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WILT DISEASES OF BLACK PEPPER

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ABSTRACT

Among the major diseases affecting black pepper, foot rot (quick wilt) and pepper yellows (slow wilt) are important, inflicting heavy losses in plantations. Foot rot is caused by *Phytophthora palmivora* and is characterised by rotting of collar and/or root and leaf, die back of twigs and spike shedding. The disease incidence is generally high when relative humidity is high (91-99%) and temperature is low (19-23°C). The nature of spread supports the view that it is mainly through soil and water. Heavy rainfall and wind contribute to aerial spread leading to foliar infections. The pathogen has got a wide host range. Its mode of perennation, population build up during different seasons and primary infections are areas requiring investigations. Existence of difference in virulence in the pathogen cannot be ruled out. *Piper colubrinum, P. obliquum,* and *P. guineense* are reported to be resistant to the pathogen. No effective curative control measures have so far been evolved against the disease. The importance of evolving resistant lines and adoption of management practices in conjunction with chemical treatment is indicated in developing control schedules.

In the case of slow wilt, involvement of fungi like *Fusarium* sp., *Rhizoctonia* sp. and nematodes like *Radopholus similis* and *Meloidogyne incognita* is indicated. The importance of deficiencies of K and P and soil water stress in the incidence of disease also has been stressed. Effective measures have not been worked out so far. Extensive screening is to be taken up for locating resistance to this disease.

INTRODUCTION

Average yield of black pepper is significantly lower in India (236 kg/ha), its native land, than in other leading pepper producing countries like Indonesia (529 kg/ha) and Malaysia (4130 kg/ha) (Anonymous, 1977). Despite an increase in area under cultivation in India, the total production has remained stagnant at less than 30,000 tonnes annually. Though high cost of cultivation and poor agrotechniques are important factors limiting pepper productivity in India, the severe incidence of diseases and pests is the major cause for this.

Seventeen fungi have been recorded affecting black pepper in India, while Turner (1971*b*) reported 40 fungi from pepper in Sarawak. Among these, only a few have been identified as causative organisms of major diseases. Pathological investigations on pepper can be traced back to the early 1900's in India. Barber (1902, 1903, 1905) was the first to study the root disease of pepper. He took up this work following a representation to the government by planters in Wynad, Kerala. Later, Butler (1906, 1918) investigated the problem in more detail.

Based on symptomatology, the time taken for the death of a vine from the expression of

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initial visible symptoms, and the pathogen involved, two types of wilt diseases are recognised in India, quick wilt and slow wilt. These diseases are common in other countries also. Quick wilt is more generally referred to as 'foot rot' and slow wilt as 'yellows' or 'slow decline'. Various aspects of foot rot have been dealt with in previous reviews by Muller (1936) and Holliday and Mowat (1963).

Quick Wilt

The first authentic record of occurrence of quick wilt (foot rot) was from Indonesia (Muller, 1936) who identified the pathogen to be *Phytophthora palmivora* var. *piperis*. Though *Phytophthora* sp. was isolated from pepper in Mysore (India) as early as in 1920's (Venkata Rao, 1929), the investigators did not consider this fungus a pathogen. It was Samraj and Jose (1966) who first identified quick wilt and confirmed Muller's (1936) identification of the pathogen.

Foot rot of pepper was reported to cause severe losses in Indonesia (Muller, 1936) and Sarawak (Holliday and Mowat, 1963). It was also recorded from Puerto Rico (Gregory Almeyda, and Theis, 1960), Brazil (Holliday, 1965; Albuquerque, 1966; Alconero et al., 1972), Jamaica (Leather, 1967) and Thailand (Tsao and Tummakate, 1977).

The disease generally occurs during July-September, coinciding with the south-west monsoon, and takes a heavy toll of the plantations. Samraj and Jose (1966) recorded 20% loss of vines due to the disease in some plantations in Cannanore (India). A loss of similar magnitude was observed in Sarawak by Robertson (1953). Holliday and Mowat (1963) estimated the total loss at 7000 tonnes. We have recorded 25-30% loss of vines in some gardens in Cannanore and Calicut districts.

