Eur J Plant Pathol (2007) 119:111–118
DOI 10.1007/s10658-007-9116-x

REVIEW

The sympatric Ascochyta pathosystems of Near Eastern legumes, a key for better understanding of pathogen biology

S. Abbo · O. Frenkel · A. Sherman · D. Shtienberg

Received: 16 October 2006 / Accepted: 23 February 2007 / Published online: 22 March 2007

Abstract The primary and secondary centres of origin of domesticated plants are often also the places of origin of their pathogens. Therefore, the Near Eastern cradle of agriculture, where crop plants, their wild progenitors, and other congeneric taxa grow sympatrically, may hold some clues on the biology of the pathogens of the respective crops. Unlike the situation in the well-studied Near Eastern cereals and their important diseases, hardly any data are available on basic questions regarding grain legumes. What is the role of genetic diversity at resistance loci of the wild hosts and is it greater compared with the cultigens? Are populations of Ascochyta pathogens infecting wild legumes genetically distinct from populations infecting their domesticated counterparts, and if so, is this differentiation related to differences in host specialization or to adaptation to different ecological conditions? Do isolates originating from wild taxa exhibit a similar level of aggressiveness and have different aggressiveness alleles compared with those originating from domesticated grain legumes? In this review we propose an experimental framework aimed at gaining answers to some of the above questions. The proposed approach includes comparative epidemiology of wild vs. domesticated plant communities, co-evolutionary study of pathogens and their hosts, phenotypic and genetic characterization of fungal isolates from wild and domesticated origins, and genetic analyses of pathogenicity and parasitic fitness among progeny derived from crosses between isolates from wild and domesticated hosts.

Keywords Ascochyta pathogens · Grain legumes · Wild hosts

S. Abbo (✉) · O. Frenkel
The Levi Eshkol School of Agriculture, The Hebrew University of Jerusalem, Rehovot 76100, Israel
e-mail: abbo@agri.huji.ac.il

O. Frenkel · A. Sherman
Genomics Department, the Volcani Center, Agricultural Research Organization, Bet Dagan 50250, Israel

D. Shtienberg
Department of Plant Pathology and Weed Research, the Volcani Center, Agricultural Research Organization, Bet Dagan 50250, Israel

Introduction

Plant pathogens are a major evolutionary force operating in natural ecosystems and in domesticated plant communities (Burdon 1987). In natural ecosystems, both hosts and their pathogens survive but the situation hardly takes the form of an epidemic. Hence, it has long been recognized that the severe epidemics that devastate crop

plants are largely artefacts of cultivation and a result of co-evolution under domestication (Harlan 1976; Burdon 1987). Therefore, gaining better understanding of host–pathogen interaction in its natural state may improve our understanding of the situation in man-made habitats.

The primary and secondary centres of origin of cultivated plants are often also the places of origin of their pathogens (Leppik 1970). Therefore, the Near Eastern cradle of agriculture (Lev-Yadun et al. 2000), where the wild progenitors of the Near Eastern crops and their con-generic taxa co-exist in natural plant formations, may hold some clues on the biology of the pathogens of the respective crops. Near Eastern farming began with the adoption of a small number of crop plants (Zohary and Hopf 2000). This 'Founder Crops' package included einkorn wheat, emmer wheat, barley, lentil, pea, chickpea, bitter vetch, and flax (Zohary and Hopf 2000). At a later stage, additional plants were added to this package including clovers, vetches, medics, fruit trees and vegetables. The farming-based economy spread from the Near East into Europe, Central and East Asia, North and East Africa, and in recent times also into the New World (Diamond 1997). Naturally, wherever the ecological conditions allow, the pathogens of the respective crop plants followed suit. For example, Ascochyta blight pathogens were detected both in the USA and Australia a few years after large scale production of chickpea and lentils was established in these countries and are now considered a major agronomic problem (e.g., Kaiser 1997).

