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Management of *Fusarium* diseases affecting conifers

Thomas R. Gordon [a,*], Cassandra L. Swett [b], Michael J. Wingfield [c]

[a] *Department of Plant Pathology, One Shields Avenue, University of California, Davis, CA 95616, USA*
[b] *Department of Plant Science and Landscape Architecture, University of Maryland, College Park, MD 20742, USA*
[c] *Forestry and Agricultural Biotechnology Institute (FABI), University of Pretoria, Hillcrest 0002, South Africa*

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ABSTRACT

Coniferous trees can suffer significant damage from seedling diseases caused by *Fusarium* spp. and from pitch canker, which is caused by *Fusarium circinatum* Nirenberg & O'Donnell. These fungi can be responsible for both pre- and post-emergence damping-off, and latent infections can result in failure of transplanted seedling to become established. *Fusarium oxysporum* Schlechtend.: Fr. has long been recognized as the most important cause of root and hypocotyl rot in seedling nurseries, but phylogenetic analysis suggests that the most virulent strains are more properly assigned to the recently described *Fusarium commune* Skovgaard, O'Donnell et Nirenberg. Management of *Fusarium* diseases in bare-root seedling nurseries has relied primarily on preplant soil fumigation. Because regulations will increasingly limit availability of the most efficacious fumigants, alternative management practices are being explored. This includes greater attention to sanitation, maintaining a robust microbial community that will inhibit root-infecting pathogens and avoiding practices that predispose trees to disease, such as excessive fertilization and poorly drained soils. *F. circinatum* Nirenberg & O'Donnell can be a problem in seedling nurseries but is also damaging to mature trees in plantations, seed orchards, landscape plantings, and native forests. A critical element of management is limiting spread of disease from existing infestations. To this end, branches and logs removed from infected trees should be disposed of locally. Seeds and seedlings can both carry the pathogen and so should not be moved from infested to non-infested areas. Quarantine restrictions should be maintained to prevent introduction of *F. circinatum* into countries where it is not yet established. Infections caused by *F. circinatum* are associated with wounds resulting from silvicultural practices, such as pruning and seed harvesting, weather related injuries and insect activity. In managed plantings, the risk of disease can be reduced by limiting pruning operations to cool, dry periods, which are less conducive to infection, and by judicious control of insects that can serve as wounding agents and vectors. Variation in susceptibility to pitch canker has been documented in a number of commercially important pine species, offering the prospect for greater utilization of genetic resistance for management of this disease in the future.

1. Introduction

A number of *Fusarium* species are recognized as causes of disease of coniferous trees, some of which have significant economic and/or ecological impacts. This includes fungi that cause damping-off and root rot in seedling nurseries, and poor establishment of out-planted seedlings (Jones et al., 2014). *Fusarium circinatum* Nirenberg & O'Donnell is among those species that are damaging to seedlings, but it is also a pathogen of mature trees, causing the disease known as pitch canker. Pitch canker affects planted pines in many parts of the world, and will be the focus of this review, along with root and hypocotyl rot caused by *Fusarium oxysporum* and *Fusarium commune*. Only brief mention will be made of *Fusarium* species that have been associated with seedling diseases but which have not yet been extensively studied. Also excluded is *Fusarium torreyae* T. Aoki, J. A. Smith, L. Mount, Geiser and O'Donnell, a recently described pathogen of *Torreya taxifolia* Arn. (Aoki et al., 2013). Although of potentially great ecological significance, the recent discovery of *F. torreyae* precludes development of a body of literature germane to management, which is the focus of this review.

2. Seedling diseases

2.1. Introduction

Seedling diseases caused by *Fusarium* spp. are problematic in

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* Corresponding author.
E-mail address: trgordon@ucdavis.edu (T.R. Gordon).

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bare-root nurseries and in container culture. Whereas bare-root nurseries rely on preplant fumigation to suppress soilborne pathogens, container operations can use pathogen-free growing media. Of course good sanitation practices are essential to avoid contamination of media and containers, but with regular monitoring, it should be possible to address problems that do arise relatively quickly. The impending loss of efficacious fumigants makes these advantages more compelling. However, owing to lower cost per seedling, bare-root nurseries still account for most of the conifer seedlings grown in North America. Likewise most of the literature on seedling diseases caused by *Fusarium* spp. deals with bare-root production systems, which consequently will be the primary focus of this review.

2.2. *Symptoms and etiology of seedling diseases*

Many coniferous trees are affected by *Fusarium* spp., including Douglas-fir (*Pseudotsuga menziesii* (Mirb.) Franco), larch (*Larix* spp.), true firs (*Abies* spp.), western white pine (*Pinus monticola* Dougl.), eastern white pine (*Pinus strobus* L.), ponderosa pine (*Pinus ponderosa* Dougl. ex Laws.), and sugar pine (*Pinus lambertiana* Dougl.) (Brownell and Schneider, 1983; Enebak et al., 1990; James et al., 1986; James, 2012). *F. oxysporum* is the species most commonly recovered from diseased conifer seedlings, and has been confirmed as a cause of damping-off (Tint, 1945), as well as hypocotyl- and root rot (Bloomberg, 1971). Damping-off can occur prior to or shortly after seedling emergence but infected seedlings can survive for several months or longer, and may or may not eventually develop symptoms. Root and hypocotyl rot affect older seedlings, with most damage to bare-root crops occurring during the first growing season. Container stock is affected throughout the growth cycle, with root symptoms often becoming evident when seedlings are stressed to initiate bud formation (Dumroese and James, 2005). Hypocotyl rot is characterized by a lesion on the stem beneath the cotyledons that can extend to the soil line or below (Brownell and Schneider, 1983). Top symptoms include stunting, yellowing and necrosis of needles and, in some cases, wilting. Similar symptoms result from root rot, but in this case the lesion develops on the tap root rather than the hypocotyl. Dead seedlings can turn black and decay quickly, and could easily escape detection in a visual survey (Dumroese and James, 2005). The symptoms described above can be caused by other pathogenic fungi, and are not by themselves sufficient to confirm the cause of disease. One indication that *F. oxysporum* is involved is the presence of sporulation in the form of sporodochia, orange to yellow in color, on the stems of infected plants (James, 2003). A definitive diagnosis requires identification of the pathogen based on morphological criteria (Leslie et al., 2006) or molecular markers (Stewart et al., 2006).

F. oxysporum is commonly found in nursery soils and can be recovered from roots of seedlings that show no symptoms of disease (Bloomberg, 1971). Inoculation tests show many of these isolates to be non-pathogenic, whereas others can cause disease to varying extents. A phylogenetic analysis revealed that isolates recovered from both diseased and healthy Douglas-fir seedlings, which were identified as *F. oxysporum* based on morphological criteria, were properly assigned to the recently described species, *F. commune* Skovgaard, O'Donnell et Nirenberg (Skovgaard et al., 2003; Stewart et al., 2006). *F. commune* is similar in appearance to *F. oxysporum* and genetic markers are required to reliably distinguish between these two species. Recent surveys have shown that *F. commune* is widespread in North American conifer nurseries (Kim et al., 2012), where it may be the principal cause of mortality in Douglas-fir seedlings. Studies conducted by Stewart et al. (2012) found some isolates of *F. oxysporum* to be damaging to Douglas-fir seedlings in inoculation tests, but all were less virulent than isolates of *F. commune*, and none were a cause of mortality. Thus, it is now unclear if *F. oxysporum* as currently circumscribed is an important pathogen of Douglas-fir. Further research on this topic will be required, along with studies to more fully characterize the host range of *F. commune* and, in particular, to determine the extent to which this species is a cause of disease in *Pinus* spp. and other conifers.

In addition to *F. oxysporum* and *F. commune*, many other species have been reported to cause seedling disease affecting one or more coniferous species in North America and elsewhere in the world, including *Fusarium proliferatum* (Matsushima) Nirenberg, *Fusarium solani* (Mart.) Sacc., *Fusarium acuminatum* Ellis & Everh., *Fusarium avenaceum* (Fr.) Sacc., *Fusarium sambucinum* Fckl., *Fusarium sporotrichioides* (Sherb.) Bilai, *Fusarium subglutinans* (Wollenweber & Reinking) Nelson, Toussoun & Marasas, and *Fusarium equiseti* (Corda) Sacc. (Huang and Kuhlman, 1990; James and Perez, 1999; James et al., 1997; James, 2012). *F. circinatum* poses a major problem in pine seedling nurseries in the U.S., South Africa and Chile, among other locations (Wingfield et al., 2008). In Algeria, *Fusarium redolens* Wr. (Lazreg and Belabid, 2013a), *Fusarium chlamydosporum* Wollenw. & Reinking (Lazreg and Belabidb, 2013b), and *F. equiseti* (Lazreg et al., 2014) were recently reported to cause damping-off of Aleppo pine (*Pinus halepensis* Mill.). Management studies described below have dealt specifically with disease caused by *F. oxysporum* but the findings and recommendations are likely also to be applicable to other pathogenic species of *Fusarium*.

2.3. *Management*

Management of *Fusarium* hypocotyl- and root rot requires maintaining soil inoculum densities below damaging levels. This has commonly been accomplished by pre-plant soil fumigation with a mixture of methyl bromide and chloropicrin. Following injection of the fumigant, treated soil is covered with a plastic tarp. This procedure is very effective in reducing populations of soilborne fungi, including *F. oxysporum*, as well as nematodes and weed seeds. However, federal regulations will ultimately prevent the use of methyl bromide, and consequently, alternative approaches to control of soilborne pests are being evaluated (Weiland et al., 2013).

Chloropicrin has good fungicidal activity and can be used as a fumigant without inclusion of methyl bromide (South et al., 1997). Based on a study conducted at a white pine (*P. strobus*) nursery in Wisconsin, Enebak et al. (1990) reported that fumigation with 100% chloropicrin at a rate of 196 kg per hectare reduced the soil population of *Fusarium* spp. as effectively as a 2:1 mix of methyl bromide: chloropicrin applied at the same rate. Inoculum densities were below 1 colony forming unit per gram in both cases. Similarly, studies conducted at three nurseries in the southern U.S. found no significant difference in efficacy between 100% chloropicrin and a 2:1 combination of methyl bromide and chloropicrin (Cram et al., 2007). Less consistent results are reported from the western U.S., where heavier soils require greater attention to proper soil preparation (Weiland et al., 2013). Notwithstanding challenges, reliance on chloropicrin as a fumigant in forest seedling nurseries is likely to increase in the near future, although longer term availability may ultimately be curtailed by enhanced regulatory restrictions (CDPR, 2011).

Another option for pre-plant treatment of soil is material that generates methyl isothiocyanate (MITC), which includes both liquid (metam sodium) and granular (dazomet) formulations. In both cases, water activation at temperatures above 10 °C is required, which limits application to summer and fall (Weiland et al., 2013). Relative to methyl bromide and chloropicrin, the efficacy of MITC-producing agents is more sensitive to soil texture, with variable

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results in denser soils. The amount of water applied is also critical, as it affects distribution and residence time of the active ingredient (Wang et al., 2006), which in turn influences the likelihood of killing fungal propagules (Nelson et al., 2014). As with other materials, the efficacy of MITC fumigations is enhanced by covering treated soil with a plastic tarp immediately after application, with maximal benefit obtained from material that is totally impermeable and hence maximizes retention of the fumigant in soil (Weiland et al., 2013).

