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Breeding for resistance to lentil *Ascochyta* blight

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Abstract

Ascochyta blight, caused by *Ascochyta lentis*, is one of the most globally important diseases of lentil. Breeding for host resistance has been suggested as an efficient means to control this disease. This paper summarizes existing studies of the characteristics and control of *Ascochyta* blight in lentil, genetics of resistance to *Ascochyta* blight and genetic variations among pathogen populations (isolates). Breeding methods for control of the disease are discussed. Six pathotypes of *A. lentis* have been reported. Many resistant cultivars/lines have been identified in both cultivated and wild lentil. Resistance to *Ascochyta* blight in lentil is mainly under the control of major genes, but minor genes also play a role. Current breeding programmes are based on crossing resistant and high-yielding cultivars and multilocation testing. Gene pyramiding, exploring slow blighting and partial resistance, and using genes present in wild relatives will be the methods used in the future. Identification of more sources of resistance genes, good characterization of the host-pathogen system, and identification of molecular markers tightly linked to resistance genes are suggested as the key areas for future study.

Keywords: *Ascochyta lentis* ; breeding methods ; disease resistance

Ascochyta blight, caused by *Ascochyta lentis*, is an important disease of lentil. It has been reported to be the major lentil disease in many lentil-producing countries, including Argentina, Australia, Canada, Ethiopia, India, New Zealand, Pakistan and The Russian Federation (Kaiser and Hannan 1986, Erskine et al. 1994). The disease has considerable effects on both seed quality and yield (Morrall and Sheppard 1981). Gossen and Morrall (1983) estimated that foliar infection has caused yield losses of up to 40%, but in Canada economic losses from infected seed may reach more than 70%. In some cases, seed infection is so severe that the lentils are unmarketable (Kaiser 1981, Gossen and Morrall 1983).

Despite extensive agronomic and chemical control studies, no efficient method has been found to control *Ascochyta* blight in lentil. Breeding for host resistance is the most effective, efficient and environmentally friendly method to control the disease (Erskine et al. 1994, Ye et al. 2000a).

This paper summarizes our understanding of *Ascochyta* blight in lentil, including the variations among pathogen populations (isolates) and the genetics of resistance incorporating recent work in this area at Lincoln University, Canterbury, New Zealand. Following this, methods for breeding for resistance and corresponding backup techniques are discussed.

The basic characteristics of *Ascochyta* blight in lentil

Bondartzeva-Monteverde and Vassilievsky (1940) first identified *A. lentis* as a pathogen of lentils in Russia in 1938. They made detailed investigations of disease symptoms. Mitidieri and De (1974) reported necrotic lesion development on leaflets, stems, pods and seeds of lentils associated with *A. lentis* infection.

Based on comparisons of cultural and morphological traits among isolates of *Ascochyta fabae* (the causal agent of *Ascochyta* blight of faba bean) and isolates of *A. lentis*, Gossen et al. (1986) suggested that *A. lentis* should be synonymized with *A. fabae*. They proposed two special forms: *A. fabae* f. sp. *fabae* for isolates from faba bean and *A. fabae* f. sp. *lentis* for isolates from lentil. This proposal has been widely accepted. However, based on the random amplified polymorphic DNA (RAPD) banding patterns, morphological characteristics and pathogenicity tests, Kaiser et al. (1997) suggested that the causal pathogen of *Ascochyta* blight in lentil is a species that is distinct from that of *A. fabae*.

Kaiser and Hellier (1993) found the sexual stage of *A. lentis* on lentil straw in the USA in 1992. They showed that the fungus is heterothallic with two mating types, and that it is probably a species of *Didymella*. Ahmed et al. (1996a) confirmed the presence of two mating types of *A. lentis* by controlled crossing in the laboratory. The roles of the two mating types in the field in promoting variability in pathogen population and in the disease cycle are not known.

