THESIS

# ASSESSING THE ROLES OF MICROBIAL AGENTS AND ABIOTIC STRESSORS IN PONDEROSA PINE DIEBACK AND MORTALITY ACROSS THE WESTERN UNITED STATES

Submitted by

Michael D. McKee

Department of Agricultural Biology

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Master's Committee:

Advisor: Jane E. Stewart

Seth Davis
Marek Borowiec

ABSTRACT

ASSESSING THE ROLES OF MICROBIAL AGENTS AND ABIOTIC STRESSORS IN PONDEROSA PINE DIEBACK AND MORTALITY ACROSS THE WESTERN UNITED STATES

Ponderosa pine (*Pinus ponderosa*) is one of the oldest and most ecologically and economically important tree species in western North America. As the most widely distributed conifer on the continent, it plays a critical role in forest ecosystems and supports recreational, environmental, and commercial values. However, in recent years, increasing levels of dieback and mortality have been observed across its native range. These declines appear to be driven by a complex combination of biotic agents, such as fungal pathogens, and abiotic stressors like drought and heat. Abiotic conditions can weaken trees and increase their susceptibility to disease, but the specific causal agents behind this widespread dieback remain poorly understood. It is unclear whether the current landscape-level decline is primarily the result of one dominant factor or the consequence of multiple interacting stressors. The research presented in this thesis aims to address these gaps in knowledge by investigating the biotic and abiotic contributors to ponderosa pine decline. In Chapter 2, I present a multi-state field study conducted across regions of the western United States experiencing noticeable tree decline. This study assesses the prevalence of fungal pathogens and records abiotic stress conditions to determine their correlation with dieback and mortality. The overarching goal is to quantify ponderosa pine decline and evaluate how its drivers vary across different geographic regions. Specifically, this project addresses three key research questions: (1) What biotic and abiotic agents are contributing to dieback and mortality?

(2) Is the prevalence and severity of disease observed sufficient to cause or contribute to significant dieback and/or mortality? (3) Are the pathogen drivers of mortality consistent across the western United States, or do they differ regionally? By answering these questions, the study will provide valuable insights into the patterns and drivers of ponderosa pine dieback and inform future monitoring and management strategies.

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## 1.1 Introduction

### 1.1.1 Ponderosa pine (*Pinus ponderosa):* background and description

The ponderosa pine (*Pinus ponderosa*) is one of the oldest and most important trees species in western North America. It is the second most widely distributed conifer on the continent, with a native range extending throughout the western United States, southern Canada, and northern Mexico (**Fig. 1**). This long-lived species, often reaching over 500 years in age, can grow to impressive sizes, with mature individuals attaining heights over 200 feet and trunk diameters exceeding 6 feet (Van Hooser & Keegan 1988). Characterized with thick, rust-orange bark with scaly plates, ponderosa pine is also known for its distinctive needle arrangement, typically bearing two to three needles per fascicle (Harlow & Harrar 1968). The ponderosa pine exhibits considerable genetic and morphological variation across its range, and two distinct geographic varieties of ponderosa pine are recognized. The Rocky Mountain variety (*Pinus ponderosa* var. *scopulorum* Engelm.) is primarily found throughout the Rocky Mountains, while the Pacific variety (*Pinus ponderosa* var. *ponderosa*) is widely distributed across the mountain regions of the Pacific Coast, extending from British Columbia down through California and into western Nevada (Little 1979).

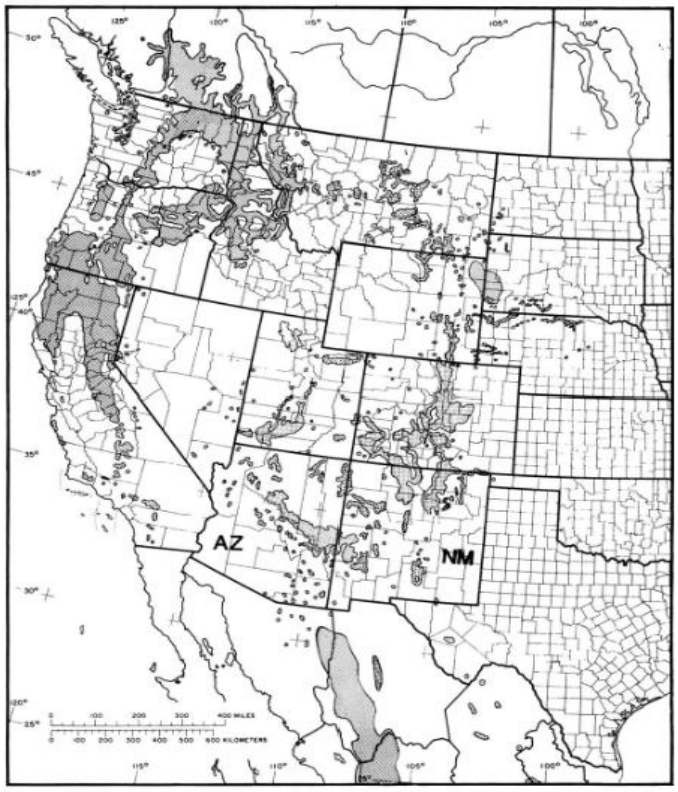


**Figure 1**. Distribution of ponderosa pine in North America (Moir et al. 1997)

1.1.2 Importance and use of ponderosa pine

The ponderosa pine has long been recognized as an ecologically and economically important species in western North America. Since the 1860s, its durable wood has been heavily harvested to support infrastructure development for settlements, agriculture, and mining operations. By the early 20$^{th}$ century, the need for railroad ties and bridge timbers led to widespread clearcutting of old growth ponderosa pine forests (Graham 2005). In addition to its commercial value, ponderosa pine provides critical ecological functions. These include providing a habitat for many diverse species, such as the northern goshawk, as well as protection of watersheds that supply domestic water (Long & Smith 2000; Reynolds et al. 1992; Robichaud 2005; Thomas 1979). The structural heterogeneity of these forests also supports biodiversity and ecosystem resilience.

Ponderosa pine forests are also highly valued for recreation and aesthetic enjoyment. Their understories, tall canopies, and scenic landscapes make them popular destinations for camping, hiking, hunting, birdwatching, and nature photography. In regions like the southwestern United States and the Rocky Mountain region, ponderosa pine dominated stands are crucial to the tourism economy, particularly in national forests and parks. Furthermore, recreational access to these areas fosters public support for conservation and sustainable management practices.

Today, ponderosa pine continues to be a key source of commercial timber for construction projects. It also supports many non-commercial uses, such as wood working, fuelwood, and in some cases, Christmas tree harvesting (Raish et al. 1997) As management priorities shift toward forest restoration and fire mitigation, ponderosa pine is often the focus of thinning and prescribed burning efforts aimed at reducing fuel loads and restoring historical stand structure.

### 1.1.3 Habitat and forest type associations

Ponderosa pine is broadly tolerant, but within clear ecological limits. It occupies many forest types across western North America, thriving on warm, dry, well-drained, fire-prone sites, but suffers under dense shade and poor drainage. Ponderosa pine can grow at elevations from sea level to 10,000 ft but are typically more abundant around 4,000-8,500 ft depending on latitude (Oliver & Ryker 1990). In dry forests, common co-occurring species include quaking aspen (*Populus tremuloides* Michx.), lodgepole pine (*Pinus contorta* Dougl. ex Loud), western larch (*Larix occidentalis* Nutt.), Douglas-fir (*Pseudotsuga menziesii*), and grand fir (*Abies grandis* (Dougl. ex D. Don) Lindl.) or white fir (*Abies concolor* (Gord. & Glend.) Lindl. ex Hildebr.) (Cooper et al. 1991; Graham 2005). In wetter forests, the ponderosa pine tends to grow on south

facing slopes or in areas with less competition. Common species that co-occur include western redcedar (*Thuja plicata* Donn ex D. Don) and other shade-tolerant species (Daubenmire & Daubenmire 1968). Because ponderosa pine can associate with a combination of different species, a variety of forest compositions with ponderosa occur both in dry and moist forest types (Graham 2005).

### 1.1.4 Increase in ponderosa pine dieback

In recent decades, a notable increase in dieback and mortality in ponderosa pine stands have been observed across its native range. This trend has been attributed to prolonged drought conditions. An example of this occurred between 1998 and 2004 in the Jemez Mountains of New Mexico; A severe drought that lasted six years resulted in significant overstory mortality, with unthinned stands experiencing far higher losses compared to thinned areas (Oswald et al. 2016). Similarly, regional studies have shown that repeated or prolonged drought events reduce tree growth, weaken physiological resistance, and increase vulnerability to mortality even in the absence of damaging biotic agents (Kolb & McDonald, 2016).

Among abiotic stressors, drought and high temperatures are very impactful on ponderosa pine. Reduction in soil moisture and elevated vapor pressure deficits (VPD) disrupt hydraulic conductivity and suppress carbon assimilation. This can lead to progressive canopy dieback and root loss (Niinemets & Valladares 2006). Drought stress also limits stomatal conductance and photosynthetic efficiency, which reduces carbon assimilation and carbohydrate reserves available for growth and defensive processes. Previous dendrochronology studies show that low spring precipitation delays growth initiation and restrictions annual ring development, compounding the impact of prolonged summer droughts (Li et al. 2024; Szymański et al. 2021).

These climate-related stressors, however, have not remained static. The frequency, severity, and duration of drought events in the western United States have significantly increased over the past several decades (Abatzoglou & Williams 2016). Across the native range of ponderosa pine, average temperatures have risen. These shifts have led to longer fire seasons, earlier snowmelt, and prolonged summer dry periods, all of which increase water deficits in forest ecosystems (Williams et al. 2013). The combined effects of warming and drying have contributed to chronic stress that decrease tree resilience, even in a species that is typically more drought-adapted like the ponderosa pine.

As abiotic pressures intensify, they can amplify biotic threats. Drought and heat can weaken resin-based defenses in pines, increasing their vulnerability to infection (Hood 2015). Warmer, drier conditions can also accelerate pathogen development and extend windows for pathogen sporulation and dispersal (Lahlali et al. 2024). As a result, stress-associated diseases often flare following drought or injury. Under these conditions, opportunistic fungi, such as needle and canker pathogens, root rots, capitalize on weakened trees, resulting in higher infection pressure and increased disease severity.

## 1.2 Common diseases affecting ponderosa pine

### 1.2.1 Needle and canker diseases

Diplodia tip and shoot blight caused by *Diplodia sapinea*

*Diplodia sapinea*, causal agent of Diplodia tip blight (DTB), is a widespread and damaging fungal pathogen of many pine species, particularly in the western and central United States, south and central Europe, and most recently in several northern European countries (Blumenstein et al. 2021; Caballol et al. 2022). This opportunistic pathogen affects new growth

of needles, cones, and in some severe cases, branch dieback and mortality. *D. sapinea* is most associated with stressed trees that are weakened by environmental stressors, such as drought, or mechanical damages, like hail (McKee et al. 2025). Once established in the host*, D. sapinea* invades developing shoots and cones leading to reduced growth, branch deformation, and decreased reproductive output (Stanosz et al. 2001). Typical symptoms of DTB include blighting (mortality) of current growth, retention of browned needles on twigs, resin-soaked dieback, and black pycnidia visible at the base of needles or on cone scales (**Fig. 2 & 3**) (Wingfield et al. 2024).

The probability of infection by *D. sapinea* is closely related to weather and plant stress. The fungus overwinters in infected tissue or cones by producing conidia that are dispersed the following spring via rain or wind. These conidia infect new shoots and needles in spring and early summer during times of high humidity and rainfall. Young trees are mostly affected by *D. sapinea*; however, mature trees can suffer severe damage during times of high stress (Blodgett et al. 2005). Outbreaks are more prevalent in plantations or urban settings where environmental stress and competition are more common. Given the increasing frequency of drought and higher temperatures, *D. sapinea* may pose an even greater threat to ponderosa pine in the coming years.

**Figure 2.** Symptomatic branch infected with *Diplodia sapinea.* Source: https://www.forestryimages.org/browse/image/1241526


**Figure 3.** Ponderosa pinecone scales covered with *Diplodia sapinea* fruiting bodies (pycnidia). Source: https://www.invasive.org/browse/detail.cfm?imgnum=2112097

Cytospora canker caused by *Cytospora* spp.

*Cytospora* species (syn. Leucostoma) are fungal pathogens that are the causal agents of Cytospora canker. This is a widespread disease affecting many conifer species, such as spruce, pines, and fir. Infection of the host typically occurs through mechanical wounds or insect damage and allows for the fungus to colonize the cambium and sapwood. This leads to the formation of sunken cankers and branch dieback (Sinclair et al. 1987). Symptoms of infected branches include resin soaked, discolored wood beneath cankered bark, browning of the needles, and eventually branch death (**Fig. 4)** (Sinclair et al. 1987). This disease often progresses following environmental stresses, like drought, mechanical injury, nutrient deficiencies, or storm damage, which weaken the tree's defenses and facilitate fungal colonization. Frequently, the area infected by a canker exudes resin which is an attempt by the tree to compartmentalize the fungus (Kamiri & Laemmlen 1981).

Although most research on Cytospora cankers exists on spruce and fir, the disease also affects pine species, including ponderosa pine. Previous studies have confirmed that *Cytospora*

*kunzei* and closely related species can infect the ponderosa pine and similar pine. *Cytospora* sp. was isolated from ponderosa pine sapwood, causing characteristic brown stain and cankering that is consistent with *C. kunzei* infection (Rogers & Noskowiak 1976). Infection in trees is typically initiated by drought stress or injury, resulting in branch girdling, resin flow, and dieback (Waterman 1955). In landscapes where ponderosa pine forests experience drought, physical damage, or other stressors, Cytospora infections can become prevalent (Schoeneweiss 1983; Kamiri & Laemmlen 1981; Guyon et al. 1996; Dudley et al. 2020).

**Figure 4.** Resin covered branch canker caused by Cytospora on blue spruce (*Picea pungens*) Source: https://www.invasive.org/browse/detail.cfm?imgnum=5055024

*Sydowia polyspora*

*Sydowia polyspora* (Bref. & Tavel) E. Müller (anamorph: *Hormonema dematioides* Lagerb. & Melin) is a common fungal species often found as an endophyte on conifers (Sieber-Canavesi & Sieber 1993). Across Europe, reports over the past decade show increasing detection linked with current-season needle necrosis (Talgø et al. 2010; Tinivella et al. 2014; Silva et al. 2020). Under favorable conditions to the host, it lives harmlessly within needle tissue. However,

when the host becomes stressed from abiotic or other biotic factors, the fungus may switch to a pathogen (Ridout & Newcombe 2018; Talgø et al. 2010). Additionally, *S. polyspora* can function as a pre-emergent seed pathogen on ponderosa pine with trials reducing emergence by up to 30% (Ridout & Newcombe 2018). Common symptoms of infection include yellow bands or spots on needles that turn reddish-brown during the growing season (**Fig. 5)**. In many *Pinus* spp., tip dieback can occur along with chlorotic lesion dispersed randomly throughout healthy needles (Beram and Demiröz 2024).

Although studies on *S. polyspora* in ponderosa pine are limited, observations from forests in western North America suggest the fungus behaves similarly. During periods of prolonged drought or after defoliation by insects, ponderosa pines exhibit needle chlorosis, reddening, and premature needle drop consistent with *S. polyspora* infection (Ridout & Newcombe 2018). Given the ecological and economic importance of ponderosa pine across its range, early detection of these symptoms and management practices, such as thinning to reduce stand stress and monitoring tree vigor, are essential to prevent localized outbreaks and maintain stand health.

**Figure 5.** Red banding on current growth needles on *Abies* sp. Source: https://pnwhandbooks.org/plantdisease/host-disease/fir-true-abies-spp-current-season-needle-necrosis

Elytroderma needle cast caused by *Elytroderma deformans*

Elytroderma needle cast is an important needle pathogen in pine species throughout western North America caused by the fungus, *Elytroderma deformans* (Smirnova et al. 2021). Across its native range in western North America, it is recognized as one of the most important foliage diseases of ponderosa pine. Unlike many needle pathogens, *E. deformans* is not confined to the foliage and eventually spreads into the shoots and branches. Repeated infections leads to reddening, or "flagging", of branches, formations of dense witches' brooms, and casting of needles (**Fig. 6)** (Childs, Shea, & Stewart 1971). Infection occurs in spring under cool, moist conditions when wind-borne spores land on current growth. The spores germinate on the needles and penetrate the epidermis. By mid-to late- summer, a long, black, football-shaped fruiting body (hysterothecia) forms on necrotic needles from which new spores are released (**Fig. 7)** (Childs 1968; Childs, Shea, & Stewart 1971).

Previous surveys of elytroderma needle cast have shown how damaging this disease can be in ponderosa pine stands. For example, mortality events in Oregon and Washington have been recorded in thousands of acres, with infected stands experiencing significant reduction in growth and branch kill (Childs 1968). Additionally, a study in 1981 on a closely related species, Jeffery pine (*Pinus jeffreyi*), showed that infected trees have a significant reduction in radial growth, live-crown ratios, and increase in tree mortality following several years of repeated outbreaks (Scharpf 1981). These compounding effects impact the overall health of individual trees and the

entire stand, making heavily infected populations more vulnerable to secondary pest and pathogen attacks such as bark beetles and root-diseases (Childs, 1968; Scharpf, 1981).

**Figure 6.** Flagged branches and brooms formed on ponderosa pine by *Elytroderma deformans.* Source: https://pnwhandbooks.org/plantdisease/host-disease/pine-pinus-spp-elytroderma-needle-cast


**Figure 7.** *Elytroderma deformans* fruiting bodies on pine needles. Source: https://www.insectimages.org/browse/image/1241507

Dothistroma needle blight caused by caused by *Dothistroma septosporum* and *Dothistroma pini*

Dothistroma needle blight (DNB) is a globally important foliar disease of pine trees caused by two closely related species of fungi, *Dothistroma septosporum and Dothistroma pini*. Typical symptoms include red banding on needles, premature defoliation, growth loss, and sometimes mortality (**Fig. 8)** (Bradshaw 2004; Bulman et al. 2016). Incidence and severity are

tightly linked to weather, especially in areas with increased precipitation leading to prolonged needle wetness and elevated summer temperatures (Woods et al. 2005; Woods et al. 2016). DNB has a wide host and geographical range, consisting of many *Pinus* species on almost every continent (Drenkhan et al. 2016). Management of DNB emphasizes avoiding planting susceptible hosts on sites with a higher precipitation regime and higher temperatures, mechanical management such as thinning or pruning of branches to reduce humidity and applying copper fungicides during extreme outbreaks (Bulman et al. 2016).

In western North America, native pines species such as ponderosa pine and lodgepole pine are highly susceptible to DNB. In British Columbia, major outbreaks have been reported on lodgepole pine in stands where uncharacteristically wet summers occur (Woods et al., 2005). Ponderosa pine has higher vulnerability in conducive climates. In New Zealand, extreme pressure from DNB resulted in the abandonment of ponderosa pine as a plantations species (Bulman et al. 2016). For ponderosa pine in the interior west, risk is highest on sites or years with prolonged needle wetness and in stands with dense canopies. Management for DNB in ponderosa stands is very similar to other *Pinus* species focusing on site/species choice, wider spacing, thinning to improve airflow, and sanitations in severely affected pockets. Copper sprays are also an effective option but are seldom used in North American forests (Woods et al., 2016; Bulman et al., 2016; Watt et al., 2011).


**Figure 8.** Red banding caused by Dothistroma needle blight on pine. Source: https://www.invasive.org/browse/detail.cfm?imgnum=2251050

1.2.2 Rust pathogens

Western gall rust caused by *Endocronartium harknessii*

*Endocronartium harknessii* (J.P. Moore), commonly called western gall rust, is an autoecious rust fungus that infects stems and lateral shoots of many species of two- and three-needle pines. The most obvious signs of western gall rust are the formation of perennial, woody galls on branches or stems that can weaken and girdle the tree (Old 1981) (**Fig. 9**). Infection of pines is initiated in the spring when aeciospores are released from mature galls of the previous year land on expanding shoots or stems of susceptible hosts (Old 1981; Peterson 1961). The spores germinate and penetrate the cortex through stomata or directly through the epidermis. By early summer, spermogonia (formerly pycnia) develop under the surface of newly formed galls (Old 1981). Later in the growing season, finger-like aecia form on the gall surface and extrude orange-yellow aeciospores that disperse locally to infect adjacent shoots (Peterson 1961). Each gall then grows incrementally every year, expanding in size up to 10 cm. The result of repeated infection and gall formation can result in deformation, girdling, reduced growth, and increased susceptibility to secondary pests or pathogens (Old 1981; Peterson 1961). Characteristic of its

autoecious, endocyclic lifecycle each gall produces successive cycles of spore producing structures without requiring an alternative host (Hiratsuka 1969).

Although western gall rust is more commonly found on lodgepole and radiata pines, it poses a significant threat in regenerating ponderosa pine stands. Egan and Merrill (1997) assessed several ponderosa pines planted on a coal strip-mine spoil bank and found rust infection levels ranging from 5% to 95% among seed sources and with mortality due to gall rust reaching up to 58% in some areas. In these plantings, trees with multiple galls exhibited reduced growth and developed deformities that diminished lumber quality (Egan & Merrill 1997). Although many of the previous studies on western gall rust have emphasized lodgepole and radiate pines, management recommendations for ponderosa pine parallel those for other pines.


