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COCONUT DISEASES OF UNCERTAIN ETIOLOGY

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COCONUT DISEASES OF UNCERTAIN ETIOLOGY

Several maladies plague the coconut palm in India; of these, four diseases have been the most baffling to the scientific investigators. These are, the root (wilt) of Kerala, Thatipaka of Andhra Pradesh, Tanjavur wilt of Tamil Nadu, and stem bleeding prevalent in almost all coconut growing states. A brief account of the symptomatology, the present status of our knowledge regarding the etiology of each of these diseases and details of investigations carried out towards their control is presented here. At the end of each section, certain salient points for discussion and some questions arising therefrom have been listed. Tear-out blank sheets have been provided for you to note down your suggestions and to raise further questions, if any.

I. ROOT (WILT) DISEASE

This disease was first noticed in three isolated pockets, one at Erattupetta in Kottayam District and two at Kathipara and Kayangulam in Alleppey District within 50 km of each other, following the floods of 1882. Since then it has been steadily spreading towards north and south Kerala and slowly extending even to Tamil Nadu, particularly along the waterways. It now covers about 2·5 lakh hectares (in south and central Kerala) out of the 7·4 lakh ha of coconut gardens (Fig. 1). The disease has been noticed in all types of soil under varying ecological conditions from foot hills to coastal sands. The disease spreads faster in sandy and sandy loam than in laterite soils. Occurrence of healthy palms within a diseased area is an important feature. Although the disease occurs in palms of all ages, young palms in pre- and early bearing stages are more susceptible. The disease is debilitating in nature but not lethal. Loss in terms of nut-yield is proportional to the intensity of the disease and varies from 10 to 80%. The annual loss has been estimated to be approximately 300 million nuts valued at about 300 million rupees.

(a) Symptomatology

1. Crown: The characteristic symptoms of the disease are general wilting of the leaves, from middle whorl outwards, yellowing,

and marginal necrosis of leaflets. The abnormal bending or ribbing of leaflets, termed as 'flaccidity' (Fig. 2), is a typical feature of the disease. In young palms this is most often the only symptom whereas in adult palms foliar yellowing and necrosis are also associated. In a diseased tract, nearly 20% of the palms exhibits leaf rot (Fig. 3) caused by *Bipolaris halodes* Dresch., which leads to a rapid deterioration of the palms. Drying up of the spathe from tip downwards, abnormal shedding of female flowers and buttons, and retardation of general growth, are other associated symptoms. As the disease advances, the whole crown gets smaller in size, due to reduction in size and number of the leaves. The reproductive vigour of the palm being affected, it produces lesser number of female flowers, and consequently a smaller number of poor quality nuts. In the final stages of the disease the tree becomes highly unproductive.

2. Root system: There is a reduction in the number of roots produced. A high percentage of roots is seen to rot from tip backwards even in apparently healthy palms in a diseased area. In certain instances, where the roots remain apparently healthy, their absorbing region gets covered with a hard hypodermis resulting in the cessation of normal root functions. In 1/8th sector of the root system studied, 9·0 to 64·0% of root rot was observed in apparently healthy and 12·0 to 90·0% in the diseased palms, as compared to 3·6 to 13·0% in healthy palms from a disease-free area. Cracks and blotches are visible on older regions, and the cortex turns brownish and dries up in flakes.

(b) Results of investigations

1. Anatomical studies: Leaf: Stomatal distribution on the abaxial side of the tender unopened leaf was 50% more in diseased palms and consequently the opening was narrower. Thickness of cuticle on the adaxial surface was considerably less. The epidermal cells were narrower and the proliferation of these resulted in the slight downward curling of the leaflets. Cells of the mechanical sclerenchyma in hypodermis and bundle sheath tissues were thinner by about 50% leading to a reduction in the rigidity of the leaflets. Xylem vessels were narrower in diameter whereas the phloem tissue appeared proliferated.

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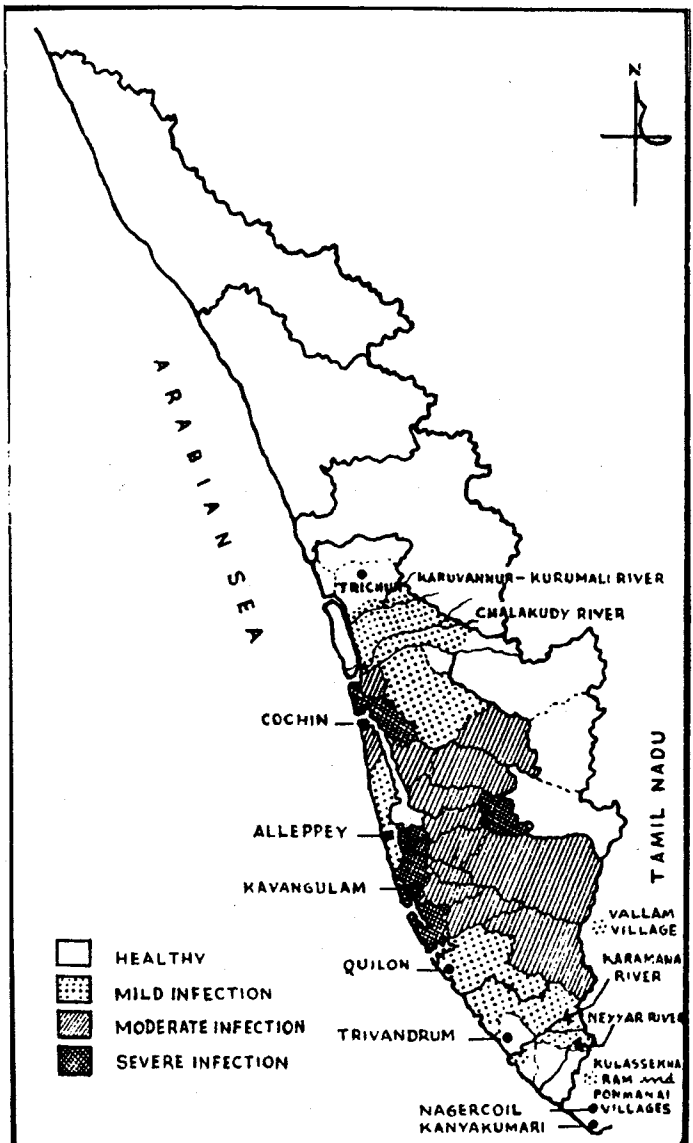

FIG. 1. Map of Kerala State showing distribution and intensity of root (wilt) disease

FIG. 2. A root (wilt) affected palm showing typical ribbing and flaccidity of leaflets

FIG. 3. Another root (wilt) affected palm exhibiting leaf rot.

