FORESTRY COMMISSION.

BULLETIN No. 6.

The Phomopsis Disease of Conifers.

BY

MALCOLM WILSON, D.Sc., A.R.C.Sc., F.R.S.E., F.L.S.,

Reader in Mycology, University of Edinburgh,

Consulting Mycologist to the Forestry Commission.

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FOREWORD.

The fungus *Phomopsis Pseudotsugæ*, which has from time to time been known under different names, has a wide distribution in Britain and also occurs on the continent of Europe. It may attack both the Green and the Blue Douglas firs, the European and Japanese larches and *Abies grandis* among the Silver firs. It is possible that other conifers may be subject to the same disease, but this matter requires further investigation.

It is part of the policy of the Commissioners to keep a strict watch for possible enemies of valuable exotic conifers such as Douglas fir and Sitka spruce and to arrange for the investigation of insects and fungi which are or may become of economic importance.

Phomopsis Pseudotsugæ cannot yet be ranked among the fungi which cause serious epidemics among forest trees. It has, however, caused enough damage to warrant the taking of precautions against its spread and it may, of course, become more important as the proportion of Douglas fir in our stands of coniferous timber increases.

Dr. Wilson's account of *Phomopsis Pseudotsugæ* embodies much original research work and is necessarily couched in technical language. A simpler description of the fungus from the point of view of practical forestry will be found in the Commission's Leaflet No. 14 (in preparation).

R. L. ROBINSON,
Commissioner.

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July, 1925.

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THE PHOMOPSIS DISEASE OF CONIFERS.

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THE PHOMOPSIS DISEASE OF CONIFERS.

INTRODUCTION.

The Douglas fir was, in the past, considered to be a tree remarkably free from disease, but unfortunately during recent years it has not sustained its good reputation. It is now attacked in this country by several fungal diseases, and amongst these that caused by *Phomopsis Pseudotsugæ* is perhaps one of the most serious. The disease often attacks the young shoots killing them back for some distance, and there is no doubt that this damage was in the past frequently put down to frost. But since 1910, it has been recognised that the disease could not be accounted for in this way and the fungus responsible for the damage has been under observation since that date.*

HISTORY OF THE DISEASE.

Although the disease has only attracted attention during recent years, there is little doubt that it has been present in this country for a considerable period. It was first definitely associated with *Phomopsis Pseudotsugæ* in 1920 (38 and 39†), but previously to this time diseases of a similar type had been noted on *Pseudotsuga Douglasii*, both in Scotland and on the Continent, and it is very probable that some at least of these were identical with the disease about to be described.

The first record of any such disease appears to be that given by Hartig (14, 15, 16, p. 138), in 1889. This investigator, working in Germany, gives an account of the constriction and death of young branches of *Abies pectinata* caused by a fungus which he described and named *Phoma abietina*. Shortly afterwards in 1890 Rostrup (32), in Denmark, described a disease of the same type which attacks the shoots of *Pseudotsuga Douglasii* and *Abies pectinata*. He considered that the disease was due to the attack of *Phoma pithya* Sacc., a species first described by Saccardo (33), in 1879, on the branches of *Pinus sylvestris* collected in the Botanic Garden of Berlin, by Magnus. Rostrup states, however, that *Phoma pithya* is identical with *Phoma abietina* Hartig.

Prillieux and Delacroix (28, 29 and 30), in France, in 1890, investigated a similar disease on *Abies pectinata*, caused by a fungus which they described and named *Fusicoccum abietinum*

* My attention was first drawn to the disease by Dr. A. W. Borthwick, of the Forestry Commission.

† The numbers refer to the Bibliography, pp. 33–34.

(Hartig) Prill. et Delacr. This disease was also described by Mer (25), in 1890, and a detailed account of the effect of this fungus on its host in France was published by the same author in 1893 (26).

Böhm in 1896 (4) gave an account of the attack of *Phoma abietina* Hartig on the Douglas fir in North Germany. It is noteworthy, however, that the editor and translator of the English edition of the *Diseases of Plants*, by Tubeuf, suggests that there appears to be some confusion in this case between *P. abietina* and *P. pithya* (37, footnote, p. 466).

Meanwhile accounts of similar disease of the Douglas fir had appeared in Scotland. In the Report of the Honorary Consulting Cryptogamist to the Royal Scottish Arboricultural Society, published in 1898 (36), Prof. Somerville records the occurrence of a minute fungus " almost certainly *Phoma pithya* Sacc." on a young tree sent from Ayrshire by Mr. George Leven. Mr. Leven remarks on the unhealthy colour and compressed appearance of the affected part which occurred about half-way up the tree ; the part above the wound was dying, while the lower part was fairly healthy. In the same year, Leven (20) published a note on this and other diseases of the Douglas fir in Scotland. These early records were referred to in a note published by the author in 1921 (41).

In 1907, Prillieux and Maublanc (31) and Henry (17) recorded the disease of *Abies pectinata*, caused by *Fusicoccum abietinum* from the Jura, France, and Maublanc (24) gave an account of the tissue changes produced in the host by the attack of this fungus.

In 1911, Farquharson (8) recorded a disease of the Douglas fir in the neighbourhood of Aberdeen caused by *Phoma pithya* Sacc., and stated that although it occurred commonly it could not be looked upon as a serious pest.

Lind (22), in 1913, in his account of the fungi of Rostrup's herbarium, includes all the species mentioned above under *Phomopsis pithya* Lind and states that in Denmark this attacks *Pseudotsuga Douglasii*, *Abies pectinata* and a considerable number of other conifers as a parasite.

In 1920 (38 and 39) an account of the disease of the Douglas fir in Scotland was published, and for reasons which will be discussed later, the fungus causing the disease was named *Phomopsis Pseudotsugæ*.

In 1921, Alcock (1) published a short account of the disease from the south of England.

The description of a very similar or identical disease of the Japanese larch (*Larix leptolepis*) was published in 1921 (2, 3 and 40), and in the same paper (40) its occurrence on *Abies pectinata* and *Tsuga Albertiana* was recorded. In all these cases of disease, *Phomopsis Pseudotsugæ* was apparently the causal fungus.

Leven, in 1921 (21), gave a description of the different forms

of the Japanese larch and discussed their susceptibility to the disease.

Finally, De Koning, in 1922 (19), has given an account of the disease of the Douglas fir in Holland, caused by *Phoma pithya* Sacc., and there is little doubt that this is similar to the disease found in Scotland.

SYNONYMY AND RELATIONSHIPS OF PHOMOPSIS PSEUDOTSUGÆ.

It will be noticed that although the diseases to which reference has been made are all very similar, yet there is considerable difference of opinion as to the identity of the causal fungus.

In the earlier descriptions of the disease the fungi concerned were usually referred to the genus *Phoma*. Later on it was considered that these should be included in *Phomopsis*, a genus suggested by Saccardo in 1884 (34), and definitely split off from *Phoma* in 1905 (5). In *Phomopsis* the pycnidium is rarely or never globose as in *Phoma*, and the two genera differ considerably in the structure of the pycnidial wall. *Phomopsis* is further characterised by the possession of long, flexuous, subulate or cylindrical sporophores which are much more permanent than those of *Phoma*. In the description of *Phoma pithya* given by Saccardo (34) there is no mention of the presence of sporophores, although these are described as especially obvious in *Phoma abietina* Hartig and in *Fusicoccum abietinum* Prill. et Delacr.

In spite of this these species were all united by Lind (22) under *Phomopsis pithya* in 1913. Von Höhnel had, however, already in 1909 (18) removed *Phoma pithya* Sacc. to his new genus *Sclerophoma*, and regarded it as a twig-growing form of *S. pithyophila* (Cda.) V. Höhn. In 1911 Diedicke (6) stated that *Phoma pithya* Sacc. is a *Sclerophoma*, and accordingly this species should be known as *S. pithya* (Sacc.) Diedicke. Grove, in this country in 1918 (10), suggested that *Phomopsis pithya* Lind and *Phoma pithya* Saac. were not identical and considered the latter to be synonymous with *Sclerophoma pithya* Diedicke. Grove confirmed his suggestion in 1921 (11), and gave the name *Phomopsis abietina* to the species with distinct sporophores.

In the genus *Sclerophoma* the young pycnidium consists of a sclerotium-like mass, the outer portions being brownish and the inner hyaline. Later on the outer wall becomes thin and brittle, and the cells of the inner hyaline part separate to form the spores, but these remain for a long time surrounded by mucilage ; there are no sporophores.

When the disease of *Abies pectinata* was first found in France it was supposed by Mer. (25) and Prillieux and Delacroix (28 and 29) to be caused by *Dothiorella pithya* Sacc., and the two latter authors figured a pycnidium and spores of the fungus

causing the disease under this name (28, Plate XV, Figs 9 to 11). But later Prillieux and Delacroix (30) described it as *Fusicoccum abietinum*, and recognised this as identical with *Phoma abietina* Hartig, and distinct from *Dothiorella pithya* Sacc.

Grove (11, p. 17) suggests that in *Phomopsis abietina* there seems to be a *Phomopsis* state, a *Fusicoccum* state and a *Dothiorella* state ; " and after all, it is by no means improbable that the fungus called *Sclerophoma pithya* is nothing but a sub-sclerotioid state of the same species." This author bases his suggestion on the known dimorphism of *Phomopsis quercina* V. Höhn. and *Fusicoccum quercinum* Sacc., in which " there can be found on twigs and branches of oak every possible transitional state between the two, the spores of the *Phomopsis* state being 7-10 × 1½-2μ, and of the extreme *Fusicoccum* state 15-16 × 3-2½μ." However, as pointed out by Van Luyk (23), connection of this kind between *Sclerophoma* and *Phomopsis* cannot be accepted without further proof.

There appears to be no doubt that *Phoma abietina* Hartig and *Fusicoccum abietinum* Prill. et Delacr. are identical. An examination has recently been made of one of Hartig's specimens of the fungus on *Abies pectinata*, and the spores agree with Hartig's description (14, 15, p. 124 ; 16, p. 138). They measure 12-14 × 5-6μ, showing little variation in size ; they are acute at the ends, rather irregular in form, and are provided with two or more large oil drops towards the ends, so that the protoplasm is almost confined to the central portion of the spore. This agrees with the description *Fusicoccum abietinum* given by Prillieux and Delacroix, and it is noteworthy that these investigators do not mention any great variation in the size of the spores.

The fungus which produces the disease described in the present communication possesses spores 5·5-8·5 × 2·5μ, usually without oil drops, somewhat obtuse at the ends, attached to well-developed sporophores. A large number of specimens have been examined from various parts of England, Scotland and Wales, and little or no variation in spore size has been found.

This fungus has been grown on artificial culture media, and has produced pycnidia which contain spores of a similar shape and size. Grove (11) has described specimens of this fungus from Scotland, and his description of the size and shape of the spores agrees almost exactly with that given above. These facts do not bear out Grove's suggestion that there is a considerable variation in spore size in this fungus. On this account the name *Phomopsis Pseudotsugæ* has been given to the fungus by the present author (38 and 39).

It differs from *Sclerophoma pithya* (Sacc.) Diedicke in the structure of the pycnidium, and in the presence of sporophores, and from *Phoma abietina* Hartig (*Fusicoccum abietinum* Prill. et Delacr.) in the size and shape of the spore (Fig. 5).

