Chapter

Fusarium Wilt in Banana: Epidemics and Management Strategies

Fatin Nadiah Jamil, Chu-Nie Tang, Noor Baity Saidi, Kok-Song Lai and Nadiya Akmal Baharum

Abstract

Fusarium wilt, caused by *Fusarium oxysporum* f. sp. *cubense* (Foc), is one of the most threatening fungal diseases affecting banana plantations across the globe. It was first discovered in Australia in 1874 and has now spread to numerous different regions in the world hinting at the persistency of the pathogen. Various management strategies have been devised aiming mainly on improving the plant's tolerance or suppressing the infection. Fungicide is commonly used to control the disease spread, but it does not provide total protection to the plants besides displaying selective effectiveness on certain Foc strains. Alternatively, farmers apply crop rotation, rice hull burning, biological soil disinfestation, and compound-supplemented soil in their banana plantations. Studies have also shown that certain biocontrol agents manage to curb the disease threat. Selection of somaclonal variants and genetic manipulation via induced mutagenesis and transformation are also among the alternatives that have been implemented in producing *Fusarium*-tolerant and *Fusarium*-resistant banana plants. This chapter will describe *Fusarium* epidemics in banana, the effectiveness and challenges of different management approaches, as well as the future alternatives that can be adopted by taking advantages of the latest advances in omics technologies.

Keywords: disease control, epidemiology, *Fusarium oxysporum* f. sp. *cubense*, Foc, fungal disease, soilborne

1. Introduction

Fusarium wilt is the most destructive fungal disease affecting banana plantation across the globe [1–4]. It is caused by a type of soilborne fungal called *Fusarium oxysporum* f. sp. *cubense* (Foc). The pathogen penetrates through banana roots and dominates the vascular tissues, which disrupts the dissemination of necessary nutrients from roots to the upper parts of the plants [5–8]. It was first discovered in Australia [9] and was later followed by reports from Tropical America (Costa Rica and Panama) in 1890 [10]. Despite the first occurrence in Australia, the disease was believed to have originated in Southeast Asia [10–13]. Drastic increase in the number of new disease records occurred in the early 1900s causing over USD 2.3 billion loss in the last century [14], most of which described the damages in the export plantations of "Gros Michel" variety affected by race 1 of Foc [1, 10, 15].

In the recent years, Foc Tropical race 4 (FocTR4) emerges as the most virulent strain causing epidemics in Taiwan, Peninsular Malaysia, Indonesia, the Philippines [16], China, Jordan, Mozambique, Lebanon, Pakistan [6, 17–20], Australia [21, 22], Vietnam [23], Laos [24], Myanmar [8], and Israel [25]. The latest outbreak of FocTR4 has been confirmed in the Americas affecting the most popular commercial variety which could have jeopardized banana production for decades [26]. According to several unofficial reports, about 15,500 hectare (ha) of plantation areas in the Philippines, 40,000 ha in China, and 80% of production area in the Jordan Valley had been jeopardized by FocTR4 [14]. Several management strategies have been executed in controlling the spread of *Fusarium* wilt. However, they did not manage to provide long-term solutions to the problem. Current cutting-edge technologies particularly through omics studies may provide better alternatives to solve this long-running issue. Here, we discuss the current knowledge on *Fusarium* wilt with special emphasis on current disease management approaches and rising new omics technologies that can be adopted.

2. Symptoms of *Fusarium* wilt

Fusarium oxysporum f. sp. *cubense* (Foc) causes a typical wilt syndrome on the infected banana plants accompanied by the necrosis and rotting of roots, rhizome, and pseudostem vessels. The most typical symptoms become visible in susceptible banana plants after the appearance of initial external symptoms such as pale green streaks on the base of the petiole and the brown-reddish discoloration of the vessels under the epidermis of the petiole (**Figure 1**). These symptoms occur between 2 and

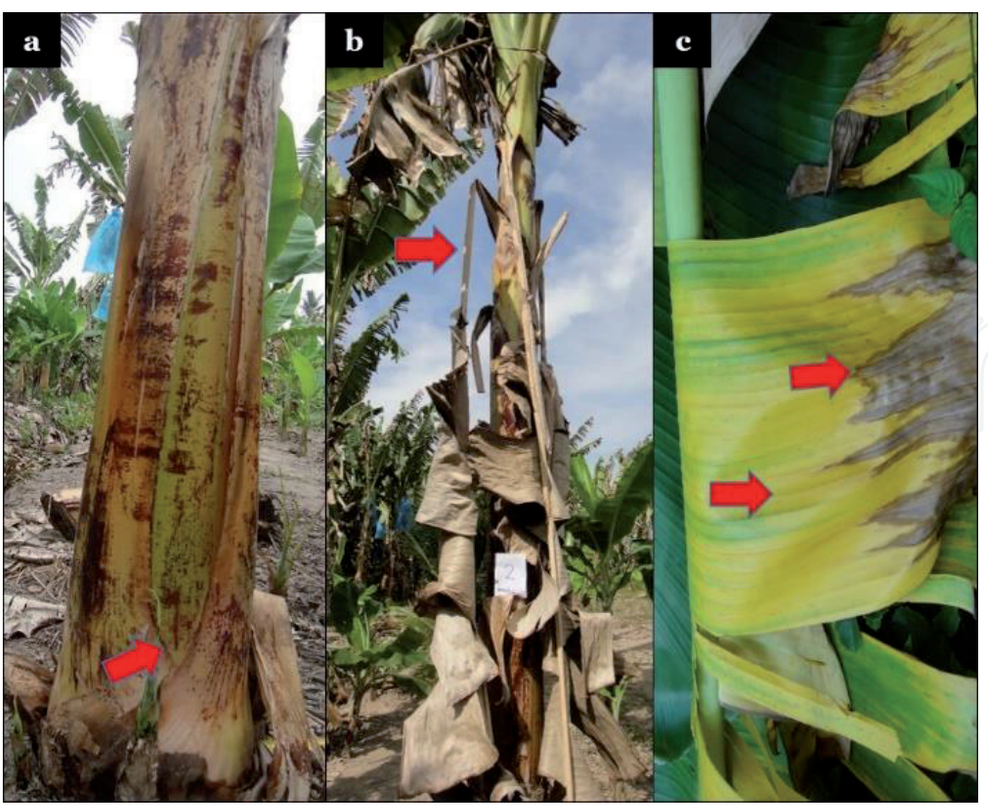

Figure 1.
External symptoms of Fusarium wilt in cv. "Berangan" banana cultivated in a plantation located in Selangor state, Malaysia (3°48'10.1"N 100°50'42.5"E). (a) Split pseudostem. (b) Skirting of wilted leaves. (c) Leaves with yellow and brown streak. Red arrows indicate external symptoms observed.

5 months after infection of roots [10]. Foc and other members of *Fusarium oxysporum* (Fo) species complex produce a type of phytotoxin called fusaric acid (FA), which has been suggested as the cause of leaf chlorosis symptom [27].

Discoloration observed in the cross and longitudinal section of the rhizome, pseudostem, as well as petiole is among the internal symptoms of *Fusarium* wilt. Golden discoloration may also be observed in infected rhizome. In more severe infection, the rhizome displays total discoloration accompanied by sticky texture and rotten smell (**Figure 2**). Meanwhile, healthy banana plants produce fresh green leaves, clear cross section of petiole with no brown ring as well as white and healthy rhizome (**Figure 3**).

The first internal symptom of the disease occurs in the hair roots which are the initial sites of infection. The infection later progresses to the rhizome. In a more prominent attack, the pathogen may colonize the cortex as well as the vascular bundle of the pseudostem. The pathogen passes through the affected vessels to the new growing shoot [28]. Appearance of brown-reddish discoloration in the internal vessels of the pseudostem confirms that the pathogen has already invaded the pseudostem. The oldest leaf sheaths may also show brownish streaks [1]. In general, no internal symptom is observed in the fruits.

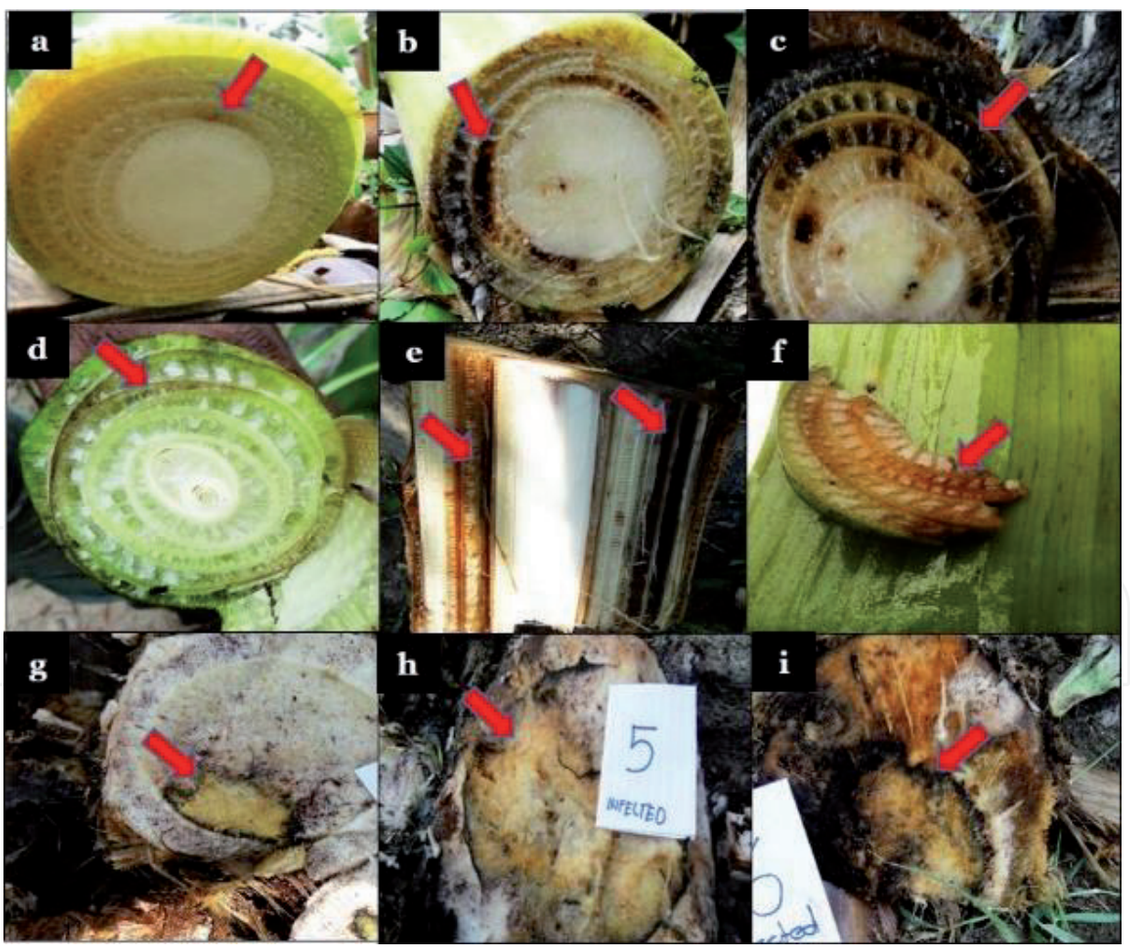

Figure 2.
Internal symptoms of Fusarium wilt in cv. "Berangan" banana cultivated in a plantation located in Selangor state, Malaysia (3°48'10.1"N 100°50'42.5"E). (a) Cross section of pseudostem showing minor discoloration. (b) Pseudostem showing moderate vascular discoloration. (c) Severe internal discoloration of the infected pseudostem. (d) Cross section of infected petiole showing minor discoloration. (e) Longitudinal section of pseudostem showing infected vascular bundle. (f) Petiole showing severe discoloration. (g) Infected rhizome showing minor golden discoloration. (h) Rhizome showing moderate discoloration. (i) Total discoloration in severely infected rhizome producing sticky texture and rotten smell. Red arrows indicate discoloration sites.

