INCIDENCE AND SEVERITY OF SWISS NEEDLE CAST IN THE COASTAL FOREST OF WESTERN WASHINGTON: EXAMINING CLIMATIC CORRELATION

by

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ABSTRACT

Incidence and Severity of Swiss Needle Cast in the Coast Forest of Western Washington: Examining Climatic Correlation

Michele Boderck

The Pacific Northwest (PNW) is experiencing an epidemic of Swiss needle cast (SNC), a foliage disease that affects Douglas-fir exclusively and is caused by the fungus, *Phaeocryptopus gaeumannii*. Symptoms include chlorotic (yellow) needles, decreased foliage retention, and reductions in growth. Although tree mortality is rare, because there are few tools to combat SNC other than tree species substitution, the epidemic is a major concern for the region's timber industry, economy, and general forest health. SNC is native to the PNW, but emerged as an epidemic in coastal Washington and Oregon in the 1970s and has steadily increased since then. The causes are not entirely understood, however, the extensive planting of Douglas-fir stands in the coastal forest where this species is naturally rare, as well as the warm, wet climate of the coast that is conducive to fungal growth, are likely contributing factors. This research sought to answer the questions of: What is the incidence and severity of SNC in western Washington? What climate variables are most correlated with its growth? Needle samples were collected in the spring of 2015 from 47 sites throughout western Washington and examined for pseudothecia (fungal fruiting body) abundance. Results indicated a colonization index of 5.3 for one-year-old needles and 22.5 for two-year-old needles. This demonstrates an increase in disease severity from ground surveys conducted in 2011 and 2012. Additionally, correlations between colonization indices and various climate variables calculated with Climate WNA software at the monthly, seasonal, and annual scale were examined using a linear regression analysis. No correlation between climate and disease severity was found in this study, however, literature suggests that climate is playing some role in disease severity: other factors may be making the relationship less clear in this study. Due to the effects that growth loss from SNC can have on the economy of Washington State and forest health in general, further research into the cause of SNC spread and how climate conditions affect the disease are recommended.

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CHAPTER I: Literature Review

Introduction

Swiss needle cast (SNC) is a foliage disease caused by the pathogenic fungus, *Phaeocryptopus gaeumannii* that exclusively infects Douglas-fir (*Pseudotsuga menziesii*). While SNC is not usually directly linked to tree mortality, symptoms include chlorosis (yellowing of foliage), decreased needle retention, and most importantly, above- and below-ground growth loss. The fungus is believed to be endemic to the Pacific Northwest (PNW) and ubiquitous across the native range of Douglas-fir in western North America (Boyce 1940) but for reasons not entirely clear, an epidemic has emerged over the last thirty years on the outer coast of Washington and Oregon. Considering the importance of the timber industry, and thus Douglas-fir, to the Pacific Northwest, this disease has turned into an important coastal forest health issue.

This literature review will discuss the importance of Douglas-fir to the Pacific Northwest timber industry and thus Washington State's economy. An explanation of the SNC disease will follow, covering the life cycle of the causal fungus, its effects on Douglas-fir, and the spread and possible causes of the current epidemic. Current research into attempts to minimize disease impacts, methods for monitoring, and favorable environmental conditions is reviewed. The lack of an environmental analysis in Washington is identified as a key gap in the literature, prompting this study. Due to the nature of the disease varying geographically, learning which conditions are most correlated with disease severity in western Washington will advance our understanding of SNC and potentially enable us to inform landowners of high-risk areas.

Significance of Douglas-fir in the Pacific Northwest

The forests of Washington are a prominent part of the state's history, shaping its economic development and identity since the 1850s. Washington, known as the Evergreen State, has relied on the logging industry since its creation, but the 1994 Northwest Forest Plan dramatically altered federal land management, reducing timber harvest by 80% on federal land to provide protection for riparian areas and late successional reserves for species associated with old-growth ecosystems such as the northern spotted owl (*Strix occidentalis caurina*) (Charnley et al. 2006). As a result, there is increased pressure on non-federal (private, state, tribal, and county/municipal) timberland to supply 95% of the demand (Smith 2015). In western Washington, the average annual harvest level across all management types dropped 45% from pre-1990 to 1998-2002 (Partridge and MacGregor 2007). In Washington State there are 21 million acres of forest, of which 8.9 million acres (42%) are working forests grown for timber production (Lippke et al. 2007). In 2014, over 3 billion board feet of timber was harvested in Washington (Smith 2015). Approximately 22% of Washington's timber supply is exported to international markets (Partridge and MacGregor 2007).

The economy of Washington, particularly rural communities, depends on these timber plantations, dating back to the settlement of the region that was made possible by timber harvest. Washington's forest industry is the second largest in the nation (behind Oregon) and drives economic activity including employment, wages, and revenue for both state and local governments. The total direct, indirect and induced impact of forestry-related industries included 105,000 jobs paying $4.9 billion in wages, gross business income totaling nearly $28 billion, and $175 million in taxes paid in 2013

(Hatfield 2013). In 2015, more than $37 million in forest excise taxes were distributed to counties, libraries, local schools, and taxing districts (Department of Revenue 2015). Forest products is the third largest manufacturing sector in the state (Hatfield 2013). An analysis of the economic impacts of SNC could not be found in Washington, but in Oregon forests there is an impact of more than 2,100 jobs, representing $117 million in labor income, $10 million in income tax, and $700,000 in harvest tax, for a total of $128 million per year lost due to the disease (Kanaskie et al. 2014). With a $12.7 billion forest sector contribution to Oregon's economy each year, this represents a loss of 4.7% (Oregon Forest Resources Institute 2015).

Approximately 42% (11.6 billion cubic feet) of non-federal timberland in Washington State is composed of Douglas-fir (Gray et al. 2005), underlining the importance of this species. Waring and Franklin (1979) found that coastal stands of Douglas-fir are among the most productive native conifer forests in the world due to a long growing season, abundant nitrogen, high precipitation, and relatively warm winter temperatures. Even with growth losses from SNC, Douglas-fir is still financially competitive with other local conifer species. Western hemlock (*Tsuga heterophylla*) is the primary alternative species in the area but is not as valuable as Douglas-fir, due to market demands for quality structural lumber. Washington Department of Natural Resources' reported log prices in October 2015 in the Coast marketing area at $551/MBF (thousand board feet) for Douglas-fir, compared to $449 for western hemlock (Smith 2015). Other alternative species that are worth more than Douglas-fir have their own drawbacks: red alder is only suitable to grow in riparian areas and western redcedar,

while highly valuable, is browsed heavily by deer and elk as a sapling, grows slowly, and is easily wounded during thinning operations (Shaw et al. 2011).

What is Swiss needle cast?

The life cycle of P. gaeumannii

Phaeocryptopus gaeumannii is the microfungus that causes the SNC disease. Numerous researchers have described the life cycle of *P. gaeumannii* since its discovery, which is an important first step in the field of pathology (Boyce 1940, Chastagner and Byther 1983, Chen 1972, Ford and Morton 1971, Hood and Kershaw 1975, Hood 1982, Michaels and Chastagner 1984a, Rhode 1937). Manter et al. (2005) identified three key seasons in the life cycle of *P. gaeumannii*: May-June is when spore dispersion, deposition, germination, and initial infection occur; August-October is when fungal development occurs within infected needles; and December-February is the period critical to the rate of pseudothecia (fungal fruiting body) growth.

In the spring, spores of the fungus are dispersed by windblown rain and infect newly emerging Douglas-fir needles. The ascospores tend to be released at temperatures between 5 and 30°C and in greatest abundance at 20°C (Michaels and Chastagner 1984a). This is not a one-time occurrence, but occurs continuously throughout spring and summer (Temel et al. 2004). The release coincides with bud break and shoot elongation of Douglas-fir and only newly emerged needles can be infected (Stone et al. 2008b, Hansen et al. 2000). Harrington and Gould (2010) predicted that budburst will occur earlier as temperatures warm with climate change, which depending on changes in the seasonality of precipitation, could influence SNC infection behavior. The spore germinates on the needle surface until it finds a suitable stomata (air pore) to grow into. Infection through

stomata is the exclusive method of *P. gaeumannii* infection (Stone et al. 2008b). Throughout the summer, the fungus colonizes the intercellular spaces of the needles (Capitano 1999). The fungus does not penetrate cells but grows within the mesophyll layer and on the leaf surface, absorbing water and nutrients, such as nitrogen and carbon, from the needles' intercellular spaces (Capitano 1999). Between October and February, pseudothecia form in the epistomatal chamber and can be seen as tiny black spheres in the rows of stomata (Stone and Carroll 1985) (Fig. 1). The pseudothecia gradually mature until May to June, when the ascospores are released.

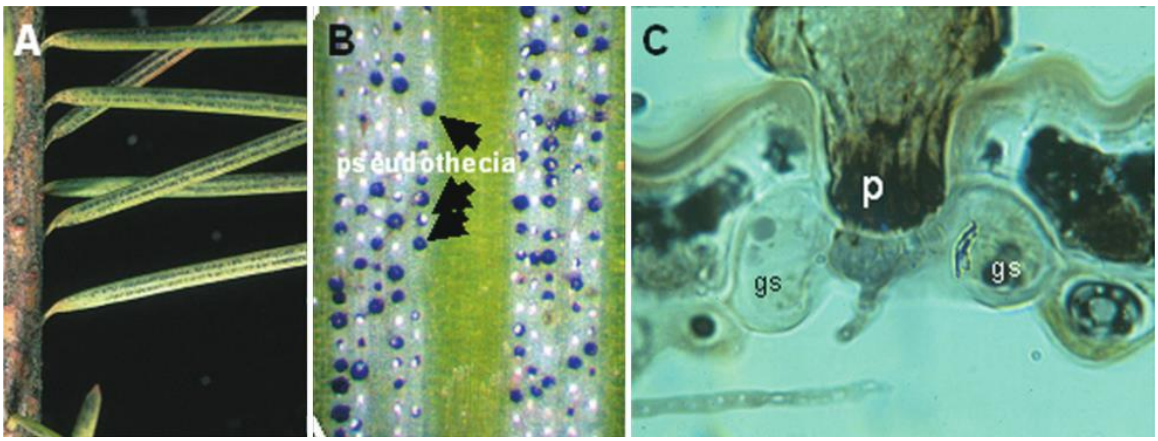


Figure 1. (A) Pseudothecia visible as black dots on the underside of Douglas-fir needles. (B) Rows of stomata with pseudothecia. (C) Cross-section through a stoma showing pseudothecium (p) between the guard cells (gs). From Stone et al. 2008b.

As the needles age, hyphae grow from the pseudothecia across needle surfaces in dense mats and continue to infect more stomata (Capitano 1999). Thus, 2-year-old (and older) needles typically have increased levels of fungal colonization (Maguire et al. 2002, Michaels and Chastagner 1984a, Temel et al. 2004, Winton et al. 2003). Epiphytic

colonization in an environment with year-round leaf moisture, such as the PNW, means that the disease can progress cumulatively over time (Capitano 1999). After several years of favorable weather conditions, the entire tree crown can be heavily infected with *P. gaeumannii*.

Effects on Douglas-fir

There is a strong physiological basis that links *P. gaeumannii* with disease severity (Watt et al. 2010). The fungus interferes with key life cycle phases of Douglas-fir, but does not kill the plant cells directly. The pseudothecia of the fungus occlude the needles' stomata, impairing gas exchange and photosynthesis, and is thus considered the primary mechanism of pathogenicity (Manter et al. 2000, 2003a). Effects include reduced respiration and photosynthesis, chlorosis (yellowing of foliage), decreased needle retention, growth loss, and altered wood quality.

As pseudothecia block stomata, the entrance of carbon dioxide (CO_2) into the leaf is restricted, resulting in reduced carbon assimilation rates. Occlusion of 20% of stomata reduces stomatal conductance by approximately 20% and daily net CO_2 assimilation by 60% (Manter et al. 2003a). Another result of this decrease in CO_2 is decreased activated rubisco, which requires a molecule of CO_2 for its activation and then catalyzes the first step in carbon fixation (Manter et al. 2000). It is believed that occlusion of about 25% of stomata results in overall negative needle carbon budgets on an annual basis (Manter et al. 2003a). As respiration exceeds assimilation, the needles switch from being carbon sources to carbon sinks (Cannell and Morgan 1990).

When stomata are occluded by pseudothecia, there is an associated decline in photosynthesis. Manter (2002) found that SNC-infected needles had lower photochemical utilization due to a decline in the efficiency of photosystem II, resulting in twice as much excess absorbed light as uninfected needles. This excess light causes photooxidative damage and might contribute to needle abscission. Unexpectedly, severely infected trees still maintain a positive carbon budget for the year as they rely on their current-year needles (that are infected but the fungus has not developed yet) as the main source of photosynthate (Saffell 2013).

One of the first visible symptoms of SNC is chlorosis, or yellowing of foliage. The mechanism causing chlorosis is not quite understood and there are certainly other factors, such as soil conditions or pollution, which can cause chlorosis (Gowin and Goral 2015, Chappelka and Grulke 2016). Therefore, it is not used as a definitive diagnosis of SNC but it can be used as one type of measure in conjunction with others or to identify general areas of disease boundaries when SNC is known to exist in a particular stand.

Decreased needle retention is another symptom that can help narrow the diagnosis to SNC. As *P. gaeumannii* colonization increases, photosynthetic rate of the needle decreases, and when the cost of maintaining a needle exceeds the benefits from retaining it, the needle is shed (Manter et al. 2000). Hansen et al. (2000) hypothesized that the proportion of stomata occluded by pseudothecia rarely exceeds 50% so needles are shed before half of the stomata are occluded, regardless of age.

Under ideal conditions coastal Douglas-fir can hold its needles for up to seven years in western Oregon and Washington (Mitchell 1974), although a more typical

number is four years (Maguire et al. 2002). Needle longevity is related to many factors, including latitude (Xiao 2003), climate (Coop and Stone 2007), site fertility (Balster and Marshall 2000, Niinemets and Lukjanova 2003), and insect and disease history (Hansen et al. 2000). Populations with high levels of SNC commonly only retain less than a year of foliage (Maguire et al. 2002), although the average in the epidemic area is 1.5 to 2.6 years (Hansen et al. 2000). Zhao et al. (2012) studied needle longevity in the Oregon Coast Range and found that most needles survived the first two years but were likely to die after that.

Foliage diseases typically reduce foliage retention more in the lower crown, probably because of moist microclimate; however, SNC is unique in that most of the effects are seen in the upper crown (Hansen et al. 2000, Manter et al. 2003b, Shaw et al. 2014, Weiskittel et al. 2006). Warmer temperatures (that favor fungal growth) in the upper crowns or photooxidative damage may cause the intensified symptoms (Manter 2002).

Defoliation also has implications for the ecosystem as a whole. Processes such as nutrient cycling, decomposition rates, and soil temperature and moisture levels can all be modified by defoliation (Kizlinski et al. 2002). Not only is the amount of litterfall increasing, but the timing is altered as well, with foliage falling not only in the fall as per usual, but also in the early summer, which is consistent with the lifecycle of *P. gaeumannii* (Weiskittel and Maguire 2007).

