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Resin-Top Disease of Scots Pine

(*Peridermium pini*)

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Forestry Commission

Figure 1. Forty-year-old Scots pine in Brandon Park, Thetford Chase, Suffolk, showing death and dieback of the upper crown caused by girdling infection by *Peridermium pini*. Branches produced below the canker are quite normal in appearance.

RESIN-TOP DISEASE OF SCOTS PINE

(*Peridermium pini*)

ALTHOUGH Resin-top disease of Scots pine, caused by the rust fungus *Peridermium pini* (Pers.) Lév., has been present for many years in British forests, it is only over the last ten years or so that the severity of damage has significantly affected production in certain areas. The forests worst affected by the disease at the present time are in the area immediately south-west and south of the Moray Firth. Widespread infection also occurs in Thetford Chase, East Anglia, but damage there is generally less severe than in the affected Scottish forests.

The disease is widely distributed throughout pine forests on the Continent, being particularly important in Scandinavia and Germany. A good deal of field and experimental work has been carried out on the disease by Continental pathologists, but in spite of this very little is known about the infection biology of the fungus.

The Fungus and Disease Symptoms

Peridermium pini is a member of the same family of rust fungi as *Cronartium ribicola*, the White pine blister rust, which has caused very extensive damage on five-needled pines in Europe and North America. The life-cycle of *C. ribicola* involves two entirely unrelated hosts, namely five-needled pines and members of the genus *Ribes* (currants), the fungus being unable to spread directly from pine to pine. A rust fungus with such a life-cycle is said to be heteroecious. *Peridermium pini* differs from *C. ribicola* in that infection can spread directly from pine to pine without intervening infection on an alternate host, and is said to be autoecious. The two genera, *Peridermium* and *Cronartium*, are very closely related and the separation is somewhat artificial. In fact, it is fairly well established now that *Peridermium pini* is a form of the heteroecious rust *Cronartium flaccidum* (also known as *C. asclepiadium*), in which infection alternates between two-needled pines, mostly Scots pine, and certain flowering plants, notably peonies and species of *Tropaeolum* (one of which is the commonly grown garden nasturtium). With *P. pini*, the part of the life cycle on the non-pine hosts no longer exists, and the spores produced on the pine are able to infect other pine trees directly. Although Scots pine is the main host, *P. pini* has been recorded occasionally on Corsican pine. As far as is known, *Cronartium flaccidum* is comparatively rare in this country.

Infection of pines by *Peridermium* is apparently most commonly initiated by penetration into healthy needles from spores deposited on to the needle surface, although there is some evidence from experimental work that infection may also be associated with mechanical damage to young shoots. The microscopic filamentous fungal hyphae develop in the needle tissue and spread eventually into the shoot. Early infection causes browning of the affected needles but as no fructifications are produced, the disease cannot be diagnosed at this stage. The mycelium, the name given to the complex of hyphae in the tissues, is perennial and slowly extends further along the shoots. Fructifications are first produced two or three years after initial infection, but are usually not observed in the forest until several years after this when general, more obvious, symptoms in the affected trees are seen. The rate at which infection can advance in the shoot is not known and may be influenced by a number of physiological factors.

The fructifications, known as aecidia, are small, irregular blister-like structures, generally about 3-6 mm. across, and usually occur in large numbers during May and June on the cankers produced by infection. The outer covering of the developing aecidium is a white papery layer, which splits irregularly at maturity to expose the pale orange mass of

aecidiospores within. (Figure 2) The aecidiospores are shaken in large numbers from the aecidia during movement of the affected stems or branches, or may be blown out of the blisters by the wind. The aecidiospores are thick-walled and resistant to desiccation and can be carried by the wind for many miles without loss of viability.

