United States
Department of
Agriculture

Forest
Service

North Central
Forest Experiment
Station

Research
Paper NC-275

Biology and Control of Scleroderris Canker in North America

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Inset, front cover: *Branch dieback on red pine caused by* G. abietina. *This symptom is usually most ovbious in early spring.*

Front cover: *Severe mortality in pole-size red pine in New York caused by the European strain of* G. abietina.

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1992 Folwell Avenue
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Manuscript approved for publication June 3, 1986
1986

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CONTENTS

BIOLOGY AND CONTROL OF SCLERODERRIS CANKER IN NORTH AMERICA

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During the 1930's when the Civilian Conservation Corps and the Work Projects Administration were involved in large-scale tree planting programs, pines were regarded as the most desirable species for reforestation. In the Lake States, red pine (*Pinus resinosa* Ait.) and jack pine (*P. banksiana* Lamb.) were the primary species planted. In the New England States the major species were red and eastern white pine (*P. strobus* L.). As a result of these planting programs, today we have 1.4 million hectares of red, jack, and eastern white pine in the Lake States and 1.3 million hectares of these three species in the New England States. These pine stands are potentially one of our most valuable conifer resources and any disease problem that threatens them would have serious economic implications. Scleroderris canker, caused by the fungus *Gremmeniella abietina* (Lagerb.) Morelet (= *Ascocalyx abietina* (Naumov) Schlaepfer-Bernhard), poses such a threat. In addition, the disease is also a possible threat to the 2.3 million hectares of ponderosa pine (*P. ponderosa* Laws.) and the 3.1 million hectares of lodgepole pine (*P. contorta* Dougl.) currently growing in the northern Rocky Mountain region.[1]

[1]*Forest statistics of the U.S., 1977 Review Draft. USDA Forest Service, North Central Forest Experiment Station.*

Two strains of the fungus, the North American and the European, are known in North America. The North American strain was not identified in the United States until 1964 (Ohman 1966), but evidence indicates that the disease had been present in Lake States stands since at least 1950. The disease, as observed from 1950 to 1964, was a problem of nurseries and young plantations. The causal fungus was spread on infected nursery stock throughout the northern Lake States and by 1965 was well established in National Forest plantations of red and jack pine in Michigan and Wisconsin. Approximately two-thirds of these plantations were infected with Scleroderris canker and losses from the disease were estimated at approximately 40 percent of the seedlings planted (Skilling and Cordell 1966). As serious as this damage was, trees that survived and reached 2 m or more in height were able to "outgrow" the disease; thus it was only considered a problem in nurseries and juvenile stands. The Forest Pathology Project of the North Central Forest Experiment Station (NCFES), developed a nursery fungicide spray program that now protects millions of conifer seedlings from infections each year. Since 1972 nursery managers have been producing healthy nursery stock for field planting. This has reduced the incidence of Scleroderris canker to manageable levels in northern Lake States pine plantations.

In 1975 Scleroderris canker was reported on red and Scotch pine (*P. sylvestris* L.) in New York State (Setliff *et al*. 1975). The symptoms observed, however, did not match those previously seen in the Lake States. Rather than infecting only young trees, Scleroderris canker killed trees of all ages from young seedlings to mature saw log trees. By 1977 Scleroderris canker was present in a 14,000-hectare area. In many 30- to 40-year-old red pine stands mortality was more than 90 percent and the remaining trees were heavily infected and had poor vigor. The disease showed the potential to move across northern New York State and kill large numbers of trees in all pine plantations infected (Miller and O'Brien 1977). In 1976 research by the NCFES and the Canadian Forestry Service determined that the fungus present in New York was different from that found in the Lake States but was serologically identical to fungus isolates found in Europe (Dorworth *et al*. 1977). The fungus was named the European strain of *Gremmeniella abietina* to distinguish it from the North American strain previously studied.

In 1976, research was begun on the European strain of Scleroderris canker in New York. Many of the problems encountered in this research were similar to those encountered with the North American strain. Therefore research work with the North American strain was used as a guide during the early research on the European strain.

The results of the research program in New York were summarized at an international symposium on Scleroderris canker in conifers held at Syracuse, New York, in June 1983. The proceedings of this symposium contains 40 papers on Scleroderris canker and represents the current status of the world's knowledge on this disease (Manion 1984).

TAXONOMY

The scientific nomenclature for the fungus causing Scleroderris canker has gone through a long series of changes dating back to the 1800's. When Van Vloten and Gremmen (1953) placed the fungus in the genus *Scleroderris*, it appeared the matter was settled, but discussion among taxonomists continued. Morelet (1969) developed the new genus *Gremmeniella* solely to accommodate this fungus, which became *G. abietina*. At about the same time in Switzerland Schlaepfer-Bernhard (1969) placed *Scleroderris lagerbergii* in the genus *Ascocalyx*. Müller and Dorworth (1983), recently published on the discomycetous genera *Ascocalyx* Naumov and *Gremmeniella* Morelet. They also support the name *Ascocalyx abietina* as the correct binomial for the causal organism of Scleroderris canker. For the present, however, we will continue to use the genus name *Gremmeniella*. The nomenclature for the imperfect stage of the fungus *Brunchorstia pinea* (Karst.) v. Hohn has remained stable and is still valid.

DISTRIBUTION OF *G. ABIETINA*

At the present time *G. abietina* is found in northern Europe, North America, and Japan. Since the original report in Norway (Brunchorst 1887), the disease has been reported in all the northern European countries. The most southerly point of distribution is in central Italy. In North America, damage from Scleroderris canker first became apparent in Michigan and Wisconsin in the 1950's. The causal fungus was identified in Canada in 1962. In 1964 Ohman identified the disease in red and jack pine plantations in northern Michigan (Ohman 1966). By 1966 the fungus was widely distributed across northern Wisconsin and the Upper Peninsula of Michigan. French and Silverborg (1967) reported Scleroderris in New York. By this time the disease was well distributed across Ontario and Quebec. Scleroderris was reported in New Brunswick (Magasi 1972), in Vermont (Tatter *et al*. 1973), Alberta (Dorworth 1975), and British Columbia (Hiratsuka and Funk 1976). Up to this time reports did not differentiate between the North American and the European strain. Based on later serological typing of sample material from this area, Smerlis' (1967) report of scleroderris on black spruce (*Picea mariana* (Mill.) B.S.P.) in Quebec probably involved the European strain. The Setliff *et al*. (1975) report in New York definitely described an occurrence of the European strain. Since 1975, a major research goal has been to determine the distribution of the European strain. The reasons for this were both its more serious impact on forest stands and its more limited distribution. Following the original report in New York and the identification of a different strain of *G. abietina* as the cause of this problem, a great deal of time and effort have gone into determining the distribution of the European strain in North America (fig. 1). Since 1976, the European strain has been found in 13 counties involving roughly 23,000 hectares in New York. In Vermont, 94 plantations in 9 counties were infected. One plantation infected with the European strain was found in Coos County, New Hampshire, in 1978. All trees in this plantation were destroyed and no additional infection sites have been found in New Hampshire. In Maine the North American strain was found in Franklin County in 1978 and the European strain was found in Hancock County in

