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Fusarium Wilt (Panama Disease) of Banana and its Management

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***Abstract*: This paper presents an overview on fusarium wilt (panama pisease) of banana and its management.**

***Keywords*: Cooking banana, Dessert banana, Fusarium oxysporum species complex, Plantain.**

1. Introduction

Banana and plantain (a type of cooking banana) rank among the world's most valuable primary agricultural commodities. Banana (*Musa* spp.) is the fourth most important food crop in the world and second in India. It is a staple food and export commodity. India's production of Banana is 21% of total world's production. It contributes to the food security of millions of people in the developing world and, when traded in local markets, provides income and employment to rural populations. India is a leading country in the world from the perspective of banana production (Table 1). Two distinct tracts growing banana in Bihar are Vaishali region and North Eastern Koshi region. Important varieties such as Malbhog and Alpan have no match for their fruit quality. These excellent varieties are on verge of extinction due to problem of diseases (panama wilt) and non-availability of disease free quality propagules. Currently, this deadly fungal disease threatens the banana cultivation in all parts of the world. The main infectious agent is the Fusarium soil fungus that enters the banana plant through the roots via the water stream. It travels into the leaves and trunks. In result, it produces gels, gums that usually cut off the flow of nutrients and water, and results in premature wilting of the plants. Since this disease cannot be stopped by application of fungicides or other chemicals, farmers are advised to cultivate their crops with great precautions to prevent the fungus from contaminating their banana plants.

2. Origins, taxonomy, and production of banana

The edible bananas are predominantly hybrids between Musa acuminata and M. balbisiana (Musaceae: Zingiberales) Local and export cultivars are usually parthenocarpic, vegetatively propagated triploids that were selected in antiquity by farmers in Southeast Asia. Banana was one of the first cultivated food crops. Its domestication is thought to have begun after the Pleistocene (approximately 12,000 B.P.), and there is archeological evidence that domesticates were used at least 6,500 B.P. A shorthand system is used to indicate the relative haploid contributions of these species to the cultivars: M. acuminata (A) and M. balbisiana (B); For example, the Gros Michel and Cavendish cultivars are triploid, pure M. acuminate and thus AAA, whereas plantains are triploid, 2/3 M. acuminata, 1/3 M. balbisiana, and AAB. The Linnaean binomials M. paradisiaca (the AAB plantains) and M. sapientum (the dessert banana, 'Silk' AAB) refer to interspecific hybrids.

Botanical description of banana

Scientific Name	Musa SP
Type of Fruit	Berry
Edible part	Endocarp
Chromosome No.	22
Commercial part	Fruit

Although hundreds of banana cultivars exist, few are responsible for most production. The Cavendish subgroup is most significant, as it is responsible for most export production (about 15% of the total) and also accounts for 28% of the locally consumed fruit. The AAB plantain subgroup, which is important in West Africa and tropical America, is responsible for an additional 21%. Thus, 2 of the 50 subgroups of banana account for 64% of all production. The importance of such a limited genetic base signals a perilous situation for global production.

Table 1
Top banana producing countries in the world

Rank	Country	Production (in tonnes)
1	India	27,575,000
2	China (mainland)	12,075,238
3	Philippines	8,645,749
4	Brazil	6,892,622
5	Ecuador	5,995,527

A. Threat to banana cultivation

Fusarium wilt of banana, popularly known as Panama disease, is a lethal fungal disease caused by the soil-borne fungus *Fusarium oxysporum* f. sp. *cubense* (*Foc*). It is the first disease of bananas to have spread globally in the first half of the 20th century. The epidemic started in Central America on the susceptible 'Gros Michel' banana, which at the time dominated the global export trade. In the 1950s, 'Gros Michel' was replaced by Cavendish cultivars. At the end of the 1980s, the so-called TR4 strain, to which Cavendish cultivars are susceptible, was isolated from samples from Taiwan. It has since spread through Asia and reached Africa in 2013.

The fungus enters the plant through the roots and colonizes the xylem vessels thereby blocking the flow of water and nutrients. Disease progression results in the collapse of leaves at the petiole, the splitting of the pseudostem base and eventually plant death. Once established in a field, the fungus persists in soil for an indefinite period of time and cannot be managed using chemical pesticides. The solution best adapted to the continued production of bananas in infested soils is replacing susceptible cultivars by resistant ones.

