Kerala Wilt

In India, Kerala Wilt is also called Root(wilt), Wilt(root) or Root Rot. However, as long as the etiology of the disease is unknown and root rot may be a secondary effect (Lal, 1968), it is better to name this disease after its locality of origin.

The disease was known in the State of Kerala, India, since 1882. By the end of the 1960s it had spread over about 250,000 ha of coconut plantations, or about one third of the total area under coconuts in that State. The disease occurs in the central and southern districts and spreads slowly to the North and South. The damage caused by the disease is estimated at 340 million nuts annually. The spread of the disease in Kerala is not continuous. New foci of infection may be observed at some distance from the main diseased area. Pockets of healthy coconuts may be found throughout the diseased area (Lal, 1968). Palms of all ages are affected. In the State of Tamil Nadu along the east coast of India, the disease was also observed after the cyclones of 1952 and 1955(Vijayan, et al., 1973a). In this State, the disease is often accompanied by Basal Stem Rot *(Ganoderma)* confusing the symptomatology.

The general yellowing and drooping of the outer whorls of leaves and flaccidity of leaflets has been indicated as the most pronounced symptoms of the disease. Snehi Dwivedi et al. (1979) made detailed observations on a large number of diseased and healthy palms. The results of this study showed that yellowing, necrosis and flaccidity were present in healthy as well as diseased palms, flaccidity occurring to a greater extent in diseased palms. Varkey et al. (1979) observed that foliar yellowing both in healthy and diseased areas is due to nutrient deficiency and that the same could be corrected by either foliar sprays of 2 per cent $MgSO_4$, 1 per cent $FeSO_4$ or by the basal application of $MgSO_4$.

The first symptom they observed, was deterioration of the spear, indicated by whitening and softening of the leaflets. Soft leaflets of the spear were whitish-brown to pale-green. Round to rod-shaped necrotic spots were prevalent along the margins. The leaflets were usually rotten at their tip margins. Close observations of the yellowing pattern of leaves in the middle and outer whorls indicated that the leaflets had interveinal chlorosis followed by marginal necrosis. The chlorosis starts from the tips and advances towards the bases of the leaflets. Sometimes scattered yellowing was seen. In leaves of outer whorls where yellowing was in an advanced stage, interveinal yellowing was not clearly visible. The interveinal yellowing of middle leaves is similar to symptoms of magnesium and manganese deficiency; however, the application of magnesium sulphate and manganese sulphate brought partial recovery only. Healthy palms growing close to diseased palms did not show the symptoms. Usually, mature leaves of diseased palms had round holes towards the margins of the leaflets, which were caused by *Myllocerus curvicornis F.* A severe attack by this insect on healthy looking palms, particularly seedlings indicated a latent stage of the disease.

Advanced necrosis and further discolouration are followed by the complete drying of the leaflet. Often the whole leaf shows bending and curving. Because fewer leaves are produced on affected trees, and because leaves fall prematurely, only a few leaves are left on the tree in an advanced stage of the disease. Leaves developing at this stage may be somewhat stunted (Maramorosch,1964). The growing point remains unaffected.

No clear symptoms have been observed on the inflorescences. They may grow weaker and produce fewer buttons and nuts. Buttons and small nuts may be shed. Maramorosch(1964) reported that the mature nuts of diseased trees had a normal size, but other writers, like Subba Raja and Ahamed (1976) reported a remarkable reduction in nut size. The endosperm of the nuts is thinner than usual and uneven in thickness and when dried remains flexible. The husks of the nuts are thinner than normal and the fibers weaker than in normal nuts. Ramadasan et al. (1971) observed that trees infected by the disease before flowering may not flower at all. Younger plantations in the infected areas also deteriorate faster than older plantations.

Progressive deterioration and drying up of the roots and rootlets from the tip backwards was observed by Maramorosch (1964), but not in all palms investigated. He did not observe discolouration of the roots having been reported by several other scientists, either. Subba Raja and Ahamed (1976) observed brown longitudinal streaks in the roots and rootlets. The disease is not lethal but if accompanied by other diseases such as fungal leaf diseases, this may result in death of the affected palms. No recovery of infected palms has been reported yet.

The sometimes contradictory descriptions of the disease symptoms may probably be explained by assuming that physiological disorders resulting from the disease may differ according to variety of palms and growing conditions, such as mineral nutrient availability and water availability.