Uncertainty about the continued availability of efficacious fumigants has motivated greater attention to other means by which disease suppression can be achieved. Fungicides with activity against *Fusarium* spp. are available and can help to control damping-off (Linderman et al., 2008) but have not been shown to reduce damage caused by hypocotyl rot or root rot. Consequently, more emphasis has been placed on other practices that 1) effect a reduction in the abundance of pathogenic *Fusarium* spp. in soil, 2) enhance the activity of microbes that inhibit plant pathogenic fungi, and 3) avoid predisposing seedlings to development of disease.

One way to reduce the inoculum density of root-infecting fungi is to include a vegetation-free period as a part of the cropping cycle. During this interval, the number of viable propagules of *F. oxysporum* would be expected to decline by attrition, although this may be offset to some degree by growth on residue from a previous crop. Hansen et al. (1990) reported that, in the absence of fumigation, the inoculum density for *Fusarium* spp. in aggregate was significantly lower in soil following fallow than in soil that had been cropped to oats or peas. At one location there was also a reduction in *Fusarium* hypocotyl rot where soil was fallowed. Rye and red clover cover crops were also found to increase mortality due to damping-off caused by *F. oxysporum* (Wall, 1984). These findings indicate that cover crops may be colonized by pathogenic strains of *Fusarium* and so increase the risk of disease, and hence that it may be better to fallow the land between seedling crops. In fact, Hildebrand et al. (2004) found that, at some nursery locations, disease caused by *Fusarium* was no more severe in plots that were fallowed prior to sowing and not fumigated than in plots fumigated with methyl bromide and chloropicrin. However, results vary by nursery, and fallowing alone cannot be expected to keep losses to root and hypocotyl rot at acceptable levels (James, 2001). Furthermore, fallowing engenders other problems, such as the need for some form of weed control and loss of soil due to wind erosion.

Drawing on experience from agricultural systems, benefit may be gained from cover crops containing glucosinolates, which are converted to isothiocyanates following incorporation of the crop. Broccoli residue, for example, has been shown to reduce populations of *Verticillium dahliae* (Njoroge et al., 2009). However, incorporating *Brassica juncea* after six weeks of growth in a forest nursery resulted in an increased population of *Fusarium* spp. and greater mortality in the subsequent crop of Douglas-fir seedlings (James et al., 2004). This result was taken to indicate that any direct or indirect toxic effect of decomposing residue on *F. oxysporum* was outweighed by the benefit of access to a source of nutrients. The same study also found the application of composted sewage sludge to increase soil populations of *Fusarium* spp.

Whereas fallowing or cover cropping aim to reduce inoculum levels of pathogenic fungi, other measures are intended to modify the soil environment in ways that will inhibit the propagules that are present. In principle, this might be achieved by enhancing the activity of soil microbes that can compete with and/or antagonize plant pathogenic fungi. Indications that co-occurring microbes can have suppressive effects on pathogens affecting conifer seedlings follow from the observation that although decline and death of seedlings induced by *F. oxysporum* is common in nurseries, disease caused by this fungus is not a problem in coniferous forest soils covered with needle litter. Smith (1967) noted whereas *F. oxysporum* was recovered from the roots of 90% of sugar pines when trees were lifted from nursery soil, this fungus was not detectable on roots of sugar pines four years after outplanting in the forest. Similarly, Dumroese et al. (1993) concluded that field performance of Douglas-fir seedlings was not negatively affected by root infection by *Fusarium* spp. at the time of transplanting. The lack of any significant impact of pre-existing infections suggests that forest soil conditions negatively affect *Fusarium* spp. Consistent with this view, *F. oxysporum* is rarely recovered from forest soils (Schisler and Linderman, 1984; Axelrood et al., 1998), wherein chlamydospores are observed to germinate, followed by lysis of the germ tube. There is evidence for both a chemical (Toussoun et al., 1969) and biological basis for this effect (Schisler and Linderman, 1984). A role for the microbiota derives from studies showing that treating soil with steam, propylene oxide or radiation greatly diminished the stimulatory effect on chlamydospore germination (Schisler and Linderman, 1984). These findings offer hope that manipulation of the microbial community in nursery soils might establish pathogen suppression comparable to what occurs in the forest.

The composition of the microbial community in soil can be modified through incorporation of organic amendments, which are commonly used in forest nurseries to improve the physical and chemical properties of soil. Most of what is known about the influence of organic amendments on diseases is based on studies of agricultural crops. This work has revealed quite variable effects, with amendments reducing disease impacts in some crop-pathogen systems and making disease worse in others, suggesting that suppression is often pathogen-specific. Based on an assessment of over 2000 studies, Bonanomi et al. (2010) concluded that both microbial biomass and measures of microbial activity (e.g., quantifying hydrolysis of fluorescein diacetate) tend to be positively correlated with suppressiveness. This may reflect direct antagonism of the pathogen as well as competition for resources. In forest nurseries, Hildebrand et al. (2004) reported a benefit of amending soil with aged sawdust. This effect was attributed in part to the complex nature of the nutrients in sawdust (mostly lignin and cellulose), which would be more readily utilized by microbes other than plant pathogenic fungi. In line with this view would be the expectation that amendments with high C:N ratios, such as composted pine bark, would provide more suppression than materials with low C:N ratios.

Efforts to impose biological barriers to pathogen activity have included the use of individual microbial taxa to protect seedlings from disease. Linderman et al. (2008) tested a number of commercial products under greenhouse conditions, including formulations of *Bacillus* spp., *Streptomyces* spp., *Trichoderma harzianum* Rifai and *Gliocladium virens* Miller, Giddens and Foster. None of the tested products provided effective control of damping-off of Douglas-fir seedlings caused by *F. oxysporum*. More promising results were obtained using a strain of *T. harzianum* obtained from a native soil in Idaho that was incorporated into soilless media (Mousseaux et al., 1998). In this case, damping-off of Douglas-fir seedlings caused by *F. oxysporum* was significantly reduced when roots encountered the pathogen after growing through a layer of medium in which *T. harzianum* was present but the pathogen was not. In contrast, mortality was not lowered when *T. harzianum* and *F. oxysporum* were both uniformly distributed throughout the medium. The value of prior colonization was also evident in a study conducted by Dumroese et al. (2012), in which Douglas-fir seedlings grown in a medium infested with a non-pathogenic strain of *F. oxysporum* prior to transplantation into a medium containing *F. commune* (= *F. oxysporum sensu lato*) manifested no symptoms of

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disease. These findings may indicate that some measure of biological control can be obtained in container culture of seedlings. On the other hand, field application of biological agents has generally not proven to be efficacious (Hildebrand et al., 2004; James et al., 2004).

The severity of *Fusarium* hypocotyl- and root rot will be influenced by the conditions under which seedlings are grown. Dumroese and James (2005) noted the importance of avoiding stress induced by water-saturated soils, which can render seedlings more prone to disease. Accordingly, nursery managers should strive to maintain well-drained soils through deep tillage to disrupt hardpans, adding organic matter to improve aeration and water penetration, avoiding compaction that can result from operating equipment on wet soils, and monitoring soil moisture so irrigation is supplied only when needed. Seedlings should not be over-fertilized because excess nitrogen can promote succulence and increase susceptibility to disease caused by *F. oxysporum* (Tint, 1945; James, 1997). Nitrogen should be supplied only in amounts that are balanced with respect to other mineral nutrients, particularly potassium and phosphorus. Timing is also important, and evidence suggests that post-emergence rather than pre-plant application can reduce disease risk (Hildebrand et al., 2004). The form of nitrogen may have an effect, with ammonium being more conducive to disease than nitrate, but this may be due at least in part to an effect on soil pH (Woltz and Jones, 1981). Disease tends to be more severe under relatively acidic conditions because micronutrients are more available to *F. oxysporum* and because bacteria are less active. Consequently, maintaining soil pH near neutrality may contribute to management of root- and hypocotyl rot.

As is the case with all soilborne diseases, sanitation is essential for effective management of seedling diseases in forest nurseries. *F. oxysporum* can quickly become re-established in fumigated soils, given the reduced numbers of competing microbes (Marois et al., 1983). Therefore it is important to avoid introducing the pathogen into production areas by moving soil from infested sites. Another vehicle for introduction of inoculum is seed, which can carry propagules of *F. oxysporum* and other species of *Fusarium* (Graham and Linderman, 1983; James, 1987; Mason and Van Arsdell, 1978). The risk of introducing *F. oxysporum* on seed can be reduced by harvesting cones directly from trees rather than fallen cones, and avoiding seed from squirrel caches, which may carry higher levels of inoculum of *F. oxysporum* (Dumroese and James, 2005). Imbibing seeds in running rather than still water can help to reduce the amount of inoculum that is retained on seed (James and Genz, 1981). Various treatment protocols utilizing ethanol, sodium hypochlorite, or hydrogen peroxide have been shown to significantly reduce seedborne inoculum, while maintaining a high rate of germination (Dumroese et al., 1988). Other appropriate sanitation measures include frequent cleaning of all seed handling equipment and periodic removal of symptomatic seedlings, which can add inoculum to the soil. Culled seedlings should be burned or otherwise disposed of in a manner that eliminates inoculum or at least ensures that it does contaminate production areas.

At present, conifer seedling production in North America remains dependent on pre-plant fumigation and it seems likely that this practice will continue as long as efficacious materials are available (Weiland et al., 2013). No alternative measures can provide comparable levels of disease control. Ideally, where fumigation is to be discontinued, a final treatment of the soil with the best available material could be employed to reduce inoculum to the lowest possible levels. Thereafter, an integrated approach that emphasizes good sanitation, coupled with cultural practices that maintain a robust microbial community in soil and promote vigorous growth of seedlings could minimize opportunities for pathogens to become established and increase to levels that result in reduced yield and quality of seedlings. Prospects for effective management would certainly be enhanced by development of lines that are less susceptible to *Fusarium* root- and hypocotyl rot. To date, however, it appears that no such efforts have been initiated (Weiland et al., 2013).

If economic losses to soilborne diseases cannot be kept to an acceptable minimum, nurseries may elect to produce seedlings in containers using soilless media. This is already a common practice in the Southern Hemisphere, where many large nurseries grow seedlings in *Fusarium*-free media such as pine bark, coco peat, vermiculite or perlite. Still, sanitation remains critically important, with seed, irrigation water, and contaminated containers being the primary means by which pathogens may be introduced (Morris et al., 2014).

3. Pitch canker

3.1. Distribution and host range

Pitch canker, caused by *F. circinatum*, was first recognized as a disease in the state of North Carolina in the U.S. in 1945 (Hepting and Roth, 1946). The pathogen was isolated from cankers that were characterized by extensive production of resin, which inspired the name “pitch canker” for the disease. Pitch canker subsequently became more widespread in the southeastern U.S. (SE U.S.), where it continues to cause problems for production of pines in plantations, seed orchards, and seedling nurseries (Dwinell et al., 1985; Storer et al., 1997). The disease was discovered in California in 1986 (McCain et al., 1987), followed by confirmed reports from Japan (Kobayashi and Muramoto, 1989), South Africa (Viljoen and Wingfield, 1994), Mexico (Guerra-Santos, 1999), Spain (Landeras et al., 2005), South Korea (Lee et al., 2000), Chile (Wingfield et al., 2002), Italy (Carlucci et al., 2007), Portugal (Braganca et al., 2009), Colombia (Steenkamp et al., 2012), and Brazil (Pfenning, 2014).

