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Tree diseases and landscape processes: the challenge of landscape pathology

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Forest pathology inherently involves a landscape perspective, because tree pathogens propagate according to heterogeneous spatial patterns of flow and isolation. Landscape pathology is a field that is now emerging from the transdisciplinary cooperation of forest pathologists with landscape ecologists. Here, we review recent broad-scale assessments of tree disease risk, investigations of site and host preferences for several root rot pathogens, and regional historical analyses of pathogen outbreak in plantations. Crucial topics include fragmentation effects on pathogen spread and geophysical features that predispose forest patches to disease expression. Recent methodological developments facilitate the spatially explicit analysis of reciprocal coarse-scale relationships among hosts and pathogens. Landscape pathology studies fill a significant research gap in the context of our understanding of sustainable forest management, the introduction of exotic organisms and how climate change might affect the spread of disease.

Together with anthropogenic influences, abiotic factors and herbivores, forest pathogens are key to the shaping of the dynamics and diversity of forested landscapes (e.g. [1]). The spread of pathogens and the expression of disease are also influenced by landscape features and by the spatial patterns of vegetation, which includes host, reservoir and resistant species. Not only can landscape features influence patterns of disease development (Figure 1), but diseases can also influence landscape patterns. The presence of yellow-cedar *Chamaecyparis nootkatensis* decline in southeast Alaska caused a 3.8-fold increase in the frequency of landslides as compared to unaffected slopes by reducing the protective effect of cedar trees against erosion [2]. Over the past few decades, many forest pathogen outbreaks have occurred over regional rather than local scales [3]. For these reasons, forest pathology (the study of diseases within tree populations) inherently involves a landscape perspective. The important role of forest pathogens within landscape disturbance processes has been demonstrated in several recent studies, including the analysis of tree mortality in Adirondack Park, New York [4] and the role of laminated root rot, caused by *Phellinus weirii*, in stand replacement processes in Oregon (e.g. [5]). Studies at a larger scale are also important in predicting rates of spread of pathogens and the spatial patterns of their impacts, as shown by studies of sudden oak death in California [6] (Box 1) and beech bark disease in the Catskill Mountains, New York [7].

Although fine-scale studies have often dominated forest pathology, it is now recognized that our understanding (and, ultimately, managing) of diseases on a regional scale requires a broader scope of investigation (e.g. [8]). As a result, there has been a widespread adoption of epidemiological approaches for managing diseases [9,10]. Increasingly, such approaches consider how the spatial structure of host populations influences epidemic spread rates [11]. Therefore, by building on the tradition of plant disease epidemiology [12], many contemporary epidemiological

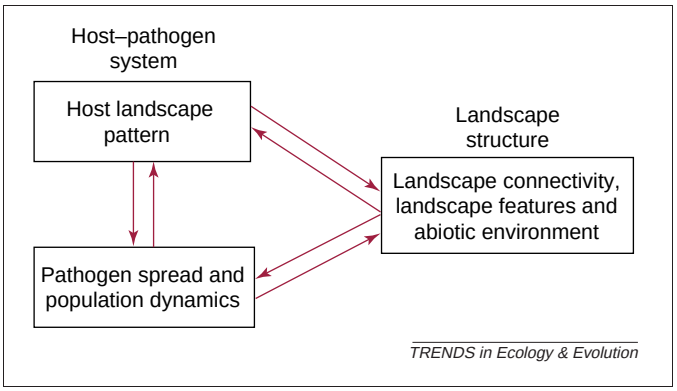

Figure 1. Conceptual model of relationships among host–pathogen systems and landscape structure. Key processes connecting host–pathogen systems with landscape structure include fragmentation effects on pathogens, effects of environmental heterogeneity upon pathogen susceptibility, interactions among pathogens and host landscape pattern, and spatially explicit, coarse-scale pathogen population dynamics. Both host and pathogen populations are influenced by landscape connectivity and spatial heterogeneity in the abiotic environment. The landscape pattern of host species on the landscape is a key determinant for landscape connectivity with respect to the pathogen. Pathogen population dynamics and spread might exert a reciprocal influence upon host landscape pattern through mortality effects.

