Impact of Diseases on Export and Smallholder Production of Banana

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Abstract

Banana (*Musa* spp.) is one of the world's most valuable primary agricultural commodities. Exported fruit are key commodities in several producing countries yet make up less than 15% of the total annual output of 145 million metric tons (MMT). Transnational exporters market fruit of the Cavendish cultivars, which are usually produced in large plantations with fixed infrastructures and high inputs of fertilizers, pesticides, and irrigation. In contrast, smallholders grow diverse cultivars, often for domestic markets, with minimal inputs. Diseases are serious constraints for export as well as smallholder production. Although black leaf streak disease (BLSD), which is present throughout Asian, African, and American production areas, is a primary global concern, other diseases with limited distributions, notably tropical race 4 of Fusarium wilt, rival its impact. Here, we summarize recent developments on the most significant of these problems.

INTRODUCTION

Banana and plantain (*Musa* spp.) rank among the world's most valuable primary agricultural commodities. In 2011, global production was 145 million metric tons (MMT), with a gross production value of US $44.1 billion (42). Although only 15% of this output reaches international markets, export production is a key economic factor in producing nations (42). In 2009, banana was the most important export product produced by Ecuador, Costa Rica, Panama, and Belize, and was ranked second or third in importance in Colombia, Guatemala, the Philippines, Honduras, and Cameroon (42). Locally consumed bananas make up the remaining 85% of global production. They are often grown by smallholder producers and are significant staple foods in Africa, Asia, and tropical America; fruit from hundreds of distinct cultivars are consumed raw, cooked, or brewed (66, 132).

In a previous edition of this series, R.H. Stover discussed the future of the banana export trades (123). He considered issues that impacted or might affect production and evaluated diseases of the Cavendish cultivars because they were the bananas utilized by the trades. Stover (123, p. 90) indicated that the trades were "extremely vulnerable to a new disease, especially a tropical race of Fusarium wilt."

Below, we revisit issues that were considered by Stover (123). Some of these factors are still relevant in export production, such as the transition of the transnational companies from producers to marketing entities, problems that export producers face in managing black leaf streak disease (BLSD; aka black Sigatoka), and increasing production costs (26, 34, 102). Unfortunately, Stover's (123) fear that a tropical race of Fusarium wilt would threaten the trades materialized shortly after his review was published. In the 1990s, tropical race 4 (TR4) emerged in Southeast Asia (97, 98). Its impact on Cavendish production and recent spread to Africa and Western Asia (19, 47) have caused concern among banana producers and consumers worldwide.

BANANA ORIGINS, TAXONOMY, PRODUCTION, AND TRADE

A shorthand system is used to indicate the relative haploid contributions of two species, *Musa acuminata* (A) and *Musa balbisiana* (B), to diploid and polyploid cultivars of banana (116–118). Although hundreds of parthenocarpic and vegetatively propagated cultivars are produced worldwide, a narrow genetic base is responsible for most production (94). The Cavendish subgroup AAA is most significant, as it accounts for 28% of the locally consumed fruit, as well as almost all export production (15%). The AAB plantain subgroup, which is important in West Africa and tropical America, is responsible for an additional 21% of the total. Thus, two of the 50 recognized subgroups of banana account for more than 60% of all production.

In descending order, the top 10 banana-producing countries are India, Uganda, China, the Philippines, Ecuador, Brazil, Indonesia, Colombia, Cameroon, and Tanzania (42). With the exceptions of the Philippines, Ecuador, and Colombia, virtually none of the fruit from these countries reach international markets (42).

In 2011, international trade was valued at US $8.9 billion (42). Production was highly concentrated, as the top five exporting countries accounted for 68% of the total and four of the top five were in the Americas (42). Five companies were responsible for 75% of international trade: Dole (26%), Chiquita (22%), Del Monte (15%), Fyffes (7%), and Noboa/Bonita (5%) (11). To a major extent, the transnational companies are now vertically integrated marketers that buy, transport, ripen, and distribute fruit (102). By becoming marketers, the companies have minimized risk by avoiding production problems, natural disasters (especially hurricanes), and environmental and social standards that confront producers. Meanwhile, they have solidified

their control of the supply chain and achieved higher profit margins by focusing on transport and distribution activities and increasing partnerships with retailers, wholesalers, and distributors.

CONSTRAINTS TO PRODUCTION

Banana is a tropical crop that is produced in a wide range of subtropical and tropical environments (125). Monocultures prevail in export production but are less common in smallholder situations, where different banana cultivars and crops are often interplanted. Although the management of production can be complicated in interplanted situations, disease pressure is often considerably lower than in monocultures (121, 122).

Diverse abiotic and biotic factors influence banana production (63, 93, 112, 125). Stover (123) listed five primary factors that affected yield in export production: (*a*) soils and plant nutrition; (*b*) drainage and irrigation; (*c*) type of cultivar; (*d*) planting densities and rhizome (sucker) management; and (*e*) BLSD management. Yields decline whenever one or more of these factors are deficient.

Although the same factors impact production in smallholder situations, yield and quality standards are generally lower than for export (102, 125). In smallholder sectors, low-yielding cultivars that produce organoleptically superior fruit are often produced. Moreover, high-yielding, disease-resistant products from the breeding programs are accepted in some of these situations even when they have less than excellent postharvest traits or taste (102).

