ETIOLOGY OF THE ROOT (WILT) DISEASE OF COCONUT PALM

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The root (wilt) disease of coconut (*Cocos nucifera* Linn.) prevalent in Central Kerala was significantly manifested after a severe flood in 1882 when the land was waterlogged for a long period. The disease seems to have made its appearance from more than one foci of infection in distant areas and since then has been spreading slowly and steadily. Menon (1963) and Lal (1966) have reviewed the work done on the various aspects of this complex disease.

Symptoms: The characteristic symptom of the disease is a slow wilting of the foliage. Flaccidity and ribbing of leaflets accompanied by an abnormal bending of petiole is an early symptom. Yellowing of the outerwhorl of leaves and marginal necrosis of leaflets contribute to complete the disease syndrome. Rarely, yellowing and drying of intermittent leaves and abnormal shedding of buttons and immature nuts may precede flaccidity of leaves. Sterility of pollen and necrosis of spadix also occurs. Production of nuts is adversely affected both in quality and quantity. As the name of the disease signifies itself, rotting of roots is an important feature.

Development of the symptoms is very slow. Generally a disease affected palm continues its life for more than 15 years after the first appearance of symptoms. Young palms at the pre-bearing and early-bearing stages (6–15 years) are more susceptible to the disease than the older palms of 35–50 years. Progress of disease is faster in young palms, which at times, succumb within 3–4 years after the onset of initial symptoms. Infection on palms younger than 4 years is rare.

Quantitative studies on the foliar symptoms of nearly 2,000 palms, young as well as adult, in a heavily infected garden in sandy loam soil reveal that flaccidity of leaflets appears as initial symptom in 4–9 year old palms. In a few cases foliar necrosis and yellowing occurred independently of flaccidity. On the other hand in adult palms (35 years and above) in majority of cases all the symptoms are manifested (Table 1).

Table 1 : Percentage of palms manifesting different symptoms

	Total number	Flacci-dity	Yellow-ing	F Y	Necro-sis	F N	F Y N
Adult palms	1592	1·3	7·6	22·9	0·08	1·3	44·9
Young palms	365	52·3	2·4	4·6	3·7	5·4	4·2

The low incidence of yellowing of leaves in younger palms and the occurrence of yellowing in more than 7·0% of the adult palms in the absence of the other symptoms is perhaps suggestive of a condition associated more with nutritional

factors than of a disease symptom. This view is strengthened by the fact that during a 3-year period the percentage of palms exhibiting foliar yellowing remained almost steady in plots V, VI and VII maintained under normal agronomic practices, whereas those supplied with higher doses of NPK fertilizers in plot I showed an increasing trend probably due to an imbalance created in the K/Mg ratio. (Table 2). Tissue analysis of palms manifesting foliar yellowing and those without yellowing indicate imbalance in K/Mg ratio, 18·3–27·1 in the former and 9·7 in the latter.

TABLE 2 : Disease incidence and foliar yellowing in an infected garden

Plots	1954		1955		1956	
	% disease incidence (a)	% Foliar yellowing (b)	(a)	(b)	(a)	(b)
I	59·0	5·2	59·3	5·6	54·0	10·8
V	67·0	7·6	74·5	9·7	72·0	7·3
VI	69·0	6·7	71·2	8·1	76·1	7·2
VII	69·0	6·7	71·2	8·1	76·1	7·2
VII	69·0	10·3	71·1	10·3	70·0	10·8

Studies on root rot in relation to foliar symptoms confirm the earlier observation that root rot is a secondary symptom and that the production of new roots is higher in the disease-affected palms than in healthy ones. (Table 3).

TABLE 3 : Percentage root rot in relation to foliar symptoms and frequency of occurrence of *Rhizoctonia*—1/8th sector of the bole

Foliar condition of palms	No. of palms studied	Total No. of roots	% Young root	% Root rot	No. of palms having *Rhizoctonia*
Healthy	4	135	35·6	66·6	0
Flaccidity	4	253	69·0	63·4	1
Flaccidity & Necrosis	4	126	73·0	90·5	2
Necrosis & Slight Flaccidity	4	289	51·6	90·0	2
Healthy	24	521	—	—	4
Diseased	31	798	—	—	14

Distribution and spread : The disease occurs in all types of soils, from the coastal sandy tract to the laterite beds in the upland hilly area. Isolated diseased gardens within the healthy tract, miles away from the diseased belt as well as healthy pockets within the heavily diseased area do occur at many places. Severe disease incidence observed along the banks of rivers has been traced up to the high ranges.

