Shoot Blight of Larches.

A destructive disease in larch plantations of Japan.

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The first authentic report of the shoot blight affecting Japanese larch (*Larix leptolepis* GORDON) was made by SAWADA (1950)[25], who described the causal organism as *Physalospora laricina* sp. nov. by the materials collected in the Tohoku district, the northeastern part of the Japanese mainland. According to KAMEI (1961)[8], however, the same disease was known to have occurred in nurseries as early as 1938 in Hokkaido, the northern island.

UOZUMI (1960[31], 1961[32]) reported that a *Macrophoma* found on the diseased parts in the growing seasons of larch was the pycnidial stage of the causal fungus, *Physalospora laricina* SAWADA.

Morphologic characteristics of the genus *Physalospora* are very similar to those of the genera *Guignardia* and *Glomerella*, so they can not be distinctly distinguished from one another, but at their imperfect stages they are distinctly different.

For the reason that the genus *Guignardia* at the imperfect stage should belong to the genus *Macrophoma* or the allied genera, the fungus was transferred to the genus *Guignardia* and was, therefore, designated as *Guignardia laricina* (SAWADA) YAMAMOTO et K. ITO, comb. nov. (YAMAMOTO 1961[33]).

Although the disease has been regarded by forest pathologists as becoming significant in larch plantations, it is only within the past few years that it has been recognized as of sufficient importance to arouse a demand by tree owners for information regarding the disease and its control.

Since 1959 in Hokkaido and 1961 in the Tohoku district, the occurrence of numerous heavily affected trees in extensive stands has often presented an alarming appearance. This disease has thus became a matter of concern to the forest managers, and it is now generally considered to be a serious obstacle to successful growth of larch stands.

The disease is widely distributed throughout Hokkaido and the Tohoku district. The total area of diseased plantations has recently been calculated to be about 70,000 ha.

It is not too much to say that the disease is the most destructive affliction of trees which Japanese forestry has experienced in its history of cultivation. On account of its importance and severity, the various aspects of the disease have been treated by the forest pathology research staff of the Government Forest Experiment Station and the Faculty of Agriculture of Hokkaido University, and numerous contributions have increased our knowledge of the disease. By the recent studies, it has been made clear that European and American larches are more susceptible than Japanese larch to the disease (YANAGISAWA & SAITO 1960[34], SATO et *al.* 1962[18][23], TAKAHASHI 1962[26]), and accordingly European and American pathologists will no doubt direct increasing attention to this destructive blight.

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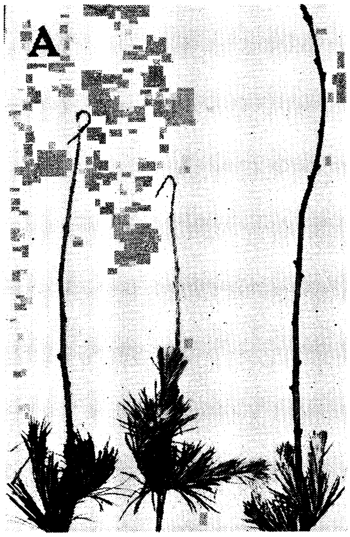
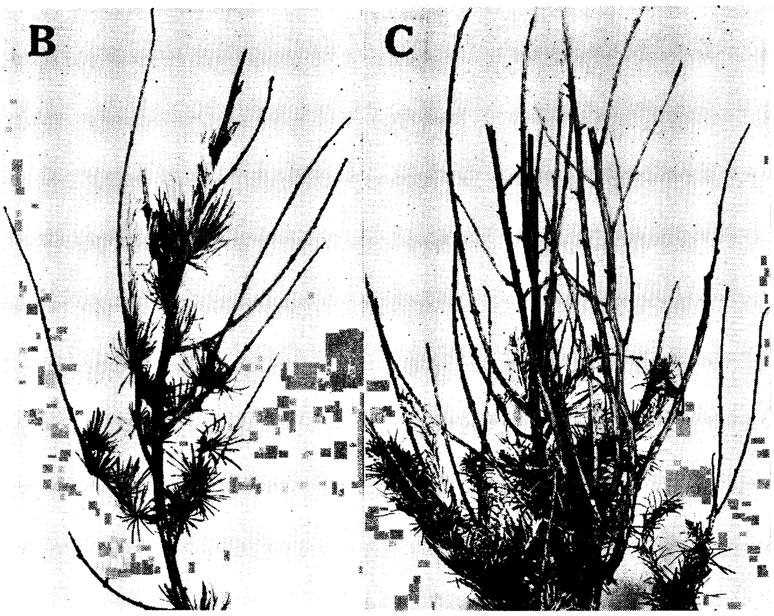
The present paper is essentially a progressive report summarizing the results of many workers' studies to date. The author wishes to express his appreciation to Messrs. Shun-ichi YOKOTA, Kunihiko SATÔ, Tadashi UOZUMI, Michio NAKAGAWA and Mrs. Hiroko HAYASHI for assistance in the preparation of the illustrations.

Symptoms

Infection is generally confined to the current season's shoots and leaves of adult trees as well as nursery stocks.

The symptoms first show themselves in late June to early July as hanging at the top of the shoot with the green to pale yellowing green leaves as a result of girdling. Then, the leaves in the infected parts defoliate, but those on the top which turned to brown remain for a long time. The diseased hanging shoots also turn brown and are frequently accompanied with exudation of resin, which, losing its more volatile constitution on exposure to the air, hardens into whitish drops. Infected shoots soon die and dry. As the disease progresses the infection occurs not only at the top of the shoot and the middle part of the shoot, but also the succulent secondary shoot, from where further infection takes place to the main shoot. From September to early October, the infected shoots do not





Text-fig. 1. Development of symptomatic pictures in the shoot blight of larch, showing in late spring.

A, 1 year after the first infection,
B, 2 years after the first infection,
C, 4 years after the first infection.

hang but remain straight, probably because of the hardening of the tissues.

Lesions on the diseased shoot are somewhat depressed, dried and shrunken. Diseased succulent shoots are actually thinner in the infected region than above or below. Minute pustules (perithecia or pycnidia of the causal fungus) are abundantly produced on the lesions.

The most conspicuous symptom of the disease is the discoloration, wilting, and death of the tender, succulent shoots of the current season's growth.

