6

Major Crops Affected by *Phytophthora*

Diversity and Management of *Phytophthora* in Southeast Asia
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6.1 *Phytophthora* on Cocoa

Peter McMahon[1] and Agus Purwantara[2]

Abstract

Phytophthora pathogens are responsible for some of the most serious diseases of cocoa including phytophthora pod rot (PPR) or black pod, stem canker, leaf and seedling blight, chupon wilt and flower cushion infections. PPR causes 10–30% annual losses in production of cocoa beans globally, and much higher losses locally in particularly wet and humid conditions. Stem canker causes further losses and also tree deaths. Eight species of *Phytophthora* have been isolated from diseased cocoa, but most losses in production are caused by *Phytophthora palmivora*, *P. megakarya* and *P. citrophthora*, which cause similar diseases with slightly varying symptoms. Worldwide, *P. palmivora* is one of the most serious pathogens on cocoa, and in Southeast Asia this species accounts for almost all of the phytophthora diseases of cocoa. The most effective control measures are the introduction of resistant cocoa genotypes and farm management practices such as removal of infected pod husks, proper pruning of the canopy and judicious selection of shade species and associated crops.

Introduction

Among the numerous pathogens of cocoa (*Theobroma cacao* L.), species of *Phytophthora*, notably *Phytophthora palmivora* with a worldwide distribution and *P. megakarya*, which is restricted to West Africa, cause serious losses. Diseases of cocoa can be grouped into those that have spread with cocoa from its centre-of-origin in the Amazon region, and new-encounter diseases, which have transferred from other plants in regions to which cocoa has been introduced (Keane 1992). Phytophthora diseases probably fall into the 'new-encounter' group. The original hosts from which the various *Phytophthora* pathogens on cocoa transferred remain unknown. Since both *P. palmivora* and *P. megakarya* have a wide host range (Erwin and Ribeiro 1996; Opoku et al. 2002); it is likely that such transfers have occurred more than once. However, a study of the genetic diversity of isolates collected from different regions around the world suggests that at least some of the distribution of *P. palmivora* on cocoa outside its centre of origin has been clonal, which suggests that it has spread with its host (Alex Appiah, pers. comm.).

Impacts of Phytophthora on Cocoa Production

The main regions of cocoa production are West Africa, Central and South America and Southeast Asia/Pacific, with more than half the world's cocoa being produced in West Africa **(**World Cocoa Foundation, **<**www.chocolateand cocoa.org/Supply/supplyindex.htm**>).** Southeast Asia, particularly Indonesia, is becoming an increasingly important centre of cocoa production. However, production in this region is affected by three main disease and pest problems: cocoa pod borer (*Conopomorpha cramerella*), vascular-streak dieback caused by *Oncobasidium theobromae* and phytophthora diseases caused by *P. palmivora* (Figure 6.1.1).

It is difficult to estimate yield losses due to phytophthora diseases since the same species may cause a number of diseases, and environmental conditions, particularly rainfall and humidity, can have a dramatic effect on disease incidence and severity (Thorold 1955; Tollenaar 1958). Most phytophthora-related losses can be attributed to phytophthora pod rot (PPR), followed by stem

[1] Department of Botany, La Trobe University, Bundoora, Victoria 3086, Australia.

[2] Biotechnology Research Institute for Estate Crops, Jalan Taman Kencana 1, Bogor 16151, Indonesia.

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cankers. It is commonly estimated that 10–20% of the world's annual production is lost due to PPR, but estimates vary from average annual losses of 10% (Padwick 1956) up to 30% (Medeiros 1977; Opeke and Gorenz 1974), with much higher losses in particularly wet locations or wet years. In Western Samoa, losses of 60–80% due to PPR in wet years were reported by Keane (1992). Data collected at Keravat, Papua New Guinea, over an 18-year period indicate a mean annual loss of cocoa yield due to PPR of 17% and a range from 5–39% (Holderness 1992). In Mexico, losses of up to 80% due to PPR were reported by (Rocha 1965). Surveys in Java indicated that the percentage of pod rot ranged from 26 to 56% (Pawirosoemardjo and Purwantara 1992). If the impact of other phytophthora diseases such as stem canker were taken into consideration, these figures would be even higher. Stem canker contributes to production losses although these are difficult to assess, and can also cause tree deaths. A survey in Solomon Islands by Friend and Brown 1971) indicated tree losses to phytophthora canker averaged 3% annually over 5 years, with losses of trees approaching 40% in one locality.

Figure 6.1.1 Black pod in cocoa caused by *Phytophthora palmivora* in Indonesia.

Cocoa Agrosystems

Wild populations of *Theobroma cacao* in the Amazonian forest are shade adapted shrubby trees growing under the rainforest canopy. Over-storey shade trees used on cocoa farms include coconuts (particularly in Southeast Asia), legumes such as *Leucaena* and *Glyricidia,* and even rainforest trees left standing after partial clearing. The shady conditions produced by over-storey shade trees and the dense foliage of cocoa itself provide favourable conditions for oomycete pathogens such as *Phytophthora* spp. Over-storey shading, unpruned cocoa canopies (self-shading) or high-density plantings can reduce the movement of air, leading to increasing humidity, highly favourable for *Phytophthora*. Conversely, removing shade trees completely may result in epidemics of *Colletotrichum*-related diseases and increase insect pest populations on cocoa (Smith 1981). To reduce pest and disease problems, a balance is needed that optimises both shade conditions and air movement within the cocoa canopy. Smith (1981) pointed out that, in Papua New Guinea, cocoa experiences fewer pest and fungal pathogen problems when grown under tall shade (e.g. coconut) than under low shade (e.g. *Leucaena*).

The choice and management of shade crops is important in integrated approaches to managing phytophthora diseases considering the fact that some shade trees (e.g. coconut) are also hosts of *Phytophthora* pathogens (Smith 1981; Opoku et al. 2002). The possibility that *P. palmivora* on coconut could infect cocoa trees growing on the same farm needs to be considered, although budrot is rare in the endemic tall palms of Southeast Asia. Judicious inter-planting with non-host plants (e.g. for wind breaks, insect breaks or alternative sources of income), or use of non-hosts as shade trees, could reduce transmission of *Phytophthora* infections. However, the economic value of the shade tree will also affect choice. In parts of Vietnam, where the cocoa industry is relatively new, durian trees, which are affected severely by *P. palmivora*, are the shade species of choice on cocoa farms because of the high financial returns from durian fruit (David Guest, pers. comm.). An important question in these areas will be whether *P. palmivora* can cross infect between the two tree crops and give rise to increased disease problems on both host plants. Disease management in intercrops and mixed plantings has to include all components, although mixed plantings are less vulnerable to explosive epidemics seen in monocultures.

Phytophthora Pathogens of Cocoa

Phytophthora pathogens thrive on all parts of the cocoa plant from the seedling to mature stages, causing a number of diseases. To date, eight species of *Phytophthora* have been isolated from cocoa: *P. palmivora* (Butler) Butler, *P. megakarya* (Brasier and Griffin), *P. capsici* (Leonian emend.) (= *tropicalis*), *P. katsurae* (Ko and Chang), *P. citrophthora* (R.E. Smith and E.H. Smith), *P. arecae* (Coleman) Pethybridge, *P. nicotianae* (van Breda de Haan) and *P. megasperma* (Dreschler) (Erwin and Ribeiro 1996; Iwaro et al. 1997; Appiah et al. 2003). Throughout the world

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most damage is caused by *P. palmivora* and, in particular localities, by *P. megakarya* and *P. citrophthora* (Brasier and Griffin 1979; Brasier et al. 1981; Kellam and Zentmyer 1981). These three pathogens cause similar diseases including PPR and stem canker, although symptoms and pathology may vary slightly (Lass 1985). For example, in West Africa both *P. palmivora* and *P. megakarya* infect cocoa pods, causing pod rot or black pod and both these species also cause stem cankers.

Following Turner's identification of distinct strains of '*P. palmivora*' isolated from West African cocoa (Turner 1960), Brasier and Griffin (1979) designated three morphological forms, MF-1, MF-3 and MF4 as separate species. Only MF-1 was clearly *P. palmivora*. MF-4 was identified as *P. capsici* or a similar species and MF-3 as a new species, *P. megakarya*. MF-4 has recently been described as a separate species, *P. tropicalis* (Aragaki and Uchida 2001). Possibly other taxa will be found in the *P. capsici–P. tropicalis* complex (Appiah et al. 2003). MF-2 (Waterhouse 1974b) was not accepted as a valid taxon by Brasier and Griffin (1979).

Chowdappa and Mohanan (1996)reported that PPR in India was associated with *P. citrophthora*. This pathogen has been reported to occur on cocoa in Brazil (Campelo and Luz 1981; Kellam and Zentmyer 1981), in Cameroon (Lass 1985) and in Indonesia (Appiah et al. 2003). In Brazil, *P. capsici* is often isolated from PPR-affected pods (pod lesions) along with *P. palmivora* (Campelo and Luz 1981), although it is likely that the main causal pathogen is *P. palmivora*. *P. capsici* has been reported on cocoa in Kerala, India (Chowdappa and Mohanan 1996). *P. megasperma* was found on cocoa in Venezuela (Zadoks 1997) and *P. katsurae* on cocoa in Sri Lanka (Liyanage and Wheeler 1989). *P. palmivora* is the main species attacking cocoa throughout Southeast Asia where, under conditions favourable it is able to infect the pods at all stages of development (causing pod rot and cherelle wilt), the flowers and flower cushions, the main trunk (causing cankers which sometimes lead to death of the tree), the chupons (causing chupon wilt), the young growing twigs and young leaves of mature trees sometimes leading to repeated defoliation, dieback and death of the tree, the petiole and lamina of old leaves (causing leaf blight), and the young seedlings (causing seedling blight) (Gregory 1974; Lass 1985).

Few studies have been done to compare the pathogenicity of different species or different isolates of *Phytophthora*. In one study, Kellam and Zentmyer (1981) transplanted germinated cocoa seeds into soil artificially infested with chlamydospores or oospores of *P. palmivora*, *P. citrophthora* or *P. capsici*. After 8 weeks, they found that *P. capsici* had not caused any seedling mortality, while infection with *P. palmivora* and *P. citrophthora* resulted in mortality rates of 67% and 53%, respectively. In Brazil, Campelo et al. (1982) reported that, on healthy, detached pods, *P. citrophthora* was more pathogenic than both *P. palmivora* and *P. capsici* (see Lass 1985). Liyanage and Wheeler (1989) found that, compared to *P. palmivora*, *P. katsurae* is only mildly pathogenic. Five days after inoculation of healthy, detached pods, *P. palmivora* had produced over 10-fold larger lesions than had *P. katsurae*.

Disease symptoms

Phytophthora pod rot begins on the surface of the pod. Infection starts as a discoloured spot, then develops into a brown or black lesion with a well-marked boundary, and spreads over the entire pod within about 2 weeks. On older pods, infections mostly start at either the tip or the stem end of the pods. Equatorial infections are usually associated with damage to the pod surface or wounds. The rot involves the whole of the fleshy tissue of the husk as well as the pulp and seeds (Figure 6.1.2). Infection of pods approaching ripeness when the seeds are no longer in close contact with the husk may not lead to infection of the seeds, which therefore can be salvaged and fermented. The pathogen appears on the surface of the pod as a whitish down on which masses of sporangia are produced. The pod ultimately blackens and shrivels, and is colonised by secondary fungi. PPR is a firm rot that can be distinguished from pod rot caused by *Botryodiplodia theobromae*, which causes loss of firmness in the pod wall and relative dryness of the diseased tissue (Thrower 1960a), and from infections by *Colletotrichum* which result in dark, often sunken lesions. Cherelle wilt (Figure 6.1.3) may be caused by *P. palmivora* but this needs to be distinguished from physiological wilt which may be related to stress associated with excessive fruit set (Thrower 1960b).

Stem canker is characterised by development of brown necrotic bark around the trunk. When the surface of the bark is scraped off, the affected tissues become watery to gummy and of a dull brownish-grey colour that often assumes a claret tone on exposure (Figure 6.1.4). The necrosis does not extend into the wood beyond the cambium layer. When the canker enlarges, it may encircle the trunk, causing 'sudden death' of the tree. In Papua New Guinea, cankers were found to be associated with channels made by larvae of the insect pests, *Pantorhytes* and *Glenea* (Prior and Sitapai 1980). Additionally,

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contaminated pruning implements, diseased pod peduncles and diseased pods in contact with the bark are sources of inoculum (Vernon 1971; Brown and Friend 1973). Flower cushion cankers result from contaminated harvesting knives, or by visits from flying beetle vectors (Konam and Guest 2004).

Figure 6.1.2 Black pod rot on the inside of infected pods.

The pathogen naturally attacks and kills unhardened (flush) leaves and young green stem tissue. It also infects mature leaves, even though this is not normally regarded as being serious (Manco 1966). Infection of flush leaves and stems can lead to death of the growing point or of the whole plants in the case of seedlings, and can cause bark cankers when the pathogen spreads down a chupon (chupon wilt). Cocoa seedlings grow very rapidly in the first few months and produce young leaves that are highly susceptible to pathogen attack.

Figure 6.1.3 Cherelle wilt in cocoa

Disease cycle

On cocoa farms, *Phytophthora* is dispersed by rainsplash (from infections on the plant, often as sporangia, and from the soil), and by vectors such as ants and flying insects (Dade 1927, 1928; Evans 1971, 1973a,b; Gregory et al. 1984; Konam 1999; Konam and Guest 2004). The most important infective propagules of *Phytophthora* are motile zoospores. Rainsplash probably disperses sporangia (*Phytophthora* spp. on cocoa have deciduous sporangia) followed by release of zoospores. Encysted (dormant) zoospores, chlamydospores and hyphae might be other forms of inoculum (Turner 1965; Gregory et al. 1984). Both *P. palmivora* and *P. megakarya* can survive for up to 4 months in cocoa roots and soil, as was shown by Opoku and Wheeler (1998) (Konam and Guest 2002).

For *P. palmivora* in the Southeast Asia–Pacific region, flower cushions are likely to be particularly important reservoirs of infection (Brown 1973). Additionally, infected plant parts and cocoa pods left on the ground or in the canopy after harvest (especially as there is a tendency not to harvest black pods) provide a large proportion of inoculum for *Phytophthora* pathogens generally (Ward and Griffin 1981; Purwantara and Pawirosoemardjo 1990; Konam 1999).

Figure 6.1.4 Stem canker in cocoa tree

In Nigeria, where the predominant *Phytophthora* pathogen is *P. megakarya*, a long-term research study on PPR demonstrated that rainsplash from or contact with infected pods accounted for more than 71% of pod losses (Gregory et al. 1984). Other sources of infection included soil (5%), ant tents (5.8%) and pod-damage due to insects and rodents (4.9%) with 10.9% attributed to 'no obvious' sources. Rather than disease spreading from a few initiator pods, it appeared it spread from numerous 'initiator' pods with sources for these initial infections being partly derived from the soil and ant tents, but also largely (40%) from 'no obvious sources' (Griffin et al. 1981; Gregory et al. 1984). Observations on infection sources of Amazonian, Amelonado and Trinitario cocoa types in Java for three years (1990–1992)

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showed similar results (Purwantara 2003). Contact or splash from infected pods accounted for about 35% of infection. On average, living vector activity accounted for about 14%, whereas infection from soil and cankers was only 3% and 7%, respectively. Almost 40% of sources of infection were not identified (no obvious source) (Table 6.1.1). This high percentage could be due to the activity of living vectors such as squirrels and rats, which carry spores that are disseminated onto healthy pods. Almost 12% of infection was associated with rodent damage.

In addition to the possibility that inoculum is carried upwards by convection of aerosol-sized water droplets as well as larger rain-splashed drops (Gregory et al. 1984), tent-building ants are likely to be important agents of vertical spread (Evans 1971, 1973a,b; Newhook and Jackson 1977; Gregory et al. 1984; McGregor and Moxon 1985; Konam 1999). In Papua New Guinea, Konam (1999) established that tent building and/or path building ants were strongly associated with PPR incidence. When Konam dislodged ants and ant tents from cocoa trees and then prevented access of ants from the soil by applying grease near the base of the trees, the incidence of PPR was significantly lower than in untreated trees, and the treatment also led to significantly increased yields. These results were obtained even when infected pod husks were scattered under the trees, suggesting that ants, rather than flying insects, provide most of the inoculum that infects healthy pods. However, it is apparent that flying insects also play an important role in inoculum dispersal (Konam and Guest 2004).

