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FIRE SCIENCE AND MANAGEMENT (ME ALEXANDER, SECTION EDITOR)

# The Intertwined Problems of Wildfire, Forest Disease, and Climate Change Interactions

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## Abstract

**Purpose of Review** I aim to contextualize wildfire-disease interactions with the goal of building a better understanding of where these may be of ecological importance and problems for sustainable forest management.

**Recent Findings** While wildfire-disease interactions have been documented, they are not well represented in the ecological literature, probably because they require considerable effort or serendipity to rigorously quantify. Examples of disease-fire interactions are relatively limited and tend to be clearer in systems where fire and disease are management problems. The most resolved systems include *Phytophthora* pathogens although wildfire-disease interactions are not limited to these pathogens. Documented interactions encompass a range of effects which include the magnification of problems associated with each disturbance. Wildfire-disease interactions are also likely to shape basic ecological function in systems where both wildfire and disease are common but not necessarily critical management problems. Climate change has altered the fundamental controls on both fire and disease suggesting it will also alter the magnitude and likelihood (occurrence or detection) of disease-fire interactions.

**Summary** I present a framework for linking wildfire-disease interactions and highlight the importance of host community/fuels structure on linking and mediating these interactions. I provide a series of examples where understanding interactive effects, interfacing with climate change, and the magnitude of changes to wildfire and disease intensification are of practical value and/or advance basic ecological knowledge. While much remains to be understood about these interactions, I make the argument that, in some cases, management can address both problems simultaneously.

**Keywords** Disease triangle · Non-additive effects · Disturbance interactions · Management

## Introduction

Wildfire and forest disease are components of rapidly changing ecological systems and while both are components of healthy ecosystems [1, 2], they are also emerging problems alone and in combination. Numerous examples illustrate the risk each disturbance poses to a diverse set of timber and non-timber forest resources as well as public safety [3–5]. We know less about how these events interact, the ecological conditions where interactions should be expected, and if those interactions rise to the level that they justify intervention [6, 7••, 8]. This uncertainty is a practical problem. Wildfire is an increasingly costly challenge in natural resource management by measures of ecological impact, severity, and cost of suppression [3, 9]. At the same time, a steady introduction of exotic pathogens to evolutionarily naive host communities and reemergent native pathogens has decreased forest biomass and often transformed forest structure and composition in ways that are likely to interact problematically with wildfire [4, 10]. Cutting across these problems is the issue of climate change which has potential to magnify the impacts of disease and wildfire [5, 6, 11]. Wildfire, climate change, and forest disease (both exotic and native pathogens) are disturbances with impacts at broad spatial scales and all three frequently overlap; thus, interaction could become more common in the near future [4, 10–12]. Given that these changes are occurring in a period

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of accelerating environmental change and stretched management resources, it becomes critical to ask: Do these interactions matter? Put another way, do interactions between wildfire and disease in the context of climate change alter the magnitude or management challenges associated with each disturbance? If so, what mechanisms are responsible for these changes? Lastly, do the answers to these questions provide guidance to strategizing or prioritizing management actions?

I attempt to inform these questions by leveraging understanding of wildfire and forest disease interactions in the context of climate change. I largely approach climate change as a backdrop upon which wildfire and disease interactions emerge or evolve; this limits the scope to one where climate change is primarily unidirectional in its effects on these disturbances, although feedbacks on climate change can certainly occur via carbon release or changes in carbon sequestration stemming from either or both fire and disease [13–15]. I also confront the relative dearth of direct tests of wildfire-disease interactions while using a robust literature on each disturbance individually to envision the mechanisms of their interaction as well as to identify when and where these could be important [5, 12]. Furthermore, the implications of climate change to both disturbances have received increasingly robust attention; these advances provide the basis for informed speculation on how each and their interactions may change in the future [3, 7••, 11, 16]. Mostly due to the practical difficulties of measurement, wildfire-disease interactions have not been well documented. However, community structure and compositional changes resulting from each disturbance provide the basis to identify systems where these interactions are most likely to emerge and where they may be the most important on longer timescales. I also present reasons for a positive outlook on what can seem like an existential threat posed by anthropogenically driven changes to the environment: While wildfire, disease, and their interactions can be intractable problems in some systems, the tools and approaches for addressing one often overlap or foster success in the other.

## Fully Reciprocal Models of Wildfire and Disease

While disease and wildfire ecology have developed on separate lines of inquiry, they share a parallel in that each are commonly envisioned as a fully reciprocal set of interactions among three broad component categories (Fig. 1). The resulting triangular renderings, known as the respective disease and fire triangles, are widely employed as teaching tools, to organize research, and structure adaptive management to emerging fire and disease problems. In order to use these models efficiently here, we must recognize first that they collapse a wide range of potentially interactive mechanisms that influence the occurrence and severity of each disturbance (Table 1). This kind of simplification is of great utility for engaging students and structuring mechanistic or process-driven inquiry [5, 17, 18]. However, alone these simplified models describe everything and nothing in particular. This approach risks a glossing over of the complexity of emergent processes driven by non-linearity, informational and material legacies, and interactions among important disturbances [2, 8]. At the onset, these fully reciprocal models are a set of black boxes which encompass a complex network of positive and negative feedbacks, hysteresis, and escalating orders of interaction limited only by the number of drivers and their interdependence. This risks the facilitation of a false mechanistic understanding of the interactions and inefficient or ineffective management interventions that follow.

