Fusarium wilt: a threat to banana cultivation and its management

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Received: 26 September 2018
Accepted: 27 November 2019

doi: 10.1079/PAVSNNR202015004

The electronic version of this article is the definitive one. It is located here: http://www.cabi.org/cabreviews

Abstract

Banana is affected by a wide number of diseases, of which, Fusarium wilt caused by *Fusarium oxysporum* f. sp. *cubense* (*Foc*) race 1 has played a major role in devastating Gros Michel banana plantations. Since 1960s, the pathogen *Foc* race 4 has threatened the survival and existence of the Cavendish group of bananas, which has necessitated detailed study on Fusarium wilt, the causal organism *Foc*, its biology, dispersal, pathogenicity, diversity and detection at a molecular level (especially in soils) and its management. The recently developed technique of transferring the gene encoding green fluorescent protein into *Foc* has assisted in visualizing and analysing the colonization and infection of banana plants by the pathogen. Studies on the pathogenicity secreted in xylem genes have helped in rapid detection of the pathogen *in planta* and techniques such as real-time fluorescence loop-mediated isothermal amplification assay have facilitated rapid and direct quantitative detection of *Foc* in soil. Several management practices, especially resistant varieties/ transgenics and biological control methods are available for the effective management of this deadly disease. Strict quarantine procedures and reduction of *Foc* inoculum are the methods undertaken to limit the spread of the disease to other un-infected regions. This review summarizes the recent developments of Fusarium wilt in banana and its management.

Keywords: Banana, Fusarium wilt, Genetic diversity, Management, Biocontrol

Review Methodology: Information for the review has been compiled from journal articles, text books, proceedings, reports etc. and to access these, ICAR-CeRA J gate, hard copy of text books (for an instance D R Jones, 2018. Handbook of Diseases of Banana, Abaca and Enset, CABI, p633) were referred. Information for the review has also been compiled from CAB Abstracts, CAB Heritage and e-Journals, Science Direct, Research Gate. In addition we used the references from the articles obtained by this method to check for additional relevant material. We also spoke to colleagues and checked for any upcoming studies not yet published.

Introduction

Banana (*Musa* sp.) is a perennial monocotyledonous herb plant belonging to the order Zingiberales. Its fruits are consumed worldwide, as dessert and cooked (plantain) forms. The fruits of edible bananas are diploid and triploid seedless parthenocarpic hybrids derived from intra- or inter-specific crosses between two diploid wild *Musa* species, *Musa acuminata* (AA) and *M. balbisiana* (BB) [1, 2]. The most common varieties of dessert are triploid AAA derived from crosses within *M. acuminata*, while common cooking triploid bananas (AAB or ABB) are derived from crosses between *M. acuminata* and *M. balbisiana*. Bananas are vital for food security in many tropical and subtropical countries and half of the banana production relies on somaclones derived from a single triploid genotype, Cavendish [3]. In 2015, global banana exports, excluding plantains, registered the first decline since 2010 after having reached an unprecedented peak of 18.6 million tonnes in 2014 [4]. Export of banana from Asia has also declined by 46% as a production drop occurred, mainly due to Fusarium wilt in the Philippines (which is the largest exporter in the region, accounting for 90% of the total export from Asia). As Fusarium wilt is becoming a major threat to banana cultivation worldwide and will be the major impediment for the export of banana in the future, the current review focuses on different aspects on status, diagnostics and management.

Figure 1 Fusarium wilt tropical race 4 devastated banana field at Katihar, Bihar, India.

Pathogen

Fusarium wilt of banana is caused by the fungus *Fusarium oxysporum* Schlechtend.: Fr. f. sp. *cubense* (*Foc*) (E.F. Smith) Snyder and Hansen [5]. It is also known as the Panama disease [6] and is poly-cyclic in nature. [7]. The diverse means of dispersal and long-term survival in infested soil could be the reason for *Foc* being the most widely distributed pathogen. There are four recognized races (race 1 to race 4) of this fungus, which are separated based on reaction on differential hosts. Race 1 causes disease in the Gros Michel (AAA), Silk (AAB), Lady Finger (AAB), Maqueno (Maia Maoli-Popoulu subgroup, AAB), Pome (AAB) and Pisang Awak (ABB) cultivars, race 2 attacks monthan, bluggoe and other closely related cooking bananas and also affects some bred tetraploids (Bodles altafort hybrid between Gros Michel and Pisang lilin) and enset (*Ensete ventricosum*) and race 4 infects Cavendish (AAA) group of bananas and also cultivars susceptible to races 1 and 2. Race 3 is not considered to be a pathogen of banana, as it only attacks *Heliconia* spp. (tropical American banana relatives). Race 4 is further divided into subtropical and tropical strains. Tropical race 4 (*Foc* TR4) is a more virulent form of the pathogen and is capable of causing disease in Cavendish grown under any conditions, whereas subtropical race 4 (*Foc* STR4) generally causes disease only in plants grown under abiotic stress, especially in cold weather [8].

Occurrence and Losses

Races 1 and 2 of *Foc* are distributed worldwide [5, 8, 9]. *Foc* STR4 is reported in Taiwan, Canary Islands, South Africa and southern Brazil [8, 9]. The infection of *Foc* TR4 was first reported in Taiwan [10] in Cavendish cultivar, affecting nearly 1200 ha of banana plantations. Later, *Foc* TR4 has been reported to cause severe damage to Cavendish cultivars in Malaysia, Indonesia, South China, Philippines, Northern Territory of Australia [11–13], Mozambique [14], Jordan [15], Lebanon and Pakistan [16]. In 2017, *Foc* TR4 was also reported in Laos [17], Vietnam [18] and Myanmar [19]. In India, its presence was reported in Bihar (Katihar and Purnea districts) [20] (Fig. 1) and Faizabad district in Uttar Pradesh [21]. Reports of *Foc* infecting Cavendish in different parts of the world are summarized in Table 1 and Fig. 2. The lack of banana diversity and the difficulties in the banana breeding process have raised serious distress that poses the threat of disappearance of banana from the shops.

Fusarium wilt epidemics in the twentieth century resulted in the devastation of more than 50 000 ha of exotic Gros Michel (AAA) plantations [8], which led to a major shift of the entire banana production to race 1-resistant Cavendish (AAA) banana varieties such as Williams, Grand Naine and Dwarf Cavendish. Between 1940 and 1960, the loss caused by *Foc* race 1 in Gros Michel variety was estimated to be US$2.3 billion [38] to the export companies alone. In 2013, the financial losses due to *Foc* TR4 infection has been estimated as high as US$121 million in Indonesia, US$253 million in Taiwan, US$14 million in Malaysia and more than US$7.5 million in northern Mozambique (www.rtb.cgiar.org). Earlier, it was stated that the occurrence of *Foc* TR4 in Latin America would cause huge losses to the banana industry, as that region along with Caribbean contributes to 80% banana export [39]. Unfortunately, the recent report in 2019 says it has spread to Colombia (Latin America), which is a major concern for the banana industries involved in export (https://www.foodnavigator-latam.com/Article/2019/08/09/Colombia-declares-national-emergency-as-TR4-banana-disease-confirmed). In banana, Cavendish cultivars comprised of 15% of the global banana production and occupied 40% of the total global area [7]. Clearly, this implied a huge risk for a pandemic outbreak of *Foc* TR4 as Cavendish clones are susceptible to the strain. The vegetative propagation of

Table 1 Chronological occurrence of Fusarium wilt disease in the Cavendish group of banana across the globe