Unlike the situation described above for the USA or Australia, domesticated crop plants grow sympatrically with their wild relatives in the east Mediterranean, (Harlan and Zohary 1966; Zohary 1973). Whenever crop plants grow adjacent to natural ecosystems harbouring stands of wild forms, gene flow between the cultigens and their wild relatives is possible. Indeed, many such examples were described, e.g., for beans in Mexico (Zizumbo-Villarreal et al. 2005), sorghum in Israel and rice in India (Abbo and Rubin 2000). In theory, similar processes can occur between the pathogen populations that exist in such sympatric cropping systems. However, to the best of our knowledge, despite old reports that Ascochyta pathogens occur on wild legumes in Israel (e.g., Barash 1960), the genetic affinities between the Ascochyta pathogens of the Near Eastern legumes and their relatives infecting the wild forms were hardly studied. In this review we address the *Cicer*/Ascochyta blight system in wild and in man-made ecosystems (cultivation) as a test case for other Ascochyta pathosystems and flag knowledge gaps relevant for better understanding of the underlying host–pathogen interaction.

Evolutionary, agronomic and ecological considerations

Wheat, barley, pea, lentil and flax spread in prehistoric times around the Mediterranean and into the temperate regions of Europe (Zohary and Hopf 2000). Chickpea, however, took a different pattern compared with the spread of the other Founder Crops and spread across the Mediterranean, but mainly to the south and south-east. Chickpea became a major crop in East Africa and India, mainly as a post-rainy season crop (Ladizinsky 1995) but not in the wheat-based temperate systems of Europe (Ladizinsky 1995; Kumar and Abbo 2001). All Founder Crops species except chickpea have retained their autumnal germination—summer maturation cycle, while across the Near East, traditionally, chickpea is a spring-sown crop (Kumar and Abbo 2001). It was suggested that this crop cycle change from autumn to spring sowing was driven by the extreme vulnerability of chickpea to Ascochyta blight during the rainy season (Abbo et al. 2003).

Ascochyta blight is a fungal disease caused by *Didymella rabiei* (anamorph: *Ascochyta rabiei*). It is one of the most important diseases of chickpea worldwide (Nene 1982; Nene and Reddy 1987; Akem 1999) affecting all above ground parts of the plants. Under environmental conditions that favour development of the pathogen, the disease is devastating. Crops are destroyed and yield losses reach 100% (Nene 1984; Akem 1999). Like many other pycnidial fungi, the pathogen spreads during the growing season mainly by rain-splash of pycnidiospores (Nene 1984; Fitt et al. 1989). Although temperature and wind influence disease

development and spread, rainfall is the environmental parameter governing Ascochyta blight epidemics and the disease develops whenever there are rains during the cropping season (Nene and Reddy 1987; Reddy et al. 1990; Diekmann 1992; Akem 1999).

Lentil and pea, two other grain legumes of Near Eastern origin, are also infected by Ascochyta blights. In lentil, the causal agent is *A. lentis* (Kaiser et al. 1997). In pea, the disease is incited by a complex of three pathogens: *A. pisi*, which causes leaf and pod spots; *A. pinodes*, the conidial state of *Mycosphaerella pinodes*, which causes blight; and *Phoma pinodella* (Syn. *Ascochyta pinodella*), which causes foot rot (Ali et al. 1994). Interestingly, Ascochyta blight did not preclude winter sowing of pea or lentil. One possible explanation is the difference in the influence of the disease on these crops. Whereas severe Ascochyta blight epidemics in chickpea are devastating, effects of the disease in lentil and pea are less conspicuous. Although substantial yield losses may occur in pea and lentil, complete destruction of the plants is uncommon even under severe epidemics (Gossen and Morrall 1983; Bayaa et al. 1992; Ali et al. 1994; Tivoli et al. 1996; Morrall 1997).

Is the difference between the aggressiveness/virulence of the chickpea Ascochyta pathogen and those of lentil and pea related to the different cropping practices? Study of the wild barley/powdery mildew system in Israel demonstrated that higher aggressiveness and wider virulence range are common in sites where the climatic conditions are unfavourable for the pathogen (Dinoor and Eshed 1987). Likewise, the summer cropping system of chickpea is less favourable to the Ascochyta pathogen than the winter cropping of lentil and pea or the autumn–winter development of wild *Cicer* to their respective Ascochyta pathogens. Could this be the reason for the extreme aggressiveness of Ascochyta in domesticated chickpea fields? Will lower aggressiveness be found in wild populations similar to the above? Contrary to the well-documented situation in the cereals and many of their pathogens (e.g., Dinoor 1974; Dinoor and Eshed 1984; Dinoor et al. 1991), hardly any information is available on the role of fungal pathogens in populations of wild relatives of Near Eastern legumes. Specifically, regarding chickpea, up to date, the only published report of *D. rabiei* from wild *Cicer* was from the perennial *C. montbretti* in Bulgaria (Kaiser et al. 1998).

