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Resilience of forests to pathogens: an evolutionary ecology perspective

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Both natural and managed forests are currently suffering from increases in damage by pathogens. Here, an evolutionary ecology approach is adopted to analyse the factors that influence the levels of pathogen damage experienced by forest tree populations and consider the conditions under which stable co-existence of trees and pathogens occurs in natural populations. The demographic and genetic responses of tree–pathogen systems to anthropogenic perturbations are explored to identify where the greatest threats to resilience lie. Problems caused by native pathogens are likely to arise as a consequence both of rapid climate change and of forest management practices that lead to increases in species density, drastic reductions in genetic diversity and planting outside the native range. The most serious threats to forest trees are posed by introduction of exotic pathogens derived from related exotic tree species. Recovery following spread of exotic pathogens is likely to be both slow and uncertain and may not be possible without intensive programmes involving rapid selection and widespread dissemination of genotypes resistant to the exotic pathogen.

Introduction

In natural and managed forest ecosystems, trees coexist with their pathogens. Under most circumstances, the damage inflicted by pathogens on tree populations is small and acceptable, giving rise to little comment. However, from time to time, pathogens have significant impacts on forests and in certain situations may effectively eliminate susceptible tree species from landscapes, or even from whole continents (Liebhold *et al.*, 1995). In recent years, there has been a significant increase in the frequency with which such damaging pathogen outbreaks have arisen in forests and there is widespread anticipation of future increases in devastating forest pathogen epidemics (Brasier, 2008; Stenlid *et al.*, 2011; Forestry Commission, 2012; Meentemeyer *et al.*, 2012). Given the power of pathogens to alter forest tree abundance and distribution, an understanding is needed of how pathogens interact with trees at the individual, population and community level in order both to identify the factors that have caused an upsurge in forest pathogen damage and to manage their impacts (Desprez-Loustau *et al.*, 2007).

When pathogens and hosts interact in natural populations, changes can occur both in the genetic composition and in the abundance and distribution of both partners. These genetic and demographic changes are interlinked. Therefore, an evolutionary ecology approach is essential for capturing and describing how pathogens affect populations of trees (Parker and Gilbert, 2004). The first section of this paper reviews our understanding of the factors affecting the amount of damage suffered by individual trees within populations. The second section considers the co-evolutionary dynamics of trees and pathogens in natural conditions where there is stable coexistence. The final section analyses the ways in which this dynamic equilibrium may be affected by anthropogenic perturbations. Examples and scenarios are drawn predominantly from forestry in Europe and North America, and the resulting perspective will necessarily differ from that drawn from experience with predominantly exotic plantations in the southern hemisphere (Wingfield *et al.*, 2000, 2001, 2008).

Factors affecting impact of pathogens on individual trees

Before considering how pathogens influence the performance of tree population in particular ecological settings, it is helpful to identify the factors that will determine the amount of pathogen damage suffered by individual trees. Some of these factors are a function of the abiotic environment, some are dependent on the biotic environment, including the genotypes of the tree and the pathogen, whereas others depend on interaction between the abiotic and biotic components of the environment.

Pathogen pressure

The first factor influencing the amount of damage suffered by a tree is pathogen pressure, defined as the number of pathogen propagules attempting to invade the vulnerable tissue of the tree within a set period of time. Pathogen pressure is determined firstly by the number of effective pathogen propagules present in the environment. This will be a function of the density of infected

host trees if the pathogen is spread only within species, by the density of alternate or secondary hosts if there is transmission between species, and will be modulated by the density of any vectors that may be required for transmission.

Thus, in snow blight caused by *Phacidium infestans*, where direct transmission occurs between hosts, pathogen pressure is a function of the density of infected Scots pine, *Pinus sylvestris* (Burdon *et al.*, 1994a,b). For *Phytophthora ramorum* on tanoak, *Lithocarpus densiflorus*, pathogen pressure is largely a function of the density of infected individuals of the primary host bay laurel, *Umbellularia californica*, which produces pathogen propagules more prolifically than tanoak (Cobb *et al.*, 2010). For white pine blister rust, *Cronartium ribicola*, pathogen pressure on five needled pines is primarily affected by the local density of the alternate host *Ribes* (Geils *et al.*, 2010). Finally, for the Dutch elm disease fungus *Ophiostoma novo-ulmi*, pathogen pressure is not merely a function of the density of diseased elm, *Ulmus* spp., but of the abundance of sympatric *Scolytus* beetles that transmit the disease (Webber and Brasier, 1984).

The other important factor influencing pathogen pressure is the suitability of the abiotic and biotic environmental conditions for pathogen invasion. This is directly related to the ecology of the pathogen concerned. Thus, high pathogen pressure of red band needle blight *Dothistroma septosporum* is found in microclimates with temperatures between 15 and 20°C and sustained periods of leaf wetness which allow for successful spore dispersal and germination (Woods *et al.*, 2005). For the pine twisting rust *Melampsora pinitorqua*, pathogen pressure is maximized under optimal biotic conditions for infection where the timing of spore release from aspen, *Populus tremula*, coincides with the date of shoot extension in maritime pine, *P. pinaster* (Desprez-Loustau and Dupuis, 1994).

When the environmental and biotic conditions influencing pathogen pressure are understood, it is possible to map variation in pathogen pressure across landscapes. In this way, disease hazard ratings can be allocated to particular areas as has been achieved for *C. ribicola* (Ostry *et al.*, 2010) and *P. lateralis* (Hansen *et al.*, 2000). Such hazard rating may ultimately be very important when planning strategic planting of resistant tree species and genotypes across the landscape to manage disease.

Probability of initial establishment

The second factor affecting the amount of damage suffered by a tree is the probability that the attempted invasion of vulnerable tissue by a pathogen is translated into establishment in the host. Defence mechanisms preventing pathogen establishment act very early in infection. They rely on the tree recognizing the presence of a pathogen and triggering an effective defence response in the appropriate local area of tissue (Eyles *et al.*, 2010; Dangl *et al.*, 2013; Kovalchuk *et al.*, 2013). Typical responses in trees are hypersensitive reactions and local cell death in needle and leaf tissues after attempted biotrophic rust invasions (Kinloch and Littlefield, 1977; Laurans and Pilate, 1999; Kovalchuk *et al.*, 2013).

