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Interacting Effects of Global Change on Forest Pest and Pathogen Dynamics

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Abstract

Pathogens and insect pests are important drivers of tree mortality and forest dynamics, but global change has rapidly altered or intensified their impacts. Predictive understanding of changing disease and outbreak occurrence has been limited by two factors: (*a*) tree mortality and morbidity are emergent phenomena determined by interactions between plant hosts, biotic agents (insects or pathogens), and the environment; and (*b*) disparate global change drivers co-occur, obscuring net impacts on each of these components. To expand our understanding of changing forest diseases, declines, and outbreaks, we adopt a framework that identifies and organizes observed impacts of diverse global change drivers on the primary mechanisms underlying agent virulence and host susceptibility. We then discuss insights from ecological theory that may advance prediction of forest epidemics and outbreaks. This approach highlights key drivers of changing pest and pathogen dynamics, which may inform forest management aimed at mitigating accelerating rates of tree mortality globally.

I. INTRODUCTION

In forest systems, pathogens and insect pests shape population structure, community composition, and ecosystem processes via their impacts on tree physiology, mortality, and morbidity (see the sidebar titled Tree Mortality and Morbidity) (Cobb & Metz 2017, Franklin et al. 1987, Mordecai 2011, Preston et al. 2016). Climate change and other anthropogenic impacts have caused the emergence of novel or altered diseases and outbreaks in forest systems across the globe, creating substantial ecological and economic challenges (Bentz et al. 2010, Ghelardini et al. 2016, Roy et al. 2014). Rapidly changing pest and pathogen dynamics have contributed to the loss of foundational tree species, disruption of food webs (Ellison et al. 2005), shifts in forest composition (Metz et al. 2012), altered biogeochemical cycling (Preston et al. 2016), the emergence of new, landscape-scale biotic disturbances, and changes to existing disturbance regimes (Johnstone et al. 2016, Metz et al. 2011, Raffa et al. 2008). Understanding future shifts in pest and pathogen dynamics will be essential to predicting tree mortality, community composition, ecosystem function, and the availability of forest resources in a changing world.

Despite their clear ecological importance, identifying the mechanisms that underlie global change effects on forest diseases and outbreaks is challenging. Firstly, forest diseases, insect outbreaks, and subsequent tree mortality are phenomena that emerge only when a susceptible host, a virulent biotic agent, and conducive environmental conditions co-occur in space and time. Further, drivers of global change, including land use change, climate change, species invasions, and human impacts on disturbance regimes, interact and impact these three key factors via contrasting or synergistic mechanisms across scales. Other abiotic drivers, such as fire and drought, further contribute to increasing patterns of tree mortality globally, interacting with or obscuring the impacts of these biotic agents (Allen et al. 2015, Anderegg et al. 2015, Millar & Stephenson 2015). These forms of complexity hamper predictive modeling in many cases (Hartmann et al. 2018), but progress in identification of causal mechanisms suggests that forecasting risk and, in some cases, preventing impacts of damaging biotic agents are possible (Garrett et al. 2006, Liebhold et al. 2012, Meentemeyer et al. 2012).

To address this complexity, we synthesize and discuss the impacts of drivers of global change as they relate to the specific mechanisms determining the virulence of biotic agents and the susceptibility of tree hosts, highlighting their relative contributions to changing disease- and outbreak-caused mortality in forest systems. Specifically, we aim to (*a*) organize the diverse possible mechanisms that cause shifting forest pest and pathogen dynamics under global change into a single framework, drawing upon principles from pathology, and (*b*) integrate these pathways into existing ecological theory to advance prediction of altered impacts of forest pests and pathogens. We leverage familiar concepts from plant health sciences and ecological theory to accomplish these goals, integrating examples primarily from nontropical forest systems, including temperate and boreal types across the globe.

TREE MORTALITY AND MORBIDITY

Pathogens and insects can affect both the vitality and survivorship of individuals or populations of trees. Here, we define mortality as the complete loss of physiological function and distinguish this from tree morbidity, in which growth and reproduction are reduced to the point that individuals and populations are unable to sustain regeneration of subsequent populations or maintain current canopy or community dominance. Morbidity as a health term is more often applied to human health, but the term is also applicable to the impacts of forest pests and pathogens.

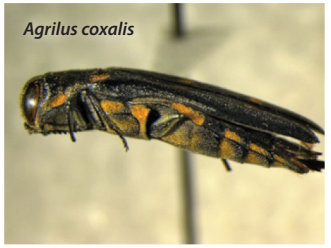
Insect pests and tree pathogens (which span fungal, viral, plant, and bacterial taxa) are different classes of organisms and are often, justifiably, treated separately in characterizing forest health problems. Yet, both insect pests and pathogens negatively impact tree growth, cause mortality, alter forest structure and composition, and shift ecosystem processes. Further, these organisms commonly attack individual trees in conjunction, jointly influencing host mortality (Dooley & Six 2015, Ploetz et al. 2013), necessitating examination of how global change will alter the dynamics of both insect pests and pathogens concurrently. In this review, we compare and contrast these classes of biotic agents while acknowledging the limitations of this approach whenever possible. There are many reasons why clear biological differences between insect and pathogen groups preclude lumping these organisms into a single class (**Table 1**). Insects are multicellular protostome animals with centralized nervous systems that regulate complex behaviors, such as directed movements and pheromone-driven mass attack (Bale et al. 2002, Bentz et al. 2010). These behaviors have no analogy in pathogen systems. Similarly, there are several examples of microbial pathogens with biological or ecological dynamics that have no parallel to insects. For example, the spores of many root rot fungi of forest trees (e.g., *Armillaria* spp., *Phellinus weirii*) are single-celled but may develop into massive, long-lived genetic individuals colonizing trees over many hectares, in stark contrast with the shorter life spans of most insects (Hansen & Goheen 2000, Rizzo et al. 2000). Although there are clear limitations to comparisons between these groups of organisms, we propose that progress toward understanding the interacting impacts of global change on disease, outbreak, and tree mortality can be made by considering pathogen and insect pests jointly while identifying cases where this combined approach is inappropriate (**Table 1**).

