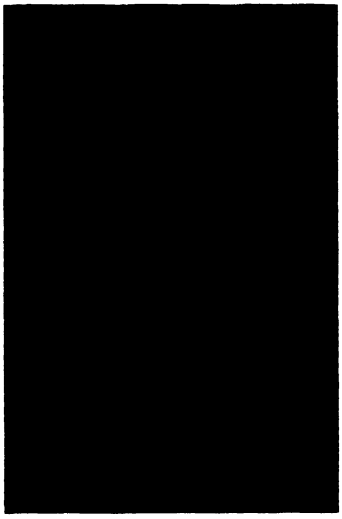

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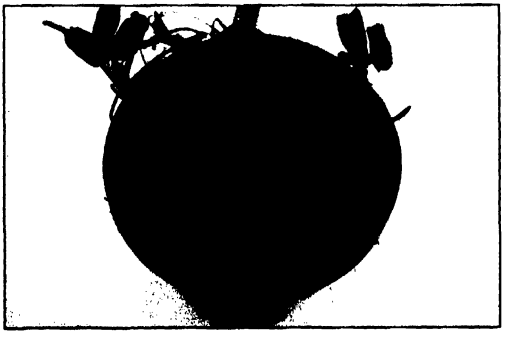

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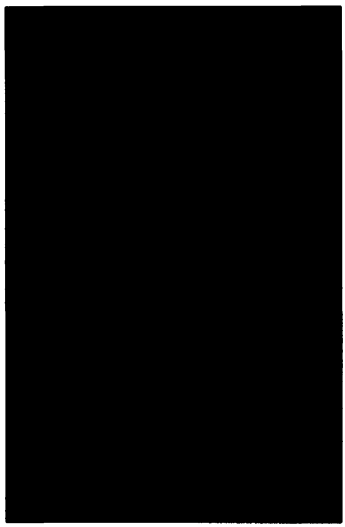
Plate 27. Section of field of red clover affected by fusarium root rot. Photo courtesy of K.T. Leath.

Plate 28. Extensive necrosis of red clover plant following inoculation with *Sclerotinia trifoliorum.* Photo courtesy of P.C. Mercer.



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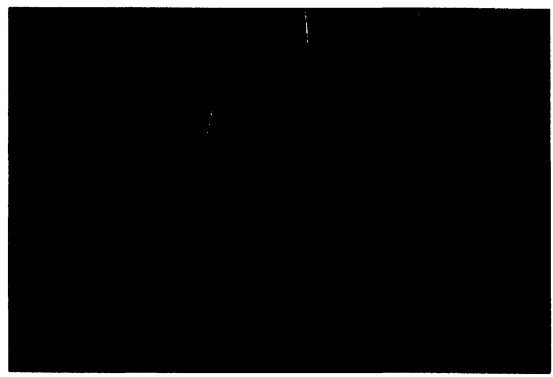

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Plate 29. Wilt-type symptoms of *Kabatiella caulivora* on red clover. Photo courtesy of C.J. O'Rourke.

Plate 30. Phyllody of white clover caused by phytoplasma. Photo courtesy of C.J. O'Rourke.

Plate 31. Symptoms of clover yellow vein on leaves of arrowleaf clover (left) compared with healthy leaves (right). Photo courtesy of M.J. McLaughlin.

Plate 32. Symptoms of white clover mosaic on leaves of white clover. Photo courtesy of M.J. McLaughlin.

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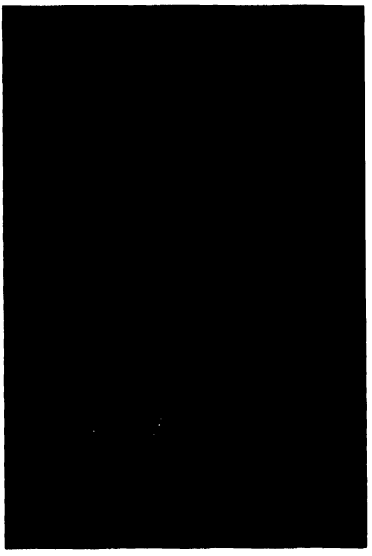

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Plate 33. Symptoms of alfalfa mosaic virus on right-hand leaf of white clover. Centre leaf infected by peanut stunt virus and left-hand leaf healthy. Photo courtesy of M.J. McLaughlin.

Plate 34. Cream to light grey lesions with dark margins on leaves of *Stylosanthes hamata* - Type A anthracnose (top), and black blight lesions on leaves of *Stylosanthes guianensis* - Type B anthracnose (bottom). Photos courtesy of J.M. Lenné and CIAT.

Plate 35. Evaluation of *Stylosanthes* spp. for resistance to anthracnose in Bahia, Brazil. Photo courtesy of J.M. Lenné.

Plate 36. Coalescence of necrotic lesions on leaves of *Centrosema brasilianum* (bottom) and profuse growth of mycelium of *Rhizoctonia solani* on *Stylosanthes guianensis* (top). Photos courtesy of J.M. Lenné.

DISEASE AS A CONSTRAINT TO PRODUCTION OF LEGUMES IN AGRICULTURE

1

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LEGUMES IN AGRICULTURE

Evolutionary Diversity

The *Leguminosae*, which is the third largest family of flowering plants, comprises some 18,000 species. Subfamilies *Mimosoideae* and *Caesalpinioideae* are mostly tropical whereas *Papilionoideae*, which contains most species of economic importance, is more widespread. Some 6600 species are African, and it has been estimated (Le Houerou, 1991) that some 2000 of these are actually consumed as herbage or browse by large herbivores, the palatable species being about evenly distributed in the three subfamilies of *Leguminosae*. Among the genera mentioned in this book, *Cassia* and *Senna* belong to the subfamily *Caesalpinioideae* and *Leucaena* is in the *Mimosoideae*; all other legumes we discuss belong to the *Papilionoideae*, which is separable into botanical tribes. Affinities among the species and their origins are shown in Table 1.1. Legumes show great variety in habit, from trees and shrubs to herbs and climbers. Many have the characteristic of being able to form a symbiotic relationship with a group of bacteria (*Rhizobium* spp.) which can utilize atmospheric nitrogen, and it may be that this peculiar ability is the key to the success of the family which has a cosmopolitan distribution. The *Leguminosae* are characterized by their fruits, which are pods, and by their usually alternate, compound, pinnate or trifoliolate leaves.

In economic importance, the legumes are second only to the grasses among plant families. They are of value firstly for their nitrogen-rich plant material for consumption by humans and their animals, and secondly for the nitrogen-rich residues legumes leave in the soil. The human food they supply is of three kinds: edible tubers, as in the genera *Pachyrhizus*, *Psophocarpus* and *Sphenostylis*; leaf, green pods and unripe seed (*Cajanus*, *Phaseolus*, *Pisum*, *Psophocarpus* and *Vigna*); and ripe dry seed, as in the various pulses or grain legumes. Some are oil-rich,

 The Pathology of Food and Pasture Legumes (eds D.J. Allen and J.M. Lenné)

Table 1.1. Affinities, centres of origin and principal areas of production of the world's major legume crops.[1]

Tribe[2]	Species origin	Centres of origin	Regions of major production (Mt dry seed)[3]
Aeschynomeneae	*Arachis hypogaea*	Central Brazil, northern Argentina, eastern Bolivia, India	India, China, USA, West Africa, Sudan (19.8)
	Stylosanthes guianensis	Central and South America	China, Latin America (n.a.)
	Stylosanthes scabra	South America	Australia (n.a)
Cicereae	*Cicer arietinum*	Middle East	Indian subcont., Mexico, Turkey, Ethiopia (7.8)
Desmodieae	*Desmodium* spp.	South-east Asia	South-east Asia, Latin America (n.a.)
Genisteae	*Lupinus* spp.	Mediterranean, South America	Australia, Poland, former USSR, South Africa (1.5)
Phaseoleae	*Glycine max*	North-east China	USA, Brazil, China, Argentina (113.1)
	Phaseolus vulgaris	Central America, Andean South America	Latin America (Brazil, Mexico), Eastern Africa (8.5)
	Phaseolus lunatus	Central America, Peru	USA, Madagascar, Peru (<1.0)
	Vigna unguiculata	Tropical Africa	West Africa, Brazil, India (2.0)
	Vigna radiata	India, or Indo-Burmese region	India, South-east Asia (1.2)
	Vigna subterranea	Tropical Africa	Tropical Africa (<1.0 ?)
	Cajanus cajan	India (Western Ghats)	India (2.0)
	Lablab purpureus	Africa?	Sudan, Egypt (as grain crop)
	Psophocarpus tetragonolobus	Unknown (Africa?)[4]	Papua New Guinea, South-east Asia
	Centrosema pubescens	Tropical America	Australia, South-east Asia (n.a)
Trifolieae	*Trifolium* spp.	Northern Europe, Near East	Europe (n.a.)
	Medicago sativa	Central Asia, Near East	USA (n.a.) and Mediterranean
Vicieae	*Pisum sativum*	Near East	USA, former Russia, China (10.0); USA, UK, France, India (as green pod)
	Lens culinaris	Mediterranean	India, Turkey (2.6)
	Vicia faba	Near East	China, Ethiopia, Egypt, Morocco, UK (3.8)
Mimoseae	*Leucaena leucocephala*	Central America	Central America, Hawaii, Australia (n.a.)

[1] Based on information supplied in Chapters 2–13 (this volume) which supplements the content of Table 1 in Allen (1997).
[2] Polhill and Raven (1981).
[3] Estimates of world production refer to million tonnes of dry grain and are not applicable (n.a.) to green vegetable, forage and pasture species.
[4] Smartt (1980).

like groundnuts (*Arachis*) and soyabeans (*Glycine*). About a quarter of the total output of crop protein in the world as a whole comes from legumes, and it is estimated that, to at least 700 million people, pulses are an essential component of their diet. Furthermore, about 1000 million hectares of land, in the tropics and subtropics alone, are suitable for pastures, and 25% of the land surface of the earth is under permanent pasture that supports a large part of our domestic livestock. Animal feeds in developed countries rely substantially on supplements from protein-rich grain legumes like soyabeans, lupins and faba beans, and improved pastures rely on clovers or lucerne. In tropical areas, *Stylosanthes* is the single most important pasture legume, and forage and browse legumes like *Leucaena* are important in some areas, while crop residues such as haulms of groundnuts, soyabeans and common beans are important animal feed supplements in developing countries. Many species have multiple uses. Woody species provide timber and fuel and often fertilizer from their ash; others provide fibre (*Vigna*) and green manures (*Crotalaria, Medicago*). Dyes (*Indigofera*), fish poisons (*Tephrosia*), insecticides (*Derris*), laxatives (*Senna*), gums (*Cyamopsis*), fruits (*Tamarindus*) and showy ornamentals (*Amherstia, Erythrina* and *Wisteria*) are among many other uses of the *Leguminosae* (NAS, 1979).

Many of the grain legumes were domesticated very early in history in the major centres of origin of agriculture. Peas, faba beans, chickpeas and lentils are among the early domestications in the Old World, dating from about 7500 BC in the Mediterranean and temperate Eurasia. Among lupins, species of Mediterranean origin appear to have been in cultivation for 3000–4000 years. The Afro-Asian group is indigenous in areas to which agriculture was introduced from the Middle East; soyabeans are more recent domesticates, emerging in north-eastern China perhaps in about 1100 BC. In the New World, the earliest domesticated types of common bean are dated between 8000 and 5500 BC while the groundnut has been dated at 3000–2000 BC. The domestication of pasture legumes is a relatively new process and most tropical pasture legume cultivars are selections from the wild (Lenné and Sonoda, 1990). Since their early origins, the grain legumes have spread widely and, in some cases well illustrated by groundnut and soyabean, the regions of major production today are far removed from centres of origin. The most obvious effects of pulse domestication involve the modification of growth habit. Stems tend to be thicker, leaves larger, branches fewer, the node number less and internode length shorter, a process culminating in the evolution of self-supporting plants well adapted to monocrop farming systems (Allen, 1983; Smartt and Hymowitz, 1985).

Production and Productivity in Legume Farming Systems

The world production of soyabean far exceeds that of any other legume crop, followed by the other oilseed legume, groundnut. Among pulses, the most important in terms of production are pea, common bean and chickpea, followed in some order by faba bean, lentil, pigeonpea, cowpea, mung bean and lupin. Monocropping is the rule in developed countries of temperate and subtropical regions, examples being the production of soyabeans, groundnuts, peas and

common beans in the USA; or common beans in Brazil, lupins in Australia and faba beans in Britain. These are produced by intensive systems of mechanized farming from which grain yields of 1.5–3.0 t $ha^{-1}$are common. However, with the exception of soyabean, the vast majority of the harvest of food legumes comes from small-scale subsistence farms where common practices are mixed cropping and landrace or cultivar diversity, strategies that avoid risk rather than maximize productivity. Added inputs are few and yields are typically low, perhaps 200–700 kg ha^{-1}. Pasture legumes, in both temperate and tropical production systems, are rarely monocropped unless the land is exclusively used for high quality hay. Most pasture legumes are incorporated into grass-based pastures, i.e. at minimum, intercropped. In tropical regions, particularly, perennial *Stylosanthes* pastures are highly heterogeneous plant communities (Lenné, 1989; Lenné, Chapter 13, this volume). It seems probable that there are cogent parallels here between such practices in the production of food legumes and the diversity implicit in pastures. Diseases have played a part in the evolution of tropical agroecosystems within which crop diversity seems a sound strategy for sustaining productivity (Allen, 1983; Smithson and Lenné, 1996). The extent to which disease alone, among other agronomic constraints, can account for the yield gap (between subsistence and 'modern' agriculture) has seldom been well quantified in legumes, and attention is drawn to the need for better assessment of yield loss in the final chapter (Lenné and Allen, Chapter 14, this volume).

Minor Legumes

The following chapters (2–13) give monographic treatment, crop by crop, of groundnut, soyabean, common bean, cowpea, pea, faba bean, lentil, chickpea, pigeonpea, lupins, clovers and tropical pasture legumes. To a point, our choice has been subjective and we can be criticized for various omissions amongst which lima bean, winged bean, mung bean and lucerne (alfalfa) are obvious examples. These and many 'minor' legumes are either locally important or have potential for greater development (NAS, 1979). Much of the substance of Chapters 4 and 5 is relevant to lima bean and mung bean, respectively, and some of the content of Chapter 12 has relevance to lucerne. The literature on the pathology of the more minor species is dispersed and fragmentary. Recognizing the need to capture what is available, we have tabulated information on the diseases of lima bean, hyacinth bean, the Asian *Vigna* species, bambarra groundnut, winged bean, *Leucaena* and lucerne in Tables 1.2 to 1.8.

Many diseases, and the pathogens that cause them, are common to a range of legume species, so that synthesis of information in a critical review could help to highlight particular problems and opportunities. For this reason, the next section of this chapter focuses on the legume pathogens themselves.

Table 1.2. Diseases of lima bean (*Phaseolus lunatus*).

Disease	Pathogen	Distribution/importance	References
Root rots	*Fusarium* spp., *Pythium* sp., *Rhizoctonia solani* Kühn, *Thielaviopsis basicola* (Berk. & Br.) Ferr.	Important in cool humid areas	Lyman *et al.* (1985)
Web blight and stem rot	*Rhizoctonia solani* Kühn	Widespread, and devastating in humid lowland tropics	Warren (1975); Lyman *et al.* (1985)
Ashy stem blight, charcoal rot	*Macrophomina phaseolina* (Tassi) Goid	Widespread; minor	Riley (1960); Toler and Wester (1966)
Stem spot	*Phomopsis phaseoli* (Desm.) Grove (teleomorph = *Diaporthe phaseolorum* (Cooke & Ell.) Sacc.)	Tanzania	Riley (1960)
Anthracnose	*Colletotrichum lindemuthianum* (Sacc. & Magn.) Bri. & Cav.	Widespread? Seldom reported and probably unimportant	Whiteside (1966); Lyman *et al.* (1985); Pastor-Corrales and Tu (1989)
Brown blotch, pod spot, stem anthracnose	*Colletotrichum truncatum* (Schw.) Andrus & Moore	Widespread; common and potentially destructive	Andrus and Moore (1935); Riley (1960); Nattrass (1961); Rothwell (1983); see Allen *et al.* (Chapter 5, this volume)
Scab	*Elsinoe phaseoli* Jenk.	Central America and USA; ? Zambia	Jenkins (1931b, 1933); Bruner and Jenkins (1933); Angus (1962–1966); Sitterly and Epps (1958)
Ascochyta blight	*Ascochyta phaseolorum* Sacc. (=*Phoma exigua* var.*exigua* Desm.), but isolates from Kenya, Zambia and Zimbabwe have been shown to be *P. exigua* var. *diversispora* (Bub.) Boerema	Widespread; locally severe under humid conditions at mid-altitude in Africa	Riley (1960); Angus (1962–1966); Peregrine and Siddiqi (1972); Gerlagh (1987); see Allen *et al.* (Chapter 4, this volume)
Phytophthora blight, 'downy mildew'	*Phytophthora phaseoli* Thaxt.	USA	Thaxter (1889); Thomas *et al.* (1952); Wester *et al.* (1966)

Continued overleaf

Table 1.2. *Continued*

Disease	Pathogen	Distribution/importance	References
Powdery mildew	*Leveillula taurica* (Lev.) G. Arnaud	Tanzania	Riley (1960)
False rust	*Synchytrium dolichi* (Cooke) Gaum.	Tanzania	Riley (1960)
Rust	*Uromyces appendiculatus* (Pers.) Ung.	USA and presumably elsewhere; rather rare	Laundon and Waterson (1965); Stavely and Pastor-Corrales (1989)
	Phakopsora pachyrhizi Syd.	Papua New Guinea, India, Nigeria	Ramakrishnan and Sundaram, (1954); Allen (1979)
	Phakopsora meibomiae (Arthur) Arthur	Central and South America	Kern and Thurston (1943); Vakili and Bromfield (1976); Ono *et al.* (1992)
Cercospora leaf spot	*Cercospora canescens* Ell. & Mart.	Widespread and common	Riley (1960); Angus (1962–1966); Steiner (1975); Williams and Liu (1976); Rothwell (1983)
Leaf spots	*Aristastoma guttulosum* Sutton	Nigeria	Allen (1979)
	Pestalotiopsis versicolor (Speg.) Steyaert	Nigeria	Allen (1979)
	Pseudoplea trifolii (Rostr.) Petr. (=*Leptosphaerulina trifolii* (Rostr.) Petr.)	Zambia	Angus (1962–1966)
	Chaetoseptoria wellmanii Stev.	Central America	Muller (1953)
	Dactuliophora tarrii Leakey	Africa	Leakey (1964)
	Alternaria sp.	Togo	Steiner (1975)
Grey mould	*Botrytis cinerea* Pers.	Tanzania	Riley (1960)
Yeast spot	*Nematospora coryli* Pegl.	Tanzania	Riley (1960)

Bacterial blight	*Xanthomonas* sp., presumably *X. campestris* pv. *phaseoli* (E.F. Smith) Dye	Probably widespread	Angus (1962–1966); Lyman *et al.* (1985)
Bacterial brown spot	*Pseudomonas syringae* pv. *syringae* van Hall	USA; East Africa	Tisdale and Williamson (1923); Thaung and Walker (1957); Riley (1960); Teverson (1991)
Halo blight	*Pseudomonas syringae* pv. *phaseolicola* (Burk.) Young *et al.* (=*Ps. savastanoi* pv. *phaseolicola* (Burk.) Gardan *et al.*)	Eastern and southern Africa; Madagascar	Riley (1960); Teverson (1991)
Cucumber mosaic	Cucumber mosaic cucumovirus	USA	Harter (1938); Anderson (1955)
Common mosaic	Bean common mosaic potyvirus	Widespread	Morales and Bos (1988)
Peanut mottle	Peanut mottle potyvirus	East Africa	Bock *et al.* (1978)
Cowpea mild mottle	Cowpea mild mottle carlavirus	Nigeria	Rossel and Thottappilly (1985)
Golden mosaic	Bean golden mosaic geminivirus	Latin America	Goodman and Bird (1978); Williams (1976);
	Lima bean golden mosaic geminivirus	Nigeria	Vetten and Allen (1983)
Root knot	*Meloidogyne* spp.	Widespread and important	Allard (1954); Toler and Wester (1966); Rachie *et al.* (1980)

Table 1.3. Diseases of hyacinth bean (*Lablab purpureus*).

Disease	Pathogen	Distribution/importance	References
Ashy stem blight, charcoal rot	*Macrophomina phaseolina* (Tassi) Goid	Australia, India, Malaysia, Egypt, Sudan and Kenya	Tarr (1958); Lenné (1990)
Collar rot, southern blight	*Corticium rolfsii* Curzi	Australia	Lenné (1990)
Stem and branch rot, white mould	*Sclerotinia sclerotiorum* (Lib.) de Bary	Australia	Lenné (1990)
Web blight, crown rot	*Rhizoctonia solani* Kühn	Widespread	Riley (1960); Lenné (1990)
Ascochyta blight, leaf spot	*Ascochyta phaseolorum* Sacc. and *A. dolichi* Gonz. Frag.	Australia, Papua New Guinea, Africa	Riley (1960); Angus (1962–1966); Rotwell (1983); Lenné (1990)
Angular leaf spot	*Phaeoisariopsis griseola* (Sacc.) Ferraris	Japan, Kenya	McDonald (1929); Lenné (1990)
Cercospora leaf spot, foliar blight	*Cercospora canescens* Ell. & Mart. and *C. dolichi* Ell. & Mart.	Widespread	Lenné (1990)
Anthracnose	*Colletotrichum lindemuthianum* (Sacc. & Magn.) Bri. & Cav.	Bangladesh, Burma, Trinidad	Nowsher *et al.* (1975); Lenné (1990)
	Colletotrichum truncatum (Schw.) Andrus & Moore	India	Lenné (1990)
Scab	*Elsinoe dolichi* Jenk., Bitanc. & Cheo	China, Philippines, eastern and southern Africa	Cheo and Jenkins (1945); Nattrass (1961); Angus (1962–1966); Lenné (1990)
	Sphaceloma sp.	Uganda	Hansford (1934)
Phyllosticta leaf spot	*Phyllosticta dolichi* Brun. and *Phyllosticta* sp.	Nigeria, Brazil, Kenya	Allen (1979); Lenné (1990)
Septoria leaf spot	*Septoria lablabina* Sacc., *S. dolichi* Berk. & Curt. and *Septoria* sp.	Tanzania, Kenya, Malawi, India, Burma, Philippines	Ebbels and Allen (1979); Lenné (1990)
Rust	*Uromyces appendiculatus* (Pers.) Ung.	Widespread	Lenné (1990)
	Phakopsora pachyrhizi Syd.	Australia	Lenné (1990)
	Phakopsora meibomiae (Arthur) Arthur	Brazil and the Caribbean	Vakili (1981); Ono *et al.* (1992)

Powdery mildew	*Erysiphe polygoni* DC	Venezuela	Stewart and Dagnatchew (1967);
	Leveillula taurica (Lev.) Arnaud	India, Ethiopia, Kenya, Nicaragua	Ondieki (1973); Lenné (1990)
Alternaria leaf spot, foliar blight	*Alternaria alternata* (Fr.) Keissler	Sudan	Lenné (1990)
	A. tenuissima (Pers.) Wilts.	Hong Kong, India, Kenya, Sudan	Lenné (1990)
	A. circinans (Berk. & Curt.) Bolle	India	Garud *et al.* (1977)
Myrothecium leaf spot	*Myrothecium roridum* Fr.	Malaysia, India, Ghana and Sierra Leone	Williams and Liu (1976); Lenné (1990)
Yeast spot	*Nematospora coryli* Pegl.	Tanzania	Riley (1960)
Pyrenochaeta leaf spot	*Pyrenochaeta dolichi* Mohanty	Ghana, Uganda	Lenné (1990)
Bacterial blight	*Xanthomonas* sp.	Tanzania	Ebbels and Allen (1979)
	X. campestris pv. *phaseoli* (E. F. Sm.) Dye	Zimbabwe, Sudan	Tarr (1958); Sabet and Ishag (1969); Rothwell (1983);
Halo blight	*Pseudomonas syringae* pv. *phaseolicola* (Burk.) Young *et al.* (=*Ps. savastanoi* pv. *phaseolicola* (Burk.) Gardan *et al.*)	Ethiopia, Kenya, Tanzania, Zimbabwe	Rothwell (1983); Teverson (1991)
Alfalfa mosaic	Alfalfa mosaic alfamovirus	Sudan	Nour and Nour (1962)
Sunn-hemp mosaic, dolichos enation mosaic	Sunn-hemp mosaic tobamovirus	India	Kassanis and Varma (1975)
Peanut stunt	Peanut stunt cucumovirus	Sudan	Ahmed and Mills (1985)
Dolichos yellow mosaic	Dolichos yellow mosaic geminivirus	India	Capoor and Varma (1950); Harrison *et al.* (1991)
Root knot	*Meloidogyne* sp.	Widespread	Anonymous (1981)
Witchweed	*Striga* sp.	? Africa	NAS (1979)

Table 1.4. Diseases of the Asiatic *Vigna* species.

Disease	Pathogen	Distribution/importance	References
Damping-off, crown rot	*Rhizoctonia solani* Kühn	Widespread; up to 57% mortality in Iran	Kaiser *et al.* (1968); Kataria and Grover (1978)
Seedling blight	*Phomopsis* spp.	South-east Asia	Yang (1978
Damping-off, radicle decay and stem rot	*Pythium aphanidermatum* (Edson) Fitzp., *P. butleri* Subram. and other spp.	Sporadic ? Important in bean sprouts	Williams and Liu (1976); AVRDC (1978); Lawn and Ahn (1985)
Stem rot	*Phytophthora vignae* Purss	On *V. angularis* in Japan	Kitazawa *et al.* (1978)
Brown stem rot	*Cephalosporium gregatum* All. & Chamb. (teleomorph *Phialophora gregata* (All. & Chamb.) Gams)	Japan	Konno and Narikawa (1978)
Stem and root rot, wilt	*Corticium rolfsii* Curzi (sclerotial state *Sclerotium rolfsii* Sacc.)	Minor?	Turner (1971); AVRDC (1978)
Fusarium wilt	*Fusarium* spp.	Minor?	Lawn and Ahn (1985)
Charcoal rot, root rot and leaf blight	*Macrophomina phaseolina* (Tassi) Goid.	Widespread?	Vidyasekaran and Arjunan (1978); Yang (1978)
Web blight, leaf rot	*Rhizoctonia solani* Kühn	Locally damaging?	Turner (1971); AVRDC (1978)
Ascochyta blight	*Ascochyta phaseolorum* Sacc. and/or *Phoma exigua* Desm. var. *diversispora* (Bub.) Boerema	Eastern Africa, India and elsewhere in Asia	Stewart and Dagnatchew (1967); Singh *et al.* (1978a); Gerlagh (1987)
Anthracnose	*Colletotrichum capsici* (Syd.) Butl. & Bisby, *C. dematium* (Pers. ex. Fr.) Grove and apparently *C. lindemuthianum* (Sacc. & Magn.) Bri. & Cav.	Probably widespread	Tripathi and Beniwal (1977); Singh *et al.* (1978b); Ebbels and Allen (1979); Lawn and Ahn (1985)
Scab	*Elsinoe iwatae* Kajiwara & Mukelar and *E. phaseoli* Jenk.	Indonesia; East Africa	Nattrass (1961); Mukelar (1978); Allen (1991)
Cercospora leaf spot	*Cercospora canescens* Ell. & Mart., *Pseudocercospora vignae-reticulatae* Deighton and *Pseudocercospora mungo* Deighton	Widespread and important	Bose (1932); Mew *et al.* (1975); Allen (1979); Deighton (1979)
Zonate leaf spot	*Dactuliophora tarrii* Leakey	Eastern Africa	Leakey (1964); Ebbels and Allen (1979)

Myrothecium leaf spot	*Myrothecium roridum* Tode ex Fr.	India	Srivastava (1980)
Black spot	*Protomycopsis phaseoli* Ramakrishnan & Subraman.	India	Pavgi and Thirumalachar (1953); Prasad *et al.* (1962)
False rust	*Synchytrium* sp.	South-east Asia	Yang (1978)
Rust	*Uromyces appendiculatus* (Pers.) Ung. *Phakopsora pachyrhizi* Syd.	Widespread; Asia and Africa	Bose (1932); Yang (1978); Ebbels and Allen (1979); Lenné (1990); Ono *et al.* (1992)
Powdery mildew	*Erysiphe polygoni* DC.	Widespread and important	Bose (1932); Lawn and Ahn (1985)
Pod rot	*Diplodia* sp.	South-east Asia	AVRDC (1978)
Bacterial blight, leaf spot	*Xanthomonas campestris* pv. *phaseoli* (E. F. Sm.) Dye, pv. *vignicola* Burkh., and pv. *vignaeradiatae* Sabet *et al.*	Probably widespread; comparative studies seem warranted	Sabet *et al.* (1969); Patel and Jindal (1972); D.J. Allen, unpublished (IMI B 6940 and B 6943)
Halo blight	*Pseudomonas syringae* pv. *phaseolicola* (Burkh.) Young *et al.* (= *Ps. savastanoi* pv. *phaseolicola* (Burkh.) Gardan *et al.*)	India, Eastern Africa, USA and New Zealand	Stewart and Dagnatchew (1967); Patel and Jindal (1972); Taylor *et al.* (1996)
Blackgram mottle	Blackgram mottle carmovirus	India and Thailand	Scott and Hoy (1981)
Urdbean leaf crinkle	?	India	Williams *et al.* (1968); Beniwal *et al.* (1980)
Cowpea severe mosaic	Cowpea severe mosaic comovirus	Trinidad	Dale (1949)
Southern bean mosaic	Southern bean mosaic sobemovirus	India	Tremaine and Hamilton (1983)
Alfalfa mosaic	Alfalfa mosaic alfamovirus	Iran, probably widespread	Kaiser (1979); Jaspars and Bos (1980)
Cucumber mosaic	Cucumber mosaic cucumovirus	Widespread?	Purivirojkul and Poehlman (1977)
Bean common mosaic, ? mung bean mosaic	Bean common mosaic potyvirus	Iran and India, probably widespread	Kaiser and Mossahebi (1974)
Bean yellow mosaic	Bean yellow mosaic potyvirus	Indonesia	Iwaki and Auzay (1978)
Tobacco ringspot	Tobacco ringspot nepovirus	Sri Lanka	Vignarajah (1978)
Tomato spotted wilt, leaf curl	Tomato spotted wilt tospovirus	India	Ghanekar *et al.* (1979)
Cowpea mild mottle	Cowpea mild mottle carlavirus	Tanzania	Mink and Keswani (1987)
Mungbean yellow mosaic	Mungbean yellow mosaic geminivirus	Widespread in South Asia	Honda *et al.* (1983); Harrison *et al.* (1991)

Table 1.5. Diseases of bambarra groundnut (*Vigna subterranea*) in Africa.

Disease	Pathogen	Distribution/importance	References
Root rot, southern blight	*Corticium rolfsii* Curzi	Malawi, Mozambique, Tanzania and Zambia	Riley (1960); Peregrine and Siddiqi (1972); Plumb-Dhindsa and Mondjane, (1984); Kannaiyan and Haciwa (1993)
Charcoal rot	*Macrophomina phaseolina* (Tassi) Goid	Togo, Uganda, Zambia	Hansford (1943); Steiner (1975); Kannaiyan and Haciwa (1993)
Fusarium wilt	*Fusarium oxysporum* Schlecht. f. sp. *voandzeiae* Armstrong *et al.*	Tanzania, ? Kenya, Zambia	Nattrass (1961); Ebbels and Billingon (1972); Armstrong *et al.* (1975); Kannaiyan and Haciwa (1993)
Ascochyta blight	*Ascochyta phaseolorum* Sacc.	Madagascar, Zambia, ? Togo	Bouriquet (1946); Angus (1962–1966); Steiner (1975)
Web blight and stem rot	*Thanatephorus cucumeris* (Frank.) Donk (*Rhizoctonia solani* Kühn)	Mozambique, West Africa	Chevaugeon (1952); Deighton (1956); Plumb-Dhindsa and Mondjane (1984)
Cercospora leaf spot	*Cercospora canescens* Ell. & Mart.	Widespread; locally important	Holliday (1980)
	C. voandzeiae Bour.	Madagascar	Bouriquet (1946)
Phyllosticta leaf spot	*Phyllosticta voandzeiae* Bour.	West Africa	Chevaugeon (1952)
	Phyllosticta sp.	Togo, Sierra Leone, Malawi	Deighton (1956); Peregrine and Siddiqi (1972); Steiner (1975)
Powdery mildew	*Erysiphe pisi* DC ex. St.-Am.	Zimbabwe	Rothwell (1983)
	E. polygoni DC	Tanzania	Riley (1960)
	Sphaerotheca voandzeiae Bour.	Madagascar	Bouriquet (1946)
	Oidium sp.	Zambia, Malawi	Angus (1962–1966); Peregrine and Siddiqi (1972)
Rust	*Uromyces appendiculatus* (Pers.) Ung.	Tanzania, Malawi, Zambia	Allen (1975); Ebbels and Allen (1979); Kannaiyan and Haciwa (1993)
	Phakopsora pachyrhizi Syd.	Tanzania	Teri and Keswani (1985)

Scab	*Elsinoe phaseoli* Jenk.	Zambia	Kannaiyan and Haciwa (1993)
Blight	*Phomopsis* sp.	Zambia	Kannaiyan and Haciwa (1993)
Sooty mould	*Meliola vignae-gracilis* Hansf. & Deight.	Sierra Leone	Hansford and Deighton (1948)
On leaves, minor leaf spots	*Mycosphaerella pinodes* (Berk. & Blox.) Vestergren	Zambia	Allen (1991)
	Pleospora sp.	Togo	Steiner (1975)
	Colletotrichum dematium (Fr.) Grove	Togo	Steiner (1975)
	Leptosphaerulina trifolii (Rostr.) Petr.	Malawi	Peregrine and Siddiqi (1972)
Bacterial pustule	*Xanthomonas* sp.	Mozambique	Plumb-Dhindsa and Mondjane (1984)
Cowpea mottle	Cowpea mottle carmovirus	West Africa	Bozarth and Shoyinka (1979)
Voandzeia necrotic mosaic	Voandzeia necrotic mosaic tymovirus	Burkina Faso, Ivory Coast	Fauquet *et al.* (1984)
Peanut mottle	Peanut mottle potyvirus	East Africa	Bock *et al.* (1978)
Root knot	*Meloidogyne javanica* (Treub.) Chitwood	Zambia	Angus (1962–1966)
Alectra	*Alectra* sp.	Zambia	Kannaiyan and Haciwa (1993)
Witchweed	*Striga gesnerioides* (Eilld.) Vatke	Africa	See Allen *et al.* (Chapter 5, this volume)

Table 1.6. Diseases of winged bean (*Psophocarpus tetragonolobus*).

Disease	Pathogen	Distribution/importance	References
Collar rot, seedling mortality	A fungal complex: *Macrophomina phaseolina* (Tassi) Goid., *Rhizoctonia solani* Kühn, *Fusarium equiseti* (Corda) Sacc., *F. moniliforme* J. Sheld., and *F. semitectum* Berk. & Rav.	Papua New Guinea	Price and Munroe (1978a)
Collar rot and leaf blight, web blight	*Rhizoctonia solani* Kühn	India, Malaysia, Papua New Guinea, Nigeria	Voelcker (1953); Price (1978); Sharma and Sohi (1980); D.J. Allen, unpublished
Anthracnose	*Colletotrichum lindemuthianum* (Sacc. & Magn.) Bri. & Cav.	Papua New Guinea	Keane (1974)
On leaves	*Colletotrichum* state of *Glomerella cingulata* (Stonem.) Spauld. & Schrenk.	Nigeria	D.J. Allen, unpublished (IMI 210205)
Leaf spot	*Cercospora* sp. and *Colletotrichum* sp.	Bangladesh	A.Z.M. Nowsher and A. Khan, Bangladesh (1977), personal communication
Leaf spot	*Corticium rolfsii* Curzi	Nigeria	D.J. Allen, unpublished
Leaf spot	*Phaeoseptoria* sp.	Nigeria	Allen (1979)
Leaf spot	*Cercospora psophocarpicola* Yen	Papua New Guinea	Price (1978)
Leaf spot	*Mycosphaerella* sp.	Papua New Guinea	Price (1978)
Leaf spot	*Myrothecium* sp.	Malaysia	McIntosh (1951)
Leaf spot	*Pseudocercospora psophocarpi* (Yen) Deighton	Papua New Guinea, Nigeria	Price and Munroe (1978b); Allen (1979)
Zonate leaf spot	*Corynespora cassiicola* (Berk. & Curt.) Wei	India	Addy and Hazarika (1980)

False rust	*Synchytrium psophocarpi* (Racib.) Gaum.	Papua New Guinea, Java, Philippines, Malaysia Tanzania (on *Psophocarpus ? scandens)*, Uganda and Ghana (on *P. palustris)*	Drinkall (1978); Drinkall and Price, (1979, 1983, 1986); Allen (1991)
Powdery mildew	*Oidium* sp., probably *Erysiphe cichoracearum* DC ex Merat	Papua New Guinea	Price (1977)
Flower blight	*Choanephora cucurbitarum* (Berk. & Rav.) Thaxt.	Papua New Guinea	Price (1978)
Bacterial wilt	*Pseudomonas solanacearum* (E. F. Sm.) E. F. Sm. (=*Burkholderia solanacearum* (E. F. Sm.) Yabuuchi *et al.*)	Malaysia	Abdullah (1980)
Ringspot	Cucumber mosaic cucumovirus	West Africa, Fiji	Fauquet *et al.* (1979); Brunt and Phillips (1981); Rossel and Thottappilly (1985)
Necrotic mosaic	Cowpea mild mottle carlavirus	Ivory Coast	Fauquet *et al.* (1979); Rossel and Thottappilly (1985)
Mosaic	Cowpea mosaic comovirus Cowpea severe mosaic comovirus	East Africa Brazil	Kitajima *et al.* (1979); Allen (1983)
Cyst nematode	*Heterodera radicicola (Greeff) Müller*	Mauritius	de Sornay (1916)
Root knot	*Meloidogyne incognita* (Kofoid & White) Chitwood and other spp.	Widespread	Price (1976); Price and Linge (1979) Eagleton *et al.* (1985)

Table 1.7. Diseases of *Leucaena* species.

Disease	Causal agent	Distribution	Importance
Seedling and root diseases			
Root rot	*Ganoderma lucidum (Fr.)* Karst; *G. applanatum* (Pers.) Pat; *G. tornatum* (Pers.) Bres.	Widespread	Potentially important
Root rot	*Fusarium oxysporum* Schlecht; *F. moniliforme* Sheldon var. *subglutinans* L.; *F. solani* (Mart.) Sacc.	Sri Lanka, India, Mauritius	Minor to locally important
Damping-off and seedling rot	*Fusarium* spp.; *Rhizoctonia solani* Kühn; *Pythium* spp.	Widespread	Locally important
Seedling rot	*Colletotrichum truncatum* (Schwein.) Andrus & Moore	Philippines	Minor
Stem diseases			
Gummosis	Unknown aetiology	Widespread	Important
Stem rot	*Pseudolagarobasidium leguminicola* Chang & Chen	Taiwan	Locally important
Stem canker	*Pirex subvinosus* (Berk. & Br.) Hjortstam	India	Locally important
Blight, canker	Complex of *Calonectria rigidiuscula* (Berk. & Br.) Sacc. and *Fusarium roseum* Fr.	Taiwan	Minor
Foliar diseases			
Leaf spot	*Camptomeris leucaenae* (F.L. Stev. & Dalbey) H. Sydow	Widespread	Locally important
Anthracnose	*Colletotrichum capsici* (H. Sydow) E. Butler & Bisby; *C. crassipes* (Speg.) v. Arx; *C. gloeosporioides* (Penz.) Penz. & Sacc.	Widespread	Minor
Pod rots			
Fusarium pod rot	*Fusarium* sp.	Brazil and Colombia	Minor
Bacterial pod rot	*Pseudomonas fluorescens* (Trev.) Migula Biovar II	Central and South America	Locally important

Main source: Lenné (1990, 1991); Lenné and Trutmann (1994).

Table 1.8. Diseases of lucerne/alfalfa (*Medicago sativa*).

Disease	Causal agent	Distribution	Importance
Root and crown diseases			
Acrocalymma root and crown rot	*Acrocalymma medicaginis* Alcorn & Irwin	Australia	Significantly contributes to poor persistence and productivity
Brown root rot	*Phoma sclerotioides* G. Preuss ex Sacc.	USA (Alaska), Canada, Australia	May be serious in winter and early spring
Phytophthora root rot	*Phytophthora megasperma* Drechs. f. sp. *medicaginis* T. Kuan & D.C. Erwin	Widespread	Especially severe under flood irrigation
Winter crown rot	*Coprinus psychromorbidus* Redhead & J.A. Traquair	USA (Alaska), Canada	Severe, may progressively kill 75–100% of a stand
Root knot	*Meloidogyne hapla* Chitwood	Widespread	Locally important, especially in association with pathogenic fungi
Wilts			
Fusarium wilt	*Fusarium oxysporum* Schlech. f. sp. *medicaginis* (Weimer) Snyder & Hansen	Widespread	Severe, especially under warm, temperate conditions
Verticillium wilt	*Verticillium albo-atrum* Reinke & Berthier	Europe, USA, Canada, New Zealand	Serious, can reduce yields by up to 50% and shorten stand life
Lower stem and crown diseases			
Anthracnose	*Colletotrichum trifolii* Bain & Essary; *C. destructivum* O'Gara; *C. truncatum* (Schwein.) Andrus & Moore	USA, Argentina Australia, Europe, former USSR	Serious, especially in areas with summer rainfall
Root canker, crown and bud rot, stem blight	*Rhizoctonia solani* Kühn (*Thanatephorus cucumeris* (Frank) Donk)	USA, Australia, Iran	Damaging under conditions of high temperature and soil moisture
Sclerotinia crown and stem rot	*Sclerotinia sclerotiorum* (Lib.) de Bary *S. trifoliorum sensu* Kohu	Widespread	Damaging to seedling stands
Leaf and stem diseases			
Common leaf spot	*Pseudopeziza medicaginis* (Lib.) Sacc.	Widespread	Reduction in yields up to 40%, reduced quality of hay
Leptosphaerulina leaf spot	*Leptosphaerulina briosiana* (Pollacci) Graham & Luttrell	Widespread	Economically important during cool, wet summers

Continued overleaf

Table 1.8. *Continued*

Disease	Causal agent	Distribution	Importance
Spring black stem and leaf spot	*Phoma medicaginis* Malbr. & Roum var. *medicaginis* Boerema	Widespread	Destructive in northern temperate regions, reducing yield and quality
Alfalfa stem nematode	*Ditylenchus dipsaci* (Kühn) Filipjer	Widespread	Serious pest in heavy soils, areas of high rainfall and irrigation
Alfalfa enation	Alfalfa enation rhabdovirus (AEV)	Europe, Saudi Arabia	Serious disease in southern France
Alfalfa mosaic	Alfalfa mosaic virus (AMV)	Widespread	Moderate to minor loss to alfalfa but host acts as an important reservoir for other crops including pea

Source: Stuteville and Erwin (1990).

LEGUME PATHOGENS

Pathogen and Disease Diversity

Legume crops throughout the world are prone to attack by the whole gamut of plant pathogens, from fungi, bacteria, phytoplasmas and viruses, to nematodes and parasitic angiosperms. Among these, fungi and viruses are the largest and perhaps most important groups. All parts of the plant are affected at all stages of growth. Among fungi, species of *Aspergillus, Fusarium Pythium and Rhizoctonia* are typically associated with seed and seedling diseases, crown rot, root and stem rots, or wilt. For accounts of *Aspergillus*, which is especially important on groundnut, the reader is referred to McDonald *et al.* (Chapter 2, this volume). The genus *Pythium* has a wide host range among legumes but it is perhaps on soyabean, pea and chickpea that it is most important (Chapters 3, 6 and 9, this volume). Many of the chapters address *Fusarium* and *Rhizoctonia* as legume pathogens. *Macrophomina*, which causes charcoal rot on a range of legume hosts, is reviewed in Allen (1997) and by Sinclair (Chapter 3, this volume) and Haware (Chapter 9, this volume). *Phytophthora* species cause various blights (Allen, 1983), the most important of which are in soyabean (Chapter 3, this volume) and pigeonpea (Reddy *et al.*, Chapter 10, this volume). A stem rot and blight, often called white mould, is caused by *Sclerotinia* species that affect a vast range of plant hosts including many legumes (Allen, 1997; Sinclair, Chapter 3; Kraft *et al.*, Chapter 6; Jellis *et al.*, Chapter 7; Mercer, Chapter 12, this volume). Grey mould and chocolate spot, caused by species of *Botrytis*, are of great importance to faba bean, lentil and chickpea (Jellis *et al.*, Chapter 7; Bayaa and Erskine, Chapter 8; and Haware, Chapter 9, this volume). *Rhizoctonia* also causes aerial blight, often called web

blight, in a range of legumes, among which the disease is particular important in lowland humid tropical environments (Sinclair, Chapter 3; Allen *et al.*, Chapter 5; Lenné, Chapter 13, this volume).

Numerous fungal genera cause various leaf spots; among them the genus *Cercospora* and its allies are especially important. This group, which has been reviewed in Allen (1983), includes the genus *Phaeoisariopsis* which causes diseases of world importance in groundnut and common bean (McDonald *et al.*, Chapter 2; Allen *et al.*, Chapter 4, this volume). *Cercospora* leaf spot fungi are treated also by Sinclair (Chapter 3, this volume), Allen *et al.* (Chapter 5, this volume) and Reddy *et al.* (Chapter 10, this volume). The genus *Stemphylium* causes foliar diseases of consequence in lentil and lupin (Bayaa and Erskine, Chapter 8; Hill, Chapter 11, this volume), and *Phomopsis* is important in soyabean and lupin (Sinclair, Chapter 3; Hill, Chapter 11, this volume). *Septoria* species cause leaf spots in soyabean, cowpea and various tropical pasture legumes (Lenné, 1990; Sinclair, Chapter 3; Allen *et al.*, Chapter 5, this volume).

Downy mildews are important in soyabean and pea (Sinclair, Chapter 3; Kraft *et al.*, Chapter 6, this volume) and powdery mildews are important especially in pea, pigeonpea and clovers (Kraft *et al.*, Chapter 6; Reddy *et al.*, Chapter 10; Mercer, Chapter 12, this volume). Smut fungi are relatively unimportant, although *Entyloma* species cause disease in cowpea and *Aeschynomene* (Allen, 1983; Lenné, 1990). Conversely, rust fungi are important on a wide range of legumes and are subjects well treated in many of the chapters. Perhaps especially notable are recent studies by Hennen and his co-workers on the taxonomy of the groundnut and soyabean rust fungi, as discussed by McDonald *et al.* and Sinclair (Chapters 2 and 3, this volume).

From the fungi affecting legumes, we have chosen four groups for more detailed treatment in the following sections of this chapter. Our choice is inevitably somewhat subjective, but we have borne in mind factors including their importance across legume crops or their comparative neglect in the literature. Our intention is to highlight aspects that seem to deserve greater attention and to derive principles that appear applicable to several host legume crops. The fungal genera we focus on are: *Synchytrium; Colletotrichum; Elsinoe* and *Sphaceloma; and Ascochyta* and *Phoma.*

Among bacterial pathogens of legumes, the most important belong to the two species *Pseudomonas syringae* van Hall and *Xanthomonas campestris* (Pammel) Dowson; we choose the latter for discussion later in this chapter. Host-specific pathovars within *P. syringae* cause major diseases in soyabean, common bean and pea (Sinclair, Chapter 3; Allen *et al.*, Chapter 4; Kraft *et al.*, Chapter 6, this volume); these and other pathovars also cause disease in other legume crops. Fluorescent pseudomonads are recorded as minor pathogens of clover and *Leucaena* (Mercer, Chapter 12; Table 1.7). Bacterial wilts are caused by *Burkholderia solanacearum* (E.F. Sm.) Yabuuchi *et al.* or by *Curtobacterium flaccumfaciens* (Hedges) Collins & Jones. The former is especially important in groundnut (McDonald *et al.*, Chapter 2, this volume) but also affects common bean (Allen, 1995) and *Stylosanthes* (Lenné, Chapter 13, this volume). *C. flaccumfaciens* affects soyabean (Sinclair, Chapter 3, this volume), and various other legumes including common bean in which it appears to have been accorded undue quarantine significance (Allen, 1995).

Phytoplasmas are also common legume pathogens but relatively few cause diseases of great economic importance. The principal exceptions include phyllody diseases of pigeonpea and clover, and little leaf of tropical pasture legumes (Reddy *et al.*, Chapter 10; Mercer, Chapter 12; Lenné, Chapter 13, this volume).

There is a rapidly expanding number of characterized viruses described from natural infections of legumes, with representatives in many of the virus groups now recognized. Among the more important groups are the carlaviruses, furoviruses and potyviruses, all with rod-shaped or filamentous particles. The potyviruses are probably the most important overall and these are selected for further comment below. Among the viruses with isometric particles, the como-, carmo-, bromo- and sobemoviruses are important in legumes in which they are transmitted by beetles; cucumo- and luteoviruses are aphidborne, the former in a non-persistent and the latter in a persistent manner; tymoviruses have no known vector. Alfalfa mosaic virus is currently the only virus known in legumes that possesses bacilliform particles. The geminiviruses, with geminate particles, are a group with members transmitted in legumes either by whiteflies or by leafhoppers. Thrips are important vectors of the tospoviruses, notably in the groundnut crop (McDonald *et al.*, Chapter 2, this volume), and both thrips and nematodes are implicated in the transmission of the nepovirus that causes bud blight in soyabean (Sinclair, Chapter 3, this volume). The furoviruses that cause peanut clump are examples of soilborne, and fungus transmitted, pathogens.

Many of these viruses are seedborne in their legume hosts, some at levels sufficient to have enabled worldwide distribution; others, though seedborne, remain relatively restricted ecologically or geographically, suggesting that seed transmission is inefficient. The carmovirus cowpea mottle seems an example (Allen *et al.*, 1982); there may be others among comoviruses. Geminiviruses are not seedborne: the emerging pattern of relationships among them suggests that whitefly-transmitted geminiviruses from the same area are closely related whereas those from different regions are more distantly related, irrespective of their host species (Natesham *et al.*, 1996).

The economic importance of legume viruses also varies widely, depending in part on the width of virus host range among major crop species, partly on geographic distribution of the virus and partly on the intensity of its damage. The prediction of disease-prone areas by means of geographic information systems, as applied to peanut clump, could prove valuable, but it is salient to note that the importance of diseases like groundnut rosette can vacillate wildly with the season, from trivial losses to catastrophic epidemics (McDonald *et al.*, Chapter 2, this volume). The potential for the more effective management of virus diseases in legumes is reviewed by Lenné and Allen (Chapter 14, this volume).

Among the wide range of nematodes, or eelworms, that parasitize legume crops worldwide, the root-knot nematodes (*Meloidogyne* spp.) are of greatest importance, particularly in tropical regions. About 40 species of *Meloidogyne* are recognized but only four account for 95% of their damage; these are *M. incognita* (Kofoid & White) Chitwood, *M. javanica* (Treub.) Chitwood, *M. arenaria* (Neal) Chitwood and *M. hapla* Chitwood. One or other of these species attacks almost all of the tropical legumes but their economic importance is variable; perhaps overall they cause greater damage to groundnut and to pasture legumes like

Desmodium (McDonald *et al.* and Lenné, Chapters 2 and 13, this volume, respectively). The cyst nematodes (*Heterodera* spp.) are perhaps better adapted to more temperate climates where they are especially damaging to soyabean (*H. glycines* Ichinohe) and to peas (*H. goettingiana* Liebs.) and to a lesser extent to faba bean and, locally, to pigeonpea (Allen, 1983; Sinclair, Chapter 3; Kraft *et al.*, Chapter 6 this volume). The stem nematode, *Ditylenchus dipsaci* (Kuhn) Filipjer, is the most important of several nematode species that attack faba bean (Jellis *et al.*, Chapter 7, this volume). The root lesion nematodes (*Pratylenchus* spp.) are widespread and often important, and the reniform nematode (*Rotylenchulus reniformis* L. & O.) is also widely distributed with a broad host range that includes many legumes. Other nematodes of economic importance to legume crops are more restricted in their distribution; their importance is not confined to their individual effects on crop yield, for nematodes are well known to be involved in disease complexes with other soilborne pathogens and some, like *Xiphenema* species, are virus vectors (Allen, 1983; Lenné and Trutmann, 1994).

Finally, among the groups of legume pathogens with which this book is concerned are the parasitic angiosperms. Those that are parasites of the aerial parts of plants include the dodders (*Cuscuta* spp.) which occasionally are reported from legume crops, including soyabean, common bean, cowpea, chickpea and pigeonpea (Allen, 1983), but they are seldom of much importance. On the other hand, the root parasites are of very great importance. Broomrapes in the genus *Orobanche* are temperate in distribution and have many hosts among legumes, including clovers (*O. minor* Sm.) and the shrubby genera *Ulex*, *Genista* and *Cytisus* (*O. rapum-genistae* Thuill.) in northern Europe, but it is in the Mediterranean region and Near East where *Orobanche* spp. are of greatest importance and rank among major field problems of the region. *O. crenata* Forskall, *O. aegyptiaca* Pers. and *O. ramosa* L. together cause substantial damage to faba bean and lentil crops (Jellis *et al.*, Chapter 7; Bayaa and Erskine, Chapter 8, this volume). In the related family *Scrophulariaceae*, two other genera of root parasites are of consequence, *Alectra* and *Striga*. Of these two, *Striga* is much the more important. *S. gesnerioides* (Willd.) Vatke, which has a relatively broad host range that includes various legume and non-legume genera, is of extreme importance to cowpeas in western and southern Africa. Within the species there is a considerable degree of host specificity, as reviewed by Allen *et al.* (Chapter 5, this volume).

Synchytrium Species as Legume Pathogens

The genus *Synchytrium* belongs to the *Chytrideales* whose species are water- or soil-inhabiting organisms. The genus *Synchytrium* is subdivided into at least six subgenera. *S. desmodii* Munasinghe belongs to *Mesochytrium* whereas all the rest of the species on legumes are placed in subgenus *Woroninella*, in which no thick-walled resting stages occur (Karling, 1977). Although the potato wart pathogen (*S. endobioticum* (Schilb.) Perc.) is well known, the species that have been recorded as parasites of legumes have received relatively little research attention; some 18 species are shown in Table 1.9. The best known of the legume synchytria are

Table 1.9. Species of *Synchytrium* on legumes.

Pathogen	Natural host and distribution	References
Synchytrium aequatoriense (H. Sydow) Gaum.	*Teramnus* sp. in Sudan	Lenné (1990)
S. alysicarpi T.S. Ramkr. & Sundar.	*Alysicarpus vaginalis* in India	Lenné (1990)
S. cassiae Lingappa	*Senna cobanensis* in India	Lenné (1990)
S. citrinum (Lagerh.) Gaum.	*Desmodium intortum* and *D. molliculum* in Caribbean and South America; *Desmodium* sp. in Caribbean, Central America, South-east Asia and Papua New Guinea	Lenné (1990)
S. cookii Lingappa	*Alysicarpus monilifer* and *A. vaginalis* in India	Lenné (1990)
S. crustatum Lingappa	*Indigofera linifolia* and *I. spicata* in India	Lenné (1990)
S. cyamopsae Gupta & Sinha	*Cyamopsis tetragonoloba* in India	Gupta and Sinha (1955)
S. decipiens Farlow	*Vigna vexillata* in Central America	Lenné (1990)
S. desmodii Munasinghe	*Desmodium adscendens* in Tanzania; *D. heterocarpon* and *D. ovalifolium* in Colombia and Ecuador	Lenné (1985, 1990)
S. dolichi (Cooke) Gaum.	*Phaseolus lunatus* in Tanzania; *Vigna unguiculata* and *Vigna* spp. in Africa; *Neonotonia wightii* in Africa; *Glycine* spp. in Australia; and *Rhynchosia* spp. in Venezuela and South Africa	Riley (1960); Allen (1983); Lenné (1990)
S. eriosematis (Henn.) Sydow	*Eriosema psoraleoides* in Uganda	Lenné (1990)
S. minutum (Pat.) Gaum.	*Pueraria* sp. in Asia, Papua New Guinea and Fiji	Lenné (1990)
S. phaseoli Weston	*Phaseolus acutifolius, P. lunatus; Vigna calcarata, V. radiata* and *Vigna* spp.; *Rhynchosia elegans* in Kenya; *Rhynchosia minima* in Zambia; *Neonotonia wightii* in Brazil; and *Macroptilium atropurpureum* in Latin America	Angus (1962–66); Lenné (1990); Lenné and Trutmann (1994)
S. phaseoli Patel., Kulk. & Dhande (=*Protomycopsis phaseoli* Ramakr. & Subram.)		Holliday (1980)

Table 1.9. *Continued*

Pathogen	Natural host and distribution	References
S. phaseoli-radiati Sinha & Gupta	*Cajanus cajan, Vigna mungo* and *V. radiata* in India	Gupta and Sinha (1951)
S. psophocarpi (Racib.) Gaum.	*Psophocarpus tetragonolobus* in Papua New Guinea and South-east Asia; *P. palustris* and *P. ? scandens* in Africa	Drinkall and Price (1979); Allen (1991)
S. rynchosiae Lingappa	*Rhynchosia aurea* in India	Lenné (1990)
S. umbilicatum (Berk. & Br.) Karling	*Cajanus cajan* in India	Ramakrishnan and Sundaram (1954)
S. zorniae Lingappa	*Zornia glochidiata* in India	Lenné (1990)

probably *S. psophocarpi* (Racib.) Gaum. which is an obligate biotroph and causes a serious disease of the winged bean (*Psophocarpus tetragonolobus*) in Papua New Guinea and South-east Asia (Drinkall and Price, 1979; Table 1.6), and *S. desmodii*, the false rust pathogen of *Desmodium* species (Lenné, 1985, 1990; Chapter 13, this volume).

In contrast to *S. endobioticum* which is now widespread in temperate regions of the world, the legume synchytria are essentially tropical. *S. dolichi* (Cooke) Gaum. and *S. phaseoli* Weston have each been recorded from five legume genera in three continents and are the most widespread species. *S. phaseoli* is more frequently recorded from Latin America whereas *S. dolichi* records show an African bias (Lenné, 1990). Many species are apparently confined to a single host genus: *S. desmodii* on *Desmodium*, *S. eriosematis* (Henn.) Sydow on *Eriosema*, *S. crustatum* Lingappa on *Indigofera* and *S. zorniae* Lingappa on *Zornia*, among others (Lenné, 1990). Some legume genera appear to be hosts to several *Synchytrium* species, mostly notably *Rhynchosia* (with *S. dolichi*, *S. phaseoli* and *S. rhynchosiae*) and *Vigna* (with *S. decipiens* Farlow, *S. dolichi*, *S. phaseoli* and *S. phaseoli-radiati* Sinha & Gupta); the extent to which synonymy confuses the picture is unclear, and there is probably scope for comparative studies including cross-inoculations.

Synchytrium species typically induce diseases in legumes variously termed false rust, wart or yellow blister. Symptoms develop especially along the midrib, veins and leaf margins and enlarge to form yellowish-orange to brownish-orange galls, developing on all aerial parts of the plant. Vegetative growth may be arrested, internodes are shortened, and leaves and branches become deformed. Severely infected stems may die back and seed production can be decreased markedly. Seedling mortality can also occur (Lenné, 1985). Typical symptoms of false rust on cowpea and on *Desmodium* and *Macroptilium* are colour-illustrated, respectively, in Singh and Allen (1979) and Lenné and Trutmann (1994). Figures 13.5 and 13.6 illustrate false rust on adult plants and seedlings of *Desmodium ovalifolium*. Losses from false rust have seldom been quantified. Under

intermittent flooding, *S. desmodii* was found to decrease adult plant yield of *Desmodium ovalifolium* by almost 73%. Seedling survival, recruitment to the adult plant population, the soil seed bank, productivity and persistence are all adversely affected (Lenné *et al.*, 1990). Standard area diagrams have been developed and found superior to disease keys in evaluating the severity of false rust in winged beans but the effects on crop loss remain undetermined (Price *et al.*, 1982). Diseased pods are consumed as a delicacy in West Java.

S. desmodii produces both summer sporangia and resting sporangia within galls that develop on leaves, petioles and stems of *D. ovalifolium* (Price, 1987; Lenné, Chapter 13, this volume). Bright yellow summer sporangia develop within a membrane which protrudes from an opened gall from which zoospores are liberated. Each sorus may contain 20–50 summer sporangia. Summer sporangia of *S. desmodii* are apparently not wind-dispersed. Zoospores are released from the sporangia within the galls in guttation droplets, moving in water films along leaf hairs (Price, 1987; Lenné, Chapter 13, this volume). When leaf tissue starts to disintegrate, thick-walled brown resting sporangia form within the gall; these resting sporangia are probably liberated passively by wind and dispersed by grazing animals (Price, 1987). They may also occur as seed contaminants and become implicated in intercontinental spread (Lenné, 1985). In contrast, *S. psophocarpi* produces only summer sporangia. Unopened sori contain 4000–5000 sporangia, each of which contains 100–150 zoospores (Drinkall and Price, 1979). Sporangia, of *S. psophocarpi*, like *S. citrinum* and *S. phaseoli*, are liberated as a dry mass and are airborne. Dispersal has diurnal periodicity, and it has been shown (Drinkall and Price, 1983) that the amount of airborne sporangia is correlated positively with temperature and negatively with humidity and dew. Sporangia are deposited within 15 m of their source. Resting spores are not formed (Drinkall and Price, 1986).

False rust of *D. ovalifolium* is favoured by humid conditions both in Sri Lanka and Colombia (Lenné, 1985; Lenné, Chapter 13, this volume). Similarly, false rust of winged bean in Papua New Guinea is especially damaging in the highlands. It has been shown in each case that high humidity and the presence of free water are essential for germination and infection, and both the humidity and temperature requirements found essential in the laboratory correspond to those prevailing where false rust is epidemic (Drinkall and Price, 1979; Price and Lenné, 1988). Zoospores of *S. psophocarpi* lose their motility within half an hour of their release from the sporangium, encysting after 2–3 h (Drinkall and Price, 1979). Zoospores probably move in water films on plant surfaces and re-infect either the same plant or move by rain-splash to adjacent plants (Price, 1987). At least 4 h of leaf wetness is necessary for infection of seedlings of *D. ovalifolium*, at an inoculum concentration in excess of 3000 zoospores per cotyledon. Price and Lenné (1988) found that maximum infection occurred at the emergence of the first primary leaf, following inoculation with 28,000 zoospores per cotyledon and a 24-h period of leaf wetness. These pathogens are not known to be internally seedborne and their survival seems to depend on sporangial viability in infected debris, in soil, or on perennial hosts (Lenné, 1985). Exposed sporangia of *S. psophocarpi* have been found to lose viability after only 4 days, but when sealed under experimental conditions survival is extended to 24 days (Drinkall and

Price, 1979). However, resting sporangia of other *Synchytrium* species are clearly long-lived in soil and plant debris (Glynne, 1926), and Karling (1977) states that some species within subgenus *Woroninella*, in which no resting stages occur, are capable of overwintering in temperate regions.

Genotypes of *Desmodium ovalifolium* with an erect growth habit tend to be less affected by false rust than semi-prostrate to prostrate ones. A Thai accession (CIAT 13089) has been shown to possess valuable adult plant resistance (Lenné *et al.*, 1990; Lenné and Trutmann, 1994; Lenné, Chapter 13, this volume). In winged bean two lines from Thailand have been found resistant to *S. psophocarpi* and *Psophocarpus scandens* is considered immune (Thompson and Haryono, 1979; Drinkall and Price, 1986). Fungistatic compounds, pterocarpans, have been found in winged bean seed (Preston, 1977) but their involvement in host plant resistance, if any, remains unknown. Resistance in potato to *S. endobioticum* involves hypersensitivity, and physiologic races of *S. endobioticum* have been shown to exist (Brooks, 1953; Olsen and Nelson, 1964). The existence of cultivar-specific pathotypes among the legume synchytria remains undetermined. However, one shred of evidence comes from the very few records of *S. psophocarpi* in Africa where the fungus is known from *P. palustris* in West Africa, but also from a wild winged bean in Tanzania (Allen, 1991) where *P. scandens* is the only species recorded (Verdcourt and Halliday, 1979). Since *P. scandens* is considered immune both in Indonesia and Papua New Guinea, this suggests that the pathogenicity of East African populations of *S. psophocarpi* differ from those in Southeast Asia.

We may conclude that *Synchytrium* species have exacting moisture requirements. Those infecting legumes are airborne and splash-dispersed pathogens of aerial habitats, confined to the humid tropics or to the wet season in semi-arid areas. The wart pathogen of potato, being a soil inhabitant, is freed of this restriction and has been able to adapt to temperate climates, to where it was presumably introduced from an Andean origin.

Colletotrichum Species as Legume Pathogens

Colletotrichum Corda belongs to the *Coelomycetes* in the *Melanconiaceae*. All known teleomorphs belong to the genus *Glomerella* Schrenk. & Spauld. in the *Phyllachoraceae*. However, many species of *Colletotrichum* have no known perfect stage; in other cases, claims of the existence of a teleomorph have not been substantiated. The taxonomy of the genus was extensively reviewed by von Arx (1957) who accepted only 11 species and placed more than 600 species in synonymy with *C. gloeosporioides* and nearly 90 with *C. dematium*. In his treatment of the genus, Sutton (1992) accepted 39 taxa. The recent development of improved diagnostic tools and methods of characterization has raised further questions as to the identity of certain species and the relationships between species. For example, anthracnose of lupins was until recently thought to be caused by *C. gloeosporioides* (Hill, Chapter 11, this volume). However, the development of a species-specific molecular probe for *C. acutatum* (Sreenivasaprasad *et al.*, 1994) has led to this and other pathogens, previously described as *C. gloeosporioides*,

being reclassified as *C. acutatum* (Sreenivasaprasad *et al.*, 1994; Reed *et al.*, 1996; Hill, Chapter 11, this volume). Similarly, investigations of the infection process and host specificity of an isolate of the cowpea anthracnose pathogen from Nigeria suggest that it is best referred to as *C. destructivum* and not *C. lindemuthianum* (Allen *et al.*, Chapter 5, this volume). Using combined biological and molecular approaches, new relationships between species are being established and a firmer foundation for the taxonomy of the genus is being developed (Bailey *et al.*, 1995).

Many *Colletotrichum* species affect legumes, attacking all parts of the plant causing anthracnoses, as well as leaf, stem and pod spots and blights, crown and root rots, and seedling diseases (Lenné, 1992). The diagnostic symptomatology of the group is typified by the ulcer-like lesions formed on legume pods, especially on bean (Fig. 4.3), although a range of other symptoms are found, e.g. brown blotch of cowpea and lima bean and type B anthracnose of *Stylosanthes guianensis* (see Allen *et al.*, Chapter 5, this volume, and Lenné, Chapter 13, this volume). Diseases caused by *Colletotrichum* spp. on soyabean, common bean, cowpea, lupins and tropical pasture legumes are reviewed in Sinclair, Chapter 3, Allen *et al.*, Chapters 4 and 5; Hill, Chapter 11, and Lenné, Chapter 13, this volume, respectively.

At least eight species of *Colletotrichum* have been commonly recorded on legumes in tropical and temperate regions. These include *C. capsici* (H. Sydow) E. Butler & Bisby, *C. crassipes* (Speg.) Arx, *C. dematium* (Pers.) Grove, *C. destructivum* O'Gara, *C. gloeosporioides* (Penz.) Penz. & Sacc., *C. lindemuthianum* (Sacc. & Magn.) Br. & Cav., *C. trifolii* Bain & Essary and *C. truncatum* (Schw.) Andrus & Moore. Legume hosts of these species are given in Table 1.10 and further information is available in Lenné (1990, 1992) and Chapters 3, 4, 5, 11 and 13. Another species, *C. acutatum* Simmonds, affects lupins (see Hill, Chapter 11, this volume) and probably occurs more widely on grain legumes, having frequently been misidentified as *C. gloeosporioides* in the past (Sutton, 1992; Sreenivasaprasad *et al.*, 1994).

The principal *Colletotrichum* species that affect grain legumes are *C. lindemuthianum*, which has a worldwide distribution on common bean (see Allen *et al.*, Chapter 4, this volume) and is thought to affect other grain legumes in the genera *Phaseolus* and *Vigna*, lima, pea, hyacinth bean, winged bean (Tables 1.2, 1.3, 1.4, 1.6); *C. capsici* which is pantropical on groundnut, cowpea, chickpea and soyabean (Table 1.4); *C. truncatum*, another worldwide species whose host range includes soyabean, cowpea, lima bean, pigeonpea and groundnut (Tables 1.2, 1.3); *C. destructivum* affecting soyabean, cowpea and lentil in the USA and Asia; *C. acutatum* on lupins (Hill, Chapter 11, this volume) and possibly other grain legumes; and *C. gloeosporioides* reported from pigeonpea, soyabean, winged bean and groundnut (Table 1.6). The wide host ranges conventionally attributed to some species, e.g. *C. lindemuthianum* and *C. gloeosporioides* (Table 1.10), may prove spurious as suggested by recent studies on cowpea and lupin anthracnose diseases, and future work seems likely to reveal additional anomalies.

The principal *Colletotrichum* species that affect pasture legumes include *C. trifolii*, on lucerne and clovers and *C. gloeosporioides* on tropical pasture legumes of the genera *Aeschynomene*, *Centrosema*, *Desmodium*, *Leucaena*, *Pueraria* and

Table 1.10. Common species of *Colletotrichum* on legumes.[1]

Pathogen	Natural host	References
Colletotrichum acutatum Simmonds	Lupins	Reed *et al.* (1996); see Chapter 11
Colletotrichum capsici (H. Syd.) Butl. & Bis.	Groundnut, chickpea, soyabean, yam bean, cowpea, *Vigna* spp., *Acacia, Desmodium, Leucaena, Stylosanthes* spp.	Lenné (1992); IMI unpublished records; Tables 1.4, 1.7; see Chapters 2, 3, 5, 9, 13
Colletotrichum crassipes (Speg.) v. Arx	Pigeonpea, *Acacia mangium, Leucaena leucocephala*	Lenné (1992); IMI unpublished records; Table 1.7
Colletotrichum dematium (Pers.) Grove	Groundnut, soyabean, pigeonpea, common bean, winged bean, cowpea, bambarra groundnut, *Vigna* spp., lucerne and many tropical pasture legumes	Lenné (1992); IMI unpublished records; Tables 1.4, 1.5; see Chapters 2, 3, 4, 5, 10, 13
Colletotrichum destructivum O'Gara (teleomorph *Glomerella glycines* Lehm. & Wolf)	Lentil, cowpea, lucerne, clovers, *Melilotus alba, Vicia* spp.	Tiffany and Gilman (1954); Lenné (1992); IMI unpublished records; Table 1.8; see Chapters 5, 8, 12
Colletotrichum gloeosporioides (Penz.) Penz. & Sacc. (teleomorph *Glomerella cingulata* (Stonem.) Spauld. & v. Schr.)	Pigeonpea, winged bean, lucerne, *Lespedeza striata, Aeschynomene Centrosema, Desmodium, Leucaena, Stylosanthes* spp. and many other tropical pasture legumes	Weimer (1946); Tiffany and Gilman (1954); Graham *et al.* (1976); Lenné (1992); IMI unpublished records; Tables 1.6, 1.7; see Chapters 10, 13
Colletotrichum lindemuthianum (Sacc. & Magn.) Br. & Cav.	Common bean, *Phaseolus* spp., soyabean, pea, yam bean, hyacinth bean, winged bean, *Vigna* spp., lucerne, *Macroptilium atropurpureum* (Siratro), *Cassia* spp.	Lenné (1992); IMI unpublished records; Tables 1.2, 1.3, 1.4, 1.6; see Chapters 3, 4, 13
Colletotrichum trifolii Bain & Essary	Clovers, lucerne, *Lupinus luteus, Melilotus alba, Vicia* spp.	Welty (1982); Lenné (1992); IMI unpublished records; Table 1.8; see Chapters 11, 12
Colletotrichum truncatum (Schw.) Andrus & Moore	Groundnut, cowpea, soyabean common bean, pea, pigeonpea, hyacinth bean, *Phaseolus* spp., clovers, lucerne, *Centrosema, Stylosanthes, Macroptilium atropurpureum* (Siratro) and many other tropical pasture legumes	Tiffany and Gilman (1954); Lenné (1990, 1992); IMI unpublished records; Tables 1.2, 1.3, 1.8; see Chapters 2, 3, 4, 5, 6, 10, 12, 13

[1] A more detailed review is given by Lenné (1992).

Stylosanthes. *C. truncatum* is also common on these legumes, but is generally less damaging than *C. gloeosporioides* (Lenné and Trutmann, 1994; Lenné, Chapter 13, this volume). Just as anthracnose of lupins was recently proven to be caused by *C. acutatum* and not *C. gloeosporioides*, further analysis may reveal that the former species affects a wider range of tropical pasture legumes. Most species affecting legumes also show considerable pathogenic variation which has necessitated the development of high-input, long-term breeding programmes to control many of them (see Chapters 3, 4, 5 and 13, this volume).

Losses due to *Colletotrichum* diseases of legumes have been described as 'significant', 'substantial', 'considerable' and even 'total'. A review of available literature, however, shows a dearth of both qualitative and quantitative studies. This lack of accurate, long-term data on crop and pasture losses hinders realistic definition of the economic importance of most *Colletotrichum* diseases of legumes. This situation is not, however, unique to *Colletotrichum* diseases of legumes and is discussed in more detail by Lenné and Allen (Chapter 14, this volume).

Colletotrichum diseases of legumes can be controlled by genetic, chemical, cultural, biological (by microorganisms) and integrated management strategies. Availability of many of these strategies in developed countries allows the use of individual and integrated systems especially for grain legumes (see Chapters 3, 4 and 5, this volume). In developing countries, the high cost of chemical control prohibits use of this strategy, except perhaps as a seed treatment; however, integrated control strategies using cultural and genetic options are feasible (Trutmann *et al.*, 1993; see Chapters 4 and 5). The most promising and widely used management strategy in developing countries is the use of resistant cultivars. They are of low cost to the farmer (Allen, 1983), can provide potentially permanent protection against disease, and their adoption does not upset the farming system.

Resistance is also the principal recommended control strategy for anthracnose of lucerne and clovers (Stuteville and Erwin, 1990). Resistance to anthracnose has been easily identified and isolated from lucerne populations and red clover and readily incorporated into productive cultivars in North America and Australia (Elgin and Ostazeski, 1982; Leath, 1985, 1989; Irwin, 1989). Selection for resistance to stem anthracnose also confers resistance to crown rot (Irwin, 1989). Resistance is also the most practical and economic method for managing anthracnose of *Stylosanthes* species (Lenné and Trutmann, 1994). Current breeding programmes are comprehensive and directed towards diverse breeding strategies (Lenné and Trutmann, 1994; Davis and Irwin, 1994). Management strategies are reviewed in detail in Sinclair, Chapter 3, Allen *et al.*, Chapters 4 and 5, Hill, Chapter 11 and Lenné, Chapter 13, this volume.

With the exception of brown blotch of cowpea (Allen *et al.*, Chapter 5, this volume), most diseases caused by *Colletotrichum* species are important under warm, humid conditions. This is directly related to fungal biology as the optimum conditions for disease development of most species affecting legumes include temperatures of 15–28°C and relative humidities greater than 90% (see Chapters 3, 4, 5 and 13). The production of conidia in a mucilaginous matrix in an acervulus facilitates dissemination by rain-splash and wind-driven rain over short distances. For example, in Canada, the spread of infection in common bean

from foci of *C. lindemuthianum* was up to 4.6 m per rainstorm (Tu, 1981). Recent studies have also shown that dry conidial masses may be dispersed as wind-blown particulate matter (Nicholson, 1992) who also noted that conidia may survive for months in the matrix at 45% relative humidity. Survival in crop and pasture residues and seeds has been shown for all *Colletotrichum* species affecting legumes (Lenné, 1992); for example, *C. lindemuthianum* may survive in seed of common bean for up to 5 years (Tu, 1983). There appear therefore to be many similarities among *Colletotrichum* diseases of legumes.

Legume Scab Fungi

The scab fungi of legumes belong to the genus *Elsinoe* in the *Myringiaceae*, with anamorphs in *Sphaceloma*. These fungi have received surprisingly little research attention since the intensive studies of Jenkins (1931a, b; *et seq.*). Among the better known species are *E. fawcettii* Bitanc. & Jenk., the cause of citrus scab, *E. veneta* (Burkh.) Jenk., the pathogen of cane spot of *Rubus*, and *E. brasiliensis* Bitanc. & Jenk., which causes super-elongation of cassava (Zeigler and Lozano, 1983). The species that attack legumes (Table 1.11) have been discussed by Allen (1983) from which this review is an expansion. The extent to which there is synonymy among the species is unclear, though they were erected on the basis of host range and symptoms, and these criteria alone seem insufficient to distinguish species (Zeigler and Lozano, 1983). Certainly, natural host ranges are narrow and essentially genus-specific (Table 1.11). The apparently wider host range of *E. phaseoli*, reported from common bean, lima bean, runner bean, cowpea and mung bean, may be illusory, judging from the much narrower pathogenicity spectra of common bean and cowpea isolates (Emechebe, 1980; Phillips, 1996). The host range of the mung bean pathogen in Africa (Allen, 1991) has not been investigated. Whether or not the host species-specific isolates should be regarded as distinct *formae speciales* of *E. phaseoli*, as proposed by Staples (1958, cited by Allen, 1983), or warrant recognition as separate species, remain unclear. However, the cowpea scab fungus does appear to be distinct. Since its teleomorph has not been reported, it is best regarded as *Sphaceloma* sp. (Emechebe, 1980; Phillips, 1996) rather than as a form of *E. phaseoli*. *E. phaseoli* was first described from *Phaseolus lunatus* in the New World by Bruner and Jenkins (1933) who considered *P. vulgaris* to be a non-host; scab of the common bean remains unknown in the Americas, despite the plant's Latin American origins. The recent work of Phillips (1996) reveals that *E. phaseoli* populations vary in their pathogenicity also at the host cultivar level, with obvious implications for scab resistance breeding.

The diseases caused by this group of fungi are characterized typically by the development of raised corky lesions that develop on most aerial parts of the plant. The plant becomes severely distorted, in some cases as a result of gibberellin production (Zeigler *et al.*, 1980). Early infection in common bean is detectable by distortion of apical growth during the plant's vegetative stage (Allen *et al.*, 1996), followed by the development of the typical corky lesions especially on upper surfaces of leaves. In cowpea it is perhaps stem and peduncle lesions (see Fig. 5.3),

Table 1.11. Species of *Elsinoe* and *Sphaceloma* on legumes.

Pathogen	Natural host	References
Elsinoe canavaliae Racib.	*Canavalia ensiformis* and *C. gladiata*	Jenkins (1931a); Lenné (1990)
Elsinoe dolichi Jenkins *et al.*	*Dolichos* sp., *Lablab purpureus*	Cheo and Jenkins (1945); Lenné (1990)
Elsinoe erythrinae Sivan. & Gomez	*Erythrina* spp. in Brazil and Costa Rica	Lenné (1990)
Elsinoe iwatae Kajiwara & Mukelar	Mung bean in Indonesia	Mukelar (1978)
Elsinoe phaseoli Jenk.	Lima bean, runner bean, common bean, cowpea and mung bean?	Jenkins (1931b); Bruner and Jenkins (1933); Allen (1983); Allen *et al.* (Chapter 5, this volume)
Elsinoe rhynchosiae Jenk, & Wats.	*Rhynchosia calycosa* in Panama	Jenkins and Watson (1962); Lenné (1990)
Elsinoe tephrosiae Hansf.	*Tephrosia candida* and *T. vogelii* in Uganda	Lenné (1990)
Elsinoe wisconsinensis H.C. Green	*Desmodium illoense* in USA	Holliday (1980)
Elsinoe sp.	*Calopogonium caeruleum* in Peru	Lenné (1990
Sphaceloma arachidis Bitanc. & Jenkins	Groundnut in Brazil and Japan, *Arachis glabrata, A. pintoi* in Brazil and Colombia	Bitancourt and Jenkins (1940); Lenné (1990); McDonald *et al.* (Chapter 2, this volume)
Sphaceloma glycines Kurata & Kuribayashi	Soyabean in Japan	Jenkins (1951)
Sphaceloma zorniae Bitanc. & Jenk.	*Zornia* spp. in Latin America	Lenné (1981, 1990)
Sphaceloma sp.	*Cassia obtusifolia* in Zimbabwe, and *Galactia eggersii* in USA	Lenné (1990)

and in *Zornia* and *Arachis* spp. the stem and petiole lesions (see Figs 13.8 and 13.9), that are especially characteristic. Since the illustration of symptoms of scab caused by *Cladosporium vignae* Gardn. (Gardner, 1925) are strikingly similar, it is tempting to suggest that there has been confusion between the two, albeit at a time when Jenkins' pioneering work on the scab fungi had not begun. It is salient to note, too, that Hansford (1937) records *Elsinoe* sp. as a host of a *Cladosporium* sp.

The economic importance of this group of pathogens on legumes is variable. Some species are destructive only locally, including the scab of groundnut in Brazil, soyabean scab in Japan (Allen, 1983), scab of *Zornia latifolia* under humid conditions in Brazil and Colombia (Lenné, 1981) and hyacinth bean scab in

China (Cheo and Jenkins, 1945). Others are widespread and very destructive, and their importance may well have been underestimated. Common bean scab causes yield losses of 50% in South Africa and up to 70% in Kenya (Phillips, 1994). Work in Mbala, Zambia (D.C. Greenberg, Chipata, 1985, personal communication) revealed a negative correlation between scab and bean seed yield of −0.82, suggesting that the disease is important under some conditions. Cowpea scab is perhaps the most important of all, causing almost complete crop loss in some cases. It is considered the most important fungal disease of cowpea both in Africa and in north-east Brazil; in northern Nigeria, losses of at least 71% can occur when scab is severe (Allen *et al.*, Chapter 5, this volume).

Most of the scab fungi are known to be seedborne and survive intercrop periods on infected debris, but the relative importance of the anamorph and teleomorph in survival and spread appears not to have been investigated. Bean scab has been estimated to spread (principally by wind) at the rate of 7.5 m over 6 weeks (Mutitu, 1979). In most cases, host plant resistance seems likely to be a sound strategy for scab disease management. Sources of scab resistance have been found in *Arachis pintoi* (Lenné and Trutmann, 1994), in cowpea (Allen *et al.*, 1981b), and in common bean (Phillips, 1995).

Ascochyta and *Phoma* Species as Legume Pathogens

Ascochyta and *Phoma* are two closely related form-genera within the *Sphaeropsidales*. They are distinguished on the basis of septation of conidia, being essentially two-celled in *Ascochyta* and without septation in *Phoma*. *Ascochyta* species show annelidic ontogeny: conidial septation is an essential part of conidial completion so that the conidia are always two-celled or more. *Phoma* species show phialidic ontogeny and the conidia are in principle aseptate, although secondary septation may occur (Holliday, 1980). Nevertheless, there remains considerable confusion between the two genera as well as synonymy between species: a good example is *Ascochyta phaseolorum* Sacc. which is considered a synonym of *Phoma exigua* Desm. by some authors (Boerema, 1972) but apparently not by others (Holliday, 1980).

The species of *Ascochyta* and *Phoma* that have been reported as legume pathogens are shown in Table 1.12. Only anamorphs are known in many of these species; known teleomorphs belong essentially either to *Didymella*, in the *Venturiaceae*, or to *Mycosphaerella* in the *Dothideaceae*. Species of *Didymella* include the ascochyta blight pathogens of chickpea, lentil and faba bean and the web blotch pathogen of groundnut, whereas the genus *Mycosphaerella* includes one of the ascochyta blight pathogens of pea, though this species, too, is sometimes placed in *Didymella* (Muller and von Arx, 1962). *Ascochyta pinodes (Mycosphaerella pinodes)* is homothallic whereas *A. fabae* f. sp. *lentis* (= *A. lentis*, teleomorph *Didymella lentis*; W.J. Kaiser, Pullman, Washington, 1996, personal communication) and *A. rabiei* (*D. rabiei*) are heterothallic (Jellis and Punithalingam, 1991; Trapero-Casas and Kaiser, 1992; Kaiser and Hellier, 1993).

There is considerable variation in the nature and severity of symptoms induced by this group of fungi. Discrete spotting of leaves, stems, peduncles and

Table 1.12. Species of *Ascochyta* and *Phoma* on legumes.

Pathogen	Natural host	References
Ascochyta adzamethica Schosch see *Phoma arachidicola*		
Ascochyta arachidis Woronich. see *Phoma arachidicola*		
Ascochyta boltshauseri Sacc. (=*Stagonosporopsis hortensis* (Sacc. & Malbr.) Petr.)	Common bean, cowpea	Allen (1983, 1995)
Ascochyta cassiae Henn.	*Senna* spp.	Lenné (1990)
Ascochyta caulicola Laub.	Sweet clover (*Melilotus* spp.)	Dickson (1956)
Ascochyta dolichi Gonz. & Frag.	Hyacinth bean, *Vigna parkeri*	Lenné (1990)
Ascochyta erythrinae Elisei	*Erythrina* spp.	Lenné (1990)
Ascochyta fabae Speg. (teleomorph *Didymella fabae* Jellis & Punith.)	Faba bean	Jellis *et al.* (Chapter 7, this volume)
Ascochyta fabae Speg. f. sp. *lentis* Gossen *et al.* (teleomorph *Didymella* sp.)	Lentil	Bayaa and Erskine (Chapter 8, this volume)
Ascochyta imperfecta Pk. see *Phoma medicaginis* Malbr. & Roum.		
Ascochyta lentis Vassil. see *A. fabae* f. sp. *lentis*		
Ascochyta lethalis Ell. & Barth. (= ? *A. meliloti* Trus.) (teleomorph *Didymella lethalis* (Stone) Sivan. (= *Mycosphaerella lethalis* Stone))	Lucerne, clovers	Dickson (1956); Holliday (1989)
Ascochyta meliloti Trus. see *A. lethalis*		
Ascochyta phaseolorum Sacc. (=*Phoma exigua* var. *exigua* Desm.)	Common bean, cowpea, soyabean, hyacinth bean, lima bean, bambarra, groundnut	Boerema (1972); Allen (1983); Lenné (1990)
Ascochyta pinodes L.K. Jones. (teleomorph *Mycosphaerella pinodes* (Berk. & Blox.) Vestergr. (=*Didymella pinodes* (Berk. & Blox.) Petrak))	Pea, common bean, *Lathyrus* and *Vicia*	Holliday (1980); Allen (1995); Kraft *et al.* (Chapter 6, this volume)
Ascochyta pisi Lib.	Pea	Kraft *et al.* (Chapter 6, this volume)

Table 1.12. *Continued*

Pathogen	Natural host	References
Ascochyta rabiei (Pass.) Lab. (=*Phoma rabiei* (Pass.) Khune & Kajoor) (teleomorph *Didymella rabiei* (Kovachevski) v. Arx (=*Mycosphaerella rabiei* Kovachevski))	Chickpea	Trapero-Casas and Kaiser (1992); Haware (Chapter 9, this volume)
Ascochyta sojicola Abramoff	Soyabean, *Neonotonia wightii, Glycine ussuriensis*	Allen (1983); Lenné (1990)
Ascochyta trifolii Bond. & Truss.	Clovers	Moore (1959)
Phoma anguina Berk. & M.A. Curtis	*Alysicarpus* spp.	Lenné (1990)
Phoma arachidicola Marasas *et al.*, (teleomorph *Didymella arachidicola* (Chochrjakov) Taber *et al.*)	Groundnut	McDonald *et al.* (Chapter 2, this volume)
Phoma bakeriana Sacc.	Cowpea	Allen (1983)
Phoma cajani Rangel	Pigeonpea	Lopez-Roza (1969); Lenné (1990)
Phoma exigua var. *exigua* Desm. (=*Ascochyta phaseolorum* Sacc.)	Common bean, cowpea, soyabean, hyacinth bean, lima bean, bambarra, groundnut	Boerema (1972); Allen (1983); Lenné (1990)
Phoma exigua Desm. var. *diversispora* (Bubak) Boerema	Common bean, cowpea, hyacinth bean	Gerlagh (1987); Allen (1991)
Phoma glomerata (Corda) Wollenw. & Hochapf.	Cowpea, pigeonpea	Lenné (1990); D.J. Allen, unpublished record (IMI 200636)
Phoma herbarum var. *medicaginis* Westend. see *P. medicaginis*		
P. macrostoma Mont.	*Macrotyloma uniflorum, Senna* spp.	Lenné (1990)
Phoma medicaginis Malbr. & Roum.	Lucerne, chickpea, *Glycine ussuriensis*	Dickson (1956); Boerema *et al.* (1965); Haware and Nene (1981); Lenné (1990)
Phoma medicaginis Malbr. & Roum. var. *pinodella* (Jones) Boerema (=*P. trifolii* E.M. Johnson & Valleau = *Ascochyta pinodella* L.K. Jones)	Pea, red clover, lentil	Boerema *et al.* (1965); Kraft *et al.* (Chapter 6, this volume)
Phoma minutella Sacc. & Penz.	*Centrosema pubescens, Vigna adenantha*	Lenné (1990)
Phoma phaseoli Desm.	Common bean	Şesan and Dumitras (1979)

Continued overleaf

Table 1.12. *Continued*

Pathogen	Natural host	References
Phoma phaseolina Pass.	Common bean	Allen (1995)
Phoma rabiei (Pass.) Khune & Kajoor. see *Ascochyta rabiei*		
Phoma sclerotioides G. Preuss ex Sacc.	Lucerne	Stuteville and Erwin (1990)
Phoma sorghina (Sacc.) Boerema *et al.*	Common bean, pigeonpea, *Centrosema, Desmodium, Erythrina, Lablab, Macroptilium, Stylosanthes, Teramnus, Zornia*	Lenné (1990); Allen (1995)
Phoma subcircinata Ell. & Everh.	Common bean	Allen (1995)
Phoma terrestris Hans.	Common bean	Schwartz (1989)
Phoma trifolii E.M. Johnson & Valleau. see *P. medicaginis* var. *pinodella*		

pods are typical of many species, including *Ascochyta pisi* on pea, *A. sojicola* on soyabean, *Phoma bakeriana* on cowpea and *P. sorghina* on a wide range of legumes. Some cause stem canker, like *Phoma cajani* on pigeonpea (Lopez-Rosa, 1969; Lenné, 1990) and *P. medicaginis* which causes spring black stem on lucerne; others including *P. medicaginis* var. *pinodella* and *P. sclerotioides* also cause foot and root rots (Boerema *et al.*, 1965, Stuteville and Erwin, 1990). However, the most important symptoms are blights which often affect all aerial parts of the plant, and ascochyta blights rank among the most important diseases of common bean, cowpea (Allen *et al.*, Chapters 4 and 5, this volume), pea (Kraft *et al.*, Chapter 6, this volume), faba bean (Jellis *et al.*, Chapter 7, this volume), lentil (Bayaa and Erskine, Chapter 8, this volume), chickpea (Haware, Chapter 9, this volume) and groundnut (as web blotch; McDonald *et al.*, Chapter 2, this volume) The reader is referred to these chapters for greater detail. The association of *Ascochyta* with leaf spots and of *Phoma* with plant parts other than leaves (Barnett, 1960) appears wholly fallacious.

The degree of host specificity found among these fungi is also very variable. Fungi like *Ascochyta phaseolorum/Phoma exigua* var. *exigua* have a very wide host range among legumes and apparently other plant families. Conventionally regarded as a weak wound parasite (Crossan, 1958; Alcorn, 1968), outbreaks of severe blight of common bean led to the recognition of a distinct taxon possessing much greater host specificity (Boerema *et al.*, 1981), and *P. exigua* var. *diversispora* is now recognized as the principal cause of foliar blight of common bean and probably also of cowpea, hyacinth bean (*Lablab purpureus*) and several other species of *Vigna* and *Phaseolus* in Africa (Gerlagh, 1987; Allen, 1991). Insufficient host range studies have been made with *P. exigua* var. *diversispora*, but on present evidence it may be host-tribe-specific, with hosts among the

Phaseoleae. There is no evidence that the ascochyta blight pathogens of either common bean or cowpea exhibit pathogenic variability within this taxon, rather that several morphologically distinct species are part of an ascochyta blight complex (Allen *et al.*, Chapter 4, this volume). Similarly, there are several related but distinct species associated with ascochyta blight of lentils (Bayaa and Erskine, Chapter 8, this volume) and in peas, wherein the existence of an 'ascochyta blight complex' is particularly well illustrated (Kraft *et al.*, Chapter 6, this volume; Bowen *et al.*, 1996). In comparing isolates of *P. medicaginis* from various hosts, plant parts and soil, Cormack (1945) found no evidence of host specificity. Subsequent studies (Schenk and Gerdemann, 1956) of both *P. medicaginis* and *P. medicaginis* var. *pinodella* revealed that, although all isolates caused the same types of symptoms on both lucerne and red clover, all were more pathogenic to the host species from which each had been isolated originally, indicating host adaptation at the species level. Isolates of *P. medicaginis* var. *pinodella* from pea in Australia appear not to vary pathogenically (Ali *et al.*, 1978). However, qualitative differences in host resistance and susceptibility do appear to exist in pea (Clulow *et al.*, 1991) and genetic variation among isolates both of *A. pinodes* (Ali *et al.*, 1978; Clulow *et al.*, 1991) and *A. pisi* (Ali *et al.*, 1978; Darby *et al.*, 1986) has been demonstrated. Both *A. fabae* and *A. fabae* f. sp. *lentis* have narrow host ranges that are essentially host-genus-specific, and the same is true of *A. rabiei* on chickpea and its close relatives within the genus *Cicer*. The extent to which host specificity is found at the cultivar level among this group of fungi appears to be debatable. No physiological races of the lentil or groundnut pathogens have yet been identified, but races of the faba bean fungus have been reported (Hanounik and Robertson, 1989; Rashid *et al.*, 1991), though some lines revealed no differential host–pathogen interactions. Further work to validate those races and to standardize methods is warranted (Jellis *et al.*, Chapter 7, this volume). The case of ascochyta blight in chickpea is especially complex and deserves more comment.

Variation both in the degree of host susceptibility of chickpea cultivars and between isolates in a range of characters including pathogenicity has been established for many years (Sattar, 1933; Luthra *et al.*, 1939). The nature of this host–parasite interaction has remained ill-understood, if not also contentious, and it is a matter of profound concern that this remains so (see Haware, Chapter 9, this volume): the amount of resources devoted to work on ascochyta blight of chickpea over recent years is evidence enough. On the one hand, the conventional view is that physiological races of the pathogen exist (Aujla, 1964; Grewal, 1984) and that the evolution of new races accounts for the reported breakdown of resistance which is under monogenic control (Singh and Reddy, 1983). Evidence for the existence of physiological races is not strong, as argued elsewhere (Allen, 1983); such variation in pathogenicity between isolates as does exist is probably attributable to variable aggressiveness, which is quantitative in nature and usually without significant reversals in the ranking order of host cultivars (Gowen *et al.*, 1989), a conclusion drawn also by Buddenhagen and Kaiser (1988) after reviewing the evidence on the nature of pathogenic variation. Studies of the nature of resistance have often also lent weight to the view that the *A. rabiei*–chickpea relationship is essentially quantitative in nature (Boorsma,

1980; Pieters and Tahiri, 1986; Gowen *et al.*, 1989; van Rheenen and Haware, 1993; Haware, Chapter 9, this volume). That this evidence appears to have been ignored seems to be attributable to several factors, including a lack of understanding of the concept of aggressiveness (Buddenhagen and Kaiser, 1988), and a tendency to regroup data on plant response to make two distinct categories ('resistant' and 'susceptible'), a dilemma nicely treated by Robinson (1987) that could be pertinent to work on other legume–blight relationships.

The relative economic importance of this group of legume pathogens perhaps has tended to be understated. Although species that cause only discrete spotting can be accepted as minor pathogens, the blight pathogens do cause severe damage to their respective hosts, in some cases accounting for complete crop failure under conditions that favour disease development. The taxa that possess the greatest host specificity tend to be the most important. *Phoma sorghina*, which has a very wide host range among legumes as well as cereals, is known to produce a mycotoxin, the significance of which appears uncertain (Rabie *et al.*, 1975).

Ascochyta and *Phoma* species are known to be seedborne, with implications both for their international dissemination and for their survival. Seedborne inoculum retains its viability for several years, in some cases over a wide range of temperature (Kaiser, 1989). Infected debris and stubble are also important sources of primary inoculum in several of the ascochyta blights but the relative significance of infected seed and infected straw seems likely to depend on the environment and the farming system as well as the biology of the pathogen. In species that produce chlamydospores, these structures may be presumed to have a role in saprophytic survival. *A. pinodes* survives overwinter in the UK in soil, whether as pycnidiospores, mycelium, chlamydospores or sclerotia. The survival of pycnidiospores depends partly on their transformation into chlamydospores in soil, and it seems that *A. pinodes*, in contrast to *A. pisi*, is a moderately successful saprophyte (Dickinson and Sheridan, 1968). *P. medicaginis* overwinters as pycnidia in dead lucerne stems (Toovey *et al.*, 1936). Factors including the burial of infected straw, and increases in humidity appear to decrease survival periods (Kaiser, 1973; Navas-Cortes *et al.*, 1995)). The main sources of primary inoculum are most widely considered to be rain-splashed conidia which spread over relatively short distances. For instance, ascochyta blight of faba bean spreads only 6–10 m, and in lentils about 10–30 m, from an infection source (Hewett, 1973; Pederson *et al.*, 1993). In view of the seasonal effects on the distance of spread sometimes reported (Bond and Pope, 1980), it seems probable that windborne ascospores from pseudothecia of the teleomorph are involved, but more work is needed to clarify the role of ascospores in dispersal of the faba bean pathogen (Jellis and Punithalingam, 1991; Jellis *et al.*, Chapter 7, this volume). Conversely, there is now solid evidence (Trapero-Casas and Kaiser, 1992) that the teleomorph of the chickpea ascochyta blight pathogen locally plays an important role in the epidemiology of the disease. Approximately 15,000 ascospores per square millimetre are estimated to be discharged energetically from the surface of infected crop residue at a time of year (early March–late May in the Pacific Northwest of the USA) when the chickpea crop is in the vegetative stage, indicating that ascospores of *Didymella rabiei* do serve as primary inoculum for

epidemics. The extent to which the teleomorph is important in other regions of the world, where the climate between chickpea crops is cool and moist, remains to be established. On the Indian subcontinent where the teleomorph is considered absent, the chickpea crop season ends with rising temperatures, contrary to the situation in those areas where *Didymella* has been found (Haware, Chapter 9, this volume). But this seems not to be the whole story. The rapidity with which blight develops is well established (Sattar, 1933) but not fully explained. Buddenhagen and Kaiser (1988) raise the suggestion that the teleomorph may, indeed, occur in a hitherto undetermined area of cold hills from where its airborne ascospores could play a key role in long-distance dissemination to foci of re-infection after the off-season. Critical studies along these lines are keenly awaited.

The effective management of this group of diseases in all cases rests on an integrated approach. Components include a range of cultural practices including crop rotation, field sanitation, the manipulation of sowing date and, especially, the use of clean seed. Seed dressing with fungicide is widely advocated and foliar sprays may possibly have application in some farming systems. In subtropical and tropical areas, the use of barrier crops and intercropping would seem to have potential (Luthra *et al.*, 1935, Moreno, 1975; van Rheenen *et al.*, 1981). The application of mulch may also deserve more attention in some areas (Allen *et al.*, Chapter 4, this volume). Success with the identification of host plant resistance to the ascochyta blights has been variable: high levels of resistance have proved elusive in many of the host–pathogen relationships, and varying degrees of partial resistance have necessitated the adoption of cultural or chemical measures in support of them. In the common bean, higher levels of resistance have been found within related species in the secondary gene pool and, in lentil and chickpea, sources of resistance have also been located in wild relatives (Allen *et al.*, Chapter 4, this volume; Bayaa and Erskine, Chapter 8, this volume; Haware, Chapter 9, this volume). Relatively little is known of mechanisms of resistance. Factors in various ways associated with resistance include straw length (Lockwood *et al.*, 1985), flower and seed colour, and so also the concentration of tannins, the accumulation of phenolics, and even the secretion of malic acid from glandular hairs (Hafiz, 1952; Vir and Grewal, 1974; Jellis *et al.*, Chapter 7, this volume). Working on chickpea, Hafiz (1952) found that penetration in blight-resistant cultivars tended to be delayed relative to the infection process in susceptible cultivars, perhaps in a manner similar to the mechanism of resistance in pea to *Ascochyta pisi* (Darby *et al.*, 1981). Very little is known about the durability of resistance, nor is there much known about its genetic control. Resistance in lentil is controlled by three major genes (Bayaa and Erskine, Chapter 8, this volume), and a total of seven genes for resistance have been identified in faba bean, all either monogenic or oligogenic (Rashid *et al.*, 1991; Jellis *et al.*, Chapter 7, this volume). A combination of minor and major genes is implicated for chickpea (Haware, Chapter 9 this volume). No major genes have been identified in pea against *Ascochyta pinodes*, and Kraft *et al.* (Chapter 6, this volume) advocate the combination of minor genes for resistance. In common bean and cowpea, too little is known, but on present evidence the relationship between host and pathogen appears quantitative (Allen *et al.*, Chapters 4 and 5, this volume). In

such situations it is tempting to suggest that partial resistance seems likely to be durable, but evidence either way remains scanty. Physiological races have been defined in several of these pathogens (Grewal, 1984; Rashid *et al.*, 1991) and claims have been made that resistance has proven transient (Aujla, 1964; Grewal, 1984). The occurrence of teleomorphs (Jellis and Punithalingam, 1991; Trapero-Casas and Kaiser, 1992; Kaiser and Hellier, 1993) and of parasexuality (Sanderson and Srb, 1965) each seem likely to have important implications for the maintenance of genetic diversity in these fungi. In general, there seems a need for more investigations into the nature of host–parasite interaction.

Legume Pathogens Among Xanthomonads

There are at least 20 apparently distinct pathogens that naturally infect legume crop plants (Table 1.13); all are currently regarded as pathovars of the single species *Xanthomonas campestris* (Pammel) Dowson (Bradbury, 1986). The taxon of pathovar, which may be regarded as synonymous with *forma specialis* in fungi (Holliday, 1989), is used for the host-specific 'nomenspecies' below the level of subspecies for which a set of international standards have been set (Dye *et al.*, 1980). However, Vauterin *et al.* (1995) emphasize that the classification of xanthomonads on the single phenotypic feature of host specificity is not sound, suggesting that DNA hybridization studies are liable to reveal truer genomic relationships, so that further revision of the genus seems imminent.

There is considerable overlap in host range of the pathovars amongst which there is perhaps confusion in the literature. One example will suffice. *X. c.* pv. *vignicola* infects cowpea and common bean (Burkholder, 1944; Vakili *et al.*, 1975), mung bean in Ethiopia (Allen *et al.*, Chapter 5, this volume) and *Vigna pubigera* in Sudan (Bradbury, 1986); *X. c.* pv. *vignaeradiatae* is known from mung bean in Sudan (Sabet *et al.*, 1969); and *X. c.* pv. *phaseoli*, the cause of common blight in *Phaseolus* species, is recorded also in India from the Asian *Vigna* species that were formerly considered as *Phaseolus* (Patel and Jindal, 1972, 1973). Clearly, there is need for comparative studies, perhaps particularly between isolates from mung bean (*Vigna radiata* = *Phaseolus aureus*). Conversely, four pathovars infecting *Desmodium* are recognized (Table 1.13). Dye (1958) has drawn attention to the close affinity also between *X. c.* pv. *phaseoli* and *X. c.* pv. *malvacearum*, the cotton bacterial blight pathogen.

Xanthomonads induce a range of symptoms in their legume hosts, from seedling blight (pv. *cassiae* in chickpea) and stem canker (pv. *cajani*) to bacterial leaf spot and foliar blight. Discrete leaf lesions, with or without chlorotic haloes, are sometimes raised in the manner typical of the bacterial pustule diseases of soyabean (pv. *glycines*) and cowpea (pv. *vignaeunguiculatae*). The most destructive are the blights in which all above-ground parts of the plant are infected, including stems, peduncles and pods as well as leaves. In some cases, separate syndromes are distinguishable (Allen *et al.*, Chapter 5, this volume) in the manner of bacterial blight of cotton, in which angular leaf spot, blackarm and boll rot are components of the disease (Innes, 1983). Estimates of crop loss range from 15% from bacterial pustule of soyabeans (Hartwig and Johnson, 1953) to as much

Table 1.13. Pathovars of *Xanthomonas campestris* as legume pathogens.

Bacterium	Host, disease and distribution	References
Xanthomonas campestris		
pv. *alfalfae* (Ricker *et al.*) Dye	Lucerne and red clover leaf spot	Bradbury (1986)
pv. *cajani* (Kulkarni *et al.*) Dye	Leaf spot and stem canker of pigeonpea; China, India, Sudan, Fiji	Kulkarni *et al.* (1950); Sabet *et al.* (1969); Davis *et al.* (1989)
pv. *cassiae* (Kulkarni *et al.*) Dye	Leaf spot of *Cassia tora* and seedling blight of chickpea; India, Sudan, China	Rangaswami and Prasad (1959); Bradbury (1986)
pv. *clitoriae* (Pandit & Kulkarni) Dye	Leaf spot of *Clitoria biflora* in India	Bradbury (1986)
pv. *cyamopsidis* (Patel *et al.*) Dye	Leaf spot and blight of *Cyamopsis tetragonoloba* in India, South Africa and USA	Patel *et al.* (1953); Bradbury (1986)
pv. *desmodii* (Patel) Dye	Leaf spot of *Desmodium diffusum* in India	Bradbury (1986)
pv. *desmodiigangetici* (Patel & Moniz) Dye	Leaf spot of *Desmodium gangeticum* in India	Bradbury (1986)
pv. *desmodiilaxiflori* (Pant & Kulkarni) Dye	Leaf spot of *Desmodium laxiflorum* in India	Bradbury (1986)
pv. *desmodiirotundifolii* (Desai & Shah) Dye	Leaf spot of *Desmodium rotundifolium* in India	Desai and Shah (1960)
pv. *erythrinae* (Patel *et al.*) Dye	Leaf spot of *Erythrina indica* in India	Bradbury (1986)
pv. *glycines* (Nakano) Dye	Bacterial pustule of soyabean; widespread and locally damaging. Also on *Neonotonia wightii* in south-central Africa, and on *Dolichos uniflorus* in India	Patel *et al.* (1949); Hartwig and Johnson (1953); Allen (1975); Lenné and Trutmann, (1994)
pv. *lespedezae* (Ayres *et al.*) Dye	Leaf spot and blight of *Lespedeza* spp. in USA	Bradbury (1986)
pv. *patelii* (Desai & Shah) Dye	Leaf spot of *Crotalaria juncea* in India	Bradbury (1986)
pv. *phaseoli* (E. F. Sm.) Dye	Common bacterial blight of common bean; widespread, a major disease. Recorded also from *Phaseolus acutifolius, P. lunatus, Lablab purpureus, Macroptilium lathyroides, Strophostyles helvola* and various Asian *Vigna* spp.	Sabet and Ishag (1969); Patel and Jindal (1972); 1973); Bradbury (1986); Allen *et al.* (Chapter 4, this volume)
pv. *pisi* (Goto & Okabe) Dye	Pea blight in Japan	Holliday (1989)
pv. *rhynchosiae* (Sabet *et al.*) Dye	Leaf spot of *Rhynchosia memnonia* in Sudan	Sabet *et al.* (1969)

Continued overleaf

Table 1.13. *Continued*

Bacterium	Host, disease and distribution	References
pv. *sesbaniae* (Patel *et al.*) Dye	Leaf spot of *Sesbania* spp. in India	Bradbury (1986)
pv. *vignaeradiatae* (Sabet *et al.*) Dye	Mung bean in Sudan and perhaps elsewhere	Sabet *et al.* (1969); Bradbury (1986)
pv. *vignaeunguiculatae* Patel & Jindal	Bacterial pustule of cowpea in Africa	Williams (1975); Patel and Jindal (1982); Allen *et al.* (Chapter 5, this volume)
pv. *vignicola* (Burkh.) Dye	Bacterial blight of cowpea; widespread and damaging. Also infects common bean, mung bean and *Vigna pubigera*	Burkholder (1944); Vakili *et al.* (1975); Allen (1983); Allen *et al.* (Chapter 5, this volume); D.J. Allen, unpublished (IMI B6940 and 6943)

as 77% in bacterial pustule of cowpea (Omotunde, 1987). Bacterial blight of cowpea has been estimated to cause losses of 26–100%, depending on environment as well as the degree of cultivar susceptibility (Kishun, 1989). In the common bean, yield losses in the range of 10% to over 40% in susceptible cultivars are reported (Wallen and Jackson, 1975; Opio *et al.*, 1992). Recent work in Uganda indicates that for each 1% increase in the incidence of the disease during reproductive growth of the common bean crop there is a concomitant loss in seed yield of 4–12 kg ha^{-1}, depending on the season (Opio *et al.*, 1992).

These bacterial pathogens are typically seedborne in legumes both as internal infections and as external contaminants (Shekhawat and Patel, 1977; Cafati and Saettler, 1983). *X. c.* pv. *phaseoli* may infect seed of both susceptible and resistant host cultivars (Cafati and Saettler, 1980; Aggour *et al.*, 1989) and this must account for the pathogen's widespread distribution. The seedborne nature of xanthomonads is often thought crucial in pathogen survival between seasons but it is also often found to constitute the main source of primary inoculum (Opio *et al.*, 1993). An initial inoculum of 0.2–0.5% infection of bean seed (Weller and Saettler, 1980; Opio *et al.*, 1993), and of 1% of cowpea seed (Shekhawat and Patel, 1977), is sufficient to initiate an epidemic. Opio *et al.* (1993) have shown that the minimum bacterial population necessary to initiate field infection was 10^2 colony-forming units per seed, and Weller and Saettler (1980) concluded that symptoms develop from primary inoculum once the bacterial population reaches a threshold of 5×10^6 colony-forming units per 20 cm^2 of tissue. Infected cotyledons and primary leaves serve as sources of secondary infection. Spread within the crop is favoured by wind-driven rain and soil particles associated with mechanical injury (Claflin *et al.*, 1973), by irrigation and agricultural implements (Saettler, 1989) and presumably by animals. The extent to which grazing herbivores are vectors of xanthomonads in pasture legumes seems uncertain, though sheep do transmit plant pathogenic pseudomonads (Starr and

Kercher, 1969). Certain insects including various beetles (Kaiser and Vakili, 1978) and whitefly (Sabet and Ishag, 1969) are known to be vectors of xanthomonads in legumes. The intercropping of legumes with cereals can sometimes retard the secondary spread of these diseases (van Rheenen *et al*, 1981; Ouko and Buruchara, 1989).

Outbreaks of blight occur occasionally where certified seed is used, suggesting the existence of primary inocula other than seed. The existence of epiphytic colonies of xanthomonads on various weeds and non-host plants has long been recognized (Gardner, 1924; Jones, 1961) and more recent work has confirmed this (Groth and Braun, 1989; Ramos *et al.*, 1991; Opio *et al.*, 1995). It seems that, under some tropical conditions at least, symptomless weeds may play an important role in xanthomonad survival by acting as reservoirs which bridge the gap between successive crops. Plant pathogenic xanthomonads do not produce resting spores, their competitive saprophytic ability is slight and their association with the soil is transient (Buddenhagen, 1965; Schuster and Coyne, 1974). Nevertheless, it is clear that both infested soil and infested crop residues are sources of primary inoculum under certain conditions. *X. c.* pv. *phaseoli* has been shown to survive for as long as 18 months in hyacinth bean debris when kept dry (Sabet and Ishag, 1969). When debris is buried, or under moist conditions, survival is greatly decreased. Under Ugandan field conditions, the bacterium was found to survive in infested soil and debris for several months between common bean crops; when two successive seasons passed without a bean crop, the pathogen was eradicated from the soil (Opio *et al.*, 1994). Essentially similar results are reported from tropical America (Santana *et al.*, 1990). However, in temperate North America, *X. c.* pv. *phaseoli* appears not to survive at all in plant residues (Saettler *et al.*, 1986). The xanthomonads of legumes are rather less susceptible to high temperatures than are the pseudomonads, borne out by differences in their ecological distribution. Whereas halo blight of common bean (*Pseudomonas syringae* van Hall pv. *phaseolicola*) and the bacterial blights of pea and soyabean (*P. s.* pv. *pisi* (Sackett) Young *et al.* and pv. *glycinea* (Coerper) Young *et al.*, respectively) are widespread and damaging in temperate and cool highland tropical areas, the xanthomonads are also prevalent in the hot lowland tropics (Allen, 1983). This indicates that disease management strategies need to be appropriate to tropical agriculture, often in subsistence settings.

Chemical control of xanthomonads has been largely unsatisfactory, whether applied as foliar sprays or as seed dressings. Various copper compounds provide some protection, and antibiotic formulations have shown some potential though there is risk of the development of antibiotic-resistant strains (Allen, 1983; Saettler, 1989). Cultural practices include field sanitation, crop rotation and the production of clean seed, the latter especially if suitably arid areas are available. Since symptomless plants can produce contaminated seed, sensitive assays for detection of the bacterium may be necessary, but the feasibility of operating conventional seed certification schemes in tropical regions (wherein farmers' save their own seed) seems remote. The best strategy is likely to be an integrated one in which several of the above-mentioned components are combined with host plant resistance in a manner deemed locally appropriate.

Sources of resistance have been found in soyabean against bacterial pustule (Lehman and Woodside, 1929), in cowpea against bacterial pustule (Williams, 1977) and blight (Sherwin and Lefebvre, 1951; Allen *et al.*, 1981b), and in common bean against bacterial blight (Coyne and Schuster, 1973). Levels of available resistance are variable, in some cases providing only partial protection, and some appears under monogenic control (Hartwig and Lehman, 1951; Patel, 1982) and in other cases it is essentially quantitatively inherited (Beebe, 1989). Physiological races of these xanthomonads appear seldom to threaten the stability of resistance as do the major legume pseudomonads (Fett and Sequeira, 1981; Taylor *et al.*, 1989, 1996). In soyabean, pustule resistance has proven durable (Jindal *et al.*, 1981) and in cowpea current evidence suggests that available sources of non-hypersensitive resistance may be race non-specific (Allen *et al.*, 1981b; Patel 1981). In common bean, it appears that populations of *X. c.* pv. *phaseoli* do vary in pathogenicity but that physiological races can only be defined by use of *Phaseolus acutifolius*; common bean cultivars cannot yet distinguish them, with the result that the host–bacterium relationship is stable (Opio *et al.*, 1996). It is tempting to suggest that there may be parallels between the bacterial blights of common bean and cotton, in which major genes for resistance have been transferred by interspecific hybridization and superimposed upon an essentially quantitative host–parasite relationship (Arnold and Brown, 1968) so that variability for virulence now poses a problem (Innes, 1983). The gradual evolution of these xanthomonads toward increasingly sustained plant-to-plant infection cycles, liberated from the requirement of a soil saphrophytic phase (Buddenhagen, 1965), may underlie the degree of host specialization they now exhibit. But whether or not one might expect the cool-climate pseudomonads to have progressed further than the more tropical xanthomonads in this regard remains less clear.

Potyviruses and Legumes

The potyviruses, which are named after potato virus Y, are the largest group of plant viruses now often referred to the family *Potyviridae*. All have flexuous filamentous particles mostly 730–790 nm long, containing single-stranded RNA. They are transmissible experimentally by inoculation with sap and, in nature, by various aphids in the non-persistent manner. Potyviruses that naturally infect legume crop plants are shown in Table 1.14; various others that have been less well characterized are omitted. Among the potyviruses of legumes, some (like bean yellow mosaic) have wide host ranges; others, that perhaps tend to be more efficiently transmitted through seed, have rather narrow host ranges. Some are transmitted through pollen. Infected plants show various mosaic and mottle symptoms.

Much attention has been paid recently to relationships within the *Potyviridae*, and increased knowledge of coat protein structure has contributed greatly in this regard. Distinct potyviruses have a 31–71% sequence homology in their coat proteins whereas homology is greater than 90% among strains of the same virus (Shukla and Ward, 1989). Subgroups of potyviruses have been

proposed (Dijkstra and Khan, 1992) and include the bean yellow mosaic and the bean common mosaic potyvirus subgroups. Work on bean common mosaic virus has led to its separation into two distinct viruses, necrosis-inducing strains being referred to the newly delineated bean common mosaic necrosis virus (Allen *et al.*, Chapter 4, this volume). A newly defined bean common mosaic virus now embraces isolates of adzuki bean mosaic, blackeye cowpea mosaic and peanut stripe viruses (Anonymous, 1994).

Potyviruses are transmitted through seed of many but not all their legume hosts. Rates of transmission vary with host species and cultivar, the time of infection and environment, as well as with the virus and its strain. Bean common mosaic and soybean mosaic viruses have long been recognized as seed-transmissible (Allen, 1983). Peanut mottle virus is transmitted in seed of groundnut but not of cowpea or soyabean (Bock and Kuhn, 1975), whereas seed transmission of bean yellow mosaic virus is less common. Certain host cultivars appear to possess a resistance to seed transmission, as shown in cowpea to blackeye cowpea mosaic virus (Allen *et al.*, Chapter 5, this volume), in soyabean to soybean mosaic virus (Goodman and Oard, 1980) and in groundnut to peanut mottle virus (McDonald *et al.*, Chapter 2, this volume). Seed transmission plays a crucial role in the ecology of potyvirus disease both in survival and in providing foci of infection for the subsequent season, and the efficiency of transmission accounts for the widespread distribution of many of the legume potyviruses.

All the legume potyviruses are transmitted also by aphids in a non-persistent manner. Whereas a relatively wide range of aphid species has been demonstrated to act as vectors, few studies have been made of their relative importance under field conditions nor is much known of aphid behaviour in relation to potyvirus transmission in legumes. One notable exception is the work on soybean mosaic (Halbert *et al.*, 1981) and another is the more recent work with blackeye cowpea mosaic in cowpea in Nigeria (Allen *et al.*, Chapter 5, this volume). Weeds and wild legumes are sometimes found to be reservoirs of potyvirus infection (Sengooba *et al.*, 1993; McDonald *et al.*, Chapter 2, this volume) and aphids are presumably responsible for virus spread to the crop.

Strains, most often defined by variation in symptoms, are recognized in most of these potyviruses. Symptom expression is extremely variable and it is often difficult to identify the causal virus by symptomatology alone. Symptomless infection is known but potyviruses typically induce a range of mosaics, mottling, vein clearing, epinasty, cupping and blotching of foliage, and sometimes plant stunting or a rosette, as in pea seedborne mosaic infection. Chlorotic stripes or rings are characteristic of infection with some strains of peanut stripe virus (McDonald *et al.*, Chapter 2, this volume). An apical necrosis that leads to the lethal 'black root' in common bean is a symptom in some strain–cultivar combinations in bean common mosaic necrosis (Allen *et al.*, Chapter 4, this volume) and apparently similiar necroses in soyabean and cowpea are associated also with infections by soybean mosaic virus (Cho and Goodman, 1979) and blackeye cowpea mosaic virus (Kannaiyan and Haciwa, 1993), respectively. Yield losses also vary widely, depending on the susceptibility of the cultivar, the virus and strain, time of infection and environment. Peanut mottle virus infection of groundnut seldom leads to losses above 6%; peanut stripe can cause as much as 70% loss and

Table 1.14. Potyviruses as legume pathogens.

Virus	Natural hosts	Distribution/importance	References
Bean common mosaic	*Phaseolus* spp., *Lupinus luteus*, *Rhynchosia minima* and perhaps *Crotalaria* and *Vigna* spp.	Worldwide; major	Morales and Bos (1988)
Bean yellow mosaic	Common bean, pea, soyabean, faba bean, chickpea, lentil, mung bean, clover and many others	Worldwide; major	Bos (1970); Allen (1983); Jellis *et al.* (Chapter 7, this volume); Mercer (Chapter 12, this volume)
Soybean mosaic	Soyabean and others incl. *Centrosema* spp.	Worldwide; major	Lenné and Trutmann (1994); Sinclair (Chapter 3, this volume)
Peanut mottle	Groundnut and other *Arachis* spp.; soyabean, common bean, lima bean, cowpea, bambarra, groundnut, lupin, *Centrosema, Desmodium* and *Stylosanthes* spp.	Worldwide; moderately important	Bock *et al.* (1978); Lenné and Trutmann (1994); McDonald *et al.* (Chapter 2, this volume); Lenné (Chapter 13, this volume)
Cowpea aphidborne mosaic	Cowpea and others	Widespread, perhaps especially circum-Mediterranean and western Asia; major	Allen *et al.* (Chapter 5, this volume)
Blackeye cowpea mosaic	Cowpea, *Crotalaria spectabilis*, *Centrosema* and *Desmodium* spp.	Pantropical; major	Purcifull and Gonsalves (1985); Allen *et al.* (Chapter 5, this volume); Lenné (Chapter 13, this volume)
Peanut stripe	Groundnut, soyabean and others	Widespread	Mishra *et al.* (1993); Demski *et al.* (1984); McDonald *et al.* (Chapter 2, this volume)

Adzuki bean mosaic	*Vigna angularis*	Japan	Tsuchizaki and Omura (1987)
Pea seedborne mosaic	Pea, faba bean, lentil and chickpea	Widespread; major	Kraft *et al.* (Chapter 6, this volume)
Groundnut eyespot	Groundnut	West Africa; minor	Dubern and Dollet (1980)
Peanut green mosaic	Groundnut	India; minor	McDonald *et al.* (Chapter 2, this volume)
Clover yellow mosaic	*Trifolium* spp.	Widespread; major	Mercer (Chapter 12, this volume)
Passion fruit woodiness	*Centrosema* spp., *Macroptilium atropurpureum*	Australia; local or minor	Lenné and Trutmann (1994)
Cassia severe mosaic	*Senna (=Cassia) occidentalis*	Yemen and Ethiopia	Walkey *et al.* (1994)

soybean mosaic often causes crop losses in excess of 50% (McDonald *et al.* and Sinclair, Chapters 2 and 3, this volume, respectively). Complete crop loss can occur from infection with bean common mosaic necrosis virus (Allen *et al.*, Chapter 4, this volume).

The production of virus-free seed has great potential in the management of legume potyvirus disease in agricultural systems wherein this is feasible. Certain insecticides may possibly have potential in controlling these diseases but, in view of the rapidity with which potyviruses can be introduced into the crop by aphids, virus incidence can actually be higher in sprayed plots relative to unsprayed controls, especially when the incidence of incoming alates is high. Host resistance against the vector seems also ineffective (Allen *et al*, Chapter 5, this volume) and in most cases the development of virus disease resistance seems the most powerful strategy for effective management of potyviruses in legumes.

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DISEASES OF GROUNDNUT

2

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INTRODUCTION

The cultivated groundnut or peanut, *Arachis hypogea*, is an annual oilseed legume native to South America but now grown in diverse environments in six continents between latitudes 40°N and 40°S. The genus *Arachis* contains 70 or more diploid and tetraploid species of which *A. glabrata*, *A. hypogea*, *A. pintoi*, *A. repens* and *A. villosulicarpa* are cultivated. *Arachis* is placed with the related genera *Aeschynomene*, *Arthrocarpum*, *Chapmannia*, *Pachecoa*, *Stylosanthes* and *Zornia* in the subtribe *Stylosanthinae* of the tribe *Aeschynomeneae*.

World groundnut production in the 1980s averaged 19.8 Mt from approximately 18.3 million hectares (Nigam *et al.*, 1991). Of over 100 countries with significant production of groundnut, the most important are India (33.4%), China (27.8%), USA (9.3%), Senegal (4.2%), Indonesia (4.2%), Nigeria (3.3%), Myanmar (3.0%), Sudan (2.7%) and Argentina (2%). Groundnut seed contains around 50% high quality edible oil and 25% protein. Groundnut is consumed as whole seed or processed as traditional dishes or snack foods. The oil may be extracted and used for cooking, and the residual cake used in production of food or, more commonly, in animal feeds. The haulms are used as hay for feeding livestock, and this is particularly important to resource-poor farmers in the arid and semi-arid tropics. After decortication, the shells may be burnt as fuel, or used for production of particle board. They may also be ground and used as a filler in animal feeds (Nigam *et al.*, 1991).

About 80% of the world's production of groundnut is grown by resource-poor, small farmers in developing countries who obtain low yields of 500–800 kg ha^{-1}. This compares poorly with yields of over 2.5 t ha^{-1} in developed countries, and with potential yields of over 10 t ha^{-1}. Poor yields are due in many cases to diseases.

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However, it should also be noted that some abiotic stresses can cause symptoms that mimic those caused by pathogens, and that biotic and abiotic stresses may interact with the host plant and environment to produce complex disease situations and significant losses.

There is extensive literature on groundnut diseases and the reader can access much of this through the reviews by Garren and Wilson (1951), Feakin (1973), Garren and Jackson (1973), McDonald and Raheja (1980), Porter *et al.* (1982, 1984), Middleton *et al.* (1994) and NRI (1996). The monograph by Jackson and Bell (1969) on diseases of groundnut caused by fungi continues to be cited because of its comprehensive coverage of the subject. For identification of groundnut diseases, we recommend the *Compendium of Peanut Diseases* published by the American Phytopathological Society (Porter *et al.*, 1984) which provides descriptions and illustrations of nearly all common diseases of the crop. A revised edition has recently been published (Kokalis Burelle *et al.*, 1996). Illustrated handbooks to assist field workers in disease diagnosis have been published in several countries and the International Crops Research Institute for the Semi-Arid Tropics (ICRISAT) has produced one in several languages that gives a comprehensive coverage of groundnut diseases worldwide (Subrahmanyam *et al.*, 1992). Computer-based 'Expert Systems' are being developed to assist with disease diagnosis and to provide advice on management.

Diseases are caused by numerous fungi, bacteria, a phytoplasma, and more than 20 viruses; at least 100 species of nematodes attack groundnut (Sharma and McDonald, 1990; Subrahmanyam *et al.*, 1990a; Reddy, 1991). A few angiosperms are also capable of parasitizing groundnut but their incidence is high only in Malawi and Burkina Faso. Fourteen diseases have been distinguished as of global importance and are featured in this chapter under aetiology, biology, symptoms, epidemiology, effects on yield and quality, and management. Fungal diseases of local or minor importance are listed in Table 2.1 according to disease type. Similarly, diseases of local or minor importance caused by viruses and nematodes are summarized in Tables 2.2 and 2.3.

ASPERGILLUS CROWN ROT

Aetiology

Aspergillus crown rot was first reported on groundnut in 1926 in Java (Jochems, 1926). Gibson (1953a, b) contributed greatly to knowledge of the aetiology of crown rot in East Africa. Although early reports suspected that two separate species of *Aspergillus* – *A. niger* van Tieghem and *A. pulverulentus* (McAlp.) Thom – were responsible for crown rot (Jackson and Bell, 1969), Porter *et al.* (1984) suggest that the latter species is a mutant of the former. The accepted causal agent is *A. niger* and further taxonomic details can be found in Raper and Fennell (1965).

Table 2.1. Fungal diseases of groundnut of local or minor importance.

Disease	Causal fungi	Distribution	Importance	References
Seed and seedling diseases				
Pre-emergence rots	*Aspergillus pulverulentus* (McAlp.) Thom *Fusarium oxysporum* Schlecht. emend Snyder & Hans. *Fusarium solani* (Mart.) Saccardo *Macrophomina phaseolina* (Tassi) Goid. *Penicillium caryophyllum* Dierckx. *Penicillium citrinum* Thom *Penicillium funiculosum* Thom *Penicillium meliagrinum* Biourge *Pythium aphanidermatum* Hesse *Pythium debaryanum* Hesse *Pythium irregulare* Buisman *Pythium myriotylum* Drechsler *Rhizoctonia solani* Kühn *Rhizopus arrhizus* Fischer *Rhizopus oryzae* Went & Gerlings *Rhizopus stolonifer* (Ehrenberg ex Fries) Vuillemin	Widespread	May reduce seedling stand substantially; yield loss rarely exceeds 1%; pathogens may be associated with *Aspergillus niger*, *A. flavus* and *A. parasiticus* to cause severe yield losses	Coleman (1916); Middleton (1943); Jackson and Bell (1969)
Post-emergence rots	*Fusarium oxysporum* *Fusarium solani* *Pythium aphanidermatum* *Pythium debaryanum* *Pythium irregulare* *Pythium myriotylum* *Rhizoctonia solani*	Widespread	Seedling losses rarely exceed 2%	Jackson and Bell (1969); Singh and Chohan (1974); Elad *et al.* (1979)

Continued overleaf

Table 2.1. *Continued*

Disease	Causal fungi	Distribution	Importance	References
Wilts				
Fusarium wilt	*Fusarium oxysporum* *Fusarium solani*	Widespread	Minor	Rothwell (1962); Jackson and Bell (1969)
Pythium wilt	*Pythium myriotylum*	West Africa and North America	Sporadic, local and minor	Perry (1967)
Verticillium wilt	*Verticillium albo-atrum* Reinke & Berthier *Verticillium dahliae* Kleb.	Australia, North America, South America	Local importance	Jackson and Bell (1969); Middleton *et al.* (1994)
Stem rots and root rots				
Diplodia collar rot and root rot	*Lasiodiplodia theobromae* (Pat) Griffon & Maubl.	Widespread	Sporadic and minor importance	Higgins (1963); Jackson and Bell (1969); Porter *et al.* (1982); Porter *et al.* (1984)
Charcoal rot	*Macrophomina phaseolina*	Widespread	Minor	Jackson and Bell (1969)
Texas root rot (Phymatotrichum root rot)	*Phymatotrichum omnivorum* (Shear) Duggar	North America	Causes severe losses in some locations	Porter *et al.* (1984)
Rhizoctonia root rot	*Rhizoctonia solani*	Widespread	Minor	Jackson and Bell (1969); Porter *et al.* (1984)
Fusarium root rot	*Fusarium oxysporum* *Fusarium solani*	Widespread	Minor	Jackson and Bell (1969)
Olpidium root rot	*Olpidium brassicae* (Woronin) Dang	South Asia	Sporadic, local and minor	Subrahmanyam and McDonald (1980)
Cylindrocladium black rot	*Cylindrocladium crotalariae* (Loss) Bell & Sobers (perfect stage *Calonectria crotalariae* (Loss) Bell & Sobers)	Australia, India, Japan, USA	Locally important; up to 50% crop losses in USA	Bell and Sobers (1966); Porter *et al.* (1984)

Pod rots				
Blackhull	*Thielaviopsis basicola* (Berk. & Broome) Ferraris	North America, South America, South Africa, Europe	Important in some locations only	Hsi (1965); Jackson and Bell (1969)
Pre-harvest pod rots caused by stem and root rot fungi	*Botrytis cinerea* Pers. *Fusarium oxysporum* *Fusarium solani* *Lasiodiplodia theobromae* *Macrophomina phaseolina* *Pythium myriotylum* *Rhizoctonia solani* *Sclerotinia minor* Jagger *Sclerotinia sclerotiorum* (Lib.) De Bary	All widespread	Generally of minor importance, but singly or in various combinations can cause serious damage of a sporadic and local nature	Bouriquet and Jaubert (1954); Ashworth *et al.* (1961); Garren (1966); Bouhot (1967); Jackson and Bell (1969); Rawson *et al.* (1972); Frank (1974); Ibrahim *et al.* (1977); Porter *et al.* (1984)
Foliar diseases				
Alternaria leaf blight	*Alternaria alternata* (Fries) Keissler *Alternaria arachidis* Kulk.	South Asia, East Asia, West Africa	Minor	Frezzi (1960); Subrahmanyam *et al.* (1981, 1992)
Ascochyta leaf blight	*Ascochyta arachidis* Woronichin	Asia	No importance	Woronichin (1924)
Botrytis blight,	*Botrytis cinerea*	Widespread	Minor	Marchionatto (1922)
Sclerotinia blight	*Sclerotinia minor* *Sclerotinia sclerotiorum*	Australasia East Asia, Africa, North America, South America	Important in some locations only	Jackson and Bell (1969); Abawi and Grogan (1979); Porter (1980); Porter *et al.* (1984)
Choanephora wet blight	*Choanephora cucurbitarum* (Berk & Revenel) Thaxt.	East Asia, East Africa	No importance	Van Hall (1924); Mukibi (1975)

Continued overleaf

Table 2.1. *Continued*

Disease	Causal fungi	Distribution	Importance	References
Anthracnose	*Colletotrichum arachidis* Sawada *Colletotrichum dematium* (Pers) Grove *Colletotrichum mangenoti* Chevaugeon	Africa, Asia North America	Minor	Chevaugeon (1952); Sawada (1959)
Zonate leaf spot	*Cristulariella moricola* (Hino) Redhead	South Asia, East Asia, North America	No importance	Smith (1984); Subrahmanyam *et al.* (1992)
Pepper spot and leaf scorch	*Leptosphaerulina crassiasca* (Sechet) Jackson & Bell	Widespread	Minor	McGill and Samples (1965); Jackson and Bell (1968,1969)
Myrothecium leaf blight	*Myrothecium roridum* Tode ex Fries	South Asia, East Asia	No importance	Subrahmanyam (1979)
Pestalotiopsis leaf blight	*Pestalotiopsis arachidis* Satya	South Asia, East Asia, West Africa	Minor	Satya (1964); Subrahmanyam *et al.* (1992)
Phoma blight	*Phoma microspora* Balasubramanian & Narayanaswamy	South Asia (India)	Can cause some damage in its restricted locations	Balasubramanian and Narayanaswamy (1980)
Phomopsis leaf scorch	*Phomopsis sojae* Lehman	South America South Asia	Minor, but found together with early and late leaf spots	Frezzi (1965); Garren and Jackson (1973); Sharma (1974)
Phyllosticta leaf spot	*Phyllosticta arachidis-hypogaea* Vasant Rao	Widespread	Minor	Rao (1963); Jackson and Bell (1969)
Scab	*Sphaceloma arachidis* Bitancourt & Jenkins	South America, East Asia (Japan)	Of local importance in areas of South America	Jackson and Bell (1969); Porter *et al.* (1984)
Melanosis	*Stemphylium botryosum* Wallr.	South America (Argentina)	Of minor importance locally	Frezzi (1960)
Powdery mildew	*Oidium arachidis* Chorin	Widespread in subtropics and temperate climates	Minor	Chorin (1961); Hirata (1966)

Table 2.2. Virus diseases of groundnut of local and minor importance.

Disease	Causal virus	Distribution	Importance	References
Groundnut leaf roll	Cowpea mild mottle carlavirus	China, India, Indonesia, Ivory Coast, Nigeria, Thailand, Philippines, Papua New Guinea, Sudan	Can cause severe yield reduction. Potential to become a threat	Iizuka *et al.* (1984)
Peanut chlorotic streak	Peanut chlorotic streak caulimovirus	India	Minor	Reddy *et al.* (1993)
Groundnut yellow mosaic	Bean golden mosaic geminivirus	India	Minor	Reddy and Demski (1996)
Not named	Cucumber mosaic cucumovirus	China	Extremely important in China. Virus is seed-transmitted	Xu and Barnett (1984)
Peanut stunt	Peanut stunt cucumovirus	Sudan, Japan, Spain, USA	In early 1960s very important in the USA. Currently minor	Tolin (1984)
Not named	Tobacco streak ilarvirus	Brazil	Not known	Reddy and Demski (1996)
Groundnut streak necrosis	Sunflower yellow blotch luteovirus	Malawi, Kenya, Zambia, Tanzania	Potential to become important	Bock (1989)
Groundnut eyespot	Groundnut eyespot potyvirus	Ivory Coast, Burkina Faso, Mali	Minor	Dubern and Dollet (1980)
Peanut green mosaic	Peanut green mosaic potyvirus	India	Minor	Sreenivasulu *et al.* (1981)
Peanut yellow spot	Peanut yellow spot tospovirus	India, Thailand, Myanmar	Potential to become economically important	Reddy *et al.* (1991)
Groundnut yellow mottle	Groundnut yellow mottle tymovirus	Nigeria	Minor	Reddy and Demski (1996)
Groundnut veinal chlorosis	Groundnut veinal chlorosis rhabdovirus	India, Indonesia	Potential to cause severe yield losses	Naidu *et al.* (1989)

Table 2.3. Nematode diseases of local or minor importance.

Disease	Causal nematode	Distribution	Importance
Kalahasti malady	*Tylenchorhynchus brevilineatus* Williams	India	Localized
Peanut chlorosis	*Aphasmatylenchus straturatus* Germani	Burkina Faso	Localized
Peanut rot	*Ditylenchus africanus* Wendt, Swart, Vrain, Webster	South Africa	Localized
Root lesion	*Pratylenchus brachyurus* (Godfrey) Filipjev & Sch. Stekh.	Widespread	Localized
Testa discoloration	*Aphelenchoides arachidis* Bos	Nigeria	Localized
Peanut yellows	*Criconemoides ornata* (Raski) Luc & Raski	India, USA	Minor
Crop growth variability	*Scutellonema cavenessi* Sher.; *S. clathricaudatum* Whitehead	Niger, Senegal	Localized
Crop growth variability	*Paralongidorus bullatus* Sharma & Siddiqi	Niger	Localized

Sources: Bridge *et al.* (1977); Minton and Baujard (1990); De Waele *et al.* (1988); Sharma and McDonald (1992).

Biology

Aspergillus niger causes rot of seed and germinating seedlings and post-emergence crown rot (Jackson and Bell, 1969; Porter *et al.*, 1984). It is a ubiquitous and very efficient saprophyte which can parasitize groundnut and other crops. It probably occurs in all groundnut growing regions of the world. There are morphological and physiological variants but little is known about pathogenic variability. Colonies on malt agar produce a loose, white to yellowish mycelium which rapidly becomes black to dark brown with the development of conidia (Onions, 1966a). The fungus typically has large black conidial heads that are globose to radiate, in columns 700 to 800 μm in diameter; conidiophores variable, 1.5–3.0 mm × 15–20 μm, smooth-walled, colourless to brown. Vesicles are 45–75 μm in diameter, at times smaller or up to 80 μm; sterigmata in two series: the primary 20–30 × 5–6 μm; when young reaching 60–70 × 8–10 μm at maturity; the secondary more uniform, ranging from 7–10 × 3.0–3.5 μm; conidia globose, 4–5 μm in diameter and irregularly roughened (Raper and Fennell, 1965; Onions, 1966a). The optimum growth temperature range is 30–40°C and the fungus persists and grows on a range of substrates (Jackson and Bell, 1969).

Symptoms

Seed may be attacked as soon as it is sown in moist, infested soil (Jackson and Bell, 1969). Seedlings are also very susceptible (Porter *et al.*, 1984). Rotted seed is reduced to a spongy mass of disintegrating tissue often covered with black or brown masses of sporulating mycelium. Emerging seedlings show rapid wilting especially during dry weather, become desiccated and the cotyledons and growing points are covered by sporulating mycelium giving them a black powdery appearance. As the disease progresses the collar region becomes shredded and discoloured and the affected seedling usually dies (Gibson, 1953b; Jackson and Bell, 1969). In mature plants, death may occur due to previously established infections. Because of the woodiness of mature plants, symptoms may not be noted until permanent wilting of branches or the entire plant is apparent. Disease incidence in mature plants is much less than in seedlings.

Epidemiology

Growth and sporulation of the fungus are favoured by warm, moist soil conditions and disease can be particularly damaging in the warm tropics (Porter *et al.*, 1984). The fungus can tolerate low soil moisture (Coleman, 1916), and is more tolerant of mercury than most fungi associated with groundnut seed (Gibson, 1953b). The disease is often more prevalent in sandy soils low in organic matter (Porter *et al.*, 1984). As a saprophyte, *A. niger* is found abundantly in soil and plant debris and these agents are probably the most important sources of primary infection. The infection process takes around 10 days (Jackson and Bell, 1969). The disease may also develop from mycelium already established in the seed. Infestation of seed lots can exceed 90% (Porter *et al.*, 1984). Plants growing from such seed are usually highly infected. The fungus can invade shells and seed as pods mature in the soil, as the harvest is dried in windrows, or as it is held in storage (Jackson, 1967). This establishes a cycle when infected seed is sown.

Effects on Yield and Quality

According to Porter *et al.* (1984), stand losses in individual fields can reach 50% but are more usually less than 1%. Infected plants may survive and produce pods, but the damaged plants may be more liable to end of season drought damage and their seeds may be invaded by various soil fungi with resulting loss of viability and free fatty acid content as well as contamination with mycotoxins.

Management

Several seed protectant chemicals are recommended for use against *A. niger* and other soil fungi infecting groundnut (Porter *et al.*, 1984). Those based on thiram or captan are usually effective but mercury-containing substances should not be

used (Jackson and Bell, 1969). It is important to sow undamaged, healthy seed, and to rotate groundnut with cereal. Sowing time should be adjusted to ensure ample soil moisture for rapid germination and seedling growth. All commonly grown cultivars can be affected by crown rot under conducive environmental conditions. Bunch types are usually less susceptible than runner types (Porter *et al.*, 1984). Some of the cultivars being bred for field resistance to pod invasion by the aflatoxigenic *Aspergillus flavus* also have resistance to invasion by *A. niger* and other soil fungi, and could be utilized in areas where crown rot is a serious and recurring problem.

YELLOW MOULD/AFLAROOT

Aetiology

In a detailed taxonomic account of the genus, Raper and Fennell (1965) noted that one synonym and 12 possible synonyms exist for *Aspergillus flavus* Link ex Fries. This species is the most common of a very large group of related fungi which tend to show similar behaviour, are frequently observed on groundnut and cause similar diseases (Onions, 1966b; Ahmed and Reddy, 1993). *A. flavus* may be distinguished from *A. flavus* var. *columnaris* Raper and Fennell, which has phialides only and conidia borne consistently in columns, and from *A. parasiticus* Spear, which has deeper green colonies, phialides only and smaller conidia (Onions, 1966b).

Biology

A. flavus is reported most commonly as a pod and seed inhabitant (Jackson and Bell, 1969). Relatively few reports associate the fungus with older plants. *A. flavus* causes diseases of seed and seedlings very similar to those incited by *A. niger*. The term yellow mould relates to the colour of the sporulating fungus. *A. flavus* and *A. parasiticus* commonly occur together as causal agents of yellow mould (Porter *et al.*, 1984). In the 1960s, mycotoxins produced by *A. flavus* were found in groundnut meal. Feeds prepared from this meal were responsible for the death of 100,000 turkeys in the UK (Middleton *et al.*, 1994). Four mycotoxins occur naturally in groundnut seed: aflatoxin, citrinin, ochratoxin A and zearalenone; of these aflatoxins are the most important and widespread, being toxic in small amounts (Porter *et al.*, 1984; Middleton *et al.*, 1994). Yellow mould and aflatoxin contamination can occur in seedlings, near harvest on pods and seed in the soil, and during harvest and postharvest on seed in storage (Jackson and Bell, 1969; Porter *et al.*, 1984). Aflatoxin contamination has had a tremendous impact on the global groundnut industry and on consumers. This has been reviewed in detail by Mehan *et al.* (1991).

Colonies are usually spreading, yellow-green, occasionally dominated by hard sclerotia (Onions, 1966b). The young conidial heads are yellowish but quickly shade through bright to dark yellow-green, jade green or cress green

colours (Jackson and Bell, 1969). Sclerotia are produced by many strains, starting as white mycelial tufts, changing to dark red-brown to near black, globose to subglobose, 400–700 μm in diameter (Onions, 1966b). Conidial heads radiate, splitting into several poorly defined columns, commonly 300–400 μm in diameter. Conidiophores are thick-walled, hyaline coarsely roughened, usually less than 1 mm long (occasionally 2–2.5 mm), with stalk diameters immediately below vesicles 10–20 μm. Vesicles elongate, becoming subglobose or globose, 10–65 μm in diameter, mostly 25–45 μm. Sterigmata are uniserate or biserate; primaries 6–10 × 4–5.5 μm (sometimes 15–16 × 8–9 μm); secondaries 6.5–10 × 3–5 μm. Conidia are globose to subglobose, echinulate, 3–6 μm in diameter, mostly 3.5–4.5 μm; sometimes elliptical, 4.5–5.5 × 3.5–4.5 μm (Onions, 1966b).

A. flavus thrives as a soil parasite in the tropics, subtropics and warm temperate regions (Onions, 1966b). Yellow mould and aflatoxin contamination of groundnut seed occur throughout the world and they also affect seed of other crop plants including legumes such as soyabean, bean and pea (Onions, 1966b; Jackson and Bell, 1969). Isolates of *A. flavus* and the closely related *A. parasiticus* vary in their ability to produce aflatoxins, but little is known of their pathogenic variability.

Symptoms

In pre-emergence attack, seed and seedlings are reduced to shrivelled, dried brown or black masses covered with yellow or greenish-yellow spores (Fig. 2.1) (Ahmed and Reddy, 1993). When infected seedlings do emerge their cotyledons have necrotic lesions with reddish-brown margins and are covered with sporulating mycelium (Porter *et al.*, 1984). This is very similar to *A. niger* crown rot and, in some cases, both fungi may be present as mixtures of yellowish-green and black spore masses covering the diseased cotyledons and growing point. When the strain involved is aflatoxigenic, the seedling may be severely stunted with chlorotic leaves and vein-clearing of leaflets that are smaller than usual and have pointed tips (Chohan and Gupta, 1968). Roots are necrotic with few or no secondary roots. This condition is known as 'aflaroot disease' (Chohan and Gupta, 1968; Porter *et al.*, 1984). Affected seedlings sometimes die. Yellow mould of pods and seed appears on or in pods before harvest, especially when plants are stressed by drought (Porter *et al.*, 1984). Yellow-green colonies of *A. flavus* may develop on seed and pods that are overmature or damaged. During and following harvest, additional pods may be contaminated.

Epidemiology

The disease is favoured by warm, dry soil conditions and it can be particularly damaging in the warm arid and semi-arid regions of the tropics (Jackson and Bell, 1969). *A. flavus* grows between 17 and 42°C and in a declining soil moisture regime, providing relative humidity is maintained between 85 and 95%,

Fig. 2.1. Seed of groundnut covered with masses of greenish-yellow spores of *Aspergillus flavus* (Photo: courtesy of ICRISAT).

while the optimum temperature for aflatoxin production is 25–35°C (Pettit *et al.*, 1971; Porter *et al.*, 1984). Mehan *et al.* (1988) showed a significant, positive linear relationship between water deficit (drought intensity) and seed infection in groundnut genotypes. Like *A. niger*, it is tolerant of mercury, low soil moisture and high temperature. The epidemiology of *A. flavus* has been reviewed in detail by Jackson and Bell (1969) and Porter *et al.* (1984), and is similar to that of *A. niger*. The literature on aflatoxin contamination of groundnut, including the relationship of *A. flavus* with other fungi present in groundnut field soils, in the rhizosphere and geocarposphere, and in shell and seed mycofloras, has been reviewed by Mehan *et al.* (1991).

Being a soil inhabitant, *A. flavus* can survive in soil and crop residues (Porter *et al.*, 1984). Its ability to cause disease is related to its ability to compete with other soil microflora, the availability of susceptible plant tissues, and the occurrence of favourable environmental conditions. Aflatoxin contamination of seed is influenced by the aflatoxin producing ability of the particular isolate of *A. flavus*, composition of the substrate, environmental conditions and harvesting, handling and storage practices (Mehan *et al.*, 1991).

Effects on Yield and Quality

Direct damage to groundnut by seed and seedling diseases is probably less than that caused by *A. niger* but pre- and/or postharvest contamination of seed with

aflatoxins creates a serious quality problem of international concern. Aflatoxin detection systems have been set up in many developed countries. In particular in the USA, the Food and Drug Administration has set levels of aflatoxin for all edible products and a detection system is in place to prevent groundnut seed contaminated with aflatoxins from being used for human and animal foods (Porter *et al.*, 1984; Middleton *et al.*, 1994). Chemical detection of aflatoxin is carried out by various chromatographic procedures (Coker and Jones, 1988; Mehan *et al.*, 1991); however, increasingly, more cost-effective serological methods based on monoclonal antibodies to aflatoxin B_1 are being used successfully (Cole *et al.*, 1988; Mehan *et al.*, 1991).

Management

Control of yellow mould and management of aflatoxin contamination in groundnut seed is best achieved by preventing *A. flavus* from infecting growing plants and by destroying contaminated seed (Porter *et al.*, 1984). Recommendations include: avoiding mechanical damage to the crop during cultivation, harvesting and processing; harvesting at the optimum time and rapid postharvest drying; removal of damaged and mouldy pods; drying to a safe moisture level (8%) before storage; and storage under conditions of low temperature and humidity (Middleton *et al.*, 1994). Use of appropriate seed protectants (Subrahmanyam, 1991) and the cultural practices recommended above for control of *A. niger* should also be helpful in managing yellow mould. Such measures have been applied with success in developed countries but have been largely neglected in developing countries (Mehan and McDonald, 1983). Thus aflatoxin contamination of groundnut continues to be a major problem and health risk in developing countries, especially in West Africa (Waliyar *et al.*, 1994a).

Groundnut genotypes resistant to seed infection by aflatoxin-producing strains of *A. flavus* that do not support aflatoxin production are being sought (Mehan *et al.*, 1991). Several lines have been identified which resist seed colonization and reduce capacity to produce aflatoxin B_1 (Mehan *et al.*, 1986; Wynne *et al.*, 1991; Waliyar *et al.*, 1994a; Rao *et al.*, 1995). Varieties are being developed that have resistance to both *A. flavus* infection and to aflatoxin contamination (Mehan *et al.*, 1991; Wynne *et al.*, 1991; Rao *et al.*, 1995). As resistance may operate at the pod surface, within the shell, at the seed surface and within the testa and/or cotyledon, different screening methodologies are needed. There appear to be different genes conferring resistance to seed colonization, postharvest infection and aflatoxin production (Utomo *et al.*, 1990; Wynne *et al.*, 1991; H.D. Upadhyay, ICRISAT, India, 1996, personal communication). As very high levels of resistance to aflatoxin production have not been found, resistant varieties should be considered as part of an integrated aflatoxin management programme incorporating cultural control strategies and crop handling practices appropriate to the particular environment (Mehan *et al.*, 1991). Current research is examining the possibility of replacing natural populations of aflatoxigenic strains of *A. flavus* with highly competitive, non-toxigenic strains (Cotty,

1990), but it would be essential to ensure that these are no more pathogenic to groundnut than the strains they replace.

STEM, ROOT AND POD ROT

Aetiology

The name *Sclerotium rolfsii* Sacc. was given by Saccardo (1911) who characterized the fungus as an imperfect form without sexual spores and belonging to this heterogeneous form-genus. The taxonomy has been reviewed by Aycock (1966). The basidial stage was first known from culture only and was given the name *Corticium rolfsii* (Sacc.) Curzi which was later changed to *Pellicularia rolfsii* (Sacc.) West (West, 1947). As the basidial stage is rarely found in nature, it is common practice to use the name of the sclerotial stage (Mehan *et al.*, 1995).

Biology

S. rolfsii causes stem, root, peg and pod rot of groundnut which are major constraints to production in many countries (Jackson and Bell, 1969; Porter *et al.*, 1984; Mehan *et al.*, 1995). Stem rot is also known as southern blight in the southern USA. It is mainly a problem of older plants but can also attack seedlings. Stem, root and pod rot is an increasing threat to groundnut production under irrigation (Mehan *et al.*, 1995).

The sclerotial stage is characterized by septate, hyaline mycelium with conspicuous branching at acute angles (Aycock, 1966). The well-developed mycelium is in cord-like strands of hyphae with clamps in the form of hooks and forks. The young mycelial mass is snow-white with a silky lustre. The developing mycelium grows in strands and sclerotial formation occurs at its tips in 6–12 days. At first white, the sclerotia turn light to dark brown at maturity. They are subspherical, sometimes flattened, with surfaces finely wrinkled or pitted, and usually measure 0.5–1.5 mm in diameter (Aycock, 1966). The hymenium of the basidial stage is at first coarsely areolate, becoming more dense with the formation of basidia but never forming a continuous fleshy layer, 30–40 μm thick, white to grey in colour (West, 1947). Basidia are ovoid, 7–9 × 4–5 μm, each bearing two to four parallel or divergent sterigmata that bear basidiospores which are hyaline, smooth, elliptical to obovate, rounded above, rounded to pointed at the base, apiculate, 3.5–5 × 6–7 μm (West, 1947).

S. rolfsii grows well in culture on a wide range of media, over a wide range of conditions (Mehan *et al.*, 1995). The optimum temperature range for growth is 27–30°C. In general, media which support mycelial growth also support sclerotial production. Considerable variability in morphological characteristics among isolates has been documented (Punja, 1985), and recent studies (Shokes *et al.* unpublished results, reported in Mehan *et al.*, 1995) have shown that isolates may vary considerably in their ability to infect groundnut genotypes. *S. rolfsii* has a very large host range including many legumes (see Bayaa and Erskine, Chapter

8, and Haware, Chapter 9, this volume) and it is found wherever groundnut is grown. All aspects of the pathogen and its interactions with groundnut have been reviewed in detail (Jackson and Bell, 1969; Porter *et al.*, 1984; Mehan *et al.*, 1995).

Symptoms

Early symptoms of stem rot are yellowing and wilting of branches near the base of the plant (Jackson and Bell, 1969; Porter *et al.*, 1984; Mehan *et al.*, 1995). Sheaths of mycelium develop around the affected areas close to the soil surface (Fig. 2.2), and the stems become shredded. Abundant formation of sclerotia occurs over infected parts of the plant and this is often diagnostic. Individual branches or the entire plant may be killed. Infected pegs first show light to dark brown lesions, but with advance of the disease become necrotic and shredded and pods are detached and left in the soil at harvest. Lesions on pods are light tan in colour but severely damaged pods are covered with a white mycelial mat. Seeds from infected pods may show 'blue damage', a characteristic bluish-grey discoloration caused by oxalic acid produced by *S. rolfsii*. The fungus can also cause leaf spots although these are not common. The grey necrotic ring spots usually develop shot holes. Under very wet conditions the lesions coalesce to cause severe blight. Minute sclerotia appear on both leaf surfaces. Jackson and Bell (1969) cover symptomatology in detail.

Fig. 2.2. Sheaths of white mycelium of *Sclerotium rolfsii* on groundnut stems (Photo: courtesy of V.K. Mehan).

Epidemiology

Environmental factors are important in determining the form the disease will take whether stem, root or pod rot. Warm (25–35°C), moist conditions favour disease development (Aycock, 1966; Rodriguez-Kabana *et al.*, 1975). Factors which tend to increase or prolong soil moisture favour stem rot while dry periods favour severe root and pod rot (Mehan *et al.*, 1995). In the USA, stem rot is most prevalent in sandy soils (Porter *et al.*, 1984) whereas in India it is more common in vertisols (Mayee and Datar, 1988). Sclerotia of *S. rolfsii* remain viable in the soil for 2–3 years and serve as the primary source of inoculum; they can initiate infection without need for an additional food base (Aycock 1966; Mehan *et al.*, 1995). High temperatures and high soil moisture reduce survival of sclerotia, as do cycles of drying and wetting which stimulate nutrient leakage and microbial antagonism. The fungus readily colonizes organic substrates in the soil, and inoculum potential and disease severity are positively correlated with the food base.

Effects on Yield and Quality

Stem, root and pod rot caused by *S. rolfsii* results in serious losses in yield of groundnut in many parts of the world (Mehan *et al.*, 1995). In the south-eastern USA, losses range from 10 to 25% (Porter *et al.*, 1984), and similar levels have been noted in India (Mayee and Datar, 1988). Discoloration of seeds from infested pods (blue damage) render them unacceptable for confectionery use.

Management

Mehan *et al.* (1995) reviewed the considerable amount of research which has been done on cultural control measures (crop sanitation, rotation, moisture control, soil amendments, soil solarization), biological control organisms, chemical control products (chemicals or mixtures of chemicals) and resistant varieties for management of stem and pod rot and provide a useful bibliography. They note that although little is known as to how different cropping systems affect soil microorganisms, especially the survival of *S. rolfsii*, there is scope for using microorganisms as biocontrol agents to manage *S. rolfsii* populations which can be linked to other management practices of stem and pod rots.

In recent years, progress has been made in the development of screening techniques for identifying sources of resistance (Shew *et al.*, 1987; Shokes *et al.*, 1994). Although no genotypes have been identified which are immune or even highly resistant to *S. rolfsii*, several genotypes and breeding lines have shown field resistance (Smith *et al.*, 1989; Grichar and Smith, 1992; Branch and Brenneman, 1993). Mehan *et al.* (1995) list 17 genotypes of groundnut resistant or partially resistant to stem and pod rot. Resistance is thought to be due to thick cuticle, thick-walled cortical cells and cork cambium activity (Cooper, 1961). Many resistant lines have non-succulent stems and smooth, hard shells (Mehan *et al.*, 1995). Further studies on this topic are needed.

An integrated management approach is recommended for stem, root and pod rot (Mehan *et al.*, 1995). Partially resistant cultivars could be grown in rotation with crops highly resistant to *S. rolfsii*. Other appropriate cultural controls such as deep ploughing could be incorporated and biological control could be part of the systems. Clearly, development of integrated management packages suitable for specific farming systems and environments should be given some attention.

RUST

Aetiology

Groundnut rust was first recorded from Surinam as *Uredo arachidis* Lagerheim (Hennen *et al.*, 1987). The second record was from Paraguay, named *Puccinia arachidis* by Spegazzini in 1884. Debate regarding the correct name followed until Hennen compared both collections with a new collection from Brazil and proposed that the latter name be accepted as the teleomorphic and holomorphic binomial for the groundnut rust fungus (Hennen *et al.*, 1976). The taxonomy of the fungus has been summarized by Subrahmanyam and McDonald (1983) and is discussed in some detail by Hennen *et al.* (1987) and Hennen and Buritica (1993).

The name universally accepted is *Puccinia arachidis* Speg. but the fungus is not regarded as a *Puccinia* by Hennen and Buritica (1993). On cultivated groundnut, a conidial anamorph occurs widely but the teleomorph has been found only rarely, most commonly on wild *Arachis* spp. in Brazil. Hennen and Buritica (1993) proposed the name *Peridipes arachidis* as the conidial anamorph of groundnut rust due to possession of a membranous peridium and pedicillate spores. *Puccinia arachidis* is retained as the holomorph. There is no knowledge of spermogonia, aecia and hosts that basidiospores will infect, thus the taxonomic position of this fungus is incomplete (Subrahmanyam and McDonald, 1983). The uredial stage is the predominant form globally.

Cummins (1978), Hennen *et al.* (1987) and Lenné (1994a, b) have commented on the relationship between *P. arachidis* and apparently closely related rusts on the legume genera *Zornia* and *Stylosanthes* (see Chapter 13, this volume). These genera together with *Arachis* are in the same subtribe of *Stylosanthinae* and they frequently occur together in natural vegetation in South America, especially Brazil (Lenné, 1994b). The rusts are *P. offuscata* Arthur and *P. zorniae* McAlpine on *Zornia* spp. and *P. stylosanthis* Viegas on *Stylosanthes* spp. Sutton (1984 – cited in Lenné, 1994b) considered that these rusts form part of a closely graded series, with relatively small differences in uredospore morphology (see Lenné, Chapter 13, this volume). J.F. Hennen, Texas, USA (1996, personal communication) also believes that the four rusts are very closely related. He noted, as did Lenné (1994b), that cross-inoculation studies are needed to elucidate the relationship between these taxa and the taxonomic position of *P. arachidis*.

Biology

Rust of groundnut probably originated in South America with cultivated groundnut and, until the late 1960s, its distribution was limited (Subrahmanyam *et al.*, 1985a). During the past 30 years, it has spread rapidly throughout most important groundnut growing areas (Subrahmanyan and McDonald, 1983; Porter *et al.*, 1984; Subrahmanyam *et al.*, 1985a). Together with late and early leaf spots with which it often occurs, it is considered to be among the most important diseases of groundnut.

Uredinial sori are pustular, predominantly hypophyllous, scattered or irregularly grouped, round ellipsoid or oblong in shape, dark cinnamon brown in colour when mature; mostly on abaxial leaf surfaces where the ruptured epidermis is conspicuous but may also occur on petioles, stipules and stems (Cummins, 1978; Hennen *et al.*, 1987). Uredospores are broadly ellipsoid or obovoid, 16–24 × 21–30 µm in size, the wall brown in colour, 1–2 µm thick, finely echinulate, with mostly two, occasionally three to four germpores, nearly equatorial, often in flattened areas. Telia are rarely observed, chiefly hypophyllous, 0.2–0.3 mm in diameter, scattered, prominent, pulvinate, chestnut brown, ruptured epidermis prominent; teliospores are oblong, obovate or ellipsoid, with rounded to acute and thickened apex, slightly or not constricted at the septum, predominantly two-celled, 33–60 × 12–18 µm, wall smooth, light to chestnut brown, 0.7–0.8 µm thick at the sides, 2.5–5 µm thick at the top, apical thickening almost hyaline, pedicel thin-walled, hyaline, up to 35–65 µm in length, but usually broken, shorter or detached at the spore base, germinating at maturity without dormancy (Cummins, 1978; Hennen *et al.*, 1987).

The complete life cycle of groundnut rust is at present unknown. The only known hosts are the cultivated groundnut and many related species of *Arachis*, including *A. glabrata*, *A. burkartii*, *A. marginata* (Hennen *et al.*, 1987), *A. cardenasii*, *A. helodes*, *A. stenosperma*, *A. nambyquarae* and *A. hagenbeckii* (J.F. Hennen, Texas, USA, 1996, personal communication), yet these are unlikely to play a role in the disease cycle of rust on groundnut as most occur in natural vegetation and only in South America, distant from major groundnut growing areas (Subrahmanyam and McDonald, 1983). It is generally assumed that rust has inherent capabilities for development of physiological races when confronted with host genotypes possessing major gene resistance (Wynne *et al.*, 1991). There are, however, no confirmed reports of pathotypes, and groundnut cultivars tested in many different parts of the world have shown comparable reactions to the local rust populations (Subrahmanyam and McDonald, 1983, Subrahmanyam *et al.*, 1985a, 1989, 1995; Wynne *et al.*, 1991; NRI, 1996). Rust resistance has remained stable over a wide range of geographical locations.

Symptoms

The characteristic orange-coloured pustules (uredia) appear first on the abaxial surfaces of leaflets and rupture to expose masses of reddish-brown uredospores (Fig. 2.3) (Subrahmanyam and McDonald, 1983; Porter *et al.*, 1984). Pustules

Fig. 2.3. Reddish-brown uredospores of *Puccinia arachidis* on the abaxial surface of groundnut (Photo: courtesy of ICRISAT).

are circular and range from 0.5 to 1.4 mm in diameter. On highly susceptible cultivars, secondary pustules may develop around the earlier ones. Pustules may later appear on the adaxial surfaces of the leaflets opposite those on the lower surfaces on susceptible varieties. Pustules may form on all aerial plant parts with the exception of flowers. Infected leaves tend to become necrotic and dry up but remain attached to the plant. Plants have a burnt appearance.

Epidemiology

Rust perpetuates, spreads, and causes disease by means of its uredospores (Mallaiah and Rao, 1979a, b; Subrahmanyam and McDonald, 1983; Savary *et al.*, 1988). These are short lived, particularly in warm climates, and are unlikely to survive for long between crop seasons in the tropics (Subrahmanyam and McDonald, 1983; Porter *et al.*, 1984). Infected volunteer plants and overlapping crops are important in carry-over of the disease (Mallaiah and Rao, 1979a; Subrahmanyam and McDonald, 1983). Long-distance dissemination of the disease may occur by airborne uredospores, the movement of infected crop debris, or the movement of pods and seeds surface contaminated with uredospores or debris. However, only circumstantial evidence exists for long-distance dispersal (Savary *et al.*, 1988). There is no reliable evidence that rust is internally seedborne and no authenticated report of rust being spread by germplasm exchange (Subrahmanyam and McDonald, 1983).

Temperatures around 20–28°C, high relative humidity, and free water on the leaf surface favour infection and disease development (Mallaiah and Rao, 1979b; Savary *et al.*, 1988). Spread of disease in growing crops is facilitated by rain-splash, wind movement and insects (Subrahmanyam and McDonald, 1983). Aerial dispersal of uredospores showed strong diurnal rhythm related to daily variations in wind velocity (Savary *et al.*, 1988).

