Breeding for disease resistance in pulse crops

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Abstract

Resistance or tolerance has been identified for 13 major diseases of pea and at least 5 major diseases in each of chickpea, faba bean and lentil. Most of the resistances are oligogenic; some have proven to be stable but many have not. Approaches to improve the stability and durability of resistance are discussed.

Introduction

Breeding resistant cultivars is the most widely used and cost-efficient method of controlling diseases in annual crops of marginal profitability. This paper reviews the status and mechanisms of resistance to major diseases of the four legume crops of topical concern. It also summarizes the concepts and terminology involved in using resistance, presents methods of testing for complete and partial resistance, and discusses approaches and techniques to improve the effectiveness, stability and duration of resistance.

The status of resistance

Disease resistance has ensured the successful production of garden and field peas, particularly in North America, Europe and Australia. Resistance, semi-resistance or tolerance has been reported for 19 of the 32 diseases of pea (Cousin, 1978; Hagedorn, 1984). Resistance or tolerance to major bacterial and fungal diseases has now been incorporated into many pea cultivars (cvs) and breeding lines (Table I). However, effective resistance to Ascochyta blight caused by *Mycosphaerella pinodes* (Berk. and Blox.), and

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Table 1. Inventory of genetic resistance to major bacterial and fungal pea diseases.

Disease	Pathogen	Resistance available as	Nature of inheritance	Pathotypes	References
Bacterial blight	*Pseudomonas syringae* pv. *pisi* (Sackett)	many cvs res. to r1, 2, 1 & 2, 3[a]	r1 = monogenic dominant + modifying genes	3 races	Hagedorn (1984) Taylor (1972)
Seedling rot	*Rhizoctonia solani* Kuehn	semi-resistant gp & bl	—	—	McCoy and Kraft (1984) Shehata *et al.* (1981)
Leaf and pod spot	*Ascochyta pisi* Lib.	4 res. cvs	3 dominant genes; duplicate dominant genes	5 races	Ali *et al.* (1978) Cousin (1974; 1985) Lyall and Wallen (1958)
Downy mildew	*Peronospora viciae* (Berk.) de Bary	many res. cvs	monogenic dominant; digenic recessive; monogenic recessive; polygenic	6 races	Cousin (1974); Dixon (1978) Hubbeling (1975) Mathews (1978)
Powdery mildew	*Erysiphe pisi* Syd.	genes er_1, er_2, in many evs	monogenic recessive	yes	Dixon (1978) Heringa *et al.* (1969)
Fusarium wilt	*Fusarium oxysporum* f. sp. *pisi* (van Hall) Smyd. and Hans.	most cvs res. to r1; some to r2; a few to r1, 5, 6.	monogenic dominant to each of 5 r	5 races	Hagedorn (1984) Kraft and Haglund (1978)
Pythium root rot	*Pythium* spp.	res. in gp & bl	—	—	Muehlbauer and Kraft (1973)
Fusarium root rot	*Fusarium solani* f. sp. *pisi* (Jones) Smyd. and Hans.	tol. in gp & bl	—	—	Kraft (1984) Kraft *et al.* (1981)

[a] cvs = cultivars; res. = resistant; tol. = tolerance; gp = germplasm; bl = breeding line; r = race.
Dashes denote that data are not available.

to root rot caused by *Aphanomyces euteiches* Drechs., remains elusive. Aphanomyces root rot is very destructive in many pea growing regions, and although tolerance has been identified, it is linked to undesirable horticultural traits (Meiners, 1981).

Hagedorn (1984) summarized the then current information on virus diseases of pea. Resistance or tolerance has been found for 8 of 11 virus diseases. The status of resistance in pea cvs to five major virus diseases is as follows: many cvs are tolerant to pea enation mosaic and bean leaf roll, which are each governed by a single dominant gene; cvs are also immune and resistant (monogenic recessive) to pea seed-borne mosaic and bean yellow mosaic virus (BYMV), respectively. In addition, tolerance to pea streak mosaic (multigenic) is now present in breeding lines.

In contrast, the pathology of each of chickpea, faba bean and lentil was largely neglected until the establishment of ICRISAT and ICARDA in 1972 and 1976, respectively (see pp. 39 and 25, this Volume). Since then, large germplasm collections of both chickpea and faba bean have been screened for resistance to several pathogens (Erskine, 1984; Hanounik and Maliha, 1985; Hawtin, 1984; Singh and Malhotra, 1984). As shown in Tables 2 and 3, resistance has been identified against many major diseases of each crop, and is being transferred to breeding lines and cultivars.

