Received: 24 May 2021 | Revised: 3 August 2021 | Accepted: 11 September 2021

DOI: 10.1111/efp.12719

REVIEW ARTICLE

Forest Pathology WILEY-BLACKWELL WILEY

Applications of a conceptual framework to assess climate controls of forest tree diseases

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Editor: Stephen Woodward

Abstract

A conceptual framework for climate involvement in forest tree diseases was applied to seven examples to demonstrate its suitability for different disease types: cases where climate favours pathogen biology which then leads to tree mortality or where diseases are caused primarily by climate-driven physiological injury or stress to trees. Hypotheses for climate involvement are derived from detection and monitoring data to express associations of weather or climate factors with disease development at several spatial and temporal scales. Research findings contribute to an understanding of temperature, precipitation and related climate variables that influence biotic and abiotic diseases. To demonstrate use of the framework, we accessed information from the literature which exposed data and information gaps. Among various simulated approaches to test associations of climate and disease, we found disease risk factor models that use climate inputs derived from monitoring and research provide the best understanding of climate–disease relationships. These model outputs project future disease scenarios that can be used to inform climate adaptation strategies. Conservation and management implications for current and likely future climatic conditions are provided for each disease example. The most common guidance in managed landscapes is to move the imperilled tree species to areas of lower projected climate risk and to favour non-host, climate-adapted tree species where the disease is occurring.

Paul E. Hennon, James J. Worrall and Charles G. Shaw are retired.

1 | INTRODUCTION

As Hepting (1963) predicted, climate and climate change have profoundly affected forest tree diseases with consequent impacts on ecological dynamics, species viability and provision of multiple forest resource values. Atmospheric changes and their impacts on tree diseases present complex management challenges (Seidl et al., 2017). The disruption of forest diseases (pathosystems) is considered among the earliest and most severe climate change impacts on forests (Mahony et al., 2018) and may be particularly difficult to predict in novel climates (Woods, 2011). The body of literature documenting cases of a changing climate affecting forest diseases continues to grow (Allen et al., 2015; Brasier, 1996; Delgado-Baquerizo et al., 2020; Desprez-Loustau et al., 2006, 2007; Hennon et al., 2012; La Porta et al., 2008; Sturrock et al., 2011; Woods et al., 2005; Worrall et al., 2013; Wyka et al., 2017). But forecasts on how these relationships will unfold are uncertain because of the critical role that precipitation plays in most pathosystems and expected rapid swings between opposite precipitation extremes in the future (Cai et al., 2015). Increased monitoring of biotic and abiotic disturbances in both managed and unmanaged forests is needed to help close the information gaps surrounding forest condition and threats (McDowell et al., 2015). Forest managers must incorporate adaptation to climate change in the development of management plans to ensure that forests are sustained, and species remain viable (Jandl et al., 2019). The great puzzle is to determine how best to adapt forest management, and what conditions to adapt to, given sustained climate change. A key piece is a better understanding of how forest diseases will respond to climate change and how they will alter future forest dynamics.

To address these questions, we proposed "A framework to evaluate climate effects on forest tree diseases" (Hennon et al., 2020) to provide concepts and methods for demonstrating climate as a cause of tree disease development. The framework, based on the plant disease triangle, calls for an examination of ways that weather or climate can influence disease outbreaks. Diseases are divided into two fundamental categories, Type 1, where the dominant influence is on the pathogen (climate-pathogen disease), and Type 2, where the primary climate effect occurs directly on the forest tree (climate-stress disease). Climate-stress diseases, in some cases, are largely abiotic where the stress alone can kill the tree (Type 2a). In others, secondary pathogens and/or insects take advantage of the stressed trees and kill them where the abiotic stress alone may have been insufficient to cause mortality (Type 2b).

Because experimentation with mature trees across landscapes is impractical or impossible, to demonstrate causality, the framework relies primarily on associations between weather or climate and disease, as well as the hosts and pathogens (Hennon et al., 2020). The associations are both spatial (varying weather or climate over geographic area drives disease distribution) and temporal (varying weather or climate over time drives disease frequency or severity). Spatial variation is considered at different scales: tree, stand, landscape or range wide. At fine spatial scales, individual tree, stand and site factors (e.g., tree age, density, basal area, soils, aspect, elevation) can affect climate-influenced diseases as predisposing factors. Temporal perspectives cover a range from short-term hourly and daily weather events to long-term climate and multi-decadal climate regime shifts (climate change).

Here we present applications of the framework with examples from western North America where a climate element is considered a driver of disease, thereby testing our conceptual framework with available data. We discuss similarities and differences that emerge from these disease examples including modelling for future predictions and management implications. Our objective is to facilitate use of this framework on other tree diseases where climate is suspected to be a major factor, thereby allowing for improved understanding and management of forest diseases. A longer-term goal is to improve integration of forest disease information into global dynamic vegetation models to more accurately reflect the role of forest diseases and other biotic disturbance agents under changing environmental conditions.

2 | APPLYING THE CONCEPTUAL FRAMEWORK OF CLIMATE INVOLVEMENT TO EXAMPLE FOREST TREE DISEASES

Below, the conceptual framework (Hennon et al., 2020) is applied to seven forest tree diseases that have increased during this or the previous century, and for which there is an adequate body of knowledge to enable analysis. A hypothesis is stated to explain climate involvement for each disease. The relationship between climate and each of the main factors in the conceptual framework is examined (Table 1). Climatic and other environmental conditions, including management history, are considered at several spatial scales along with temporal factors (i.e., alignment of temperature and moisture levels at locations with disease outbreaks). Then an assessment of the certainty of assumptions is made to gauge the strength of causality. Where weather or climate appear to be drivers, climate projection models are used to predict future disease activity.

2.1 | Dothistroma needle blight in British Columbia

Disease: Dothistroma needle blight (Figure 1a–d).

Forest tree: Lodgepole pine (*Pinus contorta* var. *latifolia*), shore pine (*P. contorta* var. *contorta*), and other pine species.

Type of climate effect: Type 1, climate-pathogen disease.

Pathogen: *Dothistroma septosporum* (Dorog.) Morelet.

Climate hypothesis: The pathogen, *D. septosporum*, is strongly controlled by the weather, so disease expression can change rapidly due to shifts in weather patterns. The disease spreads and intensifies quickly during periods of increased summer precipitation combined with warmer overnight minimum or optimal (i.e. warmer) daytime temperatures.

TABLE 1 Factors considered in the conceptual framework (Hennon et al., 2020) to evaluate hypotheses for climatic conditions that elicit increases or decreases in forest tree diseases

Elements to associate with climate	Approach, monitoring, analysis and research
Disease expression—spatial scales	Evaluate the climate–disease relationship at tree, stand, landscape and range-wide spatial scales
Disease expression—temporal scales	Evaluate the climate–disease relationship at weather (daily or seasonal), yearly to decadal, and multi-decadal to centuries time scales
Predisposing factors	Evaluate site and stand conditions that exacerbate or alleviate disease expression
Forest tree vulnerabilities	Evaluate climate impacts directly to forest trees, especially tree physiological stress
Pathogens	Evaluate climate-controls directly on pathogen reproduction, infection and virulence. Evaluate endophytic fungi that may change behaviour as climate changes

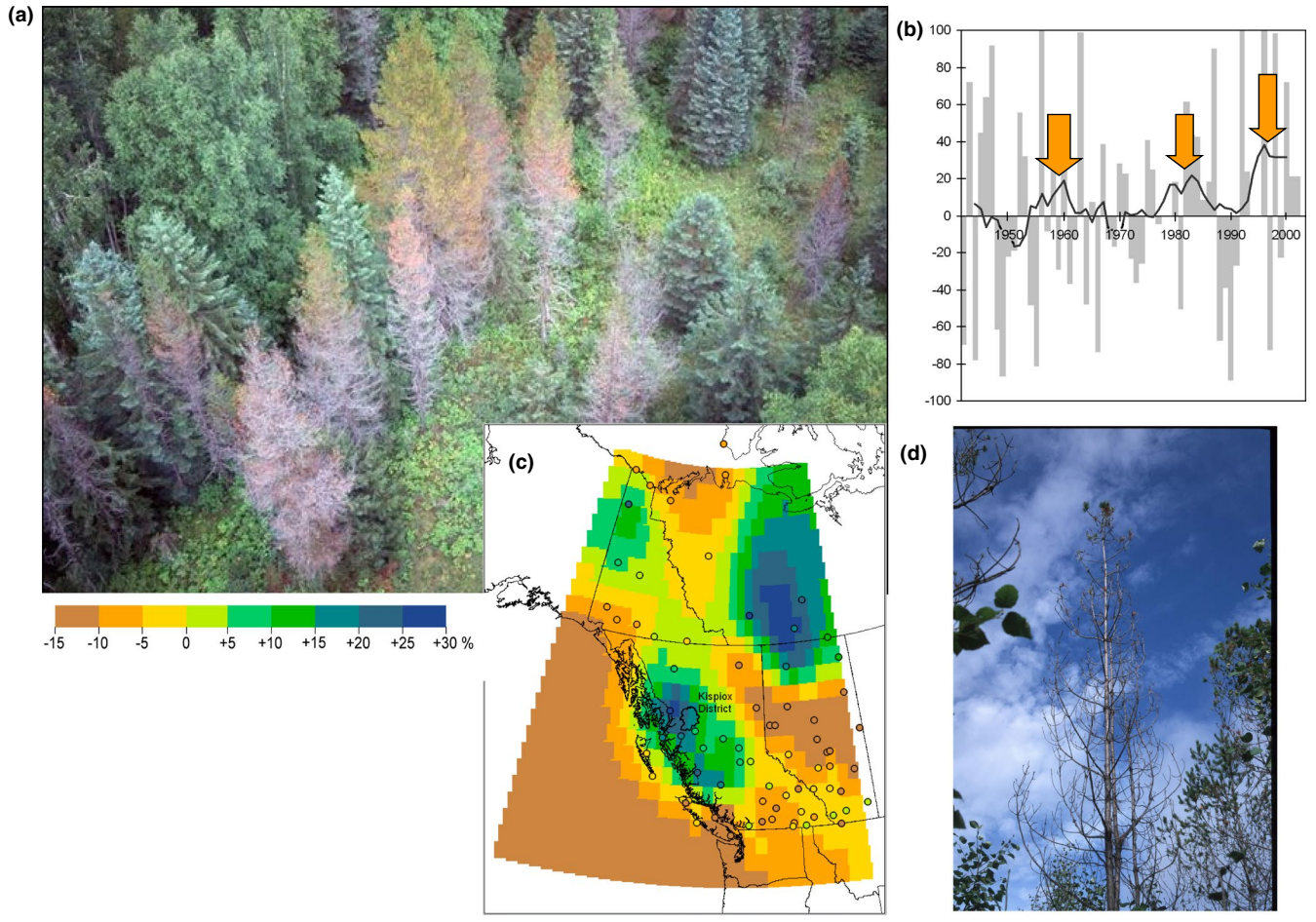

FIGURE 1 Dothistroma needle blight. (a) Photo of severe Dothistroma needle blight infestations killing mature lodgepole pine trees in the Kispiox District, British Columbia. (b) Deviation from 1961 to 1990 normal mean summer precipitation in nearby Smithers, BC (10-year moving average is added to visualize trends in noisy data) and timing of previous reports of Dothistroma outbreaks in the area based on Forest Insect and Disease Surveys (orange arrows) and Woods et al., 2005.(c) Changes in mean summer precipitation for British Columbia, Canada, displaying the percent increase in mean summer precipitation during the period of current Dothistroma epidemic initiation, 1998–2002, over the climate normal of 1961–1990. (d) Photo of young lodgepole pine severely impacted by Dothistroma needle blight

Summary: The Dothistroma epidemic in northwest British Columbia began in the late 1990s following a series of wet summers, 1997 being one of the wettest summers since the 1950s. Above-normal summer precipitation combined with warmer overnight minimum summer temperatures continued into the early 2000s, favouring this foliar pathogen in lodgepole pine stands throughout the region and beyond in a band across central British Columbia (Welsh et al., 2009). By 2001, in addition to cases of complete plantation

failure in the most heavily impacted areas, mature lodgepole pine trees were observed succumbing to the foliar disease—an unprecedented phenomenon (Woods et al., 2005).

