

Managing an Epidemic of Swiss Needle Cast in the Douglas-Fir Region of Oregon: The Role of the Swiss Needle Cast Cooperative

David C. Shaw, Gregory M. Filip, Alan Kanaskie,
Douglas A. Maguire, and Will A. Littke

ABSTRACT

Swiss needle cast (SNC) is a foliage disease specific to Douglas-fir (*Pseudotsuga menziesii*) caused by the Ascomycete fungus *Phaeocryptopus gaeumannii*. Currently, it is causing an epidemic west of the Oregon Coast Range from Coos Bay to Astoria: 2010 aerial survey estimate of almost 400,000 ac of plantations with visible symptoms. General symptoms include chlorotic needles, decreased needle retention resulting in sparse crowns, and growth reductions. Growth losses in the epidemic area have been in the range of 20–55%, although mortality from the disease is rare. The SNC Cooperative (SNCC) was established in January 1997 at Oregon State University with private, state, and federal partners. The mission of the SNCC is to conduct research on enhancing Douglas-fir productivity and health in the presence of SNC. We present an integrated pest management framework for SNC on the Oregon Coast based on research sponsored by the SNCC.

Keywords: Swiss needle cast, Douglas-fir, silviculture, integrated pest management, forest pathology/foilage disease

In the early 1980s foresters in Oregon noted that Douglas-fir trees within several miles of the Pacific Coast were yellow (chlorotic; Figure 1), had thin crowns with minimal foliage (Figure 2), and did not appear to be growing well (Hansen et al. 2000, Thies and Goheen 2002). Visible symptoms of chlorosis and foliage loss were most evident in the spring before bud break (Figure 3). Symptoms continued to become increasingly apparent throughout the coastal

strip, from Coos Bay to Astoria, Oregon, and along the Washington Coast. The problem also was reported to be increasing around the Puget Sound and some inland Oregon valleys. Mounting evidence suggested that the observed symptoms were attributable to a needle disease known as Swiss needle cast (SNC). Although the name suggests the disease is from Europe, the Ascomycete fungus that causes the disease, *Phaeocryptopus gaeumannii*, is native to North

America, occurs wherever Douglas-fir is found, and is specific to Douglas-fir (Hansen et al. 2000). Mortality is rare, but premature loss of older foliage reduces growth. In young plantations (10–30 years old) in north coastal Oregon, height growth was reduced by ~25% and basal area by 35% in heavily infected stands, resulting in an average volume growth loss of 23% over the target population and up to a 52% volume growth loss in the most heavily impacted plantations (Maguire et al. 2002). Approximately 4 million ac along the western flank of the Oregon Coast Range is affected by the disease (Figure 4). The fungus also causes significant damage in Washington State (Omdal et al. 2001, Omdal and Ramsey-Kroll 2010) and in British Columbia along the coast and in some interior ranges (Hood 1982).

SNC and its causal agent, *P. gaeumannii*, were first described in Switzerland in 1925 (Boyce 1940). The disease was then noted in other parts of Europe and in New Zealand (Hood and Kershaw 1975) and is now known to be a potentially significant dis-

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David Shaw (dave.shaw@oregonstate.edu) is extension forest health specialist, Oregon State University, Department of Forestry, 218 Richardson Hall, Corvallis, OR 97331-5752. Gregory M. Filip (gmfilip@fs.fed.us) is regional forest pathologist, US Forest Service, Forest Health Protection, Portland, OR. Alan M. Kanaskie (alan.kanaskie@state.or.us) is forest pathologist, Oregon Department of Forestry, Salem, OR. Douglas A. Maguire (doug.maguire@oregonstate.edu) is Giustina Professor of forest management, Department of Forest Engineering, Resources, and Management, Oregon State University, Corvallis, OR. Will A. Littke (will.littke@weyerhaeuser.com) is forest pathologist, Weyerhaeuser, Federal Way, WA. Funds for the activities of the Swiss Needle Cast Cooperative (SNCC) are provided by members of the cooperative and the State of Oregon. The SNCC is housed in the Department of Forest Engineering, Resources and Management at Oregon State University, College of Forestry. Special thanks to the foresters, graduate students, and scientists who worked on SNC in coastal Oregon. Thanks also to Doug Mainwaring, Travis Woolley, and Greg Latta at Oregon State University.

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ease wherever Douglas-fir is grown outside its native range. Although the fungus was recognized as ubiquitous in native Douglas-fir forests, it had never been considered a significant threat to forest health (Boyce 1940, Peace 1962). In the 1970s, however, SNC was recognized as a problem in Christmas tree plantations in western Washington and Oregon, and by the 1980s the disease was common in Christmas tree plantations throughout the region (Michaels and Chastagner 1984). SNC became increasingly apparent in forest plantations in the Coast Ranges of Oregon and Washington since the 1970s, becoming so severe by the mid-1990s to prompt formation of a research cooperative at Oregon State University (OSU; SNC Cooperative [SNCC]) to investigate the problem.

The SNCC was instigated and has been supported by a group of over 25 timber-growing companies and government land-management agencies since 1996, while also being supported by the state of Oregon. Current members include Starker Forests, Stimson Lumber, Hampton Resources, Weyerhaeuser, Oregon Department of Forestry, US Forest Service, and US Department of the Interior (USDI) Bureau of Land Management (BLM). OSU, College of Forestry and OSU Forestry and Natural Resources Extension fund the Director salary and the cooperative is housed in the Department of Forest Engineering, Resources and Management. This public/private partnership has undertaken basic and applied research regarding SNC and has made significant progress toward understanding the epidemic and its causal factors (SNCC 2010).

The disease symptoms caused by SNC appear to fluctuate within the epidemic area, and research indicates that warmer winter temperatures and wet springs and early summers may be associated with intensification of the disease in coastal plantations (Manter et al. 2005, Stone et al. 2008a, Black et al. 2010). SNC is now one of the principle forest health issues in the Douglas-fir region and is forcing changes in silviculture and management of affected lands. The purpose of this review is to summarize the state of our knowledge on SNC and to describe the influence of the research cooperative on the management of Douglas-fir in coastal Oregon. Core focus areas of the SNCC include (1) disease distribution and severity; (2) biology, ecology, and epidemiology of *P. gauemannii*; (3) effect of silvicultural treatments on disease; (4) models to predict growth im-



Figure 1. Douglas-fir with chlorotic (yellow) foliage associated with Swiss needle cast. Note the green western hemlock behind the Douglas-fir.

pacts and geographic distribution of disease severity; and (5) integrated pest management (IPM) for SNC.

Disease Distribution and Severity

Disease distribution and severity is fundamental to understanding the importance and impacts of SNC, to determining where management changes are necessary, and in developing an IPM strategy. The SNCC collaborates with the Oregon Department of Forestry and the Forest Health Protection program of the US Forest Service to conduct an aerial survey each year just before bud break in spring (late April/early May) when

symptoms are most visible (Kanaskie and McWilliams 2009; Figure 3). Symptomatic plantations are concentrated along the coast and at lower elevations west of the Oregon Coast Range crest (Figure 5). In 1996, symptoms were detected from the air on 131,088 ac (Figure 6). Acreage increased to a peak of 293,649 ac in 1999, dipped, and then rose to 387,040 in 2002. After dipping again, detections over the past 5 years have surpassed 300,000 ac and 2010 was the highest acreage recorded to date (Figure 6). All indications are that the disease has intensified within the epidemic area and spread marginally to the east (Figure 5).

Comparable aerial surveys were flown

in Washington only in 1999 and 2000, indicating visible symptoms on about 180,000 ac at low elevations close to the Pacific Coast (Omdal et al. 2001). Foresters have also noted a band of SNC-impacted Douglas-fir in coastal northern California, and a visit to the region by SNCC personnel documented the presence of *P. gaeumannii*; however, formal surveys and stand assessments have not been made in that state.