Variation in the probability of initial establishment of pathogens is genetically controlled and is the consequence of interaction between tree and pathogen genotypes. Along with other plant species, trees have a battery of resistance genes, known collectively as R genes. R genes in the tree code for molecules that detect either directly or indirectly the products of pathogen AVR ('Avirulence') genes. If either elicitor molecules/molecular patterns coded by pathogen AVR genes or damage to the tree's defence system caused by AVR gene products is detected by R gene molecules, a suite of defences is mounted that prevents establishment of the pathogen (Jones and Dangl, 2006; Dangl *et al.*, 2013).

In natural tree populations, there is genetic variation such that, for each R gene, some individuals produce R gene products (genotype *R*+) whereas others do not (genotype *R*−). Trees of genotype *R*− are unable to detect the products of corresponding AVR pathogen loci and, therefore, are susceptible to these pathogens. On the other hand, they do not invest energy in R gene products and therefore are expected to have higher fitness in the absence of pathogens (Tian *et al.*, 2003).

In natural pathogen populations, there is corresponding genetic variation at the AVR loci, with some individuals producing normal AVR products (genotype *AVR*+) and others produce no product or modified product (genotype *AVR*−). Pathogens of genotype *AVR*− are not detected by and can infect tree genotype *R*+ as well as *R*−. However, *AVR*− genotypes have lower fitness compared with *AVR*+ on host genotype *R*− possibly because they do not exploit the host so effectively without the aid of the normal AVR gene product (Huang *et al.*, 2010).

Pathogen development following initial establishment

Once a pathogen has become established within a tree, the damage that it causes will depend on its abilities to overcome the tree defence systems. Trees use a variety of strategies to limit pathogen development including chemical modification of tissues surrounding infection foci to slow down invasion, differentiation of impermeable barriers and plugging of vessels in the vascular system with tyloses (Shigo, 1984; Eyles *et al.*, 2010; Kovalchuk *et al.*, 2013). For any one of these defence mechanisms, there will be many loci that can mutate to provide quantitative variation for the defence trait. For instance, variation could occur at loci governing the intensity of the defence response (Todesco *et al.*, 2010), loci determining the chemical composition of resins used to slow down pathogen invasion (Ennos and Swales, 1991) or loci controlling the anatomy of xylem and its capacity to block vessels in the event of invasion (McNabb *et al.*, 1970; Venturas *et al.*, 2014). This means that we expect to see substantial quantitative genetic variation in pathogen resistance among trees within populations that is independent of the R genes that code for recognition systems. Indeed, for many necrotrophic pathogens which make no attempt to avoid detection by the host tree, genetic variation in resistance may not be associated with R genes responsible for pathogen recognition, but with variation at loci influencing the strength and efficacy of the defence mechanisms themselves (Eyles *et al.*, 2010).

A common feature of all of the defence mechanisms utilized by trees is that they are costly in terms of energy and non-carbon resources. Induction of defences can reduce radial growth of a seedling by 30 per cent (Krokene *et al.*, 2008). This has two implications. The first is that there will be selection against investment in these defence mechanisms in situations where pathogen pressure is low. The second is that where trees are under stress, their ability to generate the resources required for an effective defence response will be compromised, and they will be susceptible to infection by necrotrophic and opportunistic pathogens many of which would otherwise be innocuous.

There is a huge observational and experimental literature demonstrating that trees growing under stressed conditions have a reduced ability to prevent spread of necrotrophic and opportunistic pathogens in their tissues (Schoeneweiss, 1975;1981). For instance, *P. sylvestris* growing under water stress shows reduced ability both to form suberized barriers and secrete resin in response to root invasion by *Heterobasidion annosum*, leading to lower resistance (Rishbeth 1951; Gibbs, 1968). In natural populations of trees growing in heterogeneous environments varying in level of stress, a high proportion of the quantitative phenotypic variation in resistance to disease may therefore be caused by environmental variation over both space and time.

Tolerance to tissue invasion

The final factor that will determine the effect of pathogens on individual trees is the extent to which invasion of tree tissues translates into a reduction in the ability of the tree to reproduce. This could be described as the tolerance of the tree to the pathogen. Tolerance will depend firstly on the life cycle stage affected. Many large trees are likely to be able to tolerate significant amounts of tissue invasion by pathogens without detectable effects on their production of seeds. However, the same amount of tissue infection in a seedling or sapling may be fatal. Thus, the pathogen pressure generated by an infected mature tree, whose own seed production is not affected, may be sufficient to prevent regeneration of seedlings in its immediate vicinity (Augspurger and Kelly, 1984; Packer and Clay, 2000). To understand the impact of tree diseases, it is therefore essential to account for their effects over all life cycle stages.

The tolerance of trees to pathogen invasion will also be influenced by the particular tissue that is invaded. For some diseases that kill cambial tissue and girdle main stems, small amounts of tissue infection can lead to rapid death and total elimination of sexual reproduction and regeneration. This is the case for chestnut blight in the USA where killing of cambium and girdling of main stems kills mature trees preventing any further seed production within stands (Anagnostakis, 1987). In contrast, for many foliar and root diseases, tissue damage causes a reduction in growth rate that is a function of the percentage of infected tissue (Shaw and Toes, 1977; Bastiaans, 1991). Reduction in tree growth rate will ultimately translate into a reduction in tree survival and reproduction, whose severity will depend on the competitive environment in which the tree is growing.

As in the case of genetic variants that reduce disease progress, there are likely to be genes in the tree that may vary to affect tolerance of pathogen invasion (Desprez-Loustau *et al.*, 2014). Genes affecting disease progress in tissues and tolerance of infection by the pathogen will be referred to collectively, in the remainder of this article, as quantitative resistance genes.

