

# A framework to evaluate climate effects on forest tree diseases

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## Abstract

A conceptual framework for evaluation of climate effects on tree diseases is presented. Climate can exacerbate tree diseases by favouring pathogen biology, including reproduction and infection processes. Climatic conditions can also cause abiotic disease—direct stress or mortality when trees' physiological limits are exceeded. When stress is sublethal, weakened trees may subsequently be killed by secondary organisms. To demonstrate climate's involvement in disease, associations between climatic conditions and disease expression provide the primary evidence of atmospheric involvement because experimentation is often impractical for mature trees. This framework tests spatial and temporal relationships of climate and disease at several scales to document climate effects, if any. The presence and absence of the disease can be contrasted with climate data and models at geographic scales: stand, regional and species range. Temporal variation in weather, climate and climate change is examined during onset, development and remission of the disease. Predisposing factors such as site and stand conditions can modify the climate effects of some diseases, especially at finer spatial scales. Spatially explicit climate models that display temperature and precipitation or derivative models such as snow and drought stress provide useful data, and however, information on disease extent at different spatial scales and monitoring through time are often incomplete. The framework can be used to overcome limitations in other disease causality approaches, such as Koch's postulates, and allow for the integration of vegetation, pathogen and environmental data into causality determinations.

## 1 | INTRODUCTION

The changes in climate taking place in parts of North America and elsewhere in the world during the past century... are very likely affecting the distribution and severity of many of our tree diseases.

George Hepting (1963)

After nearly six decades, the prescience of George Hepting's, 1963 review of the relationship between climate change and forest diseases is striking. We now have a better understanding of the science surrounding climate change including the magnitude at which climate forcing and atmospheric greenhouse gas accumulation are occurring as well as the dominant role that human activity plays (IPCC, 2019). Globally, 2010–2019 was the hottest decade on record; the five hottest years ever recorded are the last five (2015–2019) (Kennedy et al., 2020). So, to aid investigations of tree diseases and mortality in a time of rapid climatic change, this paper builds on Hepting's review by providing a conceptual framework to address climate and climate change's influence on forest tree diseases.

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Several reviews describe the influence of climate change on forest diseases (Desprez-Loustau et al., 2007; Dukes et al., 2009; Kliejunas et al., 2009; La Porta et al., 2008; Sturrock et al., 2011; Woods et al., 2010;) and other studies examine individual climate-disease interactions (e.g., Brasier, 1996; Klopfenstein et al., 2009; Woods et al., 2005). What needs reassessment in conversations and the literature is a conceptual basis on how a changing climate affects forest tree diseases, a foundation and language to help guide discussion, and a method to critically evaluate possible climatic controls of disease. To demonstrate disease causality, plant pathologists utilize Koch's postulates but for diseases without a biotic cause, Koch's postulates cannot be conducted. There is a need for a means to demonstrate causality based on observation and integration of data from various sources. The objectives of this paper are to offer: (a) concepts for demonstrating causality in climate- and climate change-driven forest diseases and (b) methods for evaluating the extent and certainty of any climate involvement. To remain focused on these two objectives, this paper does not provide a guide to resolve the causes of all diseases (i.e., study of symptoms and aetiology), nor does it cover insects and animal damage to trees, atmospheric factors other than climate variables (e.g., not air pollution damage), or environmental effects of climate-induced disease. We recognize that these other factors are important but our aim is to elucidate the impacts of climate change (longer-term regime shifts in climate) and climate on forest diseases. Throughout much of this document, we first consider climate effects, and then as a second step, climate change effects.

## 2 | WEATHER, CLIMATE AND CLIMATE CHANGE

First, we clarify definitions of weather, climate and climate change (Table 1) as each may contribute to expression of forest tree disease. Weather and climate factors include primarily temperature, moisture (precipitation as rain and snow, and humidity) and circulation; all are influenced by geography (e.g., latitude, elevation, topography and proximity to mountains and water bodies). Historically, climate change has been initiated by natural processes such as variation in earth's orbit and exposure to solar and

volcanic activity, but more recently it has been primarily altered by the burning of fossil fuels, other industrial activity and deforestation (IPCC, 2019).

## 3 | TYPES OF CLIMATE-INDUCED DISEASES OF FOREST TREES

We outline an organizing principle to guide evaluation and discussion of how climate, and climate change, influence tree health and mortality. A fundamental concept of plant pathology, the plant disease triangle (Figure 1; Gäumann, 1950; Stevens, 1960), provides a useful structure for understanding the interacting elements needed for tree diseases and mortality to occur. Several papers that discuss climate-forest relationships also use the plant disease triangle, one with a focus on phenology and alder canker (Grulke, 2011) and another on ozone damage (Chappelka & Grulke, 2016). We use the disease triangle concept but modify two of its elements to focus on the roles of climate.

To hone in on climate, we deconstruct the 'Environment' factor of the disease triangle into its components: retaining climate/weather, but temporarily setting aside the other contributors such as soil and other site factors, stand composition and structure and microbial community. Note that these removed factors can be considered later to give a more complete picture of the forest disease once an understanding of the potential primary climate effects have been discerned. We use the word 'climate' but recognize that shorter-term weather events influence both disease development and tree mortality as well. However, when damaging weather events are repeated across longer time spans, they represent climate, or possibly climate regime shifts. Next, we replace the factor 'Host' with 'Forest tree' because direct climate-induced mortality (i.e., abiotic mortality) does not always require a pathogen so there is no host.

Here we consider this simple conceptual model to elucidate two main ways that climate contributes to tree stress and/or elevated pathogen activity that can lead to tree mortality. Examples in forest pathology reveal that there is more than one pathway to tree death but determining the actual cause and mechanism is not always simple (Das et al., 2016).

<i>Weather</i>	The condition of the atmosphere at a location over a short period of time, daily to seasonal periods of temperature, precipitation; includes extreme weather events.
<i>Climate</i>	Patterns of weather usually considered over a decade or longer, including cycles of decadal oscillation. Climate is usually expressed as cumulative statistics (mean, variation) of shorter-term weather data. Modelled future climate projections typically give averages, often covering several decades.
<i>Climate change</i>	Regime shifts of climate over multiple decades or longer, in some cases not predicted by the effects of various natural events or historic climate cycles. These regime shifts can be from natural or anthropomorphic causes. The speed and magnitude of environmental changes under anthropogenic-driven climate change are believed to be more pronounced, as much as 10 times the rate of warming after the last glacial period (IPCC, 2019).