Climate–disease spatial. At the fine scale (individual tree and stand level), *Dothistroma* first attacks the lower portion of tree crowns. Most susceptible are trees located in depressions and cool air-ponding sites, such as stream draws. Species composition of the stand appears to have little influence on infection; *Dothistroma* attacks host pine trees whether they are a prevalent or minor stand component. At the middle spatial scale (landscape level), lower elevation stands in valley bottoms are the first to be attacked, but when favourable environmental conditions persist over broad areas, this spatial pattern can break down. The influence of soil and other stand or site attributes in this host/disease relationship is minor compared to the overwhelming influence of weather conditions, but thinning treatments have been shown to reduce disease severity in several areas including New Zealand and the UK, at least temporarily (Bulman et al., 2016). At the range-wide scale, Dothistroma needle blight has severely impacted lodgepole pine plantations and natural stands in wetter interior environments throughout BC. *Dothistroma* does not yet extend to the coldest northern limits of the range of lodgepole pine in the Yukon Territory although over time, with continued warming, this is likely to change. To the south and east of BC, recent evidence suggests an expansion of the range for this pathogen (Barnes et al., 2014). Dothistroma needle blight occurs throughout the range of the shore pine in Alaska.

Climate–disease temporal. This Dothistroma epidemic example covers the temporal spectrum from daily warm-rain weather events triggering short-term outbreaks, to climatic patterns of weather over multiple years dictating disease activity in association with weather drivers such as El Niño events. An increase in the frequency and magnitude of El Niño/Southern Oscillation (ENSO) events has been forecast (Cai et al., 2015) which could influence disease expression. The strong El Niño of 1996/97 is thought to have been a critical climate turning point for the current epidemic (Woods et al., 2016). Weather events consisting of five consecutive days of rain with average high temperatures above 18°C occurred only four times from 1950 to 2002 but occurred twice in 1997 (Woods, unpublished data). This combination of precipitation and temperature is near optimal for *Dothistroma* (Gadgil, 1967; Peterson, 1967). Past outbreaks of *Dothistroma* reported in the area were associated with increased summer precipitation, but these earlier, less severe outbreaks subsided once drier conditions returned (Woods et al., 2005). Based on dendrochronological evidence, it appears periodic outbreaks of the disease have been responsible for episodic growth reductions in pines in northwest British Columbia for several hundred years (Welsh et al., 2009). These same investigations have revealed the importance of relatively warm August minimum temperatures as a determining factor in past *Dothistroma* outbreaks (Welsh et al., 2014). The increase in summer overnight minimum temperatures is one of the clearest climatic trends in northern British Columbia, increasing by 7°C at the Fort St. James weather station from 1895 to 2019 (V. Foord, pers comm., June 2, 2020).

Climate-forest tree. The host tree species, lodgepole pine, is native to the area, present for at least the past 9000 years based on pollen evidence (Gottesfeld et al., 1991). However, forest management has increased its prevalence over the past four to five decades (Woods, 2003). The impacts of *Dothistroma* on lodgepole pine growth have not been quantified in BC but in New Zealand, van der Pas (1981) found volume growth of radiata pine (*Pinus radiata*) was reduced in proportion to the percent of infected crown. At the peak of the epidemic in northwest British Columbia, over 90% of pine plantations had suffered some degree of defoliation and over 7% of the surveyed area contained trees killed by *Dothistroma* (Woods et al., 2005).

Climate-pathogen. The incidence and severity of *Dothistroma* outbreaks are strongly controlled by precipitation and temperature and are thus highly sensitive to yearly differences in weather conditions (Peterson, 1973). Each year, the first splash-dispersed conidiospores of the fungus are released in the spring from persistent dead needles infected the previous year (Bradshaw, 2004). Conidiospores continue to be released and new infections initiated throughout the year, provided temperatures are above 5°C and moisture is present (Sinclair et al., 1987). The specific environmental conditions that favour disease development are well understood (Gadgil, 1967; Peterson, 1967). Higher rates of summer precipitation combined with increased overnight minimum temperatures and warm rain events are associated with past outbreaks of Dothistroma needle blight in the US Midwest (Peterson, 1973) and Chile (Gibson, 1974).

Secondary agents are not important drivers of increased damage or mortality associated with this disease.

Certainty of climate involvement: Confirmed.

Given the consistent alignment of documented pathogen outbreaks with past favourable weather events, there are few alternative explanations for the change in disease behaviour in British Columbia. Each aspect of the disease triangle (host, pathogen, and environment) suggests a changing climate remains the best explanation for the increased disease severity in this area, making this clearly a climate-pathogen disease.

An analysis of the genetic structure of *D. septosporum* in northwest British Columbia indicates that sexual reproduction is a regular part of its life cycle, contributing to high levels of genetic diversity in the pathogen population (Dale et al., 2011). Findings from this work suggest the fungus has likely been present in the ecosystem for a considerable time. There is no evidence of a recent introduction of a new virulent strain of the pathogen that could serve as an alternate hypothesis for the cause of these outbreaks. Recent research has raised concern that tree breeding programs and material transfers of planting stock farther east in the province could increase the risk of new disease epidemics (Capron et al., 2020).

Management implications. Lodgepole pine in northwest British Columbia has shifted from being considered a favoured species, planted extensively, to a major restoration liability (Woods et al., 2005). Forests managers in BC would benefit from diversifying managed stands to mitigate previously unexpected negative effects of climate change on forest productivity. Growth forecasts need to be

adjusted to account for increased lodgepole pine volume losses due to *Dothistroma*.

Information gaps/needed research. There remains considerable uncertainty as to how climate change will affect precipitation patterns in BC and elsewhere in the range of susceptible pines. Precipitation forecasting is difficult. Extreme rain events and seasonal extremes, rather than trends, will drive disease expression. In 2018 a severe, hot, drought throughout BC led to the worst wildfire season in the history of the province; two years later, summer precipitation in northwest British Columbia was the highest in the past 70+ years. Given the importance of environmental factors in the behaviour of this pathogen, continued monitoring of forest conditions is essential.

2.2 | Swiss needle cast in the Pacific Northwest

Disease: Swiss needle cast (SNC) (Figure 2a–c).

Forest tree: Douglas-fir, *Pseudotsuga menziesii*.

Type of climate effect: Type 1, climate-pathogen disease.

Pathogen: *Nothophaeocryptopus gaeumannii* (T. Rohde) Videira, C. Nakash., U. Braun & Crous.

Climate hypothesis: In the Pacific Northwest (PNW), two climatic conditions are associated with increased Swiss needle cast (SNC) disease severity: high humidity in spring/early summer, and warm winter temperatures. Increased mean winter temperatures, evident since the 1980s, are driving disease outbreaks; warming speeds up the colonization within the needle by the fungus which appears to increase earlier formation of the stomatal blocking-fruiting bodies on Douglas-fir needles, and stomatal blockage causes disease.

Summary: Swiss needle cast was first detected on Douglas-fir along the northern Oregon Coast in the 1980s and reached epidemic proportions by the 1990s (Shaw et al., 2011). Leaf wetness during the spring/early summer sporulation period and mild winter temperatures have been identified as the two climatic factors positively correlated with disease development, with the latter likely the key in coastal Oregon and Washington (Manter et al., 2005). Since the epidemic was first identified in the mid-1980s, the mean minimum daily winter temperature in the last decade has been consistently greater than the 1961–1990 normal (~+0.8°C, ClimateNA, Wang et al., 2016). *Nothophaeocryptopus gaeumannii*, the causal agent of SNC, incites disease through stomatal plugging of infected needles by fungal pseudothecia (Manter et al., 2000). This leads to carbon starvation and subsequent needle casting in the tree host, resulting in sparse crowns and reduced growth. Due to the centrality of Douglas-fir to wood production in the region, the economic impact of SNC is substantial.

Climate–disease spatial. At the fine scale within the epidemic area, SNC is most severe in the upper crown and least severe in the lower crown of individual trees (Hansen et al., 2000; Ritóková et al., 2020). This is due to leaf wetness levels sufficient for leaf colonization in the upper crown and warmer ambient temperatures due to higher sunlight exposure. Susceptibility of Douglas-fir foliage to fungal colonization is not affected by stand species composition or density, but tolerance to *N. gaeumannii* varies across individual trees (Wilhelmi et al., 2017). Stand age is influential, as trees in older stands (120–400 years) experience consistently lower disease severity than nearby younger (20–30 years) plantation trees (Lan et al., 2019).

At the mid-scale, SNC disease severity varies across landscapes; plantations within a few miles of each other often have quite different disease severity levels. Distance from the coast is a significant factor for the Oregon epidemic, but disease severity distribution is complicated, likely due to topographic effects on weather patterns (Ritóková et al., 2020).

At the broad, geographic scale, SNC in western Oregon develops primarily within about 35 km of the Pacific Coast, with greater disease severity in the north and west. SNC is also present in southwest coastal Washington, and the disease has recently intensified in southwest coastal British Columbia (Montwé et al., 2021; SNCC, 2018, 2019). Despite a large increase in disease incidence over the last 25 years, the geographic bounds of the epidemic within Oregon have remained relatively unchanged (Shaw et al., 2021), suggesting that climatic conditions are a limiting factor (Rosso & Hansen, 2003).

Climate–disease temporal. Mildrexler et al. (2019) showed that short-term climate trends were correlated with an increase in disease expression in Oregon from 2004 to 2014; the decade was consistently cool and wet in the Pacific coastal strip from May to July. Looking to the future, average annual precipitation is not predicted to change significantly in this area (USGCRP, 2017), so SNC will likely persist even if inland sites become drier. Tree-ring studies have been used to predict and understand relationships between disease expression and climatic conditions over time. Black et al. (2010) focused on trees near Tillamook, Oregon, where SNC impacts mature forests. The study found that Douglas-fir growth reductions began in the mid-1980s and intensified in the 1990s through to the present. Decreased tree growth was correlated with increased mean temperature from March to August. Lee et al. (2017) contend that anticipated climate change in Oregon will sustain SNC development throughout the region, and disease may increase at higher elevations due to the increases in temperature as long as sufficient needle wetness is maintained during the sporulation period.

Climate-forest tree. Genetic tolerance of Douglas-fir to *N. gaeumannii* is linked to the climatic conditions at the seed source; trees grown from seed sources from higher elevations and drier regions are more susceptible to SNC when grown in common garden studies (Wilhelmi et al., 2017). Douglas-fir is well adapted to the region along the western slope of the Oregon Coast Range within the *Tsuga heterophylla* (western hemlock) vegetation series (Franklin & Dyrness, 1973). However, the near-coastal environment is in the *Picea sitchensis* (Sitka spruce) vegetation series where the current SNC epidemic is believed to have initiated (Hansen et al., 2000) and where Douglas-fir was historically a minor component. Conversion of coastal stands of non-susceptible species (e.g., *T. heterophylla*, *P. sitchensis*, *Alnus rubra* (red alder)) to Douglas-fir and large-scale reforestation of the ~150,000 ha, mid-20th century Tillamook Burn

