

EPIDEMIOLOGY OF COCONUT ROOT (WILT) DISEASE

N. GOPINATHAN PILLAI, M. SASIKALA AND K. MATHEN

*Central Plantation Crops Research Institute
Regional Station, Kayangulam,
Krishnapuram-690 533, Kerala, India*

ABSTRACT

Coconut root (wilt) disease of Kerala (S. India) is a debilitating disease of uncertain etiology. Out of 7,40,000 ha of coconut plantation, 2,50,000 ha are affected by this malady causing an annual loss of about 340 million nuts. Associations of fungi, bacteria, nematodes, and a transmissible virus-like pathogen with the disease have been reported.

Observations recorded for seven years in 10 coconut gardens, each of approximately one ha in extent with very low percentage of disease incidence revealed indiscriminate spread in sandy, sandy-loam, and alluvial soils. The percentage of infection increased from 2.3 to 18.2 with 2-5 palms contracting infection every year. The spread of the disease was slower in laterite soils, with only one palm developing the disease in two years. The spread was erratic and irregular irrespective of soil conditions and occurred in jumps/leaps. The rate of spread was 1-4 km from the nearest source of infection. The pattern of spatial distribution or galaxial outbreak of the disease is suggestive of the involvement of aerial vector(s).

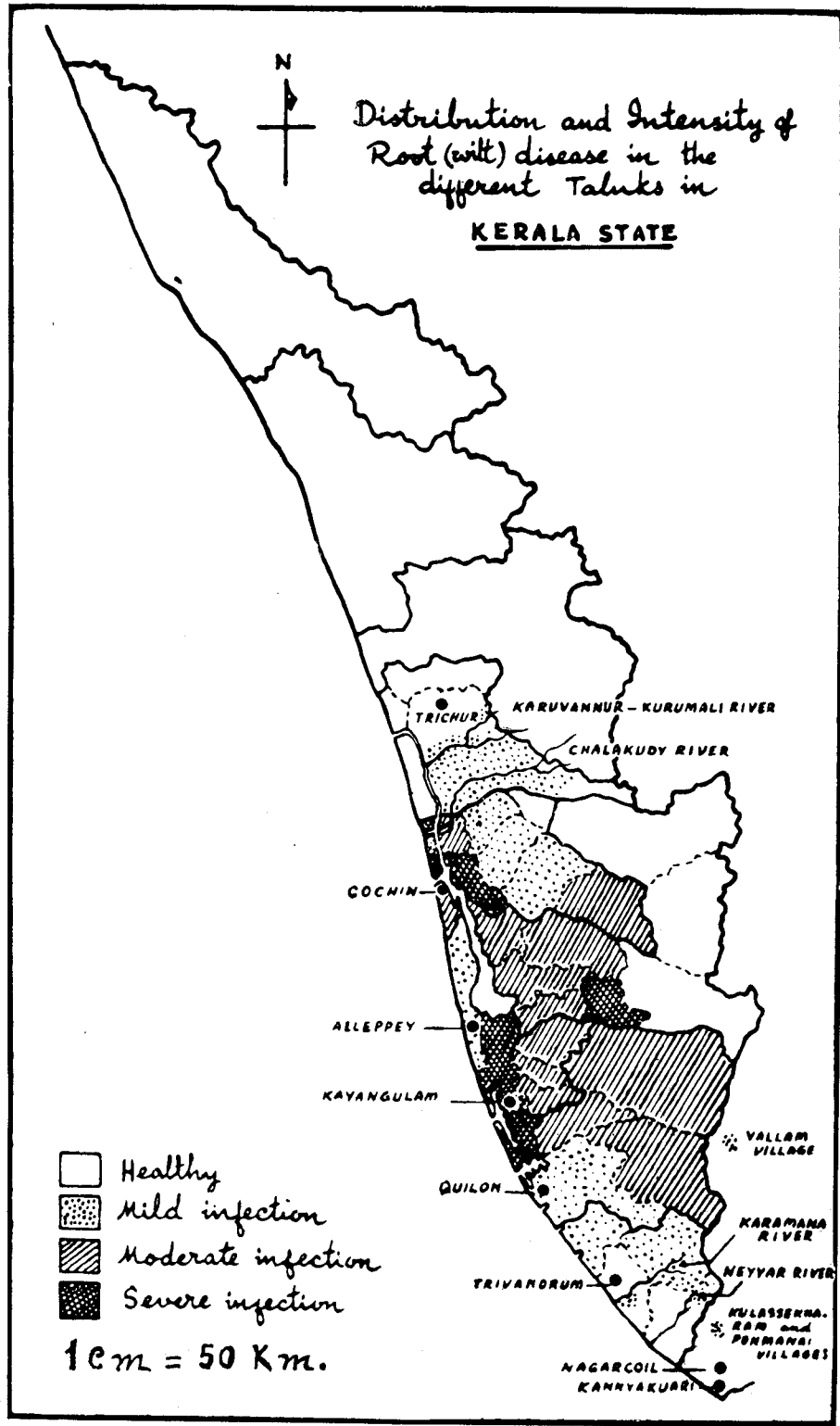
INTRODUCTION

Root (wilt) disease of coconut has been known to occur in Central Kerala since 1882. (Butler 1908, Varghese, 1934). It is not lethal, but causes a loss of 340 million nuts annually (George, Thomman, and Sukumaran, 1976). The spreading nature of the disease was emphasised first by Varghese (1934). It now occurs in about 2,50,000 ha of coconut plantations, which is about a third of the total area under coconuts in Kerala (Fig. 1). Palms of all ages are susceptible to infection. The cause of the disease is uncertain, but several agents like a virus-like pathogen (Shanta, Menon and Pillai, 1960, Summanwar et al., 1969) fungi (Joseph, 1978) and bacteria (Srivastava, Sekhawati and Rao, 1969; George, Potty, and Jayasankar, 1976) have been implicated with the disease. The present study was

undertaken to observe the pattern and rate of spread of the disease in the border areas between disease affected and disease free gardens of Kerala.

