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Review of Literature on Climate Change and Forest Diseases of Western North America

John T. Kliejunas, Brian W. Geils, Jessie Micales Glaeser, Ellen Michaels Goheen, Paul Hennon, Mee-Sook Kim, Harry Kope, Jeff Stone, Rona Sturrock, and Susan J. Frankel

Authors

John T. Kliejunas is a retired regional forest pathologist, U.S. Department of Agriculture, Forest Service, Pacific Southwest Region, 1323 Club Drive, Vallejo, CA 94592. **Brian W. Geils** is a research plant pathologist, U.S. Department of Agriculture, Forest Service, Rocky Mountain Research Station, Southwest Forest Science Complex, 2500 South Pine Knoll Drive, Flagstaff, AZ 86001. **Jessie Micales Glaeser** is a research plant pathologist, U.S. Department of Agriculture, Forest Service, Northern Research Station, One Gifford Pinchot Drive, Madison, WI 53726. **Ellen Michaels Goheen** is a forest pathologist, U.S. Department of Agriculture, Forest Service, Forest Health Protection, Southwest Oregon Forest Insect and Disease Service Center, 2606 Old Stage Road, Central Point, OR 97502. **Paul Hennon** is a research forest pathologist, U.S. Department of Agriculture, Forest Service, Forestry Sciences Laboratory, 2770 Sherwood Lane, Suite 2A, Juneau, AK 99801. **Mee-Sook Kim** is a plant pathologist, U.S. Department of Agriculture, Forest Service, Rocky Mountain Research Station, 1221 South Main Street, Moscow, ID 83843 and Assistant Professor, Department of Forest Resources, Kookmin University, Seoul, Korea 136-702. **Harry Kope** is the provincial forest pathologist, Forest Practices Branch, British Columbia Ministry of Forests and Range, 9th Floor, 727 Fisgard St., Victoria, BC, Canada V8W 1R8. **Jeff Stone** is a research professor, Department of Botany and Plant Pathology, Cordley Hall 2082, Oregon State University, Corvallis, OR 97331. **Rona Sturrock** is a research scientist, forest pathology, Natural Resources Canada, Canadian Forest Service, Pacific Forestry Centre, 506 West Burnside Road, Victoria, BC, Canada V8Z 1M5. **Susan J. Frankel** is a plant pathologist, U.S. Department of Agriculture, Forest Service, Pacific Southwest Research Station, 800 Buchanan Street, West Annex Building, Albany, CA 94710-0011; sfrankel@fs.fed.us.

Abstract

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A summary of the literature on relationships between climate and various types of tree diseases, and the potential effects of climate change on pathogens in western North American forests is provided. Climate change generally will lead to reductions in tree health and will improve conditions for some highly damaging pathogens. Sections on abiotic diseases, declines, canker diseases, root diseases, Phytophthoras, foliar diseases, stem rusts of pine, mistletoes, and wood decays present some examples of potential disease effects with predicted climate change. The effects of climate change on hosts, pathogens, and their interaction will have numerous, mostly adverse, consequences to forest ecosystems.

Keywords: Climate change, forest pathogens, forest tree diseases.

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Paul Hennon

Decline of yellow-cedar on western Chichagof Island in southeast Alaska.

Introduction

> The evidence is clear that one of the features that characterizes the world's climate is instability…
>
> We now come to the present century and find general agreement that during the first 40 or more years the warming trend has not only been continuing but possibly accelerating…
>
> As we explore what these changes in long-term weather mean with respect to forest diseases, we must realize that while mean rises or drops in temperature or moisture are important, the extremes which vary with these means may be more important.
>
> —*George Hepting*, 1963

This literature review is the result of the "Climate Change and Western Forest Diseases" project sponsored by the U.S. Department of Agriculture Forest Service (USDA FS) Western Wildland Environmental Threat Assessment Center (WWETAC) and the Pacific Southwest Research Station (PSW). The project builds on a June 2007 workshop on climate change and forest insects and diseases held by WWETAC (Beukema and others 2007) and a workshop in October 2007 that was sponsored by the USDA FS, WWETAC, and PSW in cooperation with the 2007 Western International Forest Disease Work conference.

The objectives of the Climate Change and Western Forest Diseases project are to:

1. Provide high-quality information on climate change and forest diseases to land managers and forest pathologists.
2. Integrate forest pathology and forest pathologists into climate change policy, management, and science.
3. Identify forest diseases that threaten ecosystems undergoing rapid climate change.
4. Determine needs for research, management, and extension to address future climate change and forest disease issues.

This literature review is intended to inform managers, decisionmakers, scientists, and others about the potential effects of occurring and predicted climate change on forest pathogens and forest pathogen/host interactions.

Background

Climate Change

The world's climate is changing (Houghton and others 1996, IPCC 2007, Wigley 1999). During the past 100 years, average global temperatures have risen by about 0.8 °C. Although Earth's climate has fluctuated for millions of years (Jackson and Overpeck 2000, Russell 1941), the current rate of warming has been unprecedented over the last millenium (IPCC 2007, Mann and others 1999). In the conterminous United States, average temperature has risen by 0.5 °C, and precipitation has increased by 5 to 10 percent (National Assessment Synthesis Team 2000). In addition, the variance in temperature and precipitation is increasing. Further, the frequency and severity of extreme weather effects may be increasing (Hopkin and others 2005).

Causes of climate change include changes in atmospheric concentrations of greenhouse gases and aerosols, and changes in land cover and solar radiation. The concentration of carbon dioxide, the most important anthropogenic greenhouse gas, has risen dramatically since preindustrial times. Models of the carbon cycle project that the concentration of carbon dioxide will increase from 379 parts per million (ppm) in 1995 to 540 to 970 ppm by 2100 (IPCC 2001, 2007). Even if human sources of greenhouse gas emissions were stabilized, warming would continue for centuries owing to time lags associated with climate processes and feedbacks (IPCC 2007).

Average global temperature is projected to increase from 1 to 6 °C from 1990 to 2100. Increases across much of the United States may be even greater (Houghton and others 2001). Increases in winter temperature and more frequent droughts are predicted throughout much of the Western United States under most climate change models (USDA Forest Service 2003). Precipitation may increase slightly in winter months, but the duration of seasonal heat and drought is likely to increase (USDA Forest Service 2003). Projections for the Pacific Northwest (Mote and others 2003), based on output from eight global circulation models, are illustrative. Temperature in the Pacific Northwest is projected to increase from 0.5 to 2.5 °C by 2020 and from 1.5 to 3.2 °C by 2040. Precipitation across the region is projected to increase except during summer. In British Columbia, snowmelt may occur earlier and more rapidly, with a reduction in summer streamflows (Hamann and Wang 2006).

There is consensus that the direct and indirect effects of climate change on forest dynamics may be profound (Aber and others 2001, Ayres and Lombardero 2000, Dale and others 2001). Climate change will affect the geographic distribution of vegetation types, ecosystem processes such as primary production, and the distribution and abundance of individual species of trees and other plants (Malcolm and Pitelka 2000, Malcolm and others 2006). In British Columbia, the projected future climate may result in the disappearance of subboreal and montane climate regions, resulting in the loss of suitable habitat for many ecologically and commercially important species of conifers (Hamann and Wang 2006).

Climate Change and Disturbance in Forest Ecosystems

Forest ecosystems are affected both directly and indirectly by natural disturbances including but not limited to fire, drought, insects and pathogens, hurricanes, windstorms, and ice storms (McNulty and Aber 2001). The distribution, frequency, and severity of these disturbances is affected by changes in climate (Dale and others 2000, Joyce and others 2001, Walther and others 2002). When disturbances exceed a forest's ability to maintain a stable state (that is, its resilience), fundamental changes in forest structure and function are inevitable (Drever and others 2006, Gunderson 2000). Impacts of disturbances and thus of climate change are seen over a broad spectrum of spatial and temporal scales (Peterson 2000). As climate changes, outbreaks of insects and diseases in forests may become more common (McNulty and Aber 2001). Disease reflects interaction among a susceptible host, a virulent pathogen, and environmental conditions that support the pathogen. Therefore, any climatic change that increases host susceptibility to a pathogen or results in an environment more suitable for the pathogen will result in increased incidence of disease.

When disturbances exceed a forest's ability to maintain a stable state (that is, its resilience), fundamental changes in forest structure and function are inevitable.

Some species of trees can evolve rapidly in response to changes in temperature (Jump and others 2006). However, it is unclear how trees will be affected by interactions among changes in climate and disturbances such as fire, insects, and pathogens (Anderson and others 2004). Determinants of forest response to insects and pathogens include the category, host specificity, and aggressiveness of the pathogen and the phytosociology and functional importance and distinctiveness of the host (Ayres and Lombardero 2000, Harrington 2002, Logan and others 2003, Lovett and others 2006).The forested area annually impacted by insects and pathogens in the United States is approximately 45 times the area affected by fire (20 400 000 and 450 000 ha, respectively), with an economic impact almost five times as great (Dale and others 2001, Logan and others 2003, insect and disease data from USDA 1997). The USDA FS (2004) estimated that the area of insect- and disease-caused tree mortality increased from about 1.2 million ha in 1997 to about 4.9 million ha in 2003. Because dead trees serve as fuel for catastrophic wildfires, insects and pathogens often play key roles in the occurrence and severity of the forest's second greatest disturbance agent, fire (Bergeron and Leduc 1998, Hepting and Jemison 1958).

Forests already stressed by high tree density (overstocking), pathogens, or climatic conditions such as drought may not survive additional climatic stress (Winnett 1998). Forests in southern California provide an example. In the early 2000s, a severe drought, the worst in the recorded history of the region, in combination with stress induced by overstocking, dwarf mistletoe (*Arceuthobium* spp.), and annosus root disease (caused by *Heterobasidion annosum*), predisposed conifers to bark beetle attack. The resulting bark beetle epidemic and subsequent tree mortality provided fuel for wildfires that occurred in late October and early November 2003. More than 300 000 ha of brush and timber burned during 1 week. Twenty-six lives and 3,360 homes were lost. The fires caused more than $2.5 billion in damage (Keeley and others 2004) and dramatically altered ecosystem structure and function. Similarly, recent mortality of pinyon pine (*Pinus edulis* Engelm.) across 12 000 km^2 of the Southwestern United States was attributed to the stress of pathogens and drought that left trees susceptible to infestation by bark beetles (Breshears and others 2005).

Effects of Climate Change on Forest Pathogens

Most plant pathogens are strongly influenced by environmental conditions and the vigor of the host. Climate change will directly affect the pathogen, the host, and the interaction between them, resulting in changes in disease impacts (Brasier 2005, Burdon and others 2006). Global climate change ultimately is expressed at the microclimatic scale, the scale at which plant pathogens reproduce, disperse, and infect their hosts (Waggoner 1965). Host condition, thus susceptibility to pathogens, also is affected by cycles and extremes of temperature and moisture (see Ayers and Lombardero 2000, Hopkin and others 2005).

The following sections summarize the literature on relationships between climate and various types of tree diseases, and the potential effects of climate change on pathogens in western North American forests. Rather than summarizing the entire body of literature on the effects of weather (temperature and moisture) on the host, pathogen, and disease, each section presents some examples of potential disease effects with predicted climate change.

Key points:

Although we are uncertain how specific forest pathogens will respond to climate change, existing knowledge allows us to draw some general inferences.

- The distribution of pathogens and diseases, and their influence on the status and trend of forests, will change. Increases in temperature may allow some diseases to expand their latitudinal and elevational ranges. The influence of pathogens on the status and function of forests may change coincident with changes in species composition and climate. If managers facilitate migration of tree species to new locations, we should expect new diseases to affect those trees.
- Climate change will alter the epidemiology of plant diseases. Prediction of disease outbreaks will be more difficult in periods of rapid climate change and unstable weather.
- In a rapidly changing climate, the rate at which pathogens evolve and overcome host resistance may increase.
- Because abiotic factors such as temperature and moisture affect host susceptibility to pathogens and pathogen aggressiveness, changes in interactions between biotic diseases and abiotic stressors may represent the most substantial effect of climate change on plant diseases.
- Climate change may facilitate invasion by new nonnative pathogens. New epidemics may occur as a result.
- Many pathogens currently are limited by winter temperature, and seasonal increases in temperature are expected to be greatest during winter. Accordingly, both overwintering survival of pathogens and disease severity are likely to increase.
- The effects of climate change on individual plant diseases will depend on the ecosystem and climate conditions.

Kim Camilli, California Dept. of Forestry and Fire Protection

Abiotic Diseases

Agents of abiotic forest disease include, but are not limited to, air pollutants; extremes of temperature, precipitation, and water availability; and nutrient deficiencies. Abiotic environmental factors affect plant disease in several ways. First, abiotic diseases can result from direct effects of the environment on the host. Direct effects of environmental extremes (for example, drought, low or high temperatures) are generally negative (Desprez-Loustau and others 2006). An increase in average temperature may accelerate photochemical reaction rates and increase the concentration of water vapor in the lower atmosphere, which likely will increase concentrations of ozone (Sitch and others 2007). Ozone reduces the ability of plants to sequester carbon dioxide and can increase tree susceptibility to root disease (James and others 1980a, 1980b).

Key point:

- Drought stress exacerbates the damage to trees from many pathogens, particularly facultative parasites.

Second, factors such as temperature and moisture affect host susceptibility and pathogen aggressiveness. For example, severity of the foliage disease Swiss needle cast (caused by *Phaeocryptopus gaeumannii*) in western Oregon is strongly correlated with winter temperature, which affects growth of the pathogen (Manter and others 2005). Water stress was associated positively with the severity of effects of *Sphaeropsis sapinea* on pines (Bachi and Peterson 1985; Blodgett and others 1996, 1997; Paoletti and others 2001). Drought in northwestern Alberta in the 1960s and 1980s decreased resistance of aspen (*Populus* spp.) stands to fungal cankers and wood-boring insects (Hogg and others 2002). In the Southwestern United States, widespread mortality of pinyon (*Pinus monophylla* Torr. & Frem.) and juniper (*Juniperus osteosperma* (Torr.) Little and *J. occidentalis* Hook.) is associated with several years of drought; drought, insects, and disease are responsible for local pinyon mortality rates that can approach 100 percent (Shaw and others 2005).

Kim Camilli, California Department of Forestry and Fire Protection

Salt damage on Jeffrey pine, *Pinus jeffreyi*.

Third, abiotic factors also function as predisposing, inciting, or contributing agents in tree declines (Manion 1991). Stress depletes a tree's carbohydrate reserves, increasing its vulnerability to insects and pathogens (Wargo and Haack 1991).