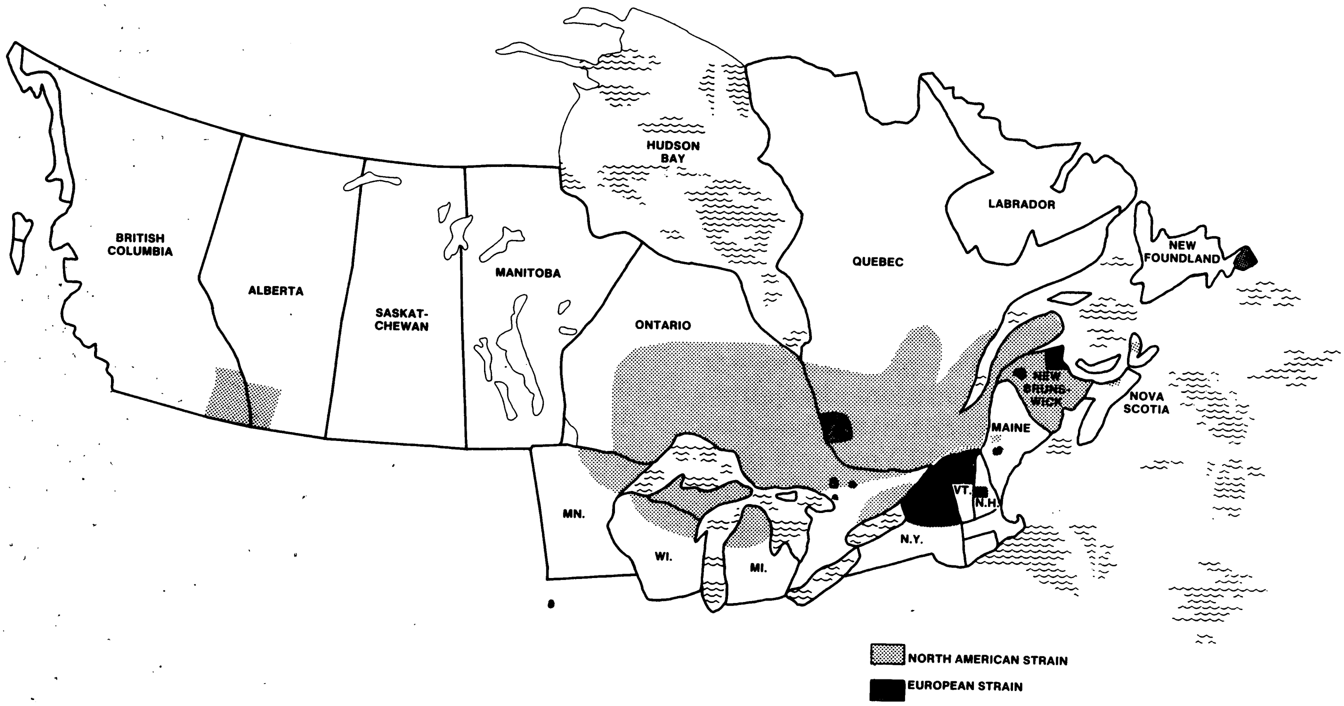

Figure 1.—Distribution of G. abietina *in North America, 1985.*

1980 and in Washington County in 1982. Infected branches were removed from the latter two plantations to prevent further disease buildup or spread. In Quebec the European strain was found in a 10-year-old red pine plantation near the New York border in 1978. This was the first positive record of the European strain in Canada. In 1979 personnel at the Laurention Forest Research Center found 19 additional plantations infected with the European strain. Infected branches were removed from these plantations, and the plantations have remained under surveillance for newly infected trees. The European strain was found in three plantations in Ontario in 1985.

Scleroderris canker is common in New Brunswick; about one-third of the jack pine plantations 4 years or older are infected (Magasi 1984). Most infections have been associated, however, with the North American strain. Isolates other than the North American strain of *G. abietina* have been identified serologically at nine locations in New Brunswick. Five samples were classified European race and four were classified as a race intermediate between the European and North American race. European race-like symptoms with large tree infection have not been observed in New Brunswick.

Scleroderris canker was present in a few plantations in Nova Scotia between 1972 and 1978. The race was never determined and this infection appears to have died out (Magasi 1984). No explanation is known for the disappearance of the disease in Nova Scotia, but some researchers have postulated that the fungus arrived on infected nursery stock but and was unable to maintain itself under Nova Scotia's climatic conditions.

The European strain of Scleroderris canker was first recorded near St. John's, Newfoundland, in 1979. In 1980 and 1981 the disease was detected at nine other nearby locations. Disease development and intensity of infection was spectacular in one plantation. Although a quarantine was established in 1980, the disease was detected outside of the quarantine area 60 km from St. John's in 1982 (Hudak and Singh 1984).

Except for the New York and Newfoundland areas, the damage in these newly discovered areas thus far is minimal. In most cases prompt detection and sanitization has prevented serious buildup. Research in New York during the last 6 years indicates that the disease requires a number of years to reach epidemic status. Except for a few areas in Newfoundland, epidemic levels are now present only in New York.

HOST RANGE

In the 1950's and 1960's, damage from Scleroderris canker in North America was found primarily in young red and jack pine plantations. Scotch pine was also attacked, but during the 1950's when the damage was occurring, few Scotch pine plantations had been established. A few eastern white pine were lightly infected in Ontario.

In inoculation tests in Wisconsin, no infection was obtained on any species of spruce or fir. These tests were all performed with the North American strain of *G. abietina*. Smerlis (1967) reported infection on white spruce (*P. glauca* (Moench) Voss) and black spruce by *G. abietina* in Quebec. This surprised investigators in the Lake States because they had been unable to infect these species. In 1978, serology tests of infected black spruce from the Quebec area where Smerlis had reported infection, revealed that these trees were infected with the European strain of *G. abietina*.

When research on the European strain was begun in New York in 1976, no information was available on the susceptibility of North American conifers to this strain. Field observations in northern New York indicated that red, jack, Scotch, and ponderosa pine were highly susceptible. Some resistance was seen in pole-size eastern white pine and white spruce showed only minor branch tip infection. Host range studies were begun in 1976 with a few species and were expanded to include most conifers native to the U.S. The objective of this study was to determine the susceptibility of North American conifers to the European strain of *G. abietina* when inoculated under field conditions.

Materials and Methods

Two sites in northern New York State were selected for the species resistance tests: the Boonville area, located 8 km NE of Boonville, New York and the Lake Clear site, located 14 km NW of Saranac Lake, New York. The European strain of *G. abietina* was present in both areas. When possible, 100 seedlings of each test species were planted in prepared transplant beds at each site. Each test plot was approximately 0.5 × 0.75 m. In most cases 2-0 seedlings were planted. In all tests infected red pine branches containing pycnidia of *G. abietina* were placed over the test trees in May or June to inoculate them. Infected branches were set on a vinyl-coated wire screen that was located 0.6 m above the ground. Inoculum remained over test seedlings throughout the year. Seedlings from 125 sources of 43 species were tested. Trees of most species were planted and tested in 2 successive years to determine an average infection rate at each site. One year after planting, the trees were rated for resistance on the basis of the number of infected seedlings. Fungus infection of seedlings was primarily determined by the presence of asexual fruiting bodies. If they were missing, isolations were made from necrotic tissue so we could identify the causal organism in the laboratory. Seedlings that were infected at the time of rating but later outgrew the infection were counted as infected. If the seedlings showed major differences in infection during the 2 years of testing, both infection ratings were given to show the range of infection.

Results

Infection rates differed widely for a given species between test sites and between years (tables 1 and 2). For example, in 1979 at Lake Clear only 8 percent of the Mugo pine (*P. mugo* Turra) seedlings were infected by *G. abietina*. In 1980 at the same site, however, the infection rate was 100 percent. Thus the potential for severe infection was present in 1979 but environmental conditions were not as favorable as in 1980.