Infection and disease development and spread are favoured by cool, wet weather (Nene et al. 1988). The highest infection frequency occurs with a wetness period of 24 or 48 h. The latent period is shortest at 20°C and longest at 10°C. Temperature has little effect on lesion size and number of pycnidia per lesion, but infection frequency is higher at 10°C and 15°C than at 25°C (Pedersen and Morrall 1994). Disease severity is tissue-age-related. Tissue below the top four or five nodes on the main stem and secondary branches are almost completely free of disease. This effect is most apparent at the podding stage (Pedersen and Morrall 1994).

Seed infection occurs up to 250 m from the primary inoculum source, but gradients generally level off within 50 m (Pedersen et al. 1993). The larger splash droplets following a ballistic trajectory are important in the short-range spread of the disease, and longer-range spread is the result of wind dispersal of detached leaflets and conidia in small air-borne droplets (Pedersen et al. 1994).

Genetic variations among isolates of *A. lentis*

A good understanding of the genetic variations of pathogenicity and virulence patterns in the pathogen is useful for designing a breeding programme and infection test method. For example, when there are many physiological races of the pathogen, race-specific resistance usually conferred by major genes can be easily overcome by the pathogen. Therefore, exploring partial resistance conditioned by polygenes and/or combining major resistance genes for different races is necessary to obtain durable resistance. However, there is only limited information on variation in *A. lentis*. Tests done by Kaiser et al. (1994), using 24 isolates from different countries, showed only small differences among isolates in their ability to attack two cultivars. Ahmed and Morrall (1996) investigated the reactions of lentil lines and cultivars (differentials) to seven isolates of *A. lentis* in the field using artificial inoculation, and percentage seed infection. Significant differences (based on initial disease severity) occurred among differentials in their reactions to the isolates. Based on the seed infection percentage and the area under the disease progress curve, the differentials and isolates were significantly different in both years. The isolate × differential

interaction was nonsignificant in both years for seed infection percentage, and significant in only one year for the area under the disease progress curve.

Ahmed et al. (1996b) investigated the virulence patterns of 84 isolates of *A. lentis* from western Canada, and isolates from 13 other countries on 10 lentil lines and cultivars (differentials). Western Canadian isolates collected in 1978 and 1985 were less virulent than 1992 collections. Cluster analysis grouped the isolates into weak, intermediate and high virulence forms. However, the virulence pattern did not relate to specific geographical locations.

Nasir and Bretag (1997a) examined the pathogenic variability of 39 isolates from Australia using 22 lentil cultivars. Based on the different reaction of six cultivars ('ILL 358', 'ILL 7537', 'ILL 7515', 'ILL 5588', 'ILL 5244' and 'Eston'), they identified six pathotypes. Most of the isolates were virulent to some of the cultivars. There were three isolates that infected all the cultivars, and five that were avirulent to all the cultivars. This study showed that resistance is most likely to be pathotype-specific.

Measuring the reaction to disease

The different genotypic reactions to *A. lentis* are the basis of breeding for resistance. To measure the reaction, a suitable infection test and disease rating scale need to be designed for the desired breeding objectives and understanding of variation in patterns of pathogenicity.

Infection test methods

Results from infection tests are the basic information on host resistance. Therefore, the reliability of the infection test used is key for a successful breeding programme.

Natural infection test

Testing in the field under natural disease epidemics is the cheapest method. With the knowledge of disease epidemics, 'hot-spots' can be selected for testing. Using this method all plants are exposed to environmental conditions that are similar to those that the resultant resistant cultivars will face. However, the expression of resistance may be masked by the interactions between different biotic and abiotic factors, and race-specific resistance cannot be identified. Moreover, resistance can only be tested in epidemic years and thus reduce breeding progress. Therefore, selection for resistance should not entirely rely on this method, although it may be the best one to test the resistance achievable in a breeding programme.

Artificial infection test in the field

To overcome some of the potential problems of the natural infection test, artificial epidemics can be generated in the field. This can be achieved by using with inoculum prepared in the laboratory, scattering diseased debris and interplanting susceptible genotypes (spreaders) to increase pathogen populations, and providing irrigation to increase relative humidity (Ahmed and Morrall 1996). This method is the preferred method in breeding programmes because of its simplicity and reliability. All resistant cultivars released and most of resistant germplasm were identified using this method. However, its effectiveness can be reduced by genotype \times environment interaction, physiological age of plant and organ specificity of resistance expression.

