SCLERODERRIS LAGERBERGII GREMMEN AND THE PINE REPLANT PROBLEM IN CENTRAL ONTARIO

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ABSTRACT

The fungus, *Scleroderris lagerbergii* Gremmen, was an active pathogen of planted Austrian pine (*Pinus nigra austriaca* Endl.) and Corsican pine (*P. nigra calabrica* Schn.) in Europe and Great Britain for at least 50 years before it began to damage plantations of red pine (*P. resinosa* Ait.) and jack pine (*P. banksiana* Lamb.) in North America. The fungus was probably imported onto this continent from Europe or Great Britain. It has nearly eliminated red pine from many plantations and promises to exact a similar toll of planted jack pine. Further replanting of areas bearing potential infection sources with jack or red pine is to be discouraged.

TABLE OF CONTENTS

	Page
INTRODUCTION	1
HISTORY OF SCLERODERRIS DISEASES IN EUROPE AND GREAT BRITAIN . .	2
THE SCLERODERRIS PROBLEM IN NORTH AMERICA	2
REFERENCES	11

INTRODUCTION

Scleroderris lagerbergii Gremmen was first described from Ontario by the Forest Insect and Disease Survey of the Canada Department of Forestry (1965) from the identification by E. Jorgensen, University of Toronto, and from Michigan by J. Ohman (1966), U.S. Forest Service, who presented a careful description of the disease in young red pine (*Pinus resinosa* Ait.) plantations. These constituted the first legitimate reports of the fungus in North America so far as I am aware, and answered the question of constant failures of planted red pine in parts of central Ontario (Algoma and immediately surrounding areas) and Michigan, noted by Martin (1964) and Benzie (1958), respectively.

Two Ontario nurseries have discontinued the production of red pine seedlings, and management foresters have nearly discontinued planting them in areas where infections were severe in the past. In general, these infections occurred on pine species commensurate with the site, e.g., red pine in central Ontario and jack pine (*P. banksiana* Lamb.) in northern Ontario. Areas in the Searchmont and Kirkwood Management Units were repeatedly planted with red pine after initial plantation failures, before the nature of the difficulty was understood. Local buildups of spore inoculum occurred in many of these areas, so that successive pine plantings succumbed even more quickly than those planted initially.

Many Ontario and Michigan foresters were initially in the unfortunate position of spectators, watching their pine plantations being decimated by *S. lagerbergii*, and a healthy respect for the fungus resulted. Two misconceptions have come to the surface, however, that deserve immediate consideration:

a) the fungus operates on a biologic cycle, which is now on the downswing, and plantations established at this time will have a reasonably good chance of succeeding;

b) jack pine is considerably less susceptible to *S. lagerbergii* than red pine, and can be used to replant or interplant red pine plantations which were partly depleted by *S. lagerbergii*.

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HISTORY OF SCLERODERRIS DISEASES IN EUROPE AND GREAT BRITAIN

Scleroderris lagerbergii has been known in Europe under a variety of names since it was first described by Brunchorst (1888), whose name has been given to the imperfect, or asexual, stage of the fungus: *Brunchorstia pinea* (Karst.) Hoehn. Lagerberg (1913) later discovered and described the perfect, or sexual, form of the fungus. Confusion surrounded the nomenclature of the fungus in the early part of this century. The confusion was dispelled for a short time when J. Gremmen of the Bosbouwproefstation, Netherlands, applied the name *S. lagerbergii* (1955). The Canadian mycologist J.W. Groves (1965) cast doubt upon the efficacy of this name, and the fungus will undoubtedly be renamed. A much more complete statement of the situation will be found in Ettlinger (1945) and Dorworth (In press).

The pathogenicity of *S. lagerbergii* was in doubt for some time in Europe. Some workers declared the organism to be nearly harmless, and at worst a natural pruning agent, while others noted heavy mortality caused by the fungus. According to Jørstad (1929), planting of Austrian pine (*P. nigra austriaca* Endl.), the principal European suscept, had been abandoned over much of northern Europe. The dilemma was resolved, at least in part, by Gremmen (1966), who noted that frost damage and attack by *S. lagerbergii* are distinctly different. Gremmen (1968) noted further that the disease became important only when Austrian pine was planted outside its native range, and in areas where relatively low temperatures and high relative humidity prevailed. Even then, the disease did not become damaging until the crowns of the young trees met, and a continually moist and cool environment was established in which the fungus could thrive. In Europe, *S. lagerbergii* is considered to be a relatively weak pathogen which remains in balance with its primary suscept while the latter is within its natural range, but which incites a serious disease problem when the primary suscept species is moved from its native areas to less suitable sites. In England, however, Read (1968) concluded that individual tree resistance was more important than site in determining whether or not Corsican pine (*P. nigra calabrica* Schn.) would succumb to attacks of *S. lagerbergii*. He also noted that many of the weaker trees would have formed a crop were it not for the fungus, and termed the fungus a primary, rather than a secondary, pathogen.

