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REVIEW PAPER

Resistance to ascochyta blights of cool season food legumes

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Abstract Ascochyta blights are the most important diseases of cool season food legumes (peas, lentils, chickpeas, and faba beans) and are found in nearly all production regions. Despite having the same common disease name, the pathogen species differ for each of the crops. These diseases cause serious yield losses under favourable cool and humid conditions. Planting resistant cultivars is often the first choice and most economical means in managing the diseases. Therefore breeding for resistance to ascochyta blights has been an important objective of many cool season food legume research programmes. Systematic screening of germplasm collections at international research centres and other national research programmes have identified useful resistance sources that have been used successfully to breed resistant or tolerant cultivars. Genetic studies have revealed inheritance patterns of the resistance genes. Genetic linkage analyses and QTL mapping have identified molecular markers that could be useful for marker-assisted selection and gene pyramiding. In general, research towards developing resistance to ascochyta blights in cool season food legume faces mainly two limitations: the lack of availability of efficient resistance sources and the lack of a good understanding of the variability of the pathogen populations. Research efforts to alleviate these limitations should be pursued. Given that modern technologies of marker development and genomics are available, further advances in deploying resistance to manage ascochyta blights in this group of legume crops will depend on concerted efforts in developing accurate screening procedures with adequate knowledge of pathogen variability and identification of additional sources of resistance.

Keywords Disease resistance · Quantitative trait loci · Marker assisted selection · Disease screening · Inheritance · Breeding for disease resistance · *Pisum sativum* · Peas · *Lens culinaris* · Lentil · *Cicer arietinum* · Chickpea · *Vicia faba* · Faba bean

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Introduction

Peas (*Pisum sativum*), lentil (*Lens culinaris*), chickpea (*Cicer arietinum*) and faba bean (*Vicia faba*) are important food crops throughout the world and are produced on nearly 25 million hectares with annual production approaching 40 million metric tons (FAOSTAT 2004). Total production ranges from over 20 million metric tons for pea to about 4 million metric tons for lentil (FAOSTAT 2004). These cool season food legumes are affected by a number of foliar and root diseases that cause wide spread damage and in severe cases cause complete crop

loss. The most important foliar diseases worldwide are ascochyta blights. Although the diseases are collectively referred to as ascochyta blights due to similar symptoms, the pathogen species differ for each of the crops (Hernandez-Bello et al. 2006) and host specificity is necessary for disease development. The ascochyta blight complex of pea involves three pathogens, *Ascochyta pisi*, *Mycosphaerella pinodes*, and *Phoma medicaginis* var. *pinodella* (formerly *Ascochyta pinodella*). The disease is a complex because the three pathogens cause more or less similar symptoms and they frequently occur together. In the case of lentil, the crop is affected by *Ascochyta lentis* that causes leaf and stem spotting, leaf drop, stem lesions and seed lesions that result in serious reductions of yield and crop quality. Ascochyta blight of chickpea is caused by *Ascochyta rabiei* (*Didymella rabiei*) that causes severe symptoms on the leaves, stem breakage and die back, and often is cited as causing complete crop loss (Nene and Reddy 1987). Similar symptoms on faba bean incited by *Ascochyta fabae* cause yield losses and reduce seed quality.

Resistance to ascochyta blight in the cool season food legumes has been sought through germplasm exploration, collection, and systematic evaluation. Sources of partial resistance have been identified in all of the cool season food legumes and the currently available resistance is being used in breeding programmes designed to develop cultivars with improved resistance. Since there are some recent reviews on breeding methods, screening procedures, the ascochyta diseases and biology of the pathogens (Bretag et al. 2006; Pande et al. 2005; Tivoli et al. 2006; Torres et al. 2006; Ye et al. 2002), we will in this review mainly focus on the recent developments in understanding the genetics of host resistance for each of the major cool season food legumes and point out immediate needs in research that in our opinion will further advance deployment of resistance in managing ascochyta blight in cool season food legumes.