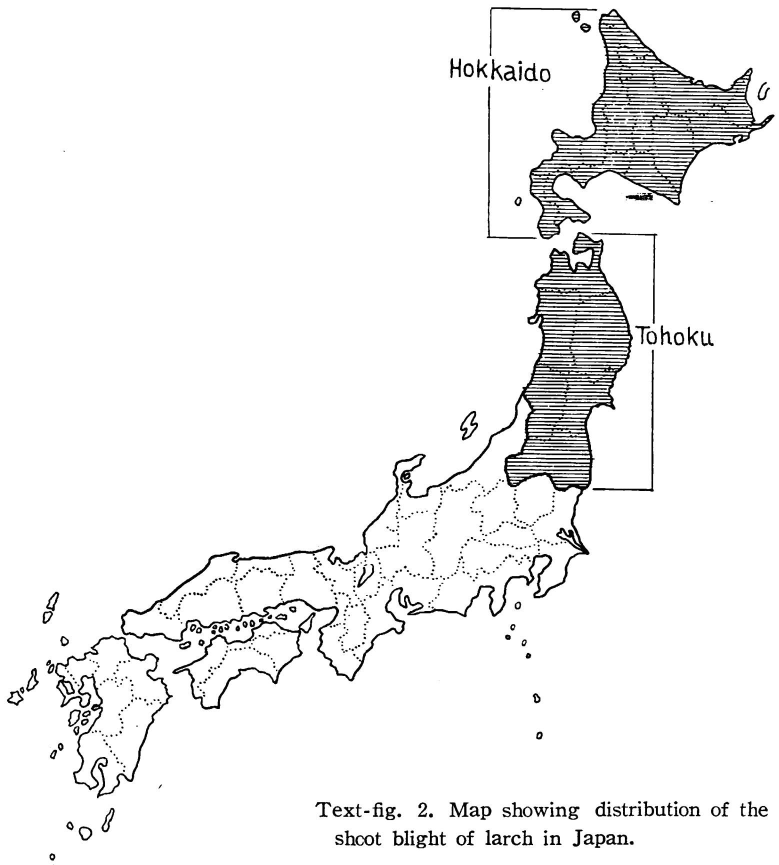
Larches that have their young shoots repeatedly killed by the disease become stunted and bushy, accompanied by many dead shoots. Repeated killing of shoots brings about a considerable decrease in growth increment, and generally results in worthless plantations (Text-fig. 1, Plates 1～7).

Distribution and damage

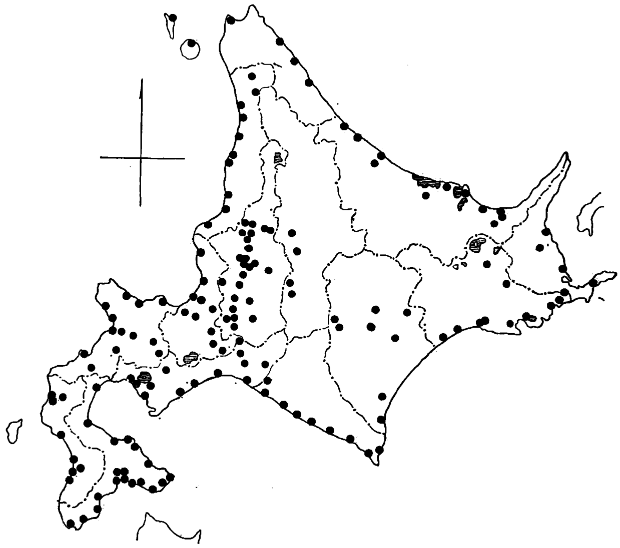
As noted already, the disease has long been locally known, but the large-scale infection of larch plantations has been found in many places only since about 1959.

The distribution of the disease is now restricted to Hokkaido and the Tohoku district, the northern parts of Japan. The accompanying maps (Text-figs. 2～4), which have been drawn up on data collected from all the sources available, show the present known distribution of the disease.

According to the results of the extensive survey carried on until 1962, the areas of diseased larch plantations are about 50,000 ha. in Hokkaido and about 20,000 ha. in the Tohoku



Text-fig. 2. Map showing distribution of the shoot blight of larch in Japan.



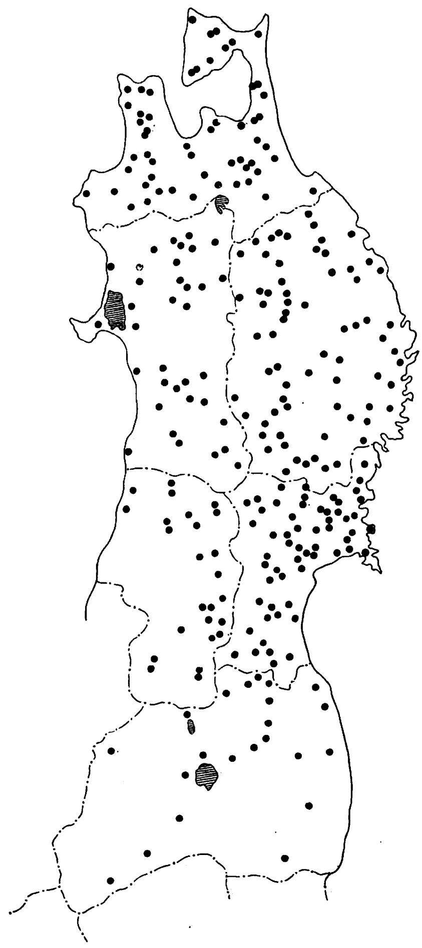
Text-fig. 3. Known distribution of the shoot blight of larch in Hokkaido in 1962.

district, and totally about 70,000 ha. In damaged plantations, almost 100 per cent of the trees are generally affected. Diseased trees which were severely infected in their younger stage have in most cases stopped growing.

Damage of larch seedlings in nurseries is also very common (Plate 4-C, Plate 5). In 1957, about 400,000 seedlings were infected in a nursery located along the coast of Uchiura Bay, Hokkaido, and in 1960, about 450,000 seedlings were badly diseased in a nursery in Iwate Prefecture, the Tohoku district. It is well known that such a conspicuous damage occurs commonly in many other nurseries situated in the disease affected region. In almost all cases the source of infection to seedlings is the diseased larch hedge cultivated as a windbreak in the nurseries. It is very important that the diseased seedlings be tranferred to new plantations and make new infection loci (ITO 1961[4)5)], YOKOTA 1961[39)]).

Damage of the disease is very severe near the seashore. In Hokkaido, the diseased plantations are generally concentrated along the coast of the Japan Sea, the Pacific coast and the coast of the Sea of Okhotsk, amounting to 60 per cent of the total damaged area in Hokkaido (YOKOTA 1962[40)]). In the Tohoku district, the most severely infected areas are also distributed along the Pacific coast and the coastal region of Aomori Bay (SATO 1961[17)], SATO et *al.* 1962[21)]). The acute disease development in these regions is believed to be largely due to the wind blowing in the growing seasons of larch trees.

Recently, considerable damage by the disease in larch plantations has also been found in the



Text-fig. 4. Known distribution of the shoot blight of larch in the Tohoku district in 1962.

inland areas. The damage in the lowlands along the Ishikari River, between Sapporo and Tomakomai, Hokkaido, and that in the Tohoku district distributed in the regions along the railway lines acrossing the Ou Range have become conspicuous. All of these regions are commonly exposed to the wind along the rivers or the railway lines. Recent experimental works have also shown that damage in the stand protected from the wind by the windbreak or the shelterbelt is conspicuously much slighter than in the untreated stand, and that the wind in the growing seasons is considered to be one of the most important environmental factors leading to a heavy outbreak of the disease (YOKOTA & INOUE 1961[36], KATO & ONO 1962[9], OKAMOTO & NAKAGAWA 1962[13], SATO et *al.* 1962[21]).

Damage is generally severe in the larch plantations that are enveloped in a dense fog in the growing seasons.