Resistance breeding has been given less priority in lentil improvement programmes since diseases have been less damaging on a global scale than in other food legumes. Nevertheless, resistance to the following more important diseases has been identified: Rust caused by *Uromyces viciae fabae* (Khare, 1981; Bejiga, 1984; Erskine, 1985); Fusarium wilt caused by *Fusarium oxysporum* f. sp. *lentis* (Khare, 1981; Bejiga, 1984; Erskine, 1985); Root rot caused by *Fusarium* sp. (Kannaiyan and Nene, 1976; Lin and Cook, 1977); Ascochyta blight caused by *Ascochyta lentis* (Khare, 1981; Gurdip Singh *et al.*, 1982; Bejiga, 1984; Erskine, 1985; Muehlbauer and Slinkard, 1985); Downy mildew caused by *Peronospora lentis* (Khare, 1981; Erskine, 1985); and Pea seed-borne mosaic virus (Haddad *et al.*, 1978). In the case of Ascochyta blight and rust, resistance was found in existing cvs with good agronomic traits (Muehlbauer and Slinkard, 1985). These are useful as interim cvs as well as resistant parents. The genetics of the resistances in lentil have not been elucidated except for Pea seed-borne mosaic virus where immunity is controlled by a single recessive gene (Haddad *et al.*, 1978). However, since most of the lentil pathogens are similar to those of the other food legumes, the resistances may also be similar and monogenic.

Stability and durability of resistance

The usefulness of cvs with oligogenic resistance has often been ephemeral, giving rise to serious disease epidemics until replaced by other resistant cultivars. This is particularly true of biotrophic, highly mobile pathogens such as rusts, downy mildews and powdery mildews which have a great capacity to

Table 2. Inventory of genetic resistance to major chickpea diseses.

Disease	Pathogen	Nature of inheritance	Pathotypes	References
Fusarium wilt[a]	*F. oxysporum* f.sp. *ciceri* (Padwick)	1. Monogenic recessive. 2. Digenic recessive complementation. 3. Multiple alleles.	yes	Ananda Rao *et al.* (1986) pers. comm. Ayyar and Iyer (1936) Sindhu *et al.* (1983) Smithson *et al.* (1983) Upadhyaya *et al.* (1983)
Dry root rot[a]	*Rhizoctonia bataticola* (Taub) Butler	Monogenic dominant.	no	Ananda Rao *et al.* (1986) pers. comm.
Ascochyta blight[ab]	*Ascochyta rabiei* (Pass.) Labr.	1. Monogenic dominant or single recessive gene. 2. Two dominant linked genes. 3. Two dominant loci and an independent recessive gene.	yes	Ahmad *et al.* (1952) Eser (1976) Hafiz and Ashraf (1953) Singh and Reddy (1983) Tewari and Pandey (1985) Vir *et al.* (1975)
Botrytis grey mould[a]	*Botrytis cinerea* Pers. Ex. fr.	Monogenic dominant	—	Tweari and Pandey (1985)
Chickpea stunt[a]	Pea leaf roll virus	Monogenic dominant?	—	Kumar *et al.* (1983)

[a] Resistance available as germplasm or breeding lines.
[b] In cvs.
Dashes denote that data are not available.

Table 3. Inventory of genetic resistance to major faba bean diseases.

Disease	Pathogen	Resistance		Pathotypes	References
		Type	Nature of inheritance		
Rust[a]	*Uromyces viciae-fabae* (Pers.) Schroet	SR rr	Monogenic dominant —	yes	Conner and Bernier (1982a) Conner and Bernier (1982b) Rashid and Bernier (1984) Khalil *et al.* (1985)
Ascochyta spot[a] (blight)	*Ascochyta fabae* Speg.	SR rr	Monogenic dominant —	yes	Kharbanda and Bernier (1980) Rashid and Bernier, unpublished Hanounik and Maliha (1984)
Chocolate spot[a]	*Botrytis fabae* Sard.	SR	Monogenic?	yes?	Hanounik (1983); Hanounik and Maliha (1985); Elliot and Whittington (1979); Khalil *et al.* (1984)
Root rot[a]	*Aphanomyces euteiches*	—	—	yes	Lamari *et al.* (1984) Lamari and Bernier (1985)
Bean yellow mosaic[a]	BYMV	SR tol.	Digenic recessive	yes	Gadh (1983); Gadh and Bernier (1984) Schmidt *et al.* (1985)
Broomrape[a]	*Orobanche crenata* Forsk.	—	—	—	Nassib *et al.* (1982)

SR = strong resistance; rr = rate reducing resistance; tol. = tolerance.
[a] Resistance available in germplasm and breeding lines.
Dashes denote that data are not available.

generate more virulent pathotypes. Most of the sources of resistance or tolerance identified in the four food legumes reviewed here are oligogenic. The only exceptions are the multigenic tolerances to streak mosaic and downy mildew in pea.