MATERIALS AND METHODS

Ten plots, each of approximately one ha size, all with low percentages of disease and distributed in different soil types, viz., reclaimed sandy, sandy-loam, alluvial and laterite, were selected in the northern borders of the root (wilt) affected areas during 1968-69. All the plots contained West Coast Tall palms and they were of different ages. The lay outs of the plots were prepared. One plot was eliminated at a later stage because of non-cooperation from the owner farmer. In another plot, the farmer uprooted the diseased palms soon after the disease was observed. The plots were



observed at half yearly intervals for seven years on disease index of affected palms. mode of fresh appearance of disease, flow of water and level of water table.

Three garden-to-garden surveys were carried out in 40 villages of Trichur District (northern most disease affected areas of the State) at 2-3 years intervals jointly with the Kerala Agriculture Department to delineate the boundary between the affected and healthy areas and to study the spread towards un-affected areas (Fig. I). This was carried out by a team of trained observers who surveyed all the coconut plantations in all the border villages.

RESULTS AND DISCUSSION

The incidence of the disease in different plots in 1968-69 was very low, ranging from 2.0-2.3%. The incidence of the disease

increased steadily in all the plots but irregularly during the seven year period beginning December, 1969, irrespective of soil conditions except in the plot in which the diseased palms were removed as soon as they had been observed. This garden did not show any fresh outbreak of the disease till 1976. Water table in plots of alluvial, sandy and sandy-loam soils ranged from 1.0-3.0 m, and in plots of laterite soils from 5.0 to 12.0 m in the different seasons.

Pattern of spread. In one plot (Fig. II), there were initially in 1968-69 four palms (2.3%) showing symptoms of root (wilt) disease. In 1970, three palms developed the disease, one near the primary focus and the other two 25-32 m away from the primary focus in the third row of palms. During 1971, four palms took up infection almost in a similar pattern. In 1972, three palms developed infection, in 1973, five palms, and in

