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STUDIES ON DISEASES OF INDIAN MEDICINAL PLANTS-II

BY

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An account is given of major diseases of Cinchona *Cinchona ledgeriana* Moens ex Trimen, and *C. succirabrg* Pavon ex Ktotzsch), namely seedling blight by *Phytophthora palmivora* Batler Pink disease caused by *Pellicularia salmonicolor* (Bark & Br.) Dastur, damping off of seedlings by *Pythium vexans* de Bary, and brown root rot by *Fomes lamaoensis* (Murr) Sacc. & Trott and stam canker, collar rot and root rot caused by *Phytophthora cinnamonis* Rand. Of these seedling blight, and pink disease are the most important ones.

CINCHONA

Botanical Name : *Cinchona ledgeriana* Moens ex Trimen, *C. succirubra* Pavon ex Klotzsch

Family : Rubiaceae

The plant is a native of South America. This is cultivated in Indonasia, Ceylon, India, Burma, Tangenyika and other countries for the bark which is the commercial source of quinine and other antimalerial drug.

C. ledgeriana and *C. succirubra* are weak, straggling but fast growing trees, of about 7 metre high. The bark contains a high percentage of quinint (upto 14 per cent) and is utilized for preparation of quinine. *C. ledgeriana* is more efficient producer of quinine.

In recent years, cinchona plantations in the world as well as in India have lost much importance and have suffered a set back owing to the discovery of a number of synthetic drugs which are being used as substitute of quinine. Cultivation of cinchona is gradually being given up and the land under cinchona plantations is being used for some other purposes. So, in the past 20-30 years virtually no work has been undertaken on the diseases of cinchona except a few stray reports.

Seedling blight

Phytophthora palmivora Butler

The disease was first observed in a very serious form in the Government farm at

Mungpoo in the Darjeeling district of West Bengal and a species of *Phytophthora* was found to be the causal agent (McRae, 1933). Mitra (1931) isolated a species of *Fusarium* from the root and collar portions of the root and collar portions of the dying seedlings in the same areas but its pathogenicity could not be established. Later Kherwalla (1935) carried out thorough investigations on this disease in India.

Symptoms

When first reported, the disease was found to be very destructive to the seedlings in the nursery. It is not known how far the disease is serious at present.

At the first onset of the disease, a discoloured area is evident in the collar region of the seedlings. The discolouration gradually extends until it reaches the cotyledons, which become flaccid and turn dowards. The leaves gradually become yellow, lose their turgidity and may fall down in extreme cases. Discoloured areas soon undergo rotting, which proceed from the bark to the vascular region, and the seedlings gradually hang downward and eventually die. Under moist conditions, copious mycelial growth over the rotted tissues is observed.

The causal organism

In the affected areas, mycelium is chiefly present in the bark, cortex and wood and the hyphae are both inter and intercellular. Haustoria are not observed but the cell walls in the infected tissues undergo dissolution and the cells collapse. Loosely attached sporangia are also present in the aerial mycelium. Sporangia on the host usually measure 30-79 μm $\times$ 21.6-45 μm, the mean being 52 μm $\times$ 44 μm.

In culture the fungus produces moderate aerial growth on oat meal agar. Aerial hyphae are much broader than the submerged ones, roughly granular and knotted occasionally. Sporangia are formed sparingly in culture. They are terminal, rarely intercalary, with a distinct pedicel, broadly ovate, pear-shaped, ellipsoidal to spherical, with a distinct papilla, measuring 17.5-58.5 μmX9.0-30.0 μm, the average being 33.3 μm X17.6 μm. Germination may be direct by germ tube under higher temperature or indirect through the production of zoospores at lower temperatures. Each sporangium produces 13-20 zoospores, and they are liberated through the papillate (Kheswalla, 1935). Oospore formation has not been noticed on the host or in pure culture, but it readily takes place in mixed culture with *Phytophthora colocasiae*, *P. oarasitics* or isolates from other hosts of *P. faberi* and *P. palmivora* (McRae, 1932 ; Khaswalla, 1935).

The optimum temperature for growth of the fungus has been found to be 24°C,

above and below which growth decreases and altogether ceases at 35°C (Khaswalla, 1945).

The pathogen has also been reported from Indonesia, where it has been observed to produce stem canker disease (Keuchenius, 1938).

Celioo (1934) reported a serious disease of cinchona from thc Philippines caused by a species of *Phytophthora*, which he identified as *Phytophthora faberi* Maubl. *Phytophthora faberi* has been considered to be morphologically similar to *Phytophthora palmivora* by Butler (1924) and Gadd (1924) Ashby (1929) and Thompson (1929) regarded them to be identical and the specific name *Phytophthora palmivora* Butler has been retained by the majority of workers. So the disease in the Philippines may be considered to be due to *Phytophthora palmivora* Butler.

