Historic, archived document

Do not assume content reflects current scientific knowledge, policies, or practices.
REVIEW OF LITERATURE ON
CINCHONA DISEASES,
INJURIES, AND FUNGI

By
FRANCES F. LOMBARD
Assistant Pathologist
Division of Forest Pathology
Bureau of Plant Industry, Soils, and Agricultural Engineering
Agricultural Research Administration
REVIEW OF LITERATURE ON
CINCHONA DISEASES,
INJURIES, AND FUNGI

By
FRANCES F. LOMBARD
Assistant Pathologist
Division of Forest Pathology
Bureau of Plant Industry, Soils, and Agricultural Engineering
Agricultural Research Administration
# CONTENTS

<table>
<thead>
<tr>
<th>Seedbed diseases</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Damping-off</td>
<td>2</td>
</tr>
<tr>
<td>Mechanical injury</td>
<td>8</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Transplant bed diseases</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Leaf spots</td>
<td>9</td>
</tr>
<tr>
<td>Other leaf diseases</td>
<td>11</td>
</tr>
<tr>
<td>Stem blights</td>
<td>12</td>
</tr>
<tr>
<td>Stem cankers</td>
<td>17</td>
</tr>
<tr>
<td>Root rots</td>
<td>18</td>
</tr>
<tr>
<td>Nematode root infections</td>
<td>20</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Plantation diseases</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Leaf spots</td>
<td>21</td>
</tr>
<tr>
<td>Other leaf diseases</td>
<td>25</td>
</tr>
<tr>
<td>Bark diseases</td>
<td>27</td>
</tr>
<tr>
<td>Systemic diseases</td>
<td>29</td>
</tr>
<tr>
<td>Trunk and branch cankers</td>
<td>29</td>
</tr>
<tr>
<td>Root and collar rots</td>
<td>36</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Injuries attributed to environmental factors other than soil conditions</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Drought</td>
<td>44</td>
</tr>
<tr>
<td>Frost or freezing</td>
<td>44</td>
</tr>
<tr>
<td>Light</td>
<td>44</td>
</tr>
<tr>
<td>Lightning</td>
<td>45</td>
</tr>
<tr>
<td>Rain and hail</td>
<td>45</td>
</tr>
<tr>
<td>Shade</td>
<td>45</td>
</tr>
<tr>
<td>Smoke</td>
<td>45</td>
</tr>
<tr>
<td>Wind</td>
<td>45</td>
</tr>
</tbody>
</table>

| Miscellaneous fungi and lichens                                      | 45   |
| Mycorhizae                                                           | 48   |
| Literature cited                                                     | 49   |
| Index                                                                | 66   |
REVIEW OF LITERATURE ON CINCHONA DISEASES, INJURIES, AND FUNGI

By

FRANCES F. LOMBARD

Assistant pathologist, Division of Forest Pathology, Bureau of Plant Industry, Soils, and Agricultural Engineering, Agricultural Research Administration

CINCHONA, the natural source of quinine, is a native tree of the slopes of the Andes in South America from Venezuela to Bolivia and in a comparatively limited area in Central America. Quinine and related alkaloids, which are extracted from cinchona bark, are the well-known medicaments for the treatment of malaria. Following the discovery of the medicinal value of the bark during the seventeenth century and the subsequent wholesale harvesting of the wild barks for the European market, efforts were made to introduce cinchona into other parts of the world. In 1865 Charles Ledger sent seeds of a high-alkaloid-yielding variety of Cinchona officinalis to England (185). Part of this shipment of seeds was sold to Dutch planters in Java, who through careful selection and breeding over a period of years were able to develop stock with a quinine content of 3 to 13 percent (99). Aided by the possession of this superior variety, known as C. officinalis var. ledgeriana, planters of the Netherlands Indies became the foremost producers of commercial bark. When the Netherlands Indies fell to the Japanese during World War II, it was producing more than nine-tenths of the world’s supply of cinchona bark (99).

Malaria occurs intermittently in northern and southern Africa, the Mediterranean countries, most of southern Asia and Asia Minor, all of the South and Central American countries north of the Rio Plata, and in most of the Pacific tropics (283). It is endemic in 17 of the Southern and Southwestern States of the United States of America (283). The quinine requirements for human consumption alone in the United States were estimated at 3,000,000 to 4,000,000 ounces per year before the war, of which approximately 60 percent was used by malarial sufferers (190). Following the outbreak of war, deaths from malaria throughout the world were estimated to have increased from 4 to 6 million (283).

1 Publication is made possible by funds provided through the Interdepartmental Committee on Scientific and Cultural Cooperation, Department of State, in cooperation with the Office of Foreign Agricultural Relations, Department of Agriculture. Valuable assistance was rendered by May Coult, translator, Office of Foreign Agricultural Relations, who translated most of the foreign literature used in the preparation of this manuscript. Thanks are due also to John A. Stevenson, principal mycologist, and Edith K. Cash, associate mycologist, Division of Mycology and Disease Survey, Bureau of Plant Industry, Soils, and Agricultural Engineering, for checking mycological details during the course of the preparation of this publication.

2 Italic numbers in parentheses refer to Literature Cited, p. 49.
During the war the important commercial sources of quinine were controlled by the Japanese. To meet the wartime needs of the Allied armed forces and the future needs of the civilian populations, the United States and other countries in the Western Hemisphere cooperated in developing the cinchona industry in this hemisphere. At the same time that cinchona production on existing and new plantations in the Western Hemisphere was being stimulated, bark was being harvested from the regenerated natural stands in Colombia, Ecuador, Peru, and Bolivia. About 18,000 tons were harvested from these wild stands in South America during a 2½-year period (81). Until a completely satisfactory synthetic product is available, prudence demands that all possible encouragement be given to the establishment of a cinchona culture sufficiently expansive to provide for any exigencies that may arise in the Western Hemisphere.

This publication has been prepared in an effort to bring together the world literature on the diseases of cinchona. With the introduction of cinchona into the Netherlands Indies, India, and other countries, the problem of developing the proper planting techniques for an exotic species and also the problem of preventing and controlling diseases confronted the planters. Many of the early workers recognized the threat to the new industry of uncontrolled diseases in the plantations and the advisability of combating them. When the plants could be brought to harvesting age, however, the incidence of disease assumed a relatively unimportant aspect, particularly where high-alkaloid-yielding varieties were used in large-scale plantings and a consequent good economic return could be realized despite the diseases.

It is not surprising therefore that the main efforts of the early workers were directed toward establishing the plant rather than investigating the disease-producing organisms and that some of the reports and descriptions of the various diseases are so general as to contribute little to the discussions based on the more accurate investigations of later workers.

Present-day pathologists and mycologists will recognize as saprophytic or weakly parasitic many of the fungi that were reported by early writers as occurring on cinchona plants or causing their diseases or death. Since the purpose of this publication is to review the pathological literature on cinchona, most of these earlier reports are included for the benefit of investigators who may not have access to them and who may wish to have the information contained in these older publications at hand.

SEEDBED DISEASES
DAMPING-OFF

The term “damping-off” is used here for any fungus infection that causes the rapid decay or death of young succulent seedlings (119). The disease is of world-wide distribution, affecting many hosts in nurseries and greenhouses (19). The fungi that cause damping-off occur commonly in the soil; and under favorable conditions of moisture and temperature the fungus hyphae attack
the host plants by directly penetrating the host tissues (19). Some fungus species that cause damping-off attack a great variety of hosts, whereas others have a rather limited host range (119). Among the fungi that have been reported to cause or that are associated with damping-off are: *Phytophthora cactorum* (L. and C.) Schreo., *P. cinnamomi* Rands, *P. parasitica* Dast., *Pythium* spp., *Botrytis cinerea* Pers., *Fusarium* spp., *Pestalozzia funerea* Desm., and *Rhizoctonia* sp. (*Corticium vagum* type) (19). A number of fungi that cause damping-off in seedbeds may continue to cause losses in transplant beds a or may cause other diseases on the mature plants (119). The so-called “late damping-off” of seedlings that have developed woody supporting tissues is usually considered an early stage of root rot (50).

Several diseases that have been reported on cinchona can be classed as damping-off diseases, although the authors may not have used that name specifically in reference to them. For example, Loble (174) stated that in cinchona seedbeds fungus infection is most likely to occur just after the seeds have begun to germinate. Following is a discussion of fungi that have been reported to be associated with or to cause damping-off.

**Cladosporium**

Losses in cinchona seedbeds caused by a fungus that attacks the radicle before it has penetrated the soil have been reported at Mulungu and Tshibinda, Belgian Congo, by Hendrickx (122), Stoffels (262), and Feilden and Garner (78). The fungus destroys the positive geotropism of the rootcap (122), with the result that the radicle does not enter the soil and the seedling is left lying flat on the ground (78). Correct watering and good ventilation during the period of germination retard the development of the fungus (78). Spraying with a copper fungicide at the time the seeds begin to swell also achieves some control, according to Feilden and Garner (78). Hendrickx (122) stated that an *Alternaria* was isolated from the diseased material but that inoculation tests had not been made. Later, Stoffels (262) wrote that the causal organism has been found to be a physiologic race of *Cladosporium herbarum* (Pers.) Lk.

*Cladosporium herbarum* (Pers.) Lk.—“Caespitulis dense aggregatis, confluentibus, stratum velutinum flavo-olivaceum dein atro-olivaceum consti-
tuentibus; hyphis erectis vel adscendentibus, brunneis vel olivaceis, paullum ramosis, septatis, 5-7μ crassiss; conidiis prope hypharum apicem nascentibus, non vel parce concatenatis, dilute brunneis vel olivaceis, forma et magnitudine variabilissima, oblongis, ovoideis, simplicibus vel oblongo-ellipticis cylin-
draceisve, 1-3-septatis, ad seipmenta constrictis, levibus.” (242, v. 4, pp. 350-351.)

---

a The term “transplant beds,” as used in the organization of this publication, includes all nursery beds in which the cinchona plants are grown from the time they are moved from the original seedbeds until they are placed in permanent field locations. The terminology used in the text follows as closely as possible that used in the original articles cited. Scientific names of fungi given within parentheses and indicated by sign of equality (=) are added to bring the nomenclature to date.
Clitocybe

In 1940 Heim (120) reported a destructive seedling wilt of Cinchona ledgeriana and C. succirubra in French Guinea. The seedlings wilted and died in patches in the seedbeds when the cotyledons were about 4 to 5 mm. long. Numerous white rhizomorphs of a tropical species of Clitocybe were found in the soil of the affected patches; and on contact with the cinchona rootlets the rhizomorphs produced mycelial threads that penetrated the host tissues. The methods recommended by Heim for preventing and combating the trouble were: Presterilization of seedbed soil by using formaldehyde (2 to 4 percent), aromatic nitrated phenols (ortho-2-4-dinitrophenol), or steam; the removal of the Clitocybe mycelial mats from the beds; and careful drying of the seed flat soil in the sun and air.

Clitocybe sp.—Fruiting body abortive, sterile; stipes dirty white, 22 mm. high; caps less than 3 mm. in diameter, with triangular profile, flattened on the top; peridium dark blue gray, highly involute; gills plicate, about 24 in number, thick, strongly decurrent, whitish, with sterile hymenium; flesh whitish, odorless; cap and stipe not separable; rhizomorphs Milky white, branching, 250µ to a few microns in diameter, composed of “constituent hyphae” and “protective hyphae”; constituent hyphae parallel, thin-walled, constricted, septate, colorless, constituting the central part of the rhizomorphs; protective hyphae of irregular contour, slender, not constricted, rarely septate, thick-walled, interlaced, often swollen, constituting the peripheral part of the rhizomorphs (120).

Fusarium

Barat (6) in 1931 reported a Fusarium attacking germinating cinchona seedlings in south Indochina. He stated that the seed appeared to have been infected because the Fusarium developed on seed that had been sown on sterilized sand.

(See fusarium stem blight, p. 12, and fusarium root rot, p. 38.)

Pestalozzia

A disease on cinchona seedlings at Pala, Sumatra, was reported by Hunger (131) to have been caused by Pestalozzia myristicae. The fungus attacked the cotyledons of the seedlings, and the plants died. Removing and burning infected plants immediately was advised.

Pythium

Tschirch (271) stated that according to Breda de Haan a species of Pythium attacks cinchona seedlings if the beds are improperly watered. Reinking 5 reported that a species of Pythium was present in cinchona seedbeds in which the seedlings were affected by damping-off in Guatemala.

4 The scientific names for the species of Cinchona used in the text are those given in the publications cited and cannot be checked for accuracy of identification.

Rhizoctonia

As early as 1882 Moens (189) recorded a disease that occurred in cinchona seedbeds in Java and that resulted in the death of seedlings in patches and was caused by a "cobwebby mycelium." Koorders (154) observed the same disease in Preanger, Java, on cinchona plants that were only a few days old. He found a thin, colorless mycelium in the stems, hypocotyl region, and roots of all dead or dying seedlings. In 1908 and 1915 Rant (220, 228, 229) described the disease under the name "mopog" (usually called "mopog" in the Dutch literature). From his cultural studies he identified the fungus as Moniliopsis aderholdii Ruhl. (229), which was shown later to be a synonym of Rhizoctonia solani Kuehn (= Corticium solani (Prill. and Del.) Bourd. and Galz. = Pellicularia filamentosa (Pat.) Rogers). Following Rant's investigations and identification of the fungus, numerous reports of the occurrence of mopog at the governmental cinchona stations and on private estates in Java and Sumatra have been made in the literature (13, 44, 45, 54-58, 61, 62, 64-67, 88-93, 100, 102-108, 110-112, 139-144, 148-150, 157-161, 167, 170, 205, 210, 227, 252, 259, 271). The disease was rather serious on a few private plantations in Java and Sumatra in 1926 and 1930 (157, 160), causing a 10 percent loss in the seedbeds in 1926 (157, 252).

Rant (228) stated that the mopog fungus may attack cinchona seeds either before or after germination. In some cases it attacks seedlings so young that at no time do the seedlings ever stand erect (161); and it also attacks seedlings large enough to transplant (92, 161). In the seedbed bare places containing dead plants and ungerminated seed appear and these are surrounded by diseased plants (13). The infected spots quickly enlarge under favorable moisture conditions, and a large number of seedlings may be lost in a short time (228). The infected seedlings collapse on the top of the ground and remain green for several days, but appear to be swollen (205). A dark spot may be seen on the lower part of the stem, and the seedling will break off at this point if touched (13). Rant (228, 229) stated that a characteristic symptom of mopog is the tendency of the particles of soil, ungerminated seeds, and dead seedlings to hang together by the mycelial filaments of the fungus when the soil is lightly stirred or lifted. If the humidity in the seedbed is high, the mycelium grows over the dead plants and seeds (229). Rant successfully inoculated hybrid cinchona seedlings 4 to 6 inches high with the mopog fungus and obtained the disease. Similar inoculations with Botrytis cinerea Pers. failed to give the typical mopog symptoms (229).

Careful adjustment of light and ventilation and moderate watering with a fine spray were recommended by Rant (229) as preventive measures. Keuchenius (147) reported that, although 5 million plants were started in the seed and transplant beds in West Sumatra in 1935, there were no losses from mopog, because the germination houses were built so as to allow proper regulation of the relative humidity. The addition of wood ashes to the soil of infected seedbeds is said to discourage the growth of the fungus,
(229) and Stoffels (262) advised replacing contaminated patches of seedbed soil with wood ashes or sulfur. Keuchenius stated that mopog was successfully controlled by spraying with a Cryptonol solution (149) and that the Bayer fungicides "Nosperit" (75-per cent solution) and "Terbolan" (5-percent solution) were being tested (150). Coster (45) reported that Rhizoctonia solani could be controlled with "Superol."

Rhizoctonia solani Kuehn.—Hyphae practically colorless when young, vacuolate, more or less irregular, septate with septa at intervals of 100μ to 200μ; some development of external hyphae on certain hosts; external hyphae somewhat colored, usually yellowish brown, and generally of two types, a purely vegetative type and a second type constituting the short tufted or mealy growth as occurring on certain hosts; vegetative hyphae, 8μ to 12μ, branches when young pointing in the direction of growth and somewhat constricted at point of union with main hyphae, branches when older approximately at right angles to the main hyphae, more deeply colored and more uniform and rigid, constrictions less marked and septa farther apart; hyphae of tufts profusely branched when young, lobed, sometimes botryoid, becoming light brown with maturity, ultimately dividing into short hyphal lengths or single ovate cells, arranged in short chains or elbowed and producing branches in a more or less dichotomous fashion, the single cells somewhat resembling conidia, which may germinate by tube within a few hours under suitable conditions, the denser masses in culture giving rise to sclerotia; hyphae in tissues colorless while in active growth, smaller in diameter but otherwise similar to the young external hyphae; sclerotia of fairly homogenous structure, generally more or less flattened, irregular, deep chestnut brown, generally smooth under natural conditions, ranging from a minute size to 1 to 2 cm. in diameter (51, pp. 439-444).

In 1941 Madarang (182) reported that damping-off is estimated to cause a 20 to 30 percent loss in the Government cinchona plantation at Bukidnon, P. I. The disease is said to be most injurious from the time the seedlings emerge from the soil until they are about 2 inches high. A species of Rhizoctonia, similar to or identical with R. solani Kuehn, was isolated from infected seedlings. This disease occurs in patches in the seed flats; and when it is allowed to go unchecked, most of the seedlings may be lost. The fungus prevents seed germination by attacking the radicle and cotyledons before they emerge from the seed, finally causing them to deteriorate. Also seedlings that have emerged are attacked at or near the ground line, and when the stems have rotted at the point of infection the seedlings topple over. The infected seedlings continue to decay if the soil and atmosphere are moist or they wilt and shrivel if the atmosphere is dry. Occasionally leaves touching the soil may become infected, while the rest of the plant escapes. Heavy mycelial growth may smother small seedlings.

Soil inoculation tests to prove the pathogenicity of the Rhizoctonia were successful on Cinchona succirubra, and the causal organism was reisolated. Limited inoculation tests on other host plants—a native species of tomato, Irish potato, and Pinus insularis Endl.—showed the cinchona strain of Rhizoctonia to have a narrow host range. Sterilization of the soil by steam pressure at 15 pounds for 2 hours or drenching the soil with 40-percent commercial formaldehyde diluted 1 : 50 with tap water and applied
at the rate of 2.5 liters per square foot before seeding effectively checked damping-off of cinchona seedlings. Seed dusted with cuprous oxide at the rate of 2 grams dust to 100 grams seed showed a higher percentage of infection than the controls and a very low percentage of germination. The poor germination of the seed dusted with cuprous oxide was believed to have been due to injurious effects of the dust or to have been caused by preemergence infection in the presence of the dust or a combination of the two factors (182).

Sundararaman (265) reported in 1928 that cinchona seedlings at the Government cinchona plantation at Anamalai, Madras, India, were dying in patches from a damping-off caused by Rhizoctonia sp.

Rhizoctonia sp. is reported to have caused 100 percent loss in certain cases in the cinchona seedbeds in Belgian Congo (135). Mercuric chloride and formaldehyde when used as soil disinfectants retarded infection but did not suppress it completely (135).

The organisms causing damping-off of cinchona in Guatemala have not been identified; however, Reinking ⁶ reported in 1945 that a species of Rhizoctonia had been isolated from infected plants and a species of Pythium was present also in affected beds. The disease was observed on C. ledgeriana and C. calisaya and was not serious in well-managed plantings visited by Reinking.⁶ Control measures suggested were the use of new soil in each new seedbed and soaking the soil of severely attacked beds with bordeaux mixture or Yellow Cuprocide.⁶ Popenoe (209) stated that cinchona is rarely attacked by damping-off if the seed is sown at the beginning of the dry season (January or February in Guatemala) and if the seedlings make 2 or 3 months’ growth before the heavy rains begin.

(See rhizoctonia root rot, p. 18.)

Undetermined

In 1877 a disease that affected cinchona seedlings immediately after germination was reported in Jamaica (241).

In 1880 seedlings at the Government cinchona plantations in Sikkim, India, were reported to have died, owing to the occasional presence of a fungus mycelium in the seedbeds during very damp weather (153). Gently stirring the soil was said to have some value in checking the disease (153).