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Box 1. Landscape pathology case study: sudden oak death

The recent dieback known as sudden oak death (SOD) has reached epidemic proportions along hundreds of kilometers of the Californian coast (Figure I). It occurred following the apparent introduction of *Phytophthora ramorum* into stands of coastal live oak *Quercus agrifolia*, black oak *Q. kelloggii*, and tanoak *Lithocarpus densiflorus* [65]. The pathogen is known elsewhere only from Europe, where it remains rare, except on *Rhododendron* spp. and a *Viburnum* sp. Its rate of spread in California surpasses that of the chestnut blight pathogen *Cryphonectria parasitica* in New England at the beginning of the 20th century [66]. It causes rapid canker development, leading to girdling and 'sudden' death of the tree, and can be spread via sales of infected material throughout the USA [67].

Analyses of this outbreak provide an apt demonstration of the landscape pathology perspective.

- The location and effects of the disease were mapped and monitored with remote sensing imagery at multiple spatial scales [68]. In addition, an interactive SOD monitoring Web-GIS application was developed, enabling the inclusion of ground-based information in the relational database that tracks disease incidence [69].
- The distribution of dead tree crowns (derived from high-resolution imagery) was analyzed with second-order spatial point-pattern analysis techniques to determine whether, and to what extent, mortality is clustered [6]. Clustering patterns between 100 and 300 m were found during both 2001 and 2002, an extent implying that fine-grained assessment of disease or pathogen presence will retain its value also in landscape pathology.
- The occurrence of disease at all slope positions, the above-ground nature of the disease, and biological knowledge about the pathogen – *Phytophthora* species reproduce through both dormant spores [either sexual (oospores) or asexual (chlamydospores)], as well as rapidly produced sporangia which can germinate directly or release swimming spores (zoospores) [49,70] – were judged to imply wind-blown rain or rain splash as mechanisms for movement of spores [65].
- Spatial patterns of risk for oak mortality were modeled on the basis of several landscape variables [6]. Proximity to forest edge was found to be the most important explanatory variable in a risk assessment of oak mortality from *Phytophthora ramorum*. Forest edge influence could be explained by the abundance of understorey foliar hosts in high-light edge environments.

Figure I. Sudden oak death. Image shows strands of dying tanoak *Lithocarpus densiflorus* (Marin County, CA, USA). Image taken during Spring 2002 by Marin County Fire Department. Reproduced with permission of Susan Frankel.

In this case, landscape fragmentation plays a key role in influencing pathogen dispersal and in altering the character of remaining habitat patches through edge effects. Further research is needed to determine the relative importance of localized edge effects and of pathogen spread behaviour for the clustering scale reported. The SOD case study integrates fieldwork, knowledge of pathogen life history, remotely sensed imagery analysis, geographic information systems and spatial modeling. The merging of these approaches can provide us with the ability to predict further spread of pests and diseases (e.g. [71]). Understanding and managing the dynamics of other exotic species invasions call for similar approaches.

models utilize concepts and mathematical tools that have been developed by landscape ecologists and metapopulation modelers [13,14]. Spatial modeling methods and landscape metric analyses have proved useful for investigating the spread of plant pathogens and the incidence and/or severity of tree diseases (e.g. [15,16]).

Landscape ecology addresses the interactions of spatial patterns and ecological processes over multiple scales [17]. Pathogen spread rates and population processes interact with the spatial patterns of host or vector species, which vary in their susceptibility or vector capacity, respectively. In turn, host landscape patterns are influenced by landscape connectivity and abiotic patterns, which also affect pathogen spread and population dynamics directly (Figure 1). Here, we review the key underpinnings of landscape pathology, an increasingly important interdisciplinary field that has emerged from the incorporation of landscape ecological concepts and methods into the science of forest pathology (Figure 2). Concentrating on fungal pathogens, we present examples of landscape pathology research and identify future research needs (Box 2).

Fragmentation effects on pathogens

Conservation biologists focus on the negative effects of habitat fragmentation, because it reduces dispersal and gene flow, degrades remnant habitats via edge effects and, thus, increases the probability of chance extinction in isolated patches (e.g. [18]). For the management of pathogens, fragmentation might not be undesirable provided the goal is to limit their presence in the landscape (but see [19]). However, landscape fragmentation generally leads to differential changes in habitat configuration for host, reservoir and pathogen species, making it difficult to predict the effects of fragmentation on pathogen population viability and spread rate. Fragmentation can hinder tree migration, reducing the capacity of host species to keep pace with climate change. Altered environmental conditions can increase the susceptibility of trees to disease, and the isolation of populations followed by genetic erosion can enhance this effect [20]. Conversely, the spread of pathogens following the artificial connection of previously divided eco-regions can have severe impacts on ecosystems [21]. For systems in which the host and pathogen have coevolved, recent modeling work shows that the possible risks of increased pathogen spread through corridors can be outweighed by the benefits resulting from increased recolonization by animal hosts [22]. However, the consequences of connectivity might be less beneficial taking into account the differential rates of spread of tree pathogens and tree species.