**Figure 8.** *Pinus* spp. branch with sporulating gall. Source: https://www.invasive.org/browse/detail.cfm?imgnum=1241722

Comandra blister rust caused by *Cronartium comandrae*

Comandra blister rust, caused by the heteroecious rust fungus *Cronartium comandrae,* is a common disease of hard pines. It requires two hosts to complete its life cycle, *Comandra pallida* (bastard toadflax) (telial host) and two- and three-needle pines (aecial hosts). In late

spring or after summer rains, basidiospores produced on infected bastard toadflax leaves are dispersed via wind and land on nearby pine needles. Spores germinate and penetrate needle tissue through stomata or directly through the epidermis (Dolezal & Tainter 1979). During the first year of infection, the fungus establishes in the cortex and produces spermogonia that ooze sporidia. The following spring, bright orange aecia erupt as blisters on infected tissue, killing underlying sapwood and releasing aeciospores that infect new shoots (Krebill 1968) (**Fig. 10)**. Over successive years, aecial cankers expand girdling branches, causing top-kill, and disrupting water transport. Geils and Jacobi (1993) showed that annual volume increments dropped 32% of trees with severe cankers on the main stem and trees that experienced top kill from girdling ceased meaningful growth (Geils & Jacobi 1993).

Although many of the previous studies have been conducted on lodgepole pine, the ponderosa pine is also recognized as an aecial host. Childs (1968) included ponderosa pine among primary host susceptible to basidiospore infection and noted similar blister formation and girdling of the cortex on saplings and mature trees. Early surveys in the Rocky Mountain region show that comandra blister rust cankers on ponderosa pine occur in about 40% of surveyed understory regeneration (Krebill 1968). Given these parallels, management recommendations developed for lodgepole pine should be used for ponderosa stands as well. Tactics such as reducing alternate-host density, pruning infected branches before aeciospore release, and thinning stands to enhance host vigor are directly applicable to maintaining the health and productivity of ponderosa pine stands (Childs 1968).


**Figure 10.** Aecia erupting as blister on main stem of lodgepole pine (*Pinus contorta* Dougl. ex Loud.), releasing aeciospores. Source: https://www.invasive.org/browse/detail.cfm?imgnum=5008067

1.2.3 Vascular diseases

Pine wilt disease

Pine wilt disease (PWD) is a lethal vascular wilt caused by the pine wood nematode (PWN), *Bursaphelenchus xylophilus* (Steiner and Buhrer 1934) Nickle 1970. This disease poses a major threat to pine forests globally as PWD is difficult to treat once a tree is infected. The virulence of PWN varies depending on the health of the host. Abiotic stressors, secondary infections, and species susceptibility can determine whether the host survives an attack from the nematode (Atkins et al. 2021). Roughly 3 weeks post-infection, needles typically wilt and fade from green to straw-colored (**Fig. 11**). This symptom expression commonly coincides with summertime heat and limited precipitation (Rutherford & Webster, 1987). A combination of tactics should be used for management of PWN. Injecting trees with nematicides, removing

diseased trees and replacing them with a non-susceptible host, and control of the vector are a few ways to limit the spread of PWN (Kamata 2008).

The PWN is believed to be native to North America with a distribution throughout most forested areas in the United States and Canada (Rutherford and Webster 1987). Additionally, PWN occurs frequently in areas where exotic pines species have been planted in historically unforested or urban areas (Malek and Appleby 1984). Out of the total number of pine species commonly found planted in North America, seventeen have been found to be susceptible to PWN; The five most susceptible pines species are Jack pine (*Pinus banksiana*), shortleaf pine (*Pinus echinata*), Monterey pine (*Pinus radiata*), sugar pine (*Pinus lambertiana*), and Scots pine (*Pinus sylvestris*) (Wingfield et al. 1982). Historically, it was thought that native pines were highly resistant to or tolerant of the PWN (Dropkin 1981). However, reports in Colorado show contradictory evidence that native pines are not as resistant as once thought with the first report of pine wilt disease affecting ponderosa pine (*P. ponderosa* Douglas ex. Lawson) in 2016 (Atkins et al. 2021).

**Figure 11.** Stand of *Pinus* sp. infected with PWN with changes in needle color from green to straw colored. Source: https://www.agriculture.gov.au/biosecurity-trade/import/arrival/pests/pine-wilt-nematode

Blue stain fungi as pathogens

Blue stain fungi are comprised of a polyphyletic assemblage of primarily ascomycetes in genera such as *Ophiostoma*, *Ceratocystis*, *Ceratocystiopsis*, *Grosmannia,* and *Leptographium.* These sap staining fungi can be vectored by a variety of insects, commonly by bark beetles such as *Dendroctonus* (including western pine beetle on ponderosa pine) and also *Ips* and *Tomicus* species, with phoretic *Tarsonemus* mites serving as important secondary vectors. It has also been recovered from galleries of pine sawyers (Ballard et al. 1984; Chang et al. 2020; Davydenko et al. 2017; Jankowiak 2007; Jankowiak et al. 2007). In host sapwood it causes blue-stain discoloration characterized by hyphal colonization of ray parenchyma cells (Ballard et al. 1984) (**Fig. 12)**. While they are often considered secondary invaders to stressed, dying, or already dead trees, blue stain fungi have been shown to sometimes act as plant pathogens. Plant pathogenicity trials have shown certain species such as *Grosmannia clavigera* and *Ophiostoma ips,* can invade living sapwood, occlude vessels, and reduce hydraulic conductivity when introduced in sufficient inoculum (Krokene & Solheim 1998; Rane & Tattar 1987).

Previous studies show that blue stain fungi can act pathogenically in ponderosa pine. In seedling assays, *Ceratocystis minor* (syn. *Ophiostoma minus*) and *Ceratocystis ips* produced necrotic lesions and significant mortality, independent of their bark beetle vectors (Owen et al. 1987). In mature trees, stem inoculations with *O. minus* resulted in measurable lesions and rapid sapwood occlusion, indicating hydraulic impairment of the xylem (Parmeter et al. 1992). Collectively, these findings support *O. minus* as an opportunistic vascular pathogen in ponderosa

pine, capable of causing tissue injury and reducing conductivity, especially in seedlings or under host stress.


**Figure 12.** Blue stain fungus staining ray parenchyma cells in the wedge shape. Source: https://foresightcac.com/article/low-water-blue-stain-free-wood-challenge

Black stain root disease caused by *Leptographium wageneri*

Black stain root disease (BSRD) is an extremely damaging root disease in western North American conifers. Caused by the ophiostomatoid fungus, *Leptographium wageneri*, its most characteristic symptom is dark sapwood staining that progresses longitudinally through xylem vessels (Hessburg et al. 1995) (**Fig. 13)**. Recent phylogenetic work revealed that former varieties of Leptographium wageneri should be recognized as separate species. Each species specializes on different hosts: *L. ponderosum* on ponderosa/Jeffrey/lodgepole pines, *L. pseudotsugae* on Douglas-fir, and *L. wageneri* sensu stricto on pinyon pines (Choi et al. 2023). First observed on Jeffery pine in 1938 and on ponderosa pine in 1941 (Wagener et al. 1961), it was found that the fungus is vectored by root-weevils and secondary bark beetles into fine roots (Hessburg et al. 1995). Once spread to a viable host, it colonizes cortical and xylem tissues, producing the characteristic black staining that impedes water and nutrient transport (Joseph et al. 1998). Centers for BSRD infected trees are often associated with soil disturbances typically associated

with logging or thinning of stands. Trees in these areas succumb to BSRD in mass and can form patches that span hundreds of hectares (Harrington et al. 1983).

In ponderosa pine, BSRD significantly reduces vigor by lowering the tree's ability to move water and hinders growth. Joseph et al. (1998) showed the hydraulic impact of BSRD on infected ponderosa pine. They reported that infected trees dropped conductivity 4.6% from healthy controls due to resin filled xylem and embolisms (Joseph et al., 1998). Additionally, field surveys in southwestern Colorado reported infection incidence exceeding 60% in drought stresses stands (Kearns & Jacobi, 2005). This highlights the importance of maintaining stand vigor through careful thinning and sanitation while minimizing soil disturbance.

**Figure 13.** Black xylem staining caused by black stain root disease. Source: https://forestpathology.org/root-diseases/black-stain/

1.2.4 Other major diseases

Southwestern dwarf mistletoe (*Arceuthobium vaginatum* ssp. *cryptopodum*)

Southwestern dwarf mistletoe (*Arceuthobium vaginatum* ssp. *cryptopodum*) is an obligate hemiparasitic flowering plant native to coniferous forests on the southwestern United State and northern Mexico (Worrall 2013). It infests roughly 9 million hectares across Arizona, New Mexico, Colorado, Utah. Texas and Chihuahua (Hoffman et al. 2007). The parasite attacks its host by penetrating the xylem and phloem via a network of haustoria, taking water, carbohydrates, and nutrients (Worrall 2013). Dispersal of the pathogen typically occurs when sticky seeds are forcibly ejected and spread via the air or attach to birds or animals (Mathiasen et al. 2008). The most characteristic signs are the formation of an aerial witches' broom on the branches (**Fig. 14)**. These shoots appear three to five years after infection and vary in color from orange to reddish brown to almost black (Worrall 2013). Severe infections of dwarf mistletoe are a chronic driver of growth loss, crown deformation, and increased fire potential (Worrall 2013; Hoffman et al. 2007).

In ponderosa pine substantial damage and growth reductions have been shown in smaller trees infected with dwarf mistletoe (Roth 1971). Dendroecological studies in mature ponderosa pine found that trees infected by a closely related species of dwarf mistletoe, western dwarf mistletoe (*A. campylopodum*), exhibited heightened sensitivity to climate change, suggesting that infected trees react differently to variability in climate. At the stand scale, severely infested ponderosa pine stands in northern Arizona had significantly lower live tree density, higher snag density, and greater surface fuel loadings than uninfected stands, indicating greater crown-fire potential (Hoffman et al. 2007). Management studies in the Grand Canyon region further show that first-year mortality following prescribed burns increases in trees with high dwarf mistletoe ratings, and scorch-pruning can help lower stand level infections (Harrington & Hawksworth

1990; Worrall 2013). Together, these findings show that in ponderosa pine forests, southwestern dwarf mistletoe not only suppresses growth but also interacts with fire to increase mortality risk.


**Figure 14.** Mistletoe shoots concentrated near the original infection site forming witches' brooms on ponderosa pine. Source: https://www.insectimages.org/browse/image/1949042

## 1.3 Effects of climate change on native plant pathogens

### 1.3.1 Effect of climate change on plant pathogens

In recent decades, changing climates have significantly influenced the behavior of fungal plant pathogens. Shifting environmental conditions such as rising temperatures, altered precipitation patterns, and increased humidity have allowed fungal pathogens to expand outside of their normal geographic range and increased their severity. Previous reviews have indicated that warming can intensify epidemics by accelerating pathogen development, increasing potential inoculum, and shifting-host pathogen windows of interaction (Garrett et al. 2006; Chakraborty & Newton 2011). Not only does this climatic shift increase the abundance and aggressiveness of fungal plant pathogens but also facilitates the geographic expansion and

emergence of novel diseases (Bebber et al. 2013). In addition, warming trends have extended the annual window for which pathogens can infect their hosts. This has resulted in additional life cycles for the pathogen per season (Chakraborty & Newton 2011; Christidis et al. 2007). Even pathogens that have not extended outside of their normal range can become more severe under prolonged growing seasons and higher humidity, allowing for more severe outbreaks and persistent disease pressure (Romero et al. 2022). For example, in the late 1900s, an unprecedented epidemic of Dothistroma needle blight in northwest British Columbia infected more than 40,000 hectares of Lodgepole pine. Infected stands saw severe defoliation due to more frequent summer rain events (Woods et al. 2005; Woods et al. 2016). Analyses show outbreaks have become more frequent and extensive in recent decades and are statistically tied to weather patterns, indicating a climate-driven shift in both distribution and intensity (Welsh et al., 2009; Welsh et al. 2014). While the complexities of climate change and the effects on plant pathogens are still being studied, collectively the evidence shows that climate change can intensify existing diseases, shift distributions, or for novel pathogens to be introduced (Pautasso et al. 2012; McDonald & Stukenbrock 2016).

### 1.3.2 Effects of climate change on plant-pathogen interactions

Plants and associated pathogens engage in what is known as a molecular “arms race”. This is driven by the ability of plants to perceive microbial signatures of pathogens and counterstrategies they evolve to avoid detection. The plants’ first layer of defense is called pattern triggered immunity (PTI). It is a general defense system that is active when host pattern recognition receptors (PRRs) recognize microbe associated molecular patterns (MAMPs) such as fungal chitin (Boller & Félix 2009). A cascade of downstream responses such as, ion fluxes, reactive oxygen species production, and transcriptional reprogramming, are triggered that help

restrict the movement of the pathogen further into the plant. A method to avoid detection that successful pathogens have developed is the ability to secrete effector proteins that block the activation of PTI and enable it to colonize the host (Jones & Dangl 2006).

When the effectors are recognized by the plant's resistance proteins, a stronger, localized and specific defense is triggered. This is known as effector triggered immunity (ETI) uses a combination of tactics to prevent the spread of the pathogen including hypersensitive response, programmed cells death, and signaling that primes other tissues (Dodds & Rathjen 2010). Both PTI and ETI rely on signaling networks that use salicylic acid (SA), jasmonic acid (JA) and ethylene pathways to tailor a defense to specific type of attack (biotrophic, necrotrophic, or environmental (Cui, Tsuda, & Parker 2015).

Climate change factors such as elevated $CO_2$ concentrations, higher temperatures, and altered precipitation regimes can affect host plant physiology and defensive capabilities. Typically, biomass production is promoted by elevated $CO_2$, however, it can dilute concentrations of defensive secondary metabolites (Walters & Heil 2007). Drought and heat stress can undermine both PTI and ETI by disrupting hormone signaling, thus making plants more susceptible to opportunistic pathogens (Ramegowda & Senthil-Kumar 2015). Additionally, changes in phenology caused by warmer and longer growing seasons can throw off host pathogen interaction. This can lead to novel disease outbreaks or shifts in epidemic timing (Garrett et al. 2006).

### 1.3.4 Pathogens directly affected by climate (Primary pathogens)

Regardless of host condition, some pathogens can cause disease if the proper environmental requirements, such as temperature, humidity, and moisture, are met. These are

known as primary pathogens, whereas secondary pathogens mainly exploit stressed hosts (Kolb & McDonald 2016). The life cycles of primary pathogens are highly sensitive to these environmental conditions and affect their ability to infect spread and reproduce (Harvell et al. 2002). For example, foliar fungal pathogens, like *Dothistroma septosporum*, responsible for Dothistroma needle blight in pines, have started to exhibit increased incidence in regions with increasing average rainfall and temperature in the spring and summer. Before the 1990s, Dothistroma needle blight was considered a minor pathogen confined to the southern hemisphere. In countries such as the United Kingdom, France, and Canada the increases in distribution and intensity of Dothistroma needle blight can be attributed to increased temperature, including overnight minimum; and increase in period of needle wetness caused by higher levels of precipitation (Sturrock et al. 2011). Similarly, Swiss needle cast (*Phaeocryptopus gaeumannii)* is a major foliar pathogen of Douglas-fir along the coast of Oregon. In this area, winter temperatures and spring precipitation have increased 0.2◦C–0.4◦C and 0.7–1.5 cm, respectively, every decade since 1970 (Stone et al. 2008). In the early 1990s, an epidemic of this disease broke out in the Oregon coast (Hansen et al. 2000) and was found to be positively correlated with the accumulation of higher degree days in winter and duration of leaf wetness in spring and fall (Manter et al. 2005).

### 1.3.5 Pathogens indirectly affected by climate (secondary pathogens)

Pathogens indirectly affected by climate change rely on host stress to cause disease. While they still rely on temperature and moisture to infect, spread, and reproduce, factors that stress their host are much more critical to successful colonization. Factors such as environmental change, primary pathogens, or insects can cause the host to become stressed and lower its ability to fight off these secondary pathogens (Lonsdale 2002; Desprez-Loustau et al. 2006). An

example of this are species of *Armillaria* that cause Armillaria root disease. These are soil-borne pathogens that cause root disease in natural and managed stands around the world (Kile et al. 1991). The hosts are primarily conifers but occasionally hardwoods, woody shrubs, and herbaceous plants can be infected. Disease severity is tightly linked to host condition. Short-term decreases in precipitation and increases in temperature can predispose stands to higher Armillaria damage (Filip 2024). Since *Armillaria* species can thrive at a wide range of temperatures ranging from 10 to 31°C (Rishbeth 1978), the incidence and severity of this disease is likely to increase (Shaw & Kile 1991; La Porta et al. 2008; Klopfenstein et al. 2009). As temperatures increase and precipitation decreases in areas with Armillaria root disease, hosts will become more stressed making them more susceptible (Kim et al. 2021). Similar to Armillaria root disease, canker pathogens are likely to reach epidemic levels when their host is weakened by heat, drought stress, or other biotic agents (Schoeneweiss 1975, 1981) but unlike *Armillaria* species, many of these canker fungi can persist in the host asymptomatically and then shift to pathogenic growth when host stress is increased by drought, heat, or wounding (Desprez-Loustau et al. 2006; Slippers & Wingfield 2007). Representative species include *Biscogniauxia mediterranea* which causes charcoal canker of oaks and is frequently recovered as an endophyte from healthy oak tissue. Outbreaks have been found to be associated with drought (Linaldeddu et al. 2011; Moricca et al. 2016); *Botryosphaeria dothidea,* known as a latent pathogen that transitions from endophyte to canker agent under water stress (Marsberg et al. 2017; Slippers & Wingfield 2007); *Diplodia sapinea,* which resides in asymptomatic pine tissues and rapidly causes disease under water stress (Stanosz et al. 2001; Wingfield et al. 2024); and Cytospora canker agents such as *Cytospora chrysosperma* and *Cytospora kunzei*, which commonly inhabit woody tissues and produce cankers when trees and injured or drought stressed (Schoeneweiss 1983)

### 1.3.6 Decline diseases

Forest declines are a disease complex resulting from interactions with multiple stressors, such as abiotic or biotic agents. Manion (1981) classifies these factors as predisposing, inciting, or contributing based on the order of occurrence and length of effect. These syndromes are often characterized by a progressive loss in tree or stand health and vigor without obvious evidence of a single identifiable causal agent. Decline diseases typically emerge when trees, weakened by long-term environmental stress, become more susceptible to secondary pests and opportunistic pathogens (Ciesla and Donaubauer 1994). A historical example of a decline disease is beech decline in Europe. It was found that the decline was likely due to long-term stress followed by infection by *Nectria* fungi and bark beetles (Houston 1987). Similarly, sudden aspen decline in North America was linked to drought-induced stress and a combination of secondary fungi and insects. The complex of abiotic stress followed by attack by biotic agents on weakened trees led to rapid canopy dieback and mortality on a landscape scale (Worrall et al. 2008, 2010).

Modern forest decline diseases are often viewed through the scope of climate change, which can exacerbate predisposing and inciting factors. For example, in yellow cedar (*Callitropsis nootkatensis*), climate-driven reduction in snowpack has resulted in root damage from freezing. This predisposes the trees to decline without any involvement from biotic factors (Hennon et al. 2012). Cases like yellow cedar decline demonstrate how climate-altered abiotic conditions can be the primary driver of forest declines, rather than a pest or pathogen.

### 1.3.7 Management strategies as climate changes

Climate change influences forest pathogens directly, indirectly, and through interactions with stressors. Therefore, elevated tree mortality should be expected and planned for. Management must be comprehensive and context-specific, despite uncertainties in biological

responses and public acceptance (Stenlid et al. 2011). While many forests and urban trees will face altered climates without active intervention, socially and economically value landscapes can be managed to minimize dieback and mortality (Spittlehouse 2005, 2009). The central task is to adapt management tactics now, because the current approaches for management will not protect forests under changing climate conditions (Moore and Allard 2008). Already, climate change is reshaping silviculture and policy, from growth and yield forecast, timber supply modeling to wildlife habitat strategies and carbon-nutrient-water cycling (Graham et al. 1990; Woods et al 2010). Increasing stand and landscape level diversity is a practical approach. Mixing conifer and broadleaf species in regenerating forests spreads climate risk, as conifers show greater plasticity to drought and deciduous species enhance water return to the environment Hasselquist et al. 2010; Tor-ngern et al. 2018). Ultimately, land managers should prioritize species likely to persist under projected conditions and tailor planting to site-specific forecasts.

A practical management framework focuses on four linked components: monitoring, forecasting, planning, and mitigation (Sturrock et al. 2011). Implementations will vary with forest type, stewardship goals, and human and financial capacity. Regardless, a first step is identifying places where increasing tree species diversity is both feasible and beneficial.

Effective monitoring of forest diseases is critical for adapting appropriate management strategies, particularly as climate change and human activities increase the risk of emerging pathogens. Coordinated programs should track disease spatial patterns relative to host ranges and interannual weather, use trained observers, establish permanent observation plots, and conduct field and remote sensing surveys (Sturrock 2012). Cross-boundary collaboration among federal, state, and local partners can help improve early detection and rapid response (Moore & Allard 2008). Additionally, urban forests and nurseries warrant special attention as sentinels for invasive

species. Finally, clear communication of results to scientists, policymakers, and the public is essential to effective monitoring. Framing the impacts of tree disease, tailored to the needs of each audience, can help gain public and political support for proactive management (Stenlid et al. 2011).

Since changes in global environmental conditions can be unpredictable, forest professionals cannot rely on historical observations and experiences to predict and plan for the future. Instead, managers need integrated models of climate, vegetation, and disturbance to anticipate trajectories (Sturrock et al., 2011). For example, bioclimatic envelope models correlate species distributions with climate future niches for hosts and pathogens (Pearson & Dawson 2003). Modeling the climate niches of pathogens in conjunction with host tree responses to climate conditions can improve the accuracy of disease outcome predictions. While uncertainty remains, forecasting clarifies directions and bounds of change.