Since SNC does not kill the trees it infects, the biggest concern is growth loss, especially in plantations where the trees are grown for timber harvest. Although

reduction in foliage mass and thus photosynthesis is the main cause of growth loss, the role of pseudothecia in inhibiting gas exchange on surviving foliage is also a contributing factor (Manter et al. 2000). These different mechanisms of growth impact are reflected in the different SNC indices used to calculate growth loss: foliage retention surveys indicate potential growth decline due to foliage loss and pseudothecia counts represent the potential growth decline caused by disruption of gas exchange by pseudothecia. Another mechanism suggested by Saffell et al. (2014b) is that SNC-infected trees redirect their carbohydrates to new needles and branches, at the expense of trunk growth.

Maguire et al. (2011) found that once infected, the decline in growth was steady, without a threshold at which the loss suddenly appeared or worsened. They also correlated growth loss with declines in foliage retention and calculated growth loss from SNC as approximately 50% after a 75% reduction in foliage retention and approximately 25% after a 50% in foliage retention. Shaw (2008) made a general recommendation to landowners to not worry about growth loss unless their trees are retaining less than three years of foliage, at which point reduced growth is typically first detected.

Growth analyses can provide another insight in the effects of SNC. A growth reconstruction analysis was performed by Maguire et al. (2002) in northwestern Oregon in which they used annual height increments and stem section radial growth measurements to calculate a volume growth loss of 23% in 1996, with the most severely diseased plantations reaching a 52% loss. Hansen et al. (2000) estimated height growth losses at 50 to 70%, corresponding to as much as a 30% volume growth loss. A tree-ring analysis on older (>80 years) trees was performed by Black et al. (2010) and found that radial growth was reduced by as much as 85% at the most severely diseased site.

The arrival of SNC in New Zealand provided a unique opportunity to quantify growth increment loss. SNC was discovered there in 1959 and has been closely monitored throughout its spread, with records of the timing of its arrival in different forests known. Kimberley et al. (2011) analyzed these growth rates and found a cumulative mean reduction of 32% for stem volume. The variation in the estimates of growth loss could be due to variation in other site factors or different methodologies in quantifying the loss; however, there is a clear consensus that growth loss on some level is occurring.

The forest products sector relies heavily on Douglas-fir wood for structural lumber so any changes to its quality are a concern for the industry. The ratio of earlywood, which is formed in early spring by relying on photosynthate from older cohorts, to latewood, dependent mostly on new foliage, dictates the structural integrity of the wood (Onaka 1950). Johnson et al. (2003) found that latewood and earlywood growth in SNC-infected Douglas-fir was 44 and 68% less, respectively, than trees sprayed with fungicide (not infected with SNC). Since there was more of a growth loss in earlywood than latewood (due to its reliance on the more heavily infected older needles), the higher latewood proportion resulted in increased wood density. Although this might seem like a benefit, the weakest portion of wood is earlywood and by reducing earlywood cell wall thickness, Johnson et al. (2003) suggest that in SNC-influenced wood, the weakest link could be further weakened.

SNC-infected trees also have a smaller sapwood area, lower sapwood moisture content, and more gas-filled space in the sapwood, probably due to a decrease in sap flow and specific conductivity (Edwards and Jarvis 1982, Granier et al. 2000, Puritch 1971).

Johnson et al. (2003) found a 21% decrease in moisture content in trees with severe SNC, partially due to increased latewood (de Kort 1993) but also because of insufficient photosynthate (energy) to reverse sapwood embolisms. Trees commonly refill air embolisms in the xylem, but photosynthate is required and it is possible that diseased trees lack the photosynthate reserves needed to reverse these common gas embolisms (Johnson et al. 2003). The reduction in sapwood area is due to decreased foliage (Grier and Waring 1974) and since sapwood is of higher green density and moisture content, this would result in reduced log weights and less opportunity to use the timber for poles or pilings (Johnson et al. 2003).

History of Swiss needle cast discovery

SNC has an interesting history, as it was first discovered outside of its native range and the consequent knowledge of its origin and spread assisted in narrowing down the environmental factors that control its distribution. The history of the disease offers a parallel explanation of its geography, and in the case of a pathogen such as *P. gaeumannii* that is heavily impacted by its environment, geographic location has turned out to be a key piece of information in understanding the disease.

Worldwide spread followed by discovery in United States

The fungus that causes SNC, *Phaeocryptopus gaeumannii*, was first discovered in 1925 in Switzerland and within fifteen years it was identified in Douglas-fir stands across Europe and the United Kingdom (Boyce 1940, Liese 1938, Peace 1962, Wilson and Waldie 1928). By 1940 SNC was discovered in the northeastern United States and reports continued to come in from Turkey, Australia, New Zealand, and Chile (Beekhuis 1978, Boyce 1940, Hood et al. 1990, Morton and Patton 1970, Osorio 2007, Marks and

Pederick 1976, Temel et al. 2003). Since *P. gaeumannii* does not live or sporulate saprophytically (on dead organic matter), its distribution may have occurred through the movement of infected Douglas-fir nursery stock (Stone et al. 2007a).

Later examination of herbarium specimens showed that *P. gaeumannii* was present in Oregon as early as 1916 and because of its wide distribution and (up until then) harmless effects in the Pacific Northwest, it was considered to be native to the region (Boyce 1940). However, by the mid-1970s Christmas tree growers in the Pacific Northwest began reporting damage from SNC, resulting in annual losses of $3.4 million (Michaels and Chastagner 1984b). By the mid-1980s, forest managers and researchers began noticing more frequent and severe cases of SNC in the coastal timber plantations of Oregon and Washington.

A worsening epidemic

The 1990s saw a dramatic increase in distribution and severity of the disease. In response, the Swiss Needle Cast Cooperative was established at Oregon State University in 1996 and scientists began conducting aerial surveys to ascertain the geographical extent of the disease. In 1999, approximately 119,303 ha of 1.1 million ha (295,000 ac of 2.9 million ac) aerially surveyed in coastal Oregon were classified as severely infected (Hansen et al. 2000) and a similar survey in coastal Washington found approximately 81,000 ha of 930,000 ha (200,000 ac of 2.3 million ac) infected with SNC (Omdal and Moore 1999).

The area of Douglas-fir showing symptoms of SNC has been increasing in Washington, from 94,000 ha (228,500 ac) (8.5%) in 2012 to 141,000 ha (349,704 ac)

(14%) in 2015 (Ramsey et al. 2015) (Fig. 2). Aerial surveys have been more consistent in Oregon and their data shows variation year-to-year but an obvious trend of rapid increase (Fig. 3). Maguire et al. (1998) calculated a growth loss due to SNC of about 3.2 m^3/ha/yr (230 board feet/acre/year) from plantations growing at a rate of 14 m^3/ha/yr (1,000 board feet/acre/year) in 1996. In Oregon this came out to be 602 m^3/ha/yr (43 million board feet/acre/year) lost to the disease (Maguire et al. 1998). Observed damage has been most severe within 18 miles of the coast, but not all coastal stands are severely damaged (Kanaskie and McWilliams 1998, Hansen et al. 2000).

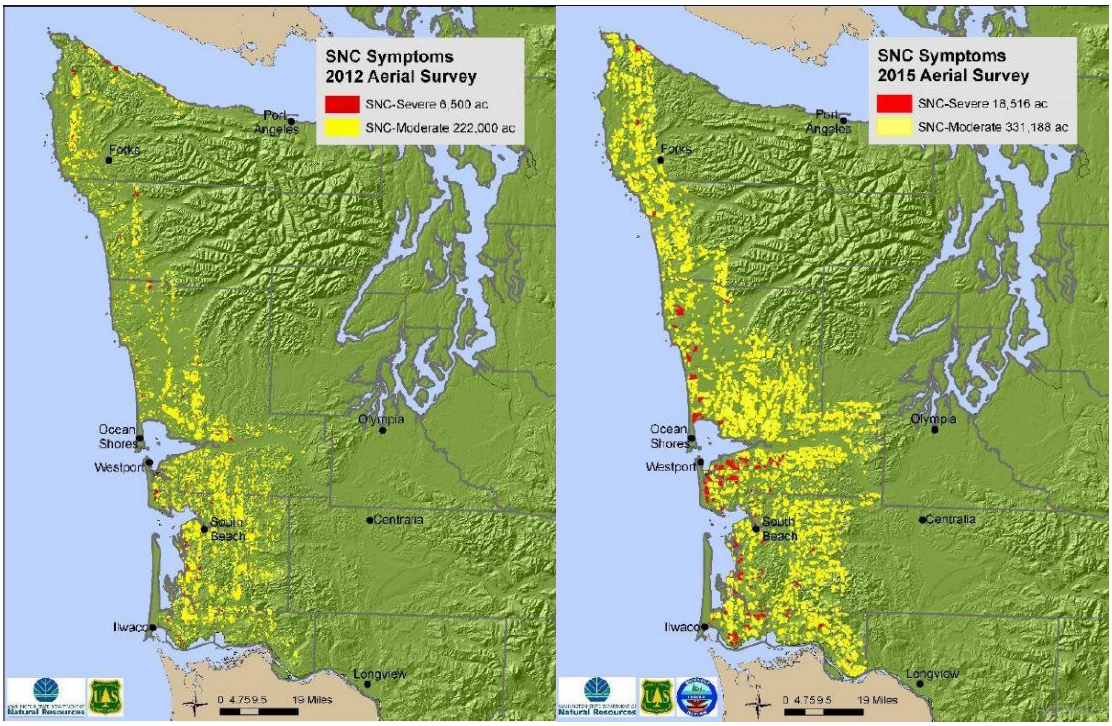


Figure 2. Washington aerial survey maps showing the spread of SNC from 2012 to 2015. From Ramsey et al. 2015.

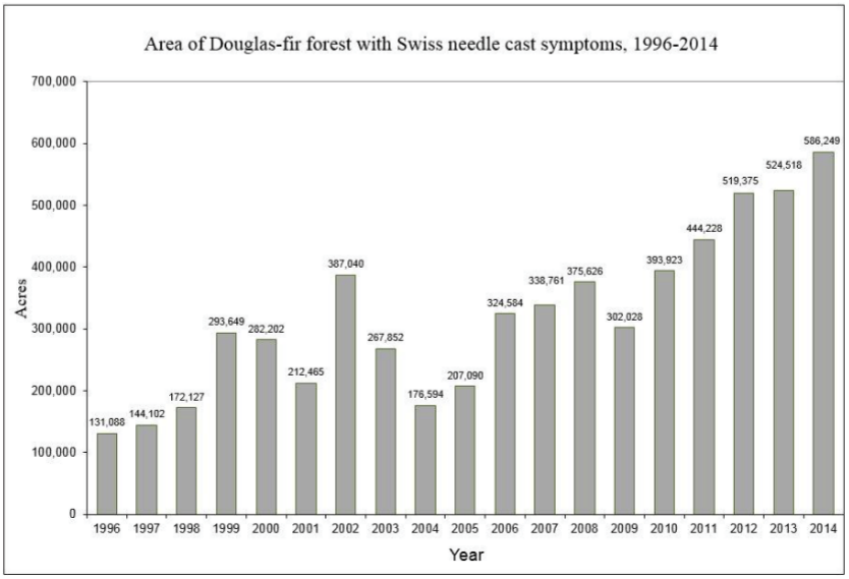


Figure 3. Area of western Oregon showing SNC symptoms during aerial surveys flown in 1996-2014. From Kanaskie and Norlander 2014.

Investigating the cause of the epidemic

The severity of the epidemic in Oregon has spurred a surge in research, particularly focusing on how an inconspicuous, insignificant native pathogen could so quickly become a serious forest health issue. A popular conceptual model in epidemiology is the disease triangle between the causal agent (*P. gaeumannii*), the host (Douglas-fir), and the environment (McNew 1960, Hansen 1996). Plant pathology requires all three components to be present and elimination of any one can prevent disease. Efforts to strengthen the understanding of this relationship have been ongoing and many questions still remain. Each corner of the triangle represents a possible explanation for the disease outbreak, some of which are examined here.

One hypothesis to explain the outbreak was that many of the coastal plantations were established from seed collected farther inland and at higher elevations than native

coastal Douglas-fir stands (Maguire et al. 1998). However, professional foresters have long known about geographic variation in tree genotypes and by 1966 many programs were in place to assist them in appropriate selection of stock, including forest tree seed certification by site of origin, seed zone maps, and seed transfer guidelines (Randall 1996). The interior form of Douglas-fir (*P. menziesii* var. *glauca*) is more susceptible than the coastal form (*P. menziesii* var. *menziesii*) but foresters do not grow the interior form in western Oregon and Washington and it does not occur naturally in the Coast Range (Manter et al. 2005).

Kastner et al. (2001) investigated SNC susceptibility of Douglas-fir from different seed zones. They compared growth of 50 seedlings from each of three seed sources (coastal fog belt, western Oregon Coast Range, and standard vendor-purchased seed) in an area heavily impacted by SNC, finding no significant difference in basal diameter, height growth, and needle retention. Additionally, Temel et al. (2004) examined 108 15-year-old Douglas-fir trees from six families, finding that the amount of *P. gaeumannii* on foliage did not differ significantly. The widespread distribution of the disease in the western Coast Range, with little variation in susceptibility among provenances and families from that area, deems inappropriate seed source an unlikely cause of the current epidemic.

Pathogen genetic variation is another possible explanation for the current epidemic. The population of *P. gaeumannii* in North America has been found to contain two distinct, non-recombining, sympatric lineages (Winton et al. 2001, 2006). While Lineage 1 exists both throughout the Pacific Northwest and abroad, Lineage 2 has not been found in any of the samples from Europe and eastern North America and has been

associated with more severe disease symptoms (Winton et al. 2001, 2006). Some preliminary work by Bennett and Stone (2014) in Washington found two out of twenty isolates that were genotyped belonged to Lineage 2, meaning that strain is present here in small numbers. Although Lineage 2 is comprised of five genotypes, it was only represented by a single genotype in most stands in western Oregon, which Maynard Smith et al. (1993) classify as an epidemic population structure, with one or a few highly successful genotypes. This is an interesting possibility that warrants further work into differences in pathogenicity of the two lineages or individual genotypes.

Since the discovery of *P. gaeumannii*, local climate has been believed to be a major cause of its pathogenicity (Boyce 1940, Hood 1982). This hypothesis has persisted as the most widely accepted explanation for European and western North American differences in disease severity. Much research has gone into analyzing how the annual fluctuations in SNC severity have corresponded to annual fluctuations in various climatic factors. A four-year study by Stone et al. (2008a) found that measures of severity within plots with the same seed source remained at similar levels over the years, suggesting that differences in severity between sites is due to environmental or other site factors. The primary hypotheses regarding climate are that mild winter temperatures are more conducive to the development of fungal mycelia in Douglas-fir needles and abundant spring moisture encourages spore germination on needles and allows hyphae to grow across the needle surface (Latta et al. 2009, Manter et al. 2005, Rosso and Hansen 2003, Stone and Coop 2006).

