Climate and Forest Diseases of Western North America: A Literature Review

by

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“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
Introduction

This literature review is the result of a “Climate Change and Western Forest Diseases” project sponsored by the United States Department of Agriculture Forest Service (USDA FS), Western Wildland Environmental Threat Assessment Center (WWETAC) and the Pacific Southwest Research Station (PSW). The project is a follow-up to a June 2007 workshop on Climate Change and Forest Insects and Diseases held by WWETAC, which catalyzed an effort to assemble relevant information on western forest diseases and climate relationships. The report of that workshop is available online (Beukema and others 2007).

The objectives of the Climate Change and Western Forest Diseases project are to:
1. Provide useable information on climate 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 disease threats to ecosystems undergoing rapid climate change.
4. Determine needs for research, management and extension to address future climate change and forest disease issues.

The project focus is weather, climate, and western North American forest diseases. Information gathered is intended for predicting and understanding how climate change (global warming) can influence forest diseases (distribution, impacts, biology, and so forth), and how those changes can influence forests and wildlands.

As a first step, a half day workshop, sponsored by the USDA FS, WWETAC and PSW, in cooperation with the 2007 Western International Forest Disease Work Conference (WIFDWC), was held on October 15, 2007 in Sedona, AZ. At the workshop, information on climate influences on abiotic diseases, canker diseases, forest declines, root diseases, rusts, mistletoes, decay organisms, and Phytophthora diseases was reviewed for the identification of information and research needs. The next step, this literature review, expands upon the material discussed at that workshop, and is intended to inform managers, decision makers, scientists, and other publics about the potential effects and impacts of occurring and predicted climate change on forest pathogens and forest pathogen/host interactions.

Contributors

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Background

Climate Changes

The world is getting warmer (Houghton and others 1996, IPCC 2007, Wigley 1999). Although the Earth’s climate has fluctuated for millions of years (Jackson and Overpeck 2000, Russell 1941), the recent acceleration in warming is alarming to many. Over the last 100 years, average global temperatures have risen by about 0.76°C. Probably of more concern, the rate of warming is increasing (IPCC 2007). These changes are anomalous compared to those that have occurred over the last millennium (Mann and others 1999). In the conterminous U.S., the average temperature has risen by 0.5°C and precipitation has increased 5 to 10 percent (National Assessment Synthesis Team 2000). The problem is not just changing temperatures; but also change in the weather patterns to which people and ecosystems have adapted. The term “global warming” is sometimes used in place of the more general, but perhaps more accurate, “climate change” to refer to the same phenomena.

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 CO₂, the most important anthropogenic greenhouse gas, has risen dramatically since preindustrial times and carbon cycle models project concentration increases from 379 ppm in 1995 to 540–970 ppm by 2100 (Intergovernmental Panel on Climate Change, 2001, IPCC 2007). Even if human-caused greenhouse gas concentrations were to be stabilized, warming due to greenhouse gases and aerosols would continue for centuries due to the time scales associated with climate processes and feedbacks (IPCC 2007).

From 1990 to 2100, average global temperature is projected to increase from 1 to 6°C (2 to 10°F), with warming in most of the U.S. expected to be even greater (Houghton and others 2001) Warmer winter temperatures and more frequent droughts are predicted throughout most of the western U.S. Extremes in weather are expected to become more common and more severe (Hopkin and others 2005). Scenarios created by climate change models include a general increase in temperature and a decrease in moisture for western forests. For example, in six of seven scenarios run through MAPSS (Mapped Atmosphere-Plant-Soil System, which models the response of plant functional types to climate), the western U.S. will have wetter winters, and warmer summers, throughout the 21st century relative to the current climate (USDA Forest Service 2003). Precipitation may increase slightly in winter months, but summers will remain hot and dry and be longer than they currently are. Interior western forests would likely have more precipitation, but it would fall mainly in the traditional October to April wet season (USDA Forest Service 2003). For the Pacific Northwest, Mote and others (2003), using output from eight climate models, projected a warming of 0.5 to 2.5°C (0.9 to 4.5°F) by the 2020s, 1.5 to 3.2°C (2.7 to 5.7°F) by the 2040s, and an increase in precipitation except in summer. In British Columbia, the snowmelt may occur earlier and quicker, and the summer low flows may be drier and earlier (Hamann and Wang 2006).
There is consensus that the effect of predicted climate change on key environmental factors may have profound implications for forest dynamics (Aber and others 2001, Ayres and Lombardero 2000, Dale and others 2001). These climate changes will cause major changes in ecosystems, including changes in the geographic distribution of vegetation types; ecosystem processes such as productivity, and the distribution and abundance of individual species (Malcolm and Pitelka 2000). Some scientists suggest that continued warming will jeopardize the integrity of many terrestrial ecosystems and will pose a threat to existing biodiversity (for example, see Malcolm and others 2006). In British Columbia, Hamann and Wang (2006) suggest that projected climate change will result in currently important sub-boreal and montane climate regions disappearing, with some of the most important conifer species in those regions losing a large portion of their suitable habitat.

Key Points

While we are uncertain how forest pathogens will respond to climate change, some general statements presented here and under individual types of diseases may be helpful in understanding logical responses by forest pathogens to various environmental conditions, new rates of change, and exposure to new climate conditions.

- Changes will occur in the type, amount and relative importance of pathogens and diseases. With warming, some diseases may be able to occur farther north or at higher elevations than under current conditions. Pathogens may play new roles in the health and functioning of newly forming forests because these conditions will be distinctive from any previous forest. If managers facilitate migration of tree species in new environments, then new disease situations should be expected.

- The epidemiology of plant diseases will be altered. Prediction of disease outbreaks will be more difficult in periods of rapidly changing climate and unstable weather.

- In a rapidly changing environment, host resistance to pathogens may be overcome more rapidly due to accelerated pathogen evolution.

- Because abiotic factors such as temperature and moisture affect host susceptibility and pathogen aggressiveness, interactions between biotic and abiotic diseases may represent the most important effects of climate change on plant diseases.

- When combined with climate change, trends in increasing invasions by non-native pathogens means that new epidemics may occur.

- Greater overwintering success of pathogens will likely increase disease severity. Because temperatures are expected to increase more in winter than in other seasons, this population bottleneck may be removed for many pathogens.

- Climate change could have positive, negative, or no impact on individual plant diseases, depending on the ecosystem and climate conditions.

