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Can we protect forests by harnessing variation in resistance to pests and pathogens?

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Our natural and commercially planted forests are currently facing an unprecedented threat from pests and pathogens. On the principle that 'prevention is better than cure', the policies and practices that influence forest management must aim to prevent epidemics rather than to fight them once they are established. Vigilance and strict security at national borders aim to prevent entry of pests and pathogens but experience shows us that this does not achieve total exclusion. Consequently, to improve the long-term resistance and resilience of tree populations to infection or herbivory, a more realistic and scientific approach may be to understand and use the resistance mechanisms that are naturally present in trees. Resistance trait variation may be genetically controlled and heritable. Populations therefore have the potential to respond to the selective pressure imposed by attack and, if the management and environmental conditions are right, adapt. This review outlines the mechanisms that trees use to defend themselves, the genetic and environmental control of these mechanisms, the subsequent phenotypic variation that we observe and how best to measure and use this to develop and maintain resilient tree populations. In order to ensure a more sustainable and stable future for commercial and native tree species there is a need to incorporate these approaches into forest management globally through collaboration between foresters and scientists and increased investment in relevant research trials.

Introduction

It is estimated that non-native pests and pathogens cost the UK forestry sector over £5 million annually through loss of production alone (Williams *et al*., 2010) although it is acknowledged that this is probably an underestimate. This figure also does not include the additional loss of income due to damage caused by native pests and pathogens or those of unknown origin, the expense incurred in research, changes in management practices or large-scale removal of affected trees needed to minimize the damage caused by these organisms. The environmental and recreational impact of forest loss are difficult to quantify accurately, although one attempt to do so arrived at an estimate of £1 billion in Britain (Willis *et al*., 2003). Loss of ecosystem services that provide environmental and recreational benefits is likely to be particularly acute in Britain where ancient and semi-natural woodland cover only 1–2 per cent of the country (Forestry Commission, 1994; Woodland Trust, 2000), and woods are important locations for recreation and leisure pursuits. Historically our forests have always faced threats from pest or pathogen attack, but it is anticipated that this will worsen with changing climate (Sturrock *et al*., 2011) and land-use patterns, and the global movement of pests and pathogens into new territories (Parker and Gilbert, 2004; McKinney *et al*., 2011).

A key element of successfully managing forests for sustainable resilience to invading pests and pathogens is to harness the genetically controlled resistance mechanisms that are naturally present in trees (Ledig, 1988; Cavers and Cottrell, 2015), as part of an integrated management strategy. This review recommends approaching this task by first identifying and understanding several key components of the interaction between the tree, the pest/pathogen and the environment. These include recognizing specific mechanisms of resistance that a tree uses in defence of biotic threats, the genetic and environmental control of resistance variation in trees and consideration of phenotypic variation in resistance and the relative importance of genetic and environmental conditions. A series of key pathosystems, including both conifer and deciduous systems, are used to provide context on how genetically controlled and heritable resistance traits have been exploited in forest management to mitigate the threat of pests and pathogens. A companion paper (Ennos, 2015) in this Special Issue provides a more detailed account of the resistance variation and damage caused by pathogens in an ecological and evolutionary context.

Mechanisms of resistance

Trees tolerate, recover from or resist pests and pathogens at either cellular, tissue or whole tree levels (Namkoong, 1991). Resistance

here is defined as the ability of an individual host tree to use genetically encoded mechanisms to defend against or withstand attack by an invading organism (Figure 1), with an associated and measurable increase in fitness compared with hosts who do not employ these mechanisms. The resilience of populations, their ability to endure and recover over time, is a complementary and distinct concept. Mechanisms which confer resistance may be encoded in the genome of the tree, be under environmental control or may result from an interaction between the genotype and the environment (g × e). It is important, although not easy, to determine the relative importance of genetic, environmental and g × e control of resistance variation, as it varies depending on the host species, the pest or pathosystem, the environment, the specific population in question, and the temporal and spatial context in which it is assessed.

Plants employ a range of phenological, morphological and physiological mechanisms to reduce damage by herbivores and pathogens (Carson and Carson, 1989). These mechanisms can be both passive (spatial and temporal avoidance of threats, tolerance to infection or herbivory) and active (confrontation through interactive resistance mechanisms which slow or prevent infection or attack) (Burdon, 1987; Kennedy and Barbour, 1992). Active resistance mechanisms exist in various forms which include mechanical or structural barriers, the production of toxic or antimicrobial chemicals or proteins, programmed cell death, the attraction of predators which target the pest, the reallocation of resources to unaffected regions of the plant, and compensatory increases in growth or reproduction (Burdon, 2001; Gilbert, 2002; McDowell and Woffenden, 2003; Eyles *et al*., 2010; Kloth *et al*., 2012). It is likely that plants use a combination of these mechanisms in a coordinated and integrated response (Bonello *et al*., 2006). Most mechanisms do not provide complete resistance but instead reduce the success of the pest and pathogens (Poland *et al*., 2011).

