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Fusarium wilt of banana in Australia: a review*

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Introduction

In 1874, when Dr Joseph Bancroft was describing for the first time in the world a wilt disease affecting banana plants at Eagle Farm, Brisbane (Bancroft 1876), he would have been unaware that he was investigating what was to become recognised as one of the most widespread and destructive plant diseases in the recorded history of agriculture (Simmonds 1966). In terms of crop destruction, it was to rank with the few most devastating plant diseases such as wheat rust and potato blight (Carefoot and Sprott 1969). This disease was next reported in banana plantations grown for export in Central America in 1890 (Ashby 1913). From 1890 to the mid 1950*s*, some 40 000 hectares of the banana cultivar Gros Michel in Central and South America were destroyed or abandoned because of the disease.

Although Bancroft first reported the disease in 1876, it was not until 1910 that Dr Erwin Smith recovered the pathogen from host tissue sent from Cuba and named it *Fusarium cubense* E. F. Smith (Smith 1910). The first detailed description of the disease and the pathogen was published by Ashby (1913), while Brandes (1919) was the first to demonstrate pathogenicity conclusively. In 1940, Snyder and Hansen proposed the name *Fusarium oxysporum* Schlecht. f.sp. *cubense* (E. F. Smith) Snyd. & Hans. (*Foc*) (Snyder and Hansen 1940).

The disease became known as Panama disease because it was first epidemic in the Central American country of Panama. The name, Panama disease, was initially used in the literature by Rorer (1911) and Drost (1912). The more descriptive term, banana wilt, was first used in Jamaica in 1915 (Stover 1962*a*), but this does not differentiate between Fusarium wilt and bacterial wilt of banana (Moko disease). Since *Foc* consists of many strains and a wide range of banana clones are affected, the name Fusarium wilt is more appropriate and in agreement with the use of Fusarium wilt for other crop diseases caused by *Fusarium oxysporum*.

Banana production in Central America was maintained during the first half of this century by the short-sighted policy of clearing and planting new land

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(usually primary forests). Due to disruptive social and political effects, such land soon became more difficult to obtain (Carefoot & Sprott 1969). Ecuador then entered the international banana trade on a massive scale in the mid 1950*s*, resulting in the end of the Gros Michel era in Central America. To compete with cheap Gros Michel fruit from Ecuador, the *Foc*-infested soils of Central America were replanted with wilt-resistant Cavendish cultivars (Stover 1986). Cavendish cultivars growing in the Latin American–Carribean region are still resistant to the disease (Stover 1990), but they are being damaged severely from Fusarium wilt in subtropical production areas (Ploetz *et al.* 1990) and in equatorial regions of Asia (Moore 1994).

Although Fusarium wilt is best known for its influence on the export banana industries, only 10% of total world banana production (an estimated 70 million tonnes) is exported. The remaining 90% is grown and consumed locally, often as a staple food. The impact of losing a few banana plants to Fusarium wilt in subsistence agriculture is not as obviously great as that of the large scale losses incurred in export plantations; however, it is very significant to the smallholders in Brazil, Asia, and East Africa. As of 1995, Panama disease has been reported from all banana-growing regions of the world except Papua New Guinea, the South Pacific Islands, and some of the countries bordering the Mediterranean. The early history of Fusarium wilt and its damage to the export industries has been reviewed comprehensively by Stover (1962*a*) and more recent events by Ploetz (1990*a*). This review summarises the history of the disease and the research conducted in Australia.

Classification of banana cultivars

Banana is a monocotyledon which belongs to the family Musaceae within the order Zingiberales. There are 2 genera in Musaceae, *Musa* and *Ensete*. Banana originated in South East Asia and in the Indian subcontinent probably several thousand years ago. All edible banana cultivars except for the Fe'i group are essentially derived from the 2 wild, seeded species, *Musa acuminata* Colla and *Musa balbisiana* Colla. Both *M. acuminata* and *M. balbisiana* are diploid ($2n = 22$) and their genomic constitutions are referred to as AA and BB, respectively (Simmonds and Shepherd 1955). Edible banana cultivars are monospecific (pure *M. acuminata*) or interspecific (*M. acuminata* × *M. balbisiana*) in origin. There are several different ploidy levels: diploid (AA and AB groups), triploid (AAA, AAB, ABB groups), and occasionally tetraploid (ABBB). The term banana refers to all plants within the genus *Musa*, but more specifically to the edible fruit which is sweet when ripe. The term plantain has been used widely and loosely to refer to both AAB and ABB groups of starchy bananas which are eaten after cooking. Plantain refers more correctly to the Plantain (AAB) subgroup and the term 'cooking banana' should be used for the ABB cultivars.

The Australian banana industry

While small in world terms, banana production in Australia is a significant horticultural industry and bananas have become the major fresh fruit line in the market. Approximately 12 700 hectares of banana annually produce 18·46 million 13-kg cartons of fruit with a market value of A$330 million, nearly all of which is consumed in Australia.