2. A FRAMEWORK FOR THE IMPACTS OF GLOBAL CHANGE ON PEST AND PATHOGEN DYNAMICS

The negative physiological impacts of forest pests and pathogens on trees are emergent phenomena that depend upon three interacting factors: the virulence of the biotic agent, the susceptibility of the host, and the environmental context of their interaction (McNew 1960). In plant pathology, the classic disease triangle framework is commonly used to describe how this three-way interaction among virulent agent, susceptible host, and conducive environment leads to disease emergence at the individual, stand, or landscape scale (McNew 1960, Scholthof 2007). However, agent virulence, host susceptibility, and their relationship with abiotic factors are multifaceted characteristics determined by a suite of underlying biological processes. The virulence of biotic agents may be primarily influenced by (*a*) the species identity, genotype, and distribution of the agent, which determine its inherent virulence, and (*b*) the population dynamics of the infectious agent, as shaped by dispersal, survival, growth, and reproductive rates, which govern transmission or infection pressure (**Figure 1**). In turn, host susceptibility varies with (*a*) the species identity, distribution, and genotype of the host (or its inherent susceptibility), (*b*) the density, spatial orientation, and local composition of hosts, which determine individual risk and transmission, and (*c*) the host's physiological state or stress (Garrett et al. 2006, 2011; Meentemeyer et al. 2012) (**Figure 1**).

Anthropogenic drivers of global change may act upon agent virulence and host susceptibility in interacting, antagonizing, or contrasting ways, concealing net effects on tree mortality. Therefore, in this review, we organize current knowledge of changing pest and pathogen dynamics in forests using these underlying mechanisms, determining agent virulence and host susceptibility as a structure for discussion. This approach identifies important components of changing disease and outbreak dynamics, highlights gaps in our existing mechanistic understanding, and draws new comparisons between the impacts of disparate global change drivers, including climate change, human land use, species translocations or invasions, and altered abiotic disturbances. Further, this

Table 1 Contrasts between insect pest and pathogen life history characteristics

	Broad taxonomic groups	
	Insect pests	**Pathogens, including fungi, oomycetes, and parasitic plants**
Example species	 	 
Behavior	Exhibit behavior with centralized command and control (e.g., pheromone-mediated mass attack)	Lack centralized command and control; spread is more strongly governed by physical processes
Size	Relatively small animals with a narrow range of body mass compared with pathogens	Range from single-celled to the largest known living single organisms
Life span	Short life spans; generally a year or shorter	Highly variable life spans; from seasons to centuries
Reproduction strategies and systems	Sexual and asexual reproduction, including pseudogamy and parthenogenesis	Extremely diverse, including clonal reproduction and a range of polyploidy
Reproduction rate	Can be rapid with egg clutch sizes of up to several hundred eggs	Rapid with spore production ranging from hundreds to billions of spores
Ecological shifting	Generally do not shift ecological roles (herbivore to predator, etc.); pre- and post-metamorphosis can be an exception	Ecological switching is frequent and includes switching from mutualistic or saprotrophic to pathogenic modes

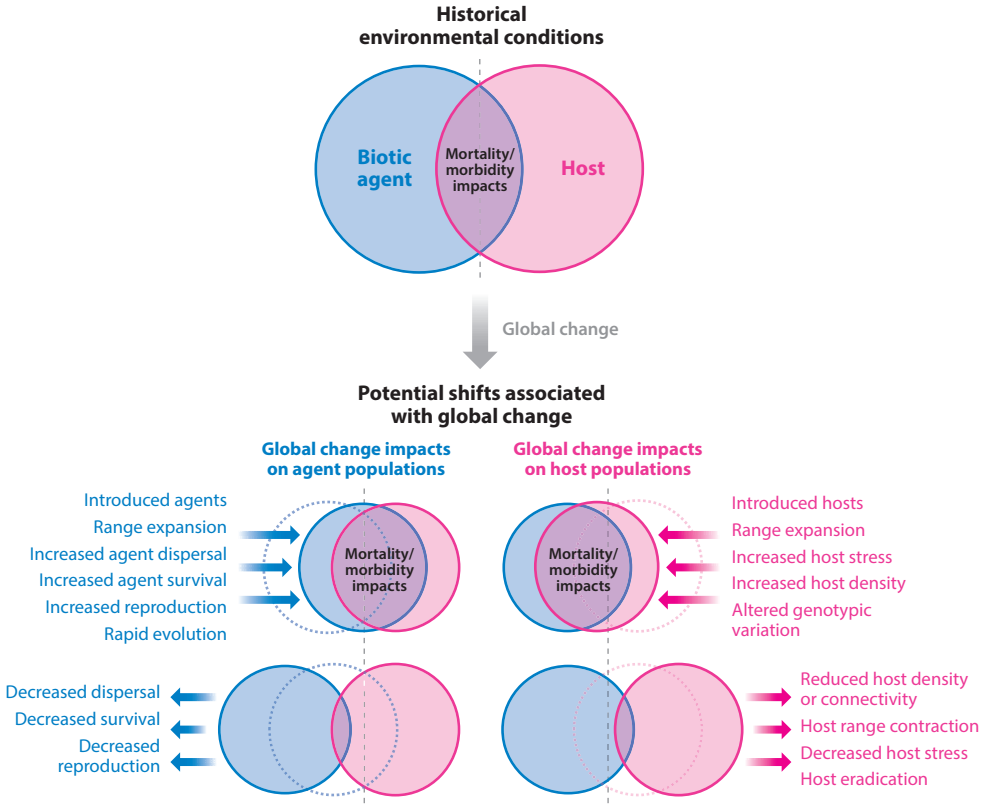


Figure 1

Mortality or morbidity impacts (*purple regions*) from biotic agents emerge only through interactions between susceptible host trees and virulent pests or pathogens in conducive environmental conditions. Drivers of global change may expand or contract the current extent of mortality or morbidity impacts in forest systems by causing shifts in the virulence of biotic agents (*blue arrows*) or in the susceptibility of tree hosts (*magenta arrows*), via the outlined mechanisms.

framework highlights instances in which a single mechanism, or a suite of interacting factors, is responsible for changing pest and pathogen dynamics (**Table 2**). Following this discussion, we propose several ways in which foundational theories from ecology may advance our understanding of agent virulence and host susceptibility to improve predictions of changing forest disease epidemics or insect outbreaks.

3. MECHANISMS DETERMINING PATHOGEN OR PEST VIRULENCE

In disease systems, pathogen virulence is typically defined as the extent to which an agent infects and causes damage to a host (McNew 1960, Scholthof 2007). Though the concept of virulence is not commonly applied to insects, here, we examine similar variation in pests' inherent abilities to colonize and cause physiological damage to host trees. Due to the indeterminate growth and compartmental morphology of trees, the impacts of most forest pests and pathogens often scale with infection intensity (or population size), exhibiting macroparasite, rather than microparasite, dynamics. Drivers of global change have altered the virulence of biotic agents in temperate forest systems via their impacts on agent occurrence, genetic variation, dispersal, survival, growth, and reproduction (Ghelardini et al. 2016, Weed et al. 2013) (**Figure 1**).

