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THE INCIDENCE OF GANODERMA ROOT AND BOLE ROT OF COCONUT IN SRI LANKA

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ABSTRACT

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A tapering disease of coconut in Sri Lanka caused by *Ganoderma boninense* Pat. is described. Affected palms show necrosis of pinnae, dropping of dead leaves, necrosis of male flowers and a reduction in nut setting. Extensive root decay is also noticed. During the final stages of the disease, a soft rot is observed at the bud. Further, stem-bleeding and rotting of the bole are observed. Fruit-bodies usually occur at the base of the bole.

INTRODUCTION

There are several diseases of unknown aetiology affecting the coconut palm (*Cocos nucifera L.*), in all parts of the world where it is grown. The leaf scorch disease recorded in Sri Lanka is one of them. Only a very few of these diseases have been described fully (Bock, Ivory and Adams, 1970). The comprehensive review of the literature on this subject by Maramorosch (1974) highlights the lack of systematic studies on coconut diseases. This is mainly because they have been studied essentially by foreign experts working in various countries on short term assignments.

In the initial stages of the CRI (Coconut Research Institute) studies, Peries (1974) reported that one of the causes for the tapering disease of coconut in Sri Lanka is infection by the fungus *Ganoderma boninense* Pat., a new record of the fungus in Sri Lanka. This causes a lethal root and bole rot of coconut palms which can become epidemic in some areas and under certain conditions in Sri Lanka. This paper describes the symptoms of the disease and its distribution in a limited area and discusses the possible methods by which it spreads.

SYMPTOMS

For the purpose of uniformity Maramorosch's (1964) system of describing symptoms is followed here, as done by Bock *et al* (1970) for describing lethal bole rot disease of coconuts.

Leaves. The infection of a tree by a lethal root disease fungus results in the progressive destruction of the roots and the basal tissues of the trunk. Therefore, the leaf symptoms they cause, general wilting and malnutrition, are characteristic of drought. The first symptom of this disease, therefore, is the wilting of the pinnae and then the fronds which turn yellowish green and later brown. The affected fronds, particularly the outermost whorls, become necrotic from the tip backwards and die. Dead fronds either droop from the point of attachment to the trunk or break somewhere along the rachis, usually close to their bases, and hang vertically downwards to form a skirt of dead leaves around the trunk (Fig. 1). As the older fronds die progressively and hang down, the affected palms go through a characteristic stage where only an erect tuft of wilted, dying leaves are left on them (Fig. 2). In the final stages, all the fronds fall off, or the crown with the remaining leaves get blown over, leaving a tapering decapitated trunk.

The development of young fronds is impaired, when palms survive the infection for some time. The fronds are then reduced in size and number with their rachis shortened. In contrast, in healthy trees the development of the fronds is uniform so that a circular pattern is seen around the stem at the basal region of the rachis, when viewed from below.

Inflorescences. The development of the spathe and inflorescence depends on the rate at which the infection spreads. If the palms collapse suddenly, normal inflorescences in various stages of development become necrotic and undergo rapid decay. If the progress of the disease is slow, necrosis of male flowers usually begins at the tip and spreads gradually towards the base and sometimes at a later stage of development they may wither away completely. The female flowers or buttons are few and poorly developed.

Apex and bud. The activity of the apical meristem is greatly reduced in severely affected trees and later the apex begins to die as shown by the tapering of the trunk just below the top. At this stage the apical region becomes moist and emits a bad odour.

The bud is normal in appearance until the final stages of the disease. When severely affected palms are split longitudinally, the buds show a brown soft rot with a characteristic foul odour, and the bases of the fronds are seen to be brown and necrotic.

Nuts. The reduction of the total leaf area affects the production of nuts which become narrow and elongated in the immature stage and small and distorted when mature. They have a narrow tip instead of the normally rounded one. In section the husk is thicker and marked with dark brown streaks in comparison with the nuts of healthy palms (Fig. 3). The nut diameter and kernel thickness are reduced and shells are often cracked. There is also considerable immature nut fall. Palms showing slow progress of the disease bear moderate clusters, but the nuts are badly distorted. In these cases the lower bunches often hang down because of the breakdown of the supporting frond or rachis.

