Phylogenetic determinants of potential host shifts in fungal pathogens

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Abstract

Understanding what determines the host range of pathogens and the potential for host shifts is of critical importance to controlling their introductions into new environments. The phylogeny of the hosts has been shown to be important: pathogens are more likely to be infectious on hosts closely related to their host-of-origin because of the similar host environments that is shared by descent. The importance of pathogen phylogenies for predicting host range has never been investigated, although a pathogen should also be able to exploit a new host that its close relative can infect. We performed cross-inoculations using a plant–fungus association and showed that both host and pathogen phylogenies were significant predictors of host range, with at least partly independent effects. Furthermore, we showed that some pathogens were better at infecting novel hosts. Our results should have implications in the context of biological invasions and emergences of new diseases due to globalization.

Introduction

Understanding what determines a pathogen’s host range has major implications, from predicting the emergence of new diseases to the biological control of pest species. The host range of a pathogen is influenced by a variety of factors, including extrinsic geographical or ecological barriers that prevent host–pathogen contact or subsequent infection (Jaenike, 1985) and intrinsic chemical, physiological or behavioural properties that determine whether a pathogen can persist on a given host when contact occurs. The combined effects of extrinsic and intrinsic factors determine the actual host range of a pathogen, i.e. those hosts on which the pathogen does persist in nature. An issue of great concern for biologists is what controls a pathogen’s potential host range, i.e. the range of hosts that a pathogen could infect barring extrinsic barriers. This question is of immediate importance because of increasing ‘biological globalization’ that brings into contact previously geographically isolated species (Desprez-Loustau et al., 2007). The past two decades have indeed seen the emergence of many new infectious diseases, mainly due to global travel, agricultural intensification or international transport of goods (Daszak et al., 2000; Desprez-Loustau et al., 2007). Also, research into biological control agents must take into account the potential host range of pathogens in order to prevent harm to nontarget hosts (Goddard et al., 2005).

Experimental cross-inoculations are a powerful means to assess the potential host ranges of pathogens (Poulin & Keeney, 2008). Such studies have been performed for a long time, and showed that some pathogens were highly specific, whereas others had a much broader potential host range than their actual one in nature (Zillig, 1921; Brisley, 1923; Bush et al., 2006; King & Cable, 2007). The underlying causes of such differences in the widths of potential vs. actual host ranges have not received much attention so far. Furthermore, although many studies have shown that pathogens can infect species outside the actual host range (Zillig, 1921; Brisley, 1923; Bush et al.,
2006; King & Cable, 2007), relatively few have looked for predictors for the success of new host–pathogen combinations. A consistently positive correlation has been demonstrated between the success of artificial inoculation and the phylogenetic proximity of the experimentally inoculated host and the pathogen’s host-of-origin (Moore & Gotelli, 1996; Perlman & Jaenike, 2003; Gilbert & Webb, 2007; King & Cable, 2007). Co-phylogenetic studies have shown that past host shifts or the expansion of a pathogen’s actual host range have involved closely related hosts in diverse groups (Futuyma et al., 1995; Reed & Hafner, 1997; Nishiguchi et al., 1998; Morehead & Feener, 2000; Hirose et al., 2005; Gilbert & Webb, 2007; Refrégier et al., 2008). Furthermore, theoretical studies have revealed that the congruence between host and pathogen phylogenies in some systems may have resulted from frequent host shifts preferentially between closely related hosts (Charleston & Robertson, 2002; de Vienne et al., 2007b).

