Emerging diseases are a growing worldwide problem accompanying global environmental changes. There is tremendous interest in identifying the factors controlling the appearance and spread of these diseases. Here, we discuss emerging fungal plant diseases, and argue that they often result from host shift speciation (a particular case of ecological speciation). We consider the factors controlling local adaptation and ecological speciation, and show that certain life-history traits of many fungal plant pathogens are conducive for rapid ecological speciation, thus favoring the emergence of novel pathogen species adapted to new hosts. We argue that placing the problem of emerging fungal diseases of plants within the context of ecological speciation can significantly improve our understanding of the biological mechanisms governing the emergence of such diseases.

The problem of emerging diseases in the context of ecological speciation

During the last century, human activities strongly contributed to changes in the environment and ecosystems on a global scale, breaking down many natural barriers to dispersal and causing an unprecedented redistribution of organisms [1]. There is growing evidence that these global changes play a key part in the emergence of infectious diseases in humans [2], wildlife [3], domestic animals [4], and plants [5].

Diseases are qualified as ‘emergent’ if they have recently become a cause for concern due to an increase in virulence, infection of a novel host, and/or occurrence in a new area. Pathogenic fungi in particular have been responsible for emerging diseases in plants and animals (and even in humans). Consequently, there is a greatly increased interest in identifying the factors driving the emergence of new fungal diseases [5–7].

Here, we focus on emerging fungal diseases in plants because of their importance for humans and because of the abundance of documented cases. We discuss the importance of novel fungal diseases of plants in agricultural production and for natural ecosystems, and we argue that host shift speciation appears to be a major route for their emergence. Host shift speciation is the formation of a novel species adapted to a novel host, and is a particular case of ecological speciation [8,9]. We argue that the problem of emerging fungal diseases is therefore best understood in the context of ecological speciation. There is a well-established body of mathematical theory dealing with the population dynamics of infectious diseases [10,11]. However, in this theoretical framework, adaptation and divergence among pathogens are largely neglected despite recognition that infection of a novel host can create strong selective pressures and raise the risk of devastating emerging diseases.

We argue that understanding the emergence of fungal diseases requires integration with established theories of ecological speciation, while taking into account the life-history characteristics particular to pathogens. We review ecological speciation. There is a well-established body of mathematical theory dealing with the population dynamics of infectious diseases [10,11]. However, in this theoretical framework, adaptation and divergence among pathogens are largely neglected despite recognition that infection of a novel host can create strong selective pressures and raise the risk of devastating emerging diseases.

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the factors that usually limit the potential for adaptation and speciation, and show that some life-history features of fungal pathogens can be conducive to rapid ecological speciation by host shifts. Recognizing the link between emerging diseases and ecological speciation should lead to more relevant theoretical models that incorporate the features of fungal pathogens, as well as novel approaches to studying emerging pathogens. Taken together, these efforts can yield important advances in our understanding of emerging fungal plant diseases, and have practical applications for designing more efficient and sustainable control programs.

**Importance of emerging plant fungal diseases**

Fungi (*sensu lato*, i.e. including Oomycetes) are responsible for ∼30% of emerging diseases in plants [5]. Epidemics caused by invasive pathogens have frequently been reported to alter natural ecosystems [5,6]. Well-documented examples include chestnut blight fungus (*Cryptonectria parasitica*), the spread of which eliminated nearly 100% of the native chestnut trees throughout eastern American forests during the twentieth century; and the Dutch elm disease fungus (*Ophiostoma ulmi*), which led to the destruction of American elms. A more recent example is *Phytophthora cinnamomi*, which currently threatens native forests throughout Australia [5,6]. Such devastating diseases affect not only the host plants, but also the whole associated fauna, including the insects, birds and mammals that are dependent upon them.

