3.4

ETIOLOGY - VIRUS

T. JOSEPH, N.G. PILLAI and J.J. SOLOMON

Virological investigations on the root (wilt) disease of coconut were initiated following the unsuccessful attempts of Menon and Nair (1951) to reproduce its characteristic symptoms through inoculation of fungal isolates including *Botryodiplodia theobromae* proposed by Butler (1908) as the causal agent. On account of the resemblance of the symptoms to those of the known plant virus disease, Nagaraj *et al.* (1954) suspected virus as the disease incitant.

The virus theory gained significance with positive transmission of the disease through sap inoculation and the insect vector, *Stephanitis typica* Distant under field conditions (Nagaraj and Menon, 1956) and under insect-proof condition. Flaccidity, paling and slight stunting of younger leaves were observed in five of the six seedlings mechanically inoculated with sap of diseased palms and in one out of six seedlings inoculated with *S. typica* (Shanta *et al.*, 1960; Shanta *et al.*, 1964).

Shanta and Menon (1960) reported crinkling and malformation in the first trifoliate leaf of 71.8 per cent cowpea *(Vigna unguiculata* (L.) Walp) mechanically inoculated on the pair of the simple leaves with crude leaf sap of diseased palms. Cowpea plants also became infected when inoculated through viruliferous lace bug, *S. typica.* Even a single insect could be infective and 16 per cent of the field population was found to be viruliferous (Joesph *et al.* 1972). Pillai, *et al.* (1970) observed that pollen, nuts, husk, kernel and embryo of diseased palms had the virus in them as inoculated cowpea developed typical symptoms.

Positive reproduction of symptoms was reported on one out of two coconut seedlings by incorporating diseased roots in the soil under insect proof conditions and in the open, four out of five seedlings grown in sterilised soil interlayered with diseased roots (Shanta *et al.*, 1964; Mathen *et al.*, 1976). Cowpea plants also showed symptoms when grown in soil collected from basins of diseased palms; in sterilised soil watered with infective leaf/root sap and also sterilised soil incorporated with infected roots (Shanta *et al.*, 1972). Cowpea plants grown in the basin of diseased palms also exhibited typical symptoms (Menon and Shanta, 1962; Pillai *et al.*, 1970).

The virus contained in the expressed sap of the diseased palm was named as 'coconut wilt virus' (Shanta and Menon, 1960). Shanta and Menon (1961) studied the physical properties of the virus using cowpea as an indicator plant. The virus had a thermal inactivation point of 76°C for 10 min. and a dilution end point of 10^{-4}. The virus was most active at pH 5 to 9 in the supernatant

solution of the extract. The longevity of the virus was eight weeks in frozen sap and three weeks at room temperature of 28-30°C at dilution of 1:2.5 (Shanta and Menon, 1961). It had a wide host range infecting many species under Leguminosae, Solanaceae and Arecae. The virus, in general caused necrosis, malformation of leaves and stunting in these hosts. Culture of the virus was maintained in cowpea through serial inoculation (Shanta and Menon, 1961).

The positive results of cowpea inoculation tests were confirmed by Holmes (1965) and Holmes *et al.* (1965). Holmes (1965), however, suggested that the sap transmissible agent may be a virus-like organism similar to spirochaete or sporozoa, in view of the peculiar nature of the symptoms on cowpea and lack of proof on its passage through bacterial filters. Production of symptom on cowpea was found to be very inconsistent in later studies. Environmental factors, especially temperature, were suspected to influence symptom expression (Anon., 1971). However, Sasikala and Pillai (1978) after a detailed study on cowpea indicated that it cannot be used as a reliable test plant.

Summanwar *et al.* (1969) isolated a virus from *Chenopodium amaranticolor* Coste & Reynier which they had infected using purified fraction from diseased coconut leaf. The purified fraction caused chlorotic lesions on *C. amaranticolor* and systemic mosaic on *Nicotiana tabacum* cv White Burley. The particles were 320-360 nm in length and 24-25 nm in width. The longevity *in vitro* was nearly one year; the thermal inactivation point 90°C and the dilution end point 10^{-5} to 10^{-6}. The virus was present in leaf and root sap of diseased coconuts but not in leaf, root sap and nut water of healthy palms. Failure of coconut leaf crude sap to infect *C. amaranticolor* was attributed to the presence of viral inhibitors (Summanwar *et al.*, 1971). The rate of infectivity could be improved by addition of 15 per cent lead acetate and 30 per cent polyclar (Summanwar and Gupta, 1976). Based on its positive reaction to antisera of three tobacco mosaic virus (TMV) strains, the virus was classified as TMV (Summanwar *et al.*, 1971). However, Shanta *et al.* (1975) through detailed pathogenicity and EM studies, indicated the non-association of TMV with the root (wilt) disease of coconut. Solomon and Sasikala (1980) also confirmed the non-involvement of TMV with the disease through serology. Maramorosch and Kondo (1977) reported the presence of icosahedral particles of 56 nm diameter. in the epidermis and ground parenchyma cells of diseased palms. But, Parthasarathy (1978) identified these particles as plasmodesmata sectioned in tangential plane.