Effects on Yield and Quality

Losses due to rust alone are difficult to assess because leaf spots are often present and also contribute to losses. Rust causes serious damage to groundnut crops in many parts of the world with pod losses of up to 70% being reported (Harrison 1973; Subrahmanyam *et al.*, 1984, 1985a). Losses of haulms are also substantial and rust can also affect the quality of fodder. In a study in Nigeria, protein content of haulms was reduced from 16 to 12% due to foliar diseases including rust (Salako and Adu, 1990). The disease can be particularly severe when it occurs in conjunction with early or late leaf spots, or if it affects the crop early (Subrahmanyam and McDonald, 1983).

Management

Although rust can be controlled very effectively by fungicides (Smith and Littrell, 1980; Porter *et al.*, 1984), these are costly and not readily available to resource-poor farmers in developing countries, who also generally lack the technical expertise to use chemicals effectively (Subrahmanyam *et al.*, 1995). Breeding for resistance is therefore a key strategy in reducing yield losses due to rust. In recent years, there have been efforts by many countries to exploit genetic resistance to groundnut rust (Subrahmanyam *et al.*, 1985a, 1995). A world collection of 12,000 accessions of groundnut germplasm from as many as 87 countries has been systematically screened for resistance at ICRISAT, India, and many accessions have also been screened in the USA, Nigeria, Mali, Burkina Faso and Malawi (Subrahmanyam *et al.*, 1982, 1985a, 1995; Subrahmanyam and McDonald, 1983; Waliyar and McDonald, 1988). Over 100 genotypes with good resistance to rust have been found, mainly in valencia-type landraces originating from Tarapoto, Peru (Ramanatha Rao, 1987; Subrahmanyam *et al.*, 1989). Several have been used successfully in breeding programmes (Reddy *et al.*, 1987; Subrahmanyam *et al.*, 1995).

High levels of resistance and, in some cases immunity, have been found in wild *Arachis* species (Subrahmanyam *et al.*, 1983a; Singh and Nigam, 1996), and through hybridization with cultivated groundnut, some rust-resistant genotypes with good agronomic characters have been bred (Singh *et al.*, 1987). Cultivars have been released in different countries for use in rust-affected areas (Hammons *et al.*, 1982a, b, c; Subrahmanyam *et al.*, 1985a, 1995). Resistance presently used by groundnut breeders appears to include factors for 'slow rusting' (polygenic, minor genes) characterized by reduced infection frequency,

increased incubation period, reduced lesion diameter and sporulation index (Subrahmanyam *et al.*, 1983a, b; Wynne *et al.*, 1991; Mehan *et al.*, 1994a). Rust-resistant cultivars should be used together with cultural practices such as eradication of groundkeepers and volunteer plants, and adjustment of sowing and harvest dates to ensure a sufficiently long break between successive groundnut crops and to avoid environmental conditions conducive to disease build-up (Subrahmanyam and McDonald, 1983). Weeds should be controlled, as a heavy growth of weeds may encourage disease development due to higher humidity in the crop canopy.

Under experimental conditions, large yield increases have been obtained through application of fungicides and, in the USA, fungicides are used widely to control rust and leaf spots with as many as six to eight applications throughout the growing season (Smith and Littrell, 1980). Integrated control packages suited to specific production systems and environments, including tropical developing countries, can be developed particularly through combining chemicals with resistance (Subrahmanyam and McDonald, 1983). The choice of fungicide(s) will be influenced by the presence of other foliar diseases and this is discussed in some detail in Smith and Littrell (1980) and Subrahmanyam and McDonald (1983). Good control of rust, leaf spots and web blotch can be obtained with chlorothalonil, while the systemic fungicide calixin is effective against rust but not against the other foliar diseases (Subrahmanyam and McDonald, 1983).

EARLY AND LATE LEAF SPOTS

Aetiology

Early leaf spot is caused by the fungus *Mycosphaerella arachidis* Deighton (Deighton, 1967; Mulder and Holliday, 1974a). The accepted anamorph is *Cercospora arachidicola* Hori (Jenkins, 1938; Chupp, 1953). Jenkins (1938) described the teleomorph as *Mycosphaerella arachidicola* but this name had already been applied to a different fungus with an *Ascochyta* conidial state. Deighton (1967) proposed that the name *Mycosphaerella arachidis* Deighton be used for the teleomorph of the early leaf spot fungus. Late leaf spot is caused by the fungus *Mycosphaerella berkeleyi* W.A. Jenkins (Jenkins, 1939; Mulder and Holliday, 1974b). The nomenclature of the anamorph has undergone several changes (Chupp, 1953; Porter *et al.*, 1984; McDonald *et al.*, 1985). Until recently the combination *Cercosporidium personatum* (Berk. & Curt.) Deighton (syn. *Cercospora personata* (Berk. & Curt.) Ell. & Everh.) was widely used (Deighton, 1967). The anamorphs of the genus *Mycosphaerella* were reorganized and 23 form-genera were enumerated, mainly on the basis of conidionatal structure and position on the host plant and the types of scars on the conidiogenous cells and conidia (von Arx, 1983). The accepted anamorph is *Phaeoisariopsis personata* (Berk. & Curt.) v. Arx. The teleomorphs of both fungi are rare. Leaf spots caused by *Cercospora* spp. and allied genera are common on legumes and are further

reviewed in Sinclair, Chapter 3; Allen *et al.*, Chapter 5; and Reddy *et al.*, Chapter 10, this volume.

Biology

Early and late leaf spots (also known as tikka spots, cercospora leaf spots and brown leaf spots) are very widespread on groundnut although early leaf spot is more restricted in distribution than late leaf spot (CMI, 1966, 1967; Jackson and Bell, 1969; McDonald *et al.*, 1985). As the names suggest, early leaf spot tends to appear earlier in the growing season and late leaf spot later in the season (Gibbons, 1966; Garren and Jackson, 1973; NRI 1996); however, it depends very much on environmental conditions and there can be both short- and long-term fluctuations in the relative proportions of either leaf spot.

Leaf spots of *C. arachidicola* are subcircular to irregular, coalescing, 1–10 mm in diameter, dark brown to black on the lower surface and reddish-brown to black on the upper surface (Mulder and Holliday, 1974a). A yellow halo usually develops. Fruiting is confined mostly to the upper surface. The stroma is present but slight, 25–100 μm in diameter and dark brown. Conidiophores are arranged in dense fascicles, five to many, pale olivaceous or yellowish-brown, darker at the base, mostly once geniculate, unbranched, septate, 15–45 × 3–6 μm. Conidia are subhyaline, slightly olivaceous, obclavate, mildly to much curved, up to 12 septa, base round, truncate, tip subacute, 35–110 × 3–6 μm. *M. arachidis* is characterized by scattered perithecia, mostly along lesion margins, amphigenous, partly embedded in host tissue, erumpent, ovate to nearly globose, 47.6–84.0 × 44.4–74.0 μm in size, black, ostiole slightly papillate; asci are cylindrical, clubshaped, short stipitate, fasciculate, 27.0–37.8 × 7.0–8.4 μm in size, aparaphysate, bitunicate, eight-spored; ascospores are uniseriate to imperfectly biseriate in ascus, bicellular, the upper cell somewhat larger, slightly curved, hyaline, 7.0–15.4 × 3–4 μm (mean 11.2 × 3.64 μm) in size (McDonald *et al.*, 1985).

Leaf spots of *P. personata* are circular, coalescing, dark brown to blackish-brown, 5–10 mm in diameter, occasionally a yellow halo appears in mature spots (Mulder and Holliday, 1974b). Fruiting is more often on the lower surface. The stroma is dense, pseudoparenchymatous, and up to 130 μm diameter. Conidiophores are numerous, sometimes in concentric circles on the spot, in dense to very dense fascicles, pale to olivaceous brown, smooth, geniculate, continuous or sparingly septate, 10–100 × 3–6.5 μm. Conidial scars conspicuous, prominent, thickened, 2–3 μm wide. Conidia are medium olivaceous, mostly concolorous with the conidiophores, cylindric, obclavate, usually straight or slightly curved, wall usually finely roughened, rounded at the apex, base shortly tapered with a conspicuous hilum, one to nine septa usually not constricted, mostly three to four-septate, 20–70 × 4–9 μm (Mulder and Holliday, 1974b). Differences between *M. berkeleyi* and *M. arachidis* are clearly exhibited in the nature of the conidia (Mulder and Holliday, 1974a, b). *M. berkeleyi* is characterized by scattered perithecia, mostly along lesion margins, amphigenous, partly embedded in host tissue, erumpent, broadly ovate to globose, 84–140 × 70–112 μm in size,

black in colour, ostiole slightly papillate; asci are cylindrical, club-shaped, short stipitate, fasciculate, 30–40 × 4–6 µm, aparaphysate, bitunicate, eight-spored; ascospores are uniseriate to imperfectly biseriate in the ascus, bicellular, the upper cell somewhat larger, slightly constricted at the septum, hyaline, 10.9–19.6 × 2.9–3.8 µm (mean 14.9 × 3.4 µm) in size (McDonald *et al.*, 1985).

There is some evidence for variation in pathogenicity in both *P. personata* and *C. arachidicola* (Gibbons, 1966; Subrahmanyam *et al.*, 1983a) but races of either pathogen have not been defined (Mulder and Holliday, 1974a, b). Although, evidence for host specificity has not been conclusively demonstrated, pathogen adaptation to local environments has been reported (Wynne *et al.*, 1991). This is thought to be due to environmentally induced alterations in host metabolism rather than pathogen specificity (Shew *et al.*, 1988). Recent studies have shown that isolates of *C. arachidicola* collected globally produce differential responses on some groundnut genotypes (Subba Rao *et al.*, 1993). The possibility of environmental adaptation of local pathogen populations should not be dismissed (Wynne *et al.*, 1991) and further studies are needed to understand the variability in both *P. personata* and *C. arachidicola*. Groundnut is the only known natural host of *C. arachidicola* and *P. personata* (McDonald *et al.*, 1985). Wild *Arachis* spp. are suspected natural hosts as some are susceptible under artificial inoculation.

Symptoms

Plants may be affected in the vegetative growing stage, flowering stage and pod filling stage and both fungi can infect leaves, stems, petioles and pegs. Symptoms are influenced by host genotype and environmental factors (Jackson and Bell, 1969; Porter *et al.*, 1984; McDonald *et al.*, 1985). Symptoms of both diseases are initially similar. Small chlorotic spots appear on leaflets about 10 days after infection and develop into sporulating lesions in a further 5 days. The subcircular lesions produced by *C. arachidicola* are up to 10 mm in diameter, reddish-brown to black on the adaxial leaf surface and lighter shades of brown on the abaxial surface (Plate 1) (Mulder and Holliday, 1974a; McDonald *et al.*, 1985). Distinct chlorotic haloes develop early on the upper surface but their presence and prominence is altered by host genotype and environmental factors. Similar haloes may be found around *P. personata* lesions (McDonald *et al.*, 1985). Lesions tend to be larger than those of *P. personata* and the dark stroma of the latter is absent. Conidia form on both leaf surfaces, the conidiophores being somewhat diffusely arranged.

The circular lesions produced by *P. personata* are up to 8 mm in diameter (usually smaller than those developed by *C. arachidicola*) and become dark brown or black (darker than those of *C. arachidicola*) (Jackson and Bell, 1969; Mulder and Holliday, 1974b; Porter *et al.*, 1984). Symptoms caused by *P. personata* are most clearly identified by the distinct dark stroma of the conidial state. On the abaxial surfaces, where most sporulation occurs, lesions are black with a slightly rough appearance (Plate 1) (McDonald *et al.*, 1985). Conidiophores and conidia are produced in concentric rings. In contrast, *C. arachidicola* forms conidia on both surfaces and has no dark, rough stroma (Mulder and Holliday, 1974b). A

distinctive, chlorotic halo is often present around *P. personata* lesions, but its presence and prominence are altered by host genotype and environmental factors. As similar haloes may be found around *C. arachidicola* lesions, the halo is not a good diagnostic character.

The colour of the lesion on the abaxial leaflet surface, light brown for *C. arachidicola* and black for *P. personata*, and the distribution of fruiting structures, randomly on the adaxial surface for *C. arachidicola* and in circular rings on the abaxial surface for *P. personata*, are useful diagnostic characters for distinguishing between the two leaf spots in the field (McDonald *et al.*, 1985). The two pathogens can also be readily identified by the morphology of conidiophores and conidia. In addition to causing leaf spots, the two pathogens also produce oval to elongate lesions on petioles, stems and pegs which have more distinct margins than the leaflet lesions. When disease attack is severe, the affected leaflets become chlorotic and necrotic and are shed. Severe attacks cause considerable defoliation.

Epidemiology

C. arachidicola and *P. personata* are both soilborne, disease onset being earliest and attack most severe when groundnut follows groundnut in the rotation (Gibbons, 1966; Garren and Jackson 1973; McDonald and Raheja, 1980; McDonald *et al.*, 1985). An attack by *C. arachidicola* normally precedes that of *P. personata*, but both diseases may appear within 3 to 5 weeks after sowing. Ascospores are generally not regarded as important sources of primary inoculum although Jenkins (1938) reported that ascospores produced on mycelium in the soil could be involved in early season infections. Conidia produced directly from mycelium in crop debris in the soil or on volunteer plants following early rains usually initiate the disease cycle when deposited on leaves of young plants by rain-splash and wind (McDonald *et al.*, 1985).

Temperatures of 18–30°C, leaf wetness of 20 hours and a total wetness period of greater than 160 hours favour infection and disease development (Butler *et al.*, 1994). The first lesions normally develop on the leaves near the soil surface and the conidia produced on them are carried by wind, rain splash and insects to younger leaves on the same plant and to adjacent plants (McDonald *et al.*, 1985). Leaves are susceptible during the entire growing season. Given favourable conditions, disease progresses throughout the season and may result in nearly total defoliation of plants. Recent studies have focused on assessment of the effects of relative humidity (RH), leaf wetness, temperature and light on conidial production by *P. personata* (Butler *et al.*, 1995). It was interesting to note that conidial production was less with continuous leaf wetness than with intermittent leaf wetness under continuous high RH (98–99%). With intermittent wetness, there was also clear evidence of trophic growth of germ tubes towards stomata and penetration (Wadia and Butler, 1994). With constant high RH (98–99%), conidial production increased linearly from 10 to 28°C during 2–6 days (Butler *et al.*, 1995).

Conidia may be detached from lesions at any time but peak release periods

occur when leaf surfaces dry in the morning (between 1 and 4 hours after sunrise), and at the onset of rainfall (Smith and Crosby, 1973). Air-dispersed conidia of both *P. personata* and *C. arachidicola* showed diurnal periodicity with peak catches occurring between 1000 and 1800 hours which increased rapidly with the onset of rain (Smith and Crosby, 1973; Alderman and Nutter, 1994). Evidence of vertical dissemination of conidia of *P. personata* to heights of 2.7 m was obtained (Smith and Crosby, 1973).

The pathogens may survive from season to season on volunteer groundnut plants and infected crop debris (McDonald *et al.*, 1985). Jackson and Bell (1969) note that conidia in crop debris have sufficient longevity to carry over from one season to another. However, in India *P. personata* survived on crop debris for 35–60 days only (Rao *et al.*, 1993). Under certain environmental conditions, crop debris may not be a major source of initial inoculum and volunteer plants and overlapping crops may be more important. Further studies on inoculum survival would be worth while. Survival of the perfect stages of both pathogens is not considered important epidemiologically. Long-distance distribution of the pathogens may be by airborne conidia, movement of infected crop debris, or movement of pods or seed that are surface-contaminated with conidia or crop debris (Jackson and Bell, 1969; McDonald *et al.*, 1985). There is no evidence of either pathogen being internally seedborne.

Effects on Yield and Quality

Late and early leaf spots are considered to be the most serious and widespread diseases of groundnut globally (Porter *et al.*, 1984; McDonald *et al.*, 1985; NRI, 1996). In the southern USA, where fungicide application is a normal practice, pod yield losses are estimated at around 10%. Where fungicides are not used, pod yield losses due to the two leaf spots alone can reach as high as 50% (Porter *et al.*, 1984). When rust is also present, losses can be as high as 70%. Losses in yield and quality of haulms can also be high due to serious defoliation during the 3–4 weeks prior to harvest (Cummins and Smith, 1973).

Management

Breeding resistant cultivars is one of the best means of reducing yield losses from diseases (Gibbons and Bailey, 1967; Cook, 1981; Porter *et al.*, 1984; McDonald *et al.*, 1985). It is the best strategy to help resource-poor farmers in the semi-arid tropics who generally lack the financial resources and technical expertise to use chemical control methods effectively. The need to breed resistant cultivars in developed countries to reduce farmers' dependence on fungicides is also critical. Many sources of resistance to early and late leaf spots have been reported and are available from various research institutes (Abdou *et al.*, 1974; Sowell *et al.*, 1976; Mixon *et al.*, 1983; McDonald *et al.*, 1985; Wynne *et al.*, 1991; Smith *et al.*, 1994; Subrahmanyam *et al.*, 1995). Genotypes resistant to late leaf spot are available from ICRISAT, listed in McDonald *et al.* (1985) and Subrahmanyam *et*

al. (1995). Some are also resistant to rust (Subrahmanyam *et al.*, 1995). As recently as 10 years ago, although many sources of resistance had been identified, there was no agronomically acceptable groundnut cultivar with resistance to either of the leaf spots (Wynne *et al.*, 1991). During the past few years, effective field and laboratory screening methods have been developed and systematic screening of groundnut germplasm for resistance to leaf spots has been intensively carried out in different parts of the world (Subrahmanyam *et al.*, 1995). Research is aimed at incorporating leaf spot resistance and high yield into cultivars with agronomic and quality characters suited to different environments (Subrahmanyam *et al.*, 1983b, 1995; Wynne *et al.*, 1991; Smith *et al.*, 1994). High-yielding breeding populations, with resistance to late leaf spot and rust, are routinely generated at ICRISAT (Subrahmanyam *et al.*, 1995). This material could be used immediately in developing countries but some quality characters will need to be improved for sophisticated markets. The recent rapid spread of groundnut rust has created a problem for breeders in incorporating resistance to all three diseases into agronomically acceptable cultivars (McDonald and Raheja, 1980). Success has been achieved for rust and late leaf spot, and sources of combined resistance to these diseases are listed in Subrahmanyam *et al.* (1995).

Considerable emphasis has been placed on screening wild *Arachis* spp. for resistance to leaf spots (Moss, 1980; McDonald *et al.*, 1985; Moss *et al.*, 1992; Singh and Nigam, 1996). Accessions of some wild *Arachis* spp. are highly resistant to both late leaf spot and rust (Subrahmanyam *et al.*, 1985b; Wynne *et al.*, 1991). Cytogenetic research aimed at incorporating leaf spot resistance from wild *Arachis* spp. into cultivated groundnut is in progress in several institutes. At ICRISAT, tetraploid lines incorporating resistance to late leaf spot and rust have been produced which are being used in breeding programmes in many countries (Sharief *et al.*, 1978; Moss *et al.*, 1992). High levels of resistance to late leaf spot and rust have been observed in a number of the derivative populations. In the near future, application of molecular tools including markers is expected to lead to the development of probes for resistance gene detection (J.P. Moss, ICRISAT, India, 1996, personal communication).

Resistance to leaf spot pathogens has been attributed to various morphological and anatomical characters of the host plant (Taber *et al.*, 1977; Mayee and Suryawanshi, 1995) and to different chemical constituents of leaves and seeds (Alabi and Naqvi, 1977). It operates by prolonging incubation and latent periods, and by reducing the number of lesions per unit area of leaf surface, defoliation, and sporulation (Nevill, 1981). Kornegay *et al.* (1980) proposed that resistance to leaf spots was quantitatively inherited. Nevill (1982) showed that late leaf spot resistance was determined by recessive alleles at five loci. At present it is accepted that resistance to both leaf spots is based on additive genetic effects (Wynne *et al.*, 1991). Waliyar *et al.* (1993a, b) found that expression of resistance to *C. arachidicola* varied across diverse geographical locations. Further studies showed that temperature affected the stability of components of resistance, especially lesion number, infection frequency and incubation period (Waliyar *et al.*, 1994b). Several groundnut genotypes were identified with stable resistance to *C. arachidicola* across all temperatures tested.

Where possible, there should be a distinct break between successive ground-

nut crops (Jackson and Bell, 1969; McDonald *et al.*, 1985). As the diseases are largely soilborne, rotation with other crops is important (Kucharek, 1975). Plant debris should be removed from the field after harvest, burned *in situ*, fed to animals or buried deeply. Volunteer groundnut plants and groundkeepers should be eradicated. Depending upon the length of the growing season and cultivars grown, the time of sowing may be adjusted to avoid infection of the crop from outside sources and to avoid environmental conditions conducive to disease build-up. Weeds should be kept under control because they may encourage disease development through modification of the crop microclimate (Jackson and Bell, 1969). Early maturing cultivars (95–100 days) may be mature before *P. personata* can build up and thus escape major disease problems. ICRISAT is placing emphasis on the development of such cultivars in several groundnut growing regions (Nigam *et al.*, 1995).

In developed countries, fungicidal control of leaf spots is effective and economic and has been widely adopted (Smith and Littrell, 1980; Subrahmanyam *et al.*, 1984; McDonald *et al.*, 1985; Culbreath *et al.*, 1995) but it has presented problems for resource-poor groundnut farmers in developing countries. In the USA, fungicides are applied by various kinds of tractor-propelled machines, aircraft, helicopters, and, more recently, through sprinkler irrigation systems. Recent studies have focused on developing disease forecasting systems based on climatological data (Jacobi *et al.*, 1995; Linvill and Drye, 1995; Wu *et al.*, 1996). According to Smith and Littrell (1980) there was a rapid move towards spray application following the introduction of highly effective fungicides such as benomyl, chlorothalonil and fentin hydroxide in the early 1970s; however, after several years of extensive use of benomyl, tolerant strains of *P. personata* and *C. arachidicola* appeared (Littrell 1974; Smith *et al.*, 1978). Benomyl is rarely used alone now for leaf spot control, but is used in mixtures with protectant fungicides. Chlorothalonil is now the most widely used fungicide for leaf spot control and it is also very effective for controlling rust and minor foliar diseases (McDonald *et al.*, 1985). An extensive review of chemical control of leaf spots can be found in McDonald *et al.* (1985). Mycoparasites, *Dicyma pulvinata* (Berk. & Curt.) v. Arx (Mitchell, 1984; Porter and Taber, 1992) and *Verticillium lecanii* (Zimmerm.) Viegas (Subrahmanyam *et al.*, 1990b) have been observed to parasitize the early and late leaf spot pathogens of groundnut. These were found to be effective in controlling leaf spots in greenhouse studies; however, no serious attempts have been made to use them in the field.

Every effort should be made to utilize all available and compatible disease control measures (Gorber *et al.*, 1982; McDonald *et al.*, 1985; Ghewande *et al.*, 1993). Breeders should endeavour to combine leaf spot resistance with resistance to rust and other diseases. Cultural and chemical control measures effective against one leaf spot will normally be effective against the other (Gibbons, 1966; Garren and Jackson 1973). Pande *et al.* (1993) noted that intercropping with pigeonpea resulted in higher levels of late leaf spot on groundnut compared to the sole crop. Similar observations have been made for angular leaf spot of *Phaseolus* bean, which is caused by *Phaeoisariopsis griseola* (Allen, 1990; see Chapter 4, this volume). If fungicides are the choice for leaf spot management, they should be capable of controlling both leaf spots and rust, and the possibility

of applying fungicides combined with insecticides should also be considered where insect pests are a problem. Recent work in Malawi has shown that partial resistance, combined with judicious use of fungicides (such as chlorothalonil) and cultural practices (such as early planting and crop rotation) can be effective in reducing early leaf spot (Subrahmanyam *et al.*, 1994a).

WEB BLOTCH

Aetiology

The causal agent of web blotch was earlier identified as a species of *Ascochyta* by many workers (Subrahmanyam *et al.*, 1994b). The taxonomy of the anamorphic state of the pathogen was clarified by Marasas *et al.* (1974) based on differences in conidiogenesis between *Phoma* and *Ascochyta*, described by Boerema (1965). The accepted anamorph is *Phoma arachidicola* Marasas, Pauer & Boerema. The teleomorph has been variously identified as a species of *Mycosphaerella*, *Didymella* and *Didymosphaeria* and details of the taxonomic debate are given in Subrahmanyam *et al.* (1994b). The now accepted teleomorph is *Didymella arachidicola* (Chochrjakov) Taber, Pettit & Philley.

Biology

Web blotch has received extensive treatment in Porter *et al.* (1984), Taber *et al.* (1984) and, more recently, by Subrahmanyam *et al.* (1994b). Web blotch of groundnut is also known as phoma leaf spot, ascochyta leaf spot, net blotch and 'spatselvlek' (Porter *et al.*, 1984). The disease has been reported from southern Africa, Australia, South America, China, Japan, the USA and the former USSR (Subrahmanyam *et al.*, 1994b). It is especially important in Zimbabwe on irrigated long-duration cultivars, in the Vaalharts, Transvaal and Natal regions of South Africa, and in Texas, USA. Leaf blight caused by *Phoma/Ascochyta* spp. is common on legumes and similar diseases are reviewed in Chapters 4, 5 and 6, this volume.

Pycnidia of *P. arachidicola* are pale to dark brown, separate, globose to flask-shaped, ostiolate, amphigenous, and immersed in leaf tissues, 85–240 μm in diameter and produced in concentric rings, corresponding to periods of light and dark (Subrahmanyam *et al.*, 1994b). Pycnidiospores arise as buds on conidiogenous cells, and are hyaline, smooth-walled, and subglobose to ellipsoid with rounded ends. They vary in size with substrate and septation; single-celled spores from culture measure 4–9 × 2.5–4 μm while on the host, spores may be larger and are often septate. Spore size is influenced by temperature, and cultural characteristics vary with temperature, light and medium (Taber *et al.*, 1984). Optimum temperature for mycelial growth is 20°C while pycnidial production is greatest at 25°C. Chlamydospores may be formed by some isolates (Subrahmanyam *et al.*, 1994b). These are brown, thick-walled, round to irregular, 8–19 × 8–17 μm, and may be formed singly or in chains. Pseudothecia of

D. arachidicola are dark brown, subglobose to globose, separate, usually immersed in host tissues, ostiolate, and measure 65–154 μm in diameter (Subrahmanyam *et al.*, 1994b). Asci are hyaline, cylindrical to somewhat clavate, mostly with a differentiated foot, eight-spored and distichous. Ascospores are uniseptate, smooth, hyaline at first, becoming dark with maturity, with the upper cell broader and more sharply tapered than the lower cell, and measure 4.5–6 × 13–17 μm.

Groundnut is the only known natural host of *P. arachidicola* but it can also be successfully inoculated to soyabean, sweet clover, alfalfa and hairy vetch (Pettit *et al.*, 1986). There is no evidence of variation in pathogenicity in *P. arachidicola*. Pettit *et al.* (1986) reported that all isolates from USA, South Africa and Argentina were equally pathogenic on groundnut and the above legumes.

Symptoms

Lesions first appear on the adaxial surfaces of lower leaves as scattered tan-coloured specks or streaks that form a webbed pattern (Plate 2) (Porter *et al.*, 1984; Subrahmanyam *et al.*, 1994b). Each strand of the web is associated with a single hyphal strand. The discoloured areas expand forming nearly circular, purplish-brown to dark brown blotches with inconspicuous margins. The blotches may coalesce and cover entire leaflets. Older lesions are dark brown to black with roughened surfaces, and tend to dry out and crack. Symptoms appear later on the abaxial leaf surface. The web and blotch symptoms may develop in sequence on the same leaflet or independently on different leaflets. When environmental conditions are favourable, hyphal growth is extensive, leaf tissue is severely damaged, and premature defoliation results with subsequent reduction in pod yield (Porter *et al.*, 1984; Pettit *et al.*, 1986; Subrahmanyam *et al.*, 1994b).

Epidemiology

Web blotch tends to be more severe under cool (15–20°C), moist conditions in the semi-arid tropics and subtropics (Blamey *et al.*, 1977). It is more severe on irrigated crops than on rainfed crops in the USA (Liddell, 1990) and Zimbabwe (Rothwell, 1962). Liddell (1990) reported that temperatures below 29°C and diurnal cycles of relative humidity above 85% with periods over 95% favour web blotch development in the USA. Subrahmanyam and Smith (1989) found a highly positive correlation between temperature and leaf wetness period on web blotch development. Increasing the duration of leaf wetness from 2 to 8 days increased web blotch development between 15 and 25°C.

The fungus survives in infected crop residues or on volunteer plants (Pettit *et al.*, 1986). Fruiting bodies are formed on fallen infected leaves under moist conditions (Luttrell and Smith, 1981; Pettit *et al.*, 1986). Pycnidia and pseudothecia provide the initial inoculum and infection and pycnidiospores, ascospores and chlamydospores are all capable of initiating infection of leaflets, petioles, stipules and stems, invasion being directly through the cuticle and subsequent growth

being intercellular. Younger plants tend to be more susceptible than older plants (Subrahmanyam and Smith, 1989).

Effects on Yield and Quality

Web blotch can be as destructive as early and late leaf spot, causing serious damage in the USA (particularly in Texas) and in southern Africa (South Africa and Zimbabwe) (Subrahmanyam *et al.*, 1994b). It is not as widespread as the two leaf spots and rust but can cause losses of 10–18% on its own and up to 40% when combined with early or late leaf spots (Blamey *et al.*, 1977; Hildebrand, 1987).

Management

High levels of resistance have been found in groundnut cultivars, germplasm lines and wild species of *Arachis*, and these are listed in Subrahmanyam *et al.* (1994b). In general, resistant accessions show low infection frequency, long incubation periods, small lesions, minimal defoliation and leaf area damage (Subrahmanyam and Smith, 1987; Subrahmanyam *et al.*, 1994b). Resistance to *P. arachidicola* in groundnut cultivars appears to be manifest as fewer successful infections from pycnidiospores and reduced development on the host. A few sources of resistance have been used in breeding programmes in Zimbabwe (Hildebrand, 1987; Subrahmanyam *et al.*, 1994b).

Crop rotation with non-hosts and eradication of infected crop debris and infected volunteer plants can reduce sources of inoculum (Subrahmanyam *et al.*, 1994b). Several fungicides commonly used against leaf spots and rust can reduce web blotch damage and are reviewed in Subrahmanyam *et al.* (1994b). Cole (1981, 1982) reported interactions between web blotch and early leaf spot and found that by delaying fungicide application for leaf spot control, suppression of web blotch was more successful. Mancozeb, benomyl and chlorothalonil gave good control of leaf spots and moderate control of web blotch, but did not give satisfactory control of severe web blotch disease. There are good prospects for developing integrated disease management packages incorporating cultural practices such as rotation and eradication of crop residues and volunteer plants, judicious use and timing of fungicides, and adequate levels of resistance to both web blotch and other foliar diseases of groundnut (Subrahmanyam *et al.*, 1994b).

BACTERIAL WILT

Aetiology and Biology

The causal agent of bacterial wilt is *Burkholderia solanacearum* (E.F. Smith) Yabuuchi *et al.* (syn. *Pseudomonas solanacearum* (E.F. Smith) E.F. Smith). The taxonomy, characteristics, natural and inoculated host range and geographical

distribution of the bacterium are reviewed by Bradbury (1986). *B. solanacearum* is an aerobic, non-fluorescent, non-spore-forming, rod-shaped, gram-negative bacterium, approximately 0.5 × 1.5 μm (Kelman, 1954; Hayward, 1964). Key diagnostic features are its cultural characteristics on tetrazolium agar medium where virulent isolates, which are mainly non-flagellate and non-motile, form irregularly round, fluidal, creamy white colonies with light pink centres (Kelman, 1954). Extracellular slime formation is a common attribute of all virulent isolates of *B. solanacearum*. Avirulent isolates usually bear one to four polar flagella and are highly motile. The bacterium produces a brown diffusible pigment on some media including tyrosine. Acid production from carbohydrates varies greatly between biovars and strains. Optimum temperature for growth varies between 25 and 35°C. Further information is available in a recent review (Mehan *et al.*, 1994b).

B. solanacearum has a very wide natural host range which includes many crop plants and weeds that are found in groundnut production systems (Bradbury, 1986). Susceptible legumes include *Phaseolus* bean, lupins, pea, soyabean, faba bean, cowpea, *Trifolium* spp. and *Stylosanthes* spp. The species is highly heterogeneous (Bradbury, 1986). Isolates are classified into five races based on host range (Buddenhagen and Kelman, 1964; He *et al.*, 1983), and into five biovars based on biochemical characteristics (Hayward, 1964; He *et al.*, 1983). Races and biovars are informal groupings at the intraspecific level. Although the two systems of classification are largely independent, each system has contributed considerably to understanding the complex pathogenicity of *B. solanacearum* (Mehan *et al.*, 1994b). Race 1 isolates cause wilt in groundnuts and in many other leguminous and solanaceous plants. Biovar 1 isolates cause wilt of groundnut in the USA; biovar 3 and, to a lesser extent, biovar 4 isolates cause wilt of groundnut in Asia and Africa (Hayward, 1991). Biovars 2 and 5 have not been reported from groundnut.

Serological techniques based on both polyclonal and monclonal antibodies have been developed for detection and identification of *B. solanacearum* (Alvarez *et al.*, 1993; Robinson, 1993). Molecular techniques are also being developed to enhance detection of the bacterium and to understand better its considerable genetic diversity (Cook *et al.*, 1989; Seal, 1994). *B. solanacearum*-specific DNA sequences have been identified and oligonucleotide primers constructed for those regions which helped in detecting single cells of the bacterium in hosts by the polymerase chain reaction (PCR) (Seal, 1994). A rapid identification test was also developed for distinguishing biovars 3, 4 and 5 based on restriction fragment length polymorphism DNA probes. Further work is focusing on the development of an immunocapture PCR test for *B. solanacearum*.

Bacterial wilt is most important on groundnut in the warm, humid and subhumid tropics, especially in South Asia and South-east Asia (Hayward, 1991; Mehan *et al.*, 1994b). It has also caused sporadic damage in wetter areas of the semi-arid tropics. In general, isolates of *B. solanacearum* from groundnut are reported to be more virulent on groundnut than isolates from other hosts (Mehan *et al.*, 1994b); however, strains from groundnut differ greatly in their pathogenicity on the host (Tan *et al.*, 1992). Further information is needed on the geographic distribution of races that infect groundnut before specific linkages

with environmental conditions can be made to explain why wilt is severe in some zones but not in others.

Symptoms

Very young seedlings may wilt but it is more common for wilting to commence at flowering (Fig. 2.4) (Mehan *et al.*, 1994b). *B. solanacearum* invades groundnut through wounds or through natural openings in roots. The bacteria enter the water-conducting tissues, multiply, and block the vessels causing wilting of the plant. Infection of young plants results in rapid wilting of stems and foliage but leaves remain green until final rotting occurs. When older plants or partially resistant varieties are infected, wilting proceeds more gradually, usually starting with the lateral branches. Infected plants have discoloured and rotted roots, and sometimes rotted pods. A useful diagnostic characteristic is the streaming of bacterial ooze from the cut ends of infected stems and roots when these are immersed in water.

Epidemiology

The disease is soilborne and its long-term survival is favoured by continuous cropping of groundnut and other host plants and by the presence of weed hosts

Fig. 2.4. Bacterial wilt of groundnut caused by *Burkholderia solanacearum* (E.F. Smith) Yabuuchi *et al.* (Photo: courtesy of V.K. Mehan).

(Mehan *et al.*, 1994b). The bacterium is mainly disseminated through water and infested soil. Soil temperatures above 25°C together with high soil moisture favour the development of wilt (Wang *et al.*, 1983). Wilt peaks when the soil temperature is over 30°C for 10 days (Tan and Liao, 1990). Under continuously wet conditions, wilt develops and spreads but severe wilt symptoms may not appear for some time. However, infected plants wilt rapidly if they are subjected to a dry period. A clear relationship between soil type and groundnut wilt has not been established. In Indonesia, wilt is predominantly a problem of heavy clay or loam soils (Machmud, 1986), whereas in China wilt is more common in sandy soils (He, 1990). Wilt is less prevalent in soils with high organic matter and preliminary information suggests that alkaline soils are wilt-suppressive (Yeh, 1990). Machmud and Middleton (1990) found seed transmission of 5–8% in freshly harvested seed, but this was greatly reduced by drying. Seed transmission is of obvious quarantine significance and more research is needed to determine the extent of transmission (Mehan *et al.*, 1994b).

Effects on Yield and Quality

Bacterial wilt is a major constraint to groundnut production over large areas of China, Indonesia and Vietnam (Mehan *et al.*, 1994b). Yield losses are usually in the range of 10–30%, but losses can reach 60% when highly susceptible cultivars are grown in heavily infested fields. In China it is estimated that the annual loss of groundnut pods from wilt exceeds 50,000 t. Severe losses are also reported from parts of Malaysia, Fiji, Papua New Guinea and Uganda (Opio and Busolo-Bulafu, 1990; Mehan *et al.*, 1994b).

Management

Crop sanitation measures such as burning of crop residues and removal of weeds, and cleaning of implements after cultivation help to reduce carry-over and spread of the disease (Mehan *et al.*, 1994b). Adjustment of sowing date to avoid periods of high temperature and soil moisture has had limited success (Kelman, 1953). Rotation with immune or highly resistant crops including rice, maize, sugarcane, soyabean and sorghum is useful (Wang and Hou, 1982; He 1990). Chemical control is not economically feasible. Varieties with high levels of resistance and good agronomic qualities have been bred and released in China and Indonesia (Liao *et al.*, 1990; Yeh, 1990; Machmud, 1993) and Mehan *et al.* (1994b) list 54 resistant genotypes. In addition, several wild *Arachis* spp. are highly resistant (Yeh, 1990). The best approach to management of wilt is to combine appropriate cultural control practices with the planting of resistant varieties. Considerable progress has been made in understanding cropping practices which significantly reduce wilt but more information is needed on how they affect the survival of the bacterium (Mehan *et al.*, 1994b).

BUD NECROSIS OR SPOTTED WILT

Aetiology and Biology

Three distinct viruses can cause bud necrosis disease or spotted wilt of groundnut. These are tomato spotted wilt virus (TSWV), groundnut ring spot virus (GRSV) and peanut bud necrosis virus (PBNV). TSWV is widely distributed in the Americas, Australasia, Africa and Europe. GRSV is reported from South America and Africa. PBNV appears to be restricted to South and South-east Asia (Reddy *et al.*, 1991a). These viruses are present in all organs of infected plants, and clusters of virus particles are often found in the endoplasmic reticulum. Particles of the three viruses are of similar shape and size: spherical, covered with projections, and of 80–120 nm diameter. The nucleocapsid (N) protein of TSWV and GRSV is 29 kDa, and that of PBNV is 31 kDa. TSWV, GRSV and PBNV are grouped under tospoviruses in the family *Bunyaviridae*. They are differentiated on the basis of serological cross-reactions (de Avila *et al.*, 1992; Adam *et al.*, 1993) and amino acid sequence homology of the N protein (de Avila *et al.*, 1993; Satyanarayana *et al.*, 1996 a, b). They all contain three single-stranded RNA species, called small RNA (*c.* 2900 nucleotides), medium RNA (*c.* 5000 nucleotides) and large RNA (*c.* 9000 nucleotides). The large RNA has negative polarity, whereas the medium and small RNAs are ambisense, i.e. both negative and positive sense RNAs can code for polypeptides. The large RNA codes for a single large protein, whereas each of the medium and small RNAs codes for two proteins (Goldbach and Peters, 1996). Tospoviruses have a very low thermal inactivation point (45°C for 10 min) and their longevity *in vitro* is less than 5 h at room temperature. The viruses have a very wide host range: over 600 plant species in more than 70 families. These include many crop and weed species commonly found in groundnut production systems (Reddy and Wightman, 1988; Peters and Goldbach, 1995).

Symptoms

Symptoms of diseases caused by TSWV and PBNV in groundnut are similar (Reddy *et al.*, 1991a, b). Initial symptoms appear on young leaflets as chlorotic spots or mottling that may develop into chlorotic and necrotic rings and streaks until the whole plant is affected (Fig. 2.5). Terminal bud necrosis (Fig. 2.6) often occurs when temperatures are relatively high. Early infection results in stunting and sometimes proliferation of axillary shoots. Leaflets produced on axillary shoots are reduced in size and may show puckering, chlorosis, mosaic, and distortion of the lamina. Any seeds produced on early infected plants are small, shrivelled, and testae have red, brown or purple mottling. Late-infected plants may produce seed of normal size; however the testa of such seed is often mottled (Reddy *et al.*, 1991a, b).

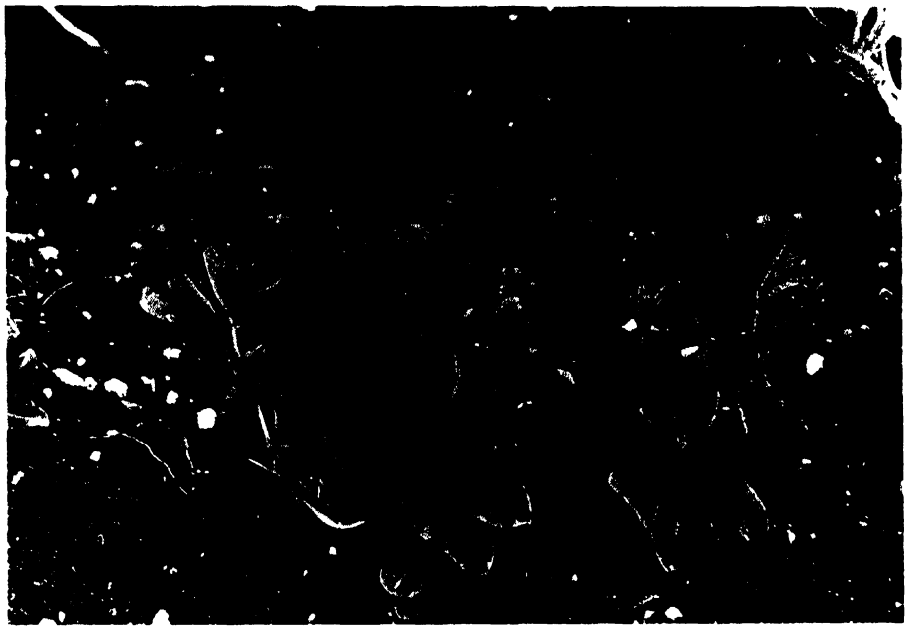

Fig. 2.5. Early infection of groundnut by tomato spotted wilt virus resulting in stunting (Photo: courtesy of D.V R. Reddy).

Fig. 2.6. Terminal bud necrosis of groundnut caused by peanut bud necrosis virus (Photo: courtesy of D.V.R. Reddy).

Epidemiology

Diagnostic hosts are *Vigna unguiculata*, concentric chlorotic and necrotic rings being formed on leaves of cvs. C-152 or California Black Eye 4–5 days after inoculation, and *Petunia hybrida*, necrotic lesions appearing on leaves within 3–4 days. While the viruses can be mechanically transmitted, natural transmission is by thrips, TSWV and GRSV by *Frankliniella occidentalis* Perg., *F. schultzei* (Trybom), *F. fusca* (Hinds), *Thrips tabaci* Lindeman, and *T. setosus* Moulton, and PBNV by *Thrips palmi* Karny. Transmission is in a persistent manner, viruses being acquired by larvae and transmitted by adults. The last instar larvae can transmit. These viruses multiply in their vectors and are not seed-transmitted in groundnut (Reddy *et al.*, 1991a, b; Goldbach and Peters, 1994). The primary source of inoculum is likely to be from a range of hosts, which include weeds as well as crop plants. Incidence will be dependent on the sources of inoculum and factors that contribute to multiplication and spread of vector thrips (Reddy *et al.*, 1983).

Effects on Yield and Quality

Yield losses can be as high as 80% when infection occurs early in the crop season and many plants are infected. PBNV has caused serious damage in India, and TSWV has been a serious problem in Australia and more recently in the USA (Reddy *et al.*, 1991). Of more than 1000 plant viruses, TSWV is included among the top ten of the most devastating plant viruses (Goldbach and Peters, 1994).

Management

Management of bud necrosis has concentrated on control of the thrips vector. Insecticide use is not recommended as this can lead to increased levels of the disease and may encourage other pest problems. If information is available on time of arrival of principal thrips vectors, and if the growing season permits, sowing dates may be adjusted so that plants are well established and the crop canopy developed before infection occurs (Reddy *et al.*, 1983). Field resistance which appears to operate mainly against the vectors has been found in some cultivars and germplasm lines. ICGV −86029, −86031, −86388, −91239, −91245, −91246 and −91249 have shown field resistance. Preliminary evidence indicates that ICGV-86388 and −91239 may also possess resistance to PBNV (Dwivedi *et al.*, 1995). In the USA, the cv. Southern Runner showed 50% less incidence of TSWV than did cv. Florunner (Culbreath *et al.*, 1993). Transgenic peanut plants expressing the 'N' gene of TSWV have been produced (Z. Li, J.W. Demski and R. Jarrett, Griffin, Georgia, USA, 1996, personal communication). They are currently being field-tested in south Georgia, USA.

PEANUT CLUMP

Aetiology and Biology

Peanut clump disease is caused by at least two distinct viruses of the furovirus group. In West Africa the disease is caused by peanut clump virus (PCV) (Thouvenel *et al.*, 1988), while that in India is caused by the variable Indian peanut clump virus (IPCV) (Reddy *et al.*, 1988). PCV and IPCV are not related serologically, and IPCV's coat protein is only 61% identical to that of PCV. Both viruses have rod-shaped particles of 24 nm in diameter with two predominant lengths of *c.* 185 and 250 nm. Each of the particles encapsidates a distinct RNA molecule. The two RNA molecules of IPCV (Hyderabad isolate) (Miller *et al.*, 1996) and PCV (Senegal isolate) (Herzog *et al.*, 1994) have been fully sequenced. Serologically distinct variants occur within PCV and, especially, IPCV, where four distinct serotypes have been identified.

Symptoms

Peanut clump is soilborne and occurs in patches in the field which recur in the same positions when groundnuts are grown again (Fig. 2.7) (Reddy *et al.*, 1988). Young leaves show mosaic, mottling, and chlorotic ring symptoms. Older leaves are darker green with faint mottling. Early infected plants are severely stunted;

Fig. 2.7. Patches of groundnut affected by peanut clump virus in Rajasthan, India (Photo: courtesy of P. Delfosse).

they may produce flowers, but the pods are not properly developed. Late-infected plants may also show stunting, and leaves are dark green; they produce pods but seeds are much reduced in weight (Reddy *et al.*, 1988).

Epidemiology

PCV and IPCV have extremely wide host ranges which include both monocotyledons (e.g. sorghum, finger millet, wheat, foxtail millet and barley) and dicotyledons (e.g. *Phaseolus vulgaris*, *Vigna unguiculata*, *V. mungo*) (Reddy *et al.*, 1988; Delfosse *et al.*, 1996). Both viruses are readily sap-transmissible. They are thought to be transmitted by the soil-inhabiting fungus *Polymyxa graminis* Ledingham (Ratna *et al.*, 1991) and are seed-transmitted in groundnut with over 6% frequency. IPCV has been shown to be seed-transmitted in millets but not in sorghum (Reddy *et al.*, 1988). Distribution of rainfall, rotation with highly susceptible cereal crops (to both the virus and the vector) and date of sowing have immense influence on the disease incidence. Developing evidence indicates that infected groundnut seed may not provide inoculum to the fungal vector. However, infected seed from cereal hosts is likely to play an important role in the disease establishment in soils infested with *Polymyxa* spp. (P. Delfosse, A.S. Reddy and D.V.R. Reddy, ICRISAT, India, 1996, personal communication). With the help of geographic information systems it is possible to locate the potential areas for the occurrence of the disease. Parameters which helped in this process were soil type, temperature, rainfall and cropping system. This information will help immensely in assessing the economic importance of the disease (P. Delfosse and D.V.R. Reddy, unpublished data).

Effects on Yield and Quality

Yield losses can be serious (up to 60% even in late-infected crops) in crops grown in light sandy soils in drier areas of the semi-arid tropics of South Asia and West Africa. The build-up of inoculum in the soil can lead to groundnut being abandoned in such areas (Reddy *et al.*, 1988).

Management

Soil solarization for about 2 months in hot summer weather can reduce IPCV incidence (Reddy *et al.* 1988). In some areas it may be possible to grow an irrigated post-rainy season crop, if temperatures remain below 25°C, as clump incidence is much reduced compared with the rainy season crop. Several soil biocides have given successful control of the disease but may not be economical and pose environmental hazards. No resistant variety has been found despite extensive screening (Reddy *et al.*, 1988). As the genomes of PCV and IPCV have been sequenced there are prospects for inducing resistance in groundnut by non-conventional methods (Mayo *et al.*, 1995; Miller *et al.*, 1996).

Care is necessary to avoid movement of infected seed to new areas. Seed of cereal crops from infested soils should not be used for planting. As several of the most important cereal crops used in groundnut production systems of the semi-arid tropics are susceptible to PCV and IPCV, management of the disease will require careful selection of rotation crops and integration of all feasible control measures. Prospects are also good for controlling the disease by sowing trap crops, such as pearl millet, prior to planting groundnut. The trap crop should be removed at the seedling stage before multiplication of the vector in the root system. In preliminary trials from groundnut crops raised in soils treated in this manner either escaped or showed much lower disease incidence than those, untreated soils (P. Delfosse, A.S. Reddy and D.V.R. Reddy, ICRISAT, India, 1996, personal communication).

PEANUT STRIPE

Aetiology and Biology

Peanut stripe disease is caused by the peanut stripe potyvirus (PStV) which is included under the 'Bean Common Mosaic' subgroup (Demski *et al.*, 1984). The disease is now present in most areas of South-east and South Asia, in North America, and has recently been found in South America and in West Africa. The virus particles are filamentous flexuous rods, about 752 nm long and 12 nm in diameter. PStV is serologically distinct from peanut mottle virus which it resembles, and is related to blackeye cowpea mosaic virus, bean common mosaic virus, and soyabean mosaic virus. The complete nucleotide sequence for the PStV genome is available (Gunasinghe *et al.*, 1995). Demski *et al.* (1993) list natural and diagnostic hosts of PStV and explain how these and serological tests can be used to differentiate the virus from peanut mottle virus.

Symptoms

There are several symptom variants of PStV, and host variety may also influence symptom expression. The name was derived from the isolate that induces discontinuous chlorotic stripes along the lateral veins of young leaflets (Fig. 2.8) (Demski *et al.*, 1984). Another variant, which causes irregular darker green blotches on young leaflets, that persist as leaflets age, is the most widely distributed (Demski *et al.*, 1993). Yet another induces chlorotic rings surrounding blotches on young leaflets. An isolate widely distributed in China induces a mild mottle on leaflets. An 'oak leaf' symptom is occasionally seen (Wongkaew and Dollet, 1990; Demski *et al.*, 1993).

Epidemiology

PStV is transmissible by sap inoculation. It is naturally transmitted by many aphids in a non-persistent manner, and this is probably its only means of spread

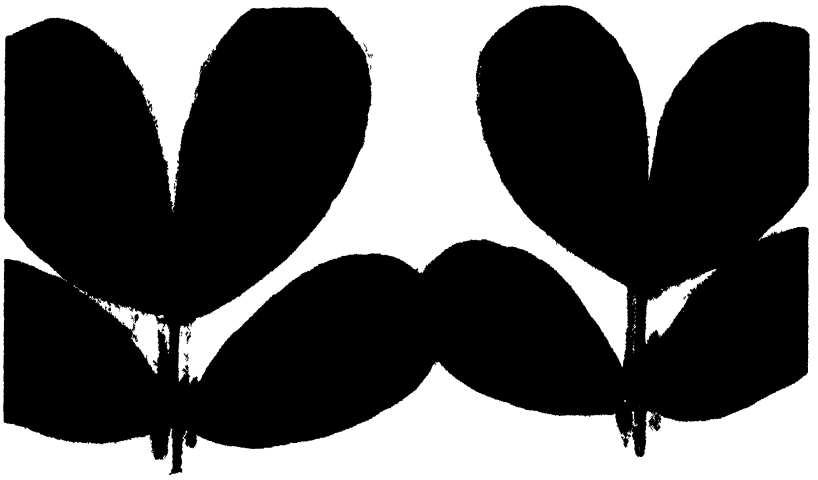

Fig. 2.8. Chlorotic stripes on groundnut leaflets caused by peanut stripe virus (Photo: courtesy of D.V.R. Reddy).

from its primary source under field conditions (Demski *et al.*, 1993). *Aphis craccivora* Koch. is a very efficient vector; *A. gossypii* Glover rather less effective. PStV can be transmitted through seed at up to 37% frequency, but the normal rate of transmission is usually less than 5%. The primary source of inoculum is likely to be seed. Secondary spread is by aphids (Demski *et al.*, 1993).

Effects on Yield and Quality

PStV can seriously affect yield of groundnut in South-east Asia. Yield loss in infected plants can be as high as 70% (Middleton and Saleh, 1988). An annual yield loss from the disease in northern China was estimated at 200,000 t (Xu, 1988).

Management

The most important factor in management of PStV is to sow only virus-free seed. For very high value germplasm, non-destructive serological testing of seed is possible to eliminate infected materials. It may be possible to find environments where the vectors are not present for seed production. Despite extensive resistance screening, no resistant germplasm has been identified in cultivated groundnut (Demski *et al.*, 1993). Some wild *Arachis* spp. have shown resistance, and interspecific hybrids may show promise. Research is in progress to produce transgenic plants with PStV genes (Reddy *et al.*, 1994).

GROUNDNUT ROSETTE

Aetiology and Biology

Groundnut rosette disease is caused by a complex of two viruses and a satellite RNA (Murant *et al.*, 1995). One component is a mechanically transmissible virus, groundnut rosette virus (GRV). It depends for transmission by *Aphis craccivora* on the presence of the aphid-transmitted groundnut rosette assistor virus (GRAV). GRV RNA was shown to contain 4019 nucleotides and can code for four proteins (Taliansky *et al.*, 1996). A satellite RNA of *c.* 900 base pairs is dependent on GRV for its multiplication. It is mainly responsible for the symptoms of rosette disease and is also needed for aphid transmission. GRV is a member of the genus umbravirus (Murant *et al.*, 1995). GRAV is a typical luteovirus and can be detected by polyclonal antisera produced against it, and other luteoviruses such as potato leaf roll or bean leaf roll viruses. GRAV can also be detected by some monoclonal antibodies produced for the potato leaf roll virus (Murant, 1990). Satellites can be detected by cDNA probes. No virus particles have been associated with GRV, but infectious single-stranded RNA is present in diseased plants. It can be identified by symptoms on *Chenopodium amaranticolor* and *Nicotiana clevelandii*, and by the presence of double-stranded RNA of 4600 and 1300 base pairs. Since satellite RNA is fully dependent on GRV for its replication, the presence of a satellite is diagnostic for GRV. Satellites occur as different variants. The 'green' and 'chlorotic' forms of rosette are caused by different satellites. Another variant was shown to cause mottle symptoms. Laboratory studies have revealed the presence of a large number of variants of satellite RNA, which can induce distinct symptoms (Blok *et al.*, 1994). Rosette is widespread in Africa south of the Sahara but there are no authenticated cases of its occurrence elsewhere.

Symptoms

Three types of rosette are recognized based on symptoms. The most common is chlorotic rosette which is found in all regions but is prevalent in East and Central Africa (Porter *et al.*, 1984). Faint chlorotic mottling appears on young leaflets and subsequently produced leaflets are pale yellow with green veins, reduced in size, curled and distorted (Plate 3). Early infected plants are severely stunted. In later infected plants, individual branches only may show symptoms. Few pods are formed and these may not develop properly. Another form, green mottle, occurs in West Africa and in Uganda (Porter *et al.*, 1984). Young leaflets have mild chlorotic mottling with isolated flecks. Older leaflets are dark green, remain small and show outward rolling of their margins (Plate 3). Early infected plants are severely stunted and produce no mature pods. The third form, mosaic mottle, is less common, being found in eastern, central and southern Africa. Young leaflets show conspicuous mosaic symptoms (Porter *et al.*, 1984).

Epidemiology

All forms of rosette are transmitted by *A. craccivora* in a persistent manner. GRV is only transmitted from plants that also contain the GRAV. It is possible to infect many plant species with rosette by aphid transmission, but no alternative host is known to be involved in perpetuation of the disease. Currently groundnut itself is suspected of being the main source of infection of the crop, possibly through infected groundkeepers and volunteer plants (Reddy, 1991). Recently developed diagnostic tools should facilitate epidemiological studies now in progress in Malawi to clarify the situation regarding perpetuation of the disease.

Effects on Yield and Quality

Groundnut rosette causes annual crop losses of under 5%, but the sporadic epidemics can be extremely serious. One such outbreak in West Africa in 1975 destroyed much of Nigeria's crop and losses were estimated at 560,000 t (Yayock *et al.*, 1976; Yayock, 1977). As a result, much of the groundnut production in northern Nigeria changed from sole to intercrop systems. A rosette epidemic occurred in southern Africa (Malawi, Zambia, Zimbabwe) in 1983. During the 1994/95 season, a rosette epidemic occurred in Malawi (Lilongwe, Kasugu and Mchingi) and in the eastern provinces of Zambia. The crop losses in Zambia were estimated to be over US$ 4.5 million. Yield reduction in both the countries was so severe that it resulted in an acute shortage of seed for the 1995/96 season (P. Subrahmanyam, Lilongwe, Malawi, 1995, personal communication).

Management

Rosette disease can be effectively managed by cultural practices such as crop sanitation, destruction of groundkeeper and volunteer plants, and early sowing of good quality seed at recommended rates to ensure rapid establishment of ground cover (Reddy, 1991). Broad spectrum insecticides are not recommended as they may worsen the situation by killing predators of the aphid vectors, but systemic aphicides can be used if cost effective. High levels of resistance are available based on germplasm originating from West Africa, and this has been used to breed medium- or long-duration rosette-resistant cultivars in West and southern Africa, e.g. RMP 41, 48, 91 and 93, RG 1 and RRI/6, /16 and /24. In on-farm trials, a long-duration virginia bunch type ICGV-SM 90704 out-performed the local cultivars (Chiyembekeza *et al.*, 1996). These cultivars are suitable for cultivation only in the wetter areas of Africa. The resistance, which is controlled by two independent recessive genes, is against the GRV and satellite components of the complex only. Recently rosette resistance was observed in 16 early maturing Spanish types (Subrahmanyam *et al.*, 1996). Many advanced short-duration breeding lines have recently been developed. Integration of resistant cultivars with cultural control practices should be effective in managing rosette disease.

PEANUT MOTTLE

Aetiology and Biology

Peanut mottle virus (PMV), a potyvirus, is present in all the major groundnut growing countries (Porter *et al.*, 1984; Brunt *et al.*, 1990). It is the most widespread virus of groundnut. Its seedborne nature and availability of aphid vectors in most environments probably account for this wide distribution (Reddy and Demski, 1996). The virus particles are similar to those of PStV, being filamentous flexible rods of 750 nm in length and of 12 nm diameter. PMV is serologically distinct from PStV, peanut green mosaic and groundnut eye spot potyviruses. The coat protein has an apparent molecular weight of 32–36 kDa. A comparison of nucleotide sequences of PMV with PStV revealed similarities of 64.4% in the coat protein gene and 34.6% in the three regions (Dietzgen *et al.*, 1994).

Symptoms

PMV produces a range of symptoms on groundnut. The youngest leaflets may show a mild mottle or a mosaic of irregular dark green islands (Porter *et al.*, 1984). In older leaflets mosaic symptoms are not so obvious but can be seen in transmitted light. In some genotypes, conspicuous interveinal depression and inward curling of the margins of leaflets can occur (Fig. 2.9). Plants are slightly

Fig. 2.9. Interveinal depressions and inward curling of leaf margins of groundnut caused by peanut mottle virus (Photo: courtesy of D.V.R. Reddy).

stunted, and both number and size of pods are reduced by the disease (Reddy and Demski, 1996).

Epidemiology

PMV has a natural host range that includes several important legume crops (cowpea, *Phaseolus* bean, soyabean, lupin) and weeds that occur in groundnut cropping systems (Porter *et al.*, 1984). The virus is both sap-transmissible and seed-transmissible in groundnut at low frequency (usually less than 1.0%, the maximum recorded is 8.5%). It is also seed-transmitted in cowpea, mung bean and common bean. PMV is transmitted in a non-persistent manner by *Aphis craccivora*, *A. gossypii*, *Myzus persicae* (Sulzer), *Hyperomyzus lactucae* (L.), *Rhopalosiphum padi* (L.) and *R. maidis* (Fitch), (Reddy and Demski, 1996).

Effects on Yield and Quality

Crop losses from PMV rarely exceed 6%, and are influenced by incidence and time of infection. Susceptible cultivars tested in India, however, sustained yield losses as high as 40% (Reddy and Demski, 1996).

Management

As infected seed appears to be the primary source of inoculum (Kuhn and Demski, 1975), it is obviously important to sow only virus-free seed. Non-destructive serological testing procedures are available to detect infected seed but this process is only feasible for small quantities of high value materials, e.g. for germplasm maintenance or exchange. Genotypes with very low levels of seed transmission have been found and are being used in breeding programmes. Resistance to PMV has been located in some wild species of *Arachis* but has not yet been transferred to the cultivated groundnut (Reddy and Demski, 1996).

ROOT-KNOT

Aetiology and Biology

Four species of *Meloidogyne* produce root-knots and galls on pegs and pods of groundnut. *Meloidogyne arenaria* (Neal) Chitwood and *M. javanica* (Treub) Chitwood are found mainly in tropical regions, and *M. hapla* Chitwood in temperate, cooler regions (Minton and Baujard, 1990). *M. incognita* Kofoid & White occurs in the Mediterranean region. *M. javanica* populations capable of infecting groundnut were originally thought to be of restricted distribution, but this species has recently been observed to be widespread on groundnut in India and Egypt (Sharma and McDonald, 1992; Tomaszewski *et al.*, 1994) and it is emerging

as an important parasite of groundnut (Sharma *et al.*, 1995). *M. arenaria* is widespread and probably the most damaging nematode pest of groundnut on a world scale (Minton and Baujard, 1990). Root-knot nematodes have wide host ranges including legumes and are further reviewed on tropical pasture legumes by Lenné, Chapter 13, this volume.

Identification of *Meloidogyne* spp., which is important for designing efficient management strategies, has been principally based on perineal pattern morphology and host preferences. However, due to considerable variation in these characters, newer criteria have been developed. Isozyme analysis (Esbenshade and Triantaphyllou, 1985), polymerase chain reaction (PCR) method (Power and Harris, 1993), random amplified polymorphic DNA (RAPD) assay (Cenis, 1993), and serological methods have been helpful in identification of *Meloidogyne* spp. populations.

According to Porter *et al.* (1984), the nematodes exist in soil as egg masses, infective second-stage larvae, and adult males. Eggs are elongate and ovate measuring 30–60 × 75–113 µm. The larvae which are 430–470 µm long, move through the soil and penetrate plant tissue through which they move to a region near vascular tissue. They lose mobility and feed on adjacent plant cells. The larvae develop into adults and the enlarged females produce large numbers of eggs. Cells of roots, pegs or pods react to the damage by producing galls. Eggs released into the soil hatch and a further cycle of infection and disease is initiated.

Symptoms

Plants infected with root-knot nematodes may or may not show chlorosis and/or stunting of above-ground parts. Infected roots and pegs are enlarged and galls of varying size are produced on them; protrusions or warts develop on pods (Porter *et al.*, 1984). Symptoms are similar for all species, but galls are rather smaller when *M. hapla* is involved and increased root branching results in a bushy root system. Severe root-knot attack reduces the efficiency of symbiotic nitrogen fixation by damaging nodules. The nematodes have also been held responsible for increasing the incidence and severity of several soilborne diseases, particularly those caused by species of *Fusarium* and *Pythium*.

Effects on Yield and Quality

Nematodes reduce groundnut yields by feeding on roots with resulting loss of vigour; they also cause direct damage to pegs and pods (Middleton *et al.*, 1994). Sasser and Freckman (1987) estimated that the annual groundnut yield loss worldwide due to nematodes was around 12%, and that equates with a monetary loss of approximately US$ 1 billion. Yield losses from root-knot in the USA are considered to be economically important but rarely exceed 5%. The disease is also considered to be important in Australia, China, Egypt and India (Sharma and McDonald 1990). Groundnut yields have been negatively correlated with numbers of infective nematode larvae in the soil.

Management

Management practices used against nematodes include crop sanitation, rotation with non-host plants, use of resistant varieties and application of nematicides. Nematicides are commonly used for control of root-knot disease in the USA and other developed countries (Middleton *et al.*, 1994). Nematode populations in the soil may be reduced by rotation with crops such as castor, maize, sesame, sorghum and pearl millet. No resistant cultivars of groundnut are available, but resistance to the various nematodes has been found in several groundnut genotypes and in related wild *Arachis* species (Wynne *et al.*, 1991). Nematode problems have not received the attention they warrant and more information is needed before effective integrated disease management packages can be assembled.

CURRENT SITUATION AND RESEARCH NEEDS

Considerable progress has been made towards the control of groundnut diseases since they were reviewed by Garren and Wilson (1951), but very few of the problems they outlined 45 years ago have been fully resolved, and new diseases and problems have appeared. Early crop protection research in North America placed much emphasis on use of pesticides, and increasingly effective fungicides were developed for control of leaf spots. These gave economic control in developed countries and for large-scale farmers in tropical developing countries, but were rarely satisfactory for use by resource-poor farmers in the tropics. For the latter, cultural control measures and resistant varieties were considered to be more appropriate. This and the increasing concern for protecting the environment led to emphasis being given to resistance breeding with a view to developing integrated crop protection packages involving use of resistant varieties, cultural control measures, biological control and appropriate use of pesticides.

International cooperation has resulted in a comprehensive world collection of groundnut and related *Arachis* species germplasm being assembled. Sources of resistance to many important diseases have been found within this collection and these are being used to breed agronomically acceptable varieties with good levels of resistance. Multiple disease resistance is required for most production systems and this has proved relatively easy for some diseases, e.g. rust and late leaf spot, but difficult to achieve in other cases, e.g. rosette and early leaf spot. If current research into the transformation of groundnut by insertion of foreign genes proves to be successful, the objective of multiple disease resistance will be much closer. This genetic engineering approach has particular relevance for developing groundnut with resistance to viruses.

It is important to have a clear understanding as to what diseases occur in each groundnut production system, their prevalence and severity over seasons and how they are influenced by other crops in the system, cultural practices etc. Such data are often missing or inadequate, and high priority should be given to disease surveys and crop loss evaluations. These activities could be coordinated at national, regional and international levels and stored in geographic information

systems where they could be correlated with information on soils, climate, and other data used for crop simulation modelling. The microclimatic data gathered for physiological growth models would have much relevance in calculating disease risk and developing forecasting systems.

Further research is needed on effects of temperature, humidity and leaf wetness in the canopy on infection by leaf spot and rust fungi, as small variations can greatly influence disease development (Butler *et al.*, 1994). The data would be incorporated into the crop models and the techniques developed in such research could be used in studying pathogen variability. Another approach to this would be to collect isolates of a pathogen from different regions of the world and screen them in one environment at one time for reaction on a selected range of groundnut genotypes. This has been tried for early leaf spot disease and considerable variability was found (Subba Rao *et al.*, 1993).

Groundnut crop protection should address the cropping system as a whole if full advantage is to be taken of cultural control measures such as adjustment of sowing dates, use of varieties of different crop duration, rotations, intercropping, cultivations and land form, plant population and spacing patterns etc. Interactions between different diseases, and with abiotic stresses such as drought and unfavourable temperatures, also require more study. The economic and socio-economic aspects of integrated crop protection packages should be examined when these packages are being field-tested.

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DISEASES OF SOYABEAN 3

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INTRODUCTION

The soyabean, *Glycine max* (*Fabaceae: Phaseoleae*), was domesticated by farmers in the eastern half of northern China during the Shang dynasty (*c.* 1700–1300 BP) or perhaps earlier. For several thousand years people in eastern Asia have used the soyabean for food and animal feed and as a medicine to treat a number of human disorders. An extensive review of the origin and early history of this crop was published with highlighted references to soyabean in books written over 4500 years (Probst and Judd, 1973). The geographic distribution suggests that the botanical home of *Glycine* may be Australia.

During the 1993/94 season, soyabeans were grown in at least 45 countries with a production of 113,069,000 metric tonnes (t). The major producers of the crop are the USA (44%), Brazil (21%), the People's Republic of China (12%) and Argentina (11%) (Soytech, Inc., 1995). Soyabeans are a primary source of vegetable oil and protein. The oil is used in cooking oil, margarine, salad oils, and shortening. The soya meal or cake remaining after the oil is extracted is used for animal feed and textured protein, used as a meat extender and in prepared foods. In addition to being used in the formulation of new, low-cost, nutritionally balanced, high-protein foods and beverages for human consumption, studies under way suggest that certain components may be beneficial to human health. Various industrial uses include making biodiesel fuels, paints, plastics, insecticides and adhesives.

The moisture, soil and temperature requirements of the soyabean are similar to those of maize (*Zea mays*), which explains why the major producing areas are in temperate and subtropical regions. Soyabean seeds require a moisture content of 50% on a dry weight basis for germination. Therefore, a good supply of moisture is critical at planting time. Another critical period is during the R3 to R4 growth stages (Fehr *et al.*, 1971). Soyabeans flower over a 3- to 4-week period and can resist short periods of moisture stress. Soyabeans grow best between soil pH 5.8 and 7.0 with an average between pH 6.3 and 6.5. Soyabeans grow well in

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a range of soil types, the type determining specific practices to obtain the maximum yield. Soil temperature should be 10°C or above before planting and about 30°C for germination and rapid emergence, but other factors may determine planting date. The ideal temperature for growth is 30°C. Sustained temperatures below 24°C will delay the onset of flowering. Flowers will not set seed if night temperatures fall below 10°C. Successful seed set occurs when night temperatures reach 21°C followed by day temperatures of at least 27°C (Scott and Aldrich, 1983). A standard method for reporting soyabean growth stages has been devised (Table 3.1). This terminology should be used in reporting data associated with growth stages. Because of their response to photoperiod, soyabean cultivars are placed in 13 maturity groups designated 000 to X. Cultivars in the 000 group are the earliest in maturity and are adapted to the northern- and southernmost world production areas; groups IX and X are used primarily in subtropical and tropical areas of low altitude. Most soyabean cultivars are adapted for full-season growth in a band no wider than 160 to 240 km (Scott and Aldrich, 1983). Therefore, unlike most other crops, improved genetic traits must be incorporated in cultivars of the appropriate maturity group to be of value.

Table 3.1. Growth stage key for soyabean disease evaluation.

Stage	Description[1]
V1	Completely unrolled leaf at the unifoliolate node
V2	Completely unrolled leaf at the first node above the unifoliolate node
V3	Three nodes on the main stem, beginning with the unifoliolate node
VN	N nodes on the main stem, beginning with the unifoliolate node
R1	One flower at any node
R2	Flower at node immediately below the uppermost node with a completely unrolled leaf
R3	Pod 0.5 cm long at one of the four uppermost nodes with completely unrolled leaf
R4	Pod 2 cm long at one of the four uppermost nodes with a completely unrolled leaf
R5	Seeds beginning to develop (can be felt when the pod is squeezed) at one of the uppermost four nodes with a completely unrolled leaf
R6	Pod contains full-sized green seed at one of the four uppermost nodes with a completely unrolled leaf
R7	Pods yellowing, 50% of leaves yellow (physiologic maturity)
R8	95% of pods brown (harvest maturity)

Source: Fehr *et al.* (1971) (Reproduced by permission of the Crop Science Society of America, Inc., Madison, Wisconsin, USA).

[1] To use the key to report disease occurrence at specific stages of soyabean growth, first select a random sample of soyabean plants for disease assessment. The sample may consist of individual leaves or plants, groups of plants, or all plants in a plot. Next, calculate the average infection for the units in the sample. Then, determine the vegetative stage by counting the number of nodes on the main stem (beginning with the unifoliolate node) that have a completely unrolled leaf and finding the corresponding entry in the table.

More than 100 diseases are known to affect soyabeans (Sinclair and Backman, 1989); about 20 are important economically. The economic importance of any single disease may vary from one geographic area to another in any one season. The major diseases that are considered in detail in this chapter are the following: frogeye leaf spot, anthracnose, stem canker, pod and stem blight, fusarium root, collar and pod rot, charcoal rot, rust, phytophthora root and stem rot, pythium root and seedling rot, rhizoctonia diseases, sclerotinia stem rot, septoria brown spot, bacterial blight, bean yellow mosaic, soyabean mosaic, the bud blights and soyabean cyst nematode. Minor diseases are presented in Tables 3.2, 3.3 and 3.4. Non-infectious and stress-related diseases are not considered. For additional information the reader is referred to *The Pathology of Tropical Food Legumes* (Allen, 1983) and the *Compendium of Soybean Diseases* (Sinclair and Backman, 1989).

During the 1994/95 growing season, a conservative estimate of the world loss to all soyabean diseases was over 19.6 Mt (about 15%). Losses in the USA alone were 2.88 Mt in 1989, 2.14 Mt in 1990, and 2.21 Mt in 1991 (Doupnik, 1993; Wrather *et al.*, 1995a).

FROGEYE LEAF SPOT

Causal Pathogen

Cercospora sojina Hara, which is classified in the subdivision *Deuteromycotina* and class *Hyphomycetes*, is highly variable. *C. kikuchii* causes a distinct disease (see Table 3.2). Leaf spots caused by *Cercospora* spp. and allied genera are also reviewed in McDonald *et al.* (Chapter 2, this volume), Allen *et al.* (Chapter 5, this volume) and Reddy *et al.* (Chapter 10, this volume).

Biology

Frogeye leaf spot occurs worldwide but is common in warmer regions during warm, humid weather. Often it is prevalent in one area and scarce in another. Conidiophores arise in fascicles of 2 to 25 from a thin stroma, and are light to dark brown, 52–110 × 4–6 μm. Conidia are non- to 10-septate, hyaline when young, and elongate to fusiform, tapering toward the tip. The base usually is rounded. On infected leaves, conidia are 24–108 × 3–9 μm, but can be even larger. The fungus appears restricted to soyabeans. Five physiologic races have been reported (Phillips and Boerma, 1988). Race 2 is seedborne but does not sporulate on soyabean seeds.

Table 3.2. Fungal diseases of soyabean of local or minor economic importance worldwide.

Disease	Causal agent	Distribution	References
Leaf spot/pod necrosis	*Alternaria alternata* (Fr.) Keissler	Worldwide	Shortt *et al.* (1982); Vaughan *et al.* (1989); Kunwar *et al.* (1986a)
	A. tenuissima (Kunze ex Pers.) Wiltshire	Worldwide	
	Alternaria spp.	Worldwide	
Ascochyta leaf spot	*Ascochyta sojicola* Abramoff, *A. phaseolorum* Sacc.	Eastern Africa	Ondieki (1973); Allen (1983)
Choanephora leaf blight	*Choanephora infundibulifera* (Curr.) Sacc.	Thailand, USA	Roy (1993); Subba Rao *et al.* (1990)
Target spot	*Corynespora cassiicola* (Berk. & M.A. Curtis) C.T. Wei	Widespread	Carris *et al.* (1986); Hansen & Nelson (1994)
Cylindrocladium black rot and red crown rot	*Cylindrocladium crotalariae* (C.A. Loos) D. K. Bell & Sobers (teleomorph *Calonectria crotalariae* D.K. Bell & Sobers.); *C. clavatum* C.S. Hodges & L.C. May	Brazil, Cameroon, Japan, USA	Berner *et al.* (1991); Dianese *et al.* (1986); Kuruppu and Russin (1995)
Cercospora blight and leaf spot; purple seed stain	*Cercospora kikuchii* (T. Matsumoto and Tomoyasu) Gardner	Worldwide	Fernandez and Sinclair (1990); Schuh (1992); (1993); Sinclair (1992); Orth and Schuh (1994)
Red leaf blotch	*Dactuliochaeta glycines* (R.B. Stewart) Hartman and Sinclair (syn. *Pyrenochaeta glycines* R.B. Stewart), sclerotial state is *Dactuliophora glycines* Leakey	Cameroon, Ethiopia, Malawi, Nigeria, Rwanda, Uganda, Zaire, Zambia and Zimbabwe	Stewart (1957); Datnoff *et al.* (1987); Hartman *et al.* (1987); Hartman and Sinclair (1988); Levy *et al.* (1990); Akem *et al.* (1992); Schilder *et al.* (1995)
Sudden death syndrome	*Fusarium solani* (Mart.) Sacc.	USA	Hershman *et al.* (1990); McClean and Lawrence, (1993); Stephens *et al.* (1993); Melgar and Roy, (1994); Hartman *et al.* (1995); Rupe and Ghur (1995); Wrather *et al.* (1995b)

usarium wilt	*Fusarium oxysporum* f. sp. *glycines* Armstr. & Armstr., *F. o.* f. sp. *tracheiphilum* (E.F. Sm.) W.C. Snyder & H.N. Hans., *F.o.* f. sp. *vasinfectum* (Atk.) W.C. Snyder & H.N. Hans.	Worldwide	Armstrong and Armstrong (1950, 1958, 1965)
'owdery mildew	*Microsphaera diffusa* Cooke & Peck	Worldwide	Lohnes and Bernard (1992); Lohnes and Nickell, (1994)
Mycoleptodiscus root rot	*Mycoleptodiscus terrestris* (J.W. Gerdemann) Ostazeski	India, North Africa, USA	Gray (1978)
leocosmospora stem rot nd wilt	*Neocosmospora vasinfecta* E.F. Sm. (anamorph *Acremonium* sp.)	Nigeria, USA	Gray *et al.* (1980); Mengistu and Grau (1986)
)owny mildew	*Peronospora manshurica* (Naumov) Syd. ex Gaum	Worldwide	Lim *et al.* (1984); Dunleavy (1987); Lim (1989)
Brown stem rot	*Phialophora gregata* (Allington & Chamberlain) W. Gams	Egypt, Japan, Mexico, North America, Yugoslavia	Willmot *et al.* (1989); Mengistu *et al.* (1991); Sills *et al.* (1991); Waller *et al.* (1992); Adee *et al.* (1995)
Phomopsis seed decay	*Phomopsis longicolla* Hobbs	Worldwide	Kunwar *et al.* (1985); Rupe (1990); Sinclair (1993)
Thielaviopsis root rot	*Thielaviopsis basicola* (Berk. & Broome) Ferraris	Canada, Germany, former USSR, USA	Anderson (1984); Chun and Lockwood (1985)
Phyllosticta leaf spot	*Phyllosticta sojaecola* C. Massal. (teleomorph *Pleosphaerulina sojicola* Miura)	Worldwide	Walters and Martin (1981)
Sclerotium blight	*Sclerotium rolfsii* Sacc. (teleomorph *Athelia rolfsii* (Curzi) Tu & Kimbrough)	Worldwide	Gazaway and Hogan (1989)
Scab	*Sphaceloma glycines* Kurata & Kuribayashi	Japan	Jenkins (1951)

Table 3.3. Bacterial diseases of soyabean of minor importance.

Disease	Causal agent	Distribution	References
Seed decay	*Bacillus subtilis* (Ehrenberg) Cohn	Worldwide	Tenne *et al.* (1977)
Tan spot and wilt	*Curtobacterium flaccumfaciens* pv. *flaccumfaciens* (Hedges) Collins & Jones	Canada, former USSR, USA; locally destructive	Hedges (1926); Dunleavy (1985)
Wilt	*Ralstonia solanacearum* (Smith) Yabuuchi *et al.*	Ukraine, USA; uncommon	Hedges (1926); Miakushko and Baranova (1984); Yabuuchi *et al.* (1995)
Bacterial brown spot	*P. syringae* pv. *syringae* van Hall	Kenya	Kaiser and Ramos (1980)
Wildfire	*P. s.* pv. *tabaci* (Wolf & Foster) Young, Dye & Wilkie	Brazil, USA	Ribeiro *et al.* (1979); Sinclair and Backman (1989)
Pustule	*Xanthomonas campestris* pv. (Nakano) Dye	Worldwide	Groth and Braun (1989); Hwang *et al.* (1992)

Table 3.4. Virus diseases of soyabean of minor importance.

Disease	Causal agent (vectors)	Distribution	References
Pod mottle	Bean pod mottle comovirus (bean leaf beetles)	USA	Lin and Hill (1983); Abney and Ploper (1994)
Yellow mosaic	Bean yellow mosaic potyvirus (aphids)	USA	Afanasiev and Morris (1952)
Mild mottle	Cowpea mild mottle carlavirus (whiteflies)	Asia, Africa	Thouvenel *et al.* (1982); Iwaki *et al.* (1986)
Severe mosaic	Cowpea severe mosaic comovirus (beetles)	Tropical America	Anjos and Lin (1984)
Stripe	Peanut stripe potyvirus (aphids)	People's Republic of China, USA	Mishra *et al.* (1993)
Dwarfing	Soybean dwarf luteovirus (aphids)	Australia, Japan, New Zealand	Fletcher (1993); Hewings *et al.* (1986)
Yellow mosaic	Mung bean yellow mosaic geminivirus (whiteflies)	India, Thailand	Honda *et al.* (1986)

Symptoms

This is primarily a foliage disease, but stems, pods and seed may be infected. Leaf lesions are circular to angular, varying from less than 1 mm to 5 mm or more in diameter. Lesions are at first brown with a reddish-brown margin, then the central area becomes light brown to ash grey with dark margins. The leaf spots may coalesce to form larger affected areas (Fig. 3.1). When lesions are numerous the leaves wither and drop prematurely.

Epidemiology

The fungus survives as mycelium in infected seeds (Singh and Sinclair, 1985) and in infested soyabean debris. Heavily infected seeds that germinate produce weak seedlings that bear lesions. Sporulation on cotyledons provides primary inoculum. On inoculated plants, lesions are visible after 9–12 days and, under ideal conditions, the first spores are produced within the next 24–48 h. Conidia are carried short distances by air currents and splashing rain. If there are alternating wet and dry periods, a layered pattern of heavily and lightly diseased leaves may develop. Seed colonization takes place through pod infection (Kunwar *et al.*, 1985). Reactions of cultivars to the pathogen vary from immunity to high susceptibility which results in variation in symptoms and conidial production.

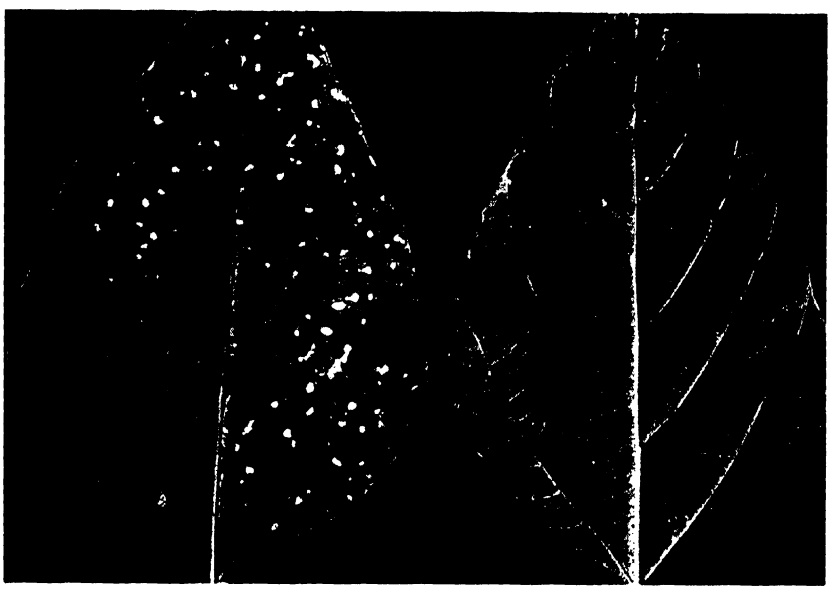

Fig. 3.1. Soyabean leaflets with symptoms of frogeye leaf spot, caused by *Cercospora sojina*, on upper leaf surface (left) and lower leaf surface (right) (photo: courtesy of J.T. Yorinori).

Damage and Crop Loss

Yield losses on susceptible cultivars may be 15% (Dashiell and Akem, 1991). Greater losses have been reported in certain areas of Brazil. Discoloured seeds due to the pathogen may reduce seed quality and value (Bisht and Sinclair, 1985).

Management

Plough under crop residues and sow high quality seeds of adapted or resistant cultivars that are relatively free of the pathogen. Use a seed dressing when planting low quality or infected seeds. Rotate soyabeans with other crops for two years. If frogeye leaf spot occurs in a seed crop, apply a foliar fungicide at growth stages R2–R5.

ANTHRACNOSE

Causal Pathogen

The most common fungal pathogen associated with anthracnose is *Colletotrichum truncatum* (Schwein.) Andrus & Moore. Other species that can be involved include *C. destructivum* O'Gara (teleomorph *Glomerella glycines* F. Lehman & F.A. Wolf), *C. gloeosporioides* (Penz.) Penz. & Sacc. in Penz. (teleomorph *G. cingulata* (Stoneman) Spauld. & H. Schrenk), and *C. graminicola* (Ces.) G. W. Wilson (teleomorph *G. graminicola* Politis). These fungi are classified in the subdivision *Deuteromycotina* class *Coelomycetes*. *C. truncatum* also affects tropical pasture legumes (see Lenné, Chapter 13, this volume), while other *Colletotrichum* spp. affect common bean (Allen *et al.*, Chapter 4, this volume), cowpea (Allen *et al.*, Chapter 5 this volume), lupins (Hill, Chapter 11, this volume) and tropical pasture legumes (see Lenné, Chapter 13, this volume).

Biology

Anthracnose is found wherever soyabeans are grown and causes severe losses in warm, humid areas following prolonged rainy spells. Isolates of *C. truncatum* vary in colony characteristics, sclerotial production (Khan and Sinclair, 1992), size of fruiting structures and pathogenicity. *C. truncatum* is characterized by crowded, black acervuli borne on well-developed stromata. The acervuli are oval to elongated, hemispherical to truncate-conical, and erumpent, with numerous black, needle-like setae intermixed long and short, 60–300 × 3–8 μm. Conidia are borne singly on conidiophores and are bluntly tapered, curved, unicellular and hyaline. Conidia measure 7–31 × 3–4.5 μm. *C. truncatum* has a wide host range including a variety of crop and weed species (Sinclair, 1988a).

Symptoms

All plant parts can be affected, causing damping-off, leaf veinal necrosis, pod lesions, stem cankers, premature defoliation, and seed discoloration (Fig. 3.2). Plants may be infected throughout the growing season and not show symptoms (Sinclair, 1991). Dark brown to black irregular lesions and cankers form on various plant parts; infected seeds appear dirty brown (Sinclair, 1992). Infected plant parts exhibit spiny bristles (setae) and black fruiting structures (acervuli) which are scattered or clustered.

Epidemiology

The fungus overseasons as mycelium in infected seeds and in crop and weed host residues, which are sources of inoculum for pre- and post-emergence damping-off. Severe damage to all plant parts results, regardless of age, under warm, moist conditions following a prolonged period of rain. The optimum soil temperature for root infection is 30°C (Khan and Sinclair, 1991). Disease forecasting methods used in the south-eastern United States predict infections from growth stages R1 to R5–6 and allow the timely use of foliar fungicides.

Fig. 3.2. Anthracnose lesions on soyabean leaf petioles, caused by *Colletotrichum truncatum*, which are similar to those that appear on stems and pods (photo: courtesy of P.R. Hepperly).

Damage and Crop Loss

The disease can reduce stand, seed quality, and yield by 16–26% or up to 100% in tropical and subtropical areas.

Management

Plough under crop residues and sow high quality seeds that are relatively free of the pathogen. If anthracnose is a chronic problem in seed production fields, spray with a foliar fungicide between growth stages R2 and R5. Polyamine biosynthesis inhibitors may offer promise for control of seedborne *C. truncatum* (Gamarnik *et al.*, 1994).

STEM CANKER

Causal Pathogens

Diaporthe phaseolorum (Cooke & Ellis.) Sacc. f. sp. *caulivora* K.L. Athow & R.M. Caldwell (anamorph *Phomopsis phaseoli* (Desmaz.) Sacc.), primarily in the northern USA, and *D. phaseolorum* (Cooke & Ellis) Sacc. f. sp. *meridionalis* F.A. Fernandez, in the southern USA. Both are classified in the subdivision *Ascomycotina*, class *Pyrenomycetes* and order *Diaporthales*. *D.p.* var. *sojae* (Lehman) Wehmeyer causes a distinct disease, pod and stem blight.

Biology

This disease is part of the *Diaporthe/Phomopsis* disease complex of soyabeans (see 'Pod and stem blight'). Although pod and stem blight and phomopsis seed decay are worldwide in distribution, stem canker has been reported only in Brazil, Europe and North America. On potato-dextrose agar, colonies of the northern type are white and closely appressed, later becoming flocculent, whereas those of *D. p.* f. sp. *meridionalis* are tan to buff, with early development of tufted aerial mycelium, which later becomes lanose (McGee and Biddle, 1987). Chlamydospores form in f. sp. *meridionalis* giving a tan to buff coloration to the colonies. Stromata of f. sp. *caulivora* are circular, while those of f. sp. *meridionalis* are irregular in shape, 2–10 mm in diameter. Perithecia readily form; the perithecial neck of f. sp. *caulivora* is half the length of f. sp. *meridionalis*. Isolates of f. sp. *meridionalis* contain only alpha conidia. Both are homothallic (Welch and Gilman, 1984).

Perithecia of f. sp. *caulivora* are black and globose and formed in caespitose groups of 2 to 12; in f. sp. *meridionalis*, perithecia are borne singly. Isolates of f. sp. *caulivora* produce perithecia embedded in cortical tissues, measure 165–340 × 282–412 μm, with a protruding beak that is variable in length and width. The

eight-spored asci, 30–40 × 4–7 μm, are sessile and elongate–clavate, with thin, evanescent walls, slightly thickened at the base. Ascospores, 8–12 × 3–4 μm, are hyaline, elongate–ellipsoidal, two-celled, slightly constricted at the septum, and biguttulate in each cell. The fungus may occur on other crops and weeds (Kulik, 1984) (see 'Pod and stem blight').

Symptoms

The first symptoms appear as small, reddish-brown lesions, usually near the leaf node. The lesions expand longitudinally to form dark brown to black, sunken cankers (Fig. 3.3). Leaf symptoms develop at this stage, with interveinal chlorosis and necrosis characteristic of diseases associated with restricted water conduction.

Fig. 3.3. Lesions and cankers associated with nodes of a soyabean plant infected by *Diaporthe phaseolorum* var. *caulivora*, cause of stem canker (photo: courtesy of the US Department of Agriculture).

Epidemiology

The source of primary inoculum is from overwintering debris. Primary spread is through infested debris, contaminated equipment, and infected or infested seeds, with the latter being of least importance (Damicone *et al.*, 1990). Perithecia develop any time after senescence or plant death but require 8 or more days of free moisture at temperatures above 20°C (Keeling, 1988; Subba Rao *et al.*, 1992; Tubajika and Russin, 1995). Mature perithecia produce viable ascospores for up to 3 weeks. Plants develop the maximal levels of disease if infected by the V3 growth stage. All cultivars are colonized by the pathogen, but only susceptible cultivars allow disease development. The quantity of spores produced by perithecia is related to cultivar susceptibility: the more susceptible, the more spores that are produced.

Damage and Crop Loss

Both fungi cause plant death as a result of stem girdling. Disease losses are minimal in the northern USA with the advent of resistant cultivars, but losses in the southern USA vary from year to year, with losses as much as 100% in susceptible cultivars.

Management

Rotate soyabeans with maize or other non-host crops and use tolerant or less susceptible cultivars. Benzimidazole fungicide sprays coordinated with infection periods have been successful in reducing disease severity.

POD AND STEM BLIGHT

Causal Pathogen

Diaporthe phaseolorum (Cooke & Ellis) Sacc. var. *sojae* (Lehman) Wehm. (anamorph *Phomopsis phaseoli* (Desm.) Sacc.). The fungus is classified in the subdivision *Ascomycotina*, class *Pyrenomycetes*, and order *Diaporthales* (Morgan-Jones, 1989).

Biology

Colonies of *D. p.* var. *sojae* on potato-dextrose agar are floccose and ropy, turning tan to brown with age. In reverse, the colonies are tan to dark brown with black, pulvinate stromata. Conidiomata are pycnidial, black, stromatic, solitary or aggregated, and usually unilocular, with no beak, or with a beak less than 200 μm long, opening by an apical ostiole. Locules are uni- to multiostiolate, lenticular,

and up to 350 μm wide. Condiophores are simple phialides, hyaline and up to 20 × 1.5–2.0 μm. Beta conidia are common, hyaline, filiform, and hamate. Perithecia are produced on old agar cultures in light or on overwintered soyabean stems. Mature perithecia are nearly spherical and slightly flattened at the base, 148–346 μm, have long tapered beaks, 60–100 × 60–150 μm. They are not usually clustered. Asci, which measure 35–51 × 3.3–1.0 μm, are elongate and clavate and dissolve before ascospore liberation. The asci are unitruncate, with a distinct apical ring. They are released in a viscous fluid that oozes out of the ostiole. Ascospores are bicellular, 9–13 × 2–6 μm, biguttulate in both cells. The causal fungi colonize debris of pigeonpea (*Cajanus cajan*), common bean (*Phaseolus vulgaris*), lima bean (*P. lunatus*), cowpea (*Vigna unguiculata*), garlic (*Allium sativum*), onion (*A. cepa*), lespedeza (*Lespedeza* spp.), lupins (*Lupinus* spp.), groundnut (*Arachis hypogaea*), okra (*Abelmoschus esculentus*), pepper (*Capsicum frutescens*), and tomato (*Lycopersicon esculentum*) (Kulik, 1984).

Symptoms

The pod and stem blight fungi first cause a latent infection (Sinclair, 1991). Pycnidia form on abscised leaves or broken branches (Fig. 3.4). In wet seasons, latent infections result in pycnidia being produced over the entire plant when it matures; in a dry season they are confined to areas on the stems near the soil line and nodes (Sinclair, 1988b). Pycnidia develop on dry, poorly developed pods.

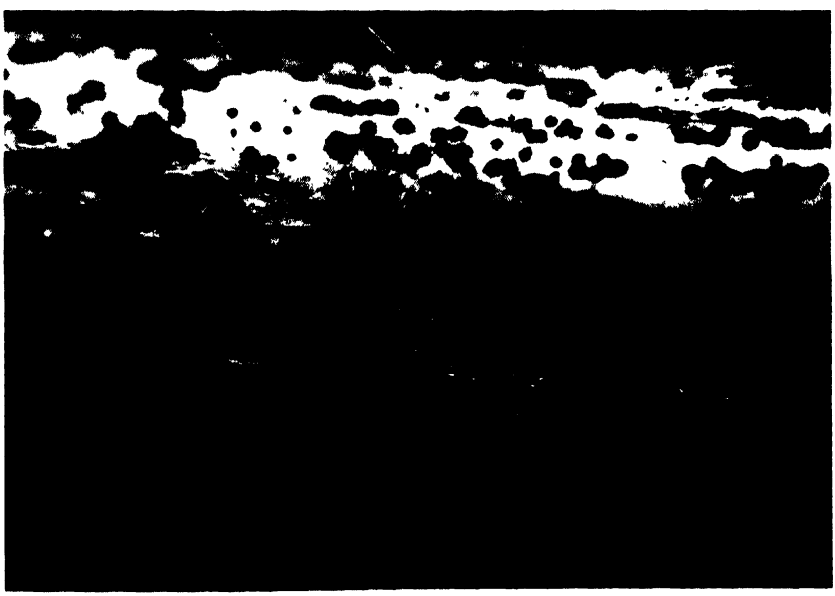

Fig. 3.4. Soyabean stem with black pycnidia of *Diaporthe phaseolorum* var. *sojae*, cause of pod and stem blight (photo: courtesy of R.F. Nyvall).

Epidemiology

The fungus overseasons as dormant mycelium in soyabeans and other host debris and in infected seeds (Kunwar *et al.*, 1985). Pycnidia are produced on petioles of the current season's abscised leaves and on crop debris, while perithecia are produced in early summer on overseasoned crop debris. The fungus colonizes plant tissues within 2 cm of the infection point until the plant begins to senesce, then spreads to tissues about 5 cm from the infection point. Progressive spread is caused by infection from conidia dispersed by splashed water. Both alpha conidia and ascospores can be splashed onto plants and initiate infection (Sinclair, 1988b).

Damage and Crop Loss

The main losses are the result of reduction in seed quality leading to grade reduction (Sinclair, 1993).

Management

Rotate with maize and plough down residues, plant high quality seeds relatively free of the pathogen, or use a fungicide seed treatment. Plant late or use late-maturing cultivars which allow for maturation during a dry period. Use a less susceptible cultivar, if available.

FUSARIUM ROOT, COLLAR AND POD ROT

Causal Pathogens

Fusarium oxysporum Schlecht.: Fr, and *Fusarium pallidoroseum* (Cooke.) Sacc. are classified in the subdivision *Deuteromycotina*, class *Hyphomycetes*. *F. oxysporum* is in the section *Elegans* and *F. pallidoroseum* is placed in the section *Arthrosporiella*. Other fusaria cause sudden death syndrome and fusarium wilt (see Table 3.2). Root, collar and pod rots and wilt caused by *Fusarium* spp. are common and serious diseases of legumes, reviewed in Chapters 6, 8, 9, 10 and 12, this volume.

Biology

Both species are extremely variable, have wide host ranges, and are made up of form-species and physiologic races. In general, *F. oxysporum* in culture is white or tinged with purple, floccose, septate, sometimes forming aerial hyphae; the underside is colourless, dark blue or purple. Conidiophores are branched or unbranched monophialides, usually short and branched and dolioform.

Sporodochia are cream, tan or orange. Chlamydospores are intercalary or terminal and formed singly or in pairs. Microconidia are single-celled, oval to kidney-shaped, and produced in false heads. Microconidia are hyaline, three- to five-septate, with a sickle-shaped basal cell and an attenuated apical cell. *F. pallidoroseum* produces either white mycelium or a peonnotal colony in culture. Aerial mycelium is tan to brown. Sporodochia, if present, are orange. The agar reverse varies from peach, tan to brown. Conidiophores are unbranched or branched mono- and polyphialides. Chlamydospores are intercalary or terminal. Microconidia are spindle-shaped and borne on aerial mycelium, with a papilla at the basal cell. Those borne in sporodochia are slightly curved with a foot-shaped basal cell. Microconidia are rare.

Symptoms

The diseases caused by *Fusarium* occur wherever soyabeans are grown and often are associated with other pathogens or stress conditions. Fusarium root rot usually appears on seedlings and young plants in cool weather (14°C). Seedling emergence may be delayed and affected plants are stunted and weak. Symptoms, which are confined to the roots and lower stems, consist of a dark discoloration of the cortex, but the vascular system may be invaded in advanced stages of root rot (Farias and Griffin, 1990) (Fig. 3.5). Pod and collar rot is characterized by depressed, water-soaked, cream-coloured lesions on cotyledons and hypocotyls of emerging seedlings. After emergence, these lesions turn dark brown to black.

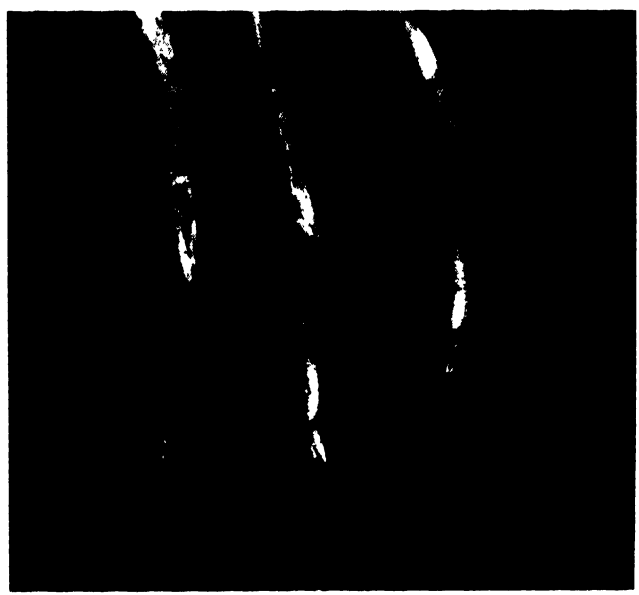

Fig. 3.5. Root rot lesions on soyabean seedlings caused by *Fusarium* (photo: courtesy of J.M. Dunleavy).

Lesions may girdle the young stem. Infected pods dry prematurely and seeds turn dark brown to black.

Epidemiology

Both fungi are soil inhabitors and overseason as chlamydospores or mycelium, which are sources of primary inoculum. Seedborne mycelium has been reported and can reduce seed germination. Host penetration is either direct or indirect. Initial penetration is followed by intercellular growth of hyphae in the cortex, later the xylem may become invaded. *F. oxysporum* is most destructive when the soil is saturated with water at 14–23°C. *F. pallidoroseum* seems most destructive when conditions are warm and dry. Infestation with the soyabean cyst nematode (*Heterodera glycines* Ichinohe), a root-knot nematode (*Meloidogyne incognita* (Kofoid & White) Chitwood), or a sting nematode (*Belonolaimus longicaudatus* Rau) predisposes seedlings and young soyabean plants to infection by *F. oxysporum*, as do dinitroaniline herbicides. *F. oxysporum* interacts with *Rhizoctonia solani* Kühn in causing a root rot of soyabeans (Summer and Minton, 1987; Datnoff and Sinclair, 1988).

Damage and Crop Loss

Losses of over 50% have been reported from severe infection by *F. oxysporum*. Data on economic losses caused by *F. pallidoroseum* are not available, though germination can be decreased by as much as 40%.

Management

Plant high quality seeds of cultivars resistant or tolerant to *Fusarium* and soyabean cyst and root-knot nematodes in warm, well-drained soil. Avoid cultivation until soil moisture is adequate. In a field with a history of these pathogens, ridge soil around the base of the plants to promote adventitious root formation.

CHARCOAL ROT

Causal Pathogen

Macrophomina phaseolina (Tassi) Goidanich. The fungus is classified in the subdivision *Deuteromycotina* and class *Coelomycetes*.

Biology

Charcoal rot, also called ashy stem blight, occurs worldwide and appears in irrigated soyabeans along with accelerated maturity when water is withheld after

flowering. The pycnidial stage is not common on soyabeans. Pycnidia, which are initially immersed in host tissues, are erumpent at maturity, and more or less globose; they are membranous or subcarbonaceous, dark greyish, becoming black with age, generally measuring 200–300 µm in diameter. On artificial media, sclerotia range from 75 to 150 µm in diameter. The optimal temperature for growth ranges from 28 to 35°C. Several selective media have been developed. Colonies are at first white to brown and become darker with age. Hyphal branches generally arise at right angles and may be constricted at the point of the union with the parent hypha. The fungus has an extensive host range and is highly variable in culture characteristics as well as in pathogenicity.

Symptoms

Infected seedlings show a reddish-brown discoloration at the emerging portion of the hypocotyl. If infection occurs through the roots, discoloration is evident at or just above the soil line. In older plants, charcoal rot usually appears after mid-season. Infected plants at first produce leaves smaller than normal, and there is a subtle loss of vigour. In advanced stages, leaves turn yellow and wilt but remain attached. After flowering a light grey or silvery discoloration of the epidermal and subepidermal tissues develops in the taproot and the lower part of the stem (Fig. 3.6). Microsclerotia form beneath the epidermis, resembling powdered charcoal. When the pathogen is seedborne, infected seeds may be symptomless; sometimes microsclerotia form in the cracks of colonized seed coats (Kunwar *et al.*, 1986b).

Epidemiology

M. phaseolina is seedborne in soyabeans and survives as microsclerotia, in soil or in crop or weed host debris (Mihail, 1989). The fungus competes well when soil nutrient levels are low and temperatures are above 30°C (Collins *et al.*, 1991). Microsclerotia germinate on root surfaces and penetration is either direct or indirect. Pathogen growth can occur early in the season and the rate of infection increases with higher soil temperatures. Low soil moisture enhances disease severity (Olaya and Abawi, 1995). Plants become infected at any time during the growing season, but disease development may be delayed until the plants are under stress (Sinclair, 1991).

Damage and Crop Loss

When severe, the disease reduces yield and seed quality through debilitation of the host.

Fig. 3.6. Charcoal rot on soyabean stem (left) and roots (right), caused by *Macrophomina phaseolina*, showing microsclerotia exposed after removal of the epidermis (photo courtesy of the US Department of Agriculture)

Management

In severely infested fields, rotate with comparatively poor hosts like cotton, for 1 or 2 years; with sorghum or maize, rotation must be extended to 3 years. Avoid excessive seeding rates: crowding produces weakened seedlings which are more vulnerable to fungal attack. Fertilize soyabeans to encourage vigorous growth, and irrigate, where possible, to keep soil moisture high, or flood fields for 3 to 4 weeks before planting. Some field-tolerant cultivars have been reported (Carvil and Smith, 1995; Wrather and Anand, 1995).

RUST

Causal Pathogens

Soyabean rust has long been regarded as caused by *Phakopsora pachyrhizi* H. Sydow & Sydow, a fungus with a wide host range including tropical pasture legumes (see Lenné, Chapter 13, this volume) and numerous synonyms (Allen, 1983; Sinclair and Backman, 1989). Recent work (Ono *et al.*, 1992) has shown that the fungus is better delineated into two distinct species, on the basis of morphological differences between their anamorphs and teleomorphs. Thus, *P. pachyrhizi* includes the Austro-Asian populations whose telia are irregularly two to seven spore layered and whose teliospores have walls that are pale yellowish-brown to colourless. *Malupa sojae* (P. Hennings) Ono, Buritica & Hennen is its uredial anamorph. *Phakopsora meibomiae* (Arthur) Arthur includes the New World populations whose telia are irregularly one to four spore layered and whose teliospores have walls that are cinnamon to light chestnut brown. *Malupa vignae* (Bresadola) Ono, Buritica & Hennen is its uredial anamorph.

Biology

P. pachyrhizi occurs on 34 natural hosts (and 61 inoculated hosts) in Australia, Burma, Cambodia, India, Indonesia, Japan, Malaysia, Nepal, Papua New Guinea, People's Republic of China, the Philippines, Taiwan, Thailand, and Vietnam. In Africa, the fungus is recorded from Ghana, Nigeria, Sierra Leone, Tanzania, Uganda, Zaire and Zambia, and elsewhere in the former USSR (Singh & Allen, 1979; Ono *et al.*, 1992; see Lenné, Chapter 13, this volume) and in the USA in Hawaii (Killgore, 1996). *P. meibomiae* occurs on 41 natural hosts (and 25 inoculated hosts) in the New World: Barbados, Belize, Bolivia, Brazil, Chile, Colombia, Costa Rica, the Dominican Republic, Ecuador, Guatemala, Mexico, Puerto Rico, St Thomas, Trinidad and Tobago, and Venezuela (see Lenné, Chapter 13, this volume). *P. pachyrhizi* is considered more aggressive than *P. meibomiae*. *Phakopsora* is classified in the subdivision *Basidiomycotina*, class *Urediniomycetes*, and order *Uredinales* (Ono *et al.*, 1992).

Soyabean rust is found extensively in areas of the eastern and western hemispheres; in the eastern hemisphere from Japan to Australia, westward to India, and the People's Republic of China. In the western hemisphere, soyabean rust occurs generally throughout Latin America and the Caribbean. The anamorph fruiting structures have a cellular basal peridium terminating in paraphyses. *P. meibomiae* teliospores are angularly subglobose, oblong to elliptical, and more or less regularly layered in rows or irregularly arranged, 12–26 × 6–12 μm, walls uniformly 1.5–2 μm thick and thickened apically to 6 μm, yellowish-brown to light chestnut brown; uredospores are ellipsoid, 16–31 × 12–24 μm, densely echinulate, colourless to pale yellowish-brown. *P. pachyrhizi* teliospores are one-celled, irregularly arranged, angularly subglobose, oblong to ellipsoid and measuring 15–26 × 6–12 μm, with walls uniformly 1 μm thick and slightly thickened apically, colourless to pale yellowish-brown. Uredospores are obovoid

to broadly elliptical, measuring 18–34 × 15–24 μm. Walls are 1 μm thick, minutely and densely echinulate, colourless to pale yellowish-brown (Ono *et al.*, 1992).

Symptoms

The most common symptom is the development of tan to dark brown or reddish-brown lesions with one to many erumpent, globose uredia, particularly on the underside of leaflets (Fig. 3.7). Lesions tend to be angular, restricted by leaf veins, reaching a size of 2–5 mm^2. Lesions may appear on petioles, pods, and stems. Telia form subepidermally among uredia and are dark brown to black at maturity. They are crustose, irregular to round, sparse to aggregated and about 150–150 μm in diameter.

Epidemiology

Rust epidemics are most severe when the mean daily temperature is less than 28°C, with long periods of leaf wetness occurring throughout the growing season. Free water is necessary for uredospore germination and penetration which take place over a temperature range of 8–28°C. Uredia appear about 9–10 days after infection, and uredospores are produced at 3 weeks. Uredospores are the primary means of disease spread. The role of individual hosts in the overseasoning of the pathogens is not known. Soyabeans are susceptible at any stage of

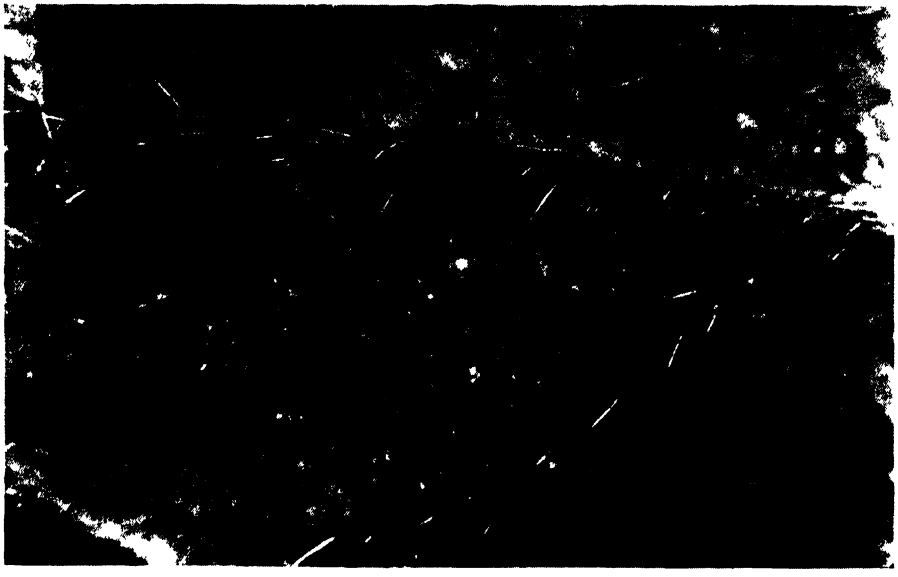

Fig. 3.7. Lower surface of a soyabean leaflet with rust lesions and pustules, caused by *Phakopsora pachyrhizi* (photo: courtesy of K.R. Bromfield).

development, but symptoms usually appear mid- to late-season because of the requirements of a prolonged wet, cool period for infection and sporulation. Spread of uredospores is by wind-blown rain. The pathogens are not seedborne in soyabeans.

Damage and Crop Loss

Significant losses have been reported only in the eastern hemisphere, 10–40% in Thailand, 10–90% in India, 10–50% in southern China, 23–90% in Taiwan, and 40% in Japan. Nearly complete losses can occur in limited areas in most of these countries. Losses are due to reduced seed weight, and fewer pods and seeds (AVRDC, 1992).

Management

Resistance has been identified in at least 11 soyabean cultivars and strains (Bromfield, 1984), and in wild *Glycine* spp. from Australia (Schoen *et al.*, 1992). However, because of the recent recognition of two distinct species within which there exists physiologic specialization, cultivars must be screened carefully. Certain fungicides can reduce rust damage. However, since frequent application is required, often before symptoms appear, it may not be cost effective.

PHYTOPHTHORA ROOT AND STEM ROT

Causal Pathogen

The causal fungus has been considered for some years to be *Phytophthora megasperma* Drechs. f. sp. *glycinea* Kuan & Erwin (1980), but more recent work (Hansen and Maxwell, 1991) suggests that the pathogen should be named *Phytophthora sojae* M.J. Kaufmann & J.W. Gerdermann. This fungus belongs to the subdivision *Mastigomycotina*, class *Oomyces* and order *Peronosporales*.

Biology

The disease was first described in the USA and has been reported in Australia, Canada, Hungary, Italy, Japan, and the former USSR. The fungus is pathogenic to soyabeans and three *Lupinus* spp. At least 25 physiologic races have been described (Schmitthenner *et al.*, 1994). The disease has been misidentified as water damage since it is usually associated with poorly drained soils. Isolates vary in cultural characteristics and morphology. Mycelium is coenocytic when young, septate with age, branching at right angles with a slight constriction at the base of each branch. Hyphae are 3–9 μm wide. The optimum for growth is 25–28°C. Sporangiophores are simple and indeterminate. Conidia (terminal

sporangia) are obpyriform, non-papillate, 65 × 32–53 μm. Sporangia may germinate directly functioning as a conidium, or indirectly by forming zoospores. The optimum for zoospore production is 20°C. Zoospores are ovoid, bluntly pointed at one or both ends, and flattened on the sides with two flagella, one directed anteriorly and the other, four to five times longer, directed posteriorly. Zoospores encyst, germinate directly, and form an appressorium for direct penetration of host tissues. The sexual antheridia and oogonial form, fertilization takes place and the resulting oospores have a thick, smooth inner and outer walls. Oospores germinate directly after about 30 days. The optimum for oospore formation and germination is 24°C.

Symptoms

The disease develops at any time during the growing season. The pathogen causes a seed rot, pre- and post-emergence damping-off, and root and stem rot of older plants. Symptoms depend upon cultivar susceptibility. In low-tolerant cultivars at the primary leaf stage, affected stems may appear water-soaked, primary leaves turn yellow and wilt, and affected seedlings may die (Fig. 3.8). On highly

Fig. 3.8. Soyabean plants with advanced symptoms of Phytophthora root and stem rot, caused by *Phytophthora sojae* (photo: courtesy of M. Ferguson).

tolerant cultivars damage may be restricted to roots, and seedlings are only stunted (Bhattacharya and Ward, 1986). Older plants of slightly tolerant cultivars are killed gradually; lower leaves develop an interveinal yellowing followed by chlorosis of the upper leaves. The plant wilts and wilted leaves remain attached to the plant. Wilted plants usually appear in groups. Roots of affected plants are dark brown, and the discoloration progresses up the stem involving the cortex and vascular system. In older plants of highly tolerant cultivars, symptoms generally are confined to the roots, plants are not killed but may be stunted and slightly chlorotic (Wagner *et al.*, 1993). Lesions occasionally form on the lower stems.

Epidemiology

Phytophthora rot is most common in heavy, tightly compacted, clay soils subject to flooding. Primary inoculum comes from oospores which survive in soil or crop residues. Oospores germinate at suitable temperatures and form sporangia which release zoospores. Zoospores are attracted to soyabean roots where they encyst and germinate. After penetration, hyphae grow intercellularly and produce globular or finger-like haustoria within the cells. Oogonia and oospores are formed in infected root and stem tissues of all cultivars, with many more being formed in susceptible than in resistant ones. The association and interaction of *P. sojae* with mycorrhizal fungi may decrease disease severity, while dense populations of *Fusarium* spp., *Pythium* spp. or *Rhizoctonia solani* may increase the disease. Infection by the nematode *Meloidogyne hapla* increases severity of root rot.

Damage and Crop Loss

Plant losses and yield reductions range from 40% in highly tolerant cultivars to 100% in susceptible ones. Disease severity depends on cultivar susceptibility, rainfall, drainage, soil type and tillage practices.

Management

Use race-specific resistant cultivars, noting that a cultivar need not be resistant to all races. Combine optimal cultural conditions for integrated disease management of highly tolerant cultivars, good drainage, autumn or spring ploughing and crop rotation. Apply metalaxyl fungicide seed treatment for highly tolerant cultivars for control of damping-off. Metalxyl may also be applied in the seed furrow at time of planting. The fungicide leaches into the root zone and is taken up by roots before moving upward into the plant. *Phytophthora* cannot colonize roots containing metalaxyl at the appropriate concentrations (Schmitthenner and van Doren, 1985; Lamboy and Paxton, 1992).

PYTHIUM ROOT AND SEEDLING ROT

Causal Pathogens

Pythium aphanidermatum (Edson) Fitzp., *P. debaryanum* Hesse and *P. ultimum* Trow. are classified in the subdivision *Mastigomycotina*, class *Oomycetes* and order *Peronosporales*. These species also cause seedling diseases of many other legumes, and diseases caused by *P. ultimum* on pea and chickpea are reviewed by Kraft *et al.* Chapter 6 and Haware, Chapter 9, this volume, respectively.

Biology

Pythium rot is primarily a seedling disease but the pathogens can cause seed decay, pre- and post-emergence damping-off, and root rot. These soil pathogens are cosmopolitan and attack a wide range of crop plants. They grow well on sugar-rich media, produce sporangia on coenocytic hyphae, have biciliate zoospores and possess oogonia which are spherical and smooth-walled. The hyphae of *P. aphanidermatum* are 2–8 μm wide. Sporangia are filamentous, branched or unbranched, and produced freely. Oogonia are terminal; antheridia are mono- or diclinous, dome-shaped, and intercalary. Oospores are smooth, aplerotic, single, and moderately thick-walled. The hyphae of *P. debaryanum* usually are 5 μm in diameter. Sporangia are spherical to oval and both terminal and intercalary. Oogonia are smooth, terminal or intercalary, and usually spherical. Antheridia are monoclinous or diclinous. Oospores are smooth and aplerotic and germinate directly. The hyphae of *P. ultimum* are 1.7–6.5 μm thick. Sporangia are mostly terminal and spherical and germinate by one or more germ tubes. Zoospores are formed rarely. Oogonia are mostly terminal. Antheridia are monoclinous and arise first below the oogonium. Oospores are aplerotic, single, spherical, smooth and thick-walled.

Symptoms

Soyabean seedlings infected with either *P. debaryanum* or *P. ultimum* may develop different symptoms. *P. aphanidermatum* causes a root rot resembling those caused by other *Pythium* spp. Infection by *Pythium* in nature is often followed by other microorganisms which mask typical symptoms. Generally, wet rot symptoms develop in seedings infected with *P. ultimum*, whereas retarded development of the growing point of seedlings infected with *P. debaryanum* is characteristic (Fig. 3.9). In cold, wet soil both species cause a seed rot and pre-emergence damping-off. Seedlings infected with *P. ultimum* generally fail to emerge, but if attacked at the root tip, they will survive. Recently invaded stem tissues are translucent; older lesions become brown. Cortical tissues may disintegrate and slough off. Smaller roots decay and break away. Seedlings infected with *P. debaryanum* develop small, black, dry, sunken, lesions on the cotyledons. Apical meristems

Fig. 3.9. Soyabean seedlings wilting and dying in the field from Pythium root rot, caused by *Pythium* (photo courtesy of H J Walters)

may be severely stunted. Hypocotyls may swell to two to three times their normal size, symptoms which may be confused with herbicide damage. Axillary buds may develop at the cotyledonary node.

Epidemiology

All species are soil inhabitants and subsist as saprophytes, colonizing the residues of many crops. They overseason as oospores. Low soil temperatures (10–15°C) are most favourable for damping-off caused by *P. debaryanum* and *P. ultimum* (Griffin, 1990). Damping-off decreases at soil temperatures above 15°C, and the number of damped-off seedlings declines sharply above 22°C. Seedlings up to 10 days old are more susceptible to damping-off than older plants (Schlub and Lockwood, 1981). Infection by *P. aphanidermatum* occurs between 25 and 36°C. Hyphae enter the host directly and spread intercellularly within the cortex and endodermis, ultimately invading the stele. In seeds, hyphae penetrate the seed coat and spread extensively in the cotyledons.

Infection by *Pythium* commonly occurs at high soil moistures. Under these conditions, not only is the oxygen content of the soil low, but water hinders uptake of oxygen by soyabean seeds.

Damage and Crop Loss

Only scattered individual plants or small groups of plants are killed, and although stands may be reduced, the disease usually does not cause economic losses alone.

Management

Plant good quality seed, free of cracks and capable of at least 85% germination in warm (above 19°C), well-drained, fertile soil that is well prepared. Where possible, plough under weeds or cover crops several weeks before planting. Rotate with less susceptible crops such as maize (Zhang and Yang, 1995). Avoid excessive irrigation during the first 10 days after planting. Where the disease persists year after year, use metalaxyl fungicides either as a seed treatment or applied in the furrow.

RHIZOCTONIA DISEASES

Causal Pathogen

Rhizoctonia solani Kühn (teleomorph *Thanatephorus cucumeris* (Frank) Donk.) *T. cucumeris* is in the subdivision *Basidiomycotina*, class *Hymenomycetes* and order *Aphyllophorales*. Most soyabean pathogenic isolates are members of anastomosis group AG-4, but some have been shown to belong also to groups AG-1, AG-2-I, AG-2-II, AG-3 and AG-5 (Liu and Sinclair, 1991). Several ungrouped isolates are also pathogenic to soyabeans. The pathogen has a wide host range including crop (e.g. cowpea, see Allen *et al.*, Chapter 5, this volume), pasture (see Lenné, Chapter 13, this volume) and weed host species. Binucleate *Rhizoctonia* spp. affect lupins causing similar diseases (see Hill, Chapter 11, this volume).

Biology

Rhizoctonia diseases, including pre- and post-emergence damping-off, root and stem decay, and web (or aerial) blight, have been reported in soyabean growing areas worldwide. The diseases occur at any time during the season when conditions are favourable. Isolates of *R. solani* are ecologically specific, differ morphologically, and are highly variable in cultural characteristics, pathogenicity, responses to environmental changes and DNA polymorphism (Liu and Sinclair, 1993; Liu *et al.*, 1995). Isolates that cause root and stem decay may not cause aerial blight. Colonies are fast-growing in culture, at first colourless, submerged or with some radiating aerial hyphae, rapidly becoming brown. Hyphae are subhyaline to pale brown, thick-walled cells about 100–150 μm long and 5–17 μm wide, often constricted near septa and where branching, with conspicuous dolipore septa, lacking clamp connections branched widely at angles. Hyphal

cells contain 2 to 28 nuclei. Some isolates produce solitary sclerotia, 1 mm in diameter, some turning brown. Basidia are resupinate, creamy, effuse and loosely attached, arising in asymmetrical cymes or racemes from tufts of ascending hyphae, variable in shape, barrel-shaped to cylindrical, measuring 10–25 × 6–12 μm with two to seven stout, straight sterigmata as long or longer than the metabasidia. Basidiospores are oblong to broadly ellipsoid, unilaterally flattened, apiculate at the base, hyaline, smooth-walled and 6–14 × 4–8 μm in size.

Symptoms

Pre-emergence blight occurs immediately after the plumule emerges. Damping-off can occur a few days after emergence, with lesions appearing at the base of seedling stems and on roots below the soil line (Fig. 3.10). These may enlarge into a sunken lesion, girding the stem. Affected plants may show wilting during the heat of the day. Young lesions are brown, dark brown, or reddish-brown. Seedlings that survive initial infection may develop reddish-brown cortical decay above the crown. The discoloration may extend into the pith of stems and roots. Decay may continue throughout the growing season with continuing death of plants. Symptoms of aerial blight on leaves, stems and pods usually begin mid-season on the lower or middle parts of infected plants and move upward (Yang *et al.*, 1990). Infected leaves are first water-soaked, then take on a greenish-brown to reddish-brown cast and later turn dark. Infected leaves drop and adhere to

Fig. 3.10. Soyabean roots, crowns and stems with symptoms of Rhizoctonia root rot, caused by *Rhizoctonia solani* (photo: courtesy of P.S. Lehman and C.C. Machado).

pods and stems below. A brownish web of fungus hyphae may form over affected plant parts (Yang *et al.*, 1991).

Epidemiology

The fungus colonizes all types of plant debris, and can overseason in soil in the absence of crop tissue. The extent of saprophytism varies among isolates. When environmental conditions are favourable for disease development, disease severity is related directly to inoculum potential. Generally, disease is most severe under conditions of high moisture and moderate to warm temperatures.

Damage and Crop Loss

Yield losses up to 35% have been attributed to the aerial web blight phase.

Management

Rhizocotonia diseases are difficult to control because of the unpredictability of their occurrence in any one field or in any one season. If rhizocotonia seedling disease is a chronic problem, ensure that there is good soil drainage, and preferably use less susceptible or tolerant cultivars (Muyolo *et al.*, 1993). Foliar application of systemic fungicides appears promising for control of the web blight phase. Use a fungicide seed treatment for both types of disease.

SCLEROTINIA STEM ROT

Causal Pathogen

Sclerotinia sclerotiorum (Lib.) de Bary is classified in the subdivision *Ascomycotina*, class *Discomycetes*, and order *Helotiales*.

Biology

The disease, which is also sometimes called white mould, is known worldwide and usually occurs locally in individual fields. The disease is most serious on soyabean when planted after highly susceptible crops such as sunflowers, and cruciferous or leguminous vegetable crops. Its effects on pea and faba bean are reviewed in this volume (see Kraff *et al.*, Chapter 6, and Jellis *et al.*, Chapter 7, this volume). One hazard associated with this disease is contamination of soyabean seed lots with sclerotia. A few sclerotia can infest a large number of seeds and may be grounds for rejection of an entire shipment. The large, black sclerotia range in size from 2 to 20 mm in diameter and germinate either directly or indirectly. Indirect germination results in the production of one to many

cup-shaped apothecia borne on slender stalks. The apothecia are 0.5–2.0 mm or more in diameter, funnel-shaped to discoid, and light tan to brown in colour. The hymenium is composed of closely packed, eight-spored asci interspersed with slender, simple, hyaline paraphyses. The asci are narrow and cylindrical or cylindrical–clavate, and measure 81–252 × 4–22 μm. The pathogen has a large host range including many crop and broadleaf weed hosts (Grau, 1988).

Symptoms

Generally, the first symptoms are the wilting and the eventual death of upper leaves of infected plants in the reproductive stage. Leaves become greyish-green, necrotic and turn brown, remaining attached to the stem. Diagnostic symptoms develop at first above the soil line and up to 50 cm above it. Water-soaked lesions develop at the nodes and change from tan to white, then girdle the stem with side branches and pods becoming infected. Cottony mycelial growth on all diseased parts is characteristic. The most conspicuous sign of the disease is the formation on and within stems and pods of large, black, round to irregularly-shaped sclerotia that are partially covered with white mycelium (Fig. 3.11). Seeds may become infected within diseased pods, appearing flattened and shrivelled.

Epidemiology

The fungus survives for long periods in soil as sclerotia which are highly resistant to fungicides as well as to dry heat and prolonged freezing and thawing. Those within 5 cm of the soil surface germinate by producing apothecia. Prolonged periods of soil temperatures in the range of 5–15°C and high soil moisture (−0.25 bar) for 10–14 days are favourable for apothecial production. Ascospores are forcibly discharged from asci and carried by wind to other plants where infection occurs if relative humidity is high. Infection may occur by direct or by indirect penetration. The disease usually develops during periods of low rainfall when the crop is planted at a narrow row spacing of less than 38 cm (Boland and Hall, 1988). Seed contamination by sclerotia is the most likely means of introducing the pathogen into new areas.

Damage and Crop Loss

This disease has become of increasing importance in soyabean fields rotated with other legumes or with sunflowers. Losses in individual fields range from trace levels to as much as 15%. Pods above a stem lesion are reduced in number and size.

Management

Soyabean cultivars range from moderately resistant to highly susceptible, varying with plant architecture, maturity and lodging characteristics (Chun *et al.*,

Fig. 3.11. Soyabean stem with external symptoms of sclerotinia stem decay (centre), with internal sclerotia in a split stem (right), and extracted sclerotia (left), caused by *Sclerotinia sclerotiorum* (photo: courtesy of M.C. Shurtleff).

1987; Nelson *et al.*, 1991). The expression of resistance is altered by cultural practices, such as row width, plant population and irrigation applied at flowering. Alternating soyabeans with non-host crops such as maize, grain sorghum or pasture grasses, or clean fallowing for 2 years, may prevent inoculum build-up. In fields heavily infested with *S. sclerotiorum*, a 3- to 4-year rotation with non-host crops may be needed. Burying crop debris bearing sclerotia at a depth of 15–25 cm with a mouldboard plough may reduce inoculum carry-over and delay disease development. Movement of soil during cultivation around soyabean stems may increase disease incidence.

SEPTORIA BROWN SPOT

Causal Pathogen

Septoria glycines Hemmi (teleomorph *Mycosphaerella uspenskajae* Mashk. & Tomil.). *M. uspenskajae* is classified in the subdivision *Ascomycotina*, class *Loculoascomyetes*, and order *Dothideales*. *Septoria sojae* Syd. & Butl. and *S. sojina* Thum. are considered less important.

Biology

Brown spot occurs worldwide particularly in temperate regions, or in subtropical and tropical areas where mild temperatures and abundant moisture prevails. The fungus has a relatively narrow host range and is variable (Kamicker and Lim, 1985).

Symptoms

Brown spot is primarily a foliar disease although stems, pods and seeds of maturing plants can also be infected. Irregular dark brown spots, varying in size up to 4 mm in diameter, appear on both upper and lower leaf surfaces (Fig. 3.12). Infected leaves quickly turn yellow and drop. Adjacent lesions may coalesce to form blotches. The disease usually progresses from lower to upper leaves. Symptoms on other plant parts are not sufficiently distinct to be diagnostic. Symptom production is due in part to a pathotoxin (Song *et al.*, 1993). The pathogen colonizes soyabean seed coats without producing conspicuous symptoms. The pycnidia, which are the most conspicuous fruiting structure in the field, form in the dead tissues of old lesions and are globose to conical–globose and generally open to the upper surface. Those formed in stems are flattened. They are embedded in the substrate and open to the surface with a large ostiole. Pycnidial walls are membranous and thin. Conidia are hyaline, filiform, curved and measure 21–50 × 1.4–2 μm. They are distinctly one- to three-septate, with septation becoming noticeable at germination. Conidia readily germinate in free water or on leaf surfaces. Mature hyphae are thick-walled and densely branched.

Epidemiology

The primary crop inoculum arises from conidia and mycelium overseasoning in diseased seeds and crop debris. Lesions in cotyledons and unifoliolate leaves are inoculum sources. Infection and disease development are favoured by warm, moist weather which promotes sporulation on primary lesions (Schuh and Adamowicz, 1993). Conidia are spread by wind and splashing rain. The fungus enters through stomata and grows intercellularly, killing cells next to the

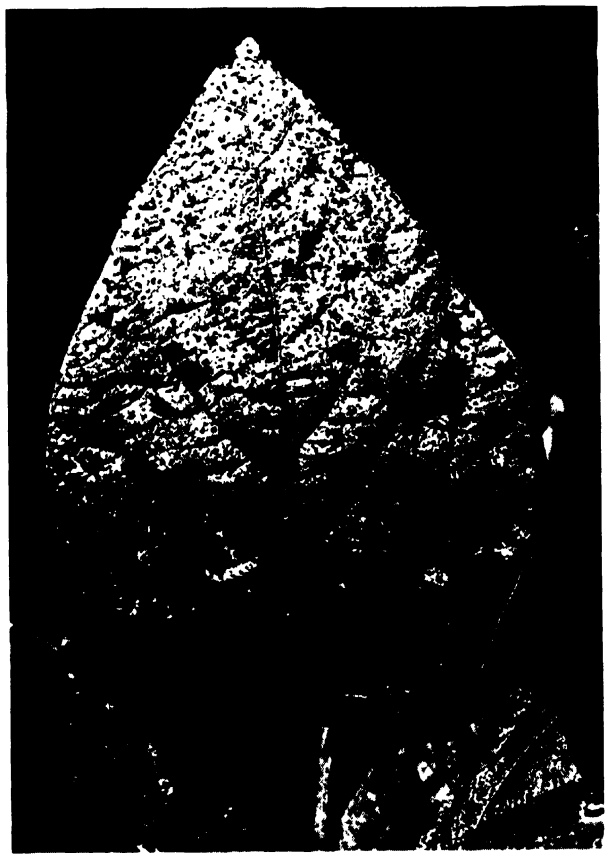

Fig. 3.12. Soyabean leaflet with leaf lesions of brown leaf spot, caused by *Septoria glycines* (photo: courtesy of R.F. Nyvall).

hyphae, or by growing through funicular and placental tissue and later invading the seed coat without producing symptoms.

Damage and Crop Loss

The disease causes premature defoliation with yield losses ranging from 8 to 15% under natural field conditions, and from 8 to 35% in inoculated field trials (Lim, 1980; Pataky and Lim, 1981). The disease is most severe when soyabeans are grown continuously in the same field.

Management

Plough under all crop residues after harvest. Rotate soyabeans with a crop that is not susceptible to the causal fungus. Plant seed of cultivars that have been shown

to be relatively free of the pathogen or are generally less susceptible. Use resistant or tolerant cultivars where available (Sebastian *et al.*, 1983; Song *et al.*, 1994).

BACTERIAL BLIGHT

Causal Pathogen

Pseudomonas syringae pv. *glycinea* (Coerper) Young, Dye & Wilkie is a motile, Gram-negative rod (1.2–1.5 × 2.3–3.3 μm) with rounded ends, and one to several polar flagella in the *Pseudomonadaceae*. Bacterial blight caused by other pathovars of this bacterium affect common bean (see Allen *et al.* Chapter 4, this volume) and pea (see Kraft *et al.*, Chapter 6, this volume).

Biology

Bacterial blight is the most common bacterial foliar disease of soyabeans, occurring wherever the crop is grown, especially during cool, wet weather. Colonies on nutrient agar are circular, smooth and glistening, with an acute margin; they are white and raised but not viscid (Basu and Butler, 1986). The optimum for growth is 24–26°C. Selective media have been developed (Alvarez *et al.*, 1995). Strains infect bean (*Phaseolus vulgaris* L.), lima bean (*P. lunatus* L.), tepary bean (*P. acutifolius* A. Gray) and cowpea (*Vigna unguiculata* (L.) Walpers). At least nine races of the bacterium have been identified (Fett and Sequeira, 1981).

Symptoms

Angular, yellow to light-brown lesions appear first on young leaflets, then enlarge to produce irregular dead tissue with yellow haloes (Fig. 3.13). These areas drop out, giving a ragged appearance, especially after strong winds and beating rains. Early defoliation of lower leaves may occur. Infected stored seeds may shrivel, develop sunken or raised lesions and become discoloured or remain symptomless.

Epidemiology

Cotyledons may be a major source of inoculum that causes secondary lesions on seedlings. The bacterium is spread during windy rainstorms and during cultivation while foliage is wet. It exists epiphytically on leaf surfaces and buds, needing only the proper temperature and wind-blown rain to enter the leaf. Cool, rainy weather favours disease development. The bacterium overseasons in crop debris on the surface as well as in seeds.

Fig. 3.13. Soyabean leaflets with symptoms of bacterial blight, caused by *Pseudomonas syringae* pv. *glyinea* (photo: courtesy of D.W. Chamberlain).

Damage and Crop Loss

Estimates of economic losses range from 5 to 18% from various world locations, but losses generally are low where resistant or tolerant cultivars are planted (Huynh *et al.*, 1989).

Management

Rotate soyabeans with non-susceptible crops and completely bury crop residues by clean ploughing before planting. Avoid planting highly susceptible cultivars and sow seeds that are relatively free of the pathogen. To prevent field spread of the pathogen, do not cultivate when the foliage is wet with dew or after rain. Antibiotic sprays have been successful in controlling bacterial blight, but cost may be prohibitive.

BEAN YELLOW MOSAIC

Causal Pathogen

Bean yellow mosaic virus (BYMV), a member of the potyvirus group, is a flexuous rod, about 750 × 12 nm. Its dilution end point is 10^{-3} to 10^{-4}, and the

thermal inactivation point varies between 50 and 62°C, depending on the strain. Longevity *in vitro* varies from 1 to 4 days at room temperature.

Biology

BYMV and the disease it causes in soyabean are found throughout Asia, in Brazil, and in certain areas of the USA and the former USSR. BYMV has a wide host range including many legumes, e.g. faba bean (see Jellis *et al.*, Chapter 7, this volume), lupins (see Hill, Chapter 11, this volume) and clovers (see Mercer, Chapter 12, this volume). It is made up of a number of strains.

Symptoms

Early symptoms include vein-clearing along the small, branching veins of young leaves. Later, a conspicuous yellowing mottling of the entire leaf develops (Fig. 3.14). Rusty, necrotic spots appear in the yellow areas as the leaves mature. Some strains produce severe mottling and crinkling of the leaves.

Fig. 3.14. Soyabean leaf with symptoms caused by bean yellow mosaic virus (photo: courtesy of J.B. Sinclair).

Epidemiology

Soyabeans are most susceptible from 3 to 9 weeks after planting and susceptibility decreases with age. The virus is sap-transmitted and more than 20 aphid species transmit the virus in a non-persistent manner. Seed-transmission has not been reported in soyabeans.

Damage and Crop Loss

Losses ranging from 15 to 75% have been reported from India due to decreased number of pods and seeds per pod, seed weight and nodulation by *Bradyrhizobium* (Dhingra and Chenulu, 1985; Dante *et al.*, 1992). Reduced oil content has been recorded in seeds from infected plants.

Management

Use resistant cultivars where available (Ram *et al.*, 1984). Insecticides have been effective in controlling transmission in India.

SOYBEAN MOSAIC

Causal Pathogen

Soybean mosaic virus (SbMV) and at least 14 closely related potyviruses from soyabean (Jain *et al.*, 1992; Qusus *et al.*, 1995).

Biology

Soybean mosaic occurs wherever soyabeans are grown. SbMV is a flexuous rod averaging 750 × 15–18 nm. However, virus particles range from 300 to 900 nm, the most infectious particles being over 656 nm long. Soybean mosaic virus virions have helical symmetry with a pitch of 34°. The nucleic acid in the particles is single-stranded RNA, constituting 5.3% of the particle mass and having a molecular weight of 3.25×10^6. Thermal inactivation points range from 55 to 70°C; longevity *in vitro* is 2 to 5 days. The virus remains infective in desiccated leaves for 7 days at 25–33°C and is most stable at pH 6 and loses infectivity below pH 4 and above pH 9. The virus can infect several host species, mostly in the *Fabaceae*. It induces systemic symptoms in soyabeans, as well as in *Canavalia ensiformis*, *Cassia occidentalis*, *Crotalaria spectabilis*, *Cyamopsis tetragonoloba*, *Dolichos falcatus*, *Lespedeza stipulacea*, *L. striata*, *Lupinus albus*, *L. luteus*, *Macroptilium lathyroides*, *Mucuna deeringianum*, *Phaseolus lunatus*, *P. nigricans*, some cultivars of *P. vulgaris*, *Sesbania exaltata*, *Trigonella caerulea* and *T. foenum-graecum*.

Latent infections occur in *Hippocrepis multisiliquosa*, *Lotus tetragonolobus*,

Lupinus angustifolius, *Phaseolus speciosus*, some cultivars of *P. vulgaris*, and *Scorpiurus sulcata*. SbMV causes local lesions on *Chenopodium album*, *C. quinoa*, *Cyamopsis tetragonoloba*, *Dolichos biflorus*, *Indigofera hirsuta*, *Lablab purpureus*, *Lourea vespertilionis*, *Macroptilium lathyroides*, *Phaseolus lunatus*, some cultivars of *P. vulgaris*, and *Vigna unguiculata*. Some isolates of SbMV cause both local lesions and systemic symptoms in *Cyamopsis tetragonoloba* and *Macroptilium lathyroides*.

SbMV also infects *Amaranthus* sp., *Physalis longifolia*, *P. virginiana*, *Setaria* sp. and *Solanum carolinense*. Several strains of the virus have been recognized, on the basis of the reactions of a differential set of soyabean cultivars (Bowers and Goodman, 1991).

Symptoms

Symptom production depends on host genotype, the virus strain, plant age, and environmental conditions. Seedlings arising from infected seeds are spindly, with rugose or crinkled unifoliolate leaves which may be mottled or curl longitudinally downward. Subsequent leaflets are chlorotic, severely stunted, mottled, and rugose (Fig. 3.15). Plants infected early in the season are stunted, have shortened petioles and internodes and often show a browning of stems and petioles. Leaves are reduced in size and generally misshapened; the youngest show the most severe symptoms (Pacumbaba, 1995). Typically, infected plants mature conspicuously later than uninfected ones, being conspicuous in the field where they remain green while most other plants have become defoliated and dried.

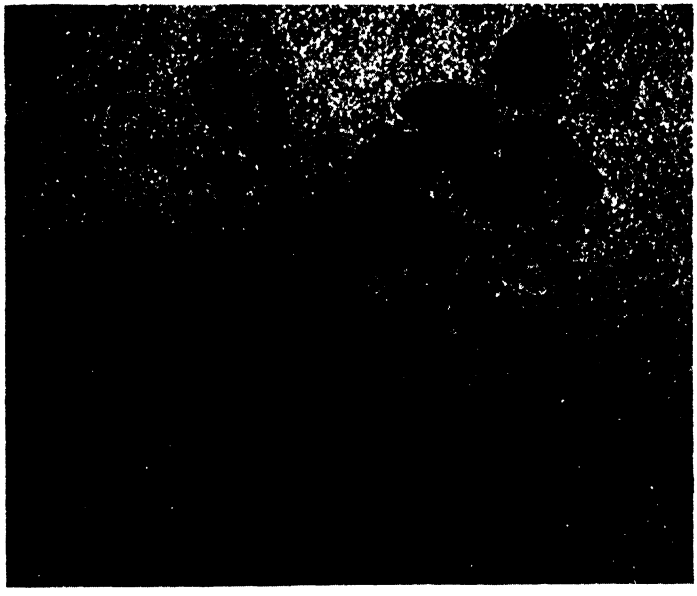

Fig. 3.15. Soyabean plant in the field with symptoms caused by soyabean mosaic virus (Photo: courtesy of G.R. Bowers, Jr.).

The pattern of infected plants in the field depends on aphid transmission. Seed from infected plants may or may not show symptoms. Yellow-seeded cultivars infected with SbMV may produce seeds that are mottled brown or black, depending upon hilum colour (Bottenberg and Irwin, 1992). The presence of symptoms does not necessarily mean the virus can be detected in seeds. Other viruses and certain environmental conditions can each cause such mottling.

Epidemiology

Seed transmission in the embryo is important in the epidemiology of soyabean mosaic, particularly in areas that lack vectors and alternative hosts of SbMV. The extent of seed transmission depends on the virus strain and host genotype. A small percentage of seeds from infected plants carry the virus for periods exceeding 2 years. The virus is sap- and graft-transmissible, but it is not transmitted by *Cuscuta*. At least 31 aphid species transmit the virus efficiently in a non-persistent manner (Gunasinghe *et al.*, 1986). The virus moves systemically in infected plants. Multiplication and movement occur most rapidly at 26°C, with no movement at 10°C.

Damage and Crop Loss

Yields may be reduced by 50%. Yield reduction of 93% has been reported in experimentally inoculated plants. Infected plants produce fewer, smaller, lighter seeds which may have mottled seed coats (Hill *et al.*, 1987). Infection of plants by the virus can predispose some cultivars to infection by *Phomopsis longicolla*, cause of Phomopsis seed decay.

Management

Control of soyabean mosaic is difficult because of the broad host range of SbMV, the number of aphid species that transmit the virus, and the importance of seed transmission. No cultivar is resistant to all strains. Cultivars vary in their resistance to the virus, seed transmission, and symptom expression (Lim, 1985). Use the most tolerant cultivar available and sow seeds relatively free of the virus (not necessarily free of the symptoms), and practise alternative-host weed control in and around soyabean fields. Controlling aphids with insecticides often increases disease incidence by promoting flights of viruliferous insects. Cross-protection by mild strains has been studied (Kosaka and Fukunishi, 1994).

BUD BLIGHT

Causal Pathogen

Tobacco ringspot virus (TRSV) and tobacco streak virus (TSV) in Brazil (see 'Brazilian bud blight'). Tobacco ringspot virus is a nepovirus, and TSV an

ilarvirus. Phytoplasma-like organisms can cause symptoms somewhat similar to those caused by TRSV and TSV.

Biology

Bud blight symptoms can be caused by different agents and the disease has been reported in all the major soyabean growing countries (Almeida, 1993). The virions are polyhedral particles, 28 to 30 nm in diameter, which can be separated into top-, middle- and bottom-sedimenting components (53, 91, and 126 S, respectively). The top component is devoid of nucleic acid. The bipartite genome consists of single-stranded RNA molecules with molecular weights of 1.4×10^6 and 2.4×10^6, encapsidated separately in the middle and bottom components, respectively. The virus is moderately immunogenic. The thermal inactivation point of TRSV is between 60 and 65°C; its dilution end point is between 10^{-4} and 10^{-5}, and longevity *in vitro* is 6–10 days at 25°C and 10 months at 2–4°C. It is sap-transmissible and is made up of many strains (Gergerich *et al.*, 1983; Tu, 1986).

The virus induces local or systemic symptoms, or both, in a wide range of plants, and some symptomless carriers also are known. The hosts among legumes, many of which act as reservoirs, include *Crotalaria intermedia*, *Cyamopsis tetragonoloba*, *Lupinus* spp., *Melilotus* spp., *Phaseolus lunatus*, *P. vulgaris*, *Pisum sativum*, *Trifolium pratense* and *Vigna unguiculata*, as well as various species in other families. Symptomless carriers include *Trifolium repens*. Several strains of the virus naturally infecting soyabeans have been reported.

Symptoms

TRSV can infect soyabeans at any time, but susceptibility decreases after blossoming begins. Plants infected while less than 5 weeks old are stunted. The stunting is not evident when grown at above 25°C. The most striking symptom is the curving of the terminal bud to form a crook. Adventitious leaf and floral buds may proliferate. Later, buds on the plant become brown, necrotic and brittle (Fig. 3.16). Petioles of the youngest trifoliolate leaves often are thickened and shortened and may be curved. Leaflets are dwarfed and tend to cup or roll, and the blades become more or less rugose and bronzed. Pods generally are severely underdeveloped or aborted. Those that set before infection often develop dark blotches, generally do not produce viable seeds, and drop early. Maturity is delayed in infected plants; they remain green until harvest, or until killed by frost. The pith and branches may show a brown discoloration, first near the nodes and then throughout the stem. Brown streaks occasionally are observed on petioles and large leaf veins. The virus reduces root and nodule growth. Nodulation is suppressed until plants are about 40 days old.

Fig. 3.16. Soyabean plant in the field with symptoms of bud blight caused by the tobacco ringspot virus (photo: courtesy of J.B. Sinclair).

Epidemiology

TRSV causes systemic infection in susceptible cultivars, moving from infected leaves to stem tips and into roots. Movement from roots to leaves is uncommon. Movement is faster at high temperatures and in long photoperiods, and movement from young leaves is greater than that from maturing leaves. Seed transmission is the most important mode of long-range dissemination and carry-over from season to season. Because all infected seeds come from plants infected before bloom, the maximum possible amount of viruliferous seeds is extremely small, barely enough to perpetuate the virus. The virus remains viable in seeds for at least 5 years. In the absence of seedborne inoculum, the disease first appears at the edge of a soyabean field and advances inward as the season progresses. The speed of spread depends on the crops and weeds next to the field and probably on an insect vector. More infection occurs in fields next to pastures, and less next to maize fields.

No efficient insect vector of the virus has been discovered. Nymphs of *Thrips*

tabaci Lind. transmit it at a low level of efficiency. A grasshopper (*Melanoplus differentialis* (Thomas)) is capable of a 2–3% transmission rate after a single feed. When feeding is extended to 30 seconds, no transmission occurs. The dagger nematode (*Xiphinema americanum* Cobb) also is an inefficient vector and the infection generally remains confined to roots.

Damage and Crop Loss

Yields may be reduced by 25–100%. In general, losses are greater when young plants are infected or when seeds with a high percentage of the virus are sown. Yields are lowered through reduced pod set and seed formation on infected plants.

Management

A few soyabean cultivars have resistance to a few strains of the virus. These should be used where available. Virus-free soyabean seeds should be used in commercial fields, and it may be desirable to avoid fields with dagger nematodes. Since the speed of spread depends on the crops and weeds next to soyabean fields and probably also on insect vector populations, locate soyabean fields next to maize fields rather than pastures. There is promise of resistance from plant introductions (Orellana, 1981).

BRAZILIAN BUD BLIGHT

Causal Pathogen

Tobacco streak virus (TSV), a member of the ilarvirus group (see 'Bud blight').

Biology

Bud blight caused by TSV occurs in Brazil (Almeida *et al.*, 1994), the USA, and probably elsewhere, because the disease is indistinguishable in the field from bud blight caused by tobacco ringspot virus (see 'Bud blight'). It has isometric particles ranging from 25 to 30 nm in diameter. The particles separate into three or four sedimenting components, with sedimentation coefficients ranging from 78 to 114 S. Particles contain four molecules of positive-sense, single-stranded RNA ranging in molecular weight from 0.3×10^6 to 1.04×10^6. The three largest RNAs are genomic, but they require either coat protein or the smallest (subgenomic) RNA for infectivity. The virus is seed- and sap-transmitted, but is readily inactivated if extracted in water. The extent of seed transmission depends on the virulence of the virus strain, susceptibility of the soyabean cultivar, and earliness of infection (Fetzer *et al.*, 1988; Almeida and Corso, 1991). In early-infected

susceptible cultivars, seed transmission may reach 30%. There is indirect evidence suggesting transmission by thrips, *Caliothrips phaseoli* (Hood).

The virus has a wide host range, including groundnut. Many strains of TSV are known (Ghanekar and Schwenk, 1980), and cross-protection between strains is common. The thermal inactivation point at pH 7 is between 55 and 60°C in phosphate buffer and in sodium sulphite solution; dilution end point is about 10^{-1} in sap and is between 10^{-5} and 10^{-6} if the virus is extracted with phosphate buffer and sodium sulphite. A dilution end point of 1:640 was reported in phosphate buffer. The virus is inactivated within 20 minutes in crude sap and within 1 h if extracted with water; infectivity is retained for up to 9 h if extracted with phosphate buffer and sodium sulphite.

Symptoms

Symptoms usually are not seen on young plants. Irregular, yellow spots form later on leaves, followed by systemic symptoms. The virus moves rapidly in soyabeans from roots to aerial parts and from leaves to shoots and roots. Infected plants tend to recover and then develop supernumerary axillary branches which are stunted and produce dwarfed leaves (Fig. 3.17). Mosaic symptoms and necrotic streaks may develop at nodes. Necrotic blotches appear on pods. TSV can be recovered from all parts of the plant except pollen. Virus concentration increases in young developing leaves and declines as they mature.

Damage and Crop Loss

Early infected plants produce fewer pods and seeds. Infection at any age delays seed maturation.

Management

Resistance in soyabean to TSV has not been reported. Therefore, control measures should emphasize protection from introducing virus-infected seeds into soyabean fields. In areas where the virus causes significant problems in soyabean performance, the production of virus-free seeds for planting should prove useful.

SOYBEAN CYST NEMATODE

Causal Pathogen

Soybean cyst nematode is *Heterodera glycines* Ichinohe which belongs to the order *Tylenchida*, suborder *Tylenchina*, superfamily *Tylenchoidea*, and family *Heteroderidae*.

Fig. 3.17. Soyabean plant with symptoms of Brazilian bud blight caused by tobacco streak virus (photo: courtesy of A.S. Costa).

Biology

The nematode and the disease occur in the People's Republic of China, Colombia, Indonesia, Japan, Korea, the former USSR and at least 26 states of the USA. *H. glycinea* has an egg stage, four juveniles stages, and an adult stage. First-stage juveniles develop within the egg and moult once to become second-stage juveniles, which emerge from the egg. Second-stage juveniles penetrate roots approximately 1.0 cm or more behind the root tip from where they migrate to the vascular tissue; when feeding begins, the nematodes begin to enlarge and become sedentary. Three more moults occur, resulting in third- and fourth-stage juveniles and adults. Males mature faster than females. Development occurs at temperatures of 18–32°C; 24–28°C is optimal. Development does not occur above 33°C and is slow below 16°C. At least 16 races have been described (Schmitt *et al.*, 1987).

Symptoms

Foliar symptoms on seedlings vary from a slight stunting to severe chlorosis and death. Mature plants may be stunted or chlorotic, or both. These symptoms, however, are not diagnostic, because nitrogen and potassium deficiencies may cause similar symptoms. The root system has symptoms ranging from slight discoloration to severe necrosis. Diagnosis must be based on the white to yellow females (cysts), which erupt from the roots (Fig. 3.18). Some populations of the nematode, especially race 1, also affect nitrogen fixation. Nodulation may be slightly or completely inhibited, and the nitrogen fixation efficiency of the remaining nodules may be reduced. Cysts are lemon-shaped and measure 560–850 × 350–590 μm. Brown bullae (internal knobs in the anal area) are present. Young females are white when young and turn yellow with age; upon death the body wall hardens and becomes a dark brown cyst. The cyst wall has a pattern of irregular, short, zigzag lines. The female produces a gelatinous matrix at the vulval cone which usually contains some eggs, and the female body also is filled with eggs. Males are veliform and 1–1.5 mm long. Second stage juveniles are approximately 450 μm long. About half of the tail of the second stage juvenile is hyaline.

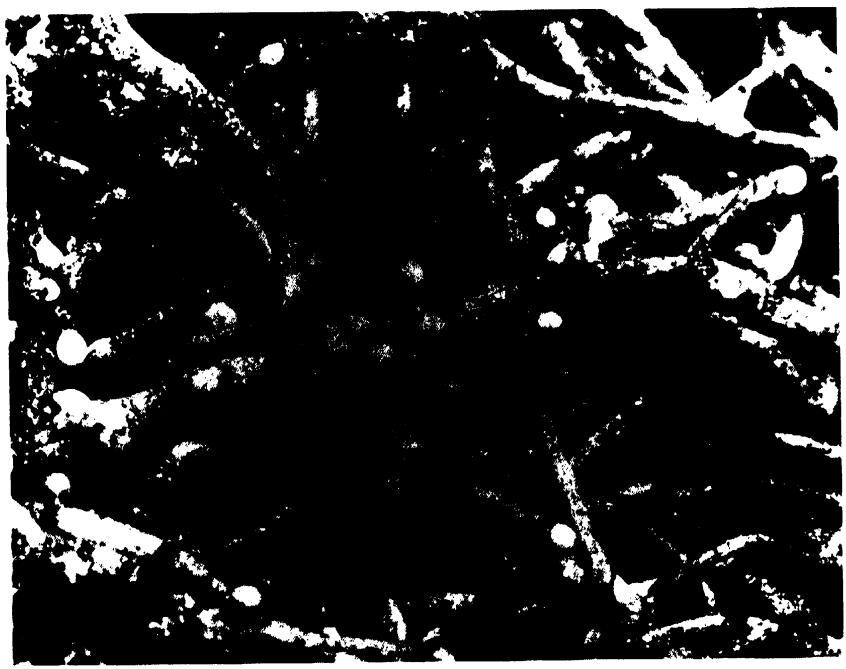

Fig. 3.18. Roots of a soyabean plant with cysts formed by female soyabean cyst nematodes, *Heterodera glycines* (photo: courtesy of US Department of Agriculture).

Epidemiology

Eggs within *H. glycines* survive for 11 years or more. The relationship between soyabean yield and the number of eggs generally is linear. This tolerance of low population levels of the nematode is dependent on soil factors, especially those related to moisture and nutrition. Soybean cyst nematode can be disseminated by wind, water, soil, pods in uncleaned seed, and machinery. As the nematode can move on its own through the soil at a rate of only a few centimetres a year, passive movement with soil is important in dissemination of this nematode.

The length of the life cycle depends on soil temperature. A generation lasts from 24–40 days under ideal field conditions varying with temperature in the range of 18 to 23°C. Thus, three to six generations are possible in a single year, depending on location. The nematode fails to develop on soyabean roots below 10°C or above 34°C. Survival in the absence of a host plant depends on soil temperature and moisture. Eggs in cysts are capable of remaining viable for up to 8 years in moist cool soil. Even in dry conditions, some eggs may survive in cysts as long as 7 years under cool conditions. Viability declines when dry soil is exposed to high temperatures or to flooding.

Damage and Crop Loss

Yield losses vary from minor to complete, depending upon the race and the population density of the nematode, cultivar susceptibility, environmental conditions, and crop management practices (Koennig and Barker, 1995). Severe losses may be confined to restricted areas in a field. Heavy infestation reduces effectiveness of *Bradyrhizobium* (McGinnity *et al.*, 1980).

Management

Long-term control of soyabean cyst nematode requires an integration of management practices, including crop rotation for at least 2 years, the use of resistant cultivars and good crop management (Koenning *et al.*, 1995). Integration of control tactics is dependent on the crop or crops, available cultivars, and ability to manage soil water. Important considerations in integrated management are the use of a non-host to maintain a low nematode population (Rodriquez-Kabana *et al.*, 1991), good weed control and the use of resistant cultivars in such a way that race shifts are minimized. The use of nematicides may be useful if other nematode species are present, or if genotypes of soyabean cyst nematode are mixed.

PROSPECTIVE

Soyabeans have good potential for production in the tropics. Yields from a newly introduced crop to an area are usually good and are larger from late-maturing

than from early-maturing cultivars. However, yields are still lower than in temperate regions, in part due to poor nodulation by nitrogen-fixing bacteria. Loss from diseases on a worldwide basis is about 15% of potential yield for any single season. These losses vary widely from year to year, and not always to the same disease in any one year. These losses are due primarily to foliar diseases, such as anthracnose, frogeye leaf spot and rust, as well as to soilborne pathogens that cause charcoal rot, pre-and post-emergence damping-off, seedling diseases, root rots and nematode damage. The use of resistant cultivars is a major means of disease management in the tropics, especially for small growers. However, the pathogens that cause the diseases described in this chapter are highly variable, made up of populations, of races, or strains, and breeding for resistance or tolerance is a long-term objective. Perhaps with the use of biotechnological methods, multiple resistance genes can be incorporated into soyabean cultivars with acceptable yield potential for the tropics.

Soyabeans have been shown to contain all but one essential amino acids required for good human nutrition and, more recently, soya protein has been shown to have beneficial effects on lowering human serum blood cholesterol and preventing heart disease. There are many new value-added products from soyabean being developed, such as a control for mosquitoes, non-toxic adhesives for composition board, plastic foam for insulation and other uses, biofuels, and flavourful prepared foods. Biotechnical tools, such as the use of random amplification of polymorphic DNA (RAPD) for DNA identification, are providing a means to identify beneficial genes influencing higher yield and preferred quality traits as well as disease resistance. The breakthrough of crossing wild *Glycine* spp. (which are immune to most soyabean pathogens) with *G. max* provides a source of genetic material yet to be exploited (Singh and Hymowitz, 1985). Soyabeans truly are the crop of the twenty-first century. Through integrated disease, insect and weed management using sustainable technology and improved cultivars, soyabean production will be profitable for growers worldwide.

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DISEASES OF COMMON BEAN

4

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INTRODUCTION

This chapter is confined to a review of the diseases of the cultivated species of the genus *Phaseolus* with emphasis on the common bean (*Phaseolus vulgaris*). *P. vulgaris* belongs to the family *Fabaceae*, in tribe *Phaseoleae* of subfamily *Papilionoideae*, which contains all the grain legumes important in world agriculture (Polhill and van der Maesen, 1985). Domesticated in Latin America, *P. vulgaris* is now the most important grain legume for small-scale farmers in intermediate altitude zones of tropical Latin America and Africa.

Its diversity of growth habit, from determinate bush types to vigorous climbers, suits common bean to a wide variety of cropping systems and environments. Pure stands of common bean are found mainly in large-scale agricultural situations though, on small farms in the Great Lakes region of central Africa, there are large areas of climbing bean grown pure (Allen *et al.*, 1989). On small farms, common bean is most frequently grown in traditional systems, in association with other crops – notably maize, but also with banana and coffee, important in central and eastern Africa (Woolley *et al.*, 1991).

Risk aversion is a major strategy of small-scale farmers in developing countries. Mixed cropping helps to avert risk through diversity (van Rheenen *et al.*, 1981; Davis and Panse, 1987). But it is vulnerable because small farmers lack wherewithal to protect crops against the many constraints that afflict them in tropical environments (Pinstrup Anderson *et al.*, 1976; Wortmann and Allen, 1994). Among these constraints, diseases feature prominently. Some 200 pathogens are known to attack the common bean but fewer than a dozen cause substantial economic damage. Diseases like anthracnose, angular leaf spot, rust, common bacterial blight, bean common mosaic and bean golden mosaic are very widespread and can decrease seed yield considerably. One or more of these diseases is almost always associated with the bean crop wherever it is grown. Other bean diseases can also cause significant crop loss, but they tend to be confined to specific environments. This group includes halo blight and ascochyta blight,

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among others. Another group of bean diseases, although widespread, tends not to cause very large losses; all the rest of the diseases are either sporadic in occurrence or are of only local importance (Beebe and Pastor-Corrales, 1991; Allen, 1995).

Here, we focus on the eight diseases referred to above. In the interests of keeping text concise, fungal pathogens not dealt with fully are listed in Table 4.1.

Table 4.1. Local or minor fungal diseases of common bean.

Disease	Causal fungi	Distribution (references)[1]
Seed and seedling diseases		
Seed decay	*Aspergillus* spp., *Botryodiplodia theobromae* Pat.	Widespread (5,7,9)
Damping-off	*Pythium ultimum* Trow., *P. myriotylum* Dresch. and other spp., *Rhizoctonia* spp. and *Sclerotium rolfsii* Sacc.	Widespread (3,4,14)
Stem and root rots and vascular wilts		
Charcoal rot, ashy stem blight	*Macrophomina phaseolina* (Tassi) Goid.	Widespread (14)
Sclerotium root rot	*Sclerotium rolfsii* (teleomorph = *Corticium rolfsii* Curzi)	Widespread in warm temperate and tropical regions (14)
Pythium root rot	*Pythium aphanidermatum* (Edson) Fitz.; *P. debaryanum* Hesse, *P. ultimum* Trow. and other spp.	Widespread (14)
Rhizoctonia root rot	*Rhizoctonia solani* Kühn (teleomorph = *Thanatephorus cucumeris* (Frank) Donk)	Widespread (14)
Aphanomyces root and hypocotyl rot	*Aphanomyces euteiches* Dresch.	USA (14)
Texas root rot	*Phymatotrichum omnivorum* (Shear) Dugg.	USA, Mexico (14)
Black root rot	*Thielaviopsis basicola* (Berk. & Br.) Ferr.	USA, Europe (14)
Fusarium dry root rot	*Fusarium solani* f. sp. *phaseoli* (Burk.) Snyder & Hansen (teleomorph = *Nectria haematococca* Berk. & Br.)	Widespread (14)
Fusarium wilt, yellows	*Fusarium oxysporum* f. sp. *phaseoli* Kendrick & Snyder	Temperate/tropical America, central Africa (14)
Foliar and pod diseases		
Web blight, mustia	*Rhizoctonia solani* Kühn	Widespread; especially important in Central America (12)

Table 4.1. Continued

Disease	Causal fungi	Distribution/references[1]
White mould	*Sclerotinia sclerotiorum* (Lib.) de Bary	Locally important temperate/tropical America, eastern Africa and Australia (13)
Floury leaf spot	*Mycovellosiella phaseoli* (Drummond) Deighton	Widespread, warrants more research (10)
Alternaria leaf and pod spot	*Alternaria alternata* (Fr.) Keissler and other spp.	Widespread, minor (10,11)
Phyllosticta leaf spot	*Phyllosticta phaseolina* Sacc.	Widespread, minor (10)
Chaetoseptoria leaf spot	*Chaetoseptoria wellmanii* Stev.	Tropical America (1,2)
Scab	*Sphaceloma* state of *Elsinoe phaseoli* Jenkins.	Eastern/southern Africa, locally and seasonally important (10,15), needs research attention
Grey leaf spot	*Cercospora vanderysti* P. Henn. and *C. castellanii* Matta & Bell.	Latin America (11)
Cercospora leaf spot or blotch	*Cercospora canescens* Ell. & Mart., *Pseudocercospora cruenta* Sacc. (Deighton) (teleomorph = *Mycosphaerella cruenta* Latham) and other spp.	Widespread, minor (10,11)
White leaf spot	*Pseudocercosporella albida* (Matta & Bell.) Deighton	Dominican Republic, Guatemala, Colombia (6,11)
Leaf smut	*Entyloma* spp., but there may be confusion with *Protomycopsis* spp.	Tropical America, minor (8,10)
Powdery mildew	*Erysiphe polygoni* DC.	Worldwide, exacerbated by shade (10)
Phytophthora pod rot	*Phytophthora nicotianae* var.*parasitica* (Dast.) Waterh. and *P. phaseoli* Thaxt	USA, Zaire, India (10)
Grey mould	*Botrytis cinerea* Pers. ex Fries (teleomorph = *Botryotinia fuckeliana* (de Bary) Whetzel)	Widespread (11)
Diaporthe pod blight	*Diaporthe phaseolorum* (Cooke & Ellis) Sacc.	Latin America (11)
Yeast spot	*Nematospora coryli* Peg. and other spp.	Widespread (11)

[1] References: 1. Muller (1953); 2. Yerkes (1956); 3. Schroth and Cook (1964); 4. Gay (1969); 5. Habish (1972); 6. Deighton (1976); 7. Ellis *et al.* (1977); 8. Vakili (1978); 9. Seenappa *et al.* (1981); 10. Allen (1983); 11. Schwartz (1989a); 12. Galvez *et al.* (1989); 13. Schwartz and Steadman (1989); 14. Abawi and Pastor-Corrales (1990); 15. Phillips (1994).