Recently however, Frenkel et al. (2007) described the isolation of two Ascochyta pathogens from *C. judaicum*, an annual wild relative of domesticated chickpea native to Israel, Jordan and neighbouring countries. The pathogens, *D. rabiei* and *P. pinodella*, were identified morphologically and the DNA sequences of the rDNA intergenic regions were used to verify the morphological identification according to their similarity with published sequence information (Frenkel et al. 2007). The infectivity of the isolates obtained from the wild was verified by following Koch's postulates. *Didymella rabiei* isolates from wild *C. judaicum* were capable of infecting a number of annual *Cicer* species including domesticated chickpea, its wild progenitor *C. reticulatum*, and *C. bijugum* from Turkey. Disease severity caused by isolates from *C. judaicum* was greater on the wild hosts compared with the domesticated host. Similarly, using isolates originating from domesticated fields resulted in higher disease severity on domesticated cultivars compared with wild *C. judaicum* accessions (Frenkel et al. 2007). Although *P. pinodella* is not the focus of this review, it is interesting to note that this pathogen, which is one of the fungi that compose the Ascochyta complex of pea, also attacks *C. judaicum*. *Phoma pinodella* isolates from *C. judaicum* were able to infect both wild and domesticated peas (*Pisum sativum* and *P. fulvum*, respectively). In the studied ecosystems, wild chickpea grow side by side with wild pea species, and both are within meters from farmland where archaeological remains testify for millennia of cultivation (Frenkel et al. 2007). Such sympatric cropping (and patho-systems) may provide better understanding of the biology of the pathogens and their interaction with wild and domesticated hosts.

Important knowledge gaps

A number of questions emerge from the above description. What is the role of genetic diversity at resistance loci of the wild hosts and is it greater

compared with the cultigen? Do natural and agricultural ecosystems function as independent pathosystems? Specifically, are populations of *D. rabiei* infecting wild *Cicer* genetically distinct from populations infecting domesticated chickpea, and if so, is this differentiation related to differences in host specialization or to adaptation to different ecological conditions? Do isolates sampled from wild *Cicer* exhibit a similar level of aggressiveness and have different aggressiveness alleles compared with those sampled from domesticated chickpea? And last but not least, can we use gene diversity measures of the pathogen to infer about its origin and past and recent migration patterns?

Proposed framework for progress and bearing for resistance breeding

Clarifying the unresolved issues above, and answering the relevant research questions, require extensive multi disciplinary experimental work.

Comparative epidemiology

Modelling approaches are often used to elucidate the influence of environmental parameters on epidemic outbreaks. This was done in domesticated chickpea (e.g., Jhorar et al. 1997), but not in wild *Cicer* populations. Wild *Cicer* populations differ from domesticated plant communities in terms of their physical structure and genetic constitution. Therefore, disease prevalence, spread and development in time and space in the wild are likely to be different from those occurring in farmers' fields. Application of modelling approaches will enable quantification of the association between climatic parameters and disease development characteristics in wild populations. This in turn will point to the differences, if such occur, between the selection pressures operating on the pathogens and their hosts, in natural vs. man-made agro-systems.

Phenotypic and genetic characterization of hosts and pathogens

The different seasonality of wild vs. domesticated chickpea may suggest that pathogen populations parasitizing wild chickpea have different ecological requirements than those infecting domesticated crops. For example, the optimal temperature for spore germination, penetration, establishment and formation of pycnidia and pycnidiospores may differ between pathogen populations from wild and domesticated origins. Comparing the effect of temperature on the components of the disease cycle of isolates originating from wild and domesticated plants will clarify if such differences do exist and what is their magnitude. Similarly, effects of other environmental parameters (such as relative humidity, wetness duration, etc.) can be studied. Analyzing the segregation of the respective phenotype among cross progeny between wild and domesticated isolates will enable the study of the genetic control of the respective traits. Challenging wild *Cicer* accessions and domesticated chickpea with *D. rabiei* isolates from both hosts under controlled conditions may clarify the role of genetic resistance in natural *Cicer* populations. Comparing population structure of the pathogens isolated from wild and domesticated hosts using neutral DNA markers will allow estimation of gene flow among populations on different hosts and between geographic regions. Such analyses will determine if wild *Cicer* populations provide a significant source of inoculum for Ascochyta blight epidemics of domesticated chickpea.