Roots. The roots are infected; initially the cortical region and then the stele turn brown, later they become friable and disintegrate. As the roots in contact with the soil die back, the palm puts out new roots from higher and higher up the trunk, and sometimes new roots can be seen coming down from healthy tissue through the affected tissue. In contrast, production of new roots in the soil rarely occurs after the palm shows external symptoms of the disease. The roots are affected long before the expression of foliar symptoms, at which stage the former are found to be almost completely decayed.

Bole. The disease tends to spread from the exterior to the centre of the stem, and primary infection appears to take place through the roots, but after the bole is infected, root infection can start from within. Fungal invasion causes decay of extensive areas of the bole, resulting in the formation of large cavities (Fig. 4). Frequently the trunks of such palms fracture at the base and they fall, leaving behind the diseased bole tissue in the ground.

No external or internal lesions were seen in the mid-section of the trunk of affected palms examined.

Stem. There is a characteristic reddish-brown discoloration accompanied by the exudation of a brown viscous gummy substance at the base of the stem. Initially, these 'bleeding' patches appear on several places as parallel vertical streaks (Fig. 5). These soon coalesce, forming a discoloured band round the trunk. These brownish patches extend up to about 1 m from ground level.

The production of a dry rot of internal tissue at the base of the stem is characteristic. Sometimes boring insects get into the affected area and leave a trail of powdery droppings in the area streaked with gum. Transverse and longitudinal sections of the base of the trunk show a light brownish area of rotting tissue marked by darker bands, often with an irregular outline. The edge of the lesion is marked with a distinctive yellow margin, 0.5 - 1 cm wide, showing the perimeter of the decayed area. As the infection advances the infected tissue gives fermenting alcoholic smell and gradually becomes darker in colour.

Prognosis. There is no evidence of the disease affecting young palms in the field, all affected ones being over 50 years old. The progress of the disease appears to be related to local climatic conditions, being rapid in dry areas and slow in wet areas. Infected palms die in 6-30 months in Ambalantota (rainfall 1000 mm/annum) whereas they survive for 5–6 years in Negombo (rainfall 2000 mm/annum). This is similar to the observations made by Vijayan, Natarajan and Krishnamurthy (1973) for coconut wilt disease in Tamil Nadu, India, also suspected to be caused by a *Ganoderma* sp.

FRUCTIFICATIONS

The identification of the disease can be clearly made from the fructifications of the fungus, which have recently been observed, for the first time, occurring naturally on affected trees in the field. The fructifications were found only in areas of the trunk showing dark brown streaks accompanied by the exudation of gum. The first sign of the formation of a sporophore is the appearance of a small white button-like structure, close to the base of the affected palm. This gradually develops into a typical bracket type fruit body, its shape being quite variable. As the sporophore develops, its upper surface turns a rich brown colour, which is usually uneven, loosely zoned and has a shiny lacquered appearance. A characteristic of the sporophore in the early stage of development is the distinct white lip running all around it (Fig. 6). The under-surface is dull white or greenish white and covered with myriads of tiny pores. Frequently, many sporophores are formed close together so that they overlap one another and often fuse to form large compound structures, which sometimes measure 45 – 50 cm across. Another feature of the sporophores is that fresh growth often commences from just below senescent areas so that the old bracket appears to be the stem of the new structure. However, this is easily distinguished on closer examination.

The location of the sporophores on the trunk gives a rough guide to the extent of the diseased area inside the tree. When the tree dies and falls over, the remaining stump is rapidly colonised by the fungus. In certain cases when infected trees show no external symptoms of the disease, except tapering of the palm, the fungus colonises the cut stump and produces sporophores on them when the palms are cut down prior to replanting (Peries, 1974). The sporophores produce large numbers of minute chocolate brown spores, which are shed and cover the surface below them. These spores are well adapted for wind dispersal.

Samples of the sporophores sent to Dr. R. L. Steyaert, Jardin Botanique De Belgique Brussels, Belgium, have been identified by him as those of *Ganoderma boninense* Pat.

DISTRIBUTION

A survey of disease incidence was carried out in a coconut estate, 44 ha in extent, in the southern-most part of Sri Lanka. It was found that 188 palms had been infected by the fungus, and of these 81 palms are already dead. The infected trees were confined to groups and 39 such patches were distributed throughout the estate. This pattern of disease incidence is similar to that of other root diseases (Marsh, 1952; Kable, 1974) where spread of the pathogen by root contact has been proven although experimental evidence is lacking in this case. There is also the distinct possibility of infection starting from wind blown spores.