Morphology and life history of the causal fungus

The fungus was described by SAWADA (1950)[25] under the name of *Physalospora laricina* sp. nov. The imperfect stage, *Macrophoma,* and the spermogonial stage of this organism were first found by UOZUMI (1961)[32]. By YAMAMOTO (1961)[33] the fungus was transferred to the genus *Guignardia* and named as *Guignardia laricina* (SAWADA) YAMAMOTO et K. ITO from the opinion that *Physalospora* or the allied genera owning *Macrophoma* in the imperfect stage were to be treated as the genus *Guignardia.*

Guignardia cryptomeriae SAWADA* (1950)[24], a very closely allied species to the fungus, is frequently found on blighted or die-backed shoots of larch trees, and this often makes diagnosis difficult. A die-back caused by *Diaporthe conorum* (DESM.) NIESSL (syn. *Phomopsis occulta* TRAVERSO) is widely distributed throughout larch plantations of Japan, but this is readily distinguishable from the shoot blight fungus by morphologic characteristics of the spores (KOBAYASHI 1960[10], 1962[11], ITO 1963[7]).

Perithecial stage (*Guignardia*) : Mature perithecia single or in groups, hypodermic, black, globose to subglobose, ostiolate, 300～410 μ in height, 265～440 μ in diameter. Asci clavate, hyaline, round at the apex, stipitate at the base, 102～140×22～45 μ. Paraphyses abundant, hyaline, straight or branched. Ascospores oblong, hyaline, round at the apex, 24～41×8～17 μ (Text-fig. 5).

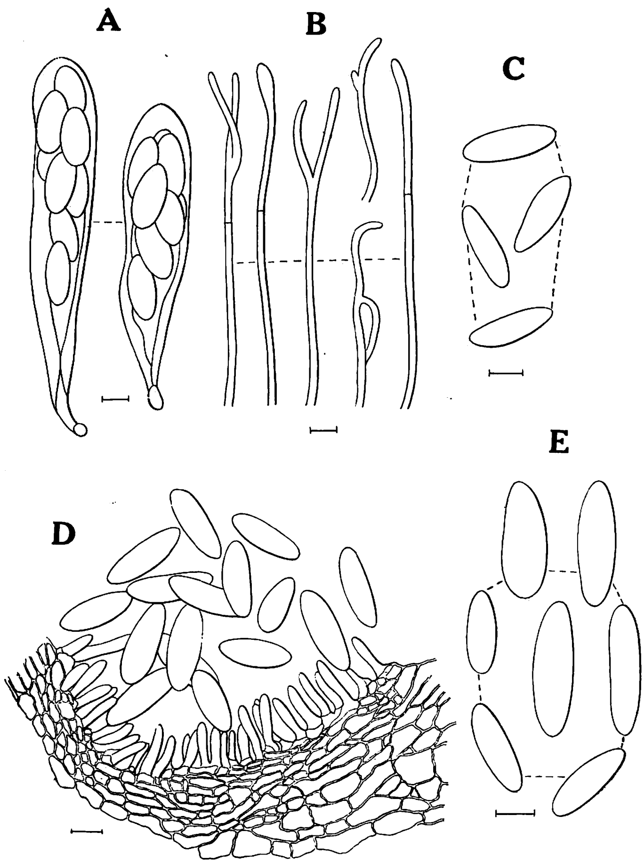
Dimensions of the fungus in the perithecial stage measured by various workers are shown

Table 1. Dimension of *Guignardia laricina* in the ascigerous stage (μ).

Perithecium	Ascus	Paraphysis	Ascospore	Literature
368 (in diam.)	114～135×22～26	3 (in diam.)	24～27×13	SAWADA '50
330×440	131～142×30～45	2.8～3.6(〃)	25～34×8～17	UOZUMI '61
170～500×130～300	60～120×20～50	3 (〃)	23～38×8～15	YOKOTA '62
300～415×265～390	102～118×25～31	—	31～41×13～17	KOBAYASHI '62

* *Guignardia cryptomeriae* SAWADA : Perithecia hypodermic, black, globose to subglobose, ostiolate, 160～310 μ in height, 300～340 μ in diameter. Asci clavate, hyaline, round at the apex, 60～85×13～22 μ, paraphysate. Ascospores fusiform to oblong, hyaline, 18～31×7～10 μ, arranged biseriately. Pycnidia (*Macrophoma*) hypodermic, black, globose, 120～440×138～390 μ. Pycnospores fusiform to oblong, hyaline, 15～31×5～10 μ.

Hab. on leaves and shoots of *Cryptomeria japonica* and *Larix leptolepis.*



Text-fig. 5. *Guignardia laricina* (SAWADA) YAMAMOTO et K. ITO. (⊢=10μ)
A, Asci; B, Paraphyses; C, Ascospores; D, A part of pycnidium (*Macrophoma* stage); E, Pycnospores.

in Table 1.

Pycnidial stage (*Macrophoma*) : Pycnidia hypodermic, black, globose to subglobose, 123～325×176～265 μ. Pycnospores oblong, straight or somewhat curved, hyaline, 22～37×6～10 μ (Text-fig. 5).

Dimensions of the fungus in the pycnidial stage measured by various workers are presented in Table 2.

Table 2. Dimension of *Guignardia laricina* in the pycnidial stage (μ).

Pycnidium	Conidiophore	Pycnospore	Literature
123～210×176～245	3～7 (in length)	23～30×6～9	UOZUMI '61
100～210×90～200	8 (〃)	15～33×5～11	YOKOTA '62
250～325×200～265	—	25～37×7～10	KOBAYASHI '62

The pycnidial stage, *Macrophoma*, is generally found from late June to early November and it is responsible for the secondary infection appearing late in the summer. Pycnospores rarely overwinter on the diseased shoots and leaves, and survive till April of the following year. The spermogonia are found in July to October, rarely in February of the following year. The mature spermogonia are filled with a great number of rod-shaped spermatia, 3～6×1～2 μ in size. Repeated attempts to germinate the spermatia in various media have been unsuccessful (UOZUMI 1961[32], YOKOTA 1962[40]).

The perithecial stage, *Guignardia*, begins its formation on the diseased current season's shoots generally in October, rarely in August, but does not become sufficiently differentiated to be recognized as perithecial primordia until the next spring. The mature perithecia are formed in May to September and they discharge abundant ascospores throughout the seasons. The ascospores act as the source of the chief primary incculation. Though almost all of the perithecia become empty by early November, a few of them overwinter in the state of containing viable ascospores, and accordingly some of the ascospores are collected even in early spring (UOZUMI 1961[32], SATO & SHOJI 1962[20], YOKOTA 1962[40]).

Physiology of the causal fungus

Germination of spores

Ascospores of the fungus germinate readily in water with over 50 per cent germination in 4 hours at 15° to 30°C. The germination occurs at the temperatures ranging from 10° to 35°C with an optimum at 25°C (SATO & SHOJI 1962[22], YOKOTA 1963[44]).

The germination of pycnospores is generally favorable at 20°～30°C, and very sparse at 15° and 35°C with an optimum at 28°C to 30°C. It is noticeable that the optimal temperature for germination of pycnospores is somewhat higher than that of ascospores (SATO & SHOJI 1962[22], YOKOTA 1963[44]).

