MYCOPLASMA DISEASES OF COCONUT WITH SPECIAL REFERENCE TO ROOT (WILT) DISEASE.

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IN

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The coconut palm (Cocos nucifera Linn) is prone to several diseases the etiology of which has been elusive. But intensive research carried out during the past few years has improved the situation to a considerable extent resulting in a breakthrough in the understanding of the etiology of some of these diseases. The Red Ring disease in the Tropical American region is now known to be caused by a nematode Rhadinaphelenchus cocophilus which is present in a systemic manner in all the parts of the palm. The Cedros Wilt in Trinidad, Hart Rot of Surinam, Ecuador, Brazil and Trinidad are associated with phloem restricted Trypanosomatid flagellate Phytomonas species (Parthasarathy et al., 1976). Rickettsia-like organisms are implicated in coconut palms afflicted by the New Disease in Tanzania (Steiner et al., 1977). The Cadang Cadang disease in the Philippines is suspected to be incited by a viroid (Randles et al., 1977). Lethal Yellowing disease causing serious threat to coconut cultivation in Jamaica, Florida, Cuba, Africa etc. has been found to be due to mycoplasma-like organisms (Plavsic-Banjac et al., 1977). More recently, MLOs have also been attributed as the causative agent for Coconut Stem Necrosis in North Sumatra and Peninsular Malaysia (Turner et al., 1978).

Mycoplasmas are procaryotes resembling bacteria without a defined nucleus but only DNA strands as genetic material and ribosomes. They are smaller than bacteria and larger than viruses and are bound by a triple layered unit membrane (devoid of a defined cell wall), highly pleomorphic and can normally be located only under an electron microscope. Mycoplasmas are filterable. They have been known to induce diseases in animals and human beings as early as in 1898. But it was only in 1967 organisms resembling mycoplasma were

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located with the aid of electron microscope, in the phloem of plants infected with several yellows type diseases which were till then thought to be caused by viruses. MLOs were also encountered in the insect vectors known to transmit these diseases. In nature, the mode of transmission of these organisms is through insect vectors (normally phloem feeders) generally by leaf hoppers and in a few cases by psyllids. At present, MLOs have been observed in over 150 plant diseases. In spite of numerous attempts, in vitro culturing has been successful only in a few cases and so is the limited success in transmission experiments. Pathogenicity has been successfully proved, satisfying Koch's postulates, only with respect to citrus stubborn, citrus little leaf, corn stunt and disease of cactus. In the case of LY, Howard et al. (1983) reported successful transmission of LY to coconut seedlings through Myndus crudus, a leafhopper. Efforts are in progress to isolate and culture the MLO associated with LY. MLOs are sensitive to tetracycline group of antibiotics. Direct injection into the plant and treatment during early stage of the disease have been observed to be promising.

As far as coconut is concerned, mycoplasmal etiology has been attributed to the lethal yellowing disease (known to occur in Jamaica, Florida, Cuba, Dominican Republics, Haiti Bahama Islands), Kaincope disease (Togo), Cape St. Paul Wilt (Ghana), Kribi disease (Cameroon), Awka wilt (Nigeria) and coconut disease prevailing in Tanzania, all of which are believed to be identical in terms of symptomatology (Maramarosch and Hunt, 1981). The coconut stem necrosis (Sumatra and Malaysia) with a different pattern of symptom is also attributed to the MLOs (Steiner, 1978).

In an excellent article, Ennis Jr. (1982) has summarised the status of research on lethal yellowing. The affected palms in the early stages of infection are characterised by necrosis of inflorescence and spear leaf and immature

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nutfall. This is followed by progressive yellowing of the foliage and defoliation of dead fronds. Ultimately the trunk snaps off below the growing point with the main stem only remaining. This results in the appearance of "telephone pole-like" coconuts stems in the affected plantations. In Florida, it is reported that stomatal closure was the early physiological symptom. The disease is lethal in that about 4-10 months elapse between the appearance of first symptom and death of palms.

Remission of symptoms has been brought about with respect to lethal yellowing selectively in palms given injections of aquous solution of tetracycline in United States and Jamaica. The treatment should be done at the earliest onset of symptoms to get the desired result. This does not guarantee freedom from the disease since the symptoms reappear when injections are stopped. According to Ennis (1982) continuous injections at four-month intervals resulted in palms being free from disease upto 7 years. Similar remission of symptoms was noticed in palms affected by Kain Cope disease consequent on Tetracycline therapy in Togo. The ultimate and best solution to LY is to exploit genetic resistance. Excellent field resistance has been shown by Malayan Dwarf and hybrid "Maypan".

