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REVIEW ARTICLE

Transmission of coconut root (wilt) disease through plant hopper, *Proutista moesta* Westwood (Homoptera:Derbidae)*

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INTRODUCTION

Root (wilt) disease, a non-lethal but debilitating malady of coconut palm was first reported from Kerala State, India about a century and quarter years ago. The spread of the disease in the course of about 125 years in the past from the original three independent foci of incidence to a contiguous tract of 4,10000 ha covering eight southern districts of Kerala (CPCRI, 1985) is sufficient proof for the transmissibility of the disease under field conditions. Sparse and sporadic occurrence of the disease was also reported from other six northern districts of Kerala (Radha *et al.,* 1985; Solomon *et al.,* 2001) and from adjoining areas of neighbouring states of Tamil Nadu (Srinivasan *et al.,* 2000) and Karnataka (Sasikala *et al.,* 2005) indicating the slow spread of the disease to the entire state of Kerala and neighbouring states.

NATURE AND SPREAD

The most obvious and diagnostic symptom of the disease is the abnormal bending of the leaflets termed ribbing or flaccidity. Foliar yellowing of the outer whorl of leaves and marginal necrosis are the other associated symptoms (Radha and Lal, 1972). In seedlings and juvenile palms yellowing of foliage is virtually absent and flaccidity is the only symptom evident. The disease affects palms of all age groups. About 65% of the root (wilt) diseased palms are affected by leaf rot disease caused by fungi (Srinivasan, 1991) and contributed to the rapid decline and reduction in the yield of the affected palm. The annual loss due to the disease was estimated to be about 968 million nuts amounting to 3000 million rupees (CPCRI, 1985). The reduction in the yield of nuts up to 80% has been reported in the advanced stages of the disease.

The root (wilt) disease occurs in all major soil types but the spread is faster in sandy, sandy loam and alluvial soils. The spread of the disease was erratic and irregular irrespective of soil conditions. The rate of spread of the disease was 1-4 km over a period of three years from the nearest source of infection. Epidemiological investigations revealed that the pattern of spread occurs in jumps or leaps suggestive of possible involvement of aerial vectors in the transmission of the disease (Pillai *et al.* 1980).

INVENTORY OF PUTATIVE VECTORS

Experimental transmission studies conducted in the past proved the transmission of the disease through the lace bug, *Stephanitis typica* (Distant) [Heteroptera : Tingidae] in the field (Nagaraj and Menon, 1956; Shanta *et al.,* 1960) and in the insect proof house (Shanta *et al.,* 1964). These results were reported when a virus etiology of the disease was suspected. Report of Solomon *et al.* (1983) on the association of Phytoplasma with the disease warranted a reinvestigation on the vectoral ability of the lace bug, *S. typica* to imbibe and to transmit the phloem bound mollicute since phytoplasma diseases are not known to be transmitted by true bugs (Heteropteran insects). The detection of phytoplasma in salivary glands and brain tissue of lace bugs, *S. typica* offered acquisition access and incubation period on root (wilt) diseased palm (Mathen *et al.,* 1987), and experimental transmission of phytoplasma from diseased palm to healthy two-year old coconut seedlings under insect proof conditions confirmed its vectoral role.

Phytoplasmas are mostly transmitted by leaf hoppers and planthoppers (Auchenorrhyncha) and rarely by psyllids. Record of insect fauna on coconut (Kurian *et al.,* 1979), however, did not include insects belonging to Auchenorrhyncha from India. Therefore, a systematic inventory of insects in root (wilt) prevalent gardens was made using various traps *viz.*, rotary trap, suction trap, light trap and sticky traps and confirmation of their

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occurrence in coconut foliage by direct examination of 500 young coconut seedlings over a period of two years. As a result, besides lace bug, a plant hopper, *Proutista moesta* (Westwood) and a leaf hopper, *Sophonia greeni* (Distant) were recorded (Rajan and Mathen 1984; 1985) to be included as putative vectors of the disease.

A rapid survey of the contiguous disease tract and freshly reported pockets of disease incidence indicated the presence of all three putative insect vectors. There was no disease occurrence independent of all the three putative vectors (Rajan and Mathen, 1985).