Management plans should be durable, well-funded, regularly updated, and policy aligned in order to meet emerging risks (Woods et al. 2010; Sturrock et al. 2011). Plans must address interactions with other disturbances, epically wildfire, which is amplified by climate-driven insect and disease mortality (Bergeron & Leduc 1998; Lertzman & Fall 1998; Kliejunas et al. 2009). Risk and hazard rating systems still remain valuable and should be revised to incorporate future climate scenarios. Many major pathogens still lack a robust rating system that incorporates future climate scenarios and as climate change reshapes how forest diseases behave and how they are distributed, forest planning must adapt (Woods et al. 2010). Despite existing global regulations for plant movement and biosecurity, gaps in enforcement still remain, leading to higher risks of outbreaks. Addressing such gaps through legislation, improved enforcement, and international coordination is vital. (Brasier, 2008).

The overall goal of forest management is resilience through sustained function despite disturbances (Thompson et al. 2009). Diversifying species, age classes, and genotypes reduces the risk of failure. Another potentially effective and cost-efficient strategy for promoting species persistence under climate change is the use of assisted migration. This practice involves the intentional relocation of species to areas where future climatic conditions are predicted to be more suitable. It can be conducted under three levels of intensity: (1) assisted population migration (within the species' current range), (2) assisted range expansion (just beyond current range limits), and (3) assisted long distance migration (into entirely new areas) (Winder et al. 2011). While assisted migration can help conserve species affected by climate change, it must be implemented with caution. Translocating trees may transmit unknown pathogens to new environments or lack of resistance to local diseases may cause mortality (Wallis et al. 2010). Breeding for disease resistance, drought tolerance, and plasticity, while maintaining genetic diversity, can raise adaptive capacity (Yanchuk et al., 1988; Liu & Ekramoddoullah, 2003; Cruickshank et al., 2010). In high-value settings, such as nurseries & heritage trees, targeted chemical controls may offer short-term protection where disease pressure is high (Prospero et al. 2021; Souder and Strimbu 2021). Across all tactics, iterative, model information management with continued monitoring and evaluation helps ensure effectiveness and limit unintended outcomes (Leech et al., 2011).

### 1.4 Research objectives and questions

Widespread dieback and mortality in ponderosa pine forests across the western United States has raised widespread concerns. While several stressors, such as drought, rising temperatures, increased insect and pathogen pressures, are known to impact tree health, their

interactions and contributions to recent mortality events remain unclear. Despite the economic, ecological, and societal importance of ponderosa pine, there is a lack of comprehensive, regional-scale studies that assess how biotic and abiotic stressors collectively influence the health of the species. Additionally, most existing research tends to focus on a single pathogen or localized outbreaks, rather than broader landscape scale patterns.

The overall goal of this project is to quantify dieback and mortality of ponderosa pine across multiple western states and assess the correlation between biotic and abiotic factors and observed declines. Understanding these relationships is essential for identifying key drivers of tree stress, anticipating future disease risk, and adapting appropriate management practices under changing climate conditions. To address the knowledge gaps, this research focuses on the following questions: (1) What are the biotic and abiotic agents contributing to dieback and mortality? (2) Is the prevalence and severity of disease we observe causing/contributing to significant dieback and/or mortality of ponderosa pine? (3) Are the pathogens observed on ponderosa pine the same throughout the western USA or do they differ across geographical regions? By integrating field-based assessments with climatic data and pathogen diagnostics, this study aims to generate valuable insights into the causes of ponderosa pine decline. The findings will contribute to a growing body of knowledge on forest health and help guide future management strategies that enhance resilience and reduce the risk of mortality under future climate scenarios.

REFERENCES


Abatzoglou, J. T., & Williams, A. P. (2016). Impact of anthropogenic climate change on wildfire across western US forests. *Proceedings of the National Academy of Sciences, 113*(42), 11770–11775. https://doi.org/10.1073/pnas.1607171113

Atkins, D. H., Davis, T. S., & Stewart, J. E. (2020). Pine Wilt Disease. *Colorado State University Extension. Fact Sheet 2.915.*

Atkins, D. H., Davis, T. S., & Stewart, J. E. (2021). Probability of occurrence and phenology of pine wilt disease transmission by insect vectors in the Rocky Mountains. *Ecological Entomology, 46*(4), 744–754.

Ballard, R. G., Walsh, M. A., & Cole, W. E. (1984). The penetration and growth of blue-stain fungi in the sapwood of lodgepole pine attacked by mountain pine beetle. *Canadian Journal of Botany, 62*(9), 1724–1729. https://doi.org/10.1139/b84-233

Bebber, D. P., Ramotowski, M. A. T., & Gurr, S. J. (2013). Crop pests and pathogens move polewards in a warming world. *Nature Climate Change, 3*(11), 985–988. https://doi.org/10.1038/nclimate1990

Beram, M., & Demiröz, M. (2024). Detection of *Sydowia polyspora* in pine species in Turkey and implications for forest health. *Turkish Journal of Forestry, 25*(1), 11–20.

Bergeron, Y., & Leduc, A. (1998). Relationships between change in fire frequency and vegetation dynamics in the boreal forest of northwestern Quebec. *Plant Ecology, 134*(1), 39–52.

Blodgett, J. T., Kruger, E. L., & Stanosz, G. R. (2005). *Sphaeropsis sapinea* and water stress in a red pine plantation in central Wisconsin. *Phytopathology, 95*(3), 388–396.

Blumenstein, K., Bußkamp, J., Langer, G. J., Langer, E. J., & Terhonen, E. (2021). The Diplodia Tip Blight Pathogen *Sphaeropsis sapinea* Is the Most Common Fungus in Scots Pines' Mycobiome, Irrespective of Health Status—A Case Study from Germany. *Journal of Fungi, 7*, 607. https://doi.org/10.3390/jof7080607

Boller, T., & Félix, G. (2009). A renaissance of elicitors: perception of microbe-associated molecular patterns and danger signals by pattern-recognition receptors. *Annual Review of Plant Biology, 60*, 379–406.

Bradshaw, R. E. (2004). Dothistroma (red-band) needle blight of pines and the dothistromin

toxin: A review. *Forest Pathology, 34*(3), 163–185. https://doi.org/10.1111/j.1439-0329.2004.00356.x

Brasier, C. M. (2008). The biosecurity threat to the UK and global environment from international trade in plants. *Plant Pathology, 57*(5), 792–808.

Bulman, L. S., Bradshaw, R. E., Fraser, S., Martín-García, J., Barnes, I., Musolin, D. L., La Porta, N., Woods, A., Diez-Casero, J. J., Koltay, A., Drenkhan, R., Ahumada, R., Poljakovic Pajnik, L., Queloz, V., Piškur, B., Doğmuş-Lehtijärvi, H. T., Chira, D., Tomešová-Haataja, V., Georgieva, M., … Tubby, K. (2016). A worldwide perspective on the management and control of Dothistroma needle blight. *Forest Pathology, 46*(5), 472–488. https://doi.org/10.1111/efp.12305

Caballol, M., Serradó, F., Barnes, I., Camarero, J. J., Valeriano, C., Colangelo, M., & Oliva, J. (2022). Tree mortality caused by Diplodia shoot blight on *Pinus* spp.: An emergent forest disease in Europe. *Forest Pathology, 52*(6), e12810.

Chakraborty, S., & Newton, A. C. (2011). Climate change, plant diseases and food security: An overview. *Plant Pathology, 60*(1), 2–14. https://doi.org/10.1111/j.1365-3059.2010.02411.x

Chang, R., Duong, T. A., Taerum, S. J., Wingfield, M. J., Zhou, X., & de Beer, Z. W. (2020). Ophiostomatoid fungi associated with mites phoretic on bark beetles in Qinghai, China. *IMA Fungus, 11*, 15. https://doi.org/10.1186/s43008-020-00037-9

Childs, T. W. (1968). Comandra rust damage to ponderosa pine in Oregon and Washington. *Pacific Northwest Forest and Range Experiment Station, U.S. Forest Service.*

Childs, T. (1968). Elytroderma disease of ponderosa pine: A review of current knowledge. *USDA Forest Service Research Paper PNW-69.*

Childs, T., Shea, K. R., & Stewart, R. J. (1971). Epidemiology of *Elytroderma deformans* in western forests. *Phytopathology, 61*, 1301–1305.

Choi, D., Harrington, T. C., Shaw, D. C., Stewart, J. E., Klopfenstein, N. B., Kroese, D. R., & Kim, M.-S. (2023). Phylogenetic analyses allow species-level recognition of *Leptographium wageneri* varieties that cause black stain root disease of conifers in western North America. *Frontiers in Plant Science, 14*, 1286157. https://doi.org/10.3389/fpls.2023.1286157

Christidis, N., Stott, P. A., Brown, S. J., Karoly, D. J., & Caesar, J. (2007). Human contribution to

the lengthening of the growing season. *Journal of Climate, 20*(21), 5441–5454. https://doi.org/10.1175/2007JCLI1568.1

Ciesla, W. M., & Donaubauer, E. (1994). Decline and dieback of trees and forests: A global overview. *FAO Forestry Paper No. 120.*

Cooper, S. V., Neiman, K. E., & Roberts, D. W. (1991). Forest habitat types of northern Idaho: A second approximation. *USDA Forest Service.*

Cruickshank, M. G., Morrison, D. J., & Merler, H. (2010). Resistance of interior Douglas-fir families to *Armillaria ostoyae* in British Columbia. *Canadian Journal of Forest Research, 40*(1), 155–166.

Cui, H., Tsuda, K., & Parker, J. E. (2015). Effector-triggered immunity: From pathogen perception to robust defense. *Annual Review of Plant Biology, 66*, 487–511.

Daubenmire, R., & Daubenmire, J. B. (1968). Forest vegetation of eastern Washington and northern Idaho. *USDA Forest Service.*

Davydenko, K., Vasaitis, R., & Menkis, A. (2017). Fungi associated with *Ips acuminatus* (Coleoptera: Curculionidae) in Ukraine with a special emphasis on pathogenicity of ophiostomatoid species. *European Journal of Entomology, 114*, 77–85. https://doi.org/10.14411/eje.2017.011

Desprez-Loustau, M.-L., Robin, C., Buee, M., Courtecuisse, R., Garbaye, J., Suffert, F., Sache, I., & Rizzo, D. M. (2006). The fungal dimension of biological invasions. *Trends in Ecology & Evolution, 22*(9), 472–480.

Dodds, P. N., & Rathjen, J. P. (2010). Plant immunity: Towards an integrated view of plant–pathogen interactions. *Nature Reviews Genetics, 11*(8), 539–548.

Dolezal, J. E., & Tainter, F. H. (1979). The infection process of *Cronartium comandrae* on hard pines. *Phytopathology, 69*, 176–180.

Drenkhan, R., Tomešová-Haataja, V., Fraser, S., Bradshaw, R. E., Vahalík, P., Mullett, M. S., Martín-García, J., Bulman, L. S., Wingfield, M. J., Kirisits, T., Cech, T. L., Schmitz, S., Baden, R., Tubby, K., Brown, A., Georgieva, M., Woods, A., Ahumada, R., Jankovský, L., … Barnes, I. (2016). Global geographic distribution and host range of *Dothistroma* species: A comprehensive review. *Forest Pathology, 46*(5), 408–442. https://doi.org/10.1111/efp.12290

Dropkin, V. H. (1981). *Introduction to plant nematology.* Wiley.

Dudley, M. M., Tisserat, N. A., Jacobi, W. R., Negrón, J., & Stewart, J. E. (2020). Pathogenicity and distribution of two species of *Cytospora* on *Populus tremuloides* in portions of the Rocky Mountains and Midwest in the United States. *Forest Ecology and Management, 468*, 118168. https://doi.org/10.1016/j.foreco.2020.118168

Egan, J. L., & Merrill, E. (1997). Disease incidence in ponderosa pine seed sources on coal mine spoil banks. *Tree Planters' Notes, 48*(1), 21–25.

Filip, G. M., Klopfenstein, N. B., Maffei, H. M., Shaw, C. G., III, & Lockman, I. B. (2024). *Armillaria root disease in conifers of western North America* (Forest Insect & Disease Leaflet 188). U.S. Department of Agriculture, Forest Service.

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. https://doi.org/10.1146/annurev.phyto.44.070505.143420

Geils, B. W., & Jacobi, W. R. (1993). Effects of comandra blister rust on growth of ponderosa pine. *Plant Disease, 77*(1), 47–51.

Graham, R. L., Turner, M. G., & Dale, V. H. (1990). How increasing $CO_2$ and climate change affect forests. *BioScience, 40*(8), 575–587.

Graham, R. T. (2005). Hayman Fire Case Study. *USDA Forest Service General Technical Report RMRS-GTR-114.*

Guyon, J. C., Jacobi, W. R., & McIntyre, G. A. (1996). Effects of environmental stress on the development of *Cytospora* canker of aspen. *Plant Disease, 80*(12), 1320–1326. https://doi.org/10.1094/PD-80-1320

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*(7), 773–778. https://doi.org/10.1094/PDIS.2000.84.7.773

Harlow, W. M., & Harrar, E. S. (1968). *Textbook of dendrology: Covering the important forest trees of the United States and Canada* (5th ed.). McGraw-Hill.

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*(5576), 2158–2162.

Harrington, M. G., & Hawksworth, F. G. (1990). Interactions of fire and dwarf mistletoe on

mortality of southwestern ponderosa pine. In J. S. Krammes (Ed.), *Effects of fire in management of southwestern natural resources: Proceedings of the symposium* (Gen. Tech. Rep. RM-191, pp. 234–240). USDA Forest Service, Rocky Mountain Forest and Range Experiment Station.

Harrington, T. C., Berryman, A. A., & Thier, R. W. (1983). Epidemiology of black stain root disease in ponderosa pine. *Canadian Journal of Botany, 61*(7), 1481–1488.

Hasselquist, N. J., Allen, M. F., & Santiago, L. S. (2010). Water relations of evergreen and deciduous trees along a seasonally dry tropical forest chronosequence. *Oecologia, 164*(4), 881–890.

Hennon, P. E., D'Amore, D. V., Schaberg, P. G., Wittwer, D. T., & Shanley, C. S. (2012). Shifting climate, altered niche, and a dynamic conservation strategy for yellow-cedar in the North Pacific coastal rainforest. *BioScience, 62*(2), 147–158. https://doi.org/10.1525/bio.2012.62.2.8

Hessburg, P. F., Goheen, D. J., & Bega, R. V. (1995). *Black stain root disease of conifers* (Forest Insect & Disease Leaflet No. 145). U.S. Department of Agriculture, Forest Service.

Hiratsuka, Y. (1969). *Endocronartium*, a new genus for autoecious pine stem rusts. *Canadian Journal of Botany, 47*(9), 1493–1495.

Hoffman, C., Mathiasen, R., & Sieg, C. H. (2007). Dwarf mistletoe effects on fuel loadings in ponderosa pine forests in northern Arizona. *Canadian Journal of Forest Research, 37*(3), 662–670. https://doi.org/10.1139/X06-259

Hood, S. M., Sala, A., Heyerdahl, E. K., & Boutin, M. (2015). Low-severity fire increases tree defense against bark beetle attacks. *Ecology, 96*(7), 1846–1855. https://doi.org/10.1890/14-0487.1

Houston, D. R. (1987). Forest tree declines of past and present. *USDA Forest Service General Technical Report NE-120.*

Jankowiak, R. (2007). Fungal flora associated with *Tomicus piniperda* L. in an area close to a timber yard in southern Poland. *Journal of Applied Entomology, 131*(8), 579–584. https://doi.org/10.1111/j.1439-0418.2007.01194.x

Jankowiak, R., Rossa, R., & Bilański, P. (2007). Contribution to pathogenicity of three blue-stain fungi associated with the pine sawyer beetle (*Monochamus galloprovincialis*) to Scots pine in Poland. *Phytopathologia Polonica, 46*, 37–46.

Jones, J. D. G., & Dangl, J. L. (2006). The plant immune system. *Nature, 444*(7117), 323–329.

Joseph, G., Kelsey, R. G., & Thies, W. G. (1998). Hydraulic conductivity in roots of ponderosa pine infected with black-stain (*Leptographium wageneri*) or annosus (*Heterobasidion annosum*) root disease. *Tree Physiology, 18*(5), 333–339. https://doi.org/10.1093/treephys/18.5.333

Kamata, N. (2008). Integrated management of pine wilt disease in Japan: Tactics and Strategies. In *Forest Insects and Pathogens* (pp. 147–153). Springer.

Kamiri, M., & Laemmlen, F. (1981). Effects of Drought-Stress and Wounding on Cytospora canker of conifers in Colorado Blue Spruce. *Plant Disease, 65*(4), 350–351.

Kearns, F., & Jacobi, W. R. (2005). Impacts of black stain root disease in recently formed mortality centers in the piñon-juniper woodlands of southwestern. *Forest Pathology, 35*(4), 251–259. https://doi.org/10.1111/j.1439-0329.2005.00354.x

Kile, G. A., McDonald, G. I., & Byler, J. W. (1991). Ecology and disease in natural forests. *Armillaria Root Disease.* USDA Forest Service Agriculture Handbook No. 691.

Kim, M.-S., Hanna, J. W., Stewart, J. E., Warwell, M. V., McDonald, G. I., & Klopfenstein, N. B. (2021). Predicting present and future suitable climate spaces (potential distributions) for an *Armillaria* root disease pathogen (*Armillaria solidipes*) and its host, Douglas-fir (*Pseudotsuga menziesii*), under changing climates. *Frontiers in Forests and Global Change, 4*, 740994. https://doi.org/10.3389/ffgc.2021.740994

Kliejunas, J. T., Geils, B. W., Glaeser, J. M., Goheen, E. M., Hennon, P. E., Kim, M. S., ... & Woods, A. J. (2009). Review of climate change and forest disease. *USDA Forest Service.*

Kliejunas, J. T., Geils, B. W., Glaeser, J. M., Goheen, E. M., Hennon, P., Kim, M. S., Kope, H., Stone, J., Sturrock, R., & Frankel, S. J. (2009). Review of literature on climate change and forest diseases (PSW-GTR-225). *USDA Forest Service, Pacific Southwest Research Station.*

Klopfenstein, N. B., Kim, M.-S., Hanna, J. W., Richardson, B. A., McDonald, G. I., & Geils, B. W. (2009). Approaches to predicting potential impacts of climate change on forest disease. *Forest Pathology, 39*(5), 362–375.

Kolb, T. E., & McDonald, P. M. (2016). Water relations and drought stress response of ponderosa pine. *Forest Ecology and Management, 380*, 190–200.

Krebill, R. G. (1968). *Cronartium comandrae in the Rocky Mountain States* (U.S. Forest Service

Research Paper INT-50). U.S. Department of Agriculture, Forest Service, Intermountain Forest & Range Experiment Station.

Krokene, P., & Solheim, H. (1998). Pathogenicity of four blue-stain fungi associated with aggressive and nonaggressive bark beetles. *Phytopathology, 88*(1), 39–44.

La Porta, N., Capretti, P., Thomsen, I. M., Kasanen, R., Hietala, A. M., & Von Weissenberg, K. (2008). Forest pathogens with higher damage potential due to climate change in Europe. *Canadian Journal of Plant Pathology, 30*(2), 177–195.

Lahlali, R., Taoussi, M., Laasli, S.-E., Gachara, G., Ezzouggari, R., Belabess, Z., Aberkani, K., Assouguem, A., Meddich, A., El Jarroudi, M., & Ait Barka, E. (2024). Effects of climate change on plant pathogens and host–pathogen interactions. *Crop and Environment, 3*(3), 159–170. https://doi.org/10.1016/j.crope.2024.05.003

Leech, S. M., Lara Almuedo, P., & O'Neill, G. (2011). Assisted migration: Adapting forest management to a changing climate. *BC Journal of Ecosystems and Management, 12*(3), 18–34.

Lertzman, K. P., & Fall, J. (1998). From forest stands to landscapes: Spatial scales and the roles of disturbances. In D. L. Peterson & V. T. Parker (Eds.), *Ecological scale: Theory and applications* (pp. 339–367). Columbia University Press.

Li, Z., Zhang, Q., Wei, Y., & Zhao, K. (2024). Radial Growth of Dahurian Larch (*Larix gmelinii*) Responses to Climate and Competition. *Forests, 15*(7), 1084. https://doi.org/10.3390/f15071084

Linaldeddu, B. T., Sirca, C., Spano, D., & Franceschini, A. (2011). Variation of endophytic cork oak–associated fungal communities in relation to plant health and water stress. *Forest Pathology, 41*(3), 193–201. https://doi.org/10.1111/j.1439-0329.2010.00652.x

Little, E. L. (1979). *Checklist of United States Trees (Native and Naturalized).* U.S. Department of Agriculture Handbook 541.

Liu, J.-J., & Ekramoddoullah, A. K. M. (2003). Isolation, genetic variation and expression of TIR-NBS-LRR resistance gene analogs from western white pine (Pinus monticola). Molecular Genetics and Genomics, 270, 432–441. https://doi.org/10.1007/s00438-003-0940-1

Long, J. N., & Smith, F. W. (2000). Restructuring the forest: Goshawks and the restoration of southwestern ponderosa pine. *Journal of Forestry, 98*(8), 25–30.

Lonsdale, D. (2002). Effect of climate change on fungal diseases of trees. *Forestry Commission Bulletin,* No. 125, 83–97.

