THE CURRENT STATUS OF THE PINK DISEASE (*CORTICIUM SALMONICOLOR*) OF COCOA IN PAPUA NEW GUINEA: A REVIEW

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ABSTRACT

The Pink disease caused by Corticium salmonicolor *(Berk.& Broome) is increasing in prominence in some cocoa growing areas in Papua New Guinea. The characteristic symptoms, its distribution, host plants, aetiology and epidemiology are discussed.The need for hygienic cultural treatment of individual tree is stressed.The localization of the conditions of the disease in restricted areas is attributed to the local environment and the prior existence of the pathogen on other host plants. It was once a relatively insignificant cocoa disease but is now becoming more prominent in some parts of Papua New Guinea. It can be locally severe in young cocoa requiring thorough and careful treatment.*

***Key words**: Pink disease, symptoms, host plants, aetiology, epidemiology, distribution.*

INTRODUCTION

Little information is available in the literature on Pink the disease of cocoa caused by the fungus, *Corticium salmonicolor*, a fungus of the subdivision Basidiomycotina. The disease is commonly localized on the bark of host plants.Thwaites first noted the disease in 1873 on coffee tree bark (Schneider-Christians *et al.* 1983 a). It is now reported from many cocoa producing nations.The limited literature available on this disease probably reflects its relatively low incidence in the past and its low impact on cocoa production. However serious economic losses can be experienced, especially on young cocoa trees in localized areas.

There are no reports on economic losses caused by the pink disease in the Pacific Islands.This can be attributed to the fact that those who are involved in cocoa production often treat this disease as of minor economic significance (Henderson 1954, Brown and Friend 1973). In Papua New Guinea the prominence of the disease has increased reflecting the extensive planting that has taken place recently (Anon. 1987). Turner (1987) remarked that the disease has reappeared as a serious problem, particularly in North Solomons, with outbreaks in New Ireland Province, the Bainings and Pomio areas of East New Britain.

This paper reviews some of the literature available and discusses the writer's views on the current status of the disease in Papua New Guinea.

SYMPTOMS

Four different stages of the disease can be observed in the field.The first stage is often referred to as the cobweb stage which relates to the cobweb-like appearance of the mycelium (Fig. 1) and can be hard to detect. A silky whitish mycelium develops on young branches in particular.This growth can be boosted by humid and moist conditions. Under favourable conditions the mycelium can grow at the rate of 8 cm a week (Anon. 1989).

The second stage is called the Sterile Pustule stage which is characterized by the formation of pale pink to whitish pustules about 1 to 8 cm

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Figure 1. **First stage - Cobweb Stage**

Figure 2. **Second stage - Sterile Pustules Stage**

Figure 3. **Third Stage - Corticium Stage**

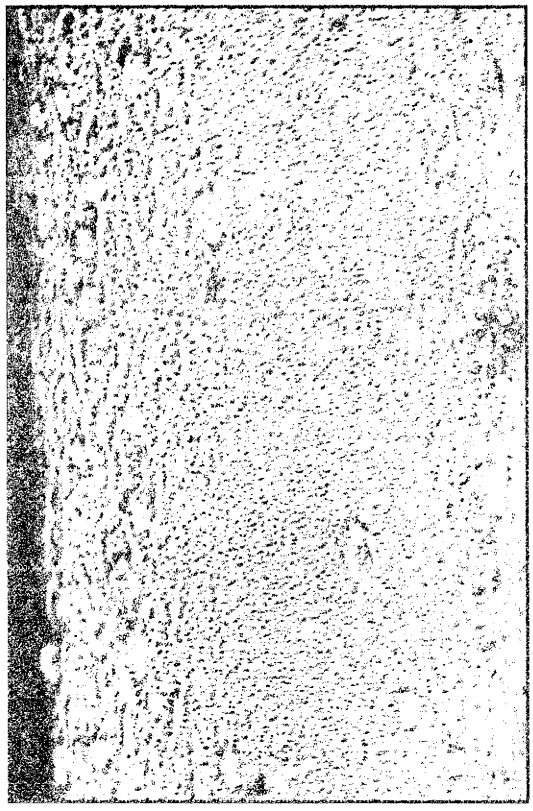

Figure 4. **Fourth Stage - Necatar Stage**

PHOTOS: *COURTESY OF PNG COCOA AND COCONUT RESEARCH INSTITUTE, 1991.*

behind the tips of the advancing hyphae of the 'cobweb' (Fig. 2).The pustules can develop longitudinally with small crack lines on the bark on both the upper and lower aspects (Henderson 1954). Unlike pustules on other hosts, those on cocoa have masses of cells packed one beneath another and do not separate easily in water (Anon. 1989).