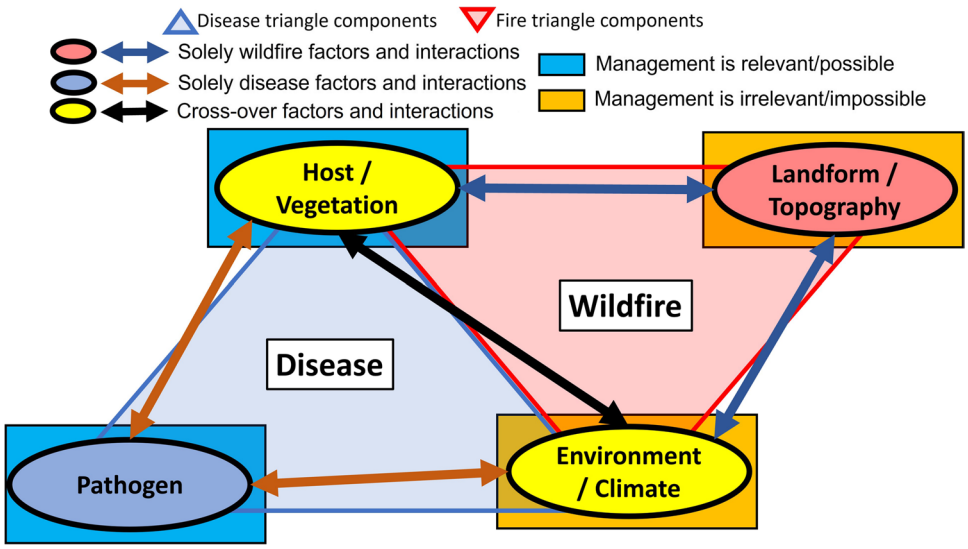


**Fig. 1** The disease and wildfire triangle frameworks rendered to illustrate overlap between the drivers of each disturbance. Each framework is depicted as fully reciprocal (arrows are two-directional) although for any individual connection between components a very broad range of causal directions, number of interactions, or effects are possible

**Table 1** Some examples of specific characteristics or dynamics encompassed by the components of the disease and fire triangle at the fire environment/fire regime scales. Overlap in these factors is noted including where an overlapping category ("host," etc.) includes a characteristic which is solely relevant to one disturbance

| | Wildfire | | |
|---|---|---|---|
| Disease | | | |
| Pathogen | Host/vegetation | Environment/climate | Landform/topography |
| Host range (number of species possible to infect) | Host competency–host capacity to transmit infection (disease only) | Temperature | Slope |
| Environmental resiliency such as dormancy | Degree of disease impact (mortality vs. growth reduction) | Precipitation | Aspect |
| Organism size vs. colonization capacity | Vertical structural vegetation change | Soil moisture | Topographic moisture variation |
| Virulence (capacity to infect hosts) | Live and dead organic matter dynamics | Timing and duration of temperature maximum and minimum | Topographic solar interception variation |
| Pathogenicity (capacity to cause mortality or morbidity) | Physiological impacts of infection including secondary chemistry | Relative humidity | Topographic influence on convective or radiative heating |
| Molecular and cellular mechanisms of infection and pathogenicity | Species-level flammability characteristics | Precipitation form (rain vs. snow) | Deposition of embers or smoke carried spores |
| Dispersal constraints (including factors influencing dispersal in wind or precipitation) | Degree of biomass loss (mortality and dead fuels) | Precipitation seasonal timing duration of precipitation events, frequency of events | Topographic or regional impacts to wind velocity and direction |

Despite their simplicity and limits, these models have a track record of success in structuring and organizing inquiry that has accelerated development of practical management solutions [17, 18]. In light of these successes, it may seem odd to criticize an approach which systematically structures inquiry and cause-and-effect understanding. However, the focus here is on interactions, which can be emergent phenomenon resulting from effects and dependency such as feedbacks and non-linearity. These kinds of models represent a common duality of ecological process: Cause and effect directionality can be simple or complex. A helpful illustration of this, as well as the risk of oversimplification of a complex process, can be gleaned from graph theory which seeks in part to identify pathways of effects, dependencies, and interactions which result in common outputs. These insights are especially useful in computer science where they can help identify simple computing pathways and restricting multi-step procedures that accomplish the same outcome [19]. This characteristic of complex systems—both simple and complex pathways result in the same outcome—is also known as model equivalence and is essential to efficient computing. However, restricting an arbitrary universe constructed from computer code is possible while the power to exert this level of control is rare or impossible in forest management and other ecological systems. Yet, model equivalence is probably prolific in ecology. This implies that a simple set of factors, as much as a complex set of factors, will lead to an outcome of interest such as interactions between disease or wildfire that influence the frequency, magnitude, or intensity/severity of one or the other [7••, 8]. It is possible to use this simple model effectively (Fig. 1), but it is important to recognize that it will sometimes obscure convoluted pathways of interaction (Table 1). At worst, this would mask cause and effect directionality and lead to their conflation.

With informed caution in hand, these models can be used to contextualize the mechanistic interactions of wildfire and disease while highlighting overlap in the structure and components of each disturbance. One of the most obvious implications of conjoining the wildfire and disease triangles is that half the component categories lie within each system and that the other half lie solely with one or the other (Fig. 1). Forest vegetation has a central importance for both wildfire and disease. In the wildfire triangle, vegetation encompasses live and dead fuel loading which itself is a function of vertical structure, density, productivity, senescence, mortality, and species composition [14, 20, 21•]. Vegetation structure and composition at the community level is equally central to understanding forest disease dynamics and impacts as it determines stand-level host competency (transmission capacity), species identity determines the likelihood of mortality or growth reduction, and density, biomass, and or spatial arrangement can determine the likelihood of disease emergence [4, 22, 23]. Disease is also a powerful way to transition fuels from live to dead pools which can influence flammability [2, 24–27]. Although changes in vegetation can have opposite effects on one of these disturbances compared to the other, it

is unsurprising that manipulation of vegetation is a central focus of management for both fire and disease [2, 27–29]. Environmental variation, here also encompassing climate, has a similar shared importance and centrality for both fire and disease [3, 5, 30, 31]. This category of effects is quite diverse for both disturbances although its importance, again, is unlikely to surprise the informed reader. Combustion is a heat and moisture influenced process and biological function—such as the sporulation of a pathogen—is often controlled by temperature and limited by water. A complex and dynamic set of climate variations can be critical to emergence of both disturbances (Table 1) including the timing, form, and range of variance in precipitation, analogous temperature variables, and edaphic factors such as soil characteristics that determine moisture availability [12, 32, 33•].

