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REVIEW PAPER

Role of host specificity in the speciation of *Ascochyta* pathogens of cool season food legumes

Tobin L. Peever

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Abstract *Ascochyta*/legume interactions are attractive systems for addressing evolutionary questions about the role of host specificity in fungal speciation because many wild and cultivated cool season food legumes are infected by *Ascochyta* spp. and most of these fungi have described teleomorphs (*Didymella* spp.) that can be induced in the laboratory. Recent multilocus phylogenetic analyses of a worldwide sample of *Ascochyta* fungi causing ascochyta blights of chickpea (*Cicer arietinum*), faba bean (*Vicia faba*), lentil (*Lens culinaris*), and pea (*Pisum sativum*) have revealed that fungi causing disease on each host formed a monophyletic group. Host inoculations of these fungi demonstrated that they were host-specific, causing disease only on the host species from which they were isolated. In contrast to the strict association between monophyletic group and host observed for pathogens of cultivated legumes, *Ascochyta* fungi causing disease on wild bigflower vetch (*Vicia grandiflora*) were polyphyletic. Genetic crosses between several pairs of closely related, host-specific, and phylogenetically distinct *Ascochyta* fungi were fully sexually compatible. Progeny from these crosses had normal cultural morphology and segregation of molecular markers indicating a lack of intrinsic, post-zygotic mating barriers between the parental taxa. However, when progeny from a cross between a faba bean-adapted isolate (*A. fabae*) and a pea-adapted isolate (*A. pisi*) were assessed for their pathogenicity to the parental hosts, almost all progeny were non-pathogenic to either faba bean or pea. These results suggest that although these fungi have retained the ability to mate and produce progeny with normal saprophytic fitness, progeny are severely compromised in parasitic fitness. The host specificity of these fungi, coupled with the inability of hybrid progeny to colonize and reproduce on a host, may constitute strong extrinsic, pre-zygotic and post-zygotic mating barriers in these fungi and promote the genetic isolation and speciation of host-specific taxa. A phylogeny of the host plants is also being developed, and with more extensive sampling of pathogens and hosts from sympatric populations in the centre of origin, the hypothesis of cospeciation of pathogens and hosts will be tested. The objectives of this review are: (1) to summarize recent phylogenetic, host specificity and speciation studies of *Ascochyta* fungi, and (2) to suggest how current and future research using these pathosystems may lead to a better understanding of the role of host specificity in the speciation of plant-pathogenic fungi and the cospeciation of pathogens and their hosts.

T. L. Peever (✉)
Department of Plant Pathology, Washington State University, Pullman, WA 99164-6430, USA
e-mail: tpeever@wsu.edu

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Biology of *Ascochyta* spp.

Species of the coelomycete genus *Ascochyta* infect a number of economically important cool season food legumes and the diseases they cause represent serious limitations to legume production worldwide. Well-known hosts include chickpea (*Cicer arietinum*), faba bean (*Vicia faba*), lentil (*Lens culinaris*), pea (*Pisum sativum*), vetches (*Vicia* spp.) and their wild relatives. These diseases are known as ascochyta blights and are characterized by tan-coloured lesions on all above-ground parts of the plant which contain concentric rings of black pycnidia exuding cirrhi of one or two-celled hyaline conidia (Nene and Reddy 1987). Conidia are dispersed short distances via rain-splash and are responsible for secondary disease cycles during the growing season of the crop (Nene and Reddy 1987; Kaiser 1992). Some species of *Ascochyta* also reproduce sexually and ascospores can be windborne and dispersed long distances by air (Trapero-Casas et al. 1996). Ascospores are typically unequally two-celled with a prominently constricted septum (Wilson and Kaiser 1995; Kaiser et al. 1997) and are considered important sources of primary inoculum in areas where both mating types occur (Trapero-Casas et al. 1996; Kaiser 1997a, b; Peever et al. 2004). Most *Ascochyta* spp. have a bipolar, heterothallic mating system (Barve et al. 2003; Cherif et al. 2006). *Ascochyta* spp. have been moved by human activity into most areas of the world where cool season food legumes are currently produced (Morrall and McKenzie 1974; Kaiser 1997a, b; Peever et al. 2004). Most of this movement has been due to the introduction of infected and/or infested seed imported for agronomic evaluation (Kaiser 1992; Peever et al. 2004). *Ascochyta* fungi have been demonstrated to be seedborne (Kaiser 1972; Morrall and McKenzie 1974; Maden et al. 1975) and have the potential to be transmitted from seed to seedling (Maden 1983; Dey and Singh 1994; Kimber et al. 2006). Cool season food legumes are native to south-eastern Turkey, Iran, Iraq, and Syria (Van Der Maesen 1987; Smartt 1990) and we hypothesize that *Ascochyta* spp. coevolved with their hosts in these areas.