Abiotic Factors

Different abiotic factors impact export and smallholder production (125). Whereas supplemental irrigation and fertilization, drainage systems, bunch support, debudding and dehanding, fruit protection (bagging), ground covers, and herbicides are used in export production, few if any inputs are made by smallholders (21, 125, 131, 134). Predictably, relatively low yields occur in the latter sectors.

High input costs are a major concern for export producers. In the future, peak oil will affect high-input agricultural commodities to a greater extent as fertilizers become more expensive and energy costs increase throughout export and marketing chains (33, 59). Climate change will influence both export and smallholder production as well as where banana can and cannot be produced (55, 59, 78). Labor and environmental issues, international trade regulations and tariffs, and production imbalances will continue as important issues for the export trades (82, 126, 136).

Biotic Factors

Diverse biotic factors affect export and smallholder production of banana (63, 93). The most important biotic constraints are diseases (in descending order, those caused by fungi, bacteria, viruses, and nematodes). Diseases cause more losses than all other biotic constraints (102) and have played prominent roles in the establishment and mandates of banana improvement programs worldwide (16, 63).

Natural, long-distance spread of plant pathogens [more than 500 km, as defined by Brown & Hovmøller (15)] is limited to those that produce propagules that can survive conditions in the upper atmosphere, usually the rusts and smuts. Thus, with the possible exceptions of *Uromyces musae* and *Uredo musae*, both of which cause rust diseases of banana (63), long-distance spread of banana pathogens is associated with the anthropogenic movement of infested seed pieces (aka suckers) and other plant tissue (13, 100). The wide dissemination of *Fusarium oxysporum* f. sp. *cubense* (FOC)

in seed pieces is well-known and was a key factor that led to the collapse of export trades based on Gros Michel AAA (121). Likewise, the prevalence of *Mycosphaerella fijiensis* and *Mycosphaerella musicola* throughout the world's banana zones resulted from the movement of infected banana leaves used as packing materials and banana germplasm that was used in the breeding programs (56). Parnell et al. (91) reported that viable ascospores of *M. fijiensis* would probably move no more than 200 km, and Rieux et al. (110) demonstrated that they usually spread far shorter distances.

Colletotrichum musae (anthracnose), *Banana streak virus* (BSV; banana streak), and the burrowing nematode (*Radopholus similis*) have also been widely disseminated with this crop (63) (**Table 1**). Given the ease with which they are moved, it is surprising that other significant problems have relatively narrow geographic distributions. For example, *Mycosphaerella eumusae* (eumusae leaf spot), *Guignardia musae* (freckle), and *Pratylenchus goodeyi* (a lesion nematode) currently have relatively restricted distributions even though they spread in some of the same manners as the widely disseminated problems. Although attributes that distinguish the narrowly and widely distributed pathogens have apparently not been studied, latent infection of, and the severity of symptoms on, traditional seedpieces (suckers) probably play important roles.

Of relevance to this review, TR4 of FOC (cause of Fusarium wilt), *Ralstonia haywardii* subspecies *celebensis* (blood disease), *Xanthomonas campestris* pv. *musacearum* [banana Xanthomonas wilt (BXW)] and *Banana bunchy top virus* (BBTV; banana bunchy top disease) had narrow distributions which have recently expanded (102). The movement of suckers (banana bunchy top disease) and infected fruit and contaminated tools and insect vectors (blood disease and BXW) were probably responsible for the spread of the latter pathogens, but factors that enabled the transcontinental spread of TR4 are unclear.

Few options exist for managing the most significant diseases of banana (63). For those with narrow distributions, effective quarantine measures and the use of pathogen-free planting materials are key interventions (13, 27). Pesticides are available to combat some of these problems, but the numbers and efficacy of products that are available are decreasing (34). Importantly, commercially important genotypes are often quite susceptible (90). For many bananas, there is a critical need for enhanced disease resistance.

Diseases and pests of banana have been reviewed extensively (63, 93, 102, 103, 121, 122, 135). Hundreds of biotic constraints are known and new threats continue to develop (32, 127). However, based on their current or potential impact, ability to spread, and difficulty with which they are managed, fewer than 20 are major concerns (**Table 1**) (100, 115). In descending order of importance, we list below the most significant emerging and pre-existing diseases of this crop. We discuss recent insights on their etiology, epidemiology, and management, and project how these and other factors could affect export and smallholder production in the future.

Emerging Diseases

TR4, BXW, and blood disease are emerging, lethal diseases of banana. Although TR4 was recognized within the past couple decades, BXW and blood disease have been known for 75 and 100 years, respectively. The geographic distributions of each of these diseases have expanded significantly in the past decade.

Tropical race 4 of Fusarium wilt. Fusarium wilt is found in most banana-producing regions (63, 95, 97). Four pathotypes of FOC are recognized on banana: race 1, which caused the epidemics on Gros Michel; race 2, which affects ABB cooking bananas, such as Bluggoe; subtropical race 4 (SR4), which affects Cavendish and race 1 and 2 suscepts in the subtropics; and TR4, which affects many of the same cultivars as SR4 but in the absence of disease-predisposing cold temperatures

Table 1 The major diseases and pests of banana, *Musa* spp.