Year	Country	Remark	Reference
1967	Taiwan	Symptoms of Fusarium wilt on Cavendish cultivar at Chiatung in South Taiwan were observed. Initially, it was observed in 0.27 ha and later it has spread to 1200 ha in 1976. In 1989, VCG 01213 of TR4 was identified	[22–24]
1970s	Philippines	About 30 000 plants of Grand Naine were eradicated due to *Foc* wilt between 1974 and 1991, and this was mainly caused by STR4 (VCGs 0122, 0123 and 0126) Samples collected from highland and lowland Cavendish banana farms in Davao in Sep 2005 confirmed the presence of TR4 (VCG 01213/16)	[25, 26]
1990s	Indonesia and Malaysia	*Foc* TR4 infected thousands of ha of banana and resulted in loss of hundreds of millions of USD. In Lampung district of Sumatra, TR4 caused a loss of 9–11 million USD between 1993 and 2002, and 5000 ha of Cavendish plantations abandoned	[27]
1997–99	Australia	*Foc* STR4 (VCGs 0120, 129, 1211) was reported in Subtropical regions of South Queensland and New South Wales in 1993. *Foc* TR4 (VCG 01213/16) infecting Cavendish was confirmed in Darwin (Northern territory of Australia) 1997	[28–30]
2001	China	The symptoms of *Foc* TR4 was first observed on Cavendish plants in localized areas along the Pearl River Delta in Guangdong Province in South China and later confirmed as VCG 01213/16. In 2006, 6700 ha plantations were severely affected in Guangdong Province	[31–33]
2009	India (*Foc* race 1 –VCG 0124 infecting Cavendish)	The *Foc* infected samples collected from Theni district of Tamil Nadu in 2010 confirmed the presence of *Foc* race 1 in cv. Grand Naine	[34]
2015	India (*Foc* TR4)	Although the *Foc* TR4 believed to be present since 2010, the *Foc* samples collected from Katihar district of Bihar in 2015 confirmed the presence of *Foc* TR4 (VCG 01213/16) in India. This TR4 has spread to the adjoining state Uttar Pradesh and the incidence was more than 50% in severely infected banana growing areas of these two states	[35]
2012–13	Oman (2012), Jordan (2013) and Mozambique (2013)	• TR4 was confirmed in Jordan in 2013, but has probably been present in the country since at least 2005 • *Foc* TR4 in Oman was reported in 2012 • The TR4 occurrence was observed in export banana plantation located in northern Mozambique in Nov. 2013. It was later found in Nampula province also	http://www.sun.ac.za/english/faculty/agri/plant-pathology/ac4tr4/background/global-distribution-of-Foc-Tr4 http://www.promusa.org/Tropical+race+4+-+TR4#footnote19
2012	Pakistan and Lebanon	• In Pakistan, symptoms of Fusarium wilt were first observed in 2012 in a 2 ha Cavendish plantation in Baoo Pooran of Sindh province and in 2014, approximately 121 ha were affected. Later, the samples collected were confirmed as *Foc* TR4 • In Lebanon, during 2013, one ha area of Grand Naine plantation located at Mansouri and Berghliyeh regions was affected by Fusarium wilt and the pathogen was confirmed as *Foc* TR4	[16]
2016	Israel	The incidence of Fusarium wilt TR4 was observed in cv. Grand Naine has grown in Shfeya (Camel coastal plain) and Kibbutz Ein Gev (Eastern Shore of Lake Galilee)	[36]

Table 1 (Continued)

Year	Country	Remark	Reference
2014–17	Vietnam, Myanmar and Laos	The symptoms of Fusarium wilt TR4 was observed in three provinces of Northern Vietnam along the Red River that originates in the Yunnan province of China i.e. Hanoi (2014), Hung Yen (2015) and Lao Cai (2015). Later in 2016, the samples collected from Laos, Vietnam and Myanmar confirmed as *Foc* TR4 (VCG 01213/16)	[18, 19, 37]
2019	Colombia	The incidence of TR4 was reported in La Guajira and declared the presence of TR4 officially on 8th August 2019	https://www.foodnavigator-latam.com/Article/2019/08/09/Colombia-declares-national-emergency-as-TR4-banana-disease-confirmed

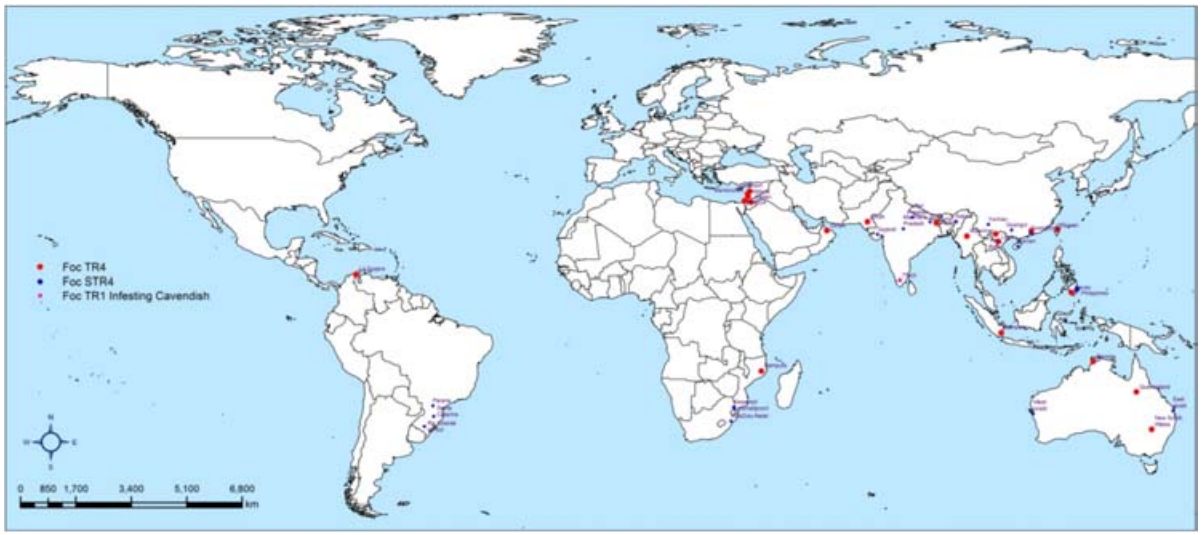

Figure 2 Occurrence of Fusarium wilt (TR4, STR4 and race 1) in Cavendish group of bananas in different banana growing countries of the world.

planting material and a lack of diversification efforts over the last century have increased the genetic vulnerability of the crop to unacceptable levels, which could threaten food security. Also there is no known substitute to the commercial Cavendish cultivars yet developed. Thus, there is a need for international, regional and local measures aimed at prevention and management of this destructive disease.

Symptoms

Development of a reddish-brown discoloration of the xylem in fine or smaller non-woody feeder roots at the sites of infection and yellowing of lower older leaves are the initial symptoms. Leaf yellowing begins along the margin and advances towards the midrib. Subsequently, the petiole turns brown and buckles. Infected plants frequently develop longitudinal splits on the pseudostem just above the soil level. The typical external symptom is hanging of dead leaves around the pseudostem, which appears like a skirt. Eventually, the heart leaf withers and the pseudostem remains standing until it is removed or collapsed [5, 40, 41]. Infection is also passed into young suckers [22]. Cross-section of the corm and pseudostem shows purplish-brown discoloration of the vascular bundles while in corm, the discoloration appears as a collection of tiny reddish or brownish dots and streaks [42]. The discoloration of the rhizome is severe where the stele joins the cortex [5]. Generally, infected plants produce no fruit bunches and if produced, the fruits are very small with few fingers having pithy and acidic flesh [43].

Mechanism of Symptom Expression

In the presence of banana roots, the chlamydospores or conidia of the fungal pathogen germinate and infect the lateral or feeder roots of banana plants [44]. The pathogen colonizes and blocks the plant vascular system [8] thereby inhibiting water and nutrient transport to the leaves and also damaging the chloroplast, and thus leading to wilting. The chloroplast damage leads to expression of leaf yellowing. The fungus produces fusaric acid and beauvericin toxins. Normally, fusaric acid (at 17.9 mg/ml) is considered to be enough for initiating leaf chlorosis symptom [45], whereas beauvericin which inhibits cholesterol acyltransferase, induces typical programmed cell death (PCD) and pore formation in cellular membranes [46]. Both beauvericin and fusaric acid are found to be detected in all the infected tissues of banana including pseudostems, fruit and leaves [47]. Besides, the fungus also secretes a mixture of hydrolytic enzymes, including cutinases, cellulolytic enzymes (cellulases, hemicellulases and xylanase), pectinases (pectin methylesterase, polygalacturonase and pectate lyase) and proteases [48] which facilitate penetration of pathogen into the plant cell wall. Unlike in susceptible cultivars, resistant cultivars respond to the initial pathogen attack by quick occlusion of the xylem lumens with tyloses (large bladder-like cells), gels and gums, hindering the pathogen from further entering the plant.

