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Biology and Control of *Sphaeropsis sapinea* on *Pinus* Species in South Africa

Sphaeropsis sapinea (Fr.:Fr.) Dyko & Sutton in Sutton is an opportunistic pathogen of *Pinus* species in more than 25 countries of both hemispheres between the latitudes 30° and 50° north and south (15,30,34). Since the fungus was first described under the name *Sphaeria pinea* Desm. by Desmazieres in 1842 as a saprophyte on needles of *Pinus sylvestris* L. in France, it has acquired at least 23 synonyms, of which *Diplodia pinea* (Desmaz.) J. Kickx fil. was the most widely used (14,30,40). The taxonomy of the fungus was reviewed by Sutton (33), and the name *Sphaeropsis sapinea* was designated to it.

During the past 20 years, several studies of the taxonomy, morphology, etiology, and epidemiology of *S. sapinea* have been conducted in the United States, Australia, Europe, and South Africa. These studies have provided new facts regarding the biology of the fungus and have stimulated many new questions that will influence hypotheses for future studies. Our aim is to place knowledge of *S. sapinea* into perspective so as to formulate procedures to reduce the pathogen's impact on pines throughout the world.

Historical Perspective

The earliest known reports of *S. sapinea* as the cause of a disease of pines were published in 1908 and 1911 from the Royal Botanical Gardens, Kew, England, in connection with infected nursery stock and *Pinus radiata* D. Don trees in the Cape Province of South Africa (40). In Europe, the importance of *S. sapinea* as a pathogen is mentioned only occasionally in the literature, despite the fungus having been present in western Europe for almost 150 years. The first reports of serious economic damage were from the Netherlands, in 1982 on *P. nigra* Arnold and *P. sylvestris* (13) and again in 1985 (19). The first report of *S. sapinea* as a pathogen in the United States was in 1917, when it was determined to be the causal agent of canker and shoot blight of pine in New Jersey (40). *S. sapinea* has been found in 32 states, 30 of them in the central and eastern regions of the country (28). The pathogen has caused most damage in plantings of exotic pine species such as *P. nigra* and *P. sylvestris*, but plantings of native pine such as *P. resinosa* Aiton, *P. banksiana* Lamb., and *P. ponderosa* Douglas ex P. Laws & C. Laws have also been severely damaged in the central United States. The pathogen is, however, of relatively minor importance in the United States and Europe compared with the destruction it has caused in exotic *P. radiata* plantations in New Zealand, Australia, and South Africa (6,7,12,44,45). The international notoriety of the pathogen has largely been attributed to the South African experience with extensive infection and mortality in plantations of *P. radiata* and *P. patula* Schlechtend. & Cham. after hail injury (15,20). Reports of *S. sapinea* from other parts of the world where the pathogen is known to occur, such as Central and South America and Asia, are few and lack significant detail (30,40).

Symptoms and Associated Losses

S. sapinea has been associated with damping-off and collar rot of seedlings, shoot blight, sap stain, canker, and root disease (24,30,34,40,41) (Figs. 1 and 2A and B). Shoot blight is the most common symptom associated with *S. sapinea* infection and occurs on seedlings and mature trees (6,7,15,17,28,35,40). In the north-central United States, losses of up to 35% resulting from shoot blight of first-year *P. resinosa* seedlings have been recorded (26). Infection of older shoots results in dieback of the current season's growth, which, if it occurs year after year, results in stunting and deformation of the tree and ultimately death. Dieback can reduce timber increment by 40% (43) and potential marketable volume by 63% (12). Infection of terminal shoots results in a condition known as dead-top, top-kill, or stag-head, which is considered the most damaging form of shoot blight (12,43) because it drastically affects the utilizable length of the bole.

Certain symptoms and situations associated with *S. sapinea* either are unique to some parts of the world or occur only rarely in other parts. The association of *S. sapinea* with a serious root disease of mature *P. elliottii* Engelm. and *P. taeda* L. trees has been reported only from South Africa (35,41). Collar rot of nursery seedlings, although becoming increasingly common in the north-central United States (24,25), has not been reported from South Africa and is very rare in other countries.

Dieback attributed to infection by *S. sapinea* after hail injury has resulted in extensive losses in South African pine plantations. In a case study, timber losses