Attempts have rarely been undertaken to quantify the phylogenetic signal for infection success among hosts and nonhost species. Moreover, the relationships among the host are considered exclusively and not among their associated phylogeny species. The rationale for examining the host phylogeny is straightforward: hosts sharing a recent common ancestor will more likely share an internal environment that the pathogen can utilize or similarly lack the specific defences that result in susceptibility. However, the pathogen phylogeny may also prove to be a predictor of successful host shifts because of similar reasoning: if closely related pathogens share physiological/nutritional requirements or similar mechanisms to overcome host defences, then they should be able to exploit the same kind of host. Thus, a particular pathogen may be able to cause disease on a host that currently harbours its close relative, independent of the relationship between the pathogen’s host-of-origin and the new host. In this way, the pathogen phylogeny may serve as an effective predictor of potential host range for the emergence of new diseases that is based upon knowledge of existing host–pathogen combinations.

Anther-smut disease of the Caryophyllaceae, caused by pathogens in the fungal genus Microbotryum, appears well suited for investigating the impact of host and pathogen phylogenies on potential host range. Microbotryum violaceum is a complex of sibling species, each with a narrow host range, typically one parasite per host (Lutz et al., 2005; Kemler et al., 2006; Le Gac et al., 2007). Co-phylogenetic analyses reveal that many host shifts have occurred in the history of the Microbotryum–Caryophyllaceae association, leading to incongruence between the pathogen and host phylogenies (Jackson, 2004; Refrégier et al., 2008).

The Microbotryum–Caryophyllaceae association is an ideal model to study the actual and potential host ranges via cross-inoculations. There is no vertical transmission of the disease. Spores are produced by the fungus in the anthers of infected hosts and are transmitted via pollinators onto healthy plants. The spores germinate and infect the meristems of the new plant (Antonovics, 2005). The fungus then resides within meristems in the infected plants (Audran & Batcho, 1982). No gene-for-gene relationships have been described in this pathosystem, differences in pathogenicity between strains or between species being only quantitative, usually measured as percentages of infected plants. This system has in fact long been used in cross-inoculation experiments by infection of plant meristems using a spore solution, demonstrating that the potential host range of Microbotryum species is larger than its actual host range (Zillig, 1921; Liro, 1924; Antonovics et al., 2002; Van Putten et al., 2003; Carlsson-Granér, 2006; Sloan et al., 2008). These studies, however, have not investigated the importance of phylogeny as a predictor of infection success.

In the current study, we performed a cross-inoculation experiment, using replicate strains belonging to seven different species of anther-smut pathogens and six host species to assess the potential host range in a phylogenetic context. We showed that: (1) the potential host ranges of Microbotryum species were larger than their actual host ranges; (2) there was variation in both the number of potential hosts among Microbotryum species and the degree of host susceptibility to various Microbotryum species; (3) the potential host range was influenced, at least partly independently, by both the host and the pathogen phylogenies.

Material and methods

Artificial inoculations and biological material

Cross-inoculations were performed on six host species (Saponaria officinalis, Silene latifolia, Silene dioica, Silene vulgaris, Silene paradoxa and Diontithus carthusianorum) with the Microbotryum species originating from these hosts. Silene vulgaris is infected by three distinct Microbotryum species in nature (Kemler et al., 2006; Le Gac et al., 2007), two of which were available for this study. A total of seven fungal species were therefore used, with three strains per species from different regions when possible (Appendix, Fig. 1). Some of these species have been given Latin names (Lutz et al., 2005; Kemler et al., 2006; Denchev, 2007a,b; Denchev et al., 2009), but others await formal description. The fungal species will be referred to as in Le Gac et al. (2007) and Refrégier et al. (2008), using an abbreviation of the pathogen and host names (Appendix). Figure 1 summarizes the treatments and presents the host and pathogen phylogenies. Fungal samples and seeds were collected from natural populations (Appendix), except for Sa. officinalis for which seeds were instead purchased from Jelitto Staudensamen GmbH (Schwarmstedt, Germany).