BIOLOGY

The planthopper, *Proutista moesta* breeds in decaying organic matter in the soil and only adult insects are seen feeding on the leaves of coconut palm. The bunch waste of oil palm was found to be a good substrate for breeding this insect. Egg to adult period of this insect was completed in 28-35 days with five instars having an average incubation period of 7 days and nymphal period of 25 days. Adult longevity ranged between 50-60 days. Adult lays on an average of 45 eggs with sex ratio of 1 : 0.59. The population build up of plant hopper was noticed with onset of summer showers in late May-June and highest population was recorded during October-November (Solomon, 2001). Alternate hosts of the insect include arecanut, oil palm and sugarcane. Planthoppers are occasionally observed on banana, jowar, maize, rice and Napier grass.

The leafhopper, *Sophonia greeni* breeds on tender leaves of the palm and its population was found maximum during July-August. Alternate hosts of the leafhopper include *Areca catechu*, *Areca triandra* and *Solanum melongena*.

FEEDING HABITS

The planthopper, *P. moesta* and leaf hopper, *S. greeni* are phloem feeders. The feeding of the former species in coconut is confined to the leaflets of middle and outer whorls while the latter species prefers tender fronds of coconut. Both these insects suck sap from the abaxial surface of leaflets and no feeding marks were observed in leaflets due to their feeding (Rajan, 1996).

ACQUISITION STUDIES WITH INSECT VECTORS

The potential of these three insects to acquire the phytoplasma was verified electron microscopically. The insects were subjected to detailed studies for the ability to acquire the phloem bound phytoplasma from diseased coconut palms. Acquisition access and incubation periods (A+I) of various combinations (no. of days) were offered to these insects by caging them in situ on the leaves of diseased palms in which the presence of phytoplasma was confirmed through EM studies. Phytoplasma has been observed in the salivary glands of *Proutista moesta* given an acquisition plus incubation period of more than 30 days on diseased palms (Rajan *et al.*, 2002). Phytoplasma was not observed in planthoppers collected from disease free areas such as Kasaragod and Minicoy in Lakshadweep and those insects offered acquisition plus incubation periods of less than 30 days. Presence of phytoplasma in insects that were fed on diseased palms clearly indicates that they were able to acquire the pathogen and sustain its multiplication inside their body. No lead could be obtained with leafhoppers in acquisition studies carried out.

EXPERIMENTAL TRANSMISSION STUDIES USING INSECT VECTORS

The experiment on transmission of root (wilt) disease was carried out under insect proof field cages using infective planthoppers. Coconut seedlings from a disease free area were planted in methyl bromide fumigated loamy sand soil collected from paddy field, held in field tanks of 1.8 x 1.8 x 1.2m and protected by insect proof cages.

In transmission studies using planthoppers, eight seedlings were inoculated with infective plant hoppers and 8 seedlings were kept as uninoculated control. Phytoplasma was observed in six out of eight planthopper inoculated seedlings 5-24 months after inoculation as evidenced by electron microscopic examination and serological tests. Two seedlings remained free from contraction of the disease till the closure of the experiment. Five out of six seedlings which have showed the presence of phytoplasma developed flaccidity of leaflets, the decisive symptoms of root (wilt) disease within 8-24 months after the first inoculation. The seedling which had expressed visual symptoms in 8th month had received by then an insect load of 610 infective plant hoppers. Uninoculated control plants remained free of the organisms (phytoplasmas) and symptom of disease. This experiment has conclusively proved the role of plant hoppers in the transmission of root (wilt) disease (Rajan *et al.*, 2000; Solomon, 2001).

The vector role of plant hopper *Proutista moesta* in root (wilt) disease gains significance in the light of its implication as the vector of other phytoplasmal diseases in Kerala *viz.*, yellow leaf disease of arecanut (Ponnamma *et al.*, 1991; 1997) and spear rot disease of oil palm (Kochu Babu, 1993; CPCRI, 1994). In sugarcane grassy shoot disease also *P. moesta* is implicated as an insect associated with the secondary transmission of the

disease (Edison *et al.,* 1976). Phytoplasma associated with the lethal yellowing of coconut in Florida was transmitted by planthopper, *Myndus crudus van* Duzee (Homptera : Cixiidae) (Howard *et al.,* 1983; 1984). Lethal yellowing group phytoplasma was detected in planthopper, *Cedusa* sp. (Homoptera : Derbidae) from Jamaica by nested-PCR assay (Brown *et al.,* 2006).