Table 2 Examples of the two primary drivers contributing to changing and emerging insect or pathogen dynamics, as they relate to host susceptibility and agent populations

Insect or pathogen system	Primary mechanism	Secondary or interacting driver	Secondary mechanisms	Reference(s)
Changes to agent virulence or population pressure				
Hemlock woolly adelgid (*Adelges tsugae*)	Naive host; introduced agent	Agent survival	Increasing minimum winter temperatures	McAvoy et al. 2017, Orwig et al. 2012
Sudden oak death (*Phytophthora ramorum*)	Naive host; introduced agent	Agent population dynamics and host occurrence	Timing and amount of precipitation, wildfire occurrence; land use change	Beh et al. 2012, Eyre et al. 2013, Meentemeyer et al. 2008
Phytophthora root rot (*Phytophthora cinnamomi*)	Naive host; introduced agent	Agent survival	Increasing minimum winter temperatures	Burgess et al. 2017
Western spruce budworm (*Choristoneura freemani*)	Drought and precipitation impacts on population growth	Host stress	Drought impacts on host susceptibility	Flower et al. 2014
Changes to host susceptibility or occurrence				
Eastern spruce budworm (*Choristoneura fumiferana*)	Changes in host density and composition	Agent population growth	Warming temperature effects on insect development	Bouchard & Auger 2014
Fusiform rust (*Cronartium quercuum*)	Changes in host density and composition	NA	NA	Randolph et al. 2015
Swiss needle cast (*Phaeocryptopus gaeumannii*)	Changes in host density and composition	Agent population growth	Incidence of warm summer rain events	Woods et al. 2005
Mountain pine beetle (*Dendroctonus ponderosae*) on jack pine (*Pinus banksiana*)	Similarity in phytochemistry between novel and historical hosts	Agent survival	Northward range expansion associated with warming temperatures	Erbilgin 2019

Abbreviation: NA, not applicable.

3.1. Occurrence, Dispersal, and Genetic Variation in Pathogen and Pest Populations

Dimensions of global change have fundamentally altered the geographical occurrence and movements of forest pest and pathogens, locally and over longer distances. Most notably, numerous novel forest diseases and epidemics have emerged from the long-distance translocation of nonnative biotic agents to new regions, such as the introduction of gypsy moth (*Lymantria dispar dispar*) and chestnut blight (caused by the fungus *Cryphonectria parasitica*) to the eastern United States (Anagnostakis 1987, Liebhold et al. 1992). Pest and pathogen introductions have primarily been facilitated by international trade and the movement of infected live plants, wood materials, or soil (Liebhold et al. 2012, Roy et al. 2014). Aukema et al. (2010) recorded the steady introduction of approximately 2.5 novel pests or pathogens per year to the United States between 1850 and the early twenty-first century. These translocations can bypass biogeographic gradients that determine the degree of coevolved defenses between nonnative biotic agents and native

hosts; thus, these agents can have dramatic physiological impacts, causing local eradication of foundational forest species (Ellison et al. 2005, Gilbert 2002). Within their introduced ranges, these agents may develop new relationships with native vectors (Ploetz et al. 2013, Wingfield et al. 2016) and can generate novel biotic disturbances, given their acute impacts on tree mortality (Cobb & Metz 2017, Johnstone et al. 2016, Weed et al. 2013).

Cultural or economic activities and human translocation of infected materials also directly influence the occurrence and dispersal of biotic agents within their existing ranges, beyond their impacts on long-distance pest and pathogen movement. For instance, within its introduced range, the occurrence of the pathogen *Phytophthora lateralis*, which causes root disease in Port Orford cedar (*Chamaecyparis lawsoniana*), is strongly linked to road infrastructure. Movement of pathogen-contaminated sediment on vehicles is an important source of within-range dispersal (Jules et al. 2002). Restoration and horticultural activities may also play a role, not only in facilitating long-distance introductions but also in intensifying the spread of forest pests, pathogens, or new strains and genotypes thereof into uninvaded regions. Hospitable conditions within nurseries may amplify agent occurrence, and dispersal may be facilitated by subsequent planting of infected hosts (Parke & Grünwald 2012, Sims et al. 2019). Changing environmental conditions may also influence genetic structure of pest and pathogen populations and facilitate the diversification of host–agent associations, which have been historically sparked by past periods of climatic change (Hoberg & Brooks 2015).

In addition to human translocations of novel forest pests and pathogens, changing climatic conditions alter the preexisting dispersal pathways of biotic agents, with implications for infection pressure on host trees. Insect pest movement may be encouraged by increasing temperatures, which determine characteristics of many insect behaviors, including flight distances and the timing of seasonal emergence (Bale et al. 2002). In warming environments, earlier emergences and longer flight season durations have been reported or predicted for a variety of bark beetle pests, such as *Ips typographus* (Jönsson et al. 2009, Wermelinger et al. 2012) and *Dendroctonus ponderosae* (Mitton & Ferrenberg 2012) as well as for defoliating species (Netherer & Schopf 2010). For flying insects with long-distance dispersal, changes in wind intensity and direction are also likely to alter local and long-distance dispersal events (Anderson & Sturtevant 2011).

Changes in insect behavior and movement also have clear implications for the dynamics of vectored forest pathogens (Wingfield et al. 2016). However, dispersal of many forest pathogens is passive, driven by physical processes like precipitation and wind, which will also shift in relation to climate change. *Dothistroma septosporum*, the causal agent of red band needle blight in Canada and the United Kingdom, produces asexual conidia that are locally transported by rainfall passing through infected canopies and, less frequently, via wind over longer distances (Boateng & Lewis 2015, Mullett et al. 2016). Similarly, spore dispersal for *Lecanosticta acicola*, the cause of white pine needle damage in the eastern United States, is positively related to not only the abundance of rainfall but also the timing of rainfall within the season (Wyka et al. 2017). In its introduced range in the coastal western United States, *Phytophthora ramorum*, the causal agent of sudden oak death, spreads prolifically within forest canopies during warm, wet springs; however, based on data examining migration rates for genotypes within stands, dispersal effectively halts during periods of drought (Eyre et al. 2013). Together, these results suggest that increased precipitation—more specifically, more variable precipitation patterns and increasing rainfall during key periods of pathogen reproduction—may intensify the behavior of certain diseases, particularly those caused by fungal or fungal-like agents.