The host range of *F. circinatum* extends to more than 60 species of *Pinus* (Hepting and Roth, 1953; Muramoto et al., 1993; Guerra-Santos, 1999; Hodge and Dvorak, 2000; Lee et al., 2000; Gordon et al., 2001; Enebak and Carey, 2003; Enebak and Stanosz, 2003) and also includes Douglas-fir, the only conifer outside the pine genus known to be susceptible (Gordon et al., 2006a). Under experimental conditions, *F. circinatum* is capable of colonizing maize (*Zea mays* L.) asymptomatically (Swett and Gordon, 2015), and natural infections of grass hosts have been confirmed in the U.S. (Swett and Gordon, 2012) and in South Africa (Swett et al., 2014). No symptoms have been observed on infected grasses, suggesting the relationship is commensal. The significance of an association with grasses has yet to be established but could influence disease development, as described below.

3.2. Symptoms and identification of the pathogen

A typical symptom of pitch canker in mature trees is dead branch tips. Branches die as a result of a girdling lesion at the site of infection, which is often a cone node or non-cone branch whorl, but infections may also occur in inter-nodal regions. The pathogen appears to grow more rapidly in succulent, current year growth than in older, more lignified tissue. The earliest symptom is wilting of needles distal to the infection site, which can be recognized by an accumulation of resin on the branch surface. Wilted needles lose their lustrous green color and eventually become chlorotic, before turning red and finally brown; abscission tends to occur quickly and uniformly, leaving naked tips. The progression of symptoms varies with the season and age of the infected branch. Young, succulent

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branches may droop, giving the appearance of a Shepherd's crook, whereas older, lignified branches remain stiff as the needles fade and die. Individual infections do not progress very far axially along the infected stem, probably because of the accumulation of host-produced resin. Although resin does not prevent growth of *F. circinatum* (Slinski et al., 2015), it will restrict the flow of water, leading to desiccation of the infected tissue and the pathogen.

Pitch canker intensifies through repeated infections. In addition to tip dieback, larger diameter branches may eventually die owing to the coalescence of multiple lesions. This can occur as well on the main stem, leading to top-kill and, in some cases, death of the entire tree. Infections on the trunk of the tree often produce copious amounts of resin, which can coat large areas of the bark. Resin streaming on the main stem typically occurs on trees that have already sustained extensive canopy dieback (Gordon et al., 2001) but can be an early symptom in some cases. The pattern of symptom development may be a reflection of the means by which infection courts are established. The death of branch tips would commonly be associated with infections mediated by twig beetles (*Pityophthorus* spp.) (Sakamoto et al., 2007; Storer et al., 2004) whereas the early appearance of cankers on the main stem may indicate infections were initiated by engraver beetles (*Ips* spp.) (Fox et al., 1991).

In addition to affecting vegetative tissues, the pitch canker fungus can infect and kill both female strobili and mature cones (Dwinell et al., 1985; Barrows-Broaddus, 1990). Infected cones can be misshapen and stunted, and often abort before reaching maturity (Correll et al., 1991). *F. circinatum* can also infect cones without doing visible damage, and seeds from such cones can carry the pathogen internally (Storer et al., 1998a). Infected seeds may or may not show visible evidence of deterioration.

Sowing infected seed can result in pre or post-emergence mortality, but it is also possible for infected seedlings to survive and remain symptomless for an extended period of time. Symptoms on seedlings killed by *F. circinatum* at or shortly after emergence are not particularly distinctive and laboratory examination is required to confirm a diagnosis. Culturing on a selective medium that contains pentachloronitrobenzene allows for reliable recovery of *F. circinatum* from infected tissue (e.g., Aegerter and Gordon, 2006). Most often the pathogen is found to be colonizing the root collar. Depending on the soil in which seedlings were growing and the condition of the sample, the inclusion of additional antibiotics (Correll et al., 1991) to suppress bacteria may be advisable.

A diagnostic feature of branches killed by pitch canker is an accumulation of resin at the junction of living and symptomatic tissue, which corresponds to the site of infection. Other causes of tip dieback, such as western gall rust, caused by *Endocronartium harknessii* (J. P. Moore) Y. Hiratsuka (Old et al., 1986) and shoot blight caused by *Diplodia pinea* (Desm.) Kickx. (Swart and Wingfield, 1991), typically lack this characteristic symptom. Paring away the bark at the infection site reveals a discolored, resin-soaked lesion, from which *F. circinatum* can be cultured. A selective medium can be used for this purpose, but water agar is often sufficient. In either case, the pathogen will grow from lesion margins and produce diagnostic microconidiophores. Where tissue was recently infected, growth of the pathogen may be apparent after only 24 h of incubation. The older the infection, the longer it may take for the pathogen to emerge.

The facility with which *F. circinatum* can be identified based on morphological criteria depends on the prevalence of other *Fusarium* spp. The formation of microconidia in false heads borne on polyphialides is sufficient to distinguish *F. circinatum* from *F. proliferatum* (Gordon et al., 1996), which can also be associated with pine seedlings and may be a cause of mortality (James et al., 1997). Morphological differences between these two species are readily visualized on carnation leaf agar amended with 1.5% KCl (Nelson et al., 1983). Some species, however, are more difficult to separate from *F. circinatum* based solely on morphological criteria, and molecular diagnostic methods can help to achieve a definitive diagnosis (Steenkamp et al., 1999; Schweigkofler et al., 2004; Ramsfield et al., 2008; Ioos et al., 2009).

3.3. The infection process

Canopy dieback caused by pitch canker is associated with wounding that creates infection courts. In seed orchards, wounds caused by mechanical shakers and by tearing cones away from branches are prone to infection by *F. circinatum* (Dwinell et al., 1985). Infection courts can also be created by pine-associated insects. In the SE U.S., the deodar weevil (*Pissodes nemorensis* Germar) (Blakeslee and Foltz, 1981) and pine tip moths (*Rhyacionia* spp.) (Runion et al., 1993) are potentially important wounding agents and/or vectors of pitch canker. In California, several species of *Pityophthorus* and *Ips* have been shown to vector pitch canker (Gordon et al., 2001), as have *Conophthorus radiatae* Hopkins (cone beetle) and *Ernobius punctulatus* Fall. (dry twig and cone beetle) (Hoover et al., 1995, 1996). Also in California, *Aphrophora canadensis* Walley (spittle bug) is an effective wounding agent (Storer et al., 1998b) and numerous other insects carry the pathogen and may serve as vectors. In South Africa, *P. nemorensis* was found to be infesting trees where the first outbreak of pitch canker on adult pines was documented in that country (Coutinho et al., 2007), and this insect has been shown to facilitate infection in greenhouse studies (Gebeyehu and Wingfield, 2003). In Spain, *Ips sexdentatus* Boerner and *Pityophthorus pubescens* Marsham among several other pine-associated insect species are reported to carry spores of *F. circinatum* (Romón et al., 2007).

The likelihood of infection may be influenced by the nature, size and age of a wound, and by ambient conditions during the period of wound susceptibility. Pruning wounds on *Pinus radiata* D. Don (Monterey pine) became less susceptible over time but a 30% infection rate was recorded even 28 days after cuts were made (Sakamoto and Gordon, 2006). In *Pinus taeda* L. (loblolly pine), pinhole wounds (made with a dissecting needle to a depth of 3–5 cm) were readily infected, whereas wounds made by removing needle fascicles or lateral branches became infected at a much lower rate (Kuhlman, 1987). In *P. radiata*, wound size had a significant effect on infection frequency, with 1.6 mm diameter wounds sustaining a higher infection rate than wounds 0.5 mm in diameter (Sakamoto and Gordon, 2006). In *P. taeda* and *P. elliotii* Engelm. (slash pine), pinhole wounds remained susceptible for 21 days, although infections occurred at a much reduced rate (20%) when compared to fresh wounds (70%) (Kuhlman, 1987). In *P. radiata*, mean infection rates for wounds 0.3 mm in diameter and 1 mm deep decreased from nearly 82% on day zero to 12.5% two days later (Inman et al., 2008). The interval of wound susceptibility defines the period within which spores of *F. circinatum* must germinate, and subsequent growth must be sufficient to establish an infection. Whether or not this occurs will be strongly influenced by ambient temperature and moisture availability. Spore germination and growth both proceed very slowly at 10 °C and more rapidly as temperature increases up to 25 °C (Inman et al., 2008). For this reason, infection rates tend to be lower in winter than during warmer periods (Inman et al., 2008). However, higher temperatures will favor infections only if wounds are deep enough to reach moisture within the plant, or if ambient humidity is high and/or free moisture is present. Thus, infections mediated by *Pityophthorus* spp., which create only very shallow wounds on healthy branches, occur at a higher frequency when relative humidity is at or close to 100% (Sakamoto et al., 2007). On the other hand, where inoculum is

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applied to deeper wounds created mechanically, the effect of ambient humidity on infection frequency appears to be diminished (Sakamoto and Gordon, 2006). The temperature and moisture requirements for infection are consistent with the widespread occurrence of pitch canker in the SE U.S., where rainfall during warm periods is common. Conversely, in California, where most areas receive rainfall only during the coolest months of the year, pitch canker is restricted to the coast, where moderate temperatures coincide with high humidity and/or condensation provided by moist marine air.

Based on the assumption that environmental requirements for infection impose limits on the distribution of pitch canker, the model CLIMEX was used to characterize the risk of disease on a global scale (Ganley et al., 2009). Predictions of the model were consistent with the distribution of pitch canker in North America, where the disease has been established long enough to have approached ecological limits on its geographic range. The model further predicts that pitch canker constitutes a limited risk to continental Europe, although pockets of optimal climate were found in Spain, Portugal, France, Italy and Georgia. Climate was found to be suboptimal in Chile, where pitch canker is a problem in nurseries but not as yet in plantations. In contrast, regions within Australia and New Zealand, where the highly susceptible Monterey pine is widely planted, appear to have a climate that would be optimal for the disease, should the pathogen become established. CLIMEX has also been used to predict how climate change may influence the risk of pitch canker in Australasia in the future (Ganley et al., 2011).

An important caveat to conclusions drawn from models based on climate limitations is that wounding agents may negate environmental limitations on infection. Insects that colonize healthy tissue, such as the cone beetle (*C. radiatae*), can allow the pathogen to access moisture within a tree, effectively shielding the fungus from limiting effects of dry ambient conditions. Furthermore, weather will have little or no effect on the activity of *F. circinatum* as a soilborne pathogen. Thus, countries, or regions within countries, with a climate deemed unsuitable for pitch canker may nevertheless sustain serious damage from seedling mortality in nurseries and a higher rate of transplant failure as a consequence of latent infections.

3.4. Management of pitch canker

F. circinatum can infect and kill susceptible pines at any stage of development, and affects commercial production of trees in seedling nurseries, plantations and seed orchards. Pitch canker can also be damaging to landscape trees and to trees in native forests. Accordingly, management considerations will be presented separately for planted and native stands of susceptible species.

3.4.1. Seedling nurseries

F. circinatum is not a common resident in soil and so is not likely to be a problem in seedling nurseries unless introduced with contaminated soil or seed. If the pathogen is not already established in soil nearby, seed is the most likely vehicle for introduction. *F. circinatum* has been confirmed to occur both on and within seeds of several pine species (Carey et al., 2005; Dwinell et al., 1985; Storer et al., 1998a). Superficial contamination of seed is common in areas where pitch canker occurs, regardless of the disease status of the tree from which cones are obtained. Such infestations are presumably due to deposition of airborne microconidia and macroconidia, both of which *F. circinatum* can produce on infected host tissue. Spores might germinate on seed, but whatever subsequent growth occurs appears not to extend beyond the seed coat because topical treatments with various anti-microbial materials will eliminate the pathogen. For example, seeds collected from healthy *P. radiata* in stands affected by pitch canker commonly carry the pathogen, but the incidence of infestation can be reduced to zero by immersion of seeds in an aqueous solution of 1.0% sodium hypochlorite (Storer et al., 1998a).