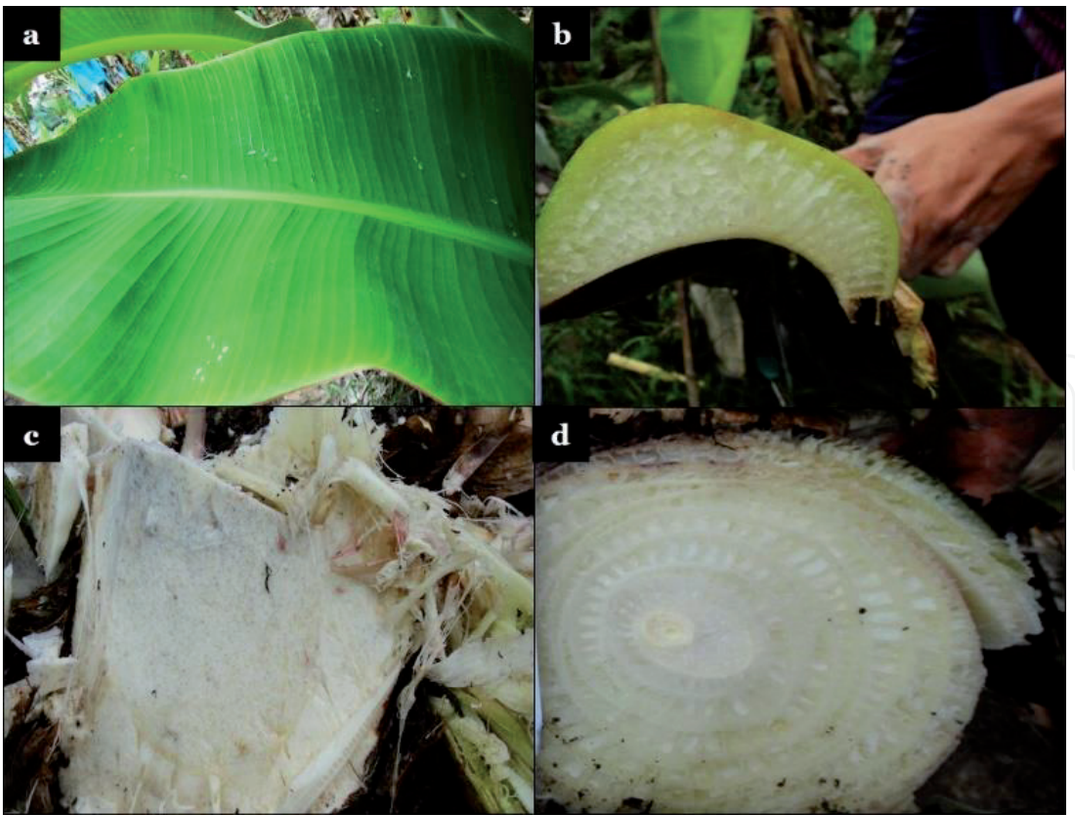

Figure 3.
Healthy banana without Fusarium wilt symptoms. (a) Fresh green leaf. (b) Clear cross section of petiole with no brown ring. (c) White and healthy rhizome. (d) Cross section of pseudostem with no brown ring.

3. *Fusarium* wilt infection

Over the years, researches have been carried out to investigate the infection process and disease cycle of *Fusarium* wilt. It was found that mycelia and conidia are produced after 6–8 hours of chlamydospore germination, while new chlamydospores will be produced after 2–3 days [28–30]. The infection is not initiated through the main root but instead takes place through secondary or tertiary feeder roots [31]. According to Beckman [32, 33], the pathogen infects the roots of both susceptible and resistant cultivars, but the infection of vascularized fragment of the rhizome is more prominent in the susceptible genotypes. In responding to the infection, tyloses, gums, and gel are produced in xylem lumen. Most of the infections are blocked, but some of them become systemic and pass through rhizome and pseudostem. The pathogen may mobilize through many different vascular pathways starting from the roots [30, 34, 35]. However, pathogen colonization in the infected rhizome is deemed the most efficient [28]. As the disease advances, the pathogen moves out of the vascular system to the adjacent parenchyma forming conidia and chlamydospores which are released to the soil when the plant died. These spores may remain dormant in the soil for years [36–38]. Ploetz [17] suggested that the pathogen's persistent survivability in the soil is contributed by the presence of living banana host.

4. Classification

Foc is a genetically and pathogenically diverse fungus. Over 20 vegetative compatibility groups (VCGs) and diverse evolutionary lineages are recognized in the global populations of the pathogen [1, 39–45]. Foc can also be classified according

to races depending on the group of cultivars they affected. To date, four races of Foc have been recognized [46–48].

Race 1 of Foc which was responsible for the "Gros Michel" epidemics also attack "Maqueno" (Maia Maoli-Popoulu subgroup, AAB), "Silk," "Pome" AAB, and "Pisang Awak" ABB. Race 2 of Foc is known to affect cooking bananas and plantains such as "Bluggoe" (ABB). On the other hand, race 3 of Foc is described as a pathogen of *Heliconia* spp., which is a tropical American banana relative [49]. It was also found to have minor impact on "Gros Michel" and seedlings of *M. balbisiana.* Race 3 Foc has not been reported since Waite's [49] work, and no voucher specimens of the pathogen exist. Of these, race 4 Foc is considered the most virulent group which does not only affect economically important cultivars like "Cavendish" but also race 1- and race 2-susceptible varieties [48, 50–52].

Race 4 Foc can be further divided into Tropical (TR4) and Subtropical (SR4) variants [50, 53]. Foc Subtropical race 4 (FocSR4) isolates cause infection on plants that are grown in an unconducive environment such as cool temperature, poor soil, and under stress condition. It also has an ability to infect banana plants regardless of the predisposing conditions [54]. Foc Tropical race 4 (FocTR4) cases have been reported in Taiwan, Indonesia, Malaysia, China, the Philippines, and Northern Australia [6]. The early outbreaks of FocTR4 in China and the Philippines were not taken seriously that they slowly developed into destructive and uncontrollable problems [38]. Recent outbreaks of FocTR4 in Mozambique and Jordan were claimed as the first reported FocTR4 cases outside the common Asian and Australian regions [6] (**Table 1**).

5. Geographical distribution

Year	Country	Description
1874	Australia	Dr. Joseph Bancroft was the first person to discover that *Fusarium* wilt was caused by a type of fungus. This was revealed using microscopic examination
1890	Panama and Costa Rica	First report in Panama and Costa Rica
1940	N/A	Snyder and Hansen suggested the pathogen be renamed as *Fusarium oxysporum* f. sp. *cubense*
1890–mid 1950s	Central and Southern America	*Fusarium* epidemic (race 1) annihilated more than 50,000 hectares of cv. "Gros Michel" plantations. "Gros Michel" cultivar was replaced with "Cavendish" as the major cultivar for trade
1911	India	*Fusarium* wilt was first reported in different countries
1916	Java, Indonesia	
1920	Philippines	
1953	Malaysia	
1967	Taiwan	
1990–present	N/A	Foc Tropical race 4 (FocTR4) severely affected the banana productions worldwide
1994	N/A	First FocTR4 case on cv. "Cavendish"
Mid-1990s	Malaysia and Indonesia	Severe *Fusarium* epidemics in Malaysia and Indonesia
1996	Panyu, Guangdong, China	First report on infected Cavendish
1997–1999	Near Darwin (northern territories), Australia	Three FocTR4 outbreaks in sites

Year	Country	Description
2000	Southern China	Epidemic severely affected cv. "Cavendish" plantations
2002	China	FocTR4 annihilated about 60,000 ha of banana plantation
2013	Oman, Jordan, and Mozambique	FocTR4 had spread outside the common Asia regions
2015	Pakistan and Lebanon	First report of FocTR4 causing Panama disease in Cavendish bananas in Pakistan and Lebanon
2018	Vietnam	First report of *Fusarium* wilt on Cavendish bananas caused by FocTR4 (VCG 01213/16) in Vietnam
2018	Laos	First report of FocTR4 (VCG 01213/16) associated with Cavendish bananas in Laos
2018	Myanmar	FocTR4 incidence was reported in Myanmar
2018	Israel	First report of FocTR4 causing *Fusarium* wilt of Cavendish bananas in Israel
2019	America	Foc TR4 incidence was reported in the Americas

Table 1.
Significant events that took place after the discovery of Fusarium wilt [8, 10, 14, 17, 18, 23–26, 55, 56].

6. Management control

The effectiveness of *Fusarium* disease management strategies has been hampered by several limitations including extremely poor fertility of certain economically important banana varieties (e.g., "Cavendish"). This has particularly hindered the improvement efforts through conventional breeding [57]. Several approaches that have been practiced to curb the infection of *Fusarium* wilt in banana are biological control, chemical control, cultural control, physical control, quarantine, exclusion and personnel awareness, breeding programs, selection of somaclonal variants, and genetic modification via transgenic approach and mutagenesis.