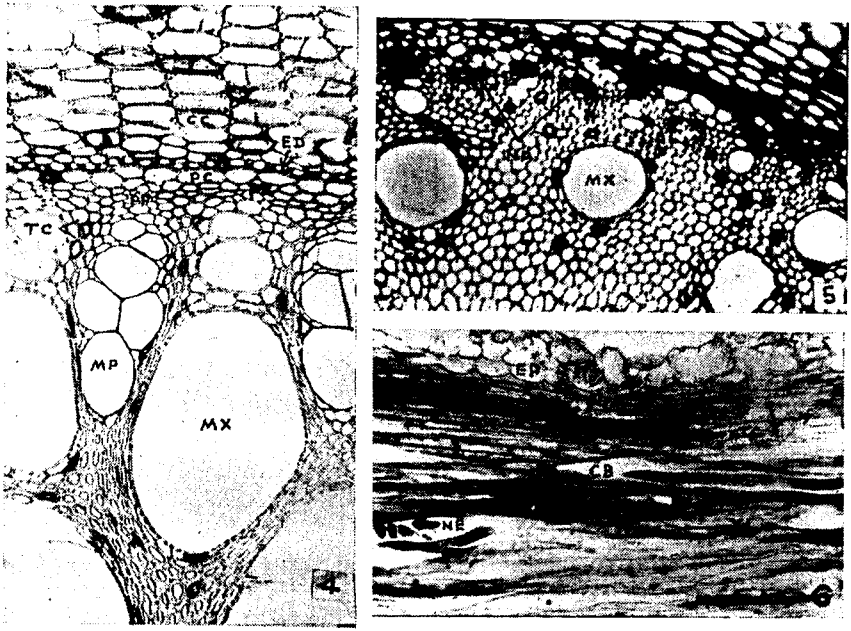

FIG. 4. Transverse section of mature region of a normal healthy coconut root.

FIG. 5. T. S. diseased root.

FIG. 6. L. S. root containing nematodes and their eggs.
CC—companion cell, ED—endodermis, PC—pericycle, PP—protophloem, PX—protoxylem, TC—tannin cells, MP—metaphloem, MX—metaxylem, NP—necrotic phloem, CB—cortical burrow, EP—epidermis, NE—nematode eggs, RS—*Radopholus similis*.

Root: Absorbing region of roots showing no visible damage in apparently healthy as well as diseased palms, revealed internal browning in the stelar region and disintegration of vascular elements (Figs. 4, 5). This was noticed in over 60% of the externally healthy roots from diseased palms (Table 1).

Table 1. Occurrence of vascular browning in healthy, apparently healthy, and diseased palms

Condition of the palm	No. of palms examined	No. of externally healthy roots examined	No. of roots showing vascular browning
Healthy in disease-free area	51	122	0
Apparently healthy in diseased area	86	739	261
Diseased (Index 1–25)*	31	139	109
Diseased (Index 50–100)*	16	18	16

*Disease index is the numerical expression of disease intensity based on the frequency and severity of foliar symptoms of flaccidity, yellowing and necrosis (George and Radha 1973).

Development of tyloses was also observed in vessels of majority of roots from diseased palms. They also showed fungal hyphae in metaxylem. Many roots showed a degenerated phloem. Protophloem and metaphloem elements were deeply stained and occluded. Mechanical and conducting tissues of diseased roots were also poorly stained as compared to the healthy one.

Sections of roots having lesions caused by *Radopholus similis* revealed the presence of nematodes and their eggs in cortical burrows (Fig. 6), which were encircled by deeply staining cells and abnormal sclerenchyma.

2. Etiology: Investigations carried out so far point to a pathogenic nature of the disease, since it could be produced on seedlings raised in pots filled with soil from disease-free area intermixed with roots from diseased palms. Two species of *Rhizoctonia, viz., R. solani* and *R. bataticola* have been isolated frequently from roots of apparently healthy and diseased palms (45.0–60.0%). Being ubiquitous in nature these fungi occur in disease-free area also but less frequently (1.3-20.0%). On

inoculation these fungi induced root-rot in healthy palms but not foliar symptoms of the disease. Waterlogging and acidic pH aggravated root-rot. Sap-inoculated seedlings in pots, which initially showed only flaccidity also developed root-rot on subsequent inoculation with *R. solani*. To check for involvement of fungal toxins, the culture filtrate was fed to coconut as well as sannhemp seedlings. Whereas it produced wilting in sannhemp seedlings, no symptoms appeared on coconut. Cowpea inoculated with *R. solani*, died due to collar rot without showing systemic symptoms, thereby ruling out the role of this fungus as a carrier of any sap-transmissible pathogen. However, the fungus was found to elaborate the enzymes pectinmethylesterase, transeliminase and polygalacturonase *in vitro*. Externally healthy roots of apparently healthy palms which showed vascular degeneration in the absorbing region yielded *Fusarium equiseti*, *Cylindrocarpon effusum*, and a white sterile fungus whose identity is yet to be established, in addition to *R. solani* and *R. bataticola*. Preliminary studies indicated that on inoculation, *C. effusum* induced vascular browning within three months, and *R. solani* caused cortical rotting while the other fungi failed to produce any change in the inoculated roots.