Similarly, the other bacterial and virus diseases that afflict the common bean are summarized in Tables 4.2 and 4.3, respectively. We have chosen to omit mention of diseases caused by phytoplasma-like organisms and other disorders caused by parasitic weeds, nematodes and nutritional factors. The reader is referred to the reviews of Allen (1983) and Schwartz and Pastor-Corrales (1989) for treatment of these topics.

Although seed treatments may hold some promise (Trutmann *et al.*, 1992), for small-scale farmers in the tropics host plant resistance usually remains the only feasible means of disease control. Its effective deployment requires understanding of the nature and genetics of pathogenicity in the pathogen and of resistance in primary and secondary hosts and of their interactions with environment. Important advances in these fields have been made recently with several of the major diseases of common bean, notably halo blight and bean common mosaic virus. We review existing knowledge of these and other diseases in this chapter.

ANTHRACNOSE

Aetiology

The fungus causing bean anthracnose belongs to the genus *Colletotrichum* which, despite recent revision, contains much taxonomic uncertainty. Citation of the pathogen has been variable (Walker, 1957) and controversial, but *C. lindemuthianum* (Sacc. & Magn.) Bri. & Cav. is used in this review following taxonomic usage at the International Mycological Institute in the UK. The disease is caused exclusively by the imperfect form of the fungus. The perfect stage, which has been

Table 4.2. Minor bacterial diseases of common bean.

Disease	Causal bacteria	Distribution/references
Brown spot	*Pseudomonas syringae* pv. *syringae* van Hall	Eastern and southern Africa (Kaiser and Ramos, 1980; Allen, 1995); temperate and tropical America (Patel *et al.*, 1964; Mohan and Hagedorn, 1989)
Wild fire	*Pseudomonas syringae* pv. *tabaci* (Wolf & Foster) Young, Dye & Wilkie	Brazil (Ribeiro *et al.*, 1979); Argentina (Mohan and Hagedorn, 1989)
Wilt	*Burkholderia solanacearum* (E.F. Sm.) Yabuuchi *et al.*	USA (Smith and McCulloch, 1919); Swaziland and Madagascar (Allen, 1995)
Wilt	*Curtobacterium flaccumfaciens* pv. *flaccumfaciens* (Hedges) Collins & Jones	USA (Schuster and Christiansen, 1957; Mohan and Hagerdorn, 1989); Kenya, Mauritius (Allen, 1995)

Table 4.3. Minor virus diseases of common bean.

Disease	Causal virus	Distribution/references
Yellow mosaic	Bean yellow mosaic potyvirus	Widespread (Galvez and Morales, 1989a), including eastern Africa (Vetten and Allen, 1991). Important in Chile, Uruguay (CIAT, 1990)
Peanut mottle	Peanut mottle potyvirus	Eastern and southern Africa (Vetten and Allen, 1991)
Soyabean mosaic	Soybean mosaic potyvirus	Widespread (Provvidenti *et al.*, 1982; Galvez and Morales, 1989a)
Cucumber mosaic	Cucumber mosaic cucumovirus	Widespread (Galvez and Morales, 1989a), including south central Africa (Vetten and Allen, 1991). Locally important in Chile (CIAT, 1991)
Peanut stunt	Peanut stunt cucumovirus	USA, north Africa, Japan (Mink, 1972; Fischer and Lockhart, 1978; Ahmed and Mills, 1985)
Yellow dot, spot mosaic	Alfalfa mosaic alfamo virus	USA (Zaumeyer, 1963); South Africa (Allen, 1995)
Pod mottle	Bean pod mottle comovirus	USA (Zaumeyer and Thomas, 1948; Semancik, 1972)
Rugose mosaic	Bean rugose mosaic comovirus	Costa Rica, El Salvador, Guatemala (Gamez, 1982)
Severe mosaic	Quail pea mosaic comovirus	El Salvador, Costa Rica, Guatemala (Moore and Scott, 1981; Morales and Castano, 1992)
Bean mild mosaic	Bean mild mosaic virus	El Salvador, Colombia (Waterworth, 1981)
Yellow stipple	Cowpea chlorotic mottle bromovirus	Southern USA, Central America (Fulton *et al.*, 1975)
Southern bean mosaic	Southern bean mosaic sobemovirus	USA, Latin America, western and central Africa, France (Tremaine and Hamilton, 1983)
Bean dwarf mosaic 'achaparramiento'	Bean dwarf mosaic geminivirus	Latin America, especially N.W. Argentina (Morales *et al.*, 1990; Hidayat, *et al.*, 1993)
Bean calico mosaic	Bean calico mosaic geminivirus	Mexico (Brown *et al.*, 1990)
Euphorbia mosaic	Euphorbia mosaic geminivirus	Latin America (Galvez and Morales, 1989b)
Mung bean yellow mosaic	Mung bean yellow mosaic geminivirus	India (Singh, 1979; Honda *et al.*, 1983)
Curly top	Beet curly top geminivirus	USA and eastern Mediterranean (Thomas and Mink, 1979)
Summer death	Tobacco yellow dwarf geminivirı	Australia (Thomas and Bowyer, 1984)
Mild mottle, leaf curl	Cowpea mild mottle carlavirus	Eastern and southern Africa (Mink and Keswani, 1987; Vetten and Allen, 1991); Brazil (Costa *et al.*, 1983)
Red node	Tobacco streak ilarvirus	USA, Latin America (Mink *et al.*, 1966; Fulton, 1971).
Stipple streak	Tobacco necrosis virus	Netherlands (Bawden and van der Want, 1949)
Tobacco mosaic	Tobacco mosaic tobamovirus	Kenya (Hollings *et al.*, 1981).
Spotted wilt	Tomato spotted wilt virus	Brazil, Canada (Allen, 1983)

found rarely in either culture or nature, has been named *Glomerella lindemuthianum* (Shear and Wood, 1913) and, more recently, *G. cingulata* (Stonem.) Spauld. & Shrenk f. *phaseoli* (Kimati and Galli, 1970). Diseases caused by other *Colletotrichum* spp. are reviewed in Allen *et al.* (Chapter 5, this volume), Hill (Chapter 11, this volume) and Lenné (Chapter 13, this volume).

Biology

Conidia are produced in acervuli formed from cushion-like masses of intra- or inter-epidermal hyphae, disrupting the outer epidermal cells of the host. The acervuli have simple short erect conidiophores, on which conidia are produced, and dark spines or setae at the margin. Conidia are hyaline, single-celled, cylindrical or dumb-bell-shaped and uninucleate. They are confined by a gelatinous coating and *en masse* appear salmon pink in colour. On a suitable host, in the presence of water, conidia germinate and produce one to four germ tubes whose tips enlarge on contact with the epidermis to form thick-walled, brown appressoria. From each appressorium a peg-like infection hypha develops and penetrates the host tissue mechanically (Dey, 1919) both inter- and intracellularly to form the primary mycelium. The invaded tissue then becomes water-soaked (Leach, 1923). A secondary mycelium continues the infection and, under suitable environmental conditions, forms lesions which produce acervuli and spores, thus completing the life cycle. Several cycles may be completed in the same season.

The host range of *C. lindemuthianum* is thus far confined to the genera *Canavalia*, *Lablab*, *Phaseolus* and *Vigna*, all belonging to the tribe *Phaseoleae*, and *Vicia* in the tribe *Vicieae*. It has been recorded on: the wild form of common bean (*P. vulgaris* var. *aborigineus*); lima bean (*P. lunatus*); scarlet runner bean (*P. coccineus*); cultivated tepary bean (*P. acutifolius* var. *latifolius*); urd bean (*Vigna mungo*); mung bean (*V. radiata*); jack bean (*Canavalia ensiformis*); horse or faba bean (*Vicia faba*); and hyacinth bean (*Lablab purpureus*) (Zaumeyer and Thomas, 1957; Mordue, 1971a, b; Onesirosan and Barker, 1971; Sherf and MacNab, 1986; Pastor-Corrales and Tu, 1989). Doubts whether *C. lindemuthianum* also causes cowpea anthracnose have been raised following the recent observation of an abnormal infection process on cowpea for a *Colletotrichum* species (Bailey *et al.*, 1990), now considered to be *C. destructivum* (J.A. Bailey, Warwick, 1995, personal communication; Allen *et al.*, Chapter 5, this volume).

Following the work of Barrus (1918) in the USA, a large number of races of *C. lindemuthianum* have been identified (Table 4.4), reflecting the wide range of pathogenic variation displayed by the fungus. Recent studies in Latin America distinguish two gene pools in *C. lindemuthianum*, corresponding with the Andean and Mesoamerican gene pools of cultivated common bean (Pastor-Corrales *et al.*, 1993; Pastor-Corrales, 1994). Andean pathotypes have a narrow virulence range and predominantly attack large-seeded common bean genotypes of Andean origin. Mesoamerican *C. lindemuthianum* pathotypes have a broader virulence range and predominantly attack small-seeded Mesoamerican common bean varieties. These observations are consistent with the separate co-evolution of the anthracnose pathogen and its common bean host in the two regions.

Table 4.4. Races of *Colletotrichum lindemuthianum* reported.

Races	Countries	References
alpha, beta	USA	Barrus (1918)
gamma	USA	Burkholder (1923)
delta	USA	Andrus and Wade (1942)
alpha, beta, gamma	Germany	Peuser (1931)
delta	Netherlands	Hubbeling (1957)
delta	France	Bannerot (1965)
epsilon	France	Blondet (1963)
kappa	France	Schnock *et al.* (1975)
alpha-brazil, lambda-mutant	France	Fouilloux (1979)
lambda, iota	Netherlands	Hubbeling (1976)
alpha, beta, gamma, delta, epsilon	Italy	Ferrante and Bisiach (1976)
alpha, beta, gamma, delta, epsilon	Canada	Tu *et al.* (1984)
Aust-1 to Aust-8	Australia	Waterhouse (1955)
alpha, beta, gamma, Mexican groups I, II, III	Mexico	Yerkes and Tellis-Ortiz (1956)
Mexico group IV, alpha group	Mexico	Garrido (1986)
alpha, beta, gamma, epsilon, lambda, kappa, zeta, theta, mu, Mexico groups I and II, Brazil groups I, II, III	Brazil	Oliarai *et al.* (1973); Balardin (1988); Menezes and Dianese (1988)
alpha, beta, gamma	Colombia	CIAT (1976)
alpha, beta, gamma	Costa Rica	M.A. Pastor-Corrales (unpublished data)
alpha, beta, gamma, delta, epsilon	Uganda	Leakey and Simbwa-Bunnya (1972)
alpha, beta, gamma, delta, lambda, alpha-Brazil	Malawi	Ayonoadu (1974); Bokosi (1985)
alpha, beta, gamma, delta, epsilon	Kenya	Kinyua (1979); Mwangi (1983)
alpha, beta, gamma, delta, kappa	Tanzania	Anonymous (1984)
alpha, alpha-Brazil, beta, gamma, epsilon	Rwanda	Nkezabera (1987)
alpha, beta, Brazil group I, Mexico Group I	Burundi	Bigirimana and Perreaux (1988)
alpha-Brazil	South Africa	Edington (1990)

The different criteria, differential cultivars and systems of nomenclature used to distinguish races creates problems in comparing results. More than 41 differential cultivars have been used, with Michelite, Michigan Dark Red Kidney, Perry Marrow, Cornell 49–242 and Kaboon the most common (Pastor-Corrales, 1988; Buruchara, 1991). The Greek alphabet has been most widely employed to designate races. Other systems include: race groups, for example, the Mexico and alpha groups (Yerkes and Tellis-Ortiz 1956); the Roman alphabet (Peuser, 1931); and numbers Aust-1, Aust-2 (Waterhouse, 1955). Thus it is obvious that there is an urgent need to standardize the system of race designation. It is equally important to use an international standard set of differential cultivars to facilitate data comparison and deploy genetic resistance to the pathogen more effectively. Recent proposals for standard sets of international cultivars (Pastor-Corrales, 1988; Drijfhout and Davis, 1989) and use of the binary system for race designation have led to the recommendation of a set of 12 differential cultivars (Table 4.5) which is now used by scientists in Latin America and Africa.

Table 4.5. The order, binary values and some characteristics of bean differentials used in characterization of the pathogen diversity (races) of *C. lindemuthianum*.

Differential cultivars	Seed size[1]	Seed colour	Growth habit[2]	Phaseolin type	Bean race	Binary value[3]
1. Michelite	S	White	III	Sb	Mesoamerica	1
2. MDRK[4]	L	Red	I	T	New Granada	2
3. Perry Marrow	L	White	III	T	Chile	4
4. Cornell 49242	S	Black	II	S	Mesoamerica	8
5. Widusa	M	White	I	S	Durango	16
6. Kaboon	L	White	I	T	New Granada	32
7. Mexico 222	M	White	I	T	Durango	64
8. PI 207262	S	Cream	III	S	Mesoamerica	128
9. TO	M	Cream	I	S	Durango	256
10. TU	M	Black	III	B	Durango	512
11. AB 136	S	Red	IV	B	Mesoamerica	1024
12. G 2333	S	Red	IV	B	Mesoamerica	2048

[1] S = small (<25 g seed^{-1}), M = medium (25–40 g seed^{-1}, L = large (>40 g seed^{-1}); [2] I = determinate, II = indeterminate bush, III = facultative climber, IV = climber; [3] The binary value when a cultivar gives a susceptible reaction. Designation of a race is obtained by adding the values corresponding to susceptible cultivars. [4] MDRK = Michigan Dark Red Kidney.

From a study of the progeny of two crosses, Bannerot and Ritcher (1968) postulated the evolution of pathogenicity within two different but related groups (A and B) of races of *C. lindemuthianum*, and suggested that alpha evolved to delta (in group A) and beta to gamma (in group B). They also showed genetic relationship of host–pathogen interactions of the fungus.

Molecular approaches confirm the vast genetic diversity in the genus (Rodriguez *et al.*, 1990). Preliminary studies with isolates of *C. lindemuthianum* from Argentina (Neema *et al.*, 1994) showed successful amplifications with several primers using a polymerase chain reaction (PCR) technique involving the random amplification of polymorphic DNA (RAPD). The genomic variability of *C. lindemuthianum* was not correlated with virulence patterns or geographic distribution of the isolates. In Colombia, RAPD techniques have identified primers that lead to the production of unique bands for individual isolates (Otoya *et al.*, 1994b; CIAT, 1994). The results indicate that *C. lindemuthianum* is highly variable but that RAPD patterns are not correlated with overall spatial distribution and virulence, as also found by Neema *et al.* (1994), suggesting that most DNA polymorphism is independent of virulence. Similar observations have been made for *C. gloeosporiodes* on *Stylosanthes* spp. (see Lenné, Chapter 13, this volume). RAPD patterns among 178 isolates suggest the existence of several lineages of genetic evolution of which only a few have evolved virulence of increased complexity.

Symptoms

Susceptible genotypes may exhibit symptoms on all aerial parts of the plant. Seedborne infection usually induces dark brown to black eye-shaped lesions longitudinally on the hypocotyl and cotyledons. On the hypocotyl, the lesions enlarge and may cause the stem to break. On older stems, lesions are sunken and may reach a length of 5–7 mm (Pastor-Corrales and Tu, 1989). Early signs of leaf infection occur on the petiole and on the lower leaf surface where small lesions extend along the veins developing a brick-red to purple-red coloration becoming black (Fig. 4.1). Later, similar symptoms appear on the upper leaf surface. Perhaps the most characteristic symptoms occur on the pods, on which brown or rusty coloured spots enlarge and develop as sunken cankers with dark brown margins surrounded by slightly raised reddish-brown borders. Under favourable environmental conditions, sporulation of conidia may occur, giving the appearance of pink viscous masses at the centres of lesions (Fig. 4.2). Seeds from heavily infected pods may show a variation of discoloration depending on the colour of the seed testa (Zaumeyer and Thomas, 1957). In severely infected seed, the lesion may extend to the cotyledons.

Fig. 4.1. Anthracnose lesions on bean leaf (photo: courtesy of CIAT).

Fig. 4.2. Anthracnose lesions on bean pod (photo: courtesy of CIAT).

Epidemiology

Infection and development of anthracnose depend on interactions among host, pathogen and prevailing environmental conditions. Infected seeds supply the primary inoculum for disease development and secondary spread (Tu, 1983; Fernandez *et al.*, 1987). Early seedling infection often leads to high disease severity on the same plant (CIAT, 1976) and greater chances for spread and infection of neighbouring plants (Zaumeyer and Thomas, 1957). Spread of the disease from a focus to other susceptible plants is influenced by environmental factors responsible for inoculum dispersal, such as rain-splash or wind-driven rain (Tu, 1983), and cultural practices, such as intercropping and growing of mixtures (Mwalyego, 1991; Trutmann and Kayitare, 1991). The number of foci of the initial inoculum has been shown to be linearly related to disease incidence on leaves but not on pods (Araya-Fernandez, 1981).

The penetration rate of the infection tube is faster in younger than in older cells (Landes and Hoffman, 1973). In susceptible genotypes, young plants have been associated with greater disease severity and yield losses (CIAT, 1976; Guzman-Vargas *et al.*, 1979). Some genotypes appear less influenced by plant age and exhibit susceptibility throughout the vegetative phase while others show an increase in disease severity with increase in plant age (Sindhan and Bose, 1981).

Free water is necessary to dissolve the water-soluble gelatin matrix associated with the spore mass of *C. lindemuthianum* in the acervuli. Local dissemination of conidia (within the same plant or crop) is achieved by water-splash from rain or irrigation water. Water-splash accompanied by strong winds is responsible for secondary spread within the field (Tu, 1983). Direction of spread depends on the direction of prevailing winds, while the rate of spread is influenced by the distance between plants and whether the crop is grown alone or in association with other species. Conidia may also be dispersed by insects, animals and humans, particularly when the foliage is moist (Zaumeyer and Thomas, 1957). The seedborne nature of the pathogen is critical in its international dissemination through the introduction of commercial seed and germplasm (Neergaard, 1979).

In both temperate and tropical climates, *C. lindemuthianum* can survive between seasons in seeds and plant debris. Length of survival is influenced by environment, especially moisture and temperature (Mordue, 1971a, b; Tu, 1983). The fungus remains viable for as long as 5 years in air-dried pods or seeds stored at 4°C, but alternating wet and dry cycles curtail its survival (Tu, 1983). In seeds, the fungus persists as a dormant mycelium (Zaumeyer and Meiners, 1975).

Infection, disease development and symptom expression are strongly influenced by environment. High relative humidity or free moisture is essential for dissemination and germination of conidia, infection, incubation and subsequent sporulation (Ferrante and Bisiach, 1976; Tu, 1983). Development of anthracnose occurs between 13 and 26°C (Zaumeyer and Thomas, 1957; Ferrante and Bisiach, 1976) with optimum temperatures ranging between 17 and 24°C (Tu and Aylesworth, 1980). Good infection is obtained when inoculated plants are incubated at or near 100% relative humidity at 22°C for 5–7 days following artificial inoculation (Pastor-Corrales, 1985).

Crop Loss

On susceptible cultivars, greater disease severity and crop loss are associated with early plant infection (CIAT, 1976; Guzman-Vargas *et al.*, 1979; Mukunya and Keya, 1979). On research stations, losses due to anthracnose have been estimated to range between 47 and 86% on susceptible cultivars in Tanzania (Shao and Teri, 1985), 92% in Malawi (Peregrine, 1971), 95% in Kenya (Mukunya and Keya, 1979) and up to 95% in Colombia (Guzman-Vargas *et al.*, 1979). Although the potential and actual effects of anthracnose in farmers' fields are generally appreciated, estimates of yield losses under these conditions are lacking.

Control

Use of clean seed is a potentially powerful control measure in areas where strict standards of seed health can be maintained (Allen, 1983; Fernandez *et al.*,

1987), since this reduces the inoculum for secondary spread of anthracnose. Successful control of anthracnose in Canada (Tu, 1986b) and the USA (Zaumeyer and Thomas, 1957) is attributed in part to the use of clean seed. In Tanzania, the rejection of discoloured seeds has been shown to reduce anthracnose by 33% and increase yield by 17% (Anonymous, 1984). Seed selection is practised in Africa and Latin America, where the majority of farmers use their own seed for planting (Allen *et al.*, 1989; Buruchara, 1990; Trutmann and Kayitare, 1991) and is probably responsible for the relatively low incidence of anthracnose in farmers' crops (Buruchara, 1990). Burial of plant debris, rogueing of diseased plants, removal of diseased basal leaves at weeding and crop rotation also decrease disease incidence (Crispin-Medina and Campos-Avila, 1976; CIAT, 1986; Trutmann and Kayitare, 1991). Seed treatment with hot water at 50°C for 20 min is reported to be effective in inactivating seedborne *C. lindemuthianum* without affecting seed viability (Hernandez and Mendoza, 1987). Use of cultivar mixtures, a practice common in the Great Lakes region of central Africa, Malawi, southern Tanzania and Uganda, may play an important role in buffering against disease and stabilizing yields (Allen, *et al.*, 1989; Mwalyego, 1991; Trutmann *et al.*, 1993). Use of mixtures for managing anthracnose of tropical pasture legumes is reviewed by Lenné (Chapter 13, this volume).

Seed treatment with thiram (Costa, 1972), ceresan, benomyl or carboxin (Guzman-Vargas *et al.*, 1979; Sindhan and Bose, 1981) is effective against infection by way of the seed coat. Satisfactory results have been obtained with foliar application of benomyl, carbendazim (Guzman-Vargas *et al.*, 1979; Sindhan and Bose, 1981) and difolatan (Navaro *et al.*, 1981). Other chemicals that have been used against anthracnose are zineb, captafol and maneb (Costa, 1972; Crispin-Medina and Campos-Avila, 1976). Limited effectiveness (Zaumeyer and Thomas, 1957; Leakey and Simbwa-Bunnya, 1972), possible development of resistant biotypes (Tu and McNaughton, 1980) and expense (Pastor-Corrales and Tu, 1989) are some of the limitations of chemical control.

Host plant resistance is a most effective and appropriate strategy for the control of anthracnose (Schwartz *et al.*, 1982) and has been widely employed in Europe (Fouilloux, 1979) and Canada (Tu, 1986b). Most sources of resistance utilized have been race specific (Andrus and Wade, 1942; Fouilloux, 1979; Beebe and Pastor-Corrales, 1991). The dominant '*Are*' gene found in Cornell 49-242 (Mastenbroek, 1960) confers resistance to the alpha, beta, gamma, epsilon and lambda races (Bannerot, 1965; Ayonoadu, 1974) and, although susceptible to a number of Latin American and African races (Hubbeling, 1976; Fouilloux, 1979; Beebe and Pastor-Corrales, 1991; CIAT, 1995), provided effective resistance to the pathogen for nearly 30 years. Other bean genotypes considered to have broad-based resistance, such as AB 136, Mexican 222, Tu, To and PI 207262 (Fouilloux, 1979; Schwartz *et al.*, 1982; Pastor-Corrales and Tu, 1989; Beebe and Pastor-Corrales, 1991) are now known to be susceptible to several Latin American and African isolates (Pastor-Corrales *et al.*, 1994b). The Mexican lines, Mexican 222, To, and Tu carry the genes *Mexique I, II* and *III*, which confer resistance to European races but not to some Latin American races (Beebe and Pastor-Corrales, 1991). Like the '*Are*' gene, they confer total resistance and are monogenic and dominant (Fouilloux, 1979).

G 2333 (PI 311998 of Mexican origin, released in the Great Lakes as cv. Umubano) has continued to exhibit resistance to most races worldwide except for one race from Costa Rica (Pastor-Corrales *et al.*, 1994b) and is a valuable source of resistance since it is also widely adapted and yields heavily. Two independent dominant genes appear to control the resistance of G 2333 to anthracnose (race 521) in the seedling and adult plant stages (Pastor-Corrales *et al.*, 1994a). Since G 2333 is resistant to a much broader range of pathogen populations, it is inferred that the two genes differ from those found in Cornell 49–242, Mexico 222, To and Tu, and are likely to prove more durable. A form of resistance not as yet explained genetically has been observed in the cultivars, ICA Llanogrande (G 12488) and Rio Negro, both of which are susceptible as seedlings but resistant in later growth stages and in the field (Beebe and Pastor-Corrales, 1991).

The use of single gene resistance to combat anthracnose is not a lasting solution and it will be necessary to identify broader spectrum resistance to reduce the chances of matching by pathogen populations. The possible coevolution of the pathogen and its host in the Andean and Mesoamerican gene pools provides a useful means of identifying appropriate sources of resistance since common bean genotypes originating from one gene pool are more likely to express resistance to pathotypes from the other. For example, the well-known source of resistance to anthracnose, AB 136, a small-seeded genotype of Mesoamerican origin, is resistant to most Andean isolates but susceptible to Mesoamerican races (CIAT, 1995). Nevertheless, several bean genotypes are resistant to both Andean and Mesoamerican races. Among 20,144 common bean accessions evaluated in Colombia, 350 (1.7%) were immune to Andean and Mesoamerican isolates inoculated separately (Pastor-Corrales *et al.*, 1994b). The majority of the resistant accessions were from the Mesoamerican gene pool but differed widely in growth habit, seed size, testa colour and adaptation.

Cattan-Toupance *et al.* (1995) have recently investigated the variability in anthracnose resistance in wild populations of common bean. Their results suggest the existence of specific resistance genes that are apparently different from those identified in the cultigen. The susceptibility of plants to local races suggests an adaptation of the pathogen toward host genotypes from the same region.

The physiological and biochemical processes associated with the expression of resistance in host–pathogen interactions have been reviewed by Bailey (1982). Expression of resistance may be due to a delay in penetration from appressoria, cell necrosis and growth of primary mycelia within the dead host cells and may be evident as hypersensitivity (Bailey and Deverall, 1971; Bailey, 1982). The cross-protection induced by inoculation with a non-pathogenic race, low inoculum concentration of the pathogenic race (Sutton, 1979), and heat treatment (32–37°C) of tissue before inoculation, are not well understood (Elliston *et al.*, 1976).

It has long been known that infection of the bean plant with an incompatible race of *C. lindemuthianum* may afford some protection against disease caused by a compatible race of the anthracnose pathogen (Rahe *et al.*, 1969). Whereas some progress has been made toward elucidating the phenomenon (Cloud and Deverall, 1987), it seems that its role in decreasing disease severity in the field, particularly in the cultivar mixtures that predominate in many parts of Africa,

has received too little attention, despite the demonstrable potential of induced resistance (Lannou *et al.*, 1995).

Recent developments in biochemical, physiological and molecular techniques have improved our understanding of the processes involved in compatible and incompatible interactions between *C. lindemuthianum* and bean cultivars. It is now known that, following infection, polysaccharides from the cell walls of the fungus elicit biochemical and physiological changes in host plants resulting in accumulation of isoflavonoid phytoalexins, deposition of wall-bound phenolic compounds and synthesis of hydroxyproline-rich glycoproteins (HRGPs) (Dixon *et al.*, 1986; Lawton and Lamb, 1987; Ellis *et al.*, 1989; Esquerre-Tugaye *et al.*, 1990). These elicitor-mediated, plant defence responses arise from a rapid but transient induction of enzyme synthesis resulting from accumulation of mRNAs due to activation of plant defence genes (Dixon *et al.*, 1986; Lawton and Lamb, 1987). Similar responses have been observed following mechanical damage of the hypocotyl tissues (Tepper *et al.*, 1989), suggesting that transcriptional activation of defence genes characteristically underlies induction of corresponding defence responses and expression of resistance. A positive correlation exists between the degree of pathogen–cultivar incompatibility and phaseolin concentration suggesting that a phytoalexin index may be of value in selecting for disease resistance (Cruickshank and Smith, 1988). Similarly, the amount of HRGP has been shown to be greater in incompatible than in compatible interactions (Esquerre-Tugaye *et al.*, 1990). Relationships between elicitor activity and host response appear complex and do not always explain race–cultivar specificity (Tepper *et al.*, 1989; De Lorenzo *et al.*, 1990) but may account for some of the host–pathogen interactions observed.

ANGULAR LEAF SPOT

Aetiology

The fungus causing angular leaf spot of common bean was first described by Saccardo (1878) in Italy as *Isariopsis griseola* and then successively as *Isariopsis laxa* (Ell.) Sacc. (Saccardo, 1886), *Graphium laxum* Ell. (Ellis, 1881), *Cercospora stuhlmanni* Henn., *Cercospora columnare* Ell. & Ev. (Ellis & Everhart, 1893), *Arthrobotryum puttemansii* Henn. and *Lindaumyces griseola* Gonz-Frag. (Gonzales-Fragoso, 1927). Harter and Zaumeyer (1944) concluded these were all synonyms of *Isariopsis griseola* and, based on conidial septation, pigmentation and conidiophore and stroma characteristics, Ellis (1971) concurred with the description by Ferraris (1909) which recognizes the angular leaf spot fungus as *Phaeoisariopsis griseola* (Sacc.) Ferr., which is used in this review. *Phaeoisariopsis griseola* belongs in the family *Stilbellaceae*, order *Stilbellales*, class *Hyphomycetes*, under the subdivision *Deuteromycotina* (Sutton, 1980). The diagnostic characteristic of *Hyphomycetes* is the apparent absence of sexual stages or teleomorphs. *Phaeoisariopsis personatum* is the causal agent of late leaf spot of groundnut (McDonald *et al.*, Chapter 2, this volume).

Biology

The conidia of *P. griseola* are borne on columnar, synnematous conidiophores which are loosely fused along most of their length, splaying out only at the apex. Synnemata separate near maturity: they are 80–680 µm long and 20–70 µm wide (Miles, 1917; Chupp, 1925; Hocking, 1967; Ellis, 1971). The conidia are pale grey, cylindrical to spindle shaped and may be slightly curved. They are 30–80 µm long and 3–8.8 µm wide. The septa usually number between three and six but can range from none to seven (Llanos, 1957; Hocking, 1967; Ellis, 1971; Buruchara, 1983). Considerable variation in conidial size and septation occurs both between and within isolates (Buruchara, 1983).

The conidia germinate in the presence of water or high relative humidity and enter the host through stomata. Growth continues intercellularly in the mesophyll and palisade layers, resulting in tissue disintegration which extends to the upper epidermis. Later, the fungus grows intracellularly in the necrotic tissues, becoming delimited by the vascular bundles in the veins (Cardona-Alvarez and Walker, 1956). Lesions may appear 5–9 days after the beginning of infection (Cardona-Alvarez, 1956; Llanos, 1957; Sindhan and Bose, 1980; Buruchara, 1983). After about 9–12 days, stromata form in the substomatal cavities and, under favourable environmental conditions, synemmata form and sporulation occurs (Cardona-Alvarez, 1956), thus completing the cycle.

A number of legumes of the genera *Phaseolus*, *Vigna*, *Macroptilium* and *Pisum* are known hosts of *P. griseola*. These include the principal host, *Phaseolus vulgaris*, as well as *P. lunatus*, *P. coccineus*, *P. acutifolius*, *Vigna mungo*, *V. angularis* (Willd.) Ohwi & Ohashi, *V. umbellata* (Thunb.) Ohwi & Ohasha, *V. unguiculata* and *Macroptilium atropurpureum* (Brock, 1951; Cardona-Alvarez and Walker, 1956; Diaz-Polanco *et al.*, 1965; Golato and Meossi, 1972; Campos-Avila, 1979; Lenné, Chapter 13, this volume). Reports that pea (*Pisum sativum* L.) (Chupp, 1925) and soyabean (*Glycine max* (L.) Merill) (Abramanoff cited by Cardona-Alvarez and Walker, 1956) are host to *P. griseola* are contradicted by Campos-Avila (1979) and Cardona-Alvarez and Walker (1956), who found no evidence of disease on these species. The present authors have found angular leaf spot only on *Phaseolus* species, perhaps suggesting a need to verify the natural versus the experimental host ranges of the pathogen.

Pathogenic variation in *P. griseola* was first demonstrated in Australia by Brock (1951), who observed differences in the pathogenicity of 13 isolates on two bean cultivars, Brown Beauty and Red Mexican. Evidence for physiological specialization in *P. griseola* has been found by: Marin-Villegas (1959) in Colombia; Hocking (1967) in Tanzania; Alvarez-Ayala and Schwartz (1979) in Colombia and Ecuador; Buruchara (1983) in Colombia and the USA; Correa-Victoria (1984) in Colombia, Brazil, the Dominican Republic, Puerto Rico, the USA and Malawi; Correa-Victoria (1987) and CIAT (1987a, b) in Latin America and Africa; and CIAT (1991) and M.M. Pyndji (unpublished data) in Zaire and Rwanda. The intensive studies of Buruchara (1983) and Correa-Victoria (1987) indicate that populations of *P. griseola* vary in aggressiveness and apparently also virulence.

Recent efforts to define pathotypes and pathogenicity groups of *P. griseola* have led to the identification of a set of common bean differential cultivars comprising six Andean and six Mesoamerican genotypes (CIAT, 1996). Results of recent studies based on these differentials clearly show that, as in *C. lindemuthianum*, there are two distinct virulence groups: one associated with large-seeded Andean cultivars and a second associated with beans of Mesoamerican origin. *P. griseola* displays a wide diversity of virulence in both Latin America and Africa (CIAT, 1994). The greatest diversity is found among Mesoamerican isolates, which exhibit a greater number of races and attack Andean cultivars as well as small-seeded genotypes of Mesoamerican origin (CIAT, 1993, 1994, 1995; R.A. Buruchara, unpublished data; Guzman *et al.*, 1995; Pastor-Corrales *et al.*, 1995). The parallel diversities of virulence and host reaction suggest that the two virulence groups have co-evolved separately with their respective common bean gene pools.

Recent biochemical and molecular studies have produced interesting results. Correa-Victoria (1987) found two distinct isoenzyme patterns among isolates of *P. griseola* from Latin America and Africa, the latter exhibiting the pattern typical of Andean cultivars (Beebe and Pastor-Corrales, 1991). Restriction fragment length polymorphism (RFLP) techniques have revealed distinct differences between isolates of *P. griseola* from Mesoamerican and Andean cultivars of common bean while isolates from bean cultivars of the same gene pool have given identical hybridization patterns with all probes tested (CIAT, 1991, 1995). Eleven primers generated reproducible and distinct RAPD patterns that separated isolates of *P. griseola* into Andean and Mesoamerican groups corresponding to their pathogenicities on differential cultivars (Guzman *et al.*, 1995). In a separate study of Colombian isolates, the correlation between virulence and RAPD similarity matrices was 0.5, conspicuously greater than those reported for other pathogens (CIAT, 1994). All these results are consistent with the hypothesis that *P. griseola* co-evolved with *P. vulgaris* separately in the two centres of diversity (Pastor-Corrales *et al.*, 1995).

Symptoms

Angular leaf spot symptoms may occur on all aerial plant parts, usually appearing close to or after flowering. On the leaves, lesions are initially greyish in colour. On primary leaves, the lesions are round and larger than those on trifoliolate leaves and may develop concentric rings. On trifoliolate leaves they become dark brown and angular in shape, surrounded by a chlorotic halo (Plate 4). Lesions increase in size and may coalesce. In severe infections, general leaf chlorosis and/or premature defoliation usually occurs. Lesions are oval to circular and reddish- to dark brown in colour on pods (Fig. 4.3) and elongated and brown in colour on stems, branches and petioles (Cardona-Alvarez and Walker, 1956; Zaumeyer and Thomas, 1957; Hagedorn and Wade, 1974). Dark grey stromata and, under humid conditions, dark grey to black synnemata bearing conidia are produced on all types of lesion (Zaumeyer and Thomas, 1957; Hagedorn and Wade, 1974). Sporulation occurs on the lower surfaces of trifoliolate leaves and on both surfaces of primary leaves.

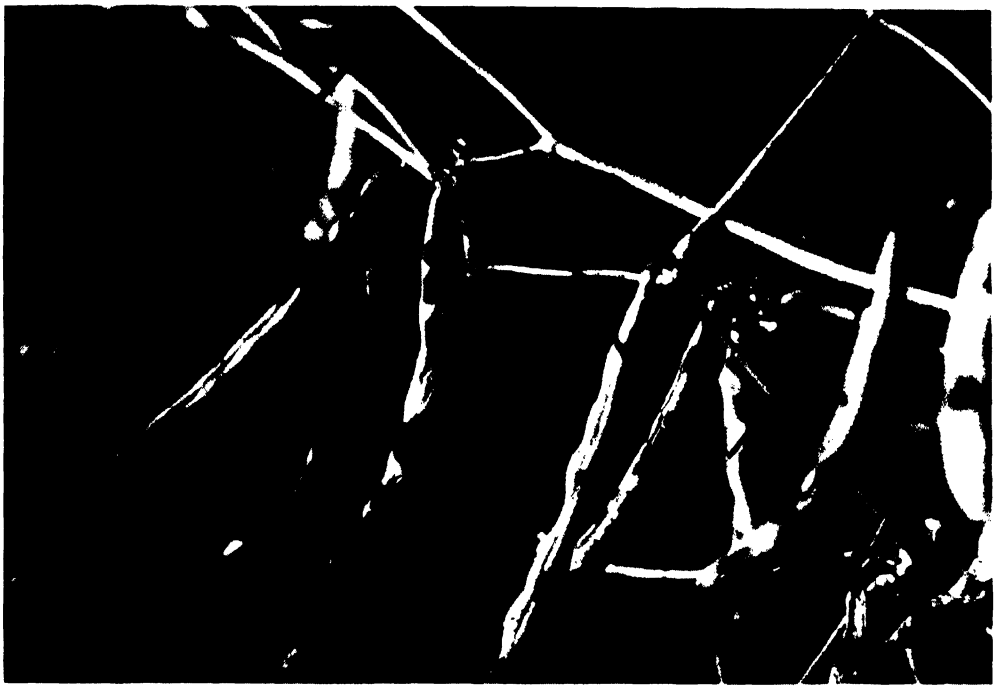

Fig. 4.3. Angular leaf spot lesions on bean pods (Photo: courtesy of D.J. Allen).

Epidemiology

P. griseola survives between seasons as dormant mycelia in seed and as stromatic growth in plant debris (Cardona-Alvarez and Walker, 1956; Sindhan and Bose, 1980). Periods of survival are variable. The pathogen can retain viability in seed for at least a year (Sindhan and Bose, 1980) and in infected debris from 4 months to more than a year, depending on environment (Sindhan and Bose, 1980; Saettler and Correa-Victoria, 1985; Sengooba and Mukiibi, 1986). Burial of debris decreases pathogen survival (Correa-Victoria, 1984). Conidia lose viability within 8 months (Sindhan and Bose, 1980).

Seedborne inoculum, by which long-distance dissemination occurs (Neergaard, 1979), leads to the development of lesions at the pod suture but seed-to-seedling transmission tends to be inefficient because the transfer of conidia from testa to leaves depends on wind and water splash (Diaz-Polanco *et al.*, 1965; Sohi and Sharma, 1974; Dhingra and Kushalappa, 1980; Correa-Victoria, 1984; Buruchara, 1985; Sengooba and Mukiibi, 1986). Although *P. griseola* sometimes is carried-over also on volunteer seedlings or off-season crops (Sengooba and Mukiibi, 1986), infected debris is the most important form of primary inoculum and leads to infection of the lower leaves of the canopy in which the disease spreads upwards (Cole, 1966; Saettler and Correa-Victoria, 1985).

Although spore release is favoured by dry conditions (Cardona-Alvarez and Walker, 1956), efficient dispersal depends on wind-driven rain, and soil and water splash. High relative humidity, rainfall or dew are also necessary to permit successful infection and subsequent formation of synnemata and conidia (Cardona-Alvarez and Walker, 1956; Llanos, 1957; Silvera, 1967; Alvarez-Ayala, 1979; Campos-Avila, 1979; Buruchara, 1983; Pastor-Corrales, 1985). A period of 48–72 h is sufficient for infection to occur (Alvarez-Ayala, 1979). Although infection and disease development occur over a range of temperatures from 16 to 28°C, 24°C is optimum. The incubation period at 24°C is about 5–7 days but this period extends to 15 days under cooler conditions (16°C) (Buruchara, 1983). Thus, epidemic development can be expected to be most rapid under conditions of high relative humidity and moderate temperatures alternating with periods of wind and low humidity.

Plant age may (Santos-Filho, 1976; Sindhan and Bhose, 1980) or may not (Cardona-Alvarez and Walker, 1956; Barros *et al.*, 1958; Marin-Villegas, 1959; Costa, 1972) influence disease development. Cultural practices, including the use of infected debris as mulch, can encourage the dissemination of *P. griseola* from spore-laden tissue (Neergaard, 1979). Other cultural practices, like the growing of bean cultivar mixtures and intercropping with a cereal, can each influence angular leaf spot development (Moreno, 1977; van Rheenen *et al.*, 1981; Pyndji, 1988) but the outcome and its underlying causes (Allen, 1990; Boudreau, 1990) have received inadequate attention.

Crop Loss

Yield losses due to angular leaf spot have been estimated at up to 50% in the USA (Cole, 1966; Hagedorn and Wade, 1974); 40–80% in Colombia (Barros *et al.*, 1958; Schwartz *et al.*, 1981; Pastor-Corrales *et al.*, 1983; Mora *et al.*, 1985); 45% in Brazil (Ravas-Seijas *et al.*, 1985); and 80% in Mexico (Crispin-Medina *et al.*, 1976). In Zaire, yield losses of 50–60% have been demonstrated in farmers' fields and on experiment stations (Pyndji, 1988). Yield losses are through reduction in seeds per pod and seed mass rather than pod number (Santos-Filho *et al.*, 1978; M.A. Pastor-Corrales, unpublished data).

Control

The use of pathogen-free seed reduces initial inoculum (Correa-Victoria, 1984; Buruchara, 1985) and decreases the possibility of introducing new variants of the pathogen (Neergaard, 1979). Since plant debris is an important means of carry-over of *P. griseola*, the removal of diseased leaves, plants and plant debris and deep ploughing are useful management strategies (Cardona-Alvarez and Walker, 1956; Saettler and Correa-Victoria, 1985; Trutmann and Kayitare, 1991). A 2-year rotation with a non-host crop was useful in Michigan (Saettler and Correa-Victoria, 1985). Intercropping with maize can either increase (Moreno, 1977) or decrease (Mora, 1978; van Rheenen *et al.*, 1981; Boudreau,

1990) the severity of angular leaf spot. Angular leaf spot has been shown to be less in cultivar mixtures than in its components grown pure (Pyndji, 1988) and the incorporation of resistant components in traditional mixtures reduces disease severity and increases yield (Pyndji and Trutmann, 1988, 1992; Trutmann and Pyndji, 1994).

Effective chemical control of angular leaf spot has been achieved by seed treatment with benomyl, captan–zineb (Correa-Victoria, 1984; Saettler and Correa-Victoria, 1985) and ceresan (Singh and Sharma, 1976). Foliar application of antracol–benomyl, benomyl, bitertanol, captafol, chlorothalonil, etiltrianol, maneb, mancozeb, metiram and zineb controls disease and increases yield (Costa, 1972; Cordoba and Martinez, 1975; Gonzales *et al.*, 1977; Pastor-Corrales *et al.*, 1983; Sengooba, 1985; Rodriguez and Melendez, 1986; Goulart, 1990). Copper oxychloride is effective against angular leaf spot but causes phytotoxicity (Gonzales *et al.*, 1977). The optimum time of initial application, number and frequency of sprays varies with chemical and prevailing environmental conditions, which thereby influence effectiveness and profitability.

For small-scale farmers, genetic resistance offers the most effective and practicable management option for the control of angular leaf spot. Resistance to angular leaf spot was first demonstrated by Gardner and Mains (1930) and various sources of resistance have since been identified (Brock, 1951; Puerta and Alonso, 1958; Santos-Filho, 1976; Singh and Saini, 1980; CIAT, 1984; Stoetzer *et al.*, 1984b; Correa-Victoria *et al.*, 1989). Though both qualitative and quantitative resistance have been recognized, no genotype with complete immunity has yet been found (Barros *et al.*, 1958; Cardona-Alvarez, 1958; Hagedorn and Rand, 1986; Buruchara *et al.*, 1988). Fifty-six genotypes with promising levels of resistance were identified in Colombia among 13,000 accessions from the World Common Bean Germplasm Collection (Schwartz *et al.*, 1982). These formed the basis of an international nursery through which several accessions have been shown to exhibit broad resistance to *P. griseola* including A 222, A 216, A 247, A 384, G 3391, G 4032, G 5207, G 5698, G 10474, G 10474, G 10613, G 14301, G 20523, CNF 5558, MEX 54 and G 5686 (CIAT, 1995; M.A. Pastor-Corrales, Cali, 1995, personal communication). Recent advances in the understanding of the evolution of the pathogen, as we discuss under 'Biology' (see above), should be expected to aid the search for further sources of resistance.

Information on the inheritance of resistance is scanty and conflicting. The resistance of some genotypes appears to be conferred by one or more independent or recessive factors, depending on the host–pathogen interaction (Barros *et al.*, 1958; Cardona-Alvarez, 1958; Santos-Filho *et al.*, 1976) about which little is known. Disease reaction is strongly influenced by environment and plant age, the latter tantamount to an 'adult plant susceptibility'. Leaf and pod reactions may also differ (Santos-Filho *et al.*, 1978; Correa-Victoria *et al.*, 1989). Whereas modern molecular techniques have confirmed the existence of considerable diversity in the pathogen, further attempts to elucidate the nature and extent of pathogenic variation and to define physiologic races in *P. griseola* seem unlikely to be successful until our understanding of the genetics controlling the interaction between the angular leaf spot fungus and *P. vulgaris* is much improved.

ASCOCHYTA BLIGHT

Aetiology

Ascochyta blight, known also as black node, leaf and pod spot, zonate leaf spot and target spot, is conventionally attributed to the imperfect fungus *Ascochyta phaseolorum* Sacc. (Alcorn, 1968; Holliday, 1980; Allen, 1983). However, Boerema (1972) has shown that *A. phaseolorum* is a synonym of *Phoma exigua* Desm., a pycnidial fungus frequently found on leaves, stems and roots of a wide range of herbaceous plants. Among the diagnostic characters of *P. exigua* is its weak parasitism: it is generally a wound parasite and appears to be a ubiquitous soil fungus (Boerema and Howeler, 1967), and this essentially conforms with the wide host range of isolates of *A. phaseolorum* from beans reported by Crossan (1958). Subsequent studies of isolates from ascochyta blight collections from Europe and eastern Africa revealed that the morphology of the pathogen, which was consistently associated with a blackening of stem nodes and petioles, enabled it to be distinguished from *P. exigua*. Boerema *et al.* (1981) described it as *P. exigua* var. *diversispora* (Bub.) Boerema, which has subsequently been shown to be the cause of ascochyta blight on a range of legume crops including the common bean in Africa (Gerlagh, 1987).

Various other related fungi are also known to cause foliar diseases in beans. These include *Ascochyta boltshauseri* Sacc. (Sprague, 1935; Sneep, 1945; Dingley, 1961; Echandi, 1976), more correctly referred to as *Stagonosporopsis hortensis* (Sacc. & Malbr.) Petr. which induces reddish-brown flecks on leaves, stems and pods (Ester, 1981). A reddish necrosis of leaf veins, branches, flowers and pods of beans is apparently caused by a *Phoma* species distinct from *P. exigua* in southern Colombia (Sanudo and Zuniga, 1982), and *P. ? sorghina* (Sacc.) Boerema, Dorenbosch & Kest. has been found associated with a red leaf spot of beans in northern Zambia (Allen, 1995), perhaps confirming a previous record (IMI 110419) from the same country (Angus, 1962–1966). Based on the above, it seems reasonable to conclude that there are at least three separate bean diseases caused by distinct taxa within the genera *Phoma* and the related *Stagonosporopsis*. For the purposes of this account, the following will refer to the more important blight, caused by *P. exigua* var. *diversispora*.

Biology

The pathogen belongs to the *Sphaeriodaceae*. Pycnidia are black and measure 160 × 120 µm, somewhat larger than in *P. exigua* var. *exigua* (Boerema *et al.*, 1981); the conidia are hyaline or pale yellowish-brown, nearly globose to ellipsoidal, 6.8 × 2.7 µm, and usually aseptate. Secondary septation can occur, apparently depending on the environment. No doubt this contributes to the common confusion between *Phoma* spp. with *Ascochyta*: conidia in *Phoma* are essentially aseptate while those in *Ascochyta* are essentially two-celled (Holliday, 1980).

Whereas the type variety of *P. exigua* is a widespread, weak, wound parasite with a very wide host range, var. *diversispora* appears to be relatively specialized.

In eastern Africa, the latter has been shown to be the cause of blight of *Phaseolus vulgaris* in Burundi, Rwanda, Tanzania, Uganda and Zambia; of *P. coccineus* in Rwanda; of *P. lunatus* in Kenya and Zambia; of *Vigna radiata* in Zambia; and *V. unguiculata* in Kenya, Zambia and Zimbabwe (M. Gerlagh and D.J. Allen, unpublished, 1986; Gerlagh, 1987). In Andean Latin America, where ascochyta blight is also an important disease, the causal agent is again attributed to *P. exigua* var. *diversispora* (Schwartz, 1989a), but there is a consistent difference in cultural characteristics between the isolates examined in The Netherlands from Latin America and those from Europe and eastern Africa. However, as regards symptoms they induce, spore form and size, and the lack of coloration of the culture medium with sodium hydroxide, they are identical to var. *diversispora* (Gerlagh, 1987).

Isoenzyme studies by Obando-Rojas (1989) indicate great uniformity within groups of isolates from the same geographic region. She concluded that the Latin American isolates as a group were probably sufficiently different from other isolates of var. *diversispora* to warrant the status of a separate species. Recent examination of a range of Latin American isolates has revealed some quantitative variation but no differential interactions were found between isolates and host genotypes. Isoenzyme studies showed little polymorphism, suggesting strong homogeneity and therefore presumably a common origin among Latin American isolates of the ascochyta blight pathogen (CIAT, 1995).

Symptoms

The first symptoms appear on leaves, on which lesions are dark grey to black, measuring 1–3 cm in diameter; later these lesions become concentric ringed. Such dark brown to black lesions develop also on petioles, stem nodes, peduncles and pods, and can cause girdling of stems leading to plant death. A blackening of the nodes is characteristic. Extensive blight and premature defoliation occurs when leaf attack is severe. Pycnidia occur at all the sites attacked. Infection of the floral remains can lead to a stem end rot of the pod but direct infection of the pod can also occur. Such pod lesions often expand and coalesce (Fig. 4.4) to cause significant damage also to the seed, in which the fungus is internally transmitted.

Epidemiology

Ascochyta blight is economically important in tropical regions, principally at altitudes above 1500 m, under cool and humid conditions. In Europe, *P. exigua* var. *diversispora* has caused severe problems only in extremely wet and cold summers. The extent of damage depends not only on environmental conditions but also on the initial inoculum dose. The fungus survives in seed and on bean straw. Work in The Netherlands has shown that the disease is carried over to the next season, even when infected bean straw is incorporated into the soil. Seed from diseased plots can be severely contaminated and are not safe even at several metres from a focus. Young plants developing from contaminated seed may produce pycnidia

Fig. 4.4. Ascochyta blight lesions on bean leaf and pods (Photo: courtesy of D.J. Allen).

from which spores may spread by rain-splash (Gerlagh, 1987). Successful infection is heavily dependent upon a high relative humidity (Boerema *et al.*, 1981) and presumably also cool temperatures, since Namekata and Figueiredo (1975) found that sporulation and germination are optimal at 21°C and mycelial growth is optimal at 24°C. The fungus is inactivated at temperatures above 30°C.

The possible role of alternative hosts in pathogen survival (Pegg and Alcorn, 1967) seems in need of investigation, because of previous confusion between *P. exigua* var. *exigua* (= *Ascochyta phaseolorum*) and *P. exigua* var. *diversispora*. Whether or not cowpea is the primary host of var. *diversispora* (Gerlagh, 1987), it is pertinent to note that the fungus apparently causes severe disease in cowpea at altitudes of about 1000 m, under conditions where beans are seldom attacked (Leakey, 1970; Allen, 1983).

Crop Loss

Seed yield losses of up to 74% have been measured in Colombia, where even moderate disease pressure can cause a 40% loss (Schwartz *et al.*, 1981; CIAT, 1984). In northern Zambia, Greenberg *et al.* (1987) failed to obtain evidence of a significant yield depression from ascochyta blight, when regression analysis of disease scores on seed yield was performed. However, studies at the same site the following season revealed that yield losses in the range of 57–341 kg ha^{-1} per unit

increase in disease score (1–9 scale) were attributed to ascochyta blight (Mulila-Mitti *et al.*, 1989). Under conditions favouring the disease in Rwanda, a 50% crop loss has been recorded (Munyemana and Trutmann, 1987).

Control

Seed dressings of benomyl combined with Dithane M45 or Mancozeb as foliar sprays have been found to decrease ascochyta blight severity in Uganda (Sengooba, 1989). In Colombia, foliar application of sulphur, benomyl, zineb, chlorothalonil and carbendazim has been found to be effective (Schwartz, 1989a). In the Netherlands, seed dressing with a combination of dichlofenthion/thiram with benomyl or carbendazim was used successfully (Ester, 1981).

The use of clean seed, field sanitation, isolation from infected reservoirs and *Pennisetum* wind-breaks have been suggested as cultural control measures (Pegg and Alcorn, 1967). The efficacy of the latter is confirmed by subsequent work with maize–bean associations, in which it has been shown that ascochyta blight is decreased in the intercrop (van Rheenen *et al.*, 1981; Msuku and Edje, 1982), but not invariably so, for some reports indicate the disease may be exacerbated in an intercrop (CIAT, 1984; Sengooba, 1989). Recent work on cultural practices to manage ascochyta blight in Colombia has shown that soil cover with banana leaves can regulate disease severity, especially when the practice is combined with rotation of beans with wheat (CIAT, 1991).

Evaluation of germplasm of *P. vulgaris* has shown that there is variation in susceptibility to ascochyta blight, but no high level of resistance has been found within the species. The more promising sources of partial resistance have been assembled into an International Bean Ascochyta Blight Nursery (CIAT, 1988). Evaluations in Rwanda, Uganda and Zambia have confirmed the existence of partial resistance which seems to be effective across locations (Munyemana and Trutmann, 1987; Mulila-Mitti *et al.*, 1989; Anonymous, 1990). Promising materials include VRA 81059, G 10823, G 10817 and G 10747, the latter two of which have been used in crosses in which heritability estimates indicate that selection for partial resistance is effective. A generation means analysis of resistant parents (G 10817 and G 10823) crossed with the susceptible parent G 12488 indicates that additive, dominance and epistatic effects may be important in the inheritance of resistance. Increasing resistance levels within *P. vulgaris* should be possible through selection in advanced generations (P.M. Hanson, M.A. Pastor-Corrales and J. Kornegay, unpublished, 1992). Many of the ascochyta blight resistant lines also have anthracnose resistance, and both bush and indeterminate climbing cultivars are among those with resistance (CIAT, 1988, 1991, 1995).

Overall, the best strategy at present seems to be the development of an integrated management of ascochyta blight, against which both cultural practices and the use of partial resistance are the most promising components.

RUST

Aetiology

Bean rust is caused by the obligate parasitic fungus, *Uromyces appendiculatus* (Pers.) Ung., of the *Basidiomycotina*. The disease was first reported in 1795 in Germany (Persoon, 1795), since when it has been recorded from almost every part of the world, occurring wherever beans are grown (Guyot, 1957; Laundon and Waterston, 1965; Stavely and Pastor-Corrales, 1989).

The morphology of *U. appendiculatus* has been well summarized (Laundon and Waterston, 1965; Wilson and Henderson, 1966). The uredial stage of the bean rust fungus was first recorded as *Uredo appendiculata* Pers. (Persoon, 1796). However, by 1804, the fungus had been reported from Germany as *Puccinia phaseoli* Reb. (Rebentisch, 1804) and Fries (1849) later re-named it *U. appendiculatus* (Pers.) Fr. Winter (1880) changed the name to *U. phaseoli* (Pers.) Wint. by which bean rust is widely known in the American literature. However, in accordance with the International Rules of Botanical Nomenclature, both Laundon and Waterston (1965) and Wilson and Henderson (1966) retain the name *U. appendiculatus* (Pers.) Ung. which is the name used here. Guyot (1957) records 33 synonyms for the bean rust fungus. Arthur (1934) erected three varieties within *U. appendiculatus* (as *U. phaseoli*) and varieties are occasionally maintained in the literature (Almeida, 1977). *U. appendiculatus* var. *crassihumiatus* is a major pathogen of *Macroptilium atropurpureum* and is reviewed in Lenné (Chapter 13, this volume).

Biology

U. appendiculatus is an autoecious, macrocyclic rust which has a full complement of spore forms (their nomenclature here follows Holliday, 1989). De Bary (1863) proved the genetic connection of the aecia with the uredia and telia, and the complete life cycle of the bean rust fungus has now been induced in the greenhouse (Andrus, 1931; Groth and Mogen, 1978). Under field conditions, it is the brown uredia and single-celled uredospores that are typical of bean rust, and are the 'repeating stage' that gives rise to secondary dispersal of the fungus. Under adverse conditions, certain isolates of *U. appendiculatus* produce within the pustule dark, thick-walled teliospores, but rust races differ in this ability (Harter *et al.*, 1935; Allen, 1975a; Groth and Mogen, 1978; Stavely, 1984a). Fusion of dikaryotic nuclei occurs in the teliospore immediately following its formation (Gold and Mendgen, 1984). Teliospores germinate to produce basidia in which meiosis occurs and on which haploid basidiospores develop (Gold and Mendgen, 1984). Some 6 days after basidiospore infection, a small chlorotic fleck containing the pycnium develops. The pycnium later produces droplets of cloudy white nectar containing heterothallic spermatia and receptive hyphae. Cross-fertilization of a pycnium by pycniospores of the opposite mating type triggers the formation of an aecidium, in which aeciospores are formed. Aeciospores in turn infect the

host plant and, after 8–10 days, a uredium develops (Stavely and Pastor-Corrales, 1989).

The pycnial and aecial stages of *U. appendiculatus* are rare under field conditions: aecia have been found in various parts of the USA (Jones, 1960; Venette *et al.*, 1978), in Germany (Heinze, 1974) and in Nigeria (Allen, 1979). The most commonly observed spore forms are the uredospore and the teliospore. Uredospores are produced in rows within the uredium, or pustule, on both leaf surfaces and other plant parts. Towards the end of the season, teliospores may form within the uredium in response to various environmental factors influenced also by plant genotype and maturity.

The host range of *U. appendiculatus* is tribe-specific, being effectively confined to the genera *Phaseolus*, *Vigna*, *Macroptilium* and *Lablab* of the *Phaseoleae* (Almeida, 1977; Allen, 1983). Between them, these genera contain crops native to the Americas (common bean, lima bean, scarlet runner bean, tepary bean and siratro), Africa (cowpea, bambarra groundnut and hyacinth bean) and Asia (mung bean); and no single ancestral host, nor region of origin, is readily discernible. The host-specific forms within *U. appendiculatus*, given specific or varietal status by some authors (Fromme, 1924; Arthur, 1934; Almeida, 1977 see Lenné, Chapter 13, this volume), may possibly reflect a New World origin of the bean rust fungus versus an Old World origin of the cowpea rust fungus (Allen, 1983). *Formae speciales*, well established among the cereal rusts, have not been erected within *U. appendiculatus*.

U. appendiculatus is a highly variable species of rust fungus in which numerous pathogenic races have been described; these have been summarized by Ballantyne (1974a) and by Stavely and Pastor-Corrales (1989). Physiologic races, each with a distinct pattern of virulence, as determined by inoculation of a set of differential cultivars (Harter and Zaumeyer, 1941; Crispin-Medina and Dongo, 1962; Davison and Vaughan, 1963; Stavely *et al.*, 1983), have long been recognized in the USA, where Harter *et al.* (1935) first reported the existence of pathogenic variation. At least 65 races have now been identified in the United States (Stavely, 1984b), 31 in Mexico (Crispin-Medina and Dongo, 1962) and at least 80 in Brazil (Carrijo *et al.*, 1980; Stavely and Pastor-Corrales, 1989). Elsewhere in the New World, rust races are known in the Caribbean, Central America, Colombia and Peru (Allen, 1983; Stavely and Pastor-Corrales, 1989). Bean rust races have also been identified in Australasia (Waterhouse, 1953; Yen and Brien, 1960; Ogle and Johnson, 1974; Ballantyne, 1978), Europe (Hubbeling, 1957; Allen, 1975a), Asia (Yeh, 1983) and Africa (Howland and Macartney, 1966; Allen, 1975a; Mmbaga and Stavely, 1988). As many as eight races may be found among collections from a single field of a susceptible cultivar (Stavely, 1984b), and changes in virulence gene frequencies occur (Augustin *et al.*, 1972; Alexander *et al.*, 1985).

Owing to the difficulty of making valid comparisons between separate studies, in which different sets of differentials, different rating scales and different systems of naming races have been used, an International Bean Rust Workshop was held in Puerto Rico in 1983 in an attempt to standardize methods (Stavely *et al.*, 1983). As a result, it is now possible to determine whether an isolate is unique or whether it is representative of a race already described (Stavely, 1984b). With

advances in knowledge of the genetics of bean–bean-rust relationships (Christ and Groth, 1979, 1982a, b), it may be expected that yet more valuable systems of race notation will be established.

Mention so far has been made only of variation in virulence. Variation in pathogenicity may also be described in terms of fitness characteristics, best referred to as aggressiveness: such variants, which do not interact differentially with host genotypes, can be distinguished by their fitness on a single universally susceptible host cultivar (Allen, 1975b). More work seems warranted on the relationships between virulence, aggressiveness and other fitness characteristics involved in survival and competitiveness of races in the bean rust fungus.

Symptoms

Symptoms of bean rust first appear about 5–6 days after infection as minute, chlorotic, raised spots on both surfaces of leaves and on pods and petioles. In a susceptible reaction, these spots enlarge and, on the eighth or ninth day after infection, the epidermis is ruptured to form reddish-brown coloured uredial pustules, about 2 mm in diameter (Fig. 4.5). These uredia reach maximum development on the fourteenth day when they are about 5 mm in diameter (Rey and Lozano, 1961). The sori may be surrounded by chlorotic haloes and, eventually, by rings of secondary sori. As the infection ages, the leaf becomes debilitated and the chlorotic areas surrounding the pustules become confluent, while the tissue ramified by the fungus remains green; these zones have been termed 'green islands' (Wingard, 1935; Wang, 1961). The sori darken as the pigmented, thick-walled and single-celled teliospores are produced and the leaf gradually dies. In severe infections, leaves may absciss after about 25 days. The green islands may remain visible after the leaf has detached and the whole of the leaf is dead.

Epidemiology

The selective retention of bean rust uredospores on bean leaves may depend on the release of film-forming substances from the spore, the amount produced depending on host cuticle wettability (Yarwood, 1968; Woodbury and Stahmann, 1970). The extent to which receptivity to *U. appendiculatus* may vary with cultivar has been examined (Groth and Urs, 1982) and there is evidence (Shaik, 1985) that leaf epidermal characters including pubescence do influence infection rate. The optimum temperature for uredospore germination is between 14.5 and 22.5°C and germ tubes are longer at temperatures whereat the most germination takes place (Harter *et al.*, 1935; Imhoff *et al.*, 1981). Dew period also influences germination (Imhoff *et al.*, 1981). Bean rust spores germinate poorly if their concentration is too dense (Yarwood, 1956) because of the presence of an auto-inhibitor (Macko *et al.*, 1970). Appressoria form over stomata in response to a surface contact stimulus (Wynn, 1976) and the infection process (Mendgen, 1973) leads to the development of uredia which attain full size some 14 days later.

Fig. 4.5. Rust uredial pustules on bean leaf (Photo: courtesy of CIAT).

Disease development is influenced by plant age. Schein and Snow (1963) have shown that whereas young primary leaves of beans are only slightly susceptible to infection, susceptibility increases about sixfold as these leaves expand, a phenomenon attributable to the maturity and distribution of stomata. Not only individual leaf age but also leaf type affects rust development: primary and trifoliolate leaves may differ substantially in susceptibility to rust infection, again attributable in part to differences in stomatal density (Allen, 1975a; Zulu and Wheeler, 1982). Furthermore, spores from old leaves and old pustules may have reduced germinability relative to those from young ones (Imhoff *et al.*, 1981).

The international dissemination of bean rust is presumed to have occurred principally by means of windborne uredospores. Secondary dispersal occurs as uredospores and is favoured by cloudy humid weather with heavy dew and temperatures in the range of 21–27°C (Schein, 1961). Uredospores, which are released and deposited frequently in clusters (Ferrandino and Aylor, 1987), are disseminated principally by wind and to a lesser extent through contact with animals including humans and their implements (Laundon and Waterston, 1965) and probably also by insects (Zaumeyer and Thomas, 1957). Uredospore production and release are also influenced by moisture and temperature (Yarwood, 1961; Imhoff *et al.*, 1982a) and apparently also by photoperiod (Cohen and Rotem, 1970). It has been estimated that *U. appendiculatus* can produce one million uredospores cm^{-1} on leaves bearing 2–100 pustules cm^{-2} (Yarwood, 1961). This spore production occurs in waves, with peaks every 3–4 days. Efficiency of

sporulation per unit of leaf area varies inversely with uredial density (Imhoff *et al.*, 1982a). The rate of radial expansion of a rust focus has been determined to be 15–16 cm day^{-1} (Habtu Assefa *et al.*, 1995). The rate of spread and severity of rust tend to decrease in cultivar mixtures (Mundt and Leonard, 1986; Davis and Panse, 1987; Aylor, 1988; Habtu Assefa *et al.*, 1995). Secondary spread of uredospores is impeded by the presence of a cereal intercropped with bean (van Rheenen *et al.*, 1981; Msuku and Edje, 1982; Moreno and Mora, 1984).

The relative importance of the different spore forms in the seasonal carry-over of bean rust apparently varies with location. The production of teliospores, which require a rest period before germinating (Zaumeyer and Thomas, 1957), is in part under environmental control and in part governed by the genotypes of both the host and the rust fungus. While teliospores may be the major source of overwintering inoculum in some areas (Milbrath, 1944), in areas where *U. appendiculatus* does not produce telia, bean rust epidemics may depend on the transport of uredospores from elsewhere (Townsend, 1939) or on the direct overwintering of uredospores (Fromme and Wingard, 1921; Marcus, 1952). In Brazil, uredospores apparently can survive in the field for only about 2 months (Zambolim and Chaves, 1974).

Crop Loss

The extent of crop loss caused by rust depends on the plant growth stage at which infection occurs and the susceptibility of the cultivar: losses are most severe when plants are infected before or during flowering. The severity of rust obviously in turn is governed substantially by environmental conditions and, at any one location, seasonal differences are pronounced. Early infection of particularly susceptible cultivars can lead to almost complete crop loss, as has been reported in the USA (Fromme and Wingard, 1921), and in white-seeded canning beans in eastern Africa (Howland and Macartney, 1966). Numerous other estimates of crop loss are reported in the literature (Stavely and Pastor-Corrales, 1989) but all too seldom are losses measured relative to agronomic constraints as a whole. In one such study (Pinstrup-Andersen *et al.*, 1976), rust was held responsible for 22% of the total crop loss from all causes. Analysis of disease progress curves (Imhoff *et al.*, 1982b) can be valuable in assessing crop loss. Leaf area index and rust severity during flowering and late pod setting have been found to give the best estimates of yield and yield loss (Habtu Assefa, 1994).

Control

Certain cultural practices are important in reducing initial infection by rust and these include: not using the same land more than once in 3 years; the burial of bean debris; not planting near stacks of old bean straw; and avoiding the use of the previous season's stakes for support of climbing beans (Milbrath, 1944; Zaumeyer and Thomas, 1957). In some areas, there may be potential for adjusting sowing date so as to minimize exposure to temperatures and dew periods that

favour infection at critical stages of crop development (Stavely and Pastor-Corrales, 1989) or to avoid spread from adjacent crops of different maturities.

In tropical areas where beans are commonly produced in complex crop associations, intercropping with cereals can afford some protection from rust (van Rheenen *et al.*, 1981; Msuku and Edje, 1982; Moreno and Mora, 1984), by acting as a barrier to uredospores or perhaps even by providing an environment wherein induced resistance might operate (Allen, 1975c; Castano and Allen, 1985). In parts of eastern and southern Africa wherein varietal mixtures of beans predominate, the maintenance of genetic diversity appears to contribute to protection against diseases, including rust (Lyimo and Teri, 1984; Davis and Panse, 1987).

Numerous chemical control measures for bean rust have been reported. Of the older fungicides, sulphur dusts have often given good control (Zaumeyer, 1946); the dithiocarbamates zineb and mancozeb as well as copper have also provided some protection, though phytotoxicity can be a problem (Oxenham, 1956; Cortado, 1969). A 7–14 day spray schedule has been recommended for preventative fungicides, including mancozeb, maneb and chlorothalonil. Materials that have shown promise include bitertanol, triadimefon and propiconazole (Stavely and Pastor-Corrales, 1989). In general, protectant fungicides tend to fail in areas where there is frequent rainfall because the deposit is washed off too soon. The persistence of fungistatic concentrations of chemicals after rainfall is directly related to the amount of fungicide initially deposited. Neely (1971) found that if the initial deposit was not twice the fungistatic deposit, then the first few centimetres of rainfall reduced it to less than the fungistatic concentration. Such shortcomings of protectant chemicals led to investigations of systemic materials.

Effective systemic activity in bean against rust has been displayed by various organic substances and antibiotics (Mitchell *et al.*, 1959; Smale *et al.*, 1961) and other substances have been found chemotherapeutic against bean rust (Davis *et al.*, 1959; Evans and Saggers, 1962). Benomyl, which remains one of the more readily available systemic fungicides, has given rather variable results with bean rust. The most important of the systemic fungicides against rust is oxycarboxin (Bates and Tweedy, 1971; Stavely and Pastor-Corrales, 1989).

While certain fungicides may be effective, the overriding factor that should regulate their use is their estimated cost-effectiveness. In tropical regions, heavy rust infections may necessitate several fungicide applications and this is often considered impracticable and uneconomic, even when technically feasible. The management decision on when to spray can be guided by information on plant maturity and rust severity. Similar observations have been made for rust of groundnut (McDonald *et al.*, Chapter 2, this volume).

There are various examples of hyperparasitism of the bean rust fungus, but their potential as biological control agents remains largely unexploited. The agents include a virus (Yarwood and Hecht-Poinar, 1973), the bacterium *Bacillus subtilis* (Ehrenb.) Cohn (Baker *et al.*, 1985) and the fungus *Verticillium lecanii* (Zimm.) Viegas (Allen, 1982). Limited evaluation of the field performance of *V. lecanii* (Grabski and Mendgen, 1985) and *B. subtilis* suggests that the bacterium may have rather greater potential in biocontrol, perhaps especially if integrated with other disease management practices.

Differences in the field susceptibility of bean cultivars to rust infection were first reported at the beginning of this century by Duggar in the USA and by Gassner in Uruguay (Fromme and Wingard, 1921). Since then, extensive selection and breeding for rust resistance have occurred in all the main bean-growing areas of the world. Resistance to rust in *P. vulgaris* is typically based on a hypersensitive reaction (Wingard, 1935) under oligogenic control (Zaumeyer and Harter, 1941) and associated with the accumulation of phytoalexins (Bailey and Ingham, 1971). Cultivars possessing hypersensitive resistance to rust usually prove transient in commercial agriculture because their resistance is race-specific, relating to the existence of a gene-for-gene relationship in which a gene for resistance in the host is matched, and overcome, by a gene for virulence in the pathogen (Christ and Groth, 1982a). Ultrastructural examination of the infection process of *U. appendiculatus* (Hardwick *et al.*, 1971; Heath, 1971; Mendgen, 1978) has revealed that there are several histologically distinct reactions within the leaf. Resistance is expressed variously, from immunity through various types of hypersensitivity with necrosis that may, or may not, support sporulation. Uredia also vary in size (Kolmer and Groth, 1984), and this range of qualitative responses has been used as the basis for developing grading scales (Stavely and Pastor-Corrales, 1989).

Quantitative resistance to rust has received rather less attention, despite the pioneering work of Fromme and Wingard (1921) who showed that there were several components of resistance, including decrease in the number of infections, smaller pustule size, and longer incubation period. Subsequent studies have confirmed that bean cultivars may differ in infection ratio (Ogle and Johnson, 1974; Allen, 1975a; Groth and Urs, 1982; Statler and McVey, 1987), latent period (van Breukelen, 1979), pustule size (Pastor-Corrales and Correa-Victoria, 1983; Statler and McVey, 1987) and sporulation capacity (Allen, 1975a; Aust *et al.*, 1984). Such components of partial resistance to rust are liable to be influenced by both environment (Imhoff *et al.*, 1982a) and host. The influence of leaf age and type on susceptibility (Zulu and Wheeler, 1982) depends in part on the density of stomata (Allen, 1975a; Groth and Urs, 1982) and in part on pubescence (Shaik, 1985; Shaik and Steadman, 1988; Mmbaga *et al.*, 1994). In the field, such components of partial resistance may contribute to 'slow rusting' or adult plant resistance (Ballantyne and McIntosh, 1976; Mmbaga and Steadman, 1991). But, in view of the proven transience of most forms of resistance as a result of their race-specificity, the crucial question is whether partial resistance is race non-specific and so likely to prove more durable. There is growing evidence that race non-specific resistance to rust in beans does exist (Ballantyne, 1974b; Allen, 1975a; Groth and Urs, 1982; Shaik and Steadman, 1988; Mmbaga and Steadman, 1992) and could perhaps be harnessed to support race-specific resistance. However, slow rusting and adult plant resistance clearly do not necessarily indicate durable resistance and these may also prove race-specific: gene-for-gene interactions can also occur in slow-rusting cultivars of wheat (Johnson, 1992) and there is now evidence (Sandlin and Steadman, 1994) that adult plant resistance to rust in beans may be race-specific in some instances. Transgressive segregants for rust resistance have been found among advanced inbred populations derived from the slow-rusting cultivars Apollo and California White Kidney

(Ballantyne and McIntosh, 1977), suggesting a polygenic inheritance of slow rusting. An innovative approach has looked at the extent of variation among races to form appressoria on surfaces of varying topography. The uniformity among diverse races in this character suggests there are opportunities for the development of race non-specific resistance based on thigmotropism, through which a leaf topography capable of providing a disruptive prepenetration signal might prevent infection (Allen *et al.*, 1991).

Genetic studies of rust resistance usually show that reaction grade is controlled by single dominant genes, of which there are many in *P. vulgaris* (Zaumeyer and Harter, 1941; Ballantyne and McIntosh, 1977; Christ and Groth, 1982a; Grafton *et al.*, 1985). Monogenic, dominant resistance genes that are effective against several races of rust have been identified; they occur in linkage groups in which there is a single gene for each of many races (Stavely, 1984c). Some genes are epistatic to others (Kolmer and Groth, 1984). Resistance expressed either as reduced pustule size (Kolmer and Groth, 1984) or as slight incompatibility (Xiang-Sheng and Deverall, 1989; Edington and Rijkenberg, 1991) is also under oligogenic control, perhaps indicating that these characters may not necessarily confer greater stability than may be expected from hypersensitive or immune reactions. Conversely leaf pubescence, which may or may not be associated with race non-specific resistance to rust, is determined by a single major gene in some crosses (Zaiter *et al.*, 1990), suggesting that there are opportunities for combining abaxial leaf pubescence with race-specific resistance through single plant selection.

A wide range of rust-resistant germplasm has been tested internationally since 1984, but as yet no single entry has remained resistant across all sites and seasons (Staveley and Pastor-Corrales, 1989). The most resistant cultivars tested include Mexico 309, Ecuador 299, Redlands Greenleaf B and C, Turrialba 1 and 4, Compuesto Chimaltenango 2 and 3 and Puerto Rico 5.

Strategies to stabilize race-specific resistance include 'gene pyramiding' which attempts to combine genes to provide protection against many races (Coyne and Schuster, 1975). More work seems warranted on a pyramiding of mechanisms of resistance, as suggested by Allen (1983). 'Gene deployment' refers to the use of certain single genes that are known to confer effective resistance to all races of rust recognized within a specific region; the cultivar Westralia provides an example (Ballantyne and McIntosh, 1977). Multiline varieties, in which each component line contains a distinct resistance gene, may also stabilize rust resistance (Coyne and Schuster, 1975) but, in tropical regions wherein pure lines are less commonly cultivated than heterogeneous landraces, the potential for effective management of rust seems especially great in varietal mixtures (Davis and Panse, 1987) and perhaps also cereal–bean intercrops, wherein induced resistance could perhaps contribute to the protective effects of such systems of cropping (Allen, 1975c). There is clear potential for the manipulation of the components of varietal mixtures (Davis and Panse, 1987) and their spatial arrangement (Mundt and Leonard, 1986; Aylor, 1988) so as to achieve a balance between protection from rust (and other agronomic restraints) and crop productivity and acceptability.

COMMON BACTERIAL BLIGHT

Aetiology

Common bacterial blight is caused by the bacterium *Xanthomonas campestris* pv. *phaseoli* (Smith) Dye. Its brown-pigmented variant is considered to cause fuscous blight (Zaumeyer and Thomas, 1957; Saettler, 1989). The bacterium was first described as *Bacillus phaseoli* in 1897 by E.F. Smith in the USA (Zaumeyer and Thomas, 1957). He renamed it *Pseudomonas phaseoli* in 1901 and *Bacteria phaseoli* in 1905 (Elliot, 1943; Zaumeyer and Thomas, 1957). In 1923, following a proposal to group all plant pathogenic bacteria in the genus *Phytomonas* (Bergey *et al.*, 1923), the name of the bacterium was changed to *Phytomonas phaseoli*. A subsequent revision by Dowson (1943) led to the creation of a new genus (*Xanthomonas*) consisting of yellow-pigmented monotrichous phytobacteria, and the common blight bacterium was renamed *Xanthomonas phaseoli* (E.F. Smith) Dowson. In 1974, Dye and Lelliot separated the genus into five species and Dye (1978) transferred the bacterium to the species *X. campestris*. Based on distinctive pathogenicity on one or more host species, Dye *et al.* (1980) placed plant pathogenic bacteria in taxa called pathovars: the common blight bacterium was accordingly named *X. campestris* pv. *phaseoli* and its fuscous variant, *X. campestris* pv. *phaseoli* var. *fuscans*. Because the two bacteria cause similar symptoms and may occur together on the same plant, in this review common and fuscous blights are treated as the same disease under the name of common blight. The bacteria are referred to as XCP and XCPF, respectively. However, a reclassification of the genus (Vauterin *et al.*, 1995) on the basis of comprehensive DNA–DNA hybridization studies now places the bean bacterial blight pathogens into *X. axonopodis* pv. *phaseoli* and its variety *fuscans*, so that our treatment may no longer be strictly correct. *X. campestris* pv. *vignicola* causes bacterial blight of cowpea and is reviewed in Allen *et al.* (Chapter 5, this volume).

Biology

Both bacteria are typical Xanthomonads: they are Gram-negative, do not form spores, are in the shape of rods measuring 0.4 × 1.0 μm and possess a single polar flagellum. On nutrient glucose or yeast dextrose calcium carbonate agar, XCP produces yellow-pigmented, smooth, convex and mucoid colonies (Dye, 1980). The yellow pigment (xanthomonadin) is due to an extracellular polysaccharide slime called xanthan, which is a brominated aryl-polyene ester (Starr *et al.*, 1977), insoluble in water but soluble in petroleum ether with absorption spectrum peaks at 418, 437 and 463 nm (Dye, 1980; Starr, 1983). XCPF produces a diffusible brown pigment on media containing tyrosine (Dye, 1980), which distinguishes it from XCP. The bacterium is strictly aerobic, metabolizes glucose and mannose and causes proteolysis of milk (Dye and Lelliot, 1974). It is catalase positive, oxidase negative, hydrolyses starch and Tween 80 (Dye, 1980) and does not induce a hypersensitive reaction on tobacco (Gilbertson *et al.*, 1990).

Common blight bacteria enter the plant passively via wounds or natural openings such as stomata and hydathodes (Zaumeyer and Thomas, 1957) and multiply in intercellular spaces dissolving the middle lamellae. Bacteria may block the vascular elements of the leaves and stem causing their disintegration and plant wilting (Santana, 1985) but do not infect the plant systemically (Haas, 1972). Macroscopically visible water-soaked spots become necrotic, enlarge and may coalesce. Bacterial exudates may appear on the surface of infected tissues and are disseminated to initiate secondary infection.

The host ranges of XCP and XCPF include *Phaseolus vulgaris*, *P. lunatus*, *P. coccineus*, *Macroptilium lathyroides*, *Vigna radiata*, *V. mungo*, *V. aconitifolia*, *V. angularis*, *V. umbellata*, *V. unguiculata*, *Pisum sativum*, *Glycine max*, *Lablab purpureus*, *Strophostyles helvola*, *Mucuna deeringiana* and *Lupinus polyphyllus* (Zaumeyer and Thomas, 1957; Vakili *et al.*, 1975; Allen, 1983; Bradbury, 1986). Bradbury (1986) disagrees with reports that soyabean and *V. radiata* are host to XCP (Vakili *et al.*, 1975) and attributes the infections to *Xanthomonas campestris* pv. *glycinea* and *X. campestris* pv. *vignaeradiatae*, respectively. However, XCP does grow epiphytically on soyabean leaves (Cafati and Saettler, 1980).

Although variation in pathogenicity of XCP isolates has been demonstrated within and among geographic regions (Schuster *et al.*, 1973; Valladares-Sanchez *et al.*, 1979), physiologic specialization on *P. vulgaris* is unknown. The available evidence indicates that the interaction between XCP and *P. vulgaris* is quantitative in nature. Conversely, it appears that certain genotypes of *P. acutifolius* differentiate races within XCP (Zapata, 1989; Opio *et al.*, 1996), having implications for resistance breeding as discussed below.

Sutton and Wallen (1970) obtained evidence of a relationship between phage type and geographic origin but Fujimoto (1985) found no such relationship. Phage typing and plasmid profile analysis have proved reliable in distinguishing XCP isolates while bacteriocin typing and polyacrylamide gel electrophoresis appear unreliable (Sutton and Wallen, 1970; Fujimoto, 1985).

Restriction fragment length polymorphism has been recently used and appears promising for the study of population structures and variability of XCP and XCPF. Initial studies (Gilbertson *et al.*, 1987; Lazo *et al.*, 1987) indicate that isolates from different geographic regions form clonal groups. Subsequent studies (CIAT, 1991; Gilbertson *et al.*, 1991) show that XCP and XCPF isolates from different regions are not clonal and that regional subpopulations do exist, having evolved from isolates that may have been introduced either via seed or with certain bean genotypes. XCP and XCPF isolates of the same geographic origin tend to have very similar RFLP patterns but, unlike the angular leaf spot pathogen, Andean and Mesoamerican groups do not appear to occur (CIAT, 1992; Otoya and Pastor-Corrales, 1992; Otoya *et al.*, 1994a). XCP and XCPF have been shown to be sufficiently different to be handled separately in breeding programmes (CIAT, 1990, 1991; R.L. Gilbertson and D.P. Maxwell, unpublished). Genetic diversity has also been shown to be greater than originally thought among populations of XCP and XCPF in Latin America (CIAT, 1991). Further investigations are required to enhance our understanding of host–pathogen interactions.

Symptoms

Common bacterial blight symptoms occur on leaves, pods and stems. On leaves, initial symptoms appear as water-soaked spots on the lower surface. As the spots enlarge the centres become necrotic. The lesions enlarge irregularly, coalesce and become surrounded by narrow chlorotic zones which turn brown (Plate 5). On susceptible cultivars, necrosis may be extensive and wind and rain shatter leaves and cause premature defoliation (Zaumeyer and Thomas, 1957).

On pods, symptoms first appear as small water-soaked spots which enlarge, turn dark reddish-brown and become slightly sunken (Fig. 4.6). Under humid conditions, a yellow slimy exudate may be produced, forming a yellow crust when dry. Heavy infection during pod and seed development may cause pods and

Fig. 4.6. Common bacterial blight lesions on bean pods (Photo: courtesy of F.J.J. Jongeleen).

seeds to shrivel. Bacteria occur on or inside seeds, on which they are sometimes symptomless (Weller and Saettler, 1980a; Aggour, 1988; Saettler, 1989). Severely infected, pale-coloured seeds bear yellow or butter-coloured lesions. Seeds may be wrinkled and spotted when less severely infected.

On stems of seedlings, symptoms often arise from seedborne infection, resulting in the destruction of growing tips and primary leaves and a condition termed 'snakehead' (Zaumeyer and Thomas, 1957). On older stems, lesions initially appear as water-soaked spots. They enlarge, become reddish in colour and may extend up the stem (Santana, 1985) or girdle it if infection is at a node (Burkholder, 1921; Zaumeyer and Thomas, 1957). The stem is weakened and may break in windy conditions.

Epidemiology

XCP survives between seasons in contaminated seed at rates up to 16% (Weller and Saettler, 1980a; Opio *et al.*, 1993). The bacterium may either be borne externally or as an internal infection, retaining viability for as long as 36 years (Saettler *et al.*, 1986; Saettler, 1989). Under certain conditions, but apparently not others, the common bacterial blight pathogen can survive in infested soil and plant debris (Gilbertson *et al.*, 1990; Opio *et al.*, 1994), with periods of survival varying from about 3 to 18 months depending on environment. Under temperate North American conditions, survival on debris may be negligible (Sutton and Wallen, 1970; Saettler *et al.*, 1986), whereas in tropical areas this means of carry-over may be significant. Survival is greater in drier environments and bacterial populations decline rapidly under moist conditions and in debris that is buried (Opio *et al.*, 1994), perhaps through increased bacterial antagonism (Habte and Alexander, 1975). The common bacterial blight pathogen also survives on weeds and non-host plants (Cafati and Saettler, 1980; Ramos, 1988; Opio *et al.*, 1992a). Epiphytic colonies have been found on a wide range of plant species in the families *Amaranthaceae*, *Commelinaceae*, *Compositae*, *Cruciferae*, *Gramineae*, *Oxalidaceae* and *Portulacaceae* in addition to various legumes. Whereas some studies suggest that, because such epiphytic populations decline rapidly, their role is probably as a source of secondary rather than primary inocula (Ramos, 1988), certain weed species including the leguminous shrub, *Senna hirsuta*, may act as reservoirs for up to 6 months (Opio *et al.*, 1992a).

Seedborne inoculum constitutes the means of international distribution of XCP (Schuster and Coyne, 1975) as well as the principal source of primary infection of susceptible common bean cultivars. The minimum population in seed that can initiate field infection is estimated at 10^2 colony-forming units; 0.2% seed infection can lead to epidemic development (Opio *et al.*, 1993). Weller and Saettler (1980b) estimated that 5×10^6 colony-forming units per 20 cm^2 was the threshold population density for the development of symptoms under North American conditions.

Disease development and secondary spread are influenced by the number of infection foci, the presence of vectors, the crop growth stage, environmental conditions and cultural practices. Among vectors are whiteflies, the borer *Diapreps*

abbreviatus (Boh.) and the beetle *Ceratoma ruficornis* (Ol.), as well as larger animals and man (Sabet and Ishag, 1969; Kaiser and Vakili, 1978; Saettler, 1989). Because infected cotyledons and primary leaves serve as sources of infection upwards through the canopy, early plant infection is thought important for the establishment and subsequent epidemic development of bacterial populations (R.L. Gilbertson and D.P. Maxwell, unpublished). However, plant susceptibility to infection generally increases from flowering to pod-filling (Coyne and Schuster, 1974c) then decreases as the crop matures (Santana, 1985). Environmental conditions that favour disease spread include warm temperatures, high relative humidity, rainfall, wind and windborne soil and irrigation water (Sutton and Wallen, 1970; Claflin *et al.*, 1973; Steadman *et al.*, 1975; Saettler, 1989). Damage is greatest at temperatures of about 28°C and *in vitro* growth is most rapid in the range of 28–32°C (Patel and Walker, 1963; Mack and Wallen, 1974; Santana, 1985). Photoperiod also influences common bacterial blight severity, damage being greater in short days; this may in part account for observed environmental effects on plant susceptibility (Coyne *et al.*, 1973; Webster *et al.*, 1983a; Santana, 1985). Cultural practices including intercropping with maize also can influence the rate of spread of common bacterial blight (van Rheenen *et al.*, 1981).

Crop Loss

Since XCP and XCPF occur together, the plant damage and yield loss caused by either bacterium cannot be ascertained and there have been few attempts to estimate the effect of common blight on yield. Yield losses of up to 38% due to natural infection were demonstrated in Canada (Sutton and Wallen, 1970) and estimated at 22 and 45% from natural and artificial infection, respectively, in Colombia (Yoshii *et al.*, 1976). In 1976, an outbreak of common blight in Michigan affected 75% of a 263,000 ha bean crop, causing an estimated yield loss of 10–20% (Saettler, 1989). Recent studies in Colombia and Uganda (CIAT, 1990, 1991) have shown yield losses of 20–47% and 40%, respectively, and work in Uganda (Opio *et al.*, 1992b) estimates that for each 1% increase in the incidence of common blight during reproductive growth there is a yield loss of 3.5–11.5 kg ha^{-1}, depending on the season.

Greater damage occurs with early plant infection due to premature defoliation, which reduces photosynthetic area, interferes with translocation of water and nutrients and reduces seed number and size (R.L. Gilbertson and D.P. Maxwell, unpublished). Lesions on pods and seeds reduce quality. In the USA, crops with pods having 4% blemishes due to bacterial blight are graded substandard and may not be harvested for seed, thereby causing substantial economic losses (Webster *et al.*, 1983b). In Uganda in 1983, an outbreak of bacterial blight at the main seed multiplication site caused the abandonment of the operation and continuing delays in release of seeds to farmers (A.F. Opio, Kampala, Uganda, 1991, personal communication).

Control

Cultural methods are important in the control of common blight. Because of the importance of seedborne infection as a source of primary inoculum, use of clean seed is a potentially effective control measure where applicable (Weller and Saettler, 1980a; Webster *et al.*, 1983b). Strict standards in seed production and use of clean seed have been the basis of successful management of common blight in the USA (Webster *et al.*, 1983b; R.L. Gilbertson and D.P. Maxwell, unpublished). Use of clean seed alone, however, does not guarantee freedom from infection in the field (Weller and Saettler, 1980a). Other useful cultural practices include: destruction or removal of plant debris or its incorporation by deep ploughing (Zaumeyer and Thomas, 1957; R.L. Gilbertson and D.P. Maxwell, unpublished); crop rotation (Saettler, 1989); effective weed control; and avoiding movement through crops when foliage is wet (R.L. Gilbertson and D.P. Maxwell, unpublished). Intercropping with maize reduces spread and incidence of bacterial blight (van Rheenen *et al.*, 1981) but non-host plants may possibly enable short-term epiphytic survival of XCP and provide a source of secondary inoculum (Santana, 1985; Saettler, 1989).

The effectiveness of chemical control of common blight is considered limited (Saettler, 1989; R.L. Gilbertson and D.P. Maxwell, unpublished), although some chemicals control foliar infection. Good control has been reported with copper oxychloride mixed with zineb or mancozeb (Maringoni, 1990), cupric carbonate, cupric sulphate (Opio, 1990), copper sulphate (Dickens and Oshima, 1969), copper hydroxide and potassium *N*-hydroxymethyl-*N*-methyldiothiocarbamate (Weller and Saettler, 1976). Their limited effectiveness, cost and the possibility of developing resistant pathotypes, particularly against antibiotics, mean that chemical control is feasible only under special circumstances such as seed multiplication or as a component of integrated control strategy.

Particularly for small-scale farmers in the tropics, where other measures may have practical limitations, host plant resistance appears the most suitable control strategy. The search for sources of resistance in *P. vulgaris* began in 1925 in the USA (Rands and Brotherton, 1925). This and subsequent efforts (Coyne *et al.*, 1963; Coyne and Schuster, 1973) have yielded only moderate levels of resistance, immunity to the disease having not yet been found in *P. vulgaris*. In evaluations of 12,000 germplasm accessions, only 39 showed moderate levels of resistance (CIAT, 1988). Most of these were from the Andean gene pool (Singh, 1989) and none was as well-adapted as bred lines. Resistant lines like Great Northern Nebraska No. 1 selection 27 and PI 207262, bred in temperate regions (Coyne and Schuster, 1976), have been used to improve foliar resistance in the tropics. But the poor adaptation and instability of seed colour of these lines and their progenies have limited their use (Beebe and Pastor-Corrales, 1991). However, some success has been achieved by combining other novel sources of resistance (CIAT, 1990). High levels of resistance have been transferred from the tepary bean, *P. acutifolius*, (Schuster, 1955; Coyne and Schuster, 1974a) to *P. vulgaris* (Honma, 1956; McElroy, 1985). Near-immune self-fertile lines, compatible with *P. vulgaris* have been obtained from some crosses

(McElroy, 1985) and resistant lines with different growth habits and grain types have been identified, such as XAN 159, XAN 160 and XAN 161 (Beebe and Pastor-Corrales, 1991).

Studies of the inheritance of resistance to XCP have given variable results due to variation in methodology and parents used (Saettler, 1989; Beebe and Pastor-Corrales, 1991). Resistance is essentially quantitative (Coyne *et al.*, 1973) but often with dominance and epistatic effects (Rava *et al.*, 1987; Silva, 1988). In bean genotypes such as PI 207262, foliage reactions are resistant, while pods are susceptible (Coyne and Schuster, 1974b). One, two or three genes appear to confer resistance depending on sources and methods of evaluation (McElroy, 1985; Drijfhout and Blok, 1987; Scott and Michaels, 1988; Silva, 1988).

The mechanisms of host–pathogen interaction are not well understood but polysaccharides produced by the bacterium appear to interfere with water and nutrient movements (Corey and Starr, 1957; Zaumeyer and Thomas, 1957). Recent studies indicate that pathogenicity is a complex trait governed by genes and/or operons (Daniels *et al.*, 1988) but further investigation is required. Phytoalexins are apparently not involved in resistance (Wyman and van Etten, 1982).

There is evidence (Opio *et al.*, 1992b) that crop loss is negligible in existing resistant lines (e.g. XAN 112), so current levels of resistance within *P. vulgaris* may confer sufficient protection against bacterial blight. Since such resistance is quantitative, it is likely to be durable. If improved techniques of hybridization open the way towards fuller use of the greater levels of race-specific resistance in the tepary bean, in which it is under oligogenic control, this could introduce a form of resistance that might invite concomitant specificity in pathogen populations and thus pave the way to epidemiological instability (Opio *et al.*, 1996).

HALO BLIGHT

Aetiology

Halo blight is caused by the bacterium, *Pseudomonas savastanoi* pv. *phaseolicola* (Gardan *et al.*, 1992) though it is still widely referred to as *Pseudomonas syringae* pv. *phaseolicola*, belonging to the family *Pseudomonadaceae*. It was first described by Burkholder (1926) and soon recognized to be the major bacterial disease of common bean (Burkholder and Zaleski, 1932) in temperate regions and above medium altitudes in the tropics.

Burkholder (1926) named the causal organism *Bacterium medicaginis* var. *phaseolicola*, then later referred to *Phytomonas medicaginis* var. *phaseolicola*. Based on host specificity, Schroth *et al.* (1971) proposed that *Pseudomonas phaseolicola*, *Ps. glycinea* and *Ps. mori* be considered a single species, *Ps. syringae*, with pathovar groups corresponding to the original species. This was implemented following the establishment of international naming standards by Dye *et al.* (1980) and the bacterium causing halo blight of common bean became known as *Pseudomonas syringae* pv. *phaseolicola*. Recent studies of DNA relatedness among pathovars of *Ps. syringae* have led to the proposal that the halo blight pathogen be re-classified as *Ps. savastanoi* pv. *phaseolicola* (Gardan *et al.*, 1992).

On agar, bacterial colonies are white to cream in colour with a bluish hue, frequently producing a green fluorescent pigment (Weber, 1973). Individual cells are rod-shaped, 1.5 μm long and 0.7–1.2 μm in diameter and have at least one polar flagellum (Kreig and Holt, 1984). The bacterium is Gram-negative, strictly aerobic and does not require growth factors (Schwartz and Galvez, 1980). Optimal temperatures for growth are 20–23°C.

Biology

The first record of an alternative host of the halo blight pathogen was that of Hedges (1927), who isolated a bacterium from *Pueraria thunbergiana* that was later identified as *Ps. syringae* pv. *phaseolicola*. Since then, natural infections of halo blight have been recorded on several other legume species (Table 4.6), all members of the tribe *Phaseoleae* including *Macroptilium atropurpureum* (Lenné, Chapter 13, this volume) of the subfamily *Papilionoideae* with the exception of the *Desmodium* spp. (*Desmodieae*) and *Pisum sativum* (*Vicieae*). Apart from the studies of Teverson (1991), most records have stemmed from chance field observations and it seems likely that the list will lengthen as more extensive research is undertaken. A larger number of species has been experimentally infected with halo blight (Table 4.7), within the same tribes as those found naturally infected.

Table 4.6. Species other than *P. vulgaris* reported naturally infected with *Pseudomonas savastanoi* pv. *phaseolicola* and the races of the pathogen.

Natural alternate hosts	Countries	Races of pathogen[1]	References[2]
Cajanus cajan	Australia, Ethiopia, Tanzania, Zambia	N,1,5	4,8,10,11
Centrosema spp.		N	9
Desmodium spp.	Rwanda, Tanzania	4,7	11
Dolichos spp.	Tanzania	8	11
Glycine max	USA, Tanzania	N,4	2,10
Lablab purpureus	Kenya, Tanzania, Ethiopia	N,5–8	5,11
Macroptilium atropurpureum	Australia	2,7	11
Neonotonia wightii	Kenya, Tanzania, Australia, Zimbabwe	1,5–7	7,11,12
Phaseolus acutifolius	Kenya	2,7	11
P. coccineus	UK, Rwanda, Italy	1,4,6	11
P. lunatus	Ethiopia, Madagascar	1,2	11
Pisum sativum		N	3
Pueraria thunbergensis		N	1
Sphenostylis stenocarpa		N	6
Vigna angularis	New Zealand	7	11
V. radiata	Tanzania, USA	1,4	10,11

[1]Races of *Ps. savastanoi* pv. *phaseolicola*: N = not known.

[2]References: 1. Hedges (1927); 2. Zaumeyer and Thomas (1957); 3. Guthrie *et al.* (1965); 4. Allen (1979); 5. Ebbels and Allen (1979); 6. Birch *et al.* (1981); 7. Mitchell *et al.* (1982); 8. Allen (1983), p. 237; 9. Schultze-Kraft and Keller-Grein (1985); 10. Gondwe (1991); 11. Teverson (1991); 12. Mabagala and Saettler (1992a).

Table 4.7. Reactions of legume species to artificial inoculation with races of *Pseudomonas savastanoi* pv. *phaseolicola* – summarized from Patel and Walker (1964, 1965) and Teverson (1991).

	Races of pathogen[1]					
Species tested	1	2	3	4	5	6
Centrosema pubescens	–	+	+	+	+	–
Desmodium intortum	–	–	–	–	I	I
Glycine max	–	–	–	–	–	–
Lablab purpureus	+	+	+	I,+	I	I,+
Macroptilium atropurpureum	I,+	+	I,+	+	+	+
M. geophilum	+	+	+	+	+	+
M. lathyroides	+	+	+	+	N	+
Neonotonia wightii	+	+	+	+	+	+
Phaseolus aborigineus	–	+	–	–	+	–
P. acutifolius	–,+	–,+	+	–,I,+	–	+
P. bracteatus	+	+	N	N	N	N
P. cerastiformes	+	+	+	+	–	+
P. coccineus	I,+	–,+	–	–	–	+
P. filiformis	–	+	–	–	–	–
P. formosus	N	–	+	N	+	N
P. leucanthius	+	+	+	+	+	+
P. lunatus	+	+	+	+	+	+
P. polyanthus	+	+	N	N	N	N
P. polystachyus	+	+	N	N	N	N
P. retusus	–	–	–	–	–	–
P. tuberosus	+	+	+	+	+	+
Pisum sativum	–	–	N	N	N	N
Vicia faba	–	–	–	–	–	–
Vigna aconitifolia	–	–	N	–	–	–
V. angularis	–,I,+	–,+	+	+	+	+
V. mungo	–	–	–	–	–	–
V. radiata	–,+	–,+	–	–	–	–
V. trilobata	–	–	–	N	+	+
V. umbellata	–	–	–,I	I,+	–	–,I
V. unguiculata	–	–	–	–	–	–

[1] Reactions: + = susceptible; I = intermediate; – = resistant or nearly resistant; N = not tested.

For many years, *Ps. savastanoi* pv. *phaseolicola* was considered to exist as two races (1 and 2), distinguished by their reactions on cv. Red Mexican (and similar genotypes), which are resistant to race 1 and susceptible to race 2 (Walker and Patel, 1964b). Subsequently, isolates with different reaction patterns on other genotypes were reported in the USA by Coyne *et al.* (1979), in Kenya by Stoetzer *et al.* (1984a), in Malawi by Msuku (1985), in eastern and southern Africa by Taylor and Teverson (1985) and in southern Tanzania by Gondwe (1991, 1992). An isolate of Taylor and Teverson (1985) induced strong hypersensitive reactions on the cvs. Tendergreen and Cascade (highly susceptible to races 1 and 2) and was designated race 3.

Following more extensive collection, Teverson (1991) and Taylor *et al.* (1996a) distinguished nine races based on their reactions on eight *Phaseolus* differentials. Race 6 (28% out of 175 isolates) was most common. Other races found in appreciable frequencies were races 4 (17%), 1 (12%), 2 and 7 (both 9%) and 8 (7%). Races 1, 2, 6 and 7 were worldwide in distribution, while races 3, 4, 5 and 8 were restricted to eastern and southern Africa. Mabagala and Saettler (1992a) have since found halo blight races 1 (45%), 2 (52.5%) and 3 (2.5%) on common bean in northern Tanzania, and races 1 (22%), 2 (12%), 6 (4%), 8 (60%) and 9 (2%) are now reported from South Africa (Fourie, 1995). Race 6 appears identical with the 'new virulent race' distinguished by Coyne *et al.* (1979) based on differences in aggressiveness. Several of the alternative hosts of the halo blight pathogen also show clear race differentiation (Table 4.7), which may involve resistance genes identical with those in *P. vulgaris* (Teverson, 1991).

Differences in aggressiveness have also been demonstrated, evidently associated with variation in the levels of a toxin, phaseotoxin, which suppresses production of antibacterial phytoalexins. The reader is referred to reviews of Allen (1983) and Schwartz (1989b) for more detailed discussion of this topic.

Symptoms

Halo blight is characterized by small, water-soaked lesions, which appear on the leaves, 3–5 days after infection and quickly develop greenish-yellow haloes (Plate 6). Pods, stems and petioles also exhibit water-soaked lesions, sometimes producing a whitish exudate. Systemic infection causes general chlorosis and stunting and distortion of plant growth. The general chlorosis, and the haloes around lesions, appear to be caused by phaseotoxin (Hoitink *et al.*, 1966). Infected seeds may be symptomless, wrinkled or have buttery-yellow patches on the seed coats. Seedlings emerged from infected seeds exhibit stem girdling and rotting at the nodes (Schwartz *et al.*, 1978).

Epidemiology

The importance of halo blight derives from the rapidity of its dispersal in favourable conditions. From initial sources of infection, halo blight is disseminated by water-splash and wind occurring during rainfall (Walker and Patel, 1964a) or sprinkler irrigation (LeBaron *et al.*, 1977). The bacterium invades the plant through wounds and natural openings and, in the presence of dew, can multiply rapidly on leaves, flowers, pods and stem internodes (Stadt and Saettler, 1981; Legard and Schwartz, 1987). In Wisconsin, Walker and Patel (1964a) recorded new infections as far as 26 m from the primary source of inoculum.

Seed infection, first reported by Burkholder (1930), is probably the most important means of transmission of the halo blight pathogen between seasons. Most recent knowledge of the process is due to Taylor *et al.* (1979). Seed infection occurs through direct contact with pod lesions, so symptomless pods or sections of pods produce healthy seeds. An infected seed harbours up to 3.7×10^7 bacteria

which are found mainly in the inner, parenchymatous layer of the seed coat or on the surface of the embryo. The viability of bacteria in seeds declines only slowly during storage: viable bacteria were isolated from seeds after 3 years of storage in ambient conditions and after 6 years in controlled conditions. Nearly 90% of seedlings infected are from seeds with slight or no symptoms, so seed sorting is unreliable as a control measure. In Wisconsin, as few as 12 infected seeds per acre were sufficient to cause an epidemic (Walker and Patel, 1964a) and in the UK a tolerance level of one infected seed in 5 kg has been estimated (Wharton, 1967).

Plant residues (either in the field or adhering to the seed) appear to be less important agents of transmission, because inoculum is less and the bacterium survives poorly in unfavourable conditions. In dry infected bean leaves, bacteria remained viable for over 12 months at 24°C but, in moist leaf–soil mixtures in warmer temperatures survival was reduced to less than 6 days (Natti, 1967). In Tanzania, survival of the pathogen varied with race, location, depth in the soil and bean genotype (Mabagala and Saettler, 1992a). In standing plants and buried debris in a banana/coffee environment (Lyamungu on the lower slopes of Mount Kilimanjaro), race 1 survived for 1–2 months and race 2 for 2–3 months. In contrast, bacteria were still viable 9 months after passage through sheep which consumed infested plant debris (Starr and Kercher, 1969).

Grogan and Kimble (1967) demonstrated disease transmission in externally contaminated seeds in artificial conditions but, in the field, Guthrie (1970) obtained transmission only with very high inoculum rates and Taylor *et al.* (1979) found 100 to 1000 times fewer bacteria on externally contaminated than in naturally infected seeds.

Weedy and cultivated alternative hosts (Table 4.6) may also transmit the halo blight pathogen. For example, *Neonotonia wightii*, which is widespread in eastern and southern Africa where it is native, almost always shows symptoms of halo blight and is presumed to serve as a perennial reservoir (Mabagala and Saettler, 1992b) from which it has been shown to spread to beans (Teverson *et al.*, 1993).

Crop Loss

Under experimental conditions, yield losses of 43% have been reported by Taylor (1969) in the UK and 23–43% by Saettler and Potter (1970) (cited in Schwartz and Galvez, 1980) in Michigan in the USA. In green beans, reduction of quality due to pod lesions may be economically as important as yield loss (Taylor and Dudley, 1977a). Although losses due to halo blight in Africa have not been properly quantified, the present authors have recorded many cases of common bean crops devastated by halo blight, notably in Lesotho, Rwanda and Zimbabwe.

Control

Some control of the disease has been achieved by use of copper formulations or streptomycin applied as foliar sprays or seed dressings (Ralph, 1976; Taylor and

Dudley, 1977a, b; Legard and Schwartz, 1987; Autrey and Saumtally, 1992). Antagonistic bacteria have been reported (Adam and Pugsley, 1935) but biocontrol potentials seem uninvestigated. Evidence on the effects of cropping system is conflicting. In Kenya, van Rheenen *et al.* (1981) reported less halo blight in maize–bean associations than in sole-cropped bean, an observation confirmed in Malawi (Msuku and Edje, 1982). In Tanzania, Mabagala and Saettler (1992c) found a denser population of the halo blight pathogen in bean intercropped with maize than in sole-cropped bean. In the USA, the severity of halo blight has been much reduced by seed production under arid conditions, notably in Idaho (Myers, 1992). Chemicals, field inspection, roguing and pod sorting help to ensure that seeds are free of infection but contamination can occur even when halo blight is negligible (Stadt and Saettler, 1981). Because seed sorting is unreliable, various tests are used to detect infected seeds, including a specific bacteriophage, serology, inoculation of excised cotyledons, ultra-violet fluorescence and *Staphylococcus aureus* agglutination (see Taylor *et al.*, 1979; Walkey *et al.*, 1990).

As with other diseases, host plant resistance is considered the most cost-efficient method of control of halo blight and, in the past two decades, a considerable amount of work has been devoted to searches for sources of resistance, notably in Nebraska (NSU), France and Bulgaria and, more recently, in the UK (HRI, Wellesbourne) and Colombia (CIAT). The resistance used in breeding common bean for resistance to halo blight traces back to two main sources: cv. Red Mexican (resistant to race 1) (Jensen and Goss, 1942) and PI 150414 (resistant to races 1 and 2) (Patel and Walker, 1965). Walker and Patel (1964b) and Patel and Walker (1965) concluded that resistance to race 1 was due to a single dominant gene and this was confirmed by Taylor *et al.* (1978) and Innes *et al.* (1984). Resistance to race 2 has been found to be recessive (Patel and Walker, 1966; Taylor *et al.*, 1978), partially dominant (Taylor *et al.*, 1978), dominant (Hill *et al.*, 1972) or polygenic (Innes *et al.*, 1984).

More recently, based on tests of F_2 populations of crosses among seven differential cultivars against nine races of halo blight, Teverson (1991) and Teverson *et al.* (unpublished) explained the interaction between races of *Ps. savastanoi* pv. *phaseolicola* and genotypes of *P. vulgaris* in terms of a gene-for-gene relationship involving five matching gene pairs (Table 4.8). The resistance genes R1, R2 and R4 are dominant and R5 is recessive. R3 (also dominant) appears to be duplicated at different loci. There is evidence that R3 is identical or very closely linked to the dominant I gene conferring resistance to Serotype B of bean common mosaic virus (see p. 229) since there is complete co-segregation of resistance and both produce enhanced hypersensitive reactions in incompatible combinations (Teverson, 1991; Taylor *et al.*, 1995). The gene also appears to confer resistance to *Ps. syringae* pv. *syringae* and four other legume viruses (Kyle *et al.*, 1988). This represents a case of true multiple resistance, the implications of which are discussed further in the section on bean common mosaic virus later in this chapter. Teverson (1991) and Taylor *et al.* (1996b) also found sources of non-specific resistance to the halo blight pathogen. Some of these trace back to the original race 2 found in PI 150414 and others may be new sources. They suggest combining race-specific and non-specific resistances to increase the chances of producing durable resistance to the halo blight pathogen. For Africa, when R3 is the

Table 4.8. Gene-for-gene relationships between *Phaseolus* differentials and races of *Pseudomonas savastanoi* pv. *phaseolicola* (adapted from Teverson, 1991).

		Races/avirulence genes								
Differential cultivars	Resistance genes	1 1	2 2,5	3 3	4 2,3	5 1,2,	6 4	7 1,2	8 5	9 1,5
Canadian Wonder		+	+	+	+	+	+	+	+	+
A52 (ZAA 54)	4	+	+	+	+	−	+	+	+	+
Tendergreen	3	+	+	−	−	+	+	+	+	+
Red Mexican UI31,4		−	+	+	+	−	+	−	+	−
1072	2	+	−	+	−	−	+	−	+	+
A53 (ZAA 55)	3,4	+	+	−	−	−	+	+	+	+
A43 (ZAA12)	2,3,4,5	+	−	−	−	−	+	−	−	−
Guatemala 196-B	3,4	−	+	−	−	−	+	−	+	−

source of specific resistance, the recessive *bc-2*^{2} gene will also be needed to provide protection against 'necrotic' strains of BCMV, which can induce 'black root' on genotypes possessing the '*I*' gene, as discussed in the next section of this chapter.

BEAN COMMON MOSAIC AND BLACK ROOT

Aetiology

Bean common mosaic virus (BCMV) was first described, as bean mosaic virus, in the USA by Stewart and Reddick (1917) and is now known to occur in all regions where common bean is grown (Galvez and Morales, 1989a). The virus has flexuous filaments 720–770 nm long and 12 nm in diameter (Morales and Bos, 1988). Based on the structure of virus-induced inclusions, it is a member of Subdivision I of the potyvirus group (Edwardson, 1974). Recent work has led to its placement in a subgroup of its own (Anonymous, 1994). Two serotypes, A and B, of BCMV are recognized (Wang *et al.*, 1984). Further investigation both of the serological relationships and coat protein sequence data has revealed that BCMV comprises two distinct viruses (McKern *et al.*, 1992; Vetten *et al.*, 1992; Khan *et al.*, 1993) that correspond to the two serotypes: serotype A embraces the temperature insensitive, necrosis-inducing strains (Drijfhout, 1978), now named bean common mosaic necrosis virus (BCMNV) (Anonymous 1994); serotype B isolates, which do not normally induce necrosis except at high temperature, retain the name BCMV that now also embraces strains of blackeye cowpea mosaic, peanut stripe, azuki bean mosaic and certain virus isolates from soyabean. For the purposes of this chapter, however, all strains are referred to as belonging to bean common mosaic virus; the reader should bear in mind that further reference to serotype B isolates relates to the newly defined BCMNV.

While serotype B strains of BCMV have been considered more common than the 'necrotic' strains, in recent years the latter have caused epidemics in parts of the USA (Kelly *et al.*, 1982, 1984; Hampton *et al.*, 1983; Provvidenti *et al.*, 1984; Myers *et al.*, 1990) and Canada (Tu, 1986a), and recent surveys reveal their predominance in eastern and southern Africa, with the exception of Ethiopia (Silbernagel *et al.*, 1986; Edington and Whitlock, 1988; Vetten and Allen, 1991; Spence and Walkey, 1994, 1995).

Biology

The known natural host range of BCMV is confined to a few legumes of subtribe *Cassieae* of subfamily *Caesalpinioideae* and subtribes *Crotalarieae*, *Genisteae* and *Phaseoleae* of the *Papilionoideae* (Table 4.9). The distribution of the 'necrotic' strains of BCMV and the occurrence in wild legumes of strains which diverge from typical BCMV strains (Spence and Walkey, 1994, 1995) in their host ranges and symptoms induced on *P. vulgaris* suggest that the 'necrotic' strains of BCMV

Table 4.9. Species reported naturally infected with BCMV and the strains of the virus.

Hosts	Countries	Isolates	Serotypes[1]	Pathotypes	References[2]
Cassia hirsuta	Uganda	465	B	I	9
Cassia hirsuta	Uganda	197	A	VIa	8
Cassia sophera	Rwanda	820,830,836	A	Novel	9
Crotalaria comanestiana	Kenya	963	B	Novel	9
Crotalaria incana	Uganda	28,30	A	Novel	9
Crotalaria juncea	India	ND	ND	ND	3
Crotalaria striata	India	ND	ND	ND	5
Centrosema pubescens	Uganda	741	A	VIa	8
Glycine max	Uganda	38	A	Novel	9
Lupinus luteus	Poland	ND	ND	ND	6
Macroptilium atropurpureum	Uganda	499	A	VIa	9
Phaseolus acutifolius	Kenya	ND	ND	ND	7
Rhynchosia minima	Colombia	ND	ND	ND	4
Rhynchosia sp.	Malawi	145	B	IVb	9
Vigna mungo	Kenya	ND	ND	ND	7
Vigna radiata	Iran	ND	ND	ND	2
Vigna unguiculata	USA	ND	ND	ND	1
Vigna unguiculata	Rwanda	531	A	VIa	9
Vigna vexillata	Kenya	956	B	Va	9
Vigna vexillata	Uganda	308	A	VIa	8

[1] Strains: A and B are serotypes; ND indicates not determined.

[2] References: 1. Zaumeyer and Thomas (1957); 2. Kaiser and Mossahebi (1974); 3. Singh and Singh (1977); 4. Meiners *et al.* (1978); 5. Sarkar and Kulshreshthra (1978); 6. Frencel and Pospiesczny (1979); 7. Bock *et al.* (1980); Sengooba *et al.* (1993); 8. Spence and Walkey (1995).

may have originated on wild legumes in eastern Africa (Spence and Walkey, 1991b, 1992a, 1995). Whether or not this is so, the ability of 'necrotic' strains of BCMV to infect wild legumes has important implications for the epidemiology of BCMV and breeding for resistance against it.

Among legumes, species of *Cajanus*, *Canavalia*, *Cassia*, *Centrosema*, *Cicer*, *Crotalaria*, *Cyamopsis*, *Indigofera*, *Glycine*, *Lens*, *Lupinus*, *Macroptilium*, *Melilotus*, *Phaseolus*, *Rhynchosia*, *Sesbania*, *Trifolium*, *Trigonella*, *Vicia* and *Vigna* have been experimentally infected with BCMV (Morales and Bos, 1988; Spence and Walkey, 1992b). Apart from *Cassia*, some species of which are now referred to *Senna* and which belongs to the *Caesalpinioideae*, all of these are members of several tribes of the subfamily *Papilionoideae*. Tests of some of these legumes against six strains of BCMV suggest a host–pathogen specificity similar to that found within *Phaseolus vulgaris* (Spence and Walkey, 1992b; Table 4.10). In several cases, sap from inoculated plants without symptoms reacted positively to BCMV antisera.

Symptoms

The symptoms of BCMV on common bean are nicely described and illustrated in colour by Morales (1989). Typical common mosaic leaf symptoms comprise distinct dark green sectors on a lighter green background, usually accompanied by downward curling of the leaf margins. In cases of more severe infection, there may be leaf distortion and blistering, stunting of growth (Fig. 4.7) and distortion of flowers and pods.

'Black root' appears first on young trifoliolate leaves as local lesions with diffuse reddish-brown lamina overlain by a reticulate pattern of darkened veins. This often leads to systemic infection, first appearing as apical necrosis (Fig. 4.8) then progressing through discoloration of the stems and vascular tissues to wilting and death. Two other characteristic symptoms, 'ring-shaped' and 'pin-point' local lesions, enable the recognition of certain host genotypes and are thereby important in breeding methodology.

Epidemiology

Seed-transmission of BCMV was first demonstrated by Reddick and Stewart (1919). Between 10 and 30% of the seeds of infected plants carry the virus, which is located mainly in the embryo (Quantz, 1961; Provvidenti and Cobb, 1975). The virus is not transmitted in seeds of genotypes with the dominant 'I' gene. Seed transmission is less if plants are infected 30 or more days after sowing (Morales and Castano, 1987).

The virus is transmitted, in a non-persistent manner, by several aphid species, including *Acyrthosiphon pisum* (Harris), *Myzus persicae* (Sulzer) and *Aphis fabae* Scopoli (Kennedy *et al.*, 1962; Zettler and Wilkinson, 1966). Other aphid vectors include *Aphis gossypii* Glover, *A. medicaginis* Koch, *A. rumicis* L., *Hyalopterus atriplicis* Davis, *Macrosiphum ambrosiae* (Thomas), *M. pisi* (Kaltenbach) and *M. solanifolii* (Ashmead) = *M. euphorbiae* (Thomas) (Zaumeyer

Table 4.10. Reactions of species to artificial inoculation with strains of BCMV (Spence and Walkey, 1992b; N.J. Spence and D.G.A. Walkey, unpublished data).

	Strains of BCMV[1]					
Host species tested	NL1	NL3	NL4	NL6	NL8	NY15
Cajanus cajan	–	–/E	–	–/E	–	N
Cassia didymobotrya	–	–	N	–	–	–
Cassia hirsuta	M/E	M/E	E	M/E	–	N
Cassia occidentalis	–	M/E	N	–	M/E	–
Cassia sophera	–	–/E	N	–/E	–/E	–
Centrosema plumieri	–	–	–/E	–	–/E	N
Centrosema pubescens	E/M	M/E	M/E	M/E	M/E	N
Crotalaria anagyroides	V/E	V/E	V/E	V	V/E	N
Crotalaria goreensis	–	–	–/E	–	–	N
Crotalaria incana	–	V/E	I	C	V/E	–
Crotalaria juncea	–	–/E	–	–	–	N
Crotalaria laburnifolia	–	–	N	–	–	–
Crotalaria lanceolata	–/E	V/E	–/E	–/E	–/E	N
Crotalaria ochroleuca	M/E	M/E	–/E	–/E	–/E	N
Crotalaria retusa	–	–	–/E	–	–	N
Crotalaria verrucosa	–	–/E	–	–/E	–	N
Crotalaria spp.	–	–/E	N	–	–	–
Desmodium heterocarpon	–	–	–	–	–	N
Desmodium triflorum	–	–	–	–	–	N
Glycine max	–	C/E	–/E	–	–/E	N
Glycine tomentella	–	–/E	–	–	–/E	N
Indigofera hirsuta	–/E	–/E	–/E	–/E	–/E	N
Macroptilium atropurpureum	–	–	–	–	M	N
Parkinsonia aculeata	–	–	N	–	–	N
Rhynchosia diversifolia	–	–/E	–	–	–/E	N
Rhynchosia edulis	–	–/E	–	–	–	N
Rhynchosia minima	M/E	B/E	B/E	B/E	B/E	N
Rhynchosia sublobata	–	–	–	–	–	N
Vigna angularis	S	–/E	–/E	S/E	–	N
Vigna radiata	–	S	–	S/E	–	N
Vigna unguiculata	–	–/E	–	–/E	–/E	N

[1]Reactions: – = no reaction; E = positive ELISA reaction; S = systemic mottle; V = systemic vein banding; C = systemic chlorosis; M = systemic mosaic; B = systemic necrosis (black root); N = not tested.

and Thomas, 1957). Sohati *et al.* (1992) observed *Toxoptera citricidus* (Kirkaldy), *Brevicoryne brassicae* (L.) and *Tetraneura nigriabdominalis* (Sasaki) on bean in farmers' fields in Zambia, though their vector status is not established. The black bean aphid (*A. fabae*) appears to be the most important vector of BCMV in eastern and southern Africa (Khaemba and Latigo, 1981; Remaudiere and Autrique, 1985; Sohati *et al.*, 1992). Necrotic strains of BCMV obtained from wild legumes in eastern Africa have been shown to be seedborne and aphid-transmissible both

Fig. 4.7. Mosaic symptoms on bean foliage due to BCMV (Photo: courtesy of CIAT).

within wild legume species and between such species and common bean, and it has been suggested that wild legumes may act as important reservoirs of infection in such areas (Sengooba, 1994; Spence and Walkey, 1995). BCMV is also readily transmitted by mechanical means, facilitating screening for host plant resistance.

Crop Loss

Crop losses caused by BCMV depend on the genotype of the host, the aggressiveness of the strain of virus, the timing and source of infection and the environment (Burke and Silbernagel, 1974). Yield losses of 50% have been recorded in Morocco (Lockhart and Fischer, 1974) and up to 68% in Oregon (Hampton, 1975), though cases of total crop failure have been reported from Latin America (Galvez, 1980).

The number of seeds per pod and pods per plant, leaf number, plant height and seed size have all been reported as adversely affected by BCMV infection. It has been estimated that for each unit increase in mosaic severity score (on 1–9 scale), there is a concomitant loss in seed yield of 213–321 kg ha^{-1} in bean cultivars that carry no resistance genes. Significant BCMV strain effects on mosaic

Fig. 4.8. Black root symptoms on bean plant due to BCMV (Photo: courtesy of D.J. Allen).

severity, plant stunting, leaf number, the number of seeds per pod, seed yield and rates of seed-transmission have been demonstrated, and isolates of pathotype VIa (to which the NL3 strain belongs) appear to be the most aggressive. In one instance, infection with a mixture of two isolates (pathotypes IVb and VIa) was shown to lead to a greater loss in the number of seeds per pod than was caused by either pathotype alone (Mukoko, 1992; Sengooba, 1994).

Control

Losses due to BCMV may be in principle curtailed through chemical control of the vectors (Schwartz *et al.*, 1978), timely sowing of crops (Burke, 1964), use of

optimum plant densities (Sithanantham *et al.*, 1992; Sohati *et al.*, 1992), sowing in association with maize (Katunzi *et al.*, 1987; Sithanantham *et al.*, 1992) and use of clean seeds, but host plant resistance remains the most effective means of limiting the damage caused by the virus.

Two main sources have featured in breeding for resistance to BCMV: the cv. Robust developed by Spragg in Michigan in 1915 (Spragg and Down, 1921); and Corbett Refugee, selected by R.D. Corbett in 1931 (Pierce and Walker, 1933). The resistances were soon shown to be distinct (Pierce, 1935) but it remained for Ali (1950) to show that, while both were conferred by single genes, the resistance of Robust (which he designated '*a*') was recessive and that of Corbett Refugee ('*I*') was dominant. Silbernagel (1969) noted that the '*I*' gene conferred effective resistance to all known strains of BCMV in the USA. However, the systemic necrosis known as 'black root' had been recorded in Corbett Refugee as early as 1938 (Jenkins, 1939) and Grogan and Walker (1948) induced 'black root' in genotypes possessing the '*I*' gene at temperatures in excess of 30°C; subsequently BCMV strains were identified in Europe that were capable of causing 'black root' at temperatures between 15 and 20°C (Hubbeling, 1969).

Based on a study of the reactions of host genotypes inoculated with an extensive collection of BCMV strains, Drijfhout *et al.* (1978) distinguished seven groups of strains, best regarded as pathotypes, and recommended a standard set of differential cultivars. The reactions of the differential cultivars against the type strains and their genetic constitutions are summarized in Table 4.11 (Drijfhout, 1978).

Table 4.11. Genetic interactions between BCMV strains and selected *Phaseolus vulgaris* cultivars (adapted from Drijfhout, 1978; Morales, 1989).

Differential cultivars	Resistance genes	Serotype B				Serotype A		
		Type	Florida	NY15	NL4	NL8	NL3	NL5
A. Cultivars with recessive alleles (*bc-u bc-1*) of the necrosis gene								
DW	*I+*	M	M	M	M	M	M	M
Imuna	*I+ bc-u bc-1*	–	M	M	M	–	M	M
RG B	*I+ bc-u bc-1*^{2}	–	M	–	M	–	M	M
Sanilac	*I+ bc-u bc-2*	–	–	M	–	M	M	M
Pinto 114	*I+ bc-u bc-1 bc-2*	–	–	M	–	–	M	M
GN 31	*I+ bc-u bc-12 bc-2*^{2}	R	R	R	M	R	R	R
IVT 7214	*I+ bc-u bc-2 bc-3*	–	–	–	–	–	–	–
B. Cultivars with dominant alleles (*II*) of the necrosis gene								
Widusa	*I*	–	–	–	–	B	B	B
Top Crop	*I bc-1*	–	–	–	–	–	B	B
Amanda	*I bc-1*^{2}	–	–	–	–	–	–	B
IVT 7233	*I* (*bc-1*^{2}) *bc-2*^{2}	–	–	–	–	P	P	P

Cultivar groups: DW = Dubbele Witte; RG B = Redlands Greenleaf B; GN = Great Northern.

Reactions: – = no reaction; M = mosaic; B = 'black root'; R = 'ring-shaped' lesions; P = 'pin-point' lesions.

When inoculated on to cultivars which lack the '*I*' gene (Subset A, Table 4.11), virulent strains predominantly induce mosaic. The exceptions are cultivars of the Great Northern group, which exhibit 'ring-shaped' lesions, and cultivars of the IVT 7214 group, which are immune. A series of recessive genes (*bc-1*, *bc-1*^{2}, *bc-2*, *bc-2*^{2}, *bc-3* and *bc-u*) is responsible for differential reactions between cultivars and strains (*bc-1*^{2} is the same as '*a*' in Robust while *bc-1* and *bc-1*^{2} and *bc-2* and *bc-2*^{2} are allelic). The 'ring-shaped' lesions induced in the Great Northern 31 group of cultivars are probably due to the presence of *bc-2*^{2}, while *bc-3* confers the immunity of the IVT 7214 group of cultivars. The *bc-u* gene, which was also found by Innes and Walkey (1980) in all cultivars they tested, is non-specific but complements the actions of the other recessive resistance genes.

When inoculated on to cultivars which have the '*I*' gene (Subset B, Table 4.11), virulent strains (NL8, NL5 and NL3, representative of pathotypes III, VIb and VIa, respectively) induce 'black root'. In combination with the '*I*' gene, the recessive *bc-1* confers resistance to pathotype III (NL8) and *bc-1*^{2} confers resistance to pathotypes III and VIb, while all three groups of 'necrotic' strains induce only pin-point lesions in the presence of '*I*' and *bc-2*^{2} (IVT 7233 cultivar group). Since 'mosaic-inducing' strains do not induce symptoms on cultivars with the '*I*' gene, cultivars of the IVT 7233 group are effectively resistant to all known strains of BCMV. However, the gene-for-gene model described by Drijfhout (1978) does not fully explain the existence of isolates expressing the novel pathogenicity that is now known, which Spence and Walkey (1995) attribute to the existence of genes controlling the temperature sensitivity of necrosis in combination with the '*I*' gene.

The regional distribution of BCMV strains clearly has important implications for the deployment of these resistance genes, especially for international bean breeding programmes. In the New World and Europe, where mosaic-inducing strains of BCMV and cultivars with resistance to them predominate, the '*I*' gene provided effective protection against the virus for more than 50 years (Zaumeyer and Meiners, 1975). Since the gene also prevents seed-transmission, it has provided valuable means of eliminating quarantine risk, and has been exploited by organizations engaged in moving seeds within and among continents. In areas where 'necrotic' strains of BCMV and cultivars lacking the '*I*' gene predominate and provide sources of inoculum, notably parts of eastern and southern Africa but, increasingly, also in other regions of the world, cultivars possessing the '*I*' gene are proving exceedingly vulnerable to 'black root'. For such situations, genotypes with *bc-3* alone or with the '*I*' gene in combination with the recessive *bc-2*^{2} or *bc-3* gene have been developed (Kornegay, 1991: Mukoko *et al.*, 1994). Use of *bc-3* alone avoids the problem of the unfavourable linkages that occur between the '*I*' gene and seed coat colour genes.

Recent evidence that the '*I*' gene confers multiple resistance not only to four other potyviruses (Kyle *et al.*, 1988) but also to race 3 of the halo blight pathogen, which predominates in the same areas of Africa as the 'necrotic' strains of BCMV (Teverson, 1991; Taylor *et al.*, 1995), supports use of the latter strategy for eastern and southern Africa. It also offers an explanation for the frequent occurrence of the '*I*' gene in unimproved germplasm and landraces (Kelly,

1988), its extensive use in breeding and the success, despite their vulnerability to 'necrotic' strains of BCMV, of improved cultivars with the gene.

BEAN GOLDEN MOSAIC

Aetiology

Bean golden mosaic is caused by bean golden mosaic virus (BGMV) of the geminivirus group (Goodman and Bird, 1978). BGMV is an ssDNA-containing virus with twin isometric particles each about 19 nm in diameter. The virus was first purified by Galvez and Castano (1976). The disease was first reported from southern Brazil in 1961, since when it has been recorded in the major areas of bean production of that country. Subsequently, BGMV has been recorded widely, from Mexico, Central America, the Caribbean, Venezuela, Colombia and Argentina (Costa, 1965; Galvez and Morales, 1989b; CIAT, 1990). An apparently similar disease affects *Phaseolus lunatus* in Nigeria (Williams, 1976) but further comparative studies are required to determine whether Nigerian lima bean golden mosaic is indeed caused by BGMV (Galvez *et al.*, 1977; Vetten and Allen, 1983). Bean golden mosaic is known variously as bean yellow mottle, bean golden-yellow mosaic, bean double-yellow mosaic and 'mosaico dorado' (Galvez and Morales, 1989b).

Biology

The host range of BGMV is narrow, being confined to legumes. Hosts include the four cultivated species of *Phaseolus*: *P. vulgaris*, *P. acutifolius*, *P. lunatus* and *P. coccineus*, as well as *P. polystachyus*; other hosts include species of *Macroptilium*, *Vigna*, *Teramnus* and *Calopogonium* (Galvez and Morales, 1989b) but it is not certain that all are natural hosts. Conventionally, *Macroptilium* spp. have been considered a major reservoir of BGMV in Latin America. DNA probes developed at the University of Wisconsin have been used to detect virus in wild hosts, and preliminary findings call to question their importance as reservoirs of BGMV (CIAT, 1989). Work in the Dominican Republic concluded that *M. lathyroides* was infected by a geminivirus distinct from BGMV, suggesting that this plant species is not the source of inoculum of BGMV (Martinez *et al.*, 1992).

Recent comparative studies of BGMV isolates obtained from Puerto Rico, Guatemala, the Dominican Republic and Brazil indicate that considerable differences exist among those isolates tested. Genomic differences between a Brazilian isolate that is not mechanically transmissible and mechanically transmissible isolates from Guatemala, the Dominican Republic and Puerto Rico show that the former (BGMV-BZ) is sufficiently distinct from the latter group (BGMV-GA, BGMV-DR and BGMV-PR) that they can be considered as distinct strains (designated types I and II) of BGMV (CIAT, 1988; Gilbertson *et al.*, 1993). More work is warranted to unravel further the relationships between isolates of BGMV from other geographical origins as well as with other geminiviruses that naturally

infect *Phaseolus*, including bean dwarf mosaic (Hidayat *et al.*, 1993) and bean calico mosaic geminiviruses (Brown *et al.*, 1990).

Symptoms

Susceptible genotypes of *P. vulgaris* develop a brilliant golden yellow coloration, starting in the veins of the first trifoliolate leaves within 2 weeks of sowing if vector populations are dense. Following exposure to viruliferous whiteflies (*Bemisia tabaci* Genn.), small yellow dots may appear near leaf veins about 4 days later. Young leaves of diseased plants usually become rolled and cupped. Severely affected plants may be stunted and leaves become almost bleached. Pods often exhibit blotching and seed may be discoloured, as well as reduced in size and number. Less susceptible cultivars develop less intense symptoms, with a tendency toward remission (Galvez and Morales, 1989b).

The concentration of virus increases during symptom development and reaches a peak 1–2 weeks after infection. Thereafter, virus titre decreases rapidly and is very low in plants with well-developed symptoms (Shock and Goodman, 1981). Electron microscopy reveals that the principal cellular symptom is a change in chloroplast morphology (Kitajima and Costa, 1974). Symptoms are restricted to the phloem and cells adjacent to the parenchyma tissue (Kim *et al.*, 1978). Viroplasms appear as packed hexagonal crystal arrangements or as loose aggregates in the nuclei of infected cells, and distinct changes occur also in the nucleoli (Goodman and Bird, 1978).

Epidemiology

BGMV is not seedborne (Costa, 1965; Pierre, 1975) so that wild or weedy relatives of *P. vulgaris*, and volunteers or groundkeepers of bean crops nearby, are presumed to act as reservoirs of infection by the virus (Gamez, 1971; Pierre, 1975). Field spread occurs efficiently through the whitefly vector, *B. tabaci*, in which BGMV is transmitted in a persistent or semi-persistent manner. Acquisition and inoculation by adults can occur in a total time of less than 6 min but efficient transmission requires a longer feeding period. Individual adult whiteflies transmit intermittently for up to 16 days after acquisition (Gamez, 1971; Goodman and Bird, 1978).

Studies of the relationship between *B. tabaci* and putative geminiviruses in other tropical food legumes (Nene, 1972; Anno-Nyako *et al.*, 1983) tend to confirm the above findings. In no case is there evidence of transovarial transmission; nymphs may acquire virus and retain it through the pupal stage.

Female whiteflies are often found more efficient vectors than males and it has been suggested by Bird and Maramorosch (1978) that this may relate to differences in feeding behaviour. With most whitefly-transmitted geminiviruses investigated, the minimum time required for inoculation is less than that for acquisition and often less than that needed for a whitefly's stylet to reach the phloem (Pollard, 1955), suggesting that virus reaches susceptible tissue before

reaching the phloem. In some studies (e.g. Anno-Nyako *et al.*, 1983), a latent period has been shown to exist.

In Latin America, BGMV is prevalent at elevations below 1500 m, where temperatures are higher. Under these conditions, virus reservoirs are more plentiful and whitefly populations denser (Galvez and Morales, 1989b). Recent increases in the range and severity of BGMV have been attributed to the expansion of soyabean production in Brazil (Costa, 1975) and to increased production of tobacco, tomatoes and cotton in Central America (Galvez and Morales, 1989b), because these crops are preferred hosts of the plurivorous *B. tabaci* whose populations have increased concomitantly.

Although whitefly populations and the incidence of virus diseases they spread are each strongly influenced by environment (Vetten and Allen, 1983), it is clear that *B. tabaci* has the capacity to act as a highly efficient vector, based on the following factors: dense populations of adults (50–100 adults) can build up within 2 days of seedling emergence; the adults' flight activity; their ability to acquire and transmit virus from plants infected only 60 hours earlier, before symptom development; and their efficient and persistent transmission. These factors underlie the often high rate of spread of BGMV and its relatives under field conditions (Anno-Nyako *et al.*, 1983; Vetten and Allen, 1983).

Crop Loss

BGMV infection can decrease pod number, the number of seeds per pod and seed weight. Crop loss depends on the time of infection, bean genotype and, possibly also, on virus strain. Estimates range from about 40 to 100% loss of seed yield (Galvez and Morales, 1989b).

Control

The incidence of BGMV decreases with increasing distance from preferred hosts of the vector. There are also opportunities for reducing disease severity by manipulating sowing time so as to escape periods of peak population density of *B. tabaci* (Galvez and Morales, 1989b). The spread of golden mosaic can also be decreased by controlling the vector by chemical or, perhaps, by biological means. Various insecticides are effective against whiteflies, and the combination of a systemic insecticide like carbofuran or aldicarb with mineral oil, which can immobilize *B. tabaci* within 3 min of its alighting on an oil-sprayed leaf, has been advocated (Nene, 1973; Galvez and Morales, 1989b). Substantial yield increases were obtained in the Dominican Republic by applying carbofuran at sowing, followed by monocrotophos after emergence (Abreu-Ramirez and Galvez, 1979). Recently, seed treatment with carbosulfan has shown promise in Guatemala (CIAT, 1989). Certain parasitic fungi including *Paecilomyces farinosus* (Dick. ex Fr.) Brown & Smith may possibly play some role in the natural regulation of whitefly populations (Nene, 1972). Differences in vector preference between bean cultivars do exist and

relate to leaf pubescence and to common bean gene pools, but such resistance has not been shown to confer resistance to BGMV (Blair and Beaver, 1993a).

The search for host plant resistance amongst bean germplasm has led to the identification of a few promising accessions. However, among about 10,000 *P. vulgaris* accessions, not one genotype was found to be immune to BGMV. Those possessing partial resistance or tolerance include Porrillo Sintetico, Porrillo 70, Turrialba 1 and ICA-Pijao, and the tolerance of several of these cultivars has been confirmed in disease nurseries run in Guatemala, El Salvador and the Dominican Republic. They have been used successfully in breeding black-seeded cultivars such as ICTA Quetzal and Negro Huasteco in Guatemala and Mexico, respectively (Galvez and Morales, 1989b).

Work in Guatemala has also led to the identification of BGMV-tolerant accessions of *P. coccineus* and work in Nigeria with lima bean golden mosaic led to the identification of partial resistance in *P. lunatus*, as well as to a high level resistance in the ecotype of the wild species, *P. ritensis*, with which *P. lunatus* is interfertile (Baudoin and Allen, 1979; Galvez and Morales, 1989b). Attempts have also been made to produce BGMV-resistant material by irradiation and chemical mutagenesis but selections from the mutant progenies did not possess resistance superior to materials selected conventionally (Tulmann-Neto *et al.*, 1977).

Recent collaborative work in Latin America has led to the identification of new, superior sources of BGMV resistance in 188 accessions selected from a set of 1660 accessions of bean germplasm. As a result, resistance is now available in seed types other than black-seeded materials, often at higher levels of partial resistance than were available previously, in some cases indicative of transgressive segregation. Three distinct resistance mechanisms have been recognized; these are: disease escape, through earliness or superior plant vigour; tolerance, *sensu* the ability to withstand yield depression despite systemic infection; and partial resistance, expressed as development of mild symptoms (CIAT, 1986; 1987a, b; 1988, 1990; Morales and Niessen, 1988). Although BGMV resistance in the line A 429 is governed by a single recessive gene (Blair and Beaver, 1993b), it seems that in other genotypes of *P. vulgaris*, resistance to BGMV is controlled largely by additive genes, so further progress may be expected from selection within and among hybrid populations derived from BGMV-resistant parents of diverse origin (Morales and Singh, 1991). Exciting advances are also being made in the development of transgenic plants, using electrical discharge particle acceleration methods, so opening a way toward the creation of transgenic plants with BGMV resistance (CIAT, 1991). Transgenic plants have so far proved to be susceptible, however (Azzam *et al.*, 1996).

CONCLUSIONS

In this chapter we have reviewed existing knowledge of the major diseases of common bean. For both small- and large-scale farmers, in temperate or tropical regions, host plant resistance remains our most important means of disease control. This is reflected in the concentration of recent research on the understanding of the nature and genetics of pathogenicity in parasites, of resistance in hosts and of their interactions with environment.

Most important have been the advances in our knowledge of halo blight and BCMV. The host ranges of the pathogens causing both diseases are now known to be rather wider than hitherto suspected. Alternative hosts provide not only sources of inoculum but also opportunities for increased variability in the pathogenicity of the parasite. For example, Spence and Walkey (1991a, 1995) suggest that strains of the 'A' serotype of BCMV may have originated in wild legume species in central Africa. Other pathogens outside the centre of origin of common bean, like that causing scab (*Elsinoe phaseoli*), may have similar origins, though this remains to be investigated.

Five matching gene pairs account for the interactions observed between the common bean and halo blight pathogen genotype combinations so far tested. Four of the resistance genes segregate normally as either dominant or recessive. There is evidence that the other resistance gene (R3) also confers resistance to the NL3 strain of BCMV and other potyviruses, an instance of true multiple disease resistance. Quantitative resistance has also been identified. Mapping of the distributions of strains of the halo blight and BCMV pathogens has helped to define more effective strategies for breeding for resistance to these pathogens. No doubt continued collection will reveal further variation in both parasite and host.

Nevertheless, there remain large gaps in our knowledge of the common bean disease system. For rust and anthracnose, parasite–host interactions have long been known to be highly specialized but, because of the wide pathogenic variation of the parasites and earlier lack of standardization of methodology, the distribution of physiological races and inheritance of resistance remain little understood. For the other major pathogens, situations are less clear but quantitative variation in pathogenicity and resistance appear to predominate, offering greater opportunities to develop durable protection against plant disease.

There also remains a need to better quantify crop loss caused by disease, among the other agronomic constraints on common bean productivity. Some useful progress has been made with common bacterial blight for which a crop loss model is now available, but such studies need to be extended to other diseases if clear priorities among constraints are to be set. That said, it is evident too that priorities among pathogens are liable to change, not only as progress is made in their management but also as common bean production moves into more marginal areas, perhaps particularly with regard to soilborne pathogens.

Better information on pathogen variability and its distribution and the genetics of host–pathogen interactions is vital for the identification and effective deployment of appropriate resistance genes. Some pathogens have evidently coevolved with their host so that diversity may be found in the *P. vulgaris* gene pool, others (BCMNV and some races of halo blight) have evolved outside the centres of origin of common bean. Improved biochemical and molecular techniques have recently provided valuable insight into some of these aspects in the cases of the angular leaf spot and common bacterial blight pathogens and will obviously contribute to further studies of these and other pathogens.

Finally, our ultimate aim must be the development of safe, economic and durable disease control strategies for all farm situations. This will probably be achievable only through a combination of measures in an integrated control system including cultural practices, crop and varietal mixtures and chemicals (at

least for large-scale situations) as well as host plant resistance. Yet studies of the biology and epidemiology of the pathogens of common bean have become unfashionable of late and are urgently needed to supplement existing knowledge to provide a sound basis for the development of effective integrated control practices.

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DISEASES OF COWPEA

5

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INTRODUCTION

The cowpea belongs to the pantropical genus *Vigna* of the tribe *Phaseoleae* in which the genus is closely allied to *Phaseolus* (Verdcourt, 1970; Baudoin and Marechal, 1985). Six species are cultivated in Asia: *V. aconitifolia*, the moth bean; *V. angularis*, the adzuki bean; *V. mungo*, black gram or urd bean; *V. radiata*, green gram or mung bean; *V. trilobata* and *V. umbellata*, the rice bean (Jain and Mehra, 1980). The geocarpic species *V. subterranea*, the bambarra groundnut, is cultivated in Africa and various other species of *Vigna* are tropical pasture species (Lenné, 1990). The cowpea, which is much the most widespread and important, belongs to the species *V. unguiculata* within which five subspecies have been recognized (Verdcourt, 1970). An alternative scheme (Marechal *et al.*, 1978) reduces the three cultivated subspecies to 'cultigroups' within *V. unguiculata* ssp. *unguiculata*, with the erection of three wild subspecies, as discussed by Ng and Marechal (1985) and Steele *et al.* (1985).

The cowpea was domesticated in Africa, where most of the 160 species of *Vigna* are native and 66 species are endemic, within the ancient sorghum and pearl millet farming systems of the savannah zone probably around the third millennium BC. West Africa is the centre of genetic diversity of *V. unguiculata* ssp. *unguiculata* which first reached India some time after 1500 BC, and Verdcourt's two other cultivated subspecies, *cylindrica* (the catjang) and *sesquipedalis* (the yard-long bean), were selected out of ssp. *unguiculata* after it reached India (Steele *et al.*, 1985). Cowpeas reached Europe before 300 BC, and Spaniards took the crop to the West Indies in the 17th century AD. More cultivars reached the New World from West Africa with the slave trade, reaching the southern USA early in the 18th century (Steele and Mehra, 1980). Now, the cowpea is grown throughout the tropics and subtropics, principally for its dry seed but also as a vegetable, for fodder and as a cover crop. In Africa, the young leaves are eaten as a spinach or dried for use in soups, the haulms are fed to livestock and some cultivars

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provide a fibre. West Africa, Brazil and India are the main centres of production of dry seed. There are probably no accurate estimates of world production; unofficial statistics suggest the world area of cowpeas harvested is about 6 million hectares, with an average seed yield of 240 kg ha^{-1} and total annual production of 1.4 million tonnes. This is almost certainly an underestimation, in part because of the omission from official statistics of quantities consumed in subsistence agriculture, and it seems probable that world seed production is significantly greater than 2 million tonnes annually (Allen, 1983; Steele *et al.*, 1985). Nigeria remains the world's largest producer of cowpeas. In the early 1970s, some 80% of the Nigerian crop was grown in the ancient cereal farming system of the savannahs where unimproved, spreading, photosensitive and locally adapted landraces are intercropped (Steele and Mehra, 1980). With the development and release of early maturing, erect cultivars that are substantially heavier cropping than traditional landraces (Smithson *et al.*, 1980; Ntare, 1989; Naik *et al.*, 1990; Singh, 1994), there has been a shift toward more intensive forms of production in some areas.

In the savannahs of Africa, cowpeas are most commonly found as a subordinate companion crop in complex crop mixtures to which few inputs are added. Whereas such traditional systems tend to be relatively stable, they are also poor yielding. Although the traditional practice of late sowing forfeits some grain yield, it is evident that in Africa insect pests and diseases are the principal constraints on seed yield of cowpea. There is evidence that the damage caused by insect pests (Perrin, 1977; Matteson, 1982) and pathogens (Moreno, 1975; Ouko and Buruchara, 1989; Allen, 1990) is often, but not invariably, less in complex crop mixtures than it is in monoculture of cowpeas. Cowpeas are susceptible to attack from a very wide range of pests and diseases which attack the crop at all stages of growth. Reviews of the literature on cowpea diseases include those of Williams (1975b), Singh and Allen (1980), Allen (1983) and Emechebe and Shoyinka (1985); reviews of cowpea virus diseases also include Thottappilly and Rossel (1985, 1992) and Mali and Thottappilly (1986). A colour illustrated field manual is also available (Singh and Allen, 1979). The economic importance of diseases, relative not only to one another but also among other biotic and abiotic constraints, has tended to be inadequately quantified; the most notable exceptions include the studies of cercospora leaf spot (Schneider *et al.*, 1976), scab (Mungo *et al.*, 1995) and bacterial blight (Kishun, 1989). It is clear that the importance of cowpea pathogens varies considerably both across regions of production and across ecological zones. Thus, in the USA the principal diseases are still considered to be fusarium wilt and root-knot (Mackie, 1934; Patel, 1985) whereas in Brazil priority diseases include scab, leaf smut, cercospora leaf spot, powdery mildew and fusarium wilt, cowpea severe mosaic and blackeye cowpea mosaic (Lin and Rios, 1985). In Africa, web blight, cercospora leaf spot, anthracnose, rust, bacterial pustule and cowpea mosaic are the major diseases of the forest belt (Williams, 1975b; Oyekan, 1979); and scab, brown blotch, septoria leaf spot, bacterial blight, blackeye cowpea mosaic and witchweed are the principal problems in the savannahs (Allen *et al.*, 1981b; Emechebe and Shoyinka, 1985; Aggarwal and Ouedraogo, 1989). There is, of course, much overlap, local variation and seasonal change. For instance, ascochyta blight is a devastating disease

under humid conditions at altitudes above about 1000 m (Allen, 1983; Price and Cishahayo, 1986) and cowpea golden mosaic is severe under conditions of high humidity and high temperature (Vetten and Allen, 1983). Intensification of cowpea production in some areas of the West African savannahs appears to have exacerbated the witchweed problem (Berner *et al.*, 1994), and irrigation schemes have sometimes encouraged the massive build-up of aphidborne virus diseases (Raheja and Leleji, 1974). Charcoal rot, which is exacerbated by drought stress, is sometimes found the most destructive disease under semi-arid to arid conditions in the Sahelian Zone (IITA, 1984; Burke *et al.*, 1986a). This chapter focuses on those diseases we consider of greatest economic importance. These are: anthracnose and brown blotch, each caused by a *Colletotrichum* species; scab, caused by a *Sphaceloma*; the cercospora leaf spots; web blight, caused by *Rhizoctonia solani*; aschochyta blight; bacterial blight and bacterial pustule, caused by distinct pathovars of *Xanthomonas campestris*, blackeye cowpea mosaic and cowpea aphidborne mosaic, caused by distinct potyviruses; and the parasitic angiosperm witchweed, *Striga gesnerioides*. Fungal diseases of local or minor importance are summarized in Table 5.1 and the viruses naturally infecting cowpea are reviewed in Table 5.2. The reader is referred to the review of Allen (1983) for information on the minor bacterial diseases and parasitic nematodes afflicting the cowpea crop. Allen and Lenné (Chapter 1, this volume) review the diseases of the Asian *Vigna* species.

ANTHRACNOSE AND BROWN BLOTCH

Aetiology

Anthracnose has been considered to be caused by a form of *Colletotrichum lindemuthianum* (Sacc. & Magn.) Br. & Cav. (Onesirosan and Barker, 1971; Allen, 1983), but recent studies suggest this is incorrect (Sherriff *et al.*, 1994). Investigations of the infection process and host specificity of an isolate (I 57) from Nigeria have revealed novel features (Bailey *et al.*, 1990; Pain *et al.*, 1992) and it now appears that the cowpea anthracnose pathogen is best referred to *C. destructivum* O'Gara (Latunde-Dada *et al.*, 1996). It remains to be shown whether or not the causal agent of the disease outside Nigeria is also *C. destructivum*. Brown blotch is caused by *C. capsici* (Syd.) Butler & Bisby and apparently also by *C. truncatum* (Schw.) Andrus & Moore (Singh and Allen, 1980; Emechebe, 1981) but it is probable that these two names are used for the same pathogen (J.A. Bailey, Bristol, 1996, personal communication). Diseases caused by other *Colletotrichum* spp. are reviewed in Allen *et al.* (Chapter 4, this volume), Hill (Chapter 11, this volume) and Lenné (Chapter 13, this volume).

Biology

Cowpea anthracnose is reported from Brazil (Lin and Rios, 1985), the USA (Onesirosan and Barker, 1971), Uganda (Hansford, 1937), Zambia (Angus,

Table 5.1. Local or minor fungal diseases of cowpea.

Disease	Causal fungi	Distribution (references)
Seed and seedling diseases		
Seed decay and seedling mortality	*Pythium aphanidermatum* (Edson) Fitz. and *Rhizoctonia solani* Kühn; *Macrophomina phaseolina* (Tassi) Goid. and *Phytophthora* spp. cause seedling mortality in some areas	Widespread (1)
Stem, collar and root rots, and wilts		
Pythium stem rot	*Pythium aphanidermatum* (Edson) Fitz.	The pathogen occurs worldwide but the disease is reported only from Nigeria, Tanzania and Brazil (1)
Phytophthora stem rot, red stem canker	*Phytophthora vignae* Purss; *P. cactorum* (Leb. & Cohn) Schroet.	Widespread but local. Reported from USA (*P. cactorum*), Australia, India and Taiwan (*P. vignae*). *P. vignae* is known from adzuki bean (*Vigna angularis*) in Japan (1,12)
Stem rot, stem canker	*Diaporthe phaseolorum* (Cooke & Ellis) Sacc.	USA (6); and in India on *Vigna caracalla* (7)
Sclerotium stem rot	*Sclerotium rolfsii* Sacc. (teleomorph = *Corticium rolfsii* Curzi)	Widespread (1)
Ashy stem blight, charcoal rot	*Macrophomina phaseolina* (Tassi) Goid.	Widespread (1,11)
Fusarium collar and dry root rot	*Fusarium solani* (Mart.) Sacc. (teleomorph = *Nectria haematococca* Berk. & Br.)	Widespread but local. Recorded from Brazil, Puerto Rico, Nigeria, Uganda, Malaysia and the Philippines (1)
Fusarium wilt	*Fusarium oxysporum* f. sp. *tracheiphilim* (E.F. Smith) Snyd. & Hans.	Widespread. Reported from the USA, Brazil, Nigeria, Uganda, India, Malaysia and Australia (1)
Verticillium wilt	*Verticillium* sp.	USA and Australia (1)
Foliar and pod diseases		
Target spot	*Corynespora cassiicola* (Berk. & Curt.) Wei	Worldwide (1)
Septoria leaf spot	*Septoria vignae* P. Henn., *S. vignicola* Vasant Rao, *S. kozopolzanskii* Nikolajeva and *S. vignae-sinensis* Sawada	Widespread. *S. vignae* reported from East and West Africa and Brazil; *S. vignicola* occurs in eastern Africa and India. *S. kozopolzanskii* is known only from the former USSR and Zambia, and *S. vignae-sinensis* is reported from Taiwan (1)

Table 5.1. Continued.

Disease	Causal fungi	Distribution/references
Chaetoseptoria leaf spot	*Chaetoseptoria wellmanii* Stevenson	USA and Central America (1)
Aristastoma leaf spot	*Aristastoma guttulosum* Sutton and *A. oeconomicum* (Ellis & Tracy) Tehon	Nigeria and the USA (1)
Myrothecium leaf spot	*Myrothecium roridum* Tode ex Fries	India (5); in Sierra Leone on *Vigna adenantha* (7)
Alternaria leaf spot	*Alternaria* spp.	India, Brazil (1) and Zambia (3)
Leptosphaerulina leaf spot, pepper spot	*Leptosphaerulina trifolii* (Rost.) Petr. (= *L. vignae* Tehon & Stout)	Widespread. Recorded from the USA, Brazil, Malawi, Ethiopia and India; on *Vigna richardsiae* in Papua New Guinea (1,7)
Phyllosticta leaf spot	*Phyllosticta* spp.	Probably pantropical; recorded from Brazil, Malaysia (1) and southern Africa (4)
Phytophthora blight	*Phytophthora* sp. Sometimes assumed to be *P. vignae* but as yet there is no published evidence (see discussion in Ref. 1)	Tanzania (P.N. Patel and C. Kuwite, Ibadan, 1979, personal communication)
Zonate leaf spot	*Dactuliophora tarrii* Leakey	Widespread in sub-Saharan Africa; also India (1,2)
False rust, yellow blister	*Synchytrium dolichi* (Cooke) Gaum.	Widespread in tropical Africa (1)
Brown rust	*Uromyces appendiculatus* (Pers.) Ung.(= *U. vignae* Barcl. = *Aecidium caulicola* P. Henn.)	Widespread (1)
Pink rust	*Phakopsora pachyrhizi* Syd. (uredial anamorph = *Malupa sojae* (P. Hennings), Ono, Buritica & Hennen) and *P. meibomiae* (Arthur) Arthur (uredial anamorph = *Malupa vignae* (Bresadola) Ono, Buritica & Hennen). (see Ref. 8)	*P. pachyrhizi*, in its newly defined sense (8), reported on cowpea from Nigeria, Sierra Leone, Ghana, Cambodia and China. *P. meibomiae* on cowpea in Brazil (8)
Leaf smut	*Entyloma vignae* Batista and/or *Protomycopsis phaseoli* Ramak. & Subram. between which there is obvious confusion (see Ref. 1 for discussion)	Probably pantropical, on cowpeas and other legumes. Collections from Brazil have been identified as *E. vignae* (9) as well as *P. phaseoli* (IMI 227229). Collections on cowpea from Nigeria (IMI 193853), Togo (IMI 187883) and India all identified as *P. phaseoli*, but there is no doubt they all represent the same taxon. *Entyloma* spp. have been recorded on legumes in Malawi (7)
Powdery mildew	*Erysiphe polygoni* DC. and *Sphaerotheca fuliginea* (Schlecht. ex. Fr.) Poll.	Widespread (1,10)

Continued overleaf

Table 5.1. Continued.

Disease	Causal fungi	Distribution/references
Cladosporium pod spot, scab	*Cladosporium vignae* Garder. Perhaps sometimes confused with scab caused by *Sphaceloma* sp.	Widespread but local. Recorded from the USA, Brazil, Uganda, Zimbabwe and Australia (1)
Choanephora pod rot, lamb's tail pod rot	*Choanephora cucurbitarum* (Berk. & Rav.) Thaxt. and *C. infundibulifera* (Currey) Sacc.	Widespread. Reported from the USA, Brazil, Nigeria and India (1)
Grey mould, pod rot	*Botrytis cinerea* Pers. ex Fr.	Brazil (1)

References: 1. Allen (1983) on which much of the table is based. The reader should refer to this source for primary references. 2. Chandrashekaraiah and Hiremath (1982); 3. Maramba (1983); 4. Mariga *et al.* (1985); 5. Singh and Shukla (1986); 6. Toler *et al.* (1963); 7. Lenné (1990); 8. Ono *et al.* (1992); 9. Prabhu and Albuquerque (1982); 10. Jhooty *et al.* (1985); 11. Burke *et al.* (1986a); 12. Kao and Leu (1982).

1962–1966; Kannaiyan and Haciwa, 1993), Nigeria (Onesirosan and Barker, 1971), India (Prasanna, 1985) and Pakistan (Quereshi *et al.*, 1985). Whereas the natural host range of the cowpea anthracnose pathogen remains unknown, it is clear that the hemibiotrophic cowpea isolate I 57 of *C. destructivum* exhibits considerable specificity: none of six cultivars of common bean was found susceptible and all developed only a superficial flecking following artificial infection. No lesions were produced on excised hypocotyls of lima bean, groundnut, soyabean, pigeonpea, pea, chickpea, lucerne or adzuki bean (*Vigna angularis*) (Bailey *et al.*, 1990). Other strains of *C. destructivum* have host ranges that include soyabean, clover, sweet clover, lucerne, leucaena and phasey bean (*Macroptilium lathyroides*) (Holliday, 1980; Latunde-Dada *et al.*, 1997) apparently as well as non-legumes including tobacco and pyrethrum (*Chrysanthemum cinerariaefolium*) (Rothwell, 1983). At least two physiologic races of the cowpea anthracnose pathogen occur in Nigeria (Skipp, 1975). In a susceptible reaction, race I 57 exhibits an infection process which results in production of water-soaked lesions in all seedling tissues. During the biotrophic phase which lasts for 72 h, the fungus produces unusual, large and multilobed infection vesicles with many septa and elongated neck regions; these remain confined within the epidermal cell first infected (O'Connell *et al.*, 1993). The necrotrophic phase of the host–pathogen interaction is characterized by the rapid development of invasive secondary hyphae which radiate from the vesicles into surrounding tissue in which water-soaked lesions, then acervuli, appear on the surface of infected tissue (Latunde-Dada *et al.*, 1997).

In contrast to the anthracnose fungus which produces straight or ovoid conidia, the brown blotch pathogen *C. capsici*, has large (22–35) curved, or falcate conidia among long setae in acervuli. *C. capsici* is a relatively unspecialized pathogen with a wide host range, including *Capsicum* pepper; among legumes, hosts include species of *Canavalia, Cassia, Clitoria, Crotalaria, Desmodium, Glycine, Indigofera, Lablab, Leucaena, Senna* (Lenné, 1990), *Cyamopsis* and *Sesbania* (Holliday, 1980). *C. truncatum* also has a wide host range which among legumes includes many pasture species (Lenné, 1990; see Lenné, Chapter 13, this volume), as well as

Table 5.2. Viruses other than members of *Potyviridae* in natural infections of cowpea.

Virus (group)	Geographical distribution	Importance/crop loss	Vectors	Seed transmission	Control
Cowpea mosaic comovirus (van Kammen and de Jager, 1978)	Subhumid tropical Africa (Chant, 1959; Bock, 1971; Patel, 1982b) and apparently elsewhere	Locally severe; 60–100% (Chant 1960; Thottappilly and Rossel, 1985)	Beetles and perhaps grasshoppers; claimed thrips transmission refuted (Chant, 1959; Whitney and Gilmer, 1974; Allen and van Damme, 1981)	0–15% (Gilmer *et al.*, 1974; Thottappilly and Rossel, 1992)	Resistant cultivars (Wells and Deba, 1961; Williams, 1977b)
Cowpea severe mosaic comovirus (de Jager, 1979)	Latin America, Caribbean and southern USA. Recent records from Senegal and Pakistan (Bashir and Hampton, 1993; Ndiaye *et al.*, 1993)	Major; up to about 85% (Valverde *et al.*, 1982)	Beetles, especially *Ceratoma* spp. (Smith, 1924; Dale, 1953; Walters and Barnett, 1964)	0–10% (Dale, 1949; Shepherd, 1964)	Resistant cultivars (Fulton and Allen, 1982; Rios and das Neves, 1982)
Cowpea chlorotic mottle bromovirus (Bancroft, 1971)	Warm temperate and tropical America (Kuhn, 1964). Also in Nigeria but on other hosts (Thottappilly *et al.*, 1992)	Negligible (Harrison and Gudauskas, 1968)	Beetle (Hobbs and Fulton, 1979)	0% (Gay, 1969)	Probably not necessary but sources of resistance exist (Sowell *et al.*, 1965)
Cowpea mottle carmovirus (Bozarth and Shoyinka, 1979)	Nigeria (Robertson,1966); Benin (Thottappilly and Rossel, 1988); Ivory Coast (Thouvenel, 1988); Senegal (Ndiaye *et al.*, 1993) and Pakistan (Bashir and Hampton, 1993)	Locally severe; 65–75% (Bozarth and Shoyinka, 1979; Thouvenel, 1988)	Beetle (Allen *et al.*, 1981a)	Apparently 7–10% (Shoyinka *et al.*, 1978), but restricted virus distribution indicates inefficient seed transmission, confirmed by Robertson (1966) and Allen *et al.* (1982) who estimate rates at 0–0.4%	Resistant cultivars (Allen, 1980; Allen *et al.*, 1982)

Continued overleaf

Tobacco streak ilarvirus (Fulton, 1971)	USA (Kaiser *et al.*, 1982)	?	Thrips	?	?
Bean leafroll luteovirus (Ashby, 1984)	Iran (Kaiser, 1972)	?	Aphid	0 (Kaiser, 1972)	?
Broad bean wilt fabavirus (Taylor and Stubbs, 1972)	Europe, China and Japan (Thottappilly and Rossel, 1985)	?	Aphid	?	?
Tobacco ringspot nepovirus (Stace-Smith, 1970)	India (worldwide in many other hosts) (Ganacharya and Mali, 1981)	?	Thrips	0	?
Tomato spotted wilt tospovirus (Ie, 1970)	Worldwide (on many hosts) (Thottappilly and Rossel, 1985)	?	Thrips	?	?

Tobacco streak ilarvirus (Fulton, 1971)	USA (Kaiser *et al.*, 1982)	?	Thrips	?	?
Bean leafroll luteovirus (Ashby, 1984)	Iran (Kaiser, 1972)	?	Aphid	0 (Kaiser, 1972)	?
Broad bean wilt fabavirus (Taylor and Stubbs, 1972)	Europe, China and Japan (Thottappilly and Rossel, 1985)	?	Aphid	?	?
Tobacco ringspot nepovirus (Stace-Smith, 1970)	India (worldwide in many other hosts) (Ganacharya and Mali, 1981)	?	Thrips	0	?
Tomato spotted wilt tospovirus (Ie, 1970)	Worldwide (on many hosts) (Thottappilly and Rossel, 1985)	?	Thrips	?	?

soyabean (see Sinclair, Chapter 3, this volume), common bean and lima bean (*Phaseolus lunatus*) on which symptoms are similar to brown blotch (Andrus and Moore, 1935; Tiffany and Gilman, 1954). Brown blotch of cowpea occurs in Nigeria (Emechebe, 1981), Burkina Faso, Cameroon, Kenya, Japan (Allen, 1983) and Zambia (Allen, 1991). There is evidence that cowpea brown blotch isolates of *C. capsici* vary considerably in their pathogenicity (IITA, 1984). But it is thought that isolates of *C. capsici* from one host will generally infect others.