It appears that three distinct fungi* have been associated with the disease of conifers which is the subject of this bulletin:—

(1) *Phoma pithya* Sacc. = *Sclerophoma pithyophila* (Cda. V. Höhn. = *Sclerophoma pithya* (Sacc.) Diedicke = *Phomopsis pithya* Lind. (in part). This is possibly the fungus described by Rostrup (32) as causing disease of *Pseudotsugæ Douglasii*. It is described as occurring "*in ramulis corticatis Pini sylvestris*" by Saccardo (34), and is said to be saprophytic on the same tree by Grove (10). It has recently been found as a parasite on young plants of *Pinus sylvestris* in England, causing serious damage.†

(2) *Phoma abietina* Hartig = *Fusicoccum abietinum* Prill et Delacr. = *Phomopsis pithya* Lind (in part) = *Phomopsis abietina* Grove. This is the parasite of *Abies pectinata* described by Hartig (16), Prillieux et Delacroix (30), and Mer (25). It is described as attacking various conifers by Lind (22) and Grove (11).

(3) *Phomopsis Pseudotsugæ* Wilson = *Phomopsis pithya* Lind (in part) = *Phomopsis abietina* Grove (in part). This is the fungus described in the present bulletin.

COMPARISON WITH OTHER SPECIES OF PHOMOPSIS ON CONIFERS.

Phomopsis thujæ has been described on *Thuja occidentalis* by Diedicke (7) from Germany and Petrak (27) has stated that this is a stage in the life-history of *Diaporthe thujana* Petrak which has also been found on *Thuja sp. Diaporthe thujana* differs in several respects from *D. pithya* Sacc.

Phomopsis juniperovora is described by Hahn (12 and 13) as a serious disease of *Juniperus virginiana* in the United States. This has been shown to grow as a parasite on *Thuja occidentalis, T. orientalis* and *Cupressus glabra* but inoculations on species of *Pinus, Pseudotsuga Douglasii, Picea parryana, P. engelmanni* and *Abies lasiocarpa* gave negative results.‡ *Phomopsis juniperovora* differs from *P Pseudotsugæ* slightly in spore size.

THE DISTRIBUTION OF THE DISEASE IN BRITAIN.

On the Douglas Fir.

The disease on the Douglas fir is known to be widespread and will probably be found over the whole of Britain. Up to

* Two other species of *Phoma* have been recorded on *Pseudotsuga Douglasii*, *P. dura* Sacc. on the leaves and *P. Douglasii* Oud. on the cone scales; these, as far as is known, have no connection with the disease described here.

† This species was forwarded to me from Rendlesham, Norfolk, by Dr. J. W. Munro, of the Forestry Commission.

‡ Mr. Hahn informs me that he has recently obtained positive results on *Pseudotsuga Douglasii*, *Picea* sp., and *Pinus* sp., the infections on the first named species being particularly striking.

the present it has been recorded in Scotland from Perthshire, Elgin, Inverness, Aberdeen, Argyllshire, Midlothian, Peebles, Dumfries and Roxburgh; in England from Cumberland and Yorkshire and, as recorded by Alcock (1), from Hampshire, Surrey, Berkshire and Sussex; in Wales from Montgomery.

On the Japanese Larch.

The disease on the Japanese larch has only been seen in four localities—in Inverness, Argyll and Roxburgh in Scotland and Yorkshire in England (2, 3 and 42).

MORPHOLOGY OF THE FUNGUS.

The Pycnidial Stage.

The pycnidia are first covered by the bark and later on break through by means of a slit. This, in the case of young branches of the Douglas fir, is elongated in the direction of the longitudinal axis of the shoot (Fig. 1) but on the older stems the ruptures are oval or rounded (Fig. 2); on the Japanese larch the slits are transversely arranged (Fig. 3). The pycnidia may occur singly but are often found in groups of two or three; they are usually densely distributed over the branch, often only 2–3 mm. apart. The upper bluntly conical part of the black pycnidium projects very slightly through the slit but can be easily seen when examined from above. Seen in section (Fig. 4) the pycnidium is globose-depressed with flattened base 0·1–0·5 mm. in diameter at the widest part, the bluntly conical upper part terminating in the short, wide ostiole which may be as much as 0·1 mm. across; the pycnidium varies in depth (up to 0·3 mm.). The wall is everywhere several cells in thickness; these cells are more or less hyaline and are tinged with green below but are more opaque and darker in colour towards the upper part of the pycnidium where the wall is considerably thicker (up to 50 μ at the ostiole). The cavity of the pycnidium is partially divided up by incomplete septa which project inwards from the wall and which like the latter are made up of almost hyaline cells tinged with green. The number and completeness of the septa vary; in the larger pycnidia they are well developed and may divide the cavity into several loculi, but in the smaller are few and incomplete or even entirely absent. The spores are borne on long colourless sporophores which form a continuous layer over the inner surface of the wall and septa; this layer is only interrupted near the ostiole. The mature spores are elliptic-fusoid, hyaline, obtuse at the ends, 5·5–8·5 $\times$ 2·5–4μ, usually eguttulate but sometimes with two small oil drops or granules; the sporophores are straight, subulate, hyaline, 13 $\times$ 1 — 2μ. The spores vary considerably in shape at different stages in their development. When a mature pycnidium (*i.e.*, one from which the spores will exude naturally if kept

sufficiently damp) is crushed most of the spores are found detached from the sporophores, and the latter form a distinct zone in contact with the wall of the pycnidium. Many of the sporophores appear to be partly disintegrated. The spores can be roughly divided into three groups, as follows:—(1) A few of small size ($5 \cdot 5 \times 2 \cdot 5 \mu$), which stain readily, and are still attached to the sporophores; (2) a large number, free from the sporophores, which have attained their full length ($6 \cdot 5$–$8\ \mu$), but which are comparatively narrow ($2 \cdot 5\ \mu$ wide), and which still stain readily; (3) a considerable number, free from the sporophores, which are mature and have attained their full size ($6 \cdot 5$–$8 \cdot 5 \times$ 3-4 μ), in which the wall is slightly thicker, and which do not stain readily. Spores which naturally exude from the pycnidium are similar to those of the third group; they are not accompanied by the sporophores, for these remain inside the pycnidium attached to its walls. The sporophores can be clearly seen in the young pycnidium but at maturity they become somewhat mucilaginous and partly disintegrate.

The dehiscence of the pycnidium takes place under damp conditions. When moisture is abundant the exuded spores form a rounded, rather glistening, drop of mucilaginous consistency above the slit (Fig. 1, *b*, *c*) but with slightly drier conditions they are squeezed out in the form of a white tendril (Fig. 1, *a*). The ostiole can be seen with a lens after the pycnidium has become emptied (Fig. 1, *d*).

The Non-occurrence of B-spores.

Several species of *Phomopsis* possess two types of spores known as A- and B-spores respectively, these being produced in the same or in different pycnidia. A-spores are usually fusiform or elliptic in shape while the B-spores are longer, rod-like and sometimes curved. In the original description of *Phomopsis Pseudotsugæ* (38 and 39) it was stated that the two types of spores are present in different pycnidia, the A-spores being found in pycnidia on the stems and the B-spores in pycnidia on the leaves or younger parts of one-year-old stems.

As the result of further investigation it has now been concluded that the spores previously described as B-spores do not belong to *Phomopsis Pseudotsugæ*, but are probably those of some species of *Cytospora*. These spores when placed in a decoction made from the leaves and branches of the Douglas fir swell up within 24 hours and, in this condition, closely resemble the spores of *P. Pseudotsugæ* previously described as A-spores. Each then forms a germ-tube which grows out and produces a colourless mycelium. When the spores are sown on Douglas fir decoction agar a mycelium is formed which in about three weeks produces pycnidia containing spores of the same type as those sown. The mycelium is clearly different in habit from that produced by *P. Pseudotsugæ* under similar conditions.

The association of the *P. Pseudotsugæ* and the *Cytospora* sp.

is almost constant when young plants of the Douglas fir are attacked, but the latter has not been found in connection with the cankers produced on older branches. This association is paralleled by that described by Prillieux and Delacroix (30), in which *Fusicoccum abietinum* on the stem of the silver fir is frequently accompanied by *Cytospora Pinastri* on leaves of the attacked plant.

The Ascigerous Stage.

While searching for cankers in a plantation 16 years old, fructifications closely resembling those of *Phomopsis* were found on lateral branches which had been killed by shade. These branches were borne low on the trees, and, apart from the fructifications, showed no signs of infection. On examination the fructications were found to belong to the ascomycetious fungus, *Diaporthe pitya* Sacc. a species which had been described on the continent on the Norway spruce (*Picea excelsa*), but which has apparently not been hitherto recorded in Britain. *Diaporthe* is a large genus, most of the species of which are found growing on trees as saprophytes. Certain species of *Phomopsis* are often found associated with *Diaporthe* Sp., and Saccardo (35) considers that the former are pycnidial stages of the latter, while Lind (22) assigns *Phomopsis pithya* to *Diaporthe pithya*. In the case of *Diaporthe thujana*, found by Petrak (27) on *Thuja* sp., it has been definitely stated that a *Phomopsis* stage known as *P. thujæ* Died. occurs in the life history of the fungus, although the connection of the two forms has not been proved by artificial cultures.

Diaporthe pithya is frequently found on trees of Douglas fir 15-25 years old on the lower branches, which have been killed by shading and here it is evidently saprophytic. It has also been found on young living branches of *Abies grandis*, causing a disease similar to that produced by *P. Pseudotsugæ* on the Douglas fir. It therefore appeared possible that *P. Pseudotsugæ* was merely the pycnidial stage of *Diaporthe pithya*, and in order to test this hypothesis ascospores of the latter fungus were sown on Douglas fir decoction agar. Germination takes place readily and a mycelium is produced quite similar in appearance to that of *P. Pseudotsugæ* and, in about three weeks' time pycnidia are developed containing spores borne on long sporophores identical with those of the latter fungus. It is therefore very highly probable that the two are merely stages in the life-history of the one species, but in order to prove conclusively that this is the case it would be necessary to produce the typical disease on the Douglas fir by infection with spores from pycnidia developed in cultures of *D. pithya*. These infection experiments are now being carried out.

The genus *Diaporthe* is placed in the family Valsaceæ and is characterised by possessing a stroma in which the perithecia are developed. The form and structure of the stroma varies considerably in different species and in the section *Tetrastaga*

of the genus in which *D. Pithya* is placed, it consists of a mass of almost unchanged cortical cells densely filled with hyphæ, the whole being delimited from the rest of the cortex by an irregular black line (Fig. 7, a). The fructifications are scattered in groups on the twigs and emerge through small longitudinal cracks in the bark. About 3–4 perithecia are found in each group ; each is oval in form with a rather flattened base and is pierced by a cylindrical ostiole which projects slightly above the bark (*see* Fig. 7, in which the cavities of two perithecia are seen ; part of the ostiole of the perithecium on the right is shown) ; the wall is membranous and subcoriaceous, black on the exterior and pale brown within. The asci are oblong-fusoid, 50–55μ long and 6–7μ wide ; paraphyses are absent. The spores are arranged rather irregularly in two indistinct rows and are fusoid, hyaline, two-celled, scarcely constricted at the septum, 10–12 by 3·5–4μ, each cell containing two rather large oil drops. The ascospores do not germinate in water, but do so readily in an extract from the Douglas fir ; a germ tube is produced from one or both of the cells.

The Mycelium.