The third stage is called the Corticium stage and is characterized by a bright pinkish-orange coloured crust developing on the lower aspects of the infected branches (Anon. 1989 and 1992). The colour makes this stage easy to identify (Fig. 3). With increasing age and intensity of infection the crust can reach a length of up to 2 metres. The crust is formed by the bascidiocarp of *C. salmonicolor* which is hard when touched. The pink colour can fade to white and the crust can crack into small pieces with the onset of drought or due to ageing.

The final stage is an asexual stage called the Necatar stage. It is characterized by orange pustules on the top aspect of the infected branch with small empty crater-like pockets through the bark with a diameter of 1 to 1.5 mm (Schneider-Christians *et al.*1983 b).The pustules contain conidia which can be easily separated and dispersed by water.

The characteristic diagnostic features of this disease includes the dark brown leaves and the pink crust which can remain attached to the tree for a considerable time on the affected branches (Brown and Friend 1973). Pink disease rarely causes death of mature trees but can kill those under the age of five years (Anon. 1983). Internally the wood of the affected branches becomes dry and brown. The leaves at the tip of the affected branches can turn dark brown and die, but commonly adhere to the branch (Fig. 4). The infected branch often shows split bark, withered leaves and dead infected areas (Henderson 1954; Brown and Friend 1973).

DISTRIBUTION

Though it has a wide distribution, extending as far as the Caucasus in Europe and the South Coast of New Zealand (Schneider-Christians *et al.* 1983 a; Seth *et al.* 1978), its main distribution is throughout the humid Tropics (Brown and Friend 1973). Sharples (1936) considered the causative fungus to be native in most areas but has spread to introduced host species such as cocoa.

In the Pacific Islands it was first reported on cocoa in Fiji by Morwood (1956). The causative pathogen was then called *C. litaco fuscum.* Johnston (1960) reported the disease from the Solomon Islands. Schneider-Christians *et. al.* (1983 a) reported the disease from Western Samoa.The disease was reported in Papua New Guinea by Henderson (1954) and Shaw (1963). Apparently, the disease is more prominent in certain areas than others in Papua New Guinea (Turner 1987) although the disease has a wide distribution in the country (pers. comm., visiting Field Officers). The prominence of the disease can be attributed to the local weather patterns, environment and prior existence of the pathogen on other host crops before cocoa was introduced.

HOST PLANTS

There are more than 100 different host plants. Some of the tropical host plants are: *Amherstia nobilie,* soursoap, custard apple, hoop pine, jackfruit, tea, *Cassia sp.*,*Hibiscus*, many *Citrus sp., Crotolaria, Tephrosia* and *Loquat, Gardenia,* silky oak, *Herma*, mango, cassava, pepper, African tulip tree, Cocoa, Rubber, pigeon pea, coffee, citrus, *Gilricidia sp., Leucaena sp.* and Avocado and many unidentified roots. (Henderson 1954; Brown and Friend 1973; Seth *et al.* 1978: Anon. 1991).

AETIOLOGY

Many aspects of the epidemiology and aetiology of the disease are uncertain, with conclusions reached from subjective observations and deductions becoming accepted as proven in the absence of adequate experimentation. The lifecycle of this fungus would appear to be uncomplicated and is largely governed by the weather. Under wet conditions, the basidiospores upon landing on a susceptible part of the plant, particularly young bark, will germinate producing whitish mycelium. Spore germination can be vigorous in the presence of sucrose (J. Dennis

and W. Waine, pers comm.) simulating the effects of honeydew on cocoa bark. In a humid and moist environment, the mycelium can grow at a rate of 8 cm per week (Anon. 1989). With a prolonged wet weather, the mycelium can form pale pink to whitish pustules behind the tips of its advancing hyphae. Given time and with increasing intensity of infection the pinkish pustules can produce a basidiocarp which is often bright pinkish-orange in colour and crust-like. This crust contains basidiospores which are sexual spores and have the potential of initiating a new infection.

In dry weather, the pink colour can fade to white and forms empty crater-like pockets containing basidiospores which maintain viability of the fungus during unfavourable conditions. With excess water, the basidiospores are easily released to complete the cycle.

EPIDEMIOLOGY

The basidiospores of the fungus (9-12 μm x 6-7 μm) develop on the pink crust and may be dispersed by wind or rain (Brooks 1953; Brown and Friend 1973; Schneider-Christians *et al.* 1983 c; Anon. 1989).