Co-evolutionary dynamics of trees and pathogens in natural populations

Having established the environmental and genetic factors that influence the impact of pathogens on the performance of individual trees, it is now possible to make, and in some cases test, predictions about the distribution of genetically determined resistance variation in naturally co-evolved tree and pathogen populations, and the response of such populations to the range of environmental fluctuations that they normally encounter. The outcome of co-evolutionary interactions between R genes and corresponding AVR genes are analysed first, followed by an analysis of the behaviour of genes conferring variation in rates of pathogen development and tolerance (quantitative resistance genes) under some simple ecological scenarios.

Co-evolution involving R *and AVR loci in natural populations*

To appreciate the co-evolutionary interactions involving R genes in trees and AVR genes in pathogens, consider a situation where all individuals in a tree population are of genotype *R*+ at a given locus and are capable of detecting the elicitor molecule or damage produced by the elicitor molecule of a given pathogen in which all individuals are of genotype *AVR*+. Initially, all pathogen individuals will be recognized by the tree, defence reactions will be mounted and pathogen invasion will be prevented. In this situation, there will be strong selection for *AVR*− pathogen genotypes, with elicitors that are modified or absent, which will no longer be recognized by trees of genotype *R*+. Though able to infect the *R*+ tree genotype, *AVR*− genotypes are likely to be less efficient at exploiting their hosts than *AVR*+ genotypes because they do not possess the most effective elicitor molecule or are unable to debilitate the tree's defence system (Huang *et al.*, 2010).

As a consequence of natural selection, the frequency of *AVR*− genotypes will rise in the pathogen population, and possession of the *R*+ genotype by the tree will come to confer no selective advantage. Indeed, because production of the R gene product is likely to be costly, there will now be selection in the host population for *R*− genotypes that fail to produce the R gene product (Tian *et al.*, 2003). When the *R*− genotype reaches sufficient frequency in the tree population, selection will once again favour the *AVR*+ pathogen genotype that can not only infect the *R*− tree genotype but exploit it most efficiently with the use of its elicitor or defence debilitating molecule. Finally, when the pathogen *AVR*+ genotype reaches high frequency, this will favour selection of trees of genotype R+ that produce the R gene product and can detect the *AVR*+ pathogen genotype. We are back at the beginning of the selection cycle (Stahl *et al.*, 1999; Brown and Tellier, 2011).

Evolutionary interactions between R and AVR loci therefore generate unstable cycles of selection that tend to maintain both *R*+ and *R*− genotypes in the tree population, and *AVR*+ and *AVR*− genotypes in the pathogen population (Salvaudon *et al.*, 2008; Brown and Tellier, 2011). The cycles may be stabilized by negative frequency-dependent selection arising under a range of ecological and environmental conditions (Brown and Tellier, 2011) and lead to a situation described as 'trench warfare' in which neither pathogen nor tree has the upper hand.

When tree and pathogen populations show variation at many R and corresponding AVR loci, respectively, what consequences will this have for the resistance of the tree population? The first is that the R genes will confer partial rather than complete resistance on the pathogen population. The resistance is partial because any one tree, with a particular multilocus R genotype, is likely to be able to recognize and eject only a proportion of the multilocus AVR pathogen genotypes encountered.

The second consequence is that there will be quantitative variation in the level of pathogen damage suffered by individual trees (Carson and Carson, 1989; King and Lively, 2012). Different multilocus R tree genotypes will be susceptible to different sets of

multilocus AVR pathogen genotypes. Furthermore, these multilocus AVR pathogen genotypes will cause different degrees of host damage. The more *AVR*− alleles possessed by a pathogen genotype, the more its ability to debilitate the defence system of the tree will be compromised, and the lower will be the damage that it causes (Thrall and Burdon, 2003).

The third consequence of polymorphism at corresponding R and AVR genes is that the partial resistance that it confers on the host population will be stable. This stable partial resistance is a population-level phenomenon. It derives from the genetic variability of the tree population at R loci and the co-evolutionary constraints this imposes on the pathogen population which ensure that a single pathogen genotype able to infect all trees does not evolve. In contrast, stable resistance is not exhibited by tree populations that lack genetic variation at R loci. This is demonstrated by the observation that when genetically uniform crop populations containing R alleles conferring resistance to all current pathogen genotypes are planted, initial resistance breaks down very quickly as a consequence of the selection of a single pathogen genotypes with appropriate *AVR*− alleles capable of infecting all individuals, leading to catastrophic disease epidemics (McDonald and Linde, 2002).

Co-evolution involving quantitative genetic resistance to tree pathogens

As emphasized earlier, variation at genes affecting many different traits may confer quantitative variation in the rate of pathogen spread and tolerance to pathogens (quantitative resistance) on tree populations. In this section, predictions are made about the levels of quantitative resistance expected to evolve in natural populations of forest trees under different ecological scenarios, and evidence is presented to support these predictions.

Consider two tree species, one of which is found at low frequency, the other at high frequency within a forest community. Suppose that each of these species has its own species-specific pathogen or pathogen community (Hersh *et al.*, 2012; Benitez *et al.*, 2013). The level of quantitative resistance that evolves in each of these species will depend on the fitness benefits gained from increased resistance, the costs associated with increased resistance and the ecological opportunity for the evolution of increased quantitative resistance.

For the species at low density, species-specific pathogen pressure is likely to be low due to the low density of hosts in the population. Furthermore, pathogen pressure will tend to be localized in the vicinity of infected trees, and it is only in these areas that increased pathogen resistance will be selectively advantageous. In the vicinity of infected trees, any genotypes with higher resistance will be competing predominantly with individuals of other tree species unaffected by the species-specific pathogen or pathogen community. Genotypes of higher resistance are therefore likely to be out-competed by individuals of alternative tree species unless the resistance increase is very substantial and comes at very little cost. Thus, for species at low density, the fitness benefits gained by increased quantitative resistance are low, and there is little ecological opportunity for natural selection to bring about increases in quantitative resistance (de Mazancourt *et al.*, 2008). Equilibrium levels of quantitative resistance are therefore expected to be low for species at low density in the forest. The most likely response of such species to an increase in pathogen pressure is a reduction in population density with no change in quantitative resistance.