The second set of components which lie solely within one framework or the other encompass topography (landform) and pathogens. Topographical effects are not ignored by the field of forest pathology but are traditionally grouped within "environment" to reflect the importance of water availability and temperature on pathogen biological dynamics [4, 30, 33•]. For example, topographic influences on vadose zone water flow can influence dispersal for pathogens adapted to aquatic life histories, which includes soil-borne *Phytophthora*, some of the most globally important forest pathogens [30, 33•, 34]. But in this case topography is determining environmental conditions—moisture levels and flows—critical to pathogen movement and survival. In fact, for many *Phytophthora*-caused diseases, drainage and contact with standing water is a more biologically meaningful measure of disease risk [33•, 34]. Treating topography (landform) independently for wildfire is important as it is critical to spatial patterns of heat exchange and the occurrence of problematic weather phenomenon such as katabatic (downslope) winds [2, 31]. This landscape factor also influences smoke dynamics, convective, and/or radiative heating which has potential crossover effects on the viability of some pathogen inoculum not accounted for in my depiction of the two processes [35, 36]. Lastly, topographic controls on wildfire dynamics and energy transfer can be so strong that they overwhelm dampening characteristics of vegetation or other environmental factors making them central to maintaining safety during firefighting operations [37].

Similarly, the category pathogen lies squarely outside the wildfire triangle and encompasses a diverse range of organisms and associated ecologies. Of course, fire can directly impact pathogens either by reducing inoculum in living tissues (host/vegetation) or by consuming inoculum reservoirs in dead organic matter [36, 38, 39]. A further contextualization of the biological agents of disease (pathogens) with wildfire reveals several fundamental differences between the two disturbances. The timeframes in which pathogens spread, reduce growth or kill trees, as well as their size, capacity to survive climate variability, and dispersal constraints often lack simple or accurate analogies to fire [30, 34, 40, 41]. Within my rendering of the combined fire and disease triangles, the mechanisms, evolutionary dynamics, and processes that confer pathogenicity also occur at a uniquely small scale (cellular and molecular) which should not be ignored as new tools to limit pathogen spread and impacts are often developed at the gene or transcription level [42–44].

Uniting the disease and fire triangles provides a framework to envision how various factors connect, interdepend, or potentially interact. However, this rendering does not suggest the possible outcomes of these interactions which, a priori, can be positive or negative as well as relatively weak or strong [45]. For example, pathogen-driven changes in biomass or species dominance can be expected to dampen or increase wildfire depending on what species are favored by disease and how disease redistributes biomass [2, 6, 26, 27]. Similarly, fire-driven changes in vegetation can be expected to have a similar range of effects, both positive and negative, on disease dynamics through direct effects on vegetation as well as pathogen populations [6, 7••, 38, 45–47]. Fire return intervals as well as disease importance vary widely across forest systems meaning that either wildfire or disease impacts will be irrelevant to a range of forest characteristics of interest or whole forest types (Fig. 2). For example, some wet tropical and wet sub-boreal systems can have wildfire return intervals so long (several centuries to millennia) they are much more likely to be structured by other factors such as disease, insects, and or anthropogenic factors associated with social upheaval and warfare [48, 49]. Equally so, many pathogens may not alter equilibrium punctuated by stand-replacing wildfire (Fig. 2, *Pinus muricata* and other closed cone forests). Wildfire and disease may also overlap on timescales which are difficult to reconcile from a management perspective. For example, compared to disease human actions such as vandalism, irresponsible cutting, or arson (which may overcome fuel limitation) may be a much more pressing and novel threats to the oldest known living trees (Great Basin bristlecone pine—*Pinus balfouriana*) than their endemic pathogens (Fig. 2). Lastly, climate change is not explicitly rendered within the combined fire-disease triangle framework, yet it directly alters or modifies the effects of each category [3, 5, 50]. This creates the likelihood that the relative strength of fire and disease interactions, the category of effects which drive these interactions when they do occur, and/or their ecological impacts will change over time [2, 6].

Clearly the potential for wildfire-disease interactions appears plausible in a simple rendering of their overlap (Fig. 1) as well as in a more detailed examination of the components within these broad categories (Table 1). A reasonable argument can be made that these interactions will not occur or be important everywhere that either wildfire,