Potential agents of horizontal spread of *Phytophthora* are wind-dispersed spores or water droplets and flying insects and other fauna. Wind appears not to be an important factor in horizontal spread of *Phytophthora* (Evans 1973a; Wharton 1955). However, in West Africa, Thorold (1954, 1955) trapped zoospores above infected pods, indicating some spores were wind dispersed (Waterhouse 1974a). Konam (1999) established that in Papua New Guinea two types of flying beetle, a scolytid and a nitidulid, preferentially visited and bored holes in infected pods. The beetle frass contained viable spores. He concluded that the beetles' frass provided a new source of inoculum that could be dispersed by water and perhaps wind (the dust-like frass could be blown around).

Intra-specific Diversity of *Phytophthora* Species

Both mating types of *P. palmivora*, A1 (once called the 'rubber' type) and A2 (the 'cacao' type), are found on cocoa with the A2 mating type predominating (Turner 1961; Zentmyer 1974). Of 70 *P. palmivora* isolates collected from around the world by Appiah et al. (2003) only 16 were of the A1 mating type. In contrast, 19 of 29 *P. palmivora* isolates collected from non-cocoa hosts for the same study were predominantly of the A1 mating type. Oospores have never been found in the field on cocoa, although they are obtained in culture when A1 and A2 types are plated together (Tarjot 1974).

Turner (1961) found that isolates of *P. palmivora* collected from cocoa around the world were remarkably uniform morphologically, consistent with sexual isolation (Zadoks 1997). Brasier and Griffin (1979) and Appiah et al. (2003) also found that the morphology of international collections of *P. palmivora* was relatively uniform. Furthermore, molecular studies indicated that *P. palmivora* isolates collected from different regions around the world (including Central America, West Africa, Southeast Asia, Taiwan and Papua New Guinea) have a greater genetic uniformity than *P. megakarya* isolates collected from different regions of Africa (A.A. Appiah et al., unpublished data).

Table 6.1.1 Percentage of incidence of pod infection on three cocoa types in Java during 1990–1992. Source: Purwantara (2003).

Likely source of infection	Incidence of pod infection (%)			Mean incidence of infection (%)
	Amazonian	Amelonado	Trinitario	
Contact/splash from another pod	39.6	33.3	33.5	35.5
Soil and litter	2.8	5.7	1.5	3.3
Cushion and canker	8.4	7.2	6.2	7.3
Rodent damage	7.9	12.0	14.9	11.6
Ant tent	0.3	0.1	0.2	0.2
Harvest damage	2.6	1.8	1.5	2.0
Insect damage	1.8	3.2	1.7	2.2
No obvious source	36.6	36.7	40.5	37.9

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Important questions requiring further study are the host-specificity of different isolates of *P. palmivora*. A case where direct inoculation demonstrates that a rubber isolate can infect cocoa, for example, might be more complex in a field situation where a variable *P. palmivora* population may be present. Thus, any *P. palmivora* population may contain a range of strains, only some of which are pathogenic and only some of these being able to infect more than one host, the others being host specific.

Host Resistance

Despite its obvious importance in disease control, the study of resistance to *Phytophthora* in cocoa has been neglected. There has been much confusion about methods for studying and measuring resistance. For example, resistance to stem canker may not be linked to resistance to PPR. The Forastero clone, Sca-6, is resistant to PPR (Okey et al. 1995) but susceptible to canker (Okey et al. 1996), and in Papua New Guinea, the KA2-101 clone is susceptible to PPR (McGregor 1981) but is less affected by canker (Prior and Sitapai 1980).

Resistance of particular cocoa clones observed in one country may not be evident in another, presumably due to varying environmental conditions or variations in the pathogenicity of different regional populations of *Phytophthora* (e.g. Lawrence 1978; Saul 1993). Resistance found in laboratory and glasshouse studies is not always evident in the field. The interaction of different species, and possibly strains, of *Phytophthora* is another factor to consider, although Zadoks (1997) considers that there is little evidence to contradict the hypothesis that host resistance to PPR is effective against different *Phytophthora* pathogens. When testing 10 cocoa clones for resistance to *P. palmivora* and *P. capsici*, Iwaro et al. (1998) obtained a similar ranking order, although *P. palmivora* was the more aggressive species. Another problem is that resistance tests on detached plant parts might not correlate with results from attached plant parts, although Iwaro et al. (1997) found that results from resistance tests on leaves and pods were similar whether they were detached or attached.

Despite all the above-mentioned difficulties, in cocoa-growing countries, there are consistent differences in the incidence of pod rot and canker on different varieties. In Indonesia, phytophthora diseases are generally most severe in Criollo type varieties. At the beginning of the last century, canker was very serious in Java, leading to the eradication of a very susceptible Criollo-type (Van Hall 1912, 1914). However, canker is no longer a menace in this area since Criollo has been replaced by relatively more resistant Forastero types or Criollo-Forastero hybrids (Tollenaar 1958).

Amelonado varieties are also susceptible to canker. The cocoa genotypes currently widely planted in Malaysia, Indonesia and Papua New Guinea are mostly hybrids between Upper Amazon and Trinitario types, with Amelonado types in Sabah and Sulawesi, which are susceptible to pod rot and canker to different degrees. In Indonesia smallholder cocoa plantations are genetically diverse, and hybridisation occurs between out-crossing genotypes, making local selections a promising source of resistance.

Some sources of resistance to *Phytophthora* are found in varieties from Upper Amazon, Costa Rica (crosses between Trinitarios and an Amazon-type local genotype), Bahia (Catongo and related clones) and Ecuador (e.g. the clones Sca-6, Sca-12) (Soria 1974). Van der Vossen (1997) lists some cocoa clones with demonstrated resistance to *P. palmivora*, including P7, PA-150, EET-50, IMC-47, Sca-7, Sca-6, Sca-12 and K82. In Malaysia, PBC-123 and BR-25 are recommended for PPR resistance. In Papua New Guinea, long-term studies have shown differences between clones in their resistance to *Phytophthora* (Saul 1993). For example, in a particular year, K82 has been consistently ranked with a lower disease incidence compared to other clones over a number of years (Figure 6.1.5).

PPR resistance is mostly partial, involving reduced incidence of pod infection and reduced rates of expansion of lesions on infected pods (Saul 1993). However, A.J. McGregor (unpublished data) recorded varying responses in lesion development. Some lesions were small black spots or even barely discernible, consistent with restricted expansion due to cell death (Saul 1993). Phillips-Mora and Galindo (1989) also described some reactions of pods that were similar to the sudden collapse of tissues associated with hypersensitive necrosis. However, resistance to PPR controlled by a single gene with a strong effect has not been demonstrated. Resistance to *Phytophthora* in certain clones (e.g. Sca-6, K82, RJ-2) appears to be durable on the evidence that field tests have been conducted over a long period of time and no erosion of resistance has been observed (Figure 6.1.5).

Mechanisms of Resistance

Mechanisms of resistance in cocoa to *Phytophthora* pathogens are poorly understood. Iwaro et al. (1997) identified two aspects of resistance to *Phytophthora* operating at the penetration and post-penetration

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stages of infection, with the poor correlation between the two suggesting that they are independent. The PPR-resistant Sca-12 clone had a high number of small lesions on pods (indicating a post-penetration rather than penetration mechanism of resistance), but it had few lesions on leaves (indicating resistance at the penetration stage in leaves). The authors therefore concluded that leaf tests for resistance could not be used to indicate resistance in pods. However, others have found good correlation between expressions of resistance in leaves (or leaf disks) and pods (Van der Vossen 1997).

Okey et al. (1995) compared the response of six genotypes of 3-month-old cocoa plants inoculated with *P. palmivora* into wounds in the stem. They found that larger lesions were obtained in genotypes that produced lower quantities of lignin at the wound sites, while poor correlation was found between lesion size and other wound healing components (suberin and callose). In a further study with 6-month-old cocoa, Okey et al. (1996) found that lower resistance to canker was associated with relatively low levels of bark hardness and relatively high levels of moisture in the bark.

Control of Diseases Caused by *Phytophthora*

Farm management practices

Various cultural management practices employed on cocoa farms can effectively control phytophthora diseases, particularly in conjunction with a degree of host resistance (Muller 1974; Toxopeus 1974). Disease is prevalent in wet areas. Humidity levels of nearly 100% during the night result in condensation of free water, which is essential for infection. Disease incidence is increased by poor drainage of the plantation, and high humidity due to a heavy canopy and low branching of the trees. Pruning of cocoa and removal of low branches, combined with a reduction of shade to the minimum required for good growth of the cocoa, can contribute substantially to the control of phytophthora diseases. Not only does pruning allow increased air circulation and more rapid drying of the pod surfaces, but it facilitates complete harvesting of pods (including infected pods) and application of fungicide if required.

Cultural practices involving sanitation contribute substantially to control of phytophthora diseases in cocoa, although experimental studies are needed to quantify this. Such practices include regular complete harvesting of both healthy ripe pods and any infected pods, including pod mummies, which can remain sources of infection for long periods, and burying of infected pods and pod husks. Addition of manure (e.g. green vegetable matter plus chicken manure) can be used to hasten decomposition of pod remains and encourage the release of ammonia and stimulation of saprophytic microbes that will kill *Phytophthora* (Konam 1999; Konam and Guest 2002). Occasional application of a protective fungicide (e.g. in the dry season) or trunk injection of phosphonate could be used to kill surviving inoculum sources (in flower cushions, pod mummies and rough bark).

Chemical control

Copper fungicides have been used since the early 1900s to control pod rot (Tollenaar 1958). Cuprous oxide has consistently been shown to give good control of the disease (Newhall 1967). Metalaxyl became available in the late 1970s, and was found to be effective in controlling the disease (McGregor

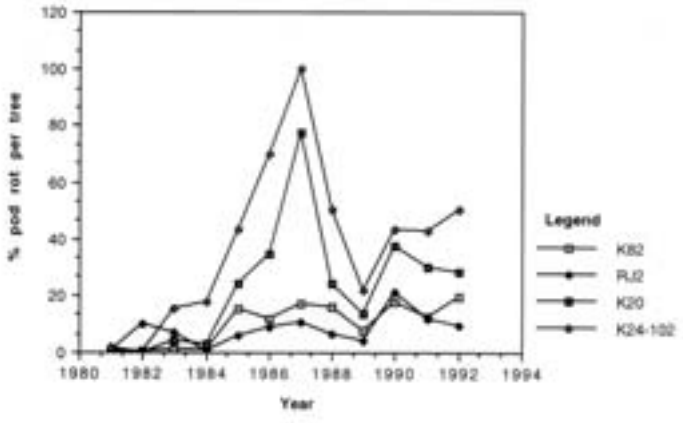

Figure 6.1.5 Percentage pods per tree infected by phytophthora pod rot in four Keravat clones, for the period July 1981 to December 1992 (from Saul 1993)

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1982, 1984). The timing of application may be important (Mabbett 1986). However, these sprays provide only limited protection, particularly during the wet season when heavy rains are likely to wash away chemical treatments. Also, development of resistance to metalaxyl is likely since such resistance developed in *P. infestans* on potato crops (Erwin and Ribeiro 1996). Even if fungicides are effective, factors such as potential yield of the cocoa tree and cocoa prices have to be considered in determining the profitability of fungicide spraying (Fagan 1984; McGregor 1983).

Work with mature cocoa in Papua New Guinea demonstrated that PPR and stem canker could be controlled effectively by potassium phosphonate applied by injection (Anderson and Guest 1990; Guest and Grant 1991; Guest et al. 1994). Yields were almost doubled with twice-yearly trunk injections of 10% phosphonate solutions (Anderson and Guest 1990; Guest et al. 1994). Phosphonate is a simple inorganic compound that apparently operates in conjunction with physiological factors in the plants. Since it appears to be more toxic to the pathogen in planta than in vitro (Guest and Grant 1991). It specifically controls oomycete pathogens and is also more economic for the farmer than other treatments (Guest et al. 1994). Moreover, it avoids the problem of removal of surface treatments by rain, and involves very simple equipment (hand-drill and spring-loaded syringes). Uptake of this method has been slow; – Indonesian growers, for example, have been reluctant to adopt this control technique because of the wounding that results from multiple and regular injections (Yohannes Junianto, pers. comm.).

Biocontrol and Natural Plant Extracts

Odigie and Ikotun (1982) showed that *Botryodiplodia theobromae*, *Gliocladium roseum*, *Penicillium* spp., *Bacillus cereus* and *B. subtilis* inhibit the growth of *Phytophthora palmivora* in vitro and in vivo.

Plant extracts are another possible 'biological' treatment and testing such extracts against various pathogens is very active in some tropical countries. Awauh (1994) identified plant extracts that suppress PPR lesion development but their effectiveness is too short-lived (only 3 hours) to be useful for control purposes. Chapter 7.5 describes the development of microbial biocontrol agents for the control of black pod.

Selecting and breeding for resistance

Since cocoa genotypes are highly variable, and resistance to *Phytophthora* pathogens has been evident in the field, there is a great deal of potential for deployment of more resistant genotypes (Toxopeus 1974; Zadoks 1997).

The resistance observed to date has been partial, additively inherited and apparently durable, and so is likely to be of long-term benefit to farmers. Quantitative trait loci (QTLs) in cocoa linked to *Phytophthora* resistance have been identified (Flament et al. 2001), providing a promising approach to improving predictability of resistance and thereby speeding up breeding programs (Van der Vossen 1997; Zadoks 1997).

Resistance to *Phytophthora* has been identified in some cocoa clones (see Host resistance), but these clones may not be suitable for propagation. For example, Sca-6 and Sca-12 are quite resistant to *Phytophthora*, but have a small bean size. To date, there has been little attempt to incorporate genotypes with known resistance to PPR (like Sca-6, PA-7, K82) into cocoa breeding programs. Such clones could be crossed with agronomically desirable clones to produce hybrids from which a wider range of genotypes with resistance could be selected on farms.

Rapid screening methods involving inoculation of pods, leaves or leaf disks may save considerable time and labour, since screening for resistance to phytophthora diseases in the cocoa field can take years (Blaha 1974; Lawrence 1978; Zadoks 1997). Good correlation may be found between rapid screening methods, such as leaf disc tests, and field tests (Nyasse 1997; Efron and Blaha 2000). It is important that rapid screening be supplemented by confirmation of resistance in the field. Saul (1993) developed an inoculation method in the field by transferring inoculum onto a pod by tape (the 'band-aid' method). This allows rapid assessments for resistance (Figure 6.1.6).

In Indonesia, trees relatively free of PPR have been observed next to heavily infected trees (Arief Iswanto, Indonesian Coffee and Cocoa Research Institute, Jember, pers. comm.). In Indonesia and Papua New Guinea, farmers recognise trees with superior yield of healthy pods; such trees are likely to have a degree of resistance to phytophthora diseases. These trees can be propagated clonally for experimental testing of their performance. Budwood can be side-grafted onto existing trees on a farm, allowing on-farm selection for PPR resistance. For example, farmers and extension officers could select budwood from potentially resistant cocoa genotypes and side-graft these onto susceptible genotypes or any rootstock that is available. The mother tree can eventually be pruned back to allow

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the side-grafted resistant genotype to replace the original tree. This approach has been initiated by an ACIAR project (PHT/2000/102) based in Sulawesi, Indonesia. It is very suitable for the smallholder farmer and local extension services, since improvement of cocoa stock can be achieved without the need for inputs of expensive technologies or expertise. Field experiments established by that particular ACIAR project will test the efficacy of this approach as well as shed light on some unknown aspects such as the effect of susceptible rootstock on the grafted genotypes selected for their resistance.

Figure 6.1.6 Artificial inoculation of pods using the 'band-aid' method: one drop (0.1 mL) of a suspension containing zoospores, sporangia or a mixture of both is placed on the central absorbent pad of a band aid which is then pressed onto the pod surface. Band-aids or modified tape moistened with distilled water can also be used to hold in place discs of agar containing mycelium or epicarp plugs of infected tissue (Saul 1993).

