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# IS WATER STRESS ASSOCIATED WITH ROOT (WILT) DISEASE OF COCONUT?*

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ABSTRACT

**The wilt disease of coconut in India popularly known as the root (wilt) disease, has flaccidity of leaflets as the primary symptom, while leaf yellowing and necrosis are the other associated symptoms of the disease. Diseased palms are characterized by damaged root system and impaired stomatal regulation. Mycoplasma-like organisms (MLOs) are found to be the causal organisms of the disease. The water relation studies revealed the complexity of the disease, which is the only 'wilt' disease on record associated with the MLOs. The present review dwells on the recent investigations on root (wilt) disease, in relation to water stress besides the comparison between the root (wilt) and the lethal yellowing, the two widely known diseases of coconut.**

## INTRODUCTION

Although Kerala ranks first with regard to area under coconut, the productivity is lower than that in the two adjoining States of Tamil Nadu and Karnataka. Coconut palms in Kerala are afflicted by a serious malady characterized by wilting of the crown and is popularly described as root (wilt) disease, (RWD) (Mathen, Gopinathan Pillai and Radha, 1983) or Kerala wilt (Maramarosch, 1964). This disease is wide spread in the eight southern districts of Kerala, with sporadic occurrences in some northern districts of the state and also in some districts in the adjoining Tamil Nadu. Reports indicate that the disease is prevalent in all the major soil types in the State (Gopinathan Pillai, Lal and Shantha, 1973). Relatively higher incidence of the disease was reported in water-logged, low-lying areas adjacent to rivers and canals. Palms of all ages are found to be susceptible to the disease.

Extensive investigations during the last two decades have excluded nutritional deficiency, hormonal imbalance or water stress as factors directly involved in symptom expression of the disease (Anonymous, 1981). The disease is pathogenic in nature. Systematic studies have ruled out the involvement of fungi, bacteria or viruses with the disease. Electron microscopic investigation carried out during 1981-1983 have revealed the association of MLOs with the disease (Solomon, Govindankutty and Neinhaus, 1983) and further confirmations were obtained through the transmission studies (Anonymous, 1986). Unlike the Lethal Yellowing Disease (LYD) of coconut in Jamaica, which also is caused by MLOs (Beakbane, Slater and Posnetti, 1972; Plasvic-Banjac, Hunt and Maramarosch, 1972; Parthasarathy, 1974), the RWD of Kerala is only a debilitating disease. Marked decline in nut yield due to RWD has been reported (Anonymous, 1985). However, palms in early stages of the disease have been shown to respond favourably, both in terms of decrease in disease intensity and nut yield, to a set

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of management practices in the field (Muraleedharan, Gopalakrishnan Nair and Jayasankar, 1986; Rajagopal, Patil and Sumathykutty Amma, 1987.

**Disease symptoms**

The disease is characterized by flaccidity of leaflets of middle and outer whorl of leaves as the primary symptom (Radha and Lal, 1972). To begin with, the upper whorl of leaves show flattening and bending of leaflets, progressively leading to drooping or wilted appearance at the time when the leaves reach middle whorl. In the advanced stage of the disease, the bending of leaflets from two sides of the midrib give the appearance of ribs of a human skeleton. Premature yellowing of older leaves, necrotic spots on the middle or outer whorls of leaves, drying up of spadices and premature shedding of nuts are the associated symptoms (Anonymous, 1981). In general, 67 to 98% palms had flaccidity and 38 to 74% had yellowing, whereas necrosis occurred only in 28 to 48% palms. Intensity of the symptoms varied according to age of the palms. Trees below ten years had rarely exhibited yellowing, but with advancement in age yellowing and necrosis became prominent. Softening and whitening of the central shoot was also reported as characteristic disease symptoms (Dwivedi et al., 1979). Based on the intensity of the three foliar symptoms *viz.*, flaccidity, yellowing and necrosis, the disease index (DI) could be computed and palms categorized as apparently healthy (DI$<$10%), diseased early (DI 11 to 25%), middle (DI 26 to 50%) and advanced (DI $>$50%) (George and Radha, 1973).

