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Coconut Wilt Disease of Tamil Nadu

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ABSTRACT

The Coconut wilt in Tamil Nadu appears to be different from the other wilt diseases and differs from the Cadang - Cadang disease of Philippines and (Root) wilt of Kerala in having a short prognosis. The lethal yellowing disease of West Indies and Malaysian wilt are the other two diseases which have some similarities with the Tamil Nadu wilt. But in lethal yellowing and Malaysian wilt stem bleeding does not occur. A new wilt disease reported from Godavari delta, Andhra Pradesh in 1940 resembles the Tamil Nadu wilt in many external symptoms but differs in the absence of rotting of bole and stem and even stem bleeding. The presence of bleeding patches on the stem is a typical symptom of Tamil Nadu wilt which is present only in the Ganoderma wilt of Mysore State. It is probable that the Tamil Nadu wilt and Ganoderma wilt or anabe-roga of Mysore are identical diseases since the symptoms of these two diseases resemble to a very great extent. The role of *Ganoderma lucidum* as the chief incitant of the disease is to be viewed with some reservation as far as the Tamil Nadu wilt is concerned and it would require further study. *Ganoderma lucidum* is only one of the factors in the disease complex.

INTRODUCTION

Since the early fifties a serious wilt disease of coconut was observed in Thanjavur district and east coast. In Thiruthuraipoondi and Pattukottai taluks about one third of the gardens were infected. There is very little information on the disease as it occurs in Tamil Nadu. It is termed as *Ganoderma* wilt. The symptoms, epidemiology and etiology of the disease are described in this paper.

SYMPTOMATOLOGY

(a) *Leaves:* The outer one or two whorls of leaves are seen suspended along the trunk. The leaves appear to be normal in size and colour at this stage. However, in some diseased trees light to moderate bronzing of older leaves is observed. Where the progress of disease is rapid the remaining leaves also droop in quick succession until the spindle alone remains. But in cases where prolonged infection takes place the outer leaves drop away and the subsequent ones are reduced in size and with a shortened spindle that does not unfold itself properly. In the final stages the spindle alone remains with the outer leaves either drooping or having fallen one by one over a period of time.

(b) *Inflorescence and bunches*: The development of the spathe and inflorescence depends on whether the progress of the disease is rapid or prolonged. If the plant dies rapidly normal inflorescence and bunches in various stages of development are seen but they do not develop further. The immature nuts and buttons dry up and are shed in quick succession. If the disease progresses slowly, there is no button shedding. At the time of drooping of leaves, heavily set lower bunches are found hanging due to the breakdown of the supporting leaf petiole.

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(c) *Apex and bud*: The bud is normal in appearance till the death of the tree and does not reveal any internal discolouration. During the final stage the bud tissues are affected by a soft rot which emits a bad odour. The spindle gets easily blown off at this stage leaving a decapitated trunk.

(d) *Nuts:* The nuts which are not fully developed in trees with rapid progress of infection are shrivelled and without kernels. In trees with slow progress of disease normal nuts are produced but in reduced numbers. Most of the trees bear very profusely just before or at the time of expression of external symptoms like stem bleeding and drooping of leaves.

(e) *Roots* : There is extensive rotting and discolouration of the root system with fewer secondary and tertiary and finer roots. The primary and secondary roots are seen distroted in certain places along its length. The roots are watery with a distinct alcoholic smell and appear reddened below the hypodermis and brownish towards the stele. The cortical tissues are in a disintegrated condition, while the stele and hypodermis retain their structure for a considerable time though discoloured. New roots are hardly ever produced after the tree had developed external symptoms of the disease. There appears to be a dieback effect on the roots with progressive reduction in production of new roots as the disease progresses.

(f) *Bole* : There is extensive decay and discolouration of the bole ranging from mustard yellow to dark brown. The discoloured tissues appear watery with an alcoholic smell. But disintegration of tissues is very slow.

(g) *Stem:* A reddish brown viscous fluid exudes from the basal portion of the stem. The bleeding patches begin from the base and extend upto about 5 m as the disease progresses. The discoloured region of the bole extends into the stem in a cylindrical form which gradually decreases in diameter and becomes less pronounced with height. The discoloured cylinder or core is usually confined to the height at which active bleeding symptoms appear on the stem. The peripheral tissues of stem along the central core of diseased tissues are also discoloured in patches and it is from these areas on the stem that bleeding occurs. In the final stages of the disease the stem tissues in the basal 2 m regions are uniformly discoloured and decayed. In the middle region of the stem (3-7 m) the discolouration is usually seen in irregular patches or streaks which are connected with the main infection core. The discoloured strand appear to be disposed in a more or less spiral manner in this region. The affected tissues are lighter coloured being either yellowish orange or light brown. Stem bleeding rarely occurs. Discolouration does not extend to the apical region of the stem and there is a clear healthy region between the apex and discoloured region. However, a soft rot of the bud sets in with loss to turgidity and death of cells resulting from the breakdown of the conducting system.

PROGNOSIS : The disease apparently passes through the following stages from initial attack upto the final wilting of the tree.

Stage 1. Die-black of roots beginning from decay and death of finer roots progresses into the main roots; light bronzing of lower leaves with partial folding of leaflets

Stage 2. Flaccidity of spindle leaves; bronzing of outer leaves more intense; fruit set arrested or unproductive inflorescence produced root decay extends upto the bole.

Stage 3. Extension of decay into the stem; appearance of bleeding patches on basal portions of the stem; drooping of the outer 1—2 whorl of leaves; bunches also hang down due to the breakdown of the supporting petiole; spindle leaves do not unfold properly and appear to be shortened and erect.

Stage 4. Extension of internal stem decay upto the middle region; increase in bleeding patches on the stem; outer leaves drop away; crown is reduced to a few undersized pale green leaves.

Stage 5. The tree dries up gradually with the spindle can be easily pulled out, but the top usually gets blown off. As the tree dries up the stem gets shrivelled.

The time taken for the progress of the disease upto the appearance of bleeding symptoms i.e. stages 1 and 2 could not be determined. Stages 3, 4 and 5 are in most cases spread over 6 to 54 months, the average being two years. In case where death occurs in 6 months, stage 4 is suppressed. In stages 3, 4 and 5, *Xyleborus* sp. is usually found boring into the stem in the region of bleeding patches.

EPIDEMIOLOGY

a) *Soil condition* : The disease is widely prevalent within 5 to 10 km from the coast where the sub soil consists of a mixture of sand and yellow clayey loam which is alkaline and becomes hard on drying. The incidence of the disease is less in loamy and clayey soils of uniform texture to a depth of 120 to 180 cm.

(b) *Weather factors* : Stem bleeding is initiated mostly during the rainy season. Deaths occur during summer.

c) *Spread in infected gardens* : The spread of the disease is rapid in infected gardens. The annual rate of spread was from 0.2 to 4.8% and intensity was from 0.8 to 25.6%. The disease appears to spread from particular foci of infection.

d) *Age of the trees* : Trees of 10-15 year old are most susceptible. Some trees may survive the initial attack and live for a number of years. Fresh attacks on trees beyond 30 years are rare.

PROBABLE CAUSES OF THE DISEASE

Unfavourable soil conditions and lack of soil moisture aggravate the disease. Weakening of the tree by unfavourable soil conditions, lack of soil moisture and nutrients and insects lead to decay of roots and *Ganoderma lucidum* gains entry and causes extensive damage to the internal tissues. The fructifications of the fungus is seen on living diseased trees which have been wounded and also on stumps of dead trees. The etiology of the disease is yet to be pin pointed.

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