For each of the 21 fungal strains, teliospores from 10 infected anthers were suspended in 1200 μL of sterile tap
water by vortexing. Teliospore concentrations were standardised and 200000 spores in 1.5 mL of water were applied to 100 × 15 mm Petri dishes containing 1% water agar. Hosts were inoculated by incubating between 50 and 75 seeds in a Petri dish with the spores at room temperature for 10 days, and sterile tap water was added from time to time to prevent dehydration of the medium. Using this protocol, the spores germinate in water and infect the meristems of the plants while they grow. Seeds are not infected vertically. To test for possible contamination between treatments, seeds were incubated on two Petri dishes without fungal teliospores. Each of 30 seedlings per treatment (fungal strains × fungal species × plant species) was planted into 0.9 L potting soil and maintained under greenhouse conditions until flowering. Plant positions were randomized with regard to treatment. Plants with the fungus sporulating in the flowers were scored as diseased, and plants with symptomless flowers were scored as healthy. All infected flower buds were removed and scored plants were segregated to avoid secondary disease transmission. The experiment was terminated after 20 months (May 2008), as no further plants seemed to flower.

Genetic distance between species

Alignments of 329 bp in the internal transcribed spacer (ITS) region of the hosts’ nuclear rRNA genes and 485 bp for fungal ITS region were used for estimating the genetic distances. Primers used, PCR conditions and sequencing protocol were as in Refregier et al. (2008). The software MEGA v3.1 (Kumar et al., 2004) was used to calculate a pairwise distance matrix between all the species, following a Kimura-2-Parameters model (Kimura, 1980). The distance between pathogen species was calculated using the mean distance from all the strains of the same species. Where a plant species has more than one endemic pathogen species (i.e. on S. vulgaris) the distance between the pathogen used as inoculum and the pathogen originating from that particular plant species was chosen as the shortest of the two possibilities; this was to avoid pseudoreplication and because subsequent results showed that the effects of genetic distance vanished at long distances. Results are however robust when using either, or both, endemic pathogen species to estimate pathogen genetic distances to the pathogen used as inoculum. A neighbour-joining tree was constructed for the pathogen isolates used as inoculum to confirm that each pathogen belonged to the Microbotryum species described on its host-of-origin and that phylogenetic relationships among species (hosts and pathogens) were concordant with previous works (Refregier et al., 2008).

Statistical analyses

Logistic regressions and \( \chi^2 \) tests were performed using JMP Statistical Software v. 5.1.2 (SAS Institute, 2004). We used a logistic regression to test whether infection status was affected by pathogen genotype nested within pathogen species, pathogen species, plant species, genetic distance between the hosts (distance between the host-of-origin and the host on which inoculation was performed) and/or genetic distance between the pathogens (genetic distance between the inoculated pathogen and the pathogen originating from the inoculated plant species). The logistic regression considers each flowering plant as a row and its infection status as a nominal variable (either diseased or not) and tests the effect of the parameters cited above on the infection status. Full models were first fit including all factors and all interactions and were then simplified by sequential removal of the least significant highest-order interaction term, retaining only significant interactions and all main effects, even when nonsignificant.

The correlation between the host and pathogen genetic distances was tested using a Mantel test because the species are involved in multiple pairs, creating pseudoreplication in a correlation. The Mantel test was performed using R (R Development Core Team, 2008). We assessed whether the plant and fungal topologies were congruent with previous works (Refregier et al., 2008).
Maximum Agreement SubTree (MAST). A MAST is the largest possible tree compatible with two given trees (Finden & Gordon, 1985) and is obtained by removing the minimum number of leaves (i.e. terminal branches) in both trees in order to obtain perfect congruence. Significant congruence is inferred when congruence between the two trees is higher than that of random trees with the same leaf number.

Results

Flowering rate

Over the 20 months of the experiment, 45% of the plants flowered. The others either died or remained in vegetative state. The proportion of flowering plants was highly variable among host species, with 67.9%, 81.4% and 93.3% for D. carthusianorum, S. latifolia and S. vulgaris respectively. Far fewer plants of Sa. officinalis, S. dioica and S. paradoxa flowered, with only 11.4%, 13.0% and 2.8% respectively. We checked that flowering rates did not affect our results by testing whether flowering rate and disease rate. There was no significant correlation between flowering rate and disease rate. (logistic regressions, Wald

\[ \chi^2 = 0.85; \]

represent standard errors on the percentage of diseased plants.