A fungus disease that occurs during periods of excessive humidity in the “nurseries” in Peru was reported by Augusto (5) in 1943. Bordeaux mixture is used to control it (5).

Damping-off in Puerto Rico is said to be caused by a basidio-mycete (216). Although the causal organism has not been identified, control measures have been investigated. Semesan and Cuprocide dusts used as seed and seedling protectants against damping-off were found to be harmful to the seed and to reduce

⁶ See footnote 5, p. 4.
the stand by 90 percent or more; however, liquid applications after germination were not injurious to the plants and appeared to control the spread of the disease (261). Untreated seed and seed treated with calcium hypochlorite, when planted in soil disinfected with formaldehyde and pretreated with hot water, germinated equally well, and neither lot of seed was attacked by damping-off organisms (215). Cinchona seeds, which were disinfected with calcium hypochlorite and planted on sterile agar slants for germination counts, were attacked by an unidentified fungus (215). The ridged, uneven surface of the seed coat was thought to be responsible for the inadequate disinfection, permitting the development of the fungus growth (215).

Boecop (15) reported a "root collar" disease in Java that affected cinchona seedlings about 1 to 2 cm. high. The stem at the soil level is infected, while the root remains healthy and the leaves remain green for a considerable time after the plant becomes affected. In the early stages of the disease the plants show a tendency to droop slightly and, when brushed lightly with the hand, do not spring back to an upright position. The seedlings fall flat on the ground finally and break off at the collar when an attempt is made to pull them up. The disease occurs in patches in the seedbed. Boecop was unable to isolate a fungus from either the diseased plants or the soil. He stated that control was obtained by removing the infected plants and soil, leaving the vacant space open to the air and light, and withholding water from the bed for a week.

**MECHANICAL INJURY**

**Coprinus and Marasmius**

Mechanical injury to seedlings caused by the lifting of the soil when the fruiting bodies of species of *Coprinus* and *Marasmius* are formed has been reported by Hendrickx (122) in seedbeds at Mulungu and Tshibinda, Belgian Congo.

**Stemonitis**

The suffocation of cinchona seedlings by the plasmodia of a myxomycete was reported from the Preanger and East Java, Netherlands Indies (154, 167, 193, 227, 271, 286). The affected plants were about 2½ cm. in height, and both sides of the leaves were thickly covered with the fruiting bodies of a species of *Stemonitis* (286). The seedlings had died when the overgrowing plasmodia obstructed the stomata of the leaves and cut off exposure to light (286). Wurth (286) recommended spraying the plasmodia with common salt or saltpeter or, after the formation of sporangia, the removal and burning of the affected plants and disinfection of the soil with unslaked lime or 1-percent carboxlic acid.

---

Turf fungus

Cinchona seedlings were reported to have died as the result of an unusual condition in the seedbeds at the Government cinchona station in Java (169, 224, 226, 227). In some spots in the beds the seeds had not germinated, and in other beds there were spots of backward plants among the healthy ones (224). The affected plants were small, and the leaves were red (169, 224). Examination of the affected seedlings failed to show any causal organisms (224). The ground felt peaty and elastic to the touch; and when such a spot was dug out, a brown mass of fungus mycelium was found a few centimeters below the surface (224). The fungus was found growing luxuriantly and forming thick brown rhizomorphs (169, 224). In the absence of fructifications Rant was unable to identify the fungus (224). If the plants succeeded in penetrating the mass of mycelium, they grew normally, although they remained smaller than the unaffected plants (224). In 1936 Goot (92) reported that a comparatively large number of plants were lost on an estate where the soil used in the seedbeds was forest humus and full of the turf fungus. Rant (224) advised replacing the contaminated seedbed soil with soil from another place in the forest or making a new seedbed.

TRANSPLANT BED DISEASES

LEAF SPOTS

Colletotrichum

Koordiers (154, 155) described a new species, Colletotrichum cinchonae, causing a leaf spot on 1-year-old seedlings of a cinchona hybrid in Java. The seedlings were growing at an altitude of 1,500 meters above sea level (227). The leaf spots were pale and the sori scattered (155, 227). The plants attacked by this fungus had already been attacked by an undetermined fungus and the Colletotrichum species did no serious damage (155, 227). Cinchona calisaya plants in the Transcaucasian region of Russia (U.S.S.R.) are reported to have been attacked by Colletotrichum cinchonae; however, the leaf spots were reddish brown (210).

Colletotrichum cinchonae Koord.—“Maculis pallidis foliicolis; acervulis sparsis, punctiformibus, epidermide erumpentibus, vix 0.1 mm. diam.; sporulis oblongis, subrectis v. curvulis, utrinque obtusis v. rotundatis, 9–18 × 3–5, plerumque 10–12 × 3.5–4, hyalinis; setulis paucis, fuscis, 1-pluri-septatis, subrectis v. curvulis v. flexuosis, 60–75μ longis, basi 4–7μ diam.; sporophoris cylindraceis, hyalinis, c. 10μ longis.” (242, v. 22, p. 1203; also described in 155, 227).

Gloeosporium

Bugnicourt (24) reported a withertip of cinchona seedlings in South Annam, Indochina. The affected seedlings were from 8 months to 1 year old. The infection started with the terminal bud. The upper three or five internodes withered and shriveled, and the adjacent leaves and diseased parts turned black. The whole top drooped. When the disease was allowed to develop, the infection spread downward and the plant died. A species of Gloeosporium was isolated. Bugnicourt suggested that the initial
infection might have been caused by infected seed. He advised disinfection of seed, cutting off the diseased parts, painting the pruning wounds, and spraying with bordeaux mixture.

Bugnicourt (24) also described an anthracnose leaf spot affecting young plants in seedbeds and nurseries in Laos, Indochina. The leaf spots, which were brown at first and then turned yellow, affected the entire thickness of the blade. Later the affected parts of the leaf dropped out. The leaf disease was also caused by a species of Gloeosporium, and Bugnicourt considered it a form of the withertip disease in South Annam.

_Gloeosporium_ sp.—Mycelium in culture dense, white, hyphae 2μ to 3μ, forming fine orange droplets; conidia abundant, hyaline, somewhat cylindrical; conidiospores 15μ × 4.5μ (24).

(See gloeosporium leaf spot, p. 22.)

**Lapp disease**

Lapp disease, or "leaf scab," occurs on the leaves of _Ledgeriana_ grafts and seedlings in the seedbeds, nurseries, and plantations in Java and Sumatra (61, 109, 126, 132, 144, 147, 148, 167, 168, 227). Grafts are said to suffer considerably less injury than the young seedlings (147). The infected plants have curled or wrinkled brittle leaves that tear easily (167). The leaves are red at the edges, and brown spots with red borders are found between the veins (167). Occasionally only one part of a plant is affected, the rest of the leaves being healthy (227); and the plants or affected parts generally appear to be rather slender (227). The disease may attack widely scattered plants in the plantations or plants that are growing close together in the seedbed (167).

Although the occurrence of the disease has been noted in the reports from the Netherlands Indies for about 30 years (1907-36), the cause is still unknown. Microscopic examination and attempts to isolate a causal organism from affected plants gave negative results (168). Rant (as reported by Leersum, 168) was of the opinion that the type or condition of the soil was responsible for the disease, although he indicated that insects might be the cause of the damage. Hall (109) stated that some plants affected with Lapp disease could always be found in young plantations and nurseries, but as the disease is generally limited to a comparatively small number of plants only a small extent of damage is done. He reported that the disease had broken out so severely in Sumatra in 1922 in both the nurseries and young groves of _Ledgeriana_ seedlings, however, that a very large number of plants had been killed (109). Hunger (132) stated that in 1907 no attempt to combat the disease was necessary, as it gradually diminished and finally disappeared.

**Phyllostictina**

In recent years (1935-1938) a species of _Phyllostictina_ has been reported to occur periodically in cinchona nurseries as a leaf disease on young grafts of Clone R. G. 1 in West Sumatra (93, 147, 148, 150). This fungus is believed to be identical with _Phyl-
losticta cinchonae Koord. (147). The application of lime, nitrogen, and phosphoric acid to the soil and repeated spraying with bordeaux mixture are said to give satisfactory control (147).
(See Phyllosticta cinchonae, p. 23.)

OTHER LEAF DISEASES

In 1921 and 1922 Vincens (273, 274) reported a disease in the cinchona nurseries at the Honbà station, Annam, Indochina. Cinchona ledgeriana was particularly susceptible, although C. succirubra and a hybrid (C. ledgeriana × C. succirubra) were also attacked (274). The diseased Ledgeriana plants were only 15 to 20 cm. high, while the better developed Ledgeriana plants were about 35 to 40 cm. in height (274). No plant was entirely healthy, heavily spotted and often perforated leaves being found on plants least affected (274). Older leaves, showing comparatively little damage, had circular brown spots a few millimeters in diameter with purplish-blue borders or perforations from which the contents of the leaf at those places had dropped out (274). The spots were more numerous on the younger leaves, often uniting to form checkerboard designs occupying a large part of the leaf area (274). The leaves near the top of the plants were misshapen, wrinkled, and tattered looking, similar in appearance to leaves severely attacked by anthracnose (274). The young green shoots were bent and deformed, sometimes slightly enlarged, and occasionally had abnormal suberose formations with a cankerous appearance (274). Only a few misshapen leaves remained on the weak, dying plants, the majority having already fallen to the ground (274).

The disease showed the same characteristics on the hybrids, except that the shoots of the hybrids looked healthy despite the diseased condition of the leaves (274). Badly infected leaves were comparatively scarce on the C. succirubra plants and all the shoots were healthy (274). Vincens (273) stated that according to Dr. Yersin, Director of the Honbà Station, the same disease has resulted in much damage in Java where its cause is unknown.

The disease at Honbà was said to be favored by the frequent mountain mists (274). The use of copper sprays proved both ineffective in the control of the disease and injurious to the young cinchona plants (273, 274). New plantings of Ledgeriana were free from disease, and further studies on control measures were unnecessary (274). In the earlier report Vincens stated that the disease appeared to be caused by a species of Guignardia (273); however, in his later report he said that it was impossible to observe any parasite on the majority of the diseased leaves still attached to the plants and that no parasite was found on or near the suberose young shoots (274). Parts of diseased plants later revealed the fruiting bodies of several fungi, however, and these were described by Vincens (274) under the following names: Dendrophoma cinchonae Vincens, Guignardia yersini Vincens, Phlyctaena cinchonae Vincens, Phoma cinchonae Vincens, Phyllosticta cinchonaeacola Vincens, P. honbaensis Vincens, P. yersini
Vincens, and Physalospora cinchonae Vincens. Vincens stated that there was not much doubt that the Phyllosticta species were harmful and that the frequent occurrence of the Phyllosticta in the abnormally suberoser bark of the plants led him to consider it a parasite (274). Phyllosticta yersini Vincens was reported on the leaves of cinchona plants in a greenhouse in Russia (U.S.S.R.) (18). (See Phyllosticta, p. 22.)

**STEM BLIGHTS**

**Diplodia**

Arens (3) reported that Cinchona ledgeriana plants, which had been topped and potted preparatory to an entomological investigation in Java, had been killed by Lasiodiplodia theobromae Griff. and Maub. The fungus attacked the plants at the wound, the newly formed shoots were killed back, and finally when the roots became infected the plants died (3). According to Nowell (194) *L. theobromae* is a synonym of Diplodia theobromae (Pat.) Nowell. Diplodia theobromae (Pat.) Nowell.—“B. perithecii gregariis, 200μ, latis, atris, plus minus villosis, stromate viloso, atro, junctis; basidiis elongatis; hyalinis (50μ) apicem versus incrassatis; sporulis ellipticis, 1-septatis, hyalinis dein brunneis, 25–35 × 12–15μ.” (201, p. 136; also described in 194 and in 242, v. 11, p. 522.)

(See Diplodia theobromae, p. 35.)

Roepke (237) reported that cinchona plants being used in entomological investigations in Java were killed by the Diplodia fungus.

**Fusarium**

An epidemic wilt disease that appeared in the Munsong cinchona plantation in Bengal, India, in 1926–27 (30), and later at Mungpoo (32), continued to be quite serious for about 10 years (30–35, 84, 178, 188, 199, 200, 246, 247). A few plants became diseased in October of one year, and by April and May of the following year the disease had become of such epidemic proportions that thousands of plants had died (178). The disease is believed to attack the plants at midnursery age (31), but usually develops just after the seedlings are set out in the field (30). Coppice shoots are said to be immune (31). Plants in 2- or 3-year-old nursery beds are more severely attacked than those in new beds, although the new beds are not entirely free of the disease (178). The disease may appear in nurseries at both high and low elevations (178). On plants 15 months old the leaves turn dingy yellow and then red and finally wilt (178). Slight longitudinal depressions appear in the bark (178), extending from the collar up to the first or second branch (30). The largest lesions are about one-fourth inch wide and several inches long (178). The bark beneath the depressions becomes brown and dry, and the brown area extends beyond the cambium into the wood (178). Mildly attacked plants may throw off the disease after being planted in the open (32); more severely attacked plants die after a period
of varying length (30). Fungal hyphae were found sparingly near the advancing edges of the lesions (178).

During the investigations on the disease, species of Botryodiplodia, Diplodia, Fusarium, Nectria, Sporotrichum, and Verticillium were isolated from a large number of the diseased plants (188). Inoculations with some of these organisms gave negative results (188), but the Sporotrichum and Verticillium isolates gave positive infections (181). Galloway (84) suggested that the Diplodia might be the causal organism. In 1939 Padwick (199) stated that a species of Fusarium was regularly isolated from the vascular tissues of the diseased plants; and Mitra (188) reported that the Fusarium had been isolated from both the roots and the collar region. One-year-old seedlings were inoculated with the Fusarium after damaging the bark, and seed and transplant bed tests were made by infecting the soil with three Fusarium isolates (200). In these tests infection had not yet taken place at the end of 3 months (200). Selection of disease-free plants for the plantation at the time of lifting from the nursery beds is difficult because the disease symptoms usually are not evident at that time (34). The continual development of new nursery bed sites (31, 35), disinfection of the nursery soils and sites (31), and transplanting to the plantations at an earlier age (178) have reduced the losses somewhat, but have not proved to be very satisfactory control measures. The effectiveness of watering the beds with weak fungicidal solutions of “Kerol” and malachite green was being tested as a control measure (84).

Fusarium and Helminthosporium were reported by Keuchenius (150) to have caused injury to 4-month-old seedlings in the West Sumatra area in 1938. Spraying with a 5-percent Cryptonol solution was recommended.

(See fusarium damping-off, p. 4, and fusarium root rot, p. 38.)

Phytophthora

In 1936 Sawada (245) reported an epidemic disease affecting cinchona seedlings in a plantation in Formosa. The 1-year-old seedlings, which were 10 to 30 cm. in height, were attacked during the rainy season from May to August, and about half of the 20,000 seedlings in the plantation were lost. The fungus isolated from the diseased seedlings was a species of Phytophthora to which the author assigned the name P. cinchonae Saw. The first symptom to be noticed is a darkening of the stem, followed by the spread of the infection to the leaves, which shrivel and die, and finally by the death of the plant. Positive results were obtained in inoculation tests, and the causal organism was reisolated. Sawada recommended presterilizing the seedbeds with formalin, draining the seedbeds well, burning infected seedlings, and spraying the infected soil in the seedbed with bordeaux mixture.

Phytophthora cinchonae Saw.—Hyphae continuous when young, forming a few “pseudo-septa” in old age, branching profusely on media, thin, membranous, colorless, commonly 5μ to 6μ in diameter on soybean agar, 4μ to 8μ on millet broth agar, 4μ to 15μ on polished rice media, 7μ to 8μ on citrus
agiar; aerial hyphae 6µ to 11µ in diameter on millet broth agar, 4µ to 7µ on polished rice media, 6µ to 10µ on citrus agar; sporangiophores about 4µ in diameter; sporangia terminal, colorless, smooth, oval-shaped or sometimes dumbbell-shaped with rounded base and flattened papilla at apex, 18µ to 68µ × 13µ to 38µ (average of 48 specimens, 41.12µ × 23.0µ), secondary sporangia borne on short sporangiophores branching from below the terminal sporangia or by proliferating through the empty terminal ones, germinating to produce free swimming zoospores or by germ tubes; zoospores biciliate, 15µ to 17µ × 12µ to 13µ while swimming, 12µ to 16µ in diameter when at rest, germinating after a short while by germ tubes 2µ in diameter to form small sporangia 13µ × 10µ (which in turn produce zoospores) or by producing zoospores directly without forming the germ tubes; oospores not formed on artificial media (245).

Thompson (269) in 1940 reported a wilt of cinchona seedlings in the Cameron Highlands, Malaya, caused by Phytophthora cinnamomi Rands. Cinchona ledgeriana, C. succirubra, and a hybrid (C. succirubra × C. ledgeriana) were affected. The plants were attacked at the collar by a canker and bark rot and the stems split to a height of 3 to 9 inches. The leaves of the diseased seedlings had previously suffered repeated insect injury. The fungus also caused a root rot and dieback of mature trees.

Phytophthora cinnamomi Rands.—“Maculis in cortice Cinnamoni burmanni perpendicularius, elongatis, linearibus vel irregularibus, 1–5 cm. latis, saepe supra terram usque ad 10 M. in altitudinem extensis insita; cortice mortuo radiatim zonato. Mycelio in cortice et ligno exteriore, irregulari; hyphis aeris in agare avenae cultis tenuibus, 5–7µ crassis, primo continuis, deinde septatis; haustoria absent. Chlamydosphoris et inter et intra cellulas matricis formatis, tenuibus, globosis vel pyriformibus, plerumque 31–50µ in diametrum, in apice ramorum brevium nascentibus, copiosis in cultris artificialibus, saepe 3–10 in racemis coalitis; hyphis 3–11 germinantibus. Conidiophoris non a mycelio distinctis, simplicibus vel sympodio-ramosis. Conidios neque in natura neque in cultris artificialibus visis, sed copiose nascentibus in mycelio e solutione nutritia in aquam translato, primo terminalibus, plerumque ovoideis vel ellipsoides, hyalini, tenuibus; plerumque 38–84 × 27–39µ, fere 57 × 39µ latis, papillis latis, brevibus; conidios ulterioribus sympodiliter et ramis conidiophori nascentibus, saepe hypha fertile proliferata et conidiis vacuis ingrediens; parietibus conidiorum expulsione finita partim collapsis; conidios in aqua fere zoosporis, sed nonnumquam tubo vel conidiis secundaris germinantibus; conidiis e cultris infrimis vel contaminatis saepe abnormally germinantibus. Zoosporis 8–40, plerumque concavoconvexis, ciliis duabus inaequalibus longitudine, lateraliter insertis, mobilibus, motis circa 11 × 18µ, quietis 10–11µ in diametrum, tubo germinantibus. Oosporis non visis.” (219, p. 41.)

(See phytophthora root rot, p. 40.)

In 1934 Celino (39) reported a serious blight on C. calisaya var. ledgeriana, C. hybrida, and C. succirubra seedlings in the Philippine Islands caused by Phytophthora faberi Maub. Infection takes place in the tips of the young shoots of the seedlings, which suddenly wilt and dry up, and spreads rapidly downward, causing the death of the leaves and petioles (39). Occasionally, infection begins at the tips or margins of the leaves (39). The dead, dry leaves persist on the seedlings, hanging downward (39, 183). If the infection is unchecked, the seedlings die within 5 or 6 days (39).

*Tucker (272) recognizes Phytophthora faberi Maub. as a synonym of P. palmivora Butler. Descriptions of both P. faberi and P. palmivora are given for the reader’s information.
One-year-old *C. ledgeriana* and *C. hybrida* plants were inoculated on the uninjured young shoots and on the petioles of the leaves; by the second day after inoculation the shoots were discolored and wilted and the leaves were slightly wilted and hung downward (39). The causal organism was reisolated 6 days later and again in 10 days (39). Celino reported good control by removing and burning infected parts of the seedlings and spraying twice with standard bordelais mixture at an interval of 2 weeks (39). Ocfemia (195) also stated that *P. faberi* causes a seedling and shoot blight of cinchona.