The incidence of abiotic diseases associated with environmental extremes is expected to increase as climate changes, as are diseases caused by interactions between biotic and abiotic agents (Boland and others 2004). Mortality of seedlings and saplings is a likely consequence of severe drought. Mature trees are less vulnerable to water limitation because they have deep roots and substantial reserves of carbohydrates and nutrients. However, severe or prolonged drought may render even mature trees less resistant to insects or disease (Hanson and Weltzin 2000, Joyce and others 2001).

Don Duriscoe

Ozone injury on blue elderberry, causing interveinal necrosis.

Tree mortality in response to drought will become more common as temperature increases. Of particular concern is regional-scale mortality of overstory trees, which rapidly would alter land cover and ecosystem function. These changes would last for decades (Breshears and others 2005, Folke and others 2004).

A variety of pests, pathogens, and parasites can infect trees stressed by climatic extremes (Jurskis 2005). An increased incidence of summer drought would make trees more vulnerable to attack by fungi whose activity is dependent on host stress, particularly root pathogens, wound colonizers, and latent colonizers of sapwood (Broadmeadow 2002, Desprez-Loustau and others 2006, Lonsdale and Gibbs 2002, Redfern and Hendry 2002). Pathogens that are more likely to infect stressed hosts will benefit from the effects of heat and drought on forest and urban trees (Boland and others 2004, Schoeneweiss 1975).

Changes in climate including increased temperature, increased evapotranspiration, and extreme weather events may create unusually high levels of stress in forest stands (Columbia Mountains Institute of Applied Ecology 2005, Sturrock 2007). Forests already stressed by high tree density, pathogens, or atmospheric conditions may not survive the additional climatic stress (Winnett 1998).

James Worrall

Declines

Tree decline is characterized by a slow and progressive deterioration in condition or vigor, decreased growth accompanied by branch dieback on mature trees, and a complex etiology often involving abiotic and biotic factors (Manion 1991, Manion and Lachance 1992). Long-term **predisposing factors** (such as unfavorable site, age, climate, and genetics) decrease the resistance of trees to short-term **inciting factors** (such as frost and defoliation). Inciting factors weaken the trees and, in conjunction with **contributing factors** (such as opportunistic pathogens), ultimately kill the trees.

Key point:
- The incidence and magnitude of forest declines are predicted to increase as local populations of trees are stressed by climate change.

Decline and mortality of western white pine (*Pinus monticola* Dougl. ex D. Don), referred to as pole blight, was observed in the Inland Empire region of the Pacific Northwest in the 1930s and 1940s. The decline appeared to be associated with the predisposing factor of shallow soils that held little moisture and the contributing factors of canker pathogens and root deterioration from Armillaria root rot (Leaphart 1958, Wellington 1954). Climate records indicated that between 1916 and 1940, regional precipitation was lower and temperature higher than during the past 280 years (Leaphart and Stage 1971). Thus, drought was an inciting factor in the pole blight, with shallow soils the predisposing factor and secondary fungi contributing factors.

Phil Kemp

Aspen stand nearly completely dead owing to sudden aspen decline, Mancos-Dolores Ranger District, San Juan National Forest, Colorado.

Mortality of yellow-cedar (*Chamaecyparis nootkatensis* (D. Don) Spach), known as yellow-cedar decline, occurs on about 200 000 ha in southeast Alaska and British Columbia (Hennon and others 2005, Wittwer 2004). Despite much study, no biotic causes could be identified for the decline, which began in the late 19th century (Hamm and others 1988, Hansen and others 1988, Hennon 1990, Hennon and others 1986, Hennon and McWilliams 1999, Shaw and others 1985). The distribution of yellow-cedar decline is associated with low snowpack in winter and spring, which is consistent with the assumption that snow protects yellow-cedar. Seasonal air and soil temperatures consistently were correlated with yellow-cedar decline, but saturated soils, acidic soils, aluminum toxicity, and calcium deficiency were not. The areas with greatest yellow-cedar mortality had temperatures that were higher in the spring and summer, lower in winter, and had greater daily ranges. Snow delays soil warming. Without the snow's protection, yellow-cedar roots were damaged during warm spring days interspersed with overnight frost (Schaberg and others 2007). Thus, the inciting factor of climate change (climate warming) is hypothesized to be the primary cause of yellow-cedar decline (Beier and others 2008; Hennon and others 2006, 2008).

Sudden aspen decline, the premature death of aspen (*Populus* spp.) stands, recently has begun to occur in southern Colorado, Utah, and Nevada. The decline is characterized by severe, rapid branch dieback and crown thinning. Widespread and severe drought from 2000 to 2005 may have caused stress in aspens, particularly in mature stands at lower elevations, reducing the trees' resistance to secondary infections and infestations. The decline is associated with predisposing factors (low elevations, south to west aspects, open stands, and mature trees), inciting factors (hot, dry conditions), and contributing factors (secondary insects and pathogens including Cytospora canker [caused by *Valsa sordida* Nitschke], aspen bark beetle [*Trypophloeus populi* Hopkins, and *Procryphalus mucronatus* LeConte], poplar borer [*Saperda calcarata* Say], and bronze poplar borer [*Agrilus liragus* Barter and Brown]) (Forest Health Management 2008, Worrall and others 2008).

Tom Gordon, University of California-Davis

Canker Diseases

Cankers are diseases that result in death of sections on branches or main trunks of trees. Cankers are usually caused by the infection of bark tissues by plant pathogens. Although numerous canker pathogens are capable of attacking vigorous trees, many canker-causing fungi are more likely to cause damage to trees with heat and drought stress (Schoeneweiss 1975, 1981). Outbreaks or epidemics of these stress-related canker diseases often follow water stress and freezing stress (Schoeneweiss 1981). Most published studies refer to a positive association between drought and disease, or to synergistic effects of drought and disease. Most canker diseases are caused by facultative parasites such as *Botryosphaeria*, *Sphaeropsis*, *Cytospora*, and *Biscognauxia* (Hypoxylon) (Desprez-Loustau and others 2006).

Key point:

- Many stem canker and dieback pathogens are more successful when attacking stressed trees. Water and drought stress, and therefore the incidence of canker and dieback pathogens, will increase as climate changes.

Botryosphaeria dothidea attacks a wide range of host trees, but causes serious damage only to those that are weakened or under environment stress (Ma and others 2001). Drought stress and winter injury have been associated with increased infection and canker expansion of *B. dothidea* (Brown and Hendrix 1981).

Severity of Sphaeropsis shoot blight, caused by the fungus *Sphaeropsis sapinea* (Fr.) Dyko & B. Sutton, consistently has been associated with water stress (Bachi and Peterson 1985; Blodgett and others 1996, 1997; Paoletti and others 2001). Cankers caused by *Septoria musiva* Peck on inoculated water-stressed trees were significantly larger than those on nonstressed trees (Maxwell and others 1997).

The distribution of pitch canker (caused by *Fusarium circinatum* Nirenberg & O’Donnell) is limited by low temperatures. Inoculation trials during winter yielded low infection rates (Inman and others 2008). Increases in minimum winter temperature may increase the range of the pathogen in the Sierra Nevada and elsewhere (Battles and others 2006).

The pathogen *Biscogniauxia mediterranea* (De Not.) Kuntze (*Hypoxylon mediterranea*) historically was widespread in oak woodlands in the Mediterranean, with a northern range limit of southern Tuscany. However, in recent years, the pathogen has been detected on damaged oaks in Slovenia (Jurc and Ogris 2006), and it is likely to continue moving north as climate warms.

Climate change may increase the incidence of many canker diseases. Most canker-causing fungi benefit from heat and drought stress to forest and urban trees (Boland and others 2004, Broadmeadow 2002, Lonsdale and Gibbs 2002, Schoeneweiss 1975). For example, incidence of *Sphaeropsis sapinea* on pines is likely to increase as drought becomes more common. The incidence of some other canker pathogens, such as *Thyronectria austro-americana* (Speg.) Seeler on honeylocust (*Gleditsia triacanthos* L.) and *Cryphonectria cubensis* Bruner on *Eucalyptus* spp., may decrease as climate changes. Incidence of these pathogens is associated with high rainfall, and their hosts display some resistance to drought stress (Despres-Loustau and others 2006).

Tom Gordon, University of California-Davis

Closeup views of a resin-soaked pitch canker lesion caused by *Fusarium circinatum* on Monterey pine, *Pinus radiata*, from a tree in coastal California near Monterey.

Phytosphere Research

Root Diseases

Tree stress is one of the major factors affecting the incidence and spread of root diseases. Several root pathogens, in particular *Heterobasidion annosum* (Fr.) Bref. and *Armillaria* spp., both of which are major pathogens of western forest conifers, are more aggressive when hosts are stressed. Climate change could increase the incidence and spread of root pathogens like these if host trees become stressed. In addition, climate change could alter the relative fitness of various mycorrhizal fungi and other microbes that currently suppress root disease. The protective effects of mycorrhizae may change in response to changes in soil temperature or moisture (Broadmeadow 2002).

Some reports suggest that hot and dry conditions typical of prolonged drought will increase incidence and spread of root diseases in forests. However, it is difficult to predict how diverse root diseases will be affected by different climate scenarios. Although empirical data are limited, a rise in atmospheric carbon dioxide and increased growth of tree roots may result in an increase in severity or frequency of root disease. More extensive root systems would increase the probability of invasion (O'Neill 1994). This increase could be offset by increased plant vigor and disease resistance (Runion and others 1994).

Several species of *Armillaria* cause root disease in forest trees, resulting in mortality or reduced growth (Shaw and Kile 1991). Armillaria root disease caused by *A. ostoyae* (Romag.) Herink is responsible for volume losses of 2 to 3 million m^3/year of timber in the forests of Canada's Pacific Northwest (Morrison and Mallett 1996). Some *Armillaria* species are primary pathogens and attack healthy trees in western forests, whereas other species act as secondary agents, infecting after hosts have experienced a predisposing stress agent such as drought, other diseases, insects, or reductions in local habitat quality. The incidence of Armillaria root disease is likely to increase under warmer or drier conditions (Shaw and Kile 1991, U.S. Office of Technology Assessment 1993). Such increases not only will lead to declines in tree growth but may increase the incidence or severity of insect attacks on stressed trees (Battles and others 2006). In eastern forests, Piercey-Normore and Bérubé (2000) found that stress from insect defoliation predisposed host trees to root infection by *A. ostoyae*, and significant damage from *Armillaria* has been observed in forests that have been stressed by drought (Wargo and Harrington 1991). In the Pacific Northwest, where the mean annual temperature currently is below the optimum (25 °C) for *Armillaria* growth, a warmer climate is likely to result in increased prevalence and rate of spread of root disease (IPCC 2001).

Key point:

- Simultaneous increases in temperature and decreases in precipitation are likely to increase incidence and spread of root diseases in forests. However, the response of root diseases to climate change differs among species and climate scenarios.

Adverse impacts from annosus root disease also are expected to increase as climate changes. The pathogen *H. annosum* generally is more common and damaging on drier sites (see Puddu and others 2003, for example). Nilson and others (1999) suggested that warming will provide more favorable environmental conditions for *H. annosum* to spread, increasing damage to trees and economic loss.

Laminated root rot, caused by *Phellinus weirii* (Murr.) Gilb., commonly kills Douglas-fir (*Pseudotsuga menziesii* (Mirb.) Franco) and several other conifer species (Thies and Sturrock 1995). The distribution and occurrence of this pathogen is closely associated with the host tree species, especially Douglas-fir. If climate change will affect the host distribution and conditions, *Phellinus* spp. can be less or more threatening to the host species depending on other environmental conditions.[1] Forest soils can buffer roots from changes in air temperature, so a few degrees increase in air temperature may not affect the fungus substantially.

Because occurrence and distribution of root rot pathogens and their host species are closely linked, it is essential to predict distribution of host species in response to climate change. A modeling approach is available to explain current and predict future distributions of forest trees using climate variables (Rehfeldt and others 2006). However, the coarse grain of such models precludes prediction of changes in microclimate that may affect both hosts and root pathogens dramatically.

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[1] Thies, W.G. 2008. Personal communication. Research forest pathologist (retired), Pacific Northwest Research Station, Forest Sciences Laboratory, 3200 SW Jefferson Way, Corvallis, OR 97331.

John W. Hanna

Armillaria root disease mortality center in northern Idaho.

John W. Hanna

Armillaria fruiting body.

Phytophthora Species

Numerous species in the genus *Phytophthora* parasitize a wide variety of host plants, including forest trees. Although some *Phytophthora* species are host specific, others are generalists. The success of *Phytophthora* partly reflects its ability to produce sporangia and zoospores rapidly when environmental conditions are favorable, increasing inoculum levels from extremely low to high within a few days or weeks. The effects of most *Phytophthora* species are greatest when temperatures are relatively mild to warm. Moisture—from sources including rainfall, dew deposition, and irrigation—is the main environmental control on population size of the pathogen. The multicyclic nature of *Phytophthora*-caused plant diseases has resulted in severe epidemics in forest and agricultural systems worldwide.

Key point:

- Increases in winter temperatures of 0.5 to 1.5 °C will allow for increases in survival of *Phytophthora cinnamomi* propagules, resulting in increased negative impacts from *P. cinnamomi*.

Phytophthora cinnamomi Rands, a widespread pathogen in tropical and subtropical regions, causes diseases in the root and stem-base of a wide range of broadleaved and coniferous species, and has been well studied in diverse forest ecosystems. The fungus-like organism is transmitted in the soil and requires warm, wet soils to infect roots. The pathogen is currently present but has limited effects in conifer forests of the Pacific Northwest. Although Douglas-fir is susceptible to the pathogen, the winter months are currently too cold for effective zoospore production and infection, and the summer months are currently too dry (Roth and Kuhlman 1966). If, as projected, winters in the Pacific Northwest become

Kerri Frangioso, University of California-Davis

Sudden oak death mortality caused by *Phytophthora ramorum* near Big Sur, California.

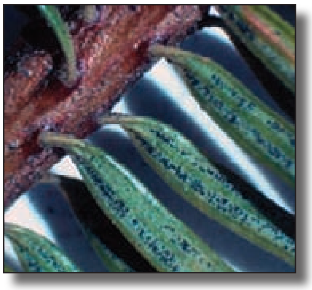
warmer, *P. cinnamomi* may infect Douglas-fir. Epidemic losses of trees owing to *P. cinnamomi* in New Zealand tend to be restricted to years when rainfall is unusually high in autumn and late spring, when soil temperatures are sufficient to support sporulation. Sporulation of *P. cinnamomi* did not occur at typical soil temperatures during the wet winter months in New Zealand (Chee and Newhook 1965).