Changes in the carbohydrate content in infected palms were studied by Mathew (1977). He found that total, reducing and non-reducing sugars were significantly higher in the leaves of infected palms. But a depletion of sugars was found in the roots, indicating a possible block in the translocation process. Starch and carbohydrates were present in the leaves and roots of infected palms at a reduced level. A decrease in total carbohydrates and starch content may be due to the combined effect of a retarded rate of net photosynthesis and an increased rate of respiration. Michael (1978) studied the changes of respiration rate and nut yield in diseased palms. Leaves and roots of diseased palms showed an increase in respiration rate. He suggested that the higher rate of respiration in the leaves might be due to better availability or accumulation of total and reduced sugars and inorganic nutrients. A negative correlation of 0.54 was recorded between respiration rate and nut production.

Michael (1978) found that the moisture content of the leaves was higher in the palms growing in the diseased tract, but Lal (1968) mentioned that earlier studies had shown that the rate of transpiration of the leaves of diseased palms was higher than of the healthy palms. The rate of water uptake by the root tissue of diseased palms was 35 per cent lower than that in healthy palms. A study of the rate of water uptake in the stem just above the level of the bole revealed that the uptake was 5-30 per cent less in diseased palms. It was concluded that these symptoms indicated a slow development of internal water deficit in diseased palms.
Studies conducted by Mathew (1981) confirmed that there was a significant increase in the transpiration rate in all leaves of the diseased palms. An excess of transpiration often leads to a progressive decrease in the water content of leaf tissues. But this could not be applied to the situation as regards Kerala-Wilt affected palms, showing an increased water content in the leaves.

The frequency of cells and stomata per unit area of leaf was higher in diseased palms. Despite differences in cell and stomatal frequencies, stomatal index did not differ significantly between healthy and diseased palms Mathew suggested that structural changes in the leaves characteristic of plants grown in dry habitat may be the consequence of severe water deficit which continuously develops within the leaves of disease-affected palms.

The causal agent of the disease is still unknown but there are indications that a virus may be involved. Since the 1950s several scientists suspected the viral nature of the disease but the final proof has yet to be presented. Summanwar et al. (1969) found rod-like structures in infected palms, suspected to be a tobacco mosaic virus. Shanta et al. (1975) examined root and leaf samples by means of electron microscopy. They found tubular particles in tender, growing tissue of healthy and diseased palms. Ultracentrifugation resulted in the breaking up of long particles into shorter sizes. Where Summanwar et al. used this technique, it was suggested that the tobacco mosaic virus-like organisms observed by them and the broken tubular structures observed by Shanta et al. were the same.

Maramorosch and Kondo (1977) examining diseased coconut material by means of electron microscopy, detected icosahedral virus-like particles in the epidermis and ground parenchyma cells. These particles appeared in small, isolated aggregates. Since they were sparse and comparatively small, their detection was difficult. The approximate size of the particles was 56 *nm* in diameter. Parthasarathy (1978), however, was of the opinion that these particles are actually transverse to slightly oblique sections of plasmodesmata, a portion of the cell wall, and some ribosomes.

Mycological studies showed that *Rhizoctonia solani* and *R. bataticola* were associated with excessive root damage of diseased trees. Lily (1975), when examining the longitudinal sections of coconut roots in connection with the investigations on Kerala Wilt observed that the vascular tissue of the roots contained sporangia-like bodies. The fungus was identified as *Gongronella butleri (Lindn.)*. However, the fungus attack may be secondary, as it appears after the development of the leaf symptoms. Lily (1981) indicated that *Fusarium equiseti* (Corda) Sacc. and *Cylindocarpon effusum* Bugn. are also pathogenic to the coconut root system. In laboratory tests he showed the pathogenicity of *C. lucidum* a fungus isolated from coconut roots as well. Although browning of roots prior to foliar symptoms has been reported, and might be associated with fungus attack, it has not been observed in all root investigations. Joseph and Jayasankar (1973) suggested that the vascular browning could result from conversion of phenolic into melanin-like compounds.

Sumathykutty and Patil (1983) succeeded in stimulating new root initiation in diseased palms by applying growth hormones and phenols in lanoline paste to the base of the stem close to the bole after debarking the same at ground level. The points of application were then covered with soil which was kept moist during the dry season. Root initiation was observed after one year of treatment. The treatment with IBA 500 ppm + phenols 400 ppm yielded the best results, followed by NAA 500 ppm + glutanic acid 500 ppm.