Infection success

Control plants that were germinated on medium without fungal spores did not become diseased, thus there was no evidence of between-treatment contaminations. Of the 1646 inoculated plants that flowered, 90 were infected, representing 5.47%. Silene paradoxa was never infected and was therefore removed from the analyses. Its pathogen species (MvSp) was able to infect other host species and was retained in the analyses.

Infection success in each treatment is shown in Figs 1 and 2. Silene latifolia became diseased at a rate of 55.2% when inoculated with its own pathogen MvSl (Fig. 2a), which was significantly higher than when inoculated with any other pathogen species (logistic regression, Wald

\[ \chi^2 = 31.39, P < 0.00001, \]

even when considering only MvSl vs. MvSd (logistic regression, Wald

\[ \chi^2 = 19.76, P < 0.00001. \]

Silene dioica became diseased at a rate of 16.7% when inoculated with its own pathogen MvSd and of 17.6% when inoculated with MvSl, which was not significantly different (logistic regressions, Wald

\[ \chi^2 = 0.008, P = 1; \]

Fig. 2a). Similarly, there were no significant differences in the ability of the different Microbotryum species to infect either Sa. officinalis, S. vulgaris or D. carthusianorum. The plant S. vulgaris was not infected by its own pathogen species, but the overall rates of infection in this species were too low for this effect to be significant (see test above). Dianthus carthusianorum was infected by all seven Microbotryum species, at similarly low rates. This plant species thus appeared to be susceptible to a higher number of fungal species than the other plant species (\( \chi^2 = 14.016, P = 0.007; \) Fig. 2a).

Among the host–pathogen combinations used, the Microbotryum species appeared to vary in their ability to cause disease on multiple hosts (\( \chi^2 = 12.953, P = 0.05; \) Fig. 2a). (a) Plant’s point of view: for each of the four plant species, infection success of the plant species thus appeared to be susceptible to a higher number of fungal species than the other plant species (\( \chi^2 = 14.016, P = 0.007; \) Fig. 2a). (b) Pathogen’s point of view: for each of the seven Microbotryum species, infection success of the plant species. MvSl for instance could be infected by three Microbotryum species, MvSd having the highest infection success, (b) Pathogen’s point of view: for each of the seven Microbotryum species, infection success of the plant species. MvSl for instance managed to infect the four plant species, infection success by each parasite species. Silene latifolia; SL. Silene dioica; SD. Silene vulgaris; SO. Saponaria officinalis. Pathogen names are as in Le Gac et al. (2007) and Refregier et al. (2008). (a) Plant’s point of view: for each of the four plant species, infection success by each parasite species. Silene latifolia plants for instance could be infected by three Microbotryum species, MvSl having the highest infection success. (b) Pathogen’s point of view: for each of the seven Microbotryum species, infection success of the plant species. MvSl for instance managed to infect the four plant species but was better at infecting the S. latifolia plants. The error bars represent standard errors on the percentage of diseased plants.

Fig. 2 Infection success of each pathogen species on each plant species (percentage of infection in each treatment). Plant names are represented by capital letters. DC, Dianthus carthusianorum; SD, Silene dioica; SL, Silene latifolia; SV, Silene vulgaris; SO, Saponaria officinalis. Pathogen names are as in Le Gac et al. (2007) and Refregier et al. (2008).
Fig. 2b). The pathogens from *S. latifolia* and *S. dioica*, MvSl and MvSd, respectively, caused disease on four host species. In contrast, MvDc, MvSp and MvSv2 caused disease in a single plant species, *D. carthusianorum*, and MvSv1 and MvSoff successfully infected two plant species each, although each at very low rates.