This survey recorded only the infected and dead palms; apparently healthy trees in the early stages of the disease as well as infected palms not showing the diagnostic fructifications of the pathogen have not been accounted for. Supplies that have been made to replace palms that have succumbed to *Ganoderma* infection have also been excluded. Therefore, the actual figures for disease incidence could be higher than those recorded here.

DISCUSSION

The disease has been observed only on palms more than 50 years old, which is similar to observations made in Malaysia on *Ganoderma* disease of oil palm (Navaratnam 1961, Turner 1965). This indicates that, like *Rigidoporus* sp., *Ganoderma* sp. are weak parasites.

Fig. 1. Dead fronds of affected palms hang vertically downwards to form a skirt of dead leaves around the trunk.

Fig. 2. The affected palms go through a characteristic stage where only an erect tuft of wilted dying leaves are left on them.

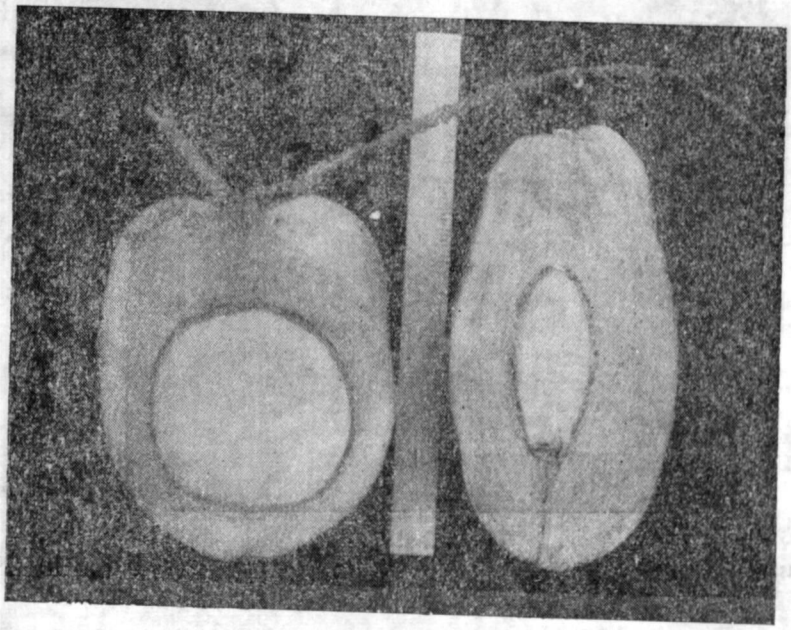

Fig. 3. Longitudinal section of a healthy nut (left) and a nut from an affected palm (right).

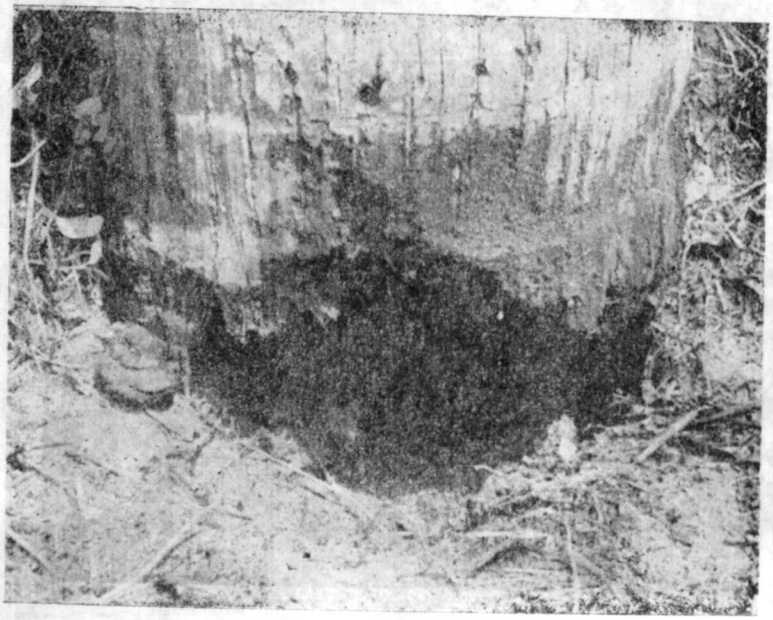

Fig. 4. Decay of base of palm affected by the disease.

Fig. 5. Basal portion of a trunk of an affected palm showing vertical streaks of 'bleeding' patches.