The mycelium as seen in the tissues consists of slender, septate, branched, colourless hyphæ about 2-3 μ across. They are found abundantly in the cortex of infected shoots, forming masses in the resin canals and in the cavities produced by the disintegration of the cells. The hyphæ become slightly yellowish in tissue which has been dead for some time, but they do not disintegrate for some years and can often be found after all sign of the pycnidia has disappeared.

CULTURES ON ARTIFICIAL MEDIA.

The fungus can be readily grown on media made from agar and extract of the Douglas fir both from the pycnospores and the ascospores. Either kind of spore germinates easily in extract of Douglas fir within 24 hours. The pycnospores swell slightly before germination, the contents becoming granular and then each produces a colourless germ-tube, which grows to some length before branching (Fig. 9). The ascospores germinate (Fig. 8) by producing a germ-tube from one or both cells, and here again a considerable length is reached before branching takes place ; the oil drops gradually decrease in size and finally disappear.

The cultures obtained from both kinds of spore are similar in appearance. The mycelium is white and rather superficial, consisting of slender, septate, frequently branched hyphæ 2–3 μ across. Pycnidia are produced in the cultures after about three weeks ; they first appear as minute, rounded structures, at first white but soon becoming black. During their development droplets of water are exuded. These pycnidia are usually well developed and contain numerous partial septa ; they are usually larger than those found on the Douglas fir and may reach 1 mm. in diameter, the spores being of the usual size.

THE DISEASE ON THE DOUGLAS FIR (PSEUDOTSUGA DOUGLASII CARR.).

The Effects of the Disease.

In the papers already published (38 and 39) two types of attack by the disease were distinguished and the subsequent examination of additional material justifies the retention of these, although some modifications in the descriptions of the types are necessary.

Infection of the young Shoot.

The actual seat of the infection is frequently on the young shoot close to its apex, but the resultant disease depends upon the relative position of the attacked shoot on the tree. This damage may be considered under two headings, which correspond to the previously described types of attack.

(1) *Die-back of the Shoot.*—The leading shoot or the terminal shoot of a large lateral branch becomes infected near its apex. The mycelium then spreads downwards and the shoot is killed back for a variable distance, the length of the dead portion depending on a number of factors, of which the relative vigour of the parasite and of the host and the time at which infection occurs are probably the most important. The shoot is often killed back about a length of 9 inches ; this distance in a rapidly elongating specimen may be wholly included in the current year's development, but in a slowly growing shoot may involve two or even more years' growth. In the early stages there is a discoloration, usually a darkening, of the infected shoot ; later on, owing to the death of the cambium, thickening ceases and at the same time there is a partial collapse of the cortical cells. In consequence even before the shoot is completely killed, the extent of the spread of the mycelium is clearly defined by the sudden increase in thickness in passing from the infected to the healthy portion. The small black fructifications of the fungus usually appear on the shoot shortly before its death. Fig. 10 shows a four-year-old tree with the leading shoot killed back for some distance. The length of dead shoot here includes almost the whole of two years' growth and the sudden increase in thickness at the base of the dead portion is clearly seen at (*a*). In Fig. 11 the apical portion of the dead leading shoot of another four-year-old tree is shown, and on this the pycnidia can be just distinguished. Some dead leaves are still attached and on these the fructifications of the species of *Cytospora* already referred to (p. 10) can be seen. The damage brought about by such an attack depends largely on the age of the infected tree. When a two-three-year plant is attacked and the leading shoot killed the tree generally succumbs, but in the case of older individuals the place of the dead leading shoot usually is taken by a lateral and the tree survives. In six-seven-year-old trees cases have been found in which the original terminal shoot has been killed by the disease and its place taken by a lateral,

which has in turn been attacked, and the examination of older trees shows that this phenomenon has sometimes taken place as many as four or five times It is unnecessary to emphasize the very serious set-back to growth which must occur in such cases. As will be explained later, the disease in this way acts as an important factor in bringing about the suppression of trees in plantations.

The course of events when the terminal shoot of a large lateral branch is attacked is similar, although here, of course, the damage to the tree as a whole is not nearly so serious.

(2) *Canker of the Stem.*—The upper part of a small lateral branch becomes infected, and the mycelium spreads downwards through it and enters the tissues of the main stem, in which it spreads upwards, downwards and laterally. The distance which the mycelium spreads in the main stem is limited and the extent probably depends on the factors mentioned above, *i.e.*, time of infection and the relative vigour of the host and of the parasite.

Infection of this kind may take place in a lateral branch close to the apex of the main shoot, as in Fig. 12, where one of the members of the whorl of branches produced at the end of the growth immediately preceding the present year has been attacked.

In this case the dead lateral is about 4 inches in length, and bears fructifications of the fungus. The mycelium, as shown by the discoloration, has spread about 2 inches upwards and 1 inch downwards in the main stem, but the latter does not bear fructifications. In such a case as this, where the main stem at the point of insertion of the infected side branch is thin, the lateral spread of the mycelium is sufficient to encircle completely the former. It is probable, too, that here the mycelium would by its upward growth have reached the apex of the main stem and brought about its death.

The results of a similar attack on a tree four years old are shown in Fig. 13. In this case the lateral through which the infection took place is seen on the right and here again, although the stem is older, it is still comparatively thin and the mycelium has encircled it, killing the cambium and bringing about a complete stoppage of secondary thickening. The place of attack is low on the tree, and most of the leaves are borne on branches above it. The food material produced by photosynthesis in these leaves has been unable, in consequence of the death of the stem, to pass downwards, and has led to increased thickening just above the dead tissues. This abnormal thickening of the stem above the place of attack is particularly characteristic of the disease in trees of 4–8 years old. Fructifications of the fungus are here present both on the infected lateral and main stem. The whole of the tree above the dead portion of the stem will of necessity die from lack of food and water, and even if the branches below survive a tree of this kind would be of no value.

In Fig. 14 the results of attack near the base of a tree 7 years old are shown. Here also the tissues all round the stem have been killed. The base of the small lateral branch through which infection probably took place is seen near the middle of the diseased portion, which involves about 5 inches of the stem. The swelling above the dead portion is not so marked as in the younger tree. In this specimen a certain amount of callus tissue has been produced by the cambium of the still living parts of the stem, and, in consequence, the bark is slightly raised, both at the upper and the lower end of the dead portion. The death of a tree attacked in this way must necessarily take place as no branches are present below the dead portion of the stem.

Fig. 15 shows a part of the stem of a tree 8 years old which has been attacked just above ground level. As usual, the basal part of the lateral branch through which infection took place is seen in the middle of the dead portion, but here the mycelium has only spread laterally about half-way round the stem, and, in consequence, only the bark on one side of the stem has been killed. The resulting appearance somewhat recalls that produced by *Dasycypha calycina* on the larch, but differs in the more regular appearance of the canker. The dead bark is not cracked, except where it abuts on the living tissues, and here the edge is raised by the development of callus tissue under it. Numerous resin blisters are present both on the dead bark and at the edges of the canker, and where these have burst and dried the whitish patches of resin are very obvious (as on the upper part of the specimen). The small black fructifications can be just distinguished by the naked eye scattered over the dead bark.

The effect of the disease on the stem can be seen very clearly when such a canker is cut across transversely, as shown in Fig. 16. In this specimen infection evidently took place through the three-years-old lateral branch seen above. The mycelium probably reached the main stem during the latter part of the fourth season's growth, and the cambium must have been killed during the fourth winter, since, on the side of the stem attacked, four annual rings are complete and the fifth has not been commenced. The fifth and sixth annual rings have been formed on the uninjured side of the stem, but are, of course, absent where the cambium has been previously killed. In this specimen the mycelium spread round about two-thirds of the circumference of the stem, and the points on each side up to which the cambium was killed are indicated in the photograph by the black lines, *a*, *a'*. The arc of cambium which remained alive between the lines *a*, *a'*, on the side of the stem away from the canker then gave rise to the partial fifth annual ring, which is of approximately equal thickness between the lines *a*, *a'*, but which narrows at each side and disappears at *b*, *b'*. The portions of the ring between *a b* and *a' b'* consist of callus tissue produced by growth from the living cambium, and were originally formed under the edges of the dead bark. Similarly in the following year's partial

ring the portion between *b b'* is due to normal secondary thickening, and the parts between *b*, *c* and *b'*, *c'* consist of callus tissue. As a result of the healing process the cambium has been replaced on each side between *a*, *c* and *a'*, *c'*. There is, however, no organic connection between callus tissue and the wood of the annual ring which it overlies, and this is indicated by the curved black lines between *a* and *c* and *a'* and *c'*. There will always be a resultant fault in the timber, even if the wound is completely healed over. The formation of the callus tissue under the bark brings about the loosening of the latter, and the dead piece is finally cast off; an early stage of this process is seen in the figure.

In the cases of the specimens shown in Figs. 15 and 16, where the canker is near ground level and extends round more than half of the stem, the tree nearly always finally succumbs, although a certain amount of healing may take place before death. In the case, however, of an older tree, where the trunk is thicker and the canker relatively smaller, the healing is usually completed and the tree survives. Examples of this are shown in Figs. 17 and 18, in which the specimens were taken from trees 16 years old. In the case of Fig. 17 the lateral branch is four years old, and five complete annual rings were produced in the stem before the cambium was killed. Five partial annual rings are also present on the side of the stem away from the canker, and healing has therefore been going on for five years. Fig. 18 shows a surface view of a somewhat similar canker from which most of the dead bark has fallen away. In this specimen the remains of a lateral branch can, as usual, be seen near the centre of the canker. The dead bark loosened by the development of the callus tissue under it, generally falls away as shown in Fig. 19 and this, in almost all cases, is perforated by a hole near the centre indicating the position of the infected lateral branch.

Cankers of the type shown in Figs. 17 and 18 have been found on the trunks of trees up to 23 years old. It is improbable that they will be discovered on trees of much greater age since it is evident from the above description that after the lapse of a sufficient number of years healing will be completed and ultimately all signs of the canker will disappear.

The callus tissue, produced during the healing, is distinctly ridged, each ridge corresponding roughly to a year's growth and the canker, even when completely healed leaves a scar which is quite different in appearance from the normal bark. (*See* Fig. 20.) This scar only slowly disappears, its disappearance being brought about by the thickening of the bark and the gradual shedding of its outer layers. As the scar becomes older it enlarges, the increase in size corresponding with the increased girth of the trunk and an old scar may be considerably larger than the size of the original canker. This is seen in the stem which is shown cut transversely in Fig. 21. The stem is 22 years old and became cankered during

the 16th year. The canker was completely healed by the end of the 19th year, but the scar is still quite obvious on the exterior, its edges being marked *a*, *a'*. The width of the original canker is shown by the dark line just outside the 15th annual ring; this measures one inch while the width of the scar is $1\frac{3}{8}$ inches.

The canker in question was a small one and healed rapidly. It was probably produced as the result of a wound infection since the plantation from which the specimen was obtained was thinned during its 16th year.

(2) *Infection through Wounds.*

Infection may take place on a branch on which bark has developed. In this case it must be assumed that the infection can only occur through a wound for there is no evidence that the germ tube can penetrate through an uninjured layer of cork cells.

Cankers similar to those shown in Figs. 17 and 18 but where no lateral branch is present are sometimes found, and although no obvious wound is present on the bark it is considered that in these cases there has been some slight injury sufficient to allow infection to take place.