G. abietina infected and produced conidia on all 16 pine species tested. Every species had more than 50 percent infection in at least one test plot. Species such as ponderosa, Monterey (*P. radiata* D. Don), red, and western white pine (*P. monticola* Dougl.) consistently had more than 90 percent seedling infection. Most of these infected seedlings died shortly after symptoms appeared. Infected Scotch and jack pine recovered somewhat although many of the recovered trees became re-infected and died during the second test year.

Within the genus *Picea*, Sitka spruce (*P. sitchensis* (Bong.) Carr.) was the most susceptible. In 1979 this species was 100 percent infected at both sites. One year later 74 percent of them died. Englemann spruce (*P. engelmannii* Parry) and Norway spruce (*P. abies* Karst.) were also severely infected in at least one test plot. The infected branch tips on seedlings of both species dropped off, however, and the trees recovered. White, red (*P. rubens*) Sarg. and black spruce showed only minor branch tip dieback; these seedlings also later recovered. Conidia of *G. abietina* were found on the dead branch tips of all three species.

Infected branch tips with conidia were found in all *Abies* species tested. Infection was light on all species except one plot of silver fir (*Abies amabil*

Table 1.—*Susceptibility of seedlings of the genera* Pinus *and* Picea *to European strain of* G. abietina

Species	Susceptibility rating[1]
Pinus spp. (pine)	3
P. virginiana (Virginia)	3
P. echinata (shortleaf)	3
P. thunbergii (Japanese black)	3
P. taeda (loblolly)	3
P. resinosa (red)	3
P. strobus (eastern white)	2-3
P. nigra (Austrian)	3
P. mugo (Mugo)	2-3
P. monticola (western white)	3
P. ponderosa (ponderosa)	3
P. lambertiana (sugar)	3
P. contorta (lodgepole)	3
P. banksiana (jack)	2-3
P. sylvestris (Scotch)	2-3
P. rigida (pitch)	3
P. radiata (Monterey)	3
Picea spp. (spruce)	
P. sitchensis (Sitka)	3
P. engelmannii (Engelmann)	3
P. abies (Norway)	2-3
P. glauca (white)	1-2
P. mariana (black)	1
P. pungens (blue)	2-3
P. rubens (red)	1

[1]Susceptibility rating is based on the number of seedlings infected 1 year after inoculation. Seedlings that later outgrew the infection were still counted as being infected. A dual rating such as 2-3 means that the average infection was 2 for 1 year but was 3 for the other year.

0 = immune; 1 = from 1 to 9 percent of seedlings infected;
2 = from 10 to 49 percent of seedlings infected;
3 = 50 percent or more of seedlings infected.

These ratings are based on 100 tree plots at two sites. The tests were repeated a second time with different trees so the total sample size was 400 trees for each species.

Table 2.—*Susceptibility of seedlings of the genera* Abies, Thuja, Tsuga, Larix, *and* Pseudotsuga *to European strain of* G. abietina

Species	Susceptibility rating[1]
Abies spp. (fir)	
A. balsamea (balsam)	0-1
A. fraseri (Fraser)	1-2
A. grandis (grand)	1
A. magnifica (California red)	1
A. procera (noble)	1
A. concolor (white)	1-2
A. amabilis (Pacific silver)	2
Thuja spp. (cedar)	
T. plicata (western red)	0
T. occidentalis (northern white)	0
Tsuga spp. (hemlock)	
T. heterophylla (western)	1-3
T. canadensis (eastern)	3
Pseudotsuga menziesii (Douglas-fir)	1-3
Larix spp. (larch)	
L. leptolepis (Japanese)	1
L. laricina (tamarack)	1
L. occidentalis (western)	3
L. gmelini dahurica (Mongolian)	3
L. sibirica (Siberian)	3
L. eurolepis (hybrid)	3
L. sudetica (Sudeten)	3

[1]Susceptibility rating is based on the number of seedlings infected 1 year after inoculation. Seedlings that later outgrew the infection were still counted as being infected. A dual rating such as 2-3 means that the average infection was 2 for 1 year but was 3 for the other year.

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(Dougl.) Forbes) where 65 percent of the test seedlings had infected tips. One year later all fir seedlings had overgrown the infection and appeared to be healthy. Douglas-fir (*Pseudotsuga menziesii* (Mirb.)) seedlings were moderately to heavily infected as measured by dead branch tips, but these seedlings also recovered from the infection. Both eastern hemlock (*Tsuga canadensis* (L.) Carr.) and western hemlock (*T. heterophylla* (Raf.) Sarg.) were heavily infected (90 + percent) in these tests but recovered within 1 year. Eastern hemlock seedlings growing wild near infected pines in northern New York were resistant to *G. abietina*. Northern white-cedar (*Thuja occidentalis* L.) and western red cedar (*T. plicata* D. Don) appeared to be immune to

G. abietina. Tamarack (*Larix laricina* (Du Roi) K. Koch) and Japanese larch (*L. leptolepis* (Sieb. & Zucc. Gord.)) were resistant with only a few branch tips infected. Other *Larix* species tested were susceptible.

Discussion and Conclusion

Based on the results of this work we know that the European strain of *G. abietina* is able to infect most of our North American conifers. The most serious damage will be to the hard pine species. Most of the infected hard pine seedlings eventually die. Sitka spruce also appears to be susceptible to the European strain and many of the infected spruce die. If the European strain of *G. abietina* spreads west into the natural range of Sitka spruce and ponderosa and lodgepole pine, it is possible that these three species could be severely damaged. No information is available, however, as to whether environmental conditions in the West would be favorable for infection by Scleroderris canker. Low precipitation and relative humidity could prevent epidemics from developing. Conditions necessary for Scleroderris canker infection are several days of cool, moist weather with high relative humidity when the fruiting bodies are mature (usually May to July). Whether these climatic conditions would develop often enough in the western States to support a Scleroderris canker epidemic is unknown. In the Lake States, the climatic conditions are similar to those found in the northeastern U.S. Our results indicate that the European strain will be very damaging in the Lake States, especially to red pine. No evidence of resistance was found in red pine seedlings from seed collected from 39 sources. Except for Sitka and perhaps Engelmann spruce, the *Picea* spp. appear to be resistant to *G. abietina*. Infection was light and seedlings that were infected usually recovered. Forest managers should be able to plant *Picea* spp. seedlings with little fear that Scleroderris canker will be a major problem. All the firs in this test were resistant and no problems should be experienced with this group. This is also true for Douglas-fir, eastern and western hemlock, eastern larch, northern white-cedar, and western red cedar. Although Japanese larch appears to be resistant, the European larch varieties were badly infected. Land managers should use caution with this group until more data are available.

SHORT DISTANCE SPORE DISSEMINATION

In a 2-year study of ascospore release of the North American strain of *G. abietina* in northern Michigan, ascospores were collected from April 9 to October 14 using Rotorod spore collectors.[2] The major spore release period was from May 15 to September 3 (Skilling 1969). Spore discharge and recovery were closely related to rainfall. During 1967, more than 96 percent of the spores were recovered within 24 hours after rain. No conidia of the asexual stage were collected by the Rotorod spore traps during the experiment. Conidia were collected, however, from May 17 to September 14 on vaseline slides placed 5 mm from infected branches. Therefore, the conidia were rain splashed from the pycnidia onto the slides but were probably not wind disseminated to the more distant Rotorod traps. Pycnidia collected at several areas in Michigan and Wisconsin during a 3-year period had viable conidia present from late April to mid-October with the maximum number present during early June.

No spore release information was available for conidia of the European strain. To determine at what time and in what numbers the conidia were present in infected plantations, we monitored spore release in two areas of New York during 1977 and 1978. These two areas were the Boonville and Lake Clear sites previously described for the host range studies. Ascospores of the perfect state of the European strain of *G. abietina* are rare in New York State, but they have been found in a few plantations. Apparently, conidia are primarily important in the spread of the fungus. Thus, the emphasis of our study was placed on characterizing conidial dispersal.