Land use change and landscape heterogeneity additionally influence pathogen and pest dispersal by altering spatial relationships between patches of suitable habitat and subsequent occurrence of so-called natural enemies (Meentemeyer et al. 2012). Trees in well-connected stands of

primeval forest suffered increased defoliation due to winter moth (*Operophtera brumata*) compared with more fragmented patches in regions of Poland (Wesołowski & Rowiński 2006). These results may be attributed to greater dispersal distances between patches of host habitat, yielding increased mortality of insect pests at specific developmental stages. Simulation modeling of gypsy moth invasion in forests of the eastern United States also suggests that rates of spread are limited by dispersal mortality as moths travel long distances across unsuitable matrix habitat (Walter et al. 2016).

3.2. Pathogen and Pest Survival

Climate change directly alters pest and pathogen survival rates, influencing agents' ranges and population growth rates. Rates of overwinter survival of forest pests and pathogens may limit epidemics and outbreaks, and increasing winter temperatures in temperate regions have been linked to changing occurrence and impacts for a variety of biotic agents (McAvoy et al. 2017, Raffa et al. 2008, Sturrock et al. 2011). Jepsen et al. (2011) associated range shifts in two defoliating geometrid moths, *Epirrita autumnata* and *O. brumata*, with increasing winter temperatures. These moth species suffer high egg mortality below −35°C, but few or no periods have crossed that threshold in recent years (Jepsen et al. 2011). Minimum winter temperatures are primarily responsible for slowing rates of hemlock woolly adelgid (*Adelges tsugae*) spread across New England and southern Canada (Orwig et al. 2012). During years with minimum temperatures below −25 to −30°C, populations experienced 95–99% overwinter mortality rates; thus, expected increases in minimum winter temperatures are likely to result in invasion of new stands, particularly in northern New England and parts of maritime Canada (McAvoy et al. 2017). Warming minimum winter temperatures may also increase overwinter survival of bark beetle species and subsequent outbreak likelihood (Bentz et al. 2010, Preisler et al. 2012). Similarly, the introduced forest pathogen *Phytophthora cinnamomi* is susceptible to frost, and subsequent disease severity is related to overwinter survival; therefore, this pathogen is expected to expand its current European range with increasing winter temperatures (Bergot et al. 2004, Burgess et al. 2017).

Many forest insect pest populations are regulated by mutualistic or parasitic relationships with symbionts or natural enemies, and these interactions may shift with changing climatic conditions. Fungal symbionts improve nutrition and survival in bark beetle species; however, future temperature shifts are projected to differentially affect the various symbiotic fungi that associate with mountain pine beetle *D. ponderosae* and possibly disrupt the coexistence of these mutualistic species, reducing benefits to this insect (Addison et al. 2013, Six & Bentz 2007). Similarly, the impacts of a fungal pathogen (*Entomophaga maimaiga*) on gypsy moth survival significantly varied across moisture gradients in the eastern United States (Reilly et al. 2014). Alterations in the strength or direction of these mutualistic and parasitic relationships have important implications for outbreak emergence, but few studies have been conducted (Addison et al. 2013).

Human-altered disturbances, generated by climate change or associated with anthropogenic activities, may also directly impact agent survival rates. Consumption of host vegetation by wildfire directly reduces the occurrence of the oomycete *P. ramorum* in the western United States (Beh et al. 2012). Though research examining direct disturbance impacts on pathogenic species is limited, changing disturbance regimes are likely to contribute to local patterns of pathogen occurrence in forest systems. For example, wildfire causes direct mortality in a variety of microbial groups, generating shifts in forest mycorrhizae (Glassman et al. 2016) and foliar endophyte communities (Huang et al. 2016). Changes in canopy cover associated with disturbance or management activities may also influence forest floor temperatures, water infiltration, or snowpack accumulation (Varhola et al. 2010). These shifts could impact the survival of forest insects that overwinter in litter and duff, but possible effects remain poorly understood.

3.3. Pathogen and Pest Reproduction

Temperature, humidity, and precipitation, altered by climate change, directly impact agent reproduction and subsequent growth rates of pest and pathogen populations. Reproduction of *D. septosporum*, causal agent of Dothistroma needle blight in the U.S. Pacific Northwest, is facilitated by periods of extended or continuous moisture. Under future climates, landscape-scale Dothistroma needle blight epidemics may be facilitated by increasing occurrence of warm rain events (Woods et al. 2005). Warming temperatures have been associated with shifts in the timing of developmental stages in many bark beetle pest species in the United States (Bentz et al. 2010). Climate change–induced range expansions of forest pests and pathogens are also likely to increase encounters between host populations with fewer coevolved defenses, which may influence agent reproductive rates, infection, or attack success. For example, higher reproductive rates were observed for mountain pine beetle in lodgepole pine (*Pinus contorta*) trees that had not previously been frequently exposed to outbreaks, suggesting weaker defenses in these hosts (Cudmore et al. 2010).

Warming temperatures and earlier emergence times may also accelerate development rates and increase the number of generations that insect pests can complete in a single year, with implications for population growth, overwinter survival, and outbreak likelihood. *I. typographus*, the Eurasian spruce bark beetle, is univoltine (completing one generation in a year) across most of its range; however, climate modeling of the development and emergence of this insect suggests that by 2050, *I. typographus* will successfully establish a second generation annually in approximately half of years in the southern extent of its range (Jönsson et al. 2009). Similarly, in univoltine mountain pine beetle populations, some broods have shown evidence of the initiation of a second generation within a single season (Mitton & Ferrenberg 2012).

However, not all populations respond strongly to changing climatic conditions (Weed et al. 2013), and dimensions of local adaptation may limit the extent to which insects can respond to climatic variation. In many pests, day length or photoperiod partially determines the initiation of diapause, in addition to temperature conditions, which could limit insect population responses to climatic shifts (Jönsson et al. 2009). Bentz et al. (2014) showed evidence of genetic variation in bark beetle development rates along a gradient of climatic conditions across the western United States, which may limit the potential for these populations to exhibit bivoltinism in response to changing environments. Underlying genetic variation, the limits to agent plasticity, and nonlinear relationships among voltinism, population growth, and fitness may constrain our capacity to predict changes in outbreak occurrence, as predicted by climate.

Forest management activities and land use further alter pest and pathogen population growth. *Heterobasidion* spp. are important drivers of tree mortality in many conifer forests across the northern hemisphere. Establishment of long-lived individuals and production of inoculum in the landscape is often determined by the prevalence of stumps and wounds associated with harvesting (Garbelotto & Gonthier 2013). Similar mechanisms have been attributed to increased abundance of *Armillaria* spp. in previously logged forests, where discrete inputs of buried organic matter (from large roots and stumps) may lead to local inoculum proliferation and disease emergence (Baumgartner & Rizzo 2001).