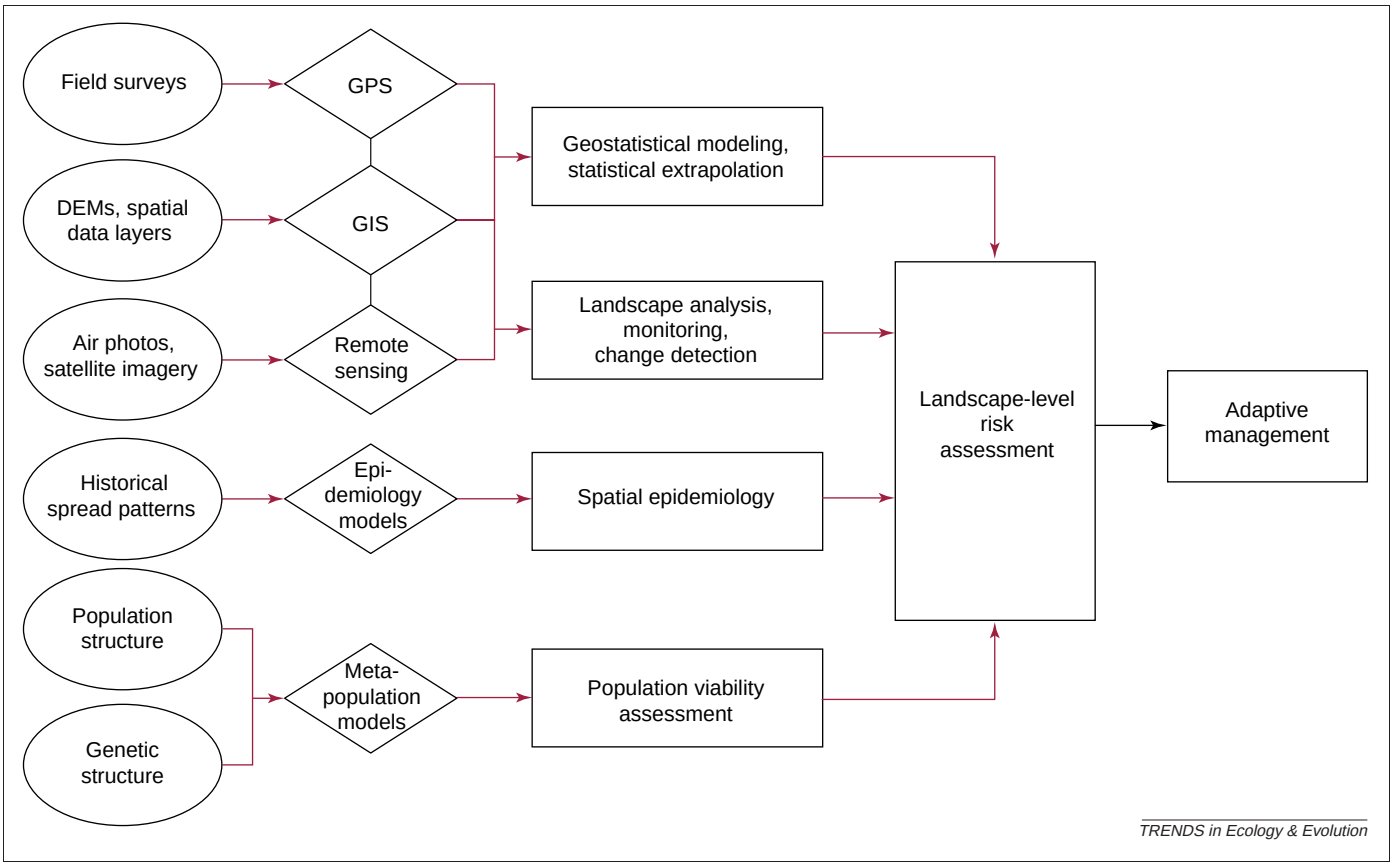

Figure 2. Data sources, quantitative tools, and analytical approaches central to landscape pathology (DEMs, digital elevation models).