6.1 Biological control

Due to the continuous spread of *Fusarium* wilt, biological control offers a commendatory disease management approach [58–61]. Diverse microbes such as *Pseudomonas fluorescens*, *Trichoderma viride*, *Bacillus* spp., *Serratia* spp., *Pseudomonas aeruginosa*, *Streptomyces violaceusniger*, *Y-Proteobacteria*, *Bacillus subtilis*, *Pseudomonas* spp., and nonpathogenic *Fusarium* had been tested against *Fusarium* wilt disease in which most of the published works resulted from in vitro assays or short-term greenhouse studies [34, 59, 62–72]. Some Indian medicinal plants such as *Calotropis gigantea* L., *Centella asiatica* L., *Ocimum sanctum* L., *Piper betle* L., and *Vitex negundo* L. have also been tested in vitro as biological control against Foc. Among those, *P. betle* L. extract exhibited the highest antifungal activity against the tested plant pathogen *Foc*, followed by *V. negundo*, *C. gigantea*, *C. asiatica*, and *O. sanctum* extracts [70]. These results proved that several plant extracts exhibit antifungal activity and have potency to reduce mycelium growth under both greenhouse and field conditions [62]. Other studies reported on an application of biosynthesized silver nanoparticles to control pathogenic fungi [73]. It has also been shown that a community of *Gammaproteobacteria* is an indicator species of healthy banana plants on *Fusarium* wilt-infested and healthy fields in Nicaragua and Costa Rica [74]. These researches provide new insights on using organisms

as bioindicators. Nevertheless, there were very few studies done on the long-term biocontrol efficacy of *Fusarium* wilt in banana [2, 75]. For example, Thangavelu and Jayanthi [76] reported the field application of a selected nonpathogenic (np) Fo isolate, Ro-3, which reduced the severity of the disease up to 89% after three rounds of treatments.

On the other hand, Belgrove et al. [77] reported that neither the nonpathogenic *F. oxysporum* and *P. fluorescens* nor combinations thereof reduced the development of *Fusarium* wilt significantly.

6.2 Chemical control

Nel et al. [78] reported that prochloraz and propiconazole notably inhibited mycelial growth. In the same study, it was found that the disease symptoms of *Fusarium* wilt can be controlled up to 80.6% when benomyl and the demethylation-inhibiting fungicide were applied via root dip treatment. Besides, certain quaternary ammonium compounds were also reported as potent sterilants against Foc [78]. Similar to biological control, future field evaluations on the efficacy of the fungicides against *Fusarium* wilt are deemed necessary.

Test injections of fungicides into *Fusarium* banana plants had also been performed. In a study conducted by Lakshmanan et al. [79], rhizome of cv. "Rasthali" was injected with 2% carbendazim (Bavistin 50 WP) using hypodermic syringe. The upper portion of the rhizome was exposed by removing a portion of soil, and a slope hole at angle 45° was made before it was injected with the fungicide. The method managed to reduce the disease incidence up to 13.5% at the time of harvest.

In another study, Herbert and Marx [80] reported that the injection with carbendazim and several other fungicides did not manage to prevent the *Fusarium* wilt infection of banana in South Africa. In Australia, pseudostems of cv. "Williams" that were injected with 20% potassium phosphonate showed some effectiveness against Foc. However, the results were inconsistent and uncertain (Pegg, unpublished).

Herbert and Marx [80] also reported another treatment against *Fusarium* wilt using soil fumigation treated with methyl bromide. However, the fumigated areas were eventually reattacked by the pathogen causing the next fruit production not possible.

Nitrogen level in the soil may also affect the severity of *Fusarium* infection as NO_3 commonly decreases the severity of the disease. In contrast, NH_4 shows the opposite effect [2]. Interestingly to date, application of N fertilization in the field and its effect on *Fusarium* wilt has not been published. Nevertheless, it was reported from in vitro and hydroponic studies that high amount of NH_4 managed to block the invasion of Foc via roots [81]. The causes of these contradicting results were not elaborated.

6.3 Cultural control

As monoculture production of susceptible cultivars is difficult in infested areas, mixed plantings have been suggested as an alternative. Mixed plantings in small-scale or subsistence agriculture, in which diverse banana cultivars are planted with different crops, often develop more moderate losses than if they had been planted in monocultures [10]. Planting of Foc-tolerant or Foc-resistant cultivars accompanied by cultural practices help to improve the disease incidence and increase the crop yield [82]. Mixed culture system involving legumes, cereals, and multipurpose trees in the banana plantation can also improve the production yield of banana and develop improved tolerance against the disease [83].

6.4 Physical control

In Southern Mindanao, Philippines, and Indonesia, heat sterilization or solarization of the soil had been suggested [84]. Rice hulls were mounded on top of an affected mat and burned, supposedly generating sufficient heat to kill the pathogen. Herbert and Marx [80] stated that solarization reduced *Fusarium* wilt disease for one cycle only when it was combined with methyl bromide fumigation showing that the impact of solarization alone was not significant. In another study in Indonesia, a 6-month delay in symptom development was reported after 10 months of solarization [85].

In 1939, Dunlap used flood fallow (a method to eradicate soilborne pathogen by flooding the land) to rejuvenate Foc-infested soil [10]. However, flooded soil was rapidly recolonized by the pathogen and became impractical due to high cost needed especially in cost of labor and machinery. Thus, this method was stopped. In contrast, this measure had been commonly used in eliminating Moko disease pathogen, *Ralstonia solanacearum*, and the burrowing nematode, *Radopholus similis*, from infested soil [10].

6.5 Quarantine, exclusion, and personnel awareness

Foc is a recalcitrant pathogen that will remain in the soil for years. Thus, in halting the spread of Foc to non-infected areas, systematic quarantine and exclusion procedures are required. Stakeholders must also be educated on the infection cycle and effects of *Fusarium* wilt on banana production. This can be executed through regional awareness programs, personal communication with farmers, congresses, and contingency trainings. For example, such programs had been discussed and conducted in Latin America and the Caribbean by Bioversity International [86].

On personal level, each personnel working in the farm must be aware of the measures that they have to take in preventing the spread of *Fusarium* wilt. Some disinfectants have been found to be effective in sterilizing the farming tools, equipment, and footwear such as Sporekill (poly dimethyl ammonium chloride), Jik (sodium hypochlorite) and Prazin agri (polymeric biquanidine hydrochloride and quaternary ammonium compound) [78].

6.6 Breeding programs

The first banana breeding program involving crossbreeding of banana plantains for *Fusarium* wilt tolerance was initiated in Trinidad in 1922. The program focused on the process of crossing, screening, testing, selecting, and identifying promising hybrids for market release [87]. Generally, the success of banana breeding programs was hindered by a few barriers such as the diploid parents were lacking good agronomic and high-quality fruit traits. Other than that, low fertility of cultivar, genetic abnormalities in the parental lines, as well as the laborious duration taken for stable integration of disease resistance trait to take place from parents into the next generations contribute to the problems [88].

6.7 Transgenic research

Increasing number of publications on the development of transgenic bananas for *Fusarium* wilt tolerance were observed in the recent years probably driven by the release of whole genome sequencing of diploid banana DH Pahang [89] (**Table 2**).

Most of the published studies only reported the transgenic plants' performance under greenhouse condition. A field trial of transgenic "Cavendish" conducted in a Foc-infested area in Northern Territory of Australia was among the very few

site studies published. The 3-year trial concluded that two lines of their transgenic "Cavendish" were able to resist Foc infection. One of the lines carried a disease resistance gene, RGA2 which was derived from a TR4-resistant diploid banana, while the other line overexpressed Ced9 isolated from nematode [102, 103].

Cultivar	Foc isolate	Description	Reference
Taijiao (AAA)	Race 4	• Human lysosome was used as the donor gene • Corm slice was used as the explant. Transformation was carried out via both particle bombardment and *Agrobacterium*-mediated transformation	[90]
Rasthali (AAB)	N/A	• Chitinase and β-1,3-glucanase were transformed into tiny single meristem bud of Rasthali via co-bombardment	[91]
Rasthali (AAB)	Race 1	• β-1,3-glucanase from *Glycine max* (soybean) was introduced into *Agrobacterium tumefaciens* and later transformed into single bud of Rasthali	[92]
Pei Chiao (AAA) and Gros Michel (AAA)	Race 4	• Multiple bud clumps (MBC) were used as explants for *Agrobacterium*-mediated transformation. The genes of interest were Arabidopsis root-type ferredoxin gene (Atfd3) isolated from *Arabidopsis thaliana* and ferredoxin-like protein (pflp)	[93]
Lady Finger	Race 1 (VCG 0124/5)	• Bcl-xL, Ced-9, and Bcl-2 3' UTR genes originated from chicken, *C. elegans*, and human, respectively, were used as donor genes. The genes were integrated into the banana genome via co-cultivation of *Agrobacterium*-embryogenic cell suspension	[94]
Rasthali (AAB)	Race 1	• Petunia floral defensins, PhDef1, and PhDef2 were introduced into *A. tumefaciens* and later co-cultivated with embryogenic cell suspension cultures of Rasthali	[95]
Furenzhi (AA)	Race 4	• Endochitinase gene chi42 isolated from *Trichoderma harzianum* was integrated into banana genome via co-cultivation of *A. tumefaciens* and embryogenic cell suspension of Furenzhi banana	[96]
Rasthali (AAB)	Race 1	• Intron hairpin RNA-mediated expression of two important Foc genes, *Fusarium* transcription factor 1 (ftf1) and velvet (vel), were constructed and expressed in banana cells via co-cultivation of *Agrobacterium*-embryogenic cell suspension of Rasthali	[97]
Rasthali (AAB)	Race 1	• Three different constructs harboring endogenous cell-death related genes (*MusaDAD1*, *MusaBAG1*, and *MusaBl1*) were prepared and introduced into *A. tumefaciens*. Transformation was performed via co-cultivation of *A. tumefaciens* culture and embryogenic cell suspension of Rasthali	[98]
Rasthali (AAB)	Race 1	• Seed defensin gene (Sm-AMP-D1) isolated from *Stellaria media* (common chickweed) acted as donor gene • Gene of interest was integrated via co-cultivation of *A. tumefaciens* and embryogenic cell suspension of Rasthali	[99]
Rasthali (AAB)	Race 1	• Antimicrobial peptide (*Ace-AMP1*) derived from onion seeds was used as donor gene • *Ace-AMP1* was cloned into pCAMBIA2301 and later introduced into *A. tumefaciens* • Embryogenic cell suspension of cultivar Rasthali was developed using male floral meristems and used as explant	[100]

Cultivar	Foc isolate	Description	Reference
Nangka (AAB)	Race 4	• Thaumatin-like protein (tlp) gene derived from *Oryza sativa* (rice) was used as donor gene • Particle bombardment was performed on cauliflower-like bodies of cultivar Nangka	[101]

Table 2.
Transgenic research for development of Fusarium-tolerant bananas.

Performing field trials present several difficulties which might explain the limited number of field evaluations performed on the newly developed cultivars. Among the most apparent bottleneck is the arduous process in generating and selecting the most promising lines which involved several stages starting from laboratory, greenhouse evaluation, and lastly field assessment. One has to regenerate as many lines in the laboratory since each stage would eliminate a number of unsuccessful lines leaving with only the most performing candidates for field study. The researchers may also face difficulties in finding a suitable trial site, obtaining the regulator's approval for biosafety clearance as well as convincing the locals on the project. Based on personal communication, some plantation owners refused to cooperate because they assume that the research will harm their plants and affect the gross yield. Even when the trial has commenced, researchers may still face unexpected event that could bring the trial to a halt. For example, [102, 103] initially planned for a 5-year trial to evaluate the performance of their transgenic bananas against *Fusarium* infection. Unsuccessfully, the trial was only carried out for 3 years due to invasion of another disease.