Conclusion and Recommendations for Future Research

Developing host resistance to *Phytophthora* pathogens is the most pressing need in attempting to achieve control of phytophthora diseases in cocoa. A wide genetic base is fundamentally important for selecting and breeding for disease resistance. Therefore, the promotion of sound conservation strategies for a wide range of cocoa germplasm should be an integral part of dealing with phytophthora diseases. In addition to establishing collections of germplasm, the maintenance of on-farm genetic variability in cocoa, which will enable local and environmentally relevant programs of selection and breeding, needs to be given serious consideration.

The importance of this is illustrated by the lack of success in selecting for disease resistance for swollen shoot virus in West African cocoa, which is largely derived from a few introductions and is genetically uniform Amelonado (Keane 1992).

In contrast, in Indonesia and Papua New Guinea, original introductions of Trinitario cocoa resulted in a high degree of genetic variability following propagation of seedlings. Since the 1960s, introductions of Amelanado and Upper Amazon material, hybridisation between all types of cocoa in mixed plantings and propagation of hybrids, have greatly increased the genetic diversity of cocoa on farms. This has allowed selection and breeding for disease resistance based on observations of resistance in the field, an approach that has been very successful in controlling vascular-streak dieback caused by *Oncobasidium theobromae* throughout the region (Keane 1992) but has yet to be fully exploited to control PPR and stem canker.

In addition to improving host resistance to *Phytophthora* pathogens, integrated disease management strategies are needed that take account of the disease cycles of *Phytophthora* pathogens of cocoa and the wider agrosystem within which cocoa is grown (Smith 1981). Information on the genetic diversity of *Phytophthora*, host–pathogen compatibility and variations in pathogenicity among *Phytophthora* populations between different cocoa-growing regions will be useful for adopting management schemes for cocoa agrosytems. Basic measures such as choosing appropriate shade species (preferably non-hosts of *Phytophthora*), pruning the canopy to improve air circulation and light penetration (which could kill zoospores), soil surface treatments such as mulching and manuring that suppress populations of *Phytophthora* in the soil, regular complete harvesting of both healthy and infected pods to reduce carryover of inoculum sources on the trees, burial of pod cases and infected pods to reduce inoculum at the soil surface, and the use of clean farm implements can all go a long way towards successful management of phytophthora diseases (See Chapter 8.5).

Combining cultural management methods with improved resistance could act to reduce disease synergistically, not just additively. Thus, cultural methods of phytophthora disease control might be quite ineffective on very susceptible cocoa, but show

dramatic results as soon as partially resistant clones are used. As is the case with most *Phytophthora* pathogens in tropical regions, no one control measure can hope to contain phytophthora diseases on cocoa, rather the diseases need to be managed using an integrated approach that aims to minimise losses.

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6.2 Phytophthora Diseases of Coconut in the Philippines

Erlene Concibido-Manohar[1]

Abstract

Coconut is an economically important crop for the Philippines and is the number one export product. Although *Phytophthora palmivora* was known to cause bud rot, and fruit and immature nut fall in the Philippines, the disease losses were relatively low. This changed dramatically after the introduction of highly susceptible MAWA hybrids, which are a cross between Malaya Yellow Dwarf and West African Tall. This chapter provides an overview of the impact of the introduction of this material on coconut production in the Philippines.

Introduction

The coconut (*Cocos nucifera* L.) is a monoecious plant and member the palm family, and is a major earner of foreign exchange for the Philippine economy. The crop provides income directly or indirectly to about one third of the country's population. The coconut industry is considered to be a major pillar of the Philippine economy, supporting 3.4 million farm families directly dependent on coconuts for their livelihood, and a further 24 million individuals who are indirectly dependent on the industry, such as traders, exporters, processors, and their employees. Three hundred million coconut palms spread over 4.09 million ha dominate the landscape of 65 of the 78 provinces in the country. Among the 15 administrative regions of the Philippines, Southern Luzon had the largest area under cultivation (19%) followed by Bicol (16%), Eastern Visayas (15%), and Southern Mindanao (12%). Coconut remains the number one agricultural export product, having generated aggregate foreign exchange earnings of USD768.5m during 1991–2000.

The Philippines was the number one coconut producer in the world during 1976–1986. However, the average productivity has declined in the past decade (1991–2000) with an average production of 669 kg/ha. It lags behind India, which produces, on average, 732 kg/ha, and Indonesia with an average production of 1041 kg/ha. This lower productivity can be attributed to a number of factors, such as slow adoption of recommended cultural management, an increasing number of senile trees, and damage brought about by pest and disease outbreaks. Bud rot and fruit rot were major causes of the large loss of coconut trees and the significant decrease in production.

Bud rot, an apical meristem decay (Reinking 1923) and fruit rot or immature nutfall (Teodoro 1925) are two destructive diseases known to be caused by *Phytophthora palmivora* in coconuts. As well as in the Philippines (Concibido 1990), these diseases were reported to have caused significant coconut yield losses in the Ivory Coast (Quillec et al. 1984) and Indonesia (Bennett, Roboth et al. 1986), in areas planted with the MAWA hybrid. This is a cross between the Malayan Yellow Dwarf and West African Tall varieties, both of which are known to be susceptible to phytophthora.

In the Philippines, bud rot was the first reported disease of coconut and was observed by Reinking in 1919 causing the death of local plantings. The disease never reached epidemic proportions and was known to be prevalent only in the highlands, where the climatic conditions favour disease development. It was only in 1989 that the Philippine Coconut Authority (PCA) became alarmed by the reported outbreak of bud rot that caused the death of over 3000 MAWA hybrid trees in large coconut

[1] Philippine Coconut Authority, Department of Agriculture, Elliptical Road, Diliman, Quezon City, Philippines.

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plantations. These included the 600 ha Ayala Agricultural Development Corporation (AADC) and the 700 ha coconut plots of the Cocoa Investors Inc. (CII) in the southern part of the island of Mindanao in the Philippines. Likewise, at pilot hybrid farms (PHFs) bud rot of the MAWA hybrids was prevalent and regularly monitored by the PCA.

Immature nut fall did not gain attention after Teodoro (1925) gave a detailed description of the disease with the observation that it did not cause significant losses in production. No further reports of the disease were made until 1986, when immature nutfall was reported to be causing significant nut losses in the germplasm collection plots of PCA and in the MAWA PHFs.

It was speculated that the plantings of the MAWA hybrids were one of the main factors that escalated disease incidence in the country, due to its susceptibility to phytophthora infection. It was believed that the genetic uniformity of the nationwide large-scale plantings of MAWA was the major factor that led to the development of disease epidemics between 1989 and 1992. The death of over 1000 palms in the PHFs indicated the potential threat of phytophthora diseases to the coconut industry in the Philippines.

The Disease

Coconut bud rot has been known to be in the Philippines since 1919 when it was reported on the foot slopes of Mt Banahaw on Luzon Island. The disease was considered to be the first serious infectious disease of coconut that causes death of palms. Early epidemics were reported in the highlands of Quezon and Laguna, and sporadic diseased trees were identified in Bukidnon in 1976 (PCA, Crop Protection Guidebook, 1977).

Early studies of the nature and aetiology of bud rot were undertaken by Reinking in 1919. They are considered as the pioneering studies in plant disease which mark the start of plant pathology in the Philippines. Unfortunately, after this initial work, no further studies were conducted due to the sporadic and infrequent incidence of the disease. Information available about the disease and its host–pathogen interaction before the introduction of the MAWA hybrid is therefore rather limited.

The PCA took serious action against the disease only when it was reported to be widespread in the 50,000 ha plantations of MAWA hybrids located on the Ivory Coast. A large-scale replanting program based on the high-yielding MAWA hybrid was under way at the time, and the death of over 1000 palms to bud rot highlighted the potential threat to the coconut industry if the replanting program were to continue. A few cases of the disease were reported on local cultivars but were mostly confined to the highlands, where the climate is humid with a long wet season that is conducive to disease development.

After Teodoro's detailed description of immature nutfall in 1925 (Teodoro 1925), no further cases of the disease were reported. It was only in 1986 that phytophthora-induced nut fall was reported to be prevalent in the germplasm collection plots of the Zamboanga Research Center (PCA–ZRC). It was first observed in the nuts of the Red Cameron Dwarf (RCD) plantings and later in the Malayan Red Dwarf (MRD) and Malayan Yellow Dwarf (MYD) collection plots. Rillo and Paloma (1988) noted that red and yellow pigmented nuts are more susceptible to nutfall than green ones, based on 5-year observations of the disease incidence amongst the various populations planted in the PCA–ARC. Nuts with symptoms of fruit rot or immature nutfall were also found in some PHFs where MAWA had been planted, particularly on Mindanao. To date, there has been no report of fruit rot incidence in local plantings, or in the PCA–ZRC and Davao Research Centers (PCA–DRC) planted with local hybrids.

The Pathogen (*Phytophthora palmivora* Butl.)

Four species of *Phytophthora, P. palmivora, P. arecae, P. katsurae, P. nicotianae,* have been implicated as the causal organisms of the bud rot and fruit rot diseases of coconut (Quillec et al. 1984). Recent studies conducted to elucidate the pathogenic nature of these four species have produced inconclusive results.

Isolation of the oomycete organisms of the genus *Phytophthora* proved to be difficult in the initial studies. Isolations from plants in the advanced stages of bud rot were generally unsuccessful, since infected tissues are prone to contamination with other fungi and bacteria. Only in the early stages of disease development can the pathogen be found at the edges of infected areas or lesions, and sometimes in the centre as mycelium (Quillec et al. 1984). Based on an initial morphological identification of *Phytophthora* isolated from sporulating infected nuts, *P. palmivora* was declared to be involved in immature nutfall. However, it was later reported that several species of *Phytophthora* can attack coconut buds and nuts, and so taxonomic studies were conducted to identify the pathogen based on morphological and molecular characteristics (Chee 1969).

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In the Philippines, *Phytophthora* samples isolated from infected nuts and bud are usually identified as *P. palmivora* (Reinking 1923). This pathogen produces a 'dry' rot before the development of rotting symptoms that are associated with other organisms such as *Fusarium* and *Erwinia* species (Joseph and Radha 1975). It was observed that, while *Phytophthora* is the primary causal agent of the disease, rotting of the bud and subsequent maceration of tissues and foul odour emission are triggered by bacterial infection. At this stage, it is no longer possible to isolate the primary cause of the disease from bud tissues.

It was noted that, in the case of fruit rot, *Phytophthora* species could be isolated from the perianth area and sometimes from the peduncle of the inflorescence. Water-soaked lesions were observed on the epidermal portion of the nut, which becomes brownish at advanced disease stages, and premature senescence results in the nut falling from the bunch. It was claimed that the organism penetrates the soft tissues of the mesocarp where the infection starts (Quillect et al. 1984). The embryo can facilitate the spread of the pathogen from the husk to the meat, through the germinative pore.

Other Hosts

Phytophthora palmivora is known to be the causal organism for many diseases of economically important tropical crops, such as black pod and stem canker of cocoa (*Theobroma cacao* L.), root rot and fruit rot of papaya (*Carica papaya* L.), and foot rot of black pepper (*Piper nigrum* L.). *Phytophthora palmivora* has also been isolated from orchids, durian (*Durio zibethinus)* and rubber. These crops are all grown in the Philippines and perform well in areas suitable for coconut growing. Durian and cocoa are economically important intercrops of coconut, with a coconut–durian mixed cropping system reported to be a profitable agricultural venture in Mindanao. However, it remains to be seen what influence intercropping of susceptible host plants will have on the severity of disease caused by *P. palmivora*. Attempts to establish an integrated disease-management system for phytophthora in a coconut-based farming system are the focus of our current research efforts.

Distribution of Bud Rot in the Philippines

Nationwide bud rot cases

To determine the extent and damage caused by the pathogen nationwide, disease surveys were conducted in the main island groups of Luzon, Visayas and Mindanao in 1992. To obtain sufficient data, two methods were adopted: (i) disease incidence reports from PCA Regional offices were consolidated; and (ii) direct farm visits were undertaken. The highest disease incidence was observed in Mindanao and mostly in areas planted to MAWA (Table 6.2.1). Bud rot incidence in local populations of coconut were reported only in elevated areas such as Mt Banahaw in Luzon, and Camiguin Island in Misamis Oriental. In 1977–78, in an effort to minimise disease spread in infected areas, PCA launched a 'cut and burn' operation on the foot slopes of Mt Banahaw, covering Laguna and Quezon. An estimated 35,000 trees infected with bud rot were felled in 1977 (N. Bondad, Assistant Manager, PCA-Region IV-A, pers. comm.), with similar operations carried out in Camiguin Island in 1985 (J. Lopez, Agriculturist II, PCA-Camiguin, pers. comm.). The yearly data on bud rot cases in pilot PHFs was analysed, revealing a high incidence of the disease in areas of Mindanao where large MAWA plantings occurred (Figure 6.2.1).

Disease assessment

As a result of the data compiled in 1978–1985, disease mapping in the high-incidence Mindanao area was carried out. In the plantings of AADC and CII, bimonthly farm visits and disease monitoring found high levels of infection in the areas planted to MAWA. The highest disease incidence was found in AADC, where 3269 palms (12.6%) succumbed to the disease in a 600 ha MAWA plantation. In CII, the total bud rot cases recorded was 5559, an average disease incidence of 6.3%. Additional data on bud rot cases were collected in PCA research centres, where it was noted that disease incidence in mixed stands which included MAWA was as high as in areas planted to a single susceptible variety of coconut.

It has commonly been observed that bud rot infection of local cultivars is limited to the highlands due to the favourable climatic conditions for pathogen survival and disease development there. However, based on the high incidence of bud rot in MAWA PHFs nationwide, it was inferred that *Phytophthora* could infect the MAWA hybrid in all environmental conditions due to the hybrid's inherent susceptibility. In addition, it is noteworthy that the inherent susceptibility of West African Tall to *Phytophthora* was reported in Ivory Coast (Quillec et al. 1984).

The MAWA experience in the Philippines easily demonstrates the risk of large-scale plantings with a single or a few coconut hybrids where the plants may be inherently susceptible to a pathogen like *Phytophthora.* It is now appreciated in varietal

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improvement programs that cultivars and hybrids recommended for replanting programs should be thoroughly and adequately evaluated in terms of their reaction to *Phytophthora* infection.

Disease symptoms

Visible symptoms

Bud rot of coconut is typically observed soon after a long dry season or after the occurrence of strong winds and typhoons. The first visible symptom of the disease is the drooping of the spear leaf, a symptom that can be easily recognised by a trained and experienced researcher or farmer. Infections initially causes the youngest or spear leaf of the coconut tree to wilt, while advanced stages result in the rotting or disintegration of bud or heart frond tissues, due to secondary infection by bacteria and other opportunistic fungi. As the disease progresses, the spear dries up completely with drooping of the young leaves becoming noticeable (Figure 6.2.1). At this stage, the bud or the coconut heart is already rotted with degenerated tissues and emanates a distinct foul odour. The spear leaf can easily be pulled out but the other leaves are still intact. Existing nuts can continue to develop and mature for 6 months to 1 year, even though the bud has already rotted.

Infection process

Infection by *Phytophthora* can be observed by felling and dissecting a newly infected tree. At this stage, the spear leaf is still green but already starting to wilt with evident loss of turgor. When leaves are sequentially removed, circular, water-soaked lesions can be observed on the smooth portion of the unopened leaf near the base of the youngest leaf frond (Figure 6.2.2).

Figure 6.2.1 Drooping of the spear leaf due to bud rot in MAWA hybrid coconut.

Typical symptoms of fruit rot or immature nutfall are conspicuous irregular patches on the epidermal surface of tissues of immature nuts. These appear as water-soaked lesions, brownish in colour, of varied size and with yellowish margins. The infected nuts can be mistaken for aborted nuts due to premature browning and immature nut fall (Figure 6.2.3). Quillec et al. (1984) observed similar symptoms on MAWA hybrids in the Ivory Coast and Indonesia. When the affected nuts were split open, they exhibited brownish husks and, in severe cases, the meat failed to develop completely. This may be due

Table 6.2.1 Disease distribution of coconut bud rot in 1992 on three major islands of the Philippines.