Although *P. gaeumannii* is widespread, significant disease only emerges in certain situations, such as Christmas tree plantations, the valley floor and wet sites along the Cascade Range, and in the current epidemic area. In the coast range of Oregon, symptoms appear most pronounced in young Douglas-fir plantations at low elevations, on south-facing aspects, in the Sitka spruce/western hemlock plant association zone (see Franklin and Dyrness, 1973, for description of vegetation zones of Oregon). Some evidence suggests that mild winters and wet spring/summers along the Oregon Coast are responsible for disease symptom development, particularly where Douglas-fir has been planted in areas previously dominated by red alder (*Alnus rubra*), western hemlock (*Tsuga heterophylla*), western redcedar (*Thuja plicata*), and Sitka spruce (*Picea sitchensis*). Research plots established collaboratively by the SNCC and US Forest Service on the western slope of the Oregon Cascades, approximately 75 mi (120 km) east of the Coast Range epidemic zone on the other side of the Willamette Valley, found no significant SNC disease expression (Filip et al. 2006). The lack of disease expression in the Cascade Range may be due to the colder winter temperatures and minimal summer rain and drizzle in that region.

One line of reasoning, therefore, suggests that SNC is limited to the west slope of the Coast Range because the genetic source of planted Douglas-fir seedlings was inappropriate for these sites and, therefore, that the disease is of no concern outside this region. Although “off-site planting” may have contributed to the disease in some areas, particularly during reforestation of the Tillamook burns, this mechanism does not explain its wider spread occurrence in the native range of Douglas-fir. The most severe foliage loss and highest growth impacts have been measured in the area around Tillamook, Oregon. Older trees up to 60–80 years old have also expressed chlorosis, premature foliage loss, and significant growth



Figure 2. Douglas-fir with poor foliage retention associated with Swiss needle cast.

reductions attributable to SNC (Mainwaring et al. 2005, Black et al. 2010).

In 1996 the SNCC installed a series of stand-level transects for ground truthing the aerial surveys and permanent plots to estimate growth impacts in the epidemic area. The target population was all 10–30-year-old Douglas-fir plantations in north coastal Oregon between Newport and Astoria and within 18 mi of the coast. Foliage retention measured on these transects established the range of foliage retention and its geographic distribution. A set of 70 growth impact plots were established in 1998 at the same locations and have formed the basis of the relationship between foliage retention and growth losses (Maguire et al. 2002). These

plots were remeasured four times and continue to be an important mechanism for monitoring foliage retention and quantifying growth losses thereby complementing the aerial survey of foliage discoloration (Figure 7).

The loss of foliage associated with SNC changes crown structure with respect to foliage longevity, its vertical distribution, and its age class structure (Weiskittel and Maguire 2007, Weiskittel et al. 2006, 2007). In trees heavily impacted by SNC, the upper crown is composed primarily of 1- and 2-year foliage while older foliage occurs mostly in the lower crown (Weiskittel et al. 2006). Branch diameters in the lower crown of trees with severe SNC are larger than those

in healthy trees (Weiskittel and Maguire 2007), presumably because the lower foliage density within the crowns of heavily impacted trees (Maguire and Kanaskie 2002) allows more light to reach the lower branches. Similarly, total branchwood surface area for a tree of given height, diameter, and crown length was lower under conditions of severe SNC (Weiskittel and Maguire 2007).

The historical impacts of SNC on mature (~80 years old) Douglas-fir trees was assessed for three even-aged stands near Tillamook, Oregon, by using dendrochronological tree ring analysis and by comparing western hemlock and Douglas-fir of similar age (Black et al. 2010). Only one of these stands experienced severe radial growth reductions in Douglas-fir compared with western hemlock, while another stand indicated moderate impacts and the third stand had no growth differences between the two species. This variation in growth impacts is consistent with geographic variation documented by Hansen et al. (2000), Maguire et al. (2002), and Rosso and Hansen (2003). Growth reductions in the severely impacted stand were documented as early as 1950, but the most severe impacts did not occur until after 1984 and became especially severe after 1996. At the most severe site, mature Douglas-fir trees had growth reductions approaching 85% with some trees showing 10 or more missing growth rings. Warm conditions from March through August were associated with reduced radial growth at the most severely impacted site, which was a distinct finding from the previous epidemiology work that associated winter temperature and spring leaf wetness as the primary environmental factors affecting disease. The study suggests that the SNC epidemic is a new phenomenon on the coast, mature stands are susceptible to disease, and that the SNC epidemic is increasing in severity.

SNC severity has an effect on wood quality that is influenced by the impact of the disease on both growth rate and proportion of earlywood. Comparison of severely infected trees that were treated with fungicides to those that were not (Johnson et al. 2003) and investigation of wood characteristics of trees across a gradient of SNC intensity (Johnson et al. 2005) showed that trees with severe SNC had narrower sapwood, narrower growth rings, higher modulus of elasticity, higher wood density, greater proportion of latewood, narrower tracheid cell-wall thickness, and lower sapwood moisture content, including narrower tracheid cell-



Figure 3. Douglas-fir plantation showing visible symptoms of Swiss needle cast foliage disease. Note the contrast between the chlorotic (yellow) Douglas-fir trees and green Sitka spruce and western hemlock trees. The hardwoods are red alder.

wall thickness (Grotta et al. 2004). The lack of older foliage and an associated reduction in earlywood production drives the increase in wood density and stiffness.

The relationship between SNC-infected trees and Douglas-fir bark beetles (*Dendroctonus pseudotsugae*; Coleoptera: Curculionidae: Scolytinae) was investigated by measuring stem ethanol, monoterpene concentrations, oleoresin flow, and host selection (Kelsey and Manter 2004). As SNC severity increased, this was associated with reduced woody tissue ethanol, wound-induced resin flow, and reduced beetle attraction to the host. The authors concluded that SNC-infected trees were not being attacked by beetles because pioneering beetles fail to recognize them as stressed because of low ethanol production. Trees with severe SNC infection also appear to be unsuitable for successful beetle reproduction.

Biology, Ecology, and Epidemiology of *P. gaeumannii*

The biology, ecology, and epidemiology of *P. gaeumannii* have been a major focus of the SNCC because an understanding of these aspects of the disease was considered fundamental to possible amelioration (Hansen et al. 2000). Hansen et al. (2000) confirmed the causal agent and basic biology on the coast. Rosso and Hansen (2003) showed strong geographic patterns in disease related to elevation, aspect, distance from coast, and relation to the Sitka spruce/western hemlock

vegetation zone. Greenhouse studies of the pathogen led to disease severity models underscoring winter temperature as a key factor in disease expression (Manter et al. 2005). Multiple efforts to model both needle retention and disease severity are now suggesting that the combined influences of mild winter and spring temperatures and later spring/early summer leaf wetness (precipitation, fog impaction, and convergent marine air zones) drive the disease epidemiology. Because *P. gaeumannii* is ubiquitous across the Douglas-fir region, fluctuations in the disease are not associated with infection per se, but rather with an intensification of fungal activity that depends on climatic conditions that have interacted with the expansion of host (Douglas-fir) into climatically suitable areas.

SNC disease expression in the epidemic area of the Oregon Coast is distinctive from dominant foliage-disease paradigms (Hansen et al. 2000). Specifically, SNC symptom expression is greatest in the upper crown, and on south-facing aspects. The spores of fungi causing foliage diseases germinate and grow on the surface of foliage before entering the leaf and hence are very susceptible to desiccation. Normally, foliage diseases are most intense in the lower and inner parts of the crown and on north aspects, presumably because of the more moist microclimates in these locations. Vegetation management and thinning, both thought to improve airflow and decrease foliage disease by creating

a drier microclimate, are not effective in coastal Oregon presumably because coastal weather (fog and drizzle) maintains high leaf wetness regardless of stand structure. Temperature may also be an important factor because the upper crowns and south aspects where symptoms intensify are generally warmer.

Bud break occurs in early May for Douglas-fir, with branch elongation largely completed within 4–6 weeks. Needles are infected by *P. gaeumannii* almost exclusively in their 1st year; although some older foliage may be infected, this later infection is relatively minor in importance (Stone et al. 2008b). The fungus, an Ascomycete (Winton et al. 2007a), reproduces only sexually, and unlike other foliage fungi does not have asexual spores to aid in intensification. Spores are dispersed from small ascospore-producing fruiting bodies, called pseudothecia, which emerge from and plug the stomates in living foliage, dispersing spores in May, June, and July after pseudothecia maturation. Continuously wet needles during late spring and early summer, a common occurrence near the coastal fog zone, allow spores to successfully germinate on the needle surface and hyphae to grow into the needles through stomates. Warm winter temperatures probably allow the fungus to develop faster in the intercellular spaces of the needles and form pseudothecia sooner.

