ADVANCES IN AGRONOMY, VOL. 45

ADVANCES IN DISEASE-RESISTANCE BREEDING IN CHICKPEA[1]

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I. INTRODUCTION

Chickpea (*Cicer arietinum* L.) is a diploid species with $2n = 16$ chromosomes. It is a self-pollinated crop with natural cross-pollination ranging between 0 and 1% (Singh, 1987). Most probably chickpea originated in southeastern Turkey (Ladizinsky, 1975). There are two types of chickpea: desi (local), characterized by small, angular, colored seeds; and kabuli (an allusion to origin in the Afghani capital, Kabul, before it reached India), characterized by large, ram-head-shaped, beige-colored seeds. The desi type is primarily grown in the Indian subcontinent and East Africa, and the

[1] [redacted] 1124 joint contribution from the International Center for Agricultural Research in the Dry Areas (ICARDA), Aleppo, Syria, and the International Crops Research Institute for the Semi-Arid Tropics (ICRISAT), Patancheru, Andhra Pradesh 502 324, India.

kabuli type is mostly grown in the Mediterranean region and Central and South America. It is believed that the small-seeded desi type is the original form of chickpea and that the kabuli type developed through mutation.

The chickpea is grown primarily on conserved moisture and rarely receives fertilizers or protection from diseases and insect pests. The protein content of the seed is comparatively low (23%), but its biological value is the best among pulses. Chickpea is consumed as fresh, immature green seed, whole seed, dhal, and flour.

Of the food legumes, chickpea ranks second in area and third in production. It was grown on 9.6 million ha with a production of 6.7 million t from 1986 to 1988. It is an important crop in South and West Asia, and is also grown in Central and South America, East Africa, North Africa, and southern Europe. The average per hectare production of 704 kg/ha is low (Food and Agriculture Organization, 1988), a major cause being susceptibility of land races to diseases.

Diseases can be controlled by application of fungicides, by cultural practices, or by use of host-plant resistance. Although effective fungicides have been identified (Hanounik and Reddy, 1984), they are often impractical. Modification of cultural practices can often reduce yield loss from diseases, but yield per se also may be reduced. Hence, the best strategy to control diseases is through use of resistant cultivars. The purpose of this article is to review the past work on disease resistance breeding in chickpea and to discuss strategies to tackle unsolved disease problems.

II. SOURCES OF GENETIC VARIABILITY

Sufficient genetic variability exists in the chickpea germplasm collections maintained at national, regional, and international genetic resources centers (Malhotra *et al.*, 1987; Pundir *et al.*, 1988; Singh *et al.*, 1983). The largest collection (15,945 accessions) is maintained at ICRISAT (International Crops Research Institute for the Semi-Arid Tropics) Center in India (R. P. S. Pundir, personal communication) and the second largest collection (over 8,000 accessions) is maintained at ICARDA (International Center for Agricultural Research in the Dry Areas), Syria (L. Holly, personal communication). Granted that some accessions are common to both collections, total accessions exceed 20,000. Evaluation of 5,000 to 15,000 accessions for reaction to six biotic and abiotic stresses at ICARDA resulted in identification of sources resistant to all except seed beetle and cyst nematode (Singh, 1989). The most extensive germplasm evaluation has been for resistance to *Ascochyta* blight and *Fusarium* wilt. Germplasm

lines maintained at ICRISAT and ICARDA warrant further evaluation for resistance to other diseases.

III. BREEDING TECHNIQUES

Methods for breeding disease-resistant chickpea cultivars are similar to those used for yield breeding, except that the segregating materials are challenged by the pathogen and selection is made for disease resistance along with other attributes. Some of the techniques employed by breeders are as follows:

1. *Selection from introductions.* Selection from introductions is a potent method of breeding, especially for countries with limited resources or area. Following this technique, Karachi was released as a wilt-resistant cultivar in Myanmar, Burma in 1923; Lebanon released Janta 2 as an *Ascochyta* blight-resistant cultivar in 1989. Many cultivars have been released in the intervening period.
2. *Hybridization.* Resistance breeding usually begins with selection from introductions, but subsequently it is dominated by hybridization as this offers an opportunity to combine desirable traits from two or more parents in one line. In chickpea, hybridization is followed by three breeding methods: (1) pedigree, (2) bulk/population, and (3) backcross. Combinations of these methods, such as bulk–pedigree and backcross–pedigree, are commonly adopted.
3. *Mutation.* Mutation techniques have been used to create new variability, but sometimes even cultivars have been developed.

IV. DISEASE RESISTANCE

Chickpea is subject to numerous diseases. Nene *et al.* (1989a) listed 115 pathogens known to infect chickpea, including fungi, bacteria, viruses, mycoplasmalike organisms, and nematodes. Fortunately, only a few of them cause economic losses, but in certain areas they severely limit chickpea production. Some diseases such as wilt, root rots, *Ascochyta* blight, and *Botrytis* gray mold can cause major losses and prevent farmers from realizing the potential yield of the crop. This is because farmers do not implement necessary practices to prevent losing the crop by diseases.

Though work on diseases such as *Ascochyta* blight and wilt has been

conducted since the beginning of the century, research effort has only occurred over the past 15 years. The establishment of international agricultural institutes such as ICRISAT and ICARDA, in which chickpea is a mandated crop, has given momentum to research on chickpea diseases. Also, national research programs in India and Pakistan, where chickpea is an important grain legume crop, have increased efforts considerably during the past 10–15 years.

Though considerable progress has been made in understanding and managing some diseases, more research is needed. Since chickpea disease research has been reviewed in detail by several workers (Greco and Sharma, 1991; Haware *et al.*, 1991; Kaiser *et al.*, 1991; Nene and Reddy, 1987; Reddy *et al.*, 1991), the scope of this paper will be restricted to summarizing host-plant resistance research on the most important chickpea diseases.

A. Fungal Diseases: Soil-Borne

Fungal diseases are by far the most important. These diseases can be broadly divided into two groups: soil-borne and foliar. Soil-borne diseases are relatively more serious in the lower latitudes (0–20°) where the chickpea growing season is short, warm, and dry. Foliar diseases are more important in higher latitudes (20–40°) with relatively long, cool, and wet growing seasons. Soil-borne diseases, such as wilt and root rots, occur regularly, whereas foliar diseases, such as *Ascochyta* blight, do not occur every season, but only when rain occurs during the cropping season. Losses from soil-borne diseases are not high; however, when foliar diseases occur in epidemic form the entire crop is usually destroyed.

Chickpea suffers from several major soil-borne diseases (Table I) including wilt, root rots, and stem rots. Very often more than one disease occurs in the same field; a single plant may be infected by more than one disease. The disease may affect the crop from seedling stage to maturity.

1. Fusarium *Wilt* [Fusarium oxysporum *Schlect. emend Snyd. & Hans f.sp.* ciceri *(Padwick) Snyd. & Hans.*]

a. General Description of Disease. *Fusarium* wilt, the most important soil-borne disease, is prevalent in most chickpea-growing countries (Table I). It is a typical vascular disease causing xylem browning or blackening. The disease affects the crop at all stages. The expression of symptoms is most rapid at high temperatures (>30°C). A susceptible cultivar (e.g., JG

Table I

Important Soil-borne Fungal Diseases of Chickpea and Their Distribution[a]

Disease	Causal organism	Countries where prevalent
Fusarium wilt	*Fusarium oxysporum* Schlecht. emend Snyd. & Hans. f.sp. *ciceri* (Padwick) Snyd. & Hans.	Algeria, Argentina, Australia, Bangladesh, Chile, Colombia, Ethiopia, India, Iran, Iraq, Kenya, Malawi, Mexico, Morocco, Myanmar (Burma), Nepal, Pakistan, Peru, Spain, Sudan, Syria, Tunisia, U.S.A.
Verticillium wilt	*Verticillium dahliae* Reinke & Berth	Pakistan, Tunisia, U.S.A.
Dry root rot	*Rhizoctonia bataticola* (Taub.) Butler [*Macrophomina phaseolina* (Tassi) Goid.]	Australia, Bangladesh, Ethiopia, India, Iran, Kenya, Lebanon, Mexico, Pakistan, Spain, Syria, U.S.A.
Collar rot	*Sclerotium rolfsii* Sacc.	Bangladesh, Colombia, Ethiopia, India, Mexico, Pakistan, Syria
Wet root rot	*Rhizoctonia solani* Khun	Argentina, Australia, Bangladesh, Chile, Ethiopia, India, Iran, Mexico, Morocco, Pakistan, Syria, U.S.A.
Black root rot	*Fusarium solani* (Mart.) sacc.	Argentina, Chile, India, Mexico, Spain, Syria, U.S.A.
Phytophthora root rot	*Phytophthora megasperma* Drechs.	Argentina, Australia, India, Spain
Pythium root and seed rot	*Pythium ultimum* Trow	India, Iran, Turkey, U.S.A.
Foot rot	*Operculella padwickii* Kheshwalla	India
Stem rot	*Sclerotinia sclerotiorum* (Lib.) de Bary	Algeria, Australia, Bangladesh, Chile, India, Iran, Morocco, Pakistan, Syria, Tunisia, U.S.A.