Peas

Ascochyta blight of pea is a disease complex caused by three pathogens: *Ascochyta pisi* which causes well-defined lesions (spots) on leaves, stems and pods; *Phoma medicaginis* var. *pinodella*, previously *Ascochyta pinodella*, which causes lesions on leaves and stems, and foot rot; and *Mycosphaerella pinodes* which causes blight starting with small purple to black spots, enlarging and turning brown to black. The disease complex, its epidemiology, screening techniques and management were recently reviewed (Tivoli et al. 2006). Methods of screening for resistance have relied on field nurseries and natural infection by the pathogen through dispersal of airborne ascospores from debris of previously infected pea crops. In general no complete resistance to the ascochyta blight complex has been identified in peas; however good sources of partial resistance have been identified and are being used in breeding programmes (Tivoli et al. 2006). Screening of the USDA-ARS collection of pea germplasm was successful in identifying sources of partial resistance to *M. pinodes* in five accessions (PI 142441, PI 142442, PI 381132, PI 404221 and PI 413691); however, none of the accessions were more resistant than the cv. Radley. Other sources of resistance have been identified and include cv. Carneval (Tar'an et al. 2003) and accessions JI 97 and JI 1089 from the John Innes Institute Collection. Accessions of the wild pea species, *P. fulvum* have also shown some resistance to ascochyta blight.

A relationship between lodging traits and resistance to ascochyta blight was found (Banniza et al. 2005) indicating that stem structural components may have a role in resistance. Thus, upright plant habit with resistance to lodging appears to be an important factor in reducing disease severity.

Most of the genetic studies on pea resistance focused on resistance to *M. pinodes* possibly because it is the most destructive pathogen of the three involved. The genetics of resistance to ascochyta blight in peas indicate a multiple gene system with some dominance and additive genetic effects (Wroth 1999). Estimates of quantitative trait loci (QTL) for resistance to ascochyta blight have ranged from three (Tar'an et al. 2003) to 13 (Timmerman-Vaughan et al. 2004) (Table 1). The complex nature of resistance, as indicated by the number of QTL that have been estimated, presents a challenge to breeders and the prospect of making use of marker assisted selection for ascochyta blight resistance in peas.

Development of cultivars with improved resistance to the disease will depend on the use of germplasm with partial resistance to ascochyta blight with

Table 1 Quantitative trait loci (QTL) associated with ascochyta blight resistance in peas

Loci	% Variation accounted for	Reference
6 QTL in seedling stage	76.0	Prioul et al. (2004)
10 QTL in adult stage	56.6–67.1	Prioul et al. (2004)
14 QTL in 7 linkage groups	Not estimated	Timmerman-Vaughan et al. (2004)
3 QTL	35.9	Tar'an et al. (2003)

consideration for plant traits such as good standing ability that has been shown to be associated with resistance. In general, developing pea cultivars resistant to ascochyta blight is rather challenging because of the number of pathogens involved, pathogenic variation (races or pathotypes) within each pathogen species and seemingly tissue or growth stage specificity of certain resistance genes, in addition to lack of efficient resistance sources. Marker-assisted selection may be attempted but the number of QTL that are estimated to be involved with disease expression would seem to make that approach difficult and time-consuming. Direct screening in the presence of the disease may be a more viable approach at the present time until efficient marker-assisted selection protocols are established. Needless to say, more efficient resistance sources are needed and additional exploration and collection in regions of diversity may be a fruitful approach. Also, the use of wild species such as *P. fulvum* may hold promise as a source of resistance genes.