The range of resistance mechanisms employed by hosts in defence is exemplified by the responses of both white spruce (*Picea glauca* (Moench) Voss) and Douglas-fir (*Pseudotsuga menziesii* (Mirb.) Franco) to eastern and western spruce budworm (*Choristoneura* spp.) respectively. Resistant phenotypes are more likely to have high concentrations of sugar (Clancy, 1992), phenolic compounds (Delvas *et al*., 2011; Despland *et al*., 2011) and monoterpenes (Chen *et al*., 2002) in their needles, a fast growth rate and late budburst (resulting in a phenological mismatch with the pest) (Chen *et al*., 2001, 2003), and a thicker epicuticular wax layer (Daoust *et al*., 2010). Transcriptome sequencing has also revealed the regulation of genes during budworm attack, in particular significant upregulation of the genes involved in octadeconoid, terpenoid and phenylpropanoid biosynthesis pathways (Ralph *et al*., 2006). Despite this wealth of information on mechanisms of resistance, there appears to be very little, if any, progress concerning our understanding of other aspects of this association, for example, the heritability of variation for host resistance traits.

Despite its importance, our knowledge of mechanisms of resistance is restricted to a few species. This is in part because identifying heritable resistance traits for use in breeding programmes can be conducted without identifying resistance mechanisms, and the two are therefore often undertaken in parallel but separately (Smalley and Guries, 1993). It may even be argued that elucidating precise resistance mechanisms employed by trees is secondary to discovering which trees are genetically most resistant, and exploiting this genetic variation in breeding programs. However, identifying the mechanisms behind the response may reveal the route and method of infection/herbivory and vice versa. Furthermore, an understanding of mechanisms of resistance may reveal phenotypic markers that can be measured and used to predict a tree's response to attack by pests or pathogens. For instance constitutively higher levels of phenolics such as ellagic acid, a fungistatic compound, in coast live oak (*Quercus agrifolia* Nee.) have been associated with resistance to *Phytophthora ramorum* (Werres *et al*., 2001; Nagle *et al*., 2011; McPherson *et al*., 2014). Induction of defence compounds, particularly terpenes, has also been strongly correlated with the inhibition of bark beetle colonization in Norway spruce (*Picea abies* (L.) H. Karst) (Zhao *et al*., 2011; Schiebe *et al*., 2012).

Defence mechanisms which deter initial herbivory/infection or affect insect/pathogen performance (Figure 1) may be either constitutive and therefore always present, or induced following recognition of a threat (Kloth *et al*., 2012). Constitutive defences often rely on the presence of toxic chemicals or anatomical structures which deter initial herbivory or infection (Bonello *et al*., 2006). This strategy allows the tree to prevent or inhibit invasion, then kill or isolate the pest or pathogen and repair any damage that has been caused (Franceschi *et al*., 2005). If this first line of defence is overcome, induced defences may be invoked (Figure 1). These are dependent on constitutive or basal (induced through direct recognition of a pathogen) defences for the recognition and induction of a response (van Loon, 1997) and may be either a general or a pathogen or pest-specific response (Franceschi *et al*., 2005). Induced defences may operate locally at the site of infection, or systemically throughout the entire plant via signalling and transportation of defence compounds to other tissues (Eyles

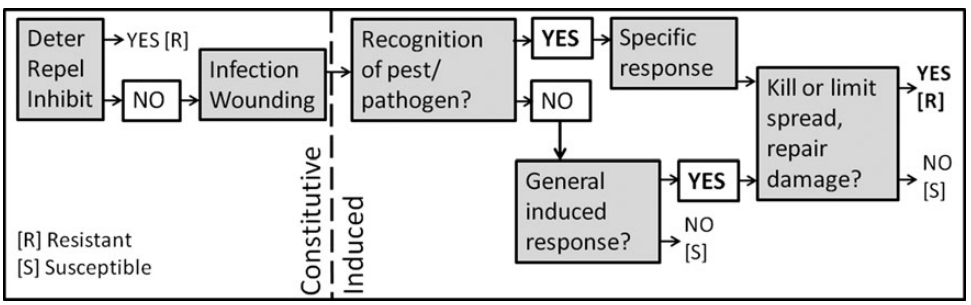

Figure 1 Process of tree response (shaded) to infection or attack, and the resultant phenotype (resistant or susceptible). Variation in the strength of the response leads to variation in the phenotype.

et al., 2010). Eyles *et al*. (2010) recognized the existence in plants of seven types of systemic induced resistance, which vary according to the inducing agent. Induced defences may also prime the host and protect it against future challenges (Bonello *et al*., 2006; Pastor *et al*., 2013). This phenomenon is poorly understood, although it is thought to result in a generally heightened defence response, rather than a pathogen-specific response to the original inducing agent (van Loon, 1997).