This contrasts with tree species at high density where, because of the density of hosts, pathogen pressure will tend to be consistently high throughout the population. Genotypes with greater quantitative resistance to pathogens will have higher fitness wherever they occur. Furthermore, trees at high density will be competing predominantly against other individuals of the same species. This provides the ecological opportunity for natural selection to lead to an increase in mean quantitative resistance of the population over time. The equilibrium level of mean quantitative resistance attained in the population will depend on a trade-off between the advantages gained and the resource costs imposed by greater quantitative resistance. In sites where environmental conditions lead to higher pathogen pressures, populations showing a higher equilibrium level of quantitative resistance are expected to evolve. In summary, species present at high density are predicted to show higher quantitative resistance to pathogens than species at low density, and in species at high density, the level of quantitative resistance in a population should be related to the pathogen pressure at the site where that population has evolved.

There is both experimental and empirical evidence consistent with the first prediction that the quantitative pathogen resistance of tree species is related to their density in the forest. Among a sample of six tree species from forests in Panama, the negative impact of species-specific soil pathogens on seedlings was inversely related to the frequency of the species in the forest (Mangan *et al.*, 2010). The implication is that tree species present at low density are less resistant to their species-specific soil pathogens than are species present at high density. In complementary analyses of seedling forest plots from both tropical and temperate forests, seedling survival in the presence of conspecific adults was reduced much more in low-density tree species than in high density species (Comita *et al.*, 2010; Johnson *et al.*, 2012). Again this is consistent with the prediction that tree species present at high density have evolved greater quantitative resistance to species-specific pathogens or pathogen communities than species present at low density.

While a relationship between tree density and levels of pathogen resistance may be a general rule, there are likely to be exceptions. Thus, if the biology of a pathogen means that it is limited in its spatial distribution within a population, only a small fraction of the host tree population will be exposed to selection, even if the tree is present at high density. As a consequence, especially if resistance imposes a high cost, increases in resistance to this pathogen may not evolve. Thus, in the Pacific Northwest of America, indigenous tree species found at high density, such as *Pseudotsuga menziesii*, show little or no resistance to the endemic root-infecting pathogen *Phellinus weirii*, even when growing vigorously (Hansen and Goheen, 2000). In this case, the limited spatial distribution of the pathogen, the small proportion of the population subject to selection by the pathogen and the high resource costs associated with appropriate levels of defence may preclude evolution of increased resistance of *P. menziesii* to in *P. weirii*.

The second prediction, that the evolved level of quantitative resistance in high density species is related to pathogen pressure at the site of origin, is supported by results from a variety of provenance experiments. For *P. sylvestris* populations in Sweden, resistance to *P. infestans* is closely related to latitude

and extent of snow cover in winter (Bjorkman, 1963). In western larch, *Larix occidentalis*, there is a strong relationship between precipitation levels at the site of origin and resistance to needle cast disease, *Meria laricis,* which is favoured by moist conditions (Rehfeldt, 1995). In a *P. banksiana* provenance trial, there was a positive correlation between the quantitative resistance of provenances to sweet fern rust, *Cronartium comptoniae* and the abundance of sweet fern, *Comptonia peregrina,* the alternate host, in the sites from which they had been derived (Hunt and van Sickle, 1984). In *Eucalyptus globulus,* high quantitative resistance to leaf disease was detected in populations from sites where high summer temperature and rainfall are found, conditions that favour pathogen infection (Hamilton *et al.*, 2013). In crosses between resistant and susceptible individuals of *E. globulus*, variation at two loci accounted for 55 per cent of the differences in resistance suggesting that changes at relatively few genes can bring about large differences in quantitative resistance to pathogens (Freeman *et al.*, 2008).

Co-evolution under fluctuating environments

In natural populations, levels of pathogen resistance are generally expected to evolve in response to the average pathogen pressure at a site. At equilibrium, both tree and pathogen populations will generally co-exist with a relatively minor impact of the pathogen on the tree population. However, from time to time, environmental conditions may be such as to favour rates of attempted pathogen infection above normal levels, and this will lead to a temporary increase in pathogen pressure. For instance, there may be an unusually wet summer providing ideal conditions for spore survival and germination in a fungal pathogen, e.g. *Lophodermium seditiosum* on Scots pine, *P. sylvestris* (Martinsson, 1979). The result will be an increase in the pathogen damage experienced by trees in the population that have evolved resistance levels compatible with lower pathogen pressure. An epidemic outbreak of the disease will then occur that will last until the unusual weather conditions cease and pathogen pressure declines, at which point tree damage levels will return to normal.

If tolerance of trees to the disease over the limited time period of the outbreak is sufficiently high, it is unlikely that significant changes either in tree density or quantitative genetic resistance will occur. However, in the absence of sufficient tolerance, tree density may be reduced and/or selection for greater quantitative resistance may occur. Therefore, species density may be lower and level of quantitative resistance may be higher than expected under current pathogen pressure as a consequence of the higher pathogen pressure generated by infrequent episodes of extreme environmental fluctuation in the past.

Apart from altering pathogen pressure, unusual weather conditions may also impose temporary stress on tree populations. This will reduce their quantitative resistance below that needed to combat the pathogen pressures normally encountered. In these situations, a temporary increase in pathogen damage is expected which ceases when the stress is removed. Typical examples of disease triggered by temporary stress are increased damage by root-infecting pathogens during periods of drought (Rishbeth, 1951; Gibbs, 1968) and outbreaks of latent endophytic pathogens (Slippers and Wingfield, 2007). Again, provided tolerance to infection is sufficiently high to allow survival over the period of stress, no significant changes either in tree density or quantitative genetic resistance are expected. However, if tolerance is insufficient, tree density will be reduced by pathogen infection. In these circumstances, species distribution and density will partly reflect past episodes of infrequent but extreme environmental fluctuations (Cavin *et al.*, 2013).