Cankers produced as the result of an obvious wound are of fairly frequent occurrence especially on the trunks of trees from which the lateral branches have recently been pruned off. In some of these cases the point of infection is on the trunk where the mycelium has evidently started from a cut and has extended through a spindle-shaped portion of the bark which, as in cankers previously described, will be ultimately cast off. In many cases, however, cankers are found around the base of fairly large lateral branches which have been cut off flush with the bark. Here infection may have taken place as described on page 14, *i.e.*, near the apex of the lateral with subsequent growth of the mycelium through it to the main stem, but it appears more likely that here also infection took place through one or more small wounds produced on the trunk close to the base of the lateral branch during its removal. It seems improbable that infection of the cut surface of a dead branch will produce a canker although this might occur if the branch was removed while still living.

Infection and Extension of the Disease.

Time and Method of Infection and Period of Extension.

As already stated it may be provisionally concluded that *Phomopsis Pseudotsugæ* is a stage in the life history of *Diaporthe pithya* and, if this is the case, it appears probable that infection, either by pycnospores or ascospores, will give rise to the characteristic disease symptoms on the young shoots and stems. Infection experiments designed to test this supposition are now being carried out.

The area of the young stem upon which infection is possible

can obviously only be definitely determined by a number of such experiments. It is however very improbable that the germ-tube produced by the spore can penetrate through periderm, and since this is formed in the Douglas fir a short distance behind the apex, infection is very probably confined to a short length of the young shoot. Repeated observations have shown that the living shoot can be attacked by the fungus, and there is no evidence that wounds are necessary for infection of the young stem.

Since the fungus is known to live as a saprophyte on dead branches it might be argued that infection takes place on dead stems and that the mycelium spreads from dead to living cells. The fact that a layer of periderm is formed at the junction of the living and dead tissues appears to preclude this suggestion for there is no evidence that hyphae can pass through a layer of cork.

Mature conidia have been found throughout the year and ripe ascospores in the spring, but in view of what is expressed above infection of the young uninjured shoot can probably take place only during the summer and early autumn.

Up to the present it has been found that whenever living tissue of the Douglas fir has become infected, the fructification produced is the pycnidium ; in other words whenever the fungus grows as a parasite it reproduces itself by means of pycnidia and pycnospores. Pycnidia may under certain conditions also be produced by the mycelium when growing saprophytically. It has been frequently found that after a young plant has been killed by the disease pycnidia are produced on those parts, which while they were living did not contain the mycelium. This further infection of the plant may take place in two ways, (1) the cork layer which separates the healthy from the infected portions while living may no longer limit the spread of the mycelium after the death of the whole plant, (2) secondary infection by pycnospores of the previously healthy portions may take place just before or just after the death of the plant as a whole. In both cases the second crop of pycnidia will not be produced until after the death of the whole plant.

As previously described (p. 11) the ascigerous fructifications, *i.e.*, the perithecia of *Diaporthe pithya*, are produced on the dead lower branches which have been killed by shading. Cankers on the main stem bearing pycnidia are frequently found close to these branches and presumably the infection takes place by pycnospores. Ascigerous fructifications are scattered irregularly over the branches, and the latter do not show any examples of the characteristic sudden decrease in diameter associated with the attack of the fungus on living shoots. There is no limitation of the mycelium by means of cork layers and, since these are always present in the infected living branches, it appears probable that infection takes place after the branches are dead or just before death. The conditions of growth of the fungus here would be very different from those obtaining where the living shoot exposed to uninterrupted illumination is

attacked, and the production of the ascigerous stage is no doubt associated with the changed environment.

In the case of living tissue the mycelium spreads throughout the winter following the infection, and ceases to grow when cambial activity commences in the succeeding spring. A layer of periderm is then formed which cuts off the infected shoot or area of bark from the healthy tissue, and soon after this has been produced the diseased tissue dies.

The reason why the mycelium ceases to spread in the spring has not been definitely determined. It has been noticed in several cases that infected tissue does not contain starch, although this is present in adjacent uninfected cells, and it is suggested that the limitation of growth probably partly depends on the diminution of food material available for the growth of the fungus. Starch stored up during the winter is usually converted into sugar and moved to the actively growing tissues in the spring, and it is just at this time that the growth of the mycelium ceases.

On leading shoots the length of stem killed by the fungus is usually 9 to 12 inches. This may include part or the whole of the current year's growth, or it may extend to the two-year-old shoot, and in exceptional cases even further. The length of lateral branch down which the mycelium can grow into the trunk has not been definitely determined, and no doubt varies under different conditions of environment. It is probable, however, that the mycelium can spread throughout lateral branches up to 12 inches long.

Examination of a normally grown Douglas fir shows that the lateral branches produced from buds formed at the end of the year's growth are usually vigorous and elongate rapidly, giving rise to the characteristic whorls of branches. The lateral branches produced from buds between the whorls, *i.e.* from buds developed in the course of the year's growth, are much weaker and elongate very slowly, remaining quite short after the lapse of several years. The lateral of which a part is seen in Fig. 17 is four years old and only about 6 inches in length, and branches up to seven years have been found which are not more than 12 inches long. It is possible then that infection of the main stem may take place by the growth of the mycelium down a lateral branch which is seven years old, and it may be that even older laterals, if sufficiently short, may transmit the infection.

The growth of the mycelium down the lateral branch is probably generally completed during the course of the summer, although it may exceptionally occupy two seasons. The mycelium evidently reaches the main stem some time after the completion of the season's growth, for transverse cuts through cankers never show on the infected side an annual ring which has not attained its complete width. It is suggested that the course of events is as follows :—

Infection takes place on the lateral shoot on the current year's growth in the summer. The mycelium then grows down the lateral shoot, and reaches the main stem in autumn after the cessation of cambial activity. The mycelium spreads in the cortex of the main stem and kills the cambium in the spring before the date at which cambial activity would normally recommence.

It has been already pointed out that trees from six to eight years old when severely cankered near ground level usually die (*see* p. 15). In such cases death of the whole tree only exceptionally takes place in the year following infection and it is generally postponed for a considerable period. The time elapsing between infection and the death of the tree usually varies from one to three years, but may be considerably longer. When the stem is completely ringed by the disease (as in Fig. 14) death usually occurs rather less than two years afer infection, *i.e.*, early in the second summer after attack. It appears that in the first season the tree does not succumb because the food material stored in the stem above the canker is still available and the water supply is not seriously interrupted. If the canker spreads about half-way round the stem death frequently takes place rather less than three years after infection, *i.e.*, in the early part of the third summer after attack. The later case is illustrated by the tree shown in Fig. 15.

Trees do not usually show obvious signs of approaching death during the winter, but those which will die in the following summer are easily distinguishable in the spring. This may probably be explained by the supposition that the diminished supplies of food and water are adequate during the winter when the tree is in a dormant condition, but rapidly become inadequate on the approach of spring when the metabolic processes and transpiration are greatly increased. The leaves of these dying trees become yellowish and, in April, they can in consequence be easily distinguished even at a considerable distance. By the middle of the summer the leaves are reddish-brown and the tree is completely dead.

The above discussion has a direct bearing on the question as to what is the greatest age at which a main stem can become infected through a lateral shoot. The lateral shoot is always one year younger than the main stem which bears it*, and if we suppose that the maximum age of the lateral which can transmit the infection is ten years, development of a canker on the main trunk cannot commence at a point where the latter is more than eleven years old. Observations made up to the present indicate that this is an approximately correct estimate. A leading shoot or large lateral shoot infected during the summer generally shows

* This statement is based on the supposition that dormant buds are not present on the Douglas fir. It appears that under normal conditions all buds do grow out in the year after their formation, except those which produce Lammas or second growths in the same year as they are formed.

signs of disease early in the following spring, the shoot becoming discoloured and the leaves turning brown. Pycnidia begin to appear usually about a year after infection, *i.e.*, soon after the extension of the mycelium has ceased. In the case of infection of the main stem through a lateral branch, pycnidia usually appear on the canker late in the summer after its formation, *i.e.*, rather more than a year after the infection of the lateral. This lateral branch usually bears fructifications, but where it has been dead for some time they cannot always be found. In all cases hyphæ are present in the cortex of the lateral. It is known that the fructifications disintegrate fairly rapidly after the discharge of the spores and, after the lapse of a few years, it is difficult or impossible to find any trace of them. This fact no doubt explains their absence in some cases, but in others it is possible that they have never been produced on the infected lateral, especially where the latter is small and thin, and where, in consequence, the food supply available for the fungus is scanty. The specimen shown in Fig. 12 was collected early in the spring and here the lateral bears fructifications, but these are not present on the main stem. Infection of this lateral evidently took place in the previous summer and the pycnidia in this case must have been produced rather less than a year after infection.

In the case of cankers produced as the result of infection through wounds the sequence of events is probably not so regular. Pruning usually takes place during the winter, and the mycelium in cankers resulting from wounds made and infected at this time probably ceases to extend in the following spring. Pycnidia are then probably produced late in the following summer, *i.e.*, about eight to nine months after infection.

Description of an Abnormal Canker.

An investigation has been made of a remarkable canker on a specimen of Douglas fir of which the history is unknown. The area of dead bark is spindle-shaped and is 34 ins. long by 3½ ins. wide in the broadest part. Several lateral branches forming part of a whorl produced at the end of a year's growth are borne near the centre of the canker, which spreads over portions of two years' growth in length. There is a series of four concentric ridges on the surface giving the impression that the canker had been extending during four years. Although no spores were discovered, hyphæ and remains of fructifications were found in the dead bark and there is no doubt that the canker was caused by *Phomopsis*.

On cutting through the stem it was found that the age is 14 years, made up in the following way. Starting from the centre there are eight well-developed annual rings followed by a thinner one, the ninth, and the tenth to fourteenth are extremely thin, in some cases only 7–8 tracheids wide, but each consisting of spring and autumn wood. The tree evidently suffered a check in the ninth year and from the tenth year onward practically

did not increase in thickness. The mycelium reached the main stem during the autumn of the tenth and killed the cambium over an area enclosed by the innermost of the concentric ridges referred to above; the ridge was produced by a slight development of callus tissue. The mycelium evidently continued to spread during the eleventh and succeeding years, the ridges marking the limit of each year's extension. The cambium was killed each year over a larger area.

Previously to examining this specimen, no instance had been found where the mycelium had continued to live in the main stem for more than one season. The hyphae were considered to spread during the winter only and to receive a check at the cessation of the dormant period of the host, the death of the fungus taking place before the succeeding winter. This course of events is still considered to be the normal one and the unusual condition of the specimen in question is believed to be due to its extremely slight growth in thickness after infection had taken place. It was evidently in a practically dormant condition throughout the whole period from the tenth to fourteenth year. The fungus, in consequence, received no check on account of renewed activity of the host in the spring and therefore continued to extend. This will account for the unusually large size of the canker.

Conditions under which Infection takes Place.

From the practical point of view it is important to determine whether any predisposing factors to the disease exist; in other words, whether trees weakened by other causes are more liable to become infected than those which are quite healthy.