Climate Change and Disturbance Agents in Forest Ecosystems

Forest ecosystems are affected both directly and indirectly by disturbances. Disturbances are natural and essential components of forest ecosystems. Fire, drought, insects and
pathogens, hurricanes, windstorms, and ice storms are the most important, and most often mentioned, natural disturbance mechanisms causing forest change in the U.S. (McNulty and Aber 2001). Climate change will affect and alter these natural disturbances (Dale and others 2000, Joyce and others 2001). There is ample evidence that climate change is already altering natural ecosystems (Walther and others 2002). When disturbances exceed ecosystem resilience, fundamental change in forest structure and function is inevitable (Gunderson 2000, Drever and others 2006). Many disturbance agents, including pathogens, will be affected by climate change. Impacts of disturbances and thus of climate change are seen over a broad spectrum of spatial and temporal scales (Peterson 2000). Fire and drought are often used as examples in the literature as disturbance agents that will likely increase and adversely affect ecosystems as climate warming occurs. As climate change occurs, forests may be more predisposed to other disturbance factors, such as insect and disease outbreaks (McNulty and Aber 2001). Because plant disease involves the interaction of a susceptible host, a favorable environment, and a virulent pathogen, any climate change that results in the host becoming more susceptible to the pathogen or in an environment more suitable for the pathogen, will result in increased disease. At least some forest species have a capacity for rapid genetic response to temperature increases (Jump and others 2006) which may or may not be ultimately adaptive under an altered disturbance regime.

Along with physiological changes caused by drought and other abiotic factors, the most important effects of climate change on forest tree species will probably be mediated through changes to disturbance events caused by fire, insects and pathogens. Ayres and Lombardero (2000), Harrington (2002) and Logan and others (2003) provide broad overviews of potential climate change impacts on forest insects and pathogens. Based on an analogy of responses of forest ecosystems to invasive pathogens, key assessment features are 1) disease type, 2) host specificity, 3) pathogen aggressiveness, 4) host importance, 5) functional uniqueness of host, and 6) phytosociology of host (Lovett and others 2006). Anderson and others (2004) further identify the potential for emerging plant diseases responding not only to anthropogenic introduction, but also to increases in extreme weather events.

The forested area impacted by insects and pathogens (20,400,000 ha annually) in the U.S. is approximately 45 times that of fire (450,000 ha annually), with an economic impact that is 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 insect and disease-caused tree mortality increased from about 1.2 million ha (3 million acres) in 1997 to about 4.9 million ha (12.2 million acres) in 2003. Since 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 overstocking, pathogens, or climatic conditions such as drought may not survive the additional climatic stress (Winnett 1998). An example is what is occurring in southern California forest ecosystems. 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 and annosus root disease, predisposed conifers to bark beetle attack. The resulting bark beetle epidemic and subsequent tree mortality provided fuels for wildfires that occurred in late October and early November 2003. More than 300,000 ha (742,000 ac) of brush and timber burned over a one week period, destroying 3360 homes, resulting in 26 deaths, and causing over $2.5 billion in damage (Keeley and others 2004). The affected ecosystems have been drastically altered—if not permanently, then certainly for a long time. And the drought in southern California continues. A second example is the recent mortality of pinyon pine (*Pinus edulis* Engelm.) across 12,000 km² of the Southwest. The mortality was attributed to trees stressed by pathogens and drought, resulting in subsequent bark beetle infestation and regional-scale die-off (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 (Brasier 2005), resulting in changes in disease impacts. Global climate change is ultimately expressed at the microclimatic scale which controls reproduction, dispersal, and infection by many plant pathogens (Waggoner 1965). Host condition is also affected by temperature, moisture, and by variations in their cycles and extremes; pathogens may then take advantage of either increased host susceptibility or availability (see Ayers and Lombardero 2000, Hopkin and others 2005). In addition to demographic and epidemiological interactions, hosts and pathogens also respond genetically with consequences in community structure and ecosystem function (Burdon and others 2006).

Information available on relationships between forest tree pathogens and climate has not been fully reviewed. However, the following sections do summarize the literature on climate relationships and various types of diseases, and the possible effects of climate change on pathogens in western North American forests. Rather than attempting to summarize 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.

**Abiotic Diseases**

Some general statements:

- Because abiotic factors such as temperature and moisture affect host susceptibility and pathogen aggressiveness, interactions between biotic and abiotic diseases may represent the most important effects of climate change on plant diseases.
- Drought-stressed trees experience greater damage by many pathogens, particularly facultative parasites.
Abiotic diseases are caused by non-living agents. Examples include air pollutants, extremes of weather (frost, winter injury, heat stress), water deficiency (moisture stress, drought) or excess, nutrient deficiencies, and others. Abiotic, environmental factors affect plant disease in several ways. First, abiotic diseases can result from the effects of the environment alone directly on the host. A warming climate may accelerate photochemical reaction rates and lead to more water vapor in the lower atmosphere, which would tend to produce more of the pollutant ozone (Sitch and others 2007). In addition to damaging plants directly and reducing their ability to sequester CO$_2$, ozone has been shown to increase tree susceptibility to root disease (James and others 1980a, 1980b). Direct effects of environmental extremes (for example drought, low or high temperatures) are generally negative (Desprez-Loustau and others 2006). An increase in drought as a result of climate warming will negatively impact many forest ecosystems.

Second, factors such as temperature and moisture can affect host susceptibility and pathogen aggressiveness. For example, severity of the foliage disease Swiss needle cast is strongly correlated with winter temperature in western Oregon, due to its effect on growth of the pathogen (Manter and others 2005). Hosts predisposed by drought can suffer very severe damage. Water stress was consistently associated with more severe symptoms on pines attacked by Sphaeropsis sapinea (Bachi and Peterson 1985, Blodgett and others 1996, Blodgett and others 1997, Paoletti and others 2001). Drought conditions in northwestern Alberta in the 1960s and 1980s predisposed aspen stands to damage by fungal canker pathogens and wood-boring insects (Hogg and others 2002). In the southwestern U.S., widespread mortality in the pinyon-juniper forest type is associated with several years of drought; a complex of drought, insects, and disease is responsible for pinyon mortality rates approaching 100 percent in some areas (Shaw and others 2005). Third, abiotic (environmental) factors are predisposing, inciting, or contributing agents in declines (Manion 1991). As a healthy tree is stressed one or more times, its carbohydrate reserves are depleted and its defense systems become impaired, making it vulnerable to attack by insects and pathogens (Wargo and Haack 1991).

The literature contains numerous predictions of the effect of climate change on abiotic diseases, on host susceptibility and pathogen success, and on declines. Some examples include:

- Future drought is projected to occur under warmer temperature conditions. Of particular concern is regional-scale mortality of overstory trees, which would rapidly alter ecosystem type, associated ecosystem properties, and land surface conditions for decades (Folke and others 2004, Breshears and others 2005).
- Mortality of small stature plants (seedlings and saplings) is a likely consequence of severe drought. In comparison, deep rooting and substantial reserves of carbohydrates and nutrients make mature trees less susceptible to water limitations caused by severe or prolonged drought. However, severe or prolonged drought may render even mature trees more susceptible to insects or disease (Hanson and Weltzin 2000, Joyce and others 2001).
- An increased incidence of summer drought would make trees more vulnerable to attack by weak pathogens (Redfern and Hendry 2002), favoring diseases caused...
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).