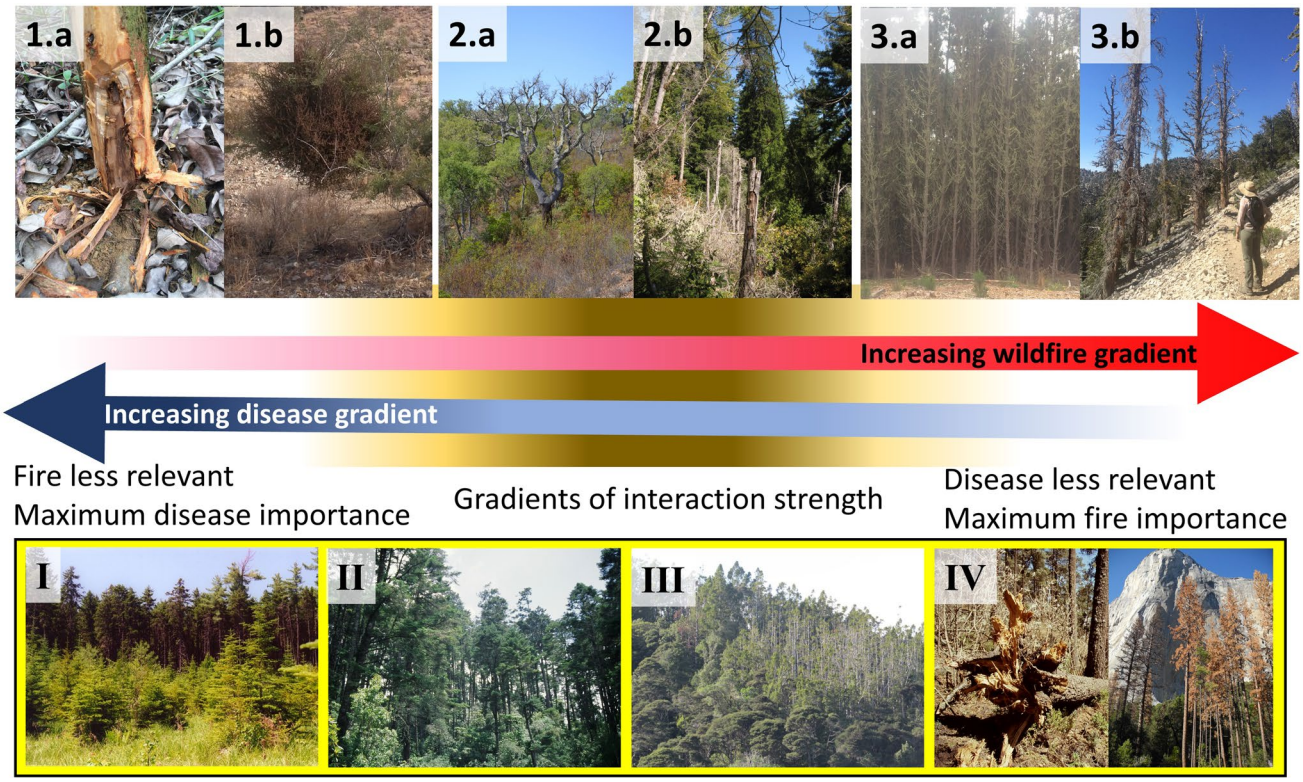


**Fig. 2** Wildfire and disease dynamics across a wide-ranging set of woody plant communities (1–3) along with a set of examples where climate change could increase importance of wildfire-disease interactions (I-IV). Examples include *Phytophthora cinnamomi* in ignition-limited tropical forests (1.a, near Hanoi, Vietnam), *Phoradendron* sp. mistletoe in fuel-limited Mojave woodlands (1.b, *Senegalia greggii*, near Barstow, CA); *P. cinnamomi*-driven canopy mortality and increased shrub dominance (2.a, *Quercus suber*, Algarve region of Portugal) sudden oak death changes to live and dead fuels (2.b, Big Sur, CA. Photo: K. Frangioso); dominant effects of wildfire in Bishop Pine (3.a, *Pinus murcata*, Point Reyes, CA) and Great Basin bristlecone pine (3.b, *Pinus balfouriana*, White Mountain, CA). Examples where climate change could magnify impacts include ignition-limited wet northern forests (I, Weymount point, ME), humid tropical montane forests (II, *Abies guatemalensis*, Cantel, Guatemala), broad-leaved temperate rainforests (III, Kauri dieback, Waitakere Ranges Regional Park, New Zealand), and fuel-limited dry-conifer forests (IV, Iztaccíhuatl, Mexico and Yosemite National Park, CA). All photos are by the author unless otherwise credited. See text for discussion

disease, or both occur. However, an equally convincing argument can be made that these interactions can be masked by the inherent complexities of each system and even more so via the potential for feedback between these common and impactful disturbances.

## Evidence and Mechanisms of Fire-Disease Interactions

In an ideal world, the field of forest management could rely on a broad set of rigorously quantified wildfire-disease interactions to contextualize and dictate the need to leverage resources to address them. Regrettably, this does not describe the state of the science. While we have many examples of fire and disease impacts alone, in only one case are the interactions between these disturbances documented across multiple spatial scales and from multiple cause and effect directions at the system level. In a few other examples, we have evidence of consequential interactions for fire, disease, or vegetation structure, but in most cases, we have potential mechanisms of interaction without clear documentation that these occur.

At the system level, sudden oak death in California is arguably the best documented example of disease and wildfire interactions in a forest system in that impacts of both disturbances are well quantified, interactions have been documented at spatial scales ranging from individual to landscape, and mechanisms of interaction are relatively well elucidated in each example. Here, wildfire-disease interactions have been demonstrated to alter both disease and fire dynamics in distinct ways, although in this case the more substantial ecological effects appear to be a magnification of wildfire impacts [7••, 14, 21•, 51, 52]. In the context of Fig. 1, the mechanisms altering wildfire impacts are realized via disease-driven changes to fuels/host dynamics with some direct negative effects of fire on the pathogen. Caused by the generalist pathogen *Phytophthora ramorum*, sudden

oak death has killed at least 42 million trees in broadleaved forests of California and Oregon after if first emerged circa 1990 [4]. The pathogen has infected at least 166 million more trees but only ~ 2.2% of apparently at-risk biomass has been killed as of 2019 suggesting an already consequential disease could grow to an even greater problem in a region also well known for devastating and costly wildfire [3, 51]. My colleagues and I have been serendipitously able to measure wildfire-disease interactions for the 2008 Basin Fire in a landscape-scale survey initially designed to quantify disease [7••, 14, 21•, 52]. At the stand level, the disease has been shown to increase fire severity during a transient period of higher canopy fuels [51]. As these fuels transition to the surface, tree mortality increases in species and individuals which would otherwise be resilient to wildfire [52]. This increase in surface fuel also exacerbates loss of soil carbon and nitrogen at the ecosystem scale while increasing available phosphorus [14] which could shift species dominance over the longer term. Lastly at the stand level, disease generated fuels and associated increases in soil burn severity can overwhelm below-ground survival capacity in communities and species which are highly adapted to fire [21•]. These analyses paint a picture—in this case—of interactions between wildfire and disease which primarily, but not exclusively, increase the impact of wildfire. There is also no epidemiological or pathological reason to expect these interactions are specific to *Phytophthora* given that these kinds of disease impacts co-occur with wildfire in many other systems. For *P. ramorum*, and likely others where they may occur, the mechanism of interaction has primarily been fuel-driven changes to fire dynamics which shift over time from canopy-fuel effects to those driven by surface fuels. This body of work also found evidence of transient negative impact to pathogen populations at the stand level which recovered to pre-fire levels within a few years [36]. Furthermore, for *P. ramorum*, the frequency or strength of wildfire-disease interactions also appear to shift over time with changes in host prevalence [7••].

While most *P. ramorum*–wildfire interactions have been documented at the stand level, evidence has accumulated for their importance at greater spatial extents. During the 2008 Basin Fire, stand-level disease intensity was positively associated with fire impact to adjacent stands, even if these adjacent stands were not dominated by susceptible species or were not disease impacted [53]. Greater fire impacts also appear to slow forest recovery which appeared to suppress disease-driven mortality in the 8 years of subsequent forest recovery [54•], results which mirror those documented at the stand scale [7••]. In both cases, heat, fire dynamics such as intensity, and smoke impacts have been invoked as possible mechanisms of these spatial dependencies. While these mechanisms have not yet been directly quantified at broader spatial extents, the overall patterns of ecological impact match those at the stand level where fuel loading, distribution, and consumption have been quantified pre-fire and post-fire.