Materials and Methods

Precipitation was recorded weekly at both areas using standard U.S. Weather Bureau rain gauge. To trap *G. abietina* conidia, two vaseline-coated microscope slides were exposed at each site. A dead red pine branch with mature pycnidia of *G. abietina* was mounted on the trap approximately 5 mm from each slide. The slides were changed at weekly intervals and the number of conidia was determined by making three passes across each slide at 400X magnification. (This procedure has been described by Ost

[2]*Mention of trade names does not imply endorsement by the USDA Forest Service.*

and Nicholls 1982.) In 1977 the spore traps were operated from March 28 to November 21. Because spores were trapped during the first week of the study in 1977, spore dispersal could have commenced prior to the beginning of the study. As a result, the trapping period was expanded in 1978 to cover the entire year. Spore counts from the two areas were combined to give an average dispersal count for the northern New York region.

Results and Discussion

In 1977 and 1978 the major spore release was during May, June, and July. Peak release was in June in both years (fig. 2). Conidia were trapped in every month during 1978. The number of conidia released was not related to total weekly rainfall, but no conidia were collected during any week without rain (table 3). Spores were only released during the winter months when precipitation was as rain; no spores were released during snowfall. Apparently, condidia dispersal with the European strain is possible anytime free moisture is present and the temperature is above freezing.

In a separate study in New York, Luley and Manion (1984) used both petroleum-coated slides and monofilament spore traps to study spore dispersal. No ascospores were collected during the 17-week sampling period on either type of trap. The maximum number of conidia were collected from early

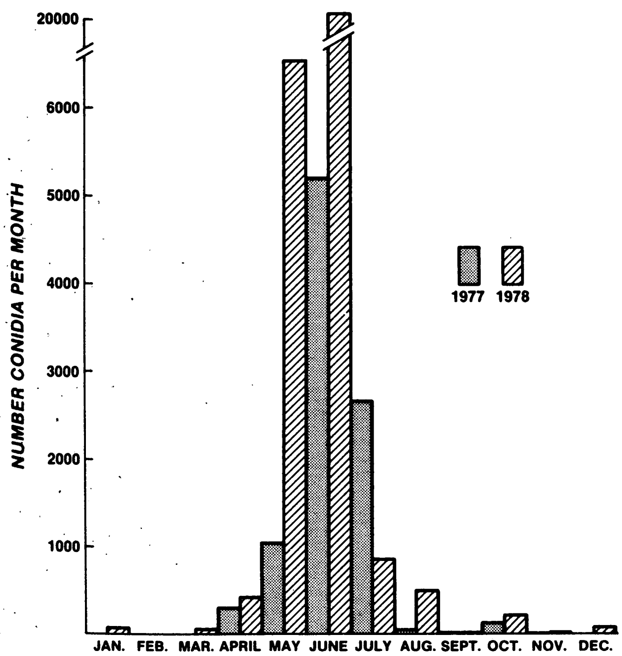

Figure 2.—Total conidia dispersal of G. abietina *at Lake Clear and Boonville, New York, 1977-1978.*

Table 3.—*Combined conidia dispersal of* G. abietina *at Lake Clear and Boonville, New York*

Month	1977		1978	
	Conida	**Rainfall**	**Conida**	**Rainfall**
	Ave. no.	*Ave. cm*	*Ave. no.*	*Ave. cm*
January	-	-	74	16.2
February	-	-	1	4.3
March	-	-	28	5.1
April	295	9.4	424	9.3
May	1,052	3.0	6,542	4.3
June	5,200	9.2	20,140	11.0
July	2,642	12.5	863	5.5
August	56	16.2	503	8.7
September	32	22.7	26	12.0
October	133	10.1	212	8.8
November	12	5.2	26	3.8
December	-	-	76	13.2

May through early June, which coincides with the period of elongation and development of red pine shoots. Conidia were trapped from ground level to 12 m above the ground with no significant difference in spore density at the different elevations. Red pine seedlings exposed to infection at the various heights were infected from the groundline to 12 m.

LONG DISTANCE SPORE DISSEMINATION

Conidia studies of the North American strain in Michigan indicated that conidia were only disseminated over short distances, such as within an individual tree, while ascospores were responsible for long distance fungus spread (Skilling 1969). Spore dispersal studies of the European strain in New York in 1977 and 1978 showed that conidia were rain-splash disseminated for short distances throughout the year. No information was available on long-distance movement of conidia in New York. Because the wind-blown ascospore stage of the European strain of *G. abietina* is rare in New York, the question of how the causal fungus moved so rapidly through New York plantations remains to be answered.

Materials and Methods

To determine the importance of the long distance movement of conidia of the European strain, a new spore-trapping procedure was used in 1979. Two areas were selected for this study. One was located on the Tug Hill plateau near Lowville, New York, and the second area was located near Saranac Lake, New York. These two sites each had a small infection area next to an open field with no other inoculum present. Vaseline-coated spore traps were placed from 0 to 610 m from the inoculum source.

Results and Discussion

The number of conidia collected at the spore trap sites dropped rapidly as distance from the inoculum source increased (table 4). Small numbers of conidia, however, were trapped at the 610 m trap indicating that at least some wind transport of conidia was possible.

The conidia of *G. abietina* are approximately the same size as the ascospores. If they become airborne, they should be able to move by air currents with about the same efficiency. The major difference between the two spore stages with respect to dispersal is that conidia are produced in spore tendrils that ooze out of the pycnidia during moist weather. These spore tentrils are probably diluted by rain, but the spores rely on wind or some other agent to propel them into air currents where they can be carried away. The ascospores, however, are forcefully expelled from the apothecium for a distance of up to 25 mm in calm air (Skilling 1968). This distance is sufficient for the spores to reach passing wind currents around the apothecium in the first step toward long-distance spread. This system explains why many more ascospores than conidia are collected in long-distance spore dissemination studies of the North American strain. In New York, however, even though the ascospores are not present in large numbers, enough conidia become airborne to spread the fungus from one plantation to another. Once airborne, these hyaline, 4-celled spores are durable. Zajechowski and Bergdahl (1983), sprayed conidia on living red pine foliage in the greenhouse and in the field. After 20 days spore viability was 27 percent on greenhouse seedlings while that of spores in the field was 49 percent on seedlings protected from rain but only 2 percent on unprotected seedlings. It appears that airborne conidia can spread the fungus to new areas if they land on a suitable target needle. The 30 to 60 km distances between certain old and new infection sites in New York and Quebec are probably the result of airborne conidial.

Table 4.—*Long-distance transport of* G. abietina *conidia in New York during 1979*

Area	Distance from inoculum source						Total
	0	61	122	305[1]	305	610	conidia[2]
	---------- *M* ----------						*Number*
Saranac lake	1,723	54	13	3	17	11	1,821
Tug	49	39	25	10	9	18	150

[1]Rotorod collection point.

[2]All conidia were collected on vaseline slides except for one rotorod collection point at 305 m.

Insects have also been considered as vectors of *G. abietina*. Frederick *et al.* (1976), were able to infect jack pine by placing conidia-bearing beetles on wounded seedlings. A study by Lanier *et al.* (1984), examined the role of the twig-infesting scolytid beetles *Pityophthorous pulicarius* and *P. puberulus* in infecting trees with Scleroderris canker. These beetles were often found breeding in twigs killed by *G. abietina*. Although these insects were exonerated from the role of suspected vectors of Scleroderris canker, a significantly reduced infection rate in re[d] pines treated with the systemic insecticide carbo furan indicated that insects may nonetheless exacer bate the disease outbreak by providing infectio courts for conidia.