- Abiotic diseases associated with environmental extremes are expected to increase, and interactions between biotic and abiotic agents might represent the most important effects of climate change on plant diseases (Boland and others 2004).
- Facultative pathogens, such as Armillaria root rot, as well as secondary canker-causing fungi, which are more successful infecting stressed hosts, would benefit from the heat and drought stress caused to forest and urban trees (Boland and others 2004, Schoeneweiss 1975).
- Climate change can be expected to create abnormal stress conditions in forest stands (Columbia Mountains Institute of Applied Ecology 2005).
- Forests under stress are likely to be further stressed. Forests on marginal sites may deteriorate if climatic changes make conditions less conducive to survival. Forests already stressed by crowding, pathogens, or atmospheric conditions may not survive the additional climatic stress (Winnett 1998).
- In a rapidly changing climate with increased temperatures, evapotranspiration, and extreme weather events, there will be an increase in the frequency and severity of stress factors, which may lead to more frequent forest declines (Sturrock 2007).
- Climatic extremes accelerate chronic declines. A variety of pests, pathogens and parasites can take advantage of trees stressed by environmental changes (Jurskis 2005).

Declines

General statement:

- Forest declines are predicted to increase as tree species become stressed by climatic conditions on particular sites and in portions of their ranges.

In the concept of tree decline developed by Manion (Manion 1991, Manion and Lachance 1992), decline is characterized by a slow and progressive deterioration in tree health or vigor, decreased tree growth accompanied by branch dieback, primarily occurring on mature trees, and a complex etiology often involving abiotic and biotic factors. Manion describes the etiology of declines as involving long-term predisposing factors (such as site, age, climate, genetic factors) that predispose the tree to short-term inciting factors (such as frost damage, defoliation) that weaken the tree and allow contributing factors (such as opportunistic pathogens that are normally considered secondary) in combination to kill the tree. There are several examples in the literature where climate change has been considered to be the main inciting factor in tree decline.
A decline and mortality of western white pine, referred to as pole blight, was observed in the Inland Empire region of the Pacific Northwest in the 1930s and 1940s. Leaphart (1958) reported that the decline involved root deterioration and shallow soils of low moisture holding capacity. He and others (for example, Wellington 1954) had observed the disease associated with shallow soils and stressed trees. Climatic records for the areas of pole blight indicated that the stands of western white pine experienced a period of low precipitation and high temperatures from 1916 to 1940 that were the longest and most intense the last 280 years (Leaphart and Stage 1971). Various contributing factors, including Armillaria root rot and canker pathogens, were associated with the pole blight. Using Manion’s concept, the pole blight can be described as caused by the combination of predisposing shallow soils, the inciting factor of drought, and the contributing factors of several secondary pathogens.

A mortality of yellow-cedar (Chamaecyparis nootkatensis [D. Don] Spach), known as yellow-cedar decline, occurs on about 200,000 hectares in southeast Alaska and British Columbia (Hennon and others 2005, Wittwer 2004). Hennon and others (2006) explain the history of the decline, and the current hypothesis to explain the phenomena. 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 areas of low snowpack in winter and spring. A risk factor study provided evidence that seasonal air and soil temperature were consistently correlated with yellow-cedar decline, but saturated soils, acidic soils, aluminum toxicity, and calcium deficiency were not. Maps of yellow-cedar decline indicate strong patterns of occurrence on the landscape where snow accumulation is lowest, which is consistent with the concept that snow provides protection for yellow-cedar. The forest zones with abundant dead yellow-cedar had temperatures that were higher in the spring and summer, lower in winter, and had greater daily ranges. Snow delays soil warming and presumably protects yellow-cedar roots through periods of spring frosts. Recent research indicates that the decline is related to climate warming, resulting in loss of cold hardiness due to a reduced protective snow pack. Warm spring days followed by freezing weather damaged the roots (Schaberg and others 2007). Thus, climate warming is hypothesized to be the primary cause (Beier and others 2008, Hennon and others 2006, Hennon and others 2008) operating most directly as an inciting factor.

A recent premature dying of aspen stands, referred to as sudden aspen decline, is occurring in southern Colorado, Utah and Nevada. The sudden decline is characterized by widespread, severe, rapid branch dieback and crown thinning leading to mortality. The current hypothesis is that the widespread and severe drought conditions of 2000 to 2005 may have caused stress in aspens, particularly in mature stands of aspens at lower elevations, making the trees weaker and more susceptible 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 of 2000 to 2005), and contributing factors (secondary insects and pathogens including Cytospora canker [Valsa sordida Nitschke], aspen bark beetles [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).

Canker Diseases

Cankers (diseases leading to dead sections on branches or main trunks of trees) 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 favored by heat and drought stress (Schoeneweiss 1975, 1981). Outbreaks or epidemics of these stress-related canker diseases usually indicate that the trees have been predisposed by stress, the most common being water stress and freezing stress (Schoeneweiss 1981). Most published studies refer to a positive association between drought and disease, or to drought and disease acting synergistically on tree health status, with a predominance of canker/dieback diseases, caused by facultative parasites, such as Botryosphaeria, Sphaeropsis, Cytospora and Biscogniauxia (Hypoxylon) (Desprez-Loustau and others 2006). Some examples include:

- **Botryosphaeria dothidea** attacks a wide range of host species, but causes serious damage only to those host plants 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) is consistently associated with water stress (Bachi and Peterson 1985, Blodgett and others 1996, Blodgett and others 1997, Paoletti and others 2001). The most severe Sphaeropsis shoot blight occurred in the driest year and the least in the wettest year (Blodgett and others 1997).
- 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, University of California Davis, 2007, unpublished). Climate change resulting in warmer temperatures may increase the range of the pathogen.
- The pathogen *Biscogniauxia mediterranea* (De Not.) Kuntze (*Hypoxylon mediterranea*), a serious problem in oaks in the Mediterranean area, and not
previously detected further north than southern Tuscany, is now found in Slovenia causing damage to oak species (Jurec and Ogris 2006).

Based on published research and predicted changes in climate, the literature contains numerous statements concerning the potential effects of climate change on canker diseases, including:

- Most canker-causing fungi would benefit from heat and drought stress caused to forest and urban trees (Boland and others 2004, Schoeneweiss 1975).
- An increased incidence of summer drought would favor diseases caused by fungi whose activity is dependent on host stress, particularly numerous canker fungi (Broadmeadow 2002, Lonsdale and Gibbs 2002). For example, incidence of *Sphaeropsis sapinea* on pines would increase as drought increases.
- Some other canker diseases, such as Thyronectria canker of honeylocust (caused by *Thyronectria austro-americana* [Speg.] Seeler) and *Cryphonectria cubensis* (Bruner) Hodges on *Eucalyptus* would decrease (presumably because the pathogens are favored by high rainfall and the hosts display some resistance to drought stress) (Despres-Loustau and others 2006).
- Climate change could lead to outbreaks of *Biscogniauxia mediterranea* further north than current distribution (Jurec and Ogris 2006).
- The pitch canker pathogen, now limited by environmental conditions, may find favorable conditions in the Sierra Nevada as milder winter minimum temperatures occur (Battles and others 2006).