In Australia, commercial plantings began around 1870 on the northern and western slopes of Buderim, just north of Brisbane. In the early 1900*s*, the main production area was in North Queensland where 1400 hectares were planted to banana in the Cairns and Innisfail areas (Watson 1988). The crop was grown almost exclusively by Chinese farmers who settled in the coastal region after the decline of the Palmer goldfields in the late 1870*s*. Each week, 30–40 bunches were transported by sea from Cairns and Innisfail to markets in Brisbane, Sydney, and Melbourne. After 1910, production declined due to banana imports from Fiji, periodic cyclones, and unreliable shipping. After World War I, there was further decline due to shortage of ships, and land being used to grow sugar cane in preference to banana. The banana industry then expanded in the subtropical areas of southern Queensland and northern New South Wales helped by an import duty on Fijian bananas, and greater proximity and access to the major domestic markets. Initially, the industry was based on the cultivar Dwarf Cavendish (AAA 'Cavendish'), but this was replaced by cv. Williams (AAA 'Cavendish'), which is more cold-tolerant.

The major production areas are now located in the wet semi-tropics of North Queensland and the coastal subtropical areas of southern Queensland and northern New South Wales. Minor production areas are located at Carnarvon in Western Australia, where the climate is subtropical, and in the semi-arid tropics at Kununurra in Western Australia and at Katherine and near Darwin in the Northern Territory.

Cultivars in production in Australia

The Australian industry is based almost exclusively on Cavendish cultivars; Williams now represents about 95% of total production. The origin of Williams is obscure. Some suggest that it appeared as a mutation in a Dwarf Cavendish plantation in the Clarence Valley in New South Wales. Others claim that it was introduced from Fiji in 1902 by a Captain Williams. Mons Mari, a cultivar which originated as a somatic mutation in a Dwarf Cavendish plantation at Buderim in 1910, is very similar to Williams and both clones are now referred to by the latter name (Turner and Hunt 1984). Lady Finger (AAB 'Pome') is the only non-Cavendish cultivar of significant commercial importance in Australia. With its sweet-acid flavour and long shelf life as a ripe fruit, Lady Finger is preferred by some consumers. This cultivar usually commands double the price of Cavendish as a consequence of its restricted availability due to its susceptibility to Fusarium wilt.

Distribution and history of Fusarium wilt in Australia

The history of Fusarium wilt in Australia is well documented. In February 1874, a medical practitioner, Dr Joseph Bancroft, investigated a banana disease at Eagle Farm near Brisbane (Bancroft 1876). It was damaging the Sugar banana (AAB 'Silk') but not affecting Dwarf Cavendish plants growing nearby. Bancroft's description of the symptoms leaves no doubt that he was dealing with Fusarium wilt. His description recorded the discoloration of vascular strands in the host and the fungus associated with them.

> 'From microscopic observation made then, and more recently, the disease appears to be of a fungoid nature. The spiral threads on fracture of a banana stool, the true stem

> of the plant, are easily seen, and if diseased, some draw out of a bright orange colour, others are darker like mahogany. This spiral thread which forms the wall of the air vessel is the part first to suffer by invasion of the mycelium, which, passing through the interior of the stool, attacks the new buds; consequently, when a young banana plant is cut off the disease is carried with it to new ground. Diseased section of the stool, when very slight, shows red marks in short lines not thicker than a fine hair... Passing down the inner face of the sheaths may be seen ruby red spots and streaks traceable to the attachment to the stool.'

As well as being the first description of Fusarium wilt in the world, it is also significant that this was the first recorded plant pathological investigation in Queensland. Bancroft advised growers to select planting material from plants free of the disease, still a current recommendation.

Tryon (1912), when describing a wilt disease of Sugar banana and Gros Michel (AAA), stated that 'the Cavendish banana either escapes its onslaught altogether or is highly resistant thereto', and also noted that 'once the disease is in the field it never leaves it'. Commercial production of Sugar bananas ceased many years ago because of the cultivar's susceptibility to Fusarium wilt. Gros Michel, although introduced into Australia on a number of occasions after 1910, was not grown widely due to its tall growth habit, lower yield than Dwarf Cavendish, and susceptibility to Fusarium wilt.

In 1947, Magee and Simmonds independently expressed concern about the susceptibility of Lady Finger, a variety first grown in Queensland in the 1880*s*, to Fusarium wilt (Magee 1947; Simmonds 1947). They noted, however, that this cultivar was less susceptible to Fusarium wilt than Sugar and Gros Michel. Fusarium wilt in Lady Finger is now found in most production areas in eastern Australia.

In 1953, Purss reported a disease resembling Fusarium wilt on 3 Williams plants at Woongoolba in southern Queensland (Purss 1953). A *Fusarium* species was isolated and proved to be pathogenic on plants of Williams and Lady Finger. At the same time, Purss found that an isolate of *Foc* from Lady Finger did not affect Williams.

By 1969, Fusarium wilt had become such a serious problem in Lady Finger plantations in Queensland that Nicholson began evaluating the resistance of possible replacement cultivars (Nicholson 1969). He planted a number of cultivars in a field at Nambour where Lady Finger had succumbed to the disease. Mysore (AAB) and the hybrid I.C.2 (AAAA) from the Banana Breeding Research Scheme in Jamaica were found to be susceptible, but Williams and 2390–2 (AAAA) and Bodles Altafort (AAAA), two other Jamaican hybrids, were not affected. However, because the fruit of these two hybrids did not meet commercial post-harvest standards, they were not accepted by the Australian industry.