Seed from cones on infected branches may sustain infestations that survive exposure to surface acting materials such as sodium hypochlorite (Storer et al., 1998a). Internally infested seeds typically suffer high mortality rates from both pre- and post-emergence damping-off. However, some seed treatments will significantly reduce the rate of mortality, which indicates that internally infested seed is capable of producing a healthy seedling if growth of the pathogen can be suppressed. Both internal and external infestations of *P. radiata* seed were eliminated by treatment either with benomyl or a combination of benzimidazole, carboxin and thiram, which increased seedling emergence to 53 and 57%, respectively, compared to 43% for untreated seed (Gordon, unpublished data). No seedlings emerging from treated seed were infected with *F. circinatum*. Runion and Bruck (1988) found that thiabendazole suspended in 10% dimethyl sulfoxide significantly reduced but did not eliminate infestations of *F. circinatum* in seed of *Pinus palustris* Mill. An alternative to chemical seed treatments is to soak seeds in hot water prior to sowing. Agustí-Brisach et al. (2012) reported that brief exposure to water at temperatures above 51 and below 53 °C eradicated *F. circinatum* from infested seed in most cases, and all seedlings emerging from treated seed were free of the pathogen.

Although seed treatments can reduce the risk of introducing *F. circinatum*, it is nevertheless advisable to avoid moving seed collected in infested areas to regions where pitch canker does not occur. Seed appears to have been an important vehicle for movement of *F. circinatum* over long distances. For example, genetic relationships between populations suggest that Mexico is a likely source of the pitch canker infestation now found in South Africa (Wikler and Gordon, 2000), and the introduction most likely occurred through importation of infested seed (Britz et al., 2001). A restriction on seed importation should be applied to pines regardless of their susceptibility to pitch canker because resistance to the disease would not preclude deposition of airborne spores on seed. If pine seed is to be imported into non-infested countries, even seed originating from areas presumed to be free of pitch canker should be subjected to appropriate fungicidal treatments. Furthermore, one should never assume that any seed treatment will be completely effective and consequently imported seed should be sown and maintained under quarantine conditions, where any occurrence of seedling disease can be investigated to determine if *F. circinatum* was the cause.

The consequences of sowing seed carrying *F. circinatum* include not only a loss of seedlings due to damping-off but also the possibility that soil will become contaminated with the pathogen (Dwinell and Barrows-Broadus, 1978; Gordon et al., 2001). Although *F. circinatum* does not produce chlamydospores or other structures adapted to survival in soil, it has been shown to persist for as long as one year under controlled conditions (Gordon, 2011).

Where the seedling phase of pitch canker already occurs, management must emphasize sanitation to eliminate sources of inoculum. In South African nurseries, *F. circinatum* has been found to contaminate planting containers, allowing for infestation of sterile media and infection of seedlings. Steam treatment of containers can substantially reduce this risk (Morris et al., 2014). *F. circinatum* can also be found in irrigation water, but was shown to be eliminated through the addition of hydrogen peroxide (Van Wyk et al., 2012). Once seedlings become infected, the pathogen can spread throughout the nursery via wind borne spores (Fourie et al., 2014). Thus, every effort must be made to prevent infections from

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becoming established.

Although it remains to be confirmed, it is possible that infected grasses serve as cryptic reservoirs of inoculum in seedling nurseries. Several grass species have been shown to sustain natural infections both in South Africa (Swett et al., 2014) and in California (Swett and Gordon, 2012), and all isolates recovered from grasses were confirmed to be pathogenic to pines. Under experimental conditions, *F. circinatum* can infect roots and grow into the shoot of corn (*Z. mays*), wherein it colonizes developing seeds (Swett and Gordon, 2015). It remains unclear if corn and other grasses can serve as reproductive hosts for *F. circinatum* but preliminary studies show that the fungus will sporulate on senescent leaves removed from inoculated plants (Swett et al., 2013). Further research is needed to determine if sporulation on infected grasses occurs in nature.

Seedlings that sustain infections may remain symptomless for up to two years (Swett, 2013). Such latent infections can eventually become active and cause disease. This has become a significant problem for *Pinus patula* Schiede: Schltdl. & Cham., which is widely grown in South Africa. Plants that leave the nursery with latent infections often fail to become established in plantations, and losses to this problem are so great as to threaten the continued use of *P. patula* in commercial forestry in South Africa (Mitchell et al., 2012a; Jones et al., 2014).

To the extent that infections remain cryptic, seedlings can serve as a vehicle for dissemination of the pathogen. In California, for example, it is likely that pitch canker first became established in nurseries, from which cryptically infected seedlings were moved to “choose and cut” Christmas tree farms. Some infected trees eventually developed symptoms of pitch canker and died, and soil at these sites likely became contaminated, which may have caused infections in subsequent plantings (Gordon et al., 2001). Also, presymptomatic trees that were sold allowed infected material to be further distributed. Depending on how such trees were ultimately disposed of, they may have served as breeding sites for bark beetles that could acquire the pathogen and introduce it into nearby landscape pines (Gordon et al., 2001).

The potential for seedlings to sustain latent infections poses a challenge for management, because visual inspections are not sufficient to identify trees that may be carrying *F. circinatum*. Furthermore, greenhouse studies have shown that extensive colonization of the root system by *F. circinatum* may not result in visible damage. Shoot symptoms become apparent only after the fungus grows into the root collar and girdles the stem. Thereafter, *F. circinatum* grows more extensively in roots and can be isolated from necrotic tissue. Thus it appears that rotting of roots is a consequence of seedling death and not the cause (Swett and Gordon, 2013). If this is typical of how disease develops in seedling nurseries, inspection of roots may not be a reliable means of identifying cryptic infections.

Several measures can reduce the risk of shipping infected seedlings. Most important is to avoid introducing the pathogen to a nursery by using only seed that is certified to be pathogen-free. This can usually be accomplished by sourcing seed from areas where pitch canker does not occur. If necessary, seed can be tested for the presence of the pathogen either by cultural methods or by detection of a conserved DNA sequence using quantitative PCR (Ioos et al., 2009; Dreaden et al., 2012). Because the pathogen does not survive well in soil, periodic intervals without a susceptible pine crop may help to maintain inoculum densities below damaging levels. Although soil fumigation can be effective in killing pathogen propagules in soil, suppression of the resident microbiota may render fumigated soil more conducive to establishment of subsequent pathogen introductions (Marois et al., 1983). Thus, management practices that maintain a robust microbiota may better serve to reduce the risk of establishment of the pitch canker pathogen. On the other hand, where a localized infestation occurs, eradicative measures may be appropriate. In these situations, infected trees should be removed, along with as much of the root system as possible. All affected plant material should be burned or placed in sealed containers for disposal off-site. In the course of this activity, care should be taken not to expand the infested area by movement of pathogen-contaminated soil. The affected site should be treated with a material that will kill the pathogen in soil. Materials that generate MITC such as Basamid or Vapam can be effective for this purpose, and may be preferable to more potent fumigants because MITC has a less suppressive effect on the bacterial component of the microbiota.

Early detection of an infestation can help to minimize the damage. To this end, it is advisable to monitor the health of a seedling crop for the occurrence of dead or symptomatic plants. Symptoms on emerging seedlings are unlikely to be diagnostic, so laboratory tests should be undertaken to determine the cause of death. This can be accomplished by cultural methods or the use of procedures based on diagnostic DNA sequences (Wingfield et al., 2008). Recovery of *F. circinatum* from any seedling indicates that some symptomless seedlings may also be infected. Thus, further sampling should be undertaken to determine the extent of the infestation. If modest in scope, affected area(s) can be treated as described above but if more extensive, destruction of the crop may be justified. If any seedlings are to be shipped from a site where pitch canker has been confirmed to occur, a representative sample of seedlings should be tested for the presence of latent infections. The size of the sample should be scaled according to the detection threshold deemed to provide the desired level of safety. The potential for the pitch canker pathogen to infest seed and become established in seedling nurseries should be a principal focus of attention for regulators concerned with exclusion of the pathogen from non-infested areas. For this purpose, restrictions on seed imports should be considered along with appropriate quarantine and inspection procedures to ensure rapid detection of any incursions.

3.4.2. *Plantations, seed orchards and other managed plantings*

A key element in management of pitch canker in plantations and seed orchards is minimizing opportunities for infection. To this end suppressing the activity of insects that can serve as wounding agents can be beneficial. Runion et al. (1993) reported that soil applications of carbofuran to one-year old *P. taeda* significantly reduced damage by pine tip moth (*Rhyacionia* spp.) and the incidence of infection by *F. circinatum*. Wounds created by pruning are potential infection sites (Sakamoto and Gordon, 2006), and Bezos et al. (2012) found a significant relationship between pruning and the number of cankers per tree. The risk of infection may be reduced if pruning and other procedures that wound trees are conducted when temperatures are not favorable for insect activity and/or for infection by *F. circinatum*.

Whereas pruning creates wounds that can become infected, judicious removal of infected branches might contribute to disease management. Where canopy dieback is limited to small-diameter branches, it should be possible to rid a tree of disease by pruning. Of course pre-symptomatic infections will be missed and new infections can occur. Consequently, multiple rounds of pruning might be required to eliminate all infections and maintain a tree in a symptom-free condition. To assess the potential benefit of eradicative pruning, all symptomatic branches were removed from a stand of 51 trees, of which only four had symptoms of pitch canker (Gordon et al., 2001). In five successive rounds of pruning over a period of 43 months, 1185 branches were cut from symptomatic trees. At the end of this period, 48 of the 51 trees had symptoms of

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pitch canker. Similar results were obtained at three other locations, showing that eradicative pruning was not effective in eliminating the disease or even in reducing its incidence or severity. It is possible that disease would have progressed more rapidly in the absence of pruning, but it is doubtful the benefit gained would justify operational costs in most situations. A valuable landscape tree might be an exception. If pruning restores the esthetic value of a tree, costs might be repaid if the tree recovers and the expense of removal and replacement is thereby avoided.

Although fungicides with activity against *F. circinatum* are available, no cost-effective strategy for the use of anti-fungal materials to manage pitch canker has yet been developed. Applications of the fungicide thiabendazole were shown to reduce the incidence of pitch canker, but the magnitude of the benefit was judged insufficient to justify the cost of application (Runion et al., 1993). Comprehensive control of pitch canker using fungicides would be difficult to achieve because nearly all aerial surfaces would require protection for an extended period of time, and the cost of frequent fungicide applications would likely be prohibitive. Various materials can induce resistance to pitch canker, and chitosan has been shown to significantly reduce lesions lengths resulting from inoculations of *P. patula* with *F. circinatum* under controlled conditions (Fitza et al., 2013). This approach may have utility in managed plantings, provided the diversion of resources to defense does not engender a significant reduction in growth.