Our primary food production is also at risk due to emerging crop diseases [12]. The most dramatic example is the epidemic caused by *Phytophthora infestans* on cultivated potato in Ireland in the 1840s [13]. The infamous ‘Irish potato famine’ led to mass emigration and >1 million deaths from starvation or famine-related diseases. More recent examples include the severe blast disease of wheat, first noted in 1970, which spread globally to most wheat-growing regions in the twentieth century, and the fungus *Magnaporthe oryzae* that caused rice blast in China around the year 1970. The fungus *Sclerotinia sclerotiorum* causes an important and widespread disease of many agricultural crops, including *S. scabies* of carrots. These examples show how emerging fungal diseases can lead to significant economic losses and human suffering.

**Emergence of plant fungal diseases via host shift speciation**

Given their importance, there has been an increasing focus on identifying the factors that drive the emergence of new fungal diseases [5–7]. Emerging diseases can result from a sudden increase in virulence and/or expansion of the geographic range of a previously unnoticed pathogen [16], but infection of a novel host is the most frequent cause of fungal emerging diseases [7]. Infection of a novel host can result from spillover, host range expansion, or host shift. Much interest has been devoted to the extrinsic factors that promote contact between a pathogen and a novel host, such as climate change and worldwide trade [5,6]. Less attention has been paid to the intrinsic genetic changes in the pathogen needed for successful infection of a novel host in cases of host range expansion and host shift. Although the pathogen can be pre-adapted for infection and transmission onto a new type of host, most often successful disease emergence requires the pathogen to adapt, as witnessed by the usual failure of experimental inoculations in novel host–pathogen combinations [17]. Horizontal gene transfer and inter-specific hybridization have been invoked to explain how pathogens might achieve an enhanced adaptive potential that allows the emergence of new fungal diseases on novel hosts [7], but such adaptation is usually the result of selection among existing genetic variants or novel mutations within a fungal population [7,18]. Existing evolutionary theory tells us that adaptation to a new host will be most efficient if the flow of ancestral genes into the population adapting to a new host has ceased completely or is significantly reduced [19]. This makes the evolution of reproductive isolation (and speciation) important for understanding many emergent diseases.

The focus of this contribution is the mechanism of the emergence of fungal disease by host shift speciation. There are many examples of formation of sibling pathogen species on different hosts, and these radiations most often involved hosts shifts [20]. An example is the anther smut fungus, constituting a complex of sibling species sterilizing Caryophyllaceae plants [21]. Comparisons of host and pathogen phylogenies have shown that host shift speciation was frequent in the history of this association [22]. In fact, several incipient host shifts have been detected in natural populations of anther smut fungi. In these cases, populations that already appeared differentiated while still sub-optimally adapted to their novel host plants were found to be sympatric with their original hosts [23,24].

Host shifts have also been involved in cases of recent emerging diseases due to introductions of fungal pathogens into new continents [25,26]. Examples include the introduction of *C. parasitica* and *Phytophthora cinnamomi* in the USA, and devastation of the American chestnut (*Castanea dentata*), the most probable source of which was Japanese chestnut trees (C. crenata) that were imported and sold throughout the country [27,28]. These diseases are very recent, having occurred within the last century. The pathogens might thus have had no time to differentiate from their population of origin (although this has not been investigated in many cases). Differentiation has been found when looked for, as in the case of differentiation in *C. parasitica* between North America and Europe [27].

Other historical cases of disease emergence, associated with plant domestication, are also consistent with ecological speciation by host shifts. Well-documented cases are provided by the wheat fungal pathogen *Mycosphaerella graminicola* that originated in the Fertile Crescent at the time of wheat domestication [29], and the fungus *Rhynchosporium secalis*, causing a disease called ‘scald’ on rye, barley and other grasses [30].

The plethora of cases of closely related (but well-recognized) species of fungal pathogens infecting different host species after host shift speciation leads to the question of whether some specificities of parasites render them particularly prone to adaptation to new niches and so to ecologial speciation.