Symptoms of infection are usually first noticed in young crops in which two or three thinnings have been carried out. Infection of lateral shoots frequently results in dieback of the affected branches before the fungus has reached the main stem. Browning of foliage on individual branches in otherwise healthy crowns is frequently the first evidence of attack. If affected branches are examined through binoculars, a dark girdling lesion can often be seen at the base of the dieback, with secondary branches produced below this point still bearing normal green foliage. At a later stage more obvious crown symptoms are produced, as infection which has progressed extensively down branches usually results eventually in girdling of the main stem, producing dieback of the whole crown above this point. During dieback of the crown above the canker, the foliage on branches lower down on the main stem appears quite normal. (Figure 1)

Girdling lesions on the main stem appear from the ground as large blackened cankers, usually about one foot in length (Figure 3), but they may be considerably larger than this. The blackened appearance of the cankers is due to copious exudation of resin from the infected tissues, hence the common name of Resin-top for the disease. During May and June, clusters of orange aecidial blisters may be seen at the periphery of branch and main stem cankers. When blisters are present, the outline of the canker is characteristically of an elongated diamond shape. Because of the progressive development of infection along shoots towards the main stem, canker lesions resulting in dieback both of branches and main stems, are usually seen at the base of a whorl

Figure 2. The fructifications (aecidia) of *Peridermium pini* produced on a canker lesion, showing the white papery outer covering and the pale orange mass of spores within

Figure 3. Infected tree in a young crop of Scots pine showing black resinous lesion on the main stem. Note that the lesion extends above and below a whorl of branches

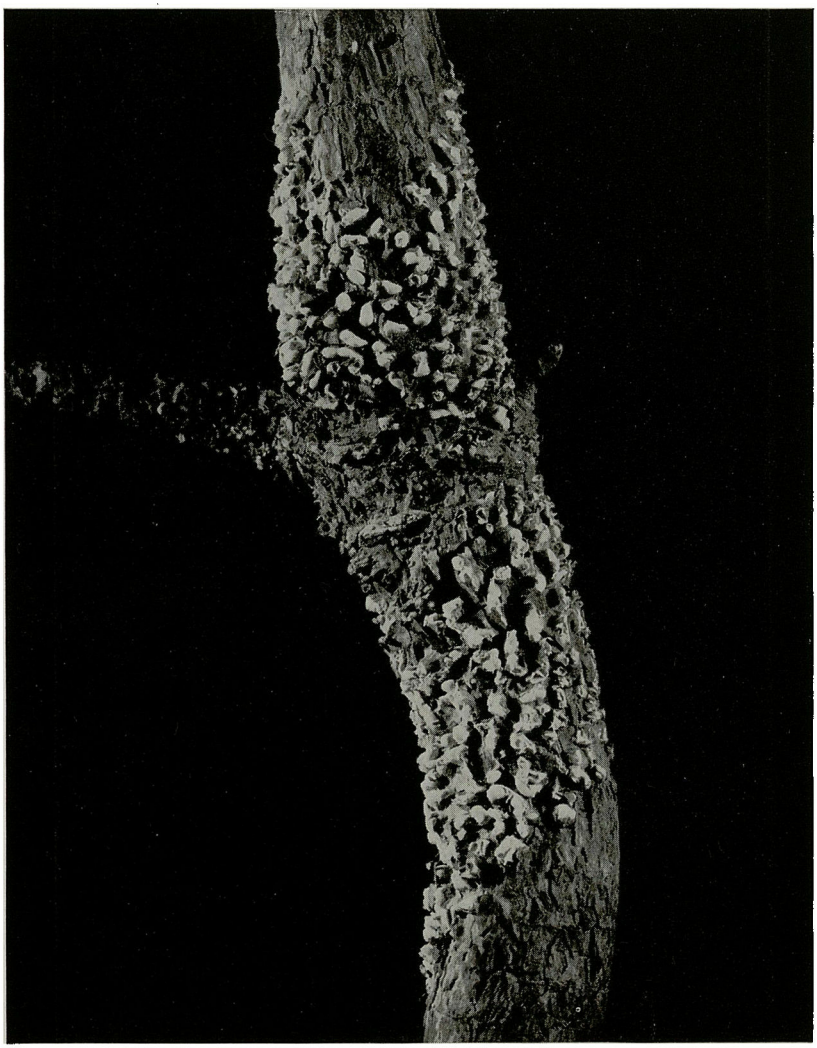

Figure 4. Lesion of *Peridermium pini* on a lateral branch of Scots pine, extending above and below a whorl of secondary branches, showing a large number of mature aecidia

of branches. (Figure 4) Cankers may, however, be formed between branch whorls, presumably as a result of infection of needles borne on this part of the stem or by direct infection through the bark before the development of the hard outer protective layer. Trees on which large cankers on the main stem are formed usually die rapidly following dieback of the upper crown.