Symptoms

Leaf rot. It is observed in all parts of the vine, but is more severe at the lower regions. Water-soaked lesions with smooth or fimbriate margins appear in all parts of the leaf and advance rapidly. Enlarged lessions of 0.5 cm size and more, some times cover upto half the lamina. They are either unformly dark or appear in concentric zonations. Muller (1936) did not observe zonate leaf lesions in Indonesia, but Holliday and Mowat (1963) observed it in leaves in Sarawak. Turner (1969) observed that the different types of lesions described by Muller (1936) and Holliday and Mowat (1963) reflected differing conditions of incubation only. Under continuous humid conditions, fimbriate lesions develop, but when wet and dry conditions alternated concentric zonations were formed. Foliar infections result in defoliation to varying degrees. Immature leaves are more susceptible than mature ones and infection is more common on the lower surface of leaves (Turner, 1969*b*).

Spike infection culminating in its shedding is another common symptom. Usually, the distal end of the spike is affected and the lesions spread towards the stalk. Occasionally, a few berries only contract infection in a spike. It is commonly observed that aerial branches show discoloration and decay at some point. The leaves of affected twigs become pale yellow, droop, and fall off. The lateral branches of the vines break off at the nodes.

Collar rot (Foot rot). It appears on the vines at ground level and above upto about one meter height. Early infections of collar rot or root usually go undetected until foliar yellowing starts. The earliest symptom is the appearance of light yellow interveinal chlorosis in the upper leaves. Gradually, the whole foliage turns yellow. The infected region appears wet and discoloured and is at times slimy to touch. Before the cortical tissues disintegrate, the vascular cylinder turns dark and the intensity of cortical disintegration increases as the disease pro-

gresses. Vascular discoloration upto 0.5 m beyond the point of infection has been observed in many cases, but not consistently. The cortical region gradually rots and peels off easily. The rotting progresses into the soft medullary ray cells resulting in loosening of xylem strands. In advanced stages, the affected tissues emit a foul smell. Infection in the collar region progresses towards the underground stem, then to the root system, and causes their rotting. The spread of infection to the roots from the collar is, however, gradual.

Occasionally, one vertical half of the stem alone is involved in the infection, leaving the other half normal. In some bushes, a single vine dies leaving the adjacent vine on the same standard unaffected.

A notable feature is the absence of foot rot in young vines. This is believed to be due to disease escape rather than to juvenile resistance, since young vines have been seen to succumb to infection upon inoculation (Holliday and Mowat, 1963).

Root rot. Though in most cases root rot follows collar rot, root infections alone are also noticed in several gardens. The earliest visible symptom in such cases is foliar yellowing and interveinal chlorosis. The infection generally starts on the tender lateral roots and progresses towards mature roots, reaching finally the underground stem. The number of roots affected and the extent of rotting determine the speed with which the vines die. In the case of both collar rot and root rot, foliar yellowing and flaccidity, defoliation, breaking of tender stems at nodal region, and spike shedding are often noticed (Muller, 1936; Holliday and Mowat, 1963).

Holliday and Mowat (1963) reported that cultivation of pepper on mounds favoured root infection. However, both collar rot and root rot are seen in India though mound cultivation is not adopted. While collar infection is more common on slopy lands, root rot symptoms are noticed in the plains on level land.

Etiology

As already reviewed, Muller (1936) was the first to identify the pathogen of foot rot as *P. palmivora* var. *piperis*. Holliday and Mowat (1963) also placed the pepper pathogen inder *P. palmivora*. Based on morphological and cultural characters, Turner (1969*b*) placed *Phytophthora* isolates affecting both *P. nigrum* and *P. betle* under *P. palmivora*. In Brazil, however, Albuquerque (1961) have siolated *Fusarium solani* var. *piperi* consistently from dead tissues of pepper vines affected by foot rot. The fungus produced typical foot rot symptoms on inoculation.

The fungus grows rapidly and luxuriantly on oats agar at 25-28°C (Turner, 1969*b*). There was no growth at 35°C (also, Holliday and Mowat, 1963). Turner (1969*a*) obtained maximum sporulation at pH 6.0 with Sarawak isolates of the pathogen. There was no sporulation at pH 3.0. Brasier (1969*a*) obtained oospores by artificial inoculation of leaves by different isolates. Oospores were not formed at 30°C and their production was favoured by darkness and low temperature (20°C). While Holliday and Mowat (1963) found fusion organs in mixed culture, Turner (1962) reported their occurrence in cultures from single isolate. Brasier (1969*b*) suggested that oospores were likely to occur in nature, though rarely, and that their production would be confined to woody tissues or debris which are protected from light. Holliday and Mowat (1963) could not observe these either in naturally or artificially infected vines.