Main island	Province	Location	Coconut variety	Age group	Total number of palms per farm	No. of cases	Disease incidence (%)
Luzon	Laguna	Liliw	Local	50	650	35	5.4
		San Pablo	Local	35	9,000	300	3.3
		Nagcarlan	Local	35	3,500	500	14.3
		Majayjay	Local	50	500	30	6.0
	Quezon	Dolores	Local	30	24,000	500	3.1
		Lucban	Local	45	2,192	72	3.3
	Batangas	Lipa City	Local	25	300	14	4.7
		Lemery	Local	20	218	38	17.4
		Calaca	Local	20	197	17	8.6
Visayas	Leyte	Baybay	MAWA	15	558	22	3.9
Mindanao	Zamboanga	PCA–ZRC	Various collections	20	6,017	39	0.6
	Bukidnon	Don Carlos	Local	20	193	13	6.7
	Cotabato	Lake Sebu	Local	15	239	79	30.5
	Mis. Oriental	Medina	MAWA		556	29	5.2
		Camiguin Is					

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to colonisation by the pathogen, which is known to produce enzymes that macerate the infected tissues (Akinrefon 1982).

Figure 6.2.2 *Phytophthora palmivora* lesion on the inner leaf sheaths of the bud of a MAWA hybrid coconut

Figure 6.2.3 Nut rot in MAWA hybrid coconut, caused by *Phytophthora palmivora.*

The initial penetration of the nut by the pathogen may occur through the spikelets, since it was noticed that infection usually starts from the perianth portion and progresses towards the apex of the nut. It is evident that the pathogen can establish itself in the husk, shell, meat and water, since it can be isolated from all of these parts.

Pathogenicity

Isolation in pure culture

Several studies were conducted to establish the host–pathogen interaction. *Phytophthora palmivora* was isolated from infected areas using baiting techniques and selective synthetic media. Cultures grown in V-8 juice agar produced mycelia and sporangia. Tissue baiting using cocoa pods also favoured mycelial growth and production of sporangia. *Phytophthora* isolates from both bud rot and fruit rot disease displayed no variation in cultural characteristics.

Pathogenicity studies

Six-month-old coconut seedlings were mechanically inoculated with a pure culture of *P. palmivora* isolated from infected buds. Inoculation resulted in the production of brownish lesions and drooping of young leaves, with white mycelial growth observed on the area of inoculation. *P. palmivora* was re-isolated from the seedlings 20 days after inoculation. The production of symptoms on inoculated seedlings and the re-isolation of the pathogen indicated the pathogenicity of *P. palmivora* on susceptible coconut host tissues, and showed an infection cycle of 8–15 days on seedlings under favourable conditions.

In the case of fruit rot, the 'single drop' technique was employed. A portion of a 6-month old healthy nut was pricked by a sharp pin, a drop of the *P. palmivora* inoculum was placed on the pinpricks and covered with Scotch™ tape to provide a humid environment (Figure 6.2.4). Lesions were seen to develop at the site of inoculation, with an average daily increment of 0.85 cm.

Inoculation of coconut fruit through the spikelets produced symptoms after 2 days. Lesion

Figure 6.2.4 Lesions on coconut infected with *Phytophthora palmivora*.

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development seemed to be faster after the third day. It was noted that 4-month-old nuts are more sensitive to infection than younger or almost mature nuts (Figure 6.2.5). In these studies, production of secondary sporangia resulting from the primary inoculum occurred within 72 hours of the time of inoculation.

Figure 6.2.5 Five-month old coconut artificially infected with *Phytophthora palmivora*.

Cross inoculation test

In order to determine the relationships of the *Phytophthora* isolated from buds and nuts, cross inoculations were performed. Isolates from the bud were used to inoculate the nut, and isolates from the nut used on the bud. Seedlings inoculated with the immature nutfall isolate displayed symptoms 30 days after mechanical inoculation, and nuts inoculated with the bud rot isolates showed symptom development after 5 days. Based on the size of the lesions that developed on the inoculated portion of the nut, the results suggest differences in the degree of specificity of different parts of the host plant are insignificant.

Bud rot observations in the germplasm collection

The Zamboanga Research Center (ZRC) of PCA maintains the largest collection of coconut germplasm in the world, with 83 cultivar collections and 42 hybrids for use in breeding programs and in genetic conservation. The average annual rainfall in this region is 1600 mm, falling predominantly between May and November, followed by a number of distinct dry months. The earliest incidence of bud rot and fruit rot disease in the germplasm plots were observed in 1986 and noted to be prevalent among the dwarf cultivars. The data collected indicate the greater susceptibility of the dwarf cultivars to nut fall and bud rot diseases, particularly the MRD and the MYD varieties, when compared to the talls and the local hybrids (Table 6.2.2). Interestingly, the incidence of bud rot in MAWA plots was negligible during the observation period. This observation can be attributed to the heterogeneity of the populations planted in ZRC, which limits the continual spread of the disease, and to the environmental factors (warm temperature, high relative humidity and soil moisture, and the absence of typhoons and strong winds) that can trigger infection development and pathogen dissemination. Appropriate cultural management and immediate 'cut and burn' of infected trees in the collection plots was conjectured to prevent disease spread and minimise disease incidence on the MAWA plots.

The first cases of fruit rot were observed on the Red Cameron Dwarfs in 1986, while the MRD and MYD populations were found to be infected later. Fruit rot was observed to be severe, with about 5% of the total nuts succumbing to infection (Table 6.2.3). Emasculated palms showed especially high levels of rot, which could be attributed to contaminated cutting tools having been used. The emasculation activity was temporarily stopped and routine, 6-monthly treatments of Ridomil by root infusion (20 mL of 1.6 g a.i./tree) were undertaken. Monitoring has shown a reduction in disease incidence since that time.

Varietal Nut Reaction to Artificial Inoculation

Two varieties/cultivars, Malayan Red Dwarf (RMD) and Malayan Yellow Dwarf (MYD), and the locally developed hybrid PCA 15-1 (Catigan × Bago-Oshiro Tall), were tested for reaction to *P. palmivora* through mechanical inoculation using the 'single drop' technique. The results showed that MRD was subject to the most rapid increments in lesion size, while PCA 15-1 had the slowest. The reaction of MYD was not significantly different from MRD. Cross-variety inoculations were trialled, using detached infected nuts from one variety as a source of inoculum with which to inoculate healthy nuts from a second variety. This technique allowed us to identify a source of inoculum that produced the most consistent pathogenic results under field conditions, and can be used to test the susceptibility and resistance of potential parent material in breeding programs. The degree of resistance or susceptibility of the infected nuts was assessed as lesion expansion over time. The increase in size and depth of the lesion were measured daily using calipers. Infected MAWA nuts used as source of inoculum to inoculate healthy MRD nuts produced symptoms similar to those observed in the field.

The initial results of the varietal nut reaction could be used in evaluation studies to determine the

performance of promising hybrids in terms of disease reaction. This study itself has already provided information on promising parental materials for hybridisation programs and in determining sources of resistance. The significant resistance to infection displayed by the local hybrids, which were produced from local dwarf and local tall cultivars, indicates that the local tall parent cultivars could be sources of parental genes with possible inherent resistance to *Phytophthora* infection.

As reflected in Table 6.2.3, significant differences in reaction to the disease were found among dwarf and tall cultivars. When artificially inoculated, the red and yellow-pigmented cultivars were found to be highly susceptible when compared to the green-pigmented cultivars, and in particular when compared to the local populations, thus confirming field observations. The results of the inoculation tests show that sources of resistance to *Phytophthora* infection can be determined, which is vital in the process of selecting promising cultivars for replanting programs, and in the formulation of control strategies to contain the disease.

Recommendations

- Collaborative efforts among breeders and pathologists are needed in breeding programs to look beyond improving agronomic characters of the hybrids to be developed while at the same time also including resistance to major diseases.
- Comprehensive assessment of recommended cultivars and hybrids for distribution and replanting is imperative to assure disease-free or disease-resistant planting materials.
- To minimise losses from the disease, areas identified as having high inoculum levels of *Phytophthora* should be avoided in planting

Table 6.2.2 Bud rot cases at the germplasm collection at the Zamboanga Research Center of the Philippine Coconut Authority.

Population	Number of palms	Bud rot incidence 1991		Bud rot incidence 1992	
		No.	%	No.	%
CAT × LAG hybrid	190	1	0.6	0	0.0
CRD × WAT hybrid	130	1	0.8	0	0.0
CAT × BAY hybrid	168	0	0.0	1	0.6
MYD × WAT hybrid	401	3	0.7	1	0.2
MRD × TAG hybrid	22	0	0.0	3	13.6
MAT × MYD hybrid	150	0	0.0	1	0.7
BAO × CRD hybrid	90	1	1.1	0	0.0
BAY × CRD hybrid	90	1	1.1	0	0.0
RNL × GDS hybrid	120	1	0.8	0	0.0
TAG × WAT hybrid	120	0	0.0	1	0.8
TAG × RCD hybrid	60	0	0.0	1	1.7
Aromatic dwarf	137	0	0.0	1	0.7
Catigan dwarf	1115	0	0.0	3	0.3
Banigan	96	0	0.0	1	1.0
Galas	110	0	0.0	1	0.9
RNL-A tall	565	1	0.2	0	0.0
Magtuod dwarf	134	1	0.7	2	1.5
MRD dwarf	488	0	0.0	4	0.8
MYD dwarf	1557	0	0.0	3	0.2
Macapuno	96	3	3.1	0	0.0
Agta tall	84	2	2.4	0	0.0
SNR tall	134	0	0.0	1	0.7

Table 6.2.3 Fruit rot incidences at the germplasm collection in PCA–ZRC (1991–1992).

Population	Number of palms	Number of palms infected	Number of bunches infected	Number of nuts infected
MRD	488	26 (5.3%)	42	301
MYD	1557	7 (0.5%)	15	88
Buswang	90	1 (1.1%)	4	34

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susceptible coconut cultivars and intercrops known to be infected by the pathogen.
- Planting of homogeneous varieties/populations in environments that may favour disease development should be discouraged to avoid disease epidemics.
- Adoption of proper cultural management and proper disposal of infected palms and plant parts is essential to eliminate possible sources of pathogen and control the spread of disease.

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6.3 Distribution and Progression of Phytophthora Bud Rot Disease of Coconut in Selected Areas in the Philippines

Nemesia San Juan-Bachiller[1]

Abstract

Geographical distribution of *Phytophthora* bud rot on coconut in the Philippines was determined from 1990 to 1999 through a survey in areas with reported incidence of the disease. Records of the disease reached to 4.1%. Over 11,000 palms were killed by *P. palmivora*, with the three provinces of Davao (Davao del Norte, Davao del Sur and Davao City) having the highest incidence. It was found that the disease infected several coconut cultivars all over the country. The Malayan Yellow Dwarf × West African Tall (MYD × WAT) hybrid (known as MAWA) introduced into the country was the most susceptible, with an incidence rate of 2.7%. Most of the affected palms were 3–15 years old with MYD or Malaysian Red Dwarf (MYD) parentage. Studies on the spatial and temporal distribution of the disease showed that it occurred in multiple foci that were distributed throughout the experimental area. It had both the regular and contagious or cluster-distribution pattern. The disease progress curves suggest that bud rot follows a continuous 'compound interest' model. It follows that the progress of the disease at any given time is a function of the initial inoculum and the number of effective contact points between a susceptible host and inoculum per unit time. Analysis of the infection rates using logistic growth model in three observation sites gave rates of 0.065, 0.074 and 0.157 per unit per year in MYD × WAT, Laguna Tall and MYD × Hijo Green Tall (HGT), respectively. Regardless of genotype, infection rate was established at 0.228 per unit per year.

Introduction

The occurrence of bud rot disease of coconut in the Philippine provinces of Laguna and Quezon was first reported by Copeland (1908). A decade later, Reinking (1919) identified *Phytophthora faveri* Maubl. (also *P. palmivora* Butler) as the causal organism of coconut bud rot, following an extensive study of its morphology, including growth in various media, mycelium, conidiophores, conidia, chlamydospores and absence of sexual bodies. Bud rot is characterised by the wilting of the spear leaf due to the rotting of the bud (Figure 6.3.1). The fungus has infected thousands of coconut palms since it was first identified in the Philippines.

[1] Agricultural Research Branch, Philippine Coconut Authority, Davao Research Center, Bago-Oshiro, Davao City 8000, Philippines.

However, a thorough investigation of its the mode of spread, rate of infection and geographical distribution was made only in 1989 to 1998, led by the Crop Protection Division, Davao Research Center, Philippine Coconut Authority (PCA). The information generated about behaviour of the disease relative to cultivar, age of the palm, location and climatic conditions prevailing in the growing area is vital in the management of the disease.

Methodology

Disease distribution and assessment of bud rot incidence

Disease surveys and mapping were conducted on coconut farms in the Philippines, in the provinces of Laguna, Quezon, Batangas (Luzon), Leyte and Samar (Visayas), Zamboanga, Bukidnon, Misamis Oriental,

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South Cotabato, Camiguin Island, Davao del Sur, Davao City, and Davao del Norte (Mindanao). This survey was conducted in collaboration with extension staff of the Field Operation Branch of PCA. Before the survey, a training course on disease identification and basic control methods was given to the Coconut Development Officers and several farm leaders nationwide. Data on the incidence of bud rot cases were reviewed and consolidated annually from 1990 to 1999.

Figure 6.3.1 (Upper left) The initial symptoms of bud rot: early wilting of the spear leaf. (Upper right) Abnormal hanging and desiccation of the spear leaf, another bud rot symptom. (Lower left) Rotted tissue shows as purple to pale pink, and has the consistency of soft cheese. (Lower right) A dissected bud showing internal rotting of the tissue. The rotten tissue emits the odour of putrefaction.

Data on bud rot incidence were collected from different experimental plots planted with various coconut hybrids/cultivars at the PCA's Davao Research Center at Bago-Oshiro, Davao City and the Zamboanga Research Center, as well as at different multi-location sites of the Breeding and Genetic Division for at least 5 years.

The following data were gathered in each area surveyed:

- cultivar/hybrid
- palm age
- number of palms
- number of infected palms
- percent of disease incidence computed as the number of infected palms
- disease incidence per cultivar.

Disease mapping of bud rot

Actual mapping of disease spread was done in at least 10 ha per planting area, with approximately 1000 coconut palms and at least 10% disease incidence. These were established in Payahan, Camiguin Province, Ayala Agricultural Development Corporation, Darong, Davao del Sur and Conception Farm in La Filipina, Davao del Norte. With the use of farm maps indicating the distribution of coconut palms, the exact location and number of diseased palms were recorded.

Data on disease incidence were collected every 3 months. In addition, rainfall data within the period of observation were gathered. The increase in disease incidence was expressed as the number of infected palms divided by the total number of palms, calculated annually in each experimental area.

Disease plant density distribution analyses

To determine the spatial pattern of bud rot distribution in the three experimental sites, a local density distributions diseased plants were compared with expected random distribution. The mean (x) and variance (s^2) of the diseased palms to the total local population at each site were taken and the goodness of fit was tested using the chi square (χ^2) distribution parameter. In events where variance is equal to the mean, the population is said to be randomly distributed. If the variance is less than the mean, then the distribution is regular. Computations were made following the formula of Gomez and Gomez (1984).

Disease progress curve

Disease progress curves for each experimental area or cultivar were determined by plotting the disease proportion against time, using the data gathered from 1990 to 1999.

Infection rate

Infection rate per site per cultivar was calculated using the same data as for the disease progress curve. Infection rates were estimated from the logistic growth model described by Van Der Plank (1963), using the differential equation:

$$dY/dt = rYt\ (1 - Yt)$$

where the change in proportion of disease Y, with time t, is equal to the rate of infection r, multiplied by the proportion of the disease at any given time and then multiplied by a correction fact or $(1 - y)$. Disease

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proportion was transformed according to the disease growth model and regressed with time. The infection rate, which was the slope of the line, was determined.