**Which factors usually restrict the possibility of speciation?**

The existing theory of ecological speciation [9,19,31–33] can be used to better understand factors constraining or
promoting the adaptation of pathogens to a new host. According to this theory, alleles providing an advantage on a new host need to greatly increase in frequencies in the local population on that new host. This increase is accomplished by strong selection for local adaptation. The theory also tells us that locally advantageous combinations of alleles need to be protected from being ‘diluted’ by ancestral alleles brought by immigrants. Such a dilution is expected if mating is random and immigration is recurrent. Therefore, protection of locally adapted allele combinations is required through the evolution of assortative mating (which can be achieved by mate choice, or habitat choice if mating occurs within habitats) or by strongly reduced viability of immigrants [34,35]. The evolution of assortative mating and speciation can be prevented by several factors, including a lack of genetic variation, immigration of ancestral genes, or the costs of being choosy in the selection of mate and/or habitat. Moreover, recombination and segregation will work against the establishment of locally advantageous allele and trait combinations by continuously destroying them. Therefore the overall success of ecological speciation in the presence of gene flow is dependent upon a delicate balance of several factors, as discussed above.

Applying the theory described above to fungi suggests that some characteristics of fungal pathogens can act to

Box 1. Lifecycle and the possibility of ecological speciation by host shift

Whether a plant pathogen mates within or outside its host has important consequences for the level of gene flow between populations adapted to different hosts. Consider two populations of an obligate biotroph fungal pathogen adapted to two different hosts. Assume there exists a single diallelic haploid locus involved in a gene-for-gene relationship, with allele A\text{1} preventing infection of host 2 (because it codes for an effector recognized by the plant and inducing a defence reaction), and allele A\text{2} preventing infection of host 1. Consider also a neutral locus with alleles B\text{1} and B\text{2}. Figures I and II depict different types of barriers that could act to restrict neutral gene flow between the pathogen populations adapted to the alternative hosts, i.e. host choice, host adaptation, geographic barriers between the two hosts, mate choice (i.e. assortative mating between the host races), and post-zygotic barriers. The arrows show potential gene flow. The red crosses indicate the steps in the lifecycle where the barriers to gene flow act to prevent gene exchange at the neutral locus. The figures illustrate that, for obligate biotrophs mating within their hosts, host adaptation alone can be a barrier to gene flow (even at neutral loci) because only individuals able to grow on the same host can eventually mate. In contrast, host adaptation cannot substantially reduce gene flow at neutral loci if mating occurs outside the host. A lifecycle of a pathogen mating outside of its host is represented in Figure I. Geographic barriers, mate choice, and post-zygotic barriers can prevent gene flow at a neutral B locus (B\text{1} and B\text{2} alleles). Host choice and host adaptation can decrease the frequencies of allele A\text{1} on host 2 and allele A\text{2} on host 1, but cannot prevent gene flow at the neutral locus B. This is a typical lifecycle of some basidiomycete fungal pathogens such as rusts and smuts. A lifecycle of an obligate biotroph mating within its host is represented in Figure II. Host choice and host adaptation can prevent gene flow at the host choice or specialization loci (A\text{1} and A\text{2} alleles), but also at the neutral locus (B\text{1} and B\text{2}; alleles), as can geographic barriers, mate choice, and post-zygotic barriers. Host adaptation can therefore pleiotropically cause specialization and reproductive isolation if mating occurs within hosts, which is not the case if mating occurs outside the host (Figure I). This is a typical lifecycle of ascomycete plant pathogens (Box 3).
constrain the possibility of adaptation and divergence. For instance, fungi lack active means of dispersal, spores are usually air-borne, and the mycelium is also passively dispersed. This means they cannot choose their most suitable hosts, a factor that should produce a high genetic load. Fungi cannot move toward their most suitable mates, which should increase the cost of waiting for a mate. Factors therefore exist that tend to oppose ecological speciation in fungi, but there are other, more powerful, mechanisms that strongly promote it.