Considerable difficulty is experienced in isolating the pathogen. It is more difficult to isolate them from infected stems and roots than from leaf lesions. Isolation of *P. palmivora* from soil using baits like apple (Holliday and Mowat, 1963), castor seeds

(Narasimhan, 1964), and pepper leaf discs (Muller, 1936) does not always give consistent results. Whether this inconsistency is due to the differences in temperatures at which these isolation were attempted, or because more than one species or strians of *Phytophthora* is involved in the disease, is uncertain. Tsao and Tummakate (1977) found that the *Phytophthora* isolates from pepper in Thailand differed markedly from those previously described. Holliday and Mowat (1963) had earlier found that different isolates differed in their *in vitro* growth, sporangial production, and pathogenicity. Turner (1973*a*) has also reported variable virulence of the pathogen from Sarawak.

The fact that vascular browning is noticed at points beyond the site of infection, both in root and stem, suggests the possible involvement of a toxin in the production of the disease. Incidentally, Lee (1973) had reported production of toxin by *P. palmivora* in black pepper and found that the virulence of the isolates was related to their toxin production capacity. He used it as a marker to screen pepper varieties for foot rot resistance. In this connection, the observation of Keen et al. (1975) on the presence of mycolaminarans, B-1-3 glucans in cultures of *P. cinnamomi, P. palmivora,* and *P. megasperma* var. *sojae,* and its phytotoxic nature on soybean, cacao, tomato, etc. is pertinent. Nambiar et al. (1977) also found that cell-free culture filtrates of the fungus could induce vascular browning and flaccidity of leaves in cut shoots of pepper.

Spread

In hilly terrain, the vines in the valley get infected early and the infection spreads gradually, more downwards, supporting the view that spread is mainly through soil and water. In the plains, infection occurs in a sporadic manner and spreads to the adjacent vines. The spread is rapid in neglected plantations with infected vines around.

We studied the spread of the disease in an arecanut plantation having pepper as an intercrop and observed that within two years, about 250 vines died. Regular irrigation as well as cultivation in the gardens could have aided the quick spread of the pathogen. The rate of spread appears to be much more rapid in Sarawak, and it has been ascribed to the prevalence of continuous wet seasons coupled with application of large amounts of organic fertilizers and bare soil cultivation without any weed growth (Holliday and Mowat, 1963). They have proposed that the grass cover and weed growth in Indian pepper plantations may be impeding the spread of the pathogen.

Vines of all age groups are infected under field conditions. However, the incidence is less common in vines during the first three years after planting and maximum incidence is noticed after the first five years. Nambiar et al. (1976) has observed that 3-months old rooted cuttings are infected.

Muller (1936) and Holliday and Mowat (1963) have suggested that infection of leaves in the lower region of the bush might be due to rain splash only. Turner (1969 *b*) observed that number of leaves exhibiting leaf lesions was more at lower heights of the vines than at the top and felt that heavy rainfall and wind could contribute to aerial spread of the disease. Aerial transmission of zoospores of *P. palmivora* has been reported in coconut (Briton-Jones, 1970), rubber (Wastie, 1967), and cacao (Thorold, 1952).

Many agents aid the dispersal of the pathogen zoospores (Turner, 1964, 1967). *Phytophthora* has been recovered from the faeces of the giant African snail, *Achatina fulica,* which often feeds on infected foliage. He opined that this might serve as an effecive mode of spread during the hotter months of the year. Spores of *Phytophthora* have been isolated from the ant runs of *Crematogaster* (Turner, 1972). The ants

carry the spores along with soil while constructing tunnels on posts supporting pepper vines and thus spread the spores.

Climatic factors

The disease incidence is generally high during the south-west monsoon (July- September), when rainfall (2270-2990 mm) and relative humidity (91-99%) are high and minimum temperature (19-23°) is low. In Sarawak, maximum disease incidence was observed during October-March when the mean maximum temperature and minimum temperatures are about 26°C and 21°C. The mean rainfall during the period is 318-653 mm and daily mean sunshine 4.3 hr (Holliday and Mowat, 1963). The pathogen was successfully isolated in 60% of cases during October-March, but only in 43% during the dry months (April-September). Sarawak does not have a long dry season as the west-coast of India has. On overcast days with low mean sunshine hours and heavy rainfall, the temperatures generally fall, and such conditions are conducive for spore release.