Host–pathogen specialization in the *Cicer*/*D. rabiei* system is another unresolved issue. Some authors refrain from using the term 'race' for *D. rabiei* isolates thereby implying incomplete specialization of the fungus (Lichtenzveig et al. 2005), while others use a race classification of this pathogen (Santra et al. 2000). Several groups have reported significant cultivar-by-isolate interaction (Chen et al. 2003; Chongo et al. 2004; Phan et al. 2003; Cho et al. 2004). Another approach was to define pathotype groups in *D. rabiei* to describe shifts in the pathogen populations that caused breakdown of resistant cultivars (e.g., Reddy and Kebbabeh 1985; Udupa et al. 1998). This yet unresolved debate regarding host–pathogen specialization in the *Cicer*/*D. rabiei* system has important implications for resistance breeding and may benefit from re-evaluation of current breeding strategies as well as disease assessment

methodologies (e.g., use of parametric scales to evaluate disease severity). If host specialization of the pathogen is the rule, it implies that breeders will have to frequently recruit new alleles and maybe even new resistance genes to combat new emerging virulent pathogen genotypes. If, however, host specialization is not a major feature of the pathogen, it may be possible to use existing resistance sources for a longer period like the Israeli cv. Bulgarit (Lichtenzveig et al. 2005). As some collections of Ascochyta isolates are heavily biased towards isolates originating from cultivated fields (e.g., WJ Kaiser collection in Pullman WA, USA), assessment of host specialization among *D. rabiei* requires larger sampling of isolates from the wild and challenging a larger representative collection of wild and cultivated hosts with both domesticated and wild isolates.

Host–pathogen coevolution

Molecular study of Ascochyta pathogens isolated from a number of legume species (wild and domesticated) enabled the assessment of the phylogenetic relationships among the sampled group (Barve et al. 2003; Peever et al. 2006). Like the host plants that undergo speciation processes on an evolutionary time scale, the pathogens are adjusting themselves to the evolutionary trends of their hosts as expected from the intimate interactions that occur through most of the life cycle of the pathogen. The study of such co-evolutionary trajectories taken by different members of the legume family and their Ascochyta pathogens may enable the reconstruction of the evolution of the pathogens. For instance, phylogenetic analyses among Ascochyta taxa from different legume species may help answer the question whether the pathogens parasitizing any given legume taxon are monophyletic or polyphyletic, and if the evolution of the pathogens actually reflects the evolutionary history of their hosts.

Phylogenetic analyses using DNA markers may expose spatial and temporal patterns of population dynamics across large geographical scales (Stukenbrock et al. 2006). Indeed, using DNA markers and hierarchical analyses enabled Zaffarano et al. (2006) to excluded both the Near Eastern cradle of agriculture and the Ethiopian barley diversity centre as the origin of the barley scald pathogen *Rhynchosporium secalis*, due to higher gene diversity found in central Europe. Similarly, study of a world collection of *D. rabiei* enabled Peever et al. (2004) to draw both a recent and a historical picture of population structure of this pathogen in the USA Pacific Northwest. However, due to the extremely small number of isolates from wild chickpea it is impossible at the present time, to hypothesize on the geographical origin of *D. rabiei* infecting domesticated chickpea. Study of Ascochyta isolates with special emphasis on isolates from wild *Cicer* both within the Near Eastern cradle of agriculture as well as in areas outside the natural distribution of the hosts, may expose the historical trends of the spread of the pathogen, and may enable the detection of the centre of origin of current pathogens or sources of recent epidemic episodes (e.g., epidemic outbreak in Australia in the late 1990s). Such analysis will also enable the refutation or corroboration of the hypothesis of Abbo et al. (2003) regarding Bulgaria and perennial *C. montbretii* as a possible origin of *D. rabiei* infecting domesticated chickpea. Such information is important since areas of maximum gene diversity of the pathogen are also likely to be important sources of host resistance genes, both wild and domesticated. In addition, identification of migration patterns and direction of gene flow in the pathogen may help in devising better quarantine measures within as well as between continents for the benefit of farmers worldwide.