A saturated atmosphere is very favorable to germination of ascospores and pycnospores, and they germinate in 100 to 94 per cent humidities, while those kept at 92 per cent humidity and below 92 per cent show no signs of germination (SATO & SHOJI 1962[22], YOKOTA 1963[44]).

Influences of H-ion concentrations upon the germination of the spores are not remarkable with exponents ranging from pH 3 to 10 (SATO & SHOJI 1962[22]).

Longevity of spores

Ascospores in the perithecia and pycnospores in the pycnidia on diseased parts in nature maintain the germinability during at least 9 months, whereas discharged ascospores as well as pycnospores are very short in their longevity and they lose the germinability within 3 or 4 days (YOKOTA 1963[44]).

Mycelial growth

Isolation of the fungus is readily obtained from mono-pycnospore, mono-ascospore and diseased tissues. Mycelial growth on such agar media as potato-dextrose agar, SAITO's soy agar and CZAPEK's solution agar is usually well. Macroscopic and microscopic characteristics of Isolate KPS43-7* on potato-dextrose plate agar at the end of 20 days' culture at about 25°C are summarized as follows (YOKOTA 1962[40]). Macroscopic appearances of the mycelial colony : Aerial mycelium abundant, grayish white, making a concentric circle; mycelium of inner part of the circle tiny floccose, that of outer part pale gray, somewhat reticulate; small mass of hyphae appearing in various parts of the surface of the colony; submerging hyphae dark green.

Microscopic appearances of the hyphae : Marginal hyphae of the colony septate, branched, thin in membrane, hyaline, rarely pale dark; sometimes hyphae swollen and shrinking at the

* The isolate from mono-ascospore on *Larix leptolepis* collected in Hokkaido.

septum or branching, having no special structure; hyaline hyphae 1.5～4 μ in diameter; pale dark hyphae 2～4 μ in diameter. Hyphae in the central part of the colony septate, branched, thin in membrane, hyaline or dark brown; hyaline hyphae often partially swollen and shrinking at the septum or branching, having no special structure; hyaline hyphae 2～5 μ in diameter; dark hyphae 2.5～6 μ in diameter. Submerging hyphae septate, thin in membrane, branching, hyaline or dark brown; hyaline hyphae often swollen and shrinking at the septum or branching; some hyphae continued to hyaline hyphae and separated by septum; hyaline hyphae 1.5～4 μ in diameter; dark hyphae 2～4 μ in diameter.

Among 12 isolates tested, the one, SG91T*1, produced many spermatia, 2.5×1 μ, and pycnospores, 18～25×7～9 μ, on the three agar media (YOKOTA 1962[40]).

The relation of temperatures to the growth of the mycelium of the fungus was studied by Petri dish method. The result shows that the fungus grows favorably at the temperatures ranging 10°C to 30°C with an optimum at 25°C, and does not grow at 0°C and 35°C, respectively (UOZUMI 1961[32]).

Sporulation on agar media

A constant supply of a great number of spores is needed for screening tests of fungicides and artificial inoculations, but the fungus has been generally found to produce few spores in pure culture (UOZUMI 1961[32], YOKOTA 1962[40]). Here, by using many isolates of the fungus, efforts have been made to find artificial media favorable for sporulation.

Recently, only the two*2*3 among 15 isolates tested have produced a great abundance of pycnospores, $\times 10^6$ in number per tube, 18 mm in diameter, after two weeks' culture at 25°C to 27°C on agar-medium of the following formula (HARA & ITO 1963[2]):

Dry yeast extract (Daigo Eiyo Chem. Co., Ltd.)	3 g
Soluble starch	10 g
$MgSO_4 \cdot 7H_2O$	0.25 g
Agar-agar	15 g
Distilled water	1 *l*
	pH 7.0

Pathogenicity of the causal fungus

According to the results obtained by field observations and artificial inoculations (YOKOZAWA & MURAI 1958[46], YOKOZAWA 1959[47], YANAGISAWA & SAITO 1960[34], SATO & SHOJI 1962[23], TAKAHASHI 1963[26]) the relative susceptibility of four species of *Larix* to the disease is as follows:

Resistant………*L. gmelinii* var. *japonica*, *L. gmelinii* var. *koreana*, *L. gmelinii* var. *japonica*×*L. leptolepis*,

Susceptible………*L. leptolepis*, *L. leptolepis*×*L. decidua* (*L. eurolepis*),

Very susceptible………*L. decidua*, *L. occidentalis*.

Generally speaking, Kurile larch (*L. gmelinii* var. *japonica*) is relatively resistant, whereas European larch (*L. decidua*) is susceptible and Japanese larch (*L. leptolepis*) is intermediate, although different races of European larch vary widely in susceptibility.

Douglas fir (*Pseudotsuga douglasii*) is the only species except the member of the genus

*1 The isolate from the diseased tissue of *Larix gmelinii* var. *japonica* collected in Hokkaido.

*2 The isolate from *Larix leptolepis* in Noheji, Aomori, by K. SATO.

*3 The isolate from *L. decidua* in Morioka, Iwate, by K. SATO.

Larix which has been known as the host of the fungus in nature (SATO & SHOJI 1962[23]).

Pathogenicity of the fungus to various conifers was tested by artificial inoculations with the mycelium from culture on potato-dextrose agar. In this study, plants of 28 species or varieties, representing 14 genera were inoculated with the fungus. Results of the experiments are given in Table 3 (SATO & SHOJI 1962[23]).

Table 3. Results of the artificial inoculation with *Guignardia laricina* to various kinds of conifers.

Tree species		Pathogenicity	Pycnidium formation on lesion
Scientific name	Japanese name		
Cephalotaxus harringtonia var. *drupacea*	Inu-gaya	+	−
Abies firma	Momi	+	−
A. homolepis	Urajiro-momi	+	+
A. mariesii	Aomori-todomatsu	+	−
A. mayriana	Ao-todomatsu	+	−
A. veitchii	Shirabe	+	−
Cedrus deodara	Himaraya-sugi	−	−
Picea glehni	Aka-ezomatsu	+	+
P. abies	Doitsu-tohi	+	−
Pseudotsuga douglasii	Dagurasu-fa	+	+
Tsuga diversifolia	Kome-tsuga	+	−
Pinus densiflora	Aka-matsu	+	+
P. koraiensis	Chosen-matsu	+	−
P. pentaphylla	Himeko-matsu	+	−
P. thunbergii	Kuro-matsu	+	−
P. rigida	Rigida-matsu	+	−
P. banksiana	Bankusu-matsu	+	+
P. strobus	Storobu-matsu	+	−
Taxodium distichum	Rakuu-sho	+	−
Cunninghamia lanceolata	Koyozan	−	−
Metasequoia glyptostroboides	Metasekoia	−	−
Chamaecyparis obtusa	Hinoki	+	−
C. pisifera	Sawara	+	−
Thujopsis dolabrata var. *hondai*	Hinoki-asunaro	+	−
Thuja standishii	Nezuko	+	−
T. occidentalis	Nioi-hiba	+	−
T. orientalis	Konote-gashiwa	−	−
Juniperus conferta	Hai-byakushin	−	−

As shown in Table 3, pathogenicity of the fungus is positive to almost all of the tree species tested, except *Cedrus deodara*, *Cunninghamia lanceolata*, *Metasequoia glyptostroboides*, *Thuja orientalis* and *Juniperus conferta*. Pycnidia of the fungus are produced on the infected parts of the following five species: *Abies homolepis*, *Picea glehni*, *Pseudotsuga douglasii*, *Pinus densiflora* and *P. banksiana*.