The ambient temperature plays an important role in the infection process. When the pathogen is inoculated on rooted cuttings, symptoms of root necrosis and foliar yellowing appear in 3-4 days when inoculated plants are incubated at 20-25°C. The symptom expression is delayed when the temperature is 28°C and above (Nambiar et al. 1976). In one year old cuttings, symptoms appeared only after 35 days at 28-35°C. Selvaraj (1966) working on betal vine wilt found that wilt incidence was maximum (100%) at low soil temperature (20-23°C).

In pure pepper plantations in India, the disease generally becomes apparent during the south-west monsoon period. In mixed cropping systems such as pepper in arecanut gardens (which are generally irrigated during the dry period), the disease is noticed during the post-monsoon period (November–January) also. Since, during the winter months, the minimum temperature falls to 16-21°C and soil water content remains high due to frequent irrigations (once in 4-5 days), the microclimatic factors are very congenial for fungal growth, sporulation, and zoospore emission.

Soil factors

In India, Nambiar, Nair, and Money (1965) observed heavy incidence of the disease in neglected pepper gardens where inorganic fertilizers were not being applied. Kliejunas and Ko (1974) reported that deficiency of inorganic nutrients contributed to the heavy incidence of ohia decline (*Metrosideros collina* (Forst.) Gray subsp. *polymorpha* (Gang.) Rock) associated with *P. cinnamomi*. Broadbent and Baker (1974) reported that exchangeable Ca, Mg, N and organic matter were high in soils suppressive to root rot of avocado caused by *P. cinnamomi* as compared to soils conducive to the incidence of root rot. Working with quick wilt, Nambiar et al. (1965) reported that surface soils in diseased gardens contained lower levels of Ca, Mg, and K with high N. They suggested that if the ratios were lower than K=1.14 and $\frac{CaO+MgO+K_2O}{N}=3.80$, then the area would be prone to the disease. Huber and Watson (1974) found that the type of nitrogenous source also determined disease incidence in several root diseases involving pythiaceous fungi. At present, no information is available on the effect of nutrition on the incidence of pepper wilt. Hence, such studies are warranted in this line.

Survival

Holliday and Mowat (1963) observed that *P. palmivora* from pepper survived for about 15 weeks in naturally infected underground stems. The pathogen, however, has a low saprophytic ability. For instance, fresh vines

replanted six months after the death of affected vines, did not contract infection. Brasier (1969*a*) observed oospore formation *in vitro* when two compatible types of the pathogen were present under congenial conditions of low temperature (20°C), darkness, and adequate food supply. Oospore formation has been presumed to occur in nature, though rarely, in woody tissue or debris not exposed to light. We have not however been able to notice oospores so far in infected tissues. Holliday and Mowat (1963) also did not observe any fusion organ in nature. The mode of perennation and population build up of the pathogen during different seasons and under varied soil, water, and temperature regimes are to be studied in depth for developing effective control measures.

Host range

P. palmivora has been recorded to infect 138 species belonging to different families of angiosperms (Chee, 1969). Turner (1971*a*) reported that the isolates of *P. palmivora* from pepper in Sarawak were highly host-specific and none of the 43 species from 40 genera, belonging to 20 families other than the Piperaceae, was susceptible. Out of 32 *Piper* species tested, 30 species including *P. betle* were susceptible. All the seven *Peperomia* spp. were resistant. Further, the leaves of *Lycopersicon esculentum*, *Solanum melongena* and *Vinca rosea* and fruits of *Areca catechu* and *S. melongena* were also occasionally infected under laboratory conditions. The authors found that *P. palmivora* isolates from pepper in Kerala infected roots of *P. betle* and *P. longum*, pods of cacao and betel nut fruits. The *P. palmivora* isolates from rubber and cacao induced root necrosis in rooted pepper cuttings (Nambiar et al., 1976). Pepper gardens in the vicinity of rubber plantations usually show heavy incidence of pepper wilt. Holliday and Mowat (1963) reported that *Phytophthora* isolates from colocasia and cacao did not infect leaves of pepper, while an isolate from citrus did. According to Muller (1936), *Phytophthora* isolate from pepper in Indonesia showed similar characters in culture to those from coconut, rubber, cacao, and papaya. Isolates of the pathogen from pepper were, however, less virulent to the other host plants than those from the respective hosts.