[a] From Nene *et al.* (1989a).

62) under such conditions may be killed within 15 days of sowing in a wilt-infested field. The freshly wilted plants show drooping of the foliage, but retain their green color. In tolerant cultivars (e.g., K 850), the disease causes general yellowing and drying of the lower leaves and late wilting. The root systems of wilted plants do not show any apparent symptoms.

Losses from wilt have not been estimated precisely. In India, the disease is suspected to cause about 10% loss annually (Singh and Dahiya, 1973).

Sattar *et al.* (1953) estimated a loss of about U.S. $ 1 million annually due to wilt in Pakistan. Estimating losses due to wilt alone in farmers' fields is difficult as it is usually accompanied by root and stem rot diseases. Wilt initially appears in a field in small patches; these patches enlarge if chickpea is cultivated in the same field year after year. In soils favorable to *Fusarium oxysporum* f.sp. *ciceri*, the field becomes completely infested within three seasons.

The wilt pathogen is both seed and soil borne and can survive in soil up to six years in the absence of a host plant. The fungus has been found to have distinct physiologic races; seven races have been reported from India, Spain, and the United States (Haware and Nene, 1982; Jimenez-Diaz *et al.*, 1989; Phillips, 1988).

b. Sources of Resistance. Field, greenhouse, and laboratory inoculation techniques have been standardized for screening chickpeas for wilt resistance (Nene *et al.*, 1981). Effective "sick plots" have been developed in almost all the important chickpea growing countries, including Bangladesh, Ethiopia, Mexico, Morocco, Myanmar (Burma), Nepal, Peru, Spain, Tunisia, and the United States. Lines resistant to *Fusarium* wilt have been identified in all these countries. A few lines with broad-based resistance to wilt, such as ICC 2862, ICC 9023, ICC 9032, ICC 10803, ICC 11550, and ICC 11551, also have been identified (Nene *et al.*, 1989b). Although resistant lines are not killed, they show internal blackening or browning indicating fungal infection. The mechanism of resistance to wilt is not fully understood. Exudates from susceptible cultivars such as JG 62 are known to stimulate mycelial growth and germination of conidia and chlamydospores, while exudates from the resistant cultivar CPS-1 inhibited these processes (Satyaprasad and Ramarao, 1983).

c. Genetics of Resistance to Fusarium *Wilt.* Knowing the genetics of resistance to diseases helps plant breeders eliminate or reduce yield losses through appropriate breeding strategies. Ayyar and Iyer (1936) were first to report that a single recessive gene conferred resistance to *Fusarium* wilt in chickpea; this finding was confirmed later by several studies (Table II). Lopez Garcia (1974) presented evidence that two pairs of recessive genes controlled the genetics of resistance to *Fusarium* wilt.

Upadhyaya *et al.* (1983a) reported that different chickpea genotypes varied as to the time required before the initial symptoms of *Fusarium* wilt appeared. In particular, C-104 wilts much later than JG-62; the difference appears to be controlled by a single gene. Upadhyaya and co-workers (1983a) found that at least two genes control resistance to race 1. Further studies by Upadhaya *et al.* (1983b) confirmed that the cultivar C-104

Table II

Inventory of Inheritance of Resistance to *Fusarium* Wilt (*Fusarium oxysporum* f.sp. *ciceri*) in Chickpea

Nature of inheritance	Genotype	References
Incomplete dominance, single gene	Strain No. 468	Ayyar and Iyer (1936)
Two pairs of recessive genes	19 Lines	Lopez Garcia (1974)
Single recessive gene	Strain 315	Pathak *et al.* (1975)
Single recessive gene	9 Lines	Haware *et al.* (1980)
Single recessive gene	JG 315	Tiwari *et al.* (1981)
Single recessive allele	WR 315, CPS1	Kumar and Haware (1982)
Monogenic recessive gene	7 Desi lines	Phillips (1983)
Single recessive gene	1231, 32-35-8/7	Sindhu *et al.* (1983)
Three independent loci designated H_1, H_2, and H_3	P 436-2, C PS1, WR 315, BG 212	Smithson *et al.* (1983)
Two recessive genes to race 1	JG 62, C 104, H 208, K 850	Upadhyaya *et al.* (1983a)
Two recessive genes to race 1	JG 62, C 104, H 208, K 850	Upadhyaya *et al.* (1983b)
Digenic nature of wilt resistance	—	Singh *et al.* (1986)
K 850 and C 104 each carry independent recessive allele	K 850, C 104	Singh *et al.* (1987)

appears to differ from WR-315 and CPS-1 by a single locus, which results in delayed wilting when in homozygous recessive form. The same researchers also suggested that data are consistent with the hypothesis that JG-62 carried the two genes in a homozygous dominant condition (H_1 H_1 H_2 H_2); C-104 is homozygous recessive at the second locus (H_1 H_1 h_2 h_2); and the resistant parents (WR-315, CPS-1, BG-212, and P-436-2) are homozygous recessive at both loci (h_1 h_1 h_2 h_2). Singh *et al.* (1987) reported that K-850 carried a recessive gene that is different than and independent of the gene in C-104 and that the two together confer complete resistance. Thus, K-850, like C-104, is a late-wilting cultivar. Early wilting is partially dominant over late wilting. They concluded that at least two loci control resistance to race 1. Unpublished data from H. Singh suggests that a third locus may be involved. Singh *et al.* (1988) have found a digenic nature of wilt resistance with epistasis.

Clearly, the inheritance of resistance to *Fusarium* wilt is not simple. All studies at ICRISAT Center have been made against race 1 of *F. oxysporum* f.sp. *ciceri*. The existence of at least four races has been reported from India (Haware and Nene, 1982). The situation may be complicated further if a study is made against two or more races. Further, resistant plants have

been recovered from crosses between two susceptible parents, indicating a complementary type gene action (Singh *et al.*, 1987). Singh and coworkers have suggested that chickpea germplasm may be classified in three categories: early wilter, late wilter, and resistant.

d. Breeding for Fusarium *Wilt Resistance.* Recognizing the severity of wilt in Myanmar, McKerral (1923) evaluated a large number of Burmese and introduced collections for resistance to wilt and yield. Based on resistance and superior yield performance, he released a cultivar, Karachi, which was subsequently grown extensively. While reviewing 50 years of progress in pulse research in Bombay state of India, Chavan and Shendge (1957) described the development of four wilt-resistant cultivars: Dohad-206-8, Dohad-1597-2-1, Chaffa Tr. 1-7, and Nagpur Tr. 1-2. These cultivars produced more seed yield than Chaffa in fields infested by the wilt pathogen, but produced less seed yield than Chaffa in wilt-free fields. This was a common feature of all resistant lines developed in the early years of breeding. As a result, wilt-resistant cultivars never became popular with farmers. In Pakistan, Khan (1954) developed C 612 from an F 8 × C 144 cross. This cultivar had the same yield potential as previously released cultivars.

Breeding for wilt-resistant cultivars in India was a discontinuous effort (Singh, 1974). Singh summarized the work on breeding for wilt resistance carried out between 1943 and 1953 and stated that, in the absence of an efficient screening technique, only limited progress was made. G 24, a cultivar from Punjab, India, was reported to be resistant to wilt. Singh listed 17 lines that were reported resistant in India up to 1974. Kanpur (India) has the distinction of being the first place in the world where a wilt-sick plot was established (Singh *et al.*, 1974). Several hundred lines were screened in this nursery and 12 lines were identified as resistant. Of these, strain Nos. 100, 101, 106, and 6002 were crossed with high-yielding lines T_1, T_2, and T_3, and promising lines were developed.