In 1976, R. A. Peterson (pers. comm.) observed Fusarium wilt in cultivar Williams at Wamuran in south-east Queensland. Wilt had been observed on Cavendish cultivars by growers in this area for at least the preceding decade, but since only occasional plants were affected, it was not considered important. Affected plants were growing in shallow clay soils subject to periodic waterlogging. Since the rate of disease spread was much slower than that observed in Lady Finger plantations, suboptimal soil and cultural factors, rather than a change in the pathogen, were considered to be the cause of the breakdown in resistance. This was in concurrence with the hypothesis of Stover and Malo (1972) and

Waite (1977) who had observed similar outbreaks of Fusarium wilt in Cavendish growing under suboptimal edaphic conditions in other countries.

However, in the late 1970*s* and early 1980*s*, the incidence of Fusarium wilt in Cavendish plantations in subtropical Queensland increased significantly. Mayers (1983) reported that Cavendish was being attacked more severely than would be expected if suboptimal edaphic conditions were interacting with previously recognised strains of the fungus. He conducted pathogenicity tests that provided evidence for a new strain of *Foc* that was capable of causing disease in Cavendish cultivars. The disease now occurs in Cavendish plantations located between Caboolture and Yandina in south-eastern Queensland. The disease was also detected in 1983 in a small Cavendish planting near Byron Bay in northern New South Wales (Brake *et al.* 1990) and has since been recorded in several plantations in the Burringbar and Eungella regions of northern New South Wales.

Fusarium wilt has been known at Carnarvon in Western Australia since 1949, when it was reported in windbreak banana plants (edible AA diploid) used to protect Cavendish cultivars from the almost continuous southwesterly winds and the occasional hot dry easterly winds in summer (Barnett 1947; Doepel 1964). It seems likely that these windbreak plants were introduced directly into the north-west of Western Australia from Java and Singapore prior to 1930 (Wise 1930). In 1992, Fusarium wilt was detected in Cavendish plants on 10 of 153 plantations at Carnarvon (Shivas *et al.* 1995). Affected plants were thought to have been predisposed to infection by waterlogging or drought.

The disease is not known to occur at Kununurra in Western Australia or in the Northern Territory (Jones 1991).

Diversity of *Foc* in Australia

Foc is a highly variable pathogen (Ploetz 1990*b*), and several analytical techniques have been used to study variation in Australian populations of *Foc*. These techniques include pathogenicity, vegetative compatibility, volatile production, pectic enzyme analysis, and DNA fingerprinting.

Pathogenicity

Four races of *Foc* are currently recognised based on their pathogenicity to different *Musa* cultivars. Race 1 is pathogenic to Gros Michel and AAB dessert cultivars such as Silk and Pome. Race 2 affects Bluggoe and other closely related ABB cooking banana cultivars. Race 3 affects *Heliconia* spp. with little or no effect on banana, and has been reported from Honduras, Costa Rica, and recently in the Northern Territory of Australia. Race 4 is pathogenic on Cavendish cultivars and all cultivars attacked by races 1 and 2. Races 1, 2, and 4 have been reported in Australia (Brake *et al.* 1990).

When applied to *Foc*, the term 'race' does not imply a defined genetic relationship with the host as it does with other pathosystems. Races of *Foc* are groups of strains which have been observed to be pathogenic to particular host cultivars in the field. It is now evident that pathogenic variation exists within each of the 3 *Foc* races which affect banana. Undoubtedly, other races of *Foc* would be revealed if additional host cultivars were included in the recognised

differential set. Characterising isolates by pathogenicity is complicated by factors that affect host resistance such as host genotype, host age, method of inoculation, and environmental conditions (Brake *et al.* 1995). Pathogenicity tests in the field are expensive and time-consuming but as yet no reliable glasshouse test using small plants has been developed.

Vegetative compatibility

Vegetative compatibility characterises groups of isolates based on the genetic relationships within the fungal populations rather than host–pathogen interaction. Classical Mendelian techniques cannot be used to study genetic diversity in *Foc* as no sexual stage has been found. Vegetative compatibility differentiates isolates that have identical alleles at each of the loci that govern heterokaryon formation and thus vegetative compatibility. These loci are referred to as vegetative incompatibility (*vic*) or heterokaryon (*het*) loci. On the basis of heterokaryon formation, isolates of *Foc* can be divided into genetically distinct groups known as vegetative compatibility groups (VCGs) (Correll *et al.* 1987; Leslie 1990). A technique developed by Puhalla (1985), based on the generation of nitrogen non-utilising (*nit*) mutants, enables heterokaryon formation to be scored macroscopically, making VCG analysis amenable to population studies. VCGs for *Foc* were first reported by Puhalla in 1985. Twenty-one VCGs have now been described for *Foc* (Correll *et al.* 1987; Ploetz and Correll 1988; Moore 1994; Pegg *et al.* 1995). Fifteen VCGs have been found in Asia where *Foc* is thought to have coevolved with *Musa* at 'centres of origin' to generate genetically diverse strains of the pathogen (Ploetz 1990*b*; Moore 1994; Pegg *et al.* 1995). In Asia, VCGs are distributed in distinct areas, with the greatest number of different VCGs found in the Indo-Malaysian region, where the greatest number of subspecies of *M. acuminata* also occurs. VCG analysis has confirmed that some of the strains of *Foc* that occur in Asia have been introduced into Australia with banana planting material (Brake *et al.* 1990; Moore *et al.* 1993). Other pathotypes of *Foc*, some of which pose a potential threat to Australia, have not been moved from Asia to Australia or, as far as can be ascertained, to other production regions of the world.