6.8 Somaclonal variation

Somaclonal variation is defined as genetic variability undergone by tissue-cultured generated plantlets [104]. Somaclonal variation instigated by in vitro micropropagation is commonly known as tissue-induced variation [105], and it occurs as a result of strong stress experienced by the in vitro-cultured plantlets [106]. Factors that contribute to somaclonal variation had been well-reviewed over the past few decades [104, 106]. The possible causes leading to this phenomenon were observed in various plant species [107–109]. However, due to complex mechanism and high degree of variation in the results, no definite conclusion was ever made [106]. Despite all of these, Larkin and Scowcroft [106] believed that chromosomal variation plays a bigger role in generating variants among the somaclones, rather than a simple base mutation. For instance, change in ploidy level, or also known as karyotypic alteration, was among the early hypothesis posed in discussing causes of somaclonal variation at chromosomal level.

The emergence of selecting *Fusarium* wilt resistant banana clones via somaclonal variation is believed to begin in Taiwan. Initiated by Taiwan Banana Research Institute (TBRI) in 1984, the institution managed to commercialize a few resistant cultivars selected from somaclonal variants in continuous greenhouse and field trials. Starting with about 20,000 tissue-cultured cv. "Cavendish" plantlets planted in Foc-infested soil, multiple screening and selection process eliminated most of the susceptible clones displaying yellowing and wilting symptoms. Rhizomes of surviving clones were collected, trimmed, and replanted again under disease condition before six final putative resistant candidates were short-listed. Among those, GCTCV-215-1 showed the most potential and was further tested on Foc-infested plantations for its disease survivability in several places in 1990. Positive results of

the first trial incited the farmers to request the Taiwanese government to officially allow the growing of GTCV-215-1 for commercial purpose, even though the variety was not yet registered at that time. With only 17.2 and 5.2% disease incidence for plantlet-originating and suckers-originating, respectively, the new variety was probably nothing but a ray of hope to the growers who have been distressed with heavy loss in production. Even in the second trial, the clone continued to exhibit consistent result with only 4.8% disease incidence in comparison with 39.1% of parental Giant Cavendish. Promising field trials armed with positive response from both local and Japanese market led to the registration of GCTCV-215-1 as a commercial variety called Tai Chiao No. 1 in 1992 [110, 111].

Tai Chiao No. 1, however, was inferior to the parental Giant Cavendish in certain areas. The variety had weaker defense against wind/typhoon due to its taller and slender stature besides lower fruit bunch production and longer maturation period [110, 112]. Another study concerning somaclonal variation was carried out in Malaysia using micropropagated plantlets of "Rastali" cultivar. In the first round of trial conducted in 1994, micropropagated cv. "Rastali" showed 51% survival rate after being planted on Foc-infested soil for about 12 months. Nevertheless, a number of these plants developed split pseudostem, which is one of the Foc symptoms. Plants showing vigorous growth and did not produce split pseudostem were further selected, micropropagated, and tested again in 1996. The second trial produced promising results proving that the selected cv. "Rastali" exhibited high tolerance against *Fusarium* infection. The selected clone was later named as "Mutiara" following two successful harvests in a Foc-infested plantation [113]. Somaclonal variation approach was also used in screening *Fusarium*-resistant clones of other varieties such as Novaria and Berangan [113].

Regardless of its simplicity and feasibility, selecting a resistant clone from somaclonal variants posed several disadvantages [110]. Since it is a completely random process, a huge number of samples are required for screening. Uncontrollable frequency of successful clone and occurrence of epigenetic and off-type variants are also associated with somaclonal variation. It is commonly known that by gaining a desired trait, the plant might be losing some others. This was also demonstrated in banana [112]. Hwang and Tang [111] observed some poorer traits in resistant somaclone that included longer maturation period, lower fruit production, as well as reduced fruit quality.

6.9 Induced mutation

Induced mutation is another way of generating genetic variability in banana genomes. Working in a more efficient manner than spontaneous mutation, induced mutation happens at a higher frequency than the former [114]. Mutation breeding will increase the chances of genetic alteration by annulling the dominant allele and thus reviving the recessive allele [114]. Similar to somaclonal variation, induced mutation procedures required a large number of samples which at one time was the limiting factor to the execution of this technique [115]. To date, studies have employed two methods of mutagenizing genes, which are chemical and physical mutagenesis.

6.9.1 Chemical mutagenesis

In general, very few studies on the development of Foc-resistant banana through mutagenesis had been reported. Even though ethyl methanesulfonate (EMS) is one of the most common chemical mutagens used in studies involving plants [116–119], Bhagwat and Duncan [120] tested other two mutagens which were sodium azide

(NaN_3) and diethyl sulfate (DES). They recommended three different dosage treatments depending on the type of mutagens used. The recommended treatments for NaN_3, DES, and EMS were 30 min/2.3 mM, 60 min/20 mM, and 30 min/200 mM, respectively. When planted in greenhouse, 95.5% of the micropropagated clones showed infection symptoms and were eradicated. A total of 48 plants proceeded into field evaluation. There were 20, 9, and 19 plants coming from NaN_3, DES, and EMS treatment, respectively [120].

6.9.2 Physical mutagenesis

In vitro mutation can also be instigated by physical means such as gamma rays [116, 121–128]. Gamma rays are preferred due to their high penetration and high energy characteristics. Secondary radioactivity does not occur in gamma irradiation, and this makes the latter as a better choice over other physical mutagens like neutron [129]. Parameters used to measure radiation sensitivity and postirradiation recovery include survival rate, propagation, shoot height, and fresh weight which are all expressed as percentage of the control [130]. References [121, 130] used the same source of gamma irradiation, which was ^{60}Co, but with different experimental methodologies. According to Roux and Wingfield [130] irradiated seven banana clones of dissimilar ploidy level and genomic institution (Calcutta4 (AA), Kamaramasenge (AB), Tani (BB), Grande Naine (AAA), Williams (AAA), Three Hand Planty (AAB), and Cachaco (ABB)) with 10 doses of ^{60}Co ranging from 10 to 100 Gy at a dose rate of 44 Gy/min. The optimal dosage for diploid cultivars Calcutta4 (AA) and Tani (BB) was in the same range, which was 10–20 Gy of gamma irradiation. However, there were two different optimal dosages recommended for triploid clones. Triploid Cachaco (ABB) required the highest range of optimal dosage, which was 40–50 Gy, while triploids Three Hand Planty (AAB), Grande Naine (AAA), Williams (AAA), and Kamaramasenge (AB) were best treated with a lower dosage treatment of 30–40 Gy. Even though Kamaramasenge is a diploid, the cultivar has more triploid-like characteristics [130]. From the results, it was evident that lower dosage treatment was preferred as it could minimize the chromosome distortion and other harmful outcomes. Bhagwat and Duncan [121] in their experiment irradiated three different explants of Highgate cultivar with ^{60}Co gamma rays. All three types of explants, Type I dissected apices, Type II 4-week-old cultured apices, and Type III in vitro-cultured corms, were treated with different dose rates, which were 101.82, 177.37, and 256.8 rads/min, respectively. All explants were inoculated twice with *Fo* suspension, uprooted and re-cultured. The plants were observed for disease symptoms. Symptomless plants were chosen and replanted. Another examination was carried out 4 weeks later, and those with no infection symptoms were regarded as resistant to *Fusarium* wilt. Twenty clones were chosen for field evaluation but no further observation was reported.

7. Future strategies and opportunities

A comprehensive understanding on the roles of molecules in a cell is essential in driving the efforts for crop improvements. By taking advantage of systems biology approach, omics technologies extend the knowledge on complex interactions between genes, proteins, and metabolites of a particular species [131]. In short, omics technologies provide deeper insight on the modifications happening in an organism instigated by both internal and external factors. This may include changes in genetics, nutritional condition, and environment of an organism [132]. In association with the study of bioinformatics, different areas that are steered by

omics technologies include genomics, transcriptomics, proteomics, metabolomics, and phenomics [133].

Genomics, which refer to the study and analysis on gene sequences of a species, assist researchers in specifying their genes of interest for the development of plants with improved agricultural traits. With the first banana genome sequence of a double haploid *M. acuminata* spp. *malaccensis* published in 2012 [89], endogenous gene modification approaches including gene editing technologies started to emerge. A review done by Dale et al. [103] had listed a few ongoing researches on genetically modified bananas especially on "Cavendish" banana in response to FocTR4. In addition, new technologies are being invented including RNA interference (RNAi) and gene editing tools such as CRISPR-Cas for the production of bananas with improved disease resistance, better fruit yields, and higher nutritional values. However, genomics only provide general information of the genes in a particular species [131]. In contrast, transcriptomics data reveal the expression patterns of those genes under different conditions such as biotic and abiotic stresses [131]. Current trends on transcriptomic approaches include microarrays and the next-generation sequencing (NGS) [133]. Another option suggested by [134] is the RNA-Seq on transcriptome sequencing which had been adapted by [135]. Guo et al. [135] studied about the transcriptome analysis between Foc1 and Foc4 at 48 hour postinoculation on banana variety "Brazil." It was believed that the gene contents and transcriptional regulations between Foc1 and Foc4 will contribute toward improving the banana's resistance against *Fusarium* wilt in the future [135].

Another branch of omics that has been explored is the study on proteins known as proteomics. Genomics and transcriptomics analysis generate a lot of useful data. However, the approaches do not explicitly reflect the protein expression patterns. This may be due to mRNA lifetime and the presence of non-translated RNAs [133]. Translated proteins may experience posttranslational modifications (PTMs) which are one of the limitations in genomics and transcriptomics approaches [133]. Thus, proteomics take over the task to underpin the roles of a protein [136] by analyzing the alterations that took place within the cells via protein pathways. Two-dimensional electrophoresis (2-DE) and mass spectroscopy (MS) [137] are among the approaches used widely in proteomics. Particularly for Foc, [138] performed proteomic analysis by using isobaric tags for relative and absolute quantitation (i-TRAQ)-based comparative proteomic approach on conidial germination of FocTR4. Upregulated proteins identified in the ergosterol biosynthesis pathway will be useful targets in designing an effective fungicide to tackle FocTR4 [138].

Metabolomics studies involve chemical processes that link the genotypes and phenotypes [131]. This is because alteration in both transcriptome and proteome will result in changes in metabolome as metabolome is the final downstream product of gene transcription [139]. Compared to other omics, metabolomics analysis is more complicated despite having the least domains (about 5000 metabolites) as metabolome involves varied biological components [140]. In addition, metabolic profiling presents an instantaneous picture on the current activities happening in the cell which aid in greater understanding on the mode of action of pesticide [131]. Mass spectrometry (MS) and nuclear magnetic resonance (NMR) analyses used in metabolic profiling reveal more about the metabolite regulations and changes in response to different environmental conditions. The MS approach had been adapted by Li et al. [141] to study the metabolic changes, transcriptional regulation, and signaling compounds during early stage of FocTR4 infection. Despite its importance in system biology, metabolomics approach is still not widely used [133].