Biology, Survival and Dispersal

The fungus initially enters into the epidermal cells and intercellular spaces of the banana root thereafter it forms numerous microconidia and macroconidia and finally chlamydospores when the plants are dead. Microconidia are either one- or two-celled, oval- to kidney-shaped and are produced in false heads. Macroconidia are four- to eight-celled and sickle-shaped with foot-shaped basal cells. Chlamydospores are usually globose and are formed singly or in pairs in hyphae or conidia [49]. The fungus moves through the conducting vessels acropetally along with the xylem sap by alternating between the sporulating and germinating phase to penetrate the barriers. After the plant wilts completely, the fungus feeds saprophytically on the dead plant parts such as leaves, pseudostem and roots, and produces numerous resting spores called chlamydospores, which are resistant to desiccation and unfavourable environmental conditions and enable to remain viable in the soil and on plant debris for more than four decades [38, 42]. The germination rate of chlamydospores and Fusarium wilt development depends on the soil topography and rhizosphere microflora [50, 51]. These spores could be transported to disease-free soils by wind, run-off waters and inadvertent dispersal *via* birds, animals, humans and even farm implements [5, 52]. The spread of the pathogen locally, nationally and internationally is through root-to-root contact, infected planting materials (rhizomes or suckers) and also through soil attached to planting materials, farm implements, vehicles, footwear and unsterilized potting compost [53]. Dispersal could also be effected through soil adhering to other crops planting materials. Aerial dissemination of *Foc* might also be possible since macroconidia or sporodochia of the pathogen were produced on artificially inoculated plants in greenhouse experiments [54].

The pathogen survives in the roots of several species of common grasses and weed species such as *Commelina diffusa*, *Chloris inflate*/*C. barbata*, *Ensete ventricosum*, *Euphorbia heterophylla*, *Tridax procumbens*, *Cyanthillium cinereum*, *Paspalum*, *Panicum* and *Ixophorus* which do not express the symptom of the disease [55–57]. Environmental factors, especially the edaphic factors such as poor soil drainage and unfavourable chemical or physical or biological conditions play a role in the predisposition of the host to the disease [58, 59]. Soil moisture content at less than field capacity (0.01 MPa) is favourable to Fusarium wilt development [60]. Also, temperature plays a major role in the progress of *Foc* invasion and symptom development in banana [61] as infection and establishment of *Foc* TR4 in the Cavendish variety takes place at 15 °C or below during winter in the subtropics. High disease severity of Fusarium wilt of banana occurred at pH 8 is also noticed [51]. During heavy rainfall, spores of the pathogen and infected tissues on the ground are carried in surface drainage water. Survival of Indian strain of *Foc* under water stagnation for a month is also reported [62]. The texture and organic matter content of the soil significantly influenced the survival of the pathogen as wilt disease has been recorded from loose soil to heavy clay soil with the pH range of 4.80–8.45 and EC range from 0.12 to 1.10/dsm [63]. Banana weevil, *Cosmopolites sordidus*, seems to be a vector or predisposing agent of *Foc* as *Foc* TR4 was detected on exoskeleton [64]. Co-infection of nematode *Radopholus similis* and *Foc* in cv. Gros Michel is reported, however, there was no influence on disease severity [65].

Colonization

Colonization of the host plant by *Foc* is a complex process which requires a series of highly regulated processes such as recognition of host roots by a signalling process, adhesion to the root surface, differentiation of the penetrating hyphae, penetration of root cortex and degradation of the physical barriers of the host (e.g. endodermis) to reach the xylem for infection. Adaptation to the host cell environment, including antifungal compounds and finally proliferation in the xylem vessels, production of reproductive structures and secretion of virulence determinants such as little polypeptides or phytotoxins is well defined [66].

Invasion of epidermal cells and penetration of cell walls by *Foc* race 1 and *Foc* TR4 have been demonstrated through

green fluorescent protein transformants [67, 68]. In an artificial inoculation study of banana (Silk, AAB), colonization of cortex and xylem vessels was observed at 5 and 15 days after inoculation, respectively [69].

Pathogenicity

Studying specific pathogenicity factors and genes in *Foc* is useful not only to identify pathogenic isolates, but also to identify the genes that could be silenced for effective management of the disease. Fungal pathogenicity genes are responsible for the formation of infection structures, cell wall degradation, toxin biosynthesis and signalling [70] so as to suppress plant immunity [71]. *Foc* secretes a wide array of proteins into the host xylem sap during colonization which contributes affirmatively to wilt disease development in banana [72], such as the specific effector proteins secreted in xylem (SIX). Genome and transcriptome analyses of *Foc* race 1 and TR4 identified several orthologous copies of SIX genes [73]. *Foc* TR4 possessed three copies of SIX1 gene as compared to the single copy present in *Foc* race 1. Additionally, *Foc* TR4 also contained SIX4, SIX6 and SIX8 [74, 75]. Isolation of a resistance gene analogous to the tomato I-3 gene would help in understanding the mechanism of pathogenicity of Fusarium wilt in the few wild-resistant cultivars of banana [76].

Certain pathogenicity genes also encode proteins that are involved in the suppression or disruption of host defence mechanisms [77]. Quantitative analysis of the transcript showed a significant increase in expression of *chsV*, MFS multidrug transporter and *ste12* genes in *Foc* STR4 and TR4 compared with that of non-pathogenic *F. oxysporum* [78]. Genome analysis reveals that the genome structures of race 1 and TR4 isolates are highly syntonic with those of *F. oxysporum* f. sp. *lycopersici* strain Fol 4287 [73].

Proteomics is an apt research tool to study microbial pathogens in terms of their proteome maps, stage-specific proteomics and pathogenicity factors. Many reports pertaining to cataloguing mycelial, conidial, sclerotial, organellar and secreted proteins (secretome) across a range of fungal species exist [79–83]. Proteome comparison studies between *Foc* race 1 and race 4 [84] and also between pathogenic *Foc* (race 1, VCG 0124) and non-pathogenic *F. oxysporum* (np*Fo*) [85] revealed the overexpression of proteins such as vesicle transport v-SNARE (soluble *N*-ethylmaleimide-sensitive factor attachment protein receptors) protein, developmentally regulated GTP binding protein, ankyrin, isocitrate np dehydrogenase [NADP], mitochondrial, homogentisate 1,2-dioxygenase and hypothetical protein in pathogenic *Foc*.

Genetic Diversity and Detection

Studying *Foc* diversity is very much essential for developing durable resistant banana cultivars and also for quarantine purposes [86]. Important methods used to study the variation in *Foc* include vegetative compatibility group (VCG) analysis and molecular characterization techniques.

VCG grouping is a useful technique for studying the genetic relationship between isolates of asexually reproducing fungi such as *Foc*. On the basis heterokaryon formation, isolates of *Foc* can be divided into genetically distinct groups known as vegetative compatibility groups. It gives an idea of the genetic diversity and evolution of the pathogen [87]. So far, 24 *Foc* VCGs have been identified across the world [88]. Isolates in some VCG were cross-compatible with those in other VCG, resulting in VCG complexes such as VCG 0120-01215, VCG 0124-0125-0128-01220 and VCG 01213-01216. The *Foc* race 1 belonged to VCG 0123, 0124, 0125, 0126, 0128, 01210 and 01215 whereas the *Foc* race 2 belonged to VCG complex 0128-01220 and 01214 [38, 89, 90]. Fusarium wilt of banana is caused by 35 different strains or genotypes of *Foc* among which, VCG 01213 called *Foc* TR4, is just one of six distinct strains that attacked Cavendish, but it is much more aggressive on Cavendish than the strains known previously [13]. The *Foc* TR4 belonged to a single group of VCG 01213/16 complex, whereas the *Foc* STR4 isolates belonged to VCG 0120, 0121, 0122, 0129 and 01211 [88, 91–93]. However, the use of VCG as a means to classify *Foc* is also considered to be incomplete as one race could comprise more than one VCG (e.g. VCG 0124 and VCG 0125 belonged to race 1) or one VCG occurs in multiple races (e.g. VCG 0124 occurs in both race 1 and race 2). Also, few of the VCG groups are cross-compatible, giving rise to VCG complexes making it more difficult for pathotype identification. However, Ghag *et al.* [76] separated 24 VCGs of *Foc* further into two clades and eight lineages, of which 21 VCGs are present in Australia and Asia. Mostert *et al.* [94] identified VCG complex 0124/5 as the most common one in the Indian subcontinent, Vietnam and Cambodia, while VCG complex 01213/16 as the dominant one in the rest of Asia. In this study, *Foc* VCG diversity in Bangladesh, Cambodia and Sri Lanka, and presence of VCGs 01221 and 01222 in Cambodia and Vietnam are documented.

