The Vulnerability of Bananas to Globally Emerging Disease Threats

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ABSTRACT

Banana cultivation has increased significantly over the last century to meet the growing demand for this popular fruit. Originating in Southeast Asia, bananas are now produced in >135 different countries in tropical and subtropical regions of the world. Most of this expansion of production is based on a single banana variety, Cavendish, which makes up almost all the export trade grown in large-scale monocultures and a large part of the local trade and represents >40% of all bananas grown globally. Over the last century several major diseases of the banana have emerged and widely expanded their geographic ranges. Cultivars within the Cavendish variety are highly susceptible to these diseases, including yellow Sigatoka, black leaf streak, Eumusae leaf spot, freckle, Fusarium wilt tropical race 4, banana bunchy top, and the bacterial wilts Moko, *Xanthomonas* wilt, and banana Blood disease. This review graphically illustrates the emergence and rapid intercontinental spread of these diseases and discusses several major disease epidemics in bananas. Evidently, the large-scale monoculture based on the single variety Cavendish has resulted in an extreme level of genetic vulnerability. The resistance to diversification in the Cavendish production chain and the lack of investment in genetics and plant breeding in the recent past means that currently limited genetic solutions are available to replace the Cavendish banana with a set of market acceptable resistant varieties from a range of different genetic backgrounds.

Keywords: bacterial pathogens, Bridgehead effect, disease control and pest management, disease resistance, ecology and epidemiology, epidemic, epidemiology, fungal pathogens, genetic vulnerability, genetics and resistance, invasion, *Musa*, mycology, pandemic, techniques, virology

Bananas evolved in Southeast Asia and have been used for food, medicine, shelter, fiber, and many other purposes for a long period of time (Kennedy 2009). About 2,500 years ago early seafarers brought bananas and plantains, a type of cooking banana, to East Africa, where they became an important food crop (Perrier et al. 2011). After discovery of the Americas, bananas and plantains were introduced into Central and South America in the 16th century, where they became an important part of the local diet. Improved transport systems enabled the development of a large banana industry in Central and South America focused on export of fruit to North America and Europe from the early 20th century onward (Simmonds 1966; Soluri 2006). At present, banana production is common in most tropical and subtropical countries, and annually >100 billion bananas are consumed. Globally >400 million people depend on bananas as a staple food or as a source of income. The combined economic value of bananas is approximately US\$52 billion, of which >US\$8 billion represents the export of Cavendish bananas (FAO 2018). The majority of bananas (85%) are consumed

locally, and the remaining 15% are exported from tropical countries to countries mainly in the temperate zones. Bananas are the most valuable and most traded fruit in the world and the sixth most valuable food crop.

The early expansion of banana cultivation experienced few disease problems, which led Gäumann (1922) to state that “in publications of tropical plant diseases until about 1910 banana diseases typically did not rate a mention.” This statement, correct at the time, is certainly no longer true today because the production of bananas is threatened by a range of different plant diseases spreading around the globe, including yellow Sigatoka, black leaf streak, Eumusae leaf spot, freckle, Fusarium wilt tropical race 4, banana bunchy top, and the bacterial wilts Moko, *Xanthomonas* wilt, and banana Blood disease. The aim of this review is to explore the events over the last century that turned this statement around to a situation today where the banana is threatened by a plethora of diseases. Several aspects of cultivated banana plants resulting in genetic vulnerability, such as clonal reproduction and large-scale monoculture based on a single variety, are discussed. Special attention is paid to the origin, distribution, and increasing impact of an expanding range of diseases of the banana plant. The combination of these factors has turned the banana business into a fragile industry that needs attention and fundamental changes to secure its long-term survival.

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GENETIC MAKEUP AND SPREAD OF THE CLONAL BANANA

Banana and plantains belong to the genus *Musa*, which originated in Southeast Asia and the Pacific. The *Musa* genus consists of about 40 different species (Perrier et al. 2011; Volkaert 2018). Two of these *Musa* species, *M. acuminata* and *M. balbisiana*, and hybrids between them, have contributed to our edible bananas and plantains. *M. acuminata* consists of several subspecies (*banksii*, *zebrina*, *microcarpa*, *truncata*, *errans*, *burmannica*, and *malaccensis*), which contribute what is called the “A” genome, whereas *M. balbisiana* contributes the “B” genome (Perrier et al. 2011). Edible banana varieties are parthenocarpic, defined as producing fruit in the absence of successful fertilization and thus containing no seed, and are often hybrids between these different subspecies containing the “A” genome. Natural hybridization between these subspecies often gives rise to sterility, resulting in the development of seedless and hence edible fruit. This natural hybridization between the different species and subspecies has created highly heterozygous diploid (AA) and triploid (AAA, AAB, ABB) banana varieties (D’Hont et al. 2000). The dark lines within the flesh of a banana fruit after it is cut lengthwise are all that remains of the shrunken seeds. Parthenocarpy, resulting in fruit without seed, can occur through either lack of pollination or lack of fertilization. In the case of edible bananas, it is often the case that both the male and female flowers are sterile (Simmonds 1962).