In each of the aforementioned studies, *P. ramorum*–wildfire interactions are non-additive, that is, they are interactive effects which cannot be predicted based on knowledge of only one or the other disturbance: The disturbance impacts are synergistic. Importantly, these interactions are driven by different mechanisms, depending on the stand, ecosystem, or landscape characteristic in question. As previously noted, changes in fire severity can be influenced by the amount and position of fuels in the vertical stand profile [2, 27]. Dead foliage in the canopy, and consequently low foliar moisture, appears to be important in driving fire severity and the associated impacts [20, 26]. However, dead canopy fuels rapidly transition to the surface where they appear to be drivers of below-ground mortality and soil impacts [14, 21•, 52]. Once disease-caused mortality occurs in this system, a fairly predictable shift of risk from canopy to surface fire impacts occurs at the tree-level over a few years [20, 26, 51, 52]. Disease, the driver of mortality and thus fuel accumulation, is temporally and spatially dynamic with inherent prediction complexities [55–57]. *Phytophthora ramorum* is an airborne pathogen of leaves, twigs, and tree boles in this ecosystem and is highly sensitive to interannual variation in temperature and moisture [55, 58, 59]. Host communities also vary in their collective capacity to generate inoculum and, for yet unknown reasons, tree-level mortality rate increases with tree size [25]. As a result, mortality rates vary among years, within stands, and across landscapes with variation in spring rainfall, host community composition, and stand structure. Sudden oak death can be intense and is progressive, but it is not uniform and its interactions with wildfire are also likely to vary with disease heterogeneity [6, 56].

*Phytophthora cinnamomi* is arguably the next best understood example of wildfire-disease interactions. Similar to *P. ramorum*, the mechanism of interaction appears to be realized by influences on fuels/host community, but unlike *P. ramorum*, direct effects of fire on pathogen dynamics may be more substantial. *Phytophthora cinnamomi* is widespread in Mediterranean and warm temperate forests where its broad host range, long-lived survival structures, and high degree of pathogenicity cause a range of disease severities across many forest communities [33•, 60]. Disease impacts to forest structure and species composition can be great enough to transform ecosystem types (Fig. 2) [33•, 61]. Mortality can increase dead fuels, disease-driven changes in species composition can facilitate fire, and fire also directly stimulates pathogen populations by enhancing germination of long-term soil survival structures [62, 63]. In a study of Australian *Eucalyptus*/*Banskia* woodlands, decreasing time since fire was found to be associated with greater disease impacts, suggesting a positive feedback between disease

and wildfire could interact to transform ecosystems from forests to a shrub and non-woody plant-dominated community [33•, 63–65]. Considering the long survival times of *P. cinnamomi* resting spores and broad pathogen host range, these changes are likely a long-term conversion of vegetation types. *Phytophthora cinnamomi*-caused mortality can be locally intense suggesting the disease could result in stand-level accumulations of fuels and potentially increase fire severity. This pattern does not appear to have been documented to date although this type of interaction could influence wildfire impacts throughout much of the Mediterranean basin given the widespread distribution of this pathogen.

Rigorous analysis of fuel dynamics could be worthwhile across several *P. cinnamomi*-impacted forests where additional environmental factors can modify wildfire and/or disease to result in interactive effects. In the southern Iberian Peninsula, *P. cinnamomi* is widespread, impactful, and actively spreading [30, 61, 66]; this region is also simultaneously experiencing shifting rural demographics which appear to increase vegetation density and accumulate fuel (Fig. 2) [67, 68]. In combination, these two landscape changes could result in increased fire impacts. The disease is also widespread in many Australian forest ecosystems where wildfire impacts have also been increasing, likely in association with intensified droughts and tree mortality which are themselves associated with climate change [50, 64, 69]. Lastly, *P. cinnamomi* is also widespread in the southeastern USA where it perpetuates the extirpation of American chestnut (*Castanea dentata*) in piedmont forests and impacts growth of economically important *Pinus* species; this region is also facing some degree of increasing wildfire impact [33•, 70] and therefore increasing interactive effects could rise to the level of management challenges. As a globally distributed pathogen, *P. cinnamomi* co-occurrence with wildfire creates many opportunities for their interaction.

While wildfire and disease frequently overlap, direct investigations of their interactions do not extend beyond these two *Phytophthora* pathogens, at least not for wildfire which can have high spatial variation in severity or temporal variability in frequency and severity. While our best examples are limited to *Phytophthora*, there is little ecological or epidemiological reason to expect these interactions are limited to this group of pathogens. This conjecture is supported by two examples of prescribed fire-disease interactions which inform our understanding of interactions in the context of wildfire and support the expectation of these interactions will occur and could be impactful in other forest systems. Oak-dominated forests which stretch across the eastern margin of the great plains region of North America have experienced changes in species composition and stand density in response to fire suppression which has led to increases in *Biscogniauxia* pathogens which cause bole canker diseases and *Bretziella fagacearum* (formerly *Ceratocystis fagacearum*) which causes a root and stem disease [71, 72]. Two experiments applying prescribed fire documented very different outcomes of prescribed fire-disease interactions. First, in a study in the cross-timbers region of Oklahoma, frequent fire was associated with improved host physiological status and lower incidence of *Biscogniauxia*-caused disease [71]. In a similar experiment in Minnesota, fire suppression appears to have increased stand susceptibility to the *B. fagacearum*-caused disease known as Oak Wilt. Here, disease-driven loss of canopy cover and shifts in community-level fire-resiliency along with repeated prescribed fire caused a long-term shift in vegetation type [72]. In the first example, loss of fire appears to alter host communities and intensify disease impacts, while in the second, fire is accelerating, and perpetuating stand structural and compositional changes caused by disease in a fire-suppressed system.

Considering the two *Phytophthora* diseases along with the prescribed fire examples, we have evidence for fire-disease interactions which (1) suppress disease, (2) increase disease, (3) intensify fire impacts, and (4) transform ecosystem structure over decadal time scales and plausibly centuries [7••, 63, 71, 72]. Why are these differences in interaction outcomes so disparate? Furthermore, in each example the primary mechanism of interaction is mediated via fuels/host community, and secondarily via direct effects of fire on pathogen populations. Will this relative degree of importance hold over a greater set of examples? Finally, climate change-associated changes in temperature and moisture are likely to modify many aspects of both wildfire, disease, and their interactions. Climate change impacts may also be primarily realized via fuel/host components such as fuel moisture or accumulation of dead fuels and secondarily via impacts to pathogen dynamics, but relative importance between the two categories will also likely be influenced by biological responses to changes in temperature and moisture. We can anticipate with some clarity why, how, and to what degree climate change is likely to increase wildfire frequency and severity in many forests, including those where disease has heretofore been far more consequential than fire (Fig. 2; Table 2). At present, we can also conclude with confidence that fire-disease interactions do occur but also that the implications of these interactions are diverse.