Results and Discussion

Geographical distribution and assessment of bud rot incidence

Geographical distribution

Figure 6.3.2 shows that bud rot disease is widely distributed throughout the country where coconut is grown. In Luzon, Laguna, Quezon and Batangas, the number of bud rot infected palms was 893, 654 and 69, respectively, during the 10 years of observation. Areas surveyed in these provinces are situated at high elevations where low temperature and high moisture favour disease development. In Visayas, where only the province of Leyte was visited, 22 infected palms were found in a 5 ha coconut farm. Disease severity was highest in Mindanao. Several provinces were affected but the incidence was highest in the three provinces of Davao, with Davao del Sur having 5224 bud rot infected coconut palms, Davao del Norte 1749, and Davao City 1163.

Figure 6.3.2 Locations in the Philippines where bud rot disease of coconuts was found.

Considering the country as a whole, Mindanao had by far the largest proportion of total disease incidence, 85.2%. Areas in Luzon areas had 14.6% of the total percentage disease incidence, while Visayas had only 0.2%. Average disease incidence across the country reached 4.1%, which translates to 11,130 palms killed in our experimental survey plots over the 10 years of observations.

Assessment of bud rot incidence by cultivar/ hybrid

Among the dwarf cultivars, the highest disease incidence was observed in Malaysian Red Dwarf (MRD) (13.7%). Among the tall cultivars, Laguna Tall (LAGT) had the highest incidence (5.6%) followed by Hijo Green Tall (HGT) (5.2%).

Among the hybrids planted in different places in the country, disease incidence was relatively higher in Malayan Yellow Dwarf × West African Tall (MAWA) hybrid (4.4%) plantings than in the local cultivars (Table 6.3.1). It should be noted that almost all areas surveyed with LAGT plantings were located in the highlands, where relative humidity is high, a critical factor that predisposes coconut palms to pathogen infection. The MAWA hybrids, on the other hand, have been used for massive planting both in high and low-lying areas of the country. It was also observed that hybrids with MRD or Malayan Yellow Dwarf (MYD) as one of the parent materials had a higher disease incidence than the other hybrids. This observation is supported by the data gathered in the multi-location trial sites of PCA. Catigan, a local dwarf cultivar, was observed to be quite tolerant to the disease.

Assessment of bud rot disease incidence by age group

Table 6.3.2 shows the effect of coconut age on the incidence of bud rot. Generally, the incidence of the disease falls in mid-aged palms, but then rises again in older trees. Coconut palms ranging in age from 3–10 years were more susceptible to bud rot with disease incidence of 4.3% or total disease occurrence of 4982 bud rot cases. This was followed with palms ranging in age from 11–15 years (2569) or 3.9% disease incidence. Coconut palms ranging in age from 41–50 years had the highest incidence (657) of bud rot infection. This trend might have something to do with the physiology of the coconut bud as it matures. As Mackenzie et al. (1983) indicated, the phenomenon of adult plant resistance may in some cases be explained by age-specific changes of the plant.

Mapping of bud rot incidence

Among the three areas, the AADC coconut plantation at Darong Davao del Sur, where MAWA was planted, had the highest bud rot occurrence with 0.37, followed by La Filipina, planted with MYD × HGT with 0.24. LAGT planted in Camiguin province had the least incidence at 0.13 (Table 6.3.3). Once again, MAWA and hybrids with MYD parentage show a significant degree of susceptibility to *Phytophthora* infection.

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Table 6.3.1 Disease distribution of bud rot by coconut cultivar/hybrid.

Genotype	Age group (year)	Total number of palms	Disease incidence	
			Number	%
Dwarf				
Catigan	20	971	1	0.1
Malaysian Red Dwarf (MRD)	14	110	15	13.7
	Total	1,081	16	1.5
Tall				
Baybay	12	288	7	2.4
Hijo Green Tall	12	96	5	5.2
Laguna Tall	20– 30	57,623	3,227	5.6
Tagnanan Tall	12– 18	33	271	0.8
	Total	91,703	3,510	3.8
Hybrid	12	96	2	2.1
CAM × BAY	20	53	3	5.7
CAT × BAO	20	70	2	2.9
GDH × WAT	12	384	8	2.1
MRD × BAY	12	384	16	4.2
MRD × HGT	12	576	16	2.8
MRD × RIT	12	480	12	2.5
MRD × TAG	12	4,500	60	1.3
MYD × HGT	12	192	5	2.6
MYD × RIT	10–20	168,429	7,357	4.4
MYD × WAT	12	73	1	1.4
NRC × WAT	12	96	7	7.3
PGD × LUP	12	96	7	7.3
TAC × BAO	12	96	2	2.1
WAT × RIT	Total	175,525	7,498	4.3

Table 6.3.2 Disease distribution of bud rot by age group.

Age group	Number of palms	Disease incidence	
		Number	%
03 – 10	115,757	4,982	4.3
11 – 15	65,920	2,569	3.9
16 – 20	39,354	673	1.7
21 – 30	30,929	1,253	4.1
31 – 35	13,924	996	7.2
41 – 50	8,342	657	7.9
Total	274,326	11,130	4.1

Table 6.3.3 Cumulative disease proportion of bud rot disease.

Location	Genotype	Number of palms	Disease incidence							
			1990	1991	1992	1993	1994	1995	1996	1999
AADC, Darong, Davao del Sur	MYD × WAT	1144	0.13	0.14	0.17	0.17	0.20	0.23	0.24	0.37
La Filipina, Tagum, Davao del Norte	MYD × Hijo Tall	911	0.05	0.08	0.08	0.08	0.10	0.10	0.11	0.24
Payahan, Camiguin Island	Laguna Tall	1025	0.07	0.08	0.09	0.09	0.10	0.12	0.13	0.13

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Diseased plant density distribution analysis

The spatial distribution of bud rot in the different experimental areas over a 9-year observation period is presented in Figures 6.3.3–6.3.5. Initial descriptive patterns of the disease indicate that it is distributed throughout the entire plots and has multiple foci. The randomness of the disease distribution was analysed using the theoretical binomial distribution under the random distribution analysis. Table 6.3.4 shows that variances of the majority of the areas throughout the observation period are greater than the means, an indication that the disease distribution is continuous or clustered.

Multiple foci were observed and the disease progressed from one infected palm to the next. This observation follows that of Steer and Coastes-Beckford (1990). Mackenzie et al. (1983) also reported that dispersal mechanisms of the

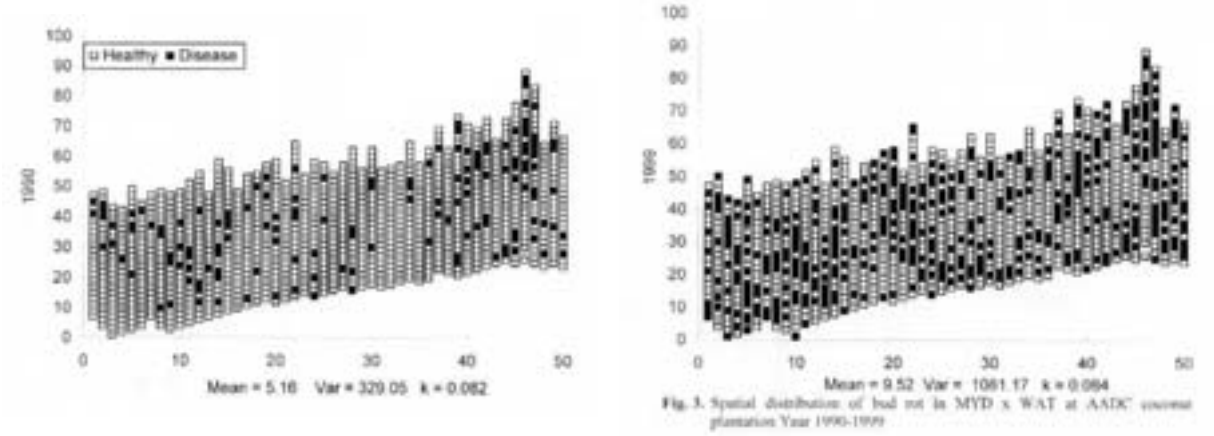

Figure 6.3.3 Spatial distribution of bud rot in MAWA hybrid at AADC coconut plantation year 1990–1999.

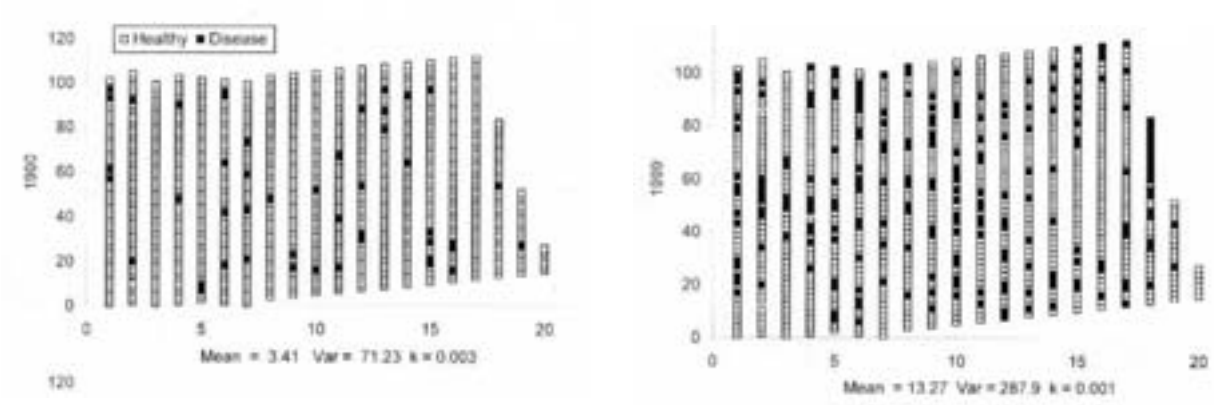

Figure 6.3.4 Spatial distribution of bud rot in MYD × Hijo Green Tall at La Filipina coconut plantation year 1990–1999.

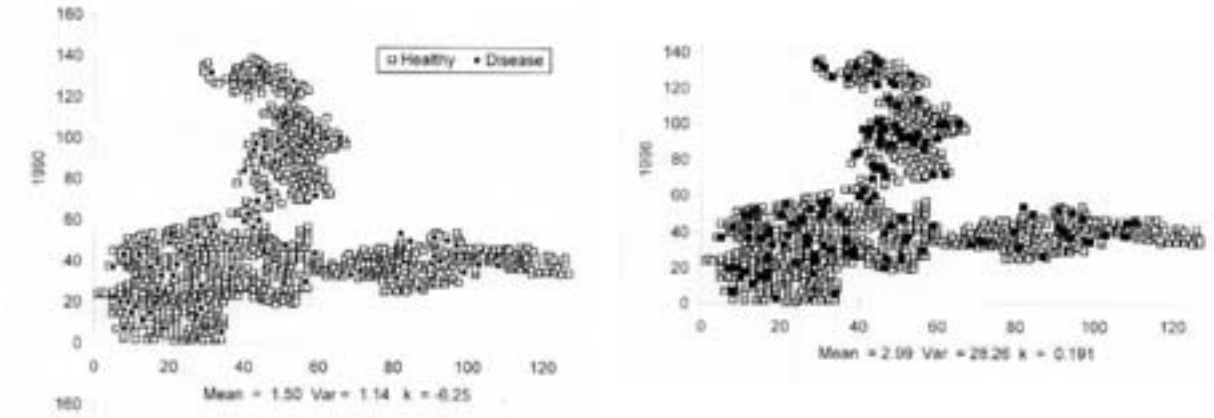

Figure 6.3. 5 Spatial distribution of bud rot in Laguna Tall coconut palms at Camiguin Island. 1990-1999.

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inoculum of *Phytophthora* spp. are closely or directly related to water. Rain splash, among other water movements, may account for local dispersal within canopies of the palm, moving the infectious spores between different palm trees and different fields and areas.

Disease progress curve

Progress of bud rot disease in the three sites during the 9-year observation period was determined by plotting the disease incidence over time. Figure 6.3.6 shows a disease progress curve that appears to follow the compounded continuous interest (CCI) type described by Van Der Plank (1963). CCI curves or epidemics, according to Van der Plank, have the potential for exponential explosion, sometimes resulting in catastrophic disease.

Infection rates

Apparent infection rates (represented by *r* values) are estimates of how fast an epidemic progresses over time when adjusted for multiple infections. They are calculated as linear regression coefficients of the logit-transformed disease proportions (Mackenzie et al. 1983). Table 6.3.5 and Figure 6.3.7 show the apparent infection rates of bud rot in the three experimental areas planted with different cultivars ranged from 0.157 to 0.065 per unit per year. The area with the highest apparent infection rate of 0.157 per unit per year is the MYD × HGT plantations at La Filipina. According to Mackenzie et al. (1983), cultivars differ in their apparent infection rates, which may be due to different levels of horizontal resistance. Regardless of genotype and area, infection rate is 0.228 per unit per year.

Table 6.3.4 Analysis of bud rot disease distribution in coconut palms at three experimental sites in the Philippines.

Area	Total population	Year	Disease incidence (no. of cases)	Mean (X)	Variance (S^2)	Aggregation index (K)
AADC	1144	1990	144	5.17	329.05	0.0824
		1991	164	5.50	438.80	0.0698
		1992	192	5.65	413.38	0.0770
		1993	197	5.87	442.96	0.0789
		1994	230	6.75	711.88	0.0647
		1995	266	7.06	706.71	0.0713
		1996	274	7.14	708.65	0.0260
		1997–99	426	9.82	1081.17	0.0845
La Filipina	911	1990	46	3.41	71.23	0.0031
		1991	69	5.09	195.59	0.0017
		1992	72	5.22	196.51	0.0017
		1993	76	5.29	194.91	0.0017
		1994	87	5.73	212.11	0.0016
		1995	95	5.92	256.78	0.0013
		1996	98	5.95	259.69	0.0012
		1997–99	222	13.27	287.95	0.0010
Camiguin	1025	1990	75	1.50	1.14	–6.2500
		1991	86	1.69	8.30	0.4358
		1992	88	1.68	8.05	0.4437
		1993–94	97	1.87	15.15	0.2636
		1995	122	1.99	19.61	0.2252
		1996	123	2.23	28.26	0.1905

Table 6.3.5 Simple linear regression analysis, using a logistic model, of progress of bud rot disease in three different locations in the Philippines.

Location	Genotype	Intercept	Apparent infection rate (r)	R-squared
AADC, Darong, Davao Del Sur	MYD x WAT	–301.900	0.065	0.982
La Filipina, Davao Norte	MYD x Hijo Tall	–377.900	0.157	0.925
Camiguin Island	Laguna Tall	–220.300	0.074	0.925
	Average	–420.600	0.228	0.894

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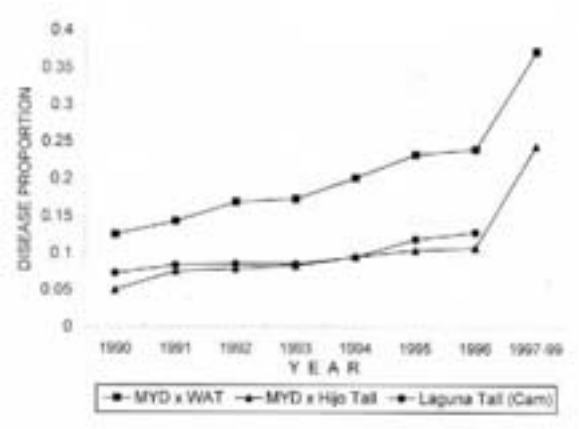

Figure 6.3.6 Disease progress curve of bud rot of coconut in AADC, La Filipina Plantation in Davao Sur and Norte and Camiguin Island 1990–1999.

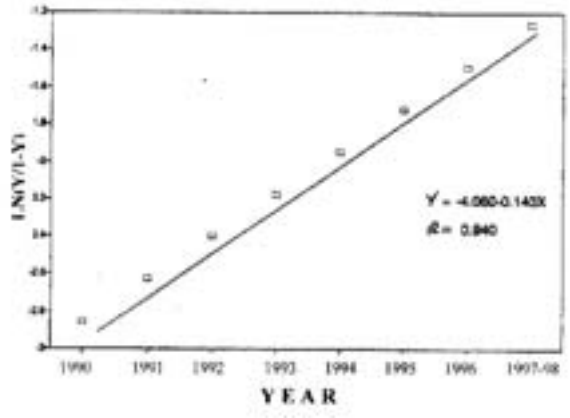

Figure 6.3.7 Regression line showing the relationship of transformed disease progress to time.