Malek, R. B., & Appleby, J. E. (1984). Epidemiology of pine wilt in Illinois. *Plant Disease, 68*(9), 651–654.

Manion, P. D. (1981). *Tree disease concepts.* Prentice-Hall.

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*(11), 1256–1265.

Marsberg, A., Kemler, M., Jami, F., Nagel, J. H., Postma-Smidt, A., Naidoo, S., Wingfield, M. J., Crous, P. W., Spatafora, J. W., Hesse, C. N., Robbertse, B., & Slippers, B. (2017). *Botryosphaeria dothidea*: A latent pathogen of global importance to woody plant health. *Molecular Plant Pathology, 18*(4), 477–488. https://doi.org/10.1111/mpp.12495

Mathiasen, R. L., Nickrent, D. L., Shaw, D. C., & Watson, D. M. (2008). Mistletoes: Pathology, systematics, ecology, and management. *Plant Disease, 92*(7), 988–1006. https://doi.org/10.1094/PDIS-92-7-0988

McDonald, B. A., & Stukenbrock, E. H. (2016). Rapid emergence of pathogens in agro-ecosystems: A grand challenge for plant pathology. *Philosophical Transactions of the Royal Society B, 371*(1709), 20160026. https://doi.org/10.1098/rstb.2016.0026

McKee, M., Dobbs, J., Tisserat, N., Blodgett, J. T., Burns, K. S., & Stewart, J. E. (2025). First Report of Diplodia Shoot Blight and Canker Disease Caused by *Diplodia sapinea* on Ponderosa Pine in Colorado, U.S.A. *Plant Disease, 109*(2), 495.

Moir, W. H., Geils, B. W., Benoit, M. A., & Scurlock, D. (1997). Ecology of southwestern ponderosa pine forests. In *Ecology and management of forest diseases* (pp. 3–27). USDA Forest Service RM-GTR-292.

Moore, B. A., & Allard, G. B. (2008). *Climate change impacts on forest health* (Forest Health & Biosecurity Working Papers FBS/34E). FAO.

Moore, R., & Allard, G. (2011). Climate change impacts on forest health. *Forest Pathology, 41*(Suppl. 1), 103–104.

Moricca, S., Linaldeddu, B. T., Ginetti, B., Scanu, B., Franceschini, A., & Ragazzi, A. (2016). Endemic and emerging pathogens threatening cork oak trees: Management options for conserving a unique forest ecosystem. *Plant Disease, 100*(11), 2184–2193.

https://doi.org/10.1094/PDIS-03-16-0408-FE

Niinemets, Ü., & Valladares, F. (2006). Tolerance to shade, drought, and waterlogging of temperate Northern Hemisphere trees and shrubs. *Ecological Monographs, 76*(4), 521–547.

Old, K. M. (1981). Western gall rust, a serious disease of *Pinus radiata* in California. *Australian Forestry, 44*(3), 178–184.

Oliver, W. W., & Ryker, R. A. (1990). *Pinus ponderosa* Dougl. ex Laws. In R. M. Burns & B. H. Honkala (Tech. Coords.), *Silvics of North America: Volume 1. Conifers* (Agriculture Handbook 654, pp. 413–424). U.S. Department of Agriculture, Forest Service. https://www.srs.fs.usda.gov/pubs/misc/ag_654/volume_1/pinus/ponderosa.htm

Oswald, B. P., Dugan, S. C., Balice, R. G., & Unger, D. R. (2016). *Overstory tree mortality in ponderosa pine and spruce–fir ecosystems following a drought in northern New Mexico*. Forests, 7(10), 225. https://doi.org/10.3390/f7100225

Owen, D. R., Lindahl, K. Q., Wood, D. L., & Parmeter, J. R., Jr. (1987). Pathogenicity of fungi isolated from *Dendroctonus valens*, *D. brevicomis*, and *D. ponderosae* to ponderosa pine seedlings. *Phytopathology, 77*(4), 631–636.

Parmeter, J. R., Jr., Slaughter, G. W., Chen, M.-M., Wood, D. L., & Stubbs, H. A. (1989). Single and mixed inoculations of ponderosa pine with fungal associates of *Dendroctonus* spp. *Phytopathology, 79*(7), 768–772. https://doi.org/10.1094/Phyto-79-768

Pautasso, M., Döring, T. F., Garbelotto, M., Pellis, L., & Jeger, M. J. (2012). Impacts of climate change on plant diseases—opinions and trends. *European Journal of Plant Pathology, 133*(1), 295–313. https://doi.org/10.1007/s10658-012-9936-1

Pearson, R. G., & Dawson, T. P. (2003). Predicting the impacts of climate change on the distribution of species: Are bioclimate envelope models useful? *Global Ecology and Biogeography, 12*(5), 361–371.

Peterson, R. S. (1961). Western gall rust cankers in Lodgepole pine. *Journal of Forestry, 59*(3), 194–196.

Prospero, S., Botella, L., Santini, A., & Robin, C. (2021). Biological control of emerging forest diseases: How can we move from dreams to reality? *Forest Ecology and Management, 496*, 119377.

Raish, C., Gonzalez-Caban, A., & Condie, C. J. (1997). A cultural and ecological assessment of

forest health management. *General Technical Report RM-GTR-295. USDA Forest Service.*

Ramegowda, V., & Senthil-Kumar, M. (2015). The interactive effects of simultaneous biotic and abiotic stresses on plants: Mechanistic understanding from drought and pathogen combination. *Journal of Plant Physiology, 176*, 47–54.

Rane, K. K., & Tattar, T. A. (1987). Pathogenicity of blue-stain fungi associated with *Dendroctonus terebrans*. *Plant Disease, 71*(10), 879–883. https://doi.org/10.1094/PD-71-879

Reynolds, R. T., Graham, R. T., Reiser, M. H., & Bassett, R. L. (1992). Management recommendations for the northern goshawk in the southwestern United States. *USDA Forest Service.*

Ridout, M., & Newcombe, G. (2018). A potential role for endophytes in mitigating drought stress in forest trees. *Current Forest Reports, 4*(3), 73–80.

Rishbeth, J. (1978). Effects of soil temperature and atmosphere on growth of *Armillaria* rhizomorphs. *Transactions of the British Mycological Society, 70*(2), 213–220. https://doi.org/10.1016/S0007-1536(78)80033-3

Robichaud, P. R. (2005). Measurement of post-fire hillslope erosion to evaluate and model rehabilitation treatment effectiveness and recovery. *International Journal of Wildland Fire, 14*(4), 475–485. https://doi.org/10.1071/WF05031

Rogers, J. D., & Noskowiak, A. F. (1976). Brown sapwood stain of ponderosa pine caused by *Cytospora* sp.: Cultural and histological aspects. *Phytopathology, 66*(1), 25–27. https://doi.org/10.1094/Phyto-66-25

Romero, F. M., Marín, C., Zürcher, C., & van der Heijden, M. G. A. (2022). Humidity and high temperature are important for predicting fungal disease outbreaks worldwide. *New Phytologist, 234*(5), 1553–1556. https://doi.org/10.1111/nph.17340

Roth, L. F. (1971). Dwarf mistletoe damage to small ponderosa pines. *Forest Science, 17*(3), 373–380. https://doi.org/10.1093/forestscience/17.3.373

Rutherford, T. A., & Webster, J. M. (1987). Distribution of pine wilt disease with respect to temperature in North America, Japan, and Europe. *Canadian Journal of Forest Research, 17*(9), 1050–1059. https://doi.org/10.1139/X87-161

Scharpf, R. F. (1981). Impact of *Elytroderma deformans* on growth of Jeffrey pine. *Plant*

*Disease, 65*(12), 1013–1014.

Schoeneweiss, D. F. (1975). Predisposition, stress, and plant disease. *Annual Review of Phytopathology, 13*(1), 193–211.

Schoeneweiss, D. F. (1981). The role of environmental stress in diseases of woody plants. *Plant Disease, 65*(4), 308–314.

Schoeneweiss, D. F. (1983). Drought predisposition to *Cytospora* canker in blue spruce. *Plant Disease, 67*(4), 383–385. https://doi.org/10.1094/PD-67-383

Shaw, C. G., & Kile, G. A. (1991). *Armillaria root disease.* USDA Forest Service Agriculture Handbook No. 691.

Sieber-Canavesi, F., & Sieber, T. N. (1993). Endophytic fungi of Norway spruce needles. *Mycological Research, 97*(12), 151–156.

Silva, A. C., Henriques, J., Diogo, E., Ramos, A. P., & Bragança, H. (2020). First report of *Sydowia polyspora* causing disease on *Pinus pinea* shoots. *Forest Pathology, 50*, e12570. https://doi.org/10.1111/efp.12570

Sinclair, W. A., Lyon, H. H., & Johnson, W. T. (1987). *Diseases of trees and shrubs.* Cornell University Press.

Slippers, B., & Wingfield, M. J. (2007). Botryosphaeriaceae as endophytes and latent pathogens of woody plants: Diversity, ecology and impact. *Fungal Biology Reviews, 21*(2–3), 90–106. https://doi.org/10.1016/j.fbr.2007.06.002

Smirnova, O. V., Vasilyeva, L. N., & Le Page, Y. (2021). *Elytroderma deformans* and needle cast of conifers in Eurasia and North America. *Forest Pathology, 51*(2), e12700.

Souder, S. K., & Strimbu, B. M. (2021). Evaluating the use of systemic fungicides in forest disease management. *Forests, 12*(8), 1104.

Spittlehouse, D. L. (2009). Integrating climate change adaptation into forest management. *Forestry Chronicle, 85*(5), 657–659.

Spittlehouse, D. L. (2009). Adapting to climate change in forest management: A management agency response. *Mountain Views Newsletter, 3*(1), 2–4.

Stanosz, G. R., Blodgett, J. T., Smith, D. R., & Kruger, E. L. (2001). Water stress and *Sphaeropsis sapinea* as a latent pathogen of red pine seedlings. *New Phytologist, 149*(3), 531–538.

Steiner, G., & Buhrer, E. M. (1934). *Aphelenchoides xylophilus* n. sp., a nematode associated

with blue-stain and other fungi in timber. *Journal of Agricultural Research, 48*(10), 949–955.

Stenlid, J., Oliva, J., Boberg, J. B., & Hopkins, A. J. M. (2011). Emerging diseases in European forest ecosystems and responses in society. *Forest Ecology and Management, 261*(11), 1639–1643.

Stenlid, J., Oliva, J., Boberg, J. B., & Hopkins, A. J. M. (2011). Emerging diseases in European forest ecosystems and responses in society. *Forest Pathology, 41*(Suppl. 1), 134–146.

Stone, J. K., Coop, L. B., & Manter, D. K. (2008). Predicting Swiss needle cast severity using a model of degree-days and needle wetness. *Phytopathology, 98*(6), 739–744.

Sturrock, R. N. (2012). Forest pathology: A key issue in forest health management. *Forest Pathology, 42*(Suppl. 1), 5–7.

Sturrock, R. N. (2012). Climate change and forest diseases: using today's knowledge to address future challenges. *Forest Systems, 21*(2), 329–336.

Sturrock, R. N., Frankel, S. J., Brown, A. V., Hennon, P. E., Kliejunas, J. T., Lewis, K. J., Worrall, J. J., & Woods, A. J. (2011). Climate change and forest diseases. *Plant Pathology, 60*(1), 133–149.

Szymański, N., & Wilczyński, S. (2021). Radial growth response of European larch provenances to interannual climate variation in Poland. *Forests, 12*(3), 334. https://doi.org/10.3390/f12030334

Talgø, V., Chastagner, G. A., Thomsen, I. M., & Stensvand, A. (2010). *Sydowia polyspora* associated with current season needle necrosis of *Abies* spp. in Scandinavia. *Forest Pathology, 40*(4), 253–261.

Thomas, J. W. (1979). *Wildlife habitats in managed forests: The Blue Mountains of Oregon and Washington.* USDA Forest Service Agricultural Handbook No. 553.

Thompson, I., Mackey, B., McNulty, S., & Mosseler, A. (2009). *Forest resilience, biodiversity, and climate change* (CBD Technical Series No. 43). Secretariat of the Convention on Biological Diversity.

Tinivella, F., Dani, E., Minuto, G., & Minuto, A. (2014). First report of *Sydowia polyspora* on Aleppo pine (*Pinus halepensis*) in Italy. *Plant Disease, 98*(2), 281. https://doi.org/10.1094/PDIS-06-13-0658-PDN

Tor-ngern, P., Oren, R., Palmroth, S., Novick, K., Oishi, A., Linder, S., Ottosson-Löfvenius, M.,

& Näsholm, T. (2018). *Water balance of pine forests: Synthesis of new and published results*. *Agricultural and Forest Meteorology, 259*, 107–117. https://doi.org/10.1016/j.agrformet.2018.04.021

Van Hooser, D. D., & Keegan, C. E. (1988). Distribution and volumes of ponderosa pine. *USDA Forest Service General Technical Report RM-166.*

Wagener, W. W. (1961). A staining-fungus root disease of Ponderosa, Jeffrey, and Pinyon Pines. *Plant Disease Reporter, 45*(11), 831–835.

Wallis, C. M., Reich, R. W., Lewis, K. J., & Huber, D. P. W. (2010). Lodgepole pine provenances differ in chemical defense capacities against foliage and stem diseases. *Canadian Journal of Forest Research, 40*(12), 2333–2344. https://doi.org/10.1139/X10-178

Walters, D., & Heil, M. (2007). Costs and trade-offs associated with induced resistance. *Physiological and Molecular Plant Pathology, 71*(1–3), 3–17.

Waterman, A. M. (1955). The relation of *Valsa kunzei* to cankers on conifers. *Phytopathology, 45*, 686–692.

Watt, M. S., Palmer, D. J., & Bulman, L. S. (2011). Predicting the severity of Dothistroma on *Pinus radiata* under current climate in New Zealand. *Forest Ecology and Management, 261*(11), 1792–1798. https://doi.org/10.1016/j.foreco.2011.01.043

Welsh, C., Lewis, K. J., & Woods, A. J. (2009). The outbreak history of Dothistroma needle blight: An emerging forest disease in northwestern British Columbia, Canada. *Canadian Journal of Forest Research, 39*(12), 2505–2519. https://doi.org/10.1139/X09-159

Welsh, C., Lewis, K. J., & Woods, A. J. (2014). Regional outbreak dynamics of Dothistroma needle blight linked to weather patterns in British Columbia, Canada. *Canadian Journal of Forest Research, 44*(3), 212–219. https://doi.org/10.1139/cjfr-2013-0387

Tor-ngern, P., Oren, R., Palmroth, S., Novick, K., Oishi, A., Linder, S., Ottosson-Löfvenius, M., & Näsholm, T. (2018). *Water balance of pine forests: Synthesis of new and published results*. *Agricultural and Forest Meteorology, 259*, 107–117. https://doi.org/10.1016/j.agrformet.2018.04.021

Wingfield, M. J., Blanchette, R. A., Nicholls, T. H., & Robbins, K. (1982). The pine wood nematode: A comparison of the situation in the United States and Japan. *Canadian Journal of Forest Research, 12*(1), 71–75.

Wingfield, M. J., Slippers, B., Barnes, I., Duong, T. A., & Wingfield, B. D. (2024). The pine

pathogen *Diplodia sapinea*: Expanding frontiers. *Current Forestry Reports, 11*(2), Article 2. https://doi.org/10.1007/s40725-024-00236-2

Winder, R. S., Nelson, E. A., & Beardmore, T. (2011). Ecological implications for assisted migration in Canadian forests. *The Forestry Chronicle, 87*(6), 731–744.

Woods, A., Coates, K. D., & Hamann, A. (2005). Is an unprecedented Dothistroma needle blight epidemic related to climate change? *BioScience, 55*(9), 761–769. https://doi.org/10.1641/0006-3568(2005)055[0761:IAUDNB]2.0.CO;2

Woods, A. J., Heppner, D., Kope, H. H., Burleigh, J., & Maclauchlan, L. (2010). Forest health and climate change: A British Columbia perspective. *The Forestry Chronicle, 86*(4), 412–422.

Woods, A. J., Martín-García, J., Bulman, L., Vasconcelos, M. W., Boberg, J., La Porta, N., Peredo, H., Vergara, G., Ahumada, R., Brown, A., & Diez, J. J. (2016). Dothistroma needle blight, weather and possible climatic triggers for the disease's recent emergence. *Forest Pathology, 46*(5), 443–452. https://doi.org/10.1111/efp.12248

Worrall, J. (2013). *Dwarf mistletoes: Ecology and management in the Rocky Mountain Region.* USDA Forest Service, Rocky Mountain Region.

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*(3–4), 686–696

Worrall, J. J., Marchetti, S. B., Egeland, L., Mask, R. A., Eager, T., & Howell, B. (2010). Effects and etiology of sudden aspen decline in southwestern Colorado, USA. *Forest Ecology and Management, 260*(5), 638–648. https://doi.org/10.1016/j.foreco.2010.05.020

Yanchuk, A. D., Yeh, F. C. H., & Dancik, B. P. (1988). Variation of stem rust resistance in a lodgepole pine provenance-family plantation. *Forest Science, 34*(4), 1067–1075.

# CHAPTER 2: PONDEROSA PINE HEALTH FIELD SURVEY

## 2.1 Summary

Ponderosa pine spans extensive dry-forest landscapes of the western United States but faces increasing climatic stress under warming and aridification. Most prior research on ponderosa pine status in the landscape has been regional and centered on a single disturbance like fire regeneration, climate, or pathogen outbreaks. This leaves questions about how climate, stand structure, and multiple biotic agents jointly relate to dieback and mortality. To fill these gaps, we surveyed 26 sites across California, Colorado, Idaho, Montana, and Wyoming, during the summers of 2024 and 2025. We established standardized plots, rated tree health, sampled symptomatic tissues for diagnosis, and collected 30-year PRISM climate data for each site. We tested health score distributions, mapped pathogen frequencies and severity, related mean health to climate and stand attributes, and built pathogen centered correlation networks for the five most common pathogens found that integrate climate, site factors, prevalence, and host conditions. Across all five study states, multi-decade warming, higher vapor pressure deficit, and a recent precipitation deficit was correlated with more severe decline at warmer, drier sites. Latent opportunistic foliar and canker fungi, like *Sydowia polyspora* and *Diplodia sapinea*, were widespread, most frequently at cooler, wetter sites and concentrated in moderate decline. Likewise, *Cytospora* spp. was associated with moderate declining trees, but prevalence increased with warmer temperatures and elevated VPD, consistent with stress canker behavior. Beetle associated, blue stain fungi (*Leptographium* spp., *Ophiostoma* spp.) were associated with hot, dry, lower elevation sites and dead trees. Taken together, the results indicate two patterns of decline where climate predisposes trees and native pathogens take advantage: (1) cooler, wetter

windows following long term heat and moisture stress that favor foliar and latent pathogen growth, manifested as moderate dieback, and (2) hot, dry regimes that accelerate blue stain activity, resulting in severe decline and tree mortality. Geographical differences in pathogen occurrence reflect the native ranges of the agents and how local climates impact infection.

## 2.2 Introduction

The ponderosa pine is a foundational conifer species across much of the western United States, providing timber, wildlife habitat, and recreational value. Over the past several decades, warmer temperatures and regional aridification have increased atmospheric water demand and altered moisture regimes. These conditions can weaken trees and heighten tree susceptibility to biotic pressures (Abatzoglou & Williams 2016; Allen et al. 2010; Littell et al. 2009). Although a widespread decline has not been formally established across all landscapes, field observations in multiple states have noted pockets of ponderosa pine canopy dieback and mortality. Yet, it's unknown whether the dieback and mortality are associated with only abiotic factors, or if biotic agents also play a role in the declining ponderosa pine health.

Multiple interacting abiotic or biotics factors can predispose, incite, and contribute to episodes of dieback and mortality in conifers. Tree decline emerges when long-term predisposing stresses lower defenses, then a short-term inciting event pushes trees past physiological thresholds, and lastly contributing organisms or agents then amplify and the decline persists (Manion & Lachance 1992). In pines, many tree-associated fungi occupy a spectrum of lifestyles, from largely endophytic or latent to actively pathogenic in vascular tissues. For example, *Diplodia sapinea* has been recognized as a stress-responsive pathogen capable of shoot blight, cankers, and occasionally mortality, with recent findings on its behaviors under dry-warm

conditions (Stanosz et al. 2001; Wingfield et al., 2025). *Sydowia polyspora* is widely detected as a foliar endophyte whose expression as a pathogen can increase following stress (Ridout & Newcombe 2018), while *Cytospora* spp. are classic canker fungi that capitalize on low vigor status or wounding of their host (Desprez-Loustau et al. 2006). Collectively, these examples illustrate that pathogen presence is widespread, but disease incidence and severity can be altered by environmental factors and host stress exacerbated by climate change.