## Where Else May Fire-Disease Interactions Be Important Now or in the Future?

As previously highlighted, the trend of increasing wildfire frequency and severity [3, 12] suggests we can reasonably expect more overlap between fire and disease. This increased overlap may increase the frequency or importance of these interactions to critical natural resources. However, any attempt to better understand wildfire-disease interactions

**Table 2** Examples of documented and potential disease-wildfire interactions with associated mechanisms

| Disease system or pathogen | Disease-wildfire interactions | Interaction mechanism | Expected influence of climate change |
|---|---|---|---|
| **Examples with evidence of disease-wildfire interaction** | | | |
| *Phytophthora ramorum* (in California and Oregon) | Live and dead fuel accumulation increases fire severity and post-fire impacts [24, 51] | Pathogen-mediated shifts in canopy fuels (live and dead) and shifts in community structure; accumulation of surface fuels [14, 53, 103] | Increased fire severity may magnify interactive impacts including long-term community changes [21•, 52] |
| *Phytophthora cinnamomi* | Disease can dramatically alter live fuels creating communities with fundamentally different fire regimes [33•, 60] | Pathogen-mediated shifts in community composition. Fire enhances pathogen germination [63] | Pathogen range expansion with increased average temperature is expected [104–106] |
| **Examples where wildfire-disease interactions are likely** | | | |
| Mistletoes | Altered canopy fuels likely to increase fire-caused tree mortality [79, 80]. Intense fire also can decrease pathogen prevalence [27, 46, 47, 83] | Disease increases canopy and surface fuels; pathogen-infested trees may be more likely to die during fire; frequent or intense fire may reduce disease | Combined infection and climate stress should magnify host impacts; increased high severity fire may reduce pathogen prevalence |
| Heterobasidion root disease | Fuel accumulation may increase wildfire impacts while long-term canopy gaps may decrease fire impacts in conifer systems [23, 41] | Disease increases canopy and surface fuel in the short term; long-lived canopy gaps create fuel discontinuity | Climate stress expected to increase tree mortality; insect-disease-fire interactions are possible [16, 107] |
| Armillaria root disease | Likely similar to Heterobasidion, but in broadleaved as well as conifer systems [85] | Probably similar to Heterobasidion | Similar to Heterobasidion, but in broadleaved and conifer systems [108] |
| Laurel wilt disease | Disease may increase fire impacts via dead fuel accumulation; canopy species shifts may also interact with wildfire with unknown consequences [109] | Pathogen-mediated changes in vertical live and dead fuel structure; dominant species changes | Climate change-driven increased fire severity would make interactions more likely |
| Chestnut Blight | Disease has likely altered community composition in ways that may make interactions less likely [92] | Loss of the most strongly fire-associated species probably drives current wildfire dynamics [91, 96] | Increased temperature is expected to increase pathogen range and complicate species restoration [33•] |
| **Examples where climate change could increase the likelihood of wildfire-disease interactions** | | | |
| Beech bark disease | Although fire is currently, increased fire severity could result from disease impacts [90] | Disease-driven increases in canopy-fuel continuity (increased stand density) | Climate change-caused increases in fire occurrence make interactions more likely |
| Pine pitch canker | Disease may increase fire impacts by increasing dead canopy fuels and possibly surface fuels | Increased fire impacts are possible, but may be weak when fire is stand-replacing [110] | Interaction strength may weaken if climate change slows pathogen spread [111] |
| White Pine Blister Rust (western North America) | Dead fuel accumulation may increase fire impacts while lower stand density could decrease them [112] | Disease-fire interaction strength is likely to vary with fuel limitation | Climate change is expanding pathogen range and disease impacts [113] |
| Swiss needle cast | Fire impacts could be increased via fuel changes [16] | Disease may increase fine fuels and can change species composition | Simultaneous increases in fire and disease impacts are possible |

must attempt to anticipate where they will occur or be important to subsequently quantify their effects. Here, it is helpful to recognize several shared components of the studies which have thus far documented these interactions. First, each disease example is widespread and can be locally intense. Second, interactions were documented in sites or monitoring networks with either long-term data collection, or rich background information built from a history of research efforts on vegetation dynamics, disease, and/or wildfire. Therefore, the general lack of studies documenting these interactions may reflect limited research resources as opposed to a lack of ecological importance. It may also reflect the complexity inherent to how these interactions may emerge, a speculation supported by recent advances in understanding wildfire, tree mortality associated with insect outbreak, and dynamics in the wildfire environment including wind, temperature, and relative humidity during fire events [48, 73••, 74, 75]. Over the last several decades, interactions between insect outbreak and wildfire have been documented in several independent studies which also found interactions have variable effects—both intensification and dampening of wildfire impacts have been documented—but the dynamics so far have been primarily mediated by fuel/host community components and secondarily by environment during wildfire [8, 74–76]. The two disturbances also vary in time in a manner which influences the likelihood of their overlap, and thus the potential importance of their interactions [48]. The state of understanding gleaned from insect outbreak and wildfire suggests at least two broad expectations for wildfire-disease interactions. First, when interaction effects occur, these could be positive or negative and act on disease, fire, or both—a pattern thus far confirmed by empirical studies of wildfire-disease interactions [36, 52, 71]. Second, we should also expect that a gradient of importance between disease and fire (wildfire and prescribed) will influence the ecological importance of these interactions, possibly their occurrence overall, and environmental conditions during wildfire could overwhelm and mask non-additive interactions (Fig. 2) [48, 75]. Thus, the most critical knowledge frontier remains centered on one of the motivating questions of this article: When, where, and why do these common disturbances interact in an ecological meaningful way (Fig. 2)?