During favorable weather, fungal hyphae can also grow from pseudothecia, across the needle surface and back into other stomates, contributing to disease intensification (Stone et al. 2008b). The fungus reaches peak development just before bud break and the pseudothecia that emerge from stomates blocks gas exchange in the leaf. In the Cascade Mountains of Oregon and Washington, the pseudothecia can be commonly found on older senescing needles with an age range of 4–7 years, but in some areas of the Coast Range, pseudothecia are abundant on 1- and 2-year-old needles.

The primary pathological effect of the fungus appears to be plugging of the stomates by fruiting bodies, subsequent carbon starvation, and resulting premature shedding of foliage. Needle abscission generally occurs after 50% or more of the stomata are occluded (Hansen et al. 2000). Photosynthesis in 1st-year needles that have been infected by *P. gaeumannii* continues unhindered for up to 6 months; therefore, net carbon gains at the whole canopy level remain positive (Manter and Kavanagh 2003, Manter et al. 2003a). Excess absorbed light

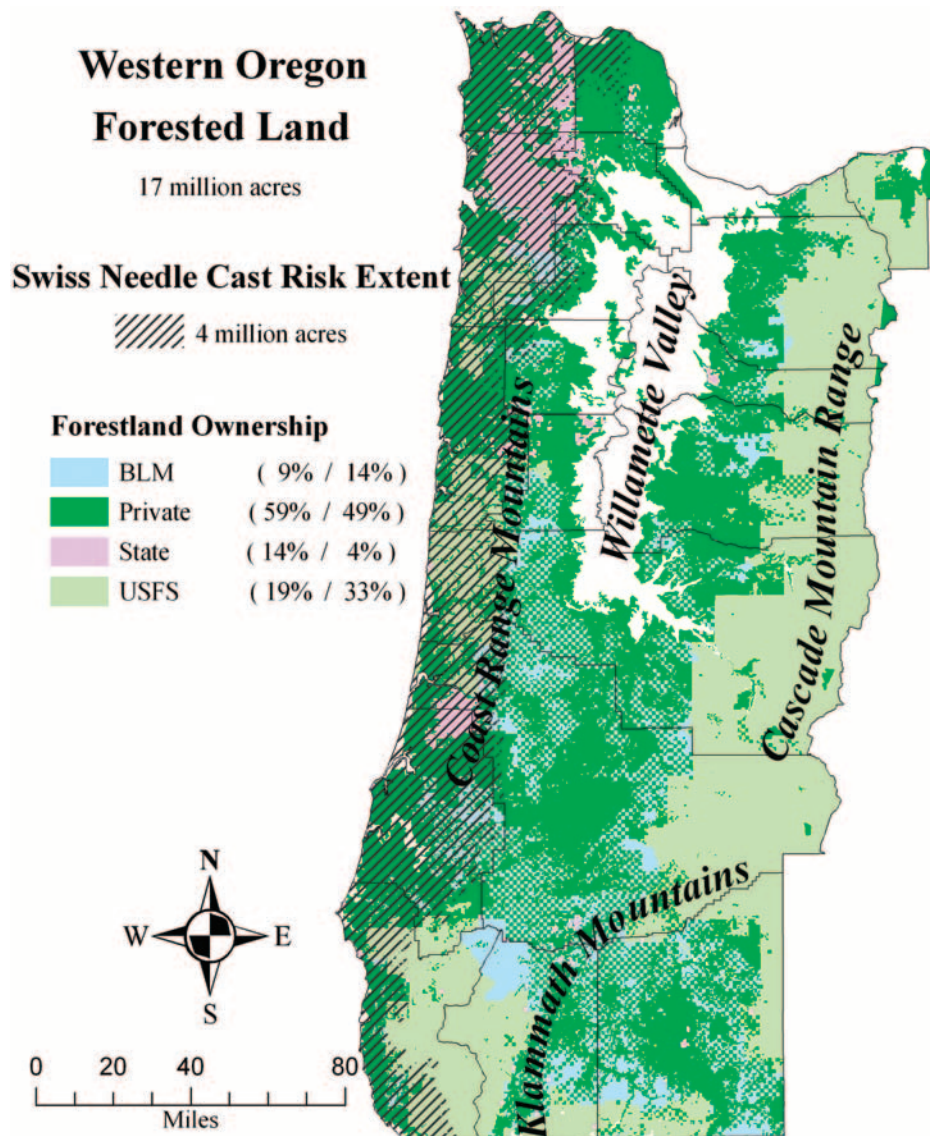


Figure 4. SNC risk area and forest ownership in western Oregon. The hatched area shows the region of the Coast Range that is threatened by the current Swiss needle cast epidemic. (Provided by Greg Latta, Oregon State University.)

in older infected needles combined with decreased photosynthetic capacity causes photooxidative damage that may also contribute to premature needle loss in sun-exposed foliage (Manter 2002); this response may explain the greater disease expression in upper crowns. The fungus appears to acquire nutrients from intercellular spaces of needles, but may also alter membrane permeability where hyphae appress host cells (Hansen et al. 2000, Stone et al. 2008b). Soil nutrient additions have revealed that *P. gaeumannii* positively responds to nutrient status in host trees and that increased N availability in Douglas-fir needles may be related to increased severity of SNC (El-Hajj et al. 2004).

The potential for genetic resistance and disease amelioration through tree breeding

was investigated early on after the disease was identified and confirmed (Kastner et al. 2001, Johnson 2002, Temel 2002, Temel et al. 2004, 2005). Research from British Columbia and northern Washington State indicated that there was variation in disease expression between the interior Rocky Mountain form of Douglas-fir (var. *glauca*) and the coastal form (var. *menziesii*) when trees were grown together in provenance trials. The coastal form was less impacted by disease in coastal areas compared with interior forms, presumably because the coastal form grows in wetter conditions and has adapted to more disease pressure in the past (Hood 1982). These observations supported hypotheses that the Oregon Coast Range epidemic was caused at least in part by planting

stock brought from outside the region. However, a 5-year study of the SNC effects on Douglas-fir from three different coastal seed sources at a high-hazard site near Tillamook, Oregon, found that all the trees grew poorly regardless of seed source (Kastner et al. 2001). The authors recommended that Douglas-fir should not be grown on sites along the north Oregon coast with intense disease pressure.

In other genetic work, Temel and others (Temel 2002, Temel et al. 2004, 2005) found that Douglas-fir has no apparent resistance to infection; i.e., all needles become infected, and that fungal biomass within the needle was similar between trees. However, disease expression and symptom development did vary by family, leading the authors to conclude that some genetic sources express disease tolerance. Some low-to-moderate genetic amelioration is apparently possible, and early testing for disease tolerance holds promise (Temel et al. 2005).

Winton et al. (2006, 2007a, 2007b) explored the population structure of *P. gaeumannii* by comparing multilocus-gene genealogies from samples across the epidemic area and from exotic plantations in the Northern and Southern Hemispheres. *P. gaeumannii* was concluded to have two sympatric, but isolated (noninterbreeding) lineages. One lineage is common throughout the world and the epidemic area, whereas the second lineage is only common in the epidemic area. It is not clear whether this second lineage or the combination of the two lineages is responsible for the current epidemic.

Effect of Silvicultural Treatments on Disease

Because of the SNCC, the effect of silvicultural treatments on disease are now better understood (Table 1). Early anecdotal evidence suggested that thinning exacerbated SNC. This possibility was a key concern because meeting silvicultural objectives on the Tillamook and Clatsop State Forests, in particular, have relied heavily on thinning. Experiments on vegetation management (Rose et al. 1999) and both commercial and precommercial thinning (Mainwaring et al. 2005, 2008) could not confirm any deleterious effects of these silvicultural treatments. This lack of any SNC response to thinning was consistent with results from studies in New Zealand (Hood and Kershaw 1975, Hood 1997).