Feeding on the phloem by an insect vector in a non-destructive manner accounts for the efficiency of the vector in transmission of phytoplasma. Usually homopterans are non-destructive feeders of phloem and heteropterans have destructive feeding pattern (Weintraub and Beanland, 2006). Hence, vector efficiency of plant hopper, which is a non-destructive feeder of phloem gains more importance in the transmission of coconut root (wilt) disease.

Leach (1940) laid down four requirements to provide adequate proof for insect transmission of plant diseases. They are 1) Demonstration of a close though not necessarily constant association of the insect with the diseased plants; 2) Regular visits to healthy plants by the insects; 3) Presence of the microorganism associated with the diseased plant in the insect following visits to diseased plants and 4) production of disease in experimental plants under controlled conditions as a result of inoculating with infective insects with sufficient checks. Plant hoppers are present as minor pests on coconut foliage throughout the year. The disease does not occur independently of insect vectors as per the results of the survey undertaken in eight districts of disease prevalent areas and isolated disease prevalent gardens beyond the contiguous areas of disease incidence. Healthy palms of disease free areas and apparently healthy (symptomless) palms of diseased tracts also harbour these insect vestors. The presence of phytoplasmas in the salivary glands of these insects were detected by EM examination of tissues when they were exposed to the diseased palms for the completion of required A+I periods and their total absence in insects from disease free areas. The experimental palms inoculated with infective insects developed flaccidity of leaflets, the diagnostic symptom of the disease and un-inoculated control plants were free of the disease. Tissues of the experimental palms inoculated with infective insects demonstrated positive results in serology and EM studies, whereas in control plants the results were negative. The norms of the acceptable insect transmission have thus been fulfilled.

VECTOR CONTROL STUDIES

Knowledge about the vector(s) is useful in attempts to regulate the disease through vector control. With this objective seven field experiments were conducted during 1985 to 1997 attempting insecticidal control of vectors on newly planted seedlings and source palms in the endemic/ mildly affected areas of the disease. Insecticides *viz.,* endosulfan and monocrotophos were sprayed at different intervals *viz.,* fortnightly, monthly, quarterly and half-yearly to the experimental seedlings. In one experiment, instead of spraying, soil application of systemic granular insecticides *viz.,* phorate and carbofuran and botanicals like neem cake were applied thrice a year. Though reduction in population of vectors could be obtained, regular spraying could not give prevention of fresh incidence of disease and spread on new plantings (CPCRI, 1993; 1997). Similar results were recorded in lethal yellowing disease in Florida. Biweekly spraying of insecticides resulted only in slight reduction in vector population and the spread of lethal yellowing disease (Howard and McCoy, 1980; Howard and Barrant, 1989). They are of the opinion that insecticides currently in use are not persistent and thus treated palms are quickly re-infested resulting in disease contraction. The perennial nature of the crop, persistent mode of transmission and presence of insect vectors almost throughout the year in coconut were attributed as the reasons for non-effectiveness of the insecticides. Conventional insecticides, even when frequently used could not control the appearance of the disease because the pathogen transmission occurs faster than insecticides can act, and there is always a constant influx of new vectors from surrounding habitats (Weintraub, 2007).

Coconut root (wilt) disease is caused by phytoplasma and is transmitted in nature by insect vectors *viz.,* lace bug, *S typica* and plant hopper, *P. moesta* as evidenced by experimental transmission studies under controlled conditions. Phytoplasmas were also detected from tender tissues of diseased seedlings in the experimental transmission studies and infective vectors through EM, LM and serological studies. Vector control trials to manage the root (wilt) disease could not give desired results as far as regulation and spread of the disease is concerned. In this context, breeding coconut cultivars / hybrids for resistance/ tolerance against the disease emerges as the most important strategy to achieve a permanent solution to this problem.

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