Symptoms

Although all above-ground parts of the plant can be affected, anthracnose is essentially a stem disease in cowpea. Individual lesions are lenticular to sunken, and tan to brown in colour. Lesion size and distribution depend on cultivar susceptibility. Highly susceptible genotypes develop large spreading lesions which coalesce to girdle stems, branches, peduncles and petioles (Fig. 5.1). Lesions may

Fig. 5.1. Lenticular, sunken lesions of anthracnose on the stem and petioles of a susceptible cowpea in southern Nigeria (Photo: courtesy of D.J. Allen).

also develop on leaves and pods but the symptoms on the stem and branches are more severe (Williams, 1975b; Singh and Allen, 1979).

Brown blotch symptoms are typified by the development of purplish-brown discoloration of petioles, leaf veins, stems, peduncles and, especially, pods (Fig. 5.2). Discoloration, which may be accompanied by cracking of stems, develops as blotches without the formation of discrete lesions. Foliar symptoms are uncommon. Sporulation on dry pods in alternating black and brown bands is diagnostic. Pod infection leads to distortion and maldevelopment of pods which bear black fruiting bodies of the pathogen. Symptoms first appear either at the stem base before flowering or on pedicels following flowering; the latter is especially characteristic (Singh and Allen, 1979). Seedlings that develop from infected seed are liable to damping-off, which is also a destructive phase of brown blotch. On the seed, symptoms arise as tiny purplish-brown spots that develop into round blotches that can cover up to one-half of the seed surface. In severe seed infection, the whole seed may be discoloured and become shrivelled, often with a cracked testa. Occasionally, severely affected seed are greyish-black due to the formation of acervuli (Emechebe, 1981).

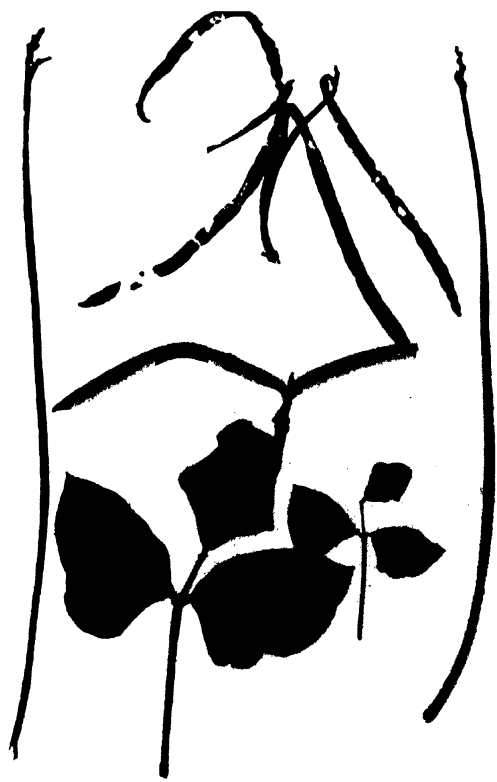

Fig. 5.2. Typical brown blotch symptoms on petioles, peducles and pods; Nigeria (Photo: courtesy of IITA and John Wiley & Sons).

Epidemiology

Both the anthracnose pathogen and the brown blotch fungi are seedborne in cowpea (Emechebe and McDonald, 1979), and both *C. capsici* and the cowpea anthracnose fungus can survive the dry season in Nigeria in infected cowpea debris (Onesirosan and Sagay, 1975; Okpala, 1981). Secondary spread of both diseases is favoured by rain-splash and wind-driven rain. Despite these similarities the prevalence of the two diseases is contrasting, at least in Nigeria. Anthracnose is essentially a disease of the subhumid forest margin belt of the south-west and is seldom encountered further north under semi-arid conditions. Conversely, brown blotch is more widely adapted: although it is sometimes damaging in southern Nigeria, the disease is most destructive under the drier conditions of the Guinea savannah (Allen, 1983).

Crop Loss

Grain yield losses in the range of 35–50% have been recorded in field experiments with anthracnose, using the highly susceptible cowpea TVu 91 in southern Nigeria (Williams, 1975a). Such losses are attributed to the destruction of stem and petiole parenchyma (Skipp, 1975). In an assessment of anthracnose progress, Latunde-Dada (1990) obtained direct correlations between visual assessment scores and the length of necrotic lesions on peduncles of cowpea cultivars of varying susceptibility. Estimates of yield loss incurred from brown blotch in the northern Guinea savannah belt range from 46 to 75%, being more severe in exceptionally wet years (Emechebe, 1981; Alabi, 1994).

Control

Both anthracnose and brown blotch can be fairly effectively controlled by foliar application of fungicides as sprays, with materials including benomyl and mancozeb (Williams, 1975a; Sohi and Rawal, 1984; Emechebe and Shoyinka, 1985; Oladiran, 1990), but whether or not spraying is practicable is likely to depend on the local cropping system. In many instances, the foliar application of fungicides is liable to remain beyond the reach of low-input farmers, but the recent spread of large-scale production of cowpeas, especially around Kano in northern Nigeria, may present opportunities for the wider use of fungicides. Paramount among these perhaps is the adoption of seed treatment that does present a viable option for the subsistence producer of cowpea (Emechebe *et al.*, 1994). However, the use of fungicides has its drawbacks as stressed elsewhere (Allen, 1983), perhaps including the development of fungicide-resistant variants of the anthracnose pathogen (Ramos and Kamidi, 1982; Naik and Anilkumar, 1991). Cultural practices that contribute to the elimination of infected debris, the adjustment of sowing date to avoid periods of heavy rainfall at times of peak susceptibility, and the sowing of clean seed harvested from disease-free production plots, would each have some value in controlling these diseases. The development of anthracnose-

resistant cultivars continues to be the most valuable single control measure, perhaps as a component in an integrated strategy of disease management. Sources of resistance to anthracnose (Williams, 1977a) and brown blotch (Allen *et al.*, 1981b) have been identified and rapid progress has been made in incorporating this resistance into improved cultivars for release to farmers (Smithson *et al.*, 1980; Singh, 1994). Genotypes including TVx 3236 possess combined resistance to both diseases (Adebitan *et al.*, 1992). The mechanisms of resistance to brown blotch are not yet fully understood, but those involved in anthracnose resistance have received substantial investigation, and the reader is referred to the review by Allen (1983) for a fuller account than is given here. There are several distinct resistance mechanisms that operate at different points in the infection process and appear to restrict fungal growth. These mechanisms include a failure to penetrate, penetration that leads to encapsulation of hyphae, a failure of the primary vesicle to develop, and hypersensitivity which is associated with the accumulation of phytoalexins. At least eight anti-fungal compounds are produced in response to infection with the anthracnose pathogen, the most important are vignafuran and methyl phaseollidin isoflavan (Preston, 1975). Hypersensitive resistance in the cowpea cultivar New Era is thought to be governed by a single dominant gene that has proved race-specific. In the wake of the demonstration of the potential transience of such hypersensitive resistance on the appearance of race I 57, interest turned to apparent sources of field resistance in the hope that it would prove durable (Skipp, 1975).

SCAB

Aetiology

Cowpea scab is caused by a *Sphaceloma* species (Emechebe, 1980) which is conventionally considered to be the anamorph of *Elsinoe phaseoli* Jenkins, but the genetic connection between the cowpea scab fungus and its teleomorph has not been demonstrated. It is clear that there is need for a fundamental study of the taxonomy of the cowpea scab pathogen and its close relatives. The establishment of *formae speciales* within *E. phaseoli* has been proposed (Holliday, 1980; Allen, 1983). Scab pathogens of tropical pasture legumes are reviewed in Lenné (Chapter 13, this volume) and the group as a whole is discussed by Allen and Lenné (Chapter 1, this volume).

Biology

The host range of *E. phaseoli*, which was first described on lima bean (*Phaseolus lunatus*) (Jenkins, 1931), includes also common bean (*P. vulgaris*) and mung bean (*Vigna radiata*) in addition to cowpea (Allen, 1983, 1991) but cowpea isolates of the pathogen appear highly specialized and are apparently restricted to *V. unguiculata*. Emechebe (1980) found that *V. radiata*, *P. lunatus* and *P. vulgaris* remained unaffected by artificial inoculation with the cowpea scab fungus, and

only hyacinth bean (*Lablab purpureus*) showed symptoms. Natural infections of hyacinth bean have been attributed to *E. dolichi* Jenk., Bitanc. & Cheo (Cheo and Jenkins, 1945). Whereas the *Elsinoe* state of the cowpea pathogen has not yet been detected either in diseased samples or in culture (Emechebe, 1980), *Elsinoe* is readily obtained in studies of the bean scab pathogen (Phillips, 1994a).

Cowpea scab is widespread, with reports of the disease from Brazil (Lin and Rios, 1985), Surinam (van Hoof, 1963) and Bangalore in India (J.B. Smithson, Hyderabad, 1981, personal communication) as well as throughout sub-Saharan Africa. The distribution of scab disease on cowpea in Africa is known to be the Guinea savannah belt of West Africa including both Burkina Faso and Nigeria; in eastern and southern Africa, the disease is recognized from Ethiopia, Kenya, Uganda, Tanzania, Zambia, Zimbabwe (Allen, 1983) and Rwanda (Price and Cishahayo, 1985). Despite its wide geographical distribution, it appears that the disease is ecologically restricted to semi-arid environments: in Nigeria, scab is seldom encountered outside a narrow latitudinal belt of about 10°30′–12°30′N that corresponds approximately with the extent of the Guinea savannah. Cowpea scab is uncommon or absent in the subhumid forest, the northern Sudan savannah and the Sahel Zones of Nigeria (Allen, 1983).

No work has been done on the pathogenic variability of the cowpea scab fungus. However, the scab resistance of cowpea cultivar TVx 3236 appears to be site-specific, suggesting the existence of pathogenic variation (A.M. Emechebe, unpublished). Studies on the pathogenic variation of isolates of *E. phaseoli* from common bean (Phillips, 1996) confirm that variability exists within, as well as between, host-species-specific populations of these legume scab fungi whose taxonomy appears to warrant revision.

Symptoms

The symptoms of cowpea scab are characterized by the development of silvery grey, circular to oval lesions first on stems, then on leaves, petioles, peduncles and pods (Fig. 5.3). Leaves of diseased plants are often cupped and bear numerous small whitish scab lesions along the veins, and 'shot-hole' may occur. In severe infections, peduncle lesions coalesce to cause distortion and abortion of flower buds. Pods that do develop are usually heavily spotted, curled and mummified, containing very few seeds. Old lesions on stems, peduncles and pods may turn black with the development of chlamydospores (Singh and Allen, 1979; Emechebe, 1980).

Epidemiology

The cowpea scab pathogen has been shown to be both seedborne and seed-transmitted. The pathogen survives the dry season in infected crop debris (Donli, 1983) in which the longevity of its survival may depend on the development of chlamydospores, which have been shown to form on old lesions as well as *in vitro* at temperatures outside the range of 20–30°C. Secondary spread of the pathogen occurs as conidia in water, rain-splash and runoff water; disease severity is

Fig. 5.3. Silvery grey, circular to oval lesions that typify scab; north-east Brazil (Photo: courtesy of D.J. Allen and John Wiley & Sons).

exacerbated by long periods of wet weather (Emechebe, 1980). No measurement of the rate of spread of scab in cowpea has been reported; work in Kenya on bean scab has estimated that scab spreads from an infection focus at rates in the range of 2.0–7.5 m in 6 weeks, varying with location (Mutitu, 1979).

Crop Loss

Scab is now regarded as the most important fungal disease of cowpea, not only in the savannahs of Africa but also in north-east Brazil, regions that account for the vast majority of cowpea production worldwide. Field surveys in Brazil have shown that as many as 16% of cowpea fields had scab (Lin and Rios, 1985). Observations in Nigeria, where heavy scabbing of the flowering axis can completely halt reproductive development, suggest that total crop loss may occur (Emechebe, 1980). Up to 60% loss has been estimated in farmers' fields (Emechebe and Shoyinka, 1985), and under experimental field conditions over

two seasons at Samaru in the northern Guinea savannah of Nigeria, it has been shown that losses in seed yield vary from 9 to 71%, depending on scab severity (Mungo *et al.*, 1995).

Control

Good control of cowpea scab has been achieved through use of foliar fungicides (Price and Cishahayo, 1985; Mungo *et al.*, 1995) and seed dressings (Emechebe *et al.*, 1994). In principle, cultural practices including the production and use of clean seed, sanitation and crop rotation would seem to hold promise (Emechebe, 1980; Holliday, 1980), but experimental work is needed to test their practicability. Sources of scab resistance including VITA 4 have been identified (Allen *et al.*, 1981) and cultivars with scab resistance have been bred by utilizing them (Rios, 1983; Singh, 1994).

CERCOSPORA LEAF SPOT

Aetiology

Two fungi are responsible for cercospora leaf spot: *Cercospora canescens* Ellis & Martin and *Mycosphaerella cruenta* Latham and its anamorph, *Pseudocercospora cruenta* (Sacc.) Deighton which was earlier known as *Cercospora cruenta* Sacc. The morphology of these two pathogens is described by Holliday (1980), and the life history of *M. cruenta* was described by Latham (1934) who first demonstrated the genetic connection between the anamorph and its teleomorph.

Biology

Cercospora leaf spot is widespread in the USA where it was first reported in Mississippi in 1891. There is an early report also from Indonesia (Latham, 1934) and the disease is now thought to be prevalent in tropical Asia. Both species are reported from cowpeas in India (Verma and Patel, 1969; Mew *et al.*, 1985). Cercospora leaf spot of cowpea is also widespread throughout Africa, from Egypt (Fahim *et al.*, 1969) to Nigeria (Williams, 1975b) and Togo (Steiner, 1975), eastwards to Uganda (Hansford, 1937) and south to Mozambique and Zimbabwe (Rothwell, 1983; Plumb-Dhinsa and Mondjane, 1984). In Latin America and the Caribbean, cercospora leaf spot is recorded from Puerto Rico (Vakili, 1977), Costa Rica (Araujo and Moreno, 1980) and Brazil (Lin and Rios, 1985).

C. canescens causes leaf spot of a wide range of legumes including species of *Vigna* and *Phaseolus* as well as *Lablab niger*, groundnut, soyabean (Verma and Patel, 1969; Holliday, 1980; Allen 1983), pigeonpea (see Reddy *et al.*, Chapter 10, this volume), *Calopogonium mucunoides*, *Canavalia ensiformis*, *Centrosema* spp., *Clitoria ternatea*, *Desmodium* spp., *Erythrina* spp., *Flemingia macrophylla*, *Gliricidium sepium*, *Indigofera astragalina*, *Leucaena leucocephala*, *Macroptilium* spp.,

Macrotyloma spp., *Neonotonia wightii*, *Pueraria phaseoloides*, *Rhynchosia* spp., *Stylosanthes* spp. and *Tetamnus* spp. (Lenné, 1990). *M. cruenta* is found on *Calopogonium*, *Lablab purpureus*, *Phaseolus*, *Stizolobium deeringianum* (Holliday, 1980), *Canavalia ensiformis* and *Macroptilium lathyroides* in addition to species of *Vigna* including *V. luteola* and *V. vexillata* (Lenné, 1990). It appears that no studies have been made of pathogenic variation in either fungus, so the extent of host specificity that exists among the cercospora leaf spot pathogens is not known.

Symptoms

C. canescens induces circular to irregular cherry red to reddish-brown lesions on both leaf surfaces (Fig. 5.4), whereas leaf spots caused by *M. cruenta* appear first as a chlorosis on the upper surface of leaves which become dotted with necrotic lesions (Fig. 5.5). These enlarge until the entire affected area becomes necrotic. The lower leaf surfaces infected by *M. cruenta* bear areas of profuse sporulation in which masses of conidiophores appear as downy, greyish-black mats (Williams, 1975b; Singh and Allen, 1979). Spindle-shaped lesions on stems, petioles and peduncles are described by Vakili (1977). Symptoms usually develop relatively late in the season on plants during reproductive growth (Verma and Patel, 1969; Vakili, 1977).

Fig. 5.4. Circular or somewhat irregular, cherry red to reddish-brown lesions induced by *Cercospora canescens*; southern Nigeria (Photo: courtesy of R.J. Williams).

Fig. 5.5. *Pseudocercospora cruenta* induces lesions on the upper leaf surface that are first chlorotic then necrotic. The lower leaf surface supports profuse sporulation in dark grey mats; southern Nigeria (Photo: courtesy of R.J. Williams).

Epidemiology

Both *C. canescens* and *M. cruenta* are seedborne in cowpea (Williams, 1975b), although Emechebe and McDonald (1979) were unable to demonstrate seed-to-plant transmission of *C. canescens*. Infected host debris probably constitutes the most important means of carry-over of the cercospora leaf spot pathogens between seasons. The role of perithecia in survival of *M. cruenta* appears to remain unclear. In view of the wide host range of both these fungi, it seems likely that alternate hosts may play a role in pathogen survival but there is no evidence to support this. Under certain conditions, volunteer seedlings may also provide sources of infection perhaps especially in humid areas, as suggested by parallel studies with related fungi on other legumes (Allen, 1983). The development and release of conidia are favoured by humid weather, and secondary spread by wind and water-splash is more rapid in dense plant populations (Amin *et al.*, 1976) but disease incidence is not affected by cropping system (Araujo and Moreno, 1980).

Crop Loss

Despite the fact that cercospora leaf spot develops late in the season, disease spread is often rapid and premature defoliation can be severe. Verma and Patel (1969) estimated that about 11% of all foliage can be diseased. A survey in Brazil showed that 55–73% of cowpea fields were affected by cercospora leaf spot (Lin

and Rios, 1985). In studies in Ibadan, Nigeria, Schneider *et al.* (1976) showed that yield loss in cowpea was correlated with leaf spot severity, and that areas under the disease progress curves were correlated with yield loss regardless of the shape of the curves. They revealed that defoliation as a result of infection with *C. canescens* and *M. cruenta* can cause crop losses up to 20 and over 40%, respectively. In USA, the crop loss caused by *M. cruenta* has been estimated at 36%, all components of seed yield being adversely affected (Fery *et al.*, 1977).

Control

Cercospora leaf spot can be controlled effectively in cowpeas with foliar fungicides applied post-flower; captafol and benomyl each give good control (Williams, 1975a; Oyekan, 1979). Numerous sources of resistance to *M. cruenta* have been identified (Verma and Patel, 1969; Fery *et al.*, 1976; Vakili, 1977; Williams, 1977a) and some possess resistance against both leaf spot pathogens (Williams, 1977). Little is known of the mechanisms that underlie such resistance, although there is some evidence that pre-formed fungal toxins may contribute to resistance against *C. canescens*. Demonstration of an effect of leaf age on disease development led Schneider and Sinclair (1975) to investigate conidial behaviour. They found that germination and germ tube growth were each inhibited on the surface of young but not old leaves of a susceptible cultivar. Similar inhibition was caused by diffusates from apical but not basal leaves of the susceptible cultivar, and by diffusates from both apical and basal leaves of a resistant cultivar. Somewhat similar results have been obtained from work with *M. cruenta* (Ekpo and Esuruoso, 1977). Resistance to *M. cruenta* is governed by at least two separate genes which are neither allelic nor linked (Fery *et al.*, 1976; Fery and Dukes, 1977).

WEB BLIGHT

Aetiology

Web blight is caused by the ubiquitous soil-inhabiting fungus *Thanatephorus cucumeris* (Frank) Donk, an aggregated species. Its anamorph is *Rhizoctonia solani* Kühn. At least 12 anastomosis groups (AG) are recognized within *T. cucumeris* and there is growing evidence that these groups are discrete evolutionary units that deem taxonomic recognition (Vilgalys, 1988). Web blight is caused by aerial types that most often belong to AG-1, but there are exceptions (Onesirosan, 1977; Bolkan and Ribeiro, 1985; Allen, 1997).

Biology

T. cucumeris has an extremely wide host range and occurs worldwide (Parmeter, 1970; Holliday, 1980). It has a wide host range among tropical pasture legumes

and is a serious disease of some species (see Lenné, Chapter 13, this volume). Web blight of cowpea is a serious disease throughout the humid tropical lowlands. It is among the major diseases in the forest belt of West Africa, though it can be damaging under locally waterlogged conditions also in the drier African savannahs (Oyekan, 1979; Allen 1983). The disease is reported from cowpeas in India (Dwivedi, 1977) as well as in the hot and humid north of Brazil (Lin and Rios, 1985).

T. cucumeris has evolved different parasitic patterns, aerial, soil surface and subterranean, and there is specialization among isolates which are referable essentially to one of three ecotypes, based on their vertical distribution on the host plant (Durbin, 1959). Furthermore, the habitat of an isolate is often associated consistently with a particular mode of host penetration as well as with a particular degree of host specificity (Dodman *et al.*, 1968). Although attempts have been made to define physiologic races (Le Clerg, 1939), the definition of pathotypes within *T. cucumeris* appears less useful than the recognition of ecotypes, which relate to some extent with groupings of hyphal anastomosis. Isolates adapted to aerial habitats are typically fast-growing, carbon dioxide-intolerant types which produce sclerotia readily (Durbin, 1959).

Symptoms

Initial symptoms on the leaves appear as small, circular, reddish-brown spots which enlarge, becoming surrounded by irregular-shaped water-soaked areas. Under humid conditions, the lesions develop rapidly and coalesce, leading to extensive blighting and defoliation (Fig. 5.6). All aerial parts of the plant are affected and become covered superficially with small brown sclerotia and by the light brown hyphae of the causal fungus, which eventually forms a web of mycelium over the plant (Williams, 1975b; Singh and Allen, 1979). Infection by basidiospores produces distinct, small necrotic and circular spots which seldom enlarge. Pods and seed may also bear lesions.

Epidemiology

Carry-over of the web blight pathogen depends on the availability of infected debris, weed hosts and seedborne inoculum (Onesirosan, 1975; Onesirosan and Sagay, 1975). Secondary spread, which is favoured by relatively high temperature and moisture as well as by dense plant populations, relies on the dissemination of sclerotia and direct mycelial growth through plant contact. Basidiospores may also play a role in web blight development (Weber, 1939; Echandi, 1965; Onesirosan, 1975). Aerial isolates of *T. cucumeris* appear not to survive long if infected debris is deeply buried, but sclerotia can remain viable in soil for several years (Weber, 1939; Onesirosan and Sagay, 1975). Soil survival is temperature dependent (Papavizas *et al.*, 1975). The pathogen is seedborne in cowpea (Onesirosan, 1975; Emechebe and McDonald, 1979).

Fig. 5.6. Web blight lesions develop rapidly under humid conditions, coalescing to cause extensive blight and defoliation; eastern Nigeria (Photo: courtesy of W. Horst).

Crop Loss

Oyekan (1979) has estimated that cowpea seed yield may be decreased by 28–40% by web blight but field observations suggest that almost complete crop loss may occur under epidemic conditions that often prevail in the humid forest belt of West Africa (Singh and Allen, 1979).

Control

A wide range of fungicides give good control of web blight when applied as foliar sprays; chemicals shown effective include benomyl, fentin acetate, carbendazim and captafol (Oyekan, 1979; Galvez *et al.*, 1989), but the economics of their use requires local analysis. Measures for the cultural control of web blight include the use of clean seed, field sanitation, the avoidance of dense monocropping during periods of peak rainfall (Onesirosan and Sagay, 1975; Williams, 1975b), improved soil drainage and, perhaps particularly, application of mulch which has proved highly effective in the suppression of web blight in common beans (Galindo *et al.*, 1983). The protective effect of a maize intercrop against web blight development appears to depend on the growth habit of the cowpea cultivar and its spatial arrangement (Allen, 1990). Inoculation of the foliage with the antagonist *Trichoderma koningii* Oudemans can restrict the spread of web blight (Latunde-Dada, 1991) but the biocontrol potential of this practice remains unclear.

There is variation among cowpea genotypes in the degree of susceptibility to web blight, and sources of partial resistance including the cultivar VITA I have been identified (Oyekan *et al.*, 1976; Allen, 1983). This has also been noted for tropical pasture legumes (see Lenné, Chapter 13, this volume). No studies have yet been made on the mechanisms conferring partial resistance to web blight in cowpea. Work on web blight in other legume hosts (Allen, 1983, 1996) suggests that resistance tends to increase with host plant age and to decrease with increasing inoculum concentration. Several distinct resistance mechanisms may operate at different stages in the infection process: a pre-penetration resistance operates in certain cases, while in others a resistance to penetration, or a hypersensitive resistance appear to operate (Flentje *et al.*, 1963). However, partial resistance affords insufficient protection of cowpea under heavy disease pressure, and the integration of several control measures appears warranted, as is the case with web blight of common bean (Galvez *et al.*, 1989).

ASCOCHYTA BLIGHT

Aetiology

Ascochyta blight of cowpea has usually been attributed to *Ascochyta phaseolorum* Sacc. (Holliday, 1980; Singh and Allen, 1980), an imperfect fungus that is a synonym of *Phoma exigua* var. *exigua* Desm. (Boerema, 1972). Whereas *P. exigua* var. *exigua* is known as a weak, wound parasite of a wide range of hosts (Boerema and Howeler, 1967), the ascochyta blight pathogen appears to be somewhat more specialized. Isolates from both Europe and Africa are now known to be distinct in morphology and pathogenicity, and are now treated as a separate taxon, *P. exigua* var. *diversispora* (Bub.) Boerema (Boerema *et al.*, 1981). *P. exigua* var. *diversispora* has so far been positively identified as the cause of ascochyta blight of cowpea only in Kenya, Zambia and Zimbabwe (M. Gerlagh and D.J. Allen, 1986, unpublished results), but it seems probable that it is the principal causal agent of ascochyta blight throughout Africa. Since there are small but consistent differences in isolates from Latin America (Gerlagh, 1987), more comparative studies of the pathogen seem warranted. The same pathogen causes a serious disease of common bean (see Allen *et al.*, Chapter 4, this volume).

Various other closely related fungi also cause a miscellany of leaf and pod spots in cowpea, including *Ascochyta boltshauseri* Sacc., now known as *Stagonosporopsis hortensis* (Sacc. & Malbr.) Petr., which is reported from Ethiopia (Stewart and Dagnatchew Yirgou, 1967), and both *Phoma bakeriana* Sacc. and *P. exigua* var. *exigua* which are quite commonly encountered on cowpea in Africa (Allen, 1983).

Biology

Ascochyta blight of cowpea is of economic importance principally under cool humid conditions at elevations above 1000 m in southern and eastern Africa,

extending westward to the highlands of north-west Cameroon (Allen, 1983). The disease is a major problem in Rwanda and in northern Zambia, in each case in environments where scab (see above) is also prevalent (Price and Cishahayo, 1985, 1986; Kannaiyan and Haciwa, 1993). Elsewhere, the disease is reported from India (Singh *et al.*, 1978), Australia (Alcorn, 1968), Brazil (Lin and Rios, 1985) and Costa Rica (Moreno, 1975). The natural host range of *P. exigua* var. *diversispora* has not been investigated fully; positive identifications have been made of isolates from *Phaseolus vulgaris* (see Allen *et al.*, Chapter 4, this volume), *P. coccineus, P. lunatus, Lablab purpureus, Vigna angularis* and *V. radiata* in addition to *V. unguiculata* which is probably the primary host of the fungus (Gerlagh, 1987; Allen, 1991). *P. exigua* var. *exigua* is recorded from a wide range of legume genera worldwide, including *Cajanus, Cassia, Dolichos, Lablab, Macroptilium* and *Pueraria*. *A. phaseolorum*, its synonym, is reported also from *Canavalia, Desmodium, Glycine, Macrotyloma, Neonotonia, Phaseolus* and *Teramnus* in addition to *Vigna*. Natural hosts within *Vigna* include *V. angularis, V. caracalla, V. coerulea, V. luteola, V. prainiana, V. richardsiae* and *V. vexillata* (Lenné, 1990), and *V. mungo, V. radiata* and *V. unguiculata* (Holliday, 1980). Clearly, more comparative work is warranted to determine the extent to which these legume species are natural hosts of the ascochyta blight pathogen, *P. exigua* var. *diversispora*.

The degree of host specificity seems also unclear (Alcorn, 1968; Holliday, 1980) and little is known about pathogenic variation. Limited information on ascochyta blight of common bean, on which more work has been done (see Allen *et al.*, Chapter 4, this volume), suggests that variation is relatively slight and apparently quantitative in nature.

Symptoms

Young lesions are irregularly circular with grey to brown centres surrounded by a yellow halo. Such lesions, which first appear on foliage, then expand, becoming zonate ringed. Under favourable conditions, such lesions coalesce, spreading rapidly through the canopy to cause extensive blighting of the foliage leading to defoliation. Pycnidia are immersed in infected tissue. Lesions also develop on stems, peduncles and pods. The large, concentrically ringed lesions (Fig. 5.7) are diagnostic (Singh and Allen, 1979) and give the disease its alternative common names of zonate leaf spot or target spot. Both *P. exigua* var. *exigua* and *P. bakeriana* appear not to cause blight; rather each is commonly associated in the field with discrete spotting of leaves and pods, but comparative studies of these taxa, together with *Stagonosporopsis hortensis* (*Ascochyta boltshauseri*), under controlled conditions are required in order to delineate their symptomatology on cowpea.

Epidemiology

The ascochyta blight pathogen is seedborne in cowpea (Noble and Richardson, 1968) and can survive in infected debris and presumably also in alternative perennial hosts. From these sources of primary inoculum, the disease spreads by

Fig. 5.7. Ascochyta blight lesions are concentrically ringed; Zambia (Photo: courtesy of D.J. Allen and John Wiley & Sons).

rain-splash and wind-driven rain that favour the dispersal of spores from pycnidia. Successful infection depends heavily on high relative humidity (Boerema *et al.*, 1981) and probably also cool temperatures. It is sometimes assumed that infection of intact tissue is rare, and disease development depends on predisposing factors including wind damage, by unfavourable growing conditions or by prior infection with another pathogen (Pegg and Alcorn, 1967; Holliday, 1980). Whereas predisposition might be expected to be important in the case of a weak wound pathogen like *Phoma exigua* var. *exigua* (*Ascochyta phaseolorum*), this may not be the case with the more specialized *P. exigua* var. *diversispora* (Boerema *et al.*, 1981). Secondary spread of ascochyta blight is retarded in cowpeas intercropped with maize which acts as a barrier against wind and water-splashed dispersal (Moreno, 1975).

Crop Loss

No estimates of crop loss incurred in cowpea from ascochyta blight have been published though it is evident that, under certain conditions, the disease can be devastating. Early infection can cause complete loss of grain yield (Angus, 1962–1966; Allen, 1983; Price and Cishahayo, 1986).

Control

The use of clean seed, field sanitation, rotation, the isolation from infected reservoirs and the use of *Pennisetum* windbreaks have been proposed as cultural measures against ascochyta blight (Angus, 1962–1966; Pegg and Alcorn, 1967), and Moreno (1975) has demonstrated the efficacy of maize intercropping in protecting cowpea. Various fungicides have been shown to give good control as foliar sprays. In Rwanda, carbendazim (Bavistin) and Brestan have proved effective in decreasing disease severity and increasing cowpea grain yield (Price and Cishahayo, 1985), and various combinations of thiram, benomyl, carbendazim and thiophanate-methyl are effective treatments of seed infected with *P. exigua* var. *diversispora* (Gerlagh, 1987). Small differences exist between cultivars in their susceptibility (Moreno, 1975; Allen, 1983; Kannaiyan and Haciwa, 1993), which appears to increase with plant age (Angus, 1962–1966) and early maturity may enable escape from total destruction. Until a more effective level of partial resistance is available against ascochyta blight, an integration of cultural practices, fungicidal seed dressings and lesser susceptibility would seem the best strategy. The potential value of mulches in controlling ascochyta blight of common beans (see Allen *et al.*, Chapter 4, this volume) suggests there may be an opportunity for cowpeas.

BACTERIAL BLIGHT AND PUSTULE

Aetiology

Bacterial blight is caused by *Xanthomonas campestris* pv. *vignicola* (Burkholder) Dye (Burkholder, 1944) and bacterial pustule is caused by *X. campestris* pv. *vignaeunguiculatae* Patel & Jindal, not a strain of pv. *vignicola* (Allen, 1983; Emechebe and Shoyinka, 1985) from which it is clearly distinct in its pathogenicity (Patel and Jindal, 1982). Common blight caused by the *X. campestris* pv. *phaseoli* is reviewed by Allen *et al.* (Chapter 4, this volume).

Minor and local diseases caused by *Pseudomonas* species are also occasionally reported from cowpea. Brown spot (*Ps. syringae* pv. *syringae* van Hall) is reported from the USA (Gardner and Kendrick, 1923; Patel, 1985), Australia (Wilson, 1936) and Tanzania (Riley, 1960), and wildfire (*Ps. syringae* pv. *tabaci* (Wolf & Foster) Young *et al.*) occurs in the USA (Tisdale, 1924) and Brazil where *Ps. solanacearum* (E.F. Sm) E.F. Sm. (now *Burkholderia solanacearum* (E.F. Sm.) Yabuuchi *et al.*) causes a wilt (da Ponte and Santos, 1973; Lin and Rios, 1985). An unidentified fluorescent group 1b pseudomonad causes a bacterial spot of cowpea in Ethiopia (Allen, 1979).

Biology

Bacterial blight, or canker, was first reported from Oklahoma in the USA in 1931, with subsequent records from other states in the 1940s (Burkholder,

1944; Hoffmaster, 1944). The disease was reported from Tanzania in 1964 (Ebbels and Allen, 1979), then from India (Patel and Jindal, 1970), Nigeria (Williams, 1975b), Puerto Rico (Vakili *et al.*, 1975) and Brazil (Rios *et al.*, 1980). It is now known to be widespread throughout Africa (Kaiser and Ramos, 1979; Allen, 1983; Kannaiyan and Haciwa, 1993) and probably occurs in all major cowpea growing regions of the world.

Bacterial pustule is much more restricted in its distribution. The first collections of the disease were made by D.R.W. Watson at two sites in Tanzania in 1964–1966 (IMI B 2281 and B 2962; Ebbels and Allen, 1979). A bacterial leaf spot, attributed to *X. campestris* pv. *phaseoli*, was recorded at about the same time in Nigeria (Bailey, 1966) where both cultivated and wild cowpeas are affected by bacterial pustule (Williams, 1975b). There is a recent report of the disease from Nepal (Dahal *et al.*, 1992) but bacterial pustule is otherwise unknown outside Africa.

The natural host ranges of these xanthomonads are difficult to determine, in part because the definition of pathovars of phytopathogenic bacteria itself depends largely on host plant identity. There is evidence that *X. campestris* pv. *vignicola* naturally infects *P. vulgaris* (Burkholder, 1944; Vakili *et al.*, 1975), whereas pv. *phaseoli* is reported from various Asiatic *Vigna* species and *Lablab purpureus* but not cowpea (Sabet and Ishag, 1969; Patel and Jindal, 1972), so there is overlapping in host ranges among pathovars and there is almost certainly some confusion in the literature. Recent revisions within *Xanthomonas* have relied upon DNA–DNA hybridization to define homology groups considered genomic species (Vauterin *et al.*, 1995) and this will permit more precise estimation of natural host ranges. Furthermore, taxonomic changes among host species, notably in *Phaseolus* and *Vigna*, seem likely to have compounded the problem. Whereas bacterial leaf spot of mung bean (*Phaseolus aureus* = *Vigna radiata*) was identified as *X. campestris* pv. *phaseoli* in India (Patel and Jindal, 1972), the pathogen of apparently the same disease of mung bean in Ethiopia was identified as *X. campestris* pv. *vignicola* (IMI B 6940; Allen *et al.*, 1976). *Vigna pubigera* (= *V. ambacensis*) is considered a host of *X. campestris* pv. *vignicola* (Sabet *et al.*, 1969) but it remains unclear whether some of the *Xanthomonas* blights of Asian *Vigna* species are correctly referred to pv. *vignicola*, despite the extensive cross-inoculation studies reported by Jindal and Patel (1980) who showed there was considerable host specificity among their isolates. Pathogenic variation has been demonstrated among isolates of the cowpea bacterial blight pathogen in which attempts have been made to define races (Sherwin and Lefebvre, 1951; Jindal *et al.*, 1981), yet race specificity of monogenic resistance has apparently not been clearly demonstrated and some variation in pathogenicity may also be quantitative (Allen, 1983). The bacterial pustule organism, *X. campestris* pv. *vignae-unguiculatae*, is also variable. Three distinct races have been defined on the basis of their reactions to infiltration inoculation of a set of four differential cowpea genotypes: Prima (TVu 76), VITA 3 (TVu 1190), TVu 1630 and TVu 43. Race 1 may be prevalent in West Africa whereas races 2 and 3 occur in East Africa (Patel, 1981).

The histology of infection of susceptible cowpea tissue with *X. campestris* pv. *vignicola* has been investigated by Shekhawat *et al.* (1977) who also have shown

that the bacterium enters intact leaf, stem and pod tissue through stomata. Thereafter, the pathogen multiplies and migrates intercellularly; middle lamellae appear to be dissolved in advance of bacterial spread. Multiplication and migration occurs intracellularly only in xylem vessels. Invasion of leaf veins results in foliar blight.

Symptoms

There are several distinct syndromes of bacterial blight: seedling mortality, stem canker, and foliar blight. Stem canker is the symptom most commonly mentioned in early American literature (Burkholder, 1944; Hoffmaster, 1944) and foliar blight is the most common symptom seen under African field conditions. However, there is evidence that seedborne inoculum of *X. campestris* pv. *vignicola* may lead to seedling mortality and stem canker whereas secondary infection causes foliar blight (Shekhawat and Patel, 1977). The initial symptoms are tiny water-soaked dots on leaves. These dots remain small and the surrounding tissue dies, developing a tan to orange coloration with a yellow halo (Fig. 5.8). On heavily infected leaves the necrosis coalesces so that large areas of leaf are affected and premature defoliation ensues. The pathogen also affects the peduncle and stem causing cracking and local swelling (canker), and water-soaking of pods leads to seed infection (Williams, 1975b; Singh and Allen, 1979).

The symptoms of bacterial pustule first appear on the adaxial surface of leaves as tiny dark, raised and water-soaked lesions which enlarge to about 3 mm in diameter. Dark necrotic spots develop on abaxial surfaces of infected leaves (Fig. 5.9). Pustules in older infections are usually dry and sunken, and heavily infected leaves become chlorotic and fall prematurely (Williams, 1975b; Singh and Allen, 1979).

Epidemiology

Both *X. campestris* pv. *vignicola* and pv. *vignaeunguiculatae* are seedborne in cowpea (Shekhawat and Patel, 1977; Emechebe and McDonald, 1979) and efficient seed transmission must account for the international distribution of the bacterial blight pathogen, although a claim that both pathogens had been introduced from West Africa into East Africa on seed (Kaiser and Ramos, 1979) is invalid, owing to prior records of each from Tanzania (Ebbels and Allen, 1979; Allen, 1981). Seed infection and contamination presumably also play a vital role in pathogen survival between seasons, a subject that appears to warrant more research. The extent to which these two bacterial pathogens survive in infected cowpea debris seems unsure, but work on the closely related pv. *phaseoli* (see Allen *et al.*, Chapter 4, this volume) suggests that infected residues are important in survival in some areas, and survival on weeds and non-host plant species has also been demonstrated. The more restricted distribution of pv. *vignaeunguiculatae* might possibly indicate a poorer survival ability relative to pv. *vignicola*. Under humid lowland tropical conditions, where seasonal changes are minor and

Fig. 5.8. Bacterial blight often appears as tan to orange lesions with yellow haloes; Nigeria (Photo: courtesy of R.J. Williams).

biological activity is continuously high, one might expect a greater attrition of plant pathogenic bacteria in the soil (Buddenhagen, 1965), and competitive saprophytes may play a significant role in xanthomonad survival (Sabet and Ishag, 1969; Allen, 1983). It is tempting to suggest that such factors may influence the greater importance of bacterial blight in the semi-arid savannahs than in the forest belt of West Africa (Emechebe and Shoyinka, 1985).

An initial inoculum load of 1% of infected cowpea seed has been shown to be sufficient to cause an outbreak of bacterial blight at an incidence of 62% (Shekhawat and Patel, 1977). Secondary spread of both diseases is more rapid during heavy rainfall and during overhead irrigation. Insects have also been implicated in the dissemination of bacterial blights. Kaiser and Vakili (1978) noticed that lesions on cowpeas in Puerto Rico were frequently associated with pest damage, and xanthomonads were recovered from washings of five leaf-feeding insects, including three beetles, a leafhopper and a sucking bug. Naturally infested beetles acted as vectors and xanthomonads survived for up to 19 days on

Fig. 5.9. Symptoms of bacterial pustule appear on the lower surface of leaves as tiny, dark raised lesions which seem greasy. Necrotic spots then develop on the upper surface often associated with leaf chlorosis; Nigeria (Photo : courtesy of D.J. Allen).

the bodies of beetles. *Ceratoma ruficornis* (Oliver) was considered the predominant vector. Heavy infestations of the whitefly (*Bemisia tabaci* Genn.) may also lead to transmission of xanthomonads in legumes (Sabet and Ishag, 1969). Secondary spread of bacterial pustule is influenced by the cropping system of which cowpeas are part. Spread within and between plants was shown to be least in relation to sole crop when cowpea was grown in a relay following maize in the long rains, and when grown as an intercrop with maize during the short rains in Kenya (Ouko and Buruchara, 1989).

Crop Loss

Ekpo (1979) has estimated that in the subhumid forest margin of south-western Nigeria, the potential crop loss incurred from bacterial blight may exceed 26% in the moderately susceptible cultivar Ife Brown, and the effect of bacterial pustule on cowpea seed yield was found to be of similar magnitude: the potential loss in seed yield of a resistant (cv. VITA 3) and a susceptible cultivar (cv. Prima) was estimated at 1.8 and 26.6%, respectively (E.J.A. Ekpo, cited in Allen, 1983). However, both these appear to be underestimates. Working in the same environment, Omotunde (1987) recorded losses from bacterial pustule of 2.3 and 76.8% in resistant (TVu 43) and susceptible (TVx 301) cowpea genotypes. In the Sudan savannah belt of West Africa and in India, bacterial blight can cause almost complete crop loss (Emechebe and Shoyinka, 1985; Kishun, 1989).

Control

The use of clean seed is a potentially valuable measure of cultural control for both bacterial blight and pustule, and too few studies appear to have been devoted to this aspect. Since the bulk of the cowpea crop is grown by small-scale farmers in tropical areas of developing countries wherein the practice of saving seed for the next season is the norm, conventional seed certification schemes probably have little applicability. However, in some areas of northern Nigeria, cowpea is being produced by larger-scale farmers who have the capacity to purchase inputs (Emechebe and Shoyinka, 1985), presumably including certified seed. Strict standards in seed production and use of clean seed have been the basis of successful management of *X. campestris* pv. *phaseoli* in common bean in the USA (see Allen *et al.*, Chapter 4, this volume), and Soni and Thind (1991) found that it was relatively easy to produce bacteria-free cowpea seed from symptomless pods in India. Hot water treatment of seed (Boettinger and Bowers, 1975) and perhaps seed dressings (Jindal and Thind, 1990) may have potential in some areas. Manipulation of sowing time, plant population and intercrop pattern (Kishun and Chand, 1989; Ouko and Buruchara, 1989) and field sanitation (Kannaiyan and Haciwa, 1993) might each be expected to have some potential as components of an integrated disease management of these two bacterial diseases. Some chemicals might be expected to give partial control when applied as foliar sprays, judging from experience with xanthomonads on common bean, but their limited effectiveness, high cost, and liability to elicit development of resistant strains probably indicate that chemical control is feasible only under special circumstances (see Allen *et al.*, Chapter 4, this volume).

Much the best strategy for control is the identification, development and use of host plant resistance. Differences in the susceptibility of cowpea cultivars to bacterial blight were first detected by Hoffmaster (1944) who identified cvs. Buff, Iron and Victor as resistant in the USA, where numerous other sources were soon identified (Sherwin and Lefebvre, 1951). Screening of germplasm has led to detection of bacterial blight resistance also in India (Patel and Jindal, 1970; Kishun *et al.*, 1980; Prakash and Shivashanker, 1982) and Africa (Allen *et al.*, 1981b, c; Kannaiyan and Haciwa, 1993). Resistance against bacterial pustule has also been identified (Williams, 1977a; Patel, 1981). Two distinct mechanisms of resistance against bacterial blight are recognized: a hypersensitivity, and a partial resistance expressed as decreased and delayed disease development (Patel and Jindal, 1970; Allen *et al.*, 1981c; Gitaitis, 1983). Hypersensitive resistance appears to be controlled either by a single dominant or a single recessive gene, depending on the cultivar. Modifying factors may also be involved (Lefebvre and Sherwin, 1950; Singh and Patel, 1977). Similarly, both a hypersensitivity and a non-hypersensitive resistance operate in cowpea against bacterial pustule: whereas the former is race-specific, on current evidence the latter is race non-specific. Examples are the reactions of cowpea cvs. VITA 3, TVu 410 and TVu 1630, which possess hypersensitive resistance, and TVu 43 which appears to have race non-specific resistance against bacterial pustule (Patel, 1981). Hypersensitive resistance appears to be governed by two dominant genes, given the symbols *Bp-1* and *Bp-2*. *Bp-1* confers resistance to race 1 alone whereas *Bp-2*

is effective against races 1 and 2. Neither is effective against race 3. Non-hypersensitive resistance is controlled by one, two or three recessive genes, with the symbols *bp-3*, *bp-4* and *bp-5*. On present knowledge, the best combination of genes for incorporation into a cultivar where bacterial pustule is epidemic would be one dominant hypersensitive gene (*Bp-2*) and two recessive genes (*bp-3* and *bp-4*) (Patel, 1982a). Some cowpea genotypes, like TVu 43, TVu 410 and VITA 3, possess some resistance against both bacterial blight and pustule (Kishun *et al.*, 1980; Allen *et al.*, 1981c; Patel, 1982a).

BLACKEYE COWPEA MOSAIC AND COWPEA APHIDBORNE MOSAIC

Aetiology

Blackeye cowpea mosaic virus (BlCMV) and cowpea aphidborne mosaic virus (CAMV) are two potyviruses that are pathogenic to cowpea. BlCMV was first reported in the USA by Anderson (1955) and CAMV was reported a decade later from Europe and Africa (Bock and Conti, 1974). BlCMV was at one time regarded as a strain of bean yellow mosaic virus, and there has been considerable confusion between BlCMV and CAMV (Taiwo *et al.*, 1982; Dijkstra *et al.*, 1987) as well as between BlCMV and bean common mosaic virus (Lana *et al.*, 1988). Other potyviruses including peanut mottle (Demski *et al.*, 1983), as well as cowpea rugose mosaic, cowpea green vein-banding and cowpea severe mottle viruses have also been reported from naturally infected cowpeas (Lin *et al.*, 1979; dos Santos *et al.*, 1980, 1981). Whereas isolates of some of these partially characterized viruses have not yet been compared so as to clarify their relationships within the *Potyviridae*, comparison of nucleotide sequence data of coat protein genes of strains of bean common mosaic virus (BCMV) and BlCMV strongly suggests that BlCMV is best regarded as a strain of BCMV as newly redefined. CAMV is a closely related but distinct virus within the BCMV subgroup of potyviruses (Khan *et al.*, 1993; Mink *et al.*, 1994).

Biology

Owing to confusion between BlCMV and CAMV, and possibly also with certain isolates of other legume-infecting potyviruses, it is not yet possible to define either their natural host ranges or their geographical distribution. Further comparative studies between CAMV and BlCMV have been hampered by the loss of the type culture of CAMV (Lovisolo and Conti, 1966). However, it seems that certain other isolates from the Mediterranean region, including a Moroccan isolate of CAMV (Fischer and Lockhart, 1976a), are distinct from BlCMV which is perhaps the more widely distributed of these two related potyviruses (Taiwo and Gonsalves, 1982; Taiwo *et al.*, 1982). It is perhaps safe to say that both are essentially restricted to legume hosts and that one or other of BlCMV and CAMV occur wherever cowpeas are grown. Together, the viruses are widespread throughout sub-Saharan Africa (Bock, 1973; Ladipo, 1976; Thottappilly and Rossel, 1985;

Burke *et al.*, 1986b), north to the Mediterranean Basin (Lovisolo and Conti, 1966; Fischer and Lockhart, 1976; Taiwo *et al.*, 1981), eastwards to Turkey, Iran and the Indian subcontinent (Kaiser and Mossahebi, 1975; Mali *et al.*, 1981), Indonesia, China and Japan (Iwaki, 1979; Tsuchizaki *et al.*, 1984; Thottappilly and Rossel, 1985), thence to Australia (Behncken and Maleevsky, 1977), Brazil (Lin and Rios, 1985) and the USA (Anderson, 1955; Taiwo *et al.*, 1982). The ecological distribution of these viruses, at least in sub-Saharan Africa, is also wide (Fig. 5.10). Various strains have been recognized. Bock (1973) distinguished the African (neo-type) strain from African mild and vein-banding strains on the basis of the reaction of cowpea cultivar Mak 1, and other variants have been reported elsewhere (Taiwo *et al.*, 1982; Purcifull and Gonsalves, 1985; Bashir and Hampton, 1992). Clearly, the use of standard isolates is vital, and host responses of known cowpea genotypes are likely to remain a valuable guide to the appearance of novel strains.

Symptoms

Natural infection of cowpea with BlCMV or CAMV causes various mosaics, mottling, interveinal chlorosis and green vein-banding (Plate 7), the type of symptom and its severity depending on the susceptibility of the cultivar, the virus strain and the time of infection. Leaf distortion, blistering and plant stunting also occur (Bock and Conti, 1974). A systemic necrosis (Fig. 5.11) sometimes develops in resistant cowpea cultivars in the field (Kannaiyan and Haciwa, 1993) in a manner reminiscent of 'black root' caused by bean common mosaic necrosis virus in *Phaseolus* (see Allen *et al.*, Chapter 4, this volume).

Epidemiology

The seed-transmissibility of BlCMV and CAMV, reflected in their wide geographical distribution, probably also governs virus survival in dry areas, in contrast to the beetleborne cowpea viruses (Allen, 1983; Rossel and Thottappilly, 1990). The extent to which weeds and wild legumes act as reservoirs of infection appears not to have been investigated; the extensive surveys throughout eastern and southern Africa of Spence and Walkey (1994) seem likely to prove relevant to cowpea. There is evidence that the use of irrigation, in addition to perennially damp areas, provide reservoirs of BlCMV in semi-arid savannah of West Africa (Raheja and Leleji, 1974; Rossel, 1977). The seed transmission of both BlCMV and CAMV has been demonstrated with various isolates (Zettler and Evans, 1972; Bock and Conti, 1974; Mali *et al.*, 1983). A Nigerian isolate has been shown located in the cotyledon and embryo of infected cowpea seed but the virus was not recovered from the testa or pod (Ladipo, 1977). Rates of transmission range from 0 to 40% (Kaiser and Mossahebi, 1975), though rates up to about 20% appear more common in the literature (Bock and Conti, 1974; Ladipo, 1977; Aboul Ata *et al.*, 1982; Mali *et al.*, 1983). Seed transmission rates depend in part on the cowpea cultivar, and some cultivars appear to possess a resistance

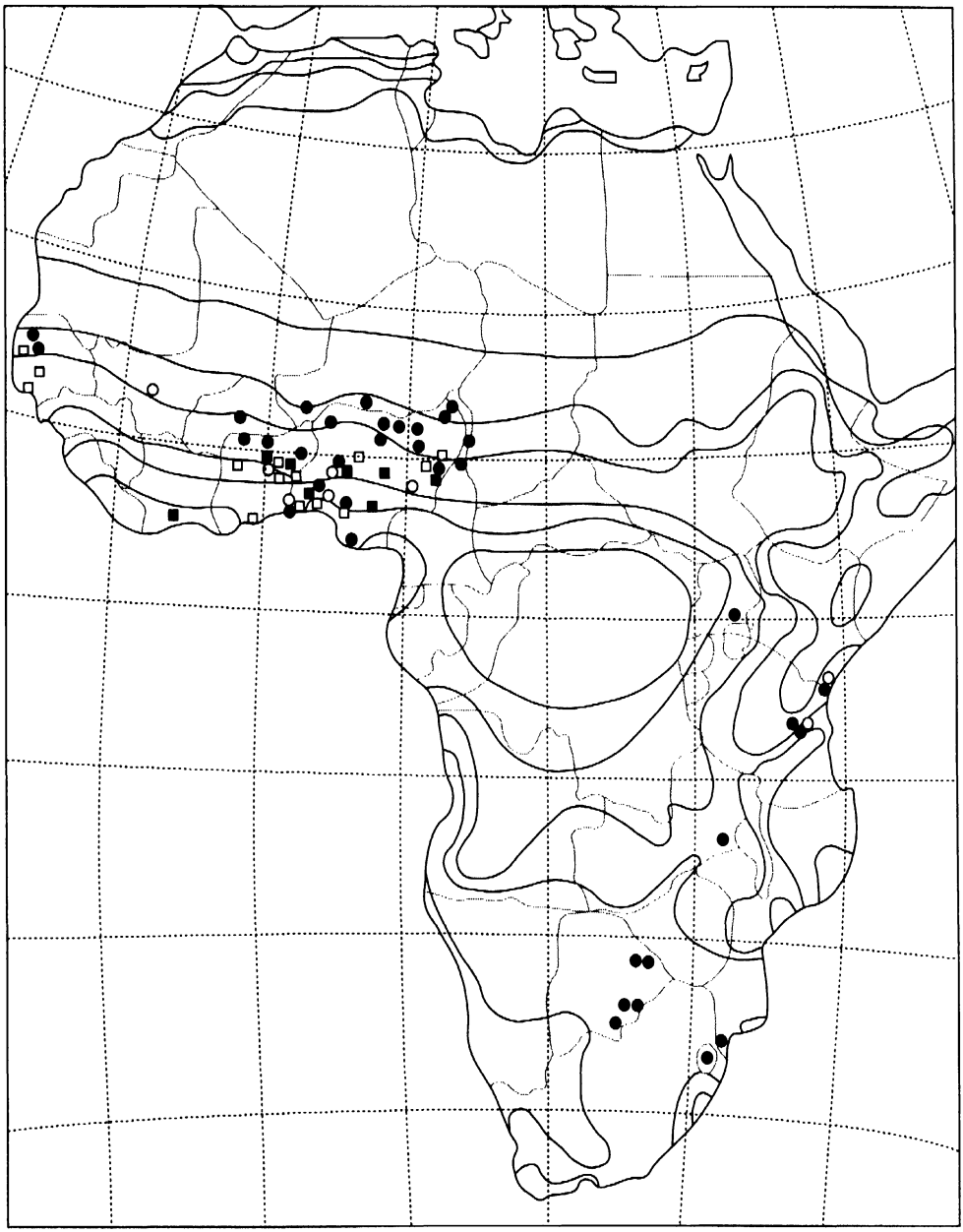

Fig. 5.10. Geographical distribution of viruses in cowpea in Africa: cowpea aphidborne mosaic virus/blackeye cowpea mosaic virus = filled circles; cowpea mosaic virus = open circles; cowpea mottle virus = filled squares; southern bean mosaic virus = open squares. (Courtesy of H.W. Rossel, agroclimatic map after FAO/AGLS, 1997.)

Fig. 5.11. An apical necrosis sometimes develops in resistant cultivars when challenged by potyvirus; Zambia (Photo: courtesy of J. Kannaiyan).

to seed transmission that is independent of resistance to infection (Ladipo, 1977). The virus isolate (Thottappilly and Rossel, 1985) and the severity of symptoms may also influence the rate of seedborne infection, although virus is transmitted at low rates occasionally also in symptomless plants (Aboul Ata *et al.*, 1982).

The secondary spread of BlCMV and CAMV in the field depends on the presence and activity of various species of aphid by which the viruses are transmitted in a styletborne, non-persistent manner. Species reported as vectors include *Aphis citricola* van der Groot, *A. craccivora* Koch, *A. fabae* (Scopoli), *A. gossypii* Glover, *A. medicaginis* Koch, *Cerataphis palmae* (Boisduval), *Rhopalosiphum maidis* (Fitch.), *Myzus persicae* (Sulzer) and *Macrosiphum euphorbiae* (Thomas) (Bock and Conti, 1974; Purcifull and Gonsalves, 1985; Thottappilly and Rossel, 1985; Atiri *et al.*, 1986; Roberts *et al.*, 1993). The extent to which aphid species' prevalence as vectors varies between cowpea-producing regions seems not to have been investigated. Both colonizing and transient species of aphid appear important in the epidemiology of these virus diseases, but colonial species of *Aphis* are principally responsible for secondary spread; other aphids including *R. maidis* may play a role in the development of infection foci (Atiri *et al.*, 1986). In Nigeria, *A. citricola* is sometimes abundant and it has been suggested (Roberts *et al.*, 1993) that, whereas alates of this aphid may be the most important primary vectors, apterous *A. craccivora* is responsible for secondary spread of virus through the crop. Rates of virus spread may be greater in cultivars possessing aphid resistance or to which synthetic pyrethroid insecticides have been applied. Feeding behaviour of *A. craccivora*, which is the most important species on cowpea in Africa, is influenced by cowpea cultivar. Atiri *et al.* (1984) have shown that the abundance and

relative size of aphids is less on aphid-resistant cowpea genotypes than on aphid-tolerant or susceptible ones. Probes are more numerous and of shorter duration on the resistant cultivars from which less phloem sap is ingested (Meslin *et al.*, 1992). Efficiency of BlCMV transmission by *A. craccivora* following probes of less than 1 min can be as much as 50%. *Myzus persicae* has been shown to acquire and transmit BlCMV in mixed infections with cucumber mosaic virus, and their interaction causes a serious, distinct disease known as cowpea stunt (Pio-Ribeiro *et al.*, 1978; Purcifull and Gonsalves, 1985).

Crop Loss

Few attempts have been made to quantify crop loss. Natural field infection has been estimated to have caused an 87% loss in cowpea yield in Iran (Kaiser and Mossahebi, 1975) and from 48 to 60% in Zambia (Kannaiyan and Haciwa, 1993). Complete loss of an irrigated crop has been reported from northern Nigeria (Raheja and Leleji, 1974).

Control

Cultural practices including early sowing and intercropping of cowpeas with cereals may possibly decrease disease incidence (Kannaiyan and Haciwa, 1993), and the use of virus-free seed is potentially important, particularly in preventing spread to new areas (Zettler and Evans, 1972). Field inspection and roguing may help to eliminate seedborne virus but, since there is evidence that BlCMV may occasionally be seed-transmitted in symptomless plants (Aboul Ata *et al.*, 1982), a rapid indexing procedure for the detection of virus in cowpea seed lots would be a valuable supplement. Since some cultivars apparently do not transmit virus through seed (Ladipo, 1977; Mali *et al.*, 1983), selection for 'resistance to seed transmission' could prove a useful strategy.

Certain insecticides may possibly have potential in controlling aphidborne mosaic. Whereas an organophosphate and a carbamate had no effect, Atiri *et al.* (1987) found that the synthetic pyrethroid cypermethrin restricts the acquisition and inoculation of virus, and protects against its transmission. However, subsequent studies with other synthetic pyrethroids have shown that the chemicals do not prevent the initial introduction of virus into the cowpea crop and, when the incidence of incoming alate aphids was high, virus incidence can be higher in sprayed plots relative to unsprayed controls (Roberts *et al.*, 1993).

There is no doubt that the best management strategy against BlCMV and CAMV is to breed for virus disease resistance. Sources of resistance have been identified among cowpea germplasm in the USA (Kuhn *et al.*, 1965; Taiwo *et al.*, 1982), Brazil (Lin *et al.*, 1981); Nigeria (Ladipo and Allen, 1979a), Tanzania (Patel *et al.*, 1982a), Iran (Kaiser and Mossahebi, 1975) and India (Mali *et al.*, 1981). Resistance is often expressed as an immunity (Kuhn *et al.*, 1965; Ladipo and Allen, 1979a; Bashir and Hampton, 1996) that is governed by a single dominant or single recessive gene (Taiwo *et al.*, 1981; Walker and Chambliss,

1981; Quattara and Chambliss, 1991), sometimes in association with modifiers (Patel *et al.*, 1982a). Resistance is also expressed as development of very mild mosaic without adverse effects on plant growth (Patel *et al.*, 1982a) as well as by tolerance, in which systemic infection occurs without the appearance of symptoms (Ladipo and Allen, 1979a). Resistance to the aphid vector is now known not to be a component of virus disease resistance (Atiri *et al.*, 1984). No cowpea genotype has yet been found to possess resistance to both CAMV and BlCMV and lines have been identified as useful differentials of the two viruses. However, it is also evident that some lines, including TVus 22, 612, 1453, 1948, 2331, 2480, 2657, 2740, 3433, Big Boy, Corona and Serido (Ladipo and Allen, 1979; Patel *et al.*, 1982a; Taiwo *et al.*, 1981; Bashir and Hampton, 1996), possess resistance to a range of isolates of diverse geographical origin. Other lines have isolate-specific resistance (Table 5.3). Sources of virus resistance including those with resistance combined against several distinct viruses (Allen, 1980, 1983) have now been widely utilized in cowpea breeding both in Nigeria (Singh *et al.*, 1987) and elsewhere in Africa (Kannaiyan and Haciwa, 1993).

Table 5.3. Reaction[1] of cowpea genotypes to mechanical inoculation with various isolates of blackeye cowpea mosaic and cowpea aphid-borne mosaic viruses.

Genotype of *Vigna unguiculata*	Isolate[2]							
	TZ	Nig1	Nig 2	Ken	Fla2	Flo	Mor	Cyp[3]
TVu 1582	+	–	+	+	+	+	–	–
TVu 1593	+	–	+	+	+	+	+	–
Serido	NT	NT	–	–	–	–	+	+
TVu 2845	+	+	–	–	–	–	+	+
TVu 3273	+	+	–	–	–	–	+	+
TVu 3433	+	+	–	–	–	–	+	+
TVu 2480	+/–	–	–	–	–	–	+	+

Compatible (susceptible) reactions denoted by +; incompatible (resistant) reactions denoted by –.
NT = not tested.
TZ = Tanzania isolate of Patel *et al.* (1982a); Nig 1 = Nigerian isolate of Ladipo and Allen (1979a);
Nig 2 = Nigerian isolate of Taiwo *et al.* (1982); Ken = Kenyan isolate of Taiwo *et al.* (1982);
Fla 2 = Florida isolate of Taiwo *et al.* (1982); Flo = New York isolate of Taiwo *et al.* (1982);
Mor = Moroccan isolate of Taiwo *et al.* (1982); Cyp = Cypriot isolate of Taiwo *et al.* (1982).
Mor and Cyp are considered isolates of CAMV by Taiwo *et al.*, (1982); all the rest are probably BlCMV.

WITCHWEED

Causal Agent

The witchweed of cowpea is the angiosperm root parasite, *Striga gesnerioides* (Willd.) Vatke, of the *Scrophulariaceae*. *S. gesnerioides* is an erect, annual autogamous species; though the plant bears green shoots its foliage is reduced to scale leaves, and so appears more dependent on host nutrients than its relatives. The

inflorescence consists of a loose spike of dull, mauve to pale purple flowers (Plate 8). The yellow-flowered *Alectra vogelii* Benth. also parasitizes cowpea in Africa (Rattray, 1932; Polniaszek *et al.*, 1991). Other parasitic weeds of legumes are reviewed in Jellis *et al.* (Chapter 7, this volume) and Bayaa and Erskine (Chapter 8, this volume).

Biology

S. gesnerioides is widely distributed throughout sub-Saharan Africa, from the semi-arid savannahs of West Africa, eastwards to Kenya (Agnew, 1974) and south to Malawi (Binns, 1968), Zimbabwe (Wild, 1954) and Botswana (Musselman *et al.*, 1991). It is found also in the Arabian peninsular, the Indian subcontinent (Lane and Bailey, 1992) and the USA, presumably to where the species was accidentally introduced (Musselman and Parker, 1981). The natural host range of *S. gesnerioides* includes tobacco, in the *Solanaceae*, the genera *Jacquemontia, Ipomoea* and *Merremia* in the *Convolvulaceae* and *Pterodiscus* in the *Pedaliaceae*, as well as various legumes which are the most important hosts. Musselman *et al.* (1991) give additional hosts in *Vitaceae, Euphorbiaceae* and *Acanthaceae*. Species of legume attacked include *Alysicarpus vaginalis, Indigofera hirsuta*, various *Rhynchosia* and *Tephrosia* spp., and *Vigna subterranea* as well as cowpea (Musselman and Parker, 1981; J.A. Bailey, Bristol, 1996, personal communication).

Host-specialized forms (morphotypes) of *S. gesnerioides* are recognized at the host species level and races (pathotypes) are distinguishable within host-specific populations. Isolates from tobacco are confined to *Nicotiana* (Wild, 1954) and, similarly, the indigo witchweed has a host range much narrower than *S. gesnerioides* as a whole. Isolates from cowpea vary in virulence (Parker and Polniaszek, 1990; Lane *et al.*, 1994) and now five physiologic races are distinguished by use of four differential cowpea cultivars, Blackeye, 58–57, IT 81D 994 and B 301 (Lane *et al.*, 1996). The geographical distribution of these races has been mapped (Fig. 5.12). It is not known whether additional diversity may exist among populations of *S. gesnerioides* on wild legume hosts, nor has the inheritance of virulence been investigated.

The host rhizosphere strongly influences the growth and development of witchweed seedlings. Host roots appear to exert a positive chemotropic response on the parasite's radicle, and subsequent branching of the root and shoot production of the parasite each depend on the secretion of growth substances from the host (Williams, 1961). The tip of the radicle swells at the site of host penetration, a primary haustorial bulb or tubercle develops and a shoot with scale leaves appears. Once vascular contact with the host is established, the parasite draws water and nutrients from the cowpea host, and the shoot grows and emerges after several weeks (Okonkwo and Nwoke, 1975). Seedlings of *S. gesnerioides* later produce secondary roots which, in turn, establish secondary haustoria. After emergence from the soil, *S. gesnerioides* is able to photosynthesize but most assimilates continue to be derived from the host (Press *et al.*, 1991).

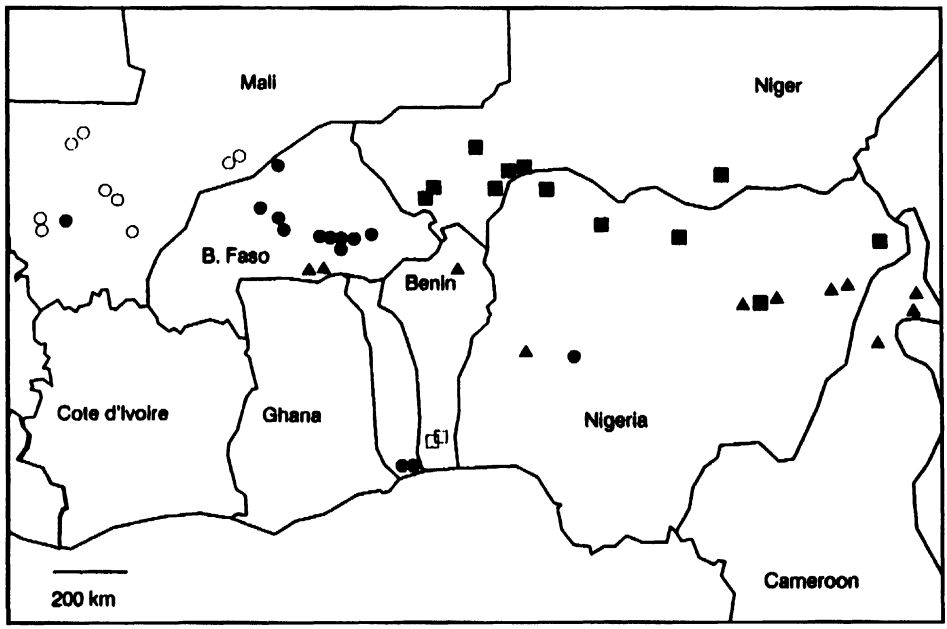

Fig. 5.12. The geographical distribution of races of *Striga gesnerioides* on cowpea in West Africa. Race 1 = filled circles; race 2 = open circles; race 3 = filled squares; race 4 = open squares; race 5 = triangles (Courtesy of J. A. Lane and the American Phytopathological Society).

Symptoms

Typically, affected plants develop an interveinal chlorosis (Plate 9) followed by a wilt and collapse of the plant within which vascular tissues are discoloured (Singh and Allen, 1979). *Striga* plants may or may not emerge before the appearance of chlorosis in the host. Heavy infestation causes foliar desiccation which may be preceded by the development of brownish-purple interveinal spots that then turn straw coloured as desiccation of the tissue sets in. Coalescence of spots leads to blighting of the lamina and rachis. Less severe attack causes mild chlorosis and plant stunting (Emechebe *et al.*, 1991).

Ecology and Epidemiology

In West Africa, *S. gesnerioides* is adapted particularly to the northern Guinea savannah, Sudan savannah and Sahel fringes (Emechebe *et al.*, 1991; Lane *et al.*, 1996), ecological zones that are subject to a long, dry intercrop period (Wien and Summerfield, 1980). In south-central Africa, *S. gesnerioides* is a plant of seepage slopes and decomposing rock in *Brachystegia* (miombo) woodland at altitudes between 450 and 1350 m (Banda and Morris, 1986). It is sometimes claimed that witchweed is common only on 'worn-out' agricultural land, though it is

more often considered to be the cause not the effect of soil infertility (Wilson-Jones, 1953), but there is evidence that increases in soil nitrogen decrease the incidence of witchweed infection (Emechebe *et al.*, 1991). *Striga* seed, which can survive in soil for periods of 7–14 years (Wilson-Jones, 1953; Berner *et al.*, 1994), tends to lose viability under conditions of high soil moisture (Parker, 1965). The reproductive capacity of *S. gesnerioides* is clearly very considerable: it is estimated that a single plant produces 50,000–500,000 seed (Wilson-Jones, 1953; Berner *et al.*, 1994). Because of its small size, it has been assumed that seed is principally windborne, as well as spread by water, animals and humans (Parker, 1965). However, a recent study has shown that witchweed seed is neither widely nor efficiently dispersed by wind: rather, it appears that dispersal on cowpea seed is common and may well account for most new establishments of witchweed, since only a single seed of the autogamous *S. gesnerioides* is needed to form a new focus of infestation. Witchweed seed is also dispersed locally in cattle dung, and perhaps also on hooves, in animal fodder and by birds (Berner *et al.*, 1994).

Crop Loss

As an obligate parasite, *S. gesnerioides* is completely dependent upon its cowpea host during the early stages of its development below soil level, and it is during this time that maximum damage to crop growth and subsequent seed yield occurs (Lane and Bailey, 1992). Cowpea seed yield has been estimated to be decreased by 20–60%, with an average of 30% in susceptible cultivars, irrespective of plant type and maturity. Since infestation in their study did not reach 100%, Aggarwal and Ouedraogo (1989) concluded that theirs was an underestimate, borne out by field observations that suggest complete crop loss can occur (Singh and Emechebe, 1990; Reiss *et al.*, 1995). Yield loss cannot be attributed to witchweed's effect on any one yield component alone (Aggarwal and Ouedraogo, 1989).

Control

Cowpea and its witchweed have co-evolved in the cereal farming systems of the African savannahs, traditional systems that have entailed complex patterns of intercropping and rotation interspersed with long periods of fallow (Steele *et al.*, 1985). There is evidence, discussed for instance in Allen (1983), that co-evolved crops in traditional farming systems have achieved an equilibrium not only with one another but also with their parasites. Thus, it seems that witchweed populations have been kept at tolerable population densities, perhaps with the added influence of the gall weevil (*Smicronyx* sp.) that parasitizes *S. gesnerioides* (Williams and Caswell, 1959). Cultural control practices have also included hand-pulling and hoeing of witchweed plants, and the use of both catch crops and trap crops, the latter stimulating *Striga* seed to germinate without themselves becoming infected (Wild, 1948; Lagoke *et al.*, 1991). Together, these methods

continue to be the chief means of witchweed control currently available to the small-scale farmer.

With intensification of cowpea production in some areas of the West African savannahs, populations of *S. gesnerioides* appear to be increasing both in density and range (Berner *et al.*, 1994), indicating that the intrinsic balance of the traditional farming system has been upset so that greater intervention is now required to manage the witchweed. Chemical control, aimed both at preventing witchweed reproduction and depleting the soil of viable seed, is employed in the USA where both foliar- and soil-applied herbicides have been shown to be effective. The use of seed germination stimulants, including ethylene gas which is injected under pressure into the soil, is partially effective in depleting witchweed seed reservoirs in the soil in the absence of a susceptible host. Analogues of strigol, a natural witchweed stimulant, may also have potential in inducing suicidal germination of *Striga* seed, but soil fumigation with materials including methyl bromide is the only procedure currently available to kill witchweed seed in soil. Various systemic herbicides including dicamba are effective against witchweed when applied as foliar sprays, and pre-emergence herbicides like metalachlor suppress *Striga* attachment to its host (Eplee *et al.*, 1991). Imazaquin applied to cowpea as a seed dressing also has been shown to decrease attachment and emergence of *S. gesnerioides* (Berner *et al.*, 1992). However, there are two major constraints to the use of herbicides against witchweed: cost and availability. It seems unlikely that chemical control alone will become widely used in the farming systems of which cowpeas are part in the tropics. Herbicides may have a part to play under special circumstances in local eradication, particularly as a component of an integrated witchweed management strategy.

The most promising approach against cowpea witchweed, and perhaps the area in which the most spectacular progress has been made in cowpea pathology over recent years, is the identification and incorporation of *Striga* resistance into cowpea cultivars for the African savannahs, a topic reviewed by Aggarwal (1991). In 1981, a 'sick-plot' was first established at Kamboinse, near Ouagadougou in Burkina Faso, where a set of cowpea germplasm was screened for resistance against *S. gesnerioides*. Two lines, SUVITA-2 and 58–57, were found free of witchweed and subsequent tests confirmed their resistance under both greenhouse and field conditions. Testing across locations soon revealed that resistance was site-specific: whereas SUVITA-2 proved resistant to witchweed isolates from Burkina Faso and Mali, 58–57 proved resistant only to isolates from Burkina Faso; each was susceptible to samples from elsewhere in West Africa, so confirming that the site effects were attributable to variations in virulence (Parker and Polniaszek, 1990). A line from Botswana, B 301, which had been selected for its field resistance to *Alectra vogelii*, proved resistant to all populations of *S. gesnerioides* to which it was exposed. An *in vitro* screening technique (Lane *et al.*, 1993) has led to the identification of further sources of resistance, including two West African landraces (APL-1 and 87–2) and a breeding line (IT82D-849). Most known sources of resistance appear to be race-specific (Aggarwal, 1991; Moore *et al.*, 1995; Lane *et al.*, 1996). Several distinct mechanisms of resistance can be discerned. For instance, in the cowpea line 58–57, host tissue around invading radicles may become necrotic, tubercles do not form and the parasite

dies early. A second mechanism, observed in B 301, is characterized by radicles infecting the roots and the formation of tubercles, but their subsequent growth is very limited and the parasite develops no further. In neither type is resistance due to decreased parasite germination, nor have *Striga*-tolerant cultivars of cowpea been identified; each mechanism is known in cereals against their witchweeds (Lane *et al.*, 1993; Reiss *et al.*, 1995). Witchweed resistance in B 301 is governed by a single dominant gene assigned the symbol R_{sg1} (Singh and Emechebe, 1990) and has been shown to be non-allelic and independent of the duplicate dominant gene controlling resistance to *Alectra vogelii* in this cultivar (Atokple *et al.*, 1992; Singh *et al.*, 1993). Additional independent genes, R_{sg2} and R_{sg3}, are available in the witchweed resistant lines IT82D-849 and SUVITA-2, respectively (Atokple *et al.*, 1995).

Now that the sympatric distribution of races of *S. gesnerioides* is known, armed with an understanding both of the expression and genetic control of witchweed resistance, there seem to be good opportunities for the cowpea breeder to pyramid these genes in cultivars and to deploy them judiciously throughout West Africa. Good progress has already been made in incorporating the combined resistance to both *Striga* and *Alectra* from B 301 into backgrounds of improved plant type, together with resistance to a range of other biotic constraints (Atokple *et al.*, 1995; Lane *et al.*, 1996).

PROBLEMS, PROGRESS AND PROSPECT

Rapid progress has been made in cowpea pathology most notably since the inception of a research programme at IITA in about 1970. Work accomplished by this programme and its many collaborators up to 1984 has been critically reviewed elsewhere (Allen, 1982, 1983; Emechebe and Shoyinka, 1985; Thottappilly and Rossel, 1985). Perhaps the most spectacular progress made subsequently has been in the understanding of witchweed and its management, as reviewed in detail in this chapter.

Good progress has also been made in elucidating the aetiology of several other diseases. Ribosomal DNA sequence analysis has led to better understanding of relations among species of *Colletotrichum*, with implications for the identity of the cowpea anthracnose pathogen (Sherriff *et al.*, 1994; Latunde-Dada *et al.*, 1997). It remains to be seen whether isolates from locations outside Nigeria have the same aetiology. Fundamental work on relations among legume potyviruses (Dijkstra *et al.*, 1987; Lana *et al.*, 1988; Khan *et al.*, 1993) has led to a clearer understanding of relatedness of blackeye cowpea mosaic virus to bean common mosaic and cowpea aphidborne mosaic viruses: comparison of nucleotide sequences has revealed that the coat proteins of certain isolates of BlCMV and BCMV are sufficiently similar to indicate they are strains of the newly re-defined BCMV. Conversely, there are gaps in our understanding of a number of other diseases, the most important of which is scab. Beyond the work of Emechebe (1980), we remain ignorant of the relationship between *Sphaceloma* and its putative teleomorph, *Elsinoe phaseoli*, and little is known of pathogenic variation within the cowpea pathogen or between the cowpea scab fungus and the scab fungi of other legumes, many of which are con-

ventionally considered to belong to the same species. There is considerable host specificity within this group and much more work on these pathogens is warranted, as stressed in Allen (1983) and by Allen and Lenné (Chapter 1, this volume). The work of Phillips (1994a, b, 1995) is pertinent.

Among virus diseases, the most conspicuous gap in knowledge remains the aetiology of cowpea golden mosaic, the epidemiology of which has received substantial attention (Anno-Nyako *et al.*, 1983; Vetten and Allen, 1983). There is some evidence that a geminivirus is involved in its aetiology in Nigeria (IITA, 1980). Similar diseases of cowpea occur in Niger, Kenya, Tanzania (Singh and Allen, 1979), Mozambique (D.J. Allen, 1989, unpublished observation), Pakistan (Ahmad, 1978) and Brazil (Lin and Rios, 1985) but comparisons between them must await the fuller characterization of the causal agents.

Sound methods of virus detection have enabled the mapping of both the geographical and ecological distribution of cowpea viruses in Africa (Fig. 5.10). There may be a case for better documentation of the distribution of cowpea pathogens and their variants, as has been done with common bean in Africa (Allen, 1995), both as a basis for updating quarantine legislation and as a guide to national cowpea breeding teams in setting their objectives. A better understanding of the nature and extent of pathogenic variation in pathogens including *Colletotrichum* is clearly necessary, and it has been suggested (Singh *et al.*, 1992) that restriction fragment length polymorphism and polymerase chain reaction biotechnology may prove useful in this respect.

These perceived gaps in research should not detract from the solid progress made toward the identification of combined resistance to a wide range of diseases (Williams, 1977a; Allen, 1978, Allen *et al.* 1981b) and its effective incorporation in improved cultivars (Smithson *et al.*, 1980; Patel *et al.*, 1982b; Singh *et al.*, 1987; Singh 1994).

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DISEASES OF PEA

6

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INTRODUCTION

The pea (*Pisum sativum*) is considered among the earliest cultivated plants. Archaeological evidence indicates that peas were cultivated in the Near East and Greek Neolithic settlements around 6000 BC (Marx, 1977). It is difficult to determine a time for domestication of peas because carbonized seeds are all that remain. Among the few shreds of evidence for domestication is the seed coat surface, a rough or granular testa being the primitive trait and a smooth surface being characteristic of cultivated varieties. Thick or hard seed coats are also considered primitive traits for they are associated with scarification required for germination. In addition, dehiscent pods which expel and scatter seeds when mature are also considered a primitive trait.

Wild and primitive forms are found in the Near East and areas of central Asia, the Mediterranean and Ethiopia. The dispersal of peas throughout the world is directly associated with the migrations and activities of humans. Whether the crop was simply collected or cultivated, peas were desirable to early man. The protein- and carbohydrate-rich seeds could be stored and transported easily, and for much of recorded history peas were grown primarily for dry seed. Hedrick *et al.* (1928) state that peas were called *pisos* by the Greeks and *pisum* by the Romans, and became 'peason', then 'pease', and finally 'peas' by the English. Peas became an important crop in northern Europe in the Dark and Middle Ages and were grown as commonly as cereals. Peas became a chief crop in England and significant improvements were made there by such pioneers as Thomas Andrew Knight. Knight introduced several improved cultivars with wrinkled seed and many of today's commercial cultivars have parents that trace their origins to those developed in England during the nineteenth century.

Today, peas fall into two main categories; those that are harvested at the green, immature stage and those harvested when fully mature as seed. Those harvested at the green stage are further subdivided into vining peas, which are

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harvested and shelled mechanically for processing as canned and frozen, and those which are picked by hand and marketed as fresh peas. Peas used for human consumption and harvested when fully mature are called dried peas. An increasing proportion of dried peas are now being grown for animal consumption as a substitute for soyabeans (*Glycine max*) and are called combining or protein peas.

Peas are subject to a number of bacterial, fungal, viral and nematode diseases which can significantly decrease both yield and quality. Soilborne fungal diseases discussed in this chapter include seedling diseases and root rots. Two important seedling diseases are *Pythium ultimum* Trow. and *Rhizoctonia solani* Kühn. Factors which delay emergence or result in uneven plant stands, such as cold wet soil, poor seed vigour (Rush, 1987), and herbicide injury, can predispose young, developing plants to seedling disease. Processing or vining peas are frequently planted in early spring into cold, wet soil. Often emergence can take as long as 6–8 weeks. The ability to germinate and emerge under cold, wet conditions is defined as seed vigour. One test to measure seed vigour is the electroconductivity (EC) test as described by Biddle *et al.* (1988). This test measures the amount of inorganic salts released during imbibition. The higher the EC rating, the greater the risk of planting this seed into cold, wet soil. Smooth-seeded peas do not exude comparable amounts of carbohydrates and inorganic salts relative to wrinkle-seeded peas and are less prone to seed and seedling infection.

Root rots of peas are caused by a number of different soilborne fungal pathogens that can produce similar symptoms. This disease complex can encompass the entire root system and even extend a short distance above the soil surface. Root rots are enhanced by short rotations and conditions of plant stress. Conditions which limit root growth, such as compaction, poor fertility, anoxia, excess or deficient moisture, herbicide injury, and suboptimal temperatures can all increase the damage due to root rot (Allmaras *et al.*, 1988). Root rots are exacerbated by production of pea crops in short rotations. Serious root rots are caused by *Aphanomyces euteiches* and *Fusarium solani* f. sp. *pisi*; are wilts caused by *Fusarium oxysporum* f. sp. *pisi* races 1, 2, 5 and 6.

Foliar diseases caused by fungal pathogens include white mould caused by *Sclerotinia sclerotiorum*, powdery mildew caused by *Erysiphe pisi*, downy mildew caused by *Peronospora pisi* and ascochyta blight caused by *Ascochyta pisi*, *Mycosphaerella pinodes*, and/or *Phoma medicaginis* var. *pinodella*. Foliar disease caused by bacteria is primarily due to the bacterial blight organism *Pseudomonas syringae* pv. *pisi*.

Yield losses and reduction of quality due to virus diseases can be substantial during years of epidemic virus incidence. All the virus disease agents discussed in this chapter are aphid transmitted and can overwinter in perennial legumes such as clover and alfalfa (Hagedorn, 1974). In this section, we discuss five of the important viruses which occur in peas as well as other legume crops, including lentil, chickpea, faba bean, clover and alfalfa, and these include pea enation mosaic, pea (bean) leaf roll, alfalfa mosaic, pea seedborne mosaic, and pea streak (alfalfa latent virus). The transmission of non-persistent viruses, such as pea seedborne mosaic potyvirus, can occur more rapidly by aphid species which do not have peas as a preferred host. Aphid species which do not prefer peas are more transient in their feeding habits and are more difficult to

detect in field monitoring. Monitoring fields for pea aphid build-up may be totally inadequate in reducing virus spread, especially with a potyvirus. Growing a virus-resistant cultivar, monitoring aphid populations, and awareness of perennial virus sources are essential for a healthy crop. Also discussed is the problem caused by pea cyst nematode. Diseases of only minor or local importance are summarized in Table 6.1.

PYTHIUM SEED AND SEEDLING ROT

Causal Pathogen

Pythium ultimum Trow. var. *ultimum* (Van der Plaats-Niterink, 1981) and sporangial forms similar to *P. ultimum* are the most frequently described pathogens of peas in the genus *Pythium*. Other species including *P. aphanidermatum*, *P. irregulare*, *P. splendens*, *P. debaryanum*, *P. acanticum*, *P. spinosum* and *P. andrum* have been described as pathogens of peas. Oogonia of *P. ultimum* are smooth, spherical and terminal. Antheridia of *P. ultimum* are monoclinous and arise just below the oogonium with usually one per oogonia. Oospores are aplerotic, single, spherical, smooth and thick-walled with an average diameter of 22 μm. Germination is via a germ tube. Sporangia are usually terminal and always spherical. Some isolates of *Pythium*, obtained from rotted seeds or seedling roots, resemble *P. ultimum* but do not produce oospores. *Pythium* is most easily isolated on water agar, diluted V8-juice agar, or various selective media.

Biology

Diseases of pea caused by *Pythium* spp. are referred to as seed rot, damping-off or root rot. *P. ultimum* also causes root rot of chickpea (see Haware, Chapter 9, this volume). Temperatures below 23°C are most favourable for infection. Figure 6.1 illustrates the root pruning and seedling damping-off characteristic of this disease in young pea seedlings. *Pythium* damage is characterized by a watery, soft rot and the infection of juvenile tissues, such as root tips. The pathogenic capacity of any given isolate will largely be determined by the production of pectolytic and cellulolytic enzymes. Damage by *Pythium* tends to be more severe when soil moisture is high and soil temperatures are in the 10 to 15°C range (Kraft *et al.*, 1988).

Control

Appropriate seed treatment chemicals, especially for wrinkled-seeded varieties, planting seed with high germination rates, and cultural practices that promote rapid germination, emergence, and uniform plant stands should help to minimize this disease problem. Such factors as planting too deep, soil crusting, poor seed vigour, and herbicide injury will often increase *Pythium* seed and seedling damage.

According to Stasz *et al.* (1980), there are three types of genetic resistance in

Table 6.1. Diseases of pea of local or minor importance.

Disease	Causal agent	Symptoms	Control
Anthracnose	*Colletrotrichum pisi* Pat.	Foliar symptoms are irregularly oval, 2–8 mm in diameter, grey to tan, brown on margin. Pod lesions are circular, sunken, and red in colour	Use disease-free seed and crop rotation practices
Septoria	*Septoria pisi* West.	Lesions are not distinct on lower portion of plant. Yellow to straw coloured, numerous pynidia develop in a scattered pattern	Crop rotation and disease-free seed
Cladosporium blight	*Cladosporium pisicolum* Cugini & Macch.	Lesions on leaves and stipules are circular or irregular, under humid conditions, immature lesions covered with grey spores. Stem lesions are elongated and brown or black. Pods can be distorted with dark pimples, both raised and sunken	Crop rotation where disease is present. Use clean seed
Grey mould	*Botrytis cinerea* Pers. ex. Fr.	Fuzzy, grey lesions that streak or encircle stem. Infected leaves become dry, grey, and shrivelled as infection progresses. Pod damage can cause economic loss	Apply potassium if deficient. Use systemic fungicides
Black root rot	*Thielaviopsis basicola* Berk. & Br.	Black necrosis of tap root and lateral roots. Abundant chlamydospores form in diseased root tissue	Avoid planting in fields with high inoculum levels of *T. basicola*
Pea early browning	Pea early browning virus, transmitted by stubby root nematodes (*Paratrichodorus terres*, *P. pachydermus* in the Netherlands, *Paratrichodorus primitivus* and *Trichodorus viruliferus* in the UK)	Irregular purplish-brown necrotic discolorations on stems and leaves. Vascular necrosis and localized wilt follows vein necrosis. Stunting and distortion with overall yellow hue or mottling	Use clean seed. Preplant nematicide where stubby root nematode and virus are each present

Fig. 6.1. Root pruning and root tip necrosis caused t
J.M. Kraft).

peas to *Pythium* seed and seedling rot: (1) germinating seeds lose susceptibility within 48 h after imbibition begins, which decreases the number of rotted seeds; (2) round-seeded peas exude reduced amounts of substances stimulatory to *Pythium*; and (3) peas with pigmented seed coats are resistant to pythium rot due to the presence of fungistatic, anthocyanin compounds. Seeds with pigmented seed coats can be susceptible if sufficient sugars are also released, which allows *Pythium* to overcome the fungistatic affects of anthocyanins (Kraft, 1978). Haware (Chapter 9, this volume) also noted that chickpea with pigmented *desi*-type seed are more resistant to *P. ultimum*. There are a number of pea breeding lines that have shown resistance or tolerance to *Pythium* under greenhouse conditions and in the field where no seed treatment chemicals are used (Kraft *et al.*, 1988).

RHIZOCTONIA SEEDLING BLIGHT

Causal Pathogen

Rhizoctonia solani Kühn is most often named in defining this seedling pathogen and is classified using the anastomosis grouping (AG) concept whereby hyphal fusion occurs only between isolates of the same AG grouping. The pea pathogen is assigned to the AG-4 grouping (Anderson, 1982). Isolates in this group usually cause seed, epicotyl, and hypocotyl rots, and isolates in the AG4 group are considered the practicola type in that the perfect stage is listed as *Thanatephorus practicola* (Frank) Kotila (Anderson, 1982). *R. solani* is a serious disease of both adult plants and seedlings of *Centrosema* spp.; however both AG-1 and AG-4 groups are involved (see Lenné, Chapter 13, this volume). Binucleate *Rhizoctonia* spp. affect lupin seedlings (see Hill, Chapter 11, this volume). The mycelial branches of *R. solani* are characteristically at right angles and are restricted at the point of attachment. Thick-walled sclerotia, the survival structure of this fungus, are formed by multiple hyphal branching and aggregation. *Rhizoctonia* can survive in field soil for extended periods of time and can attack emerging pea seedlings when conditions are favourable. For seedling infection to occur, the sclerotium or hyphal fragment must germinate or resume growth. This fungus can grow several millimetres through soil to form an infection cushion on the host epicotyl surface prior to penetrating the host. Seedlings most often become resistant or less susceptible with age. This fungus is limited to the top few centimetres of soil, and plant parts at or near the soil surface are most vulnerable.

Biology

Rhizoctonia seed and seedling rot occurs more frequently in fields that are trashy tilled or in no-till and reduced tillage farming systems. The disease is usually most severe where conditions are warm and moist, especially in lighter or sandy soils. In irrigated areas of the Pacific Northwest, this disease can be devastating where susceptible crops such as dry beans (*Phaseolus vulgaris*) are planted prior to peas and the residue is disked but not ploughed. Symptoms of this disease include a water-soaked appearance of the hypocotyl and epicotyl, and reddish-brown or brown lesions above and below the cotyledonary node. Figure 6.2 illustrates typical symptoms of *Rhizoctonia* damage to seedling peas. Especially diagnostic is the death of stem radicles as they emerge. Often, auxiliary stems are also affected so that the plant never fully recovers. On older plants, reddish-brown, sunken lesions may occur on the epicotyl, sometimes resulting in girdling and severe plant stunting.

Control

There are no resistant pea cultivars, but those which are vigorous, have thick epicotyls, and emerge rapidly can escape serious damage (McCoy and Kraft, 1984).

Fig. 6.2. Seedling stem necrosis, seed rot caused by *Rhizoctonia solani*. Observe secondary shoot development (Photo: courtesy of J.M. Kraft).

Rotation with cereals, clean tillage prior to planting, and fungicidal seed treatments provide some protection. Biological seed treatment organisms, such as *Trichoderma harzianum* Rifai, have also shown promise for protecting germinating seed against rhizoctonia seed and seedling rot (Kraft and Papavizas, 1983).

APHANOMYCES ROOT ROT

Causal Pathogen

Aphanomyces root rot is caused by the fungus *Aphanomyces euteiches* Drechs. Pfender and Hagedorn f. sp. *pisi*, a water mould of the family *Saprolegniaceae* (Hagedorn, 1984). Cultures of *A. euteiches* grow sparsely in an arachnoid fashion on a minimal medium, but on a rich medium, such as maltose–peptone agar, it produces copious, whitish, aerial mycelium. Mycelia are 3–10 μm in diameter, without cell walls which are relatively sparsely branched and almost at right angles and the branches are relatively short. This fungus produces two types of zoospores (diplanetism). Primary zoospores are first extruded from the sporangia in single file and encyst at the mouth of the sporangium. Usually in 1–3 h these encysted zoospores germinate to form secondary zoospores which possess two flagella and are the motile, infective form of this pathogen. Secondary zoospores are *c.* 13 μm long and 7–8 μm in diameter. Motile zoospores will move in the soil water, surround soil particles, and can encyst, germinate, and infect or re-infect pea root tissue.

Oospore formation occurs when the fungal thallus is exposed to nutrient deficiency or adverse conditions within root cortical tissue. Thin-walled oogonia are fertilized by one to five diclinous antheridia. Resultant oospores are hyaline, spherical, 18–25 μm in diameter, and thick-walled with a characteristic, large oil globule surrounded by granular material (see Fig. 6.3).

Biology

Aphanomyces root rot, which is one of the most important root pathogens of peas worldwide, has been reported to occur in the Midwest, Northeast, and Pacific Northwest regions of the United States, in northern Europe, and in Australia, New Zealand and Japan (Hagedorn, 1984). *Aphanomyces euteiches* can infect peas at any stage of plant development. Visible lesions can appear 1–2 weeks after infection, depending upon the environment. Plate 10 illustrates typical symptoms of aphanomyces root rot on roots of seedling plants. Typically, soft, water-soaked lesions appear on the surface of the lower stem and root, which then become tan coloured. The lesions spread through the cortex causing eventual discoloration of the entire root system (Kraft *et al.*, 1988). The infected cortical tissue darkens as other organisms invade. When infected plants are pulled from soil, a strand of vascular tissue is often all that remains of the root system. Microscopic observation of infected, cortical tissue reveals diagnostic, thick-walled oospores that can survive for years in soil. Dissemination of *A. euteiches*

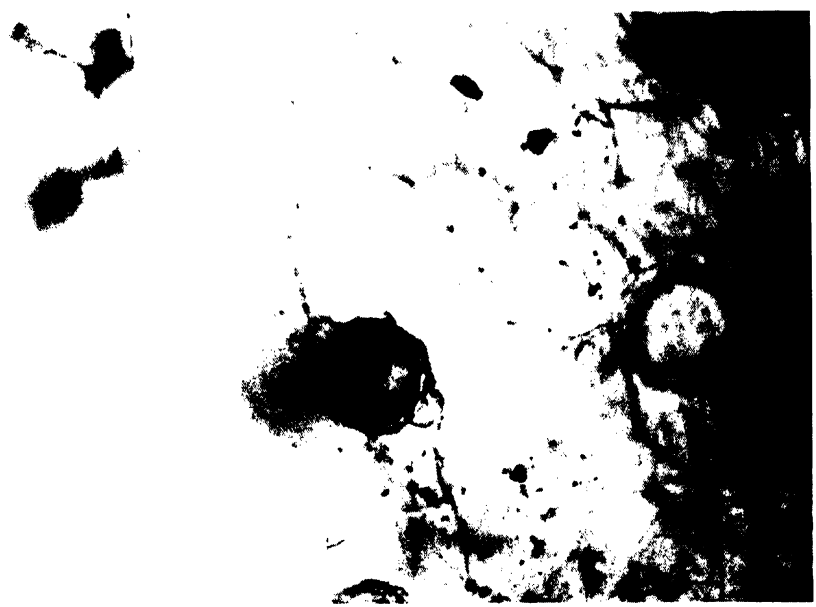

Fig. 6.3. Mature oospore of *Aphanomyces euteiches* embedded in cortical tissue of a pea root. Note diagnostic, large oil globule in centre of oospore. Oospore is *c.* 25–30 μm in diameter (Photo: courtesy of J.M. Kraft).

occurs in infested soil by water movement, infected root debris, or by farm implements. Other legumes, such as alfalfa (*Medicago sativa*), common bean, faba bean (*Vicia faba*), clover (*Trifolium* spp.) and lentil (*Lens culinaris*) are also hosts for *A. euteiches*.

Control

Control of aphanomyces root rot is extremely difficult. Once a field is heavily infested, a non-host (wheat, oats, barley) must be planted for at least 10 years before a profitable pea crop can be grown again. All potential hosts should be avoided as crops to include in any rotation (Grau *et al.*, 1991). Oats (*Avena sativa*) have been shown to aid in reducing the inoculum potential of *A. euteiches*, perhaps due to the production of fungistatic compounds called saponins in the roots and tops (Fritz *et al.*, 1995). Soil can be tested for aphanomyces root rot potential to avoid severely or heavily infested fields (Sherwood and Hagedorn, 1958; Kraft *et al.*, 1990). If the root rot potential is moderate to severe, the field is not recommended for peas. If the root rot potential is moderate to slight, an early-maturing cultivar should be planted. Currently, there are no commercially available seed treatment chemicals or biological control organisms that will consistently control this disease. However, several experimental compounds and organisms have shown promise (Kraft *et al.*, 1995).

Genetic resistance to *A. euteiches* appears to be quantitatively inherited with low heritability (Lewis and Gritton, 1988). Resistance is associated with slower lesion development and pathogen build-up (Kraft and Boge, 1994, 1995). Currently, no pea cultivars are available with resistance to this disease. However, over the last 10 years, several resistant/tolerant germplasm lines with horticulturally acceptable traits have been released (Kraft and Kaiser, 1993). Resistance/tolerance, together with cultural practices (Fritz *et al.*, 1995), improved seed-treatment chemicals, and soil indexing, will help reduce the economic impact of this disease problem in the future.

FUSARIUM ROOT ROT

Causal Pathogen

Fusarium root rot of pea is caused by *Fusarium solani* (Mart.) Appel & Wr. f. sp. *pisi* (F.R. Jones) Snyd. & Hans. In culture, this pathogen produces sporodochia which are blue-green to buff in colour (Kraft *et al.*, 1981). Root rots caused by *Fusarium* spp. on soyabean, chickpea and pigeonpea are reviewed by Sinclair (see Chapter 3, this volume), Haware (see Chapter 9, this volume) and Reddy *et al.* (see Chapter 10, this volume), respectively. Macroconidia are primarily three-septate, 4.4–5 μm × 27–49 μm, curved and hyaline. Microconidia are less abundant, except in liquid culture where they are numerous. Chlamydospores, produced in the mycelium or by conversion of conidia, are abundant, intercalary, terminal, single or in chains. The teleomorph, *Nectria haematococca* Berk. & Br.

(Kraft *et al.*, 1981) has been found in nature only on diseased branches of mulberry (*Morus* sp.) in Japan. Matuo and Snyder (1972) reported that *F. solani* f. sp. *pisi* was identical, by mating tests, with the pathogen causing branch blight of mulberry trees and root rot of ginseng (*Panax* sp.). Distinct heterothallic isolates of *F. solani* f. sp. *pisi* exist and do not intercross; however, homothallic isolates are also prevalent. This pathogen can be isolated from infected plant material and from field soil on acidified potato dextrose agar (PDA) or by use of a *Fusarium* selective medium such as PCNB (Nash and Snyder, 1962).

Biology

Fusarium root rot of pea has been reported as a serious pathogen in Minnesota, Wisconsin and the Pacific Northwest of the USA, as well as in Europe (Kraft *et al.*, 1981). In pea seedlings, the initial centre of attack by *F. solani* f. sp. *pisi* is the cotyledonary attachment area, below-ground epicotyl, and upper tap root. Figure 6.4 illustrates typical symptom expression of *F. solani* f. sp. *pisi* on the epicotyl, hypocotyl, and cotyledonary attachment area. Infection extends upward to the soil line and downward into the root zone. The degree of root infection and damage depends on the soil environment (Allmaras *et al.*, 1988). A red discoloration of the vascular system may occur in the root but usually does not progress above the soil line. Only when primary and secondary roots are infected by *F. solani* f. sp. *pisi* is the disease serious enough to adversely affect that plant's productivity. Above-ground symptoms consist primarily of stunted growth and yellowing of the basal foliage. Initial symptoms on seedling roots consist of reddish-brown to blackish-brown streaks which coalesce.

Fusarium root rot is enhanced by conditions adverse to root growth, including soil compaction, soil temperatures exceeding 30°C, soil moisture contents of -0.5 to -1.2 MPa, soil acidity (pH lower than 5.1), and poor soil fertility (Kraft *et al.*, 1981; Allmaras *et al.*, 1988; Kraft *et al.*, 1988). Any factor, such as soil compaction, that significantly reduces the rate and distribution of rooting, increases the chances of *Fusarium* contact with the pea root. In friable, well-aerated soil, pea roots can grow at a nominal rate of 0.4 mm h^{-1} and the rhizosphere influence is approximately 1 mm away from the root surface (Huisman, 1982). The root apex and region of elongation are the sites of most root exudation. The resting spore (chlamydospore) of *F. solani* f. sp. *pisi* may detect the approaching root tip, via root exudates stimulating germination, only a few hours before the root arrives. To ensure contact with a root tip, a propagule must germinate 1–3 h after receiving a stimulus from the root. Such germination times have been recorded for *Pythium* spp., but not for *Fusarium* chlamydospores which do not germinate as quickly. These differences in germination times help to explain why *Pythium* attacks root tips and *F. solani* f. sp. *pisi* attacks the stationary cotyledonary attachment, epicotyl and hypocotyl areas. It is only when root growth ceases because of stress factors like compaction, anoxia, etc., that *F. solani* f. sp. *pisi* attacks the root tips, resulting in severe plant injury.

Fig. 6.4. Hypocotyl, epicotyl and cotyledonary attachment area infected with *Fusarium solani* f. sp. *pisi*. Lesion colour is reddish to blackish-brown. This area (crown) is the primary centre of attack by *F. solani* (Photo: courtesy of J.M. Kraft).

Control

Where fields are heavily infested with *F. solani* f. sp. *pisi*, peas should not be planted more frequently than once in 5 years. The yield constraints of this disease can be significantly reduced by tillage practices to reduce soil compaction, better fertility, and practices promoting favourable soil moisture and root penetration.

Genetic resistance of fusarium root rot has been reported to be dominant and affected by cytoplasmic factors (Knavel, 1967). Resistance in pea to fusarium root rot is evidenced by lower disease indices and higher fresh weights of plants when grown in *Fusarium*-infested ground (Kraft *et al.*, 1988). Seedling exudates and seedling vigour directly affect susceptibility and resistance to *F. solani* f. sp.

pisi (Kraft, 1986). In fact, resistant lines can be predisposed to fusarium root rot by physiologic ageing of seed (Kraft, 1986). Resistance to fusarium and pythium root rot are thought to be conditioned by the same genetic factors (Muehlbauer and Kraft, 1973). Currently, no commercial cultivars are resistant to *F. solani* f. sp. *pisi*. Germplasm with measurable levels of resistance to this pathogen has been released and commercially acceptable cultivars are likely to be developed within the next few years (Kraft *et al.*, 1988).

FUSARIUM WILT

Causal Pathogen

Fusarium wilt is caused by several races of *Fusarium oxysporum* Schlecht. emend. Snyd. & Hans. f. sp. *pisi* (van Hall) Snyd. & Hans. Wilt caused by *Fusarium* spp. is also a major disease of lentil (Bayaa and Erskine, Chapter 8, this volume), chickpea (Haware, Chapter 9, this volume) and pigeonpea (Chapter 10, this volume). According to Booth (1971), *Fusarium oxysporum* belongs to the section *Elegans* of the genus *Fusarium*. Snyder and Hansen (1940) reduced all the species in this group to be synonyms of *F. oxysporum*. Snyder and Hansen then designated 25 forms of this species based primarily on pathogenic host specificity. Hence, f. sp. *pisi* indicates specificity to peas. The average growth rate *in vitro* is *c.* 4.5 cm day^{-1}. Mycelium is usually white, often with a purple or pink tinge. Microconidia are borne on simple phialides or from short, sparsely branched conidiophores. Microconidia are abundant, variable, oval–ellipsoid, cylindrical, straight to curved, 5–12 × 2.2–3.5 μm. Macroconidia are sparsely produced. Colony morphology is sporodochial to aerial. Pionnotal colony morphology usually indicates low or avirulence. Because of the high genetic variability in races 1, 2, 5 and 6, isolates of these races should be maintained in a dormant state, such as autoclaved soil (Toussoun and Nelson, 1968), to reduce vegetative growth as much as possible.

Biology

By 1928, fusarium wilt of pea was reported to occur in most pea growing areas of the United States. Races 1 and 2 can be economically important in most pea growing areas of the world (Kraft *et al.*, 1988), whereas races 5 and 6 are reported to cause economic losses to pea crops primarily in north-western Washington state and British Columbia (Hagedorn, 1984). Races 3 and 4 are considered to be variants of race 2 (Kraft and Haglund, 1978), based on the reaction of pea cultivars with known single gene resistances to races 1 and 2.

Fusarium oxysporum f. sp. *pisi* is a soil-inhabiting fungus that survives as thick-walled chlamydospores, primarily in the tilled layer, and is fairly host-specific. Invasion of a susceptible host is thought to occur through root tips (Kraft *et al.*, 1981). Symptoms due to races 1, 5 and 6 include downward curling of leaves and stipules. The basal internode may be thickened, and the leaves and

stems can become more brittle and rigid than leaves of healthy plants. The root system appears normal. However, a longitudinal section may have a yellow to orange colour in the vascular tissue of the root and stem. Often this vascular discoloration will extend to the upper stem. As the disease develops, yellowing progresses from the lower leaves to the stem apex. At soil temperatures of 20°C and above, fusarium wilt develops rapidly, resulting in plant death. In fields where races 1, 5, and 6 are prevalent, symptoms usually occur in small to large patches.

Symptoms of race 2 on individual plants are similar to symptoms of races 1, 5 and 6. Usually, field symptoms consist of occasional plants exhibiting symptoms unless the inoculum level is extremely high. Symptoms of race 2 usually occur later in the growing season, often at bloom; hence the name near wilt has been given to these symptoms. Often, vascular discoloration caused by race 2 is more intense being an orange or dark red in colour. Plate 11 illustrates vascular discoloration of the above-ground stem, typical of pea wilt.

Resistance to races 1, 2, 5 and 6 is governed by single, separate, dominant gene factors in the host. Table 6.2 lists a number of differential pea lines which can be used to determine the race designation of any isolate of *F. oxysporum* f. sp. *pisi*. Using near-isogenic lines differing by one major gene for resistance/susceptibility to races 1 and 5, the response of resistant cultivars to each race was similar. The resistance response was based on physical containment and reduced fungal growth in lateral roots, hypocotyl and epicotyl regions, and no colonization of the upper stem in cultivars resistant to either race (Charchar and Kraft, 1989). The wilt pathogen can sustain itself and even increase in population on resistant cultivars. However, the resistance to all economically important races of fusarium wilt has been stable. There is an array of wilt-resistant, commercial varieties

Table 6.2. Response of differential pea lines to the fusarium wilt pathogen, *F. oxysporum* f. sp. *pisi*.

		Wilt reaction[2]			
Pea line	Source[1]	R1	R2	R5	R6
M 410	Brotherton	S	S	S	S
Vantage	Brotherton	R	S	S	S
Mini	Asgrow	S	R	S	S
Mini 93	Asgrow	R	R	S	S
Sundance II	Pure Line	R	S	R	S
Grant	Brotherton	R	S	S	R
WSU 23	Haglund	R	R	R	S
WSU 28	Haglund	R	S	R	R
74SN5	Kraft	R	R	R	R

[1] Brotherton Seed Company, Inc., PO Box 1136, Moses Lake, WA 98837; Asgrow Seed Company, PO Box 1235, Twin Falls, ID 83303; Pure Line Seeds, Inc., Box 8866, Moscow, ID 83843; Research & Extension Unit, Washington State University, 1468 Memorial Highway, Mt Vernon, WA 98273, USA.

[2] Races 3 and 4 are considered variants of race 2 (Kraft and Haglund, 1978).

available on the market. Table 6.2 lists a number of commercial pea cultivars and public breeding lines, their sources, and their reactions to races 1, 2, 5 and 6. It is suggested that these lines can be used to determine the specific race of a wilt isolate. To isolate from an infected plant, we suggest that only above-ground tissue from the fourth or higher node be used. The stipule leaf is removed, the nodal tissue is surface disinfected in 10% household bleach, and plated on acidified potato dextrose agar or a *Fusarium* selective medium.

Control

Control consists primarily of planting resistant cultivars, and avoiding severely infested fields and planting peas back in the same field more frequently than 1 in 5 years. Seed transmission can occur with race 2 because infection usually occurs during maturity. Seed transmission with races 1, 5 and 6 would primarily occur as external plant debris or in soil particles because these pathogens commonly attack and kill susceptible peas before anthesis.

WHITE MOULD

Causal Pathogen

White mould is caused by *Sclerotinia sclerotiorum* (Lib.) de Bary which is classified as an *Ascomycete* in the order *Helotiales* because this pathogen produces sclerotia. The pathogen also causes stem rots of soyabean (see Sinclair, Chapter 3, this volume) and faba bean (see Jellis *et al.*, Chapter 7, this volume). Figure 6.5 illustrates the production of apothecia by sclerotia. Sclerotia vary in size from 2.5 to 6 mm and are the principle means of survival from one crop to the next. They germinate to form mycelium, which can invade seedling roots causing a damping-off, or they can produce ascospores which are the primary means of foliar infection. Ascospores can be dispersed up to several miles by wind and remain viable to infect a susceptible crop. This pathogen has an exceedingly wide host range.

Biology

White mould can be serious where humidity is high, and is usually associated with the formation of a dense canopy which restricts air movement on the soil surface. This disease has been reported to occur in Ireland, England, The Netherlands, Brazil, Argentina, Morocco, New Zealand, the USA and Canada (Hagedorn, 1984). Symptoms usually do not appear until the canopy has completely covered the soil surface. Fluffy, white mycelium develops and later dense mycelial mats form at the soil surface on prostrate vines, pods and leaves. (Plate 12). Affected areas become slimy and dark, and sclerotia (irregular in size and shape), form within the mycelial mats.

White mould is initiated from direct germination of sclerotia in soil and from

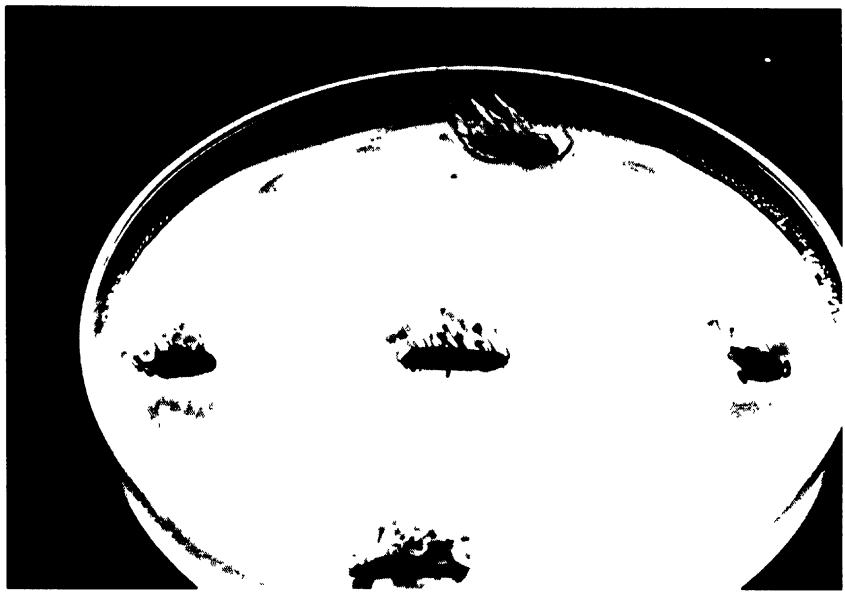

Fig. 6.5. Apothecial formation on sclerotia produced by *Sclerotinia sclerotiorum* (Photo: courtesy of J.M. Kraft).

ascospores released from apothecia which form on the sclerotia (Fig. 6.5). Direct penetration of the cotyledonary attachment area is thought to occur from sclerotial germination. However, infection via ascospore discharge, germination and penetration, during periods of cloudy weather and high moisture, is more common. Ascospores can originate in the same field or be carried by air currents from neighbouring fields. Free water or dew is necessary for infection as is a nutrient source such as senescent flower petals or other decaying tissues. Once infection occurs, incubation will only take a few days and symptoms can be severe within 2 days (Hagedorn, 1984).

Control

To control white mould, sclerotia should be deep-ploughed after harvesting a crop infested with the fungus. Crops such as potatoes, *Brassica* species, and beans should be avoided preceding the pea crop. Residual N should not exceed 22–44 kg ha^{-1} to avoid lush vine growth. In addition, in fields which have had infestations of *S. sclerotinia*, seeding rates should be reduced and cultivars with an open vine habit, such as semi-leafless types, should be planted. There are no resistant cultivars available nor has germplasm been developed that is resistant or tolerant to this disease (see Sinclair, Chapter 3, this volume). Some progress has been made in breeding for resistance to *S. rolfsii* in faba bean (see Jellis *et al.*, Chapter 7, this volume). Some recommendations for applications of fungicides have been

made. However, it is difficult to penetrate to the soil line through the canopy with effective chemicals to arrest the spread of white mould, once it is evident.

POWDERY MILDEW

Causal Pathogen

The pathogen which causes powdery mildew is *Erysiphe pisi* Syd. (syn. *E. polygoni* DC.) and is an obligate parasite found on *Pisum, Medicago, Vicia, Lupinus* and *Lens* spp. Powdery mildew is also an important disease of pigeonpea (see Reddy *et al.*, Chapter 10, this volume). The haustoria of *E. pisi* lie outside the host cell cytoplasm, and special structures form between the haustoria and cytoplasm. Cleistothecia are dark coloured, up to 180 μm in diameter, with flexuous, unbranched appendages. Each cleistothecium contains two to eight ascospores. Ovate, hyaline conidia are formed successively in loosely connected chains from the mycelium on the plant surface. These conidia can germinate and penetrate a plant surface at variable and rather low humidities (Hagedorn, 1984).

Biology

Powdery mildew is found wherever peas are grown. The disease is most serious when days are warm and dry, and nights are sufficiently cool for dew formation (Hagedorn, 1984). The disease can also be devastating where peas are grown in late season or in low, wet areas with high soil moisture, which allows the plant to remain in a vegetative state. The disease is least serious where there is high rainfall or sprinkler irrigation. In areas where the weather becomes warm too quickly, powdery mildew usually does not become severe before plant senescence.

Symptoms include white, off-coloured spots on the upper surface of the lowest and oldest leaves. These spots then increase in size and appear as white, powdery areas. Figure 6.6 illustrates the symptoms on the upper surface of leaves of a susceptible plant. The disease can progress in susceptible cultivars until the entire plant is covered with white, powdery mycelial growth. Tissue beneath infected areas may turn purplish in colour and cleistothecia form in the mature lesions. Severe infection results in early crop senescence and reduced quality, as well as decreased green pea and seed yields. The pathogen overwinters on infected plant debris, on alternative hosts and is seedborne (Hagedorn, 1984).

Control

Resistance to powdery mildew is readily available in commercial cultivars and this resistance has been stable for at least 30 years. Currently, there is some controversy as to whether there are one or two genes which govern resistance to powdery mildew. It is thought that the single recessive gene, *er*, will not suffice under conditions of extreme pathogen pressure (Hagedorn, 1984). In that case, resis-

Fig. 6.6. Lesions on upper surface of pea leaflets caused by the powdery mildew pathogen, *Erysiphe pisi* (Photo: courtesy of J.M. Kraft).

tance conferred by a second gene, *er2*, is needed in combination with *er*. In areas where the disease frequently occurs and resistant cultivars are not readily available, early maturing cultivars plus chemical sprays are the most viable control option. Chemical control includes spraying powdery mildew infested fields with flowable, elemental sulphur at a rate of 3–4 kg ha^{-1}. Other control measures include crop rotation and immediate ploughing under of the infested crop debris.

DOWNY MILDEW

Causal Pathogen

Downy mildew is caused by *Peronospora viciae* (Berk.) de Bary which belongs to the family *Peronosporaceae*. Members of this group produce sporangiophores which are dichotomously branched at acute angles and taper to curved, pointed tips where sporangia are produced. The sporangia germinate directly to form a germtube. Oospores are produced within senescent, infected pea tissue. Oospores are spherical, light brown to yellowish-pink, and are 25–37 μm in diameter.

Biology

In contrast to powdery mildew, downy mildew is most prevalent under cool moist growing conditions. In areas where green or processing peas are grown, downy

mildew is often present in the early part of the season. The disease has been reported in the United Kingdom, Sweden, Norway, Australia and New Zealand (Dixon, 1981).

The downy mildew pathogen can be either systemic or localized on leaves and/or pods. Systematically infected plants are stunted and usually distorted, and *P. viciae* sporulates readily on the plant surface. Figure 6.7 illustrates downy mildew symptoms on a systemically infected plant. The plant usually dies before flowering. Late infection is usually restricted to the plant apex. On the upper side of the foliage, white, yellow, and/or brown lesions with diffuse margins appear. On the underside of the leaves, opposite the discoloured areas, grey-brown patches of mycelia and fruiting bodies appear (Fig. 6.8). Flowers and tendrils can also exhibit symptoms and sporulation. There appears to be a positive correlation between semi-leafless foliage peas, phenotypes with restricted stipule leaves, and increased susceptibility to downy mildew (Matthews, 1981).

The pathogen overwinters in the soil and on plant debris, and from this source, systemic and local infections develop. Soilborne oospores are a primary source of potential systemic infection of developing pea seedlings and can survive for 10–15 years in soil. Pod infection can occur especially during periods of high relative humidity and can occur without foliage symptoms. Infected pods are deformed and a mycelial mass can occur on the pod wall. Oospores can form in this mycelial mass and are recorded to occur within the seed coats as well.

Fig. 6.7. Downy mildew symptoms on a systemically infected plant (Photo: courtesy of J.M. Kraft).

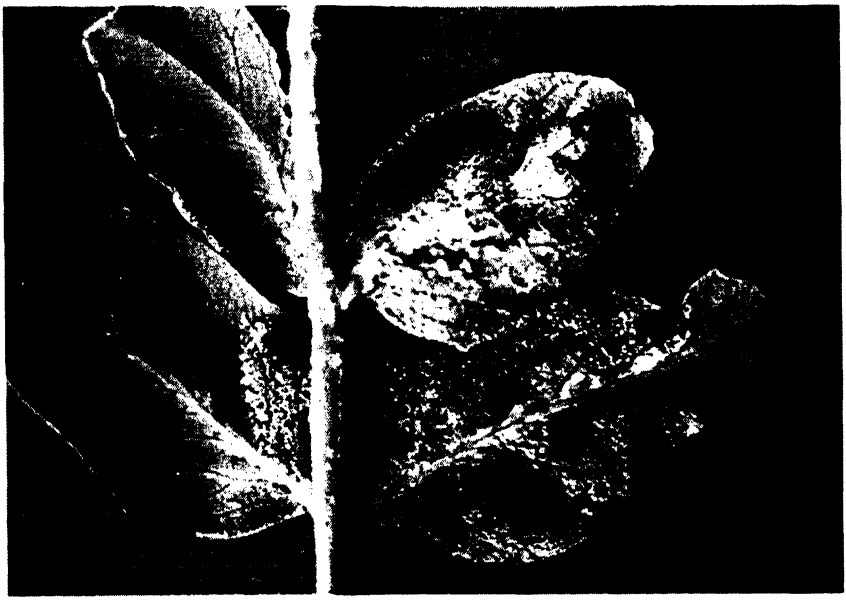

Fig. 6.8. Grey-brown lesions on underside of pea leaflets caused by the downy mildew pathogen, *Peronospora viciae* (Photo: courtesy of D. Inglis).

However, no successful case of seed transmission of downy mildew has been recorded.

Control

To control downy mildew, all crop debris containing oospores should be removed or deep ploughed. Acylalanine fungicides (such as metalaxyl) are effective seed treatment chemicals that reduce or eliminate the primary systemic infection phase of downy mildew. The practice of not growing peas more than once every 5 years in the same field, deep ploughing crop debris, use of an appropriate seed treatment, and a resistant or tolerant cultivar will reduce the incidence and severity of downy mildew in any given year.

Several sources of resistance to downy mildew are known (Ali *et al.*, 1994). As many as six physiologic races of downy mildew occur in northern Europe (Stegmark, 1990) and race-specific resistance has been reported for several cultivars (Hubbeling, 1975). Unfortunately, there are no pea genotypes with complete resistance to all known pathotypes (Stegmark, 1988). Stegmark stated that the cultivar Dark Skin Perfection is more resistant to downy mildew than other cultivars used for canning and freezing in Europe. Unfortunately, Dark Skin Perfection can be affected severely enough to cause significant losses in yield and quality when conditions are optimum for severe downy mildew development (Stegmark, 1988). In Sweden, such cultivars as Starcovert, Gastro, Cobri and

Puget, previously reported as resistant, were susceptible to the prescence of strains of *P. viciae*. Stegmark further stated that some breeding lines exhibited low susceptibility to all known isolates but no complete resistance to any isolate. These lines should be good prospects for developing stable resistance to *P. viciae*.

ASCOCHYTA BLIGHT

Causal Pathogen

Ascochyta blight is a disease complex caused by three separate pathogens: *Ascochyta pisi* Lib. which causes a leaf, stem and pod spot; *Phoma medicaginis* Malbr. & Roum. var. *pinodella* (Jones) Boerema which causes leaf spots, stem lesions and root rot; and *Mycosphaerella pinodes* (Berk. & Blox) Vestergr. which causes a leaf, stem and pod spot, and root rot (Hagedorn, 1984). Ascochyta blight is a common and serious disease of legumes and has been reviewed in Chapters 4, 5, 7, 8 and 9, this volume. Under field conditions, it is not easy to distinguish among the three pathogens and it is practical to consider the three as causing a single disease. *Mycosphaerella pinodes* is considered the most aggressive and causes the most economic loss. The three pathogens can be distinguished by the presence or absence of chlamydospores and the presence or absence of a perfect stage. Such characters as size and shape of conidia are not predictable and are not recommended to be used in distinguishing between pathogens. *Mycosphaerella pinodes* is the perfect stage of *Ascochyta pinodes*. When embedded in pea tissue it produces perithecia which are dark brown, globose, with papillate ostioles 90 × 180 μm in diameter (Hagedorn, 1984). Ascospores are hyaline, two-celled, and average 7.9 × 17.2 μm. Pycnidia are freely formed as are chlamydospores. Conidia (pycnididospores) are hyaline and ellipsoid and most are one-septate. *M. pinodes* is homothallic.

Ascochyta pisi does not produce a perithecial or perfect stage nor does it produce chlamydospores. *Phoma medicaginis* var. *pinodella* does not produce a perfect stage but does produce chlamydospores.

Biology

Infection from all three pathogens can develop on seedlings during cool, wet weather, resulting in blackening and death, beginning at the soil line and extending up the stem for 4–12 cm. Leaf infection by *M. pinodes* results in small purple to black spots (Fig. 6.9). Under dry growing conditions, these lesions remain small but with moist conditions the lesions enlarge turning from brown to black. Affected leaves can be killed. Stem lesions caused by *M. pinodes* enlarge and often coalesce to girdle stems completely giving the entire lower plant a blue-black appearance. In contrast to *M. pinodes* and *P. medicaginis* var. *pinodella*, *A. pisi* seldom attacks the plant base and does not cause a root rot.

All three pathogens can be seed-transmitted and spread in pea trash. Chlamydospores are produced by *M. pinodes* and *P. medicaginis* var. *pinodella*

Fig. 6.9. Leaf infection and small purple to black spots caused by *Mycosphaerella pinodes* (Photo: courtesy of J.M. Kraft).

which allow for soil survival. Only *M. pinodes* produces a perfect stage in culture. In fields previously cropped to peas, *M. pinodes* and *P. medicaginis* var. *pinodella* can overwinter as mycelium on infected pea debris or in soil as chlamydospores. When peas are grown in fields recently cropped to peas, either of these pathogens can cause severe root rot and can spread to adjacent plants. *Ascochyta pisi* survives poorly in soil but can sometimes overwinter on volunteer plants and pea stubble.

Moist conditions are necessary for infection and plant-to-plant spread of all three pathogens. Plants can be attacked at any age but mature leaves are most susceptible. The production of seed in drier growing areas and the use of seed treatment chemicals have essentially eliminated *A. pisi* as an important pathogen of peas. However, the overwintering abilities of *M. pinodes* and *P. medicaginis* var. *pinodella* via chlamydospores are the reason they are more difficult to control. Under field conditions, with and without fungicide sprays, there was a 5–6% loss in yield for every 10% of stem area affected by *M. pinodes*. *Mycosphaerella* was found to be most severe on early-maturing cultivars (Bretag, 1991).

Control

Control of ascochyta blight begins with growing seed crops in dry areas and avoiding the harvest of seed from bypassed production fields. Seed treatment chemicals effectively reduce seedborne *Ascochyta* to manageable levels. Pea refuse should be disked and ploughed under immediately after harvest before these pathogens can be dispersed by wind and rain.

No single gene or major gene resistance to *M. pinodes* has been found despite extensive searches in the available pea gene pool. It is our opinion that minor genes need to be compiled to develop horizontal resistance or tolerance to *M. pinodes* utilizing a recurrent selection programme and severe disease pressure.

BACTERIAL BLIGHT

Causal Pathogen

Pea bacterial blight is caused by *Pseudomonas syringae* pv. *pisi* (Sackett) Young, Dye & Wilkie, which is a one-celled, aerobic, Gram-negative, non-spore-forming rod with polar flagella. Colonies on King's medium B are fluorescent. Average rod measurements are 0.7 × 2–3 μm. Six races of *P. syringae* pv. *pisi* have been described based on reactions to a set of differential pea cultivars (Taylor *et al.*, 1989). Race 2 was found to be the most common and race 6 was the most virulent on all cultivars tested.

Biology

Bacterial blight is a seedborne disease which has been reported in most pea growing areas of the world (Hagedorn, 1984). Symptoms of the disease can occur any time during the growing season, especially following heavy rain, after hail damage, or when the crop is grown under overhead sprinkler systems. Symptoms usually develop initially at the nodal area and on stipule leaflets. Lesions often develop first on the underside of leaves as water-soaked lesions that appear dark green or brown on the upper surface. Older lesions tend to have light brown centres and dark borders. Pods may become severely infected with lesions that are circular, water-soaked and sunken. Seed infection is via pod infection. In seed production fields in arid areas, infection with bacterial blight can go unnoticed. The lower leaf nodes can become infected but the disease does not progress if conditions are not optimum. The debris from these infected leaves can become a source of infection on seed during harvest. Bacterial blight can be disseminated from one seed lot to another during threshing and milling, even when fungicides are applied. Farm machinery can also spread the pathogen if not cleaned between seed lots.

Control

Planting clean seed and using resistant cultivars are the primary means of control. Seed should be produced in arid areas, not under sprinkler irrigation, and seed fields should be inspected for the presence of blight. Sodium hypochlorite as a 1% concentration can reduce seed infection by 85 to 90%. In addition, a streptomycin slurry can also be used but recommendation to use an antibiotic is questionable both in terms of effectiveness and economics (Hagedorn, 1984). Resistance to races 1 to 5 was found to be widespread in available cultivars (Taylor *et al.*, 1989).

ALFALFA MOSAIC

Causal Pathogen

Evidence has shown that the alfalfa mosaic virus (AlMV) genome is composed of three bacilliform and one spherical-shaped particle. However, as many as six nucleoprotein components have been reported (Jaspers and Bos, 1980). Infectious virus consists of a tripartite RNA genome (RNAs 1, 2 and 3) and a subgenomic RNA4, which contains a repeat of the coat protein gene located on RNA3. Genomic RNAs 1, 2 and 3, and the presence of some coat protein or coat protein mRNA are required for infection (Bol and Jaspers, 1994). Graaff *et al.* (1995) demonstrated that while the protein coat is required for infection, it is not required for assembly and targeting of the viral polymerase. Frequently, only minor changes in the nucleotide sequence of the coat protein gene can drastically alter symptom expression. Nelleman *et al.* (1991) reported that nucleotide changes resulting in a single amino acid mutation in the virus coat protein can affect symptom expression in the host plant.

Biology

Alfalfa mosaic virus (AlMV) is probably the most geographically widespread virus in the world. The virus has a natural host range of approximately 150 plant species. When combined with the experimental host range, the list of susceptible plants is increased to over 600 plant species representing 70 families (Bol and Jaspers, 1994) and includes *Trifolium* spp. (see Mercer, Chapter 12, this volume). An immense population of AlMV strains exists, varying in both pathogenicity and virulence in any geographical area. Symptoms on pea include chlorosis, purple or brown necrotic streaks in leaves and the vascular system along the stem, and stunting of the plant (Plate 13). Ford and Baggett (1965a) reported that AlMV consistently caused severe plant stunting in all pea cultivars tested. Curling and brown necrotic lesions frequently occur on leaves of plants. Leaves of infected plants are often brittle, presumably due to carbohydrate accumulations. Symptoms on pods include malformation, discoloration, and the occurrence of

necrotic lesions (Zaumeyer, 1938). Identification of pea plants infected with AlMV can be difficult, as the necrotic streaking of the vascular system is often identical to streaks produced by pea streak carlavirus infection (Ford and Baggett, 1965b).

AlMV is transmitted in a non-persistent manner by at least 14 different aphid species (Crill *et al.*, 1970b). The pea aphid (*Acyrthosiphon pisum* Harris) and the green peach aphid (*Myzus persicae* Sulzer) are considered the most important vectors of AlMV in peas. Both are distributed worldwide. Other aphid species which should also be considered as vectors of AlMV in pea crops are the foxglove aphid (*Aulacorthum solani* Kaltenbach), the bean aphid (*Aphis fabae* Scopoli), and the potato aphid (*Macrosiphon euphorbiae* Thomas). The wide range of naturally occurring annual and perennial hosts provides abundant inoculum sources for AlMV spread by aphids. Figure 6.10 illustrates the build-up of pea aphids on a pea plant. However, the main source of AlMV worldwide is generally considered to be alfalfa. Two-year-old alfalfa fields can have infection levels of 80 to 90% (Crill *et al.*, 1970a; Rahman and Peaden, 1993), so that AlMV inoculum can be abundant when peas are grown near infected alfalfa fields.

Control

Knowledge of inheritance traits for resistance to AlMV in peas is lacking. Numerous USDA Plant Introduction (PI) accessions have been identified (Table 6.3), but no commercial cultivars resistant to the virus are currently available. Ford and Baggett (1965b) reported 31 of 900 pea lines tested were resistant against a single isolate of AlMV from white clover (*Trifolium repens*). In contrast, Hagedorn (1968) tested 397 accessions against an isolate from red clover and found none of the PI lines immune to AlMV. Most of the lines reported as resistant to AlMV by Ford and Baggett (1965b) were tested again by Hagedorn and found to be susceptible to the virus isolate from red clover. Further studies with the PI accessions are currently needed, using several virus strains from various regions and sources, in order to identify lines either with high levels of tolerance or with true resistance.

An effective control to limit the incidence of AlMV is to avoid planting peas near alfalfa fields older than 3 years. There are no known commercial pea or alfalfa cultivars with resistance. Aphid populations may be reduced by application of registered insecticides. While it is unlikely that control of aphid populations will prevent virus infection, the use of aphicides can significantly reduce plant-to-plant spread in the field.

LEAF ROLL

Causal Pathogen

Bean leaf roll virus (BLRV) is a member of the large group of yellowing viruses, the luteoviruses. The virus consists of icosahedral particles 28 nm in diameter.

Fig. 6.10. Massive build-up of pea aphid *Acyrthosiphon pisum* on pea pods (Photo: courtesy of J.M. Kraft).

The single-stranded RNA species has a molecular weight of 2.02×10^6 under denaturing conditions (Reijnders *et al.*, 1974) consisting of approximately 57,000 nucleotides. The coat protein gene of BLRV has been sequenced and consists of a 22 kDa open reading frame (Prill *et al.*, 1990). Amino acid sequence homologies ranged from 42.6 to 56.1 when the 22 kDa protein was compared to coat proteins of barley yellow dwarf (PAV strain), beet western yellows, and potato leaf roll luteoviruses.

Biology

The virus was first described in Europe in 1954 on faba bean and pea (Quantz and Volk, 1954; Tinsley, 1959), and is also common in Iran (Kaiser, 1972), Africa and India (Ashby, 1984). BLRV had not been reported in the United States until 1980, when it occurred in epidemic proportions and caused severe losses in Washington and Idaho (Hampton, 1983). BLRV is sometimes referred to as 'top

Table 6.3. Pea germplasm accessions, breeding lines, and cultivars with resistance or tolerance to at least one isolate of alfalfa mosaic virus (AlMV), bean leaf roll virus (BLRV), pea enation mosaic virus (PEMV), pea seedborne mosaic virus (PSbMV), pea streak virus (PSV), and red clover vein mosaic virus (RCVMV). Accessions and breeding lines listed are white-flowered, intermediate to tall in stature, and may be readily used for crossing purposes.

AlMV	BLRV	PEMV	PSbMV	PSV	RCVMV
PI 121977	Abador	Aurora	B 442-15	OSU B442-15	OSU B442-15 [b]
PI 164148	Alderman	Commando	B 442-66	OSU B445-66	OSU B445-66
PI 166129	Almota	Freezer 50	OSU 547-29	OSU 644-2	OSU 663 [d]
PI 180701	Centurion	Freezer 52	OSU 559-6	OSU 663 [d]	OSU 668 [d]
PI 184131	Champ	Freezer 6650	OSU 564-3	OSU 668 [d]	OSU 677 [b,d]
PI 193838	Climax	Freezer 60	OSU 584-16	OSU 677 [b,d]	PI 116056
PI 197044	Cobri	H 286-1-1	OSU 589-12	OSU 709-4 [c]	PI 194339
PI 197449	Coquette	H 294-5-1-1	OSU 615-15	PI 140297	PI 195026
PI 197988	Elf	H 312-2-3	OSU 620-1	PI 195405	
PI 197989	Frisky	H 543-3-1-11	OSU 663 [d]	PI 203066	
PI 201391	Jubilee	H 890-3-2	OSU 668 [d]	PI 212029	
PI 210684	Juwel	Maestro	OSU 677 [d]		
PI 244116	OSU 559-6	New Era	VR74-410-2		
Thomas Laxton	OSU 564-3 [a]	Novella II	VR74-1492-1		
OSU 33	OSU 584-16 [a]	Olympia	WI 7105		
OSU 176-2	OSU 589-12 [a]	OSU 547-29	WI 7106		
OSU 709-4 [c]	Perfected 400	OSU 559-6	X 78006		
	Rika	OSU 564-3 [a]	X 78122		
	Sparkle	OSU 584-16 [a]	X 78123		
	Splendor	OSU 589-12 [a]	X 78124		
	Superlaska	OSU 615-15	X 78125		
	Surpass	OSU 620-1	X 78126		
	Telephone	OSU B442-15 [b]	X 78127		
	Wando	OSU 445-66 [b]	X 78128		
		OSU 663 [d]	PI 193586		
		OSU 668 [d]	PI 193835		
		OSU 677 [b,d]	PI 347328		
		OSU 33	PI 347494		
		OSU G113	PI 378158		
		OSU 225			
		OSU 709-4 [c]			
		PI 140295			
		Shoshone			
		Surprise 60			
		Tempter			
		Trident			

Sources: AlMV: Ford and Baggett (1965 a,b).
BLRV: Drijfhout (1968); Hagedorn (1974, 1984).
PEMV: Schroeder and Barton (1958); Hagedorn and Hampton (1975); Baggett and Hampton (1977); Baggett and Hampton (1983); Baggett (1984); Baggett and Kean (1988); Baggett *et al.* (1994);
PSbMV: Hagedorn and Gritton (1971); Hagedorn (1974); Baggett and Hampton (1977); Kraft and Giles (1978); Hampton and Braverman (1979); Muehlbauer (1983); Baggett and Kean (1988); Provvidenti and Alconero (1988c); Baggett *et al.* (1994).
PSV: Ford and Baggett (1965 a,b); Hagedorn (1968); Baggett and Hampton (1977); Baggett *et al.* (1994).
RCVMV: Hagedorn (1968); Baggett and Hampton (1977); Baggett *et al.* (1994).

Breeding lines with resistance to more than one virus are denoted as follows:
BLRV + PEMV; [b] PSV + RCVMV; [c] AlMV + PEMV + PSV; [d] PEMV + PSV + RCVMV + PSbMV

yellows' in pea crops. However, other diseases or agronomic conditions can produce similar symptoms. Originally described as pea leaf roll, bean leaf roll is now the commonly accepted term because of the typical leaf rolling symptoms produced in *Vicia faba* (see Jellis *et al.*, Chapter 7, this volume). BLRV is restricted to a small group of plants in the legume family, and most are of important economic significance. Economic food crop hosts include pea, faba bean, chickpea, lentil and common bean. The aetiology of the virus on chickpea is complex (see Haware, Chapter 9, this volume). BLRV is also widespread in alfalfa and in several clover species which serve as important alternative hosts. It is believed that alfalfa was the primary reservoir host responsible for the epidemic outbreaks of BLRV in peas and other cool-season food legume crops in the USA in 1980 (Hampton, 1983), 1987 (Kraft and Kaiser, 1993), and 1990 (Klein *et al.*, 1991).

Many aphid species have been reported to transmit BLRV in a persistent manner (Ashby, 1984; Edwardson and Christie, 1991). The pea aphid, however, is considered the most important vector. Aphids can acquire the virus within 2 h (Thottappilly *et al.*, 1977), but the latent period of BLRV in the aphid vector is 16 to 20 h. Consistent with other phloem-limited viruses, Kaiser (1972) found that BLRV was not transmissible by seed and the virus is not transmitted mechanically. BLRV shares close serological relationships with other luteoviruses including legume yellows (Duffus, 1979) and beet western yellows (Waterhouse *et al.*, 1988). Other viruses which share serological relationships with BLRV include soyabean dwarf virus, subterranean clover redleaf virus, and potato leaf roll virus. No specific strains of BLRV have been clearly identified. Baggett and Hampton (1991) examined the host response to 11 BLRV isolates collected from Idaho on a wide range of pea genotypes and could detect no differences in virus strains or pathotypes.

Control

Resistance in pea to BLRV is inherited as a single recessive gene designated *lr* (Drijfhout, 1968). Crampton and Watts (1968) described resistance to BLRV as an additive system of inheritance. Baggett and Hampton (1991) observed the operation of *lrv*, a single recessive tolerance gene described on the basis of symptom expression in selected pea cultivars. The relationship between the *lrv* gene and the *lr* gene reported by Drijfhout is not known. Several PI accessions possess resistance and many commercial varieties are now available with resistance to BLRV (Table 6.3). In addition, this resistance has not shown any evidence of being overcome by the virus.

PEA ENATION MOSAIC

Causal Pathogen

Pea enation mosaic virus (PEMV) is the only member in its group and shares no known serological relationship with any other plant virus. The infectious

genome is packaged in two isometric spherical nucleoprotein particles c. 25 and 28 nm in diameter. The coat protein molecular weight is 21 kDa. The genome is composed of two ssRNA species consisting of 5706 nucleotides (RNA1) and 4253 nucleotides (RNA2) (Demler and de Zoeten, 1994). A third small RNA (RNA3) is occasionally observed and is considered to be satellite RNA. RNA1 has been shown to share close organizational and sequence homology with several members of the luteovirus group. Its aphid transmissibility has been linked to the presence of a 54 kDa protein. Demler and de Zoeten have also shown that the 54 kDa minor protein is lost after repeated mechanical transmission and resultant loss of transmissibility by the aphid vector.

Biology

Pea enation mosaic is an important virus disease not only in pea but other economically important legumes. Pea enation mosaic virus has been responsible for severe losses in food legumes in the Pacific Northwest region of the USA during 1983, 1987, and 1990 (Klein *et al.*, 1991; Kraft and Kaiser, 1993). PEMV was first identified by Osborn (1935) on faba bean in New York State. The virus has also been reported in Europe (Cockbain and Gibbs, 1973), Iran, and Sicily (Peters, 1982). The host range is narrow and restricted primarily to *Leguminosae*, although *N. clevelandii* and *Chenopodium album* are useful diagnostic non-legume hosts (Hagedorn *et al.*, 1964). Alfalfa (lucerne) has been considered to be the primary perennial host and inoculum source for PEMV (McWhorter and Cook, 1958). However, Hagedorn *et al.* (1964) and Cockbain and Gibbs (1973) later attempted to infect alfalfa with PEMV, and were unable to recover the virus from this host. Recent work by Larsen *et al.* (1996a) has shown that PEMV could not be detected by enzyme-linked immunofluworescence assay (ELISA) or dot blot hybridization in any of 3230 alfalfa samples collected in Washington State between 1988 and 1994. These findings further suggest that alfalfa is not a host of PEMV.

Mechanically inoculated peas display symptoms in 5–10 days, depending on cultivar, environmental conditions, and plant maturity. Diagnostic symptoms in pea include translucent flecks or 'windows', together with vein-clearing and malformation in leaves and stipules (Fig. 6.11). Plants are usually severely stunted and distorted. Many cultivars undergo gross cytopathological changes, including small growths or enations on the undersides of leaves as a result of the infection. Pods are typically deformed severely and produce characteristic outgrowths or proliferations on the pod surface. The virus causes death of plants in susceptible cultivars or when plants are infected at an early stage.

The virus is transmitted in a persistent manner by eight aphid species, with the pea aphid (*Acyrthosiphon pisum*) considered to be the most significant vector (Demler and de Zoeten, 1994). Several biotypes of the pea aphid exist which transmit the virus with varying efficiencies. Pea aphid nymphs, given an acquisition access period of 3 h, had a latent period of 10 h (Toros *et al.*, 1978). Following the latent period, the aphid was able to transmit the virus during brief probes into the cytoplasmic tissue. PEMV is also mechanically transmissible but

Fig. 6.11. Typical symptoms of pea enation mosaic showing translucent windows, vein-clearing, malformed pods and leaves (Photo: courtesy of J.M. Kraft).

aphid transmissibility is usually lost after several serial mechanical transfers. Only a single biological strain of PEMV exists as defined by host range and symptom expression. Different geographical isolates exist which may vary slightly in biological properties, the most significant difference being aphid specificity. Bath and Tsai (1969) were able to separate a New York isolate from a California isolate of PEMV using the pea aphid and comparing latent periods, inoculation retention times, and acquisition and inoculation access periods. Symptomatology and virus physical properties of the two isolates could not be differentiated.

Control

Control of the virus can be greatly enhanced by elimination of aphid populations and reservoir hosts. Control of virus movement is often difficult when peas are grown near clover fields because of restrictions in pesticide application on this host.

Resistance in pea to PEMV is primarily conferred by a single dominant gene, *En*, first found in PI 140295 from Iran (Schroeder and Barton, 1958). No complete resistance to PEMV is yet available in pea, although many cultivars or PI accessions exhibit varying levels of tolerance (Table 6.3). Cultivars expressing tolerance generally exhibit mild symptoms when infected with PEMV, or 'recover' after initial infection (Baggett and Hampton, 1983). Plants then usually progress

to normal production of peas. The knowledge accumulated to date on the genomic organization, including the complete nucleotide sequence information (Demler and de Zoeten, 1994), may provide opportunities for development of transgenic resistance to this virus in future pea cultivars.

PEA SEEDBORNE MOSAIC

Causal Pathogen

Particle lengths of pea seedborne mosaic potyvirus (PSbMV) have been reported in Czechoslovakia measuring from 700 nm (Musil, 1970) to 750 nm (Inouye, 1967), and 770 nm by Bos (1970). Hampton *et al.* (1974) found that particle lengths varied by fixative treatment, and whether the preparations were from leaf dips or purified preparations. Leaf-dip preparations fixed in gluteraldehyde were 700 nm in length, while particles from purified preparations fixed in gluteraldehyde measured 750–770 nm. Coat protein molecular weight is reported to be 34 kDa (Hampton and Mink, 1975). The entire virus genome has been sequenced. It consists of a positive-sense ssRNA of 9924 nucleotides in length containing a 9618 base open-reading frame which codes for a 364 kDa polypeptide (Johansen *et al.*, 1991).

Biology

PSbMV was first discovered in Europe (Musil, 1966) and, at about the same time, it was reported in Japan by Inouye (1967), who described the virus using the name by which it is now commonly accepted. The virus was reported shortly after in the United States (Mink *et al.*, 1969; Stevenson and Hagedorn, 1969), and described by Hampton (1969) as 'pea fizzle top virus'. It was not until 1980 that PSbMV was found in the southern hemisphere in New Zealand (Fry and Young, 1980), then in England (Matthews *et al.*, 1981). Largely because of the potentially high levels of seed transmission, the ease of dissemination through international exchange of breeding lines, and the numerous species of aphid that can transmit PSbMV, this virus continues to be an economically important disease of pea (Mink *et al.*, 1969; Stevenson and Hagedorn, 1971; Hampton *et al.*, 1976; Hampton and Braverman, 1979; Kraft and Hampton, 1980; Fletcher, 1993). It is also an important virus disease of faba bean (see Jellis *et al.*, Chapter 7, this volume). The virus is seedborne in pea, lentil and faba bean, but it is not known to be seedborne in chickpea (W.J. Kaiser, Washington, 1996, personal communication). Pea seed with physical cracks in the seed coat transmitted PSbMV at 33%, compared to 4% in seed with normal intact seed coat (Stevenson and Hagedorn, 1970). However, seed cracking was not a reliable diagnostic tool for PSbMV.

Symptoms in pea vary greatly with cultivar, temperature, and environmental conditions as well as virus strain or pathotype. Common symptoms include epinasty or downward leaf rolling, mild chlorosis, vein-clearing, mosaic, and a

general stunting of the plant. Symptoms are often most severe on plants emerging from infected seed. Terminal rosetting, a result of the reduction in internodal growth, is common and pods may be deformed or fail to set. Plants grown in the field frequently may display fewer obvious symptoms than those which are grown in the greenhouse or in growth chambers (Hampton *et al.*, 1976). Mid-season pea cultivars typically display more severe rosetting symptoms than the early cultivars (Hampton and Baggett, 1970).

The host range of PSbMV includes 47 plant species in 12 families, only a few of which are considered highly susceptible (Aapola *et al.*, 1974). Other hosts have since been reported, including chickpea (Alconero *et al.*, 1986). Because *Phaseolus* spp. are non-host members of the legume family, they are important to include in biological assays for the presence or absence of PSbMV. Important diagnostic indicator plants include faba bean, *Chenopodium quinoa* and *C. amaranticolor* (Hampton and Mink, 1975). In the original work by Aapola *et al.* (1974), alfalfa was reported as a host of the virus in aphid transmission tests. However, there has been no recent evidence to support their findings and it is now generally considered a non-host (R.C. Larsen, 1996, unpublished results).

PSbMV exists as several strains or pathovars. Hampton *et al.* (1981) reported that seven isolates could be distinguished on pea germplasm differentials. The isolates could be placed into one of five groups based on host response in selected pea lines. All strains were closely related in serological tests. Furthermore, all test isolates from the USA and Japan were considered to be closely related serologically. Two isolates from pea (P-1 and P-4) and one from lentil (L) were described by Alconero *et al.* (1986). The isolate from lentil, which only infected pea cultivars also susceptible to bean yellow mosaic potyvirus, produced much more severe symptoms in pea than those of the other two pea isolates.

PSbMV is transmitted by at least 21 different aphid species on a worldwide basis (Khetarpal and Maury, 1987). *Acyrthosiphon pisum*, *Myzus persicae*, and *Aphis craccivora* Koch are likely to be the three most common aphid species which transmit the virus in a non-persistent manner. However, the potato aphid (*Macrosiphon euphorbiae* Thom.) is of concern wherever potatoes are grown near pea fields. Aphids can typically acquire the virus within 5 min. Gonsalez and Hagedorn (1971) reported that 7–10% of test aphids transmitted PSbMV after a single acquisition probe. They also found that *M. euphorbiae* was significantly more efficient than either *M. persicae* or *A. pisum* in transmission of PSbMV.

Control

Many pea germplasm accessions currently available are resistant to PSbMV (Table 6.3). Resistance was first characterized as a single factor recessive *sbm* gene (Hagedorn and Gritton, 1973). Gritton and Hagedorn (1975) later identified *sbm-1* to confer resistance against the P-1 pathotype and to be linked with the gene *wlo* on chromosome 6 of peas. Recessive genes *sbm-2* and *sbm-3* confer resistance to the L and related L-1 pathotype (Provvidenti and Alconero, 1988a). Gene *sbm-2* is linked to *mo* on chromosome 2. A fourth gene, *sbm-4*, is monogenic recessive and confers resistance to the P-4 pathotype (Provvidenti and

Alconero, 1988b). The location of *sbm-1* on chromosome 6 has been identified using molecular markers (Timmerman *et al.*, 1993). This information is useful for rapid identification of resistant progeny as early as the F_2 generation without inoculation of test plants with PSbMV.

The most efficient control of PSbMV is to grow resistant cultivars, when available. Also, care should be taken by plant breeders and commercial growers to ensure that seed lots are relatively free of PSbMV. Seed lots are now routinely tested for the presence of PSbMV. In addition, as for other viruses in the potyvirus group, aphid control is extremely important in limiting plant-to-plant spread.

PEA STREAK

Causal Pathogen

The pea streak carlaviruses (PeSV) consist of slightly flexuous, rod-shaped particles *c.* 619–653 nm in length. Examination of purified virions of the PeSV-Walla Walla strain, however, revealed three distinct particle sizes of 640 nm, 140 nm, and 95 nm in length (Larsen *et al.*, 1993). Veerisetty and Brakke (1977) reported that PSV and alfalfa latent virus (ALV) were two distinct viruses based on coat protein molecular weight and comparative sizes of their RNAs. Hampton (1981) later suggested, however, that both ALV and PSV comprised a single particle length of 630 nm, were serologically indistinct in ELISA tests, and could only be distinguished by minor differences in host range and symptomology. A recent comparison of the nucleic acid sequences of AlMV with the putative coat protein sequence of ALV revealed an 82% base homology (Hampton, 1981; R.C. Larsen, 1996, unpublished results). Homology was 95% when the amino acid sequence of both viral proteins were compared. The capsid protein of PeSV has a molecular weight of 28 kDa and the ssRNA resolved in glyoxal-denaturing gels as 8.1 kilobases (Larsen *et al.*, 1993).

The virus is transmitted in a non-persistent manner by the pea aphid, and alfalfa is considered the most important perennial reservoir of PeSV (Hampton and Webster, 1983). Aphid specificity apparently occurs in nature. Pea aphids transmitted the Wisconsin strain of PeSV at a low percentage rate (Kim and Hagedorn, 1959), while a western strain from Idaho could be transmitted with ease.

Biology

Pea streak carlavirus (PeSV) was first reported on peas in Virginia by Zaumeyer in 1938 and in Wisconsin pea fields by Hagedorn and Walker (1949a). The virus has been reported to occur in the USA, Canada and Germany (Bos, 1973), but is likely to have a wider geographic distribution. The host range of PeSV is limited primarily to food and forage legumes but bean (*Phaseolus vulgaris*) is not generally considered a host. Symptoms in peas are characterized by purple to brown necrotic streaks on stems and petioles, brown necrotic lesions on leaves, and wilting

of the plant. Symptoms on affected pods include brown necrotic lesions often associated with sunken areas (Plate 14). Pods fail to fill properly if plants are infected at an early age by PeSV (Hagedorn and Walker, 1949a). General leaf chlorosis can occur, but is usually not as severe as in peas infected with AlMV. Severe strains of PeSV can cause death of younger plants that become infected before flower set. Crop losses caused by PeSV can occasionally be significant in peas as well as other food legumes including chickpeas, lentils and faba beans. Several strains of PeSV exist and have been described as PeSV-Walla Walla (Larsen *et al.*, 1993), PeSV-Central Ferry (Kaiser *et al.*, 1993), and alfalfa latent virus (Veerisetty and Brakke, 1978).

Control

Control consists primarily of avoiding planting peas near alfalfa fields and close monitoring and suppression of increasing aphid populations with timely aphicide applications. No pea cultivars that are resistant to PeSV currently exist, although several PI accessions have been reported to have some levels of resistance or tolerance under field or greenhouse conditions (Table 6.2). Four Plant Introduction accessions including PI 193845, 203066, 212029, and 261677 were found to be resistant to the PSV (P-42) isolate (Ford and Baggett, 1965b). Hagedorn (1968) later reported that three additional PI accessions (116944, 140297, and 195405) were resistant to the Wisconsin PSV isolate. Baggett and Hampton (1977) reported that two Oregon State University breeding lines (OSU B442–15 and OSU B445–66) exhibited moderate to good resistance to PSV.

RED CLOVER VEIN MOSAIC

Causal Pathogen

Virus particles of red clover vein mosaic carlavirus (RCVMV) are slightly flexuous rods 645 nm in length (Varma, 1970). They are composed of a single-stranded RNA species with a length of 7.05 kilobases as determined by glyoxal-denaturing agarose gels (Larsen *et al.*, 1996b). The virus coat protein has an apparent molecular weight of 32–33.5 kDa (Veerisetty and Brakke, 1977; Larsen *et al.*, 1996b).

Biology

RCVMV was first described in red clover by Osborn in 1937. Hagedorn and Walker (1949b) later described the virus in pea as 'Wisconsin pea stunt', by which it is still often referred. Symptoms include marked vein clearing accompanied by a mosaic in pea leaves. Specific symptoms may vary with virus strains and environmental conditions. Diagnostic symptoms in field-infected plants include severe stunting and a pronounced shortening of internodes resulting in

rosetting of leaves. A lack of apical dominance and a proliferation of axillary buds frequently occurs. Pod formation is severely affected when plants are infected before flower set thus reducing yields, or plants can be killed if infected at an early stage of growth (Hagedorn, 1984). Distribution of the virus is not well documented, but the incidence is of increasing concern in the Willamette Valley of western Oregon.

RCVMV is transmitted in a non-persistent manner by the pea aphid and by several other aphid species. Aphids acquire the virus from perennial host plants including red clover, alsike clover, crimson clover (*T. incarnatum*), white sweet clover (*Melilotus alba*), and alfalfa (Hagedorn and Hanson, 1951). In the spring, aphids feed on these important virus reservoir perennial sources and move to pea fields usually as alate forms. Several strains of RCVMV have been reported. Apparently they differ only by symptom expression or minor variations in host range. A strain of RCVMV was recently isolated from chickpea, a previously undocumented host (Larsen *et al.*, 1996b). Transmission of RCVMV through seed has been reported in red clover (Varma, 1970). Sander (1959) reported that RCVMV was seed-transmitted in faba bean through six generations. It has not been reported as seedborne in alfalfa. The strain currently available from the American Type Culture Collection (RCVMV pv110) was recently reported to be seedborne in pea but at a very low rate (Larsen *et al.*, 1996b).

Control

As with other non-persistently transmitted viruses, care should be given to avoid planting peas near established clover or alfalfa fields. Also, close attention should be given to monitoring aphid populations and/or flights into the pea field.

Little information is available on resistant cultivars, PI accessions, or breeding lines. None has been identified with resistance to RCVMV, although Hagedorn (1968) reported six PI lines with varying degrees of tolerance (Table 6.3). Only a single virus isolate from red clover was used in the evaluations. None of the PI accessions with reported tolerance to RCVMV was resistant or tolerant to PSV.

PEA CYST NEMATODE

Causal Pathogen

The pea cyst nematode, *Heterodera goettingiana* Liebs., was first described as a pathogen of peas in 1892 (Jenkins and Taylor, 1967). Members of this nematode genus form a cyst at the end of their life cycle. The cyst, the oxidized cuticle of the female, forms a tough, leathery sac which can contain as many as 500 eggs. The cyst wall protects the eggs and is the survival structure. Second-stage larvae constitute the motile infective stage, possess a pointed tail, and range from 0.4 to 0.6 mm in length. Once a feeding site is found, the larvae become stationary. Mature females are white and have the same body size and shape as the mature

cysts. At maturity, the posterior portion of the female body lies outside the root with only the neck embedded. After the final moult, males assume a vermiform shape, range in length from 0.7 to 1.6 mm, and leave the root to reproduce sexually with sedentary females.

Biology

The pea cyst nematode occurs throughout many regions of Europe and the Mediterranean Basin, including Germany, The Netherlands, Great Britain, Belgium, the former USSR, Spain, Portugal, France, Italy, Israel, Algeria and Malta (Di Vito and Greco, 1986). To a limited extent, it also occurs in the USA, having been reported from greenhouse cultures in Idaho, Illinois and Pennsylvania (Thorne, 1961; Di Vito, 1991), and in commercial pea fields in western Washington (Handoo *et al.*, 1994).

Symptoms in the field occur as clearly defined patches of stunted, chlorotic plants (Fig. 6.12). Infected areas are first limited to small circular areas, but then may extend to the entire field (Di Vito and Greco, 1986). The plants may be upright and small leaved (Biddle *et al.*, 1988). Yellowing may not be apparent until the time of flowering, but then can progress rapidly from the base to the top of the plant. The root system is poorly developed with reduced *Rhizobium* nodule development. Lemon-shaped cysts, about 50 μm in diameter, can be found embedded in the roots. However, the cysts will slough off towards the end of the

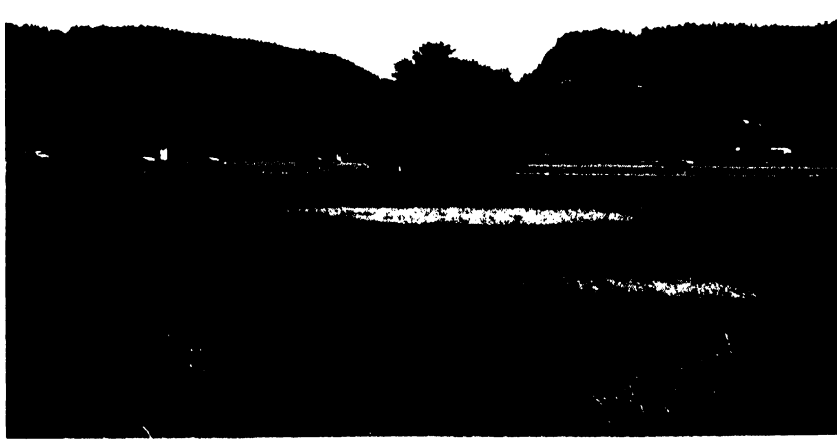

Fig. 6.12. Circular patches of peas infected with pea cyst nematode. Infected plants are yellow in colour (Photo: courtesy of D. Inglis).

season, particularly when the roots are decayed (Fig. 6.13). Fungal infections are frequently associated with pea cyst nematode infestations, and increase symptom severity (Stone and Course, 1974). Plant damage can be magnified by co-infection with *F. oxysporum* f. sp. *pisi* race 1 (Biddle *et al.*, 1988).

Infestations of pea cyst nematode can cause considerable crop loss (Di Vito and Greco, 1986; Biddle *et al.*, 1988). Heavily infested fields require long rotations out of peas before peas can be grown again economically. Tolerance limits for peas, broad bean (*Vicia faba*), and vetch (*Vicia* spp.) are listed as 0.5, 0.8 and 2.0 eggs g^{-1} of soil, respectively (Greco *et al.*, 1991). Yield losses of 20 and 50% in Italy can occur at 3 and 8 eggs g^{-1} of soil for peas; 5 and 15 eggs g^{-1} soil for broad bean; and 20 and 78 eggs g^{-1} soil for vetch, and complete crop failure at 32 and 64 eggs g^{-1} of soil for pea and faba bean, respectively (Greco *et al.*, 1991). Similar losses have been sustained in western Washington where dry pea seed yields averaged 18% less at 5 eggs g^{-1} soil and 87% less at 25 eggs g^{-1} soil compared to non-infested soil (D.A. Inglis, 1996, unpublished results). However,

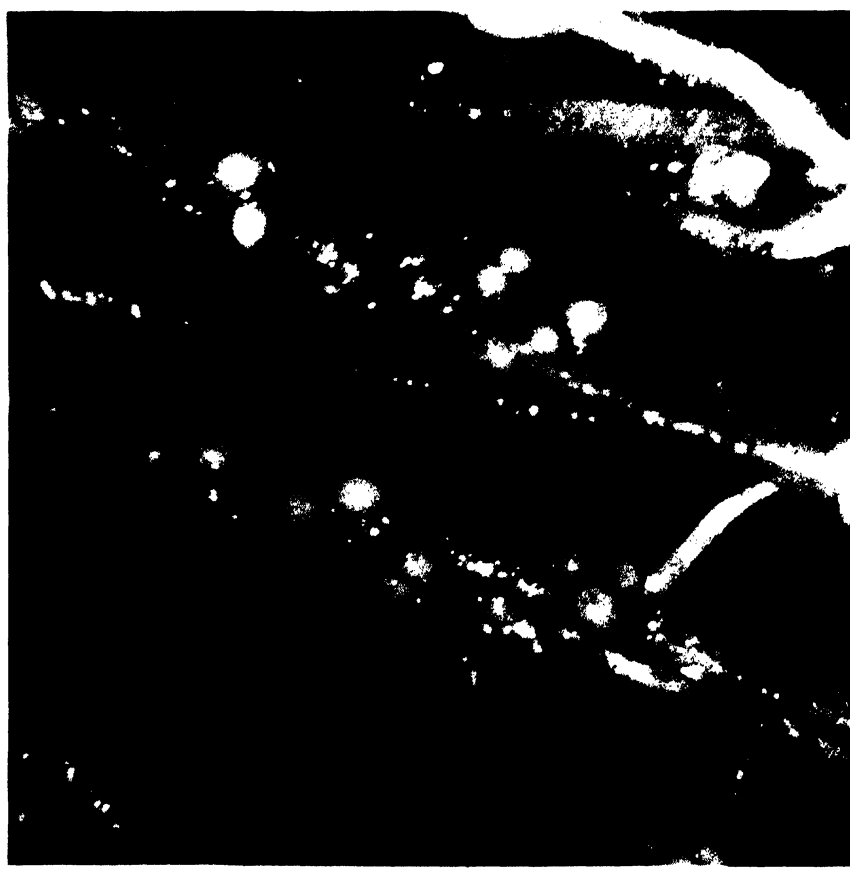

Fig. 6.13. Cysts of pea cyst nematode on surface of decayed pea root. These are visible to the naked eye (Photo: courtesy of D. Inglis).

Stone and Course (1974) reported variable relationships between initial pea cyst nematode densities and pea yields.

The number of generations of this nematode per year may vary according to host species and environmental conditions (Di Vito and Greco, 1986). Jones (1950) reported that one or two generations per year are completed on pea and faba bean in England, compared to southern Italy where three generations per year can occur on some host plants (Di Vito *et al.*, 1974; Greco *et al.*, 1986). On short-growing-season hosts, such as peas, *H. goettingiana* usually produces only one generation per year (Di Vito, 1991). Egg production of up to 100 eggs per cyst occurs at 10–13°C if soil moisture is adequate (Greco *et al.*, 1986). Above 14°C, females develop into cysts but no, or very few, egg masses are produced. Above 25°C, egg hatch and juvenile penetration of roots are suppressed even in irrigated crops (Di Vito, 1991).

Invasion of pea roots by pea cyst nematode under constant and fluctuating soil temperatures in both greenhouse and field experiments in western Washington also showed that soil temperature directly affected pea root penetration (Tedford and Inglis, 1995). After 286 degree hours (basal temperature of 4.4°C) in the greenhouse, a greater number of juveniles penetrated pea roots at 10 than at 18 or 26°C. In the field, later plantings, hence warmer soil temperatures, increased green pea yields.