Australian isolates of *Foc* can be grouped into 7 VCGs, viz. VCGs 0120, 0124, 0125, 0128, 0129, 01211, and 01220 (Brake *et al.* 1990; Moore *et al.* 1993; Pegg *et al.* 1995). With race 1 and race 4 isolates, there has been a complete correlation between pathogenicity and VCG. All race 1 isolates belong in VCGs 0124 and 0125, whereas all race 4 isolates belong in VCGs 0120, 0129, and 01211. Race 1 isolates belonging to VCGs 0124 and 0125 have been recovered from Lady Finger, Gros Michel, Sugar, Mysore and Ducasse (ABB 'Pisang Awak') in northern New South Wales and southern Queensland, but also from Agnes Waters, Yeppoon, Bowen, Ingham, Innisfail, Tully, South Johnstone, Mena Creek, Cairns, Mossman, Mareeba, and Atherton in central and North Queensland.

Isolates belonging in VCGs 0120, 0129, and 01211 were obtained from Cavendish cultivars as well as race 1- and race 2-susceptible cultivars, and are classified as race 4. These are found mainly in the Caboolture–Yandina region, but also occur at Currumbin, Bundaberg, Kandanga, Gunalda, Gympie, Cooran, and Wahpunga in southern Queensland as well as Byron Bay, Eungella, Mullumbimby, and Burringbar in northern New South Wales.

Previously, isolates belonging to VCG 0128 were recovered from Bluggoe and Blue Java (ABB) and other ABB cooking banana cultivars (Brake *et al.* 1990). However, isolates belonging to VCG 0128 were recovered recently from 2 plants of Sugar and one Tuu Gia (AA) plant growing at Kamerunga in North Queensland (Moore 1994). This was unexpected as isolates in VCG 0128 were previously only recovered from race 2-susceptible ABB cooking banana cultivars. Further investigations are required to determine the pathogenicity of isolates in VCG 0128.

An anomaly within the race classification system has been found with a unique population of *Foc* causing wilt in banana plants used as windbreaks and Cavendish cultivars growing under suboptimal conditions at Carnarvon in Western Australia. This population of *Foc* belongs to VCG 01220 (Pegg *et al.* 1995). By definition, any strain of *Foc* attacking Cavendish is regarded as race 4. However, these Carnarvon isolates only caused localised chronic outbreaks of disease in Cavendish plants growing in poorly drained clay soils or, in one case, growing on a dry ridge.

Worldwide, there are at least 21 VCGs of *Foc* which is unusually high compared with other *formae speciales* of *F. oxysporum.* The number of VCGs is greatest in Asia where it is believed that *Foc* arose (Moore 1994). Continuing analysis of *Foc* populations from Asia will enhance the understanding of pathogenic and genetic variation in the pathogen and assist in producing banana cultivars with durable resistance. Such research is also essential to enable the commercial development of banana in that region. The presence of strains of *Foc* (VCGs 01213 and 01216) causing the collapse of Cavendish plantations in Indonesia and Malaysia is of concern. The introduction of these strains into Australia and other world production areas would likely precipitate a new 'Gros Michel era' of devastating plant losses. Fortunately, Australia is an island continent, and *Foc*, being soil-borne, can only spread very slowly around the world through transport of diseased planting material. Now that plantlets produced by tissue culture are available for major cultivars, there should be no need to move rhizomes locally, nationally, or internationally from infested areas.

Volatile production

Brandes (1919) found that isolates of *Foc* grown on steamed rice and different liquid media either produced or did not produce a characteristic volatile odour. Isolates from Panama, Costa Rica, and Jamaica produced 'aromatic aldehydes', whereas those from Cuba did not produce these compounds and were designated as 'var. *inodoratum*'. Stover (1962*b*) also used volatile compounds to differentiate strains of this pathogen. Isolates of *Foc* were cultured on a starch substrate of sterilised, steamed rice and the gases in the headspace above the cultures were analysed using gas chromatography. Stover assigned isolates to either the 'odoratum' or 'inodoratum' group based on the presence or absence of volatile substances. This technique has been used to characterise Australian and Asian isolates of *Foc* (Moore *et al.* 1991; Pegg *et al.* 1993). These studies indicated that the production of volatile compounds on rice medium could be used to differentiate between strains of *Foc.* There was absolute correlation between the production of volatile substances, VCG, and pathogenicity in the Australian isolates; race 1 and race 2 isolates did not produce a detectable volatile odour

and gave gas chromatogram profiles with no peaks, while race 4 isolates produced easily detectable volatile odours with characteristic gas chromatogram profiles. Volatile analysis is a simple and inexpensive method of characterising isolates of *Foc* based on the biochemistry of cultures *in vivo*.

Pectic enzyme analysis

Foc isolates from Carnarvon, Western Australia, and from eastern Australia exhibited 2 distinct phenotypes when assayed for pectic enzyme activity on citrus pectin gels (Pegg *et al.* 1995). Race 4 isolates from eastern Australia belonged in one zymogram group and were characterised by a slow-moving smeared band which indicated polygalacturonase activity. Race 1 isolates from Queensland and Carnarvon isolates produced a fast-moving smeared band which also indicated polygalacturonase activity.