Constraint	Cause(s)	Distribution	Impact	Management	References
Diseases					
	Bacteria				
Blood disease	*Ralstonia haywardii* subspecies *celebensis*	Indonesia, Malaysia (?)	All cultivars are susceptible, but those with ABB genomes are at greatest risk	Male bud removal, sanitation, budless mutants	18, 58, 64
Moko disease	Phylotypes IIA-6, IIB-3, and IIB-4 of *Ralstonia solanacearum*	Primarily tropical America	All cultivars are susceptible, but those with ABB genomes are at greatest risk	Male bud removal, sanitation, budless mutants	17, 63, 122
Banana Xanthomonas wilt (BXW)	*Xanthomonas campestris* pv. *musacearum*	Sub-Saharan Africa	All cultivars are susceptible, but those with ABB genomes are at greatest risk	Male bud removal, sanitation, budless mutants	119, 129
	Fungi				
Anthracnose	*Colletotrichum musae*	Humid tropics and subtropics	Serious postharvest disease for export; pathogen also causes crown rot	Bunch covers and pre- and postharvest fungicide applications	63, 122
Black leaf streak disease (BLSD)	*Mycosphaerella fijiensis*	Humid tropics and subtropics	Significant reductions in yield and postharvest quality of fruit in export and smallholder production	Applications of fungicides and spray oils; some cultivars (especially ABBs) are resistant	9, 26, 34
Crown rot	*C. musae* and *Fusarium semitectum*	Humid tropics and subtropics	Postharvest problem on exported Cavendish fruit	Postharvest fungicides	63, 121
Eumusae leaf spot	*Mycosphaerella eumusae*	Southern Asia, Nigeria	Serious problem in some areas; can be confused with black leaf streak disease, which causes similar symptoms	Applications of fungicides and spray oils	63
Freckle	*Guignardia musae* (anamorphs: *Phyllosticta musarum*, *Phyllosticta cavendishii*, and *Phyllosticta maculata*)	Eastern Hemisphere	Different strains of the pathogen cause serious damage to leaves and fruit of different cultivars/genomes	Applications of fungicides and spray oils	63, 137
Fusarium wilt	*Fusarium oxysporum* f. sp. *cubense*	Global	Pathogenically diverse pathogen affects diverse host cultivars	Host resistance, pathogen exclusion	97, 98, 121

(*Continued*)

Table 1 (*Continued*)

Constraint	Cause(s)	Distribution	Impact	Management	References
Diseases					
Sigatoka leaf spot	*Mycosphaerella musicola*	Humid tropics and subtropics	Causes significant damage; displaced by *M. fijiensis* in most locations	Applications of fungicides and spray oils	63, 121
	Viruses				
Banana bunchy top disease	*Banana bunchy top virus*	Eastern Hemisphere	Most damaging of the virus-induced diseases	Cultural management, pathogen-free planting material	13, 63
Banana streak disease	*Banana streak virus*	Global	Serious problem in some sectors (e.g., sub-Saharan Africa)	Pathogen-free planting material	51, 63
Bract mosaic	*Banana bract mosaic virus*	Southern Asia	Quarantine significance, variable or unclear impact where found	Pathogen-free planting material	63
Infectious chlorosis	*Cucumber mosaic virus*	Global	Primarily affects young plantations	Pathogen-free planting material, elimination of alternative weed hosts of pathogen	63
	Pests				
Burrowing nematode/ blackhead toppling syndrome	*Radopholus similis*	Global	Causes serious losses in some areas, especially when bunches approach harvest	Clean planting material	63
Lesion nematodes	*Pratylenchus coffeae* and *Pratylenchus goodeyi*	Global, but primarily in Eastern Hemisphere	Can cause serious losses on AAB plantains; damage can be confused with that caused by *R. similis*	Clean planting material	63
Banana weevil	*Cosmopolites sordidus*	Global	Causes serious losses in some areas	Clean planting material, trapping, insecticides	52, 93
Stem borer	*Odoiporus longicollis*	Southeast Asia	Causes serious losses in some areas		93

that occur in the subtropics (race 3, which does not affect banana, is not considered here). Although these races facilitate comparisons among different populations of FOC, they imprecisely classify pathogenic variation in this pathogen (97). There is a critical need for a better understanding of this important trait in FOC.

Stover (123, p. 88) indicated that Cavendish succumbed to Fusarium wilt in subtropical Australia, the Canary Islands, South Africa, and Taiwan, and that the responsible strain of FOC was "not highly virulent." He also indicated that this strain was introduced from Taiwan to tropical Mindanao, where it had caused little damage by 1986. Afterward, it became clear that SR4 was responsible for the Cavendish outbreaks in subtropical Australia, the Canary Islands, and South Africa, and that TR4 was not introduced to Mindanao until recently (it was confirmed

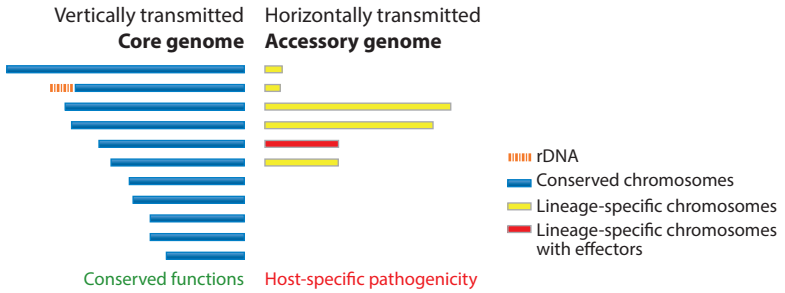

Figure 1

Structural and functional compartmentalization of the *Fusarium oxysporum* f. sp. *lycopersici* genome. The genome is divided into the core genome, which is presented in the form of conserved chromosomes, is vertically transmitted, performs conserved functions, and is almost identical among all *F. oxysporum* strains, and the accessory genome, which is in the form of lineage-specific chromosomes, and is horizontally transmitted, responsible for host-specific pathogenicity, and unique to each forma specialis. Abbreviation: rDNA, ribosomal DNA.

there in 2006) (97). Furthermore, TR4 was probably in Taiwan and other areas in Southeast Asia when Stover (123) wrote his review.