Priming can be considered a key element of induced resistance (Pastor *et al*., 2013), and recent work (predominantly in *Arabidopsis thaliana* (L.) Heynh, reviewed by Conrath (2011) has started to reveal its molecular basis. In contrast to animals, plants do not possess an immune system and therefore induced defences are particularly important, as they allow plants to respond to threats when they are present rather than having constantly to invest in metabolically expensive defences. It is generally believed that the metabolic costs of primed defences are less than those of other induced defences (van Hulten *et al*., 2006), which are less than those of constitutive defences (when considering resource allocation) (Franceschi *et al*., 2005). However most of this work has been done on short-lived perennials and may therefore not apply to trees (Eyles *et al*., 2010).

There are many limitations to investigating mechanisms of resistance in trees which contribute to the current scarcity of detailed studies. These include the anatomical complexity of trees in comparison to crop species, the practical difficulties of working with organisms of large size, long lifespan and long generation times, and the potential for long-lived trees to show different responses at different developmental stages (Fenning, 2006). This means that most of the research into plant resistance mechanisms has been undertaken in short-lived model plants. It is therefore important to recognize that there are features of the defence mechanisms of trees that are additional to and distinct from those found in short-lived model plants.

An example of a defence tissue unique to trees and shrubs is bark, which comprises the major constitutive barrier to pest and pathogen invasion. A comprehensive review by Franceschi *et al*. (2005) describes the various mechanisms by which conifer bark protects the tree against pests, including production of antifungal tannins, resin synthesis and storage structures, and layers of dead cells which provide a barrier to invasion. The anatomical complexity of woody tissue in trees also enables them to defend themselves through the process of compartmentalization (Shigo, 1984). This relies on a single tree effectively acting as a series of perennial plants, with each growth ring forming a 'new' tree compartment which envelopes the last. Compartmentalization, in addition to strengthening the structure of the tree, also serves to isolate damage and restrict the spread of pests and pathogens, as demonstrated by analysis of patterns of pathogen spread and containment within the invaded woody tissues of trees (Shigo, 1984). Modification of many structures which confer resistance, both constitutive and induced, contributes to this process of compartmentalization. These include alterations to xylem vessel size, resin canals and structural changes such as wound callus formation (Shigo, 1984).

Genetic control of variation in disease resistance traits

Variation in host disease resistance traits is genetically controlled by a range of genes whose effects may be additive, dominant, heterotic or epistatic (Young, 1996). These genes may have evolved specifically to defend the plant against threats, or they may control differences in growth, phenology and metabolism that result in differential susceptibility of the host (Namkoong, 1991).

Differences between individuals in terms of the genetic control of defence mechanisms may be due to variation in single or multiple genes, although these are not necessarily completely distinct (Poland *et al*., 2009). The former is often referred to as complete, major-gene, R-gene mediated, vertical or qualitative disease resistance. The latter is known variously as incomplete, polygenic, horizontal or quantitative disease resistance (Burdon, 1987). For the purposes of this review, the terms major-gene resistance and polygenic resistance are used.

Induced defence is initiated following recognition of a threat and is mediated by a major-gene known as a resistance (R) gene, which responds to a restricted set of pathogens (McDowell and Woffenden, 2003) and provides a high degree of resistance. Major-gene disease tolerance (maintaining fitness despite infection) has also been demonstrated in *A. thaliana* to bacterial wilt (*Ralstonia solanacearum*, van der Linden *et al*., 2013), raising the possibility that although outwardly resistant, plants carrying these major-genes would in fact sustain pathogen populations in large numbers. The consequences of maintaining a reservoir of pathogens may range from the benign to extremely severe, depending on the adaptive potential of the pathogen, and the proximity and number of host species. In infected plant cells R-proteins induce a response through recognition of the pathogen, either directly or indirectly (Bent and Mackey, 2007). R-genes may directly recognize the products of avirulence (*Avr*) genes produced by the pathogen, or indirectly recognize modifications to host defence systems brought about by the products of Avr genes (Guard R genes, Jones and Dangl, 2006). Following recognition of the pathogen the R protein initiates a defence response in the host (McDowell and Woffenden, 2003). The signalling pathways involved are complex; they are thought to be nonlinear and linked with both positive and negative feedback loops (Eyles *et al*., 2010). The presence of a single dominant R-gene is often easy to identify in crop plants as progeny segregate into resistant and susceptible phenotypes (Fang *et al*., 2010). The discovery and use of major-gene resistance has therefore been common in agricultural crops, where domestication has involved backcrossing to wild varieties or cultivars with observable resistance to a pest or pathogen. Major-gene mediated resistance to pests and disease is generally considered to be qualitative for this reason. However in natural systems and in tree species, which are more complex, resistance variation may often appear to be quantitative due to the interaction of many induced defences, variation in the genetics and biology of the threat and climatic conditions.

A frequently cited example of major-gene resistance in trees is found in native North American white pine species (Kinloch *et al*., 1970). White pines have been significantly affected by the causative agent of white pine blister rust (WPBR), *Cronartium ribicola* J.C. Fisch, since the pathogen was accidently introduced into North America in the early 1900s (Kinloch, 2003). Early assessments of stands in the 1930s by A.J. Riker found very low numbers of WPBR resistant eastern white pines (*Pinus strobus* L.) (0.25 per cent) (David *et al*., 2011). One mechanism of resistance is due to an R gene controlling hypersensitive response (HR) in

needles (leading to the premature shedding of needles) (Sniezko, 2006; Kinloch *et al*., 2011).