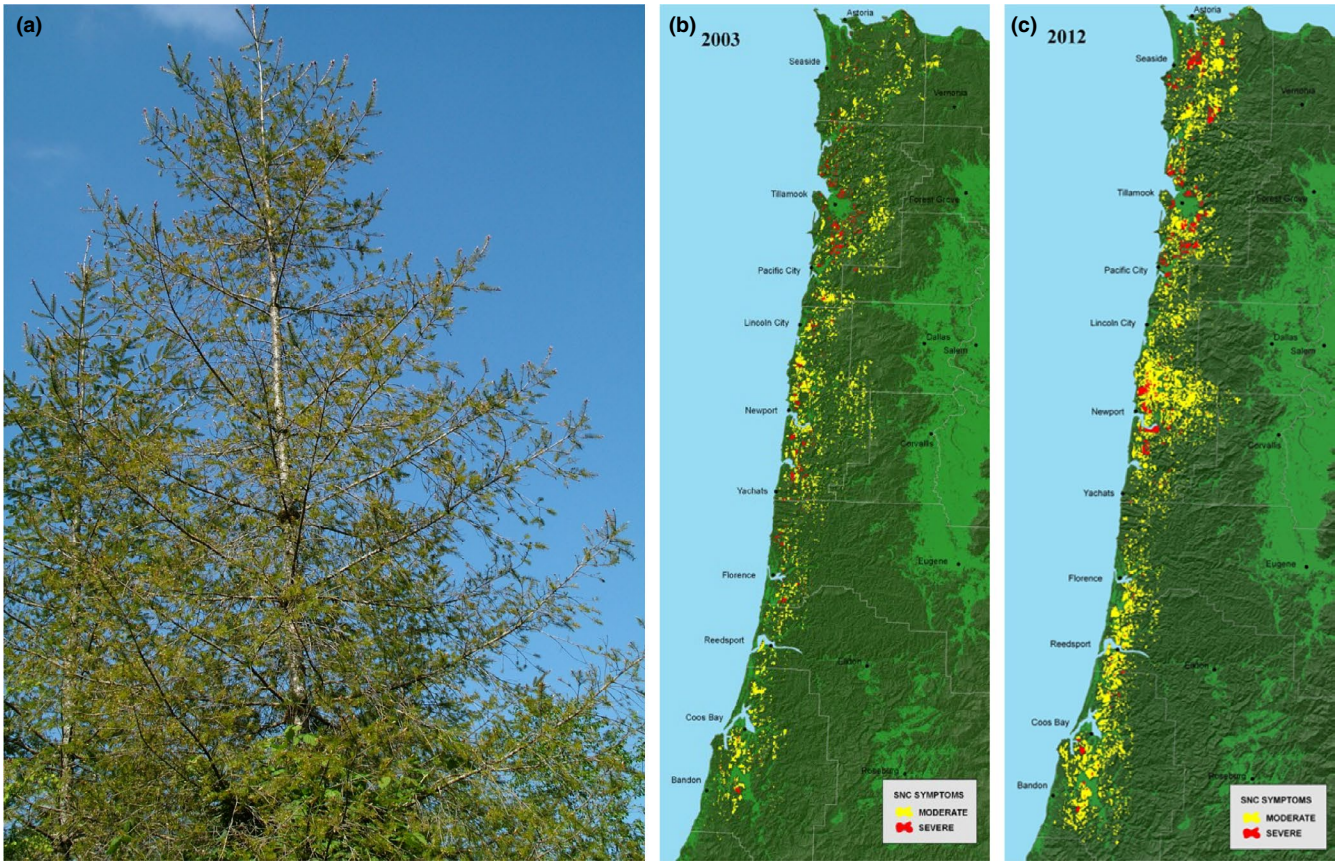

FIGURE 2 Swiss needle cast. (a) Douglas-fir infected with the Swiss needle cast pathogen near Hemlock, Oregon. Photo: Dave Shaw, 2006. (b, c) Maps from aerial detection survey for Swiss needle cast along the Oregon Coast in 2003 (108,369 ha mapped with symptoms) and 2012 (210,184 ha mapped with symptoms). This is the period of study used by Mildrexler et al. (2019) which showed that decade was cooler and moister in May–August. Figure from Oregon Department of Forestry

with seemingly inappropriate seed sources resulted in stands highly susceptible to this native pathogen.

Climate-pathogen. Leaf wetness during May-August promotes needle colonization and subsequent inoculum buildup, while mild fall and winter temperatures appear to facilitate early fruiting of pseudothecia on young needles due to faster colonization within the needle by the fungus (Manter et al., 2005).

Climate models for this disease have used Douglas-fir needle retention as a surrogate for disease severity and are based on precipitation during the spore dispersal period (May–August) and temperatures in winter (non-growing season). Higher precipitation during spore dispersal and warmer winter temperatures decrease needle retention and increase disease severity (Lee et al., 2017; Manter et al., 2005; Rosso & Hansen, 2003; Stone et al., 2008; Watt et al., 2010; Zhao et al., 2011, 2012). Models have had considerable variation in specific months used for climate variables, and this may be related to geographic (elevational) variation causing a shift in which climate variables are most influential (Lee et al., 2017).

Certainty of climate involvement: Confirmed.

The association of mean winter (non-growing season) temperature with disease development has been supported by several studies (Lee et al., 2017; Manter et al., 2005; Stone et al., 2008; Watt et al., 2010). NOAA records indicate that winter temperatures have increased within the symptomatic area during the epidemic (1985 to the present). Additionally, disease expression requires spore germination, which research has associated with spring/early summer leaf wetness (Manter et al., 2005). The amount of low cloud cover in the near coastal area has remained consistent since 1996 (Dye et al., 2020) so is not considered responsible for changes in disease incidence.

Management implications. Douglas-fir remains the most economically valuable timber resource in the PNW due to its abundance, normally short crop rotation interval, and high log prices. Nonetheless, the presence of stagnating or unproductive, diseased Douglas-fir stands has resulted in some liquidation of pre-merchantable stands and reforestation with non-susceptible but less valuable timber species.

Planting of alternative tree species (e.g., *T. heterophylla* and *Thuja plicata* (western redcedar)) is increasing and is recommended in areas prone to severe disease, with Douglas-fir included in planting mixes in increasing proportion as disease pressure decreases. Breeding for tolerance guides tree improvement efforts, with disease-tolerant stock recommended for areas with moderate to low disease levels.

Information gaps/needed research. The role of temperature at the stand and foliar scale needs more attention. SNC severity (expressed as amount of foliage retention) has been modelled across

the landscape with interpolated climate variables (Zhao et al., 2011). Still, there remains a great deal of unexplained variation, probably due to the complex topography of the Oregon coastal mountains. Furthermore, forests along the southern Oregon Coast exhibit limited disease despite a climate conducive to SNC. It is thought that anomalies within the disease zone may be due to wind patterns that reduce spore loads or spring moisture levels. To better understand these factors, leaf wetness sensors and spore trapping will be employed in otherwise similar areas exhibiting contrasting disease levels.

2.3 | Hard pine rusts

Disease: Hard pine rusts (comandra, stalactiform and western gall rust) in western North America (Figure 3).

Forest tree: Lodgepole pine, *P. contorta* var *latifolia*; ponderosa pine, *Pinus ponderosa*; Jack pine, *Pinus banksiana*.

Type of climate effect: Type 1, climate-pathogen disease.

Pathogen: *Cronartium comandrae* Peck, *Cronartium coleosporioides* Arthur, *Cronartium harknessii* (JP Moore) E. Meinecke.

Climate hypothesis: Hard pine rust pathogens are very responsive to weather conditions with narrow environmental windows for portions of their life cycles. These narrow windows have resulted in 'wave years' where most infections are restricted by the timing of a few key climatic events. However, due to climate warming, increasing overnight minimum temperatures have eliminated a critical environmental barrier that had been restricting infection frequency. The result is an increased probability of rust infection each year, particularly in environments where cool overnight temperatures had hitherto reduced infection success.

Summary: A trend towards a greater frequency of years with more spring and summer precipitation and higher overnight minimum temperatures in late summer throughout interior BC has reduced the period between rust wave years (Woods, 2011) to as little as three years on some sites (Reich et al., 2015). This represents an apparent change in disease behaviour from that observed earlier in BC and farther south in the Rocky Mountain states where outbreaks of comandra blister rust had been considered rare (Krebill, 1968). Wave years of western gall rust caused by *C. harknessii* have also been relatively rare historically, occurring approximately once a decade (Peterson, 1971).

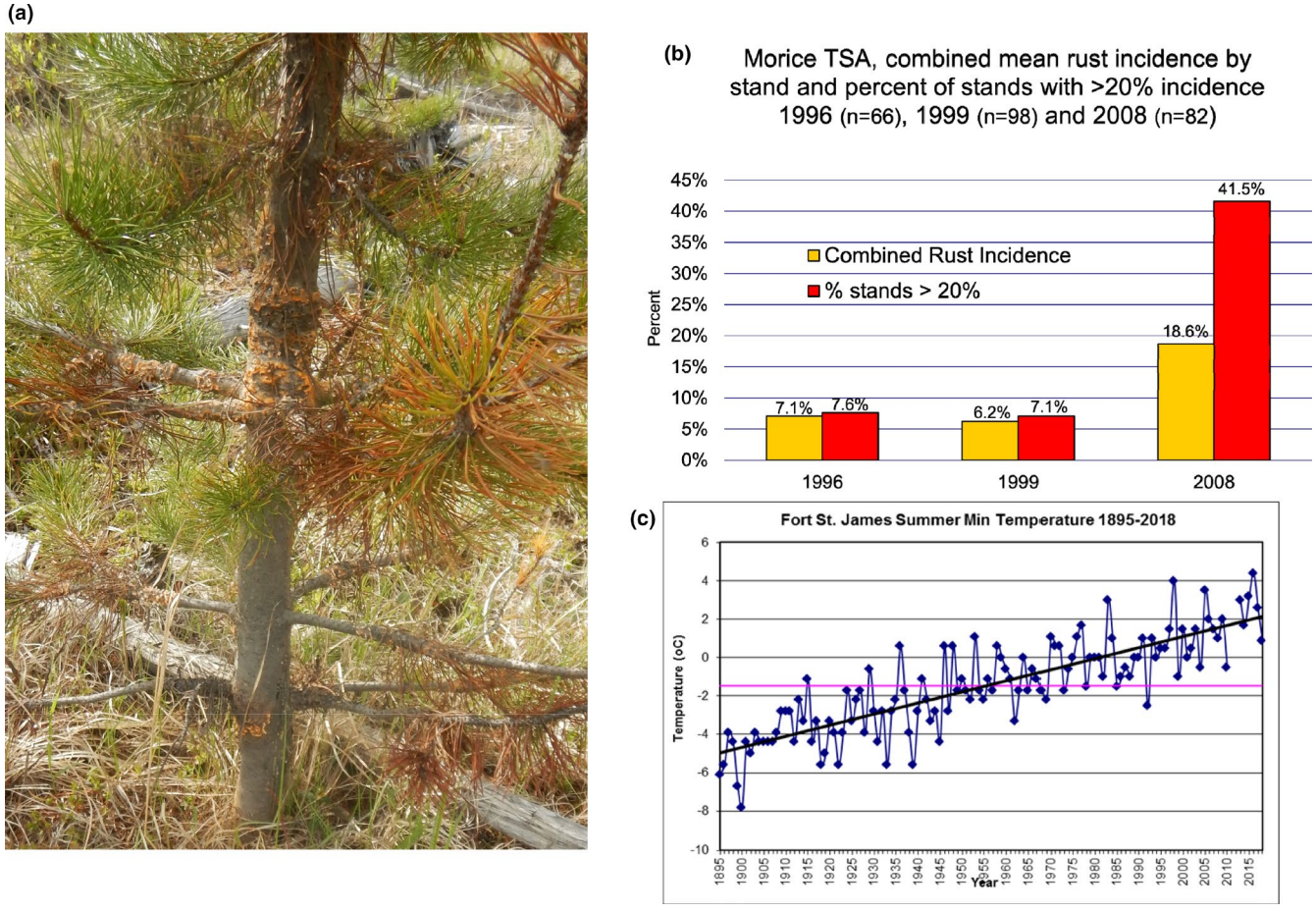

FIGURE 3 Hard pine rusts. (a) Multiple comandra blister rust infections on a young lodgepole pine in British Columbia. (b) Increase in landscape incidence of rust in the Morice timber supply Area (TSA) in central British Columbia. (c) Increase in overnight minimum summer temperatures in the region (Woods, 2011)

Climate–disease spatial. Although a consistent means of recording rust incidence and severity across western North America is lacking, the geographic range of reported increases in disease activity is broad, extending across BC (Heineman et al., 2010; Maclauchlan & Brooks, 2020; Woods et al., 2017) and the western United States (Jacobi et al., 2002). This broad-scale increase in rust incidence has occurred, while the climate across the range of lodgepole pine has warmed, particularly in terms of overnight minimum temperatures in spring and summer. Precipitation patterns across western North America both spatially and temporally are less predictable, so it is difficult to link a trend of increased hard pine rust incidence across broad areas to moisture patterns. The weakening of the jet-stream across North America contributes to this uncertainty (Francis & Vavrus, 2015).

At stand and landscape scales, hard pine rusts are most conspicuous in young stands where the majority of infections occur by age 10 (Blenis & Li, 2005). Stand density appears to have little influence on disease incidence, but lower density pine stands (<1000 sph) suffer greater timber productivity impacts, losing as much as 50% of projected volume at rotation in the most severely rust-affected, lower density stands (Woods et al., 2000). In the lodgepole pine dominated interior plateau of central BC (latitude 52–56°N), hard pine rust incidence is generally higher at lower elevation sites (<800 m). Farther south in the province both the landscape and the forest species composition are more varied and rust incidence generalizations for elevational bands are less clear (Heineman et al., 2010). As widespread warming and more extreme variation in precipitation occurs, prior generalizations about rust incidence levels at different elevational ranges and latitudes, (i.e., hazard ratings) are expected to break down.

Although the influence of stand structural attributes, including host age and the presence of alternate host plants for both comandra and stalactiform rusts, is critical, the influence of weather plays a large part in this host/pathogen relationship (Krebill, 1968; Peterson, 1971).