DNA fingerprinting

Although vegetative compatibility is a useful means of characterising *Foc* into genetically isolated groups, it may not be a true indication of the genetic relatedness between different isolates. Isolates within a VCG are thought to be genetically similar as they have identical alleles at each of the *vic* loci; however, a mutation at a single *vic* locus could result in closely related isolates becoming vegetatively incompatible, or, although unlikely, it is also possible that distantly related isolates could have the same *vic* loci and be vegetatively compatible even though they are not genetically similar. Arbitrary primer techniques (RAPD-PCR and DAF) have been used to generate genome-specific DNA fingerprints to further characterise isolates of *Foc* (Sorensen 1993; Bassam and Bentley 1994, 1994; Bentley *et al.* 1995). Using these DNA fingerprinting techniques, it is possible to determine the genetic similarity between isolates within each VCG and the genetic relatedness among the VCGs. Molecular analysis is also useful for characterising isolates that do not belong to known VCGs. Comparison of the DNA fingerprints generated for *Foc* has subdivided isolates from a worldwide collection into 2 major groups (Bentley *et al.* 1995; S. Bentley unpublished data). Group 1 contains isolates belonging in VCGs 0120, 0121, 0122, 0126, 0129, 01210, 01211, 01213, 01215, 01216, and 01219; Group 2 contains isolates belonging in VCGs 0123, 0124, 0125, 0128, 01212, 01214, 01217, 01218, and 01220. Isolates within each VCG generally produce a very similar banding pattern and are closely related, independent of geographical origin or host source.

The 2 groups within *Foc* differentiated by DNA fingerprinting analysis correspond with the 'odoratum' and 'inodoratum' groups described by Moore *et al.* (1991). Group 1 contains all 'odoratum' isolates and Group 2 contains all isolates belonging to the 'inodoratum' group. There was good, but not absolute, correlation between the groups determined by DNA fingerprinting and the classification of isolates based on electrophoretic karyotype (EK) variation described by Boehm *et al.* (1994).

Among Australian isolates of *Foc* there was direct correlation between DNA fingerprint group and race; Group 1 included all race 4 isolates (VCGs 0120, 0129, 01211) and Group 2 included all race 1 isolates (VCGs 0124, 0125) (Sorensen 1993). Isolates in VCG 0128 also belonged to Group 2. The genetic similarity between the 2 groups was 30% based on their DNA fingerprints. Molecular

analysis of the Carnarvon isolates (VCG 01220) indicated that these isolates were more similar (genetic similarity of 80%) to the race 1 isolates, which further supports the proposal that these isolates are only capable of attacking Cavendish under suboptimal growing conditions (Sorensen 1993; Pegg *et al.* 1995).

Information derived from these studies should be useful for developing a PCR-based detection system for the organism in soil and plant tissue. Such a detection system would be useful for assessing fields for the detection and race identification of *Foc* prior to planting, screening rhizomes or suckers used for planting material, and characterising isolates of *Foc* directly from infested soil or infected plant tissue.

The genetic relationships between the VCGs determined using arbitrary primer technology also provide insight into the evolution of *Foc.* The subdivision of isolates of *Foc* into 2 groups based on their DNA fingerprint patterns suggests a biphyletic origin for the pathogen which supports the hypothesis that the pathogen coevolved with edible banana cultivars and their wild diploid progenitors in South East Asia (Sorensen 1993; Bentley *et al.* 1995).

Race 4 in Cavendish cultivars

Since the mid 1950*s*, Cavendish cultivars have remained the basis of the world export industries which are situated in tropical areas of Central and South America, the Caribbean, West Africa, and the Philippines. Cavendish cultivars are also grown for domestic consumption in countries such as Australia, China, Vietnam, India, Pakistan, Egypt, and South Africa. Recently, plantations of Cavendish cultivars in subtropical countries such as Taiwan, Canary Islands, Australia, and South Africa have been attacked increasingly by strains of *Foc.* It is thought that Cavendish plants growing in subtropical environments are predisposed to systemic infection by certain strains of *Foc* by low temperatures during winter. Moore *et al.* (1993) and Moore (1994) studied the influence of winter temperatures on the Cavendish cultivars Dwarf Parfitt and Williams in Australia. Dwarf Parfitt has high resistance to Australian race 4 populations of *Foc.* It was found that Dwarf Parfitt maintains a higher chlorophyll concentration and more effective photosynthetic activity during winter than Williams. Host defence reactions, such as the formation of gums, gels, tyloses and other xylem-occluding products used to block the invading pathogen, are presumably driven by photoassimilates, either from storage or current photosynthesis. It was proposed that cold-induced stress disrupts photoassimilation mechanisms thus compromising the resistance of Williams to race 4 populations of *Foc* (Moore *et al.* 1993; Moore 1994). Further studies are required to test this hypothesis.

Strains of *Foc* capable of affecting Cavendish cultivars growing under tropical conditions have been recently identified. Cavendish cultivars such as Valery, Grande Naine and Williams grown for export in Indonesia and Malaysia have been seriously affected by Fusarium wilt. Although Cavendish growing in the tropical areas of Latin America–Caribbean region succumb only occasionally, the world export industries may once again be threatened by Fusarium wilt.