Species of *Ascochyta*

Ascochyta fabae, A. pisi, A. lentis, *A. rabiei*, and *A. viciae-villosae* are pathogens of faba bean (*V. faba*), pea (*P. sativum*), lentil (*L. culinaris*), chickpea (*C. arietinum*) and hairy vetch (*Vicia villosa*), respectively (Nene and Reddy 1987; Nene et al. 1988; Kaiser 1997a, b; Mel'nik et al. 2000). Several *Ascochyta* anamorphs have been connected to *Didymella* teleomorphs (Jellis and Punithalingam 1991; Kaiser et al. 1997). The taxonomy of *Ascochyta* spp. is based first on morphological characters such as the shape and size of conidia, conidial septation, and second on host of isolation and molecular markers (Gossen et al. 1986; Kaiser et al. 1997; Fatehi and Bridge 1998). *Ascochyta rabiei*, the chickpea pathogen, grows approximately five times more slowly in culture and has darker colony morphology compared to *A. lentis*, *A. fabae*, and *A. pisi*. Genetic crosses made between *A. rabiei* and A. *fabae* and between *A. rabiei* and *A. lentis* failed to produce any pseudothecia while crosses between *A. fabae* and *A. lentis* produced pseudothecia (Kaiser et al. 1997). Results of these crosses predict that *A. fabae* and *A. lentis* are more closely related to each other than either is to *A. rabiei*. *Ascochyta fabae*, *A. lentis*, *A. viciae-villosae* and *A. pisi* are morphologically similar and have been historically difficult to separate using morphological criteria alone. Efforts to differentiate *A. fabae* and *A. lentis* provide an interesting case study in fungal species concepts. Gossen et al. (1986) demonstrated that *Ascochyta* spp. isolates from lentil and faba bean only caused disease on lentil and faba bean, respectively. However, these host-specific taxa could not be differentiated by statistical analyses of conidium length, proportion of septate conidia and cultural morphology (Gossen et al. 1986). These authors proposed that these two fungi be synonymized under *A. fabae* using the *formae speciales* designations *A. fabae* f.sp. *fabae* and *A. fabae* f.sp. *lentis* to denote their morphological similarity and host specificity. Crosses of these taxa were fertile and produced pseudothecia with viable ascospore progeny (Kaiser et al. 1997) but strong post-zygotic mating effects were observed which included abnormal numbers of ascospores in each ascus, variable ascospore size, and progeny isolates that grew abnormally in culture. In addition, all progeny isolates failed to infect either of their parental hosts. Kaiser et al. (1997) also scored these isolates for RAPD-PCR markers and showed that the fungi from each host each had distinct RAPD-PCR banding profiles and clustered separately in a UPGMA phenogram. The combination of host

specificity, strong genetic differentiation in molecular markers (i.e., lack of gene flow) and post-zygotic mating effects observed in this study were used to justify the elevation of *A. fabae* f.sp. *lentis* to *A. lentis* Vassilevsky (Kaiser et al. 1997), and represents a rare example of application of the biological species concept to plant-pathogenic fungi. Currently, we consider *A. rabiei*, *A. fabae* and *A. lentis* to be well-supported biological and/or morphological species.