FOC is a member of the *F. oxysporum* species complex (FOSC), which contains nonpathogens as well as plant and animal pathogens (36, 81). Plant-pathogenic isolates in the FOSC often exhibit considerable host specificity, and single pathogenic forms affect a single or limited set of host plants. A comparative genomic study revealed structural and functional compartmentalization of the genome of *F. oxysporum* f. sp. *lycopersici* (FOL), a tomato pathogen (77) (**Figure 1**). Structurally, the *Fusarium* genome is divided into core and accessory regions (75, 76, 109). The core region, which resides on conserved chromosomes, is preserved among all *Fusarium* species and is present in all strains in the FOSC that have been studied. Whereas core regions are vertically transmitted and perform all essential functions, accessory regions are located on supernumerary (SP) chromosomes that are transferred horizontally. SP chromosomes encode host-specific factors but generally lack housekeeping genes that are involved in primary metabolism; they are responsible for host-specific pathogenicity in the FOSC. Owing to its horizontal transfer, host specificity in the FOSC, including FOC, is often polyphyletic (10, 89).

Although phylogenetic relationships in the FOSC have been examined with genes from the core genomic region (88), additional markers are needed to easily and reliably place all strains into a phylogenetic framework. A recent comparison of sequences of 12 phylogenetically diverse isolates of *F. oxysporum* from diverse hosts identified 10 single-copy, informative loci (L.-J. Ma, personal communication) based on the recommendation of the Fungal Tree of Life (62, 74). Phylogenetic markers for strains in the FOSC are under development.

Conversely, genes related to pathogenicity would be expected in accessory genomic regions. We have sequenced TR4 strain II5 (aka CBS 102025 and NRRL 36114) and identified a total of 838 genes that can be coarsely defined as accessory (L.-J. Ma, unpublished data). Potentially, these genes could be used to investigate the pathogenicity of this deadly race. On the basis of comparisons with FOL strain 4287, small secreted cysteine-rich proteins (potential effectors) and candidate secondary metabolite biosynthesis gene clusters were identified in II5. Some of the *SIX* (secreted in xylem) genes that were identified in FOL (108, 109) have also been detected in FOC. *SIX1* has been observed consistently in strains of FOC, and three homologs of *SIX1*, *SIX1a*, *b*, and *c*, are found in TR4 (L.-J. Ma, unpublished data; M. Rep, personal communication). Preliminary results indicate that *SIX1a* has a virulence function on Cavendish (M. Rep, personal

communication). Interestingly, *SIX8* is present multiple times in the subtelomeric regions of the FOL genome and is also found as multiple copies in strains of TR4 and SR4 (L.-J. Ma, unpublished data). PCR (polymerase chain reaction) amplification revealed two *SIX8* homologs, *SIX8a* and *SIX8b*, in more than 500 strains of FOC (46).

Many questions remain on TR4 and other races in FOC. For example, what are the genetic mechanisms that contribute to the virulence of TR4? Has TR4 accumulated mutations from other races of FOC to overcome the resistance of Cavendish, or are novel genetic materials responsible for this outcome? Furthermore, nothing is known about the genetic basis of resistance in the host, which needs to be elucidated for a good understanding of the banana:FOC pathosystem. Nevertheless, current data indicate that the tropical outbreaks of Fusarium wilt on Cavendish are caused by a single clonal lineage of the pathogen VCG (vegetative compatibility group) 01213–01216 (37, 97). As host specificity in the FOSC is often polyphyletic, due to horizontal transfer of the SP chromosomes, pathogenicity toward Cavendish may also be the result of convergent evolution. If so, genes from core genomic regions of these fungi would not be expected to identify novel pathotypes. To understand evolutionary processes behind the development of plant-pathogenic races in the FOSC and to define TR4-specific virulence factors, genomic and phenotypic comparisons of diverse strains of FOC are needed.

In general, there are no effective, long-term biological, chemical, or cultural treatments to protect susceptible cultivars from Fusarium wilt (96, 101). Many of the world's important banana cultivars are susceptible to TR4, and other important cultivars have unclear responses (102). Better information is needed on the susceptibility of cultivars and the various subgroups, as well as on genotypes that could be used in improvement programs (**http://www.panamadisease.org**).