As noted above, *F. circinatum* appears to grow more aggressively on succulent than on lignified tissue, which may help to explain the observation that trees managed for rapid growth are more susceptible to pitch canker (Fisher et al., 1981; Fraedrich and Witcher, 1982). Accordingly, the benefits of fertilization to promote productivity must be balanced against the countervailing effect of enhanced susceptibility to disease. In addition to applied fertilizers, susceptibility to pitch canker may be influenced by proximity to volatile sources of nitrogen, such as ventilated poultry farms (Lopez-Zamora et al., 2007). Drought stress may also render trees more susceptible to pitch canker (Dwinell and Phelps, 1977). A protracted drought in California from 1987 to 1991 is believed to have contributed to mortality in *P. radiata* stands on soils with poor water holding capacity (Owen and Adams, 2001). Accordingly, matching stocking densities to the available water supply should reduce the impact of pitch canker. Stress may help to explain the observation that pitch canker is more severe in plantings started with seed from non-local provenances. Thus, genotypes that are not well-adapted to a planting site may be more prone to damage from the disease (Dwinell et al., 1985).

Genetic resistance offers an attractive approach to disease management because it can be highly effective, requires no inputs during the production cycle and engenders no adverse environmental impacts. Pine species show a wide range of variation in susceptibility to pitch canker, and greenhouse tests of young trees can provide results that are predictive of field performance. For example, lesion lengths resulting from branch inoculations on four pine species maintained in a greenhouse were strongly correlated with the incidence of pitch canker on the same four species in a landscape, where they were exposed to the pathogen under natural conditions (Gordon et al., 1998a). Among species grown in plantations in the SE U.S., inoculations of one year-old seedlings revealed *P. taeda* to be the most resistant, with shortleaf pine (*Pinus echinata* Mill.) and Virginia pine (*Pinus virginiana* Mill.) being the most susceptible, and *Pinus elliottii* ranked as intermediate (Dwinell, 1978). Even for susceptible species, such as *P. elliottii* and *P. radiata*, intraspecific variation in susceptibility has been demonstrated (Gordon et al., 1998b; Matheson et al., 2006; Rockwood et al., 1988; Roux et al., 2007; Storer et al., 1999), and can be exploited to develop planting stock with higher levels of resistance than is currently available. In *P. radiata*, families that manifested rapid growth were, in some cases, also among the most resistant to pitch canker (Matheson et al., 2006). Consequently, it may be possible to develop families and genotypes that are less prone to damage caused by pitch canker without compromising commercially important characteristics. Likewise, there appears to be considerable opportunity to select resistant families of *P. patula*, which is widely grown in South Africa (Mitchell et al., 2012b). Other species, such as *P. tecunumanii* Eguiluz & J. P. Perry (low elevation provenance) are also being tested for resistance to infection by the pitch canker fungus (Mitchell et al., 2012c) and for possible future deployment.

An alternative to genetic improvement through family selection within species is to develop more resistant germplasm through hybridization. This option has been pioneered by CAMCORE (http://www.camcore.org/overview/history.php) at North Carolina State University, where various species combinations are being tested. In countries such as South Africa and Colombia, where *P. patula* is a favored tree, hybrids between *P. patula* and *P. tecunumanii* (low elevation provenance) have shown promise (Kanzler et al., 2014). To the extent that plantation forestry can make greater use of resistant trees, the impact of pitch canker can be reduced accordingly.

It is possible that deploying resistant genotypes will select for novel pathotypes that compromise the efficacy of genetic resistance in the future. In *P. radiata*, resistance to pitch canker appears to be a quantitatively inherited trait (Matheson et al., 2006), which should reduce the likelihood that a change at a single genetic locus (through mutation or selection of a rare variant allele) in *F. circinatum* would be sufficient to negate genetic resistance. However, incremental changes in the pathogen population could occur over the course of several generations (Slinski, 2012) if the sexual cycle is operative in nature. Although out-crossing is readily accomplished in a controlled environment (Britz et al., 1998; Wikler et al., 2000), population studies have not revealed any indications that sexual reproduction occurs in nature (Gordon et al., 1996, 2006b; Iturritxa et al., 2011; Berbegal et al., 2013; Steenkamp et al., 2014). Of course, changes in local populations of *F. circinatum* could also result from the introduction of exotic strains that differ in virulence. For this reason, movement of infested material should be avoided, even into areas where pitch canker is already present.

3.4.3. Native forests

Several lines of evidence suggest the pitch canker pathosystem may have originated in Mexico (Gordon et al., 2001), where the disease affects a number of pine species in native forests (Guerra-Santos, 1999). That pitch canker appears to cause relatively little damage to native pines suggests the pathogen may be of relatively long residence in Mexico, consistent with an origin of the pathosystem in that part of the world. Further support for this hypothesis is based on interfertility of *F. circinatum* with *Fusarium temperatum*, a commensal associate of a grass species (*Z. mays* ssp. *mexicana* (Schrader) Iltis) that co-occurs with pines in Mexico and Central America (Desjardins et al., 2000; Friel et al., 2007). Other close relatives of *F. circinatum* are also commonly associated with grasses. Thus, the ability of *F. circinatum* to colonize corn (Swett and Gordon, 2015) and other members of the grass family (Swett and Gordon, 2012) may be seen as retention of ancestral characteristics, following a host jump from grasses to pines.

If this hypothetical origin of pitch canker is correct, *F. circinatum* must have been moved from Mexico to the SE U.S. (most likely on seed), where the disease was first described (Hepting and Roth, 1946). Thereafter, pitch canker developed into an increasingly serious and widespread disease of managed commercial plantings.

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The exception was in California, where pitch canker became established in native forests of *P. radiata*, *P. muricata* D. Don (bishop pine) and *P. attenuata* Lemmon (knobcone pine) (Gordon et al., 2001). In 1996, monitoring plots were established to characterize the dynamics of disease development in *P. radiata* in California. Data gathered over a three year period showed that *P. radiata* in managed stands (landscape trees in urban areas or in proximity to golf courses) had more severe disease than trees in native stands, independent of geographical location (Wikler et al., 2003). All the managed stands were fragmented, whereas trees in wild-land plots were mostly within relatively large, contiguous stands. Thus, many more trees in managed stands occupied edges, which may render them more prone to stress and may also enhance their exposure to inoculum. Disease was more severe in plots close to the coast than in plots located farther inland (Wikler et al., 2003). This difference remained apparent in 2013 (Gordon, unpublished data), and is likely due to greater frequency and duration of fog closer to the ocean. Farther inland, moisture is less available when temperatures are within a range that will support a high infection frequency. This concept is supported by studies under controlled conditions that document a significant effect of ambient humidity on the frequency of infections mediated by twig beetles (Sakamoto et al., 2007). Overall, the impact of pitch canker on native *P. radiata* forests has moderated in recent years and the rate of mortality has been relatively low. This likely reflects the operation of systemic induced resistance (Bonello et al., 2001), which has allowed many infected trees to recover from pitch canker (Gordon et al., 2011).

The low rate of mortality observed in native stands of *P. radiata* contrasts with a much more dramatic impact of pitch canker on landscape plantings of this species. Extensive mortality occurred in stands that were located outside the native range of *P. radiata*, and most tree deaths occurred during a protracted drought between 1987 and 1991. Thus, the impact of pitch canker was likely aggravated by stress, and hence native populations may also suffer high mortality where and when drought stress occurs in stands affected by pitch canker. Disease development in a native population of *P. muricata* appears to be consistent with this expectation. At Pt. Reyes National Seashore on the California coast north of San Francisco, a stand replacement fire occurred in 1995. Thereafter, abundant regeneration resulted in dense stands of *P. muricata* that far exceeded historic stocking levels. Consequently, trees were likely subjected to drought stress, and this may have facilitated the rapid development of pitch canker that was observed to occur between 2007 and 2009. Since that time disease severity has continued to increase. Based on plots that were established at the periphery of infection centers, mean disease severity (as a percent of maximum) rose from 2% in 2011 to 6% in 2012 and 11% in 2013 (Gordon, unpublished data).

Management of pitch canker in native populations should focus on minimizing impacts of the disease. Where pitch canker is recently established, efforts should be made to limit opportunities for expansion of the infestation. To this end, branches removed from infected trees should be disposed of locally either by burning or chipping and composting. Chipping will dramatically reduce insect emergence but will not preclude survival of *F. circinatum* for one year or more (McNee et al., 2002), and hence there is a need for further treatment. As noted above, Agustí-Brisach et al. (2012) documented that *F. circinatum* was killed by temperatures above 50 °C under controlled conditions. Consistent with that result, exposure to moist heat (50 °C or higher) for ten days was found to eliminate the pathogen in branches taken from infected trees (Gordon, unpublished data). Thus proper composting can render infested material pathogen-free.

Whereas composting will be appropriate for small diameter branches and chipped wood, this will not be an option for logs. *F. circinatum* can survive in logs cut from diseased trees for up to 18 months. Consequently, logs known or suspected to have cankers should not be transported out of an infested area without treatment, such as exposure to high temperatures (minimum continuous core temperature of 70 °C for more than 4 h) or fumigation with an effective material such sulfuryl fluoride.

Stands affected by pitch canker should be managed to promote regeneration and to achieve stocking densities that reduce the likelihood of water stress. Regeneration is desirable because intraspecific variation in susceptibility to pitch canker is common in pines, so natural selection may serve to increase the frequency of relatively resistant individuals within a population over time. Where mortality is extensive, it may be necessary to suppress the growth of invasive plant species, which might otherwise cover open ground to the exclusion of pine seedlings.

4. Conclusions

An overarching goal in managing diseases of conifers caused by *Fusarium* species should be to enhance genetic resistance in susceptible species. Whereas breeding for resistance to pitch canker has shown great promise, comparable efforts to develop resistance to *Fusarium* root- and hypocotyl rot have not been undertaken (Weiland et al., 2013). In part, this differential can be attributed to the fact that pre-plant fumigation has historically provided adequate control of root diseases. The impending loss of efficacious fumigants should inspire more attention to development of resistant lines of commercially important species. These efforts will be aided by the recent recognition of *F. commune* as a principal cause of root rot in Douglas-fir (Stewart et al., 2006), which should help to resolve ambiguities concerning the etiology of this disease.

Notwithstanding the global distribution of pitch canker, many areas where susceptible species are grown are as yet unaffected. Consequently, quarantine restrictions remain important. The principal risk lies with movement of infected or infested plant material, such as seed, seedlings, scion wood or logs. The interception of infected Douglas-fir cuttings in New Zealand (Vogler et al., 2004), where the highly susceptible *P. radiata* is widely planted, illustrates how vigilant attention to quarantine procedures can prevent introductions.

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References

Aegerter, B.J., Gordon, T.R., 2006. Rates of pitch canker induced seedling mortality among *Pinus radiata* families varying in levels of genetic resistance to *Gibberella circinata* (anamorph *Fusarium circinatum*). For. Ecol. Manag. 235, 14–17.

Agustí-Brisach, C., Pérez-Sierra, J., Armengol, J., García-Jiménez, J., Berbegal, M., 2012. Efficacy of hot water treatment to reduce the incidence of *Fusarium circinatum* on *Pinus radiata* seeds. Forestry 85, 629–635.

Aoki, T., Smith, J.A., Mount, L.L., Geiser, D.M., O'Donnell, K., 2013. *Fusarium torreyae* sp. nov., a pathogen causing canker disease of Florida torreya (*Torreya taxifolia*), a critically endangered conifer restricted to northern Florida and southwestern Georgia. Mycologia 105, 312–319.

Axelrood, P.E., Chapman, W.K., Seifert, K.A., Trotter, D.B., Shrimpton, G., 1998. *Cylindrocarpon* and *Fusarium* root colonization of Douglas-fir seedlings from British Columbia reforestation sites. Can. J. For. Res. 28, 1198–1206.