Artificial infection test in glasshouse

Glasshouse testing has several advantages over field testing. First, off-season testing can be done and testing can also be done at any development stage of the plants. Second, environmental conditions can be more easily adjusted to enhance disease development. Third, possible interference caused by other biological agents and environmental factors can be avoided. Fourth, the inoculum can be more evenly distributed and consequently reduce the risk of escapes. Glasshouse tests may be the best testing method for studying the genetic mechanism of resistance. It also facilitates the selection of lines with multiple resistance genes by using a range of pathotypes or isolates. However, glasshouse test is costly, and cannot be used for a large-scale resistance screening.

Testing in the laboratory

A method based on a detached leaf test has been developed at Lincoln University (Russel and Hill 1998). The correlation of disease severity or disease incidence values obtained from this method and the disease severity values for intact plants was high. The resistant or susceptible category for each cultivar in relation to each race was not changed by assay on young detached leaflets. However, for older leaflets (from the first or second oldest leaves) a susceptible rating was sometimes altered to a resistant rating. Sharma et al. (1995) developed the 'cut-twig' method to screen *Ascochyta rabiei* resistance in chickpea. This involves inoculating single cut branches with spores.

Once its reliability is confirmed, laboratory testing will find use in breeding for resistance, particularly at earlier generations when the number of seeds per line is limited. Because disease reaction can be tested using several leaflets (or a branch), the intact plant is protected for seed production.

Blight reaction in lentil has been commonly measured by a visual scale. At the International Centre for Agricultural Research in Dry Areas (ICARDA, Aleppo, Syria), a 9-point scaling system based on visual judgement of disease severity was developed, and can be used to describe the disease reaction in field plots.

The area under the disease progress curve (AUDP) has also been used to quantify host resistance (Ahmed and Morrall 1996). A high correlation between AUDP and the initial disease severity measured was found by Ahmed and Morrall (1996) using a visual scale. However, a high disease pressure was required to obtain a good correlation. This may imply that under high disease pressure they both measure the resistance conferred by major genes and, consequently, are highly correlated. Incubation period (days from inoculation to first symptom appearance) and latent period (days from inoculation to appearance of fruiting structures) have been used by Pedersen and Morrall (1994) and Ahmed et al. (1996b). These scales will be useful for identifying slow blighting and resistance conferred by minor genes.

Per cent seed infection has been used in Canada to measure resistance in lentil (Slinkard and Vandenberg 1993). The rationale is to alleviate the problem of subjectivity of foliar rating. However, the difficulties with this methods are: it is time consuming and expensive; it is not suitable for early generation selection for resistance because the limited number of seeds per line; and the resistance of early maturing materials may be underestimated because of the saprophytic infection from late maturing materials. Nevertheless, per cent seed infection is the ultimate measurement of resistance in practice, it can be used in the late stages of breeding when the amount of seed is not a problem and lines can be grouped according to their maturity.

Genetics of Ascochyta blight resistance

Breeding for resistance has been suggested as an efficient means to reduce/avoid the economic loss caused by Ascochyta blight in lentil (Erskine et al. 1994, Ye et al. 2000a). To be successful and efficient in breeding, genetic resources that contain different genes for resistance must be available, the genetic mechanisms of resistance need to be well understood, and the techniques for utilizing different genes need to be established.

Genetic resources of Ascochyta blight resistance

Genotypic differences for Ascochyta resistance are present in cultivated lentil. Table 1 gives the resistant cultivars/lines identified in several countries. Resistance to blight has also been found in wild *Lens* species. Erskine and Bayaa (1991) found 30 accessions of wild lentils with strong resistance reaction and a disease score of 1, which means no visible lesions. In 1993, they identified another 12 lines with resistance scores of 1–3, which means no visible lesions or very few scattered lesions. Bayaa and Erskine (1994) further tested the response of wild lentils to *A. lentis* from Syria and found that 24 *Lentis orientalis*, 12 *Lentis odemensis*, three *Lentis nigricans* and 36 *Lentis ervoides* accessions were resistant.