Evolutionary relationships among *Ascochyta* spp.

Despite the economic importance of the cool food season legumes and the *Ascochyta* spp. that cause devastating losses of these plants, little is known about the evolutionary history of either the hosts or the pathogens. Peever et al. (2007) recently estimated phylogenies among the *Ascochyta* spp. pathogens of the cool season legumes using DNA sequence data from several regions of the genome including a glyceraldehyde-3-phosphate dehydrogenase gene (*G3PD*), a chitin synthase 1 gene (*CHS*) and translation elongation factor 1 alpha gene (*EF*). The analysis employed an extensive collection of *Ascochyta* spp. from cool season food legumes established by W. J. Kaiser, USDA-ARS and maintained at Washington State University. This collection contains isolates from chickpea, pea, lentil, hairy vetch and faba bean sampled on a worldwide scale. Currently, the collection is biased heavily towards fungi from cultivated legumes, but has been augmented in recent years with isolates sampled from wild legume species including some of the closest known relatives of cultivated crops. Isolates from wild legumes have been obtained during collecting trips to Armenia, the Republic of Georgia and Spain. Phylogenetic analyses of the combined *G3PD*, *CHS* and *EF* datasets using maximum likelihood methods revealed that *A. rabiei*, the pathogen of chickpea (*C. arietinum*), was distinct from the *Ascochyta* pathogens of pea, faba bean, wild vetches and lentil which were found in two differentiable but closely related clades (Fig. 1, Peever et al. 2007). The differentiation observed between *A. rabiei* and *A. lentis*/*A. fabae* in the combined phylogeny correlates well with the results of genetic crosses among these same taxa made previously (Kaiser et al. 1997). The combined phylogeny also revealed that isolates sampled from wild *Cicer* spp. (*C. montbretii* and *C. ervoides*) had sequences that were identical or nearly identical to isolates from cultivated chickpea (*C. arietinum*). *Cicer arietinum* is an annual species which is genetically distinct from the perennial species, *C. montbretii* and *C. ervoides* (Javadi and Yamaguchi 2004; Sudupak et al. 2004). The genetic similarity of fungi colonizing distantly related annual and perennial *Cicer* hosts suggests that the source of the ascochyta blight fungus for epidemics on cultivated chickpea may be wild, perennial chickpea relatives.

Two major clades were apparent in the combined phylogeny, one corresponding to isolates from cultivated lentil (*L. culinaris*), hairy vetch (*V. villosa*) and wild *Vicia* spp. (the *A. lentis*/*A. viciae-villosae* clade) and one corresponding to isolates from cultivated pea (*P. sativum*) and faba bean (*V. fabae*), wild pea (*P. elatius*) and wild *Vicia* spp. (the *A. fabae*/*A. pisi* clade) (Fig. 1). Isolates sampled from wild legume hosts displayed more sequence variation for all genomic regions compared to isolates from cultivated hosts (Fig. 1) consistent with the hypothesis that *Ascochyta* spp. pathogens of cultivated legumes represent a subset of the variation present in pathogen populations on wild hosts. More intensive sampling of *Ascochyta* spp. from sympatric legume hosts in the centre of origin will be required to definitively test this hypothesis. Isolates sampled from the cultivated hosts lentil, pea, faba bean, and chickpea were each monophyletic with strict correlation between phylogenetic clade and host of origin. In contrast, isolates from the wild hosts *V. villosa and V. lathyroides* formed a well-supported sub-clade within the *A. lentis* clade and isolates from *V. grandiflora* and *V. cordata* and from *V. grandiflora* and *V. sepium* also formed well-supported sub-clades within the *A. fabae*/*A. pisi* subclade (Fig. 1). Perhaps the most interesting result of the phylogenetic analysis was that isolates sampled from wild *V. grandiflora* were polyphyletic, distributed in three clades (Fig. 1). There are at least two hypotheses that may explain the polyphyly of the fungi sampled from this host. The first is that *V. grandiflora* may be colonized by different evolutionary lineages of *Ascochyta* pathogens. This would imply that the apparent tight correlation between pathogen clade and host of origin seen with isolates from cultivated hosts is the result of a founder event or strong selection by each