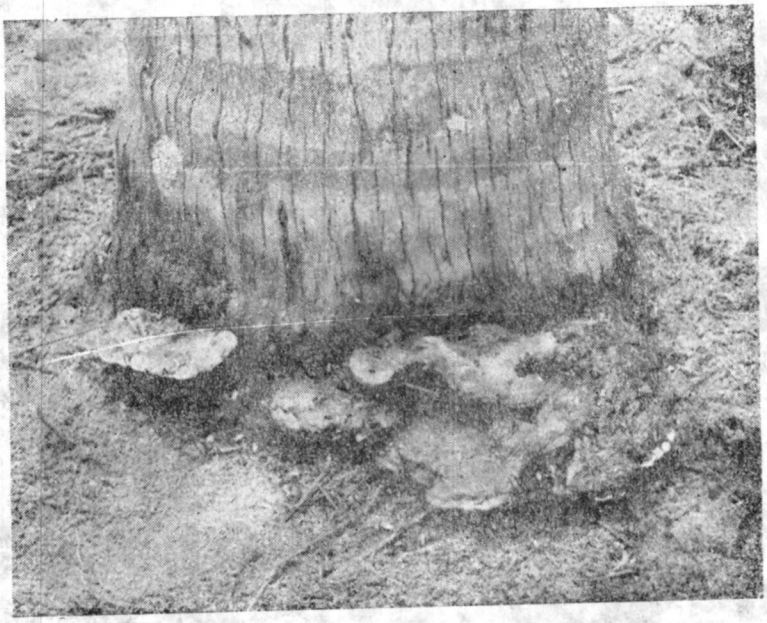

Fig. 6. The distinct white lip on the sporophose at the initial stages and the tiered appearance of the fructification in the final stage of development.

However, unlike *Rigidoporus* sp., they do not affect young, vigorous palms, but only those weakened by age, environmental stress or other factors. It is important to establish whether young plants are resistant to this disease, and steps have been taken to do this. In the meantime the apparent resistance of young palms show the importance of replanting coconut palms when they have passed their prime.

The original infection as in root diseases caused by weak parasites in general, would be expected to be from a food base of considerable size. The spread of the disease, is typical of the distribution of a root disease by root contact. It appears that, after the first palm is infected, the spread of the disease occurs through root contact. But direct experimental evidence is lacking. However, Peries (1974) has observed that spore infection too can be important under certain conditions, specially where trees are damaged or are cut down prior to replanting, when the injuries on the cut surfaces of the stumps can become infected by spores. This is most important in dry areas, where the base of the palm is liable to crack during periods of prolonged dry weather, and the disease can spread over long distances through spore infection of such cracks, particularly as the palms too would be under stress during such periods and their resistance to disease could be expected to be low.

When trees infected by the disease are left standing, they eventually fall over leaving a considerable amount of infected material in the soil. These are potential foci of infection (food bases) for the seedlings supplied to fill the vacancies. This is important as Peries (1974) has shown by artificial inoculation that seedlings are susceptible to infection by *G. boninense* under control conditions. Therefore, it is important to uproot and burn all infected material when trees succumb to this disease.

It was observed that in the estate, where this disease is a serious problem, that vacancies have been supplied in the identical spots where the diseased trees originally stood. This is an undesirable practice, particularly because of the possibility of fresh infection occurring from infected stumps. It is better, from the point of view of disease control, to supply vacancies in between the existing rows. However, when this is impracticable for other considerations, it is essential to remove as much as possible of the infected material of the old palm. We further recommend in such cases that the pits dug for the new palms be heat sterilized as far as possible, prior to planting, by filling them with coconut husks which are later burnt.

Systematic studies are being undertaken to develop control measures for this disease. In the meantime, it is recommended that when a diseased palm is recognised, the extent of damage be assessed and the infected area of the palm be excised and treated with a readily available fungicide, such as 10% copper sulphate in water, if the palm can be saved. However, if the damage is too extensive, the palm, together with as much of its root system as possible, should be uprooted and burnt on the spot. Planting holes, particularly those at the sites of palms that had succumbed to the disease, should be heat sterilized.

The role of the spore has not yet been fully assessed. However, studies in comparable fungal diseases have shown that they can cause stump infection thus providing a base for infection of healthy trees. Therefore, palms should be uprooted rather than cut above ground level, when thinning plantations.

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