By providing enormous genetic information to the researches, current technologies help to escalate the crop improvement studies. However, development of new cultivars also comes with extensive phenotypic evaluations [142]. Moreover,

association of genotype to phenotype established in model systems might be deceitful [143]. In addressing this issue, plant phenomics approaches provide a noninvasive technology to study traits such as growth, performance, and composition of plants [142]. Ideally, it is hoped that the link between genotype and phenotype can eventually be described.

Unlike genotyping analysis with DNA-based molecular markers, which have been widely applied in breeding strategies, sensor-based, automated, or semiautomated phenotyping is currently underway causing delay in the plant assessment study [143]. In the study of plant pathology, several examples of sensor-based phenomics approaches include chlorophyll fluorescence imaging (CFI), multi- and hyperspectral imaging, infrared thermography (IRT), and magnetic resonance imaging (MRI) [142]. Phenomics techniques may aid in unraveling the causes of a certain plant disease as well as allowing early detection of disease-related changes in plants [142].

Omics strategies indeed provide us with gigantic amounts of data. However, the data must be validated to minimize the possibility of getting false-positive results which may affect the whole study [140]. Moving forward, it is expected that the combination between omics technology and plant breeding will generate a huge impact on crop improvement [144]. From the review done by Zhang et al. [145], multi-"omics" analyses will be the next level of omics approaches as it is able to provide more discrete and testable biological hypotheses from a large scale of high-throughput datasets. With integrated approaches, a more complex process at different levels can be analyzed, resulting in new insights to produce a more resistant species [145].

8. Conclusions

Fighting the spread of *Fusarium* wilt is a race against time. Once an area is invaded by Foc, it is almost impossible to use the area again for growing bananas. Thus, preventing the spread of *Fusarium* infection is very critical in ensuring the continuity of banana production as well as securing the nutritional supply to the consumers. In curbing the epidemics, knowledge on the etiology of *Fusarium* wilt and its infection process must be provided to the farmers and plant breeders. In addition to the current management strategies, various recent technologies introduced may shed some light into the production of *Fusarium*-resistant banana varieties thus finally putting a stop to this longstanding threat.

Acknowledgements

This work was supported by Putra Grant (Inisiatif Putra Muda) (GP-IPM/2019/9672100) financed by Universiti Putra Malaysia and Fundamental Research Grant Scheme (FRGS) (FRGS/1/2019/STG05/UPM/02/8) financed by the Ministry of Education Malaysia.

Conflict of interest

The authors declare that no conflict of interest exists.

DOI: http://dx.doi.org/10.5772/intechopen.89469

Author details

Fatin Nadiah Jamil[1], Chu-Nie Tang[2], Noor Baity Saidi[1,2], Kok-Song Lai[2,3] and Nadiya Akmal Baharum[2*]

1 Institute of Bioscience, Universiti Putra Malaysia, Serdang, Selangor, Malaysia

2 Faculty of Biotechnology and Biomolecular Sciences, Department of Cell and Molecular Biology, Universiti Putra Malaysia, Serdang, Selangor, Malaysia

3 Health Sciences Division, Abu Dhabi Women's College, Higher Colleges of Technology, Abu Dhabi, United Arab Emirates

*Address all correspondence to: nadiya_baharum@upm.edu.my

References

[1] Ploetz RC, Pegg KG. Fungal disease of the root, corm, and pseudostem: *Fusarium* wilt. In: Jones DR, editor. Diseases of Banana, Abaca and Enset. Wallingford, UK: CAB International; 2000. pp. 143-159

[2] Ploetz RC. Management of *Fusarium* wilt of banana: A review with special reference to tropical race 4. Crop Protection. 2015;**73**:7-15. DOI: 10.1016/j.cropro.2015.01.007

[3] Siamak SB, Zheng S. Banana *Fusarium* wilt (*Fusarium oxysporum* f. sp. *cubense*) control and resistance, in the context of developing wilt-resistant bananas within sustainable production systems. Horticultural Plant Journal. 2018;**4**(5):208-218. DOI: 10.1016/j.hpj.2018.08.001

[4] Butler D. Fungus threatens top banana. Nature News. 2013;**504**(7479):195. DOI: 10.1038/504195a

[5] Moore NY, Bentley S, Pegg KG, Jones DR. Musa Disease Fact Sheet No 5. *Fusarium* Wilt of Banana. Montpellier, France: INIBAP; 1995

[6] García-Bastidas F, Ordóñez N, Konkol J, Al-Qasim M, Naser Z, Abdelwali M, et al. First report of *Fusarium oxysporum* f. sp. *cubense* tropical race 4 associated with Panama disease of banana outside Southeast Asia. Plant Disease. 2014;**98**(5):694. DOI: 10.1094/PDIS-09-13-0954-PDN

[7] Ordonez N, García-Bastidas F, Laghari HB, Akkary MY, Harfouche EN, Al Awar BN, et al. First report of *Fusarium oxysporum* f. sp. *cubense* tropical race 4 causing Panama disease in Cavendish bananas in Pakistan and Lebanon. Plant Disease. 2016;**100**(1):209. DOI: 10.1094/PDIS-12-14-1356-PDN

[8] Zheng SJ, García-Bastidas FA, Li X, Zeng L, Bai T, Xu S, et al. New geographical insights of the latest expansion of *Fusarium oxysporum* f. sp. *cubense* tropical race 4 into the greater Mekong subregion. Frontiers in Plant Science. 2018;**9**:457. DOI: 10.3389/fpls.2018.00457

[9] Bancroft J. Report of the board appointed to enquire into the cause of disease affecting livestock and plants. Queensland. In: Votes and Proceedings of Legislative Assembly 1877; 1876. p. 1011-1038

[10] Stover RH. Fusarial Wilt (Panama Disease) of Banana and Other *Musa* Species. Kew, Surrey, United Kingdom: Commonwealth Mycological Institute; 1962. 117p

[11] Ploetz RC. Diseases of tropical perennial crops: Challenging problems in diverse environments. Plant Disease. 2007;**91**(6):644-663. DOI: 10.1094/PDIS-91-6-0644

[12] Ploetz RC, Pegg KG. *Fusarium* wilt of banana and Wallace's line: Was the disease originally restricted to his indo-Malayan region? Australasian Plant Pathology. 1997;**26**(4):239-249. DOI: 10.1071/AP97039

[13] Vakili NG. *Fusarium* wilt resistance in seedlings and mature plants of *Musa* species. Phytopathology. 1965;**55**(2):135-140

[14] Pérez-vicente L, Dita MA, Martínez-de La parte E. Technical manual: Prevention and diagnostic of *Fusarium* Wilt (Panama disease) of banana caused by *Fusarium oxysporum* f. sp. *cubense* tropical race 4 (TR4). Workshop Diagnosis *Fusarium* Wilt. 2014;**4**:1-74

[15] Ploetz RC. *Fusarium* wilt of banana (Panama disease). In: Mukhopadhyay AN, Chaube HS, Kumar J, Singh US, editors. Plant

Diseases of International Importance. Vol. III. Englewood Cliffs: Pretence Hall; 1992. pp. 270-282

[16] Mostert D, Molina AB, Daniells J, Fourie G, Hermanto C, Chao CP, et al. The distribution and host range of the banana *Fusarium* wilt fungus, *Fusarium oxysporum* f. sp. *cubense*, in Asia. PLoS One. 2017;**12**(7):e0181630. DOI: 10.1371/journal.pone.0181630

[17] Ploetz RC. *Fusarium* wilt of banana. Phytopathology. 2015;**105**:1512-1521. DOI: 10.1094/PHYTO-04-15-0101-RVW

[18] Ordonez N, Seidl MF, Waalwijk C, Drenth A, Kilian A, Thomma BP, et al. Worse comes to worst: Bananas and Panama disease—When plant and pathogen clones meet. PLoS Pathogens. 2015;**11**(11):e1005197. DOI: 10.1371/journal.ppat.1005197

[19] Qi YX, Zhang X, Pu JJ, Xie YX, Zhang HQ, Huang SL. Race 4 identification of *Fusarium oxysporum* f. sp. *cubense* from Cavendish cultivars in Hainan province, China. Australasian Plant Disease Notes. 2008;**3**(1):46-47. DOI: 10.1007/BF03211234

[20] Thangavelu R. Status report on Fusarium wilt disease in India. In: 2016 ISHAS ProMusa Symposium; Montpellier, France; 2016

[21] Cook DC, Taylor AS, Meldrum RA, Drenth A. Potential economic impact of Panama disease (tropical race 4) on the Australian banana industry. Journal of Plant Diseases and Protection. 2015;**122**(5-6):229-237. DOI: 10.1007/BF03356557

[22] O'Neill WT, Henderson J, Pattemore JA, O'Dwyer C, Perry S, Beasley DR, et al. Detection of *Fusarium oxysporum* f. sp. *cubense* tropical race 4 strain in northern Queensland. Australasian Plant Disease Notes. 2016;**11**(1):33. DOI: 10.1007/s13314-016-0218-1

[23] Hung TN, Hung NQ, Mostert D, Viljoen A, Chao CP, Molina AB. First report of *Fusarium* wilt on Cavendish bananas, caused by *Fusarium oxysporum* f. sp. *cubense* tropical race 4 (VCG 01213/16), in Vietnam. Plant Disease. 2018;**102**(2):448. DOI: 10.1094/PDIS-08-17-1140-PDN

[24] Chittarath K, Mostert D, Crew KS, Viljoen A, Kong G, Molina AB, et al. First report of *Fusarium oxysporum* f. sp. *cubense* tropical race 4 (VCG 01213/16) associated with Cavendish bananas in Laos. Plant Disease. 2018;**102**(2):449. DOI: 10.1094/PDIS-08-17-1197-PDN

[25] Maymon M, Shpatz U, Harel YM, Levy E, Elkind G, Teverovsky E, et al. First report of *Fusarium oxysporum* f. sp. *cubense* tropical race 4 causing *Fusarium* wilt of Cavendish bananas in Israel. Plant Disease. 2018;**102**(12):2655. DOI: 10.1094/PDIS-05-18-0822-PDN

[26] Lambert J. Alarm as Devastating Banana Fungus Reaches the Americas. Nature News. 19 August 2019. ISSN: 1476-468. Available online: https://www.nature.com/articles/d41586-019-02489-5

[27] Dong X, Ling N, Wang M, Shen Q, Guo S. Fusaric acid is a crucial factor in the disturbance of leaf water imbalance in *Fusarium*-infected banana plants. Plant Physiology and Biochemistry. 2012;**60**:171-179. DOI: 10.1016/j.plaphy.2012.08.004