Outside the Indian subcontinent, Mexico is the only country where concentrated wilt-resistance breeding has been practiced. Singh (1987) reviewed work conducted in Mexico and reported that three large-seeded kabuli chickpea cultivars, Surutato 77, Sonora 80, and Santo Domingo, were bred following the hybridization technique; a wilt-sick plot established at Culiacan in 1960 was utilized. Later, the wilt-sick plot was found to be infested with other soil-borne diseases and viruses (I. W. Buddenhagen, personal communication).

Despite progress made in resistance to wilt, confusion existed in the identity of the causal organism of wilt disease. To tackle this problem, a

symposium on gram (chickpea) wilt was organized in 1973 at New Delhi and the recommendations were summarized by Jain and Bahl (1974). The need to distinguish the differences between wilts caused by pathogens and wilts caused by environmental factors was stressed. It was clear that as late as 1973, the mystery of the wilt complex remained unresolved, an indication that whatever progress made until then was not based on sound scientific knowledge. The work of Nene *et al.* (1978) at ICRISAT, India, resolved the mystery of the wilt complex. Wilt, it was clearly suggested, is caused by several distinct pathogens and not by environmental factors. Among the pathogens, wilts and a number of root rot and stem rot diseases were separated out. This work helped put *Fusarium* wilt-resistance breeding on a sound scientific footing.

Since 1980, wilt-sick plots have been established at many research centers including ICRISAT Center and Ludhiana (India), Faisalabad (Pakistan), Beja (Tunisia), Santella (Spain), Debre-Zeit (Ethiopia), and the Central Valley (California, U.S.A.). The establishment of wilt-sick plots in these and other places has facilitated planned breeding programs and led to the breeding of a number of high-yielding, wilt-resistant cultivars. Singh (1987) listed chickpea cultivars released up to 1984; Table III presents an updated list of resistant released cultivars.

Fusarium wilt-resistant cultivars have been bred by researchers in many countries (Table III), but they have seldom been grown on a large scale by farmers for two reasons. First, *Fusarium* wilt incidence in the field is usually associated with other soil-borne diseases. Wilt-resistant cultivars are thus affected by other soil-borne diseases. Second, most breeders have developed race-specific resistant genotypes, whereas different races of *F. oxysporum* f.sp. *ciceri* have been identified from various locations within countries (Haware and Nene, 1982; Jimenez Diaz *et al.*, 1989).

This suggested that breeders and pathologists should consider a different strategy. First, they should pyramid genes for resistance to different races in one line for use in hybridization programs. Second, soil-borne disease-sick plots should be developed rather than wilt-sick plots as in the past. In the soil-borne disease-sick plot, pathologists could then incorporate in the plot pathogens of *Fusarium* wilt, root rots, and other soil-borne diseases, including nematodes, that are prevalent in the region. To some extent, this is being done at ICRISAT. Sick plots for wilt and dry root rot have been developed. Pathologists and breeders together should screen germplasm lines in the soil-borne diseases-sick plot, identify sources of resistance, and use these in hybridization programs to breed resistant lines.

Table III

List of Disease-Resistant Cultivars Released between 1923 and 1989[a]

Country	Disease	Cultivar
Algeria	Blight[b]	ILC 482, ILC 3279
Bangladesh	Unspecified	Sabour-4, Fatehpur-1, Bhaugora
Bulgaria	Blight	Plovdiv 19, Obraztsov, Chijlik-1, Plovdiv 8
Chile	Root rot	California-INIA, Guasos-SNA
Cyprus	Blight	Yialousa, Kyrenia
France	Blight	TS 1009, TS 1502
India	Blight	F 8, C 12/34, C 235, G 543, Gaurav, BG 261, GNG 146, PBG 1
	Wilt[c]	C 612, S 26, G 24, C 214, G 130, H 208, H 355, GL 769, Pusa 212, Pusa 244, Pusa 256, Pusa 408, Pusa 413, Pusa 417, JG 315, Avrodhi
Italy	Blight	Califfo, Sultano
Lebanon	Blight	Janta 2
Mexico	Wilt	Surutato-77, Sonora-80, Santo Domingo
Morocco	Blight	ILC 195, ILC 482
Myanmar	Wilt	Karachi
Pakistan	Blight	F 8, C 12/34, C 235, C 727, CM 72, C 44, AUG 480
	Wilt	C 612
Portugal	Blight	Elmo, Elvar
Spain	Blight	Alcazaba, Almena, Atalaya, Fardan, Zegri
Syria	Blight	Ghab 1, Ghab 2
Tunisia	Blight	Chetoui, Kassab
	Wilt	Amdoun 1
Turkey	Blight	ILC 195, Guney Sarisi 482, Damla 89, Tasova 89
U.S.A.	Root rot	Mission
	Wilt	UC 15, UC 27
U.S.S.R.	Blight	Alpha, Mugucii, Skorospelka, Vir 32, Nut Zimiston

[a] From Nene *et al.* (1989a).
[b] *Ascochyta* blight.
[c] Mainly *Fusarium* wilt.

2. Verticillium *Wilt* (Verticillium albo-atrum *Reinke & Berth*)

Verticillium wilt has been reported from Pakistan, Tunisia, and the United States. Both *Fusarium* and *Verticillium* wilts were found to occur in the same field and plant in Tunisia. *Verticillium* wilt is difficult to distinguish from *Fusarium* wilt, based on symptoms. Sources of resistance to *Verticillium* wilt have been reported from Tunisia (Halila and Harrabi, 1987).

3. Dry Root Rot [Rhizoctonia bataticola (*Taub.*) *Butler* = Macrophomina phaseolina (*Tassi*) *Goid.*]

Dry root rot is the most important and widely spread root rot affecting chickpea. Though infection can occur in the early stages of growth, maximum disease expression occurs from podding time onwards. The maximum disease incidence usually coincides with moisture stress and high temperature (>30°C), stresses that are favorable for disease development. Under field conditions, the disease is manifested as scattered dead plants, whereas wilt appears in patches. The root system of diseased plants shows extensive rotting with most of the lateral roots destroyed. Affected roots are brittle, and there is shredding of the bark. Sclerotial bodies of the fungus sometimes can be seen on the surface of the root or inside the wood.

Susceptibility of chickpeas to dry root rot increases with age. At ICRISAT, screening numerous germplasm lines in a wilt and root rot nursery helped identify a few chickpea lines, such as ICC 2862 and ICC 4023, having resistance to wilt and tolerance to dry root rot. In spite of extensive root rotting, these lines do not die until maturity. High levels of resistance may be difficult to develop as the pathogen has a very wide host range. Both wilt and dry root rot infections can be found in the same plant in wilt and root rot-sick plots at ICRISAT and Beja, Tunisia. Monogenic dominance was found to confer resistance to dry root rot (Ananda Rao and Haware, 1987).

4. Other Root and Stem Rots

Collar rot (*Sclerotium rolfsii* Sacc.), wet root rot (*Rhizoctonia solani* Kuhn), black root rot [*Fusarium solani* (Mart.) Appel & Wr.], stem rot [*Sclerotinia sclerotiorum* (Lib.) de Bary], phytophthora root rot (*Phytophthora megasperma* Drechs.), pythium root and seed rot (*Pythium ultimum* Trow), and foot rot (*Operculella padwickii* Kheshwalla) are the other important soil-borne diseases affecting chickpea. Most of these diseases mainly affect chickpea in the seedling stage when soil moisture is relatively high. Collar rot, wet root rot, black root rot, and stem rot are more widespread than the phytophthora and pythium root rots and foot rot.

Collar rot usually affects the crop in the seedling stage; susceptibility decreases with age. High soil moisture, presence of undecomposed organic matter on the soil surface, and high temperatures at sowing time favor disease development. The disease is usually a problem in areas

where chickpeas are sown following paddy. Kabuli types are more susceptible than desi types. Diseased plants show yellowing of foliage before death. They develop a cankerous lesion at the collar region, or rotting of most of the root system, which is covered with white mycelial growth and sclerotial bodies. Little research has been conducted on standardization of inoculation techniques, or on identification of resistance sources. Though a few lines are reported to be resistant under field conditions, their resistance under artificial inoculation remains unconfirmed. It may be difficult to obtain high levels of resistance to a fungus such as *S. rolfsii* having a wide host range.