Effects of anthropogenic perturbation on tree–pathogen systems

Anthropogenic perturbation of forest – pathogen systems may occur as a consequence of rapid anthropogenic climate change, as a result of different types of intensive forest management (plantation and clonal forestry), from deliberate movement of tree species outside their natural ranges (exotic forestry) and from inadvertent introduction of exotic pathogen species. The anticipated consequences of these perturbations are analysed in the following section.

Anthropogenic climate change

If rapid, anthropogenic climate change occurs in an area, one consequence may be the natural spread of a pathogen, previously absent, into this area, and/or to an increase in the pathogen pressure of an indigenous pathogen. A current example of this phenomenon appears to be the epidemic outbreak of *Dothistroma* needle blight on *P. contorta* in British Columbia associated with a local increase in summer precipitation (Woods *et al.*, 2005; Welsh *et al.*, 2009). The resulting chronic increase in pathogen pressure may initially reduce the density of individuals and for species at high density will impose long-term selection for an increase in quantitative resistance. If there is sufficient genetic variation for quantitative resistance available, a tree population with greater mean quantitative resistance may evolve provided genetic change is sufficiently rapid that it occurs before the population goes extinct. The process by which populations avoid extinction by adapting to the relevant threat has been termed 'adaptive escape' (Gomulkiewicz and Holt, 1995; Hoffmann and Sgro, 2011).

Adaptive escape is most likely where populations are initially large and genetically diverse. It will also be facilitated by gene flow from populations of the same species elsewhere in the landscape that are already adapted to higher pathogen pressure. Adaptive escape can only occur if increases in pathogen pressure do not prevent the process of natural regeneration, because without population turnover and the opportunity for natural selection to operate, there can be no long-term response to natural selection (Cavers and Cottrell, 2015). As a result of adaptive escape, damage levels should return to those found before environmental change occurred.

Evidence that evolution of higher quantitative resistance can occur in the presence of long-term increases in pathogen pressure comes from studies of live oak, *Quercus fagacearum*, populations in Texas into which oak wilt, *Ceratocystis fagacearum*, has recently spread and caused significant death of mature trees (Juzwick, *et al.*, 2008). When inoculated with oak wilt, seedlings from adult trees that have survived oak wilt show significantly higher survival (82 per cent) than seedling from populations that have not yet been affected by oak wilt (62.5 per cent) (Greene and Appel, 1994). Effective selection for higher quantitative resistance has occurred and is associated with better containment of the disease by the seedlings.

Another possible consequence of rapid anthropogenic climate change will be loss of local environmental adaptation in tree populations and a corresponding permanent increase in stress. If this

occurs, increase in damage not by one but by a suite of opportunistic pathogens is expected. In conjunction with other manifestations of reduced fitness, this may drive the population towards extinction. Adaptation of the tree population to the new environment may prevent this occurring, if it takes place sufficiently rapidly. Once again adaptive escape is most likely where tree populations are initially large and genetically diverse, where pathogens do not prevent natural regeneration, and where gene flow is possible from populations located elsewhere that are already adapted to the new climatic conditions (Gomulkiewicz and Holt, 1995; Hoffmann and Sgro, 2011).

Reduction in species diversity

A common objective in commercial forestry is to encourage dominance by the most valuable species. In the process, this necessarily decreases species diversity in the stand and increases the density of the favoured species. This is taken to the extreme in single species plantation forestry. For pathogens that are transmitted from tree to tree, this may increase pathogen pressure to a level above that to which the host is adapted in the native forest, and an increase in damage by pathogens is therefore anticipated.

This phenomenon is exemplified by the rubber tree *Hevea brasiliensis* that occurs as scattered individuals in natural forests in the Amazon (Lieberei, 2007). At these low densities, the indigenous pathogen South American Leaf Blight, *Microcyclus ulei,* causes little damage. However, when *H. brasiliensis* is grown in plantations in South and Central America, it is severely damaged by *M. ulei* infection, and successful plantations are only possible on other continents where *M. ulei* is absent. Further evidence for increase in pathogen damage with increase in tree species density is provided by a meta-analysis of damage to conifers by root infecting *H. annosum*. In 9 out of 13 studies, greater damage was found in the pure than in the mixed stands (Korhonen et al., 1998).

Increased pathogen pressure at high densities is not only expected to increase disease prevalence and damage, but also to decrease stand productivity as a consequence of non-lethal loss of tissue to pathogens. There is now very good experimental evidence from grassland systems that multispecies communities suffer substantially lower root disease than mono-specific communities and that this leads to productivity gains of 40 per cent for multispecies stands over monocultures that are directly attributable to decreased root pathogen damage (Maron *et al*., 2011; Schnitzer *et al*., 2011). If the same were true for forest systems, this would be a powerful commercial argument for increasing species diversity in managed plantations.

If, nevertheless, mono-specific plantations are to be established using tree species that are naturally present at low densities in forests, the considerations outlined above indicate that genetic selection programmes for increased quantitative resistance will be required (Lieberei, 2007). Such quantitative resistance breeding is an integral component of a wide range of domestication programmes that involve both animal and plant crop species grown at commercial densities far in excess of those found in natural populations (Simmonds, 1979; Bishop *et al*., 2010).

Reduction in genetic diversity

Where vegetative propagation of tree species is feasible, selection of the best performing clones may be favoured, and genetically, invariant clonal plantations may be established. Deliberate and extreme reduction in genetic diversity of this kind is common practice in the silviculture of *Populus*, *Salix* and *Eucalyptus* (Labrecque and Teodorescu, 2005; Stanton *et al*., 2010; Rezende *et al*., 2014). This form of management has potentially very serious effects on the level of damage caused by pathogens. As explained earlier, as a consequence of host-pathogen co-evolution, natural populations of trees are genetically variable at a suite of R loci that are responsible for detecting the presence of (principally) biotrophic pathogens. The pathogens in turn are genetically variable at corresponding AVR loci. Although not wholly effective at preventing pathogen establishment, the presence of R diversity in the population provides a filter that decreases infection rates and limits the pathogen's ability to evolve mechanisms for debilitating the host defence system.