Anthropogenic warming has increased fuel aridity (drier fuels under high atmospheric demand) and expanded burning area across western forests as well as elevated background stress on ponderosa pine (Abatzoglou & Williams, 2016). In fire-prone landscapes, conditions like higher fire severity, increases in temperature, and decreases in moisture, lead to unsuccessful post-fire regeneration. (Davis et al. 2019, 2023). Beyond fire, it has been reported that, even in unburned stands, ponderosa pine is experiencing losses from climate-linked stress (Woolman et al. 2022). Multiple native, biotic agents have also been demonstrated to reduce ponderosa pine performance and increase risk. Chronic parasitism by dwarf mistletoe (*Arceuthobium* spp.) reduces growth and alters stand structure and fuels, shaping fire behavior and stand vigor (Hoffman et al. 2007; Shaw & Agne 2017). Endophytic fungi, such as *Sydowia polyspora*, can become a pathogen when the host is under stress with measurable effects on ponderosa pine regeneration (Ridout & Newcombe, 2018). Heterobasidion root disease and *Armillaria* spp. can further compound drought stress eventually leading to growth loss and mortality (Filip et al., 2024; Oester et al. 2018). Defoliation events by the pandora moth (*Coloradia pandora*) can depress radial growth in ponderosa pine and when coincident with drought or fire, entire stands can be impacted (Ciesla et al., 2010; O'Neill et al., 2023). Native bark beetles like western pine beetle (*Dendroctonus brevicomis*) and mountain pine beetle (*D. ponderosae*) respond to heat and

drought. When conditions turn hot and dry, pine beetle numbers can surge, and pine mortality can rapidly increase. Together, these biotic agents that are already present in low numbers in natural stands can interact with changing climate factors that result in increased prevalence and severity of the diseases they cause, amplifying ponderosa pine decline risk. Most prior research on ponderosa pine tree health has focused on single biotic or abiotic factors, such as fire severity, climate, pathogens, or insects, rather than examining ponderosa health on a landscape scale. As a result, we lack a landscape-wide, plot-based study of ponderosa pine that assesses tree health by integrating biotic agent presence with stand and climate data. The aim of this research was to use a landscape scale study, that jointly samples pathogens and records stand characteristics and climate conditions across ponderosa pine's distribution. To provide information on factors contributing to ponderosa pine dieback and mortality, this study (i) establishes the biotic and abiotic agents contributing to ponderosa decline, (ii) tests associations between pathogen prevalence and severity with climate variables, and (iii) determines regional differences among pathogens observed on ponderosa pine.

## 2.3 Material and methods

### 2.3.1 Sampling design and implementation

Field plots were established to collect tree health data and samples from symptomatic ponderosa pine in five states. USDA Forest Service, Forest Health Protection (FHP) collaborators were consulted about ponderosa pine health in their designed regions. Damage was reported in California, Colorado, Idaho, Montana, and Wyoming. A drought index was then created within forest with reported damage to find areas with potential precipitation anomalies. This was done by first collecting weather and climate data from Prism Group at Oregon State

University (https://prism.oregonstate.edu/). Thirty years (1993-2023) of precipitation data was downloaded for each forest at a 4-kilometer (km) resolution. Next, the averages for the past five years (2019-2023) and the past twenty-five years (1993-2018) were calculated for each 4 km square. The averages were then subtracted (5-year average-25-year average) to create a grid that showed any areas with precipitation anomalies. Areas with less precipitation on average in the past five years were targeted over areas with roughly the same or more precipitation. Along with precipitation data, cover type was mapped to stay in areas dominated by ponderosa using aerial survey data from 2020 with ArcGIS.

2.3.2 Plot design and data collection.

Plot surveys and sampling took place in the summers of 2024 and 2025 in five (California, Colorado, Idaho, Montana, and Wyoming) states with reported damage. Each plot consisted of three, 100ft transects that were approximately equidistant. The transects converged at a center tree and ran south, northeast, and northwest. GPS coordinates, slope, and aspect were measured at plot center. Every 15ft along each of the three transects, a $10ft^2$/acre prism was used to estimate basal area of the plot. Along each transect, trees that were within 5ft of either side were given an identification name, health score, and measured diameter at breast height (DBH). Health scores were recorded on a 1-5 scale based on the following criteria: (1) healthy: <15% damage to crown/stem, (2) declining: 16-50% damage to crown/stem, (3) dying: >50% damage to crown/stem, (4) recently dead: no green needles, red needles/fine twigs present, (5) grey dead: fine twigs, no needles. Signs and symptoms of disease and damage, such as insects or fire, were also recorded. Based on the health score and observable symptoms, multiple samples were taken. Sample types included branches, needles, cones, drill shavings for pinewood nematode (PWN). Healthy trees were not sampled but were still given an ID, health rating, and DBH was recorded.

### 2.3.2 Sample Processing

Field samples were processed in the lab to detect any possible pathogens. Different methods were used for each type of sample.

Symptomatic ponderosa pine needles were sampled for fungal plant pathogens. Needles with banding or flecking were cut to approximately 5mm or along the margin of healthy and infected tissue. A total of 6-8 pieces per sample were then surface sterilized by placing needle samples in 70% ethanol for 60s, 1% bleach for 3 mins, and then sterile water for 30s. The needle samples were then air dried in a laminar flow hood briefly and then placed on ½ strength potato dextrose agar (PDA) (Hardy Diagnostics; Santa Maria, CA) media, sealed with parafilm, and placed in an incubator at 20C. For samples with fruiting bodies, the needle was surface sterilized using the same methods but were placed in a humid chamber constructed with an empty agar plate with a moist filter paper on the lid. After 2-3 days in the incubator, the samples were then placed in ½ strength PDA media and returned to the incubator.

For wood and cone samples, the same general workflow was followed but modified slightly for different sample types. Wood samples were obtained by scraping the very outer surface of bark off to locate areas with necrotic lesions. Once located, 3-4 small wood chips (5-10mm) per sample were excised along the margin of healthy and infected tissue. These samples were then surface sterilized in 70% ethanol for 60s, 1% bleach for 5 mins, and rinsed in sterile water for 30s. Samples were dried briefly in the laminar flow hood and then place on ½ strength PDA media.

Cone samples were only taken from plots suspected to have Diplodia Tip and Shoot Blight (DTSB). The pathogen, *Diplodia sapinea*, overwinters on fallen cones and many times

fruiting bodies are visible. To isolate *D. sapinea* from cones, 2-3 scales from each cone were surface sterilized in 70% ethanol for 30s, 1% bleach for 1 min, and sterile H20 for 30s. The samples were dried and then placed in a humid chamber in an incubator at 23C. After 2-3 days, they were then placed on ½ PDA media and returned to the incubator.

After branch, needle, and cone samples were grown out sufficiently, anything growing from the sample was sub-cultured and placed on a new ½ strength PDA media plate and returned to the incubator. Once a pure culture was obtained and grown until it covered roughly 2/3 of the agar plate, the culture placed in storage at 4°C until DNA was extracted.

2.3.3 DNA extraction and amplification

The DNA was extracted by scraping a single strand of mycelium and placing it directly into a 0.2mL PCR strip tube with a 5% Chelex 100 resin solution and mixed for 30s using a pipette tip. Samples were then spun down and placed in a thermocycler for 20 mins at 98°C (Kozhar et al. 2023). The samples were then stored at -80°C.

Polymerase chain reaction (PCR) was then used to amplify the internal transcribed spacer region (ITS) from the extracted DNA. The master mix contained 1 µl of the forward and reverse primers ITS1F/ITS4 (White et al. 1990), 12.5 µl of GoTaq Green 1x (Promega), and 8.5 µl of MH2O. 2 ul of extracted DNA was used for a total reaction volume of 25 µl. The cycling parameters followed those described by White et al (1990). PCR products were run on a 1.5% agarose gel to visualize amplified PCR products using GelRed® and sent to Eurofins Genomics for forward and reverse sequencing. The obtained sequences were then compared to reference strains found in the NCBI database using the BLAST algorithm. The top results from the BLAST

search were then recorded,and culture samples were either destroyed or stored in 20% glycerol at -80°C. These samples are stored at Colorado State University, Fort Collins, CO.

For wood shavings or increment wood cores taken for PWN, a DNA extraction method was developed using Chelex 100 resin (BioRad; Walsh et al. 1991). In addition, 2% polyvinylpyrrolidone (PVP) was added to the solution to help reduce phenolic compounds contamination (Heikrujam et al., 2020). Approximately 0.01g of shavings or increment wood core were added to 200ul of the 5% Chelex and 2% PVP solution. Samples were then incubated at 98°C in the MyBlock mini dry bath for 30 minutes. All samples were tested using a commercial loop-mediated isothermal amplification (LAMP) kit (*B. xylophilus* detection kit: Nippon Gene Co. Ltd., Tokyo, Japan) developed based on the methods by Kikuchi et al., 2009. Positives samples and a subset of negatives were then confirmed using the field-based colorimetric LAMP assay for PWN (McKee, in prep).

2.3.4 Abiotic factor assessment

To assess the effects of abiotic factors, weather and climate data from Prism were collected for each site using the GPS coordinates for a site. Using a 4-kilometer (km) resolution, mean precipitation, minimum, mean, and maximum temperature, and minimum and maximum VPD was downloaded for the past 30 years.

Differences in climate between the most recent five years and the past twenty-five years were analyzed. Site-specific averages for each climate variable were calculated separately for the five-year (2019–2024) and twenty-five-year (1994–2018) periods. The five-year average was subtracted from the twenty-five-year average to quantify recent changes. These differences were

then visualized using bar graphs in RStudio (version), highlighting shifts in climate conditions at each site in recent years.

### 2.3.6 Data analysis

All data handling, summarization, statistics, and graphics were scripted in R (v4.4.1). Raw plot- and tree-level datasets were imported, validated, and cleaned. Data wrangling and summaries used the tidyverse (dplyr, tidyr) and base R; inferential tests used stats with p.adjust for multiple-testing control (Holm and Benjamini–Hochberg), with broom/rstatix to tidy outputs. Figures were produced with ggplot2 (patchwork for multi-panel layouts) and networks with igraph/ggraph; tables and spreadsheets were generated with gt and openxlsx2.

*Health score frequency.* To quantify dieback and mortality of ponderosa pine across all plots across states, health score frequency. For each tree at every site, a health score was recorded on an ordinal score (1-5). Out of range or blank entries were treated as missing. Frequency tables were then generated for the full dataset and then stratified by state.

We tested whether the overall distribution differed from a priori expectation and whether distributions varied by state. For the state-combined data, each score (1-5) was tested against a uniform 20% expectation using a two-sided exact binomial test, with a Holm step-down correction across the five categories. For state differences, a state × score table was generated, and a chi-square test of independence was run. Departures were localized with adjusted standardized residuals (over-representation if positive, under-representation if negative). Residuals were converted to two-sided p-values and Holm-adjusted across the 25 state×score cells ($\alpha = 0.05$).

*Pathogen frequency.* To determine if pathogens differ geographically, pathogen frequency was quantified across all plots and for each state. For each tree recorded, binary pathogen detections (0/1) were used. For each pathogen, overall frequency and by state counts were reported and visualized using bar graphs. In state graphs, pathogens with zero detections were removed.

*Pathogen x health score.* To observe trends between pathogen and health score frequency, pathogen presence/absence was cross tabulated against health score. To stabilize sparse cells, pathogens with less than 5 detections were collapsed into an "Other" category. One two-way table was generated for all plots, plus five state-specific tables that reported counts.

*Pathogen focused subset.* To determine individual pathogens were significantly associated with diseased trees, Fisher's exact test was used on a pathogen focused subset. Analysis was restricted to trees with a health score greater than 2 and severity was dichotomized as moderate (2-3) and dead (4-5) for statistical stability and simplicity. Effect size was summarized as the odds ratio (OR) for severe disease, comparing presence vs absence of a biotic agent, with 95% confidence intervals computed on the log scale. To address multiple comparisons across pathogens, the false discovery rate was controlled using the Benjamini–Hochberg (BH) procedure and reported q-values, designating $q \leq 0.05$ as statistically significant (Benjamini & Hochberg 1995). The analysis was repeated on the state level with BH adjustment applied separately.

*Correlation analysis (site level).* To see how abiotic and site factors contribute to tree health, a correlation analysis was conducted at the site level. For the correlation screen, we performed a site-level, bivariate correlation analysis linking a response variable (health score) to stand-structure and climate predictors drawn from anomaly data made from the difference of 5-

and 25-year averages. Trees were aggregated to one row per site and used the mean health score as the response variable. After standardizing column the datasheets, tables were merged by state and site. Predictors included minimum/mean/maximum temperature (°F), maximum/maximum VPD (hPa; mean VPD used when present), mean precipitation (in), mean DBH, basal area, elevation (ft), and aspect eastness (sin) and northness (cos). Pathogen variables were excluded from this correlation because testing was non-uniform and preferential to symptomatic trees, so prevalence would be biased and not comparable across sites. For the analysis, Spearman's rank correlation (ρ) between health score and the predictor variables using a pairwise-complete observations and two-sided p-values were reported.

*Network analysis for top pathogens*. To explore associations between site conditions, climate, pathogen occurrence, and host conditions correlation-based networks were constructed. For each focal taxon, five pathogens (*Sydowia polyspora, Diplodia sapinea, Leptographium* spp., *Cytospora* spp. *Ophiostoma* spp.) with the highest detection frequency across all plots were identified and analyzed in separate, pathogen centric networks. Raw plot and tree level data was aggregated to site level: continuous climate metrics (mean Tmin/Tmean/Tmax, VPDmin/VPDmax, and precipitation) and site factors (elevation, basal area, DBH, numeric aspect transformations) were averaged per site. Pathogen occurrence was treated as prevalence (mean of 0/1 presence across observations within a site). Host health was averaged as the site level mean health score. A Spearman rank correlation was computed between all variables. For each network, only associations meeting $|\rho| \geq 0.30$ and $p < 0.05$ were kept. Networks were rendered as unidirectional graphs with notes representing variables grouped in 4 categories (climate, site, pathogen, mean health) and edges represented the filtered Spearman associations.

Edge color indicates the sign of the correlation (red = positive, blue = negative) and edge width scales with |ρ|.

## 2.4 Results

### 2.4.1 Abiotic factor assessment

When comparing weather and climate data from the past five years to the past twenty-five years, consistent trends emerged across all climate factors across all states. All sites experienced a decline of average precipitation over the past five years **(Fig. 1).** California sites showed the most significant change, with an average deficit of 6.48 inches per site. Colorado and Wyoming had average losses of 1.94 and 1.97 inches, respectively. Idaho and Montana sites had the smallest reductions, averaging 1.54 and 1.30 inches of precipitation lost, respectively.

Changes in mean temperature also varied by region. Sites in California, Colorado, and Wyoming all experienced increases in average temperature over the past five years. Wyoming sites had the largest increase, averaging 0.52°F, followed by California with an increase of 0.37°F and Colorado with 0.31°F. In contrast, mean temperatures in Idaho remained relatively stable, with only a 0.01°F increase, while Montana sites experienced a decrease of 0.25°F (**Fig. 2)**. For vapor pressure deficit (VPD), maximum values increased at 23 of the total 26 sites. Only three sites—two in Colorado and one in Montana—saw a decrease in maximum VPD. In contrast, minimum VPD exhibited greater variability. More than half of the California sites experienced a decrease in minimum VPD, while three showed an increase. Nearly all sites in Idaho and Montana saw declines in minimum VPD, whereas all Colorado and Wyoming sites experienced increases (**Fig. 3 & 4)**.

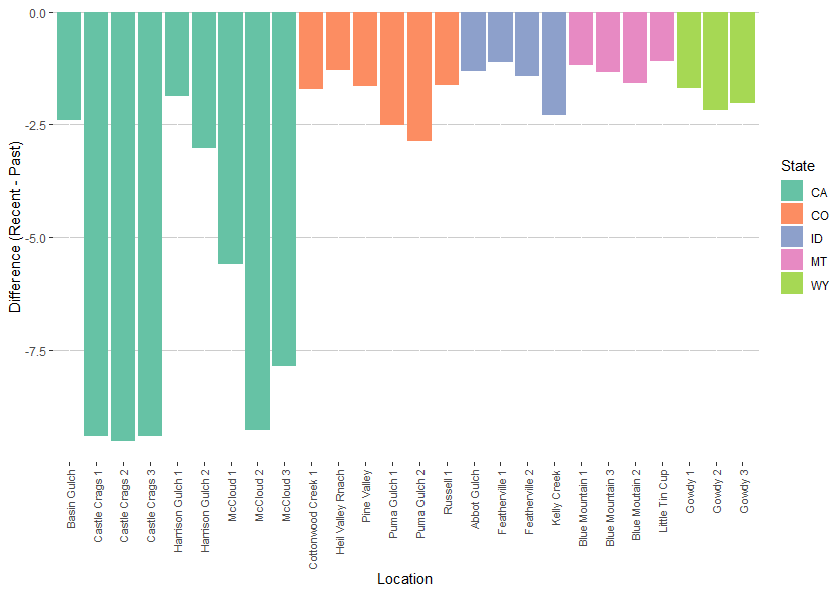


**Figure 1.** Differences in yearly precipitation averages in the past 5 and 25 years (5-25).

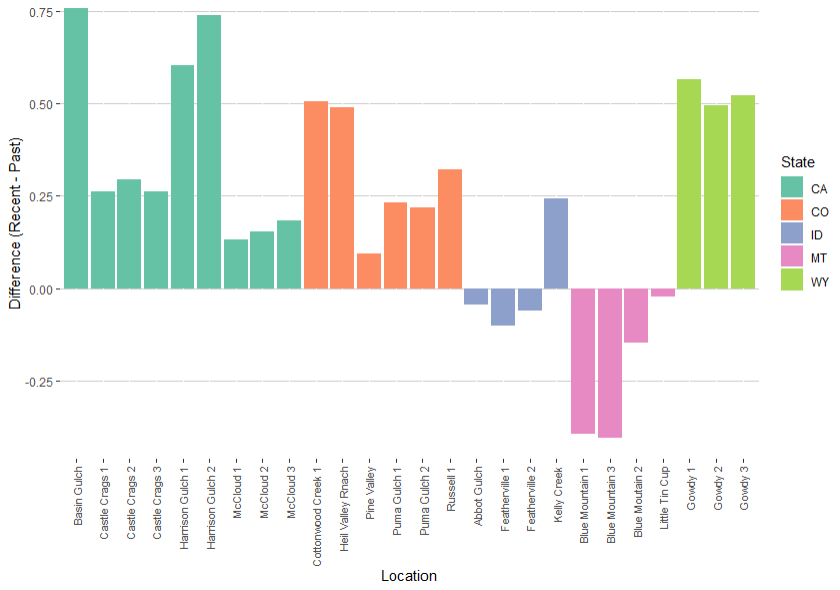


**Figure 2**. Difference in yearly average mean temperatures for the past 5 and 25 years (5-25).

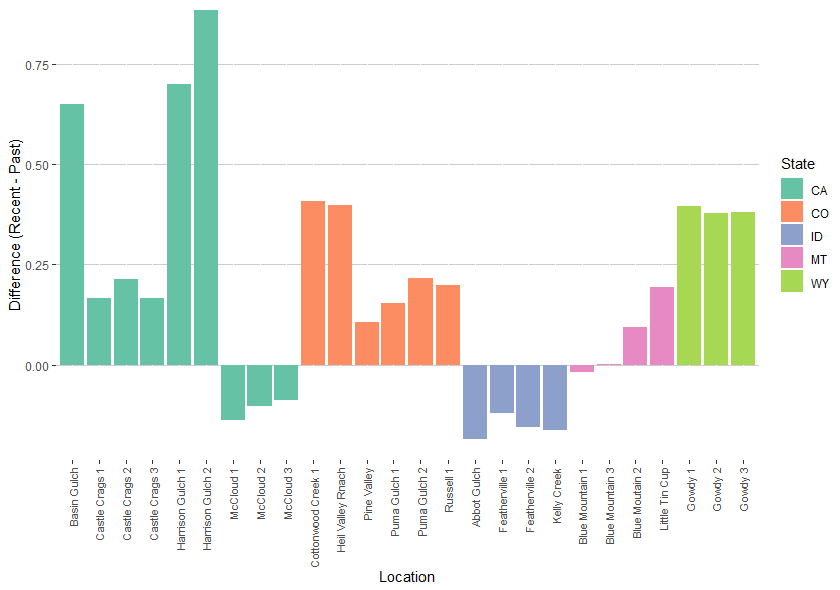


**Figure 3**. Difference in yearly average minimum VPD for the past 5 and 25 years (5-25).

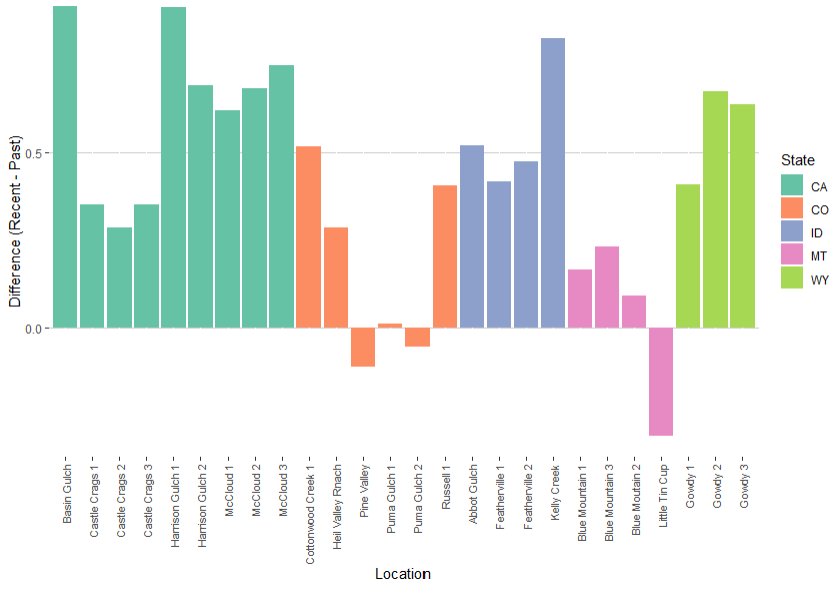


**Figure 4**. Difference in yearly average maximum VPD for the past 5 and 25 years (5-25).

2.4.2 Site and stand characteristics

Twenty-six plots were established during the summers of 2024 and 2025. Nine plots in northern California (9), eight plots in Montana and Idaho (8), and nine in northern Colorado and southern Wyoming (9). A total of 404 trees were examined, and 80% (ca. 324) were ponderosa pine. Other species included in the survey were Douglas-fir (*Pseudotsuga menziesii var.*

*menziesii*) (ca. 42), Juniper (*Juniperus* sp.) (ca. 20), black oak (*Quercus veluntina*) (ca. 13), and common manzanita (*Arctostaphylos manzanita*) (ca. 5). The average diameter at breast height (DBH) for ponderosa pine trees was 11.06 inches with an average health score of 2.26. Elevation for plots ranged from 3248 ft to 8655 ft and was 7,105 ft on average. Most plots were on southeast-facing slopes, and the most common site disturbance was fire with 5 sites experiencing fire in previous years.