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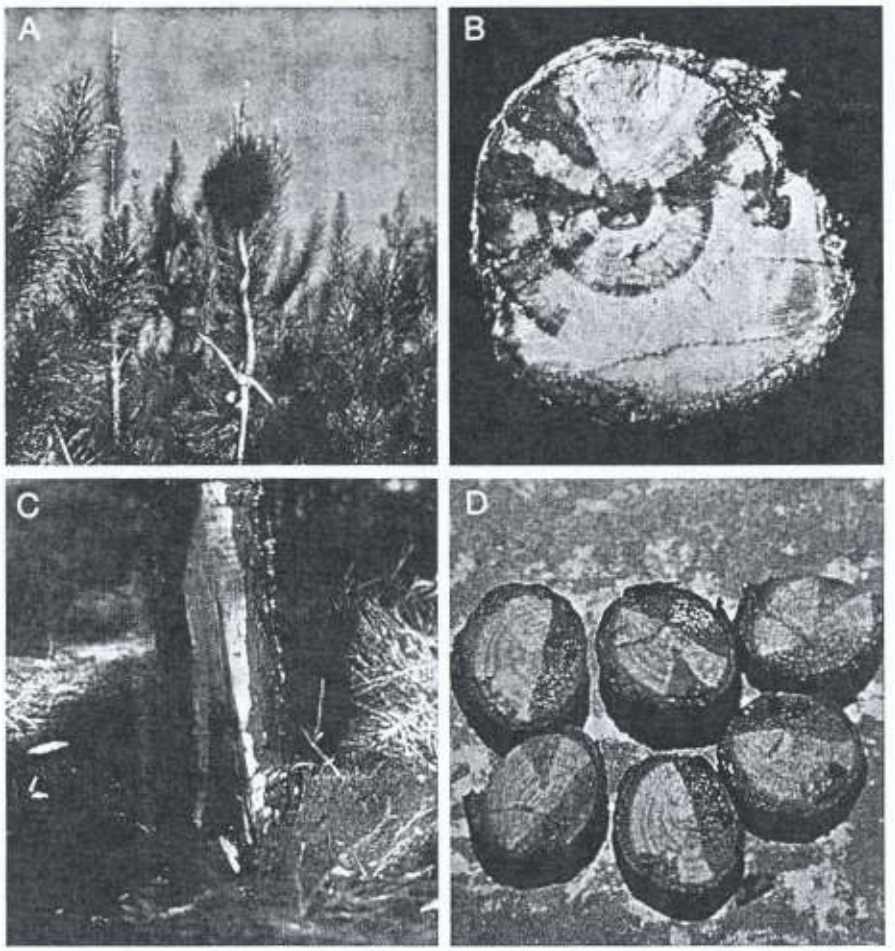

Fig. 1. Symptoms of infection by *Sphaeropsis sapinea*: (A) Dieback of leader of *Pinus radiata*. (B) Cross section through canker on branch of *P. radiata*. (C) Discoloration of bole of *P. taeda* resulting from root disease. (D) Cross sections through infected root.

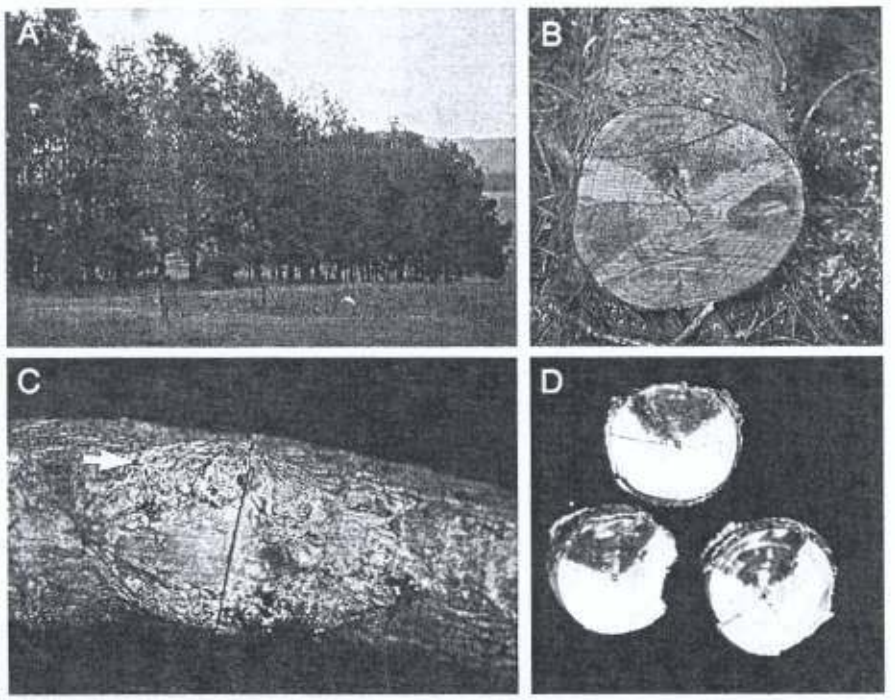

Fig. 2. Symptoms of infection by *Sphaeropsis sapinea* after hail damage: (A) Dieback of *Pinus patula*. (B) Sap stain of bole of *P. patula*. (C) Pycnidia (arrow) surrounding wound on hail-damaged branch of *P. radiata*. (D) Cross sections through hail wound showing infected tissue.

in 2,000 ha of local pine plantations were evaluated as 28% of potential volume and 55% of potential value. Losses caused by *S. sapinea* and hail-associated dieback in South Africa are estimated as approximately $3.8 million (U.S.) annually (45). Although a few isolated—and far less extensive—instances of hail-associated infection have been reported from Australia (4,14), it is virtually unknown from other parts of the world.