When looking conjointly at the number of plant species that the pathogens can infect and the number of pathogen species to which the plant species are susceptible (Fig. 3), the different host–pathogen associations appear to exhibit contrasted patterns: in the susceptible (Fig. 3), the different host–pathogen associations appear to exhibit contrasted patterns: in the *S. latifolia/MvSl* and *S. dioica/MvSd* associations for instance, the *Microbotryum* species can infect several hosts and the hosts are quite resistant to most of the pathogens, whereas in the *D. carthusianorum/MvDc* association the host is susceptible to many pathogens and the *Microbotryum* species cannot infect other hosts. The relationship between the number of hosts infected by each pathogen and the number of pathogens on each host was negative, although the correlation was not significant ($r = -0.32$, $P = 0.54$).

### Effect of the genotype of the fungus on the success of infection

Logistic regressions gave no significant effects for fungal genotypes within their species and this component was thus removed from the following models (Table 1).

### Effect of the host genetic distance and the pathogen genetic distance when tested separately

We performed a logistic regression to investigate the effects of host species, pathogen species and genetic distance between hosts on infection status of the plants (Table 2). The effect of pathogen species was highly significant, confirming that some pathogen species caused more infections over all hosts than others. The effect of host species was also highly significant, confirming that some plant species were overall significantly more susceptible than others. The effect of genetic distance between hosts was also highly significant: pathogen species infected better the host species that were genetically closer to their host-of-origin than those that were more distantly related (Fig. 4a). When we removed the hosts-of-origin from the analysis (with a genetic distance of zero), the effect of the host genetic distance became only marginally significant ($P = 0.09$).

The similar analysis considering genetic distance between pathogens on infection status of the plants (Table 3) revealed the effects of host and pathogen species as above and also a significant effect of the genetic distance between pathogens: pathogen species infected better those host species that are naturally infected by genetically close pathogens than those that are naturally infected by distant pathogens (Fig. 4b). When the pathogens-of-origin were removed from the analysis (with a genetic distance of zero), the effect of the pathogen genetic distance remained significant ($P = 0.04$).

### Effects of host genetic distance and pathogen genetic distance when tested conjointly

The genetic distances between hosts and the genetic distances between pathogens were not significantly correlated (Mantel test, $z = 0.065$, $P = 0.08$), and the host and parasite phylogenies (Fig. 1) were not more similar than expected by chance ($P = 0.11$), in agreement with previous studies (Refrégier et al., 2008). This justifies our testing of the effects of these genetic distances on infection success conjointly. We performed a logistic

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**Table 1** Minimal model for the effect of pathogen species and pathogen genotype nested within pathogen species on the infection status of plants.

<table>
<thead>
<tr>
<th>Source</th>
<th>d.f.</th>
<th>Wald $\chi^2$-value</th>
<th>$P$-value</th>
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<tr>
<td>Pathogen species</td>
<td>6</td>
<td>72.00</td>
<td>&lt; 0.00001</td>
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<td>Pathogen genotype nested within pathogen species</td>
<td>14</td>
<td>6.19</td>
<td>0.9615</td>
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</table>

Logistic regression, $R^2 = 0.2230$, $P < 0.0001$.

**Table 2** Minimal model for the effect of pathogen species, plant species and genetic distance between hosts on the infection status of plants.

<table>
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<td>Plant species</td>
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</tr>
<tr>
<td>Genetic distance between hosts</td>
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<td>8.56</td>
<td>0.0035</td>
</tr>
</tbody>
</table>

Logistic regression, $R^2 = 0.3518$, $P < 0.0001$.

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**Fig. 3** For each host–pathogen association, number of pathogens able to infect the plant species as a function of the number of hosts infected by the pathogen.
When we removed the genetic distances of zero from the analyses, the effect of the pathogen genetic distance remained significant \((P = 0.01)\) but not that of the host genetic distance.