Resistance to TR4 is found in several bred hybrids, especially those developed by the Fundación Hondureña de Investigación Agrícola (FHIA) in Honduras (45). FHIA hybrids have been widely deployed and are especially important in Cuba, where they are grown without significant input of fertilizers or fungicides (5, 6). Unfortunately, hybrids from FHIA and other improvement programs cannot be used as export replacements. For example, the high-yielding dessert clones FHIA-01 and FHIA-02 have lower pulp-to-peel ratios, are not as sweet, and have lower overall consumer acceptance than the Cavendish cultivars Grand Nain and Williams (29). In this regard, somaclonal mutants of Giant Cavendish from Taiwan (the so-called GCTCV lines) produce better fruit (60) but must be replanted every one or two cycles in TR4-infested sites. Despite their slight susceptibility to TR4, poor hand and finger architecture, and lower yields, they are the best alternatives for export production where TR4 is present. Conversion to and production of the GCTCV lines by the trades would be costly and would radically change production norms.

The release of the genome sequence of *M. acuminata* ssp. *malaccensis*, a wild diploid banana, is a turning point for banana improvement (35), particularly because wild diploids have excellent resistances to various diseases (N. Garcia & G.H.J. Kema, unpublished data) and can be used as parents in breeding programs (63, 90). Markers that are linked to the various resistances could facilitate their introgression into new lines, thereby assisting traditional breeding. However, such programs require ample time. Given the rapid spread of TR4 and the urgent need for solutions, all possible tools and methods should be considered.

Genetic transformation of banana has become relatively commonplace (90, 107), and there are convincing arguments for using genetic transformation to create resistant genotypes, especially when targets, such as Cavendish-like export bananas, are difficult to improve via conventional breeding (1). When and whether engineered bananas will be accepted in the marketplace is not clear (71). However, even if they were accepted it would take many years to evaluate and deregulate these products. More work and patience will be needed to realize the promise engineered bananas hold for combating this disease.

Banana Xanthomonas wilt. Lethal bacterial wilts of banana occur in Africa (BXW), Southeast Asia (blood disease), and the Western Hemisphere (Moko disease). BXW emerged as a significant threat to banana when it arrived in Uganda in 2001 (119, 129), whereas blood disease and Moko disease (caused by phylotypes IIA-6, IIB-3, and IIB-4 of *Ralstonia solanacearum*) have each been recognized on banana for over a century (63).

The epidemiologies of these diseases are remarkably similar (17, 18, 63, 79, 102, 129). The pathogens are disseminated in soil and water and on farm tools, and all appear to be soil inhabitants that enter host roots through natural openings or wounds. In addition, insects that visit banana inflorescences, particularly those of cultivars with dehiscent bracts and an ABB genome, can spread the pathogens rapidly and over great distances. Large geographic jumps have been reported for Moko disease (90 km) (17) and blood disease (between 100 and 200 km/yr) (79).

BXW has been present in Ethiopia on a banana relative, enset (*Ensete ventricosum*), for at least 75 years (22). Although banana is not an important crop in Ethiopia, Yirgou & Bradbury (138, p. 114) were concerned that the disease might "escape and establish itself on banana in other parts of the world." BXW was reported in Uganda in 2001 and has since spread to Burundi, the Democratic Republic of Congo, Kenya, Rwanda, and Tanzania (129). The disease has caused losses of more than $2 billion over the past decade.

X. campestris pv. *musacearum* is related to *Xanthomonas vasicola*, a pathogen of sorghum (8). Sequences of the internally transcribed spacer (ITS) locus and gyrase B (*gyr*B) gene revealed limited (<2%) nucleotide divergence among a geographically diverse set of isolates from banana and enset (129).

The modes by which the pathogen is transmitted depend on the production situation (40, 128, 138). Insect vectors are less important in cold, high-altitude regions, and transmission via farm tools is more important in highly managed plantations. For example, where Pisang Awak ABB is grown in low-input systems, transmission is primarily via insects; whereas spread via infested tools is most common where the East African Highland Bananas (EAHBs) AAA are produced in intensive commercial systems.

BXW can cause total losses, and all cultivars develop symptoms after artificial wound inoculation (129). However, mutants of ABB clones that do not produce male flower buds are not infected via insects and have been promoted where ABB bananas are produced with minimal management. Recently, Tripathi et al. (130) constitutively expressed the *Hrap* and *Pflp* genes in Sukali Ndizi AAB and Nakinyika AAA (which is an EAHB), and tested 65 lines (40 *Hrap* lines and 25 *Pflp* lines) in a field trial in Uganda. After artificial inoculation with *X. campestris* pv. *musacearum* and evaluation over two crop cycles, many of the transformed genotypes exhibited outstanding resistance to BXW and produced significantly greater yields than the nontransformed controls (L. Tripathi, personal communication).

Blood disease. Blood disease (aka Penyakit Darah) was first reported in 1906 in the Salayar Islands south of Sulawesi (63, 111). It destroyed a developing dessert banana trade there and was found later on Sulawesi by Gäumann (48–50). The Dutch imposed a quarantine to limit its spread, as it was unknown at the time in other areas. The eventual spread of blood disease to Java around 1987 preceded its dissemination to other areas in Indonesia (Bali, Kalimantan, Moluccan Islands, Papua, Sumatra, and West Nusa Tenggara) (18, 31, 41, 58, 63). The transmigration of people from Java to less populated islands in the country was associated with these outbreaks. Anthropogenic spread was thought to be responsible for a 2007 outbreak in peninsular Malaysia, although that appears to have been caused by Moko disease rather than blood disease (57, 139). Losses will escalate as blood disease spreads. If it reaches the Southeast Asian mainland there would be no barriers to its eventual movement to the Indian subcontinent (64).