INFECTION BIOLOGY

Primary infection for the North American stra is by windblown ascospores. These are formed fruit bodies on dead infected branches. The spor are disseminated during moist weather from April October although major infection is in June a July. Although most spores are disseminated onl few meters from the infected branch, many tin new infections have appeared several kilomet from any existing infections. The ascospores inf through buds or at the bases of needles in early su mer, and infected branch tips are usually dead following summer. According to Patton *et al.* (19 the spore germ tube infects shoots through stom on the abaxial surface of bracts that subtend sh shoots. The germ tube penetrates between gu cells, sparsely colonizes branch tissue, and reac the periderm that separates branch from stem tis by late summer or fall. The fungus crosses this p derm in early February when the host plant is mant. It then rapidly colonizes the short shoot surrounding cortical tissue. This invasion produ resinous brown necrotic area of cortical parench and phloem beneath the branch as the first vi symptom of infection. Sletten (1971), workin Norway with *P. sylvestris*, found that trees in lated with *G. abietina* during winter dorm showed symptoms the following spring and sum However, trees inoculated in the growing seas

1 year showed no symptoms until the growing season of the next year. Sletten's work agrees with studies at the North Central Forest Experiment Station in that most damage to the host plant by Scleroderris occurs while the host is dormant. Lang and Schütt (1974), in Germany also found that the mycelium remained under and between the leaf scales during the growing season, invading them in the autumn by penetrating the outer wall of the epidermis directly and growing through the periderm into the inner tissues of the shoots. Siepmann (1976) found that after inoculation with conidia in July 1974, hyphae were found in the epidermis and hypodermis of long shoots by the end of August and in the cortical parenchyma of the needle base by January 1975. Invasion of the bud scales of short shoot buds was observed in early December 1974, beginning in the dead cells of the blade margin and extending into the green tissue of the scales.

The long period of time between spore infection and the first visible symptoms frequently leads to the shipment of infected nursery stock. This is especially true with fall shipments because no visable symptoms are present in the fall.

Dorworth (1979), studied the effect of inoculum concentration on infection of red pine by the North American strain of *G. abietina*. Conidial suspensions were sprayed on red pine seedlings during June. A concentration of 2.5×10^8 conidia per square meter of seedbed was required to kill 50 percent of the seedlings.

The first symptom of infection by *G. abietina* is usually the dieback of buds and the discoloration at the base of needles on the previous season's growth (Inset front cover). Beginning at their base, infected needles turn orange during early May approximately 9 months after infection. By midsummer the needles are brown. These needles are loose and usually drop off leaving a bare branch tip. A few months after an infected branch dies, pycnidia appear near the base of dead needle fasicles. Asexual spores (conidia) ooze out of these pycnida during wet weather from April to October (fig. 3). As mentioned earlier, these spores are primarily transported by rain splash to nearby branches, but recent studies have shown that these conidia can also be transmitted long distances by the wind. The apothecial fruiting bodies of the North American strain appear in early summer on branches that have been dead for 1 or 2 years (fig. 4). The apothecia are also commonly found at the base of dead needle fasicles and are often found in association with the pycnidial stage (fig. 5).

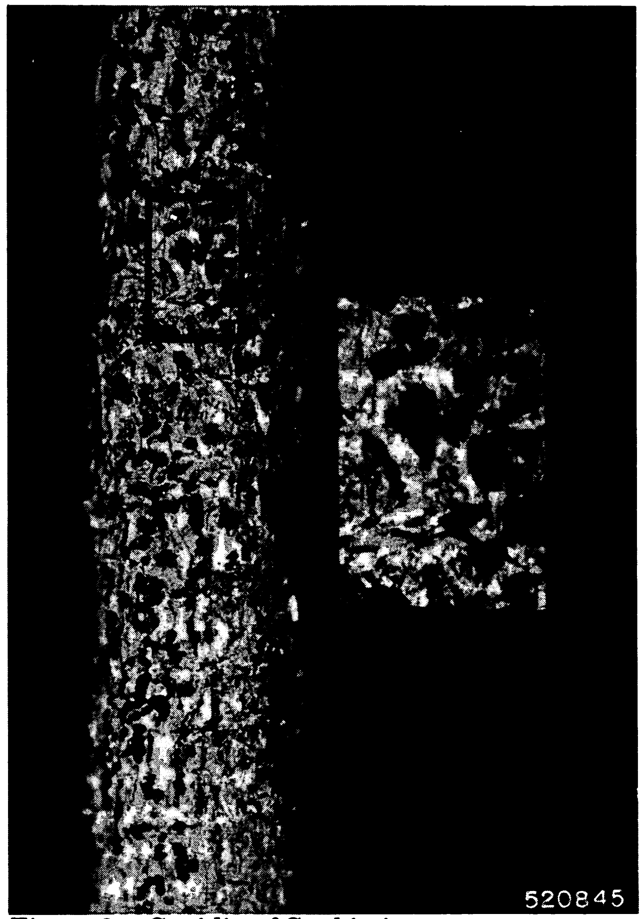

Figure 3.—Conidia of G. abietina.

After infection, the fungus may grow down the branch for a short distance until it reaches the main stem of the tree; young trees are girdled quickly and die within a few months. In many cases, however, the fungus only grows outward for 3 to 4 cm from the infection spot. At this point the fungus is unable to survive competition from saprophytic fungi. By this time, however, it has girdled the branch and the branch dies from the infection point to the tip. It is this dieback of branch tips that is most characteristic of Scleroderris canker and when multiple branches are killed, is the primary cause of tree death. Stem infections frequently form a canker, and it is this canker that gives the disease its name (fig. 6). This canker may eventually be overgrown but will usually deform the stem. Small cankers are common on branches infected with the European strain but are rarely found on branches infected with the North American strain. A characteristic yellow-green stain is commonly observed in the cambial zone of recently killed tissue.

The infection biology of the European strain is similar to the North American strain but with the following exceptions. The apothecia that produce windblown ascospores are rare, at least in New York

Figure 4.—Apothecia of G. abietina.

Figure 6.—Canker on red pine caused by G. abieti

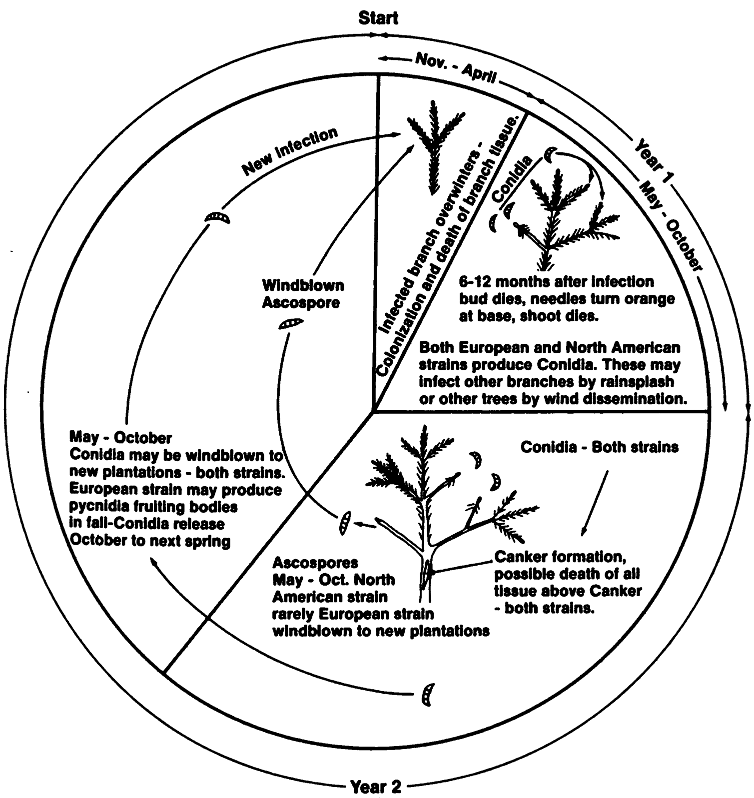

Figure 5.—Disease cycle of G. abie

State and in some areas in Europe. Most spread appears to be the result of wind and rainsplash movement of conidial. The European strain produces a second crop of pycnidia during the late fall in New York. Conidia from these fruiting bodies are sometimes released during wet winter periods when the temperature is above freezing.