Pea, broad bean, Austrian winter pea (*P. sativum* var. *arvense*), gross-pea (*Lathyrus cicera*), and vetch (*Vicia* spp.) are listed as major hosts of economic importance (Stone and Course, 1974; Di Vito, 1991). Di Vito *et al.* (1980) tested the reaction of several leguminous species to six populations of *H. goettingiana* under greenhouse conditions and found soyabean, common bean, chickpea, lentil and white lupin (*Lupinus albus*) among other hosts to be resistant. In addition, Jones (1950, 1965) and R.L. Huettel (Maryland, 1993, personal communication) concluded that soyabean and sweet pea (*Lathyrus odoratus*) are not hosts of pea cyst nematode.

Seventeen leguminous crops found in the Pacific Northwest of the USA were evaluated as potential hosts under greenhouse and field conditions (Tedford and Inglis, 1995). Nearly all tested plants, including processing peas, dry edible peas, faba bean, chickpea, lentil, hairy vetch (*Vicia villosa*), lima bean (*Phaseolus lunatus*), snap bean (*Phaseolus vulgaris*), alfalfa, red clover (*Trifolium pratense*), yellow clover (*Trifolium agrarium*), black medick (*Medicago lupulina*), alsike clover (*Trifolium hybridum*), lupin (*Lupinus alpinus*), and sweet pea (*Lathyrus odoratus*), had detectable levels of root infection. However, cyst development only occurred on green pea, dry pea, and faba bean roots. Faba bean roots yielded more cysts and eggs per plant, thus the reproductive potential of cyst nematode was higher on faba bean.

Cyst nematodes, in general, are often undetected under field conditions for years. For example, it takes 3–6 years before population densities of the golden nematode reach levels readily detected by regulatory surveys (Brodie and Mai, 1989). With the high susceptibility of peas, their wide geographic distribution, and the ease with which pea cyst nematode can be disseminated (infested soil transported on equipment, and on plant parts), the probability is high that pea growing areas where short pea rotations are practised could be infested.

Control

In the absence of host plants, cysts can persist in soil for several years (Brown, 1958). A population decline of 50% over the first 3 years was reported in both The Netherlands and England (Stemerding, 1960; Moriarty, 1963). In southern Italy, Ferris and Greco (1992) reported that pea cyst nematode can be managed by a combination of nematicides and crop rotation where populations of the nematode are low.

Chemical control has been reported with both liquid and granular fumigants and non-volatile nematicides (Di Vito and Greco, 1986). Continuous hatching of *H. goettingiana* eggs during the growing season prevents adequate control with low dosage rates of fumigants (Di Vito and Greco, 1986). Chemical control with non-fumigant nematicides should be initiated early in the growing season because the cyst nematode reacts strongly to hatching factors and can inflict substantial damage early in the growing season (Sikora, 1992). In the UK, oxamyl when broadcast and incorporated prior to planting, provided good control (Green *et al.*, 1981; A. Biddle, UK, 1993, personal communication).

Di Vito and Perrino (1978) reported that some accessions of *P. sativum* subsp. *arvense*, *P. sativum* subsp. *elatium*, and *P. abyssinicum* exhibited moderate resistance. Hybrid lines obtained by crossing susceptible *P. sativum* cv. Progress 9 with a resistant line of *P. abyssinicum* also exhibited moderate resistance in the F_2 generation (Di Vito and Greco, 1986). Inglis *et al.* (1995) evaluated the USDA *Pisum* collection for resistance in western Washington. Out of nearly 2500 accessions, only 22 accessions were more resistant than the susceptible controls. Readings were based on root ratings at the white cyst stage and foliar ratings at processing maturity.

CONCLUSION

In this chapter, we have attempted to describe the most important diseases of peas on a national and international scale, the disease symptoms, the pathogens, and to give currently acceptable control practices. Listed in Table 6.1 are several foliar diseases, one root disease and one virus disease, the causal pathogens, symptoms and known control which we consider of minor or local importance only. Peas are subject to an array of bacterial, fungal, nematode and viral diseases which affect the foliage and/or roots. Many of these diseases can be readily controlled by growing resistant cultivars. Such diseases include bacterial blight, downy mildew, powdery mildew and fusarium wilt. Also, many viral diseases, such as pea seedborne mosaic, bean leaf roll and pea enation mosaic, that cause serious diseases in peas are controlled by growing resistant cultivars. What is encouraging is that resistance to most of these diseases has been relatively stable over the years. The notable exceptions are bacterial blight and downy mildew, where new races or strains of the pathogens keep appearing.

Research on viruses of peas can be complex because of the interaction with the aphid vector and with overwintering hosts. More information is required on the aetiology of the viruses and their related strains, as well as on vector relations

and epidemiology, to develop predictive models for aphid build-up and insecticide spray scheduling. Most of the active work in legume virus research has occurred from about 1940 to 1985. Since this period, there has been a paucity of information in the literature on virus incidence and progress in developing new resistant cultivars, and more work is needed in these areas.

The severity of downy mildew can be lessened by longer rotations and the use of acylalanine fungicidal seed treatments. Also, the avoidance of planting reduced-foliage-type peas will help to manage this disease. Bacterial blight severity can be reduced by growing seed crops under arid conditions and the strict avoidance of centre pivot irrigation systems. In-furrow irrigation and wheel-line irrigation systems should only be used for pea seed increases.

Diseases such as the cortical root rots and *Mycosphaerella* blight have been difficult to control through conventional breeding efforts. The levels of resistance available in the pea genome are low and in many cases can be overcome by increased pathogen virulence, inoculum level and/or environmental stress. Currently, an integrated control approach is needed for these diseases which includes cultural practices, genetic resistance, seed treatments and appropriate foliar fungicide applications. Hopefully, genetic engineering technology will come to the rescue. The successful transformation of peas, starting with immature cotyledons (Grant *et al.*, 1995), will greatly facilitate the insertion of genes such as those for class II chitinase and class-II B-1,3-glucanase which may enhance quantitative resistance to root and foliar pathogens (Jach *et al.*, 1995).

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DISEASES OF FABA BEAN 7

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INTRODUCTION

No wild progenitor of faba bean (*Vicia faba*) is known but the geographical origin is generally thought to be the Near East. The small-seeded form, *V. faba* subsp. *paucijuga*, now mainly grown in Afghanistan and India, is considered to be more primitive than the commonly cultivated *V. faba* subsp. *faba*, and its varieties *minor*, *equina* and *major*. The expansion of cultivation from the centre of origin to Mediterranean countries, central Europe, Ethiopia, Afghanistan and China was accompanied by an evolution of diversity in seed size and shape and degrees of allogamy (Cubero, 1974). Large-seeded types did not appear until about 500 AD and the species only reached the New World with the Spaniards in the 16th century.

The total world production was estimated as 3,762,000 t on 2,855,000 ha in 1994 (FAO, 1995). China is the largest producer with 2,330,000 t from 1,700,000 ha, but over 100,000 ha in each country are grown in Ethiopia, Egypt, Morocco and the UK. In Europe, the area is generally declining but the crop still forms an important legume break from cereals in the UK, both from a spring and an autumn sowing.

The seeds are mainly used for human food in Africa, Asia and South America; faba beans are, for example, a significant proportion of the human protein intake in Egypt, but in Europe, Canada and Australia most of the production of small-seeded types goes for animal feed, the larger-seeded broad beans being used on a small scale as vegetables, fresh, canned or frozen.

The plant is a stiff-strawed annual legume with a tap root and secondary roots which establish symbiosis with *Rhizobium* and fix nitrogen. Flowers are about 35% cross-pollinated, mainly by bumble bees; heterosis and inbreeding depression are marked, and some cultivars are composites or populations rather than pure lines. In regions with mechanized farming, the crop has been adapted to the cultivation and harvesting methods that are used for cereals.

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The plant requires a considerable amount of water to maintain turgor, support its erect stems and prevent pod loss, so it is particularly subject to drought stress. High and low temperatures can limit pollination and fertilization, so in arid regions the crop is often grown in the winter or at high altitudes. There are winter-hardy cultivars but severe winters limit cultivation in cold continental climates. However, the above abiotic stresses are matched by the effects of various pests and diseases and where these are not controlled, or resistant cultivars have not been bred, the result is some instability of yields. This chapter describes the major diseases, but other diseases, shown in Table 7.1, also constrain production in some places or seasons.

CHOCOLATE SPOT

Aetiology

Chocolate spot is caused by both *Botrytis cinerea* Pers. ex Pers. and *Botrytis fabae* (Sard.). *B. cinerea* is the anamorph of *Botryotinia fuckeliana* (de Bary) Whetzel (≡ *Sclerotinia fuckeliana* (de Bary) Fuckel). A teleomorph of *B. fabae*, *Botryotinia fabae*, was described by Wu and Lu (1991), but apothecia were obtained experimentally and no confirmation of this report has appeared.

Biology

B. fabae is usually regarded as the more important causal agent (Mansfield, 1980), although Harrison (1984) has reported that *B. cinerea* may be more important than previously thought. *B. cinerea* can certainly sometimes cause rotting of green pods, gaining entry through dead flowers (Jellis and Bond, 1980) (Fig. 7.1). *B. fabae* can only be distinguished from *B. cinerea* on conidia size, not on appearance of lesions (Harrison, 1983). *B. cinerea* is a serious pathogen of lentil and chickpea (see Bayaa and Erskine, Chapter 8, this volume and Haware, Chapter 9 this volume).

The pathogen comprises mycelium which spreads in healthy leaves in humid conditions, but once leaves are senescent or falling, hyphae produce conidiophores and conidia. These are normally macroconidia but occasionally microconidia are formed in response to unfavourable fungal growth (Harrison, 1988). Conidia are dry spores which are dispersed and re-infect leaves. Sclerotia are the main survival structures of both *Botrytis* spp.; they are readily formed *in vitro* and can be found in dead stems in the field where they remain viable (Harrison, 1979). Sclerotia germinate on exposure to light, producing mycelium or conidiophores. Airborne conidia produced in this way can then re-initiate chocolate spot disease.

B. cinerea can produce the sexual stage *Botryotinia fuckeliana* by germination of sclerotia to produce apothecia and ascospores. Genetic recombination may occur during sexual reproduction and may account for the more rapid development of fungicide-tolerant strains in *B. cinerea* than in *B. fabae*. *B. cinerea* is a parasite and saprophyte on a wide range of host plants, whereas *B. fabae* is specialized for the invasion and colonization of *Vicia* spp., especially *V. faba*.

Table 7.1. Minor diseases of faba bean.

Disease	Pathogen	Distribution	References
Powdery mildews	*Erysiphe pisi* DC (*Erysiphe polygoni sensu lato*)	Widespread	Iqbal *et al.* (1988)
	Erysiphe cichoracearum DC	Middle East[1]	Al-Hassan (1973)
	Leveillula taurica (Lev) Arn.	Middle East	Tarr (1955)
	Microsphaera penicillata (Wall ex Fr.) Lev. var. *ludens* (Salmon) Cooke	Canada	Morrall and McKenzie (1977)
Leaf spots	*Cercospora zonata* Wint.	China, Europe, Middle East	Lang *et al.* (1993)
	Alternaria alternata (Fr.) Keissler	Middle East, E Europe	Simay (1987)
	Pleospora herbarum (Pers. ex Fr.) Rabenh. (anamorph *Stemphylium botryosum* Wallr.)	Middle East, E Europe	Simay (1992)
Stem canker	*Phomopsis fabae* Ondrej	E Europe	Ondřej (1991b)
Stem rot	*Sclerotium rolfsii* Sacc. (teleomorph *Corticium rolfsii* Curzi)	India	Singh *et al.* (1990)
Bacterial soft rots	*Pseudomonas fabae* (Yu) Burkholder	China	Salt (1983)
	Erwinia carotovora (Jones) Bergey *et al.* subsp. *atroseptica* (van Hall) Dye	Russia	Salt (1983)
Bacterial blight	*Pseudomonas syringae* pv. *syringae* van Hall	Middle East	Abd-El-Moneem *et al.* (1994)
Virus diseases[2]	Alfalfa mosaic virus	Widespread	Cockbain (1983)
	Broad bean wilt fabavirus	Widespread	Cockbain (1983)
	Faba bean necrotic yellows virus	Middle East	Katul *et al.* (1993)
	Pea enation mosaic pemovirus	Widespread	Cockbain (1983)
	Pea streak carlavirus	N America, Asia	Bos (1973)
Phyllody	Phytoplasma-like organism	Sudan	Nour (1962)
Dodder	*Cuscuta campestris* Yunck and other *Cuscuta* spp.	Middle East	Sauerborn and Saxena (1987)

[1] Middle East is used to describe West Asia and North Africa.

[2] Approximately 50 viruses and virus-like diseases have been reported from faba bean. Only those of more than local importance are listed here. For further details see Bos *et al.* (1988) and Cockbain (1983).

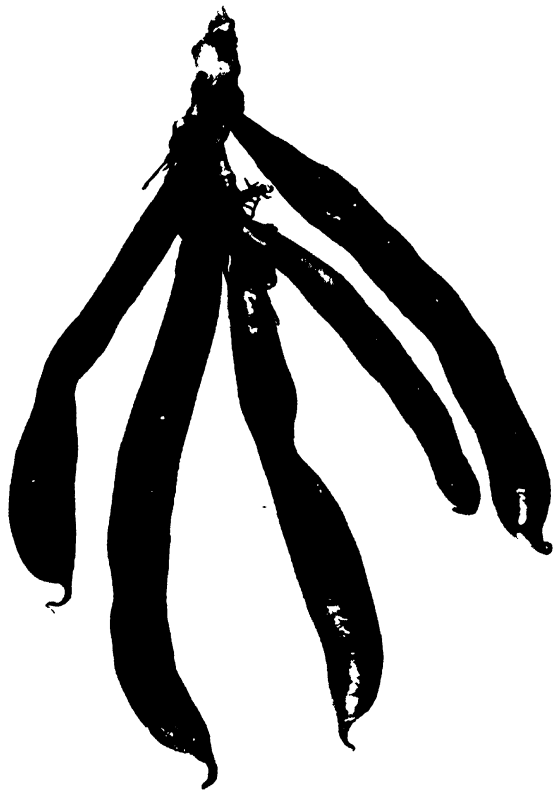

Fig. 7.1. *Botrytis cinerea* infection of pods.

The existence of races of *B. fabae* has been proposed on the basis of reaction to given differentials in seven Mediterranean countries (Hanounik and Mahila, 1986) but the resistance is quantitative and influenced by the environment so confirmation is needed. There are no demonstrable physiological differences among the putative races other than their suggested reactions to the differentials. Isolates of *B. fabae* in China were separated according to the abundance of sclerotia versus mycelium that they produced (Liang, 1993). Isolates of *B. cinerea* vary widely in many characters, and forms have been proposed according to type of sporulation and on the basis of certain hosts, but validity is doubtful (Ellis and Waller, 1974), and none is described especially for *V. faba*.

Symptoms

The main symptoms are on the leaves; flowers can also be infected and occasionally pods, but rarely stems. In the non-aggressive phase of the disease the classic symptoms are regular chocolate-brown spots giving an intensely 'peppered'

Fig. 7.2. Chocolate spot (*Botrytis fabae*) infection of leaves. top: non-aggressive spots; below: aggressive spreading lesions.

appearance especially on the older leaves (Fig. 7.2). However the non-aggressive symptoms can vary: the lesions can become more grey than reddish and more irregular, even elongated, instead of circular. These blotches can even be concentric under alternately wet and dry conditions and can be mistaken for *Cercospora zonata* or *Ascochyta fabae*.

In mild wet weather, especially in dense or lodging crops of winter beans on slow-drying soils, the disease becomes aggressive. Lesions enlarge, especially from the margin, until the whole leaflet is infected (Fig. 7.2). With a continued humid microclimate in the crop, defoliation occurs rapidly. The fungus can also grow saprophytically on fallen dead leaves, sporulate and re-infect younger, growing leaves. The disease is often first seen on damaged tissue of seedlings, especially frost-damaged winter beans in north-west Europe and, given humid conditions, progresses through the plant, infecting older leaves first.

B. cinerea and *B. fabae* damage leaf tissues of *V. faba* with the help of pectic enzymes and phytotoxins. The host responds by producing phytoalexins which inhibit germ tube growth. However, wyerone acid, the most important

phytoalexin, is metabolized by *B. fabae* more rapidly than by *B. cinerea*, and is prevented from accumulating in invaded tissues (Mansfield, 1982). Hence, *B. fabae* lesions become aggressive more frequently.

Epidemiology

Sporulation is usually on senescing or dead leaves at relative humidities above 80%, and can continue on the ground after leaves have fallen (Gondran, 1975). Spread through the crop is by conidia and is fast in a high plant-density and/or lodging crop; that is, in any situation where relative humidity is over 70% and temperature is 15–20°C (Harrison, 1980).

Carry-over from one crop to the next is by mycelium on crop debris followed by sporulation in favourable conditions, when crops are harvested in the autumn and the new crop sown in adjacent fields only 1 month later (Gaunt, 1983). Sclerotia in crop debris are probably a more important means of survival when there is a longer gap between crops (Harrison, 1979), for example where only spring beans are grown or in Mediterranean climates with a long summer gap.

B. fabae infection has been found in faba bean seeds but is unlikely to be an important source of carry-over because the frequency of infected seeds is very low; the fungus remains viable for only 9 months in the seed and has not been detected after sowing infected seeds (Harrison, 1978). Seed dispersal is therefore much less important in chocolate spot than in *Ascochyta fabae*.

The main feature of chocolate spot is the highly critical effect of environmental conditions. *B. fabae* infection and spread can change very rapidly from day to day according to temperature and humidity. Geographical distribution is also much influenced by rainfall and the water-retentive nature of the soil. The regions where aggressive attacks occur include the Nile Delta, near rivers in China, and humid maritime climates in western Europe. *B. fabae* is present, however, in almost all parts of the world where faba beans are grown; *B. cinerea* is ubiquitous.

Damage and Crop Loss

Damage is more frequent in humid than in arid regions of the world. In eastern England (where about 100,000 ha are grown, about half as winter beans) damage was severe in 1935, 1944 and 1971, producing up to 50% yield loss attributable to chocolate spot, a frequency of about 1 year in 15. Less severe epidemics occurred in 1954, 1958, 1968 and 1981 in the UK, that is in about 1 year in 10. Chocolate spot is also the most widespread disease of faba beans in China (Liang, 1989), where 50% yield losses have been experienced (Liang, 1986).

The extent of damage is very much influenced by the timing of a change to aggressive infection in relation to flowering and pod setting. Early severe infection during wet weather at flowering can cause almost complete loss of pods except for field borders. Yield loss is often correlated with premature *Botrytis*-induced leaf fall (Gondran, 1983). At the other extreme, non-aggressive spotting may not affect yield at all, photosynthetic capacity of green areas being flexible

enough to meet demands of the pods (Williams, 1975). The probability of aggressive chocolate spot in Scotland depends not only on weather conditions but on the amount of inoculum present (Harrison, 1988), but in southern England there always seems to be ample inoculum whenever the weather is humid. Other important factors are weakening of faba bean plants by poor soil, waterlogging, overcrowding, lodging or virus attacks.

Seed quality is also affected. Seeds from infected pods – and *B. cinerea* is important in this respect – are often blemished and may not be saleable for human consumption, especially tannin-free broad bean seeds which are white or light grey when healthy.

Management

Factors that predispose faba bean to chocolate spot can be partly avoided. A crop rotation that avoids bean crop debris and volunteer plants (Yarham and Gladders, 1993) in the field and adjacent fields, and crop hygiene where debris and straw are burned (if legally permitted) or deeply ploughed very soon after harvest all help to reduce inoculum. Adequate levels of soil nutrients and good drainage prevent premature senescence on which *Botrytis* increases severity. Open rather than sheltered aspects are less likely to develop chocolate spot.

Two major factors are to avoid early sowing (late October preferred for winter beans in north-western Europe) so as to reduce frost damage; and to use only moderate seed rate (20 seeds m^{-2}). The dangers of high plant density in the absence of chocolate spot control have been well demonstrated (Ingram and Hebblethwaite, 1976; Taylor, 1993). Winter beans are more prone to chocolate spot than spring beans in north-western Europe and south China but spring crops may also become infected in humid situations. Seed treatment with a mixture of benomyl and thiram reduced chocolate spot and improved yield on one occasion when a severe epidemic occurred in the UK early in the season (Bainbridge *et al.*, 1985), but this was an effect on the seedling rather than on seed infection (Harrison, 1988). Soaking seed in ethephon also reduced the severity of chocolate spot in Egypt (Salem *et al.*, 1992). However, it is generally better for the fungicide to be applied as the disease is beginning to develop, usually at the onset of flowering or mid-flowering (Creighton *et al.*, 1985). The current recommendation in the UK is that carbendazim + chlorothalonil or iprodione + thiophanate methyl vinclozolin, or tank mixes of these, be applied at onset of flowering and then a second spray 3 or 4 weeks later (Knott *et al.*, 1994). Chlorothalonil + vinclozolin sprayed at early flowering and 3 weeks later gave a significant (16%) increase in yield attributable to chocolate spot (Gladders *et al.*, 1991). Similar partial control has been reported in Syria (Hanounik, 1981). Mancozeb has given some control in Egypt (Abou-Zeid *et al.*, 1990). Enhanced control can be obtained by mixing adjuvants with fungicides though the two chemicals are very specific in effects (Green *et al.*, 1992), and there is a close correlation between adjuvant concentration and effectiveness (Amer *et al.*, 1994). However, some fungicides, first benomyl and more recently carbendazim, have

become less effective with usage, presumably due to the evolution of resistant strains of *Botrytis* (Knott *et al.*, 1994), particularly *B. cinerea*. Similar experiences with management of *B. cinerea* on lentil and chickpea are reviewed by Bayaa and Erskine (Chapter 8, this volume) and Haware (Chapter 9, this volume).

Morris and Lane (1990) concluded that biological control may be a viable alternative to chemical control of chocolate spot after they tested four isolates of *Trichoderma viride* as preventative inoculations. When Jackson *et al.* (1991) tested over 500 bacterial and 100 fungal isolates for antagonism to *B. fabae in vitro*, they found at least four bacterial isolates giving inhibition of the *B. fabae* cultures. However, we know of no successful field testing of biocontrol agents. Field tests are none the less well advanced for chickpea (see Haware, Chapter 9, this volume).

Twenty-one pure lines in the International Center for Agricultural Research in Dry Areas (ICARDA) collection in Syria were listed by Robertson (1995) as having some resistance to chocolate spot. Resistance has also been reported in Italy (Santorelli *et al.*, 1992) and China (Liang, 1993). The two lines with the highest and most consistent levels of resistance are BPL 710 and BPL 1179. These originated in Ecuador but were first recognized in the Nile Delta, and their resistance has since been transferred to Egyptian populations, by ICARDA to other adapted genetic stocks including some with a Moroccan base, as well as lines in South Australia (Luminis Pty Ltd, 1994).

However, transfer to the more widely divergent stocks of winter-hardy beans in England and France (where resistance is particularly required) is proving a more difficult and slower process than in eastern Mediterranean countries. This is not surprising when resistance is thought to be conferred by a combination of factors, including total free amino acids and phenols, and thickness of cuticle (Kararah *et al.*, 1991); also seedling reactions to infections do not always correlate with those of adult plants, nor detached leaves with field infections (Tivoli *et al.*, 1986). Moreover, resistance is thought to be additive (El-Hady Mohamed, 1988) and quantitative rather than qualitative (Robertson and Saxena, 1993) so it is difficult to trace identifiable gene(s) across segregating populations. BPL 710 is resistant at all locations where it has been tested (Hanounik and Maliha, 1986) and there is a possibility that this line and derivatives will remain durable, especially if bean cultivars are maintained as mixed populations. Where the BPL 710 source is proving difficult, breeding for (a) early maturity to set pods before aggressive attacks (Sass and Frauen, 1991), (b) slow leaf senescence, or (c) resistance to predisposing factors (e.g. frost and virus infection), should all help to reduce the effects of chocolate spot.

Multiple resistances, for example involving resistance to rust as well as chocolate spot, are known among the ICARDA lines (Bond *et al.*, 1994; ICARDA, 1995); and a combination of resistances to pathogens (including chocolate spot) with reduced levels of anti-nutritional factors is the objective of a European joint breeding programme. A check will need to be made however on whether zero vicine and convicine may increase *B. cinerea* infections, as suggested by Bjerg *et al.* (1984).

ASCOCHYTA BLIGHT

Aetiology

Ascochyta blight, also known as leaf, stem and pod spot, is caused by *Didymella fabae* Jellis & Punith. (anamorph *Ascochyta fabae* Speg.). The teleomorph was first reported by Jellis and Punithalingam (1991) on overwintering faba bean straw at Cambridge, UK, and has also been found in Australia (J. Dennis, Waite Agricultural Research Institute, 1990, personal communication). Both stages are described in Jellis and Punithalingam (1991). Ascochyta blight caused by several different fungi is reviewed in Chapters 4, 5, 6, 8 and 9, this volume.

There is some confusion in the literature between the ascochyta blight pathogens of lentil (see Bayaa and Erskine, Chapter 8, this volume) and faba bean. Despite their host specificity, Gossen *et al.* (1986) found that the two fungi were indistinguishable in cultural and morphological characters, proposing that they should be treated as *formae speciales* within the single species *Ascochyta fabae*. Recent work on the teleomorph suggests that such treatment is unsatisfactory, and that there is evidence on morphological and molecular grounds that the two pathogens belong to distinct species of *Didymella* (Kaiser and Hannan, 1994; W.J. Kaiser, Pullman, Washington, 1996, personal communication).

Biology

The fungus is highly specialized to faba bean and inoculations with conidia of *A. fabae* have been largely unsuccessful on other legumes, although infection may be possible under certain specific conditions (Gaunt, 1983). Differential interactions between faba bean genotypes have been reported by Hanounik and Robertson (1989) and Rashid *et al.* (1991a, b) and isolates have been classified into physiological races based on these. In the material studied by Rashid *et al.* (1991a), a total of seven genes for resistance was identified, and additional genes were also thought to be present. Resistance was either monogenic or oligogenic. The races reported by this group did not correspond to those of Hanounik and Robertson (1989) and work needs to be done to standardize methods and differential cultivars. *A. fabae* is very variable in culture; in growth rate, the production and size of pycnidia and conidia, and the number of conidial septa (Kharbanda and Bernier, 1980; Filipowicz, 1988).

Symptoms

Lesions on leaves are more or less circular, slightly sunken with a definite margin, and are usually dark brown with a lighter centre in which pycnidia develop. Later, lesions may coalesce to cover the whole leaf surface (Plate 15). Browning of the foliage, particularly in the leaf area surrounding the vascular tissue, sometimes occurs; this has been noticed particularly in determinate lines (Lockwood *et al.*, 1985) and may be due to the production and spread of the toxin ascochitine

(Foremska *et al.*, 1990), although Beed *et al.* (1994) were unable to isolate the toxin from infected plants. On stems and pods, lesions tend to be more sunken and a darker colour (Fig. 7.3). Infected seed may be stained or symptomless. The stains are not well defined and are indistinguishable from those resulting from other pathological and physiological conditions.

Epidemiology

Little is known about the factors influencing the dispersal of and infection by ascospores of *D. fabae*. However Pritchard *et al.* (1989) studied the conditions needed to establish successful infection by conidia. A minimum of 2–4 h of leaf wetness was required for successful germination at between 15 and 25°C and this was extended to over 8 h at 10°C. Conidial germination was significantly higher at 20–25°C than at 10–15°C. Germ tube penetration occurred almost directly beneath a spore adjacent to the anticlinal cell wall. Nothing has been reported on the infection conditions required by ascospores.

Van Breukelen (1985) found young leaves to be more susceptible to infection, contrasting with the findings of Dodd (1971) and Hanounik (1980). Differences in microclimate may contribute to the differences in lesion distribution over the plant, and a young furled leaf will provide a more favourable microclimate for infection than unfurled older leaves (van Breukelen, 1985). Spread from leaf to leaf, stem and pods is by splash dispersal of conidia. In general conidia do not travel more than short distances. During a whole season, Hewett (1973) found that infection did not spread beyond 6–10 m from an infective

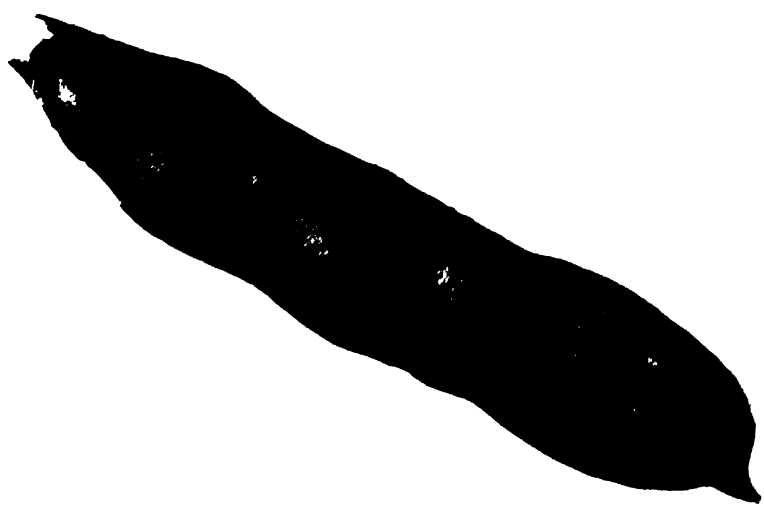

Fig. 7.3. *Ascochyta fabae* on a faba bean pod.

source in spring-sown beans. However in winter crops, spread of up to 200 m has been reported by Bond and Pope (1980). It could be that ascospores are implicated in such situations. Long-term survival of the pathogen has not been studied although, in our experience, the fungus can retain its viability in seed for at least 2 years.

The fungus is largely regarded as a seedborne pathogen. Disease transfer from the seed to the emerging seedling appears not to be systemic and is probably by physical contact (Pritchard *et al.*, 1989). Transmission levels vary depending on environmental factors and varietal resistance. Conidia from pycnidia produced in the centre of leaf lesions are splash dispersed to developing leaves, stems and later to pods. The fungus grows through the pod wall and infects the seed, completing this cycle. Volunteer crops in adjacent fields, and infected debris can also be sources of inoculum, especially for autumn-sown beans. The discovery of the teleomorph adds another dimension to pathogen dispersal. Pycnidia develop in infected straw over winter, maturing early in the year in the UK (Jellis and Punithalingam, 1991). Ascospores can be dispersed aerially over large distances, infecting seedlings of autumn-sown beans. However, no work has yet been reported on the relative importance of the teleomorph in disease transmission.

Kharbanda and Bernier (1978) observed chlamydospores in lesions on maturing faba bean stems and presumably these may be involved in survival of the fungus on straw lying in fields, which has been implicated in disease carry-over (Geard, 1962). However, their role in the life cycle of the pathogen has not been established.

Damage and Crop Loss

Ascochyta blight is a common and occasionally destructive disease reported from countries in all five continents (Gaunt, 1983). The pathogen attacks the leaves, stems, pods and seeds, leading to premature defoliation, stem break, reduced pod filling and blemished seed. The extent of the damage depends on climatic conditions and cultivar resistance. In the UK, Madeira *et al.* (1988) reported that disease reduced the leaf area index and the dry matter production of the crop. The reduction in seed yield was significant, representing a decrease of 15% compared with a prophylactic fungicide treatment.

In the Czech Republic, between 1984 and 1989, Ondřej (1991a) found between 18.0% and 30.7% infection in harvested seed. Lots with over 31% infection had a thousand-seed weight 32.8% lighter than those with infection under 10%. Plots sown with seed with 32.2% infection yielded 41.1% lower than those sown with seed with 2.2% infection.

In many years in eastern England, the disease is not destructive. Lesions are seen on young plants of autumn-sown crops during the winter and early spring but no further progress of the disease occurs until lesions appear on pods just before ripening. In this situation, the disease is of particular concern to the seed producer, who has to reach the required health standard (see next section).

Management

As ascochyta blight is both seed- and strawborne, the measures for avoiding crop debris and volunteer plants discussed for controlling chocolate spot also apply here. In western Europe, ascochyta blight is generally regarded as a more important disease of autumn-sown beans than spring beans. Crops sown in early autumn frequently show lesions of *A. fabae* before winter and considerable damage can be caused. In disease-prone areas, or with very susceptible cultivars, late sowing of autumn crops may therefore be advisable.

Much emphasis has been put on the production of healthy seed, particularly in the UK, as ascochyta blight has been regarded principally as a seedborne disease. Hewett (1966) developed a routine agar plate method for detecting the pathogen and until 1995 this was the basis of a statutory certification scheme. This has now developed into a marketing standard, with the following requirements:

Pre-basic seed	–	1 infected seed per 1000
Basic seed	–	2 infected seeds per 1000
Certified seed, first generation	–	2 infected seeds per 500
Certified seed, second generation	–	1 infected seed per 100

These standards only apply to seed being sold for commercial use, and are not applicable to farm-saved seed. However, it is recommended to farmers in the UK that farm-saved seed should be tested and discarded if infection exceeds 3%. For batches with between 1 and 3% infection, seed treatment is recommended (Knott *et al.*, 1994).

Control of ascochyta blight by treating seed using slurries and dips has generally proved to be erratic (Gaunt, 1983). Seed treatments containing thiabendazole are currently recommended for use in the UK (Knott *et al.*, 1994) but, used without additional foliar sprays, they may not provide adequate protection of susceptible cultivars (Jellis *et al.*, 1988). Foliar sprays have also been investigated, particularly for reducing infection in seed crops, in particular chlorothalonil has been found to be moderately effective (Kharbanda and Bernier, 1979; Lockwood *et al.*, 1982; Jellis *et al.*, 1984b).

Most breeding programmes for resistance to ascochyta blight have only been established in the last two decades. Probably the largest screening and parental development programme was that developed by ICARDA. ICARDA developed a number of resistant lines; for example 24 sources of resistance have been listed by Hanounik and Robertson (1988). In addition to these, locally adapted lines and cultivars with high resistance have been reported from a number of countries, including the UK ('Striker', NIAB, 1996, and 'Quasar', also known as IB18–1/30, Jellis *et al.*, 1984c), France (line 29H, Maurin and Tivoli, 1992), and Poland ('Fioletowy Czyzowskich' and 'Krasnoyarskii', Zakrzewska, 1986).

As well as genotypic variation for resistance, Lockwood *et al.* (1985) found that there was a strong correlation between straw length and the incidence of disease on the pod, tall cultivars having a lower incidence of disease. Zakrzewska (1986) also reported that genotypes with short internodes were the more suscep-

tible. Furthermore, she found that white-flowered cultivars (tannin-free) were very susceptible. This, to some extent, agrees with the findings of Jellis and Vassie (1995) who reported possible association between the gene for white flower (*sp-a*) derived from Ch170 and susceptibility to ascochyta blight. However, in the same experiment, a second gene for white flower (*sp-b*), derived from the cv. Toret, showed no associated blight resistance or susceptibility. Using near-isogenic pairs of lines, Helsper *et al.* (1994) also found no relationship between tannin content of seed and resistance. This work was done by inoculating foliage. When seed was artificially infected with *D. fabae*, white-flowered bean cultivars were highly susceptible (Fagbola and Jellis, 1994).

The mechanisms of disease resistance have not been studied in most of the resistant material which has been described, but Maurin *et al.* (1993) described a hypersensitive response or highly restricted lesion development in line 29H. Some other sources of resistance do not appear to function in the same way. Little is known about the durability of the resistance in different accessions; Hanounik and Robertson (1989) tested accessions in different locations and found line BPL 471 to be consistently resistant.

RUST

Aetiology

Brown rust of faba beans is predominantly caused by the fungus usually described as *Uromyces viciae-fabae* (Pers.) Schröter, although Boerema and Verhoeven (1979) consider that *Uromyces fabae* (Grev.) de Bary ex Fuckel is the correct name.

Biology

As well as infecting *V. faba*, *U. viciae-fabae* attacks many other leguminous species, including other *Vicia* spp. and those belonging to the genera *Pisum*, *Lathyrus* and *Lens* (Conner and Bernier, 1982a; see Bayaa and Erskine, Chapter 8 this volume).

U. viciae-fabae predominantly produces uredospores on leaves of faba bean, although aeciospores can also be found and teliospores are sometimes formed in large black sori on stems and petioles, particularly towards the end of the season. Pod infection has also been reported. Studies on host specialization in *U. viciae-fabae* have been carried out by a number of workers, summarized by Conner and Bernier (1982a). The overall picture presented by this work is far from clear. Gaümann (1934) proposed nine *formae speciales* based on host range but later workers have not been able to substantiate this, and Conner and Bernier (1982a) concluded that isolates share so many hosts in common that it was impossible to classify them meaningfully.

Evidence for distinct physiologic races of *U. viciae-fabae* infecting faba bean is much stronger. Conner and Bernier (1982b) developed a series of inbred lines in

which resistance was expressed as immunity or hypersensitive flecking. Using these lines, they described seven races of rust; a further four races were designated using pea lines as differentials. Resistance to these races proved to be controlled by several independent genes (Conner and Bernier, 1982c; Rashid and Bernier, 1986a).

Symptoms

Symptoms of rust appear initially on leaves as small round necrotic spots, in the centre of which reddish-brown raised powdery pustules occur (Fig. 7.4). These are often arranged as a central pustule surrounded by daughter pustules, each of which has a yellow halo. The pustules release masses of orange-brown uredospores which are wind dispersed to other plants. Between five and ten cycles of urediniospores are produced each season. Pods and stems can also be infected; the pustules may form elongated blisters below the epidermis before erupting

Fig. 7.4. Rust (*Uromyces viciae-fabae*) on faba bean leaf (Photo: courtesy of Processors and Growers Research Organization).

(Thomas and Sweet, 1990; Knott *et al.*, 1994). Black elliptical teliosori may be formed on leaves, petioles and stems, particularly in the later stages of infection (Lang *et al.*, 1993; Sache, 1995).

Epidemiology

Optimum conditions for germination and penetration of uredospores are 18–22°C and 95–100% relative humidity, and a humidity of 80% is essential. This is because the basis of adhesion of uredospores to the cuticle of leaves is a pad which develops on contact with an aqueous environment (Deising *et al.*, 1992). Light does not affect germination (Mohamed *et al.*, 1986). After a latent period of 8–10 days, sporulation of a single lesion continues for about 50 days, during which time about 1×10^5 uredospores are released (Mohamed *et al.*, 1986; Sache and Zadoks, 1995). In experiments conducted by Sache and Zadoks (1996), from an initial focus, disease within a plot spread at a radial velocity of expansion slightly lower than 0.1 m day^{-1}. However, the severity of disease in trap plots distant from the initial source of infection led them to postulate that two mechanisms of spore dispersal were operating, one over short distances at high frequency and the other over long distances at low frequency.

The fungus can survive in the absence of *V. faba* on wild species of *Vicia* and *Lathyrus* (Conner and Bernier, 1982a), both of which are common in areas of northern Europe where faba bean is cultivated. The method of survival probably depends on location. In the absence of the host, spores can remain viable for 1–2 years. Teliospores can withstand a wide range of temperature and germinate readily; uredospores can survive in cooler climates. Aeciospores survive in mild, Mediterranean climates but not through severe northern European winters (Gaunt, 1983; Virányi 1988). Within infected tissues, uredo-mycelium is probably an important means of survival.

Damage and Crop Loss

Rust is usually regarded as only a rather minor pathogen of faba beans in Europe (Virányi, 1988) although in recent years there have been more frequent and severe attacks (Lapwood *et al.*, 1984; Knott *et al.*, 1994); losses can be severe (Gaunt, 1983; Bond *et al.*, 1994). In extreme cases, losses can be as high as 70–80%, as recorded in China in 1933/34 (Lang *et al.*, 1993).

Studies on the effect of rust on components of yield have established that the extent of infection on leaves at the flowering nodes during pod filling has the strongest influence on yield. A reduction in the weight of seeds per stem and in seed weight have generally been the yield components most affected (Williams, 1978; Lapwood *et al.*, 1984; Rashid and Bernier, 1991), although Sache and Zadoks (1995) also found a significant relationship between rust severity and number of pods per stem.

Management

In northern Europe, where faba beans can be autumn or spring sown, rust is generally more of a problem on the latter, as the autumn-sown crop usually matures before rust develops strongly. However, as noted earlier in this chapter, autumn sowing favours other diseases such as chocolate spot and ascochyta blight. Factors other than disease will usually determine whether winter or spring cultivars are grown. As for many other diseases, removal or burial of plant debris reduces the inoculum available the following season.

Chemical control of rust can be very effective and give substantial yield benefits if applied at the right time, that is, from early to late flowering (Marcellos *et al.*, 1995). Effective fungicides are those containing fenpropimorph, tebuconazole, maneb, mancozeb, propiconazole, thiram or triadimefon (Yeoman *et al.*, 1987; Knott *et al.*, 1994; Marcellos *et al.*, 1995).

Screening for resistance to rust has been done in a number of countries. Two broad types of resistance have been described. Working in Canada, Bernier and his co-workers used glasshouse tests to identify race-specific resistance in a number of selected inbred lines. Resistance was expressed as immunity or hypersensitive flecking. This type of resistance has proven to be highly race-specific (Conner and Bernier 1982b, c; Rashid and Bernier 1986a). The same group (Conner and Bernier, 1982c; Rashid and Bernier, 1986b) recognized the existence of quantitative resistance ('slow rusting') and of tolerance to rust. Bhalla and Bernier (1984) evaluated this resistance and found that the most important component in slow rusting was infection frequency. The length of the latent period was also important, at least for some accessions. In the slow-rusting population 2N43, yield losses of only 1–2% were recorded in a trial where susceptible lines lost up to 68% of yield. However, other slow rusting populations incurred higher losses of between 6 and 43% indicating that partial resistance and tolerance are independent characters (Rashid and Bernier, 1991). To date, there is no evidence that slow rusting is race-specific.

At ICARDA, selection for resistance to rust was carried out over several years (Hanounik and Maliha, 1986) and a number of resistant lines, probably of the slow-rusting type, have been described (Bond *et al.*, 1994). Resistance has also been reported in a number of other collections, including Chinese (Luo *et al.*, 1991), Egyptian (Khalil *et al.*, 1984) and Ethiopian (Polignano *et al.*, 1990). Some existing cultivars are also reported to be highly resistant, for example 'Gidou I Hao' in China (Lang *et al.*, 1993) and resistance breeding programmes are under way in a number of countries.

DOWNY MILDEW

Aetiology

Faba bean downy mildew is caused by the fungus *Peronospora viciae* (Berk.) Casp. The species also attacks other species of *Vicia* and members of the genera *Lathyrus* and *Pisum* (Mukerji, 1975; see Kraft *et al.*, Chapter 6, this volume) but

isolates appear to be host-specific (Mukerji, 1975; Thomas and Sweet, 1990). Those attacking faba beans have recently been described as *P. viciae* f. sp. *fabae* (Jacz & Serg.) Boerema *et al.* (Boerema *et al.*, 1993).

Biology and Symptoms

Bean downy mildew has generally been regarded as a minor disease of faba bean but in recent years it has become more common and damaging on spring-sown beans in northern Europe, associated with the introduction of susceptible cultivars. Primary, systemic infection occurs occasionally on young plants via soil-borne inoculum and gives rise to pale green, stunted and distorted plants which die early. Sporulation occurs under humid conditions; this has a greyish-fawn and velvety appearance, and particularly occurs on the underside of the leaves. Secondary infection from wind-blown spores appears as pale yellowish-green patches on the upper leaf surfaces which enlarge to cover the entire leaf area. The characteristic greyish-fawn sporulating mycelium occurs on the underside of these patches (Plate 16). Infected material quickly becomes necrotic. Infection is generally restricted to the young leaves on the top third of the plant.

Epidemiology

The life cycle of the disease in *V. faba* has not been carefully studied. As the pathogen appears to be highly specialized, inoculum for infection must carry over from previous crops. Oospores are produced abundantly throughout the growing season in all plant parts except seeds (van der Gaag *et al.*, 1993) and these are disseminated in the soil with crop residues and provide inoculum to infect developing seedlings in subsequent crops. Little is known about optimum conditions for infection and spread. The disease is favoured by cool, humid conditions according to Knott *et al.* (1994), but in our experience spread of the disease continues at temperatures which inhibit pea downy mildew (>*c.*20°C). In growth chamber tests, van der Gaag *et al.* (1993) found that sporangia developed more rapidly at 15°C than 10°C. Oospores were also formed earlier at higher temperatures (tested up to 20°C) but the ultimate numbers produced in leaves were highest at 10–15°C.

Damage and Crop Loss

Primary, systemic infection can lead to early plant death and subsequent loss of yield. Destruction of young leaves as a result of secondary infection might also be expected to have serious yield effects when infection is severe but published data are not available. There have been reports of serious effects on broad beans (used fresh for human consumption) (Thomas and Sweet, 1990).

Management

Long rotations and good crop hygiene can help prevent the build-up of resting spores (oospores) in the soil. Late-sown crops tend to be the more susceptible.

Differences in susceptibility between cultivars have been recorded. Thomas and Sweet (1991) found Troy, Vector, Alfred and Octopus to be very susceptible and Maris Bead to have moderately high resistance. NIAB (1996) list 'Alpine', 'Luna', 'Maris Bead', 'Spear' and 'Titch' as resistant to downy mildew and 'Alfred', 'Aribo' and 'Maya' as susceptible. In growth chamber tests, van der Gaag *et al.* (1993) found 'Toret' to be more resistant than 'Maris Bead' (intermediate) or 'Melissa' (susceptible) when sporulation area on leaves was assessed. However, fewer oospores developed in leaves of 'Maris Bead' than the other two cultivars.

Seed treatment with fungicide (metalaxyl + thiabendazole + thiram) and foliar sprays (metalaxyl + thiabendazole or chlorothalinil, or fosetyl-aluminium) applied at early flowering, possibly repeated 10–14 days later, can give effective control of the disease.

STEM ROT

Aetiology

Stem rot, or sclerotinia disease, is caused by the fungi *Sclerotinia trifoliorum* Erikss. and *S. sclerotiorum* de Bary. Keay (1939) and Loveless (1951) found morphological differences between the *S. trifoliorum* attacking red clover and that mainly attacking faba beans. Keay described the latter as *S. trifoliorum* var. *fabae* Keay. In the UK, *S. sclerotiorum* attacks spring beans while *S. trifoliorum* attacks winter beans; these were distinguished on electrophoretic patterns of sclerotial proteins by Jellis *et al.* (1984d; 1990).

Biology

Sclerotia remain dormant in the soil for 6 to 8 years (Archer, 1988) but eventually they germinate near the surface in cool moist conditions and produce apothecia and ascospores. Entry to the host by germinating ascospores causes enzymatic breakdown of cell walls but is much assisted by wounding. Mycelium spreads throughout stems and then as the host matures, sclerotia form within the hollow bean stems in which they turn from white to black as they mature. Sclerotia of *S. trifoliorum* germinate in the autumn, those of *S. sclerotiorum* do so more frequently in the spring (Williams and Western, 1965).

S. trifoliorum infects red clover (*Trifolium pratense*; see Mercer, Chapter 12, this volume), trefoil (*Medicago lupulina*), lucerne or alfalfa (*M. sativa*), sainfoin (*Onobrychis viciifolia*), as well as winter (*Vicia*) bean. *S. sclerotiorum* infects a very wide range of hosts including peas (see Kraft *et al.*, Chapter 6, this volume), *Phaseolus* beans, and oilseed rape as well as spring (*Vicia*) beans. *S. trifoliorum* var. *fabae* may be specialized in infection of *V. faba* as well as having distinct ascospore

size, because an incompatibility line forms between the two varieties in culture (Loveless, 1951).

Symptoms

The first symptom is a slimy wet rot of the lower parts of the stem; the plant begins to wilt and can easily be pulled up. Single or small groups of affected plants are usually found scattered throughout the field (Knott *et al.*, 1994). White mycelium and/or black sclerotia can be found within or on the stems. In the UK, symptoms on winter beans occur in the spring and on spring beans in early summer. When mature plants are infected sclerotia form within pods.

Epidemiology

Ascospores are released over several weeks and blown by wind over several kilometres before germinating on host tissue (Salt, 1983). Then, sclerotia can survive in debris and in soil for 6 to 8 years. It is possible for sclerotia to spread with seed, though not often with well-cleaned bean seed because sclerotia are separated on size. *Sclerotinia* has been found in fruits of *Orobanche crenata* (Al-Menoufi, 1986). Sclerotia produce apothecia; there is no direct infection from sclerotia and only a small amount of direct movement of mycelium from plant to plant as there is with red clover, peas and oilseed rape.

Damage and Crop Loss

Sclerotinia occurs in most cool moist regions including North America, Europe and Mediterranean coastal countries (Hawtin and Stewart, 1979). Affected plants are killed or yield very little, but in northern Europe, where faba beans are normally grown only as a break from cereals, and especially for *S. sclerotiorum* on spring beans, the proportion of plants infected is usually too small to reduce the yield of a crop significantly. However, production can be seriously curtailed in some other regions including Greece (Karamanos, 1995) and Italy (Caruso and D'Anna, 1984). In many countries, *Sclerotinia* restricts the frequency of faba beans in crop rotations.

Management

Long rotations usually prevent build-up of the disease in the soil. Four years are recommended between winter bean crops and between red clover (*Trifolium pratense*) and winter beans (though white clover, *T. repens*, and alsike clover, *T. hybridum*, which are less susceptible, can substitute for *T. pratense*). As *S. sclerotiorum* has an extremely wide host range, at least 4 years should be allowed between spring beans and any other susceptible crop in the rotation (Jellis *et al.*,

1984a). Low plant density reduces infection, and infected crop residues should be destroyed and not fed to livestock, because this maintains inoculum. A possible chemical control is pentachloronitrobenzene (PNNB) for use on seed (Salt, 1983).

A breeding programme has been conducted in Greece. Lines KU 189, 190 and 191 were reported as showing some resistance though further work is needed to relate laboratory tests to field observations (C. Podimatas, Larissa, Greece, personal communication 1995; Karamanos, 1995).

FOOT AND ROOT ROT, AND WILT

Aetiology

A number of pathogens have been associated with the foot and root rot complex of faba beans, particularly when plants are stressed due to adverse conditions such as heat, drought or poor drainage. Species of the genus *Fusarium* are the most common pathogens isolated, including *F. oxysporum* Schlecht, *F. solani* (Mart.) Sacc., *F. avenaceum* (Fr.) Sacc., *F. graminearum* Schwabe and *F. culmorum* (W.G. Smith) Sacc. (Salt, 1983; Pascual Villalobos and Jellis, 1990; Helsper *et al.*, 1994), but other fungi may also be present, including *Rhizoctonia* spp., *Pythium* spp., *Phoma* spp., *Aphanomyces euteiches* Drechs. and *Cylindrocarpon destructans* (Zinssmeister) Scholten (Salt, 1983; Lamari and Bernier, 1985). In China (Yu and Fang, 1948), Japan (Yamamoto *et al.*, 1958) and the Sudan (Ibrahim and Hussein, 1974) a specialized form of *F. solani*, designated *F. solani* f. sp. *fabae* (Yu and Fang, 1948) has been identified and described. Wilt is also caused by *Fusarium* spp., principally *F. oxysporum* f. sp. *fabae* (Yu and Fang, 1948), although other species have also been recorded (Hanounik *et al.*, 1993; Salt, 1983). Similar diseases affect soyabean, pea, chickpea, pigeonpea and clovers and are reviewed in Sinclair (Chapter 3, this volume), Kraft *et al.* (Chapter 6, this volume), Haware (Chapter 9, this volume), Reddy *et al.* (Chapter 10, this volume) and Mercer (Chapter 12, this volume), respectively.

Biology

Fusarium spp. can attack seedlings or adult plants. At the seedling stage, high soil moisture has been shown to be a pre-disposing factor (Lang *et al.*, 1993). Infection of adult plants may also have occurred early in the growing season but the pathogen does not become aggressive until the host is stressed, often at flowering time. Restriction and decay of the root system is followed by blackening and decay of the stem base and chlorosis, wilting and eventual death of the foliage.

Symptoms

The pathogens become established in the xylem after penetrating young rootlets directly and through wounds in older roots. The first visible symptom is a marked

chlorosis of leaves, followed by reversible and ultimately irreversible wilting, which spreads upwards from the older leaves at the base of the plant. In moribund plants, the vascular tissue extending up the stem is necrotic. This is different from vascular tissue affected by foot and root rot pathogens which is only locally necrotic (Salt, 1983).

Epidemiology

With the exception of *F. solani* f. sp. *fabae*, the pathogens associated with foot and root rot are unspecialized, with a wide host range, and are capable of surviving in the soil for long periods in the absence of the host. *Fusarium* spp. have been isolated from faba bean seed (Zakrzewska, 1991), and tannin-free seed in particular can be heavily contaminated (Jellis *et al.*, 1993).

Damage and Crop Loss

Both foot and root rots, and wilt affect yield by reducing plant population density, if infection occurs early, and also adversely affect grain size and quality. Yield loss is sometimes serious (Leocata and Sesto, 1995).

Management

In areas where foot and root rot or wilt are a problem, rotations must be chosen with care to avoid build-up of the pathogens. In China, disease incidence due to *F. solani* was reduced from 38.1% with continuous cropping to 5.7% with a 1-year break (Lang *et al.*, 1993). As the diseases are frequently associated with stress, care should be taken to ensure optimum growing conditions. Soil pH does not affect the incidence of foot rot (Lang *et al.*, 1993). Seed dressing with metalaxyl + thiabendazole + thiram controls seedborne inoculation but is not effective against the more common soilborne inoculum. Foliar sprays will not control the disease.

High resistance to foot and root rot has been claimed for the Russian cultivar 'Burshtyn 56' (Yartiev, 1976) and tolerance of the disease has also been identified, for example in the German line KK13 which is being used in breeding programmes (Bond *et al.*, 1994). No useful sources of resistance to wilt are currently available, although some varietal differences have been noted in Egypt, Poland and Russia (Salt, 1983). In general, differences between cultivars are probably due to their relative ability to withstand stress and those with a high degree of heterogeneity and which display heterosis will generally withstand conditions conducive to foot or root rot and wilt better than inbred lines (Bond *et al.*, 1994). Pascual Villalobos and Jellis (1990) and Helsper *et al.* (1994) found that the tannin-free member of near-isogenic pairs was generally more susceptible to *Fusarium* spp. than the tannin-containing member. However, differences between white-flowered, tannin-free cultivars do exist and it is possible to select for resistance in the absence of tannin (Pascual Villalobos and Jellis, 1990).

BEAN LEAF ROLL

Aetiology

Bean leaf roll virus (BLRV) is in the luteovirus group of plant viruses. Synonyms include pea leaf roll virus (see Kraft *et al.*, Chapter 6, this volume) and legume yellows virus (Duffus, 1979). The luteovirus group appears to comprise a continuum of serologically related viruses (Waterhouse *et al.*, 1988) and other members of this group which have been reported to cause yield losses in faba bean include subterranean clover red leaf virus (SCRLV) (= soyabean dwarf virus) in New Zealand (Wilson and Close, 1973) and Australia (Johnstone, 1978), beet western yellows virus (BWYV) in the USA (Duffus, 1964) and chickpea stunt virus (CpSV) (Haware, Chapter 9, this volume).

Identification of BLRV, and related luteoviruses infecting faba bean, can be done on the basis of symptoms, their persistence in aphid vectors, and failure to be mechanically transmitted (Cockbain, 1983). A serological test such as ELISA will offer a more definitive and rapid diagnosis.

Symptoms

BLRV on faba bean produces symptoms of upward leaf-rolling and thickening, accompanied by interveinal chlorotic yellowing (Fig. 7.5; Cockbain, 1983). Early infection can suppress flowering and pod set. SCRLV and BWYV produce symptoms on faba bean similar to those of BLRV. Infection with BLRV or related viruses may be symptomless on other hosts, although SCRLV produces distinctive leaf reddening on subterranean clover (*Trifolium subterraneum*) (Ashby, 1984; see Mercer, Chapter 12, this volume).

Epidemiology

Luteoviruses are transmitted in a persistent manner by several species of aphid. They are not transmissible mechanically nor through seed. BLRV is common in Europe, the Middle East, North Africa and India (Ashby, 1984); BLRV infects pea in the USA (see Kraft *et al*; Chapter 6, this volume). Incidence of infection of up to 70% was recorded in spring-sown crops in England (Cockbain, 1980) and up to 60% in Germany (Schmutterer and Thottappilly, 1972). Makkouk *et al.* (1988) found BLRV to be the most common virus on faba bean in six Middle Eastern countries, with incidence usually of not more than 20%, but of 100% in exceptional cases. Incidence of 100% has also been reported from China (Yu, 1979). In Tasmania, up to 84% infection by SCRLV in broad bean was found by Johnstone and Rapley (1979).

Fig. 7.5. Symptoms of bean leaf roll virus (Photo: courtesy of A.J. Cockbain, IACR-Rothamsted).

Damage and Crop Loss

Ashby (1984) stated that BLRV rarely causes economic losses in Europe but, nevertheless, it is potentially a very damaging disease of faba bean (Thomas and Sweet, 1990). Naturally infected plants yielded 50–90% less than uninfected plants in the UK and in Germany (Tinsley, 1959; Heathcote and Gibbs, 1962; Schmutterer and Thottappilly, 1972). Yield loss due to infection with SCRLV was

as high as 91% in late-sown plots of broad bean in Tasmania (Johnstone and Rapley, 1979). Total loss of faba bean crops following 100% incidence of natural infection with BLRV occurred in the coastal region of Syria in 1986 (Makkouk *et al.*, 1988).

Tinsley (1959) noted that BLRV-infected plants in crops of broad bean were more susceptible to *Botrytis fabae*. Omar *et al.* (1986a) confirmed increased susceptibility to both *B. fabae* and *B. cinerea*, and noted that virus symptoms resemble premature leaf senescence, thereby inducing the fungus to sporulate and spread. Makkouk *et al.* (1988) also observed that BLRV-infected crop plants were usually more severely infected by *Botrytis* spp., and that this greatly increased the importance of this virus. In contrast, prior infection of faba bean with BLRV decreased pustule density on leaves subsequently infected by the bean rust pathogen *Uromyces viciae-fabae* (Omar *et al.*, 1986b), thus decreasing the severity of rust.

BEAN YELLOW MOSAIC

Aetiology

Bean yellow mosaic virus (BYMV) is a member of the potyvirus group. Synonyms include bean virus 2, and pea mosaic (or pea common mosaic) virus (PMV). However, some controversy exists as to whether pea mosaic virus, also known as the pea strain, is in fact a strain of BYMV or a distinct but closely related virus. It has also been recorded on soyabean (see Sinclair, Chapter 3, this volume), lupins (Hill, Chapter 11, this volume) and clovers (Mercer, Chapter 12, this volume).

Tentative identification can be done by symptom observation, but care must be taken since pea seedborne mosaic virus infection can produce similar symptoms on some faba bean genotypes. BYMV may be distinguished from other potyviruses infecting faba bean by the use of bioassay plant species (Bos, 1970), and identified by ELISA or cDNA dot blot hybridization.

Symptoms

BYMV is the most common cause, worldwide, of mosaic symptoms in faba bean (Cockbain, 1983). Symptoms typical of BYMV are vein-clearing in the youngest leaves 7–10 days after infection, followed by mild green mosaic, vein-banding and sometimes chlorosis (Fig. 7.6). A strain of BYMV causing mosaic, dwarfing, and necrosis and plant death was found on broad bean in Italy (Vovlas and Russo, 1978). The pea strain produces a distinctive bright yellow mosaic on faba bean and pea (Fig. 7.7).

Epidemiology

BYMV is transmitted by many aphid species in a non-persistent manner, and is transmitted easily by mechanical inoculation. The virus can also be seedborne in

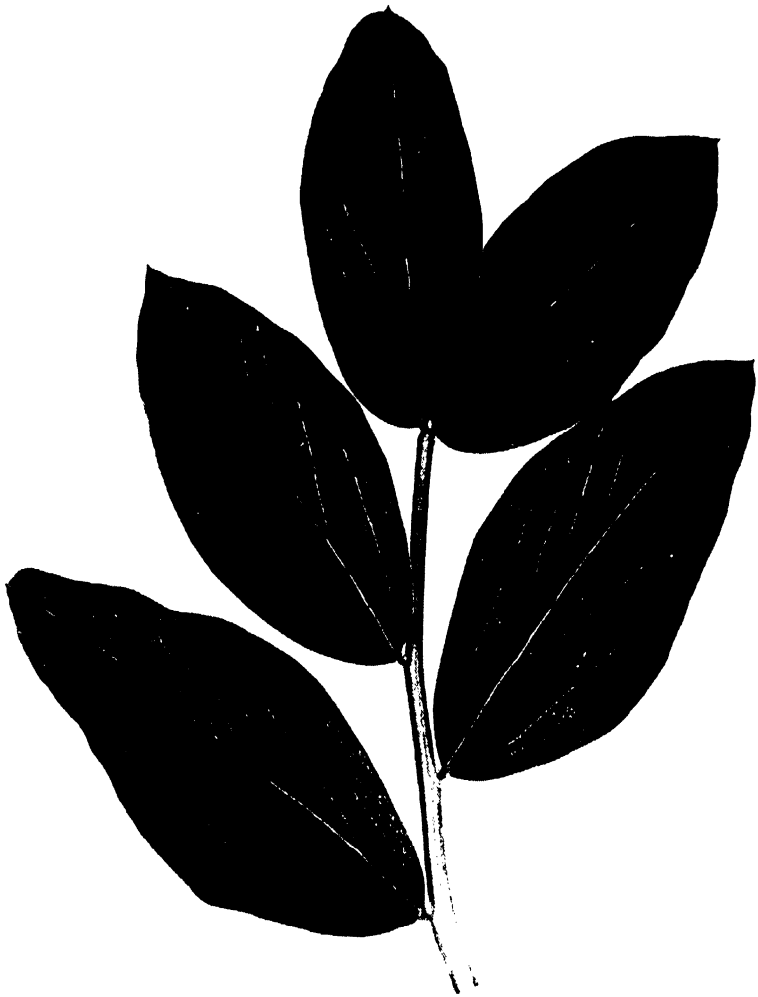

Fig. 7.6. Symptoms of bean yellow mosaic virus (Photo: courtesy of A.J. Cockbain, IACR-Rothamsted).

faba bean, but with usually a low transmission rate (Cockbain, 1983). Nevertheless, virus spread by aphids from random multiple foci of infected seedlings can be very efficient (Bos *et al.*, 1988). In a trial in Iran where seed-borne BYMV inoculum was less than 1%, as many as 34% of plants were infected within 15 weeks and 93–100% were infected after 22 weeks (Kaiser, 1973). Crop plants infected through seed transmission were the main source of inoculum. Infected faba bean seeds were regarded as a major source of infection of BYMV in

Fig. 7.7. Symptoms of bean yellow mosaic virus, pea strain (Photo: courtesy of A.J. Cockbain, IACR-Rothamsted).

Japan (Sasaya *et al.*, 1993), and seed transmission as high as 9% occurred in up to 10 of the 12 cultivars tested.

BYMV has worldwide distribution, occurring in most countries where legumes have been investigated for viruses (Bos, 1970). An incidence as high as 77% was recorded in Germany in plots untreated with aphicide (Schmutterer and Thottappilly, 1972). Makkouk *et al.* (1988) recorded an incidence of 67% in plants from naturally infected crops in Egypt, and noted that incidences of 100% infection are not uncommon in the Sudan late in the growing season. A survey of faba bean viruses in Egypt in 1993 and 1994 found that, of plants with symptoms suggestive of virus infection, 25% and 31% respectively were infected with BYMV (Makkouk *et al.*, 1994). Faba bean necrotic yellows virus was the most common in this survey with an incidence of 51% in 1993 and 62% in 1994.

Damage and Crop Loss

Infection with BYMV at or before flowering is much more serious than later infection. Frowd and Bernier (1977) inoculated faba bean plants at 5, 7 or 9 weeks after sowing. Compared to uninoculated plants, those inoculated with a mild isolate yielded, respectively, 59, 48 and 17% less, and with a severe isolate 96, 70 and 17% less. Makkouk *et al.* (1988) found that BYMV induced 81, 56 and 39% yield loss in experimentally inoculated plots following inoculation 11, 15 and 20 weeks after sowing, respectively. Mixed inoculation with BYMV and broad bean mottle virus before or during flowering resulted in almost complete failure of the crop. Pod set is affected by early infection. Nour and Nour (1962) reported that broad bean plants naturally infected with the pea strain set 82% fewer pods. Omar *et al.* (1986a, b) found, as with PLRV infection, that BYMV increased susceptibility to *Botrytis* spp. but decreased the pustule density of *Uromyces viciae-fabae*.

BROAD BEAN TRUE MOSAIC AND BROAD BEAN STAIN

Aetiology

Broad bean true mosaic (BBTMV) and broad bean stain (BBSV) viruses are both members of the comovirus group. The synonym of BBTMV is Echtes Ackerbohnenmosaik-Virus. The two viruses are unrelated serologically, and ELISA can be used for identification.

Symptoms

Foliar symptoms on faba bean are very similar for both viruses, with chlorotic mottling in patches on leaves (Fig. 7.8) and sometimes leaf deformation. Some leaves on an infected plant may appear normal (Gibbs *et al.*, 1968). Apical dieback may occur in cooler conditions. Seed from plants infected with BBSV often have a brown necrotic stain around the testa (Fig. 7.9).

Epidemiology

BBTMV and BBSV have been reported from Europe, North Africa and Asia but not from the Americas; BBSV also is reported from Australasia. Bos *et al.* (1988) regarded BBTMV and BBSV as economically important viruses of faba bean, with frequent seed transmission, although usually this occurs in only 1–3% of seed (Fiederow, 1980; Cockbain, 1983). However, a seed transmission rate of 46% for BBSV in a pea cultivar was reported by Musil and Kowalska (1993). The vectors of both viruses are weevils of the genera *Apion* and *Sitona*, and both viruses are also readily sap-transmissible. Both viruses may persist in the weevil vector for some weeks, and may spread rapidly from seed-infected foci if conditions are

Fig. 7.8. Symptoms of broad bean stain virus on leaf (Photo: courtesy of A.J. Cockbain, IACR-Rothamsted).

Fig. 7.9. Symptoms of broad bean stain virus on seed (Photo: courtesy of A.J. Cockbain, IACR-Rothamsted).

favourable. The viruses are widespread only in occasional years in the UK, presumably because of low vector numbers or few sources of inoculum in most years.

Fiederow (1980) found that 35% of plants with virus-like symptoms collected from field crops in Poland were infected with BBTMV. Makkouk *et al.* (1987) detected BBSV in 12% of faba bean samples with virus-like symptoms. A further survey (Makkouk *et al.*, 1988) found BBSV in 19% of all samples tested. BBTMV was also found but was the least common of the nine faba bean viruses detected.

Damage and Crop Loss

Vorra-Urai and Cockbain (1977) found up to 92% less pod set and 94% less seed, compared to that of symptomless plants, in plants showing symptoms of natural infection with BBTMV and BBSV through seed transmission. BBTMV reduced the seed yield of sap-inoculated plants of horse bean by 44% under glasshouse conditions (Fiederow, 1983). Bailiss and Senananyake (1984) inoculated faba bean plants in the glasshouse and in the field, and found that BBTMV reduced yield by 89%. Yield losses were progressively less the older the plants at the time of inoculation. Makkouk *et al.* (1988) also experimentally inoculated plants grown in field plots; BBSV reduced yield by 84% with inoculation 11 weeks after sowing but by 39% with inoculation after 20 weeks. As well as loss of yield *per se*, the stain induced by BBSV can render faba bean seed useless for processing for human consumption (Bos *et al.*, 1988).

PEA EARLY BROWNING

Aetiology

Although primarily a disease of pea, and locally important in The Netherlands and England (Bos and van der Want, 1962; Gibbs and Harrison, 1964; Boulton, 1996), pea early browning tobravirus (PEBV) can also infect faba bean. There is a very wide diversity of serological relationships among isolates and serotypes of PEBV, and infective isolates also exist which lack the protein coat necessary for serological detection. ELISA can thus be inconclusive and misleading for PEBV (Boulton, 1996). For accurate detection, cDNA hybridization or polymerase chain reaction can be used.

Symptoms

PEBV is almost always symptomless on faba bean (Bos and van der Want, 1962; Fiederow, 1980, 1983; Cockbain *et al.*, 1983) although virus concentration in plants may be high. Mahir *et al.* (1992) stated that symptomless infection appears to be widespread in faba bean in North Africa, although Lockhart and Fischer (1976) reported a very mild mosaic caused by PEBV in Morocco. The broad bean yellow band virus (BBYBV) serotype of PEBV (Russo *et al.*, 1984),

however, can produce yellow vein-banding, rings and line patterns on faba bean leaves, and brown necrotic rings on the pods, although symptomless infection with BBYBV can also occur. A Libyan isolate of PEBV which was not serologically related to BBYBV could also induce vein-banding symptoms (Bos *et al.*, 1993). Cockbain *et al.* (1983) reported interactions between BLRV and PEBV or pea enation mosaic virus, inducing necrotic symptoms on faba bean that were not typical of infection with the viruses alone.

Epidemiology

PEBV is transmitted through sandy or light-textured soils by species of trichodorid nematodes. Transmission is also common through pea seed, and occurs to a lesser extent through seed of faba bean. Fiederow (1980, 1983) reported seed transmission of up to 8% in faba bean seed grown in the glasshouse. Cockbain *et al.* (1983) found that two out of three cultivars of faba bean showed transmission of PEBV of up to 5%, and Mahir *et al.* (1992) found transmission of up to 45% in seed from sap-inoculated plants.

Damage and Crop Loss

Fiederow (1983) reported an average reduction in seed yield of 27% in faba bean when sap-inoculated with PEBV under glasshouse conditions, even though infected plants were symptomless. When plants were inoculated both with PEBV and BBTMV, infected plants showed symptoms only of BBTMV, with a yield loss of 77%. Cockbain *et al.* (1983) found that leaf and stem necrosis, and sometimes early death of plants, was induced by inoculation of PEBV and PLRV together. No further losses in faba bean due to PEBV have been described, although the severe symptoms of BBYBV on broad bean in Italy (Russo *et al.*, 1984) imply that yield losses may occur there.

PEA SEEDBORNE MOSAIC

Aetiology

Pea seedborne mosaic potyvirus (PSbMV) causes a disease of prime importance in pea (see Kraft *et al.*, Chapter 6, this volume) but the virus can affect faba beans as well. Three pathotypes (strains) have been reported from pea: P-1, L-1 and P-4. All can infect faba bean, but pathotype L-1 is the only one which can infect lentil (*Lens culinaris*). The virus can be identified in foliage and seed by ELISA.

Symptoms

Foliar symptoms of PSbMV in faba bean are vein-clearing and mosaic, particularly on younger leaves, and are quite similar to those of BYMV. Fagbola *et al.*

(1996) noted varying severity of symptoms, including stunting and severe leaf, flower and pod distortion, on different cultivars following sap-inoculation in the glasshouse with the three pathotypes of PSbMV. Some cultivars remained symptomless, although infected. A proportion of abnormal seed was produced by all the cultivars. This was smaller than seed from uninfected plants, with frequent splitting of the seed coat, and was also often necrotic, in some cases showing as a wavy line around the seed coat.

Epidemiology

PSbMV is transmitted non-persistently by several species of aphid, and is sap-transmissible. The disease has a worldwide distribution, occurring wherever the pea crop is grown (Khetarpal and Maury, 1987). Kvičala *et al.* (1975) found a number of viruses including PSbMV occurring on pulse crops in the former Czechoslovakia, and Lundsgaard (1981) isolated PSbMV from the broad bean cv. Wieselburger growing in a field near to pea plots in Denmark. PSbMV and BYMV were reported to be the most important viruses in faba bean growing areas of Japan (Tachibana, 1981). Makkouk *et al.* (1988) found PSbMV in 6% of samples tested in a survey of faba bean viruses, and Bos *et al.* (1988) and Fortass and Bos (1991) also reported isolation of PSbMV from naturally infected faba bean in West Asia and North Africa. Boulton *et al.* (1996) reported natural infection of faba bean in the UK, the virus most probably having been aphid-transmitted from adjacent infected peas, but in some cases the nearest known source was 1 km distant.

PSbMV is transmitted through pea seed with an efficiency of up to 100%, although this varies greatly with cultivar (McKeown and Biddle, 1991; Wang *et al.*, 1993). In faba bean, transmission through seed following sap-inoculation of the parent plants has been reported by Musil (1980), Lundsgaard (1981) and Fagbola *et al.* (1996), but there has been no report of seed transmission following natural infection, even though virus may be present in seed lots (Boulton *et al.*, 1996).

Damage and Crop Loss

Although PSbMV has a wide distribution in faba bean there are few reports of yield loss due to natural infection. Tachibana (1981) reported that PSbMV and BYMV considerably reduced both the number of pods and the size of seed.

Following sap inoculation under glasshouse conditions Fagbola *et al.* (1996) reported that, in addition to severe stunting in some cultivars, significant amounts of smaller and poor quality seed were produced. This would be an important economic factor not only where faba bean seed is destined for freezing, canning and other human consumption, but would also reduce acceptable yields of seed for animal feed.

MANAGEMENT OF VIRUS DISEASES

Since there is no curative treatment for virus diseases once crop plants are infected, only two control strategies are available; avoidance or prevention of infection, and selection for resistance or tolerance to viruses.

Measures for avoidance of infection with aphidborne viruses include growing crops at a distance away from known early sources of infection, such as clover fields. Even so, isolation may not always be effective where the vectors are particularly active (Bos *et al.*, 1988). It is difficult to isolate crops from infection by viruses such as BLRV and pea enation mosaic virus, which persist over long distances in their aphid vectors. It may be possible, especially for seed multiplication, to grow crops in areas such as higher ground where the vectors may be less active or arrive at later stages of crop maturity. Sowing earlier in the spring when possible may allow the crop to be more mature when the most damaging period of vector activity occurs. PEBV may be avoided by not growing susceptible crops on land where the virus is known to be present.

Strict statutory seed certification schemes, such as that successfully used for many years in The Netherlands to control the spread of PEBV in pea (Bos and van der Want, 1962; Boulton, 1996), may not be practical nor justified in many instances for seedborne virus diseases of faba bean. Even so, the production and use of seed carrying little or no virus is desirable. Seed lots, or preferably sample plants grown from seed, which better indicate the actual rate of transmission, can be screened for infection by ELISA. Those involved with distribution of seed for commercial, experimental or breeding purposes should also ensure that only healthy seed is exchanged or acquired.

Insecticides are effective in preventing infection, and spread within a crop, of luteoviruses such as BLRV since the vector is deterred or killed before virus is transmitted to the host phloem cells. They are less effective when viruses are transmitted in the non-persistent manner. Spread within the crop of non-persistent viruses in epidemic situations, nevertheless, may be significantly limited by insecticide use. It is particularly important to prevent infection up to the time of pod set. Table 7.2 lists insecticides currently approved for use on faba bean in the UK (Whitehead, 1996).

Breeding for resistance to faba bean viruses has been reviewed by Cockbain (1983), Bos *et al.* (1988) and Makkouk *et al.*(1993). However, there are few good sources of resistance, and breeding programmes are not as advanced in faba bean as in other cool-season crop legumes, particularly pea.

Although it is obviously of benefit to maintain crops as virus-free as possible, the degree of control measures taken must be economically viable and environmentally sound. Much will depend on local conditions and the epidemiology of particular viruses, the availability of, and means to implement, an efficient control measure, and the extent to which virus disease in the crop can be tolerated by the growers and users.

Table 7.2. Chemicals for control of virus vectors in faba bean, approved for use in the UK (Whitehead, 1996).

Aphids	Organophosphorus	Demeton-S-methyl
		Dimethoate
		Disulfoton
		Heptenophos
		Malathion
		Phorate
	Carbamate	Pirimicarb
	Alkaloid	Nicotine
	Pyrethroid	Remethrin
	Fatty acids	
Weevils	Organophosphorus	Phorate
		Triazophos
	Pyrethroid	Cypermethrin
		Deltamethrin
		Esfenvalerate
		Lambda-cyhalothrin

BROOMRAPE

Aetiology

Broomrapes (*Orobanche*) are higher plant species in the family *Orobanchaceae* and are obligate parasites on many crops and wild plants. By far the most important species attacking *V. faba* is *O. crenata* Forskall, though occasionally *O. ramosa* L. *O. aegyptiaca* Pers., *O. foetida* Poiret and *O. minor* Sm. have been seen on faba beans (Fig. 7.10).

Biology

Seeds of broomrape germinate in the proximity of roots when stimulated by substances excreted by the host (Cubero and Moreno, 1979). Penetration of the haustorium into the host's root is by mechanical pressure but growth within the roots is by enzymatic processes (Dörr and Kollmann, 1974). Connection between the two xylems is established before connection of the phloems. After attachment, three other growth phases of the parasite are recognized: as a 'nodule', formative and adult. Flowering tends to coincide with that of the host; there are 10–100 flowers per plant, and cross-pollination is by bees. Each capsule contains on average 4000 seeds, with the number of seeds per parasite plant ranging from 50,000 to 500,000. Seeds remain in the soil for long periods, up to 19 years (Cubero and Moreno, 1979). The population of *O. crenata* at Alameda, Spain,

Fig. 7.10. Broomrape: *Orobanche crenata* (right); *O. aegyptiaca* (left).

reached 4 million seeds m^{-2}, though only 0.003% attached to faba bean roots (Lopez Granados and Garcia Torres, 1993).

O. crenata, unlike many other species of *Orobanche*, is adapted to hot and dry regions; hence it is found on faba beans in Mediterranean countries and the Middle East. Southern Spain, southern Italy, Greece, Egypt and Morocco are particularly affected. Although *O. minor* is common on clover, it rarely infects faba bean and there are very few reports of broomrape as a pest of faba beans in northern Europe, China or the Americas. In fact there are proposals for quarantine procedures, including *O. crenata* on faba bean, on *Orobanche* entering USA (Musselman, 1993).

Although *O. crenata* is mainly found on faba beans, peas, lentils (see Bayaa and Erskine, Chapter 8, this volume) and vetches, it can also infect carrot, lettuce and sunflower. Pathotypes of *O. crenata* from host-adapted populations that vary in virulence on faba bean have been described by Cubero and Moreno (1979); and cultivars that were resistant to *O. crenata* in Spain, Syria and Morocco were susceptible to a red-stemmed species identified as *O. foetida* in Tunisia (Cubero *et al.*, 1993).

Symptoms

The first symptoms are pale, slightly pink, fleshy shoots of the adult stage of the parasite that can be seen emerging from the soil near the host bean plants. The faba bean plant may tolerate a few broomrapes and transpiration through the parasite is relatively small, but eventually the effect on the host plant is wilting and desiccation due to competition for water. In a severe infestation of a susceptible host, the bean collapses and the field appears like a crop of broomrape.

Epidemiology

O. crenata is a persistent pest of *V. faba* in Mediterranean countries because the seeds are so small and numerous and able to survive in the soil for periods over 19 years, germinating only when stimulated by a faba bean crop. More than one compound from the host is needed to trigger germination (Whitney, 1995). Although seeds germinate at cool temperatures (13–23°C), growth of the parasite is not optimum below about 25°C. Drying winds also increase the amount of infestation (Cubero and Moreno, 1979). As the seeds are so small, it would be quite possible for *O. crenata* to spread with *V. faba* seed samples. The fact that there are few reports of such-long distance dissemination probably indicates the critical nature of conditions for germination and attachment.

The parasite has evolved to exploit the host fully and quickly so as to produce as many as possible of its own seeds before it dies of desiccation along with the host (Whitney, 1995). Thus the host suffers in proportion to the amount of infestation unless some roots extend deeply beyond the parasitized region.

Damage and Crop Loss

Schmitt (1979) evaluated damage in an area in Morocco where 80% of the bean plants were infested and 14% of the bean plants showed severe yield losses. Fifty per cent and up to 100% yield losses are not uncommon. Cropping is constrained by length of rotations (Lutzeyer *et al.*, 1994), and farmers divert land to other crops.

Management

Late sowing (January in Morocco) can avoid some of the infestation but this results in lower yield; or early-maturing cultivars have to be used which are often not so high yielding. High plant density (50 plants m^{-2}) had less attack and higher yield than 17 plants m^{-2} in trials conducted by Pieters and Aalders (1986).

There is less broomrape in irrigated regions of Spain (Cubero and Moreno, 1979), but *V. faba* is not such a high-value crop that it often warrants irrigation. Hand weeding of broomrapes has been ineffective. Trap crops, like flax or vetch

(*Vicia sativa*), which are ploughed or grazed once the broomrape seeds have been stimulated to germinate, have been tried but with limited success; not all *Orobanche* seeds germinated and the system did not fit well with agricultural practice.

Good control of *O. crenata* has been obtained with glyphosate and pronamide (Zahran *et al.*, 1980), chlorsulfuron (Garcia Torres *et al.*, 1991) and imazethapyr (Saber *et al.*, 1994). Imazethapyr, imazaquin and glyphosate also gave some control of *O. foetida* (Kharrat and Halila, 1996). Applications are usually made at the subterranean stage of the parasite, and the chemical is translocated from host to parasite. However, soil treatment with dazomet also prevented infestations of *O. crenata* (Khalaf *et al.*, 1994). Though the effect of spraying is greatest on susceptible cultivars of faba beans, there is still a significant improvement in yield in spraying some resistant cultivars (Robertson and Saxena, 1993). Chemical germination-stimulants include gibberellic acid but there may be further development of strigol analogues (Saghir, 1994).

A number of pests attack broomrape. Some success was achieved using the fly *Phytomyza orobanchiae* (Kalt.) in Russia (Cubero and Moreno, 1979) and in Turkey (Giray and Nemli, 1983). However, these trials were directed at species other than *O. crenata*; also the fly is itself attacked by a wasp. The fungus *Fusarium orobanche* Jacz. is another candidate, but as with most biological control of *Orobanche*, even 95% control means that many seeds escape and can re-infect beans next season. *Fusarium* isolated from *O. crenata* by Al-Menoufi (1986) was non-pathogenic on *V. faba* and therefore could be a potential candidate.

There have been several reports of genetic resistance to *O. crenata* in *V. faba* (Cubero, 1983, 1994; Karamanos and Avgoulas, 1989; Kheir *et al.*, 1989). The first reports suggested an association of resistance with small seeds (VF 172, a *V. faba* subsp. *paucijuga* line) or late maturity (F402 in Egypt) and a skew toward small seeds and late maturity has been noted in resistance scores. In fact, Aalders and Pieters (1986) warned that plant vigour is a misleading factor in the search for resistance. However, it is now clear from crosses and their derivatives that resistance can be transferred into other genetic backgrounds. One of the most successful was from VF 1071 (a 402 derivative) × the Spanish susceptible 'Brocal' which resulted in the resistant cv. Baraca (Cubero *et al.*, 1992). In an infested trial, Baraca had 0.25 broomrapes per plant (cf. 3.79 for susceptible control) and three times the yield. Resistance of the F402 and Baraca type is thought to be quantitative and strongly additive, and breeding is by recurrent selection (Cubero, 1994) or pedigree breeding (Khalil *et al.*, 1994). However, polycross testing leading to synthetic cultivars has also been proposed by Radwan and Darwish (1991). The resistance of VF1071 has proved to be stable (Cubero *et al.*, 1993) as, so far, has that of Baraca, but lines in Egypt (cf. BPL 241 with BPL 1656) varied in their reaction to different *O. crenata* accessions (Radwan *et al.*, 1988), there was genotype–environment interaction in terms of resistance in Spain (Flores *et al.*, 1996) and the parasite is known to be very polymorphic (Verkleij *et al.*, 1991). There may be different mechanisms of resistance, including a lack of exudates which stimulate pathogen germination (Cubero *et al.*, 1993). Thus, new sources of resistance are needed. The resistance of ICARDA lines 18009S, 18025S, 18105S, 8/972 and 8/9/128 to *O. crenata* was confirmed in

Algeria, and in Tunisia the tolerance of BPL 818, 838, 911, 990 and 1015 to *O. foetida* was demonstrated. Breeding for *Orobanche* resistance in Egypt resulted in the cultivars Giza 674 and 429 which yield 80% more than the commercial cultivar in infested fields (ICARDA, 1995). Chiaro TL was reported as resistant in Greece (Karamanos and Avgoulas, 1989), Locale di Castellano in Italy (Perrino *et al.*, 1988), BPL 2830 in Morocco (Robertson and Saxena, 1993), while Aalders and Pieters (1987) drew attention to BPL 2210, a line which showed lower *O. crenata* attack than predicted on its vigour. Evidence that resistance of new lines is not due to reduced stimulation of *Orobanche* seeds by root exudates (Woerden *et al.*, 1994) nor to avoidance mechanisms, and therefore is probably associated with gene-controlled histological differences, was also given by ter Borg *et al.* (1994).

Although improved sources of resistance are becoming available, control of broomrape may, for some time, require an integration of resistant cultivars with agronomic measures, including delayed sowing and the post-emergence application of glyphosate or imazethapy, as shown by the work of ICARDA (Saxena *et al.*, 1994). However, the price of herbicides may limit their use. Many of the above control measures and cultivars are not yet being applied (Lutzeyer *et. al.*, 1994).

STEM NEMATODE

Aetiology

Stem nematode *(Ditylenchus dipsaci* (Kühn) Filipjer) is a bisexual species with worm-like juveniles and adults measuring up to 1–1.5 mm long. It is a migratory endoparasite of stems and bulbs. Eggs are laid within plant tissue and there are four juvenile stages, the life cycle taking 3–4 weeks (Hooper, 1991). Juveniles and young adults can withstand desiccation; the nematodes clump together, can be dispersed as 'wool', then revived with moisture.

Other nematodes attacking faba bean include *Heterodera goettingiana* Liebs., the pea cyst nematode, as described by Sikora and Greco (1990), and also *Meloidogyne* species (Greco and Di Vito, 1993), the root-knot nematode, which is more damaging in warm soils.

Biology and Crop Loss

There are two distinct races that commonly attack faba beans, the 'Giant' race and 'Oat' race. The giant race is tetraploid, with slightly longer adults (up to 2.0 mm); it is specialized, infesting mainly *V. faba* (Hooper, 1984) but also certain weeds including *Lamium album* and *Chenopodium album*. The oat race, on the other hand, has a wide host range including oats, onions, rye, peas, maize, strawberry and several weeds. The races of *D. dipsaci* that attack red clover and lucerne can reproduce within faba beans but do not cause much damage. The oat race is indicated by reddish-brown coloration of the host stems starting at the base and stopping at a leaf node. Heavy infestations cause thin, brown distorted stems and

shortened internodes. The giant race induces more severe distortion and twisting of the stem including the top of the plant, similar to swellings and distortions of the red clover race in red clover plants. Both races can infest seed giving necrotic patches under the testa, the seed sample being blemished, darkened or cracked.

Large populations of nematodes can build up within faba bean plants, with up to 100,000 individuals per stem (Hooper, 1971), returning to the soil in plant debris. Both races persist in the soil, in the absence of host crops, for at least 10 years (Hooper, 1984), and also survive on certain weeds. The disease can spread in bean seed and undoubtedly seed has infested previously clean land. The oat race is adapted to cool conditions and is found more often in northern Europe whereas the giant race is more common in the warmer Mediterranean region, surviving also in central Europe.

Hooper (1991) reported a yield decrease of 26% by the oat race and 58% by the giant race in plots with infested straw. Normally, infestations are only very damaging if dense populations are allowed to build up. However, any detectable level of infestation prevents the crop from being used for seed. The faba bean crop was severely infested in Morocco recently, eliminating the export trade (Robertson and Saxena, 1993). Like broomrape, *Sclerotinia*, foot or root rots and wilt, stem nematode limits cropping to long rotations.

Management

A rotation with infrequent oats, onion and faba beans helps prevent build-up of the oat race, and *Vicia faba* should not be grown more than one year in six (less often on heavy soils) even if only the giant race is involved. Care should be taken not to allow debris from infested crops on to adjacent fields. Seed should be tested and only used if free of stem nematode.

Methyl bromide is used to fumigate clover, lucerne and onion seed, but for faba beans there is too narrow a margin between efficacy and seed toxicity. Aldicarb and carbofuran were successfully used as seed and soil treatments in spring beans but not winter beans (Whitehead and Tite, 1987). The cost is only justified for valuable seed crops (Hooper, 1991).

There have been some reports of resistant cultivars. Twelve ICARDA lines were found to have moderate to high resistance in Syria (Robertson and Saxena, 1993) and 11 also in Tunisia (Hanounik *et al.*, 1986). Caubel and Leclercq (1989) confirmed some resistance in BPL 1696, BPL 1827 and FLIP 84–154 but only the INRA line 29H was very resistant to the giant race (Caubel and Le Guen, 1992). This resistance is controlled by a single gene inherited maternally through cytoplasm of 29H, which also has resistance to *Ascochyta fabae*.

CONCLUSIONS

Many of the diseases of faba bean have not been researched extensively; there is still a poor understanding of some aspects of the epidemiology, and control methods have not been optimized. A sound knowledge of the life cycle of a disease is

essential for effective control strategies to be formulated. For example, if the teleomorph of *Ascochyta fabae*, which is windborne, proves to occur frequently on bean straw, the current emphasis on healthy seed as the major means of controlling the disease may need to be re-evaluated.

The importance of infected plant debris in the carry-over of diseases from one crop to another also needs to be re-examined, particularly as debris can now remain on fields longer after harvest with current farming practices. In the UK, straw burning has now been banned and in some areas minimal cultivation has become popular. Clearly, these changes will have an influence when crops are grown adjacent to infected straw but for some diseases longer-distance spread is not clearly understood. For example, although the aerially dispersed teleomorph of the ascochyta blight pathogen has now been described, it is not yet known how frequently it occurs or how significant it is in the epidemiology of the disease.

In many areas, beans are grown as a low-input crop and fungicides are uneconomic. Also, the relatively small area of the crop in western Europe has meant that pesticide manufacturers have been reluctant to invest in the research and development of new pesticides or in some cases even to do the necessary tests to permit recommendation of existing products. In such a situation, disease resistance and other non-chemical control measures assume greater importance.

Relative to other arable crops, disease resistance breeding in faba beans is of recent origin. However, considerable progress has been made for a number of diseases. Of the major diseases, high resistance to chocolate spot has proved to be the most elusive. Although sources of resistance have been identified, these have been in lines which are poorly adapted for western Europe and have complex inheritance, making transfer of resistance to winter-hardy genotypes difficult. Furthermore, current screening methods for resistance to chocolate spot are not very reliable, at least partially due to the nature of the resistance and the strong interaction between genotype and environment.

Having achieved high resistance to a number of diseases, it is now important that these are combined to give comprehensive resistance to diseases of importance in a particular area and also to ensure that resistant cultivars also have the agronomic traits and quality components necessary for beans to be used to optimal advantage in both food and feed. There are indications that such combinations may not always be easy; for example cultivars low in tannin tend to be more susceptible to infection by *Fusarium* spp. However, a recent major programme sponsored by the European Commission has provided breeding material containing resistance to a range of diseases despite low tannin or low vicine and convicine.

Durability of disease resistance remains largely untested, because of the relatively recent development of resistant cultivars. It is known that some sources of resistance to some diseases, for example rust, are highly race-specific and that others have proved effective over a range of locations and several years, but generally only in small-scale trials. Durability can only be proven by growing cultivars on a large area for many years.

In many crops, there is now considerable interest and research effort being devoted to transgenic research, and novel techniques for disease control are

being developed. Currently progress has been most rapid for virus diseases. However, faba beans are proving to be difficult to transform, although recently a protoplast regeneration system has been developed (T. Pickardt, personal communication).

Integrated control measures for a number of diseases are being investigated and such an approach should bring considerable benefits to both the crop and the environment. There have already been encouraging results from studies involving resistant cultivars, herbicides and planting dates for controlling *Orobanche*.

In order to make rapid and sustained progress in faba bean research, a coordinated international effort is required; particularly as in many countries the national resource is low. In recent years, there has been encouraging progress in collaborative research and this needs to continue and be strengthened, particularly in areas such as collecting, evaluating and maintaining genetic resources, and in molecular biology.

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DISEASES OF LENTIL

8

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INTRODUCTION

Lentil (*Lens culinaris*) is a short, annual cool-season food legume. Its seeds are a source of good quality protein for the human diet and its straw is a valued animal feed in West Asia. The putative progenitor of the cultivated lentil is *Lens orientalis*, which is distributed from Greece to Uzbekistan (Cubero, 1981). From its Near East origins, the lentil has now spread in the Old World to be cultivated in a band stretching from Morocco to Bangladesh, with India and Turkey as the two largest producing countries (Table 8.1). The crop has been introduced to Australia, Canada, New Zealand and the USA. The world production stands at 2.6 million metric tonnes from an area of 3.3 million ha (1991–1993) (FAO, 1995).

Recent reviews have assessed the relative economic importance of key biotic stresses affecting lentil (Saxena, 1993; Johansen *et al.*, 1994). The most serious biotic constraints facing the crop are the foliar diseases, ascochyta blight, rust, stemphylium blight and grey mould. Rust is a key yield reducer in Morocco and Ethiopia and is important also in India. Ascochyta blight is important in Canada and in wetter parts of South Asia. Fusarium wilt and collar rot are also important universally; the former is more important in dry areas where foliar diseases are of minor importance and the latter is more prominent under humid conditions. The parasitic angiosperm weed, broomrape, is a major threat to lentil production in parts of the Mediterranean region.

In this chapter research on the above key diseases is reviewed. Colour plates of major lentil diseases are in Beniwal *et al.* (1993). Information on other fungal diseases of lesser economic importance than those already covered is to be found in Table 8.2. Bacterial diseases are unimportant on lentil. There is a report of a bacterium causing root rot in the former USSR (Javornokova, 1932) and lentil is reported as a host of *Xanthomonas campestris* pv. *phaseoli* (Kore and Shirshikar, 1980). The lentil crop is affected by a number of virus diseases worldwide (Bos *et al.*, 1988; Makkouk *et al.*, 1992), some of which have the potential to adversely

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Table 8.1. Area (x1000 ha) and production (1000 t) of lentil worldwide and in the ten major lentil producing countries, ranked in descending order of the 1991–1993 production data (FAO, 1995).

	Area (x1000 ha)		Production (1000 t)	
Country	1979–1981	1991–1993	1979–1981	1991–1993
World	2218	3298	1327	2642
India	934	1183	411	811
Turkey	206	747	219	655
Canada	38	279	32	331
Bangladesh	290	209	171	158
China	n.a.	72	n.a.	98
Iran	58	139	45	86
Nepal	98	134	49	84
USA	78	53	84	79
Syria	82	92	62	73
Ethiopia[1]	51	50	45	35

n.a. Not available.
[1] In 1991–1993, former Ethiopia was divided into Eritrea and PDR Ethiopia; 90% of lentil production is in PDR Ethiopia.

affect lentil seed yield and quality. The major virus diseases of lentil are listed in Table 8.3. Earlier reviews of lentil pathology are to be found in Khare (1981) and Diaz Moral (1993). Information on different species of nematode reported from lentils is presented in Table 8.4.

RUST

Causal Pathogen

The disease is caused by *Uromyces viciae-fabae* (Pers.) Schroet. (*Pucciniaceae, Uredinales*). It is an autoecious fungus, completing its life cycle on lentil. Spermagonia are subepidermal and globoid. Aecia are subepidermal in origin, erumpent later. Aeciospores are elliptical, yellowish-brown, measuring 14–22 μm in diameter and with a finely warty wall. Uredia are first subepidermal, then erumpent. Uredospores are borne singly on pedicels, mostly echinulate, with three to four germination pores and measure 22–28 × 19–22 μm. Telia are subepidermal in origin, then erumpent on leaves but remain covered by the epidermis on stems for an extended period. Teliospores, borne singly on pedicels, are globose to subglobose, very rarely ellipsoid or ovoid, one-celled, measuring 25–40 × 18–26 μm, with a single germination pore; the wall is obviously pigmented (Viennot-Bourgin, 1949).

Table 8.2. Minor fungal diseases of lentil.

Disease	Causal fungi	Distribution and references
Above-ground		
Powdery mildew	*Erysiphe polygoni* DC and/or *Leveillula taurica* (Lév.) Arnaud	Bangladesh (24), Chile (33), Cyprus (17), Ethiopia (16), India (6), Jordan (28), Spain (40), Sudan (25), Syria (36), former USSR (2)
Downy mildew	*Peronospora* sp.; *Peronospora lentis* Gäumann	Egypt (32), France (1), India (7), Syria (23), Turkey (35)
Anthracnose	*Colletotrichum truncatum* (Schwein) Andrus & Moore; *C. trifolii* Bain & Essary	Canada (38), Ethiopia (5), Pakistan (34), Syria (26), USA (43)
Leaf spot	*Alternaria alternata* (Fr.) Keissler	Bangladesh (37), Canada (10), Ethiopia (42), India (3), Jordan (28), Turkey (41), USA (4)
Cercospora leaf spot	*Cercospora lensii* Sharma, Mishra & Jain; *C. zonata* Wint.	India (14); Mauritius (8)
Cylindrosporium leaf spot	*Cylindrosporium* sp.	Syria (23)
Septoria leaf spot	*Septoria* sp.	Syria (23)
Mycosphaerella leaf spot	*Mycosphaerella* sp.	Syria (23)
Phoma leaf spot	*Phoma medicaginis* Malbr. & Roum. var. *pinodella* (Jones) Boerema	Syria (23), Turkey (44)
Leaf mould	*Cladosporium herbarum* (Pers.) Link	Canada (10), USA (4)
	Ulocladium atrum G. Preuss.	Turkey (35)
	Helminthosporium sp.	Turkey (35)
Sclerotinia stem and root rot	*Sclerotinia sclerotiorum* (Lib.) de Bary	Worldwide (15)
Root rots		
Rhizoctonia root rot	*Rhizoctonia solani* Kühn (teleomorph: *Thanatephorus cucumeris* (Frank) Donk.)	Worldwide (21), especially on irrigated lentils

Continued overleaf

Table 8.2. Continued.

Disease	Causal fungi	Distribution and references
Dry root rot, charcoal rot	*Macrophomina phaseolina* (Tassi) Goid.	Worldwide (21). On lentil: Ethiopia (42), India (15), Nepal (9), Syria (26)
Black root rot	*Thielaviopsis basicola* (Berk. & Br.) Ferr.	USA (29)
Pythium root rot	*Pythium aphanidermatum* (Edson) Fitzp.; *P. ultimum* Trow. (most commonly reported)	Iran (19), Syria (23), Turkey (18), USA (20,39)
	P. butleri Subramanian;	Egypt (39)
	P. irregulare Buis.	USA (39)
Ozonium root rot and wilt	*Ozonium texanum* Neal & Wester var. *parasiticum* Thirum.	India (22)
Fusarium root rot	*Fusarium solani* (Mart.) Appel & Wr.	Canada (27), India (13)
	Fusarium roseum 'avenaceum'	USA (12)
	F. roseum 'gibbosum'	USA (4)
Aphanomyces root rot	*Aphanomyces euteiches* Drechsl.	Canada (31), USA (30)

1. Viennot-Bourgin (1949). 2. Golovin (1956). 3. Sengupta and Das (1964). 4. Wilson and Brandsberg (1965). 5. Stewart and Dagnachew (1967). 6. Sankhla *et al.* (1967). 7. Beniwal and Srivastava (1968). 8. Anonymous (1971). 9. Manandhar (1975). 10. McKenzie and Morrall (1975). 11. Abdel-kader (1977). 12. Lin and Cook (1977). 13. Shukla and Bhargrava (1977). 14. Sharma *et al.* (1978). 15. Khare *et al.* (1979). 16. Mengistu (1979). 17. Photiades and Alexandrou (1979). 18. Soran (1977). 19. Kaiser and Horner (1980). 20. Kaiser (1981). 21. Khare (1981). 22. Shukla (1982). 23. Bellar and Kabbabeh (1983). 24. Fakir (1983). 25. Sarrag and Nourai (1983). 26. Bellar (1984). 27. Bhalla *et al.* (1984). 28. Mamlouk *et al.* (1984). 29. Bowden *et al.* (1985). 30. Kraft *et al.* (1985). 31. Lamari and Bernier (1985). 32. Abou-Zeid (1987). 33. Sépulveda (1987). 34. Bashir and Malik (1988). 35. Sagir (1988). 36. Bayaa (1989). 37. Fakir and Rahman (1989). 38. Morrall (1988). 39. Ingram and Cook (1990). 40. Diaz Morall (1993). 41. Karahan and Katircioglu (1993). 42. Mengistu and Negussie (1994). 43. Venette *et al.* (1994). 44. Beniwal *et al.* (1995).

Table 8.3. Virus diseases of lentil.

Disease	Causal agent	Legume host[1]	Manner of transmission	Morphology	Distribution and references[2]
Lentil yellows	Bean leaf roll luteovirus (BLRV)	P,L,C,F	Aphids (persistent) only	Sphere (25–28 nm)	NA, EU, AF, Asia (Bos *et al.*,1988)
	Beet western yellows luteovirus (BWYV)	P,L,C,F			
	Subterranean clover red leaf luteovirus (SCRLV)	P,L,C,F			Only in Aust. and NZ
Yellow mosaic	Bean yellow mosaic potyvirus (BYMV)	P,L,C,F, others	Sap, seeds[3], aphids (non-persistent)	Flexuous and rod-shaped (750 nm)	Worldwide (Bos *et al.*, 1988)
Pea seedborne mosaic	Pea seedborne mosaic potyvirus (PSbMV) = Pea leaf rolling virus = Pea seedborne mosaic virus = Pea fizzle top virus = Pea leaf roll virus = Pea leaf rolling mosaic virus	P,L,C,F, *Vicia villosa*	Sap, seeds[3], aphids (non-persistent)	Flexuous and filamentous (680–900 nm)	NA, AF, Asia, Aust. and NZ (Inouye, 1967; Kvicala and Musil, 1967; Hampton, 1969; Bos, 1970; Musil, 1970)
Pea enation	Pea enation mosaic enamovirus (PEMV)	P,L,C,F	Aphids (persistent), sap	Sphere (28 nm)	NA, EU, Asia (Aydin *et al.*, 1987)
Pea streak	Pea streak carlavirus	P,L,C	Aphids (non-persistent)	Filamentous (619–630 nm)	NA, EU (Bos *et al.*, 1988)
Broad bean stain	Broad bean stain comovirus (BBSV)	P,L,F, vetch	Sap, seeds[3], snout beetles	Isometric (28 nm)	NA, EU, W. Asia, Aust and NZ (Lloyd *et al.*, 1965; Fisher and Lockhart, 1976; Tolba, 1980; Makkouk *et al.*, 1987; Bos *et al.*, 1988; Fortass and Bos, 1991)

Continued overleaf

Table 8.3. Continued.

Disease	Causal agent	Legume host[1]	Manner of transmission	Morphology	Distribution and references[2]
Cucumber mosaic	Cucumber mosaic cucumovirus (CMV)	P,L,F, common bean and non-legumes	Sap, aphids (non-persistent), seed[4]	Sphere (30 nm)	Wordwide (Bos *et al.*,1988). On lentil: Iran (Kaiser, 1973), Pakistan (Bashir *et al.*, 1994)
Faba bean necrotic yellow	Faba bean necrotic yellow (ungrouped) virus (FBNYV)	P,L,C,F	Aphids (persistent)	Isometric (18 nm)	Egypt and Spain (Makkouk *et al.*, 1992; Katul *et al.*, 1993)
Alfalfa mosaic	Alfalfa mosaic alfamovirus (AMV)	P,L,C,F and non-legumes	Sap, aphids (non-persistent)	Bacilliform (28–58 nm)	Worldwide (Bos *et al.*, 1988)
Tomato spotted wilt	Tomato spotted wilt tospovirus	L	Sap	–	Brazil (Fonseca *et al.*, 1995)

[1] P = Pea, L = lentil, C = chickpea, F = faba bean.
[2] NA = North America, EU = Europe, AF = Africa, Aust. = Australia, NZ = New Zealand.
[3] Seed transmission reported in lentil.
[4] Seed transmission reported but not in lentil.

Table 8.4. Nematodes reported to infect lentil.

Nematode	Distribution and references
Heterodera ciceri Volvas & Di Vito	Syria (Greco *et al.*, 1988)
Ditylenchus dipsaci (Kühn) Filipjev	Syria (Greco *et al.*, 1984, 1988)
Pratylenchus thornei Sher. & Allen	Syria (Greco *et al.*, 1984)
Meloidogyne sp.	India (cited in Khare, 1981)
Tylenchorhynchus sp.	Syria (Greco *et al.*, 1988)

Biology

Rust is a widespread foliar disease of lentil. It occurs in Algeria (Khare, 1981), Argentina (Bascur, 1993), Bangladesh (Talukdar, 1974), Bulgaria (Christoff, 1939), Canada (Conner and Bernier, 1982), Chile (France and Tay, 1987), Colombia (Bascur, 1993), Cyprus (Nattrass, 1932), Ecuador (Bascur, 1993), Egypt (Rajab *et al.*, 1986), Ethiopia (Stewart and Dagnachew, 1967), India (Prasad and Verma, 1948), Iran (Scharif and Ershad, 1966), Italy (Canonaco, 1937), Jordan (Mamlouk *et al.*, 1984), Morocco (Malencon, 1936), Pakistan (Bashir and Malik, 1988), Palestine (Rayss, 1937), Peru (Bascur, 1993), Portugal (De Souza da Camara *et al.*, 1939), Nepal (Manandhar, 1975), Syria (Hanounik, 1979) and Turkey (Bremer *et al.*, 1947). It is economically important in such countries as Bangladesh, Chile, Ecuador, Ethiopia, India, Morocco, Nepal and Pakistan.

Accatino (1963–1964) and Plaza de Los Reyes (1964) reported that the rust affecting lentil may attack faba bean (*Vicia faba* L.) (see Jellis *et al.*, Chapter 7, this volume). Additionally, Singh and Sokhi (1980) identified six pathotypes on the basis of their differential reactions on cultivars of lentil, pea (*Pisum sativum* L.) and sweet pea (*Lathyrus odoratus* L.). Laundon and Waterston (1965) reported that the fungus may attack species of *Lathyrus*, *Lens*, *Pisum* and *Vicia*.

In summary, *Uromyces viciae-fabae* has some degree of host specialization with some forms restricted to a single host species and others having a wider host range, often including several genera (Gaunt, 1983). However, in some countries, such as Morocco, the crops faba bean, pea and lentil are sympatric; clearly, research in this area is required. Discussion on host specialization of rust in the cultivated lentil is discussed under 'Management'.

Symptoms

Rust starts with the formation of yellowish-white pycnidia and aecial cups on leaflets and on pods, singly or in small groups in a circular form (Beniwal *et al.*, 1993). Later, brown uredial pustules, oval to circular and up to 1 mm in diameter, develop on either surface of leaflets, branches, stem and pods. They may coalesce to form larger pustules (Plate 17).

The telia, which are formed late in the season, are dark brown to black, elongated and present mainly on branches and stems. At a certain moment, the three stages, aecia, uredia and telia, are present in the centre of the field: uredia and

aecia in the intermediate areas and aecia on plants at the border of the field. The plants dry following the same pattern, leading to the appearance of circular brown patches of dry plants in the field (Khare, 1980).

In severe infections, the affected plant dries without forming any seeds in pods or with small shrivelled seeds. The plant has a dark brown to blackish appearance, visible in affected patches of the field or in the whole field if it is totally infected.

Epidemiology

The disease first occurs during the flowering/early podding stage. In South Asia, lentil rust occurs mostly in January–February, in the form of pycnia and aecia. Aeciospores germinate at 17–22°C and infect other plants forming either secondary aecia or uredia at 25°C. Teliospores remain viable on faba bean over a wide range of temperatures and germinate readily (Gaunt, 1983). At lower temperatures, uredospores are probably an important means of survival in the absence of the host. Uredomycelium is, on the other hand, highly resistant to heat and sunlight and is probably important for continued development and survival of rust in hot, dry conditions. The predominant form of survival, therefore, varies with the environment and location. Teliospores germinate at 17–22°C without a resting period and cause outbreaks of the disease.

The slow build-up of epidemics may be due to the slow infection rate. Williams (1978) reported rates of infection of between 0.136 and 0.077 calculated as $\log_e x(1\text{-}x)$ on faba bean (van der Plank, 1963).

The disease generally starts from low-lying patches in the field and radiates towards the border (Khare and Agrawal, 1978). Uredosori develop late in the season and are rapidly followed by telia. After harvest, aecia and uredia present on the plant die out, but teliospores resist the heat. The fungus survives the summer as teliospores. It is also carried with seed as concomitant contamination (Richardson, 1979). It may also perpetuate on weed hosts (*Lathyrus* and *Vicia* spp.) from where it may infect lentil by windborne spores. High humidity and cloudy or drizzly weather with temperatures of 20–22°C favour disease development (Khare, 1981).

Damage and Crop Loss

As stated earlier, the crop is vulnerable to infection at all stages of its growth but is most susceptible at flowering (Accatino, 1963–1964). Damage to the crop depends upon the stage at which it is attacked, the cultivar and severity of the attack (Sépulveda, 1985). Yield losses of 60–69% have been reported in India and Chile (Sépulveda 1985; Singh *et al.*, 1986). Early infection accompanied by conducive environmental conditions can result in complete crop failure, as observed in Morocco (Sakr, 1990). Singh *et al.* (1986) reported a yield loss of 11.5 kg ha^{-1} for every 1% increase in disease intensity.

Management

Adjusting the sowing date may help reduce losses from rust. The early-sown crop in India is more affected by the disease than the late-sown one (Singh and Dhingra, 1980), whereas the converse is true in some other countries such as Russia (Cajlevic, 1963).

It is advisable to use clean seed without concomitant contamination (Cajlevic, 1963). Also, it is recommended to burn or bury diseased crop residues after harvest in order to reduce or eliminate the inoculum (Canonaco, 1937; Prasad and Verma, 1948). US Federal regulations prohibit the importation of seed of *Lens* spp. from South America because of the threat posed by the fungus (Anonymous, 1959).

Seed treatment with phenyl mercuric acetate (Agrosan) is reported to eliminate the inoculum from the seed (Prasad and Verma, 1948), but being a mercury compound, Agrosan is incompatible with *Rhizobium*. Accatino (1963–1964) reported that the following were the most effective fungicides in controlling lentil rust: wettable sulphur 2%, zineb (Dithane Z-78) 2% and nickel nitrate 0.5%. Singh (1984) reported that wettable sulphur spray eliminated lentil rust. Lentil grown from seed treated with diclobutrazol (Vigil) is reported to remain free from rust infection for up to 60 days after sowing, while the control crop was infected severely after only 35 days (Singh, 1985). Spraying the crop with zineb (Dithane M 45) at 2500 ppm (3 l ha^{-1}) at an interval of 10–12 days from the initiation of the disease, controlled the disease effectively and increased the yield by 82% (Agrawal *et al.*, 1976a; Singh *et al.*, 1985). Sépulveda and Alvarez (1989) stated that triademfon (Bayleton) + propineb (Antracol) at 0.5 + 2 kg ha^{-1}, applied as foliar spray controlled the disease. However, the application of fungicides on a large scale, on a crop such as lentil, is neither environmentally nor economically sound.

The use of host plant resistance is the best means of rust control. Several resistant lines are available in different parts of the world. Examples include the following: BARI Masur 2 in Bangladesh (ICARDA, 1995); Centinela-INIA (ILL 5540) (Bascur and Sépulveda, 1989) in Chile; INIAP-406 (ILL 5764) in Ecuador (INIAP, 1988); NEL 358 (ILL 358) (Million and Beniwal, 1988), Chikol (ILL 2704), FLIP84–7L (ILL 5680), Gudo (ILL 5748) and Ada'a (ILL 6027) in Ethiopia (Bejiga and Anbessa, 1994); Precoz (ILL 4605) in Morocco and Manserha'89 (ILL 4605) in Pakistan (ICARDA, 1994). The international testing of lentil genotypes differing in resistance to rust has shown that the reaction to lentil rust of individual lines is the same across the Old World. However, on pea the fungus exhibits a high degree of physiological specialization and nine races have been reported on the basis of a differential set of cultivars (Kispatic, 1949). Although the resistance on lentil is now holding, it is likely to break down.

In India, screening for rust resistance has been conducted at several centres (Jabalpur, Kanpur, Pantnagar and Faridkot in Punjab) under natural epiphytotic conditions. Cultivars with resistance to rust include the following: L 9–12, T 36, Bombay 18 (Nene *et al.*, 1975), Pant L 236, Pant L 406 (Pandey, 1981), and NP 47 and T 36 (Mishra *et al.*, 1985), Pusa 10 (Khare and Agrawal, 1978) and HPL

5 (Singh and Sandhu, 1988). Resistant cultivars were reported to have more surface wax, phenol, P, K, S, Zn, Fe and Cu, whereas susceptible cultivars had higher leaf permeability and a higher amino acid content, protein N, sugars and Mn (Reddy and Khare, 1984). Resistance to rust in lentil is reported to be conferred by a single dominant gene (Sinha and Yadav, 1989; Singh and Singh, 1990).

ASCOCHYTA BLIGHT

Causal Pathogen

Ascochyta blight is caused by *Ascochyta fabae* Speg. f. sp. *lentis* Gossen *et al.* (*Sphaerioidaceae, Sphaeropsidales*). The perfect stage of the fungus is reported to be a *Didymella* sp. (Kaiser and Hellier, 1993). *Phoma medicaginis* Malbr. & Roum. var. *pinodella* (Jones) Boerema and *Mycospherella* sp. also infect lentils (Table 8.2) but these are clearly distinct from ascochyta blight.

Aerial mycelium is substantially reduced on culture media, but tends to increase with subculturing (Gossen *et al.*, 1986). The fungus produces its spores in a flask-shaped fruiting body (pycnidium). Pycnidia are globose to subglobose, dark, ostiolate, and 75–225 μm in diameter (Khan *et al.*, 1983). Conidia are cylindrical and hyaline with 1, 2 or 3 septa, with uni-septate spores predominating. Conidia ranged from 10–20 × 3–6 μm, averaging 13.1 × 3.8 μm when grown on oatmeal agar (Sattar, 1933; Grewal, 1988) and averaged 14.7 × 4.1 μm when isolates from Canada were grown on V8 agar (Morrall and Sheppard, 1981).

Biology

Ascochyta blight has been reported in most lentil-producing countries, including Argentina (Mitidieri, 1974), Australia (Luig *et al.*, 1982), Brazil (Veiga *et al.*, 1974), Canada (Morrall and Sheppard, 1981), Chile (Sépulveda and Alvarez, 1982), where it is considered as the most important limitation especially in rainy seasons (Tay and Kramm, 1984), Cyprus (Photiades and Alexandrou, 1979), Ethiopia (Seid and Beniwal, 1988), Greece (Davatzi-Helena, 1980), India (Khatri and Singh, 1975), Iran (Ahmadinejad, 1991), Jordan (Mamlouk *et al.*, 1984), Morocco (S.P.S. Beniwal, Morocco, 1991, personal communication), New Zealand (Cromey *et al.*, 1987), Pakistan (Khan *et al.*, 1983), Russia (Voluzneva and Golubev, 1982), Spain (Diaz Moral, 1993), Syria (Hanounik, 1979), Turkey (Sagir, 1988) and the USA (Kaiser and Hannan, 1987). In addition, this pathogen has been isolated from seeds originating in Australia, Canada, Chile, Ethiopia, Greece, Hungary, India, Italy, Morocco, Pakistan, Russia, Spain, Syria, Turkey and the former Yugoslavia (Kaiser and Hannan, 1982; Kaiser, 1983; Kaiser and Hannan, 1986). It is of economic importance in Australia, Canada, Chile, Ethiopia, India, New Zealand and Pakistan. Ascochyta blight affects common bean, cowpea, pea, faba bean and chickpea, as reviewed in Chapters 4, 5, 6, 7 and 9.

Two matings types (1 and 2) are known for the fungus (Kaiser and Hellier,

1993). Gossen *et al.* (1986) examined the range of variability of cultural and morphological characters of 68 isolates of *Ascochyta fabae* f. sp. *lentis* from 12 countries and 13 isolates of *A. fabae* from Canada and used multivariate analysis to delineate different isolate groups. No differences in cultural and morphological characters were found among the isolates tested. They further reported that isolates were pathogenic only on the host species (*L. culinaris* or *V. faba*) from which they were originally isolated. Seid and Beniwal (1991) reported that the fungus, isolated from lentil seed, was only pathogenic to lentil and not to any of the seven other legume species tested. In addition, f. sp. *fabae* produces ascochytine but f. sp. *lentis* does not (Boerema, 1984). This suggests that the fungus exhibits a high degree of host specialization and indicates the inability of *A. fabae* f. sp. *lentis* to cross-infect the related food legumes, faba bean (see Jellis *et al.*, Chapter 7, this volume), pea and chickpea (*Cicer arietinum*). Although races of *A. fabae* f. sp. *lentis* have not been reported, the differential reaction of some lines of lentil in different countries suggests pathogenic variation in the fungus (ICARDA, 1992). Information on pathogenic variation could lead to a more efficient screening of lentil germplasm.

Symptoms

The above-ground parts of the host may be infected at different growth stages (Morrall and Beauchamp, 1988). Tan spots, surrounded by dark margins, are seen on the leaflets (Beniwal *et al.*, 1993). The centre of the spot is light coloured and speckled with tiny, black, fruiting bodies (pycnidia) that are characteristic of the disease (Fig. 8.1). Pycnidia form most readily within lesions of ageing leaves and under moist conditions. Pycnidia are numerous, prominent and concentrically arranged and may develop oozing spore masses under very humid conditions. Coalescing lesions lead to blight (Plate 18) and leaflet abscission. Stem lesions are elongated, sunken and darker in colour than leaf lesions (Fig. 8.2) with scattered pycnidia. Stems may break at the point of infection and lodging may occur.

The tips of branches wilt, turn brown and die. The crop then has a distinctly blighted appearance. Lesions on the pods are generally darker than those on the leaves. The infected areas often have a purplish hue after the pods ripen. Seeds from heavily infected plants become purplish-brown (Fig. 8.3), shrivelled and greatly reduced in size, which adversely affects their quality. Severely affected seeds may have whitish patches of mycelia and tiny, black, fruiting bodies. We have obtained the fungus from symptomless seeds.

Epidemiology

The fungus occurs in all parts of the infected seed. Testa and cotyledons have a higher level of infection than the plumule and radicle (Morrall and Beauchamp, 1988). The amount of infected plant debris, frequency of seed transmission, which varies over genotypes according to their resistance, and wet conditions are

Fig. 8.1. Characteristic concentric rings of pycnidia of *Ascochyta fabae* f. sp. *lentis* in leaf lesions (Photo: courtesy of CODIS, ICARDA).

factors determining the onset of ascochyta blight (Beauchamp *et al.*, 1986a). The frequency of transmission from infected seed to seedling is low, especially at moderate to high soil temperature. Frequent rainfall can cause a major epidemic of ascochyta blight in a lentil field if inoculum is present. The source of primary inoculum may be either infected seed or stubble. Conidia spread from infected stubble to plants and from plant to plant within a crop. Subsequent disease development occurs by transmission of the pathogen from seed to the epicotyl and random dispersal of conidia from infected stubble (Gossen and Morrall, 1986). Russell *et al.* (1987) reported 100% foliage infection in lentil plants grown from naturally infected seeds. The cold and wet conditions in New Zealand during crop establishment in the winter are possibly favourable to pathogen establishment on the slowly developing seedlings. Horizontal spread of ascochyta blight was often observed 10–30 m from the infected crop, but declined sharply at 50 m (Pederson *et al.*, 1993).

Long-distance spread of *Ascochyta fabae* f. sp. *lentis* is through sowing infected seed in previously disease-free areas. Infected seed provides the fungus with an important survival mechanism (Gossen and Morrall, 1981; Kaiser, 1987). Kaiser (1989) reported that the storage of infected lentil seeds for 4 years at 20, 5, −18, −160, and −196°C did not adversely affect the pathogenicity of the fungus. It survived for more than 3 years in infected pods and seeds at 4– 5°C or in a shelter outdoors, and for 1.5 years on the soil surface, but lost its viability within 29 weeks at a soil depth of 16 cm. The fungus was found to remain viable for more than 30 years (Kaiser and Hannan, 1986).

Fig. 8.2. Stem lesions in ascochyta blight often coalesce to girdle stems (Photo: courtesy of B. Bayaa).

Damage and Crop Loss

Ascochyta blight can cause poor plant stand, reduced seed yield, seed discoloration and reduced seed quality. Seed germination and seedling vigour are adversely affected by fungal infection by *Ascochyta* (Neergaard, 1977; Morrall and Sheppard, 1981; Harman, 1983; Sépulveda, 1985; Cromey *et al.*, 1987; Kaiser, 1989). Seed size was significantly correlated to the level of seedborne infection while stunting and poor vigour were the most apparent symptoms in lentil seedlings developed from naturally infected seeds (Kaiser and Hannan, 1986). Gossen and Morrall (1983) suggested that the destruction of photosynthetic area by leaflet lesions and by defoliation reduced the photosynthates available for seed formation and development, resulting in reduced seed yield. Malik (1983) in Pakistan reported 30–40% crop damage by the disease. Gossen and Morrall (1984) in Canada reported seedborne infection levels of 35–49% and

Fig. 8.3. Seed from plants heavily infected with ascochyta blight may be discoloured and bear white patches of mycelial growth (Photo: courtesy of B. Bayaa).

23–28% in artificially inoculated field plots of cv. Common Chilean and cv. Laird, respectively.

Seed discoloration, which results from pod infection, may develop both before and after the crop is swathed, especially if the swathe requires a long drying time due to limited air circulation. Rain during the interval between swathing and threshing favours spread of ascochyta blight and loss of seed quality. The commercial seed grade is dependent upon percentage natural colour. Yield losses and reduced seed grades due to ascochyta blight can cause a loss of more than 70% of potential income to the Saskatchewan farmer (Gossen and Morrall, 1984).

Management

An integrated approach has been recommended for the management of the disease. Components include crop rotation to reduce the spread of the pathogen

from infected lentil debris, use of disease-free seed or seed with a low level of infection, and use of seed treatments, foliar fungicides and early seeding to avoid wet weather at harvest (Morrall and Sheppard, 1981; Russell *et al.*, 1987). Morrall and Sheppard (1981) suggested testing seeds for the presence of seedborne infection prior to planting. Their recommendation is that seeds with zero to a low per cent seedborne infection with the pathogen should be used in seeding.

Many researchers have tried to use fungicidal seed treatments to prevent the establishment of the fungus in a lentil crop. Morrall and Gossen (1979) studied the effect of several such treatments and reported no effect on infection. Subsequently, Morrall and Beauchamp (1984) and France *et al.* (1987) indicated that some fungicides may increase seed germination and reduce the frequency of transmission from seed to seedling. Kaiser and Hannan (1987) reported that treating seed with thiabendazole greatly reduced seed transmission. Seed treatment with thiram reduced infection but did not control the disease effectively (Guerrero, 1987). In laboratory tests, seed treatment with thiabendazole (Tecto) and benomyl (Benlate) resulted in a significant reduction in the number of seeds giving rise to colonies of *A. fabae* f. sp. *lentis* and a significant increase in seed germination (Russell *et al.*, 1987). In a field trial, using highly infected seeds (71%) of cv. Titore treated with four fungicides at 3 g kg ^{1}, thiabendazole and benomyl treatment resulted in significantly greater field emergence than chlorothalonil and mancozeb. In 1994, Agriculture and Agri-Food Canada granted Crown (thiabendazole + carboxin) a temporary registration for the control of seedborne *A. fabae* f. sp. *lentis* infection in lentil (Andrahennadi, 1994).

Hot water and dry heat treatment at 55°C for 25 min and 70°C for 24 h, respectively, partially inhibited fungal growth from seed (Seid and Beniwal, 1991). In contrast, Kaiser and Hannan (1987) stated that treatment of infected seeds with aerated steam or hot water at 45–75°C for 30 min did not control *A. fabae* f. sp. *lentis*. Beauchamp *et al.* (1986b) studied the effects of foliar application of fungicides on disease severity, seed yield and percentage seed infection in cv. Chilean in inoculated plots. A single application at early bloom to early pod set with chlorothalonil, captafol, folpet and metiram provided the best protection, increased seed yield by 8–30% and decreased seedborne infection by 12%. Yield losses and seed infection were reduced by early application, as opposed to late application, but a second application may be necessary under extremely wet growing conditions.

In a 3-year field trial in Ethiopia, Seid and Beniwal (1991) reported that chlorothalonil, benomyl and a mixture of tridemorph and maneb provided the best disease control and highest yield. Iqbal *et al.* (1989) evaluated seven fungicides as foliar sprays on the susceptible cv. Masoor-85. Benomyl and chlorothalonil both at 0.2% controlled the disease effectively when applied three times. Benomyl increased yield by 80% in comparison with the untreated check.

Seedborne inoculum is more likely to introduce the pathogen to new areas, as compared to stubble-borne inoculum, which helps the pathogen become established early in the season on lentil plants in the immediate vicinity (Gossen and Morrall 1981; Bedi and Morrall, 1990). This implies the necessity of cultural control, including crop sanitation, as part of an integrated approach to ascochyta blight management.

By far the most economical and environmentally sound measure of disease management is the use of host plant resistance. Resistance to ascochyta blight has been reported from Canada (Tay, 1989), India (Singh *et al.*, 1982), New Zealand (Cromey *et al.*, 1987), Pakistan (Iqbal *et al.*, 1990) and International Center for Agricultural Research in the Dry Areas (ICARDA) (1984). Cultivars with resistance to ascochyta blight include Manserha '89 (ILL 4605) in Pakistan, Talya 2 (ILL 5588) in Lebanon (Abi Antoun *et al.*, 1990), CDC Redwing and CDC Matador in Canada (A.E. Slinkard, Canada, 1995, personal communication) and INIAP-406 (ILL 5764) in Ecuador (Villacis and Acuna, 1988).

The ICARDA wild lentil germplasm collection has been screened for ascochyta blight resistance (Bayaa *et al.*, 1994b). Twenty-four out of 86 accessions of *Lens culinaris* ssp. *orientalis* were resistant, as were 12 of 35 accessions of *L. culinaris* ssp. *odemensis*, 3 of 35 accessions of *L. nigricans* ssp. *nigricans*, 36 of 89 accessions of *L. nigricans* ssp. *ervoides*, and all three accessions of *Vicia montbretii*. Of resistant sources 64% were from Syria and southeastern Turkey.

According to Tay (1989) and Tay and Slinkard (1989), resistance to ascochyta blight in lentil is controlled by three major genes. They identified two dominant genes (Ral_2 and Ral_3) and one recessive gene (ral_1) in the line ILL 5588 lentil. The line ILL 5684 had two genes for resistance (Ral_2 and Ral_3), while cv. Laird had one recessive gene (ral_1) that was ineffective at the late-podding stage.

GREY MOULD

Causal Pathogen

The disease is caused by *Botrytis cinerea* Pers. ex Fr. (*Mucedinaceae, Hyphales*), which has a teleomorph: *Sclerotinia fuckeliana* (de Bary) Fuckel (= *Botryotinia fuckeliana* (de Bary) Whetzel) (*Sclerotiniaceae, Helotiales*).

In vivo, the fungus has many different but characteristic forms: mycelial, conidial, sclerotial and apothecial (ascospores). The mycelium is septate, cylindric, sometimes vesicular, brown, branched and measures 8–18 μm in diameter. The conidiophores are light brown, erect, septate, slightly ramified, tips of its ramifications slightly enlarged and bearing small pointed sterigmata. Conidia are hyaline, one-celled, ovoid or spherical, smooth, thin-walled, borne in a cluster, measuring 10–12 × 8–10 μm. These dimensions vary (9–12 × 7–10 μm) depending on environmental conditions and the host plant. The fungus produces another type of conidium, much smaller 'microconidia', usually on old colonies or in response to a prolonged period of unfavourable conditions (Urbasch, 1984). Their major function may be that of spermatization in the production of apothecia.

Under unfavourable conditions the fungus is capable of producing sclerotia, consisting of densely interwoven, brown, septate hyphae. Sclerotia are always small (2–4 × 1–3 mm), globose or oval: at first they are white in colour later becoming brown or black, with a very shiny surface, regularly and finely punctate. Sclerotia produced by *B. cinerea* vary depending on the isolate (Harrison, 1976).

The teleomorph, *S. fuckeliana*, has been shown to develop from sclerotia of *B. cinerea* (de Bary, 1886; Groves and Drayton, 1939). Polach and Abawi (1975) reported its occurrence on bean in New York, but it has not been reported on lentil. Apothecia are brown, most often on stalks. Asci are cylindrical, mixed with filiform paraphyses. Ascospores, generally eight in number, are oblong to elliptical, measuring 6–9 × 5–6 μm.

Biology

The disease has a worldwide distribution on lentil, occurring especially in Chile (France *et al.*, 1988), India (Mukerji and Bhasin, 1986), Nepal (NGLRP, 1989) and Pakistan (Bashir and Malik, 1988). Additionally, it has been reported on lentil from Canada (Beauchamp *et al.*, 1986a), Morocco (W. Erskine, 1991, unpublished observation), New Zealand (Cromey *et al.*, 1987), Syria (Bellar and Kabbabeh, 1983) and the USA (Wilson and Brandsberg, 1965).

B. cinerea is an ubiquitous, non-host-specific pathogen. Its host range comprises weeds, ornamentals, vegetables, field and fruit crops. Among legumes, hosts include chickpea (see Haware, Chapter 9, this volume), faba bean (see Jellis *et al.*, Chapter 7, this volume) and common bean.

Heterokaryosis as a source of genetic variability in *B. cinerea* was clearly demonstrated by obtaining morphologically distinct strains of the fungus from single spore culture (Menzinger, 1966, Lauber, 1971). In Italy, Saponario (1953) concluded that isolates of *B. cinerea* obtained from different areas of Italy could be divided into seven morphological races based on conidial size. Rewal and Grewal (1989) isolated *B. cinerea* from chickpea from different locations in India and divided the purified isolates into six groups based on variation in growth, formation of sclerotia, sporulation, size and shape of conidia and temperature requirement. They classified the six groups into five physiologic races based upon their reaction on a set of chickpea differentials, and concluded that physiologic specialization existed in *B. cinerea*. However, Harrison (1988) induced aggressive lesions by inoculating bean leaves with young conidia of several isolates of *B. cinerea* from raspberry canes. Furthermore, Paul (1929) demonstrated that although isolates of *B. cinerea* varied in their pathogenicity, there was 'no evidence of selective parasitism'. Clearly there is conflicting evidence from other crops on the level of host specificity, but there is no information in this regard on isolates from lentil.

Sclerotia may germinate in either of three different forms: as mycelium, common on synthetic media but rare under field conditions (Coley-Smith, 1980); apothecia, but apparently not on lentil; and conidiophores bearing conidia, most frequently observed in the field.

Symptoms

When seed is heavily infected, seedling blight may occur. Otherwise, symptoms first appear on flowers (Beniwal *et al.*, 1993). Especially in the morning, they are

covered with a dirty white mouldy growth of the fungus which kills them. If a favourable microenvironment (cool moist conditions and dense plant canopy) prevails, infection develops on lower leaves on which the characteristic white fungal growth is clearly visible. This results in the shedding of lower leaves. Later, the whole plant becomes infected and dries up. The infected portions of the stem are light brown or blanched and covered with a grey mouldy growth and rot at the plant crown. Infected pods fail to fill properly, rot, turn brown and are covered with mould (Fig. 8.4). Infected seeds may be discoloured and shrivelled. Affected plants may appear in patches that enlarge as the disease spreads.

Epidemiology

There are several sources of inoculum. The pathogen is reported to be seedborne on lentil (Richardson, 1979; Kaiser, 1981). Conidia are common in the atmosphere

Fig. 8.4. Grey mould on pods is characterized by the development of conidiophores and conidia (Photo: courtesy of B. Bayaa).

(Gregory and Hirst, 1957). However, the three main sources of inoculum are conidia from diseased host plants, saprophytic mycelium in plant debris and sclerotia.

Conidia germinate over a wide temperature ranging from 7 to 26°C (Doran, 1922), with an optimum *c.* 20–25°C and minimum between +1 and −1°C (Wilson, 1937; de Haas and Wennemuth, 1962). Spore germination is considerably reduced at low temperatures, and requires extended incubation. Conidial germination is also favoured by high relative humidity (95–100%). At humidity <93%, germination does not occur (Snow, 1949). Lower disease severity in the field may be due to reduced relative humidity often associated with warm weather (Harrison, 1988). The disease is severe when growth is luxurious, as in dense stands, with abundant weeds or wherever conditions provide a humid microclimate. Conidia retain germinability for approximately 1 month (Wilson, 1937). However, the percentage germination of spores from old cultures was lower and germ tubes shorter than from young ones (Singh, 1940). Brown (1922) reported that the germination of conidia of *B. cinerea* was inhibited at high spore concentrations. Masuta (1984) attributed this phenomenon to a heat-stable self-inhibitor of germination.

Mycelium, in plant debris, may survive for extended periods and produce conidia under humid conditions. Sclerotia are the main survival structure. They are highly resistant to adverse conditions and are produced over a wide range of temperature, light and culture media. Sclerotia may survive for long periods if they are not buried. They die more quickly in wet than dry soil (Townsend, 1952). Their survival was reported to be favoured by low temperature and decreased rapidly above 25°C (Nair and Nadtotchei, 1987).

It was shown on faba bean that prior infection with either bean yellow mosaic virus or bean leaf roll virus increased susceptibility to infection by *B. cinerea* (Omar *et al.*, 1986). The same phenomenon was observed in pea infected with pea leaf roll (Tinsley, 1959). Equivalent information is lacking for lentil. Epiphytic microorganisms may reduce germination of *B. cinerea* spores and infection by competing for nutrients on leaf surfaces (Blackeman and Brodie, 1977).

Damage and Crop Loss

Detailed information is unavailable on lentil. One author observed 50% crop loss at Parwanipur, Nepal (W. Erskine, 1995, unpublished results).

Management

Several agronomic practices are possible to avoid a dense canopy which is conducive to disease outbreak. These include adjustments to sowing date and rates, row spacing, fertilizer and weed control. Similar measures have been found to be successful in chickpea and are reviewed in detail by Haware (Chapter 9, this volume).

Seed treatment with fungicides such as benomyl or chlorothalonil (0.1–0.3%) can be carried out to minimize seedborne inoculum and seedling blight. Foliar sprays with synthetic fungicides are extensively used to manage the disease caused by *B. cinerea* on high-value crops. This has led to the development of strains resistant to the commonly used groups of fungicide (dicarboximides, diethofencarb and ergosterol biosynthesis inhibitors) (Elad *et al.*, 1992). However, the use of fungicides to control grey mould on lentil is uneconomic.

Several fungi, most of which are common soil inhabitants, can attack sclerotia. *Gliocladium virens* Bain, *Trichoderma viride* Pers., *T. harzianum* Rifai and a *Verticillium* sp. are particularly vigorous parasites of *B. cinerea* (Coley-Smith, 1980).

No information is available on resistant lentil cultivars. Systematic screening for host plant resistance should be started. However, experience with chickpea indicates that it may be difficult to find high levels of resistance to the disease (Porta-Puglia *et al.*, 1994; see Haware, Chapter 9, this volume). The most effective control will come from combining some of the above individual measures into an integrated control package.

STEMPHYLIUM BLIGHT

Causal Organism

The asexual stage of the causal organism is *Stemphylium botryosum* Wallr. (*Dematiaceae, Hyphales*). *Pleospora herbarum* (Pers. ex Fr.) Rab. (*Pleosporaceae*) is the sexual stage.

Conidiophores are short, arise singly or in groups and are aseptate and swollen at the apex. After a conidium is produced the end of the conidiophore grows out, producing new cells and a new conidium. The conidiophore may grow to considerable length and have a nodulose appearance. Conidia are olive brown, muriform and echinulate measuring 24–40 × 14–25 μm. Conidia are oblong with three to four septae and often constricted at the centre by a median cross-wall. Echinulations to fine warts are numerous over the surface.

Perithecia are globose, membranous and black and sometimes have a slender neck. Asci (183–267 × 27–37 μm) are oblong to clavate with outer and inner walls. Ascospores (32–48 × 12–21 μm) are elongate to ovate, characteristically with seven cross-walls and three to five longitudinal septa, and yellowish to brown in colour and muriform when mature.

Biology

Stemphylium blight has been reported on lentil from Bangladesh (RARS, 1981; Bakr and Zahid, 1987), Egypt (B. Bayaa, 1993, unpublished observation), Syria (Hanounik, 1979) and the USA (Wilson and Brandsberg, 1965).

The fungus has a wide host range that includes leguminous and non-leguminous crops (Smith, 1940; Wells *et al.*, 1956; Graham, 1957; Slade, 1961;

Tate, 1970). Specialized races of the fungus grow on the clovers and alfalfa (Smith, 1940). Tate (1970) showed that the race attacking lupin is different in temperature requirement from the one attacking lettuce. In some cases *P. herbarum* is considered as a simple saprophyte. It grows as a greyish or olivaceous rot, which later becomes black and produces spores in great quantity.

Symptoms

The disease first appears as small pin-headed light brown to tan coloured spots on the leaflets (Beniwal *et al.*, 1993). The spots enlarge rapidly, covering the entire leaf surface within 2–3 days. The foliage and twigs gradually turn dull yellow, giving a blighted appearance to the affected crop. The infected leaves are shed severely, leaving only the terminal leaves on the twigs. The twigs bend down, dry up and gradually turn ashy-white, but pods remain green. On careful observation, white mycelial growth is seen on the infected twigs.

Epidemiology

In Bangladesh, the pathogen initiates its infection when the ambient night temperature remains above 8°C, the mean day temperature rises above 22°C and the relative humidity inside the canopy is at least 94% (Bakr, 1993). In India, an average temperature of 18 ± 2°C, humidity above 50%, a mean of 7.7 h or less of daily sunshine and cloudy or foggy weather; all favour disease development (Sinha and Singh, 1993). The pathogen seems to be airborne: no report is available on its seedborne nature on lentil, either externally or internally.

Damage and Crop Loss

Preliminary studies have indicated that the disease can cause up to 62% yield reduction (Bakr, 1993).

Management

In Bangladesh, the application of iprodine (Rovral 50 WP) controlled the disease when sprayed three times at an interval of 7 days starting from the initiation of the disease (Bakr, 1993). Also, incidence of the disease was lowered significantly when sowing was delayed beyond 20 November, but the yield of the late-sown crop was drastically reduced because of poor growth and a heavy infection of rust.

Screening to identify sources of resistance is under way in Bangladesh by using highly susceptible spreader rows. Several genotypes have shown good tolerance over three consecutive growing seasons (Bakr, 1993).

COLLAR ROT

Causal Pathogen

The disease is caused by *Sclerotium rolfsii* Sacc. (*Corticaceae, Aphyllophorales*) (Mathur and Deshpande, 1968). The perfect state of the fungus is *Corticium rolfsii* (Sacc.) Curzi. According to Talbot (1973), the basidial state of *S. rolfsii* is a species of *Athelia* (*Athelia rolfsii* (Curzi) Tu & Kimbrough in *Corticiaceae*. The basidial stage is rarely found in nature. The mycelium is septate and hyaline, branching at acute angles. Well developed mycelium is in cord-like strands. The hyphae have clamps in the form of forks and hooks or H-like connections (Aycock, 1966). Newly developed mycelium is slender, lacking clamp connections, snow-white in colour with a silky lustre. Hyphal cells are binucleate to multinucleate. Basidia are obovoid, 7–9 × 4–5 μm, each bearing two to four sterigmata that bear basidiospores. Basidiospores are hyaline, smooth elliptical, apiculate and 3.5–5.0 × 6–7 μm (Mehan *et al.*, 1995).

Sclerotia are formed laterally from main hyphal strands (Willetts, 1971). They are at first white, becoming light brown to dark brown at maturity. They are subspherical, 0.5–1.0 mm in diameter, with their surface being finely wrinkled or pitted. The sclerotium is differentiated into rind and medulla.

Biology

Collar rot of lentil, also described as 'root rot' (Pavgi and Upadhyay, 1967), occurs in almost every region where lentil is cultivated, and is especially prevalent in warm areas where high soil moisture and temperature at the seedling stage of the crop are relatively high. The disease is economically important in some parts of North India and Bangladesh, where the crop is grown after rice, causing considerable death to seedlings in the early stages of plant growth (Ishaque and Talukder, 1967). It has been also reported on lentil in Ethiopia (Stewart and Dagnachew, 1967) and Pakistan (Bashir *et al.*, 1987).

The fungus has an extensive host range which comprises nearly 500 plant species, mainly *Compositae* and *Leguminosae*. It is the causal agent of stem, root and pod rot of groundnut (see McDonald *et al.*, Chapter 2, this volume). Graminaceous species are less susceptible (Punja, 1985).

The fungus survives in the form of sclerotia that remain viable in the soil for 2–3 years. They serve as the primary source of inoculum and are capable of initiating infection with or without an additional food base (Aycock, 1966; Punja, 1985). Sclerotia germinate following two forms: hyphal and eruptive (Punja and Grogan, 1981). Eruptive germination in soil is greatest at 21–30°C and is less common below 15°C or above 36°C. Germination is greatest at the soil surface and decreases with soil depth. This could in part be due to a response to light. Volatile compounds from re-moistened, undecomposed plant tissue stimulate germination and the growth of *S. rolfsii* (Beute and Rodriguez-Kabana, 1979; Punja and Grogan, 1981). Optimal growth occurs at low pH (Punja and Jenkins, 1984) and is markedly less above pH 8.0 (Aycock, 1966). Germination of sclerotia

can be inhibited by several means (applying fungicides and ammonium compounds, burying sclerotia deeply, altering the composition or activity of soil microflora and adding antagonistic microorganisms) and thereby limit disease incidence (Punja, 1985)

The fungus produces a number of extracellular enzymes including pectin methylesterase (Bateman and Beer, 1965), cutinase (Baker and Bateman, 1978), phosphatidase (Sellam *et al.*, 1982), arabanase (Cole and Bateman, 1969), galactanase, mannase and xylanase (van Etten and Bateman, 1969) and β-glucosidase (Shewale and Sadana, 1979). The production of cell-wall degrading enzymes in conjunction with oxalic acid would account, in part, for the extensive host range of the pathogen.

S. rolfsii is a variable fungus. Single basidiospore strains from parental field isolates show pronounced variability in morphological characteristics (Punja and Grogan, 1983), suggesting that field isolates may be heterokaryotic.

Symptoms

The pathogen infects the collar region of the plant, causing a yellowish-brown discoloration, and a rotting of tissue (Beniwal *et al.*, 1993). The young seedlings show damping-off symptoms. Plants infected at an advanced stage gradually turn pale, droop and dry. White feathery growth of the fungus, generally associated with dirty white to brown coloured, mustard-seed-like sclerotia, can be seen on infected plant parts, so prolonging the survival of the pathogen under unfavourable conditions. Sometimes the fungus may proceed downward causing root rot. Infected plants are easily pulled up as the root system is poorly developed and side roots are destroyed.

Epidemiology

Collar rot incidence may increase following periods of temperature and moisture fluctuation; cycles of drying and wetting have been reported to stimulate germination of sclerotia (Smith, 1972). The presence of an organic substrate for mycelial growth may enhance disease severity (Beute and Rodriguez-Kabana, 1979).

S. rolfsii survives well in the soil as sclerotia in the presence of sufficient organic matter even under adverse weather conditions (Khare *et al.*, 1979). The disease is favoured by high soil moisture with high temperature (25–30°C) and good sunshine after rains (Khare, 1981). Extensive plant-to-plant spread occurs in closely spaced crops. *In vitro* research showed that disease incidence can be directly or indirectly affected by non-target pesticides. However, the importance of such compounds in suppressing disease under field conditions has yet to be determined (Punja, 1985). Continuous rotation with crops susceptible to *S. rolfsii* may increase disease incidence. Temperatures above 50°C for an extended period are lethal to sclerotia (Porter and Merriman, 1983; Mihail and Alcorn, 1984), possibly by enhancing nutrient leakage accompanied by an increased microbial antagonism (Lifshitz *et al.*, 1983).

Mycelium survives better in sandy soils than in fine-textured soils (Chattopadhyay and Mustafee, 1977). Factors that increase nutrient leakage or predispose sclerotia to antagonism may accelerate their death. These include drying, heating, deep burial, exposing sclerotia to chemicals and inducing changes in the integrity of the sclerotial rind (Punja, 1985). The disease becomes severe if the stubble of the previous crop, such as paddy or sorghum, is left in the field.

Management

Collar rot in lentil can be reduced greatly by manipulating the sowing date so that the seedling stage does not coincide with high soil moisture and a temperature above 25°C. Under Jabalpur conditions, 15 October was found to be a suitable sowing date resulting in low seedling mortality and a high yield (Agrawal *et al.*, 1976b, 1986b). In Bangladesh, the incidence of the disease declined gradually when sowing was delayed until the first week of November (Fakir and Rahman, 1989). Further delay in sowing reduced the disease incidence greatly but curtailed the yield drastically. The disease is reduced considerably by the application of 60 kg K ha^{-1} (Prasad and Chaudhary, 1984) and calcium fertilizer (Punja *et al.*, 1986). Increased calcium levels in the tissue may partly offset the effect of oxalic acid and cell-wall degrading enzymes. There is controversy regarding the effects of nitrogenous fertilizer, with Fakir and Rahman (1989) reporting an increase of the disease in lentil and Punja *et al.* (1986) reporting a decrease in carrot. Crop rotation is unlikely to be an effective method of control in view of the wide host range of the fungus and its persistence on almost all types of crop debris. Soil organic amendments such as oat or maize straw have been found to limit disease incidence on lentil (Mehrotra and Claudius, 1972)

Shahid *et al.* (1990) evaluated the effect of several fungicides on mycelial growth and sclerotial production *in vitro*; captan and metalaxyl (Ridomil) at 10, 20, 50 μg ml^{-1} dosage rates were the most effective. The disease was partially controlled by seed treatment with thiram, captan or methyl arsenic sulphide (Rhizoctol) (Khare *et al.*, 1974), with guazatine (Fakir and Rahman, 1989), and with triadimenol and carboxin at 0.25% w/w of dry seed. Later, it was shown that the early-stage seedling mortality in lentil can be controlled best by treating the seed with combinations of fungicides such as thiram + pentachloronitrobenzene or thiram + carbendazim (Agrawal *et al.*, 1975). Mancozeb has also been found effective (Singh *et al.*, 1985).

Trichoderma viride Pers. ex Fr., *Streptomyces gougeroti* (Duche) Waskman & Henrici and some bacterial species were reported antagonistic to *S. rolfsii*, isolated from lentil (Mehrotra and Claudius, 1972). *Trichoderma harzianum* Rifai and *Bacillus subtilis* Cohn emend Prazmowski were found to be antagonistic to *S. rolfsii* and also their application with seed or soil controlled collar rot in pot culture (Agrawal *et al.*, 1977). Similarly, Mukhopadhyay *et al.* (1989) reported the control of collar rot in lentils by using *T. harzianum* or *B. subtilis* applied to seeds with a 2% solution of *Gur* (molasses); the collar rot of lentil was reduced by about 80% over that of the control.

The use of resistant cultivars provides good control of this soilborne disease. Several resistant lines have been identified in Bangladesh (BARI, 1986), India (Kannaiyan and Nene, 1976; Khare *et al.*, 1979; Mohammad and Kumar, 1986) and in Pakistan (Anonymous, 1988).

VASCULAR WILT

Causal Pathogen

Several species of *Fusarium* have been found associated with the wilted lentil plant (Khare *et al.*, 1979) but in India *Fusarium orthoceras* App. and Woollen. var. *lentis* Vasudeva and Srinivasan was reported to be the cause of the disease (Vasudeva and Srinivasan, 1952). Later, the name of the fungus was changed to *F. oxysporum* Schlecht. ex Fr. f. sp. *lentis* Vasudeva and Srinivasan (*Tuberculariaceae, Hyphales*) by Chattopadhyay and Sengupta (1967).

The fungus produces three types of spore: oval or kidney-shaped microconidia; thin-walled, multicellular (four to six cells) macroconidia, each with a definite foot cell and a pointed apical cell; and chlamydospores, formed singly in macroconidia, terminally or intercalary in the hyphae.

Biology

The disease is widespread in most countries where lentil is grown. It has been reported from Argentina (Ravenna and Negri, 1979), Canada (Bhalla *et al.*, 1984), Chile (Cafati and Andrade, 1983), Colombia (A. van Schoonhoven, Colombia, 1990, personal communication), former Czechoslovakia (Ujevic *et al.*, 1965), Egypt (Mansour *et al.*, 1976), Ethiopia (Mengistu, 1979), France (Moreau, 1978), Hungary (Fleischman, 1937), India (Vasudeva and Roy, 1950), Jordan (Mamlouk *et al.*, 1984), Morocco (ICARDA, 1987), Nepal (Manandhar, 1975), Sudan (Sarrag and Nourai, 1983), Syria (Bellar and Kabbabeh, 1983; Bayaa *et al.*, 1986), Turkey (Sagir, 1988), Tunisia (Djerbi *et al.*, 1979), Uruguay (Carrera and Noll, 1941), the USA (Wilson and Brandsberg, 1965; Kaiser, 1981) and the former USSR (Kotova *et al.*, 1965). Fusarium wilt caused by *Fusarium* spp. is also a serious disease of pea, chickpea and pigeonpea (see Kraft *et al.*, Chapter 6, this volume; Haware, Chapter 9, this volume; Reddy *et al.*, Chapter 10, this volume), respectively.

The natural host range of the fungus is limited to lentil, although *Vicia montbretii* can be infected with the fungus under artificial inoculation (Bayaa *et al.*, 1995). Wilt caused by *Fusarium* spp. also affects pea, chickpea and pigeonpea (see Kraft *et al.*, Chapter 6, this Volume; Haware, Chapter 9, this volume; Reddy *et al.*, Chapter 10, this volume). The *F. oxysporum* f. sp. *lentis* has great variability. Isolates of the fungus have been differentiated on the basis of their nutritional requirements (Kushwaha *et al.*, 1974; Khare *et al.*, 1975), temperature (Dhingra *et al.*, 1974), their sensitivity to fungicides (Agrawal and Khare, 1977), morphology and virulence (Sharma and Agnihotri, 1972; Claudius and Mehrotra, 1973).

Symptoms

The disease appears in the field in patches at both seedling and adult stages. Seedling wilt is characterized by sudden drooping, followed by drying of leaves and seedling death. The roots appear healthy, with reduced proliferation. Adult wilt symptoms (Fig. 8.5) appear from flowering to late pod-filling stage and are characterized by sudden drooping of top leaflets of the affected plant, leaflet closure without premature shedding, dull green foliage followed by wilting of the whole plant, or of individual branches. The root system appears healthy, with a slight reduction of lateral roots and usually no internal discoloration of the vascular system. Seeds from plants affected in mid to late pod-fill are often shrivelled.

Fig. 8.5. Vascular wilt, caused by *Fusarium oxysporum* f. sp. *lentis*, during early reproductive growth of a lentil crop (Photo: courtesy of B. Bayaa).

Epidemiology

In India, the disease appears in two phases: seedling stage and during reproductive growth. The disease is seen at early stages of crop growth (as a seedling wilt) during November, then incidence falls during December and January. At flowering and podding during late February and March, adult plant wilt symptoms appear (Vasudeva and Srinivasan 1952; Kannaiyan and Nene, 1976). In Syria, wilt appears only during a single phase – that of flowering and podding (April/May). The temperature during the seedling stage of growth in India is around 20°C, whereas in Syria it is lower and only allows slow fungal growth (Erskine *et al.*, 1990).

Plant age affects the germination of fungal spores. Claudius and Mehrotra (1973) found that the root exudate of 21-day-old seedlings contained glycine and phenylalanine which had an inhibitory effect upon spore germination.

External contamination of seeds by the fungus is usual, and high inoculum levels may be carried in plant debris. The fungus may survive in the soil for more than 5 years. Chlamydospores are probably the main fungal structure for long-term survival.

Various factors governing the growth of the pathogen have been studied (Dhingra *et al.*, 1974 ; Khare, 1980; Erskine *et al.*, 1990; Saxena and Khare, 1988; Vasudeva and Srinivasan, 1952). Optimum temperature for fungal growth is around 22°C. Low soil moisture, coupled with moderately high soil temperatures, seem to be the key factors determining symptom expression. Lentil suffers more damage in sandy loam soil (48%) than in clay soil (22%), and the mortality of lentil plants increases with soil pH up to 7.5, above which it declines.

Damage and Crop Loss

Wilt is an important disease of lentil that can cause complete failure of the crop, especially in a warm spring and dry, hot summer (Izquierdo and Morse, 1975; Bayaa *et al.*, 1986; Agrawal *et al.*, 1993). Baraimer and Izquierdo (1977) found that the degree of *F. oxysporum* infection ranged from 25 to 95% depending on the cultivar tested.

Wilt incidence during reproductive growth was correlated with yield loss estimates with a reduction in seed yield per unit change in wilt incidence of 0.846 ± 0.118% in northern Syria. In the laboratory, disease reaction was positively correlated to inoculum density (Erskine and Bayaa, 1996). In the field, inoculum density was unrelated to disease incidence in susceptible lentils, precluding the prediction of disease incidence from inoculum density. *In vitro*, fungal culture filtrate caused 90% seedling mortality (Agrawal *et al.*, 1986a).

Management

Sowing date affects wilt incidence because it determines the proportion of the growth cycle of the crop that is at an optimum or near-optimum temperature for

fungal growth. In India, delayed sowing reduces disease incidence, but late sowing dramatically reduces yield potential and its effect on disease development differs over locations and seasons (Kannaiyan and Nene, 1975a). A crop rotation of 4–5 years reduces inoculum density in the field, but does not completely eradicate the disease. In India, cultivation of paddy or sorghum in the rainy season reduced lentil wilt incidence in the succeeding winter (Kannaiyan and Nene, 1979). Soil amendment with organic matter (wheat or barley straw) enhances antagonism by other soil microorganisms.

Seed treatment with benomyl at 0.3% is reported to check the disease (Kannaiyan and Nene, 1974). A degradation product of benomyl was detected in the root and the shoot up to 45 days after sowing treated seed (Kannaiyan *et al.*, 1975). Agrawal *et al.* (1975) reported that seed treatment with thiram + pentachloronitrobenzene or thiram + carboxin reduced the disease. Wilt incidence can also be decreased by the application of Mn and Zn (Mehrotra and Claudius, 1973). Insecticides like dimethoate, trichlorfon and monocrotophos can also reduce seedling wilt of lentil (Kannaiyan and Nene, 1975b). Benomyl and captan have been shown to be effective in Pakistan (Khalid, 1990).

Biological control is a desirable alternative to chemical control of vascular wilt. In Syria, several antagonistic bacteria isolated from soil were detected by challenging the pathogen, and were identified as *Bacillus* sp. The isolates did not affect seed germination, stimulated plant growth in pots and reduced disease severity (B. Bayaa and W. Erskine, 1994 unpublished results). In India, *Trichoderma viride* Pers. ex Fr., *Streptomyces gougeroti* and some bacterial species were found antagonistic to *F. oxysporum* f. sp. *lentis* (Mehrotra and Claudius, 1972). Similarly, *Trichoderma harzianum* Rifai and *T. koningii* Oudemans showed antibiosis and mycoparasitism (Mukhopadhyay *et al.*, 1989), and in Pakistan *Arachniotus* sp. and *T. harzianum* have been studied (Akhtar, 1989; Aslam, 1989). The possibility of their use as biocontrol agents on a large scale should be explored through field trials and the development of a suitable delivery system. The addition of organic matter to the soil enhances the activity of the antagonists. Complete control of wilt was achieved when *Arachniotus* sp. or *T. harzianum* were used in combination with chopped wheat straw along with 1% N_2 urea + 1% glucose + 1% K_2SO_4 or 1% N_2 urea + 1% K_2SO_4 + 0.001% $MgSO_4$.

Host plant resistance is considered the most feasible and environmentally sound means of vascular wilt management for lentil. Screening of lentil for wilt resistance is done in the field (Plate 19; Kannaiyan and Nene, 1976; Khare *et al.*, 1993; Bayaa *et al.*, 1994a), greenhouse (Bayaa and Erskine, 1990), or in the laboratory (Omar *et al.*, 1988; Bayaa *et al.*, 1994a). Screening methods have been compared and a strong correlation found between field and greenhouse disease reaction (Bayaa *et al.*, 1994a). Plant age has a dramatic effect on resistance; for example, many lines exhibiting resistance at the seedling stage lose their resistance at the adult stage (ICARDA, 1990).

Cultivars with resistance to wilt have been released. These include Naslada, Zhana, Anicia and Tadzhikskaya 95 in Bulgaria (Mihov *et al.*, 1987), Talya 2 in Lebanon (Abi Antoun *et al.*, 1990) and Pant L 406 (Pandya *et al.*, 1980) and Pant 4 (Singh *et al.*, 1994) in India. Sources of resistance to wilt have been found by many authors in the cultivated lentil (Nene *et al.*, 1975; Kannaiyan and Nene,

1976; Khare *et al.*, 1979; Khare, 1980; Tiwari and Singh, 1980; Hossain *et al.*, 1985; Bayaa and Erskine, 1990; Hamdi *et al.*, 1991; ICARDA 1993, 1994) and made available through the Lentil International Fusarium Wilt Nursery. Resistance to wilt has also been found among lentil wild relatives in three accessions (ILWL 79 and ILWL 113 of *L. culinaris* ssp. *orientalis* and ILWL 138 of *L. nigricans* ssp. *ervoides*) (Bayaa *et al.*, 1995).

Saxena and Khare (1988) reported that cultivars with short roots or a low number of secondary roots showed a low incidence of the disease. Such cultivars had compact cork cambium and narrow metaxylem; high levels of amino acids, sugars, phenols, ortho-dihydric phenols, phosphorus, potassium; low permeability and low percentage nitrogen in roots. A large number of cultivars should be studied to correlate such characteristics with resistance.

Kamboj *et al.* (1990) reported that the inheritance of resistance to vascular wilt was controlled by five independently segregating genes based on the reaction of individual plants. Recently, Abbas (1995) highlighted the problems of studying the reaction of individual plants to wilt and, using F_3 progeny rows, found resistance to be governed by a single dominant gene.

BROOMRAPE

Causal Organism

Orobanche spp. (*Orobanchaceae, Dicotyledoneae*) are parasitic higher plants whose hosts are either wild or cultivated plant species. Species attacking lentil are: *O. crenata* Forskall, *O. aegyptiaca* Pers. and *O. ramosa* L. The following is a description of *O. crenata*, the primary problem (Cubero, 1983): the main fleshy stem, which emerges from the ground, is covered by small, scale-like, alternate leaves and is terminated by an inflorescence. The latter is a spike 17–70 cm long, bearing many two-lipped white flowers with purple markings (Plate 20). There are four stamens and a style with a two- to four-lobed stigma. The gynaecium is superior. The fruit is a capsule which contains many minute reticulate brown seeds (4000 seeds per capsule). A mean number of 150,000 seeds per plant has been reported on faba bean (Ponce de León *et al.*, 1974). Cubero (1983) reported 270 ± 25 seeds mg^{-1}, which means that the seed weight is around 3.7×10^{-6} g. The seeds are protected by a black pigment that seems to act as a germination inhibitor and is very difficult to remove. The embryo is poorly differentiated, with small cells in the micropylar zone and vacuolated ones in the chalazal region (Aber and Sallé, 1983). During a survey of south-eastern Anatolia, Turkey (B. Bayaa and W. Erskine, 1996, unpublished observation), it was found that *P. aegyptiaca* was the predominant species of broomrape; *O. crenata* infestation was less common.

Biology and Epidemiology

O. crenata is endemic to the Mediterranean area, southern and eastern Spain, southern Italy and some parts of Greece, North Africa and the Near East (Cubero

et al., 1988). *O. crenata* has a wide host range that includes members of the *Compositae*, *Umbelliferae* and *Leguminosae*. Among legumes, faba bean (see Jellis *et al.*, Chapter 7, this volume), vetch and clovers are hosts in addition to lentils. However, the extent of host specificity of lentil isolates of *O. crenata* appears unknown: the parasite is frequented by bees so is presumably cross-pollinated; seed from a single flower spike is probably heterogeneous. The seeds do not germinate unless they are pre-conditioned (ripened and exposed to moisture) and stimulated by certain components of the host root exudate. Some chemicals, including gibberellic acid and sodium hypochlorite, increase germination in the presence of a germination stimulant (Hiron, 1973; Pieterse, 1981).

Germination occurs between 18 and 25°C and is inhibited below 8°C (Kasasian, 1973). However, Sauerborn (1989) observed delayed germination and host plant attachment at 5°C. Seeds are damaged when the temperature reaches −5°C (Canizo, 1946). Germination does not occur during winter. Also crops sown late in the season are often less infected. Light is not required for germination and may even have a negative effect (Cubero, 1983).

In the proximity of a host root (*c.* 1 cm), the embryo produces a haustorium which penetrates into the root by mechanical pressure (Privat and Andary, 1973). Thereafter, the growth of haustorium within the root is achieved by enzymatic processes (Dörr and Kollmann, 1974; Dörr, 1979). The establishment of connections between the host and parasite is achieved by fusion of both xylems but not between the phloem of host and parasite (Aber and Sallé, 1983). The effects of temperature on haustorial attachment and development are unknown.

Attachment is followed by the formation of a spherical nodule, which has an intense red-orange colour. A bud is formed on the nodule, when it reaches 1–2 cm in diameter, which ultimately produces a whitish stem bearing floral buds. This occurs about 10 weeks after germination (Cubero, 1983).

O. crenata is facultatively autogamous (Cubero, 1983). The stamen growth places the anther close to the base of the stigma, allowing a portion of the pollen to brush against it. Cross-pollination is by large *Hymenoptera*, especially bumble bees. The seeds are distributed by the wind and may remain dormant for up to 18 years.

Orobanche spp. generally occur on poor dry soils (Kasasian, 1971) and increasing soil fertility restricts *Orobanche* development. *In vitro* the presence of P, urea, or NH_4 (but not NO_3^-) inhibit germination of *O. crenata* (ter Borg, 1986).

Symptoms

The appearance of the parasite itself is the most diagnostic feature of infestation. The parasite has erect, branched or unbranched aerial flowering shoots. *O. crenata* has white flowers and *O. aegyptiaca* has blue flowers (Plate 20).

Damage and Loss

Broomrape infestation may cause severe yield loss. The host can be completely destroyed and a highly infested field may give the impression that the cultivated

plants are broomrape! Yield reduction is particularly correlated with the number of parasites per host plant and the earliness of attack. In faba bean, Cubero and Hernández (1991) found that there was an extremely high correlation between the number of emerged broomrape shoots per host plant and the total number of broomrape tubercules attached to the host roots.

The most important damage is apparently caused by competition for water: by water flow from the host to the parasite and the reduced ability of the host roots to extract water from the soil. Weakness of the host is attributed to the flow of sugar, especially sucrose, to the parasite, which is translocated to the broomrape spike and at once split into glucose and fructose (Whitney, 1972)

Management

Orobanche control in lentil is difficult, for several reasons: (i) the large number of seeds produced on a single inflorescence, (ii) extended survival of the seed in the soil, and (iii) attachment with the host which necessitates very specific control measures (Pieterse, 1979; Cubero, 1983; Parker and Wilson, 1986). Control measures include physical, cultural, chemical and biological methods and their combined use into an integrated control package.

Flooding the field for 1 month led to the loss of broomrape seed viability (Kasasian, 1971). Generally, irrigated fields have less of a problem than rainfed fields. Hand-weeding of inflorescences reduces the seed bank, if done before seed maturity, but is time consuming, may cause damage to the host crop and is only economic when the level of infestation is low. Weekly hand-pulling reduced infestation by 95% over 5 years (Krishnamoorthi and Krishnan, 1967) but was unable to eradicate the parasite from a plot artificially infested with *Orobanche* continuously cropped with faba bean, even after 14 years of hand-pulling (Cubero, 1983).

Deep ploughing delays seed germination and reduces *Orobanche* infestation (Kasasian, 1971); however, in practice it is difficult to invert the topsoil completely. Delayed sowing is a common practice in the Middle East to reduce the level of infestation. Following the suggestion of Cubero and Moreno (1979), early maturing genotypes, such as ILL 8, have been sown late to reduce the level of infestation (ICARDA, 1990, 1991, 1992, 1993). However, late sowing has a reduced yield potential compared to normal December sowing in West Asia (Silim *et al.*, 1991). Crop rotation is of little importance due to the persistence of the seeds for extended periods (up to 18 years) and the non-specificity of the parasite attack.

Trap plants are non-hosts which are able to stimulate *Orobanche* seeds but do not permit the development of the haustorium. These can be used as secondary (trap or catch) crops in the rotation either for forages or green manuring. Ciccarone and Piglionica (1979) suggested *Astragalus baeticus* L., flax and *Helminthia echoides* Gaert as possible trap crops for *O. crenata*. A limitation of this method is that only *Orobanche* seeds in the rhizosphere of the trap crop will germinate under its stimulus; in addition the longevity of viable broomrape in the soil is a limiting factor.