Recent work highlights the importance of landscape connectivity for influencing tree pathogen spread rate over multiple spatial scales. In the coastal plain of the south-eastern USA, Perkins and Matlack studied three 10×10-km^2 independent blocks of a mosaic of plantations of loblolly pine *Pinus taeda*, slash pine *P. elliottii* (which is susceptible to fusiform rust *Cronartium quercuum*) and longleaf pine *P. palustris* (which is moderately resistant to *C. quercuum*) and naturally seeded stands (mainly of oak *Quercus* spp., the alternate host for *C. quercuum*) in a matrix of agricultural land [23]. For each block, land use and forest distribution were compared to pre-settlement data at 100 points on a 1-km grid. The degree of fragmentation, expressed as the distance between susceptible stands, was judged to be the crucial variable controlling the spread of the pathogen. In this case, modern forestry has reduced mean distance between stands and so increased connectivity. This has resulted in the facilitation of pathogen spread, which

Box 2. Outstanding questions: challenges for landscape-scale tree pathology research

Disease management with a landscape perspective can contribute much to sustainable land use and is an important challenge for tree pathologists. Landscape pathology is developing as a subdiscipline at the interface between forest pathology and landscape ecology. It meets the challenge by analyzing interactions between spatial features and disease processes. Key research questions amenable to landscape-based pathological investigation include:

- What is the overall effect of the degree of landscape fragmentation on pathogen spread, and can the value of connectivity for biodiversity [72] be outweighed by its potentially negative pathological effects?
- At which spatial scale(s) does the presence of resistant tree species provide a significant buffering effect against pathogen spread?
- What is the relative role of the topoclimatic environment versus landscape vegetation structure in determining the tendency for a pathogen to occur and to cause disease at a particular landscape site?
- How can we develop an understanding of pathogen effects within a historical context, considering the changes that occur in host and pathogen populations, often over many years?
- How can we design land-use systems that create landscape patterns that are suitable for sustainable ecological control of plant diseases?

Further research questions should consider the role of the various biotic and abiotic factors that are often invoked as causes of complex diseases (e.g. [73]), the role of insects both as vectors of pathogens and as debilitating factors (e.g. [74]), and the geographical scales over which tree diseases should be managed, taking into account their differential spread according to landscape features (e.g. *Phytophthora* along rivers; see [75,76]). The spread of pathogens from one pathosystem to another could be modeled as Brownian motion [77], but, in many cases, passive dispersal cannot be assumed because there are vectors (insects, birds or humans) to be considered carrying pathogens with them in a non-random way [78]. The issue is complicated by the heterogeneity of the landscape encountered by the pathogen, which needs to be understood in several ways, including: (i) spatial variability of primary or alternate host population presence, density and susceptibility; (ii) the number and abundance of resistant species and of antagonists potentially buffering the host–pathogen interaction; and (iii) pathogen dispersal rates, virulence and resistance to control [79]. Analogous to the drainage-basin scale of forest and landscape hydrology, the patho-region scale might emerge as a key frame of reference for forest pathologists. The pathoregion could be identified by the presence of a particular pathogen population and/or by a significant reduction in disease incidence at its borders.

occurs passively through wind dispersal of fungal spores, thought to be limited to a few hundred meters.

Support for the hypothesis that increased connectivity facilitates pathogen spread is also provided by a 23-yr study of the pathosystem formed by Port Orford cedar *Chamaecyparis lawsoniana* and its exotic root pathogen *Phytophthora lateralis* in a 37-km^2 area of the west coast of the USA [24]. Here, pathogen spread occurs through the transfer of propagules contained in mud and organic material on timber-harvesting vehicles moving through a network of forest roads, and then along river courses. The authors assessed the relative importance of large-scale vehicular dispersal compared with small-scale dispersal processes involving wildlife, cattle, hikers and workers in the woods. Vehicular dispersal was found to be the strongest influence on the rate of spread of *Phytophthora* to previously uninfested regions. At a considerably larger scale, the spread of white pine blister rust *Cronartium ribicola* is inhibited by fragmentation [25]. The high genetic differentiation of western and eastern American populations of *C. ribicola* is explained by a barrier to gene flow owing to the absence of five-needled pines and the alternate host *Ribes* spp. within a belt several hundred kilometers wide across the Great Plains. If this natural large-scale barrier were to be breached by the artificial transfer of the pathogen from one geographical range to the other, it could result in increased genetic diversity and, perhaps, virulence of the pathogen and disease expression within either of these geographical areas.