The effect of the interaction between these two genetic distances on infection status was significant (Table 4). The increasing genetic distance between pathogens indeed had a negative effect on the proportion of infected plants only for the class of host distances that were small but not for the class of host distances that were large (Fig. 4a). Reciprocally, the genetic distance between hosts appeared to have an effect on the proportion of infected plants only for the class of short pathogen distances but not for the class of large pathogen distances (Fig. 4b).

Because MvSv1 and MvSv2 infected few plants and *Sa. officinalis* had a low rate of overall infection, we also performed the analyses removing these fungal and plant species. The results remain similar, with both the host and genetic distances significantly impacting infection success.

### Discussion

#### Local adaptation vs. host specificity in *Microbotryum*

We found no significant effect of fungal genotype nested within fungal species on the ability to infect different host species. Within-species variation in infection ability and adaptation is known to occur in *Microbotryum* (Kaltz et al., 1999), but they produce only weak effects that are well below the strong differences that we see here for inter-species cross-inoculations. The difference in percentages of infection by fungal strains from allopatric vs. sympatric plant populations was only 32% vs. 40% (Kaltz et al., 1999). It is therefore likely that intraspecific variation in infection success on the native host plant has different underlying mechanisms and therefore does not affect the pattern of interspecific adaptation.

#### Potential host ranges larger than actual host ranges in *Microbotryum*

We found that most of the *Microbotryum* species used in this experiment were able to infect hosts that were
different from their host-of-origin. Similar results have been obtained in other cross-inoculation experiments in this system (Zillig, 1921; Liro, 1924; Antonovics et al., 2002; Van Putten et al., 2003; Carlsson-Granér, 2006; Sloan et al., 2008). Such a potential host range that is larger than the actual host range probably facilitates host shifts by Microbotryum, which have indeed been shown to be frequent by cophylogenetic analyses (Jackson, 2004; Refrégier et al., 2008) and by reports of incipient host shifts (Antonovics et al., 2002; Lopez-Villavicencio et al., 2005).

**Host specificity and host’s specific susceptibility**

Our study revealed a high degree of variation in host specificity among Microbotryum species. The species parasitizing *S. latifolia* (MvSl) and the species parasitizing *S. dioica* (MvSd) were able to infect all the four host species. MvSl seemed even better at causing disease in *S. dioica* than the Microbotryum species endemic to that host, as was also found previously (Van Putten et al., 2003). A previous study also found that MvSl was the only one of four Microbotryum species able to cause disease on multiple hosts (Sloan et al., 2008). In contrast, MvDc was found to be very specific, infecting only its host-of-origin. Pathogen-specific susceptibility was also variable among hosts. *Silene latifolia* and *S. dioica* were only parasitized by one or two pathogen species different from their usual one, whereas all seven pathogen species used in the experiment caused disease on *D. carthusianorum* at approximately equal rates.

When looking jointly at the host specificity and the degree of pathogen-specific susceptibility of host–pathogen pairs, a striking pattern emerges. The fungal species MvDc is strictly host specific and *D. carthusianorum* can be infected by all the tested fungal species. This could be interpreted as a slow coevolution in this association, host and pathogens evolving few sophisticated weapons and defences, which will render them largely inefficient against novel species combinations. In contrast, in the *S. latifolia*/MvSl and *S. dioica*/MvSd associations the plants have a high degree of specific susceptibility to the pathogen and the pathogens are able to infect many other host species. This could be interpreted as the result of a rapid arms race, where the host and pathogen regularly evolve new weapons and new defence mechanisms, that would make a ‘super pathogen’ able to infect many hosts and a host able to defend against many foreign pathogens. This concept of super-pathogen arising from the hot spot of coevolution has been invoked at the within-species level (Thrall & Burdon, 2002), i.e. in terms of local adaptation in a geographical context, and we suggest here that it could also be relevant at the interspecific level in terms of potential host range and without any geographical implication. Further investigations with more host–pathogen pairs are however required to be conclusive given the low infection and flowering rates obtained here in some species.