The coconut Stem Necrosis, on the other hand, has a discoloured spear leaf which turns brown and dies showing light brown necrotic flecking. The change in colour from green to brown is direct without a marked transition-yellowing symptomatic of lethal yellowing. The older fruits successively die with a similar shift in colour from green to brown. The palm dies in about five months after the appearance of the first symptom. By the time the external symptoms become manifested, extensive necrosis occurs within the stem, both above and below the apical bud. Mycoplasma induced diseases

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in general are non-lethal but affect the productivity of the crop by way of induced sterility, metamorphosis of floral parts, yellowing, stunting etc. Exception to this pattern is observed in lethal yellowing and stem necrosis where the rapid death of the palm is imminent.

Though the lethal yellowing type of disease of coconut in the Caribbean area and the West African Coast have similarity in the nature of symptoms and both are suspected to be MLO induced diseases, there is marked difference in the pattern of spread and in the resistance of varieties/hybrids to the diseases. In the former the pattern of spread is characterised by leaps or jumps suggestive of the involvement of aerial vectors. This situation is comparable to that observed in root (wilt) disease. In the Kaincope disease of coconut the spread is continuous from a single focus of infection like an oil stain resembling a soil borne disease. Moreover, the Malayan Dwarf which is considered to be resistant/tolerant to lethal yellowing succumb to MLO diseases in Africa so also the Mava hybrid to stem necrosis of Malaysia and Sumatra. Possible strainal variation in the MLOs may be one of the reasons for this differential behaviour.

With reference to the coconut root (wilt) disease, the important foliar symptoms are the flaccidity of the leaflets (which is most consistent), yellowing of leaves and marginal necrosis of leaflets. The root (wilt) disease is only debilitating unlike lethal yellowing (Jayasankar and Radha, 1982).

Recently, MLOs have been detected under electron microscope in ultra thin sections of developing leaves, unopened inflorescences, root tips, and terminal bud tissues (in the sieve tubes of phloem) in coconut root (wilt) affected palms and were conspicuously absent in samples from althy palms. No virus-like particles or microorganisms

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other than MLOs were made out in these ultra thin preparations (Solomon et al. 1983). Sample preparations and ultra microtomy were performed at the CPCRI Regional Station, Kayangulam and ultrathin sections were examined under electron microscopes at the Christian Medical College, Vellore and the Cotton Technological Research Laboratory, Bombay. Representative samples of four disease affected palms were also sent to the Institute for Plant Diseases, Bonn, West Germany for electron microscopic studies where again the presence of MLOs was observed in disease affected palms.

Numerous attempts to control coconut root (wilt) disease with plant protection chemicals have been made in the past. Neither pesticides nor major and micronutrients altered the course of disease contraction and symptom development. Elaborate studies are in progress with antibiotics like oxytetracycline hydrochloride through stem injection.

Based on the results obtained at the Institute, an adaptive research programme was started for containing the disease in collaboration with Kerala Agricultural University, Kerala Agricultural Department and Coconut Development Council. Seven hundred diseased palms in 309 gardens of 14 villages were eradicated in three years. A scrutiny on the recurrence of disease in 156 gardens where 300 disease affected palms were uprooted and burnt in 1979 showed that 90% of the gardens remained free of disease in 1982 indicating thereby the effectiveness in eliminating the foci of infection by the systematic eradication programme.

Since the disease affected palms respond well to management practices, it is possible to live with the disease if proper care and management are given to these palms. The recent research results conducted have shown that regular

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recycling of organic matter by way of mixed farming increased the yield by 26,1 per cent. Growing intercrops like Elephant foot yam and yam and mixed cropping with cacao had also resulted in the increased yield and income, besides improving the soil fertility. The D x T hybrid under good management condition had resulted in the increased yields and income when compared to the local WCT. The disease incidence was also comparatively low in D x T. A detailed programme of screening cultivars with emphasis on yield and disease resistance/tolerance is in progress under better management for root (wilt) disease.

In the light of the present level of understanding, the following lines of investigations are necessary for tackling the root (wilt) disease:

1. Isolation, culture and characterisation of mycoplasma pathogen
2. Transmission of the disease through biological agents and detection of the pathogen in their tissues using electron microscope
3. Study of the biology and bionomics of the insect vector(s)
4. Devising serological and other biochemical methods to radpidly detect the pathogen in the plant
5. Chemotherapy through antibiotics
6. Containing the disease through eradication of infected palms
7. Management of the disease
8. Field evaluation of coconut germplasm for resistance/ tolerance

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