*Phytophthora faberi* Maub.—“Maculis irregulariter brunneis; mycelio ramoso, primum continuo demum septato; sporangiophoris 150–200μ longis; haustoriis non visis; sporangii plerumque ovoides sed magnitudine variabilibus, 42–80 × 25–30; oogoniis intramatricalibus; antheridiis nullis v. raris; oosporis plerumque parthenogenetice ortis, cavitatem oogonialem late occupantibus, sphegoideis, elongatis v. irregularibus 22–45μ diam.” (242, v. 21, p. 860.)

As early as 1928 a seedling blight of cinchona was reported at the Mungpoo plantations in the Darjeeling District, Bengal, India (177). A species of *Phytophthora*, later identified as *P. palmivora* Butl.,\(^8\) was isolated and studied (33, 177-180); and in 1935 Kheswalla (152) reported the results of the cultural studies on the pathogen and the inoculation tests. The disease occurs in very wet weather or when the nursery has been overwatered (178). Usually, infection first becomes evident at the root collar, where the tissues become discolored, and then infection extends up the stem (152). The cotyledons wilt; and the leaves turn yellow and curl inward and, in severe cases, are shed (152). Occasionally, seedlings become infected at the tip and the infection spreads downward (152). When the discolored areas of the bark tissues have rotated, the plant droops (152). During damp weather copious mycelial growth may be seen on infected seedlings (152).

At Pusa (temperature about 27° C.) healthy cinchona seedlings 1 foot high were successfully inoculated either at the base of the stem or on the tender leaves and the fungus reisolated (152). Seedlings inoculated at Sureil (temperature about 20° C.) did not become infected (178). Other inoculations indicated that *Phytophthora parasitica* Dast. and *P. colocasiae* Rac. were unable to parasitize cinchona seedlings, while the *P. palmivora* isolate from palm gave only partial infection (152). All cinchona seedlings inoculated with the *P. palmivora* isolate from cinchona succumbed (152).

*Phytophthora palmivora* Butl.—“Maculas sicas, brunneas, profundas in vaginis fol. generans; mycelio intercellulari, haustoriis vero intra cellulas penetrantibus; hyphis latis, interdum irregulariter inflatis, 7μ diam., ramosis, haustoriis digitatis; sporangii obverse piriformibus, raro subrotundis, acrogenis, 50 × 35; zoosporis 8–10μ diam.; oosporis sphaericis, 35–45μ diam., membrana 4μ cr.” (242, v. 21, p. 855; v. 24, p. 36.) McRae (177) gave the extreme range in sporangia size of the cinchona isolate as 30μ–79μ × 21.6μ–45μ.

\(^8\)Tucker (272) recognizes *Phytophthora faberi* Maub. as a synonym of *P. palmivora* Butl. Descriptions of both *P. faberi* and *P. palmivora* are given for the reader’s information.
(See Phytophthora palmivora, p. 32.)

Crandall and Davis (49) described a Phytophthora disease affecting seedlings of *C. pubescens* Vahl., *C. officinalis* L., *C. officinalis* var. *ledgeriana*, and *Ledgeriana* grafted on *C. pubescens* in Guatemala and on *C. officinalis* var. *ledgeriana* in Peru and Puerto Rico. Infection appears to take place in the petiole region of the tender young leaves or in the succulent stem tissue. Brown necrotic spots with indefinite margins are formed on the leaves in the early stages of the disease. The fungus grows downward through the petiole and into the cambial region of the stem, and the seedling dies. A *Phytophthora*, tentatively identified as *P. parasitica* Dast., was isolated from Peruvian seedling, leaf, petiole, and stem material. Inoculation studies indicated that infection may take place through wounds on the stem or on uninjured bud tips or mature leaves. In Peru some control was obtained by removing the diseased plants from the beds. Applying a copper spray with sticker at regular intervals was suggested as a possible control.

*Phytophthora parasitica* Dast.—"Maculis amphigenis, orbicularibus, centro arescentibus, dilute umbrinus, annulatis, solitariis vel confluentibus; mycelio internatriciali, ex hyphis inter- et intracellularibus, primo continuis, tandem septatis, 3-9μ crassis, constantibus; haustoriiis sparsis, digitatis vel sub-globosis, raro ramosis, sporangiphoris 100-300μ longcis, sporangis terminalibus, interdum intercalaribus vel lateralibus, plerumque ovoides, subinde globosis 25-50 × 20-40μ; zoosporis 8-12 × 5-8μ; sporis perdurantibus globosis, flavidis, 20-60μ, membrana crassa, levi; oogoniis et antheridis in vitro cultis; oogoniis intercalaribus vel lateralibus, globosis, levibus vel rugosis, melleis, 18-25μ (23.8μ), pedicellis per antheridium penetrantibus; oosporis globosis, 15-20μ (18.6μ), membrana crassa, mellea, levi." (272, p. 168.)

(See Phytophthora parasitica, p. 33.)

A species of *Phytophthora* is said to cause severe damage in some instances as a postemergence damping-off organism in Guatemala. The diseased plants are sprayed with bordeaux or Yellow Cuprocide for control.

In 1945 Pinkus (206) reported that a species of *Phytophthora* has been identified by A. S. Müller as the cause of a disease affecting grafted cinchona nursery stock in Guatemala. The loss from this disease was said to be 3.75 percent in 1943, and the disease was of comparable prevalence in 1944. Small coffee-colored spots occur on the leaves, and the infection spreads downward through the midrib to the main stem, eventually killing the grafts. Nurseries exposed to the morning sun and on good soil sustain less damage than those less favorably situated. Marked variations in susceptibility of individual clones have been noted. Control has been effected by removing and burning diseased foliage bi-weekly during the rainy season and weekly during the dry season.

Reinking 9 described a dieback and canker disease affecting *C. ledgeriana* and *C. calisaya* nursery stock in Guatemala. The young growing tips of affected plants wilt and darken, and the disease may move down the shoots and eventually kill the seedlings.

---

9 See footnote 5, p. 4.
Frequently, the graft region is infected. Often when plants are attacked at the bases of leaf petioles, sunken stem cankers are produced that may girdle and kill the upper parts of the plants. Reinking stated that Dr. Albert Müller isolated a species of Phytophthora from diseased plants and attributes the disease to this fungus. Control is attained by good sanitary and cultural practices, pruning diseased parts well below point of infection and spraying the most severely attacked plants with bordeaux mixture, burning diseased plants and prunings, watering nursery by flooding rather than spraying, and spacing plants in nursery to allow good aeration.

(See phytophthora cankers, p. 32, and phytophthora root rots, p. 40.)

**STEM CANKERS**

Olpidiaceae

In 1910 Commelin (43) described a disease that affected the collar region of Cinchona succirubra seedlings in Java. Microscopic examination of sections through the affected region showed the presence of a fungus belonging to the Olpidiaceae of the Chytridiales (43). The affected plants were small, having a stem diameter of about 2 mm., and were growing at a high elevation (43). The bark had dried at the soil line for a distance of 1 to 2 or more centimeters, but the roots remained in a healthy condition (43). In the early stages of attack, i.e., before the stem was completely girdled, the leaves were green (43). In the later stages of the disease, however, the leaves faded and withered and the plant tissues turned brown (43). Corky tissues occurred occasionally in the diseased bark (43). Adventitious roots frequently formed on the stem above the diseased parts (43). The sporangia of the Chytrid measured about 0.02 mm. in diameter and were found in various stages of development in the cells of the bark and woody tissues (43). Blücher (13) suggested that the fungus probably gained entrance through hoe wounds. Leersum (170) stated that experiments in controlling the disease by cutting out the diseased spots and treating the wounds with carboleineum, slaked lime, or chloride of lime produced no results in the majority of cases. The same or a similar Chytrid was found in the dead areas of the outer bark of cinchona trees affected with "trunk canker" (43, 227).

**Physiological**

Owen (198) reported a physiological canker affecting the collar region of small cinchona plants in nursery beds in India. The disease was not found generally in the seedbeds but occurred in nursery beds situated above the mist line in damp locations. He suggested placing the beds at lower elevations and putting in a better drainage system.

**Undetermined**

In 1904 Zimmermann (289) reported very severe damage to Cinchona succirubra and C. ledgeriana hybrid seedlings at Amani,
German East Africa (Tanganyika since 1919). The leaves of the affected plants wilted and became discolored, and the stems usually turned brown near the root collar, while the root systems remained in a healthy condition (289). Sometimes only the upper part of the stems turned brown and then only the higher leaves withered and became discolored (289). Occasionally the leaves alone showed brown discolorations, principally along the midrib (289). A fungus mycelium having characteristically lobed haustoria was observed in microscopic sections of diseased leaf and stem material; however, fructifications of this fungus were not observed (289). The disease diminished considerably with the advent of dry weather, and transplanting the seedlings to the plantation brought the disease to a standstill (289). Control measures were unnecessary at the time (289). Zimmermann (289) isolated a number of fungi that developed on parts of the diseased stems when left in damp soil for a time and from these he described the following new species: Calosphaeria cinchonae Zimm., Nectria amanitana Zimm., N. cinchonae Zimm., and Pestalozzia cinchonae Zimm. Nectria coffeicola Zimm. was also isolated from the diseased stems (289). Inoculations with these fungi failed to produce the disease (192, 289). In 1906 Koorders (154) stated that, of the fungi described by Zimmermann, Calosphaeria cinchonae, Nectria cinchonae, and Pestalozzia cinchonae had not been reported in Java.

In the report of the Government cinchona plantation at Tjinjireoean, Java, for 1917 (56, 104), a disease of unknown cause, which is said to correspond in every way with the collar disease described by Zimmermann, occurred in several seedbeds where a great deal of sprinkling had been done. The disease noticeably appeared in spots where the young plants stood very close together (56).

ROOT ROTs

Rhizoctonia

In 1940 a root rot was reported on Cinchona ledgeriana plants in the nurseries at Mayaguez, Castañer, El Semil, and Las Mesas, P. R., and also to some extent on some plants at Maricao (216). The fungus responsible for the disease has been identified as a species of Rhizoctonia (216).

Heubel (127) reported a root collar disease in the seedbeds in South Sumatra caused by Rhizoctonia, the fungus also causing the "mopo" disease.

(See rhizoctonia damping-off, p. 5.)

Sclerotium

Sclerotium rolfsii Sacc. has been reported by Rhind (235) on cinchona seedlings in the nursery of the Government cinchona plantation in the Tenasserim District, Burma, and by Crandall 10

on Ledgeriana transplants at the agricultural experiment station, Tingo Maria, Peru. Crandall observed the disease in isolated patches in the nursery following the rainy season, where it caused a root and collar rot. He isolated the fungus from diseased collar tissue and found the sclerotia of the fungus in the soil within which diseased plants were growing.\textsuperscript{11} Eradication of diseased and adjacent healthy plants and the removal of a part of the soil in the bed are said to have checked the spread of the disease in Peru.\textsuperscript{11} The extent of damage was not serious in Burma (235). Weber (281) listed Cinchona sp. as one of the hosts of \textit{S. rolfsii}.

\textit{Sclerotium rolfsii} Sacc.—“Superfaciale, subrotundum v. horizontaliter ellipsodeum, 0.5–0.8 mm. diam., facile secedens, levigatum, nitrillum, roseum, demum fulvescenti-brunneum, carnoso-firmulum, intus pallidum; cellulis e globoso polyedris 6–8μ diam. interdum sinuosis, subhyalinis, ad peripheriam sclerotii bruneolis; fructificatione nulla observata.” (243, p. 257.)

\textbf{Sporodesmium}

In 1906 and 1907 Koorders (154, 155) reported a new species, \textit{Sporodesmium cinchonae} Koord., as a wound parasite on 1- to 2-year-old cinchona hybrid seedlings in the Kedoe Residency in Middle Java. Most of the seedlings had been rather severely damaged by the nematode \textit{Heterodera marioni} (Cornu) Goodey; but nematode-free seedlings, which were from a different plantation and were suffering from a root disease, were found to be infected with the same species of \textit{Sporodesmium} (155). Koorders (154) also isolated \textit{Sporodesmium} from diseased plants sent from the Government cinchona plantation in the Preanger; therefore, he considered the nursery bed root disease in the Preanger to be identical with the disease in the Kedoe.

Leersum (162), who had reported the disease in the Preanger in 1891, stated that \textit{Cinchona ledgeriana} seedlings quickly succumbed to the disease, while \textit{C. succirubra} plants were not so quickly killed by it. The bark of young plants cracked at the root collar (167). The tops of the plants appeared healthy for a while after the roots were found to be seriously infected; later the leaves turned red, and the plants withered and died (162). Leersum (162) considered the white mycelium that he found between the root bark and wood to be characteristic of the disease. The wood of the infected roots was found to be brown and the bark rotten (162). In 1903 there was considerable disease in nurseries on soil rather impermeable to water; 50,000 plants were lost in old nursery beds at Kawah–Tjiwidei, while the newly laid-out nursery beds remained free of the disease (2, 154). Using new nursery beds composed of rich humus, building up the beds for drainage, and transplanting to field positions when the plants are 2 years old were measures suggested for control (154).

\textit{Sporodesmium cinchonae} Koord.—“Parasiticum, conidiis acrogenis subsolitariis, in radicis cellulis inordinate dispositis, primo juventute hyalinis vel
pallide olivaceis v. fuscis, continuis, dein aterrimis, oblongis v. irregularibus, plerumque 30-45 × 14-21μ usque ad 50-65 × 15-18μ, multicellularibus.” (155, pp. 234–235; also described in 227 and in 242, v. 22, p. 1402.)

Stigeosporium

In March 1941 Marañon and Bartlett (183) reported on a canker disease of cinchona in the Philippine Islands from information and data given in an unpublished manuscript by Servando S. Madarang. All of the cinchona species grown at Impalutao and Kaatoan, namely, Cinchona ledgeriana, C. succirubra, C. officinalis, and C. hybrida, were infected. The bark canker is said to have appeared in such epidemic proportions between 1929 and 1934 as to threaten to destroy the plantation at Impalutao. From July to December 1939, 22.4 percent of the 1,916,639 plants raised in the Kaatoan nursery were eradicated because they showed the canker symptoms. In the first 10 months of 1940, 62 percent of the 1,067,592 nursery plants were discarded from the beds and of this group 94 percent were cankered. The disease occurs as a leaf and stem blight, root necrosis, and a canker of seedlings and nursery plants. "The root necrosis is said to be the initial stage of infection, which later manifests itself as seedling stem and leaf blight and finally as tree canker.” (183, p. 125.) Illustrations of diseased older trees show the canker near the soil line.

The causal organism of this disease complex is said to be an undescribed soil-borne fungus and was to have been published as Stigeosporium cinchonae Roldan n. sp. Infected plants up to 3 years are grubbed out and destroyed. Madarang advised leaving older trees, if not too badly infected, until at least 6 years of age, when they can be harvested. After diseased branches are pruned, the wounds are painted with coal tar. Resistant trees are to be used in future breeding and selection work.

NEMATODE ROOT INFECTIONS

Heterodera

Plant-parasitic nematodes (eelworms, roundworms, or threadworms) are minute organisms of complex structure. The most widely known species, Heterodera marioni (Cornu) Goodey, the root knot nematode, has been reported on seedlings of Cinchona spp. in southern India (8, 37, 133, 156, 191) and cinchona hybrids in Java (12, 100, 154, 155, 184, 230).

Koorders (154) found large numbers of this nematode in the roots of cinchona seedlings that were diseased and stunted in growth. Galls were not numerous and only 0.5 to 1 mm. in size at the most (154). Swollen females were frequently found in the bark of roots that showed no external evidence of their presence (154). They were present in both the dead rotten bark and the living bark of the cinchona roots (154). Koorders (154) isolated a species of the fungus Sporodesmium from the roots of most of the infected plants as well as from the roots of diseased plants that were not infected by nematodes, and he was not able to state conclusively which of the organisms involved had caused the
injury to the seedlings. Rant (226) reported in 1913 that Cinchona ledgeriana plants had been successfully inoculated with the nematode Heterodera marioni. The extent of damage done to cinchona in both India and Java is said to be unimportant (191, 230).

Dorylaimus

In Java Koorders (154) observed another species of nematode in the rotten bark of a cinchona seedling infected with Heterodera marioni and the fungus Sporodesmium. He tentatively identified the nematode as Dorylaimus sp.

Tylenchorhynchus

Tylenchus alatus Cobb was described originally from root specimens, said to have been Cinchona sp., from Africa (42). Goodey (87) stated that it was a root of C. succirubra fav. from Belgian Congo. Tylenchus alatus Cobb was placed by Filipjev into the genus Tylenchorhynchus, and therefore it is called Tylenchorhynchus alatus (Cobb) Filipjev (79).

Tylenchus

In 1941 Fluiter and Mulholland (80) reported Cinchona succirubra as a host of the nematode Tylenchus coffeae Zimm. in Java.

PLANTATION DISEASES

LEAF SPOTS

Cephaleuros and other algae

An algal leaf spot, caused by Cephaleuros virescens O. Kunze, has been reported by Hendrickx (122) on Cinchona spp. in the Mulungu region, Belgian Congo; by Reinking 12 on cinchona in Guatemala; and by Crandall and Davis 13 on C. succirubra in Guatemala, on C. pubescens in Colombia, and on the Ledgeriana form of C. officinalis in Guatemala and Peru. The circular and raised spots are light reddish brown with some light-green coloring and occur mostly on the upper leaf surfaces. 12 In Guatemala the disease is usually found at higher altitudes where the humidity is high. 12 The algal leaf spot in the Mulungu region was not serious enough to warrant a program of control (122), nor was it considered to be of economic importance in the Americas. 12 13

Cephaleuros virescens O. Kunze.—Thallus a roundish disk, more or less notched at the edge, generally uninterruptedly complete, red yellow or red brown, up to 1 cm. broad, 1- to many-layered, subcuticular or subepidermal, composed of radial dichotomously branched, radiate, closely connected filaments attached to the leaf by a moderate number of short, 1- to 3-celled rhizoids; cells brown-walled, polygonal-rounded, varying between 4μ to 12μ in diameter, 1 to 3 times longer; hairs absent in young thalli, sterile and fertile hairs abundant on the older ones and forming a furry covering that

12 See footnote 5, p. 4.
penetrates the cuticle; sterile hairs simple, stiff, consisting of several cells, the apical cell pointed; fertile filaments branched, capitately swollen, bearing 4 to 8 lateral or terminal clustered sporangia with 1 oval or ellipsoidal sporangium mother cell, $25\mu$ to $30\mu \times 38\mu$ to $40\mu$, appearing particularly during the rainy period; zoospores biciliate, elongated ovoid, $5\mu$ to $8\mu$ long; globular sporangia occurring in the thallus, chiefly near the edge or terminal on the creeping filaments, borne on the same thallus as the cluster sporangia, oval-ellipsoid, of the same size as the mother cell of the cluster sporangia. The thalli can live and grow on the leaves for years (219).

Martin and Crandall (186) reported a green algal leaf spot on Ledgeriana cinchona trees in Peru.

Hunger (132) reported that specimens of C. ledgeriana leaves, which were preserved in the station museum at Malang, Java, showed injury caused by an alga belonging to the Chroolepidaceae.

In 1944 Martin and Crandall (186) observed a brown algal leaf spot on a cinchona tree in Peru.

Cercospora

In 1887 Ellis and Everhart (74) described a new species of Cercospora collected on cultivated cinchona in the United States of America. Hansford (113, 115, 116) also reported C. cinchonae on cinchona in Uganda. This species of Cercospora was listed by Lieneman (173).

*Cercospora cinchonae* Ell. and Ev.—“Spots amphigenous, definite, nearly black above, with a narrow, slightly raised margin, brownish-black below, 2-3 millim. in diam.; hyphae epiphyllous in small, scattered, sphaeriform tufts, very short; conidia cylindrical, yellowish-hyaline, nearly straight, becoming faintly 3-septate, 25-35 $\times 21\frac{1}{2}\mu$.” (74, p. 17; also described in 227 and in 242, v. 10, p. 645.)

Gloeosporium

An undetermined species of Gloeosporium was observed by Koorders (154) in 1905 on mature leaves of a 2-year-old cinchona hybrid in Middle Java. The characteristics of the fungus are said to correspond to some extent with the genus *Zythia* Fr. (*Phomopsis* Sacc.).