THE SCLERODERRIS PROBLEM IN NORTH AMERICA

Much of the damage done to red pine plantations in central Ontario between the 1930's and the 1950's has since been attributed to *S. lagerbergii*. Dorworth (In press) considers the almost simultaneous outbreaks of the disease in Canada and in the United States along the Great Lakes, the lack of earlier reports of the fungus, and the complete

or nearly complete lack of genetic resistance in the principal local suscept species, red pine, as sound indications that *S. lagerbergii* is an exotic pathogen in North America.

Effects of environment in the balance between suscept resistance and pathogen virulence are less clear in North America than in Europe. Both red pine and jack pine are attacked and killed from the time they are in the nursery until they exceed approximately 8 ft in height, contrary to the European experience. Successful infection and/or colonization of the suscept tissues does not require that the crowns of the trees meet, but rather occurs long before that point in their development. There is some agreement that trees die more quickly in low areas, which may be frost pockets, than in the surrounding higher countryside (Ohman, 1966; French and Silverborg, 1967). The frost pocket explanation is certainly not applicable in every case of plantation failure in which *S. lagerbergii* is involved, particularly on and adjacent to sites where healthy trees had previously developed. Sexual spores of *S. lagerbergii* (ascospores) are windborne, and are deposited in random fashion on the current year's branches and foliage of pines, from which points the fungus grows into the internal tissues of the trees, and causes branch death (Figure 1). Such branch tip infections on small trees often progress into the main stem, where the fungus grows in the cambium. Death of the cambium leads to the formation of a canker (Figure 2) which enlarges year after year and often kills the tree. Branches are killed in random fashion by *S. lagerbergii* within the first 3 to 4 ft of the stem, whereas frost damage most often results in the death of all branch tips up to a certain height, as illustrated by Pomerleau and Ray (1957). Cankers incited by *S. lagerbergii* develop progressively from the point at which the fungus enters the main stem, and are characterized by stem "fasciation", or flattening. Frost cankers, which are not true cankers, are relatively rare on red pine and ordinarily heal with the formation of a protuberant "rib" of tissue, which may enlarge if frost cracking recurs in subsequent years. Teich (1968) reported that frost did not predispose jack pine in a northern Ontario provenance test to attack by *S. lagerbergii*. More actual information of the sort presented by Teich is in order for red pine in central Ontario before rigid viewpoints are developed.

Planting of red pine in certain areas contaminated with *S. lagerbergii* has been discontinued for the present. The disease itself is presently at low ebb in the Kirkwood and Searchmont Management Units, with *S. lagerbergii* merely cleaning up the remainder of the red pine which were previously infected or are still small enough to succumb. Once red pine have reached heights of 6 to 8 ft, they are relatively safe from fatal attacks by *S. lagerbergii*, and any lower branch infections that occur usually die out as the branches die. The present lack of high-level activity by *S. lagerbergii* is only relative,