Lentil

Ascochyta blight of lentil, caused by *Ascochyta lentis* (teleomorph: *Didymella lentis*), has world-wide distribution and causes extensive damage to yields and crop quality. The disease causes necrotic spots on the leaflets, stems, pods and seeds. The lesion spots are initially light grey and turn tan, and are surrounded by darker margins. Lesions often enlarge and coalesce causing blight and stem breakage. Tivoli et al. (2006) provided a thorough review of sources of resistance and screening techniques for ascochyta blight of lentil and Ye et al. (2002) gave an account of breeding techniques for selection of lentils with resistance to ascochyta blight. Partial resistance to the disease is available in the germplasm. Most notable of the partially resistant germplasm accessions are lentil accessions PI 339283, PI 374118, ILL5588, ILL5684, PR86-360, and ILL7537. Other accessions have been reported as resistant and have been used in breeding. The sources of resistance are readily available from gene banks at ICARDA, the U.S. Department of Agriculture-Agricultural Research Service, Canada and Australia. Screening for resistance has generally relied on field screening; however, screening in controlled environments has been practiced with good results (Muehlbauer, personal observations).

Resistance to ascochyta blight in lentil has been reported, but theories abound with the number and nature of genes responsible for the observed resistance. Two complementary dominant genes for resistance were postulated (Ahmad et al. 1997) in a cross of *L. ervoides* × *L. odemensis* whereas a single dominant gene was found in crosses within *L. culinaris*. Ford et al. (1999) identified a single dominant gene, *Abr*(1), in accession ILL5588 that conferred resistance to ascochyta blight in lentil and also identified molecular markers flanking the resistance gene that may be useful in marker-assisted selection. Chowdhury et al. (2001) postulated that a single recessive gene conferred resistance to ascochyta blight in lentil and was linked to RAPD markers, UBC227 and OPD-10. These RAPD markers are currently being used in marker-assisted selection. Nguyen et al. (2001) studied the resistance in germplasm accession ILL7537 and found that two complementary dominant genes conferred resistance. Ye et al. (2003) found two dominant genes in ILL5588 with one gene for resistance and the other for partial resistance, and one or two recessive genes in Laird and Indianhead, respectively. Additionally, two complementary resistance genes were found in the susceptible lines W6-3192 and Titore (Ye et al. 2003). At least five QTL for blight resistance have been mapped on four linkage groups and they together accounted for 50% of phenotypic variation (Rubeena et al. 2006). It appears that at least two genes are involved in resistance to ascochyta blight in

lentil, and the nature of the genes (whether dominant or recessive) depends on the sources. Based on current information it is not possible to make comparisons of the genes that have been identified or to draw conclusions on the number of genes involved. Appropriate allelism tests need to be conducted using common parents followed by uniform and systematic screening of the progenies. It is clear that the expression of those genes may be altered by variable environmental conditions which can alter the interpretation of the mode of action of the genes. Nevertheless, their use in breeding has led to the development of resistant cultivars such as Milestone (Vandenberg et al. 2001) and other candidate breeding lines with resistance.

Variation in virulence patterns of *Ascochyta lentis* has been reported (Ahmed et al. 1996; Ahmed and Morrall 1996; Nasir and Bretag 1997) and six pathotypes have been classified in Australia (Nasir and Bretag 1997). The cultivar Laird, released in Canada, was initially described as resistant to ascochyta blight but is now classified as susceptible. The reduced resistance of cv. Laird was reported to be due to the appearance of more virulent pathotypes (Ahmed et al. 1996). The pathogenic variation has undoubtedly contributed to the confusion about the genetics of resistance in lentil to ascochyta blight. Needless to say, these findings have important implications for lentil breeding and point out the need to consider pathogen variation during evaluation and selection for improved resistance.