Gloeosporium sp.—Sori round or irregular; spots large, straw-colored, on both the upper and under sides of mature leaves, about $120\mu$ in diameter; spores oblong, straight or slightly bent, 1-celled, hyaline, without appendages, $8\mu$ to $10\mu \times 3\frac{1}{4}\mu$ (154; also described in 227).

An undetermined species of Gloeosporium was isolated from brown circular spots on cinchona leaves in the Darjeeling District of Bengal, India (84, 136, 199).

(See gloeosporium leaf spot, p. 9.)

Phyllosticta

In 1892 Patouillard and Lagerheim (201) described a fungus causing a leaf spot on *Cinchona* sp., to which fungus the name *Phyllosticta cinchonae* was assigned. It was collected at San Nicolas during a trip to the coastal and mountainous regions of Ecuador (201). The fungus causes angular, dull-yellow spots on the living leaves (259). *P. cinchonae* has been reported to cause brown leaf spots on the leaves of *C. succirubra* in the Transcaucasian region of Russia (U.S.S.R.) (210).
Phyllosticta cinchonae Pat.—"P. maculis exaridis, sparsis vel confluentibus, 4–8 millim. latis, quadrangularibus aut irregulariter 5–6-gonis, ochraceis, aestigi brunneo-marginatis; perithecis epiphyllis, 80–100 μ latis, brunneis; sporulis hyalinis, ovoidis, biguttulatis, 7 × 2 μ." (201, p. 135; also described in 242, v. 11, p. 473.)

Koorders (155) also described a species of Phyllosticta under the name P. cinchonae. His material was obtained from the leaves of young high-percentage cinchona hybrids in Middle Java at an altitude of 1,600 meters (155). The irregular leaf spots were straw-colored, not bordered, often 0.5 cm. in size, appearing both on the upper and lower surfaces of the leaves (155). The fungus was said to cause little damage at the time and had attacked only a few plants (155). Because of the confusion with Patouillard's fungus, Rant (227) renamed the species P. cinchonicola. Later, Stevenson (259) stated that the name Phyllosticta cinchonae Koord. is untenable and that P. cinchonae Pat. should have priority.

Phyllosticta cinchonicola Rant (P. cinchonae Koord. non Pat.).—Pyenidia scattered, straw-colored, thin-walled, flattened globose or ovoid, about 120 μ in diameter; conidia cylindrical, hyaline, 1-celled, oblong, straight or nearly straight, rounded at both ends, 8 μ to 10 μ × 3.25 μ, generally 10 μ long, not frequently containing a few large drops of oil, sometimes broader at the tip than the base (227; also described in 155 and in 242, v. 22, p. 346.)

(See Phyllostictina, p. 10, and Phyllosticta spp., p. 11.)

Prillieuxina

Stevenson (260) reported a species of Prillieuxina occurring on living leaves of Cinchona pubescens Vahl in Costa Rica. The fungus caused definite leaf spots, and infected leaves turned red and fell prematurely. The diseased tree was standing in a forest near a commercial planting of cinchona.

Prillieuxina cinchonae Stevenson.—"Hypophylla, plagulas 1 cm. latas formans; mycelio anastomosanti 3–3.5 μ crasso; hyphopodiis nullis; thyriothecis hemisphaerico-lobatibus, 150–300 μ diam.; ascis subglobosis usque ovatis, apaphysatis, 30–45 × 25–35 μ, octosporis; sporis medio 1-septatis et constrictis, fulvis, 24–27 × 12–15 μ." (260, p. 632.)

Sclerotinia

In 1904 Lutz (176) reported a leaf disease caused by Sclerotinia fuckeliana D By. on cinchona plants cultivated in the greenhouses of the Paris School of Pharmacy, France. Small, semitransparent spots appeared first on the leaves, and these spots turned brown in 1 or 2 days (176). The causal organism continued to spread from the original spots in concentric zones until the entire leaf blade was covered in 5 or 6 days (176). The leaves withered, shriveled, and finally fell from the plants (176). A light-gray mycelial growth appeared on the leaf spots on either the upper or lower sides of the leaves, which, when isolated and cultured, produced the conidia of S. fuckeliana (176). Healthy plants that were inoculated with the disease organism showed the leaf spots after 2 days and necrosis followed in 24 hours (176). Control consisted of the removal of seriously affected leaves, repeated spray-
ings with neutral bordeaux mixture, and repotting the plants (176).

Rant (227) stated in his 1914 publication that the disease had not been reported from Java. While investigating the mopog disease of cinchona in Java, Rant (229) inoculated leaves of 4- to 6-inch cinchona hybrid seedlings with Botrytis cinerea and obtained leaf spots similar to the spots caused by S. fuckeliana as described by Lutz. There was also abundant spore production (229). Rant's further tests, however, indicated that B. cinerea was able to produce the leaf symptoms described by Lutz only at the points of inoculation (229).

_Sclerotinia fuckeliana_ D By.—“Stipitata, minuta, patellaris, ½–3 mm. lata, 5–10 mm. alta, flavo-brunnea ex _Sclerotio echinato_ Fuck. nascens; ascis teretico-clavatis, 130 × 12–18; sporidiis monostichis, ovoideis, hyalinis, 10–11 × 6–7.” (242, v. 8, p. 196.)

**Uredo**

In 1902 Hennings (125) described a new species of _Uredo_ collected by Zimmermann on _Cinchona_ sp. growing in an experimental garden in Java. The fungus formed brown-rust-colored pustules on the leaves (259).

_Uredo cinchonae_ P. Henn.—“Maculis fuscidulis vel obsoletis, soris epiphyllis, sparsis, minutissimis, pulvinatis, ditius tectis. brunnealis, ca. 0.3 mm. diametro; sporis ovoideis vel ellipsoides, intus flavidis, 13–18 × 10–15 μ, episporio hyalino, granulato-verrucoso.” (125, p. 140; also described in 227 and in 242, v. 17, p. 493.)

In 1924 P. and H. Sydow (266) stated that Hennings’ species is not valid, because examination of the original material showed no fungus formation in the light-brown emarginate spots and the “spores” described by Hennings seemed to be the cells of the matrix of the host leaves.

**Undetermined**

Reinking 14 has reported a leaf spot of undetermined cause affecting _Cinchona ledgeriana_, especially the narrow-leaved strains, and to a less extent _C. succirubra, C. calisaya_, and hybrids on certain plantations in Guatemala. The disease is characterized in the earliest stage by minute water-soaked spots, which later have dark red-brown borders and sunken centers that turn ash gray eventually. The spotting begins at the leaf tip and edge and moves inward. Severely attacked young leaves are wrinkled, and the margins are curled under. Defoliation may occur, although apparently normal trees may have severe spotting without defoliation. The disease was observed on Ledgeriana seedlings in seedbeds and on grafted Ledgeriana plants grown in plots with and without coffee plant shade. Specimens examined for a causal organism failed to show a fungus or bacterium, and it is suggested that the disease might be of nutritional origin.

14 See footnote 5, p. 4.
OTHER LEAF DISEASES

Capnodium and other molds

Sooty mold, which is found on a number of tropical and subtropical plants, has been reported on the leaves and stems of *Cinchona ledgeriana* in Java (167, 220, 227) and on *Cinchona* spp. in Indochina (24), Uganda (250), and Guatemala. The characteristic black sooty covering on the affected parts is made up of the hyphae of fungi that live on the “honeydew” excreted by scale and other insects (24, 220). Although a large number of the fungi causing sooty mold belong mainly to the Perisporiaceae of the Ascomycetes (19), *Capnodium brasiiliensis* Putt. was the organism reported from Indochina (24) and Uganda (250). Heavy growth of the mold may prevent the proper functioning of the covered leaf, but damage to the host plant is caused primarily by the feeding of the insects (227) and is comparatively unimportant on cinchona (24, 220). Control consists of the destruction of the insects (24, 167).

(See hормисциум sooty mold, p. 27.)

Mineral deficiencies

Two distinct types of chlorophyll deficiency are reported on some cinchona trees at Maricao and Las Mesas, P. R. (117). One type is said to be caused by iron deficiency. In the other type the leaves develop asymmetrically, causing the midrib to be curved. A light-pink color predominates over the natural green in some parts of these leaves, and the leaves vary widely in shape and size. The causes of these disturbances were not determined.

Reinking reported a leaf mottle or chlorosis on cinchona in Guatemala, believed to be a nutritional trouble. The yellow-green mottling usually occurred between the main lateral veins.

Harper (117) in 1948 reported an abnormality of the leaves of some cinchona trees at Maricao and Las Mesas, P. R., probably caused by nutrient deficiencies or by the mineral toxicity of the soil. The young leaves stop growing at the margins, with the result that mature leaves have an inverted-cup shape and are wrinkled, with dark-red mottling. Leaves of the fruiting branches are smaller, thicker, and flatter than other leaves. Generally, sterile flowers develop with only miniature corollas.

Mosaic

In 1909 a mosaic disease was reported to occur occasionally on otherwise normal *Cinchona ledgeriana* plants in the Java plantations (167). One entirely variegated plant of *C. succirubra* was found. It was stated that this symptom is not to be confused with variegation of the edges, which is probably an indication of weakness.

In 1931 Petri (204) reported that the leaves of *Cinchona* sp. at Faghenia, Eritrea, were affected with mosaic.

15 See footnote 5, p. 4.
Pellicularia and other thread blights

Davis and Crandall reported a thread blight caused by *Corticium koleroga* (Cke.) Hoehn. (= *Pellicularia koleroga* Cke.) on 10-year-old trees of *Ledgeriana* stock in Guatemala. The mycelial threads of the fungus extended as a web over the trunk, branches, and leaves, and the dead leaves hung in clusters, suspended from the branches by the fungal threads.

Horse hair blight (*Marasmius* sp.) was observed by Davis and Crandall on a few wild trees of *Cinchona barbacoensis* Karst. in Colombia. The fungus strands were found on trunks, branches, and leaves.

Hunger reported a “cobweb” disease on the twigs and leaves of *C. ledgeriana* in the Soekaboemi area in western Java. White fungus threads crept along the branches and forked at the side branches, twigs, and leaves. The threads grew over the leaf petioles and split into a many-branched system of fine threads on the leaf blade to form the “cobweb”. The leaves died and became detached from the twig but hung from it by the fungus thread. The disease is said to occur quite infrequently in the cinchona plantations in Java, and cutting and burning the affected branches is recommended as a means of control.

Rant reported another thread blight that he found on *C. succirubra* and on a young cinchona hybrid planted in a damp shady spot. The mycelial threads were honey yellow to brown and crept along the trunk for more than 120 cm. The threads were 1 to 1.5 mm. wide and did no damage to the tissues of the plant until the leaf blades were reached. The leaf blades died finally and hung from the twigs by the fungus filaments. Rant found a white to brownish powdery fruiting stage on the under surface of some of the leaves, but he did not observe a “cobweb” formation.

Scab

Jenkins reported a scab caused by a species of *Elsinoë* affecting the leaves, stems, and fruits of cinchona in South America. The disease occurs on *Cinchona pubescens* Vahl and rarely on *C. officinalis* L. in Colombia and Peru and rarely on *C. delesser-tiana* Standley in Peru. The fungus causes spots on the upper or lower surfaces of the leaves, and occasionally the spots fall out, leaving perforations or tissue network. Raised cankers are formed on the young stems and rachis and branches of the inflorescence. Diseased fruit capsules are frequently curved or otherwise bent and distorted and may also be dwarfed. Lesions on the capsules are often numerous and usually more conspicuous than on leaves and stems. The possible economic importance is not known as yet; but it is suggested that, since the leaves and young stems

---

are affected, it might prove destructive under favorable conditions in the nursery. Jenkins described the causal fungus under the name *Elsinoë cinchonae* Jenkins.

_Elsinoë cinchonae* Jenkins.—“Maculae plerumque numerosissimae, conspersae, circulares, subcirculares, usque ellipticae, interdum elongatae, elevatae, centro saepe plus minusve apiculiformi, interdum aggregatae vel confluentes, in foliis amphigenae, interdum nervisequentibus, usque 1.5 mm, rare 2 mm, superne conspicuiores et saepe cinnamonea-griseae, margine nigro-vinaceo-brunneo circumdatae, inferne vinaceo-brunneoae; cancri in caulibus generaliter elliptici, usque 4 × 5 mm, avellanei, vel discolors, in capsulis usque 8 mm diameter, avellanei vel discolors; ascomata plus minusve numerosa, in maculis foliorum epigena conspicuoria, rotunda usque elliptica, pulvinata, exposita, usque 300μ diameter, 75μ crassa, superficialiter nigro-brunnea; epithecium fuscum, 10μ crassum; asci numerosi, sub-epithecio in regione stromatica hyaline distributi, globosi usque ellipsoidei, apice incras- sati, 18–20μ diameter; ascosporae immaturae, 1–3-septatae, hyalinae, 15 × 15μ; status conidiophorus (*Sphaceloma*) in maculis foliorum epigena promi- nens; conidiophora in palum compactum, expositum superficialiter nigro- brunneum, plus minusve continuum, ex stromate hyaline oriundum, fructifica- tione tota 30μ crassa, vel marginem maculae versus usque 50μ; conidiophora cylindrica apice acuminato, generaliter continua vel unisepata, 3.5–5 × 5–15μ; conidia rare visa, brunnea, elliptica, 4–5 × 8–10μ.” (188, pp. 348, 550.)

Undetermined

Owen (198) reported a disease on cinchona leaves in Ballangen- godde, India, which is controlled by an application of lime and sulfur.

In 1909 an abnormal leaf condition of otherwise healthy cin- chona trees was reported in Java (167). Corky growths were formed in spots on the young leaves; and as these spots were un- able to keep pace with the rapid growth of the surrounding healthy parts of the leaf blades, the tissues tore around the spots. Fre- quently the corky material dropped out, leaving holes in the blades.

In 1942 Kevorkian (151) reported a virus disease of cinchona in Puerto Rico.

**BARK DISEASES**

**Hormiscium**

In 1888 Fürth (82) and Gorkom (95) reported the occurrence of a black sooty fungus on the trunks and branches of cinchona, some of which were *Cinchona succirubra*, in a few plantations in Java. The spots were jet black and soft in the earlier stages, drying out and becoming firmly attached to the bark later (82), and thereby making the bark unfit for pharmaceutical use (95). Gorkom (95) stated that the fungus belonged to the genus *Hormiscium*, to which he gave the specific name *pannosum* without publishing a description.

The fungus multiplies by conidia and spreads very rapidly (95). It penetrates the outer dead “cork cells” but does not enter the living tissues; however, Gorkom believed that the thick fungus growth interfered with the normal functions of the bark, in addition to making the bark unfit for use (95). Very crowded nurser- ies, high humidity, and shade were thought to be favorable to the growth of the fungus (95).
Rant (227) stated that he observed a species of *Hormiscium*, which he thought was probably *H. pannosum*, on living, sound trunks of old *C. succirubra* trees at the Government cinchona plantation in Java. He described the fungus as a saprophyte, forming rather thick, black, irregular coverings, which varied in thickness and which bore dark olive-green conidia having a diameter of 6μ to 17.5μ and underdeveloped conidiophores (227). Gorkom (95) recommended removing the fungus growth by mechanical means and burning and washing the infected spots with milk of lime or limewater. He advised against the use of copper sulfate or carbon bisulfide solutions on the spots, fearing that the poisonous component might affect the bark unfavorably (95).

(See capnodium sooty mold, p. 25.)

**Septobasidium**

The sporadic occurrence of species of *Septobasidium* in the cinchona plantations in the Netherlands Indies has been reported for a period of about 35 years (131, 160, 167, and others). According to the classification by Couch (46) there are three species of *Septobasidium* that have been reported on cinchona, namely *S. bogoriense* Pat., *S. cinchonae* Rac., and *S. lichenicolum* (Berk. and Br.) Petch. *S. bogoriense*, the gray dadap fungus, has been reported on *Cinchona* spp. in Java and Sumatra (11, 16, 17, 46, 66, 67, 85, 86, 88, 121, 157, 236, 252-254); *S. cinchonae*, on young branches of *Cinchona* sp. in Java (16, 46, 218, 227, 253); and *S. lichenicolum*, on *Cinchona* sp. in Java (17, 46). Attacks of *S. bogoriense* are said to be especially heavy on young cinchona grafts and seedlings after heavy blooming (67). *S. cinchonae* has been found on cinchona at elevations of 2,300 to 2,700 meters above sea level and occurs more frequently in young nurseries where the plants are low and produce leaves close to the ground than in older plantations where the trees are widely spaced and the circulation of the air takes place more freely (16). While the *Septobasidium* species on cinchona are generally considered to be epiphytic, they are capable of causing indirect damage to the host plants by providing a damp atmosphere and shelter for other fungus organisms, such as the disease-producing *Corticum salmonicolor* Berk. and Br., and by harboring the scale insects associated with *Septobasidium*, which live at the expense of the host plant (11, 211, 253). Although the plants recover from attack, the death of a large number of branches in the plantation represents a considerable crop loss at the time of harvesting (11, 236). Steinmann (253) advised spraying for the control of scale insects, the insecticide used depending on the type of insect present.

**Tulasnella**

Raciborski (218) reported a species of *Tulasnella* as being common on young branches of *Cinchona ledgeriana*, *C. officinalis*, and *C. succirubra* in the Prêanger, Java. Although the fungus
hyphae grow only on the surface, the young bark underneath the mycelium is killed and later the tips of such affected branches die even though the fungus has not extended on to them (218). Stevenson (259) reported the fungus as occurring in thin layers on lower leaf surfaces. Leaves that are infected are promptly killed (218). *Tulasnella cinchonae* is said to spread in the plantations and to be injurious (218, 227).

*Tulasnella cinchonae* Rac.—“Effusa, aplanata, tenuissima, caules et paginam inf. foliorum obducens, pallide rosea; hyphis hyalinis septatis usque ad 60μ long., depressis sursum copiose ramulosis; basidiis in apice ramulorum nascentibus, anguste ovoideis, 20-24 × 8-11, apice tenuiter tenuiter lateraliter ad basidia quaternis globosis v. breviter ovoideis, hyalinis, tenuiter tunicatis, 7-9μ diam., non secedentibus.” (242, v. 21, p. 452; also described in 218.)

Rogers (238) examined the type specimens from Raciborski's herbarium and stated that he did not believe that the fungus described was a species of *Tulasnella*, being somewhat like a species of *Septobasidium*.

Undetermined

Martin and Crandall (186) reported a few cases of rough bark on Ledgeriana trees in Peru. The condition was said to be so rare at the time as not to be alarming.

Owens (198) reported that “hide-bound” trees are sometimes found in the cinchona plantations in India. The condition is not infrequently mistaken for canker. A vertical slit in the bark relieves the condition.

**SYSTEMIC DISEASES**

Undetermined

In 1939 Hendrickx (122) reported a tracheomycosis disease at Kalonge, Belgian Congo, on *Cinchona ledgeriana* and *C. succirubra*. Stoffels (262) stated that the disease is not limited to the wood, but also causes a bark necrosis. The trees succumb quickly (122). A species of *Fusarium* of the section "Elegans" and a species of *Verticillium* have been isolated and inoculations made (135).

A disease affecting *C. robusta* and occasionally *C. succirubra* has been reported in Java, in which large trees begin to die about a meter from the ground, with the necrosis continuing upward, while the lower part of the tree remains alive and can send out new shoots (167). About 30 trees were found to be affected by the disease in one locality. Inoculation tests with fungi isolated from the diseased trees were negative.