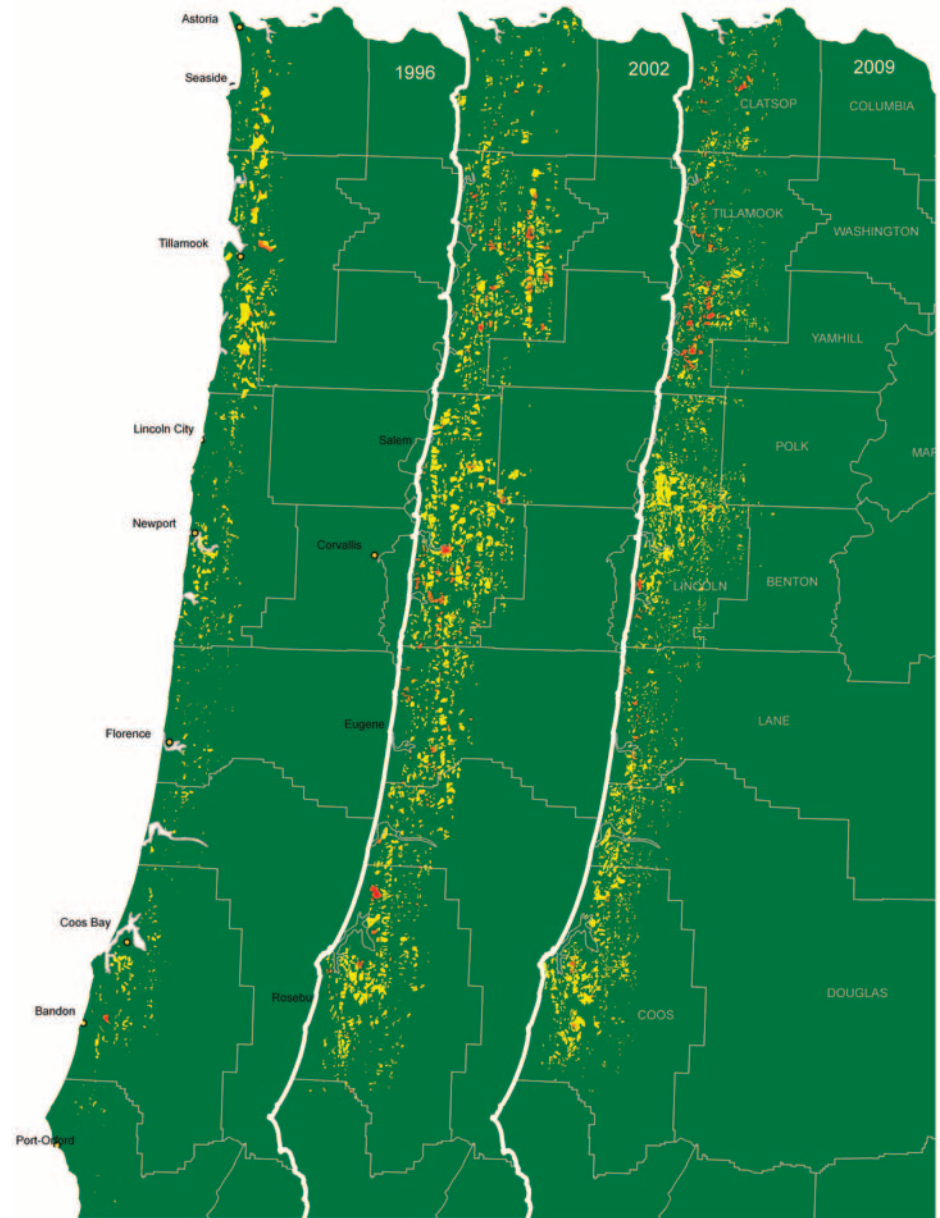


Figure 5. Swiss needle cast (SNC) aerial survey area with visible symptoms of SNC in Douglas-fir forests with detected in 1996 (the first aerial survey) in the left box and 2002 (the survey with the most mapped acres) in the middle box, and 2009 (the most recent aerial survey). Yellow indicates stands of Douglas-fir that show moderate symptoms of yellowing and crown thinning, while red indicates stands that appear severely affected.

Fungicides, including chlorothalonil and sulfur, are effective in preventing *P. gaeumannii* infection of current-year needles (Chastagner and Stone 2001), but annual applications of fungicide are required to consistently protect new foliage (Stone et al. 2007). The cost of annual operational spraying in managed forests is prohibitive, and potential environmental risks to both terrestrial and, especially, aquatic systems are high also.

Fertilization with nitrogen is not recommended in the SNC epidemic area. Nu-

trient availability varies from east to west across the Oregon Coast Range, with lower nitrogen and higher calcium availability east of the crest where SNC is not a factor and higher nitrogen and lower calcium availability closer to the coast where SNC is in epidemic (Perakis et al. 2005). Waring et al. (2000) found a strong negative correlation between increased foliar nitrogen concentration and foliage retention and proposed that excess nitrogen in the foliage may be stored in the form of soluble amino acids that promote the growth and development of *P.*

Area of Douglas-fir forest with Swiss needle cast symptoms detected by aerial surveys, Coast Range, Oregon, 1996-2010

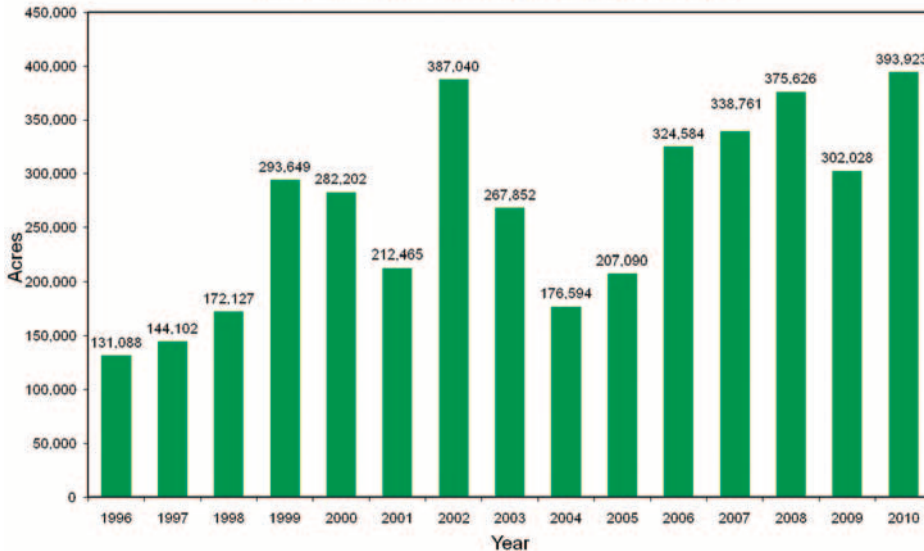


Figure 6. Trend in area (acres) of Douglas-fir forest in western Oregon with visible symptoms of Swiss needle cast detected during aerial surveys in April and May 1996–2010. (Provided by Kanaskie and McWilliams 2009.)

gaeumannii. Nitrogen fertilization of Douglas-fir has also been shown to increase N leaf concentration, *P. gaeumannii* biomass, and SNC severity in Idaho (El-Hajj et al. 2004). These observations have been the basis for avoiding nitrogen fertilization to ameliorate SNC, despite the prevalence of nitrogen amendment as a conventional management response to chlorotic foliage. Other nutritional amendments are currently being tested with respect to their potential for re-establishing a balance among nutrients, influencing disease severity and reversing growth impacts to Douglas-fir (Mainwaring et al. 2009).