Failure to observe any virus particle associated with the root (wilt) disease of coconut and the inconsistency in the response of cowpea to the root (wilt) pathogen presently stand out against a virus etiology for the disease.

REFERENCES

ANONYMOUS, 1971. Annual Report for 1969-70. Central Plantation Crops Research Institute, Kasaragod, India. pp. 198.

BUTLER, E.J. 1908. Report on coconut palm disease in Travancore. *Agric. Res. Inst. Pusa. Bull.* No. **9** : 1-23.

HOLMES, F.O. 1965. Investigation on the etiology of coconut root (wilt) disease. Report to the Government of India, Report No. 1958. FAO of the UN. pp.13.

HOLMES, F.O., LAL, S.B. and SHANTA, P. 1965. Cowpea inoculation test for diagnosis of coconut wilt disease in India. *FAO Pl. Prot. Bull.* **13**:30-34.

JOSEPH, T., SHANTA, P. and LAL, S.B. 1972. Role of *Stephanitis typicus* Distant in the spread of coconut root (wilt) pathogen. *Indian J. Agric. Sci.* **42**: 414-417.

MARAMOROSCH, K. and KONDO, F. 1977. Electron microscopy of leaf sections from Kerala wilt diseased coconut palm. *J. Plantn. Crops* **5** : 20-22.

MATHEN, K., PILLAI, N.G., MATHEW, A.S. and SHANTA, P. 1976. Reproduction of symptoms of root (wilt) disease of coconut in potted coconut seedlings. *J. Plantn. Crops* **4** : 78-79.

MENON, K.P.V. and NAIR, U.K. 1951. Scheme for the investigation of the root and leaf diseases of the coconut palm in South India. Consolidated final report of work done from 8th March, 1937 to 31st March, 1948. *Indian Cocon. J.* **5** (1) : 5-19.

MENON, K.P.V. and SHANTA, P. 1962. Soil transmission of the coconut wilt virus. *Cur. Sci.* **31** : 153-154.

NAGARAJ, A.N. and MENON, K.P.V. 1956. Note on the etiology of the wilt (root) disease of coconut palms in Travancore-Cochin. *Indian Cocon. J.* **9** : 161-165.

NAGARAJ, A.N., DAVIS, T.A. and MENON, K.P.V. 1954. Sap transfusion, a new device for virus transmission trials in palms. *Indian Cocon. J.* **7** (3) : 91-95.

PARTHASARATHY, M.V. 1978. Have virus like particles been found in leaf of Kerala wilt diseased coconut palms? Not yet. *J. Plantn. Crops* **6** : 87-89.

PILLAI, N. G., SHANTA, P. and LAL, S.B. 1970. Distribution of root (wilt) pathogen in developing seed coconut. *Cur. Sci.* **39**:188-189.

SASIKALA, M. and PILLAI, N.G. 1978. Transmission of coconut root (wilt) disease - Reaction of 170 test plants. *J. Plantn. Crops.* **6** : 17-20.

SHANTA, P. and MENON, K.P.V. 1960. Cowpea (*Vigna sinensis* Endl.) as indicator plant for the coconut wilt virus. *Virology* **12** : 309-310.

SHANTA, P. and MENON, K.P.V. 1961. Studies on some properties of coconut wilt virus. *Indian Cocon. J.* **15** : 36-46.

SHANTA, P., HARIHARASUBRAMANIAN, V. and PILLAI, N.G. 1975. Possible association of tobacco mosiac virus with

the root (wilt) disease of coconut. *J. Plantn. Crops* **3** : 77-80.

SHANTA, P., JOSEPH, T. and LAL, S.B. 1964. Transmission of root (wilt) disease of coconut. *Indian Cocon. J.* **18** : 25-28.

SHANTA, P., MENON, K.P.V. and PILLAI, K.P. 1960. Etiology of root (wilt) disease : Investigations on its virological nature. *Indian Cocon. J.* **13** : 56-60.

SHANTA, P., PILLAI, N.G. and LAL, S.B. 1972. Additional evidence of soil transmission of coconut root (wilt) pathogen. *Indian J. Agric. Sci* **42** :623-626.

SOLOMON, J.J. and SASIKALA, M. 1980. A serological appraisal of the connection of the tobacco mosaic virus isolate with the root (wilt) disease of coconut. *Phytopath. Z.* **99** : 26-32.

SUMMANWAR, A.S. and GUPTA, M.D. 1976. A quick method for isolating coconut root (wilt) virus from diseased palms. *Indian Phytopath.* **29** : 108.

SUMMANWAR, A.S., RAYCHAUDHURY, S.P. and JAGADISH CHANDRA, K. 1971. Further studies on coconut root (wilt) disease. 2nd *International Symp. on Pl. Path.* January, 27th to Feb. 3rd.

SUMMANWAR, A.S., RAYCHOUDHURY, S.P., JAGADISH CHANDRA, K., NAMPRAKASH and LAL, S.B. 1969. Virus associated with coconut root (wilt) disease. *Cur.Sci.* **38** : 208-210.