Features of fungal pathogens of plants promoting ecological speciation
Several features of life-history traits in fungal pathogens are conducive to ecological speciation by reducing the constraints that usually impair speciation. We detail these features below and examine their consequences for the possibility of ecological speciation.

Very large numbers of spores: population persistence and large mutational input
Pathogenic fungi can produce thousands of spores per lesion per day [36,37], and multiple asexual cycles on the same individual plant can yield hundreds of separate infections. This means that billions of spores can be released from a given plant during an infection by a single fungal genotype. Such large numbers of spores can allow the population to persist on a new host even if selection against allele combinations adapted to infection of the ancestral host is extremely strong and the initial degree of adaptation to a new host is very low. Moreover, such large numbers of spores allow the rapid and recurrent creation of genetic variation by mutations. Empirical examples support this logic. For instance, consider evolution of virulent strains able to overcome resistance in crops. New cultivars of plants with resistance genes conferring complete resistance often become susceptible in just a few years if deployed over large areas due to the rapid appearance of new fungal genotypes by mutations that can infect the hitherto resistant plants. Such rapid evolution occurs even for clonal fungal pathogens with very low genetic diversity [38]. If the number of offspring is relatively small, the choice of host and/or mate is crucial for providing the offspring with the best possible genetic and environmental background [19,39]. Producing a very large number of spores represents an alternative reproductive strategy which can make the evolution of the mechanisms for the choice of host and mate unnecessary.

Mating within hosts: pleiotropy between host adaptation and assortative mating
Many pathogenic fungi can disperse over large distances after mating or by asexual spores, but they cannot disperse between infection of the host and mating. This is the case for the many ascomycete fungal pathogens (Boxes 1 and 2) responsible for most of the devastating crop diseases. In cases of obligate biotrophs undergoing sex within their host plant, mating occurs only between individuals able to grow on the same host. This means that mutations providing adaptation to a new host will pleiotropically affect local adaptation and mating patterns. This ‘magic trait’ scenario is one of the most favourable for ecological speciation [19]. A theoretical model has shown that, because of this characteristic of the lifecycle of some fungal pathogens, adaptation to a new host can significantly restrict gene flow even in sympathy without the need for mate choice or host choice [18,35,40] (Boxes 1 and 3). In this model, the barrier to gene flow is the reduced viability of immigrants [34,35]. With very strong selection this can completely prevent neutral gene flow. Several studies provide empirical support for the generality of this mechanism in natural fungal plant pathogens (Box 2).
Box 2. Host specialization as the only barrier to gene flow

The model of speciation described in Boxes 1 and 3 [35,40] predicts that pathogens mating within their hosts can become different species just by host specialization. In contrast, additional reproductive barriers (e.g. intrinsic prezygotic isolation) are required to prevent gene flow in species mating outside of their host or substrate. Data on reproductive isolation in fungi support these predictions.

A comparative analysis has been conducted on the type and degree of reproductive isolation found in vitro among closely related fungal species [55]. Basidiomycetes (e.g. mushrooms, smuts, rusts) mate outside of their substrate after having dispersed their spores, whereas most Ascomycetes (which include many crop pathogens) mate on their host or substrate after mycelial development. Results showed that closely related species of Basidiomycetes have all evolved strong intrinsic reproductive isolation at any genetic distance unless they are allopatric. For instance, sympatric species pairs of Pleurotus and of Lentinula (Figures I and II) show complete intersterility, whereas allopatric species pairs show a high degree of compatibility [55].

In contrast, closely related species of Ascomycetes could be as interfertile in vitro in sympatry as in allopatry, indicating that host adaptation can be an efficient barrier to gene flow. A good example is provided by Ascochyta pathogens. Recent multilocus phylogenetic analyses of Ascochyta fungi causing blights of chickpea, faba bean, lentil, and pea have revealed that fungi causing disease on each of these hosts form distinct species [56] (Figure III). Experimental inoculations demonstrated that each species was highly host-specific, yet experimental crosses showed that the species were completely interfertile. The host specificity of these fungi might therefore constitute the sole reproductive barrier [56].