Initial strategies for managing Douglas-fir in the presence of SNC focused on three general levels of SNC severity: low (needle retention, 2.6–3.5 years), moderate (needle retention, 1.6–2.5 years), and high (needle retention, 1.5 years); Filip et al. (2000). Within high-severity areas, it was recommended that Douglas-fir comprise less than 20% of planted seedlings, and in medium-severity areas less than 50% of planted seedlings. Recommendations for low-severity areas called for carefully restricting planted Douglas-fir seedlings to only those originating from only local Coast Range seed sources. These guidelines remain, although they can be modified by local knowledge of restricted microenvironments, local SNC severity, and new IPM approaches. The primary alternative species in this area is western hemlock, but its value is only about half

the value of Douglas-fir. Red alder is appropriate for some sites, and, currently, offer economic value equivalent to or exceeding Douglas-fir. Unfortunately, the feasibility of growing Sitka spruce is limited both by its lower value and the Sitka spruce weevil (white pine weevil, *Pissodes strobi*; Coleoptera: Curculionidae). Western redcedar is an economically attractive alternative, but it is heavily browsed by deer and elk, grows

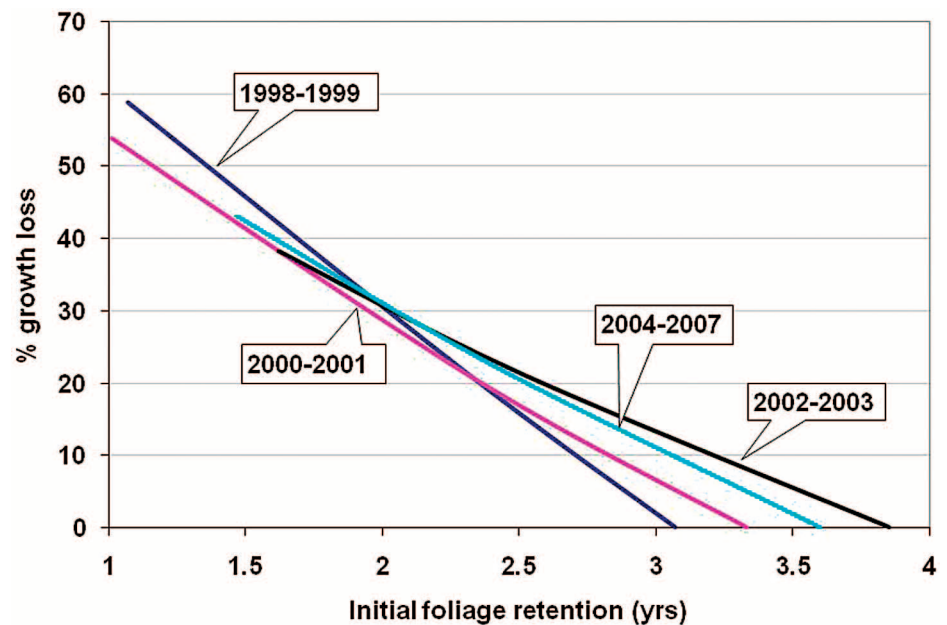


Figure 7. Implied relative growth losses for the four Growth Impact Study (GIS) growth periods. The GIS measures the effect of Swiss needle cast on growth of plantation Douglas-fir. Foliage retention varied at the start of each growth period. (Provided by Mainwaring et al. 2008.)

slower than Douglas-fir, and is easily wounded in thinning operations. Few tree improvement programs for these alternative species have been pursued to date.

Models to Predict Growth Impacts and Geographic Distribution of Disease Severity

Growth Impact Modeling

The severity of growth losses attributable to SNC is fundamental to understanding its economic impact and the viability of any management options. Hansen et al. (2000), Manter et al. (2001), and Winton et al. (2002, 2003) found a close correlation between needle retention, pseudothecia density, and fungal-biomass estimates. However, because needle retention (number of annual cohorts of needles) is the most practical index for assessing SNC severity in the field, Maguire et al. (2002) linked this index to growth impacts. Cubic volume growth losses ranged from approximately 50% in stands retaining only a single age class of needles to 0% in stands retaining at least 3½ years of needles. Diameter and height growth multipliers for an individual-tree growth model (ORGANON; Hann 2009) have also been developed as a function of needle retention (Garber et al. 2007), allowing landowners to estimate the impact of SNC on their projected timber inventory.

Disease Severity Models

Several modeling efforts have contributed to our ability to map the geographic variation in disease severity and hazard prediction. Rosso and Hansen (2003) were first to estimate the geographic nature of disease expression, with elevation and distance from the coast driving the estimates. Manter et al. (2003b, 2005) refined this approach after gaining additional insights into the epidemiology of SNC through greenhouse studies. Winter temperature in December, January, and February together with spring and summer precipitation were selected as key model inputs. Coop and Stone (2007) applied these relationships to create a web-accessible model of SNC severity for Oregon and Washington (Integrated Plant Protection Center 2010).

Latta et al. (2009) further developed a needle-retention model for estimating landscape-level growth losses. This model was based on field assessment of needle retention at many sites ranging from southern Oregon to southwestern Washington and within 20 mi of the coast. The economic impact of these growth losses is currently being assessed by considering the effects on timber inventories (through ORGANON growth multipliers), log markets, and milling infrastructure, with regional estimates based on US Forest Service Forest Inventory Analysis plots.

Climate Change Models

Because of the close association of weather variables to disease severity, the link between climate change and future disease is of significant interest (Stone et al. 2008a, Kliejunas et al. 2009). Stone and colleagues have begun using their disease severity models to predict how climate change will influence future disease severity. They contend that since 1970, winter temperature on the Oregon Coast has increased from 0.4 to 0.7°F (0.2 to 0.4°C) per decade, while spring precipitation has increased 0.3–0.6 in. (0.8–1.5 cm) per decade (Stone et al. 2008a). These trends in temperature and precipitation may have influenced the distribution and severity of SNC over the last 30 years. Continued warming during winter combined with continued or increasing precipitation during spring and summer will likely intensify SNC in the current high-risk zone and possibly allow it to spread to other areas where it is only endemic at present.

Table 1. Silvicultural techniques and their impacts on Swiss needle cast disease of Douglas-fir in the Oregon Coast Range.

Technique	Impact
Vegetation control	No impacts on disease; may improve tree growth
Fungicide application	Control of disease is effective, requires annual applications; environmental effects of fungicides too great for general forestry applications; not economical
Precommercial thinning	No impacts on disease; may improve individual tree growth
Commercial thinning	No impacts on disease; may improve individual tree growth
Fertilization	Unknown; excess N can increase disease severity; addition of Ca may balance excess N; S may act as fungicide on leaf surface
Pruning	Not studied but thought to reduce foliage in lower crown; because disease causes foliage biomass to shift to the lower crown, pruning is thought to have a negative impact on growth
Alternatives to Douglas-fir	Coast Range of Oregon has several alternatives to Douglas-fir, including red alder, western redcedar, western hemlock and Sitka spruce; each species has silvicultural difficulties, however (see section on effect of silviculture treatments on disease), and markets favor Douglas-fir.
Longer rotations	In areas of moderate to low needle cast severity, it may make sense to increase rotation length to accommodate growth losses that peaked when stands were 10–30 years old; This approach is untested and hypothetical

Table 2. Framework for an integrated pest management strategy for Swiss needle cast of Douglas-fir in the Oregon Coast Range.

Determining thresholds
Site and landscape risk assessment
Qualitative estimate of potential impacts
Aerial survey, Cascades Mountains plots
Disease severity and needle retention models
Stand impact assessment
Quantitative estimate of impacts
Needle retention estimates from the stand
ORGANON adjustment
Stand-growth assessment tool
Basal area growth from increment coring
Monitoring
Annual aerial survey for disease distribution and visual severity Oregon Coast Range
Cascades Mountains plots (disease assessment 5-yr remeasurement)
Long-term permanent plot growth impacts monitoring (Maguire et al. 2002)
Prevention/control
Silviculture
-Douglas-fir plantation management
Impacts of various silvicultural treatments on disease
Mixed species management
Alternative species management
Rotation length
Uneven-aged management
Implications for old-growth management on federal lands unknown
Long term
Maintenance of monitoring program (long-term monitoring of growth impacts)
Tree Improvement: PNW Tree Improvement Coop
Continued research
Economic and market models

IPM for SNC

Research funded at least in part by the SNCC has provided the framework for developing an IPM strategy for SNC in western Oregon (Table 2). An IPM strategy for SNC involves providing tools for landowners to determine economic and management activity thresholds, for monitoring distribution and impacts of disease, and for selecting silvicultural activities that prevent or ameliorate the disease. Thresholds are determined through qualitative risk assessment

and quantitative stand assessments. Monitoring is essential to an IPM strategy because disease distribution, disease severity, and tree growth impacts provide the basis for stand management. Current prevention and control of disease impacts applies silvicultural knowledge compiled from the latest research, while long-term strategies involve continuous monitoring of growth impacts; tree improvement through the Northwest Tree Improvement Cooperative (2010); continued research on the biology, ecology, and

epidemiology of *P. gaemannii*; and economic modeling and market simulation to inform landowners about future conditions.

