SUMATRA DISEASE IN THE CLOVES *(EUGENIA CARYOPHYLLUS)* [1]

Sumatera disease pada tanaman cengkeh di Sumatera [1]

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ABSTRACT

Sumatra disease is not restricted to West Sumatra. Epidemiological and analytical evidence strongly indicate that the primary cause is pathogenic. Presumed causes have been *Phytophthora cinnamomi* and a Rickettsia like bacterium (RLB). The results of pesticide trials and electron microscopy further implicate the role of a RLB. Long term control is likely to depend upon selection for resistance but the high value of the crop might permit expensive pesticide control measures. All clove-growing areas in Indonesia are considered in danger of Sumatra disease. Crop diversification is recommended.

RINGKASAN

Sumatera disease (Penyakit Sumatera) tidak hanya ditemukan di Sumatera Barat. Menurut bukti penyebaran penyakit dan analisa laboratorium penyebab utama ialah patogen. Penyebab penyakit yang diduga adalah *Phytophthora cinnamomi* atau suatu Rickettsia like bacterium (RLB). Hasil dari percobaan penggunaan pestisida dan pemeriksaan dengan mikroskop elektron menunjukkan bahwa RLB tersebut memainkan peranan yang penting. Untuk pencegahan dalam jangka panjang kemungkinan sangat tergantung kepada seleksi untuk ketahanan terhadap penyakit, tetapi dengan harga cengkeh yang mahal memungkinkan cara pemberantasan yang lebih mahal dapat ditempuh. Semua daerah cengkeh di Indonesia dikhawatirkan dapat terjangkit oleh Sumatera disease. Tanaman campuran dianjurkan terutama di Sumatera Barat.

INTRODUCTION

West Sumatra is well-known for its mass decline of cloves. However, in North Sumatra and Lampung there has been a similar decline. In these provinces stem-borers and minimal agronomic practices are a constraint to successful clove culture (Hadiwidjaja, 1956). But the most destructive disorder has been the mass decline renamed by Nutman as Sumatra disease (Waller and Sitepu, 1975).

Between 1968 and 1976 10 000 hectares of cloves were devastated. Its cause has yet to be established and no satisfactory control is known.

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SYMPTOMS

Two major symptom types have been recognized (Bennett, Hunt and Asman, 1979), possibly sharing the same cause. In the first type which we have named *wilt die-back,* leaf fall occurs branch by branch. If the bark is peeled back discoloured bands can be traced in the wood from roots to branch extremities. During the later stages of disease all the leaves on one branch may wilt giving a scorched appearance. Young trees (3-6 years of age) can die in three months and older trees usually within 18 months but may suffer a sudden wilt syndrome at any time following first symptom.

The second major symptom type we have named *senescence die-back* and is characterized by a much slower death (about four years) and leaf loss starting throughout the canopy. There may be a temporary recovery but the tree becomes progressively thinner. Discoloured bands in the wood develop much slower than in wilt die-back.

Senescence die-back is common in the lowlands and wilt die-back in the highlands. Intermediate symptom types exist in different areas, perhaps an indication of environmental influence on the development of a common causal organism.

PRIMARY CAUSE

It has not been possible to consistently correlate with occurrence of disease either rainfall, soil, cultural practices or tree physiology. In certain areas disease started during long dry spells but elsewhere during the rainy season. Disease will often follow a heavy yield but can also affect comparable non-bearing trees*. It is found where there are fertile or poor soils whether trees are neglected or well-maintained. Nevertheless, it is likely that the severity of disease is influenced by the above factors and that hitherto unidentified soil characteristics could predispose plants to infection.

The limited documentary evidence indicates that disease spread from the South (Bengkulu) in the 1930s and continued Northwards through West Sumatra. Spread within localized areas is better recorded particularly in gardens where wilt die-back progresses outwards from primary foci of infection. Thus the epidemiological data point to an entomological or pathogenic cause of Sumatra disease.

* Heavy bearing may equally result from or be the cause of plant stress.

Stem-borers can cause extensive damage and death of clove trees but are not invariably associated with disease. They or other insects could alternatively be vectors of the causal agent. Fungi, bacteria and nematodes found in and around diseased cloves have been investigated. Arising from these studies at L.P.T.I. Solok two presumed causes have been postulated.