[28] Li C, Yang J, Li W, Sun J, Peng M. Direct root penetration and rhizome vascular colonization by *Fusarium oxysporum* f. sp. *cubense* are the key steps in the successful infection of Brazil Cavendish. Plant Disease. 2017;**101**(12):2073-2078. DOI: 10.1094/PDIS-04-17-0467-RE

[29] Guo L, Yang L, Liang C, Wang G, Dai Q, Huang J. Differential colonization patterns of bananas (Musa spp.) by physiological race 1 and race

4 isolates of *Fusarium oxysporum* f. sp. *cubense*. Journal of Phytopathology. 2015;**163**(10):807-817. DOI: 10.1111/jph.12378

[30] Li C, Chen S, Zuo C, Sun Q, Ye Q, Yi G, et al. The use of GFP-transformed isolates to study infection of banana with *Fusarium oxysporum* f. sp. *cubense* race 4. European Journal of Plant Pathology. 2011;**131**(2):327-340. DOI: 10.1007/s10658-011-9811-5

[31] Trujillo EE. Pathological-anatomical studies of Gros Michel banana affected by *Fusarium* wilt. Phytopathology. 1963;**53**(2):162

[32] Beckman CH. The Nature of Wilt Diseases of Plants. St Paul: America Phytopathological Press; 1987

[33] Beckman CH. Host responses to the pathogen. In: Ploetz RC, editor. Fusarium Wilt of Banana. USA: APS Press; 1990. pp. 93-105

[34] Ho YN, Chiang HM, Chao CP, Su CC, Hsu HF, Guo CT, et al. In planta biocontrol of soilborne *Fusarium* wilt of banana through a plant endophytic bacterium, *Burkholderia cenocepacia* 869T2. Plant and Soil. 2015;**387**(1-2):295-306. DOI: 10.1007/s11104-014-2297-0

[35] Zhang L, Yuan T, Wang Y, Zhang D, Bai T, Xu S, et al. Identification and evaluation of resistance to *Fusarium oxysporum* f. sp. *cubense* tropical race 4 in *Musa acuminata* Pahang. Euphytica. 2018;**214**(7):106. DOI: 10.1007/s10681-018-2185-4

[36] Stover RH. Studies on *Fusarium* wilt of bananas: IX. Competitive saprophytic ability of *F. oxysporum* f. *cubense*. Canadian Journal of Botany. 1962;**40**(11):1473-1481

[37] Simmonds NW. Bananas. 2nd ed. London: Longmans; 1966. p. 512

[38] Buddenhagen I. Understanding strain diversity in *Fusarium oxysporum* f. sp. *cubense* and history of introduction of 'tropical race 4' to better manage banana production. Acta Horticulturae. 2009;**828**:193-204. DOI: 10.17660/ActaHortic.2009.828.19

[39] Bentley S, Pegg KG, Dale JL. Genetic variation among a world-wide collection of isolates of *Fusarium oxysporum* f. sp. *cubense* analysed by RAPD-PCR fingerprinting. Mycological Research. 1995;**99**(11):1378-1384. DOI: 10.1016/S0953-7562(09)81225-2

[40] Bentley S, Pegg KG, Moore NY, Davis RD, Buddenhagen IW. Genetic variation among vegetative compatibility groups of *Fusarium oxysporum* f. sp. *cubense* analyzed by DNA fingerprinting. Phytopathology. 1998;**88**(12):1283-1293. DOI: 10.1094/PHYTO.1998.88.12.1283

[41] Boehm EW, Ploetz RC, Kistler HC. Statistical analysis of electrophoretic karyotype variation among vegetative compatibility groups of *Fusarium oxysporum* f. sp. *cubense*. Molecular Plant-Microbe Interactions. 1994;7(2):196-207

[42] Fourie G, Steenkamp ET, Ploetz RC, Gordon TR, Viljoen A. Biology and evolution of *Fusarium oxysporum*, with specific reference to the banana wilt pathogen. Infection, Genetics and Evolution. 2011;**11**:533-542

[43] Koenig RL, Ploetz RC, Kistler HC. *Fusarium oxysporum* f. sp. *cubense* consists of a small number of divergent and globally distributed clonal lineages. Phytopathology. 1997;**87**(9):915-923. DOI: 10.1094/PHYTO.1997.87.9.915

[44] O'Donnell K, Kistler HC, Cigelnik E, Ploetz RC. Multiple evolutionary origins of the fungus causing Panama disease of banana: Concordant evidence from nuclear and mitochondrial gene

genealogies. Proceedings of the National Academy of Sciences. 1998;**95**(5):2044-2049. DOI: 10.1073/pnas.95.5.2044

[45] Islands C, Province M. Vegetative compatibility among races of *Fusarium oxysporum* f. sp. *cubense*. Plant Disease. 1988;**72**:325-328

[46] Stover RH. Fusarium wilt of banana: some history and current status of the disease. In: Ploetz RC, editor. Fusarium wilt of banana. Minnesota: American Phytopathological Society; 1990. p. 1-7

[47] Stover RH, Buddenhagen IW. Banana breeding: Polyploidy, disease resistance and productivity. Fruits. 1986;**41**(3):175-214

[48] Stover RH, Simmonds NW. Bananas. Tropical Agricultural Series. Vol. 10. New York: John Wiley and Sons, Inc.; 1987. pp. 158-468

[49] Waite BH. Wilt of *Heliconia* spp. caused by *Fusarium oxysporum* f. sp. *cubense* race 3. Tropical Agriculture Trinidad. 1963;**40**:299-305

[50] Leong SK, Latiffah Z, Baharuddin S. Genetic diversity of *Fusarium oxysporum* f. sp. *cubense* isolates from Malaysia. African Journal of Microbiology Research. 2010;**4**(11):1026-1037

[51] Mayers PE. *Fusarium* wilt of Cavendish bananas in Queensland. In: Liddell C, editor. Proceedings of the International *Fusarium* Workshop. Australia: The University of Syndey 5; 1983. p. 60

[52] Su HJ, Hwang SC, Ko WH. Fusarial wilt of Cavendish bananas in Taiwan. Plant Disease. 1986;**70**(9):814-818

[53] Ploetz RC. *Fusarium* wilt of banana is caused by several pathogens referred to as *Fusarium oxysporum* f. sp. *cubense*. Phytopathology. 2006;**96**(6):653-656. DOI: 10.1094/PHYTO-96-0653

[54] Thangavelu R, Mustaffa MM. Current advances in the Fusarium wilt disease management in banana with emphasis on biological control. In: Cumagun CJ, editor. Plant Pathology. Rijeka: Intech; 2012. pp. 273-298

[55] Ploetz RC. Panama disease: An old nemesis rears its ugly head: Part 1. The beginnings of the banana export trades. Plant Health Progress. 2005;**6**(1):18. DOI: 10.1094/PHP-2005-1221-01-RV

[56] Snyder WC, Hansen HN. The species concept in *Fusarium*. American Journal of Botany. 1940;**27**(2):64-67. DOI: 10.1002/j.1537-2197.1940.tb14217.x

[57] Morán JF. Improvement of Cavendish banana cultivars through conventional breeding. Acta Horticulturae. 2013;**986**:205-208. DOI: 10.17660/ActaHortic.2013.986.21

[58] Fravel D, Olivain C, Alabouvette C. *Fusarium oxysporum* and its biocontrol. The New Phytologist. 2003;**157**(3):493-502. DOI: 10.1046/j.1469-8137.2003.00700.x

[59] Jie L, Zifeng W, Lixiang C, Hongming T, Patrik I, Zide J, et al. Artificial inoculation of banana tissue culture plantlets with indigenous endophytes originally derived from native banana plants. Biological Control. 2009;**51**(3):427-434. DOI: 10.1016/j.biocontrol.2009.08.002

[60] Gang G, Bizun W, Weihong M, Xiaofen L, Xiaolin Y, Chaohua Z, et al. Biocontrol of *Fusarium* wilt of banana: Key influence factors and strategies. African Journal of Microbiology Research. 2013;**7**(41):4835-4843. DOI: 10.5897/AJMR2012.2392

[61] Raza W, Ling N, Zhang R, Huang Q, Xu Y, Shen Q. Success evaluation of

the biological control of *Fusarium* wilts of cucumber, banana, and tomato since 2000 and future research strategies. Critical Reviews in Biotechnology. 2017;**37**(2):202-212. DOI: 10.3109/07388551.2015.1130683

[62] Akila R, Rajendran L, Harish S, Saveetha K, Raguchander T, Samiyappan R. Combined application of botanical formulations and biocontrol agents for the management of *Fusarium oxysporum* f. sp. *cubense* (*Foc*) causing *Fusarium* wilt in banana. Biological Control. 2011;**57**(3):175-183. DOI: 10.1016/j.biocontrol.2011.02.010

[63] Getha K, Vikineswary S, Wong WH, Seki T, Ward A, Goodfellow M. Evaluation of *Streptomyces* sp. strain g10 for suppression of *Fusarium* wilt and rhizosphere colonization in pot-grown banana plantlets. Journal of Industrial Microbiology and Biotechnology. 2005;**32**(1):24-32. DOI: 10.1007/s10295-004-0199-5

[64] Getha K, Vikineswary S. Antagonistic effects of *Streptomyces violaceusniger* strain G10 on *Fusarium oxysporum* f. sp. *cubense* race 4: Indirect evidence for the role of antibiosis in the antagonistic process. Journal of Industrial Microbiology and Biotechnology. 2002;**28**(6):303-310. DOI: 10.1038/sj/jim/7000247

[65] Thangavelu R, Mustaffa MM. First report on the occurrence of a virulent strain of *Fusarium* wilt pathogen (Race-1) infecting Cavendish (AAA) group of bananas in India. Plant Disease. 2010;**94**(11):1379. DOI: 10.1094/PDIS-05-10-0330

[66] Ting AS, Meon S, Kadir J, Radu S, Singh G. Endophytic microorganisms as potential growth promoters of banana. BioControl. 2008;**53**(3):541-553. DOI: 10.1007/s10526-007-9093-1

[67] Ting AS, Mah SW, Tee CS. Prevalence of endophytes antagonistic towards *Fusarium oxysporum* f. sp. *cubense* race 4 in various plants. American-Eurasian Journal of Sustainable Agriculture. 2009;**3**(3):399-406

[68] Ting AS, Mah SW, Tee CS. Detection of potential volatile inhibitory compounds produced by endobacteria with biocontrol properties towards *Fusarium oxysporum* f. sp. *cubense* race 4. World Journal of Microbiology and Biotechnology. 2011;**27**(2):229-235. DOI: 10.1007/s11274-010-0447-y

[69] Thangavelu R, Palaniswami A, Velazhahan R. Mass production of *Trichoderma harzianum* for managing *Fusarium* wilt of banana. Agriculture, Ecosystems and Environment. 2004;**103**(1):259-263. DOI: 10.1016/j.agee.2003.09.026

[70] Mohammed AM, AL-Ani LK, Bekbayeva L, Salleh B. Biological control of *Fusarium oxysporum* f. sp. *cubense* by *Pseudomonas fluorescens* and BABA *in vitro*. World Applied Sciences Journal. 2011;**15**(2):189-191