The pathogenic isolates are classified into races based on the cultivars on which they cause disease. For example, the isolates that affect cultivars in the Gros Michel, Silk and Pome subgroups, among others, are classified as race 1. When Cavendish cultivars exhibiting symptoms of Fusarium wilt were first observed, the isolates were classified as race 4. They were later subdivided into subtropical race 4 (STR4) and tropical race 4(TR4) to distinguish the strains that need predisposing factors to cause the disease from the ones that don't (see Race 4). The race concept has been criticized for being an imperfect measure of pathogenic diversity, but it is still considered useful to describe host reaction and new disease outbreaks.

3. Fusarium wilt (panama disease)

A. History

The early history of this disease is tightly linked with the first banana export trades. Many of the first reports in a given country were on damage in export plantations of ‘Gros Michel’, the banana on which the export trades were then based, and one of the disease’s common names, Panama disease, refers to damage in export plantations in that country, first reported in 1890. Throughout the banana belt, production was eliminated or became increasingly difficult. In 1933, the Governor of Jamaica indicated that the final destruction of Gros Michel was not a question of if, but when, this would occur. He indicated that the island’s banana industry depended upon the development of “an immune variety which also fulfills market requirements.” Fortunately, productive Cavendish cultivars that resisted Fusarium wilt were available, and they ultimately replaced Gros Michel in the American and African trades (Stover 1962). Simmonds (1966) considered Fusarium wilt of banana to be one of the most destructive of all plant diseases. When Stover’s (1962) figures were converted to 2005 dollars, losses in the Gros Michel-based trades totaled at least US$2 billion. Increasing losses and the scarcity of pathogen-free soil played significant roles in the trades conversion to the Cavendish subgroup (Stover 1962). Cavendish is resistant to race 1 of Fusarium oxysporum f. sp. cubense, the cause of the Gros Michel epidemics. As Cavendish replaced Gros Michel, Fusarium wilt disappeared as a problem for the trades. Black leaf streak, caused by Mycosphaerella fijiensis, became the new primary disease, and Fusarium wilt was forgotten as a threat to the industry.

The risk of relying on such a narrow genetic base (the Cavendish cultivars are a closely related set of clones) was recognized by those who were familiar with the above history. Stover (1986) indicated that the export trades were “…extremely vulnerable to a new disease, especially a tropical race of Fusarium wilt that could devastate the basis of the industry—the Cavendish varieties.” Shortly after Stover’s (1986) warning, the tropical race of Fusarium wilt that he feared began to devastate Cavendish in Southeast Asia. In the 1990s, new plantations of Cavendish began to succumb in the region and a new race of F. oxysporum f. sp. cubense, tropical race 4 (TR4), became apparent. In the ensuing decades, TR4 spread rapidly, first within Southeast Asia but more recently to Africa and Western Asia. As of the writing of this article, TR4 had been confirmed in Australia (Northern Territory and Queensland), China (Hainan, Hunan, Guangdong, and Guangxi), Indonesia (Bali, Halmahera, Kalimantan, Java, Papua Province, Sulawesi, and Sumatra), Jordan, Lebanon, Malaysia (Peninsular and Sarawak), Mozambique, Oman, Pakistan, the Philippines (Mindanao), and Taiwan (Fig. 1)

Fig. 1. Geographic distribution of tropical race 4 of Fusarium oxysporum f. sp. cubense.

4. Causal agent: Notes on taxonomy and nomenclature

The causal agent of Fusarium wilt or Panama disease of banana is the fungus Fusarium oxysporum f. sp. cubense (E.F. Sm.) W.C. Snyder & H.N. Hansen (Foc). Its taxonomic position is as follows:

Domain: Eukaryota
Kingdom: Fungi
Phylum: Ascomycota
Class: Ascomycetes
Subclass: Sordariomycetidae
Order: Hypocreales

Fusarium oxysporum is a complex of anamorphic, filamentous, morphologically undifferentiated fungal species featuring saprophytes, antagonists and pathogens to plants, animals and humans. In the case of plant pathogens, these mostly cause wilting, damping off and root and organs necrosis and rots. From an agricultural and economical point of view, it is the most important taxon of Fusarium. Specialization of pathogenicity to plant genera and families gave rise to the formae speciales (f. sp.) classification. Initially, it was believed that formae speciales were specific to one host and henc, the name was taken from the host, e.g. betae, callistephi, apii, mori, and about 60 others. This early concept of highly specific pathogenicity led to the establishment of several formae speciales, which are merely races of formae speciales described in other hosts. Armstrong and Armstrong (1968) demonstrated that F. oxysporum f. sp. batatas from sweet potato could also attack tobacco.