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Explanation of plates

Plate 1.

A, Young stand of Japanese larch affected destructively by the shoot blight, Tarumae, Hokkaido.

B, Japanese larch affected severely by the shoot blight, Hokkaido.

Plate 2.

A, A part of Japanese larch stand (about 10-year-old) affected moderately by the shoot blight, Tomakomai, Hokkaido. Photo. October, 1961.

B, Japanese larch (about 10-year-old) affected moderately by the shoot blight Tomakomai, Hokkaido. Photo. October, 1961.

C, Ditto. Photo. September, 1962.

Plate 3.

A-B, Japanese larch (about 10-year-old) affected severely by the shoot blight, Tomakomai, Hokkaido. Photo. September, 1962.

C, Top of Japanese larch affected severely by the shoot blight, Tomakomai, Hokkaido. Photo. September, 1962.

D, Japanese larch (about 10-year-old) affected severely by the shoot blight, Ryugamori, Iwate. Photo. September, 1961.

Plate 4.

A, Japanese larch (about 10-year-old) affected severely by the shoot blight, Ryugamori, Iwate. Photo. September, 1961.

B, Top of young Japanese larch (6-year-old) affected severely by the shoot blight, Shiraoi, Hokkaido. Photo. October, 1961.

C, Damage of the shoot blight in a nursery, Tomakomai, Hokkaido. Photo. October, 1961.

Plate 5.

A, Initial stage of the shoot blight in a 2-year-old seedling in Matsuo Nursery, Iwate. ×1.4 d, Diseased secondary shoots.

B, Primary and secondary shoots of a 2-year-old seedling affected by the shoot blight in Matsuo Nursery, Iwate. ×1.5

C, Killed and defoliated shoots of a 2-year-old seedling affected severely by the shoot blight, accompanied with dried resin (r) on the surface, in Matsuo Nursery, Iwate. ×1

D, Apical part of a 2-year-old seedling affected severely by the shoot blight in Matsuo Nursery. ×0.7

Plate 6.

A, Top of the shoot of young larch affected severely by the shoot blight, Shiraoi, Hokkaido. ×0.7

B, Primary and secondary shoots of young larch affected severely by the shoot blight, Tomakomai, Hokkaido. ×1.2

C, Top of the primary shoot affected severely by the shoot blight, showing many pycnidia of the causal fungus. Magnified. Photo. by Mr. S. Yokota.

D, Dried resin (r) produced on the surface of the diseased shoot. ×0.8

E, Slender part of the diseased shoot, accompanied with dried resin (r). Magnified. Photo. by Mr. S. Yokota.

Plate 7.

A, Enlargement of the lesion of the diseased shoot, showing perithecia of the causal fungus produced in a slit (p) of the host. ×4

B, Enlargement of the lesion of the diseased shoot, showing perithecia of the causal fungus and dried resin (r). ×8

C, Enlargement of the lesion of the diseased shoot, showing pycnidia of the causal fungus. ×9

D, Photomicrograph of perithecia of the causal fungus. ×150

E, Photomicrograph of pycnidium of the causal fungus. ×230

カラマツの先枯病

伊 藤 一 雄[1]

摘 要

カラマツの先枯病（梢枯病，枝枯病）菌は東北地方で採集された資料をもとに沢田（1950）によってはじめて記載された。もっとも亀井（1961）によれば本病はすでに 1938 年ごろ北海道で見い出されていたということである。

本病は北海道では 1959 年以来，また東北地方では 1961 年以来一般林業家の注目をひく激害林分が各地で発見され，1962 年現在その被害面積は北海道約 50,000 ha，東北地方約 20,000 ha，計 70,000 ha をかぞえ，今後の調査によってこの面積はさらに増大するものと予想されている。

本病は造林木のみならず苗木も侵し一苗畑で 400,000 本以上の被害をうけた例も少なくない。また罹病苗木が造林地に植栽され，これを中心にして蔓延，激害林分をもたらしたと考えられる事例もまた普通にみとめられる。

本病の分布は現在北海道および東北6県に限られているが，他の地方への被害の拡大が憂慮されている。

本病に侵されたカラマツはただちに枯死することはないが，連年新梢が枯死するため樹形は傘型あるいは盆栽型を呈し，発育はいちじるしく阻害される。本病はその伝染力の猛烈なこと，被害の顕著なことおよび防除の困難なことなどからまれにみる悪質な疾病で，わが国におけるカラマツ造林の成否を左右するほどの大きな障害と考えられ，またわが国林木病害史上これに匹敵するものはなく，今やこれは欧米諸国におけるストローブマツの発疹さび病（blister rust），クリの胴枯病（Chestnut blight）およびニレの立枯病（Dutch elm disease）にまさるともおとらない流行病の様相を呈している。

本病はこのように重要なものであるから，農林省林業試験場本場，北海道支場，東北支場および北海道大学農学部が中心になって本病に関する広汎な試験研究が着手され，まだ数年しか経過していないにもかかわらず，数々の見るべき成果があげられている。本病はニホンカラマツ（*Larix leptolepis*）のみならず，オオシュウカラマツ（*L. decidua*），西部アメリカカラマツ（*L. occidentalis*）にも発生，特にオオシュウカラマツおよび西部アメリカカラマツは著しく感受性なこともあって，本病に対する海外の関心もはなはだ高い。それでこれまで得られた試験研究の概要を広く紹介する目的で本稿の筆をとった。

本病の病原菌は最初 *Physalospora laricina* sp. nov. と記載され（沢田 1950），さらに柄子殻時代 *Macrophoma* が見出されてこれと *Physalospora* の同根関係が立証された（魚住 1961）。のちにこれは転属されて *Guignardia laricina* (SAWADA) YAMAMOTO et K. ITO, comb. nov.（山本 1961）と改められた。本菌に近似のものとしてわが国に *Guignardia cryptomeriae* SAWADA（*Macrophoma sugi* HARA）があるが，これらはおのおの別種で，また海外においてこれに類似のものはなく，本菌はわが国の特産と考えられている。

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本病が今日のように広く蔓延をみるに至ったのは，まず苗畑周辺にあるカラマツの生垣あるいは防風林が本病に侵され，これから伝染源胞子が苗木に到達して罹病，初期病徴が気象災害等と一見似ているので悪質の疾病と考えずに罹病苗木が造林地に植栽され，近年広く実施されたカラマツの単純一斉大面積造林がわざわいして広大激甚な被害をもたらしたものとみられている。なお本病の被害と環境条件との間には密接な関連があり，特に風衝地に本病が激発する傾向は顕著で，従来風衝地ではカラマツが盆栽状を呈して育たないもの，と巷間伝えられたことがらを裏書きしており，カラマツの生育期における常風は本病の被害発生およびその程度を左右する大きな因子になっていることが明らかにされつつある（横田・井上 1961，加藤・小野 1962，岡本・中川 1962，佐藤ら 1962）。