Climate–disease temporal. The incidence and severity of hard pine rusts are strongly controlled by precipitation and temperature and are thus highly sensitive to yearly differences in weather (Krebill, 1968; Peterson, 1971). A close relationship between comandra blister rust outbreaks and the timing of specific environmental conditions may occur over periods of just a few hours (Krebill, 1968). Wave years for western gall rust require cool moist conditions in late spring (van der Kamp, 1988). Wave years for other stem rusts, including white pine blister rust (*Cronartium ribicola*), comandra and stalactiform blister rust, require moderate temperatures and moist conditions in mid- to late summer (Hunt, 2004; Krebill, 1968; Van Arsdel et al., 1956) during the vulnerable basidiospore stage of the rust life cycle (Peterson, 1971). Optimum overnight minimum temperatures are also critical for wave year events (Reich et al., 2015). Long-term climatic trends at Fort St. James, BC, a weather station located near the centre of the latitudinal range of lodgepole pine in western North America (Wheeler & Critchfield, 1985), indicate a 7°C increase in summer overnight minimum temperatures over the period of 1895–2019 (V. Foord, pers comm., June 2, 2020). The result is an increase in landscape-level rust incidence spatially, as specific infection conditions are met in more locations. Temporally, there is a more even distribution of infection events rather than a concentration in less frequent wave years.

Climate-forest tree: The host tree species, lodgepole, Jack and ponderosa pine, have historically been well adapted to the climate of their respective ranges. Environmental stress on the host trees is, in general, not considered a factor in development of these rust diseases.

Climate-pathogen. The life cycle of most stem rusts has three distinct spore stages which have similar environmental requirements to those well documented for comandra rust (Krebill, 1968) and the introduced pathogen that causes white pine blister rust (*C. ribicola*) (Van Arsdel et al., 1956). The first two spore stages, aeciospores and urediniospores, have relatively wide environmental windows for germination with temperatures ranging from 3 to 28°C, but the most favourable conditions span 8–18°C, provided free water is available (Krebill, 1968). Aeciospores spread from infected pine to alternate hosts in late spring and urediniospores can further increase infection levels spreading among alternate host plants throughout the summer. The narrowest environmental window in the life cycle is when basidiospores are released from alternate host plants that will infect the conifer host, as they require >98% relative humidity, favourable temperatures of 13–23°C and low intensity light in order to germinate (Krebill, 1968). Krebill posits these conditions are only met when warm storm systems remain in areas for several consecutive days in August to early September, resulting in wave years of infection. Wave years for western gall rust require cool moist conditions in late spring during the period of shoot elongation (van der Kamp, 1988).

To date there is no evidence that the native hard pine rust pathogens have increased in virulence due to genetic changes.

Management implications. The extent of lodgepole pine plantations in the central interior landscape of BC has increased significantly since the 1980s. It is possible that the creation of plantations full of young susceptible age classes of trees within a landscape once dominated by mature timber could itself influence rust incidence.

The warmer, wetter conditions and their effect on forest pathogens in central BC are forcing forest managers to consider assisted migration of species such as Douglas-fir (*P. menziesii*) and western larch (*Larix occidentalis*) that are ecologically suitable to these new environmental conditions and immune to these ubiquitous pathogens. The increasingly variable weather conditions across western North American forests call for a greater emphasis on monitoring forest pathogens including hard pine rusts.

Certainty of climate involvement: Probable. A lack of detailed, historic records of disease incidence, and difficulties to precisely date infections, limit our ability to link weather patterns and past climatic conditions with disease expression.

Information gaps/needed research. One of the key information gaps is the translation of stand-level rust incidence in young stands to the volume impact at rotation. A consistent means of long-term

monitoring of rust pathogens in operational managed stands coupled with on-site weather stations capable of hourly assessments would add valuable information.

2.4 | Hemlock dwarf mistletoe

Disease: Hemlock dwarf mistletoe (Figure 4a–c).

Forest tree: Western hemlock, *T. heterophylla*.

Type of climate effect: Type 1, climate-pathogen disease.

Pathogen: *Arceuthobium tsugense* (Rosend.) G.N. Jones.

Climate hypothesis: An aspect of the reproductive or dispersal biology of this dwarf mistletoe, likely either fruit and seed survival/maturation or over-winter seed retention in tree crowns, is favoured by a warmer climate with longer growing seasons, reduced snow or alternatively by non-lethal winter temperatures.

Summary: The elevational and north-western (geographic) range distributions of hemlock dwarf mistletoe relative to its primary host, western hemlock, suggests climate limitations on the disease. This is apparent to the north in Alaska where mistletoe brooms recorded in forest inventory plots show the extent of the disease. Interpretation of the plot data in the southern distribution of the tree and pathogen is complicated by the presence of multiple dwarf mistletoe species and numerous susceptible host tree species. Climate controls on pathogen distribution are suspected to be a limiting factor, but the mechanism has not been demonstrated. It is theorized that climate operates as a threshold to constrain distribution, but the pathogen is already well adapted to climate within its range, so further warming and reduced snow are not expected to produce greater disease severity in areas that are currently infested.

Climate–disease spatial. The main evidence for a climate control of hemlock dwarf mistletoe is the restriction of the pathogen and disease to a subset of the host tree range, which occurs from California to south-central Alaska. The mid- and broad-scale spatial distribution of hemlock dwarf mistletoe is limited by elevation and to the northwest range in the northern portion of its range in Alaska. Western hemlock is abundant to about 800 m elevation in Alaska at latitudes 54–59°N where hemlock dwarf mistletoe occurs (Caouette et al., 2016). Near sea level at that latitude, hemlock dwarf mistletoe infects about 20 percent of western hemlock trees, but the pathogen becomes far less common above 200 m and drops to about two percent infection at 400 m in elevation (Barrett et al., 2011). Climate modelling of the host tree and pathogen supports the hypothesis that geographic influences limit disease in Alaska but cannot confirm which aspect of climate is responsible for limiting disease (Barrett et al., 2012). For climatic or other unknown reasons such as the discontinuous presence of susceptible host trees, hemlock dwarf mistletoe does not occur in the continental interior populations of western hemlock in Idaho, Washington, Montana and British Columbia.

Using vegetation inventory plots to determine elevational limits of the disease is more challenging in the southern portion of the range (California through Washington) due to the presence of both multiple subspecies of the pathogen and several susceptible tree species. Here, the best evidence of climate influence on disease extent is from field observations. The mistletoe subspecies that affects western hemlock, *A. tsugense* subsp. *tsugense*, is reported from sea level to 1300 m in Oregon (Hawksworth & Weins, 1996), but susceptible hosts are also found at higher elevations. Observations of elevational limits on the other subspecies of dwarf mistletoe are also reported (Hawksworth & Weins, 1996; Mathiasen, 2021). Climate modelling for this dwarf mistletoe species has not been conducted for its southern distribution.

The occurrence of dwarf mistletoe on western hemlock at the finer spatial scale, within and between stands, is likely controlled more by disturbance history (Trummer et al., 1998), including fire and timber harvest, than by climate or soils. Dwarf mistletoe thrives in late-successional forests because gap-phase dynamics facilitate mistletoe seed dispersal (generally <10 m distance) (Robinson & Geils, 2006; Shaw & Hennon, 1991).

Climate–disease temporal. Dwarf mistletoes are chronic, slow-developing tree diseases. Individual hemlock dwarf mistletoe infections can survive for a century or more. The lack of annual and probably decadal fluctuation of disease complicates attempts to link the disease to recent and mid-term climate data. Relationships between climate and disease severity may be more discernable by examining long durations (i.e., decades), suggesting that bioclimatic niche modelling, based on the association of the pathogen distribution, may be useful. Using several climate scenarios, Barrett et al. (2012) projected a 374–757 percent increase in extent over a century in the potential climate space for hemlock dwarf mistletoe on western hemlock in Alaska; however, slow migration limited by pathogen dispersal will limit the actual extent of broad-scale spread.

Climate-forest tree. The primary host tree, western hemlock, appears well suited to current and future precipitation and warming conditions in northern portions of its range where the species is likely to extend its distribution (Barrett et al., 2012). Short-term droughts may increase dwarf mistletoe damage to western hemlock (Bell et al., 2020) and fire can eliminate both the host tree and disease (Shaw & Agne, 2017).

Climate-pathogen. The main climate effects that increase dwarf mistletoe intensity and range likely influence pathogen reproductive and dispersal biology. Short growing seasons or snow may explain the current climatic restrictions of disease. As these conditions are lifted by a warming climate, there are three likely scenarios for future upslope and north-westward expansion of the disease: (1) longer, warmer growing seasons allow fruit to mature and seeds to disperse before first frost in the fall; and (2) reduced snow due to warmer winters lessens sloughing snow that previously could remove overwintering seeds; (3) freezing damage to seeds no longer occurs. More seeds would persist in canopies to enable spring infection. The basis for the first hypothesis, freezing of mistletoe berries, has been observed in British Columbia (Baranyay & Smith, 1974). Expansion of range of this dwarf mistletoe would serve as a classic example of the lifting of climate restrictions through climate warming; yet, dwarf mistletoe may have a slow rate of migration to the newly suitable

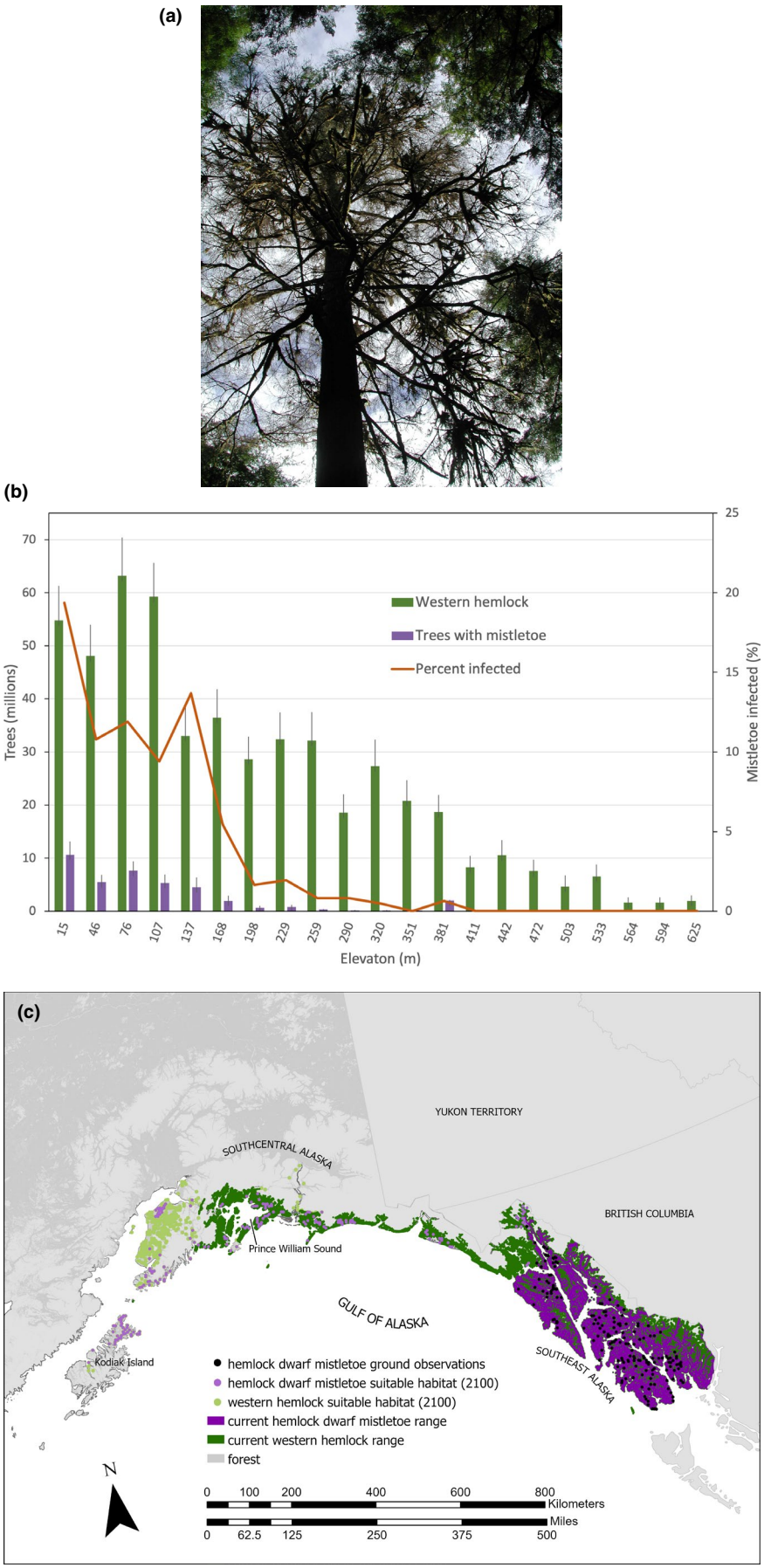

FIGURE 4 Hemlock dwarf mistletoe. (a) Dead mature western hemlock showing numerous hemlock dwarf mistletoe brooms used to detect the disease in forest surveys. Photo: US Forest Service. (b) Estimated numbers of western hemlock and trees with hemlock dwarf mistletoe and percentage of mistletoe-infected western hemlock trees (brown line) in 30 m (100 ft) elevation classes in southeast Alaska, latitude 54.5–59°N. Modified from Barrett et al. (2011). (c) The current ranges of western hemlock in Alaska where hemlock dwarf mistletoe is present (purple) and absent (dark green). Disease presence is based on Forest Inventory and Analysis plots and other ground observations. Projected potential 2100 ranges of the western hemlock habitat with (light purple) and without mistletoe infection (light purple) from bioclimate niche models (Barrett et al., 2012)

climatic habitat within the current and projected potential ranges for the tree (Hawksworth & Wiens, 1996). Analysis and modelling identified a range of environmental factors that currently restrict hemlock dwarf mistletoe in Alaska (growing degree-days, indirect and direct solar radiation, rainfall, snowfall, slope, and minimum temperatures (Barrett et al., 2012)), but because of covariance in these factors, they could not be used to distinguish the actual mechanism that limits the disease.