**TRUNK AND BRANCH CANKERS**

The word “canker” has been used so variously in connection with cinchona diseases that it is difficult to judge from the literature the actual number of different canker diseases involved. Among the earlier workers in the Netherlands Indies who wrote about the canker diseases of cinchona were Eekhout (73), Kessler (146), Warburg (279, 280), and Wurth (as reported by Hunger, 131). Since their discussions are rather general, however, and
do not contribute greatly toward an accurate understanding of the symptoms and causal organisms involved in the various canker diseases, they will not be reviewed at length here. A similar general treatment is found likewise in some of the early reports by the Government cinchona station in Java, where the terms “stem canker” and “stem rust” were used interchangeably to designate one disease (54–59), but which were later separately used for two diseases (60–62, 64, 65, 67, 70). Numerous other reports have been made of the occurrence of stem, trunk, or branch canker in Java and Sumatra (2, 88–93, 98, 100, 106–109, 112, 129, 131, 150, 154, 157–161, 167, 168, 170, 214, 227, 240, 252, 263). Zimmermann (290) stated that canker is the most common disease of cinchona. Some of the fungi observed on or believed to be connected with cinchona canker diseases are Botryodiplodia (161), Nectria (154, 214, 227), Peziza (279), and Tubercularia (227).

Corticium

Pink disease, called “djamoer oepas” in Java, is caused by Corticium salmonicolor Berk. and Br. (synonymous with C. javanicum Zimm.) (227). It occurs on more than 140 different species of plants in the tropical rain-forest regions of the world (249), including such economic plants as citrus species, coffee, tea, rubber, cacao, cinnamon, and green-manure plants (225). Butler (27) stated that C. salmonicolor probably has a wider range of recorded hosts, belonging to the most diverse families, than any other tropical fungus known. It has been reported on cinchona in India and Ceylon (29, 40, 41, 178, 187, 264), Burma (48, 285), Java and Sumatra (1, 2, 13, 14, 20, 21, 27, 52–70, 71, 80, 88–93, 100–112, 121, 127, 131, 139–145, 147, 150, 154, 157–162, 164, 165, 167–170, 203, 205, 207, 210, 221, 223, 225–227, 239, 240, 244, 249, 251, 252, 259, 262, 263, 271, 275, 282, 287), Belgian Congo (122, 134), Guatemala,17 Colombia,17 and Peru (186).17 Rant (225) stated that he has observed the djamoer oepas on Cinchona ledgeriana, C. robusta, C. sucrirubra, C. officinalis, C. pahudiana, and hybrids in Java.

Davis and Crandall17 reported that pink disease occurs on cinchona in both plantations and wild stands and is possibly one of the primary enemies of cinchona in the Western Hemisphere. Symptoms of the pink disease on cinchona are the characteristic pink or whitish crusts on the bark, ringed or cracked bark, and occasionally calluses on the affected branch or trunk (225). The leaves turn red and dry up, and they remain attached to the diseased twig if the twig is young and dies suddenly (225). Frequently brown rust spots, caused by the bleeding and drying of the sap that exudes from the diseased areas, are found on the affected branches (225). Zehntner (287) stated that the symptoms of callus formation, cracking, and peeling in the absence of the fruiting stages of pink disease are sometimes mistaken for the symptoms of other stem and branch cankers by the cinchona growers in Java. Davis and Crandall17 stated that when the attack is most severe,

the cankers girdle and kill the branches or trunks on which they occur. Rant (223) reported that by the time the fungus fruits on cinchona the attacked branch is almost or quite dead.

Rant (223, 225, 227) described four forms of the fungus on cinchona. In the first stage the mycelium, consisting of fine, shining white hyphae, grows over the bark and is known as the "cobweb mycelium" (225). The fungus is believed to exist saprophytically in this form. In the second stage the mycelium forms white nodules composed of dense coils of fine, colorless hyphae (225). These nodules occur chiefly in the lenticels and connect with the cobweb mycelium on the bark (27). Rant (225) believed that the hyphae of these nodules enter the lenticels and infect the host tissues. The Corticium, or third, form is a fruiting stage (237). It appears as a light-pink or faded-white crust, often somewhat cracked, on the under surfaces of branches and twigs and occasionally on the leaves (225). Usually the bark underneath these crusts is killed (225). Necator decretus Mass., which is said to be an imperfect form of Corticium salmonicolor, is also a fruiting stage, and its orange-red fruiting bodies occur usually on the upper surfaces of branches and twigs (225). These fruiting bodies are produced in the cracks of the bark or superficially on the bark (225). The Necator stage is usually found with the perfect stage or on the same cinchona plant on which the Corticium is fruiting elsewhere (225).

Rant (225) states that the hyphae of the fungus penetrate the bark and wood of affected cinchona plants, and in the tender succulent twigs of Cinchona robusta he found the pith to be infected in advance of the surrounding wood. Rant's inoculations proved that the disease organism can pass readily from one host to another, cinchona having been successfully infected from tea, coffee, hevea, loquat, and others (225). There is no evidence of biological races (27, 225).

Inoculations made by Rant (225) show that Corticium salmonicolor is able to infect the sound bark of Cinchona ledgeriana twigs when the atmospheric humidity is high. The moisture conditions favoring the attack of the fungus depend not only on the weather but also upon the density of the planting and the habit of the plant. C. ledgeriana makes a rather dense, bushy growth and suffers severely from pink disease, while C. succirubra makes a more open growth through which the air can circulate freely and is rarely injured (27). The disease attacks are said to diminish during the dry season, but they increase soon after the rains begin (157).

The extent of injury to cinchona varies: Young plants are frequently killed by the disease; but old trees are seldom killed by it, although the branches are badly stunted (100). In 1905 all the plants in low-lying nurseries on one Government plantation in Java were attacked and had to be replaced by grafts (2). Rant stated that the use of fungicides alone will not control the disease (223); the usual method recommended in Java is to cut and burn the affected branches during dry weather (223, 287).
Corticium salmonicolor Berk. and Br.—“Fructifications broadly effused, thin, adnate, membranaceous-soft, separable when moist, pale ochraceous buff to orange-pink when fresh, fading in the herbarium to pale olive-buff and cartrige-buff, pulvulent, even, cracking a little in drying, the margin thinning out; in section 100-200μ thick, composed of hyphae running longitudinally over the substratum and bearing a broad layer of suberect, branching, loosely interwoven hyphae 4-7μ in diameter, not incrusted, not nodose-septate; no gloeostigidia; basidiospores hyaline, even, 9-12 × 6-8μ. The conidia of the imperfect Necator stage are catenate, 14-18 × 7-8μ, according to Massee.” (26, pp. 227-228; also in 242, v. 6, p. 620.)

Dasyscypha

Hennings (123, 124) reported that Dasyscypha warburgiana P. Henn. causes cankerous overgrowths on the branches of Cinchona ledgeriana in the Preanger, Java. He stated that according to Warburg's observations this fungus is probably the cause of “a disease” that occurs in the cinchona plantations in Java and has killed numerous trees for years (124). Engler and Prantl (75) and Stevenson (259) stated that it causes a serious canker disease in cinchona plantations.

Dasyscypha warburgiana P. Henn.—“Ascomatibus sparsis, brevissime stipitatis vel sessilibus, hemisphaerico-cupuliformibus, extus flavo-villoso, disco aurantio-flavo, margine integro, inflexo, ⅔-1 mm. diametro; ascis fusiformibus vel cylindraceo subcalvatis carnescentibus dein hyalinis, octosporis 50 × 70 und 7-9μ; sporidios elongato-ellipsoides vel subfusoides, hyalino-subcarnescentibus 5-10 × 3-4μ; paraphysibus filiformibus.” (123, p. 226; also described in 75, 227, and in 242, v. 11, p. 413.)

Phytophthora

Although “stripe canker” of cinchona has been reported from Java and Sumatra only within recent years (44, 45, 88, 90, 93, 147-150, 258), it has probably been prevalent there for many years. In 1886 Eekhout (73) described cinchona canker as being easily recognized by the vertical furrows in the bark and the death of the wood underneath the furrows. Stripe canker is caused by Phytophthora palmivora Butl. and is characterized by the nearly parallel vertical depressions in the bark of the stem that precede a cankerous condition, the death of the tissues underneath the strips and extending into the root, rust-colored spots on the bark resulting from the bleeding and drying of the exuding sap, and leaf fall (150). For many years conspicuous rust-colored spots on the bark of diseased or dying cinchona trees were observed by the planters in the Netherlands Indies and the condition was reported as “stem rust” (60-62, 64-67, 70, 88-93, 106-109, 112, 126-129, 131, 148, 157-161, 227, 252).

Since diseases other than stripe canker or injuries to the bark of the tree might cause bleeding and drying of the sap, it is not possible to state with certainty that all the reports of stem rust refer to the stripe canker; however, when rust spots are accompanied by dying of the tissues in strips, as reported by some authors (131, 166, 227), then it is probably the stripe canker disease. Goot (90) reported that in 1953 stripe canker was of common occurrence in the plantations of South and West Sumatra. It is said to be especially injurious to some grafts and in Cinchona
succirubra seedling nurseries (149). Keuchenius reported that the stripe canker disease is the most serious of the cinchona trunk diseases in Sumatra (147), especially in damp, unterraced plantations and where pruning and thinning of the groves have not been done early (148). Failure to practice crop rotation in the seedbeds and mature stands was observed on plantations in Sumatra reporting damage to C. ledgeriana (9). Pruning the diseased tissue down to the wood and applying 5-percent carbolinium in Socony grease was found to be an effective control (9). Stoffels (262) recommended using 15 percent “carbolinium plantarum” and Coster (44) advised the selection of resistant clones.

(See Phytophthora palmivora, p. 15.)

Crandall and Davis (46) and Martin and Crandall (186) have reported a stem canker and top wilt on Cinchona spp. in Central America and South America. The disease was observed on C. officinalis and C. pubescens in the plantations and on C. officinalis var. ledgeriana grafted on C. pubescens in the nurseries and plantations in Guatemala, on C. ledgeriana plantation stock in Peru, and on C. pubescens and C. pitayensis growing wild in forest stands in Colombia (49). The same disease symptoms were reported on seedling cinchona stock in Guatemala and Puerto Rico (49). Inoculations of 18-inch potted Ledgeriana seedlings with the causal organism, tentatively identified as Phytophthora parasitica Dast., resulted in girdling stem cankers, dieback, and leaf infections (49). Considerable evidence of host resistance of individuals and of certain clonal lines was observed wherever the disease occurred (49). In Guatemala control of the disease in the plantations was obtained apparently by cutting back the tops of infected grafted stock (49).

In 1942 Kevorkian (151) reported a stem canker of cinchona in Puerto Rico. C. pubescens is particularly susceptible, but C. officinalis, C. ledgeriana, and the hybrids C. pubescens × C. officinalis and C. officinalis × C. pubescens are also attacked. The disease is especially prevalent on young plants (151). Harper and Winters (118) stated later that symptoms of the canker disease in Puerto Rico were similar to those of the disease in Guatemala and Peru caused by Phytophthora parasitica.

(See Phytophthora parasitica, p. 16.)

Undetermined

King (153) and Owen (198) reported a canker disease in the cinchona plantations in India. Dark, shriveled, brittle patches were found on the stems and branches (153). The patches varied in size and were not numerous on one tree, often being confined to one branch (153). The tissue of the bark was destroyed, adhering firmly to the wood of the tree, and a canker was produced (198). When a large spot occurred on a small tree, involving the bark almost around the stem, death of the tree resulted (153). If a tree were cut back, healthy shoots would be produced (153). The disease was most prevalent during the rainy season and
usually was not fatal (153). Owen (198) stated that fungus fila-
ments were found in the decayed bark, occasionally penetrating
the living tissue; however, he did not consider the fungus to be
the cause of the disease. Pruning the diseased parts was recom-
mended (198).

In 1879 Hooker (130) reported that large Cinchona succirubra
trees in Jamaica were affected by a disease of the trunks in which
an area of a few inches to a few feet in the middle of the trunk
was said to be “contracted.” The leaves showed a slight dis-
coloration, and the bark and sapwood in the affected areas were
dead. All the parts above the ringed areas died eventually, but
the lower trunks and roots remained healthy. Suckers formed
when the trees were cut back. The number of trees attacked was
comparatively small. The mycelium of a fungus was believed to
be responsible.

In 1930 and 1931 a destructive canker disease that girdled
the branches or trunks of cinchona trees was reported from the
Philippine Islands by Brown (22, 23). Trees up to 7 years of
age were attacked and many were killed (22). Efforts to isolate
a causal organism failed (23). Eradication methods were recom-
mended for field control (23).

Martin and Crandall (186) reported a few cases of a canker
of unknown cause on cinchona trees in Peru. The numerous
cankers on both trunk and branches were about 1 inch in diameter
and closely resembled the cat’s-eye cankers caused by Nectria sp.
No fungus or fruiting bodies were observed.

In 1945 Reinking 18 reported a stem and crown canker affecting
Ledgeriana grafts on C. succirubra stock in Guatemala. The
grafts were dead at the point of union with the stock. Frequently
a species of Nectria was observed on the dead areas above the
graft, but it was not considered to be the cause of the disease.
The disease was not epidemic, apparently; and faulty grafting
methods and incompatibility of stock and scion were suggested as
contributing factors.

Mitra (187) and Rhind (235) have reported a diseased condi-
tion of young cinchona plants in the Government cinchona plan-
tation in Lower Burma. C. succirubra and a hybrid are affected,
and C. ledgeriana is especially injured (235). The collar of an
affected plant swells to about three times its normal diameter, and
the bark turns dark brown to black and has many small longitu-
dinal fissures (187). The lower leaves fall, and the leaves that
remain on the extremities of the branches are subnormal in size
(187). Badly affected plants usually die within a short time
(187). Most of the abnormal size in the swollen collar region is
caused by the thickening of the bark (187). The brown discolora-
tion extends into the wood (187).

Microscopic examination failed to reveal the presence of either
bacteria or fungi in any of the affected parts, and the disease
was thought to be physiological (187). The plantation is located

18 See footnote 5, p. 4.
on a low hill with the top exposed to the southwest monsoon, while the sides are sheltered (187). Eighteen percent of the plants died on one-fourth acre on top of the hill, but only 3 percent died on the whole field of 20 acres (187). Too low an elevation (300 to 500 feet), too high temperatures, insufficient drainage, and over-exposure during the monsoon season are thought to be the causes of the abnormal development and disease in the plantation (187). Although a causal organism was not found, saprophytic fungi are said to invade the tissues of dead plants (187). *Botryodiplodia theobromae* Pat. (= Diplodia theobromae (Pat.) Nowell) was isolated from a few plants (29, 187).

(See *Diplodia theobromae*, p. 12.)

In 1930 McRae (178) reported a "bleeding disease" affecting cinchona trees at the Mungpoo and Munsong plantations in Bengal, India. The disease is said to be worse in plantations between 2,500 and 3,500 feet in elevation (178). Narrow red streaks occur on the surface of the bark near the base of dying trees (178). The bark under these patches is brown and dead to a varying depth, sometimes to the cambium (178). Bleeding takes place on the trunk and occasionally on the branches, chiefly during the hot weather during April (178). The "bleeding fluid" was found to be soluble in acetic acid and alcohol, leaving a residue of collapsed cells (178). Considerable areas of rust-brown stain are also present sometimes, especially toward the base of the stem, under which the bark is dead to the cambium and sometimes even the wood is stained brown (178).

It is stated that in the plantation the bleeding and rust stains are considered part of the same disease, although both symptoms are not always found together at the same time (178). Usually no hyphae are to be found in the bark tissues observed in the sections of the bark of either of these two types of stained bark (178). The bark of a few dying trees, however, which had the rust spots near the ground line but few obvious symptoms of bleeding, had cracked in longitudinal fissures several inches in length, exposing the wood (178). A layer of white to pale-yellow septate hyphae 3.5\(\mu\) to 5.5\(\mu\) in diameter was found between the dead bark and the wood of one tree (178). The hyaline to brown hyphae of another fungus, measuring 4.7\(\mu\) to 7\(\mu\) in diameter and sparingly septate, were found in the wood and sparingly in the bark of another tree (178). These fungi were not identified, although a later report by Mitra (188) stated that attempts were being made to isolate a fungus from diseased material. Padwick (199) in 1939 reported that a species of *Rhizoctonia* had been isolated from a plant with the "splitting disease" symptoms.

A disease on *C. succirubra* and on Ledgeriana grafts was reported in Java, in which the leaves turned yellow and the entire plant withered (132). Brown, sharply outlined spots were found in the region of the graft union. Several plants died from the disease.

Rutgers (240) reported a disease of unknown cause that affected the trunks of 2- to 4-year-old grafts in Java. Numerous cracks
appeared in the bark just above the root collar, and, although the roots were entirely sound, the wood in the collar region was killed. The disease appeared when the rains came and attacked plants on poor soils.

ROOT AND COLLAR ROTS

Frequently accurate diagnosis of root diseases is difficult because the symptoms on the upper parts of the tree may be similar to those of other diseases and because secondary organisms may have entered the wholly or partially destroyed root system by the time the visible symptoms are evident (19). Furthermore, unfavorable climatic and soil conditions, as impermeable subsoils and high or low soil-moisture content, may so weaken the plants as to afford easy entrance for soil-inhabiting fungi that would not attack vigorous, living roots (19). Consequently, the isolation of a particular fungus species from the roots of a dying tree does not indicate always that the isolate is the sole causative agent. It would be equally misleading to consider the death of less vigorous plants growing on unfavorable sites and invaded by weakly parasitic organisms as caused by physiological causes alone.

The terms "root rot," "root disease," "root canker," "root collar canker," "collar disease," "collar canker," and "collar rot" were used indiscriminately in the earlier literature from the Netherlands Indies in describing root and collar diseases of cinchona. The causal agents were unknown, and frequently such meager information was given on the symptoms as to make it difficult to associate with certainty these reports with the works of later investigators who described specific root diseases and fungi. Consequently, the contributions of such writers as Berkhout (10), Eekhout (73), Kessler (146), and Warburg (280) are of historical interest mainly and will not be reviewed here.

Many references have been made in the Dutch literature to the "root diseases" or "root fungi" of cinchona (14, 54–56, 58, 72, 96, 108, 126, 170, 240, 282, and others). Wurth (as reported by Hunger, 131) stated that diseases of the root system are rather widespread and are the most dangerous diseases of cinchona in Java. The symptoms are not recognized usually until the disease is too far advanced for control. Also, control is difficult because the roots are not easily accessible (131). Cases of root diseases generally occur locally (157), and attacked plants are uprooted and harvested (56, 257). Cinchona succirubra and its hybrids are less susceptible than C. ledgeriana (205), and grafted stock is said to show much greater resistance than seedling stock to root fungi (59). As a result, in Java the vacancies left by uprooting diseased plants are filled usually with grafts on C. succirubra stocks or hybrids (56, 205). In some nurseries where the loss from root diseases has been high, the number of grafted trees used as replacements is said to exceed the number of original seedling stock still left in the planting (57) and this interplanted grafted stock usually remains vigorous and healthy (56).
Armillaria

*Armillaria mellea* Vahl ex Fr., the “white root fungus” or honey agaric, causes a root rot of cinchona in the nurseries and plantations in Java and Sumatra (13, 28, 52, 60–62, 64–67, 69, 88–93, 100, 109–112, 131, 144, 147, 148, 150, 157–160, 169, 171, 210, 231, 251, 252, 257, 262, 263, 271), in Belgian Congo (122, 185), in Nyasaland (20, 278), and in Uganda (268). *Cinchona ledgeriana* is more susceptible than *C. succirubra* (227); and 1- to 2-year-old plants in the nurseries are most frequently attacked (131), although older trees may be attacked when grown in partially cleared fields in which there are many standing stumps and dead-wood to serve as sources of infection (227). The presence of the disease is not apparent on the parts of the tree above ground until the fungus is well established in the roots (227). By the time the leaves begin to turn and fall, the main roots have become infected and the plant dies (131). Thin, rather tough white mycelium may be seen between the bark and wood of infected trees, and black rhizomorphs are sometimes found on the outside of the roots (227). Stoffels (262) states that the mycelial elements on cinchona are phosphorescent. Diseased plants should be grubbed out and the roots burned (131). If the area is to be replanted, all broken pieces of roots should be removed and well-slaked lime applied (131). Diseased stock in the nurseries in Java is replaced by Ledgeriana grafted on *C. succirubra* root-stocks (227).

*Armillaria mellea* Vahl ex Fr.—“Pileo carnos, tenui, explanato, squamoso-piloso; margine expanso, striato; stipite spongioso-farcto; annulo floccoso patente; lamellis adnatis, dente decurrentibus, subdistantibus, pallidis, demum sub Rufescenti-maculatis, farinosis.” (242, v. 5, p. 80.)