In contrast to major-gene variation, polygenic resistance variation is due to the integrated action of multiple genes each contributing a small effect to a defence response. This type of resistance is usually associated with genes that affect the strength or efficacy of the resistance response, rather than those that recognize a specific threat (R-genes). As a result of the number of genes involved, a continuous distribution of disease resistance phenotypes is observed (Quesada *et al*., 2010). Polygenic resistance has long been attributed to quantitative responses to diseases and pests. However, the identification of mechanisms and the specific genes that underlie them is challenging; each QTL (quantitative trait locus) that contributes to resistance variation is unlikely to be individually distinguishable (Poland *et al*., 2011).

Poland *et al*. (2009) put forward six hypotheses regarding mechanisms whose variation would give rise to differences in quantitative resistance, although it is probable that all of these are involved to some extent: (1) combined effects of genes which contribute to development and morphology; (2) genes involved in neutralization of toxins produced by the pathogen; (3) elements contributing to signal transduction during an attack; (4) allelic variants of R genes; (5) as yet unidentified mechanisms; (6) variants of basal defence genes.

In the white pine species mentioned earlier, quantitative variation in disease resistance is associated with genetically determined variation in the strength of defence reactions in the bark and the ability to inactivate cankers (Sniezko, 2006). Additional information on mechanisms of polygenic resistance comes from analysis of needle morphology demonstrating that the stomata of more susceptible phenotypes are significantly wider, rounder and have a greater area than stomata of resistant phenotypes (Woo *et al*., 2001). Genetically susceptible phenotypes may therefore allow easier pathogen access to the vulnerable internal regions of the needle. At the molecular level real-time PCR has demonstrated that more resistant seedlings up-regulate genes earlier than susceptible seedlings, and comparative proteomic profiles have shown that differential expression and more active synthesis of proteins in resistant seedlings contribute to a faster, more effective response to infection (Zamany *et al*., 2012).

Another example where quantitative resistance to an important tree pathogen has been studied is for the host *Pinus radiata* D. Don attacked by *Dothistroma* needle blight (DNB) (*Dothistroma septosporum* (Dorog) Morelet). The disease causes defoliation of trees that leads to long-term reduction in timber yield and occasionally tree death (Brown *et al*., 2012). Polygenic variation in resistance to DNB occurs with narrow-sense heritability estimates ranging from 0.18 to 0.51 (Wilcox, 1982; Carson and Carson, 1989; Chambers *et al*., 2000; Jayawickrama, 2001; Devey *et al*., 2004; Ivković *et al*., 2010). Quantitative trait loci (QTL) for resistance to DNB have also been found in *P. radiata* (Devey *et al*., 2004). Breeding programmes in New Zealand and Australia that exploit this genetic variation have achieved an average reduction in defoliation of 12 per cent after one generation of artificial selection (Carson, 1989).

Although traits that confer increased resistance are likely to contribute to an improved fitness of a host in the presence of corresponding threats, there are also associated costs (Parker and Gilbert, 2004). These costs may result directly from the metabolic investment in the production of resistance proteins, indirectly from the production of induced defence responses even at basal levels, or involve the reaction to environmental signals which trigger responses in the absence of a threat (Tian *et al*., 2003). Alternatively, they may be a result of an overall reduction of fitness due to the covariance of resistance traits with other traits such as an altered growth form (Burdon, 2001). However, the cost of resistance is considered to be small according to a multilocus model developed by Frank (1993) although sufficient to reduce host fitness in the absence of disease or infection. These costs act to maintain diversity of resistance alleles as fixation of such alleles is less likely in the absence of consistently strong selection pressure. Without this resistance associated cost (in combination with virulence in pathogens) resistance alleles would continuously reach fixation in plant populations (Tian *et al*., 2003), resulting in plants with universal resistance (Parker and Gilbert, 2004). Those organisms with resistance alleles which do reach fixation may also subsequently be targeted by specialized pests or pathogens in a co-evolutionary arms race (Ennos, 2015).

The contribution of heritable resistance traits in protecting our natural forests and plantations from pests and pathogens depends on the durability of the trait. Durability is affected by multiple factors which include heritability of the trait: climate, the genetic diversity (Hirst *et al*., 1999) and the reproductive and dispersal mechanisms (Carson and Carson, 1989; Frank, 1993) of both host and pest/pathogen, and the genetic basis for resistance (major-gene or polygenic-mediated resistance) (McDonald and Linde, 2002). Polygenic resistance is likely to remain stable due to the complexity of mechanisms controlled by multiple genes (Lindhout, 2002), whereas pest or pathogen-specific R-gene mediated defence can be defeated by pathogens through the loss or modification of *Avr* genes (Poland *et al*., 2009).