[71] Gnanasekaran P, Salique SM, Panneerselvam A, Uma Maheswari K. *In vitro* biological control of *Fusarium oxysporum* f. sp. *cubense* by using some Indian medicinal plants. International Journal of Current Research and Academic Review. 2015;**3**:107-116

[72] Sekhar AC, Thomas P. Isolation and identification of shoot-tip associated endophytic bacteria from banana cv. Grand Naine and testing for antagonistic activity against *Fusarium oxysporum* f. sp. *cubense*. American Journal of Plant Sciences. 2015;**6**(07):943-954. DOI: 10.4236/ajps.2015.67101

[73] Ali SM, Yousef NM, Nafady NA. Application of biosynthesized silver nanoparticles for the control of land snail *Eobania vermiculata* and some plant pathogenic fungi. Journal of Nanomaterials. 2015;**2015**:3. DOI: 10.1155/2015/218904

[74] Köberl M, Dita M, Martinuz A, Staver C, Berg G. Members of Gammaproteobacteria as indicator species of healthy banana plants on *Fusarium* wilt-infested fields in Central America. Scientific Reports. 2017;**7**(45318):1-9. DOI: 10.1038/srep45318

[75] Ploetz RC. Biological control of *Fusarium* wilt: A review and an evaluation. In: Abstract Booklet, International Congress on Musa: Harnessing Research to Improve Livelihoods. International Network for the Improvement of Banana and Plantain (Bioversity International) Online Publication; 2004. p. 141

[76] Thangavelu R, Jayanthi A. RFLP analysis of rDNA-ITS regions of native non-pathogenic *Fusarium oxysporum* isolates and their field evaluation for the suppression of *Fusarium* wilt disease of banana. Australasian Plant Pathology. 2009;**38**(1):13-21. DOI: 10.1071/AP08071

[77] Belgrove A, Steinberg C, Viljoen A. Evaluation of nonpathogenic *Fusarium oxysporum* and *Pseudomonas fluorescens* for Panama disease control. Plant Disease. 2011;**95**(8):951-959. DOI: 10.1094/PDIS-06-10-0409

[78] Nel B, Steinberg C, Labuschagne N, Viljoen A. Evaluation of fungicides and sterilants for potential application in the management of *Fusarium* wilt of banana. Crop Protection. 2007;**26**(4):697-705. DOI: 10.1016/j.cropro.2006.06.008

[79] Lakshmanan P, Selvaraj P, Mohan S. Efficacy of different methods for the control of Panama disease. Tropical Pest Management. 1987;**33**(4):373-374. DOI: 10.1080/09670878709371187

[80] Herbert JA, Marx D. Short-term control of Panama disease of bananas in South Africa. Phytophylactica. 1990;**22**(3):339-340

[81] Zhang MX, Zhang MC, Chen P, Ruan Y, Zhu YY, Shen QR. Influence of nitrate/ammonium ratio on the plant growth of banana and related wilt disease development. Journal of Plant Nutrition and Fertilizer. 2013;**19**:1241-1247

[82] Haddad F, Rocha LS, Soares AC, Martins IP, Teixeira LA, Staver C, Dita M. Management of Fusarium wilt of bananas in Minas Gerais, Brazil. Acta Horticulturae 1196; 2016. pp. 137-146. DOI: 10.17660/ActaHortic.2018.1196.16

[83] Dowiya NB, Rweyemamu CL, Maerere AP. Banana (*Musa* spp. Colla) cropping systems, production constraints and cultivar preferences in eastern Democratic Republic of Congo. Journal of Animal and Plant Sciences. 2009;**4**(2):341-356

[84] Molina A, Williams R, Hermanto C, Suwanda M, Komolong B, Kokoa P. Mitigating the threat of banana *Fusarium* wilt: Understanding the agroecological distribution of pathogenic forms and developing disease management strategies. Australia: Australian Centre for International Agricultural Research, GPO Box. 2010;**1571**:2010-2012

[85] Hermanto C, Djatnika I, Emilda D. Pre-planting treatments for management of banana *Fusarium* wilt. Journal of Agricultural and Biological Science. 2012;**7**(4):260-265

[86] Pocasangre LE, Ploetz RC, Molina AB, Vicente LP. Raising awareness of the threat of *Fusarium* wilt tropical race 4 in Latin America and the Caribbean. 2011;**897**:331-337. DOI: 10.17660/ActaHortic.2011.897.45

[87] Ortiz R. Conventional banana and plantain breeding. In: Amorim EP, Pires de Matos, Ven den Gergh I, editors. VII International Symposium on Banana: ISHS-ProMusa Symposium on Bananas

and Plantains: Towards Sustainable Global Production 986; 10-14 October 2011; Salvador, Brazil, Belgium: International Society for Horticultural Science; 2013. pp. 177-194. DOI: 10.17660/ ActaHortic.2013.986.19

[88] Ortiz R, Swennen R. From crossbreeding to biotechnology-facilitated improvement of banana and plantain. Biotechnology Advances. 2014;**32**(1):158-169. DOI: 10.1016/j.biotechadv.2013.09.010

[89] D'Hont A, Denoeud F, Aury JM, Baurens FC, Carreel F, Garsmeur O, et al. The banana (*Musa acuminata*) genome and the evolution of monocotyledonous plants. Nature. 2012;**488**(7410):213-217. DOI: 10.1038/nature11241

[90] Pei XW, Chen SK, Wen RM, Ye S, Huang JQ, Zhang YQ, et al. Creation of transgenic bananas expressing human lysozyme gene for Panama wilt resistance. Journal of Integrative Plant Biology. 2005;**47**(8):971-977. DOI: 10.1111/j.1744-7909.2005.00141.x

[91] Sreeramanan S, Maziah M, Abdullah MP, Sariah M, Xavier R. Transient expression of *gusA* and *gfp* gene in *Agrobacterium*-mediated banana transformation using single tiny meristematic bud. Asian Journal of Plant Sciences. 2006;**5**(3):468-480. DOI: 10.3923/ajps.2006.468.480

[92] Maziah M, Sariah M, Sreeramanan S. Transgenic banana Rastali (AAB) with *β-1, 3-glucanase* gene for tolerance to *Fusarium* wilt race 1 disease via *Agrobacterium*-mediated transformation system. The Plant Pathology Journal. 2007;**6**:271-282. DOI: 10.1007/s11816-011-0179-y

[93] Yip MK, Lee SW, Su KC, Lin YH, Chen TY, Feng TY. An easy and efficient protocol in the production of *pflp* transgenic banana against *Fusarium* wilt. Plant Biotechnology Reports. 2011;**5**(3):245-254

[94] Paul JY, Becker DK, Dickman MB, Harding RM, Khanna HK, Dale JL. Apoptosis-related genes confer resistance to *Fusarium* wilt in transgenic 'lady finger' bananas. Plant Biotechnology Journal. 2011;**9**(9):1141-1148. DOI: 10.1111/j.1467-7652.2011.00639.x

[95] Ghag SB, Shekhawat UK, Ganapathi TR. Petunia floral defensins with unique prodomains as novel candidates for development of *Fusarium* wilt resistance in transgenic banana plants. PLoS One. 2012;**7**(6):e39557. DOI: 10.1371/journal.pone.0039557

[96] Hu CH, Wei YR, Huang YH, Yi GJ. An efficient protocol for the production of *chit42* transgenic Furenzhi banana (*Musa* spp. AA group) resistant to *Fusarium oxysporum*. In Vitro Cellular and Developmental Biology. Plant. 2013;**49**(5):584-592. DOI: 10.1007/s11627-013-9525-9

[97] Ghag SB, Shekhawat UK, Ganapathi TR. Host-induced post-transcriptional hairpin RNA-mediated gene silencing of vital fungal genes confers efficient resistance against *Fusarium* wilt in banana. Plant Biotechnology Journal. 2014;**12**(5):541-553. DOI: 10.1111/pbi.12158

[98] Ghag SB, Shekhawat UK, Ganapathi TR. Native cell-death genes as candidates for developing wilt resistance in transgenic banana plants. Annals of Botany Plants. 2014;**6**:plu1037. DOI: 10.1093/aobpla/plu037

[99] Ghag SB, Shekhawat UK, Ganapathi TR. Transgenic banana plants expressing a *Stellaria media* defensin gene (*Sm-AMP-D1*) demonstrate improved resistance to *Fusarium oxysporum*. Plant Cell, Tissue

and Organ Culture. 2014;**119**(2):247-255. DOI: 10.1007/s11240-014-0529-x

[100] Mohandas S, Sowmya HD, Saxena AK, Meenakshi S, Rani RT, Mahmood R. Transgenic banana cv. Rasthali (AAB, silk gp) harboring *Ace-AMP1* gene imparts enhanced resistance to *Fusarium oxysporum* f. sp. *cubense* race 1. Scientia Horticulturae. 2013;**164**:392-399. DOI: 10.1016/j.scienta.2013.09.018

[101] Mahdavi F, Sariah M, Maziah M. Expression of rice thaumatin-like protein gene in transgenic banana plants enhances resistance to *Fusarium* wilt. Applied Biochemistry and Biotechnology. 2012;**166**(4):1008-1019. DOI: 10.1007/s12010-011-9489-3

[102] Dale J, James A, Paul JY, Khanna H, Smith M, Peraza-Echeverria S, et al. Transgenic Cavendish bananas with resistance to *Fusarium* wilt tropical race 4. Nature Communications. 2017;**8**(1):1496. DOI: 10.1038/s41467-017-01670-6

[103] Dale J, Paul JY, Dugdale B, Harding R. Modifying bananas: From transgenics to organics? Sustainability. 2017;**9**(3):333. DOI: 10.3390/su9030333

[104] Lee M, Phillips RL. The chromosomal basis of somaclonal variation. Annual Review of Plant Physiology and Plant Molecular Biology. 1988;**39**(1):413-437

[105] Kaeppler SM, Kaeppler HF, Rhee Y. Epigenetic aspects of somaclonal variation in plants. In: Matzke MA, Matzke AJM, editors. Plant Gene Silencing. Dordrecht: Springer; 2000. pp. 59-68. DOI: 10.1007/978-94-011-4183-3_4

[106] Larkin PJ, Scowcroft WR. Somaclonal variation—A novel source of variability from cell cultures for plant improvement. Theoretical and Applied Genetics. 1981;**60**(4):197-214. DOI: 10.1007/BF02342540

[107] Krishnamurthi M, Tlaskal J. Fiji disease resistant *Saccharum officinarum* var. Pindar subclones from tissue cultures. Proceedings of International Society Sugar Cane Technologists. 1974;**15**(1):130-137

[108] Liu MC, Chen WH. Tissue and cell culture as aids to sugarcane breeding. I. Creation of genetic variation through callus culture. Euphytica. 1976;**25**(1):393-403. DOI: 10.1007/BF00041572

[109] Nishi T, Yamada Y, Takahashi E. Organ redifferentiation and plant restoration in rice callus. Nature. 1968;**219**(5153):508-509. DOI: 10.1038/219508a0

[110] Hwang SC, Ko WH. Cavendish banana cultivars resistant to *Fusarium* wilt acquired through somaclonal variation in Taiwan. Plant Disease. 2004;**88**(6):580-588. DOI: 10.1094/PDIS.2004.88.6.580

[111] Hwang SC, Tang CY. Unconventional banana breeding in Taiwan. In: Disease of Banana, Abaca and Enset. Wallingford: Centre for Agriculture and Biosciences International Publishing; 1999. 544p

[112] Tang CY. Somaclonal variation: A tool for the improvement of Cavendish banana cultivars.In: Chang CW, Drew R, editors. II International Symposium on Biotechnology of Tropical and Subtropical Species 692; 5 November 2001; Taiwan; Belgium: International Society for Horticultural Science; 2005. pp. 61-66. DOI: 10.17660/ActaHortic.2005.692.6

[113] Ho YW, Mak C, Liew KW. Selection of banana cultivars for tolerance to fusarium wilt race 4 in Malaysia.