Fomes

In 1928 Sundararaman (265) reported *Fomes lamoensis* as causing a collar rot associated with *Rosellinia* sp. on 3-year-old cinchona trees at the Government cinchona plantation, Anamalai, India, and in 1931 Butler and Bisby (29) listed *F. lamoensis* (Murr.) Sacc. and Trott. as occurring on cinchona in India. In 1939 Keuchenius (150) reported a single case of *F. noxius* on cinchona in West Sumatra. According to Sharples (249), the brown root disease of rubber, tea, and other tropical plants was believed to be caused by *Hymenochaete noxia* until 1917. He reported that in 1917 Petch had found fructifications of a fungus on tea and hevea that had been killed by the brown root disease, and this fungus had been identified by Lloyd as *F. lamoensis* Murr. (249). Sharples (249) stated further that Corner pointed out in 1932 that two similar fungi had been confused: The first, *F. lamoensis*, is a harmless saprophyte, and the second, to which Corner assigned the name *F. noxius*, is a facultative parasite and the true cause of the brown root disease of *Hevea brasiliensis*.

In view of the above information it seems probable that the authors reporting *F. lamoensis* on cinchona in India were recording the occurrence of *F. noxius* Corner on this host. In reference to the occurrence of the disease at Anamalai, Sundararaman
(265) stated that the sanitation conditions on the plantation were very poor, since the ground was littered with dead trunks and rotting stumps and there was a lack of adequate surface and subsoil drainage. He advised the installation of a good drainage system and the removal of the trees and stumps of species likely to harbor the fungus (265).

*Fomes noxius* Corner.—Effuso-reflexed, pileus applanate, dimidiate, slightly ascending, up to 13.5 cm. radius, 25 cm. wide, resupinate part spreading up to 35 cm. wide, upper surface rapidly glabrescent, growing margin white, creamy white, or pale ochraceous; flesh 6 to 19 mm. thick at base, rarely up to 5 cm. thick, 1.5 to 12 mm. at 5 mm. from margin, 0.5 to 2 mm. in the resupinate part, crust 0.5 to 1 mm. thick, with black crustaceous lines; tubes short in the first season, 2 to 5 mm. at the base, 0.3 to 1.5 mm. at 5 mm. from margin, developing 2 to 5 layers, with a total thickness up to 11 mm., carbonaceous; pores 80μ to 110μ wide, dissepiments 40μ to 100μ thick; spores 3.5μ to 4.5μ × 3.0μ to 3.5μ; cystidia sparse; hymenial setae none; extrahymenial setae in the flesh up to 600μ × 4μ to 10μ, in the dissepiments up to 100μ × 9μ to 16μ; generative hyphae 2.5μ to 5μ wide (240).

**Fusarium**

Barat (6, 7) and Bugnicourt (24) have reported a serious root and collar disease on cinchona species in Indochina. Ledgeriana, Malabar, and *C. succirubra* plants are affected and also grafts (24); and the disease is found throughout the cinchona plantations, causing high mortality in seedbeds and plantations at an approximate elevation of 1,100 meters (7). Hendrickx (122) stated that cinchonas that have been planted too deeply are attacked by the disease. A species of *Fusarium*, possibly *F. moniliforme* Sheldon or an odorless variety of *F. vasinfectum* Atk., was isolated from the stems and roots of diseased plants (7). *Nectria* perithecia obtained from branches also produced *Fusarium* in culture (24).

The fungus attacks the inner bark of the stem, root, collar, and large roots (7) and forms cracks and cankers on the large roots extending up through the collar region (24). The cankers gradually become deep and circular in shape (24). The disease is initiated in the nurseries, and infected nursery plants showing the characteristic cracks and cankers wither soon after being transplanted and the leaves turn red (24). The growth of the plants is slowed down or stopped, and the plants die very slowly (7). The alkaloids in the bark are reduced very rapidly; however, the damage at elevations less than 1,100 meters has not been so great but that most of the trees could be exploited in 5 to 10 years (7). Better care and maintenance of the plantations is said to retard the disease (7).

The following control measures were suggested: Disinfection of the seed, use of soil disinfected with carbon disulfide or formaldehyde in the seed and nursery beds, discarding at transplanting time all plants showing symptoms, and destruction of all diseased plants in the nurseries and plantations (24). Barat (7) suggests that the disease might be overcome by planting at higher elevations, if these were available in Indochina, or by breeding disease-resistant varieties.
**Fusarium** sp.—Pustules on roots and stems whitish, projecting, round, isolated or in groups; spores hyaline, curved, 3- to 5-septate, 32µ to 42µ × 4µ to 4.5µ; *Nectria* perithecia spherical, reddish orange, isolated or in groups, with distinctly red papilla; asci 8-spored, 14µ × 6µ; ascospores 2-celled, hyaline, rounded at both ends, constricted (24).

In 1939 Bugnicourt (25) listed *Fusarium vasinfectum* Atk. as occurring on the stems and roots of *C. ledgeriana* in Central and South Annam, Indochina.

(See fusarium damping-off, p. 4, and fusarium stem blight, p. 12.)

**Ganoderma**

*Ganoderma pseudoferreum* (Wakef.) v. Over. and Steinm. has been reported on cinchona in Sumatra and Java (18, 89, 90, 158-161, 257) and Belgian Congo (134). In 1933 Leefmans (160) reported that a great deal of damage was done in the older Ledgeriana nurseries by the “root fungi” (including *Armillaria mellea, Rosellinia* sp., and *Ganoderma pseudoferreum*). In 1935 Goot (90) stated that in Sumatra the extent of damage caused by the red root fungus was exceeded only by that caused by *Rosellinia* spp., both in the nurseries and on plants up to 4 years old. The red root fungus, however, is said to remain thoroughly localized (160). Steinmann (257) stated that burning diseased plants, digging isolation ditches around infected areas, clear-cutting, and removing stumps from forest lands should be adequate for control.

*Ganoderma pseudoferreum* (Wakef.) v. Over. and Steinm.—Rhizomorphs light red becoming dark wine red to violet black in older specimens, flattened, branching, closely adpressed to surface of host roots, sometimes forming extensive networks or coverings, white mycelia from rhizomorphs penetrate host roots to cause a rot; sporophores solitary or only a few together, with more or less clearly developed stalk and cap, resupinate when on lower surface of exposed roots; cap more or less round, up to 30 cm. in diameter, upper surface rough in center, brown to brown black or brown red with violet or greenish tinge, margin zonate, light red brown; stalks when present same color as center of cap; inner tissue of cap black brown when fresh, cinnamon-colored with black-brown radial stripes when dry, composed of very thick-walled, branching hyphae, 4.5µ to 8.5µ in diameter; tubes darker than tissue, drying to a brownish color on the outside, barely 1 mm. long at edge of cap but up to 10 mm. at base of cap, not stratified; pore surface dirty yellowish white when dry, gray violet when drying, pores 90µ to 140µ in diameter; basidia colorless, 4-spored, short, about 20µ × 7µ; spores ellipsoid, hyaline at first, later with yellow-brown inner wall, slightly roughened, 6µ to 9.5µ × 2.8µ to 4µ (197).

**Physiological**

King (153) reported a physiological disease affecting cinchona in the plantations in Sikkim and the Nilgiris, India, in 1871-72. The disease was confined to trees in damp soils (153). The roots of the trees were attacked first, and the cortical and woody tissues gradually shriveled from the root upward (153). The leaves became discolored and fell, and the plants usually died (153). Owen (198) stated that the shriveled appearance of the young plants, the shrinking of the leaves, and the stunted growth of the primary branches were noticeable for some time before the roots
decayed. Draining the soil and harvesting the diseased trees before the bark became worthless were recommended (198).

Calder (36, 37) and Cowan (47, 48) reported a collar canker disease on cinchona in the Burma plantations in 1924-27. Only a limited amount of canker was present in 1923-24 (36); but in 1926-27 approximately 21,000 plants were killed, while many more were affected by the disease (48). The first symptoms were "flagging" of the whole plant, and the bark at the soil level and extending a few inches above became thick and dark and cracked longitudinally (37). Calder (37) stated that the symptoms increased rapidly during the rains in 1924-25. Trees in a state of collapse were observed on the sides of ridges, but were much more prevalent on the flat damp wind-swept crests (37).

Microscopic and cultural investigations failed to reveal a fungus in the affected plant tissues (37). The disease was believed to be a physiological disorder caused by planting too deeply, combined with wind action (37). When the young trees were swayed by the wind, hollow cone-shaped depressions were formed in the soil at the collar of the stems, in which water collected and was retained (37). Plants with the root systems near the soil level and with the collar completely free seldom exhibited the symptoms (37). Removing the earth from around the collar, shallow planting, good drainage (37), and the use of windbreak hedgerows were found to minimize the trouble (47).

The occurrence of root rot in cinchona plantings in Puerto Rico is believed to be favored by soil unsuitable for cinchona (117). The plantings are located on Nipe clay, a ferruginous laterite, which has a very low air content and becomes waterlogged during the rainy season. The taproot and some of the deeper roots become diseased and die. A mat of new roots is formed at the base of the diseased roots near the surface of the soil. This condition results in a check in the vigor of the trees, and some of the trees attain a pathological maturity after one or two seasons.

**Phytophthora**

Thompson (269) reported a root rot and dieback of *Cinchona ledgeriana* trees in the Cameron Highlands, Malaya. The affected trees were growing on a combination of shallow, sandy topsoil and a stiff subsoil into which the roots did not penetrate. The main root system partly decayed, and the bark just above and below the soil line was cankered or split. The cortex was soft and mottled with red-brown lines. *Phytophthora cinnamomi* Rands was isolated from the roots but not from the cankered tissue above the soil line. Often a species of *Phomopsis* was found in the affected cortex. Inoculation of roots and stems of trees and seedlings, however, indicated that the *Phytophthora* was a slow-acting, weak parasite and that the *Phomopsis* was probably a secondary organism. The *Phytophthora* did not reproduce the typical bark canker symptoms and advanced only a slight distance in the cortex in 2 months; and 2 to 5 months elapsed before wound-inoculated seedlings died. Thompson advised planting
Ledgeriana grafted on \textit{C. succirubra} rootstocks, unless good rich virgin soil was available for Ledgeriana seedlings.

(See phytophthora stem blights, p. 13, and phytophthora cankers, p. 32.)

In 1944 Crandall and Davis\footnote{Crandall, B. S., and Davis, W. C. \textit{Occurrence of Cinchona Root Rots in the Americas.} U. S. Bur. Plant Indus., Soils, and Agr. Engin., Plant Dis. Rptr. 28: 926–929. 1944. [Processed.]} reported high mortality from a root disease affecting the calisaya form of \textit{C. officinalis} in field plantings located in the valleys of the eastern slopes of the Andes in Bolivia. The infected plants had been grown from locally collected seed of wild trees. In several cases an entire planting had been destroyed. Faulty field-planting practices were said to be contributing factors. The young plants had been placed in the bottoms of holes about 1½ feet deep, and the drowning of the roots during the rainy season and the inability of the roots to make normal growth in the impervious clay subsoil apparently allowed rot fungi to gain entry. A fungus with the hyphal characteristics of a \textit{Phytophthora} was isolated from infected roots. Wild plants of the same cinchona species were found with rot lesions on the roots but otherwise were in healthy condition. The selection of root-rot-resistant plants and the modification of field-planting methods were suggested as means of cutting down plantation losses.

In 1944 Martin and Crandall (186) reported a root disease on Ledgeriana variety of \textit{C. officinalis} at an elevation of about 3,700 feet in Peru. A species of \textit{Phytophthora} was said to be the probable cause. Part of the planting was located on a well-drained hillside, where the death rate was about 35 percent, but the planting in the poorer drained bottom land had a mortality rate of about 56 percent. Infection appeared to be in the collar region, and death resulted when girdling took place at or above the collar within a year or two after infection. A root and collar rot in a newer planting was considered to be caused by the same fungus, although planting too deeply was thought to be partly responsible for the disease.

\textbf{Rosellinia}

Species of \textit{Rosellinia} cause the black or gray root rots of many tropical plants (230). In 1935 Heusden reported that "root fungi (\textit{Rosellinia} sp.)" were the principal cause of disease in old plantations of cinchona seedlings in South Sumatra (128). \textit{Rosellinia} sp. has been recorded on \textit{Cinchona} spp. in India (265), Belgian Congo (122, 135), Indochina (6), and Java and Sumatra (45, 60–62, 64–67, 69–71, 88, 89, 92, 93, 100, 101, 108–112, 128, 129, 144, 145, 157, 158, 160, 171, 172, 202, 210, 227, 231, 251, 252, 263, 271).

\textit{Rosellinia arcuata} Petch, frequently called the "gray root fungus" in the Dutch cinchona literature, has been reported on cinchona in Java and Sumatra (13, 45, 90–93, 147, 148, 150, 159, 161, 230, 257, 262). In 1931 Leefmans (159) wrote that \textit{R.}
arcuata occurred quite generally throughout South Sumatra and was rather serious on some plantations. Keuchenius (147) reported that in 1935 trees were still dying regularly from Rosellinia attack on an estate in West Sumatra where the young cinchona plantation had been injured by fire in 1930. Rant (230) states that the gray root fungus attacks young plants of Ledgeriana, C. succirubra, C. robusta, and hybrids in the nurseries and also occurs in plantations on the roots of older trees of the same species. It has been reported to affect seedlings in the germination beds occasionally (54). C. succirubra is more resistant than C. ledgeriana (257).

Since the fungus spreads by means of aerial-borne conidia or by root contact, diseased plants may occur in the plantations as isolated specimens or roughly grouped in circles (257). The fungus attacks the tips of primary and secondary roots; and the mycelium, which spreads over the surface of the root, is a gray-white color at first, later turning black (257). The white mycelium found between the bark and wood is characteristically arranged in small fan-shaped, starlike figurations about 1.5 cm. in diameter (257). The external mycelium may grow up over the collar and the base of the trunk under favorable conditions of moisture (262). Rant (230) successfully inoculated Ledgeriana seedlings with the fungus. He states that good drainage and cultivation of the soil in the nurseries are desirable and that diseased plants should be dug out and burned immediately (230). When the disease occurs in the plantation, the diseased trees should be removed and the area of contaminated soil should be isolated by drains and ditches (230). The use of resistant plants, as Ledgeriana grafts on C. succirubra rootstocks, is recommended by Steinmann (257).

Rosellinia arcuata Petch.—"Peritheciis gregariis, primum in mycelio purpureo-brunneo immersis, fusco-brunneis, dein nigris, liberis, carbonaceis, globosis, leniter depressis, 1.5—2.4 mm. diam., levibus, ostiolo conico 0.1 mm. alto, basi 0.4 mm. cr.; asci cylindraceis 300 × 8; sporis oplique monostichis; paraphysibus 2μ circ. cr., ascos aequantibus; sporis nigris, cymiformibus, apicibus acutis et saepe max contractis, 30—47 × 5—7." (242, v. 24, p. 834.)

Rosellinia bunodes (Berk. and Br.) Sacc. has been reported on Cinchona spp. in Java and Sumatra (90, 121, 147, 148, 150). Goot (90) stated that R. bunodes and R. arcuata were first in importance among the root fungi causing losses in the nurseries and on older plants (up to 4 years old) in the South and West Sumatra plantations in 1933.

Rosellinia bunodes (Berk. and Br.) Sacc.—"Peritheciis magnis, 2 mill. diam., globosis verrucosis e strato strigoso oriundis; sporidiis maximis fusiformibus, apicibus elongatis acutissimis, multinucleatis... Mycelium ex hyphis fasciculatis fuscis, articulatis, ramosis, sursum pallidioribus constans; conidiis moniliformi-catenuulatis 4—5μ long.; asci lumiinis apice capitato-fovealati." (242, v. 1, p. 254.)

Thompson (270) reported a fatal root and collar rot of two young cinchona hybrid trees in Malaya in 1940. The disease was caused by a species of Rosellinia.
Kevorkian (151) and Harper and Winters (118) have reported a root disease of cinchona in Puerto Rico caused by a fungus that appears to be similar to the "gray root fungus" of Java (the Graphium or imperfect stage of a species of Rosellinia). The disease is not detected until the trees, which appear to be healthy but making poor growth, suddenly wilt (151). The leaves dry out and remain attached for some time before falling (151). The main roots are found to be rotted off, leaving short stubs (151). When the adventitious roots have likewise rotted, the trees wilt and eventually die (151). Kevorkian stated that the prevalence of the disease in Puerto Rico may be caused in part by the fact that the plantation is on poorly drained clay soil (151).

Reinking 20 reported a root rot disease killing Ledgeriana plants on their own roots in Guatemala. In one case several trees in one row had been killed, while C. succirubra plants in the next row were not affected. Although a Rosellinia-like fungus was found on the roots of dead trees in one planting, the type of soil in which the trees were planted was also considered as a possible contributing factor.

Undetermined

Crandall and Davis 21 observed a dry root rot in a wild stand of Cinchona pubescens in Colombia, but the disease was not considered to be of economic importance.

Crandall and Davis 21 reported a root rot and collar canker disease on C. officinalis var. ledgeriana grafted on C. succirubra in Guatemala. The foliage of infected trees was thin, and the leaves were reddish and somewhat smaller than normal. A very narrow streak of infected cambial tissue was found to extend from a root lesion in the rootstock to a girdling lesion in the Ledgeriana scion, connecting through the graft union. These canker lesions on the scion extended up the trunk as much as 3 feet before complete girdling occurred just above the graft union. Some plants of Ledgeriana growing on their own roots were also attacked by the disease. This disease is thought to be caused by a soil-borne parasite that is probably also an active root rot on susceptible rootstocks.

The occurrence of a root disease in a 10-year-old planting of Ledgeriana in Peru has been reported by Crandall and Davis 21. The mortality rate is about 20 percent, and in some cases as many as three replants have been killed by the disease. The infection apparently starts in the collar region below the soil line, either on the main roots or at the base of the trunk. Cambial tissue is said to be invaded as high as 10 inches above the soil line before the tree is girdled and dies.

20 See footnote 5, p. 4.
21 See footnote 19, p. 41.
INJURIES ATTRIBUTED TO ENVIRONMENTAL FACTORS OTHER THAN SOIL CONDITIONS

DROUGHT

Calder (28) reported an unusual reaction by cinchona plants to drought injury in the plantations in South Burma in 1928-29. During a long period of dry weather a large area of cinchonas, which had been planted shallow in order to minimize the risk of collar disease, became quite dry and the plants lost almost all of their leaves. Within a week after the rains began the plants had leafed out and made healthy growth.

Rant (226) reported drought-killing of cinchona seedlings in seedbeds in Java. The humus, before being placed in the seedbed, had become rather impermeable to water, owing to overexposure to sunlight. Consequently, when the beds were watered, the moisture stood on the surface of the bed while the underlayer of soil remained quite dry.

FROST OR FREEZING

Leersum (163) reported severe frost injury to cinchona plants in old and young nurseries following a drought in Java. When the trees are badly hit by frost, the bark splits from the wood and remains loosely attached. The wood is brown and the bark is hard and brittle and fibrous. As thawing takes place rapidly in the Tropics, the plants quickly wilt and turn black. The sap exudes as a dark-brown liquid and runs down the trunk, drying to a rust-brown color. The alkaloidal content is not appreciably lowered if the bark is harvested within a short time.

LIGHT

Tests at the Puerto Rico Agricultural Experiment Station, at Mayaguez, showed that cinchona seedlings with poor root development caused by excess moisture in the seedbed are more sensitive to light than plants with good root development.22 Seedlings that are exposed to a slight excess of light become weak and the leaves develop a pinkish-red color, while overexposure to direct sun rays results in severe leaf burning (217). Reinking 23 has reported a leaf scorch characterized by red-brown scorched areas extending from the margin inward and thought to be caused by sunburning of weakened leaves, as occurring in Guatemala. Augusto (5) stated that lack of adequate shade results in a stunted condition and subnormal size of the leaves of Cinchona calisaya. Severe injury can result in the death of the seedlings (117), and Popenoe (208) stated that seedlings in the seedbed may be killed within a few hours if exposed to the direct rays of the sun. Cowan (48) reported that in 1926-27 the exceedingly hot sun at Mergui, Burma, caused more deaths in the cinchona plantation than did insects and fungus diseases.