Another excellent example of host adaptation acting as an efficient barrier to gene flow is provided by Venturia inaequalis, an ascomycete responsible for the scab disease on apples [57,58] (Figure IV) and pyracantha [59]. The two host plants grow in sympatry, but the populations on apples and pyracantha remain highly differentiated [60], indicating a lack of gene flow despite interfertility [59]. Reproductive isolation might therefore be due to a mere host specialization because cross-inoculations are unsuccessful [59] and because the fungus mates within the leaves of its host tree. Similarly, an emerging disease caused by a V. inaequalis population carrying a virulence gene allowing infection of apple trees with the resistance gene Vf has remained genetically separated from the avirulent population since its appearance 10 years ago. This is despite their co-occurrence in cider orchards containing resistant and susceptible trees [57,58], and despite interfertility [61].

We thank T. Peever, B. Le Cam, and J. Guinberteau (INRA MycSA) for the images.
Box 3. A model of speciation by host specialization

Mathematical models have shown that specificities in the life-cycles of pathogens can favour ecological speciation [35,40]. Consider sexual, haploid individuals of pathogens with four possible genotypes at two unlinked, diallelic loci A and B: A1B1, A1B2, A2B1, and A2B2. Assume that the population on the old host is close to fixation for the locally adapted genotype A1B1. The genotype fitnesses on the new host are (1 – s)2, 1 – s, 1 and 1, respectively. That is, genotype A2B2 has the highest fitness on the new host whereas the ancestral genotype A1B1 has the smallest fitness. Each spore (both immigrating from the old host and produced locally) survives viability selection and becomes an adult with a probability equal to its fitness. If the number of surviving spores is larger than the carrying capacity \( K \), they are randomly culled to have the population of adults of size \( K \). Mating is random between adults surviving on the new host. Each mating results in a random number of spores taken from a Poisson distribution with mean \( c \). Their genotypes are determined in a stochastic way subject to recombination at rate \( r \) and mutation at rate \( \mu \). A proportion \( m \) of locally produced spores immigrates. The parameters \( K \), \( c \), \( m \) and \( \mu \) are assumed to be the same for both hosts.

In numerical simulations, the dynamics of the colonization of new hosts proceed in two stages. During the first stage, all immigrating spores and surviving adults (if any) on the new host have the ancestral genotype A1B1 and thus have a very low fitness (Figures Ia and b). This stage ends when a mutation in one of the loci enters the local population and then becomes quickly fixed. The increase in the frequency of the mutation is then fast due to the assumption of a very large number of spores produced by each individual. The duration of the first stage is mostly dependent upon the overall probability of an advantageous mutation entering the population (which is approximately \( K^*c^* \mu \)). The second stage (during which the individuals present on the new host have intermediate genotype A1B2 or A2B1) ends if an advantageous mutation in the other locus enters the population and then becomes quickly fixed. The second stage is much shorter than the first one (Figures Ic and d). This is because fixation of the first advantageous mutation results in a significant increase in the population size, which in turn increases the overall number of advantageous mutations.

Figure IV. Lesion on a leaf and fruit of an apple tree caused by the fungus Venturia inaequalis.

