

CURRENT STUDIES ON ETIOLOGY AND MANAGEMENT OF STEM BLEEDING DISEASE OF COCONUT

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INTRODUCTION

Stem bleeding disease of coconut has been reported from all countries where coconut is grown. The disease was first reported (Petch, 1906) from Sri Lanka. Later the disease was found to occur in many coconut growing countries like India, Indonesia, Malaysia, the Philippines, Fiji, Papua New Guinea etc. In India, the disease is seen in Kerala, Karnataka, Tamil Nadu, Andhra Pradesh, Goa, Orissa, Maharashtra, Andamans, West Bengal etc. Restricted surveys conducted in Kannur and Kasaragod districts of Kerala showed that the average disease incidence is 8.8% (Anon, 1990).

Symptoms:

The disease is characterised by the presence of dark brown patches along the growth cracks, mostly at the base of the trunk. A dark reddish brown liquid exudes from these cracks. Adjacent patches may coalesce together to form larger patches. The gummosis later dries up. On chiselling the affected portion of the bark, the lesions can be found to penetrate up to about 2-3 cm in depth. Histopathological studies revealed disorganization of the parenchymatous tissues of the cortex in the affected stem. In young palms, the lesions can be deep seated and the tissues and bark decay leaving only internal fibrous tissues.

Under cool conditions, the degeneration of affected tissues, especially in young palms, is very fast and the palms succumb to the disease in 2-3 years. As the disease progresses, the patches also traverse higher and higher up on the trunk. The crown starts expressing the symptoms by that time. The lower leaves turn yellow gradually and the yellowing spreads upwards. In advanced stages, the size of leaves is reduced with a consequent reduction in size of the crown. Shedding of buttons and immature nuts are also observed. Tapering of the trunk is noticed in severely affected palms. The leaves dry gradually, and fall away leaving a crownless trunk in the final stage. In the initial stages the roots are normal, but in advanced stages of disease, the roots also show abnormal decay.

Etiology:

Petch (1906), observed that *Thielaviopsis paradoxa* is a weak wound pathogen associated with the disease. This fungus remained as a suspected pathogen till recently. Nambiar *et. al.* (1986) confirmed its etiologic role from artificial inoculations carried out at Kasaragod (Kerala State) and Appangala & Vittal (Karnataka State). Characteristic rusty brown discolouration of the bark was noticed around the inoculated sites within 2-8 weeks depending on the location and period

of inoculation. Copious gushing of brown liquid was noticed on palms at Appangala, where the atmospheric temperature was the lowest and humidity the highest among the three locations. The gummy exudates contained conidia of *T. paradoxa*. Recently the authors have observed *Ceratocystis* stage of the fungus from affected palms in Karnataka (Anon, 1992).

The lesion depth/size was maximum in palms inoculated during or after South West monsoon (July to November), and the lesion size was comparatively less when inoculation was done during April-May. Palms in the age group of 10-12 years were found to be more susceptible and showed more internal decay compared to 45-60 year-old palms. High humidity and moderate temperature prevalent during July to November favoured the disease development while reduced moisture and high temperature during April-May adversely affected lesion development (Nambiar, *et. al.*, 1989).

Many workers failed to isolate the pathogen consistently from infected tissues. So a method was developed at this Institute for isolating *T. paradoxa* from infected tissues (Anil Kumar and Nambiar, 1991). This simple and highly reproducible method consists of inoculation of old frond pieces with the pathogen using bore hole method, incubation in polythene bags at 30°C

for 10 days and isolation of the fungus from the margin of lesions on sugarcane juice agar. Similarly, a baiting technique was also developed to recover the fungus from the soil and to estimate the population in the soil (Anon, 1990).

Different isolates of the pathogen were collected from various localities. Among isolates of *T. paradoxa* wide variability was found to exist with regard to characters like colour, nature of colony, pattern of growth on various media, production of conidia and chlamydospores, reaction towards antagonistic fungi, etc. (Gowda and Nambiar, 1992). The isolates also expressed wide variations with regard to their requirement of temperature, pH, carbon, nitrogen sources, etc. (Nishita Naik, 1990). Maximum survival of the chlamydospore was seen in red loamy soils and the least in sandy soils (Usman, 1988). In the case of neem cake-amended soil, the survival of chlamydospores was minimum. All isolates of *T. paradoxa* were completely inhibited *in vitro* by lower concentrations of Bavistin (carbendazim) and calixin (Tridemorph) (Nishitha Naik, 1990).