For many years, blood disease was presumed to be an aberrant outbreak of Moko disease (124, 135). However, clear genetic differences between the blood disease and Moko pathogens were revealed in recent studies (23, 92, 106). Although Buddenhagen (18, p. 61) speculated that "...the genes for pathogenicity and field biology...of the Moko bacterial wilt pathogen and the blood bacterial wilt pathogen would seem to be identical," the genomic data of Remenant et al. (106) indicated that convergent evolution, not lateral gene transfer, was probably responsible for their similar disease cycles.

The original name of the blood disease pathogen, *Pseudomonas celebensis* (50), has undergone several changes (63), most recently to *R. haywardii* ssp. *celebensis* (106). It has been suggested that the blood disease pathogen and banana coevolved (63). However, Buddenhagen (18) indicated that this was unlikely because of when and where the disease first appeared. Blood disease was originally found where wild bananas were not found (111), supporting Buddenhagen's (18) suggestion that the bacterium originated on something other than banana. Interestingly, the putative Moluccan origin of *R. haywardii* ssp. *celebensis* coincides with the center of origin for clove, which is affected by a close relative of the pathogen, *Ralstonia syzygii* (12, 106). Although more work is needed to understand the evolution of these and other members of phylotype IV of the *R. solanacearum* species complex, the circumstantial evidence indicates that the *R. haywardii* ssp. *celebensis* × banana interaction may have a short history.

Gäumann (48–50) recognized that blood disease differed from Fusarium wilt and demonstrated that transmission via the inflorescence was possible. He suggested that flying insects that visited these organs were vectors of the pathogen 40 years before this was demonstrated for the SFR (slimy fluidal round) strains of the Moko pathogen (63). Recently, Mairawita et al. (79) reported that a flying insect, *Trigona minangkabau*, was commonly infested with the blood disease pathogen in Sumatra. Gäumann (49) reported that the pathogen survived in soil for at least a year in infested plant residues and infected the banana host through its roots. Infested soil, tools, and vehicles move the pathogen within plantations, and infected fruit and planting material are capable of long-distance spread.

All edible bananas may be susceptible to blood disease, as Gäumann (49) found no resistance in 100 cultivars that he tested. However, infection can be impeded by using mutants that do not produce male buds, such as Pisang Sepatu Amora (18, 38, 58).

Ongoing Threats

Several banana diseases with relatively long histories impact both export and smallholder production (**Table 1**). We discuss recent insights on the causal agents, epidemiology, and management of four significant problems.

Black leaf streak disease. At least 20 species of *Mycosphaerella* have been described on *Musa* (9, 26). *M. fijiensis* is most important and has displaced *M. musicola*, cause of Sigatoka leaf spot, throughout the humid tropics (26, 34).

BLSD reduces yields, causes premature ripening, and shortens the postharvest green life of fruit (26, 34, 63). It has decreased subsistence production of diverse banana and plantain cultivars, and makes the production of Cavendish for export increasingly difficult. To maximize yields in export plantations and ensure that premature ripening does not occur, frequent aerial applications of fungicides are required (34). Several different types of protectant and systemic fungicides are used with or without spray oils. This is an enormously expensive practice. In tropical America, 25% of the purchase prices of fruit was related to the impact of BLSD and the infrastructures (hangers for aircraft, runways, fungicide mixing, storage areas, etc.), materials (mainly fungicides and spray oils),

and application equipment that were required for management (125). Nevertheless, a reliance on susceptible Cavendish cultivars and production in what are often disease-conducive environments currently leaves the export producers with no other options. A new paradigm for exporters is needed wherein disease-resistant replacements for the Cavendish standards would be produced (34). To that end, there is an urgent need for productive BLSD-resistant genotypes that would be accepted by consumers. Scant progress has been made toward the development of such bananas.

M. fijiensis is considered a high-risk pathogen (14, 80). Its heterothallic bipolar mating system (28) enables it to continuously recombine and adapt to various abiotic and biotic stresses, and its asexual stage allows beneficial genetic combinations to be fixed in populations. Resistance or tolerance has developed to many of the fungicides that are used against BLSD. Owing to their single gene mode of action and the ease with which resistance to them develops (single nucleotide mutations are sufficient), the once efficacious benzimidazoles and strobilurins are now useless (7; R. Arango, unpublished results). Fungicides with more complex modes of action, such as the demethylation inhibitors (DMIs), are slowly losing efficacy as tolerance to them develops (20, 25; Chong & Kema, unpublished results). Where DMI tolerance has become serious, application frequencies have reached 70 times per year and production has become unsustainable (34).

Less is known about the adaptation of *M. fijiensis* to other factors. For example, whether resistant cultivars such as Yangambi Km 5 developed more severe BLSD because of the development of new pathotypes or the selection of pre-existing pathotypes is not known (26, 83). Likewise, in the past only Sigatoka leaf spot affected plantains above 500 MASL (meters above sea level) (84, 125). Although it is unclear whether the recent impact of BLSD at higher elevations is due to environmental adaptations in the pathogen or global warming (i.e., higher temperatures at higher elevations), the possibility that *M. fijiensis* may adapt to multiple factors should be kept in mind when one considers the future impact of this disease.