防除薬剤のスクリーンニング・テストに，また人工接種試験に，常に多量の胞子を必要とするのであるが，本菌は人工培地上に胞子を生成しがたい菌に属しており，これらの試験研究遂行上多大の隘路になっていた。最近特定の菌株に限られるが，胞子を多量に生成する培地の探索に成功，能率的に実験を行ないうる見とおしが得られた（原・伊藤 1963）。

カラマツ属樹種間に本病に対する抵抗性あるいは感受性の差がある事実が野外観察および人工接種によってたしかめられた。すなわちグイマツ（*L. gmelinii* var. *japonica*），チョウセンカラマツ（*L. gmelinii* var. *koreana*）およびグイマツ×ニホンカラマツは抵抗性，ニホンカラマツおよびニホンカラマツ×オオシュウカラマツ（*L. eurolepis*）は感受性，なおオオシュウカラマツおよび西部アメリカカラマツはきわめて感受性である（横沢・村井 1958，横沢 1959，柳沢・斎藤 1960，佐藤・庄司 1962，高橋 1963）。

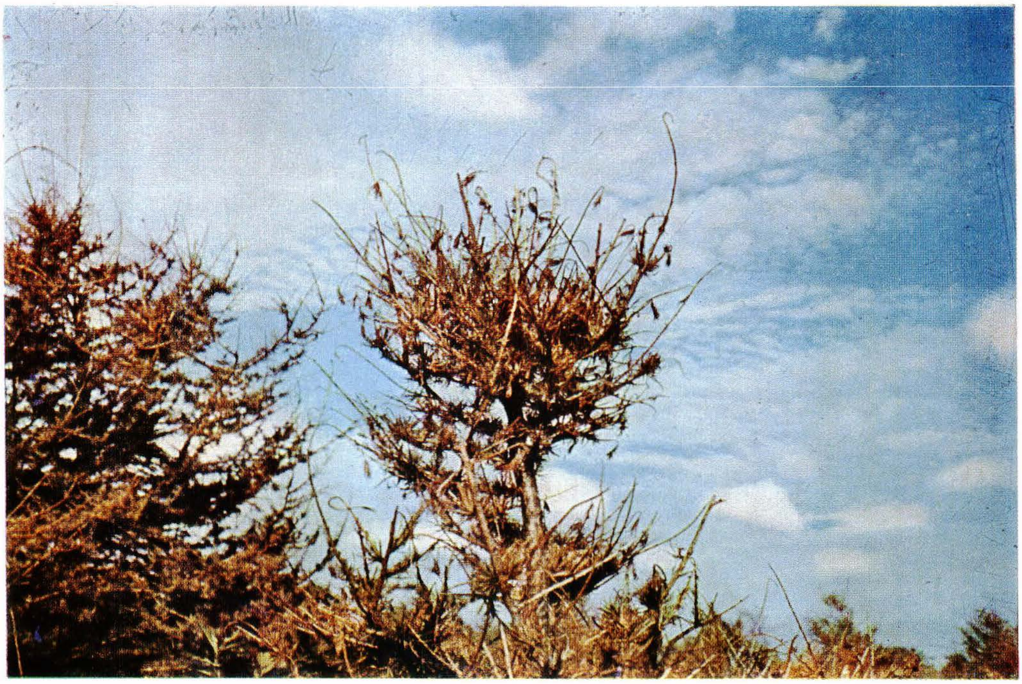
本病病原菌は自然状態においてはカラマツ属樹種以外ではダグラスファー（*Pseudotsuga douglasii*）にだけ発見採集されている。培養菌糸による人工接種試験結果によれば本菌はイヌガヤ（*Cephalotaxus harringtonia* var. *drupacea*），モミ（*Abies firma*），ウラジロモミ（*A. homolepis*），アオモリトドマツ（*A. mariesii*），アオトドマツ（*A. mayriana*），シラベ（*A. veitchii*），アカエゾマツ（*Picea glehni*），ドイツトウヒ（*P. abies*），ダグラスファー，コメツガ（*Tsuga diversifolia*），アカマツ（*Pinus densiflora*），チョウセンマツ（*P. koraiensis*），ヒメコマツ（*P. pentaphylla*），クロマツ（*P. thunbergii*），リギダマツ（*P. rigida*），バンクスマツ（*P. banksiana*），ラクウショウ（*Taxodium distichum*），ヒノキ（*Chamaecyparis obtusa*），サワラ（*C. pisifera*），ヒノキアスナロ（*Thujopsis dolabrata* var. *hondai*），ネズコ（*Thuja standishii*）およびニオイヒバ（*T. occidentalis*）に病原性をしめし，なおこれらのうち，ウラジロモミ，アカエゾマツ，ダグラスファ，アカマツおよびバンクスマツには柄子殻が形成された（佐藤・庄司 1962）。

本病の薬剤防除にはおよそ3つの分野がある。その1は苗畑における予防，その2は山出苗木の消毒，その3は造林木の予防および治療である。山出苗木の消毒はその休眠期においてEMP（エチル燐酸水銀）剤による浸漬被覆法（遠藤・横田 1963）によってほぼ実用化の見とおしが得られ，苗木の予防には抗生物質シクロヘキシイミドおよびTPTA（有機スズ剤）の散布により，また造林木の予防治療には同じくシクロヘキシイミドとTPTA剤の茎葉散布およびシクロヘキシイミド油剤の樹幹塗布など有望な試験成績（斎藤ら 1961 ）が報告されているが，これらについてはそれぞれ文献をあげるにとどめた。