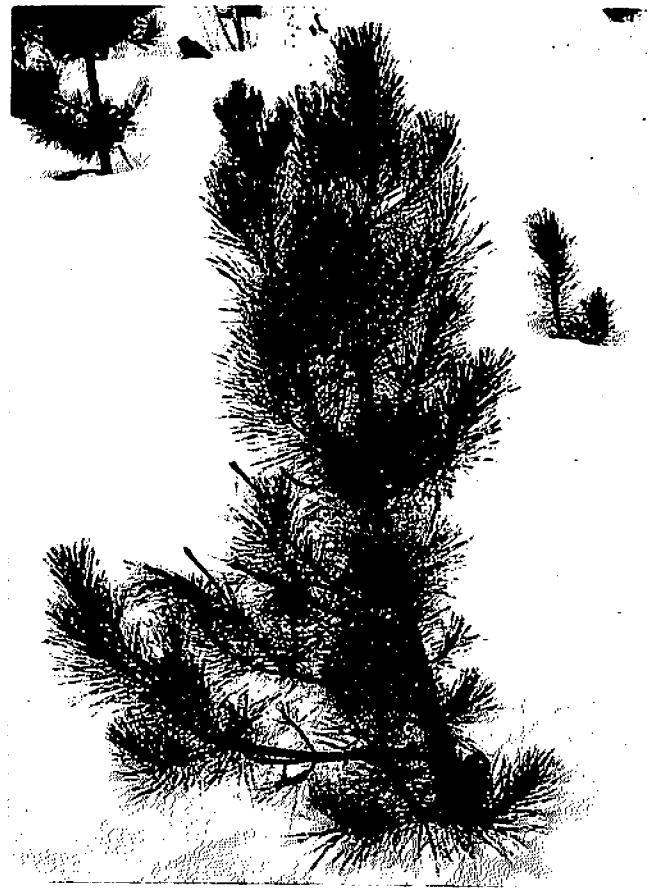

Figure 1. Early branch infections by Scleroderris lagerbergii on 6-year-old red pine.

and reflects a paucity of red pines in their most susceptible stage of development. It should not be assumed that the virulence of the fungus has in any way diminished.

I suspect strongly that the disease situation which existed in the past with red pine will shortly be repeated to some degree with jack pine. Cordell *et al.* (1968) in Michigan noted that, while jack pine was infected somewhat later in life than red pine, jack pine was ultimately more severely affected by the infections on the same sites. It is possible that the red pine were the more heavily infected when planted and that *S. lagerbergii* spread later into the jack pine. Furthermore, there is always the possibility that research findings from one geographic area will not prove applicable in another, although the proximity of Michigan to Algoma District makes it unlikely that such will apply here.

Figure 2. Scleroderris lagerbergii stem canker of 18-year-old red pine.

Even with these reservations, I believe that we should predict failures of jack pine plantations established, and being established, adjacent to and intermingled with infected red pine. My observations in central Ontario indicate that jack pine is susceptible to *S. lagerbergii*, can be killed by the fungus, and will fare little better than the red pine under the influence of a heavy concentration of spores.

Symptoms of the disease in red pine are familiar to most involved management foresters, noted as die-back of the infected branch tips in the spring, after which the foliage turns first a dark chocolate-brown, and then a silvery brown, and finally falls (Figure 3). The foliage of *S. lagerbergii*-infected jack pine branches, on the other hand, most often turns a straw-yellow colour in the spring, becoming darker toward autumn, and falls from the branches (Figure 4). Symptoms

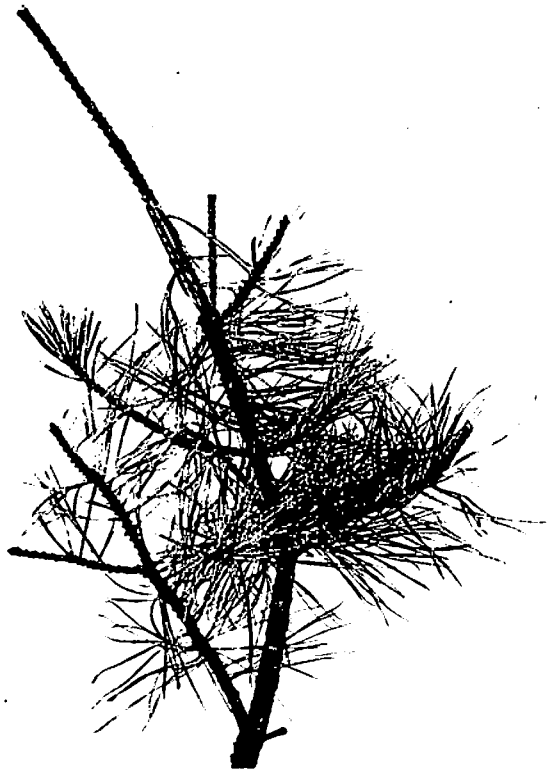

Figure 3. Branch tip symptoms of Scleroderris lagerbergii infection of red pine.

of *S. lagerbergii* infection on jack pine are clearly distinguishable from those caused by root rot. In the latter case, the predominant causal agent, *Armillaria mellea* (Vahl. ex Fr.) Kummer, creates an infection which causes all of the foliage to turn dark brown simultaneously in the spring. Where the forest manager is in doubt about the cause of disease symptoms in a particular case, he should contact the Insect and Disease Survey Unit of this department in Sault Ste. Marie for verification.