Wet root rot is most likely to attack at the seedling stage, but can affect the crop in advanced stages of growth if the soil moisture level is high. The root system of affected plants shows rotting, which may extend up the stem.

In the case of black root rot, affected plants initially show a black cankerous lesion at the point of attachment of cotyledons to the stem. Rotting later extends to the whole root system. Wet, black root rots favor relatively lower temperatures (around 25°C) than those favored by collar rot. Little research has been directed toward inoculation techniques or identification of resistance sources.

Stem rot is a problem at higher latitudes where cool, wet weather prevails. Excessive vegetative growth favors its development. The disease may affect the collar region killing the whole plant, or may affect individual branches. Affected plants or branches turn chlorotic before dying. White mycelial growth and large irregular-shaped sclerotial bodies can be seen on the affected portions of the plant. At present, stem rot is not considered to be a serious disease. However, standardization of inoculation techniques and identification of resistance sources would be useful.

Phytophthora root rot has been reported from Argentina, Australia, India, and Spain. Disease symptoms include yellowing and drying of the foliage and decay of the lateral roots and the lower portion of the tap root. Lesions on the remainder of the tap root are dark brown to black and extend to and, in some cases, reach above ground level. The advancing margins of these lesions are often preceded by a reddish-brown discoloration (Vock *et al.*, 1980). Screening tests in Queensland, Australia, have revealed that some lines, such as CPI 56564, have field resistance.

India, Iran, Turkey, and the United States have reported pythium root rot and seed rot, but it is particularly a serious problem in the Palouse region, Washington, U.S.A. The disease is more common in kabuli types than in desi types. Seed rotting is usual. The fungus is pathogenic to the roots of chickpea seedlings, which become stunted. Larger roots are

necrotic and are devoid of feeder rootlets. Affected plants frequently die before flowering (Kaiser and Hannan, 1983).

Foot rot is reported only from India. The disease appears under wet soil conditions. Aboveground symptoms are similar to those of wilt; rotting of the root is evident from the collar region downward. Internal discoloration appears above the rotten portion, but this discoloration is brown and does not involve the pith as do *Fusarium* and *Verticillium* wilts (Nene *et al.*, 1978).

B. Fungal Diseases: Foliar

Foliar diseases seriously limit chickpea yields in several important chickpea-producing countries. Foliar diseases occur in areas (20°–40° latitude) that are otherwise highly suited to chickpea production. These areas usually receive winter rains during the crop season, which benefit crop growth but promote foliar diseases. Lack of precipitation eliminates the foliar diseases problem, but reduces yields due to drought. A relationship between chickpea yields and *Ascochyta* blight is shown in Fig. 1. Foliar diseases control is a prerequisite for increasing chickpea yields in these regions.

The most important foliar diseases are *Ascochyta* blight [*Ascochyta rabiei* (Pass.) Labr.], *Botrytis* gray mold (*Botrytis cinerea* Pers. ex. Fr.), *Alternaria* blight [*Alternaria alternata* (Fr.) Kiessler], rust [*Uromyces ciceris-arietini* (Grogn.) Jaj & Beyer], and Stemphylium blight [*Stemphylium sarciniforme* (cav.) Wilts.] (Table IV). Among these, *Ascochyta* blight occurs in slightly cooler (20°C) environments than the other diseases (25°C). While rain is essential for infection and spread of *Ascochyta* blight, the other foliar diseases can develop in its absence if high humidity is created in the crop canopy by irrigation, heavy dew, high soil moisture, or excessive vegetative growth.

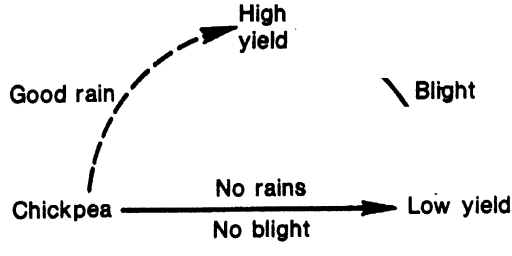

Fig. 1. Relationship between chickpea yield and *Ascochyta* blight.

Table IV

Important Foliar Fungal Diseases of Chickpea and Their Distribution[a]

Disease	Causal organism	Countries where prevalent
Ascochyta blight	*Ascochyta rabiei* (Pass.) Lab. (*Mycosphaerella rabiei* Kovachevski)	Algeria, Australia, Bangladesh, Bulgaria, Canada, Colombia, Cyprus, Egypt, Ethiopia, France, Greece, Hungary, India, Iran, Italy, Jordan, Lebanon, Mexico, Morocco, Pakistan, Portugal, Romania, Spain, Sudan, Syria, Tanzania, Tunisia, Turkey, U.S.A., U.S.S.R.
Botrytis gray mold	*Botrytis cinerea* Pers. ex Fr.	Argentina, Australia, Bangladesh, Canada, Colombia, India, Nepal, Pakistan, Spain, Turkey, U.S.A.
Alternaria blight	*Alternaria alternata* (Fr.) Kiessler	Bangladesh, India, Nepal
Stemphylium blight	*Stemphylium sarciniforme* (Cav.) Wills	Bangladesh, India, Iran, Syria
Rust	*Uromyces ciceris-arietini* (Grogn.) Jacz & Beyer	Algeria, Afghanistan, Bulgaria, Chile, Cyprus, Ethiopia, France, India, Iran, Lebanon, Libya, Malawi, Mexico, Morocco, Nepal

[a] From Nene *et al.* (1989a).

1. Ascochyta *Blight* [Ascochyta rabiei (*Pass.*) *Labr.*]

a. General Description of Disease. *Ascochyta* blight is by far the most destructive disease of chickpea. It is particularly serious in India, Pakistan, and the countries around the Mediterranean Sea. It does not occur every season, but usually in cycles of about 5 years. Once it does occur, it continues for 2–3 years.

The disease usually appears in epiphytotic form from the flowering stage onwards when temperatures are optimum for blight infection and development. Earlier in the season, temperatures are too low for disease development. The disease initially appears in small patches and, under favorable conditions (15 to 25°C, rains accompanied by winds and cloudy days), spreads very rapidly. Rain splash helps spread the disease. Figure 2 shows the relationship between temperature, humidity, and *Ascochyta* blight. When both temperature and relative humidity are optimum, *Ascochyta* blight develops in epiphytotic form.

The disease affects all aboveground parts of the plant. If the infection

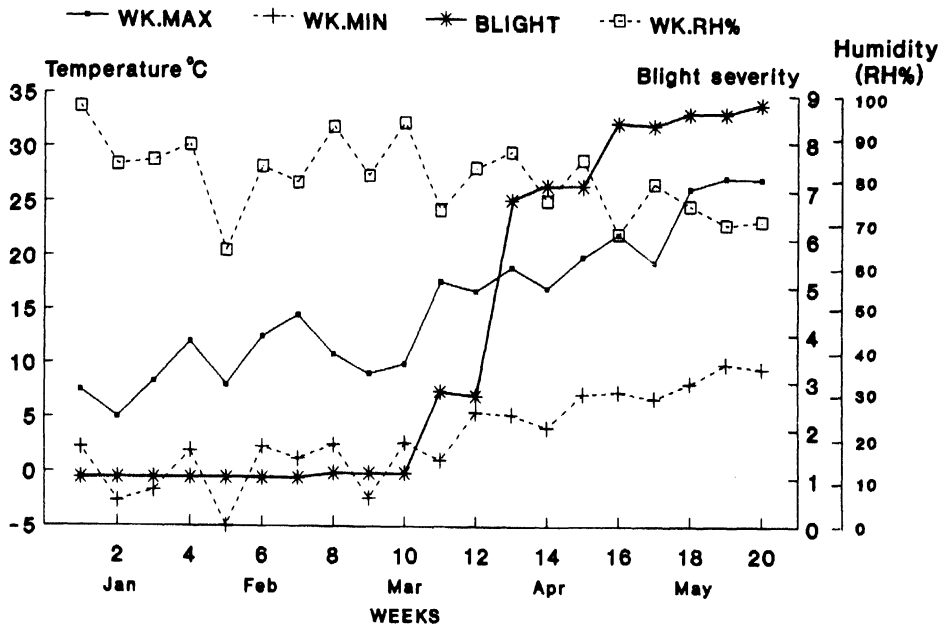

FIG. 2. Relationship between temperature, humidity, and chickpea *Ascochyta* blight at Tel Hadya, Syria, 1982–1983.

occurs through seed-borne inoculum, the seedlings show brown cankerous lesions at the collar region before they collapse. Symptoms on leaves and pods are circular spots with pycnidia of the fungus usually arranged in concentric rings. On stems, the lesions are elongated and, when the lesions engirdle the stem, portions above the lesions either dry up or break off. The pathogen infects seed and sometimes causes deep cankerous lesions.