—Plate 1—

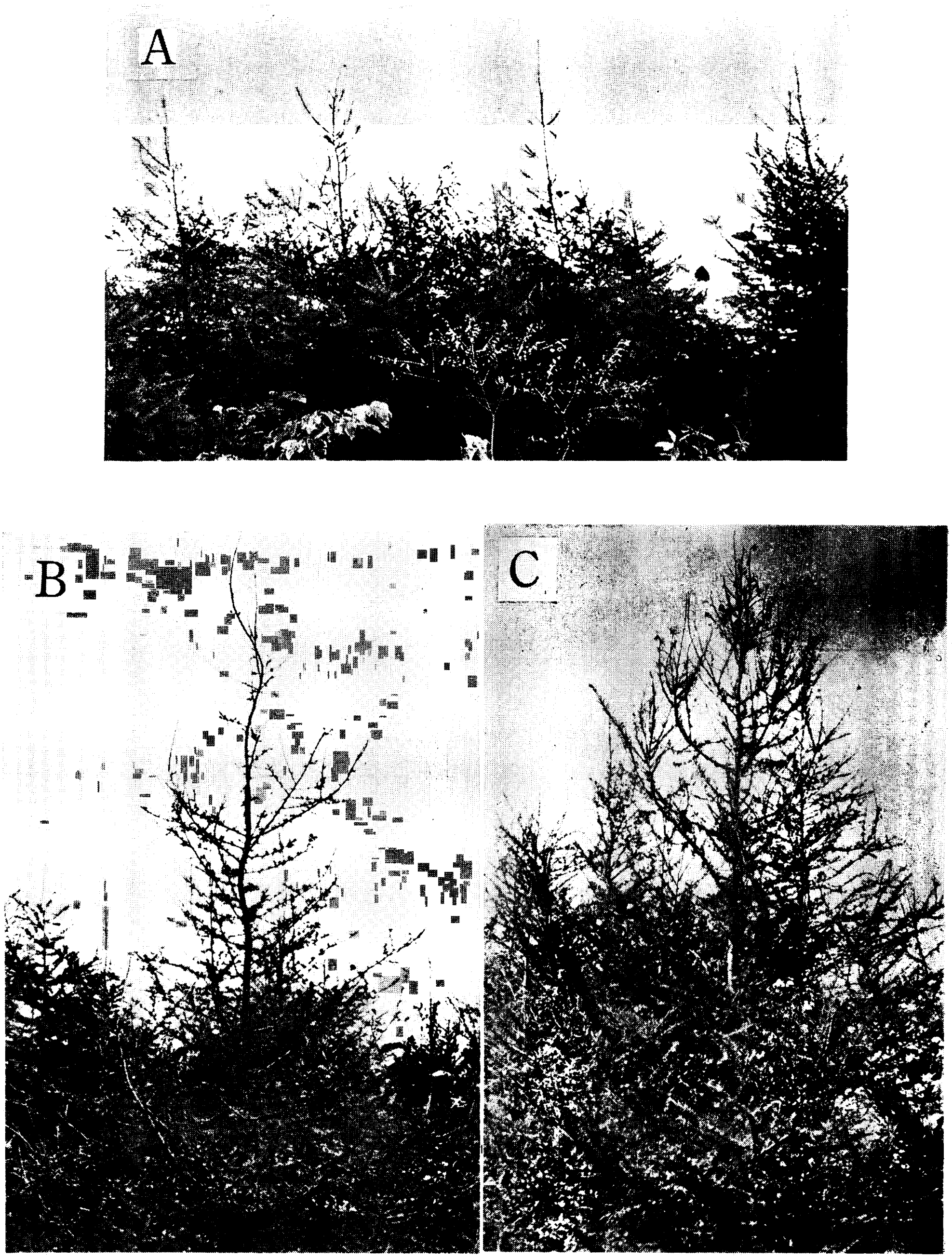
A

B

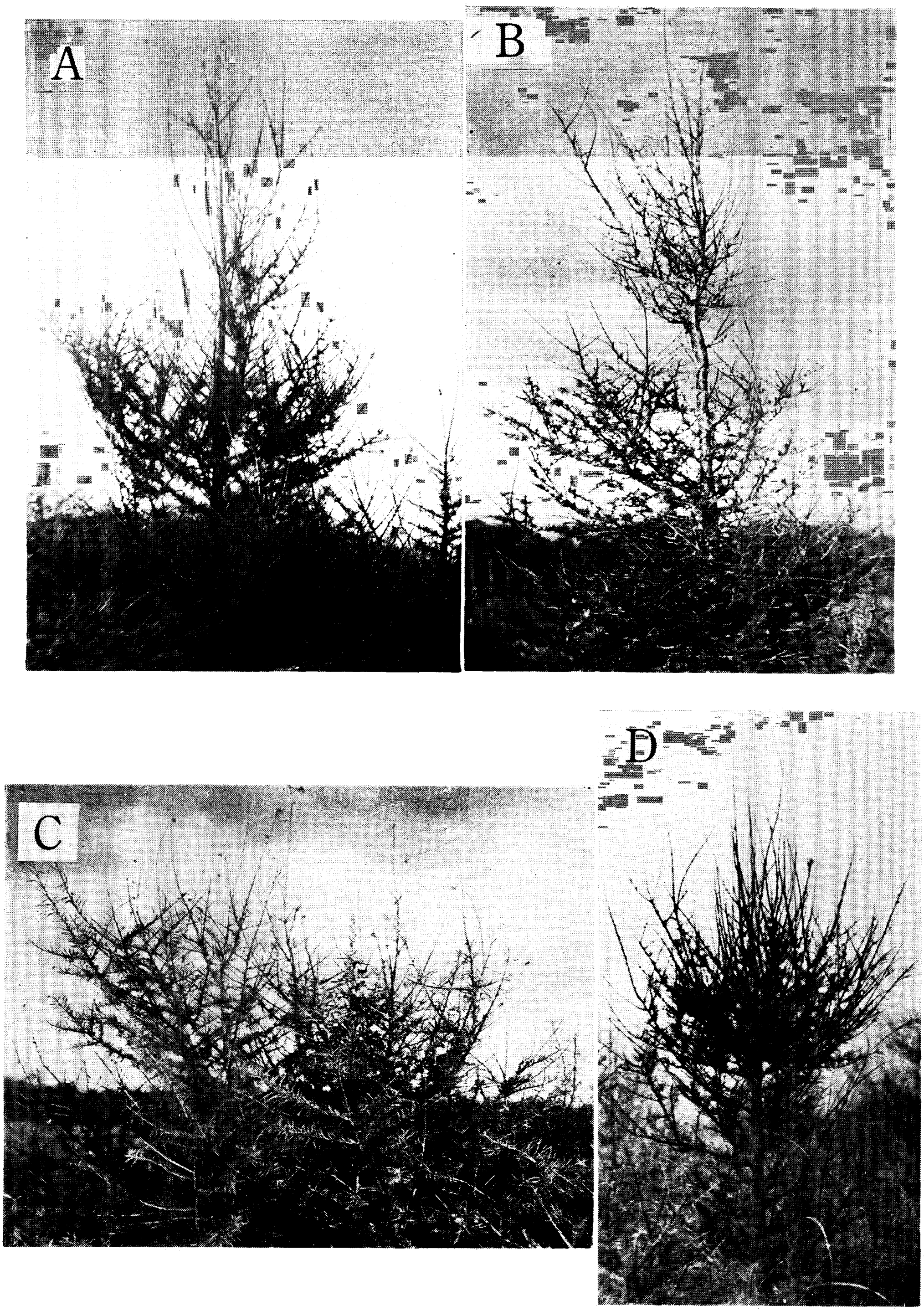


By courtesy of Zenkoku Ringyo Kairyo Fukyu Kyokai, Tokyo.