Barrows-Broaddus, J., 1990. Colonization of cones and seed of loblolly pine following inoculation with *Fusarium subglutinans*. Plant Dis. 74, 1002–1005.

Berbegal, M., Pérez-Sierra, A., Armengol, J., Grünwald, N.J., 2013. Evidence for multiple introductions and clonality in Spanish populations of *Fusarium circinatum*. Phytopathology 103, 851–861.

Bezos, D., Lomba, J.M., Martinez-Alvarez, P., Fernandez, M., Diez, J.J., 2012. Effects of pruning in Monterrey pine plantations affected by *Fusarium circinatum*. For. Syst. 21, 481–488.

Blakeslee, G.M., Foltz, J.L., 1981. The deodar weevil, a vector and wounding agent associated with pitch canker of slash pine. Phytopathology 71, 861.

Bloomberg, W.J., 1971. Diseases of Douglas-fir seedlings caused by *Fusarium oxysporum*. Phytopathology 61, 467–470.

Bonanomi, G., Antignani, V., Capodilupo, M., Scala, F., 2010. Identifying the characteristics of organic soil amendments that suppress soilborne plant diseases. Soil Biol. Biochem. 42, 136–144.

Bonello, P., Gordon, T.R., Storer, A.J., 2001. Systemic induced resistance in Monterey pine. For. Pathol. 31, 99–106.

Braganca, H., Diogo, E., Moniz, F., Amaro, P., 2009. First report of pitch canker on pines caused by *Fusarium circinatum* in Portugal. Plant Dis. 93, 1079.

Britz, H., Wingfield, M.J., Coutinho, T.A., Marasas, W.F.O., Leslie, J.F., 1998. Female fertility and mating type distribution in a South African population of *Fusarium subglutinans* f. sp. *pini*. Appl.. Environ. Microbiol. 64, 2094–2095.

Britz, H., Coutinho, T.A., Gordon, T.R., Wingfield, M.J., 2001. Characterization of the pitch canker fungus, *Fusarium circinatum*, from Mexico. S. Afr. J. Bot. 67, 609–614.

Brownell, K.H., Schneider, R.W., 1983. Fusarium hypocotyl rot of sugar pine in California forest nurseries. Plant Dis. 67, 105–107.

Carey, W.A., Oak, S.W., Enebak, S.A., 2005. Pitch canker ratings of longleaf pine clones correlate with *Fusarium circinatum* infestation of seeds and seedling mortality in containers. For. Pathol. 35, 205–212.

Carlucci, A., Colatruglio, L., Frisullo, S., 2007. First report of pitch canker caused by *Fusarium circinatum* on *Pinus halepensis* and *P. pinea* in Apulia (Southern Italy). Plant Dis. 91, 1683.

[CDPR] California Department of Pesticide Regulation, 2011. Toxic Air Contaminant Program. Sacramento, CA. www.cdpr.ca.gov/docs/emon/pubs/tac/finaleval/chloropicrin.htm.

Correll, J.C., Gordon, T.R., McCain, A.H., Fox, J.W., Koehler, C.S., Wood, D.L., Schultz, M.E., 1991. Pitch canker in California: pathogenicity, distribution, and canker development on Monterey pine (*Pinus radiata*). Plant Dis. 75, 676–682.

Coutinho, T.A., Steenkamp, E.T., Mongwaketsi, K., Wilmot, M., Wingfield, M.J., 2007. First outbreak of pitch canker in a South African pine plantation. Australas. Plant Pathol. 36, 256–261.

Cram, M.M., Enebak, S.A., Fraedrich, S.W., Dwinell, L.D., Zarnoch, S.J., 2007. Evaluation of fumigants, EPTC Herbicide, and *Paenibacillus macerans* in the production of loblolly Pine seedlings. For. Sci. 53, 73–83.

Desjardins, A.E., Plattner, R.D., Gordon, T.R., 2000. *Gibberella fujikuroi* mating population A and *Fusarium subglutinans* from teosinte species and maize from Mexico and Central America. Mycol. Res. 104, 865–872.

Dreaden, T.J., Smith, J.A., Barnard, E.L., Blakeslee, G., 2012. Development and evaluation of a real-time PCR seed lot screening method for *Fusarium circinatum*, causal agent of pitch canker disease. For. Pathol. 42, 405–411.

Dumroese, R.K., James, R.L., 2005. Root diseases in bare root and container nurseries of the Pacific Northwest: epidemiology, management, and effects on outplanting performance. New For. 30, 185–202.

Dumroese, R.K., James, R.L., Wenny, D.L., Gilligan, C.J. 1988. Douglas-fir seed treatments: effects on seedborne organisms and germination. In: Landis, T.D. (Tech. Coord.) (Ed.), Proceedings: Combined Meeting of the Western Forest Nursery Assoc. USDA Forest Serv., Rocky Mtn. Forest and Range Exp. Sta., Fort Collins, Colorado. Gen. Tech. Rep. RM–167. pp. 155–160.

Dumroese, R.K., James, R.L., Wenny, D.L., 1993. Fusarium root infection of container-grown Douglas-fir: effect on survival and growth of outplanted seedlings and persistence of the pathogen. New For. 7, 143–149.

Dumroese, R.K., Kim, M.-S., James, R.L., 2012. *Fusarium oxysporum* protects Douglas-fir (*Pseudotsuga menziesii*) seedlings from root disease caused by *Fusarium oxysporum*. Plant Pathol. J. 28, 311–316.

Dwinell, L.D., 1978. Susceptibility of southern pines to infection by *Fusarium moniliforme* var. *subglutinans*. Plant Dis. Rep. 62, 108–111.

Dwinell, L.D., Barrows-Broaddus, J., 1978. Recovery of the pine pitch canker fungus from pine plantations and seed orchard soil. Phytopathol. News 12, 207.

Dwinell, L.D., Phelps, W.R., 1977. Pitch canker of slash pine in Florida. J. For. 75, 488–489.

Dwinell, L.D., Barrows-Broaddus, J.B., Kuhlman, E.G., 1985. Pitch canker: a disease complex of southern pines. Plant Dis. 69, 270–276.

Enebak, S.A., Carey, W.A., 2003. Pitch canker caused by *Fusarium circinatum* identified on spruce pine in Alabama. Plant Dis. 87, 449.

Enebak, S.A., Stanosz, G.R., 2003. Responses of conifer species of the Great Lakes region of North America to inoculation with the pitch canker pathogen *Fusarium circinatum*. For. Pathol. 33, 333–338.

Enebak, S.A., Palmer, M.A., Blanchette, R.A., 1990. Managing soil-borne pathogens of white pine in a forest nursery. Plant Dis. 74, 195–198.

Fisher, R.F., Garbett, W.S., Underhill, E.M., 1981. Effects of fertilization on healthy and pitch-canker infected pines. South J. Appl. For. 5, 77–79.

Fitza, K.N.E., Payn, K.G., Steenkamp, E.T., Myburg, A.A., Naidoo, S., 2013. Chitosan application improves resistance to *Fusarium circinatum* in *Pinus patula*. S. Afr. J. Bot. 85, 70–78.

Fourie, G., Wingfield, M.J., Wingfield, B.D., Jones, N.B., Morris, A.R., Steenkamp, E.T., 2014. Culture-independent detection and quantification of *Fusarium circinatum* in a pine-producing seedling nursery. South. For. 76, 137–143.

Fox, J.W., Wood, D.L., Koehler, C.S., O'Keefe, S.T., 1991. Engraver beetles (Scolytidae: *Ips* species) as vectors of the pitch canker fungus, *Fusarium subglutinans*. Can. Entomol. 123, 1355–1367.

Fraedrich, B.R., Witcher, W., 1982. Influence of fertilization on pitch canker development on three southern pine species. Plant Dis. 66, 938–940.

Friel, C.J., Desjardins, A.E., Kirkpatrick, S.C., Gordon, T.R., 2007. Evidence for recombination and segregation of virulence to pine in a hybrid cross between *Gibberella circinata* and *G. subglutinans*. Mycol. Res. 111, 827–831.

Ganley, R.J., Watt, M.S., Manning, L., Iturritxa, E., 2009. A global climatic risk assessment of pitch canker disease. Can. J. For. Res. 39, 2246–2256.

Ganley, R.J., Watt, M.S., Kriticos, D.J., Hopkins, A.J.M., Manning, L., 2011. Increased risk of pitch canker to Australasia under climate change. Australas. Plant Pathol. 40, 228–237.

Gebeyehu, S., Wingfield, M.J., 2003. Pine weevil *Pissodes nemorensis*: threat to South African pine plantations and options for control. S. Afr. J. Sci. 99, 531–536.

Gordon, T.R., 2011. Biology and management of *Gibberella circinata*, the cause of pitch canker in pines. In: Alves-Santos, F.M., Diez, J.J. (Eds.), Control of Fusarium Diseases. Research Signpost, Kerala, India, pp. 195–208.

Gordon, T.R., Storer, A.J., Okamoto, D., 1996. Population structure of the pitch canker pathogen, *Fusarium subglutinans* f. sp. *pini*, in California. Mycol. Res. 100, 850–854.

Gordon, T.R., Okamoto, D., Storer, A.J., Wood, D.L., 1998a. Susceptibility of five landscape pines to pitch canker disease, caused by *Fusarium subglutinans* f. sp. *pini*. HortScience 33, 868–871.

Gordon, T.R., Wikler, K.R., Clark, S.L., Okamoto, D., Storer, A.J., Bonello, P., 1998b. Resistance to pitch canker disease, caused by *Fusarium subglutinans* f.sp. *pini*, in Monterey pine (*Pinus radiata*). Plant Pathol. 47, 706–711.

Gordon, T.R., Storer, A.J., Wood, D.L., 2001. The pitch canker epidemic in California. Plant Dis. 85, 1128–1139.

Gordon, T.R., Kirkpatrick, S.C., Aegerter, B.J., Wood, D.L., Storer, A.J., 2006a. Susceptibility of Douglas fir (*Pseudotsuga menziesii*) to pitch canker, caused by *Gibberella circinata* (anamorph = *Fusarium circinatum*). Plant Pathol. 55, 231–237.

Gordon, T.R., Kirkpatrick, S.C., Petersen, J.C., Friel, C.J., 2006b. Potential diversity in vegetative compatibility groupings in the California population of *Gibberella circinata*. Mycol. Res. 100, 936–940.

Gordon, T.R., Kirkpatrick, S.C., Aegerter, B.J., Fisher, A.J., Storer, A.J., Wood, D.L., 2011. Evidence for the occurrence of induced resistance to pitch canker, caused by *Gibberella circinata* (anamorph *Fusarium circinatum*), in populations of *Pinus radiata*. For. Pathol. 41, 227–232.

Graham, J.H., Linderman, R.G., 1983. Pathogenic seedborne *Fusarium oxysporum* from Douglas-fir. Plant Dis. 67, 323–325.

Guerra-Santos, J.J., 1999. Pitch canker in Monterey pine in Mexico. In: Devey, M., Matheson, C., Gordon, T. (Eds.), Current and Potential Impacts of Pitch Canker in Radiata Pine. Proc. IMPACT Monterey Workshop, Monterey, CA. CSIRO, Australia, pp. 58–61.

Hansen, E.M., Myrold, D.D., Hamm, P.B., 1990. Effects of soil fumigation and cover crops on potential pathogens, microbial activity, nitrogen availability, and seedling quality in conifer nurseries. Phytopathology 80, 698–704.