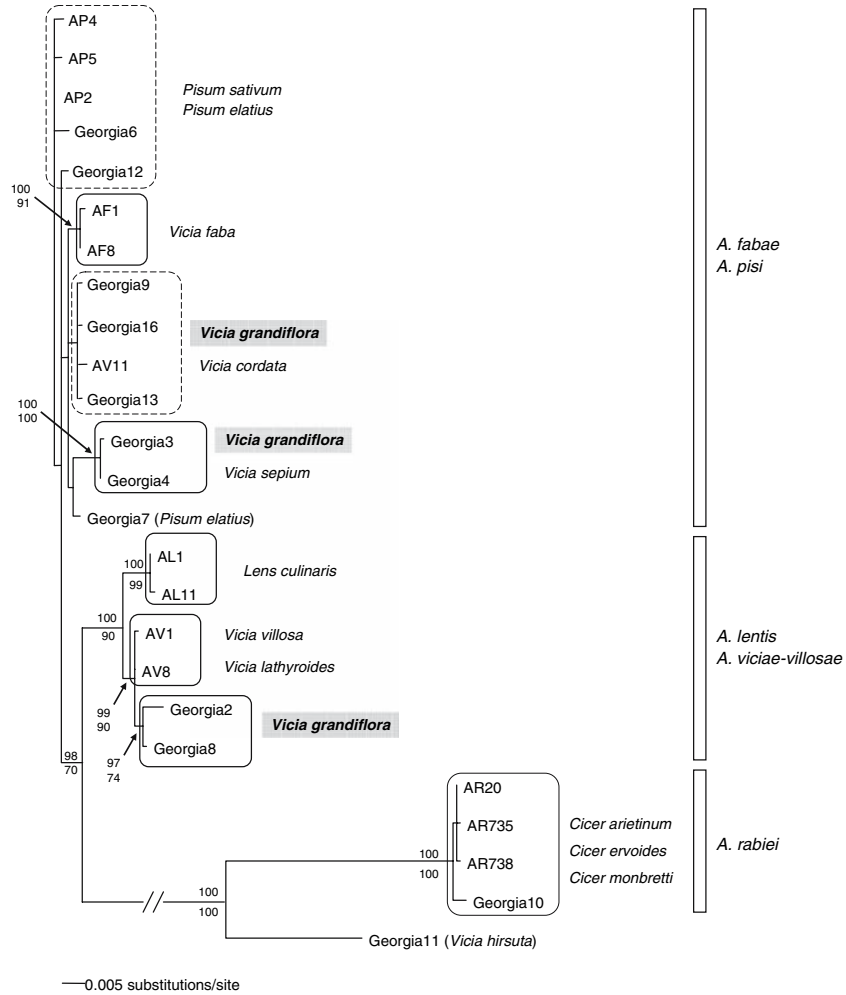

Fig. 1 Maximum likelihood phylogeny estimated from the combined chitin synthase (*CHS*), translation elongation factor alpha (*EF*) and glyceraldehyde-3-phosphate-dehydrogense (*G3PD*) datasets for *Ascochyta* and *Didymella* spp. sampled from various legume hosts. Upper numbers at major nodes indicate Bayesian posterior probabilities of sampling the node among 6,000 trees (600,000 generations of the MCMC chain) and lower numbers indicate percent ML bootstrap values from 1,000 bootstrapped datasets. Clades were inferred based on ML bootstrap values greater than or equal to 70% and posterior probabilities greater than or equal to 95%. Major clades are identified by open vertical bars and well-supported sub-clades by solid-line boxes. Clades with bootstrap values and posterior probabilities below the significance criteria are indicated by dashed-line boxes. Branch lengths are proportional to the inferred amount of evolutionary change and the scale represents .01 nucleotide substitutions per site. Host of isolation is indicated to the right of the taxon labels and isolates sampled from *Vicia grandiflora* are indicated in grey