The occurrence of stem or whorl canker was first reported in the United States in 1976 on *P. resinosa* (23). Previously, stem canker had been reported only from New Zealand and Australia in association with pruning damage of *P. radiata* (16). The problem has increased considerably in New Zealand during the past 10 years, with mortalities of up to 19% being reported from some *P. radiata* plantations (11). There are no reports of stem canker from Europe, and losses resulting from stem canker are relatively rare in South Africa (35).

Factors Influencing Disease Severity

Damage caused by *S. sapinea* can be either direct or indirect. Direct losses usually occur incrementally from death of shoots and branches. Indirect losses are associated with timber degradation from sap stain and cutting of diseased trees before they reach maturity and maximum value. We believe that damage caused by *S. sapinea* may be more common than generally thought, since the role of this opportunistic pathogen may easily be overlooked or misinterpreted. The economic importance of *S. sapinea* also may be underestimated because few loss assessment studies have been done.

Manifestation of the host-pathogen interaction, in terms of both the qualitative and the quantitative nature of symptom expression by *S. sapinea*, may vary according to intrinsic factors related to the pathogen or its host or to the influence of the physical environment on both components. Published work based on studies of these factors has often been contradictory, and as a result, much controversy has surrounded the pathogenicity of *S. sapinea*. This is probably because past research concentrated more on factors related to the environment than on the physiological interaction between the pathogen and its host.

Wounding and physiological stress. Infection affects trees that are wounded (21,40) or physiologically stressed by drought or nutrient deficiencies (1,2,4,10, 22,35,43). Hail damage of moisture-stressed trees was associated with 70% of reported cases of shoot blight and dieback during a survey over 2 years in South Africa (35) (Table 1). Infection associated with drought only was less

extensive but more common than that associated with hail damage only (Fig. 2C and D). Hail in the absence of drought was associated with 7% of trees affected by dieback, and drought in the absence of hail was associated with 23%. In accordance with findings of other workers (2,4), we therefore conclude that infection is more severe when stressed trees are damaged by hail.

Hail damage or physiological stress are not necessarily requisites for successful infection by *S. sapinea*. Pine shoots are susceptible to infection under optimal climatic conditions in the absence of both hail and drought (3,6,7,18,35,37). Association of infection by *S. sapinea* with insect damage in the absence of physiological stress has also been observed (17,35,42).

Pine oleoresin contains monoterpenes toxic to *S. sapinea* and may be significant in host resistance (4). Physiological stress caused by overstocking, drought, and nutrient deficiencies has been shown to reduce oleoresin flow in plantations of *P. taeda*. Further studies should elucidate the relative importance of physiological stress in predisposing trees to infection by *S. sapinea*. If stress in response to the factors could be measured quantitatively, the measurements could be correlated with disease severity.

Tree vigor and age. Fast-growing trees on productive sites may be more severely infected than stressed trees on inferior sites (9). The ability of *S. sapinea* to infect unwounded shoots and the extent of damage probably are related also to age of trees and maturity of host tissue. In the midwestern United States, trees over 30 years of age were most severely affected by *S. sapinea*-induced dieback (28). Chou (8) reported that shoot dieback decreased after trees reached 7–8 years of age, but further investigations revealed no genetic basis for this phenomenon. Studies we conducted in South Africa have provided no evidence that susceptibility is related either to age or to maturity. Older plantations, however, generally have higher inoculum levels of *S. sapinea* than do young plantations (3,28). Increasing stand age, in conjunction with site index (i.e., the mean predicted height of trees at 20 years of age) or site quality, influences financial loss resulting from premature clear-cutting after hail damage and infection by *S. sapinea* (45) (Fig. 3). Financial loss primarily reflects the decrease in value of highly priced classes of timber due to sap stain caused by *S. sapinea*.

Relative susceptibility of host species. More than 33 *Pinus* and various other conifer species have been reported to be susceptible to *S. sapinea* (15,31). Interspecific differences in susceptibility of some tropical pines to *S. sapinea* have been demonstrated in glasshouse and field trials (40,31,37). Intraspecific resistance in *P. radiata* has also been recorded (5). In South Africa, field observations over many decades have indicated that *P. radiata* and *P. patula* are far more susceptible to infection than *P. elliottii* or *P. taeda* (20,37). When results of glasshouse (Fig. 4) and field trials with five *Pinus* spp. grown commercially in South Africa were compared, susceptibility in both environments was similar and was consistent with field observations.

Instances of resistant pine species showing more damage than susceptible species growing nearby have been documented (4). Similarly, hail damage of susceptible pine species in South Africa is not always followed by *S. sapinea*-induced dieback, despite sufficient inoculum in a pine stand. In such instances, absence of dieback probably is attributable to insufficient predisposition to infection by physiological stress. Infection also could be suppressed by factors related to the host-pathogen interaction or to climatic conditions.