**TABLE 1** Definitions of weather, climate and climate change

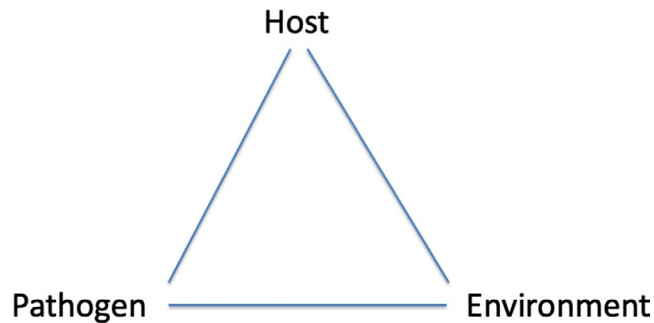


FIGURE 1 The plant disease triangle

### 3.1 | Type 1. Climate-pathogen disease

For 'climate-pathogen diseases', forest disease develops because climate directly favours pathogen biology (Figure 2). The tree may remain physiologically well suited to the climate and experience no related stress under the novel conditions.

This type of climate-induced mortality is illustrated by *Dothistroma* needle blight on lodgepole pine (*Pinus contorta* Douglas ex Loudon), in northwest British Columbia. Conidiospores of *Dothistroma septosporum* (Dorog.) Morelet are released and new infections initiated throughout the year, provided temperatures are above 5°C and moisture is present (Sinclair et al., 1987), but there are clear environmental optima at temperatures of 15–20°C (Gadgil, 1974). The incidence of *Dothistroma* needle blight infection is highly sensitive to yearly differences in weather: warm, wet summer weather repeated over several consecutive years stimulates sporulation and colonization, creating a build-up of pathogen populations (inoculum) (Peterson, 1973). In modelling projections, lodgepole pine in northern British Columbia does not appear to be stressed directly by the current or future climate and in the absence of the pathogen could benefit from warmer wetter conditions (Wang et al., 2006). The disease develops due to the pathogen's intensification on susceptible trees; the increase in disease incidence and severity and eventual tree mortality is due to climate effects that favour the pathogen.

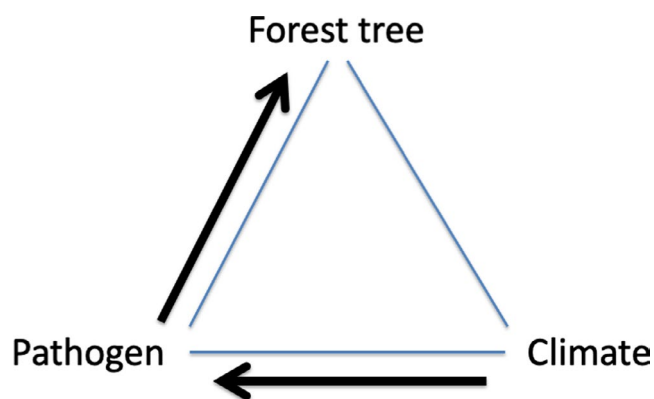


FIGURE 2 Type 1. Climate-pathogen disease. Modified plant disease triangle to show how climate can favour a pathogen to increase forest tree disease

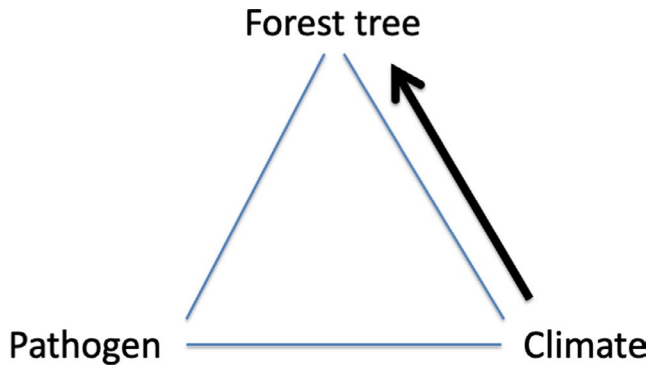
### 3.2 | Type 2. Climate-stress disease

In 'climate-stress' diseases, climate directly stresses and may kill forest trees resulting in a physiological impairment or abiotic disease that may or may not require a pathogen. Forest trees are directly damaged and killed when their physiological limits are exceeded by stressors such as drought, water inundation, excessive heat and freezing (Type 2a, Figure 3). In many cases, the climatic stress is insufficient to kill trees directly and secondary agents are required for mortality (Type 2b, Figure 4). It is possible that the climate conditions that stress the forest tree may also increase the reproduction or virulence of some pathogens making them more aggressive secondary agents. These climate-stress events can be considered climate effects when they play out over longer time periods or when shorter-term damaging weather events become a repeated pattern.

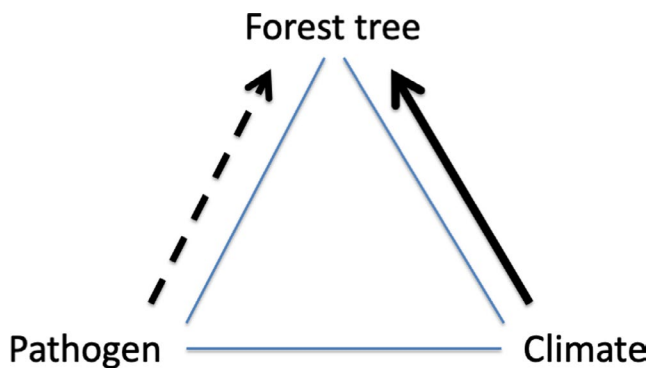
In western North America, three examples of the direct climate stress to forest trees are pole blight of western white pine (*Pinus monticola* Douglas ex D. Don) (Leaphart & Stage, 1971), yellow-cedar decline (on *Callitropsis nootkatensis* (D. Don) Oerst. ex D.P. Little) (Hennon et al., 2016) and sudden aspen decline (on *Populus tremuloides* Michx., Worrall et al., 2010). In all three of these examples, investigations began by considering pathogens as the likely cause. Further research concluded that prolonged drought was the primary cause of pole blight (Leaphart & Stage, 1971) and sudden aspen decline (Worrall et al., 2013); freezing injury to fine roots when not covered by protective snowpack is the cause of yellow-cedar decline (Hennon et al., 2012).