Determining Thresholds

A qualitative assessment of risk is possible for a particular geographic site by evaluating the maps compiled from aerial surveys. These maps show the locations of forest stands with visible symptoms of SNC during spring surveys and are available at the Oregon Department of Forestry or US Forest Service Forest Health Protection website Oregon Department of Forestry 2010. If a stand is repeatedly mapped as discolored by aerial survey, SNC risk is high. Regional maps of needle retention (as a surrogate for SNC severity) are being refined by Coop and Stone (2007) and Latta et al. (2009). The former map is widely available at the Integrated Plant Protection Center (2010).

A landowner interested in assessing SNC severity can walk transects through the stand to estimate average foliage retention, preferably on branches in the midcrown and on the south side of the tree. Needle retention should be estimated on at least 4-year-old lateral branches off a main branch, rather than on the main axis of the branch. If average needle retention is 3 years or greater, then SNC should not be a serious concern.

For stands older than 10 years, a stand-growth assessment tool is available to compare current stand growth relative to its expectation in the absence of SNC (SNCC 2010). This assessment requires measurement of sample plots in the target stand, including estimation of needle retention and coring of trees to obtain past 5-year basal area growth. Various diagnostics are computed that allow the land manager to assess SNC growth impact and design a management strategy for minimizing losses. In general, we advise collecting stand-growth information because some stands can be growing relatively well even at low and moderate needle retention. For younger stands or for major landscape assessments, disease severity prediction models are the best available tools.

Given that winter, spring, and early summer temperature and precipitation/leaf wetness are the primary factors influencing disease, long-term climatic average facilitates prediction of regions with greatest SNC risk. However, landscape characteristics that influence marine air movement and onshore flow may cause divergent and convergent patterns of airflow. These conver-

gent zones may cause winds laden with drizzle and precipitation to pass over infected stands, pick up spores, and cause increased disease severity in downwind stands. These forces may help explain some of the unusual variation in disease impacts across the coastal landscape.

Monitoring

The SNCC and collaborators have developed a diverse monitoring system. The annual aerial survey provides the most extensive assessment of disease distribution and relative severity (Figures 5 and 6). The aerial survey in the Coast Ranges has been supplemented with a plot network across Douglas-fir plantations in the foothills of the Cascade Range (Filip et al. 2006). SNC is not currently causing significant growth losses in the Cascades, but this plot network will continue to be remeasured on a 5-year cycle. A network of growth impact plots likewise provides a long-term, ground-based monitoring system for SNC severity and influence on tree growth in the Coast Ranges (Maguire et al. 2002).

Prevention/Control: Silviculture

Prevention and control of SNC is currently achieved through adaptive silviculture. Depending on landowner objectives, this may include establishment and management of even-aged, short-rotation (approximately 45 years) Douglas-fir plantations on private land, but longer rotations on public lands. The USDI BLM, e.g., establishes rotation age based on culmination of mean annual increment, usually about 80 years depending on site. Although the US Forest Service may also manage even-aged stands on long rotations, uneven-aged systems are being explored as an alternative. Both the BLM and US Forest Service manage land within the SNC epidemic zone with the objective of maintaining late-successional reserves for spotted owl and other late-seral species. Potential complications from SNC for meeting these varied management objectives are still not clear. Strategies for dealing with SNC are therefore still evolving and will vary considerably among landowners.

The SNCC has devoted much research to understanding the influence of silvicultural techniques on disease severity and growth impacts in even-aged, Douglas-fir plantations (Table 1). Standard silvicultural operations do not seem to have a strong negative or positive influence on SNC; i.e., no interactions between SNC and silvicultural

treatments have been identified. However, the role of tree nutrition is not yet fully understood. Also, it has been shown that fungicides can prevent needle infection, reduce fungal growth, and limit SNC growth impacts, but widespread application of fungicides is not considered economically or environmentally feasible.

The SNCC has recommended mixed species plantations to minimize the economic risk but not necessarily to reduce disease severity associated with Douglas-fir. This recommendation includes a smaller proportion of Douglas-fir near the coast and a greater proportion as disease risk declines with distance from coast (Filip et al. 2000). Another option is to promote only alternative species immediately near the coast. In some areas, it may be appropriate to grow pure, even-aged stands of alder, western hemlock, Sitka spruce, or western redcedar, despite the disadvantages mentioned previously.

Increasing rotation length may be an attractive option in some situations, but it is not clear whether disease severity or growth impacts are any less or possibly greater in older stands. Most growth impact and epidemiology research has targeted stands between 10 and 60 years of age. The younger end of this range received the most early attention because it was possible that some of the severely impacted stands would not reach commercial size, implying a very significant economic loss if the plantations required total rehabilitation and no timber revenue. One of the 80+-year-old stands that Black et al. (2010) investigated was severely infected, indicating that age may not be a factor in tree susceptibility to SNC.

Uneven age management generally shifts species composition from Douglas-fir to more western hemlock, but mixed species composition does not seem to protect Douglas-fir trees from infection. Old, large Douglas-fir trees are important compositional and structural elements of late-successional reserves, and we currently do not know how SNC may challenge our ability to maintain or produce old Douglas-fir trees where this is a management objective.

Long-Term Planning and Management

The long-term management of SNC requires maintenance of the monitoring program, especially the long-term monitoring of SNC symptoms and growth impacts on permanent plots. Continued research in the biology, ecology, epidemiology, and amelioration of the disease is important, as is

economic and market modeling and analysis. Temel et al. (2004, 2005) initiated the activities of Douglas-fir tree improvement programs within the Northwest Tree Improvement Cooperative. Two progeny trials in the epidemic area (South Central Coast Tree Improvement Cooperative and Trask Tree Improvement Cooperative) have incorporated knowledge of SNC in their family selections (Shaw et al. 2007). Moderate genetic gains may be possible, but Kastner et al. (2001) emphasized that Douglas-fir should simply not be grown in the highest risk areas.

Evidence suggests that the SNC epidemic is tied to annual fluctuations in temperature and precipitation, so climate change may potentially have a profound influence on temporal and spatial trends in the disease. With warming temperatures and increasing precipitation, SNC could both expand and intensify (Stone et al. 2008a). Alternatively, a general drying trend or shift away from summer precipitation on the coast could dampen the disease. Climate change modeling will likely be important for future economic assessments and forest management strategies.

Conclusions

The SNCC is a unique applied research cooperative housed at the College of Forestry, OSU. This cooperative has been central to elucidating the dynamics and features of SNC, including growth impacts and the scientific basis for an IPM approach to the epidemic. SNC is not abating and could be a potential threat to the Douglas-fir in at least parts of the Coast Ranges. This extensive foliage disease impacts previously productive Douglas-fir forests and hovers on the margins of one of the world's most productive forest types. SNC intensity continues to fluctuate annually within the epidemic area covering about 4 million ac in Oregon. The disease appears linked to climatic changes such as warming temperatures in winter and increasing spring and summer leaf wetness. Other contributing factors probably include a greater abundance of suitable Douglas-fir substrate in the fog belt, high nitrogen and low calcium availability possibly related to previous occupation by red alder, and an increasing source of fungal inocula as the disease has intensified. The SNCC has been central to exploring potential silvicultural strategies for ameliorating the disease and for conducting research on growth impacts and economic implications. Landscape dis-

ease severity models based on needle retention as an index of potential risk are aiding landowners in developing management strategies and estimating regional disease impacts. IPM of SNC, including tree improvement and long-term monitoring, is fundamental to the future success of growing Douglas-fir in the SNC epidemic zone.