Forest pathology requires a landscape perspective and anthropogenic impacts on landscape patterns in relation to disease is another important perspective to examine

(Holdenrieder et al. 2004). A 1997 survey by the Oregon Department of Forestry found that 80% of the 76,970 hectares of Douglas-fir plantations within 29 kilometers of the Oregon Coast had been occupied by other species in the previous rotation (Manter et al. 2005). Much of this converted land and where SNC is most severe, lies within the Sitka spruce (*Picea sitchensis*) vegetation zone (Fig. 4). Characterized as wet maritime, winter and summer temperatures are moderate and average annual precipitation is 150 inches, in addition to fog and clouds that contribute further moisture (Henderson et al. 1989). Coincidentally, these are also ideal growing conditions for *P. gaeumannii*. The subtle climatic differences that dictate the different vegetation zones could also be dictating disease severity. Douglas-fir is the early seral dominant species in the western hemlock (*Tsuga heterophylla*) vegetation zone, commonly bordering the Sitka spruce zone to the east; however, Douglas-fir usually only occurs sporadically within the Sitka spruce zone, not as pure stands (Stone et al. 2008a). This may mean that the Sitka spruce zone exists not only because of favorable habitat for spruce and hemlock, but also because its main competitor, Douglas-fir, isn't able to outcompete it due to SNC in coastal environments (Stone et al. 2008a).

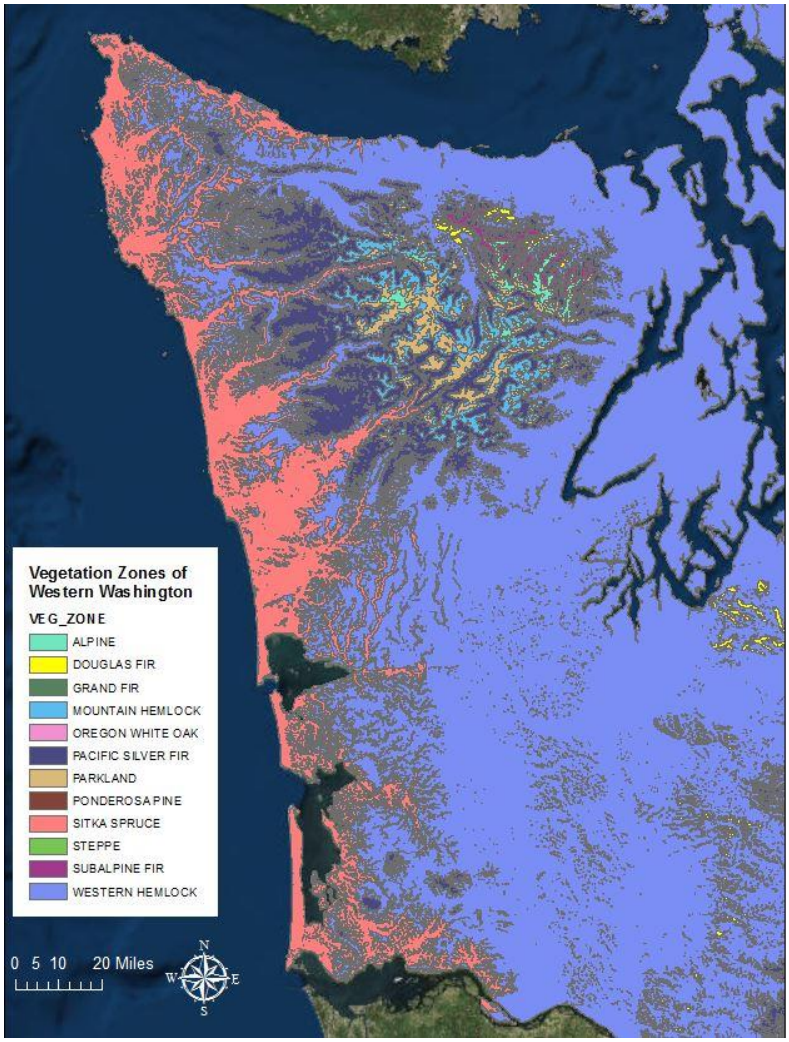


Figure 4. Map of vegetation zone in western Washington.

The combination of increased levels of Douglas-fir within the coastal Sitka spruce zone and a climate favorable to fungal infection and growth is the most likely cause of the current epidemic. However, the complex gradient of moisture, temperature, and fog within this zone is a very broad explanation and detailed climatic variables alone (discussed in the next section) are more useful in explaining which specific conditions are the most influential.

Current research

When SNC was first recognized as a problem in the Pacific Northwest, the initial reaction was to find a cure. While there have been no outright successes to date, this is still an area of interest to many land managers trying to develop their management strategies. More recently the research focus has shifted to learning where are, and which specific environmental factors are causing, the most severe disease impacts. This seems to be the most promising avenue, to learn high-risk areas that should be avoided, but first the disease control research will be discussed.

Attempts to control/minimize disease impacts

Traditional approaches to reduce or prevent SNC infection have met limited success; however, landowners can practice adaptive silviculture to meet their objectives. With techniques ranging from fungicide or fertilizer application to selective Douglas-fir genetics, a combination that is most suitable for individual landowners' management goals can be formulated. The Oregon State University Swiss Needle Cast Cooperative recommends mixed species plantations not to reduce disease severity, but to at least minimize economic risks (Shaw et al. 2011). This can also help to provide better habitat that is less impacted by disease for late-seral species such as northern spotted owls. In some situations where an area is known to be high-risk, Douglas-fir simply should not be grown and other species such as western hemlock should be considered (Kastner et al. 2001).

The fungicide chlorothalonil (Bravo) has been recommended as a disease control measure in Christmas tree plantations for quite some time (Skilling 1981, Hadfield and Douglas 1982, Chastagner and Byther 1983, Pscheidt and Ocamb 2005), with

recommendations to start applying annually three years prior to planned harvest. Crane (2002) used elemental sulfur (Thiolux) as a fungicide, as well as chlorothalonil (Bravo) fungicide, and found that Bravo was the most successful in reducing infection levels. Although only one year of treatment was applied, no difference in growth (height or diameter at breast height) was observed.

Other potential fungicides have been tested with mixed results. It has been proposed that sulfur can act as a fungicide, decreasing disease incidence and improving foliage color and retention (Chastagner 2002, Stone et al. 2004). However, aerially applied treatments of sulfur and sulfur plus nutrients showed no significant increase in volume of SNC-infected stands (Younger et al. 2008). Copper fungicide was aerially sprayed in a Douglas-fir plantation in New Zealand with no effects; however, switching to hand spraying the same material at the same concentration reduced the proportion of infected needles to below 42%, compared to 100% in unsprayed controls (Hood and van der Pas 1979). This would suggest that the hand-sprayed application covered foliage more thoroughly.

While fungicides can be a successful method of disease control, the effects are brief. Stone et al. (2007) sprayed chlorothalonil (Bravo) fungicide aerially for five years and while fruiting bodies decreased and needle retention increased, these effects only lasted one year post-treatment, probably due to decreased inoculum production, and then foliage returned to prior levels of infection. It appears that enough residual inoculum remained in the tree crowns to allow rapid re-infection as soon as treatments stopped (Stone et al. 2007b). The prohibitively high costs associated with annual application as well as the environmental risks involved with prolonged exposure of both terrestrial and

aquatic systems to these compounds are enough to discourage fungicides as a long-term solution.

Soil nutrition has long been recognized as important in sustained and high production of timber and has been found to have a significant impact on biomass production and overall growth (Mitchell et al. 1996). In particular, nitrogen fertilization as urea has been common in the Douglas-fir region of the Pacific Northwest (Bengston 1979). However, Filip et al. (2000) suggest that nitrogen fertilization may be worsening SNC severity. Waring et al. (2000) found a strong correlation between increased foliar nitrogen concentration, low soil calcium and low soil pH, and decreased foliage retention, possibly due to excess nitrogen being stored as soluble amino acids that promote the growth and development of *P. gaeumannii*. El-Hajj et al. (2004) used nitrogen (urea) to fertilize Douglas-fir and found 2.2 to 3.6 higher levels of pseudothecia density on 2-year-old needles. Some managers have begun to fertilize with lime to lower the ratio of nitrogen to other essential nutrients (Mulvey et al. 2013).

It is true that fertilizer treatments can impact disease severity in different ways, depending on the pathosystem, the specific application materials, timing of application, and other factors (Datnoff et al. 2007, Engelhard 1989). Fertilization may reduce disease by bolstering plant resistance or tolerance but may also increase disease by improving pathogen access to nutriment through its host (Mulvey et al. 2013).

Many researchers have found that fertilization has neither positive nor negative effects on *P. gaeumannii* severity. An experiment on fertilization impacts of nitrogen (urea), calcium as lime (calcium carbonate), calcium as calcium chloride, phosphorus

(monosodium phosphate), and a site-specific blend resulted in no significant effect on the *P. gaeumannii* infection levels of Douglas-fir both across and within sites (Mulvey et al. 2013). Crane (2002) found that fertilizer (N-P-K) treatments had no significant impact on growth of trees affected by SNC. Mainwaring et al. (2009) also looked at foliage retention, volume growth, form quotient, and sapwood area in response to seven types of fertilizer treatments, finding no improvements. Realistically, managers should assess the soil chemistry at each site and fertilize accordingly, but not expect it to have much effect on SNC.

Although traditional fertilization does not appear to affect SNC, soil nutrition still may contribute to Douglas-fir susceptibility in other ways. The soil microbial community is a key component of stable and healthy growing conditions and some common types of ectomycorrhizal fungi commonly found with Douglas-fir, such as *Cenococcum* and *Rhizopogon*, were found to be less widespread in SNC stands than uninfected stands (Luoma and Eberhart 2014). This suggests that "stress tolerant" ectomycorrhizal fungi may be important for sustaining Douglas-fir against the disease.

Pre-commercial thinning is a common management practice in young timber stands where high stand densities limit individual tree growth and leave trees more susceptible to insects and disease (Mitchell et al. 1983). Intraspecific and interspecific competition can also decrease resource availability and reduce tree growth (Cole and Newton 1987). Some suspect that thinning can increase air circulation, resulting in a drier microclimate, in which the fungus will not grow as well. Conversely, it is possible that the stress of thinning in stands already damaged by SNC may actually have negative impacts on residual trees (Filip et al. 2000).

Mainwaring et al. (2005) found that pre-commercial thinning does not intensify disease development. Infected stands do respond positively to thinning but growth is still lower than in uninfected stands. Severely infected stands experienced a volume growth loss of 36% when thinned (Mainwaring et al. 2005), compared to Maguire et al. (2002)'s finding of 50% in unthinned stands. In northwest Oregon, monitoring four seasons after thinning resulted in no difference in needle retention, positive or negative (Kanaskie et al. 2002). Thinned stands in New Zealand had infection rates similar to unthinned controls and had as much foliage five years after thinning (Hood and Sandberg 1979).

The overall recommendation is that thinning in low- or moderate-severity stands will still be beneficial in most stands, but should be avoided in high-severity stands (Filip et al. 2000). Even if thinning does not produce results acceptable for timber production, it can be a reasonable strategy in forests managed for timber along with other purposes such as wildlife and aesthetics (Oliver 1992, Oregon Department of Forestry 2001).

Douglas-fir trees growing within the same environment can exhibit different levels of symptoms, due in part to each tree's genetics (Johnson 2002, Temel et al. 2005). The main focus of genetic testing has revolved around determining if trees exhibiting less severe SNC symptoms are more resistant (less fungal colonization) or more tolerant (retained foliage under similar levels of infection) (Temel et al. 2004). Temel et al. (2004) examined trees from six families, categorizing them into three disease severity groups: mild, moderate, and severe. From this study, they found that the amount of *P. gaeumannii* did not differ significantly between the groups that were exhibiting different levels of symptoms; therefore differences in severity were attributed to tolerance, rather than resistance. Hood and Kimberley (2005) found similar results in New Zealand.

This variation in tolerance appears to be a result of variation in environmental conditions. Many researchers have noted that susceptibility and symptom severity are higher in Douglas-fir genotypes originating from areas of lower rainfall and humidity (Hood 1982, McDermott and Robinson 1989, Stephan 1997). The reasoning is that higher rainfall and humidity increase disease pressure, resulting in increased selection pressure for disease resistance to SNC. Therefore, seed source originating from the interior Rocky Mountain form of Douglas-fir (var. *glauca*) is more susceptible to the disease than the coastal form (var. *menziesii*) when grown together in provenance trials (Hood 1982).

The practical outcome of this research is that family selection for SNC tolerance at the seedling stage could be used to increase tolerance of managed stands. Foliage traits of crown density and color are reasonable indicators of tolerance, which are fairly simple to assess (Johnson 2002). This would also give land mangers a head start on culling poorly performing trees during thinning as the trees age.

Increasing our understanding of the disease: the importance of monitoring surveys

The first step in studying plant pathogens is to obtain an accurate measure of pathogen abundance. There is an important distinction between disease symptoms and signs: a symptom is a visible change in the plant, whereas a sign is the actual presence of the organism causing the disease (Temel et al. 2004). Some new methods of quantifying fungal colonization have been established, such as ergosterol content analysis, where a cell membrane sterol found only in fungi is measured for fungal biomass (Manter et al. 2001, Winton et al. 2003) and quantitative PCR, where amplification of fungal DNA with a polymerase chain reaction is monitored in real-time (Winton et al. 2003); however, the

more widely used methods of needle retention surveys, aerial surveys, and pseudothecia counts will be discussed here.

Since foliage loss is a primary symptom of SNC, needle retention surveys are a straightforward, easy to assess method that is widely used. Needle retention is typically estimated on a scale of 0 to 9, with 0 representing 0-9% retention and 9 representing 90-100% retention. Ideally three to five years-worth of needles are evaluated on each tree and then averaged together. Needle retention is best surveyed in April and May by examining lateral branches in the middle to upper crown of at least ten randomly chosen dominant or codominant trees (Fig. 5).

Figure 5. A healthy Douglas-fir (left) compared to one showing decreased needle retention due to SNC (right). From the Oregon Department of Forestry.

When the symptoms of foliage color, foliage density, needle color, and needle retention were compared to levels of infection (signs), needle retention was the only related trait (Temel et al. 2004). Needle retention also shows a strong positive correlation with tree growth, which is a primary concern of many surveyors (Maguire et al. 2002, 2011). The ease of foliar retention surveys translates to wider geographic areas that are able to be surveyed and a more accessible method for landowners. However, the objectivity of this method is questionable. Temel et al. (2005) pointed out that these visual assessments could be quite subjective in assigning numbers on a continuous scale of 0-9, leading to questions of their value.

In 1996 the Oregon Department of Forestry began flying annual aerial surveys to assess the extent of damage caused by SNC, through the sponsorship of the Swiss Needle Cast Cooperative. Objectives are "to identify and map areas of Douglas-fir forest with obvious symptoms of SNC in the Oregon Coast Range; and to monitor change over time in the amount of forest with visible symptoms of the disease" (Maguire et al. 1998, p. 705). Flights are typically flown in April or May and areas showing the color signature of SNC chlorosis are drawn as polygons on topographic maps. It is important to note that aerial surveys only map where symptoms are severe enough to be visible from the air and are not meant to be a comprehensive inventory. Another complication is the fact that chlorosis can be caused by other factors besides SNC, and appear in stands that aren't even composed of Douglas-fir so overestimation is a possibility as well. The idea is to map general areas to obtain acreage estimates for documenting trends over time (Kanaskie et al. 2001).