Many pathogens of the food legumes have evolved more virulent pathotypes locally, or cvs resistant in one location have been found to be susceptible elsewhere (Tables 1 to 3). In pea for example, new pathotypes or races have been found for bacterial blight (3 races); *Ascochyta pisi* (resistant Canadian and Bristish cvs are susceptible in Australia); Downy mildew (6 races); Powdery mildew (2 races); and Fusarium wilt (7 races). Fortunately, new races do not always become widely distributed and resistance to race 1 of Fusarium wilt has remained stable for more than 40 years in some regions.

Not all single gene resistances have been short-lived. Resistance to BYMV and bean leaf roll in pea, for example, has lasted for many years. Why monogenic resistance is stable in some host-pathogen systems and not in others remains unknown.

The breakdown or transient nature of monogenic resistance in many host-pathogen systems has focused greater attention on partial or field resistance that operates by reducing the rate of pathogen development, and on the management of monogenic resistance to provide greater stability against changes in the population of the pathogen (Allen, 1983; Buddenhagen, 1983; Johnson, 1984; Parlevleit and Zadoks, 1977). A clear understanding of the concepts of resistance, and of the terminology used, will ensure effective selection for resistance.

Concepts of resistance and terminology

The meaning of and the terms used to describe *resistance* have not been consistent; it has been defined and named by many authors working on different host-pathogen systems either in terms of genetics, epidemiology, differential interaction between host and pathogen, or on magnitude of effect (Parlevleit and Zadoks, 1977; Nelson, 1973; Parlevleit, 1979).

Host resistance is defined as "the ability of the host to hinder the growth and/or development of the pathogen" Parlevleit (1979). the term "complete resistance" (CR) is used when the multiplication of the pathogen has been completely prevented, i.e. spore production (SP) is inhibited completely. "Incomplete resistance" (ICR) refers to all resistances that allow some SP. "Partial resistance" (PR) is used when the SP is reduced even though the host plants are susceptible to infection (susceptible infection type).

Horizontal resistance (HR) is used in the sense of race-nonspecific resistance, characterized by the absence of genetic interactions between host and pathogen genotypes. Vertical resistance (VR) then is characterized by the presence of genetic interactions between host and pathogen genotypes.

The cultivar-isolate test suggested by Van der Plank (1963) to distinguish between VR and HR on the basis of differential interaction is laborious, and

many researchers have instead measured disease reduction as an indicator of HR. Terms such as PR (Parlevleit, 1979), rate-reducing resistance (Nelson, 1973), and slow-rusting (Conner and Bernier, 1982b) are now widely used. When resistance is complete, the cultivar-isolate test may not be feasible because of inadequate knowledge of races, in which case resistance may be referred to as "strong resistance".

According to the proposed terminology, CR, i.e. infection type (IT) of 0 or; in rusts, can be governed by a single gene, or by several "minor genes" with additive effects (Sharp and Volin, 1970). PR, i.e. IT of 2 or X in rusts, can also be due to a single gene, or to polygenes. Thus, selection on the basis of low IT to biotrophs does not by itself ensure a more complex and stable genetic resistance.

Methods of testing for resistance

Testing and selection methods must prove effective in the identification of CR as well as PR in germplasm, individual F_2 plants and in advanced breeding lines. To ensure success with foliage pathogens, the plant material must be adequately challenged with a single race or pathotype at a realistic inoculum dose to allow disease development but, at the same time, not obscure minor differences in host response. To illustrate, three cvs each having a single gene for resistance to a given race would be identified only when inoculated singly with each isolate but not if the isolates were used in a mixture. The ability to recognize PR in the presence of complete resistance in plants exposed to a mixture of races was recently discussed by Parlevleit (1983). He concluded that using a single race provides the best conditions for the selection of PR in the presence of CR, and that the race should have the broadest possible virulence spectrum to suppress the expression of as many CR genes as possible.