Secondary agents are generally incapable of killing healthy trees, but their attack and development are favoured in trees stressed and weakened by an altered climate. These secondary agents have relatively unimportant roles for pole blight on white pine and yellow-cedar decline (Type 2a, Figure 3) but more essential roles for sudden aspen decline (Type 2b, Figure 4). Each of these diseases could be considered a variant of a classic forest decline (Manion & Lachance, 1992; Sinclair & Hudler, 1988) where the repeated challenges to tree health occur over time and are primarily due to environmental conditions. Many secondary organisms are ubiquitous in the forest environment and will be found on dying or recently dead trees. Some secondary pathogens also have an endophytic stage that is typically benign but can be triggered into a pathogenic state by host stress. A continuing challenge will be to document the relative importance of secondary organisms in tree stress diseases, that is, distinguishing between the Type 2a and Type 2b diseases.

Once the primary causes of death have been determined, the additional environmental factors (i.e., site factors, stand structure) that were initially set aside from the plant disease triangle can be examined along with the primary role of climate. These would be deemed predisposing factors in forest decline terminology. Looking at our examples: pole blight occurs on soils that have little ability to retain moisture and in young dense white pine stands in which root systems of dominant, affected trees had insufficient ability to extract sufficient moisture from the soil (Leaphart & Stage, 1971);



**FIGURE 3** Type 2a. Climate-stress disease. Modified plant disease triangle to show how climate can directly stress or kill a forest tree with minimal effect of secondary contributing agents



**FIGURE 4** Type 2b. Climate-stress disease with one or more secondary agents. Modified plant disease triangle to show how climate can directly stress a forest tree but secondary pathogens (or insects) are required to cause tree

and yellow-cedar decline occurs within a certain climate envelope where there is a lack of snow cover and frequent late winter freezes, but trees are only injured and killed during cold events when predisposed by shallow rooting on either water-saturated bog or thin soils (Hennon et al., 2012).

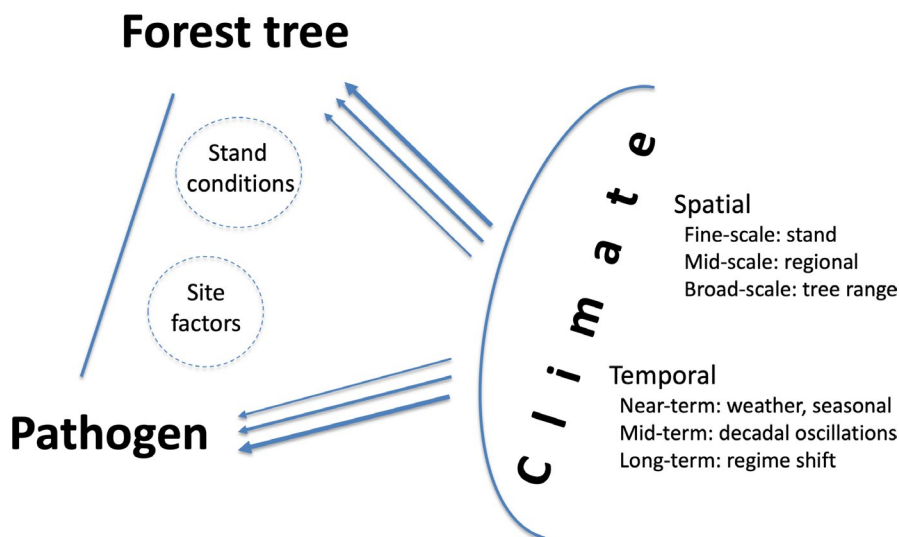
The possible predisposing factors, site and stand conditions, have less effect in sudden aspen decline but tree characteristics generally play a more important role in tree death (Worrall et al., 2008). One of the first well-documented examples of climate change impacts on forest disease is that of *Phytophthora cinnamomi* Rands in oak forests of southern Europe; Brasier (1996) noted increased rates of infection and tree decline following physiological stress caused by drought.

#### 4 | DOCUMENTING ROLES OF CLIMATE IN FOREST DISEASES

Plant pathologists have long relied on Koch's postulates to formally demonstrate the causative role of pathogens in diseases (Agrios, 2005). This is a linear approach of consecutive steps: consistent observation of a pathogen with the disease (symptoms, and related disruption in physiological function), isolation of the pathogen to pure culture, replication of the disease with inoculation and then recovery of the pathogen from the inoculated plant.

Our understanding of disease processes has progressed since Koch's postulates were introduced in the 1880s, with advances in genetics, molecular biology, spatial ecology, climate modelling and statistics to list just a few disciplines. Applicability of Koch's postulates is limited for numerous plant diseases because they may be caused by unculturable, obligate parasites such as viruses (Fox, 2020) or rusts (McDowell, 2011), or by the interaction of multiple microbes such as for acute oak decline (Denman et al., 2018). Also, Koch's postulates cannot be used to demonstrate causality for diseases without a biotic cause, such as those driven directly by weather or climate. There is a need for a means to demonstrate aetiology and causality that goes beyond the traditional framework of Koch's postulates—to allow observation and integration of data from various sources to document causality.