Studies of soil conditions have indicated that in the diseased areas the soils contain larger amounts of moisture, organic matter and nitrogen than the soils in healthy areas. Diseased soils were poorer in Ca, available K, total exchangeable bases and had a lower pH (Verghese 1966; Cecil (1975) Maramorosch(1964) could not find any evidence to substantiate any relationship between disease outbreaks and conditions of poor drainage, high water table, poor nutrient supply, high acidity and generally poor growing conditions having been associated with the disease. But Lal (1968) stated that the disease was more intense in poorly aerated land and that water stagnation and high water table seem to play an important role in the initiation and spread of the disease. The rate of spread was also higher after flooding.

The mineral nutrition in affected coconut palms was thoroughly investigated by Pillai et al. (1975) in a nutritional survey, collecting soil and leaf samples of perfectly healthy and diseased palms covering all the major coconut growing soil groups of Kerala State. The chemical analyses of these samples confirmed the accumulation of NPK in diseased palms. The nitrogen status of soils of disease-affected tracts was higher than that of healthy soils. Among the secondary nutrients, deficiency of S was

tis typicus Dist. possibly being the vector. Cowpea, *Vigna sinensis* is suspected to be a host plant of the pathogen (Holmes, 1965) and many transmission trials have been carried out on cowpea seedlings. The symptoms produced on cowpea seedlings are malformation and sometimes necrosis of the trifoliate leaf. But the behaviour of the cowpea seedlings seemed to be very erratic, influenced by temperature and differing between varieties.

Transmission trials by mechanical inoculation between coconuts produced flaccidity of leaflets on coconut seedlings both in the field and under controlled conditions. Shanta et al. (1972) carried out a series of experiments to study the infectivity of the soil. Cowpea plants became diseased when grown in soil taken from a spot close to a diseased palm, or in sterilized soil water with leaf or root sap of diseased palms, or in soil to which diseased roots were added. Treatment with pentachloronitrobenzene destroyed infectivity. Air-drying for more than a week or fine grinding did not destroy infectivity. It was concluded that the pathogen is soil transmitted and that a biological vector does not seem to be essential to the transmission. It was suggested that either fine particles of infective clay or fungal zoospores acting as vectors are carried by water.
Sasikala and Gopinathan Pillai (1978) carried out transmission trials with 170 different species of test plants and cultivars belonging to 30 families. The plants were raised in sterilised pots in an insect-proof screen house under natural conditions of temperature and light. They employed mechanical inoculation with leaf and root extracts, partially purified preparations, root exudates and soil suspension. Soil transmission, leachates, and insect feeding, using *S. typicus* were conducted on more than 22,000 plants. *Lycopersicon esculentum* cultivar San Marzano, and *Physalis minima* showed a very low percentage of infection when mechanically inoculated with diseased leaf extracts. Plants which were reported as suspects earlier did not show any symptoms in these trials. Cowpea, reported as an indicator host earlier, also failed to show symptoms consistently. Mathen et al. (1976) grew coconut seedlings in pots with soil taken from areas close to diseased palms. After three years, having transplanted the seedlings into larger pots, containing a mixture of soil and roots of infected palms in equal portions, foliar symptoms began to appear. No foliar symptoms were discernible in seedlings which had not been transplanted. No completely effective control method has been developed as yet. However, if soil conditions could be of influence, improved cultural practices could help to contain the disease. Although improvement of the general situation in infected coconut gardens has been achieved through improved management, the disease has not been stopped by it.

Lal (1968) reported a low disease incidence in palms growing on a waterlogged clay soil that is comparatively rich in nutrients and is highly acid. He suggests that these soil conditions may keep the palms free from disease. Rawther and Pillai (1972) studied three age groups of palms, viz. non-bearing palms, bearing palms up to the age of 15, and those of 15 and older to verify the relationship between age and disease incidence. Among the seedlings observed, Dwarf x Tall (DXT) (natural cross) and Green Dwarf (GD) were completely disease free, whereas Tall x Dwarf (TxD) and Orange Dwarf (OD)

evident. Imbalances in cationic ratios such as K/NA, K/Mg, K/(Ca+Mg) and K(Na+Ca+Mg); and anionic ratios such as P/S and N/S were quite apparent. Similarly, the deficiencies of Fe,Mn, Mo and Zn particularly in diseased palms were other findings of importance. Cecil (1975) found that the percentage of Ca-saturation of soils from disease-affected areas was considerably lower than that of healthy areas and that diseased palms had significantly lower Ca and Mg levels than healthy palms. Mathew and Varkey (1976) found higher Ca and Mg contents in soils of diseased tracts than in healthy tracts. Analytical data of leaves of diseased and healthy palms did not show variations in Ca content. Total magnesium content in the leaves was higher in the case of laterite and loamy but not in the case of sandy soils. They concluded that the status of Ca and Mg of soil and leaf could not be considered a factor for the incidence of the disease.