Table 1: Ascochyta blight-resistant cultivars/lines in cultivated lentil

Country	Cultivars/lines
Canada	'ILL 358', 'ILL 5588', 'ILL 5684', 'Laird'
Chile	'ILL 358', 'ILL 4605'
Ethiopia	'ILL 358', 'ILL 857'
India	'HPL5', 'L442', 'L448', 'LG169', 'LG170', 'LG171', 'LG172', 'LG173', 'LG174', 'LG176', 'LG177', 'ILL 179', 'ILL 195', 'ILL 201', 'LG209', 'LG217', 'LG218', 'LG219', 'LG221', 'LG223', 'LG225', 'LG231', 'LG232', 'LG236', 'Pant L406', 'Piant 4'
Morocco	'ILL 5698', 'ILL 5700', 'ILL 5883', 'ILL 6212'
New Zealand	'ILL 5684', 'ILL 5588', 'ILL 5714', 'Rajah'
Pakistan	'FLIP84-27L', 'FLIP84-43L', 'FLIP84-55L', 'FLIP84-85L', 'FLIP86-9L', 'FLIP86-12L', 'ILL 358', 'ILL 4605', 'ILL 5588', 'ILL 5684', 'ILL 6024', '78 S 26018', '78 S 26052', '88518', '88527', '88547', 'Masoor-93'
Syria	'ILL 857', 'ILL 2439', 'ILL 4605', 'ILL 5244', 'ILL 5588', 'ILL 5562', 'ILL 5590', 'ILL 5593', 'ILL 5684', 'ILL 5725'

Table 2: Major gene(s) conferring resistance to Ascochyta blight in lentil

Donor lines	Susceptible line	Organ	Genetic model	Reference
'Indian head'	'PI 345635'	Seed	Two duplicated recessive genes	Andrahennadi (1994)
'Indian head'	'Titore'	Foliar	Two additive recessive genes	Ye et al. (2001a)
'W6 3241' (<i>L. orientalis</i>)	'Invincible'	Foliar	One dominant gene	Ahmad et al. (1996)
'W6 3241'	'Titore'	Foliar	One dominant gene	Ye et al. (2001a)
'W6 3261' (<i>L. orientalis</i>)	'Invincible'	Foliar	One dominant gene	Ahmad et al. (1996), Parh (1998)
'W6 3261'	'Titore'	Foliar	One dominant gene	Ahmad et al. (1996)
'W6 3261'	'Titore'	Foliar	One partial dominant gene with large effect and one dominant gene with less effect	Ye et al. 2000b
'W6 3261'	'Olympic'	Foliar	One dominant gene	Ahmad et al. (1996)
'W6 3192' (<i>L. eroides</i>)	'Titore'	Foliar	Two dominant complementary genes	Ahmad et al. (1996)
'W6 3192'	'Olympic'	Foliar	Two dominant complementary genes	Ahmad et al. (1996)
'W6 3222' (<i>L. odemensis</i>)	'Titore'	Foliar	Two dominant complementary genes	Ahmad et al. (1996)
'Laird'	'Eston'	Seed	Single recessive gene	Tay and Slinkard (1989)
'Laird'	'Titore'	Foliar	Single recessive gene	Ye et al. (2001a)
'ILL 5588'	'Eston'	Foliar	Single dominant gene	Ford et al. (1999)
'ILL 5588'	'Eston'	Seed	Single dominant gene	Andrahennadi (1997), Vakulabharanam et al. (1997)
'ILL 5588'	'Eston'	Seed	Two dominant genes, one recessive gene	Tay (1989)
'ILL 5588'	'Eston'	Seed	One dominant gene one recessive gene	Sakr (1994)
'ILL 5588'	'Titore'	Foliar	One dominant gene for high resistance and one dominant gene for moderate resistance	Ye et al. (2001b)
'ILL 5684'	'Eston'	Seed	Single dominant gene	Tay and Slinkard (1989)
'ILL 5684'	'Titore'	Foliar	Single dominant gene	Ye et al. (2001a)
'ILL 5684'	'Eston'	Seed	Two dominant genes	Tay (1989)