Since there is no doubt that infection often takes place through wounds, injuries to the trees, however produced, must evidently be looked upon as predisposing factors. The possibility of injuries produced by insects leading to attack by the disease has already been suggested by Leven (21) in the case of canker of the Japanese larch and this suggestion is equally applicable to the Douglas fir. In one severely attacked plantation the young trees had also been injured by weevils (*see* p. 24) and the wounds produced by these insects may have served as points of entrance for *Phomopsis*. Attacks by rabbits or deer may, in the same way lead to the disease. The frequent occurrence of cankers just above gound level in trees six to eight years old (*see* p. 24) may be the result of infection through wounds caused by rabbits; injuries near the collar are, however, also sometimes produced during planting and these may similarly lead to infection by the disease. Pruning as a predisposing factor is fully discussed subsequently (*see* p. 32). It is possible that weakening of the tree, apart from actual injury, serves as a predisposing factor. In one case of severe infection shortly after planting out, the trees had been grown in a sheltered nursery and planted out on an exposed hillside in January. Such treatment would probably result in

weakening the trees and this may perhaps account for the severity of the attack. In other cases plantations previously damaged by fire have been found to be extensively diseased and, here again, the resulting weakening of the trees may have proved to be the predisposing factor. The possible effect of suppression as a predisposing factor is discussed subsequently (*see* p. 27).

The planting of trees in situations unsuitable as regards altitude or exposure may lead to increased infection, but at present there is not sufficient evidence to make any definite statement in this connection. In the case of many diseased trees, however, there is no obvious predisposing factor, and it cannot be definitely concluded at present that weakened or badly grown trees are more liable to attack.

Changes in Microscopic Structure induced by the Disease.

Both in the case of die-back of the young shoot and canker of the stem a layer of cork is found between the healthy and the infected tissue. The development of the layer has not been followed out in detail and it is not known exactly at what time it arises, but it is present as a zone four to six cells in width when fully developed. In the case of the die-back of the shoot, the cork layer is not developed across the cortex regularly at right angles to the longitudinal axis, but the diseased tissue dips downwards in tongue-like projections into the healthy tissue. One such projection, consisting partly of infected phloem and partly of cortex, generally lies against the woody cylinder and another is frequently found near the middle of the cortex.

Abnormal wood is produced by the cambium in the vicinity of the infected tissue. In the shoot this extends some distance downwards below the cork layer which limits the diseased tissue. It consists of almost isodiametric cells of irregular size provided with simple pits ; the walls of these are of moderate thickness and are lignified. It merges gradually into wood of the normal structure, but it can often be traced for a centimetre or more below the limiting cork layer. Similar abnormal wood has been described by Mer (26) and Maublanc (24) in *Abies pectinata* attacked by *Fusicoccum abietinum*.

The Extent of the Damage caused by the Disease.

In the Nursery.

Infected plants have been found in a number of nurseries and, in the majority of these, the disease is in the form of a die-back of the leading shoot (Fig. 10), but in some cases the young plant may be attacked just above ground level, the infection passing into the main stem from a lateral branch (Fig. 13). The youngest plant on which the disease has been found was four years old, but the attacks are most prevalent where the plants are kept in the nursery until five or six years old, *i.e.*, in two-year-three-year,

and two-year-four-year plants. The infected plants are found scattered irregularly in the lines and are usually not numerous. It has been found impossible to estimate the percentage of diseased plants in any nursery since, in the majority of cases, the infected plants are pulled out as soon as observed or the diseased stems are cut off. In the latter case a new leader is generally formed and if re-infection does not take place, the plant may, in course of time, develop sufficiently to be suitable for planting out. The removal of diseased shoots is a regular practice in some nurseries but plants treated in this way are always smaller than healthy ones of the same age and would be placed in the second grade or discarded when the trees are planted out.

As already pointed out, attacks of the disease in the nursery have in the past been usually regarded as due to frost damage.

In Plantations shortly after planting out.

A large number of trees may suffer from the disease in the form of die-back of the leading shoot within a year of planting out. This may be due to the fact that the trees are already infected when planted out but do not show any obvious signs of the disease until the following summer. In a young plant infected in the autumn the mycelium as usual spreads in the shoot during the winter but may not produce discoloration of the stem or leaves until active growth commences late in the following spring. As planting out usually takes place in March or April a diseased plant may in this way escape notice.

It is probable, however, that a number of plants become infected during the first or second summer after planting out and it is possible that the disturbance produced by the digging up and re-planting predisposes the plant to attack. There is some evidence that when this operation is carried out at an unsuitable time the predisposition to attack is more marked. In a plantation in which the trees were planted out in January it was estimated two years later that almost half the plants were attacked by *Phomopsis*. But in this case other factors probably influenced the large amount of disease for the plants were removed from a sheltered nursery and planted out on an exposed hillside and, later on suffered from a rather severe attack by weevils.

The plants attacked by the disease do not necessarily die for in a good many cases the die-back does not extend very far and the dead shoot is replaced by a lateral. But it is not unusual in several localities in Scotland to find that, within two years of planting out, 10 per cent. of the total number of plants have been killed by the disease.

In Plantations 6–10 *Years old.*

Extensive damage is frequently caused by the disease in plantations 6–10 years old. Here the most serious damage consists of cankering near the ground level usually as the result

of infection of the stem through a lateral shoot but perhaps in some cases through wounds caused during planting out (Figs. 14 and 15). Die-back of the leading and lateral branches is also not uncommon. A plantation in Perthshire about 6-8 years old was examined in the spring of 1920 and it was estimated that half of the total number of trees had been killed by the disease. The dead trees were removed and burnt and the spaces were filled up by planting fresh trees. In the following September about 20 per cent. of the trees were showing signs of attack and these included some of those planted in the previous April. In the case of another plantation of similar age in the same district practically all the trees were infected either near ground level or as a die-back of the leading or lateral shoots.

In a plantation about eight years old situated in the south of Scotland it was noticed that a considerable number of the trees were of a bushy habit and possessed no distinct leader; these trees were also distinctly shorter than those having the normal appearance. An examination showed that this unusual habit was due to the fact that the original leading shoot had been destroyed by *Phomopsis* and that the lateral which had taken its place had also been killed by the disease. In some cases a third and even a fourth lateral which had assumed the position of the leading shoot had been killed in turn. In such cases the tree usually survives but it is obvious that there will be a great decrease in the height increment as the result of successive attacks.

In Plantations 16–20 *Years old.*

An investigation of the distribution and the amount of damage caused by the fungus has been made in a plantation of Douglas fir in the neighbourhood of Peebles. The plantation is 16 years old and is at an altitude of about 700 feet in a sheltered position. Distance of planting is about 4 feet, and the trees are now about 18 feet high. The attack, which is almost entirely confined to the lower parts of the trees up to about 6 feet above ground level, can be described under three headings as follows :—

(1) A few of the apices of the lower lateral branches have been killed back and bear the pycnidial fructifications.

(2) Cankers are found on the trunks and occasionally near the bases of the larger lateral branches; pycnidial fructifications were originally present on these.

(3) Ascigerous fructifications are present on the lower lateral branches which have been killed by shade.

The damage done under (1) is negligible. An estimate has been made of the number of trees attacked as described under (2). The cankers are mostly present on the first 4 feet of the trunk, but a few were found between 4 and 6 feet. In the case of one tree eight cankers were present up to 3 feet above ground level. Three hundred trees were examined; in the first hundred 20 were attacked, 18 in the second hundred and 22 in the third

hundred, giving a mean of 20 per cent. In many cases more than one canker was present on the attacked trees; the attacked trees also obviously occurred in groups. Of the 300 one tree bearing six cankers was dead and had evidently succumbed as the result of the attack. There were a number of gaps amongst the trees, showing that some had previously been removed The 20 per cent. can be taken as a minimum, since only obvious cankers were considered; probably a number which had completely healed were omitted, and some may have been entirely missed owing to their small size.

A plantation in the Tay valley at an altitude of about 120 feet, near Dunkeld, has been examined in a similar way by Dr. Mark L. Anderson of the Forestry Commission and I am indebted to him for the following results. The plantation is 23 years old, and the planting distance about 4 feet.

" Of the total 388 trees 76 were attacked, *i.e.*, 19·6 per cent. These 76 attacked trees consisted of 17 suppressed trees and 59 main crop trees. The number of suppressed trees in the whole crop was 62 and since 17 of these were attacked the percentage of attack among suppressed trees was 27·4 per cent. The total number of main crop trees was 316 and of these 59 were attacked, *i.e.*, 19 per cent. The percentage of attack was therefore considerably greater among suppressed trees. The 76 mentioned above were undoubtedly attacked. More may have been, but the scars may have completely healed. The scars were from 2 inches to over a foot long. Other dead and suppressed trees had been previously removed. There was one scar at about 7 feet up and not more than three between 6 and 7 feet. All the others were below 6 feet from the ground. In the felled sample trees there was no evidence at all higher up the stem. It required careful investigation to discover many of these scars as, owing to the comparatively slow girth increment, they had not cast off their skin of dead bark.

" Starting at the N.E. side, I was at first under the impression that all the scars were at breast-height. As I proceeded to the centre of the wood, I found most of the wounds nearly on the ground or about a foot above it. As I neared the S.W. side, there were very few scars low down; they were again at about breast-height. I thought that this indicated a spread of the disease from the centre outwards and that the disease must have been on the plants when put out. The attack could not be called severe and I think that the locality is well suited for Douglas fir. The disease noticeably occurs in groups of trees."

The results obtained from the examination of the two plantations grown under rather different climatic conditions are strikingly similar. The disease is known to be widespread in both localities, and the similarity of result may be connected with the similar sylvicultural methods applied in each plantation.

Suppression caused by the Disease.

It has already been pointed out that successive attacks of the disease on the leading shoots of a young tree greatly lessen increase in height, and the question arises whether suppression in older plantations is due to the disease. Examination was therefore carried out of a number of suppressed trees removed from a plantation about 18 years old in which the disease was known to be widespread. It was found that in the majority forking of the main stem had occurred one or more times, and that, while in some it had taken place at various heights, including the middle portion, in most it was confined principally either to the upper or to the lower parts of the tree.

Repeated forking in the upper portion had produced trees with bushy tops, while in those in which the forking was confined to the lower portion fairly strong branches had been formed not far above ground level. In some cases the cause of the forking could not be determined, but in many others there was no doubt that it was due to the death of the leading shoot as the result of attack by *Phomopsis*. In the latter the remains of the dead shoot still bearing traces of fructifications were often found at the fork. The suppressed trees frequently bear a number of cankers and it is noteworthy that these cankers although usually found on the lower parts of the stems are not altogether confined to this portion.

A number of suppressed trees bearing large branches near the base were carefully examined. In these death of the leading shoot had usually taken place several times within five feet of ground level, and each time that this had occurred usually two of the laterals had grown out strongly; of these one had ultimately become the leading shoot, but the other had grown for some time and formed a branch of considerably more than usual size. The terminal shoots of these branches had been generally killed by *Phomopsis*. It is obvious that such trees were severely attacked by the disease soon after they were planted out and that in consequence growth must have been greatly retarded. In these cases there is little doubt that the early retardation of growth caused by the attack by *Phomopsis* was responsible for the ultimate suppression.

In the case of trees with bushy tops the course of events is more difficult to follow. The condition is due to repeated forking following on death of the leading shoot caused by *Phomopsis*. The attack by the disease must have taken place for the most part at a fairly late stage, since there are usually few or no cankers near the base of the stem. The attack, however, must have commenced before the canopy closed, since vigorous growth has taken place in the upper part—growth which would not have gone on if the tree had been shaded. There is little doubt that in these cases suppression is due to the lessened growth increment just before closure of the canopy.