Aerial surveys are costly and have many limiting factors including varying weather conditions, which can affect the appearance of chlorosis and smaller areas that may be overlooked (Hilker 2014). Kanaskie et al. (2002) acknowledged that the same areas sometimes were not mapped each year due to timing of the survey, cloud conditions at the time of flight, changes from harvesting, and precision errors in sketch-mapping polygons. However, the value in being able to get a picture of the landscape as a whole is undeniably important in monitoring a widespread disease such as SNC.

Pseudothecia counts are commonly recommended as the preferred method for quantifying SNC due to their high correlation with symptom severity (Hood 1982, Michaels and Chastagner 1982a, Hansen et al. 2000, Manter et al. 2001, Winton et al. 2003). Typically ten one-year-old and ten two-year-old needles per tree are examined under a microscope to count the percentage of stomata occluded. "Because the physiological effects of the disease (impaired CO_2 uptake and photosynthesis) are quantitatively related to the abundance of the pathogen (proportion of stomata occluded by ascocarps), pathogen ascocarps abundance [pseudothecia counts] is a suitable response variable for assessing effects of climatic factors on disease" (Stone et al. 2008a). While needle retention surveys and aerial surveys are operationally useful, the best way to understand the epidemiology of this outbreak is by observing the response of the pathogen itself, i.e. pseudothecia counts. Winton et al. (2003) found a -0.644 Pearson correlation coefficient between the pseudothecia count and needle retention, whereas ergosterol content was only -0.578 and quantitative PCR was only -0.451. There is also a proven pathway that exists between pseudothecia density and physiological impacts (Manter et al. 2000).

Some challenges associated with pseudothecia counts are high labor and statistical challenges. Costs of equipment are low with microscopes being the only major cost, but manually counting stomata and pseudothecia is time-consuming and subject to misidentification error (Winton et al. 2003). Since only a small fraction of stomata are examined and pseudothecia can be very unevenly distributed, clustering can result in estimates that are too high or too low (Winton et al. 2003).

Identifying favorable conditions

Various environmental conditions such as climate, topography, soil characteristics, and forest stand characteristics are associated with disease in Oregon (Rosso and Hansen 2003). By studying the natural spatial and temporal variations of the disease, we can increase our understanding of the outbreak and isolate the conditions that facilitate fungal growth. In 1940, Boyce hypothesized that the most likely explanation for differences in how the disease was expressing itself in Europe versus the United States was climatic differences, specifically the humid, rainy summers in Europe that the disease thrived in, compared to the warm, dry summers of western North America.

Spatial variation (both topographical and within each tree) in how the disease is expressed is high across forest plantations (Hansen et al. 2000). Disease severity tends to fluctuate from year to year; however, the relative ranking of sites by severity has remained mostly constant, which leads researchers to believe that site-related factors play an important role in disease severity (Hansen et al. 2000, Stone et al. 2001).

Much research has gone into climatic factors, but additionally, studying different temporal scales is recommended. Zhao et al. (2012), Stone et al. (2007), and Manter et

al. (2005) looked at climatic variables at the annual, seasonal, and monthly scales, finding that the seasonal variables were most suitable according to statistical scores, which makes sense considering the life cycle of *P. gaeumannii*. In contrast, Zhao et al. (2011) performed a similar study and found that annual and monthly variables explained more variation than seasonal variables. In New Zealand, Watt et al. (2010) found monthly variables to have the strongest correlations. The scale being used is critical, as Zhao et al. (2011) predicted an increase in foliage retention with their annual and monthly models, but a decrease in foliage retention with their seasonal model. While the annual resolution seemed adequate to Zhao et al. (2011), they cautioned that the possibility of greater monthly variation in future climate could deem the monthly resolution most accurate.

Free (surface) moisture on needles provides ideal conditions for spore germination and initial infection (Stone et al. 2008b). Capitano (1999) identified free moisture as a key factor in hyphal growth of *P. gaeumannii* in culture after observing that dry conditions for 24 hours or more significantly reduced germination and growth. Infection of inoculated needles occurred after 24 hours of exposure to moisture (Capitano 1999). However, high amounts of spring rainfall and summer fog in coastal Oregon (and Washington) may negate the importance of this as a limiting factor (Manter et al. 2003b).

Most of the severe disease symptoms appear in the low elevation coastal fog zone, so the amount of free water on foliage during summer months is another possible contributing factor to disease severity (Hansen et al. 2000, Rosso and Hansen 2003). Saffell et al. (2014a) used stable isotopes of carbon and oxygen in Douglas-fir tree rings and western hemlock as a non-susceptible reference species to identify changes in growth that could be related to climate factors. Results found that high relative humidity in the

previous two summers was most strongly correlated with the diagnostic tool of decreased tree-ring $\Delta^{13}C$, and thus disease severity. The researchers speculated that this was due to higher leaf wetness from increased nightly dew frequency, lower evaporation rates of dew, and less spores washed off than rainfall, all resulting in more successful germination on foliage.

Zhao et al. (2012) identified March precipitation as a general surrogate for spring wetness, due to its correlation with late spring precipitation. Rosso and Hansen (2003) categorized variables of fog occurrence, precipitation, vapor pressure deficit, altitude, and aspect all into the category of "moisture" that was correlated with higher SNC severity.

Precipitation has long been believed to play a critical role in the spread of SNC. Rainfall during the infection period aids the spread of ascospores and contributes to the moisture discussed above (Stone et al. 2008b). Hood (1982) observed a positive correlation between spring rainfall and *P. gaeumannii* abundance with more than 80% of needles infected in wet areas, compared to only 5% in the rain shadow of eastern Vancouver Island, Olympic Peninsula, and Coast Mountains of British Columbia. Precipitation during the infection period of May and June has been recognized as a contributor to regional variation in British Columbia (Hood 1982, McDermott and Robinson 1989). In New Zealand, Watt et al. (2010) found a significant quadratic correlation with November (late spring) rainfall. Fungal colonization increased until 149 mm per month was reached and then declined, possibly due to ascospores being diluted or washed off needles.

In the United States, Zhao et al. (2011) found that foliage retention decreased with greater precipitation with the greatest influence coming from spring and early summer precipitation. Spring and summer precipitation was a key model input for Manter et al. (2003c, 2005) during their greenhouse studies. Lee et al. (2013) examined Douglas-fir tree-ring chronology records dating back to the 1590s, with nonsusceptible western hemlock as the climate proxy, and found that summer precipitation was correlated with SNC impact. Regional climate trends are reflected in SNC severity as well, seen by the 1.6 to 2.6 cm per decade increase in precipitation in coastal Oregon and Washington since 1966 (U.S. Department of Commerce, National Oceanic and Atmospheric Administration 2005).

Warm conditions, identified by variables such as winter temperature, vapor pressure deficit, altitude, and aspect, were positively correlated with SNC severity (Rosso and Hansen 2003). Other studies have shown that *P. gaeumannii* ascospore production (Michaels and Chastagner 1984a) and hyphal growth and germination (Capitano 1999) increase with temperature. Labwork by Capitano (1999) found that optimum germination and growth temperatures were 18°C and 22°C, respectively.

Manter et al. (2003c, 2005) found that average winter (December-February) daily temperature had the biggest influence on the disease, likely acting as the limiting factor in fungal colonization and growth. Similar results were also found in New Zealand, with mean June (winter) temperature accounting for 53% of the variance in fungal colonization (Stone et al. 2007a). The extreme growth losses documented in forests older than 80 years by Black et al. (2010) were associated with warm conditions in late winter and early spring. Winter temperatures were also found to be a strong correlative in the

tree-ring analysis dating back to the sixteenth century by Lee et al. (2013). Disease severity models in western Oregon based on winter temperature were found to explain 77% and 78% of the variation in one- and two-year-old needles, respectively, and 80% in New Zealand (Stone et al. 2007a).

Climate-based models created by Zhao et al. (2011, 2012) found that increasing summer temperature, decreasing winter temperature, or increasing difference between the two resulted in increased foliar retention. The influence of summer temperature is not supported by other studies, and Zhao et al. (2011) suspect the reason it came up in their analysis was due to other mechanisms occurring during summer, such as the negative effects of high vapor pressure deficits, low water potential within foliage, low water availability to fungal hyphae, and water stress on the tree itself (Zhao et al. 2011). Manter et al. (2003c, 2005) also found that summer temperature (July maximum) corresponded to lower disease values probably because high summer temperatures combined with low moisture may inhibit fungal development.

Regional climate trends also seem to correspond with the increased SNC severity seen since the 1990's (Manter et al. 2005). Temperatures in the Pacific Northwest have increased by approximately 0.8°C over the last century, with most of that change occurring over winter months (Mote et al. 1999). In coastal Oregon and Washington, average January to March temperatures have increased by approximately 0.2 to 0.4°C per decade since 1966 (U.S. Department of Commerce 2005).

Manter et al. (2005) used their climate-based model to calculate that a 1°C increase in average daily winter temperature corresponds to an increase of 3.3 and 5.9%

in *P. gaeumannii* colonization for one- and two-year-old needles, respectively. Predictions for continued regional warming of 1.5°C by 2020 and 2.3°C by 2040 (Mote et al. 1999) therefore suggest that SNC severity is likely to increase (Manter et al. 2005). However, there is uncertainty in these climate predictions and one possible outcome is that spring moisture will become more limiting to fungal colonization than it currently is, resulting in winter temperatures being less relevant to disease severity (Zhao et al. 2011). Lee et al. (2013) hypothesize that continued warming in the coastal fog zone will increase SNC intensity and frequency, since needle wetness is not a limiting factor, but in areas where summers are dry, continued warming and drying will likely decrease SNC intensity and frequency.

Lack of environmental condition analysis in Washington

The relative importance of different climatic factors varies spatially. For example, Stone et al. (2008a) found that winter temperature was the best predictor of *P. gaeumannii* abundance in coastal sites (where wetness isn't a limiting factor) but spring leaf wetness was a more accurate predictor for inland sites. Similarly, Lee et al. (2013) found that winter conditions were more strongly associated with disease impact at wetter, cooler sites, but summer conditions were more important at less humid, warmer sites. Stands experiencing similar climatic conditions but lighter spore loads may be protected by lower disease pressure, resulting in the same climate variables having less of an effect on needle longevity (Zhao et al. 2012).

While Oregon and Washington have fairly similar climatic conditions, there are subtle differences that may impact the prevalence of SNC. Manter et al. (2003, p. 349) state that "relatively slight differences in microclimate can influence the colonization of

foliage by *P. gaeumannii* as well as the severity of disease symptoms." Hansen et al. (2000) noted that plantations of the same age and seed source frequently exhibited fewer disease symptoms if located just a few miles further inland or a few hundred meters higher in elevation. These nearby sites had subtle environmental differences but those differences may be significant. Care must be taken when using models outside the area for which they were created, as Zhao et al. (2012) found that another model (Latta et al. 2009) had significantly underpredicted foliage retention at nearly every site that occurred outside the geographic range.

An analysis on climatic factors associated with SNC severity in Washington specifically has not been completed. Considering the site-specificity of the disease's behavior, it could be that different variables are more strongly correlated with severity. It could also turn out to be the same as in Oregon; however, an analysis must be done in order to identify those variables.

Once this information is known, high-risk areas for SNC can be identified. "…Geographic variations in environmental determinants of pathogen growth rate could lead to predictable spatial patterns of disease severity in a long-lived, perennial host" (Manter et al. 2005, p. 1263). Identifying high-risk areas can help forest managers adjust their harvest schedules and species planting plans. Plantations in western Oregon have undergone unexpected changes in species composition whereby natural regeneration of western hemlock is replacing Douglas-fir (Hansen et al. 2000). Many landowners are also purposely planting more mixed species or switching entirely to species other than Douglas-fir. Perhaps Douglas-fir is no longer a suitable plantation species in the coastal Pacific Northwest due to SNC.

The importance of using an adaptive management framework to understand and respond to this epidemic is increasingly important in light of climate change. The Pacific Northwest is expected to have warmer and wetter winters (Mote and Salathe 2010), which could result in increased intensity, frequency, and range of SNC over the twenty-first century (Zhao et al. 2011, Watt et al. 2011). The pathogen appears to be responsive to slight variations in climate, and warmer, moister winters will likely increase infection rates. Thus, plantations that were only considered low-risk when planted might experience increased levels of impact in the changing climate (Watt et al. 2010). However, this also might provide some opportunity to extend the species range into areas that were previously unsuitable for Douglas-fir (Watt et al. 2010). Model simulations of natural vegetation in western Washington over three different future climate scenarios all showed the disappearance of optimal conditions for the Sitka spruce zone by 2100, with the western hemlock zone remaining the most extensive and the Douglas-fir zone growing (Conklin et al. 2015). Due to the specific windows in the life cycle of *P. gaeumannii*, the timing of changes in temperature and precipitation will dictate how SNC responds.

Conclusion

The current SNC epidemic appears to be a result of many factors. The rise in Douglas-fir plantations due to their economic value has increased the abundance of the fungal host species, and therefore inoculum, in the area most favorable for the growth and reproduction of the pathogen. Unfortunately, the environment that Douglas-fir grows well in is also the environment in which *P. gaeumannii* grows well. The current epidemic is likely the result of several weak points in the disease triangle.

Since the disease is not showing any signs of diminishing in the Pacific Northwest, we must learn to manage our forests in coexistence with it. To make these informed decisions, we need (1) an accurate description of the incidence and severity of the disease, and (2) some insight into climatic variables that encourage fungal growth. With this knowledge, Washington landowners can choose the most suitable locations for planting Douglas-fir and adjust their silvicultural strategies to meet their own goals.

CHAPTER II. Manuscript

Introduction

Swiss needle cast (SNC) is a foliage disease that exclusively affects Douglas-fir (*Pseudotsuga menziesii*). The pathogen was first described in Douglas-fir transplanted from its native range in the Pacific Northwest region of North America to Europe. In the 1970s, widespread symptoms were recognized in coastal Oregon and Washington, first in Christmas tree plantations, then in timber plantations. There are many mysteries about SNC extent and behavior in response to changing commercial plantation practices, climate change, and potential changes of pathogen or host. This thesis uses field data to examine the current extent and behavior of the disease along the coast of Washington State.

SNC is caused by the fungus, *Phaeocryptopus gaeumannii*. Symptoms include chlorotic (yellow) needles, decreased foliage (needle) retention, and reductions in growth; however, tree mortality is rare. Although SNC occurs throughout the natural range of Douglas-fir, the most pronounced expression is along the outer coast of Washington in the Sitka spruce forest zone.

In the early 1990s, SNC reached epidemic levels in Douglas-fir plantations of the Pacific Northwest. It is believed that prior outbreaks had occurred, but the level of severity and length of outbreak is unprecedented. Climatic factors are believed to have some effect, but many subtle factors can be involved and there is geographic variation in severity. The environmental factors that appear to influence the extent and severity of the

disease include topographical features, such as elevation and aspect, and climatic factors, such as temperature and precipitation.