Chickpea

Ascochyta blight, caused by *Ascochyta rabiei* [teleomorph: *Didymella rabiei* var. Arx], is responsible for widespread damage to chickpea crops worldwide. The disease causes necrotic lesions on the leaflets, stems, pods and seeds. Symptoms initially appear as water-soaked lesions on stems and leaves and turn to sunken, dark brown lesions with concentric black speckles of pycnidia. Lesions enlarge and coalesce under conditions favourable to the disease, causing leaf blight, stem girdling, stem breakage and plant death. Pande et al. (2005) provided a review of pathogen biology and the disease management of ascochyta blight. A thorough review of ascochyta blight of chickpea and available sources of resistant germplasm was recently completed (Tivoli et al. 2006). Most notable of the partially resistant germplasm include accessions from ICARDA such as ILC-72, ILC-3279, ILC-3868, ILC-3870, ILC-3996 and numerous FLIP lines that have shown resistance at multiple locations (Reddy and Singh 1984). Breeding lines from ICARDA such as FLIP90-98C, FLIP91-22C, FLIP91-46C, FLIP91-2C, FLIP91-24C, FLIP91-50C, FLIP91-54C, and FLIP91-18C, developed from resistance sources ILC-72 and ILC-3279, have also shown a degree of resistance in the field and in controlled environments (Singh and Reddy 1994). These accessions and others developed at ICARDA have been used in breeding programmes worldwide to develop resistant cultivars. Progress continues to be made in the development of breeding lines with improved resistance to the disease.

Studies of the genetics of resistance to ascochyta blight have relied on the use of recombinant inbred lines (RILs) from crosses between resistant and susceptible parents and QTL analyses. Santra et al. (2000) used a set of RILs from the cross of FLIP84-92C × *C. reticulatum* (PI 599072) to identify two QTL (*QTL*-1 and *QTL*-2) that in combination accounted for 50.3 and 45.0% of the variation in blight scores, respectively, over two years of evaluation. Other studies (Flandez-Galvez et al. 2003; Collard et al. 2003; Iruela et al. 2006) have identified QTL for blight resistance in comparable regions of the genome as those found by Santra et al. (2000) providing confidence in the presumed locations of the resistance genes and prospects for marker-assisted selection and eventual map-based cloning. Likewise, Lichtenzveig et al. (2006) found three QTL for resistance that were located on comparable linkage groups, and a significant epistatic interaction of the resistance QTL on linkage group 8 with flowering time.

Marker density in the *QTL*-1 region of the chickpea genome was increased by Rakshit et al. (2003) who used bulked segregant analysis and DNA amplified fingerprinting (DAF) to identify a marker directly at the peak of *QTL*-1 of Santra et al. (2000). Millan et al. (2003) also identified additional markers linked to resistance and showed their potential use in selection. Efforts are currently underway towards fine mapping of *QTL*-1 using Bacterial Artificial Chromosome (BAC) libraries (Rajesh et al. 2004). The BACs of interest are being identified through the use

of markers associated with *QTL*-1 followed by BAC end sequencing to identify single nucleotide polymorphisms for conversion to CAPs and dCAPs markers. Those markers are then being used to increase marker density within *QTL*-1. The increased marker density provides additional markers for possible use in marker-assisted selection and should facilitate cloning and characterization of the resistance genes.

The existence of pathotypes of *Didymella rabiei* must be considered in breeding programmes designed to develop resistant cultivars. There has been a plethora of classification schemes for pathogenic variation in *D. rabiei*, ranging from an initial description of six races of the pathogen (Singh and Reddy 1993) to 14 virulence forms or pathotypes (Chen et al. 2004). The current trend is a more workable classification into either two or three pathotypes (Udupa et al. 1998; Chen et al. 2004). Using a mini-dome technique, Chen et al. (2004) was able to assign isolates of *A. rabiei* from the U.S. Pacific Northwest into two pathotypes (I&II). Isolates from the two-pathotype system were used to map pathotype-specific QTL conferring resistance and to study the mechanisms of resistance in the host (Chen et al. 2005; Cho et al. 2004; Cho et al. 2005). The two-pathotype system explains the evolution of US chickpea cultivars (Chen et al. 2004). The initial chickpea cultivars (such as Spanish White and UC-5) introduced into the US Pacific Northwest were shown to be highly susceptible to both pathotypes I and II; cultivars developed through breeding for resistance (such as Sanford and Dwelley) released in the early 1990s had only resistance to pathotype I, while more recently released cultivars (such as Sierra) were shown to have resistance to pathotype I and a high level of tolerance to pathotype II (Chen et al. 2005). Our current chickpea breeding efforts are to incorporate more efficient resistance genes to improve resistance against pathotype II and to avoid the potential emergence in the US of a new pathotype that is highly virulent on chickpea ICC 12004 reported in Syria (Bayaa et al. 2004).