It might be argued that attack by *Phomopsis* is not the cause but the result of suppression, *i.e.*, that trees weakened by some other factor are more liable to attack than trees which are otherwise healthy. This possibility is not excluded and it is strengthened by the examples of supposed predisposition to the disease which have already been described (*see* p. 22). The figures of percentages of attack on main crop and on suppressed trees given on p. 26 are in the same way open to two interpretations.

The two views are, however, not mutually exclusive and, although in the opinion of the author it appears more likely that the disease is the cause of suppression, cases may occur in which the disease attacks trees which, already weakened by other factors, will in any case be ultimately suppressed.

The Effect of the Disease on the Quality of the Timber.

The description of the healing of a canker has already been given (p. 16), and it is obvious that after the lapse of a sufficient number of years all trace of the canker on the exterior of the trunk will disappear. There will, however, always be discontinuity between the normal wood and the wood produced by the wound cambium, and this is marked by a dark line seen both in transverse (Figs. 16 and 21) and in longitudinal radial section (Fig. 22). A canker must evidently always produce a fault in the timber, the extent of which will depend on the size of the canker. The effect of a single canker is comparatively small, but where the cankers have been large and numerous the timber will be appreciably weakened.

THE DISEASE ON THE BLUE DOUGLAS FIR

(*Pseudotsuga glauca* Mayr.).

Specimens of the disease have been occasionally found on *Pseudotsuga glauca* and on the variety *cæsia* of *P. Douglasii*. The disease on both these trees is similar to that described on *P. Douglasii*. There is no definite information available at present as to the relative susceptibility of the two species.

THE DISEASE ON THE JAPANESE LARCH

(*Larix leptolepis* Murr).

Effects of the Disease.

The disease on the Japanese larch has only been found in the form of cankers on the main stem similar to those found on the Douglas fir. The cankers on the larch, however, are often considerably larger, reaching 10 inches in length and 5 inches wide, and in the majority of cases there is no lateral branch in the centre (Fig. 23). The cankers are usually more obvious than those on the Douglas fir owing to the fact that

the diseased area is rather more sunken and that usually there is a marked exudation of resin around its margin. As already described the fructifications are elongated horizontally and are rather larger than those on the Douglas fir (Fig. 3). These modifications in shape and size of the fructifications probably depend on differences in structure of the bark of the two trees and not on dissimilarity of the fungus. As in the Douglas fir the diseased is separated from the healthy tissue by a layer of cork cells.

A rather older canker is seen in Fig. 24, and in this the dead bark is raised at its edges by the formation of callus tissue beneath. The bark usually cracks soon after it is dead and in consequence is not cast off as a whole. This is seen in the older canker shown in Fig. 25. Healing by the formation of callus tissue goes on as in the Douglas fir, but the process is not so rapid and is often not completed.

Cankers on the Japanese larch caused by *Phomopsis Pseudotsugæ* may be distinguished from those produced by *Dasycypha calycina* by their more regular form and the sharp delimitation between the healthy and diseased tissue as well as by the difference in appearance of the fructifications of the two fungi. Even in the old cankers where the latter have disappeared the distinction is not difficult for, in the case of *Phomopsis*, the callus tissue is formed more freely and more regularly than it is in the *Dasycypha* canker; the regular casting-off of the bark in the former also serves as a good distinguishing character.

The fungus has not been found as a saprophyte on dead branches of the Japanese larch either in the pycnidial or the perithicial condition.

Conditions under which Infection takes place.

Examples of canker of the Japanese larch have only been found in four plantations situated far apart, and in all these the trees were of approximately the same age, viz., 12 to 18 years. Usually the cankers appeared a year or two after the plantation had been thinned and the dead lateral branches removed. As already described, there is usually no lateral branch associated with the canker and it must be concluded that infection, in the majority of cases, took place as the result of wounding. The fact that cankering has usually been found shortly after thinning is very suggestive, and there is little doubt that it results from accidental wounding of the trees during the removal of the thinnings and the dead lateral branches.

No "die back" of young shoots has been found and no cankers have been discovered before thinning has taken place, although careful search has been made for these on trees closely adjoining older plantations in which cankers have been found. It therefore appears improbable that infection has taken place from diseased larch trees. The four plantations in which

cankers were found were, however, in each case in close proximity to Douglas firs attacked by the disease, and it is probable that infection of the larches has taken place from the fungus on the Douglas firs. Definite evidence on the point can only be obtained by infection experiments and these are now being carried out.

Leven in 1921 (21) distinguished three types of Japanese larch—the "fissure barked," the "flake barked," and the "smooth barked." He points out that, up to the present, the flake-barked type has proved to be immune to the *Phomopsis* disease, while the other two types have been badly affected. Whether this will prove to be the case in other districts is not at present known, but there is no doubt that it holds good in one of the infected plantations.

THE DISEASE ON THE EUROPEAN LARCH (*Larix europæa* D.C.).

Only one example has been found of the disease on the European larch. This was obtained from a nursery near Aberdeen and consists of a five-year-old tree which has been completely ringed by a canker 6 inches above ground level (Fig. 26). The canker, which bears the pycnidial fructifications, is $1\frac{1}{2}$ inches long; it also bears fructifications of *Dasycypha calycina*, but it is probable that attack by this fungus has followed the infection by *Phomopsis*. As regards appearance and structure the canker closely resembles those found on Douglas firs of a similar age, but there is no lateral branch through which the infection can have taken place. There is also little or no swelling of the stem either above or below the canker. Trees of the Douglas fir attacked by the disease were also present in the nursery.

THE DISEASE ON ABIES GRANDIS Lindl.

Only one example of the disease on *Abies grandis* has been found. This occurred on a tree about nine years old, the disease causing a "die-back" of the leading shoot for about 12 inches and also producing elongated cankers of irregular shape on the stem. As in the case of the Douglas fir, there is a marked increase in thickness in passing from the diseased to the healthy tissue and the diseased portions are delimited by cork layers. Fructifications are present on the diseased portions, but the specimen is unique in that these consist of perithecia and not of pycnidia; they are, in fact, the ascigerous stage of the fungus *Diaporthe pithya*. This is the only example found up to the present in which the fungus, after having killed the shoot, has reproduced itself by perithecia. Whether this is the normal condition for this host species cannot, of course, be determined until further specimens are found. It is in any case additional

indirect evidence of the identity of *Phomopsis Pseudotsugæ* and *Diaporthe pithya.*

The perithecia and asci agree closely with those found on the Douglas fir and the stromata bearing the fructifications are similarly delimited by a black line (*see* p. 12).

THE DISEASE ON OTHER SPECIES.

Examples of " die-back " of young shoots have been found on *Abies pectinata* D. C., *Tsuga Albertiana* Sénéc, and *T. Sieboldi* Carr. but it is not at present certain whether, in these cases, the damage has been caused by the disease just described. The dead shoots in all cases bear fructifications of a *Phomopsis* sp. which closely resemble *P. Pseudotsugæ,* but further investigation is required before any definite statements can be made as to their indentity with this species.

THE CONTROL OF THE DISEASE.

At present any measures suggested for the control of the disease must be provisional and are, for the most part, indirect. As already described, the disease is most dangerous to young plants, and it is important that infection should be prevented in the nursery and that all trees planted out should be healthy and in good condition. In this connection the site of the nursery is of importance. A nursery placed at some distance from any plantations is less liable to infection than one in close proximity to existing woods.

Any diseased plants in the nursery should be at once removed and burnt. The practice of removing diseased shoots and leaving the plants is not one that can be recommended. The use of the branches for shading the seedlings or for protection against frost should not be allowed since, if these are diseased, almost ideal conditions for the infection of the plants are thereby obtained. Planting should be carried out carefully and at the most suitable time in order to lessen, as far as possible, any predisposition to the disease.

The prevalence of cankers not far above ground level has already been noted and probably a certain proportion of these result from wound infections. The wounding in such cases may be brought about in various ways—by insects, animals, mechanical damage during planting, etc.—and any measures which remove these sources of injury will at the same time lessen the risks of infection by the disease. Any young trees seriously injured or killed by the disease should be removed from the plantations and burnt and this should be carried out before beating up takes place. The planting of a healthy tree beside one already killed by the disease should not be allowed ; it is known that the fungus can go on living and can produce its fructifications and spores after the death of the tree has taken place, and the dead tree therefore remains a potential centre of infection for some time.

There is no doubt that a considerable amount of cankering results from the pruning of the side branches of trees fifteen to twenty years old and, although it is probable that this cannot be entirely avoided, it can undoubtedly be considerably lessened by the exercise of care in this operation. It is improbable that infection takes place on the cut surfaces of dead branches and if these only were removed without any wounding of the trunk there appears to be no reason why cankers should be produced. Infection probably does take place as the result of the pruning off of living branches and through the formation of accidental wounds on the trunk. When a saw is used for the removal of the branches there is no reason why wounds should be produced on the trunk if sufficient care is taken. Cankers are, however, frequently found at the base of branches removed by a saw and these probably result from small wounds formed on the trunk near the base of the branch when the latter is cut off almost flush with the surface. In the endeavour to remove as much of the branch as possible the cut should not be made so close to the trunk as to risk wounding the latter. The use of a billhook for pruning is deprecated for in this case, especially with an inexperienced worker, the risk of wounding the trunk is greatly increased. Care should be taken not to remove any branches which are still living.

As already described, the ascigerous stage of the fungus is found on branches killed by shading, and the removal and burning of the slash produced by pruning would certainly diminish the amount of material capable of spreading infection. In view of the expense and difficulty involved in such removal it is doubtful whether it should be recommended, especially in districts where the disease is already widespread. The case of suppressed trees is, however, on a somewhat different footing and since these are known to be frequently infected with both the pycnidial and perithecial stages of the fungus their removal is recommended. Generally speaking, since the fungus is known to exist as a saprophyte, the removal and destruction of dead trees should be undertaken wherever it is practicable.

The conditions under which the Japanese larch becomes infected have already been discussed (p. 29), and it is only necessary here to emphasize the necessity for care in removing suppressed trees and dead branches in order to avoid wounding the trunks.

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PLATES I–XII.

All figures refer to *Phomopsis Pseudotsugæ* on *Pseudotsuga Douglasii* unless otherwise stated.

PLATE I.

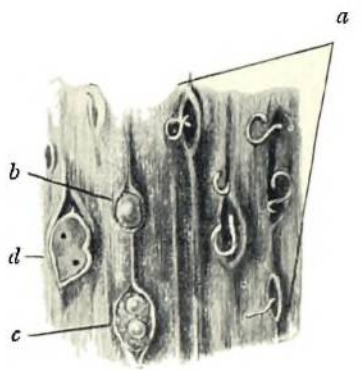


FIG. 1. Portion of a young shoot showing pycnidia; (*a*) spores in the form of mucilaginous tendrils emerging from several pycnidia; (*b*) a single pycnidium from which a rounded mass of spores has just emerged; (*c*) a group of two pycnidia from each of which a rounded mass of spores has emerged; (*d*) a group of two empty pycnidia each showing an ostiole (as a rounded black dot). x about 16.