Among bacterial pathogens, *Pseudomonas* has been identified in the "vascular streaming" oozing from the cut end of old roots, from both healthy and diseased areas, although it was more in those from diseased areas. *Enterobacter* was isolated from growing root tips of apparently healthy and diseased palms on an enriched agar medium with 15.0% root extract. This bacterium produced an alcohol precipitable polysaccharide-like substance in culture filtrates from stationary cultures, which caused a reversible wilting of tomato seedlings *in vitro*. Positive results were obtained by Gaumann's test performed with the diluted sap of diseased roots.

Among the 23 plant parasitic nematodes recorded in the root zone of coconut from both healthy and diseased areas, the burrowing nematode *Radopholus similis* is the most important. In addition, we also come across the virus vectors *Xiphinema*, *Longidorus* and *Trichodorus* species. Healthy seedlings in pot culture on inoculation with *R. similis* produced

characteristic reddish brown cortical lesions. These lesions coalesced and enlarged to cause severe root-rot. Although the inoculated seedlings exhibited stunting and retardation of growth, typical root (wilt) symptoms have not been observed so far. The burrowing nematode population on coconut at Kayangulam identified as the "banana race" has been found to infect the cultivars West Coast Tall, Spicata, Laccadive Ordinary, Gangabondam, Dwarf Orange, Dwarf Green, T×D, D×T and T×G.

A sap, vector- and soil-transmissible virus-like agent has been found to produce leaf symptoms such as flaccidity and chlorosis, besides causing stunting of the palms. The insect vector *Stephanitis typicus* which is significantly more numerous on diseased palms, could also transmit this agent to cowpea causing necrosis and malformation of leaves. Results of soil transmission trials were also corroborated by cowpea inoculation tests (Table 2).

Table 2. Disease incidence on cowpea when inoculated with different samples

Method of inoculation	Infection %
Cowpea sown in infective field soil	32
Infective field soil with infective roots	65
Infective field soil without roots	17
Sterilised soil watered with infective leaf sap	84
Sterilised soil watered with infective root sap	76
Sterilised soil watered with infective soil suspension	44
Sterilised soil with artificially infected plant parts	65

In *Capsicum annum* and *Lycopersicum pimpinellifolium*, this agent produced stunting and epinasty of leaves besides delayed flowering. Although a TMV strain has been implicated in this disease by some workers, based on positive reactions obtained with three strains of its antisera, the finding could not be substantiated by further studies on infectivity, serology and electron microscopy. Hence the viral nature of this pathogen still remains to be confirmed.

3. Soil Chemistry: In view of the high rate of spread of the disease along waterways, and the aggravating role of waterlogging

associated with extensive root damage, one is compelled to look for nutritional and soil factors that might be the root cause or would atleast favour the disease expression. The disease occurs in all soil groups, the incidence and spread being more in ill-drained and waterlogged areas. Disease infested soils were poor in available potassium, exchangeable calcium and magnesium. Tissue analysis revealed accumulation of NPK but low status of Ca and Mg. Fertiliser supplements failed to afford any remission of disease symptoms. Detailed nutritional survey was carried out in Kerala on healthy and disease-infested areas. No consistent variation was observed in the major or secondary nutrient status of soils. Sulphur content, exchangeable iron, dithizone-extractable zinc and active manganese were comparatively lower in disease infested areas (Tables 3, 4).

Table 3. Mean values (ppm) of available soil nutrients (0·50 cm) of perfectly healthy tracts of Kerala

Nutrient element	Laterite	Alluvial	Reclaimed	Coastal sandy	Sandy loam
	(82)	(31)	(16)	(31)	(31)
N*	469·70	379·40	451·50	269·40	432·30
P	8·46	8·06	5·44	16·13	10·81
K	95·11	78·77	81·13	13·61	104·90
Na	69·30	433·84	300·38	33·81	66·65
Ca	458·80	486·10	426·90	421·60	480·00
Mg	152·50	217·80	201·80	63·50	116·30
S	30·95	94·65	134·06	4·94	17·58
($Fe^{++}+Fe^{+++}$)	11·35	7·23	17·75	5·32	4·45
Cu	2·42	2·14	2·20	1·23	2·62
Zn	1·70	2·44	2·15	1·97	0·88
B	0·22	0·33	0·24	0·15	0·20
Mo	0·07	0·02	0·04	0·01	0·02
Al	12·74	17·65	65·19	8·07	28·15
Active Mn	70·37	51·48	15·76	4·19	62·19

Figures in parentheses indicate the number of samples; *total N.

Table 4. Mean values (ppm) of available soil nutrients (0-50 cm) of root (wilt) affected tracts of Kerala

Nutrient element	Laterite (82)	Alluvial (69)	Reclaimed (68)	Coastal sandy (43)	Sandy loam (95)
N*	769·01	787·74	774·85	338·82	469·89
P	4·48	12·71	10·81	9·28	37·98
K	83·31	77·53	42·14	13·90	42·97
Na	54·01	59·12	113·83	42·44	44·76
Ca	390·99	335·97	585·00	449·49	332·23
Mg	121·64	143·72	219·11	68·47	75·20
S	28·49	15·81	78·60	3·77	2·24
$(Fe^{++}+Fe^{+++})$	4·26	5·44	11·94	2·68	4·75
Cu	2·97	3·03	2·23	1·40	2·59
Zn	0·78	0·72	0·73	0·36	0·58
B	0·29	0·25	0·26	0·20	0·20
Mo	0·05	0·05	0·04	0·01	0·02
Al	39·43	40·09	47·15	0·97	10·07
Active Mn	31·18	41·48	6·27	0·84	14·47

Figures in parentheses indicate the number of samples; *total N.

Data on leaf analysis clearly indicate that major nutrients were not related to disease incidence. Zinc and molybdenum followed by manganese, iron and boron were limiting in leaf tissue (Table 5). Apparently healthy palms of the disease-affected tract had the same pattern of nutrient composition as the disease-affected palms, and were distinctly different from the healthy ones of disease-free areas. The conspicuous imbalance of nutrients in diseased and apparently healthy palms, will be evident from Table 5. In the case of cations, the ratios directly increased with the severity of symptom expression.