In: Molina AB, Nik Masdek NH, Liew KW, editors. Banana *Fusarium* Wilt Management: Towards Sustainable Cultivation; International Network for the Improvement of Bananas and Plantains—Asia and the Pacific Network; Los Banos, Laguna, Philippines; 2001. pp. 234-242

[114] Jain SM, Swennen R. Banana improvement: Cellular, molecular biology, and induced mutations. In: Proceedings from a Meeting Held on 24-28 September 2001; Enfield, NH, USA: Science Publishers Inc.; 2004. p. 382

[115] Jain SM. In vitro mutagenesis in Banana (Musa spp.) improvement. In: IV International Symposium on Banana; International Conference on Banana and Plantain in Africa. Harnessing International 879; 2008. pp. 605-614. DOI: 10.17660/ActaHortic.2010.879.67

[116] Epp D. Somaclonal variation in banana: A case study with Fusarium wilt. In: Persley GJ, De Langhe EA, editors. Banana and Plantain Breeding Strategies. Canberra: Australian Centre for International Agriculture Research; 1987. pp. 140-150

[117] Kim Y, Schumaker KS, Zhu JK. EMS mutagenesis of *Arabidopsis*. In: Salinas J, Sanchez-Serrano JJ, editors. *Arabidopsis* Protocols. Methods in Molecular Biology™. Vol. 323. Totowa, NJ: Humana Press; 2006. pp. 101-103. DOI: 10.1385/1-59745-003-0:101

[118] Lai KS, Kaothien-Nakayama P, Iwano M, Takayama S. A tilling resource for functional genomics in *Arabidopsis thaliana* accession C24. Genes and Genetic Systems. 2012;**87**(5):291-297. DOI: 10.1266/ggs.87.291

[119] Omar MS, Novak FJ, Brunner H. *In vitro* action of ethylmethanesulphonate on banana shoot tips. Scientia Horticulturae. 1989;**40**(4):283-295. DOI: 10.1016/0304-4238(89)90102-7

[120] Bhagwat B, Duncan EJ. Mutation breeding of banana cv. Highgate (*Musa* spp., AAA Group) for tolerance to *Fusarium oxysporum* f. sp. *cubense* using chemical mutagens. Scientia Horticulturae. 1998;**73**(1):11-22. DOI: 10.1016/S0304-4238(97)00141-6

[121] Bhagwat B, Duncan EJ. Mutation breeding of Highgate (*Musa acuminata*, AAA) for tolerance to *Fusarium oxysporum* f. sp. *cubense* using gamma irradiation. Euphytica. 1998;**101**(2): 143-150. DOI: 10.1023/A:1018391619986

[122] Espino RR, Zamora AB, Pimentel RB. Mutation breeding on selected Philippine fruit crops. In: Nuclear Techniques and In Vitro Culture for Plant Improvement. Vienna: International Atomic Energy Agency; 1986. pp. 429-433

[123] Hautea DM, Molina GC, Balatero CH, Coronado NB, Perez EB, Alvarez MTH, et al. Analysis of induced mutants of Philippine bananas with molecular markers. In: Jain SM, Swennen R, editors. Banana Improvement: Cellular, Molecular Biology, and Induced Mutations. Enfield, NH, USA: Science Publishers Inc.; 2004. p. 382

[124] López J, Strosse H, de la C Ventura J, Sánchez R, Rodriguez S, Swennen R, Panis B, Afza R. Field evaluation of potential mutants obtained after gamma irradiation of banana and plantain (*Musa* spp.) shoot-tip and embryogenic cell cultures. In: Jain SM and Swennen R, editors. Banana Improvement: Cellular, Molecular Biology and Induced Mutations. Science Publishers Inc., Enfield, NH, USA; 2004. pp. 87-96

[125] Mak C, Ho YW, Tan YP, Rusli I, Liew KW. Mutation induction by gamma irradiation in a triploid banana Pisang Berangan. Malaysian Journal of Science Series A Life Sciences. 1995;**16**(2):77-81

[126] Novak FJ, Afza R, Phadvibulya V, Hermelin T, Brunner H, Donini B. Micropropagation and radiation sensitivity in shoot-tip cultures of banana and plantain. In: Nuclear Techniques and *In Vitro* Culture for Plant Improvement. Vienna: International Atomic Energy Agency; 1986. pp. 167-174

[127] Roux NS, Toloza A, Dolezel J, Panis B. Usefulness of embryogenic cell suspension cultures for the induction and selection of mutants in *Musa* spp. In: Mohan Jain S, Swennen R, editors. Banana Improvement: Cellular, Molecular Biology and Induced Mutations. Enfield, NH, USA: Science Publishers Inc.; 2004. pp. 33-43

[128] Smith MK, Hamill SD, Langdon PW, Pegg KG. *In vitro* mutation breeding for the development of bananas with resistance to race 4, *Fusarium* wilt. In: *In Vitro* Mutation Breeding of Banana and Plantain. Proceedings of Reports of an FAO/IAEA Coordinated Research Programme (1988-1993). Vienna, Austria: IAEA; 1995. pp. 37-44

[129] Kulkarni VM, Ganapathi TR, Suprasanna P, Bapat VA. *In vitro* mutagenesis in banana (Musa spp.) using gamma irradiation. In: Mohan JS, Haggman H, editors. Protocols for Micropropagation of Woody Trees and Fruits. The Netherlands: Springer; 2007. pp. 543-559. DOI: 10.1007/978-1-4020-6352-7_48

[130] Roux J, Wingfield MJ. Survey and virulence of fungi occurring on diseased *Acacia mearnsii* in South Africa. Forest Ecology and Management. 1997;**99**(3):327-336. DOI: 10.1016/S0378-1127(97)00110-2

[131] Van Emon JM. The omics revolution in agricultural research. Journal of Agricultural and Food Chemistry. 2015;**64**(1):36-44. DOI: 10.1021/acs.jafc.5b04515

[132] Chassy BM. Can–omics inform a food safety assessment? Regulatory Toxicology and Pharmacology. 2010;**58**(3):S62-S70. DOI: 10.1016/j.yrtph.2010.05.009

[133] Yadav D, Tanveer A, Malviya N, Yadav S. Overview and principles of bioengineering: The drivers of omics technologies. In: Barh D, Azevedo V, editors. Omics Technologies and Bio-Engineering: Volume 1: Towards Improving Quality of Life. Cambridge: Academic Press; 2018. pp. 3-23. DOI: 10.1016/B978-0-12-804659-3.00001-4

[134] Sharma CM, Vogel J. Differential RNA-seq: The approach behind and the biological insight gained. Current Opinion in Microbiology. 2014;**19**:97-105. DOI: 10.1016/j.mib.2014.06.010

[135] Guo L, Han L, Yang L, Zeng H, Fan D, Zhu Y, et al. Genome and transcriptome analysis of the fungal pathogen *Fusarium oxysporum* f. sp. *cubense* causing banana vascular wilt disease. PLoS One. 2014;**9**(4):e95543. DOI: 10.1371/journal.pone.0095543

[136] Vlahou A, Fountoulakis M. Proteomic approaches in the search for disease biomarkers. Journal of Chromatography B. 2005;**814**(1):11-19. DOI: 10.1016/j.jchromb.2004.10.024

[137] Eldakak M, Milad SI, Nawar AI, Rohila JS. Proteomics: A biotechnology tool for crop improvement. Frontiers in Plant Science. 2013;**4**:35. DOI: 10.3389/fpls.2013.00035

[138] Deng GM, Yang QS, He WD, Li CY, Yang J, Zuo CW, et al. Proteomic analysis of conidia germination in *Fusarium oxysporum* f. sp. *cubense* tropical race 4 reveals new targets in ergosterol biosynthesis pathway for controlling *Fusarium* wilt of banana. Applied Microbiology and Biotechnology. 2015;**99**(17):7189-7207. DOI: 10.1007/s00253-015-6768-x

[139] Urbanczyk-Wochniak E, Luedemann A, Kopka J, Selbig J, Roessner-Tunali U, Willmitzer L, et al. Parallel analysis of transcript and metabolic profiles: A new approach in systems biology. European Molecular Biology Organization reports. 2003;**4**(10):989-993. DOI: 10.1038/sj.embor.embor944

[140] Horgan RP, Kenny LC. SAC review: omic technologies: Genomics, transcriptomics. Proteomics and metabolomics. The Obstetrician and Gynaecologist. 2011;**13**:189-195. DOI: 10.1576/toag.13.3.189.27672

[141] Li W, Li C, Sun J, Peng M. Metabolomic, biochemical, and gene expression analyses reveal the underlying responses of resistant and susceptible Banana species during early infection with *Fusarium oxysporum* f. sp. *cubense*. Plant Disease. 2017;**101**(4):534-543. DOI: 10.1094/PDIS-09-16-1245-RE

[142] Simko I, Jimenez-Berni JA, Sirault XR. Phenomic approaches and tools for phytopathologists. Phytopathology. 2016;**107**(1):6-17. DOI: 10.1094/PHYTO-02-16-0082-RVW

[143] Furbank RT, Tester M. Phenomics–technologies to relieve the phenotyping bottleneck. Trends in Plant Science. 2011;**16**(12):635-644. DOI: 10.1016/j.tplants.2011.09.005

[144] Witzel K, Neugart S, Ruppel S, Schreiner M, Wiesner M, Baldermann S. Recent progress in the use of omics technologies in brassicaceous vegetables. Frontiers in Plant Science. 2015;**6**:244. DOI: 10.3389/fpls.2015.00244

[145] Zhang W, Li F, Nie L. Integrating multiple 'omics' analysis for microbial biology: Application and methodologies. Microbiology. 2010;**156**(2):287-301. DOI: 10.1099/mic.0.034793-0