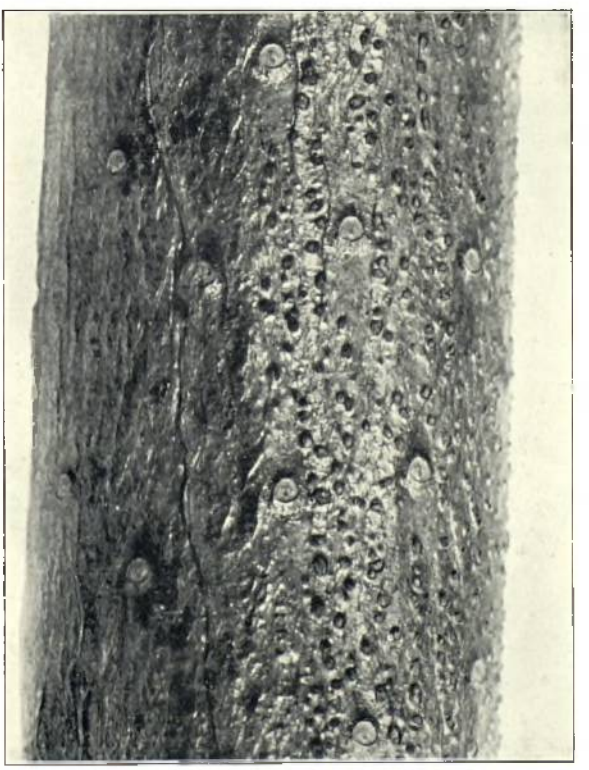


FIG. 2. Pycnidia on the bark of a cankered branch 4 years old; the margin of the canker is seen on the left. x about 5.

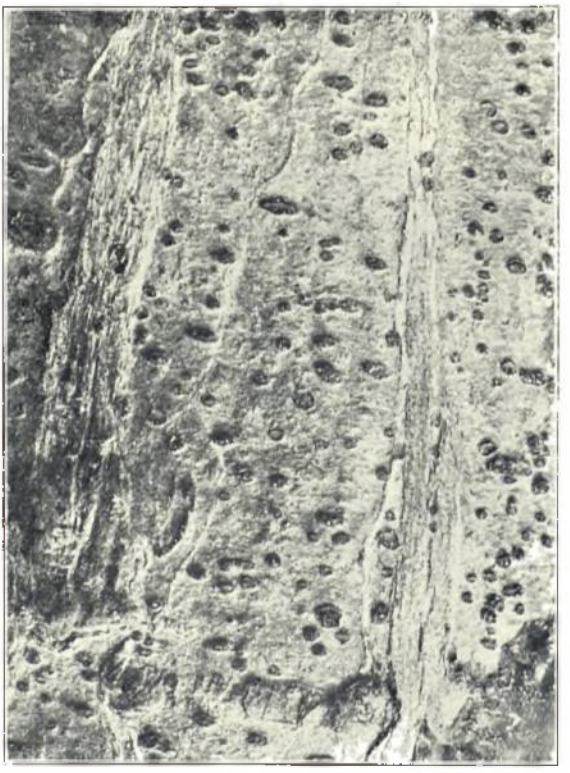


FIG. 3. Pycnidia on the bark of a cankered branch of *Larix leptolepis* 14 years old. x about 4.

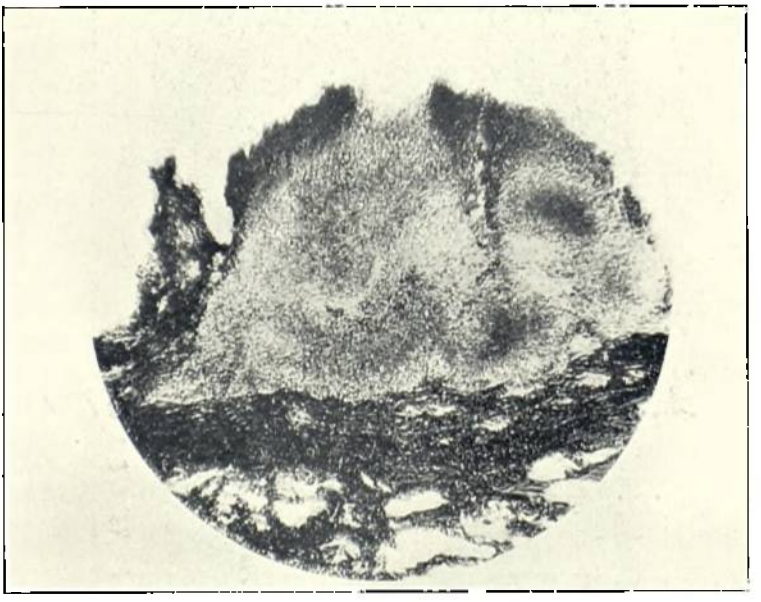
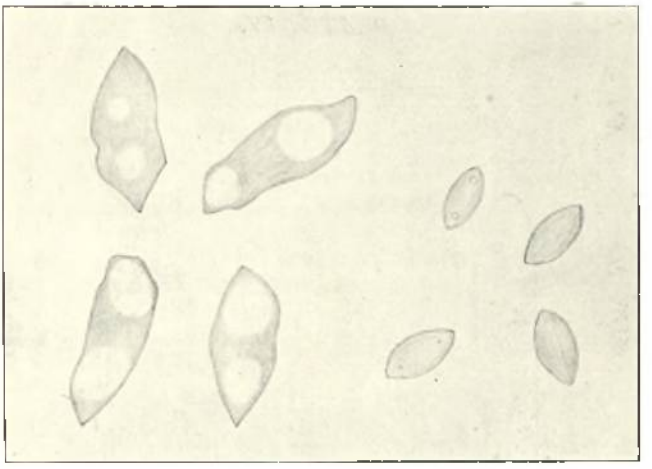


FIG. 4. A pycnidium showing the ostiole at apex; the ruptured bark is seen only on the left side. x about 110.



a *b*

FIG. 5. Spores of (*a*) *Phoma abietina* Hartig on *Abies pectinata*, (*b*) *Phomopsis Pseudotsugæ* Wilson on *Pseudotsuga Douglasii*; both drawn at the same magnification. x 950.

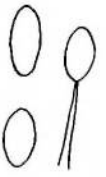
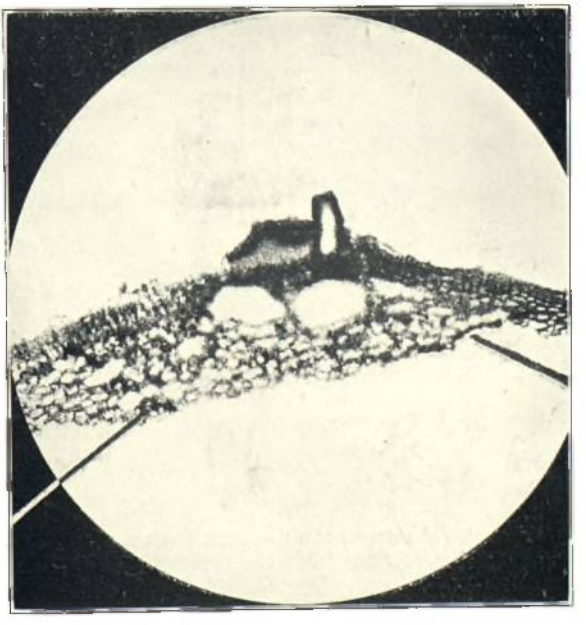


FIG. 6. Spores and sporophore from a pycnidium. x 1,000.



b

a

FIG. 7. Transverse section of a stem bearing a group of fructifications of *Diaporthe pitya*. The cavities of two perithecia are shown and a part of the neck of the one on the right. At (*a*) part of the black line limiting the cells densely filled with mycelium. x about 30.

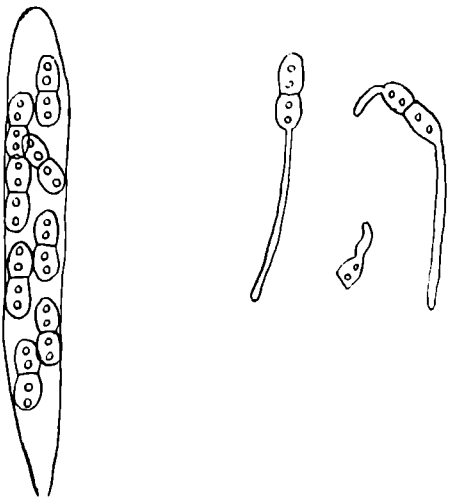


FIG. 8. Ascus and germinating ascospores of *Diaporthe pitya*. x about 660.

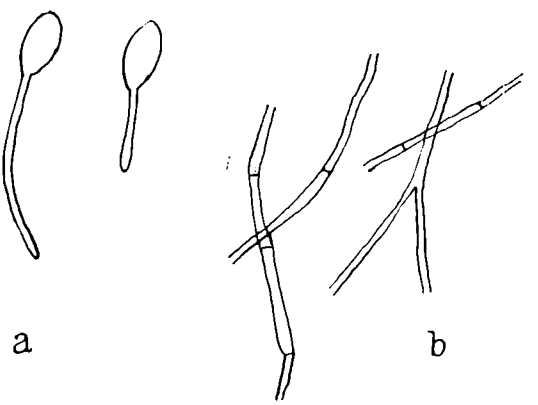


FIG. 9. (*a*) Germinating spores from a pycnidium. (*b*) mycelium from the cortex. x about 950.

PLATE V.

FIG. 10. Four-year-old tree with leading shoot killed. The dead portion of the shoot extends to (*a*). About $\frac{1}{2}$ nat. size.

PLATE VI.

FIG. 11. Apical portion of dead leading shoot of a four-year-old tree; the pycnidia can be seen on the stem; the leaves bear the pycnidia of *Cytospora* sp. About ¾ nat. size.

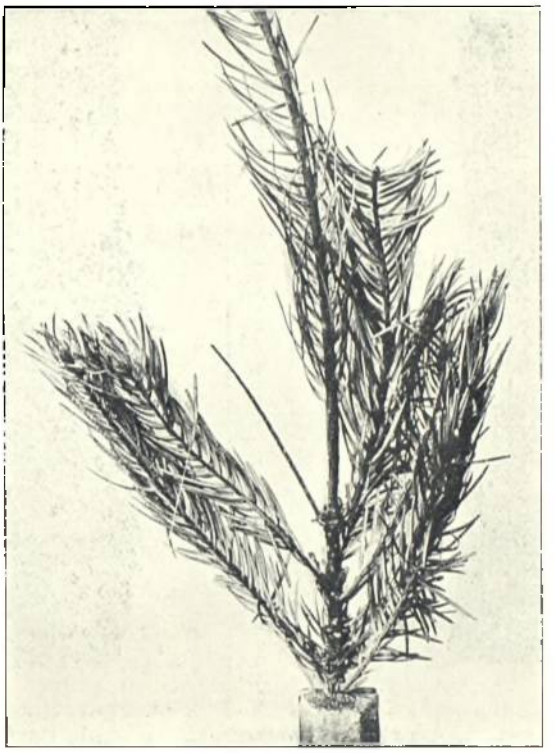


FIG. 12. Upper part of a leading shoot showing a lateral branch (on the left) killed by the fungus; the dead branch bears pycnidial fructifications. The mycelium has spread into the main stem and caused discoloration above and below the point of attachment of the lateral.