Climatic factors. Differences in the amount or frequency of damage resulting from *S. sapinea* infection have been

Table 1. Percent incidence of *Sphaeropsis sapinea* infection on *Pinus* species in South Africa during 1985–1986 in relation to predisposing factors

Predisposing factors	*Pinus* species						
	radiata	*pinaster*	*patula*	*taeda*	*elliottii*	Other	Total
Hail	4	4	2	...	...	...	10
Drought	6	2	...	2	6	4	20
Drought and hail	26	8	6	...	2	4	46
Insects	4	...	...	...	...	...	4
Nutrient stress	2	...	...	...	...	...	2
Undetermined	14	4	...	...	...	...	18
Total	56	18	8	2	8	8	100

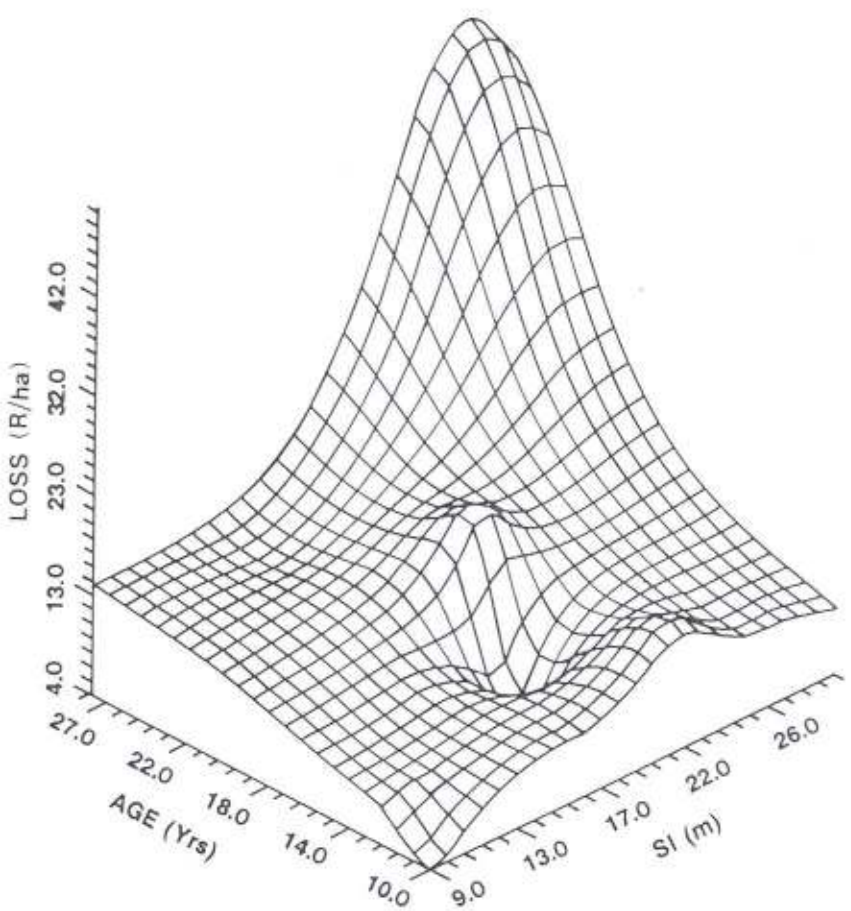

Fig. 3. Relationship of site index (SI = mean predicted height of trees at 20 years of age) and age of stands to loss of value (R [rand] 1.00 = U.S. 40¢) of timber from *Pinus radiata* stands prematurely clear-cut because of infection by *Sphaeropsis sapinea* in South Africa. (Reproduced with permission from Zwolinski et al [45])

ascribed to various site-related climatic factors. These factors may influence both the initiation and the outcome of infection. One example is the possible relationship of dieback incidence to topography. Enclosed valley sites may be more conducive than slopes to severe infection (8,43), an interpretation substantiated by developments after severe hail damage and *S. sapinea*-induced dieback of 2,000 ha of pine plantations in South Africa (44). Microclimatic conditions seemed to be the most important factor affecting *S. sapinea* infection. In enclosed valley sites and sheltered stands, with higher humidity and less temperature variation, the intensity of damage by *S. sapinea* was far greater than in open country (Fig. 5).

The interaction of microclimatic conditions with other predisposing factors is also important. If, for instance, temperature and humidity are minimal for infection, physiological stress could increase the chances of successful infection. Similarly, infection would not occur on stressed or wounded trees where temperature and humidity were not within the required range for infection.

Climatic factors related to time of year have been shown to influence both the availability of inoculum and the susceptibility of pine tissue to colonization by *S. sapinea*. Conidia disperse primarily during rainfall, when pycnidia on pinecones and pine needles release spores (25,36). Wind not accompanied by rain plays a very limited role in dispersing conidia. Studies based on spores trapped in three climatic regions of South Africa (36) (Fig. 6) suggest that maximum dispersal of conidia is not directly related to highest periods of rainfall but to temperatures prevailing after periods of maximum rainfall. In the southwestern Cape Province, which has a Mediterranean climate, the number of conidia trapped over a period of 2 years was lowest during winter, when most rainfall occurred. Conidial dispersal was maximal 16 weeks later during spring, when rainfall was considerably less but temperatures were more ideal for formation of conidia. These findings are consistent with those of similar studies conducted in the north-central United States (25).