Role of other associated organisms

Holliday and Mowat (1963) have often isolated *P. solani* and *R. bataticola* from the roots and stems of pepper. They appear to be the earliest colonisers of *Phytophthora* infected tissues. The present authors have also made similar observations. We have isolated *Pythium* sp. from tender discoloured roots of pepper vines showing foliar yellowing, and also from roots of quick-wilt affected vines and also established the pathogenicity of *Pythium* sp. on pepper. Incidentally, Holliday and Mowat (1963) have frequently isolated *P. splendans* from small roots of pepper in Sarawak and saw that the fungus caused damping off in pepper seedlings.

We have also isolated *Trichoderma* from roots of healthy pepper vines. The fungus is known to be an efficient biological agent in controlling *Phytophthora* (Baker and Cook, 1974). Since biological control of *Phytophthora* root rot of avocado (Zentmyer, 1963) and betel vine (Tiwari and Mehrotra, 1968) has been reported, investigations on the use of *Trichoderma* against foot rot pathogen in pepper may also yield valuable results.

The plant parasitic nematodes *Meloidogyne incognita* and *Radopholus similis* are being increasingly observed in pepper plantations. Holliday and Mowat (1963) observed that infestation by *Meloidogyne* sp. did not significantly enhance the susceptibility of pepper vine to foot rot. Selvaraj (1966) also observed similarly in the case of betel vine wilt. However, critical studies of the fungus-nematode interaction are required to be

carried out in the case of pepper under conditions of both pure cropping and mixed cropping.

Resistance

Muller (1930) reported the black pepper variety Belantung from Indonesia as resistant to foot rot. Holliday and Mowat (1963) found the Indian pepper cultivar Uthirankotta and the Indonesian varieties Djambi and Belantung possessed appreciable resistance. However, the present authors tested 40 Indian cultivars including Uthirankotta and 45 wild types and found all of them to be susceptible. Uthirankotta has also been found to be susceptible in Puerto Rico (Alconero et al., 1972) and Sarawak (Truner, 1973*a*). Turner (1973*a*), however, found Balancotta to be highly resistant. The observations of differences in reaction of a particular type may be attributed to the differences in virulence of the isolates of the pathogen.

As already reported, Turner (1971*a*) screened 32 *Piper* species and found that *P. colubrinum* and *P. obliquum* var. *eximum* were resistant. Albuquerque (1968*a*, *b*) had earlier reported resistance in *P. colubrinum* in Brazil. In Ghana, *Piper guineense* has been reported to be resistant (Anonymous, 1977). Ruppel and Almeyda (1965) reported that out of five *Piper* species tested, *P. aduncum*, *P. scabrum*, and *P. treleasanum* showed partial resistance. Several workers in Puerto Rico, UK, Brazil, and Malaysia (Sarawak) have successfully grafted *P. nigrum* on to a number of *Piper* spp., both resistant and partially resistant (Gaskins and Almeyda, 1969; Garner and Beakbane, 1968; Albuquerque 1968*a*, *b*; Turner 1973*a*). However, field establishment has been reported only in the combination involving *P. nigrum* and *P. colubrinum* (Albuquerque, 1968*a*, *b*; Gaskins and Almeyda, 1969). Either slow development of vascular tissues or healthy callus formation was suggested to be the cause for graft incompatibility. In some combinations involving *P. colubrinum* and cultivars like Balancotta, Kalluvalli, and Singpuri, Alconero et al. (1972) observed that longitudinal cracks developed at the graft union after a normal growth of four years.