While vegetative compatibility provided a clear measure of phenotypic relatedness, the molecular technique is useful to determine the genetic similarity between isolates within each VCG and the genetic relatedness among VCGs. The multigene phylogeny analysis done by Fourie *et al.* [91] separated *Foc* isolates into eight distinct and mostly unrelated lineages. It also indicated that the rRNA IGS (intergenic spacer) region found as an excellent marker for the diagnosis of *Fusarium* spp. in which lineage-specific polymerase chain reaction-restriction fragment length polymorphism (PCR-RFLP) fingerprints could be developed for *Foc* isolates since IGS region is a relatively quickly evolving region with the potential for more than one sequence to reside within a single genome [95]. The other regions utilized for phylogenetic study are translation elongation factor-1α (TEF), the mitochondrial small subunit (*MtSSU*) rRNA genes and a repeat region

encoded in the mitochondrial genome (*MtR*). The separation of *Foc* using DNA fingerprinting techniques such as RFLP [96], randomly amplified polymorphic DNAs and DNA amplification fingerprints [97] and amplified fragment length polymorphisms (AFLP) [98] correlated well with *Foc* VCG clusters of distinct phylogenetic lineages. A number of DNA-based studies have been employed to determine the phylogenetic relationships between *Foc* VCGs. These studies all suggested that *Foc* could be separated into two main clades and eight to ten lineages. Clade A (which included VCGs 0126, 0122, 0121, 01213/16) affected mainly *M. acuminata* hybrids, whereas Clade B (which included VCGs 0124/5, 0128, 01220, 0124/22, 0123, 01217, 01218, 01221) affected mainly *M. acuminata* × *M. balbisiana* hybrids. The lineages in *Foc* each contained one to five closely related VCGs. Zheng *et al.* [19] reported that isolates from Vietnam, Laos and Myanmar were genetically close related and resembled the *Foc* TR4 strain from Yunnan. Furthermore, the authors demonstrated genetic association between the *Foc* TR4 strains from Pakistan and the Philippines as well as between the strains from Lebanon and Jordan.

The early and correct diagnosis of plant pathogenic fungi is a crucial component of any crop management system. In this direction, Sharon [99] first designed oligonucleotide primer sequences R1F and R1R for the specific amplification of the Australian *Foc* race 1. Similarly, PCR markers [100] for the VCG complex 01213/16 (*Foc* TR4) [92, 101] have been developed for rapid identification. Dita *et al.* [92] have reported a PCR diagnostic that uniquely amplified a 463 bp amplicon in isolates belonging to *Foc* TR4, along with *in planta* detection method which provided a fastest receipt-to-result efficiency within 6 h. Lin *et al.* [102] used real-time PCR for quantification and detection of *Foc* TR4 by using the SCAR primer set *Foc*Sc-1/*Foc*Sc-2, which was designed according to the sequence of a 242 bp DNA fragment (Foc_{242}). They found that levels of *Foc* gDNA present in severely symptomatic banana pseudostems and leaves were 6946-fold and 26.69-fold higher than in those of mild-symptomatic banana, respectively. Zhang *et al.* [103] developed a real-time fluorescence loop-mediated isothermal amplification assay (RealAmp) for the rapid and quantitative detection of *Foc* TR4 in naturally infested soil samples, which could detect 100 times lower concentration than that of real-time PCR. The RealAmp assay was highly specific because it used four primers that recognized six regions on the target DNA. Previously, real-time PCR [102] and loop-mediated isothermal amplification assay have been developed to detect the *Foc* TR4 in banana tissues [104]. Peng *et al.* [105] used RealAmp for the rapid and quantitative detection of TR4 in soil and it facilitated to detect and differentiate ST4 isolates from TR4 isolates simultaneously. Besides, this method was highly tolerant to inhibitor substances in soil. Fraser-Smith *et al.* [75] detected *Foc* TR4 strains from an international collection of *Foc* isolates by screening for the presence of the putative effector SIX8. *Foc*-SIX8a was present in all race 4 isolates, whereas *Foc*-SIX8b was present only in all subtropical race 4 isolates. *Foc*-SIX8 was neither detected in any of the race 1 and 2 isolates nor in the putative non-pathogens assessed. These results suggest that *Foc*-SIX8 is a suitable candidate for the molecular differentiation of race 4 from race 1 and 2 isolates and also for the further differentiation of tropical and subtropical race 4 isolates. Neill *et al.* [106] reported the infection of *Foc* TR4 in Queensland, Australia by confirming the PCR results after sequencing the IGS region of the ribosomal DNA (rDNA) of the isolates obtained. A diagnostic PCR assay was optimized and used by Muhammad *et al.* [107] for specific detection of *Foc* TR4 in Basrai banana variety grown in Sindh province of Pakistan. An original approach, using orthogonal arrays and the Taguchi method, was employed by Aguayo *et al.* [108] to improve the sensitivity of detection of *Foc* TR4 strains causing disease to Cavendish bananas in the tropics, i.e. VCGs 01213/16 and 0121, without compromising its specificity. FWB-TR4 primer was developed and it was found to be specific, in which the DNA regions targeted by the FWB-TR4 primer were all 100% conserved within all the VCG 01213/16 and 0121 isolates.

Foc isolates could also be grouped as odoratum or inodoratum based on production or non-production of volatile aldehydes, respectively [40, 109]. Accordingly, *Foc* VCGs 0120, 0121, 0122, 0126, 0129, 01210, 01211, 01213, 01215, 01216 and 01219 are classified as 'odoratum' group while VCGs 0123, 0124, 0125, 0128, 01212, 01214, 01217, 01218 and 01220 are described as 'inodoratum' group [30]. It is speculated that the genes conferring race 4 virulence are linked to those governing volatile production [110].

Management of Fusarium Wilt

Since the occurrence of Fusarium wilt of banana, various management strategies such as flood-fallowing [41], application of organic amendments [5], planting of resistant banana varieties [111], crop rotation [22], fungicidal treatment [112] and soil fumigation [113] have been attempted. However, factors such as co-evolution with the plant host and the spread of virulence determinants *via* processes such as parasexuality, heterokaryosis and sexual recombination led to the evolution of new race [114], which in turn made these disease management practices much more complicated. Planting of resistant varieties is the effective way to control Fusarium wilt disease but it is limited due to consumer preference, extremely poor fertility particularly in the Cavendish subgroup [115, 116] and therefore somaclonal variation and genetic transformation are being exploited. Usage of antagonistic microbes, which protects and promote plant growth by colonizing and multiplying in both rhizosphere and plant system, also a potential environment safe alternative approach for the management of Fusarium wilt of banana has been

attempted [117]. Besides, botanicals with antifungal compounds have been attempted for the management of the disease [118]. Even though there are multiple options for managing Fusarium wilt of banana including prophylactic measures, the perennial nature of this pathosystem and the corresponding polycyclic nature of the disease hindered the advance of long-term management measures [119]. Hence, an integrated disease management programme for Fusarium wilt should be undertaken by involving planting disease-resistant varieties and following other strategies such as quarantine measures, cultural, chemical and biological control measures, etc.

Pathogen exclusion

The important aspect of management of Fusarium wilt at the initial stage is preventing the spread of the pathogen to other uninfected areas. Prevention of the spread of fungal propagules is vital since once introduced, the eradication of the fungus from a field could be very difficult by either soil or plant-applied disease control treatments [8]. Preventive measures include adopting strict quarantine practices, checking for infected plant materials and thorough cleaning of farm implements, which might help in slowing the spread of this disease [120]. Regional awareness and contingency programmes have been created in the Western hemisphere to ensure that stakeholders are informed about the symptoms and potential impact of *Foc* TR4 [121]. When *Foc* TR4 arrives in new areas, early recognition and delineation of the affected areas are desirable. Daniells *et al*. [122] observed that combination of clean planting material, clean fields and effective quarantine have been cost-effective in *Foc* prevention besides growing resistant cultivars and application of soil additives.