Despite 50 years of efforts to manage the disease, *Ascochyta* blight continues to cause heavy losses. In Pakistan during the 1979–1980 season, the disease caused about 50% yield loss (Malik and Tufail, 1984), while in India during the same period, it was estimated to have destroyed the crop on about one million hectares.

The perfect state of the fungus (*Mycosphaerella rabiei* Kovachevski) is reported from a few chickpea-growing countries—Bulgaria, Greece, Hungary, Syria, the United States, and the Soviet Union (Gorlenko and Bushkova, 1958; Haware, 1987; Kaiser and Hannan, 1987; Kovachevski, 1936; Kovics *et al.*, 1986; Zachos *et al.*, 1963). The epidemiology of the disease is not clearly understood. Infected seed and diseased debris have long been known as primary sources of inoculum. In the United States, ascospores were found to play an important role in disease survival and spread (Kaiser and Muehlbauer, 1988).

Studies indicate that the blight pathogen is highly variable. Variation in the pathogen has been reported from all the major chickpea-growing countries such as India, Pakistan, Syria, and Turkey (Acikgoz, 1983; Bedi and Aujla, 1970; Qureshi, 1986; Reddy and Kabbabeh, 1985; Vir and Grewal, 1974). Frequent changes in pathogen virulence have resulted in a breakdown of resistance of several cultivars. There is further need for standardization of the method for race identification in *Aschocyta rabiei.*

b. Sources of Resistance. Efforts have considerably increased during the past 15 years to identify resistance sources and to breed resistant cultivars. Efficient inoculation techniques for use in greenhouse and field have been standardized. Inoculating plants grown in pots, bags, or trays and covering them with polyethylene or cloth bags or cages for 24–48 hr results in good infection. Temperatures congenial for infection range from 15 to 25°C. Presence of a moisture film on the leaf surface is essential for infection. In the field, inoculation by scattering diseased debris or spraying a spore suspension over plants followed by sprinkler irrigation results in a high and uniform disease level (Reddy *et al.,* 1984). Rating scales for scoring disease severity have been standardized.

Available chickpea germplasm has neither high nor stable resistance to all the prevalent races of *A. rabiei* (Singh *et al.,* 1984). In general, pods are more susceptible than vegetative parts. Lines such as PK 51836 × NEC-138-2 show resistance in the vegetative stage against a wide range of isolates, but are not resistant to pod infection. Several lines with foliage resistant to isolates prevalent in the countries around the Mediterranean Sea have been identified (Singh *et al.,* 1984; Reddy and Singh, 1984). Through the Chickpea International *Ascochyta* Blight Nursery, resistant lines have been evaluated in blight-prone areas between 1979 and 1989 and a few lines with broad-based resistance have been identified. These include kabuli types (ILC 72, ILC 182, ILC 187, ILC 196, ILC 200, ILC 202, ILC 2506, ILC 2956, ILC 3279, ILC 3346, ILC 3866, ILC 3868, ILC 4421) and desi types (ICC 5035, ICC 5566, ICC 6304, ICC 7028, Pch 70, NEC 138-2) (K. B. Singh and M. V. Reddy, unpublished data). However, there are no lines in India and Pakistan that have a high and stable level of resistance. Most of the lines that showed resistance in the vegetative and podding stages in the Mediterranean region are tall, erect, and late maturing. Preliminary studies carried out at ICARDA in Syria showed that when plants of these lines had their stems bent over mechanically, some developed a higher level of infection on pods.

c. Genetics of Resistance to Ascochyta *Blight.* Reported inheritance studies results indicate that resistance is conferred by either a single

dominant gene or a single recessive gene (Table V). Allelic studies by Tewari and Pandey (1986) indicated the presence of three independent dominant genes in EC 26446, P 1252-1, and PG82-1. Similarly, Halila *et al.* (1989) found that ILC 182, ILC 191, and ILC 482 had an independent dominant gene, though Singh and Reddy (1989) found through allelic tests that the same dominant resistant gene was present in ILC 72, ILC 202, ILC 2956, and ILC 3279. The variations in reaction of four resistant lines, when tested in 13 countries and against six races of *A. rabiei*, appeared to be due to the presence of some other resistance genes in addition to a common gene.

All inheritance studies have been made in the field or against a single isolate or race of a given country. Further, genotypic reactions at the seedling and podding stages of the plant vary. Temperature and relative humidity also influence disease reaction. Duration of favorable disease development conditions also influences disease reaction.

d. Breeding for Ascochyta *Blight Resistance.* *Ascochyta* blight resistance breeding began in the 1930s in India and the Soviet Union. The first resistant desi cultivar, F 8, was released 50 years ago in India (Luthra *et al.*, 1941). This line was a selection from an introduction of material from France. In the Soviet Union, three cultivars—Skorospelka, Alpha, and

Table V

Inventory of Inheritance of Resistance to *Ascochyta* Blight (*Ascochyta rabiei*) in Chickpea

Nature of inheritance	Genotype	References
Single dominant gene	F 8, F 10	Hafiz and Ashraf (1953)
Single dominant gene	I-13	Vir *et al.* (1975)
Single dominant gene	Code No. 72-92	Eser (1976)
Single dominant gene	ILC 72, ILC 183, ILC 200, ICC 4935	Singh and Reddy (1983)
Single recessive gene	ILC 191	
Single dominant gene	ILC 200, ILC 201	Acikgoz (1983)
Single recessive gene	72012, ILC 195, NEC 138-1	
Single dominant gene	EC 26446, PG 82-1 P 919, P 1252-1, NEC 2451	Tewari and Pandey (1986)
Single recessive gene	BRG 8	
Single dominant gene	ILC 72, ILC 202, ILC 2956, ILC 3279	Singh and Reddy (1989)
Single dominant gene	ILC 182, ILC 191, ILC 482	Halila *et al.* (1989)
Single recessive gene	ILC 195	

Mogucii—were developed, following a complex hybridization technique, and were released in 1946 (Guscin, 1946). Later, a hybridization technique was adopted in India and Pakistan and several cultivars released. Table III lists resistant cultivars.

Until 1984, no *Ascochyta* blight-resistant cultivar was released in the Mediterranean region where *Ascochyta* blight is the most serious disease. Cultivars released in India, Pakistan, and the Soviet Union never became popular with farmers, with the exception of C 235. One of the main reasons for their unpopularity was that they yielded less than susceptible cultivars during the disease-free years, and disease-free years are more frequent than blight years. Two other factors contributed to the lack of sustained breeding work: resistant cultivars soon became susceptible due to the occurrence of new races of *A. rabiei*, and a reliable and simple screening technique, which could be adopted by breeders to evaluate large segregating populations, was lacking.

The ICRISAT–ICARDA Kabuli Chickpea Project was established in 1978 at ICARDA, Syria. The project helped to develop an easy, reliable screening technique (Singh *et al.*, 1981), which was further refined by Reddy *et al.* (1984). Using this screening technique, more than 15,000 germplasm accessions maintained at ICARDA and ICRISAT were evaluated and a large number of resistant lines were identified. Many of the original accessions were mixtures of resistant and susceptible plants; these were purified and assigned new accession numbers. Resistant lines were evaluated for yield potential on the ICARDA farm, and high-yielding lines were provided to national programs.