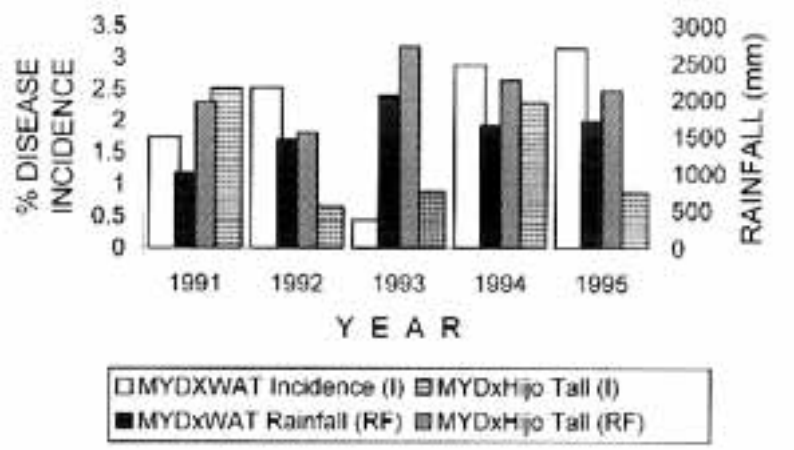

Figure 6.3.8 Incidence of bud rot in MYD × WAT and MYD × Hijo Tall coconut palm hybrids.

Effect of rainfall on bud rot incidence

Rainfall increased the incidence of disease in the MYD × HJT hybrid in La Filipina but not with the MAWA hybrid in AADC. This might be related to high relative humidity in the area.

Infection by *P. palmivora* on coconut occurs when relative humidity is higher than 94% and the temperature is below 24°C. It might be inferred from the inverse relationship of bud rot incidence to rainfall in MAWA plantation at AADC (Figure 6.3.8) that disease development in this area is not largely dependent on climatic conditions, particularly rainfall, but rather on the susceptibility of the MAWA hybrid.

Conclusions and Recommendations

Bud rot is indeed a major fatal disease of coconut palms in the Philippines. It is widely distributed, has the ability to infect several, if not all coconut genotypes, and most important of all, it has the potential for exponential growth, an occurrence that would will be catastrophic to the coconut industry.

The establishment of the apparent susceptibility of hybrids with MYD and MRD as parent materials is important information and such materials are to be avoided by breeders in breeding *Phytophthora*-resistant cultivars/hybrids.

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Establishing the pattern of disease spread is vital in framing recommendations for preventive control measures. Based on the results of this study, it is recommended that preventive measures such as sanitation (cutting and burning of affected palms) and fungicide application be applied to neighbouring palms in infected areas to prevent further spread. Monocropping with highly susceptible cultures such as MAWA is to be discouraged. Also, genetically uniform planting leads to continuity of spread of the disease, leading to outbreaks.

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6.4 *Phytophthora capsici* on Black Pepper in Indonesia

D. Manohara,[1] K. Mulya,[2] A. Purwantara[3] and D. Wahyuno[1]

Abstract

Foot rot of black pepper (*Piper nigrum* L.) is an important constraint to production of pepper in Indonesia and many other parts of Southeast Asia where pepper is grown. Cultivation practices and the intensity of management is dependent on the highly variable price of pepper. This chapter summarises the symptoms of the disease and describes its epidemiology, and provides an outline of the options for disease control.

Introduction

Phytophthora capsici Leonian causes the most destructive and economically significant disease of black pepper (*Piper nigrum* L.). *P. capsici* attacks all parts and growth stages of the black pepper plant. The disease, which was first reported in Lampung in 1885, has been called foot rot disease since 1928 (Muller 1936). The causal agent was first identified as *P. palmivora* var. *piperis* (Muller 1936), and later determined as *P. palmivora* MF4 (Tsao et al. 1985). Later still, it was renamed *P. capsici sensu lato* (Tsao and Alizadeh 1988). The disease is now found in almost all pepper grown in Indonesia.

Pepper (black and white) is the seventh largest export income earner for Indonesia. The total area under pepper cultivation is about 136,450 ha, and the activity involves over 130,000 farmers. Smallholders conduct almost all pepper cultivation in Indonesia. They have limited access to capital, and fully manage their cultivations only whenever the pepper price is high, abandoning them if the price falls. They usually use systemic fungicides to control foot rot disease, to which all cultivated pepper varieties grown in Indonesia are susceptible.

[1] Research Institute for Spice and Medicinal Crops, Bogor 16111, Indonesia.
[2] Research Institute for Agricultural Genetic Resources and Biotechnology, Bogor 16111, Indonesia.
[3] Biotechnology Research Unit for Estate Crops, Bogor 16151, Indonesia.

Lampung and Bangka are the main black pepper producing areas. Foot rot disease destroyed the pepper area in Lampung before the second world war, while in Bangka, the disease damaged about 32% of pepper plants in 1965. The other pepper areas are in West, Central and East Kalimantan.

We collected 168 *Phytophthora* isolates causing foot rot. The resulting population of *P. capsici* consisted of 148 A1 mating type isolates and 20 A2 mating type isolates. Both mating types were found in Lampung and Kalimantan, while in Bangka only the A1 mating type was found. Among those isolates, 43 were morphologically and physiologically characterised. The results showed that all isolates were *P. capsici* except one, which was identified as *P. nicotianae* (Manohara and Sato 1992).

Disease Symptoms

The first symptom of foot rot is a slight wilt of the vine. The leaves become pale and the vines droop (Figure 6.4.1). At this point, the leaves may fall prematurely, puckering along the edges and becoming yellow before they fall. Occasionally, necrosis is observed at either end of the leaf. After defoliation, the fruit begins to wrinkle and dry out. The flower spikes and lateral stems become necrotic and break off at the nodes. The post holding the vine is left bare of all but the three climbing stems. The decline of the vine is rapid, 75% of the leaves may fall within 7–14 days of the first signs of wilt. The wilting is caused by the destruction of the underground parts of the main stem, although the

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root, collar, leaves, flower spikes and fruits are also susceptible to attack. Complete destruction of the main lateral roots and girdling of the stem at the crown cause the wilt. In some cases, collar rot may occur rapidly at the base of the plant, so there is no time for the leaves to abscise and drop. This, so called sudden-death, leads to dead plants with all the leaves still attached. Infected leaves are found on the lower foliage close to the mound below the vine. Necrotic lesions are observed on the leaves. These may be circular and deep brown in colour, with a distinct fimbriate edge. Fimbriate lesions are diagnostic of foot rot. They tend to occur on younger leaves; the fimbriate edge becomes less distinct when the infection becomes less active in drier weather. Concentric rings may appear around the lesions after continued wet weather. Stems can also become infected, showing water-soaked patches. The vine may become locally defoliated near the site of stem infection. Dieback of the stem can occur as the infection progresses along the vine. It is more difficult to isolate *P. capsici* from infected roots and stems than from leaf lesions. Below-ground symptoms are sometimes detectable at the first sign of wilt. Vines older than 3 years seem to be the most susceptible to foot rot (Holliday and Mowat 1963; Erwin and Ribeiro 1996. The A1 types isolated from Lampung and East Kalimantan are more pathogenic than A2 type. Conversely, the A1 type isolated from West Kalimantan is less pathogenic than A2 type.

Figure 6.4.1 Foot rot in pepper, caused by *Phytophthora capsici* and giving rise to pale leaves and drooping of the vine (plant on the right).

Disease Epidemiology

The principal source of inoculum of *P. capsici* is infected plant debris. Leaves are infected by inoculum splashed up from the soil. The severity of foot rot increases during periods of rainfall in the monsoon season, and when day and night temperatures vary between 19 and 23°C (Erwin and Ribeiro 1996). Other predisposing factors include planting pepper in soils that are low in organic matter and nutrients such as calcium, magnesium, and potassium, but high in nitrogen (Nambiar et al. 1965). Vectors such as termites and slugs can transport inoculum within and between vines (Erwin and Ribeiro 1996).

Soil moisture is one of the most important environmental factors for the survival of *Phytophthora*. Propagules of *P. capsici* (isolated from Lampung) survived for more than 20 weeks in Latosol soil at 100% field capacity. The fungus survived as a saprophytic stage on pepper leaves for 11 weeks in soil at 60–100% field capacity, while on the stem survival time fell to 8 weeks (Manohara 1988).

P. capsici infects leaves close to the soil surface, usually after heavy rain at the start of the wet season. Penetration by zoospores occurs 4–6 hours after interaction. There are two methods of infection: direct penetration through epidermis, and indirect penetration through stomata. Brown–black minute spots appear 18 hours after infection (Manohara and Machmud 1986). Collar infection causes sudden wilting, the leaves turn brown–black and dry while they are still attached to the plant.

Disease Control

The first step in preventing the disease is to plant on well-drained sites not planted to black pepper for at least a year beforehand. Removal of diseased vines, followed by application of a copper-based fungicide around the diseased roots to prevent spread to other vines is highly recommended. Bordeaux mixture has been reported to be effective, as have metalaxyl and fosetyl-A1 when applied to the foliage (Erwin and Ribeiro 1996). There is limited resistance to foot rot in *P. nigrum* and other species of *Piper* (Sitepu 1993), but some success has been achieved in using disease-tolerant species as rootstocks for current cultivars (Manohara et al. 1991). Application of metalaxyl as a root soil drench has been used to control root and stem root in black pepper (Erwin and Ribeiro 1996). Application of fungicides is recommended at the beginning of the wet season, with follow-up sprays at 7–10-day intervals (Sitepu 1993). Another successful disease-control method developed in Sarawak uses root infusion of phosphorous acid, as described in chapter 7.4. There are some cultural practices that can minimise the impact of foot rot

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disease. These include weeding around the bases of the vines to discourage the build-up of moisture that can encourage the proliferation of inoculum, and pruning the lower canopy to prevent it from coming into contact with soil-borne inoculum. However, clean weeding usually done by farmers may in many cases cause faster disease spread than limited weeding. Improving soil drainage also discourages disease development. In areas where *P.capsici* is endemic, rows of black pepper should be alternately planted with a perennial crop that is resistant to foot rot. The application of organic waste matter (such as trash from maize, rice, mungbean, peanut or soybean crops) to the soil can encourage the development of microorganisms that are antagonistic to *P. capsici.* The eradication and burning of infected vines is also highly recommended (Sitepu 1993).

An integrated approach is needed to control foot rot in pepper. This will include introduction of adequate drainage systems, limited weeding, fertilising of the pepper plants at recommended dosages and times, pruning the lower branches of pepper plant, especially during rainy season to reduce humidity at the collar and prevent the lower leaves coming into contact with soils that might be infected by *P. capsici*, and the use of phytophthora-tolerant varieties. The use of tolerant varieties such as Natar 1 is recommended when farmers want to expand their plantings. Planting cover crops such as *Arachis pintoii* among pepper plants is believed to be better than clean weeding, as *A. pintoii* inhibits the dissemination of *P. capsici.* During the rainy season, it prevents splashing, onto the lower leaves, of soil particles that may be contaminated with *P. capsici*. Inorganic fertiliser (NPK) that contains more potash than nitrogen has also been reported as reducing *P. capsici* infection (Zaubin et al. 1995).

The amendment of organic matter such as rice straw, and maize, soybean, peanut and mungbean waste, reduced the disease intensity by about 20–50% (Kasim 1985). Root exudates of *Allium fistulosum, A. ascalonicum, A. shoenorapsum* and *A. sativum* have also been reported to inhibit zoospore germination. The rhizospheres of *Allium* spp. are suitable for the growth of some microbial antagonists such as *Trichoderma* spp. and fluorescent bacteria, and the planting of these species around pepper plants is therefore recommended (Manohara et al. 1994).

Trichoderma harzianum Rifai (BLT 1), in the form of substrate or a pelletised formulation, has shown good potential for control foot rot disease. Incorporating it with some organic materials has been shown to reduce the severity of foot rot disease by up to 50% in greenhouse tests (Manohara and dan Wahyuno 1995).

Future Research

Introducing resistant varieties is an effective and economic way to control foot rot disease on black pepper. Even though black pepper is a perennial crop, it is commonly propagated vegetatively. Therefore, breeding programs for resistance can be accelerated through the rapid multiplication of resistant hybrid clones. Conventional and somatic hybridisation could be adopted in the production of such hybrids.

Currently, the management of foot rot in pepper is conducted without much knowledge of the population biology of the causal organism. Different mating types occur in the pathogen populations, and differences in the pathogenicity between isolates within and between may exist. Therefore, analysis of the structure of pathogen population should be initiated in parallel with screening for sources of resistance. Sources of genetic resistance have been identified in wild black pepper species such as *Piper hirsutum, P. aurifolium* and *P. cubeba* (Kasim 1981). Some varieties of black pepper showed tolerance to *P. capsici* infection. These included Natar I, Bangka, Pulau Laut, Merapin and Banjarmasin Daun Lebar. In order to select a number of competent strains, representing the diversity of pathogen populations in the field for use in selection for disease resistance, more research is needed to characterise the pathogen populations and gain further insight into the nature of the host–pathogen interaction.

Disease resistance alone has as yet not been able to halt the serious economic impact of foot rot disease in pepper. To control the disease, resistance therefore needs to be combined with other management practices in an integrated approach.

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6.5 Phytophthora Diseases of Rubber

Ratana Sdoodee[1]

Abstract

Rubber is affected by a group of phytophthora diseases including pod rot, leaf fall, black stripe of the tapping panel, and stem or patch canker. Black stripe disease was the first noted in Sri Lanka and is widespread in Southeast Asia as well as Africa and America. Other phytophthora diseases are also common throughout most rubber-growing areas. Black stripe and leaf fall cause serious damage but economically important outbreaks are confined to areas with long periods of high rainfall. Although patch or stem canker is widespread, recent records of high economic impact are few. At least six species of *Phytophthora* have been reported to be associated with diseases of rubber. The most common species are *Phytophthora palmivora* (Butl.) Butl., *P. meadii* McRae and *P. botryosa* Chee.

Introduction

In the late nineteenth century, rubber was introduced from South America to Sri Lanka and later to Malaysia and other countries in Southeast Asia. By 1910 Asia had become the main supplier of natural rubber. FAO statistical records from 1990–1998 indicate that 6.9 million ha of rubber were planted in India and Southeast Asia including Indonesia, Malaysia, Myanmar, Sri Lanka, Thailand and Vietnam. The major rubber-grower countries are Indonesia, Malaysia and Thailand, each with more than a million hectares.

Like most other cultivated crops, rubber is facing serious problems from several diseases, of which at least 40 have been reported. Among these, phytophthora diseases are affecting rubber in most growing areas. Infection occurs in most parts of the rubber tree including seedpod, leaf, leaf petiole, tapping panel, stem and trunk. However, there is no record of root disease caused by phytophthora in rubber. Black stripe, a disease of the tapping panel, was the first phytophthora disease to be recognised in Sri Lanka in the early 1900s. Later, pod rot, leaf fall, stem or patch canker were reported. The impacts of phytophthora diseases on rubber production are a reduction in latex yield, caused by the panel and stem diseases, and a reduction in growth due to leaf fall. In addition, pod rot affects seed production for root stock propagation.

Prophylactic fungicidal spraying is extensively used to control phytophthora leaf fall in various parts of the world, including India, Malaysia and Sri Lanka. However, application of chemicals to control leaf fall from mature rubber trees is impractical and costly, due to the height of the trees and the large plot sizes. In contrast, disease control using fungicide is more effective and economically attractive to control black stripe and stem canker than leaf fall. In addition, clones that are tolerant to leaf fall — RRIM712, PR255, PR261 and GT1 — have been recommended and are replacing the highly susceptible rubber clones RRIM600 and PR107 in the areas conducive to disease development. Agronomic practices such as reduction of plant density and avoidance of excessively moist conditions by removal of vegetation are also recommended.

In this paper an attempt is made to summarise information regarding phytophthora diseases in rubber, with emphasis on disease incidences in the main rubber-producing countries in Southeast Asia.

Epidemiology

Annual occurrences of phytophthora leaf fall are common in India (Pillai et al. 1989), the southwest coast of Thailand (Kajornchaiyakol 1977, 1980), the

[1] Department of Pest Management, Faculty of Natural Resources, Prince of Songkhla University, Hat Yai, Thailand 90112.