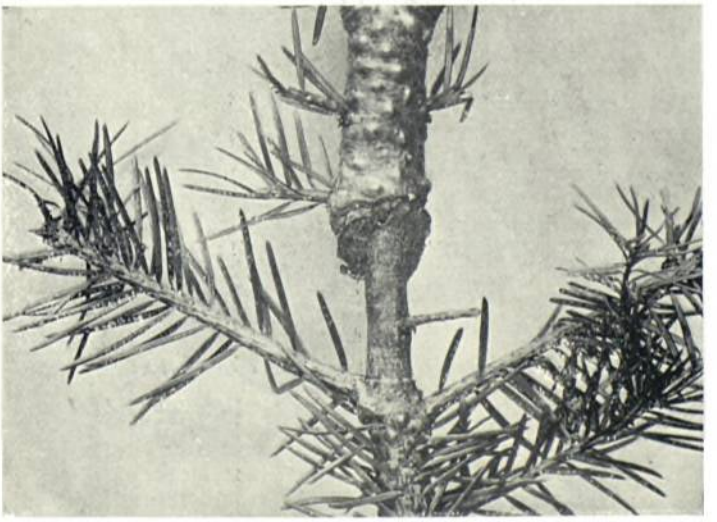


FIG. 13. Four-year-old tree attacked near ground level. The infected lateral branch is seen on the right.

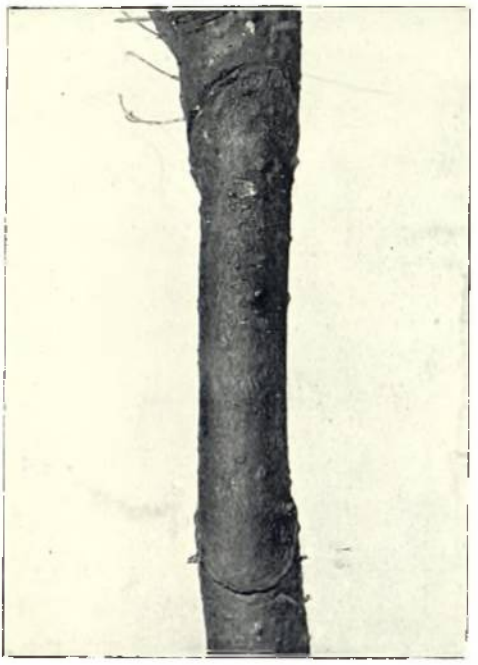


FIG. 14. Part of main stem near the base of a tree 7 years old. The infection has spread all round the stem.

FIG. 15. Part of main stem near the base of a tree 8 years old. The base of the infected lateral is seen in the middle of the canker.

FIG. 16. Transverse section through a canker on a stem 6 years old. The infected lateral is seen above. *a*, *a'*, points on each side up to which cambium was killed. *b*, *b'*, limits of callus growth during fifth year. *c*, *c'*, limits of callus growth during sixth year.

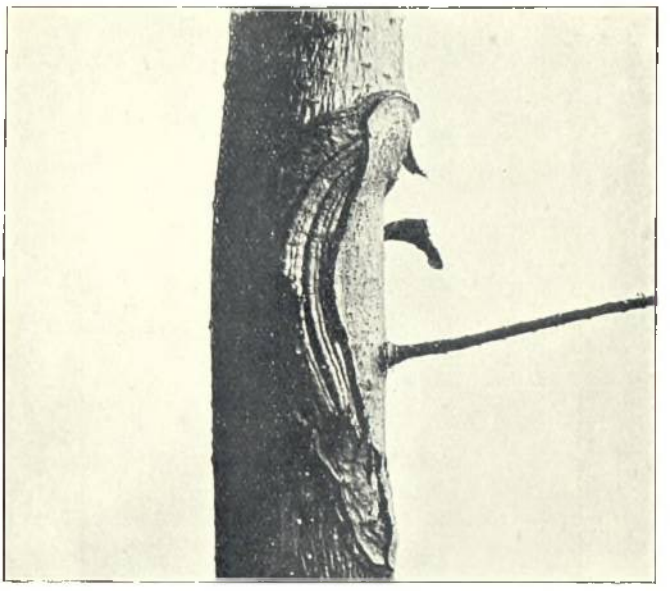


FIG. 17. Part of main stem of a tree 16 years old showing a canker in side view. The infected lateral branch is on the right hand; the callus tissue is well seen. The dead bark bearing the small black pycnidial fructifications is about to be cast off.

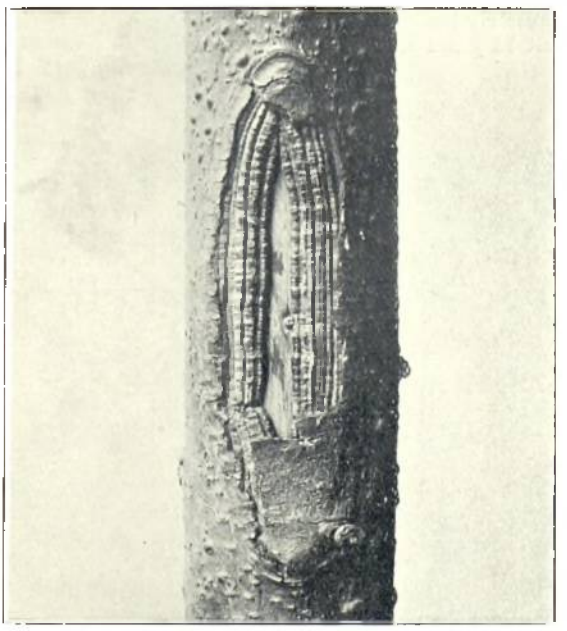


FIG. 18. Part of main stem of a tree 16 years old showing a canker in surface view. Nearly all the dead bark has been cast off exposing the surface of the wood and the callus tissue.

FIG. 19. Portion of dead bark cast off from healing canker, perforated in the centre by a hole through which the infected lateral branch passed.

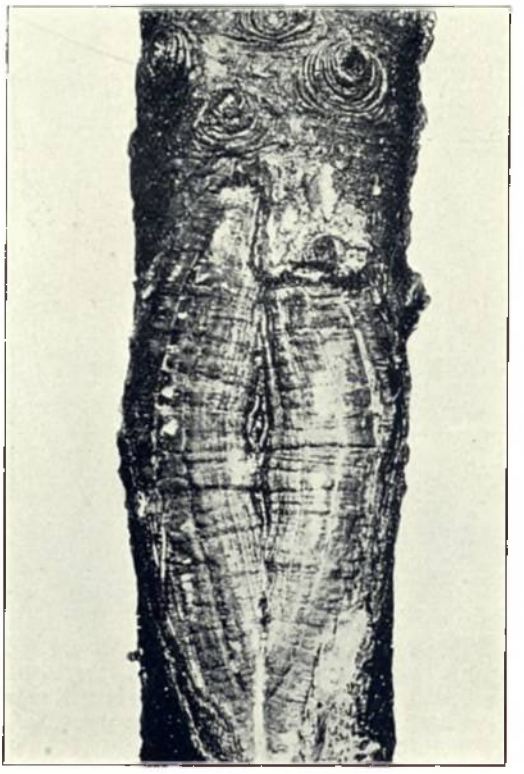


FIG. 20. A large completely healed canker from a tree about 18 years old.

a *a'*

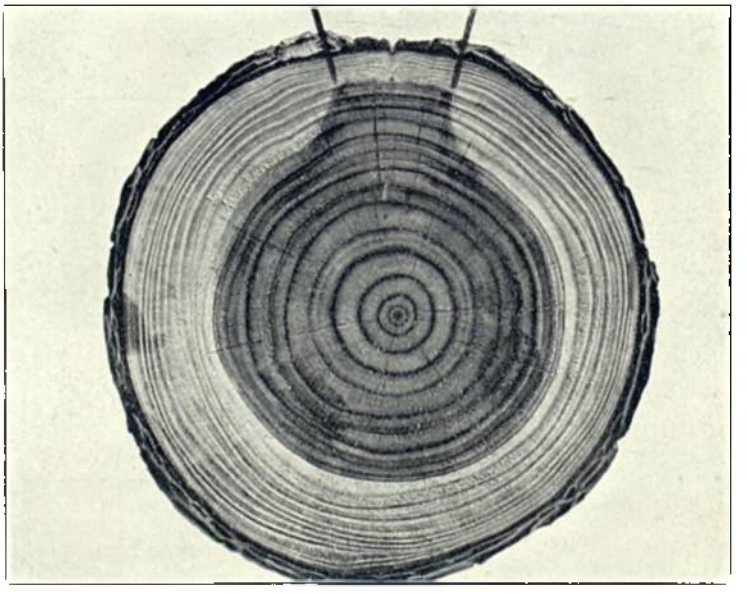


FIG. 21. A stem 23 years old cut across transversely to show the effect of a canker on the wood at *a*, *a'*.

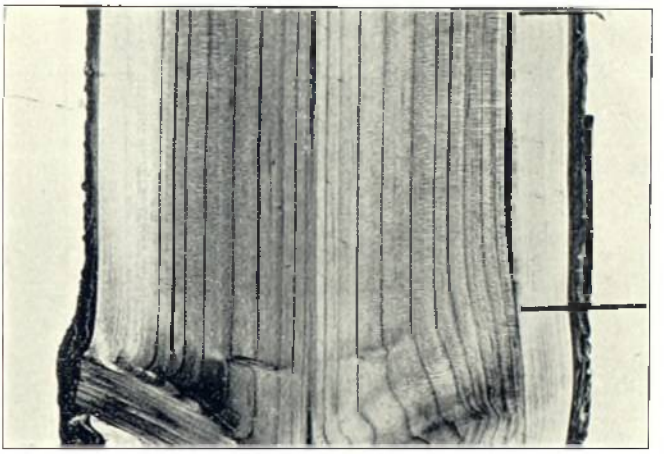


FIG. 22. A portion of the same stem cut longitudinally to show the effect of a canker on the wood. The dark line is seen on the right at *a*, *a'*.

FIG. 23. A young canker on the stem of Japanese larch.

FIG. 24. An older canker on the stem of Japanese larch; the dead bark is still present.

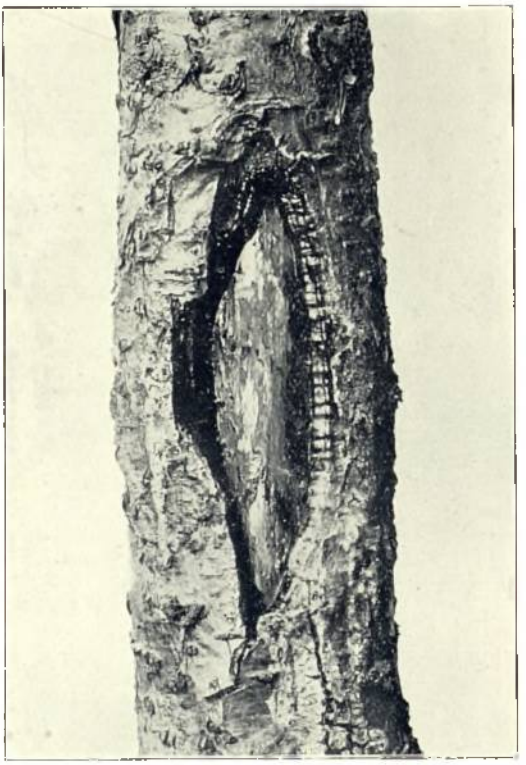


FIG. 25. A canker on stem of Japanese larch; the bark has been cast off and callus tissue is forming.

FIG. 26. A canker encircling the stem of a five-year-old plant of European larch.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