Predisposing factors:

The fungus enters the stem through growth cracks or wounds. The growth cracks may develop after sudden rains following prolonged dry period. Sudden heavy manuring, trash burning at the base of the palm or injury made during tractor ploughing etc. cause damage to the palms, paving way for infection. Imbalanced fertiliser application has also been reported to lead to disturbed physiological condition of the palms predisposing the palms to infection. Ill drainage, hard lateritic pan etc. are also found to be other

predisposing factors. Soil reaction and electrical conductivity were found to have no influence on the incidence of the disease. In Indonesia, chlorine deficiency was reported to play an important role in the manifestation of disease symptoms. Chlorine deficiency does not seem to be a contributing factor in India, especially in Kerala where on the banks of back waters (lakes and rivers) incidence of the disease is noticed. Excessive salinity with high sodium was reported to be associated with the disease (Nagarajan, 1985).

Recently reports from Indonesia show that *T. paradoxa* is associated with the disease in that country also (Sitepu and Darwis, 1989). In addition to the damage caused by fungal infection, the affected bark is often infested by scoletid beetles which make small bore holes in the stem.

Disease management:

The earliest recommendation for the control of the disease included removal of affected bark tissues and applying hot coal tar on the chiselled surface. This is being practised even now, though with limited success. Hot coal tar should never be applied on the stem of young palms below 10-15 years since such treatment was found to cause phytotoxicity resulting in oozing of gum from the coal tar applied surface. Systemic fungicides like Calixin, Bavistin, Aureofungin sol etc. and organic amendments like neem cake were also tried by some research workers later. The fungus was completely inhibited *in vitro* by lower concentrations of Bavistin (carbendazim) and calixin (Tridemorph). However, Dithane M-45 (Mancozeb), Vitavax (Carboxin) and Aureofungin sol inhibited the growth only at very high concentra-

tions (Anil Kumar and Nambiar, 1990). The results of field trials conducted at CPCRI, Kasaragod and Kerala Agrl. University, Pilicode using systemic fungicides like Bavistin, Calixin, Aureofungin sol and vitavax showed that Calixin was more efficient in the disease management (Radhakrishnan, 1990, Anon, 1992). The treated palms had not only lower disease index, but also increased yield. The persistence of the fungicide in the treated palms was also studied. Carbendazim, the active ingredient of Bavistin was detected up to 2 M height in stem up to 120 days when palms were root fed @ 10 kg Bavistin/palm and up to one metre for 20 days when treated with 0.5 g Bavistin/palm. No fungicide was detected in tender nut samples after 23 days of treatment (Anil Kumar *et al.*, 1992). Tridemorph, the active ingredient of Calixin was detected from both feeding side as well as opposite side of the coconut trunk upto 3 metres height in the trunks upto 45 days of root feeding with Calixin @ 2 - 10 ml/palm. No residues of tridemorph were detected in nut water of the treated palms (Ramanujam *et al.*, 1991).

Another area of current research in the field of disease management is the biological control. Stem bleeding lesions especially old ones, harboured many mycoflora which are found to be antagonistic to the pathogen. Important among these are *Trichoderma harzianum* and *T. viride*. Soil samples collected from neem cake amended soils also yielded higher percentage of *T. harzianum*, along with other antagonistic fungi. Maximum inhibition of *T. paradoxa* in *in vitro* studies was effected by *T. viride* (90%) followed by *T. harzianum* (86.6) (Gowda and Nambiar, 1991). *In vivo* interaction of these antagonists with *T. paradoxa* was studied

using detached leaf petioles and almost similar trends of results were obtained (Usman and Nambiar, 1992). *Trichoderma* was found to cause lysis of the hyphae of *T. paradoxa* either by physical contact or through diffusible compounds. Studies on the effect of fungicides on the antagonists showed that carbendazim was fungicidal to *Trichoderma* spp. even at 1000 ppm, while Tridemorph was only fungistatic even at 5000 ppm concentration. (Ramanujam *et al.*, 1991).