Perpetuation and Spread

The fungus perpetuates in soil in the form of oospores. The fungus has also a wide host range on which it can complete its life cycle. The spread is mainly through water and zoospores.

Control Measures

In India no control measure has so far been tried. In other cinchona growing countries, attempts have been made to control the disease by the process of soil fumigation or soil sterilization. How far these measures have been successful and economical is not known. In Indonasia effective control measures have been obtained by weekly applications of Socony product 2295 A, together with 5 per cent carbolineum plantarium. Winters (1951) in Puerto Rico obtained gcod results in preventing diseases caused by Phytophthora species by (i) the application of chloropicrin or DD mixture at the rate of 336 kg per hectare, (ii) injecting 2ml of the chemical at 20 cm interval at 10-12 cm depth or (iii) sprinkling dilute formalin, (5 table spoonfuls of 40 per cent formalin in 340 litres of water applied at the rate of 365 litres per square meter) or (iv) fermate a proprietory this cerbamate fungicide (1 table spoonful in 100 litres of water) at the rate of 365 litres per square meter. Spraying on the foliage and at the base of the stem and drenching the soil with 10% Bordeaux mixture or any other proprietory copper fungicide as practiced in the control of other Phytophthora diseases are likely to be effective in controlling this particular one.

Pink Disease

Pellicularia salmonicolor (Berk. & Br.) Dastur
(= *Corticium salmonicolor* Berk. & Br.)

This disease was first reported from Mansung Plantations of the Darjeeling

district in 1930 by McRae. The disease has been found to be present for a long time in other countries like West Indies, Indonesia etc (Butler, 1918). In India this disease has been observed in orange, tea, hevea rubber, coffee, camphor, mango, jack fruit, crotelaria and *Cassia mimosoides* (Butler, 1918). It has also been found to attack a large number of host plants in other countries (Butler, 1918). Out of two species, Cinchona ledgeriana suffers more than *C. succirubra* because of the more crowded development of the branches and the foliage in the former. The disease is believed to cause wide spread damage to cinchona plants.

Symptoms

The disease usually affects the smaller twigs and branches. The leaves of one or several branches lose their green colour, gradually turn yellow and often wither without being shed. The branches may also wither rapidly when attacked. The The pathogen is easily recognised on the host in the *Corticium* stage, when the affected part is covered by a rose-coloured or whitish crust and somewhat cracked on the underside of the infected horizontal branches and on all sides in vertical branches. This stage is very common on *Cinchona* spp. The spread of the disease is influenced by moisture and exposure, the crust being more pronounced on the moist and shaded side. The pink layer becomes ochraceous or may be bleached to white with age and exposure. After the pink crust stage comes the spore-forming stage, which was previously known as necator stage of the disease, when irregularly rounded orange coloured pustular bodies (0.5—3.0 mm in diameter) appear, often crowded together, either superficialy or bursting through the bark.

The extent of damage depends upon the size of the tree. Under the crust the tissues are usually dead, sometimes as far as the cambium. There may be longitudinal cracking of the bark, resulting in formation of a wound, wherein hyphae with clamp connections may be found in layer next to wood. The basidual stage is rather rare.

The Causal Organism

The mycelium consists of fine white, sparsely septate, thin walled hyphae cropping over the surface of bark, either forming white sheets or remaining thread like with frequent clamp connections. Hyphae later aggregate into dense coils to form nodules, chiefly in the lenticels. It is suggested that the entrance into the host tissue occurs at the stage. In the neighbourhood of the nodules, the cells of the host tissue are killed and turned brown and this appears to be the first stage of attack. When the wood and medullary rays are invaded, hyaline hyphae are formed inside the vessels and tyloses are frequently formed. In the necator

stage, fruit bodies arising from the internal mycelium consist of a stromatic mass of pseudoparenchymatous calls. The outer layer, after its emergence through the bark, gradually and continuously develops into spores, until all the cells of the entire stroma are completely transformed into spores. The spores are individually hyaline, collectively pink, thin walled, roundish to irregularly angular in shape, very variable in size 10-35 μm X 7-17 μm in diameter. The spore readily germinate in water, produciug one or more germ tubes which give rise to septate mycelium. The basidial stage, when formed, is resupinate, made up of loose basal layer of interwoven hyphae, and densely packed, club shaped basidia, 17-33 μm long and 5-8 μm broad. Four basidiospores are produced on eaeh basidium. The spores are hyaline, pear shaped and 6-7 μm in diameter.

Under artificial conditions, the fungus forms only sterile creeping mycelium often with nodular form. No fruit bodies are formed in culture.