**Root system**

As the name of the disease itself implies the root system of diseased palms was damaged in as much as the number of active roots and diameter of the bole (lower part of the stem) were drastically reduced (Nagaraj and Menon, 1955; Menon and Pandalai, 1958; Michael, 1964). The percentage of dead roots was higher in diseased palms than in healthy ones. Roots of RWD palms also exhibited poor regeneration capacity. Indira and Ramadasan (1968) recorded disintegration of vascular tissues in 60% of the roots from RWD palms, as compared to 33% in roots from the apparently healthy palms. This is reflected on the appearance of foliar symptoms in the former. Majority of diseased roots had tyloses in the vessels and were characterized by deranged permeability. Root sap from diseased palms contained 65 to 72% more dry solids than that from healthy palms. However, Joseph and Jayasankar (1981) reported that root rot was not the characteristic symptom of the disease.

**Water uptake**

The uptake of water is affected in RWD palms. While a single root of a healthy palm could absorb 250 to 500 ml day$^{-1}$, that of a diseased palm could take upto 150 ml or less day$^{-1}$ (Davis, 1964). The uptake and upward transport of water through the trunk in diseased palms was 35% less than that of healthy palms (Ramadasan, 1964). Excised roots of infected palms had poor water absorption capacity (Mathew, 1968). The profile of soil moisture content in the basins of healthy and diseased palms, at one day interval for five days after irrigation, also indicated that healthy palms caused greater depletion of water than RWD palms, reflecting thereby the poor uptake of water by the root system of the latter (Rajagopal et al., 1986). The absorption of radio active phosphorus by roots and its accumulation in leaves was significantly lower in RWD palms than in healthy ones, indicating poor absorption and translocation in the former (Dwivedi, Ray and Ninan, 1979). A similar study with $32_P$ revealed a decreased water transport in LY diseased coconut palms (Eskafi, Basham and Mc Coy, 1986).

Further evidence on the root damage or derangement was provided by the biochemical analysis of roots. Roots of diseased palms had greater depletion of carbohydrates (Mathew, 1977), reduced C/N ratio (Varkey, Michael and Ramadasan, 1969) and increased respiration rate (Michael, 1978), as compared to roots of healthy palms. Higher activities of polyphenol oxidase and peroxidase (Joseph, Potty and Jayasankar, 1976), cellulase (Padmaja and Sumathykutty amma, 1979) and pectinlyase (Sumathykutty amma and Patil, 1984) were recorded in the roots of RWD palms than in those of healthy palms.

**Leaf characteristics**

The deterioration in the structure and function of roots of RWD palms is associated with several abnormalities in leaves. In the diseased leaf, degeneration of chloroplasts, less cuticular deposition and reduced lignin content in the cell wall were reported (Shantha, Menon and Patchu Pillai, 1959; Shantha, Joseph and Lal, 1964; Govindankutty, 1981). Mature leaflets of diseased palms were thinner, measuring 265 to 295 $\mu$ in cross section, than those of healthy palms which measured 365 to 380$\mu$ (Govindankutty, 1981). Transverse division of upper epidermal cells in RWD palms are accelerated and longitudinal division curtailed. These differences in the rate of cell divisions in the two planes resulted subsequently in the slight downward curling of leaflets. There was reduction in the wall thickness of cells in mechanical sclerenchymatous fibres and bundle sheaths, leading to loss of rigidity in hypodermis of the leaflets of diseased palms. RWD palms had higher stomatal frequency than the healthy palms (Mathew, 1981). An abnormal stomatal opening in the infected palms with impaired regulation leading to excessive water loss, irrespective of time of the day or season or growing conditions was reported (Rajagopal, Patil and Sumathykutty amma, 1986). For instance, the stomatal resistance at 14.00 h was only 5.5 sec. $cm^{-1}$ in the leaves of RWD palms, as against 14.9 sec. $cm^{-1}$ in those of healthy palms.

**Stomatal regulation**

Milburn and Zimmermann (1977) reported the controlling influence of stomata on the water balance of coconut palms. According to them the water balance of palms is more protected in the 'dry' than in the 'wet' season. This is supported by data on healthy palms, which exhibited relatively high stomatal resistance in response to dry weather (Rajagopal et al, 1986). However, the infection affected the stomatal regulation in both the seasons, though with greater magnitude in the dry (7.95 sec. $cm^{-1}$) than in the wet season (1.47 sec. $cm^{-1}$), thus indicating that the diseased palms could be distinguished from healthy palms better during the dry than during wet season. The differences in the relative humidity between the two seasons also influenced the stomatal regulation. It could be inferred that healthy palms develop a protective mechanism, in response to the prevailing dry conditions, to regulate their water balance through an effective stomatal system, while this protective mechanism appeared to have been greatly impaired in RWD palms. There existed a relationship between the disease index and transpiration rate in that with the advancement of the disease there was greater disturbance in stomatal regulation resulting ultimately in excessive transpiration. Palms in the early stage of the disease with an index of 20% had the transpiration rate in the range of 4.20 to 4.35 $\mu g\ cm^{-2}\ sec^{-1}$, while those in the advanced stage with the index above