Hybridization work also was initiated in 1978 to combine high yield with resistance to *Ascochyta* blight. Using off-season advancement facilities, more than 900 *Ascochyta* blight-resistant and high-yielding lines were bred between 1981 and 1989 and freely shared with national programs. Eleven countries released 26 cultivars from these materials between 1984 and 1989. This rapid progress was possible in the 12-year period because *Ascochyta* blight-resistant segregating populations were grown on an 8-ha plot each year during the main season, and three generations (F_1, F_3, F_6/F_7) were advanced in the off season on a 4-ha plot each year. The bulk–pedigree method to breed *Ascochyta* blight-resistant chickpeas at ICARDA is shown in Fig. 3. In addition to resistance to *Ascochyta* blight, this method is designed to breed photoperiod-insensitive chickpeas with resistance to other stresses.

ICARDA research had a catalytic effect on national programs. Now *Ascochyta* blight resistance breeding work has been launched in the Mediterranean region, India, Pakistan, and the United States. Many cou

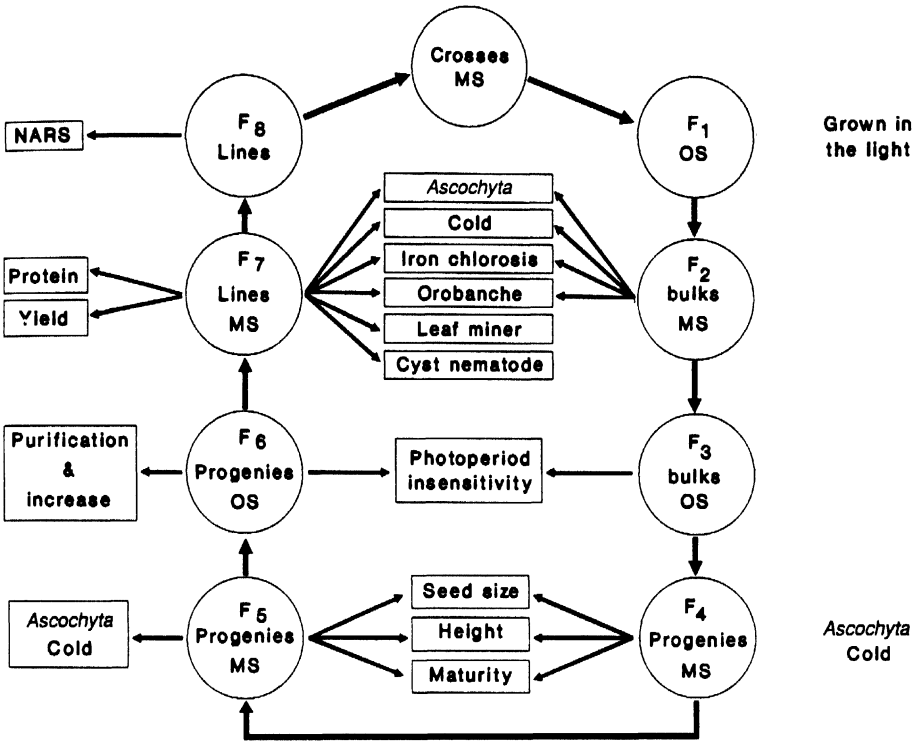

FIG. 3. Bulk–pedigree method for breeding chickpeas resistant to *Ascochyta* blight and other stresses. MS, Main season; OS, off season.

have developed resistant lines and may soon release them for commercial cultivation. Table III lists the release of resistant cultivars.

Several new problems emerged after the initial success in breeding for *Ascochyta* blight-resistant chickpeas. Most of the previously released cultivars have succumbed to new races; the life span of resistant cultivars has been short. No cultivar has been developed with resistance to all known races. Some strategies for development of durable blight-resistant cultivars are discussed here.

1. *Pyramiding multiple gene resistance.* Eight lines (ILC 72, ILC 201, ILC 202, ILC 2506, ILC 2956, ILC 3279, ILC 3856, and ILC 5928), are resistant to 4–5 races out of 6 races prevalent in Syria and Lebanon (Singh and Reddy, 1990). Since none of the lines was resistant to all 6 races, an attempt is being made to combine genes that will confer resistance against all 6 races in one line. A similar effort is being made at Ludhiana, India (G. Singh, personal communication). Lines with resistance to all existing races in a given country or region would be very useful in a breeding program.

2. *Polygenic resistance.* Although most published work on genetics of resistance to *Ascochyta* blight in chickpea suggests monogenic resistance, there are indications in at least some parents that the inheritance of resistance is governed by polygenes. If this is true, then breeding for partial resistance should be considered.

3. *Release of more than one cultivar in each country.* Release of several cultivars, possibly with known reactions to different races, will be useful; if resistance breaks down in one cultivar, others are then available to farmers. Morocco is an example. ILC 195 and ILC 482 were released in 1987; ILC 482 became susceptible in 1989 and was withdrawn; and now Moroccan farmers are cultivating ILC 195.

4. *Withdrawal of susceptible cultivars from cultivation.* Once resistant cultivars are released, even though their resistance may be weak, farmers should be advised to stop cultivating susceptible cultivars. This will reduce the build-up and spread of the disease. Earlier, when a susceptible check was included in all ICARDA yield trials grown in Jindiress (Syria) and Terbol (Lebanon), *Ascochyta* blight infected the susceptible checks and spread to other lines almost every year. After this practice was stopped in 1986, the disease has been seen only once in 4 years.

5. *Mapping of races.* There is a pressing need to map the existing races in the world. This will assist breeders to develop resistant cultivars suited to different regions.

No single strategy in breeding for *Ascochyta* blight-resistant cultivars may succeed, so a combination of different strategies should be employed. Genes conferring resistance to blight in wild *Cicer* species should be transferred to cultivated species. Likewise, mutation techniques could be used to develop higher levels of resistance. Breeders and pathologists working on blight resistance should meet periodically to discuss strategies to control *Ascochyta* blight disease.

2. Botrytis *Gray Mold* (Botrytis cinerea *Pers. ex Fr.*)

Botrytis gray mold is the second most important foliar disease after *Ascochyta* blight. The disease occurs on a regular basis, but damage is greatest in years of extensive winter rains and high humidity. The extent of losses due to this disease has not been precisely estimated. In Nepal, visual estimations during the 1987–1988 season indicated about 40% loss (Reddy *et al.*, 1988). Only limited research has been conducted on this disease whose importance has been recognized only recently. The disease is visible in the field from flowering stage onwards.

The disease affects all aboveground parts, producing brown necrotic lesions on leaves, stems, flowers, and pods. Seeds are also infected and, under certain conditions, the crop killed. Sporulation of the fungus can be seen on affected parts in the morning hours when dew is present. Many times, without any apparent symptoms on leaves and stems, the disease can cause flowers to drop, resulting in poor pod set. This type of damage usually goes unnoticed. Plants produce few pods at the upper nodes late in the season when conditions become unfavorable for the disease. It results in extended duration of the crop. Close planting, excessive vegetative growth, early sowings, and irrigation favor disease development of *Botrytis* gray mold.

Limited screening of germplasm and breeding material in "hot-spot" locations in India and Nepal has failed to identify high levels of resistance. There are a few lines, such as ICC 1069, ICC 6250, ICC 7574, and ICC 10302, which show field tolerance under moderate levels of disease (Rathi *et al.*, 1984; Sahu and Sah, 1988). Chickpeas are more susceptible in the flowering stage than in the vegetative stage. A few reports indicate variation in the pathogen *B. cinerea* (Singh and Bhan, 1986). Laboratory inoculation techniques and rating scales need to be standardized.

Inheritance of *Botrytis* gray mold resistance was studied in the resistant line ICC 1069. When ICC 1069 was crossed with BGM 413 and BG 256, monogenic dominance conferred resistance, but when ICC 1069 was crossed with BGM 419 and BGM 408, a ratio of 13 susceptible : 3 resistant was obtained indicating the presence of epistatic interactions. Thus, major gene resistance was found for *Botrytis* gray mold disease in chickpea (Rewal and Grewal, 1989). *Botrytis* gray mold is a highly devastating disease in certain years and regions, and a breeding program has been recently initiated jointly by ICRISAT and Pant University of Agriculture and Technology in India. No resistant cultivars have been released so far.