Figure I. The dynamics of adaptation to a new host. (a) Number of adults and (b) the number of spores on the new host. (c) Genotype densities and allele frequencies. The blue line represents the density of A1B2 or A2B1 genotypes, and the red line the frequencies of A1B2 genotypes. (d) Frequencies of the A2 and B2 alleles, allowing infection of the new host.
These arguments about plant pathogenic fungi are similar to those made about host shift speciation in phytophagous insects mating on their host plants [39]. However, there are important differences between the two types of organisms. Insects can disperse at any stage of their lifecycle, so divergence in local adaptation traits and host plant preference traits is required for substantial ecological differentiation. Selection must therefore most often generate linkage disequilibrium between the genes controlling these two types of traits [19,39]. In contrast, in many plant pathogens there is no dispersal stage between selection for local adaptation and mating. This implies that mating always happens only between individuals who survived selection on the same host, removing the need to evolve strong linkage disequilibrium and thus making conditions for speciation easier. It has been suggested that, in some phytophagous insects, host choice traits can be magic traits pleiotropically controlling local adaptation and mating [41,42].

**Strong disruptive selection by host plants acting on a small number of pathogenicity-related genes**

Another factor that could facilitate ecological speciation in fungal pathogens is strong selection imposed by their host plants. Strong selection is effective in increasing the frequency of locally advantageous alleles and preventing the immigration of locally deleterious ancestral alleles [19,43]. Many plants have resistance genes involved in immunity whereas pathogens have effector genes that contribute to pathogenicity. Recognition of the pathogen effectors by a resistant plant activates a defence reaction that completely prevents the establishment of a harmful infection. This resistance mechanism imposes a maximum fitness cost: fitness of a fungal strain carrying an ‘avirulent’ allele on a plant carrying the resistant allele is zero because the resistant plants can recognize these pathogen genotypes and completely prevent their growth (see Glossary).

Such resistance mechanisms can promote ecological speciation not only because of the strength of fitness costs, but also because they involve a single diallelic locus in the host and a single diallelic locus in the pathogen (thus being called ‘gene-for-gene’ relationships) [44]. In general, selection is most efficient if it acts on a small number of genes [43]. Gene-for-gene relationships are usually identified based on within-species polymorphisms, but the genetics underlying the ability to infect a particular plant species by a given fungal species (as compared with a closely related fungal species unable to parasitize this plant species) looks similar to gene-for-gene relationships (at least in some cases) [45–47]. Further, even if the ability of infecting particular host species does not correspond to the canonical gene-for-gene relationship, there are several examples in plant pathogenic fungi in which infection of a particular host is due to a change in a single locus or in a cluster of tightly linked loci. In the fungal plant pathogen *Alternaria alternata*, a cluster of several genes involved in the production of toxins controls host-specific pathogenicity [48]. Similarly, several genes that contribute to the ability of the fungus *Nectria haematococa* to cause disease on pea plants are linked on a supernumerary chromosome, and are therefore not present in all strains [49]. In these cases, lack of the matching genes will result in zero fitness on particular hosts. The genes involved in the quantitative features of pathogenicity, such as those encoding secondary metabolites [50], are also frequently tightly linked in fungi.

**Asexuality and selfing: reducing gene immigration**

Many fungal pathogens can have multiple asexual cycles and most often only a single event of sexual reproduction per year. Ecological speciation should be facilitated by multiple cycles of asexual reproduction because these correspond to multiple cycles of selection for local adaptation without recombination breaking down locally advantageous allelic combinations and introducing locally deleterious immigrant alleles. The mixture of sexual and asexual reproduction present in most fungal pathogens allows the creation of new genetic combinations and the rapid amplification by selection of those combinations that facilitate speciation by host shifts means that two distinct pathogenic species could be considered as one. This can lead to the development of control measures that overlook the specificities of each species, such as specific fungicide resistance [62]. Accurate delimitation and identification of species is also fundamental for making sound quarantine decisions and policies, and for the implementation of strategies specifically designed to target the right taxa. Furthermore, host adaptation alone can allow for very rapid speciation via host shift, so rapidly evolving markers would be needed to delimit the species [63].