Here we outline a framework to evaluate and document climate drivers of forest diseases by considering a number of factors simultaneously and at different scales rather than as a series of sequential



**FIGURE 5** A framework diagram that modifies the plant disease triangle and expands climate and other environmental components to consider climate drivers on biotic and abiotic forest tree diseases

**TABLE 2** Criteria to assess causality of climate and related factors in controlling forest tree disease (adopted from Fox, 2020)

Criterion	Possible climate causality in forest tree diseases
Experiment	The disease can be replicated with experimentation under climate controls and can be prevented by alleviating the climate stress on trees or climate influence on the pathogen. Often experimentation with climate controls on mature forest trees may not be feasible but exposing seedlings, leaves or stems to climate conditions may indicate abiotic injury. If pathogens are involved, inoculations under specific conditions may be useful to indicate their ability to cause harm.
Check for other explanations	Alternative hypotheses with factors other than climate are carefully considered and eliminated as possible primary causes of disease outbreak.
Consistency	The pattern of disease outbreak, intensification and remission corresponds with spatial models of climate and temporally with weather data. A lack of disease is observed when or where climate is not favourable for disease.
Coherence and plausibility	The natural history and biology of the disease is consistent with a climate-pathogen or climate-stress disease hypothesis. Knowledge from physiology or silvics suggests that the tree species affected is more vulnerable than associated species to the climate stress, or that the pathogen's infection biology is favoured by specific climate conditions.

steps. Published criteria developed to assess causality from other disease disciplines are used to consider the validity of climate hypotheses for disease causation. These approaches are then used to express a sense of certainty of the climate effects; certainty may vary widely between new forest disease outbreaks with little information and diseases that have been relatively well studied.

## 5 | A FRAMEWORK TO EVALUATE CLIMATE AND RELATED ENVIRONMENTAL FACTORS IN FOREST TREE DISEASES

A framework is offered to evaluate the possible roles of climate, climate change and related environmental factors as contributors to forest tree diseases (Figure 5). Collectively, these factors can be used to demonstrate the roles of climate in forest disease, if any. To use this approach, we first construct a hypothesis to state a possible linkage of climate (either occasional extremes or climate change) to the abiotic or biotic disease under study. The more factors that are shown to strongly correspond to the hypothesis of climate involvement, the higher the level of confidence in the general relationship between climate and disease. Certainty may be increased by experimental evidence that supports the climate hypothesis. An assertion that 'climate change' has initiated a disease should be made after climate and weather relationships have first been established.

The framework factors offer a basis to evaluate hypotheses regarding climate's involvement in a forest disease. To use this approach, consider to what degree each of these factors corresponds with the onset and development of a particular forest tree disease which may have a climatic cause. A foundational principle for this approach is to provide context by checking historical records of climate and outbreaks, and: (a) making observations and measurements in diseased and non-diseased (unhealthy/healthy) forests to understand the contribution of each of these factors to disease (spatial evaluation); and

(b) evaluating weather and climate data when diseases are in outbreak and when they subside to determine if there are any relationships between the fluctuation of atmospheric conditions and disease expression (temporal evaluation). We acknowledge that spatial and temporal scales are actually continuous gradients, but we divide these gradients into three classes of scale to aid communication.

## 6 | CRITERIA TO EVALUATE CLIMATE CAUSATION FOR FOREST TREE DISEASES

Sir A. Bradford Hill (1965) developed criteria to assess causation in human diseases related to exposure to environmental hazards and named these criteria 'rules of evidence'. These criteria were simplified and adapted by Fox (2020) to investigate the causal role of viruses in plant diseases. We adapt Fox's criteria to evaluate causality for forest tree diseases (Table 2).

To determine whether climate is the cause for a particular tree disease evaluate the framework criteria (i.e., check the hypothesis of climatic causation for other explanations, consistency, coherence and plausibility) for spatial, temporal, tree species and pathogen factors. Approaches, examples, and considerations are described below.

## 7 | CLIMATE-SPATIAL CONSIDERATIONS

### 7.1 | Does the disease align spatially with geographic climate patterns that are consistent with the hypothesis of a climate-induced disease?

The relationship between climate and disease can be examined at several spatial scales from the stand level (or within stands) to the regional or broader to the natural range of the pathogen and/or tree species. The climate-tree relationship could also be evaluated in

circumstances where trees are planted beyond their natural range (e.g., assisted migration).

### 7.1.1 | Fine scale (local, stand)

Investigations at the fine spatial scale (within or at the stand level) can reveal which individual trees and tree species are affected and unaffected on vegetation plots, and how other features such as site factors and forest stand conditions may be controlling disease expression. Soil conditions can exacerbate or ameliorate climate stress to forest trees; for example, tree mortality is expressed in low moisture holding soils in drought diseases and root freezing occurs where soils force shallow rooting.

Likewise, stand-structural elements such as tree density and canopy cover may affect microclimate to predispose trees to biotic and abiotic diseases. Less precipitation falls to the ground in dense stands with greater canopy cover, which may also have greater evapotranspiration and therefore drier soils. Greater competition for water among trees in dense stands may lead to moisture deficiency and drought stress. Tree density and canopy cover also affect ambient temperature fluctuation, with lower daily minimums and higher daily maximums in less dense, stands. These factors can alter tree physiology and phenology and predispose trees and regeneration to direct heat and freezing injury (Vitasse et al., 2014).

For biotic diseases, stand conditions that alter temperature, humidity, wetting and drying are important microclimate regulators for many foliar, shoot and stem diseases, including rusts (Van Arsdel et al., 2006; Chappelka & Grulke, 2016). Stand conditions that promote and maintain the abundance of understory plant species may be important for some rust diseases due to their effect on alternate host species (Zambino, 2010). Local topography is often also important for rust diseases because of the way that rust spores can be carried downslope on diurnal airflow, or in convective air flow near bodies of water (Van Arsdel et al., 2006).

### 7.1.2 | Mid-scale (watershed to regional)

Landscape features such as elevation and aspect can become relevant at this mid-scale because they directly affect weather and climate, which in turn modify tree phenology and pathogen reproductive biology. Widely distributed plots, such as U.S. Forest Service Forest Inventory and Analysis (Bechtold & Patterson, 2005), can be used to establish how patterns of disease expression vary by mid-scale factors. However, some vegetation plot systems do not include data on specific tree diseases or their pathogens, or despite collection, may be considered unreliable. If tree symptoms can be observed from the air, forest health aerial surveys may be useful to determine spatial patterns of disease outbreak. Historical climate data and climate projections, linked to GIS layers through models, may provide spatial extrapolations of climate to variation in disease

activity. Variables important to a climate–disease hypothesis, such as moisture stress and snow patterns, can be created in these spatially explicit climate models.