Studies on the pattern of spread of the disease currently in progress in different types of soil, in healthy pockets having single of focus infection indicate that the

disease spreads faster and in an irregular pattern in gardens subject to flooding and inundation. Studies based on appearance of symptoms of disease during the course of three years have revealed that there was some increase in alluvial, sandy and reclaimed soils but none in laterite soil. In an infected plot in sandy loam soil the rate of spread ranged from 2 to 7%.

Soil factors: The nutrient status of the soils from healthy and diseased areas essentially had no differences, except that the diseased soils had comparatively lower values for calcium, available potash, total exchangeable bases and pH. Nevertheless, results of a series of manurial trials using major and minor nutrients, sulphur, calcium, magnesium and several activated fertilizers proved that these soil amendments were neither able to improve the foliar condition of diseased palms nor prevent the incidence of disease, although in general all the treatments increased the yield of nuts.

Poor soil aeration and waterlogged conditions appear to have profound influence on the development of disease. Intensity of disease is high in heavy reclaimed soils, more so in extensive gardens than on bunds. Similarly disease incidence is severe in sandy and sandy loam tracts where high water table and frequent inundation prevail than in laterite soils.

Results of a field experiment conducted in a garden where severe foliar yellowing and stunted growth were associated with high water table revealed that the provision of drainage channels significantly improved the growth of palms and completely cured foliar yellowing within a period of 15 months. Under pot culture conditions waterlogging resulted in accumulation of soluble iron, manganese and ammoniacal nitrogen while the uptake of major nutrients was inhibited.

The changes following flooding or factors associated with the prevalence of high water table in the chemical and biological status of the soil environment may constitute favourable conditions for the onset of the disease. The possible role of a biological entity as the determining fact in initiating the disease under conducive environments seems probable.

Biological Factors

Fungal aspect: Earlier work indicated that *Rhizoctonia solani* Kuhn. and *R. bataticola* Butl. were associated with rotting of roots in diseased palms, former being more specific and pathogenic than the latter. Frequency of occurrence of *R. solani* was higher in the diseased palms, more so in the advanced stages of disease as compared to that in the healthy plants (Table 3). Based on the view that root rot as well as the role of *R. solani* are secondary in the incidence of root (wilt) disease, infection trials were conducted with this fungus to study the predisposing factors for its infectivity. Waterlogged soil condition inhibited root growth and favoured fungal infection (Menon *et al.*, 1952), but failed to initiate the foliar symptoms of disease. Similarly primary inoculation of palms in the field with the suspected pathogen (virus?) increased pathogenicity of *R. solani* which was inoculated subsequently. In the controls which were not

inoculated with the suspected pathogen infectivity of the fungus increased with the onset of natural incidence of disease (Table 4). Identical results were obtained on inoculation with *R. solani* on roots of palms infected with the pathogen (virus?) under controlled conditions.

TABLE 4 : Infectivity of *R. Solani* in relation to the suspected pathogen

No. of palms		1960		1961		1962		1963		1964	
		a/b	c	a/b	c	a/b	c	a/b	c	a/b	c
Inoculated with the suspected pathogen	24	105/105	100	127/118	92.9	68/66	97.0	136/133	97.8	56/56	100
Uninoculated control	12	59/18	30.5	70-29	41.4	39/26	66.6	59/40	67.7	32/24	75

a—No. of roots inoculated with *R. solani*.
b—No. of roots infected.
c—Percentage infection.

Evaluation of the soil and rhizosphere microflora of coconut from healthy and diseased areas has not, so far, revealed the association of the disease with any organism other than *Rhizoctonia* spp. A general reduction in the rhizosphere microflora in the diseased palms occurred, although it varied with soil type and depth of sampling. Radha and Rawther (1959) indicated that the rhizosphere fungi are at the minimum during periods of heavy rainfall and high water table which showed a rising trend when the water table receded (Table 5). Reduction in the microbiological activity in the soil in the root zone of diseased palms was consistently noticed in sandy loam, alluvial and laterite soils (Table 6).