Hepting, G.H., Roth, E.R., 1946. Pitch canker, a new disease of some southern pines. J. For. 44, 742–744.

Hepting, G.H., Roth, E.R., 1953. Host relations and spread of the pine pitch canker disease. Phytopathology 43, 475–475.

Hildebrand, D.M., Stone, J.K., James, R.L., Frankel, S.J., 2004. Alternatives to Preplant Soil Fumigation for Western Forest Nurseries. General Technical Report PNW-GTR-608. USDA Forest Service.

Hodge, G.R., Dvorak, W.S., 2000. Differential responses of Central American and Mexican pine species and *Pinus radiata* to infection by the pitch canker fungus. New For. 19, 241–258.

Hoover, K., Wood, D.L., Fox, J.W., Bros, W.E., 1995. Quantitative and seasonal association of the pitch canker fungus, *Fusarium subglutinans* f. sp. *pini* with *Conophthorus radiatae* (Coleoptera: Scolytidae) and *Ernobius punctulatus* (Coleoptera: Anobiidae) which infest *Pinus radiata*. Can. Entomol. 127, 79–91.

Hoover, K., Wood, D.L., Storer, A.J., Fox, J.W., Bros, W.E., 1996. Transmission of the pitch canker fungus, *Fusarium subglutinans* f. sp. *pini*, to Monterey pine. Can. Entomol. 128, 981–994.

Huang, J.W., Kuhlman, E.G., 1990. Fungi associated with damping-off of slash pine seedlings in Georgia. Plant Dis. 74, 27–30.

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

Ioos, R., Fourrier, C., Iancu, G., Gordon, T.R., 2009. Sensitive detection of *Fusarium circinatum* in pine seed by combining an enrichment procedure with a real-time polymerase chain reaction using dual labeled probe chemistry. Phytopathology 99, 582–590.

Iturritxa, E., Ganley, R.J., Wright, J., Heppe, E., Steenkamp, E.T., Gordon, T.R., Wingfield, M.J., 2011. A genetically homogenous population of *Fusarium circinatum* causes pitch canker of *Pinus radiata* in the Basque Country, Spain. Fungal Biol. 115, 288–295.

James, R.L., 1987. Occurrence of Fusarium on conifer tree seed from Northern Rocky Mountain nurseries. In: Proceedings: Combined Western Forest Nursery Council and Intermountain Nursery Assoc. Meeting. T.D. Landis (Tech. Coord.). USDA Forest Serv., Rocky Mtn. Forest and Range Exp. Sta., Fort Collins, Colorado, pp. 109–114. Gen. Tech. Rep. RM–137.

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James, R.L., 1997. Effects of Fertilizer on Selected Potential Plant Pathogens in Bare Root Forest Nurseries. Symposium Proceedings: Forest Seedling Nutrition from the Nursery to the Field. Nursery Tech. Coop., Oregon State Univ., Corvallis, Oregon.

James, R.L., 2001. Effects of Pre-sowing Soil Treatments on Root Colonization of 1–0 Ponderosa and Lodgepole Pine Seedlings by Potentially-pathogenic Fungi – USDA Forest Service Lucky Peak Nursery. USDA Forest Service, Boise, Idaho, p. 9. Forest Health Prot. Rep. 01–9.

James, R.L., 2003. Fusarium Blight of Container-grown Ponderosa Pine Seedlings – Montana State Nursery, Missoula, Montana. USDA Forest Serv., Forest Health Protection, Missoula, Montana, p. 14. Nursery Disease Notes No. 149.

James, R.L., 2012. Fusarium root and stem diseases. In: Cram, M.M., Frank, M.S., Mallams, K.M. (Eds.), Forest Nursery Pests, Agriculture Handbook No. 680. USDA Forest Service, pp. 117–120.

James, R.L., Genz, D., 1981. Ponderosa Pine Seed Treatments: Effects on Seed Germination and Disease Incidence. USDA Forest Serv., Forest Health Protection, Missoula, Montana, p. 13. Rep. 81–16.

James, R.L., Perez, R., 1999. Pathogenic Characteristics of *Fusarium Sporotrichioides* Isolated from Inland Pacific Northwest Forest Nurseries. USDA For. Serv., Northern Region. For. Health Prot. Rep. 99–8.

James, R.L., Militante, E.P., Woo, J.Y., Gilligan, C.J., 1986. Pathogenicity of *Fusarium* from Forest Seedling Nurseries on Douglas-fir and Ponderosa Pine Seedlings. For. Pest Manage. Rep. No. 86–8. USDA For. Serv., North. Reg.

James, R.L., Dumroese, R.K., Wenny, D.L., 1997. Pathogenicity of *Fusarium proliferatum* in container-grown Douglas-fir seedling. In: James, R.L. (Ed.), Proc. Third Meeting of IUFRO Working Party S7.03-04 (Disease and Insects in Forest Nurseries). USDA For. Serv., Northern Region, pp. 26–33. For. Health Prot. Rep. 97-4.

James, R.L., Knudsen, G.R., Morra, M.J., 2004. Preplant Soil Treatment Effects on Production of Douglas-fir Seedlings at the USDA Forest Service Nursery, Coeur D'alene, Idaho. Forest Health Protection. USDA Forest Service Report 04–10.

Jones, N.B., Ford, C.M., Light, M.E., Nadel, R.L., Greyling, I., Fourie, G., Wingfield, M.J., Morris, A.R., 2014. Effect on nursery and field performance of *Pinus patula* seedlings after inoculation with *Fusarium circinatum*. South. For. 76, 125–136.

Kanzler, A., Nel, A., Ford, C., 2014. Development and commercialisation of the *Pinus patula* x *P. tecunumanii* hybrid in response to the threat of *Fusarium circinatum*. New For. 45, 417–437.

Kim, M.-S., Stewart, J.E., Dumroese, R.K., Klopfenstein, N.B., 2012. Occurrence of the root rot pathogen, Fusarium commune, in Forest nurseries of the Midwestern and western United States. J. Phytopathol. 160, 112–114.

Kobayashi, T., Muramoto, M., 1989. Pitch canker of *Pinus luchuensis*, a new disease of Japanese forests. For. Pests 40, 169–173.

Kuhlman, E.G., 1987. Effect of inoculation treatment with *Fusarium moniliforme* var. *subglutinans* on dieback of loblolly and slash pine seedlings. Plant Dis. 71, 161–162.

Landeras, E., Garcıa, P., Fernández, Y., Braña, M., Fernández-Alonso, O., et al., 2005. Outbreak of pitch canker caused by *Fusarium circinatum* on *Pinus* spp. in northern Spain. Plant Dis. 89, 1015.

Lazreg, F., Belabid, L., 2013a. First Report of *Fusarium redolens* as a causal agent of Aleppo Pine damping-off in Algeria. Plant Dis. 97, 997.

Lazreg, F., Belabid, L., 2013b. First Report of *Fusarium chlamydosporum* causing damping-off disease on Aleppo pine in Algeria. Plant Dis. 97, 1506.

Lazreg, F., Belabid, L., Sanchez, J., Gallego, E., Garrido-Cardenas, J.A., Elhaitoum, 2014. First report of *Fusarium equiseti* causing damping-off disease on Aleppo Pine in Algeria. Plant Dis. 98, 1268.

Lee, J.K., Lee, S.-H., Yang, S.-I., Lee, Y.-W., 2000. First report of pitch canker disease on *Pinus rigida* in Korea. Plant Pathol. J. 16, 52–54.

Leslie, J.F., Summerell, B.A., Bullock, S., 2006. The *Fusarium* Lab Manual. Blackwell, Ames, IA.

Linderman, R.G., Davis, E.A., Masters, C.J., 2008. Efficacy of chemical and biological agents to suppress *Fusarium* and *Pythium* damping-off of container-grown Douglas-fir seedlings. Plant Health Progr. http://dx.doi.org/10.1094/PHP-2008-0317-02-RS.

Lopez-Zamora, I., Bliss, C., Jokela, E.J., Comerford, N.B., Grunwald, S., Barnard, E., Vasquez, G.M., 2007. Spatial relationships between nitrogen status and pitch canker disease in slash pine planted adjacent to a poultry operation. Environ. Pollut. 147, 101–111.

Marois, J.J., Dunn, M.T., Papavizas, G.C., 1983. Reinvasion of fumigated soil by *Fusarium oxysporum* f. sp. *melonis*. Phytopathology 73, 680–684.

Mason, G.N., Van Arsdell, E.P., 1978. Fungi associated with *Pinus taeda* seed development. Plant Dis. Rep. 62, 864–867.

Matheson, A.C., Devey, M.E., Gordon, T.R., Werner, W., Vogler, D.R., Balocchi, C., Carson, M.J., 2006. Heritability of response to inoculation by pine pitch canker of seedlings of radiata pine. Aust. For. J. 69, 101–106.

McCain, A.H., Koehler, C.S., Tjosvold, S.A., 1987. Pitch canker threatens California pines. Calif. Agric. 41, 22–23.

McNee, W.R., Wood, D.L., Storer, A.J., Gordon, T.R., 2002. Incidence of the pitch canker pathogen and associated insects in intact and chipped Monterey pine branches. Can. Entomol. 134, 47–58.

Mitchell, R.G., Coutinho, T.A., Steenkamp, E., Herbert, M., Wingfield, M.J., 2012a. Future outlook for *Pinus patula* in South Africa in the presence of the pitch canker fungus (*Fusarium circinatum*). South. For. 74, 203–210.

Mitchell, R.G., Wingfield, M.J., Steenkamp, E.T., Coutinho, T.A., 2012b. Tolerance of *Pinus patula* full-sib families to *Fusarium circinatum* in a greenhouse study. South. For. 74, 247–252.

Mitchell, R.G., Wingfield, M.J., Hodge, G.R., Steenkamp, E.T., Coutinho, T.A., 2012c. Selection of *Pinus* spp. in South Africa for tolerance to infection by the pitch canker fungus. New For. 43, 473–489.

Morris, A.R., Fourie, G., Greyling, I., Steenkamp, E.T., Jones, N.B., 2014. Re-use of seedling containers and *Fusarium circinatum* association with asymptomatic *Pinus patula* planting stock. South. For. 76, 161–166.

Mousseaux, M.R., Dumroese, R.K., James, R.L., Wenny, D.L., Knudsen, G.R., 1998. Efficacy of *Trichoderma harzianum* as a biological control of *Fusarium oxysporum* in container-grown Douglas-fir seedlings. New For. 15, 11–21.

Muramoto, M., Tashiro, T., Minamihashi, H., 1993. Distribution of *Fusarium moniliforme* var. *subglutinans* in Kagoshima prefecture and its pathogenicity to pines. J. Jpn. For. Soc. 75, 1–9.

Nelson, P.E., Toussoun, T.A., Marasas, W.F.O., 1983. *Fusarium* Species: an Illustrated Manual for Identification. Penn. State Univ. Press, University Park, PA, p. 193.

Nelson, S.D., Ajwa, H.A., Trout, T., Stromberger, M., Yates, S.R., Sharma, S., 2014. Water and methyl isothiocyanate distribution in soil after drip fumigation. J. Environ. Qual. 42, 1555–1564.

Njoroge, S.M.C., Kabir, Z., Martin, F.N., Koike, S.T., Subbarao, K.V., 2009. Comparison of crop rotation for Verticillium wilt management and effect on *Pythium* species in conventional and organic strawberry production. Plant Dis. 93, 519–527.

