

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

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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 *Xiphinema* 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. botryosa* 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).

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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

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

(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 dripping 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.

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.

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

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.



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.

Acknowledgments

We thank Dr T.K. Lim for critical comments during preparation of the manuscript.

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