Fertilizers such as urea can reduce *Orobanche* infestation, whereas K promotes *O. crenata* growth (Kasasian, 1973). Soil solarization, using transparent polyethylene sheets on the soil surface in the hot season, has been extensively tried for *Orobanche* control at ICARDA (Sauerborn and Saxena, 1987). Although the control was dramatic and the dry weight of *Orobanche* was reduced by 90%, polyethylene is too expensive for economic use by farmers on a low-value crop like lentil.

Among the various chemicals tested for their use on broomrape on lentil, imazethapyr and imazaquin are the most promising (Sauerborn *et al.*, 1987b; ICARDA, 1991, 1992, 1993). Glyphosate, which is recommended for use at sublethal doses for foliar application on faba bean to control broomrape, is not recommended for lentil because of the extreme sensitivity of the crop to the chemical.

Although several phytophagous insects attack broomrapes, only *Phytomyza orobanchia* Kalt. (*Agromyzidae*) has shown biocontrol potential (Cubero, 1983; Giray and Nemli, 1983; Mihajlovic, 1986; Linke *et al.*, 1990). The larvae feed on the seeds and bore into the stalks of *Orobanche* until they reach the underground parts of the host where they pupate. The fly infests almost 90% of the capsules of *O. crenata* and an attempt has been made to use the fly in integrated broomrape control (Linke *et al.*, 1990).

The fungus *Fusarium orobanche* Jacz. was found effective against both the seeds and seedlings of the parasite (Kott, 1969). *Fusarium oxysporum*, *F. solani*, *Alternaria* spp. and *Sclerotinia* spp. have been isolated from rotted *O. crenata* fruits (Al-Menoufi, 1986). They were responsible for a reduction of germination of *Orobanche* seeds between 21 and 85%, and caused no damage on several host crops.

Host plant resistance has been widely used in faba bean to control broomrape (see Jellis *et al.*, Chapter 7, this volume). In lentil, a total of 1774 germplasm accessions have been screened in the field in infected soil and a range in reactions recorded (Erskine and Witcombe, 1984). However, the reaction of the most resistant accessions was examined in petri dishes in the laboratory (Sauerborn *et al.*, 1987a) and there were no significant differences among accessions in the number of infections of *Orobanche* per unit length of root. The low incidence of *Orobanche* infection on the roots of 'resistant' accessions in the field was probably due to poor root growth. Despite further extensive screening in petri dishes, resistance to *Orobanche* has not been found in the cultigen. Screening has continued with the wild *Lens* spp., but resistance remains elusive (Erskine *et al.*, 1994).

The integration of some of the above control measures has been tried in Spain (Garcia-Torres and López-Granados, 1991) and Syria (ICARDA, 1990, 1991, 1992, 1993). The methods included using delayed sowing, an early-maturing cultivar (ILL8) adapted to late sowing, in combination with either two post-emergence applications of imazaquin (7.5 g active ingredient (a.i.) ha^{-1}) or one pre-emergence application of imazethapyr (60 g a.i. ha^{-1}). For *Orobanche* control and both seed and straw yield, imazaquin was the better chemical particularly on the late sowing of the early cultivar.

CONCLUDING REMARKS

Lentil is a low-value food legume predominantly grown in the dry areas of Asia by resource-poor farmers. The most economic and feasible method of disease control is through host plant resistance. In this, considerable progress has been made in breeding for resistance individually to rust, wilt, ascochyta blight and stemphylium blight in the last decade. Focus should now be on their appropriate recombination as stresses often appear together in the field. A search for markers, using random amplified polymorphic DNA (RAPD) in particular, is under way for rust, ascochyta blight and vascular wilt, so that marker-assisted selection may be used to increase selection efficiency in the future. In the absence of genetic variation in resistance to broomrape in lentil, and in view of the phytotoxicity of current selective herbicides to the crop, genetic engineering to incorporate herbicide resistance is needed. Research on grey mould of lentil is neglected, but substantial losses occur in wet seasons. Experience with other crops suggests that it is difficult to obtain a suitable level of resistance to this variable pathogen, so an integrated disease management package should be developed for control.

To date, resistances to rust, wilt, ascochyta blight and *Stemphylium* have not broken down. However, information from other crop/pathogen systems suggests that this situation will not continue indefinitely. Vigilance is required to identify pathogenic variability in wilt, rust and ascochyta blight. Once identified, DNA-based systems to study variability may be of assistance.

Several biological control agents, fungal and bacterial, have been identified *in vitro*. Further tests for their effectiveness in the field are required. Problems remain to be solved in their economic bulk production, the development of appropriate delivery systems, the compatibility with chemicals used in seed dressing and their safety on non-target organisms including *Rhizobium*.

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DISEASES OF CHICKPEA 9

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INTRODUCTION

Chickpea (*Cicer arietinum*) is the third most important grain legume globally, after common bean and pea (soyabean and groundnut being considered as oilseed crops). It is particularly important as a source of protein to the largely vegetarian population of South Asia and is used as an animal feed in developed countries. According to FAO (1994), chickpea is cultivated on 10.2 million hectares and its total production is nearly 7.8 million tonnes. The yield potential of present-day chickpea cultivars exceeds 5 t ha^{-1}, however, average yield is less than 0.8 t ha^{-1}. The gap between the average and potential yield is mostly due to diseases, pests and poor management practices. Although chickpea may be treated as a low priority crop by farmers, recent price increases in pulses has renewed interest in expanding the area sown.

Two races are recognized within cultivated chickpea which are roughly equivalent to the widely recognized *kabuli* and *desi* types. *Kabuli* types are tall with white flowers and produce large, rounded seed usually pale cream in colour. They are common in the Mediterranean and the Near East. *Desi* types are relatively short, sometimes prostrate, commonly with anthocyanin pigmentation in flowers and stems and produce small, irregularly shaped seed of various colours (Allen, 1983). Differences in resistance and susceptibility between the two types to major widespread diseases is highlighted in this chapter.

About 67 fungi, 3 bacteria, 22 viruses and 80 nematodes have been reported on chickpea (Nene *et al.*, 1996), but only a few of these cause economically important diseases. Several detailed reviews of chickpea diseases have already been published (Nene and Reddy, 1987; Nene *et al.*, 1991). This chapter focuses on the most important root diseases of chickpea: fusarium wilt and root rots caused by a complex of soilborne fungi; the most economically important foliar diseases: ascochyta blight and botrytis grey mould; and the most important and prevalent virus disease: stunt, in most of the chickpea growing areas of the world.

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Table 9.1. Fungal diseases of chickpea of minor or local importance.

Disease	Causal fungus	Distribution	Importance
Stem rots, root rots and wilts			
Stem anthracnose	*Colletotrichum capsici* (Syd.) Butl. & Bisby	India	Occasional, minor
Stem rot, white mould	*Sclerotinia sclerotiorum* (Lib.) de Bary	Iran, India, Pakistan, Australia, Chile	Locally important, especially under cool wet conditions
Foot rot	*Operculella padwickii* Kheswalla	North India	Locally important
Rhizoctonia root rot	*Rhizoctonia solani* Kühn	Widespread	Minor
Root rot	*Phytophthora megasperma* Drechs.	India	Minor
Neocosmospora root rot	*Neocosmospora vasinfecta* Smith	India	Minor
Root rot	*Thielaviopsis basicola* (Berk. & Br.) Ferr.	USA	Minor
Acrophialophora wilt	*Acrophialophora fusispora* (Saksena) Samson	India	Minor
Verticillium wilt	*Verticillium dahliae* Kleb.	USA	Minor
Foliar diseases			
Phoma blight	*Phoma medicaginis* Malbr. & Roum.	Australia, India, Bangladesh, USA	Minor
Stemphylium blight	*Stemphylium sarciniforme* (Cav.) Wilts.	India, Iran, Syria	Minor?
Alternaria blight	*Alternaria alternata* (Fr.) Keissler	India, Bangaldesh, Nepal	Minor
Powdery mildew	*Leveillula taurica* (Lév.) Arn.	India, Ethiopia, Sudan	Minor
Rust	*Uromyces ciceris-arietini* Jacz. apud Boy. & Jacz.	Widespread in Mediterranean region, SE Europe and Asia; present also in E Africa and Mexico	Locally important; regarded as the major factor affecting chickpea production in Central Mexico

Sources: Allen (1983), Nene *et al.* (1996).

Table 9.2. Virus diseases[1] of chickpea of minor or local importance.

Disease	Causal virus	Distribution	Importance
Yellow mosaic, yellowing	Bean yellow mosaic potyvirus (BYMV)	USA, Iran, India	Locally important; yield loss estimated at 77–92% in Iran
Yellow mosaic	Beet western yellows luteovirus (BWYV)	Australia, India Spain, Syria, USA	Minor
Mosaic, bud necrosis, wilt	Alfalfa mosaic virus (AMV)	Widespread	Locally important in Iran where yield losses of 22–96% have been recorded
Mosaic	Cucumber mosaic cucumovirus (CMV)	Widespread	Locally important in Iran where yield losses of 52% have been recorded
Enation mosaic	Pea enation mosaic virus (PEMV)	USA, Italy	Minor?
Necrotic yellows	Lettuce necrotic yellows rhabdovirus (LNYV)	Australia	Potentially important in northern New South Wales and southern Queensland

[1] All viruses are aphid-transmitted and not seedborne. Adapted from Allen (1983).
Sources: Kaiser and Danesh (1971 a, b); Brunt *et al.* (1990); Kaiser *et al.* (1990); Nene *et al.* (1996).

Table 9.3. Nematode diseases of chickpea of minor or local importance.

Disease	Causal virus	Distribution	Importance
Root-knot	*Meloidogyne artiellia* Franklin	Italy, Spain, Syria	Potentially serious in spring-sown crops
	M. incognita (Kofoid & White) Chitwood	Bangladesh, India, Nepal, Pakistan	Locally important
	M. javanica (Treub.) Chitwood	Widespread	Locally important
Cyst	*Heterodera ciceri vovlas*, Greco & Dvito	Jordan, Lebanon, Syria	Severe damage in Syria
	H. rosii Duggen & Brennan	Syria	
Root lesion	*Pratylenchus brachyurus* (Godfrey) Goodey	Australia, Brazil	Potentially serious
	P. thornei Sher. & Allen	Australia, India, Myanmar, Syria	Locally important
Decline	*Rotylenchulus reniformis* Lindford & Oliveira	Ghana, India	Minor

Sources: Greco and Sharma (1990); Nene *et al.* (1996).

Other diseases of local and minor importance caused by fungi, viruses and nematodes are listed in Tables 9.1–9.3.

FUSARIUM WILT

Aetiology

Chickpea wilt was first reported in India by Butler (1918). McKerral (1923), while working in Myanmar, considered the disease to be soilborne. Narsimhan (1929) and Dastur (1935) reported an association of *Fusarium* spp. and *Macrophomina phaseolina* (Tassi) Goid. with wilted plants. Dastur (1935) could not prove pathogenicity of the isolated *Fusarium* sp. and concluded that wilt was due to abiotic factors. In a detailed account, Prasad and Padwick (1939) reported *Fusarium* sp. to be the causal agent of chickpea wilt. Padwick (1940) named the fungus *Fusarium orthoceras* Appel & Wollenw. var. *ciceri*. Erwin (1958) named it *F. lateritium* (Nees) Snyder & Hansen f. sp. *ciceri* (Padw.) Erwin. Following the classification of Snyder and Hansen (1940), Chattopadhyay and Sen Gupta (1967) renamed the pathogen *Fusarium oxysporum* Schl. f. sp. *ciceri* (Padw.) Snyder & Hansen. This was accepted as the correct name of the pathogen (Booth, 1971) but has since been revised to *F. oxysporum* Schl. f. sp. *ciceris* (Padw.) Matuo & Sato (Holliday, 1980).

Biology

Fusarium wilt is the most important disease of chickpea. It is widespread in chickpea growing areas in Asia, Africa, southern Europe and the Americas, having been recorded in at least 33 countries (Nene *et al.*, 1996). Fusarium wilt is also a serious disease of pea (see Kraft *et al.*, Chapter 6, this volume), lentil (see Jellis *et al.*, Chapter 7, this volume) and pigeonpea (see Reddy *et al.*, Chapter 10, this volume). *F. oxysporum* has a worldwide distribution as a soilborne fungus and is considered to be the most economically important member of the genus (Holliday, 1980). It is also one of the most labile and variable. The fungus on potato sucrose agar at 25°C appears as delicate, white and cottony growth, becoming felted and wrinkled in older cultures (Nelson *et al.*, 1983). Hyphae are septate and profusely branched. Microconidia are borne on simple short conidiophores, arising laterally on the hyphae. Microconidia and macroconidia are generally sparse on solid media. They are formed abundantly in potato sucrose broth. Microconidia are oval to cylindrical, straight to curved and measure 2.5–3.5 × 5–11 µm. Macroconidia develop on the same conidiophores on which microconidia are formed (Nelson *et al.*, 1983). Macroconidia are lesser in number than microconidia, borne on branched conidiophores, thin-walled, three- to five-septate, fusoid, pointed at both ends, and measure 3.5–4.5 × 25–65 µm. Chlamydospores, formed in 15-day-old cultures, are smooth or rough walled, terminal or intercalary, and may form singly, in pairs, or in a chain. Optimum conditions for growth of *F. oxysporum* f. sp. *ciceris* are 25°C and pH 6 (Haware and Nene, 1982).

Pathogenic variability in *F. oxysporum* f. sp. *ciceris* has been reported. Seven

Table 9.4. Reaction of ten differential lines of chickpea to different races* of *Fusarium oxysporum* f. sp. *ciceris*

Line	Race 0	Race 1	Race 2	Race 3	Race 4	Race 5	Race 6
JG 62	R[2]	S	S	M	S	S	S
C 104	M	S	S	R	S	S	S/M
JG 74	R	R	M	R	R	S	R
CPS 1	R	R	S	S	M	S	R
BG 212	R	R	S	M	M	R	R
WR 315	R	R	R	M	R	R	R
Annigeri	–	S	S	S	S	–	R
Chafa	–	S	S	M	S	–	R
L 550	–	S	S	M	S	–	S/M
850–3/27	–	S	M	M	M	–	R

[1] Races 1, 2, 3 and 4 identified in India by Haware and Nene (1982c); Races 0 and 5 identified in Spain by Cabrera *et al.* (1985); Race 6 identified in California, USA, by Phillips (1988).
[2] R = Resistant (0–20% mortality); M = Moderately susceptible (21–50% mortality); S = Susceptible (>50% mortality).

races have been identified from their differential reactions on chickpea lines (Table 9.4) (Haware *et al.*, 1990). Races 1, 2, 3 and 4 were first described in India by Haware and Nene (1982c) and races 0 and 5 in Spain by Jiménez-Díaz *et al.* (1989, 1991, 1993). Race 6 was described from California (Phillips, 1988). In India, races 1, 2, 3 and 4 are generally geographically distinct and only race 1 and, to a lesser extent, race 2 are widespread and appear to be more virulent than others (Haware and Nene, 1982c). Chickpea cultivars to be used in the northern plains of India should have resistance to race 2 (Jagdish Kumar, ICRISAT, India, 1996, personal communication). It is interesting to note that cultivar JG 62 is highly susceptible to race 1 but is resistant to wilt in Tunisia and Spain (Cabrera *et al.*, 1985). Race 0 causes yellowing of leaves and no wilting, whereas all other races from India, Spain and the USA, cause typical vascular wilt. Recently, genetic fingerprinting and random amplified polymorphic DNA (RAPD) analyses have been used to characterize pathotypes of *F. oxysporum* f. sp. *ciceris*. With RAPDs, it was possible to distinguish f. sp. *ciceris* from other *formae speciales* of *F. oxysporum* and other *Fusarium* spp. (Kelly *et al.*, 1994).

Symptoms

Although several fungal pathogens cause diseases of seedlings and roots of chickpea, wilt can be differentiated from other root diseases by careful examination of the infected plant. Chickpea genotypes show different rates of symptom expression after infection with *F. oxysporum* f. sp. *ciceris* and can be classified in early and late wilting categories on the basis of days from sowing (Haware and Nene, 1980). Wilt can be observed in a susceptible cultivar within 25 days after sowing in infected soil and this is known as 'early wilt' (Haware and Nene, 1980).

Affected seedlings show drooping of the leaves and are a dull green colour. Seedlings collapse and, when uprooted, may show uneven shrinkage at the collar. Isolates of *F. oxysporum* f. sp. *ciceris* may induce either fast wilting or a progressive yellowing syndrome which develops 15 to 40 days after inoculation depending on the cultivar.

Wilting may also occur during reproductive growth and is known as 'late wilt'. Drooping of the petioles, rachis and leaves in the upper part of the plant together with the pale green colour of foliage is the most common symptom (Fig. 9.1). Lower leaves also become chlorotic (Nene *et al.*, 1978). When uprooted before completely dried, affected plants show no external root discoloration. However, when roots are split vertically, internal discoloration may be seen extending to the stem (Fig. 9.1). Roots also show this symptom in early wilting. Internal discoloration is due to infection of the xylem tissues of the root and stem. Partial wilting can be seen in the field, however it is not common in chickpea. Transverse sections of the infected root examined under the microscope show the presence of hyphae and spores of the fungus in the xylem (Nene *et al.*, 1978). Their presence also confirms the diagnosis of vascular wilt. In some cultivars, typical wilt symptoms are absent. The lower leaves turn yellow and dry, and the plant remains stunted. Roots show internal discoloration. Plants grown from infected seed wilt faster than plants orginating from clean seed.

Fig. 9.1. Drooping of leaves and petioles and discoloration of the main stem of chickpea caused by *Fusarium oxysporum* f. sp. *ciceris* (Photo: courtesy of M.P. Haware).

Epidemiology

Wilt is important between the latitudes 30°N and 30°S of the equator where the chickpea growing season is dry and warm. The epidemiology of root-infecting fungi in the soil is complex and factors such as inoculum density and pathotype, plant age, host resistance and its genetic potential, air and soil temperature, soil moisture, soil nutrients and plant density may affect wilt development (Haware *et al.*, 1990). Wilt severity and populations of *F. oxysporum* f. sp. *ciceris* increased with decreasing soil matrix potential (Bhatti and Kraft, 1992).

F. oxysporum f. sp. *ciceris* is internally seedborne and the fungus is found as chlamydospore-like structures in the hilum region of the seed (Haware *et al.*, 1978). Infected seed plays an important role in long-distance dispersal and in transmitting the disease to new areas. Once the inoculum is established in the soil, it is difficult to eradicate. The fungus also survives in the soil as chlamydospores. It can survive in infected crop residues buried in the soil in the absence of the host for at least 6 years (Haware *et al.*, 1996b). The fungus can also affect other *Cicer* spp. under artificial inoculation conditions (P. Stevenson, ICRISAT, India, 1995, personal communication); it may affect such species naturally. Lentil, pea and pigeonpea are symptomless carriers of the chickpea wilt fungus (Haware and Nene, 1982) and other legumes may be affected. Further work on the extent and role of natural hosts in the epidemiology of this pathogen may enhance our understanding of the disease.

Losses

Crop losses caused by wilt have mainly been made from field estimates based on the percentage incidence of the disease. No precise information on losses caused by wilt is available. Based on a rough estimate, an annual loss of US $ 1 million was reported from Pakistan in 1953 (Sattar *et al.*, 1953). In India, an annual loss of 10% has been reported (Singh and Dahiya, 1973) while in Spain an annual loss of 12–15% due to both wilt and root rots was estimated (Trapero-Casas and Jiménez-Díaz, 1985). In the drier areas of North Africa, wilt of chickpea is a serious disease and is especially common in Tunisia (Haware *et al.*, 1990). The production of chickpea in California has declined in recent years largely because of wilt (Haware *et al.*, 1990). At ICRISAT, an attempt was made to estimate yield losses on a single plant basis. Early wilting caused 77–94% yield loss while late wilting caused 24–65% loss (Haware and Nene, 1980). Seed harvested from the late wilted plants was lighter and duller than that harvested from healthy plants.

Management

The chickpea wilt pathogen is both soilborne and seedborne (Haware *et al.*, 1996b). It is difficult to eliminate inoculum from the field. Where land is not limiting, avoidance of planting in heavily infested fields can minimize the effects of this disease on yield. As the fungus can survive in soil for long periods, crop rotation

is not an effective practice for reducing wilt incidence (Haware *et al.*, 1996b). Deep ploughing during the summer and removal of host debris from the field can reduce inoculum levels. Solarization, by covering the soil with transparent polythene sheeting for 6–8 weeks during the summer months, effectively controls wilt in chickpea and improves plant growth and yield (Chauhan *et al.*, 1988). Although it is useful in commercial production, it is not a practical option for resource-poor farmers. Seed should be produced from disease-free plants and in disease-free areas to avoid transmission. Seed movement without risk is possible if seed is treated with appropriate fungicides for eradication of seedborne inoculum (Haware *et al.*, 1978). A mixture of 30% benomyl + 30% thiram at 1.5 g kg^{-1} seed successfully eradicates inoculum of the pathogen from seed. Such control technologies still need to be effectively transferred to chickpea farmers.

Due to the difficulty of widespread application of available cultural and chemical control measures for wilt, especially for resource-poor farmers with limited land, considerable emphasis has been placed on the development of resistant cultivars (Nene and Haware, 1980; Haware *et al.*, 1992b). At ICRISAT Center, India, effective field-screening and laboratory procedures have been developed including the use of wilt-sick plots (Plate 21) (Nene *et al.*, 1981; Nene and Reddy, 1987). Over 160 wilt-resistant sources have been identified (Harjit Singh *et al.*, 1987a, b; Haware *et al.*, 1990, 1992b). Interestingly, many of these are *desi* types as resistance to wilt is less common in *kabuli* types (Harjit Singh *et al.*, 1987a, b). Accessions of several wild species including *C. bijugum*, *C. echinospermum*, *C. judaicum* and *C. pinnatifidum* are also resistant to wilt (Haware *et al.*, 1992a; P. Stevenson, ICRISAT, India, 1995, personal communication).

Many lines have been developed with resistance to races 1 and 2. In spite of the existence of races of the pathogen, it has not been difficult to identify high levels of resistance which operate over wide areas due to the geographical isolation of some races (Haware and Nene, 1982). Resistant lines, such as ICC 12237 and ICC 12269, have additional resistance to dry root rot and black root rot (Nene, 1988). Wilt resistance genes have been incorporated into high-yielding *desi* and *kabuli* backgrounds (Jagdish Kumar *et al.*, 1985). Chickpea lines resistant to wilt such as ICCV 2–10, ICCC 4 and ICCC 37 are becoming popular in India. Chickpea breeding programmes at Culiacan and Sonora, Mexico, have released highly resistant cultivars including Suratato 77 (Morales, 1986). Two large-seeded, wilt-resistant cultivars, UC 15 and UC 27, have been released in California (Buddenhagen *et al.*, 1988). Amdoun-1, a wilt-resistant cultivar, was released in Tunisia in 1986 (Haware *et al.*, 1992b). There is no evidence to date of breakdown of resistance to *F. oxysporum* f. sp. *ciceris*. Induced resistance to wilt has been reported using non-pathogenic isolates of races 0 and 1 of *F. oxysporum* f. sp. *ciceris* (Hervás *et al.*, 1995).

Earlier reports suggested that resistance to wilt in chickpea was conferred by a single recessive gene (Jagdish Kumar and Haware, 1982). Subsequent studies have indicated that the genetics of this host–parasite system are more complex. Resistance to race 1 of *F. oxysporum* f. sp. *ciceris* appears to be controlled by at least three independent loci, designated H_1, H_2 and H_3. Partly recessive alleles in homozygous form at either of the first two loci (such as in cultivars K 850 and C 108), and the dominant allele at the third locus in cultivar H 208, independently

delay wilting but any of these two alleles together confer complete resistance (Upadhyay *et al.*, 1983a, b). Further studies have shown that some resistant cultivars have other recessive alleles at both of the first two loci which separately confer complete resistance (Harjit Singh *et al.*, 1987a, b). Complete resistance is obtainable from crosses involving only susceptible (through late wilting) parents (Smithson *et al.*, 1983; Harjit Singh *et al.*, 1987). Resistance to race 2 is controlled by two genes, the first of which must be present in the homozygous recessive form and the other in the dominant form whether homozygous or heterozygous for complete resistance (Gumber *et al.*, 1995). Late wilting occurs if both are dominant. Recent studies suggest that one of the genes probably confers resistance to both races 1 and 2 (Jagdish Kumar, ICRISAT, India, 1996, personal communication). Collaborative work with the John Innes Centre, UK, is identifying molecular markers for wilt resistance using amplified fragment length polymorphisms (AFLPs) (ICRISAT, 1994).

Spore germination and hyphal growth of *F. oxysporum* f. sp. *ciceris* were significantly inhibited in the presence of root exudates from wilt-resistant chickpea cultivars CPS 1 and WR 315 (Haware and Nene, 1984; Stevenson *et al.*, 1994, 1995). High-pressure liquid chromatography analysis identified the pterocarpans medicarpin and maackiain in the exudate of CPS 1 at more than ten times the concentration in JG 62, a wilt-susceptible cultivar (Stevenson *et al.*, 1994; 1995). Further, thin layer chromatographic analysis showed that the relative mobility of these compounds was close to those isolated bands which inhibited germination and hyphal growth, suggesting that the activity of these compounds may have been due to the constitutive presence of medicarpin and maackiain. Recent work (P.C. Stevenson and M.P. Haware, unpublished data) has confirmed that medicarpin and maackiain are induced in seedling roots of chickpea when inoculated with both race 1 and race 2 of *F. oxysporum* f. sp. *ciceris*. The results suggest that the resistance of chickpea to wilt depends, at least in part, upon the antifungal activity of the root exudates (Stevenson *et al.*, 1995). Further work is in progress.

ROOT ROTS

Aetiology

Root rot, collar rot and pre- and post-emergence damping-off are important soilborne fungal diseases of food legumes including chickpea (Nene and Reddy, 1987; Nene *et al.*, 1989, 1991). As most of these pathogens can occur singly or in a complex, it is common for more than one soilborne pathogen to cause problems in any one field. It is difficult to rate one as being more important than the other although one may predominate at a particular location. Pre- and post-emergence damping-off is caused by *Pythium ultimum* Trow. and has been reported from Iran, Turkey and the USA (Kaiser and Hannan 1983). It also causes a serious disease of pea (see Kraft *et al.*, Chapter 6, this volume). Root rots are frequently caused by *Macrophomina phaseolina* (Tassi) Goid. (sclerotial state *Rhizoctonia bataticola* (Taub.) Briton-Jones), *Rhizoctonia solani* Kühn and *Fusarium*

solani (Mart.) Sacc. (Haware *et al.*, 1990). *M. phaseolina*, the causal agent of dry root rot, is especially favoured by hot tropical climates. It also causes charcoal rot of many crops including soyabean (see Sinclair, Chapter 3, this volume). Collar rot is caused by *Sclerotium rolfsii* Sacc. It is also reviewed on groundnut (see McDonald *et al.*, Chapter 2, this volume) and lentil (see Bayaa and Erskine, Chapter 8, this volume). All of these pathogens are soilborne, facultative saprophytes and have wide host ranges. The diseases caused by these pathogens are important production constraints depending on the prevailing conditions in the chickpea growing area.

Biology

Among root rots, dry root rot caused by *M. phaseolina* is the most severe in the semi-arid tropics (Nene *et al.*, 1989, 1996). It has been reported from Australia, Ethiopia, Iran, Pakistan, Sudan and the USA. Collar rot is most common in seedlings and, despite considerable research, *Sclerotium rolfsii* continues to plague farmers, affecting over 100 crops and causing considerable losses in wet soils associated with warm climates (Haware *et al.*, 1990). The disease is particularly serious in Bangladesh, eastern India, Nepal and Myanmar, where chickpea is sown after rice. Black root rot caused by *F. solani* has been reported from India, the USA, Mexico and Chile. It is considered serious in Mexico and in Washington State, USA (Kraft, 1969; Grewal *et al.*, 1974; Westerlund *et al.*, 1974; Nene *et al.*, 1989, 1996). It is a well-known fungus causing root rot of chickpea and other legumes such as soyabean, faba bean, pea and phaseolus bean as well as cucurbits and onion in temperate and tropical soils (Booth, 1971; Holliday, 1980).

Pythium ultimum belongs to the *Phycomycetes*. The mycelium is white and well developed. Hyphae are usually non-septate, but septate in old cultures, branched, 1.7–6.5 µm in diameter. Sporangia are spherical if terminal or barrel-shaped if intercalary, 12–30 × 25 µm in size (Holliday, 1980). They are formed abundantly in culture and germinate by forming germ tubes. Oogonia are smooth, terminal, spherical, rarely intercalary, 20–23 µm in diameter. Oospores are single, spherical, smooth, thick-walled and 14–20 µm in diameter (Holliday, 1980).

Pycnidia of *M. phaseolina* have not been observed on chickpea but may form in culture. Small black sclerotia of the *R. bataticola* state are formed abundantly in culture and in the bark and pith of infected roots. Sclerotia are 80–174 µm in size, irregular and remain viable for at least 12 months in soil. The mycelium is composed of individual septate, hyaline hyphae. Pycnidia are dark brown, more or less erumpent, globose with inconspicuous truncate ostioles and 100–200 µm in size (Holliday, 1980). Conidia are aseptate, hyaline, elliptical to oval, thin-walled, 16–29 × 6–9 µm in size being formed on short, cylindrical condiophores, 10–15 µm long (Holliday, 1980).

On agar media, *F. solani* develops a blue to bluish-brown discoloration. The mycelium is aerial, greyish-white and septate and conidia develop in young cultures. Microconidia develop abundantly on microconidiophores which are elongated, unbranched and up to 300 µm long. Macroconidia form on short,

multibranched conidiophores of 8–15 μm. They are inequilaterally fusoid with some spores having the widest diameter in the penultimate cells and measure 35–60 × 4–6 μm (Booth, 1971). Chlamydospores develop abundantly on the mycelium as globose to oval, smooth to rough-walled spores which form terminally or intercalary and measure 10–12 × 7–11 μm. They remain dormant in the soil for some time.

The mycelium of *S. rolfsii* is white, densely floccose, with septate hyphae. Sclerotia are formed in the medium and on host tissues as numerous, olive brown to clove brown, globose, hard bodies, measuring 0.8–2.5 mm in diameter. They germinate easily in water by forming hyphae. The fungus belongs to the Mycelia Sterilia.

Symptoms

In the Palouse region of eastern Washington, *P. ultimum* and *F. solani* are isolated most frequently from decayed chickpea seeds in the soil (Kaiser and Hannan, 1983). Affected seedlings collapse. When examined closely, diseased plants show rotting of the roots and collar region. Sudden drying of plants in the field is the most important symptom caused by *M. phaseolina* generally under dry and hot conditions (Singh and Mehrotra, 1982). Leaves and stems of diseased plants are straw-coloured. The affected plants can be pulled easily from the ground because of rotting of lateral roots. The tap root is dark and brittle and can easily be broken (Fig. 9.2). Using a hand lens (10 ×), dark brown, minute sclerotia can be seen on and inside the bark. The name 'dry root rot' indicates the common appearance of the disease in the dry and hot semi-arid tropics. *F. solani* causes black root rot of chickpea particularly in cool and wet climates (Kraft, 1969; Westerlund *et al.*, 1974). Affected plants turn yellow and wilt, and roots turn black and rot (Fig. 9.3), especially in the presence of excessive soil moisture. *S. rolfsii* causes collar rot as well as pre-emergence and post-emergence damping-off, thus affecting field stands (Haware *et al.*, 1990). Plants are susceptible at the seedling stage and affected seedlings turn yellow and dry. Seedlings show rotting at the collar region and white mycelium and small, round, dark brown sclerotia can be seen on affected host tissues. Sclerotia are also seen on the soil surface near the plant. In general, *kabuli* types are more susceptible to root rot pathogens than *desi* types (Haware *et al.*, 1990). This may be due to the thin seed coat of *kabuli* types (Kaiser and Hannan, 1983).

Epidemiology

F. solani, *M. phaseolina*, *S. rolfsii* and *P. ultimum* are soilborne, facultative saprophytes. These fungi have wide host ranges and are widely distributed in warm climates. Soilborne pathogens causing wilt, damping-off and root rots of chickpea do not produce functional secondary inoculum that induces secondary infection in neighbouring plants in the same season (Fry, 1982). Such pathogens become severe only when large amounts of initial inoculum are present in the soil.

Fig. 9.2. Dry root rot of chickpea showing discoloration and breakage of the brittle tap root caused by *Macrophomina phaseolina* (Photo courtesy of ICRISAT)

Therefore, these diseases can be effectively managed by reducing the amount of initial inoculum in the soil.

Root diseases caused by *P. ultimum* and *S. rolfsii* can be severe in most soils (Cook and Papendick, 1972), however, dry root rot caused by *M. phaseolina*, usually becomes severe when plants are subjected to water stress. Disease development is favoured by dry soil conditions and temperatures around 30°C especially at flowering (Singh and Mehrotra, 1982). Sclerotia may survive for several years in the soil. *S. rolfsii* is more important where chickpea is sown after rice. Sclerotia formed on undecomposed rice residues in the field are the primary inoculum sources for disease and are capable of initiating severe infection (Haware *et al.*, 1990). Chlamydospores are the main survival structures of *F. solani* in naturally infested field soil. Chlamydospore germination occurs within 20 h after seeds are

Fig. 9.3. Black root rot of chickpea caused by *Fusarium solani* (Photo: courtesy of ICRISAT).

planted in soil with at least 9% soil moisture. Yield reduction caused by fusarium root rot is enhanced by inadequate rotation, soil temperatures of 25–30°C, soil moisture levels of −5 to 12 atm, extreme soil compaction, soil acidity (pH 5–6), and low soil fertility (Kraft *et al.*, 1981).

Losses

No precise information is available on yield losses due to root rots, although estimates of losses due to both wilt and root rots in Spain have been mentioned under fusarium wilt.

Management

The most important component of disease management for root rots is disease-free, good quality seed (Haware *et al.*, 1990). Seed quality greatly influences chances of seed and seedling infection. Poor quality chickpea seed with split seed coat exudes more water-soluble and volatile exudates than does healthy seed and is more stimulatory to *Pythium* spp. Fungicides can make a significant contribution to suppression of seed inoculum and seed infection. Chemicals can suppress the amount of initial infection induced by *Fusarium*, *Pythium* and *Rhizoctonia* spp. Seed treatments with metalaxyl (0.3 g kg^{-1}) and captan (3 g kg^{-1}) are effective in preventing seed rot and pre-emergence damping-off caused by *Pythium* spp. (Kaiser and Hannan, 1983). Seed treatment with tolclophos-methyl alone at 3 g kg^{-1} and in a mixture of tolclophos-methyl and thiram in 1:1 proportion (3 g kg^{-1}) are effective in controlling collar rot of chickpea (Haware and Narayana Rao, 1994). Strategies need to be developed to incorporate traditional practices of good soil management into modern agriculture without loss of productivity. In traditional agriculture, fallow and crop rotation are widely used cultural practices which reduce root rot intensity. Rotation is very effective for wilt pathogens with restricted host ranges. Pathogens such as *S. rolfsii* and *M. phaseolina* have broad host ranges, and crop rotation with a non-host may be difficult. High soil moisture is especially important in favouring infection by *S. rolfsii* and *P. ultimum* (Cook and Papendick, 1972). Therefore, waterlogging in fields should be avoided to reduce plant mortality. Cultural practices including roguing, increasing plant spacing, eliminating weed hosts and removing crop residues from the soil surface will reduce damage from root rot. Seed treatment with conidia of *Penicillium oxalicum* Sacc. significantly reduced seed rot and pre-emergence damping-off of chickpea caused by *P. ultimum* (Kaiser and Hannan, 1984). Soil application of *Trichoderma harzianum* Rifai integrated with seed treatment with carboxin or ziram resulted in 63% control of mortality due to *R. solani*, *S. rolfsii* and *F. oxysporum* f. sp. *ciceris* (Kaur and Mukhopadhyay, 1992).

Resistance to *M. phaseolina* has been reported (Haware *et al.*, 1990); however, even resistant cultivars may develop disease if grown in infected soil for a long period. Several sources of combined resistance to wilt and dry root rot have been identified through multilocation screening. These are ICCS 2862, 9023, 10803, 11550 and 11551 (Nene *et al.*, 1989; Haware *et al.*, 1990). Such resistances are being incorporated into breeding programmes and integrated management strategies. Resistance to pre-emergence damping-off caused by *P. ultimum* has been identified in *desi* types (Jagdish Kumar *et al.*, 1991) and is inherited polygenically. It will be difficult to develop combined resistance to all root rotting pathogens or a single control methodology with wide application. Integration of appropriate control measures is essential to manage such pathogen complexes (Jiménez-Díaz and Trapero-Casas, 1985).

ASCOCHYTA BLIGHT

Aetiology

Ascochyta rabiei (Pass.) Lab., the causal agent of blight, was first named *Zythia rabiei* by Passerini on the basis of its unicellular pycnidiospores (Khune and Kapoor, 1980). Comes in 1891 named the fungus *Ascochyta pisi* Lib. while Trotter in 1918 concluded that the fungus was not a species of *Ascochyta* and proposed the name *Phyllosticta rabiei* (Pass.) Trotter (Khune and Kapoor, 1980). Later, because of the ability of the fungus to produce 2–4% single-septate spores, Labrousse (1931) suggested that the pathogen should be called *Ascochyta rabiei* which is now accepted by the majority of pathologists.

Kovachevski (1936) recorded *Mycosphaerella rabiei* (syn. *Didymella rabiei* (Kovachevski) Arx) on overwintered straw. According to him, when ascospores were plated, cultures producing pycnidia were obtained (Holliday, 1980). Punithalingam and Holliday (1972) suggested that until further clarification of the perfect–imperfect state association, the chickpea pathogen should be retained under *A. rabiei* (Holliday, 1980). In a detailed study, Trapero-Casas and Kaiser (1992a) clearly proved the relationship between the perfect state and the imperfect state of *A. rabiei* under both field and laboratory conditions and confirmed the identity of the perfect state as *Didymella rabiei*. In Spain, Navas-Cortes *et al.* (1995) reconfirmed the relationship through similar studies. The perfect state has been found in a number of other countries (Nene, 1982; Nene and Reddy, 1987).

Biology

Ascochyta blight is one of the most important diseases of chickpea in West Asia, North Africa and the Mediterranean region (Nene and Reddy, 1987). The best documented account of blight epidemics exists for the former Punjab province of British India, now a part of Pakistan, where the disease was first observed in 1911 (Butler, 1918). Records of subsequent epidemics have been reviewed by Kausar (1965). In recent years, epidemics of the disease have been reported from Pakistan (Nene, 1984) and India (Singh and Kapoor, 1986). Blight is now reported from at least 35 countries (Nene *et al.*, 1996). Changing the date of sowing from spring to winter in the Mediterranean region, resulted in severe epidemics of blight (Hawtin and Singh, 1984). Ascochyta blights of various food legumes are reviewed in Chapters 4, 5, 6, 7 and 8, this volume.

Most workers have reported *Cicer* spp. to be the only hosts of *A. rabiei* (Nene, 1982, 1984). However Kaiser (1973) reported that the pathogen could infect cowpea and common bean when inoculated artificially. In contrast, Sprague (1930) was unable to produce symptoms on common bean under artificial conditions.

A. rabiei produces pycnidia on both chickpea and on artificial media. The dark brown, minute bodies are embedded in diseased tissues on stems, leaves, pods and seed. Pycnidia are immersed becoming erumpent, globose and 65–245 µm in size (Holliday, 1980; Nene, 1984). The pycnidial wall is composed of one to

two layers of elongated, pseudoparenchymatous cells; the ostiole is 30–40 μm wide. Pycnidiospores (also called conidia or spores) are hyaline, oval to oblong, straight or slightly curved at one or both ends, non- to one-septate, some slightly constricted at the septum, rounded at each end and measure 10–16 × 3.5 μm. They are formed on hyaline, ampulliform phialides. The growth of the fungus on potato dextrose agar at 20–25°C is initially creamy to pinkish in colour turning darker with age. Pycnidia are formed within 4–5 days and appear as black concentric rings in culture. The optimum temperature for growth, pycnidial production and spore germination has been reported to be 20°C (Nene, 1982, 1984). Kaiser (1973) noted increased sporulation under continuous light.

D. rabiei produces dark brown to black, globose or applanate pseudothecia with a hardly perceptible beak and ostiole, 70–150 × 120–250 μm in size (Kovachevski, 1936; Nene, 1982). Asci are cylindrical–clavate, more or less curved, pedicellate, 48–70 × 9–14 μm in size with eight ascospores which are monostichous, rarely distichous, ovoid, divided into two very unequal cells, strongly constricted at the septum, 12.5–19 × 6.7–7.6 μm in size. Until recently, perithecia had been found only on overwintered chickpea refuse under field conditions (Nene, 1982). However, Trapero-Casas and Kaiser (1992a) were able to induce production in the laboratory under conditions of high moisture and temperatures of 5–10°C. It is clear that cold temperatures are critical for the production of the perfect stage.

A. rabiei shows variability in morphological and physiological characters including colony colour, growth rate and size of pycnidia and pycnidiospores (Luthra *et al.*, 1939; Kaiser, 1973; Quareshi and Alam, 1984; Chaube and Mishra, 1992). Considerable pathogenic variability in *A. rabiei* has been reported (Vir and Grewal, 1974; Quareshi and Alam, 1984; Reddy and Kabbabeh, 1985; Gowen *et al.*, 1989; Singh 1990). Gowen *et al.* (1989) noted that the pathogenicity of isolates from the Indian subcontinent and West Asia was greater than that of most isolates from the western Mediterranean. Some workers have postulated that races of the pathogen exist (Luthra *et al.*, 1939; Singh *et al.*, 1981; Grewal, 1984; Nene, 1984; Porta-Pulgia *et al.*, 1986; Singh and Reddy, 1990, 1993). Riahi *et al.* (1990) developed a quantitative scale for assessing the reaction of chickpea to *A. rabiei*. The linear infection index was based on a quantitative measurement of disease expression and satisfactorily separated resistant and susceptible plants.

A review of these studies indicates no clear-cut differential interactions between isolates of *A. rabiei* and host genotypes. In order to analyse many of the data sets to prove the existence of races, it has been necessary to group together different disease scores. It appears that differences in aggressiveness may have been confused with differences in virulence. The problem of fungal variability and the existence of 'races' is very complex. Intensified studies are needed to understand and characterize the extent of variability in the pathogen and to determine how this variability is generated if a breeding strategy is to placed on secure grounds. A standard set of well-characterized genotypes, a common inoculation technique, and a well-defined disease-rating methodology should be used by workers who wish to determine the extent and distribution of variability in *A. rabiei* in different geographic regions.

The potential of DNA fingerprinting for characterizing variability in *A. rabiei* was evaluated by Weising *et al.* (1991) who demonstrated considerable DNA polymorphisms in *A. rabiei* isolates which allowed distinction between isolates using optimal enzyme/probe combinations. Using restriction fragment length polymorphisms (RFLPs) and RAPD analyses, Klein-Bölting and Barz (reported in Barz *et al.*, 1993) reconfirmed the results of Weising *et al.* (1991). Recent work has shown that molecular techniques can be used to distinguish super-aggressive isolates from moderately and weakly aggressive isolates (ICARDA, 1995). Further work may allow characterization of the geographical distribution of isolates and variability in pathotypes.

Symptoms

All above-ground parts are attacked. Initial symptoms of blight are expressed as water-soaked lesions on stems and leaflets which turn into sunken, dark brown lesions (Nene, 1982). The disease appears at any growth stage as patches in the field depending on the climate. Seedlings raised from infected seed show symptoms at the base of stems. These may remain restricted in the absence of high humidity and cool temperatures. Under favourable microclimatic conditions, brown to dark brown elongated lesions appear on the stems (Fig. 9.4). The lesions may girdle the stems which may break at the girdle. On leaflets, the lesions develop into well-defined, round or elongated, brown to dark brown spots which are sunken in the centre and surrounded by a reddish margin (Fig. 9.4). Under favourable climatic conditions, these lesions enlarge rapidly and coalesce, blighting the foliage. Lesions on pods are prominent and usually circular with dark margins (Fig. 9.4). On petioles and stems, lesions are brown and elongated and may girdle the infected portion. Dark pycnidia in concentric circles can be observed partly embedded in host tissues. Under severe infection, the entire plant dries. The developing seed is small and wrinkled and may have dark brown lesions which are especially prominent on white seed (Haware *et al.*, 1986).

Epidemiology

Epidemics of blight are favoured by temperatures of 10–20°C and moderate to high relative humidity (more than 60%) (Nene, 1982, 1984; Reddy and Singh, 1990b). In a detailed field study, Weltzien and Kaack (1984) found that blight development was favoured by temperatures of 9–24°C and wetness periods of 10 h or more. Under controlled conditions, Trapero-Casas and Kaiser (1992b) noted that severe infection by *A. rabiei* in chickpea occurred at an optimum temperature of 20°C and 17 h of leaf wetness. Wet, windy conditions are critical for rapid disease spread.

The frequency and success of epidemics of *A. rabiei* is at least partly related to efficient mechanisms of survival of the pathogen from season to season (Nene, 1982). The pathogen can survive in infected plant debris and seed (Nene, 1982; Haware *et al.*, 1986). Under controlled conditions, *A. rabiei* remained viable for

Fig. 9.4. Lesions of stems, leaves and pods of chickpea caused by *Ascochyta rabiei* (Photo: courtesy of ICRISAT).

more than 2.5 years in debris of infected chickpea plants kept at 4–35°C with relative humidity of 30–40% (Kaiser *et al.*, 1987). Reports from India (Luthra *et al.*, 1935), Greece (Zachos *et al.*, 1963) and Iran (Kaiser, 1973) indicate that the fungus can survive for at least 2 years in plant debris on the soil surface. However studies conducted in Syria, report survival for only 8 months (ICARDA, 1993). The pathogen loses viability rapidly under high relative humidity (60–100%) and at soil depth (10–40 cm) (Kaiser, 1973) and apparently survives well in debris only at the soil surface if conditions are dry. Weltzien and Kaack (1984) concluded that plant debris is a very inefficient soilborne source of inoculum. Further studies on the ability of the fungus to survive in plant debris are needed.

In recent studies in Spain, it was shown that *Didymella rabiei*, the perfect stage of *A. rabiei*, can grow saprophytically on infected chickpea tissues left on the

soil surface and remain viable for at least 2 years (Navas-Cortés *et al.*, 1995). When the debris was buried, *D. rabiei* was restricted to original lesions and remained viable for only 2–5 months. Nene (1982) noted that if cold is a prerequisite for production of the sexual stage, it would be unlikely that the sexual stage would be observed in the agroclimatic regions where chickpea is grown in the Indian subcontinent as hot summers follow the chickpea season.

Infected chickpea seed is an efficient method of survival and dissemination into new areas. Considerable research has been done on survival of the fungus in seed (Nene, 1982, 1984). The fungus may be present both on the seed surface and within the seed coat and cotyledons. Pycnidiospores obtained from pycnidia from 14-month-old seed stored at 3°C showed 33% germination (Maden *et al.*, 1975). Weltzien and Kaack (1984) considered infected seed to be a very efficient source of inoculum. Pycnidiospores of *A. rabiei* produced in crop residues and from diseased seedlings produced from infected seed at the beginning of the growing season are the primary inoculum for disease development and spread (Luthra *et al.*, 1935; Kaiser, 1973; Haware *et al.*, 1986). Secondary spread depends on pycnidiospores produced on diseased plants.

Diekmann (1992) analysed climatic data from chickpea growing areas to identify parameters that allowed discrimination of locations with or without occurrence of blight. A linear discriminant function based on mean daily temperature, mean precipitation and mean number of rainy days in the first and/or second months of the growing season could be used to predict blight risk for various agrogeographical zones and seasons. The model can help to concentrate disease control measures, such as quarantine, on high risk areas or identify areas or seasons for production of healthy seed (Diekmann, 1992).

Losses

There are many reports of serious losses caused by blight (Nene, 1982; Nene and Reddy, 1987). Yield losses of 25–70%, 20–50%, up to 100%, 10–20%, 40% and 5–30% are given for Pakistan (Sattar, 1933; Nene, 1982; Nene and Reddy, 1987), Bulgaria (Kovachevski, 1936), the former USSR, Greece (Demetriades *et al.*, 1959), Tunisia and Syria (Nene and Reddy, 1987), respectively. Reddy and Singh (1990a) studied the relationship between blight severity and yield loss. Yield losses of 10%, 16%, 27% and more than 80% were recorded in chickpea genotypes which were slightly, moderately, severely or totally affected by blight.

Management

The most important component of blight management is to reduce or prevent the entry of primary inoculum to the field. Pathogen-free seed with high germinability and the ability to produce vigorous plants is the first prerequisite of an effective disease control programme. Sattar (1933) was the first researcher to try to eradicate seedborne inoculum of *A. rabiei*. He reported good control with both copper sulphate and hot water treatment. Zachos (1951) observed that hot water

adversely affected seed germination. Many recommendations have been made concerning efficacy of various seed treatment chemicals to eradicate inoculum of *A. rabiei* from chickpea seed (Nene, 1982).

Chemical seed treatment and prophylactic sprays suppress the amount of initial inoculum induced by polycyclic pathogens like *A. rabiei*. A mixture of 11% tridemorph and maneb as seed treatment was reported to eradicate seedborne inoculum of *A. rabiei* (Reddy, 1983). Thiabendazole seed treatment (3 g kg^{-1} seed) has been reported to be more effective and safer than tridemorph and maneb. Seed treatment with an effective fungicide will control blight economically and allow the free movement of healthy seed internationally without danger of introduction of blight to new areas (Kaiser and Muehlbauer, 1988). The need to use clean seed directly or to disinfect through simple and effective seed dressings cannot be overemphasized (Nene, 1982; Kaiser and Hannan, 1985).

Foliar applications of zineb, maneb and daconil have been reported to significantly reduce disease intensity (Nene, 1982; Bashir and Ilyas, 1983). As many as four to six foliar sprays may be necessary to reduce disease significantly. When the blight appears in a field, however, disease development may be rapid under favourable environmental conditions and foliar fungicidal applications may not be effective. Foliar sprays with presently available fungicides have limited scope and are of no relevance for resource-poor farmers, especially in dry areas where water is limiting.

Sattar (1933) suggested that removal and destruction of crop debris, crop rotation and deep sowing of seed (to prevent infected seed from emerging) could reduce blight. In addition to sanitation, Luthra *et al.* (1935) suggested intercropping chickpea with non-hosts such as wheat, barley and mustard could reduce disease spread. In traditional agricultural systems, fallow, summer ploughing, crop rotation and intercropping are widely used and probably have an effect on reducing blight and soilborne diseases. Rotation is a very effective way to reduce the primary inoculum of *A. rabiei* since the pathogen has a relatively narrow host range. Adopting specific cultural practices could help especially where there is group action by all farmers of a region (Nene, 1982).

A considerable amount of research has been done by ICRISAT and the International Centre for Agricultural Research in Dry Areas (ICARDA), Aleppo, Syria, on developing techniques and screening for resistance to blight. Research was intensified after the epiphytotics of 1981 and 1982 (Reddy and Singh, 1984; Nene and Reddy, 1987). At ICARDA, over 19,000 germplasm accessions of chickpea have been evaluated for resistance to six isolates of *A. rabiei* between 1979 and 1991. Only three *desi* accessions (ICC 4475, ICC 6328 and ICC 12004) and two *kabuli* accessions (ILC 200 and ILC 6482) showed resistance during repeated greenhouse and field screening (Singh and Reddy, 1993). Mutilocational evaluation of chickpea germplasm and breeding lines for resistance to blight in 48 locations throughout 20 countries indicated that *kabuli* germplasm generally shows higher resistance to blight than *desi* germplasm (Reddy *et al.*, 1992; Haware *et al.*, 1995a, b). From the material generated at ICARDA, 29 blight field-resistant cultivars have been released in 14 countries (Singh, 1993). In India, lines with moderate levels of resistance have been identified (Singh, 1989). There is a need to quantify the level of yield gained by the use of these lines in comparison to susceptible lines.

Resistance to blight is partial and the existence of immunity has not been confirmed (Allen, 1983). Studies reviewed in Nene (1982) suggested that resistance was governed by a single dominant gene. Subsequent studies (Boorsma, 1980; Pieters, 1984) reported that blight resistance is quantitatively inherited. Van Rheenen and Haware (1993) noted that resistance against blight was quantitative with a significant vertical component, while Dey and Singh (1993) observed that resistance was governed by different genes in different cultivars: for example, by two dominant complementary genes in GLG 84038 and GL 84099 and by one dominant and one recessive independent gene in black-seeded ICC 1468. Rate-reducing resistance to blight has been shown in two chickpea cultivars (Reddy and Singh, 1993). From the varied and often contradictory results obtained to date, further work on the inheritance of resistance to blight appears essential to developing a sound breeding strategy. Very little is known of the mechanisms which underlie blight resistance although a correlation with seed coat colour has been established. Resistant genotypes are predominantly black-seeded which suggests that pigment may be associated with blight resistance (Allen, 1983). Hafiz (1952) noted that penetration of the pathogen in two resistant cultivars was delayed possibly due to the greater secretion of malic acid from the dense covering of glandular trichomes in the resistant cultivars; however, follow-up work was not able to substantiate these findings (Nene, 1982).

High levels of resistance to blight are available in wild *Cicer* species including accessions of *C. bijugum*, *C. judaicum* and *C. pinnatifidum* (Haware *et al.*, 1992a; Singh *et al.*, 1992). With biotechnology tools, the utilization of resistance genes from wild *Cicer* spp. to improve resistance in chickpea could be useful if the genetic base of resistance is different to that in the crop and once transformation techniques for chickpea have been perfected. Several research groups in Europe are presently attempting to move genes for inhibiting production of polygalacturanase and cutinase into chickpea for resistance to blight (G. Ramsey, SCRI, UK, 1996, personal communication).

GREY MOULD

Aetiology

Botrytis cinerea Pers., the causal agent of grey mould in chickpea, was first reported on chickpea by Shaw and Ajrikar (1915). It is regarded as an aggregate species with the associated teleomorph *Botryotinia fuckeliana* (de Bary) Whetzel (Grooves and Loveland, 1953), however, it should be noted that for each disease caused by *B. cinerea* the teleomorph would not necessarily be *B. fuckeliana*. In fact, the teleomorph has not been reported on chickpea.

Biology

Grey mould is an important disease of chickpea in northern India, Nepal, Bangladesh and Pakistan (Haware and McDonald, 1992). It was first reported

from India in 1915 (Shaw and Ajrikar, 1915) and has since been reported from more than 15 countries (Nene *et al.*, 1996), mostly in Asia and North Africa but also in Australia and the Americas (Carranza, 1965; Corbin, 1975; Kharbanda and Bernier, 1979; Sepulveda and Alvarez, 1984). Grey mould is also an important disease of lentil (see Bayaa and Erskine, Chapter 8, this volume).

B. cinerea is a necrotrophic fungus well known for its extensive host range, wide distribution globally, extreme variability and adaptability to a wide range of environmental conditions. The fungus grows profusely on dead flowers and potato dextrose agar. Initially, growth is white and cottony, turning light grey with age (Ellis and Waller, 1974). Some strains of *B. cinerea* produce small, black sclerotia of varying size and shape. The mycelium is septate, hyaline to lightly pigmented and branched, sometimes dichotomously, near the apex. Conidia are hyaline, one-celled and oval, measuring 4–20 × 4–6 μm in culture, and are borne in clusters on short sterigmata (Ellis and Waller, 1974). Conidia in mass are ash grey in colour. They germinate easily in water forming a thin, hyaline germ tube. Sporodochia are formed on host tissues. Small (4–8 μm) unicellular, round, microconidia are formed on the sporodochia. These conidia do not germinate; sporodochia cease to produce spores and develop into a sclerotial mass.

B. cinerea is a facultative saprophyte and infects a wide range of crops including chickpea, faba bean, pea, lentil, grape, apple, strawberry and many vegetables (Rathi and Tripathi, 1993). Isolates can be grouped on the basis of sclerotia and spore formation. There have been few studies on physiologic specialization in *B. cinerea* isolates from chickpea. Singh and Bhan (1986) identified four physiological races based on the reactions of nine isolates on nine chickpea lines. Rewal and Grewal (1989) divided six strains of *B. cinerea* infecting chickpea into five distinct pathotypes on the basis of their reaction on five chickpea lines. Experience suggests that such a variable pathogen with such a wide host range would be unlikely to exhibit strong physiological specialization. It is clear that further work is necessary to characterize the variability in the host–pathogen interaction.

Recent studies have focused on the use of molecular techniques to characterize variability in the fungus. Using RAPD markers, Chung *et al.* (1996) compared 34 isolates of *B. cinerea* from nine hosts in Korea. All isolates except one showed different molecular phenotypes and there was no relationship with host, geographic origin, year of isolation, and pathogenicity. The RAPD data suggested the existence of high levels of genetic variability in populations of *B. cinerea* in Korea (Chung *et al.*, 1996). Similar studies have been done with populations of the fungus from Spain (Alfonso *et al.*, 1996).

Symptoms

The pathogen infects all aerial parts of the plant, flowers being the most susceptible (Haware *et al.*, 1986; Haware and McDonald, 1992). Flower drop is common, resulting in poor pod formation. Initial symptoms on the leaves, stems and pods are expressed as grey to dark brown lesions covered with hairy sporophores and masses of single-celled, hyaline spores (Fig. 9.5). Grey fungal growth is evident on

Fig. 9.5. Grey fungal growth and profuse sporulation of *Botrytis cinerea* on petioles and leaves of chickpea (Photo: courtesy of M.P. Haware).

flowers and petioles on cloudy days if observed early in the morning. Drooping of affected, tender, terminal branches is a common field symptom. Under cloudy weather, rotting of foliage and flowers is conspicuous. The affected foliage is discoloured and dries, becoming greyish (Haware *et al.*, 1986; Haware and McDonald, 1992). Under favourable weather conditions of high humidity and moderate temperature (20°C), discrete brown spots develop on the leaves and circular to elongated spots form on the branches. Chlorosis and defoliation occur at higher temperatures. Sometimes, tiny dark brown to black sclerotial masses appear on dead tissue. These sclerotia should not be confused with the large dark brown sclerotia of *Sclerotinia sclerotiorum* (Lib.) de Bary which are usually embedded in a white mycelial mat covering the chickpea stem (Joshi and Singh, 1969). In cases of flower infection, no pods are formed. If pods are affected, small, shrivelled seed is formed.

Epidemiology

The fungus infects the aerial parts of the plant and produces masses of spores on infected tissues which can cause secondary spread of the disease (Haware and McDonald, 1992). As with many diseases caused by *B. cinerea*, rapid disease development over time and space is environmentally driven (Marois, 1996). Relative humidity and temperature are the most important factors determining infection and spread of grey mould in chickpea. Under conditions of 95% or above relative humidity during day temperatures of 22–25°C in a dense foliage canopy, infection and spread can occur rapidly (Haware and McDonald, 1992, 1993; Rathi and Tripathi 1993). Heavy rainfall during vegetative growth leads to increased disease severity. Under optimum conditions of 19°C and more than 95% relative humidity, the entire disease cycle can be completed in 7 days (Marois, 1996).

It was considered that conidia require free moisture on the plant surface for germination and infection (Haware and McDonald, 1992). Recent studies have shown that dry conidia of *B. cinerea* can germinate at high relative humidity (Williamson *et al.*, 1995; Cole *et al.*, 1996). Pollen has been found to stimulate germination of conidia (Chou and Preece 1968; Borecka and Millikan, 1973) which supports the observations that chickpea is commonly and seriously affected during the flowering stage.

The fungus survives on infected chickpea seed and is also thought to survive on plant debris (Laha and Grewal, 1983; Haware *et al.*, 1986; Singh and Tripathi, 1992). Seedborne inoculum is important in Australia (Cother, 1977). The fungus may remain viable in infected seed and plant debris stored at 18°C for 5 years (Grewal, 1988). As *B. cinerea* infects a very wide range of plants, some of which are perennial, the role of alternative hosts in the survival of the fungus and infection of chickpea from one season to another also needs to be studied. The relative epidemiological importance of seedborne inoculum and other sources of inoculum such as plant debris produced by *B. cinerea* has not been fully elucidated for grey mould of chickpea (Haware and McDonald, 1993). It is therefore difficult to predict the likelihood of epiphytotics. Further work on the epidemiology of grey mould in chickpea is clearly warranted.

Losses

The economic importance of grey mould in India was first realized during the cropping season of 1978/79 when the disease destroyed 20,000 ha of chickpea in the states of Punjab, Haryana, Uttar Pradesh and Bihar in India, as well as in parts of Pakistan (Grewal and Laha, 1983; Grewal, 1988). Since then, the disease has occurred in epiphytotic form on several occasions in most northern states of India causing serious losses up to 70–100%. Similarly, in Bangladesh, losses caused by grey mould were estimated to be 80–90% in 1988; 70–80% in 1989 (Bakr and Ahmed, 1992) and a survey in 1992 to 1993 found 100% losses in many farmers' fields. In Nepal, grey mould occurs every year and annual yield losses on farm are estimated to be at least 15%. Carranza (1965)

reported a 96% crop loss in the Jujuy Province of Argentina. Grey mould is therefore one of the most important diseases affecting chickpea in regions where the crop is grown primarily by resource-poor farmers.

Management

Over 6000 chickpea lines have been screened at ICRISAT for resistance to *B. cinerea* with limited success. Lines with moderate resistance to grey mould have been identified (Haware and Nene, 1982; Rathi *et al.*, 1984; Haware and McDonald, 1993) but these studies have not revealed any line with a high level of resistance. This is not surprising, as attempts to find high levels of resistance to grey mould in other crops during the past 50 years have also been unsuccessful. Some erect chickpea morphotypes have been identified – ICCL 87322 and ICCV 88510 – which are only slightly affected by grey mould under moderate disease pressure (Haware and McDonald, 1993). Although detailed studies are still in progress, it appears that such morphotypes are escaping grey mould damage due to the open canopy architecture which encourages air movement to allow rapid drying of foliage after rainfall. This apparent escape mechanism reduces the rate of grey mould build-up as high humidity encourages infection of growing shoots, leaves, flowers and developing pods by B. *cinerea*. Plant architecture expressed as canopy density is also important in phaseolus bean for escaping white mould damage caused by *Sclerotinia sclerotiorum* (Schwartz *et al.*, 1978; Blad *et al.*, 1978). A similar finding was made for loose versus tightly clustered grapes affected by *B. cinerea* (Vail and Marois, 1991). However, even the protection afforded by plant morphotype is insufficient under high disease pressure and integrated strategies for managing the disease are being developed.

Thirty-six accessions belonging to seven annual wild *Cicer* spp. were evaluated for reaction to grey mould in a controlled environment growth room at ICRISAT, India. Only three accessions of *C. bijugum* (ICCW 41, 42 and 91) were found to possess good resistance (Haware *et al.*, 1992a). ICCW 41 and 42 were also resistant to *A. rabiei*. Further work is in progress to assess the potential of using this resistance.

Seed treatment with triadimefon (1 g kg^{-1} seed) followed by mancozeb (3 g kg^{-1}), or triadimenol (1 g kg^{-1}) or thiabendazole (1 g kg^{-1}) or carbendazim and thiram (3 g kg^{-1}) is effective in eradicating *B. cinerea* from seed (Singh and Bhan, 1986). Field trials conducted at Pantnagar, India, indicated that two to three foliar sprays of vinclozolin (0.2%) could effectively control grey mould in chickpea (Grewal and Laha, 1983; Haware and McDonald, 1993).

Field trials have been conducted at Pantnagar, India, from 1991 onwards to test various components of an integrated management strategy for grey mould of chickpea (Haware and McDonald, 1992, 1993; Reddy *et al.*, 1993; Haware *et al.*, 1996a). Delayed sowing resulted in low levels of disease, even in susceptible cultivars. However there was significant reduction in yield in late sown plots (Table 9.5). It may be possible to manage grey mould of chickpea by manipulating date of sowing but superior chickpea genotypes that can perform better when sown

Table 9.5. Influence of date of sowing and growth habit of chickpea genotypes on grey mould severity and grain yield, Pantnagar, India.

Cultivars	DS I[1]		DS II		DS III		DS IV	
	Disease rating[2]	Plot yield (kg)	Disease rating	Plot yield (kg)	Disease rating	Plot yield (kg)	Disease rating	Plot yield (kg)
H 208	7.7[3]	0.9	6.7	1.1	4.7	1.0	3.7	1.0
Pant G-114	6.3	0.9	5.7	1.0	3.3	0.9	3.3	1.3
K 850	6.0	1.0	6.0	1.1	4.3	0.9	3.3	1.3
ICCV 88510	5.3	1.2	5.0	1.0	4.0	0.7	2.3	1.2
ICCL 87322	4.7	1.2	4.7	1.1	3.3	0.9	2.3	1.1

Standard error

	Disease rating	Plot yield (kg)
Cultivar	±0.169**	±0.086
Sowing	±0.152**	±0.077**
Cultivar × Sowing	±0.339	±0.173
Coefficient of variation (CV) (%)	12.6	31.6

[1] Dates of sowing; DS I = 31 Oct. 1992; DS II = 14 Nov. 1992; DS III = 29 Nov. 1992; DS IV = 14 Dec. 1992.
[2] Disease rating on 1–9 point scale.
[3] Mean of three replications.
** Significant at 1%.

late need to be identified. Using the erect cultivar ICCL 87322 at lower planting density, there was less grey mould and higher grain yield than in dense plantings in both sprayed and unsprayed treatments (Reddy *et al.*, 1993; Haware *et al.*, 1996). This indicated that it is possible to manage grey mould in chickpea by sowing erect, moderately resistant genotypes at acceptable density under a judicious regime of fungicide use (Table 9.6) (Haware and McDonald, 1993; Reddy *et al.*, 1993; Haware *et al.*, 1996a). Similar observations have been made for lentil (see Bayaa and Erskine, Chapter 8, this volume).

The need to summarize and synthesize available information on the contribution of agronomic practices is critical to developing integrated management of grey mould. A wide range of practices including date of planting, spacing, nutrition, intercropping, mixed cropping and detopping have been studied.

Table 9.6. Effect of row spacing and fungicide sprays on grey mould severity and grain yield in chickpea, Pantnagar, India.

Treatment	Cultivar	Spacing	Disease severity (1–9 scale)	Yield[1] (kg ha^{-1})
Sprayed[2]	ICCL 87322	30 × 10	4.3	1192
		60 × 10	3.3	1291
		45:15:45	4.3	1200
		60:40:60	3.3	1176
	H 208	30 × 10	5.7	1265
		60 × 10	4.7	1139
		45:15:45	4.7	1157
		60:40:60	4.7	1086
Non-sprayed	ICCL 87322	30 × 10	5.7	989
		60 × 10	4.3	989
		45:15:45	4.3	911
		60:40:60	4.0	1044
	H 208	30 × 10	8.0	459
		60 × 10	6.3	349
		45:15:45	6.3	392
		60:40:60	6.0	399
Standard error				
Cultivar			±0.124*	± 85.1
Spacing			±0.175*	±120.4
Spraying			±0.124*	± 85.1*
Spacing × Spraying			±0.248	±170.2
Cultivar × Spacing			±0.248	±170.2
Cultivar × Spraying			±0.175**	±120.4**
Cultivar × Spraying × Spacing			±0.350	±240.7
Coefficient of variation (CV) (%)			12.1	45.4

* Significant at 1%; ** Significant at 5%.

[1] Mean of three replications.

[2] Vinclozolin (0.2%).

Further work is especially needed on the potential contribution of intercropping in reducing grey mould. It was recently shown that soil solarization can eradicate *B. cinerea* from soil (López-Herrera *et al.*, 1994). Additional gaps in our current knowledge need to be identified and priority areas for further research targeted.

Beneficial microorganisms are part of the biological resources available in nature that, with little manipulation, could be used to control plant diseases (Cook and Baker, 1983). Recently it was confirmed that spraying spores of *Trichoderma harzianum* Rifai on foliage in greenhouse experiments successfully controlled grey mould of chickpea (Plate 22) (Mukherjee *et al.*, 1995). Field experiments at Pantnagar and Hisar, India, are giving encouraging results (Haware *et al.*, 1996a). An integrated management package which involves the erect cultivar ICCL 87322, appropriate plant spacing and a combination of judicious use of foliar sprays of vinclozolin and a vinclozolin-resistant isolate of *T. harzianum* is being tested for the first time on farm in northern India during the 1996/97 cropping season (M.P. Haware, ICRISAT, India, 1996, personal communication).

Research at the Scottish Crops Research Institute (SCRI), UK, has shown that immature raspberry fruit contain a polygalacturonase-inhibiting protein (PGIP) effective against endo-polygalacturonases produced constitutively by *B. cinerea* (Johnston and Williamson, 1992a, b; Johnston *et al.*, 1993, 1994; Williamson, 1994). The PGI activity declines rapidly as the fruit ripens which is correlated with susceptibility to grey mould. Polygalacturonases are key enzymes in the invasion of plant tissues by many facultative fungal pathogens. A strategy for enhancing resistance to *B. cinerea* involves isolation of the PGIP gene and *Agrobacterium*-mediated transformation of cultivars and breeders' selections with constructs of the gene, using a constitutive promoter to ensure expression in all tissues (Williamson *et al.*, 1993). PGIP genes are presently being used for transformation of chickpea with the objective of reducing the serious losses caused by grey mould. This approach has already been successful in transformed tomatoes with a PGIP gene from pear (Stotz *et al.*, 1993; Powell *et al.*, 1994). Successful utilization of the PGIP genes for resistance to grey mould in chickpea will be a significant breakthrough in the management of this major disease.

STUNT

Aetiology and biology

Stunt is the most important and widespread virus disease of chickpea. It was first reported in Iran by Kaiser and Danesh (1971a, b) and the casual agent of stunt was originally attributed to pea leaf roll virus (Kaiser and Danesh, 1971a, b) which was found to be a synonym of bean leaf roll luteovirus (BLRV) (Brunt *et al.*, 1990). Chickpea stunt in India was also ascribed to BLRV (Nene *et al.*, 1978; Nene and Reddy, 1987) although the true identity of the virus first isolated has not been established (Horn *et al.*, 1996). Brunt *et al* (1990) refer to the chickpea

virus both as BLRV (pg. 111) and as chickpea stunt luteovirus (CpLV) (pg. 177). They indicate that CpLV is a probably a strain of BLRV but no comparative studies are reported. Recent studies have shown that a leafhopper-transmitted geminivirus is also capable of producing stunt of chickpea in India and Pakistan (Horn *et al.*, 1993). This hitherto undescribed virus has been named chickpea chlorotic dwarf virus (CCDV) (Horn *et al.*, 1993). Recent surveys of chickpea with stunt symptoms in both India and Pakistan and follow-up serological and electron microscope studies have shown that the aetiology of stunt disease is more complex than was previously thought. In contrast to previous studies, it has been shown that a geminivirus and, not one, but several luteoviruses including CpLV-like, beet western yellow (BWYV)-like virus and BLRV-like isolates cause similar, if not identical, symptoms in chickpea (Horn *et al.*, 1996).

Stunt occurs in almost all chickpea growing areas in the world, including North Africa, the Middle East, the Indian subcontinent, Spain, Turkey and the USA (Nene and Reddy, 1987; Nene *et al.*, 1996). In California, USA, additional luteoviruses such as subterranean clover red leaf virus, legume yellows virus and BWYV infect chickpea. In Spain, both BWYV and BLRV were reported to cause stunt of chickpea. Recently, faba bean necrotic yellows virus has also been noted to cause chickpea stunt in Syria. BLRV is an important disease of pea (see Kraft *et al.*, Chapter 6, this volume) and faba bean (see Jellis *et al.*, Chapter 7, this volume). Clearly more work is needed to elucidate the relationships between this virus and the causal agents of stunt on chickpea.

The relative prevalence of the luteoviruses appears to vary among different chickpea growing areas of the Indian subcontinent. CCDV and CpLV-like isolates were widely distributed in India and Pakistan whereas BLRV-like and BWYV-like isolates were of minor importance (Horn *et al.*, 1996). CCDV is the predominant virus causing stunt at the ICRISAT Center, Andhra Pradesh, and in chickpea growing areas in the states of Haryana and Rajasthan, India, and in Pakistan (Horn *et al.*, 1996). In contrast, luteoviruses are predominant in chickpea growing areas in the states of Madhya Pradesh and Gujarat, India. No detailed information is presently available on the distribution of the various causal viruses of stunt in other chickpea growing areas of the world.

BLRV is described as isometric, not enveloped particles of 27 nm in diameter (Brunt *et al.*, 1990). CpLV appears to be similar, being characterized by isometric, not enveloped particles, 27–29 nm in diameter (Brunt *et al.*, 1990). CpLV is, however, serologically distinct from BLRV (Horn *et al.*, 1996). The natural host range of BLRV includes common legume crops such as pea, phaseolus bean, faba bean, cowpea and lentil, as well as chickpea, lucerne and white clover. Apart from chickpea, pea is the only other known natural host of CpLV but it has a wide experimental host range including many of the legumes listed as natural hosts of BLRV (Brunt *et al.*, 1990). CCDV is an ssDNA-containing virus with mainly dimer but also trimer and tetramer isometric particles (Horn *et al.*, 1993). Dimer particles are 25 × 15 nm. It is serologically unrelated to common leafhopper-transmitted geminiviruses known to infect dicotyledons (Horn *et al.*, 1993). Through leafhoppers, CCDV could be successfuly inoculated to species of the *Leguminosae*, the *Solanaceae* and the *Chenopodiaceae* (Horn *et al.*, 1993).

Symptoms

Stunt disease is characterized by stunting of the plant, reduction in internode length together with phloem discoloration in the collar region (Nene *et al.*, 1978). If infection occurs early, the whole plant remains small and stunted. In late infection, growth reduction is seen at the tip of the plant only. In the field, stunted plants are conspicuous due to plant and leaf reddening in *desi* types (Plate 23) and leaf yellowing in *kabuli* types. Phloem browning is an important symptom, which can be observed by removing the bark at collar region. Stems and leaves of affected plants are thick and brittle. A transverse cut of the root shows a brown ring. Other tissues including the xylem of the root appear normal unless infected by root pathogens (Nene *et al.*, 1978). Stunting and leaf reddening or yellowing may be caused by phloem injury due to insects or moisture stress; however, in such cases, phloem discoloration will be absent.

Epidemiology

BLRV is transmitted by a number of aphid vectors (including *Aphis craccivora* Koch and *Myzus persicae* Subzer) in a persistent manner (Kaiser and Danesh, 1971a, b). It can be transmitted by grafting but not by mechanical inoculation or by seed or pollen. CpLV is reported to be transmitted in nature by *A. craccivora* in a persistent manner (Brunt *et al.*, 1990). Recent studies, however, have indicated that none of the luteoviruses associated with stunt could be transmitted by *A. craccivora* (S.V. Reddy and D.V.R. Reddy, unpublished results). However, BWYV-like isolates could be transmitted by *M. persicae*. Clearly more detailed work is required to identify the key natural vectors of the luteoviruses associated with stunt of chickpea. CCDV is transmitted by the leafhopper *Orosius orientalis* Matsumura which is also known as a vector of several phytoplasma diseases (Horn *et al.*, 1993). Leafhoppers could acquire and transmit the virus within 2 h. Mechanical transmission of CCDV was not successful.

Insect populations may play an extremely important role in the epidemiology of stunt (Kaiser *et al.*, 1990). During the recent survey in India and Pakistan, the incidence of CCDV and CpLV-like viruses in a few fields suggested that the spread and sources of infection was/were limited (Horn *et al.*, 1996). It was also observed that the incidence of stunt was greater in sparsely planted chickpea fields and where chickpea was grown in monoculture rather than in mixed cropping systems. The role of alternative hosts is unknown although weeds in some chickpea fields have been found to harbour both luteo- and geminiviruses. It is clear that further epidemiological research on stunt and the viruses and vectors associated with the symptomatology is greatly needed. At minimum, further surveys are needed in all countries where stunt occurs on chickpea and across seasons to better understand the seemingly complex distribution of the causal viruses and possible shifts in virus incidence and occurrence (Horn *et al.*, 1996).

Losses

For BLRV, field incidence is usually low but if infection occurs before flowering, yield loss can be as high as 80% (Allen, 1983).

Management

Most effort in managing stunt has been directed at identifying sources of resistance and developing stunt-resistant cultivars (Reddy *et al.*, 1979). The discovery of the association of more than one virus with the disease has complicated this strategy. Over 10,000 germplasm lines have been screened for resistance to stunt at Hisar, India, which is a hot-spot for CCDV. GG 669 and ICCC 10 are field resistant. Resistance was expressed as slower symptom development, compared to the susceptible line WR 315. Chickpea lines identified as resistant at Hisar showed 40–70% infection when screened at Junagadh, Gujarat. This was not surprising as luteoviruses predominate in Gujarat whereas the geminivirus predominates in Hisar (Horn *et al.*, 1996). Four wild *Cicer* species, *C. cuneatum*, *C. echinospermum*, *C. judaicum* and *C. reticulatum*, also expressed symptoms later than susceptible chickpea genotypes and were not severely stunted (N.M. Horn, S.V. Reddy and D.V.R. Reddy, ICRISAT, India, unpublished). Sources of resistance from wild species may be used in the future. Effective field screening for resistance to chickpea stunt viruses should include serological assaying of both susceptible and resistant genotypes and evaluation under greenhouse conditions against virus types and strains.

LOOKING AHEAD

Chickpea is a popular component of dietary protein in Asia. The bulk of the crop is grown in developing countries where resources available to farmers are limited. Foliar fungal diseases of chickpea are important in West Asia and North Africa, and on the Indian subcontinent, and root diseases are present in all chickpea growing areas. Seeds of resistant cultivars for many of these important diseases are still not available commercially. Efforts must be enhanced to provide seed.

In the absence of high levels of resistance to some of the diseases, various ways of reducing field inoculum should be used in integrated disease management system. Short-duration cultivars of chickpea mature earlier and thus may escape disease if sown late. Tall, erect cultivars do not allow humidity to build up in the crop canopy which reduces grey mould damage. To ensure the effectiveness of integrated disease management systems, we will have to fill gaps in our knowledge on several pathogens. Characterization and understanding of variablility in *A. rabiei* and the stunt virus complex is essential. The epidemiology of ascochyta blight and grey mould need further attention. Control measures will be more effective if our knowledge of the source of primary inoculum of these diseases is improved. Satisfactory progress has been made in identifying high to

moderate levels of resistance to individual diseases (Table 9.7). Both conventional and biotechnological-based germplasm enhancement for disease resistance may prove useful. Efforts should concentrate on the understanding and re-establishment of more genetic diversity in chickpea grown commercially.

Extensive consultation and research progress over the past 20 years has enabled us to identify the most important constraints in each region of chickpea production and a priority listing for disease management is given (Table 9.8). Research conducted to date has resulted in the development of resistant cultivars (Haware *et al.*, 1990; Jiménez-Díaz *et al.*, 1993; Singh, 1993). Resistance has generally been durable for wilt. Less success has been enjoyed with collar rot, ascochyta blight and grey mould which remain important production constraints. Combined resistance to several pathogens has been difficult to achieve but needs more effort (Table 9.7). Disease management systems which integrate the traditional management without reducing crop productivity, use of resistant/tolerant cultivars, production of healthy seed, improved cultural practices, strategic use of crop protection chemicals and biological control agents could provide more sustainable solutions to management of diseases of chickpea.

Table 9.7. Chickpea disease-resistant lines.

Wilt	: 160 lines (see Haware *et al.*, 1992b for details)
Wilt-resistant lines (high yielding)	: ICC 11223, -12265, -12269, -12969, ICCC 32
Wilt/dry root rot/black root rot	: ICC 12237, ICC 12269
Wilt/dry root rot/stunt	: ICC 12435
Wilt/dry root rot	: ICC 11315, -12241, -12257, -12268, -12270, -12271, -12273, -12437, -12444, -12450, -12454, -12460, -12467, -12472, -12428, -12430, -12435, -12440, -12452, -12470, -12471, -14372, -14374, -14376, -14396, -14440, -14442, -14443
Wilt/black root rot	: ICC 11313, -11316, -11317, -11320, -11324, -12236, -12237, -12239, -12242, -12245, -12249, -12255, -12256, -12258, -12259, -12269, -12274, -12275
Dry root rot	: ICC 4928, -11550, -14735, -15178, -15236
Wilt/stunt	: ICCL 83408, -86401, ICCV 88106, ICC 10136, -10805, -11502, -11551
Botrytis grey mould	: ICC 1069, -1918, ICCL 87322, ICCV 88510, GL 85056, GL 85103, GL 85105, GG 829, GL 90159, GL 91040
Ascochyta blight	: ILC 202, -3279, -3856, -196, -201, ICC 1467, -1468, -2160, -4616, -5033, NEC 138-2

Table 9.8. Regional distribution of chickpea diseases and priority listing for disease management.

Regions	Countries	Diseases[1]
South-east Asia	Bangladesh, Myanmar, India, Nepal, Pakistan	FW, RR, AB, ST, BGM, RK
West Asia	Afghanistan, Iran, Iraq, Syria, Turkey	AB, FW, ST, Cyst
North Africa	Algeria, Morocco, Tunisia	FW, AB, ST, SR
Southern Europe	Portugal, Spain	FW, AB, RR
Eastern Africa	Ethiopia	RR, FW
Southern Africa	Malawi, Tanzania	FW, RR
Central and South America	Chile, Colombia, Mexico, California, USA	FW, RR, ST

[1] FW – Fusarium wilt; BGM – Botrytis grey mould; RR – Root rots; SR – Stem rot; AB – Ascochyta blight; ST – Stunt; RK – Root-knot nematode; Cyst – Cyst nematode.

Source: Compiled by the author from extensive consultation with scientists at conferences and workshops over the past 10 years.

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DISEASES OF PIGEONPEA

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INTRODUCTION

Pigeonpea (*Cajanus cajan*) originated in peninsular India and probably spread quite early to other countries (van der Maesen, 1990). A secondary centre of diversity is found in eastern Africa. The genus *Cajanus* as accepted in the broad sense, including the former genus *Atylosia*, also has two centres of diversity with 17 species occurring in the Indian subcontinent and another 13 in Australia. Pigeonpea is an important grain legume crop of rainfed agriculture in the semi-arid tropics of the Indian subcontinent and is also widely grown in eastern and southern Africa, Latin America and the Caribbean (Nene and Sheila, 1990). India, Myanmar, Kenya, Uganda, Malawi, Tanzania and the Dominican Republic are the main producers with almost 90% of production from the Indian subcontinent. It is a multipurpose crop, being grown not only for grain but also for fuel and fodder. The decorticated, split, dried seeds are used as dhal and the green pods are cooked as a vegetable (Nene and Sheila, 1990). The seed husks, pod walls and green leaves are used as cattle feed. The dry stems are used for household fuel, field fences and making huts and baskets. Because pigeonpea is a hardy, multipurpose crop, it is popular with resource-poor farmers. It is mostly grown as an annual crop and, in a limited way, as a perennial on field bunds or in backyards. The medium- and long-duration types are typically grown in inter- or mixed-cropping systems with a variety of crops such as sorghum, millet, maize, groundnut, mung bean, cotton, castor and cassava in traditional agriculture systems in Asia and Africa (Nene and Sheila, 1990). To a limited extent, these types are also grown as a sole crop. Recently short-duration types have been developed which are primarily grown in monoculture.

Worldwide, the crop is cultivated on about 3.4 million hectares with an annual production of 2.7 million tonnes, an average yield at 790 kg ha^{-1} (Nene and Sheila, 1990). Because of its high protein content, pigeonpea is a significant

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component of the diet of vegetarians in the semi-arid tropics. As the present daily per capita protein consumption in this region is 35 g, which is much lower than the desired level of 50 g, pigeonpea has great potential to bridge this gap. But its production and productivity are constrained by several major diseases.

As for other leguminous crops covered in this book, pigeonpea is susceptible to many diseases and insect pests. Over 210 pathogens have been recorded on pigeonpea (Nene *et al.*, 1996) but only a few of them are widely distributed and of economic importance. Pigeonpea diseases have been extensively reviewed (Kaiser, 1981; Reddy *et al.*, 1990b; Ghanekar *et al.*, 1992; Singh and Chauhan, 1992; Upadhyay and Rai, 1992) and readers are referred to these reviews. In this chapter, we critically review the available information on the major diseases and their management. Management of pigeonpea diseases can be one of the most important means of improving productivity of the crop and the protein consumption of resource-poor people in the semi-arid tropics.

Available information on diseases is based mostly on medium- and long-duration pigeonpea types of 6–11 months duration which are traditionally grown in inter- or mixed-cropping systems with cereals and other legume crops. However, in recent years, short-duration pigeonpea types which mature in $3\frac{1}{2}$–$4\frac{1}{2}$ months have been developed. These types are mostly recommended for sole cropping with closer spacing. In experimental fields and on a limited number of farmers' fields, differences in disease severity between short-duration and traditional types are emerging. It should be noted, however, that the information on diseases of short- and extra-short-duration pigeonpea is presently very limited and one must be careful in drawing conclusions at this stage. A greater understanding of the major disease problems will emerge as these types are cultivated more widely.

In medium- and long-duration pigeonpea, fusarium wilt and sterility mosaic in Asia; wilt and cercospora leaf spot in southern and eastern Africa; and witches' broom in Central America and the Caribbean are the major disease problems (Reddy *et al.*, 1990b; Nene *et al.*, 1996). When short-duration pigeonpea is grown in these regions, it also suffers from the same diseases, but disease intensity may differ due to different agronomic practices to which such pigeonpea types are subjected and different phenology of the crop. For example, in Asia, phytophthora blight and rhizoctonia dry root rot are more important on short- and extra-short-duration rather than medium- and long-duration types. At the same time, short- and extra short-duration pigeonpeas are less affected by wilt compared to medium- and long-duration types (Reddy *et al.*, 1980). In southern and eastern Africa, short- and extra short-duration types are affected by cercospora leaf spot and powdery mildew to a greater degree than are the medium- and long-duration types (Shakoor and Kumar, 1982). In temperate regions, it is also expected that short-duration pigeonpea will suffer from these foliar diseases. Close monitoring of diseases in such regions is strongly recommended.

Major diseases that affect pigeonpea globally are dealt with below with particular reference to causal pathogen, biology of the pathogen, symptoms of the disease, epidemiology, economic importance and management. For wilt, sterility mosaic and phytophthora blight, disease management has been covered in detail. For those diseases where information is very meagre, suggestions have been made for further research. Diseases of local or minor importance are

Table 10.1. Diseases of pigeonpea of local or minor importance

Disease	Pathogen	Distribution	Importance
Root and collar diseases			
Damping-off	*Pythium aphanidermatum* (Edson) Fitz.	India	Minor
	Rhizoctonia solani Kühn	India, Malaysia, Philippines, Puerto Rico, Sierra Leone, Uganda, Zambia	Minor
Collar rot	*Sclerotium rolfsii* Sacc.	Australia, India, Pakistan, Sri Lanka, Puerto Rico, Venezuela, Zambia	Locally important to minor
Wilting and rotting	*Pellicularia filamentosa* (Pat.) Rogers; *Rhizoctonia* sp.; *Sclerotinia sclerotiorum* (Lib.) de Bary; *Heliocobasidium purpureum* (Tul.) Pat; *Macrophomina phaseolina* (Tassi) Goid.; *Sclerotium rolfsii* Sacc.	Australia, Puerto Rico, Bermuda, Trinidad	Minor
Crown canker	Sterile white basidiomycete fungus	Puerto Rico	Minor
Root cyst	*Heterodera cajani* Koshy	India, Egypt	Locally important to minor
Pearly rot	*Rotylenchus reniformis* Linford & Diverta	India, Fiji, Jamaica, Trinidad	Locally important to minor
Root-knot	*Meloidogyne* spp.	Widespread	Locally important to minor
Root and stem diseases			
Macrophomina stem canker and root rot	*Macrophomina phaseolina* (Tassi) Goid.	India, Myanmar, Nepal, Sri Lanka, Tanzania, Uganda	Locally important to minor
Canker	*Botryosphaeria xanthocephala* (Syd. & Butler) Theiss	Australia, Puerto Rico, Fiji, India	Locally important to minor
	Macrophomina phaseolina (Tassi) Goid.	Trinidad	
	Botryodiplodia sp.	Puerto Rico	Minor
	Pellicularia filamentosa (Pat.) Rogers	Puerto Rico	Minor
	Phoma cajani (Rangel) Khune & Kapoor	Brazil, India	Minor
	Phoma sp.	Puerto Rico	Minor
	Phomopsis sp.	Puerto Rico	Minor

Continued overleaf

Table 10.1. Continued

Disease	Pathogen	Distribution	Importance
	Phytophthora nicotionae Breda de Haan var. *parasitica* (Dastur) Waterhouse	Puerto Rico, USA	Minor
Stem and foliar diseases			
Bacterial stem canker and leaf spot	*Xanthomonas campestris* pv. *cajani* (Kulkarni *et al.*) Dye *et al.*	Widespread	Minor
Phoma stem canker and leaf blight	*P. cajani* (Rangel) Khune & Kapoor	Malawi, Tanzania, Zambia	Minor
Foliar diseases			
Rust	*Uromyces dolicholi* Arth.; *Uredo cajani* Syd.	Widespread	Locally important to minor
Alternaria leaf spot	*Alternaria tenuissima* (Kunze: Fries) Wiltshire	India, Nepal, Puerto Rico, USA, Zambia	Locally important to minor
Colletotrichum blight	*Colletotrichum capsici* (Syd.) Butl. & Bisby; *C. cajani* Rangel; *C. graminicola* (Ces.) Wilson	India, Brazil, Puerto Rico	Minor to locally important
Phoma leaf spot	*Phoma* sp.	Dominican Republic, Panama, Puerto Rico	Minor
Phyllosticta leaf spot	*Phyllosticta cajani* Syd.	Jamaica, Trinidad, Brazil, Puerto Rico, India, Sri Lanka	Minor to locally important
Web blight	*Thanatephorus cucumeris* (Frank) Donk	Trinidad	Minor
Bacterial leaf spot	*Pseudomonas syringae* pv. *phaseolicola* (Burkh.) Dowson	Ethiopia, Zambia, Uganda, Brazil	Minor
Phyllody	PLOs transmitted by the leafhopper *Orosius albisinetus* Evans	India	Minor
Yellow mosaic	Mung bean yellow mosaic virus	India, Myanmar, Nepal, Sri Lanka, Philippines, Puerto Rico, Jamaica	Minor
Cowpea mosaic	Cowpea mosaic comovirus	El Salvador, Kenya, Puerto Rico, Trinidad	Minor
Inflorescence disease			
Flower blight	*Choanephora* sp.	Trinidad	Minor

Source: Braithwaite (1981); Kaiser (1981); Kannaiyan *et al.* (1984); Reddy *et al.* (1990b, 1993c); Nene *et al.* (1996).

tabulated in Table 10.1. A key to the diagnosis of major diseases is given in the handbook of pigeonpea diseases (Reddy *et al.*, 1993c).

FUSARIUM WILT

Aetiology

Butler (1910) described the causal pathogen as *Fusarium udum*, a soilborne fungus. Snyder and Hansen (1940) named it *F. oxysporum* Schlecht. f. sp. *udum* (Butler) Snyder & Hansen, which was supported by Chattopadhyay and Sen Gupta (1967). However, the name *F. udum* Butler is the accepted anamorph as the macroconidia of *F. udum* can be distinguished by their prominent hook (Booth, 1971). For general pathologists, the taxonomy of the wilt fungus continues to be problematic, as identification based solely on the presence of a macroconidial hook is not always accurate. Rai and Upadhyay (1979) named *Gibberella indica* Rai & Upadhyay as the perfect state of *F. udum* whereas Singh (1980) described it as *G. udum* Singh. Holliday (1980), however, does not accept the existence of a teleomorph.

Biology

Wilt was discovered in India by E.J. Butler in the early 1900s (Butler, 1906). It is the oldest and most widespread disease of pigeonpea and has been reported from all major pigeonpea-growing countries in Asia and Africa (Nene *et al.*, 1996). Although research on the disease has been carried out for the past 90 years, limited information on methods of management other than the development of resistant/tolerant cultivars, is available. Loss of resistance has been reported in some cultivars in India, suggesting the need for more concentrated efforts on understanding the pathogen and in identifying and breeding for stable resistance.

Butler (1910) described the fungus as parasitic within the roots of the host plant, or saprophytic. The hyphae are hyaline, slender, much branched, usually with little aerial growth. Microconidia are produced successively on the ends of short simple or clustered, verticillately branched conidiophores (Holliday, 1980). They are usually aseptate, elliptical, hyaline singly but salmon-pink in mass, occasionally develop from the surface of minute spherical stromata, and are 6–11 × 2–3 µm in diameter. In culture, the microconidial stage is usually white to salmon-pink, occasionally orange-red but never green or purple. Macroconidia are formed on short conidiophores and detach soon after abjunction. They are hyaline, three- to five-septate, 15–50 × 3–5 µm in size, falcate with a distinct foot cell and an atypical cell of decreasing diameter towards the tip which may be curved or hooked (Holliday, 1980). The chlamydospores are round or oval, rather thick-walled, hyaline, intercalary in the mycelium, sometimes in short chains and 5–10 µm in diameter.

Limited information on the perfect state considered to be associated with *F. udum* is available. Perithecia of *G. indica* are formed in fields but infrequently on wilted pigeonpea (Rai and Upadhyay, 1979). If formed on exposed roots or in the collar region of the plant, they are superficial, commonly aggregated, globose to subglobose, sessile and smooth-walled. *G. indica* is a heterothallic fungus and

perithecia can be induced in culture on nutrient medium or on sterilized host substrate at 25±2°C only after mating different strains. The ascus contains eight ascospores which are two- to three-celled. Ascospores germinate to produce micro- and macroconidia. The association of the perfect state with *F. udum* and its role in pathogenesis, however, needs further investigation. The life cycle of the fungus is shown in Fig. 10.1.

Other *Fusarium* spp. which cause wilt of legumes are reviewed in Kraft *et al.*, Chapter 6, and Haware, Chapter 9, this volume. *F. udum*, however, is specific to pigeonpea and its wild relatives *Cajanus* spp. (Kannaiyan *et al.*, 1985). Pathogenic variability and physiologic races have been reported (Baldev and Amin, 1974; Shit and Sen Gupta, 1978; Reddy and Chaudhary, 1985; Pawar and Mayee, 1986; Gupta *et al.*, 1988). Based on the reaction of four pigeonpea lines, 11 isolates from India were divided into three distinct groups (ICRISAT, 1996) (Table 10.2). One of

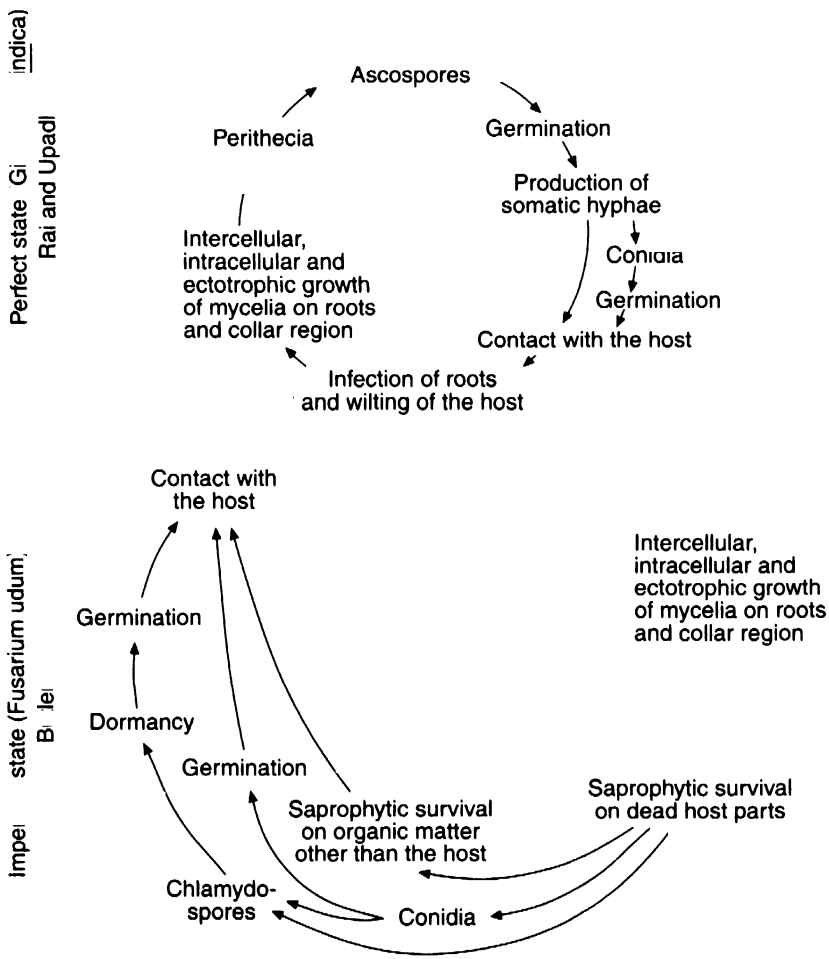

Fig. 10.1. Life cycle of *Fusarium udum*, causal agent of wilt of pigeonpea (Courtesy of Upadhyay and Rai, 1992).

Table 10.2. Reaction of four differential pigeonpea lines to 11 isolates of *Fusarium udum* in pot experiments in a greenhouse at ICRISAT Asia Center, 1995/96.

	Wilt reaction		
Line	Strain 1	Strain 2	Strain 3
ICP 2376	S[1]	S	S
C 11	R	S	S
ICP 8863	R	R	S
ICP 9174	R	R	R
Isolates	Gwalior	Dholi	Patancheru
	Akola	Kanpur	Rahuri
	N-isolate[2]	Varanasi	Badnapur
		Bangalore	Gulbarga

[1] S = Susceptible, R = Resistant.
[2] Northern Indian isolate.

the problems encountered in using differential lines for race identification was variation in the reaction of the lines between experiments, indicating the need for further standardization of the inoculation technique. Preliminary studies have begun on differentiation of isolates of *F. udum* using molecular techniques including random amplified polymorphic DNA (RAPD) analyses (T.N. Raju, unpublished data). To date, results show close correlation with race differentiation.

Symptoms

Patches of dead plants in the field, usually when the crop is flowering or podding, are the first indication of wilt (Fig. 10.2) (Reddy *et al.*, 1990b). Isolated wilted plants are also noted about a month after sowing. The most characteristic symptom in adult plants is a purple colour extending upwards from the base of the main stem. This band is more easily seen in pigeonpea with green stems than in lines with coloured stems. Partial wilting of the plant is a definite indication of wilt and distinguishes this disease from termite damage, drought, and phytophthora blight which also kill the plant. Partial wilting is associated with lateral root infection while total wilt is a result of tap root infection (Reddy *et al.*, 1990b).

The other characteristic symptom of wilt is browning of the stem below the purple band and browning or blackening of the xylem which is visible when the main stem or primary branches are split open (Reddy *et al.*, 1990b). The intensity of browning or blackening decreases from the base to the tip of the plant. Sometimes, branches, especially lower ones, are affected even when there is no band on the main stem. These branches show dieback symptoms with a purple band extending from the tip downwards, and intensive internal xylem blackening. When young (1–2 months old) plants die from wilt; they usually do not show external banding but have obvious internal browning or blackening. Plants infected by *F. udum* also exhibit loss of leaf turgidity, interveinal clearing and chlorosis before death.

Fig. 10.2. Patches of wilted pigeonpea plants in a wilt-sick plot at ICRISAT Center, Patancheru, India (Photo: courtesy of M.V. Reddy).

Epidemiology

The disease is both seedborne and soilborne (Reddy *et al.*, 1990b; Haware and Kannaiyan, 1992). Untreated seed showed levels of internal infection with *F. udum* of 13–19% (Haware and Kannaiyan, 1992). Seed treatment effectively eradicates the pathogen. Infected seed may be the primary means of spread of *F. udum* over long distances and to new areas (Haware and Kannaiyan, 1992). The fungus can survive on infected plant debris in the soil for about 3 years. Disease incidence is more severe on vertisols than on alfisols and ratooning predisposes the plant to wilt (Reddy *et al.*, 1990b). Early sowing, weed management and vigorous crop growth favour wilt development. Long- and medium-duration types suffer more from wilt than short- and extra short-duration types. Pigeonpea intercropped with castor, sorghum, maize and groundnut is less affected by wilt than sole-cropped plants (Fig. 10.3).

Though infection may occur in the seedling stage, maximum expression of the disease is, however, at flowering and podding (Reddy *et al.*, 1990b). This seems to be due to the extended time needed by the fungus to colonize the plant. Recent work at ICRISAT has shown that infected plants wilt only after the basal half of the main stem is colonized by the fungus which takes approximately 3–4 months (Reddy *et al.*, 1993b). This explains why there are low levels of wilt in short-duration types compared to long-duration and ratooned pigeonpea, as the former morphotypes are escaping wilt (Reddy *et al.*, 1980). Any practice which

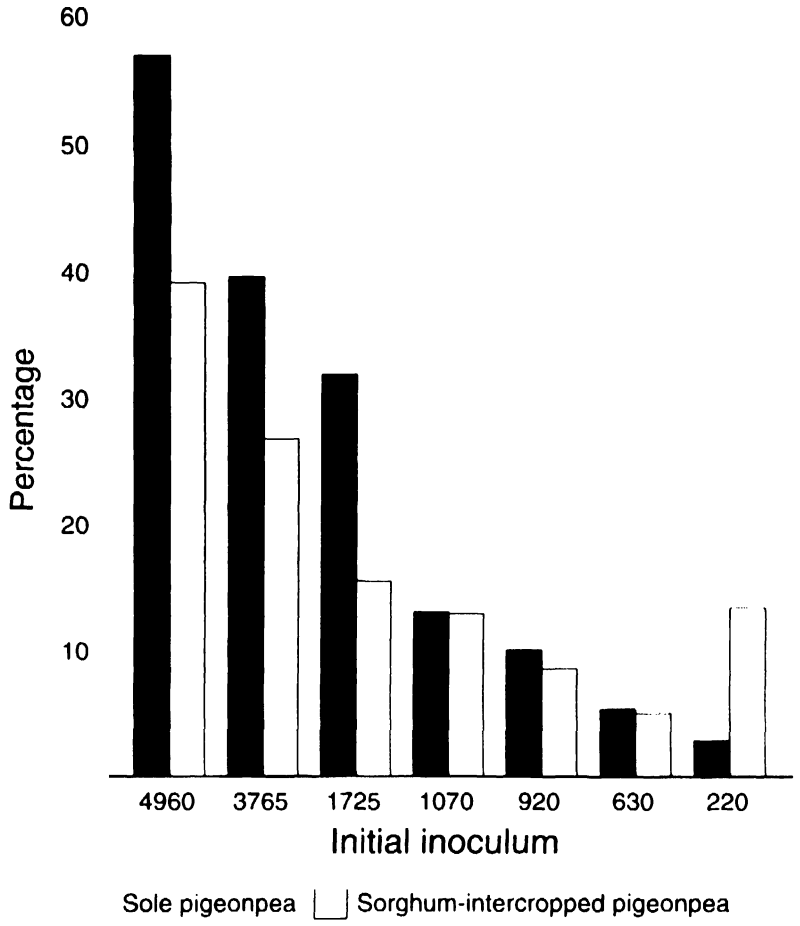

Fig. 10.3. Wilt incidence in sole and sorghum-intercropped pigeonpea at different inoculum densities (colony-forming units) in a vertisol at ICRISAT Center, Patancheru, India.

leads to increased plant biomass in pigeonpea was found to increase susceptibility to wilt (Reddy *et al.*, 1994a). Higher biomass is produced when the crop is sown early, under weed-free and well-drained conditions, in fertile fields at low plant density and when the rains are well distributed.

Recent work has indicated that the fungus can survive in the soil up to 120 cm depth (Naik, 1993). Limited variation in fungal population was found from the crop season to the off-season, especially at lower depth. Inoculum placed at 100 cm depth was found to infect pigeonpea but did not result in wilt. Only inoculum at ≤50 cm depth resulted in both infection and wilt (Naik, 1993). The economic threshold level of 20% wilt incidence in the susceptible cultivar was found to vary for vertisols and alfisols. It was slightly lower for alfisols (830 colony-forming units (cfu) g^{-1} soil) than for vertisols (920 cfu g^{-1} soil). Threshold levels were higher for tolerant cultivars than for susceptible cultivars.

The initial inoculum level in soil was found to be the major factor influencing final wilt incidence compared to soil moisture and temperature.

Sterility mosaic and phyllody-affected plants were less affected by wilt than unaffected plants of the same genotypes (Chadha and Raychaudhuri, 1966; M.V. Reddy, ICRISAT, India, unpublished results). Root-knot nematode (*Meloidogyne* spp.) infection increased wilt-incidence in both wilt-susceptible and wilt-resistant pigeonpea cultivars including ICP 8863 (Reddy *et al.*, 1990b) while cyst nematode (*Heterodera cajani* Koshy) enhanced the pathogenicity of *F. udum* in wilt-susceptible genotypes but the fungus suppressed the reproduction of the nematode (Sharma and Nene, 1989). The reaction of wilt-resistant genotypes such as ICP 8863 was not altered by the presence of cyst nematode.

Effects on Yield and Quality

When infected plants die before podding, yield loss is total (Reddy *et al.*, 1990b). When plants wilt during the pod-filling stage, losses are partial and depend on the physiological maturity stage at which the plant wilts. Wilting during pod filling can also result in seed infection (Haware and Kannaiyan, 1992). Long-duration varieties with good plasticity can compensate for loss of early wilted plants. The yield loss in plants that are infected but do not wilt greatly has not been quantified. To date, no known cultivated pigeonpea line with high resistance to infection by the isolate of *F. udum* common at ICRISAT Center, India has been identified. However, some genotypes, such as ICP 8863, do not suffer much yield loss when infected and may be tolerant.

Surveys conducted between 1975 and 1980 (Kannaiyan *et al.*, 1984) indicated that annual crop loss due to wilt in India alone was US$ 36 million, while in eastern Africa annual losses were estimated at US$5 million. It is clear that further surveys should be done at regular intervals to collect accurate information on current losses.

Disease Control

Use of resistant/tolerant cultivars is the best available strategy for the management of wilt (Reddy *et al.*, 1990b). A number of moderately resistant lines in all the maturity groups are available (Table 10.3). Some of these lines also show resistance across seasons and locations (Nene *et al.*, 1981b, 1989; Amin *et al.*, 1993c). Maruti (ICP 8863), a recently released variety (Konda *et al.*, 1986), has become very popular in peninsular India. Another variety, ICP 9145, released in Malawi, has also become popular (Reddy *et al.*, 1995). Lines combining wilt resistance and resistance to other major diseases have also been identified (Table 10.5)

A resistance screening technique of transplanting seedlings with injured, inoculated roots into autoclaved sand/soil in pots followed by assessment of disease incidence gave erratic results in trials at ICRISAT Center (Nene *et al.*, 1981a). A more reliable screening technique was developed using inoculum of *F. udum* multiplied on sand:pigeonpea flour (9:1) medium mixed with autoclaved

Table 10.3. Single sources of resistance to pigeonpea diseases of global and local importance.

Disease	Resistant varieties/lines
Fusarium wilt	ICP 8863 (Maruti), ICP 9145, ICP 9174, ICP 12745, ICPL 333, ICPL 8363, ICPL 88047, BWR 370, DPPA 85–2, DPPA 85–3, DPPA 85–8, DPPA 85–13, DPPA 85–14, Bandapalera, ICP 4769, ICP 9168, ICP 10958, ICP 11299, C 11 (ICP 7118), BDN1 (ICP 7182)
Sterility mosaic	Bahar, DA 11, DA 13, ICP 999, ICP 6997, ICP 7035, ICP 7197, ICP 7234, ICP 7353, ICP 7867, ICP 8094, ICP 8109, ICP 8129, ICP 8862, ICP 10976, ICP 10977
Phytophthora blight	ICP 9252, Hy 4, ICPL 150, ICPL 288, ICPL 304, KPBR 80–1-4, KPBR 80–2-1, KPBR 80–2-2 (field resistant)
Cercospora leaf spot	UC 796/1, UC 2113/1, UC 2515/2, UC 2568/1, ICP 8869, ICP 12792, ICP 12165, 657/1, ALPL 6–2, 66, 666
Powdery mildew	ICP 7035, ICP 9177, ICP 9179, ICP 9188, ICP 9189, ICP 9192
Alternaria blight	DA 2, DA 11, MA 128–1, MA 128–2, 20(105)

Source: Reddy *et al.* (1993c); Raju (1988).

pigeonpea stem pieces and non-autoclaved alfisol soil (Nene *et al.*, 1981a). For studies on pathogenic variability and inheritance of resistance, a root-dip technique has been developed Haware and Nene 1994).

Wilt-sick plots have been used for a long time to screen crops against vascular wilts. At ICRISAT, sick plots are developed more quickly on alfisols than on vertisols, and wilt becomes evident earlier on alfisols (Nene *et al.*, 1980). In early tests, the pathogen was multiplied on materials other than pigeonpea stubble. It was later realized that the best way to induce disease is to incorporate stubble from diseased plants into the soil and grow wilt-susceptible cultivars in intermittent rows throughout the field.

Although the search for sources of resistance to wilt began as soon as the disease was recognized as a major problem, very few studies on inheritance of resistance have been undertaken and the picture is not clear. Studies carried out to date reveal multiple factors (Pal, 1934), complementary genes (Shaw, 1936), duplicate dominant genes (Joshi, 1957), a single dominant (Pawar and Mayee, 1986) and a single recessive gene (Jain and Reddy, 1995) to be controlling wilt resistance. Further work on the genetic basis of resistance would help to develop a targeted breeding strategy.

Resistant and tolerant cultivars were found to suppress the *F. udum* population in the rhizosphere compared to susceptible cultivars (Murthy and Bhagyaraj, 1983). This was at least partly due to root exudates from resistant cultivars suppressing the germination and germ tube growth of conidia of *F. udum*. Murthy and Bhagyaraj (1983) identified chlorogenic acid, caffeic acid and an unknown phenolic acid from the root exudates of the resistant cultivar C 11-6. Though information on several physical and cultural factors affecting wilt incidence is available, the interactions between these factors and wilt incidence in the field are not well understood. Further work is needed in this area.

Pigeonpea is usually grown in inter- and mixed-cropping systems in rotation with several other crops. This provides an opportunity to exploit the cropping systems and rotations in the management of the wilt. Natarajan *et al.* (1985) found that both crop rotation and intercropping had a considerable effect on wilt incidence. One-year breaks with either sorghum or fallow reduced wilt incidence in following pigeonpea crops from 60–90% to 16 and 31%, respectively. Intercropping with sorghum but not maize also reduced wilt incidence to 20–30% consistently across 14 pigeonpea genotypes after 2 years (Natarajan *et al.*, 1985). This area of research needs more emphasis. Field studies carried out at ICRISAT Center have indicated that crops such as sorghum, castor, maize and groundnut inhibit soil populations of *F. udum* (Himani Bhatnagar, 1995). Ongoing studies are showing that root exudates from a range of crops frequently intercropped with pigeonpea may either directly reduce *F. udum* populations in the soil or encourage the growth of antagonistic fungi such as *Trichoderma* spp. which has the same effect of reducing wilt incidence (M.V. Reddy, ICRISAT, India, 1996, unpublished results). Further work with various soils from a range of localities in Andhra Pradesh, India, is in progress.

Suggested cultural practices to reduce the incidence and severity of wilt include: (i) select fields with no previous record of wilt for at least 3 years; (ii) select seed from disease-free fields; (iii) grow pigeonpea in inter- or mixed-cropping systems with cereals, e.g. sorghum or maize; (iv) rotate pigeonpea with sorghum, tobacco or castor every 3 years; (v) uproot wilted plants for fuel wood; and (vi) solarize the field in summer to help reduce inoculum (Tables 10.5 and 10.6). Suggested chemical control measure is through seed dressing with benomyl 50% + thiram 50% mix at 3 g kg^{-1} seed.

PHYTOPHTHORA BLIGHT

Aetiology

Phytophthora blight of pigeonpea was apparently discovered less than 30 years ago. Williams *et al.* (1968) first isolated a phycomycetous fungus from wilting pigeonpea plants with stem canker symptoms at New Delhi. The fungus was identified as *Phytophthora drechsleri* Tucker var. *cajani* Pal, Grewal & Sarbhoy. Kannaiyan *et al.* (1980) studied several isolates of the fungus from different parts of India and renamed it *Phytophthora drechsleri* Tucker f. sp. *cajani* (Pal, Grewal & Sarbhoy) Kannaiyan, Riberio, Erwin & Nene based on sporangium shape and size, oogonium and oospore formation, temperature requirements and pathogenicity tests. The use of *forma specialis* was considered appropriate because of the specificity of the isolates to pigeonpea and its close relatives (Reddy *et al.*, 1990b). Ho and Jong (1991) studied the taxonomy of various *Phytophthora* spp. and redefined *P. drechsleri* to accommodate only those isolates that grow well at 35°C, with the pigeonpea blight pathogen treated as non-specific.

Biology

Relatively limited information is available on the pathogen, its epidemiology and disease management. Though the disease has been reported from several countries including India, the Dominican Republic, Kenya, Panama and Puerto Rico (Nene *et al.*, 1996), precise information on its distribution and severity is still lacking. It is possible that the disease was mistaken for fusarium wilt in the past because the general symptoms of the two diseases are similar (Reddy *et al.*, 1990b). In India, *P. drechsleri* Tucker f. sp. *cajani* affects the collar region and all above-ground parts of the plant. In Australia, *P. drechsleri* was reported to cause chlorosis and lesions on stems of pigeonpea in addition to serious root rot (Wearing and Birch, 1988). The true identity of *Phytophthora* spp. from other pigeonpea-growing countries clearly needs to be confirmed.

The disease is relatively more serious in short-duration pigeonpea than in medium- and long-duration types (Reddy *et al.*, 1990b). This seems to be related to the cropping system used for short-duration pigeonpea which is characterized by increased plant population which leads to rapid canopy development and high relative humidity in the crop. As a result, losses can be considerable as the short-duration types have neither the time nor the plasticity to compensate for lost plants (Reddy *et al.*, 1990b). Short-duration pigeonpea has great potential to increase grain yield and to extend the adaptation of the crop to non-traditional areas but management of blight is essential for the realization of this potential.

The morphology and disease cycle of the blight pathogen have been described in detail (Kannaiyan *et al.*, 1980; Singh and Chauhan, 1992). The optimum temperature for growth of *P. drechsleri* f. sp. *cajani* on clarified V8 juice agar is 27–33°C. Sporangia are the proliferating type, hyaline, terminal, ovate to pyriform, non-papilate and ranged in size from 42–83 × 28–48 µm. The sporangial stalks were either narrowly tapered or widened somewhat at the base of the sporangium (Reddy *et al.*, 1990b). Each sporangium produces 8–20 zoospores (Singh and Chauhan, 1992). Zoospores are hyaline, ovoid to reniform, tapering slightly, biflagellate, and swim for 2–5 h before becoming non-motile and forming a spherical cyst which usually germinates with one or more germ tubes. *P. drechsleri* f. sp. *cajani* belongs to the mating type A1 with bicellular antheridia in some interspecific crosses. Oogonia are hyaline when immature but become thick-walled and purple-yellow to brown after maturity, smooth, spherical, and measure 19–29 × 34–44 µm (Singh and Chauhan, 1992). Antheridia are simple, hyaline, amphigynous, persistent 12.5–19 × 10–17 µm. Oospores are spherical to globose, thick-walled, and 20–32 µm in diameter. No chlamydospores were formed on any of the media tested. The life cycle of the disease is given in Fig. 10.4.

Although the disease can be identified by the symptoms produced, several workers have encountered difficulties in isolating *P. drechsleri* f. sp. *cajani*. The fungus can be isolated on dehydrated potato dextrose agar medium after surface sterilization of the infected tissues with 0.1% mercuric chloride for 30 seconds. Bisht and Nene (1988) formulated a selective medium to isolate the fungus based on a mixture of antimicrobial agents with potato dextrose agar. Sheila *et al.* (1983) reported pigeonpea seed meal agar prepared from an infusion of 40 g of

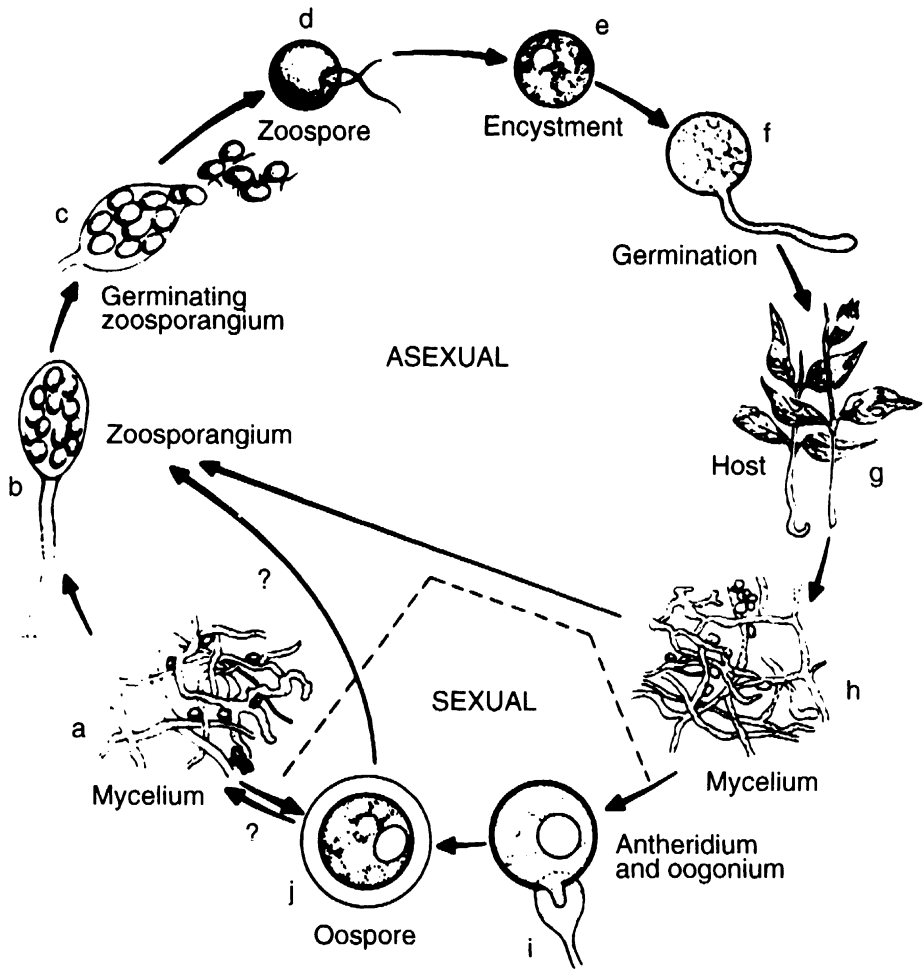

Fig. 10.4. Disease cycle of *Phytophthora drechsleri* f. sp. *cajani*, causal agent of phytophthora blight (courtesy of Singh and Chauhan, 1992).

seed meal boiled in 1000 ml distilled water for 1 h as an excellent substitute for V8 juice agar. Pscheidt *et al.* (1992) evaluated the sensitivity of a *Phytophthora*-specific monoclonal antibody-based immunoassay kit on 17 species of *Phytophthora*, including *P. drechsleri*. All the *Phytophthora* species tested produced a positive reaction. A similar kit could be developed specifically for the pigeonpea blight pathogen as a useful diagnostic tool when the diseased plants are old, dried or rotted and are thus in poor condition for isolation of the pathogen.

Research over the past 15 years has shown that *P. drechsleri* f. sp. *cajani* is variable (Reddy *et al.*, 1990b). When work on phytophthora blight was initiated at ICRISAT in 1976, screening for resistance was carried out with isolate P2 which was prevalent in the ICRISAT fields. Several pigeonpea lines such as ICP

2376 and ICP 7065 were resistant to the this isolate (Kannaiyan *et al.*, 1981). However, in subsequent tests, these lines were susceptible to the disease in the same field. The re-isolated strain was more virulent than P2 and was designated P3. At present, no pigeonpea genotype has a high level of resistance to P3. Studies with other isolates collected from different locations in India have confirmed the highly variable nature of the fungus (Nene *et al.*, 1991). Precise studies to characterize the pathogen better are needed. Characterization of host reaction by lesion type and size may be a better criterion than disease incidence for the differentiation of variants. Studies carried out to date show that P2 can be readily distinguished from P3 by the reaction on ICP 2376 which is resistant to P2 (0% blight incidence) but highly susceptible to P3 (100% blight incidence and mortality). Morphological variation also exists among Indian isolates of *P. drechsleri* f. sp. *cajani* (Kannaiyan *et al.*, 1980; Nene *et al.*, 1992). Phytophthora blight of soyabean is reviewed by Sinclair, Chapter 3, this volume.

In experimental plots at ICRISAT, natural infection with *P. drechsleri* f. sp. *cajani* was observed not only on pigeonpea but also on wild *Cajanus* spp. In pot-screening tests, P2 infected 13 wild *Cajanus* spp. (Sarkar *et al.*, 1991). However, none of 55 plant species of genera other than *Cajanus* were either hosts or symptomless carriers of the pathogen (Sarkar *et al.*, 1991). Wild *Cajanus* spp, especially *C. scarabaeoides*, a common perennial weed, may serve as an alternative host of the fungus.

Symptoms

Phytophthora blight resembles damping-off, as it causes seedlings to die suddenly (Reddy *et al.*, 1990b). Young plants are more susceptible than older plants (Sarkar *et al.*, 1992) and young tissues are more susceptible than older tissues. Infected plants have water-soaked lesions on leaves and brown to black, slightly sunken lesions on stems and petioles. Infected leaves lose turgidity, becoming blighted (Fig. 10.5) (Reddy *et al.*, 1990b). Stem lesions may appear from a few centimetres to approximately 1.5 m above ground level, expand rapidly and girdle affected portions. The stems or branches break at the point of infection. Under favourable conditions for the disease, many plants may die. Infected plants that are not killed often produce large galls on their stems especially at the edges of the lesions. The pathogen infects the foliage and stems but not the root system.

Epidemiology

The pathogen survives in soil, even in the absence of a living host, and also in infected crop debris for at least one year (Bisht and Nene, 1990). Sarkar (1988) reported that *P. drechsleri* f. sp. *cajani* survives as chlamydospores in field soil and diseased stubble. Singh and Chauhan (1992) observed that a living host is not essential for oospore formation but that temperature is critical, oospores being produced at 25°C only. The role of chlamydospores and oospores in survival and perpetuation of the fungus needs further study. This is important because the

Fig. 10.5. Phytophthora blight on pigeonpea: upper photo shows blight lesions on leaves; lower photo shows a stem lesion resulting in girdling and breakage of the stem at the point of infection (photos: courtesy of M.V. Reddy).

disease may suddenly appear in a severe form in fields where pigeonpea has not been cultivated for several years. The fungus is soilborne and waterborne, but not seedborne. Rain-splash and wind also contribute to short-distance dispersal of the zoospore inoculum (Bisht and Nene, 1990). The role of the wild relative *Cajanus scarabaeoides* in the perpetuation and spread of phytophthora blight has not been established (Kannaiyan *et al.*, 1985).

Disease development is influenced by inoculum density and by environmental factors. Agrawal and Khare (1987) reported that the maximum infection index (49.2%) was observed at 28.1 mm day^{-1} rainfall, 100% rainy days, 27.4°C maximum temperature, 21.4°C minimum temperature, and 92.4% relative humidity. In contrast, 6.5% infection index was observed at 9.4 mm day^{-1} rainfall, 30% rainy days, 31.7°C maximum temperature, 22.7°C minimum temperature, and 84.1% relative humidity. They inferred that infection index was

positively correlated with rainfall (mm day^{-1}), rainy days (%) and relative humidity (%), and that number of rainy days (%) was more important than the amount of rain. Chauhan and Singh (1991a) observed that light inhibited zoospore germination and maximum germination occurred in the dark. Singh and Chauhan (1992) further reported that light and darkness affected disease development; lesion size increased more rapidly in the dark than in continuous light. Rainfall, maximum temperature and solar radiation influence blight infection and disease development; outbreaks occur when day temperatures are less than 28°C and accompanied by rainy and cloudy weather (Reddy *et al.*, 1992). Increase in inoculum level and blight incidence is associated with a decrease in day temperature, and with high rainfall and cloudy weather (Reddy *et al.*, 1992). Recent work carried out under controlled conditions in growth chambers and in the laboratory at ICRISAT Center showed that the duration of leaf wetness is more critical for pathogen infection than temperature and inoculum load (T.N. Raju, M.V. Reddy and A.C. Kushalappa, unpublished results). A leaf wetness period of 12 h was necessary for infection and infection occurred between 10 and 35°C.

Effects on Yield and Quality

Surveys carried out in India between 1975 and 1980 indicated that phytophthora blight was prevalent in the states of Andhra Pradesh, Bihar, Orissa, Uttar Pradesh and West Bengal, but not in Gujarat, Karnataka, Madhya Pradesh, Maharasthra, Rajasthan and Tamil Nadu (Kannaiyan *et al.*, 1984). The average incidence was 2.6% but in West Bengal it was as high as 26.3%. A severe epidemic was reported in experimental plots on alfisols at ICRISAT Center in the 1975/76 season (Reddy *et al.*, 1990b). However, this information is based on observations in medium- and long-duration pigeonpea only on which blight is not considered to be a serious problem. The disease increased in importance from the early 1980s with the introduction and dissemination of short-duration types. Total yield loss has been observed in some short-duration pigeonpea crops in southern India (M.V. Reddy, unpublished results). A further comprehensive survey of damage, especially in short-duration pigeonpea in India, is urgently needed to reassess the distribution and severity of phytophthora blight and estimate crop losses. There is also a need to assess the potential importance of the disease in countries which are presently adopting short-duration types.

Disease Control

Pigeonpea is mainly cultivated by resource-poor farmers on marginal lands with minimal inputs where present yield levels of 700 kg ha^{-1} are not conducive to management of phytophthora blight through the use of expensive foliar fungicides such as metalaxyl. The ideal way to manage phytophthora blight is through resistant cultivars.

Although considerable work has been done on standardization of glasshouse

and field inoculation techniques for evaluation of blight resistance (Kannaiyan *et al.*, 1981; Reddy *et al.*, 1990a), obtaining uniform disease incidence in the field and under greenhouse conditions is still a problem. Establishment of uniform humidity after inoculation is critical, but is difficult to achieve under field conditions. Using a level field with bunds at close intervals to cause temporary inundation within the first 2–3 weeks after sowing is recommended for obtaining high and uniform blight infection (Reddy *et al.*, 1990a).

Evaluation of the world collection of pigeonpea germplasm of more than 15,000 accessions at ICRISAT against common isolates of *P. drechsleri* f. sp. *cajani* failed to identify any source of high resistance to blight (Reddy *et al.*, 1990b, 1991). All short-duration accessions were highly susceptible. These types may be genetically more susceptible to *P. drechsleri* f. sp. *cajani* than medium- and long-duration types or the high plant populations used for short-duration crops also provides a favourable environment for blight development. As well, the short plant stature of short-duration types facilitates splashing of zoospores from the soil onto younger tissues. Thus the higher susceptibility of short-duration types could be due to a complex combination of interrelated factors.

A few medium- and long-duration lines, such as KPBR 80–2-1, KPBR 80–2-2 and ICP 9252, have been identified with field resistance to P2 and P3 isolates expressed in the adult plants (Table 10.3) (Reddy *et al.*, 1991; Amin *et al.*, 1993b). However, even these lines are susceptible when phytophthora blight occurs within 2–3 weeks of sowing. The reasons for higher susceptibility to *P. drechsleri* f. sp. *cajani* in younger tissues compared to older tissues needs further study. It is also common for plants to die from blight at later stages of growth (>60 days) when conditions for disease development remain favourable. Mortality in older plants may also be due to disease progress from lesions produced during early infections and not due to new infections.

Some accessions of the wild species *C. platycarpus* (ICWP 61, ICWP 66, ICWP 67) have high levels of resistance to phytophthora blight and are able to be crossed with pigeonpea using embryo rescue techniques (ICRISAT, 1993, 1994). Enhancing resistance to phytophthora blight in pigeonpea by making use of the resistance in *C. platycarpus* through introgression into the cultivated germplasm is being strongly supported at ICRISAT. Information on the genetics of resistance to blight is limited. Resistance in ICP 7065 and ICP 2376 against the P2 isolate is controlled by a single dominant gene (Sharma *et al.*, 1982). No information is available on the genetics of adult plant resistance to the P3 isolate. This information is essential for the formulation of future disease resistance breeding strategies, including the use of genes from wild *Cajanus* spp.

Chauhan and Singh (1991b) reported that the weed canopy interferes with splash dispersal of *P. drechsleri* f. sp. *cajani* from soil to aerial plant parts, thus reducing disease intensity. They suggested that blight may be reduced and pigeonpea yields may be increased by mulching or by intercropping with short leguminous crops such as mung bean and urd bean. Recent studies at ICRISAT Center have confirmed that phytophthora blight incidence and severity are substantially reduced when short-duration pigeonpea is intercropped with short leguminous crops such as black gram and groundnut (Plate 24) (M.V. Reddy and T.N. Raju, ICRISAT, India, 1995–1996, unpublished data). Suggested cultural

practices for control of phytophthora blight include: (i) select fields with no previous record of blight; (ii) avoid sowing pigeonpea in fields with low-lying patches that are prone to waterlogging; (iii) prepare raised seedbeds and provide good drainage; (vi) use wide inter-row spacing, crop rotation and intercropping with short legumes; and (vi) use potassium fertilizers (Tables 10.5 and 10.6).

Kannaiyan and Nene (1984) reported that seed dressing with metalaxyl controlled phytophthora blight in greenhouse trials but not in field tests. However, Bisht *et al.* (1988) found metalaxyl to be effective when used as a foliar spray alone, or in combination with seed dressing. The compound ajoene, isolated from garlic, inhibits growth and reproduction of *P. drechsleri* f. sp. *cajani* (Singh *et al.*, 1992). It may be effective in controlling phytophthora blight under field conditions if applied at low concentrations before zoospore formation (Singh *et al.*, 1992). However, this requires further experimentation and economic evaluation. Sheila and Nene (1987) reported reduced infection when Phytoalexin 84® and Induce® were applied to the soil before drench inoculations with the fungus but the plants were susceptible to foliar inoculation. Suggested chemical control measures include firstly, dressing seed with metalaxyl at 3 g kg^{-1} seed to protect young plants. Vyas *et al.* (1983) reported upward movement of metalaxyl in pigeonpea seedlings when roots were treated but not vice versa. The fungicide persisted in the seed coat and cotyledons for 11 days after seed treatment and in leaves and roots for 20 and 15 days after root treatment. Secondly, two foliar sprays with metalaxyl or fentin acetate at 15-day intervals starting from 15 days after germination will also control blight.

Until high levels of resistance to blight are widely available in pigeonpea, the best option for integrated management of the disease is through adult plant resistance, seed dressing and cultural control methods (Table 10.6). As the pigeonpea plant is most susceptible to blight for the first 45 days, seed dressing with fungicides such as metalaxyl will protect the crop during the early stages and is not expensive.

CERCOSPORA LEAF SPOT

Aetiology and Biology

Cercospora leaf spot caused by *Cercospora* spp. is widespread but it is a problem in humid regions only (Rubaihayo and Onim, 1975). Three species of *Cercospora* have been reported to affect pigeonpea in different parts of the world (Reddy *et al.*, 1990b; Nene *et al.*, 1996). The most common species is *Cercospora cajani* Hennings (syn. *Mycovellosiella cajani* (Henn.) Rangel ex Trotter) which occurs throughout eastern and southern Africa, Asia, Latin America, the Caribbean and the Pacific (Nene *et al.*, 1996). It was first reported from Puerto Rico by Stevenson in 1917 (Reddy *et al.*, 1990b). At least two varieties have been recognized: *M. cajani* var. *indica* (Singh) Deighton in Fiji and India and *M. cajani* var. *tricophila* (Curzi) Deighton in Kenya, Somalia and Taiwan (Nene *et al.*, 1996). *Cercospora thirumalacharii* Sharma & Mishra has been reported from India while *C. instabilis* Rangel is known from Brazil, Puerto Rico, India, the Philippines and

the USA (Reddy *et al.*, 1990b; Nene *et al.*, 1996). As *C. cajani* is the most prevalent species, the following section relates to this pathogen only.

The disease is important on pigeonpea in humid highland areas of eastern and southern Africa (Onim, 1980; Shakoor and Kumar, 1982; Shakoor *et al.*, 1983) because flowering usually occurs during cool and humid weather which favours the development of the pathogen. Pigeonpea is known to suffer from foliar diseases such as cercospora leaf spot and powdery mildew at the reproductive stage more so than in the vegetative stage and both diseases often occur together (Reddy *et al.*, 1993c). Cercospora leaf spot is a problem on short-duration pigeonpea in north-western states of India such as Uttar Pradesh and Haryana (M.V. Reddy, unpublished results). Cercospora leaf spots and those caused by allied genera affect a wide range of legumes and others are reviewed by McDonald *et al.*, Chapter 2, Sinclair, Chapter 3, and Allen *et al.*, Chapter 5, this volume.

Symptoms

Symptoms first appear as small circular to irregular necrotic spots or lesions, usually on older leaves (Fig. 10.6). These lesions coalesce causing leaf blight and defoliation (Reddy *et al.*, 1993c). During epidemics, lesions appear on young branches and cause tip drying and dieback. The Indian isolates of *Cercospora* spp. produce fluffy mycelial growth on lesions while the African isolates produce zonate lesions. Under humid conditions, sporulation can be observed on leaf lesions (Reddy *et al.*, 1990b).

Epidemiology

Cool temperatures (25°C) and humid weather favour the disease, which normally appears when plants are flowering and podding (Reddy *et al.*, 1993c). Cyclonic rains in southern and north-eastern India can result in sudden outbreaks of the disease in certain years. No systematic work has been done on the biology and epidemiology of *C. cajani* (Reddy *et al.*, 1990b). It is logical to expect that the pathogen survives on leaf debris and perennial plants.

Effect on Yield and Quality

Yield losses up to 85% have been reported from eastern Africa and losses are severe when defoliation occurs before podding (Onim and Rubaihayo, 1976; Onim, 1980; Reddy *et al.* 1993c).

Disease Control

It is possible to control cercospora leaf spot with periodic sprays of benomyl, mancozeb and maneb (at 3 g l^{-1} water) (Reddy *et al.*, 1990b); however, this is

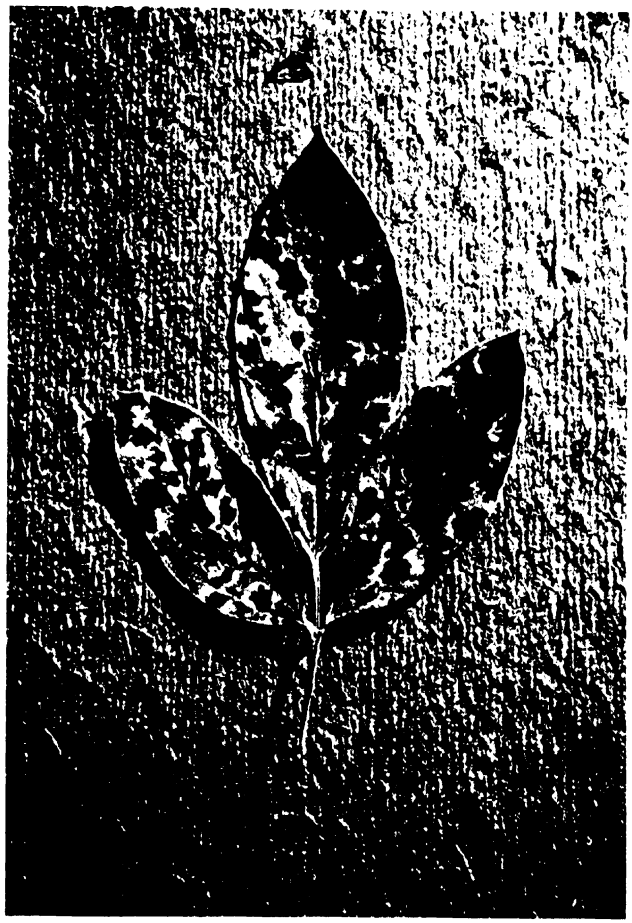

Fig. 10.6. Necrotic spots caused by *Cercospora cajani* on leaves of pigeonpea (Photo: courtesy of M.V. Reddy).

unlikely to be an economical option for resource-poor farmers in Asia and Africa. Onim and Rabaihyao (1976) attempted to screen and breed for resistance to cercospora leaf spot and identified lines with high levels of resistance and increased yields (Table 10.3). Suggested cultural control practices include: (i) select fields away from perennial pigeonpea which may be a source of inoculum and (ii) select seed from healthy crops (Tables 10.5 and 10.6).

POWDERY MILDEW

Aetiology and Biology

Powdery mildew, caused by *Leveillula taurica* (Lev.) Arnaud (anamorph *Oidiopsis taurica* (Lev.) Salmon) is a widespread disease affecting pigeonpea (Nene *et al.*,

1996), and many other economic plants (Holliday, 1980). Probably the first report of its occurrence on pigeonpea was from Tanzania (Wallace, 1930). It is particularly common in southern and eastern Africa (Reddy *et al.*, 1990b; Nene *et al.*, 1996). Powdery mildew usually assumes importance during the reproductive stage in long-duration types but can severely infect short-duration types as well. Long-duration landraces often escape from disease if they flower and pod when the season is dry and warm. Short-duration pigeonpea is vulnerable as it flowers when the season is cool and humid, especially in some areas of the southern Indian states of Karnataka and Tamil Nadu. Although the disease has potential to be serious, limited research has been done with the exception of the studies of Raju (1988). Powdery mildew of pea is reviewed by Kraft *et al.*, Chapter 6, this volume.

Symptoms

Infected plants show white powdery fungal growth on all aerial parts, especially leaves, flowers and pods (Raju, 1988). Severe infection results in heavy defoliation. The disease causes stunting of young plants, followed by the visible symptoms of white powdery growth that appear gradually before flowering. The initial symptoms develop as small chlorotic spots on the upper surface of individual leaves and subsequently with corresponding lower surfaces. When the fungus sporulates, this white powdery growth covers the entire lower leaf surface. In severe infection, leaves turn yellow, twist and crinkle, then fall (Plate 25) (Narayanaswamy and Jaganathan, 1975).

Epidemiology

Although the pathogen is generally considered to be monocyclic, Raju (1988) found that initial infection with powdery mildew in pigeonpea was followed by secondary spread. Infection is directly proportional to the quantity of conidial inoculum available and disease progress is exponential (Raju, 1988). Indian cultivars with thin, succulent leaves that are easily colonized by the fungus are more susceptible than those from Kenya that have thicker leaves. The disease develops at temperatures ranging from 20 to 35°C, but 25°C is optimal. A cool, humid climate favours fungal infection and colonization while a warm humid climate is suitable for sporulation and spore dispersal. Sporulation is more frequent on young leaves than on older ones. Plants attacked by sterility mosaic or phyllody support abundant sporulation and since such plants remain green in the field for long periods, they provide a continuous source of inoculum (Reddy *et al.*, 1984; Raju, 1988; Prameela *et al.*, 1989). The fungus survives on perennial pigeonpea and volunteer plants growing in the shade, and on the ratoon growth of harvested stubbles (Raju, 1988). It also survives as dormant mycelium on infected plant parts such as the axillary buds. In India, early sowing, shade and irrigation encourage disease establishment (Raju, 1988).

Effect on Yield and Quality

Although limited information is available on the effect of powdery mildew on yield of pigeonpea, the disease caused 100% defoliation and yield loss when newly developed short-duration pigeonpea lines were tested in eastern and southern Africa (Raju, 1988).

Disease Control

Studies on the effect of cultural, chemical and biological control methods indicated that powdery mildew incidence is greater in early sowings than in late sowings (Raju, 1988). Triadimefon, a systemic fungicide, was effective in controlling the disease (Raju, 1988). Suggested cultural control practices include: (i) select fields distant from perennial pigeonpea affected with powdery mildew and (ii) sow late (after July) in India, to reduce disease incidence (Tables 10.5 and 10.6). Suggested chemical control measures include spraying with wettable sulphur at 1 g l^{-1} or triadimefon at 0.03%. *Cladosporium* sp. was identified as a hyperparasite of the powdery mildew pathogen and has potential to control the disease biologically (Raju, 1988).

A number of lines with resistance to powdery mildew were identified (Table 10.4) (Raju, 1988). ICP 9177, a germplasm accession from Kenya, was immune

Table 10.4. Multiple sources of resistance to pigeonpea diseases of global and local importance.

Disease	Resistant varieties/lines
Wilt + phoma stem canker + phyllody + halo blight + phyllosticta leaf spot	ICPL 87, C 11
Wilt + sterility mosaic + phytophthora blight	ICPL 83024
Wilt + sterility mosaic + powdery mildew	ICP 7867, ICP 8861 (ICP 7035), ICP 8862 (Hy 3C)
Wilt + phytophthora blight + halo blight	BDN 1
Sterility mosaic + powdery mildew + halo blight	Hy 3C, ICP 7035
Wilt + sterility mosaic	ICP 9174, ICPL 227, ICPL 87119, NPWR 15, Purple 1
Wilt + halo blight	ICPL 81
Sterility mosaic + alternaria blight	ICPL 366, DA 11, ICP 2630, ICP 3782, ICP 3783, ICP 4725, ICP 7188, ICP 7201, ICP 7869, ICP 7904, ICP 7906, ICP 8850, ICP 8852, ICP 8856, ICP 8857
Wilt + alternaria blight	ICP 8861, ICP 8862, ICP 8867, ICP 8869, ICP 10960

Source: Reddy *et al.* (1993c); Raju (1988).

to the disease. Some of the resistant lines, such as ICP 8862 and ICP 7035, also have resistance to wilt and sterility mosaic (Table 10.4). In general germplasm from Kenya was found to be highly resistant (Raju, 1988; Reddy *et al.*, 1993b).

WITCHES' BROOM

Aetiology and Biology

Witches' broom is the most important disease of pigeonpea in the Caribbean and South America (Brathwaite, 1981). It also occurs in Australia, Bangladesh, Papua New Guinea and the USA (Nene *et al.*, 1996). McCoy *et al.* (1983) reported serious damage due to witches' broom in pigeonpea in southern Florida. The actiology of the disease is not fully known. Though phytoplasma-like-organisms (PLOs) and rhabdovirus particles have been found in infected plants, the role of the latter organism in disease development has not been fully elucidated (Maramorosch *et al.*, 1974). Licha-Baquero (1979) and McCoy *et al.* (1983) confirmed the association of witches' broom with PLOs in Puerto Rico and Florida, respectively, but the rhabdovirus was found associated with a pale mosaic or mild vein-yellowing condition only, and not with witches' broom. Reddy *et al.* (1990b) suggested that possibly a mixed infection resulted in the association of both a PLO and a rhabdovirus with witches' broom in the studies of Maramorosch *et al.* (1974). A similar disease called pigeonpea rosette has been reported from India (Maramorosch *et al.*, 1976). Clover phyllody and little leaf of tropical pasture legumes are also caused by PLOs and are reviewed by Mercer, Chapter 12, and Lenné, Chapter 13, this volume. Harrison *et al.* (1991) extracted total DNA from enriched preparations of the PLOs from affected pigeonpea and selected recombinant plasmids as probes in dot and Southern hybridizations with total DNAs from plants affected by various PLO-associated diseases. The probes did not hybridize with DNA from plants affected by aster yellows, periwinkle witches' broom, maize bushy stunt, beet leafhopper-transmitted virescence, Western-X, and lethal yellowing PLOs, nor with DNA from healthy plants. They also demonstrated the presence of extrachromosomal DNA associated with the Florida isolate of the witches' broom PLO. The leafhopper *Empoasca* sp. is reported as the vector of the disease by Vakili and Maramorosch (1974); in Puerto Rico, Licha-Baquero (1979) reported the leafhopper *Empoasca fabae* Harr while McCoy *et al.* (1983) reported two leafhopper species *Empoasca plebeia* DeLong & Davidson and *Acinopterus* sp. Although witches' broom is considered serious in Central America and the Caribbean, research on the disease has been limited due to its restricted geographical range and the minor importance of the crop in some of the countries in which witches' broom occurs. Further studies on the aetiology of the disease are needed.

Symptoms

Infection results in excessive proliferation and clustering of branches and small pale green leaves (Brathwaite, 1981) (Fig. 10.7). This gives to the plant a

Fig. 10.7. Proliferation and clustering of branches and small, pale green leaves on pigeonpea caused by witches' broom.

'witches' broom' appearance. Such plants rarely produce flowers and pods. Flowers, if produced at all, appear in clusters with elongated pedicels. All or part of the plant may exhibit symptoms depending on the intensity of the disease. Affected plants do not produce any grain.

Effect on Yield and Quality

Although quantitative yield losses in pigeonpea due to witches' broom have not been estimated, large areas of the Dominican Republic have up to 100% incidence in certain years (Reddy *et al.*, 1990b) and extensive damage with as many as 75% of plants affected has been reported from southern Florida (McCoy *et al.*, 1983).

Disease Control

Suggested cultural practices include: (i) select fields away from perennial pigeonpeas affected with witches' broom; (ii) pull out and destroy any infected plants to minimize secondary spread of the disease; and (iii) avoid ratoon cropping (Tables 10.5 and 10.6). Possible chemical control measures include insecticides (e.g. metasystox at 0.1%) to control the leafhopper but this will not be economic where pigeonpea is grown by resource-poor farmers.

STERILITY MOSAIC

Aetiology

In spite of numerous attempts during the past 20 years to identify the causal agent of sterility mosaic, it remains a disease of unknown actiology. As it was assumed that a virus may be involved, various protocols for the purification and detection of viruses were tested using young infected tissue. No virus-like particles, viral inclusions or prokaryotes were found consistently (Reddy *et al.*, 1990b). Total nucleic acids, single and double stranded RNAs were extracted and compared in infected and healthy leaves (Reddy *et al.*, 1994b). No consistent differences were found. Occasionally, two double-stranded RNAs occurred in infected but not in healthy plants; however, it was later discovered that they were derived from *Leveillula taurica* (Lev.) Arnaud, the causal agent of powdery mildew, which, for unknown reasons, preferred sterility-mosaic-infected plants (Reddy *et al.*, 1994b). The possible involvement of a viroid or a low molecular weight RNA was examined in infected tissues from young and old infected plants (Ishikewa *et al.*, 1984). No consistent differences in nucleic acid profiles between healthy and infected tissues were observed (Reddy *et al.*, 1994b). No phytoplasma, spiroplasma or rickettsia-like organisms have been observed in thin sections of infected tissues examined by electron microscopy (Reddy *et al.*, 1994b). Recent studies at the Scottish Crops Research Institute (SCRI), Invergowrie, UK, on the similar reversion disease of blackcurrant have detected a virus as the causal agent (A.T. Jones, SCRI, Invergowrie, UK, 1996, personal communication). New investigations are now under way at SCRI on sterility-mosaic-infected pigeonpea tissues using the same molecular-based methodologies to attempt to identify the causal agent.

Biology of the Disease and the Vector

Sterility mosaic is the most important disease of pigeonpea in India and Nepal (Reddy *et al.*, 1990b). It was first reported from Pusa in the state of Bihar, India, more than 65 years ago by Alam (1931) who gave the first detailed description of the disease. Sterility mosaic is present in major pigeonpea-producing states of India and, recently, it has become a serious problem in north-eastern (especially Bihar and Uttar Pradesh) and southern (especially Tamil Nadu and Karnataka)

Table 10.5. Steps in the integrated management of major pigeonpea diseases.

Treatment/control measure	Fusarium wilt	Sterility mosaic	Phytophthora blight	Dry root rot
Field selection	No previous record of wilt and no pigeonpea for past 3 years	No perennial or ratooned pigeonpeas in the vicinity	Well-drained field to avoid water stagnation	–
Cultural practices	Uproot and destroy wilted plants	Eliminate volunteer and ratooned plants	Provide better drainage	–
Seedbed preparation	Normal	Normal	Ridges or broad beds	Normal
Resistant varieties/ genotypes				
(i) Short-duration	Pragati (ICPL 87), AL 1, DL 82, H 76–51*, H 76–44*, H 76–51*, H 76–65*, ICPL 81*, Prabhat	Jagriti (ICPL 151)	ICPL 150*	ICPL 86005*, ICPL 86020*, ICPL 87105*, ICPL 91028*,
(ii) Medium-duration	Maruti, BDN 1, BDN 2, Mukta, Sharda, C 11, Birsa, Arhar 1, ICPL 87119, ICPL 87051, TT 5, TT 6	Hy 3C, PDA 2, DA 11, DA 13, PDA 10, ICPL 86*, ICPL 146*, MA 165, MA 166, Rampur Rahar, Bageswari, ICPL 87119, ICPL 87051	BDN-1 (tolerant), Hy 4, ICPL 304*	–
(iii) Long-duration	ICP 9174*, ICP 7867*, ICP 9145, NPWR 15	ICP 9174*, ICP 7867*, ICPL 366*, Bahar, NPWR 15	KPBR 80–1-4*, KPBR 80–2-1*	
Seed dressing	Benomyl	Carbofuran (25%) or aldicarb (10%)	Metalaxyl	PCNB (3 g kg^{-1})
Sowing date	Late sowing but yield will be reduced	Early or normal sowing to escape from disease build-up	Early or normal sowing	Avoid sowing during hot weather
Spacing	Normal	Normal	Wider-row	Normal
Crop rotation	3 years with tobacco/sorghum	3 years with cereals	3 years with cereals	–
Intercropping	Sorghum/pigeonpea	Sole	–	–
Foliar spray	Not needed	Acaricide spray like Karathane at 0.1%	Metalaxyl (Ridomil MZ) 6 g l^{-1}	–
Irrigation	Can help	May increase the problem	Can increase the problem	Will be useful

– = no information available; * = Not released cultivar.
Source: Reddy *et al.* (1993c).

Table 10.6. Six proven packages of practices for integrated management of major pigeonpea diseases in different production zones/systems in India and eastern Africa.

Production zone	Duration of varieties	Major diseases	Suggested control measures
North-eastern India	Long-duration	(1) Wilt (2) Sterility mosaic (3) Alternaria blight	Select fields with no previous record of wilt Select fields which were not under pigeonpea during the past 3 years Destroy volunteer, perennial or ratooned pigeonpea in the vicinity prior to planting Select multiple disease resistant variety such as DA 110, ICP 9174
South-western India	Medium-duration Short-duration	(1) Wilt (2) Sterility mosaic (3) Dry root rot	Select fields in which wilt was not noticed in the previous years Select a field which was not under pigeonpea during the past 3 years Destroy volunteer, perennial and ratooned pigeonpea in the vicinity Select wilt and sterility-mosaic-resistant varieties such as ICPL 87119, ICPL 87015
North-western India	Short-duration Long-duration	(1) Wilt (2) Sterility mosaic (3) Phytophthora blight	Select a well-drained field Provide better drainage through broad beds or ridges Select tolerant varieties such as ICPL 83024, ICPL 87, ICPL 151 Treat seed with metalaxyl Spray with metalaxyl 15 and 30 days after sowing if needed
Eastern Africa	Long-duration	(1) Wilt (2) Cercospora leaf spot (3) Powdery mildew	Select wilt-resistant varieties such as ICP 9145 Intercropping with sorghum and maize Follow 3-year crop rotations with cereals such as sorghum and maize
Centra America	Long-duration	(1) Witches' broom	Resistant varieties Vector control Avoid perennial cultivation

Source: Reddy *et al.* (1993c).

states of India (Reddy *et al.*, 1990b). The disease appears to be restricted to Asia, being reported from Bangladesh, Myanmar, Sri Lanka and Thailand, as well as India.

Capoor (1952) established the infectious nature of the disease through graft transmission to healthy pigeonpea. He also reported sap transmission of the causal agent but further attempts at mechanical transmission have failed. However, the possibility of mites hidden in the stem pieces contributing to transmission existed. At ICRISAT, graft transmission of sterility mosaic was proven by utilizing stocks or scions of pigeonpea completely devoid of mites (Reddy *et al.*, 1990b). A most important contribution was made by Seth (1962) who showed that the sterility mosaic causal agent is transmitted under natural conditions by an eriophyid mite, *Aceria cajani* Channa Basavanna. *A. cajani* is a worm-like, eriophyid mite, about 200–250 µm long (Reddy *et al.*, 1990b). The mites have short life cycles of less than 2 weeks and include egg, two nymphal and adult stages. Mites feed by puncturing the plant tissues and sucking sap through their stylets. The presence of *A. cajani*, even in large numbers, is often unnoticed as it does not cause visible damage to the leaves.

As the causal agent of sterility mosaic is unknown, information on host range and physiological specialization is based on symptoms observed on inoculated plants using the mite vector (Reddy *et al.*, 1990b). To date, the causal agent is known to infect only *Cajanus* spp. Recently, a wild relative of pigeonpea, *Cajanus scarabaeoides*, was found to support both the causal pathogen and mite vector during the off-season (Reddy *et al.*, 1993d). Previously off-season survival was attributed only to volunteer and ratooned pigeonpea. Three wild relatives of pigeonpea can be infected with the sterility mosaic pathogen through artificial inoculation using the mite vector: *Cajanus scarabaeoides*, *C. platycarpus* and *C. cajanifolius*. These three hosts also support limited multiplication of the *A. aceria*. The life cycle of sterility mosaic is shown in Fig. 10.8.

Between 1987 and 1990, 16 pigeonpea genotypes were tested for their reaction to nine different sterility mosaic samples (Reddy *et al.*, 1993a). Large variation in disease incidence and symptom expression of the genotypes was noted between locations, in different seasons, and in tests at the respective locations and at ICRISAT Center. The variation in symptom expression was less than that in disease incidence. Based on the reaction of seven genotypes (ICP 2376, ICP 7035, ICP 8862, ICP 8863, ICP 10976, ICP 10984 and ICP 11146) in 51 field and pot tests, the nine samples were grouped into five distinct variants (Table 10.7). The sample from Gwalior was designated as variant 1; the Badnapur and Patancheru samples represented variant 2; the Coimbatore, Kumarganj and Pudukottai samples represented variant 3; the Bangalore and Dholi samples represented variant 4; and the Kanpur sample represented variant 5.

Variability among different pigeonpea genotypes with regard to reaction to sterility mosaic is likely to be due to the presence of different *A. cajani* biotypes or different species of *Aceria* or the occurrence of strains of the causal agent. Polymerase chain reaction (PCR) based techniques (primers prepared for a conserved region in the ribosomal DNA) developed at SCRI allow distinction of different species of eriophyid mites (Fenton *et al.*, 1995). These techniques are being applied to the sterility mosaic problem to determine if different species of *Aceria* or

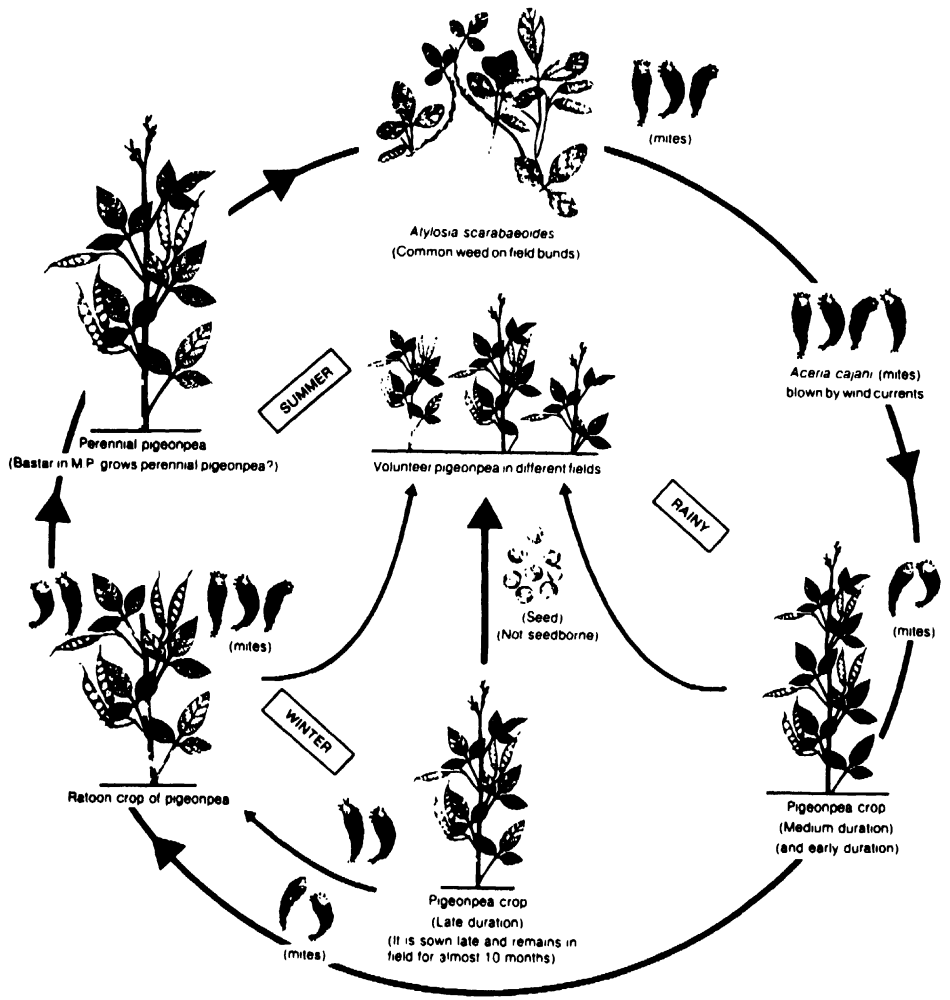

Fig. 10.8. Proposed disease cycle for sterility mosaic (courtesy of Ghanekar *et al.*, 1992).

different biotypes of *A. cajani* exist on pigeonpea and related legumes in Asia in a collaborative project between SCRI and ICRISAT (M.V. Reddy and A.T. Jones, ICRISAT, India, 1996, personal communication). It is hoped that suitable diagnostic tools will be developed for facilitating epidemiological studies and for critical evaluation of sources of resistance.

Symptoms

In the field, sterility mosaic can be easily identified as patches of bushy, pale green plants without flowers or pods (Reddy *et al.*, 1990b). The leaves of infected plants

Table 10.7. Reaction of pigeonpea differential genotypes to different variants of the sterility mosaic pathogen in India (1987–1990).

	Sterility mosaic reaction				
Genotyes	Variant 1	Variant 2	Variant 3	Variant 4	Variant 5
ICP 2376	R	RS	S	S	S
ICP 7035	R	R	R	R	S
ICP 8862	R	R	R	R	S
ICP 8863	S	S	S	S	S
ICP 10976	R	RS	R	R	S
ICP 10984	R	R	R	S	R
ICP 11146	R	R	R	S	S
Isolates	Gwalior	Badnapur Patancheru	Coimbatore Kumargunj Pudukottai	Bangalore Dholi	Kanpur

R = Resistant – no symptoms; S = Susceptible – mosaic symptoms; RS = ring spot.
Source: Reddy *et al.* (1993a).

are small with light and dark green mosaic (Plate 26). Mosaic symptoms initially appear as vein-clearing on young leaves. When infection occurs at 45 days after emergence or later, only some parts of the plant may show symptoms, while the remaining parts appear normal. Some pigeonpea varieties, e.g. ICP 2376, exhibit ringspot leaf symptoms (green islands surrounded by chlorotic areas) against some variants. These indicate localized infection as such plants produce normal flowers and pods. Strains of sterility mosaic prevalent in the Bihar state of India and in Nepal cause severe internodal shortening of the branches and clustering of leaves which sometimes become filiform.

Epidemiology

As mite colonies are usually associated with diseased plants, the possibility that sterility mosaic may be caused by mite feeding damage was considered (Reddy *et al.*, 1990b). The establishment of mite colonies in healthy pigeonpea cultivars known to be susceptible to sterility mosaic was therefore tested to eliminate this possibility. ICP 8136, which favours mite multiplication but is resistant to the disease, was used to develop healthy mite colonies which could be used for further experiments.

Understanding of the pathogen–vector relationship is critical to our knowledge of the epidemiology of the disease (Reddy *et al.*, 1989). The acquisition access period was 5–10 min while the inoculation access period was 30 min. Transmission of the pathogen by the mites appears to be of the persistent type (Reddy *et al.*, 1989). The adult and all nymphal stages of mites are able to transmit the sterility mosaic pathogen. Since they do not possess wings, the dispersal of *A. cajani* is passive, and in nature mostly by wind.

Although pigeonpea is the most common host of the vector, *C. scarabaeoides* also supports *A. cajani* under natural conditions including during the summer season (Reddy *et al.*, 1993d). *C. scarabaeoides* is the most widely distributed wild relative, being predominant in ungrazed fields. Symptoms similar to sterility mosaic have also been observed on plants under natural conditions, indicating that *C. scarabaeoides* is an alternative host of the sterility mosaic pathogen and *A. cajani*. This may explain the recurrence of sterility mosaic in areas where cultivated pigeonpeas are absent during the summer season (Reddy *et al.*, 1993d). The disease is not seedborne.

Variation in disease incidence over locations and seasons is one of the characteristic features of sterility mosaic. Experiments conducted at ICRISAT Center showed a negative correlation between *A. cajani* populations and low relative humidity (Reddy and Raju, 1993). Relative humidity appears to be a critical factor for mite survival during the summer season. During the period 1980 to 1990, a sterility mosaic epidemic occurred in 1990 when the highest summer rainfall was recorded. It is clear that further investigations on the epidemiology of sterility mosaic and its vector and the effects of macro- and microclimatic parameters on disease incidence and severity will enhance our understanding of the epidemiology of this disease.

Effects on Yield and Grain Quality

As for wilt, when a pigeonpea plant is infected by sterility mosaic early, the plant becomes sterile and yield loss is total (Reddy *et al.*, 1990b). Susceptible genotypes infected during the first 45 days of crop growth were mostly sterile and showed crop losses up to 95% (Reddy and Nene, 1981). As the plants became older, their susceptibility to sterility mosaic decreased. Such plants show partial sterility and still produce pods on unaffected branches. The disease was estimated to cause an annual loss of 205,000 tonnes of grain in India during the period 1975–1980 (Kannaiyan *et al.*, 1984). In recent years losses caused by the disease have increased but precise estimates are lacking (Zote *et al.*, 1991).

Disease Control

Most effort in managing sterility mosaic has been placed on the identification and use of host plant resistance. Considerable progress has been made in developing screening techniques, identifying resistance sources and developing resistant cultivars. A leaf-stapling technique has been developed to inoculate plants both in field and pot experiments (Nene and Reddy, 1976a). A diseased leaflet is folded on to a primary leaf of a healthy seedling in such a way that the undersurface of the diseased leaflet contacts both surfaces of the healthy one. These are then stapled together. The advantages of this method are that it facilitates inoculation at the primary leaf stage and disease symptoms are rapidly expressed (Nene and Reddy, 1976a). This technique is very useful for confirming resistance of lines

observed as promising under field conditions, and for disease inheritance and strain identification studies.

The infector-hedge field-inoculation technique, described by Nene *et al.* (1981a), consists of growing a hedge of a susceptible cultivar on the upwind border of a field to be used as a screening nursery. When the seedlings of the hedge are about 10 days old, they are inoculated either by leaf-stapling (Nene and Reddy, 1976a), or by spreading diseased twigs infested with mites among the seedlings. The pathogen and mites multiply on the hedge plants and serve as a source of inoculum for wind dispersal on to test materials during the cropping season. Once a good hedge is established, it can be effective for two or three seasons. The hedge is frequently pruned to promote fresh growth and encourage mite multiplication. In the screening nursery, rows of a susceptible cultivar (BDN 1 or ICP 8863) are sown after every 10 rows of test cultivars to serve as indicator rows for disease spread.

The spreader-row inoculation method is another field inoculation technique, wherein instead of a single hedge several rows of a susceptible cultivar are sown throughout the field about 4 months in advance of the test crop (Nene *et al.*, 1981a). The frequency of spreader to test rows is 1:10. In this method, although a more uniform disease spread is achieved more quickly than by the infector-hedge method, the maintenance of several spreader rows in the field poses land preparation, irrigation and other management problems.