3. Alternaria *Blight* [Alternaria alternata (*Fr.*) *Kiessler*]

Alternaria blight, though not very widespread, occurs in Bangladesh, India, and Nepal. It has been reported to be serious in parts of northeast India in certain seasons and usually occurs along with *Botrytis* gray mold and *Stemphylium* blight as the conditions favoring these diseases are similar. Necrotic lesions are produced on all aboveground parts. In severe cases, the disease causes defoliation. A few lines, tolerant under field conditions, have been reported, but their resistance to artificial inoculation is yet to be confirmed.

4. Stemphylium *Blight* [Stemphylium sarciniforme (*Cav.*) *Wilts*]

Stemphylium blight has been reported in Bangladesh, India, Iran, and Syria and is particularly serious in parts of Bangladesh. Kabuli-type genotypes with a compact and erect canopy appear to suffer less from the disease than desi, spreading types. The disease is also favored by high humidity within the crop canopy.

5. *Rust* [Uromyces ciceris-arietini (*Grogn.*) *Jaj & Beyer*]

Rust, a very widespread disease occurring in almost all chickpea-growing countries, is not considered to be important, as it occurs late in the season when the crop is maturing and does not cause significant losses. However, in some years it causes yield loss in Ethiopia and Mexico. It produces brown or black powdery pustules on leaves and stems. Cool, humid weather favors its development.

C. Viral Diseases

Though as many as 16 viruses are known to infect chickpea, only stunt caused by bean (pea) leaf roll virus is economically important at present. This virus belongs to the luteo virus group. It is prevalent in most of the chickpea-growing countries. Stunting, browning (desi types), or yellowing (kabuli types), and thickening of the foliage and phloem browning are the characteristic symptoms of the disease. It has a wide host range and is transmitted by aphids such as *Aphis craccivora* Koch. and *Acyrthosiphon pisum* (Harris). The first symptoms of the disease in the field are noticed a month after sowing; plants affected early may wilt before maturity. Diseased plants produce few pods. The virus is not seed borne. Diseased plants are usually scattered in the field.

Extensive screening of germplasm and breeding materials at Hissar, northern India, a hot-spot location for the disease, revealed quite a few lines, such as ICC 403, ICC 591, ICC 685, ICC 2385, ICC 2546, ICC 3718, ICC 4949, ICC 6433, ICC 10466, ICC 10596, and ICC 11155, to have field resistance. Desi types are comparatively less susceptible than kabuli types. Early sowing and wide spacing were found to increase disease incidence at Hissar. Most of the cultivars bred in northern India, such as L 550, G 130, and JG 62, are tolerant to the disease. The high natural incidence of the disease in these areas might have inadvertently aided

selection of resistant plants. Breeding efforts to develop virus-resistant, high-yielding lines are under way at ICRISAT Center, India.

D. Nematode Diseases

Though more than 50 nematodes are known to infect chickpea, only a few are economically important. Work on nematode diseases has been very limited; more research is needed to obtain a clear picture of nematode problems. Root-knot, cyst, and lesion nematodes are relatively more important than the others.

1. Root-Knot Nematodes

Meloidogyne incognita (Kofoid and White) Chitw., *M. javanica* (Treub) Chitw., and to a lesser extent, *M. arenaria* (Neal) Chitw., are of importance in the Indian subcontinent, Egypt, and Malawi, along with *M. artiellia* Franklin in the Mediterranean area. Infected roots show characteristic galls whose size depends upon nematode species and plant cultivar. The first three of these species have wide host ranges, including wild plant species. These species prefer hot weather and can cause serious problems in regions where summers are long and winters are short and mild, such as peninsular India. However, severe damage also occurs in north India and in the Terai region of Nepal where minimum temperatures fall below 15°C for many days during the winter crop season.

Meloidogyne artiellia can infect chickpea even at soil temperatures below 15°C (Di Vito and Greco, 1988). Galls caused by this nematode are small, or may be absent with the only visible symptoms on infected roots being egg masses. These can be seen by early April on roots of winter chickpea. *Meloidogyne artiellia* survives during dry seasons as anhydrobiotic second-stage juveniles. Its host range is confined to cereals, legumes, and crucifers.

Spring chickpea is more susceptible to *M. artiellia* than winter chickpea, the tolerance limits being 0.016 and 0.014 eggs/cm^3 of soil, respectively. Complete crop failure occurs in fields infected with more than 1 egg/cm^3 of soil (Di Vito and Greco, 1988). Although *M. artiellia* is widespread in the Mediterranean area, severe damage to chickpea has been reported only from Italy, Spain, and especially Syria.

2. *Cyst Nematodes*

The chickpea cyst nematode, *Heterodera ciceri* Vovlas, Greco, and Di Vito, has been reported from northern Syria and is the only cyst nematode that causes severe damage to chickpea. It develops when the soil temperature rises above 10°C. Cysts are evident from late April onwards and can persist in soil for several years (Greco *et al.*, 1988). Infected roots show small necrotic spots from which females later emerge.

Heterodera ciceri causes damage whenever its population density exceeds 1 egg/g^{1} of soil (Greco *et al.*, 1988); complete crop failure occurs where there are over 64 eggs/g^{1} of soil. Its host range is, however, rather narrow compared to root-knot nematodes. Other good hosts are lentil, pea, and grass pea.

3. *Root-Lesion Nematodes*

Among root-lesion nematodes, *Pratylenchus thornei* Sher and Allen is distributed worldwide and damages chickpea in Syria and India.

Other *Pratylenchus* spp. (*P. zeae, P. brachyurus*) are also common on legumes and may infect chickpea as well. They cause cavities within the cortical parenchyma. Infected roots show many necrotic segments. Even though *P. thornei* seems to develop better from late winter to early spring, lesion nematodes are adapted to a large range of environmental conditions and have wide host ranges. Damage caused by *P. thornei* is less impressive than that caused by the previous two species, but the tolerance limit of chickpea to this species has not been determined in the field.

4. *Sources of Resistance to Nematodes*

At ICARDA, Syria, 8,200 chickpea accessions have been evaluated up to April 1990, but no source of resistance was found (Di Vito *et al.*, 1988; K. B. Singh, M. Di Vito, N. Greco, and M. C. Saxena, unpublished). However, when 137 accessions of eight wild *Cicer* species were evaluated, 21 accessions of *C. bijugum* K. H. Rech. were found resistant to cyst nematode (Singh *et al.*, 1989a). In recent screening, five accessions of *C. pinnatifidum* Jaub & Sp. and one accession of *C. reticulatum* Ladiz. were found to be resistant. Efforts are under way to transfer the gene for resistance to the nematode from *C. reticulatum* to a high-yielding line of *C. arietinum*.

Strain No. 501 has been identified as resistant to root-knot nematode (Mani and Sethi, 1984). Several mutants resistant to root-knot nematode have been developed (Bhatnagar *et al.*, 1988). Despite identification of resistance sources, planned hybridization work to transfer the gene for resistance to high-yielding lines has yet to be undertaken.

E. Breeding for Resistance to Other Diseases

Diseases other than *Ascochyta* blight and *Fusarium* wilt are only of localized importance, and include *Botrytis* gray mold, rust, stunt, *Phytophthora* root rot (*Phytophthora megasperma* f.sp. *medicaginis*), and two nematodes (root-knot and cyst). Some progress has been made toward development of cultivars resistant to *Botrytis* gray mold disease and to stunt (pea leaf roll virus) at ICRISAT, India (Nene and Reddy, 1987), *Phytophthora* root rot in Australia (Brinsmead, 1985), cyst nematode at ICARDA, Syria (Di Vito *et al.*, 1988; Singh, *et al.*, 1989a), and root-knot nematode at ICRISAT, India (Sharma and Mathur, 1985).

V. BREEDING FOR MULTIPLE DISEASE RESISTANCE

Disease-resistant cultivars of chickpea have never been grown widely, mainly because they lack resistance to all the important diseases of a country or region. Singh *et al.* (1991) have strongly advocated the breeding of cultivars with resistance to all the important diseases of a country, and have also suggested that attempts should be made to breed cultivars with multiple stress resistance. For north Africa, cultivars with resistance to *Ascochyta* blight and *Fusarium* wilt are required. If cultivars are resistant to only one disease they will not be grown extensively. In west Asia, *Ascochyta* blight and cold-tolerant cultivars are required for winter sowing of chickpea, where the crop is traditionally grown in spring. In south India, cultivars with resistance to pod borer (*Helicoverpa* spp.) and *Fusarium* wilt are required.