Management implications. There is a wealth of information on silvicultural treatments (e.g., thinning to foster healthy regeneration) to manage dwarf mistletoes (Hawksworth & Wiens, 1996). Climate change may not be a major factor in disease management decisions because of slow apparent response to changes in atmospheric conditions by the pathogen, and the relative simplicity of the management actions needed in areas where disease impacts are considered unacceptable. For resource managers interested in promoting the positive wildlife and ecosystem values of this mistletoe, increasing fires and drought in the southern portions of hemlock dwarf mistletoe's range due to climate change may be the biggest limit to this disease.

Certainty of climate involvement: Tentative. Climate involvement in hemlock dwarf mistletoe incidence, severity and potential range expansion is provisional. Certainty would be greater if the specific climate mechanisms that limit reproduction or dispersal of this dwarf mistletoe were firmly established through observation or experimentation. A similar scenario of the host extending higher in elevation than the pathogen has been described for lodgepole pine dwarf mistletoe (*Arceuthobium americanum* Nutt. ex Engelm.) on lodgepole pine (*P. contorta*) in the Rocky Mountains (Hawksworth, 1956; Hawksworth & Wiens, 1996) and western spruce dwarf mistletoe (*Arceuthobium microcarpum* (Engelm.) Hawksw. & Wiens) on Engelmann spruce (*Picea engelmannii*) (Acciavatti & Weiss, 1974; Mathiasen & Hawksworth, 1980).

Information gaps/needed research. Monitoring could be directed to the elevational and northwest limits of the pathogen to detect any migration of the disease consistent with model predictions (Barrett et al., 2012; Muir & Hennon, 2007). The complete absence of the disease in the interior continental western hemlock populations is not well understood (Smith & Wass, 1979) and may be related to geographic gaps in host tree distributions and details of parasite dispersal biology.

2.5 | Sudden aspen decline

Disease: Sudden aspen decline (Figure 5a,b).

Forest tree: Quaking aspen, *Populus tremuloides*.

Type of climate effect: Type 2b, climate-stress disease, important secondary agents.

Pathogen or other associated organisms: *Cytospora* spp., aspen bark beetle (*Trypophloeus populi*), bronze poplar borer (*Agrilus liragus*), and others.

Climate hypothesis: Severe, hot drought stresses aspen, which increases susceptibility to secondary agents. Some agents then become more aggressive due to their increased population density.

Summary: Sudden aspen decline (SAD) occurred following the turn-of-the-century drought in western North America, considered a climate-change type drought because it was hot as well as dry (Breshears et al., 2005). The drought peaked in 2002 and affected a great swath from western Mexico to the prairie provinces of Canada, with record extreme conditions. Branch dieback, crown thinning, and stem and root mortality of aspen occurred on a landscape scale.

Climate–disease spatial: At fine (stand) and middle spatial (landscape) scales, SAD occurs primarily in stands older than about 35 years. Open stands are more vulnerable than dense stands (Worrall et al., 2008, 2010), as the forest floor and stems are exposed to the drying effect of sun and wind. SAD is inversely related to elevation, low elevations having higher temperatures and lower precipitation. It also occurs preferentially on south and west aspects and on upper slope positions, all related to drought vulnerability. At

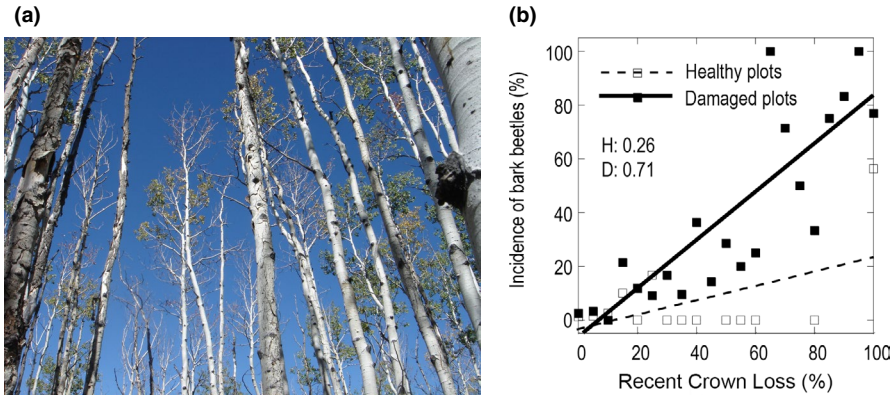

FIGURE 5 Sudden aspen decline. (a) Sudden aspen decline, Colorado, USA, in 2011. (b) Incidence of aspen bark beetles in trees grouped into crown-loss classes, in plots with SAD damage vs. healthy plots. Even in trees with high crown loss, few trees in healthy plots had beetles except for dead ones. In plots with damage, incidence of beetles increased strongly with crown loss. Because beetles were abundant in damaged plots, they could cause more damage for a given amount of stress (as indicated by crown loss) than in healthy plots. From Marchetti et al. (2011)

the range-wide scale, SAD occurred within the swath of the turn-of-the-century drought and where aspen was a dominant species, including the aspen parkland of Alberta and Saskatchewan, southern Wyoming, Colorado, Utah, northern Arizona, and New Mexico (Michaelian et al., 2010; Worrall et al., 2013). All these areas had warm, dry extremes during the drought. In Colorado, areas with SAD had greater moisture deficits in 2002 than did healthy areas (Worrall et al., 2010). A random forests bioclimate model showed that areas where sudden decline occurred are on the fringe of the realized climate niche for aspen and, consequently, are and have been the most vulnerable to such a drought (Rehfeldt et al., 2009).

Climate–disease temporal. The turn-of-the century drought was the most severe in the instrumental record, both locally and westwide (Schwalm et al., 2012; Worrall et al., 2013). Schwalm et al. (2012) estimated that it was the most severe in over 800 years. It peaked in 2002 and, in most areas, ended by the following winter. What later became known as SAD was first detected in southwest Colorado in 2004 and spread and intensified over the following 4–5 years. However, mortality was already widespread and intense in the aspen parkland of Alberta when surveyed in 2004 (Michaelian et al., 2010). The bioclimate model mentioned showed that, from 1950 to 2006, 2002 had the most extreme deviations in variables most important in determining the distribution of aspen (Rehfeldt et al., 2009). These include an annual dryness index, the ratio of summer to annual precipitation, and an interaction of growing season precipitation with the summer–winter temperature differential.

Climate-forest tree. *Populus tremuloides* has the lowest drought tolerance of all the co-occurring tree species in the southern Rocky Mountains except for *Populus angustifolia*, which has equal tolerance (Niinemets & Valladares, 2006). The latter is strictly a riparian species that depends on water tables rather than upland soil moisture. The stems have a short lifespan, and there is a fair number of pathogens and insects that attack it, either killing stressed trees or killing or weakening vigorous trees.

Climate-pathogen (and other secondary agents). There is no evidence to suggest, and it seems unlikely, that drought directly favours the biotic agents that are important in SAD. Instead, the drought-stressed trees become more susceptible to the agents. In addition, there is evidence that when populations are increased by host stress and mortality, some agents become more aggressive and can then successfully attack healthy trees (Marchetti et al., 2011).

Certainty of climate involvement: Confirmed.

The factors above consistently support the turn-of-the-century drought being the inciting factor for SAD. The drought was considered a climate-change-type drought. The drought was a weather event, but bioclimate models indicated that sites where SAD occurred are climatically vulnerable to drought. SAD distribution was consistent with projections that link the bioclimate model to future climates. In three aspen bioclimate models with different spatial scales and sample data, the most important variables were all relevant to drought (Rehfeldt et al., 2009, 2015; Worrall et al., 2013).

Management implications. Timely coppice cutting, before about 50% of the stand is killed, can stimulate adequate regeneration (Ohms, 2003; Shepperd et al., 2015). Stands < 40 years old were more tolerant, so regeneration of aspen should increase resilience to drought and SAD. However, avoid management for aspen where bioclimate models indicate that future suitability is very low. Instead invest management in areas where suitability is moderate to good. Because of its light, airborne seeds, aspen is more likely than most species to colonize newly suitable climatic habitat, and this can be enhanced by appropriate disturbance.

Information gaps/needed research. Although soil traits should affect drought vulnerability, field assessment of soil pits in healthy and diseased plots revealed no significant soil traits (Worrall et al., 2010); this bears further study. Because the secondary agents are ubiquitous, it is unclear whether, or how commonly, severe drought in poor sites can kill trees directly, without intervention of pathogens and insects. It is also unknown why there is a stand age threshold for SAD of about 35–40 years. Determining why the disease began earlier and advanced more quickly in the aspen parkland than in the southern Rockies would also advance understanding of the disease.

2.6 | Western white pine pole blight

Disease: Western white pine pole blight.

Forest tree: Western white pine (*Pinus monticola*).

Type of climate effect: Type 2a, climate-physiological disease. Abiotic stress is the cause of mortality; secondary organisms also occur but are not considered significant.

Pathogens or other associated organisms: *Armillaria* spp., *Leptographium* sp., and *Dendroctonus ponderosae* (mountain pine beetle) occur on some trees in advanced stages of decline at some locations.

Climate hypothesis: Prolonged stress from severe, multi-year drought at sites with thin soils cause dominant and codominant western white pines in pole-stage stands to undergo slow, whole-tree decline leading to mortality. The disease results from interacting effects of extended or recurrent, severe drought, soils prone to water deficit, and physiological factors including root system and resource allocation characteristics of the tree species.

Summary: Western white pine pole blight (pole blight) is a historically important, but little-recognized climate-stress disease of 40- to 100-year-old western white pine characterized by reduced radial and leader growth, crown thinning, chlorosis, and long, narrow, resinous lesions at the bole cambium, resulting in crown deterioration and mortality (Leaphart, 1958a, 1958b; Leaphart & Gill, 1955; Molnar, 1955; Wellner, 1947) (Figure 6a). Pole blight was first reported circa 1929 in northern Idaho (Leaphart et al., 1957), many years into a severe, prolonged drought that extended from about 1915 to 1960 (Leaphart & Stage, 1971). Since then, pole blight has not been considered a significant disease. However, in 2017–2020, pole-sized white pines with characteristic symptoms were observed at two drought-prone locations in Idaho (P. J. Zambino, personal observations).