An example of the comparative durability of both major-gene and polygenic resistance is provided by the white pine blister rust pathosystem. The major-gene (*Cr1*) in sugar pine (*Pinus lambertiana* Douglas) is responsible for mediating resistance to *C. ribicola* via a rapid HR. It has a counterpart in the pathogen, the virulence genotype *vcr1* that is able to infect the Cr1 host genotype (Kinloch and Dupper, 2002). Pathogens sampled from a plantation where a high proportion of trees carrying the *Cr1* gene were present, themselves possessed the *vcr1* genotype at high frequency. The major-gene resistance conferred by the Cr1 gene had not proved to be durable (Richardson *et al*., 2008). In contrast, pathogens sampled from populations of trees originally selected for polygenic resistance traits maintained a high genetic diversity (Richardson *et al*., 2008). The virulent genotype in the pathogen population is rapidly selected for in a forest where a single major-gene resistance is the primary defence.

In general, tree breeding programmes are expected to move away from traditional techniques (phenotypic selection) to genomic selection in the near future (Grattapaglia and Resende, 2010), and disease resistance will undoubtedly be a key trait of interest. However the need to measure phenotypic variation in resistance (which will then be associated with markers for future selection), will remain, and evaluating the heritability and durability of particular resistance traits will still be highly relevant. Our understanding of genetic control of variation in disease resistance and associated complexities will, however, certainly increase.

The role of environmental variation on disease resistance traits

The phenotype of an individual is the product of both its genetic composition and the effect of the environment in which it is grown. This environment effect acts over spatial as well as temporal scales. When multiple copies of a single genotype are exposed to different environments following planting in different locations it is here referred to as spatial environmental variation (SEV). Temporal environmental variation (TEV) refers to the variation over time that a single genotype experiences at its planting location. SEV tends to have a greater impact on plantations where trees are planted outside their native range. In contrast, TEV affects both natural forests and commercial plantations, and is expected to increase in a changing climate. The magnitude of the scale over which SEV operates can be explored via clonal relocation and reciprocal transplant trials. These trials identify the extent to which individual genotypes vary in their phenotype in different environments, and how much of the observed phenotype is attributable to its genetic composition (local adaptation) or to the environment in which it is grown (phenotypic plasticity).

Although similar, TEV differs from SEV in two fundamental aspects. In the former: (1) the change in the environment is usually temporary; (2) the change may occur at any stage of development (transplantation leading to SEV usually occurs when the tree is young). During their lifetimes trees experience a range of extreme environmental events, and although they have developed a range of strategies to cope, and even thrive, during these events, stress is inevitably part of this process. When a tree's resources are depleted or diverted, for example, following extreme climatic events such as fire or drought, it may no longer be able to meet the metabolic costs associated with resistance (Figure 2) and as a consequence may become more susceptible to infection and predation (Namkoong, 1991). Coincidentally the changing climate may also be more conducive to pests and pathogens and may even increase their diversity by allowing migration to higher latitudes and altitudes (Woods, 2011). Although changes in insect populations can be modelled through changes in temperature, efforts to predict alterations in pathogen populations are hampered by the difficulty of forecasting changes in precipitation (Woods, 2003), which is often the limiting factor in their growth and reproduction.

In addition to directly affecting a forest's resistance to disease and herbivory, environmental instability can also have indirect effects through reduction and fragmentation of tree populations (Figure 2). Forests that have become fragmented or smaller in size will also tend to have a reduced genetic diversity (King and Lively, 2012), and consequently lower frequency of and low variation in resistance alleles. This would reduce the variation in resistance traits expressed in the population, and may also mean that the population is unable to protect itself against new threats. Increased susceptibility is exacerbated by stress (Figure 2). If favourable conditions return or there are additional features such as a highly effective resistance response, the vicious cycle (which ends with the extinction of the tree population) may be broken (see examples in Figure 2).