Preliminary studies suggest that the root exudates from some apparently healthy palms and those exhibiting very early symptoms of disease are favourable for the growth of the fungus *in vitro*. Microbiological activity at the root zone of these palms was found to be lower than in healthy palms.

Virological : Nagaraj (1956) reported the possible involvement of a sap trans-

TABLE 5 : Rhizosphere fungi during different seasons

Depth of sampling in ft.	April		June		August	
	R.F. in ten thousands per g. soil	% fungal colonizers in roots				
1	4·4	80	1·9	69	13·7	108
2	10·4	35	0·14	60	9·7	84
3	2·94	85	0·27	—	4·2	85
4	1·0	40	—	—	—	—

(a) During June the water-table was above 4 ft.
(b) In August the water-table was at 4 ft. level.
(c) No samples were collected below the level of the water-table.

TABLE 6 : Bacterial population and respiratory Co_2 in soils at the base of healthy and diseased palms in sandy loam soil

Condition of palms	No. of palms studied	No. of samples	Bacterial flora in lakhs/g soil	Co_2 in mg. per g soil
Healthy	12	48	47·6	138·5
Diseased	12	48	13·2	73·5

missible virus in this disease. He also suggested that *Stephanitis typicus* Dist. is a probable vactor of the pathogen. The symptoms of the disease were consistently reproduced on artificial inoculation of palms (of different age groups from 4 to over 50 years) by the abrasion method and by feeding infective adults of *S. typicus*. Introduction of root sap from diseased to healthy palms by direct root connection also produced disease symptoms. Flaccidity of leaves appeared within 8–12 months after the first inoculation.

Transmission trials were conducted under insect proof conditions on 20 potted, 2-year old seedlings by different methods. Within four years five seedlings out of the six inoculated by the abrasion method and two of the six seedlings fed by infective *S. typicus* showed paling, flaccidity and slight stunting of leaves. Neither root rot nor foliar necrosis developed in any of the infected seedlings (Table 7).

Shanta and Menon (1960) reported cowpea (*Vigna sinensis* Endl.) to be an indicator host for the pathogen, producing 'vein clearing' and malformation of leaves. The symptoms were produced in six to ten days old plants within seven days. Cowpea plants raised at the base of the infected coconut seedlings, 2-1/2 years after the development of foliar symptoms showed diagnostic symptoms of disease in four of the sap inoculated seedlings. Soils from the base of diseased palms from the field, clay and silt fractions, gave positive infection on cowpea while those from healthy palms induced no symptoms.

Serological trials using different preparations of infective material from both coconut and cowpea yielded no conclusive results so far.

Physiological and biochemical considerations: Considerable changes in the water economy of the diseased palm have been noticed. The fresh wt./dry weight ratio and the loss of wt. per minute per unit area of leaf tissue are higher in the diseased palm while absorption remain low. Preliminary studies indicate that the permeability of root tissue of diseased palm is deranged.

The organic and inorganic contents in the root sap of diseased palms are higher than in healthy. Accumulation of non-protein nitrogen, inorganic phosphorus and reduction of protein nitrogen has been noticed in the leaf tissue of diseased palms. Considerable reduction in C/N ratio of both leaves and roots of diseased palms was also indicated. Increasing trend in free amino acids, tannins and RNA content of tender leaves of diseased palms was observed. It is evident from the foregoing account that gross derangement in the physiolo-

TABLE 7 : Results of transmission trials on coconut conducted in the insect proof house

Treatment	Seedling No.	Condition of seedling in				
		1960 June	1961 Jan.	1961 June	1962 Jan.	1962 June
Sap inoculation (SI)	1	H	H (d)	H (d)	DE	DE
	2	H	H	H	H	H (d)
	3	H (d)	H (d)	H (d)	DE	DE
	4	H	H (d)	H (d)	H (d)	DE
	5	H	H (d)	H (d)	DE	DE
	6	H	H (d)	DE	DE	DE
Insect transmission (IT)	1	H	H	H	H (d)	H (d)
	2	H	H	H (d)	H (d)	H (d)
	3	H	H	H	H (d)	H (d)
	4	H	H	DE	DE	DE
	5	H	H	H (d)	H (d)	DE
	6	H	H	H	H (d)	H (d)
Control	1	H	H	H	H	H
	2	H	H	H	H	H

H : Healthy. H (d) : healthy (doubtful) that is, slight symptoms of flaccidity or paling noticed.
DE : Disease early stage.

gical processes of the palm leading to improper utilization of nutrient ions is associated with the disease.