Reduction of inoculum level

Since Fusarium wilt is a soil-borne systemic disease, control strategies are mainly addressed to reduce soil inoculums before planting banana. Flooding the *Foc* infested field for 3–4 months with a minimum of 30 cm of water significantly reduced populations of *Foc* in soil and controlled Fusarium wilt [123] as it creates anaerobic conditions [5, 41]. Removing and burning infected plants and spraying the soil with the fungicide Triadimefon three times at 25-day intervals are found effective in China [124]. Temperatures of 65 and 90 °C are necessary to eliminate microconidia and chlamydospores, respectively [125]. Rice hull burning to heat sterilize the soil [126], burning the diseased plant parts such as rhizome, pseudostem, leaves and sterilization of infected soils [127], and elimination of weeds, insect vectors and plant-parasitic nematodes harbouring wilt pathogen are proved to be the effective *Foc* management practices [54].

Chemical measures

Chemical control is an essential component of an integrated disease management programme. The use of fungicides [128] or surface sterilants [129] is effective at preventing the spread of *Foc*.

Fungicides such as cyproconazole, propiconazole and prochloraz showed Fusarium wilt disease reduction of around 80% in banana plants [120] and their mode of action is to be inhibiting the demethylation step in the biosynthesis of sterol. Fungicides belonging to the benzimidazole group such as benomyl, carbendazim and thiabendazole which act as a multiplication inhibitor during fungal mitosis [130] have shown effective at controlling *Foc in vitro* and in greenhouse conditions [128]. Injection of rhizome with 2% carbendazim in cv. Rasthali was effective at controlling Fusarium wilt but the same treatment was not effective in South Africa. However, potassium phosphate (20%) injection into the pseudostem had some positive effect [54]. Surface sterilants, such as quaternary ammonium compounds (10%), sodium hypochlorite (5%), detergent and cleansers are essential components of a disease management programme and they are used to disinfect any equipment capable of transporting *Foc*-infected soil such as farm machinery and implements such as tractors, shovels, cane knives and also footwear. Farmcleanse® containing 10% alkali metal salts of alkylbenzene sulphonic acid, 5% coconut diethanolamide and 1% pyridine-2-thiol 1-oxide sodium salt is found to be the most effective, totally inhibiting germination of conidia when applied at the recommended rate of 10% [29]. Surface sterilant Sporekill® containing 12% polydiallyldimethylammonium chloride reported to inhibit spore germination of *Foc* TR4 [129], hence it could be utilized as an effective disinfecting agent of *Foc* TR4. However, most of these chemicals are non-reliable, as they are known to cause some environmental hazards and harmful to banana industry workers [131].

Soil amendments

Soils with higher biological diversity and activity, such as natural or organically managed agricultural soils are often more suppressive to root infecting fungi than conventionally managed agricultural soils [132–135]. Application of bioorganic fertilizers increased the bacterial diversity in the rhizosphere of banana [136]. The microbial community structure in soil amended with organic fertilizers for a long period is significantly different from that in soil amended with chemical fertilizer as revealed by PCR-DGGE or 454 pyrosequencing methods [137–140]. Biocontrol agents when applied in combination with organic materials have shown the enhanced activity of biocontrol microbes, resulting in better disease control than the application of biocontrol microbes alone [136, 141, 142].

Application of chemical amendments in soil also shows some effects on disease control. Liming of soil reduces the

survival period of the *Foc* to 2 months [143]. High lime (CaO) content (175–280 ppm) enhances disease suppression in the soil [144] as it reduces germination of chlamydospores. Addition of calcium carbonate ($CaCO_3$), calcium hydroxide [$Ca(OH)_2$], calcium sulphate ($CaSO_4$) or iron chelates to the soil, reduces *Foc* germination and thus disease severity. Reduction of iron availability increases soil suppression [145] as well as reducing chlamydospore germination [51]. High P content in soil reduces Fusarium wilt incidence [146]. Soil application of calcium compounds and phosphate salts such as $Ca(OH)_2$, $Ca(NO_3)_24H_2O$, $CaCO_3$, $CaSO_4$, K_2HPO_4 and $NaH_2PO_42H_2O$, strongly inhibits chlamydospore germination and promotes lysis of germ tubes of *Foc* in soil [147, 148]. The impact of nitrate (NO_3) and ammoniacal (NH_4) nitrogen is well documented on Fusarium wilts of annual hosts [149] and generally, NO_3 decreases the severity of these diseases, whereas NH_4 increases the severity.

Soil amendment using silicon (Si) is found effective at controlling Fusarium wilt in banana [150–152]. Silica is used by the plant in the form of silicic acid and deposits as elemental Si in the cell wall which in turn blocks the entry of *Foc*. In addition to this, Si also induces plant defence mechanism and enhances the production of few compounds such as phenolics, lignin-thioglycolic acid and enzymes such as peroxidases, polyphenol oxidases and chitinases which are involved in plant protection [153]. Influence of soil abiotic factors such as soil pH, N and Mn; on *Foc* race 1 incidence in 'Gros Michel' banana variety showed that a high soil pH, lower Ca and Mg content results in a higher bunch weight from plants under infected conditions [154].

Disease suppressive soils

Disease development depends on the condition of the soil [8, 51]. Healthy and disease-resistant soil for a longer period termed as disease suppressive soils. In the suppressive soils which contained more microbial population, suppress the pathogen development and such soils have been reported in Central America, the Canary Islands, Australia and South Africa [42]. In general, suppressive soils have higher pH values and on the other hand soils with lower pH values are significantly correlated with a higher incidence of Fusarium wilt in Peru [155]. The nature of disease-suppressive soil is known to be influenced by its mineral content, microbiome and soil structure [156]. In tropical America, a close relationship is found between suppression of Fusarium wilt and the presence of clay (montmorillonite type) soils, whereas in the Canary Islands, suppression is associated with host mineral nutrition [157]. Smith *et al.* [158] proposed that by the application of biocontrol agents isolated from banana roots grown in Fusarium wilt suppressive soil of tissue culture plantlets in the nursery had a better chance of protection against *Foc*. Non-pathogenic *F. oxysporum* (np*Fo*) and *Trichoderma* isolates from suppressive soils in South Africa suppresses Fusarium wilt of banana in the glasshouse [159]. Addition of organic matter boosts the general suppressiveness of soils [160–163]. Specific suppressiveness due to the combined activity of specific groups of microorganisms, which could interfere at a particular stage of the life cycle of the soil-borne pathogen, could be transferred to conducive soils by mixing smaller amounts (1–10% w/w) of the suppressive soil into the conducive soil [164, 165]. For Fusarium wilt suppressive soils, competition for carbon by non-pathogenic *F. oxysporum* [166, 167] and siderophore-mediated competition for iron by rhizosphere bacteria [168, 169] are shown to be the key mechanisms. Also, antimicrobial volatiles such as sesquiterpenes [170], methyl 2-methylpentanoate and 1,3,5-trichloro-2-methoxy benzene [171], 2-methylfuran, 2-furaldehyde, 2-(methylthio)benzothiazole and murolool [172] have been studied for their potential role in disease suppressive soils *in vitro* and it shows that these volatiles strongly reduce the hyphal growth of the Fusarium wilt pathogen. Suppression of Fusarium wilt is usually more related to microbial characteristics and enzymatic activities than to any of the chemical soil parameters tested [173, 174]. Application of *Bacillus amyloliquefaciens* NJN-6 (BIO) isolate along with compost revealed a significant decrease in Fusarium wilt disease incidence of 68.5%, resulting in a two-fold increase in yield. Study on impact of application of biofertilizer (BIO), pig manure compost (PM) and chemical fertilizer (CF) on the composition of rhizosphere microbial community revealed that significant increase in Acidobacteria (Gp1 and Gp3), Firmicutes, Leptosphaeria and while vice versa for Proteobacteria and Ascomycota including Fusarium, a causal pathogen for Fusarium wilt disease in the BIO treatment [175] than in CF and PM treatments. Other potentially beneficial bacterial communities associated with disease suppression are *Burkholderia*, Gaiellaceae, Paenibacillaceae and Streptomycetaceae. Both Gaiellaceae and Streptomycetaceae, belonging to Actinobacteria, are the dominant groups in soils resistant to Fusarium wilt of banana [51]. *Burkholderia* sp. is capable of colonizing the surface of *Foc* hyphae and cause mycelial deformation with terminal and intercalary swelling [176]. Paenibacillaceae produces fusaricidins, an antifungal compound group that suppresses *F. oxysporum* f. sp. *nevium* [177]. Understanding the temporal and spatial microbial dynamics of disease-suppressive soils as well as the corresponding modes of action is needed to facilitate the development of effective, consistent and durable disease management tools. A model predicting Fusarium wilt suppressiveness, including several soil factors combined with the abundance of three keystone microbial tax, such as Actinobacteria, Firmicutes and Acidobacteria as the major microbial predictors for bulk soil suppressiveness at a continental scale, in Australia, has been developed [178].