**Root Diseases**

**General statement:**

- Hot and dry conditions (for example, prolonged drought) are expected to increase incidence and spread of root diseases in forests. However, it is difficult to specifically predict how climate change will affect diverse root diseases under various projected climate scenarios.

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. For root pathogens like these, any climate change could increase their incidence and spread if host trees become stressed. In addition, climate change could alter fitness of various mycorrhizal fungi and other beneficial microbes that currently suppress root disease. The protective effects of mycorrhizas against various root diseases may be altered by changes in the relative fitness of different mycorrhizal fungi under conditions of altered soil temperature or moisture regime (Broadmeadow 2002). Some reports suggest that hot and dry conditions (prolonged drought) are expected to increase incidence and spread of root diseases in forests. However, it is difficult to
specifically predict how this climate change will affect diverse root diseases under various projected climate scenarios. Although actual data is limited, a rise in atmospheric CO$_2$ and subsequent increased growth of host roots, may result in an increase in severity or frequency of root disease. More extensive root systems would increase the probability for 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 reduced growth and mortality (Shaw and Kile 1991). *Armillaria* root disease caused by *A. ostoyae* (Romag.) Herink is responsible for losses of 2 to 3 million m$^3$/year 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 while other species act as secondary agents, infecting after hosts have experienced a predisposing stress agent. These stress agents may include drought, other diseases, insects, site conditions, or other factors. *Armillaria* root disease can be favored by hot, dry conditions (Shaw and Kile 1991, U.S. Office of Technology Assessment 1993), and its incidence can be expected to increase under warmer or drier conditions. In California, *Armillaria* spp. reduce growth and increase mortality in several Sierra Nevada conifers, and disease severity is expected to increase under conditions of frequent summer drought. Such increases will lead to overall timber productivity declines and may increase insect attacks on stressed trees, resulting in more overall mortality (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 undergone drought stress (Wargo and Harrington, 1991). In the Pacific Northwest where the mean annual temperature presently is below the optimum (25°C [77°F]) for *Armillaria* growth, a warmer climate is likely to result in increased prevalence and rate of spread of root disease (Intergovernmental Panel on Climate Change 2001).

Adverse impacts from annosus root disease are also expected to increase with predicted climate change. In general, the pathogen *H. annosum* is more common and damaging on drier sites (see Puddu and others 2003 for example). Nilson and others (1999) suggest that global warming will provide more favorable environmental conditions for *H. annosum* spread, resulting in increased damage and greater economic loss.

Laminated root rot, caused by *Phellinus weirii* (Murrill) R.L. Gibertson, 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 (W.G. Thies, personal communication). Forest soils can be a great buffer for the temperature changes in the atmosphere, so a few degrees of increased temperature perhaps may not significantly affect the fungus (W.G. Thies, personal communication).
Because occurrence and distribution of root rot pathogens are closely linked with their host species, it is essential to predict distribution of host species in response to climate change. Currently, a modeling approach is available to predict current and potential future distributions of forest trees using climate variables (Rehfeldt and others 2006). However, the gross scale of such models precludes prediction of microclimate changes that will dramatically affect both hosts and root pathogens.

**Phytophthoras**

Numerous species in the genus *Phytophthora* are parasitic on a wide variety of host plants, including forest trees. While some *Phytophthora* species are quite host specific, others have rather broad host ranges. *Phytophthora* owes its success, in part, to its ability to increase inoculum levels from low, often undetectable levels to high levels within a few days or weeks due to the rapid production of sporangia and zoospores when environmental conditions are favorable. Most *Phytophthoras* are considered to be most damaging under relatively mild to warm conditions. Moisture—including rainfall, dew deposition, and irrigation—is the main environmental factor that controls pathogen population levels. The multi-cyclic nature of *Phytophthora*-caused plant diseases has resulted in severe epidemics in forest and agricultural systems worldwide.

*Phytophthora cinnamomi* Rands, a widespread pathogen in warmer, tropical and subtropical regions of the world, causes root and stem-base diseases of a wide range of broadleaved and coniferous species, and has been well studied in a variety of forest ecosystems. The fungus-like organism is soilborne and requires warm, wet soils to infect roots. The pathogen is currently present, but limited as a major pathogen in conifer forests of the Pacific Northwest. Although Douglas-fir is susceptible to the pathogen, the wetter winter months are currently too cold for effective zoospore production and infection, and the warmer summer months are currently too dry (Roth and Kuhlman 1966). This could change if climate change results in the predicted warmer winters in the Pacific Northwest. Chee and Newhook (1965) also found that sporulation of *P. cinnamomi* did not occur at temperatures that prevail in the soil during the normally wet winter months in New Zealand. This explains earlier observations that epidemic losses due to *P. cinnamomi* in New Zealand tend to be restricted to years when rainfall is abnormally high in autumn and late spring when soil temperatures are sufficient to support sporulation.

In the case of *P. cinnamomi* and similar organisms, it may not only be overall climatic warming, but an increase in weather periods favorable to inoculum production and spread that will lead to an increase in activity (Brasier and Scott 1994). A warming climate
would not only be favorable for *P. cinnamomi* in areas where it already occurs, but also would favor the northward movement of the pathogen into new areas (Hepting 1963). *Phytophthora cinnamomi* is likely to become more prevalent across Europe. Models show a probable significant increase in the activity of the pathogen across the U.K. and Europe in general as a result of higher year-round temperatures and northward movement of the pathogen as temperatures become favorable for its development (Brasier 1996, Brasier and Scott 1994). In Europe, the winter survival of the pathogen is the limiting factor for amount of the disease it causes to oaks, especially *Quercus robur* L. and *Q. rubra* L.

Bergot and others (2004) compared the potential pathogen and disease geographic ranges of *P. cinnamomi* in France between a reference period from 1968–1998 and a projected period from 2070–2099. Simulations were obtained by predicting the pathogen winter survival in relation to microhabitat temperature (in the phloem of infected trees) based on a regionalized climatic scenario derived from a global circulation model. Increases in winter temperatures calculated with this scenario were in the range 0.5–5.1˚C between the two periods. As a consequence, higher annual rates of *P. cinnamomi* survival were predicted, resulting in a potential range expansion of the disease of one to a few hundred kilometers eastward from the Atlantic coast where it occurs now within one century. Using the CLIMEX model, Desprez-Loustau and others (2007) demonstrated that predicted warming would be favorable to most of the pathogens they studied, especially those such as *P. cinnamomi* for which winter survival is a limiting factor linked to low temperatures.