Earlier studies on Fusarium wilt in Central America produced symptoms of Panama disease in Gros Michel banana with inoculation of isolates obtained from Heliconia sp. (R.H. Stover, personal communication, 1990). The formae speciales designated as cubense is applied, based solely on evidence from pathogenicity tests in banana. Bancroft (1876) isolated for the first time the fungal organism from diseased banana wilt plants. Higgins (1904) noted a fungal association in banana plants suffering of a lethal wilt. Smith in 1908 (Smith, 1910) realized the first isolation of the fungus from Cuban diseased banana plants and named the species as Fusarium cubense. Ashby (1913) gave the first detailed description of the causal agent in culture and Brandes (1919) confirmed Koch postulates, not only in Gros Michel (AAA) and Manzano (Apple, AAB), but also in the cultivar Bluggoe (ABB). Wollenweber and Reinking (1935) recognized that Fusarium cubense as a variant of the almost omnipresent Fusarium oxysporum. When Snyder and Hansen (1940) developed the formae speciales system, all species of the complex Fusarium oxysporum that produced wilt symptoms in Musa were renamed as Fusarium oxysporum f. sp. cubense (Foc). Phylogenetic studies reveal that Foc is an asexual polyphyletic fungus with various strains due to convergent evolution.

A. Symptoms

Fusarium wilt or Panama disease of banana produces two types of external symptoms: “yellow leaf syndrome” and “green leaf syndrome”.

Yellow leaf syndrome: this is the most conspicuous and classic symptom of Fusarium wilt on banana. It is characterized by the yellowing border on older leaves that can at times be confused with potassium deficiency, especially in drought and cold environment. The yellowing of the leaves progresses from older to younger leaves. The leaves collapse gradually, bending at the petiole, commonly close to the midrib and hang down, forming a “skirt” of death leaves around the pseudostem. Green leaf syndrome: In contrast to the yellow leaf syndrome, the leaves of affected plants in some cultivars eventually remain predominantly green until the petioles bend and collapse.

The various symptoms of *Fusarium* wilt on banana are described here:

1. Fusarium wilt is a typical vascular wilt disease. The fungus invades the vascular tissue through the roots causing discolouration and wilting, eventually killing the plant. The progress of the internal symptoms can influence the first appearance of the external symptoms. The fruit do not exhibit any symptom.
2. The characteristic internal symptom of Fusarium wilt is vascular discolouration, which varies from pale yellow in the early stages to dark red or almost black in later stages. Internal symptoms first develop in the feeder roots, which are the initial infection sites. The fungus spreads to the rhizome and then the pseudostem.
3. Externally, the first signs of disease are usually wilting and yellowing of the older leaves around the margins. The yellow leaves may remain erect or collapse at the petiole (Fig. 2). Sometimes, the leaves remain green, except for spots on the petiole, but still snap. The collapsed leaves hang down the pseudostem like a skirt. Eventually, all the leaves fall down and dry up.
4. Splitting of the base of the pseudostem is another common symptom. Other symptoms include irregular, pale margins on new leaves and the wrinkling and distortion of the leaf blade (Fig. 2).

Infected suckers do not start showing symptoms of Fusarium wilt until they are about 4 months old, a situation that has contributed to the spread of the disease through planting material. The fruit does not show any specific disease symptoms.


(a)


(b)


(c)


(d)

Fig. 2. (a) Splitting of the pseudostem associated with Panama disease infection, (b) Initial external symptoms of Panama disease include yellowing leaf margins on older leaves, (c) Internal browning of stems and corms is the key diagnostic symptom of Panama disease, (d) Rings of discoloured tissue in the stem are a symptom of Panama disease

B. Similar disease

The leaf symptoms of Fusarium wilt can be confused with those of the bacterial disease Xanthomonas wilt. In plants affected by Fusarium, yellowing and wilting of the leaves typically progresses from the older to the younger leaves. The wilted leaves may also snap at the petiole and hang down the pseudostem. In plants affected by Xanthomonas, the wilting can begin with any leaf and the infected leaves tend to snap along the leaf blade.

