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STUDIES ON BUD ROT DISEASE OF COCONUT

by

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Introduction.

Bud rot of coconut is known to kill young palms in India during the monsoons. A better understanding of the correlation of the disease with weather factors and biology of the pathogen became necessary because of the seasonal occurrence of the disease and the reported influence of the environmental factors on Phytophthora palmivora which was attributed to be the causal agent (Butler, 2). Of late, unsuccessful efforts in many instances to isolate P. palmivora from coconut and the appearance of disease syndrome at the detectable stage as a wet rot cast doubt on its role as the causal agent (11). Hence the possibility of pathogen(s) other than P. palmivora particularly bacteria having a positive role in the incidence of the disease was also examined. Other hosts in the vicinity of coconuts may serve as source of inoculum, which pointed to the need to screen such hosts also. These aspects were investigated recently.

Survey on the distribution and intensity of the disease in different coconut growing states in India.

The coconut growing states lie chiefly along the west and east coasts of the peninsula, between 8° N and 27° N. Starting from Sourashtra (Gujarat State) in the north-west and Assam in the north-east the western and eastern coconut tracts converge to meet at Kanyakumari (Tamil Nadu) at the southern end of the peninsula. Survey on the distribution and intensity of the disease was conducted in Kerala, Karnataka, Maharashtra, Gujarat and the Union Territories of Goa and Mahe on the west coast, and Tamil Nadu, Andhra Pradesh, Orissa, West Bengal and Assam on the east. Percentage incidence of the disease was estimated on the basis of infection recorded in groups of palms in half-an-acre plots.

In general the disease was noticed in palms growing in various types of soil namely sandy to sandy loam, reclaimed claeyey, red loam and gravelly laterite, distributed from sea-shore to hill top on river banks and backwater areas. Intensity of incidence varied from 0.1 to 6.5 per cent in Kerala and 1.45 to 3.6 per cent in Tamil Nadu. Occasionally heavy incidence of the disease to the extent of 35 to 40 per cent occurred in Kerala. The disease appeared rampantly in coastal districts of Karnataka. In Andhra Pradesh it was scattered. Young palms in Maharashtra, Goa and Mahe had infection. Few palms have been infected in Gujarat, Orissa, West Bengal and Assam.

Occurrence of the diseaseת was confined to the South-West and North-East monsoon periods. Generally palms aged between 5 and 15 years were more susceptible to the disease.

Studies on the meteorological and microclimatic factors in relation to disease.

Data on rainfall, temperature and humidity were recorded at the Central Plantation Crops Research Institute observatories at Kayangulam and Kasaragod in Kerala. Through a series of experiments temperature and relative humidity were recorded at leaf axil of palms in different age groups, using Assman Psychrometer. In the first, data were collected between 0900 h and 1000 h at Kayangulam from palms in five age groups ranging from 3 to 40 years. Based on statistical soundness, a Latin square method of randomisation was adopted in the second. Data were collected between 0930 h and 1630 h at Kayangulam and Kasaragod. Temperature and relative humidity were recorded on five occasions a day at leaf axil levels of groups of 10 palms in the 5 age groups. Since then the method was modified to record the data between 0730 h and 0930 h as it was found that the late forenoon and afternoon readings had no bearing on disease development. Besides Kayangulam and Kasaragod on the west-coast, Pattukottai (Tamil Nadu) on the east-coast was also selected as a centre for recording the data.

Pre-monsoon data hardly indicated any difference between the microclimate of palms in different age groups. However with the onset of monsoon, palms in the age groups 5 to 10 and 11 to 15 years recorded lower temperature and higher relative humidity as compared to those still younger and older. Positive correlation between rainfall and disease incidence was observed at Kayangulam and Kasaragod.

Reduction in rainfall was reflected in the microclimate as well as development of disease. A critical perusal of the data revealed that the number of days which recorded high humidity (97-100%) and 21° c temperature termed as "favourable days" for infection, was

directly correlated with disease incidence. During two successive monsoons at Kayangulam 31 and 13 palms in different age groups developed infection when the number of "favourable days" ranged from 13 to 20 and 3 to 5 respectively. Incidence of the disease showed an upward trend for about four weeks during the monsoon period. Subsequently it decreased when there was fall in relative humidity.

The data on microclimate for a 10 week period from June to September revealed that 3 to 20-year old palms have higher humidity, the maximum being in the 11 to 15-year old. This holds good for open air data at the crown level of palms.

Analysis of microclimatic data with regard to temperature at Kayangulam and Kasaragod revealed significant difference between palms in different age groups. On an average palms aged 16 years and above recorded significantly higher temperature than those of 3 to 15 years. At Kayangulam the average temperature in the leaf axils and open air at crown level of palms in different age groups also showed significant difference. Both the values were found to be minimum in those aged 11 to 15 years.

Neither the temperature nor the relative humidity recorded at Pattukottai touched the "favourable" level. However, they fell in line with the observations recorded on the west-coast in that the young palms had lower temperature and higher humidity.

Studies on the pathogen.

Attempts to isolate P. palmivora from several samples of bud rot affected coconut crowns having wet rot lesions, were unsuccessful. But samples collected deeper down from the crown free of wet rot symptoms yielded P. palmivora. Seven naturally infected crowns on incubation at 22°c produced fluffy growth of

P. palmivora from the basal cut end which was free of wet rot, whereas the top portions of the crown on isolation yielded Pseudomonas sp, Xanthomonas sp and Erwinia sp.