Research on management practices in order to select and stimulate indigenous microbial communities or activities that enhances suppressiveness in the soil is yet to be

explored. Few studies pertaining to the use of specific soil amendments including chitosan [179], chitin [180], fish emulsion [181], application of agricultural practices such as crop rotation or minimum tillage [182, 183] or use of cover crops [184] or even by host-mediated microbiome engineering, where the protective microbiome is artificially selected over multiple generations [185], have been conducted in order to identify an apt integrated disease management system for Fusarium wilt.

Crop rotation

The monotony in the characteristics of the cultivated soil which might lead to the conditions favourable for pathogen multiplication and spread could be disrupted by following crop rotation. Different crop rotation systems have a varied effect on suppression of different diseases *via* mechanisms such as interrupting the pathogen life cycle, variation in establishment of antagonistic microbes exhibited due to variation in plant root exudates and production of allelochemicals [186, 187]. The bacterial community that occurred either in soil or in plant endosphere could be one of the main reasons for disease suppression [188] and variations in bacterial diversity and structure in relation to crop rotation have been studied in detail by Fierer *et al*. [189, 190]. Crop rotation with paddy and flooding for 3–4 months before planting banana is found to be effective [63, 147] and also inter-planting with cassava lowered the inoculum [13]. Banana rotation with Chinese leek and paddy could control Fusarium wilt, and it is speculated that the control is due to release of antifungal compounds from root exudates or leaf leachates such as 1-dimethyl-2-pentenal and dimethyl trisulphide [191].

Instead of monoculture, adoption of mixed planting by growing diverse banana cultivars often incurred moderate losses [5]. Two-year crop rotation systems of banana with other crops such as maize, pea, pineapple and cassava demonstrated that pineapple–banana system reduced *Foc* population and suppressed the disease incidence, along with significantly higher abundances of *Acidobacteria*, antagonistic *Burkholderia*, *Planctomycete* and *Chloroflexi* and Basidiomycetes rather than Ascomycetes [192]. Soil with favourable abiotic properties and a proper plant arrangement also could help to promote Fusarium wilt suppression in susceptible banana variety [193].

Soil solarization and cover crops

Disease control through cultural practices (crop rotation and flooding) and solarization are practiced in Latin America, Taiwan, India, Malaysia, Australia and Indonesia [5, 194–196]. Soil solarization has the power to convert conducive to suppressive soil for wilt diseases [197]. The process of soil solarization by means of heating soils under transparent plastic tarps to raise temperature which is detrimental to fungal pathogens has successfully been used to control variety of plant diseases without destroying the beneficial microbes [198–200]. Solarization could increase the soil temperature to 52.35 °C, which consequently could suppress the Fusarium population in the soil and reduce Fusarium wilt incidence on banana compared to practices such as fallowing and crop rotation with maize [201].

Cover cropping followed by incorporation of plant residues into the soil is effective to suppress certain soil-borne pathogens [202] as they increase the nutrient availability, reduce groundwater contamination and stimulate beneficial microflora in the soil [203]. The incorporation of plant residues to the soil is also helpful to increase the benefits of solarization [204]. In addition to direct effects on plant pathogens, many cover crops impact plant pathogens indirectly by triggering the plants host defence response and induce specific suppression by enhancing individual beneficial organisms such as *Trichoderma harzianum* and mycorrhizae in soils. Also no-till cover crops provide many of the above said benefits and additionally, they act as a physical barrier that reduces the splash of soil, soil-borne pathogens onto foliage, stems or fruit and the presence of free moisture on the plant. Several plant species of Fabaceae and Poaceae served as cover crops in banana cropping [205–207]. Addition of *Brachiaria decumbens* as a cover crop alters the food web of macrofauna in soil litter which might be helpful in controlling the banana weevil, *C. sordidus*, a dispersal agent of Fusarium wilt pathogen [208]. Management of ground cover at the base of the banana plantations is found to be a significant factor in reducing the incidence and severity of Fusarium wilt in bananas [209] as the cover crops reduces the weed population and plant-parasitic nematode infection [210] which are involved in the dispersal of Fusarium wilt pathogen. Application of silicon, *T. harzianum*, compost, differentiated sources of NPK and growing *Crotalaria juncea* as a cover crop could reduce the Fusarium wilt disease severity index up to 23% as compared to that of control (81%), in 14-month-old banana plants [211].

In general, volatiles released by living plants are effective for soil-borne pathogens. For example, the aqueous leachates and volatiles emitted from the intact growing roots of Chinese chive (*Allium tuberosum* Rottler) inhibit spore germination of *Foc*. The characterization of the volatiles revealed the presence of five different compounds and among which 2-methyl-2-pentenal and dimethyl trisulphide showed stronger inhibition on *Foc* TR4 [191].

Soil microbiome modification

Soil suppression of disease induced by organic amendments and biocontrol agents have been widely described [212, 213] and are more frequently related to the

modification of the soil microbiota [214, 215], which mainly promote general and specific suppression mechanisms [216] against the disease causing fungal pathogens. It is observed that the rhizosphere soils amended with biocontrol *B. amyloliquefaciens* NJN-6 (BIO) enriched the bacterial genera *Sphingobium* (Gp6 and Gp4), *Lysobacter*, *Sphingopyxis*, *Cryptococcus* and *Dyadobacter* at significant levels, and steadily suppressed the *Fusarium* as compared to those in compost-enriched soils [217]. Therefore, specifically high diversity communities create a competitive environment deleterious to pathogens where competition for nutrients is a mechanism that limits survival and invasion by soil-borne pathogens [218].

Biological control

The continuous cropping causes the loss of soil biodiversity and destroys the ecological balance of soil, which offers favourable conditions for the build-up of Fusarium wilt pathogens [219]. Expediting the growth of antagonistic microorganisms, in both rhizosphere and endosphere of the plant is an important approach in the management of Fusarium wilt of banana. Several reports have demonstrated the successful use of different species of *Trichoderma*, *Pseudomonas*, *Bacillus*, *Burkholderia cepacia*, *Streptomyces*, non-pathogenic *F. oxysporum* (np*Fo*) of both endophytic and rhizospheric in nature against Fusarium wilt disease [175, 218, 220]. Application of np*Fo* strain Ro-3 three times resulted in the reduction of Fusarium wilt disease severity by up to 89% and significant enhancement in plant growth [221]. Application of *B. amyloliquefaciens* strain NJN-6 along with the organic mixture of pig manure compost and amino acid fertilizer (2:3 w/w) has also effectively suppressed Fusarium wilt disease in banana [222, 223]. In general, the genera *Bacillus* remain the most promising biocontrol agents involved in suppression of various soil-borne pathogens as they form a stable and extensive biofilm [224] and also secrete many antifungal compounds such as surfactin, bacillomycin and macrolactin [223, 225].