Literature Cited

- BLACK, B.A., D.C. SHAW, AND J.K. STONE. 2010. Impacts of Swiss needle cast on overstory Douglas-fir forests of western Oregon Coast Range. *For. Ecol. Manag.* 259:1673–1680.
- BOYCE, J.S. 1940. A needle-cast of Douglas-fir associated with *Adelopus gaeumannii*. *Phytopathology* 30:649–659.
- CHASTAGNER, G., AND J. STONE. 2001. Fungicidal control of Swiss needle cast in stands of Douglas-fir timber, P. 89–95 in *Swiss needle cast cooperative annual report 2001*, Filip, G. (ed.). College of Forestry, Oregon State Univ., Corvallis, OR. Available online at www.cof.orst.edu/coops/sncc/index.htm; last accessed Feb. 4, 2010.
- COOP, L., AND J. STONE. 2007. Prediction maps of Swiss needle cast needle retention based on climatic factors. P. 15–21 in *Swiss needle cast cooperative annual report 2007*, Shaw, D. (ed.). College of Forestry, Oregon State Univ., Corvallis, OR. Available online at www.cof.orst.edu/coops/sncc/index.htm; last accessed Feb. 4, 2010.
- EL-HAJJ, Z., K. KAVANAGH, C. ROSE, AND Z. KANAAN-ATALLAH. 2004. Nitrogen and carbon dynamics of a foliar biotrophic fungal parasite in fertilized Douglas-fir. *New Phytol.* 163: 139–147.
- FILIP, G., A. KANASKIE, K. KAVANAGH, G. JOHNSON, R. JOHNSON, AND D. MAGUIRE. 2000. *Silviculture and Swiss needle cast: Research and recommendations*. Research Contribution 30, Forest Research Laboratory, College of Forestry, Oregon State Univ., Corvallis, OR. Available online at www.cof.orst.edu/coops/sncc/index.htm; last accessed Feb. 4, 2010.
- FILIP, G., A. KANASKIE, W. LITTKER, J. BROWNING, D. HILDEBRAND, AND D. MAGUIRE. 2006. Impacts of Swiss needle cast on Douglas-fir in the Cascade foothills of Northern Oregon after five years. P. 12–19 in *Swiss needle cast cooperative annual report 2006*, Shaw, D. (ed.). College of Forestry, Oregon State Univ., Corvallis, OR. Available online at www.cof.orst.edu/coops/sncc/index.htm; last accessed Feb. 4, 2010.
- FRANKLIN, J.F., AND C.T. DYRNESS. 1973. *Natural vegetation of Oregon and Washington*. US For. Serv. Gen. Tech. Rep. PNW-8. 417 p.
- GARBER, S., D. MAGUIRE, D. MAINWARING, AND D. HANN. 2007. Swiss needle cast ORGANON module update. P. 63–66 in *Swiss needle cast cooperative annual report 2007*, Shaw, D. (ed.). College of Forestry, Oregon State Univ., Corvallis, OR. Available online at www.cof.orst.edu/coops/sncc/index.htm; last accessed Feb. 4, 2010.
- GROTTA, A.T., R.J. LEICHTI, B.L. GARTNER, AND G.R. JOHNSON. 2004. Effect of growth ring orientation and placement of earlywood and latewood on MOE and MOR of very-small clear Douglas-fir beams. *Wood Fiber Sci.* 37: 207–212.
- HANN, D.W. 2009. *ORGANON user's manual*, Edition 8.4 (computer manual). Oregon State Univ., Department of Forest Resources, Corvallis, OR. 129 p.
- HANSEN, E.M., J.K. STONE, B.R. CAPITANO, P. ROSSO, W. SUTTON, L. WINTON, A. KANASKIE, AND M.G. MCWILLIAMS. 2000. Incidence and impact of Swiss needle cast in forest plantations of Douglas-fir in coastal Oregon. *Plant Dis.* 84:773–778.
- HOOD, I.A. 1982. Phaeocryptopus gaeumannii on *Pseudotsuga menziesii* in southern British Columbia. *N.Z. J. For. Sci.* 12:415–424.
- HOOD, I. 1997. Swiss needle cast. P. 55–56 in *Compendium of conifer diseases*, Hansen, E.M., and K.J. Lewis (eds.). APS Press, St. Paul, MN.
- HOOD, I.A., AND D.J. KERSHAW. 1975. Distribution and infection period of *Phaeocryptopus gaeumannii* in New Zealand. *N. Z. J. For. Sci.* 5:201–208.
- INTEGRATED PLANT PROTECTION CENTER (IPCC). 2010. Available online at www.uspest.org/sncc/; last accessed Oct. 15, 2010.
- JOHNSON, G.R. 2002. Genetic variation in tolerance of Douglas-fir to Swiss needle cast as assessed by symptom expression. *Silvae Genet.* 51:80–86.
- JOHNSON, G.R., B.L. GARTNER, D. MAGUIRE, AND A. KANASKIE. 2003. Influence of Bravo fungicide applications on wood density and moisture content of Swiss needle cast affected Douglas-fir trees. *For. Ecol. Manag.* 186:339–348.
- JOHNSON, G.R., A.T. GROTTA, B.L. GARTNER, AND G. DOWNES. 2005. Impact of the foliar pathogen Swiss needle cast on wood quality of Douglas-fir. *Can. J. For. Res.* 35:331–339.
- KANASKIE, A., AND M. MCWILLIAMS. 2009. Swiss needle cast aerial survey. P. 9–12 in *Swiss needle cast cooperative annual report 2009*, Shaw, D., and T. Woolley (eds.). College of Forestry, Oregon State Univ., Corvallis, OR. Available online at www.cof.orst.edu/coops/sncc/index.htm; last accessed Feb. 4, 2010. Aerial Survey data available online at www.oregon.gov/ODF/privateforests/fhMaps.shtml; last accessed Feb. 4, 2010.
- KASTNER, W., S. DUTTON, AND D. ROCHE. 2001. Effects of Swiss needle cast on three Douglas-fir seed sources on a low-elevation site in the northern Oregon Coast Range: Results after five growing seasons. *West. J. Appl. For.* 16:31–34.
- KELSEY, R.G., AND D.K. MANTER. 2004. Effect of Swiss needle cast on Douglas-fir stem ethanol and monoterpene concentrations, oleoresin flow, and host selection by the Douglas-fir beetle. *For. Ecol. Manag.* 190:241–253.
- KLIEJUNAS, J.T., B.W. GEILS, J.M. GLAESER, E.M. GOHEEN, P. HENNON, M. KIM, H. KOPE, J. STONE, R. STURROCK, AND S.J. FRANKEL. 2009. *Review of the literature on climate change and forest diseases of western North America*. US For. Serv. Gen. Tech. Rep. PSW-GTR-225. 54 p.
- LATTA, G., D. ADAMS, AND D. SHAW. 2009. Map-