4. MECHANISMS DETERMINING HOST SUSCEPTIBILITY AND ITS INTERACTION WITH ENVIRONMENT

In regions where pathogen or insect populations have not been altered via the mechanisms determining agent virulence discussed so far, novel outbreaks or diseases may additionally emerge from the impacts of global change on dimensions of host susceptibility (**Figure 1**). Susceptibility, a host's inability to resist infection, invasion, and damage by an agent, varies among species as well as over

space and time owing to changes in host physiology, morphology, and population or community structure (McNew 1960, Scholthof 2007). Specifically, changing climate, land use, disturbance, and other human activities influence three primary factors determining host susceptibility: the distribution of host species or genotypes, host density or spatial orientation, and host stress.

4.1. Occurrence and Genetic Structure in Host Populations

Over time, spatial and temporal variation in pressure from biotic agents has determined the occurrence of host species and of resistant genotypes within host populations (Laine et al. 2011). Drivers of global change both directly alter and interact with these underlying species-level and population-level patterns in host susceptibility. As with the movement of nonnative pests and pathogens, translocation or planting of nonnative trees through forestry and horticulture can introduce novel hosts to systems. These events may encourage endemic pathogens to jump or shift hosts from native to introduced species (Burgess & Wingfield 2017). Risk of host shifts is thought to be highest when species are translocated into regions containing native host plants with similar phylogenetic histories (De Vienne et al. 2009). For instance, following the introduction of *Eucalyptus* plantations to South and Central America, this exotic host has become infected by native pathogens found on closely related endemic species in the order Myrtales, including the rust fungus *Puccinia psidii* and *Chrysoporthe cubensis*, which causes Cryphonectria canker (Burgess & Wingfield 2017). Each of these emerging diseases causes significant negative impacts on commercially planted *Eucalyptus* trees.

However, even within existing ranges, spatial variation in host genotypic resistance suggests that local tree translocations or possible migrations may alter disease and insect impacts. In Australian blue gum forests, patterns of disease caused by the endemic pathogen *Corymbia citriodora* on *Eucalyptus globulus* provided evidence that this pathogen had driven spatially variable selection on this host (Freeman et al. 2018). Decreased susceptibility was observed for hosts collected from regions with climatic conditions more conducive to fungal proliferation (Freeman et al. 2018). Similarly, in a provenance study, Douglas-fir (*Pseudotsuga menziesii*) trees, grown from seed and translocated from different regions in the western United States, exhibited variation in resistance to *Rhabdocline* spp., the causal agents of Rhabdocline needle cast, and to *Phaeocryptopus gaeumannii*, the causal agent of Swiss needle cast, when grown in a common environment (Wilhelmi et al. 2017). These results indicate that spatial mosaics of evolved host resistance may influence emergent patterns of disease, as climate change causes local and regional shifts in pathogen occurrence. Clear genetic variation in other forest species' responses to forest pests and pathogens (Cobb et al. 2019, Dodd et al. 2008) also suggest that even short-distance translocations of hosts, through management or conservation actions, could generate important ecological or economic impacts (Simler et al. 2019).

Underlying genetic variation in host populations may also interact with or determine pest and pathogen range shifts. In Alberta and British Columbia, Canada, mountain pine beetle is rapidly expanding into regions more dominated by jack pine (*Pinus banksiana*). Similarity in the secondary chemistry of this new host and the beetle's historical host, *P. contorta*, may facilitate range expansion (Erbilgin 2019). Across jack pine populations, spatial variation in the composition of monoterpenes in host phloem influences beetle aggregation and pheromone production (Taft et al. 2015). Mountain pine beetles were also observed to reproduce more prolifically on their historic host, *P. contorta*, in regions that had not previously been impacted by frequent outbreaks, providing additional evidence of pest-induced selection pressures on hosts (Cudmore et al. 2010). Underlying spatial variation in host genotypes and secondary chemistry is hypothesized to have important implications for the expansion of insect pests.

Limited forms of resistance may also exist within naive host populations exposed to introduced agents, which may be, in turn, shaped by global change drivers. Single genes may determine complete resistance, as observed in sugar pine populations (*Pinus lambertiana*) impacted by white pine blister rust (*Cronartium ribicola*) (Devey et al. 1995), or more variable forms of resistance may exist, as observed in tanoak and oak populations impacted by sudden oak death, or ash populations (*Fraxinus excelsior*) impacted by ash dieback (*Hymenoscyphus fraxineus*) (Conrad et al. 2019, Hayden et al. 2011, Landolt et al. 2016). In some cases, resistance may be linked to other traits determining susceptibility to abiotic stressors, suggesting potential interactions with climate change or other anthropogenic drivers. For instance, limber pine (*Pinus flexilis*) seedlings resistant to white pine blister rust have been observed to exhibit greater cold hardiness and lower stomatal conductance during drought, compared with disease-susceptible individuals (Vogan & Schoettle 2015). The extent to which existing variation in resistance will impact longer-term shifts in pest and disease dynamics will depend on the heritability of these traits, genetically informed management actions, other human movements of hosts, and the selection pressures exerted on populations by global change drivers.

4.2. Density and Spatial Orientation of Hosts

Climate change, land use change, altered disturbances, and other human impacts shift host density, connectivity, and composition, with implications for susceptibility at local and landscape scales. At the stand level, land use change and silvicultural activities have altered tree susceptibility by changing host density and forest composition. In the Pacific Northwest of the United States, many coastal lands have been converted from a dominant Sitka spruce (*Picea sitchensis*) and western hemlock (*Tsuga heterophylla*) forest to Douglas-fir (*P. menziesii*) timber plantations. Swiss needle cast is an important disease affecting forests of natural origin in this region (Black et al. 2010), but impacts are often most severe on formerly spruce–hemlock dominated sites that have been converted to Douglas-fir plantations (Hansen et al. 2000). Increased density of Douglas-fir, previously a minor component of these forests, may be an important factor in determining these shifts, in addition to climatic conditions at these converted sites (Ritóková et al. 2016). Similarly, intensification of silviculture in the southeastern United States has increased the occurrence of loblolly and slash pine stands, compared with historical distributions. Increased connectivity of these susceptible hosts has been attributed to greater spread and impacts of fusiform rust (*Cronartium quercuum*) and southern pine beetle (*Dendroctonus frontalis*) (Perkins & Matlack 2002). Host aggregation generally increases the severity of southern pine beetle impacts in these regions (Cairns et al. 2008), and management activities may seek to decrease connectivity or density to reduce outbreak occurrence.