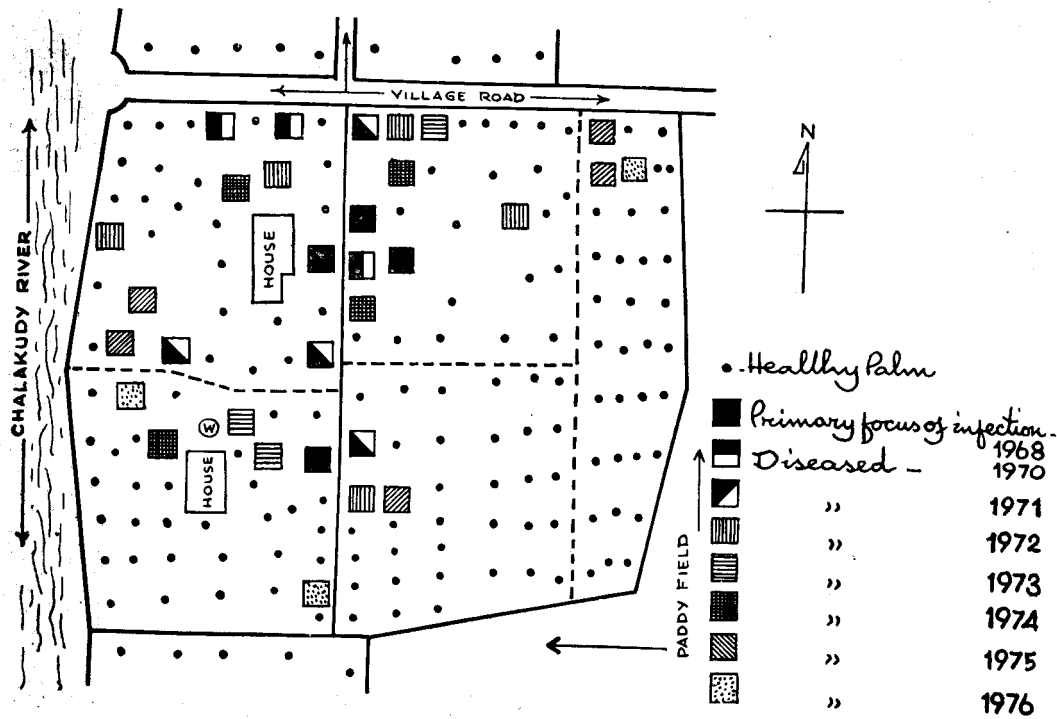


Fig. II Observational plot—Cheruval Village Soil type—Alluvial.

1974, four palms showed symptoms. In 1975, five more cases of fresh infection were observed jumping across several rows of coconut palms. Thus by the end of 1976, 31 out of 170 palms showed the disease (Fig. II). That is, within a span of seven years, the disease had increased from 2.3% to 18.2% within the garden in an irregular manner hitting one palm, and jumping across several rows to hit another, showing a discontinuous spread of the disease.

The pattern of spread of the disease was the same in all the other plots. But the percentage increase in the incidence varied in the plots (Figs. III and IV). In a laterite soil garden, only two palms were affected in 1968, no fresh occurrence was recorded till 1971. In December 1971, one palm standing 35m away from one of the two diseased palms showed disease symptoms, and in June, 1974, another palm took up infection jumping three rows of coconut palms from