cultivated host for a single monophyletic lineage of fungus and possibly coevolution of host and pathogen. The alternative hypothesis is that *Ascochyta* fungi causing disease on both wild and cultivated hosts have tight coevolutionary relationships with their hosts but the host taxa identified in this study are polyphyletic. The host plants sampled in our study were all identified morphologically in the field and it is possible that several distinct evolutionary lineages were classified as *V. grandiflora*. In order to distinguish between these two hypotheses, more detailed sampling in the centre of origin and more careful morphological analysis of the hosts needs to be performed as well as controlled inoculations of hosts and phylogenetic analyses of the hosts based on DNA sequence data.

Isolates from wild pea (*P. elatius*), the presumed ancestor of cultivated pea (Smartt 1990), clustered

with isolates from cultivated pea (*A. pisi*) in the *A. fabae*/*A. pisi* clade (Fig. 1). This is consistent with the hypothesis that *A. pisi* on cultivated pea evolved on *P. elatius* or other wild relatives, becoming a pathogen of pea during its domestication. Preliminary host inoculations have demonstrated that isolates of *A. pisi* sampled from cultivated pea are able to cause disease on both cultivated and wild pea but that isolates from wild pea are only able to cause disease on wild pea (T. Horton, M.I. Chilvers and T.L. Peever, unpublished). These data, although preliminary, may indicate that certain genotypes of the pathogen have a wider host range that allowed an expansion of host range during the domestication of pea. Crosses between isolates that are exclusively pathogenic on wild pea and isolates capable of inducing disease on both wild and cultivated pea may provide insight into the genetic control of host range and the mechanism responsible for this difference in host range.

Host specificity and speciation of *Ascochyta* spp.

Artificial inoculations in the greenhouse and in growth chambers have demonstrated that legume-associated *Ascochyta* fungi are host-specific (Kaiser 1973; Tripathi et al. 1987; Kaiser 1991; Kaiser et al. 1997; Khan et al. 1999; Hernandez-Bello et al. 2006). *Ascochyta fabae*, *A. pisi*, *A. rabiei*, *A. lentis* and *A. viciae-villosae* caused disease when inoculated onto faba bean, pea, chickpea, lentil and hairy vetch, respectively (Hernandez-Bello et al. 2006). The results of Hernandez-Bello et al. (2006) agree with previous inoculation studies where *A. rabiei* failed to cause disease on lentil, pea and vetch (Kaiser 1973; Tripathi et al. 1987; Kaiser 1991; Khan et al. 1999) and *A. fabae* and *A. lentis* could only cause disease on their respective hosts (Kaiser et al. 1997). The phylogenetic analyses demonstrated that *A. rabiei*, *A. pisi*, *A. lentis*, *A. fabae*, and *A. viciae-villosae* are each monophyletic (Fig. 1). These taxa are also host-specific (Hernandez-Bello et al. 2006). *Ascochyta pisi* is most closely related to *A. fabae* and *A. lentis* is most closely related to *A. viciae-villosae* (Fig. 1). Crosses made between these pairs of host-specific taxa were fertile and did not appear to have any of the genetic abnormalities observed in the crosses between *A. fabae* and *A. lentis* made previously by Kaiser et al. (1997). Interspecific hybridization of *A. pisi* × *A. fabae* and *A. viciae-villosae* × *A. lentis* was confirmed by the segregation of mating type and molecular markers. Segregation ratios of amplified fragment length polymorphism (AFLP) markers in these interspecific crosses were not significantly more distorted when compared to intraspecific crosses (Hernandez-Bello et al. 2006) demonstrating a lack of obvious intrinsic postzygotic mating defects. Both crosses produced viable ascospore progeny with normal cultural morphology and growth rates. However, artificial inoculations of progeny isolates from the *A. pisi* × *A. fabae* cross in the greenhouse and growth chamber resulted in very few progeny that were able to induce disease on either parental host. These data indicate that *A. fabae* and *A. pisi* are closely related phylogenetic species, can be experimentally crossed and that host specificity is likely to be polygenic. These data also suggest that fitness deficits suffered by the progeny of such a cross (i.e. the inability to cause disease and reproduce on a host plant) may be high and contribute a strong mating barrier. The results of the inoculation study with progeny from the *A. fabae* × *A. pisi* cross (Hernandez-Bello et al. 2006) were similar to those reported by Kaiser et al. (1997) for the much wider *A. fabae* × *A. lentis* cross. Mechanisms of speciation of fungi are poorly understood but host specificity may play an important role in facilitating the speciation of *Ascochyta* spp. and other host-specific, plant pathogenic fungi (Kohn 2005; Giraud et al. 2006). It is possible that host specialization of *Ascochyta* spp. acts as a prezygotic isolating mechanism as in other plant pathogens, including *Phytophthora* spp. (Goodwin and Fry 1994; Goodwin et al. 1999), formae speciales of *Blumeria graminis* (Hiura 1962; Hiura 1978) and *Puccinia graminis* (Johnson 1949). All of our observations, taken together, suggest that host specificity in *Ascochyta* may represent both a prezygotic and a postzygotic mating barrier and that these barriers have played important roles in the speciation of *Ascochyta* fungi. The evolution of host specificity may represent the initial step in the speciation of these fungi. In addition to uncovering the role of host specificity in fungal speciation, crosses between closely related pairs of *Ascochyta* taxa and inoculation of the progeny may allow determination of the genetics of species-level host specificity which is a largely unexplored area in plant pathology (Heath 1991).