### 7.1.3 | Broad scale (range-wide)

Weather and climate can be controlled by landscape factors including proximity to oceans (i.e., continentality), other water bodies, and mountains (e.g., for effects on deposition of precipitation vs. 'rain-shadow'; interaction of slope, insolation and elevation on water stress and frost exposure). Variables capturing these factors are often built into GIS-based climate models. It may be challenging to develop pathogen or disease data at the spatial scale of tree ranges, which often extend beyond country boundaries. For biotic diseases, the distribution of a pathogen may not align with the entire natural range of the host tree species, sometimes presumably due to differing climate envelopes, but also from long-term natural history factors such as post-glacial migration (Barrett et al., 2012) or fire extirpating and delayed reestablishment of pathogens with dispersal slower than that of host trees, as in the case of some dwarf mistletoes (Hawksworth & Wiens, 1996).

## 8 | CLIMATE-TEMPORAL CONSIDERATIONS

### 8.1 | Does the disease occurrence align over time with historic and future climate patterns in a manner that is consistent with a climate–disease hypothesis?

The timing of disease onset, progression, outbreak events and remission should be compared to weather and climate data to establish possible atmospheric drivers. Weather station data, climate summaries and climate projection models are generally available, but reliable information on the variation in disease occurrence through time is critical.

#### 8.1.1 | Near-term scale (weather, seasonal)

Where recent weather, such as one or more extreme weather events, are suspected as the direct cause of physiological damage to trees or trigger of pathogen activity, weather station or locally monitored weather data should be assessed. Episodes of weather presumed favourable for disease can be compared to the timing of disease expression. Repeated measurements on monitoring plots or annual forest health surveys are needed to document short-term changes in disease. It is important to recognize there will usually be a time lag between direct climate stress or pathogen infection and the development of tree symptoms or mortality; thus, complicating the linking of weather or short-term climate events to disease occurrence.

### 8.1.2 | Mid-term scale (decadal oscillation)

Basic historical daily data from weather stations may be available from over the past century (Jones et al., 1999; Sun et al., 2018), while data sets with numerous variables (e.g., snow) may be available at many locations for the past few decades. Data can be mined as daily values, consecutive days (important for some diseases to identify infection periods for spores), or averaged over months, seasons or years for associations with disease activity. Awareness of decadal oscillations in climate can be integrated at this mid-temporal scale. Forest health aerial survey maps can be used to document active periods of some diseases for the last 50–60 years in many regions of North America. On-the-ground observations or vegetation plot data of disease activity is probably only available for some diseases as many long-term plot systems record tree mortality but not specific diseases or cause of death. Dendrochronology may be helpful for documenting past outbreaks of some diseases (Lee et al., 2013; Welsh et al., 2014).

### 8.1.3 | Long-term scale (regime shift)

If long-term records of climate and disease are available and disease incidence and severity have changed, the potential that climate change has affected disease needs to be considered. Anecdotal reports of diseases may provide points in time to pair with climatic events where long-term weather station are available but not consistent disease monitoring. Stand reconstruction, including snag and log dating, and dendrological techniques can be used to link disease activity with climate data. Plots could be established in research forests or other locations where plot integrity would not be altered, and researchers could take measurements over long periods of time. Pollen analysis and other paleo-ecological tools can provide information on tree species occurrence, population fluctuations and range expansion/retraction over longer time periods, but generally there will be no corresponding disease information. Climate projections a century into the future might be used to estimate habitat suitability for trees as well as some disease vulnerability to about the year 2100. Because climate predictions are based on 30-year averages, their greatest potential for success would be for diseases that relate to long-term interactions of forest tree species, climate and pathogens (if any).

## 9 | FACTORS RELATED TO FOREST TREES

### 9.1 | Is the tree species known to be vulnerable to the stress stated in the climate-disease hypothesis?

For any hypothesis of abiotic disease with direct impact from climate, the relative vulnerability of the affected tree species to climate stress and injury compared to other tree species in the same vicinity should be considered. Knowledge of this vulnerability may

come from anecdotal information on the tree species autecology or silvics, or from direct experimentation by ecophysiologicals or others on drought, inundation, heat and freeze tolerance limits.

Tree age and genetics may be important in the expression of biotic and abiotic diseases where climate involvement is suspected. For example, data from provenance tests have been used to identify populations within species that are similar in climatic adaptation (i.e., climatypes) for growth (Rehfeldt et al., 2014), survival (Warwell & Shaw, 2017) and disease resistance (Rehfeldt, 1995). Climate change risk assessments are available that explicitly consider the extent of within species differentiation for climatic adaptation (Aubry et al., 2011; Rehfeldt et al., 2015). In general, species that exhibit climatypes with relatively narrow climatic ranges (i.e., 'specialist') are expected to be widely disrupted by ongoing climate change. In contrast, species with climatypes having broad climatic ranges (i.e., 'generalist') are expected to exhibit maladaptation predominately at the trailing edge of their distributions (e.g., lower elevation) in response to climate change.

Climatic conditions may influence tree phenology to exacerbate abiotic disease by predisposing trees to direct climatic injury, while other conditions could cause a reduction in the same disease. Likewise, climate effects on tree and pathogen phenology may enhance or disrupt biotic diseases by altering the concurrence of events necessary for infection, such as sporulation and spore dispersal by the pathogen and bud burst (Grulke, 2011) or stomatal maturation and opening on needles or leaves and increasing leaf resistance to infection related to stress (Zambino, 2010).

## 10 | FACTORS RELATED TO PATHOGENS

### 10.1 | Is the pathogen known to be favoured by weather and climate conditions consistent with the climate-disease hypothesis?

Weather and climate often have profound effects on a tree pathogen's sporulation (or fruit and seed production for dwarf mistletoe), dispersal, infection and growth in the host tree. Very short-term weather factors such as humidity and leaf wetness can determine the success of a pathogen's reproduction and infection for many foliar diseases. Changes in disease expression for pathogens that cause chronic infections such as root diseases (Klopfenstein et al., 2009), stem decays and dwarf mistletoes (Barrett et al., 2012) may show a relationship with climate on the order of decades or longer.