Seasonal differences affect susceptibility of *P. radiata* seedlings and young shoots to infection by *S. sapinea* (9,25,28) and of pruning wounds to infection (11). In a preliminary study, lesions in the cambium of *P. radiata* were more extensive after artificial inoculations in spring than after inoculations in fall (*unpublished*). These results confirm findings in New Zealand that infection of pruning wounds is confined to a short period in the summer (11) and suggest that higher temperatures facilitate *S. sapinea* infection and colonization of pine tissue. Further research is necessary to enhance understanding of the interacting roles of seasonal and physiological predisposition in infection of pines by *S. sapinea*.

Genetic variability of the pathogen. In recent years, the degree to which pathogenic variability among *S. sapinea* isolates can influence disease has become evident. Conflicting results from independent investigations regarding the necessity of wounds for infection might be explained in part by the existence of different strains of the fungus (27).

Isolates of *S. sapinea* differ in cultural characteristics, conidial size and morphology, and pathogenicity (27,35,39). On the basis of some of these criteria, two groups of *S. sapinea* isolates—designated type A and type B—have been distinguished in the north-central United States (27,38,39). Type A isolates have fluffy white to gray-green mycelium, produce conidia with smooth surfaces (Fig. 7A), and can infect nonwounded tissue. Type B isolates have white to black mycelium appressed to the agar surface, produce conidia with pitted surfaces (Fig. 7B), and require wounds to infect pine tissue.

Significant differences in conidial dimensions and morphology, cultural

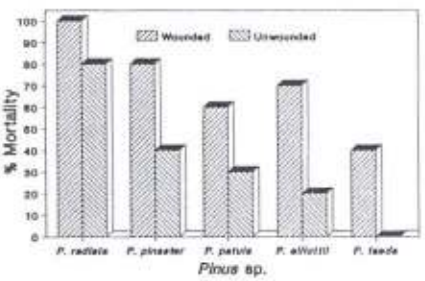

Fig. 4. Relative susceptibility of seedlings of five commercially cultivated *Pinus* spp. to infection by *Sphaeropsis sapinea* after glasshouse inoculations.

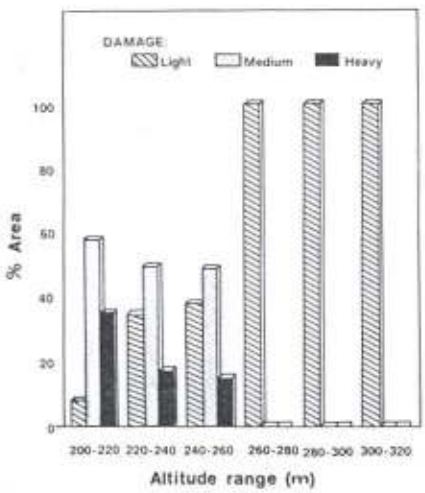

Fig. 5. Effect of topography on intensity of infection by *Sphaeropsis sapinea*. (Reproduced with permission from Zwolinski et al [44])

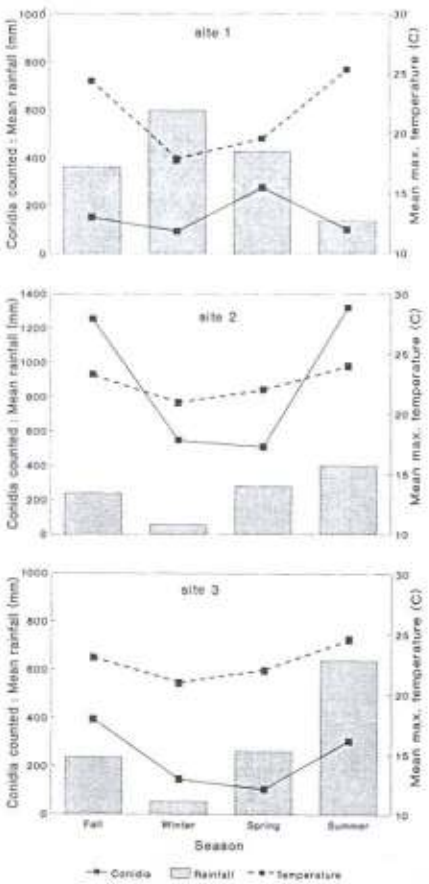

Fig. 6. Dispersal of *Sphaeropsis sapinea* conidia in *Pinus radiata* plantations in three climatic regions of South Africa: site 1, winter rainfall; site 2, all-year rainfall; site 3, summer rainfall.