Tree-level quantification of SNC severity is time-consuming and labor intensive, but pseudothecia (fruiting body) counts are unique in directly quantifying the amount of fungus, as opposed to broad estimates of severity based on symptoms. On a landscape level aerial surveys have been used to estimate disease occurrence by observer interpretation of foliage color, but there have been few efforts to link the two scales of survey. Aerial surveys have taken place in Oregon every year since 1996, but have occurred more sporadically in Washington. Both states have shown a dramatic three-fold increase in the acreage affected by SNC according to these aerial surveys.

Viewing the epidemic as a pathologist

Biotic (fungi, insects) and abiotic (temperature, moisture) factors are involved in what is commonly visualized as a "disease triangle." This conceptual model helps visualize that a susceptible host, virulent pathogen, and favorable environment are all required in order for disease to develop (Fig. 6). While humans are not directly represented on one of the vertices, they undoubtedly play a role in this interaction.

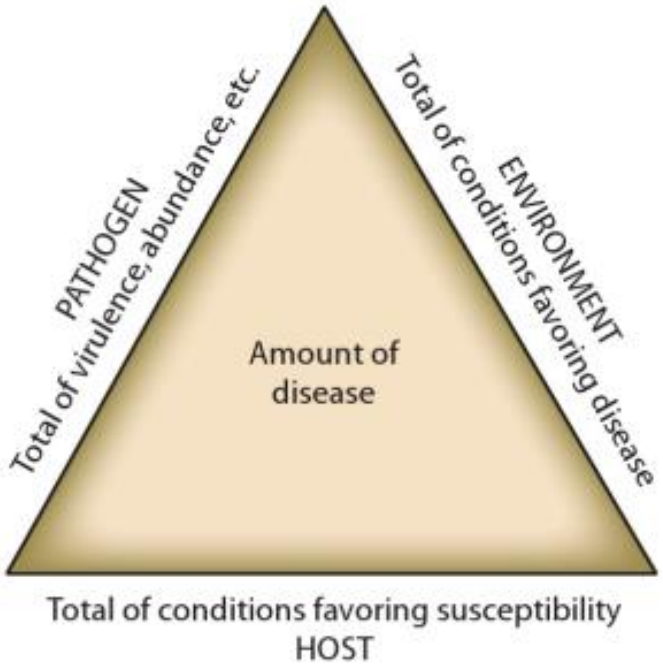


Figure 6. The disease triangle showing the interaction between pathogen, environment, and host that results in plant disease. From Agrios 2005.

SNC is caused by the pathogenic fungus, *Phaeocryptopus gaeumannii*, and is believed to have always existed in northwestern North America. *P. gaeumannii* has likely been transported to other areas around the world with the introduction of Douglas-fir from northwestern North America, but otherwise has not been affected by humans. However, humans have altered the other two vertices of the disease triangle allowing the fungus to proliferate. Douglas-fir, *P. gaeumannii's* only host, is now grown is large monocultures outside of its range of historic dominance, not only increasing the host abundance, but also placing it in the coastal environment that the fungus thrives in. Also, current climate trends are warmer and wetter making conditions more favorable for fungal growth. As a result of these factors, fungal abundance has also grown, causing the current SNC epidemic in the Pacific Northwest.

During the time the northwestern United States was being colonized, old-growth forests were increasingly cleared for settlement and timber production. Larger scale clearcuts became increasingly common as logging technology and new machinery progressed. Around the 1950s, a new type of forestry developed in which stands were logged in shorter rotations and replanted with monocultures (single species) known to grow quality wood more quickly. The extent of Douglas-fir in the forests of the outer coast grew from scattered, rare individuals to the dominant species on the landscape. This human intervention provided unprecedented access to the "host" aspect of the disease triangle.

More recently, climate change has emerged as an all-encompassing effect on the environment. No longer a debate, scientists agree that Earth's climate is warming due to anthropogenic effects. While different parts of the world are experiencing these changes in different ways, there is now a new "normal," or rather a lack thereof. The Pacific Northwest has already experienced a regionally averaged warming of about 1.3° F, with a projected increase in average annual temperature of 3.3° F to 9.7° F by 2070 to 2099 (Mote and Snover 2014). While precipitation trends have both increased and decreased depending on location, season, and time period of analysis, most models agree that the Pacific Northwest will see increased precipitation during spring, winter (up to 20%), and fall with decreased precipitation during summer (Walsh et al. 2014). These seasonal changes of warmer and wetter coincide with the life cycle of *P. gaeumannii*, whose spore deposition and initial infection occurs in spring and pseudothecial proliferation occurs in winter. Anthropogenic climate change clearly represents a human impact on the "environment" aspect of the disease triangle.

Searching for climate correlations

It is understood that environment plays a role in virtually all forest diseases, but it can be more difficult to pin down specific variables that have the strongest effects. While warm and wet are very general environmental conditions, they can be further delineated into variables such as continentality (the difference between warmest and coldest days), heat-moisture index, and frost-free period. By identifying which variable or combination of variables are most strongly correlated with severe levels of SNC, a more specific set of environmental conditions that foster disease growth can be identified.

This type of analysis has been completed in Oregon and New Zealand, but not in Washington. Due to subtle changes in environment having potentially large impacts on fungal growth, it is important to analyze different geographic areas separately. Although Oregon and Washington are geographically close and have similar climates, they are not identical. This research will first answer the question: What is the incidence and severity of Swiss needle cast in Washington? The first step in understanding any disease is getting an accurate measure of where it is occurring, the level of infection, and preferably, how it is changing over time. The Washington Department of Natural Resources (WA DNR) completed these surveys in 2011 and 2012, and the 2015 survey was included in this graduate research. The second research question is: What climate variables are most correlated with disease severity? Models developed in Oregon and New Zealand were tested with the data from Washington to see if similar variables could be identified as being related to disease. From this, models could be created to guide land managers to areas that are suitable for Douglas-fir plantations. While they are a preferred species, some areas may be too high-risk to grow healthy (and profitable) stands of Douglas-fir.

Methods

The objective of this thesis was to further examine SNC in western Washington through two means. First, the incidence and severity of the disease was quantified by collecting needle samples from 47 sites throughout western Washington during April and May of 2015. Data from surveys completed in 2011 and 2012 was also included in the analysis to further our understanding of how the disease is (or is not) spreading or intensifying over time. Second, an analysis to find correlation between disease severity and climate variables was performed in order to understand the specific conditions under which *P. gaeumannii* is most active in Washington. A range of environmental variables such as average winter temperature and spring precipitation were calculated for each sample site through the software, ClimateWNA, and linear regressions were run in JMP with the colonization indices of each site.

Field assessments

The study area consisted of 47 sites throughout western Washington (Fig. 7, Appendix A). This area covered the entire length of the state north to south (46.19° N to 48.21° N latitude) and ranged from the coast to Capitol Forest (123.22° W to 124.63° W longitude). According to the Western Regional Climate Center (2016), this "rainforest" area west of the Olympic Mountains receives the heaviest precipitation in the continental United States. Annual precipitation is 178 to 254 cm (70 to 100 inches) in the Coastal Plains and more than 381 cm (150 inches) along the western slopes of the Olympic Mountains. Average maximum temperature in July is 21° C (70° F) along the coast and 24° C (75° F) in the foothills. Minimum temperatures in January range from 0° to 3° C (32° to 38° F), with warmer areas near the coast (WRCC 2016).

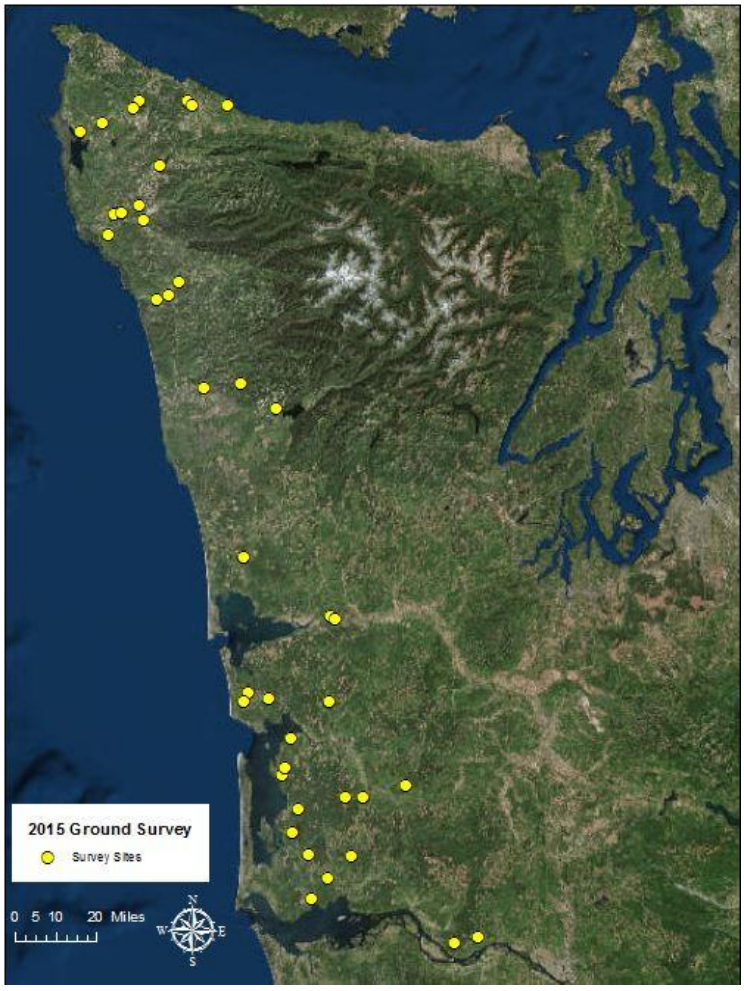


Figure 7. Map of 2015 ground survey sites in western Washington.

Sites were chosen based on previous years' survey locations. When possible, the same site was visited; however, some sites were inaccessible due to locked gates, construction, or logging activity. Additionally, the trees at some sites had grown too tall to enable sampling with pole pruners. In these instances, a nearby similar, but shorter stand was selected as a replacement. Most stands are part of large timber company holdings where the trees are harvested in varying rotations, enabling this ease of

substitution. Sites were located in Douglas-fir plantations of the same age, approximately 10 to 15 years old, primarily due to previously mentioned logistics, but also to minimize variation in canopy condition.

Transects were run at each location to collect stand data. Each transect was 75 m beginning approximately 15 m into the stand from the road and consisted of five points spaced 15 m apart. At each point, one dominant or codominant Douglas-fir was selected to be sampled from alternate sides of the transect, equaling a total of ten trees sampled at each site (Fig. 8). Field data was recorded with an electronic tablet. For consistency with previous surveys, initial samples were collected with the help of one of the DNR forest pathologists who completed the 2011 and 2012 surveys.

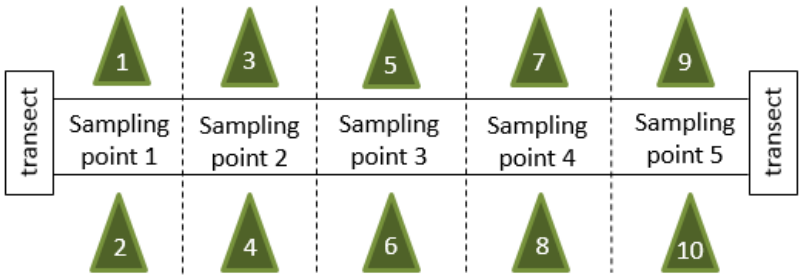


Figure 8. Layout of the 75 m transect used at each site for sampling.

Site conditions were also documented along each transect. These conditions included landscape position, elevation, aspect, stand color, average tree age, stand density, and species composition. Landscape position (coast, valley, ridge top, etc.), elevation, aspect, and coordinates were recorded. The stand as a whole was assigned a

number for the crown color, or degree of discoloration, ranging from 1 (normal green) to 4 (severe yellow or yellow-brown). Average tree age was determined based on the number of whorls on the Douglas-firs. At the second and fourth sampling points along each transect, 40 m^2 fixed radius plots were used to calculate stand density by counting the number of trees in the plot and tree species composition by counting the number of each tree species of tree.

Individual trees were also evaluated for needle retention and crown color. In most cases, unless not clearly visible or in some way abnormal, the fifth whorl from the top of the tree was examined. The secondary laterals (coming off the side of the main branch) were examined for each of the past four years of growth to estimate the percentage of needles remaining on the branch. A 0 to 9 scale was used, in which 0 represented 0 to 10 percent of needles present and 9 represented 90 to 100 percent of needles present. The fifth whorl was also evaluated for crown color using the same scale used at the stand level. One-year-old and two-year-old foliar samples were collected from the fifth whorl with pole pruners and stored in paper bags (Fig. 9). Tree diameter at breast height (DBH; 1.3 m) was also measured.

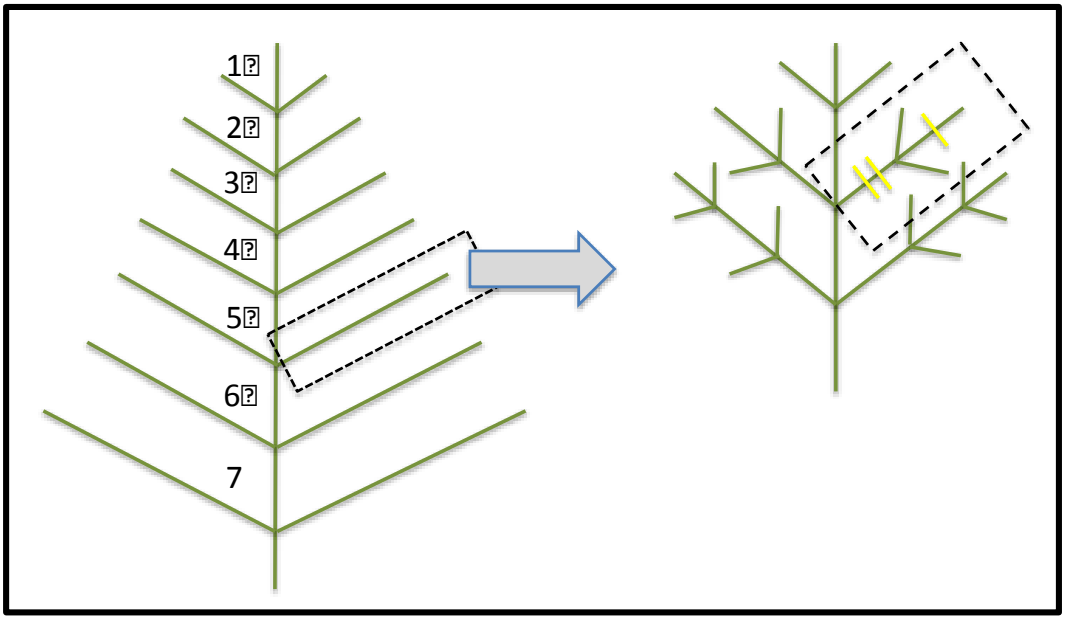


Figure 9. Location of branch sampled: fifth whorl from the top of the tree (left) and first and second cohort from a secondary lateral branch (right). Figure adapted from Temel et al. 2004.

Quantifying SNC severity

Branch samples were stored in paper bags in a refrigerator until processed, for a maximum of 3 to 4 days. Tweezers were used to carefully remove 15 to 20 needles from each branch and then 10 were selected that were relatively untwisted, whole, and of similar length. Those needles were arranged onto an index card with double-sided tape, ready for microscopic examination.