Thus, it is reasonable to assume that the disease may be related to the lower status of zinc and molybdenum. It is likely that boron may also be involved. Direct or indirect involvement of Mn due to the high Fe/Mn ratio and the indirect role of K/Mg based on imbalance of cationic ratios are suggested.

Table 5. Leaf nutrient concentrations and ratios in healthy and disease affected tracts (mean values)

A. Nutrients

Tract	N	P	K	Na	Ca	Mg	S	Fe	Cu	Zn	B	Mo	Al	Mn
	(%)							(ppm)						
Healthy	1·71	0·12	0·82	0·36	0·35	0·38	0·14	254	7·29	20·29	12·67	0·77	18·42	130·9
Apparently healthy	1·87	0·14	0·95	0·35	0·35	0·37	0·12	163	6·59	8·63	11·61	0·44	4·50	63·0
Root (wilt) with foliar yellowing	1·79	0·13	1·12	0·32	0·33	0·37	0·12	182	6·49	7·26	11·03	0·35	4·25	77·6
Root (wilt) with foliar yellowing and rubbery kernel	1·84	0·14	1·31	0·28	0·33	0·31	0·12	158	7·30	7·73	10·98	0·37	3·76	61·7

B. Nutrient Ratios

Tract	N/P	N/K	K/Na	K/Mg	$K\frac{}{Ca+Mg}$	$K\frac{}{Ca+Mg+Na}$	Ca/B	P/Fe	Mn/P*	N/S	P/S	Zn/P*	Ca/Mg
Healthy	14·9	2·6	1·4	0·68	0·46	0·34	329	6·0	1197	13·5	0·9	178	1·6
Apparently healthy	13·8	2·3	1·6	0·78	0·53	0·45	407	10·1	468	17·5	1·3	64	1·7
Root (wilt) with foliar yellowing	14·0	2·1	2·1	0·95	0·65	0·48	452	9·1	634	18·3	1·3	58	1·7
Root (wilt) with foliar yellowing and rubbery kernel	13·9	1·8	2·8	1·25	0·86	0·60	633	10·3	459	17·1	1·3	59	1·9

*Values multiplied by 10^4

Loss of permeability of roots, imbalance in water relations, increased respiratory activity (Table 6), and the accumulation of inorganic nutrients, nonprotein N, total sugars, and inorganic P (Table 7) have been recorded in diseased palms. There was a fall in the concentration of total phenols in coconut roots with increasing intensity of the disease and a concomitant increase in the level of polyphenol oxidase. An unidentified toxic substance which could induce epinasty and wilting of tomato plants was detected in the root sap of diseased palms.

Table 6. Rate of respiration in the leaf and root tissues of healthy and root (wilt) affected coconut palms (expressed as mm^3/mg dry wt./hour)*

Condition of palms	Leaf	Root
Healthy	0·59	0·35
Diseased, early	0·59	0·39
Diseased, advanced	0·71	0·40

*Mean of 20 palms.

Table 7. Nitrogen and phosphorus fractions in healthy and root (wilt) affected coconut palms (expressed as percentage of dry wt.)

Fraction	Healthy	Diseased	
		Early stage	Advanced stage
Total nitrogen	1·93	2·08	2·22
Total protein-N	0·66	0·06	0·03
Total nonprotein-N	0·08	0·09	0·12
Total phosphorus	0·47	0·49	0·46
Inorganic phosphorus	0·33	0·26	0·29
Organic phosphorus	0·14	0·29	0·17

4. Control measures attempted: Since heavy manuring with NPK fertilisers, particularly K, increased foliar yellowing, a judicious application of these fertilisers was recommended and this resulted in increased yield during early stages of the disease. Magnesium application was, however, found to be effective against foliar yellowing.

Intercropping coconut with fodder crops and recycling animal wastes under mixed farming conditions resulted in increased production of inflorescence, female flowers and consequent increase in yield of nuts to the tune of 28%. This also resulted in improved soil conditions particularly with reference to organic carbon, exchangeable Ca and Mg, available K as well as the microbial status. Soil microbiological studies revealed the profitability of cultivating *Stylosanthes gracilis* alone or in combination with hybrid napier, as the intercrops.

No positive result has so far been obtained with the use of fungicides and bactericides in the control of the root (wilt) disease. All attempts to isolate resistant varieties by progeny testing of healthy palms from diseased areas, and also using pollen from these for making crosses, have not so far yielded positive results.

(c) Current Research Programmes

Investigations are in progress on the following areas:

(1) Detailed histopathology of infected parts of the palm, combined with a histochemical analysis of host-pathogen interactions and similar studies on roots artificially inoculated with suspected pathogens.

(2) Sequence of symptom development on newly planted seedlings in diseased area.

(3) The sequence of development of the microbial and parasitic associations right from the onset of disease syndrome, in palms from the transitional areas, coupled with studies on artificial inoculation of these pathogens, individually and in several combinations.

(4) The distribution and intensity of the nematode (*R. similis*) population in relation to the disease, and epidemiology after artificial infection by this nematode.

(5) Characterisation of the polysaccharide-like material elaborated by the *Enterobacter* sp. and the pectinolytic capabilities of the fungi so far identified.

(6) EM studies to determine the nature of the sap transmissible agent of the disease. Possible association of infectious RNA as well as mycoplasma also being investigated.

(7) Physiological analysis of post-infection changes with reference to permeability, water absorption, stomatal movement, as well as absorption, translocation and distribution of nutrients, particularly P and K, using radiotracer techniques.

(8) Effect of soil and foliar application of Zn and Mo, alone and in combination.

(9) Possibility of using the specific antiserum prepared against partially purified preparations from diseased coconut tissues, to diagnose the disease prior to manifestation of foliar symptoms, and to use this technique to detect possible secondary weed hosts.