In sudden oak death–impacted regions of the western United States, *P. ramorum* disease severity has been shown to increase in areas surrounded by a higher proportion of contiguous forest, a landscape characteristic influenced by past human land use and management (Condeso & Meentemeyer 2007). In New England, the distribution of eastern hemlock (*Tsuga canadensis*) was also historically shaped by human land use and disturbance (Foster & Zebryk 1993). As a result, the patterns and extent of forest conversion by hemlock woolly adelgid reflect centuries of landscape change, often driven by management or land use, that determined the preinvasion distribution of hosts (Nuckolls et al. 2009, Orwig et al. 2012).

Modern human-impacted disturbance regimes, which are shifting in severity, timing, frequency, and type, may further alter disease or outbreak dynamics by favoring or locally eradicating important host species. Moore and colleagues (2014) found that more recent and frequent fire yielded increased disease severity associated with *P. cinnamomi*, a pattern that they attributed to the abundance of susceptible, newly germinated plants in addition to wetter and warmer soil conditions. In ecosystems invaded by *Discula destructiva*, the causal agent of dogwood anthracnose,

wildfire reduced disease severity on declining *Cornus florida* populations via changes to forest structure and composition. These results suggested that recent fire suppression may have intensified the impacts of this emerging disease (Holzmueller et al. 2008). *P. ramorum* also occurs more commonly in forest stands that have not recently burned; this pattern may be explained by the impacts of wildfire on the size and maturity of important host canopies, which may better intercept and spread inoculum suspended in wind or rain (Metz et al. 2012, Moritz & Odion 2005). Meentemeyer and colleagues (2008) suggested that land cover changes between 1942 and 2000, associated with human fire suppression, resulted in expansions of forest and woodland cover, which increased the landscape occurrence of *P. ramorum*'s primary sporulating hosts.

4.3. Host Physiological Condition

The growth–differentiation balance hypothesis suggests that trade-offs may occur between plant allocation to growth, defense, or other physiological processes, depending on stress or resource availability (Herms & Mattson 1992). Thus, increasing drought severity or other environmental stressors associated with climate change may deplete trees' nonstructural carbohydrate reserves and limit allocation to defense, impacting host susceptibility to pathogen and pest impacts (Anderegg et al. 2015, Kolb et al. 2016, McDowell et al. 2011, Oliva et al. 2014). Further, plants can suppress pathways of secondary chemical synthesis to respond to more critical environmental threats to survival (Bostock et al. 2014). For example, in *Arabidopsis*, the general hormonal response to stress—abscisic acid—both closes stomata and suppresses jasmonic acid and salicylic acid pathways associated with defense (Bostock et al. 2014).

Studies examining interactions between abiotic stress and host susceptibility to pests and pathogens are rare in adult trees and at forest stand scales. Gaylord and colleagues (2013) found evidence that drought predisposes mature piñon pines (*Pinus edulis*) to bark beetle attack, though water stress had variable impacts on tree resin defenses. Further, defense against native bark beetles decreased in whitebark pine (*Pinus albicaulis*) individuals that also carried high levels of white pine blister rust infection (Dooley & Six 2015). Water stress may further influence the nutritional content and structural properties of host leaves, with implications for feeding, infection, and reproduction by foliar pathogens and defoliating insects (Kolb et al. 2016). Though potential interactions between environmental stress and tree susceptibility remain poorly understood, theoretical frameworks suggest that the impacts of abiotic stress on host predisposition to pathogens (Oliva et al. 2014) and forest pests (Anderegg et al. 2015) may differ depending on agent life history strategies and the primary pathways of attack. Changes in forest density and stand-level competition associated with past management activities or land use may interact with or antagonize these climatic drivers of host stress (Cobb et al. 2017, Fettig et al. 2007).

4.4. Phenological Shifts: Interactions Between Susceptible Hosts and Virulent Agents

Lastly, rather than altering host susceptibility or agent virulence directly, climate-induced changes in pest, pathogen, and plant phenology can increase or decrease the extent to which susceptible hosts and virulent biotic agents may interact, altering subsequent disease or outbreak impacts. Young, developing leaves of European oak trees are susceptible to the impacts of European powdery mildew, caused by fungi in the genus *Erysiphe*, and phenological synchrony between oak bud burst and the pathogen's production of ascospores influenced disease severity (Marçais & Desprez-Loustau 2014). Similarly, lesions associated with *P. ramorum* on coast live oak (*Quercus agrifolia*) correlated with the timing of bud burst and initiation of cambial activity, suggesting that

successful infection of hosts' vascular tissue by this pathogen requires availability of active cambial tissue (Dodd et al. 2008). Host phenology may also shape trade-offs between resource allocation to growth and defense. In Norway spruce (*Picea abies*), susceptibility to lesion-forming fungal pathogens increased during periods of bud development and growth, associated with decreased phloem nonstructural carbohydrates, and may suggest growth–defense trade-offs (Krokene et al. 2012).

For leaf-feeding forest caterpillars, high synchrony between larval emergence and host plant phenology, including bud opening, has been attributed to increased herbivore fitness, population growth, and outbreak dynamics (van Asch & Visser 2007). Early larval emergence can lead to starvation and high mortality rates in forest pest species, whereas later emergence may create conditions in which caterpillars must feed on leaves with reduced or altered nutritional content, impacting agent fecundity. For example, early and delayed emergence have each been shown to reduce fitness in the winter moth *O. brumata* (Tikkanen & Julkunen-Tiitto 2003), though defoliating agents vary in their dependence on host synchrony and tolerance of starvation (van Asch & Visser 2007). Shifting tree phenology may further determine the potential for host shifts or variation in host susceptibility to defoliation within communities. For instance, differences in host susceptibility to spruce budworm defoliation correlate with synchrony between trees' bud flush timing and budworm development (Nealis & Régnière 2004).

Phenological cues, such as temperature and photoperiod, can have differing controls on key developmental stages in agents and hosts, such as larval emergence, pathogen sporulation, or bud burst (van Asch & Visser 2007). Predictions of host–agent interactions under future climatic change require understanding of key phenological events determining agent fitness or host physiology, the degree of phenotypic plasticity within host and agent populations, and the extent to which agent populations may rapidly adapt to changing cues.