It is common for the crust to be sterile and devoid of spores (Sharples 1936; Shaw 1963) but when fertile, the pink incrustation is rather thick and has uniform surface with scattered basidia bearing the basidiospores. When dry, the pink incrustation cracks into larger pieces than the sterile incrustation (Sharples 1936).

Under a cold environment, the fungus can produce conidia on bright orange-red pustules that protrude through the bark on the upper aspects of the infected branches (Brown and Friend 1973).

Luz and Ram (1980) state that wet and humid environment with shade and cool temperatures promote the disease but Schneider-Christians *et. al.* (1983 b) found that a humid environment alone may not promote the disease. This suggest that all environmental parameters may collectively pre-dispose the plant to the pathogen and that if one factor is less favourable the pathogen itself may not become established.

Rao (1974) found that basidiospores of *C. salmonicolor* were only released during or after a rainfall. The disease incidence on rubber and eucalyptus was abundant in areas of high rainfall (Sharples 1936, Seth *et al.* 1978). The qualitative and quantitative dependence of basidiospore release on the amount and time of rainfall certainly exists but light rainfall (<1.5mm) of short duration (< 2 hrs) can result in the greatest number of spores being released (Luz *et al.* 1985). Schneider-Christians *et al.* (1983 b) and Luz *et al.* (1985) found that rain fall may play an important role in the dispersion of the initial inoculum but was not the primary physical agent of the pink disease dissemination during its period of major activity. It is unlikely therefore, that rain is the sole agent of spore dispersal. Hence wind and insects could be the possible agents of dispersal (Simmonds 1931; Rao 1972; Mordue and Gibson 1976).

Basidiospore production can be prolonged if enough moisture and shade are available (Luz *et. al.* 1985). Free water availability is important for spore release from the basidium and for germination of basidiospore (Rao 1972, Schneider-Christians *et al.* 1983 b, Luz *et al.* 1985). Basidiospore production is favourable within the temperature range of 18 to 32°C and that the disease is most prevalent in densely shaded areas, particularly under natural bush shades (Rao 1974).

It has been suggested that young plants are highly susceptible to the pink disease, particularly those 18-30 months old (Anon. 1983). This susceptibility may be correlated with the phase of growth which is marked by considerable development of woody tissue. In the older plants, the disease commonly occurs on young branches undergoing lignification.

CONTROL

Chemical: Chemical and cultural control of this disease are widely practised. In some countries, fungicidal sprays and paints such as tar or bordeaux mixture are used to treat the disease on rubber.

Potassium is shown to have some physiological role in the process of lignification and it has been suggested that susceptibility to pink disease

may be associated with nutritional stress where a low level of Potassium may be particularly critical (Anon. 1983). Lower disease incidence has been reported in plots that were treated with fertilizers than in untreated plots (Anon. 1983). Therefore, the disease may be dependent upon the soil fertility and thus the manipulation of soil nutrients be important in the control of this disease.

In Malaysia, Calaxin was reported to be effective in controlling the pink disease on rubber (Wastie and Yeon 1972). It is often used as a paint which can remain active for up to 4 months. It was found that brush-on formulations incorporating Calixin with natural rubber latex as a binder promised positive results. Yeon and Tan (1974) reported a similar finding in Western Samoa where brushed-on formulation incorporating calixin with natural rubber latex as a binder was used. However, in Western Samoa there have been indications of phytotoxicity despite the control conferred by the treatment (Anon. 1983). Thus Calixin may have disadvantages as a control agent.

In practice, copper gives very good control and is the cheapest fungicide available. In Papua New Guinea, promising results are reported from field trials on young cocoa with Calixin and bordeaux mixture (J. Dennis, pers. comm.). Attempts at using PA injection did not provide a feasible protective measure at Karu Development in the New Ireland Province (Anon. 1989). Curative and prophylactic spraying and painting with copper-based fungicides can be partially effective but does not kill the fungus thus suggesting the need for integrating chemical control with cultural control methods. However many host crop plants of this disease are cultivated in this country and thus eradication of the pathogen is impractical as the pathogen will always take refuge in the various host plants available. It is likely that this reservoir will be the source of future outbreaks of the disease.

CONCLUSION

It is most likely that this disease has existed in all the cocoa growing areas of Papua New Guinea for decades. The low impact of the disease on cocoa production in most cocoa growing areas of Papua New Guinea suggests the need to develop control strategies that require minimal effort for controlling the disease. Such control measures could be the use of biological control systems or the use of moderately resistant genetic material which will suppress and maintain the pathogen's population at low levels at a minimum cost. Future research should be orientated in this direction.

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