Inheritance of Ascochyta blight resistance

The genetics of blight resistance in lentil has been studied using segregation analysis. Several major resistance genes have been discovered (Table 2). However, the allelic relationships among them are mostly unknown. Ye et al. (2001a) found that the gene conferring high resistance in 'ILL 5588' is allelic to that in 'ILL 5684'. Different models have been proposed using the same cross.

Using recombinant inbreds from two crosses between resistant ('ILL 5684' and W 63241) and susceptible ('Titore') lines, which are homozygous for the major resistance gene, Ye et al. (2001b) showed that minor genes contribute to the resistance. Although the accumulated effect of minor genes was much less than the effect of a major gene, they would provide more durable resistance than major genes at least from a theoretical viewpoint. When attempting selection within populations with the same major genotype, the use of a 1–9 scoring system is less likely to be successful. A novel system that distinguishes minor differences between infection levels is required.

Using generation mean analysis with six basic generations of four crosses, Ye (2000) showed that genetic regulation of resistance is more complicated with significant interloci interaction in three of the four crosses. Dominant effects played an important role in all crosses.

Based on these studies, it is clear that Mendelian segregation analysis, although useful, can provide only an oversimplified genetic model and that more comprehensive analysis of genetics of *Ascochyta* blight resistance in lentil is required.

Breeding for resistance

Current breeding method

Breeding for resistance to *Ascochyta* blight in lentil has been initiated only recently. It is still at a very preliminary stage, that is, large scale screening of collections of germplasm to identify resistant resources. Multilocation testing of promising cultivars from germplasm screening coordinated by ICARDA has led to the registration of several resistant cultivars in many countries (Russell 1994, Singh et al. 1994, Erskine et al. 1996). Because knowledge of the genetic variation of the pathogen and genetic regulation of host resistance is very limited, well-designed breeding programmes are not yet possible. However, the recent identification of resistance genes and their relationships in several cultivated lines (Ye et al. 2001a) and the confirmation of the presence of different pathotypes (Nasir and Bretag 1997a) has provided the basis to design a breeding programme aimed at transferring and combining these genes. Breeding for resistance should use the same method as that for other traits except that several tests for resistance should be added. A combined bulk population and pedigree selection has been used successfully in lentil and chickpea breeding (Singh 1993, Muehlbauer et al. 1995) at ICARDA. A modified version of this procedure has been used with success in breeding for *Ascochyta* blight resistance in chickpea (Singh 1993). It could be also used in breeding for *Ascochyta* blight resistance in lentil. This procedure consists of following steps.

- 1** Lines with *Ascochyta* blight resistance to multiple pathotypes are crossed to locally adapted superior cultivars. They are then backcrossed to the adapted parent (if necessary). F₁S are grown under optimal conditions to produce more seeds.
- 2** F₂S are tested for resistance in the field in epiphytotic form by artificial inoculation using inoculum prepared in the laboratory or diseased debris. Selected resistant (rating 1–4) and partially resistant (5–6) plants are bulk harvested.
- 3** F₃ and F₄ bulks are advanced under disease-free conditions and selected for growth habit, branch number, flowering time, maturity and seed characteristics. The selected plants in the F₄ generation are harvested individually.

- 4 F₅/F₆ progenies are tested for resistance using artificial infection in the field. The effects of minor genes are more obvious because of several generations of selfing and recombination.
- 5 Selected resistant lines are evaluated in preliminary yield trials. High-yielding and *Ascochyta* blight-resistant lines are selected.
- 6 Multilocation testing of the selected lines. The locations should cover the target cultivation region of the new cultivars. Tests for yield and other important traits and tests for *Ascochyta* blight resistance may need to be done separately.
- 7 On-farm testing of the selected lines and cultivar registration.