Genetic analysis of pathogenicity

The quantitative nature of the *Cicer/D. rabiei* interaction suggests polygenic control of resistance/aggressiveness. Evidence for quantitative resistance in the host was published (e.g., Santra et al. 2000; Tekeoglu et al. 2000; Lichtenzveig et al. 2002; Flandez-Galvez et al. 2003; Lichtenzveig et al. 2006) but we currently lack data concerning the genetic control of aggressiveness in the pathogen. Such information could be obtained from the genetic analysis of progeny derived from crosses between isolates with different aggressiveness phenotypes on wild and domesticated hosts. Application of quantitative

genetic tools, in conjunction with DNA markers (e.g., Lichtenzveig et al. 2002, 2006) and phenotypic assessment (e.g., above) will enable the determination of whether aggressiveness genes are genetically linked to loci governing ecological adaptation (e.g., temperature- or wetness-response loci). This may help in answering the question: what is the role of environmental determinants in the co-evolution of resistance/aggressiveness in the *Cicer/D. rabiei* pathosystem.

Concluding remarks

A combination of factors determines that host/pathogen co-evolution under domestication is likely to follow a different trajectory compared with the situation in natural ecosystems. Among the factors relevant to the *Cicer/D. rabiei* pathosystem are: plant density (dense vs. thin), the genetic structure of host populations (uniform vs. variable), seasonal profile (warmer and drier vs. colder and wetter) under cultivation and in natural ecosystems, respectively. Therefore, study of (domesticated) biased collections of fungal isolates and their interaction with domesticated cultivars is unlikely to expose the full spectrum of the host–pathogen interaction in the respective pathosystem (Harlan 1976). This, in turn, might limit our ability to develop effective management strategies or efficient breeding methodology (see above). To complement the partial picture obtained from the study of domesticated host–pathogen interactions, the above experimental approach is proposed. It is anticipated that recent initiatives taken by the present authors and other groups to study the ecology and the genetics of the respective legume sympatric pathosystems will provide plant breeders, agronomists and pathologists with better tools for more effective disease management.

References

Abbo, S., & Rubin, B. (2000). Transgenic crops: A cautionary tale. *Science, 287*, 1927–1928.

Abbo, S., Shtienberg, D., Lichtenzveig, J., Lev-Yadun, S., & Gopher, A. (2003). Chickpea, summer cropping, and a new model for pulse domestication in the ancient Near-East. *The Quarterly Review of Biology, 78*, 435–448.

Akem, C. (1999). Ascochyta blight of chickpea: Present status and future priorities. *International Journal of Pest Management, 45*, 131–137.

Ali, S. M., Sharma, B., & Ambrose, M. J. (1994). Current status and future strategy in breeding pea to improve resistance to biotic and abiotic stresses. *Euphytica, 73*, 115–126.

Barash I. (1960). The causal organisms of Ascochyta diseases on pea in Israel. M.Sc. Thesis. The Hebrew University of Jerusalem, Rehovot, Israel. 65 pp. (Hebrew, English summary).

Barve, M. P., Arie, T., Salimath, S. S., Muehlbauer, F. J., & Peever, T. L. (2003). Cloning and characterization of the mating type (*MAT*) locus from *Ascochyta rabiei* (teleomorph: *Didymella rabiei*) and a *MAT* phylogeny of legume-associated *Ascochyta* sp. *Fungal Genetics and Biology, 39*, 151–167.

Bayaa, B., Erskine, W., & Hamdi, A. (1992). Response of wild lentil to *Ascochyta fabae* f.sp. *lentis* from Syria. *Genetic Resources and Crop Evolution, 41*, 61–65.

Burdon, J. J. (1987). *Disease and plant population biology*. Cambridge, UK: Cambridge University Press.

Chen, W., Coyne, C., Peever, T. L., & Nuehlbauer, F. J. (2003). Characterization of chickpea differentials for *Ascochyta rabiei* using an improved virulence assay. *Phytopathology, 93*(Suppl.), S16 (Abstract).