Control

Initially, control of Fusarium wilt in Central America was aimed at reducing the pathogen population in infested fields. However, fungicides, fumigants, flood

fallowing, crop rotation, and organic amendments have rarely provided long-term control in any production area. Quarantine and exclusion procedures are effective in controlling the spread of the disease by restricting the movement of corms, suckers, and soil that could carry *Foc* from infested to non-infested areas. In eastern Australia, legislation provided by the Queensland Banana Industry Protection Act, which operates in association with the Plant Protection Act 1989 and the New South Wales Banana Industry Act 1987, prohibits the transfer of planting material from certain localities where the disease is prevalent or from any plantation not approved as a source of planting material. Planting material is only approved if the source plantation has no previous record of the occurrence of the disease and it is apparently free from Fusarium wilt when subjected to row-by-row inspection by trained personnel in the autumn and spring preceding the removal of the material. However, due to the presence of suppressive soils in which microbial populations suppress the pathogen population, and since infected rhizomes or suckers may not exhibit external symptoms, the pathogen may still be moved in approved planting material. In recent years, the use of micropropagated planting material has been encouraged. If managed correctly, such material should be free of *Foc*.

It is now generally accepted that the most effective means of controlling Fusarium wilt is by host resistance. Since banana is a perennial crop, resistance must be durable. Natural sources of resistance exist in wild species and cultivars of banana and in synthetic diploids developed by breeding programs. Banana breeding for Fusarium wilt resistance began in 1922 at the Imperial College of Tropical Agriculture in Trinidad and in 1924 in Jamaica (Stover and Buddenhagen 1986). There are now 4 major conventional banana breeding programs and these are located in Honduras (FHIA), Brazil (EMBRAPA-CNPMF), Nigeria (IITA), and Guadeloupe (CIRAD-FLHOR). These programs have concentrated on using resistance in Pisang Jari Buaya (AA), Pisang Lilin (AA) and *Musa acuminata* ssp. *burmannicoides* (Calcutta IR 124) (AA_w). Although it has not yet been possible to breed a Cavendish replacement because of fertility constraints, it seems that useful tetraploid hybrids can be bred to replace AAB dessert and ABB cooking banana types. For example, FHIA-01 (Goldfinger), a primary tetraploid dessert banana from the FHIA breeding program in Honduras with an acidic or 'apple' flavour, has been identified as having resistance to race 1 and race 4 of *Foc*. However, Stover and Buddenhagen (1986) have warned against using tetraploid hybrids derived from susceptible triploid females as 75% of the genome is derived from the disease-susceptible female parent. Stover and Buddenhagen (1986) questioned the durability of such resistance and advocated an alternative method of genetic improvement by developing novel triploid clones from diploid stocks to overcome this problem. This approach is being attempted by the CIRAD-FLHOR group in Guadeloupe. Biotechnology, mutation breeding, and somaclonal variations are also being used to produce resistant genotypes. These programs are likely to provide replacement clones in the future. Since Australia does not have a banana breeding program, close collaboration with international breeding and plant improvement programs is essential for obtaining improved germplasm.

Conclusion

Cavendish cultivars are resistant to race 1 in Australia, but strains belonging to this race are still causing appreciable losses in Lady Finger plantations.

However, the success of Cavendish cultivars has resulted in a production system with an extremely narrow genetic base. As in any monoculture, this system is highly vulnerable to new pests and diseases. Race 4 strains capable of attacking Cavendish as well as Lady Finger cultivars are currently confined to south-eastern Queensland and northern New South Wales. Quarantine restrictions have been imposed to prevent these strains being introduced into the major Cavendish-producing areas of North Queensland, the Northern Territory, and Western Australia. If race 4 were introduced into these areas, the Australian banana industry would be in serious difficulties as no suitable replacements for AAA dessert cultivars (Cavendish, Gros Michel) are available. Over 70 years of banana breeding has failed to provide a hybrid which matches the agronomic, fruit quality, and marketing attributes of Cavendish or Gros Michel.

Since disease control is based on quarantine and host resistance, an understanding of pathogen variability is essential. Results from Australian research presented in this review have demonstrated the importance of understanding genetic and pathogenic diversity among populations of Australian pathogens relative to global diversity. This research has demonstrated the importance of using several techniques to characterise isolates of *Foc.* Vegetative compatibility, volatile production, pectic enzyme analysis, and DNA fingerprinting proved to be reliable *in vitro* techniques for race classification of Australian isolates of *Foc.* A future challenge is to determine whether such *in vitro* tests can be used to group isolates of *Foc* from countries other than Australia according to pathogenicity. Investigating the genetic variation within *Foc* should also provide insights into the evolutionary origins of different populations and pathotypes which may benefit research into mechanisms of plant resistance.

There is an urgent need to develop a rapid and reliable small-plant test to provide information on pathogenic variability and host resistance. Field evaluation of cultivars and new hybrids is very expensive and requires large areas of land. Also, the pathogen may not be distributed uniformly and more than one pathogen genotype may be present. However, the development of such a test appears remote due to the current lack of understanding of host resistance mechanisms and how they are mediated. Defence mechanisms, such as the formation of tyloses and the infusion of phenolic compounds, require energy to operate efficiently. Environmental factors such as low temperature or waterlogging may reduce the energy efficiency of the roots and thus suppress the normal host responses to infection. Clarification should come from stress physiology research.