Second, it is important to link emerging diseases with ecological speciation to assess if new diseases are due to spillover, host range expansion, or host shift speciation. These different scenarios affect the control measures to be taken (e.g. whether one or multiple hosts should be targeted by fungicides). Also, the dynamics and evolution of the disease will be different if the pathogen is adapted to a single versus multiple hosts [64] or if the newly attacked host is only a reservoir for the second host.

Finally, if host adaptation is sufficient for speciation onto a new host via host shifts, then disease emergence can be relatively rapid. This should be taken into account in theoretical models aiming to understand and predict disease emergence. Furthermore, models of the evolution of fungal pathogens characteristics (e.g. virulence) should take into account the specificities of the pathogen lifecycle. For instance, the software package Quantinemo [65] in which no dispersal between selection and mating is allowed, can be used for modeling pathogens mating within their hosts. Also, the lifecycle should be considered when predicting which pathogens are the most serious threats: plant fungal pathogens mating within their hosts are expected to cause disease emergence on novel hosts more readily. This feature should be accounted for in quarantine policies, control design, and plant breeding programs.

**Box 4. Some implications of linking emerging diseases and ecological speciation**

Recognizing that emerging diseases caused by fungal plant pathogens often result from host shift speciation, and that several characteristics of fungal plant pathogens render them conducive to this type of ecological speciation, will improve our knowledge of the mechanisms responsible for disease emergence and the biodiversity of fungi. In addition, this recognition has several important consequences for our understanding of disease dynamics and evolution, as well as for designing more efficient and sustainable control programs.

First, if host adaptation alone can be sufficient for speciation, then intersterility, one of the most commonly applied criteria for delimiting species, will not be an appropriate criterion. Interfertility can be retained long after gene flow has ceased between plant pathogens species if the sole reproductive barrier is host adaptation. Failure to recognize that host adaptation can be an efficient barrier facilitating speciation by host shifts means that two distinct pathogenic species could be considered as one. This can lead to the development of control measures that overlook the specificities of each species, such as specific fungicide resistance [62]. Accurate delimitation and identification of species is also fundamental for making sound quarantine decisions and policies, and for the implementation of strategies specifically designed to target the right taxa. Furthermore, host adaptation alone can allow for very rapid speciation via host shift, so rapidly evolving markers would be needed to delimit the species [63].

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promote infection of a new host [51]. Selfing in some pathogenic fungi can also facilitate ecological speciation by reducing the probability of mating with pathogens adapted to other hosts [52].

Conclusions

We argue that host shift speciation is one of the main routes for emergence of new fungal diseases of plants, and that some life-history traits of fungal plant pathogens can facilitate rapid ecological divergence. These are: (1) strong disruptive selection imposed by the hosts; (2) a large number of spores increasing the possibility of survival on a new host and levels of adaptive variation created by mutation; (3) mating within the host, creating pleiotropy between host adaptation and assortative mating; (4) a small number of genes underlying the specificity of host–pathogen interactions; and (5) frequent asexual reproduction with rare events of sexual recombination. These specificities of plant pathogenic fungi make them prone to rapid ecological speciation. These features have been neglected but require more focused attention, in particular given their importance in emerging diseases on plants.

We have focused here on plant fungal pathogens, but the same arguments can be applied to many pathogens, including nematodes, bacteria, and viruses. These share many traits with plant pathogenic fungi that should render them prone to ecological speciation by host shifts, i.e. the production of numerous propagules, gene exchange occurring within hosts, linkage of traits experiencing selection, and strong selection imposed by the hosts [53,54].

In conclusion, we argue that existing models of ecological speciation are not appropriate for understanding how pathogens form new species on novel hosts, thereby causing emerging diseases, because they ignore the specificities of the lifecycle of plant pathogens. We need to think differently about life-history traits to tailor models based on specificities of pathogens if we are to fully understand emerging diseases. Linking ecological speciation with emergent diseases can also have practical implications (Box 4). In case studies, we should focus on the mechanisms allowing rapid adaptation and those preventing gene flow between emerging populations and their population of origin.

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