Host resistance for BLSD is reminiscent of that which exists for TR4 in that many important cultivars are affected (61, 63, 102). Although some bred hybrids, ABB cooking and assorted AA, AB, and AAB dessert clones resist BLSD and are accepted by smallholders (39, 87); none of the tolerant dessert cultivars meet export standards (29, 61). Notably, differences in the responses of host genotypes to BLSD are largely anecdotal, and basic information on host and cultivar specificity is lacking. For example, little is known about how individual isolates of *M. fijiensis* vary genetically for pathogenicity on different banana genotypes. This is vital for understanding the banana–*M. fijiensis* pathosystem. Similar studies in the related Dothideomycete *Zymoseptoria tritici* (formerly *M. graminicola*) have been instrumental in the discovery of a range of specific *Stb* resistance genes against the disease it causes on wheat, Septoria leaf blotch (53, 68, 69, 104). Investigations of the population biology and effectors of *M. fijiensis* have revealed the importance of its mating system for specificity in this pathosystem and the need for a deeper understanding of diversity in order to develop successful breeding strategies (110, 120). Recently, Goodwin & Kema (54) compared the genomes of *M. fijiensis* and *Z. tritici* and the respective BLSD and Septoria leaf blotch pathosystems.

Relatively few publications have dealt with bananas that have been genetically transformed for resistance to BLSD. Vishnevetsky et al. (133, p. 61) indicated that "several transgenic banana lines with improved tolerance" were found in a four-year field study, even though statistical differences were not evident between transformed and nontransformed Grand Nain. On the basis of leaf disc assays, Kovács et al. (72, p. 118) reported a "considerable delay in disease development" and a decrease in leaf necrosis that developed on transformed lines versus the susceptible Gros Michel check. Unfortunately, leaf disc assays are not reliable predictors of BLSD response (26). The need for field results for resistant materials was confirmed by the disappointing performance of the lines of Kovács et al. (72) in Uganda (64).

Banana bunchy top disease. Banana bunchy top disease, caused by BBTV, is the most destructive virus-induced disease of banana. It is currently recognized in approximately 40 banana-growing countries and regions, and, with the exception of the Hawaiian Islands, all reports come from the Eastern Hemisphere (13, 103). Serious outbreaks occurred recently in Pakistan (70, 105) and Hawaii (44), and the disease has spread rapidly in sub-Saharan Africa in the past decade or so (13, 73, 86). That it has remained outside production areas in tropical America is a stroke of considerable luck.

BBTV is a double-stranded DNA virus that infects edible banana cultivars, as well as *Ensete ventricosum*, *M. acuminata* ssp. *banksii*, *M. acuminata* ssp. *zebrina*, *M. balbisiana*, *Musa coccinea*, *Musa jackeyi*, *Musa ornata*, *Musa textilis*, and *Musa velutina* (103 and references therein). Two populations of BBTV are recognized, the Asian and South Pacific groups, based on nucleotide sequence differences (67). The Asian group is found in China, Japan, Indonesia, the Philippines, Taiwan, and Vietnam, whereas the South Pacific group is more widely spread and is found in Angola, Australia, Burundi, Cameroon, the Democratic Republic of Congo, Egypt, Fiji, Gabon, Hawaii, India, Malawi, Myanmar, Pakistan, Rwanda, Taiwan, Tonga, and Western Samoa (13 and references therein). The latter group is responsible for the recent outbreaks that are mentioned above.

The black banana aphid, *Pentalonia nigronervosa*, is found worldwide on banana and transmits BBTV in a circulative, nonpropagative manner (63, 103). It is responsible for secondary dissemination over short distances, whereas long-distance spread occurs primarily via infected, vegetative planting material (13, 86).

In Australia, the average distance of secondary spread from primary foci of banana bunchy top disease was about 16 m, and approximately two-thirds of new infections were within 20 m, with 99% within 86 m (2–4). The probability that new plantations would be affected was related to their proximity to affected plantings (4). Within 12 months, 88% of the adjacent plantations were affected, but only 27% and 5% of those that were separated by, respectively, 50–1,000 m and more than 1,000 m were affected.

Immunity to banana bunchy top disease does not exist in the edible clones, but differences in susceptibility are recognized (103 and references therein). For example, the Cavendish subgroup is highly susceptible, but symptom development is slower and less severe on Gros Michel (63, 103).

Moko disease. Moko disease was first recognized in Trinidad in the 1890s (114). It affects diverse dessert bananas, plantains, and cooking bananas, although Bluggoe ABB is especially susceptible (63). The disease is named after a synonym of Bluggoe, Moko, and it is currently difficult to produce this banana wherever the disease is found. When Moko disease is found in export plantations of Cavendish, rigorous management is needed to maintain production.

In the Western Hemisphere, Moko disease is recognized on banana from the Amazon Basin to Guatemala and southern Mexico; recently, it has spread in the Caribbean Basin, where it is now found on Carriacou, Grenada, Jamaica, and Trinidad (63, 99). In the Eastern Hemisphere, it has been confirmed in the Philippines, where the introduction of infected planting material from Honduras is thought to be responsible (17). A similar outbreak on ornamental heliconia in Australia was eradicated (63). Reports from Malaysia (139) that were thought to refer to blood disease (57, 64) appear to be of Moko disease, but reports in Cambodia, India, and Africa have not been confirmed (30, 63). Moko disease continues to spread to new areas in the Western Hemisphere and has become a serious problem in areas with a prior history of the disease. For example, in Colombia, lax control measures in smallholder situations have jeopardized neighboring export plantations (85, 113).