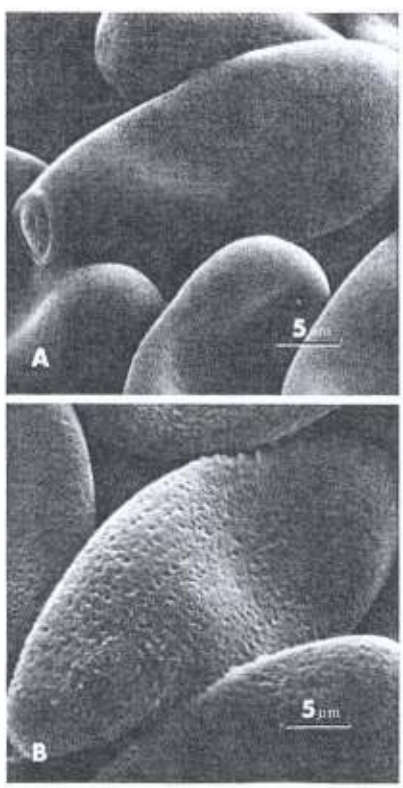

Fig. 7. Conidia of *Sphaeropsis sapinea* with (A) smooth and (B) pitted walls. (Courtesy R. A. Blanchette)

morphology, virulence, growth rate at different temperatures and on different media, and isozyme profiles have been demonstrated for South African isolates of *S. sapinea*. A significant positive correlation was shown between in vitro growth rate and virulence of isolates in both greenhouse and field inoculations. There was, however, no clear indication that local isolates could be classified into two or more distinct groups based on these criteria. *S. sapinea* appears to be a highly variable species that represents a continuum without definite types or strains. This hypothesis must be examined with isolates from many parts of the world.

Control Measures

S. sapinea can infect pines in many different ways and under a wide variety of conditions by virtue of its opportunistic nature. Consequently, suitable control measures are a problem. In the United States, fungicide application programs have been developed to control shoot blight caused by *S. sapinea* in ornamental and shelterbelt plantings (26,28,29,32). The cost and practical problems involved in spraying pine plantations in South Africa, however, limit the use of fungicides to nurseries.

The most practical disease control measure is exploitation of intraspecific and interspecific resistance of pines to *S. sapinea* (5,15). So far, however, screening for resistance to *S. sapinea* among selections of *P. radiata* from Australia (5) and within South Africa has offered little encouragement. The indiscriminate planting of resistant pines also is not always feasible. Variations in the site and climatic requirements of different species and provenances limit this option to certain geographic areas only. However, pine species that are susceptible to *S. sapinea* should not be planted in enclosed valleys or in small groups sheltered by other stands. If such planting is unavoidable, thinning should be intensive to modify microclimatic conditions and reduce physiological stress resulting from competition for water and nutrients. Nutrient deficiencies can be alleviated by fertilizing at planting or after thinning, or both.

Various options exist for controlling or limiting infection of pruning wounds. Increasing intensity of pruning has the effect of enhancing *S. sapinea* infection of *P. radiata* stems (11). In stub inoculation trials, infection was significantly greater in trees pruned to remove 39% (regular pruning) or 49% (heavy pruning) of green crown than in trees pruned to remove only 25% of the crown. This effect was ascribed to physiological stress similar to that caused by drought, provision of more wounds for infection, and a gradient of resistance along the vertical axis of the stem in which resistance decreased with age of the stem. The fact that drought stress may aggravate infection after pruning (11) suggests that intensity of pruning should be adapted according to site conditions.

The time of year when pruning is done also has an important bearing on controlling infection by *S. sapinea*. Dispersal of *S. sapinea* conidia was studied in three climatic regions of South Africa (36), and the data are substantiated by results of a separate study dealing with the effect of season on host colonization by the pathogen. Both studies suggest that pruning is best performed during winter, irrespective of the time of the year when most rainfall occurs. Low temperatures at that time restrict formation and availability of conidia as well as the rate at which the pathogen colonizes host tissue.

Sanitation measures that reduce the amount of inoculum can be implemented in nurseries and plantations. Removing infected seedlings from nursery beds, slash resulting from thinning and pruning operations, and diseased trees is easy. In South Africa, pine stands that have been damaged by hail usually are thinned or clear-cut prematurely to salvage as much of the potential production as possible. This practice obviously has sanitary advantages, but the decision to clear-cut infected trees because of potential mortality cannot be made reliably when hail damage occurs. The decision should not be influenced by extensive foliage discoloration soon after a hailstorm because foliage can regenerate after a year and trees can recover depending on the amount of physiological stress (21,23). Our studies indicate that timber discoloration rather than foliage discoloration is a more reliable indicator of disease development. We therefore recommend that during the first 6 months after symptoms appear, a small number of hail-damaged trees should be cut and examined periodically for cambium, wood, and pith discoloration. Infected trees in commercial stands should be removed when discoloration spreads to the lower crowns below the minimum utilizable stem length. Stands should be clear-cut only when infected trees are too numerous to be eliminated by thinning.

Conclusions

S. sapinea is by virtue of its opportunistic nature a complex pathogen, and considerable controversy surrounds its pathogenicity. Many different aspects of the pathogen's epidemiology have been investigated to improve understanding of the infection process and spread. However, observations of researchers working in different environments and with different isolates of *S. sapinea* have often been contradictory and have sometimes added to the confusion.