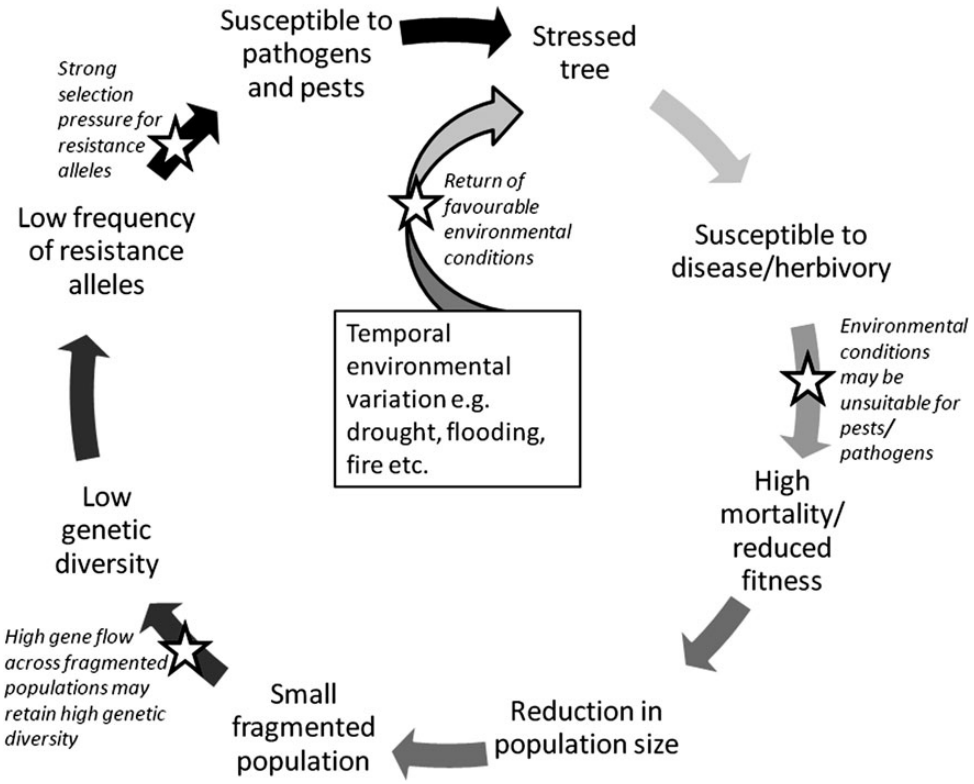

Figure 2 The effects of temporal environmental variation on the susceptibility of tree populations to pests and pathogens. Examples where the vicious cycle may be broken are indicated by stars.

Given the speed with which the climate and the environment are changing, it is not surprising that consequences such as changing pest and pathogen ranges, host shifts and catastrophic epidemics within existing pathosystems are now a threat. *Dothistroma* needle blight, predominantly thought of as an exotic pathosystem, also causes epidemics in native forests of lodgepole pine (*Pinus contorta* Douglas) in BC, where it is an endemic pathogen. Reports across Europe from both commercial and native pine forests indicate it is now widespread and causing severe damage (Barnes *et al*., 2008; Drenkhan and Hanso, 2009; Muller *et al*., 2009; Watt *et al*., 2009; Brown *et al*., 2012). In both cases it seems likely that climate change has played a role (Woods *et al*., 2005; Welsh *et al*., 2014). In addition to a more favourable climate, DNB also benefits from the increasing availability of alternative hosts which are commercially planted in large numbers at high density. In natural populations the host trees are usually at lower density which reduces the spread of DNB; by allowing build-up of pathogen populations, commercial planting densities may act to increase inoculum load, so that cross-over to natural populations is far more likely.

Resistance can also, under certain circumstances, be enhanced by interaction with the environment. Just as a host which would usually be considered resistant may become susceptible when under stress from extreme environmental events, a susceptible host can also appear resistant if the environment particularly favours the host, while being suboptimal for the pest or pathogen. For example, outbreaks of mountain pine beetle (*Dendroctonus ponderosae* Hopkins) are rarely recorded in whitebark pine (*Pinus albicaulis* Engelm.), primarily because the high-elevation environment in which these pines are found is inhospitable to the beetles (Logan and Powell, 2001). Whitebark pines are now under threat as this effective form of 'resistance' is being lost due to the warming climate and the subsequent expansion of the mountain pine beetles' range into new habitats (Logan *et al*., 2010).

In addition to environmentally induced resistance variation (Smalley and Guries, 1993) and the various types of heritable and non-heritable variation in resistance, there is also evidence of ontogenetic resistance (Ekramoddoullah and Hunt, 2002; Solla *et al*., 2005), associational resistance and maternally transmitted resistance (Gilbert, 2002). Ontogenetic (age-related) resistance has been reported in many plant species, including elm trees (*Ulmus* spp.) to Dutch elm disease (*Ophiostoma novo-ulmi* and *O. Ulmi*, Heybroek, 1957), *P. radiata* to *Dothistroma* needle blight (Bulman *et al*., 2004) and apple trees (*Malus* × *domestica* Borkh.) to apple scab (*Venturia inaequalis*) (Gusberti *et al*., 2013). Whether ontogenetic resistance is a result of developmental changes in the host, or the build up of induced defences resulting in effective resistance, is not yet known (Bonello *et al*., 2006). The phenomenon of associational resistance (or susceptibility) has been reviewed by Barbosa *et al*. (2009) and focuses on the likelihood of a herbivore being attracted to, or repelled by, a particular plant, as a direct result of its neighbours. Maternally transmitted resistance is a relatively recently discovered phenomenon mediated by epigenetic mechanisms (Luna and Ton, 2012) whereby resistance induced in the mother plant is enhanced in seedlings which have not been challenged (recently reviewed by Holeski *et al*. 2012). Another form of resistance is endophyte-mediated induced resistance, where seedlings with endophytes (non-disease-causing fungi found within the tissue of host trees) exhibit reduced disease severity as compared with endophyte-free seedlings, (Ganley *et al*., 2008; Eyles *et al*., 2010). Endophytes may directly compete with other microorganisms (Arnold *et al*., 2003), thereby conferring resistance, or they may act to 'prime' the tree by inducing systemic immunity (Conrath, 2011). These different recognized types of resistance make separating the environmental and genetic components of variation in resistance traits difficult, and it is important at each stage to consider the specific mechanisms of resistance, the genes underlying these mechanisms and the interaction of other organisms and the environment (Iason *et al*., forthcoming).