R. solanacearum is widespread, diverse, and typically soilborne. Some strains of the pathogen are adapted to nonspecific transmission by flying insects (23, 63, 106). *R. solanacearum* had been divided into five biovars on the basis of carbohydrate utilization and five races that were determined by host range. Moko strains were in biovar 1 and race 2. Currently, four phylogenetically distinct lineages, phylotypes I–IV, are recognized. They are based on sequences of the intergenic spacer region of the 16S-23S rRNA gene and the endoglucanase gene, and have been used to classify members of the *R. solanacearum* species complex (43). Phylotype II, which contains strains that cause Moko disease, is subdivided into phylotypes IIA-6, IIB-3, and IIB-4, based on comparative genomic hybridizations (23).

Although root-to-root infection is possible and moving water can disseminate the pathogen, spread usually involves insects or man (17, 63). Trigona bees, wasps, and other flying insects have been reported to disseminate some strains of the pathogen (especially the SFR and, to a lesser extent, B strains) (17, 63, 122). Insect-driven epidemics develop rapidly due to the strength and range of the vectors and the speed with which plants become infectious. Contaminated farm machinery, machetes used for pruning, and infected fruit and rhizomes are also effective vehicles of dissemination (17, 63, 122).

Due to the explosive means by which Moko epidemics can develop, regular inspection and eradication programs must be established where the disease is found (17, 63, 122). Male buds should be removed to discourage insect transmission, and farm implements must be frequently disinfested. When diseased plants are found they and the neighboring plants should be destroyed with herbicides. These sites can be replanted after host residues have decayed (approximately 6–12 months). Removal of alternative weed hosts may be helpful (113). Stover (122, p. 199) stated that "all varieties of commercial bananas and plantains are susceptible. . ." However, Bluggoe and other ABB cooking bananas with dehiscent bracts are especially vulnerable because of their attractiveness to flying insect vectors; they are significant sources of inoculum for commercial bananas (63). In these situations, Pelipita ABB, which has persistent bracts, has been recommended as a replacement for other ABB cooking bananas. The identification of factors that are associated with new outbreaks could also help focus disease control efforts. For example, because 76% of the new Moko disease foci in export plantations in Colombia were associated with cableways used to transport fruit to packing stations (90), sanitation/disinfestation in these areas could be beneficial.

Banana streak disease. Banana streak disease is caused by a heterogeneous group of badnaviruses collectively called BSV (24). Infection by these single-stranded-DNA pararetroviruses can be devastating because of its direct impact on production and the indirect effect that the pathogens have had on the dissemination of materials from breeding programs. The international movement of improved but infected A × B hybrids has been impeded by the infection of some by species of BSV that integrate into the genome of the B parents (24, 51). The appearance of de novo copies of these species in tissue-culture progeny of what were virus-free materials resulted in a moratorium on their release by Bioversity's International Transit Center for *Musa* germplasm. Geering (51) listed several reasons why this policy should be reconsidered.

Parents of all domesticated bananas have integrated badnavirus DNA, but only those in the B genome of A × B hybrids are known to initiate infection (51, 63). Estimates of yield loss caused by banana streak disease vary widely for unknown reasons. Likewise, how or whether the different BSV species affect yield, how they spread (natural dissemination by mealybug vectors appears to be uncommon in most situations), and why the occurrence of episomal BSVs varies among different cultivars need study. Managing the impact of this disease and the occurrence of infectious integrants will require greater understandings of these variable viruses and how they interact with their banana hosts.

OUTLOOK FOR EXPORT AND SMALLHOLDER PRODUCTION

During the past half-century, dramatic changes have occurred among the world's leading producers and consumers of banana. Global production has increased by 323% since FAO began compiling figures in 1961 (42). Changes in the two leading Asian producers were most striking, as India and China registered 12- and 57-fold increases, respectively, from 1961 to 2011. Virtually all of the fruit that is produced in these countries is for domestic markets. In addition, dramatic changes occurred in the international commerce of this fruit (42). Although Ecuador has consistently led the exporting nations, virtually no bananas were exported in 1961 from the current number two exporter, the Philippines. Similarly, three of the top five importers in 2011 imported miniscule volumes of banana in 1961.

Much of the global production of this fruit comes from a very narrow genetic base. Although advances have been made in understanding the pedigrees and domestication of this ancient crop (94), considerable work remains to recreate disease-resistant versions of the currently grown clones and to develop new disease-resistant varieties that would appeal to consumers (90). Although some of these bananas might come from conventional breeding programs, others may eventually be produced via genetic engineering (90, 107). For all but TR4 and BLSD, natural resistance in banana is scarce or nonexistent. Although bananas that resist Moko disease, blood disease, Xanthomonas wilt, bunchy top, and banana streak might be produced by genetic engineering, willingness in the marketplace to accept engineered foods is needed. New and highly specific genome-editing technologies using transcription activator-like effector nucleases (TALENs) or clustered regularly interspaced short palindromic repeats (CRISPRs) (65) could potentially contribute to a new generation of improved products that meet consumer preferences. Overall, there is a critical need for intensified and modern banana breeding programs, as well as an open-mindedness for new or different organoleptic qualities in this fruit. Without these changes, it could be exceedingly difficult to solve the biotic and abiotic challenges that threaten a major staple crop and the world's favorite fruit.

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