Not only increases in mean temperature but increases in the duration of relatively warm temperature may increase production of inoculum and northward expansion of *P. cinnamomi* and similar organisms (Brasier and Scott 1994, Hepting 1963). *Phytophthora cinnamomi* is likely to become more prevalent across Europe. Models indicate that activity of the pathogen across the United Kingdom and continental Europe is likely to increase substantially as a result of higher year-round temperatures and northward expansion of the pathogen (Brasier 1996, Brasier and Scott 1994). In Europe, winter temperatures limit survival of the pathogen and its effects on oaks, especially *Quercus robur* L. and *Q. rubra* L.

Bergot and others (2004) compared the geographic ranges of *P. cinnamomi* in France from 1968 to 1998 and projections for 2070 through 2099. Predictions of winter survival of the pathogen were based on temperature in the phloem of infected trees, which in turn was estimated from a regional climate scenario derived from a global circulation model. Projected increases in winter temperatures between the two periods were 0.5 to 5.1 °C. Higher annual rates of *P. cinnamomi* survival were predicted, resulting in a potential range expansion of 100 to several hundred kilometers eastward from its current range along the Atlantic coast. Using the CLIMEX model, Desprez-Loustau and others (2007) estimated that predicted warming would be favorable to most of the pathogens they studied, especially those like *P. cinnamomi* for which minimum winter temperatures limit survival.

Warming may increase habitat quality for *P. cinnamomi* in northern California. Garbelotto and others (2006) reported infections of *P. cinnamomi* among coast live oaks (*Q. agrifolia* Nee.) in southern California (San Diego County). Inoculations in February (average temperature of 19 °C) resulted in small lesions (26 ± 15 mm), whereas September (average temperature of 24 °C) inoculations resulted in larger lesions (135 ± 68 mm), consistent with previous research that suggested colder temperatures are unfavorable to the pathogen.

Increased mortality from other *Phytophthora* species, including *P. ramorum* Werres, De Cock & Man in`t Veld, the cause of sudden oak death, is expected if temperature increases in the summer and precipitation increases in the spring. These changes will lead to increases in inoculum production by the pathogen (Frankel 2007, Rizzo and others 2005). *Phytophthora ramorum* is addressed in more detail in the following section.



Alan Kanaskie, Oregon Department of Forestry

Foliar Diseases

Because they tend to be strongly influenced by weather, pathogens that cause foliar diseases are among the pathogen groups most likely to be affected by changing climate. Sporulation and infection often occur within a narrow temperature range. Spore release usually coincides with periods of precipitation. Spores of foliar pathogens are typically dispersed by wind during wet conditions, and spore germination and infection typically require free moisture on the foliar surface. The severity of these "threshold diseases" (Hepting 1963) usually varies in response to annual weather patterns, and is likely to increase in regions that become warmer and wetter. Conversely, increased incidence of drought likely will decrease the severity of some foliar diseases. Timing of sporulation of many foliar pathogens is synchronous with emergence and development of new tissue on host plants. Changes in climate that favor development of susceptible host foliage during favorable conditions for the pathogen also will increase outbreaks of foliage disease. Elevated concentrations of carbon dioxide increase host growth and pathogen fecundity (Chakraborty and Datta 2003, Chakraborty and others 2000a, Coakley and others 1999); when humidity also increases, the incidence and severity of foliar diseases are likely to increase (Coakley and others 1999, Manning and von Tiedemann 1995).

Key point:

- The majority of foliar pathogens are likely to increase in occurrence and severity as temperature and precipitation increase in late winter and early spring.

Information on interactions between specific foliar diseases and weather is limited, but general inferences can be based on the known dependence of these pathogens on weather. The incidence and severity of most foliar diseases will likely increase if late winter and spring become wetter and warmer, but may decrease if summer becomes drier (Broadmeadow and Ray 2005). The effect of increasing night temperature and, in turn, changes in humidity, will differ among pathogens (Coakley and others 1999, Harvell and others 2002).

Swiss needle cast of Douglas-fir, caused by the native pathogen *Phaeocryptopus gaeumannii* (Rohde) Petrak, occurs throughout the range of the host (Boyce 1940). Although previously considered a minor disease caused by a weak pathogen, a severe epidemic has occurred since the early 1990s in the coastal fog belt of Oregon (Hansen and others 2000, Kanaskie and others 2004). Although the causes of the current outbreak are not known definitively, habitat quality for the pathogen may have been increased by development of dense Douglas-fir plantations in moist, coastal locations where Douglas-fir was not historically dominant. Manter and others (2005) reported that abundance of fruiting bodies and premature needle loss were positively correlated with mean daily temperature in winter and cumulative leaf wetness in spring, and developed a temperature-based model to predict

Alan Kanaskie, Oregon Department of Forestry

Black fruiting bodies of the Swiss needle cast fungus, *Phaeocryptopus gaeumannii*, on the underside of Douglas-fir foliage.

geographic variation in disease severity. Although the authors could not project changes in disease severity based on winter mean daily temperature, they detected a relationship between recent regional climate patterns and the observed increase in the disease. Average temperatures for the period from January to March have increased by approximately 0.2 to 0.4 °C per decade since 1966 in the coastal area of Oregon and Washington. In New Zealand, where Douglas-fir is not native, winter mean temperature was the climate variable most closely correlated with severity of disease caused by *P. gaeumannii*, accounting for about 80 percent of the variation (Stone and others 2007).

Climate change provided ideal temperature and humidity for the recent increased incidence and severity of Dothistroma needle blight (caused by *Dothistroma septosporum* (Dorog) Morelet; *Mycosphaerella pini* Rostr.) on lodgepole pine (*Pinus contorta* var. *latifolia* Dougl. ex Loud.) in Canada (Woods and others 2005). The impact of the native needle pathogen traditionally has been low, rarely causing damage. However, the needle blight is currently causing extensive defoliation and mortality in plantations of lodgepole pine in northwestern British Columbia. These plantations, heavily planted since 1990 and covering close to 40 000 ha in northwest British Columbia, are most severely affected;

entire plantations of lodgepole pine are failing. Even mature pine trees are dying (Woods 2003). Previously, Dothistroma needle blight has caused defoliation and reduced host growth—but not mortality—only in exotic plantations (mainly *Pinus radiata* D. Don) in the Southern Hemisphere (Gibson 1972). In British Columbia, the pathogen is now attacking a native host, and causing mortality even in mature stands, rather than merely retarding growth via defoliation (Bradshaw 2004).

Noting that the current epidemic of Dothistroma needle blight coincided with an increase in frequency of warm rain events during the mid to late 1990s, Woods and others (2005) analyzed climate data for the region. They found a strong positive relationship between increased precipitation and increased disease severity. Their studies demonstrated that a relatively small change in climate can have serious implications for a tree species, particularly if that change passes an environmental threshold that restricted the development of a pathogen.

Drought produces favorable conditions for some foliar pathogens. A reduction in the number of summer rain days may reduce the incidence of Marssonina leaf spot (caused by *Marssonina* spp.) of poplar (*Populus* sp.) (Lonsdale and Gibbs 2002). Wagener (1959) reported increased damage to ponderosa pine (*Pinus ponderosa* Dougl. ex Laws.) from the needle cast pathogen *Davisomycella medusa* (Dearn.) Darker during periods of drought. An unusually wet summer in 1999 may have precipitated an increase in the abundance of the needle cast pathogen *D. ponderosae* (Staley) Dubin. Subsequent drought years allowed that pathogen to develop and kill infected needles of ponderosa pine on the San Juan National Forest in Colorado (Worrall and Sullivan 2002).

The effects of introduced or emerging pathogens may increase as a result of climate change and forest management. *Phytophthora ramorum* recently emerged and is strongly influenced by climate. In addition to causing lethal stem infections in oaks (*Quercus* spp.) and tanoak (*Lithocarpus densiflorus* [Hook. & Arn.] Rehder), *P. ramorum* causes nonlethal foliar infections in a range of other species. Inoculum of *P. ramorum* increases rapidly with heavy rains associated with El Niño events (Davidson and others 2005). Dry conditions are unfavorable. Chlamydospores and zoospores of the pathogen can survive more that 30 days under moist conditions in the laboratory, but survive less than 30 minutes when the relative humidity is less than 30 percent (Davidson and others 2002). Venette and Cohen (2006) used the CLIMEX model to identify areas of the United States suitable for development of *P. ramorum*. Increased precipitation during spring would favor development of the pathogen in areas where it already occurs and expansion of the pathogen to new locations.

Stem Rusts of Pine


David Shaw

Stem rusts of pine (*Cronartium*) are aerially-dispersed, biotrophic, fungal pathogens that have coevolved with their hosts. The complexity of their life cycles differ (Ziller 1974). In western North America, the introduced and invasive *C. ribicola* (J.C. Fischer in Rabenh.) is the best known and most serious blister rust of white pines (*Pinus* subg. *Strobus*) (Mielke 1943). Comandra blister rust (*C. comandrae* Peck) and western gall rust (*Peridermium harknessii* J. P. Moore) are important native pathogens of other pines (subg. *Pinus*). Except for their spores, which are exposed to the atmosphere, these rusts require living hosts. Microcyclic rusts such as *P. harknessii* spread directly from pine to pine; *Cronartium* rusts, however, must alternate between the pine host (pycnial and aecial stages) and an angiosperm host (uredial, telial, and basidial stages). The abundance of these rusts is therefore influenced both by weather and by host populations—each of which is affected by climate change. Severe epidemics of stem rust profoundly affect the genetics and demography of the infested pine (Kinloch 2003). Phylogeo-graphy, biogeography, and ecology of the stem rust pathosystem provide the basis for assessing rust-climate interactions, threats to forest eco-systems, and potential mitigations.

Key points:

- Although rusts can adapt to a wide range of environmental conditions, their tolerances are unknown. Under a changing climate, the incidence of rusts will be determined chiefly by host distribution. Rusts are likely to remain the cause of damaging and lethal diseases.
- Typically, rusts increase in intensity and distribution in “wave years” during which the weather is especially favorable for sporulation, dispersal, and infection. As climate changes, the frequency of such wave years is expected to change.

Spore development, dispersal, and germination of rust fungi are directly affected by abiotic factors (for example, see Chang and Blenis 1989, Powell 1972). The environmental requirements for rust infection are complex and have been especially well-documented for *C. ribicola* (Bega 1960, Mielke 1943, Spaulding 1922, Van Arsdel 1954). Epidemiology and ecophysiology are well studied for *P. harknessii*, *C. comandrae*, and *C. ribicola* (Van Arsdel and others 2006). These pathosystems commonly display a wave year phenomenon when especially favorable weather facilitates substantial increases in rust spread and intensification (Jacobi and others 2002, Peterson 1971). Because stem rusts respond to weather events, monthly average climate variables at the regional or even site level may not be sufficient for predicting rust hazard. Increasing heat and aridity may decrease infection either by inhibiting the rust or defoliating the telial host (Kimmey 1944, Kimmey and Wagener 1961). In some dry locations unfavorable for the repeating stage on the telial host, *C. coleosporioides* J.C. Arthur drops the uredial stage of its life cycle (van der Kamp 1993). Because infection is usually through stomates, whatever affects stomatal opening (for example, carbon dioxide concentration, drought) affects infection.

Climate currently does not appear to limit white pine blister rust in British Columbia (Campbell and Antos 2000). However, climate may restrict the rust elsewhere. After rapid expansion of blister rust from its introduction near Vancouver (Mielke 1943), spread stalled to the east (eastern Montana), southeast (Yellowstone), and south (California). Because hosts occur beyond the range of blister rust, its limits were ascribed to unfavorable climates (Kinloch 2003). However, the rust has since expanded across Montana, into New Mexico, and down the Sierra Nevada (Geils and others 2003, Kliejunas 1985). Colonization of these generally warmer and drier regions could indicate either that the rust is more adaptable, or suitable microsites are more common, than initially thought. For example, the Yellowstone region was described as too cold and dry to support severe infestation of blister rust (Hendrickson 1970). This may be the case on the central Yellowstone plateau, but the effective environmental limit may be separation from critical *Ribes* species rather than a direct microclimatic effect. Severe blister rust outbreaks have occurred on the periphery of the Yellowstone region (for example, Teton Pass; see Kearns and Jacobi 2007), but a credible analysis has not been conducted to examine correlations between climate and rust severity throughout the Yellowstone region. The size of rust populations may be decreased by a series of extreme winter cold fronts such as those that purged ponderosa pine (*Pinus ponderosa* Dougl. ex Laws.) of comandra blister rust at Mink Creek, Idaho (Wagener 1950). Before the cold snap, there were infected trees; after, there were no expanding or sporulating cankers.

Ecological interactions with other species, especially insects, also affect the severity of rust impact. These interactions usually have direct or indirect climate effects and biotic responses (Hiratsuka and others 1987). For example, spermatization of the rust is mediated by pycnia-associated insects (Hunt 1985); factors influencing insect abundance or behavior could affect reproduction of the rust. Likewise, various other insects and fungi are associated with aecial sporulation and host necrosis, but their influence on disease progress is unclear (for example, Furniss and others 1972, Jacobi 1993, van der Kamp and Blenis 1996). Relationships between stem rusts and bark beetles also are also complex and vary in space and time (Campbell and Antos 2000, Six and Adams 2007). Lodgepole pines severely cankered by *C. comandrae* have lower sugar levels, which increases their susceptibility to mountain pine beetle (*Dendroctonus ponderosae* Hopkins) (Nebeker and others 1995). Rust-infected trees may provide high-quality habitat for bark beetles between outbreaks. Host regeneration following a bark beetle outbreak could provide many susceptible new hosts.

David Shaw, Oregon State Universtiy

Aecia appearing on whitebark pine in the Wallowa Mountains, Oregon, in late July 2009.

Hazard models and epidemiological simulation models illustrate how climate, host, and some stem rusts may interact. Elevation, vegetation type, or topographic position are correlated with distribution or incidence of some stem-rust species (for example, Beard and others 1983, Howell and others 2006). Because these factors are assumed to be surrogates for climate, inferences about the distribution of the stem rusts under different climate scenarios are possible. Projections of the location of *Cronartium comandrae* outbreaks and resulting loss of tree volume have been based on site and weather variables (Jacobi and others 1993, Woods 2006). Meteorological records indicated that weather conditions considered suitable for infection of lodgepole pine by *C. comandrae* (more than 6 continuous hours during the months of July, August, and September in which temperature was 10 to 20 °C and the air was nearly saturated) occurred at least every few years within the central Rocky Mountains. The frequency, but not the duration, of potential infection episodes was related to these weather patterns (Jacobi and others 2002). Patterns of upper level airflow conducive to long-distance transport of rust spores from a source to a target, coupled with data on surface conditions favorable for infection at a designated target, were used to identify potential periods of transport and infection by *C. ribicola* spores (Frank and others 2008). Regional warming likely would alter the frequency of potential infection episodes.