Since the mid 1980s, attempts have been made to breed cultivars with multiple disease resistance. It is hoped that in the 1990s cultivars with multiple stress resistance will be bred and released. Singh *et al.* (1991) have listed important diseases and insect pests in different regions. This list needs to be expanded to include other stresses; the multiple stress-

resistant accessions of wild *Cicer* species, described in the succeeding section, will be useful in breeding.

VI. ANNUAL WILD *CICER* SPECIES AS A POTENTIAL SOURCE OF GENES FOR RESISTANCE

Forty-three *Cicer* species, including 9 annual and 34 perennial types, have been reported (van der Maesen, 1987). Since maintenance of perennial species is difficult, scientists other than germplasm botanists are mainly interested in annual species. ICARDA holds 233 accessions of 8 wild *Cicer* species and ICRISAT maintains 97 lines of both annual and perennial wild species. When accessions maintained at ICARDA were evaluated for resistance to *Ascochyta* blight, *Fusarium wilt,* and cyst nematode, higher levels of resistance than any available in cultivated species were found for the two diseases, as well as the only known source of resistance to cyst nematode (Table VI). Wild *Cicer* species have been investigated for resistance to diseases. *Cicer judaicum* was found resistant to *Ascochyta* blight, *Fusarium* wilt, and *Botrytis* gray mold (van der Maesen and Pundir, 1984). Nene and Haware (1980) also found *C. judai-*

Table VI

Evaluation of *Cicer* spp. for Resistance to *Ascochyta* Blight, *Fusarium* Wilt, and Cyst Nematode, at Tel Hadya, Syria, 1987–1989[a]

Cicer species	*Ascochyta* blight		*Fusarium* wilt[b]	Cyst nematode
	Mixture of race 1–4	Mixture of race 1–6		
C. bijugum K.H. Rech.	R	R	F	R
C. chorassanicum (Bge) M. Pop.	S	S	NT	S
C. cuneatum Hochst. ese Rich	S	S	NT	S
C. echinospermum P.H. Davis	S	S	F	S
C. judaicum Boiss.	R	R	F	S
C. pinnatifidum Jaub. & Sp.	R	S	F	R
C. reticulatum Ladiz.	S	S	F	R
C. yamashitae Kitamura	S	S	NT	S

[a] F, Free from damage; R, resistant; S, susceptible; NT, not tested.

[b] Evaluation was done at Istituto Sperimentale per la Patologia Vegetale, Rome, Italy.

cum resistant to *Fusarium* wilt. Singh *et al.* (1982) have reported *C. pinnatifidum* to be resistant to *Botrytis* gray mold.

Accessions of wild species have been evaluated for reaction to six different biotic and physical stresses, and resistance sources have been identified for all stresses including seed beetle (*Callosobruchus chinensis L.*) and cyst nematode, for which no sources of resistance were found in the collection of cultivated species (Singh *et al.*, 1989b). The most important achievement in evaluation of wild species was identification of genotypes having genes for resistance to four or five stresses. For example: accession No. ILWC 7-1 of *C. bijugum* is resistant to *Ascochyta* blight, *Fusarium* wilt, leaf miner, cyst nematode, and cold; accession No. ILWC 33/S-4 of *C. pinnatifidum* is resistant to *Ascochyta* blight, *Fusarium* wilt, seed beetle, and cyst nematode. No accession of the cultivated species has been found to have genes for resistance to more than one stress. Wild species are therefore potentially most important for disease and other stress-resistance breeding. Hence, it is strongly advocated that (1) wild species should be evaluated for other diseases, (2) embryo and ovule rescue techniques should be employed to transfer genes for resistance from noncrossable wild species to cultivated species, and (3) in view of the usefulness of wild species, more collections should be made.

VII. RESISTANT CULTIVARS IN DISEASE MANAGEMENT

Chickpea is grown primarily by resource-poor farmers on residual moisture with little if any inputs. The short growing season at lower latitudes (0–20°) also limits yields. The fast-rising temperatures at the reproductive phase force the crop into premature drying. Thus, yields are low (less than one t/ha). At present productivity levels, use of disease-resistant cultivars appears to be the best alternative for management of chickpea diseases.

Singh (1987) listed the disease-resistant cultivars developed in different countries; an updated list is presented in Table III. Several cultivars are resistant to soil-borne diseases (mostly resistant to *Fusarium* wilt) and *Ascochyta* blight. Though cultivars bred for resistance to soil-borne diseases have maintained their resistance, *Ascochyta* blight-resistant cultivars have shown frequent resistance breakdown due to appearance of new races.

As there are no cultivars with high levels of *Ascochyta* blight resistance, especially when the disease develops in epiphytotic form, tolerant cultivars should be used in combination with other management practices. Seed free of the *Ascochyta* blight pathogen should be produced under arid

conditions (Kaiser, 1984) or alternatively, seeds should be dressed with fungicide before sowing (Reddy, 1980). If seed yields of over 2 t/ha are expected, as is generally the case with winter-sown chickpea in the Mediterranean region, then a combination of a tolerant cultivar like ILC 482 and one foliage spray with chlorothalonil (tetrachloroisophthalonitrile) can be beneficial (Reddy and Singh, 1990).

VIII. CONCLUSIONS AND FUTURE NEEDS

The disease problems of chickpea are well identified and their distribution and importance are known. Though considerable progress has been made in managing the diseases, more work remains. Among soil-borne diseases there has been encouraging progress on *Fusarium* wilt in standardization of inoculum techniques, identification of resistance sources, and understanding the genetics of resistance, variability in the pathogen, and breeding for resistant cultivars. The mechanisms of resistance, however, need to be investigated further. Progress with other soil-borne diseases has been very limited. Standardization of inoculation techniques, identification of resistance sources, and breeding for resistant cultivars all require much more research. As wilt and root rots usually occur together, there is a need to breed cultivars having multiple disease resistance to soil-borne diseases.

Progress on the management of foliar diseases during the past 10–15 years, especially through the use of resistant cultivars, has been remarkable. In the case of *Ascochyta* blight, effective inoculation techniques and rating scales have been standardized. Some information on the genetics of resistance and on variability in the pathogen have been obtained. Steady progress has been made in identifying resistance sources and in breeding resistant cultivars for countries around the Mediterranean basin. However, progress on identification of resistance sources and breeding resistant cultivars in India and Pakistan has been limited. In these two countries, high, stable resistance levels need to be identified. Though the existing germplasm collection does not appear to have high levels of resistance, there is a significant amount of variability in susceptibility to the disease. A germplasm enhancement program to accumulate available genes for resistance may prove useful. Further studies on genetics of resistance, mechanisms of resistance, and the relationship between plant height, maturity, and resistance will be useful for better exploitation of resistance sources.

Research on the other foliar diseases has been very limited. Losses

caused by these diseases, though yet to be estimated, may be substantial. Work on standardization of inoculation techniques, development of rating scales, and identification of resistance sources to *Botrytis* gray mold, *Alternaria* blight, and *Stemphylium* blight needs to be undertaken.

Though field-tolerance sources and cultivars are available for stunt disease (pea leaf roll virus), there is a need to develop cultivars with combined resistance to stunt and major soil-borne and foliar diseases. Nematodes can cause substantial damage to chickpeas; efforts should be made to locate sources of resistance to root-knot, cyst, and lesion nematodes and to incorporate them in disease-resistant backgrounds.

Unlike cereals or major grain legume crops such as soybean, disease-resistant cultivars of chickpea have never been grown widely. The main reason for this is that the yield potential of the resistant cultivars is lower than susceptible cultivars. Second, most released cultivars possess resistance to only one disease, whereas under most situations the chickpea plant is attacked by two or more diseases. Therefore, future breeding programs should attempt to upgrade the yield potential of resistant cultivars to, or above, that of susceptible cultivars, and to combine genes for resistance to the most important diseases prevalent in the region.

Wild *Cicer* species are known to possess genes for resistance to several diseases, but they have never been transferred to cultivated species. Genes for resistance from two species, *C. echinospermum* and *C. reticulatum,* could easily be transferred to cultivated species by normal hybridization techniques. Furthermore, through the use of embryo and ovule rescue techniques, efforts should be made to transfer genes for resistance from the currently noncrossable *Cicer* species to the cultigens.

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