50% transpired at the rate of 9.15 to 10.50 $\mu g. cm^{-2} sec^{-1}$. The involvement of metabolites similar to fusicoccin, a phytotoxic terpenoid glucoside produced by the fungus *Fusicoccum amygdali* with the characteristic feature of inducing stomatal opening (Chain, Mantle and Milborrow, 1971; Heichel and Turner, 1972), in the tissue of RWD palms was surmised on the basis of relatively high content of $K^+$ in the guard cells of leaves with flaccidity symptom, as revealed by the histological staining technique for locating $K^+$ under the fluorescent microscope (Rajagopal and Govindankutty, unpublished). Based on the response of two cultivars of almond (resistant and susceptible to the fungus) to fusicoccin treatment Turner and Graniti (1976) suggested that the toxin moves to the leaves and induces $K^+$ accumulation and turgor changes that open the stomata. A similar mechanism might be operating in RWD palms, although the nature of toxins is not known yet. In the case of LYD of coconut, stomatal closure was reported (Eskafi et al, 1986), — a characteristic feature of all other yellows disease in different plant species (Matteoni and Sinclair, 1983).

**Leaf water potential**

As a consequence of impaired stomatal regulation the leaf water potential components of infected palms were affected. Diseased palms had consistently lower leaf water potential than the healthy palms at any given time (Rajagopal, Sumathykutty amma and Patil, 1987). For instance, the pre-dawn value for leaf water potential was −0.41 $MP_a$ in diseased leaves, as against only −0.23 $MP_a$ in healthy ones. The nature of symptoms on the leaflets of different whorls of leaves *i.e.*, of different physiological ages, reflected the changes in leaf water potential. The unopened leaf (spindle leaf) of diseased palms, with white to dull cream colour, thin and papery structure and brown spots, had a leaf water potential of −0.72 $MP_a$, as compared to −0.37 $MP_a$ in green, thick and stiff leaf in healthy palms. Similarly, middle whorl leaves with the flaccidity, yellowing and necrosis symptoms recorded lower water potential (−1.28 $MP_a$), while the normal erect green leaves of healthy palms had high leaf water potential (−0.79 $MP_a$). From this it is evident that although the most perceptible symptom of the disease *viz.*, flaccidity occurred on middle whorl leaves, changes in leaf water potential had already begun in the spindle leaf, thereby indicating that changes in leaf water potential preceded foliar symptom expression. The leaf turgor potential was 0.34 $MP_a$ in the turgid leaves of healthy palms, as against only 0.04 $MP_a$ in the flaccid leaves of RWD palms.

**Relationship between water balance and the flaccidity symptom**

Changes in the overall water relations of diseased palms reflected on the development of the flaccidity symptom. Table I clearly indicates the distinct features of RWD palms as compared to healthy palms. The appearance of turgid leaves without any symptoms in the healthy palms could be attributed to the good root system and well-regulated stomata resulting in balanced water

Table I. *Water relations of coconut palms*

| Parameters | Healthy | Diseased |
|---|---|---|
| Root system | Good | Decayed |
| Water uptake | Optimum | Less |
| Water transport | Effective | Hindered |
| Stomatal regulation | Normal | Abnormal |
| Transpiration rate | Optimum | Excessive |
| Leaf water potential | High | Low |
| Leaf condition | Turgid | Flaccid |

economy of palms. In palms affected by RWD, excessive transpirational water loss through abnormal stomatal opening is not replenished adequately, since the uptake of water by roots is significantly affected and the water transport hindered. This would mean that the imbalance in the water economy of palm caused by deranged root system and impaired stomatal regulation, the two important facets of water transport in plants, ultimately culminated in an irreversible flaccidity symptom of leaves through changes in leaf water potential components.