Numerous models for predicting outbreaks of white pine blister rust are available (Van Arsdel and others 1961). An epidemiological simulation model has been used to examine interactions among climate and the ecology and genetics of white pine blister rust (McDonald 1996, McDonald and others 1981). Northern Idaho and the southern Sierra Nevada in California have contrasting climates. Northern Idaho had severe rust epidemics as early as the 1920s; the southern Sierra became infested many decades later. Simulations projected outbreaks on the basis of typical Idaho weather and temperature response functions of rusts in that state. If stand development is simulated with meteorological data typical for California and the rust's epidemiological parameters for Idaho, no outbreak occurs. But if the exercise is repeated with the parameters adjusted to represent shift in the rust's response function, the model generates an outbreak (McDonald 1996). This exercise suggests that although the rust, which was adapted to cool, moist conditions in Idaho, initially did not survive under warmer and drier conditions in California, a local strain of the rust evolved that could survive in California. Ultimately, extremes of temperature and humidity restrict the rust, but the rust may be able to adapt to changing climate. The response of stem rusts to climate change can be predicted and managers can allow for surprise by managing for resilience (Drever and others 2006).

Mistletoes


Paul Dunham

The dwarf mistletoe genus *Arceuthobium* and the true mistletoe genus *Phoradendron* occur on various conifer and hardwood species in western North America (Geils and others 2002). *Arceuthobium* spp., parasitic seed plants on various Pinaceae and Cupressaceae, cause reductions in tree growth, seed and cone production, and wood quality, and result in increased tree mortality and susceptibility of the host to attacks from pathogens and insects (Hawksworth and Wiens 1970). In addition, *Arceuthobium* often form brooms that create fuel ladders into tree crowns (Hawksworth and Wiens 1970). *Phoradendron* spp., common on hardwoods and some conifers, are less damaging to their hosts than *Arceuthobium* because they have leaves and produce chlorophyll, but heavy infections can result in significant tree stress (Geils and others 2002).

Mistletoes may play a significant role in tree mortality as trees become stressed by drought and other changes in climate. For example, the interactive effects of *Arceuthobium* and bark beetles are responsible for 40 to 60 percent of the pine mortality in southern California during years of average precipitation. Mortality is more frequent in the presence of other stress factors, such as drought, aerial oxidants, or extremely high density of trees (Schultz and Allison 1982, Schultz and Kliejunas 1982). Wood and others (1979) attributed 90 percent of the mortality of Jeffrey pines (*Pinus jeffreyi* Grev. & Balf.) at Laguna Mountain in southern California to a combination of California flatheaded borer (*Melanophila californica* Van Dyke), western dwarf mistletoe (*A. campylopodum* Engelm.), and annosus root disease. In the central Sierra Nevada, red fir (*Abies magnifica* A. Murr.) are predisposed to fir engraver (*Scolytus ventralis* LeConte) attack by several stress factors, including severe drought, high tree density, annosus root disease, and dwarf mistletoe (Frankel and others 1988). Childs (1960) reported that death of conifer branches in the Pacific Northwest following an unusually hot summer in 1958 and an unusually dry summer in 1959 was particularly widespread in trees stressed by dwarf mistletoe and other agents. He also suggested that the death of branches infected by dwarf mistletoe actually may reduce inoculum and thus reduce spread of the disease.

Key points:

- Both genera of mistletoes common to the Western States can infect healthy host trees. Severe infection can induce stress and predispose hosts to serious damage in combination with other biotic agents and drought.
- Mistletoe reproduction is often limited by cold temperatures, so warming could allow for geographic range expansion.

Harry Kope

Lodgepole pine dwarf mistletoe.

The combined stress from dwarf mistletoe, drought (especially via reduction in soil moisture), and other agents often results in tree mortality. Surveys during the 1976–77 drought in California estimated that 12.3 million trees (equivalent to about 30 000 m^3 of wood) had died. All sites and forest types were affected. Forest insects and pathogens contributed to 98 percent of the lost volume. Of this loss, 65 percent was the result of a combination of dwarf mistletoe and root diseases predisposing the hosts to drought stress (Byler 1978, Craig 1979). More recent surveys in California also indicated that trees infected with dwarf mistletoe were the first to die during drought (Byler 1978). Dwarf mistletoe predisposed many stands to insect attack and has induced 60 to 80 percent of all Jeffrey pine mortality in years of severe drought (Jenkinson 1990). In ponderosa pine in southwest Colorado, the occurrence and severity of effects of dwarf mistletoe were positively correlated with dry sites (Merrill and others 1987). Wilson and Tkacz (1992) reported that severe drought, dense stand conditions, and presence of pinyon dwarf mistletoe (*A. divaricatum* Engelm.) predisposed pinyon pine (*Pinus edulis* Engelm.) in northern Arizona to attack by pinyon ips, *Ips confusus* (LeConte). Gehring and Whitham (1995) found that levels of the true mistletoe *Phoradendron juniperinum* Engelm. ex Gray in junipers (*Juniperus monosperma* (Engelm.) Sarg.) growing in droughty cinder soils were three times those in sandy-loam soils.

Because climate limits the ranges of many dwarf mistletoes, climate change likely will result in extensions in range. Southwestern dwarf mistletoe (*A. vaginatum* (Willd.) J. Presl ssp. *cryptopodum*) does not occur throughout the entire range of ponderosa pine; distinct northern, upper elevational, and lower elevational limits exist (Mark and Hawksworth 1976). Surveys of occurrence, slope association, and severity of several dwarf mistletoe species in Colorado revealed elevational zones specific to each host-parasite interaction (Williams 1971). For example, *A. vaginatum* was present to the upper elevational range of its host (2800 m), but absent below 1860 m. Below 2130 m, ponderosa pine is vigorous; relatively high temperature may increase its resistance to mistletoe. Lodgepole pine dwarf mistletoe (*Arceuthobium americanum* Nutt ex. Engelm.) on lodgepole pine occurs at the lower but not at the upper elevational limits of its host (Hawksworth 1956). The elevational limit for the mistletoe may reflect that the growing season at higher elevations is not long enough for the fruit to mature before severe autumn frosts occur. Although ponderosa pine in the Black Hills is susceptible to dwarf mistletoe infection, the parasite is absent from the Black Hills and Bearlodge Mountains, apparently because early onset of the cold season kills young embryos of an essentially tropical mistletoe (Alexander 1987). Bloomberg (1987) suggested that differences in incidence and severity of hemlock dwarf mistletoe (*A. tsugense* [Rosendahl] G.N. Jones) among geographic areas (low in Alaska, moderate to high in British Columbia, and moderate in Washington and Oregon) reflect the effects of climate on seed production and spread, stand composition and growth rate, and dwarf mistletoe biotypes. Lodgepole pine dwarf mistletoe is a damaging pathogen of jack pine (*Pinus banksiana* Lamb.) in western Canada, but is absent from jack pine forests in the colder, more northern areas, again suggesting limitation by low temperature (Brandt and others 2004).

In California, the range of several *Phoradendron* species is limited by low temperatures. Wagener (1957) reported that occasional low temperatures restrict *P. densum* Torr. on western juniper (*Juniperus occidentalis* Hook.) and *P. pauciflorum* Torr. on white fir (*Abies concolor* (Gord. & Glend.) Lindl.) to their present limits and account for their absence at the northern extent of their hosts' ranges. A warming climate likely would extend the range of these *Phoradendron* species.


Jessie Micales Glaeser

Wood Decays

Wood decay is caused by fungi that either break down holocellulose and lignin (white rots) or holocellulose but not lignin (brown rots). The decay is often described according to its position in the tree (for example, root rot, butt rot, stem rot) (Tainter and Baker 1996). Decay of wood in living trees and of woody debris is heavily dependent on moisture and temperature (Boddy 1983, Griffin 1977, Loman 1965). Some decay fungi are quite tolerant of high temperatures and dry conditions and are often found in the outer layers of slash piles, whereas other fungi require more water and lower temperatures and are restricted to the center or bottom of slash piles (Loman 1962, 1965; Spaulding 1929, 1944).

Key points:

- Decay of wood is dependent on moisture and temperature.
- With climate warming, white rot fungi, which break down carbon compounds as well as lignin, may move northward. This would alter carbon sequestration rates by accelerating carbon breakdown.

Heart-rot fungi are pathogens that colonize the central portion of living trees. Although many heart-rot fungi are host generalists and have a broad geographic range, others are more restricted and colonize specific tree species (Tainter and Baker 1996). Actively growing trees can often limit fungal colonization via active chemical and physical barriers that restrict decay to the central column (Shigo 1984). However, trees under stress, particularly drought stress, may have fewer reserves available for host defense. Low humidity and dry conditions make tree-wound surfaces less favorable for spore germination, possibly resulting in fewer established infections. Certain heart-rot fungi can persist in dry wood for many years, so colonization can resume under more favorable conditions (Wright 1934).

The breakdown of woody debris is primarily caused by numerous species of sap-rot fungi that saprotrophically degrade downed wood, recycling nutrients back into the soil. Many of these fungi are not host specific (Gilbertson and Ryvarden 1986); therefore, there is a wide array of decay fungi available to colonize a specific substrate under a given set of conditions. Wood moisture content is the most important influence on decay and is dependent on humidity, precipitation, and uptake of water from ground contact. Decay fungi cannot degrade wood with moisture content below 30 percent (fiber saturation point; moisture content expressed on a dry-weight basis), and upper moisture limits generally do not exceed 90 percent, owing to lack of oxygen (Griffin 1977). Wood temperature is the second most important influence on fungal activity. Fungi can generally grow when wood temperature exceeds 0 °C, the temperature at which free water in the wood cell wall is available to the fungus. The optimum temperature for growth varies among species; most decay fungi have temperature optima between 20 to 35 °C. For example, *Gloeophyllum sepiarium* (Fr.) Karst and *Phlebia subserialis* (Bourdot & Galzin) Donk., with temperature optima of 38 °C and 31 °C, respectively (Loman 1962), can colonize case-hardened logs and the upper portions of slash piles under conditions of high temperatures and relatively low moisture (Loman 1962, 1965; Spaulding 1929, 1944). *Coniophora puteana* and *Stereum sanguinulentum*, isolated from lower portions of the same slash piles, have temperature optima of 24 °C and 20 to 24 °C, respectively (Loman 1962).

Brown rot fungi are commonly associated with conifers, and white rots with hardwoods (Gilbertson 1980, 1981; Nobles 1958). Hardwoods that are currently limited by freezing temperatures are likely to spread northward and replace conifers as the climate warms (Shafer and others 2001). Expansion of hardwood taxa may increase the overall occurrence of white rot fungi. Because white rots break down carbon compounds as well as lignin, carbon sequestration rates will be altered by expansion of this category of pathogens.

Dead wood can represent a substantial fraction of stored carbon in forest ecosystems, and decay rates of coarse woody debris may be affected by climate change. Kueppers and others (2004) found differences in biomass and rates of decay along an elevational gradient, suggesting that warming will lead to a loss of dead wood carbon from subalpine forest. Models of decomposition of woody debris suggest that an increase in regional warming and drying in the Western United States will increase rates of decay, leaving less debris on the forest floor and increasing release of carbon dioxide, except when limited by extremely xeric conditions (see Yin 1999, for example). Therefore, it appears that a changing climate will substantially influence the development of wood decay, especially in the decomposition of downed wood and slash. The potential effects of climate on individual heart-rot diseases are unknown.


Jessie Micales Glaeser

Fruiting body of the oyster mushroom (*Pleurotus*), a common wood decay organism.

Summary of Climate Change and Forest Diseases

Based on existing knowledge, it is reasonable to infer that climate change generally will lead to reductions in tree health and will improve conditions for some highly damaging pathogens.

Because there is considerable uncertainty about the type and magnitude of climate changes that will occur, the responses of forest ecosystems and forest tree pathogens to climate change are also uncertain (Millar and others 2007). Information on interactions among the environment, forest pathogens, and their hosts is limited. Effects of climate changes on host physiology, adaptation or maladaptation, and population genetics that affect host-pathogen interactions will be altered by climate change in ways essentially unknown.

Despite these uncertainties, the potential effects of climate change on forest tree pathogens can be projected by analyzing current relationships between the behavior of individual pathogens and a suite of abiotic and biotic variables (Lonsdale and Gibbs 1994). The environmental conditions that many individual diseases can exploit have been quantified. Based on existing knowledge, it is reasonable to infer that climate change generally will lead to reductions in tree health and will improve conditions for some highly damaging pathogens. Drought stress will exacerbate the impacts of many pathogens. Increases in winter temperatures will increase the occurrence and severity of pathogens such as *Phytophthora*. Increases in spring precipitation will increase the occurrence and severity of foliar pathogens, whereas increases in autumn precipitation will promote expansion of stem rusts. Predictions are relatively feasible and credible for pathogens whose range or activities primarily are limited by temperature. It is more difficult to predict how climate change may affect pathogens dependent on interactions with other organisms such as insect vectors (Lonsdale and Gibbs 1994). Few data are available to validate predicted interactions among hosts, pathogens, and climate (Runion 2003).

Generalizations about the effects of climate change on forest ecosystems and forest pathogens in western North America are difficult because different hosts and pathosystems in different locations will have distinct responses to climate change (Runion 2003, Sturrock 2007). For example, decreases in precipitation may drive changes in host-pathogen relationships in Arizona, whereas increases in temperature may have the greatest influence on these relationships in Alaska. Tree species differ significantly in their ability to adapt to increases in temperature and atmospheric concentrations of carbon dioxide and their tolerance of disturbances (Lemmen and Warren 2004).

Implications

Various models have been used to predict how climate change will affect forest ecosystems. Some of these models have been used to predict the response of individual pests to climate change (Baker and others 2000, Logan and others 2003). For example, CLIMEX has been used to explore the response to climate change of *P. cinnamomi* (Desprez-Loustau and others 2007) and *P. ramorum* (Venette and Cohen 2006). Unfortunately, as sophisticated as some climate models have become, many do not account for or consider all the important factors involved in the effects of climate change. Current climate model projections do not include pathogen and insect impacts on vegetation, and most models do not incorporate the effects of pathogens as agents of regime change (Folke and others 2004). It is clear that climate change will alter current host-pathogen interactions.

Although the impacts of individual disturbances such as forest pathogens on forest structure and function have been studied, there is little research on the interactions of climate and disturbance (Dale and others 2000). Thus, the extent to which climate change will affect the frequency, severity, or magnitude of disturbances is difficult to predict (Loehle and LeBlanc 1996). Research on impacts of climate change on plant pathogens has been limited, with most work concentrating on the effects of a single disturbance on the tree host, or the interaction of a single disturbance and climate. Disturbances may interact in a cumulative or cascading manner, with increases in one type of disturbance increasing the potential for other types of disturbances. For example, drought in southern California forests increased mortality of trees from insects and root pathogens. The cumulative mortality resulted in extremely high fuel loads that facilitated severe wildfire.