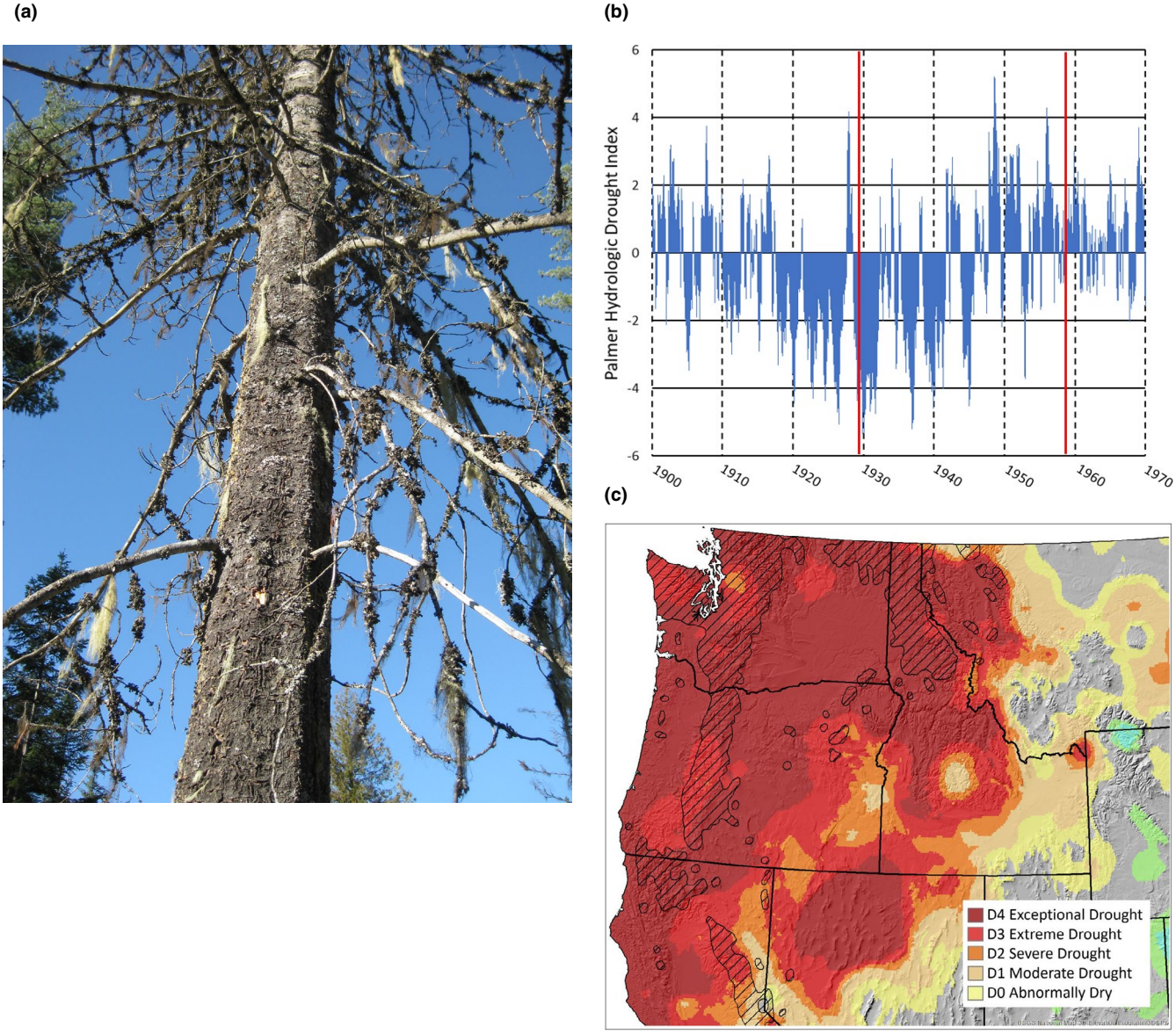

FIGURE 6 Pole blight of western white pine. (a) Recent symptoms in pole-aged trees in a stand that historically had pole blight, including slowed growth and thin crowns progressing to whole-tree mortality and multiple flat faces from long, linear, resinous lesions of the cambium typical of pole blight (Zambino, pers. obs. 2019). (b) Palmer Hydrologic Drought Index for Idaho Climate Division 1 ("Idaho Panhandle Climatological Division") from 1900 to 1970 (NOAA, 2021a). Vertical lines represent the first record of pole blight mortality (Leaphart et al., 1957) and the last surveyed increase in disease extent and severity (Graham, 1955, 1958). (c) Standardized Precipitation Index for the 9-month period ending November 1929 (NOAA, 2021b), the year when pole blight was first recorded (Wright & Graham, 1952). Cross-hatched areas indicate the geographic range of western white pine (Little, 1971)

Climate–disease spatial. Pole blight occurs during prolonged drought. In affected areas in both the panhandle and central mountains of Idaho in the first half of the 1900s, the Palmer Hydrologic Drought Index (PHDI) rarely departed from −1 to −4 for recurrent periods and occasionally reached PHDI-5 (Figure 6b) (NOAA, 2021a). Drought was widespread throughout the PNW (Figure 6c), with pole blight documented in most of the interior range of western white pine including northern Idaho, western Montana, eastern Washington (Graham, 1955, 1958; Wright & Graham, 1952) and interior southern British Columbia (Parker et al., 1950). Only a few affected locations were recorded outside the interior white pine region, including coastal British Columbia (Parker, 1951), where drought also occurred.

Differences in pole blight incidence and severity are discernable at fine, middle, and broad spatial scales. At the middle-to-broad spatial scale, aerial surveys displayed a patchwork of diseased and healthy, naturally regenerated, pole-sized western white pine stands (Wright & Graham, 1952). Differences in disease prevalence between stands were associated with the spatial distribution of soil types and other factors (Copeland & Leaphart, 1955; Leaphart, 1958a, 1958b). Areas with pole blight were characterized as having low soil moisture-holding capacity, less than 12.7 cm available water

in the top 81.3 cm of soil (Copeland & Leaphart, 1955; Graham, 1959). At the fine spatial scale (within stands), the disease affected dominant and codominant trees but not suppressed trees (Leaphart, 1958a).

Climate–disease temporal. Pole blight was supported by a mid-scale, temporal, climatic event: a decades-long period of recurring droughts corresponding with Pacific Decadal Oscillation (PDO)-conditions that typically cause warmer and drier winter conditions in coastal and interior areas of the PNW (Newman et al., 2016). Onset and expansion of the disease occurred in the late 1920s–1950s, within the period considered by Leaphart and Stage (1971) as the most prolonged and severe drought in the Inland Northwest over the previous 280 years. The disease appeared several years into the drought and developed and intensified (Graham, 1955, 1958) until drought severity lessened. Some severely affected trees continued to die even after drought abated. Symptoms and mortality generally correlated with the severity and duration of the moisture deficit (Graham, 1958). Current and predicted increased drought severity in the upper reaches of the Missouri basin (Martin et al., 2020) and Inland Northwest (Gutzler & Robbins, 2011; Overpeck & Udall, 2020; Sheffield & Wood, 2008) could increase the threat of pole blight at susceptible locations.

Climate-forest tree. Western white pine is vulnerable to pole blight because of its rooting pattern in thin soils. Western white pine has a low rootlet to structural root ratio; pine rootlets support larger amounts of structural root than other species (Leaphart, 1958b). The species thrives in deep soils, two feet or more in depth. In stands on thin soils that developed pole blight, both symptomatic and asymptomatic western white pines were shown to have dramatically reduced numbers of fine roots and mycorrhizae, with the greatest reductions present on symptomatic trees (Gill et al., 1949). Western white pine is considered more drought intolerant than co-occurring species (Graham, 1990; Leaphart & Wicker, 1966; Minore, 1979); Douglas-fir and western redcedar (*T. plicata*) have total root length per soil volume and fine roots in upper soil layers that are typically many times that of western white pine (Leaphart & Grismer, 1974). In soils conducive to pole blight, these species do not show whole-tree mortality from drought stress.

Climate-pathogen. *Armillaria* and *Leptographium* root pathogens were frequently isolated from trees with pole blight at some sites (Gill et al., 1949; Leaphart et al., 1957; Leaphart & Gill, 1955). There are many unknowns as to how their behaviour in western white pine stands is affected by climatic conditions.

Management implications. In areas within the geographic range of western white pine where drought may be severe, of long duration, or recurrent, land managers should consider planting this species only on sites with deep soils, good water-holding capacity and adequate soil moisture recharge (Leaphart, 1958a). On naturally regenerating sites with less favourable soils, managers may consider retaining a mixture of canopy classes and including tree species with greater drought tolerance.

Certainty of climate involvement: Supported. There is little contemporary literature on pole blight, but research published in the 1950s–1970s demonstrated that drought is the principal factor for pole blight.

Information gaps/needed research. A predictive pole blight risk factor model could be constructed based on current understanding. Such a model would include the geographic distribution of white pine, soil types and age classes as predisposing factors, and potential for drought index extremes as a dynamic climate input factor. Also needed are investigations of root system development for pole-sized trees growing in multi-cohort stands (Jain et al., 2020) versus uniform stands and evaluations to determine if uneven management, with white pines developing in gaps of mature stands, would have less inherent stress and a reduced tendency to develop pole blight.

2.7 | Yellow-cedar decline

Disease: Yellow-cedar decline (Figure 7a–c).

Forest tree: Yellow-cedar, Alaska-cedar, *Callitropsis nootkatensis*.

Type of climate effect: Type 2a, climate-physiological disease.

Pathogen or other associated organisms: *Armillaria* spp., *Phloeosinus* sp. (bark beetles), others, but none are important as a cause of death.

Climate hypothesis: Yellow-cedar trees die from freeze injury when shallow fine roots are killed by temperatures <−5°C during repeated cold weather events on sites that lack protective insulating snowpack to mitigate the disease.

Summary: Research on yellow-cedar decline was initiated in the 1980s to explain the cause of long-term, widespread mortality of this tree species. After ruling out other possible causes, the role of climate in yellow-cedar decline was proposed (Hennon & Shaw, 1994), and research progressed by establishing associations of climate and disease (Hennon et al., 2012) including a range-wide analysis for yellow-cedar (Buma et al., 2017). Root freezing injury was replicated experimentally (Schaberg et al., 2008), and the hypothesis stated above is now generally accepted (Oakes et al., 2016).

Climate–disease spatial. The occurrence of yellow-cedar decline has been mapped by aerial survey in Alaska and British Columbia and placed into a range-wide context for this tree species (Buma et al., 2017). Mortality is centred on landscapes with a modelled mean temperature in the coldest month of −5 to 0°C (Buma, 2018). In Alaska, yellow-cedar decline consistently aligns with climate patterns where annual precipitation as snow is below 250 mm (Hennon et al., 2012). The relationship between this threshold snow level and mortality was observed at spatial scales ranging from fine (stand), watershed (D'Amore & Hennon, 2006) to regional and Alaska-wide (Hennon et al., 2016). In British Columbia, mortality and related climate factors are present at Haida Gwaii, indicating that periodic subfreezing cold temperatures can extend to the most ocean-moderated temperature (hypermaritime) areas to kill trees (Comeau et al., 2019). Soils interact with climate at the fine spatial scale, as water-saturated soils cause shallow soil-root profiles that promote the disease, whereas trees remain healthy where they are deeply rooted—even in areas without adequate snow protection (D'Amore & Hennon, 2006; Hennon et al., 1990). Open-canopy stands on