In countries with Moko disease, which is caused by *Ralstonia solanacearum* race 2, and also causes vascular discoloration, it is possible to confuse the two diseases (fig. 3). Unlike Moko, Fusarium wil does not cause wilting and blackening of young suckers or a dry rot in the fruit. The first symptoms of Moko on rapidly growing plants are the chlorosis, yellowing and collapse of the three youngest leaves, not the older leaves as with Fusarium wilt. Finally, with Moko the vascular discoloration is concentrated near the centre of the pseudostem and not peripherally, which is common with Fusarium wilt.

Fig. 3. Moko disease (Symptoms looks like fusarium wilt)

5. Mode of transmission

The fungus is commonly spread through infected planting material, infested soil and water. Planting material. Symptomless but infected suckers or rhizomes can transmit the disease when planted in a new area. Infected planting material is often responsible for the local, national and international spread of the disease. Certified tissue-culture plantlets should be free of the fungus and would not contribute to the spread of the disease.

A. Soil

The fungus can persist in soil for decades, even in the absence of bananas. It can survive in infested plant debris and in the roots of alternative hosts. Staff and visitors to a banana plantation have the potential of moving the fungus in or out through infested soil attached to vehicles, tools and shoes. Untreated soil used as a potting medium can transmit the fungus and animals can also move around fungal spores present in soil.

B. Water

Spores can be carried in surface run-off water. They can also contaminate irrigation reservoirs. It is said that in China pumping water from sources contaminated with TR4 spores contributed to the spread of the Fusarium wilt in plantations of Cavendish bananas.

6. Disease cycle

F. oxysporum is the most widely dispersed of the Fusarium species and is found worldwide. F. oxysporum has no known sexual stage, but produces three types of asexual spores: microconidia, macroconidia, and chlamydospores. The microconidia are the most abundantly produced spores. They are oval, elliptical or kidney shaped and produced on aerial mycelia. Macroconidia, which have three to five cells and have gradually pointed or curved edges, are found on sporodochia on the surface of diseased plant (in culture the sporodochia may be sparse or nonexistent). Chlamydospores are usually formed singly or in pairs, but can sometimes be found in clusters or in short chains. They are round thick walled spores produced within or terminally on an older mycelium or in macroconidia. Chlamydospores unlike the other spores can survive in the soil for a long period of time.

F. oxysporum is a common soil pathogen and saprophyte that feeds on dead and decaying organic matter. It survives in the soil debris as a mycelium and all spore types, but is most commonly recovered from the soil as chlamydospores. This pathogen spreads in two basic ways: it spreads short distances by water splash, and by planting equipment, and long distances by infected transplants and seeds. F. oxysporum infects a healthy plant by means of mycelia or by germinating spores penetrating the plant’s root tips, root wounds, or lateral roots. The mycelium advances intracellularly through the root cortexand into the xylem. Once in the xylem, the mycelium remains exclusively in the xylem vessels and produce microconidia (asexual spores). The microconidia are able to enter into the sap stream and are transported upward. Where the flow of the sap stops the microconidia germinate. Eventually the spores and the mycelia clog the vascular vessels, which prevents the plant from up-taking and translocating nutrients. In the end the plant transpires more than it can transport, the stomata close, the leaves wilt, and the plant dies. After the plant dies the fungus invades all tissues, sporulates, and continues to infect neighboring plants.

A. Disease management

The fungus cannot be controlled using fungicides and cannot be eradicated from soil using fumigants. Drainage, environmental conditions and soil type influence host-pathogen interactions. Soils that suppress the disease have been reported in Central America, the Canary Islands, Australia and South Africa. However, the chemical, biological and physical factors responsible for this phenomenon are not well understood.

The solution best adapted to the continued production of bananas in infested soils is replacing susceptible cultivars with resistant ones. However, in the case of TR4, experts stress the importance of preventing the spread of the fungal strain.

F. oxysporum is a major wilt pathogen of many economically important crop plants. It is a soil-borne pathogen, which can live in the soil for long periods of time, so rotational cropping is not a useful control method. It can also spread through infected dead plant material, so cleaning up at the end of the season is important.