It is now generally accepted that conventional breeding approaches to improve Cavendish may not be feasible and biotechnology will be required. With the proximity of Australia to the centre of origin of *Musa* and its coevolved pests and pathogens in Asia, and with its large stable banana production base and research expertise, a breeding program based in Australia and linked to biotechnology is highly desirable and would undoubtedly yield useful disease resistant cultivars. Meanwhile, Australia needs to maintain its links with international breeding programs and could assist these programs by evaluating their banana parents and progenies for resistance to Fusarium wilt. No one since Vakili (1965) has screened wild, seeded diploid bananas against known populations of *Foc.* This may be critical now that populations of *Foc* have been identified which threaten the production of Cavendish cultivars in the tropics.

References

Ashby, S. F. (1913). Banana diseases in Jamaica. *Bulletin of the Department of Agriculture Jamaica* **2**, 95.

Bancroft, J. (1876). Report of the board appointed to enquire into the cause of disease affecting livestock and plants. Queensland, 1876. *Votes and Proceedings 1877* **3**, 1011–38.

Barnett, G. B. (1947). Banana culture in Western Australia. *Western Australian Journal of Agriculture* **24**, 79–139.

Bassam, B. J., and Bentley, S. (1994). DNA fingerprinting using Arbitrary Primer Technology (APT): a tool or torment? *Australasian Biotechnology* **4**, 232–6.

Bentley, S., Pegg, K. G., and Dale, J. L. (1994). Optimization of RAPD-PCR fingerprinting to analyze genetic variation within populations of *Fusarium oxysporum* f.sp. *cubense*. *Journal of Phytopathology* **142**, 64–78.

Bentley, S., Pegg, K. G., and Dale, J. L. (1995). Genetic variation among a world-wide collection of isolates of *Fusarium oxysporum* f.sp. *cubense* analyzed by RAPD-PCR fingerprinting. *Mycological Research* **99**, 1378–84.

Boehm, E. W. A., Ploetz, R. C., and Kistler, H. C. (1994). Statistical analysis of electrophoretic karyotype variation among vegetative compatibility groups of *Fusarium oxysporum* f.sp. *cubense*. *Molecular Plant–Microbe Interactions* **7**, 196–207.

Brake, V. M., Pegg, K. G., Irwin, J. A. G., and Langdon, P. W. (1990). Vegetative compatibility groups within Australian populations of *Fusarium oxysporum* f.sp. *cubense*, the cause of Fusarium wilt of bananas. *Australian Journal of Agricultural Research* **41**, 863–70.

Brake, V. M., Pegg, K. G., Irwin, J. A. G., and Chaseling, J. (1995). The influence of temperature, inoculum level and race of *Fusarium oxysporum* f.sp. *cubense* on the disease reaction of the banana cv. Cavendish. *Australian Journal of Agricultural Research* **46**, 673–85.

Brandes, E. W. (1919). Banana wilt. *Phytopathology* **9**, 339–83.

Carefoot, G. L., and Sprott, E. R. (1969) 'Famine on the Wind.' (Angus and Robertson: London.) 222 pp.

Correll, J. C., Klittich, C. J. R., and Leslie, J. F. (1987). Nitrate non-utilising mutants of *Fusarium oxysporum* and their use in vegetative compatibility tests. *Phytopathology* **77**, 1640–6.

Doepel, R. F. (1964). A revised list of fruit diseases recorded in Western Australia. *Western Australian Journal of Agriculture* **5**, 449–56.

Drost, A. W. (1912). The suriname Panama disease of the Gros Michel banana. Translation by S. F. Ashby. *Bulletin of the Department of Agriculture, Jamaica* **2**, 1913.

Jones, D. R. (1991). Status of banana diseases in Australia. *In* 'Banana Diseases in Asia and the Pacific: Proceedings of a Technical Meeting on Diseases affecting Banana and Plantain in Asia and the Pacific'. Brisbane, Australia. pp. 21–37.

Leslie, J. F. (1990). Genetic exchange within sexual and asexual populations of the genus *Fusarium*. *In* 'Fusarium Wilt of Banana.' (Ed. R. C. Ploetz.) pp. 37–48. (APS Press: St Paul, MN.)

Magee, C. J. (1947). Root diseases of the banana. *Agricultural Gazette of New South Wales* **58**, 419–22.

Mayers, P. E. (1983). Fusarium wilt of Cavendish bananas in Queensland. (Abstract.) Proceedings International Fusarium Workshop Australia **5**, 60. (Ed. C. Liddell.) (The University of Sydney.)

Moore, N. Y. (1994). Fusarium wilt of banana: pathogen variability and host pathogen interaction. Ph.D. Thesis, The University of Queensland, Brisbane, Australia.

Moore, N. Y., Hargreaves, P. A., Pegg, K. G., and Irwin, J. A. G. (1991). Characterisation of strains of *Fusarium oxysporum* f.sp. *cubense* by production of volatiles. *Australian Journal of Botany* **39**, 161–6.

Moore, N. Y., Pegg, K. G., Allen, R. N., and Irwin, J. A. G. (1993). Vegetative compatibility and distribution of *Fusarium oxysporum* f.sp. *cubense* in Australia. *Australian Journal of Experimental Agriculture* **33**, 797–802.

Nicholson, M. E. (1969). Clonal testing for Panama. *In* 'Papers presented to Pacific-Asian Banana Culture Interchange, Australia'.