Climate warming may provide more favorable conditions for *P. cinnamomi* in northern California than occur now. Garbelotto and others (2006) reported *P. cinnamomi* infecting 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) while September (average temperature of 24˚C) inoculations resulted in larger lesions (135 ± 68 mm), confirming previous research that 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 climatic conditions (in particular hotter summers and wetter springs that favor greatly increased inoculum production by the pathogen) become more extreme (Frankel 2007, Rizzo and others 2005). *Phytophthora ramorum* is addressed specifically in the foliar diseases section.

### Foliar Diseases

**General statement:**

- The majority of foliar pathogens are likely to increase from climate change as a result of extended conducive conditions in the late winter and early spring (warmer temperatures and increased moisture) and reduced winter mortality.
Because they tend to be strongly influenced by weather, pathogens causing foliar diseases are one of the pathogen groups most likely to be affected by changing climate. Sporulation and infection often occur within a very 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. Hepting (1963) calls these “threshold diseases”. These threshold diseases usually vary in severity depending on annual weather patterns, and so are very likely to increase in regions where climate changes bring warmer and wetter conditions. Conversely, climate changes that decrease periods of precipitation and increase periods of drought would result in a decrease of some foliar diseases. Timing of sporulation of many foliar pathogens is also synchronous with emergence and development of new tissue on host plants. Climatic changes that favor development of susceptible host foliage during more favorable environmental conditions will also influence foliage disease outbreaks. Elevated CO$_2$ concentrations increase host growth and pathogen fecundity (Coakley and others 1999, Chakraborty and Datta 1993, Chakraborty and others 2000a); when combined with increased humidity, foliar diseases are likely to increase (Coakley and others 1999, Manning and von Tiedemann 1995).

Information on specific foliar diseases and weather is limited, but general statements or predictions have been made, based on the known dependence of these pathogens on weather. The incidence and severity of most foliar diseases will likely increase if climate change leads to wetter, warmer late winters and springs, but may decrease with drier summers (Broadmeadow and Ray 2005). Increasing nighttime temperatures may work for or against various pathogens, as the humidity requirement is often met during nighttime hours. Increasing temperatures will likely benefit some foliar pathogens and prove detrimental to others, depending on the specific pathogen (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 native pathogen, a severe epidemic has continued since the early 1990s in the coastal fog belt of Oregon (Hansen and others 2000, Kanaskie and others 2004). Although the reasons for the current outbreak are not known, an explanation offered is that the pathogen was favored by the dense planting of Douglas-fir in plantations in moist, coastal locations where Douglas-fir was not a dominant species in presettlement forests. Manter and others (2005) reported that the weather factors most highly correlated with both fruiting body abundance and premature needle loss were winter mean daily temperature and spring cumulative leaf wetness, and developed a temperature-based model to predict geographic variation in disease severity. Although the authors could not project changes in disease severity based on winter average daily temperature, they did suggest a relationship between recent regional climate patterns (average temperatures for the period of 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) and the observed increase in the disease. In New Zealand, where Douglas-fir is not native, winter mean temperature was the single climate
variable most closely correlated with *P. gaeumannii*, accounting for about 80 percent of the variation in disease severity (Stone and others 2007).

Climate change provided ideal environmental conditions (correct temperature and humidity) for the recent buildup of Dothistroma needle blight (caused by *Dothistroma septosporum* (Dorog) Morelet) on lodgepole pine (*Pinus contorta* var. *latifolia* Dougl. ex Loud.) in Canada (Woods and others 2005). The native needle pathogen, *D. septosporum* (*Mycosphaerella pini* Rostr.), has traditionally been a low-impact pathogen in Canada, rarely resulting in damage. However, the needle blight is currently causing extensive defoliation and mortality in plantations of lodgepole pine in northwestern British Columbia. Lodgepole pine plantations, heavily planted over the last 30 years and covering close to 40,000 ha in northwest British Columbia, are most severely affected, and entire plantations of lodgepole pine are failing. The severity of the disease is such that mature pine trees are also dying (Woods 2003). Previously Dothistroma needle blight has been a serious forest pathogen—causing 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 growth retardation due to defoliation (Bradshaw 2004).

Noting that the current Dothistroma needle blight epidemic coincides with a prolonged period of increased frequency of warm rain events throughout the mid-to-late 1990s, Woods and others (2005) analyzed climatic data for the region. They found a strong relationship between the observed trend of increasing precipitation and disease severity. More significantly, their studies demonstrate how a relatively small change in climate can have serious implications for a tree species, particularly if that change surpasses an environmental threshold that has restricted the development of a pathogen previously not important because of unfavorable climate.

Other foliar pathogens are more successful during periods of drought. A reduction in the number of summer rain-days may reduce the incidence of various foliar diseases such as Marssonina leaf spot of poplar (Lonsdale and Gibbs 2002). Wagener (1959) reported increased damage to ponderosa pine from the needle cast pathogen *Davisomyccella medusa* (Dearn.) Darker during periods of drought. A second needle cast pathogen, *D. ponderosae* (Staley) Dubin, caused severe damage to ponderosa pine on the San Juan National Forest in Colorado (Worrall and Sullivan 2002). The authors suggest that an unusually wet summer in 1999 was favorable for an increase in populations of the pathogen and infection of the host. Subsequent drought years allowed that pathogen to develop and kill infected needles.

In addition to changes in climate providing a favorable environment for known pathogens, novel, introduced or emerging pathogens may become more important as a result of climate change and forest management. A *Phytophthora* foliar pathogen that has recently emerged and is strongly influenced by environmental conditions is *P. ramorum*, the sudden oak death pathogen. In addition to causing lethal stem infections in oaks (*Quercus* spp.) and tanoak (*Lithocarpus densiflorus* [Hook. & Arn.] Rehder), *P. ramorum*
causes non-lethal foliar infections in a range of other species. Inoculum of *P. ramorum* increases rapidly with heavy rains associated with El Nino events (Davidson and others 2005). Dry conditions are unfavorable. Chlamydospores and zoospores of the pathogen can survive more than 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 show areas of the U.S. suitable or not for development of *P. ramorum*. Climate change resulting in wetter springs would favor development of the pathogen not only in areas where it already occurs, but may provide favorable conditions for establishment of the pathogen in new locations.

**Stem Rusts of Pine**

- Although rusts can adapt to a wide range of environmental conditions, their limits are unknown. Under a changing climate, the incidence of rusts will be determined chiefly by host distribution and are likely to remain the cause of damaging and lethal diseases.

- Typically, rusts exhibit wave year increases of intensity and expansion in distribution when the weather is especially favorable for sporulation, dispersal, and infection. With climate change, such wave years are expected to occur but with different frequency.