With the microscope adjusted to view approximately 50 stomata in each column, incidence and severity was recorded (Fig. 10). Incidence, the presence or absence of pseudothecia (fungal fruiting body), was recorded as 1 (present) or 0 (absent) by scanning the entire needle. Next, the basal, middle, and upper thirds of each needle were examined to count the number of pseudothecia present to measure severity. Within each third, 50

stomates were counted in one row on each side of the midrib for a total of 300 stomates counted per needle.

Figure 10. Microscopic view of pseudothecia on the underside of a Douglas-fir needle. Photo by Sue Hagle Service Archive.

Climate Data

Climate data was calculated through the software ClimateWNA v5.21 (Wang et al. 2012), from the University of British Columbia's Centre for Forest Conservation Genetics. The program extracts and downscales PRISM (Daly et al. 2008) generated monthly data (2.5 x 2.5 arcmin) and calculates annual, seasonal, and monthly climate variables for specific locations by latitude and longitude input. The output is 23 annual, 56 seasonal, and 168 monthly climate variables (Table 1). In the output for these 47 sites, most of the solar radiation (RAD) values were incorrectly reported as 0, so that

variable, along with any others that included RAD as part of their calculation (Hargreaves reference evaporation and Hargreaves climatic moisture deficit) were eliminated for this analysis. Additionally, while Climate WNA reported data for winter, spring, summer, and fall of the year entered (i.e. 2013 for the two-year-old needles collected in 2015), the life cycle of *P. gaeumannii* involves spring infection so the winter (2013) data initially reported was substituted with the following winter (2014) to align with the fungal life cycle.

Table 1. ClimateWNA variables tested for correlation with disease severity.

1) Annual variables:	
MAT	mean annual temperature (°C)
MWMT	mean warmest month temperature (°C)
MCMT	mean coldest month temperature (°C)
TD	temperature difference between MWMT and MCMT, or continentality (°C)
MAP	mean annual precipitation (mm)
MSP	mean annual summer (May to Sept.) precipitation (mm)
AHM	annual heat-moisture index ((MAT+10)/(MAP/1000))
SHM	summer heat-moisture index ((MWMT)/(MSP/1000))
DD<0	degree-days below 0°C, chilling degree-days
DD>5	degree-days above 5°C, growing degree-days
DD<18	degree-days below 18°C, heating degree-days
DD>18	degree-days above 18°C, cooling degree-days
NFFD	the number of frost-free days
FFP	frost-free period
bFFP	the day of the year on which FFP begins
eFFP	the day of the year on which FFP ends
PAS	precipitation as snow (mm) between August in previous year and July in
RH	mean annual relative humidity (%)
2) Seasonal variables:	
Seasons:	
Winter (_wt)	Dec. (prev. yr) - Feb for annual, Jan, Feb, Dec for normals
Spring (_sp)	March, April and May
Summer (_sm)	June, July and August
Autumn (_at)	September, October and November
Tave_wt, sp, sm, at	winter, spring, summer, autumn mean temperature (°C)
Tmax_wt, sp, sm, at	winter, spring, summer, autumn mean maximum temperature (°C)
Tmin_wt, sp, sm, at	winter, spring, summer, autumn mean minimum temperature (°C)
PPT_wt , sp, sm, at	winter, spring, summer, autumn precipitation (mm)
DD_0_wt, sp, sm, at	winter, spring, summer, autumn degree-days below 0°C
DD5_wt, sp, sm, at	winter, spring, summer, autumn degree-days below 5°C
DD_18_wt. sp, sm, at	winter, spring, summer, autumn degree-days below 18°C
DD18_wt, sp, sm, at	winter, spring, summer, autumn degree-days above 18°C
NFFD_wt, sp, sm, at	winter, spring, summer, autumn number of frost-free days
PAS_wt, sp, sm, at	winter, spring, summer, autumn precipitation as snow (mm)
RH_wt, sp, sm, at	winter, spring, summer, autumn relative humidity (%)
3) Monthly variables	
Tave01 – Tave12	January - December mean temperatures (°C)
TMX01 – TMX12	January - December maximum mean temperatures (°C)
TMN01 – TMN12	January - December minimum mean temperatures (°C)

PPT01 – PPT12	January - December precipitation (mm)
DD_0_01 – DD_0_12	January - December degree-days below 0°C
DD5_01 – DD5_12	January - December degree-days above 5°C
DD_18_01 – DD_18_12	January - December degree-days below 18°C
DD18_01 – DD18_12	January - December degree-days above 18°C
NFFD01 – NFFD12	January - December number of frost-free days
PAS01 – PAS12	January – December precipitation as snow (mm)
RH01 – RH12	January – December relative humidity (%)

Statistical Analysis

Summary statistics of mean, median, and range for each one-year-old and two-year-old cohort were calculated in Excel. A colonization index (CI) was also calculated for each site's two cohorts. The CI was calculated by multiplying the incidence and severity in order to reach one number to represent the level of disease at that site for each cohort. While severity alone is commonly used as the measure for disease, it does not take into account varying levels of incidence. Since most Douglas-fir needles have SNC, incidence is commonly rated at 1 (as opposed to 0 for no visible pseudothecia); however, a site may have some needles that don't exhibit signs of the disease. This is an important piece of information that should be factored into the overall disease measure, which the colonization index does.

Starting with separated cohorts, the percentage of needles with visible pseudothecia (incidence, $n = 10$) was multiplied by the average proportion of stomata occluded (pseudothecial density, $n = 10$) for the initial CI. Initial CI was then normalized within each cohort by taking the maximum CI value and calculating all other values as a percentage of that maximum.

Correlation analysis between CI and climate variables was conducted on the 2012 needle survey data, consisting of 70 sample sites. Appropriate data from Climate WNA was not available for the 2015 collection season. The one-year-old needles collected in 2015 needed climate data from March-December 2014 and January-February 2015, however Climate WNA did not yet have downscaled PRISM data from 2015.

Statistical analysis was performed with the software, JMP Pro 12.1.0 64-bit (SAS Institute Inc. 2015). CI values were tested for normality by examining the distribution with a Shapiro Wilk W test for goodness-of-fit. Pearson correlation coefficients were calculated for CI and monthly, seasonal, and annual climate variables. Finally, linear regressions were run to test different combinations of variables as well as with models found in previous research for comparison.

Results

Direct quantification of SNC severity

Results from the 2015 SNC ground survey indicate that the average colonization index (CI) is 5.3 in one-year-old needles and 22.5 in two-year-old needles. The disease was present at all 47 sites sampled, reinforcing the ubiquitous nature of *Phaeocryptopus gaeumannii*. There was strong correlation ($r = 0.70$) between the two cohorts, as expected. The sites sampled throughout western Washington also showed a considerable range in disease levels (Fig. 11). One-year-old CI values showed a weak normal distribution after a square root transformation while two-year-old CI values were normally distributed (Fig. 12). Based on this normal distribution plus the fact that two-year-old needles are a better indicator of disease and typically show a stronger correlation

with climate data (Stone, personal communication 2015), this cohort was primarily used in the subsequent climate analysis.

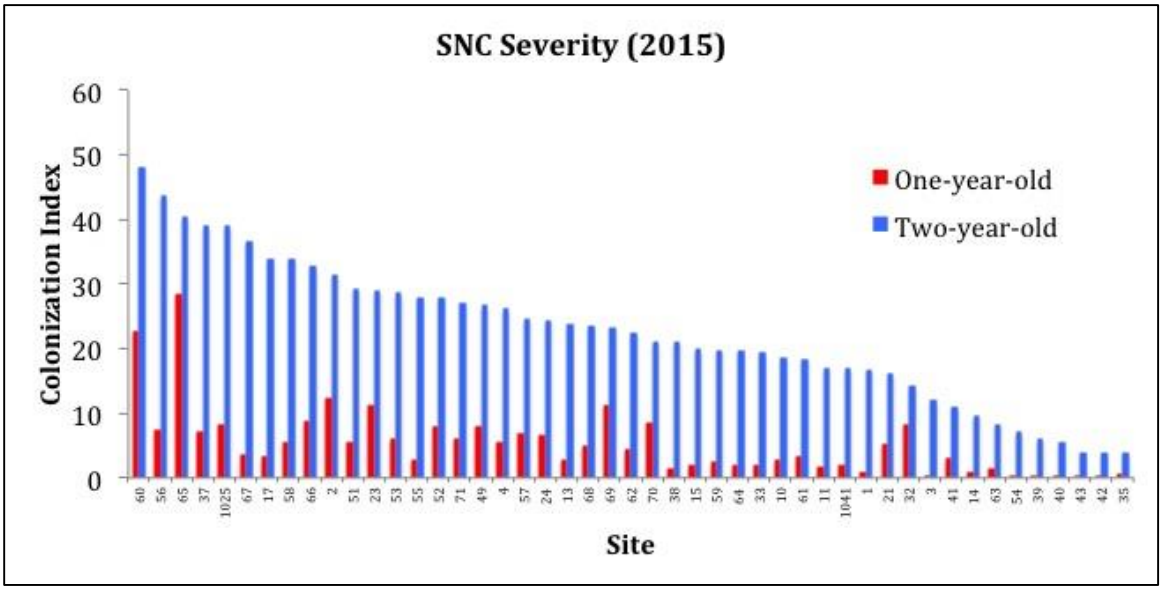


Figure 11. SNC colonization index by site sampled in the spring of 2015 in western Washington. One-year-old needle values ranged from 0.0 to 28.5 and two-year-old needle values ranged from 3.8 to 48.2.

(a) (b)

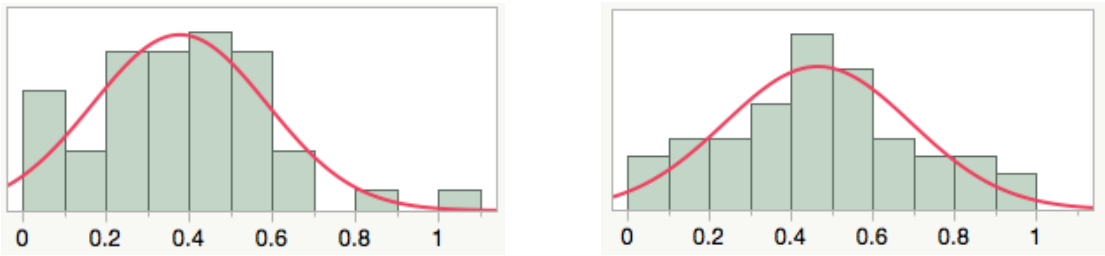


Figure 12. Distribution of normalized CI values for (a) one-year-old needles after square root transformation (Prob<W 0.1202) and (b) two-year-old needles (Prob<W 0.5613).

In both cohorts, this represents an increase since the 2012 survey (Tables 2 and 3). Severity has more steadily increased in one-year-old needles, with a decrease in two-year-old needles from 2011 to 2012. The disease appears to be worse in 2015 than in either year previously surveyed (Fig. 13). The colonization index does dampen the extremes of severity that can be seen when observing only percentage of stomata occluded without factoring in lower levels of incidence. Severity by percent occlusion shows high values of 48.1 for one-year-old needles and 69.8 for two-year-old needles.

Table 2. Summary statistics of colonization index for one-year-old needles.

One-Year-Old Colonization Index

	2011	**2012**	**2015**
Average	1.7	3.3	5.3
Median	1.2	2.6	3.8
Low	0.0	0.0	0.0
High	7.4	29.3	28.5

Table 3. Summary statistics of colonization index for two-year-old needles.

Two-Year-Old Colonization Index

	2011	**2012**	**2015**
Average	20.2	15.8	22.5
Median	20.0	15.6	22.45
Low	5.0	1.2	3.8
High	39.8	32.5	48.2

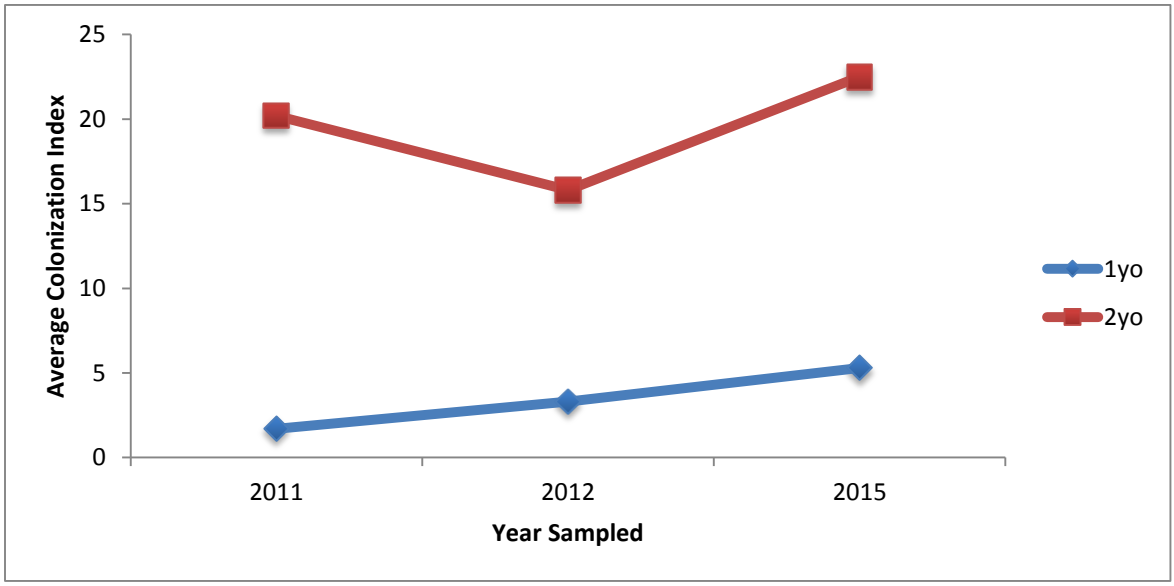


Figure 13. The trend in colonization index over the years of ground surveying in Washington: 2011, 2012, and 2015.

Disease levels may increase and/or decrease from year to year, but the relative ranking of sites by severity should be consistent. This is due to geographic and climate conditions at each site remaining the same. However, the three years of surveys in

Washington do not show this consistency in the 19 sites that were sampled each year (Figs. 14 and 15).