In a clonal population, there is no diversity at R loci. In this situation, biotrophic pathogens are expected to evolve *AVR*− alleles corresponding to the *R*+ alleles in the clone, so enabling them to evade detection. Once this pathogen genotype has evolved, it will be capable of infecting all individuals within the plantation without eliciting a hypersensitive defence response. Moreover, because the probability of successful transmission to a new host is very high, further evolution is expected that will increase the net reproductive rate of the pathogen (Holguin and Bashan, 1992; Lannou, 2012). The anticipated outcome is high disease incidence of a pathogen that rapidly colonizes tissue and causes high levels of damage.

Compelling evidence supporting this scenario is provided by experience with clonal *Populus* plantations. These are very seriously affected by a variety of highly specialized races of damaging pathogens that include fungi in the genera *Marssonina*, *Melampsora*, *Septoria*, *Dothichiza* and the bacterium *Xanthomonas* (Nesme *et al*., 1994; Pinon and Frey, 2005). A major criterion for breeding of clonal genotypes for large-scale deployment is that they are resistant to the current population of pathogen genotypes within, for instance, *Melampsora spp.* rusts. However, when clones are released into the field on a large scale, the experience has been that this resistance breaks down, often within less than a decade, as a consequence of adaptation of the pathogen population to the *R* alleles that have been incorporated into the clone (Pinon and Frey, 2005). Further clones must then be selected that are resistant to the newly evolved populations of pathogens. The result is an unstable boom-bust cycle where there is no lasting resistance.

A number of experiments in which multi-clonal willow and poplar populations have been established suggest that they may suffer less pathogen damage (Mundt, 2002; McCracken and Dawson, 2005; Pinon and Frey, 2005). However, it is unlikely that the scale of R diversity found in natural populations will be replicated in these multi-clonal plantations, and while they may perform better than single clone populations, they do not appear to achieve the level of durable resistance required.

Alteration in species distribution

It is common practice in forest management to extend the planting of exploited species to sites where they would not naturally occur. This may be within the geographic range of the species, or in the case of exotic plantations well outside these boundaries. Many such plantings have suffered from pathogen problems and

indeed have revealed the importance of pathogens in defining the natural ranges occupied by tree species.

Pathogen problems may occur firstly because plantations have been established in areas with higher pathogen pressures than those from which the planting stock was derived. In southeastern USA, slash pine, *P. elliottii*, and loblolly pine, *P. taeda*, have been planted in areas previously occupied by longleaved pine, *P. palustris*. In these areas, there is increased pathogen pressure from the native fusiform rust *Cronartium fusiforme*. This is exacerbated by fire suppression, which has allowed an increase in the density of native oak *Quercus* spp. from which infectious basidiospores are released. As a consequence, rust infection is 2–3 times greater in these off-site plantings than within the natural range (Schmidt, 2003).

Continued planting of *P. elliottii* and *P. taeda* in areas with high pathogen pressure requires tree genotypes with greater quantitative resistance (Schmidt *et al.*, 2000). There is clear evidence that natural selection for greater quantitative resistance can occur within high hazard sites. In a plantation where rust incidence on *P. elliotii* was as high as 90 per cent, progeny collected from rust-free parents showed both a reduction in incidence of rust (61 vs. 85 per cent) and that in mean number of galls per tree (3.2 vs. 4.9) compared with seedling from unselected trees (Goddard *et al.*, 1975). Thus, there appears to be sufficient and appropriate genetic variation to allow *P. elliotii* to evolve increased quantitative resistance to adapt to elevated pathogen pressure exerted by a native pathogen. Artificial selection for increased quantitative resistance is currently being employed successfully, using seedling assays, to develop populations that can, in the future, be used to restock high hazard sites (Schmidt, 2003).

In exotic forestry, similar pathogen problems can occur when species are transported to areas where the environmental conditions are much more favourable to their endemic pathogens than in the native range. This situation is exemplified by the case of *P. radiata*. The species has been planted as an exotic in many areas around the world where it has repeatedly been devastated by the needle-infecting pathogen, *D. septosporum*, which causes little damage in its native range in Monterey (Gibson, 1972). In areas such as New Zealand, the warm humid conditions present in dense plantations are ideal for infection by *D. septosporum* leading to much higher pathogen pressure than in the native range. In the absence of environmentally unsound chemical treatment of the pathogen, successful growth of *P. radiata* at high density can only occur if quantitative resistance to *D. septosporum* can be increased. Breeding programmes have therefore been established to increase quantitative resistance (Carson and Carson, 1989). These have shown both that significant quantitative genetic variation for resistance to *D. septosporum* exists and that increases in resistance of 16 per cent can been achieved within a single generation of artificial selection.

Apart from transposing tree populations into higher hazard areas, range extension may also lead to the establishment of plantations that are poorly adapted to local environmental conditions. Under stress, their quantitative resistance to local pathogens will be compromised and severe pathogen damage may ensue. A particularly vivid illustration of this is provided by Corsican pine *P. nigra* planted in Britain. In sites where winter sunlight levels are too low, trees are susceptible to killing by *Gremmeniella abietina* (Read, 1968). Before the current problems with *D. septosporum*, this prevented the establishment of successful Corsican pine plantations on the north-facing slopes of river valleys, and north of the Mersey and Humber except in coastal locations. The pathogen effectively defined the environmental range within which Corsican pine could be grown.

Extensive planting of exotic species in areas where they are under stress may have repercussions for the health of adjacent native species that share the same pathogens. Epidemic development of the pathogen on stressed exotic trees will substantially increase the pathogen pressure beyond the level that can be controlled by the evolved quantitative resistance in the native species. Thus, damage to the native species is anticipated in the immediate area of the susceptible exotic species. This phenomenon may account for increased disease levels caused by *D. septosporum* on native *P. sylvestris* following outbreaks of the disease on susceptible exotic plantations of *P. contorta* in Britain (A.V. Brown, personal communication). A similar situation is found in Sweden where *P. contorta* susceptible to *G. abietina* has been planted close to native *P. sylvestris*, leading to elevated pathogen pressure on the latter species (Ennos, 2001).