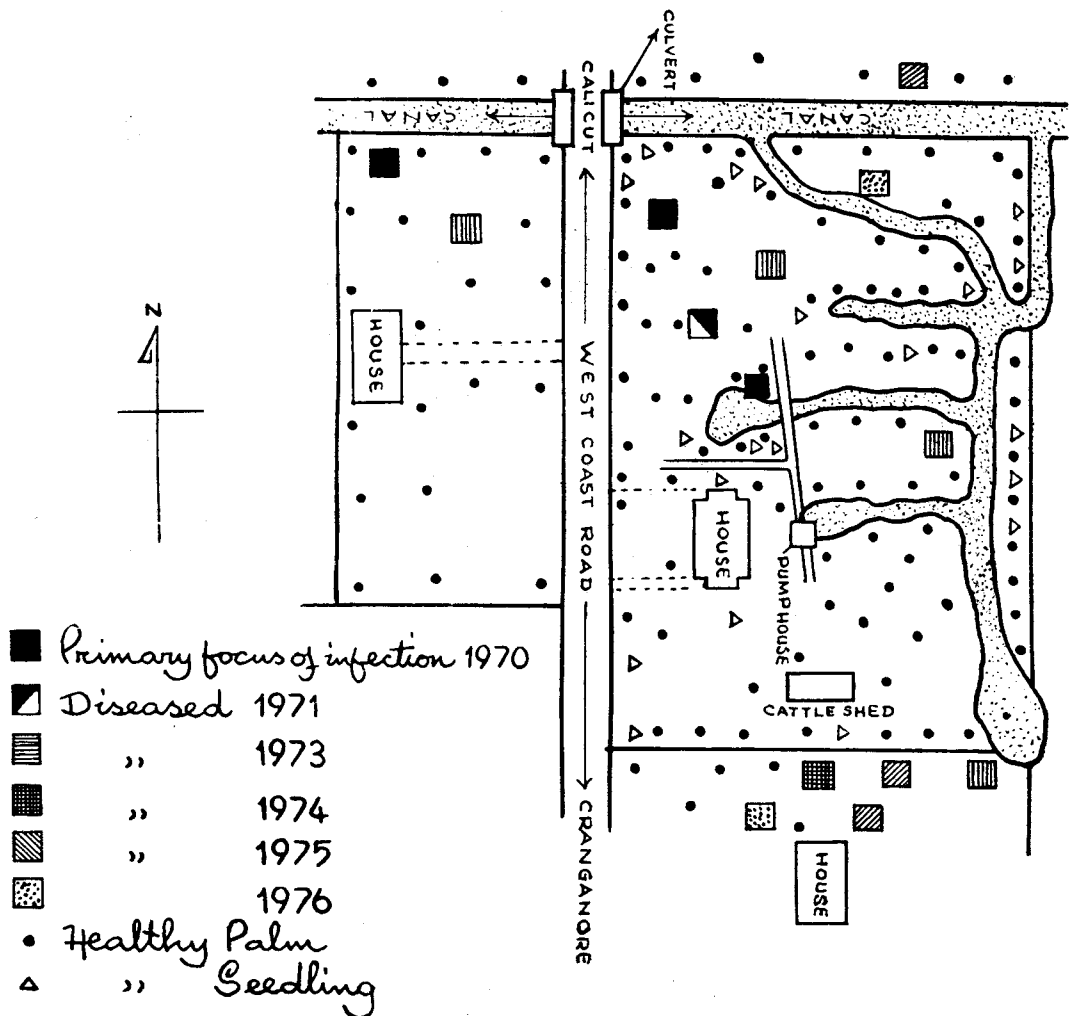


Fig. III Observational Plot—Ala Village Soil type Sandy-loam

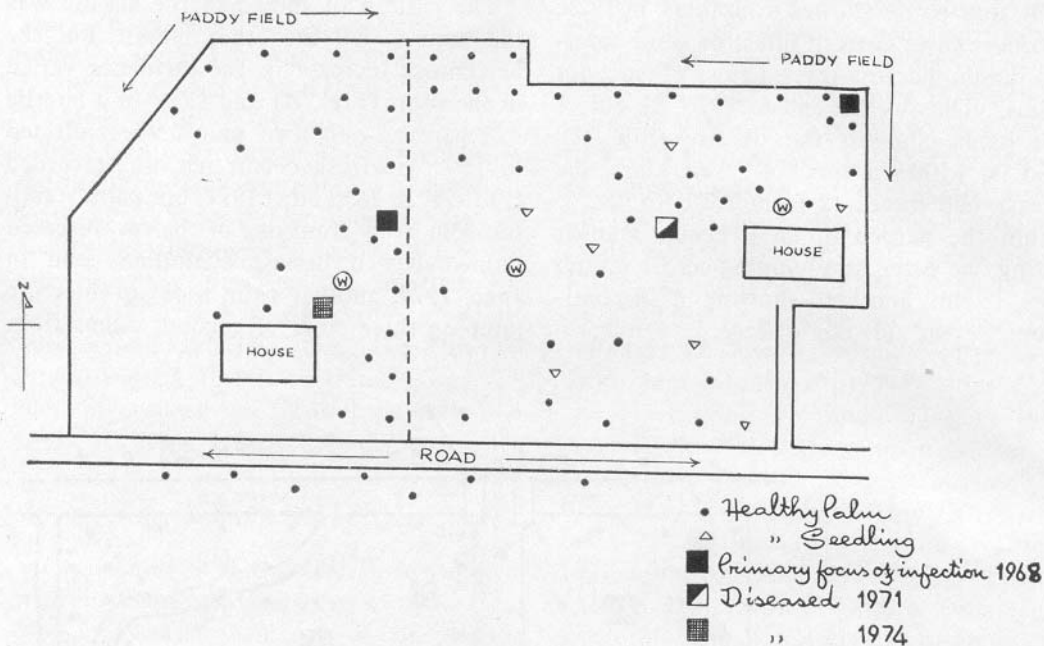


Fig. IV. Observational Plot—Amballor Village Soil types—Laterite.