23 See footnote 5, p. 4.
LITERATURE ON CINCHONA DISEASES AND FUNGI

LIGHTNING
Gorkom (97) reported lightning injury to *Cinchona succirubra* and hybrid trees in Java. The trees were stripped of their leaves, and the bark was brown and difficult to peel (97). Rant (223, 225) stated that the sap exudes and runs down the trunk, drying in rust-brown spots.

RAIN AND HAIL
Popenoe (208) stated that during the early months in the nursery many of the young cinchona plants may die if not protected from heavy rains. Washing of the soil in the seedbed may kill germinated seedlings by mechanical injury (202). Hail, if heavy, is said to strip a plantation of its leaves, but the check in growth is only temporary usually, as new leaves appear speedily (153). In 1932 Calder (33) reported considerable damage to the Munsong cinchona plantation in India by a severe hailstorm in May 1931.

SHADE
Tests at the Puerto Rico Agricultural Experiment Station, at Mayaguez, have shown that undesirable etiolation occurs in excessive shade (217).

SMOKE
Pring (212) reported that all the leaves on *Cinchona officinalis* growing in the City Garden, St. Louis, Mo., U. S. A., showed leaf burn after the cloud of smoke of December 17–18, 1931. The plants died.

Reinking* reported black blotching of cinchona leaves in Guatemala, caused by the rubbing of leaves covered with a layer of pumice from an active volcano.

WIND
Gorkom (94) quoted from the 1870 report on the cinchona crop in Java the statement that “the wind remains” the worst enemy of the cinchona plants. King (153) stated that excessive and frequent wind appears to do considerable and permanent damage, especially to *Cinchona succirubra*, the leaves of which are large and tender. In 1887 some cinchona trees in Jamaica were reported to have died, owing to wind action during a hurricane (137). The bark was injured at the collar region, and the subsequent invasion of the root bark by fungal mycelium resulted in the bark rotting (137).

MISCELLANEOUS FUNGI AND LICHENS
A number of fungi have been reported on cinchona that are not listed in this publication with those causing the diseases of cinchona, either because the reports were brief and failed to indicate diseased conditions caused by the fungal organisms or because the fungi are usually considered nonparasitic, although frequently found on plants dying from other causes. Brief discussions of these reports follow.

*See footnote 5, p. 4.*
Reichert and Hellinger (234), in reporting a tip end rot of banana fruits and flowers, listed cinchona as one of the many hosts attacked by Botrytis cinerea Pers.

A species of Chaetomium has been reported on dead twigs of cinchona in Uganda by Hansford (114).

In 1907 Koorders (155) reported a parasitic species of Diplodia, which he described under the name of D. cinchonae Koord., on the bark of old branches of Cinchona succirubra trees 1,500 meters above sea level in Middle Java. These branches were also attacked at the same time by Corticium salmonicolor Berk. and Br. and its so-called imperfect stage, Nectoc decretus Mass. (155). The Diplodia was scattered sparingly in the plantations (155). Rant (227) also collected D. cinchonae on dead branches of Cinchona sp. in Java and considered it to be saprophytic.

Diplodia theobromae (Pat.) Nowell has been reported as one of the fungi associated with root diseases on cinchona in Belgian Congo (135, 262).

An unidentified species of Diplopeltis was observed by Koorders (154) in 1905 in the bark of dying branches of 30-year-old C. succirubra trees in Middle Java. Numerous pycnidia with spores in various stages of development were found. Koorders (154) also found Diplopeltis on a 1-year-old cinchona hybrid that had died primarily because of a root disease. A few hyaline 2-celled spores were found on the later collection, but no pycnidia.

A species of Fusoma was observed by Koorders (154) in Java on the bark of a 1-year-old cinchona hybrid that had died primarily because of a root disease.

Glomerella sp. is listed as occurring on C. ledgeriana in India in 1936 (136).

In 1936 Keuchenius (147) reported Helicobasidium compactum Boed. as a root fungus on cinchona in West Sumatra.

A species of Helicobasidium has been reported on cinchona in Sumatra (160) and in Belgian Congo (135). It is said to be associated with root diseases (135, 262).

Oclemia and Celino (196) and Wollenweber (284, 285) listed cinchona as one of the hosts of Hypomyces haematococcus (Berk. and Br.) Wr.

Hypomyces ipomoeae (Halst.) Wr. has been recorded by Wollenweber (285) as occurring on cinchona.

Hansford (113, 115, 116) has reported Rhizoctonia lamellifera Small on Cinchona sp. in Uganda. The fungus form with the large sclerotia was the type observed on cinchona (115). In 1929 Leefmans (158) reported R. bataticola (Taub.) Butl. on Cinchona sp. in South Sumatra. R. bataticola and R. lamellifera are synonyms of Macrophomina phaseoli (Maub.) Ashby (4).

Koorders (154) reported a species of Pestalozzia on mature living leaves of old C. succirubra trees and on the dead stem of a 1-year-old cinchona hybrid in Middle Java. He stated that perhaps the species was identical with P. cinchonae Zimm.

Wallace (276, 277) reported Phomopsis sp. on twigs of Cinchona sp. in Tanganyika.
Polyporus rubidus Berk., the pink root fungus, has been reported on Cinchona sp. in West Sumatra (93, 148, 150).

Seymour (248) listed Polystictus fimbriatus Fr. (= Polyporus fimbriatus Fr.) on Cinchona sp.

Keuchenius (148, 150) has reported Poria sp., “the brick red root fungus,” on cinchona in West Sumatra.

Rant (227) and Leersum (167) reported the occurrence of Stilbum minutula Penz. and Sacc. on dying branches of young Ledgeriana grafts and seedlings and on branches of C. succirubra and hybrids in Java. The fungus, which is usually a saprophyte on deadwood and stems, forms red fruiting bodies on the cinchona branches (167). On cinchona branches that have been weakened from other causes, however, the fungus can invade the living host tissues and kill them (227). Although Rant considered the fungus identical to Stilbum minutula Penz. and Sacc., he preferred to call it Stilbella (227).

The following fungi were reported on commercial cinchona barks: Cystodium coccinenum Fée on bark of Cinchona lancifolia Mutis in South America (77); Gloniella chinincola Rehm and Gloniopsis regia Rehm on bark offered for sale as “cinchona regia” (227, 232, 242); Himantia cinchonarum Fée on cinchona trees in Peru (76); Hysterium enteroluecum (Ach.) Fr. on cinchona trade bark from South America (227, 242); Myriangium cinchonae Rehm on bark of “cinchona regia” offered for sale in India (29, 233, 242); Rhabdospora thallicola Tassi in a lichen on cinchona bark in Brazil (242, 267); Rhizomorpha cinchonarum Roth on bark of cinchona trees in “America” (76, 227, 242); R. erinum Fée on bark of C. cordifolia Mutis from South America (76, 227, 242); Stilbospora fumosa Fée on bark on C. floribunda Sw. from South America (76, 227, 242); Syncphalastrum elegans El. Marchal on bark of “cinchona rubra” offered for sale at Brussels (227, 242); Thelephora aurae Zenker on “china rubra” in South America (288); T. cyanescens Fée on cinchona bark in Lima, Peru (77); and T. lactea Fr. (= Corticium lacteum Fr.) on several species of Cinchona in South America (288).

Lichens, which are usually classed as a distinct group of plants, are composed of a fungus and an alga living in a symbiotic relationship (19). They are found on rocks and the bark of living or dead trees (19). The presence of lichens on a tree causes little or no damage to the host, although an abundant growth of lichens may cause the death of small twigs or small branches by smothering them and thus interfering with the normal plant processes (19).

The lichens in the following lists were reported by Fée (76, 77) and Zenker (288) as collected from trade barks of Cinchona species.


On Cinchona officinalis L.: Arthonia gregaria Fée, A. leucocheila Fée, A. leucocheila var. pallida Fée, A. obtrita Fée, A. patellula


**MYCORHIZAE**

Since the investigations of Frank in 1885, it has been known that the roots of many plants live symbiotically with certain kinds of fungi (255). Ludowyke (175) stated that there are two
schools of thought regarding the significance of the presence of mycorhizal fungi in the roots of plants. On the one hand, it is believed that the association is of mutual benefit to the host and fungus, and on the other, that the condition is one of suppressed parasitism and that the host plant receives no benefit from harboring the fungus \((175)\). It is possible that there is a range of conditions from the one extreme to the other \((175)\). If the condition is one of suppressed parasitism, then any factor that reduces the vitality of the plant will lessen its resistance to the fungus, with a consequent increase in degree of parasitism and the possible death of the plant \((175)\). Ectotropic mycorhizae form a sheath around the root and endotropic mycorhizae live within the host root cells \((255)\).

Steinmann \((255, 256)\) reported the presence of endotropic mycorhizae in the roots of healthy plants of *Cinchona succirubra* in Java. The mycorhizae are found in the thin capillary roots as well as in smaller roots 2 to 3 mm. in diameter \((255)\). The fungus is not present throughout the parenchymatous tissue of the root cortex but only in certain places \((255)\). These places may be recognized sometimes from the exterior of the root by irregular and sometimes nodular swellings on some parts of the root \((255)\). Sometimes the fungi are found several cell layers deep in the cortex, but do not penetrate the endodermis \((255)\). The hyphae are mostly brown, inter- and intra-cellular, branched, and generally 1.7μ to 3.5μ thick \((255)\). In some cases the hyphae unite into bundles, which change into rough yellow nodular masses \((255)\). In other cases dark-gray to brown fungal tissue of pseudoparenchymatic structure is found, which strongly resembles sclerotia \((255)\). Steinmann found the mycorhizae in cinchona roots in February and April on estates at very high elevation where the pH of the soil was 5.1 to 6.2 \((255)\).

**LITERATURE CITED**

(1) **Anonymous.**


(2) **Steinmann, F.**


(3) **Arens, P.**


(4) **Asby, S. F.**


(5) **Augusto, H.**


(6) **Barat, H.**

1931. *II. Laboratoire de Cryptogamie. Études de la division de phytopathologie (section sud-indochinoise de l'Institut des recherches agronomiques) au cours de l'année 1930.* Bul. Écon. de l'Indochine 34: 779B–796B.
(7) Barat, H.  

(8) Barber, C. A.  

(9) Batavia, Landbouw Syndicaat.  

(10) Berkhout, [A. H.]  

(11) Bernard, C.  
1925. SEPTOBASIDIUM BOGORIENSE OP JONGE KINABOOMEN. Cinchona 2: 46-48, illus.

(12) Bessey, E. A.  

1938. ANBAU DER CHINARINDE (CINCHONA) IN NIEDERLÄNDISCH-INDIEN. Tropenpflanzer 41: 231-245, illus. [Transl. by Burns, W., Cinchona Cultivation in the Netherlands-Indies. Indian Farming 1: 311-317. 1940.]

(14) Boecop, M. van.  
1929. SCHADUW IN KINA-PLANTSOENEN. Bergcultures 3: 1532-1533.


(16) Boedijn, K. B., and Steinmann, A.  

(17) and Steinmann, A.  

(18) Bondarzewa-Monteverde, V. N., Gutner, L. S., and Novoselova, E. D.  

(19) Boyce, J. S.  
1938. FOREST PATHOLOGY. 600 pp., illus. New York and London.

(20) Briton-Jones, H. R.  
1934. THE DISEASES AND CURING OF CACAO. 161 pp., illus. London.

(21) Brooks, F. T.  
1928. PLANT DISEASES. 386 pp., illus. London.

(22) Brown, W. H.  

(23)  

(24) Bugnicourt, F.  

(25)  

[27] Butler, E. J. 1918. Fungi and Disease in Plants. 547 pp., illus. Calcutta.


(45) Coster, C.  

(46) Couch, J. N.  
1938. THE GENUS SEPTOBASIDIUM. 480 pp., illus. Chapel Hill.

(47) Cowan, J. M.  

(48) ———  

(49) Crandall, B. S., and Davis, W. C.  

(50) Davis, W. C., Wright, E., and Hartley, C.  

(51) Duggar, B. M.  

(52) Du Pasquier, R.  
1933. PRINCIPALES MALADIES PARASITAIRES DU THÉIER ET DU CAFÉIER EN EXTRÈ-ORIENT. Bul. Écon. de l'Indochine (n. s.) 36: 1-144, illus.

(53) [Dutch East Indies] Departement van Landbouw, Nijverheid, en Handel.  

(54) ———  
1916. VERSLAG VAN DE GOVERNEMENTS KINA-ONDERNEMING TE TJIJNJIROEAN (BANDOENG) OVER 1915. 50 pp., illus. Bandoeng.

(55) ———  
[1917.] VERSLAG VAN DE GOVERNEMENTS KINA-ONDERNEMING TE TJIJNJIROEAN (BANDOENG) OVER 1916. 75 pp., illus. Bandoeng.

(56) ———  
1918. VERSLAG VAN DE GOVERNEMENTS KINA-ONDERNEMING TE TJIJNJIROEAN (BANDOENG) OVER 1917. 56 pp., illus. Bandoeng.

(57) ———  
1919. VERSLAG OMTRENT DE GOVERNEMENTS KINA-ONDERNEMING TE TJIJNJIROEAN (BANDOENG) OVER 1918. 54 pp., illus. Bandoeng.

(58) ———  
1920. VERSLAG OMTRENT DE GOVERNEMENTS KINA-ONDERNEMING TE TJIJNJIROEAN (BANDOENG) OVER 1919. 56 pp., illus. Bandoeng.

(59) ———  
1922. VERSLAG OMTRENT DE GOVERNEMENTS KINA-ONDERNEMING TE TJIJNJIROEAN (BANDOENG) OVER 1920. 56 pp., illus. Bandoeng.

(60) ———  
1922. VERSLAG OMTRENT DE GOVERNEMENTS KINA-ONDERNEMING TE TJIJNJIROEAN (PENGALENGAN) OVER 1921. 47 pp., illus. Bandoeng.

(61) ———  
1923. VERSLAG OMTRENT DE GOVERNEMENTS KINA-ONDERNEMING TE TJIJNJIROEAN (PENGALENGAN) OVER 1922. 53 pp., illus. Bandoeng.
LITERATURE ON CINCHONA DISEASES AND FUNGI

(62) [Dutch East Indies] Departement van Landbouw, Nijverheid, en Handel.
1924. Verslag omtrent de gouvernements kina-onderneeming te Tjinjiroean (pengalengan) over 1923. 44 pp. Weltevreden.

(63) ———

(64) ———
1925. Verslag omtrent de gouvernements kina-onderneeming te Tjinjiroean (pengalengan) over 1924. 48 pp. Weltevreden.

(65) ———
1926. Verslag omtrent de gouvernements kina-onderneeming te Tjinjiroean (pengalengan) over 1925. 48 pp. Weltevreden.

(66) ———

(67) ———

(68) ———

(69) ———

(70) ———

(71) ———

(72) ———

(73) Eekhout, G. W.

(74) Ellis, J. B., and Everhart, B. M.

(75) Engler, A., and Prantl, K.

(76) Fée, A. L. A.

(77) ———
[1837.] Supplément à l'essai sur les cryptogames des écorces exotiques officinales. 178 pp., illus. Paris.

(78) Feilden, G. S. C., and Garner, R. J.

(79) Filipjev, I. N.
(80) Fluiter, H. J. de, and Mulholland, J. J.

(81) Fosberg, F. R.

(82) Fürth, J.

(83) Galloway, L. D.

(84) Galloway, L. D.

(85) Gäumann, E. A.

(86) Gäumann, E. A.

(87) Goodey, T.
1933. plant parasitic nematodes and the diseases they cause. 306 pp., illus. London.

(88) Gooot, P. van der.

(89) Gooot, P. van der.

(90) Gooot, P. van der.

(91) Gooot, P. van der.

(92) Gooot, P. van der.

(93) Gooot, P. van der.

(94) Gorkom, K. W. van.

(95) Gorkom, K. W. van.
1888. een nieuwe plaag der kinaboomen. Indische Mercurur 11: 355. [For corrections see Indische Mercurur 11: 371. 1888.]

(96) Gorkom, K. W. van.
1891. de wortelziekte der kina en de gouvernements—rapporten. Indische Mercurur 14: 507.

(97) Gorkom, K. W. van.
(98) Gorkom, K. W. van.
Haarlem.

(99) [Great Britain Imperial Institute.]

(100) Groothoff, A.
Haarlem.

(101) Guillaume, M.

(102) Hall, C. J. J. van.

(103) ———

(104) ———

(105) ———

(106) ———

(107) ———

(108) ———

(109) ——— [1923.]

(110) ———

(111) ———

(112) ———

(113) Hansford, C. G.
(114) Hansford, C. G.  

(115) ———  

(116) ———  

(117) Harper, R. E.  

(118) ——— and Winters, H. F.  

(119) Hartley, C.  

(120) Heim, R.  

(121) Hendrickx, F. L.  

(122) ———  

(123) Hennings, P.  

(124) ———  

(125) ———  
1902. Fungi javanici novi a cl. prof. dr. zimmermann collecti. Hedwigia 41: 140-149.

(126) Heubel, G. A.  

(127) ———  

(128) Heusden, W. C. Van.  

(129) ———  

(130) Hooker, J. D.  

(131) Hunger, F. W. T.  


Kheswalla, K. F.

King, G.

Koorders, S. H.
1906. Resultaten van een voorloopig mikroskopisch onderzoek eener wortelziekte van jonge kinaplantjes veroorzaakt door Heterodera-aaltjes en een schimmel. Cultuurgids 7: 901-919, illus.

Krishna Ayyar, P. N.

Leefmans, S.


Leersum, P. van.
1891. Iets over de wortelziekte der kinaboomen Indische Cult. (Teysmannia) 2: 327-338.


1907. Bericht omtrent de gouvernements kina-onderneming over het 1e kwartaal 1907. Cultuurgids 9: 149-152.


(168) Leersum, P. van.

(169) ———

(170) ———
1913. KINA. In Gorkom, K. W. van, Oost-Indische Cultures, deel 3, pp. 57–154, illus. Amsterdam.

(171) ———

(172) ———

(173) Lieneman, C.

(174) Loble, [M.]

(175) Ludowyke, H. [Translator.]

(176) Lutz, M. L.

(177) McRae, W.

(178) ———

(179) ———

(180) ———

(181) ———

(182) Madarang, S. A.

(183) Marañon, J., and Bartlett, H. H.

(184) Marcinowski, K.

(185) Markham, C. R.
1880. PERUVIAN BARK. A POPULAR ACCOUNT OF THE INTRODUCTION OF CINCHONA CULTIVATION INTO BRITISH INDIA. 550 pp., illus. London.
(186) MARTIN, W. E., and CRANDALL, B. S.

(187) MITRA, M.

(188) ———

(189) MOENS, J. C. B.
1882. DE KINACULTUR IN AZIÉ, 1854–1882. 393 pp., illus. Batavia.

(190) MOSER, C. K.

(191) NOACK, F.

(192) ———
1907. KRANKHEITEN TROPISCHER NUTZPFLANZEN. Ztschr. f. Pflanzenkran. 16: 90–100.

(193) ———

(194) NOWELL, W.

(195) OFCEMIA, G. O.

(196) ——— and CELINO, M. S.

(197) OVEREEM, C. VAN.

(198) OWEN, T. C.

(199) PADWICK, G. W.

(200) ———

(201) PATOUILLARD, N., and LAGERHEIM, G. DE.

(202) PENNOCK, W.

(203) PETCH, T.

(204) PETRI, L.
LITERATURE ON CINCHONA DISEASES AND FUNGI


(207) Popenoe, W. 1941. CULTIVO DE LA QUINA (CINCHONA) IN GUATEMALA. 39 pp., illus. [Guatemala City.]


(213) Printz, H. 1940. VORARBEITEN ZU EINER MONOGRAPHIE DER TRENTEPOHLLIACEEN. NYT Mag. 80: 137-210, illus.