In our examples of *Phytophthora* wildfire-disease interactions, not only are disease impacts widespread and locally intense, these forests are also shaped by frequent or impactful wildfire. If this is a good guide for where interactions will occur, then semi-arid conifer systems, such as those which dominate the western USA, could be or are likely shaped by these effects via several disease systems. Diseases in these forests attack and modify a range of plant parts, have variable intensity in time and space, and include both invasive and endemic (native) diseases. Invasive *Dothistroma* and foliar *Phytophthora* pathogens can kill a substantial amount of the canopy without necessarily increasing surface fuels given that overstory mortality is less intense than the previous examples I have highlighted [77, 78]. Although these pathogens are very sensitive to interannual variability in rainfall and precipitation form (rain vs. snow), they can be expected to result in lower bulk canopy-fuel moisture in some years which could be impactful to interannual variation in canopy fire potential [2, 26].

A likely powerful driver of wildfire-disease interactions is represented by the broad body of native mistletoes, a widespread and impactful set of canopy pathogens in both conifer and broadleaved forests [27]. Mistletoes, in the genera *Arceuthobium* (dwarf mistletoes) and *Phoradendron* (leafy mistletoes), are widespread native perennial parasitic plants which cause canopy diseases less dependent on interannual climate variability compared to *Dothistroma* and canopy *Phytophthora*; these diseases also tend to intensify over decades. Mistletoe diseases can alter canopy architecture, reduce growth, and cause mortality, impacts which are well-known management problems in the case of dwarf mistletoes in conifer forests [16, 27, 79]. Several important dwarf mistletoe pathogens have a global distribution in fire-prone forests as well as forests where fire is expected to have increased impacts, making them prime candidates to examine for interactive effects (Table 2). Mistletoe pathogens attack the woody components of the canopy, compromise growth, can increase water stress, and have been shown to increase mortality and surface fuel loading [79–82]. Increased mistletoe impacts have been associated with fire suppression-associated densification of host communities and the absence of direct pathogen suppression by fire [79]. Furthermore, a body of post-fire surveys (both wildfire and prescribed fire) demonstrate diminished mistletoe pathogen populations, particularly following fires with relatively high levels of mortality or scorching [27, 47, 83]. Given documented disease-associated changes in fuels, as well as fire-driven reductions in pathogen populations, these diseases appear to hold great potential for a range of non-additive ecological interactions and could be fruitful areas for further investigation.

For both mistletoes and canopy diseases restricted to leaves (*Dothistroma* and some *Phytophthora*), it is plausible that disease-wildfire interactions could increase fire severity or alter the spatial patterns and intensity of disease. Furthermore, because several of these examples are endemic (native) pathogens and represent a range of disease intensities or outcomes (c.f. dwarf vs. leafy mistletoes), such interactions could represent deeper basic ecological function or unanticipated pathways for problematic impacts of global change. The set of documented wildfire-disease interactions imply that impacts to host density and surface fuels will be key drivers. Furthermore, these interactions are likely to be

dependent on the severity of disease and impacts of fire on pathogen populations in addition to temporal controls on disease progression and fuel transitions from the canopy to the surface [14, 26, 51]. The temporal dynamics of both fuels and canopy diseases suggest positive or negative feedbacks on fire, disease, or both, and that these interactions may shift overtime which may complicate efforts to detect or properly attribute them.

Wildfire-disease interactions are unlikely to be restricted to canopy diseases. Several long-lived root pathogens also hold potential for wildfire-disease interaction in semi-arid conifer systems, although the mechanisms of interaction and outcomes may differ substantially from canopy diseases. Several native root diseases, including those caused by *Heterobasidion*, *Armillaria*, *Pseudoinonotus*, and *Phellinus* root pathogens, are notable for the substantial size of these organisms, their long life spans, and potential or realized disease severity [23, 84–88]. Long-lived root diseases are likely to cause surface fuel accumulation which could increase wildfire impacts to soils and increase fire-associated mortality [14, 21•, 89]. However, these diseases can develop slowly, sometimes over decades, which has two important outcomes: (1) Canopy or surface fuel accumulation will be distributed over time and thus may or may not be substantial in any particular year at the landscape scale and (2) the canopy gaps created by these diseases are persistent for many decades [23, 41], a disease impact which is not comparable to the previous examples of canopy diseases. Persistent canopy openings may have the greatest implication for wildfire because they create fuel discontinuity and reduce canopy fuels overall. This latter effect is similar to some aspects of bark beetle impact although changes in canopy continuity from root disease can be persistent over much longer time periods [23, 74, 80]. Fire—both wildfire and prescribed—is less likely to suppress many of these diseases because they persist via below-ground inoculum on deeply buried woody roots or in soil, both reservoirs may be protected from heating or combustion [41, 85, 87]. While root-disease canopy gap expansion slows over time, root pathogens continue to infect the roots of neighboring trees which may increase drought stress or predispose individuals to insect attack [86, 87] creating the potential for even more complex wildfire-disease-insect interactions which at times increase fire severity, at times suppress fire, or in other cases have no effect. For example, surface fuel accumulation could alter soil burn severities [14], canopy openings may weaken fire contagion [74], or environmental conditions could overwhelm any potential interactions [75]. Fire suppression appears to be an important factor in the dynamics of many of these root pathogens, again primarily via alteration of host communities [86–88]. Similarly to mistletoe-caused diseases, root diseases are so frequent in fire-prone forests, they are probably useful or important disease case studies to reexamine through the lens of fire ecology [2, 6]. Furthermore, one well-replicated study demonstrated a reduction in disease following prescribed fire, likely due to reduction of inoculum in stumps and possibly stimulation of antagonistic soil fungi [39]. This result is counter to several of these expectations I have presented and should be considered an even stronger argument for investigating wildfire-disease interactions in root-disease systems.