Faba bean

Ascochyta blight of faba bean is caused by *Ascochyta fabae*, (teleomorph *Didymella fabae*) which is highly specific for faba bean. Lesions with definite margins are more or less circular or oval, slightly sunken on leaves, and more sunken on stems and pods. The disease, screening procedures and procedures for breeding disease-resistant faba bean cultivars were the subjects of a recent review (Tivoli et al. 2006). Screening for resistance has relied on the use of field nurseries and natural infection by the pathogen which may be supplemented by artificial inoculation with the pathogen or by the spreading of infected crop debris in the nursery area. Races of the pathogen have been suggested; however, classification into races has been controversial. Numerous sources of resistance listed by Tivoli et al. (2006) are being used in breeding programmes to develop improved cultivars.

A major dominant gene for resistance to ascochyta blight of faba bean was reportedly found in ILB752 and two complementary recessive genes for resistance were found in NEB463 (Kohpina et al. 2000). A detailed analysis of resistance using an F_2 population from the cross of 29H (resistant) × VF136 (susceptible) was used to identify six QTL (Avila et al. 2004). The F_2 population was evaluated for resistance to two isolates differing in their pathogenicity. Four of the QTL were effective against both pathotypes while the effectiveness of the two other QTL varied. Some QTL appeared to be tissue (either leaf or stem) specific (Avila et al. 2004), complicating selection protocols in breeding.

Variability of isolates of the ascochyta blight pathogen like those observed in Australia (Kohpina et al. 1999) is problematic for breeding and it is necessary to evaluate segregating breeding material against a range of isolates to ensure success.

Summary and conclusions

Ascochyta blights are an important yield constraint of all cool season food legumes, and using host resistance is the most economical means in managing the diseases. Resistance to ascochyta blights is present in the germplasm of all cool season food legumes; however, in most cases no complete resistance is found in the cultivated germplasm and the resistance is considered to be partial. Nevertheless, the available resistance has been demonstrated capable of reducing losses of yield and quality of these grain legumes. There is a pressing need for increased understanding

of pathogen variability, and for the standardization of screening procedures including the methods of inoculation and disease-scoring procedures, since the isolates being used for inoculation will be location-specific and disease progression will vary. Nevertheless, standardization of scoring procedures and the use of common host differentials and isolates as controls will enable comparisons of the data and results of evaluations across research locations.

The inheritance of resistance to ascochyta blights in cool season food legumes appears to be quantitative and controlled in most cases by multiple QTL. It is interesting to note that the number of QTL estimated using early generation populations such as F_2 is greater than the number of QTL estimated using nearly-homozygous recombinant inbred line populations, indicating that the latter may be a more realistic estimate of the inheritance of resistance and the location of the important genes. The use of marker-assisted selection for resistance to ascochyta blights is being developed in all of the cool season food legumes. However, it is still limited in scope, and its practical application requires further experimentation and confirmation. Selection under natural conditions in the field using a mixture of isolates remains the primary means of selection for resistance. The mini-dome procedure (Chen et al. 2005) has greatly improved the efficiency of evaluation of selections for resistance to multiple pathotypes in chickpea. Improved cultivars with resistance to ascochyta blights have been the result of breeding programmes worldwide. Seeking new resistance sources of additional germplasm lines or wild relatives will make it possible to continue to improve on that resistance. The prospect of pyramiding of genes, once identified, from various sources with the aid of modern molecular techniques has been discussed, and remains a possible fruitful approach for further improving resistance to ascochyta blights in cool season food legumes.