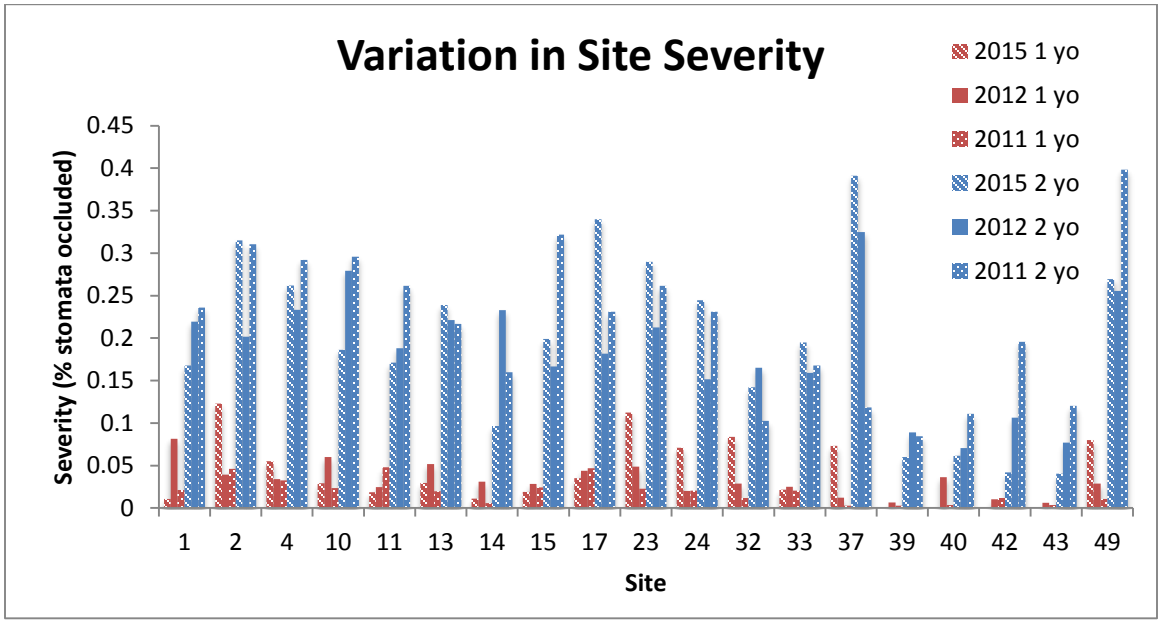


Figure 14. Variation in site severity across the 19 sites that were sampled during all three surveys (2011, 2012, and 2015).

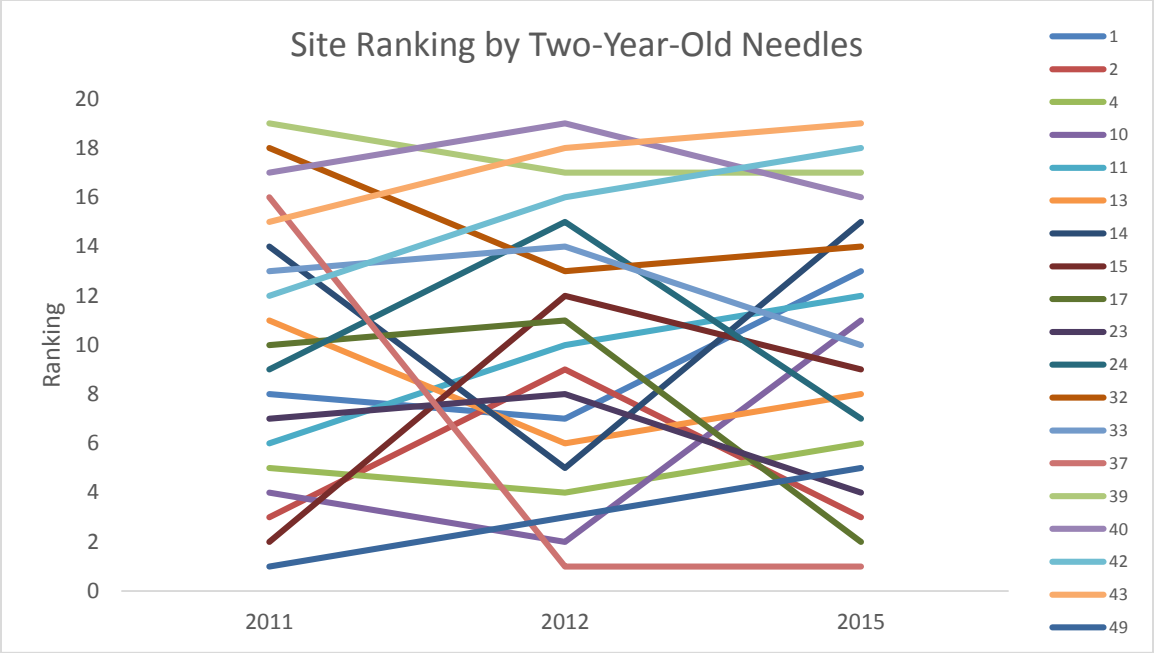


Figure 15. Ranking of site severity by two-year-old needles over the three years of surveying in Washington.

These survey sites are all within the Sitka spruce zone of coastal Washington (Fig. 16). As Douglas-fir is not the naturally dominant species in this area, monoculture plantations are a significant deviation from historical conditions. This research is not an experiment comparing SNC inside and outside the *Picea sitchensis* (PISI) zone; therefore, conclusions cannot be drawn regarding this effect, but it is an interesting part of the story.

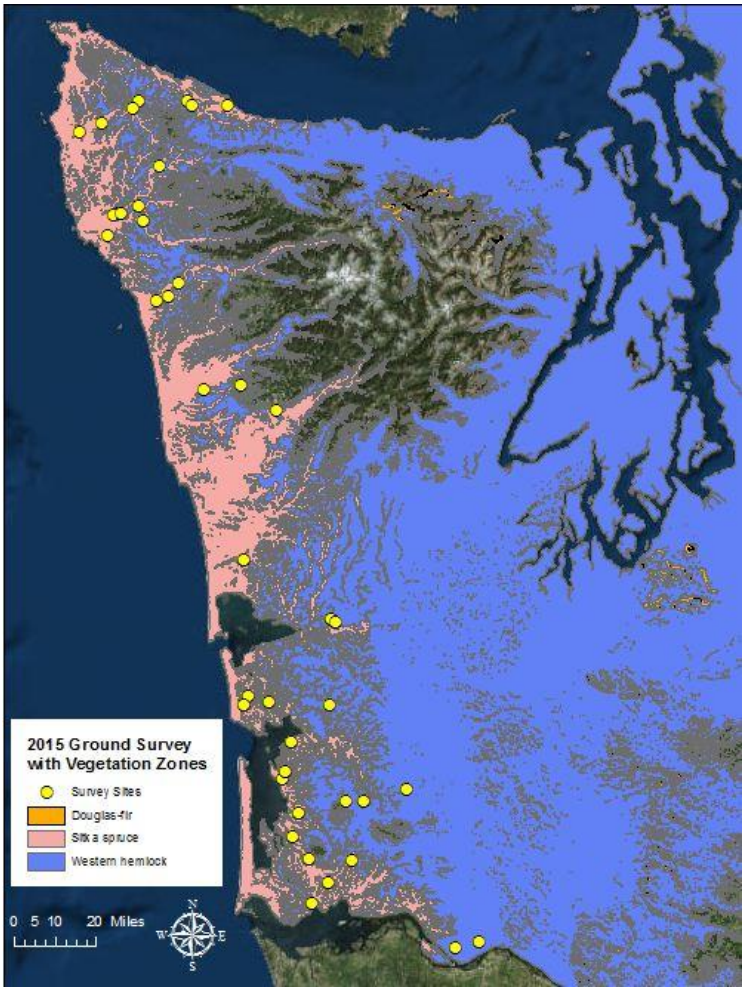


Figure 16. Map of 2015 ground survey sites over Sitka spruce forest zone.

Testing for correlation between disease severity and climate

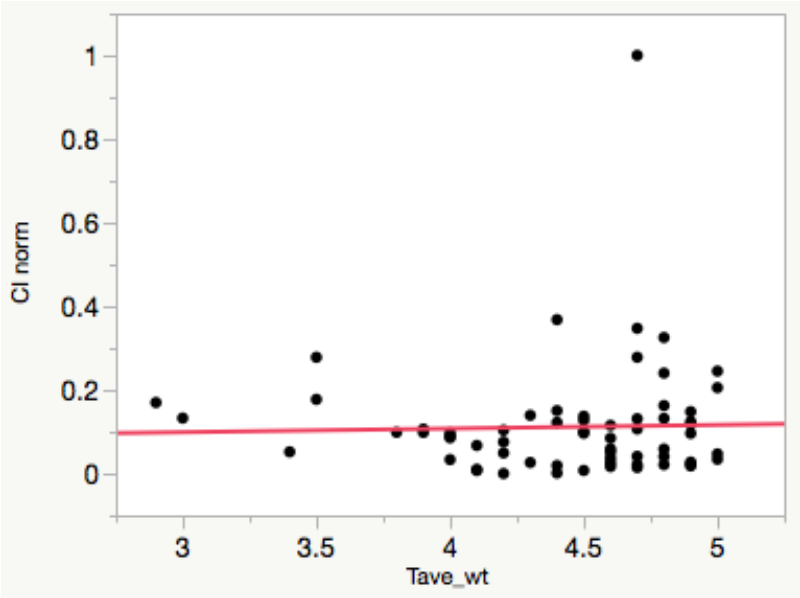
No correlation was found between CI values and climate variables produced by Climate WNA (Table 4). CI values were used for one-year-old, two-year-old, and combined cohorts. Climate variables included those at the monthly, seasonal, and annual scale. Various combinations of variables were also tested in multiple linear regressions with no significant R^2 values. To make comparisons with previous studies, CI was plotted with average winter temperature and showed an extremely low R^2 value (Fig. 17).

Additionally, in the one-year-old needles there was a small positive relationship while the two-year-old needles show a small negative relationship.

Table 4. Pearson correlation coefficient values between 2012 two-year-old CI and seasonal climate variables. Monthly and annual variables were also tested for and showed similar results.

	CI		**CI**		**CI**
Tmax_wt	-0.0253	DD_0_wt	0.0966	NFFD_wt	-0.1363
Tmax_sp	0.1032	DD_0_sp	-0.0563	NFFD_sp	0.0463
Tmax_sm	0.1332	DD_0_sm	0.0000	NFFD_sm	0.0940
Tmax_at	-0.0252	DD_0_at	0.1522	NFFD_at	-0.0745
Tmin_wt	-0.1302	DD5_wt	-0.0806	PAS_wt	0.0931
Tmin_sp	0.0380	DD5_sp	0.1360	PAS_sp	-0.1313
Tmin_sm	0.0895	DD5_sm	0.1771	PAS_sm	-0.0867
Tmin_at	-0.0926	DD5_at	-0.0408	PAS_at	-.0313
Tave_wt	-0.0861	DD_18_wt	0.0906	RH_wt	-0.0354
Tave_sp	0.1272	DD_18_sp	-0.1231	RH_sp	-0.0603
Tave_sm	0.1819	DD_18_sm	-0.1702	RH_sm	-0.0811
Tave_at	-0.0540	DD_18_at	0.0822	RH_at	-0.0237
PPT_wt	-0.0739	DD18_wt	-0.1132		
PPT_sp	-0.0762	DD18_sp	0.1446		
PPT_sm	0.0364	DD18_sm	0.1718		
PPT_at	-0.1584	DD18_at	0.0672		

(a)



(b)

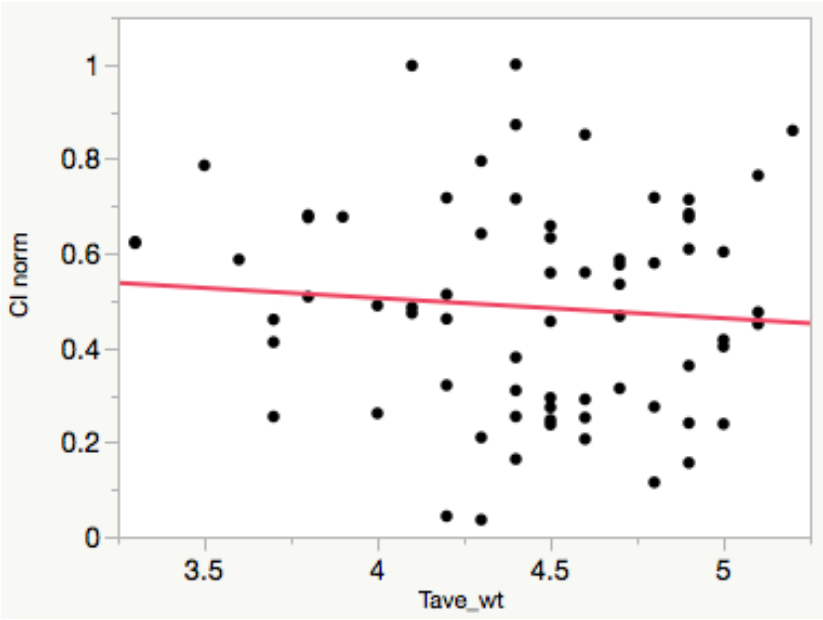


Figure 17. Relationship between colonization index (CI) and average winter temperature for (a) one-year-old needles (R^2 = 0.0008) and (b) two-year-old needles (R^2 = 0.007).

Very little range was predicted in CI values based on average winter temperature, although actual values varied significantly more (Fig. 18). Data sets from different years and different cohorts showed similar results. Additionally, removing the most severely diseased sites did not improve the correlation. In geographic terms, there does not appear to be any particular location in which high levels of disease were focused. The varying levels of severity appear to be evenly distributed throughout western Washington (Fig. 19).

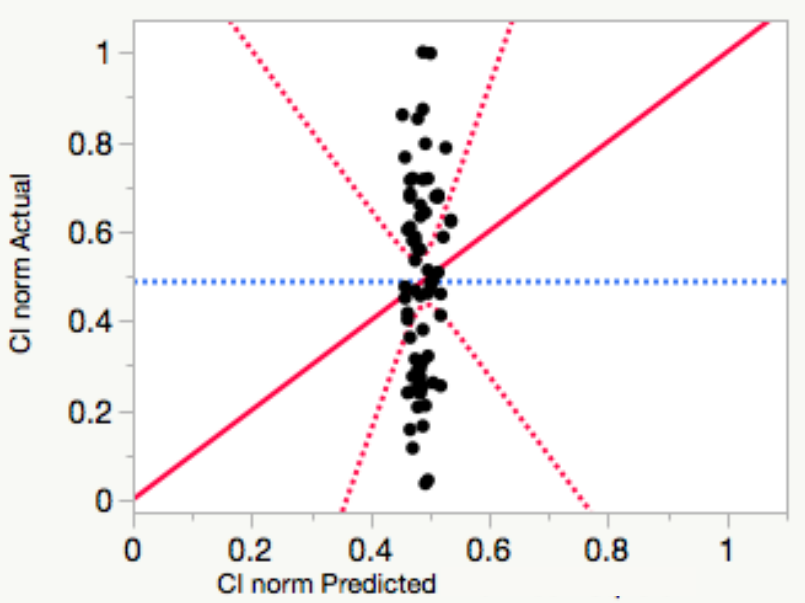


Figure 18. Relationship between actual and predicted colonization index based on average winter temperature for two-year-old needles sampled in 2012.