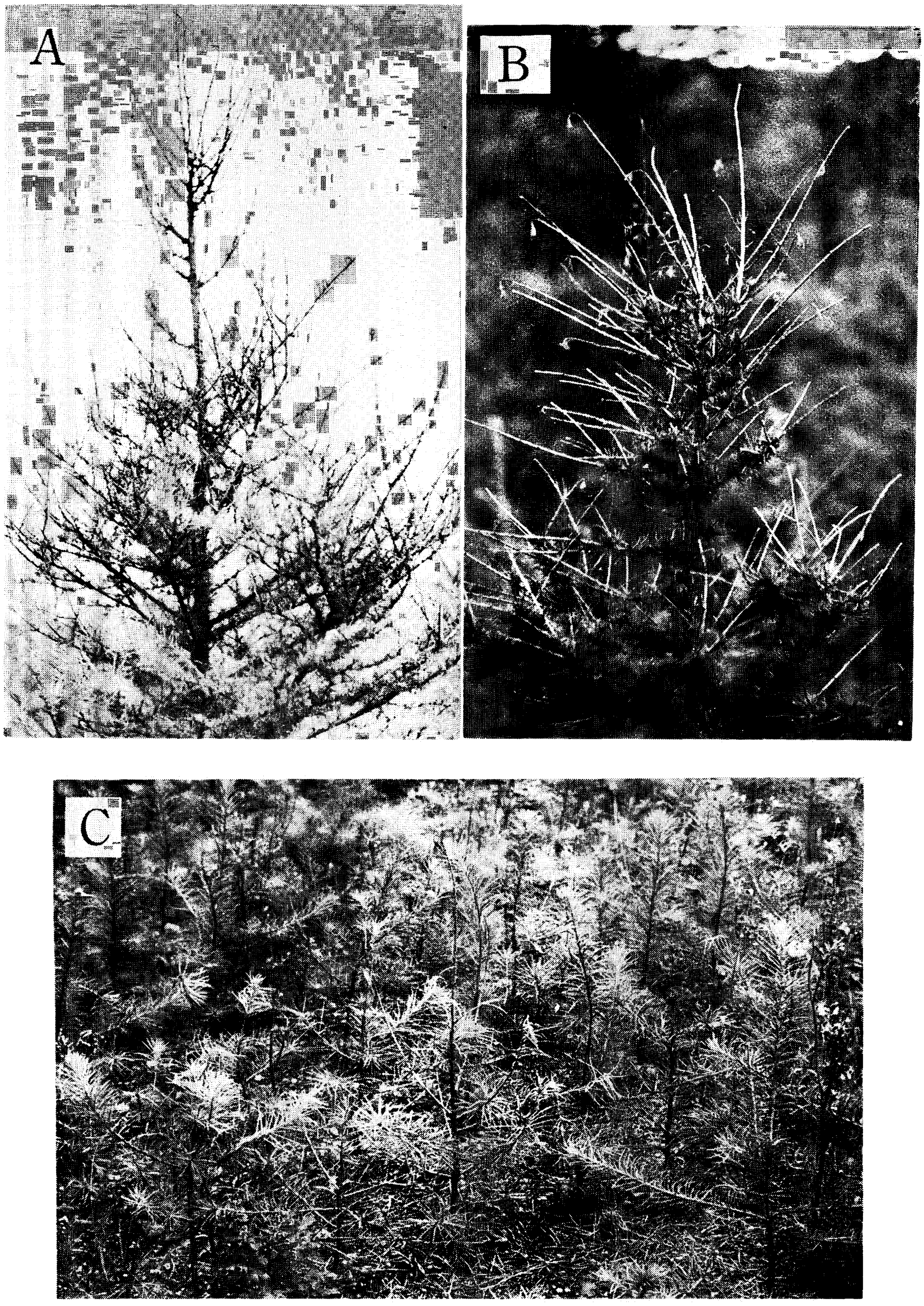
—Plate 2—



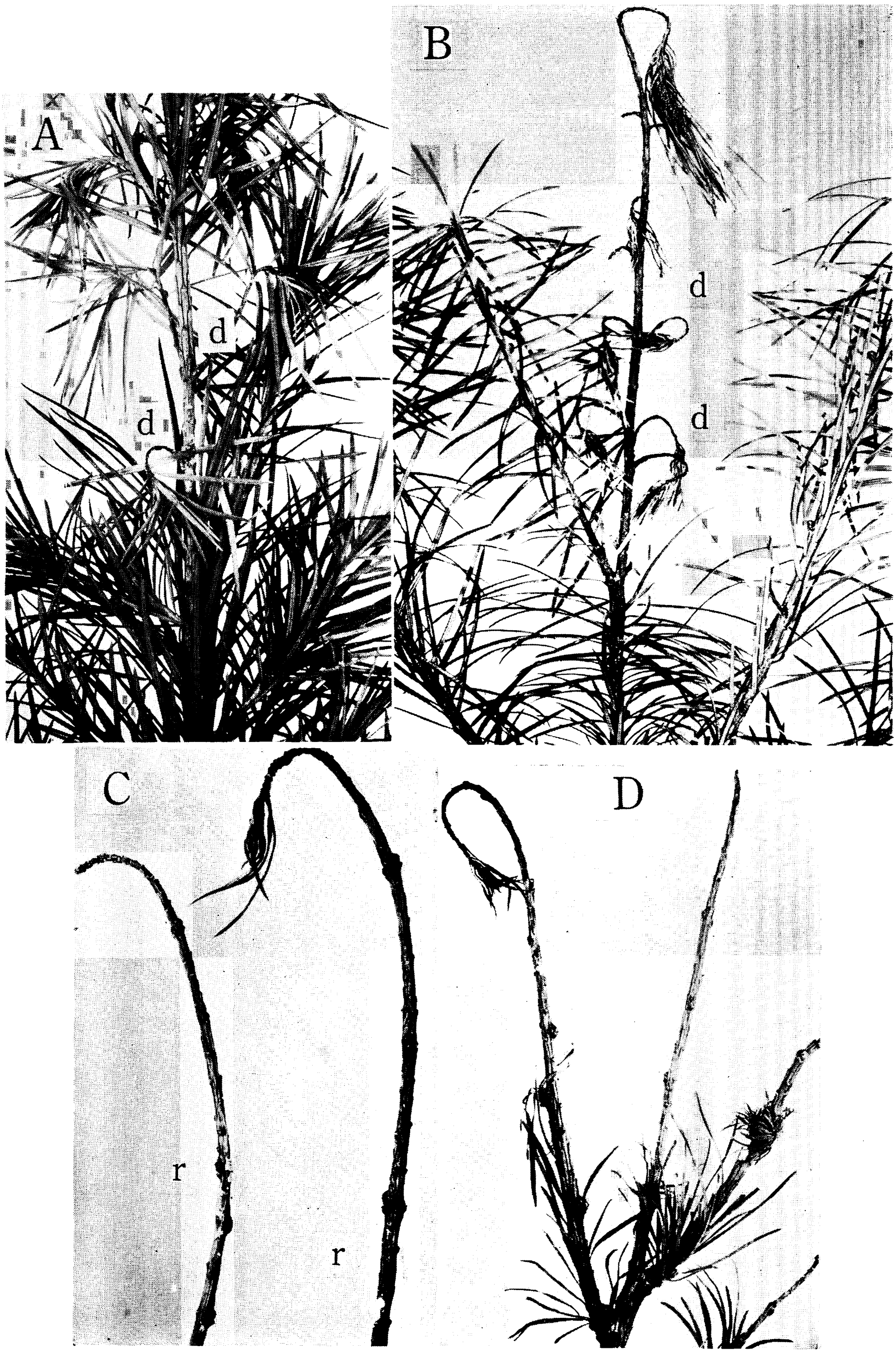
—Plate 3—



—Plate 4—



—Plate 5—



—Plate 6—

—Plate 7—