The pathogen attacks a large number of perennial plants and the disease easily passes from one plant to another. The existence of biologic races is not known (Butler, 1918).

Perpetuation and Spread

The fungus perists from one season to another as dormant mycelium in the callus formed around the canker or in bark and is disseminated during wet season. Humid atmosphere and shady conditions are essential for the establishment of the parasite.

Control Measures

The measures for reducing the losses caused by the fungus have been (i) creation of less favourable conditions for establishment of the parasite, (ii) direct treatment of trees already infected and (iii) protective spraying for prevention of infection. Less favourable condition for the establishment of the disease can be created by providing less humid atmosphere, free circulation of air, reduction of shade, thin planting, avoidance of intercrops and good drainage. Direct treatment of the infected trees consists in cutting off the affected branches and their destruction by burning and putting a fungicidal pests or tar paint on the cut surface. Prophylactic spraying with 1%Bordeaux mixture has been recommended for the pink disease of rubber (Butler, 1918) Mundkur (1949) expressed doubt about the efficacy of the prophylactic measure in the case of pink disease of orange. Subba Rao (1936) stated that in pink disease of tea, removal of diseased branches and spraying them with Agral Shirlan (Selicylanilide) keeps the disease under control during the rainy season. It is not

known how far the prophylactic spraying recommended in other cases may be helpful in controlling the disease in cinchona, which is more affected than tea. Moreover, spraying of cinchona is much more difficult and costlier than spraying tea from the operational point of view.

Damping off of Seedling

Pythium vexans de Bary

Damping off of cinchona sesdlings and also the wilting of sprouts from the vegetatively propagated nurseries owing to the attack of *Pythium vexana* de Bery nave been reported from the Annalalais at an elevation of 1.167-1500 m (Ramakrishnan 1949).

Symptom

In the affected seedlings or sprouts, the roots undergo rotting and watet soaked lesions appear at the base below the soil level. One to three year old plants are also effected. The infected plants show reddening of levels, which droop down and are shed. Large proportions of roots of such plants are blackened and die. The cortex easily sloughs off and the entire plant is killed. Hyphae are present in the contex and sometimes even in the xylem vessels.

Causal Organisms

Hyphae without septa, and with a defferential distribution of protoplasm. Zoosporangia not formed except on germination of the oospore. Oogonia smooth spherical, light yellowish to brown, sessile. Antheridia single, club shaped, rising close to the oogonium frow the same hyphae. Oospore single, spherical aplerotic. wall thin, light yellowish brown, germinating by hyphae or zoospores.

Peretuation and Spread

The fungus survive in soil as oospore and spread through zoospores.

Control Measures

Control measures recommended for other diseases caused by *Pythium* sp. can be adopted successfuily for this disease. No specific control measure has been worked out for this disease on cinchona.

Brown Root Rot

Fomes lamaoensis (Murr.) Sacc. & Trott,

The disease was first described by Butler (1918) on tea and the pathogen was described as *Hymenochaete noxia* Berk. The fungus was later found to be synonymous with *Fomes lamaoensis* (Murr.) Sacc & Trott. (Butler and Bisby, 1931).

Its occurrence on cinchona was reported by Sundararaman (1928). The fungus has a wide host range including rubber, coffee, cacae, camphor and bread fruit and is widely distributed in the tropics (Butler, 1918). Extent of damage by this pathogen in cinchona plantation is not known, though it is fairly wide-spread and one of the commonest causes of root diseases on tea in the same region.

The disease has not been described in detail on cinchona but it is presumed that it follows the same pattern as in tea, coffee etc.

Observations have shown that the roots are effected and the plant dies slowly. The fungus begins to colonize first on the decaying wood of the felled stumps. A large number of different trees such as Melia, Cassia, and Grevellia are attacked. If the dead bush or tree is allowed to remain, the mycelium spreads along the roots until it comes in contact with healthy roots and infects them. The mycelium is unable to travel through soil (Butler, 1918).

The treatment of the disease is very simple. The diseased bushes or old stumps should be removed as thoroughly as possible. Quick lime may also be applied near the decaying bushes or stumps to destory all the remnants of affected roots. If all the fragments or roots are removed and destroyed, the chances of further infection are effectively eliminated.

STEM CANKER, COLLAR ROT AND ROOT ROT

Phytophthora cinnamoni Rand.

Ramakrishnan (1951) reported the occurrence of *Phytophthora cinnamoni* on *Cinchona succirubra* and *C. ledgeriana* as causing stem canker, collar rot and root rot, finally resulting in drying of branches aud death of plants. The incidence of infection has been found to be severe after the south-west monsoon. No other details about the disease are known.

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