That the appearance of distinct flaccidity on the middle whorl leaves may not have been a consequence of any sudden event, but rather the result of progressive changes beginning at the spindle leaf stage had been clearly shown by the values for leaf water potential between the leaves of different maturity. During the period between the spindle leaf stage and the first leaf stage, the leaf water potential of RWD palms decreased from $-0.72$ $MP_a$ to $-1.24$ $MP_a$. The initial change associated with flaccidity, namely flattening and bending of the leaf tip could be seen on the first whorl of leaves, although it might have been initiated much earlier. Once it had started, the later development of symptoms appeared to depend more on histological alterations in the tissues rather than on any significant change in leaf water potential. This is supported by little differences in leaf water potential between the leaves of middle and outer whorls. According to Govindankutty (1981) the accelerated transverse division and curtailed longitudinal division of the upper epidermal cells might explain the typical bending of leaflets of RWD palms. These changes might have been initiated by the tissue water potential differences at the spindle leaf and first-leaf stages. Thus, the overall disturbances in the water relations seem to definitely precede the manifestation of flaccidity symptom. The uniqueness of RWD appear to lie in the impairment caused to both aspects of water transport *i.e.*, absorption and transpiration, leading to internal water stress (whatever amount of water is absorbed by roots is also lost rapidly due to stomatal disfunction) the degree of which depend on the intensity of the disease.

**Relationship between the mycoplasma–like organisms, stomatal regulation and leaf water potential**

Abnormal stomatal opening is characteristic of certain fungal diseases (Table II),

Table II. *Occurrence of abnormal stomatal opening in different plant species with varying symptoms caused by different organisms*

| Causal organism | Host | Symptom |
|---|---|---|
| Virus | Tomato | Mosaic, Spot wilt |
| *Phytophthora infestans* | Potato | Blight |
| *Uromyces phaseoli* | Bean | Rust |
| *Rhyncosporium secalis* | Barley | Leaf blotch |
| *Microsphaera alphitoides* | Oak | Mildew |
| *Fusicoccum amygdali* | Peach, almond | Canker |
| *Mycoplasma*-like organisms | Coconut | Wilt |

h reflect on high rate of transpiration. , RWD of coconut is the only MLO dis- (Solomon et al., 1983) associated with e opening of stomata (Rajagopal et al., 6). The other MLO-caused diseases have characteristic yellows symptom, including LYD of coconut, with stomatal closure a distinctive feature (Matteoni and Sin- ir, 1983). It is presumed that MLOs pro- e metabolites that influence the stomatal ulation, either their closure or opening ble III). Toxins have been implicated in lows disease of periwinkle caused by *Spi- plasma citri* (Daniels, 1979). As discussed rlier, some unidentified metabolites akin to sicoccin might be involved in causing the matal opening through the accumulation $K^+$ in the guard cells of RWD palms. he xylem pressure potential of LYD affected oconut palms was higher than that of ealthy palms and this was attributed to early tomatal closure (Mc Donough and Zimmer- nann, 1979). Similarly in American elms howing yellows symptoms caused by MLOs, he stomatal resistance and xylem pressure potential were higher than in healthy plants ndicating the influence of the organism on tomatal closure (Matteoni and Sinclair, 1983). In contrast, Mc Donough and Zimmermann (1979) found reduced xylem potential in the Malayan Dwarf variety of coconut with symptoms of wilt (the causal organism had not been mentioned) and attributed the same to the excessive water loss, a characteristic feature associated with the wilt disease. This is in agreement with the RWD palms which showed higher transpiration rate with lower water potential than the healthy palms (Rajagopal et al., 1987). Thus, there seems to exist an intricate relationship between MLOs and water relations of plants irrespective of the nature of symptoms. Eskafi et al., (1986) also suggested that the stomatal closure in LY disease of coconut palms and phloem necrosis in elm and periwinkle and of ash witche's broom in ash and periwinkle (Matteoni and Sinclair, 1983), indicate a possible relationship in the mode of pathogenicity of these MLO-associated diseases.

### Diagnosis of the disease

The fact that the manifestation of visual foliar symptoms of the disease is preceded by the changes in water relations of palms

Table III. *Stomatal regulation in diseases caused by MLOs in different plant species*

| Plant species | Disease | Stomatal condition |
|---|---|---|
| *Catharanthus roseus* | Elm yellows (pholem necrosis) | Closure |
| *Catharanthus roseus* | Ash yellows (witches broom) | -do- |
| *Fraxinus americana* | Ash yellows | -do- |
| *Ulmus americana* | Elm yellows | -do- |
| *Ulmus rubra* | Elm yellows | -do- |
| *Prumus virginiana* | X - disease | -do- |
| *Zea mays* | Corn stunt | -do- |
| *Cocos nucifera* | Lethal yellowing | -do- |
| *Cocos nucifera* | Root (wilt) | Open |