Predisposing environmental features

The dynamics of disease expression are influenced not only by limitations on the dispersal of pathogens arising from mosaic-level landscape and geographical structures, but also by the effects of site-related factors, either directly on the pathogens or on the susceptibility of their hosts. Manter *et al*. assessed the influence of slope exposition on disease expression of Swiss needle cast disease, caused by *Phaeocryptopus gäumannii* in Douglas-fir *Pseudotsuga menziesii* plantations in the western USA [26]. At the same level of pathogen incidence, southerly aspects had more needle cast than did plots with northern exposures.

Landscape pathology can help discern which site factors affect susceptibility. In sclerophyll vegetation of south-eastern Australia, Wilson *et al*. investigated the suitability of 17 site variables for predicting the distribution of *Phytophthora cinnamomi*, a pathogen causing generalized dieback in several types of Australian native vegetation [27]. Of the variables considered (e.g. aspect, slope, altitude, distance and elevation of sites from roads, average height of trees, canopy cover, several climatic variables, soil depth and drainage), only altitude and solar radiation index significantly affected variations in infection probability. In other studies, the pathogen was limited mainly by soil moisture [28]. Extrapolation from static spatial models such as these could be improved by considering the dynamics of the driving variables and feedback processes whereby pathogens affect the site environments (e.g. [2]).

The integration of predisposing site features in spatial risk assessment models of pathogen occurrence or disease expression is a significant research challenge [29]. Understanding and managing disease at a landscape level also requires information from finer scales. However, a restricted scale of investigation can bias the outcome if study plots are unrepresentative of disease at a broader scale. Unfortunately, not all studies report study area size or the spatial arrangement of sample plots. Care must be taken in scaling up from tree-level measurements as averaging within-site presence of the pathogen might produce a distorted image of the pathosystem (the system comprised of the interaction between host and pathogen in a disease prone environment) [30]. The importance of an appropriate landscape-level sampling methodology is exemplified by an investigation of the effects of topographical features on the occurrence of *Gremmeniella abietina* in Scots pine *Pinus sylvestris* forests in southern Finland [31]. Here, the effects of altitude upon disease expression are scale dependent, with positive correlations between mortality and altitude at a coarse scale, but negative correlations between mortality and local elevation. It might be that large-scale analyses give different results compared with local studies because large-scale analyses are unsuitable for explaining disease at a local scale. However, local studies might tend to overemphasize disease because they are often performed in those stands with the highest disease incidence.

If the selected study plots are representative of the surrounding landscape, a site categorization linking ecological features, the probability of disease development and appropriate management prescriptions can be developed. Examples include: a study of site and soil characteristics correlated with *Inonotus tomentosus* root disease in the sub-boreal spruce forests of British Columbia [32]; modeling work predicting the probability of occurrence of *Armillaria* root disease in South Dakota [33]; a study of the geographical distribution of *Armillaria* spp. in the Netherlands [34]; and a coarse-scale analysis of soil features related to *Collybia fusipes* root rot disease in French oak forests [35]. Together, these examples illustrate the increasing importance of addressing the broad-scale effects of site factors that influence tree pathogens and the susceptibility of their hosts.

Host and pathogen landscape patterns

In addition to the topographic, edaphic and climatic landscape features of sites at various geographical scales, vegetation patterns at the landscape level can play a key role in influencing disease expression [36] (Figure 1). For example, coupling the analysis of site factors associated with *Collybia fusipes* root disease with the distribution of host plants results in a more thorough understanding of the disease than do separate studies of environmental and host patterns [37]. An analysis of 54 subalpine stands of whitebark pine *Pinus albicaulis* in British Columbia found insignificant relationships between blister rust *Cronartium ribicola* disease incidence and differences in microclimate or site variables. The determining factors were stand structure and the presence of alternate hosts [38]. For eastern white pine *Pinus strobus* in the Lake States region, USA, climatic and topographic features were found to influence the risk of infection with blister rust [39].

Research is needed to assess whether this is related to a different pattern of host distribution compared with the risk of infection in *P. albicaulis*.