S. lagerbergii will grow through the branches of young jack pine as well as those of young red pine, and into the main stem, where it girdles the tree or causes formation of a canker. The branch which was the original infection court is often visible at the center of the developing canker (Frontispiece, cut surface showing). The presence of the fungus may often be confirmed by slicing the infected stem longitudinally, and noting the green to yellow-green pigment in the wood and inner bark. The green colour is not to be confused with that of residual chlorophyll, which may remain in young, newly-killed jack pine branches until well into the summer.

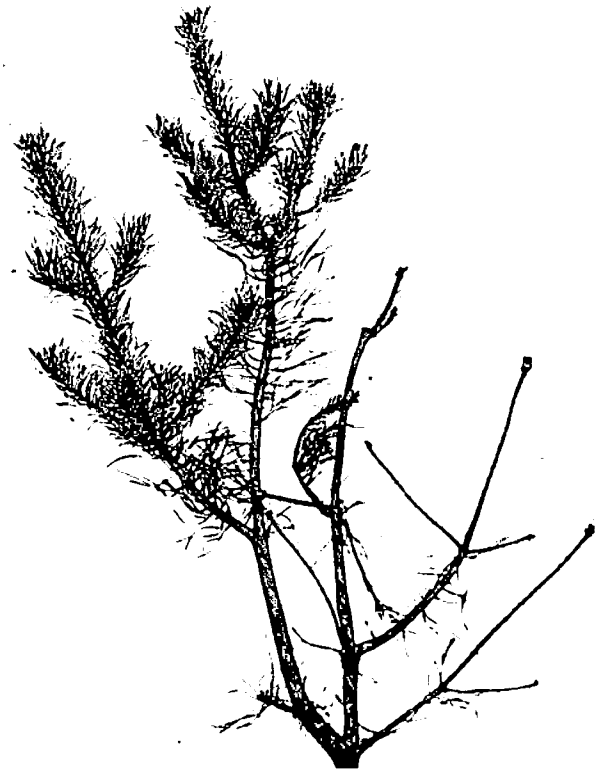

Figure 4. Jack pine seedling partly killed by Scleroderris lagerbergii, with symptoms visible on surviving shoot.

Killing of jack pine in the 4- to 6-year-old plantations examined does not at present exceed 5% of the trees examined, but each tree that dies provides a source of spore inoculum for subsequent infection of surrounding trees. The wet spring of 1969 probably enhanced dispersion of the spores and infection of trees and accelerated secondary infection will be visible in the spring of 1970. The situation is further aggravated by the presence of naturally-occurring jack pine which have become infected in the course of earlier disease outbreaks. Observations to date indicate that jack pine will not be killed if it reaches approximately 12 ft in height before infection occurs. Branch infections, however, will supply inoculum to contaminate further plantings of pine on an area. Both the jack and red pine (Figure 5) are infected, and it is no longer possible to determine which was infected first. Furthermore, infections are found as high as 20 ft from the ground on the foliage and new branches of jack pine, whereas red pine branches are seldom infected where they occur 4 ft or more above the ground. These high-branch infections on jack pine would presumably spread inoculum over a much greater area than would be possible from infections on red pine.

Figure 5. Young red pine, killed by Scleroderris lagerbergii, adjacent to pole-sized jack pine with numerous lower branch infections and lower branch mortality.

Certain pine sites in central Ontario were thoroughly contaminated with *S. lagerbergii*, and plantation failures were a regular occurrence, for at least 20 years before the cause was defined. Certain of these areas were replanted one or more times, the level of spore inoculum increased with each successive disease outbreak, and the areas are no longer acceptable sites for establishment of red pine plantations. Jack pine seedlings were planted on certain of these sites on the assumption that the species was less susceptible than red pine to *S. lagerbergii*. The young jack pine have reached heights of 2 to 3 ft and are beginning to die, as are a few of the older volunteer stock on the poorer sites. Unless several especially dry years follow, which could inhibit spread of and infection by the fungus (Skilling, 1968), stocking of the remaining jack pine will be substantially reduced before they reach a height where they become relatively immune to lethal infections. These experiences lead us to make certain suggestions with regard to future plantings of red and jack pine on public and private lands.

1. Do not replant red pine seedlings among or adjacent to older pines which have been infected by *S. lagerbergii*. This merely repeats the general rule which instructs one not to replant an infection area with a susceptible species until the source of infection is eliminated.

2. Jack pine should not be planted in place of red pine under the premise that it is resistant to *S. lagerbergii*. All sources of potential infection should be removed before an area is replanted.

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