(10) A sensitive biochemical test based on chelating compounds as extractants is being perfected for detecting the incipient stage of the disease.

(11) Studies on transmission of the disease through agents such as *Stephanitis typicus*, *Longidorus sp.*, through seed, sap and aerial means.

(12) Chemical control of the disease through fungicides, bactericides and nematicides, separately and in various combinations.

(13) Screening for sources of resistance among 78 different germplasm material comprising both cultivars and hybrids.

(14) Estimation of micro- and macronutrients in soil and leaves, on WCT and D × T seedlings where application of Mg had increased the number of functioning leaves and induced early flowering. Fertiliser trials with different NPK combinations and Ca and Mg are also being continued on these plants.

(15) Detection of biologically active inorganic nutrients and metallo-enzymes in plant tissues.

(16) Effect of mixed cropping of coconut with cacao, pineapple and tuber crops on nut yield and their influence on the beneficial rhizosphere microflora.

(d) Future programmes

(*i*) Fractionation and characterisation of the factors identified through serology and screening the native flora to locate alternate hosts.

(*ii*) Screening for possible involvement of mycoplasma-like organisms and viroids.

(*iii*) Characterisation and assay of peroxidase and polyphenol-oxidase enzymes.

(*iv*) Collection of fresh germplasm and screening them for reaction to the disease.

(*v*) Development of suitable crop combinations and management practices for increasing the productivity of the already affected gardens.

(*vi*) Identification and characterisation of a biochemical compound extracted by the chelating complexes and ethyl acetate from leaves and roots.

(*vii*) A field trial involving micronutrients applied from seedling stage onwards.

(*viii*) Anatomical studies of stem tissue from diseased palms.

(*ix*) EM studies of insect-vector tissues to check for viruses and/or mycoplasma-like organisms.

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Points to ponder

1. Could the causal agent of the root (wilt) disease be an organelle-bound, free viral nucleic acid, or MLO, RLO or any other protista, since it has so far evaded isolation by conventional methods?

2. What could be the reason for the gradation of symptom expression? Is it due to multiplicity of etiological factors, variable susceptibility of the host or the result of interaction between ecological condition and primary pathogen, or due to variation in the involvement of associated factors?

3. Root (wilt) affected palms in most cases, but not all, are also affected by leaf rot. Is it possible that root (wilt) predisposes the palm to the invasion by *Bipolaris halodes'* or is the latter an independent infection? If so, what should be our strategy for controlling leaf rot effectively?

4. Since root (wilt) is more prevalent along waterways, sandy loam and sandy soils, what are the chances for atmospheric pollutants or other edaphic factors being the causative agents or at least the preconditioning factors for the onset of disease?

5. What are the chances for soil-borne vectors other than nematodes being the agents of disease spread?

6. The fact that the disease appears afresh far remote from infected zones perhaps points to some additional means of spread other than through soil alone. Does this call or careful screening of seedlings by the antiserum or other sensitive methods?

7. How does one partition the relative roles of fungi, bacteria and nematodes, all of which have been implicated with this disease?

8. What would be the best method for establishing the effect on coconut of the *Enterobacter* polysaccharide-like materials which caused reversible wilting and epinasty of tomato seedlings *in vitro?*

9. How best can we favourably alter the rhizoplane microflora of coconut roots through appropriate mixed cropping pattern, so as to ameliorate diseased palms?

II. THATIPAKA DISEASE OF ANDHRA PRADESH

The disease derives its name from the village 'Thatipaka' in Razole Taluk of East Godavari District, where it made its first appearance following the devastating cyclone of 1949. Palms in the age group of 26-60 years were most susceptible although the disease was found to occur even on 10 year old palms. The highest incidence (of 32%) was in black alluvial and clayey soils, as well as in red sandy soils where stagnation occurred almost throughout the year. The spread of the disease was more along the Vasishta branch of Godavari and it has been recorded even as far away as Srikakulam. The rate of appearance of this disease was increased by prolonged drought followed by a sudden downpour, as was the case during 1973 and 1974.

(a) Symptomatology

1. Crown; Initially, the infected tree produces more number of leaves than what it sheds, as compared to a normal tree. Size of leaflets increases at this stage resulting in an abnormally large crown. Colour of inner leaves is darker than normal green, whereas the outermost whorl shows yellowing. The fronds bend abnormally from the middle instead of bending at the distal end. Larger number of nuts are produced, and the yields may go up. After this stage, the visible decline starts with the lengthening of petioles which decreases their capacity to hold the weight of bunches, and the trees start dropping more number of leaves than do healthy ones. The stem begins to taper suddenly, producing smaller sized, pale leaves. In some cases the trunk assumes a S-shaped curve just below the crown. Watersoaked chlorotic spots appear on the foliage and improper unfolding of leaflets gives them a fasciated, fan-like appearance (Fig. 7). In some cases the leaves become so narrow that it gives the appearance of a datepalm crown. Such trees produce smaller bunches with atrophied nuts, which become smaller, lose their trigonal shape and appear round. The mesocarp becomes spongy, and the kernel is lost gradually, resulting in pigmy sized nuts filled only with mesocarp. Some nuts show longitudinal cracks with occasional gumming at the stalk end. Subsequently, even the inflorescence fails to appear and the barren tree perishes slowly.

FIG. 7. A Thatipaka-affected palm

Root: Infected trees exhibited extensive root rot. In the initial stages of the disease, a large number of vigorously growing roots are seen. Gradually, they dwindle in number and thickness as the disease advances, and the number of dead roots also increases. Regeneration of new roots was also poor in diseased palms.

(b) Results of investigations

Three types of trees were examined in Thatipaka village, near Razole, namely, (1) healthy bearing tree (2) one showing the disease since a year, with reduced nuts and tapering stem, and (3) a tree in advanced stage of disease with atrophied barren nuts, fasciated leaflets and fewer spadices. When roots were dug out it was found that the percentage of healthy roots was more in healthy trees than in the other two trees. When the pits were refilled with river sand and the root system re-examined 3 months later, it was seen that the healthy tree regenerated the largest number of roots which were also stouter than those produced in diseased tree. Thus, root decay appears to be a key symptom of this disease.