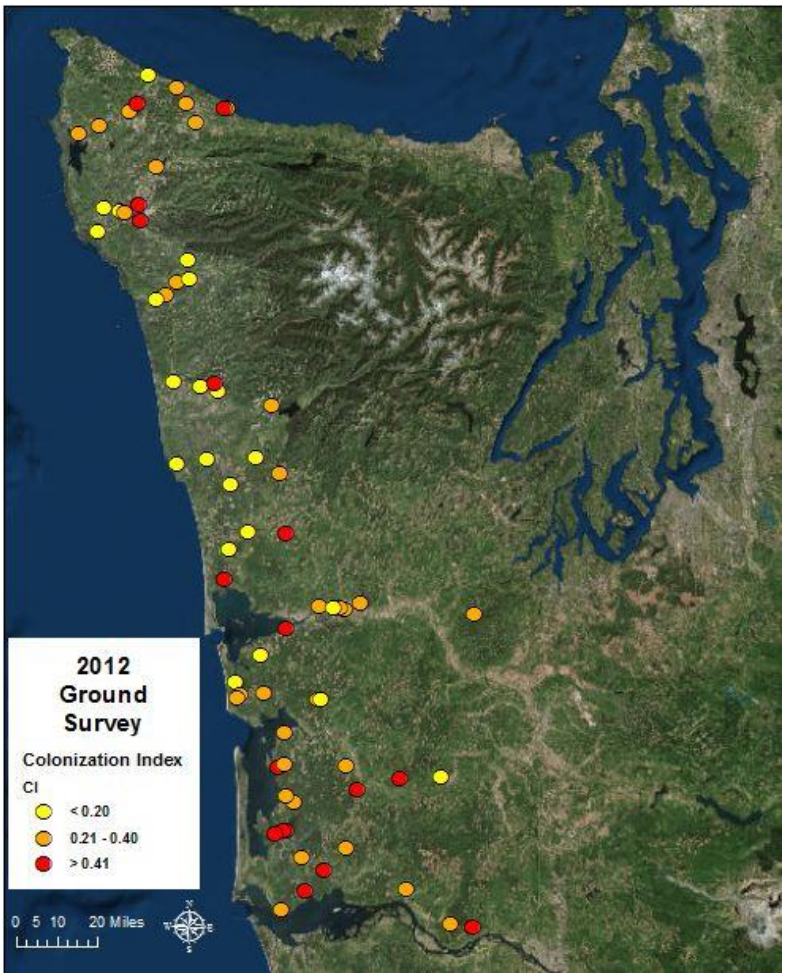


Figure 19. 2012 ground survey sites categorized by colonization index level.

Discussion

Direct quantification of SNC severity

Washington State has not had consistent ground surveying of SNC; however, a trend of gradual increase in disease severity since 2011 is clear. This parallels what is seen in aerial surveys which have found geographic spread to be increasing, as well. The Olympic Mountains are constraining the disease within the western portion of the Olympic Peninsula, but the lowlands in southwestern Washington are seeing an eastward spread of the disease. The total acreage reported with SNC symptoms increased by 53% between 2012 and 2015, which is significant in itself, but the acreage showing severe symptoms increased 184.9% (49.2% for moderate symptoms). While aerial surveys only represent estimates, it does indicate some level of increase in severity that corroborates the pseudothecia counts.

The high values of percent stomata occluded (48.1% and 69.8% for one- and two-year-old needles, respectively) are alarming when compared to previous research that has shown the greatest proportion of occluded stomata on attached needles was seldom above 50% (Hansen et al. 2000). Trees heavily affected by SNC rarely hold onto more than two years' worth of needles because, at that point, pseudothecia are sufficiently reducing stomatal conductance and CO_2 fixation to the point that the needles are carbon sinks instead of sources, so the needle falls off (Cannell and Morgan 1990, Manter et al. 2003a). From past research, it typically takes two years for needles to reach a 50% level of occlusion; however, in this study, some needles were already reaching that point in their first year. Although the average is not close to 50%, it is significant that some needles are reaching such a high level of severity so quickly. In all years surveyed, two-

year-old needles were found to be far above the 50% threshold, reaching a high of 79% in 2011, 65.4% in 2012, and 70% in 2015.

The beginning of the frost-free period has gotten significantly earlier over the course of the climate data used in this analysis. In 2009, the beginning of the frost-free period occurred on April 21st while in 2015, the frost-free period began on March 27th. As temperatures increase, budburst is occurring earlier causing WA DNR to move up the date of their aerial survey in 2015 to compensate for this change. Harrington and Gould (2015) projected Douglas-fir budburst to occur up to more than 60 days earlier by 2080. This change in Douglas-fir phenology is a benefit to *P. gaeumannii* as it allows more time to infect needles to higher levels.

Although it was not the focus of this study, foliage retention was estimated during the ground surveys to collect needle samples. Needle retention was quite low in the second cohort, with an average of 7.4 in 2012 and 6.9 in 2015. These low foliar retention rates in combination with the high levels of severity suggest that the disease may be having greater impacts in Washington State than in Oregon. Decreased foliage retention could also bias CI values, in that the most heavily infected needles have already abscised by the time of sampling so disease severity is estimated at lower levels than reality.

When looking at the nineteen sites sampled each of the three years, increases and decreases of severity are not consistent across the sites. It is clear that site severity is quite dynamic when looking at a line graph of site ranking. Monitoring in Oregon since 1996 has found a fairly constant ranking of sites, leading to the existing paradigm that

site-related factors determine severity (Hansen et al. 2000). However, the sites in Washington do not appear to follow this.

Testing for correlation between disease severity and climate

Boyce (1940) suggested that seasonal patterns in local climate might affect fungal growth in different ways, so it was expected to find similar or perhaps only slightly different correlation results than found in previous studies. The similarity of models for Oregon (Manter et al. 2005) and New Zealand (Stone et al. 2007a) suggests that winter temperature does indeed explain disease severity variation (Fig. 20). Both studies showed a strong relationship between the two variables ($R^2 = 0.847$ in Manter et al. 2005 and $R^2 = 0.75$ in Stone et al. 2007a). However, this study in Washington State found no correlation between colonization index and any climate variables at the monthly, seasonal, or annual scale.

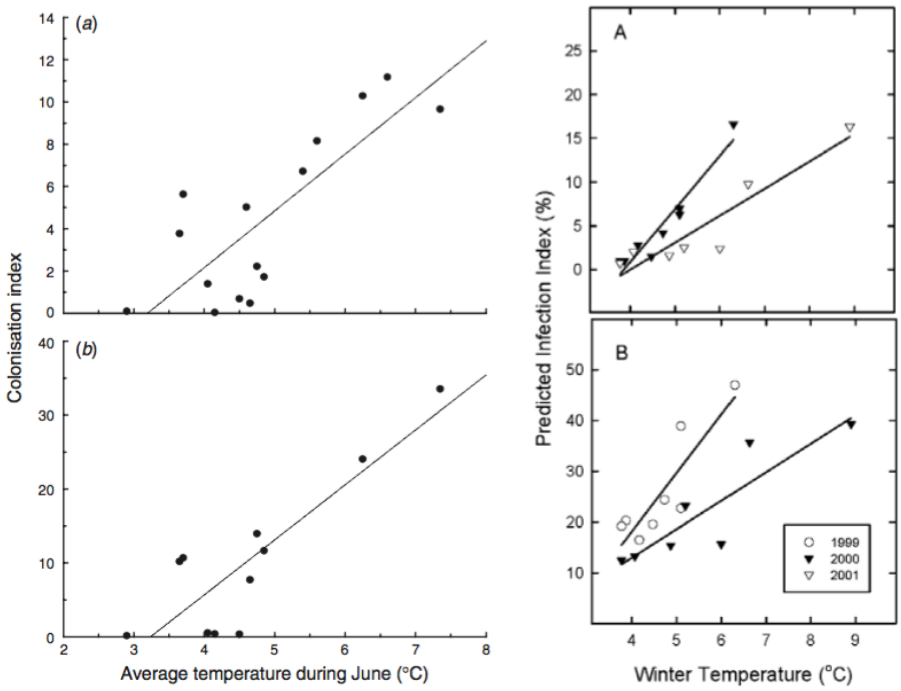


Figure 20. Relationship between colonization index and winter temperature in (a) New Zealand, where June is during the winter, $R^2 = 0.75$ for both (top) one-year-old and (bottom) two-year-old needles and (b) Oregon, where the relationship varied by year sampled but still showed a strong relationship in both (top) one-year-old and (bottom) two-year-old needles. From Stone et al. 2007a and Manter et al. 2005.

Regression showing actual CI by predicted CI shows much more variation in levels of disease actually seen than what was predicted by average winter temperature. Other variables showed a nearly identical graph. This minimal range in predicted values indicates that some other factor is influencing actual levels of disease outside the climate variables for which this study tested.

The sites sampled in Washington do not have a very large range in average winter temperature; only 2.1 to 5.3° C. In contrast, Manter et al. (2005) had a winter temperature range of approximately 4 to 9° C across only 9 sites while Stone et al. (2007)

winter temperatures ranged from approximately 3 to 7.5° C across 16 sites. Perhaps with more sites or a smaller range in temperature the correlation wouldn't be so strong.

In an ecological context, having Douglas-fir plantations covering a large amount of the Sitka spruce vegetation zone cannot be ignored as an influencing factor in the spread of SNC. The timber industry has primarily been replanting Douglas-fir trees following harvest for approximately 80 years resulting in an increasing amount of potential hosts for *P. gaeumannii*. More hosts mean more inoculum available for future years of infection allowing the cycle to feed itself.

Conclusion

While climate is likely playing some role in the increase in SNC severity and spread, this relationship is not clearly defined. Some studies have found a simple, strong relationship, but the data in Washington State indicates that a higher level of complexity in disease behavior is occurring. Due to uncertainties in how climate affects the disease and how climate change will likely bring new factors into play, it is difficult to predict the future of the SNC epidemic.

As these environmental factors play out, we as forest pathologists can observe and increase our understanding of the disease. Controlling the disease, however, is unlikely. In the meantime, perhaps the most practical action to be taken is to manage forests as responsibly as possible, even if that means planting less Douglas-fir in the coastal forest of western Washington.

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Appendix A - Site conditions from the 2012 ground survey

Site	Latitude	Longitude	Elevation	Landscape position	Avg tree age	Avg CI norm	Tave_wt	PPT_sp	MAT	MAP	NFFD	RH
1	46.20086	-123.22141	236.22	Ridge top	14	0.3480643	2.8	509	9.8	1719	278	67.6
2	46.18652	-123.30476	431.597	Ridge top	7	0.6545568	2.1	519	9.3	1728	273	69.7
3	46.39645	-123.66933	117.653	Upper slope	13	0.2486693	4.1	717	10.1	2273	294	70.2
4	46.29225	-123.81173	70.4088	Mid-low slope	11	0.5436756	4.7	619	10.3	1948	309	73.5
10	46.5962	-123.9165	.	.	.	0.3866035	5	544	10.2	1648	310	74.5
11	46.51114	-123.85731	43.2816	Coast	8	0.3553506	4.8	574	10.2	1754	307	73.3
13	46.56899	-123.47896	253.594	Ridge top	11	0.496577	3.4	661	9.7	2172	283	68.8
14	46.54113	-123.69298	75.5904	Lower slope	10	0.2007631	3.5	747	9.6	2287	285	69.3
15	46.7735	-123.75114	160.63	Ridge top	11	0.4130181	4	710	10	2190	293	70.5
17	46.77255	-124.05259	53.0352	Mid slope	10	0.7060629	4.8	562	10.1	1673	310	74.3
21	46.97814	-123.74339	11.8872	Coast	10	0.3372362	5	615	10.7	1853	311	72.3
23	46.54111	-123.62986	82.9056	Mid slope	12	0.6017165	4	647	10	2029	291	69.7
24	46.77888	-123.96409	123.139	Mid slope	7	0.5082767	4.3	659	9.9	1957	301	73.3
32	47.47782	-123.93646	.	Coast	12	0.2953294	4.3	1000	10.5	2979	304	72
33	47.1221	-124.05045	121.92	Coast	10	0.4050741	4.5	710	10.3	2083	308	72.7
35	47.7468	-124.31338	.	Coast	15	0.0789899	5.3	870	10.8	2472	316	73.2
36	47.7788	-124.27871	43.2816	Coast	10	0.8123981	5.2	917	10.9	2525	316	71.8
37	47.92403	-124.40505	.	Coast	9	0.4378711	5.3	883	11	2434	319	72.3
38	47.95956	-124.42002	.	Coast	10	0.1246808	5.3	848	10.7	2332	318	73.8
39	47.94328	-124.49361	.	Coast	12	0.1161684	5.3	840	10.7	2308	319	73.9
40	47.93893	-124.50938	.	Coast	6	0.2287314	5.3	855	10.8	2351	318	73.3
41	47.94072	-124.48324	.	Coast	10	0.0793776	5.3	831	10.6	2286	318	74.3
42	47.89113	-124.52851	.	Coast	10	0.084182	5.3	860	10.6	2429	317	74
43	47.73587	-124.35653	.	Coast	12	0.5541849	4.3	864	10.3	2475	310	74.2
49	48.2052	-124.41958	.	Coast	10	0.6083972	4.2	735	9.9	2280	300	72.5
51	46.40239	-123.81924	145.694	Upper slope	8	0.578613	4.4	638	10	1953	303	72.4
52	46.45435	-123.87709	97.536	Upper slope	7	0.593774	4.8	561	10.2	1702	308	73.9
53	46.61164	-123.90497	23.4696	Coast	11	0.1471801	4.5	619	10	1875	303	73.3
54	46.61164	-123.90493	73.7616	Mid slope	9	0.5797206	4.9	565	10.3	1711	309	73.3
55	46.68546	-123.88573	28.3464	Mid slope	8	0.9053721	2.7	787	9.2	2508	271	68.3
56	46.54111	-123.62986	355.397	Ridge top	6	0.5134744	4.6	597	10.1	1774	304	73.7
57	46.79568	-124.03685	94.7928	Ridge top	6	0.7035014	4.7	578	10.1	1719	308	74.2
58	46.77255	-124.05259	73.7616	Lower slope	7	0.4121873	3.9	737	10.1	2286	297	71.3
59	46.97814	-123.74339	343.205	Mid slope	7	1	5	611	10.7	1839	312	72.4
60	46.97814	-123.74339	0	Ridge top	6	0.3842189	4.8	639	10.7	1906	310	71.7
61	46.97319	-123.72729	0	Coast	9	0.4662549	5	649	10.6	1931	311	72.8
62	47.12212	-124.05042	28.0416	Coast	11	0.1744562	4.5	930	10.6	2847	305	71.1
63	47.538111	-124.06222	.	Bench	6	0.4090215	4.9	884	10.6	2548	311	72.3
64	47.52475	-124.18938	.	Coast	12	0.8405049	4.5	860	10.5	2417	310	73.1
65	48.05511	-124.34442	.	Coast	10	0.6801021	5.1	571	10.5	1754	320	76.5
66	48.19659	-124.10595	.	Coast	9	0.7588843	4.6	687	10.3	2038	314	75.3
67	48.20547	-124.24929	.	Coast	10	0.4876465	4.6	682	10.3	2023	311	75.1
68	48.195	-124.22991	.	Coast	10	0.483562	4.4	871	10.3	2485	310	74.2
69	48.18907	-124.44137	.	Ridge top	10	0.4392398	5.2	768	10.6	2153	323	77.5
70	48.13507	-124.62953	.	Coast	9	0.5644903	4.9	819	10.6	2318	319	75.3
71	48.15487	-124.55069	.	Coast	9	0.8103738	0.2	1216	7.9	3420	247	71
1041	46.34494	-123.75117	272.186	Ridge top	11	0.3509892	4	782	9.9	2453	296	71.4