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.

Chongo, G., Gossen, B. D., Buchwaldt, L., Adhikari, T., & Rimmer, S. R. (2004). Genetic diversity of *Ascochyta rabiei* in Canada. *Plant Disease, 88*, 4–10.

Diamond, J. (1997). *Guns, germs, and steel. The fates of human societies*. New York, USA: Norton WW and Company.

Diekmann, M. (1992). Use of climatic parameters to predict the global distribution of Ascochyta blight on chickpea. *Plant Disease, 76*, 409–412.

Dinoor, A. (1974). Role of wild and cultivated plants in the epidemiology of plant diseases in Israel. *Annual Review of Phytopathology, 12*, 413–436.

Dinoor, A., & Eshed, N. (1984). The role and importance of pathogens in natural plant communities. *Annual Review of Phytopathology, 22*, 443–466.

Dinoor, A., & Eshed, N. (1987). The analysis of host and pathogens populations in natural ecosystems. In: M. S. Wolfe & C. E. Caten (Eds.), *Populations of Plant Pathogens: Their Dynamics and Genetics* (pp. 75–88). Oxford, UK: Blackwell Scientific Publications.

Dinoor, A., Eshed, N., Ecker, R., Gerechter-Amitai, Z., Solel, Z., Manistersky, J., & Anikster, Y. (1991). Fungal diseases of wild tetraploid wheat in a natural stand in northern Israel. *Israel Journal of Botany, 40*, 481–500.

Fitt, B. D. L., McCartney, H. A., & Walklate, P. J. (1989). The role of rain in dispersal of pathogen inoculum. *Annual Review of Phytopathology, 27*, 241–270.

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.

Frenkel, O., Shtienberg, D., Abbo, S., & Sherman, A. (2007). The sympatric Ascochyta complex of wild *Cicer judaicum* and domesticated chickpea. *Plant Pathology* (in press).

Gossen, B. D., & Morrall, R. A. A. (1983). Effect of ascochyta blight on seed yield and quality of lentils. *Canadian Journal Plant Pathology, 5*, 168–173.

Harlan, J. R. (1976). Diseases as a factor in plant evolution. *Annual Review of Phytopathology, 14*, 35–51.

Harlan, J. R., & Zohary, D. (1966). Distribution of wild wheat and barley. *Science, 153*, 1074–1080.

Jhorar, O. P., Mathauda, S. S., Singh, G., Butler, D. R., & Mavi, H. S. (1997). Relationship between climatic variables and Ascochyta blight of chickpea in Punjab, India. *Agricultural and Forest Meteorology, 87*, 171–177.

Kaiser, W. J. (1997). Inter- and intranational spread of ascochyta pathogens of chickpea, faba bean and lentil. *Canadian Journal of Plant Pathology, 19*, 215–224.

Kaiser, W. J., Wang, B. C., & Rogers, J. D. (1997). *Ascochyta fabae* and *Ascochyta lentis*: Host specificity, teleomorphs (*Didymella*), hybrid analysis and taxonomic status. *Plant Disease, 81*, 809–816.

Kaiser, W. J., Hannan, R. M., & Muehlbauer, F. J. (1998). First report of Ascochyta blight of *Cicer montbretii*, a wild perennial chickpea in Bulgaria. *Plant Disease, 82*, 830.

Kumar, J., & Abbo, S. (2001). Genetics of flowering time in chickpea and its bearing on productivity in semi arid environments. *Advances in Agronomy, 72*, 107–138.

Ladizinsky, G. (1995). Chickpea. In: J. Smartt & N. W. Simmonds (Eds.), *Evolution of crop plants* (pp. 258–261). Harlow, UK: Longman Scientific & Technical.

Leppik, E. E. (1970). Gene centers of plants as sources of disease resistance. *Annual Review of Phytopathology, 8*, 323–344.

Lev-Yadun, S., Gopher, A., & Abbo, S. (2000). The cradle of agriculture. *Science, 288*, 1602–1603.

Lichtenzveig, J., Shtienberg, D., Zhang, H. B., Bonfil, D. J., & Abbo, S. (2002). Biometric analyses of the inheritance of resistance to *Didymella rabiei* in chickpea. *Phytopathology, 92*, 417–423.