References

Ahmed, S., & Morrall, R. A. A. (1996). Field reactions of lentil lines and cultivars to isolates of *Ascochyta fabae* f. sp. *lentis*. *Canadian Journal of Plant Pathology, 18*, 362–369.

Ahmed, S., Morrall, R. A. A., & Sheard, J. W. (1996). Virulence of *Ascochyta fabae* f. sp. lentis on lentil. *Canadian Journal of Plant Pathology, 18*, 354–361.

Ahmad, M., Russell, A. C., & McNeill, D. L. (1997). Identification and genetic characterization of different resistance sources to Ascochyta blight within the genus *Lens*. *Euphytica, 97*, 311–315.

Avila, C. M., Satovic, Z., Sillero, J. C., Rubiales, D., Moreno, M. T., & Torres, A. M. (2004). Isolate and organ-specific QTLs for Ascochyta blight resistance in faba bean (*Vicia faba* L). *Theoretical and Applied Genetics, 108*, 1071–1078.

Banniza, S., Hashemi, P., Warkentin, T. D., Vandenberg, A., & Davis, A. R. (2005). The relationships among lodging, stem anatomy, degree of lignification, and resistance to Mycosphaerella blight in field pea (*Pisum sativum*). *Canadian Journal of Botany, 83*, 954–967.

Bayaa, B., Udupa, S. M., Baum, M., Malhotra, R. S., & Kabbabeh, S. (2004). Pathogenic variability in Syrian isolates of *Ascochyta rabiei*. In *Proceedings of the 5th European Conference on Grain Legumes* (p. 306, Abstract). Dijon France, 7–11 June 2004.

Bretag, T. W., Keane, P. J., & Price, T. V. (2006). The epidemiology and control of Ascochyta blight in field peas: A review. *Australian Journal of Agricultural Research, 57*, 883–902.

Chen, W., Coyne, C., Peever, T., & Muehbauer, F. J. (2004). Characterization of chickpea differentials for Ascochyta blight and identification of resistance sources for *Ascochyta rabiei*. *Plant Pathology, 53*, 759–769.

Chen, W., Mcphee, K. E., & Muehlbauer, F. J. (2005). Use of a mini-dome bioassay and grafting to study resistance of chickpea to Ascochyta blight. *Journal of Phytopathology, 153*, 579–587.

Cho, S., Chen, W., & Muehlbauer, F. J. (2004). Pathotype-specific genetic factors in chickpea (*Cicer arietinum* L.) for quantitative resistance to Ascochyta blight. *Theoretical and Applied Genetics, 109*, 733–739.

Cho, S., Chen, W., & Muehlbauer, F. J. (2005). Constitutive expression of the Flavanone 3-hydroxylase gene related to pathotype-specific Ascochyta blight resistance in *Cicer arietinum* L. *Physiological and Molecular Plant Pathology, 67*, 100–107.

Chowdhury, M. A., Andrahennadi, C. P., Slinkard, A. E., & Vandenberg, A. (2001). RAPD and SCAR markers for resistance to Ascochyta blight in lentil. *Euphytica, 118*, 331–337.

Collard, B. C. Y., Pang, E. C. K., Ades, P. K., & Taylor, P. W. J. (2003). Preliminary investigation of QTLs associated with seedling resistance to Ascochyta blight from *Cicer echinospermum*, a wild relative of chickpea. *Theoretical and Applied Genetics, 107*, 719–729.

FAOSTAT. (2004). Source of statistical data on production. http://faostat.fao.org.

Flandez-Galvez, H., Ades, P. K., Ford, R., Pang, E. C. K., & Taylor, P. W. J. (2003). QTL analysis for Ascochyta blight resistance in an intraspecific population of chickpea (*Cicer arietinum* L.). *Theoretical and Applied Genetics, 107*, 1257–1265.

Ford, R., Pang, E. C. K., & Taylor, P. W. J. (1999). Genetics of resistance to Ascochyta blight (*Ascochyta lentis*) of lentil and the identification of closely linked RAPD markers. *Theoretical and Applied Genetics, 98*, 93–98.