Resistance to sterility mosaic was first reported by Alam (1931) in sabour 2E (Rahar) and other sabour types of pigeonpea. Ramakrishna and Kandaswamy (1972) reported that NP(WR) 15, P 1100, P 1289, P 1778, P 2621 and P 4835 showed mild mosaic symptoms, and less than 3% sterility mosaic incidence in Coimbatore. Systematic efforts to identify sources of resistance were initiated at ICRISAT Center in 1975 (Nene and Reddy, 1976b; Nene *et al.*, 1981b). After screening all the pigeonpea germplasm accessions in the ICRISAT gene bank, 326 resistant lines (i.e. with no visible symptoms) and 97 tolerant lines (ringspot symptoms) were identified (Nene *et al.*, 1981b). Among the resistant lines, 62 were original germplasm accessions while the remainder were selections from accessions that showed segregation for resistance and susceptibility. Since 1976, by using the ICAR–ICRISAT Uniform Trial for Pigeonpea Sterility Mosaic Resistance, it has been possible to re-test resistant sources identified at ICRISAT at different locations within India to confirm their resistance to sterility mosaic (Nene *et al.*, 1989; Amin *et al.*, 1993a). Lines ICP 6997, 7035, 7197, 7234, 7867, 8094, 8862, 10976, 10977, 10979, 10996, 11049, 11204, 11206, ICPL 342, 355, 366, 8324, BSMR 235, DPPA 85-2, 85-13, 85-14 and 85-15 have been (Tables 10.3 and 10.4) identified as resistant or tolerant across all the locations.

With the identification of good sources of resistance to sterility mosaic, breeding for resistance has progressed at many different locations in India including Pantnagar, Pudukkotai, Dholi, Badnapur, Kanpur, Rahuri, Faizabad and ICRISAT Center. Among the earlier varieties developed, NPWR 15 has some tolerance to sterility mosaic, Bahar is resistant to sterility mosaic but highly susceptible to fusarium wilt, and the recently released ICRISAT early-maturing line ICPL 151 has tolerance to sterility mosaic. Several other lines – ICPL 146, 269,

8327. DA 11, 12, 13, 15, 51, MA 97, Sehore 367, DPPA 84-61-3, 84-8-3, Pant A 104, 8505, 8508, Bhavanisagar 1 and NPRR 1, are being tested in the All India Coordinated Trials and have shown resistance to sterility mosaic.

Sharma *et al.* (1984) reported that susceptibility to sterility mosaic disease was dominant over resistance and tolerance, and that the tolerant reaction was dominant over resistance in certain lines. Two loci and more than two alleles at each locus were suggested to be controlling reactions in different crosses. Singh *et al.* (1983) found resistance to be governed by four independent non-allelic genes (Sv_1, Sv_2, sv_3, and sv_4). At least one dominant and one recessive gene are necessary for expression of resistance.

In sterility-mosaic-resistant genotypes, the leaf cuticle and the epidermal cell walls were found to be thicker than in susceptible genotypes (Reddy *et al.*, 1995). For example, in ICP 7035 and ICP 8862 the cuticle thickness was 3.79 and 3.03 μm, respectively. The cuticle thickness in susceptible genotypes varied from 1.52 to 2.27 μm. Interestingly, the stylet length of the mite vector *A. cajani* was found to be less than 3.0 μm indicating that the stylets may not reach the epidermal cells of resistant genotypes. Therefore it is likely that the resistance in these genotypes is based on the inability of the vector to transmit the pathogen. Currently graft inoculation tests are in progress to assess whether the pathogen can multiply in the resistant lines.

Some attention has also been paid to cultural and chemical methods to control sterility mosaic. Suggested cultural practices include: (i) select fields away from perennial or ratooned pigeonpea; (ii) destroy sources of sterility mosaic inoculum, i.e. perennial or ratooned pigeonpeas; (iii) uproot infected plants at an early stage of disease development and destroy them; (iv) rotate crops to reduce inoculum levels and vector populations; and (v) use a sole cropping system with optimum plant population (Tables 10.5 and 10.6). Suggested chemical control measures include (i) dressing seed with 25% carbofuran or 10% aldicarb at 3 g kg^{-1} seed and (ii) spraying acaricides or insecticides like karathane, chinomethionate, metasystox at 0.1% to control the mite vector in the early stages of plant growth (Reddy *et al.*, 1990b) (Table 10.5).

CONCLUSIONS

Though the pigeonpea plant is infected by more than 210 pathogens including fungi, bacteria, viruses, phytoplasmas and nematodes (Nene *et al.*, 1996), broad knowledge is limited to only a few diseases including wilt, sterility mosaic, phytophthora blight, alternaria blight, cercospora leaf spot and powdery mildew. For these diseases, information on biology, epidemiology and management is available, yet information on mechanisms of resistance and variability in the major pathogens is very limited. Future research in these areas is essential to support ongoing resistance breeding. In general, knowledge of diseases occurring in Africa and the Americas is very limited. Wide-reaching surveying is needed to provide comprehensive information on the global distribution of pigeonpea diseases.

For wilt, several lines with high levels of resistance have been identified

(Tables 10.3 and 10.4) and cultural practices such as inter- or mixed-cropping with cereal crops such as sorghum and maize (Tables 10.5 and 10.6) have been shown to enhance disease management. For sterility mosaic, basic questions such as its aetiology remain unanswered. Urgent attention is being given to identifying the causal agent of this disease through a collaborative project with SCRI. Though the causal agent of the disease is not known, management options are available through the identification of several apparently immune lines (Tables 10.3 and 10.4). For phytophthora blight, lines with field tolerance to the disease have been identified and an integrated disease management strategy involving raised seedbeds, field tolerant lines, seed dressing with metalaxyl and intercropping with fast-growing, short-stature legumes such as urd bean, appears to have promise (Tables 10.3, 10.4, 10.5, 10.6). For foliar diseases such as powdery mildew, alternaria blight and cercospora leaf spot, field tolerant lines and effective chemical sprays are available (Tables 10.3, 10.4, 10.5 and 10.6).

As local landraces are being rapidly replaced by improved cultivars, some of the minor diseases listed in Table 10.1 may assume major importance as has been seen for phytophthora blight and cercospora leaf spot on short-duration pigeonpea. Further work on monitoring the distribution, incidence and severity of these diseases is needed. Also, most of the information on disease management has been developed independently for individual diseases of pigeonpea and largely relates to host plant resistance and chemical control. Very little information is available on management of multiple diseases and on other methods of disease management, and especially, strategies for integrated management. Biological control using antagonistic fungi and bacteria should be further investigated, especially for soilborne diseases, and possibly exploited to a greater degree. Available information on integrated management of individual diseases and their management in different agroecological zones/production systems is presented in Tables 10.5 and 10.6. An additional major drawback to the impact of management methods for pigeonpea diseases is that, with the exception of resistant cultivars such as ICP 8863 (Maruti) in India and ICP 9145 in Malawi, few of the other disease management methods reach farmers' fields. For example, the simple method of elimination of sterility-mosaic-affected plants in the off-season can totally control this disease but this is very rarely practised. There is an urgent need for adaptive research and farmer participatory research on the management of pigeonpea diseases.

Since pigeonpea is grown in different production systems and frequently suffers from more than one disease at the same time, there is need to develop management packages for the several major diseases prevalent in a particular system. When pigeonpea is affected by more than one disease and grown in a specific cropping system, irresolvable problems may be encountered for disease management. For example, in Nepal and India, in the majority of pigeonpea production systems, both wilt and sterility mosaic affect the crop. Certain management practices which reduce wilt, increase sterility mosaic. Intercropping with cereals such as sorghum and millet reduces wilt incidence but increases incidence of sterility mosaic. Hence, the importance of multiple disease resistance in pigeonpea, where the resource-poor farmers cannot afford chemical inputs, cannot be overemphasized. Recently, the line ICPL 87119 (developed at ICRISAT), both wilt and

sterility resistant, has been released for general cultivation in India under the name 'Asha', meaning hope.

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DISEASES OF LUPINS

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INTRODUCTION

When Gladstones (1970) wrote his review the total world area sown to lupins was 980,702 ha, with an annual production of 753,224 t of seed. In 1995, in Australia alone, more than 1.31 million hectares of lupins were sown and the total seed production was 1.33 million tonnes (FAO, 1996). The vast majority of this production was from *Lupinus angustifolius*. However, lupins are also important in other parts of the world for seed protein as a substitute for soyabean meal. Their forage is important for ruminants (Hill, 1977, 1986, 1988, 1991). Their ability to fix atmospheric nitrogen on infertile soils of poor nitrogen status is also important (Gadgil, 1971a, b, c; Wendelken, 1974; Palaniappan *et al.*, 1979).

Research on lupins began in Germany (Gladstones, 1970). From Germany, lupins spread to Poland and the former USSR. They were then carried by migrants from the Mediterranean region to many other parts of the world including South Africa (Packendorf, 1986; Mey, 1994), and the United States, particularly Georgia and Alabama (Wells and Forbes, 1982; Reeves and Mask, 1992; Santen *et al.*, 1994) and more recently to California (Feyler, 1986; D.H. Putnam, California, 1994, personal communication). In Chile, *L. albus* and *L. mutabilis* are important (Baer, 1982; Baer *et al.*, 1994); in New Zealand *L. arboreus* and the Russell lupin are used; while in Germany, *L. polyphyllus* is important.

Unlike many of the other species covered in this book, commercial lupins are not a single species. There are three major regions of genetic diversity of lupins: South America (Planchuelo-Ravelo, 1984), the Mediterranean region and North Africa (Gladstones, 1974) and the west coast of North America (Dunn, 1984). Gross (1986) considers that all of the lupins in the world originated in South America. They have spread by either continental drift, as with the Mediterranean group, or by natural migration assisted by humans, and evolution. The Mediterranean group can be divided into rough- and smooth-seeded lupins (Gladstones, 1970). The latter small group provides most cultivated lupins. The

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smooth-seeded group consists of *Lupinus albus* (white lupin), *L. angustifolius* (narrow-leafed lupin) and *L. luteus* (yellow lupin). Among the rough-seeded species, the only commercial species is *Lupinus cosentinii* (Western Australian sand plain lupin). Low alkaloid genotypes of this species have been selected and Buirchell (1994a, b) is working on other rough-seeded Mediterranean lupins to select similar genotypes from *L. atlanticus*, *L. digitatus*, *L. palaestinus*, *L. pilosus* and *L. princei*. Among the many lupins that originate from South America (Planchuelo-Ravelo, 1984), only *L. mutabilis* is important as a crop. Breeding of a low alkaloid genotype of this species by Baer and Gross (1983) should increase its potential considerably; however, initial agronomic tests in South America with sweet *L. mutabilis* have not been promising (Baer *et al.*, 1994).

Besides their seed, all the above species have considerable potential for the production of high quality green forage for ruminants (Burtt and Hill, 1990a, b). They are also efficient fixers of atmospheric nitrogen for following or companion non-legume crops (McKenzie and Hill, 1984; Doyle *et al.*, 1988; Herridge, 1988; Herridge and Doyle, 1988). North American *Lupinus arboreus* is used for nitrogen fixation with *Pinus radiata* and in soil conservation (Gadgil, 1971a, b, c; Wendelken 1974). *Lupinus polyphyllus* is used in forestry in Germany (Melzer and Hertel, 1981; Melzer and Lucke, 1984) and *L. nootkatensis*, from Alaska, is used for soil conservation in Iceland (Magnússon, 1995). The final lupin of commercial interest is a hybrid, the Russell lupin comprised of *L. polyphyllus* and *L. arboreus*; however, George Russell left no specific details of the other species he had used in the cross (Gorer, 1970). The Russell lupin is being investigated in New Zealand as a potential forage for sheep, especially on low phosphate, acidic, hill and high country soils (Scott *et al.*, 1989; Covacevich, 1991; Kitessa, 1992; Hill, 1994).

There has been a major increase in the total world production of lupins since 1970. The FAO statistics (FAO, 1996) only provide figures for lupins sown for seed production and give no indication of their utilization in areas such as soil conservation or forestry. Although the total area sown to lupins has increased by about 50% since 1970 to 1.53 million ha, changes occurring over the period have masked major shifts in the regions of world production. In 1970, the former USSR grew 620,000 ha of lupin to produce 525,000 t of seed but, by 1995, it was reduced to only 24,200 t of seed from 27,000 ha. There has also been a major reduction in lupin production in South Africa from 179,000 ha in 1970 to 20,000 ha in 1995. In Europe, only Poland remains a major producer with 145,000 t from 110,400 ha in 1995. There has been an increase in interest in lupin seed production in South America since 1970 (Mora, 1986), and of over 18,000 ha now grown, 14,000 ha are in Chile (FAO, 1996).

These substantial changes in the major lupin-producing areas from northern Europe and the Ukraine to the predominantly Mediterranean environment of south-western Western Australia, which now grows over 1 million hectares (Perry *et al.*, 1994) of the total Australian production, seem likely to reflect changes in the type and significance of the major diseases that affect the lupins. Indeed, Gladstones (1970) made no mention of *Diaporthe toxica*, the recently discovered teleomorph of *Phomopsis leptostromiformis* (Williamson *et al.*, 1994), as a problem of lupin cultivation. However, by 1988 it was seen as a major limiting

factor in the utilization of lupin crop residues by ruminant animals (Gladstones, 1988). It has also led to a large breeding effort in Western Australia for producing resistant genotypes safe for grazing sheep during the Western Australian summer (Gladstones, 1994).

Four reviews (Gladstones, 1970; Bellido, 1984; Plancquaert, 1984; Baylis and Hamblin, 1986) have extensively covered the major diseases of domesticated lupins globally. One book has reviewed diseases of *L. albus* (Gondran *et al.*, 1994). At a regional level, Gladstones (1971, 1977) reviewed diseases of lupins in Western Australia. Pennycook (1989) lists all diseases that have been recorded on species of lupins in New Zealand. In addition, Frey (1980) recorded the diseases of *L. mutabilis* in Peru and Bolivia and Jaarsveld (1985) diseases of lupins in South Africa. Gladstones (1970) listed the major diseases of lupins as powdery mildew, anthracnose, fusarium wilt, brown leaf spot, grey leaf spot, bean yellow mosaic and cucumber mosaic. Other fungal diseases and pathogens of lesser importance were pythium root rots, *Rhizoctonia solani*, *Sclerotium rolfsii*, *Botrytis cinerea*, *Ascochyta gossypii* and *Phomopsis leptostromiformis*. In 1970, the role of *P. leptostromiformis* in lupinosis of sheep was not understood and the pathogen was therefore reported as a minor problem of *Lupinus luteus* (Gladstones, 1970).

At the 1984 International Lupin Conference, Bellido (1984) presented information from many countries on the major features of lupin cultivation including their pathogens. Baylis and Hamblin (1986) further contributed to this information in their review of world lupin production. However, in both papers the information on pathogens was not complete. A major review of lupin viruses was made by Jones and McLean (1989). They divided them into aphid-transmitted, thrips-transmitted, nematode-transmitted and two virus-like diseases, leaf curl and witches' broom. By far the largest group are aphid-transmitted and they fall mainly into the potyvirus and cucumovirus groups. Recent literature reports that a range of nematode species are capable of infecting different lupin species; however, none indicates that nematodes are a major problem in the commercial cultivation of lupins with the exception of the review of Baylis and Hamblin (1986), where nematodes are noted as a major problem of lupins in Brazil.

After a synthesis of all of these reviews, the following major diseases have been selected for extensive treatment in this chapter: anthracnose, brown leaf spot, lupinosis, *Rhizoctonia* diseases, bean yellow mosaic and cucumber mosaic. The information on other fungal pathogens of lupins is summarized in Table 11.1 while that on minor virus diseases is given in Table 11.2.

ANTHRACNOSE

Aetiology

Until recently it was considered that anthracnose of lupins was caused by the fungus *Colletotrichum gloeosporioides* (Penz.) Penz. & Sacc., the anamorph of *Glomerella cingulata* (Stonem.) Spauld. & Schrenk. (Gondran *et al.*, 1994). However, in a recent review of this disease, Gondran *et al.* (1996a) indicated that the causal organism is probably *Colletotrichum acutatum* Simmonds. A subsequent

Table 11.1. Minor fungal diseases of lupins.

Disease	Causal agent	Distribution	Importance
Fusarium wilt	*Fusarium oxysporum* Schlecht. f. sp. *lupini* Snyd. & Hans.; *F. oxysporum* f. sp. *radici lupini* Weimer and other spp.	Widespread	Important in Eastern Europe
Cylindrocarpon root rot	*Cylindrocarpon obtusiflorum* Looke	France	Minor
Thielaviopsis root rot	*Thielaviopsis basicola* (Berkley & Broom) Ferraris	France	Locally important
Grey mould	*Botrytis cinerea* Pers.	Widespread	Mainly a problem in windy and cold conditions
Charcoal rot	*Macrophomina phaseolina* (Tassi) Goid.	France	Locally important; leads to empty pods
Pythium root rot	*Pythium irregulare* Buisman	Australia, Denmark, New Zealand, South Africa	Minor
White mould	*Sclerotinia sclerotiorum* (Lib.) de Bary	Australia, Brazil, Denmark, New Zealand, Peru	Locally important
Grey leaf spot	*Stemphylium vesicarium* (Wallr.) Simmons; *S. botryosum* Wallr.	Australia, France, Poland, SE USA	Major problem in some seasons
Powdery mildew	*Erysiphe polygoni* DC.; *E. cichoracearum* DC.	Denmark, France, New Zealand, Peru, South Africa, Spain	Generally minor but can be a major problem of *L. angustifolius* in Spain
Rust	*Uromyces lupinocolus* Bubak.; *U. renovatus* Sydow	Bolivia, Denmark, England, France, Peru	May be a problem in countries with a Mediterranean climate
Fusarium pod rot	*Fusarium solani* (Mart.) Sacc.	France, South Africa	Locally important

Sources: Gladstones (1970, 1977); Frey (1980); Rataj-Guranowska (1981); Oram (1983); Bellido (1984); Plancquaert (1984); Jaarsveld (1985); Baylis and Hamblin (1986); Golovchenko (1986); Gondran (1988); Pennycook (1989); Sweetingham (1989); Bateman *et al.* (1991); Lewartowska *et al.* (1991); Gondran *et al.* (1994); Lewartowska and Frencel (1994).

Table 11.2. Virus diseases of minor importance in natural infections of lupins.

Virus	Distribution	Importance
Aphid-transmitted viruses		
Clover yellow vein potyvirus	Australia, Europe, USA	Only a problem when *Trifolium repens* and lupins are grown together
Bean common mosaic potyvirus	Poland	Minor
Peanut mottle potyvirus	SE USA	Potentially serious in peanut growing areas of Georgia
Bidens mottle potyvirus	Florida, USA	Potential problem in Florida
Soyabean mosaic potyvirus	South Africa	Minor
Peanut stunt cucumovirus	Poland	Minor
Alfalfa mosaic alfamovirus	Australia, Germany, Poland	Minor
Broad bean wilt fabavirus	Germany	Insignificant
Pea enation mosaic virus	Australia, Germany	Insignificant
Soybean dwarf luteovirus (syn. subterranean clover red leaf luteovirus)	Tasmania, New Zealand	Minor except in *L. albus* and *L. cosentinii*
Lettuce necrotic yellows rhabdovirus	Queensland	Insignificant
Thrips-transmitted viruses		
Tomato spotted wilt tospovirus	USA	No report of infection of cultivated lupins
Nematode-transmitted viruses		
Pea early-browning tobravirus	Poland	Insignificant
Tomato black ring nepovirus	Germany, Poland	Insignificant

Sources: Gladstones (1970); Frencel and Pospieszny (1977, 1985); Schmidt (1979); Borges (1982); Garnett and McLean (1983); Bellido (1984); Plancquaert (1984); Foster and Musgrave (1985); Pospieszny and Frencel (1985); Baylis and Hamblin (1986); Garnett (1988); Vroon *et al.* (1988); Jones and McLean (1989); Pietersen and Garnett (1992); Pennycook (1989); Milford *et al.* (1993).

report by Reed *et al.* (1996) also cited this species as the causal pathogen of anthracnose of ornamental lupin in the United Kingdom. The availability of molecular-based diagnostic tools has led to this and other pathogens, previously described as *C. gloeosporioides*, being reclassified as *C. acutatum* (Sreenivasaprasad *et al.*, 1994). A recent paper from the United Kingdom (Pring *et al.*, 1995) reported that *L. angustifolius* was susceptible to infection with three strains of the tropical species *Colletotrichum capsici* (Syd.) Butler & Bisby. However, there are no reports of this species being a problem for lupins in the field possibly because at present few lupins are being grown in tropical regions. Anthracnose diseases are also reviewed on soyabean (see Sinclair, Chapter 3, this volume), bean (see Allen *et al.*, Chapter 4, this volume), cowpea (see Allen *et al.*, Chapter 5, this volume) and tropical pasture legumes (see Lenné, Chapter 13, this volume).

Biology

The characteristic growth of *Colletotrichum* is formation of conidia in acervuli under the cuticle or epidermis of the host plant. When the conidia mature, they erupt through the epidermis to produce the characteristic pink lesions. The conidia, which are released in droplets, are elongated with rounded ends and are narrower in the middle. They are produced among scattered long dark brown setae in the acervulus.

Symptoms

The symptoms of anthracnose can be confused with a number of other diseases which infect lupins. Infected seed are small, malformed and have distinct lesions. Once seedlings emerge, under humid conditions pink spots form on the hypocotyl, the young root, the stem and the cotyledons. Stems become bent over by flowering. Pink lesions with a brown halo are formed on leaves and pods. Because of the similarity of the symptoms to those produced by *Fusarium avenaceum* and *F. culmorum*, a definite diagnosis can only be made by microscopic identification of the spores (Gondran *et al.*, 1994).

Epidemiology

Anthracnose is a major disease of lupins in areas with wet humid summers. It is a common disease in western Europe (Gondran *et al.*, 1994, 1996b) and is considered to be the most dangerous threat to increased lupin production in Germany. The major form of transmission is by infected seed and diseased crop residues. Under warm humid conditions, anthracnose can be spread rapidly by rain-splash from both diseased crop residues and newly infected plants.

Crop Loss

Crop losses from anthracnose can be almost total. In Russia, it has caused major problems in *L. albus* and *L. luteus* crops in which 86% of *L. luteus* and 89% of *L. albus* plants were infected by pod formation (Yakusheva, 1996a). It is not surprising that similar levels of infection were reported in the same two lupin species in the Ukraine (Korneichuk, 1996). The disease has also been widely reported in the Americas (Baer and Hashagen, 1996). In trials in Chile, susceptible cultivars of *L. albus* yielded 2060 kg ha^{-1} of seed compared with a resistant cultivar which produced 5720 kg ha^{-1}. Similar major crop losses have been reported from Florida (Prime *et al.*, 1996). A recent outbreak in an introduction of *L. albus* was recorded in Western Australia (Sweetingham *et al.*, 1995; Cowling *et al.*, 1996; Agriculture Western Australia, 1996).

In New Zealand, anthracnose killed extensive areas of *L. arboreus*, used in forestry with *Pinus radiata* (Dick, 1994). The outbreak began in 1988 and, over a 4 year period, both the longevity of this perennial lupin and its seed production were considerably reduced. It is not known how the fungus reached New Zealand but it was probably introduced on seed.

Control

It has been suggested that the presence of high levels of lupin alkaloids may help plants to resist anthracnose. The devastation of the bitter *L. arboreus* in New Zealand (Dick, 1994) and the work of Baer *et al.* (1996) seem to indicate that this is not the case. Baer *et al.* (1996) found that bitter cultivars could be susceptible and that lines with low levels of alkaloids could show some degree of resistance. This was confirmed by the work of Baer and Hashagen (1996) who found that the sweet cultivar Rumbo of *L. albus* showed tolerance to the disease. Cultivar differences in levels of resistance to anthracnose were also observed in *L. angustifolius* and *L. mutabilis*. Similar results were obtained by Gondran *et al.* (1996a) working with *L. albus* in France. Thus, although it would appear that breeding of resistant cultivars is possible, progress in this area has not been great.

If the disease is not present, probably the best line of defence is an efficient quarantine system, as the recent Australian experience shows (Sweetingham *et al.*, 1995; Cowling *et al.*, 1996). If the disease is already present, the presence of the fungus on the seed can be tested before sowing and in regions where anthracnose is a problem seed should be routinely tested before sowing. The disease can also be transmitted from crop residues, so stringent crop hygiene is important. There is some suggestion of the possibility of chemical control. Seed infected up to 13% can be dressed before sowing with a mixture of 105 g ipridione with 53 g carbendazim per 100 kg of seed (Gondran *et al.*, 1994). Further, Gondran *et al.* (1996a) found that when plants were artificially infected both prevention or cure of the symptoms could be obtained by spraying with mixtures of cyproconazole and chlorothalonil, carbendazim and iprodione or flutriafol and chlorothalonil at various doses.

BROWN LEAF SPOT

Aetiology

This disease is caused by the fungus *Pleiochaeta setosa* (Kirchn.) Hughes and affects lupins at two stages of their life cycle. In the seedling stage, it is know as pleiochaeta root rot. On older plants, as the common name suggests, it initially attacks the leaves causing distinct brown lesions (Sweetingham, 1989; Gondran *et al.*, 1994; Sweetingham *et al.*, 1994a).

Biology

With regard to the foliar form of the disease, Gondran *et al.* (1994) reported that it is common in all areas where lupins are grown but is particularly prevalent on autumn-sown crops of *L. albus* in France. They consider that in association with anthracnose it is the most serious disease of autumn-sown lupins. Infection is usually from rain-splash. Conidial germination on lupin leaves can reach 80–90% within 4–6 h of inoculation; the germ tube can produce an appressorium and subcuticular infection hyphae can form (Harvey, 1977). Infection is usually through direct penetration of the cuticle, though entry via stomata can occur (Harvey, 1977). Once inside the cell, the infection peg expands within the softened cell wall material to produce subcuticular infection hyphae. Sporulation occurs from brown necrotic areas on leaves from which conidia are dispersed. The spores are brown in colour with four setae and are multicellular with thick walls. Infected leaves fall to the ground within a few days of their infection and thus provide a reservoir of inoculum for subsequent seasons.

Symptoms

In fields that had previously grown lupins, emerging seedlings were attacked on the tap root and the hypocotyl. The cortex of the tap root was girdled leaving the stele intact. In the foliar form, it can either be seed-transmitted or arise from infected crop residues. In humid conditions, with temperatures in the range 10–15°C, after seedlings emerge brown spots form on the cotyledons and leaflets. These spots enlarge, become oval, and eventually reach about 1 cm in diameter. The disease spreads from leaves to stems and plants die. Harvey (1977) reported invasion of the fungus in leaves where symptoms may appear as little as 17 h after infection. Plants that survive continue to show brown lesions on leaves and pods. Gondran *et al.* (1994) give a series of coloured illustrations of symptoms in *L. albus*. Because frost can produce similar symptoms, definite identification can only be made microscopically.

Epidemiology

In Western Australia, the major factor determining severity of both forms of the disease is the soil population of *P. setosa* spores. The greatest concentration of spores is in the top 2 cm of the soil profile (Sweetingham, 1991). After spore population, the major determinant of disease severity is climate. Thus, in France, it is a major problem in autumn-sown crops but only a minor problem with spring sowings (Plancquaert, 1984; Gondran *et al.*, 1994). In Western Australia, where all lupin crops are autumn sown, the risk of major infection is highest in the colder, lower rainfall regions of the south-west of the state. Because of the lower temperatures, and thus slower growth, young plants in these regions remain at the susceptible four-leaf stage for longer. They are therefore more likely to be infected by rain-splash. After climate soil type is important. Soils with a high clay content apparently produce more splash than coarse sands. Waterlogging also favours the development of the disease (Sweetingham *et al.*, 1993a).

Crop Loss

In Western Australia, the two diseases caused by *P. setosa* are the most common and widespread diseases of lupins, causing greatest damage when they attack plants at the seedling stage. Large numbers of plants are killed. Plants that survive have substantially reduced vigour and the combination of the two factors leads to major reductions in crop yield (Sweetingham, 1991; Sweetingham *et al.*, 1993a). In France, Plancquaert (1984) listed brown leaf spot as a major problem in autumn-sown crops of *L. albus* and *L. angustifolius*. In spring sowings, it occurred on the same two species but tended only to be a problem on *L. luteus* (Plancquaert, 1984). Gondran *et al.* (1994) stated that it was the most serious disease of autumn-sown *L. albus* in France. In winter trials with *L. albus* and *L. angustifolius* in the United Kingdom, the disease had no significant effect on crop yield (Bateman *et al.*, 1991). In other parts of the world such as Canada (Paulitz and Atlin, 1992), Germany (Motte and Beer, 1991) and the USA (Kalis-Kuznia *et al.*, 1991), there are reports of the disease on lupins but it is currently of minor importance.

Control

As with most pathogens, the most suitable method of control is breeding resistant genotypes. Cowling (1988) assessed resistance to both pleiochaeta root rot and brown leaf spot and was able to find a high degree of resistance for leaf spot which was highly heritable. In contrast, there was virtually no resistance to root rot. Resistance to the two diseases did not appear to be genetically related and thus the two characters should be selected for independently. Sweetingham *et al.* (1994b) conducted tests of seedlings of *L. albus*, *L. angustifolius* and *L. luteus* with sand infested with *P. setosa* spores. *Lupinus luteus* was highly resistant to root rot, *L. albus* was highly susceptible while *L. angustifolius* was intermediate. In the field, among the three species there was a high degree of association between resistance to root rot and foliar disease. Subsequently Sweetingham *et al.* (1996)

have reported resistance to root rot in *L. albus* landraces obtained from the Azores and Crete. The resistance appears to be controlled by a single gene. In France, Gondran (1988) has also selected for resistance to brown leaf spot in *L. albus*. Gladstones (1994) suggested that resistant plants tend to have high trace alkaloid levels. However, to date no cultivars resistant to brown leaf spot have been released from either species.

In the absence of resistant lines the most effective control of the seedling disease is to grow plants in areas where lupins have not been previously sown. Sweetingham (1989) found no infection in fields where lupins had not been grown previously. Crop rotation is also important in reducing disease incidence. In Victoria, when lupins were grown after lupins, 63% of plants were infected with brown leaf spot. When lupins followed wheat the level of infection was only 18% (Reeves *et al.*, 1984). If the use of new ground or a long rotation is not possible, the alternative is to sow seed at depths of more than 5 cm. The highest concentration of fungal propagules is in the top 2 cm of the soil profile and their number declines to zero at 10–14 cm below the surface (Sweetingham, 1991). As this form of the disease only infects roots and hypocotyls, deep sowing places germinating seed out of the infection zone and germinating plants elude the disease (Sweetingham *et al.*, 1996).

For plants that are infected with brown spot after emergence, reduction of rain-splash from soil containing fungal spores can reduce the incidence of the disease (Sweetingham *et al.*, 1993a). If a cereal stubble mulch was left on the surface of the soil, the severity of disease decreased as the amount of stubble increased. As the amount of mulch was increased from 0 to 4 t ha^{-1}, lupin seed yield in *L. angustifolius* increased from 0.86 to 1.44 t ha^{-1}. There was little yield response beyond 2 t ha^{-1} of stubble which gave a seed yield of 1.48 t ha^{-1}. The use of procymidone at 1 ml kg^{-1} of seed also helped to reduce the early incidence of the disease. However, only the mulch increased the total dry matter and seed yield at harvest (Sweetingham *et al.*, 1993a).

Chemicals such as prochloraz at 450 g a.i. ha^{-1} and iprodione at 500 g a.i. ha^{-1} can also slow development of the disease (Gondran *et al.*, 1994). However, the most promising form of control would appear to be use of resistant plants. Gondran (1990) reported selection for resistance in *L. albus*. In Western Australia, Sweetingham *et al.* (1994b) developed a method for resistance testing in the glasshouse and compared the resistance of a number of genotypes of *L. albus*, *L. angustifolius* and *L. luteus*. There was considerable interspecific and intraspecific variation in resistance (Sweetingham *et al.*, 1994a). *Lupinus luteus* displayed the highest degree of resistance followed by *L. angustifolius* and *L. albus*. Field studies confirmed the resistance, and use of *L. luteus* on acid sandy soils which have high levels of spores of the pathogen has been recommended.

LUPINOSIS

Aetiology

Lupinosis is an animal disease caused by the infection of lupins by the fungus *Phomopsis leptostromiformis* (Kühn) Bubák. The teleomorph of this fungus was

formerly described as *Diaporthe woodii* Punith. (Holliday, 1989), but is now treated as a new species *D. toxica* Williamson *et al.* (Williamson *et al.*, 1994). In this chapter, I will continue to use the former name because most of the published literature refers to *P. leptostromiformis*.

Biology

In South Africa, Jaarsveld (1985) reported that although the disease was widespread in Cape Province, he was unable to isolate the pathogen from green immature plants of *L. angustifolius*. Similarly Cowling *et al.* (1984) found it difficult to find symptoms on immature glasshouse-grown plants of *L. angustifolius*. However, if plants were infected with the fungus and plant segments were treated with a paraquat/diquat mix then incubated for 12 days, most treated plants developed *P. leptostromiformis* stromata. Untreated plants remained disease free. Infected plants that had been surface sterilized also showed disease symptoms. Field tests confirmed the reliability of the method. Surveys in Western Australia also established the widespread presence of the disease in most lupin crops (Wood and Petterson, 1985, 1986), while in South Africa it was found on lupin stubbles in all parts of the lupin-producing area of south-western Cape Province.

Symptoms

It is ironical that a disease which is such a major problem to animals eating lupins appears to have very little direct effect on the overall production of the growing plant. It is only after crop maturity that the lesions become obvious. In Western Australia, Gladstones (1977) reported that the fungus developed mainly in stem tissue of *Lupinus angustifolius* and only rarely did lesions appear on the leaves. As the season progresses, sunken bleached patches about 0.5 mm deep appear on stems, and only after crop maturity do stems have a flecked appearance due to the formation of fruiting bodies by the fungus. If there is summer rain or high humidity after the crop maturity, the fungus continues to grow (Gondran *et al.*, 1994). The fungus could only be detected on lupin stubble in South Africa (Jaarsveld, 1985).

Epidemiology

Brown (1984) showed that in *L. albus*, *L. angustifolius* and *L. cosentinii* fallen leaves contained pycnidia but did not show stromata. If the fallen leaves were moistened, conidia were then dispersed from the ground by rain-splash. Recently, Williamson *et al.* (1991) showed that conidia took 4 days to penetrate the cuticle of the lupin plant. Seven days after inoculation, a distinctive coralloid hypha was present between the cuticle and the epidermis. At 20 days, coralloid hyphae could be detected with a microscope at a frequency of 148 cm^{-2} but there were still no visible symptoms. Normal mycelia invaded the subepidermal region of

senescent stems from the coralloid hyphae. Variation in resistance to the disease in *L. angustifolius* is reflected in the size of the subepidermal coralloid hyphae. Highly resistant lines had virtually no large coralloid masses but many darkly staining small coralloid masses. As the degree of resistance fell, the number of large coralloid hyphae increased. The growth rate of the hyphae from the large coralloid hyphae was three to four times faster than from the small hyphae. In highly resistant lines, most of the small coralloid hyphae remained latent and did not colonize the host stems (Shankar *et al.*, 1996).

Wood and Petterson (1985) conducted a survey of the degree of infection of Western Australian lupin seed with *P. leptostromiformis* between 1976 and 1981. They found that 51% (548) of all lupin crops sampled were infected. There was also a highly significant effect of rainfall on the level of infection but no effect of cultivar, year of sampling, or district. In a further survey, Wood and Petterson (1986) discovered that 61% of all lupin crops were infected. There was a strong relationship between seed discoloration and its level of phomopsin A. However, in most samples between 57 to 97% of the phomopsins were located in the testa which makes up only about 22% of the total seed in *L. angustifolius*. Wood *et al.* (1987) found that in an infected crop it was possible to recover viable *P. leptostromiformis* from 96% of seed showing visible symptoms. Primary pods had the highest level of discoloured seed and it was also possible to isolate the fungus from 56% of symptomless seed from primary pods. However, the highest level of phomopsins was in discoloured and furry discoloured seed.

Toxicology and Animal Losses

Although *P. leptostromiformis* is a minor problem of lupins having little direct effect on plant yield, it is a major limiting factor to the utilization of lupin crop residues by ruminants (Gladstones, 1970, 1988, 1994). There was a long search by veterinary researchers in both Australia and South Africa for the cause of the disease of sheep known as lupinosis (Allen, 1986). In 1972, workers in South Africa reported for the first time that lupinosis was caused when sheep ate lupin plant residues that had been infected with *P. leptostromiformis* (Warmelo and Marasas, 1986). The fungus produces two potent hepatotoxins, phomopsin A and phomopsin B (Culvenor and Petterson, 1986). It is thus a major limitation on the use of lupins and lupin residues for animal feeding, particularly sheep.

In sheep, when administered by subcutaneous injection, the LD_{50} of the toxin was 10 μg kg^{-1} of body weight. When 1000 μg of the toxin were injected directly into the rumen as a single dose it had a similar LD_{50}. When the same total amount was administered into the rumen at the rate of 50 or 200 μg per day all the sheep died (Peterson *et al.*, 1987). Sheep that were given intermediate doses of phomopsins suffered moderate to severe liver damage. Even at relatively low doses of phomopsins there were marked reductions in body weight as the sheep stopped eating (Peterson *et al.*, 1987). Hill (1991) reviewed possible strategies that could be used to reduce the incidence of lupinosis in sheep.

There is variation among animal species in their susceptibility to phomopsins. In yearling cattle grazing *L. angustifolius* stubble in New South

Wales, 70 of 80 heifers grazing lupin stubble for 3 weeks developed symptoms of lupinosis. In the following 2 weeks, 35 of the affected animals died and the remaining 35 had to be culled (Mackie *et al.*, 1992). In lactating dairy cows, Hough and Allan (1993, 1994) were able to feed 8 kg of *L. angustifolius* seed with symptoms of *P. leptostromiformis* infection (up to 0.1%) to which 360 μg of phomopsin A kg^{-1} of lupin seed fed was added. This dose was equivalent to the highest amount of phomopsin detected in a lupin seed sample from Western Australia. The experimental rations were fed for 8 weeks and animals were observed for a further 4 weeks. At the end of the experiment there were no differences between control and phomopsin-fed animals in any of the measured parameters which, besides milk yield, included liver function tests, body weight and cow condition. In a comparative trial with pigs and sheep, when fed on seed that was 10% infected, over a 40-week feeding period the sheep developed lupinosis while the pigs remained free of the disease (Allen *et al.*, 1984). Both infected stubble (Allen *et al.*, 1983a; Mackie *et al.*, 1992) and seed (Allen *et al.*, 1983b) can cause the disease. It also appears that the toxicity of the material does not diminish over time as Allen *et al.* (1983a) reported an outbreak of the disease in sheep in the following winter and the second summer after the growth of a lupin crop.

Control

Given the almost complete lack of obvious symptoms on growing lupin crops, the control of the disease by fungicides is not likely to be feasible, or economically viable. Due to the extreme toxicity of the mycotoxins to sheep, large numbers of deaths can follow grazing of infected lupin plants in the summer in Western Australia. At present the only available control measure is to remove animals from grazing on lupins as soon as possible. Although it appears to be less of a problem with cattle, animal deaths will follow extended grazing of infected plant material (Mackie *et al.*, 1992).

The potentially most effective means of control is by breeding for resistance. Under Western Australian conditions, *L. albus* is apparently naturally resistant (Wood and Allen, 1980). However, *L. angustifolius* is highly susceptible. Work by Hamblin *et al.* (1984) pointed the way for resistance breeding with a comparison of four cultivars of *L. angustifolius* sown at three different sowing dates. Although there was no difference in the resistance to the disease among the cultivars, averaged over all sowing dates, there were highly significant interactions between cultivar and sowing date, suggesting that the susceptibility of the plant to the fungus was related to its stage of development. Any resistance breeding programme will need to take into account the host genotype × pathogen genotype × environmental interaction if it is to have any chance of success. In 1986, Allen and Cowling (1986) compared three resistant breeding lines of *L. angustifolius* with susceptible cultivars in a grazing trial. Although sheep grazing on susceptible cultivars and one of the breeding lines rapidly developed clinical lupinosis, sheep on two of the resistant lines showed no sign of the disease and gained weight during the trial.

Resistance to the fungus has been identified in a number of wild populations

of *L. angustifolius* and had been incorporated into advanced breeding lines by the time of the International Lupin Conference in 1986 (Cowling *et al.*, 1986). The heritability of resistance was high and ranged from 0.86 to 0.92 (Cowling *et al.*, 1987). Field trials with advanced breeding lines in Western Australia and the eastern states of Australia confirmed both the resistance to the disease and reduced toxicity to sheep (Cowling *et al.*, 1988; Cowling and Wood, 1989). Young Merino lambs that were weaned onto resistant lupin stubble grew well and showed a low rate of liver damage (Morecombe and Allen, 1993). This breeding programme has led to the release of three new cultivars of *L. angustifolius*: Warrah, Gungurru and Yorrel, which show moderate to moderately high resistance to the disease (Komoll, 1989a, b, c). At present, the disease would appear to be under control in *L. angustifolius*. Animal scientists are also working on the production of sheep vaccine (Ralph, 1990).

RHIZOCTONIA DISEASES

Aetiology

A complex of three diseases of lupins is caused by *Rhizoctonia* spp. Although initially thought to be caused by *Rhizoctonia solani* (Kühn), Sweetingham (1989) conducted a survey in Western Australia and was only rarely able to isolate *R. solani*. Binucleate *Rhizoctonia* spp. were commonly isolated from diseased plants from areas where lupins had previously been grown. Such *Rhizoctonia* spp. are of worldwide distribution and the genus is a member of the *Basidiomycetes*. The fungus produces sclerotia which are embedded in a mycelial matrix. Sclerotia can survive for long periods in soil and germinate and infect host plants when the conditions are favourable. Identification is complicated by the large number of alternative host species. Diseases of lupins caused by *Rhizoctonia* spp. have been reported as a problem on lupin crops in Australia (Sweetingham, 1986a, b, 1989; Sweetingham *et al.*, 1993b), France (Gondran *et al.*, 1994), South Africa (Jaarsveld, 1985) and the United States (Leach and Clapham, 1992; Kuznia *et al.*, 1993). Similar diseases caused by *Rhizoctonia* spp. have also been reported on soyabean (see Sinclair, Chapter 3, this volume), pea (see Kraft *et al.*, Chapter 6, this volume) and chickpea (see Haware, Chapter 9, this volume).

Biology

The fungus survives on organic matter in the soil. At the start of the growing season, fine hyphae expand into the surrounding soil and infect susceptible hosts. Because of the different strains of *Rhizoctonia* spp., there is not only variation in the types of disease caused on lupin plants but there is also a wide range of alternative hosts. There is considerable variation among strains in both pathogenicity and host specificity. The strains that infect lupins have been identified as being from zymogram groups ZG1, ZG3, ZG4 and ZG6. Alternative hosts for ZG1 are

most agricultural plants while ZG3, ZG4 and ZG6 seem to be confined to legumes (Sweetingham, 1989; Sweetingham *et al.*, 1993b).

Symptoms

In Western Australia the disease manifests itself in three distinct forms apparently caused by the four different zymogram groups of *Rhizoctonia* spp.: ZG1, ZG3, ZG4 and ZG6. They produce three different forms of the disease: rhizoctonia patch (ZG1), rhizoctonia hypocotyl rot (ZG3, ZG4) (Sweetingham, 1989) and a root and hypocotyl rot caused by *Rhizoctonia* strain ZG6. Rhizoctonia patch produces characteristic dark brown 'spear-tipped' lesions on roots which pinch off roots. In the field, it manifests itself as circular patches of stunted plants 0.3–5.0 m in diameter (Sweetingham *et al.*, 1993b). Tap and lateral roots of infected plants are pinched off but late infected plants may grow secondary roots and survive (Jaarsveld, 1985). In rhizoctonia hypocotyl rot, the population of the stand is reduced as the fungus infects and kills germinating seedlings. Lesions on the hypocotyls are red-brown. The final form of the disease produces lesions on both hypocotyls and roots. The hypocotyl lesions cannot be distinguished from those of hypocotyl rot but the roots of infected plants have characteristic stubby ends (Sweetingham *et al.*, 1993b). Sweetingham *et al.* (1993b) contains a series of coloured illustrations showing the three forms of the disease.

Epidemiology

In rhizoctonia patch, the incidence of the disease is highest in zero-tilled and direct-drilled lupin stands. Hypocotyl rot is exacerbated by deep sowing (>7 cm). It is also associated with early sowing into warm soils. Sweetingham (1989) also found that the disease was most common in Western Australia on infertile sands.

Crop Loss

There appear to be no published data on the effect of the *Rhizoctonia* spp. complex of diseases on lupin yield.

Control

Because of the large number of alternative hosts and the longevity of the fungus in the soil, control is not easy. Pre-seeding tillage appears to reduce the incidence of rhizoctonia patch by breaking up the fungal hyphae into smaller, less infective fragments. It is possible to do this at seeding by making minor modifications to the drill. Seed treatment prior to sowing with the fungicides Rovral and Sumisclex is sometimes effective in reducing hypocotyl rot. Increasing plant population, to allow for seedling deaths, and sowing at 2–3 cm, to reduce the

exposure of the hypocotyl to infected soil, can also help. It is also recommended not to plant lupins after clovers or medics because of the build-up of the fungus in the soil. Late sowing is also recommended. Control of hypocotyl rot is in some ways counter to the control of the patch in that although late sowing, fungicides, and increased plant population can help so can deeper sowing at 5–6 cm. Again levels of infection tend to be high when lupins are sown following clovers and medics (Sweetingham *et al.*, 1993b). Tests in the USA with a range of fungicides gave no significant consistent effect on either final plant stand or the number of roots showing lesions (Kuznia *et al.*, 1993).

BEAN YELLOW MOSAIC

Aetiology and Biology

Bean yellow mosaic is caused by bean yellow mosaic virus (BYMV) which is a member of the potyvirus group and is of worldwide distribution. It infects a wide range of legumes including soyabean (see Sinclair, Chapter 3, this volume), faba bean (see Jellis *et al.*, Chapter 7, this volume) and clovers (see Mercer, Chapter 12, this volume) and a number of non-legume species. In general, plants infected with BYMV have considerably reduced growth and few survive to produce viable seed. However, where seed is produced, the virus is transmitted in seed and can survive in seed stored for 5 years (Gladstones, 1970). In the field, the main means of transmission of the virus is by aphids from one infected plant to another (Jones and McLean, 1989).

Symptoms

Plants infected with BYMV initially show yellow mottling of leaves, followed by the formation of many small leaves near the top of the plant and curling over of the stem into the form of a shepherd's crook and then death (Gondran *et al.*, 1994). A full description of the symptoms on a range of lupin species can be found in Jones and McLean (1989).

Epidemiology

The virus is transmitted in a non-persistent manner by more than 20 species of aphids (Jones and McLean, 1989; Jones, 1991). However, in Australia the major vectors were reported by Garnett and McLean (1983) as *Aphis craccivora* (Koch); *Aulacorthum solani* (Kaltenbach) and *Myzus persicae* (Sulzer). In *L. angustifolius*, very few infected plants survive to produce seed, so seed transmission is not seen as a major problem. However, Gondran *et al.* (1994) report up to 3% seed transmission in *L. albus* and Jones and McLean (1989) also report seed transmission in *L. pilosus*. Because there is no seed transmission in *L. angustifolius* and only limited transmission in *L. albus*, the rate of spread of the virus is a function of the

population of aphid vectors. With the exception of the American lupin aphid (*Macrosiphum albifrons* Essig), the virus has only limited chance of transmission in crops of bitter lupin.

The American lupin aphid *M. albifrons*, which is not killed by alkaloids common in some lupins, arrived in Europe in 1981 and spread rapidly from west to east. By 1990, it had reached the former East Germany, Czechoslovakia and Poland (Eppler and Hinz, 1987; Piron, 1987; Karl and Schmidt, 1990a; Müller *et al.*, 1990; Karl, 1991; Karl *et al.*, 1991a, b). By 1989, Schmidt and Karl (1989) were reporting its role in the transmission of BYMV. It had also been observed infesting *L. polyphyllus* (Karl and Schmidt, 1990a; Karl *et al.*, 1991b) and *L. albus*, *L. angustifolius*, *L. luteus* and *L. mutabilis* at a number of sites (Eppler and Hinz, 1987; Karl and Schmidt, 1990b; Karl *et al.*, 1991a). Bournville *et al.* (1991) tested the survival and reproduction of the aphid on a range of genotypes of *L. albus* and found virtually no resistance to the aphid in any of the cultivars and lines which they tested.

Crop Loss

The degree of crop loss is mainly associated with the population of transmitting aphids. In *L. luteus*, seed yield dropped 15–20% from late infected plants. However, early infection produced a total loss of seed production. In *L. cosentinii*, losses of seed yield of 50–90% have been reported. As the virus normally kills plants of *L. angustifolius*, no seed is produced. In an epidemic in the Great Southern Region of Western Australia in 1987, nearly 50% of plants were infected and there was a major reduction in both seed and forage yield. In some years in the same region there has been a 100% crop loss in the same species. Although *L. mutabilis* can be heavily infected by BYMV, there are no published results on yield reduction (Jones and McLean, 1989).

Control

Although Gladstones (1984) has looked for resistance to BYMV in *L. angustifolius*, no sources of resistance have been found to date. However Schmidt (1988) detected quantitative resistance to BYMV in *L. luteus* and Yakusheva (1996b) recently reported the release in Russia of two BYMV-resistant cultivars. In countries such as Australia, given the wide range of alternative hosts, if aphid vectors are present the virus will almost certainly spread to lupin crops. Jones (1991) investigated the effect of a reflective mulch on the rate of transmission in *L. angustifolius*. Jones (1993) investigated the effect of plant population of the lupins, growing cereal borders around the lupins or mixing the lupins with a cereal on the spread of virus infection in *L. angustifolius*. Subsequently Jones (1994) investigated the effect of mulching with cereal straw and of lupin population on the spread of the virus. In two out of three trials, the use of reflective polythene mulch reduced the rate and the extent of spread of BYMV from outside the trial area (Jones, 1991). Further, over a number of years the use of cereal borders

reduced the number of infected lupin plants by 43–60%. Sowing mixtures of lupins and a cereal also significantly reduced the number of infected plants by 76–96%. Infection rates with BYMV were also lower when the lupins were sown at high population. Jones (1993) suggested that the aphids were more attracted to plants surrounded by bare ground than to the crop canopy. His subsequent work with the use of a straw mulch supported this hypothesis. The presence of straw in a badly infected crop reduced infection by 70%. Plant population also had a significant effect on the rate of infection. In the absence of straw, plants sown in narrow rows had a 38% lower rate of infection than widely spaced plants. It appears that the best way to obtain some degree of control of the virus is to sow lupins at a high population into a straw mulch and to sow a cereal border around the lupin crop to reduce the rate at which the aphids colonize the lupin stand (Jones, 1993, 1994).

CUCUMBER MOSAIC

Aetiology and Biology

Cucumber mosaic is caused by cucumber mosaic virus (CMV) which is a member of the cucumovirus group and, like BYMV, is transmitted by aphids. It has a wide host range and infects plants from over 40 families. In Poland, CMV was considered to be the second most important virus of *L. luteus* (Frencel and Pospieszny, 1985). In South Australia, Wahyuni and Francki (1992) tested 16 strains of the virus against lupins and other legume species. They found that most of the current Australian commercial cultivars of *L. angustifolius* were susceptible, as were lentils and faba beans. More worrying however was the infection of commonly used southern Australian pasture legume species such as *Medicago truncatula*, *M. rugosa*, *M. littoralis*, *M. polymorpha*, *Trifolium subterraneum* and *T. resupinatum*. Given the extensive use of these species it would appear that aphid vectors would have adequate access to infected alternative hosts in most Australian lupin growing areas.

Jones (1988) was able to isolate the virus at one site in Western Australia from 15 species of weeds and other legumes including *Medicago murex*, *Melilotus indica*, *Ornithopus sativus*, *Trifolium arvense* and *Vicia sativa*. Virus symptoms were common in plants of *Trifolium subterraneum* at two sites (Jones, 1988). In more recent work, McKirdy and Jones (1994) tested a further range of possible alternative host species and confirmed the findings of Wahyuni and Francki (1992). They were also able to recover the virus from infected plants of the genera *Arctotheca*, *Cerastium*, *Melilotus*, *Misopates*, *Raphanus* and *Stachys*.

Symptoms

According to Jones and McLean (1989) the initial symptoms of aphid-transmitted CMV are not unlike those of BYMV. Further, unlike BYMV-infected plants, CMV-infected plants set some seed. Many seedlings arising from infected seed die

shortly after emergence or within 6–8 weeks of emergence. Plants that survive are stunted and have downcurled leaflets.

Epidemiology

CMV is the second most important viral disease of lupins. Jones and McLean (1989) suggest that up to 60 different aphid species can transmit the virus. However, McKirdy and Jones (1994) considered that in Western Australia, with its hot dry summers, weed species were unlikely to be a major source of lupin crop infection due to their lack of survival over the long summer drought. Thus the major initial cause of loss of lupins from the virus is from infected seed. Infected plants that survive in the crop then provide further foci of infection for the aphids to transmit the virus (Jones and McLean, 1989).

Unlike BYMV the degree of seed transmission in CMV is higher. Infected seedlings frequently die either shortly after emergence or may survive up to 6–8 weeks. Surviving plants are stunted but may set some seed under good growing conditions.

Crop Loss

Like BYMV, crop losses as the result of CMV can be high (Jones and McLean, 1989). Early infection can lead to virtually no seed production at all. In Western Australia, a crop of *L. angustifolius* which had been expected to yield 2.5 t ha^{-1} yielded less than 1.0 t ha^{-1}, a reduction of over 70%. In a late infected crop, yield reduction was up to 59%. In 1988 in the northern agricultural region of Western Australia where 104,000 ha were infected, yield losses were generally around 50% but in some cases crops were not worth harvesting.

Control

Considerable variation in resistance to CMV occurs in lupins both among and within species. In Western Australia, Jones and Latham (1994) were unable to detect CMV in seed of lines of *L. albus*, *L. atlanticus*, *L. cosentinii*, *L. digitatus* and *L. pilosus*. However, they were able to recover the virus from all lines of *L. angustifolius* and *L. mutabilis* and from most lines of *L. luteus* and *L. hispanicus*. The results indicated considerable potential for breeding CMV-resistant genotypes suited to Australian conditions. Cowling and Jones (1994) screened *L. angustifolius* breeding lines for resistance to CMV and discovered considerable variation in the degree of infection among breeding lines and cultivars ranging from moderately resistant (1–6% seed transmission) to very susceptible (35–75% transmission). Differences in seed transmission rates were highly significant and consistent from year to year. It was suggested that the resistance is under polygenic control. However, as yet no resistant lines have been released and other methods of control have to be used to limit the spread of the disease. Because the

virus is seedborne in up to 34% of seedlings (Jones, 1988), one method of control is to test seed before sowing. Only seed with a low level of virus infection should be sown. Initially seed testing was done using ELISA; however, recently Wylie *et al.* (1993) developed a polymerase chain reaction (PCR) test. The method was able to detect one infected seed in 200 and, because it was also able to detect one infected leaf in 1000, it may be suitable for use as a screen in routine plant breeding.

Jones (1988) found considerable differences in the level of infection among *L. angustifolius* cultivars. Reguing virus-infected plants of the new cultivar Gungurru reduced the level of infection in harvested seed to 0.1–0.2%. Highest levels of seed transmission occurred in crops from high rainfall areas. Jones (1988) was also able to show that if seed was graded on size, small seed had the highest level of virus infection and the level of infection varied as to when and where pods were formed. Pods from the main stem were 3% infected, those from first-order laterals 8%, and those from second and higher orders 13%. Bwye *et al.* (1994) suggested that the use of high populations helps to suppress the virus by the shading out of infected plants by healthy ones reducing their contribution to seed production. Further, sowing of lupins at high plant populations also limits production of higher order pods by suppressing lateral branching (Herbert and Hill, 1978). Jones and Proudlove (1991) investigated the effect of sowing seed that was 0.5 and 5% infected with CMV on the subsequent development of disease in the crop. Because of the death of infected seedlings, the final populations contained 0.2–0.3% and 1.5–2.9% infected plants. However, in plots sown to 5% infected seed, aphids rapidly spread the disease. Seed yield was reduced by 35% and the seed produced was 6–13% infected. However, aphid transmission had no effect on seed yield from plots sown to 0.5% infected seed. The aphids present in the trials were *Acyrthosiphon kondoi* (Shinji), *Aphis craccivora* (Koch), *Lipaphis erysimi* (Kaltenbach) and *Myzus persicae* (Sulzer). In glasshouse trials all of these aphid species were capable of transmitting the virus.

The timing of the infection of the plant with CMV is important for both plant survival and its ability to produce infected seed (Geering and Randles, 1994). When seedlings of *L. angustifolius* were inoculated 2 days after emergence, 45% died. Plants that were inoculated 58 days after emergence, while still in vegetative growth, produced less than 27% dry matter and 9% seed of healthy plants. Late inoculation at 114 days after emergence had no effect on dry matter yield but seed yield was reduced by 25%. The highest rate of virus transmission from seed – 24.5% – was from plants that were inoculated with the virus at the mid-vegetative growth stage (Geering and Randles, 1994). Besides sowing virus-tested seed there are other ways of limiting the spread of the infection. As with BYMV, the use of a reflective mulch also reduced the spread of infection of CMV from primary infection foci (Jones, 1991). Jones (1991) suggested that the method could be used by breeders to reduced the spread of infection in single row breeders plots. On a field scale, if virus-infected seed was sown at depths of 8 and 11 cm instead of the usual 5 cm the incidence of the disease in established seedlings was reduced by 15 and 50% (Jones and Proudlove, 1991). In a study on the effect of plant density and the proportion of infected seed sown, Bwye *et al.* (1994) found that virus spread was favoured by high seed infection levels, good

establishment of infected plants and the early arrival of aphids. In some seasons sowing seed that was only 0.5% infected could lead to seed yield reductions of 16–19%. Sowing at high plant populations, which leads to rapid canopy closure, reduced the number of seed infected plants that survived and the current season spread of the disease (Bwye *et al.*, 1994). Nutrient status also appears to be important. Work by Wahyuni and Randles (1993) suggests that the ability of CMV to infect *L. angustifolius* was reduced when plants had been inoculated with *Bradyrhizobium* prior to virus infection.

In summary, the overall recommendation for the management of CMV in lupin crops is to use seed with a low level of infection (< 0.5%), and to sow early at a high plant population to provide both good ground cover and rapid canopy closure (Bwye *et al.*, 1994)

CONCLUSIONS

As has been the case with crops like soyabean, one country has tended to become dominant in the commercial cultivation of lupins. At present the major diseases of lupins are therefore the major diseases that are present in the south-west of Western Australia. Given the large areas that are involved, and the relatively low value of lupin seed, it is unlikely that diseases in lupins merit control with fungicides. This being the case, it can be expected that the major thrust of disease control research in lupins will continue to be screening, selection and breeding of resistant cultivars. Australian plant breeders have done excellent work in this regard and their efforts are being enhanced by plant breeders in Chile, France, Germany and Poland. Given the ever-changing relationships between host plant and pathogen, their job looks as if it will continue to be a long one.

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Diseases of Clover

12

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INTRODUCTION

Clover species are attacked by a wide range of fungi, phytoplasmas, bacteria and viruses, and it would be impossible in a book of this size to cover all in detail. The seven most important fungal diseases – fusarium root rot, clover rot, scorch, powdery mildew, pepper spot, leaf spot and black blotch – are listed in order of citation in literature, followed by the phytoplasma disease, phyllody and a short section on bacterial diseases. Other fungal and phytoplasma diseases which are either of lesser importance or referred to less frequently are listed in Table 12.1.

Clover species are infected by a wide range of viruses. Johnstone and McLean (1987) recorded nine viruses on subterranean clover in Australia. Hampton *et al.* (1978), observed reactions by white and/or red clover to 17 out of 38 virus isolates tested from a number of locations in the USA, Europe and Japan. Carr (1984) stated that herbage legumes in Britain are infected by 15 viruses, although only six were considered important. A literature search of three databases ranging from 1975 to 1995 showed the following to be the most frequently quoted: clover yellow vein, bean yellow mosaic, alfalfa mosaic, white clover mosaic, red clover necrotic mosaic and soyabean dwarf (syn. subterranean clover red leaf). Other virus diseases that are quoted less often or are of minor importance are listed in Table 12.2. To add to this wide range of viruses is the further complication that clover is frequently infected by more than one virus (Alconero, 1983), some with at least four viruses (Helms *et al.*, 1993). This can obviously produce confusing symptoms. However, for the sake of clarity and simplicity, the most frequently quoted viruses are described individually.

It should also be borne in mind that in many pastures where clover is growing either as a pure sward or in a grass/clover mixture, not only may there may be more than one virus, but there may be combinations with fungal and/or bacterial pathogens.

 The Pathology of Food and Pasture Legumes
(eds D.J. Allen and J.M. Lenné)

Table 12.1. Less frequently recorded fungal, bacterial and phytoplasma diseases of clovers.

Disease (Refs.)	Pathogen	Host	Distribution	Importance
Fungal diseases				
Anthracnose (southern anthracnose in USA) (22,24)	*Colletotrichum trifolii* Bain & Essary	Red clover, white clover?	Europe, N America, Kenya, S Africa	Most records on alfalfa and lucerne
Black stem (spring black stem in N America) (9,22)	*Phoma medicaginis* Malbr. & Roum. in Roum. (syns. *P. herbarum* var. *medicaginis*, *Ascochyta imperfecta*, *A. trifolii*)	Berseem clover, red clover, Persian clover, subterranean clover, white clover	Europe, Russia, N America Australia	Common and destructive in N America; noted as important in Romania and Australia
Cercospora leaf spot (1,20,23)	*Cercospora zebrina* Passer.	Subterranean clover	Europe, N America, Australia, Japan	Moderately important in Australia and N America
Chocolate spot (24)	*Botrytis cinerea* Pers. ex Fr.	Alsike clover, white clover	Europe	Mild symptoms only
Downy mildew (8,12,22)	*Peronospora trifoliorum* de Bary	White clover, red clover	Europe, N America	Occasional on red clover, more common on white, but most common on lucerne
Rusts (1,2,8,11,21,22,26)	*Uromyces trifolii-repentis* Liro ex Liro (syns. *U. trifolii* var. *trifolii-repentis*, *U. hybridi*, *U. trifolii-hybridi*); *Uromyces trifolii-repentis* Liro ex Liro var. *fallens* (Arth.) Cummins (syns. *U. fallens*, *U. trifolii*)	Alsike clover, crimson clover, Persian clover, red clover, subterranean clover, white clover	Europe, Russia, N America, S Africa, Australia, New Zealand, Japan	Occasionally damaging; some evidence for reproductive disorders in grazing cattle, losses of herbage and seed yield
	Uromyces trifolii (R. Hedw.) Fuckel (syns. *Nigredo trifolii*, *Uromyces nerviphilus*)	White clover	Europe, N America	Serious damage in glasshouse material; less damaging in field plots
	Uromyces minor Schroet.	Yellow suckling clover	Europe, N America	Uncommon, but occasionally severe
Stagonospora leaf spots (20,24)	*Stagonospora meliloti* (Lasch.) Petr. (syn. *S. compta*); *Stagonospora recedens* (Massal.) Jones & Weimer	Red clover, white clover, subterranean clover	Europe, N America	Occasional
Stemphylium leaf spots (ring spots; target spots) (3,14,20,24)	*Stemphylium sarcinaeforme* (Cav.) Wiltsh.; *S. botryosum* (Wallr.)	Red clover	Worldwide	Common; serious in Canada

Verticillium wilt (7,13,18,22)	*Verticillium albo-atrum* Reinke & Berth.; *Verticillium dahliae* Kleb.	Alsike clover, crimson clover, red clover, white clover,	Europe, N America, New Zealand	More common on lucerne
Violet root rot (16,24)	*Helicobasidium purpureum* (Tul.) Pat. (syns. *Rhizoctonia crocorum* and *R. violacea*)	Red clover	Europe, N America	Occasional
Root rots (24,25)	*Thielaviopsis basicola* (B. & Br.) Ferraris; *Cylindrocarpon ehrenbergi* Wollenw.	Red clover, sweet clover	Europe, New Zealand	Damaging in New Zealand
Bacterial diseases				
Bacterial root rot (4,28)	*Pseudomonas* sp. (=*P. radiciperda* (Zhavorononokova) Savulescu	Red clover	Russia, Italy?	Locally important?
Root growth inhibition (5)	*Pseudomonas* spp. (fluorescent)	White clover	New Zealand	Some root growth restriction
Leaf spot (4,15)	*Pseudomonas syringae* pv. *syringae* van Hall (=*Bacterium trifoliorum* Jones *et al.*)	Red clover	USA	Not known
Leaf spot/black spot (6,19,20)	*Pseudomonas andropogonis* (Smith) Stapp.	White clover	USA	Prevalent in N Carolina
Phytoplasma diseases				
Clover club leaf (17,24)	Rickettsia	Crimson clover, white clover	Europe, N America	Occasional
Clover red leaf (English stolbur) (8,10)	PLO	Red clover, white clover	Europe, USA	Occasional
Clover witches' broom (8)	PLO	White clover	Europe	Unimportant

References: 1 = Barbetti (1989); 2 = Barbetti and Nichols (1991); 3 = Booth (1980); 4 = Bradbury (1986); 5 = Brown *et al.* (1994); 6 = Caruso (1984); 7 = Celetti *et al.* (1990); 8 = Carr (1984); 9 = Christensen *et al.* (1994); 10 = Dale and Cheyne (1993); 11 = Farr *et al.* (1989); 12 = Francis (1983); 13 = Hay and Skipp (1993); 14 = Higgins and Lazarovits (1978); 15 = Jones (1923); 16 = Mackinaite and Strukcinskas (1992); 17 = Markham *et al.* (1975); 18 = Milton and Isaac (1976); 19 = Nelson and Campbell (1991b); 20 = Nelson and Campbell (1992); 21 = O'Donovan and Fothergill (1992); 22 = O'Rourke (1976); 23 = Pratt and Ocumpaugh (1994); 24 = Sampson and Western (1941); 25 = Subramanian (1968); 26 = Walker (1978); 27 = Windsor and Black (1973); 28 = Javoronkova (1932).

Table 12.2. Less frequently reported virus diseases of clovers.

Disease/virus (Refs)	Host	Symptoms	Distribution	Importance
Arabis mosaic (3)	White clover	Mild chlorotic mottling, symptomless	Europe	Low incidence
Bean yellow severe mosaic (4)	Red clover, white clover	Local lesions, necrosis in red clover, latent in white	Northern temperate regions	Low incidence?
Clover blotch (4)	Red clover	Mottle, mosaic	Europe	Low incidence?
Clover mild mottle (1)	Red clover	Mottle, mosaic	Europe, Russia	Reduction of 95% in fresh weight in controlled conditions in Czechoslovakia
Clover mild mosaic (2)	Red clover, alsike clover	Mosaic, leaf deformation	USA, Sweden	Occasional
Clover yellow mosaic (7)	Alsike clover, white clover	Yellow/light green leaf stripping, stunting, distortion	Russia, N America, Europe	Occasional
Cucumber mosaic (6,8,9)	Subterranean clover	Mild mottle, leaf downcurling and distortion	Worldwide, especially temperate regions	Dry yield reduced by 17–24% in Australia
Pea enation mosaic (6,12)	Crimson clover, subterranean clover	Mosaic, translucent spots, small necrotic flecks, enations	Northern temperate regions, Australia	Common on subterranean clover, but main importance as means of transmission to peas
Peanut (groundnut) stunt (10)	Arrowleaf clover, alsike clover, crimson clover, kura clover, red clover, subterranean clover, white clover	Systemic mottle, leaf necrosis	USA, Japan, Hungary	A possible major source for lack of persistence of white clover in USA
Red clover vein mosaic (11)	Alpestrine clover, alsike clover, red clover, white clover, yellow suckling clover, zigzag clover	Vein yellowing and clearing, mild interveinal mottling	Europe, Russia, USA, S Africa	Common, but effects on yield not certain

Strawberry latent ringspot (3)	White clover	Symptoms indistinct	Europe, USA	Not very important
Subterranean clover mottle (6,13)	Subterranean clover	Leaf mottle, deformation, dwarfing	Australia	Common, but at low incidence
Subterranean clover stunt (13)	Subterranean clover	Yellowing, vein marginal chlorosis, distortion, small leaves	USA, Australia	Cause of large losses in legume-based herbage production in Australia
Sweet clover necrotic (5)	Sweet clover	Systemic mosaic, ringspot, veinal necrosis	Canada	Widespread
Tomato black ring (3)	White clover	Symptomless	UK, USA	Very low incidence

References: 1 = Gerhardson and Lindsten (1973); 2 = Gerhardson (1977); 3 = Gibbs *et al.* (1966); 4 = Hampton *et al.* (1978); 5 = Hiruki *et al.* (1984); 6 = Johnstone and McClean (1987); 7 = Jones *et al.* (1981); 8 = Jones and McKirdy (1990); 9 = Jones (1991); 10 = McLaughlin (1986); 11 = McLaughlin *et al.*(1992); 12 = McWhorter and Cooke (1958); 13 = Wroth and Jones (1992).

For the purposes of this chapter, clover is defined as any plant of the genus *Trifolium* commonly used for grazing or conservation.

FUSARIUM ROOT ROT COMPLEX

Aetiology

Although *Fusarium oxysporum* Schlect. alone has been shown to be capable of producing a wilt of crimson clover (*Trifolium incarnatum*) (Pratt, 1982), *Fusarium* spp. are more usually associated with other *Fusarium* spp. or with other fungi in what is commonly known as fusarium root rot complex. A relatively large number of *Fusarium* spp. are thought to be involved, including *F. acuminatum* Ellis & Everhart (*F. roseum*), *F. avenaceum* (Fr.) Sacc., *F. culmorum* (Smith) Sacc., *F. lateritium* (Fr.) Nees., *F. moniliforme* Sheld., *F. nivale* (Fr.) Ces., *F. solani* (Mart.) Appel & Wallr., *F. sambucinum* Fuckel, *F. semitectum* Berk. & Ravenel, *F. sporotrichioides* Sherb. as well as *F. oxysporum* (O'Rourke, 1976; Mezentseva, 1989). Other fungi include weakly pathogenic *Rhizoctonia* and *Pythium* spp. Normally saprophytic *Alternaria*, *Penicillium*, *Aspergillus*, *Mucor* and *Trichoderma* spp. are also found associated with the complex (Leath *et al.*, 1971). In Australia, *Aphanomyces euteiches* Drechs. is also an important component in root rots (Greenhalgh *et al.*, 1986). In Britain, although no vascular wilts of herbage legumes have been attributed to *Fusarium* spp., a range of fungi, similar to those described, can be isolated from the roots of unhealthy looking plants in the field (Carr, 1971). In the USA, Jin *et al.* (1992) have indicated that decline of red clover (*Trifolium pratense*) is the result of an interaction between *Fusarium avenaceum* and the beetle *Hylastinus obscurus* Marsham.

Biology

Hyphae of components of the complex and particularly of *Fusarium* spp. are often found within root tissues from only a few days after germination and remain for the lifetime of the host plant (O'Rourke, 1976). Although damping-off is occasionally caused by *Fusarium* spp. (Carr, 1984), *Pythium* spp. are more commonly associated with this disease. In the early stages of plant growth *Fusarium* spp. often appear to cause little damage. However, within 2–3 years of sowing, the level of *Fusarium* spp. often builds up markedly at the expense of the normally non-pathogenic fungi. Fungi in the complex are usually transmitted via the soil but it is also probable that some at least can be seed-transmitted. Isolates of *F. oxysporum*, *F. culmorum* and *F. avenaceum* pathogenic to linseed have been found on linseed seed in Britain (H.C. McGimpsey, Belfast, Northern Ireland, 1996, personal communication).

Fusarium root rot is a common and important problem in many parts of the world where clover is grown. In the USA and Canada, it has been recorded as a serious problem for over 50 years (O'Rourke, 1976). Wanson and Maraite (1984) stated that it was the main reason for the decline of red clover in Belgium,

citing *F. avenaceum* as the most virulent pathogen. Similarly in the Czech Republic (Nedelnik, 1993) *F. avenaceum* was predominant on red clover followed by *F. culmorum*, *F. oxysporum* and *F. solani*. Burgess *et al.* (1973), in Australia, also cited *F. avenaceum* first in their list of isolates from rotted roots of subterranean clover (*Trifolium subterraneum*). On the other hand, Mills (1985) found *F. solani* to be most common in almost all clover fields inspected in the Cape area of South Africa in 1979. In New Zealand, *F. oxysporum* was the predominant *Fusarium* spp. isolated from roots of white clover (*Trifolium repens*) (Skipp and Christensen, 1983), while *F. avenaceum* and *F. culmorum* were not common and never predominated at any particular site. Mohamed *et al.* (1980b), in Egypt, also found *F. oxysporum* to be one of the most commonly isolated of clover fungi. Root rots caused by *Fusarium* spp. are also reviewed on soyabean (see Sinclair, Chapter 3, this volume), pea (see Kraft *et al.*, Chapter 6, this volume) and chickpea (see Haware, Chapter 9, this volume).

Although many of the *Fusarium* spp. associated with root rot are known to attack a wide range of plants, some host specificity has been found. *F. solani*, *F. oxysporum* and *F. culmorum* isolated from red clover were pathogenic on a range of legumes, but not capable of infecting non-legumes (Chi, 1965). On the other hand, Vargo and Baumer (1986), in the USA, found that isolates of *F. sporotrichioides*, from the roots of white clover, were also pathogenic on spring wheat.

Although Pratt (1982) noted differences in the ability of various isolates of *Fusarium oxysporum* to attack crimson clover there is limited literature to support particular races of *Fusarium* spp. being specific to individual clover cultivars. On the other hand, there is clear evidence of differences between individual *Fusarium* spp. in their ability to attack clover, when inoculated under controlled conditions (Stutz and Leath, 1983).

Symptoms

Affected crop stands often contain bare patches where plants have died out (Plate 27). The remaining plants may also thrive poorly (H.C. McGimpsey, Belfast, Northern Ireland, 1996, personal communication). Examination of individual plants shows necrosis of the tap and lateral roots. This may occur from 5 months after seeding (Wanson and Maraite, 1984). In severe attacks, the crop may become totally unproductive within as little as 2–3 years (O'Rourke, 1976; Skipp and Christensen, 1990). McMurchy and Higgins (1984) have correlated increases in severity of root rot with decline in the pterocarpenoid glycoside, trifolirhizin, and increase in the aglycone, maackiain.

Epidemiology

F. oxysporum, *F. avenaceum* and *F. solani* are capable of penetrating unwounded epidermal cells directly (Chi *et al.*, 1964; Stutz and Leath, 1983; Kovácikóvá and Kudela, 1984). However, many of these tests have been carried out under aseptic conditions and may not reflect the practical situation in the field. Indeed,

Greenhalgh and Taylor (1985) have questioned the ability of *F. avenaceum* to cause root rot under normal field conditions, by pointing out that, although it could cause rot in pasteurized soil, it could not do so in unpasteurized soil. Further tests are needed in both strictly controlled and field conditions to clarify these contradictions.

However, most workers consider that the *Fusarium* spp. associated with clover are only weakly pathogenic and that predisposing stress factors are required for root rot to develop. Such factors may include too-frequent cutting, drought, cold, mineral deficiencies (particularly potash), insect, nematode or other damage to the roots (Carr, 1984; Stutz *et al.*, 1985; Jin *et al.* 1992), or infection by foliar pathogens, such as *Ascochyta imperfecta* (O'Rourke and Millar, 1966). The interaction with nematodes appears to be complex. Nordmeyer and Sikora (1983) noted that simultaneous inoculation of *Heterodera daverti* Woutts, *F. solani* and *F. oxysporum* did not reduce the yield of two clones of subterranean clover. However, inoculation of the nematode, 1–2 weeks after the fungi, reduced plant weight synergistically.

Effects on Yield and Quality

Because of the complex nature of the disease, it is difficult to reproduce exactly under controlled conditions. Use of a fungicide drench or microwave treatment of the soil in pots of red clover naturally infected with a root rot complex (Nan *et al.*, 1991) decreased fungal populations but surprisingly also decreased emergence and growth. However, later work in plots (Nan *et al.*, 1992) showed increases in yield following fungicide drenches. Kovácikóvá and Kudela (1984) found an increase in plant weight at the first cut after inoculation with a range of *Fusarium* spp., although there was a decrease at the second cut. In spite of these contradictory experimental findings, it is clear that yields of farmers' pastures can be considerably reduced by root rots because of decline in mature stands (O'Rourke, 1976). Indeed, decline may reach such a stage that the ploughing up of the pasture may be needed.

Disease Control

Carr (1984) stated that no ready solution to the problem of fusarium rot had been found in the UK other than management to reduce stress. Similarly, Oliva *et al.* (1994) in the USA, showed that relieving of water stress by irrigation reduced the incidence of the fusarium root rot complex in red clover. In the former USSR, Khar'kov and Kashmanova (1973) claimed markedly decreased infection with *F. oxysporum* through the use of ammonium molybdate seed treatment; Karvyanskii and Mazur (1978) also claimed control, but with the use of thiram seed treatment. In Egypt, Mohamed *et al.* (1980b) obtained best control with the use of benomyl seed treatment, while Pung *et al.* (1991) in New Zealand, found no reduction in *F. oxysporum* in roots of subterranean clover with benomyl. However, Nan *et al.* (1992), also in New Zealand, obtained good control of a range of root-infecting fungi, including *F. oxysporum* and *F. solani*, using a soil drench of prochloraz, although this is unlikely to be economically viable. Barbetti

and MacNish (1983), in Australia, stated that fungicidal control on subterranean clover was ineffective, but observed that the level of root rot could be reduced by techniques such as fallowing or rotation with oats (Barbetti and MacNish, 1984). However, later work (Barbetti, 1991) indicated that the remission of disease through fallowing was only effective for one season.

A degree of resistance to the root rot complex has been observed in some cultivars (Barbetti *et al.*, 1986; Nagovitsyna and Gracheva, 1990; Pratt and Rowe, 1993; Andersson, 1994; Coulman and Lambert, 1995). Paplauskiene (1992) has indicated a close correlation between the ability to produce phenolic compounds and resistance to *Fusarium* sp. in red clover.

CLOVER ROT

Aetiology

The existence of a distinct variety, var. *fabae*, of *Sclerotinia trifoliorum* Erikss. was postulated by Keay (1939) (Loveless, 1951). However, subsequent work by Wong and Willetts (1975) and Jellis *et al.* (1984) has shown that *S. trifoliorum* var. *fabae* is in fact an isolate of *S. trifoliorum*. This situation has been thoroughly reviewed by Scott (1984) who opted for the single name *S. trifoliorum*, but added that this designation should be treated with great circumspection. The fungus is a member of *Helotiales* in the *Discomycetes*.

Biology

Sclerotia are produced around the crown of dead and dying plants in early spring. The dark, irregular sclerotia range in size from 2 to 20 mm (average 5 mm) and have a convoluted surface. They remain dormant in the ground until the following autumn when they germinate to produce buff-coloured apothecia consisting of a disc *c.* 4 mm across on the top of a slender stalk which may be up to 8 mm tall (Fig. 12.1). Sclerotia can produce a second or even a third crop of apothecia in successive years. Germination is sometimes delayed for a number of years (Keay, 1939). Infection by ascospores takes place in the autumn, leading to damage and death of leaves and sometimes the whole plant. Ascospores are hyaline and elliptical (12–18 × 5–8 μm) (O'Rourke, 1976) and are produced from asci, 170–190 × 10–15 μm. Although it was originally supposed that *S. trifoliorum* was homothallic (Carr, 1954) and produced apparently functionless microconidia, more recent work (Uhm and Fuji, 1983) has indicated that the microconidia are in fact functional spermetia and the fungus is indeed heterothallic. One pathogenic determinant of *S. trifoliorum* is its production of oxalic acid, although research by Callahan and Rowe (1991) has indicated that there are almost certainly other factors.

Clover rot has generally been regarded as the most serious disease of red clover in Europe, particularly in short rotations (Carr, 1984). Importance and distribution can, however, be irregular. For example, a survey of incidence in

Fig. 12.1. Apothecium and sclerotium of *Sclerotinia trifoliorum* (Photo: courtesy of P.C. Mercer).

Bohemia and Morovia from 1900 to 1982 (Kudela and Kováciková, 1987) showed peak incidence from 1912 to 1928, with only local and sporadic occurrences afterwards. Also, in Ireland, where red clover is not extensively grown, surveys (Mercer, 1981) showed very low general incidence, but considerable damage where the disease did occur. The disease was noted in Britain as early as 1849 (Lawes and Gilbert, 1860) and again by Carruthers (1898) who stated that in some districts the crop had been almost completely destroyed. Although crimson, alsike (*Trifolium hybridum*) and white clovers could also be infected, damage was generally not as serious as on red clover. However, in recent years in Britain the pattern of damage on white clover appears to have changed. Dixon (1975) noted a number of reports of serious damage on white clover cultivars while Carr (1984) observed that more serious damage could occur on white clover cultivars than on red. In the USA, a large-leaved white clover variety, Ladino, was noted as particularly susceptible as early as 1949 (Kreitlow, 1949). *S. trifoliorum* has also been recorded on lucerne, trefoil, sainfoin (*Onobrychis viciifolia*), crown vetch (*Coronilla varia*), *Phaseolus* and faba bean (see Jellis *et al.*, Chapter 7, this volume) and a wide range of leguminous and non-leguminous weed species (Dillon Weston *et al.*, 1946; O'Rourke, 1976).

More research appears to have examined variation in host resistance to *S. trifoliorum* (Raynal, 1981) rather than variation in the pathogen. For example, Pratt (1992) in a comparison of isolates of *S. trifoliorum* from crimson, berseem (*Trifolium alexandrium*) and small hop (*Trifolium dubium*) clovers in the USA, showed significant and strong specialization between hosts. However, the under-

standing of variation of the pathogen on a single host appears confused. Thus Kreitlow (1949) showed differences between two isolates from white clover reinoculated onto white clover, suggesting physiologic specialization and Held (1955) showed differences in the production of pectinase enzymes between 'normal' and 'degenerate' strains of *S. trifoliorum* which were correlated with the production of a substance which caused wilting of clover leaves. However, Carr (1954) stated that although there was variation in pathogenicity among two isolates from red clover and one each from bean and sainfoin, there was no evidence for biologic races. Furthermore, Scott and Fielding (1985), in a similar experiment but with isolates from two cultivars each of lucerne and red and white clovers, showed differences dependent both on the origin of the isolates and on the plants inoculated. These differences were, however, only marked between different host species.

Symptoms

The first signs of clover rot appear in late autumn. Foliage becomes finely spotted due to infection by ascospores. This condition may persist for up to 12 weeks and it has been suggested (Valleau *et al.*, 1933) that it is only after the leaves die from natural causes that the fungus can advance further. Once this happens the remainder of the plant will then frequently also become severely affected and plant death may occur, often with visible growth of white mycelium, especially if conditions are warm and wet (Plate 28). Conversely, if conditions are dry and frosty, advance of the disease is severely restricted and plants may resprout from the crown.

Epidemiology

Ascospores are produced in abundance by *S. trifoliorum*. J.P. Malone and H.C. McGimpsey (unpublished) have shown a higher percentage spore germination in the presence of clover leaf leachate compared to that in pure water. Nevertheless, because of problems of inducing infection artificially with ascospores (Keay, 1939), doubts have arisen as to whether natural infections can occur unless the host is already weakened or senescent (Butler and Jones, 1949). Raynal (1981) and P.C. Mercer and H.C. McGimpsey (unpublished) produced symptoms in plants grown under conditions of high humidity. Raynal (1981) found mycelial plugs to be more successful than ascospores. P.C. Mercer and H.C. McGimpsey (unpublished) were not able to repeat the process under field conditions. In spite of these findings, however, it still seems likely that ascospores are important in disease spread, but that the precise conditions for infection, like those for much of the life cycle of the pathogen, are difficult to mimic artificially. Ascospores are forcibly shot into the air, possibly up to a height of 6.5 cm (Nilsson-Leissner and Sylven, 1929) and can then be carried to neighbouring plants by wind currents.

Although infection by ascospores is believed to be the main method of infection in Britain (O'Rourke, 1976), it is thought that in Scandinavia mycelial transfer is much more important (Fransden, 1946). Pape (1937) noted fastest

mycelial growth for most isolates of *S. trifoliorum* at 16.5°C, although two isolates had an optimum of 19°C. J.P. Malone and H.C. McGimpsey (unpublished) noted maximum mycelial growth between 15°C and 16.5°C. Mycelium may come from an extension of that present on crop debris or it may grow from sclerotia. Although Keay (1939) was unable to induce mycelial growth from sclerotia, Williams and Western, (1965a) observed the production of secondary sclerotia via a mycelial 'neck'. The authors also indicated that sclerotia, buried in soil, were capable of an increase in weight of up to 50%, either by absorption or by a closely adhering mycelial system.

Another possibility for disease spread is seed, either as mycelium on seed or as sclerotia mixed with seed. Kietreiber (1979) reported 3% of clover seed samples in Austria containing sclerotia of *Sclerotinia* spp. However, although J.P. Malone and H.C. McGimpsey (unpublished) examined 10,000 seeds from samples which subsequently gave rise to crops infected with *S. trifoliorum*, they were unable to find any sclerotia. They also made the point, as did Williams and Western (1965b), that sclerotia the size of clover seed were only likely to produce a relatively small number of apothecia. However, Scott and Evans (1984), examining the seed of apparently healthy plants from an area in Wales which had had a severe attack of clover rot, found 160 seed-sized sclerotia in 400 g of seed and came to the conclusion that this was indeed an important means of spread. There is also evidence for direct transmission of the fungus via mycelium on seed. Leach (1958), in the USA, found viable mycelium of what is now considered to be *S. trifoliorum* on seed of crimson clover. He also noted that the mycelium lost viability over a 2-year storage period. Scott (1981), in Wales, also found mycelium of a *Sclerotinia* sp. on seed of both white and red clover. Although the mycelium was non-viable, the seed had been stored for 2 years at room temperature.

A further possibility for transmission is by slugs. Shakeel and Mowat (1992) showed that transfer of excreta from slugs which had been feeding on sclerotia or apothecia of *S. trifoliorum* could be used to infect healthy white clover plants.

Under artificial conditions, sclerotial production occurs when a culture reaches the edge of a Petri dish or a barrier, such as a cut in the agar. Richer media and light tend to encourage higher numbers of sclerotia, but numbers are not affected by the wavelength of light (J.P. Malone and H.C. McGimpsey, unpublished). It is not clear what the trigger for sclerotial production is under natural conditions, but it does tend to occur as the host plant dies.

Although sclerotia can remain viable in the soil for at least 7½ years (Pape, 1937) their survival is dependent on soil moisture and temperature. Sclerotia which are close to the surface or buried in a shallow layer of soil dry out quite quickly and viability decreases sharply over just a few months. Similarly, J.P. Malone and H.C. McGimpsey (unpublished) noted a reduction in sclerotial germination after only 6 days at 30°C. It is therefore very doubtful if sclerotia can remain viable in the temperate regions for longer than 3 years under warm, dry conditions unless under clover (Williams and Western, 1965b). Increasing soil moisture up to 30% moisture-holding capacity always accelerated the degeneration of sclerotia. Above that level, the rate of degeneration was offset by the production of secondary sclerotia.

In temperate regions, apothecia are produced from sclerotia in the autumn,

probably triggered by the drop in temperature. McGimpsey and Malone (1979) produced maximum numbers of apothecia artificially by placing sclerotia (formed at 20°C) at −18°C for 48 h prior to incubation at alternating temperatures of 2°C and 8°C. Raynal and Picard (1985) also produced apothecia artificially by keeping sclerotia at 30°C for a month and then dropping the temperature to 15°C. Light, high humidity and depth of sclerotia in soil also appear to be important. McGimpsey and Malone (1979) found that sclerotia grown in the dark produced only stipes, confirming earlier work by Björling (1952). On the other hand, Hawthorne (1975) found that light inhibited stipe formation in the closely related *Sclerotinia minor* Jaggo. H.C. McGimpsey and J.P. Malone (unpublished) found that apothecial production in growth cabinets at 10°C was much greater when humidity was 60–70% than 30–35%. Williams and Western (1965a) found production of apothecia down to a depth of 5 cm. At 15 cm depth, stipes up to 6 cm long were produced, but no disc. Numbers of apothecia also tend to be higher in the presence of susceptible clover varieties (McGimpsey and Mercer, 1984). Growth of *S. trifoliorum* and apothecial production are also dependent on other microorganisms. J.P. Malone and H.C. McGimpsey (unpublished) found a higher number of apothecia produced in unsterilized rather than sterilized soil, whereas Dorenda (1984) showed inhibition of *S. trifoliorum* by saprophytic fungi from soil, rhizosphere, planosphere and roots of clover. Pfeffer and Luth (1990) reported an increase of 22% in the incidence of the mycoparasitic fungus *Coniothyrium minitans* Campb. on sclerotia of *S. trifoliorum* and an 88% decrease in apothecial production from fields under red clover monoculture compared with fields under crop rotation. Pratt and Knight (1982), in the USA, found that apothecia were much more readily produced under field conditions from sclerotia originally collected from the field rather than from ones produced artificially, thereby underlining the earlier comment about difficulties in mimicking the exact details of the life cycle.

Effects on Yield and Quality

Damage is most severe on red clover and the disease was cited as one of the main factors checking growth of the crop in Britain (Dillon Weston *et al.*, 1946). Surveys in the UK carried out in the late 1950s showed 70% of red clover fields to be infected, with losses of over 24% in 10% of crops (Lester and Large, 1958; Carr, 1984). If the plant stand is severely decimated, the only remedy is to plough the pasture in. Although white clover is not generally so badly affected, it may die out with time as the disease progresses (Scott, 1984).

Disease Control

Although clover rot can be reduced by the use of fungicides, such as quintozene (Sundheim, 1971); benomyl (Jenkyn, 1975; McGimpsey and Mercer, 1984); propiconazole (McGimpsey and Mercer, 1984); and vinclozolin (Sauer, 1984), this is not likely to be an economic option unless possibly for high-value seed

crops. The general recommendation for control is crop rotation of 4–5 years (Carr, 1984). However, Pfeffer and Luth (1990) suggested that because of the natural biological control of the pathogen in red clover monoculture, it was not considered worth while to select for resistance to *S. trifoliorum* under monoculture conditions. Pratt (1991) in the USA, noted that cutting crimson clover in November versus at other times of the year resulted in a much lower incidence of *S. trifoliorum*. As already observed, white clover tends to be more resistant than red clover, although it can still be badly affected. Lehman *et al.* (1991) suggested that resistance in the Irish white clover variety Aran was due to a high content of cyanogenic glycosides, which could have detrimental effects on cattle and sheep growth. Newer, tetraploid red clover varieties are reported to show good resistance (Raynal, 1986; Nüsch, 1989), although Vanco (1991) found selection of diploid cultivars to be more effective.

SCORCH

Aetiology

Scorch disease of clovers is known as northern anthracnose in the USA. It is caused by the fungus *Kabatiella caulivora* (Kirchn.) Karak. and is classified as a *Hyphomycete*. Although Cooke (1962) transferred the genus *Kabatiella* to the genus *Aureobasidium* and it is quoted as such in Farr *et al.* (1989), it has remained common practice to refer to the pathogen of clover scorch as *Kabatiella*. The best known synonyms are *Aureobasidium caulivorum* (Kirchn.) Cooke and *Gloeosporium caulivorum* Kirchn.

Biology

It is thought that the pathogen survives the winter on infected plants and decayed leaves, although it can also survive on seed for up to 2 years (Leach, 1962). The disease is not normally transmitted through the soil (Sampson and Western, 1941). Sporulation of conidia (8–24 × 2.5–3.5 μm) begins in the spring, and these are spread, mainly by rain-splash, to infect neighbouring plants and young seedlings. An ascigerous perfect state was observed in culture in the USA by Grinchenko and Colotelo (1963), although they did not indicate its taxonomy. However, no perfect state has yet been found in the field.

This is one of the most important diseases affecting red clover in temperate regions, occurring in Europe, North America, Asia (O'Rourke, 1976) and Australia (Helms (1975). It was described as the most serious disease of red clover in Wales by Stapledon *et al.* (1922). Scorch also occurs on crimson, subterranean and berseem clovers, but white and alsike clovers and lucerne are all fairly resistant (O'Rourke, 1976).

In Australia, Helms (1975) found a number of physiologic races of *K. caulivora*, and Chandrashekar and Halloran (1992) noted significant differences in symptoms produced by six isolates of *K. caulivora* on six cultivars of

subterranean clover, with one cultivar, Daliak, being completely immune at the seedling stage, and highly resistant at the adult stage. On the other hand, Chatel and Francis (1974) found no significant differences between two isolates from widely separated areas, when used on a range of cultivars. Ignatavichyute and Treigene (1988), in Lithuania, noted differences in virulence to three cultivars of red clover among 13 isolates of *K. caulivora*.

Symptoms

Initial symptoms, on stems and petioles, are dark brown, sunken lesions, surrounded by dark margins; smaller round spots develop on the leaves. Expanding lesions lead to girdling and cracking of stem tissues. Stems may curve over at the end to produce the crozier shape, characteristic of wilt diseases (Plate 29) (O'Rourke, 1976). The effects of the disease may be particularly serious if the crop is being grown for seed and if flower stalks are girdled. Leaves which have been infected wilt and dry up, although they may remain attached to the plant. A severe attack of *K. caulivora* produces a blackened, scorched appearance in the crop, hence the common name.

Epidemiology

Scorch is encouraged by high humidity and tends to be more common in spring and early summer before the first cut for hay. Gaurilcikiene (1992) noted that red clover plants artificially inoculated with *K. caulivora* were most susceptible at tillering and budding stages especially between 17 and 23°C. Although Barbetti *et al.* (1991) showed a strong plant-age × cultivar interaction in subterranean clover in Australia, there is some confusion on the effect of tissue age. Helms (1975) reported that leaf tissue of subterranean clover rapidly becomes more resistant to *K. caulivora* with time while Chandrashekar and Halloran (1990) stated that disease resistance in a range of cultivars decreases with time. Dense swards, which lead to high humidity and low light density, also tend to favour scorch. As spread of the pathogen is by rain-splash, periods of showery weather will also encourage disease build-up. Barbetti (1985a), in Australia, noted interrelationships between *K. caulivora* and *Cercospora zebrina* Pass., disease appearing later on the petioles if *C. zebrina* were established first. Conversely, levels of *C. zebrina* tended to be lower when *K. caulivora* were present.

Effects on Yield and Quality

Because of the girdling effect of the pathogen, the disease can be potentially very serious. In Canada, 50% losses in herbage yield of red clover have been recorded (Creelman, 1967) while, in Australia, Dear *et al.* (1987) described *K. caulivora* as a limiting factor in the growth of subterranean clover and Walker (1956) noted losses of up to 90% in herbage yields. Johnstone and Barbetti (1987) reported

K. caulivora to be one of the two fungal pathogens chiefly responsible for loss of productivity. Seed yield and crop quality may also be severely reduced.

Disease Control

In Australia, Bokor *et al.* (1978) and Little and Beale (1988) found *K. caulivora* could be successfully controlled using benomyl sprays, particularly when applied in early spring. This approach was confirmed by Helms and Andruska (1981) who showed, under controlled environmental conditions, that the level of scorch on subterranean clover was much lower when benomyl was applied before inoculation of the pathogen – only 36% control was obtained when plants were sprayed 8 days after inoculation compared with 100% control when plants were sprayed 3 days before inoculation. However, it is not clear whether such control is economically viable, particularly for hay crops. On the other hand, treatment of seed with fungicide can reduce the level of seedborne disease (Carr, 1984) and this is more likely to produce a profitable return. Resistant cultivars can play a part in disease reduction. 'Marathon' red clover is reported to have good resistance to *K. caulivora* in the USA (Smith, 1994) and Chatel and Francis (1974) showed large differences in susceptibility among five cultivars of subterranean clover tested in Western Australia. Generally, tetraploid and late-flowering cultivars of red clover are more resistant than diploid and early-flowering ones (Stapledon *et al.*, 1922; O'Rourke, 1976). Craig (1989), in Western Australia, found high resistance to *K. caulivora* and good recovery from grazing and cutting with the Persian clover (*Trifolium resupinatum*) cv. Kyambro.

Rotation can also be used as a control measure, but a break of several years may be required, at least in red clover, to prevent immediate recurrence (O'Rourke, 1976). The use of phosphate and potash fertilizers in the USSR has also been shown to reduce the percentage of red clover plants showing scorch disease (Khar'kov and Kashmanova, 1970).

POWDERY MILDEW

Aetiology

Powdery mildew is caused by *Erysiphe polygoni* DC. (syn. *Erysiphe trifolii* Grev.) and is a member of the *Erysiphales* and classified in family *Pyrenomycetes*. *E. polygoni* can be regarded as a collective species (O'Rourke, 1976), as it has a very wide host range, being recorded on at least 582 species including 212 legumes (Stavely and Hanson, 1966a). Kapoor (1967) suggested a division of the species into two – *E. trifolii* and *E. pisi* – on the basis of host range and cleistothecial morphology. Sivanesan (1976) has suggested that the individual 'species' making up the collective *E. polygoni* are in fact *formae speciales*.

Biology

The disease generally appears from the middle of the growing season onwards (O'Rourke, 1976). Conidia (25–40 × 16–22 μm) land on leaves and after the production of germ tubes, appressoria, infection pegs and bulbous haustoria, form a weft of prostrate mycelium which gives rise to secondary appressorial swellings and further haustoria (Sampson and Western, 1941). Haustorial penetration from the mycelial weft tends to be confined to the epidermal cells, although infection of the inner tissues does occur from time to time (Klika, 1922). The mycelial weft also gives rise to upright, separate conidiophores, which produce single-celled conidia from about 5 days after the initial infection. Production of conidia follows a diurnal cycle (Yarwood, 1936), being much more active around midday than during darkness, when clover leaves are naturally closed, a process which renders them more difficult to infect. Towards the end of the season, spherical cleistothecia (90–125 μm) may be formed which bear 10–30 appendages (Kapoor, 1967). At first they are a light straw colour but become dark and carbonaceous as they mature (Carr, 1971). Each cleistothecium contains five to ten ovoid asci (50–80 × 25–40 μm) which in turn contain two to six (average four) ascospores (20–25 × 10–15 μm).

E. polygoni is most severe on red clover but will also attack sweet pea (*Lathyrus odoratus*) and sainfoin as well as white clover. *E. polygoni* exists as specialized physiologic races. Staveley and Hanson (1966a), in the USA, showed 12 distinct races on six clones of red clover. Stavely and Hanson (1966b, c, 1967) studied the genetics of the fungus and host in great detail and showed that host plant resistance was dominant and that the resistance to most races was monogenically inherited.

Symptoms

Symptoms are characterized by off-white, powdery areas of conidia and mycelium on the upper surfaces of leaves often combined with a yellow mottling (Fig. 12.2). Severe infections result in death and browning of the leaves. Mika and Bumeri (1984) noted that attack of red clover by *E. polygoni* resulted in a marked increase in wax secretion by the leaves.

Epidemiology

Infection of leaves by conidia takes place via appressoria and infection pegs which then produce bulbous haustoria (Smith, 1900). Inoculation experiments (Yarwood, 1936) showed that infection is more successful during daylight than at night and can take place within 12 h of a spore landing on the leaf surface. From this initial infection, a prostrate white/grey mycelium is formed, which is largely superficial, but does produce secondary appressoria and haustoria. The mycelium also gives rise to erect conidiophores, which in turn produce elliptical

Fig. 12.2. Powdery mildew symptoms on red clover due to *Erysiphe polygoni* (Photo: courtesy of C.J. O'Rourke).

conidia. A detailed study of their formation was undertaken by Yarwood (1936) who showed that only one conidium was mature at any one time and that this happened at the same time each day on each conidiophore. Production of conidia begins within 5 days of infection. Conidia are carried to other leaves by air currents and are the main method of infection during the growing season. The ascospores, produced towards the end of the season, are forcibly shot out of the cleistothecia and are then carried by air currents to other plants. It is not considered that cleistothecia are particularly important for overwintering in the UK (O'Rourke, 1976), as the fungus can overwinter in mycelial form in host tissues. This stage may, however, be important in the formation of new physiologic races by genetic recombination.

As with other powdery mildews, *E. polygoni* tends to be more severe in warm, dry seasons. Such conditions also exacerbate wilting produced by severe infections, as infected leaves tend to transpire more readily than uninfected leaves (Foëx, 1924).

Effects on Yield and Quality

Both yield and quality of clover crops may be reduced by severe attacks of *E. polygoni*, although the effect can be variable (Horsfall, 1930). Carr (1984) stated that the disease could reach quite serious proportions on red clover in the UK, particularly in warm, dry seasons. However, there appears to be little quantitative information available.

Disease Control

E. polygoni has been shown to be controlled by fungicides such as tridemorph in small-scale trials (Carr, 1984). However, it is not clear whether this would be economic on a field scale. In the USA, breeding has developed red clover cultivars with resistance to *E. polygoni* (Stavely and Hanson, 1967; Taylor *et al.*, 1990). In Australia, breeding of subterranean clover has also resulted in lines with resistance to *E. polygoni* (Anonymous, 1992). In Poland, Mikolajska and Majchrzak (1988) showed reduced occurrence of *E. polygoni* on red clover when it was planted with a cover crop.

PEPPER SPOT

Aetiology

Pepper spot is also known as burn, leaf scorch and leaf spot. It is caused by the fungus *Leptosphaerulina trifolii* (Rost.) Petrak and is a member of *Dothideales* and classified in the *Loculoascomycetes*. Because this species shows a wide variation in spore size on its hosts, there is a high degree of synonymy, e.g. *Sphaerulina trifolii* Rost., *Pseudoplea trifolii* (Rost.) Petr., *Pleospora trifolii* (Rost.) Petr. and *Pseudosphaeria trifolii* (Rost.) Hähn. However, differences in spore size are not noted under controlled conditions (Booth and Pirozynski, 1967) (see below).

Biology

Small roundish ostiolate perithecia (100–150 μm) are formed in larger lesions on leaves, petioles or pedicels, where they are immersed and therefore relatively inconspicuous (Sampson and Western, 1941). Each perithecium contains broadly ovate asci with thickened apices. The asci themselves contain eight hyaline, three-septate ascospores measuring 27–39 × 10–15.5 μm. Ascospores are forcibly discharged from the perithecia and spread by wind and rain to further plants; moisture is required for infection (Carr, 1984). Perithecia are highly resistant to extremes of weather and survive the winter on dead leaves. Spores are liberated from perithecia in the spring to renew the infection cycle. Conidia have not been found (O'Rourke, 1976). In a comparison between isolates of *L. trifolii* from lucerne and white clover, Olanya and Campbell (1990a) showed an optimum growth rate for isolates on culture medium of 20–24°C, an average incubation period of 3–4 days, a latent period of 14–15 days and an infectious period of 21 days. Dorozhkin *et al.* (1989) gave a similar optimum growth rate for isolates from white clover at 21–25°C. Perithecia were reported to form after 5–7 days under optimum light conditions of alternating 12 h darkness and 12 h illumination at 5000–6000 lux. Numerous asci and ascospores were formed after 10 days.

L. trifolii is one of the most common pathogens of white clover (Carr, 1984), but will also attack other clovers. Barbetti (1985b) found it to be one of the

predominant pathogens on subterranean clover in Australia. As well as attacking clovers, *L. trifolii* can attack a wide range of plants, particularly those belonging to the *Cruciferae*, *Euphorbiaceae*, *Gramineae*, *Leguminosae* and *Solanaceae* (Booth and Pirozynski, 1967). A further *Leptosphaerulina* sp., *L. briosiana*, has been postulated as primarily attacking *Medicago* spp., although also attacking clovers (Graham and Luttrell, 1961). However, others consider *L. briosiana* to be merely a race of *L. trifolii* rather than a separate species (Booth and Pirozynski, 1967).

Although differences in cultivar resistance are known in Ladino white clover (Sakai, 1983), there is no general evidence for races of the pathogen. However, O'Rourke (1972), in Ireland, found that isolates of *L. trifolii* from lucerne, although capable of producing disease symptoms on white clover, did so to a lesser extent than on lucerne. This was confirmed by Olanya and Campbell (1990a, b) in the USA. The size of ascospores can vary widely depending on the host tissue from which they are isolated, but no differences are apparent when isolates are grown on potato dextrose agar under standard conditions (Booth and Pirozynski, 1967).

Symptoms

Leaves are the most severely attacked (Fig. 12.3), but unlike other leaf spot diseases, lesions can also be found on petioles, pedicels and even on flowers (Sampson and Western, 1941). Attacks by *L. trifolii* produce a 'peppering' of the leaves with spots, some as small as 0.2 mm in diameter (O'Rourke, 1976), hence one of the common names. Generally, more severe attacks produce more spots, but individual spots do not usually exceed 1 mm. Lighter attacks produce a few, larger spots, up to 3 mm. More severe attacks (>50% of leaf area affected) also cause the leaf to shrivel up, giving the plants a burnt appearance, hence the other common name. Pandey and Wilcoxson (1970) showed that, in lucerne, the type of lesion was also dependent on light intensity, larger lesions with light centres and dark brown margins occurring at 21,500 lux in a 12 h light : 12 h dark cycle, but only black spots developing at 4800 lux in an 8 h light : 16 h dark cycle. Olanya and Campbell (1990a) found greater disease severity at 15 and 22°C than at 30°C under controlled environmental conditions.

Epidemiology

As *L. trifolii* requires moisture to germinate, the incidence of pepper spot is higher in wetter areas. Unlike *K. caulivora*, *L. trifolii* is generally more severe towards the end of the summer in temperate regions (Skipp and Lambert, 1984; Mercer and McGimpsey, 1986; Holmes, 1989, Nelson and Campbell, 1992). Disease also tends to be worse in dense stands and particularly on the lower leaves (O'Rourke, 1976). As *L. trifolii* survives on trash, good crop sanitation will also reduce disease (Barbetti, 1986). There is evidence from the USA (Kilpatrick, 1958) that *L. trifolii* can be transmitted via seed, at least in white clover.

Fig. 12.3. Pepper-spotting of white clover caused by *Leptosphaerulina trifolii* (Photo: courtesy of P.C. Mercer).

Effects on Yield and Quality

Attacks by *L. trifolii* tend to reduce both yield and quality. For example, Hopkins and Gilbey (1987), in England, noted an increase of 21% in the dry matter due to white clover in a mixed grass/clover sward, after the application of chlorpyrifos, methiocarb, and benomyl and indicated that some of this improvement was due to control of *L. trifolii*. Although, Mercer and McGimpsey (1986), in Ireland, found no increase in yield with control of the disease, O'Rourke (1970), also in Ireland, noted a decrease in crude protein content following attack by *L. trifolii*. He also observed increases in the level of oestrogen in leaves, a factor which can have effects on animal health and reproduction.

Disease Control

Carr (1984) stated that no control measures for *L. trifolii* were known. However, sprays of benomyl in England were probably responsible for increases in white

clover dry matter in mixed swards (Hopkins and Gilbey, 1987). On the other hand, although Mercer and McGimpsey (1986) obtained decreases in the incidence of *L. trifolii* on leaves of white clover in Northern Ireland, following monthly sprays of propiconazole, the yield did not increase. Clearly, further research on fungicidal control is required. Some resistance to *L. trifolii* exists (Sakai, 1983), but it is not yet widely used as a control measure.

LEAF SPOT

Aetiology

Leaf spot is caused by the fungus *Pseudopeziza trifolii* (Bivona Benardi) Fuckel and is a member of the *Helotiales* and classified in the *Discomycetes*. Synonyms are *Ascobolus trifolii* Biv.-Bern. and *Peziza trifoliorum* Lib. Schüepp (1959) has suggested that *P. trifolii* and the related *P. medicaginis* (see below) are *formae speciales* – *P. trifolii* f. sp. *trifolii* and *P. trifolii* f. sp. *medicaginis sativae* of the single species *P. trifolii*. Other divisions have been indicated by Schmiedeknecht (1964) (see below).

Biology

Infection of leaves takes place in spring by ascospores (10–15 × 4–6 μm), carried by wind after discharge from asci (50–80 μm long) contained in small, jelly-like, light coloured apothecia (0.2 mm diameter) which overwinter on leaves infected in the previous autumn. New apothecia and spores are then produced on freshly infected leaves. As far as is known there are no conidial or pycnidial stages of the pathogen (Butler and Jones, 1949).

P. trifolii is known from most countries in the world where clover is grown. Leaf spot was listed as the most serious disease causing yield losses in Romania from 1971 to 1975 (Ignatescu and Suceava, 1975). Butler and Jones (1949) claimed that the pathogen caused more economic damage to red clover in the USA than in Britain. However, Carr (1984) stated that it is a serious problem on both white and red clover in Britain, and O'Rourke (1976) also found *P. trifolii* commonly throughout the growing season on both clovers in Ireland. The disease does, however, appear to be generally more severe on red clover and although damage does occur on white clover, it tends to be more sporadic in occurrence, at least in Britain (Carr, 1971). Although *P. trifolii* is only known to attack *Trifolium* spp., a closely related but biologically distinct species, *P. medicaginis*, attacks *Medicago* spp. (Schmiedeknecht, 1964). *P. trifolii* will also attack crimson and alsike clovers (Carr, 1971). Forms of *P. trifolii* attacking red and white clovers are physiologically distinct and will not attack each other's hosts (Carr, 1984). Schmiedeknecht (1964) described them as *P. trifolii* f. sp. *trifolii repentis* and *P. trifolii* f. sp. *trifolii pratensis* on white and red clovers, respectively.

Symptoms

Many dark brown spots, ranging in size from 0.5 to 3 mm (O'Rourke, 1976) can be found on leaves, on both upper and lower surfaces (Fig. 12.4). Spots may also be found on stems and petioles, although, unlike those on the leaves, they rarely fruit (Carr, 1984). The apothecia are just about visible to the naked eye, especially after rain when they swell; there is usually one per leaf spot, although this may be exceeded if the spot is large. Larger spots may also be lighter coloured in the centre compared with the borders. The centre may also fall out producing a shot-hole effect (O'Rourke, 1976). Severe attacks may also result in defoliation (Schmiedeknecht, 1967).

Epidemiology

As moisture is required for the forcible discharge of ascospores from asci, the disease tends to be worse under moist conditions, such as occur in spring and autumn (Skipp and Lambert, 1984).

Effects on Yield and Quality

O'Rourke (1976) reported a general reduction in crop vigour, quality and potential to regrow by damage to leaves and premature leaf-shedding in Ireland. On the other hand, although tests by Lewis and Asteraki (1987), on a range of 12

Fig. 12.4. Leaf spot on white clover caused by *Pseudopeziza trifolii* (Photo: courtesy of C.J. O'Rourke).

white clover cultivars in the UK, showed an average of 53% of leaves infected by *P. trifolii*, only 2.5% of the leaf area was damaged. Generally, there is little quantitative information on the effect of *P. trifolii* on yield, although Barbetti (1992), in Australia, showed a 32% increase in herbage yield of burr medic (*Medicago polymorpha*), partially attributable to control of the related *P. medicaginis* with an end of season propiconazole spray. In addition to any effect on the yield of clover, *P. trifolii* is also known to increase its oestrogen content (Wong *et al.*, 1971), which can lead to effects on the reproductive physiology of grazing animals.

Disease Control

There are little quantitative data available on the control of *P. trifolii* in clover and there are no specific recommendations for the use of fungicides in the UK (Carr, 1984). However, Khar'kov and Kashmanova (1973), in the former USSR, claimed that treatment of red clover seed with sodium molybdate markedly decreased the level of *P. trifolii*, and Kolosov (1972), also in the former USSR, claimed a reduction in pathogen incidence with two aerial sprays of zineb in clover seed beds. In the USA and Australia, fungicide sprays containing benomyl, carbendazim, zinc/maneb, chlorothalonil, flutriafol, propiconazole or triadimefon are effective (the latter three most effective) against *P. medicaginis* on lucerne (Wilcoxson and Bielenberg, 1972; Summers and McClellan, 1975; Barbetti, 1987, 1992).

Tetraploid red clover cultivars tend to be more susceptible to *P. trifolii* than diploid ones (O'Rourke, 1976). Koromyslova *et al.* (1981), in the former USSR, noted some resistance in four red clover cultivars out of 125 tested, and in a population of white clover plants in North Wales, Burdon (1980) observed that, although there was significant variation in the resistance of individual genotypes to *P. trifolii*, the frequency distribution was skewed strongly in the direction of resistance. Although statistically significant differences in resistance were observed among white clover cultivars in the UK by Lewis and Asteraki (1987), the differences did not appear large enough to be of practical significance. The subterranean clover cultivar Denmark is resistant to *P. trifolii* in Australia (Anonymous, 1992).

O'Rourke (1976), in Ireland, recommended early cutting of severely affected crops to avoid premature leaf-shed. This also helps to reduce pathogen inoculum. Similarly, in a comparison between two grazing systems in the UK, O'Donovan and Fothergill (1992) reported a dramatic decrease in the incidence of *P. trifolii* in April, a time corresponding to the onset of continuous grazing with sheep. Mikolajska and Majchrzak (1988) in Poland found that sowing red clover with a cover crop delayed and restricted the occurrence of *P. trifolii* in the first year.

BLACK OR SOOTY BLOTCH

Aetiology

Sooty blotch is caused by the fungus *Cymadothea trifolii* (Pers.) Wolf which is a member of the *Dothideales* in the family *Loculoascomycete*. The conidial state is

known as *Polythrincium trifolii* Kunze ex Ficinus & Schubert. Synonyms include *Dothidella trifolii* (Pers. ex Fr.) Bayliss-Elliott & Stansfield and *Sphaeria trifolii* Pers.

Biology

Plants are infected in the spring by ascospores. As the disease develops, an asexual stage is formed which produces wavy conidiophores (100 × 6–9 μm) which may possess a single septum. The conidiophores bear obovate, pale to light brown conidia (17–24 × 13–24 μm) with a single septum (Booth and O'Rourke, 1973). Conidia are spread to other leaves and penetration occurs either directly (Killian, 1923; O'Rourke, 1976) or through stomata (Elliot and Stansfield, 1924). However, Roderick (1993), in a study under artificial inoculation, could find no evidence of direct cuticular penetration and observed penetration only via the stomata on the adaxial surface. Relative humidity in the range 98–100% was necessary for germination which was greatest between 15 and 22°C. The highest penetration success (via appressoria) occurred at 15°C.

Towards the end of the growing season, shiny black stromata develop on the underside of the leaves next to the existing conidial stromata. The new stromata contain pycnidia which exude pycnidiospores (3–5 × 1.5–2 μm) in slimy, tendril-like masses through papillate ostioles. *C. trifolii* overwinters on fallen leaves where it develops a sexual state, leading to the production of asci from within the pycnidial stromata. It is possible that the leaves become completely decomposed by the end of the winter and that the asci will therefore be found freely in the soil (Killian, 1923). Ascospores are liberated from asci in the spring.

C. trifolii occurs worldwide on most clover species (Wolfe, 1935; Milovidova, 1974; O'Rourke, 1976). Lewis and Thomas (1991) found it to be the most frequently recorded disease on white clover at 16 sites in England and Wales monitored from 1985 to 1987. However, Nelson and Campbell (1992) rated *C. trifolii* as only a minor component of a leaf spot disease complex on white clover in North Carolina. There appear to be differences in resistance between species, cultivars and even members of the same population. Kuprewicz (1935), in Russia, found that ascospores of *C. trifolii*, collected from white clover, successfully re-infected white clover, but infected alsike clover to a limited extent. Ascospores collected from zigzag clover (*T. medium*) infected neither white nor alsike clovers except occasionally under greenhouse conditions. There also appeared to be differences in ascospore sizes between the two sets of isolates. O'Rourke (1971), in Ireland, reported that spores isolated from the white clover cultivar S100 infected other white clovers to varying degrees, but did not infect alsike clover. There does not appear to be any information on physiologic specialization of *C. trifolii*.

Symptoms

Initial symptoms appear in early summer as dark green, granular spots, *c.* 1 mm diameter, usually on the abaxial leaf surface, although in severe cases they may also appear on the adaxial surface (Fig. 12.5). Surrounding leaf tissue usually

Fig. 12.5. Black blotch of white clover caused by *Cymadothea trifolii* (Photo: courtesy of C.J. O'Rourke).

remains green for some time after infection, but in time becomes brown and dry; leaves do not, however, usually fall off immediately. Eventually, especially with a severe attack, defoliation will occur. Changes in leaf ultrastructure, as the result of infection by *C. trifolii*, have been described by Camp and Whittingham (1972) and include disruption of the lamellar organization of the chloroplasts.

Epidemiology

The disease builds up in the field from late spring onwards and reaches its peak in the autumn. McKenzie (1971), in New Zealand, showed that this coincided with a peak in spore numbers. Because of the method of transmission of spores, the disease will be worse under wet conditions.

Effects on Yield and Quality

Although black blotch is a common disease, the amount of damage actually caused in the field is relatively small. Some loss in herbage yield due to defoliation may result, but a greater problem can be the production by the fungus of flavonoid oestrogens which may kill domestic livestock which have ingested quantities of infected forage (Amelung, 1966). There is also evidence of reproductive disorders (Newton *et al.*, 1970) and the formation of mouth ulcers (Anonymous, 1960).

Disease Control

Little information is available on the effectiveness of or economic justification for chemical control. Although McKenzie (1971), in New Zealand, reported a decrease in spore numbers of *C. trifolii* in 1969 following sprays of benomyl, there was no effect the following year. Where persistent problems with the disease occur, use of resistant cultivars should be considered. Burdon (1980), in Wales, found significant differences in disease resistance between individuals of a single population of white clover and Lewis and Asteraki (1987) noted significant differences in the field in levels of *C. trifolii* on 12 cultivars of white clover in England. The possibility that disease resistance was linked to levels of cyanogenic activity was not, however, confirmed by Angeesing and Angeesing (1973). Gibson and Chen (1975) reported a cross from *T. uniflorum* × *T. occidentale* which was resistant to *C. trifolii*.

CLOVER PHYLLODY

Aetiology

Clover phyllody is caused by a phytoplasma-like organism (PLO). Systems for the classification of PLOs have been proposed based on their symptomatology in infected plants, e.g. Chiykowski and Sinha (1990). However, more recent work by Schneider and Seemüller (1994) using molecular techniques, has indicated that there is no close correlation between groupings drawn from analysis of the symptomatology in infected plants and those drawn from analysis of Southern blot hybridizations. The latter indicated that although most PLOs from herbaceous plants were interrelated, there were four main subgroups, designated A, B, C and D. Clover phyllody belonged in Group A along with PLOs associated with American aster yellows, virescence of *Primula* spp., safflower phyllody, Hydrangea phyllody and witches' broom of lime. Lee *et al.* (1993), however, indicated that, based on analysis of 16S rDNA sequences, clover phyllody could be separated from other aster yellows type PLOs. There may also be differences at a microscopic level between PLO groups – Musetti *et al.* (1992) showed clear differences in PLO and host ultrastructure between clover phyllody and apple proliferation, the latter PLO being designated as Group D by Schneider and Seemüller (1994).

Biology

Phyllody is transmitted by various leafhoppers (Jassids), such as *Euscelis plebeja* Fall., *Macrosteles* spp., *Aphrodes bicinctus* Schr. (Carr, 1984) and *Paraphlepsius irroratus* Say (Chiykowski, 1991). The leafhoppers do not feed exclusively on clover, and initial infection of the clover crop by phyllody usually occurs from an outside source. Transmission of phyllody by leafhoppers does not occur immediately after feeding on an infected plant as there is a latent period of about 1 month during which the phytoplasma multiplies inside the leafhopper

(O'Rourke, 1976; Chiykowski, 1991). There is also a suggestion that it may be transmitted through the insect's eggs. Although O'Rourke (1976) suggested that the phytoplasma might also have a deleterious effect on the leafhopper, Chiykowski (1991) could find no evidence of an effect on fecundity or longevity in *P. irroratus*.

Phyllody is primarily a disease of white clover, although other clover species are affected to some extent. Incidence is variable, governed to some extent by the presence of the leafhoppers. Data from Britain (Carr and Large, 1963) showed 40% of white clover seed crops to be affected, with levels ranging from a trace to 35% in individual crops. O'Rourke (1976) stated that up to 10% of plants in old pastures in Ireland were infected. Although there is a wide range of host reaction by white clover cultivars to different isolates of the phyllody phytoplasma, there does not appear to be any information on physiologic specialization of clover phyllody. Little leaf caused by PLOs is also important on tropical pasture legumes (see Lenné, Chapter 13, this volume).

Symptoms

The most obvious symptom of phyllody is the change of the flowering parts into leafy structures, due to an alteration of the auxin balance (Carr, 1961) (Plate 30). This usually occurs in mid-summer after flowering (O'Rourke, 1976) and the altered parts do not generally set seed. Affected plants also tend to be pale with vein-clearing and to be stunted; older leaves may be bronzed. The general lack of vigour may result in the dying-out of infected plants.

Epidemiology

Mild winters favour the survival of leafhoppers and therefore also tend to increase the incidence of phyllody. The disease also tends to be worse in older crops.

Effects on Yield and Quality

Few data are available on the quantitative effect of phyllody on a field scale, although vigour was significantly reduced in plot experiments on mixed white clover/grass swards (Carr, 1984). The reduction in vigour was not completely compensated by the companion grass, probably because of the effect of the phyllody phytoplasma on nodulating *Rhizobium* bacteria, generally rendering them less effective at nitrogen fixation, resulting in an increase in the growth of broad-leaved weeds (Joshi and Carr, 1967; O'Rourke, 1970). The largest effect is likely to be in seed crops where a significant reduction in yield must occur.

Control

Insecticides can be used to control the leafhopper vectors, but because of the latent period, are more successful at controlling the disease within a crop than

from preventing its initial occurrence. Use of insecticides is not considered to be a practical method of control (Carr, 1984). Also not practical on a field scale, but useful for the treatment of small amounts of valuable breeding material, are tetracycline antibiotics, heat treatment and ultra-violet radiation (Carr, 1968). Dale and Cheyne (1993) showed that shoot tip culture (with tip lengths of between 2.4 and 3 mm) could be used to eliminate phyllody and solutions containing zinc sulphate have also been shown to reduce the incidence of infected flowers (Carr and Stoddart, 1963). However, most of these methods do not have long-term effects and plants gradually regain their phyllody condition (Carr, 1984). Breeding for resistance is also not practical as no white clover plants immune to phyllody have been found (Carr, 1966). Although smaller-leaved cultivars are less severely affected than larger-leaved (Carr, 1962), breeding for tolerance is also problematical because of the wide range of host reactions to different isolates of the pathogen. Carr and Stoddart (1963) noted that the level of phyllody was lower where clover was grown in mixed swards as against pure stands, presumably because of restricted vector movement.

CLOVER YELLOW VEIN MOSAIC

Aetiology

Clover yellow mosaic is caused by clover yellow mosaic virus (CYVV) of the potyvirus group. The virus is filamentous with particles *c.* 760 nm long. It infects several species in the *Leguminosae*, but particularly *Trifolium* spp. It is readily transmitted by inoculation of sap and by aphids in a non-persistent manner (Hollings and Stone, 1974).

Biology

CYVV has been shown by immunological studies (Scott *et al.* 1989; Jordan and Hammond, 1991) and genome sequence analyses (Tracy *et al.*, 1992; Uyeda, 1992) to be closely related to bean yellow mosaic virus and pea mosaic virus, together forming the bean yellow mosaic potyvirus subgroup. Bednarek *et al.* (1989), in Poland, indicated that CYVV exists as at least two strains.

The virus has been reported from many clover growing areas of the world: Australia (Johnstone and McLean, 1987), Britain (Carr, 1984), Czechoslovakia (Musil *et al.*, 1986), New Zealand (Foster and Musgrave, 1985) and the USA (Ragland *et al.*, 1986). CYVV can infect a wide range of cultivars of a number of *Trifolium* spp., although some species, notably white clover, appear to be more commonly infected. White, alsike and alpestrine (*T. alpestre*) clovers were most heavily infected in two surveys in the USA (Alconero *et al.*, 1986; McLaughlin and Boykin, 1988) and CYVV was the second most common virus isolated from white clover by Gibbs *et al.* (1966) in a survey of 26 old permanent pastures from a wide range of sites in Britain. Further, in a survey of 63 white clover cultivars in Czechoslovakia (Musil *et al.*, 1986), CYVV accounted for 80–100% of virus

infection and, in a large-scale survey of the effects of 38 legume viruses on 23 hosts in USA and Europe, Hampton *et al.* (1978) reported white clover to be preferentially infected compared to red clover. In addition, Carr (1984) indicated that symptoms due to CYVV on red clover in Britain are rare.

Symptoms

Hampton *et al.* (1978), in the survey just mentioned, observed mottling or mosaic (Plate 31) when CYVV was artificially inoculated into white clover, but none when inoculated onto red clover, although Pokorny (1989) in Czechoslovakia, observed symptoms when an isolate of CYVV was inoculated onto each of four red clover cultivars. Gibbs *et al.* (1966), however, stated that naturally infected, white clover plants were symptomless unless also infected with white clover mosaic virus and/or red clover vein mosaic virus, when mild vein, yellowing symptoms were observed. These apparent contradictions may be due to differences between cultivars. Nelson and Campbell (1991a) in the USA, reported that infections of CYVV on one clone of white clover (T17) gave symptoms of stunting, veinal yellowing and necrosis, mottling and necrotic flecks, whereas infections of another clone (T7) were virtually asymptomatic. On arrowleaf clover (*T. vesiculosum*) in the USA, Pemberton *et al.* (1991) observed curling and wilting of leaflets, systemic wilting, necrosis, leaf reddening and blackening and chlorosis.

Epidemiology

CYVV is transmissible by both sap and aphids, the latter in a non-persistent manner (Carr, 1984). Karl *et al.* (1992) observed successful transmission of a Czechoslovakian isolate of CYVV by 20 aphid species in faba bean.

Effects on Yield and Quality

Artificial inoculation of virus-free white clover with CYVV in the USA showed a reduction in dry matter yield of 27% in the first season and 73% in the second season compared with uninoculated controls, although these reductions were not as severe as those caused by peanut stunt virus (PSV), 64% and 85% respectively (Ragland *et al.*, 1986). However, when both CYVV and PSV were present, reductions were not significantly lower than with PSV alone. Similar trends were noted by McLaughlin *et al.* (1992), again in the USA, who observed a positive relationship between the decline of white clover and increase in the incidence of diseases caused by clover yellow vein, white clover mosaic and peanut stunt viruses, although the latter was the predominant virus throughout the study. On the other hand, Gibson *et al.* (1979) in a study of the effects of CYVV, alfalfa mosaic, bean yellow mosaic and peanut stunt viruses in arrowleaf clover in the USA, found the greatest reduction in yield with CYVV. Nelson and Campbell

(1991a) in the USA, noted that the presence of CYVV in white clover plants infected by *Cercospora zebrina* leaf spot served to alter the epidemic components compared with plants that were virus-free – lesions were fewer but larger with a greater proportion of sporulating lesions. There was also less defoliation.

Disease Control

Although the use of aphicide could reduce transmission, the most practical method of control is the use of resistant cultivars. Gibson *et al.* (1989) indicated that the white clover germplasm registered as Southern Regional Virus Resistant remained 95–100% free of CYVV over 2 years of trials. A polymerase chain reaction test has been developed by Bariana *et al.* (1994) to detect CYVV along with alfalfa mosaic, bean yellow mosaic, cucumber mosaic and subterranean clover mottle.

BEAN YELLOW MOSAIC

Aetiology

Bean yellow mosaic is caused by bean yellow mosaic virus (BYMV) of the potyvirus group. The virus is filamentous with particles *c.* 750 nm long. It causes diseases in various cultivated and wild legumes and also has hosts in a number of non-legume families, especially the *Liliiflorae*. It is transmitted by many aphid species in a non-persistent manner and by inoculation of sap (Bos, 1970).

Biology

As noted under clover yellow vein virus, BYMV is a member of the bean yellow mosaic potyvirus subgroup. A molecular hybridization and immunological study of North American and Australian isolates of the BYMV subgroup by Barnett *et al.* (1987) indicated a diversity of relationships among seven Australian BYMV isolates with some isolates closely related to a North American isolate. Hagita (1986), in Japan, refers to two strains of BYMV, an ordinary and a necrotic strain, the latter causing severe damage to phaseolus bean and possibly having a reservoir in wild white clover.

BYMV is widely distributed in Australia (Johnstone and McLean, 1987), Britain (Carr, 1984), Czechoslovakia (Musil *et al.*, 1986), Korea (Ryu *et al.*, 1986), New Zealand (Foster and Musgrave, 1985) and the USA (Ragland *et al.*, 1986). McLaughlin and Boykin (1988), in the USA, found relatively high incidence of BYMV in alsike, subterranean, red, arrowleaf, and crimson clovers, but not in white clover. Alconero (1983), also in the USA, found BYMV to infect alpestrine, alsike and red clovers but, again, not white clover. Musil *et al.* (1986), in the survey of white clover in Czechoslovakia cited above, found BYMV to occur only sporadically. In Britain, BYMV appears symptomless in white clover (Carr,

1984), but causes necrosis in red clover which can act as a reservoir for inoculum for infection of the pea crop where symptoms can be particularly serious. Hampton *et al.* (1978), in the survey of the effects of legume viruses in Europe and USA noted above, also reported latent and variable symptoms when BYMV was inoculated into red clover but again no reaction when BYMV was inoculated into white clover, although they did obtain a reaction with another virus described as bean yellow severe mosaic. Ashby (1976) noted BYMV on alsike clover in New Zealand and McKirdy *et al.* (1994) on subterranean clover in Australia with 23 out of 87 pastures having an incidence of 1–64%. BYMV also affects other legumes including soyabean (see Sinclair, Chapter 3, this volume), faba bean (see Jellis *et al.*, Chapter 7, this volume) and lupins (see Hill, Chapter 11, this volume).

Symptoms

BYMV generally causes mottling or mosaic symptoms in legumes (Fig. 12.6). Johnstone and McLean (1987), for example, reported yellow-green leaf mottle with some vein banding in 12 cultivars of subterranean clover. Ashby (1976) noted yellowing and streaking of infected leaves of alsike clover.

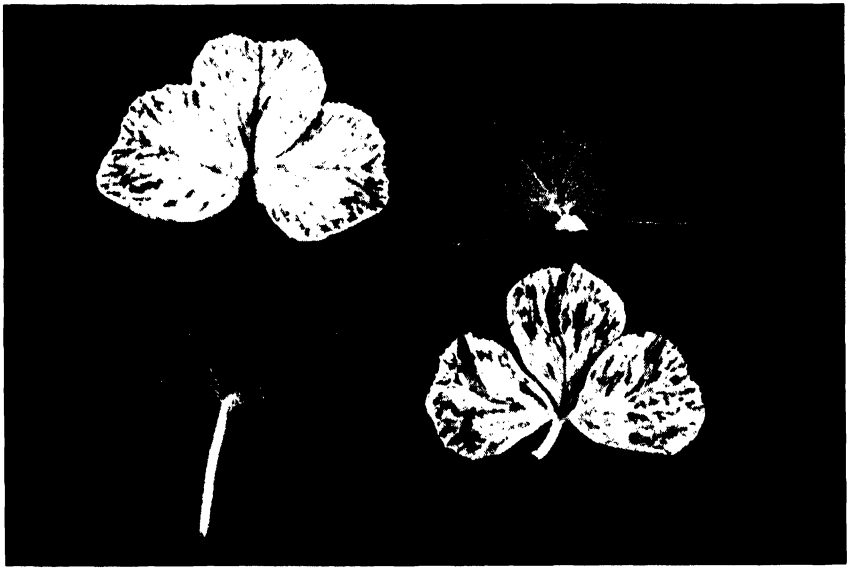

Fig. 12.6. Mosaic symptoms of bean yellow mosaic on leaves of crimson clover (top left and bottom right) compared with healthy leaves (top right and bottom left) (Photo: courtesy of M.J. McLaughlin).

Epidemiology

The virus is generally aphid-transmitted (Jones, 1994) but can also be transmitted by mechanical means (Alconero, 1983) and to a limited extent by seed (McKirdy and Jones, 1995a; Pathipanawat *et al.*, 1995). Hobbs and McLaughlin (1990) noted an isolate of BYMV that was not aphid-transmissible in pea unless accompanied by pea mosaic virus.

Effects on Yield and Quality

Pratt *et al.* (1982) noted a reduction in foliar dry weight and root volume following infection of arrowleaf clover, and a reduction in root volume but an increase in foliar dry weight following infection of alsike clover. These authors also noted a synergistic affect of BYMV with *Phytophthora* spp. in causing root rot symptoms in arrowleaf clover. Yield losses of 25–50% have been reported in the USA in Ladino white clover infected with both BYMV and lucerne mosaic virus (Carr, 1984). Pemberton and Smith (1987), also in the USA, noted survival rates of two breeding lines of arrowleaf clover of 94 and 55% following inoculation with BYMV. Plant losses were greater, the younger plants were when inoculated (Pemberton *et al.*, 1989). Jones (1992), in Australia, noted reduction in root and herbage of subterranean clover of 31–40% with a mild strain of BYMV but 60–63% with a severe strain.

Disease Control

Some control can be obtained by the use of cultivars with a higher level of resistance to BYMV, e.g. in red clover (Sim *et al.*, 1985; Pokorny, 1991) and in arrowleaf clover (Pemberton *et al.*, 1989). Control of insect vectors might be expected to reduce disease, although Jayasena and Randles (1985), in Australia, found little effect of the insecticides malathion, demeton-*S*-methyl and disulfuron on spread of the virus from faba bean. They did, however, observe some reduction in spread with the use of a barrier row of barley. Servítová and Pokorny (1987), in Czechoslovakia, were able, by the use of meristem cultures, to eliminate BYMV from 76% of regenerated plants.

WHITE CLOVER MOSAIC

Aetiology

White clover mosaic is caused by white clover mosaic virus (WCMV) of the potexvirus group. Virus particles are *c.* 480 × 13 nm. Most known hosts are members of the *Leguminosae*. It is readily transmitted by inoculation of sap, but normally not by vectors (Bercks, 1971).

Biology

Comparisons of the nucleotide sequence of WCMV show homologies with potato virus X, barley stripe mosaic and beet necrotic yellow vein (Forster *et al.*, 1988; Huisman *et al.*, 1988; Morozov *et al.*, 1989). There appears to be little information available on variability amongst isolates of WCMV.

The virus is found in many temperate clover-growing areas of the world including Australia (Johnstone and McLean, 1987), Brazil (Mulder *et al.*, 1987), Czechoslovakia (Musil *et al.*, 1986), New Zealand (Forster *et al.*, 1988), the UK (Gibbs *et al.*, 1966) and the USA (McLaughlin and Boykin, 1988). WCMV, as the name suggests, is commonly isolated from white clover (Carr, 1984), but is also isolated from a number of other clovers such as red (Martin *et al.*, 1990), alpestrine, caucasian (*Trifolium ambiguum*), alsike, zigzag (Alconero, 1983) and arrowleaf, crimson and subterranean clovers (McLaughlin and Boykin, 1988). Gibbs *et al.* (1966), in the UK, found WCMV in only 4% of pastures containing white clover compared with 12% for red clover vein mosaic virus. In Northern Ireland, WCMV was found to be widespread in red clover trials surveyed between 1977 and 1978 and, although in most cases only present to a limited extent, it was found in 80% of plants of cv. Hungaropoly in one trial (P. Cooper, Belfast, Northern Ireland, unpublished results). Musil *et al.* (1986), in Czechoslovakia, found the incidence of WCMV to be considerably lower than that of CYVV (see above). Johnstone and McLean (1987) stated that WCMV was widespread in white clover in Australia, particularly in parks and gardens where lawns are mown regularly and in pastures that are cut for hay or silage. Although occasionally isolated from subterranean clover, it was not thought to be a major threat to that species as there were generally few sources of infected white clover in the vicinity of subterranean clover.

Symptoms

Symptoms of WCMV on white clover have been described by Gibbs *et al.* (1966) as chlorotic rings, patches and flecks, later developing brown necrotic flecking. Carr (1984) described the commonest symptoms on white, red, alsike and crimson clovers in Britain to be a light green striping or flecking of the leaves between the veins (Plate 32), although infected white clover was sometimes symptomless. Hampton *et al.* (1978) noted that both red and white clovers could have mottle or mosaic symptoms. Johnstone and McLean (1987) observed that WCMV caused systemic chlorotic mottle and vein-clearing in subterranean clover.

Epidemiology

The sap of WCMV-infected plants is highly infectious and the virus can be spread by mower blades (Johnstone and McLean, 1987). It may also be spread by aphids (Carr, 1984), but this is not certain. Cook *et al.* (1989) noted transmission of WCMV in white clover by the slug *Deroceras reticulatum* Mueller.

Effects on Yield and Quality

Average yield losses in Britain of 10% due to WCMV were recorded in a range of white clover cultivars by Carr (1984). Potter (1993), also in the UK, noted an average reduction of 40% over two cuts of white clover. Guy *et al.* (1980) in Australia, noted an average decrease in white clover plant weight of 83% and a reduction in nodulation of 71% following inoculation with WCMV, while Khadair *et al.* (1984) in Canada, recorded significant reductions in top and root growth, nodulation, rhizobial populations, nitrogenase activity and leghaemaglobin concentration. McLaughlin *et al.* (1992), in the USA, noted a linear increase in diseases of white clover caused by a number of viruses, including WCMV, resulting in a decline in the clover population. Carr (1984) observed that persistence of red clover could be reduced and susceptibility to winter kill increased by infection with WCMV.

Disease Control

Variation in the resistance of clover cultivars to WCMV is known, e.g. in white clover (Carr, 1984) and in red clover, where resistance to the virus is believed to be polygenically controlled (Martin *et al.*, 1990). Tolerance rather than absolute resistance to WCMV appears to be more common (Alconero, 1983; Knight, 1985; Martin *et al.*, 1990). Lewis *et al.* (1985) noted that the incidence of WCMV was lower in red clover when it was grown with Italian ryegrass rather than on its own. Similarly, Brink and McLaughlin (1990) noted a reduction in WCMV incidence when sown with tall fescue. They also found reduced incidence of the virus when clover was sown at lower seed rates.

ALFALFA MOSAIC

Aetiology

Alfalfa mosaic is caused by alfalfa mosaic virus (AMV) and is classified in the alfamovirus group. Virus particles are bacilliform with three different lengths, the longest being *c.* 60 nm. It is readily transmitted both by sap inoculation and non-persistently by aphids to a wide range of host plants (Bos and Jaspars, 1971).

Biology

AMV is the type member of the small group of viruses known as the alfamoviruses. Comparison of non-structural proteins with those of other viruses showed significant homologies with cucumber mosaic and tobacco mosaic (Ziegler *et al.*, 1992). Pietersen *et al.* (1985) indicated a measure of variation among isolates from white clover and lucerne in South Africa and Honda *et al.*

(1986) were able to distinguish immunologically between isolates from white clover and pepino (*Solanum muricatum*) in Japan.

AMV has been observed in clovers from *inter alia*, Australia (Johnstone and McLean, 1987), Canada (McDonald and Suzuki, 1983), Japan (Akita, 1981a), New Zealand (Hickey and Harris, 1989), Kenya (Cameron, 1986), and the USA (Campbell and Moyer, 1984), but it was not isolated in the survey of white clover in Britain, noted above (Gibbs *et al.*, 1966). As the name suggests, AMV is commonly isolated from alfalfa, e.g. Rahman and Peaden (1993) in the USA found the virus in 215 out of 216 fields of alfalfa, but AMV is also found in a number of clovers – Barnett and Gibson (1975) found AMV in 7 out of 19 white clover pastures in the southern USA; McLaughlin *et al.* (1992), also in the USA, found AMV present in white clover but at a relatively low incidence. Similarly, McLaughlin and Boykin (1988) found AMV in red, crimson, alsike, subterranean, white and arrowleaf clovers in the southeastern USA, but all at considerably lower incidences than CYVV, BYMV or PSV. In the Czech white clover survey, noted previously, Musil *et al.* (1986) also found a relatively low incidence for AMV, ranking it with that of WCMV, both being considerably below CYVV although above BYMV. Johnstone and McLean (1987) noted AMV in 77–95% of white clover plants surveyed in one set of pastures in Western Australia, but did not indicate the severity of the infection. They considered that white clover and lucerne were prime sources for aphid transmission of AMV to annual legumes. This was disputed by McKirdy and Jones (1995b), also in Western Australia, on the grounds that although they found AMV in 16 out of 21 pastures of white clover with infection levels of 1–100%, the area of white clover was small and spread of AMV from infected fields was slow. Johnstone and McLean (1987) also referred to reports of AMV on subterranean clover, although this was again questioned by McKirdy and Jones (1995b), who were completely unable to detect AMV in samples from 94 subterranean clover pastures. AMV also affects other legumes including pea (see Kraft *et al.*, Chapter 6, this volume).

Symptoms

AMV has been noted by Hampton *et al.* (1978) as causing malformation of leaves and a mosaic of both red and white clovers (Plate 33). Akita (1981b) described symptoms on red clover as yellow mosaic and leaf wrinkling, while some symptomless plants had a latent infection. Fletcher (1983), in New Zealand, reported that leaves of infected subterranean clover plants were smaller than normal and displayed vein-banding and interveinal yellowing.

Epidemiology

AMV can be transmitted mechanically by infected sap (Hampton *et al.*, 1978), by aphids (Johnstone and McLean, 1987) and via seed (Pathipanawat *et al.*, 1995). In alfalfa, it is also known to be transmitted by pollen (Johnstone and McLean 1987; Pathipanawat *et al.*, 1995).

Effects on Yield and Quality

Jones (1992), in Australia, reported a 20–49% decrease in yield of subterranean clover due to AMV, while Akita (1981c), in Japan, recorded a reduction of around 50% in white clover infected with the virus. As noted above (Carr, 1984) infection of Ladino white clover by AMV and BYMV reduced yields of white clover by 25–55% in the USA.

Disease Control

Cultivars resistant to AMV are known, e.g. Southern Regional Virus Resistant white clover (Gibson *et al.*, 1989) and interspecific hybrids between (caucasian × white) clovers (Pederson and McLaughlin (1989). As with tolerance to WCMV, resistance to AMV in red clover appears to be polygenic (Martin, 1989). A genetic engineering project using tobacco and AMV as a model system has produced tobacco plants transformed with the coat proteins of AMV which were then resistant to AMV infection (Bol and Linthorst, 1993). Barnett and Gibson (1975) were able to produce healthy white clover plants from shoot tip cuttings while Servítová and Pokorny (1987) were able to regenerate 51% of red clover plants free from AMV, also using meristem cultures. Akita (1981a) noted that infection by AMV was lower where pastures were surrounded by windbreaks and where pastures were of grass/clover mixtures. Brink and McLaughlin (1990) found AMV to be lower in white clover crops sown at lower seed rates. McKirdy and Jones (1995b) found very little AMV in white clover crops sown less than 5 years previously compared with longer established crops, suggesting that resowing may be an option where there is severe disease.

RED CLOVER NECROTIC MOSAIC

Aetiology

Red clover necrotic mosaic is caused by red clover necrotic mosaic virus (RCNMV) which is classified in the dianthovirus group. Virus particles are isometric *c.* 27 nm. The virus is readily sap-transmissible to a wide range of herbaceous plant species. No vector is known (Hollings, 1977).

Biology

RCNMV is a member of the dianthovirus group, which includes carnation ringspot (CRV) and sweet clover necrotic mosaic (SCNMV) viruses. Considerable nucleotide sequence studies have been made of this group of viruses indicating the homologies among its members (Osman *et al.*, 1991; Ge *et al.*, 1992; Xiong and Lommel, 1989; Kendall and Lommel, 1992). Serological (Musil *et al.*, 1982; Chen *et al.*, 1984) and electrophoretic studies (Pappu and Hiruki, 1989) indicate

a range of distinct strains or serotypes. A virus designated clover primary leaf necrosis virus was identified as a strain of RCNMV (Rao and Hiruki, 1985).

RCNMV has been identified in the USA (Edwardson and Christie, 1986), Canada (Rao and Hiruki, 1985), Czechoslovakia, Poland and Sweden (Musil *et al.*, 1983) and Britain (Bowen and Plumb, 1979; Frame and Harkness, 1987), where it was first isolated only in 1971. As its name implies, RCNMV attacks red clover, but is also found on other clovers. Hampton *et al.* (1978), in the USA, noted the virus as infecting white clover, although Gilmour and Pemberton (1976), in Britain, were only able to transfer it to white clover with difficulty. It was, however, readily transferred to crimson and alsike clovers.

Symptoms

In red clover, RCNMV causes veinal chlorosis, often followed by severe necrosis and deformation and the plants become weakened and stunted and may die (Gilmour and Pemberton, 1976; Bowen and Plumb, 1979). If the plants survive, the symptoms tend to fade over the summer months (Carr, 1984). The symptoms of RCNMV can be confused with those of other viruses, e.g. symptoms originally attributable to RCNMV in Northern Ireland were found to have been caused by WCMV (P. Cooper, Belfast, Northern Ireland, unpublished results).

Transmission

The virus is easily transmitted mechanically, but there is little evidence of insect transmission. Healthy red clover plants grown in soil in which infected plants had been growing did contract RCNMV (Bowen and Plumb, 1979; Gerhardson and Insunza, 1979). It is possible that in both instances the virus was capable of transmission via the agency of the soil-inhabiting fungus *Olpidium* sp., but Gerhardson and Insunza also noted transmission in the absence of the fungus. Although there have been suspicions of seed transmission, these have never been proven (Carr, 1984). The related sweet clover necrotic mosaic virus is thought to be transmitted via infected pollen carried by western flower thrips (Hiruki *et al.*, 1989).

Effects on Yield and Quality

In England, Bowen and Plumb (1979) reported a yield loss due to RCNMV of 57% in the red clover cultivar Hungaropoly over three cuts.

Disease Control

Control through the use of resistant cultivars is a possibility (Carr, 1984), but there is little evidence from the literature that such cultivars are being produced.

Lewis *et al.* (1985) noted that mixing red clover cv. Hungaropoly with Italian ryegrass reduced the incidence of RCNMV to 1% compared with 9% in a pure stand.

SUBTERRANEAN CLOVER RED LEAF

Aetiology

Subterranean clover red leaf is caused by soyabean dwarf luteovirus (SDV) (syn. subterranean clover red leaf virus, SCRLV). Virus particles are isometric *c.* 25 nm. The virus is restricted to members of the *Leguminosae* and is transmitted by the aphid *Aulacorthum solani* Kaltenbach in a persistent manner, but not by sap-inoculation (Tamada and Kojima, 1977).

Biology

SDV is a member of the luteovirus group which includes barley yellow dwarf virus. It occurs as a number of strains, one of which, the yellowing strain (SDV-Y) is believed to be synonymous with subterranean clover red leaf virus (SCRLV) (Damsteegt *et al.*, 1990), although Ashby *et al.* (1979b) indicated that while the viruses are identical in many aspects, SDV-Y does not infect red clover whereas SCRLV does. Johnstone and McLean (1987) also regarded the two viruses as similar but indicated that although they generally caused no symptoms in white and red clovers, both hosts were major sources of SDV infection of subterranean clover in Australia. Johnstone *et al.* (1984), on the other hand, found no infection of red clover by two isolates designated SCRLV, one from New Zealand and one from Tasmania. Tamada (1973), in Japan, noted differences in symptoms and host ranges of different isolates of SDV and Helms *et al.* (1984) reported two isolates of SDV from New South Wales distinctive in their symptomatology but not serologically.

SDV has been mainly recorded as SDV from the USA (Edwardson and Christie, 1986; Damsteegt *et al.*, 1995) and Japan (Tamada *et al.*, 1969; Tanimura *et al.*, 1985) and SCRLV from Australasia (Ashby *et al.*, 1979b; Jayasena and Randles, 1984; Johnstone and McLean, 1987). Ashby *et al.* (1979a) has recorded SDV on yellow suckling (*Trifolium dubium*), alsike, and subterranean clovers while Johnstone *et al.* (1984) additionally recorded it on crimson clover and Johnstone and McLean (1987) on white and red clovers. McKirdy and Jones (1995b), however, were completely unable to isolate SDV from 94 subterranean clover pastures in Western Australia but were able to isolate it from 7 out of 21 white clover pastures.

Symptoms

Symptoms on subterranean clover are intense reddening of the leaflets developing progressively from the leaflet margins (Fig. 12.7) (Johnstone and McLean,

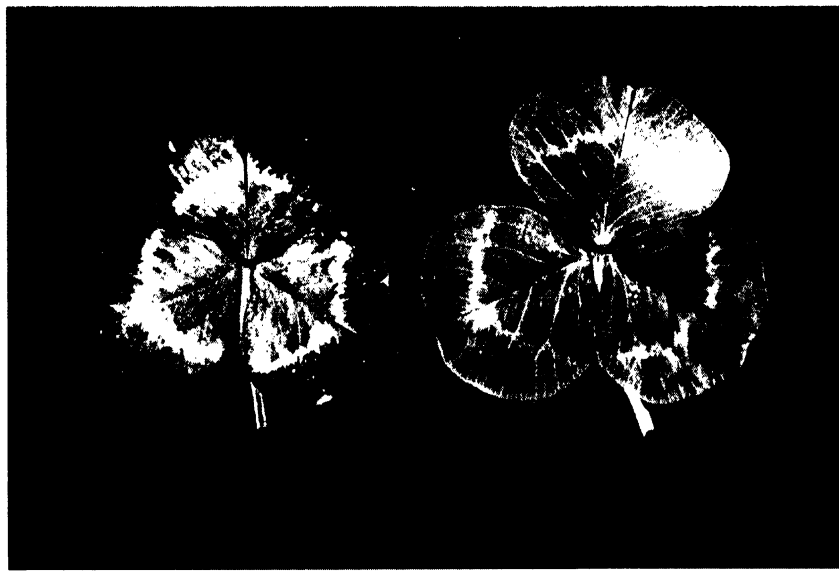

Fig. 12.7. Symptoms of subterranean clover red leaf caused by soyabean dwarf virus on subterranean clover (left) compared with uninfected healthy leaf (right) (Photo: courtesy of M.J. McLaughlin).

1987). The symptoms resemble those attributable to manganese deficiency and indeed the plants have only about half the manganese content of healthy plants. They do not, however, respond to sprays of manganese sulphate. Helms *et al.* (1987) reported that a high light intensity increased the amount of reddening in leaves of subterranean clover infected with SDV. They also noted (Helms *et al.*, 1985) that the optimum temperature for pigment development was in the range 20–30°C. SDV also causes slight stunting and reddening of older leaves of crimson, alsike and yellow suckling clovers (Ashby *et al.*, 1979a). As already indicated, symptoms on red and white clovers are slight.

Transmission

SDV is persistently transmitted by aphids, the most important on subterranean clover being the foxglove aphid, *Aulacorthum solani* (Johnstone and McLean, 1987).

Effects on Yield and Quality

Helms *et al.* (1985) reported a 66-fold reduction in top growth of plants inoculated with SDV compared with virus-free plants. Johnstone (1983) reported an average reduction in growth of 61% in single subterranean clover plants grown

following inoculation. Failures of subterranean clover in autumn, due to almost total infection of seedlings, have been observed in Tasmania, with later infections sometimes also causing failure, but this is unusual (Johnstone and McLean, 1987). Generally, a few plants become infected and die out, making way for healthy ones or for dominant grasses.

Disease Control

Some control has been achieved in faba bean by the use of insecticides (Jayasena and Randles, 1985), but it is not thought that this would normally be economic in clover crops (Johnstone and McLean, 1987). No seed lines of subterranean clover have been identified with resistance to SDV, but some have a degree of tolerance to the virus and may not even exhibit symptoms (Johnstone and McLean, 1987).

CONCLUSIONS

Although diseases of clover can cause significant economic losses, data are often hard to obtain and because frequently more than one disease or condition is associated with a clover crop which is performing poorly, it is not always very easy to quantify the effects of control measures on individual pathogens. Control by chemicals has been shown to be effective for some fungal diseases, but it is probably rare for this to be an economical course of action. Alternative means of control, such as cultural methods or the use of disease-resistant or tolerant cultivars are more likely to be economic.

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DISEASES OF TROPICAL PASTURE LEGUMES

13

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INTRODUCTION

Approximately 25% of the earth's land surface is covered by native or naturalized tropical pastures (Thomas, 1994). The productivity of these pastures can be substantially increased through the establishment of improved pasture legumes and grasses (Fig. 13.1). This has already occurred in northern Australia and parts of tropical America and South-east Asia but is still to be realized in other tropical regions.

Many genera and species of legumes have potential as tropical pasture plants and comprise herbaceous plants, creepers, browse shrubs and trees (Lenné and Sonoda, 1990). The most important tropical pasture legume genera originated in the American tropics (Williams, 1983) and include *Stylosanthes*, *Centrosema*, *Macroptilium* and *Arachis*. In addition, *Desmodium*, a pantropical genus, has useful pasture species from South-east Asia (Lenné and Sonoda, 1990). Other important genera and species include *Aeschynomene*, *Cassia rotundifolia*, *Lablab purpureus*, *Leucaena* spp., *Neonotonia wightii* and *Pueraria phaseoloides* (Skerman 1977; 't Mannetje and Jones, 1992; Lenné and Trutmann, 1994; see Allen and Lenné, Chapter 1, this volume).

Stylosanthes is the most widespread, successful tropical pasture legume genus and has produced many cultivars in Australia, tropical America and Asia (Stace and Edye, 1984; Thomas and Grof, 1986a; Thomas, 1994). In Australia alone, nearly 1 million hectares of grazing land has been sown to *Stylosanthes* cultivars, especially to *S. hamata* cv. Verano and *S. scabra* cv. Seca. The value of *Centrosema* as a source of tropical pasture legumes has been highlighted by Schultze-Kraft and Clements (1990) and successful pasture species from the genus *Desmodium* have been reviewed by Imrie *et al.* (1983) and Thomas and Grof (1986b). *Macroptilium atropurpureum*, best known as cultivar 'Siratro', is a widespread pasture legume in the dry tropics and subtropics (Jones and Jones, 1978; 't Mannetje and Jones, 1992) while forage *Arachis* spp., *A. glabrata* and *A.*

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Fig. 13.1. Animal productivity can be substantially increased through improved legume-based pastures (Photo: courtesy of J.M. Lenné).

pintoi, are well adapted to the wet tropics and subtropics (Prine *et al.*, 1981; Grof, 1986; Cook *et al.*, 1990; Argel and Pizarro, 1992; Kerridge and Hardy, 1994).

Increased use of major tropical pasture legumes has uncovered limitations to their wider cultivation. Over the past 20 years, diseases have emerged as constraints to their productivity and persistence (Lenné and Trutmann, 1994). Many fungi, bacteria, phytoplasmas, viruses and nematodes have potential to cause serious losses. The most important diseases are almost exclusively caused by fungi. There are no major bacterial pathogens of tropical pasture legumes. Although viruses are common and serious pathogens of food legumes (see Chapters 2–10, this volume) and clovers (see Mercer, Chapter 12, this volume), limited attention has been given to virus diseases of tropical pasture legumes until recently (Morales, 1994). The known viruses affecting key tropical pasture legumes are listed in Tables 13.1–13.5. Most are presently considered of minor importance; some are locally important (Morales, 1994). There are no important angiosperm parasites of tropical pasture legumes.

This chapter particularly focuses on the most important, widespread diseases of key herbaceous tropical pasture legumes: anthracnose and foliar blight. Major host-specific diseases and common diseases with broad host ranges among legumes, some of which are featured in other chapters of this book, are also considered. Diseases of local or minor importance of key pasture legumes are summarized in Tables 13.1–13.5. Other legume genera of interest (*Aeschynomene*, *Lablab*, *Leucaena*, *Pueraria* and *Zornia*) are mentioned as hosts of specific diseases where relevant. An extensive list of fungal pathogens of pasture legumes has been published (Lenné, 1990a). Diseases of fodder tree legumes are referred to in

Table 13.1. Diseases of *Stylosanthes* spp. of local or minor importance.

Disease	Causal agent	Distribution	Importance
Seed and seedling diseases			
Pre- and post-emergence rot	*Rhizopus stolonifer* (Fr.) Lind.	Colombia	Minor
Stem and collar rots and wilts			
Sclerotium wilt	*Sclerotium rolfsii* Sacc.	Widespread	Locally important
Neocosmospora wilt	*Neocosmospora vasinfecta* E.F. Smith	South America	Locally important
Bacterial wilt	*Burkholderia solanacearum* (Smith) Yaboucni, Kosako, Oyaizo, Yano, Hotta, Hashimoto, Ezaki & Arakawa	Australia	Moderate
Pythium root rot	*Pythium butleri* Subram.; *P. aphanidermatum* (Edson) Fitzp.; *P. irregulare* Buisman	Australia	Minor
Charcoal rot	*Macrophomina phaseolina* (Tassi) Goid.	Colombia, India	Locally important
Foliar fungal diseases			
Cercospora leaf spot	*Cercospora stylosanthis* Speg.; *C. commonsii* Chupp; *C. canescens* Ellis & Martin	South America USA Malawi	Minor Minor Minor
Leaf mottle	*Pseudocercospora* sp.	South America	Minor
Inflorescence diseases			
Head blight	*Botrytis cinerea* Pers.	Australia, South America, Zimbabwe	Locally important
Inflorescence blight	*Rhizopus stolonifer* (Fr.) Lind.	Colombia	Minor
Viruses			
Peanut mottle	Peanut mottle potyvirus	Colombia	Locally important
Cowpea mild mottle	Cowpea mild mottle carlavirus	East Africa	Minor
Nematodes			
Root-knot	*Meloidogyne* spp.	South America, USA, India	Minor
Root lesion	*Tylenchorhynchus vulgaris* Upadhyay, Swarup & Sethi	India	Locally important

Main source references: Lenné (1990a); Lenné and Trutmann (1994).

Table 13.2. Diseases of *Centrosema* spp. of local or minor importance.

Disease	Causal agent	Distribution	Importance
Root diseases			
Wilt syndrome	Unidentified oomycete	Colombia	Locally important
Foliar fungal diseases			
Leaf blight	*Pseudomonas fluorescens* (Trev.) Migula Biovar II	Central and South America	Locally important; up to 50% forage loss and reduced seed production
Leaf spot	*Cercospora canescens* Ellis & Martin	Widespread	Minor to locally important
Leaf spot	*Pseudocercospora bradburyae* (Young) Deighton	Widespread	Minor to locally important
Viruses			
Mosaic	Blackeye cowpea mosaic potyvirus	Brazil	Minor
Mosaic virus	Cowpea aphidborne mosaic potyvirus	Brazil	Minor
Mosaic leaf distortion	Soybean mosaic potyvirus	Colombia	Locally important
Mosaic leaf distortion	Centrosema mosaic potexvirus	Papua New Guinea	Locally important
Leaf crinkling	Groundnut crinkle carlavirus	West Africa	Minor
Mottling, rugosity	Passion fruit woodiness potyvirus	Australia	Minor
Mosaic	Cowpea mosaic comovirus	Brazil	Minor
Mosaic	Cowpea severe mosaic comovirus	Brazil	Minor
Mild mosaic	Cucumber mosaic cucumovirus	Guadeloupe	Minor
Yellow vein	Clitoria yellow vein tymovirus	Kenya	Minor

Main source references: Lenné (1990a); Lenné *et al.* (1990a); Lenné and Trutmann (1994); Morales (1994).

Chapter 1 and further information is available in Lenné (1992a), Boa and Lenné (1994) and Lenné and Trutmann (1994).

ANTHRACNOSE

Aetiology

Anthracnose is primarily caused by *Colletotrichum gloeosporioides* (Penz.) Penz. & Sacc., the anamorph of *Glomerella cingulata* (Stonem.) Spauld. & Schrenk (Irwin

Table 13.3. Diseases of *Desmodium* spp. of local or minor importance.

Disease	Causal agent	Distribution	Importance
Foliar diseases			
Pink disease	*Phanerochaete salmonicolor* (Bok & Broome) Julich	Malaysia	Locally important
Leaf spot	*Cercospora canescens*; *C. melaleuca* Ell. & Evesh.; *C. desmodiicola* Atk.; *Pseudocercospora meibomiae* (Chupp) Deighton; *P. desmodii-salicifolii* Deighton	Widespread	Minor
Powdery mildew	*Oidium* spp.	South America, Caribbean, India	Minor
Viruses			
Mosaic, mottling	Cowpea severe mosaic comovirus	USA	Minor
Mosaic	Centrosema mosaic potexvirus	Papua New Guinea	Minor
Mosaic	Peanut mottle potyvirus	USA	Locally important
Mild Mosaic	Blackeye cowpea mosaic potyvirus	USA	Minor
Mosaic	Cowpea aphidborne mosaic potyvirus	India	Minor
Streaking vein yellowing	Tomato spotted wilt tospovirus	Hawaii	Minor
Yellow mottle	Uncharacterized geminivirus	Colombia	Minor
Nematodes			
Stem galling	*Pterotylenchus cecidogenus* Siddiqi & Lenné	Colombia	Locally important
Cysts; pearly root	*Heterodera trifolii* Goffart	Hawaii	Minor
Root lesions	*Pratylenchus* spp.	West Africa	Minor

Main source references: Lenné (1990a); Lenné and Stanton (1990); Lenné and Trutmann (1994); Morales (1994).

and Cameron, 1978). The teleomorph is occasionally reported on *Stylosanthes* spp. in Australia (Irwin *et al.*, 1984a) and is often isolated from lesions at the end of the wet season in tropical America (Lenné and Sonoda, 1978a; Lenné, 1994a). Anthracnose is also caused by *Colletotrichum truncatum* (Schwein.) Andrus & Moore (Lenné and Sonoda, 1978b; Sonoda and Lenné, 1986) but rarely by other *Colletotrichum* spp. The former species is more common yet both species can occur together on some tropical pasture legumes (Lenné and Sonoda, 1978a; Lenné *et al.*, 1990a; Lenné and Trutmann, 1994). Anthracnose diseases caused by *Colletotrichum* spp. are also reviewed in Sinclair, Chapter 3, Allen *et al.*, Chapters 4 and 5 and Hill, Chapter 11, this volume.

Table 13.4. Diseases of *Macroptilium atropurpureum* of local or minor importance.

Disease	Causal agent	Distribution	Importance
Foliar diseases			
Angular leaf spot	*Phaeoisariopsis griseola* (Sacc.) Ferraris	Central and South America, Florida, USA	Moderate
Powdery mildew	*Oidium* sp.	South America	Minor
Viruses			
Mosaic	Bean common mosaic potyvirus	India	Minor
Mottle	Passion fruit woodiness potyvirus	Australia	Locally important
Mosaic	Cucumber mosaic cucumovirus	Guadeloupe	Minor
Yellowing mottle	Bean golden mosaic geminivirus	Puerto Rico	Minor
Yellowing mottle	Rhynchosia mosaic geminivirus	Puerto Rico	Minor
Mosaic	Cowpea severe mosaic comovirus	Brazil	Locally important

Main source references: Lenné (1990a); Lenné and Trutmann (1994); Morales (1994).

Table 13.5. Diseases of *Arachis* spp. (principally *A. glabrata* and *A. pintoi*) of local or minor importance.

Disease	Causal agent	Distribution	Importance
Foliar diseases			
Leaf spots	*Mycosphaerella berkeleyi* W.A. Jenkins, *Mycosphaerella arachidis* Deighton	Brazil	Minor
Pepper spot or leaf scorch	*Leptosphaerulina trifolii* (Rostr.) Petrak	Brazil, Colombia	Minor
Viruses			
Mottle	Peanut mottle potyvirus	South America	Minor

Main source reference: Lenné and Trutmann (1994)

Biology

C. gloeosporioides is characterized by immersed, branched, septate, hyaline to brown mycelium and separate to confluent acervuli which may produce brown, smooth, septate, tapered setae (Sutton, 1980, 1993). Conidiophores are hyaline to brown, septate and smooth with enteroblastic, phialidic, hyaline, determinate conidiogenous cells. Conidia of both *C. gloeosporioides* and *C. truncatum* are hyaline, aseptate prior to germination, smooth and thin-walled with those of the former species being cylindrical, straight and 9–24 × 4–12 μm and those of the

latter species being falcate with acute apices and 19–24 × 2–4 μm (Sutton, 1980, 1993). On germination, *Colletotrichum* spp. produce brown, entire to lobed appressoria. Both pathogens exhibit wide variation and it is difficult to provide a standardized description. In general, both species grow well on artificial media (Sutton, 1993). The optimum temperature for mycelial growth ranges from 20 to 30°C. A comparative morphological study of isolates of *C. gloeosporioides* from Australia, Central and South America and Asia has been published (Davis *et al.*, 1992) and a life cycle for *Colletotrichum* spp. on *Stylosanthes* spp. was proposed by Irwin *et al.* (1984a).