In summary, the planting of tree species on non-native sites either within their natural range or as exotics may lead to significant disease problems involving indigenous pathogens either as a consequence of greater pathogen pressure in the new planting site or due to poor adaptation to the site and increased stress. These pathogen-related issues are highly relevant to and must be addressed in the policy debate concerning predictive provenancing of tree species (Broadmeadow and Ray, 2005). Decisions about relocation of provenances must take into account not only matching to climate but also the disease problems that may arise with off-site planting. It should also be borne in mind that historically the range of many commercial species has been extended by deliberate planting and that the source of many current disease problems may lie in these past planting practices. For instance, current damage by *Dothistroma* on *P. contorta* in British Columbia may be due not only to climate change but also to planting of *P. contorta* on sites that would naturally have carried western hemlock, *Tsuga heterophylla*, and subalpine fir, *Abies lasiocarpa* (Dale *et al.*, 2011).

Introduction of exotic pathogens

Global trade in live plants, in the absence of adequate phytosanitary controls, has been and continues to be responsible for the most serious perturbation of our natural forests ecosystems, namely the introduction of exotic pathogens (Liebhold *et al.*, 1995; Brasier 2008). In the past, exotic pathogens have led to the virtual elimination of taxa such as the American elm, *Ulmus americana*, and American chestnut, *Castanea dentata*, from the North American continent, and reduced other species to remnant populations in areas of low pathogen pressure (Gibbs, 1978; Anagnostakis, 1987; Kinloch, 2003). At the present time, numerous tree species are threatened worldwide by a plethora of exotic pathogens including a wide variety of *Phytophthora* taxa (Hansen *et al.*, 2012). The dangers of exotic pathogens and their association with trade in live plants have been brought most vividly into contemporary focus by the spread of ash dieback *Hymenoscyphus pseudoalbidus* across Europe from its origin in Asia (Gross *et al.*, 2014; McKinney *et al.*, 2014) and the spread of *P. ramorum* in both North America and Europe (Grunwald *et al.*, 2012).

As a general rule, most exotic pathogens will be unable to infect most native tree species. However, exotic pathogens can have such devastating effects that even very low probabilities of establishment of exotic pathogens on native trees represent serious risks to forest health. The probability of an exotic pathogen being able to infect a native species is not random but depends on the evolutionary distance between the exotic host from which the pathogen is derived and the native host (Gilbert and Webb, 2007). If the exotic and native hosts are closely related, there is a high probability that the exotic pathogen will have the capability of infecting the native host. This probability declines with the evolutionary distance between the two hosts. Thus, it is possible to predict the risk that will be posed by the introduction of an exotic host, and inadvertently its associated pathogens, to a native forest ecosystem. The risk will be greatest where the exotic is closely related to native tree species (Gilbert *et al*., 2012).

Given that an exotic pathogen is able to infect a native tree species, why is the damage it causes so severe? The exotic pathogen has sufficient shared evolutionary history with the native tree that it is able to use it as a host. However, it has experienced no intimate co-evolution with that species that would normally lead to stable co-existence through diversifying selection on R and AVR genes and selection of appropriate levels of host quantitative resistance. Initially, therefore, those native host populations that can be infected are virtually universally susceptible, and disease spread is likely to be limited only by abiotic conditions adverse to the pathogen. Pathogen pressure and damage levels to trees are likely to be very high.

Once the exotic pathogen is established, a process of co-evolution will begin in which dramatic changes in tree density and possible changes in genotype frequencies will occur. In all except low-hazard sites, rapid elimination of most, if not all, host individuals from the landscape is anticipated. However, within a genetically diverse tree population, a low frequency of individuals possessing *R*+ alleles capable of detecting and eliminating the pathogen are likely to be found. These may survive the initial phase of the epidemic. However, corresponding *AVR*− alleles will rapidly be selected from within the enormous epidemic pathogen population, even if they are present at very low frequency, leading to loss of resistance in the surviving trees. Little or no change in the frequency of the R genes will occur in the tree population because they are effective for less than a generation. It would therefore appear that in this initial epidemic situation, R genes provide little potential for the evolution of higher resistance levels in the tree population.

In contrast, any tree genotype that is able to reduce the spread of the pathogen within its tissues and/or enable the tree to tolerate infection (possessing increased quantitative resistance) to a level at which host reproduction and regeneration can occur will be at a selective advantage. Such genotypes may be present in the population, but probably only at a very low frequency. Opportunity for natural evolution of greater quantitative resistance of tree populations before their elimination (adaptive escape) is most likely in low-hazard areas. This is because the size of the host population and therefore genetic diversity will remain high, giving maximum opportunity for a response to selection for greater quantitative resistance. Moreover, small increases in quantitative resistance in these areas will be sufficient to decrease damage and increase fitness. Resistance may build up slowly from a low base making use of the small amounts of genetic variability for quantitative resistance present in the initial population. As selection proceeds and mean resistance rises, the pathogen pressure will decrease allowing tree population sizes to rise and natural selection for greater resistance to be more effective.

In a high hazard area, in contrast, population sizes and genetic variation will be low, reducing the opportunity for a response to selection. Moreover, even the best genotypes available in the population may have quantitative resistance that is insufficient to allow survival and reproduction under very high pathogen pressure, so that natural selection for increased resistance will be ineffective. A possible scenario for recovery of tree populations from introduction of exotic pathogens is therefore the gradual build-up of levels of quantitative resistance in refugial populations occupying low-hazard sites. Once quantitative resistance is sufficient, natural selection for individuals possessing R genes capable of recognizing and eliminating the pathogen may occur. Over many tree generations, the same degree of corresponding polymorphism at R and AVR loci could evolve as that found in native pathogen systems. At this point, extension of the population to areas previously occupied before the introduction of the exotic pathogen may be possible.