Phenotypic variation in tree populations

For a host to be classified as resistant there must be a contrasting host which is considered susceptible. In order to be meaningful, the description 'resistant' must be set in the context of a scale of variation in host response to a particular pest or pathogen. The huge range of defence mechanisms, combined with other factors such as environment and pathotype which influence the interaction, mean that variation in the response of individuals in a population of plants to infection and herbivory is common, especially when the interaction is endemic (Wilcox *et al*., 1996). Responses may range from pest/pathogen-associated mortality, to complete resistance with no discernible impact to health (Burdon, 1987).

One of the most important first steps in estimating the actual or potential threat of pests or pathogens to forests is to evaluate the extent of resistance within these populations, ideally through inoculation in controlled conditions (Ekramoddoullah and Hunt, 2002) over a long period of time (Solla *et al*., 2005). Precise and consistent phenotyping is often the limiting factor with this research as it is time consuming and expensive (Myles *et al*., 2009), but it is vital in order to ensure that the information that is collected is accurate and representative (Ingvarsson and Street, 2011). The ecological, economic and aesthetic impacts of disease (Carson and Carson, 1989) must also be considered. Best practice demands both growth chamber/glasshouse and field trials, as the genetic resistance of the host may be either over- or under-estimated depending on the conditions at the time (Smalley and Guries, 1993). Trials established in glasshouses or growth chambers are useful as conditions can be controlled, however, there are inevitable difficulties with extrapolating disease severity data to the field. Complicating factors include the age of the plants used (ontogenetic resistance may be an issue), the pathotypes available (which will almost certainly not reflect the diversity present in the field) and the climatic conditions. Although whole-plant inoculation is preferable, detached-leaf assays are often performed where space or facilities are limited. While useful information can be obtained with this approach, it does not always mimic field symptoms in some species (P. Gadgil, personal communication). Artificial inoculation is usually developed as a tool to predict host resistance response (Kabir *et al*., 2013), but it can also be used to identify other at-risk species in the range (Hansen *et al*., 2005). An important consideration is also the correlation between disease severity in seedlings, and that in mature trees. Data on this are difficult to obtain due to the long time periods involved, but there is evidence that coastal Douglas-fir seedlings exhibiting tolerance (such as higher needle retention) to Swiss needle cast (*Phaeocryptopus gaeumannii* (Rohde) Petrak) are more tolerant to infection in the field when mature (Temel *et al*., 2005). The space and time constraints involved in doing glasshouse trials using tree species, however, mean that there are simply not as many studies as there have

been in herbaceous plants. Greater collaboration between scientists and foresters in establishing appropriately designed trials, long-term field trials, or in granting access to existing forests as study systems, would ensure efficient use of available resources and expertise.

The range of definitions for 'resistance' in different populations must also be taken into account. Where the impacts of pests or pathogens are particularly severe, a resistant individual may be defined as any tree which survives infection or herbivory, such as American elms in response to Dutch elm disease (Smalley and Guries, 1993). In contrast, *P. radiata* trees have been classed as resistant to *Dothistroma* needle blight if defoliation is <10 per cent (Wilcox, 1982). It is possible that these situations arise as a result of either major-gene or polygenic-mediated disease resistance, respectively (Quesada *et al*., 2010), although even in systems where major-gene resistance has been found, there can still be variation in disease symptoms (Wilcox *et al*., 1996). In cases where the distribution of damage to the tree is continuous, the tail-ends of the distribution of tree response to infection or predation are usually considered resistant and susceptible. In some circumstances, resistance may refer to the persistence of an entire population rather than the characteristics of one individual (Burdon, 2001). In this case resistance is only possible when infection or herbivory is restricted to a proportion of the population. Genetically diverse hosts which possess a range of resistance alleles protect against new threats by reducing the inoculum load and cross infection (Carson and Carson, 1989). Reflecting the fact that the response of trees to disease and pests is nearly always quantitative, the resistance trait should be recorded using a continuous scale.

The extent to which the variation in these resistance mechanisms is genetically encoded, and the degree of influence that external factors such as the environment have on expression will affect the heritability of resistance traits. For a phenotypic resistance trait to be most 'useful' in protecting forests either through adaptive change in natural populations or through breeding, variation in the resistance trait must also be heritable (McKinney *et al*., 2011). Assessments of heritability of disease resistance can be obtained through progeny trials. In forest trees, narrow-sense heritability (additive genetic variance which contributes to phenotypic variance, Brookfield, 2012) has rarely been found to be >0.3 (Carson and Carson, 1989), although heritability will vary depending on the environment in which the measurements are made.