Control

Only limited success has been achieved in controlling the disease with fungicides in trials carried out in Malaysia (Sarawak) and India. Holliday and Mowat (1963) reported that heavy doses of a copper oxide (Perenox) reduced the disease incidence slightly when it was forked into the soil in the basins of the vines. In Indonesia, Harper (1974) has recommended copper fungicides against *Phytophthora*. Among the ten different commercial formulations of fungicides tested by the present authors as foliar sprays and soil drenches around the vines, and before and after south-west monsoon, only Bordeaux mixture spraying and application of Bordeaux paste to the stem from the collar region to a height of one meter reduced the incidence. In tests against betel vine wilt in India, spraying and drenching the soil with Bordeaux mixture alone checked the disease (Narasimhan et al., 1976). None of the 29 chemicals tested by Turner (1973*b*) was fungicidal. He opined that concentration-volume-time interaction determined the efficacy of the formulation and soil permeability would be a major factor in penetration. In Sarawak, captafol has been reported to be useful as a soil drench against *P. palmivora* (Anonymous, 1972). We, however, did not find it to be effective. Recently, Noveroske (1975) reported pyroxychlor (Dowco 269), which is known to have basipetal translocation, to be effective against *P. parasitica* in tobacco. But, in our *in vitro* studies, this fungicide was ineffective even at 2000 ppm concentration.

Holliday and Mowat (1963) and Nambiar

and Sarma (1976) have stressed the need for adopting phytosanitary measures under field conditions to reduce the inoculum in soil and thus check the disease. These include isolation of infected plants from the surrounding healthy vines, ensuring better drainage facilities, and burning infected pits or drenching them with Bordeaux mixture before replanting. Chemical control measures are meaningless without adequate phytosanitary measures to check the disease spread. Turner's (1969*a*) observation of complete inhibition of sporulation and disease incidence at pH 3.0 indicates that some control of the disease is theoretically possible in soils with low pH, but it will not be possible to rule out nutritional disorders under such circumstances (de Waard, 1969). In India, where the pepper soils have a pH of 4.5-5.8, Nambiar et al. (1965) recorded low K, Mg, and Ca levels in diseased soils and recommended application of lime, magnesium, and potassic fertilizers in balanced amounts to prevent the disease.

The forgoing discussions show that application of fungicides alone will not be sufficient to control the disease. Chemical treatment is likely to be effective only when done in conjunction with other measures that will help to prevent the disease. Management practices play a major role in this context. While priority should be given to screening wild types of *Piper* spp. for locating resistance to the pathogen, ecological studies on the factors that predispose the vines to infection under different cropping system are also to be made. This will help in developing an efficient forecasting system to make the control measures effective and meaningful.

Slow Wilt

It is another serious disease of pepper in Kerala, India. Its etiology is only little understood. The disease is generally noticed in neglected gardens. It has been described variously as 'slow decline' (Rutgers, 1915) and 'yellows' (Bregman, 1940). In Guyana, the yellows disease caused 30% loss to the crop (Bisessar, 1969), while in Bangka island (Indonesia), Hubert (1957) estimated the loss at 90%.

Symptoms

Slow wilt is usually observed after the north-east monsoon. The initial symptoms are general yellowing of lower leaves and loss of natural lustre in them. The yellowing gradually progresses upwards. The affected leaves become flaccid and fall off. The tip burn symptoms in leaves and die back of twigs are very common. The vines exhibiting foliar yellowing show root knots and varying degrees of necrosis of both feeder and mature roots in most of the gardens examined by the authors. The affected vines die gradually after the appearance of external symptoms. The affected shoots and roots show vascular browning, but not consistently.

Etiology

Fungi. Menon (1949) isolated *Fusarium*, *Rhizoctonia*, and *Diplodia* from roots of affected vines. Though *R. bataticola* and *Fusarium* sp. have been often isolated from the roots, pathogenicity tests gave negative results except in the case of one isolate of *Fusarium* sp. (Anonymous, 1973). Incidentally, *R. bataticola* has been reported to cause considerable loss in *P. betle* in Karnataka (Nema and Nayak, 1976).

Nematodes. Koshy (1977, unpublished) has observed that many affected gardens in Cannanore District (Kerala) are infected by the root knot nematode *Meloidogyne incognita*. Delacroix (1902) was the first to record *Meloidogyne* sp. in pepper. Other reports of the occurrence of this nematode in pepper gardens are of Sharma and Loof (1974) in Brazil and Ting (1975) in Malaysia. We have noticed root knot nematode infestation in

Erythrina indica, a popular live standard of pepper in India, and in weeds such as *Ageratum conyzoides* and *Corchorus acuntangulus*. Koshy (*loc. cit.*) has found another popular standard *Garuga pinnata* to be resistant to root knot.