The role of abiotic stressors, pathogens, and their synergistic interactions with the host often are not included in climate change scenarios (Loehle and LeBlanc 1996, Scherm 2004). There is consensus that climate change likely will stress trees and increase their vulnerability to insects, pathogens, and emerging diseases (Brasier 2001, McNulty and Aber 2001).

Maladaptation of hosts and their pathogens resulting from climate change could alter sporulation and rates of development of the pathogen, modify host resistance and phenology, and result in changes in the physiology of host-pathogen interactions. For example, increased carbon dioxide concentrations will affect plant diseases by changing host physiology and anatomy, such as lowered nutrient concentration, greater accumulation of carbohydrates in leaves, more waxes, extra layers of epidermal cells and increased fiber content, and greater number of mesophyll cells (Chakraborty and others 1998). Two important effects of elevated carbon dioxide on host-pathogen interactions will be a delay in initial establishment of a pathogen because of modifications in pathogen aggressiveness and host susceptibility, and increased fecundity of pathogens (Coakley and others 1999). An increase in carbon dioxide may increase tree canopy size and density, resulting in a higher microclimate relative humidity, and a subsequent increase in foliar and rust diseases Manning and Tiedemann 1995).

Additional consequences of climate change are shifts in the geographical distribution of host and pathogen and altered impacts of the pathogen on the host (Coakley and others 1999). For example, a warmer climate may result in extensions of northern range limits for some pathogens currently limited by winter extremes (Loehle and LeBlanc 1996). The extension of the range of the subtropical pathogen *P. cinnamomi* to more northern latitudes is often given as an example. Warmer temperatures at northern latitudes and higher elevations may favor other pathogens and insect vectors as well, resulting in increased damage to hosts and to forest ecosystems. Relatively rapid changes in climate may result in host resistance to pathogens being overcome more rapidly, owing to accelerated pathogen evolution, a result of the shorter regeneration time of fungal pathogens relative to trees. This decrease in host resistance may also result in greater than expected ecosystem damage. Some studies suggest that with regional warming and drying, subalpine forests will be a net source of carbon (Kueppers and Harte 2005). Increases in temperature at northern latitudes may result in greater activity by decay fungi and potentially reduce carbon sequestration in northern forests. The literature on decline diseases, such as pole blight and yellow-cedar decline, provides examples of how a tree species can be affected by a change in climate that affects host physiology and site conditions.

The interactions and synchrony developed over time are crucial components of the sustainable ecosystems existing under current climatic conditions. As the climate changes, these components will change, resulting in changes in host–pathogen relationships. The failure to consider and include detrimental changes such as an increase of pathogen species favored by warmer temperatures or increased number of insect species capable of vectoring pathogens in climate change models where data are available means that those models underestimate, or in some instances overestimate, the effects of climate change on forest ecosystems.

Conclusions

The effects of climate change on hosts, pathogens, and their interaction will have numerous, mostly adverse, consequences to forest ecosystems. Most of these consequences are not currently accounted for in climate change models. Some key points that emerged from this literature review are:

- Climate change will alter the frequency, intensity, duration, and timing of disturbances in forest ecosystems. These disturbances include fire, drought, introduced species, outbreaks of insects and pathogens, hurricanes, windstorms, ice storms, and landslides (Dale and others 2001). The relative role of pathogens and other stressors will differ among ecosystems (Ayres and Lombardero 2000).
- Climate change will affect the geographical distribution of plant diseases, the losses they cause, and the efficacy of disease management strategies.
- Climate change may precipitate irreversible changes in ecosystem state (Folke and others 2004, Pascual and Guichard 2005). Ecosystems may be resilient to individual stresses, but multiple simultaneous stresses could seriously impact status and sustainability of forests (Aber and others 2001), especially forests on marginal sites.
- Risk assessments developed previously for invasive and emergent diseases can provide a framework for organizing and presenting information on interactions between forest disease and climate change (for example, Yang 2006). The information needs of decisionmakers differ, but all assessments should communicate assumptions, potential bias, uncertainty, and appropriate applications. Assessments on forest disease-climate change should include an adequately supported and responsive infrastructure that includes research, stakeholders, and decisionmakers at various agencies and levels (Hain 2006).
- Efforts to model host-pathogen interactions in forest ecosystems would benefit from active networking among multiple disciplines across a gradient of scales (Garrett and others 2006, Lundquist and Hamelin 2005, Proulx and others 2005). Models must consider the synergistic impacts of climate change and other environmental stressors.

- As climate changes, managers and policymakers will need input and guidance from pathologists in the form of viable management alternatives. Pathologists are uniquely trained to understand host-pathogen-environment interactions as integrated systems (Bruck and Shafer 1991). Achieving management objectives for forest ecosystems as climate changes will require multiple approaches, including models, formal assessments, distributed learning networks, and adaptation policies that are responsive to a wide variety of environmental circumstances (Norgaard and Baer 2005, Spittlehouse and Stewart 2003).

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English Equivalents

When you know:	Multiply by:	To find:
Millimeters (mm)	0.0394	Inches
Meters (m)	3.28	Feet
Cubic meters (m^3)	424	Board feet
Square kilometers (km^2)	.386	Square miles
Celsius (°C)	1.8 and add 32	Fahrenheit

References

Aber, J.; Neilson, R.F.; McNulty, S.; Lenihan, J.M.; Bachelet, D.; Drapek, R.J. 2001. Forest processes and global environmental change: predicting the effects of individual and multiple stressors. BioScience. 51: 735–751. http://www.usgcrp.gov/usgcrp/Library/nationalassessment/forests/bioone3.pdf. (July 13, 2009).

Alexander, R.R. 1987. Silvicultural systems, cutting methods, and cultural practices for Black Hills ponderosa pine. Gen Tech. Rep. RM-139. Fort Collins, CO: U.S. Department of Agriculture, Forest Service, Rocky Mountain Forest and Range Experiment Station. 32 p.

Anderson, P.K.; Cunningham, A.A.; Patel, N.G.; Morales, F.J.; Epstein, P.R.; Daszak, P. 2004. Emerging infectious diseases of plants: pathogen, pollution, climate change and agrotechnology drivers. Trends in Ecology and Evolution. 19(10): 535–544.

Ayres, M.P.; Lombardero, M.J. 2000. Assessing the consequences of global change for forest disturbance from herbivores and pathogens. Science of Total Environment. 262: 263–286. http://www.dartmouth.edu/~mpayres/pubs/gepidem.PDF. (July 14, 2009).

Bachi, P.R.; Peterson, J.L. 1985. Enhancement of *Sphaeropsis sapinea* stem invasion of pines by water deficits. Plant Disease. 69: 798–799.

Baker, R.S.; Jarvis, C.H.; Cannon, R.J.C.; MacLeod, A.; Walters, F.K.A. 2000. The role of climatic mapping in predicting the potential geographical distribution of non-indigenous pests under current and future climates. Agriculture, Ecosystems and Environment. 82: 57–71.

Battles, J.J.; Robards, T.; Das, A.; Waring, K.; Gilless, J.K.; Schurr, F.; LeBlanc, J.; Biging, G.; Simon, C. 2006. Climate change impact on forest resources. A report from: California Climate Change Center. CEC-500-2005-193-SF. 45 p. http://www.energy.ca.gov/2005publications/CEC-500-2005-193/CEC-500-2005-193-SF.PDF. (July 13, 2009).

Beard, T.H.; Martin, N.E.; Adams, D.L. 1983. Effects of habitat type and elevation on occurrence of stalactiform blister rust in stands of lodgepole pine. Plant Disease. 67(6): 648–651.

Bega, R.V. 1960. The effect of environment on germination of sporidia of *Cronartium ribicola*. Phytopathology. 50: 61–68.

Beier, C.M.; Sink, S.E.; Hennon, P.E.; D'Amore, D.V.; Juday, G.P. 2008. Twentieth-century warming and the dendroclimatology of declining yellow-cedar forests in southeastern Alaska. Canadian Journal of Forest Research. 38: 1319–1334.

Bergeron, Y.; Leduc, A. 1998. Relationships between change in fire frequency and mortality due to spruce budworm outbreaks in the southeastern Canadian boreal forest. Journal of Vegetation Science. 9: 493–500.

Bergot, M.; Cloppet, E.; Pérarnaud, V.; Déqué, M.; Marçais, B.; Desprez-Loustau, M.L. 2004. Simulation of potential range expansion of oak disease caused by *Phytophthora cinnamomi* under climate change. Global Change Biology. 10: 1539–1552.

Beukema, S.J.; Robinson, D.C.E.; Greig, L.A. 2007. Forests, insects & pathogens and climate change: workshop report. Prepared for The Western Wildlands Environmental Threat Assessment Center, Prineville, Oregon. 20 p. + appendices. http://www.essa.com/documents/Forests,%20Pests%20and%20Climate%20-%20Workshop%20Report.pdf. (July 15, 2009).

Blodgett, J.T.; Kruger, E.L.; Stanosz, G.R. 1996. Effects of moderate water stress on disease development by *Sphaeropsis sapinea* on red pine. Phytopathology. 87: 422–428. http://apsnet.org/phyto/PDFS/1997/0221-01R.pdf. (July 13, 2009).

Blodgett, J.T.; Kruger, E.L.; Stanosz, G.R. 1997. *Sphaeropsis sapinea* and water stress in a red pine plantation in central Wisconsin. Phytopathology. 87: 429–434. http://apsjournals.apsnet.org/doi/abs/10.1094/PHYTO.1997.87.4.429. (July 13, 2009).

Bloomberg, W.J. 1987. Comparison of dwarf mistletoe effects on hemlock in Alaska, British Columbia and the Pacific Northwest. In: 34th western international forest disease work conference. [Place of publication unknown]: [Publisher unknown]: 35–40.

Boddy, L. 1983. Effect of temperature and water potential on growth rate of wood-rotting basidiomycetes. Transactions of the British Mycological Society. 80: 141–149.

Boland, G.J.; Melzer, M.S.; Hopkin, A.; Higgins, V.; Nassuth, A. 2004. Climate change and plant diseases in Ontario. Canadian Journal of Plant Pathology. 26: 335–350.

Boyce, J.S. 1940. A needle-cast of Douglas-fir associated with *Adelopus gaeumannii*. Phytopathology. 30: 649–659.

Bradshaw, R.E. 2004. *Dothistroma* (red-band) needle blight of pines and the dothistromin toxin: a review. Forest Pathology. 34: 163–185.

Brandt, J.P.; Hiratsuka, Y.; Pluth, D.J. 2004. Extreme cold temperatures and survival of overwintering and germinated *Arceuthobium americanum* seeds. Canadian Journal of Forest Research. 34: 174–183.

Brasier, C.M. 1996. *Phytophthora cinnamomi* and oak decline in southern Europe: environmental constraints including climate change. Annales Des Sciences Forestieres. 53: 347–358.

Brasier, C.M. 2005. Climate change and tree health. Proceedings, trees in a changing climate conference. http://www.rhs.org.uk/NR/rdonlyres/A9B45B3B-5DED-4251-B57E-6886487E8720/0/tiaccBrasierSum.pdf. (September 15, 2009).

Brasier, C.M. 2001. Rapid evolution of introduced plant pathogens via interspecific hybridization. BioScience. 51(2): 123–133.

Brasier, C.M.; Scott, J.K. 1994. European oak declines and global warming: a theoretical assessment with special reference to the activity of *Phytophthora cinnamomi*. Bulletin-OEPP. 24(1): 221–232.

Breshears, D.D.; Cobb, N.S.; Rich, P.M.; Price, K.P.; Allen, C.D.; Balice, R.G.; Romme, W.H.; Kastens, J.H.; Floyd, M.L.; Belnap, J.; Anderson, J.J.; Myers, O.B.; Meyer, C.W. 2005. Regional vegetation die-off in response to global-change type drought. Proceedings of the National Academy of Sciences of the United States of America. 102: 15144–15148.

Broadmeadow, M.S.J., ed. 2002. Climate change: impacts on UK forests. UK Forestry Commission Bull. 125. Edinburgh, United Kingdom: Forestry Commission. http://www.forestresearch.gov.uk/fr/INFD-5ZYHMC. (July 13, 2009).

Broadmeadow, M.; Ray, D. 2005. Climate change and British woodland. Information Note 69. Edinburgh, United Kingdom: Forestry Commission. http://www.forestresearch.gov.uk/pdf/fcin069.pdf/$FILE/fcin069.pdf. (July 13, 2009).

Brown, E.A., II; Hendrix, F.F. 1981. Pathogenicity and histopathology of *Botryosphaeria dothidea* on apple stems. Phytopathology. 71: 375–379.

Bruck, R.I.; Shafer, S.R. 1991. A role for plant pathologists in global climate change. Plant Disease. 75: 437.

Burdon, J.J.; Thrall, P.H.; Ericson, L. 2006. The current and future dynamics of disease in plant communities. Annual Review of Phytopathology. 44: 19–39.

Byler, J.W. 1978. The pest damage inventory in California. In: Scharpf, R.F.; Parmeter, J.R., Jr., tech. coords. Proceedings of the symposium on dwarf mistletoe control through forest management. Gen. Tech. Rep. PSW-031. Berkeley, CA: U.S. Department of Agriculture, Forest Service, Pacific Southwest Research Station: 162–171. http://www.fs.fed.us/psw/publications/documents/psw_gtr031/psw_gtr031_05_byler.pdf. (July 13, 2009).

Campbell, E.M.; Antos, J.A. 2000. Distribution and severity of white pine blister rust and mountain pine beetle on whitebark pine in British Columbia. Canadian Journal of Forest Research. 30: 1051–1059.

Chakraborty, S.; Datta, S. 2003. How will plant pathogens adapt to host plant resistance at elevated CO_2 under a changing climate? New Phytologist. 159: 733–742.

Chakraborty, S.; Murray, G.M.; Magarey, P.A.; Yonow, T.; O'Brien, R.G.; Croft, B.J.; Barbetti, M.J.; Sivasithamparam, K.; Old, K.M.; Dudzinski, M.J.; Sutherst, R.W.; Penrose, L.J.; Archer, C.; Emmett, R.W. 1998. Potential impact of climate change on plant diseases of economic significance to Australia. Australasian Plant Pathology. 27: 15–35.

Chakraborty, S.; Pangga, I.B.; Lupton, J.; Hart, L.; Room, P.M.; Yates, D. 2000a. Production and dispersal of *Colletotrichum gloeosporioides* spores on *Stylosanthes scabra* under elevated CO_2. Environmental Pollution. 108: 381–387.