5. SYNTHESIS AND KNOWLEDGE GAPS

Examining the specific mechanisms that determine agent virulence and host susceptibility highlights commonalities in the impacts of global change drivers as well as where knowledge gaps exist in our understanding of altered forest pest and pathogen dynamics. For instance, in addition to the observed shifts in host susceptibility discussed here, global change impacts may locally eradicate or reduce certain hosts, which may influence existing disease or outbreak dynamics. Potential outcomes, which require further study, will depend upon whether other host or nonhost species respond to tree declines, generating community shifts (e.g., Metz et al. 2012). Though range shifts by tree species may occur more gradually than expansions by pests and pathogens, tree migrations may also generate novel interactions with endemic biotic agents at range edges, but possible impacts remain poorly understood. Further, underlying genetic structure, phytochemistry, and phenotypic plasticity in host populations have clear potential to shape agent range expansions and impacts on tree mortality in forest systems (e.g., Erbilgin 2019, Wilhelmi et al. 2017), and additional study is required to understand the extent and determinants of spatial or temporal variation in host susceptibility.

Accidental human translocations and introductions are perhaps the most pervasive global change threat associated with pests and pathogens (Aukema et al. 2010, Liebhold et al. 2012). The mechanisms determining these introductions are clear, and emerging policies emphasize sanitation, inspection at indicated points of entry, and quarantine. However, though these regulations and policy interventions may be effective, they cannot keep pace with the extent of growing global trade (Roy et al. 2014). Studies of which pathogens or pests may prove to be destructive invaders have made progress in identifying traits and phylogenetic relationships that predict potential

threats, but additional research is needed to help refine assessments of risks (Philibert et al. 2011). Lastly, changes in host availability or acute climatic changes have clear potential to initiate rapid evolution, host switching, or variation in the virulence of biotic agents, but this remains a poorly understood dimension of forest pest and pathogen dynamics (Hoberg & Brooks 2015).

The framework presented in this review, structured by the mechanisms that underlie agent virulence and host susceptibility, also highlights how forest management actions may target specific mechanisms driving disease or outbreak emergence (**Table 2**). These insights may be particularly useful when a primary mechanism is responsible for shifting disease or outbreak occurrence (such as in the case of introduced pests and pathogens, or diseases emerging primarily through anthropogenically altered host density) or when management interventions may be limited to a specific scale or timeframe due to logistical or financial constraints.

6. INTEGRATION WITH ECOLOGICAL THEORY

Synthesis of the existing literature emphasizes the importance of processes like dispersal, stand development, disturbance history, and inter- and intraspecific competition in determining the impacts of pests and pathogens on tree mortality. These processes have been the focus of ecological theory. Though not always focused on forest, pest, or pathogen systems, existing ecological frameworks have the potential to improve predictions or modeling of the factors that determine global change impacts on epidemics and outbreaks. Here, we review three core concepts from ecology and their potential to improve understanding of the impacts of global change on biotic agents, their hosts, and environmental interactions in forest systems.

6.1. Metapopulations and Island Biogeography: Predicting Agent Occurrence and Persistence

Metapopulation theory, metacommunity theory, and the theory of island biogeography describe patterns of species occupancy and community richness within patches of suitable habitat as a function of dispersal distance, competition, and patch size or quality (Levins 1969, MacArthur & Wilson 1967). Patch occupation increases with dispersal or colonization rates and decreases with competition and local extinction rates. More recently, these theories have been applied to understand the drivers of microbial community composition, pathogen occurrence and dispersal, and disease emergence in host populations (Borer et al. 2016, Mihaljevic 2012, Peay et al. 2010, Seabloom et al. 2015). In pest- or disease-related applications of these models, individual hosts (or individual host cells) may represent patches or islands of suitable habitat that are infected or uninfected. At a larger scale, patches may be visualized as stands of susceptible tree hosts. A key exception to the application of metapopulation theory is the fact that host patches may also become locally extinct owing to disease or pest impacts on tree mortality (Borer et al. 2016). In general, however, these extensions of theory may help inform the ecological implications of global change on forest pest and pathogen dispersal, host density, and host connectivity via changes to colonization, extinction rates, and patch sizes.

Given that propagule pressure can strongly determine species occurrence in microbes (e.g., Peay et al. 2010) and that dispersal traits may play a key role in the invasive potential of forest pathogens (Philibert et al. 2011), metapopulation and biogeography theory could advance prediction of which insects and pathogens will become problematic when introduced in new habitats. Economic activity and policy controlling quarantine, plant hygiene, and agent detection can be visualized as manipulations of propagule pressure or dispersal distance and readily extended to management (Thompson et al. 2016). Patterns of pest and pathogen dispersal and the composition

or orientation of host patches may also shape the coevolution of trees and their biotic agents, influencing spatial patterns of host resistance and agent virulence depending on these groups' rates of contact and extinction (Laine et al. 2011, May & Nowak 1994).

Further, metapopulation theory and island biogeography may also be useful in predicting endemic forest pest and pathogen outbreaks that emerge from specific foci to invade at local to regional scales. Disease and insect outbreak occurrence is influenced by changes in the size and spatial relationships between continuous patches of suitable hosts, which are impacted by drivers of global change, as described above. Increases in host density and dominance may form larger and more suitable contiguous host patches that facilitate emergence of an eruptive insect or pathogen outbreak. For example, western and eastern spruce budworm (*Choristoneura freemani* and *Choristoneura fumiferana*, respectively) insect outbreaks are influenced by changes in host density and age from stand to landscape scales owing to local population intensification and spread among stands (Bouchard & Auger 2014, Flower et al. 2014). Concepts from metapopulation theory may also predict how endemic and novel emerging outbreaks may change over time, as host patches decrease in size or become more spatially isolated owing to mortality (Burdon & Thrall 2014).

6.2. Competition: Predicting Host Stress

Competitive interactions can result in water, nutrient, and light limitation for individual plants, leading to stress, depressed effectiveness of defenses, and increased susceptibility to pest and pathogen impacts (see the section titled Host Physiological Condition) (Anderegg et al. 2015, Herms & Mattson 1992, McDowell et al. 2011). Arguably, competition is one of the ecological concepts most integrated in the study of disease or outbreak dynamics and the larger effort to predict global change-induced tree mortality (Allen et al. 2015, Millar & Stephenson 2015). A range of forest-thinning treatments are frequently directed at reducing competition-related stress to mitigate tree mortality and enhance stand resilience (Cobb et al. 2017, D'Amato et al. 2013, Fettig et al. 2007). For instance, bark beetle pest impacts can be managed by reducing competitive stress, which in turn may increase production of defensive compounds and improve plant–water relations (Fettig et al. 2007).