Lichtenzveig, J., Gamliel, E., Frenkel, O., Michaelido, S., Abbo, S., Sherman, A., & Shtienberg, D. (2005). Distribution of mating types and diversity in virulence of *Didymella rabiei* in Israel. *European Journal of Plant Pathology, 113*, 15–24.

Lichtenzveig, J., Bonfil, D. J., Zhang, H. B., Shtienberg, D., Abbo, S. (2006). Mapping quantitative trait loci affecting time to flowering and response to Ascochyta blight in chickpea. *Theoretical and Applied Genetics, 113*, 1357–1369.

Morrall, R. A. A. (1997). Evolution of lentil diseases over 25 years in western Canada. *Canadian Journal of Plant Pathology, 19*, 197–207.

Nene, Y. L. (1982). A review of Ascochyta blight of chickpea. *Tropical Pest Management, 28*, 61–70.

Nene, Y. L. (1984). A review of Ascochyta blight of chickpea (*Cicer arientum* L.). In: M. C. Saxena & K. B. Singh (Eds.), *Ascochyta blight and winter sowing of chickpea* (pp. 17–33). The Hague, Netherlands: Martinus Nijhoff/Dr W Junk Publishers.

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). Wallingford, UK: CAB International.

Peever, T. L., Salimath, S. S., Su, G., Kaiser, W. J., & Muehlbauer, F. J. (2004). Historical and contemporary multilocus population structure of *Ascochyta* rabiei (teleomorph: *Didymella rabiei*) in the Pacific Northwest of the United States. *Molecular Ecology, 13*, 291–309.

Peever, T. L., Barve, M. P., Stone, L. J., & Kaiser, W. J. (2006). Evolutionary relationship among *Ascochyta* species infecting wild and cultivated hosts in the legume tribes Cicereae and Vicieae. *Mycologia* (in press).

Phan, H. T. T., Ford, R., & Taylor, P. W. J. (2003). Population structure of *Ascochyta rabiei* in Australia based on STMS fingerprints. *Fungal Diversity, 13*, 111–129.

Reddy, M. V., & Kabbabeh, S. (1985). Pathogenic variability in *Ascochyta rabiei* (Pass.) Lab. in Syria and Lebanon. *Phytopathologia mediterranea, 24*, 265–266.

Reddy, M. V., Nene, Y. L., Singh, G., & Bashir, M. (1990). Strategies for management of foliar diseases of chickpea. In: H. A. van Rheenen & M. C. Saxena (Eds.), *Chickpea in the winter* (pp. 117–127). Patancheru, India: ICRISAT.

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

Stukenbrock, E. H., Banke, S., & McDonald, B. (2006). Global migration patterns in the fungal wheat pathogen *Phaeosphaeria nodorum*. *Molecular Ecology, 15*, 2895–2904.

Tekeoglu, M., Santra, D. K., Kaiser, W. J., & Muehlbauer, F. J. (2000). Ascochyta blight resistance inheritance in three chickpea recombinant inbred line populations. *Crop Science, 40*, 1251–1256.

Tivoli, B., Béasse, C., Lemarchand, E., & Masson, E. (1996). Effect of ascochyta blight (*Mycosphaerella pinodes*) on yield components of single pea (*Pisum sativum*) plants under field conditions. *Annals of Applied Biology, 29*, 207–216.

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

Zaffarano, P. L., McDonald, B., Zala, M., & Linde, C. C. (2006). Global hierarchical diversity analysis suggests the fertile crescent is not the center of origin of the barley scald pathogen *Rhynchosporium secalis*. *Phytopathology, 96*, 941–950.

Zizumbo-Villarreal, D., Colunga-GarcíaMarín, P., Payró de la Cruz, E., Delgado-Valerio, P., Gepts, P. (2005).

Population structure and evolutionary dynamics of wild–weedy–domesticated complexes of common bean in a Mesoamerican region. *Crop Science, 45*, 1073–1083.

Zohary, M. (1973). *Geobotanical foundations of the middle east*. Stuttgart, Germany: Gustav Fischer Verlag.

Zohary, D., & Hopf, M. (2000). *Domestication of plants in the old world*. Oxford, UK: Clarendon Press.