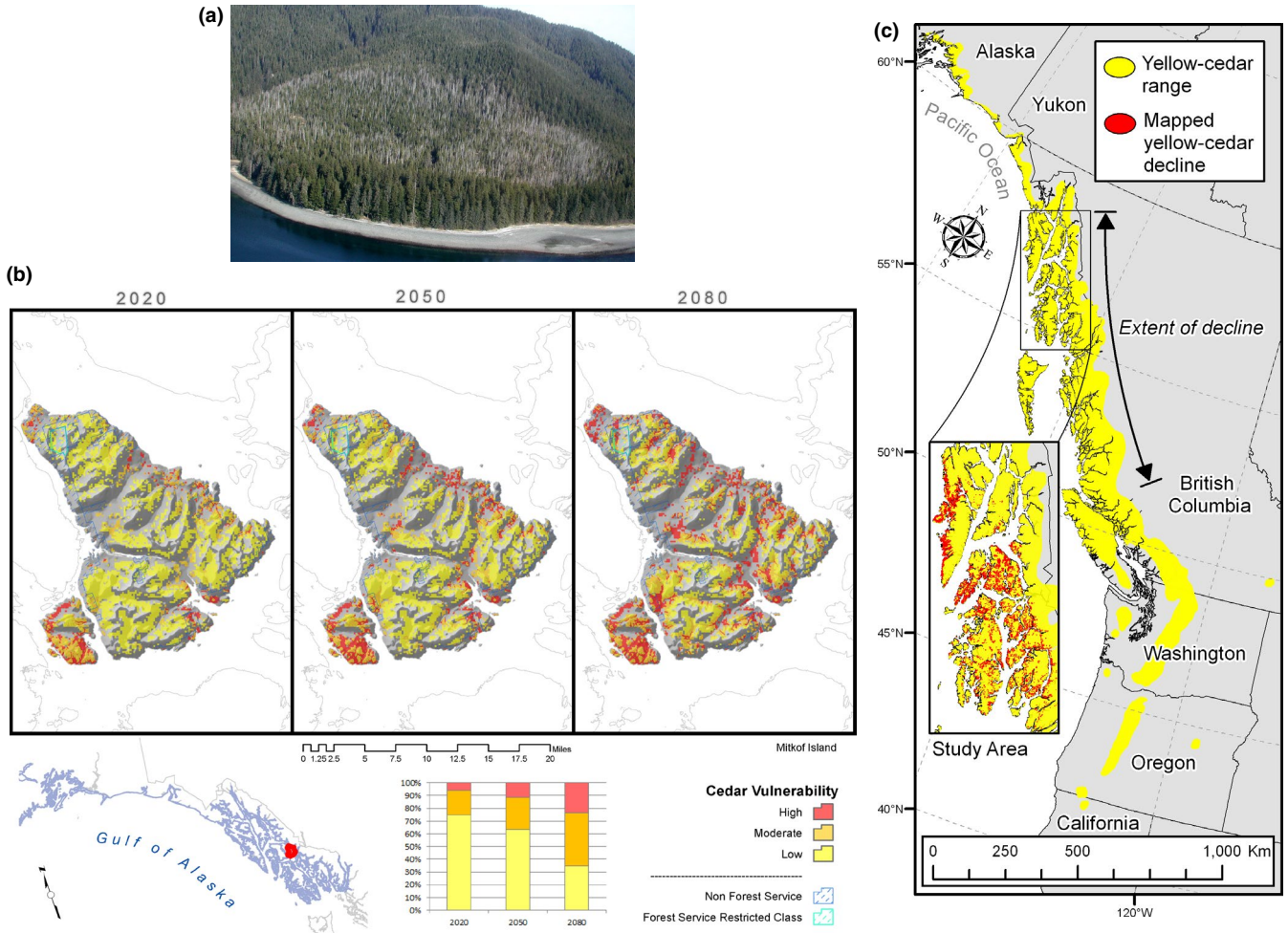

FIGURE 7 Yellow-cedar decline. (a) Patch of yellow-cedar decline at Poison Cove, Alaska, the site of some of the research on fine-scale aspects of decline such as microclimate, soils, and stand structure. Photo US Forest Service, 2004. (b) Risk of yellow-cedar decline at Mitkof Island, one of 33 mid-scale analysis zones in coastal Alaska, modelled at 30 m pixel resolution but displayed at 240 m. Modelled low (yellow), medium (orange) and high (red) risk of decline overlays the occurrence of yellow-cedar; risk categories are derived from equally weighted annual snow accumulation and drainage models. Future projections of snow accumulation from averages of general circulation models with conservative emission scenarios. From Hennon et al. (2016). (c) The range of yellow-cedar showing the latitudinal extent of decline and (inset) mapped yellow-cedar decline in Alaska. From Hennon et al. (2012); see Buma et al., 2017 for a complete mapped distribution of yellow-cedar decline including occurrence in British Columbia

wetter bog soils create conditions conducive to more extreme cold and fluctuating temperature microclimates that exacerbate the disease at the stand scale (Hennon et al., 2010).

Climate–disease temporal. Dating the year of snag creation allowed a reconstruction of tree mortality over time and established that the earliest period of broad-scale mortality coincided with the end of a natural climate cycle—the Little Ice Age—before 1900 (Hennon et al., 1990b). Weather station and cedar dendrochronological data showed that snow decreased throughout the twentieth century, but the frequency of damaging short-term, late-winter freeze events was maintained, so mortality was consistent with the climate hypothesis stated above (Beier et al., 2008). Accelerated mortality in the 1970s and 1980s was associated with a departure from typical climate decadal oscillations to indicate anthropogenic climate change (Hennon et al., 2012). Hourly monitoring of soil temperatures in forests revealed that lethal in situ soil temperatures (<−5°C) do not occur when snow is present, and when snow is absent, temperatures lethal to fine roots are confined to shallow soils during cold weather events (Hennon et al., 2010). Known risk factors for the decline disease (snow and drainage) were incorporated into a general risk model with climate projections to predict future spread of the disease (Hennon et al., 2016). Northerly spread of the disease, consistent with these models, has been confirmed by field observation (Oakes et al., 2014). A 'transitional mortality', where a climate window for tree vulnerability that moves across geographic space, explains this spatio-temporal movement of the disease appearing and abating through time at the range-wide scale (Buma, 2018).

Climate-forest tree. Cold tolerance hardening and dehardening of yellow-cedar are driven by weather conditions more than by photoperiod (Schaberg et al., 2005). Yellow-cedar fine roots are more vulnerable to freeze injury than co-existing tree species (Schaberg et al., 2011) which explains why yellow-cedar is the tree that is

impacted. Hypothetical root freeze injury to trees has been replicated experimentally as has the mitigation-protection afforded by snow (Schaberg et al., 2008). Fine roots, which are the initial tissues affected in the disease (Hennon et al., 1990c), are killed by soil temperatures <−5°C as established by cold tolerance laboratory testing, whether roots are from seedlings or mature trees.

Climate-pathogen. A full examination of biotic factors including inoculations showed minimal involvement of fungi (Hennon et al., 1990c, 2016; Hennon & Shaw, 1997), indicating that this is primarily a tree stress disease. The *Phloeosinus* bark beetle could be favoured by warmer weather, but this insect is clearly of secondary importance to root freeze injury.

Management implications. Resource managers are now using a climate adaptation strategy to guide conservation and active management, including where to favour yellow-cedar by planting and thinning in climate and soil environments favourable to yellow-cedar but not to conditions driving mortality (Hennon et al., 2016).

Certainty of climate involvement: Confirmed.

The Fox (2020) causation criteria have been met, including consistency between climate and the disease, experimentation to replicate the cause and no plausible alternative hypothesis.

Information gaps/needed research. Quantifying the horizontal and vertical distribution of fine roots in mature trees has been a vexing problem but is needed to better understand shallow root freezing injury. This would help explain whether the 20–30 percent of yellow-cedar trees that typically survive in disease-affected stands (Hennon et al., 2012; Oakes et al., 2014) differ in their rooting habits or genetic traits. The most pressing information needs are now related to adapting management of yellow-cedar to climate change, including conservation monitoring of decline in young managed stands (FS-R10-FHP, 2020) and regeneration strategies in favourable climate and edaphic habitats.

3 | DISCUSSION

3.1 | Demonstrating climate-causality for tree diseases

The climate–disease framework of Hennon et al. (2020) facilitated the systematic evaluation of climate involvement in the tree disease examples presented. These examples from western North America include climate-pathogen diseases (climate modulates disease by directly influencing a pathogen, Type 1) and both types of climate-stress diseases (climate directly influences host trees to alter disease, with or without substantive involvement of secondary pathogens and insects, Types 2a and 2b). Example diseases of conifers and hardwoods, in both coastal and continental environments, were affected by climate; all are suitable for this climate causality framework but with varying levels of certainty. Disease increases and decreases were linked to key meteorological conditions at different times of year.

In the examples where climate modulates disease by its direct influence on the pathogen (Type 1), the causal agent was already known, but, initially, the reason for altered disease expression was not. Specific weather conditions (or climate trends of repeated weather patterns) enhanced various aspects of pathogen reproductive and infection biologies in the Dothistroma needle blight, Swiss needle cast and hard pine rust pathosystems. Weather conditions can alter fungal biology and thereby change the impact on the host from parasitic or endophytic, causing no apparent symptoms, to pathogenic, as occurs in Swiss needle cast in the PNW.

The example of hemlock dwarf mistletoe differs from the other examples in that there is no evidence of varying disease intensity within the pathogen's current range to compare with weather records, but range extensions are expected as climatic barriers are lifted by warming. Range expansions are likely to develop gradually, as spread rates are limited by the ability of the pathogen to disperse into new environments.

The other type of climatic influence on tree disease is by direct physiological stress or injury to the trees. In our three 'Type 2, climate-stress disease' examples, the cause of disease was not initially known. Preliminary investigations examined and eventually deduced that insects or pathogens were not the primary cause. Climate factors were then considered as part of the cause by exploring the relationship between disease expression and weather records. The onset, duration and remission of sudden aspen decline and pole blight of western white pine were found to be closely associated with droughts. The cause of yellow-cedar decline was determined to have two climate factors: short-term cold temperatures that kill fine roots and, as an essential predisposing condition, reduced insulating snowpack that historically protected against injury. For this long-lived species, sites which were previously adequately protected by snow have become unsuitable with the loss of snowpack.

Opportunistic secondary organisms are often present on dying trees, regardless of the primary cause of death. It is a challenge to distinguish between the Type 2a and 2b diseases; that is, do weather conditions stress the tree sufficiently for direct mortality (e.g., western white pine pole blight and yellow-cedar decline)? Or, do secondary insects or pathogens kill stressed trees that otherwise might recover (e.g., sudden aspen decline)? This distinction requires focused observation, knowledge of the behaviour of the secondary agents following other kinds of stress, and possibly experimentation with all three factors (climatic conditions, tree species, secondary organisms) to determine individual and combined effects. For instance, a shoot blight fungus *Diplodia sapinea*, (formerly *Sphaeropsis sapinea*)—not one of our examples—was found to occur as a latent organism until its host, red pine (*Pinus resinosa*), experienced water stress (Stanosz et al., 2001). When trees are under stress, endophytic fungi, such as members of the Botryosphaeriaceae, have been implicated in mortality of numerous woody hosts worldwide (Slippers & Wingfield, 2007). During early stages of mortality investigations, or in response to intense drought or other stressors, the large number of secondary and saprophytic agents recovered,

possibly from multiple declining tree species, can complicate causality determinations.

It is also conceivable that climate factors may influence both the host tree and the primary pathogen to alter disease expression, thereby blurring our distinction between Type 1 and 2 diseases. None of our seven disease examples followed this scenario. However, for many stress-related Cytospora cankers, warming could increase the rate of growth and sporulation and extend the active season of the pathogen, while also increasing moisture stress of the host tree due to warmer conditions and extended growing season (Bloomberg, 1962; Butin, 1955). Similar dual effects of climate influence on the host and pathogen could be anticipated for many stress-related diseases (e.g. some Armillaria root diseases (Sturrock et al., 2011; Wargo & Harrington, 1991), sooty-bark disease of maple (*Cryptostroma corticale*, Ogris et al., 2021), pitch canker (*Fusarium circinatum*, Elvira-Recuenco et al., 2021; Swett et al., 2016), Hypoxylon canker of poplar, and bacterial leaf scorch, caused by *Xylella fastidiosa*. For insects, bark beetles are known to increase their populations and related damage during the combination of warm conditions that enhance overwintering success (in some cases, number of generations per year) and periods of increased vulnerability of trees facilitated by drought or other stress (Bentz et al., 2010). Also, changes in weather could affect tree and pathogen phenology differentially to alter disease infection or expression. Disease incidence may be reduced as the tree and pathogen react to different environmental cues, and the timing of pathogen sporulation and infection becomes unlinked to critical stages in host tree development.

3.2 | Predisposing factors and tree genetics

Predisposing site and stand factors, such as soil characteristics: texture, depth and saturation; location factors: elevation and aspect; and stand variables: structure, density, age and species composition, are considered in the climate–disease framework because of their effects on tree vulnerability to disease and microclimate. These factors have not yet been found to be the most significant factors in development of the Type 1 diseases (climate-pathogen) we evaluated; however, thinning treatments can reduce disease severity for Dothistroma (Bulman et al., 2016) and altering stand structure affects the response of Swiss needle cast to short-term directional climate changes (Mildrexler et al., 2019). For Type 2 (climate-stress) diseases, stand conditions are considered very important in the development of pole blight of western white pine, where a particular size-age class is vulnerable, and in yellow-cedar decline, where canopy cover alters microclimate to create conditions conducive to abiotic injury. Soil properties are also critical in the expression of both of these forest declines; soil moisture-holding capacity and limits on rooting depth (for pine and cedar, respectively) distinguished between areas with diseased versus healthy trees. The occurrence of sudden aspen decline was not strongly linked to soil factors despite investigation, but elevation, aspect, slope position, basal area, and tree slenderness were related to damage (Worrall et al., 2008, 2010).