The importance of estimating heritability of resistance variation to ascertain its potential for controlling disease is demonstrated by studies of ash-dieback involving an interaction between *Fraxinus excelsior* L. and the ascomycete fungus *Chalara fraxinea*, Kowalski and Holdenrieder, 2009). Evidence of variation in susceptibility of ash to this disease in Denmark (McKinney *et al*., 2011; Kjaer *et al*., 2012), Sweden (Stener, 2013) and Lithuania (Pliura *et al*., 2011) indicates low levels of variation in genetic resistance mechanisms (McKinney *et al*., 2011; Kjaer *et al*., 2012; Stener, 2013), but that variation in these resistance traits is under strong genetic control. Quoted values of narrow-sense heritability of resistance variation are 0.37–0.52 (Kjaer *et al*., 2012) and 0.40–0.49 (Pliura *et al*., 2011), with broad sense heritability of 0.25–0.54 (McKinney *et al*., 2011) and 0.07–0.57 (Pliura *et al*., 2011). With such high heritability values it is hoped that, despite low natural levels of variation in resistance, these resistance traits can be incorporated into breeding programmes to ameliorate some of the potentially devastating effects of this disease. Mortality and infection rates also seem to vary depending on the infection pressure. In Lithuania, where the inoculum load is high, the mortality rate in a trial of 27 000 trees was 90 per cent five years after planting (Pliura *et al*., 2011), whereas mortality of ash in Sweden over the same time period was only 7 and 33 per cent in two sites, respectively (Stener, 2013). This discrepancy highlights the importance of establishing the context of the disease before predicting the impact.

Resistance to a pest/pathogen can involve several different processes (Figure 1): (1) deterrence, repulsion or inhibition; (2) killing the threat; (3) limiting spread; (4) host repair and recovery (Shigo, 1984; Franceschi *et al*., 2005; Bonello *et al*., 2006; Kloth *et al*., 2012). The deployment and degree of success of each mechanism will affect the resistance phenotype of the host (Figure 1). If (1)–(3) occur quickly in response to a highly virulent pathogen and before much damage has occurred, an individual will be categorized as highly resistant, and susceptible if they do not. If (1)–(3) occur slowly or not at all in response to a pathogen with low virulence, the impact may also be low, and an observer may identify the tree as being resistant with extensive infection and little damage. If (1)–(3) do not occur, but (4) does, then the tree might also be considered resistant, as it will not show the associated reduction in fitness expected of a susceptible tree, although the term tolerance is more commonly applied to this case.

The type of population that is being assessed for resistance to disease or herbivory will also affect the way in which it is measured. Relative yield and/or quality of the product can be measured in crop species, whereas the visualization of symptoms must serve as a proxy for disease severity in natural populations (Kover and Schaal, 2002). In the latter case, it is assumed that symptoms are correlated with a reduction in fitness, which might not always be the case. When considering large, long-lived organisms such as trees it can be difficult to correlate disease symptoms with a reduction in fitness, and the ramifications could be large if the two are not significantly associated. For example, if a species can maintain a high reproductive fitness even in the face of high infection rates, it is likely to survive into the future. Conversely, if a species shows few symptoms of infection, but its mode of reproduction is affected, it may be severely threatened, despite the lack of obvious problems.

Conclusion

Our natural forests and commercial plantations are facing increasing threats from exotic pests and pathogens due to the global movement of people and goods. In addition, environmental instability and extensive planting of exotic host species are driving an increased rate of attack by native pests and pathogens. Variation in response to disease or herbivory within a host population, usually viewed on a sliding scale between resistant and susceptible, results from genes, the environment and an interaction between the two. A genetically diverse population which has co-evolved with a specific pest or pathogen is most likely to be resistant and to have a suite of resistance alleles, which may involve the interaction of many genes, or few genes of large effect. However, changes in the environment can impose stresses on host trees that can render a previously resistant population susceptible, even to native pests or pathogens.

In order to improve resilience of forests to these threats, the degree to which variation in resistance mechanisms are heritable

and durable must be established through appropriately structured growth chamber, glasshouse and field trials. These two properties will affect the extent to which the resistance mechanisms can be used as part of an integrated management strategy to protect the trees in the long term. Collaboration between scientists and foresters in establishing trials would therefore be extremely valuable. Our current understanding in this area is limited, especially when compared with short-lived perennials, and an increased investment is required in the future. As tree breeding programmes gradually progress towards genomic selection, it is likely that a growing body of information available on the genetics underlying variation in disease resistance. However phenotypic studies, measuring the heritability and durability of resistance traits in forest trees, are essential both for identifying and for exploiting the genes affecting resistance, and should therefore be a research priority. In addition, a greater emphasis should be placed on assessing the response of our tree species to a range of pressures in advance of the arrival of new threats. This understanding would provide a better measure of their vulnerability to emerging pests and pathogens, especially following introduction or environmental perturbation. Delaying such efforts until epidemics arise will be too late.

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