Chakraborty, S.; Tiedemann, A.V.; Teng, P.S. 2000b. Climate change: potential impact on plant diseases. Environmental Pollution. 108: 317–326.

Chang, K.F.; Blenis, P.V. 1989. Survival of *Endocronartium harknessii* teliospores in a simulated airborne state. Canadian Journal of Botany. 67: 928–932.

Chee, K.H.; Newhook, F.J. 1965. Variability in *Phytophthora cinnamomi* Rands. New Zealand Journal of Agricultural Research. 8: 96–103.

Childs, T.W. 1960. Drought effects on conifers in the Pacific Northwest, 1958–1959. Res. Note PNW–182. Portland, OR: U.S. Department of Agriculture, Forest Service, Pacific Northwest Research Station. 5 p.

Coakley, S.M.; Scherm, H.; Chakraborty, S. 1999. Climate change and plant disease management. Annual Review of Phytopathology. 37: 399–426.

Columbia Mountains Institute of Applied Ecology. 2005. Appendix two: case study for a managed forest under climate change—Tembec's Tree Farm Licence No. 14. Implications of climate change in BC's southern interior forests, Revelstoke, BC: 115–121.

Craig, G.A. 1979. Integrated forest pest management as seen by a forest land owner. In: Executive summaries; proceedings 1978 western forestry conference. [Place of publication unknown]: Western Forestry Conservation Association: 61.

Dale, V.H.; Joyce, L.A.; McNulty, S.; Neilson, R.P. 2000. The interplay between climate change, forests, and disturbance. The Science of the Total Environment. 262: 201–204.

Dale, V.H.; Joyce, L.A.; McNulty, S.; Neilson, R.P.; Ayres, M.P.; Flannigan, M.D.; Hanson, P.J.; Irland, L.C.; Lugo, A.E.; Peterson, C.J.; Simberloff, D.; Swanson, F.J.; Stocks, B.J.; Wotton, B.M. 2001. Climate change and forest disturbances. BioScience. 51: 723–734. http://www.usgcrp.gov/usgcrp/Library/nationalassessment/forests/bioone2.pdf. (July 13, 2009).

Davidson, J.M.; Rizzo, D.M.; Garbelotto, M.; Tjosvold, S.; Slaughter, G.W. 2002. *Phytophthora ramorum* and sudden oak death in California: II. Pathogen transmission and survival. In: Staniford, R.; McCreary, D.; Purcell, K.L., tech. cords. Fifth symposium on oak woodlands: oaks in California's changing landscapes. Gen. Tech. Rep. PSW-GTR-184. Albany CA: U.S. Department of Agriculture, Forest Service, Pacific Southwest Research Station: 741–749.

Davidson, J.M.; Wickland, A.C.; Patterson, H.A.; Falk, K.R.; Rizzo, D.M. 2005. Transmission of *Phytophthora ramorum* in mixed-evergreen forest in California. Phytopathology. 95: 587–596.

Desprez-Loustau, M-L.; Marcais, B.; Nageleisen, L.M.; Ouiym, D.; Vannini, A. 2006. Interactive effects of drought and pathogens in forest trees. Annals of Forest Science. 63(6): 597. http://www.afs-journal.org/index.php?option=article&access=standard&Itemid=129&url=/articles/forest/pdf/2006/06/f6061.pdf. (July 13, 2009).

Desprez-Loustau, M-L.; Robin, C.; Reynaud, G.; Deque, M.; Badeau, V.; Piou, D.; Husson, C.; Marcais, B. 2007. Simulating the effects of a climate-change scenario on the geographical range and activity of forest pathogenic fungi. Canadian Journal of Plant Pathology. 29: 101–120.

Drever, C.R.; Peterson, G.; Messier, C.; Bereron, Y.; Flannigan, M. 2006. Can forest management based on natural disturbances maintain ecological resilience? Canadian Journal of Forest Research. 36: 2285–2299.

Folke, C.; Carpenter, S.; Walker, B.; Scheffer, M.; Elmqvist, T.; Gunderson, L.; Holling, C.S. 2004. Regime shifts, resilience, and biodiversity in ecosystem management. Annual Review of Ecology Evolution and Systematics. 35: 557–581.

Forest Health Management. 2008. Sudden aspen decline in Colorado. Lakewood, CO: U.S. Department of Agriculture, Forest Service, Forest Health Management, Rocky Mountain Region. 4 p. http://www.aspensite.org/SAD/sad_faqs.pdf. (July 13, 2009).

Frank, K.L.; Geils, B.W.; Kalkstein, L.S.; Thistle, H.W., Jr. 2008. Synoptic climatology of the long-distance dispersal of white pine blister rust. II. Combination of surface and upper-level conditions. International Journal of Biometeorology. DOI: 10.1007/s00484-008-0157-3.

Frankel, S.J. 2007. Climate change's influence on sudden oak death, PACLIM 2007. Monterey, CA. http://www.fs.fed.us/psw/cirmount/meetings/paclim/pdf/frankel_talk_PACLIM2007.pdf. (July 13, 2009).

Frankel, S.; Denitto, G.; Hart, D. 1988. An evaluation of tree mortality in the Sayles summer home tract, Placerville Ranger District, Eldorado National Forest. For. Pest Mgmt. Report 88-19. San Francisco, CA: U.S. Department of Agriculture, Forest Service, Pacific Southwest Region. [Pages unknown].

Furniss, M.M.; Hungerford, R.D.; Wicker, E.F. 1972. Insects and mites associated with western white pine blister rust cankers in Idaho. Canadian Entomologist. 104: 1713–1715.

Garbelotto, M.; Hüberli, D.; Shaw, D. 2006. First report on an infestation of *Phytophthora cinnamomi* in natural oak woodlands of California and its differential impact on two native oak species. Plant Disease. 90: 685. http://nature.berkeley.edu/garbelotto/downloads/garbelotto2006.pdf. (July 13, 2009).

Garrett, K.A.; Dendy, S.P.; Frank, E.E.; Rouse, M.N.; Travers, S.E. 2006. Climate change effects on plant disease: genomes to ecosystems. Annual Review of Phytopathology. 44: 489–509.

Gehring, C.; Whitham, T. 1995. Environmental stress influences above ground pest attack and mycorrhizal mutualism in pinyon-juniper woodlands: implications for management in the event of global warming. In: Shaw, D.W.; Aldon, E.F.; Sapio, C.L., eds. Desired future conditions for pinyon-juniper ecosystems. Gen. Tech. Rep. RM-258. Fort Collins, CO: U.S. Department of Agriculture, Forest Service, Rocky Mountain Forest and Range Experiment Station: 30–37.

Geils, B.W.; Conklin, D.; Frank, K.; Guyon, J.; Harris, J.L.; Hoffman, J.; Jacobi, W.; Kearns, H.; Newcomb, M.; Smith, E.; Van Arsdel, E.; Vogler, D. 2003. New information on the distribution of white pine blister rust for 2002. In: Stone, J.; Maffei, H., comps. Proceedings of the 50th western international forest disease work conference. Bend, OR: U.S. Department of Agriculture, Forest Service, Central Oregon Service Center: 94–99.

Geils, B.W.; Cibrian-Tovar, J.; Moody, B., tech. coords. 2002. Mistletoes of North American conifers. Gen. Tech. Rep. RMRS-GTR-98. Ogden, UT: U.S. Department of Agriculture, Forest Service, Rocky Mountain Research Station. 123 p.

Gibson, I. 1972. *Dothistroma* blight of *Pinus radiata*. Annual Review of Phytopathology. 10: 51–72.

Gilberston, R.L. 1980. Wood-rotting fungi of North America. Mycologia. 72: 1–49.

Gilbertson, R.L. 1981. North American wood-rotting fungi that cause brown rots. Mycotaxon. 12(2): 372–416.

Gilbertson, R.L.; Ryvarden, L. 1986. North American polypores, Vols. 1 and 2. Oslo, Norway: Fungiflora. 885 p.

Griffin, D.M. 1977. Water potential and wood-decay fungi. Annual Review of Phytopathology. 15: 319–329.

Gunderson, L.H. 2000. Ecological resilience—in theory and application. Annual Review of Ecology and Evolution. S31: 425–439.

Hain, F. 2006. New threats to forest health require quick and comprehensive research response. Journal of Forestry. 104(4): 182–186.

Hamann, A.; Wang, T. 2006. Potential effects of climate change on ecosystem and tree species distribution in British Columbia. Ecology. 87: 2773–2786.

Hamm, P.B.; Hansen, E.M.; Hennon, P.E.; Shaw, C.G., III. 1988. *Pythium* species from forest and muskeg areas of southeast Alaska. Transactions of the British Mycological Society. 91: 385–388.

Hansen, A.J.; Neilson, R.P.; Dale, V.H.; Flather, C.H.; Iverson, L.R.; Currie, D.J.; Shafer, S.; Cook, R.; Bartlein, P.J. 2001. Global change in forests: responses of species, communities and biomes. BioScience. 51: 765–779. http://www.usgcrp.gov/usgcrp/Library/nationalassessment/forests/bioone5.pdf. (July 13, 2009).

Hansen, E.M.; Hamm, P.B.; Hennon, P.E.; Shaw, C.G., III. 1988. *Phytophthora drechsleri* in remote areas of southeast Alaska. Transactions of the British Mycological Society. 91: 379–388.

Hansen, E.M.; Stone, J.K.; Capitano, B.R.; Rosso, P.; Sutton, W.; Winton, L.; Kanaskie, A.; McWilliams, M.G. 2000. Incidence and impact of Swiss needle cast in forest plantations of Douglas-fir in coastal Oregon. Plant Disease. 84: 773–778.

Hanson, P.J.; Weltzin, J.F. 2000. Drought disturbance from climate change: response of United States forests. The Science of the Total Environment. 262: 205–220. http://web.ornl.gov/info/ornlreview/v40_3_07/documents/article17web_Hanson_Weltzin_2000.pdf. (July 13, 2009).

Harrington, R. 2002. Insect pests and global environmental change. In: Douglas, I. ed. Encyclopedia of global climate change, Vol. 3: Causes and consequences of global climate change. Chichester, United Kingdom: John Wiley. 381–386.

Harvell, C.D.; Mitchell, C.E.; Ward, J.R.; Altizer, S.; Dobson, A.P.; Ostfeld, R.S.; Samuel, M.D. 2002. Climate warming and disease risks for terrestrial and marine biota. Science. 296: 2158–2162.

Hawksworth, F.G. 1956. Upper altitudinal limits of lodgepole pine dwarf mistletoe in the central Rocky Mountains. Phytopathology. 46: 561–562.

Hawksworth, F.G.; Wiens, D. 1970. Biology and taxonomy of the dwarf mistletoes. Annual Review of Phytopathology. 8: 187–208.

Hendrickson, W.H. 1970. Assessing the potential of white pine blister rust to limber pine and whitebark pine in Yellowstone National Park. Final report RSP YELL-N_40. [In-Service Research Paper]. Washington, DC: Office of Natural Science Studies. 96 p. + appendices.

Hennon, P.E. 1990. Fungi on *Chamaecyparis nootkatensis*. Mycologia. 82: 59–66.

Hennon, P.E.; D'Amore, D.; Wittwer, D.; Caouette, J. 2008. Yellow-cedar decline: conserving a climate-sensitive tree species as Alaska warms. In: Deal, R., ed. Integrated restoration of forested ecosystems to achieve multi-resource benefits: proceedings of the 2007 national silviculture workshop. Gen. Tech. Rep. PNW-GTR-733. Portland, OR: U.S. Department of Agriculture, Forest Service, Pacific Northwest Research Station: 233–245.

Hennon, P.E.; D'Amore, D.; Wittwer, D.; Johnson, A.; Schaberg, P.; Hawley, G.; Beier, C.; Sink, S.; Juday, G. 2006. Climate warming, reduced snow, and freezing injury could explain the demise of yellow-cedar in southeastern Alaska, USA. World Resource Review. 18: 427–450. http://www.fs.fed.us/r10/spf/fhp/cedar/pubs/WRR_Hennon_etal_b.pdf. (July 13, 2009).

Hennon, P.E.; D'Amore, D.; Zeglen, S.; Grainger, M. 2005. Yellow-cedar decline in the North Coast District of British Columbia. Res. Note RN-549. Portland, OR: U.S. Department of Agriculture, Forest Service, Pacific Northwest Research Station. 16 p.

Hennon, P.E.; McWilliams, M.G. 1999. Decline symptoms do not develop with grafting from dying yellow-cedar. Canadian Journal of Forestry Research. 29: 1985–1988.

Hennon, P.E.; Newcomb, G.B.; Shaw, C.G., III; Hansen, E.M. 1986. Nematodes associated with dying *Chamaecyparis nootkatensis* in southeastern Alaska. Plant Disease. 70: 352.

Hepting, G.H. 1963. Climate and forest diseases. Annual Review of Phytopathology. 1: 31–50.

Hepting, G.H.; Jemison, G.M. 1958. Forest protection. In: Timber resources for America's future. Forest Resource Report 14. Washington, DC: U.S. Department of Agriculture, Forest Service: 184–220.

Hiratsuka, Y.; Blenis, P.; Chang, K.-F. 1987. The role of biotic and climatic factors in the epidemiology of western gall rust. Agriculture and Forestry Bulletin 10. Edmonton, AB: University of Alberta: 11–13.

Hogg, E.H.; Brandt, J.P.; Kochtubajda, B. 2002. Growth and dieback of aspen forests in northwestern Alberta, Canada, in relation to climate and insects. Canadian Journal of Forest Research. 32(5): 823–832.

Hopkin, A.; Fleming, R.A.; Candau, J.N.; McKenney, D. 2005. Exploring the vulnerability of eastern Canadian forests to shifting ranges of tree diseases due to climatic change. In: Adapting to climate change in Canada 2005: understanding risks and building capacity conference. http://adaptation2005.ca/abstracts/hopkin_e.html. (July 13, 2009).

Houghton, J.T.; Ding, Y.; Griggs, D.J.; Noguer, M.; van der Linden, P.J.; Xiaosu, D., eds. 2001. Climate changes 2001: the scientific basis. Cambridge, United Kingdom: Cambridge University Press.

Houghton, J.T.; Meira Filho, L.G.; Callander, B.A.; Harris, N.; Kattenberg, A.; Maskell, K. 1996. Climate change 1995. The science of climate change. Cambridge, United Kingdom: Cambridge University Press.

Howell, B.; Burns, K.; Kearns, H. 2006. Biological evaluation of a model for predicting presence of white pine blister rust in Colorado based on climatic variables and susceptible white pine distribution. Biological Evaluation R2-06-04. Lakewood, CO: U.S. Department of Agriculture, Forest Service, Rocky Mountain Region. 15 p.