Although incubation of roots from both healthy as well as diseased trees gave rise to *Diplodia* and *Thielaviopsis* colonies, their precise role in the pathogenesis or as causal incitants is yet to be demonstrated. Analysis of soils collected from healthy and diseased areas has not so far revealed any differences nor deficiencies of nutrients. However, a chemical analysis of affected palms indicated a deficiency of nitrogen and zinc, but attempts to remedy the situation through urea (10%) and zinc sulphate (2%) sprays as well as soil application of ammonium sulphate (1 kg/tree) and $ZnSO_4$ ($\frac{1}{2}$ kg/tree) did not produce any recovery. Similarly, the spraying of hormones such as IAA and IBA (250 ppm each), GA (500 ppm) *etc.* did not produce favourable results, but on the other hand, enhanced the rate of decline.

Studies on seed transmission showed that nuts from infected tree germinated late and poorly, producing weak seedlings with long petioles and lesser number of leaflets which were flaccid and bent abnormally. These symptoms closely resembled those of healthy seedlings underplanted in close proximity to diseased trees, and finally developed the

typical "Thatipaka" syndrome. Attempts were made to detect the disease in its early stages. Since the infected palms started with profuse bearing it was necessary to distinguish a genetically superior heavy yielder from an early stage of Thatipaka affected tree. A study of nuts revealed that those of diseased trees on storage for a month, developed a shrivelled mesocarp. However, this was observed only in nuts collected from trees that have already assumed early stages of Thatipaka appearance, and not in the incipient stages of the infection. It was further observed that only 17·6 to 42·9% of the prolific bearers ended up with Thatipaka, indicating thereby that the remaining could be genetically superior yielders. Table 8 gives a comparative picture of foliar and nut characters in diseased and healthy heavy yielders.

Table 8. Initial and final symptoms of foliage and nuts of heavy yielding coconut palms that remained healthy and those that turned diseased in 8 years

Sl. No.	Nature of palm	Foliage		Symptoms of fully ripe nuts	
		Initial symptoms (1961)	Final symptoms (1968)	(1961)	(1968)
1.	Heavy yielder* (remained healthy upto 1968)	Dark green big leaves bent like a bow from the middle of frond	No change	Normal size, mesocarp hard, no shrivelling when stored	No change
2.	Heavy yielder†	–do–	Leaves normal green for some period, then pale and small, still showing faint bending from the middle	Normal size, mesocarp hard but shrivelled when stored for a month	Small, round mesocarp soft, copra does not stand storage

3.	Normal yielder* (remained healthy upto 1968)	Normal green normal size	No change	Normal size, mesocarp hard, no shrivelling, copra stood storage	No change
4.	Diseased palm** since 1961	Small pale leaves faintly showing bending from the middle.	Leaves pale, further reduced in size, chlorotic spots developed on lower leaves in 1966. In 1968 only 2 leaves remained green.	Nuts small, round, mesocarp soft, copra does not stand storage	Nuts further reduced in size, devoid of copra, shrivelled, developed cracks, dropped like buttons until the year 1968. The tree stopped bearing from 1967 onwards.

*They remained in the same stage even in the year 1974.

**Died by the year 1974.

†In the final stages of disease by the end of the year 1974.

It would be seen that a long time is required before one can distinguish between the two. Attempts to locate indicator plants to afford early detection of the malady have so far not succeeded. In sap transmission studies, the seedlings receiving diseased sap exhibited pale green foliage compared to the normal green leaves of control plants. Experiments on *in vitro* inhibition of the infective principle indicated that a virus may be involved, since tetracycline failed to inhibit the infection, whereas disease sap mixed with 8-azaguanine or thiouracil caused suppression of the disease. However, the actual administration of these base analogues into diseased trees could not effectively check the infection. A comparison of Thatipaka disease with other diseases of unknown etiology such as root (wilt) of Kerala, cadang cadang of Philippines, Tinangaza of Guam, and Chinese coconuts of Jamaica (Table 9) shows that the most distinctive symptom is the augmented crown size, followed by a sudden decline leading to the formation of typical Thatipaka syndrome.

Table 9. A comparison of Thatipaka disease with other complex coconut diseases of the world

S. No.	Name of the disease	Similarities with Thatipaka disease	Differing symptoms of	
			Thatipaka disease	Other disease
1.	Root (wilt) disease of Kerala (India)	Abnormal bending of petiole; yellowing of leaves; stunting of crown.	*a) Yield of diseased palms & nut characters* Diseased palms yield more nuts that are 100% atrophied and do not stand for storage.	Diseased palms continue to yield progressively lesser number of nuts.
			b) Leaf necrosis Not seen	Leaf necrosis is invariably met with:
2.	Cadang-Cadang (Philippines)	Infection mostly of bearing palms, nonrecovery of affected trees; rounding and smalling of nuts.	*Leaf fall in quick succession* Occurs in early stages of the disease	Occurs in advanced stage of the disease.
3.	Tinangaza disease (Guam)	Chlorotic mottling of leaflets closely resemble the chloric water soaked spots of Thatipaka disease.	—	—
4.	Chinese coconuts (Jamaica)	—	*Nuts* Mesocarp becomes spongy, nuts become small and round and in the advanced stage they remain as buttons only.	Nuts are highly deformed without shell or copra and more than one nut produced on each rachilla. (Maramorosch 1964).

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Points to ponder

1. The sudden spurt of vegetative growth before the decline, perhaps points to excessive hormonal activity. What tiggers off this stimulus deserves critical examination, in order to understand the nature of hormonal imbalance.

2. Does the nonshedding of leaves in the initial stages of Thatipaka disease indicate slow senescence or prevention of abscission?

3. The developmental processes leading to abortive nuts needs to be studied in detail to locate the possible causes of the malady.