Performance of certain microbial groups such as oligotrophic bacteria and actinomycetes or non-pathogenic Fusarium species is better in suppression of Fusarium wilt than other groups [226]. Both higher bacterial diversity and lower fungal diversity are associated with disease suppression [187, 191, 227] and the most evaluated strains are np*Fo* (32%) followed by *Trichoderma* and *Pseudomonas* [228, 229]. Biocontrol agents also applied along with botanicals for enhancing protection of plants from various diseases. Combined application of botanical formulation (*Datura metel* Wanis EC and Damet 50 EC) and biocontrol agents (*Pseudomonas fluorescens* Pf1 and *Bacillus subtilis* TRC 54) has reduced the wilt incidence significantly under greenhouse by 64% and field conditions by 75% [230]. Zimmu (*Allium cepa* × *Allium sativum*) leaf extract alone could effectively suppress Fusarium wilt disease under both greenhouse and field conditions in the Cavendish cultivar 'Grand Naine' and enhanced the yield at significant level [231]. There are fewer studies on the biocontrol effects of microbial strains against Fusarium wilt of banana conducted under field conditions [219, 223, 225, 232, 233]. Many potential biocontrol agents which exhibited excellent suppression of Fusarium wilt *in vitro* and in greenhouse have failed under field conditions [234]. A possible reason could be a failure in the selection of biocontrol agents with multiple functions (biological control and plant growth promotion activities) and actions [8]. A biocontrol agent that showed antagonistic activity against Fusarium wilt in one field sometimes failed in another, which could be due to the significant differences in colony morphology and virulence of pathogenic strains infecting the other field. Therefore, it is necessary that newly isolated and characterized biocontrol strains should be evaluated under field conditions to assess their biocontrol potential.

For commercial purposes, the suitable methods for the mass production of biocontrol agents with the advantages of long shelf life, easy preparation and supportive to the growth of biocontrol agents should be evaluated. For instance, *T. harzianum* Th-10 on dried banana leaf formulation survived for 4–6 months and served as cheaper formulation for control of *Foc* [232]. In banana, particularly, *Bacillus* strains showed maximum average biocontrol with the nursery application method [228] while *Trichoderma* strains were more effective with the drenching method [235]. *Pseudomonas* and np*Fo* strains showed maximum average biocontrol with root dipping methods [236, 237].

In addition, several reports have documented that the use of biocontrol agents in combinations are more effective than individual agents for the management of plant diseases [238–242]. Combined application of two endophytes *viz.*, *Pseudomonas* sp. UPMP3 and *Burkholderia* sp. UPMB3 showed significant reduction of Fusarium wilt disease in susceptible banana cv. Berangan [243]. Banana sucker treatment before planting with biocontrol agents *Trichoderma viride* and *P. fluorescens* and soil drenching with same biocontrol agents twice at 30 and 180 days after planting as booster application, effectively reduced the Fusarium wilt disease incidence and intensity and enhanced the yield [244]. The application of endophytic *Trichoderma asperellum* Prr2 + rhizospheric *Trichoderm*a sp. NRCB3 recorded 100% reductions of Fusarium wilt disease under both pot and field conditions [245]. A series of soil treatments are required for combating *Foc* as the combination of endophytic and rhizospheric bacterial microbes applied on three occasions (at the time of planting, and 2 months and 4 months after planting) resulted in significant reductions of Fusarium wilt and also promoted plant growth parameters. These studies have therefore provided useful information to assist the determination of suitable biocontrol agent(s) and application intervals required for the successful suppression Fusarium wilt disease of banana [246].

There is growing evidence in use of arbuscular mycorrhizal fungi for the control of several fungal diseases and also to promote plant growth [247]. Application of *Glomus mosseae* + *T. harzianum* in plants challenged with *Foc* under field conditions could sustain 61 and 70% improvement in plant height and girth, respectively and gain of 75% in bunch weight [248] besides reducing the *Foc* population. Application of *Glomus etunicatum* (KPV) + *Pseudomonas aeruginosa* (Ge-A + Ge-B) or *G. mosseae* (TPV) + *Pseudomonas* sp. (Gm-A) combination also significantly suppressed the Fusarium wilt disease under pot culture conditions [249].

Currently, banana planting materials used are derived from tissue culture where the plants are raised under axenic conditions, thus the plants devoid of rhizospheric/endophytic microbes including beneficial organisms. Hence the tissue-cultured plantlets are succumbing to soil-borne disease quickly. In another study, application of mixture of native endophytic bacteria (mostly *γ*-proteobacteria) into tissue-cultured banana recorded 67% control of Fusarium wilt disease under greenhouse conditions [250]. Similarly, re-introduction of naturally occurring endophytes to tissue cultured banana plantlets resulted in a substantial reduction in the infection and severity of Fusarium wilt disease as well as increase in plant growth parameters [250]. It is also important to note that incorporation of endophytic and rhizospheric microbes during *in vitro* culturing of tissue culture plants showed greater advantages than they are applied in field conditions. For an instance *in vitro* bacterization of tissue culture plantlets with endophytes, *B. subtilis* strain EPB56 and EPB10 and the rhizobacteria, *P. fluorescens* strain Pf1 showed high-level reduction (more than 70%) of Fusarium wilt disease and doubling of yield under field conditions [251].

The possible mechanisms involved in the reduction of Fusarium wilt severity by biocontrol agents have been well documented by senior authors and others [252]. Among the mechanisms of pathogen inhibition, production of antimicrobial compounds, which could be volatile or non-volatile compounds, antibiotics, chitinase and other lytic enzymes, siderophores and hydrogen cyanide, are the most important traits possessed by the biocontrol agents and these traits are involved in the destruction of the cell wall integrity of the pathogen, leading to effective control [253].

Host resistance

Conventional breeding

Though chemical, cultural and biological means give considerable protection against highly virulent strain *Foc* TR4, complete protection is achieved only through cultivating resistant cultivars. As there is no commercially important Cavendish cultivar (AAA), identification of resistant sources in the related groups of banana and utilizing them in breeding for obtaining resistant against the wilt is hour of the need. Already a successful cross between 'Sukali Ndizi' and 'TMB2X8075-7' denoted complete resistant against *Foc* race 1 and described the gene responsible for resistant is single recessive and the gene was also named as Panama disease 1 [254]. Recently, wild banana *viz*., *Musa basjoo*, *M. itinerans* [255], *M. nagensium*, *M. ruiliensis*, *M. velutina* and *M. yunnanensis* showed resistance to *Foc* TR4 under screening and these can be utilized in breeding programme to obtain desirable resistant cultivars. In addition, *M. acuminata* subsp. *malaccensis* has shown resistance to *Foc* TR4 [256]. In the Caribbean and Mozambique regions, diploids and wild sp. such as Pahang (AA), Calcutta 4 (AA) and *M. itinerans* showed high degree of resistance to *Foc* TR4 [257]. Researchers from China have developed five different *Foc* TR4 resistant/tolerant varieties and among those, ZJ 9-triploid was completely resistant to *Foc* TR4 in China (developed by crossing diploid with tetraploid-FHIA 01) (personal communication).

Some level of resistance to Fusarium wilt has been obtained by using somaclonal variation techniques [23] and the resistant cultivars developed from Taiwan Banana Research Institute (TBRI) were GCTCV-44, GCTCV-53 and GCTCV-119. These improved variants were not acceptable for commercial planting because of shorter shelf-life and longer de-greening time of fruit in comparison with the parental Giant Cavendish. The variant GCTCV-215-1 (Tai Chiao No. 1) is found to be a promising candidate for commercial planting but the disadvantage of this cultivar is that it requires longer time to complete the crop cycle and produce a lighter fruit bunch in comparison with Giant Cavendish. Another variant GCTCV-218 (Formosana) is less susceptible to *Foc* TR4, high yielding with high-quality fruits. Thus the cv. Formosana ranks higher for consumer preference both in local and Japanese markets. Some of the cultivars or wild genotypes such as cv. Rose (AA, *M. accuminata* ssp. *malaccensis*), and numerous AAA and AAB-Plantain types are found to be resistant to *Foc* TR4 [258–260]. Tolerance/resistance to *Foc* TR4 is also found in several bred hybrids, especially in those developed by the programme at the Fundación Hondureña de Investigación Agrícola (FHIA) in Honduras [261] such as FHIA-01 (Gold finger), FHIA 18, FHIA 2 (Mona lisa), FHIA 25 and SH-3640/10 (High Noon) [262]. However, the somaclonal variants obtained so far relied on quantitative resistance that is highly dependent on the inoculum concentration of the pathogen and they required other management strategies such as adopting annual cropping systems, which are not practicable for most banana producers worldwide. Instead, the substantial genetic diversity for TR4 resistance in wild banana germplasm, such as accessions of *M. acuminata* ssp. *malaccensis* [263], could be exploited in breeding programmes and/or along with various transformation techniques [264] to develop a new generation of banana cultivars in conformity with consumer preferences [265]. The whole genome

sequence of banana genotype, DH-Pahang, which is resistant to the *Foc* TR4 pathogen, might form valuable information for crop improvement in future [266]. Methods such as protoplasts transformation and gene knockout systems for *Foc* would enhance the study of plant pathogen interactions and also the early monitoring and screening resistant materials for banana disease resistance breeding [267].