Nevertheless, there are limits to the role that competition and host stress play in determining disease and outbreak emergence. Many invasive as well as native insects and pathogens kill healthy trees and forests, regardless of stand density or competition-related stress. At the other extreme, many insects and pathogens play no role in mortality and emerge only on trees that, through resource limitation, cannot defend against otherwise insignificant biotic agents (**Table 2**). Sitting midway between these extremes is a broad range of insects and pathogens that are opportunistic and emerge as causes of mortality with variable degrees of host resource limitation, which may be best predicted by competitive interactions and potentially mitigated by management actions designed to reduce host stress.

In a synthesis of 14 tree mortality events from a global distribution that included native and exotic pests and pathogens, Cobb et al. (2017) found that management to change forest density was the most frequently applied approach to increasing forest resilience to drought and/or outbreak. Management aimed at changing species composition was the second most frequently applied approach and was applied in cases where community factors drove pathogen or insect population dynamics and impacts. Manipulation of community composition is likely the superior approach when insects or pathogens cause mortality independent of host stress, but community composition itself drives outbreak dynamics (Cobb et al. 2017, Meentemeyer et al. 2012, Perkins & Matlack 2002). However, integrating concepts related to stand-level competition and the role of stress in determining host susceptibility may improve prediction of the effectiveness of management responses in mitigating tree mortality (D'Amato et al. 2013, Hartmann et al. 2018).

6.3. Succession: Predicting Temporal Patterns of Susceptibility

Ecological succession describes the assembly of biological communities following perturbations and their development over time. These changes in forest composition, tree demography, size, and spatial structure have important implications for host and community susceptibility across spatial scales. Leveraging concepts from ecological succession, including the roles that species dispersal, species traits, disturbance history, and site characteristics play in determining community assembly, may inform pest and pathogen dynamics in heterogeneous systems impacted by altered disturbances and land use change.

Succession following natural or human-caused disturbances can drive the spatial occurrence of individual hosts, connectivity between hosts, or spatial pattern in patches of susceptible habitat (Meentemeyer et al. 2012). For instance, mortality caused by the endemic root pathogen *P. weirii* is tied to changes in root-to-root connectivity that increase with canopy closure and increased root-to-root contact with neighboring trees (Hansen & Goheen 2000). Outbreak drivers may also be a function of stand age or development at the time of invasion. For the native pathogen *Heterobasidion irregulare* in Yosemite Valley, root-to-root connectivity at the time of pathogen establishment determined the extent of individual disease centers for decades following initial emergence (Rizzo et al. 2000). Host age and size distribution, which may covary with time since disturbance, can also influence the dominant biotic agents driving mortality in forest stands (Cobb et al. 2012, Flower et al. 2013). Heart rot pathogens can become more important as stands age, a reflection of the dependence of these organisms on the chemical and physical characteristics of heartwood, a component of older trees. As forest management has intensified and stand age has generally shifted to younger age structure, the principle challenges in forest pathology have shifted from slow heart rot pathogens to root pathogens (cf., Hartig 1894).

Changes in susceptibility driven by succession can also follow an opposite temporal trend; younger stands or early successional species can have greater susceptibility, which declines as these species age or are replaced by later-successional species. Fusiform rust, a native pathogen of loblolly pine (*Pinus taeda*), has lower impacts on the later-successional longleaf pine (*Pinus palustris*), and disease severity would likely decrease with stand age at the landscape scale because of either successional change or management (Randolph et al. 2015). Despite notable exceptions for very long-lived clones, root pathogens such as *Armillaria* and *Heterobasidion* are often more important in younger stands (fewer than 50 years) because inoculum accumulates on and spreads from buried wood, particularly postharvest residues (Rizzo et al. 2000). The underlying mechanisms determining how insect or pathogen impacts change with age or size class distribution may vary across systems and are often poorly understood. However, larger trees may represent larger targets or nets for interception of aerially dispersed inoculum, age-related changes in bark texture may facilitate establishment on the bole, and/or age-related changes in tissue chemistry may influence within-plant insect and pathogen growth and development (Garnas et al. 2011, Metz et al. 2012, Raffa et al. 2008).

7. CONCLUSION

Insect and pathogen dynamics are important drivers of changing patterns of tree mortality at the global scale. Understanding, predicting, and responding to emerging and changing disease and outbreak events are critical for sustaining forest habitats, diversity, and resources valuable to human populations (Allen et al. 2015, Ghelardini et al. 2016, Millar & Stephenson 2015). However, drivers of global change do not occur in isolation, and their interactions may obscure a mechanistic understanding of changing pest and pathogen dynamics, threatening to slow conservation and management responses (Cobb et al. 2017, Hartmann et al. 2018). We argue that an explicit

examination of the underlying mechanisms that separately determine virulence of biotic agents and the susceptibility of hosts (**Figure 1**) may improve our understanding of disease and pest impacts in rapidly changing forests. This approach identifies gaps in our existing understanding of agent–host interactions and allows explicit comparison between insects and pathogens (which also interact or co-occur). This framework also highlights instances in which a single mechanism may be the primary driver of changing dynamics (**Table 2**), which may form the basis of effective forest management. Clear examination of these underlying mechanisms additionally creates an opportunity to leverage broader ecological theories, describing dispersal, competition, community composition, and succession, to improve our understanding of the impacts of global change on disease and outbreak emergence.

FUTURE ISSUES

1. Both host susceptibility and agent virulence are impacted by a diverse set of global change drivers including climate change, land use change, species invasions, and human-impacted disturbances; this necessitates an integrative approach to understand and forecast future patterns of forest tree mortality.
2. Shifts in the occurrence of biotic agents can occur rapidly owing to their short generation times and dispersal abilities; however, our understanding of more gradual changes in host tree occurrence, associated with range shifts or other human impacts on forest composition, and their implications for future pest and pathogen dynamics can be greatly improved.
3. Models from plant physiology and forest health fields that integrate biotic and abiotic stressors tend to be overly simplistic or general, limiting our capacity to forecast forest mortality at scales relevant to management.
4. Under changing climates and increasing human translocations of both hosts and agents, the potential for host jumps and the rapid evolution of biotic agents remain important knowledge gaps for predicting changing pest and pathogen impacts.
5. General ecological theory can advance understanding and prediction of biologically driven tree mortality, yet leveraging these advances is likely to require thinking about familiar ideas in forest pathology, entomology, plant physiology, and community ecology in different ways.

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