the second diseased (Fig. IV). From 1969 to 1976, only these two fresh outbreaks could be noticed in the garden showing thereby the slow pace of spread in laterite soil. Probably here, the spread of the disease was limited by a poor source of primary infection and/or by a lack of sufficient number of active vectors.

Although the initial diseased palms of the observational plots were presumably the primary sources of infection, spread from neighbouring plots could not be ruled out.

No palms recovered from the disease in any of the plots during the period of study. On the other hand, there was a slow and steady increase in the disease index scores of the affected palms (Fig. V). The infected palms continued to be productive during the period but their condition deteriorated rapidly when the root (wilt) infected palms were affected by leaf rot (incited by the fungus *Bipolaris halodes* Dresh).

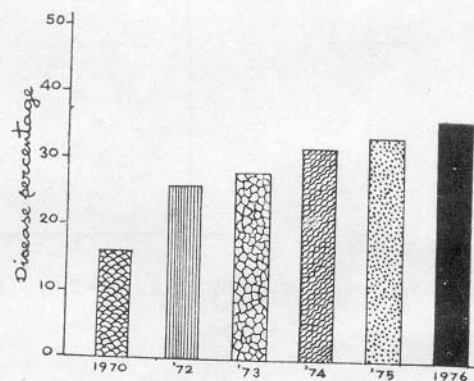


Fig. V. Increase in index series of Root (wilt) disease affected palms in the observational plots. (Average Values).

Rate of spread. This has been very slow (Table I). In gardens of sandy, sandy-loam, and alluvial soils, the disease spread at an average rate of 2-5 palms/year, while in laterite soils, the spread was limited to one palm in two years.

Shanta, Pillai, and Lal (1972) and Gopinathan Pillai, Lal and Shanta (1973) reported rapid and indiscriminate spread of the disease in reclaimed sandy and sandy-loam soils and in water-logged low lying areas. In the present study, the incidence of the disease increased by about 9% in plots having sandy, sandy-loam, and alluvial soils as compared to laterite soils (Table II). Thus, the spread of the disease was comparatively more rapid in alluvial, sandy and sandy-loam soils.

From the three comprehensive garden-to-garden surveys carried out during the study in the transition zone between the affected

areas and disease-free areas in Trichur District the rate of spread was found to be 1-4 km (Table III). Although the spread was almost contiguous in the coastal areas, the disease appeared only sporadically in jumps of several kilometers in the eastern hilly areas. The boundary of the disease as delineated during the study is shown in Fig. VI. The northernmost villages where diseased palms occurred were Ala and Panangad in the coastal area, Keezhapallikara and Oorakom in the midland region, and Nadathra in the eastern hilly area.

The study clearly showed that the coconut root (wilt) disease spreads at only a very slow pace, though the percentage increase in incidence varies in different soil types.

Further, the pattern of spread appears to be erratic and irregular in all the plots (cf. Figs. II-IV). This is suggestive of the involvement of aerial vector(s). Two other major diseases of coconut of uncertain aetiology, the lethal yellowing of West Indies (Heinze, Schuilling, and Romney, 1972; McCoy, 1976) and the cadang cadang disease of the Philippines (Price and Bigornia, 1972) have been also suggested to be transmitted by aerial vectors. But unlike root (wilt), lethal yellowing spreads at a faster rate and kills the palms within 3-15 months of contracting the disease depending on the age of the palms

Table I. Rate of spread of root (wilt) disease

Sl. No.	Soil type	Year of observation	No. Total	infected/ no. of palms
1.	Laterite	1969	4	/208
		1976	8	/207
2.	Sandy	1969	4	/282
		1976	21	/278
3.	Sandy loam	1969	5	/105
		1976	32	/102
4.	Alluvial	1969	6	/310
		1976	41	/302