The presence of both the host and pathogen is a precondition for disease. A pathogen cannot persist if its sole host is wholly absent from a landscape, as might occur following an outbreak of high severity [11]. However, even at the stand scale, both host and pathogen rarely disappear completely. Within a stand, host trees might locally persist as a result of a lack of lethal infection. Equally, the pathogen might survive independently of the host in the form of dormant propagules [40]. The impracticability of eradicating a pathogen through complete elimination of its host is even more apparent at a regional level (e.g. [41]). A more sensible management strategy is to promote tree species diversity, thus reducing the susceptibility of forests to pathogens at both the stand and regional levels [42,43].

Historical factors (i.e. previous host and pathogen species distributions) must also be considered in landscape pathological studies, not only because they provide a baseline upon which to establish ecosystem management objectives, but also because they can affect disease development. For instance, in plantations of lodgepole pine *Pinus contorta* and Scots pine in northern Sweden there is a higher rate of infection by *Gremmeniella abietina* and *Phacidium infestans* on sites previously stocked by Norway spruce *Picea abies* compared with those stocked by Scots pine, because of greater pathogen inoculum [44]. Conversely, patterns in vegetation change over coarse spatial scales can result in changing patterns of susceptibility to forest pathogens over longer timeframes (e.g. [45]). Hence, adding a temporal dimension to landscape pathological studies can enable the detection of lag effects: pathogens might be out of phase with both the current landscape structure and host distribution. Rapid climate change might trigger the onset of disease by exacerbating this lack of synchronization (e.g. [16,46,47]).

Host spatial and temporal patterns are closely connected to the differential pathogenicity and distribution of fungal pathogens (e.g. *Armillaria* [48], *Phytophthora* [49], various fungi interacting with insects [50]). Across a region, the spatial distribution of different genotypes of a pathogen can significantly account for the variation in related tree mortality. The matching of a coarse-scale *Armillaria* population structure with data on host distribution, site relationships, and the incidence and severity of the disease has been presented for Oregon, with genet (the genetic individual that develops from the fusion of two sexually compatible homocaryotic mycelia) size estimates ranging up to 950 ha, corresponding to an estimated age of at least ~1900 years [51] and for Ontario, with rhizomorphs and infected wood samples found at 110 out of 111 sites [52]. These examples highlight the temporal and spatial pervasiveness of fungal tree pathogens. The elucidation of spatially explicit genetic structures of plant pathogens at a coarse scale [53,54] contributes increasingly to our understanding of ecological and evolutionary processes [55,56]. Together with landscape approaches and the reconstruction of gene flow in trees and their pathogens [57,58], these studies provide insights for achieving ecological sustainability in the context of land use and climate change.

Landscape perspectives for forest pathology

Tree species and forests are key elements in many landscapes. Understanding their spatiotemporal patterns is crucial not only for explaining landscape dynamics, but also for developing sustainable land-use strategies. The evolutionary consequences of diseases in 'natural' populations are receiving increasing attention [59,60]. In this way, the application of spatial and genetic tools has migrated from agricultural systems, land-use models, and general plant disease applications to specific cases of forest disease epidemiology and management. The Australian, European and North American bias in the case studies discussed could reflect the current effective landscape pathology effort. The extension of such effort towards developing and tropical regions will be facilitated by newly available high-resolution satellite data [61,62], to be validated by ground inspection of tree mortality [63].

Forest pathology will develop as a field only if it succeeds in collaborating further with other evolving disciplines (e.g. landscape genetics, evolutionary epidemiology, and adaptive ecosystem management). This challenge is analogous to the integration of diseases into future empirical and modeling studies of ecosystems. Currently, most landscape forest pathology investigations focus on one, or occasionally two, of the disease triangle corners (pathogen, host and environment [64]). Quantitative spatial analyses at a broad scale of variation in susceptibility, resulting both from physical predisposing features and host genetic characters, are generally lacking. To improve our understanding of tree diseases and to support science-based management decisions, we must include dynamic patterns of host and pathogen heterogeneity in spatially explicit models. There is scope for further research to merge concepts of landscape ecology with available molecular techniques, mapping the genetic structure of trees together with the genetic variation of pathogens, and identifying predisposing factors before infection becomes evident.

Forest and land-use management must incorporate the functional role of tree pathogens, not only when operating in the long term at a restricted scale, as was traditionally the case in forestry, but also in the short term for larger regions, as climate change and biological globalization will require in the future. Landscape pathology has management relevance in the larger context of landscape health and restoration and will call for the quantitative investigation of anthropogenic impacts on landscape patterns related to diseases. The merging of perspectives from landscape ecology and forest pathology will ultimately improve our understanding of tree diseases at a functionally relevant scale.

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