Hernandez-Bello, M. A., Chilvers, M. I., Akamatsu, H., & Peever, T. L. (2006). Host specificity of *Ascochyta* spp.

infecting legumes of the *Viciae* and *Cicereae* tribes and pathogenicity of the interspecific hybrid. *Phytopathology, 96*, 1148–1156.

Iruela, M., Rubio, J., Barro, F., Cubero, J. I., Millan, T., & Gil, J. (2006). Detection of two quantitative trait loci for resistance to Ascochyta blight in an intra-specific cross of chickpea (*Cicer arietinum* L.): Development of SCAR markers associated with resistance. *Theoretical and Applied Genetics, 112*, 278–287.

Kohpina, S., Knight, R., & Stoddard, F. L. (1999). Variability of *Ascochyta fabae* in South Australia. *Australian Journal of Agricultural Research, 50*, 1475–1481.

Kohpina, S., Knight, R., & Stoddard, F. L. (2000). Genetics of resistance to Ascochyta blight in two populations of faba bean. *Euphytica, 112*, 101–107.

Lichtenzveig, J., Bonfil, D. J., Zhang, H. B., Shtienberg, D., &Abbo, S. (2006). Mapping quantitative trait loci in chickpea associated with time to flower and resistance to *Didymella rabiei* the causal agent of Ascochyta blight. *Theoretical and Applied Genetics, 113*, 1357–1369.

Millan, T., Rubio, J., Iruela, M., Daly, K., Cubero, J. I., & Gil, J. (2003). Markers associated with Ascochyta blight resistance in chickpea and their potential in marker-assisted selection. *Field Crops Research, 84*, 373–384.

Nasir, M., & Bretag, T. W. (1997). Pathogenic variability in Australian isolates of *Ascochyta lentis*. *Australasian Plant Pathology, 26*, 217–220.

Nene, Y. L., & Reddy, M. V. (1987). Chickpea diseases and their control. In M. C. Saxena & K. B. Singh (Eds.), *The chickpea* (pp. 233–270). Oxon, UK: CAB Wallingford.

Nguyen, T. T., Taylor, P. W. J., Brouwer, J. B., Pang, E. C. K., & Ford, R. (2001). A novel source of resistance in lentil (*Lens culinaris* ssp *culinaris*) to Ascochyta blight caused by *Ascochyta lentis*. *Australasian Plant Pathology, 30*, 211–215.

Pande, S., Siddique, K. H. M., Kishore, G. K., Bayaa, B., Gaur, P. M., Gowda, C. L. L., Bretag, T. W., & Crouch, J. H. (2005). Ascochyta blight of chickpea (*Cicer arietinum* L.): A review of biology, pathogenicity, and disease management. *Australian Journal of Agricultural Research, 56*, 317–332.

Prioul, S., Frankewitz, A., Deniot, G., Morin, G., & Baranger, A. (2004). Mapping of quantitative trait loci for partial resistance to *Mycosphaerella pinodes* in pea (*Pisum sativum* L.) at the seedling and adult plant stages. *Theoretical and Applied Genetics, 108*, 1322–1334.

Rajesh, P. N., Coyne, C., Meksem, K., Sharma, K. D., Gupta, V., & Muehlbauer, F. J. (2004). Construction of a *Hind*III bacterial artificial chromosome library and its use in identification of clones associated with disease resistance in chickpea. *Theoretical and Applied Genetics, 108*, 663–669.

Rakshit, S., Winter, P., Tekeoglu, M., Munoz, J. J., Pfaff, T., Ko-Iseppon, A. M., Muehlbauer, F. J., & Kahl, G. (2003). DAF marker tightly linked to a major locus for Ascochyta blight resistance in chickpea (*Cicer arietinum* L.). *Euphytica, 132*, 23–30.