Hunt, R.S. 1985. Experimental evidence of heterothallism in *Cronartium ribicola*. Canadian Journal of Forest Botany. 63: 1086–1088.

Inman, A.R.; Kirkpatrick, S.C.; Gordon, T.R.; Shaw, D.V. 2008. Limiting effects of low temperature on growth and spore germination in *Gibberella circinata*, the cause of pitch canker in pine species. Plant Disease. 92: 542–545.

Intergovernmental Panel on Climate Change [IPCC]. 2001. Chapter 5: Ecosystems and their goods and services/ 5.6.2.2.2. Pressures from diseases and insect herbivory. http://www.grida.no/climate/IPCC_tar/wg2/pdf/wg2TARchap5.pdf. (July 14, 2009).

Intergovernmental Panel on Climate Change [IPCC]. 2007. Summary for policymakers. In: Solomon, S.; Qin, D.; Manning, M.; Chen, Z.; Marquis, M.; Averyt, K.B.; Tignor, M.; Miller, H.L., eds. Climate change 2007: The physical science basis. Contribution of working group I to the fourth assessment report of the Intergovernmental Panel on Climate Change. Cambridge, United Kingdom and New York: Cambridge University Press. http://www.ipcc.ch/pdf/assessment-report/ar4/syr/ar4_syr_spm.pdf. (July 13, 2009).

Jackson, S.T.; Overpeck, J.T. 2000. Responses of plant populations and communities to environment changes of the late Quaternary. Paleobiology. 26(4):194–220. http://links.jstor.org/sici?sici=0094-8373%28200023%2926%3A4%3C194%3AROPPAC%3E2.0.CO%3B2-L. (July 13, 2009).

Jacobi, W.R. 1993. Insects emerging from lodgepole pine infected by comandra blister rust. Res. Note RM-519. Fort Collins, CO: U.S. Department of Agriculture, Forest Service, Rocky Mountain Forest and Range Experiment Station. 3 p.

Jacobi, W.R.; Geils, B.W.; Taylor, J.E. 2002. Frequency of comandra blister rust infection episodes on lodgepole pine. Res. Pap. RMRS-RP-36. Fort Collins, CO: U.S. Department of Agriculture, Forest Service, Rocky Mountain Research Station. 13 p.

Jacobi, W.R.; Geils, B.W.; Taylor, J.E.; Zentz, W.R. 1993. Predicting the incidence of comandra blister rust on lodgepole pine: site, stand, and alternate-host influences. Phytopathology. 83: 630–637.

James, R.L.; Cobb, F.W., Jr.; Miller, P.R.; Parmeter, J.R., Jr. 1980a. Effects of oxidant air pollution on susceptibility of pine roots to *Fomes annosus*. Phytopathology. 70: 560–563.

James, R.L.; Cobb, F.W., Jr.; Wilcox, W.W.; Rowney, D.L. 1980b. Effects of photochemical oxidant injury of ponderosa and Jeffrey pines on susceptibility of sapwood and freshly cut stumps to *Fomes annosus*. Phytopathology. 70: 704–708.

Jenkinson, J.L. 1990. *Pinus jeffreyi* Grev. and Balf. Jeffrey pine. In: Silvics of North America. Vol. 2 Conifers. Agric. Handb. 654. Washington, DC: U.S. Department of Agriculture, Forest Service: 359–369.

Joyce, L.; Aber, J.; McNulty, S.; Dale, V.; Hansen, A.; Irland, L.; Neilson, R.; Skog, K. 2001. Potential consequences of climate variability and change for the forests of the United States. In: National Assessment Synthesis Team, eds. Climate change impacts on the United States: the potential consequences of climate variability and change. Cambridge, United Kingdom: Cambridge University Press: 489–522. http://www.srs.fs.usda.gov/pubs/ja/ja_joyce001.pdf. (July 13, 2009).

Jump, A.S.; Hunt, J.M.; Martinez-Izquierdo, J.A.; Penuelas, J. 2006. Natural selection and climate change: temperature-linked spatial and temporal trends in gene frequency in *Fagus sylvatica*. Molecular Ecology. 15: 3466–3480.

Jurc, D.; Ogris, N. 2006. First reported outbreak of charcoal disease caused by *Biscogniauxia mediterranea* on Turkey oak in Slovenia. Plant Pathology (Oxford, United Kingdom). 55: 299.

Jurskis, V. 2005. Eucalypt decline in Australia, and a general concept of tree decline and dieback. Forest Ecology and Management. 215(1–3): 1–20.

Kanaskie, A.; McWilliams, M.; Sprengel, K.; Overhulser, D. 2004. Swiss needle cast aerial surveys, 1996 to 2004. In: Mainwaring, D., ed. Swiss needle cast cooperative annual report 2004. Corvallis, OR: College of Forestry, Oregon State University: 7–12.

Kearns, H.S.J.; Jacobi, W.R. 2007. The distribution and incidence of white pine blister rust in central and southwestern Wyoming and northern Colorado. Canadian Journal of Forest Research. 37: 462–472.

Keeley, J.E.; Fotheringham, C.J.; Moritz, M.A. 2004. Lessons from the October 2003 wildfires in southern California. Journal of Forestry. 102: 26–31. http://nature.berkeley.edu/moritzlab/docs/Keeley_etal_2004.pdf. (July 13, 2009).

Kimmey, J.W. 1944. The seasonal development and the defoliating effect of *Cronartium ribicola* on naturally infected *Ribes roezli* and *R. nevadense*. Phytopathology. 35(6): 406–416.

Kimmey, J.W.; Wagener, W.W. 1961. Spread of white pine blister rust from Ribes to sugar pine in California and Oregon. Tech. Bull. 1251. Washington, DC: U.S. Department of Agriculture, Forest Service. 71 p.

Kinloch, B.B., Jr. 2003. White pine blister rust in North America: past and prognosis. Phytopathology. 93: 1044–1047. http://apsjournals.apsnet.org/doi/pdf/10.1094/PHYTO.2003.93.8.1044?cookieSet=1. (July 13, 2009).

Kliejunas, J. 1985. Spread and intensification of white pine blister rust in the southern Sierra Nevada. Phytopathology. 75: 1367.

Kueppers, L.M.; Harte, J. 2005. Subalpine forest carbon cycling: short- and long-term influence of climate and species. Ecological Applications. 15(6): 1984–1999.

Kueppers, L.M.; Southon, J.; Baer, P.; Harte, J. 2004. Dead wood biomass and turnover time, measured by radiocarbon, along a subalpine elevation gradient. Oecologia. 141: 641–651.

Leaphart, C.D. 1958. Pole blight–how it may influence western white pine management in light of current knowledge. Journal of Forestry. 56: 746–751.

Leaphart, C.D.; Stage, A.R. 1971. Climate: a factor in the origin of the pole blight disease of *Pinus monticola* Dougl. Ecology. 52: 229–239.

Lemmen, D.S.; Warren, F.J., eds. 2004. Climate change impacts and adaptation: a Canadian perspective. Ottawa, Ontario: Natural Resources Canada. 174 p.

Loehle, C.; LeBlanc, D. 1996. Model-based assessments of climate change effects on forests: a critical review. Ecological Modeling. 90: 1–31.

Logan, J.A.; Régnière, J.; Powell, J.A. 2003. Assessing the impacts of global warming on forest pest dynamics. Frontiers in Ecology and the Environment. 1: 130–137. http://www.usu.edu/beetle/documents/Loganet.al.2003.pdf. (July 13, 2009).

Loman, A.A. 1962. The influence of temperature on the location and development of decay fungi in lodgepole pine logging slash. Canadian Journal of Botany. 40: 1545–1559.

Loman, A.A. 1965. The lethal effect of periodic high temperatures on certain lodgepole pine slash decaying basidiomycetes. Canadian Journal of Botany. 43: 334–338.

Lonsdale, D.; Gibbs, J. 2002. Effect of climate change on fungal diseases of trees. In: Broadmeadow, M.S.J., ed. Climate change: impacts on UK forests. Bulletin No. 125. Edinburgh, United Kingdom, UK Forestry Commission: 83–97.

Lonsdale, D.; Gibbs, J.N. 1994. Effects of climate change in fungal disease of trees. In: Frankland, J.C.; Magan, N.; Gadd, G.M., eds. Fungi and environment change: symposium of the British Mycological Society. Cambridge, United Kingdom: Cambridge University Press: 1–19.

Lovett, G.M.; Canham, C.D.; Arthur, M.A.; Weathers, K.C.; Fitzhugh, R.D. 2006. Forest ecosystem responses to exotic pests and pathogens in eastern North America. BioScience. 56(5): 395–403.

Lundquist, J.E.; Hamelin, R.C., eds. 2005. Forest pathology: from genes to landscapes. St. Paul, MN: American Phytopathological Society, APS Press. 175 p.

Ma, Z.; Morgan, D.P.; Michailides, T.J. 2001. Effects of water stress on *Botryosphaeria* blight of pistachio caused by *Botryosphaeria dothidea*. Plant Disease. 85: 745–749.

Malcolm, J.R.; Liu, C.; Neilson, R.P.; Hansen, L.; Hannah, L. 2006. Global warming and extinctions of endemic species from biodiversity hotspots. Conservation Biology. 20: 538–548.

Malcolm, J.R.; Pitelka, L.F. 2000. Ecosystems and global climate change: a review of potential impacts on U.S. terrestrial ecosystems and biodiversity. 47 p. http://www.pewclimate.org/docUploads/env_ecosystems.pdf. (July 13, 2009).

Manion, P.D. 1991. Tree disease concepts. 2nd ed. Englewood Cliffs, NJ: Prentice-Hall. 402 p.

Manion, P.D.; Lachance, D.L. 1992. Forest decline concepts: an overview. In: Manion, P.D.; Lachance, D.L., eds. Forest decline concepts. St. Paul, MN: APS Press: 181–190.

Mann, M.E.; Bradley, R.S.; Hughes, M.K. 1999. Northern hemisphere temperatures during the past millennium: inferences, uncertainties, and limitations. Geophysical Research Letters. 26(6): 759–762.

Manning, W.J.; von Tiedemann, A. 1995. Climate change: potential effects of increased atmospheric carbon dioxide (CO_2), ozone (O_3), and ultraviolet-B (UV-B) radiation on plant diseases. Environmental Pollution. 88: 219–245.

Manter, D.K.; Reeser, P.W.; Stone, J.K. 2005. A climate-based model for predicting geographic variation in Swiss needle cast severity in the Oregon Coast Range. Phytopathology. 95: 1256–1265. http://www.cof.orst.edu/coops/sncc/pdfs/pubs/manter%20et%20al%202005.pdf. (July 13, 2009).

Mark, W.R.; Hawksworth, F.G. 1976. Distribution of ponderosa pine dwarf mistletoe (*Arceuthobium vaginatum* subsp. *cryptopodum*) in relation to climatic factors. In: 4th national conference on fire and forest meteorology. [Place of publication unknown]: Society of American Foresters and American Meteorological Society. [Pages unknown].

Maxwell, D.L.; Kruger, E.L.; Stanosz, G.R. 1997. Effects of water stress on colonization of poplar stems and excised leaf discs by *Septoria musiva*. Phytopathology. 87: 381–388. http://www.apsnet.org/phyto/PDFS/1997/0220-01R.pdf. (July 13, 2009).

McDonald, G.I. 1996. Ecotypes of blister rust and management of sugar pine in California. In: Kinloch, B.B., Jr.; Marosy, M.; Huddleston, M.E., eds. Sugar pine: status, values, and roles in ecosystems. Proceedings of a symposium presented by the California Sugar Pine Management Committee. Publication 3362, Davis CA: University of California, Division of Agriculture and Natural Resources: 137–147.

McDonald, G.I.; Hoff, R.J.; Wykoff, W.R. 1981. Computer simulation of white pine blister rust epidemics. I. Model formulation. Res. Pap. INT-258. Ogden, UT: U.S. Department of Agriculture, Forest Service, Intermountain Forest and Range Experiment Station. 136 p.

McNulty, S.G.; Aber, J.D. 2001. United States national climate change assessment on forest ecosystems: an introduction. BioScience. 51: 720–723. http://www.usgcrp.gov/usgcrp/Library/nationalassessment/forests/bioone1.pdf. (July 13, 2009).

Merrill, L.M.; Hawksworth, F.G.; Jacobi, W.R. 1987. Frequency and severity of ponderosa pine dwarf mistletoe in relation to habitat type and topography in Colorado. Plant Disease. 71: 342–344.

Mielke, J.L. 1943. White pine blister rust in western North America. Bulletin 52. New Haven, CT: Yale University, School of Forestry. 155 p.

Millar, C.I.; Stephenson, N.L.; Stephens, S.L. 2007. Climate change and forest of the future: managing in the faces of uncertainty. Ecological Applications. 17(8): 2145–2151.

Morrison, D.J.; Mallet, K.I. 1996. Silvicultural management of Armillaria root disease in western Canadian forests. Canadian Journal of Plant Pathology. 18: 194–199.

Mote, P.W.; Parson, E.A.; Hamlet, A.F.; Keeton, W.S.; Lettenmaier, D.; Mantua, N.; Miles, E.L.; Peterson, D.W.; Peterson, D.L.; Slaughter, R.; Snover, A.K. 2003. Preparing for climatic change: the water, salmon, and forests of the Pacific Northwest. Climatic Change. 61: 45–88.

National Assessment Synthesis Team. 2000. Climate change impacts on the United States: the potential consequences of climate variability and change. Washington, DC: U.S. Global Change Research Program. 541 p.

Nebeker, T.E.; Schmitz, R.F.; Tisdale, R.A.; Hobson, K.R. 1995. Chemical and nutritional status of dwarf mistletoe, Armillaria root rot, and Comandra blister rust infected trees which may influence tree susceptibility to bark beetle attack. Canadian Journal of Botany. 73(3): 360–369.

Nilson, A.; Kiviste, A.; Korjus, H.; Mihkelson, S.; Etverk, I.; Oja, T. 1999. Impact of recent and future climate change on Estonian forestry and adaptation tools. Climate Research. 12: 205–214.

Nobles, M.K. 1958. Cultural characteristics as a guide to the taxonomy and phylogeny of the Polyporaceace. Canadian Journal of Botany. 36: 883–926.

Norgaard, R.B.; Baer, P. 2005. Collectively seeing climate change: the limits of formal models. BioScience. 55(11): 961–966.

O'Neill, E.G. 1994. Responses of soil biota to elevated atmospheric carbon dioxide. Plant and Soil. 165: 55–65.