Lastly, many of the most impactful diseases hold little potential for these interactions because fire rarely occurs in the associated forest ecosystems. Numerous examples from temperate forests demonstrate substantial changes in host community structure and composition that implicitly alter fuel amounts and species-specific fuel characteristics [90, 91]. Yet, fire return intervals have, historically, been so long in many systems that little or no effect of fire is evident in terms of either adaptation or current forest structure [92, 93]. To be clear, the modern structure and composition of this frequent scenario reflects the infrequency of wildfire, but this does not shield these forests from deleterious impacts of wildfire, or the potential for problematic wildfire-disease interactions. For example, montane tropical cloud forests such as those dominated by Guatemalan fir (*Abies guatemalensis*) and wet temperate rainforests would be expected to be shielded from wildfire impacts during climate and social eras where fire is uncommon [2, 49]. Here, native or invasive diseases are more likely to shape forest structure and evolution, and are important conservation or natural resource problems (Fig. 2). However, climate change trends, including anthropogenically driven warming and ecological drought [11], are likely to expand the range of wildfire to include areas where wildfire has been uncommon in the modern era [70, 93]. Thus, wildfire-disease interactions could emerge in many forests where they would otherwise be expected to be rare and where forests would be particularly vulnerable because of little or no evolutionary adaptation to fire. Similar to the Guatemalan fir example, Kauri (*Agathis australis*) is a species of concern threatened by habitat loss in a wet ecosystem where wildfire is historically infrequent. Kauri is also threatened across its native range by the emergent *Phytophthora agathidicida*, a pathogen epidemiologically similar to *P. cinnamomi* (Fig. 2) [94]. Here, even relatively small changes in wildfire impacts which emerge from interactions with disease could be highly consequential due to the general lack of adaptation to fire.

An even wider range of disease examples has shaped forest structure and composition in eastern North America, with a series of devastating diseases appearing to permanently alter many ecological processes and incur significant economic impacts (Table 2) [90, 95–97]. A legacy of disease impacts, emergence of novel pathogens, and expansion of wildfire impacts into historically fire-free ecosystems sets the stage for deleterious interaction effects. For even the

most conservative estimates of anthropogenically driven climate alteration [11], increased overlap of wildfire and forest disease must be expected including in systems where fire is currently relatively unimportant.

## Management: Doing Nothing Is Probably the Worst Option

At the onset of this article, I promised the reader reasons for a positive outlook. My optimism is rooted in the importance, so far, of host community effects on wildfire-disease interactions (Fig. 1). Of course, in my rendering, this is synonymous with fuels and the implication is that management aimed at host communities and/or fuels could yield benefits to both wildfire and disease problems. This is especially the case where fuel management alters community-level epidemiological drivers (and vice versa) [28, 29, 40]. Many characteristics of integrated disease and fire triangles cannot be altered by direct management, at least not at the stand level or with any degree of practically (i.e., topography). Most directly, environmental influences on disease, wildfire, or their interactions can be confidently expected to shift as climate change scenarios play out and stand-level management is unable to address this directly. One cannot make it rain or, for that matter, make the rain stop when those outcomes are what is most needed to protect a resource. Increased heat and drought will increase tree physiological stress which may in turn reduce or inhibit defensive responses, including to pathogens or insects which would otherwise be expected to be relatively inconsequential [11, 29, 98]. Yet, as is the case with most of the expected impacts of climate change, the worst outcomes may be avoidable, and tools are in hand to execute sensible responses.

To reiterate, the examples at hand point to the primary importance of host community composition (fuels) and secondarily, pathogens as the mechanisms of fire-disease interactions. Both components can be directly managed via typical stand alteration of density, community composition, or size class distribution [73••]. When designed and executed with epidemiological understanding, these kinds of stand manipulations can simultaneously address current fuel loadings, reduce current disease, and also reduce potential for future disease-associated fuel accumulation [28, 79, 99]. Furthermore, pathogen populations can be directly suppressed or eradicated via host removal, prescribed fire, or chemical pathogen suppression [38, 40, 100, 101]. Breeding of resistant lines or selection of resistant individuals within a population to increase resiliency may become more practical in the future [42], but any advance through resistance breeding will also likely need to be evaluated in the context of wildlife, wildfire, and climate change. It must also be noted that management outcomes which simultaneously address both fuels and disease are not preordained. Shifts to community or stand composition that address wildfire can create conditions ripe for disease outbreak and vice versa. After all, the components of the linked disease and fire triangles can themselves be an entangled collection of effects and dependencies (Table 1) implying that damaging positive feedback could be the outcome of management which otherwise seems wise [29]. Identification of outcomes which address both problems rely on a clear understanding across forest disease ecology and fire ecology, disciplines which will need to build a shared experimental mission in both research and management to make progress on this topic [102]. Deepening collaborations and performing the difficult work of linking what may appear as narrow and irrelevant knowledge from the outside is critical for successful collective responses. From the strict perspective of forest diseases, study of forest insect outbreak again provides an informative starting point and wildfire-disease-focused inquiry would benefit from confronting the plausibility of large-scale forest health goals [73••]. But likely most importantly, the experimental approach at the center of adaptive management will be critical to long-term successful management of disease and wildfire because of the multi-step processes and non-linearities that will often determine the overall outcome [19, 29].

## Conclusions

Both wildfire and diseases are part of healthy forests, but each can rise to the level where they impact management goals and natural resources, through either their individual or interactive impacts. Although we have relatively few examples of wildfire-disease interactions, the current state of the science shows that these interactions can result in a range of responses which increase or suppress disease and/or wildfire and should be expected to occur beyond the *Phytophthora* pathogen systems where they are best documented. To date, wildfire and disease processes appear to be most strongly linked and mediated by the dynamics of host communities and to a lesser degree by direct impacts to pathogen populations. Furthermore, host communities are largely synonymous with fuels when viewed through a purely fire-ecology lens. This creates natural potential for management which addresses both wildfire and disease problems. In general, the best expectation for forest pathogens—particularly invasive pathogens—is to expect the unexpected. This is even more true for wildfire-disease interactions given the accelerating pace and severity of climate change which may increase the occurrence, magnitude, and direction of interactive effects by increasing the overlap of these disturbances as well as the severity of each disturbance individually. This creates the potential for intensified interactions in those systems where they are already known to occur as well as systems

where one or the other disturbance has not been historically at the forefront of importance. Building a better understanding of these interactions, including sensible management responses, relies on integration of knowledge on disease and wildfire which is wholly achievable given the strong foundation of both fields. Such integration holds potential to better elucidate the critical knowledge frontier of when, where, and by what mechanisms these common disturbances interact in an ecological meaningful way.

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## Declarations

**Conflict of Interest** The author declares no conflict of interest.

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•• Of major importance

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