Reddy, M. V., & Singh, K. B. (1984). Evaluation of a world collection of chickpea germplasm accessions for resistance to Ascochyta blight. *Plant Disease, 68*, 900–1001.

Rubeena, T., Taylor, P. W. J., Ades, P. K., & Ford, R. (2006). QTL mapping of resistance in lentil (*Lens culinaris* ssp. *culinaris*) to Ascochyta blight (*Ascochyta lentis*). *Plant Breeding, 125*, 506–512.

Santra, D. K., Tekeoglu, M., Ratnaparkhe, M., Kaiser, W. J., & Muehlbauer, F. J. (2000). Identification and mapping of QTLs conferring resistance to Ascochyta blight in chickpea. *Crop Science, 40*, 1606–1612.

Singh, K. B., & Reddy, M. V. (1993). Resistance to 6 races of *Ascochyta rabiei* in the world germplasm collection of chickpea. *Crop Science, 33*, 186–189.

Singh, K. B., & Reddy, M. V. (1994). Registration of 8 Ascochyta blight-resistant, early-maturing, large-seeded chickpea germplasms. *Crop Science, 34*, 1416–1417.

Tar'an, B., Warkentin, T., Somers, D. J., Miranda, D., Vandenburg, A., Blade, S., Woods, S., Bing, D., Xue, A., DeKoeyer, D., & Penner, G. (2003). Quantitative trait loci for lodging resistance, plant height and partial resistance to Mycosphaerella blight in field pea (*Pisum sativum* L.). *Theoretical and Applied Genetics, 107*, 1482–1491.

Tivoli, B., Baranger, A., Avila, C. M., Banniza, S., Barbetti, M., Chen, W., Davidson, J., Lindeck, K., Kharrat, M., Rubiales, D., Sadiki, M., Sillero, J. C., Sweetingham, M., & Muehlbauer, F. J. (2006). Screening techniques and sources of resistance to foliar diseases caused by major necrotrophic fungi in grain legumes. *Euphytica Special Issue: Resistance to Biotic and Abiotic Stresses in Legumes, 147*, 223–253.

Timmerman-Vaughan, G. M., Frew, T. J., Butler, R., Murray, S., Gilpin, M., Falloon, K., Johnston, P., Lakeman, M. B., Russell, A., & Khan, T. (2004). Validation of quantitative trait loci for Ascochyta blight resistance in pea (*Pisum sativum* L.), using populations from two crosses. *Theoretical and Applied Genetics, 109*, 1620–1631.

Torres, A. M., Roman, B., Avila, C. M., Satovic, Z., Rubiales, D., Sillero, J. C., Cubero, J. I., & Moreno, M. T. (2006). Faba bean breeding for resistance against biotic stresses: Towards application of marker technology. *Euphytica, 147*, 67–80.

Udupa, S. M., Weigand, F., Saxena, M. C., & Kahl, G. (1998). Genotyping with RAPD and microsatellite markers resolves pathotype diversity in the Ascochyta blight pathogen of chickpea. *Theoretical and Applied Genetics, 97*, 299–307.

Vandenberg, A., Kiehn, F. A., Vera, C., Gaudiel, R., Buchwaldt, L., Kirkland, K. J., Morrall, R. A. A., Wahab, J., & Slinkard, A. E. (2001). CDC Milestone lentil. *Canadian Journal of Plant Science, 81*, 113–114.

Wroth, J. M. (1999). Evidence suggests that *Mycosphaerella pinodes* infection of *Pisum sativum* is inherited as a quantitative trait. *Euphytica, 107*, 193–204.

Ye, G., McNeil, D. L., & Hill, G. D. (2002). Breeding for resistance to lentil Ascochyta blight. *Plant Breeding, 121*, 185–191.

Ye, G., McNeil, D. L., & Hill, G. D. (2003). Inheritance of foliar resistance to Ascochyta blight in lentil (*Lens culinaris*). *New Zealand Journal of Crop and Horticultural Science, 31*, 77–83.