Studies on biological control have received great emphasis now-a-days. Isolation of more fungal and bacterial antagonists, especially in the presence of different amendments, methods of mass multiplication of the efficient candidates, standardisation of methods of application of these organisms to soil or to the affected stem are some of the areas which are receiving our attention. These together with judicious application of systemic chemicals along with organic matter supplemented with neem cake, besides a better management of soil moisture regime, will be useful in managing the disease to an economic threshold level. Since insect pests are also associated with the disease, especially in the middle and late stages, they are also to be checked through application of proper insecticides. Screening of cultivars and hybrids for resistance/tolerance to the disease is yet another area of investigation. Studies are in progress in this line also. The above investigations will help in better understanding of the problem and arriving at a satisfactory answer towards better management of the disease.

REFERENCES

1. Anil Kumar and Nambiar, K.K.N.

1990. Effect of certain physical and chemical factors on the germination of endoconidia and chlamydo spores of *Thielaviopsis paradoxa*. *Indian Phytopath.* 43: 460-462.
2. Anil Kumar and Nambiar, K.K.N. 1991. An improved method for isolation of *Thielaviopsis paradoxa* from stem bleeding affected coconut palm. *CORD* 7(1): 49-53.
3. Anil Kumar, Nambiar, K.K.N. and Voleti, S.R. 1992. Uptake, translocation and persistence of carbendazim in coconut in relation to control of stem bleeding disease. *CORD* 8(2): 40-47.
4. Anonymous 1990. Annual Report, 1989-1990, Central Plantation Crops Research Institute, Kerala, India, pp. 262.
5. Anonymous 1992. Annual Report 1990-1991, Central Plantation Crops Research Institute, Kerala, India, pp. 198.
6. Gowda, P.V. and Nambiar, K.K.N. 1991. *In vitro* interaction of antagonistic fungi with *Thielaviopsis paradoxa*, the pathogen of coconut stem bleeding disease. *J. Plant. Crops* 18(Suppl.): 233-238.
7. Gowda, P.V. and Nambiar, K.K.N. 1992. Variation in cultural characters among isolates of *Thielaviopsis paradoxa*, causal agent of stem bleeding disease of coconut. *J. Plant. Crops* 20 (Suppl.): 69-72.
8. Nagarajan, M. 1985. Influence of saline environment on the incidence of stem bleeding in coconut (*Cocos nucifera* L.) *Sci. and Cult.* 57: 349-351.
9. Nambiar, K.K.N., Joshi, Y., Venugopal, M.N. and Mohanan, R.C. 1986. Stem bleeding disease of coconut. Reproduction of symptoms by inoculation with *Thielaviopsis paradoxa* *J. Plant. Crops* 14: 130-133.
10. Nambiar, K.K.N., Anil Kumar, Kalpana Sastry, R. and Joshi, Y. 1989. Seasonal effect on infection by coconut stem bleeding pathogen, *Thielaviopsis paradoxa*. *Curr. Sci.* 58: 34-35.
11. Nishitha Naik, V. 1990. Variability and characteristics of different isolates of *Thielaviopsis paradoxa*, a

pathogen of coconut stem bleeding disease. M. Phil Dissertation, Mangalore Univ. pp. 94.

12. Petch, T. 1906. Diseases of the coconut palm. *Trop. Agriculturist* 27: 489-491.
13. Radhakrishnan, T.C. 1990. Control of stem bleeding disease of coconut. *Indian Coconut J.* 20(9): 13-14.
14. Ramanujam, B., Nambiar, K.K.N. and Anil Kumar, 1991. Chemical control of stem bleeding disease of coconut. Paper presented at the second Intern. Symp. on coconut Res. and Development. 26-29 Nov. 1991, CPCRI, Kasaragod.
15. Sitepu, D and Darwis, S.N. 1989. Current Knowledge, management techniques and areas of future work for major coconut diseases in Indonesia. pp. 201-219. In: *Coconut Production and Productivity: Proc. 26th COCOTECH Meeting* d. Sumith de Silva, APCC, pp. 377.
16. Usman, N.M. 1988. Studies on stem bleeding disease of Coconut. M.Phil. Dissertation, Mangalore Univ., pp. 74.
17. Usman, N.M. and Nambiar, K.K.N. 1992. Effect of some antagonists on *Thielaviopsis paradoxa* (de Seynes) Hohnel, the pathogen of stem bleeding disease of coconut. *J. Plant Crops* 20(1): 68-70.