The best studies on the evolutionary ecology of exotic pathogens involve the introduction of white pine blister rust *C. ribicola* into North America around 1900, since when it has affected a wide variety of North American five needles pines (Kinloch, 2003). These pines are highly susceptible to the disease and can only be grown commercially on low-hazard sites. In two of the species, sugar pine, *P. lambertiana,* and western white pine, *P. monticola*, individuals possessing R genes (Cr1 and Cr2) were discovered that led to hypersensitive response on infection, and early elimination of the pathogen (Kinloch and Littlefield, 1977; Kinloch *et al*., 1999). These genes are at frequencies of ~0.001 within populations and are geographically localized. In the first wave of infection Cr1 and Cr2, individuals within these natural populations were not affected. However, within a period of 20 years, evolution of corresponding avr1 and avr2 alleles had occurred in the *C. ribicola* population, leading to infection and death of the trees carrying the Cr genes (Kinloch and Comstock, 1981; Kinloch *et al*., 2003; Kinloch *et al*., 2004).

Individuals with elevated and heritable quantitative resistance to *C. ribicola* have also been detected and selected from natural populations of both *P. lambertiana* and *P. monticola* (King *et al*., 2010). In field trials, these genotypes showed significant reductions in disease damage, doubling their probability of survival, and this resistance was stable over time (Sniezko and Kegley, 2003; Kinloch *et al*., 2008). Seed sources with increased quantitative resistance in conjunction with Cr genes are currently being used in commercial planting in order to promote the recovery of five needle pines as commercial species. Operational problems remain with effective deployment of these scarce genotypes to high hazard sites, and with ensuring that the resistance genotypes deployed are selected from within appropriate locally adapted seed sources for planting throughout the natural distributions of the species (discussed in King *et al*., 2010). A similar programme involving intensive artificial selection for quantitative resistance is currently being used to generate a resistant population of Port Orford cedar, *Chamaecyparis lawsoniana*, which can be deployed to mitigate the effects of the introduced pathogen *P. lateralis* in the western US (Hansen *et al*., 2000; Oh *et al*., 2006).

These examples demonstrate that if suitable quantitative genetic variation for resistance is available that is not based

solely on R genes, recovery from the effects of exotic forest pathogens can occur naturally provided that some regeneration of trees remains in low-hazard sites. However, this is a process that is likely to take many tree generations, and during the period of recovery, species distribution will be dramatically reduced and the species will run the risk of extinction. Natural selection of the very low proportion of genotypes showing high quantitative resistance may be insufficient to allow recovery of populations over short time-scales, and intensive artificial selection programmes may be necessary in order to generate populations with quantitative resistance that can be used to accelerate the recovery of populations.

In selecting for quantitative resistance, it will be essential to establish its genetic basis and ensure that it is not determined by R gene variation (Samils *et al*., 2011). Given the expected low heritability of quantitative resistance in natural populations, it will also be important to conduct selection under strictly controlled environmental conditions. Populations selected for greater quantitative resistance also need to be locally adapted to the site where they are used to ensure that they do not suffer stress and additional pathogen problems when out-planted. Recovery programmes deploying naturally occurring quantitative resistance in this way have been advocated as an appropriate response to combat the present ash dieback outbreak (McKinney *et al*., 2014).

Conclusions

The review presented above is an attempt to understand forest diseases from the perspective of the evolutionary ecology of trees and their pathogens. In terms of practical forestry, what can this analysis tell us about the disease threats to our forests and the resilience of forests to pathogens? A strong conclusion from the review is that rapid anthropogenic climate change is likely to lead to increased disease problems in forests caused both by greater pathogen pressure and by elevated stress resulting from loss of environmental adaptation. The possession of appropriate genetic variation for resistance and climate adaptation will therefore be crucial for ensuring the resilience of forests in the face of climate change (Telford *et al*., 2015). Adaptive escape of individual tree species is most likely where populations are large and genetically diverse and have access via gene flow to appropriate genetic variants present in populations elsewhere within the species' distribution (Cavers and Cottrell, 2015). Forest management policies should ensure that future forests are composed of tree populations that possess these attributes.

The second point to emerge from this review is that a number of current management practices lead to forests that have reduced resilience to pathogens. Indeed, many current forest disease problems are of our own making. Management practices that exacerbate disease problems include the growth of clonal forests, creation of single-species plantations from species normally found at much lower density and extensive off-site planting of commercially desirable species. In order to ensure that disease levels are kept to acceptable levels in these managed populations, without the need for external inputs, it is likely that appropriate resistance breeding programmes will be required (Lannou, 2012; Burdon *et al*., 2014). Thus, in clonal forests, it may be necessary to introduce genetic variation at R loci, and possibly loci associated with quantitative resistance to prevent the evolution of highly aggressive clonally adapted pathogen genotypes. In single-species plantations, or plantations in high hazard sites, concerted selection programmes for greater quantitative resistance to pathogens may need to be implemented. This requirement for greater investment in resistance breeding, as the intensity of management increases, is a theme common to all crop populations.

However, the most important point to take home from this review is that the greatest threat to the resilience of forest trees is that posed by exotic pathogens derived from closely related exotic tree species. The opportunity for adaptive escape of native trees from exotic pathogens is far more limited than is the case for indigenous pathogens. It is likely to involve far greater 'selective death' of the host, and there is likely to be a very prolonged period in which trees are confined to small refugial populations that are vulnerable to extinction. Moreover, if the same species is subject to multiple exotic pathogens, the size of refugial populations may be insufficient to allow adaptive escape from the second or any subsequent threats. Priority must therefore be given to regulating the global plant trade and other practices responsible for the introduction of exotic pathogens, if the cumulative losses of forests and forest biodiversity currently exemplified by the spread of *P. ramorum* and *H. pseudoalbidus* are to be halted (Brasier, 2008; Grunwald *et al*., 2012; Gross *et al*., 2014).

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