We have presented some of the more important variables that influence the pathogenicity of *S. sapinea* in South Africa and elsewhere. Recent additions to knowledge of the biology of *S. sapinea* have provided new perspectives on ways to minimize damage to pine plantations caused by this pathogen. Further research to address some of the remaining questions about *S. sapinea* will enable foresters to cope more effectively with this troublesome pathogen. Past research has emphasized the environment's role in the incidence and severity of diseases associated with *S. sapinea*. Factors that are intrinsically related to the host and, especially, the pathogen have received far less attention. Recent research, however, demonstrating significant genetic variability within *S. sapinea* is a potentially important breakthrough for resolving some of the existing controversy. It is imperative that this line of research be followed to a logical conclusion.

Numerous new techniques in molecular biology have potential to provide further insight into the biology of *S. sapinea* and elucidate the real role of predisposing factors in its epidemiology. More effective control strategies against this pathogen then should be possible.

Literature Cited

1. Bachi, P. R., and Peterson, J. L. 1985. Enhancement of *Sphaeropsis sapinea* stem invasion of pines by water deficits. Plant Dis. 69:798-799.
2. Bega, R. V., Smith, R. S., Martinez, A. P., and Davis, C. J. 1978. Severe damage to *Pinus radiata* and *Pinus pinaster* by *Diplodia pinea* and *Lophodermium* spp. on Molokai and Lanai in Hawaii. Plant Dis. Rep. 62:329-331.
3. Brookhouser, L. W., and Peterson, G. W. 1971. Infection of Austrian, Scots, and ponderosa pines by *Diplodia pinea*. Phytopathology 61:409-414.
4. Brown, B. N., Bevege, D. I., and Stevens, R. E. 1981. Site stress and *Diplodia*-induced dieback and death of hail damaged slash pine. Pages 2.01-2.03 in: Proc. Int. Union For. Res. Organ. Congr. 17th.
5. Burdon, R. D., Currie, D., and Chou, C. K. S. 1980. Responses to inoculation with *Diplodia pinea* in progenies of apparently resistant trees of *Pinus radiata*. Aust. J. Plant Pathol. 11:37-39.
6. Chou, C. K. S. 1976. A shoot dieback in *Pinus radiata* caused by *Diplodia pinea*. I. Symptoms, disease development and isolation of pathogen. N.Z. J. For. Sci. 6:72-79.
7. Chou, C. K. S. 1976. A shoot dieback in *Pinus radiata* caused by *Diplodia pinea*. II. Inoculation studies. N.Z. J. For. Sci. 6:409-420.
8. Chou, C. K. S. 1977. Effect of tree age on *Diplodia pinea* infection of *Pinus radiata*. Plant Dis. Rep. 61:101-103.
9. Chou, C. K. S. 1982. Susceptibility of *Pinus radiata* seedlings to infection by *Diplodia pinea* as affected by pre-inoculation conditions. N.Z. J. For. Sci. 12:438-441.
10. Chou, C. K. S. 1987. Crown wilt of *Pinus radiata* associated with *Diplodia pinea* infection of woody stems. Eur. J. For.