- ping western Oregon Douglas-fir foliage retention with a simultaneous autoregressive model. P. 37–51 in *Swiss needle cast cooperative annual report 2009*, Shaw, D., and T. Woolley (eds.). College of Forestry, Oregon State Univ., Corvallis, OR. Available online at www.cof.orst.edu/coops/sncc/index.htm; last accessed Feb. 4, 2010.
- MAGUIRE, D.A., AND A. KANASKIE. 2002. The ratio of live crown length to sapwood area as a measure of crown sparseness. *For. Sci.* 48(1):93–100.
- MAGUIRE, D.A., A. KANASKIE, W. VOELKER, R. JOHNSON, AND G. JOHNSON. 2002. Growth of young Douglas-fir plantations across a gradient in Swiss needle cast severity. *West. J. Appl. For.* 17:86–95.
- MAINWARING, D.B., D.A. MAGUIRE, A. KANASKIE, AND J. BRANDT. 2005. Growth responses to commercial thinning in Douglas-fir stands with varying intensity of Swiss needle cast. *Can. J. For. Res.* 35:2394–2402.
- MAINWARING, D., D. MAGUIRE, AND A. KANASKIE. 2008. Growth impact study: Growth trends during the fourth period following establishment of permanent plots. P. 13–17 in *Swiss needle cast cooperative annual report 2008*, Shaw, D., and T. Woolley (eds.). College of Forestry, Oregon State Univ., Corvallis, OR. Available online at www.cof.orst.edu/coops/sncc/index.htm; last accessed Feb. 4, 2010.
- MAINWARING, D., D. MAGUIRE, AND S. PERAKIS. 2009. Three year response of young Douglas-fir to fertilization treatments on the beyond nitrogen plots. P. 52–62 in *Swiss needle cast cooperative annual report 2009*, Shaw, D., and T. Woolley (eds.). College of Forestry, Oregon State Univ., Corvallis, OR. Available online at www.cof.orst.edu/coops/sncc/index.htm; last accessed Feb. 4, 2010.
- MANTER, D.K. 2002. Energy dissipation and photoinhibition in Douglas-fir needles with a fungal-mediated reduction in photosynthetic rates. *J. Phytopathol.* 150:674–679.
- MANTER, D.K., B.J. BOND, K.L. KAVANAGH, P.H. ROSSO, AND G.M. FILIP. 2000. Pseudothecia of Swiss needle cast fungus, *Phaeocryptopus gaeumannii*, physically block stomata of Douglas fir, reducing CO₂ assimilation. *New Phytol.* 148:481–491.
- MANTER, D.K., R.G. KELSEY, AND J.K. STONE. 2001. Quantification of *Phaeocryptopus gaeumannii* colonization in Douglas-fir needles by ergosterol analysis. *For. Pathol.* 31:229–240.
- MANTER, D.K., AND K.L. KAVANAGH. 2003. Stomatal regulation in Douglas-fir following a fungal-mediated chronic reduction in leaf area. *Trees Struct. Funct.* 17:485–491.
- MANTER, D.K., B.J. BOND, K.L. KAVANAGH, J.K. STONE, AND G.M. FILIP. 2003a. Modelling the impacts of the foliar pathogen, *Phaeocryptopus gaeumannii*, on Douglas-fir physiology: Net canopy carbon assimilation, needle abscission and growth. *Ecol. Model.* 164:211–226.
- MANTER, D.K., L.M. WINTON, G.M. FILIP, AND J.K. STONE. 2003b. Assessment of Swiss needle cast disease: Temporal and spatial investigations of fungal colonization and symptom severity. *J. Phytopathol.* 151:344–351.
- MANTER, D.K., P.W. REESER, AND J.K. STONE. 2005. A climate-based model for predicting geographic variation in Swiss needle cast severity in the Oregon coast range. *Phytopathology* 95:1256–1265.
- MICHAELS, E., AND G.A. CHASTAGNER. 1984. Distribution, severity and impact of Swiss needle cast in Douglas-fir Christmas trees in western Washington and Oregon. *Plant Dis.* 68:939–942.
- NORTHWEST TREE IMPROVEMENT COOPERATIVE. 2010. Available online at www.fsl.orst.edu/nwtic/; last accessed Oct. 15, 2010.
- OMDAL, D., J. MOORE, AND C. BURNS. 2001. Three years of Swiss needle cast aerial survey in Washington state: 1998–2000. *Poster presentation at the Forest Health Monitoring Program Work Group Workshop*, Las Vegas, NV, Feb. 5–8, 2001.
- OMDAL, D., AND A. RAMSEY-KROLL. 2010. *Swiss needle cast on Washington state lands, 1999–2009*. Forest Health Note, 2010-001, Washington Department of Natural Resources, Olympia, WA. 4 p.
- OREGON DEPARTMENT OF FORESTRY. 2010. Available online at www.oregon.gov/ODF/privateforests/fhMaps.shtml; last accessed Oct. 15, 2010.
- PEACE, T.R. 1962. *Pathology of trees and shrubs*. Oxford University Press, New York. 723 p.
- PERAKIS, S.S., D.A. MAGUIRE, T.D. BULLEN, K. CROMACK, R.H. WARING, AND J. BOYLE. 2005. Coupled nitrogen and calcium cycles in forests of the Oregon Coast Range. *Ecosystems* 9:63–74.
- ROSE, R., S. KETCHUM, AND D. HASSE. 1999. Effect of fertilization and vegetation control on Swiss needle cast infection and growth of coastal Douglas-fir. P. 39–43 in *Swiss needle cast cooperative annual report 1999*, Filip, G. (ed.). College of Forestry, Oregon State Univ., Corvallis, OR. Available online at www.cof.orst.edu/coops/sncc/index.htm; last accessed Feb. 4, 2010.
- ROSSO, P.H., AND E.M. HANSEN. 2003. Predicting Swiss needle cast disease distribution and severity in young Douglas-fir plantations in coastal Oregon. *Phytopathology* 93:790–798.
- SHAW, D., A. KANASKIE, K.J. AYAWICKRAMA, T. YE, S. LIPOW, D. WALTERS, R. JOHNSON, D. MAGUIRE, AND J. STONE. 2007. Evaluation of Swiss needle cast effects on young Douglas-fir plantations: Measurement techniques, severity indices, growth, and genetic effects. P. 61–62 in *Swiss needle cast cooperative annual report 2007*, Shaw, D. (ed.). College of Forestry, Oregon State Univ., Corvallis, OR. Available online at www.cof.orst.edu/coops/sncc/index.htm; last accessed Feb. 4, 2010.
- STONE, J.K., P.W. REESER, AND A. KANASKIE. 2007. Fungicidal suppression of Swiss needle cast and pathogen reinvasion in a 20-year-old Douglas-fir stand. *West. J. Appl. For.* 22:248–252.
- STONE, J.K., L.B. COOP, AND D.K. MANTER. 2008a. Predicting the effects of climate change on Swiss needle cast disease severity in Pacific Northwest forests. *Can. J. Plant Pathol.* 30:169–176.
- STONE, J.K., B.R. CAPITANO, AND J.L. KERRIGAN. 2008b. The histopathology of *Phaeocryptopus gaeumannii* on Douglas-fir needles. *Mycologia* 100:431–444.
- SWISS NEEDLE CAST COOPERATIVE (SNCC). Available online at www.cof.orst.edu/coops/sncc/; last accessed Oct. 15, 2010.
- TEMEL, F. 2002. *Early testing of Douglas-fir for Swiss needle cast tolerance*. PhD thesis, Oregon State Univ., Corvallis, OR. 177 p.
- TEMEL, F., G.R. JOHNSON, AND J.K. STONE. 2004. The relationship between Swiss needle cast symptom severity and level of *Phaeocryptopus gaeumannii* colonization in coastal Douglas-fir (*Pseudotsuga menziesii* var. *menziesii*). *For. Pathol.* 34:383–394.
- TEMEL, F., G.R. JOHNSON, AND W.T. ADAMS. 2005. Early genetic testing of coastal Douglas-fir for Swiss needle cast tolerance. *Can. J. For. Res.* 35:521–529.
- THIES, W.G., AND E.M. GOHEEN. 2002. Major forest diseases of the Oregon Coast Range and their management. P. 191–211 in *Forest and stream management in the Oregon Coast Range*, Hobbs, S.D., J.P. Hayes, R.L. Johnson, G.H. Reeves, T.A. Spies, J.C. Tappeiner II, and G.E. Wells (eds.). Oregon State University Press, Corvallis, OR.
- WARING, R.H., J. BOYLE, K. CROMACK, JR., D. MAGUIRE, AND A. KANASKIE. 2000. Researchers offer new insights into Swiss needle cast. *West. For.* 45(6):10–11.
- WEISKITTEL, A.R., D.A. MAGUIRE, S.M. GARBER, AND A. KANASKIE. 2006. Influence of Swiss needle cast on foliage age-class structure and vertical foliage distribution in Douglas-fir plantations in north coastal Oregon. *Can. J. For. Res.* 36:1497–1508.
- WEISKITTEL, A.R., AND D.A. MAGUIRE. 2007. Response of Douglas-fir leaf area index and litterfall dynamics to Swiss needle cast in north coastal Oregon, USA. *Ann. For. Sci.* 64:121–132.
- WEISKITTEL, A.R., D.A. MAGUIRE, AND R.A. MONSERUD. 2007. Modeling crown structural responses to competing vegetation control, thinning, fertilization, and Swiss needle cast in coastal Douglas-fir of the Pacific Northwest, USA. *For. Ecol. Manag.* 245:96–109.
- WINTON, L.M., J.K. STONE, S. WATRUD, AND E.M. HANSEN. 2002. Simultaneous one-tube quantification of host and pathogen DNA with real-time polymerase chain reaction. *Phytopathology* 92:112–116.
- WINTON, L.M., D.K. MANTER, J.K. STONE, AND E.M. HANSEN. 2003. Comparison of biochemical, molecular, and visual methods to quantify *Phaeocryptopus gaeumannii* in Douglas-fir foliage. *Phytopathology* 93:121–126.
- WINTON, L.M., E.M. HANSEN, AND J.K. STONE. 2006. Population structure suggests reproductively isolated lineages of *Phaeocryptopus gaeumannii*. *Mycologia* 98:781–791.
- WINTON, L.M., J.K. STONE, AND E.M. HANSEN. 2007a. The systematic position of *Phaeocryptopus gaeumannii*. *Mycologia* 99:240–252.
- WINTON, L.M., J.K. STONE, AND E.M. HANSEN. 2007b. Polymorphic microsatellite markers for the Douglas-fir pathogen, *Phaeocryptopus gaeumannii*, causal agent of Swiss needle cast disease. *Mol. Ecol.* 7:1125–1128.