### 2.4.3 Current health status of surveyed trees

*Health score frequency.* After cleaning the data, n=382 trees had valid health scores (1-5) and state labels. Overall counts (percent of total) were: score 1 = 176 (46.07%), score 2 = 77 (20.16%), score 3 = 55 (14.4%), score 4 = 47 (12.3%), and score 5 = 27 (7.07%) (**Fig. 5**). Compared to a relative uniform 20% expectation, per-category exact binomial tests with Holm correction indicated that trees with a health score of 1 was over represented ($p < 0.001$), trees with a health score of 2 did not differ from expected ($p = 0.94$), and scores 3–5 were under-represented (score 3: $p = 0.01$; score 4: $p < 0.001$; score 5: $p < 0.001$). The state × score chi-square test was significant ($\chi^2(16) = 83.23$, $p < 0.001$), indicating that score distribution was significantly different across states. After the Holm adjustment across 25 state×score tests, only a few cells remained significant: California had fewer tree health scores of 3 and more of 4 than expected (both $p < 0.001$); Colorado had fewer tree health scores of 1 ($p < 0.01$) and more 2s ($p < 0.05$); Idaho had more 3s ($p < 0.01$). No individual scores in Montana or Wyoming were significant after correction (**Fig. 6**).

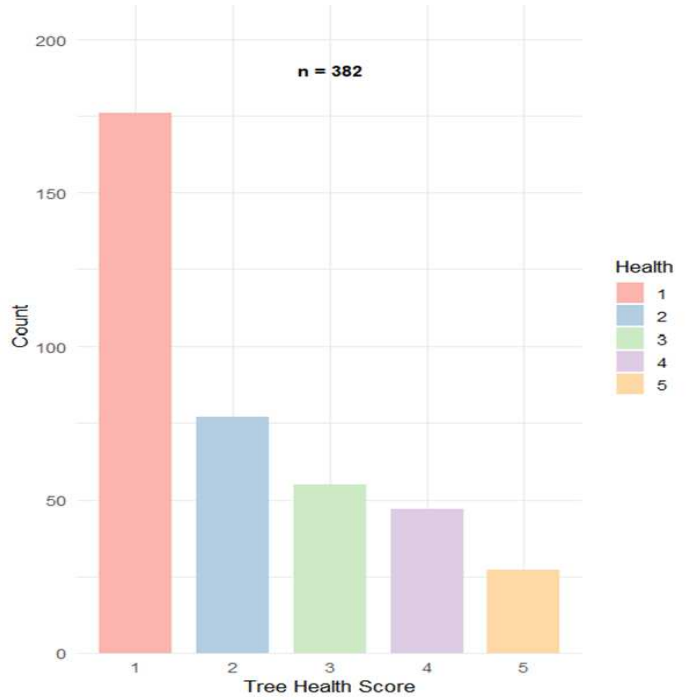


**Figure 5**. Tree health score distribution across all plots. Health scores are as follows: (1) healthy: <15% damage to crown/stem, (2) declining: 16-50% damage to crown/stem, (3) dying: >50% damage to crown/stem, (4) recently dead: no green needles, red needles/fine twigs present, (5) grey dead: fine twigs, no needles

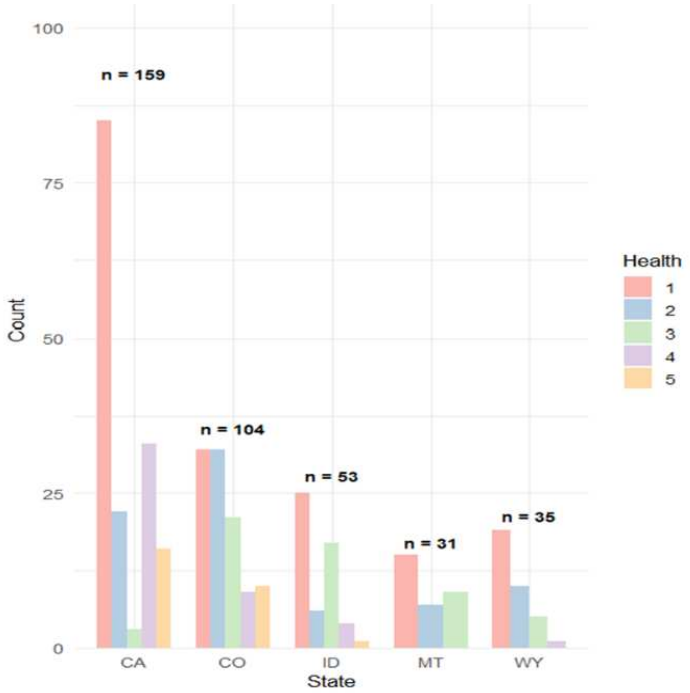

**Figure 6**. Tree health score distribution stratified by state. CA=California, CO=Colorado, ID=Idaho, MT=Montana, WY=Wyoming. Health scores are as follows: (1) healthy: <15% damage to crown/stem, (2) declining: 16-50% damage to crown/stem, (3) dying: >50% damage to crown/stem, (4) recently dead: no green needles, red needles/fine twigs present, (5) grey dead: fine twigs, no needles

2.4.4 Biotic factor assessment

*Pathogen frequency.* Across all sites, detections were dominated by *Sydowia ploysspora*, which was the most observed taxon. The second tier consisted of other frequently observed pathogens such as *Diplodia sapinea, Leptographium* spp., *Cytospora* spp., *Ophiostoma* spp., and Western gall rust. Rarely observed taxa, typically less than 5 detections across all sites, included *Biscogniauxia mediterrania, Bursaphelenchus xylophilus, Cenangium ferruginosum, Epicocum nigrum, Lophodermium baculiferum,* and *Thyronectria pinicola* (**Fig. 7).**

State-level patterns showed clear geographical differences in pathogen distribution (**Fig. 8).** Similar to the overall findings, *S. polyspora* was found in every state and was the most frequently detected species in Colorado, Idaho, Montana, and Wyoming. California had a different pathogen profile, with *Leptographium* spp. having the most detections followed by *D. sapinea, Ophiostoma* spp., and Western gall rust. *Cytospora* spp. was identified consistently across multiple states with moderate frequencies, while *Elytroderma deformans* and dwarf mistletoe were localized in Idaho and Colorado, respectively. The rare taxa listed above was infrequent in every state panel and in several cases, absent from most states.

The pathogen x health score tables corroborate these distributions. *S. polyspora* emerged as the most frequent taxon and was highly concentrated in moderately declining trees in every state. *Leptographium* spp. and *Ophiostoma* spp. had fewer detections but were disproportionately represented among dying trees, almost entirely in California. *D. sapinea* was regularly detected

but skewed towards moderate decline. Western gall rust was confined to trees with health scores of 1-2. *Cytospora* spp. was isolated from trees showing moderate and dying trees in California, Colorado, and Wyoming and *Elytroderma deformans* was localized in Idaho and was identified most on trees with health scores of 3. Rare taxa were sparse and showed no consistent state- or tree score-specific concentration. These two-way tables link pathogen frequency to severity by showing how often each pathogen occurs in each health category (**Table 1; Supp. Table 1**).

Additionally, 26 trees (6.8%) were found to have multiple infections from fungal pathogens. These trees represented a small subset of the total 384 trees but tended to be in poorer conditions than the overall populations with an average health score of 2.9.

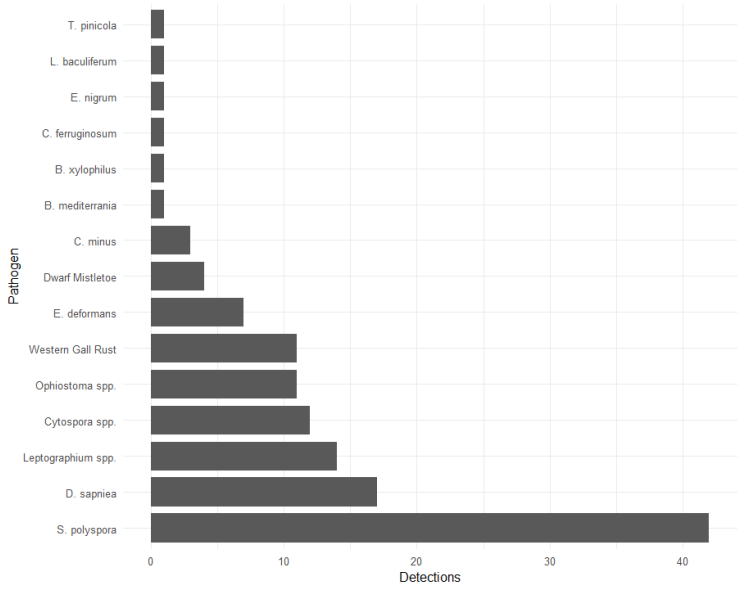


**Figure 7.** *Overall pathogen detections*. Horizontal bars show the number of trees with a positive detection for each pathogen across the study dataset. Taxa are ordered by total detection. Pathogens are as follows (top to bottom): *Thyronectria pinicola, Lophodermium baculiferum, Epicocum nigrum, Cenangium ferruginosum, Bursaphelenchus xylophilus, Biscogniauxia mediterrania, Cyclaneusma minus, Dwarf mistletoe, Elytroderma deformans,* Western gall rust*, Ophiostoma* spp.*, Cytospora* spp. *Leptographium* spp.*, Diplodia sapinea, Sydowia polyspora.*

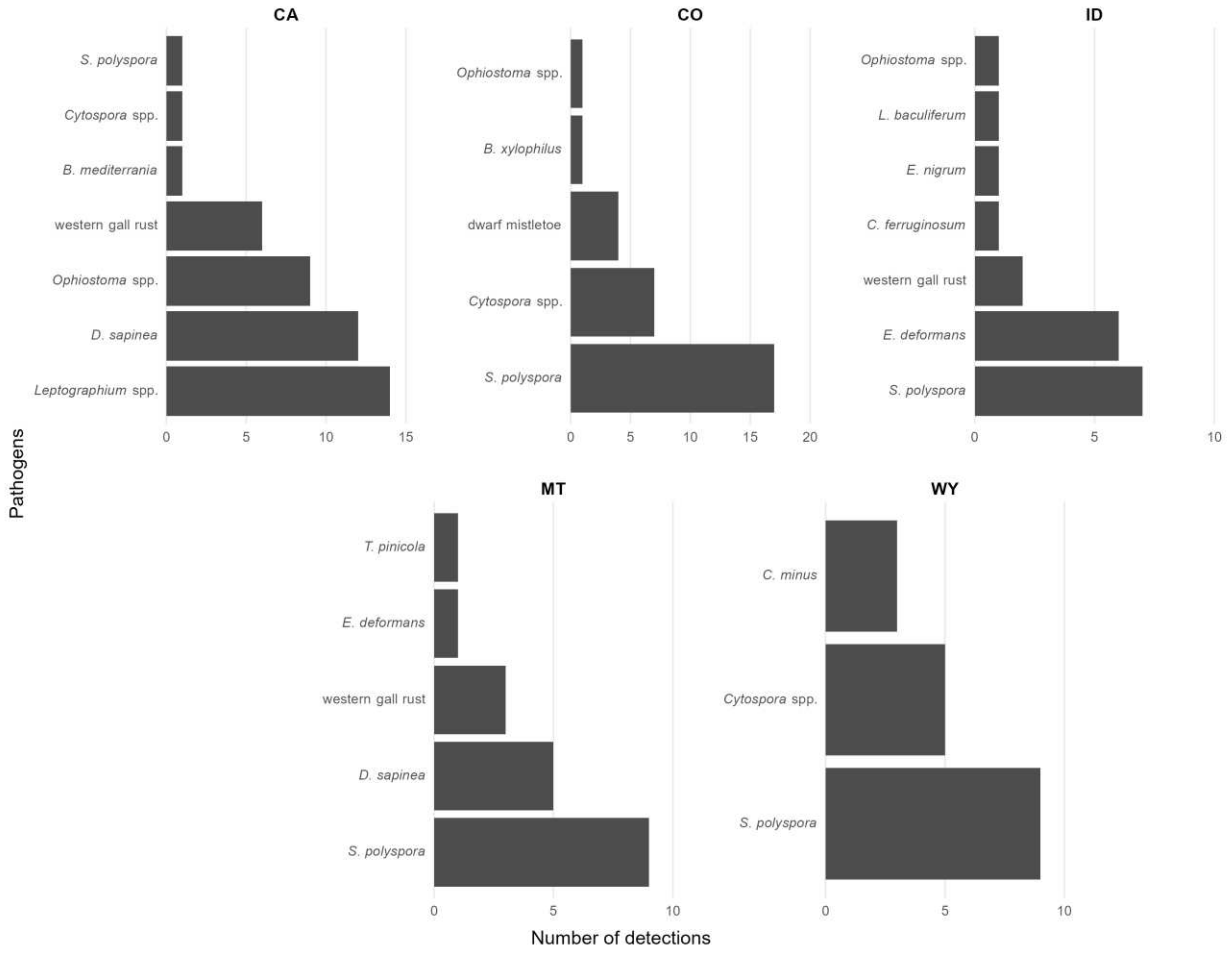


**Figure 8.** *Pathogen detections by state*. Horizontal bar charts show positive detections for each pathogen within states. Within each panel, pathogens are ordered by frequency. Pathogens without any detection were removed. Axes: x=detections; y=pathogen

**Table 1.** Two-way table showing pathogen frequency for each tree health score across all plots.

| Pathogen | 1 | 2 | 3 | 4 | 5 |
|---|---|---|---|---|---|
| *E. deformans* | 1 | 1 | 5 | 0 | 0 |
| *Leptographium* spp. | 0 | 1 | 1 | 11 | 1 |
| *Ophiostoma* spp. | 1 | 0 | 1 | 8 | 1 |
| *C. ferrugnosum* | 1 | 0 | 0 | 0 | 0 |

| | | | | | |
|---|---|---|---|---|---|
| *D. sapinea* | 4 | 8 | 3 | 2 | 0 |
| *B. xylophilus* | 0 | 0 | 0 | 0 | 1 |
| *B. mediterranea* | 0 | 0 | 0 | 1 | 0 |
| *T. pinicola* | 0 | 0 | 1 | 0 | 0 |
| *E. nigrum* | 0 | 0 | 1 | 0 | 0 |
| Western gall rust | 6 | 4 | 1 | 0 | 0 |
| *Cytospora* spp. | 1 | 5 | 3 | 1 | 2 |
| Dwarf mistletoe | 2 | 1 | 1 | 0 | 0 |
| *C. minus* | 0 | 2 | 1 | 0 | 0 |
| *L. baculiferum* | 0 | 0 | 1 | 0 | 0 |
| *S. polyspora* | 2 | 20 | 18 | 2 | 0 |

**Note:** Health scores are as follows: (1) healthy: <15% damage to crown/stem, (2) declining: 16-50% damage to crown/stem, (3) dying: >50% damage to crown/stem, (4) recently dead: no green needles, red needles/fine twigs present, (5) grey dead: fine twigs, no needles. Pathogens are as follows (top to bottom): *Elytroderma deformans, Leptographium* spp., *Ophiostoma* spp., *Cenangium ferruginosum, Diplodia sapinea, Bursaphelenchus xylophilus, Biscogniauxia mediterrania, Thyronectria pinicola, Epicocum nigrum,* Western gall rust, *Cytospora* spp., Dwarf mistletoe*, Cyclaneusma minus, Lophodermium baculiferum, Sydowia polyspora*

*Pathogen focused subset.* Analysis of the pathogen focused subset resulted in significant associations between pathogen presence and disease severity. *Sydowia polyspora* was negatively associated with dead trees (health scores 4-5) and occurred more frequently in moderately affected stands (health scores 2-3). The odds of dead trees being present were substantially lower when *S. polyspora* was present (OR = 0.02, 95% CI: 0.01-0.55). This relationship was statistically significant after controlling multiple comparisons (p_bh < 0.02) (**Table 3**).

In contrast, vascular pathogens showed strong positive associations with dying trees. *Leptographium* spp. were more present in dead or dying trees (health scores 4-5) compared to only moderately affected trees (health scores 2-3). The presence of *Leptographium* spp. was

associated with an increase in odds of dying trees (OR = 76, 95% CI: 12.6-878) and was highly significant following a false discovery rate correction (p_bh < 0.001)

Similarly, *Ophiostoma* spp. were also highly present in dying trees with an increase in odds (OR = 24, 95% CI: 4.4-255). The result after BH adjustment was significant (p_bh < 0.001) (**Table 3**).

**Table 3. Results of the pathogen focus subset Fisher's exact test between pathogen presence and disease severity.**

| Pathogen | adj. p (FDR) | Odds Ratio (95% CI) |
|---|---|---|
| ***Leptographium* spp.** | <0.001 | 76 (12.6–878) |
| ***Ophiostoma* spp.** | <0.001 | 24 (4.4–255) |
| ***S. polyspora*** | 0.02 | 0.12 (0.01–0.55) |

**Note:** Pathogen name followed by the Benjamini–Hochberg adjusted p-value for significance and odds ratio of severity with 95% confidence interval.

At the state level, patterns were much more variable. In California, *Diplodia sapinea* was disproportionately associated with moderately affected stands. The pathogen was present in 28.0% of moderately diseased trees but only 4.1% of dead or drying trees, corresponding to a significant reduction in the odds when present (OR = 0.11, 95% CI: 0.02–0.58; p_bh = 0.039). The OR < 1 indicates trees with *D. sapinea* had reduced odds of being present on dead or dying trees and were more often in moderate categories. For the remaining pathogens, though several remained suggestive ($0.05 < q \leq 0.10$), no other state-level associations survived FDR correction ($q > 0.05$), so we cannot infer robust geographic effects from these data (**Table 4**).

**Table 4. Results of the pathogen focus subset Fisher's exact test between pathogen presence and disease severity by state**

| State | Pathogen | *p* | test | OR (95% CI) |
|---|---|---|---|---|

| | | | | |
|---|---|---|---|---|
| **CA** | *Diplodia sapinea* | 0.04 | Fisher | 0.11 [0.02, 0.58] |

**Note:** Pathogen name followed by the Benjamini–Hochberg adjusted p-value for significance and odds ratio of severity with 95% confidence interval.

2.4.5 Statistical analysis

*Correlation analysis between tree health and abiotic factors.* For the correlation analysis of the anomaly (5-25) window, two signals emerged overall across all sites (n=26): health score increased with mean and temperature anomaly ($\rho = 0.45$; $p = 0.02$) and decreased with precipitation anomaly ($\rho = -0.41$; $p = 0.04$) (**Table 5**). No other anomaly predictors were significant at $\alpha = 0.05$. These results indicate that warmer sites and sites increased in temperature compared to the 25-year baseline tended to have higher health scores, while sites that were wetter compared to 25-year baseline tended to have lower health scores over the same period.

**Table 5. Results of the correlation analysis between tree health and abiotic factors**

| **Window** | **Predictor** | **Rho** | ***p*** |
|---|---|---|---|
| **Anomaly (5–25)** | Temp (mean) [5to25] | 0.45 | 0.02 |
| **Anomaly (5–25)** | Precip [5to25] | -0.41 | 0.04 |

**Note:** Time window used for the correlation analysis (5-25 anomoly), abiotic predictor significantly correlated with health score, rho refers to the correlation coefficient, p-value for significance.

*Network analysis for the top pathogens found.* The five pathogens chosen based on highest frequency in all plots were *Sydowia polyspora, Diplodia sapinea, Leptographium* spp., *Cytospora* spp., and *Ophiostoma* spp. The Spearman correlation network analysis revealed three

significant associations between *Sydowia polyspora* occurrence and site level variables (**Fig. 13**). The pathogen was positively correlated with average precipitation ($\rho = 0.58$, $p = 0.002$), suggesting higher prevalence in wetter conditions. Elevation was also positively correlated with *S. polyspora* ($\rho = 0.41$, $p = 0.04$), suggesting higher occurrence at sites with higher altitudes. In contrast, a negative correlation was observed with basal area ($\rho = –0.52$, $p = 0.006$), showing that the pathogen was less frequent in denser stands (**Supp. Table 2**). Collectively, these results suggest that *S. polyspora* is more commonly associated with cooler, higher elevation sites with lower stand densities.