Our conclusions here should be robust to the low flowering rates of some plant species. In particular, the plants *D. carthusianorum*, *S. vulgaris* and *S. latifolia* were among the most contrasted cases here and they had the highest flowering rates. The dramatic difference in the host’s specific susceptibility between *D. carthusianorum* compared with *S. vulgaris* or *S. latifolia* cannot be due here to differences in flowering rates. The differences in the ability of the fungal species to infect different plant species cannot be due either to flowering rates because all treatments had similar flowering rates for a given plant species. Only the low apparent specific susceptibility of the hosts *S. dioica* and *S. officinalis* could potentially have revealed that different flowering rates had been higher.

**Effect of genetic distances between hosts and pathogens in the infection success**

We showed that the infection success of a novel host was negatively correlated with genetic distance between that inoculated host and the pathogen’s host-of-origin. This result is in accordance with studies on other organisms (Futuyma et al., 1995; Reed & Hafner, 1997; Nishiguchi et al., 1998; Morehead & Feener, 2000; Gilbert & Webb, 2007; Refrégier et al., 2008). We also showed that the infection success on new hosts was negatively correlated with genetic distance between pathogen used as inoculum and the pathogen found naturally on the target host, and that this effect was, at least partly, independent of the host phylogeny effect. This means that a pathogen can be predicted to cause disease more easily on a new host if that host is usually infected by a close relative of the pathogen. The effect even appeared stronger than that of the host genetic distance. As far as we know, this effect has not been tested previously. The interaction between the effects of host and pathogen genetic distances showed that the effects hold only for relatively short genetic distances, and was lost at farther distances. This seems intuitive: although a pathogen should be able to infect better a new host that shares more similar resistance mechanisms by descent with the pathogen’s host-of-origin host; this phylogenetic inertia is not expected to last across too many speciation events.

Here again, our main conclusions should be robust to the low flowering rates of some plant species and low infection ability of some fungal species. Indeed, we performed logistic regressions that take into account effective sizes. Such tests use as input a table with one line per plant, their infection status and all other recorded parameters. Furthermore, we performed the tests removing the plant species and fungal species that could have introduced a bias and the results regarding the effects of the genetic distances were similar. Given the low flowering rates however, our conclusions
Implications of this study for biological invasions

Our study has implications in the frame of new emerging diseases and biological pest control, for predicting the outcome of introductions, deliberate or not, of pathogens into new environments. The increasing biological globalization brings into contact previously geographically isolated hosts and pathogens (Desprez-Loustau et al., 2007) giving opportunity for new host-shifts and invasions. Dramatic epidemics have been induced by invasive fungal pathogens, such as Cryphonectria parasitica that eliminated the dominant Chestnut forests throughout eastern North America or Phytophthora infestans that caused the Irish Potato Famine (Anderson et al., 2004). Similarly, releases of fungal pathogens for biological control of exotic species (weeds for instance) brings into contact new hosts and pathogens (Barton, 2004). After release, a biocontrol agent is expected to become established, replicate, spread and become a permanent part of its environment without being a threat for the new environment (Barton, 2004). Our study shows that for predicting the outcome of introductions and for imposing quarantine against undesired pathogen species, one need pay attention to the host species present in the new environment and their phylogenetic relationship with the host-of-origin of the focal pathogen. Moreover, it is important to recognize that a pathogen will be a greater potential threat to a new environment if there are closely related pathogen species already present.

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References


Appendix: Microbotryum strains used in this study

Species names are indicated as in Le Gac et al. (2007) and Refrégier et al. (2008). Latin names as in Lutz et al. (2005), Kemler et al. (2006), Denchev (2007a,b), Denchev et al. (2009), places and dates of collection.

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