Anthracnose is the most widely distributed and damaging disease of key tropical pasture legumes (Lenné, 1992b; Lenné and Trutmann, 1994). Both *C. gloeosporioides* and *C. truncatum* are common on tropical pasture legumes including species of *Aeschynomene*, *Arachis*, *Calopogonium*, *Cassia*, *Centrosema*, *Desmodium*, *Lablab*, *Leucaena*, *Macroptilium*, *Pueraria* and *Stylosanthes* throughout the tropics (Irwin *et al.*, 1984a; Lenné and Calderon, 1984; Lenné, 1990a, b; Lenné and Trutmann, 1994). For example, *Colletotrichum gloeosporioides* has been recorded naturally on at least 19 different species of *Stylosanthes*; at least seven species of *Centrosema*; and at least five species of *Desmodium* (Lenné, 1990a). *Colletotrichum* spp. also cause diseases of many other legumes (Lenné, 1992a, b; see Sinclair, Chapter 3, and Allen *et al.*, Chapters 4 and 5, this volume).

Irwin and Cameron (1978) distinguished two anthracnose diseases of *Stylosanthes* in Australia caused by different strains of *C. gloeosporioides*. The diseases are designated Type A and Type B anthracnose and their symptomatologies are described below. Type A disease-producing strains have a wide natural host range among tropical pasture legumes while Type B isolates are largely restricted to *S. guianensis* (Davis and Irwin, 1994). Some isolates of *C. gloeosporioides* from *Stylosanthes* spp. may infect pasture and crop legumes through artificial inoculation (Lenné and Sonoda, 1978a; Ogle *et al.*, 1986; Vinijsanun *et al.*, 1987). In Arkansas, *C. gloeosporioides* f. sp. *aeschynomene* was pathogenic to lupin, chickpea, lentil and faba bean as well as pasture legumes under artificial inoculation (TeBeest, 1988; Weidemann *et al.*, 1988; Weidemann, 1991). Results suggest that some isolates of *C. gloeosporioides* have very wide host ranges among legumes while others have narrow ranges. To date, however, relatively few isolates have been tested in cross-inoculation studies and few have been designated as *formae speciales*. Further studies of the host range of *C. gloeosporioides* across a broad range of crop and pasture legumes are needed to assess the risks involved in growing different legumes in close association.

Extensive pathogenic specialization and variation for virulence have been identified among *C. gloeosporioides* isolates causing anthracnose of *Stylosanthes* spp. In Australia, several studies have identified from 7 to 16 pathogenicity groups (Davis, *et al.*, 1984, 1987b, 1994a, b; Irwin, 1989). In tropical America, at least eight different isolate groups have been identified (Lenné *et al.*, 1983). The most complex pathotypes of *S. capitata* occur in Brazil while those of *S. guianensis* are widely distributed throughout tropical America ((Lenné *et al.*, 1983; Lenné, 1994a). Comparative pathogenicity studies among isolates from South America, Africa, Asia and Australia showed much greater variability within populations from South America than from any other region (R.D. Davis,

Brisbane, Australia, 1990, cited in Trutmann, 1994a). Regional pathogenic variation in *C. gloeosporioides* from *Stylosanthes* spp. is discussed in Lenné and Trutmann (1994).

In recent years, biochemical and molecular techniques (protein profiles, double-stranded ribonucleic acid (dsRNA) as markers, random amplified polymorphic DNA (RAPD) analyses, restriction fragment length polymorphisms (RFLP), and electrophoretic karyotyping) have been used to assess variability among *C. gloeosporioides* isolates especially from *Stylosanthes* spp. (Lenné and Burdon, 1990; Maclean *et al.*, 1993; Manners *et al.*, 1993; Brown and Sreenivasaprasad, 1994). These analyses have confirmed the considerable pantropical genetic diversity among isolates of *Stylosanthes*-infecting *C. gloeosporioides* and, especially, that populations of the pathogen are more heterogeneous in South America and Africa than in Australia (Brown and Sreenivasaprasad, 1994). These findings support results from pathogenicity studies.

In Australia, Type A and B anthracnose pathogens can be readily distinguished by various molecular techniques (Braithwaite *et al.*, 1990a, b) and subsequent molecular studies have shown that these pathogen groups represent two distinct clonal populations with independent origins (Manners *et al.*, 1993). Subsequent studies have shown that the population of *C. gloeosporioides* which causes Type A anthracnose is remarkably uniform (S. Chakraborty, Brisbane, Australia, 1994, personal communication). Comparative pathogenicity and molecular studies on some of the isolates collected from South America, Africa, Asia and Australia, however, indicated that on a worldwide basis, results from molecular analyses did not correlate with pathogenicity, disease type, host species, or country of origin (Brown and Sreenivasaprasad, 1994). Chromosome transfer between isolates of *C. gloeosporioides* which cause Type A and Type B anthracnose was recently verified in Australia (J.M. Manners, Brisbane, Australia, 1994, 1996, personal communications). This could markedly increase the rate of evolution of new pathogenic races. These findings highlight the need for further studies to elucidate the complex relationship between *C. gloeosporioides* on *Stylosanthes* spp.

Symptoms

C. gloeosporioides produces different symptoms on different species of tropical pasture legumes even when these are congeneric. Symptoms on *Stylosanthes* spp. including *S. capitata* (some accessions), *S. hamata*, *S. humilis*, *S. macrocephala*, *S. scabra* and *S. viscosa* are typically limited leaf and stem lesions (Sonoda *et al.*, 1974; Irwin and Cameron, 1978; Lenné *et al.*, 1983; Irwin, 1989; Davis and Irwin, 1994). Leaf and petiole lesions are 1–3 mm in diameter with cream to light grey centres and dark margins and elliptical stem lesions 2–6 mm in length and similar in colour to leaf lesions (Plate 34). Acervuli are generally visible in lesions. Under humid warm conditions, leaf and petiole lesions coalesce causing defoliation while stem lesions develop into cankers and girdle stems. In contrast, symptoms on *S. guianensis*, *S. grandifolia*, *S. erecta*, *S. montevidensis* and *S. capitata* (some accessions) are commonly expressed as necrosis and blight (Irwin and

Cameron, 1978; Irwin, 1989; Davis and Irwin, 1994). They include dark brown to black, irregular leaf, petiole and stem lesions, leaf chlorosis, defoliation, terminal shoot necrosis, blight and plant death (Plate 34). Under humid conditions, dark lesions are frequently covered by orange-pink spore masses. In Australia, these two contrasting symptomatologies are called Type A (most *Stylosanthes* spp.) and Type B (*S. guianensis*) anthracnoses (Irwin and Cameron, 1978). *C. truncatum* produces similar macroscopic symptoms to those caused by *C. gloeosporioides* (Lenné, 1994a). It is often a secondary colonizer of lesions caused by *C. gloeosporioides*.

Leaf symptoms caused by *C. gloeosporioides* also vary with *Centrosema* species affected (Lenné, 1994b). On *C. macrocarpum*, necrotic lesions develop on leaf borders and on veins; on *C. pubescens* and *C. acutifolium*, symptoms are manifest as death and necrosis of young leaves; while on *C. brasilianum*, round to irregular, cream lesions are formed. In all species, immature pods may be severely blighted and round to irregular, light coloured, sunken, ulcer-like lesions form on mature pods (Fig. 13.2) (Lenné *et al.*, 1983, 1990a; Lenné, 1994b). Often orange spore masses and dark acervuli can be detected in pod lesions. Descriptions of anthracnose on other pasture legumes are given in Lenné and Trutman (1994).

Epidemiology

The infection process of *C. gloeosporioides* is initiated by conidial germination and appressorium formation. Appressoria produce infection pegs which penetrate the

Fig. 13.2. Pod anthracnose caused by *Colletotrichum gloeosporioides* on *Centrosema pubescens* (Photo: courtesy of J.M. Lenné).

cuticle and infect the host (Irwin *et al.*, 1984b). The histopathology of compatible interactions between *C. gloeosporioides* and *S. scabra* cv. Fitzroy and *S. guianensis* cv. Endeavour was examined by light and scanning electron microscopy (Irwin *et al.*, 1984b). The processes were similar on both hosts. After 6 h for *S. scabra* and 12 h for *S. guianensis*, most appressoria were melanized. The first evidence of penetration occurred 12 h after inoculation and more successful infections occurred on *S. guianensis* than on *S. scabra*. In a further study, Ogle *et al.* (1990) noted that the level of penetration was very low (< 9%). Subcuticular infection hyphae were present in transverse leaf sections of both host–pathogen associations after 12 h and both intercellular and intracellular invasions were observed after 24 h. Acervuli developed on cv. Fitzroy by 96 h and on cv. Endeavour by 144 h.

In another study on *S. scabra*, penetration occurred only from melanized appressoria in both compatible and incompatible interactions and was higher for compatible interactions (Trevorrow *et al.*, 1988). Differences in the extent of fungal growth in the cells of compatible and incompatible interactions were apparent 48 h after inoculation. In incompatible interactions, the fungus rarely penetrated more than two cells (Trevorrow *et al.*, 1988; Ogle *et al.*, 1990). Collapse of epidermal and mesophyll cells was evident in susceptible hosts by 72 h. For *C. gloeosporioides* on *S. guianensis*, runner hyphae emerged from the epidermal cells to grow subcuticularly or, more often, superficially, initiating new infections without the formation of appressoria (Ogle *et al.*, 1990).

Anthracnose development on all tropical pasture legumes is favoured by prolonged high relative humidity and moderately high temperatures (Lenné, 1994a). The influence of temperature, leaf wetness period after inoculation, and relative humidity on the development of anthracnose has been studied on *Stylosanthes* spp. under controlled conditions in Australia only. Irwin *et al.* (1984a) reported severe disease developed on plants incubated at 20–30°C and 24 h of leaf wetness after inoculation. In addition, the severity of anthracnose on two accessions of *S. scabra* increased with increasing duration of the leaf surface wetness periods (Chakraborty *et al.*, 1990b). A period of 12 h or longer favoured disease development in the susceptible cv. Fitzroy and maximum severity was reached after 36 h. It appears that anthracnose can develop over a range of leaf surface wetness duration. Similar observations have been made for *C. gloeosporioides* on *Aeschynomene virginica* (TeBeest *et al.*, 1978) and *C. truncatum* on *Desmodium tortuosum* (Cardina *et al.*, 1988).

Anthracnose is spread mostly through rain-splash and windborne spores under wet and windy conditions. There have been limited studies on the effect of climatic parameters on anthracnose under field conditions (Davis and Irwin, 1994). In field experiments during three wet seasons at two sites in northern Queensland, the incidence of anthracnose on *Stylosanthes* spp. varied from moderate to high in some accessions, while others showed good field resistance for the duration of the experiment (Davis *et al.*, 1987a). It was difficult to determine a relationship between rainfall, temperature, relative humidity and disease incidence and severity, especially as severe anthracnose developed under dry conditions (Davis and Irwin, 1994). It was concluded that more devastating anthracnose epidemics may be expected to occur during seasons when there is a break in the rainfall pattern than under continuous rainfall (Davis and Irwin,

1994). Subsequent work has suggested that anthracnose incidence in an average rainfall year is linked with rainfall, relative humidity and temperature but, in a dry year, the availability of inoculum is most critical (Chakraborty and Billiard, 1995).

Both *C. gloeosporioides* and *C. truncatum* are seedborne in most tropical pasture legumes (Ellis *et al.*, 1976; Lenné and Sonoda, 1978a, 1982; Davis, 1987; Lenné and Trutmann, 1994). Seed transmission is believed to be responsible for the rapid spread of anthracnose in northern Australia (Irwin and Cameron, 1978) and probably for its global movement. In addition to facilitating the spread of anthracnose, the association of *Colletotrichum* spp. with seed of *Stylosanthes* spp. can significantly reduce seedling emergence and survival and affect pasture persistence (Lenné and Sonoda, 1979). As most tropical pasture legumes are perennial, *Colletotrichum* spp. can readily survive from season to season on living and dead host tissue. In annuals such as *Stylosanthes humilis*, the pathogens may survive on host debris (Lenné and Trutmann, 1994); however, alternative perennial legume hosts of *C. gloeosporioides* may also contribute to survival of the pathogen.

Losses

Anthracnose is noted as the most serious disease of *Stylosanthes* spp. throughout the tropics (Irwin and Cameron, 1978; Lenné and Calderon, 1984; Irwin, 1989; Lenné, 1994a). In the early 1970s, approximately 2 million hectares of *S. humilis* pastures were destroyed by anthracnose in Australia (Davis and Irwin, 1994). This was considered the single most devastating blow to the development of the tropical beef industry in Australia (B. Walker, Brisbane, Australia, 1994, personal communication). Anthracnose epidemics have also been recorded on cultivars of *S. scabra* and *S. guianensis* in Australia (Davis and Irwin, 1994). Forage yields of *S. hamata* were reduced by up to 58% in Florida (Lenné and Sonoda, 1982) and of *S. guianensis* from 64 to 100% in Colombia with associated reduction in nutritive value (Lenné, 1986). In Australia, yield and seed losses were 22 and 16% in *S. scabra* cv. Fitzroy, 67 and 49% in *S. hamata* cv. Verano and 53 and 42% in *S. guianensis* cv. Graham, respectively (Davis *et al.*, 1987b). Anthracnose has become the major limiting factor to development of otherwise promising selections of *Stylosanthes* spp. into commercial cultivars in Australia and tropical America (Irwin, 1989; Davis and Irwin, 1994; Lenné, 1994a).

Serious foliage and pod anthracnose has been reported on *Aeschynomene*, *Cassia*, *Centrosema*, *Desmodium* spp. and *Pueraria phaseoloides* in tropical America and Australia (Lenné and Sonoda, 1978a; Sonoda and Lenné, 1986; Chakraborty *et al.*, 1994; Lenné, 1994a, b ,c, d, e). However, apart from studies on the effect of anthracnose on *Stylosanthes* spp., no quantitative information is available on losses in these pasture legumes. Even for important and widely grown cultivars of *Stylosanthes* spp., limited information is available on the relationship between disease severity and loss expectation. It is an area in need of further study as the economic impact of diseases of tropical pasture legumes will have to be related to animal output and not to fodder losses in grazed pastures

(Lenné, 1989, 1992b). It is possible for a pasture to sustain substantial dry matter loss but minimal loss in animal production (Anderson *et al.*, 1982; Lenné, 1989).

Management

Resistance is the most practical and economic method of managing diseases of tropical pasture legumes and anthracnose is no exception (Lenné, 1986, 1989; Trutmann, 1994b). Where resistance to anthracnose has been sought in germplasm collections of tropical pasture legumes including *Stylosanthes*, *Centrosema* and *Aeschynomene* spp. (Sonoda and Lenné, 1986; Lenné *et al.*, 1990a; Lenné and Trutmann, 1994), resistant accessions have been identified (Plate 35). Most work has been done on *Stylosanthes* spp.

Major programmes for selection and breeding for resistance to anthracnose of *Stylosanthes* spp. have been in progress in Australia and South America since the 1970s (Thomas and Grof, 1986a; Davis and Irwin, 1994; Trutmann, 1994a, b). In the 1970s and 1980s, effort was focused on selecting genotypes with resistance and acceptable agronomic characters. The rapidity with which *C. gloeosporioides* has apparently overcome previously resistant *Stylosanthes* spp. genotypes, especially in Australia, has increased the challenge to researchers developing resistant cultivars (Irwin, 1989). Recently, advanced breeding techniques and molecular marker technologies have been incorporated into breeding programmes, especially in Australia (Clements, 1989; D.F. Cameron, Brisbane, Australia, 1994, 1996, personal communication) and South America (J.W. Miles, CIAT, Colombia, 1996, personal communication).

The Australian breeding programme is particularly comprehensive and includes the development of composite cultivars, e.g. *Stylosanthes scabra* cv. Siran composed of three genotypes each of which contains different major genes for resistance to common races of *C. gloeosporioides*, and *S. scabra* cultivars with quantitative resistance through a recurrent selection programme (Cameron *et al.*, 1993). Quantitatively expressed resistance to anthracnose was identified in *S. scabra* in Australia (Chakraborty *et al.*, 1988, 1990a; Chakraborty, 1990). Other approaches include pyramiding genes for resistance facilitated by marker technology and using advanced molecular technologies for inserting antifungal genes for resistance to *C. gloeosporioides* (Cameron *et al.*, 1993; Davis and Irwin, 1994; D.F. Cameron and J.M. Manners, Brisbane, Australia, 1994, 1996, personal communications).

In Colombia, heterogeneous *S. guianensis* populations resulting from a 45 biparental diallele cross involving ten selected *S. guianensis* var. *vulgaris* and var. *pauciflora* accessions have been managed by yearly bulk generation advance since 1983 (Cameron *et al.*, 1993; Trutmann, 1994a; J.W. Miles, CIAT, Colombia, 1996, personal communication). Advanced breeding lines continue to show high levels of resistance to local pathotypes of *C. gloeosporioides*, markedly better than that of all released cultivars of *S. guianensis*. In Brazil, *S. guianensis* var. *pauciflora* cv. Bandeirante has been released as an adapted, highly anthracnose-resistant accession for savannah regions (Thomas and Grof, 1986a) while *S.*

guianensis var. *vulgaris* cv. Mineirão was recently released as an anthracnose tolerant cultivar (CIAT, 1993). Research is in progress to insert an antifungal toxin producing gene from *Bacillus subtilis* into *Stylosanthes* spp. (Kelemu and Badel, 1994).

Interest has been shown in Australia and South America in developing improved pastures based on diverse mixtures of *Stylosanthes* spp. In South America, resistant accessions of *S. guianensis* in mixtures were able to protect susceptible accessions over two seasons (Lenné, 1985; Hernández, 1986). Cv. Capica – a blend of five accessions of *S. capitata* – was released in Colombia in 1983 and has been widely grown in Colombia during the past 14 years without major anthracnose problems (Trutmann, 1994a). In Australia, however, results have been variable. Some studies have shown reduced anthracnose on susceptible accessions of *S. scabra* compared to disease levels in pure stands (Chakraborty *et al.*, 1991), while other studies have not shown effective control of anthracnose in mixtures of *Stylosanthes* spp. (Davis *et al.*, 1994d). Although results to date suggest that mixtures of perennials may not be as effective as mixtures of annuals in controlling disease, very limited work has been done on perennials (Smithson and Lenné, 1996). Evidence from studies on natural populations and long-established mixed pastures contradicts experimental results. In Colombia and Brazil, high proportions of susceptible plants occur in native populations of *Stylosanthes* spp. although the populations sustain only slight to moderate anthracnose (Miles and Lenné, 1984; Lenné, 1988). Similarly, in a 14-year-old mixed *Stylosanthes* spp. pasture in northern Queensland, diverse stands of susceptible and resistant plants supported variable, virulent populations of *C. gloeosporioides* with limited damage (Davis *et al.*, 1994c). This demonstrated that stand variability contributed to anthracnose management. It is clear that further studies are merited on the function and role of pasture legume mixtures in controlling anthracnose.

Tropical pasture legumes are usually associated with grasses and other vegetation in pastures (Davis and Irwin, 1994; Thomas, 1994). Different grass associations may have varying effects on anthracnose incidence and severity (CIAT, 1981, 1983). Anthracnose developed more rapidly in associations of *S. guianensis* with tall, erect grasses than in association with short grasses or in unassociated stands (CIAT, 1983). It had been expected that grass would act as a barrier to movement of conidia of *C. gloeosporiodes*. However, microclimatic conditions in some associations actually favoured the development of anthracnose. Lenné *et al.* (1987), Lenné (1989) and Davis *et al.* (1994a) have noted that where the pasture sward is dense, the incidence of anthracnose on *Stylosanthes* spp. is greater. In associations of *S. guianensis* with *Andropogon gayanus* and native *Trachypogon* spp., there was considerably less anthracnose under higher versus lower stocking rates in both associations (Lenné *et al.*, 1987). Clearly, grazing pressure may be manipulated to reduce the development of anthracnose.

Recovery of *C. gloeosporioides* was reduced or prevented when pods and seed of *Stylosanthes* spp. were scarified in sulphuric acid for 3 min (Ellis *et al.*, 1976). Benomyl can significantly reduce the rate of seed transmission of anthracnose in *Stylosanthes* spp. both through application to seed crops and through seed treatment (Lenné and Sonoda, 1982; Davis, 1987, 1990). Harvested seed can be satisfactorily treated prior to sowing by dehulling and dusting with benomyl but

unfortunately, in Australia, the procedure is rarely used (Davis and Irwin, 1994). In Colombia, annual and biennial burning reduced anthracnose in *S. capitata* by up to 78% (Lenné, 1982) while, in northern Queensland, burning pastures dramatically lowered recruitment of anthracnosed seedlings (Davis, 1991). Although the advantages of burning for anthracnose control have been demonstrated, the use of burning as a pasture management tool is viewed cautiously by graziers (Davis and Irwin, 1994).

In the humid tropics of Peru, *S. guianensis* is generally not severely affected by anthracnose although pathogenic isolates of *C. gloeosporioides* are common. Studies have shown that phylloplane bacteria on *S. guianensis* (Fig. 13.3) are antagonistic to *C. gloeosporioides* (Lenné, 1986). Pathogenic isolates of *C. gloeosporioides* were more severely inhibited by antagonistic phylloplane bacteria than weakly pathogenic isolates (Lenné and Brown, 1991). It also appears that climatic conditions in the humid tropics favour the activity of these antagonists. In the same environment, it was also shown that *C. gloeosporioides* may remain latent after invading *S. guianensis* (Lenné, 1986). Latent infection by *C. gloeosporioides* is common and has been detected on several *Stylosanthes* spp. throughout tropical America (CIAT, 1982, 1983). However, its frequency is higher in the humid tropics (Lenné, 1986). Latency is an extremely effective way of reducing the infection rate. Although further studies are needed, it appears that an effective naturally occurring biological control system for anthracnose may be operating on *S. guianensis* under humid conditions (Lenné, 1986).

Fig. 13.3. Phylloplane bacteria antagonistic to *Colletotrichum gloeosporioides* on leaves of *Stylosanthes guianensis* (Photo: courtesy of J.M. Lenné and DANI).

FOLIAR BLIGHT

Aetiology

Foliar blight is caused by *Rhizoctonia solani* Kühn, binucleate *Rhizoctonia* sp. (BNR) and *R. zeae* Voorhees (Lenné *et al.*, 1989). All three species may affect *Centrosema* spp.; however, only the first has been recorded on other tropical pasture legumes (Lenné and Trutmann, 1994). The basidial state of *R. solani* – *Thanatephorus cucumeris* (Frank) Donk – has been recorded on *Macroptilium atropurpureum* in Thailand (A. Pachinburavan, Kyoto, Japan, 1985, personal communication), however, the *Ceratobasidium* sp. state of binucleate *Rhizoctonia* sp. has not been reported on any tropical pasture legumes to date.

Biology

Rhizoctonia spp. belong to the Mycelia Sterilia and do not produce conidia. They are distinguished primarily by vegetative characters. Many isolates produce sclerotia. In a study of almost 300 isolates of *Rhizoctonia* spp., mostly obtained from foliage of *Centrosema* spp. in tropical America, 54% were *R. solani* anastomosis groups AG-1 and AG-4 while 42% were binucleate *Rhizoctonia* sp. (BNR) (Lenné *et al.*, 1989). It was interesting to note that more than 14% of the isolates isolated from blighted leaves were AG-4, which is normally associated with root rots and soil. Extensive variation within these groups was also found through morphological and isozyme studies (Olaya, 1985). Considerable variation was also found in growth, colour, zonation, sclerotial characteristics, mycelial texture and virulence among seven isolates of *R. solani* from *Stylosanthes* spp. in Colombia (Olaya and Lenné, 1986). Six of seven isolates were multinucleate; one was binucleate. Among the multinucleate isolates, anastomosis groups AG-1, AG-2 and AG-4 were identified.

Foliar blight is the most widespread disease of *Centrosema* spp. in the humid tropics, being recorded in more than 15 countries (Lenné, 1990a). Similarly, it is common on *Stylosanthes* spp. in tropical America and has also been recorded in Florida, Malaysia, Papua New Guinea, the Solomon Islands and Zambia. It also affects many other legumes and is reviewed further in Allen *et al.*, Chapter 5, this volume. Highly pathogenic AG-1 isolates are commonly obtained from blighted leaves of *Centrosema* and *Stylosanthes* spp. in tropical America (Lenné *et al.*, 1989). As expected, AG-1 isolates were, in general, more pathogenic to *Centrosema* spp. foliage than AG-4 and BNR isolates, also isolated from foliage. In seedling inoculations, however, some isolates of BNR were as pathogenic as *R. solani* AG-1 (Olaya and Lenné, 1987). *Rhizoctonia zeae* was occasionally found on blighted *C. brasilianum* leaves (Alvarez and Lenné, 1988).

Foliar blight is also the most widespread disease of Siratro. It was first reported in the early 1960s in Queensland (Tothill, 1966; Skerman, 1977) and in 1970 in Florida (Sonoda *et al.*, 1971). It has since been recorded throughout humid tropical regions (Lenné and Sonoda, 1985; Lenné, 1981, 1990a). Ecologically specialized isolates have been found associated with different symptoms on *Macroptilium atropurpureum* in Florida (Sonoda, 1980). All *R. solani* isolates

causing foliar blight belonged to AG-1 while most isolates obtained from stem cankers belonged to AG-4 (Sonoda, 1980). Ecological specialization has also been reported for web blight of cowpea (Allen, 1983). A complex of at least three species of *Rhizoctonia* contributes to foliar blight of tropical pasture legumes globally (Lenné and Trutmann, 1994).

Recent studies of variability in *R. solani* using molecular techniques including RAPD analyses and RFLPs have revealed a much greater level of variability between strains than that detected by AG typing (Vilgalys and Gonzalez, 1990; Duncan *et al.*, 1993). Such analyses are needed for isolates from tropical pasture legumes.

Rhizoctonia spp. are noted for their extensive host ranges. They are widely recognized as the cause of legume foliar blight or web blight. Both tropical pasture (*Aeschynomene, Centrosema, Desmodium* and *Stylosanthes* spp.) (Lenné and Trutmann, 1994) and food legumes including common bean, lima bean, soyabean (see Sinclair, Chapter 3 this volume) and cowpea (see Allen *et al.*, Chapter 5, this volume) are important hosts. Foliar blight has been observed on at least ten *Centrosema* spp. and at least five *Stylosanthes* spp. (Lenné *et al.*, 1983, 1989, 1990a; Lenné, 1990a); however, no cross-inoculation studies have been reported with these genera. Similarly, no extensive studies have been made of isolate cross-compatibility among legume genera. In one study of *R. solani* in Brazil, isolates differed in pathogenicity and capacity to produce sclerotia and one isolate from *Neonotonia wightii* was highly pathogenic to cowpea (Cardoso *et al.*, 1982). This finding suggests that further studies on cross-compatibility should be given priority as potentially susceptible food and pasture legumes (e.g. as green manures) are commonly grown in the same cropping systems.

Symptoms

On all tropical pasture legumes affected, symptoms appear as small water-soaked lesions which enlarge, and become cream to light brown, necrotic, irregular to rounded shaped spots which may coalesce under prolonged conditions of high humidity to cover whole leaflets and leaves (Plate 36) (Lenné and Trutmann, 1994). Profuse growth of fungal mycelium throughout the foliage produces mats of leaves stuck together with mycelial strands – hence the common use of the term 'web blight' for this symptomatology (Plate 36). Sclerotia are common on blighted leaves. Considerable defoliation can occur under humid conditions.

Rhizoctonia spp. may also cause crown and root rot of *Centrosema* spp. BNR caused severe crown rot of *C. macrocarpum* in Palmira, Colombia, in 1985 (Olaya and Lenné, 1987). On *Macroptilium* spp., stem canker caused by AG-4 isolates is manifest as tan-coloured cankers up to 0.5 cm long occurring frequently on the lower side of trailing vines (Sonoda *et al.*, 1971).

Epidemiology

The initial primary inoculum for foliar blight of *Centrosema* spp. is probably sclerotia. Although infection from basidiospores has been implicated for other hosts

(e.g. Echandi (1965) for *Phaseolus* bean), the sexual stages of the two common pathogenic species have not been found on *Centrosema* spp. In all tropical pasture legumes, initiation and development of foliar blight from foci at the beginning of the wet season is favoured by high relative humidity, frequent prolonged rain and moderately high temperatures. In Central and South America, foliar blight is most severe in regions with greater than 1500 mm mean annual precipitation. Foliar blight is usually most severe during the first months of the wet season, causing considerable forage loss, but the epidemic commonly decreases in intensity toward the middle and end of the wet season as periods of prolonged rainfall become infrequent.

Foliar blight of Siratro is more severe under warm, wet conditions and especially in subhumid to humid tropical regions with >1500 mm annual rainfall (Sonoda, 1975, 1976; Shaw and Whiteman, 1977; Lenné and Sonoda, 1985). The disease may occur throughout the wet season but epidemics are most common at the beginning of the wet season as observed for *Centrosema* spp. (Lenné and Sonoda, 1985). Although the sexual stage of *R. solani* has been reported from Thailand, only *R. solani*, as sclerotia and aerial mycelium, has been implicated for *M. atropurpureum* in tropical America (Sonoda *et al.*, 1971; Sonoda, 1980). Whether initiating from sclerotia or hyphal fragments, actively growing hyphae form an infection cushion on contact with the host. Penetration occurs directly or through stomata, and subepidermal hyphae spread both inter- and intracellularly.

The pathogen complex is primarily soilborne either directly or in plant residues (Lenné *et al.*, 1983, 1990a). Sclerotia readily survive in soil and plant debris for several years and are disseminated by wind, rain and animals. Being a perennial, infected adult plants of *Centrosema* probably also contribute to pathogen survival. In Thailand, propagules of the fungus were isolated from soil to a depth of 5 cm where foliar blight had occurred on *M. atropurpureum* (Pachinburavan, 1986). Ability to survive in soil as fungal mycelium, sclerotia or associated with host debris possibly varies with AG, but this has not been studied for tropical pasture legumes.

Losses

Foliar blight or web blight, caused by *Rhizoctonia* spp., occurs worldwide and often causes serious economic damage (Holliday, 1980). It is particularly serious in humid tropical and subtropical regions on a wide range of tropical pasture legumes (Lenné, 1990a; Lenné and Trutmann, 1994). Although forage losses of more than 50% dry matter have been measured in pastures of *C. brasilianum*, adult plants usually survive and regrow after the epidemic has abated (Alvarez and Lenné, 1988; Lenné *et al.*, 1989) in contrast to cowpea and common bean where plant mortality is a common feature (Allen, 1983). In grazed pastures, however, seedlings of *C. brasilianum* are often killed, reducing survival and persistence (Lenné *et al.*, 1990a). In Siratro, forage losses due to foliar blight can be as high as 50–80%, yet adult plants usually recover as noted for *Centrosema* spp.

(Sonoda *et al.*, 1971; Sonoda, 1980). Stem canker has also been detected on Siratro in southern Florida but its effect on yield is not known (Sonoda, 1980).

In northern Queensland, web blight affects pastures of *Stylosanthes hamata*, *S. humilis* and *S. guianensis* during the wet season, resulting in extensive foliage death (O'Brien and Pont, 1977) but there is no quantitative information on losses. Foliar blight may also cause moderate to severe defoliation of *Aeschynomene* spp. (Sonoda and Lenné, 1986); *Desmodium* spp. (Lenné and Stanton, 1990; Lenné, 1994d); *Arachis* spp. (Lenné, 1990a, 1994c); *Cassia rotundifolia* (Lenné, 1990a, b, 1994c); *Neonotonia wightii* (Lenné, 1990b, 1994c) and *Pueraria phaseoloides* (John, 1963; Lenné, 1994c), depending on environmental conditions, in tropical America, Asia and northern Australia.

Management

Multi-locational field screening has shown that foliar blight severity varies among *Centrosema* spp. In Florida, Sonoda *et al.* (1971) noted differences in susceptibility among *C. plumieri* and other species. Variation in reaction to *Rhizoctonia* spp. among accessions of *C. acutifolium*, *C. brasilianum*, *C. macrocarpum* and *C. pubescens* has been noted in both field and glasshouse studies (Olaya and Lenné, 1987). In South America, screening of more than 200 accessions of *C. brasilianum* has shown that most accessions are moderately to severely blighted (Schultze-Kraft and Belalcázar, 1988; Lenné *et al.*, 1989; Lenné, 1994b). Although useful resistance has not been identified in *C. brasilianum*, high levels of resistance have been found in *C. acutifolium* and *C. macrocarpum* which may be used in future breeding programmes (Lenné *et al.*, 1989). At present, it is recommended that *C. brasilianum* should not be grown in the humid tropics (>1500 mm mean annual rainfall).

In southern Florida, Sonoda *et al.* (1971) found all accessions of *M. atropurpureum* susceptible while evaluating 72 introduced pasture legumes for reaction to foliar blight. Although Hutton (1970) reported tolerance in *M. atropurpureum* in Australia, observations of these same lines in the 1980s noted that all were susceptible to some degree under conditions conducive to foliar blight development (Lenné, 1994f). Further evaluation of more germplasm is therefore necessary in the search for resistance to this disease.

Use of small plot evaluations with natural inoculum is not reliable in selecting for resistance (Lenné, 1994b). Improved field screening methodology has been developed using inoculated spreader rows (Lenné *et al.*, 1989; Trutmann, 1994a). Development of a seedling inoculation method using liquidized frozen mycelium and targeted inoculation have also greatly improved the reliability of studies on reaction of *Centrosema* spp. seedlings to *Rhizoctonia* spp. isolates (Lenné *et al.*, 1989; Lenné, 1994b). Choice of isolate is critical and a range of virulence should be included. In comparative pathogenicity tests, Olaya (1985) found accessions of *S. guianensis* var. *pauciflora* to be more susceptible to foliar blight than *S. guianensis* var. *vulgaris*. It was thought that sticky secretions from glandular trichomes on var. *pauciflora* stimulated mycelial growth. In general, foliar blight increased with age in accessions of var. *pauciflora* and decreased with or was independent of age for accessions of var. *vulgaris* (Olaya, 1985).

Research on the resistance of *Phaseolus* bean to foliar blight has found that growth habit is an important factor. More erect accessions were less affected by foliar blight than prostrate accessions (Galindo *et al.*, 1983a, b). Evaluation of growth habit within existing *Centrosema* spp. and *M. atropurpureum* collections may be of value in selecting less affected germplasm. Because foliar blight of tropical pasture legumes is caused by a complex of *Rhizoctonia* spp. and anastomosis groups, selection and breeding for resistance will be difficult. Studies of the effect of environment on the population dynamics of the pathogen complex should also be given priority.

Fungi with potential as biocontrol agents against *Rhizoctonia* spp., including *Gliocladium* spp., *Penicillium* spp. and *Trichoderma* spp., have been isolated from leaves of *Centrosema* spp. (Alvarez and Lenné, 1983; Lenné *et al.*, 1990a). In addition, species of the bacteria *Acinetobacter*, *Chromobacterium*, *Pseudomonas*, *Serratia*, *Bacillus* and *Enterobacter* were also resident on leaves of *C. brasilianum* with foliar blight. *In vitro* studies confirmed the antagonism of these fungi and bacteria to mycelial growth of *R. solani*. This indicates that they have potential to contribute to the management of *R. solani* on *Centrosema* spp. in the field. The natural association of such microorganisms with *Centrosema* spp. affected by foliar blight may contribute to the decline of foliar blight epidemics toward the middle and end of the wet season.

Galindo *et al.* (1983a) showed that mulching was effective in controlling web blight of *Phaseolus* bean. Mulch was found to form a barrier to splashing of pathogen propagules from the soil to plant tissues. A range of mulches were effective including rice husks, maize leaves or weeds killed by herbicide (Galindo *et al.*, 1982). Although similar trials were conducted with *Centrosema* spp. in Colombia with mulches formed from dried pasture grasses, results were variable (CIAT, 1984). Foliar blight was often more severe in the mulched treatments as *R. solani* colonized the mulch and then infected *Centrosema* spp., especially semi-prostrate materials. Preliminary studies were made using heavy grazing as a means of keeping the pasture canopy open and reducing foliar blight (J.M. Lenné, CIAT, Colombia, unpublished results); however, heavy grazing can also lead to legume dominance in a mixed pasture which could lead to increased problems with foliar blight. Observations in Australia in Siratro pastures have found increased damage due to foliar blight under grazing as animals move the fungus around the pasture (J.M. Hopkinson, Walkamin, North Queensland, 1985, personal communication).

ZONATE LEAF SPOT

Aetiology and Biology

Zonate leaf spot is caused by *Cylindrocladium colhounii* Peerally (Lenne and Alvarez, 1990; Lenné *et al.*, 1990a). The fungus is characterized by branching conidiophores arising from a stipe, hyaline phialides 7–14 μm long and hyaline, cylindrical, three septate conidia, 40–60 × 4–5.5 μm (Peerally, 1974; Lenné and Alvarez, 1990). On potato dextrose agar, the fungus produced uniform,

dark-tan coloured colonies. The host range of the pathogen includes *Centrosema acutifolium*, *C. arenarium*, *C. brasilianum*, *C. macrocarpum*, *C. pubescens*, *C. schiedianum*, *C. tetragonolobum* and *C. virginianum* (Lenné and Alvarez, 1990). Leaf spots caused by this pathogen have also been recorded on other hosts, and among legumes pod rot has been recorded on *Canavalia ensiformis* (Lenné and Alvarez, 1990). It is possible that isolates from these hosts may infect *C. acutifolium* and vice versa or isolates may be host-specific. Further studies with *C. colhounii* from a range of hosts are needed to clarify the relationships between these diseases. No clear evidence of pathogenic specialization has been found among isolates from *Centrosema* spp. (Lenné and Alvarez, 1990).

Symptoms

Lesions first appear as small, irregular, mid-brown spots which enlarge to 1–2 cm in zones of varying shades of brown (Fig. 13.4) (Lenné and Alvarez, 1990). Affected leaves become chlorotic and defoliation is common under humid conditions.

Losses

Zonate leaf spot causes moderate to severe defoliation of *C. acutifolium*, *C. brasilianum* and *C. pubescens* in the eastern plains of Colombia and in Costa Rica

Fig. 13.4. Irregular zonate lesions caused by *Cylindrocladium colhounii* on *Centrosema acutifolium* (Photo: courtesy of CIAT).

(Lenné and Alvarez, 1990; Lenné, 1994b). During the period 1986–1988, zonate leaf spot caused 30% loss of dry matter and associated reduction in quality of *C. acutifolium* in grazed pastures in Colombia (Lenné and Alvarez, 1990). This disease has potential to cause serious losses to *Centrosema* spp. under humid conditions but it is regarded as a secondary foliage disease on other hosts (Peerally, 1974).

Management

Among 28 accessions of *C. acutifolium* screened for reaction to zonate leaf spot in Colombia, those from Mato Grosso, Brazil, and Vichada, Colombia, were highly susceptible (Lenné and Alvarez, 1990). Moderate levels of resistance only have been identified in accessions of *C. acutifolium* from Minas Gerais and Goias, Brazil. High levels of resistance have been found in other *Centrosema* spp. As the present collection of *C. acutifolium* is small, further collection of germplasm from Minas Gerias and Goias is encouraged.

FALSE RUST

Aetiology and Biology

False rust (also referred to as wart), caused by *Synchytrium* spp., damages *Desmodium* spp. (two species) and *Macroptilium atropurpureum* (one species) (Lenné and Trutmann, 1994). False rust is a serious disease of *D. ovalifolium* in Colombia and Ecuador (Lenné, 1985; Lenné and Stanton, 1990; Lenné *et al.*, 1990b). It is caused by *Synchytrium desmodii* Munasinghe and belongs to the subgenus *Mesochytrium* (Munasinghe, 1955; Lenné, 1985) and was first recorded in Sri Lanka in 1952 in *D. ovalifolium* cover crops in rubber plantations (Munasinghe, 1955). The pathogen was introduced to South America in the 1970s on infected debris associated with seed produced in South-east Asia (Lenné, 1985). It has also been recorded in China, Colombia and Tanzania (Lenné and Stanton, 1990). It is not known whether false rust affects native stands of *D. ovalifolium* in South-east Asia.

Galls of *S. desmodii* containing both summer sporangia and resting sporangia are produced on infected leaf, petiole and stem tissues of *D. ovalifolium* (Price, 1987). Gall diameter varies from 120 to 300 μm while orifice diameter varies from 20 to 210 μm. Bright yellow, polygonal to roughly spherical, summer sporangia, 16–25 μm in diameter, develop within a membrane which protrudes from the recently opened gall (Munasinghe, 1955; Price, 1987). Unlike other *Synchytrium* spp., summer sporangia of *S. desmodii* are not liberated from the open gall but zoospores are released from within the gall (Price, 1987). Each sorus may have 20–50 summer sporangia. Zoospores are hyaline, spherical, uniflagellate, 3 × 5 μm with the flagellum up to 16 μm in length (Munasinghe, 1955; T.V. Price, unpublished data). Resting sporangia, produced within galls,

are liberated when leaf tissue breaks down, and are brown, oval, thick-walled and 64–176 × 57–96 µm.

False rust of *D. intortum* is caused by *Synchytrium citrinum* (Lagerh.) Gäumann and belongs to the subgenus *Woroninella* (Karling, 1964; Lenné, 1985). It is common in native populations of *D. intortum* in Colombia and is also known from Ecuador, Jamaica, Guatemala and Venezuela (Karling, 1964; Lenné, 1985, 1990a). The fungus produces lemon yellow, ovoid–subspherical, thin-walled sori, 120–200 µm in diameter (Karling, 1964). Summer sporangia are polyhedral, 11–25 µm in diameter, with fine, granular, pale lemon contents and thin hyaline walls. Zoospores are unknown and resting sporangia are not produced.

The known host range of *S. desmodii* is confined to species of *Desmodium* including *D. adscendens*, *D. barbatum*, *D. heterocarpon* and *D. ovalifolium* (Lenné and Stanton, 1990). In glasshouse inoculation studies, *D. canum*, *D. heterophyllum*, *D. intortum* and *D. uncinatum* were immune to the fungus (Lenné, 1985). The host range of *S. citrinum* includes *D. axillaris*, *D. canescens* and *D. intortum* (Karling, 1964). In inoculation studies, *Desmodium ovalifolium* was immune to *S. citrinum* (Lenné, 1985).

False rust of *M. atropurpureum* is caused by *Synchytrium phaseoli* Weston and belongs to the subgenus *Woroninella* (Karling, 1964; Walker, 1983; Lenné, 1994f). It has a short life cycle producing only summer sporangia (Walker, 1983). More than 500 sporangia may be produced in a single sporangial gall or sorus on the host. Sporangia are aseptate, golden in colour, thin-walled, smooth, 16–22 × 14–20 µm, angular and polygonal to polyhedral in shape. Sporangia are released by rupture of the soral membrane and appear as powdery masses in open pustules (Walker, 1983). Zoospores and resting spores are unknown. False rust of *M. atropurpureum*, was first recorded near Jamundi, Colombia, in 1929 (Weston 1930). It was later observed causing considerable damage to cv. Siratro in central Brazil in the 1970s (Namekata *et al.*, 1974; Lenné, 1980a, 1994f). It has also been reported from Ecuador and Mexico (Lenné, 1990a). False rust is not known outside the native and naturalized habitat of *M. atropurpureum* in the Americas. It has, however, been found on closely related genera *Phaseolus* and *Vigna* in Africa, Australasia and Oceania (Walker, 1983). The known hosts of *S. phaseoli* include members of the *Leguminosae* only, including *Phaseolus acutifolius*, *P. lunatus*, *P. vestitus*, *Vigna calcarata* and *V. radiata* as well as *Macroptilium* spp. and *Rhynchosia minima* (Karling, 1964; Walker, 1983). As the above-mentioned *Vigna* spp. and *M. atropurpureum* were previously classified as *Phaseolus* spp. and another species of the pathogen *Synchytrium dolichi* (Cooke) Gäumann has been commonly recorded on cowpea, several other *Vigna* spp. and *Neonotonia wightii* in Africa and elsewhere (Allen, 1983; Lenné, 1990a), comparisons of the two *Synchytrium* spp. and cross-inoculation studies among *Phaseolus* and *Vigna* spp. and related genera would be of value.

Synchytrium spp. commonly occur on legumes and only limited work has been done on the genus as a whole since the earlier studies of Karling (1964). Many species appear to have been named for the hosts on which they were first found. Although limited attempts to inoculate specific *Synchytrium* spp. to legumes other than their hosts have not been successful to date, further studies

on the relationships between *Synchytrium* spp. on legumes are still needed especially where food, pasture and weedy legumes occur together. Detailed studies have been made on *Synchytrium psophocarpi* (Rac.) Gäum on winged bean (Drinkall and Price, 1979, 1986) which may be a useful guide for further studies on other legumes.

Symptoms

On *D. ovalifolium*, the disease first appears as small galls formed by abnormal displacement of epidermal tissues (Munasinghe, 1955). The galls are mainly formed on very young leaf and petiole tissue and on the flowering apex. Infection of leaflet midribs, veins and margins and petioles is common (Lenné, 1985). Normal shoot growth becomes arrested resulting in shortened internodes and leaflet and leaf deformation and plants develop a rosetted appearance (Fig. 13.5). Severely infected stems and seedlings often die and flowering and seed production are reduced. Adult plants generally regrow healthy tissue even after severe attack (Lenné, *et al.*, 1990b). Yellow-orange to brown galls form on all affected tissues.

On *D. intortum*, false rust is manifest as bright, lemon-yellow galls filled with dry, powdery sporangia on both upper and lower leaflet surfaces, petioles and stems. Leaf deformation and distortion is generally not as severe as that caused by *S. desmodii* on *D. ovalifolium*. On *M. atropurpureum*, symptoms usually appear

Fig. 13.5. Rosette symptoms on *Desmodium ovalifolium* caused by *Synchytrium desmodii* (Photo: courtesy of J.M. Lenné).

first on the lower leaflet surface as small, whitish, raised spots (Lenné, 1980a, 1994f). These enlarge to form bright orange galls which may turn light brown with age. Some galls develop into sporangial galls or sori which delimit sporangia within the sorus wall. Galls also form abundantly on petioles, stems and occasionally on pods (Lenné, 1980a, 1994f). These galls may coalesce to form large confluent masses which cause stem deformation, defoliation and stem and plant death, as observed in Ecuador in 1979 (Lenné, 1980a). Diseased seedlings of *M. atropurpureum* usually wilt, become defoliated and die.

Epidemiology

In Sri Lanka, false rust of *D. ovalifolium* is confined to humid environments (Munasinghe, 1955). The heavy shade canopy of rubber foliage encourages severe disease development due to prolonged high humidity. In the eastern plains of Colombia, false rust is most severe during May to August when rain showers are common and relative humidity greater than 90% (Lenné, 1985). False rust development on *M. atropurpureum* is also favoured by high humidity, and transmission of sporangia is probably facilitated by wind or rain-splash (Walker, 1983). Nametaka *et al.* (1974) recommended that Siratro should not be grown in humid areas which favour disease development.

Free water and high humidity are essential for infection (Price and Lenné, 1988). In glasshouse tests, a minimum of 4 h of leaf wetness was necessary for infection of *D. ovalifolium* by *S. desmodii* (Price and Lenné, 1988). Drinkall and Price (1986) noted that a minimum of 12 h of leaf wetness was necessary for infection of winged bean by *S. psophocarpi* in Papua New Guinea. The optimum temperature for zoospore liberation in *S. desmodii* ranged from 20 to 30°C and liberation occurred following 1.5 h incubation at 24–28°C when infected material containing recently opened galls was pre-soaked overnight (Price, 1987). In contrast to other *Synchytrium* spp. on legumes, zoospore release in *S. desmodii* occurs under natural conditions from attached sporangia within the gall in water droplets which accumulate through guttation, dew or rain (Price, 1987). It is probable that released zoospores move in water films on the plant surfaces and re-infect the same plant or move by raindrop splash to adjacent plants (Price, 1987). For both *S. desmodii* and *S. psophocarpi* only very young tissue is susceptible to infection by zoospores (Drinkall and Price, 1986; Price and Lenné, 1988). The ease of extraction of resting sporangia from tissues of *D. ovalifolium* suggests that resting sporangia may be dispersed by wind and by animals.

The *Synchytrium* spp. which cause false rust of legume foliage are essentially splash-dispersed, aerial pathogens of humid tropical environments. In this respect, they are ecologically different to the potato wart pathogen *Synchytrium endobioticum* (Schilb.) Perc. of temperate regions which is a soil inhabitant (Hooker, 1981). False rust fungi of legumes most probably survive in plant debris in soil and on perennial hosts (Lenné, 1985; Lenné and Trutmann, 1994). Resting spores of *Synchytrium* spp. may survive for up to 20 years in soil and plant debris (Walker, 1983). The survival potential of *S. desmodii*, which produces resting spores, is considerably greater than that of the other two *Synchytrium* spp. discussed.

Losses

False rust caused by *S. desmodii* reduces seedling survival, recruitment to the adult plant population and soil seed reserves of *D. ovalifolium* (Fig. 13.6) (Lenné *et al.*, 1990b). Under intermittently flooded conditions, adult plant yield of *D. ovalifolium* was reduced by 72.5%. The productivity and persistence of *D. ovalifolium*-based pastures is seriously reduced by false rust. Although *S. citrinum* has not been recorded in pastures of *D. intortum*, it has potential to cause severe disease. Although yield loss has not been quantified, the disease devastates stands of Siratro in Brazil and Ecuador (Namekata *et al.*, 1974; Lenné, 1980a, 1994f; Lenné and Sonoda, 1985) and severely affects seed production in Brazil (Namekata *et al.*, 1974).

Management

In Colombia, evaluation of a germplasm collection of 70 accessions of *D. ovalifolium* from South-east Asia identified CIAT 13089 from Thailand as having valuable adult plant resistance (Lenné *et al.*, 1990b). Accessions with erect growth habit were less affected than semi-prostrate to prostrate accessions which were exposed to standing water and inoculum for longer periods than erect accessions. Treatment of seed with concentrated sulphuric acid for 5 min failed

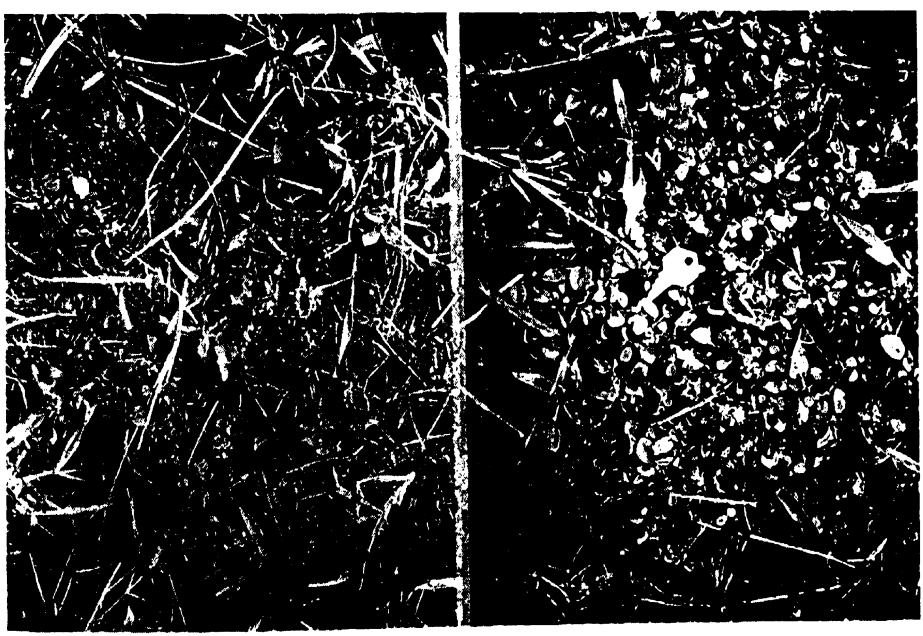

Fig. 13.6. Mortality of seedlings of *Desmodium ovalifolium* caused by *Synchytrium desmodii* (Photo: courtesy of J.M. Lenné).

to kill resting spores of the fungus. Strict quarantine should be practised to prevent further spread of *S. desmodii*. In addition, in regions such as Australia and South-east Asia, where *D. intortum* is an important pasture legume, strict quarantine should be enforced to avoid chance introduction of *S. citrinum* (Lenné and Stanton, 1990). Although *M. atropurpureum* is most important as a pasture legume in Australia and Florida where false rust does not occur, some insurance against future introduction of the pathogen would be worth while (Lenné and Sonoda, 1985; Lenné, 1994f). Germplasm collections or, at the least, elite lines from breeding programmes, could be evaluated for resistance to false rust in collaboration with institutions in Central and South America where this disease is common.

RUST

Numerous rusts, especially species of *Phakopsora*, *Puccinia* and *Uromyces*, have been recorded on tropical pasture legumes (Lenné, 1990a; Lenné and Trutmann, 1994). Rusts of the same genera and, in some cases, the same species, have also been recorded on crop legumes (Allen, 1983; see Chapters 2, 3, 4, 7, 8, this volume). For example, rust of common bean, *Uromyces appendiculatus* (Pers.) Unger (see Allen *et al.*, Chapter 4, this volume), has also been recorded on *Centrosema pubescens* and *Lablab purpureus*, while rusts of soyabean, *Phakopsora pachyrhizi* H. Sydow & Sydow and *P. meibomiae* (Arthur) Arthur (Ono *et al.*, 1992; see Sinclair, Chapter 3, this volume), have been identified on *Aeschynomene americana*, *C. pubescens*, *Desmodium* spp., *L. purpureus*, *Macroptilium atropurpureum*, *Neonotonia wightii* and *Pueraria phaseoloides* (Lenné, 1990a). Rust of groundnut (see McDonald *et al.*, Chapter 2, this volume) affects forage *Arachis* species and very similar rusts affect other tropical pasture legumes. With the exception of rusts affecting *Macroptilium atropurpureum*, *Stylosanthes* spp., *Arachis* spp. and *Zornia* spp., very limited information is available on rusts of tropical pasture legumes. Although the following discussion is unfortunately limited to few rusts, it must be emphasized that further work is needed to understand the relationships between rusts which affect both crop and pasture legumes and, especially, the disease risks associated with growing susceptible legumes together in the same cropping systems.

Aetiology and Biology

For many years, rust of *M. atropurpureum* cv. Siratro was considered to be caused by *Uromyces appendiculatus* (Pers.) Unger, which affects *Phaseolus* bean globally (see Allen *et al.*, Chapter 4, this volume). However recent comparative studies of isolates from common bean and Siratro from Australia and South America found significant differences between the isolates (Code *et al.*, 1985; Irwin, 1988). Uredospores of Siratro rust were similar in size to those of bean rust being 24–28 × 20–23 µm but their walls were significantly thicker (2.1–2.4 µm) than those of the bean pathogen (1.3–1.7 µm) and showed more closely spaced echinulae

(Irwin, 1988). *U. appendiculatus* is reviewed in detail in Allen *et al.*, Chapter 4, this volume.

In contrast to the common occurrence of the telial stage of bean rust, the telial stage of Siratro rust has been observed in Mexico only. Teliospores were similar to those of bean rust, except that the side walls were thicker (3.4–3.8 μm versus 2.1–3.2 μm) and darker than those of bean rust, with lineal rows of warts over the entire surface, a conspicuous channel in the base, thinner hyaline apical thickening and more broadly attached pedicels (Irwin, 1988). Siratro rust was described as a distinct variety var. *crassitunicatus* by Irwin (1988). *Uromyces appendiculatus* var. *crassitunicatus* is an heterothallic, obligate parasite specific to *M. atropurpureum*.

U. appendiculatus var. *crassitunicatus* is widely distributed on *M. atropurpureum* (Sonoda, 1983; Lenné and Sonoda, 1985). It was first recorded on *M. atropurpureum* in 1910 (Sydow and Sydow, 1910) and has since been found on this host including Siratro in Guatemala (Guyot, 1957), Florida (Sonoda and Kretschmer, 1982), Australia (Jones, 1982), Bolivia, Brazil, Colombia, Ecuador, Peru, Venezuela, Central America and the Caribbean (Lenné, 1990a). It is a common disease of roadside populations of *M. atropurpureum* in Colombia (Lenné and Sonoda, 1985). Whereas *Uromyces appendiculatus* has a wide host range within the tribe *Phaseoleae* including *Phaseolus* and *Vigna* spp., e.g. common bean, lima bean, scarlet runner bean and cowpea (Holliday, 1980; see Allen *et al.*, Chapter 4, this volume), var. *crassitunicatus* is presently only known from *M. atropurpureum*. Although var. *crassitunicatus* from Siratro produced small, sporulating uredia on the bean rust differential Golden Gate Wax, all members of the bean rust differential set are classified as resistant to this rust (Irwin, 1988). As no other host range studies have been reported, it would be worth while to carry out further studies with isolates of var. *crassitunicatus* from the Americas and other species within the *Phaseoleae*.

On beans, *Uromyces appendiculatus* is known as a highly variable pathogen (Ballantyne, 1974; Staveley and Pastor-Corrales, 1989; Allen *et al.*, Chapter 4, this volume). Knowledge of the race structure of var. *crassitunicatus* is essential to selecting resistant lines of *M. atropurpureum* and breeding for stable resistance. *U. appendiculatus* uredospores collected from infected *M. atropurpureum* plants at 19 sites throughout southern Mexico were inoculated onto Siratro (Sonoda and Kretschmer, 1990). All 19 isolates produced rust pustules. Siratro was provisionally designated a universal host of *U. appendiculatus* var. *crassitunicatus*.

Previous studies in Australia indicated that some strains of *U. appendiculatus* var. *crassitunicatus* exhibited differential virulence (Bray, 1988). In a more recent study, ten isogenic lines of *M. atropurpureum*, all selected for resistance to an Australian isolate of var. *crassitunicatus* and carrying resistance genes from diverse geographic origins, were developed by backcrossing to susceptible cv. Siratro, subsequent selfing and then screening against one isolate from Australia, two from Florida and seven from Mexico (Bray *et al.*, 1991). All lines were resistant to the isolates from Australia and Florida, and six lines were resistant to all seven isolates from Mexico. Each isolate from Mexico produced a susceptible reaction on at least one line. Three resistant lines with genes from accessions from Oaxaca, Mexico, gave susceptible reactions to two isolates from that area.

Although races of *U. appendiculatus* var. *crassitunicatus* have not been formally proposed, these results indicate the existence of pathogenic variation.

The underlying genetic differences between bean and siratro rusts were assessed using molecular methods (Davis and Irwin, 1994). Total DNA hybridization was used to obtain an overall assessment (Braithwaite *et al.*, 1991). Nucleotide sequence homology between bean and siratro rusts was poor. This was further quantified using restriction fragment length polymorphism (RFLP) analysis with random genomic clones. Only 7.6% of bands were common, indicating wide genetic differences. These results were also subsequently confirmed by RFLP analysis with cDNA clones (Braithwaite *et al.*, 1991).

Rust of *Stylosanthes* spp. is caused by *Puccinia stylosanthis* Viegas (Viegas, 1945). Uredospores, produced in red-brown, discrete, rust sori, are yellow-brown, globose to broadly ellipsoid, 18–25 × 20–30 µm with two, rarely three, equatorial to subequatorial pores and very finely echinulate (Viegas, 1945; Lenné and Sousa Costa, 1985). Teliospores are two-celled, oblong to ellipsoid, 30–45 × 20–25 µm with walls 2–4 µm thick, being slightly thicker at the apex, and with hyaline pedicels up to 30 µm long (Lenné and Sousa Costa, 1985). Similar measurements are given for *P. arachidis* on groundnut (see McDonald *et al.*, Chapter 2, this volume). Teliospores on *Stylosanthes* spp. vary in abundance from very common to rare in sori on rusted leaves.

The name universally accepted for rust of *Arachis* spp. including groundnut is *Puccinia arachidis* Speg. (Allen, 1983; Subrahmanyam and McDonald, 1983; Porter *et al.*, 1984) but the fungus is not regarded as a *Puccinia* by Hennen and Buritica (1993). On cultivated groundnut, the conidial anamorph *Peridipes arachidis* occurs widely but the teleomorph has been found only rarely, most commonly on wild *Arachis* spp. in Brazil. Its actiology and biology have been reviewed in detail in McDonald *et al.*, Chapter 2, this volume. Two rusts have been described on *Zornia* spp.: *P. offuscata* (Authur) Cummins and *P. zorniae* (Berk.) McAlp. From published descriptions and specimens from the International Mycological Institute, Egham, UK, the *Puccinia* spp. found on related legume genera *Stylosanthes*, *Arachis* and *Zornia* of the subtribe *Stylosanthinae* appear to be very closely related (Cummins, 1978; Hennen *et al.*, 1987; Lenné, 1994a, c; B.C. Sutton, IMI, Kew, UK, 1984, personal communication). As was observed in McDonald *et al.*, Chapter 2, this volume, the three host genera frequently occur together in Brazil and *Zornia* spp. are common weeds in *Stylosanthes*- and forage *Arachis*-based pastures in tropical America. Cross-inoculation studies are needed to confirm the true status of these taxa.

Rust is common in native populations of *Stylosanthes aurea*, *S. guianensis*, *S. macrocephala* and *S. viscosa* on the central Brazilian plateau (Lenné and Sousa Costa, 1985) and on *S. fruticosa* in East and West Africa (Lenné, 1990a). During the 1980s, rust severely affected native stands of *S. aurea* near Diamantina and *S. macrocephala* near Pirapora, Minas Gerias, Brazil. The severity of rust in native *Stylosanthes* populations suggests that it is a potentially serious disease of sown pastures of *Stylosanthes* spp. although it has not yet been reported in such agroecosystems. As observed by Hennen (see McDonald *et al.*, Chapter 2, this volume), rust is also common on native *Arachis* spp. populations in Brazil and has been observed in experimental plantings of *A. glabrata* in Brazil (Lenné, 1994c;

Kerridge and Hardy, 1994). It has not, however, been noted on the more commonly grown forage species *A. pintoi* (Lenné, 1990a). Rust caused by *P. arachidis* Speg. var. *offuscata* (Authur) Cummins has been found on native populations of several *Zornia* spp. in humid areas of Central and South America and the Caribbean (Lenné, 1990a), while *P. zorniae* (Berk.) McAlp. has been found on *Zornia* spp. in West Africa (Lenné, 1990a, 1994c). Very limited studies have been carried out on the *Puccinia* spp. on *Zornia*.

Symptoms

On *M. atropurpureum*, *U. appendiculatus* var. *crassitunicatus* manifests itself as minute, greenish-white, raised spots on the lower surface of Siratro leaflets which enlarge to form brown-red sori containing uredospores (Fig. 13.7) (Sonoda, 1975, 1976). Fully expanded young leaves were the most susceptible while unexpanded young and mature leaves were less susceptible. Heavily infected leaflets and leaves become defoliated (Sonoda, 1975). The rust has not been reported on stems or pods (Lenné, 1994f).

Symptoms caused by *P. stylosanthis* on *Stylosanthes* appear as red-brown discrete rust sori, 1–5 mm in length, mostly on the lower surfaces of leaflets and on stems (Lenné and Sousa Costa, 1985). These rupture to expose masses of yellow-brown uredospores. Severe infection leads to leaf necrosis and considerable defoliation of some species such as *S. macrocephala*. Symptoms caused by *P. arachidis*

Fig. 13.7. Brown rust sori caused by *Uromyces appendiculatus* var. *crassitunicatus* on *Macroptilium atropurpureum* (Photo: courtesy of J.M. Lenné).

on forage *Arachis* spp. are very similar to those produced on groundnut (see McDonald *et al.*, Chapter 2, this volume) although pustules tend to be smaller (0.3–0.6 mm in diameter), and are more commonly on both leaf surfaces (Lenné, 1994c). On *Zornia* spp., symptoms appear as chlorotic spots on the adaxial leaf surface and dark brown pustules on the abaxial leaf surface and stems (Lenné, 1994c). Chlorotic patches may become necrotic as pustules and uredospores mature.

Epidemiology

The apparent lack of a telial stage in most regions where *U. appendiculatus* var. *crassitunicatus* is common on Siratro suggests that infection and survival from season to season depends mostly on the uredospore stage. This is in contrast to *U. appendiculatus* on *Phaseolus* bean where telia are common (Allen, 1983). Uredospores germinate to produce germ tubes which develop appressoria on contact with host stomata (Wynn, 1976; Lenné, 1994f). An infection peg develops from the appressorium, pushes between the guard cells and fungal cytoplasm is transferred into the substomatal vesicle (Wynn, 1976). Haustorial mother cell development is stimulated by contact of the infection hypha with a host cell. Once the host cell is penetrated, intercellular hyphae ramify through the host tissue.

Rust of Siratro is most damaging during cooler, spring and autumn months and least damaging in the wet summers in Florida and Australia (Sonoda, 1975, 1976; Jones, 1982). This is in contrast to soyabean rust (*Phakopsora* spp.) which is most serious under wet conditions (Allen, 1983; see Sinclair, Chapter 3, this volume). Optimum temperature conditions for maximum disease development for Siratro rust tend to be slightly higher than those for bean rust (Code *et al.*, 1985). Moisture is required for uredospore germination but is of less importance after penetration of the host. Day length and light intensity were found to be important for development of bean rust (Harter and Zaumeyer, 1941; Augustin *et al.*, 1972) but have not been studied for Siratro rust (Lenné, 1994f). Optimal conditions for bean rust development and survival have been reviewed in Stavely and Pastor-Corrales (1989) and Allen *et al.*, Chapter 4, this volume. The infection process results in development of uredia which may mature within 14 days (Mendgen, 1973). Uredospore production and release are influenced by moisture and temperature. Spore production increases under conditions of high humidity.

Wind-distributed uredospores of *Puccinia stylosanthis* which contact wet leaf surfaces germinate and produce appressoria. Infection hyphae penetrate the host through stomata. Infection is favoured by conditions of high humidity and moderate temperatures (18–25°C). The incubation period varies from 9 to 20 days, being greatly influenced by environmental conditions. The natural occurrence of rust on *Stylosanthes* spp. appears to be restricted to the medium-altitude tropics in both the Americas and Africa (J.M. Lenné, CIAT, Colombia, 1983–1987, personal observations). Rust may not be a problem in the tropical lowlands where most *Stylosanthes*-based pastures are grown. However, because of the potential devastating effect of rust, care must be taken to ensure that it is not moved intercontinentally. Because rust is presently restricted to natural populations of

Stylosanthes spp., few germplasm collections have been screened against it. It would therefore be worthwhile to evaluate existing collections of promising species in the savannahs of Brazil for rust resistance.

The epidemiology of rust on groundnut has been described in detail in McDonald *et al.*, Chapter 2, this volume, and further information is available in Subrahmanyam and McDonald (1983) and Porter *et al.* (1984). No information is available on the epidemiology of rusts on *Zornia* spp. The spread of all rusts is facilitated mainly by wind movement, rain-splash, contact between plants, and vectors such as insects and grazing animals. Long-distance dissemination may occur through airborne uredospores, by movement of infected debris and vegetative planting material, or by movement of pods and seeds contaminated with uredospores. Uredospores of all rusts may overwinter in debris of host plants as well as on their perennial hosts. Perennial hosts probably provide primary as well as secondary inoculum in most areas where these rusts are found.

Losses

In Queensland, Australia, 30% reduction in yield of Siratro was recorded, with associated lowered digestibility and nitrogen content during 2 years, due to rust (Jones, 1982). Rust also reduces the subsequent vigour of affected plants (Sonoda, 1975, 1976) and seed yields by 25% through a reduction in inflorescence numbers (English and Hopkinson, 1983). Jones (1982) concluded that rust has the potential to reduce animal production from Siratro-based pastures over large areas of subtropical and tropical Australia. Cameron (1985a) described rust as the most serious disease affecting Siratro. Groundnut rust can reduce yield of susceptible cultivars of groundnut by over 50% (Lenné, 1994c; see McDonald *et al.*, Chapter 2, this volume) but losses caused by rust on *A. glabrata* in Brazil have not been reported. No quantitative information is available on losses caused by rusts on *Stylosanthes* and *Zornia* spp.; however, considerable defoliation of natural stands of several *Stylosanthes* spp. have been observed in Brazil (Lenné and Sousa Costa, 1985).

Management

There is considerable potential for controlling rust through selection and breeding for resistance. Although early screening of *M. atropurpureum* lines, including Siratro, in southern Florida failed to identify adequate resistance (Sonoda, 1976), later screening of 104 introductions found 18 from Mexico and two from Colombia to be highly resistant to rust (Sonoda and Kretschmer, 1982; Sonoda, 1983). Resistant accessions included broad-leafleted types similar to Siratro, and others with narrow leaflets which were considered less productive than Siratro (Sonoda and Kretschmer, 1982). Furthermore, of over 100 accessions of *M. atropurpureum* collected from Colombia and Mexico and evaluated in Santander de Quilichao and Palmira, Colombia, during 1978 to 1988, more than 50% showed field resistance to local strains of rust (Lenné, 1994f). This suggests that

germplasm of *M. atropurpureum* from Colombia and Mexico may be useful parents in breeding programmes.

Sixteen accessions of *M. atropurpureum* showed a range of reaction types when seedlings were inoculated with a single-uredospore isolate of *U. appendiculatus* cv. *crassitunicatus* (Bray, 1988). Analysis of F_2 families of crosses with susceptible Siratro showed that resistance was dominant in seven accessions and regulated by a single locus in four of these but by more than one locus in three other accessions. At least three of these, loci were identified as non-allelic. In one accession, resistance was near-recessive and regulated at a single locus, while combinations of dominant and recessive alleles at different loci explained the segregation of resistance in other accessions. Many accessions were multigenic in response to rust, some with more than one dominant gene and others with additional minor effect genes (Bray, 1988). There was some evidence that the expression of resistance could be temperature sensitive (Bray, 1988).

Quantitative histological studies of the infection process of *U. appendiculatus* cv. *crassitunicatus* on *M. atropurpureum* were made for one compatible interaction and five interactions showing varying degrees of incompatibility (Ogle *et al.*, 1988). Four histologically different resistant responses to infection were observed. CPI 91348 exhibited a response characterized by significantly fewer pustules per unit area and a longer generation time than the susceptible cv. Siratro. Significantly fewer uredospores produced appressoria on CPI 91348 than on Siratro. Resistance in CPI 84997 was associated with the development of large areas of necrotic tissue with 99.8% of colonies showing an indeterminant hypersensitive response. The remaining lines developed varying degrees of chlorosis only. Colonies in CQ 1382 and CPI 92643 grew only slowly and, as with CPI 84997, growth continued after necrotic tissue appeared (indeterminant hypersensitivity). In CQ 1398, colony growth ceased by 96–144 h after inoculation, coinciding with the appearance of necrotic tissue (determinant hypersensitivity) (Ogle *et al.*, 1988). The existence of four histopathologically different expressions of resistance to infection suggests that at least four different resistance genes may exist in the accessions studied (Ogle *et al.*, 1988). These may be useful for the development of a multiline or mixture with durable rust resistance.

High levels of resistance and, in some cases, immunity to rust, have been found in wild *Arachis* spp. (Subrahmanyam *et al.*, 1983). If there is need to develop rust-resistant forage *Arachis* spp., the potential for selecting for resistance appears high. As weeds may encourage disease development due to higher humidity in the canopy, choice of associate grass in forage *Arachis* spp. pastures may be important.

SCAB

There are many records of scab caused by *Elsinoe* spp. on tropical pasture legumes (Lenné, 1990a) and crop legumes (see Allen, 1983; Allen *et al.*, Chapter 5, this volume), but few have been investigated (Holliday, 1980). Some have been identified only to genus. With the exception of the scab pathogens affecting

Arachis spp., *Lablab purpureus* and *Zornia* spp., very limited information is available for tropical pasture legumes. Species of *Sphaceloma* and *Elsinoe* have been described more on the basis of host, pathogenicity and colony characters than on morphology (Allen, 1983). There is apparently considerable host specificity within scab pathogens (Emechebe, 1980; Phillips, 1996; see Allen *et al.*, Chapter 5, this volume); however, it is possible that some of the species are synonymous. Extensive cross-inoculation studies are needed to define *formae speciales*. In general, progress has been hampered by the relative difficulty of isolation and manipulation of *Elsinoe* spp. on artificial media. Much remains to be clarified in this group, whose economic importance is underrated (Allen, 1983).

Aetiology

Groundnut scab is caused by *Sphaceloma arachidis* Bitanc. and Jenk. (Bitancourt and Jenkins, 1940; Porter *et al.*, 1984; Lenné, 1994c) while scab of *Zornia* spp. is caused by *Sphaceloma zorniae* Bitanc. & Jenk. (Lenné, 1981, 1994c). *Elsinoe* states of these species have not been reported. Scab of *Lablab purpureus* is caused by *Elsinoe dolichi* Jenk., Bitanc. & Cheo (Cheo and Jenkins, 1945). In both East Africa and China, the *Sphaceloma* state is also associated with the teleomorph in diseased tissue and has been cultured in China (Cheo and Jenkins, 1945).

Biology

Groundnut scab was first observed in Brazil in 1937 (Bitancourt and Jenkins, 1940; Porter *et al.*, 1984). It was noted sporadically in Brazil and Argentina during the following 50 years and was observed on forage *Arachis glabrata* in Brazil and *A. pintoi* in Colombia in the mid-1980s (Lenné, 1994c). Scab is locally serious on groundnut in Argentina and Brazil (Porter *et al.*, 1984) and on the forage *A. pintoi* in Colombia (Lenné, 1994c). With the recent widespread promotion and dissemination of the susceptible *A. pintoi* CIAT 17434 (cv. Amarillo (Australia); cv. Mani Forrajero Perenne (Colombia); cv. Pico Bonito (Honduras)) in Central and South America (Kerridge and Hardy, 1994) and Australia, scab must be acknowledged as a serious threat. The host range of *Sphaceloma arachidis* has not been investigated in detail but includes *A. glabrata*, *A. hypogaea* and *A. pintoi*. On *Arachis* spp., acervuli of *S. arachidis* are amphigenous, numerous, effuse, erumpent, and 300 × 45 μm. Conidiophores are hyaline, globose and conical, in aggregations resembling a palisade, and 8–11 × 3–5 μm (Bitancourt and Jenkins, 1940; Porter *et al.*, 1984; Lenné, 1994c). Conidia are hyaline, mainly unicellular, and 9–17 × 2.5–3 μm. On agar media, at 22–25°C, the fungus grows slowly as convoluted, dark red colonies.

Scab occurs on *Zornia* spp. in forage evaluation sites and grazed pastures throughout subhumid and humid areas of Brazil, Colombia, Peru and Venezuela (Lenné, 1990a, 1994c). It is also common in native populations of *Zornia* spp. in Brazil and Colombia. Scab causes serious defoliation and dieback of *Z. latifolia* under humid conditions (Lenné, 1981; Thomas *et al.*, 1986). The known host

range of *S. zorniae* includes *Z. brasiliensis*, *Z. glochidiata*, *Z. glabrata*, *Z. latifolia* and *Z. reticultata* (Lenné, 1990a). On acidified potato dextrose agar colonies of *S. zorniae* are small, raised, crusty and slow growing, varying in colour from cream to reddish-brown (Lenné, 1981, 1994c). Conidia are one-celled, hyaline, ovoid to elliptical, 3.5–8.5 × 2–4.5 μm and produced sparsely on agar. They are borne on-one celled, cylindrical conidiophores, 4–9 μm long. Scab of *Lablab purpureus* has been reported from East and Southern Africa (Kenya, Uganda and Malawi) and China (Cheo and Jenkins, 1945; Peregrine and Siddiqi, 1972). In Uganda, cover crops of *L. purpureus* have been severely attacked and plants killed, while in Yunnan, China, all aerial parts of the plant are severely attacked and pod yield is substantially reduced. Although it has been assumed that the scab of *L. purpureus* reported in China is the same species as that reported in East Africa in 1930, it appears that only herbarium specimens have been compared (Cheo and Jenkins, 1945). *Elsinoe phaseoli* Jenkins is very widespread on legumes in Africa (see Allen *et al.*, Chapter 5, this volume). Cultural comparisons of single isolates of *E. dolichi* from *L. purpureus* and *E. phaseoli* from lima bean were made in China and both fungi were found to be distinct (Cheo and Jenkins, 1945). Extensive cultural comparisons of isolates of *Elsinoe batatas* Viegas from Asia and the Pacific revealed considerable variation within the species (Lenné *et al.*, 1994) suggesting that single isolate comparisons alone are not adequate to distinguish species of *Elsinoe*.

On *L. purpureus*, ascomata of *E. dolichi* are amphigenous, appearing as elevations in the dark stromatic tissue covering lesions or as separate, dark, punctate bodies, 60–300 μm in diameter and up to 100 μm high (Cheo and Jenkins, 1945; Lenné, 1994c). Asci are subglobose, pyriform to ellipsoid, 20–32 × 15–22 μm, and ascospores are one- to three-septate. Stromatic tissue may be covered with well-defined conidiophores which produce hyaline, spherical to elliptical conidia, 3–7.6 × 1.5–3 μm (Cheo and Jenkins, 1945).

Cross-inoculation studies are needed to clarify the taxonomic status of *Sphaceloma* spp. of legumes (Lenné, 1981) and their relationship with telemorphic *Elsinoe* spp. In the light of the close taxonomic relationship between the host genera *Arachis* and *Zornia*, it would be worth while to make detailed comparisons of the *Sphaceloma* spp. affecting them. In China, an isolate of *E. phaseoli* failed to infect *L. purpureus* while an isolate of *E. dolichi* infected only *L. purpureus*, failing to infect several legumes including lima bean (Cheo and Jenkins, 1945). Further studies of the relationship between the *Elsinoe* spp. reported on *L. purpureus* in different countries are needed.

Symptoms

All aerial parts of *Arachis* spp. including groundnut can be affected by *S. arachidis* (Bitancourt and Jenkins, 1940; Porter *et al.*, 1984; Lenné, 1994c). Scab first appears as numerous, circular or irregular, chlorotic lesions, up to 1 mm diameter. On leaflets, lesions on the upper surface are light tan and sunken with raised

dark margins while those on the lower surface are yellowish-red with brown margins. Lesions on veins increase to 2 mm in diameter and coalesce near the central vein. Leaflets become necrotic and curl upwards. On petioles and stems, lesions are oval, up to 3 mm in length, irregular, corky and cankerous (Fig. 13.8). Stem lesions coalesce and may cover the entire stem. Plant growth becomes distorted and stunted (Porter *et al.*, 1984; Lenné, 1994c). The most spectacular example of scab-induced distortion is 'superelongation' disease caused by *Sphaceloma manihoticola* Bitanc. & Jenk. on cassava (Zeigler, 1982). Elongation is caused by the production of gibberellic acid in the plant in response to the pathogen.

On *Zornia* spp., *S. zorniae* produces small, elliptical to elongate, pale brown spots on leaves, petioles, stems, inflorescences and pods (Lenné, 1981, 1994c). Lesions expand and coalesce to form raised, pale to reddish-brown, corky scabs, often involving entire petioles, extended lengths of stem and the inflorescence (Fig. 13.9). Defoliation and dieback are common. All aerial parts of *Lablab purpureus* may be affected by *E. dolichi*. Symptoms are described as light buff leaf spots with dark margins, along leaf veins especially the midrib (Cheo and Jenkins, 1945; Lenné, 1994c). When several veins are involved, general chlorosis may develop. Interveinal lesions may reach 4 mm in diameter. On stems, cankers are

Fig. 13.8. Scab lesions on *Arachis pintoi* caused by *Sphaceloma arachidis* (Photo: courtesy of J.M. Lenné).

Fig. 13.9. Scab lesions on *Zornia latifolia* caused by *Sphaceloma zorniae* (Photo: courtesy of J.M. Lenné).

circular to linear. They may be numerous and scattered, grouped or coalescent. Individual lesions range from minute spots to cankers 3 mm × 1 cm. They may be flat or slightly depressed, brownish-grey, with dark margins. Pod lesions are 5 mm in diameter, scattered or grouped or coalescent, and brown to purplish-brown with light centres (Cheo and Jenkins, 1945).

Epidemiology

Groundnut scab occurs under both dry and humid conditions; however, fructification is favoured by high humidity (Porter *et al.*, 1984). The major source of infection for *Arachis pintoi* is the perennial crop itself. The common practice of vegetative propagation of pastures by stolons helps to spread scab (Lenné, 1994c). The possibility of seed transmission in South America in groundnut has been noted (Porter *et al.*, 1984) but not for forage species. Prolonged high humidity favours disease development in *Zornia* spp. (Lenné, 1981, 1994c). Although scab on perennial *Zornia* spp. is probably the main source of inoculum for infecting seedlings and healthy adult plants, *S. zorniae* is also seedborne especially with pod debris (Lenné, 1981). Seed could be an important means of transmitting scab to new areas. No information was found on the epidemiology of *E. dolichi*

Oestrogenic activity has been recorded in various forage legumes infected by foliar pathogens. A mouse bioassay indicated that foliage of *Z. latifolia* affected with *S. zorniae* showed oestrogenic activity in contrast to healthy foliage (Lenné *et al.*, 1990c). Forages containing oestrogens can cause infertility in female

animals and weight gains in castrated male animals. There is a need to investigate this possibility in forage *Arachis* spp. and *L. purpureus*.

Losses

No quantitative information is available on the effect of scab on any of the pasture legumes mentioned. As scab causes defoliation and dieback in pastures, dry matter losses could be serious under conducive conditions.

Management

The principal method for control of scab in tropical pasture legumes is resistant cultivars. In both *Arachis* and *Zornia* spp., field screening has identified accessions which appear resistant to scab. Sources of resistance to *S. arachidis* in groundnut have also been identified (Porter *et al.*, 1984) which could be used in breeding programmes with other *Arachis* spp.

LITTLE LEAF

Aetiology

The association of wall-less prokaryotes with plants is manifest as a range of diseases such as yellows, little leaf, phyllody, witches' broom, proliferation, stunting, etc. on a variety of crops and pastures of agricultural importance (Arora and Sinha, 1988). Little leaf of tropical pasture legumes is caused by polymorphic, phytoplasma-like organisms (PLOs) which vary in form from simple rounded bodies to filaments, usually less than 1 μm in diameter, bound by a single-unit membrane and containing ribosomes and DNA-like fibrils (McCoy *et al.*, 1983). Their presence in the sieve tubes of the phloem in affected *Desmodium ovalifolium*, *Macroptilium atropurpureum*, *Stylosanthes gracilis* and *Neonotonia wightii* in China (Chen *et al.*, 1982) and Brazil (Chagas and Oliveira, 1986; Kitajima *et al.*, 1987) has been confirmed. Recent studies on relationships between PLOs from different hosts have been reviewed in Mercer, Chapter 12, this volume, but none of the PLO diseases of tropical pasture legumes were included. Witches' broom of pigeonpea is also caused by PLOs (see Reddy *et al.*, Chapter 10, this volume).

Biology

Little leaf is common on tropical pasture legumes throughout the tropics. It has been recorded on at least ten *Desmodium* spp. in Australia, tropical America, Asia (McCoy *et al.*, 1985; Lenné and Stanton, 1990) and the Pacific (Jackson and Zettler, 1983) and also affects species of *Centrosema* (at least six species), *Macroptilium atropurpureum* and *Stylosanthes* (at least eight species) to varying

degrees in the same regions (Hutton and Grylls, 1956; Lenné and Calderon, 1984; Lenné and Sonoda, 1985; Lenné *et al.*, 1990a; Lenné and Trutmann, 1994). In spite of its frequent and often serious nature, the relationships between the PLOs on different pasture legume hosts have not been studied.

Symptoms

Symptoms develop initially on all legumes as interveinal chlorosis accompanied by leaf deformation (Lenné and Stanton, 1990; Lenné and Trutmann, 1994). Proliferation of shoots and leaves occurs from the axilliary buds while shortening of internodes and stunting is common (Fig. 13.10). The proliferation of buds, leaves and branches increases progressively and plants become chlorotic and stunted. Flowering and fruiting cease on affected parts and plants with severe little leaf die within 1 to 2 years. Plants affected by little leaf frequently become chlorotic. This is due to the presence of the PLOs interfering with the legume's symbiotic relationship with rhizobia which reduces the number, size and effectiveness of nodules (Joshi and Carr, 1967; O'Rourke, 1976). The ability of the affected plant to fix nitrogen is thus impaired.

Fig. 13.10. Proliferation of leaves and shortening of internodes caused by little leaf (phytoplasma-like organisms) on *Desmodium* spp. (Photo: courtesy of J.M. Lenné).

Epidemiology

Almost all PLOs causing little leaf diseases are disseminated by leafhoppers of the family *Cicadellidae* in a persistent manner and the pathogen undergoes multiplication in both vector and host plant (Arora and Sinha, 1988). Different genera and species have been recorded in different countries (Lenné and Stanton, 1990). The leafhopper *Orosius argentatus* Evans has been confirmed as the vector of little leaf of *Stylosanthes* spp. in Australia (Hutton and Grylls, 1956) and legume little leaf in the Solomon Islands (Jackson and Zettler, 1983). Little leaf incidence and severity varies greatly from site to site and from season to season (Lenné and Stanton, 1990). Spatial and temporal variation in the incidence and severity of little leaf could be due to variable leafhopper activity related to seasonal conditions and perhaps different species of leaf hoppers.

Losses

Severe little leaf has been observed in small plot trials of *Desmodium* and *Centrosema* spp. in Colombia and Brazil (Kitajima *et al.*, 1987; Lenné and Stanton, 1990; Lenné *et al.* 1990a). Its incidence in grazed pastures, however, is usually low. Under field testing in southern Queensland in the 1950s, *Stylosanthes erecta*, *S. fruticosa* and *S. montevidensis* were seriously affected and plants were killed (Hutton and Grylls, 1956). Eighty per cent of accessions of *S. scabra* under evaluation in Central Brazil in the late 1970s were killed by little leaf (Lenné and Calderon, 1984). Little leaf, however, is regarded as a minor disease of *M. atropurpureum* (Hutton, 1962; Skerman, 1977; Chen *et al.*, 1982; Lenné and Sonoda, 1985). No quantitative assessment of losses in any tropical pasture legume has been documented.

Management

Observations on the reaction of different species of *Desmodium* to little leaf are contradictory. In Australia, Hutton and Grylls (1956) reported that *D. uncinatum* was highly susceptible while *D. intortum* was highly resistant. In a subsequent study, however, Imrie (1973) found 34 accessions of *D. intortum* to be susceptible to little leaf. In Brazil, *D. uncinatum* was more susceptible than *D. intortum* (Oliveira, 1979). Throughout Central and South America, slight to severe little leaf has been observed periodically in accessions of *D. barbatum*, *D. gyroides*, *D. heterocarpon*, *D. heterophyllum*, *D. incanum*, *D. ovalifolium* and *D. strigillosum* (Lenné and Stanton, 1990). Because of the lack of controlled screening, present information is insufficient to declare any species of *Desmodium* resistant. It should be noted that attempts to select for resistance to little leaf in any crop have not yielded much success (Jackson *et al.*, 1984).

ROOT-KNOT

Aetiology and Biology

The most common nematode pest of tropical pasture legumes, *Meloidogyne* spp., is the causal agent of root-knot (Stanton, 1994). *Meloidogyne* spp. are distributed worldwide and affect many hundreds of plant species. Further information can be found in McDonald *et al.*, Chapter 2, this volume. The main tropical pasture genera affected include *Aeschynomene*, *Calopogonium*, *Cassia*, *Centrosema*, *Desmodium* and *Dolichos* (Stanton, 1994). There are over 40 species of *Meloidogyne* although four, *M. javanica* (Treub) Chitwood, *M. arenaria* (Neal) Chitwood, *M. incognita* (Kofoid & White) Chitwood and *M. hapla* (Chitwood), cause about 95% of the damage worldwide (Taylor and Sasser, 1978).

The disease cycle commences when second-stage juveniles hatch from eggs, move through the soil and invade roots near the root tip (Stanton, 1994). Juveniles then become sedentary and feed on nurse cells, thus establishing a specialized host–parasite relationship with the plant. Developing nematodes become flask-shaped and moult three times to become adults. In most *Meloidogyne* spp., males are rare and reproduction occurs by parthenogenesis. Mature females lay hundreds of eggs in an egg mass on the root surface and these eggs hatch in warm, moist soils to continue the life cycle. The length of the life cycle is temperature-dependent but, under ideal conditions, takes only 4–6 weeks. Identification of *Meloidogyne* is difficult, inaccurate and time-consuming using current methods and several researchers are now developing molecular methods of differentiating species and races of *Meloidogyne* (Powers and Harris, 1993; Hugall *et al.*, 1994; see McDonald *et al.*, Chapter 2, this volume).

Meloidogyne spp. are generally important on *Desmodium* spp. although most species vary greatly in their resistance (Hernández and López, 1985; Lenné and Stanton, 1990; Stanton, 1994). *Desmodium gyroides* and *D. ovalifolium* were more susceptible while *D. intortum* and *D. heterophyllum* were resistant to *M. javanica* (Lenné, 1981). *D. intortum*, *D. uncinatum*, *D. barbatum*, *D. heterocarpon*, *D. ovalifolium* and *D. strigillosum* varied in their responses to *M. arenaria* race 1, *M. incognita* race 3 and *M. javanica* from highly resistant to highly susceptible (Quesenberry and Dunn, 1987). Infected *D. ovalifolium* plants become stunted, chlorotic and wilted; some become defoliated and die (Lenné, 1981). Some accessions of *D. heterocarpon* were resistant to *M. javanica* (Stanton and Hernández, 1986). However, Stanton and Hernández (1986) found that the above-ground appearance was an unreliable guide to the extent of root galling of *D. heterocarpon* infected by *M. javanica*. This suggested that tolerance, the ability of the plant to yield well in the presence of the nematode, was also important in controlling yield loss (Stanton, 1994).

Centrosema spp. are generally resistant to *Meloidogyne* spp. Lenné (1981) showed that a *Centrosema* hybrid was slightly susceptible to *M. javanica* but various accessions of *C. pubescens*, *C. macrocarpum* and *C. pubescens* × *C. macrocarpum* were immune to *M. javanica* while others were less resistant (Sharma *et al.*, 1985). However, *M. javanica* causes unthrifty growth, chlorosis, necrosis and death of *C. pascuorum* in Brazil. None of the 12 accessions tested flowered and all senesced prematurely (Sharma and Grof, 1988). *Centrosema pubescens* is moderately

resistant to *M. incognita* (Thankamony *et al.*, 1989). Also, *C. pubescens* and *C. acutifolium* developed a few small galls but did not support reproduction of *Meloidogyne arabicida* Lopez & Salazar although *C. macrocarpum* was a host (Domínguez-Valenzuela *et al.*, 1990). *Meloidogyne* spp. are considered to be minor pathogens of *M. atropurpureum* cv. Siratro (Lenné and Sonoda, 1985). *Macroptilium* spp. are generally slightly susceptible to *M. javanica* (Lenné, 1981) but *M. atropurpureum* is a poor host of *M. incognita* so is used as a cover crop for nematode control in black pepper plantations in the Amazonian region (Koshy and Bridge, 1990). However, the top growth of *M. atropurpureum* cv. Siratro may be reduced by infestation with several *Meloidogyne* spp. (Lynd and Ansman, 1989). On sandy and other friable soils in Queensland, Australia, *Meloidogyne* spp. caused severe galling of the roots of *M. lathyroides* and reduced plant vigour (Cameron, 1985b).

Stylosanthes spp. are generally resistant to *M. javanica* (Lenné, 1981), including *S. humilis* (Minton *et al.*, 1967), *S. capitata*, *S. macrocephala* and *S. guianensis* (Sharma, 1984), although *S. gracilis* is a good host of both *M. javanica* and *M. incognita* (Netscher and Sikora, 1990). *S. humilis* (Minton *et al.*, 1967), *S. scabra*, *S. hamata* and *S. viscosa* (Azmi, 1985b) are moderately resistant to *M. incognita*; however, *S. humilis* is susceptible to *M. hapla* and *M. arenaria* (Minton *et al.*, 1967) and *S. guianensis* to *M. incognita*. Rhizobium nodulation in all these species was reduced by 20% by *M. incognita* and plant growth was also reduced (Azmi, 1985b).

Symptoms

The presence of nematodes in the root stimulates the surrounding tissues to enlarge and produce the galls which are the typical symptom of infection by the nematode (Stanton, 1994). Galling restricts root volume and hinders the normal translocation of water and nutrients within the plant so that plants exhibit above-ground symptoms of stunting, wilting and chlorosis, consistent with water stress and nutrient deficiency (Fig. 13.11). Damage caused by the nematode also pre-disposes plants to attack by other soilborne pathogens, particularly fungi and bacteria. The end result of attack by root-knot nematode is yield loss.

Epidemiology

For several reasons, plant-parasitic nematodes are generally more damaging in the tropics (Stanton, 1994). Higher temperatures and humidity are more favourable to most species while longer growing seasons allow more life cycles and, therefore, greater population increase.

Losses

It is very likely that significant yield losses due to root-knot nematodes occur in tropical pasture legumes. However, because of the relative lack of nematological

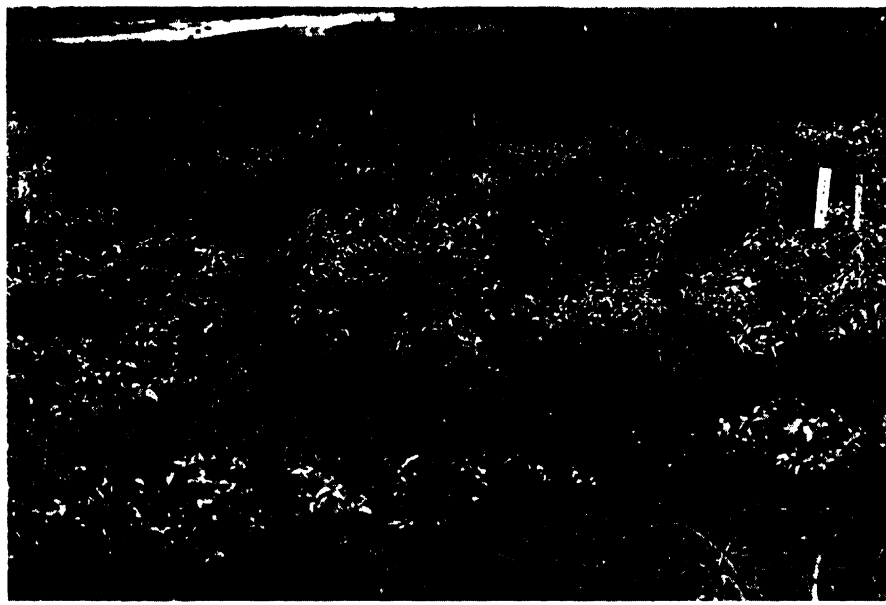

Fig. 13.11. Chlorosis, stunting and wilting of *Desmodium* spp. caused by *Meloidogyne javanica* (Photo: courtesy of J.M. Lenné).

expertise in developing countries and because pastures are usually of low value per unit area, there have been no studies to assess the impact of nematode pests on yield of tropical pastures (Stanton, 1994).

Management

There are limited management solutions to nematode problems of pastures in the tropics (Stanton, 1994). Many controls used for crops are inappropriate for tropical pastures. Chemical control is rarely likely to be economic. Non-chemical control may include the use of resistant pasture species, rotation with non-host crops or weedy fallows, and organic amendments.

Resistant genotypes will often be the only feasible method of controlling plant parasitic nematodes of tropical pasture legumes (Stanton, 1994). Cultivars of several legumes with resistance to *Meloidogyne* spp. are already available (see above). For other legumes, the solution may require collection and screening of germplasm followed by breeding for resistance. Rotations and weedy fallows can be successfully used to control nematodes in crops but further study is needed on their role in pasture systems. Many weedy species in fallows and pastures can be hosts of the same nematodes to be controlled and will, therefore, maintain populations high enough to affect the pasture legume. Where a suitable rotation crop can be found, it may reduce loss of productivity in following pastures but the

economics of rotating a pasture and the length of time over which one rotation will be successful need to be carefully considered.

Organic amendments effectively reduce populations of plant parasitic nematodes by increasing fertility and water-holding capacity of soil and thus increasing tolerance of plants to nematodes (Stanton, 1994). They may stimulate activity of naturally occurring biological control agents and may be nematoxic through production of ammonia upon decomposition. There is potential for the use of organic amendments to control nematodes in pastures by incorporating oil cakes of *Azadirachta indica* (neem) and *Brassica campestris* (Hasan and Jain, 1984). The feasibility of this type of control would depend on the availability and cost of application of large amounts of organic matter.

THE FUTURE

Tropical pasture pathology is a young, challenging and evolving science (Lenné and Sonoda, 1990). Much progress has been made during the past 20 years in identifying and characterizing pathogens, understanding pathogen epidemiology and ecology, and breeding for resistance in tropical pasture legumes (Cameron and Lenné, 1994). Overwhelmingly and justifiably, the major effort has been focused on anthracnose of *Stylosanthes* spp. and much of this research is reviewed in this chapter. For many other important diseases of tropical pasture legumes, however, there are considerable gaps in our knowledge. In a review of the history, evolution and prospects of tropical pasture pathology, Lenné and Sonoda (1990) highlighted future research needs for diseases of tropical pasture plants and these needs have not changed in the interim period. Priority recommendations for future research can be clearly defined in four key areas.

Firstly, simple, precise and reproducible methodologies for characterizing variability and understanding the origin of sources of new variability (e.g. the role of the sexual stage) for the most important pathogens of tropical pasture legumes such as *Colletotrichum gloeosporioides* and *Rhizoctonia solani* are critical for the development of durable management strategies. In spite of the efforts devoted to these pathogens, there is room for improvement (Cameron and Lenné, 1994). Molecular tools should be used wherever appropriate as for *C. gloeosporioides*, but rational decisions are needed on which technologies merit application and utilization. Proven methodologies should then be applied to understanding the relationships between isolates of the same species (e.g. *C. gloeosporioides*, *R. solani*, *Uromyces appendiculatus*, *Phakopsora* spp. and *Meloidogyne* spp.) and species of the same genera (e.g. *Synchytrium* and *Sphaceloma*) on a range of legume hosts. At the same time, it should be noted that determination of variability in many pathogens of tropical pasture legumes will remain difficult in the near future without suitable host lines, in the absence of disease assessment guidelines and lack of understanding of the underlying genetics in both the host and the pathogen.

Secondly, the difficulties of evaluating diseases affecting dynamic, heterogeneous swards of tropical pasture legumes are immense (Lenné, 1989; Lenné and Sonoda, 1990). Initial evaluation should be done in simulated pasture swards

rather than small monoculture plots. More attention should be paid to the effect of diseases on plant demography, especially the effect of diseases on the legume seedling component and the effects of grazing, key factors in long-term productivity and persistence of tropical pastures. This information is useful in developing pasture management strategies that minimize losses from diseases. More studies are needed on a range of pasture legume/grass and disease associations to understand the effects of diseases on size and age structure of legume populations. Pasture pathologists could learn from studies by crop pathologists in intercropping systems.

Thirdly, improved methods are needed to quantify economic losses and to identify which diseases most merit management (Lenné, 1989). This is necessary for tropical pasture legumes in intensive and extensive systems and for acute and chronic diseases. Methodologies are available to measure losses in the quantity and quality of forage produced due to diseases (Lenné, 1989), but this information cannot be readily converted to actual animal production losses. There is a need to evaluate effects of diseases over time as progressive pasture decline can result in reduction in animal production after several years.

Meaningful assessment of the economic significance of biotic damage requires the difficult conversion of losses in pasture legume production to animal production. It would be worthwhile now to develop models which relate yield and quality losses and changes in botanical composition to animal production losses. No matter how severe diseases of pasture legumes appear to be, animal production may not be affected if the pasture is not fully utilized. There is a critical need to study the most important diseases under grazing to quantify the direct effect of diseases on animal production. Only with such information can rational economic decisions be made concerning the need for management strategies.

Fourthly, the plethora of tropical pasture legumes, the complexity of diverse pasture environments, and the potential for multiple interactions among pathogens, hosts, environmental factors and the grazing animal necessitate a multidisciplinary approach to research on diseases of tropical pasture legumes (Lenné, 1989). There is a need for pathologists to work together with plant breeders, agronomists, entomologists, plant physiologists and animal scientists to develop integrated disease management strategies (Lenné and Sonoda, 1990). Where resistance to diseases is not adequate, resistance can be combined with cultural controls such as burning and grazing management (Lenné and Sonoda, 1990). Research is needed to develop strategies to integrate resistance with cultural control.

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TOWARD IMPROVED UNDERSTANDING AND MANAGEMENT OF LEGUME DISEASES

14

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INTRODUCTION

By bringing together for the first time information on the pathology of grain legumes, leguminous oil-seed crops and pasture legumes from both temperate and tropical environments, this book has clearly demonstrated the great diversity of pathogens affecting legumes. These pathogens include fungi, bacteria, phytoplasmas, viruses, nematodes and parasitic angiosperms, each with wide ecological and climatic adaptation and with great variability in natural host range and pathogenicity.

Although legumes were relatively neglected by agricultural scientists until about 30 years ago, both food and pasture legumes now receive considerable emphasis by research teams including pathologists, both nationally and internationally. Advances in understanding and managing the major diseases of the most economically important crops including soyabean, groundnut and common bean have been relatively rapid. The availability of large germplasm collections and use of multilocational testing have contributed to progress in breeding for resistance. In other legume crops such as pigeonpea, chickpea, lupin and tropical pasture legumes, progress has been less rapid due to more limited funding, fewer pathologists and fewer research teams. By fostering communication between plant scientists working on different legume crops, the sharing of information across crops with common problems could be enhanced, and further progress in solving pathological problems of the neglected crops and pasture species could be made. In both well-researched and relatively neglected legume crops, there still remain many gaps in our knowledge of their pathology, especially critical information for the improved understanding and management of major diseases. In the present climate of dwindling donor support, a keener focus on the most important problems that cause greatest loss is crucial. This chapter therefore focuses on the major gaps in our knowledge and the most important

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issues identified by contributors to other chapters of this book. These topics, on which further research is warranted, are :

- diagnosis and characterization;
- assessment of economic losses;
- more relevant and effective management methods; and
- the vital importance of international cooperation in research on major diseases of legumes.

DIAGNOSIS AND CHARACTERIZATION

Identification and Diagnostic Tools

Accurate identification of both the host plant and the pathogen is fundamental to research in legume pathology. However, the ability to recognize and name pathogens and their hosts is becoming increasingly rare (Webster, 1996). For common and well-characterized pathogens, field identification manuals which show macroscopic symptoms continue to be of value to pathologists. Manuals for identifying diseases are produced by the international centres of the Consultative Group on International Agricultural Research (CGIAR), including Centro Internacional de Agricultura Tropical (CIAT), International Center for Agricultural Research in the Dry Areas (ICARDA), International Crops Research Institute for the Semi-Arid Tropics (ICRISAT) and International Institute of Tropical Agriculture (IITA), on common bean, tropical pasture legumes, lentil, faba bean, groundnut, chickpea, pigeonpea and cowpea; these are widely available especially in developing countries. Compendia published by the American Phytopathological Society on diseases of pea, common bean, groundnut, alfalfa (lucerne) and soyabean, many of which are cited in relevant chapters in this book, are also useful. The recent development of the electronic compendium concept at CAB INTERNATIONAL has led to successful production of the Crop Protection Compendium which comprises diagnostic systems, descriptions, illustrations, distribution maps, taxonomic information and literature for hundreds of important diseases, pests and weeds of major crops in South-east Asia. But, in many cases, microscopic examination is needed, and additional laboratory studies using advanced diagnostic tools may also be required.

Although rapid progress has been made with the aetiology of legume diseases, more information is needed on the aetiology of some legume pathogens, especially viruses (see Kraft *et al.*, Chapter 6, Haware, Chapter 9, and Reddy *et al.*, Chapter 10, this volume). For sterility mosaic disease of pigeonpea, the causal agent is still unknown, although recent evidence indicates that it is most probably a virus (Lavakumar, Scottish Crops Research Institute, UK, 1997, personal communication). Studies on stunt disease of chickpea have found that a number of viruses including luteo- and geminiviruses cause stunt in different locations in Asia (see Haware, Chapter 9, this volume). Simple, precise, rapid and reproducible methods for identifying important pathogens of legumes are critical for the development of effective management strategies. Two main types of laboratory-based diagnostic methods have recently been developed for plant pathogens.

Immunological methods have been revolutionized by the use of monoclonal antibodies and enzyme-linked immunosorbent assay (ELISA). The assays are rapid and inexpensive and diagnosis can be completed in several hours (Randles *et al.*, 1996). They are particularly well advanced for the detection of plant viruses where antibodies are prepared against the viral coat protein and are being increasingly used for the detection of bacteria and fungi (Schots *et al.*, 1994).

Increasingly, cost-effective serological methods based on monoclonal antibodies to aflatoxin B1 are being used successfully for detection of contamination in groundnut (Mehan *et al.*, 1991; see McDonald *et al.*, Chapter 2, this volume). Serological tests are widely used for detecting infection by the halo blight and common blight pathogens in common bean (see Allen *et al.*, Chapter 4, this volume) and viruses in groundnut, pea, faba bean, lentil, chickpea and clovers (see McDonald *et al.*, Chapter 2; Kraft *et al.*, Chapter 6; Jellis *et al.*, Chapter 7; Haware, Chapter 9; Mercer, Chapter 12, this volume, respectively). The development of serological tests is still needed for some pathogens, including bean leaf roll virus (see Jellis *et al.*, Chapter 7, this volume), to ensure a more definitive and rapid diagnosis, while for other viruses, such as pea early browning of faba bean (Jellis *et al.*, Chapter 7, this volume) and peanut clump (Wesley *et al.*, 1996), there is such a very wide diversity of serological relationship between isolates and serotypes, that ELISA is not an appropriate test. For accurate detection, nucleic acid-based detection methods are needed. Although more complex, the exploitation of nucleic acid-based diagnostic methods for plant pathogens is approaching the simplicity of immunological methods, with the advantage of greater sensitivity and specificity (Fox, 1993; Randles *et al.*, 1996). Methods such as hybridization, the polymerase chain reaction (PCR), restriction and amplified fragment length polymorphisms (RFLPs and AFLPs), and random amplified polymorphic DNA (RAPD) analyses are now commonly and successfully applied to identify many fungal, bacterial, phytoplasmal, viral and nematode pathogens (Schots *et al.*, 1994).

There are many examples in this book of the successful use of molecular tools to identify legume pathogens, including fungi, bacteria, viruses and nematodes. The development of a species-specific molecular probe for *Colletotrichum acutatum* (Sreenivasaprasad *et al.*, 1994) permitted the accurate identification of the anthracnose pathogen of lupins (Reed *et al.*, 1996; see Hill, Chapter 11, this volume). Molecular studies have confirmed the distinction between strains of *Uromyces appendiculatus* var. *crassitunicatus*, the causal agent of rust on *Macroptilium atropurpureum*, from strains of bean rust, *U. appendiculatus* var. *appendiculatus* (Braithwaite *et al.*, 1991; see Lenné, Chapter 13, this volume). Oligonucleotide primers have been constructed for *Burkholderia solanacearum*-specific DNA sequences which help in detecting single cells of the bacterium in hosts by PCR (Seal, 1994). A rapid identification test has also been developed for distinguishing biovars 3, 4 and 5, based on restriction fragment length polymorphism DNA probes (see McDonald *et al.*, Chapter 2, this volume). Comparison of nucleotide sequences has revealed that the coat proteins of certain isolates of blackeye cowpea mosaic virus (BlCMV) and bean common mosaic virus (BCMV) are sufficiently similar to indicate they are strains of the newly re-defined BCMV (Khan *et al.*, 1993; see Allen *et al.*, Chapters 4 and 5, this volume). This has led to a clearer understanding of relatedness of BlCMV to BCMV and cowpea

aphid-borne mosaic viruses. In addition, PCR methods (Power and Harris, 1993) and RAPD assays (Cenis, 1993) are proving helpful in the identification of *Meloidogyne* species.

One of the most comprehensive studies using molecular tools to understand relationships between fungi is that of Bailey *et al.* (1995) who studied the diversity in *Colletotrichum* at the specific and intraspecific levels. Ribosomal DNA analyses suggested that *C. lindemuthianum* from common bean, *C. trifolii* from lucerne, *C. orbiculare* from *Cucumis* and *Xanthium* and *C. malvarum* from *Sida* constitute a single species (designated the *C. orbiculare* group), in contrast to von Arx (1957) who proposed that they were special forms of *C. gloeosporioides*, and Sutton (1992) who considered them to be separate species (Bailey *et al.*, 1995; Sherriff *et al.*, 1994). Morphological and cytochemical parameters and distinct infection processes of these same species support their close relationship and their distance from other species, including *C. gloeosporioides* (Bailey *et al.*, 1995; Sherriff *et al.*, 1994). Using combined biological and molecular approaches, new relationships between species were established and a firm foundation on which to base the taxonomy of this genus has been provided.

There remain serious gaps in our understanding of a number of other groups of pathogens. Serological and molecular tools should be used wherever appropriate, together with biological approaches, but rational decisions on which technologies merit application and utilization are crucial. At the same time, such tools should be checked for reproducibility and specificity and their limitations should be realized (Janse, 1995). These tools should be applied to understanding the relationships between isolates of the same species and species of the same genera (e.g. *Synchytrium* and *Sphaceloma)* on a range of legume hosts. For example, we remain ignorant of the relationship between *Sphaceloma* and its putative teleomorph, *Elsinoe phaseoli*, and little is known of the variation within the cowpea scab pathogen or between it and the scab fungi of other legumes, many of which are conventionally considered to belong to the same species (Allen, 1983; Allen and Lenné, Chapter 1; Allen *et al.*, Chapter 5; Lenné, Chapter 13, this volume). In certain cases including work on bean scab, the connection between *Sphaceloma* and *Elsinoe* has been well-demonstrated (Phillips, 1996). Studies which use both conventional and advanced diagnostic tools and integrate different parameters would be useful in studies of relationships within other problematic genera, including *Ascochyta, Phoma, Fusarium, Cercospora* and its allies, *Xanthomonas* and potyviruses which together affect many legumes (Allen and Lenné, Chapter 1, this volume).

Distribution and Spread

Information on the geographical distribution of plant disease can be useful both in setting priorities for disease management and in the possible prediction of a pathogen's spread into new areas. Whereas some legume pathogens have spread widely, many remain restricted to specific areas often defined by ecological boundaries (Allen, 1983). The geography of plant disease, or geophytopathology (Weltzien, 1972; Diekmann, 1993), concerns the mapping of pathogen distribu-

tion, and the assessment of the probability of a disease occurring under given climatic conditions. Models are developed by using stepwise discriminant analysis of climatic parameters specific to each disease. Disease risk elsewhere can then be assessed, with obvious relevance to plant quarantine. Such an approach is especially useful for pathogens that are efficiently seedborne. Lists of seedborne pathogens can be useful guides, with at least one important reservation: the literature abounds with reports of seed transmissibility, often assessed following artificial inoculation alone, and seldom relating the apparent degree of transmissibility to any seed inoculum threshold for epidemic development thereafter. Clues to the efficiency of seed transmission can be obtained from the distribution of a pathogen; if it is restricted geographically, or perhaps ecologically (to an environment free of a closed season, for example), then that pathogen is unlikely to be seedborne with much efficiency. Evidence of this kind comes from work on cowpea mottle carmovirus (Allen *et al.*, 1982).

Geographic information systems (GISs) can also help in defining areas where specific constraints are likely to occur and where several constraints may occur together. Recent applications of GIS in plant pathology have targeted diseases in forest and woodland environments (e.g. Shelstad *et al.*, 1991; Thomson and Silvertson, 1994) and other studies focus on general stress in plants rather than specific diseases (Nilsson, 1996). Recently, a study has been made using GIS to predict the occurrence of peanut clump virus disease in India (P. Delfosse, A.S. Reddy and D.V.R. Reddy, ICRISAT, India, 1996, unpublished data). Clump mainly occurs in soils of light texture in cropping systems that involve cereals and where summer temperatures exceed 40°C prior to the onset of the monsoon. Using the two parameters of soil texture and mean air temperature, areas in India sharing the same characteristics have been identified as potential locations for the disease, and these include sites where the disease is known to occur.

Territorial, regional and global checklists of plant pathogens (Lenné, 1990; Allen, 1995; Nene *et al.*, 1996) are a valuable basis for the preparation of distribution maps as well as for the formulation of regulations governing import. However, quarantine legislation often runs the risk of seeming 'set in concrete', and pathogen checklists as well as the regulations stemming from them do need to be updated frequently. However, there are various problems associated with the interpretation of checklists and distribution maps, as stressed elsewhere (Allen, 1983). One concerns the frequent difficulty in distinguishing between the distribution of a pathogen and the distribution of a disease: they are not synonymous. Mapping of pathogens with wide host ranges may give relatively little useful information on the geographical distribution of a particular disease. Where *formae speciales* are distinguised, and when physiological races are clearly defined, these are best mapped separately (see Fig. 5.14), for knowledge of race distribution aids selection of sites for resistance screening as well as the deployment of cultivars with race-specific resistance as, for example, with fusarium wilt pathogens of chickpea and pigeonpea (Haware, Chapter 9, Reddy *et al.*, Chapter 10, this volume). The geographical delineation of races of the pea and chickpea wilt pathogens is also crucial in the development of regional breeding strategies (Kraft *et al.*, Chapter 6; Haware, Chapter 9, this volume).

From the above, it is clear that new methods of detection coupled with

new tools for prediction together provide opportunities to further improve our understanding of the geographical and ecological distribution of disease, as well as the factors that currently restrict pathogen spread. There remains the threat that current concentration on funding of advanced laboratory technology could divert research attention away from the whole plant and from epidemiology, and continued attention to field aspects of legume pathology does seem warranted. A particularly notable exception (Butler *et al.*, 1994, 1995) is recent work on microclimatic factors within the groundnut canopy and their effects on foliar fungi.

Co-evolution and New Encounter

Legumes are notorious for the seed-transmissibility of a large proportion of their pathogens, with the result that many are distributed worldwide with their hosts. Others like the rust fungi are not seedborne but have spread widely nevertheless, sometimes after a period of separation. It is commonly supposed that pathogen and host have co-evolved in a common centre of origin in a long-established relationship. Evidence to this effect is nicely shown in a study of the angular leaf spot pathogen (*Phaeoisariopsis griseola*) and its host common bean, in which it is well established that there are two separate centres of origin in Latin America, in the Andes and in Central America (Gepts and Debouck, 1991). Distinct RAPD patterns divide the pathogen into two groups that correspond with the two centres of host origin: Andean isolates were generally recovered from the Andean gene pool and Mesoamerican isolates were recovered from the Mesoamerican gene pool of *Phaseolus vulgaris*. Furthermore, each pathogen group was found to be more pathogenic to its 'own' host gene pool (Guzmán *et al.*, 1995). Examples of co-evolved host and pathogen relationships that have been interrupted and rejoined spatially include anthracnose of *Stylosanthes* in which *Colletotrichum gloeosporioides* and its host co-evolved in South America, and were separated on the introduction of stylo to Australia where later the pathogen caught up in a 're-encounter' (Lenné and Trutmann, 1994). Similarly, groundnuts were introduced into West Africa and India from their centres of origin in South America sometime in the sixteenth century, whereas the groundnut rust fungus (*Puccinia arachidis*) which is not seedborne has spread from South America to Asia and Africa only during the last few decades (Allen, 1983). In several important legume crops, the region of major production is now far removed from the region of origin (Allen and Lenné, Chapter 1, this volume), and it has been suggested that there is greater productivity outside centres of crop origin that can be attributed to a lesser diversity of parasites (Jennings and Cock, 1977). But is this necessarily so; what origins of epidemics are there other than a co-evolution between host and pathogen?

In an analysis of the origin of epidemics, Buddenhagen (1977) drew attention to the existence of genetically new-encounter diseases that follow reunion of two long-separated components of isolated evolutionary systems, often as a result of intercontinental movement of a crop into a new environment. With the single exception of Allen (1983), who suggested examples of new-encounter diseases among tropical food legumes, the concept has been ignored. New encounters

appear to warrant greater attention, for the origin of an epidemic clearly has implications for effective disease management strategy. A few examples will suffice. In the groundnut crop, of South American origin, groundnut rosette in Africa, peanut clump in both Africa and India, and peanut stripe in Asia and the USA are each new encounters, and the same seems to be true of cowpea severe mosaic in Brazil (Allen, 1983; McDonald *et al.*, Chapter 2, this volume). Black root of common bean in Africa is now considered to be caused by bean common mosaic necrosis virus which is probably of African origin (Sengooba *et al.*, 1997). It appears that new encounters are especially common among virus diseases, but there are examples among fungal diseases too, including *Phytophthora* stem rot of cowpea in Australia, bean scab in Africa and red leaf blotch of soyabean in Africa (Allen, 1983; Hartman and Sinclair, 1996). Among bacterial diseases, some of the nine races of the halo blight pathogen are apparently confined to Africa, but whether or not they too represent new encounters of the introduced common bean remains unclear (Taylor *et al.*, 1996). In all these cases, the question arises from what alternative native legume host plant did the pathogen come, with obvious implications for its epidemiology.

Gene centres of plants have long been recognized as rich sources of resistance against diseases where host and parasite have co-evolved (Leppik, 1970), perhaps in a gene-for-gene relationship (Thompson and Burdon, 1992). It is sometimes claimed that secondary centres of genetic diversity are rich sources of resistance to new-encounter pests and diseases, and this assumption is then liable to be used to justify local landrace collection. There is some evidence to this effect from disease resistance but there is also another explanation. For instance, an important source of resistance in maize to its new-encounter streak geminivirus is cv. Revolution that comes from Reunion where streak was thought not to occur (Soto *et al.*, 1982). West African landraces of groundnut are sources of rosette resistance (Daniel and Berchoux, 1965), but new sources of resistance to rosette have been found among landraces from Tamil Nadu in India where they have never been exposed to rosette (P. Subrahmanyam, ICRISAT, Malawi, 1997, personal communication). Furthermore, some wild species of *Arachis* show resistance to all components of the rosette virus complex, including the first demonstration of resistance to the assistor virus. Since the wild species have not been exposed previously to rosette, their resistance is clearly not co-evolved. In common bean scab, sources of resistance have been found not only among African landraces but also among germplasm freshly introduced from Latin America where scab does not occur (Kannaiyan and Haciwa, 1993; Phillips, 1995). All these are presumably examples of 'allopatric resistance' in which there is an association between resistance to the new-encounter disease and some other character. Recent evidence suggests that a single gene can sometimes confer resistance against several widely different pathogens, as suggested may be the case with the 'R3' and 'I' genes in *Phaseolus vulgaris* that appear to be identical and confer a truly multiple resistance, indicated by reactions to halo blight, brown spot and five potyviruses (Taylor *et al.*, 1995). (Most cases of 'multiple resistance' in the literature refer to a pyramiding of resistance genes in a single host cultivar in which they confer individual protection against separate parasites; this may be better referred to as combined resistance.) Further work on the

identification and sequencing of resistance genes seems likely to elucidate the basis of allopatric resistance. Work to date shows there can be very close homology between genes for resistance in the same crop against distinct pathogens as well as across widely different plant species. This has been demonstrated among genes conferring resistance to fungal and bacterial pathogens as well as to viruses. Kanazin *et al.* (1996) used oligonucleotide primers for conserved sequences from coding regions of genes for disease resistance from tobacco, flax and *Arabidopsis* to amplify related sequences from soyabean. Their data indicate that it may be possible to use sequence homologies from conserved motifs of cloned resistance genes to identify candidate resistance loci from widely diverse plant taxa.

Host Range

There is great diversity in the degree of host specificity shown by legume pathogens. Those with narrow host ranges, like *Cercosporidium arachidicola* and *Phaeoisariopsis personata* which affect only groundnut (McDonald *et al.*, Chapter 2, this volume), may be considered genus-specific. Others including *Phoma exigua* var. *diversispora*, *Pseudomonas syringae* pv. *phaseolicola* and *Uromyces appendiculatus* are effectively tribe-specific (Allen *et al.*, Chapter 14, this volume), and yet others have broader host ranges across several tribes within the *Leguminosae*. Examples of the latter group include *Phakopsora pachyrhizi* (Sinclair, Chapter 3, this volume) and bean common mosaic necrosis virus (Sengooba *et al.*, 1997). Finally, there are those pathogens with very broad host ranges within and beyond *Leguminosae*; an example is *Rhizoctonia solani* (Chapters 2, 5, 6, 9, 11 and 13, this volume).

A sound knowledge of legume taxonomy and the affinities between genera can be expected to aid prediction of disease outbreaks when certain hosts are brought together. The concept of bridging hosts may have relevance here. Genera, including *Neonotonia*, appear to act as a 'bridge' between soyabean and common bean: *N. wightii* shares the red leaf blotch pathogen (*Dactuliochaeta glycines*) with soyabean but not with common bean, whereas *N. wightii* shares the halo blight pathogen (*Pseudomonas syringae* pv. *phaseolicola*) with common bean but not with soyabean (Hartman *et al.*, 1987; Allen *et al.*, Chapter 4, this volume). Taxonomic confusion among host genera is likely to have led to confusion in the identity of pathovars of bacterial pathogens like *Xanthomonas campestris* (Allen and Lenné, Chapter 1, this volume). It has been shown (Savile, 1954, 1979) that fungi are themselves useful aids to host plant taxonomy, and rust fungi can be used as indicators of the times of domestication of their hosts. The suspected relationship between the rusts of *Arachis*, *Stylosanthes* and *Zornia* is supported by the close affinity of these three host genera within the subtribe *Stylosanthinae* (McDonald *et al.*, Chapter 2; Lenné, Chapter 13, this volume). Similarly, a close relationship is suspected between the rusts of faba bean and lentil that are two closely allied genera within *Vicieae* (Jellis *et al.*, Chapter 7; Bayaa and Erskine, Chapter 8, this volume).

Natural, or at least 'semi-natural', host ranges of pathogens may be expected

to be narrower than artificial host ranges, and all too often monographic reviews of particular legume pathogens tend to regurgitate long lists of host plants devoid of comment on whether those species are naturally infected or merely experimental hosts. Gibbs and Watson (1980) draw attention to the bias implicit in the choice of legume species used by virologists; they propose a sample of species that could be used in host range studies so that a pathogen should indicate its taxonomic preferences for the subfamilies or tribes of *Leguminosae*.

A final comment on host range concerns not host affinity but host plant ecology. Whereas it is well known that some cereal rust fungi like *Puccinia graminis* Pers. are heteroecious and have hosts in *Gramineae* and *Berberidaceae*, it is pertinent that no rust of legumes mentioned in this book is considered heteroecious. However, there are examples of temperate species of *Uromyces* on legumes that have alternative hosts on *Euphorbia* (Butler, 1958). In areas with a marked closed season, one might expect some advantage to pathogen survival from alternation between a legume cultivated as an annual and a perennial host; it is tempting to suggest that the taxonomically diverse, but ecologically related hosts of peanut clump virus in *Gramineae* and legumes (McDonald *et al.*, Chapter 2, this volume) provide a situation analogous to this alternation.

Characterization of Pathogenic Variation

Pathologists' preoccupation with pathogenic variation stems principally from the risk that host plant resistance proves transient in agriculture, when cultivars with race-specific resistance are challenged by virulent physiological races. Races themselves are conventionally differentiated by use of a set of host genotypes that show clear-cut responses to infection as is usually conferred by major genes. Physiological races have been shown convincingly to exist within a wide range of the legume pathogens discussed in this book. These include the following examples: the *Phytophthora* stem rot fungi of soyabean and cowpea (Purss, 1972; Sinclair, Chapter 3, this volume); the downy mildew pathogen of pea (Kraft *et al.*, Chapter 6, this volume); the fusarium wilt pathogens of pea, chickpea and pigeonpea (Kraft *et al.*, Chapter 6; Haware, Chapter 9; Reddy *et al.*, Chapter 10 this volume); the anthracnose pathogen of common bean (Allen *et al.*, Chapter 4, this volume); the rust fungus of faba bean (Jellis *et al.*, Chapter 7, this volume); the pseudomonad blights of soyabean, common bean and pea (Sinclair, Chapter 3; Allen *et al.*, Chapter 4; Kraft *et al.*, Chapter 6, this volume); bean common mosaic and bean common mosaic necrosis viruses (Allen *et al.*, Chapter 4, this volume); and witchweed of cowpea (Allen *et al.*, Chapter 5, this volume).

In a range of other cases, claims have been made for the existence of races but the evidence is insubstantial and not yet fully convincing. Examples include *Botrytis fabae*, in which there are no demonstrable physiological differences among the putative races (Jellis *et al.*, Chapter 7, this volume), a number of the ascochyta blight fungi and most of the xanthomonads that infect legumes (Allen and Lenné, Chapter 1, this volume). Good progress has been made in refining methods for characterizing diversity but the value of molecular tools in race identification has so far been rather limited. DNA-based studies of *Colletotrichum*

gloeosporioides populations from *Stylosanthes* in Australia have revealed considerable intraspecific variation, permitting the distinction of two types, but physiological races as such have not been identified (Manners *et al.*, 1992, 1993; Lenné, Chapter 13, this volume). Attempts at characterization of *Rhizoctonia solani* by use of RAPD-PCR have failed to distinguish isolates known to differ substantially in pathogenicity (Yang *et al.*, 1995). Some further refinement of molecular methods to better monitor virulence shifts in pathogen populations seems necessary, although preliminary studies of *Fusarium udum* from pigeonpea are encouraging: RAPD analyses do show close correlation with conventional methods for differentiation of the two physiological races recognized (Reddy *et al.*, Chapter 10, this volume).

Whereas the mapping of race distribution can guide deployment of cultivars with race-specific resistance (Allen *et al.*, Chapters 4 and 5, this volume), the current fashion for the demonstration of genetic diversity is accompanied by certain risks. One is obviously that it is not merely the *extent* of diversity within a pathogen population that is of concern. It is also the degree of host specificity of the interaction between the pathogen and its host that has important implications for the plant breeder (Allen, 1983). Interactions are often quantitative in nature, and pathogenic variation may be cultivar non-specific. Such variation in aggressiveness, as distinct from qualitative, cultivar-specific variation in virulence, seems often to be either misunderstood or ignored. The acceptance of the dual concept of vertical and horizontal resistance in hosts is widespread, but the parallel concept (van der Plank, 1968) that pathogenicity is also both vertical (virulence) and horizontal (aggressiveness) is much less commonly understood. Failures to distinguish these have sometimes led to costly errors in grain legume improvement programmes. There is also sometimes an alarming, even criminal, tendency to regroup data on plant response to make two distinct categories ('resistant' and 'susceptible') when in reality there is no such clear cut distinction (Robinson, 1987). There is one other important point to be made here. Whatever the method used to detect major genes for virulence within a pathogen, if *the host itself* cannot detect those genes then the durability of its resistance seems unlikely to be threatened. An example comes from work on the bacterial blight pathogen of *Phaseolus* species (Opio *et al.*, 1996). Study of the nature of pathogenic variation among *Xanthomonas campestris* pv. *phaseoli* on a range of common bean genotypes revealed quantitative host non-specific differences. On tepary bean, however, the same isolates interacted differentially and a range of physiologic races was defined, suggestive of an underlying gene-for-gene relationship. Despite this apparent gene-for-gene interaction, resistance in common bean has remained non-specific and essentially durable, and it is suggested that breeders should resist the temptation to incorporate the new complete resistance from tepary bean as it would risk destabilizing the host–bacterium relationship.

Remarkably little remains known about the origins of pathogenic variation. Alternative hosts are likely to provide opportunities for increased variability in pathogenicity, and better understanding of relationships between anamorph and teleomorph may prove crucial in certain cases, including the scab fungi and some of the ascochyta blight pathogens (Allen and Lenné, Chapter 1, this volume).

ASSESSMENT OF ECONOMIC LOSS

Loss Assessment

Crop losses due to disease are among the most significant worldwide constraints to increasing productivity and total food production (James *et al.*, 1991). The importance of crop loss assessment and approaches for collecting data have been discussed frequently and the reader is referred to reviews by James and Teng (1979), Madden (1983), Teng (1987) and Madden and Nutter (1995). In spite of the emphasis given to this aspect of plant pathology, the ability to relate crop yield to plant disease has remained difficult in both theory and practice (Madden and Nutter, 1995). The major challenge continues to be narrowing the gap between potential yield and actual on-farm yield. There is also a need to better appraise the place of disease in the production system among other constraints including insect pests, edaphic and climatic stresses, so as to better focus research attention on the crucial priorities, at a time when funds are shrinking.

There is an alarming lack of sound quantitative data on economic losses due to major diseases of many important food and pasture legumes. In soyabeans, losses from diseases on a worldwide basis have been estimated at about 15% of potential yield for any single season (Sinclair, Chapter 3, this volume) but losses vary widely from year to year. They are due primarily to foliar diseases but not always to the same disease in any one year.

The potential economic consequences of the entry of one single disease (soyabean rust) into the USA was estimated some 13 years ago to exceed $7.2 billion per year (Kuchler *et al.*, 1984). In other legumes reviewed in this book, estimates of yield losses from specific diseases vary from 1 to 80%, but it is difficult to determine whether these data represent measurements over many years and over wide areas. For many diseases, recent data are lacking (Reddy *et al.*, Chapter 10, this volume), especially from developing countries where many constraints occur together. Decreasing research resources are partly responsible, but so too are increasing human population pressures that bring about changes in cropping pattern, with more marginal production subject to increasing stress.

In common bean, Allen *et al.* (Chapter 4, this volume) emphasize the need for better quantification of crop loss caused by diseases, among the other agronomic constraints on common bean productivity. Diagnostic trials on farm can be valuable in determining the economic importance of disease relative to other agronomic stresses (Trutmann and Graf, 1993). In chickpea, Haware (Chapter 9, this volume) identifies the most important constraints in each region of chickpea production, though information on global losses is lacking. Yield losses and reduction of quality due to virus diseases can be substantial in pea during years of epidemic virus incidence; however, this is not quantified (see Kraft *et al.*, Chapter 6, this volume). Although diseases of clover can cause significant economic losses, data are often hard to obtain (see Mercer, Chapter 12, this volume). Frequently, more than one stress is associated with a clover crop which is performing poorly and it is not always easy to quantify the effects of individual pathogens. The difficulties of evaluating diseases affecting dynamic, heterogeneous swards of tropical pasture legumes are immense (see Lenné, Chapter 13,

this volume). More attention should be paid to the effect of diseases on the legume seedling component and the effects of grazing, key factors in long-term productivity and persistence of tropical pastures. Improved methods are needed to quantify economic losses and to identify which diseases most merit management (Lenné, 1989).

For some legumes, loss of product quality is as important as yield losses. The quality of oil, particularly the free fatty acid content, can be greatly affected in groundnut by soilborne diseases such as aspergillus crown rot and yellow mould (see McDonald *et al.*, Chapter 2, this volume). Aflatoxin contamination due to yellow mould has had a tremendous impact on the global groundnut industry and on consumers (Mehan *et al.*, 1991). Discoloured seeds due to frogeye leaf spot may reduce soyabean seed quality and value (see Sinclair, Chapter 3, this volume). The stain induced by broad bean stain virus and the small seeds produced from infection with pea seedborne mosaic virus can each render faba bean seed useless for processing for human consumption (see Jellis *et al.*, Chapter 7, this volume). Downy mildew can cause significant losses in quality of peas when conditions are optimum for development (see Kraft *et al.*, Chapter 6, this volume). Varietal susceptibility to diseases including rust and anthracnose, coupled with exacting market demands for quality, together account for the frequent and heavy doses of fungicides that are applied to snap beans (Silbernagel *et al.*, 1991).

For groundnut, the effect of rosette can be felt far beyond the year of the epidemic. For example, after the epidemic in 1975 in West Africa had destroyed Nigeria's crop, much of the groundnut production in northern Nigeria changed from sole to intercrop systems. Similarly for clump virus, the build-up of inoculum in the soil can lead to groundnut being abandoned (see McDonald *et al.*, Chapter 2, this volume). In faba bean, stem rot caused by *Sclerotinia sclerotiorum* can restrict the frequency of faba beans in crop rotations while infestations of broomrape can result in farmers diverting land to other crops (see Jellis *et al.*, Chapter 7, this volume).

Is the lack of quantitative information due to a lack of sound methodology to evaluate crop losses? This is certainly the case for tropical pasture legumes (see Lenné, Chapter 13, this volume) but may be part of a wider problem. Pathologists often apply internationally approved disease assessment scales, developed from knowledge of the relationship between severity and actual losses measured under specific environmental conditions. How widely applicable are these scales? To what extent have actual losses been measured in a range of environments? Is the key problem a failure to measure disease severity and incidence accurately? In a far-reaching review of the value of mixtures, Smithson and Lenné (1996) showed that in spite of tremendous reductions (as much as 80%) in disease on many crops, the yield increase in mixtures was slight, usually less than 10% and often less than 5%. Similarly, multilocational trials with common bean cultivars in Africa have revealed that disease severity scores are seldom related to grain yield. Data from 29 trials of 25 genotypes grown in 12 countries showed that 'seed yields were larger in environments where diseases were more severe. Pod numbers were fewer where diseases were more severe, but otherwise regression analysis provided no evidence that diseases were deleterious to yield' (Smithson *et al.*, 1994). These examples question the accuracy of disease evaluation scales

and their relationship with crop loss. They also suggest that sometimes diseases are conspicuous rather than economically significant, at least in small plots under experimental conditions.

It is important to have a clear understanding as to what diseases occur in legume production systems, their relevance and severity over seasons and how they are influenced by cultural practices and by other crops in the system. Priority should be given to disease surveys and crop loss evaluations which can be coordinated at national, regional and international levels to save resources. Information generated could be stored in geographical information systems for wider use and for correlating with information on soils, climate, and other data for future modelling.

Aerial photography is extremely useful for assessing rapidly and reliably the extent of damage caused by soilborne diseases (Lee, 1989). Toler *et al.* (1981) showed that colour and colour infrared films are most useful for the detection of foliar colour changes that result from damage caused by soilborne diseases. Computer digitizing cameras have been used to digitize aerial photographs and these data have been successfully used to detect diseased crops (Bronson and Klittich, 1984) including lucerne root rot, caused by *Phymatotrichum omnivorum* (Shear) Duggar (Lee, 1989). Computer-aided photo-interpretation of digitized aerial photographs, using an image analysis system, may allow scientists to deal with detection of soilborne diseases more effectively (Lee, 1989). Results from the computer classification of the damaged areas delineated can be transferred directly to agricultural maps and used simultaneously to update databases using GIS. Recent methods use 35 mm aerial photographs with digital elevation models to measure crop losses directly with a standard digitizing tablet (Warner, 1994), and digital cameras are also available. Using measurements of canopy reflectance taken at 800 mm wavelength with a multispectral radiometer, more accurate predictions of pod yield loss in the groundnut–late leaf spot pathosystem were obtained using assessments based on defoliation (Nutter and Littrell, 1996). Similar results were obtained by Bryson *et al.* (1995) for the wheat–yellow rust pathosystem. These methods are less costly than extensive ground surveys and sampling and have broad application for assessment of legume diseases, especially of extensive areas of pasture legumes.

Modelling

Progress in sampling theory and application, and advances in instrumentation together may make estimation of crop losses more feasible (Madden and Nutter, 1995). Recent research on crop loss assessment has led to a better understanding of losses in relation to plant diseases. However, there are too few cases where severity has been related to expected losses and insufficient development of models that accurately relate the severity of disease to loss. Advances in understanding crop losses depend in no small part on research in modelling crop losses in relation to disease intensity (Madden and Nutter, 1995) and the validation of the models. Some useful progress has been made with common bacterial blight and rust for which crop loss models are now available (Lindgren *et al.*, 1995; see

Allen *et al.*, Chapter 4, this volume), but such studies need to be extended to other diseases if clear priorities among constraints are to be set. Further research is needed on the effects of temperature, humidity and leaf wetness in the crop canopy on infection by legume foliar pathogens, as small variations can greatly influence disease development. Significant advances have been made for the leaf spot and rust fungi on groundnut (Butler *et al.*, 1994) and a model is being developed (D.R. Butler, ICRISAT, Patancheru, India 1997, personal communication). Microclimatic data gathered for physiological growth models also have much relevance in calculating disease risk and developing forecasting systems.

Meaningful assessment of the economic significance of damage in tropical pastures requires the difficult conversion of losses in pasture legume production to animal production (Lenné, 1989; Lenné, Chapter 13, this volume). It would be worthwhile now to develop models which relate yield and quality losses and changes in botanical composition to animal production losses. There is a critical need to study the most important diseases under grazing to quantify the direct effect of diseases on animal production. Only with such information can rational economic decisions be made concerning the need for management strategies.

There are additional areas where work is needed to describe and understand disease–loss relationships. These include measurement error, model validation and the problems of dealing with multiple diseases, pests and other stresses, as raised by McDonald *et al.* (Chapter 2), Allen *et al.* (Chapter 4), Reddy *et al.* (Chapter 10), Mercer (Chapter 12) and Lenné (Chapter 13, this volume). Significant advances have been made by Savary and Zadoks (1992a, b) in analysing crop loss in the multiple groundnut–rust–late leaf spot pathosystem in West Africa. Using correspondence analysis, categorization of yield levels, damage and injury patterns have been established. Competition between injuries appeared to be mediated more from leaf spot to rust, due to leaf spot induced defoliation (Savary and Zadoks, 1992b). It is recommended that this approach be applied to other legume–multiple disease pathosystems.

Progress has been made in rice with the development of the CERES-rice crop growth model for simulation of multiple species pest damage (Pinnschmidt *et al.*, 1995). The approach is considered to be sufficiently mechanistic and generic to be incorporated into other crop growth models, such as SOYGRO, PNUTGRO (Batchelor *et al.*, 1993; Boote *et al.*, 1993) and BEANGRO (Hoogenboom *et al.*, 1987). Thus, applications to multiple biotic constraints in groundnut, soyabean and common bean will be possible. Crop growth models should be developed for other legumes to ensure wider application of this approach.

As the ability to assess economic losses accurately is critical to making rational decisions about management strategies, this is a key area of research for most legumes reviewed in this book. Of crucial importance is the development of methodologies to quantify losses across constraints; diseases cannot be viewed in isolation, especially in legume production systems in developing countries (Wortmann and Allen, 1994).

DISEASE MANAGEMENT

Management of Seedborne Pathogens

Production of clean seed is a principal objective in the management of many legume diseases and is often an integral part of disease management strategies. Clean seed is a concern for the farmer, especially if seed is retained for sowing the following year; for the seed producer who has to reach the required health and quality standards for certification; and for quarantine if seed is moved from country to country. As we have seen, infected seed plays an important role both in pathogen survival and in long-distance dispersal of pathogens to new areas, and explains why many of the most important diseases of legumes are widespread. That many important pathogens of legumes are seedborne is clearly demonstrated in Chapters 2–13 of this book. In the *Leguminosae*, with their dehiscent fruit, the strong vascular connections between the mother plant and developing seed and the large cotyledons together favour bacterial and viral infection of seeds, particularly by those pathogens which invade through the vascular system (Agarwal and Sinclair, 1987). Since seeds are enclosed in a pod, this provides further protection and conditions favourable for penetration of pathogens from pods to seed. Management of seedborne pathogens of legumes is possibly more important than in other crops, including cereals. The best way to avoid seedborne infection is to identify locations or seasons for production of healthy seed, so avoiding high risk areas or periods. Diekmann (1992) has developed a model, based on climatic data from chickpea growing areas, by which to identify agrogeographical zones and seasons with the highest risk of ascochyta blight. The model helps to concentrate disease control measures, like quarantine, on high risk areas, and identifies sites or seasons for production of healthy seed (Diekmann, 1992). Such studies are needed for other important seedborne pathogens of legumes. Spray regimes can also help to reduce the risk of seed infection by fungal and bacterial pathogens as reported by Haware (Chapters 9, this volume) and Lenné (Chapter 13, this volume), while field inspection and roguing can reduce seed infection by viruses (Allen *et al.*, Chapter 5, this volume). Seed treatment chemicals and biocontrol agents can suppress the amount of initial infection induced by many pathogens (see Chapters 4, 5, 6, 9, 10 and 13, this volume).

Accurate information on the importance of seed transmission of a legume pathogen is essential for making decisions on whether to apply strict control and quarantine measures. This is illustrated by the case of *Curtobacterium flaccumfaciens* (Hedges) Collins & Jones, the causal agent of bacterial wilt of common bean, which has been found on common bean in several locations in the USA and Africa and on other legumes in Latin America (Allen, 1995). Owing to its seed transmissibility and restricted geographical distribution, *C. flaccumfaciens* has been assigned to the high risk category of quarantine objects, with the result that this bacterium is the 'hot potato' among common bean pathogens in the eyes of plant quarantine officials. With the benefit now of hindsight, it seems timely to review this status: nowhere is bacterial wilt important and the ecological efficiency of its seed-transmissibility now seems dubious.

Resistance

Heritable resistance that provides comprehensive protection of a crop to disease is perhaps the most valuable first gift that science can proffer to the hundreds of millions of small farmers in the poor countries of the world. The incorporation of reliable resistance into locally acceptable cultivars, or cultivar mixtures, need not disrupt the farming system, nor does it impose on the farmer a dependence on expensive inputs. Recent developments in molecular genetics appear to have brought nearer the prospect of science providing permanent solutions to susceptibility to disease. Meanwhile, on the other hand, scientists have come to appreciate and learn from farmers' experience so that farmer participation in the development of new cultivars is an increasingly common feature of crop improvement programmes (Sperling *et al.*, 1993).

Considerable progress has been made towards the successful management of important diseases of most legume crops through the search for host resistance. Sources of resistance to many important diseases have been found and these are being used to breed agronomically acceptable cultivars with good levels of resistance. However, where pathogens are highly variable, breeding for resistance continues to be a long-term objective. It is encouraging to see that resistance to many diseases has been relatively stable over a wide range of locations over many years. Examples include rust resistance in groundnut (McDonald *et al.*, Chapter 2); the 'Arc' gene for resistance to anthracnose in common bean (Allen *et al.*, Chapter 4); resistance to wilt, powdery mildew and several viruses in pea (Kraft *et al.*, Chapter 6); resistance to rust, wilt and ascochyta blight in lentil (Bayaa and Erskine, Chapter 8); and resistance to wilt in chickpea (Haware, Chapter 9, this volume). Notable exceptions include: rust of common bean; bacterial blight and downy mildew of pea; ascochyta blight of chickpea, and anthracnose of *Stylosanthes* spp. (Allen *et al.*, Chapter 4; Kraft *et al.*, Chapter 6; Haware, Chapter 9; Lenné, Chapter 13, this volume, respectively). The durability of resistance against many of the diseases of legumes remains inadequately tested, in part because of the relatively recent development of resistant cultivars and perhaps partly because of the protective effects of the complex cropping systems in which most legumes are grown. From the examples above, it is clear that in some cases existing resistance already provides adequate protection; in other cases, either levels of resistance are insufficient or afford only local or transient protection. In yet other cases, attempts to develop a more complete resistance may risk undermining durability (Opio *et al.*, 1996). Multilocational testing of legume germplasm remains the chief means by which wide-spectrum disease resistance is identified, especially at the International Agricultural Research Centres (IARCs). However, the apparent stability of reaction across environments is no guarantee that resistance is in fact durable, as stressed previously (Allen, 1983). Whereas multilocational testing identifies resistance that is effective on a small scale against several pathogen populations in different environments in the short term, durable resistance is recognized only after a long period of testing in large-scale production in one environment. The extent to which location non-specific resistance is an index of durability is fundamental to the strategy of multilocation testing, yet the relationship remains uncertain. Much must depend on the choice

of site. If variation across locations does approximate the range of environmental variation across seasons at a given site, then it seems probable that multilocation testing would accelerate the identification of stable resistance. Conversely, if those sites differ so widely that the variation in environment and their pathogen populations far exceed those experienced across seasons at the target location, then factors other than durability are likely to be sought. Underlying this may be the relationship between site non-specific resistance and race non-specific resistance. Clearly, there are a number of causes of site differential interactions only one of which is the 'breakdown' of race-specific resistance (Allen, 1983). If race non-specific resistance tends to be a quantitative trait, then it may be highly significant that technology is now available for genetic marking for characters under quantitative control (Edwards, 1992; Dudley, 1993). Recent advances in marker technology may well have paved the way to a new revolution in our ability to manipulate quantitative traits in crop improvement.

Combined disease resistance is required in most legume production systems. This has proved relatively easy to attain in some cases, like rust and late leaf spot of groundnut and a range of viruses in cowpea, but rather difficult in other cases, including early leaf spot and rosette of groundnut (McDonald *et al.*, Chapter 2, Allen *et al.*, Chapter 5, this volume). A case of true multiple resistance conferred by the R3/I gene in common bean has already been mentioned earlier in this chapter. The conserved sequences among genes for disease resistance cloned from widely different plant hosts (Kanazin *et al.*, 1996) seem likely to be useful in identifying evolutionarily related genes in legumes including soyabean.

Combined resistances, like those to rust and chocolate spot, are known among ICARDA lines of faba bean; however, there are indications that it will be difficult to combine high levels of resistance to a number of diseases with the desired agronomic and quality traits. For example, cultivars low in tannin tend to be more susceptible to *Fusarium* spp. (Jellis *et al.*, Chapter 7, this volume). A recent major programme sponsored by the European Commission has provided breeding material with a combination of resistances to pathogens (including chocolate spot) with reduced levels of anti-nutritional factors. In chickpea, considerable effort has been given to developing lines with combined resistance to several diseases (e.g. wilt and root rots) with limited success (Haware, Chapter 9, this volume). The importance of combined disease resistance in pigeonpea for resource-poor farmers cannot be overemphasized; recently, the line ICPL 87119, which is resistant to both wilt and sterility mosaic, has been released as 'Asha' (meaning 'hope') for general cultivation in India (Reddy *et al.*, Chapter 10, this volume).

If current research into the transformation and insertion of foreign genes into crops proves to be widely successful, the objective of combined disease resistance will be much closer. In the past, attempts to move genes from wild species into crops has been highly successful for wheat, tobacco, tomato and potato but largely a failure for legume crops (Lenné and Wood, 1991), mainly due to the lack of good transformation and regeneration systems for many legumes. Successful methodologies for *Stylosanthes* spp. have been known for some time. The breakthrough in crossing wild *Glycine* spp. (which are immune to most soyabean pathogens) with soyabean provides a source of genetic material yet to be

exploited (Sinclair, Chapter 3, this volume). Attempts to transform and regenerate groundnut are in progress (McDonald *et al.*, Chapter 2, this volume). Pea has been successfully transformed using immature cotyledons (Grant *et al.*, 1995) and a similar method is showing some success in chickpea (Haware, Chapter 9, this volume). However, other legumes such as faba beans are proving to be very difficult to transform, particularly because of the lack of success in regenerating plants in tissue culture. Providing efficient transformation and regeneration systems are developed, the genetic engineering approach will be promising for developing cultivars with resistance to viruses using viral coat protein genes, like the work in progress on several viruses of groundnut (see McDonald *et al.*, Chapter 2, this volume). The insertion of antifungal genes to enhance quantitative resistance to root and foliar pathogens in pea (Jach *et al.*, 1995) and resistance to grey mould in chickpea (see Haware, Chapter 9, this volume) are further examples.

The widespread application of DNA marker technology to the construction of genetic maps has allowed location of genes affecting both simple and complex traits (Paterson *et al.*, 1991). With a large number of genetic markers one can build a complete genetic map which is informative about all regions of all the chromosomes in an organism. Although marker technology is tedious, laborious and expensive, it can accelerate breeding endeavours and provide new approaches especially to introgression of valuable traits from exotic germplasm into domestic cultivars (Paterson *et al.*, 1991). Many examples of linkage between genetic markers and genes influencing simply inherited traits have been found, including those for disease resistance in crops such as tomato, maize, rice, and *Brassica* spp. (Paterson *et al.*, 1991), but fewer examples are available for legumes, partly because of their inbreeding nature and lack of polymorphisms. An exception is soyabean as has been mentioned above. A search for RAPD markers is under way in lentil for rust, ascochyta blight and vascular wilt, so that marker-assisted selection may be used to increase selection efficiency in the future (Bayaa and Erskine, Chapter 8, this volume). However, the use of markers to search for single-gene resistances to legume pathogens offers a no more lasting solution than the use of conventional technologies to utilize the same genes. The main advantage of marker technologies will be the increased efficiency in pyramiding several dominant resistance genes into a single line with the objective of achieving more durable resistance.

Integrated Management

Management of legume diseases should address the cropping system as a whole, if full advantage is to be taken of available control measures. Components include the adjustment of sowing date, use of cultivars of different duration, rotation, intercropping, cultivation and land form, plant population and spacing patterns. Interactions between different diseases, and with abiotic stresses such as drought and unfavourable temperatures, also must be considered. The economic and socio-economic aspects of integrated management packages should be examined when these packages are being field tested. Our ultimate aim must be the development of safe, economic and durable management strategies for a range of farm

situations. This will probably be achieved only through a combination of measures into an integrated management system including cultural practices, crop and varietal mixtures, and in some systems also chemicals, as well as host plant resistance.

Many examples of the use of integrated management strategies for legume diseases which cannot be adequately controlled by resistance alone are given throughout this book. In groundnut, efforts are directed at developing integrated management programmes for aflatoxin; stem, root and pod rot (Mehan *et al.*, 1991, 1995); rust and leaf spots; and root-knot nematode (McDonald *et al.*, Chapter 2, this volume). In soyabean, integrated management packages are being developed for phytophthora root and stem rot and cyst nematode (Sinclair, Chapter 3, this volume). In common bean, attention has focused on ascochyta blight (Allen *et al.*, Chapter 4, this volume), while for cowpea, integrated management is considered a desirable approach for bacterial blight and pustule and witchweed (Allen *et al.*, Chapter 5, this volume). Integrated management has been recommended for downy mildew, cortical root rots and ascochyta blight in pea (Kraft *et al.*, Chapter 6, this volume) and broomrape in faba bean (Jellis *et al.*, Chapter 7, this volume). In the absence of high levels of resistance, integrated management packages are being developed for grey mould of both lentil and chickpea (Bayaa and Erskine, Chapter 8, and Haware, Chapter 9, this volume). For pigeonpea, strategies for integrated management are recommended for the management of multiple diseases and, especially, for soilborne diseases (Reddy *et al.*, Chapter 10, this volume). The plethora of tropical pasture legumes, the complexity of diverse pasture environments, and the potential for multiple interactions between pathogens, hosts, environmental factors and the grazing animal necessitate the development of integrated management strategies where resistance to diseases is not adequate (Lenné, Chapter 13, this volume).

The history of the use of integrated management strategies in traditional farming systems has been extensively reviewed by Thurston (1992). More recently, investigation of farmers' management of common bean diseases in the Great Lakes region of Africa found that local strategies were based on microclimate regulation (through sowing density, time of sowing, choice of soil type, foliage reduction, weeding and staking), genetic diversity (use of species and varietal mixtures) and sanitation (seed selection and removal of debris) (Trutmann *et al.*, 1993). In these systems, it was concluded that enhanced disease management should be possible through improved resistance while maintaining variability and improved seed health, but emphasis should be given to technologies which did not decrease the existing management flexibility. Many traditional systems offer additional options to disease management in modern systems.

Changes will occur in priorities given to legume pathogens as management strategies are successful in controlling the most important diseases; as cropping systems change (e.g. reduced periods of fallow and rotation or movement from productive to marginal lands due to pressure for land) and soilborne pathogens, in particular, become more important (see Haware, Chapter 9, this volume); and as legume crops move into different regions with different climatic conditions (e.g. moving chickpea from the spring to the winter season in the Mediterranean region resulted in severe epidemics of ascochyta blight (Hawtin and Singh,

1984). It is clear that flexibility will be needed in choice of target diseases as well as management strategies.

COLLABORATION

Organization of Research on Legume Pathogens

In both developed and developing countries, research on legume pathogens is generally organized on a crop by crop basis. In some cases, a crop may have its own institute (e.g. the International Winged Bean Institute in Sri Lanka). In other cases, grain, oil-seed and pasture legumes may be grouped at different institutes and subdivided into separate programmes on each crop (e.g. ICRISAT). Oil-seed legumes are more commonly grouped with non-leguminous oil-seed crops such as sunflower, rape and mustard, as at the National Oil Seed Crop Institute in India. Pathologists usually work in multidisciplinary teams with other scientists on a specific crop but, in general, there are few formal structures for collaboration among pathologists working on different legume crops. Such an organizational structure can result in isolation and fragmentation of effort and is not conducive to collaboration between pathologists working on different crops which may share the same diseases. The result is a 'crop myopia' as already mentioned several times throughout this volume.

Common Pathogens Link Different Crops

That the same pathogen may affect a number of legumes, and that the same genus of pathogen may have a very wide host range among grain, oil-seed and pasture legumes, has been emphasized on numerous occasions in this volume. For example, *Rhizoctonia solani* causes seedling or foliar blight and/or root rot of soyabean, common bean, cowpea, pea, chickpea and tropical pasture legumes (see Chapters 3, 5, 6, 9 and 13, this volume); root-knot nematodes are important on groundnut and tropical pasture legumes (see McDonald *et al.*, Chapter 2 and Lenné, Chapter 13, this volume); while *Botrytis cinerea* causes grey mould of both lentil and chickpea (see Bayaa and Erskine, Chapter 8, and Haware, Chapter 9, this volume). Similarly, *Fusarium* spp. are widespread pathogens of soyabean, pea, faba bean, lentil, chickpea, pigeonpea and clovers causing diseases such as wilt, collar, root and pod rot (see Chapters 3, 6, 7, 8, 9, 10 and 12, this volume), as are species of *Ascochyta* and *Phoma* which are major pathogens of groundnut, common bean, cowpea, pea, faba bean, lentil and chickpea (see Chapters 2, 4, 5, 6, 7, 8 and 9, this volume).

For some crops (e.g. soyabean, common bean and groundnut) a considerable amount of research has been done on the biology, epidemiology and management of some of the above pathogens. This can benefit research programmes on the same pathogens of legume crops such as lentil, chickpea, pigeonpea and tropical pasture legumes where more limited studies have been made. This is especially important for diagnosis and identification, where advanced serological and

molecular technologies have broad application, as well as in the utilization of generic models for crop loss assessment. Where certain pathogens on well-researched crops have proven very difficult to manage by single strategies and, as a result, integrated management packages have been developed, if the same pathogen occurs on less well-researched crops, existing packages may be readily modified and applied in preference to costly re-development of a similar package. Pathologists need to be increasingly aware of the advances made in legumes other than the legume on which they may be specifically working. As advances continue to be made with marker technology, as in soyabean (Kanazin *et al.*, 1996; Yu *et al.*, 1996), the possibility of moving resistance genes from one legume to another may become reality. A comprehensive knowledge of common pathogens across crops will be needed to assess the likelihood of success of this approach.

Difficulties Restricting Collaboration

No matter how individual pathologists may try to enhance collaboration with their colleagues, several factors may restrict the level of collaboration which can be achieved. The relative importance of specific legumes in specific countries will determine the involvement of the research, the private sector and the availability of funding, especially in developed countries. In developing countries, staple cereal grain crops such as maize, rice, wheat and sorghum are often given higher priority and the greatest share of limited research funds relative to legume crops. In many developing countries, research on legumes is given low priority.

Although most of the major diseases of common legumes are widespread, some diseases may be restricted to certain continents. This may be a reflection of the centre of origin of the host–pathogen association but in some cases it reflects new encounter associations, topics we have discussed earlier in this chapter. Quarantine restrictions can be a barrier to international collaboration on diseases of restricted geographical distribution. However, this may be resolved through collaboration with a third country in which the specific legume crop is not grown, for example ICRISAT's collaboration with the Natural Resources Institute (NRI) and the Scottish Crops Research Institute (SCRI), UK, on African groundnut viruses (see McDonald *et al.*, Chapter 2, this volume) and CIAT's collaboration with Horticulture Research International (HRI), UK, on global pathogens of common bean (see Allen *et al.*, Chapter 4, this volume).

Political and economic problems are major disincentives to the development of sustained research efforts required to solve many of the major disease problems of legumes (e.g. programmes breeding for resistance to major pathogens). Donors are becoming less likely to fund projects in countries with such problems, due to the reduced likelihood of impact of the research. Successful international collaboration depends on all partners having an understanding of the difficulties faced by their colleagues, especially those from developing countries. Lack of understanding between scientists from both developed and developing countries of economic, cultural and political issues, as well as lack of understanding of the farming systems and agroecological conditions in their respective countries, may make collaboration less effective.

Potential Collaborative Mechanisms

During the past 25 years, International Agricultural Research Centres (IARCs), USAID-funded Collaborative Research Support Programs (CRSP) through US universities, such as INTSOY and the Peanut and Bean–Cowpea CRSPs, as well as ACIAR-funded efforts through Australian institutes on tropical pasture legumes, have all developed extensive collaborative, international research on many grain, oil-seed and pasture legumes of importance in world agriculture. The extent of this pioneering effort is not always fully realized or appreciated (Summerfield and Roberts, 1985). As a result, disease-resistant cultivars of major grain and pasture legumes have been widely disseminated throughout developing countries via international trials networks, and superior cultivars are being increasingly adopted.

Research into the understanding and management of diseases of most major grain, oil-seed and pasture legumes in the great diversity of farming systems in which they are produced is included in the programmes of five of the CGIAR centres: CIAT (common bean and tropical pasture legumes), ICARDA (lentil, faba bean and chickpea), ICRISAT (groundnut, chickpea and pigeonpea), IITA (cowpea), ILRI (clovers) as well as AVRDC (soyabean and mung bean). Legume germplasm is collected, maintained, evaluated and distributed through international nurseries of the most disease-resistant germplasm, and technical information is disseminated through workshops, conferences and information services (Summerfield and Roberts, 1985). All of these programmes collaborate wherever possible with legume scientists in national programmes, and strong emphasis is placed on training, in accordance with the needs of the collaborating country.

With the current serious funding crisis now being faced, for example by CIAT, ICRISAT and the Peanut CRSP, considerably less funding is now available for legume research. The potential for CIAT and ICRISAT to sustain alone collaborative initiatives on common bean and tropical pasture legumes, and groundnut, chickpea and pigeonpea, respectively, is being severely eroded. Institutes in developed countries with skilled scientists, modern equipment and advanced technologies, and relatively more secure funds must now become proactive in international collaboration with IARCs and national programmes in developing countries to strongly support collaborative research projects on problems of mutual interest. One such mechanism is the ODA Holdback Facility which is a powerful initiative to build strategic linkages between UK scientific institutes, IARCs and NARS. Other donors should follow this successful model.

This can also be effectively accomplished through networks, a rapidly growing mechanism, as funding tightens and the benefits of collaboration are realized. International networking in agricultural research is not new (Plucknett and Smith, 1984). Pathologists have always cultivated informal contacts for exchange of information, pathogen isolates, diagnostic tools, or breeding lines. Informal associations sometimes develop later into formal networks as needs are better defined and technology and product exchange require a more formal structure. As funding to agricultural research continues to decrease, international networks may be the only way to ensure that goals are accomplished by cutting costs, avoiding duplication, optimizing resources and accelerating

efficient transfer of technology (Plucknett and Smith, 1984). Donors must continue to play an important role in sustaining networks.

Many international networks are now operating. A good example of a collaborative and productive network is the International Working Group on Groundnut Viruses which is managed by ICRISAT (Reddy *et al.*, 1994, 1997; Reddy and Gowda, 1996). This group has a membership of national programme virologists in Asia and Africa; scientists from advanced institutes in Europe, the USA and Australia; and ICRISAT virologists. It was initiated in 1983 and has met frequently since then to coordinate collaborative research on the most important groundnut viruses: spotted wilt and bud necrosis viruses, the groundnut rosette complex, peanut clump virus and peanut stripe virus. The meetings clearly show the progress which has been made in understanding and managing groundnut viruses globally. Funding from ICRISAT, Peanut CRSP, ODA, GTZ, the Belgian and Netherlands governments and others have supported this group, and, especially, the participation of developing country participants, over the past 14 years. However, funding is becoming more difficult to find. Progress made to date should be sufficient to convince donors that this network is the most efficient way of ensuring progress in managing virus diseases of groundnut.

Similar networks have been developed by CIAT, ICARDA and IITA. CIAT's Pan-African network for research on common bean has its origins in the Great Lakes Region of Central Africa, founded on support from the Swiss Development Cooperation (SDC) which began in 1983. An eastern Africa programme followed, with support from Canada and the USA, and, by 1987, a regional programme for southern Africa was also in place. Linkages across the three regions have developed and been strengthened in subsequent years so that now the three programmes operate as a single scientific network, with substantial devolution of management from CIAT to national scientists in planning, the allocation of resources, peer review of research progress and the exchange of germplasm and information. Now, the bean network in Africa is considered a model to be emulated by new networks (Kirkby, 1990). An example of a new potentially very successful network is the initiative on Integrated Pest Management (IPM). It is managed by the CGIAR Inter-Centre Working Group on IPM which is made up of representatives of IARCs, FAO and ARIs. Its main objective is to develop a coherent, coordinated CGIAR policy on IPM; strengthen inter-centre collaboration; identify priority global IPM opportunities and develop joint projects; and support IPM implementation through research and training (Anonymous, 1996). Although still in its infancy, it is a key global network for crop protection.

Training

There is a shortage of trained pathologists in developing countries. The need is for general training rather crop-specific training. Recognizing both this need and the opportunity, training courses and workshops have been run jointly in eastern and southern Africa, linking the crops groundnut, common bean and cowpea by inter-centre collaboration between ICRISAT, CIAT and IITA. Such initiatives need to be expanded. There are opportunities too for such cooperation in training in

virology at a regional level. The current scarcity of able and committed trainees reinforces the need for effective collaboration between institutions and their donors.

Funding

The current trend of reduced funding for agricultural research both internationally and nationally will limit the amount of research that can now be done on diseases of major legume crops. While every effort should be made to attract available research funds and to influence policy-makers and donors as to the critical importance of legumes in the diets of the world's poorest people, reduced funding may be expected also to have positive consequences: improved research efficiency, and enhanced international collaboration. For legume pathologists, this should break both the crop myopia and institutional isolation, and further foster linkages and networks. Research will need to be more targeted at the highest priority problems.

CONCLUSION

Grain legumes are essential nutritional components of the diets of most of the world's poorest people. However, despite considerable international effort during the past 25 years, their production has scarcely increased. Dramatic and widespread increases in production, similar to those of the 'Green Revolution' in cereal crops in several Asian countries, are not a realistic objective for legumes especially in subsistence production systems in which most are cultivated (Summerfield and Roberts, 1985). But modest increases which will provide food, increased nutritional status and cash for such farmers should be possible.

For oil-seed legumes such as soyabean and groundnut, the potential for increased production is higher, as illustrated by the increase in soyabean production in Brazil from the 1960s onwards (Summerfield and Roberts, 1985) and their growing role as cash crops in many developing countries. Similarly, the potential for increasing productivity of land not suitable for grain or oil-seed legumes through the use of improved pasture legumes is substantial in both temperate and tropical regions. Although this has been realized to some degree in temperate regions (e.g. Australia, Europe, USA) and in limited areas of the tropics (e.g. Australia, South America) there is considerable untapped potential.

This book has emphasized the importance of diseases in the failure to increase food and pasture legume production in spite of considerable research effort. It has also stressed that diseases cannot be considered in isolation, especially in subsistence production systems where many biotic and abiotic constraints and their complex interactions prevent legume crops from achieving their yield potential. Management of diseases in such systems requires an holistic understanding of the system and especially the importance of diseases relative to other biotic and abiotic constraints. This can be achieved if collaboration between pathologists, entomologists, breeders, agronomists, soil scientists and

farmers is strengthened. Without this understanding, unrealistic expectations of yield increases through disease management alone will be fostered. There is an urgent need to develop widely applicable disease management methodologies, based on a detailed understanding of pathogen biology and its interaction with other key parameters of the system, which can be modified for specific problems. Such an approach will make more efficient use of limited research funds.

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APPENDIX
Acronyms and abbreviations used in the text

ACIAR	Australian Centre for International Agricultural Research
AFLP	amplified fragment length polymorphism
ARI	Agricultural Research Institute
AVRDC	Asian Vegetable Research and Development Center
CABI	Centre for Agriculture and Biosciences International
CGIAR	Collaborative Research Support Program
DFID	Department for International Development (formerly ODA)
DNA	deoxyribose nucleic acid
ELISA	enzyme-linked immunosorbent assay
FAO	Food and Agriculture Organization
GIS	geographic information systems
GTZ	Deutsche Gesellschaft für Technische Zusammenarbeit
HRI	Horticulture Research International
IARC	International Agricultural Research Centre
ICARDA	International Center for Agricultural Research in Dry Areas
ICRISAT	International Crops Research Institute for the Semi-Arid Tropics
IITA	International Institute of Tropical Agriculture
ILRI	International Livestock Research Institute
INTSOY	International Soybean Program
IPM	integrated pest management
NARS	National Agricultural Research System
NIAB	National Institute of Agricultural Botany
NRI	National Resources Institute
ODA	Overseas Development Administration (now DFID)
PCR	polymerase chain reaction
RAPD	random amplified polymorphic DNA
RFLP	restriction fragment length polymorphism
RNA	ribonucleic acid
SCRI	Scottish Crops Research Institute
SDC	Swiss Development Corporation
USAID	US Agency for International Development

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