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northern and western states of Malaysia (Johnston 1989), and in Myanmar (Turner and Myint 1980) and Sri Lanka (Jayasinghe and Jayaratne 1996). In these regions the disease is most prevalent during the monsoon with long periods of high rainfall and constant high relative humidity (Wastie 1973). In Thailand, leaf fall epidemics occur during June–December (Pattanakul et al. 2001), and in Sri Lanka during May–September (Jayasinghe and Jayaratne 1996). In most cases, infection first takes place on immature pods, giving rise to pod rot, which then acts as a source of inoculum to fuel the leaf fall epidemic (Pattanakul et al. 2001). The occurrence of black stripe is correlated with leaf fall, and it is often categorised as the second phase of the leaf fall disease. Spores of the pathogen are spread by rain splash from the infected leaves to the tapping panel (Johnston 1989). Experiments in Sri Lanka showed that, under field conditions, a tapping knife did not transmit black stripe and that naturally infected trees showed a high incidence of panel infection close to ground level (Liyanage et al. 1984). Later experiments indicated that *Phytophthora meadii* was isolated from soil in a rubber plantation during epidemics of pod and leaf diseases (Liyanage and Wheeler 1991). Stem or patch canker, another phytophthora disease on rubber, is also associated with the occurrence of black stripe disease, leaf fall and pod rot. Stem or patch canker is common in rubber-growing countries but recent economical losses are relatively minor. Stem canker has been reported in countries in Southeast Asia including Malaysia (Chee 1971), Myanmar (Johnston 1989), and India (Mondal et al. 1994).

Extensive surveys of rubber diseases caused by *Phytophthora* in Thailand have been made since 1976. In general, leaf fall and black stripe are estimated to affect around 10% of the total growing area. An early record of severe damage from leaf fall and black stripe diseases was in 1976 (Kajornchaiyakol 1977). Leaf fall and black stripe outbreaks occurred on the east and the southwest coasts, of Thailand including Chuntaburi, Trad, Ranong, Phanga, Krabi, Phuket, Trang and Satun provinces (Figure 6.5.1 and Table 6.5.1), which cover about 100,000 ha. In the susceptible clone RRIM600, leaf fall occurred in 90–100% of the trees, which led to a 40% drop in yield (Kajornchaiyakol 1977). In 1979, although the area affected was reduced to 2000 ha, the disease severity was similar to that recorded in 1976 (Kajornchaiyakol 1980). A later survey indicated that damage by phytophthora diseases was reduced in southwest Thailand (Chantarapratin et al. 2001) due to replanting with rubber clones that are more resistant to *Phytophthora*.

Disease Symptoms

Phytophthora infection on rubber often begins on young pods and causes pod rot. The infected pods turn black and remain on the tree, dried up and unopened. After pod rot, the infection spreads to leaves and causes leaf fall (Figure 6.5.2). Infected leaves fall in large numbers, forming a carpet on the ground. Leaf blades of shed leaves show few signs of infection (Figure 6.5.3). A typical symptom of phytophthora leaf fall is the appearance of dark-brown lesions on the petioles with one or two drops of coagulated latex in the centre of the lesion (Figure 6.5.4). The lesion is often found near the base of the petiole and causes the premature abscission of the leaf. However, the lesions can occur anywhere along the length of the petioles. Heavy defoliation may lead to dieback of terminal branches (Chee 1968; Runner 1969; Johnston 1989).

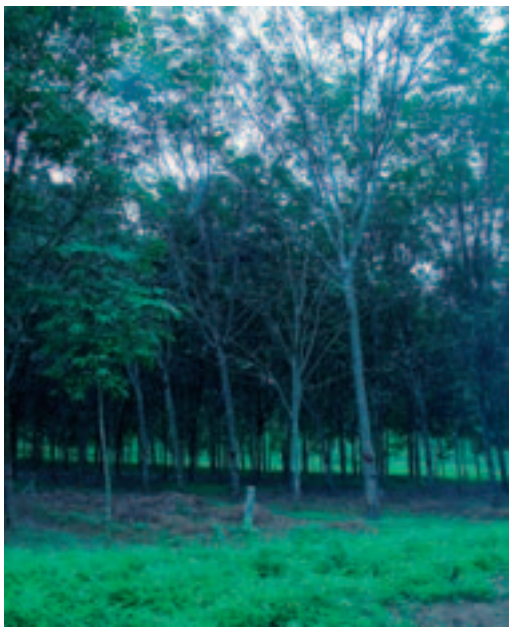

Figure 6.5.1 Rubber trees showing the effects of an outbreak of leaf fall and black stripe disease in Thailand.

Table 6.5.1 Distribution and severity of *Phytophthora* leaf fall disease on rubber in Thailand.

Provinces	Location	Infested area (ha)	Leaf drop (%)
Trad	East	32	3
Chumporn	Southwestern coast	2	10–75
Songkhla	Southwestern coast	5,280	80
Phangnga	Southwestern coast	9,197	80
Krabi	Southwestern coast	5,596	100
Trang	Southwestern coast	76,800	100
Satun	Southwestern coast	560	80

Source: Kajornchaiyakol (1977).

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Figure 6.5.2 Leaf fall from rubber trees, caused by *Phytophthora* infection.

Figure 6.5.3 Leaf blades from *Phytophthora*-infected rubber trees.

The rubber panel is continually injured in the tapping process, so it is vulnerable to fungal infection. *Phytophthora* attacks the tapping panel and causes black stripe disease. Symptoms of black stripe at the early stage of infection appear as a slightly discoloured area above the tapping cut. Later vertical depressions occur on the renewing bark (Figure 6.5.5). When the bark is removed, dark lines are visible, corresponding to the depressions on the panel surface (Figure 6.5.6). As the infection progresses, the black lines extend internally into the wood, coalesce forming broad lesions (Figure 6.5.7) and finally spread the full width of the panel. The infection also causes uneven regeneration of the panel bark. In susceptible clones, protuberance may be formed (Figure 6.5.7). This makes it difficult to tap again (Johnston 1989).

Occasionally, infection occurs on untapped bark and induces stem canker. Symptoms of stem canker begin with discolouration of the bark. This is followed by latex exudation (Figure 6.5.8). A dark-purplish liquid oozes from the damaged bark, forming a coagulum with a distinct odour, and which often causes the bark to bulge and split open (Figure 6.5.8). Internally, the disease symptoms are similar to black stripe disease but occur on the stem, mature branches and/or the branch–stem intersection (Pereira et al. 1995). When the disease occurs at the base of the trunk, it is called patch canker. In comparison, stem canker is less important than black stripe disease in terms of disease incidence.

Figure 6.5.4 Dark-brown lesions on the petioles with one or two drops of coagulated latex in the centre of the lesion are a typical symptom of phytophthora leaf fall

Pathogens

Several species of *Phytophthora* have been reported to be responsible for diseases in rubber, including *P. botryosa* (Chee), *P. capsici* (Leonian), *P. citrophthora* (Smith and Smith) Leon, *P. meadii* McRae,

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P. nicotianae Breda de Haan, and *P. palmivora* (Butl.) Butl. However, the most common *Phytophthora* species causing disease in rubber are *P. palmivora*, *P. meadii,* and *P. botryosa* (Table 6.5.2). In Brazil, *P. capsici* was reported to be the main species associated with black stripe and stem canker, but *P. palmivora* and *P. citrophthora* were also isolated from diseased rubber (Dos Santos et al. 1995). The predominant *Phytophthora* species infecting rubber in India, Myanmar, and Sri Lanka is *P. meadii* (Liyanage 1982; Kochuthresiamma et al. 1988; Johnston 1989), whereas in Malaysia, Thailand and Vietnam *P. palmivora* and *P. botryosa* are implicated (Chee 1969, 1971; Tsao et al. 1975; Duong et al. 1988). In China, although the main species involved appears to be *P. citrophthora*, other species including *P. palmivora*, *P. meadii*, *P. nicotianae* and *P. capsici* were also found to infect rubber (Zeng and Ward 1998). *P. citrophthora* was reported for the first time infecting rubber in Indonesia in 1989 (Liyanage and Wheeler 1989).

Figure 6.5.5 Symptoms of black stripe disease, caused by *Phytophthora* on the tapping panel of a rubber tree.

Figure 6.5.6 Under-bark depressions.

Figure 6.5.7 Lesions extending from the bark into the wood.

Figure 6.5.8 Latex exudation from a stem canker.

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Disease Control

Control measures for phytophthora diseases on rubber involve fungicide application, planting of tolerant clones, using appropriate cultural practices, and disease forecasting. Copper oxychloride in mineral oil is extensively used in India, Malaysia and Sri Lanka as a preventive spray in the management of phytophthora leaf fall (Jayasinghe and Jayaratne 1996). However, application of chemicals to control leaf fall from mature rubber trees is impractical and costly due to the height of the trees and the large plot sizes. Metalaxyl, oxadixyl, catafol, folpet or mancozeb are recommended for panel treatment to control black stripe (Tan 1983; Jayatissa et al. 1994; Jacob et al. 1995). In India, 0.8% phosphorous acid gave effective and economic protection of tapping panels of the rubber trees from black stripe disease when applied at weekly intervals (Jacob et al. 1995).

Chemical control alone is increasingly becoming an unacceptable strategy due to the impact on the environment. Steps have already been taken to introduce an integrated approach to phytophthora disease management on rubber, with special emphasis on genetic resistance (Radziah and Hashim 1990; Jayasinghe and Jayaratne 1997). Screening and genetic improvement of rubber for resistance to *Phytophthora* have been implemented in Southeast Asia (Pattanakul et al. 1975; Pillai et al. 1989; Jayasinghe and Jayaratne 1996,). Several tolerant clones have been established and successfully planted in areas where the diseases are endemic. Tolerant clones recommended for Southeast Asia include RRIM712, PR255, PR261 and GT 1 (Anon. 1986). Previously popular rubber clones RRIM600 and PR 107 have been found to be susceptible to phytophthora diseases in most countries in Southeast Asia (Johnston 1989).

Forecasting phytophthora epidemics on the basis of weather data is saving unnecessary fungicide applications. Since rainfall coincides with the presence of pod rot in the field, which gives rise to phytophthora leaf fall and is subsequently followed by black stripe, fungicide should be applied with the onset of the leaf fall and continued for 2–4 weeks after the rain has ceased (Satchuthananthavale and Dantanarayana 1973).

Cultural practices also pay an important role in phytophthora disease management. In Thailand, weed control in rubber plantations is recommended as a means of suppressing disease development by reducing humidity during the long periods of rainfall (Pattanakul et al. 2001). In addition, experiments conducted in Malaysia indicated that factors leading to black stripe disease were the tapping of wet rubber trees during pod infection

Table 6.5.2 *Phytophthora* species associated with rubber diseases.

Species	Country	Reference
P. botryosa	Malaysia Thailand Vietnam	Chee (1968) Tsao et al. (1975) Duong et al. (1998)
P. capsici	Brazil China	Dos Santos et al. (1995) Pereira et al. (1995) Zeng and Ward (1998)
P. citrophthora	Brazil China Indonesia	Dos Santos et al. (1995) Zeng and Ward (1998) Liyanage and Wheeler (1989)
P. meadii	India Myanmar Sri Lanka	Kochuthresiamma et al. (1988) Johnston (1989) Liyanage (1982) Jayatissa et al. (1994)
P. palmivora	Brazil China Indonesia Malaysia Sri Lanka Thailand Vietnam	Dos Santos et al. (199 Pereira et al. (1995) Zeng and Ward (1998) Parnata (1983) Chee (1969) Dantanarayana et al. (1984) Tsao et al. (1975) Duong et al. (1998)
P. nicotianae	China	Zeng and Ward (1998)

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(Peries 1976). Also, it has been found that phytophthora disease intensity increased at tree-planting densities above 500 per ha (Anon. 1973).

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6.6 Phytophthora Diseases of Durian, and Durian-Decline Syndrome in Northern Queensland, Australia

Emer O'Gara,[1] David I. Guest,[1,2] Lynton Vawdrey,[3] Peter Langdon[3] and Yan Diczbalis[3]

Abstract

Durian is the most popular fruit in Southeast Asia, with high economic and cultural value to the producing countries, which include Indonesia, Malaysia, Philippines, Thailand and Vietnam. The greatest threat to durian production in all countries is *Phytophthora palmivora*, which affects all stages of the cropping cycle. This chapter describes the diseases caused by *P. palmivora*, and their epidemiology. The chapter also describes a perplexing durian-decline syndrome which occurs in northern Queensland, where it appears that *P. palmivora* is operating in a complex with *Pythium vexans* and nematodes from the *Xiphenema* genus. Early control recommendations and their limitations are described, which leads to a discussion of integrated disease management principles and their applicability to the control of phytophthora diseases in durian.

The high-rainfall conditions under which durian is grown are conducive to the development of phytophthora diseases. In Southeast Asia. the most serious diseases of durian are caused by *Phytophthora palmivora. Phytophthora palmivora* causes seedling dieback, leaf blight, root rot, trunk cankers, and pre- and postharvest fruit rots (Lim 1997). *Phytophthora nicotianae* has also been reported as being a causal agent of durian root rot and canker on a few occasions in Malaysia (Bong 1993). Postharvest fruit rots result in 10–25% losses of durian fruits (Lim 1990).

Phytophthora Diseases in Durian

The genus *Phytophthora* is considered to be one of the most important plant pathogens worldwide. It has been identified as a major impediment to the development of a sustainable durian industry in Australia (Zappala 2002). *Phytophthora nicotianae*, *P. botyrosa* and *P.* spp (durian) have been identified as pathogens of durian (Bong 1993; Erwin and Ribeiro 1996; Brown 1997; M. Weinert, pers. comm.), but the most destructive and economically significant diseases are caused by *P. palmivora* (Navaratnam 1966; Pongpisutta and Sangchote 1994; Lim 1998). *Phytophthora palmivora* is endemic to Southeast Asia, where there is much genetic diversity, and balanced populations of the A1 and A2 mating types occur (Lee et al. 1994; Mchau and Coffey 1994) To date only the A1 mating strain has been associated with diseases in durian (Lim 1990; Lee et al. 1994).

Although essentially a soil-borne pathogen, *P. palmivora* is adapted to attack aerial parts of the plant (Chapter 3.1) and, as a result, can affect all organs of durian and all stages of the cropping cycle. The most devastating diseases include seedling dieback, foliar blight, patch canker of the trunk and branches, and pre- and postharvest fruit rots (Lim 1990).

[1] School of Botany, The University of Melbourne, Victoria 3010, Australia.

[2] Current address: Faculty of Agriculture, Food and Natural Resources, The University of Sydney, New South Wales 2006, Australia.

[3] Centre for Wet Tropics Agriculture, Department of Primary Industries, South Johnstone, Queensland 4859, Australia.

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Seedling dieback and foliar blight

Seedling dieback is common in durian nurseries and, where disease management is poor, losses can be as high as 50% (Lim 1990). Infection is commonly initiated at the young stem, or at the graft union in double rootstocks, with a conspicuous lesion. Under suitable conditions the infection quickly spreads to the roots and leaves, producing dieback symptoms. When the root system becomes extensively rotted, and/or the main stem is girdled, the seedling will die.

Leaf blight may occur on individual leaves or, in extreme cases, the whole foliage may become diseased (Figure 6.6.1), killing the seedling from the top (Lim 1990). Although more common in nurseries, foliar blight can occur also in orchards under conditions of extremely high disease pressure. By the time foliar symptoms become apparent in an orchard, infections in other organs of the tree are generally well advanced and remediation is difficult if not impossible (Bong 1993).

Figure 6.6.1 Seedling blight of durian caused by *Phytophthora palmivora*.

Patch canker of the trunk and branches

Patch canker may begin at the soil line or at the crotch region (Lim 1990), although in Thailand cankers are often first observed on branches high in the tree canopy (S. Sangchote, pers. comm.). Cankers first become evident as discrete wet-looking patches on the bark. The patches eventually coalesce to produce a conspicuous canker that exudes a reddish/brown resinous substance. When the bark is removed, a reddish/brown lesion is revealed in the cortex which, in a healthy state, is cream to pink (Figure 6.6.2). Infection commonly extends into the xylem and, when the main trunk or root is girdled, leaves wilt and become chlorotic and branches desiccate, producing classical dieback symptoms. Lesions may also be found on feeder and large lateral roots (Bong 1993), in which case root rot will contribute to the above-ground symptoms. Infected trees may survive many years from the time of initial infection, as pathogen activity slows considerably during the dry season, although the stress of drought on the host may speed up infection in the following rainy season (Cook 1975; Lim 1990).