indicates the availability of the potential tool for the early diagnosis of RWD palms. Based on the relationship between the severity of visual symptoms and stomatal resistance there existed the possibility of measuring the leaf diffusive resistance for the pre-visual detection of yellows disease in periwinkle and white ash (Matteoni and Sinclair, 1981) and RW disease in coconut (Rajagopal et al., 1988). In fact, a comparative study was made between the serological test using the cross absorption technique and stomatal resistance determinations in coconut palms with incipient infection. The palm devoid of visual symptoms, had serologically positive reaction and low stomatal resistance (thus considered as disease suspects) and were monitored for symptom expression. It was found that the two tests could detect disease affected palms six to 20 months earlier than the actual manifestation of flaccidity symptom. Basham and Eskafi (1980) also employed the stomatal resistance determinations for diagnosing the LY affected coconut palms. Even the wilt disease in apricot, caused by the fungus *Cytospora cincta,* could be detected three months earlier using the symptomatic changes in stomatal resistance (Korner, 1981). That the leaf water potential measurements could also serve for the early diagnosis of palms affected by MLOs was shown both in YLD (Basham and Eskafi, 1980) and RWD (Rajagopal and Sumathykutty amma, 1989). Thus, it is clear that the two parameters of plant water relations *viz.*, stomatal resistance and leaf water potential, could compliment each other in detecting the latent infection of plants prior to symptom expression.

**Comparison between the lethal yellowing disease and root (wilt) disease**

These two well known diseases of coconut palms show certain similarities, but in some features they exhibit distinct differences (Table IV). Although caused by MLOs, LY affected palms differ from the RWD ones not only in the primary foliar symptoms but also in the water relations. The water transport is affected to different degrees in both the diseases, but it is the stomatal regulation which seems to play a key role in the ultimate expression of foliar symptoms. As the stomatal closure was greatly impaired there was excessive transpirational loss of water resulting in wilt (flaccidity) symptom in RWD palms, whereas rapid closure of stomata due to infection led to a different type of symptom expression namely yellowing in YLD palms. Interestingly, although both the water absorption and transpiration rate are affected, the root (wilt) has remained as a debilitating disease, in contrast to the lethal nature of LY disease where transport alone was affected. This shows that the metabolic status of these palms differed in some respects. The possible cause for the differential response of the same species of coconut, growing in different geographical regions, could either be due to the variation in the strains of MLOs or to the nature of metabolites produced in the tissues of the infected plants. It may be mentioned that the insects involved in the transmission of MLOs are also different for the two diseases.

## CONCLUSIONS

Root (wilt) disease of coconut caused by MLOs, has the typical flaccidity symptom of leaves under all conditions of cultivation. The fact that the 'wilt' appearance of leaves occur even in flooded areas excludes the possibility of drought-induced effect. That the stomata remain abnormally open in the infected palms indicates clearly that the 'wilt' symptom on the leaves is not drought-induced (which in fact facilitate stomatal closure) but only disease-induced through the internal water stress. The relationship between the changes in the stomatal regulation and symptom development could be

Table IV. *Comparison between the Lethal Yellowing Disease (LYD) and Root (wilt) Disease (RWD) of coconut palms*

| Description | LYD | RWD |
|---|---|---|
| Earliest record/location | 1904, Cayman Islands | 1874–1884, Erattupetta, Kottayam, Kerala |
| Symptoms, primary associated | Yellowing, Inflorescence necrosis, immature fruit drop | Flaccidity, yellowing, necrosis |
| Nature of disease | Lethal | Debilitating |
| Causal organism | Mycoplasma-like organisms | Mycoplasma-like organisms |
| Insect associated with transmission | Plant hopper (*Myndus crudus*) | Lace bug (*Stephanitis typica*) |
| Transmission through dodder | — | *Cassytha filiformis* |
| Water absorption/transport | Decreased | Decreased |
| Stomata | Closed | Abnormal opening |
| Transpiration rate | Normal | Excessive |
| Leaf water potential | Low | Low |
| Early diagnosis | Stomatal resistance and water potential | Serological reaction, stomatal resistance, water potential |
| Flow of phloem sap | Nil or very low | Low |
| Arginine content | High in leaves | High in leaves and phloem sap |
| Antibiotic therapy | Tetracycline | Tetracycline |

established in that the former precedes the latter in time and space. There is ample evidence to show the diagnostic value, with immense potential for large scale field studies, of water relation determinations to detect latent infection of the disease several months before the manifestation of visual symptoms. The comparisons between the 'wilt' diseases caused by different organisms, between the MLOs and different plant species and finally between the LYD and RWD coconut palms brought out an interesting array of knowledge, which could lead to identifying exciting areas of research to unravel the disease complex in plant species.

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