Cospeciation of *Ascochyta* spp. and their legume hosts?

The host specificity of closely related *Ascochyta* fungi indicates that this character has likely played an important role in the speciation of these fungi and it is possible that coevolutionary interactions between pathogen and host may have resulted in cospeciation of pathogen and host (Thompson and Burdon 1992). In support of this hypothesis, the combined glyceraldehyde-3-phosphate dehydrogenase (*G3PD*), translation elongation factor 1 alpha (*EF*), and chitin synthase (*CHS*) phylogeny estimated among the *Ascochyta* spp. correlates well with a plastid *matK* phylogeny of the hosts (Fig. 2) (Steele and Wojciechowski 2003; Steele and Wojciechowski, unpublished). The differentiation seen between *Cicer* spp. (tribe Cicereae) and *Pisum*, *Vicia* and *Lens* spp. (tribe Vicieae) in the host *matK* phylogeny is mirrored by the pathogen phylogeny. Steele and Wojciechowski (2003) identified two subclades within the Vicieae including Clade 1 which contained *P. sativum* and Clade 2 which contained *L. culinaris* and *V. grandiflora*. Although there was not complete overlap in the hosts sampled for the Steele and Wojciechowski (2003) study and our study (Peever et al. 2007), there appears to be broad congruence between pathogen and host phylogenies with the Steele and Wojciechowski