Figure 6.6.2 Lesion beneath the bark at the lower trunk of a durian tree. The lesioned tissue is brown compared to the creamy/pink colour of the healthy tissues.

Pre- and postharvest fruit rot

The incidence of preharvest fruit rot due to *P. palmivora* in Malaysian durian orchards can be as high as 30%, depending on the weather and microclimate (Lim 1990; Lee 1992). The following disease description is from Lee et al. (1994) and

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applies to pre- and postharvest diseases (see also Figure 6.6.3):

> The disease first appears as tiny water-soaked lesions on the outer skin which later coalesce to form dark to black brown regions. White powdery masses of sporangia form on the lesion surface, especially when conditions are moist and humid.

The rot spreads rapidly through the skin and pulp to the seed, making the fruit unmarketable and inedible (Lim 1990; Lee et al. 1994).

P. palmivora can infect fruit at all stages of development, and preharvest infections can result in postharvest rots (Johnson and Sangchote 1994). Preharvest infection may not be apparent at the time of harvest, or infection can occur during harvest when fruit is allowed to come into contact with infested orchard soils. In either case, if conditions are favourable during transit, *P. palmivora* can spread throughout, and ruin whole consignments of fruit. Favourable conditions for postharvest infection of non-wounded fruit include high humidity (at least 98% relative humidity) for at least 72 hours (Chapter 3.2).

Figure 6.6.3 Durian fruit with large brown lesion caused by *Phytophthora palmivora*. Sporangia have formed in white powdery masses between the spines.

Disease Epidemiology

The most important characteristics of *P. palmivora*, from an epidemiological perspective, are short generation time, great reproductive capacity under favourable conditions, and the production of deciduous sporangia that readily release zoospores in the presence of free water (Erwin and Ribeiro 1996). We have a good understanding of the epidemiology of *P. palmivora* in cocoa (Chapter 4.1).

P. palmivora is endemic to tropical Southeast Asia and survives in soil and on abscised or thinned durian fruit that has been left on the orchard floor (Lee 1992; Chapter 3.1). Disease develops in durian nurseries where humidity is consistently high due to a high density of seedlings, excessive watering (sometimes with infested water), excessive shade, inadequate ventilation and poor drainage. The situation is exacerbated by the maintenance of seedlings at ground level where they are exposed to soil-splash of infested water (Figure 6.6.4). The deciduous sporangia produced on the surface of stem or foliar lesions are spread by seedling-to-seedling contact, irrigation and human activities. Potential infection courts include wounds or stomata, which are prevalent on leaves, petiole and young stems (Chapter 3.2).

Figure 6.6.4 Durian seedlings maintained in a nursery on bare soil at ground level. Water has ponded around the plants and the seedlings are subject to splash of soil and water infested with *Phytophthora palmivora*.

Of particular concern is the practice in some nurseries of using phosphonate as a soil drench, because although it will suppress disease development in the plant, the pathogen remains viable, and its presence is merely masked. In this way, infested soil is unwittingly introduced into orchards.

Conditions that encourage high humidity in the orchard exacerbate disease. These include close plantings with intertwining dense canopies (Figure 6.6.5), poor drainage (Figure 6.6.6), poor hygiene

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(Figure 6.6.6) and cultivation of susceptible varieties (Erwin and Ribeiro 1996; Lim 1990).

Evidence from research in Papua New Guinea indicates that beetles are key agents in the transmission and spread of *P. palmivora* in cocoa (Konam 1999; Konam and Guest 2004; Chapter 6.2). Durian patch cankers are attractive to boring beetles (Cook 1975) and it is likely that some of the many insects that occur in durian orchards (Figure 6.6.8) act as vectors of the abundant deciduous sporangia that form on infected organs, particularly fruit. Tent-building ants and termites also carry infested soil up the tree. The transmission of sporangia by insects may explain the initiation of infections high in the canopy, as observed in Thailand.

Durian fruit generally ripens in the early rainy season when climatic conditions for infection and colonisation of the host are optimal. The pathogen can penetrate the cuticle of the fruit in the region between the spines, or invade through wounds or stomata (Chapter 3.2). Abundant sporangia are produced on the developing lesions (Figure 6.6.3), and the wind and rain associated with the monsoon facilitate both wounding and the dissemination of sporangia within the already infected tree and throughout the orchard. Drops of rain carrying sporangia collect at the stylar end of the fruit, causing infection that spreads upwards on the fruit in a concentric pattern (Lee et al. 1994), and water dropping from the fruit carries sporangia to fruit and branches below. Infected fruit or leaves drop prematurely, returning inoculum to the soil. Failure to remove infected fruits will provide an energy source for an explosive increase of inoculum. Cryptic infections on ripe fruit will initiate postharvest rots during transit and storage.

Figure 6.6.5 Dense plantings and closed canopies lead to high humidity in the orchard providing ideal conditions for the proliferation of *Phytophthora palmivora* and infection of durian. Note the high watertable.

Figure 6.6.6 In some durian growing regions of Vietnam 'moats' are created around trees to facilitate manual irrigation (water is pumped into the moat in the dry season). However, water is trapped against the trunk of the tree in the wet season causing disease.

Figure 6.6.6 *Phytophthora*-infected durian fruit in an irrigation channel where they will produce inoculum for further infections within the orchard.

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Disease Control Options — a Historical Perspective

An understanding of the epidemiology of the moisture-loving *Phytophthora* led to recommendations for cultural disease control as early as the 1960s; they include good drainage and methods to improve ventilation and reduce humidity, such as wider spacing of trees, pruning of lower branches and the removal of weeds from under the canopy (Navaratnam 1966; Cook 1975).

Durian cultivars have historically been selected for fruit quality and productivity. Disease resistance was a secondary concern and reports of it anecdotal until 1971, when the first screening studies were conducted in Malaysia (Lim 1998). An underutilised source of resistance potentially exists in wild and semi-wild populations of *Durio* spp. and closely allied genera growing in Malaysia and Indonesia, the centre of diversity (Lim 1998). Techniques developed to identify disease resistance characteristics in durian are discussed in Chapter 8.4. Once identified, resistance can be exploited through plant-breeding programs, although both require a long-term commitment of funds and time. An alternative and more rapid method of producing disease-resistant planting material is to use the resistant cultivar as a rootstock, onto which a scion with desirable commercial qualities is grafted (Lim 1998). This method is practised in Thailand where farmers routinely use Chanee as a rootstock due to a perceived disease-tolerance relative to other cultivars.

Recommendations for the chemical control of patch canker in durian did not change greatly between 1934, when the disease was first reported, and the mid 1990s (Lim 1990; Erwin and Ribeiro 1996). The main control option was the removal of the cankered tissue and painting the wound with an antimicrobial chemical and, in some cases, covering it with a dressing or tar (Cook 1975; Lim 1990; Lee 1992; Bong 1993; Erwin and Ribeiro 1996). This method gave inconsistent results, probably as there is limited penetration of the chemical into woody tissues and the fungicide is easily washed away. In addition, the process is laborious and expensive, and there were varying levels of diligence in reapplication (Lee et al. 1994).

The choice and effectiveness of fungicides to treat phytophthora diseases has increased over the years. The use of basic disinfectants gave way to protectants, including improved copper

Figure 6.6.8b A millipede moving over a weeping canker on the trunk of a durian tree, with the potential to pick up infectious propagules for distribution elsewhere in the orchard or further up the tree.

Figure 6.6.8a Termites build mounds around durian trunks with *Phytophthora*-infested soil increasing the risk of trunk canker.

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formulations, dithiocarbamates (e.g. mancozeb) and phthalimides (e.g. captafol), followed by systemic fungicides effective against oomycetes, such as the acylalanines (e.g. metalaxyl) and the phosphonates (e.g. fosetyl-al, phosphorous acid) (Navaratnam 1966; Lim 1990; Kendrick 1992).

New formulations with different modes of action brought alternative recommendations for the methods of application. These included soil drench, foliar spray and, most recently, for woody perennials, direct injection into the trunk with the systemic formulations (Lim 1990). Some systemics, including metalaxyl, act on specific biochemical targets within the fungus, so it wasn't long before resistance to the fungicide was reported in *P. infestans* (Davidse et al. 1981; Kendrick 1992; Fungicide Resistance Action Committee (FRAC) website at <www.frac.info/publications/FRACCODE_sept2002.pdf>). New reports of fungicide resistance in other species of *Phytophthora*, and in *Pythium*, continue to mount (Parra and Ristiano 2001; Taylor et al. 2002). To reduce the risk of fungicide resistance in *P. palmivora*, a combination of protectant fungicides and metalaxyl is recommended for topical application (Lim 1990; Bong 1993).

Durian fruit rot was controlled by spraying with the same formulations recommended for patch canker and other diseases. However, there were unresolved issues about residues, stains on the skin left by the chemicals, and the difficulty of reaching fruit in the upper canopy without the aid of expensive high-pressure equipment (Lim 1990; Lee et al. 1994).

In the late 1970s, phosphonate emerged as a chemically simple, relatively inexpensive, yet highly effective weapon against *P. cinnamomi* diseases in avocado. Due to its systemic nature and ambimobility it was particularly suited to application as a trunk injection (Darvas et al. 1984), which circumvented the problem of fungicide wash-off. By the late 1980s, phosphonate trunk-injection was being successfully applied in other *Phytophthora* pathosystems, including *P. palmivora* on cocoa (Guest et al. 1994) and durian (Lim 1990; Lee et al. 1994) although phytotoxicity was reported in durian when rates of phosphonate application exceeded 25 g active ingredient (a.i.)/year (Lee 1992).

A common theme in disease control recommendations is the importance of early treatment, and the difficulty of saving trees that are suffering several phytophthora diseases simultaneously (Bong 1993: Erwin and Ribeiro 1996). Initial inoculum level is the key element in Vanderplank's model for epidemics in multi-cyclic pathogens such as *P. palmivora* (Erwin and Ribeiro 1996). Erwin and Ribeiro (1996) make the following points:

- inoculum can be reduced but not entirely eliminated through scrupulous hygiene
- the pathogen is less likely to sporulate on planting material with vertical resistance, but vertical resistance is elusive (especially in woody perennials like durian), and usually not durable because of the reliance on a single gene, which puts great selection pressure on the pathogen to adapt
- a chemical blitz can potentially reduce the inoculum levels to zero, but eradicants such as methyl bromide are being phased-out due to the environmental hazards they pose and, as already mentioned, *Phytophthora* is showing tolerance to some of the most-effective selective fungicides currently available.

In highlighting the fact that no single method will effectively and sustainably reduce inoculum levels and thus control multi-cyclic pathogens, Erwin and Ribeiro (1996) succinctly present the case for integrated disease management. The case for integrated disease management is bolstered by a rise in our consciousness of environmental and health issues, which makes our past reliance on chemicals for disease control unacceptable.

Integrated Disease Management

Integrated disease management (IDM) is the long-term control of crop diseases to economically acceptable levels through a holistic approach which combines:

- the use of resistant varieties where available
- cultural control methods
- biological control methods
- the judicious application of appropriate chemicals.

Durian is an ideal model for the development of IDM strategies because the high value of the fruit provides impetus for the intensive and continuous orchard management practices required in a perennial tree crop.

The principle of integrated management of phytophthora diseases in durian has been promoted since the early 1990s (Lim 1990; Bong 1993; Lee et al. 1994) but, for the most part, detailed recommendations were lacking or implementation patchy. A systematic approach to developing recommendations was undertaken as part of an

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ACIAR-funded project 'Management of *Phytophthora* diseases in durian' (Project No. PHT/1995/134), which commenced in 1998. As part of the project, practical disease-control options were investigated, regionally optimised and disseminated to durian farmers in Thailand, Vietnam and Australia. The project culminated in a workshop that was held in Chiang Mai, Thailand in November 2002. The presentations there formed the nucleus for the production of this monograph.

The recent, rapid expansion of the durian industries in Thailand and Vietnam has seen the establishment of orchards on marginal sites, including rice paddy in Vietnam (Figure 6.6.9), where phytophthora diseases can be exacerbated. Major issues facing the durian industries in Thailand and Vietnam and investigated as part of Project PHT/1995/134 included:

- the need to identify sources of disease resistance in durian and the development of tolerant rootstocks (Chapter 8.2)
- poor practice in durian nurseries resulting in the release of infected planting material (Chapters 7.1 and 8.3)
- an incomplete understanding of the epidemiology of *P. palmivora* in durian, which hampers effective management (Chapters 3.1 and 2.2)
- an incomplete understanding of the effect of current management practices on disease incidence and development (Chapter 7.2 and 8.3)
- the lack of specific recommendations for the rate and timing of phosphonate trunk-injection to ensure efficient application and effective disease control (Chapter 6.3 and 8.4).

Durian-Decline Syndrome in Australia

Although the fledgling durian industry in Australia is facing many of the same issues as Thailand and Vietnam, the major problem in northernmost growing areas in Queensland is a devastating decline syndrome. Durian-decline syndrome (DDS) involves the rapid dieback of branches, necrosis in the cortex of feeder roots and eventually tree death (Figure 6.6.10). The symptoms are initially suggestive of disease caused by *P. palmivora*, except that cankers are rare and trees do not respond to trunk-injection with phosphonate. In an attempt to determine the cause of DDS, 13 affected farms were surveyed in a dry season (July–September 2001) and the following wet season (February–April 2002). *P. palmivora* was isolated from the roots of affected trees on 12 of the 13 farms in the dry season, and all farms in the wet season. *Pythium vexans* de Bary was recovered from the roots of diseased trees on all 13 farms in both seasons. *Pythium vexans* was isolated from 68% of diseased trees, while *P. palmivora* was isolated from 24% of diseased trees in the dry season. In the wet season *P. vexans* was isolated from 45% of diseased trees, while *P. palmivora* was isolated from 35% of diseased trees. *Xiphenema* sp., a root-hair-feeding, plant-parasitic nematode, was also recovered from 12% of trees sampled. These results suggest a possible synergism between *P. palmivora, P. vexans* and plant-parasitic nematodes as the complex cause of DDS in northern Queensland.

The pathogenicity of *P. palmivora*, *Pythium vexans*, or a combination of the two pathogens, was tested on 3-month-old durian seedlings cv. Monthong. Inoculum of *P. palmivora* (chlamydospores) and *P. vexans* (oospores) was prepared using the submerged culture method described by Tsao (1971). A spore suspension (approximately 1×10^5 spores) was applied to the potting medium in each pot. Four replicate plants were used per treatment. An uninfected treatment was included for comparison. Two weeks after the inoculum was

Figure 6.6.9 The establishment of a new durian orchard in a rice paddy in the Mekong Delta region of Vietnam. The mounds on which the seedlings are planted, are expanded each year to accommodate the lateral growth of the root system. Eventually there will no longer be room to plant the rice.

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Figure 6.6.10 Advanced symptoms of durian decline syndrome in far-north Queensland, Australia.

applied, the pots were placed in plastic trays and filled with water to a depth of 25 mm to saturate the soil by capillary action, which stimulates chlamydospore and oospore germination, sporangial development and zoospore release.

After 3 days, the pots were removed from the trays and the soil allowed to drain. Thereafter, plants were hand-watered as required. Plant roots were assessed for root rot after a further 6 weeks. Disease-affected roots were plated onto selective culture media and *P. palmivora* and *P. vexans* were re-isolated from infested plants.

Plants inoculated with *P. palmivora* showed obvious rotting of, and a reduced number of, feeder roots. Feeder roots of plants inoculated with *P. vexans* appeared necrotic compared with controls but there was no obvious reduction in the number of roots. *P. vexans* may cause a reduction in the efficiency of affected feeder roots. A combination of *P. palmivora* and *P. vexans* failed to increase the severity of root rot compared with *P. palmivora*, which may have been a function of insufficient time under waterlogged conditions. Further experiments, including nematodes, are warranted.

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