In addition to the root knot nematode, occurrence of the burrowing nematode *R. similis* in the root zones of pepper has also been reported from Karnataka (Kumar, Viswanathan, and D'Souza, 1971) and Kerala (Seshadri, 1972, unpublished). Van der Vecht (1950) in Indonesia was the first to ascribe the slow wilt disease to infestation by the burrowing nematode. Recently, Koshy (1977, unpublished) observed in an arecanut garden mix-cropped with pepper at Kasaragod, high populations of *R. similis* in the roots of pepper vines showing foliar yellowing. Venkitesan (1976) established the pathogenicity of *R. similis* on pepper in India and discussed its possible role in the incidence of slow wilt. *R. similis* infestation has been known to wipe out pepper cultivation in Bangka in Indonesia damaging about 90% vines (Hubert, 1957). Christie (1957, 1959), studying the symptoms of pepper yellows in Indonesia caused by the nematode, observed numerous root lesions which promoted discoloration and degeneration of feeder roots. The presence of *R. similis* in roots of pepper trained on arecanut gains more importance since the nematode has been reported to infect arecanut also, in addition to coconut and banana (Koshy et al., 1975). Thus, both root knot and burrowing nematodes are associated with the root system of pepper vine either individually or jointly. Pepper is grown in a majority of homesteads in Kerala along with coconut or arecanut, and with intercrops like banana, ginger, turmeric, and vegetables like okra, brinjal, etc., the situation is very congenial for multiplication of both the nematodes.

Barat (1952) working with *Meloidogyne* sp. and Throne (1961) with *R. similis* infesting pepper in Indonesia considered that the nematodes initiated the attack on roots and the destruction was completed by secondary organisms. Thereafter, the pathogens in combination with nemas incited the yellows disease. The nematodes may predispose the host to attack by other microorganisms either because of the injury they inflict, or due to alteration of host metabolism conducive to parasitic invasion (Powell, 1971). The study of host parasite interaction and the changes in the host-metabolism in pepper as a result of attack are to be clearly understood to assess the role played by these agents in the slow wilt disease complex.

Soil factors. Harper (1975) has stressed the importance of nutrition in pepper in the control of pepper yellows in Indonesia. De Waard (1969) reported that in Sarawak, potassium-deficient pepper vines showed necrosis of the distal end of the mature leaves. We have observed here similar symptoms in leaves in a few of the pepper vines. Analysis of a limited number of leaf and soil samples from healthy and disease affected gardens has shown a lower content of K and P in the tissues of affected vines and their basins (Wahid and Haridas, 1976, unpublished).

Foliar yellowing is first noticed after the monsoon and reaches its maximum in April-May. It is more intense in gardens where the vines are exposed to direct sunlight. In a majority of cases, it persists throughout the year in varying intensities. However, nearly one-fourth of the vines which exhibit yellowing during summer months recover during the rainy season. This indicates that water stress may also be associated with slow wilt. If yellowing recurs during two to three consecutive summers, the vines seldom recover.

Control

The general recommendation is drenching the base soil with 0.1% ceresan wet before and after the monsoon rains. But this does not control the disease in majority of cases.

However, recent field trials indicate that application of fensulfothion granules @ 20 g per standard may be helpful in reducing foliar yellowing (unpublished). Since root knot infestation is very common in pepper nurseries, disinfectation of potting mixture using methyl bromide @ 500 g ton of soil under polythene cover for 24-48 hr is advisable to prevent the spread of nematodes to pepper gardens.

Harper (1975) observed that many a time application of fungicides and nematicides, both alone and in combination, did not give the desired results, but gave better results when these treatments were combined with fertilizer application. The practice of earthing up adopted in certain plantations helps to induce regeneration of roots, which is important since the ratio of root degeneration to root regeneration determines the health of vines. In Cambodia, Litzenberger and Lip (1961) found that vines mulched with *Eupatorium odoratum* controlled pepper yellows caused by *H. marioni* and *H. splendans*. Since *Garuga pinnata* is reported to be resistant to root knot nematode, this species may be used as standards. As a long term measure, resistant types of pepper are to be located by taking up extensive screening trials.

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