Pathol. 17:398-411.
11. Chou, C. K. S., and Mackenzie, M. 1988. Effect of pruning intensity and season on *Diplodia pinea* infection of *Pinus radiata* stems through pruning wounds. Eur. J. For. Pathol. 18:437-444.
12. Currie, D., and Toes, E. 1978. Stem volume loss due to severe *Diplodia* infection in a young *Pinus radiata* stand. N.Z. J. For. 23:143-148.
13. Dam, B. C. van, and de Kam, M. 1984. *Sphaeropsis sapinea* (= *Diplodia pinea*), oorzaak van het afsterven van eindscheuten bij *Pinus* in Nederland. Ned. Boschbouw. Tijdschr. 56:173-177.
14. Eldridge, K. G. 1957. *Diplodia pinea* (Desm.) Kickx, a parasite on *Pinus radiata*. M.Sc. thesis. University of Melbourne, Australia. 68 pp.
15. Gibson, I. A. S. 1979. Diseases of forest trees widely planted as exotics in the tropics and southern hemisphere. Part II. The genus *Pinus*. Commonw. Mycol. Inst., Kew, and Commonw. For. Inst., Univ. Oxford. 135 pp.
16. Gilmour, J. W. 1964. Survey of Diplodia whorl canker in *Pinus radiata*. N.Z. For. Serv. Res. Leafl. 5.
17. Haddow, W. R., and Newman F. S. 1942. A disease of the Scots pine (*Pinus sylvestris* L.) caused by the fungus *Diplodia pinea* Kickx associated with the pine spittlebug (*Aphrophora paralella* Say.). Trans. R. Can. Inst. 24:1-18.
18. Johnson, D. W., Peterson, G. W., and Dorset, D. W. 1985. Diplodia tip blight of ponderosa pine in the Black Hills of South Dakota. Plant Dis. 69:136-137.
19. Kam, M. de, and van Dam, B. C. 1987. Scheutserfte en bastnecrose, veroorzaakt door *Sphaeropsis sapinea* in Nederland. Ned. Boschbouw. Tijdschr. 59:215-219.
20. Laughton, E. M. 1937. The incidence of fungal disease on timber trees in South Africa. S. Afr. J. Sci. 33:377-382.
21. Marks, G. C., and Minko, G. 1969. The pathogenicity of *Diplodia pinea* to *Pinus radiata* D. Don. Aust. J. Bot. 17:1-12.
22. Minko, G., and Marks, G. C. 1973. Drought index and the sensitivity of *Pinus radiata* to *Diplodia pinea* infection. Res. Act. 72. Victoria, Australia, For. Comm. 47 pp.
23. Nicholls, T. H., and Ostry, M. E. 1990. *Sphaeropsis sapinea* cankers on stressed red and jack pines in Minnesota and Wisconsin. Plant Dis. 74:54-56.
24. Palmer, M. A., McRoberts, R. E., and Nicholls, T. H. 1988. Sources of inoculum of *Sphaeropsis sapinea* in forest tree nurseries. Phytopathology 78:831-835.
25. Palmer, M. A., and Nicholls, T. H. 1985. Shoot blight and collar rot of *Pinus resinosa* caused by *Sphaeropsis sapinea* in forest tree nurseries. Plant Dis. 69:739-740.
26. Palmer, M. A., Nicholls, T. H., and Croghan, C. F. 1986. Fungicidal control of shoot blight caused by *Sphaeropsis sapinea* on red pine nursery seedlings. Plant Dis. 70:194-196.
27. Palmer, M. A., Stewart, E. L., and Wingfield, M. J. 1987. Variation among isolates of *Sphaeropsis sapinea* in the north central United States. Phytopathology 77:944-948.
28. Peterson, G. W. 1977. Infection, epidemiology, and control of Diplodia blight of Austrian, ponderosa, and Scots pines. Phytopathology 67:511-514.
29. Peterson, G. W., and Wysong, D. S. 1968. Diplodia tip blight of pines in the Central Great Plains: Damage and control. Plant Dis. Rep. 52:359-360.
30. Punithalingam, E., and Waterston, J. M. 1970. *Diplodia pinea*. No. 273 in: Descriptions of Pathogenic Fungi and Bacteria. Commonw. Mycol. Inst., Kew, Surrey, England. 2 pp.
31. Rees, A. A., and Webber, J. F. 1988. Pathogenicity of *Sphaeropsis sapinea* to seed, seedlings and saplings of some Central American pines. Trans. Br. Mycol. Soc. 91:273-277.
32. Schweitzer, D. J., and Sinclair, W. A. 1976. Diplodia tip blight on Austrian pine controlled by benomyl. Plant Dis. Rep. 60:269-270.
33. Sutton, B. C. 1980. The Coelomycetes. Commonw. Mycol. Inst., Kew, Surrey, England. 696 pp.
34. Swart, W. J., Knox-Davies, P. S., and Wingfield, M. J. 1985. *Sphaeropsis sapinea*, with special reference to its occurrence in *Pinus* spp. in South Africa. S. Afr. For. J. 35:1-8.
35. Swart, W. J., Wingfield, M. J., and Knox-Davies, P. S. 1987. Factors associated with *Sphaeropsis sapinea* infection of pine trees in South Africa. Phytophylactica 19:505-510.
36. Swart, W. J., Wingfield, M. J., and Knox-Davies, P. S. 1987. Conidial dispersal of *Sphaeropsis sapinea* in three climatic regions of South Africa. Plant Dis. 71:1038-1040.
37. Swart, W. J., Wingfield, M. J., and Knox-Davies, P. S. 1988. Relative susceptibilities to *Sphaeropsis sapinea* of six *Pinus* spp. cultivated in South Africa. Eur. J. For. Pathol. 18:184-189.
38. Wang, C. G., and Blanchette, R. A. 1986. Ultrastructural aspects of the conidium cell wall of *Sphaeropsis sapinea*. Mycologia 78:960-963.
39. Wang, C.-G., Blanchette, R. A., Jackson, W. A., and Palmer, M. A. 1985. Differences in conidial morphology among isolates of *Sphaeropsis sapinea*. Plant Dis. 69:838-841.
40. Waterman, A. M. 1943. *Diplodia pinea*, the cause of disease of hard pines. Phytopathology 33:1018-1031.
41. Wingfield, M. J., and Knox-Davies, P. S. 1980. Association of *Diplodia pinea* with a root disease of pines in South Africa. Plant Dis. 64:221-223.
42. Wingfield, M. J., and Palmer, M. A. 1983. *Diplodia pinea* associated with insect damage on pines in Minnesota and Wisconsin. (Abstr.) Page 249 in: Int. Congr. Plant Pathol. 4th.
43. Wright, J. P., and Marks, G. C. 1970. Loss of merchantable wood in radiata pine associated with infection by *Diplodia pinea*. Aust. For. 34:107-119.
44. Zwolinski, J. B., Swart, W. J., and Wingfield, M. J. 1990. Intensity of dieback induced by *Sphaeropsis sapinea* in relation to site conditions. Eur. J. For. Pathol. 20:167-174.
45. Zwolinski, J. B., Swart, W. J., and Wingfield, M. J. 1990. Economic impact of a post-hail outbreak of dieback induced by *Sphaeropsis sapinea*. Eur. J. For. Pathol. 20:405-411.

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