Table II. Percentage increase in disease incidence in the observational plots

Sl. No.	Soil type	Location of the plot	Percentage disease incidence		Percentage increase over 1969
			1969	1976	
1.	Laterite	Amballoor and Varandarapally	2.03	4.08	2.05
2.	Sandy/Sandy-loam	Ala and Irinjalakuda	2.32	13.42	11.10
3.	Alluvial	Nemum/Cheruvallloor	1.96	13.57	11.61

Table III. Range of spread of coconut root (wilt) disease

Year	Coastal	Midland	Eastern Highland
1968-1971	1 km	3 km	1-3 km
1971-1974	1 km	3 km	4 km*
1974-1976	1-2 km	1-2 km	1 km

*not contiguous but in jumps

(Heinze et al. 1972). Though cadang cadang spreads very slowly, it is a disorder of older palms with maximum percentage of infection seen in palms of the age group 51-60 years (Sill, Bigornia, and Pacombaba, 1963). In contrast, palms of all age groups are equally

susceptible to infection by root (wilt) disease.

The coconut root (wilt) disease has been reported to be soil-borne by several workers (Menon and Shanta, 1962; Holmes, 1964; Holmes, Lal and Shanta, 1965; Shanta et al., 1972; Mathen et al., 1976). Shanta et al. (1972) suggested faster spread of the disease in reclaimed sandy and waterlogged areas. Rapid and indiscriminate spread of the disease in plots with reclaimed sandy and sandy-loam and alluvial soils with comparatively high water table of 1-3 m as observed here is in conformity with the above findings. All these findings suggest that the spread of the disease is most likely to be through soil and/or aerial vector(s).

There have been also reports of the occurrence of diseased palms several km away

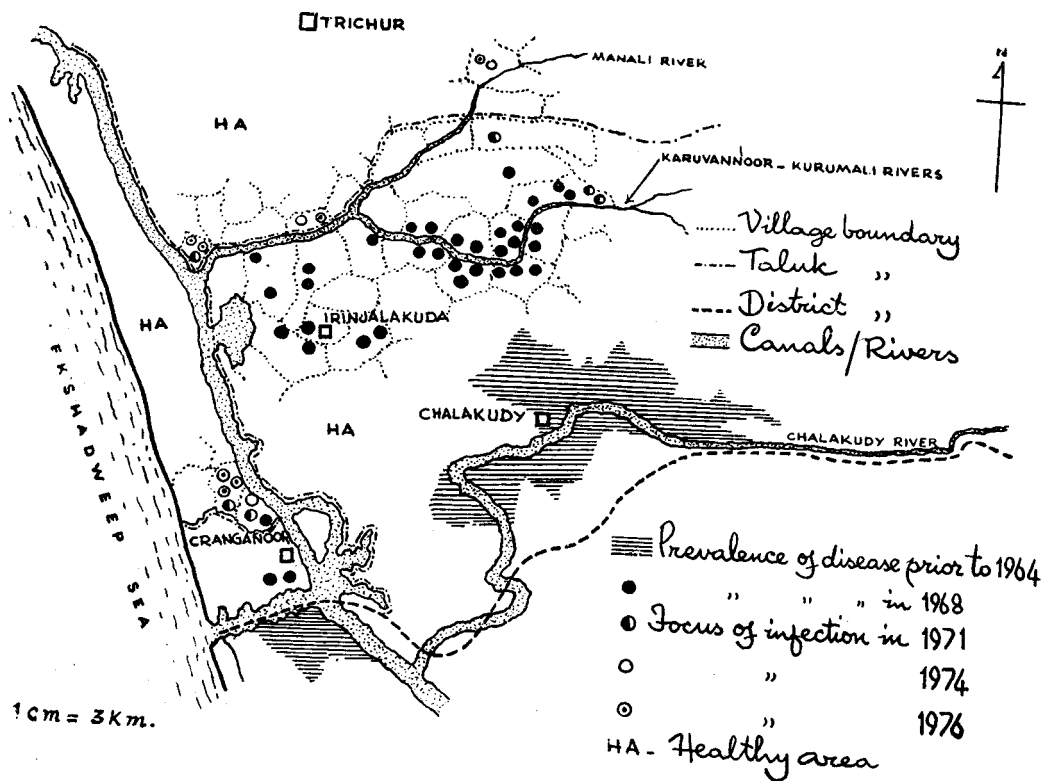


Fig. VI. Spread of Coconut Root (wilt) disease in Trichur Dist. since 1968.

from diseased areas. This suggests transmission of the disease through planting material and/or soil, if any, adhering to them. It also appears possible that the infective principles adhere to soil particles which are then carried to distant places by water during floods and remain viable for years. Miyamoto (1959) has observed this in the case of wheat and barley yellow mosaic viruses.