Stem rusts of pine (*Cronartium*) are aerially-dispersed, biotrophic, fungal pathogens with long evolutionary associations to their hosts, and with simple to complex life cycles (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 subgenus Strobus*) (Mielke 1943). Natives, comandra blister rust (*C. comandrae* Peck) and western gall rust (*Peridermium harknessii* J.P. Moore), are important pathogens of other pines (subgenus *Pinus*). Except for the spores which are exposed to the atmosphere, these rusts require living hosts of a taxa specific for the pathogen. The microcyclic rusts such as *P. harknessii* spread directly from pine to pine; the *Cronartium* rusts, however, must alternate between the pine host (pycnial and aecial stages) and an angiosperm host (uredial, telial, basidial stages). The abundance of these rusts is therefore influenced by both weather and by host populations—each of which is affected by climate change. Severe epidemics of stem rusts occur in favorable times and places with profound effects to the genetics and demography of the infested pine (Kinloch 2003). Phylogeography, biogeography, and ecology of the stem rust pathosystem provide the bases for assessing rust–climate interactions, threats to forest ecosystems, and potential mitigations.

Spore development, dispersal, and germination of rust fungi are directly affected by the abiotic environment (for example, see Powell 1972, Chang and Blenis 1989). The environmental requirements for rust infection are complex and have been especially well-
documented for *C. ribicola* (Bega 1960, Mielke 1943, Spaulding 1922, Van Arsdale 1954). Epidemiology and ecophysiology are well studied for *P. harknessii, C. comandrae*, and *C. ribicola* (Van Arsdale and others 2006). These pathosystems commonly display a wave year phenomena when especially favorable weather is accompanied by great increases in rust spread and intensification (Peterson 1971, Jacobi and others 2002). Because stem rusts respond to weather events, the regional or even site-specific climate as monthly averages may not be sufficient for predicting rust hazard. An increase in hot and dry conditions as a result of climate warming may decrease infection either by inhibiting the rust or defoliating the telial host (Kimmey 1944, Kimmey and Wagener 1961). In some typically 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). Since infection is usually through stomates, whatever affects stomatal opening (for example: CO\(_2\) concentration, drought) affects infection.

The lack of an apparent climate signal for white pine blister rust in British Columbia indicates that climate is not presently a limiting factor there (Campbell and Antos 2000); climate, however, may restrict the rust elsewhere. After rapid expansion of blister rust from its introduction near Vancouver, BC (Mielke 1943), spread stalled to the east (eastern Montana), southeast (Yellowstone), and south (California). Because susceptible hosts occur beyond the prevailing 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 Californian Sierra Nevada (Kliejunas 1985, Geils and others 2003). Colonization of these generally warmer and drier regions could indicate either the rust has greater adaptability, or suitable microsites are more common than initially thought. For example, the Yellowstone region was described as too cold and too dry to support severe infestation of blister rust (Hendrickson 1997). 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 are present at some locations on the periphery of the Yellowstone region (for example, Teton Pass and see Kearns and Jacobi 2007), but a credible analysis has not been done to correlate climatic factors with rust severity throughout the Yellowstone region. One factor which may reduce rust populations is a series of extremely cold, winter episodes 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 their 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 importance on disease progress and impact is variable and disputed (for example, Furniss and others 1972, Jacobi 1993, van der Kamp and Blenis 1996). The relationships between stems rusts and bark beetles are also complex and situational (Campbell and Antos 2000, Six
and Adams 2007). Lodgepole pines severely cankered by *C. comandrae* have lower sugar levels which increase susceptibility to mountain pine beetle (Nebeker and others 1995). Rust-infected trees may provide valuable habitat for bark beetles between outbreaks; host regeneration following a bark beetle outbreak, however, could provide many, especially susceptible, new hosts. Disease, insect attack, climate affects on hosts and symbionts are sufficiently interrelated and contingent that climate–ecosystem responses appear chaotic, patterned but unpredictable in specifics.

For several stem rusts, hazard models and epidemiological simulation models illustrate how climate, host, and pathogen are expected to interact and provide a basic assessment tool. In several examples, elevation, habitat type, or topographic position are correlated with rust distribution or incidence (for example, Beard and others 1983, Howell and others 2006). These factors are assumed to be surrogates for climate so inferences under different climate scenarios are possible. *Cronartium comandrae* is related to site and weather variables for projecting where severe outbreaks are likely (Jacobi and others 1993) and even expected volume loss (Woods 2006). Meteorological records indicated that weather conditions considered suitable for infection of lodgepole pine by *C. comandrae* (continuous periods longer than 6 hours during the months of July, August, and September when temperature ranged between 10 and 20˚C and the air was nearly saturated) occurred at least every few years within various regions of the Central Rocky Mountains. The frequency, but not the duration, of potential infection episodes was related to the prevailing summer weather patterns (Jacobi and others 2002). Changes in weather patterns resulting from climate change would likely decrease the frequency of potential infection episodes. Patterns of upper-level air flow favorable for the long distance transport of rust spores from a source to a target, coupled with data for surface conditions favorable for infection at a designated target, were used to identify periods favorable for transport of and infection by *C. ribicola* spores (Frank and others 2008). Climate warming would likely alter the variables involved.

Numerous models for white pine blister rust are available for different regions and landscape scales and using various approaches (Van Arsdel and others 1961). One example (McDonald 1996) illustrates how an epidemiological simulation model (McDonald and others 1981) could investigate climate by ecological and genetic interactions in the white pine blister rust. Northern Idaho and the southern Sierra in California have contrasting climates. Historically, northern Idaho had an early, severe rust epidemic; and southern Sierra only became infested many decades later. The rust simulator projects outbreaks using typical Idaho weather and rust parameters (in temperature response functions) derived from epidemiological studies of Idaho rusts. If one simulates stand development with meteorological data typical for California with the rust’s epidemiological parameters for Idaho, no outbreak occurs. But if one repeats the exercise with the parameters adjusted to represent shift in the rust’s response function, the model generates an outbreak (McDonald 1996). The inference from this exercise is that initially the warmer and drier California climate was unsuitable for the rust from cooler and moister Idaho; but a local, California rust successfully adapted to the prevailing climate through ecophysiological change. Ultimately, abiotic extremes of temperature and humidity restrict the rust, but the evolutionary potential of the rust allows for
alteration of its environmental envelop from what is currently observed in its present range. The response of stem rusts to climate change can be predicted; but managers must also allow for surprise and can do so by managing for resilience (Drever and others 2006).

Mistletoes

Some general statements:

- Both genera of mistletoes common to the western states can infect otherwise healthy host trees. When 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.

The dwarf mistletoe genus *Arceuthobium* and the true mistletoe genus *Phoradendron* (and other genera) 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 reduced tree growth, increased tree mortality, reduced seed and cone development, reduced wood quality, increased susceptibility of the host to pathogen and insect attack, and 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 as they have leaves and produce chlorophyll, but heavy infections can result in significant tree stress (Geils and others 2002). As stress agents, mistletoes may play a significant role in tree mortality as trees become stressed by drought and other factors as a result of climate change.