Old, K.M., Libby, W.J., Russell, J.H., 1986. Genetic variability in susceptibility of *Pinus radiata* to western gall rust. Silvae Genet. 35, 145–149.

Owen, D., Adams, D., 2001. Impact of pitch canker on ornamental Monterey pines in Santa Cruz County, California, U.S., 1987-2000. J. Arboric. 27, 298–305.

Pfenning, L.H., da Silva CostaI, S., de Melo, M.P., CostaII, H., Ventura, J.A., Auer, C.G., dos Santos, A.F., 2014. First report and characterization of *Fusarium circinatum*, the causal agent of pitch canker in Brazil. Trop. Plant Pathol. 39, 210–216.

Ramsfield, T.D., Dobbie, K., Dick, M.A., Ball, R.D., 2008. Polymerase chain reaction-based detection of *Fusarium circinatum*, the causal agent of pitch canker disease. Mol. Ecol. Res. 8, 1270–1273.

Rockwood, D.L., Blakeslee, G.M., Lowerts, G.A., Underhill, E.M., Oak, S.W., 1988. Genetic strategies for reducing pitch canker incidence in slash pine. South. J. Appl. For. 12, 28–32.

Romón, P., Iturrondobeitia, J.C., Gibson, K., Lindgren, B.S., Goldarazena, A., 2007. Quantitative association of bark beetles with pitch canker fungus and effects of verbenone on their semiochemical communication in Monterey pine forests in northern Spain. Environ. Entomol. 36, 743–750.

Roux, J., Eisenberg, B., Kanzler, A., Nel, A., Coetzee, V., Kietzka, E., Wingfield, M.J., 2007. Testing of selected South African *Pinus* hybrids and families for tolerance to the pitch canker pathogen, *Fusarium circinatum*. New For. 33, 109–123.

Runion, G.B., Bruck, R.I., 1988. Effects of thiabendazole-DMSO treatment of longleaf pine seed contaminated with *Fusarium subglutinans* on germination and seedling survival. Plant Dis. 72, 872–874.

Runion, G.B., Cade, S.C., Bruck, R.I., 1993. Effects of carbofuran and thiabendazole on incidence of pitch canker in loblolly pine. Plant Dis. 77, 166–169.

Sakamoto, J.M., Gordon, T.R., 2006. Factors influencing infection of mechanical wounds by *Fusarium circinatum* on Monterey pines (*Pinus radiata*). Plant Pathol. 55, 130–136.

Sakamoto, J.M., Gordon, T.R., Storer, A.J., Wood, D.L., 2007. The role of *Pityophthorus* spp. as vectors of pitch canker affecting *Pinus radiata*. Can. Entomol. 139, 864–871.

Schisler, D.A., Linderman, R.G., 1984. Evidence for the involvement of the soil microbiota in the exclusion of *Fusarium* from coniferous forest soils. Can. J. Microbiol. 30, 142–150.

Schweigkofler, W., O'Donnell, K., Garbelotto, M., 2004. Detection and quantification of *Fusarium circinatum*, the causal agent of pine pitch canker, from two California sites by using a real-time PCR approach combined with a simple spore trapping method. Appl.. Environ. Microbiol. 70, 3512–3520.

Skovgaard, K., Rosendahl, S., O'Donnell, K., Hirenberh, H.I., 2003. *Fusarium commune* is a new species identified by morphological and molecular phylogenetic data. Mycologia 95, 630–636.

Slinski, S.L., 2012. Factors Affecting Virulence of *Gibberella Circinata* on *Pinus Radiata*. Dissertation. University of California, Davis, p. 56.

Slinski, S.L., Zakharov, F., Gordon, T.R., 2015. The effect of resin and monoterpenes on spore germination and growth in *Fusarium circinatum*. Phytopathology 105, 119–125.

Smith Jr., R.S., 1967. Decline of *Fusarium oxysporum* in the roots of *Pinus lambertiana* seedlings transplanted into forest soils. Phytopathology 57, 1265.

South, D.B., Carey, W.A., Enebak, S.A., 1997. Chloropicrin as a soil fumigant in forest nurseries. For. Chron. 73, 489–494.

Steenkamp, E.T., Wingfield, B.D., Coutinho, T.A., Wingfield, M.J., Marasas, W.F.O., 1999. Differentiation of *Fusarium subglutinans* f. sp. *pini* by histone gene sequence data. Appl. Environ. Microbiol. 65, 3401–3406.

Steenkamp, E.T., Rodas, C.A., Kvas, M., Wingfield, M.J., 2012. *Fusarium circinatum* and pitch canker of *Pinus* in Colombia. Australas. Plant Pathol. 41, 483–491.

Steenkamp, E.T., Makhari, O.M., Coutinho, T.A., Wingfield, B.D., Wingfield, M.J., 2014. Evidence for a new introduction of the pitch canker fungus *Fusarium circinatum* in South Africa. Plant Pathol. 63, 530–538.

Stewart, J.E., Kim, M.-S., James, R.L., Dumroese, R.K., Klopfenstein, N.B., 2006. Molecular characterization of *Fusarium oxysporum* and *Fusarium commune* isolates from a conifer nursery. Phytopathology 96, 1124–1133.

Stewart, J.E., Abdo, Z., Dumroese, R.K., Klopfenstein, N.B., Kim, M.-S., 2012. Virulence of *Fusarium oxysporum* and *F. commune* to Douglas-fir (*Pseudotsuga menziesii*) seedlings. For. Pathol. 42, 220–228.

Storer, A.J., Gordon, T.R., Wood, D.L., Bonello, P., 1997. Pitch canker disease of pines:

current and future impacts. J. For. 95, 21–26.

Storer, A.J., Gordon, T.R., Clark, S.L., 1998a. Association of the pitch canker fungus, *Fusarium subglutinans* f. sp. *pini* with Monterey pine seeds and seedlings in California. Plant Pathol. 47, 649–656.

Storer, A.J., Wood, D.L., Wikler, K.R., Gordon, T.R., 1998b. Association between a native spittlebug (Homoptera: Cercopidae) on Monterey pine and an introduced tree pathogen which causes pitch canker disease. Can. Entomol. 130, 783–792.

Storer, A.J., Bonello, P., Gordon, T.R., Wood, D.L., 1999. Evidence of resistance to the pitch canker pathogen (*Fusarium circinatum*) in native stands of Monterey pine (*Pinus radiata*). For. Sci. 45, 500–505.

Storer, A.J., Wood, D.L., Gordon, T.R., 2004. Twig beetles, *Pityophthorus* spp. (Coleoptera: Scolytidae), as vectors of the pitch canker pathogen in California. Can. Entomol. 136, 685–693.

Swart, W.J., Wingfield, M.J., 1991. Biology and control of *Sphaeropsis sapinea* on *Pinus* spp. in South Africa. Plant Dis. 75, 761–766.

Swett, C.L., 2013. The Biology and Ecology of Cryptic Life History Strategies of the Pitch Canker Pathogen *Fusarium Circinatum* in *Pinus Radiata* Seedlings and Alternate Grass Hosts. Dissertation. University of California, Davis, p. 150.

Swett, C.L., Gordon, T.R., 2012. First report of grass species (Poaceae) as naturally occurring hosts of the pine pathogen *Gibberella circinata*. Plant Dis. 96, 908–908.

Swett, C.L., Gordon, T.R., 2013. Dualism in symbiosis: growth and defense enhancement of symptomless infection by the pathogen *Fusarium circinatum* in *Pinus radiata* seedlings. Phytopathology 103. S191–S191.

Swett, C.L., Gordon, T.R., 2015. Endophytic association of the pine pathogen *Fusarium circinatum* with corn (*Zea mays*). Fungal Ecol. 13, 120–129.

Swett, C.L., Huang, M., Begovic, A., Steenkamp, E.T., Wingfield, M.J., Gordon, T.R., 2013. A new dimension to pitch canker epidemiology: Biology of *Fusarium circinatum* as a grass colonist in native and managed pine systems. In: Proceedings of the 2012 Western International Forest Disease Work Conference.

Swett, C.L., Porter, B., Fourie, G., Steenkamp, E.T., Gordon, T.R., Wingfield, M.J., 2014. Association of the pitch canker pathogen *Fusarium circinatum* with grass hosts in commercial pine production areas of South Africa. South. For. 2014, 1–6.

Tint, H., 1945. Studies in the Fusarium damping-off of conifers. I. The comparative virulence of certain Fusaria. Phytopathology 35, 421–439.

Toussoun, T.A., Menzinger, W., Smith, R.S., 1969. Role of conifer litter in the ecology of *Fusarium*: stimulation of germination in soil. Phytopathology 59, 1369–1399.

Van Wyk, S.J.P., Boutigny, A.-L., Coutinho, T.A., Viljoen, A., 2012. Sanitation of a South African forestry nursery contaminated with *Fusarium circinatum* using hydrogen peroxide at specific oxidation reduction potentials. Plant Dis. 96, 875–880.

Viljoen, A., Wingfield, M.J., 1994. First report of *Fusarium subglutinans* f. sp. *pini* on pine seedlings in South Africa. Plant Dis. 78, 309–312.

Vogler, D.R., Gordon, T.R., Aegerter, B.J., Kirkpatrick, S.C., Lunak, G.A., Stover, P., Violett, P., 2004. First report of the pitch canker fungus (*Fusarium circinatum*) in the Sierra Nevada of California. Plant Dis. 88, 772–772.

Wall, R.E., 1984. Effects of recently incorporated organic amendments on damping-off of conifer seedlings. Plant Dis. 68, 59–60.

Wang, D., Fraedrich, S.W., Juzwik, J., Spokas, K., Zhang, Y., Koskinen, W.C., 2006. Fumigant distribution in forest nursery soils under water seal and plastic film after application of dazomet, metam-sodium and chloropicrin. Pest Manag. Sci. 62, 263–273.

Weiland, J.E., Littke, W.R., Haase, D.L., 2013. Forest nurseries face critical choices with the loss of methyl bromide fumigation. Calif. Agric. 67, 153–161.

Wikler, K., Gordon, T.R., 2000. An initial assessment of genetic relationships among populations of *Fusarium circinatum* in different parts of the world. Can. J. Bot. 78, 709–717.

Wikler, K.R., Gordon, T.R., Clark, S.L., 2000. Potential for outcrossing in an apparently asexual population of *Fusarium circinatum*, the casual agent of pitch canker disease. Mycologia 92, 1085–1090.

Wikler, K.R., Storer, A.J., Newman, W., Gordon, T.R., Wood, D.L., 2003. The dynamics of an introduced pathogen in a native Monterey pine (*Pinus radiata*) forest. For. Ecol. Manag. 179, 209–221.

Wingfield, M.J., Jacobs, A., Coutinho, T.A., Ahumada, R., Wingfield, B.D., 2002. First report of the pitch canker fungus, *Fusarium circinatum*, on pines in Chile. Plant Pathol. 51, 397.

Wingfield, M.J., Hammerbacher, A., Ganley, R.J., Steenkamp, E.T., Gordon, T.R., Wingfield, B.D., Coutinho, T.A., 2008. Pitch canker caused by *Fusarium circinatum*: a growing threat to pine plantations and forests worldwide. Australas. Plant Pathol. 37, 319–334.

Woltz, S.S., Jones, J.P., 1981. Nutritional requirements of *Fusarium oxysporum*: basis for a disease control system. In: Nelson, R.E., Toussoun, T.A., Cook, R.J. (Eds.), Fusarium: Diseases, Biology and Taxonomy. The Pennsylvania State University Press, University Park, pp. 340–349.

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