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REFERENCES

- BUTLER, E. J. 1908. Report on coconut palm disease in Travancore. Bull. No. 9, 23 pp. Agric. Res. Inst., Pusa.
- GEORGE, M. V., THOMMEN, K. J., AND SUKUMARAN, A. S. 1976. Root (wilt) disease and rhinoceros beetle (in Malayalam). *Kalpadhenu* 4: 27-29.
- GEORGE, M., POTTY, V.P. AND JAYASANKAR, N. P. 1976. Association of *Enterobacter* with coconut root (wilt) disease. *Curr. Sci.* 45: 677-678.
- GOPINATHAN PILLAI, N., LAL, S. B., AND SHANTA, P. 1973. Distribution and intensity of root (wilt) disease of coconut in Kerala. *J. Plant. Crops* 1: (Suppl.): 107-112.
- HEINZE, K. G., SCHUILING, M. AND ROMNEY D. H. 1972. The possible cause of lethal yellowing disease of coconut. *FAO Plant Prot. Bull.* 20: 58-69.
- HOLMES, F. O., 1964. Coconut root (wilt) can be controlled. *Coconut Bull.* 18: 368-369.
- HOLMES, F. O., LAL, S. B., AND SHANTA, P. 1965. Cow pea inoculation test for diagnosis of coconut (wilt) disease in India. *FAO Plant Prot. Bull.* 13: 30-34.
- JOSEPH, T. 1978. Some fungi associated with the root system of coconuts in the root (wilt) affected areas. *Curr. Sci.* 47: 586-587.
- MATHEN, K., GOPINATHAN PILLAI, N., MATHEW, A. S. AND SHANTA, P. 1976. Reproduction of symptoms of root (wilt) disease of coconut in potted coconut seedlings. *J. Plant Crops* 4: 78.
- McCOY, R. E., 1976. Comparative epidemiology of the lethal yellowing, kaincope and cadang cadang disease of coconut palm. *Pl. Dis. Reporter* 60: 498-502.
- MENON, K. P. V. AND SHANTA, P. 1962. Soil transmission of the coconut wilt virus. *Curr. Sci.* 31: 153-154.
- MIYAMOTO, Y. 1959. Further evidence for the longevity of soil-borne plant viruses absorbed on soil particles. *Virology* 9: 290-291.
- PRICE, W. C. AND BIGORNIA, A. E. 1972. Evidence for spread of cadang cadang disease of coconut from tree to tree. *FAO Plant Prot. Bull.* 20: 133:135.
- SHANTA, P., MENON, K. P. V., AND PATCHU PILLAI, K. 1960. Aetiology of the wilt (root) disease: Investigations on its virological nature. *Indian Cocon. J.* 13: 56-66.
- SHANTA, P., GOPINATHAN PILLAI, N. AND LAL, S. B. 1972. Additional evidence of soil transmission of coconut root (wilt) pathogen. *Indian J. Agri. Sci.* 42: 623-6326.
- SILL JR. W. H., BIGORNIA, A. E. AND PACOMBABA, R. P. 1963. Incidence of cadang cadang in coconut trees in different ages. *FAO Plant Prot. Bull.* 11: 49-58.
- SUMMANWAR, A. S., RAYCHAUDHURI, S. P. JAGADISHCHANDRA, K., NAMPRAKASH, AND LAL, S. B. 1969. Virus associated with coconut (wilt) disease. *Curr. Sci.* 38: 208-210.
- SRIVASTAVA, D. N., SEKHAWAT, G. S. AND RAO, Y. P. 1969. Association of bacteria with root (wilt) of coconut in Kerala. *Indian J. Agric. Sci.* 39: 395-397.
- VARGHESE, M. K. 1934. Diseases of coconut palm. 105 pp. Govt. Press, Trivandrum.