The above breeding procedure relies on multilocation testing to obtain cultivars with broad resistance. This method is justified because only resistance to the prevalent pathotypes in the target region is required by producers.

Future breeding methods

With the development of novel techniques and the increasing understanding of the host–pathogen system, more efficient breeding methods will be applicable in breeding for resistance to *Ascochyta* blight in lentil.

Multiple resistance by gene pyramiding

When there are different pathotypes and corresponding resistance genes, one method to breed for resistance is to combine different resistance genes into a single cultivar (gene pyramiding). By combining genes conferring resistance to different pathotypes, the cultivar can be used in more diversified environments where different pathotypes are likely to be dominant. In addition, multiple resistance genes may have additive effects. Even if they do not show additive effects, the presence of more genes implies that pathotypes have to be virulent to all the genes before a resistant cultivar will lose resistance (Crute 1988). This procedure has been suggested for breeding for durable resistance in many crops. A breeding programme in ICARDA adopted this method for improving resistance to *Ascochyta* blight of chickpea (Singh 1993). This method can also be used in breeding for lentil *Ascochyta* blight resistance as the different pathotypes and pathotype-specific resistance have been identified (Nasir and Bretag 1997b). The introduction of resistant cultivars with major resistance genes into production such as ‘Laird’ and ‘ILL 5588’ will increase the chance of the development of resistant pathotypes (Burdon 1993). It has been suggested that the large-scale cultivation of the moderately resistant cultivar ‘Laird’ is the cause of the increased aggressiveness of Canadian isolates of *A. lentis* (Ahmed and Morrall 1996).

Exploring slow blighting and partial resistance

Some cultivars/lines may not be highly resistant in late stages but do have considerable resistance in the early stages. Relatively slow development of disease has been identified in rust diseases in cereals, where it has been used successfully in breeding for resistance (Wilcoxson 1981). In breeding for *Ascochyta* blight resistance in chickpea, slow blighting has been explored and, consequently, a cultivar (‘ILC 482’) with partial but durable resistance was released in eight countries (Singh 1993). Although there are no reports on the existence of slow blighting in lentil, it is highly likely that this type of resistance can be identified in lentil after more carefully examining the disease reactions of more germplasm.

Using wild relatives

As discussed above, genes for Ascochyta blight resistance have been identified in wild lentil species. Therefore, to transfer the resistance genes from wild species into elite cultivars will be an important approach in breeding for resistance. The only wild species that can be intercrossed with cultivated lentil easily is *L. orientalis*, the progenitor of cultivated lentil (Ladizinsky 1979, 1993). A recent report showed that viable hybrids could be obtained from the crosses between the cultivated and four wild lentil species by applying gibberellic acid (GA₃) after pollination (Ahmad et al. 1995). Upon improvement, this technique may lead to an efficient method to transfer useful resistance genes from these wild species into cultivated species.

In vitro culture has been used to promote the use of wild relatives in lentil. Cohen et al. (1984) established a two-stage *in vitro* technique for the development of interspecific hybrid embryos. Fourteen-day-old fertilized ovules were cultured on Murashige and Skoog (MS) medium supplemented with zeatin, followed by release of the embryos from the ovular integuments. Ladizinsky et al. (1985) also obtained vegetatively normal *L. culinaris* × *L. ervoides* hybrids using embryo culture techniques. Micropropagation of the limited F₁ materials has been explored as a way to enlarge the F₁ population and eliminate the requirement of large-scale pollination to obtain enough hybrid material for further genetic study and breeding (Ye et al. 2000c).

With the refinement of *in vitro* culture techniques and an artificial crossing method, it will be possible to use resistance genes from wild species by repeated backcrossing.

Molecular technology

Genes conferring resistance are conventionally introgressed into an elite background by repeated backcrossing. If, instead of tracking the gene itself, a marker tightly linked to the gene is used to trace its segregation in the selection process, then this method is referred as marker-assisted introgression.