Pegg, K. G., Moore, N. Y., and Sorensen, S. (1993). Fusarium wilt in the Asian Pacific region. *In* 'Proceedings International Symposium on Recent Developments in Banana Cultivation Technology'. (Eds R. V. Valmayor, S. C. Hwang, R. Ploetz, S. W. Lee, and V. N. Roa.) pp. 225–69. (INIBAP/ASPNET: Laguna, Philippines.)

Pegg, K. G., Shivas, R. G., Moore, N. Y., and Bentley, S. (1995). Characterisation of a unique population of *Fusarium oxysporum* f.sp. *cubense* causing Fusarium wilt in Cavendish bananas at Carnarvon in Western Australia. *Australian Journal of Agricultural Research* **46**, 167–78.

Ploetz, R. C. (Ed.) (1990*a*). 'Fusarium Wilt of Banana.' (APS Press: St Paul, MN.) 140 pp.

Ploetz, R. C. (1990*b*). Population biology of *Fusarium oxysporum* f.sp. *cubense*. *In* 'Fusarium Wilt of Banana'. (Ed. R. C. Ploetz.) pp. 63–7. (APS Press: St Paul, MN.)

Ploetz, R. C., and Correll, J. C. (1988). Vegetative compatibility among races of *Fusarium oxysporum* f.sp. *cubense*. *Plant Disease* **72**, 325–8.

Ploetz, R. C., Herbert, J., Sebasigari, K., Herndandez, J. H., Pegg, K. G., Ventura, J. A., and Mayato, L. S. (1990). Importance of Fusarium wilt in different banana-growing regions. *In* 'Fusarium Wilt of Banana'. (Ed R. C. Ploetz.) (APS Press: St Paul, MN.)

Puhalla, J. E. (1985). Classification of strains of *Fusarium oxysporum* on the basis of vegetative compatibility. *Canadian Journal of Botany* **63**, 179–83.

Purss, G. S. (1953). A disease in Williams hybrid bananas produced by *Fusarium* sp. *Queensland Journal of Agricultural Science* **10**, 126.

Rorer, J. B. (1911). A preliminary list of Trinidad fungi. *Board of Agriculture, Trinidad and Tobago, Circular* **4**, 37–44.

Shivas, R. G., Wood, P. M., Darcy, M. W., and Pegg, K. G. (1995). First record of *Fusarium oxysporum* f.sp. *cubense* on Cavendish bananas in Western Australia. *Australasian Plant Pathology* **24**, 38–43.

Simmonds, J. H. (1947). Report of the Plant Pathology Section. Report Department of Agriculture, Queensland, 1946–47, pp. 33–5.

Simmonds, N. W. (1966). 'Bananas.' (Longmans: London.) 512 pp.

Simmonds, N. W., and Shepherd, K. (1955). The taxonomy and origins of the cultivated bananas. *Journal of the Linnaean Society (Botany)* **55**, 302–12.

Smith, E. F. (1910). A Cuban banana disease. *Science* **31**, 745.

Snyder, W. C., and Hansen, H. W. (1940). The species concept in Fusarium. *American Journal of Botany* **27**, 64–7.

Sorensen, S. (1993). Genetic variation within *Fusarium oxysporum* f.sp. *cubense* in banana. Ph.D. Thesis, Queensland University of Technology, Brisbane, Australia.

Stover, R. H. (1962*a*). 'Fusarial Wilt (Panama Disease) of Bananas and other Musa Species.' (Commonwealth Mycological Institute: Kew, UK.) 117 pp.

Stover, R. H. (1962*b*). Studies on Fusarium wilt of bananas VIII-Differentiation of clones by cultural interaction and volatile substances. *Canadian Journal of Botany* **40**, 1467–71.

Stover, R. H. (1986). Disease management strategies and the survival of the banana industry. *Annual Review of Phytopathology* **24**, 83–91.

Stover, R. H. (1990). Fusarium wilt of banana: some history and current status of the disease. *In* 'Fusarium Wilt of Banana'. (Ed. R. C. Ploetz.) pp. 1–7. (APS Press: St Paul, MN.)

Stover, R. H., and Buddenhagen, I. W. (1986). Banana breeding; polyploidy, disease resistance and productivity. *Fruits* **41**, 175–91.

Stover, R. H., and Malo, S. E. (1972). The occurrence of fusarial wilt in normally resistant 'Dwarf Cavendish' banana. *Plant Disease Reporter* **56**, 1000–3.

Turner, D. W., and Hunt, N. (1984). Growth, yield and leaf nutrient composition of 30 banana varieties in subtropical New South Wales. In Department of Agriculture New South Wales Technical Bulletin **31**, 36 pp.

Tyron, H. (1912). Natural enemies of the banana occurring in Queensland. *Queensland Agricultural Journal* **28**, 116–19.

Vakili, N. G. (1965). Fusarium wilt resistance in seedlings and mature plants of *Musa* species. *Phytopathology* **55**, 135–40.

Waite, B. H. (1977). Inoculation studies and natural infection of banana varieties with races 1 and 2 of *Fusarium oxysporum* f.sp. *cubense*. *Plant Disease Reporter* **61**, 15–19.

Watson, B. (1988). Chinese farmers of the north. *In* 'Harvests and Heartaches'. (Eds T. Barker and I. Byford.) pp. 94–5. (Department of Primary Industries: Queensland.)
Wise, F. J. S. (1930). The banana in Western Australia. *Western Australian Journal of Agriculture* **7**, 560–71.

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