During the winter of 1980-1981, potted red pine seedlings were exposed at Lake Clear, New York, to determine if infection could occur during the winter months from these conidia. During February 1981 the weather was unusually warm for several days. The mature pycnidia present released conidia during this period and exposed seedlings were infected (table 5).

The European strain, therefore, can release conidia during the winter months and these conidia are able to infect conifer seedlings if the weather is suitable. Temperature must be above freezing and sufficient rain must fall to liberate spores, but the needed duration of these conditions is not known.

SPREAD WITHIN STANDS

Since Scleroderris canker first appeared in North America, investigators have been trying to determine why epidemics develop within certain plantations. Early work by Skilling and Cordell (1966) showed that the highest infection rates were in plantations less than 5 years of age. Plantations more than 15 years old were lightly infected. Furthur examination revealed that infected nursery stock was being shipped to new plantations. Damage from Scleroderris canker was related to the amount of new infection that became established during the early life of the plantation. If a high percent of seedlings became infected during the first 3 or 4 years after planting, most of the seedlings died. If the trees became infected at a later period, the trees were too tall to be killed by the time the infection was distributed throughout the stand. The lower branches (up to 2 m above ground) would die but the rest of the tree would remain alive and few trees would be killed.

Table 5.—*Red pine seedling exposed to pycnidia of* G. abietina *at Lake Clear, New York*[1]

Period exposed to infection	Seedlings exposed	Seedlings infected	Seedlings infected
	------ *Number*	*------*	*Percent*
12/28/80 to 1/12/81	10	0	0
1/12/81 to 1/26/81	10	0	0
1/26/81 to 2/9/81	10	8	80
2/9/81 to 2/23/81	10	9	90
2/23/81 to 3/9/81	10	1	10
3/9/81 to 3/23/81	10	0	0

[1]Seedlings were only exposed to the inoculum for the test period shown. At the end of each exposure period the seedlings were removed to an area with no inoculum and observed in May 1981 for symptom development.

The situation with the European strain is different because the fungus has the ability to attack trees of all sizes. When the initial epidemic of Scleroderris canker was observed in New York State, disease incidence was so high that it appeared that most of the red pine stands would soon be killed. Many of these stands had a large amount of highly susceptible live lower branches available for rapid buildup of the pathogen. Within 2 years the branches on the lower 5 m of most red pine stands were killed by the fungus. Then, either because of less favorable climate for infection or because the upper branches were less susceptible, the upper crown infection advanced at a slower rate. Although thousands of trees were killed during the first infection wave (Front cover), surviving trees continued to grow even though their lower branches died. At this point information was needed to determine what the future disease impact would be on these remaining stands. It was important to know when stands would reach the critical stage where severe upper crown infection would kill the trees.

Materials and Methods

In 1976 the New York Department of Environmental Conservation established eight 20- by 30-m permanent study plots in Lewis County, New York. The purpose of these plots was to monitor the rate of upward spread of Scleroderris canker in older stands. All plots were located in 45-year-old stands—two plots were in Scotch pine and six plots were in red pine stands. At the time the plots were established, from 90 to 100 percent of trees in the stands were infected with Scleroderris canker. Each plot was measured annually. The height of each tree and the height of the highest infected branch in each tree were recorded.

Results and Discussion

We observed large differences in the amount of healthy crown from year to year in each of the eight

Table 6.—*The rate of vertical spread of Scleroderris canker in 45-year-old red and Scotch pine stands in Leu Co., New York*[1]

(In meters)

	Year	Plot 1	Plot 2	Plot 3	Plot 4	Plot 5	Plot 6[2]	Plot 7	Plot
Average	1976	16.7	14.9	16.7	14.7	15.2	11.2	14.9	14.
tree height	1980	18.2	16.4	18.4	16.2	16.4	13.2	16.6	16.
	1983	19.3	17.4	19.2	17.3	17.6	14.4	17.8	17.
Average	1976	10.6	9.8	11.9	9.3	9.3	5.8	10.1	10
infection	1980	13.9	11.6	15.4	11.8	11.7	7.8	11.8	12
height	1983	14.5	12.2	16.1	12.6	14.0	9.8	12.6	12
Healthy	1976	6.1	5.1	4.8	5.4	5.9	5.4	4.8	4
crown	1980	4.3	4.8	3.0	4.4	4.7	5.4	4.8	4
	1983	4.8	5.2	3.1	4.7	3.6	4.6	5.2	5

[1]Plots 3 and 5 are located in Scotch pine stands. All remaining plots are in red pine.
[2]Plot 6 was not measured in 1981.

sample plots as well as between plots (table 6). In 1980 moderate Scleroderris canker infection occurred throughout Lewis County. During this period the length of healthy crown was reduced in six of the eight sampled stands. An average of only 3 m of healthy crown was present on the trees in one of the Scotch pine plots in 1980. This is a small crown for trees averaging 18.4 m in height. In contrast, the years from 1980 to 1983 were light infection years for *G. abietina*. As a result the length of healthy crown on these same trees actually increased. Looking at the extent of disease development in the eight stands for the 7-year period we observed that total healthy crown length decreased in five stands and increased slightly in three stands. The decrease in healthy crown appears important in one of the Scotch pine plots (plot 5) but so far has had no measurable effect on tree height growth. No heavy infection years have been recorded since the plots were established, so we still do not know the final disease impact on these eight stands. The tree crowns appear to be getting smaller in general but we have observed no growth reduction or dead trees. If weather conditions are conducive to heavy infection for 2 or 3 successive years, we believe that many of the trees will die. Up to this time, however, the plot data do not support this conclusion and a final analysis must depend on additional data.

Carvin and Manion (1984) followed the development of Scleroderris canker on 13 plots located in 8 red pine stands in northern New York from 1978 to 1982. The trees in these stands were from 15 to 20 years old and from 3.7 to 5.9 m tall. During the first 2 years of this survey, disease incidence increased as reflected by the positive disease rates. However, the disease slowed dramatically during the 1979 to 1980 period. They concluded that int sification of Scleroderris canker in young red p plantations in New York appeared to be prog sively slowing down. They also found some plai tions that showed signs of recovery. Their data s gest that environmental factors may hav significant affect on the rate of disease incid both on a local and a regional basis. They cau that, "although the disease seems to be moving slow rate currently, there is always the potentia a return to the serious and rapid intensificatic the mid-1970's".