Transcriptome sequencing of *Foc* TR4 resistant cv. 'Yueyoukang 1' indicated that the genes related to CEBiP, BAK1, NB-LRR proteins, PR proteins, transcription factor and cell wall lignification have been found to induce strongly upon infection with *Foc* TR4 [268].

Mutation breeding is also an important approach for the development of resistant cultivars against *Foc* TR4. Ethyl methane sulphonate (EMS) induced mutants of Brazil banana (*Musa* sp. AAA) and Williams 8818-1 showed resistance against *Foc* TR4 [269, 270]. In our laboratory also, the putative gamma-irradiated embryonic cell suspension derived plants of cv. Grand Naine showed resistance to *Foc* TR4 under glasshouse conditions. Though the success of a positive mutant is a rare event, it suggests that it is possible to develop banana plant resistant against *Foc* TR4 by attempting mutagenesis at large scale especially in commercial cultivars.

Transgenic approach

Genetic modification of banana through appropriate methods is important for developing elite edible banana plants resistant to different races of *Foc*. Genetic transformation of elite banana cultivars for resistance to *Foc* has been accomplished by using techniques such as particle bombardment, sonication-assisted vacuum infiltration of apical meristem and *Agrobacterium*-mediated gene transformation [271–275]. Microarray studies have also been used to identify the genes significantly involved in the early stages of interaction between banana and *Foc* TR4. It showed that the cell wall strengthening genes might be important for banana resistance to Fusarium wilt [276]. Besides, genes encoding antimicrobial peptides are strong candidate for fungal resistance in *Musa* sp. as they are highly inhibitory to *Foc in vitro* [277].

Understanding the molecular mechanisms of certain proteins in banana–*Foc* interaction would reveal how these proteins are associated with the induction of resistance against *Foc*. Overexpression of the plant pectin methylesterase inhibitor protein reduced the activity or expression level of PME, resulting in enhanced resistance of plants to pathogens [278–280]. In banana, increased PMEs and followed by decreased degrees of pectin methylesterification accompanied by increased low methylesterified homogalacturonan (HGs) in the root vascular cylinder appeared to play a key role in specifying the susceptibility of banana plant to *Foc* [281].

Overexpression of antimicrobial proteins (AMP) such as magainin [282] and defensins [283] in transgenic banana plants has led to some level of tolerance to Fusarium wilt. Other groups have tried animal cell-derived apoptosis-inhibition-related genes namely CED9, Bcl-xL and Bcl-2 3′-UTR to prevent the necrotrophic death of banana plants after the *Foc* established itself in the host plant. In the transgenic banana cv. Rasthali higher AMP content was negatively correlated with *Foc* disease symptoms [284]. Similarly, overexpression of PCD gene *MusaBAG1* in response to *Foc* infection in banana is found to confer resistance to *Foc* in greenhouse bioassays [275].

Among defence-related genes, those encoding nucleotide-binding site leucine-rich repeat proteins are found to be less represented in the *Musa* sequence (89 genes) compared to *Oryza sativa* (464 genes) and *Vitis vinifera* (459 genes) [263]. Pathogen-triggered immunity-related genes such as chitin elicitor-binding protein (CEBiP) and the chitin elicitor receptor kinase (CERK1), the important components of the plant signalling pathway that recognized chitin oligosaccharide, are found to express more in resistant cultivar than in susceptible cultivars against *Foc* TR4 [285].

The identified SCAR markers and Quantitative Trait Loci linked to Fusarium wilt resistance mostly corresponded to resistance genes that might play major role in recognition of pathogen. Hence wherever the genome sequence is available, studies can be carried out in future to characterize genes involved in defence mechanism pathways. Petunia floral defensins, *PhDef1* and *PhDef2* (antimicrobial protein), have been overexpressed in transgenic banana plants using embryogenic cells as explants for Agrobacterium-mediated genetic transformation. The high-level constitutive expression of these defensins in elite banana cv. Rasthali led to significant resistance against infection of *Foc in vitro* and *ex vivo* bioassays [283]. Expression of rice thaumatin-like protein gene in transgenic banana plants showed enhanced resistance to *Foc* TR4 [286]. RNAi-based strategy for banana resistance using dsRNAs of adenylate cyclase, DNA polymerase alpha and delta subunits against *Foc* spores *in vitro* displayed varying degrees of inhibition of spore germination [287]. Recently two lines of transgenic Cavendish have been developed, of which one was transformed with *RGA*2, a putative nucleotide-binding and leucine-rich repeat (NB-LRR)-type resistance (R) gene, from a seedling of *M. acuminata* ssp. *malaccensis* and the other with *Ced*9 an anti-apoptosis gene derived from the nematode *Caenorhabditis elegans*, and the lines were free from *Foc* TR4 disease [288]. Transgene expression in the *RGA*2 lines was strongly correlated with resistance. However, these transgenic lines are to be tested for their resistance with the *Foc* isolates collected from different parts of the world as the *Foc* strains may differ in its virulence from place to place.

Although development of resistance is achieved normally through conventional breeding or by genetic engineering, recent approach of genome editing using

Clustered Regularly Interspaced Short Palindromic Repeats associated protein9 (CRISPR)/Cas9) tool has shown to achieve desirable traits by modifying plant genome [289]. Recently, banana genome editing using this tool for targeting MaATG8s gene for developing resistance against *Foc* TR4 [290] and *MaSWEET-1a*, *MaSWEET-4b*, *MaSWEET-14b*, *MaSWEET-4c*, *MaSWEET-14c*, *MaSWEET-4d*, *MaSWEET-14d* and *MaSWEET-14 h* [291] or *MaAPS1* and *MaAPL3* genes for *Foc TR4* and abiotic resistance together have been suggested. Since transcriptional up-regulation of *MaAGPase* genes occurs in response to *Foc* TR4 infection, these genes may play a role in modulating the response to fungal infections in banana [292]. Thus, *CRISPR/Cas9* could, therefore, modify banana gene expression to enhance resistance to *Foc* and further improvement of banana.

Conclusion

Among various production constraints, Fusarium wilt of banana is becoming the most devastating disease affecting commercial and subsistence of banana production worldwide. The aggressive strain of *Foc* TR4 which was first detected in Asia in the 1990s is now in found most of the banana growing regions of the world including Central America. Because of this, the global banana production is under severe threat, which in turn will have a calamitous impact on livelihoods and food security of millions of smallholders who grow more than 85% of the crop. Since no single-method is available to contain the disease effectively, integration of different management strategies such as creation of awareness and sensitization among all the stakeholders including the plant tissue culture companies; quarantine and sanitation measures to prevent the spread of disease to un-infected areas; soil health improvement which includes crop rotation, intercropping, cover cropping, need-based application of fertilizers, application of effective microbes and soil amendments such as cakes, organic manures, ashes and banana waste recycling; use of resistant varieties; disease-free planting material; good agricultural practices have to be followed to effectively manage this lethal disease. As 'time and tide waits for none', it is better to quicken the enforcement of various preventive and management strategies to cease the spread of the disease before it engulfs the entire banana reign.

Acknowledgements

The authors are very grateful to Dr R. Viswanathan, Principal Scientist and Head, crop protection division, Sugarcane Breeding Institute, Coimbatore and Dr E. Edwin Raj ICAR-NRC for Banana, Tiruchirapalli for suggesting corrections to the manuscript.

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