The first, a fungus named *Phytophthora cinnamomi* Rands, was frequently isolated from cloves in pre-and post symptom stages. *P. cinnamomi* has killed thousands of Eucalypts (same family as the clove : Myrtaceae) in Australia, symptoms resembling, in part, those of Sumatra disease.

The second is a bacterium (Bennet *et al.*, 1979; Sastraatmadja, 1971). Wherever there are discoloured bands in the wood there are high numbers of bacteria clogging the xylem vessels. As disease progresses so the extent of bacterial invasion increases. A bacterial ooze flows out from the cut portions of the stained wood. Electron microscopy has revealed the bacterium to be a Rickettsia like bacterium (RLB). RLBs have only in recent years been recognized in association with plant diseases.

There has been failure to inoculate either the bacteria or *P. cinnamomi* into healthy clove seedlings, produce disease symptoms and reisolate the causal organism. The reason could lie in juvenile resistance, absence of suitable vectors or inoculation techniques which do not reflect the method of natural infection.

Disease control was sought as a means to determine the cause of Sumatra disease. So-called shotgun trials were set up at four locations using broad spectrum and specific pesticides. Fungicides (Benlate, Copper oxychloride and Difolatan) including phytophthoracides (Aliette and Ridomil), a nematicide and insecticide (Temik), soil fumigation (Formalin) and antibiotics (Streptomycin and Oxytetracycline) were applied intensively by soil drenching, by spraying and by trunk injection (Oxytetracycline).

Evidence of disease control by the phytophthoracides was not significant. Furthermore *P. cinnamomi* is essentially ubiquitous* in West Sumatra having been isolated also from non-clove areas and even the forest.

Apparent response to antibiotic trunk injection was inconclusive (treatments could only be repeated once because of antibiotic supply problems).

* The first identification of *P. cinnamomi* was made by Rands in West Sumatra.

Healthy trees in a diseased locality were trunk injected with antibiotic (Streptomycin + Oxytetracycline) in a new experiment for which there have been three applications since October 1978. The treated trees are still healthy but 50 % of the controls (no antibiotic mixture) are now diseased.

This indirect evidence coupled with the correlation between disease severity and bacterial presence suggest that the RLBs play an important part in the decline of the clove (should they be secondary invaders they are likely to be more damaging than the primary causal agent itself). Their physical presence alone causes ever increasing and irreversible occlusion of the water conducting vessels of the wood. This bacterial phenomenon has been seen in North Sumatra (Asahan and Tapanuli Selatan) where symptoms similar to those in West Sumatra are widespread and in West Java (Sukamantri).

Another experiment has recently begun which incorporates a broad spectrum fungicide formulation (Benlate + Difolatan), the phytophthoracide Ridomil, antibiotics (Streptomycin + Oxytetracycline), and optimal agronomic treatments (chemical and organic fertilizing, mulching and watering during dry spells). The object is to test the capacity of proper fertilizing and tree care to prevent predisposition to disease and to further resolve the bacterial and phytophthoral roles in Sumatra disease.

CONTROL

There is not yet a practical method of control. The high value of the crop makes an expensive control programme feasible. A simplified antibiotic technique might at least allow one or two extra yields, the number of injection sites in a tree being limited. An easier solution would depend upon cultural practices which reduced predisposition to disease.

However for long term control resistant clove material must be found. Seed has been collected from surviving Zanzibar trees (previously diseased but now recovered) and will have to be screened for resistance by appropriate artificial inoculation or natural infection. If the pathogen invades through the roots potentially resistant rootstocks (*Psidium guajava, Syzygium cordatum* and *Eugenia* spp.) should be grafted with cloves and screened.

MASS DECLINE OF CLOVES IN INDONESIA

The aetiology of Sumatra disease remains uncertain. There is localised and potential inter-provincial spread. Its relative unimportance in some regions may be because of the absence of its vector (s) or predisposing conditions. Each major clove area in West Sumatra is now seriously affected by disease. Sumatra disease is considered a threat to cloves throughout Indonesia.

ADVICE TO THE CLOVE GROWER

Farmers are advised to fertilize and properly maintain their gardens. But in Sumatra if land is about to be planted to cloves, we recommend the use of no more than half of it for clove culture, the remainder being used for other crops. Disease has already devastated the clove replants of Koto Anau where in the early 1960s almost every tree was lost to mass decline. Ideally there should be no more planting of cloves in diseased areas until practical control is feasible.

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