Carvin and Manion's results in young stand similar to our results from the spread studies i year-old red and Scotch pine stands. The New Scleroderris epidemic apparently developed a result of favorable infection conditions for se consecutive years along with the availability large volume of lower crown pine foliage. This bination resulted in the rapid development of a tremely serious epidemic. With less favorable tion conditions and the loss of the susceptible foliage, the infection is now progressing at a slower rate. Individual stands are still showing ous infection conditions, but the Statewide epi that developed in the mid-1970's has not reapp

Kurkela (1984) reported on the spread of G *etina* within stands in Finland. He stated tha demics can develop only when a large amount stands are potentially susceptible. This closely with the situation that developed in no New York in the mid-1970's. Kurkela also that during the winter resistance may be bro fail to develop. Pines introduced from differe matic conditions or planted on unfavorable si most likely to become susceptible. In that s

Gremmeniella epidemic in pine stands can be a manmade problem. Kurkela also feels that with increased inoculum potential the pathogen is also able to attack more resistant local pines. This situation also developed in New York during the 1970's epidemic. Strip plantings of red pine became severely infected by *G. abietina*. At the same time the fungus infected adjacent strip plantings of resistant white spruce resulting in branch dieback. Following the death of the red pine and subsequent reduction in inoculum, the white spruce infection declined to undetectable levels. Thus, the white spruce became infected under heavy inoculum loads but when these were removed, the causal fungus was not able to continue on white spruce alone. Hudler *et al*. (1983) have also shown that species differ in susceptibility based on inoculum dose.

VARIATION OF FUNGUS ISOLATES

In the introduction, we referred to the North American and European strains of *G. abietina*. These two strains have been separated on the basis of their ability to infect the upper crown of large trees and on their immunogenic reaction to European isolates of *G. abietina*. Dorworth and Krywienczyk (1975) proposed three races.[3] The North American race was based on a group of serologically similar isolates from Canada and the United States that differed predictably in serologic reaction from a group of isolates from Europe, termed thereafter the European race. They also established an "Asian race" based on a different serologic reaction shown by five isolates from Hokkaido, Japan. This grouping of three races has proved useful thus far. However, as more isolates have been serologically tested in eastern North America, additional differences in serological reaction have appeared. Both Skilling (1981) and Wendler *et al*. (1980) have characterized an "intermediate race" that responds both to European and North American antisera when subjected to serologic examination. For practical regulatory purposes, the intermediate race or strain has been grouped with the European strain isolates because these isolates have shown field pathogenicity reactions that were identical to the European isolates (they were able to infect trees of all sizes). As the serological techniques are increasingly refined, more physiological strains probably will be defined. It is not clear whether these physiological strains will also differ in pathogenicity. The intermediate physiological strain as characterized by serology appears pathogenically similar to the European strain.

[3]*The words race and strain are used interchangeably in this paper.*

Thus all pathogenic strains are physiologic strains but the converse may not be true (Dorworth 1981). At this time no pathogenic studies have been conducted using the Asian strain under North American conditions so we cannot comment on its pathogenic relation to the European and North American strains. Yokota (1975) describes the Asian strain as a top invader of pole-sized *Abies sachelinenis* Mast. weighted to or near the ground during winter by snow at higher altitudes in Hokkaido, Japan. The most pronounced symptom, upper crown dieback, appeared after the trees resumed an erect position in the spring. It is unknown how the Asian strain would react to North American species under North American climatic conditions.

Two confusing situations with fungus variation were reported by Skilling (1984a). These should be mentioned to avoid the impression that strain identification is completely understood. In the first case an isolate obtained from black spruce in Laurentide Park in Quebec, Canada, tested as European strain. However, this isolate only infected the lower branches of black spruce trees and produced abundant apothecia of *G. abietina*. Both characteristics are normally found only on trees infected with the North American strain. In the second case an unusual isolate was identified in northern New York on red and jack pine trees. This isolate was found in the upper crowns of large trees, which is characteristic of the European strain. It also produced large quantities of ascospores under field conditions. Single ascospore cultures derived from one apothecium produced mycelial cultures that gave either North American or European immunogenic reactions depending on which single-spore culture was used. This suggests the possibility of hybridization between European and North American strains (Furnier *et al*. 1984). Conidia produced from mycelium of these two strains were used to inoculate seedlings of white spruce, and jack, Scotch, lodgepole, red, and white pine. Both types of conidia were able to infect all species inoculated, but the conidia produced from the North American mycelium killed many more seedlings of every tree species inoculated. This was the reverse of what would be expected and emphasizes that much more information is still needed on the relation between physiological and pathological strains.

At the present time the North American strain has been identified in Michigan, Wisconsin, Minnesota, New York, Vermont, Maine, Quebec, Ontario, New Brunswick, Alberta, British Columbia, and probably Nova Scotia. The European strain has been found in New York, Vermont, New Hampshire, Maine, Newfoundland, Quebec, Ontario, and New Brunswick (Skilling 1984a) (fig. 1).

CONTROL

Silvicutural

Because of the damage caused by *G. abietina* in pine stands, a number of studies have been established to develop control systems. Studies by Skilling (1968) showed that much of the damage and spread of this fungus in the Lake States was caused by planting infected nursery stock. Two recommendations were made to reduce these losses. The first was to avoid fall planting of nursery stock because it is impossible to identify latent infections during this period. The other recommendation was to prevent nursery stock infection by implementing a fungicide spray program, which will be discussed later. Skilling also determined that young seedlings planted within 700 m of plantations infected with the North American strain would become infected at an early age and some seedlings would die. He also determined that Scleroderris canker is most serious in forest areas above latitude 45° north or at high elevations where summers are cool and the frost-free period is less than 90 days. No relation was found between site quality and disease severity (Skilling 1984b).

Pruning lower branches of infected and healthy trees has delayed disease buildup. in test plots in Vermont (Bergdahl and Ward 1981). A 12-year-old red pine plantation with 44 percent of the trees infected was pruned to a height of 1.5 to 2.0 m to remove branches infected with the European strain of *G. abietina*. Two years after pruning less than 1 percent of the pruned trees were infected while 54 percent of the trees in the untreated plantation were infected. A similar study by Bergdahl in an 18-year-old red pine stand with 59 to 100 percent of the trees infected was less effective. In this stand 22 percent of the trees in the pruned area were infected 2 years later. Most of the infection in the pruned area appeared to be latent infection missed during initial pruning.

If sites in high Scleroderris hazard zones are suitable for growth of resistant or immune species, forest managers would be wise to grow these to reduce the impact of this disease.

As the European strain moves into new geographic areas, sanitation may be one of the most effective controls. This procedure has been used successfully in New Hampshire, Maine, and Quebec. Although it will not prevent reinfection, it may prevent establishment of the disease for many years. If only a few trees are infected, pruning of infected branches may be adequate. If infection is wid spread, removing the entire plantation may be ne cessary. Harvesting merchantable material pl chipping needle-bearing slash will greatly redu the disease hazard although the fungus can surv on infected pruned branches for 10 months after t branches are removed (Dorworth 1972). Placing t infected branches in plastic bags has been suggest as an alternative to removing them from the plant tion but a recent study showed that *G. abietina* cou survive for up to 14 days in green plastic bags e posed to the sun (Davis and Dorworth 1984). T fungus was isolated from 33 to 70 percent of branches checked. Davis and Dorworth reco mended that the infected material be removed fr the plantation and destroyed.

Silvicultural control in most cases is less effect with the European strain because plantation tr are susceptible throughout the entire rotation. W the North American strain, little damage will res from later fungus infections if infection can be k at low levels until the trees are about 2 m tall.

Legal

Quarantines have been used effectively in N York, Vermont, and eastern Canada to prev movement of plant material infected by the ropean strain of *G. abietina* (Barrett 1984, Laid 1984). Quarantines reduce the danger of l distance spread and appear to have prevented movement of the European strain on cut Christ trees and nursery stock. Because most quarant have been specific for the European strain, strain involved must be determined by serolo procedures prior to regulatory action. The t niques have been published in manual f (Skilling and Kienzler 1983) and they are expe and time-consuming.