For resistance conferred by major genes, classical Mendelian linkage analysis can be used to identify linked markers. Using bulk segregation analysis, Ford et al. (1999) identified seven RAPD markers linked to the resistance locus conferring Ascochyta blight resistance in 'ILL 5588'. Five of the seven RAPD markers were within 30 cM of the resistance locus and the closest flanking markers were approximately 6 cM and 14 cM away from the resistance locus.

Transgenic technology provides plant breeders with new tools in breeding for resistance (Bent & Yu 1999). Genetic transformation in lentil can be achieved, as confirmed by GUS assay, but regeneration of transgenic plants was very difficult (Warkentin and McHughen 1993). However, electroporation of DNA into intact nodal meristems has resulted in the production of transgenic plants (Chowrira et al. 1995). Oktem et al. (1999) obtained transgenic shoots from cotyledonary nodes containing *GUS* gene transferred by particle bombardment. Therefore, production of transgenic lentil plants and consequently the application of transgenic techniques may soon become feasible.

Factors which need to be considered in breeding for resistance

Correlated selection response

The changes in other traits caused by selection for a trait is termed the correlated selection response (Falconer 1989). The correlations among Ascochyta blight resistance and other

agronomic traits have not been fully investigated. A recent study showed that the major gene for resistance in 'ILL 5588' and W63241 has adverse effects on seed yield/plant (Ye et al. 2001b). From the experiences of breeding for resistance in other legume species, selection for Ascochyta blight resistance may have adverse effects such as: (1) antinutritional factors may be increased, which is undesirable both for human and animal consumption (Helsper 1998); (2) digestibility may be reduced (Helsper 1998); and (3) maturity may be delayed (Singh 1993, Porta-Puglia et al. 1994).

Genotype × environment interaction

Genotype × environment (GE) interaction is of concern if the resultant cultivar is to be used across a large area. There are two reasons why GE interaction is more important in breeding for resistance than for other traits. First, pathogens may vary in their aggressiveness under different environments. Furthermore, physiological races may be different across environments. Second, the growth, development and physiological status of host genotypes may change across environments. There is a paucity of information regarding the GE interaction in lentil. However, the different levels of aggressiveness among isolates from different locations and the recent identification of pathotypes suggest that GE interaction could be important.

Conclusion

Considerable progress has been made in the last two decades in understanding the Ascochyta blight pathogen and the resistance of its host. Resistance sources are available both in cultivated and wild lentil species. Several major resistance genes have been identified, and minor genes have also been shown to play a role in resistance. Different pathotypes are present in the pathogen population and resistances conferred by major genes seem to be pathotype-specific. However, many challenges remain both for plant pathologists and for breeders. From a plant breeding viewpoint, more studies in the following areas are urgently required to sustain breeding progress.

Identification of more resistance resources

Large-scale screening of germplasm for resistance is required. What is important for the future screening of germplasm is to characterize the resistance reactions more carefully. It is necessary to evaluate the resistance several times and to test against different isolates. Three types of germplasm should be selected: (1) highly resistant with rating 1–2, (2) germplasm with slow blighting and (3) germplasm with multiple resistance.

Careful characterization of the host–pathogen interaction system

To identify and incorporate new sources of resistance genes into the breeding programme, it is necessary to have a good understanding of the pathogenic variability of *A. lentis*. While six different pathotypes are known, more pathotypes may be present. Molecular marker-based methods have been used successfully to provide additional information on the pathogenic variability and have the potential to fingerprint pathotypes in many host–pathogen systems, including Ascochyta blight in chickpea (Weising et al. 1991, Udupa et al. 1998). It can be expected that a better understanding of the pathogenic variability of *A. lentis* will soon be obtained by using these novel techniques. Armed with the understanding of pathogen populations, the host genotypes can be easily determined using different pathotypes to challenge host genotypes and, consequently, gene pyramiding becomes feasible.

Identification of molecular markers tightly linked to different resistance genes

With the development of modern molecular techniques, more and more marker systems become available and their costs will be reduced. It is possible to develop rapid and cost-effective techniques for screening large populations for markers linked to resistance genes. Once developed, the benefits of marker-assisted selection (introgression) will be available.

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