*Arceuthobium* infection stresses host trees, in particular host trees already stressed by drought. For example, the dwarf mistletoe/bark beetle complex is responsible for 40 to 60 percent of the pine mortality in southern California during years of normal precipitation. Mortality is more frequent when other stress factors occur, such as drought, oxidant air pollution damage, or competition in overstocked stands (Schultz and Allison 1982, Schultz and Kljejunas 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, dwarf mistletoe (*Arceuthobium campylopodum* Engelm.), and annosus root disease. In the central Sierra Nevada, red fir (*Abies magnifica* A. Murr.) are predisposed to fir engraver attack by several stress factors, including severe drought conditions, overstocking, annosus root disease, and dwarf mistletoe (Frankel and others 1988). Childs (1960) reported that branch killing of conifers in the Pacific Northwest, following an unusually hot summer in 1958 and an unusually dry summer the following year, was particularly widespread in trees stressed by dwarf mistletoe and other agents. He also suggested that the death of
dwarf mistletoe-infected branches may actually be beneficial by reducing inoculum and thus reducing spread of the disease.

The combined stress from dwarf mistletoe, drought and other stress agents often results in tree mortality. Increased drought conditions resulting from climate change would increase this mortality. Damage surveys during the 1976–1977 drought in California estimated that 12.3 million trees with 8.6 million bd. ft. had died. All sites and forest types were affected. Forest insects and pathogens were important contributors to 98 percent of the volume loss. 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 dwarf mistletoe-infected trees were the first to die during drought periods (Byler 1978), as the trees already stressed from dwarf mistletoe infection were less able to survive the additional stress resulting from low soil moisture. 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). Southwestern dwarf mistletoe was found most frequently and was most severe on the driest ponderosa pine sites, while severity of dwarf mistletoe was least in the wetter habitat types in southwest Colorado (Merrill and others 1987). Wilson and Tkacz (1992) reported severe drought combined with several other factors including dense stand conditions, and presence of pinyon dwarf mistletoe (A. divaricatum Engelm.), predisposed areas of pinyon pine (Pinus edulis Engelm.) in northern Arizona to attack by Ips confusus (Leconte). Gehring and Whitham (1995) found levels of true mistletoe (Phoradendron juniperinum) in junipers (Juniperus monosperma [Engelm.] Sarg.) three-fold higher on trees growing in droughty, cinder soils than in sandy-loam soils.

Since climatic factors limit the ranges of many dwarf mistletoes, climate change will likely result in geographic shifts, most notably extensions in range. Southwestern dwarf mistletoe appears limited by climate and does not occur throughout the entire range of ponderosa pine; distinct northern, upper altitude, and lower altitude limits exist (Mark and Hawksworth 1976). Surveys of occurrence, possible slope preference, and severity of several dwarf mistletoe species in Colorado revealed distinct altitudinal zones of parasitism specific for each host–parasite interaction (Williams 1971). For example, A. vaginatum was present to the upper range of its host (2,800 m = 9200 ft), but absent below 1,860 m (6100 ft). Below 2,130 m (7000 ft), ponderosa pine is vigorous and may be resistant to mistletoe, with temperature possibly being important. Arceuthobium americanum Nutt ex. Engelm. on lodgepole pine occurs at the lower, but not at the upper elevation limits of host growth (Hawksworth 1956). The altitude limit for the mistletoe may be due to the short growing season at the higher elevations, which is not long enough for the fruit to mature before severe frosts occur in the fall. Although Black Hills ponderosa pine is susceptible to dwarf mistletoe infection, the parasite is absent from the Black Hills and Bearlodge Mountains, apparently because the early onset of the cold season is destructive to 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 (*A. americanum*), a damaging pathogen of jack pine (*Pinus banksiana* Lamb.) in western Canada, is absent from jack pine forests in the colder, more northerly areas in western Canada, again suggesting that mistletoes are often limited 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 over other more northern parts of their host ranges. A warming climate would likely extend the range of these *Phoradendron* species.

**Wood Decays**

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<thead>
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<th>Some general statements:</th>
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<tr>
<td>• Decay of wood is dependent on moisture and temperature and thus could be heavily impacted by future climate change.</td>
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<tr>
<td>• With climate warming, white rot fungi, which break down carbon as well as lignin, may move northward. This would alter carbon sequestration rates by accelerating carbon breakdown.</td>
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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 by 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 conditions (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, while other fungi require more water and lower temperatures and are restricted to the center or bottom of slash piles (Loman 1962, Loman 1965, Spaulding 1929, Spaulding 1944).

Heart-rot fungi are pathogens that colonize the central portion of living trees. Although many heart-rot fungi have broad host ranges, others are more restricted and colonize specific tree species (Tainter and Baker 1996). Actively growing trees can often limit the amount of fungal colonization by mounting active chemical and physical barriers to restrict decay to the central column (Shigo 1984), but 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 factor for decay and is dependent on humidity, precipitation, and uptake from ground contact. Decay fungi cannot degrade wood with moisture contents below 30 percent (fiber saturation point; moisture content expressed on a dry-weight basis), and the upper moisture limits generally do not exceed 90 percent, due to lack of oxygen (Griffin 1977). Wood temperature is the second most important factor for fungal activity. Fungi can generally begin growth 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, but some can be higher. 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, Loman 1965, Spaulding 1929, Spaulding 1944). *Coniophora puteana* and *Stereum sanguinulentum*, isolated from lower portions of the same slash piles, had 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, Gilbertson 1981, Nobles 1958). Various studies (see Shafer and others 2001 for example) suggest that warm temperate tree taxa, largely hardwoods, that are currently limited by freezing temperatures will spread northward and replace conifers as the climate warms. This increase in hardwood taxa may increase the overall occurrence of white rot fungi. As white rots break down carbon as well as lignin, carbon sequestration rates will be altered by accelerating carbon breakdown.

Dead wood can be a substantial fraction of stored carbon in forest ecosystems, and decay rates of coarse woody debris may be sensitive to climate change. Kueppers and others (2004) found differences in biomass and decay rates along an elevation gradient, suggesting that warming will lead to a loss of dead wood carbon from subalpine forest. Modeling studies that have considered the decomposition of woody debris have concluded that an increase in regional warming and drying in the West would increase rates of decay, leaving less debris on the forest floor and resulting in increased 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. More research is needed to determine climatic effects on individual heart-rot diseases.
Summary

It is generally agreed that changes in climate will have influences on forest ecosystems. However, 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). The literature reviewed here indicates that information on interactions among the environment, forest pathogens and host susceptibility is limited. Over the longer term, 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.

In spite of these uncertainties, the potential effects of climate change on forest tree pathogens can be projected by analyzing the existing role of climate and weather on pathogen behavior (Lonsdale and Gibbs 1994). We know that individual diseases are favored under certain environmental conditions, and we know these specific conditions for many diseases. Studies of environmental effects on the distribution of pathogens and the severity of the diseases they cause today can provide valuable insight into likely direct effects of future climate change on forest diseases (Lonsdale and Gibbs 1994). Numerous general predictive statements, based on what is known about pathogen behavior under current environmental conditions, are found in the literature. Some examples include: climate change is likely to be broadly detrimental to tree health and favor some highly damaging pathogens; drought stress will increase impacts of many pathogens; warmer winters will increase pathogens such as Phytophthora; wetter springs will increase foliar pathogens; wetter falls will increase stem rusts; and so on. Predictions are relatively easy for pathogens whose range or activity are primarily affected by temperature, and become more difficult with pathogens affected by interactions with other organisms such as insect vectors (Lonsdale and Gibbs 1994). While this current knowledge has been used to predict host–pathogen response or interaction with changing climate, few data are actually available to validate the predictions (Runion 2003).