Genetic

The long-term control of the European stra *G. abietina* will require finding native and e trees that are resistant to this fungus. Marti (1984) tested seedlings from 11 lodgepole pine p nies from western Canada at 2 sites in Sweden. resistance seemed to only slightly depend on g origin of seed. Disease incidence differed grea the two sites and no single seed source was c resistant.

In northern New York Skilling and Ri schneider (1984) tested 15 full-sib families of pine from Sweden as well as seed from seven northern European and Asian sources. None of the seed sources showed much resistance under the high inoculum levels present at the test sites. Skilling also tested 39 seed sources of red pine for resistance to both the European and North American strains of *G. abietina*. None of these seed sources was resistant to either strain. Some preliminary evidence of resistance to *G. abietina* has been found in jack pine. In 1982, 60 families of jack pine from the Lake States were screened against both strains. These seedlings were outplanted in New York and Wisconsin. The Wisconsin test showed severe infection on all families tested. However, in the New York test, 7 seed sources from southern Wisconsin and southern Minnesota had 42 percent infection while 17 seed sources from the northern Lower Peninsula of Michigan had only 7 percent infection. Although these data are preliminary, they show evidence of some resistance in jack pine (Skilling 1984c).

Yeatman (1984) has also reported resistance of jack pine to the North American strain of *G. abietina*. In a series of range-wide jack pine provenance tests in boreal and eastern Canada, resistance to the North American strain was found among seed from western, northern, and eastern Quebec. Low infection levels were also evident in particular provenances from New Hampshire, New Brunswick, and Cape Breton. Generally, provenances from the milder, southern limits of the jack pine range were highly susceptible to the disease. They were also subject to winter desiccation when planted in severe boreal or high altitude environments.

Significant clonal differences in susceptibility to *G. abietina* were observed in 14 clones of Austrian pine (*Pinus nigra* ssp. *nigra*) (Stephan *et al*. 1984). Clones with higher resistance possessed a higher buffering capacity of pH in homogenized bark tissue of 1-year-old shoots. The monoterpenes of the bark tissue of 1-year shoots were also related to disease resistance. Myrcene and limonene content showed significant negative correlations with the attack by *G. abietina*.

Stephan (1977) also has tested four provenances of ponderosa pine in northwest Germany. After 19 years in the field the provenances from British Columbia, Washington, and Oregon grew best. The Utah provenance was slow growing and almost 100 percent were killed by *G. abietina*. In 1983, the North Central Forest Experiment Station tested 47 seed lots of ponderosa pine and 26 seed lots of lodgepole pine from 5 western regions of the United States for resistance to *G. abietina*. Duplicate plots were established at two locations in New York and one location in Wisconsin. Trees at the New York locations were exposed to the European strain *G. abietina*, while trees in the Wisconsin plot were exposed to the North American strain. The results with ponderosa pine agree with tests conducted by Stephan that show extensive mortality with this species. After 1 year of exposure to high inoculum levels, seedling mortality ranged from 87 to 100 percent for those trees exposed to the European strain and 78 to 100 percent mortality for trees exposed to the North American strain. Although lodgepole pine had lower mortality rates due to shoot sprouts developing below the infection site, the infection levels were 87 to 100 percent for the European strain and 91 to 100 percent for the North American strain. Although these data are preliminary, both species appear to be highly susceptible. Jack pine is the only pine species that has shown any major variation in resistance to *G. abietina*.

CHEMICAL

In 1959 Elmar Kohh established fungicide tests in northern Sweden at the Lovkulla Nursery to protect Scotch pine from infection by *G. abietina* (Kohh 1964). Of five fungicides tested, maneb proved to be the most effective. Kohh recommended a concentration from 0.7 to 1.0 percent active ingredients plus a wetting agent. Spray applications began when the current year's needles were 0.5 cm in length and were repeated at 2-week intervals. He recommended five to six sprays during the summer. Kohh's paper was published at an opportune time for pathologists in North America because this was the time of the first identified losses from Scleroderris canker in North American nurseries. Kohh's nursery control recommendations were used as a starting point until better fungicide controls could be developed.

New control recommendations were released for the Lake States region in 1970. They advise the use of maneb at 3 percent active ingredient using 12 applications from May 21 to October 30 (Skilling and Waddell 1970). In 1974 these recommendations were updated based on tests with additional fungicides (Skilling and Waddell 1974). They recommended chlorothalonil or chlorothalonil and cycloheximide at 0.3 percent concentration of active ingredients. The number of sprays was reduced to seven and the application period was from late May to mid-August. All of Skilling's fungicide tests were against the North American strain. The fungicide mixture chlorothalonil and cycloheximide was originally tested by Klingstrom in Sweden. He found that the addition of 1 part of cycloheximide to 1,000 parts of chlorothalonil resulted in better disease control of *Lophodermium*, *Phacidium*, and *Scleroderris*

(Klingstrom 1974). He felt that the cycloheximide had a systemic action that made the process less dependent on weather conditions at the time of spraying. This fungicide mixture has never been commercially available in North America and at present chlorothalonil is the only fungicide registered for control of *G. abietina* in the United States.

In Quebec, Smerlis (1980) has also tested fungicides for control of *G. abietina*. He tested 17 fungicides against the North American strain and the strain found on black spruce, which he called the Quebec strain. He was able to control the North American strain on jack and red pine seedlings with two applications at 2-week intervals using a 2 percent water suspension of chlorothalonil or anilazine. He recommended spraying when the current-year leaders were from 5- to 10-cm long. He was able to control the disease on black spruce with only one spray application of chlorothalonil in mid-July. Skilling *et al*. (1984) were not able to control the European strain with the limited spray schedule reported by Smerlis. Although they applied chlorothalonil and chlorothalonil plus cycloheximide up to 12 times during the growing season, they were not able to achieve the 100-percent control they felt was necessary for nursery stock. They achieved the same degree of control with 4 sprays of chlorothalonil at 1 percent active ingredients as with 12 sprays at 0.3 percent active ingredients. The longer period of spore dissemination by the European strain makes fungicide control in nurseries more difficult. In a fungicide test in New York during 1980-1981, test seedlings were infected during an unusually warm, wet period in February. These seedlings had not been treated with fungicides since the previous fall. The results of this study indicate that because of the long infection period for the European strain, it will be difficult for nursery managers to protect seedlings throughout the entire year. To produce disease-free stock it will be necessary to keep a protective fungicide on seedlings from early spring until the seedlings are covered by snow in late fall. This is the only way to fully protect seedlings and prevent the spread of this fungus from nurseries into plantations. At this time no pesticide treatment can be recommended for infected plantations because of the excessive cost of the large number of fungicide applications necessary. It is important to keep susceptible plantations free of this disease wherever possible.

ACKNOWLEDGMENTS

Numerous individuals contributed to the research for this paper. We would like to acknowledge the assistance of Eileen Haynes, Clint Waddell, J Sullivan, TerrancePodoski, Carl Mohn, Denis pont, and Mariann Kienzler. Their help was in able.

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PESTICIDE PRECAUTIONARY STATEMENT

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U.S. DEPARTMENT OF AGRICULTURE

Skilling, Darroll D.; Schneider, Bruce; Fasking, Donald.
Biology and control of Scleroderris canker in North America. Res. Pap. NC-275. St. Paul, MN: U.S. Department of Agriculture, Forest Service, North Central Forest Experiment Station; 1986. 19 p.

Describes the taxonomy, distribution biology, and control of the European and North American strains of Scleroderris canker as it applies to North America.

KEY WORDS: Scleroderris canker, tree diseases, forest management, fungus variation, disease control.

1966-86

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