At many forests in which the disease occurs, killing of pine caused by root infection by the fungus *Fomes annosus* is common. The symptoms of *Fomes* infection are quite different from those described above. Infection of the root system affects foliage development over the whole crown, the needles becoming progressively shorter and more sparsely produced over a period of years before the eventual death of the tree. Just before, or soon after, the death of the tree, the characteristic leathery bracket fructifications of *Fomes* can be found attached to the base of the stem, usually hidden beneath needle litter. Trees may of course be attacked by both fungi simultaneously (although this occurrence is rare), but the symptoms of *Peridermium* infection are still clearly recognisable in a crown of declining vigour resulting from *Fomes* root infection.

The Incidence and Severity of Damage

The incidence of *Peridermium* attack is generally greatest in crops between 25 and 40 years old. Death due to infection by *Peridermium* is fairly common in the early establishment years, but the importance of the disease at this stage is obscured by the relatively high mortality due to other factors. Very little infection has been seen in thicket stage crops, but it is not clear whether the conditions in the crop at this stage mitigate against disease development, or whether the low level of infection observed is due to the difficulty of access and inspection in Scots pine thicket. The latter supposition is unlikely because at the time of first thinning in plantations of Scots pine, in areas where the disease is prevalent, very little infection is observed. It is quite clear that following first thinning the incidence of infection increases markedly, but the reasons for this are, as yet, not known. It is thought that changes in crown depth, vigour and longevity of branches, together with changes of the microclimate in the crowns following the opening of the crop, may all be important. It seems unlikely that damage to branches during thinning operations plays any significant part in the initiation of fresh infection, and the low level of infection in crops in which first thinning is delayed is more probably associated with physiological and other environmental factors.

In crops more than forty years old, although the incidence of infection may be high, there appears to be a decrease in the rate of development of the disease within the tree. However, mortality may occur regularly in crops up to ninety years old, and as the volume increment in such crops is lower, the volume of timber in trees killed each year, expressed as a proportion of the annual increment of the crop, is high. In very old isolated trees, many infections, some up to twenty years old, may occur in the crown, causing only limited die-back.

In early pole-stage crops in which there is a high incidence of infection, there is a marked correlation between vigour of individual trees and their susceptibility to infection. Surveys in such crops in Scotland showed that approximately 90 per cent. of the infected trees were dominants, and of the remainder there was a decreasing incidence of infection in subdominants, whips and suppressed trees in that order. A similar relationship between vigour and infection occurs in different plantations and on many occasions comparison of adjacent crops of similar age has shown more severe infection in the better-grown crop. Such differences in vigour of crop growth are usually related to changes of soil type and depth, but the provenance (seed origin) of the crops compared may be of importance. Observations on the Continent have shown very clearly that susceptibility to infection by *Peridermium* is strongly linked with genetic factors. The progeny from infected trees is

more susceptible to infection than that from healthy trees, and marked differences in susceptibility may occur between crops of different provenance. The importance of provenance has been indicated by observations in Britain, but in many of the middle-aged stands in affected forests, the exact source of the original seed is not known. There is sufficient evidence to show that resistance to infection by *Peridermium* is a most important factor in any breeding programme with Scots pine, and in the selection of stands to be used as seed orchards.

At the present time no control measures can be recommended to reduce the risk of infection in the crop. Some loss of produce may be prevented in badly infected vigorous crops by more frequent thinning, or the insertion of salvage thinnings into the normal thinning cycle. As mentioned previously, infected trees on which large girdling cankers of the main stem occur, normally die rapidly after the development of dieback in the crown above the canker. The timber in these killed trees soon deteriorates as a result of beetle infestation and the activity of Blue-stain and other fungi. Where the number and volume of the trees seriously affected by disease is high, more frequent thinning is certainly justified and in certain crops even annual salvage thinning may be warranted. To utilize the timber of affected trees, it is important to fell and extract the trees as soon as possible after death, or even *before* the death of the whole tree. In salvage thinnings, trees which are showing large stem cankers associated with wholesale dieback of the upper crown should be taken out, as well as trees which have died completely since the last thinning.

Because of our lack of knowledge of the fundamental infection biology of *Peridermium pini*, this leaflet is of necessity a preliminary account, in which the aim has been mainly to familiarize readers with the appearance and importance of the disease.

The photographs are drawn from the Forestry Commission collection.

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