One control method is to improve soil conditions because F. oxysporum spreads faster through soils that have high moisture and bad drainage. Other control methods include planting resistant varieties, removing infected plant tissue to prevent overwintering of the disease, using soil and systemic fungicides to eradicate the disease from the soil, flood fallowing, and using clean seeds each year. Applying fungicides depends on the field environment. It is difficult to find a biological control method because research in a greenhouse can have different effects than testing in the field. The best control method found for *F. oxysporum* is planting resistant varieties, although not all have been bred for every forma specialis.

Different races of *F. oxysporum* f. sp. *cubense*, Panama disease on banana, can be susceptible, resistant and partially resistant. It can be controlled by breeding for resistance and through eradication and quarantine of the pathogen by improving soil conditions and using clean plant material. Biological control can work using antagonists. Systemic and soil fungicides can also be use.

B. Cultural method

- Practice proper crop rotation with paddy/sugarcane once or twice followed by banana for 2-3 cycles.
- Plant wilt resistant cultivars such as Poovan and Nendran in endemic areas.
- Proper care should be given when planting susceptible cultivators such Rasthali, Monthan, Karpuravalli, Kadali, Pachanadan by selecting healthy suckers from disease fields.
- Remove and destroy infested plant material after harvest.
- When only 1-3 plants are infected, kill and chop up the diseased plants and stew all the material in water at a temperature of at least 70 deg C for 30 minutes.
- Grow healthy plants with proper fertilization, irrigation, weed control.
- Provide good drainage especially during rainy season.
- Soil application of rice chaffy grain or dried banana leaf formulation or well decomposed compost around the plants.

C. Mechanical method

- Machinery and equipment should be treated with a sanitary solution such as Farmcleanse.
- Footwear, which may have contacted banana plants or soil around banana plants elsewhere, should not be worn on the farm.
- Provide mechanical barriers in and around the infected plants.

D. Biological method

- Application *Pseudomonas fluorescens* @ 2.5kg/ha bactericide can also be applied along with farmyard manure and neem cake.
- About 60 mg of *Pseudomonas fluorescens* (in a capsule) can be applied in a 10 cm deep hole made in the corm.
- Application of bio control agents like *Trichoderma viride* @ 25 g for 4 times once at the time of planting in the planting pit and remaining doses at third, fifth and seventh month after planting.
- Application of *T.harzianum* Th-10, as dried banana leaf formulation @ 10g/platn in basal + top dressing on 2,4,and 6 months after planting.

E. Chemical method

- Application of 2 per cent of Carbendazim as injection of Carbendazim 50 ml capsule application.
- Paring (pralinge removal of roots and outer skin of corm) and dipping of the suckers in clay slurry and sprinkled with Carbofuran granules at 40g/corm.
- Soil drenching of Carbendazim 0.2 per cent solution alternated with Propiconozole 0.1% around the pseudostem at bimonthly intervals starting from five months after planting.
- Application of urea + sugarcane trash (250g/pit) followed by lime (1Kg/pit) and neem cake (1-2Kg/pit).
- Application of neem cake @ 250 Kg/ha was most effective in controlling Fusarium wilt in Rasthali cultivar.

F. By use of tissue cultured plantlet

Tissue cultured plantlets are free from bacteria, fungus and nematode pathogens and should be used to establish new planting whenever possible.

About 70% of the farmers are using banana suckers as planting material while the rest 30% of the farmers are using tissue culture seedlings. Sword suckers with well-developed rhizome, conical or spherical shape having actively growing conical bud and weighing approximately 450-700 g are commonly used as propagating material. Suckers are generally infected with some pathogens and nematodes. Similarly, due to the variation in age and size of sucker, crop is not uniform, harvesting is prolonged and management becomes difficult.

Therefore, *in vitro* clonal propagation i.e. tissue culture plants are recommended for planting. They are healthy, disease free, uniform in growth and early yielding.

7. Conclusion

In India, most of the farmers use suckers as planting material and are not aware of the serious nature of TR4 in banana. As the banana bunches are transported to various States, the pathogen can spread through peduncle of the bunches and there is a chance for the spread of this virulent TR4 strain to other States as well. "Therefore, creation of awareness and providing training to all the stakeholders on the importance and identification of Fusarium wilt disease, preventive steps to be taken to arrest the further spread of the disease to the uninfested areas in Bihar and other states". There are no mechanisms or effective control methods available (except from prevention and quarantine). We need a comprehensive, concerted and long-term action plan with stakeholders: industry, small farmers, governments, NGOs, associations, research institutions, international organizations.

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