4. Is the excessive shoot development taking place at the cost of root growth?

5. Can we rule out the possibility of seed transmission of the disease, in view of its sporadic occurrence even as far as Srikakulam District. Pedigree records of infected trees as well as fresh plantings need to be examined.

6. Since underplanted seedlings also take up the infection, this definitely points to a transmissible pathogenic entity. Is root anastomoses playing any role in disease transmission?

III. TANJAVUR WILT

The disease was first reported from the coastal areas of Tanjavur district following the cyclones of 1952 and 1955. It has now spread to the interior also and the loss due to the disease is estimated at Rs. 6,00,000/-. It is prevalent in all districts of Tamil Nadu except the Nilgiris and the incidence ranges from 3.3 to 20%.

(a) Symptomatology

A protracted dry summer and waterlogging in rainy season favoured the occurrence of disease symptoms. The subsoil of Tanjavur district consisting of a mixture of sand and yellow clay which hardens in summer forming a hard pan and impeding root penetration, predisposes the coconut trees to infection.

1. Aerial parts: Wilting of trees was also accompanied by stem bleeding in many cases. Trees in the age group of 10-30 years were more susceptible than younger trees. Some palms showed only wilting symptoms unaccompanied by stem bleeding. In fact some dead palms showed no bleeding at all. In palms that bleed, a reddish brown viscous fluid is exuded from the peripheral tissues of the basal portions of the stem and such patches extend upwards up to five meters with the advance of the disease. The discolored core of the stem is confined to the height at which active bleeding occurs. In the final stages of disease the basal portion of stem gets decayed and discolored. Affected tissues are lighter in color.

The outer one or two whorls of leaves droop down, and later exhibit light to moderate browning. As the disease advances, the remaining leaves also droop down in quick succession (Fig. 8) until the spindle alone remains. Under prolonged infection, the outer leaves fall away and subsequent ones get reduced in size with a shortened spindle that does not unfold properly. A soft rot sets in the bud in certain cases, resulting in loss of turgidity and death of the cells due to breakdown of conducting elements. The affected bud emits a bad smell and in advanced stages the spindle gets blown off leaving a decapitated stump. Fructifications of the bracket fungus *Ganoderma lucidum* occur in few cases on the trunk of wilted trees just above the soil level.

FIG. 8. A Tanjavur wilt-affected palm

2. Root: Extensive rotting and discoloration of root system and fewer secondary and tertiary roots, distortion of primary and secondary roots are characteristic symptoms of this disease. Cortical tissues disintegrate and the stele turns brown. There is progressive reduction in number of new roots produced.

3. Flowers: Normal development of flowers and bunches gets arrested with the rapid advent of disease symptoms, leading to shedding of immature buttons. When disease is less acute and slow in progress, there is no button shedding. As the leaves droop down, the subtended bunches also hang down. Even the nuts that develop are without kernels. Where disease progress is slow, only a few normal nuts are produced, and in fact most trees bear profusely just prior to and at the time of initiation of symptoms.

Thus, five distinct stages in the progress of Tanjavur wilt disease can be recognised, as follows:

1. Decay and death of fine roots, then main roots; light bronzing of lowest leaf whorl and partial folding of leaflets.
2. Flaccidity of spindle leaves, intense bronzing of outer leaves; unproductive inflorescence with arrested fruit set; root decay extending to bole region.
3. Decay extends into stem; bleeding patches appear on basal part of the trunk; outer 1-2 whorls of leaves droop down, resulting in their bunches hanging down; spindle shortened and erect without unfolding properly.
4. Internal stem decay extends up to middle; bleeding patches increase; outer leaves drop away and crown gets reduced in size due to undersized pale leaves.
5. Tree dries up with the spindle and can easily be uprooted, or the top gets usually blown off and stem gets shrivelled and dries up.

Stages 3, 4 and 5 last between 6 to 54 months, the average being 24 months. Where death occurs in 6 months, stage 4 is absent. In stages 3,

4 and 5, *Xyleborus* is usually found boring into the stem at the bleeding patches. In certain areas, *Scolytid* beetles and *Diocalandra* weevils aggravate the disease, causing rapid death.

(b) Results of Investigations

Although the application of NPK fertilisers did not ameliorate the diseased palms, organic manuring afforded some protection to healthy trees. Four out of five organic manurial treatments in irrigated trials showed less intensity of disease than in the non-irrigated series. In many cases withdrawing chemical fertilisers and application of farmyard manure produced partial recovery of diseased palms.

Among the fungicides tried, Bordeaux mixture was effective and was hence used along with fertilisers. The best combination was with tank silt and green leaves. Burying coconut husks around trees also reduced intensity of infection in older trees. Heptachlor (3%) was effective in controlling *Xyleborus* beetle and thus reduce disease intensity. Aureofungin was better than other fungicides when applied as stem injection.

(c) Future Programmes

i. Survey of different areas to assess the latest position of the disease incidence.

ii. Isolation of microorganisms from roots and basal portion of trunk of diseased palms as well as surrounding healthy palms.

iii. Monthly recording of symptom manifestation and the meteorological data.

iv. Estimation of sugars from the exudate.

v. Estimation of pH and electrical conductivity of soils at 0-30, 31-60 and 61-90 cm depths.

vi. Pathogenicity tests with suspected organisms.

vii. Induction of bleeding symptoms by heavy chemical manuring.

viii. Control measures using chemicals, antibiotics, organic manures, management practices, and NPK fertiliser trials.

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Points to ponder

1. The amelioration of diseased palms following organic manure application needs critical examination, to see if it is due to improvement in C/N ratio or in soil texture, water holding capacity, and prevention of hard pan formation. What is the effect on soil reactions?

2. Since fructification appears as the last stage of *Ganoderma*, its presence in tissues long before the appearance of any symptoms is essential. Quick detection techniques, including serology and chromatography would be useful.

3. The common mode of origin of root (wilt), Thatipaka and Tanjavur wilt diseases, following a natural calamity such as a flood or a cyclone is intriguing. Perhaps we should check meteorological data around these times and see if any correlation exists.