One to two-year old West Coast Tall cocoonut seedlings on inoculation in the leaf axils with sporangial suspension of P. palmivora isolated from diseased coconut, and incubation at controlled conditions of 22°c temperature and 97 to 100 per cent relative humidity, developed bud rot. When succumbed subsequently these yielded P. palmivora from dry rot lesions. Similarly 5-year old palms in the field also developed bud rot when inoculated in young axils, during South-West monsoon. On dissection of the crown it was observed that rotting had spread farther down from the site of inoculation, which was also true of the seedlings that succumbed under the controlled conditions.

Under the controlled conditions the coconut isolate of P. palmivora proved fatal to different hybrids and varieties of coconut namely Tall x Chowghat Dwarf, Tall x Gangabondam, Chowghat Dwarf x Tall, Chowghat Dwarf x Gangabondam, WCT and Malayan Dwarf. Treated similarly the same isolate infected one-year old oil palm and arecanut seedlings also. While oil palm succumbed to the infection arecanut sustained only brownish lesions on the young petiole bases.

A span of six days was required for P. palmivora to complete an infection cycle on tender coconut petiole. Establishment of primary infection, formation of secondary sporangia and spread of rotting were involved in this cycle which culminated in formation of oospores four weeks later.

Oospores also developed in culture medium which got contaminated with Thielaviopsis sp, a situation similar to that reported by Brasier (1).

Attempts to artificially infect coconut seedlings and uninjured tender petiole of cocoonut with the isolates Pseudomonas sp, Xanthomonas sp and Erwini a sp failed. However the same organisms were able to increase severity of rotting through primary lesions in the tender petiole produced by P. palmivora. Infectivity of bacteria on artificially wounded tissues was not tested.

P. palmivora was also isolated from diseased arecanut, oil palm, palmyra, rubber and cardamom, besides coconut. The first three belong to the "caocao group" while the rest are atypical. On cross-inoculation these isolates, except that from cardamom which was not tested, infected one-year old seedlings of coconut, oil palm and arecanut. The first two developed bud rot symptoms while the last developed only brownish lesions in the tender tissues at the collar.

Discussion.

Bud rot disease may be classified along with the rain-favoured diseases as it is season-bound and occurs during the monsoon seasons in India. Butler (3), Keitt and Jones (8) and Menon and Pandalai (10) have reported that bud rot disease of coconut i s favoured by rain and high atmospheric humidity. The disease is observed to be mainly confined to young pa lms. Our observations made in the different States in India confirm this. The importance of climatic conditions favourable to the pathogen and the susceptible age of the palm in the occurrence of the disease are evident from the non-prevalence of the disease in the adult plantations of Goa and Maharashtra (where young palms have been infected) and young plantations in Sourashtra. Weather conditions congenial for the incidence of the disease occur in the former while in Sourashtra they do not. Availability of young plantations

together with congenial climatic factors would aggravate the incidence of bud rot is evident in that the rate of infection goes as high as 35 to 40 per cent in Kerala.

Occurrence of Phytophthora diseases is known to be highly influenced by weather factors as has been experienced with P. infestans on potato. Our observations that the microclimate of palms in different age groups is directly related to weather factors particularly rainfall, conforms to this. Number of "favourable days" i.e. the duration having 97 to 100 per cent relative humidity and 21°c temperature, appears to influence the development of the disease. Observations of Crosier (5), Kyron (9) and Hicks (7) are in line with this. Humidity and temperature as shown by Crosier, are the two cardinal factors that determine sporulation, germination and infection of P. infestans. Laboratory trials showed that P. palmivora needs nearly a week to complete one infection cycle. A series of such cycles may be required to enable the pathogen to penetrate the thickly set whorls of leaf bases and reach the primordial region for the onset of the disease. This is correlated with the span of "favourable days". Observations that a minimum period of five weeks after the inoculation was required for the occurrence of the disease both under the controlled conditions and in the field during South-West monsoon, agrees with the above.

Identity of the casual agent of bud rot disease of coconut was accepted as P. palmivora (Butler, 4). Successful isolation of P. palmivora from tissues free of wet rot suggests that the earlier failures were due to the selection of wrong samples. It is likely that infection of P. palmivora causes only a dry rot in coconut and the wet rot observed therein at later stages

is due to secondary invaders like species of bacteria. Reinking (12) and 13) reported from Philippines that P. faberi (Maubl.) is the primary cause of bud rot of coconut with bacteria occurring as secondary infection in the injured and weakened tissues. Results of our infection trials also conform to this. The species of Pseudomonas, Xanthomonas and Erwinia failed to infect the WCT seedlings and the uninjured petiole thereby showing that they have no primary role in the disease development. Failure of parasitic or saprophytic bacteria to penetrate undamaged potato tissues (Dowson and Jones, 6) and effect of fungal parasitism in inciting pathogenic potential of endogenous organisms (Sturdy and Cole, 14) are pertinent. Killing of the bud of coconut perhaps takes place much in advance of the secondary wet rot usually found on the upper part of the affected crown. Successful isolation of P. palmivora from tissues affected by dry rot, its fluffy growth from the basal cut end of the affected crowns and lastly its pathogenicity on coconut seedlings both under the controlled conditions and in the open go to prove its primary role in the disease.

Susceptibility of arecanut, oil palm and hybrids and varieties of coconut to artificial infection by P. palmivora and its capability to form oospores in infected tissue which could serve as source of survival, point out to the vast pathogenic potential of the pathogen. Isolation of P. palmivora (Butl.) Butl. also from naturally infected arecanut, oil palm, palmyra, rubber and cardamom and its proved infectivity on cross-inoculation confirm this.

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Bliding. due to Erwinia in Jamaica.

Bud rot. is high - yellow