Exposure of plant material to the pathogen should be as uniform as possible so as to prevent any escape from infection. This is seldom achieved for foliage pathogens when material is evaluated in field plots under conditions of natural infection. Furthermore, in some years, disease may develop poorly or not at all. Adequate exposure can be achieved readily in the field if small plots are sprayed with inoculum and then covered with a polyethylene sheet to maintain leaf wetness overnight. This approach has been used successfully with several faba bean pathogens (Hanounik and Maliha, 1985; Rashid and Bernier, 1984). In this way, plants are evaluated for IT and/or size and frequency of lesions at the same growth stage, as they would be in the glasshouse. Later, plants with ICR can also be rated for disease severity on upper non-inoculated leaves, fruits and stems, as a measure of PR to autoinfection.

Field testing can be supplemented by glasshouse and growth room testing which is more rapid and allows the testing of several isolates sequentially on different sets of leaves (Nene *et al.*, 1981). Large collections of pathogen

isolates from a given region can be evaluated in this way to provide information on the pathogenic variability within the population and on the stability of the resistance. However, results of indoor tests must be shown to correlate reliably with field results.

The ultimate evaluation of lines or cvs judged to be resistant in a local programme is through international, multilocational trials, as conducted by ICRISAT and ICARDA, for example.

Partial resistance (slow-rusting) is best evaluated in adjacent or isolated field plots (Conner and Bernier, 1982b; Parlevleit, 1979; Rashid and Bernier, 1986). Inoculum is applied to spreader rows sown at right angles to the plots or at a point source in larger plots. Disease severity (proportion (%) leaf area infected) is assessed several times from the beginning to the end of the epidemic. The data are used to calculate the apparent infection rate or the area under the disease progress curve.

When several single plant selections were made amongst heterogeneous faba bean populations on the basis of reduced disease severity, the performance of the progenies was similar to that of the original selection (Rashid and Bernier, 1986). This suggests that individual F_2 plants could be effectively selected for PR. Lines and cvs shown to be slow-rusting in the field should be evaluated further in growth rooms to established which components of resistance are present in each host genotype (Parlevleit, 1979). Lines with the greatest number of components are likely to be more genetically complex and so more stable.

Screening for resistance to soil-borne diseases is best achieved by developing sick plots. This technique has been used successfully in chickpea (Nene *et al.*, 1981), pea (Hagedorn, 1980) and faba bean (Lamari *et al.*, 1984). Screening can also be conducted indoors to confirm resistance identified in the field (Kraft, 1980).

For large-scale inoculation of field trials with mechanically transmissible viruses, an artist air-brush is effective and rapid (Gadh and Bernier, 1984). For insect transmitted viruses, insects can be reared indoors on virus-infected plants in cages and then released onto spreader rows several times during the season.

Strategies for more stable and durable resistance

The task of developing cvs with stable resistance is most challenging for pathogens such as rusts, downy mildews and powdery mildews. The only recourse is to attempt to increase the complexity of the genetic resistance of the cvs through breeding. Partial resistance based on several components of resistance would seem most appropriate. Alternatives are the accumulation of several CR genes in a single cv. or the development of intraspecific within-field diversity by mixing host genotypes (Wolfe, 1985). Such diversity may be achieved by cultivar mixtures, multiline varieties or synthetic cvs (Bond, 1982) comprised of CR, PR or both. The use of CR genes as

described here would require the diligent monitoring of races and so the demanding inputs of time and labour may exceed the capabilities of most programmes. Recently, the enhancement of resistance by interactions of genes confering moderate resistance has been demonstrated for stripe rust (Sharp and Volin, 1970) and for leaf rust of wheat (Samborski and Dyck, 1982). This approach should be pursued with food legume host-pathogen systems, including soil-borne pathogens, in the development of improved breeding lines through recurrent selection.

Conclusions

Resistant cvs, breeding lines and germplasm have been developed for an impressive number of major diseases of food legumes. The prospects are excellent for further improvement in the stability and durability of resistance in future cvs through either vertical gene management, partial resistance or enhanced resistance by interaction of minor genes. Success, however, will require the close co-operation of plant breeders and pathologists, and adequate resources and funding.

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A complete bibliography of the sources of resistance cited in Tables 1, 2 and 3, and for lentil diseases and virus diseases of pea in the the text, is available on request to the senior author.

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