IV. STEM BLEEDING IN COCONUT

Stem bleeding was first reported from Ceylon in 1906 and from India in 1922. It is known to occur in nearly all coconut growing countries and in all soil types from coastal sands to laterite. It may occur sporadically in isolated palms or gardens or endemically in certain areas. Although mainly occuring in coastal sandy loams and reclaimed clay soils, stem bleeding has also been found to occur in shallow laterite and loamy soils inland, where a hard pan of clay or granite has been noticed. The extent of damage varies from reduction in yield to complete death of the palms.

(a) Symptomatology

The exudation of a reddish brown liquid through growth cracks on the lower part of the trunk, is a characteristic symptom (Fig. 9). This fluid turns black as it dries up, and the tissues below the bleeding patch become decayed and yellowish. Initially the palm bears heavily and when the bleeding spreads upwards, the vigour and yield go down, the crown becomes smaller, and in extreme cases the palm may become barren and die. Usually, palms of 20 years and above are the victims, whereas younger ones are seldom affected. Sometimes the whole stem in young palms may rot internally without external bleeding. On piercing the stem, a yellow fluid gushes out and the palm collapses.

Four types of stem bleeding can thus be recognised, as follows:

1. Profuse bleeding through cracks in basal region, progressing upwards, in soils subject to alternating inundation and drought; death rate variable from rare to common, in Kerala, Southern coastal Tamil Nadu, and coastal Goa.
2. Moderate to extensive bleeding in isolated gardens, shallow loamy and laterite soils with clay or rock underneath, and in reclaimed soils.
3. Sparse bleeding limited to lower half of the trunk, causing extensive internal damage, and death very commonly occurs in patches, in sandy loam soils of Goa.

FIG. 9. Lower part of a coconut trunk showing stem bleeding

4. Sparse bleeding accompanied by wilting of leaves of outermost whorl, leaving an erect central spindle. This is associated with the root infection by *Ganoderma lucidum* prevalent in Andhra Pradesh, 'Anabe roga' in Karnataka and Tanjavur wilt in Tamil Nadu.

(b) Etiology

The disease was believed to be caused by infection with *Ceratostomella paradoxa* through the growth cracks on the stem which are vulnerable to the fungus. *Thielaviopsis paradoxa* as well as its perfect stage *C. paradoxa* have been isolated from the stem tissues underlying the bleeding patches, as well as the exudate. Isolations made from roots of healthy and diseased palms yielded mainly a sterile fungus yet to be identified.

The occurrence of the growth cracks have been connected with strongly marked alteration of the wet and dry seasons. This has also been implicated with the drastic and sudden changes in moisture content of the soil.

Infestation by stem borers like *Diocalandra* and *Xyleborus* has been found to accentuate the damage to vasculature causing rapid deterioration of the palm.

(c) Results of Investigations

1. Inoculation trials were conducted with *T. paradoxa* and *C. paradoxa* in two localities of Kerala (*a*) where the disease occurs naturally, and (*b*) where it is not prevalent. Stem bleeding was induced in 2 out of 25 palms in the former area. In Andhra Pradesh, nearly 8 per cent of the inoculated palms developed stem bleeding.

2. Soil samples from 17 sites (five each in Kerala, Tamil Nadu and Goa and two in Karnataka) taken at 0-50 cm and 51-100 cm when analysed for pH and electrical conductivity, showed no significant differences between healthy and diseased gardens.

3. Calcium content of leaf also did not reveal significant variation between healthy and diseased palms. Studies on cell sap conductivity

indicated higher values (0.01 to 4.70 millimhos) in diseased than in healthy (0.85 to 0.92 millimhos) roots.

(d) Control measures attempted

1. **Organic manuring:** In a garden at Adat in Trichur district and in Goa, stem bleeding could be cured by discontinuing the application of NPK fertilizers and applying organic or fish manure instead.

2. **Coal tar treatment:** The decayed and discoloured tissues beneath the bleeding spots are chiseled away and burnt. The cleaned area is smeared with molten coal tar or Bordeaux paste (1 kg copper sulphate + 1 kg quick lime in 10 litres of water, dissolved separately and mixed together).

3. Where stem bleeding was associated with *Ganoderma* infection, aureofungin sol + copper sulphate (100 ppm) and Vitavax (500 ppm) administered through roots proved to be effective in checking the disease in early stages.

4. Improvement in drainage in lowlying waterlogged areas, and soil moisture conservation measures in drought areas has proved beneficial in checking the incidence of stem-bleeding.

5. Opening of a hole right through the stem is reported to check stem bleeding in Goa.

(e) Future programmes

i. Seasonal variation on symptom expression—to be recorded at 4 locations, namely laterite and sandy soils at Kasaragod, reclaimed soil at Kayangulam and one at Karnataka.

ii. Isolation of fungi and bacteria to be made from roots and stem tissues and from the liquid oozing out of affected palms.

iii. Sucrose content and elemental composition to be estimated from exudates.

iv. Osmotic pressure of root sap and the electrolytes in it are to be determined.

v. Soil samples from 0-30, 31-60 and 61-90 cm from observational plots to be analysed for pH and conductivity.

vi. New treatments such as antibiotics, management practices and the hole method to be evaluated critically.

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Points to ponder

1. It has been suggested that stem bleeding is purely a physiological disorder whereby the excessive nutrients find their way out through cracks, owing to a lack of sink above. Perhaps root pruning would help in stimulating additional sink capacity by way of new roots. This is worth checking up.

2. Investigating the role of the fungus, *Thielaviopsis* and its perfect stage, *Ceratostomella* by detecting it in early stages of the disease, before appearance of bleeding symptoms is important, since fungicide treatment checks the malady.

3. Infection by *Ganoderma* is considered to be unrelated to stem bleeding as such, since they can occur independently of each other also. Early detection of *Ganoderma* infection in root and stem tissues is called for.

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