(219) Rands, R. D. 1922. STREEPKANKER VAN KANEEL, VEROORZAAKT DOOR PHYTOPHTHORA CINNAMOMI N. SP. Buitenzorg Inst. v. Plantenziekten Meded. 54, 53 pp., illus.


(221) 1909. KORTE AANTEKENINGEN OVER KINA. III. Indische Cult. (Teysmannia) 20: 409-417, illus.

(222) 1910. KORTE AANTEKENINGEN OVER KINA. IV. Indische Cult. (Teysmannia) 21: 777-780.
(223) Rant, A.

(224) KORTE AANTEKENINGEN OVER KINA. V. Indische Cult. (Teysmannia) 23: 610-612, illus.


(228) KORTE AANTEKENINGEN OVER KINA. VI. Indische Cult. (Teysmannia) 26: 54-57.


(232) Rehm, H.
1903. ASCOMYCETEN-STUDIEN. I. Hedwigia 42 (Beiblatt): (172)–(176).


(234) Reichert, I., and Hellinger, E.
1932. ON BOTRYTIS TIP-END ROT OF BANANA FRUITS IN PALESTINE. Hadar 5: 162-163, illus.

(235) Rhind, D.

(236) Ritzema Bos, J.
1926. BEKNOPTE AANTEKENINGEN OP PLANTENZIEKTENKUNDIG GEBIED. 56. SEPTOBASIDIUM BOGORIENSE PAT. OP JONGE KINABOOMEN. Tijdschr. over Plantenziekten 32: 303-304.

(237) Roepke, W.

(238) Rogers, D. P.

(239) Roggen, M. A. van.

(240) Rutgers, A. A. L.

(241) S., J. L.
1877. CULTURE DU CINCHONA ET DU JALAP, À LA JAMAÏQUE. Jour. de Pharm. et de Chim. (ser. 4) 26: 215-216.


(266) Sydow, P., and Sydow, H. 1924. UREDO CINCHONAE. MONOGRAPHIA UREDINEARUM SEU SPECIEUM OMNIUM AD HUNC USQUE DIEM COGNITARUM DESCRIPTIO ET ADUMBRATIO SYSTEMATICA. v. 4, fasc. 4, p. 561. Leipzig.
(270) 1941. NOTES ON PLANT DISEASES IN 1940. Malayan Agr. Jour. 29: 241-245.


Deficiencies:
chlorophyll 25
iron 25
nutrient 25

Dendrophoma cinchonae 11

Diplodia—
cinchonae 46
species 13
stem blight 12
theobromae 12, 35, 46

Diplopteltis 46
Djamoer oepas 30
Dorylaimns 21
Drought injury 44
Ecuador 22
Eelworms 20
Elsinoe—
cinchonae 27
scab 26
Eritrea 25
Fomes—
lamoensis 37
lamoensis 37
noxius 37
root rot 37

Formaldehyde, control of—
damping-off 4, 6, 7
fusarium root rot 38
Formalin, control of phytophthora stem blight 13
Formosa 4
France 23
French Guinea 4
Frost or freezing injury 44
Fungi on commercial barks 47
Fungicides:
aromatic nitrated phenols 4
bordeaux mixture 7, 10, 11, 13, 15, 16, 24
calcium hypochlorite 8
carbolic acid 9
carbonate 17, 33
carbolime plantarum 33
chloride of lime 17
copper 3
copper spray 11, 16
copper sulfate 28
Cryptonol 6, 13
Cuprocide 7
cuprous oxide 7
formaldehyde 4, 6, 7, 38
formalin 13
Kerol 13
lime 8, 11, 17, 27, 37
malachite green 13
mercuric chloride 7
Nosperit 6
Semesan 7
sulfur 6, 26
Superol 6
Terbolan 6
Yellow Cuprocide 7, 16

Fusarium—
damping-off 4
moniliforme 38
root rot 38
species 3, 4, 13, 29
stem blight 12
vasinfectum 38

Fusoma 46

Ganoderma—
pseudoferreum 39
root rot 39

German East Africa 18
Gloeosporium 46
Glomerella 46
Goniella chinincola 47
Gloniopsis regia 47
Graphium 43
Gray dadap fungus 29
Green algal leaf spot 22
Guatemala 4, 7, 16, 21, 24, 26, 30, 33, 34, 43, 45

Guignardia yersini 11

Hail and rain injury 45
Helicobasidium compactum 46
Helminthosporium 13
Heterodera marioni 19, 20, 21
Hevea, host for—
Corticium salmonicolor 31
Fomes noxius 37
Hevea brasiliensis, host for
Fomes noxius 37
Hide-bound condition 29
Himantia cinchonarum 47
Honey agaric 37
Hormiscium—
pannosum 27
sooty mold 27

Horsehair blight 26
Hymenochaete novia 37
Hypomyces—
haematococcus 46
ipomoeae 46
Hysterium enteroleucum 47

India 7, 12, 15, 17, 20, 21, 27, 29, 30, 33, 35, 37, 39, 41, 45, 46, 47
Indochina 4, 9, 10, 11, 25, 38, 39, 41

Injuries attributed to environmental factors:
drought 44
frost and freezing 44
light 44
lightning 45
rain and hail 45
shade 45
smoke 45
wind 45

Irish potato, host for Rhizoc-
tonia solani 6
Iron deficiency 25
<table>
<thead>
<tr>
<th>Disease/Subject</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jamaica</td>
<td>7, 34, 45</td>
</tr>
<tr>
<td>Java. See Netherlands Indies.</td>
<td></td>
</tr>
<tr>
<td>Kerol, control of fusarium stem blight</td>
<td>13</td>
</tr>
<tr>
<td>Lapp disease</td>
<td>10</td>
</tr>
<tr>
<td>Lasiodiplodia theobromae</td>
<td>12</td>
</tr>
<tr>
<td>Late damping-off</td>
<td>3</td>
</tr>
<tr>
<td>Leaf mottle</td>
<td>25</td>
</tr>
<tr>
<td>Leaf scab</td>
<td>10</td>
</tr>
<tr>
<td>Leaf spot:</td>
<td></td>
</tr>
<tr>
<td>algae</td>
<td>21</td>
</tr>
<tr>
<td>cephalurose and other algae</td>
<td>21</td>
</tr>
<tr>
<td>cercospora</td>
<td>22</td>
</tr>
<tr>
<td>colletotrichum</td>
<td>9</td>
</tr>
<tr>
<td>gloeosporium</td>
<td>9, 22</td>
</tr>
<tr>
<td>Lapp disease</td>
<td>10</td>
</tr>
<tr>
<td>phyllotecta</td>
<td>11, 22</td>
</tr>
<tr>
<td>phyllotectina</td>
<td>10</td>
</tr>
<tr>
<td>prillieuxina</td>
<td>23</td>
</tr>
<tr>
<td>sclerotinia</td>
<td>23</td>
</tr>
<tr>
<td>uredo</td>
<td>24</td>
</tr>
<tr>
<td>Lichens</td>
<td>47</td>
</tr>
<tr>
<td>Light injury</td>
<td>44</td>
</tr>
<tr>
<td>Lightning injury</td>
<td>45</td>
</tr>
<tr>
<td>Lime, control of</td>
<td></td>
</tr>
<tr>
<td>armillaria root rot</td>
<td>37</td>
</tr>
<tr>
<td>leaf disease</td>
<td>11, 27</td>
</tr>
<tr>
<td>myxomycete</td>
<td>8</td>
</tr>
<tr>
<td>stem canker</td>
<td>17</td>
</tr>
<tr>
<td>Loquat, host for Corticium salmonicolar</td>
<td>31</td>
</tr>
<tr>
<td>Macrophomina phaseoli</td>
<td>46</td>
</tr>
<tr>
<td>Malachite green, control of fusarium stem blight</td>
<td>13</td>
</tr>
<tr>
<td>Malaya</td>
<td>14, 40, 42</td>
</tr>
<tr>
<td>Marsamus</td>
<td>8, 26</td>
</tr>
<tr>
<td>Mechanical injury</td>
<td>8</td>
</tr>
<tr>
<td>Mercuric chloride, control of rhizoctonia damping-off</td>
<td>7</td>
</tr>
<tr>
<td>Mineral—</td>
<td></td>
</tr>
<tr>
<td>deficiencies</td>
<td>25</td>
</tr>
<tr>
<td>toxicity</td>
<td>25</td>
</tr>
<tr>
<td>Mold, sooty</td>
<td>25, 27</td>
</tr>
<tr>
<td>Monilopsis aderholdii</td>
<td>5</td>
</tr>
<tr>
<td>Mopog (mopo)</td>
<td>5</td>
</tr>
<tr>
<td>Mosaic</td>
<td>25</td>
</tr>
<tr>
<td>Mycorhizae:</td>
<td></td>
</tr>
<tr>
<td>ectotropic</td>
<td>49</td>
</tr>
<tr>
<td>endotropic</td>
<td>49</td>
</tr>
<tr>
<td>Myrangium cinchonae</td>
<td>47</td>
</tr>
<tr>
<td>Myxomycete</td>
<td>8</td>
</tr>
<tr>
<td>Necator decretus</td>
<td>31, 46</td>
</tr>
<tr>
<td>Nectria—</td>
<td></td>
</tr>
<tr>
<td>amaniana</td>
<td>18</td>
</tr>
<tr>
<td>cinchonae</td>
<td>18</td>
</tr>
<tr>
<td>coffeicola</td>
<td>18</td>
</tr>
<tr>
<td>species</td>
<td>13, 30, 34, 38</td>
</tr>
</tbody>
</table>

Nematodes                                          | 19, 20 |
Netherlands Indies                                  | 25   |
Nosperit, control of mopog damping-off              | 6    |
Nutrient deficiency                                 | 25   |
Nyasaland                                          | 37   |
Olpidiaceae                                        | 17   |
Palm, host for Phytophthora palmivora               | 15   |
Pellicularia—                                       |      |
filamentosa                                         | 5    |
koleroga                                            | 26   |
Peru                                                | 7, 16, 19, 21, 22, 26, 29, 30, 33, 34, 41, 43, 47|

Pestalozzia—                                        |      |
cinchonae                                           | 18, 46|
funerea                                             | 3    |
myristica                                           | 4    |
Peziza                                              | 30   |
Philippine Islands                                  | 6, 14, 20, 34 |
Phlyctaena cinchonae                                | 11   |
Phoma cinchonae                                     | 11   |
Phomopsis                                           | 22, 40, 46|
Phyllotecta—                                        |      |
cinchonae                                           | 10, 22, 23|
cinchonacola                                       | 11   |
cinconicola                                         | 23   |
konbaensis                                          | 11   |
leaf spots                                          | 11, 22|
yersini                                             | 11, 12|
Phyllostictina leaf spot                            | 10   |
Physalospora cinchonae                              | 12   |
Physiological—                                       |      |
stem canker                                         | 17   |
root rot                                            | 39   |
Phytophthora—                                        |      |
cactorum                                            | 3    |
canker                                              | 32   |
cinchonae                                           | 13   |
cinnamomoni                                         | 3, 14, 40|
colocasiae                                         | 15   |
faberi                                              | 14   |
palmivora                                           | 15, 32|
parasitica                                         | 3, 15, 16, 33|
root rot                                            | 40   |
species                                             | 16, 17, 41|
stem blight                                         | 13   |
stripe canker                                       | 32   |
trunk and branch canker                             | 32   |
Pink disease                                        | 30   |
Pink root fungus                                    | 47   |
Pinus insularis, host for Rhizoctonia solani        | 6    |
Polyporus—                                           |      |
fimbriatus                                          | 47   |
rubidos                                            | 47   |
Polystictus fimbriatus                              | 47   |
Poria                                               | 47   |

Most of the diseases discussed in this publication occur in the Netherlands Indies.
<table>
<thead>
<tr>
<th>Disease/Pathogen</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prillieuxina cinchonae</td>
<td>23</td>
</tr>
<tr>
<td>Puerto Rico</td>
<td>7, 16, 18, 25, 37, 40, 43, 44, 45</td>
</tr>
<tr>
<td>Pythium</td>
<td>3, 4, 7</td>
</tr>
<tr>
<td>Rain and hail injury</td>
<td>45</td>
</tr>
<tr>
<td>Rhododospora thallicola</td>
<td>47</td>
</tr>
<tr>
<td>Rhizoctonia — bataticola</td>
<td>46</td>
</tr>
<tr>
<td>damping-off</td>
<td>5</td>
</tr>
<tr>
<td>lamellifera</td>
<td>46</td>
</tr>
<tr>
<td>root rot</td>
<td>18</td>
</tr>
<tr>
<td>solani</td>
<td>5</td>
</tr>
<tr>
<td>species</td>
<td>3, 35</td>
</tr>
<tr>
<td>Rhizomorpha — cinchonarum</td>
<td>47</td>
</tr>
<tr>
<td>crinum</td>
<td>47</td>
</tr>
<tr>
<td>Root and collar rot</td>
<td>36</td>
</tr>
<tr>
<td>Root rot:</td>
<td></td>
</tr>
<tr>
<td>armillaria</td>
<td>37</td>
</tr>
<tr>
<td>fomes</td>
<td>37</td>
</tr>
<tr>
<td>fusarium</td>
<td>38</td>
</tr>
<tr>
<td>ganoderma</td>
<td>39</td>
</tr>
<tr>
<td>physiological</td>
<td>39</td>
</tr>
<tr>
<td>phytophthora</td>
<td>40</td>
</tr>
<tr>
<td>rhizoctonia</td>
<td>18</td>
</tr>
<tr>
<td>rosellinia</td>
<td>41</td>
</tr>
<tr>
<td>sclerotium</td>
<td>18</td>
</tr>
<tr>
<td>sporodesmium</td>
<td>19</td>
</tr>
<tr>
<td>stigecosporium</td>
<td>20</td>
</tr>
<tr>
<td>Rosellinia — arcuata</td>
<td>41</td>
</tr>
<tr>
<td>bunodes</td>
<td>42</td>
</tr>
<tr>
<td>root rot</td>
<td>41</td>
</tr>
<tr>
<td>species</td>
<td>37, 39</td>
</tr>
<tr>
<td>Rough bark</td>
<td>29</td>
</tr>
<tr>
<td>Roundworms</td>
<td>20</td>
</tr>
<tr>
<td>Rubber tree, host for — Corticium salmonicolor</td>
<td>30</td>
</tr>
<tr>
<td>Fomes noxius</td>
<td>37</td>
</tr>
<tr>
<td>Russia. See U. S. S. R.</td>
<td>8</td>
</tr>
<tr>
<td>Salt, control of myxomycete</td>
<td>8</td>
</tr>
<tr>
<td>Saltpeter, control of myxomycete</td>
<td>8</td>
</tr>
<tr>
<td>Scab</td>
<td>26</td>
</tr>
<tr>
<td>Sclerotinia — fuckeliana</td>
<td>23</td>
</tr>
<tr>
<td>leaf disease</td>
<td>23</td>
</tr>
<tr>
<td>Sclerotium — rolfsii</td>
<td>18</td>
</tr>
<tr>
<td>root rot</td>
<td>18</td>
</tr>
<tr>
<td>Semesan, control of damping-off</td>
<td>7</td>
</tr>
<tr>
<td>Septobasidium — bogoriense</td>
<td>28</td>
</tr>
<tr>
<td>cinchonae</td>
<td>28</td>
</tr>
<tr>
<td>lichenicolum</td>
<td>28</td>
</tr>
<tr>
<td>Shade injury</td>
<td>45</td>
</tr>
<tr>
<td>Smoke injury</td>
<td>45</td>
</tr>
<tr>
<td>Sooty mold:</td>
<td></td>
</tr>
<tr>
<td>capnodium</td>
<td>25</td>
</tr>
<tr>
<td>hormiscium</td>
<td>27</td>
</tr>
<tr>
<td>Sporodesmium — cinchonae</td>
<td>19</td>
</tr>
<tr>
<td>root rot</td>
<td>19</td>
</tr>
<tr>
<td>species</td>
<td>20, 21</td>
</tr>
<tr>
<td>Sporangium</td>
<td>13</td>
</tr>
<tr>
<td>Stem blight:</td>
<td></td>
</tr>
<tr>
<td>diplodia</td>
<td>12</td>
</tr>
<tr>
<td>fusarium</td>
<td>12</td>
</tr>
<tr>
<td>phytophthora</td>
<td>13</td>
</tr>
<tr>
<td>Stem canker:</td>
<td></td>
</tr>
<tr>
<td>Olpidiaceae</td>
<td>17</td>
</tr>
<tr>
<td>physiological</td>
<td>17</td>
</tr>
<tr>
<td>Stem rust</td>
<td>30</td>
</tr>
<tr>
<td>Stemonitis</td>
<td>8</td>
</tr>
<tr>
<td>Stigeosporium — cinchonae</td>
<td>20</td>
</tr>
<tr>
<td>root rot</td>
<td>20</td>
</tr>
<tr>
<td>Stilbella</td>
<td>47</td>
</tr>
<tr>
<td>Stilbospora fumosa</td>
<td>47</td>
</tr>
<tr>
<td>Stilbun minuta</td>
<td>47</td>
</tr>
<tr>
<td>Stripe canker</td>
<td>32</td>
</tr>
<tr>
<td>Sulfur, control of — leaf disease</td>
<td>27</td>
</tr>
<tr>
<td>mopog damping-off</td>
<td>6</td>
</tr>
<tr>
<td>Sumatra. See Netherlands Indies.</td>
<td></td>
</tr>
<tr>
<td>Superol, control of mopog damping-off</td>
<td>6</td>
</tr>
<tr>
<td>Syncephalastrum elegans</td>
<td>47</td>
</tr>
<tr>
<td>Systemic diseases</td>
<td>29</td>
</tr>
<tr>
<td>Tanganyika</td>
<td>18, 46</td>
</tr>
<tr>
<td>Tea, host for — Corticium salmonicolor</td>
<td>30, 31</td>
</tr>
<tr>
<td>Fomes noxius</td>
<td>37</td>
</tr>
<tr>
<td>Terbolan, control of mopog damping-off</td>
<td>6</td>
</tr>
<tr>
<td>Thelephora — aurea</td>
<td>47</td>
</tr>
<tr>
<td>cynescens</td>
<td>47</td>
</tr>
<tr>
<td>lactea</td>
<td>47</td>
</tr>
<tr>
<td>Thread blight</td>
<td>26</td>
</tr>
<tr>
<td>Theadworms</td>
<td>20</td>
</tr>
<tr>
<td>Tomato, host for Rhizoctonia solani</td>
<td>6</td>
</tr>
<tr>
<td>Tracheomycosis</td>
<td>29</td>
</tr>
<tr>
<td>Trunk and branch canker:</td>
<td></td>
</tr>
<tr>
<td>corticium</td>
<td>30</td>
</tr>
<tr>
<td>dasyscypha</td>
<td>32</td>
</tr>
<tr>
<td>phytophthora</td>
<td>32</td>
</tr>
<tr>
<td>Tubercularia</td>
<td>30</td>
</tr>
<tr>
<td>Tulasnella cinchonae</td>
<td>29</td>
</tr>
<tr>
<td>Turf fungus</td>
<td>9</td>
</tr>
<tr>
<td>Tylenchorhynchhus alatus</td>
<td>21</td>
</tr>
<tr>
<td>Tylenchus — alatus</td>
<td>21</td>
</tr>
<tr>
<td>coffeae</td>
<td>21</td>
</tr>
<tr>
<td>Uganda</td>
<td>22, 25, 37, 46</td>
</tr>
<tr>
<td>United States of America</td>
<td>22, 45</td>
</tr>
<tr>
<td>Uredo — cinchonae</td>
<td>24</td>
</tr>
<tr>
<td>leaf spot</td>
<td>24</td>
</tr>
<tr>
<td>Page</td>
<td>Page</td>
</tr>
<tr>
<td>------</td>
<td>------</td>
</tr>
<tr>
<td>U.S.S.R.</td>
<td>9, 12, 22</td>
</tr>
<tr>
<td>Verticillium</td>
<td>13, 29</td>
</tr>
<tr>
<td>Virus disease</td>
<td>27</td>
</tr>
<tr>
<td>White root fungus</td>
<td>37</td>
</tr>
</tbody>
</table>

☆ U.S. GOVERNMENT PRINTING OFFICE: 1947—748314