## 11 | CONSIDERING CERTAINTY FOR CLIMATE INVOLVEMENT IN FOREST DISEASE DEVELOPMENT

Climate change will continue to cause elevated levels of stress and mortality to forest trees and contribute to increases in pathogen populations and their impacts. As new tree mortality patterns are

encountered, each situation needs to be evaluated for cause by forest pathologists and other specialists. Some emerging diseases may be elucidated rather quickly (e.g., introduction of an aggressive invasive pathogen or insect–pathogen complex) and climate analysis may show little or no role in causality. Other outbreaks may show preliminary associations of climate and disease that warrant further study. In those cases, studies and field reports may show consistent fluctuations of disease expression that correspond with climatic conditions spatially and temporally, and there may be experimental evidence demonstrating the mechanism of tree stress or favouring of pathogen biology, so climate's involvement may be generally accepted.

Once a climate hypothesis is formed, a level of certainty of the climate–disease relationship can be described along a gradient from none to confident, based on the documented strength of the associations of climate and related factors. The climate control of disease could be considered *tentative* if there is an association through time and at locations between a proposed climate effect and at least one disease outbreak and remission. There are typically other, alternative plausible explanations for disease increase to consider. More thorough observation and research is needed to establish a firm cause and effect.

Involvement of climate would be considered as *probable* if repeated outbreaks and remission of the disease consistently follow the proposed spatial and temporal patterns of the climate hypothesis. These outbreaks would have been evaluated at several spatial and temporal scales; each indicating an association with climate. The disease activity would be expected to subside when weather or climate returns to favourable conditions for the tree or unfavourable for the pathogen. The relationship of weather or climate stressing the tree or favouring pathogen development would be substantiated in scientific reports. Under the particular climatic conditions, site factors or stand conditions may appear to be predisposing trees to the disease, and the disease would have little to no impact in areas where these local factors do not exacerbate tree stress.

Climate as a primary driver in disease could be considered *confirmed* when most or all of the factors listed above have been evaluated at spatial and temporal scales and they consistently support a particular climate effect stated in the climate–disease hypothesis. Predisposing factors such as site factors and stand conditions may not apply to all climate-induced diseases. Multiple lines of evidence would have been published in peer-reviewed scientific literature. There is no credible alternative hypothesis to plausibly explain the pattern of onset, outbreaks and remissions of the disease other than that of climate effects. Also, the mechanism by which the physiologic injury to trees occurs or the pathogen population increases would have been established experimentally, or observationally, and documented in scientific literature.

## 12 | THE FURTHER CASE OF CLIMATE CHANGE EFFECTS ON FOREST DISEASES

The above factors may be used to establish links between fluctuations in climate and forest diseases. It is a second step to make the

assertion that *climate change* is exacerbating the disease. This can only be approached after climatic factors have been solidly linked to a disease. To establish a claim that the climatic conditions attributed to an outbreak are present due to climate change, additional analysis is required to make the case that a climate regime—one beyond perceived normal decadal oscillations—has emerged or is emerging and is responsible for the disease outbreak. This is justified when a regime favourable to disease is consistent with long-term climate cycles or projections that fit with climate change. Climate change is an alteration of climatic factors that may naturally fluctuate but the magnitude and directionality of such fluctuations indicate departure from past patterns. These fluctuations are being accelerated by human alterations to greenhouse gases, and the observed effects on climatic parameters are accounted for in general circulation models that project future climate (IPCC, 2019).

## 13 | CONCLUSIONS

There are two primary types of climate-induced forest tree disease, one where climate favours pathogens to increase disease, and another where climate directly stresses trees. A framework is offered which allows scientists to consider important factors in disease expression that may be influenced by climate. Spatial and temporal relationships of atmospheric conditions and forest disease, examined at different scales, are the main basis for evaluating associations of climate. Criteria of causation can be applied to each framework factor.

Understanding the complexities of how forest tree diseases may be affected by climate factors requires diverse expertise and collaboration. Forest pathologists may need to seek out ecophysiologists to assess temperature and moisture stresses that trigger abiotic stress diseases, vegetation and landscape ecologists to interpret fine and broad scale patterns of disease and climate, entomologists when insects are part of the cause of tree death, mycologists to determine physiology of forest pathogens under different environmental conditions, dendrochronologists to evaluate climate and disease effects in tree rings, and climatologists to use the most appropriate sources of weather data and the appropriate climate projections.

Since George Hepting (1963) proposed the importance of climate change in forest diseases, access to climate data and advances in climate modelling, spatial analysis and computing power are providing rich sources of information to be used in testing relationships of disease with climate. Aerial survey and remotely sensed vegetation data, coupled with reduced costs and improved diagnostic methods, for example, metagenomics, in situ diagnostics, will strengthen data for application to disease cause analysis. However, spatial and temporal data on specific forest tree diseases may continue to be difficult to obtain.

Further use of this framework by forest pathologists will improve our ability to communicate with ecologists, land managers, policy-makers, and others concerned with plant health and also aid in recognition of the importance of biotic agents, host genetics, climate



and other environmental factors in the expression of forest disease. As climate change accelerates, methodical consideration of climate and climate change's role in forest disease development is essential to fully understand the cause of tree diseases and mortality. By using this framework, the involvement and certainty of climate drivers may be better assessed.