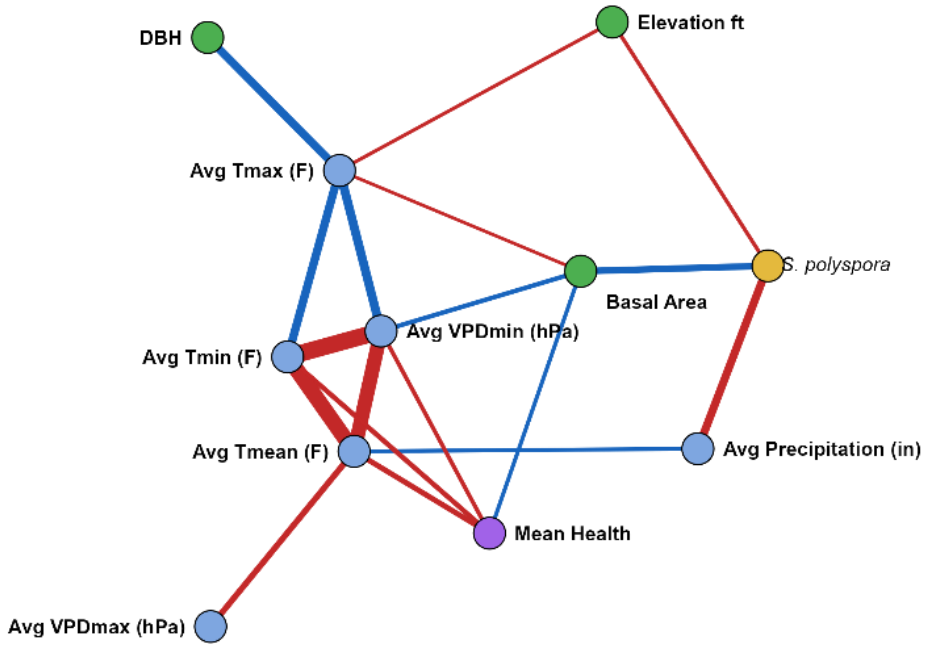


**Figure 13.** Network analysis – *Sydowia polyspora.* Undirected correlation networks summarizing relationships between climate (blue nodes), site factors (green), focal pathogen (gold), and mean health (purple). Nodes are site-level variables and edges connect pairs with Spearman correlations meeting $|\rho| \geq 0.30$ and $p < 0.05$. Line color encodes direction (red = positive, blue = negative and line width scales with $|\rho|$).

For *Diplodia sapinea*, the Spearman network analysis identified three significant correlations between occurrence and environmental factors (**Fig. 14**) A moderate negative correlations was observed with elevation ($\rho = –0.51$, $p = 0.007$), showing that the pathogen was

more prevalent at lower elevation sites. A negative association with maximum VPD (ρ = –0.43, $p$ = 0.03) and average maximum temperature (ρ = –0.42, $p$ = 0.03) suggested that *D. sapinea* occurrence declines under hotter and drier conditions (**Supp. Table 3**). Together, these findings suggest that *D. sapinea* favors cooler, less water limited environments and becomes less prevalent in sites with higher temperatures and atmospheric dryness.

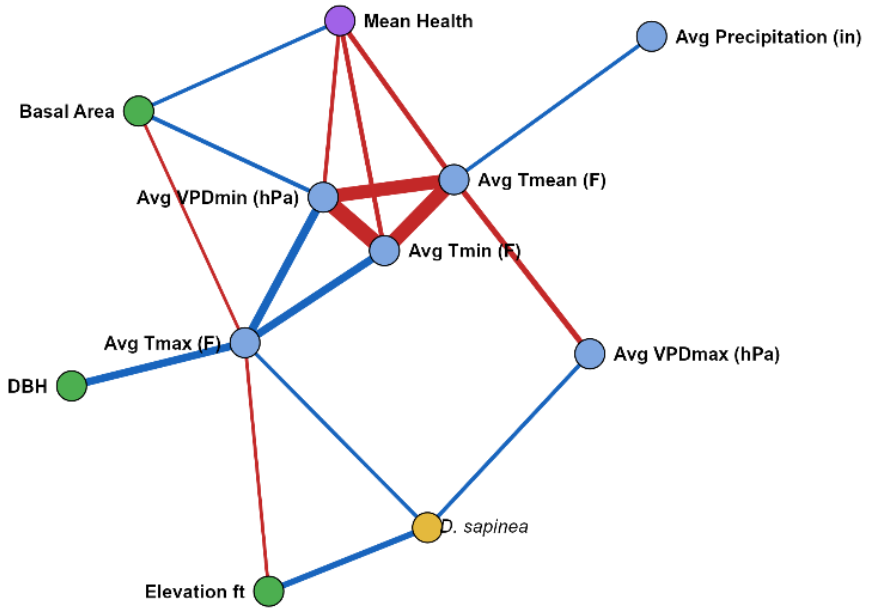


**Figure 14.** Network analysis – *Diplodia sapinea.* Undirected correlation networks summarizing relationships between climate (blue nodes), site factors (green), focal pathogen (gold), and mean health (purple). Nodes are site-level variables and edges connect pairs with Spearman correlations meeting $|\rho| \geq 0.30$ and $p < 0.05$. Line color encodes direction (red = positive, blue = negative and line width scales with $|\rho|$).

The Spearman correlation network showed that *Leptographium* spp. were significantly associated with several environmental and host factors (**Fig. 15**). Occurrence was negatively associated to elevation (ρ = –0.63, $p < 0.001$), indicating a stronger presence at lower altitude sites. In contrast, positive associations emerged with host condition and temperature, including mean health score (ρ = 0.55, $p$ = 0.003), minimum temperature (ρ = 0.52, $p$ = 0.007), and mean temperature (ρ = 0.46, $p$ = 0.02). Maximum VPD showed a weaker but significant positive

relationship (ρ = 0.4, $p$ = 0.04), indicating that the pathogen can persist under drier atmospheric conditions. Conversely, a negative correlation with precipitation (ρ = –0.46, $p$ = 0.02) was found, suggesting that *Leptographium* spp. were less common in wetter environments (**Supp. Table 4**). Collectively, these results suggest that *Leptographium* spp. are most closely associated with warmer, lower elevation forests, where host stress and atmospheric dryness may facilitate colonization.

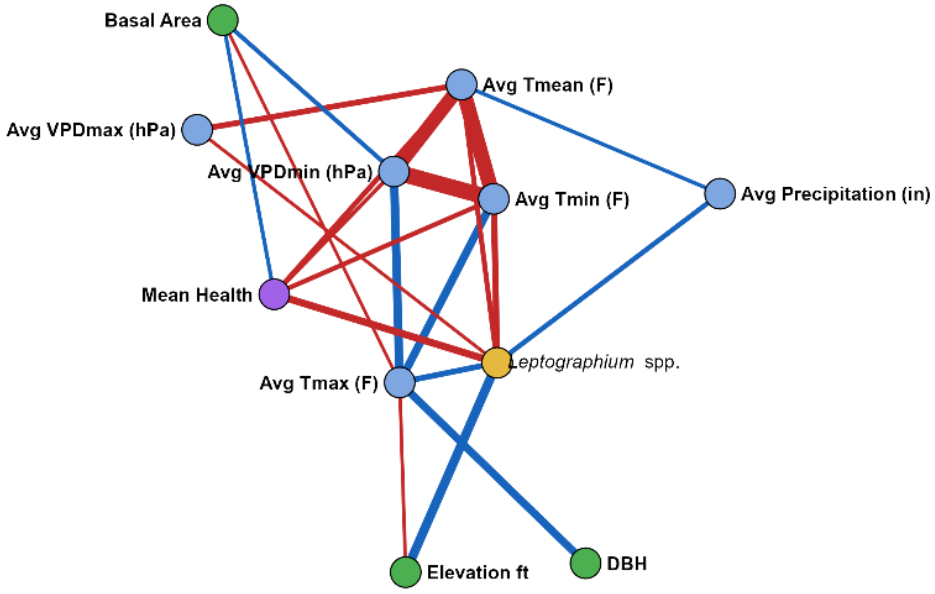


**Figure 15.** Network analysis – *Leptographium* spp. Undirected correlation networks summarizing relatioonships between climate (blue nodes), site factors (green), focal pathogen (gold), and mean health (purple). Nodes are site-level variables and edges connect pairs with Spearman correlations meeting $|\rho| \geq 0.30$ and $p < 0.05$. Line color encodes direction (red = positive, blue = negative and line width scales with $|\rho|$).

The correlation network for *Cytospora* spp. highlighted several significant associations (**Fig. 16**). Pathogen occurrence increased with warmer conditions, showing positive correlations with minimum temperature (ρ = 0.49, $p$ = 0.01) and mean temperature (ρ = 0.47, $p$ = 0.01). *Cytospora* also had as positive relationship with minimum VPD (ρ = 0.44, $p$ = 0.02). In contrast,

*Cytospora* spp. prevalence declined in denser forests, with basal area negatively associated with occurrence ($\rho = -0.48$, $p = 0.01$) (**Supp. Table 5**). These findings suggest that *Cytospora* spp. thrive in warmer, moderately drier environments but are found less in denser stands.

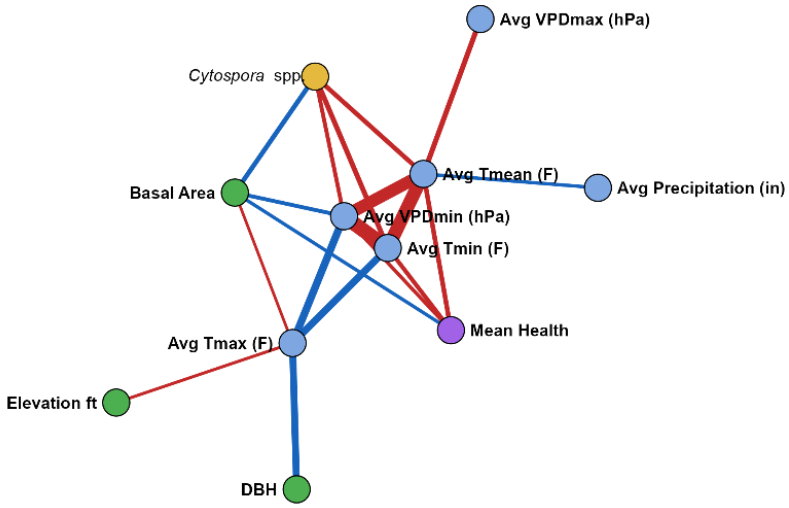


**Figure 16.** Network analysis – *Cytospora* spp. Undirected correlation networks summarizing relationships between climate (blue nodes), site factors (green), focal pathogen (gold), and mean health (purple). Nodes are site-level variables and edges connect pairs with Spearman correlations meeting $|\rho| \geq 0.30$ and $p < 0.05$. Line color encodes direction (red = positive, blue = negative and line width scales with $|\rho|$).

The network analysis of *Ophiostoma* spp. revealed only two significant associations (**Fig. 17**). Occurrence decreased in wetter conditions, as shown by a negative correlation with average precipitation ($\rho = -0.54$, $p = 0.004$). In contrast, the pathogen occurrence was associated with drier atmospheric conditions, highlighted by the positive correlation with maximum VPD ($\rho = 0.44$, $p = 0.02$) (**Supp. Table 6**). These findings indicate that *Ophiostoma* spp. more commonly occur in sites with limited moisture availability, suggesting that water stress plays a central role in disease development.

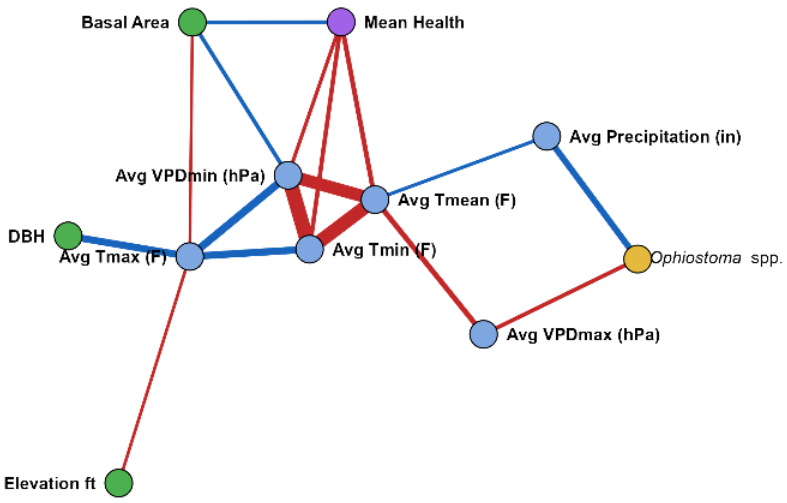


**Figure 17.** Network analysis – *Ophiostoma* spp. Undirected correlation networks summarizing relationships between climate (blue nodes), site factors (green), focal pathogen (gold), and mean health (purple). Nodes are site-level variables and edges connect pairs with Spearman correlations meeting $|\rho| \geq 0.30$ and $p < 0.05$. Line color encodes direction (red = positive, blue = negative and line width scales with $|\rho|$).

## 2.5 Discussion

This study evaluated the health of ponderosa pine across multiple states and examined the interactions of site and stand structure, biotic agents (pathogens), and abiotic stressors (precipitation, temperature, and VPD) leading to dieback and mortality. Broadly, long-term climate signals (warming, changing precipitation patterns, and higher VPD levels) align with previous studies that show recent increases in temperature and aridification across the western United States (Abatzoglou & Williams 2016; Weed et al. 2013). These patterns were reflected in our data, where trees in regions experiencing the most pronounced warming and drying showed greater decline severity.

Patterns of pathogen occurrence and disease severity revealed how biotic agents contribute to decline and tree mortality. Pathogen frequencies varied on trees in different stages of decline (health score) and regionally. Foliar and latent-opportunistic pathogens (e.g., *Sydowia*

*polyspora*, *Cytospora* spp., *Diplodia sapinea*) were most common in stands showing moderate decline (health scores 2-3) while beetle-associated vascular fungi (*Leptographium* spp., *Ophiostoma* spp.) were consistently associated with dying and dead trees (health score 4-5). Further, there were clear significant associations among pathogen presence and severity, and climate-health correlations driven by warming and drying trends. These relationships underscore the differing roles of pathogens in decline, some acting as contributors to decline and others as accelerants once host defenses are lowered. Network analyses of the most frequently identified five pathogens characterized the complex interactions among pathogens, host conditions, and environmental factors. Together, these findings suggest that ponderosa pine decline is a complex phenomenon arising from an interplay of chronic climate stress and opportunistic pathogens.

Climatic stress emerged as a dominant predisposing driver of decline across the western U.S. Multi-decade warming and aridification have predisposed conifers to decline, as the past 30 years show rising temperatures and atmospheric dryness (VPD) alongside decreasing precipitation. These trends were confirmed with the anomaly datasets for all climate factors (5-year minus 25-year), as departures from the long-term means were documented. Furthermore, more trees in decline were observed in areas where these climate shifts were more extreme. The observed climate patterns are consistent with studies linking regional warming and drying to increased conifer stress, greater disturbance vulnerability, and higher mortality in the western U.S. (Abatzoglou & Williams 2016; Littell et al. 2009). Taken together, the current literature indicates that increased heat and atmospheric dryness (VPD) with less precipitation elevates host stress directly and indirectly heightening disturbance from biotic agents, resulting in tree health declines and mortality that was observed in our study.

Network analyses provided perspective on how climate and pathogens interact by combining climate anomalies, stand structure, and pathogen detections. From these analyses, two groups of pathogens were formed based on how they responded to climate factors. One group was comprised of latent-opportunistic taxa which included foliar or stress-induced canker pathogens. These pathogens, *Sydowia polyspora, Cytospora* spp*.,* and *Diplodia sapinea,* were linked to moderately declining trees, but were found in different abiotic niches depending on the pathogen. The second group included vascular, beetle associated blue-stain fungi, *Leptographium* spp. and *Ophiostoma* spp. These fungal genera were more associated with hotter and drier climate variables, stands at lower elevations, and with dying and dead trees.

Latent-opportunistic pathogens displayed climatic and geographic variability consistent with their ecology. *S. polyspora* was associated with cooler, wetter nodes (positive with precipitation and elevation; negative with basal area). This is consistent with its endophytic lifestyle and its propensity to cause disease during moist infection windows following host stress (Ridout & Newcombe 2018; Sieber 2007; Talgø et al. 2010). *D. sapinea,* likewise, was found associated with cooler, more humid conditions (negative correlations with VPDmax and Tmax) in the network analyses. These analyses concur with the well-documented fungal biology of these pathogens that includes host stress-responsive and opportunistic behaviors on pine. Likewise, foliar pathogens have the tendency to spike with short, cool-wet periods that promote sporulation and shoot infection (Stanosz et al. 2001; Wingfield et al. 2025; Pandit et al. 2020). *Cytospora* spp. showed a different abiotic correlation where detections increased with higher temperatures and atmospheric dryness and decreased with denser stands (positive with temperature and VPD and negative with basal area). This pattern matches stress-canker ecology, where drought, heat, and wounding lower host defenses and accelerate lesion development

(Schoeneweiss 1975, 1981, 1983; Sinclair et al. 1987; Dudley et al. 2020; Kamiri & Laemmlen 1981). Taken together, the pathogen-specific networks indicate that climate does not act uniformly on disease development.

Vascular, beetle-associated fungi represented the terminal stage of decline. *Leptographium* and *Ophiostoma* had a greater presence at lower elevation, positive associations with heat and dryness (VPDmax for *Leptographium*; lower precipitation for *Ophiostoma*) and concentrated in dead and dyingtrees. Although we deliberately avoided sites with obvious bark beetle damage to focus on mortality from pathogens, these fungi were still found, consistent with their vector-mediated ecology (Six & Wingfield 2011; Linnakoski et al. 2012; Chang et al. 2017). Beetle colonization is coordinated by aggregation pheromones, and outbreak-level densities can overpower host defenses. However, host condition remains important as drought-weakened trees are generally more susceptible when beetle populations are high (Wallin & Raffa 2004; Raffa et al. 2008; Kolb et al. 2019). Even limited or transient beetle activity can inoculate trees with ophiostomatoid fungi (Ballard et al. 1982; Six & Wingfield 2011). Once introduced, rapid sapwood colonization and xylem blockage provide a direct hydraulic pathway to decline and tree mortality (Hansen & Lewis, 1997; Krokene & Solheim, 1998; Arango-Vélez et al., 2016). Warming and drought amplify this vector pathway by boosting beetle survival and flight windows and expanding their elevation and latitudinal ranges, allowing their fungal associates to expand to new areas (Bentz et al. 2010; Carroll et al. 2003; Seidl et al. 2014). This helps explain the concentration of *Leptographium* and *Ophiostoma* in hotter, drier, lower-elevation sites and their tight linkage with severely declining and dead trees despite our conservative site selection.

Results from our study suggest that these interactions among pathogen, climate, and host stress fit the classic decline-disease model. Ponderosa pine decline likely starts with predisposing

factors, specifically long-term warming, higher VPD, and site conditions that result in chronically stressed trees. Following these long-term trends, acute inciting factors like heat waves, multi-month droughts, hail storms, or insect attacks push trees past physiological limits. The pathogens we detected then act mainly as contributing factors. Foliar and latent-opportunistic fungi add shoot loss and cankers or lesions cause disruption in the trees' ability to obtain resources, while beetle-vectored blue or black stain fungi block water transport and speed up decline once infected. Although most trees were infected with a single pathogen, a small portion of trees exhibited two or more fungal infections concurrently (6.8%). These multi-infected trees generally had a higher health score than those infect with just one (2.9). This suggests that co-occurring pathogens may compound physiological stress and exploit weakened hosts. The presence of multiple pathogens on individual trees emphasizes the additive and interactive nature of biotic stress contributing to decline and eventual mortality. Depending on the location, the contributing factors (pathogens) can vary. Climate filters which organisms can thrive by shaping infection windows and vector activity. For example, cool-wet spring periods favor foliar and latent fungi while hot and dry weather boosts bark beetle flights and success, bringing blue stain fungi. Furthermore, several agents have limited native ranges or depend on vectors within their own geographical limits. As a result, the pathogens that are present locally reflect where these fungi and beetles can exist. Local site factors, such as elevation, aspect, slope, and past disturbances also play a role in the presence of these pathogens. These factors can shift microclimates enough to change which pathogen dominates on a site. The results suggest a classic decline disease where the predisposing factors include chronic climate stress and exposure. These primary drivers of ponderosa pine decline are followed by inciting events (acute drought/heat and beetle pulses) that push trees over the edge. Finally, contributing factors, such

as foliar, canker, or vascular pathogens, accelerate the decline rather than serving as the primary cause by amplifying damage and speeding dieback and producing the regional patterns of ponderosa pine decline and mortality.

Several limitations should be considered when interpreting the results of this study. First, although the PRISM climate data provides high quality, long-term climate estimates, topographic variation can be overlooked, potentially obscuring microclimatic conditions in individual stands, especially on mountainous terrain. Second, this study did not incorporate data on insect activity or abundance. Given the strong ecological interactions between bark beetles, root weevils, and fungal pathogens, the absence of insect data limits inferences about biotic interactions driving decline. Third,the site selection methods may have introduce sampling bias, as plots were established in landscapes exhibiting visible symptoms of decline. This could overrepresent stressed stands even though healthy trees were measured within each plot to help mitigate the bias. Finally, regional variation in ponderosa pine subspecies and the presence of Jeffery pine in parts of California introduce potential physiological and ecological differences, such as drought tolerance and host specificity to pathogen, that may influence disease dynamics and health outcomes (Willyard et al. 2021). These factors collectively highlight the need for future work integrating climate data that takes into account topography and represents microclimates within a stand, insect monitoring, and subspecies level analyses to refine understanding of ponderosa pine decline drivers across the west.