Disease resistance : Investigations initiated from 1950 to study the heritability of disease and disease resistance of indigenous and exotic varieties have not yielded any positive results so far. Seedlings raised from diseased parents were more susceptible than those from healthy parents. Progenies of healthy parents in the diseased tract as well as hybrids from a healthy tract behaved almost similarly (Table 8). Of the 69 healthy parents in the diseased tract 32 contracted the disease later.

Out of the 20 exotic varieties under trial, those from the Andaman, Laccadive, Philippines, New Guinea, Java, Cochin-China, Ceylon and Strait Settlements have already contracted the disease.

DISCUSSION

The possibility of a virus or a virus like pathogen being the casual agent of the disease has already been surmised (Menon, 1963; Lal, 1966 and Holmes *et al.* 1965) although no direct evidence on the nature of primary pathogen is yet available. Nevertheless, the accumulated data on the infectious principle justifies a discussion on its possible role in the disease complex. According to Allen (1966) "the symptom of disease in plants are largely the results of altered rates of host activities and altered levels of host metabolites". Based

TABLE 3 : Disease incidence in progenies of healthy and diseased parents

Percentage		No. of progenies	1960	1961	1962	1963	1964	1965
				% disease incidence				
Healthy	Open Pollinated in diseased area.	19	26·2	26·2	36·8	57·8	84·3	100
Diseased		40	33·3	55·5	67·5	81·3	88·9	100
Healthy hybrids	Diseased area	216	23·6	—	36·0	—	63·2	79·0
Healthy hybrids	Healthy area	600	11·3	12·6	29·8	57·8	60·0	78·0

on this view, it appears that the role of the pathogen is to alter the host metabolism bringing about consequent changes in the host metabolites which are directly responsible for the production of disease syndrome. It is quite probable that the pathogen involved in the root (wilt) disease is responsible for initiating the physiological aberrations associated with the disease leading to the eventual symprom picture as in the case of Pierce's disease of grape vine where slow wilting of the foliage has been reported (Esau, 1948) and the virus induced wilt of tobacco pepper in which permeability changes in the host tissue sets in within a few hours after injection (Glabreil and Pirone, 1964).

The role of the soil factors, apart from the biological entity, in contributing to the development of the disease needs consideration. The significant effect of high water table and waterlogged conditions in enhancing incidence and intensity of disease requires further elucidation. The series of chemical and biological changes that ensues such unfavourable conditions probably act as conditioning factors in pathogenesis. The role of soil factors in the disease complex is evident from the fact that in the transmission trials conducted under controlled conditions the initial symptom of the disease alone was apparent. Perhaps, given the relevant soil conditions, the complete symptom picture might be reproduced.

The observations on the occurrence and activity of *R. solani* further tends to prove its secondary role in the root (wilt) disease. The increased susceptibility of wilt affected palms to *R. solani* has a parallel in the increased root rot in virus infected peas (Farley and Lockwood, 1964) and also the pre-emergence damping off caused by *Rhizoctonia* sp. in cucumber affected by cucumber mosiac virus (Bateman, 1961). McCarter and Halpin (1961) reported that the bean yellow mosaic virus (BYMV) reduced only the yield of top growth of clover plants while *Fusarium oxysporum*, F. *roseum* and *Rhizoctonia solani* and to a lesser extent *Sclerotium bataticola* caused extensive damage to the root system. Papavizas and Davey (1962) have shown that the occurrence and activity of *R. solani* is closely related to the Co2 in the soil. "The pathogenic phase of *R. solani* was more sensitive to Co2 than its active saprophytic phase and the latter was more sensitive to Co2 than its surviving ability within precolonized substrate seg-

ments', the depressing effect of Co_2 being dependent on its concentration, type of soil and inoculum potential of the fungus. The higher incidence of *R. solani* in root (wilt) affected palms with lower quantities of Co_2 in the root zone is thus explained.

That the slow development of symptoms and the debilitating nature of its effect is the culmination of the metabolic derangement in the host initiated by the pathogen which is conditioned by the soil environment is perhaps a logical hypothesis.

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