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## PEER REVIEW

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## REFERENCES

- Agrios, G. N. (2005). *Plant pathology* (5th ed.). Elsevier-Academic Press.
- Aubry, C., Devine, W., Shoal, R., Bower, A., Miller, J., & Maggiulli, N. (2011). *Climate change and forest biodiversity: A vulnerability assessment and action plan for National Forests in Western Washington* (p. 130). USDA For. Serv., Pacific Northwest Research Station.
- Barrett, T. M., Latta, G., Hennon, P. E., Eskelson, B. N. I., & Temesgen, H. (2012). Host-parasite distributions under changing climate: *Tsuga heterophylla* and *Arceuthobium tsugense* in Alaska. *Canadian Journal of Forest Research*, 42, 642–656.
- Bechtold, W. A., & Patterson, P. L. (2005). *The enhanced forest inventory and analysis program-national sampling design and estimation procedures*. Gen. Tech. Rep. SRS-80. US Department of Agriculture, Forest Service, Southern Research Station.
- Brasier, C. M. (1996). *Phytophthora cinnamomi* and oak decline in southern Europe: 25 environmental constraints including climate change. *Annales des Sciences Forestiere*, 26(53), 347–358.
- Chappelka, A. H., & Grulke, N. E. (2016). Disruption of the 'disease triangle' by chemical and physical environmental change. *Plant Biology*, 18(S1), 512.
- Das, A. J., Stephenson, N. L., & Davis, K. P. (2016). Why do trees die? Characterizing the drivers of background tree mortality. *Ecology*, 97(10), 2616–2627.
- Denman, S., Doonan, J., Ransom-Jones, E., Broberg, M., Plummer, S., Kirk, S., Scarlett, K., Griffiths, A. R., Kaczmarek, M., Forster, J., & Peace, A. (2018). Microbiome and infectivity studies reveal complex polyspecies tree disease in Acute Oak Decline. *The ISME Journal*, 12, 386–399.
- Desprez-Loustau, M. L., Robin, C., Reynaud, G., Déqué, M., Badeau, V., Piou, D., Husson, C., & Marçais, B. (2007). Simulating the effects of a climate-change scenario on the geographical range and activity of forest-pathogenic fungi. *Canadian Journal of Plant Pathology*, 29(2), 101–120.
- Dukes, J. S., Pontius, J., Orwig, D., Garnas, J. R., Rodgers, V. L., Brazee, N., Cooke, B., Theoharides, K. A., Stange, E. E., Harrington, R., & Ehrenfeld, J. (2009). Responses of insect pests, pathogens, and invasive plant species to climate change in the forests of northeastern North America: What can we predict? *Canadian Journal of Forest Research*, 39(2), 231–248.
- Fox, A. (2020). Reconsidering causal association in plant virology. *Plant Pathology*, 69(6), 956–961. <https://doi.org/10.1111/ppa.13199>
- Gadgil, P. D. (1974). Effect of temperature and leaf wetness period on infection of *Pinus radiata* by *Dothistroma pini*. *New Zealand Journal of Forest Science*, 4, 495–501.
- Gäumann, E. A. (1950). *Principles of plant infection: A text-book of general plant pathology for biologists, agriculturists, foresters and plant breeders*. Hafner Publishing.
- Grulke, N. E. (2011). The nexus of host and pathogen phenology; understanding the disease triangle with climate change. *New Phytologist*, 189(1), 8–10. <https://doi.org/10.1111/j.1469-8137.2010.03568.x>
- Hawksworth, F. G., & Wiens, D. (1996). *Dwarf mistletoes: Biology, pathology and systematics*. *Agricultural Handbook 709* (410 p.). US Dept. of Agriculture Forest Service.
- Hennon, P. E., D'Amore, D. V., Schaberg, P. G., Witter, D. T., & Shanley, C. S. (2012). Shifting climate, altered niche, and a dynamic conservation strategy for yellow-cedar in the North Pacific coastal rainforest. *BioScience*, 62(2), 147–158. <https://doi.org/10.1525/bio.2012.62.2.8>
- Hennon, P. E., McKenzie, C. M., D'Amore, D. V., Wittwer, D. T., Mulvey, R. L., Lamb, M. S., Biles, F. E., & Cronn, R. C. (2016). *A climate adaptation strategy for conservation and management of yellow-cedar in Alaska* (382 p). PNW-GTR-917. U.S. Dep. Agric., For. Serv., Pacific Northwest Research Station.
- Hepting, G. H. (1963). Climate and forest diseases. *Annual Review of Phytopathology*, 1, 31–50. <https://doi.org/10.1146/annurev.py.01.090163.000335>
- Hill, A. B. (1965). The environment and disease: Association or causation? *Proceedings of the Royal Society of Medicine*, 58, 295–300. <https://doi.org/10.1177/003591576505800503>
- IPCC (2019). Summary for policymakers. In P. R. Shukla, J. Skea, E. Calvo Buendia, V. Masson-Delmotte, H.-O. Pörtner, D. C. Roberts, P. Zhai, R. Slade, S. Connors, R. van Diemen, M. Ferrat, E. Haughey, S. Luz, S. Neogi, M. Pathak, J. Petzold, J. Portugal Pereira, P. Vyas, E. Huntley, ... J. Malley (eds.) *Climate Change and Land: An IPCC special report on climate change, desertification, land degradation, sustainable land management, food security, and greenhouse gas fluxes in terrestrial ecosystems*, in press. [https://www.ipcc.ch/site/assets/uploads/sites/4/2020/02/SPM\\_Updated-Jan20.pdf](https://www.ipcc.ch/site/assets/uploads/sites/4/2020/02/SPM_Updated-Jan20.pdf)
- Jones, P. D., New, M., Parker, D. E., Martin, S., & Rigor, I. G. (1999). Surface air temperature and its changes over the past 150 years. *Reviews of Geophysics*, 37(2), 173–199. <https://doi.org/10.1029/1999RG900002>
- Kennedy, J. J., Kendon, M., Killick, R. E., Dunn, R. J., Allan, R. J., Rayner, N. A., & McCarthy, M. (2020). Global and regional climate in 2019. *Weather*, 75(9), 264–271. <https://doi.org/10.1002/wea.3822>
- Kliejunas, J. T., Geils, B. W., Glaeser, J. M., Goheen, E. M., Hennon, P., Kim, M.-S., Kope, H., Stone, J., Sturrock, R., & Frankel, S. J. (2009). *Review of literature on climate change and forest diseases of western North America*. General Technical Report, PSW-GTR-225. US Department of Agriculture, Forest Service, Pacific Southwest Research Station.
- Klopfenstein, N. B., Kim, M.-S., Hanna, J. W., Richardson, B. A., & Lundquist, J. E. (2009). *Approaches to predicting potential impacts of climate change on forest disease: An example with Armillaria root disease* (10 p). Res. Pap. RMRS-RP-76. U.S. Department of Agriculture, Forest Service, Rocky Mountain Research Station.
- La Porta, N., Capretti, P., Thomsen, I. M., Kasanen, R., Hietala, A. M., & Von Weissenberg, K. (2008). Forest pathogens with higher damage potential due to climate change in Europe. *Canadian Journal of Plant Pathology*, 30(2), 177–195. <https://doi.org/10.1080/07060661.2008.10540534>
- Leaphart, C. D., & Stage, A. R. (1971). Climate: A factor in the origin of the pole blight disease of *Pinus monticola* Dougl. *Ecology*, 52(2), 229–239. <https://doi.org/10.2307/1934581>
- Lee, E. H., Beedlow, P. A., Waschmann, R. S., Burdick, C. A., & Shaw, D. C. (2013). Tree-ring analysis of the fungal disease Swiss needle cast in western Oregon coastal forests. *Canadian Journal of Forest Research*, 43(8), 677–690. <https://doi.org/10.1139/cjfr-2013-0062>
- Manion, P. D., & Lachance, D. (1992). *Forest decline concepts*. APS Press.