Taken together, the results indicate that climate induces ponderosa pine vulnerability, while the effects of already present biotic agents are amplified by climate. In hot, dry conditions, blue stain fungi colonize the sapwood, disrupt water transport, and were most often linked to trees under severe decline and tree mortality. In contrast, during cooler and wet conditions, foliar

and latent opportunistic pathogens were more prominent, typically resulting in moderately declining trees (defoliations and branch dieback) that can intensify under extended heat and drought, but this rarely kills the tree alone. Regional patterns of pathogen prevalence and tree decline likely arise from interacting effects of climate and biogeography. In California, blue stain fungi are established, and hotter, drier conditions amplify their activity. Likewise, in the Rocky Mountain region, foliar and canker pathogens are broadly present, but regional climates intensify disease severity. *Cytospora* spp. become more severe under higher temperatures and vapor pressure deficients, whereas *S. polyspora* and *D. sapinea* show greater severity under cooler, wetter climates. These differences reflect that pathogens are in different locations, and climatic conditions within those regions explain the observed difference in prevalence and severity. Overall, the evidence suggests that regional climate, pathogen behavior, and their native range, differ throughout the western U.S. and contribute to different levels of ponderosa pine decline.

REFERENCES

Abatzoglou, J. T., & Williams, A. P. (2016). Impact of anthropogenic climate change on wildfire across western US forests. *Proceedings of the National Academy of Sciences, 113*(42), 11770–11775. https://doi.org/10.1073/pnas.1607171113

Allen, C. D., Macalady, A. K., Chenchouni, H., *et al.* (2010). A global overview of drought and heat-induced tree mortality reveals emerging climate change risks for forests. *Forest Ecology and Management, 259*(4), 660–684. https://doi.org/10.1016/j.foreco.2009.09.001

Arango-Vélez, A., El Kayal, W., Copeland, S., Lusebrink, I., Cooke, J. E. K., & Erbilgin, N. (2016). Differences in defence responses of *Pinus contorta* and *Pinus banksiana* to the mountain pine beetle fungal associate *Grosmannia clavigera* are affected by water deficit. *Plant, Cell & Environment, 39*(4), 726–744. https://doi.org/10.1111/pce.12615

Ballard, R. G., Walsh, M. A., & Cole, W. E. (1982). Blue-stain fungi in xylem of lodgepole pine: A light-microscope study on extent of hyphal distribution. *Canadian Journal of Botany, 60*(11), 2334–2341. https://doi.org/10.1139/b82-285

Benjamini, Y., & Hochberg, Y. (1995). Controlling the false discovery rate: A practical and powerful approach to multiple testing. *Journal of the Royal Statistical Society: Series B (Methodological), 57*(1), 289–300. https://doi.org/10.1111/j.2517-6161.1995.tb02031.x

Bentz, B. J., Régnière, J., Fettig, C. J., Hansen, E. M., Hayes, J. L., Hicke, J. A., Kelsey, R. G., Negrón, J. F., & Seybold, S. J. (2010). Climate change and bark beetles of the western United States and Canada: Direct and indirect effects. *BioScience, 60*(8), 602–613. https://doi.org/10.1525/bio.2010.60.8.6

Carroll, A. L., Taylor, S. W., Régnière, J., & Safranyik, L. (2003). Effect of climate change on range expansion by the mountain pine beetle in British Columbia. In *Mountain Pine Beetle Symposium: Challenges and Solutions* (Kelowna, BC). https://digitalcommons.usu.edu/barkbeetles/195

Chang, R., Duong, T. A., Taerum, S. J., Wingfield, M. J., Zhou, X., & de Beer, Z. W. (2017). Ophiostomatoid fungi associated with conifer-infesting beetles and their phoretic mites in

Yunnan, China. *MycoKeys, 28*, 19–64. https://doi.org/10.3897/mycokeys.28.21758

Ciesla, W. M., Eglitis, A., & Hanavan, R. (2010). *Pandora moth* (Forest Insect & Disease Leaflet 114). U.S. Department of Agriculture, Forest Service, Forest Health Protection. https://www.fs.usda.gov/foresthealth/docs/fidls/FIDL-114-PandoraMoth.pdf

Davis, K. T., Dobrowski, S. Z., Higuera, P. E., *et al.* (2019). Wildfires and climate change push low-elevation forests across a critical climate threshold for tree regeneration. *Proceedings of the National Academy of Sciences, 116*(13), 6193–6198. https://doi.org/10.1073/pnas.1815107116

Davis, K. T., Robles, M. D., Kemp, K. B., Higuera, P. E., Chapman, T., Metlen, K. L., *et al.* (2023). Reduced fire severity offers near-term buffer to climate-driven declines in conifer resilience across the western United States. *Proceedings of the National Academy of Sciences, 120*(11), e2208120120. https://doi.org/10.1073/pnas.2208120120

Desprez-Loustau, M.-L., Marçais, B., Nageleisen, L.-M., Piou, D., & Vannini, A. (2006). Interactive effects of drought and pathogens in forest trees. *Annals of Forest Science, 63*(6), 597–612. https://doi.org/10.1051/forest:2006040

Dudley, M. M., Tisserat, N. A., Jacobi, W. R., Negrón, J., & Stewart, J. E. (2020). Pathogenicity and distribution of two species of *Cytospora* on *Populus tremuloides* in portions of the Rocky Mountains and Midwest in the United States. *Forest Ecology and Management, 468*, 118168. https://doi.org/10.1016/j.foreco.2020.118168

Filip, G. M., Klopfenstein, N. B., Maffei, H. M., Shaw, C. G., III, & Lockman, B. (2024). *Armillaria root disease in conifers of western North America* (Forest Insect and Disease Leaflet 188). U.S. Department of Agriculture, Forest Service, Forest Health Protection. https://research.fs.usda.gov/treesearch/68329

Hansen, E. M., & Lewis, K. J. (Eds.). (1997). *Compendium of conifer diseases*. APS Press.

Heikrujam, J., Kishor, R., & Mazumder, P. B. (2020). The chemistry behind plant DNA isolation protocols. In *Biochemical Analysis Tools—Methods for Bio-Molecules Studies* (pp. 1–18). IntechOpen. https://doi.org/10.5772/intechopen.92206

Hoffman, C. M., Mathiasen, R. L., & Sieg, C. H. (2007). Dwarf mistletoe effects on fuel

loadings in ponderosa pine forests in northern Arizona. *Canadian Journal of Forest Research, 37*(3), 662–670. https://doi.org/10.1139/X06-259

Kamiri, L. K., & Laemmlen, F. F. (1981). Effects of drought-stress and wounding on *Cytospora* canker development on Colorado blue spruce. *Journal of Arboriculture, 7*(5), 113–116. https://auf.isa-arbor.com/content/7/5/113

Kamiri, L. K., & Laemmlen, F. F. (1981). Epidemiology of *Cytospora* canker caused in Colorado blue spruce by *Valsa kunzei*. *Phytopathology, 71*(9), 941–947. https://doi.org/10.1094/Phyto-71-941

Kikuchi, T., Aikawa, T., Oeda, Y., Karim, N., & Kanzaki, N. (2009). A rapid and precise diagnostic method for detecting the pinewood nematode *Bursaphelenchus xylophilus* by loop-mediated isothermal amplification. *Phytopathology, 99*(12), 1365–1369. https://doi.org/10.1094/PHYTO-99-12-1365

Kolb, T. E., Keefover-Ring, K., Burr, S. J., Hofstetter, R., Gaylord, M., & Raffa, K. F. (2019). Drought-mediated changes in tree physiological processes weaken tree defenses to bark beetle attack. *Journal of Chemical Ecology, 45*(10), 888–900. https://doi.org/10.1007/s10886-019-01105-0

Kozhar, O., Ibarra Caballero, J. R., Burns, K. S., & Stewart, J. E. (2023). Field ready: Development of a rapid LAMP-based colorimetric assay for the causal agent of white pine blister rust, *Cronartium ribicola*. *Forest Pathology, 53*, e12814. https://doi.org/10.1111/efp.12814

Krokene, P., & Solheim, H. (1998). Pathogenicity of four blue-stain fungi associated with aggressive and nonaggressive bark beetles. *Phytopathology, 88*(1), 39–44. https://doi.org/10.1094/PHYTO.1998.88.1.39

Littell, J. S., McKenzie, D., Peterson, D. L., & Westerling, A. L. (2009). Climate and wildfire area burned in western U.S. ecoprovinces, 1916–2003. *Ecological Applications, 19*(4), 1003–1021. https://doi.org/10.1890/07-1183.1

Linnakoski, R., de Beer, Z. W., Niemelä, P., & Wingfield, M. J. (2012). Associations of conifer-infesting bark beetles and fungi in Fennoscandia. *Insects, 3*(1), 200–227.

https://doi.org/10.3390/insects3010200

Manion, P. D., & LaChance, D. (1992). Forest decline concepts: An overview. In P. D. Manion & D. LaChance (Eds.), *Forest decline concepts* (pp. 181–190). St. Paul, MN: APS Press.

Oester, P. T., Shaw, D. C., & Filip, G. M. (2018). *Managing insects and diseases of Oregon conifers* (EM 8980). Oregon State University Extension Service. https://catalog.extension.oregonstate.edu/em8980

O'Neill, L., Fulé, P. Z., & Hofstetter, R. W. (2023). Multi-century reconstruction of Pandora moth outbreaks at the warmest/driest edge of a wide-ranging *Pinus* species. *Forests, 14*(3), 444. https://doi.org/10.3390/f14030444

Pandit, K., Smith, J., Quesada, T., Villari, C., & Johnson, D. J. (2020). Association of recent incidence of foliar disease in pine species in the southeastern United States with tree and climate variables. *Forests, 11*(11), 1155. https://doi.org/10.3390/f11111155

Raffa, K. F., Aukema, B. H., Bentz, B. J., Carroll, A. L., Hicke, J. A., Turner, M. G., & Romme, W. H. (2008). Cross-scale drivers of natural disturbances prone to anthropogenic amplification: The dynamics of bark beetle eruptions. *BioScience, 58*(6), 501–517. https://doi.org/10.1641/B580607

Ridout, M., & Newcombe, G. (2018). *Sydowia polyspora* is both a foliar endophyte and a preemergent seed pathogen in *Pinus ponderosa*. *Plant Disease, 102*(3), 640–644. https://doi.org/10.1094/PDIS-07-17-1074-RE

Schoeneweiss, D. F. (1975). Predisposition, stress, and plant disease. *Annual Review of Phytopathology, 13*, 193–211. https://doi.org/10.1146/annurev.py.13.090175.001205

Schoeneweiss, D. F. (1981). The role of environmental stress in diseases of woody plants. *Plant Disease, 65*(4), 308–314. https://doi.org/10.1094/PD-65-308

Schoeneweiss, D. F. (1983). Drought predisposition to *Cytospora* canker in blue spruce. *Plant Disease, 67*(4), 383–385. https://apsnet.org/publications/PlantDisease/BackIssues/Documents/1983Articles/PlantDisease67n04_383.pdf

Seidl, R., Schelhaas, M.-J., Rammer, W., & Verkerk, P. J. (2014). Increasing forest disturbances

in Europe and their impact on carbon storage. *Nature Climate Change, 4*(9), 806–810. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4340567/

Shaw, D. C., & Agne, M. C. (2017). Fire and dwarf mistletoe (Viscaceae: *Arceuthobium* species) in western North America: Contrasting *Arceuthobium tsugense* and *Arceuthobium americanum*. *Botany, 95*(3), 231–246. https://doi.org/10.1139/cjb-2016-0245

Sieber, T. N. (2007). Endophytic fungi in forest trees: Are they mutualists? *Fungal Biology Reviews, 21*(2–3), 75–89. https://doi.org/10.1016/j.fbr.2007.05.004

Sinclair, W. A., Lyon, H. H., & Johnson, W. T. (1987). *Diseases of trees and shrubs*. Ithaca, NY: Comstock Publishing Associates, Cornell University Press.

Six, D. L., & Wingfield, M. J. (2011). The role of phytopathogenicity in bark beetle–fungus symbioses: A challenge to the classic paradigm. *Annual Review of Entomology, 56*, 255–272. https://doi.org/10.1146/annurev-ento-120709-144839

Stanosz, G. R., Blodgett, J. T., Smith, D. R., & Kruger, E. L. (2001). Water stress and *Sphaeropsis sapinea* as a latent pathogen of red pine seedlings. *New Phytologist, 149*(3), 531–538. https://doi.org/10.1046/j.1469-8137.2001.00052.x

Wallin, K. F., & Raffa, K. F. (2004). Feedback between individual host selection behavior and population dynamics in an eruptive herbivore. *Ecological Monographs, 74*(1), 101–116. https://doi.org/10.1890/02-4004

Walsh, P. S., Metzger, D. A., & Higuchi, R. (1991). Chelex 100 as a medium for simple extraction of DNA for PCR-based typing from forensic material. *BioTechniques, 10*(4), 506–513. https://doi.org/10.2144/000114018

Weed, A. S., Ayres, M. P., & Hicke, J. A. (2013). Consequences of climate change for biotic disturbances in North American forests. *Ecological Monographs, 83*(4), 441–470. https://doi.org/10.1890/13-0160.1

Willyard, A., Gernandt, D. S., Cooper, B., Douglas, C., Finch, K., Karemera, H., Lindberg, E., Langer, S. K., Lefler, J., Marquardt, P., Pouncey, D., & Telewski, F. (2021). *Phylogenomics in the hard pines (Pinus subsection Ponderosae; Pinaceae) confirms paraphyly in Pinus ponderosa, and places Pinus jeffreyi with the California big cone*

*pines. Systematic Botany, 46*(3), 538-561. https://doi.org/10.1600/036364421X16312067913435

Wingfield, M. J., Slippers, B., Barnes, I., Duong, T. A., & Wingfield, B. D. (2025). The pine pathogen *Diplodia sapinea*: Expanding frontiers. *Current Forestry Reports, 11*, 2. https://doi.org/10.1007/s40725-024-00236-2

White, T. J., Bruns, T., Lee, S., & Taylor, J. (1990). Amplification and direct sequencing of fungal ribosomal RNA genes for phylogenetics. In M. A. Innis, D. H. Gelfand, J. J. Sninsky, & T. J. White (Eds.), *PCR protocols: A guide to methods and applications* (pp. 315–322). Academic Press

Woolman, A. M., Coop, J. D., Shaw, J. D., & DeMarco, J. (2022). Extent of recent fire-induced losses of ponderosa pine forests of Arizona and New Mexico, USA. *Forest Ecology and Management, 520*, 120381. https://doi.org/10.1016/j.foreco.2022.120381

APPENDIX

**Supplemental Table 1: Two-way table showing pathogen frequency for each tree health score separated by states.**

| State | CA | | | | | CO | | | | | ID | | | | | MT | | | | | WY | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **Health Score** | **1** | **2** | **3** | **4** | **5** | **1** | **2** | **3** | **4** | **5** | **1** | **2** | **3** | **4** | **5** | **1** | **2** | **3** | **4** | **5** | **1** | **2** | **3** | **4** | **5** |
| *E. deformans* | * | * | * | * | * | * | * | * | * | * | 1 | * | 5 | * | * | * | 1 | * | * | * | * | * | * | * | * |
| *Leptographium* spp. | * | 1 | 1 | 11 | 1 | * | * | * | * | * | * | * | * | * | * | * | * | * | * | * | * | * | * | * | * |
| *Ophiostoma* spp. | 1 | * | * | 8 | * | * | * | * | * | 1 | * | * | 1 | * | * | * | * | * | * | * | * | * | * | * | * |
| *C. ferrugnosum* | * | * | * | * | * | * | * | * | * | * | 1 | * | * | * | * | * | * | * | * | * | * | * | * | * | * |
| *D. sapinea* | 3 | 7 | * | 2 | * | * | * | * | * | * | * | * | * | * | * | 1 | 1 | 3 | * | * | * | * | * | * | * |
| *B. xylophilus* | * | * | * | * | * | * | * | * | * | 1 | * | * | * | * | * | * | * | * | * | * | * | * | * | * | * |
| *B. mediterranea* | * | * | * | 1 | * | * | * | * | * | * | * | * | * | * | * | * | * | * | * | * | * | * | * | * | * |
| *T. pinicola* | * | * | * | * | * | * | * | * | * | * | * | * | * | * | * | * | * | 1 | * | * | * | * | * | * | * |
| *E. nigrum* | * | * | * | * | * | * | * | * | * | * | * | * | 1 | * | * | * | * | * | * | * | * | * | * | * | * |
| Western gall rust | 3 | 3 | * | * | * | * | * | * | * | * | 2 | * | * | * | * | 1 | 1 | 1 | * | * | * | * | * | * | * |
| *Cytospora* spp. | * | * | * | 1 | * | 1 | 2 | 1 | * | 2 | * | * | * | * | * | * | * | * | * | * | * | 3 | 2 | * | * |
| Dwarf mistletoe | * | * | * | * | * | 2 | 1 | 1 | * | * | * | * | * | * | * | * | * | * | * | * | * | * | * | * | * |
| *C. minus* | * | * | * | * | * | * | * | * | * | * | * | * | * | * | * | * | * | * | * | * | * | 2 | 1 | * | * |
| *L. baculiferum* | * | * | * | * | * | * | * | * | * | * | * | * | 1 | * | * | * | * | * | * | * | * | * | * | * | * |
| *S. polyspora* | * | * | * | 1 | * | 2 | 8 | 5 | 1 | * | * | 1 | 6 | * | * | * | 4 | 5 | * | * | * | 7 | 2 | * | * |

**Note:** Health scores are as follows: (1) healthy: <15% damage to crown/stem, (2) declining: 16-50% damage to crown/stem, (3) dying: >50% damage to crown/stem, (4) recently dead: no green needles, red needles/fine twigs present, (5) grey dead: fine twigs, no needles. Pathogens are as follows (top to bottom): *Elytroderma deformans, Leptographium* spp., *Ophiostoma* spp., *Cenangium ferruginosum, Diplodia sapinea, Bursaphelenchus xylophilus, Biscogniauxia mediterrania, Thyronectria pinicola, Epicocum nigrum,* Western gall rust, *Cytospora* spp., Dwarf mistletoe*, Cyclaneusma minus, Lophodermium baculiferum, Sydowia polyspora.* States are as follows: California (CA), Colorado (CO), Idaho (ID), Montana (MT), Wyoming (WY).

**Supplemental Table 2**: Pairwise associations between site/stand or climate variables and *Sydowia polyspora*.

| Variable 1 | Variable 2 | rho | *p* | sign |
|---|---|---|---|---|
| **Avg Precipitation (in)** | *S. polyspora* | 0.58 | 0.002 | positive |
| **Basal Area** | *S. polyspora* | -0.52 | 0.01 | negative |
| **Elevation (ft)** | *S. polyspora* | 0.41 | 0.04 | positive |

**Note:** Variable 1 = abiotic or stand attribute; Variable 2 = pathogen taxon; rho = Spearman correlation coefficient; p = two-sided p-value; sign = direction of the association (positive/negative).

**Supplemental Table 3**: **Pairwise associations between site/stand or climate variables and *Diplodia sapinea***.

| Variable 1 | Variable 2 | rho | *p* | sign |
|---|---|---|---|---|
| **Elevation_ft** | *D. sapinea* | -0.51 | 0.01 | negative |
| **Avg VPDmax (hPa)** | *D. sapinea* | -0.43 | 0.03 | negative |
| **Avg Tmax (F)** | *D. sapinea* | -0.42 | 0.03 | negative |

**Note:** Variable 1 = abiotic or stand attribute; Variable 2 = pathogen taxon; rho = Spearman correlation coefficient; p = two-sided p-value; sign = direction of the association (positive/negative).

**Supplemental Table 4: Pairwise associations between site/stand or climate variables and *Leptographium* spp.**

| Variable 1 | Variable 2 | rho | *p* | sign |
|---|---|---|---|---|
| **Elevation_ft** | *Leptographium* spp. | -0.63 | 0.001 | negative |
| **Mean Health (score)** | *Leptographium* spp. | 0.55 | 0.003 | positive |
| **Avg Tmin (F)** | *Leptographium* spp. | 0.52 | 0.01 | positive |
| **Avg Tmax (F)** | *Leptographium* spp. | -0.49 | 0.01 | negative |
| **Avg Tmean (F)** | *Leptographium* spp. | 0.46 | 0.02 | positive |
| **Avg Precipitation (in)** | *Leptographium* spp. | -0.46 | 0.02 | negative |
| **Avg VPDmax (hPa)** | *Leptographium* spp. | 0.40 | 0.04 | positive |

**Note:** Variable 1 = abiotic, stand, or health attribute; Variable 2 = pathogen taxon; rho = Spearman correlation coefficient; p = two-sided p-value; sign = direction of the association (positive/negative).

**Supplemental Table 5: Pairwise associations between site/stand or climate variables and *Cytospora* spp.**

| Variable 1 | Variable 2 | rho | *p* | sign |
|---|---|---|---|---|
| **Avg Tmin (F)** | *Cytospora* spp. | 0.49 | 0.01 | positive |
| **Basal Area** | *Cytospora* spp. | -0.48 | 0.01 | negative |
| **Avg Tmean (F)** | *Cytospora* spp. | 0.47 | 0.02 | positive |
| **Avg VPDmin (hPa)** | *Cytospora* spp. | 0.44 | 0.02 | positive |

**Note:** Variable 1 = abiotic or stand attribute; Variable 2 = pathogen taxon; rho = Spearman correlation coefficient; p = two-sided p-value; sign = direction of the association (positive/negative).

**Supplemental Table 6: Pairwise associations between site/stand or climate variables and *Ophiostoma* spp.**

| Variable 1 | Variable 2 | rho | *p* | sign |
|---|---|---|---|---|
| **Avg Precipitation (in)** | *Ophiostoma* spp. | -0.54 | 0.004 | negative |
| **Avg VPDmax (hPa)** | *Ophiostoma* spp. | 0.44 | 0.02 | positive |

**Note:** Variable 1 = abiotic or stand attribute; Variable 2 = pathogen taxon; rho = Spearman correlation coefficient; p = two-sided p-value; sign = direction of the association (positive/negative).