Our approach has been to primarily consider climatic conditions, or climate zones, for the distribution of each tree species to evaluate climate effects on disease, but individual tree characteristics also modify disease development in various ways. Genetic resistance to pathogens plays a role in climate-pathogen diseases (Sniezko & Koch, 2017). Genetic variation in drought responses, for populations or for individual trees (Moran et al., 2017), influences outbreak patterns of climate stress diseases. Natural variation associated with climate of seed source and disease tolerance has been demonstrated using provenance tests, for the Swiss needle cast example (Montwé et al., 2021; Wilhelmi et al., 2017) and for other tree diseases (e.gMcDermott & Robinson, 1989; Morton & Zhu, 1986; Steiner et al., 2019). These data may be coupled with climate change models to identify populations at risk of climate maladaptation and associated disease (Rehfeldt, 2004; Rehfeldt et al., 1999; Rehfeldt et al., 2014; St. Clair & Howe, 2007).

3.3 | Links to climate may be difficult to document for some diseases

Some diseases are challenging to monitor through time, and therefore, it becomes problematic to make any links to climatic conditions. Changes in perennial, chronic diseases of roots and stems (including decays) are particularly difficult to monitor because they are hidden either below ground or within the tree bole. These locations are also somewhat buffered from short-term weather events due to the insulating properties of those niches.

For stem decays, such as those caused by *Phellinus tremulae*, *Porodaedalea pini* and *Ganoderma applanatum*, it is difficult to accurately measure the volume of decayed wood; however, both direct and indirect estimation methods have been developed (Soge et al., 2021). Stem decay rates vary depending on factors that include wood moisture content and temperature (Boddy, 2001), and growth temperature optima have been measured (Humphrey & Siggers, 1933). Assuming that mycelial growth rate is correlated with decay rate in live trees, it is logical to conclude that greater decay volume will develop in many cool temperate and boreal forests if growth temperature optima are met over extended periods of time, provided adequate moisture is also present. Excessive heat could reduce vegetative growth of stem decay fungi in some warmer pathosystems. Carbon dioxide (CO_2) and methane (CH_4) are released to the atmosphere during the decay process (Hietala et al., 2015); therefore, it is predicted that increased stem decay rates will lead to increased rates of emission of these greenhouse gases. The weather triggers for spore production are complex (Kadowaki et al., 2010). Additionally, under changing climatic conditions the rate and distribution of establishment for new infections could also be altered. All of these effects are likely to be related to climate affecting the fungus (Type 1) rather than by initiating tree stress (Type 2).

Likewise, variation in root disease activity, such as caused by *Armillaria* spp., is notoriously difficult to monitor through time. Challenges include attributing death to *Armillaria* species, which may

act as a primary pathogen or as a secondary agent that infects hosts where immunity is impaired by drought, temperature extremes, other pathogens, insect or reductions in site quality (Goheen & Otrosina, 1998; Wargo & Harrington, 1991). These factors contribute to inoculum quantity and quality, which needs to be assessed to account for changes in disease behavior. However, Murray and Leslie (2021) showed that levels of Armillaria root disease intensity are associated with drought events where the disease is present in a plantation in southern British Columbia (i.e., Type 2 climate–disease). Some root diseases such as laminated root rot caused by *Coniferiporia weirii* (formerly *Phellinus weirii*) are not associated with host stress and any climate effects on disease behavior may be related to vegetative growth and sporulation similar to stem decay fungi (i.e., Type 1 climate–disease).

3.4 | Research and monitoring needs

Most of the information that contributed to our understanding of climate-tree disease interactions for the examples described was derived from a combination of monitoring surveys and research. To evaluate climate associations with tree disease expression, widely available weather data can be extrapolated with models at all spatial scales and may be used for back-casting (e.g., Welsh et al., 2009), retrospective studies (e.g., Woods et al., 2005) as well as forecasting using general circulation models (e.g., Desprez-Loustau et al., 2007); Hennon et al., 2016).

Incomplete monitoring records of spatial and temporal information on forest diseases and other biotic disturbance agents continues to represent a critical gap in efforts to better understand climate-induced forest disturbances (McDowell et al., 2015). Disease-specific aerial surveys documented the extent of sudden aspen decline, yellow-cedar decline, and Swiss needle cast, all of which can be identified from the air without the need for labour-intensive ground-checking and laboratory confirmation. When conducted annually, these surveys provide useful data to compare disease response with contemporaneous weather records. Many of the more diffusely distributed diseases (i.e., those without a unique aerial signature), such as pine rusts, some root diseases and hemlock dwarf mistletoe, require ground-based monitoring methods to determine increases or decreases in disease. Data from most ground-based forest inventory plots are often not disease specific and not analysed to determine the cause of damage or mortality.

Research findings are necessary to build hypotheses on climate–disease interactions that can lead to more refined model projections. Without more detailed information derived from focused mechanistic research, we assume that climate affects pathogen reproductive biology but cannot build risk projection models with relevant weather-climate inputs to capture the timing of key events such as sporulation or infection. Physiological investigations brought clarity to the specific mechanistic roles of weather and climate in disease development in the three tree-stress disease examples. Both tree and fungal physiological data either did, or could, help identify specific weather-climate variables needed for more refined disease risk factor models (as discussed further in Section 3.6).

3.5 | Modelling of climate and disease

Various forest ecological modelling approaches, including physiological, biotic interaction, species distribution, ecological niche, bioclimate niche and others, are used to project future forest conditions. All incorporate climate variables as inputs, but each model type has a specific goal—not all are appropriate for forest diseases or particular situations, so they need to be applied cautiously. Relevant to our disease examples, Watt et al. (2009) demonstrated in a global model that climatic stresses that commonly limit the occurrence of Dothistroma needle blight are more likely to constrain the distribution of the host before they affect the fungus. However, in British Columbia, the pathogen has not yet reached the northern range of lodgepole pine, which has wide climatic suitability.

Bioclimatic niche models offer the potential to predict both the future distribution of forest trees and their associated damaging biotic agents. These models are based on assumptions that the distribution of an organism is synchronous with the current climate, and as climate changes, so will the potential distribution of the organism. Using bioclimate niche modelling, significant potential range expansions for both western hemlock and hemlock dwarf mistletoe in Alaska have been forecast (Barrett et al., 2012). However, lags in migration for both the tree and pathogen seem to have created a disconnect between current distributions and current climate, and it is unlikely that either the tree or the pathogen will disperse to keep pace with the predicted expanding suitable climate space. A bioclimatic niche model prediction accurately identified locations where sudden aspen decline occurred (Rehfeldt et al., 2009) but would not have predicted yellow-cedar decline, which occurs in the heart of the yellow-cedar range (Buma et al., 2017; Hennon et al., 2016). Desprez-Loustau et al. (2007) cautioned that the use of the bioclimate models for pathogens like rusts, with their complex life cycles involving multiple spore types with differing responses to climatic variables, could be problematic.

3.6 | Applying climate–disease concepts to forest management through climate–disease risk factor models

For some disease outbreaks tied to weather or climate drivers, forest management strategies may be designed by using the type of information presented in the examples to develop climate–disease risk factor models. A climate–disease risk factor model predicts risk of disease occurrence or severity in a particular location based on disease presence and condition of the forest tree, key climate variables and relevant predisposing factors. Such a model will be only as

robust as the supporting tree species distribution, and additionally for Type 1 diseases, the quality of the pathogen or disease distribution layer.

The steps needed to develop and use a management strategy that incorporates climate influences on tree diseases include recognizing the occurrence of disease increase or decrease, distinguishing between biotic and abiotic causes, considering climate inputs in creating and refining models that predict disease incidence and/or severity, and employing the resulting climate–disease risk models in conservation and management strategies (Table 2).

Climate–disease risk factor models have been partially or fully developed for a few of the disease examples presented. For yellow-cedar decline, the risk factor model required two major factors, the climate variable 'snow' and the predisposing factor 'hydrology impacting rooting depth' (Hennon et al., 2016). The climate variable that causes actual injury to yellow-cedar, short-term freezing events, was not included in the model because these conditions occur anywhere in the broad areas where the disease occurs including hypermaritime environments (Comeau et al., 2019). Two other climate tree-stress diseases, sudden aspen decline and western white pine pole blight were shown to be drought-driven, so drought models essentially serve as disease risk factor models. Stand age and soil moisture-holding capacity were important risk factors that could be added as input variables for the white pine pole blight risk model. For most of our other examples, the necessary components for constructing similar models are known. For example, risk factor models could be constructed to predict future activity for Swiss needle cast (Lee et al., 2017; Manter et al., 2005; Stone et al., 2008) and pine rusts based on the specific weather variables described as essential for those diseases to develop. A climate–disease risk model has been created for Swiss needle cast on Douglas-fir in North America (Manter et al., 2005) and New Zealand (Watt et al., 2010) where neither host nor pathogen is native.

3.7 | Conclusions and implications for conservation and management

Based on the examples presented above, several generalizations for disease management emerged to adapt to changing climate conditions. First, favour the desired tree species through active management in climate zones where that species is well adapted, and the future climate–disease risk is expected to be low. Unfortunately, the magnitude of forecast climatic changes, high levels of uncertainty particularly with precipitation trends, and the increased frequency of extreme events limit the opportunities to apply this approach. A second broad theme is to favour a greater number of alternative tree species in areas of high climate–disease risk. But, the limitations of this approach are illuminated by the sudden aspen decline example: there is no replacement for the aesthetic and biodiversity values of quaking aspen and, similarly, no mitigation for the loss of keystone species in other ecosystems.

Our understanding and ability to manage tree diseases is often limited by a lack of recurring tree disease monitoring data to track annual disease expression with weather records. Focused research is also needed to determine specific temperature and precipitation influences and physiological limits of the host and pathogen. Collaboration between pathologists, physiologists, ecologists, entomologists and others is needed to understand the causes of tree mortality or decline. Greater integration of the impacts of climate-driven tree diseases on forest dynamics is needed to manage and sustain forests under threat from numerous disturbance agents and processes.

To solve problems, one needs to understand their causes. This is true for forest health as it is in human and animal illness (Rizzo et al., 2021). Expanding on our climate–disease framework (Hennon et al., 2020), we used information from previous studies to form and test hypotheses for the strength of climate involvement for seven forest

TABLE 2 Steps and methods to develop a disease risk factor model with climate inputs. The model can be used to create tree conservation and disease management strategies

Step	Method
1. Recognize change in disease incidence	Detection of increased or decreased disease by survey and monitoring
2. Preliminary assessment of biotic or abiotic cause	Evaluate diseased trees to find primary biotic agents or their absence
3. Initial associations of climate and disease	Develop information on spatial and temporal disease expression, compare with weather station data and spatial climate layers
4. Climate controls in disease confirmed	Analyse consistency of biotic or abiotic disease with climate conditions at several spatial and temporal scales; research mechanistic links; develop climate hypothesis consistent with knowledge of tree or pathogen physiology
5. Check for predisposing factors	Observe and measure relationship between relevant site factors or stand characteristics that influence climate effects and disease expression
6. Develop climate–disease risk factor model	Analyse key aspects of climate involvement in disease; develop models with climate inputs to explain past, current and future disease outbreaks
7. Refine and validate model	Continually improve and validate model with new monitoring and research information
8. Conservation and management implications	Use climate–disease risk factor model to develop conservation/management strategy

tree diseases. Using the framework structure, we found key climatic and related factors that control disease development. These variables can be incorporated into climate–disease risk factor models to project favourable and unfavourable locations for forest trees across landscapes under a changing climate. Incorporating these concepts into strategies for forest management can lessen the negative effects of tree diseases and improve adaptation to climate change.

ACKNOWLEDGMENTS

This paper expands on discussions in the Climate Change Committee of the Western International Forest Disease Work Conference. We thank Robert Mathiasen for data on elevational limits of dwarf mistletoes. The comments from two anonymous reviewers are also appreciated. Financial support for this work was partially provided by the USDA Forest Service. The assessment of western white pine pole blight was partially funded by the USDA Forest Service, Forest Health Protection, Special Technology Development Program (R1-FY2020-01).

PEER REVIEW

The peer review history for this article is available at https://publons.com/publon/10.1111/efp.12719.

DATA AVAILABILITY STATEMENT

Data sharing not applicable – no new data generated.

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How to cite this article: Hennon, P. E., Frankel, S. J., Woods, A. J., Worrall, J. J., Ramsfield, T. D., Zambino, P. J., Shaw, D. C., Ritóková, G., Warwell, M. V., Norlander, D., Mulvey, R. L., & Shaw, C. G. III (2021). Applications of a conceptual framework to assess climate controls of forest tree diseases. *Forest Pathology*, 00e1–25. https://doi.org/10.1111/efp.12719