- McDowell, J. M. (2011). Genomes of obligate plant pathogens reveal adaptations for obligate parasitism. *Proceedings of the National Academy of Sciences of the United States of America*, 108(22), 8921–8922. <https://doi.org/10.1073/pnas.1105802108>
- Peterson, G. W. (1973). Infection of Austrian and ponderosa pines by *Dothistroma pini* in Eastern Nebraska. *Phytopathology*, 63, 1060–1063. <https://doi.org/10.1094/Phyto-63-1060>
- Rehfeldt, G. E. (1995). Genetic variation, climate models and the ecological genetics of *Larix occidentalis*. *Forest Ecology and Management*, 78, 21–37. [https://doi.org/10.1016/0378-1127\(95\)03602-4](https://doi.org/10.1016/0378-1127(95)03602-4)
- Rehfeldt, G. E., Leites, L. P., St Clair, J. B., Jaquish, B. C., Sáenz-Romero, C., López-Upton, J., & Joyce, D. G. (2014). Comparative genetic responses to climate in the varieties of *Pinus ponderosa* and *Pseudotsuga menziesii*: Clines in growth potential. *Forest Ecology and Management*, 324, 138–146. <https://doi.org/10.1016/j.foreco.2014.02.041>
- Rehfeldt, G. E., Worrall, J. J., Marchetti, S. B., & Crookston, N. L. (2015). Adapting forest management to climate change using bioclimate models with topographic drivers. *Forestry: An International Journal of Forest Research*, 88(5), 528–539. <https://doi.org/10.1093/forestry/cpv019>
- Sinclair, W. A., & Hudler, G. W. (1988). Tree declines: Four concepts of causality. *Journal of Arboriculture*, 14(2), 29–35.
- Sinclair, W. A., Lyon, H. H., & Johnson, W. T. (1987). *Diseases of trees and shrubs*. Cornell University Press.
- Stevens, R. B. (1960). In J. G. Horsfall, & A. E. Dimond (Eds.), *Plant pathology, an advanced treatise* (vol. 3, pp. 357–429). Academic Press.
- Sturrock, R. N., Frankel, S. J., Brown, A. V., Hennon, P. E., Kliejunas, J. T., Lewis, K. J., Worrall, J. J., & Woods, A. J. (2011). Climate change and forest diseases. *Plant Pathology*, 60, 133–149. <https://doi.org/10.1111/j.1365-3059.2010.02406.x>
- Sun, Q., Miao, C., Duan, Q., Ashouri, H., Sorooshian, S., & Hsu, K. L. (2018). A review of global precipitation data sets: Data sources, estimation, and intercomparisons. *Reviews of Geophysics*, 56(1), 79–107. <https://doi.org/10.1002/2017RG000574>
- Van Arsdell, E. P., Geils, B. W., & Zambino, P. J. (2006). Epidemiology for hazard rating of white pine blister rust. In: Guyon, J. C. comp. *Proceedings of the 53rd Western International Forest Disease Work Conference; 2005 September 26–30*. US Department of Agriculture, Forest Service, Intermountain Region, Ogden UT.
- Vitasse, Y., Lenz, A., & Körner, C. (2014). The interaction between freezing tolerance and phenology in temperate deciduous trees. *Frontiers in Plant Science*, 5, 541. <https://doi.org/10.3389/fpls.2014.00541>
- Wang, T., Hamann, A., Yanchuk, A., O'Neill, G. A., & Aitken, S. N. (2006). Use of response functions in selecting lodgepole pine populations for future climate. *Global Change Biology*, 12, 2404–2416.
- Warwell, M. V., & Shaw, R. G. (2017). Climate-related genetic variation in a threatened tree species, *Pinus albicaulis*. *American Journal of Botany*, 104, 1205–1218.
- Welsh, C., Lewis, K. J., & Woods, A. J. (2014). Regional outbreak dynamics of *Dothistroma* needle blight linked to weather patterns in British Columbia, Canada. *Canadian Journal of Forest Research*, 44(3), 212–219.
- Woods, A., Coates, K. D., & Hamann, A. (2005). Is an unprecedented *Dothistroma* needle blight epidemic related to climate change? *BioScience*, 55, 761–769.
- Woods, A. J., Heppner, D., Kope, H. H., Burleigh, J., & Maclauchlan, L. (2010). Forest health and climate change: A British Columbia perspective. *The Forestry Chronicle*, 86(4), 412–422. <https://doi.org/10.5558/tfc86412-4>
- Worrall, J. J., Egeland, L., Eager, T., Mask, R. A., Johnson, E. W., Kemp, P. A., & Shepperd, W. D. (2008). Rapid mortality of *Populus tremuloides* in southwestern Colorado, USA. *Forest Ecology and Management*, 255, 686–696. <https://doi.org/10.1016/j.foreco.2007.09.071>
- Worrall, J. J., Marchetti, S. B., Egeland, L., Mask, R. A., Eager, T., & Howell, B. (2010). Effects and etiology of sudden aspen decline in southwestern Colorado, USA. *Forest Ecology and Management*, 260, 638–648. <https://doi.org/10.1016/j.foreco.2010.05.020>
- Worrall, J. J., Rehfeldt, G. E., Hamann, A., Hogg, E. H., Marchetti, S. B., Michaelian, M., & Gray, L. K. (2013). Recent declines of *Populus tremuloides* in North America linked to climate. *Forest Ecology and Management*, 299, 35–51. <https://doi.org/10.1016/j.foreco.2012.12.033>
- Zambino, P. J. (2010). Biology and pathology of *Ribes* and their implications for management of white pine blister rust. *Forest Pathology*, 40(3–4), 264–291. <https://doi.org/10.1111/j.1439-0329.2010.00658.x>

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