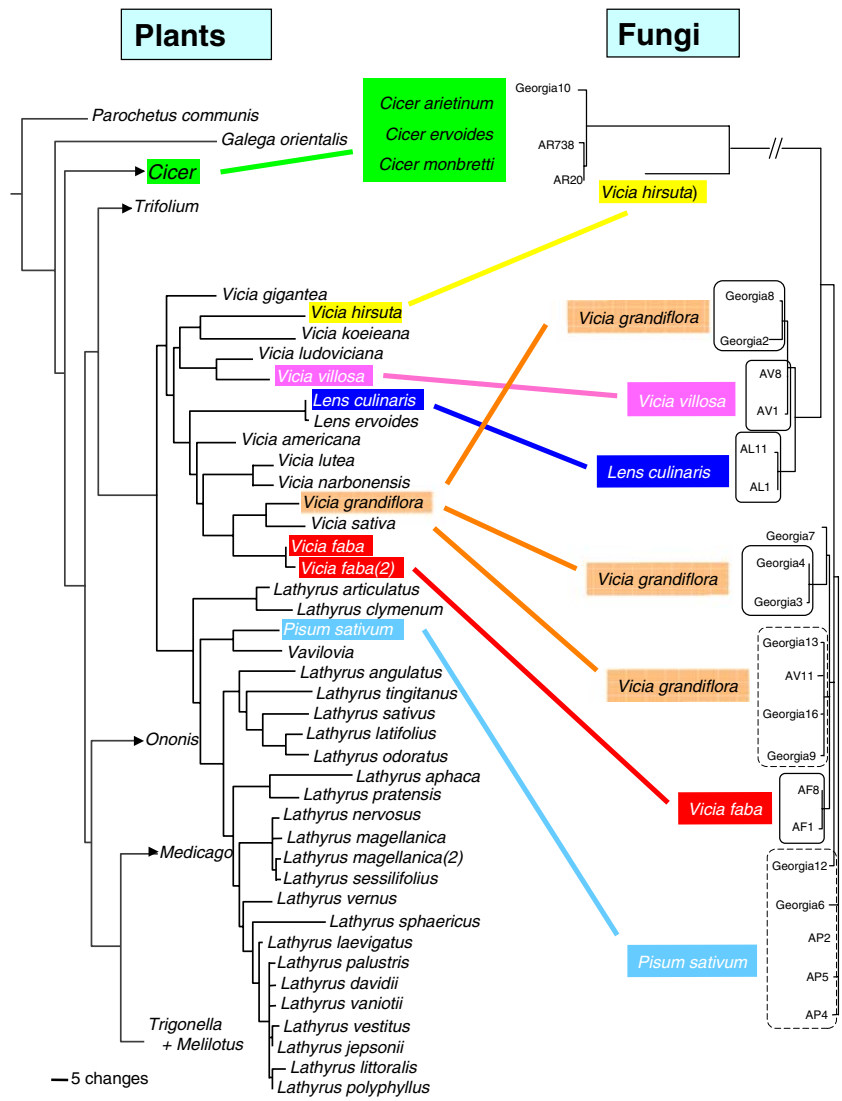

Fig. 2 Phylogeny of Vicioid clade (left) based on parsimony analysis of complete *mat*K gene aligned with combined *CHS*, *EF*, and *G3PD* phylogeny of *Ascochyta* fungi (right-simplified and inverted representation of Fig. 2). Plant tree shown is one representative of 1,000 equally parsimonious trees (1,865 steps), 1,524 characters included (168 excluded of 1,692 total), 485 of which are parsimony informative; CI = .5727, RI = .8444; tree rooted using *Glycyrrhiza* and *Callerya* (not shown). Bootstrap proportions shown near nodes for all nodes resolved in strict consensus tree; support for larger clade, the IRLC = 100%. Each host species is colour-coded and black lines connect legume hosts and fungi isolated from those same hosts. Orange lines illustrate polyphyly of *Ascochyta* fungi isolated from *Vicia grandiflora*

(2003) Clade 1 corresponding to the *A. pisi*/*A. fabae* clade in the combined analysis reported here and the Steele and Wojciechowski (2003) Clade 2 corresponding to the *A. lentis* clade reported here (Fig. 2). In order to rigorously test the cospeciation hypothesis, more extensive sampling of pathogen and host from sympatric host populations in the centre of origin are required. Cospeciation analyses will also require lower-level phylogenetic and phylogeographic analyses of the host using faster-evolving regions of the genome. Statistical tests of congruence between robust pathogen and host phylogenies will allow critical tests of cospeciation (Paterson and Banks 2001). Additional fast-evolving regions of the legume genome have been identified and are currently being used to resolve the evolutionary relationships within the Vicieae and Cicereae tribes (Steele and Wojciechowski 2003) and these regions will be useful for resolving phylogenetic relationships among closely related hosts. Sampling of *Ascochyta* fungi from sympatric hosts in these tribes in their centre of origin coupled with estimation of robust lower-level phylogenies for both hosts and pathogens will provide interesting insights into the coevolution of these pathosystems.

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