Paoletti, E.; Danti, R.; Strati, S. 2001. Pre- and post-inoculation water stress affects *Sphaeropsis sapinea* canker length in *Pinus halepensis*. Forest Pathology. 31: 209–218.

Pascual, M.; Guichard, F. 2005. Criticality and disturbance in spatial ecological systems. Trends in Ecology and Evolution. 20(2): 89–95.

Peterson, G.D. 2000. Scaling ecological dynamics: self-organization, hierarchical structure, and ecological resilience. Climatic Change. 44: 291–309.

Peterson, R.S. 1971. Wave years of infection by western gall rust on pine. Plant Disease Reporter. 55: 163–167.

Piercey-Normore, M.D.; Bérubé, J.A. 2000. Artificial inoculation with *Armillaria ostoyae* in established conifers stressed by defoliation, planting, and thinning in Newfoundland. Canadian Journal of Forestry Research. 30: 1758–1765.

Powell, J.M. 1972. Seasonal and diurnal periodicity in the release of Cronartium comandrae aeciospores from stem cankers on lodgepole pine. Canadian Journal of Forest Research. 2: 78–88.

Proulx, S.R.; Promislow, D.E.L.; Phillips, P.C. 2005. Network thinking in ecology and evolution. Trends in Ecology and Evolution. 20(5): 345–353.

Puddu, A.; Luisi, N.; Capretti, P.; Santini, A. 2003. Environmental factors related to damage by *Heterobasidion abietinum* in *Abies alba* forests in southern Italy. Forest Ecology and Management. 180: 37–44.

Redfern, D.; Hendry, S. 2002. Climate change and damage to trees caused by extremes of temperature. In: Broadmeadow, M.S.J., ed. Climate change and UK forests. Forestry Commission Bulletin No. 125. Edinburgh, United Kingdom: Forestry Commission. [Pages unknown].

Rehfeldt, G.E.; Crookston, N.L.; Warwell, M.V.; Evans, J.S. 2006. Empirical analyses of plant-climate relationships for the Western United States. International Journal of Plant Sciences. (167)6: 1123–1150.

Rizzo, D.M.; Garbelotto, M.; Hansen, E. 2005. *Phytophthora ramorum*: integrative research and management of an emerging pathogen in California and Oregon forests. Annual Review of Phytopathology. 43: 309–335. http://nature.berkeley.edu/garbelotto/downloads/rizzo2005.pdf. (July 13, 2009).

Roth, L.F.; Kuhlman, E.G. 1966. *Phytophthora cinnamomi*, an unlikely threat to Douglas-fir forestry. Forest Science. 12: 147–159.

Runion, G.B. 2003. Climate change and plant pathosystems—future disease prevention starts here. New Phytologist. 159: 531–538.

Runion, G.B.; Curl, E.A.; Rogers, H.H.; Backman, P.A.; Rodriguez-K´abana, R.; Helms, B.E. 1994. Effects of free-air CO_2 enrichment on microbial populations in the rhizosphere and phyllosphere of cotton. Agriculture and Forest Metereology. 70: 117–130.

Russell, R.J. 1941. Climatic change through the ages. In: U.S. Department of Agriculture, ed. Climate and man. Yearbook of agriculture. Washington, DC: U.S. Government Printing Office: 7–9, 67–97.

Schaberg, P.G.; Hennon, P.E.; D'Amore, D.V.; Hawley, G.J. 2007. Influence of simulated snow cover on the cold tolerance and freezing injury of yellow-cedar seedlings. Global Change Biology. 14: 1282–1293.

Scherm, H. 2004. Climate change: Can we predict the impacts on plant pathology and pest management? Canadian Journal of Pathology. 26: 267–273.

Schoeneweiss, D.F. 1975. Predisposition, stress, and plant disease. Annual Review of Phytopathology. 13: 193–211.

Schoeneweiss, D.F. 1981. Infectious diseases of trees associated with water and freezing stress. Journal of Arboriculture. 7: 13–18. http://joa.isa-arbor.com/request.asp?JournalID=1&ArticleID=1703&Type=2. (July 13, 2009).

Schultz, D.; Allison, J. 1982. Tree mortality in the Cleveland National Forest. For. Pest Mgmt. Rep. 82-12. San Francisco, CA: U.S. Department of Agriculture, Forest Service, Pacific Southwest Region. 17 p.

Schultz, D.; Kliejunas, J. 1982. A biological evaluation of pine mortality of the San Jacinto Ranger District, San Bernardino National Forest. For. Pest Mgmt. Rep. 82-5. San Francisco, CA: U.S. Department of Agriculture, Forest Service, Pacific Southwest Region. 10 p.

Shafer, S.L.; Bartlein, P.J.; Thompson, R.S. 2001. Potential changes in the distributions of western North America tree and shrub taxa under future climate scenarios. Ecosystems. 4: 200–215.

Shaw, C.G., III; Eglitis, A.; Laurent, T.H.; Hennon, P.E. 1985. Decline and mortality of Chamaecyparis nootkatensis in southeastern Alaska, a problem of long duration but unknown cause. Plant Disease. 69: 13–17.

Shaw, C.G., III; Kile, G.A. 1991. Armillaria root disease. Agric. Handb. 691. Washington, DC: U.S. Department of Agriculture, Forest Service. 233 p.

Shaw, J.D.; Steed, B.E.; DeBlander, L.T. 2005. Forest Inventory and Analysis (FIA) annual inventory answers the question: What is happening to pinyon-juniper woodlands? Journal of Forestry. 103: 280–285.

Shigo, A.L. 1984. Compartmentalization: a conceptual framework for understanding how trees grow and defend themselves. Annual Review of Phytopathology. 22: 189–214.

Sitch, S.; Cox, P.M.; Collins, W.J.; Huntingford, C. 2007. Indirect radiative forcing of climate change through ozone effects on the land-carbon sink. Nature. 448: 791–794.

Six, D.L.; Adams, J. 2007. White pine blister rust severity and selection of individual whitebark pine by the mountain pine beetle (Coleoptera: Curculionidae, Scolytinae). Journal of Entomological Science. 42(3): 345–352.

Spaulding, P. 1922. Investigations of the white-pine blister rust. Bull. 957. Washington, DC: U.S. Department of Agriculture, Forest Service. 100 p.

Spaulding, P. 1929. Decay of slash of northern white pine in southern New England. Tech. Bull. No. 132. Washington, DC: U.S. Department of Agriculture: 1–20.

Spaulding, P. 1944. Decay of logging slash in the Northeast. Tech. Bull. No. 876. Washington, DC: U.S. Department of Agriculture: 1–21.

Spittlehouse, D.L.; Stewart, R.B. 2003. Adaptation to climate change in forest management. BC Journal of Ecosystems and Management. 4(1): 1–11. http://www.forrex.org/publications/jem/ISS21/vol4_no1_art1.pdf. (July 13, 2009).

Stone, J.K.; Hood, I.A.; Watt, M.S.; Kerrigan, J.L. 2007. Distribution of Swiss needle cast in New Zealand in relation to winter temperature. Australasian Plant Pathology. 36(5): 445-454.

Sturrock, R.N. 2007. Climate change effects on forest diseases: an overview. In: Jackson, M.B., comp. Proceedings of the 54th annual western international forest disease work conference. [Place of publication unknown]: [Publisher unknown]: 51–55.

Tainter, F.H.; Baker, F.A. 1996. Principles of forest pathology. New York: John Wiley & Sons, Inc. 805 p.

Thies, W.G.; Sturrock, R.N. 1995. Laminated root rot in western North America. Gen. Tech. Rep. PNW-GTR-349. Portland, OR: U.S. Department of Agriculture, Forest Service, Pacific Northwest Research Station. 32 p.

U.S. Department of Agriculture, Forest Service [USDA Forest Service]. 1997. Forest insect and disease conditions in the United States 1996. Washington, DC: Forest Health Protection. [Pages unknown].

U.S. Forest Service [USDA Forest Service]. 2003. Climate models predict wetter winters, warmer summers in the West. Science Daily. (November 14, 2008). http://www.sciencedaily.com/releases/2003/11/031114072917.htm. (July 13, 2009).

U.S. Department of Agriculture, Forest Service [USDA Forest Service]. 2004. Forest insect and disease conditions in the United States, 2003. Washington, D.C.: Forest Health Protection. 156 p.

U.S. Office of Technology Assessment. 1993. A primer on climate change and natural resources. Preparing for an uncertain climate. Washington, DC: Government Printing Office. [Pages unknown].

Van Arsdel, E.P. 1954. Climatic factors affecting the distribution of the white pine blister rust in Wisconsin. Madison, WI: University of Wisconsin. 105 p. Ph.D dissertation.

Van Arsdel, E.P.; Geils, B.W.; Zambino, P.J. 2006. Epidemiology for hazard rating of white pine blister rust. In: Guyon, J., comp. 2006. Proceedings of the 53rd western international forest disease work conference. Ogden, UT: U.S. Department of Agriculture, Forest Service, Intermountain Region: 49–64.

Van Arsdel, E.P.; Riker, A.J.; Kouba, T.F.; Suomi, V.E.; Bryson, R.A. 1961. The climatic distribution of blister rust on white pine in Wisconsin. Station Paper 87. St. Paul, MN: U.S. Department of Agriculture, Forest Service, Lake States Experiment Station. 34 p.

van der Kamp, B.J. 1993. Production of uredinia and telia by stalactiform blister rust in British Columbia. Canadian Journal of Botany. 71(3): 519–521.

van der Kamp, B.J.; Blenis, P.V. 1996. A model of hyperparasite regulation of the gall rust-lodgepole pine pathosystem. Canadian Journal of Forest Research. 26(7): 1256–1265.

Venette, R.C.; Cohen, S.D. 2006. Potential climatic suitability for establishment of *Phytophthora ramorum* within the contiguous United States. Forest Ecology and Management. 231: 18–26.

Wagener, W.W. 1950. Severe cold reduces rust in pine plantations. Plant Disease Reporter. 34: 193.

Wagener, W.W. 1957. The limitation of two leafy mistletoes of the genus *Phoradendron* by low temperatures. Ecology. 38: 142–145.

Wagener, W.W. 1959. The effect of a western needle fungus (*Hypodermella medusa* Dearn.) on pines and its significance in forest management. Journal of Forestry. 57: 561–564.

Waggoner, P.E. 1965. Microclimate and plant disease. Annual Review of Phytopathology. 3: 103–126.

Walther, G.R.; Post, E.; Convey, P.; Menzel, A.; Parmesan, C.; Beebee, T.J.C.; Fromentin, J-M.; Hoegh-Guldberg, O.; Bairlein, F. 2002. Ecological responses to recent climate change. Nature. 416: 389–395.

Wargo, P.M.; Haack, R.A. 1991. Understanding the physiology of dieback and diseases and its management implications for oak. In: Laursen, S.B.; DeBoe, J.F., eds. Proceedings, Oak resource in the upper midwest, implications for management. St. Paul, MN: University of Minnesota Extension Service: 147–158.

Wargo, P.M.; Harrington, T.C. 1991. Host stress and susceptibility to Armillaria. Agric.Handb. 691.In: Shaw, C.G., III; Kile, G., eds. Armillaria root disease. Washington, DC: U.S. Department of Agriculture: 88–101.

Wellington, W.G. 1954. Pole blight and climate. Ottawa, Ontario: Canadian Department of Agriculture, Forest Biology Division, Bi-monthly Progress Report. 10: 2–4.

Wigley, T.M.L. 1999. The science of climate change: global and U.S. perspectives. Arlington, VA: Pew Center on Global Climate Change. http://www.pewclimate.org/docUploads/env_science.pdf. (July 13, 2009).

Williams, W.T. 1971. Distribution of three species of dwarf mistletoe on their principal pine hosts in the Colorado Rocky Mountain Front Range. Phytopathology. 61: 1324–1325.

Wilson, J.L.; Tkacz, B.M. 1992. Pinyon *Ips* outbreak in pinyon juniper woodlands in northern Arizona: a case study. Gen. Tech. Rep. RM-218. Fort Collins, CO: U.S. Department of Agriculture, Forest Service, Rocky Mountain Forest and Range Experiment Station: 187–190.

Winnett, S.M. 1998. Potential effects of climate change on U.S. forests: a review. Climate Research. 11: 39–49. http://www.int-res.com/articles/cr/11/c011p039.pdf. (July 13, 2009).

Wittwer, D. comp. 2004. Forest health conditions in Alaska, 2004. Protection Rep. R10-PR-3. Anchorage, AK: U.S. Department of Agriculture, Forest Service, Alaska Region. 96 p.

Wood, R.E.; Shuft, M.J.; Schultz, D.E. 1979. An evaluation of tree mortality in Laguna Mountain Recreation Area, Cleveland National Forest. Forest Insect and Disease Mgmt. Report 79-l. San Francisco, CA: U.S. Department of Agriculture, Forest Service, Pacific Southwest Region. 22 p.

Woods, A.J. 2003. Species diversity and forest health in northwest British Columbia. Forestry Chronicle. 79: 892–897.

Woods, A.J. 2006. Predicted impacts of hard pine stem rusts on lodgepole pine dominated stands in central British Columbia: Were our assumptions valid? In: Guyon, J.C., comp. Proceedings of the 53rd western international forest disease work conference. Ogden, UT: U.S. Department of Agriculture, Forest Service, Intermountain Region: 160.

Woods, A.J.; Coates, K. D.; Hamann, A. 2005. Is an unprecedented *Dothistroma* needle blight epidemic related to climate change? BioScience. 55: 761–769.

Worrall, J.; Sullivan, K. 2002. Discoloration of ponderosa pine on the San Juan National Forest, 1999–2001. Biological Evaluation R2-02-06. Gunnison, CO: U.S. Department of Agriculture, Forest Service, Forest Health Management, Rocky Mountain Region: 1–20. http://www.fs.fed.us/r2/fhm/reports/be_r2-02-06.pdf. (July 13, 2009).

Worrall, J.J.; Egeland, L.; Eager, T.; Mask, R.A.; Johnson, E.W.; Kemp, P.A.; Shepperd, W.D. 2008. Rapid mortality of *Populus tremuloides* in southwestern Colorado, USA. Forest Ecology and Management. 255: 686–696.

Wright, E. 1934. Survival of heart-rots in down timber in California. Journal of Forestry. 32: 752–753.

Yang, X.B. 2006. Framework development in plant disease risk assessment and its application. European Journal of Plant Pathology. 115(1): 25–34.

Yin, X. 1999. The decay of forest woody debris: numerical modeling and implications based on some 300 data cases from North America. Oecologia. 121: 81–98.

Ziller, W.G. 1974. The tree rusts of western Canada. Publication 1329. Victoria, BC: Department of the Environment, Canadian Forestry Service. 272 p.

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