Nematological studies or bacteriological studies have not revealed any evidence of these pathogens being involved in Kerala Wilt either. A higher percentage of soil and root samples from the diseased localities yielded *Radopholus similis* nematodes as compared to those from healthy areas and a characteristic streaming movement of a bacterium tentatively identified as *Pseudomonas sp.* in the vascular tissues of the roots of diseased palms (Shanta and Radha, 1975). But neither of these two organisms has been further associated with the disease.
A possible association of river water with the spread of the disease has been suggested by several scientists. Gopinathan Pillai et al. (1973) who made a survey of the spread of the disease in Kerala, support this view as the coconut palms on the banks along the course of all rivers from Trivandrum to Trichur were invariably heavily infested. The banks of the lower tidal parts of the rivers were still disease free. The first outbreaks of the disease in Kerala in 1882 occurred after heavy floods. They stated that in general the disease incidence was less in laterite soils than in other soils. There are instances of individual diseased palms having stood in laterite areas for many years without any fresh outbreak in the neighbourhood.
Further observations by Gopinathan Pillai et al. (1980) showed that the spread of the disease was most rapid in palms growing on alluvial soils, and of decreasing intensity in sandy-loam and sandy soils. The spread was slowest in palms growing on laterite soils. Lal (1968) had also observed a slower spread among palms growing on laterite soil than on other soils.

In general, the spread is erratic and irregular irrespective of soil conditions and occurs in jumps. The span of spread can be up to about four kilometers from the nearest source of infection. In one plot where diseased palms were removed as soon as they had been observed, once a fresh outbreak occurred only after seven years without any disease incidence.

The transmission mechanism of the disease has not yet been determined. It is generally accepted that the transmission is soil-borne, but the pathogen is also suspected to be air-borne, the banana lace wing bug, *Stephani-*

recorded 6, 17, and 24 per cent disease incidence respectively. Among the young bearing palms DxT was found to be the most resistant with a disease incidence of 2 per cent only. TxD, OD and West Coast Tall (WCT) were diseased in varying degrees ranging from 6 per cent in OD to 65 per cent in WCT. Among adult palms GD and DxT showed better tolerance than others, the incidence percentage being 8 and 13 respectively. TxD and OD ranked in succession, whereas WCT was found to be highly susceptible, with an incidence percentage of 63. The overall performance indicated that DxT was the most tolerant, following by GD and TxD. OD was partially resistant and WCT was highly susceptible. DxT also recorded the highest yields followed by TxD. OD and GD were moderate yielders. WCT produced relatively low yields in both healthy and diseased groups. In WCT the yield decreased sharply with the progress of the disease, whereas in the hybrids and the dwarf varieties though there was a considerable reduction in yield due to disease, the loss was not as heavy as in WCT. Bavappa (1975) reported a disease incidence of 37.5 per cent for WCT, 22.9 per cent for OD, 9.3 per cent for DxT, 11.4 per cent for TxD and 10.4 per cent for GD. Screening of many local and exotic cultivars is still going on but no cultivar or hybrid has shown the desired degree of resistance or tolerance as yet.

Infection by a pathogen may result in the release of free phenols which are readily converted into toxic oxydation products. Joseph and Jayasankar (1973) tried to correlate the phenolic content of the roots in the healthy palms to resistance to the disease. TxD, which is compartively more resistant than WCT had lower phenolic content than WCT. The natural cross dwarf that was found to be the most tolerant to Kerala Wilt (Rawther and Pillai, 1972) had a much lower phenolic content than TxD and WCT. The results suggest that tolerance or resistance is correlated to low phenolic content. Low phenolic content may be related to a higher rate of oxydation of the polyphenols. In fact, incidence of disease was associated with a fall in the concentration of total phenols. It was concluded that further studies were required to come to more definite conclusions with a view to the correlation of phenolic content and disease resistance.

Apart from the development of resistant varieties or cultivars of coconut, improved management and thorough elimination of diseased palms, as soon as disease symptoms are observed, may reduce the disease incidence and the damage caused by the disease to an extent which may be economically tolerable.