Generalizations about the effects of climate change on forest ecosystems and forest pathogens in the West are difficult since the effects will tend to be different for different pathosystems in different locations (Sturrock 2007) and would be influenced by factors including species composition, site conditions and local microclimate. Tree species differ significantly in their ability to adapt to warming, their response to elevated CO₂ concentrations and their tolerance to disturbances (Lemmen and Warren 2004). For example, compare the potential effects of climate change in Arizona where drought may dominate with the effects of climate change in Alaska where increased warmth may be important. Whether disease incidence or severity decreases, increases, remains the same, or shifts in geographic occurrence with predicted climate change will vary, depending on the host response, the specific pathosystem, and with the specific environment in which they occur (Runion 2003).
Implications

Various models have been used to predict changes in forest ecosystems due to climate changes. Some of these models have been utilized in predicting changes to individual pests (Baker and others 2000, Logan and others 2003). For example, the CLIMEX model has been used for *P. cinnamomi* (Desprez-Loustau and others 2007) and for *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 result in maladaptation of hosts and pathogens to the new climate and alter host–pathogen interactions that occur under the present climate.

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 conditions in southern California forests resulted in additional mortality of moisture-stressed trees from insect attack and root pathogens. The cumulative mortality resulted in tremendous fuel loads and catastrophic wildfire.

The role of abiotic stress factors, pathogens, and their synergistic interactions with the host are often not included under climate change scenarios (Loehle and LeBlanc 1996, Scherm 2004). There is consensus that climate changes will likely increase host stress and thereby predispose them to disturbance factors such as insects, pathogens, and to emerging diseases (Brasier 2001, McNulty and Aber 2001). Current information suggests that some disturbances affecting forests will shift in geographic region (for example, ice storms), or in frequency (for example, fire) or increase in severity (insect and disease outbreaks) (Hansen and others 2001, McNulty and Aber 2001) as a result of climate change. These factors need to be considered by models where information is available.

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 CO$_2$ 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 CO$_2$: 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 CO$_2$ 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 Tiedmann 1995).

Additional consequences of climate change and resulting maladaptation 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 sub-tropical pathogen _P. cinnamomum_ 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). Some decay fungi may also find warmer temperatures at northern latitudes favorable, resulting in greater activity by these fungi and potentially reduced carbon sequestration in northern forests. The literature on decline diseases, such as pole blight and cedar decline, provides examples of how a tree species can be affected by a change in climatic factors affecting the host physiology and site conditions. As future climate change occurs, additional examples of tree decline and resulting changes to the forest ecosystem will likely occur and need to be considered by models.

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. As pointed out in the report of the June 2007 workshop on Climate Change and Forest Insects and Diseases held by WWETAC (Beukema and others 2007), there are few models or tools which quantify how ecosystems might change, particularly those that include insects and pathogens.
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 forest ecosystems primarily by its effect on the frequency, intensity, duration and timing of disturbance factors, including fire, drought, introduced species, insect and pathogen outbreaks, hurricanes, windstorms, ice storms, or landslides (Dale and others 2001). Although significant as stress factors, pathogens will probably play a lesser role than other disturbances such as fire and drought in some ecosystems (Ayers and Lombardero 2000).

- Climate change could have positive, negative, or no impact on individual plant diseases. The most likely impact of climate change on forest pathogens will be felt in three areas: in the geographical distribution of plant diseases, in the losses they cause, and in the efficacy of disease management strategies. Changes will occur in the type, amount and relative importance of pathogens and diseases. Currently, insufficient information is available to quantify these effects. More research is needed to obtain fundamental understanding on different disease systems and supporting various potential mitigations (Chakraborty and others 2000b, McLachlan and others 2007).

- Climate change may cause a crossing of an environmental threshold, resulting in irreversible changes to ecosystems (Folke and others 2004, Pascual and Guichard 2005). Ecosystems may be able to withstand individual stresses, but multiple stresses occurring together could seriously impact forest health and sustainability (Aber and others 2001). Forests on marginal sites may deteriorate if climatic change makes conditions less conducive to host survival.

- Disease risk assessments developed previously for invasive and emergent diseases can provide a framework for organizing and presenting information on forest disease–climate change interactions (for example, Yang 2006). Information needs vary for decision makers at different levels, but all assessments ought to communicate assumptions, bias potential, uncertainty, and appropriate utilization. Assessments on forest disease–climate change should include an adequately supported and responsive infrastructure that includes research, stakeholders, and decision-makers at various agencies and levels (Hain 2006).

- Plant pathologists have a critical role in providing input to other disciplines and various publics on the effects of climate change on forest ecosystems. Pathologists are uniquely trained to understand host–pathogen–environment interactions as integrated systems (Bruck and Shafer 1991). They need to actively collaborate with other disciplines working on predicting the effects of climate change and planning on how to deal with them.

  - Modeling efforts would benefit from active networking among multiple disciplines working across scales from genomes to ecosystems (Proulx
and others 2005, Garrett and others 2006, Lundquist and Hamelin 2005). Although the complexity of host–pathogen interactions with climate change may be difficult to model, pathologists need to provide information so the synergistic impacts of climate change and other environmental stresses are considered in models of forest ecosystems.

- Managers and policymakers will need input and guidance from pathologists, in the form of viable management alternatives, in order to guide decisions on management as expected climate change occurs. Because managing climate change and forest ecosystems are complex problems, multiple approaches including models, formal assessments and distributed learning networks (Norgaard and Baer 2005) and adaptation policies that are responsive to a wide variety of environmental circumstances (Spittlehouse and Stewart 2003) are required. Pathologists should begin now to formulate ways for adapting to climate change and forest disturbance by maintaining forest ecosystem diversity and resilience.

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Citations


Manning, W.J.; von Tiedemann, A. 1995. Climate change: potential effects of increased atmospheric carbon dioxide (CO2), ozone (O3), and ultraviolet-B (UV-B) radiation on plant diseases. Environmental Pollution. 88: 219–245.


Nobles, M.K. 1958. Cultural characteristics as a guide to the taxonomy and phylogeny of the


55–65.


Pascual, M.; Guichard, F. 2005. Criticality and disturbance in spatial ecological systems. Trends in


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

Piercy-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


Heterobasidion abietinum in Abies alba forests in southern Italy. Forest Ecology and Management. 180:
37–44.


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.

Forest Science. 12:147–159.

Phytologist. 159: 531–538.