The less diverse *M. balbisiana* also evolved in Southeast Asia and contains the “B” genome. Interspecific hybridization between *M. acuminata* and *M. balbisiana* has given rise to banana varieties such as ‘Lady Finger’ (AAB), ‘Pisang Awak’ (ABB), ‘P. Kepok’ (ABB/BBB), and all plantains (AAB). All *Musa* species producing seed in abundance are diploid, with a basic chromosome number of 11 (Osuji et al. 1997). Another group of bananas called Fe’i occur in New Guinea and the Pacific, carry 10 chromosomes, and have an unresolved genetic origin (Volkaert 2018).

Wild bananas contain many seeds, which makes the fruit inedible, but occasional intraspecific and interspecific hybrids occur that are often sterile because of incompatible combinations of fertility genes giving rise to pollen or seed not developing normally, resulting in sterility. This process occurs spontaneously where wild diploid bananas grow naturally in close proximity and is common in the center of origin in Southeast Asia, where there are anywhere between 350 and 1,000 known sterile banana varieties. In the natural environment these sterile offspring are no longer part of the evolving species because of their inability to pass on their genes in the form of gametes or seed. Only through involvement of humans, who learned how to identify such plants and propagate them through suckers sprouting from the corm, did they survive long term and spread. Interspecific and intraspecific hybridization between these *Musa* species and subspecies also gave rise to the production of unreduced gametes. When combined with the regular gamete it enables the production of triploids (AAA) among different *M. acuminata* subspecies and (BBB) within *M. balbisiana* but also between *M. acuminata* and *M. balbisiana*, resulting in interspecific hybrids designated as genome groups AAB and ABB. Triploids are often highly productive and produce larger plants with bigger bunches having large fruit with no seeds, which made them highly desirable for early farmers. The increased productivity of these triploids may result from significant inherent genetic diversity among the different species and subspecies. A variety such as Cavendish, being a triploid (AAA) made up of different subgroups within *M. acuminata*, is known to be highly heterozygous (Perrier et al. 2009; Risterucci et al. 2009). This may result in a very strong heterosis effect and explain the larger plants, bigger bunches, and high level of productivity of triploid hybrids compared with their diploid ancestors.

It has been hypothesized that the first edible diploid (AA) bananas were propagated by humans about 8,000 years ago in the

New Guinea region (De Langhe et al. 2009; Denham et al. 2003). For the next 5,000 years banana cultivation expanded in Southeast Asia through propagation of cuttings and selection of varieties with favorable characteristics and improvements in cultivation. Approximately 2,500 years ago a limited number of plants were taken on a few occasions by early seafarers from Southeast Asia to Africa (Volkaert 2018). These voyages formed the foundation for the cultivation of Mlali (Mchare) (AA), and the East Africa Highland banana Matoke (AAA) in East Africa and, through a separate introduction, the plantains (AAB) in West Africa (Karamura 1999). The African banana and plantain varieties grown today are all derived from these few introduction events of sterile plants thousands of years ago. It is of interest to note that the diploid banana varieties we find today in eastern Africa belonging to the subgroup Mlali turn out to be one of the major ancestors of the Gros Michel and Cavendish varieties (Raboin et al. 2005). However, material with the same genetic makeup has not been identified, or even may no longer exist, in the center of origin in Southeast Asia (Volkaert 2018). As part of the Columbian Exchange following the discovery of the Americas, bananas and plantains were introduced into Central and South America in 1516 (Reynolds 1951) and became an important food crop. At the end of the 19th century, refrigerated steamships, called reefers, gave rise to a large export industry in Central and South America to North America and Europe from the early 20th century onwards (Simmonds 1966). This export industry was first based on large-scale monoculture of a single variety called Gros Michel. This variety succumbed to *Fusarium* wilt race 1 and was replaced with the variety Cavendish, which subsequently became the most commonly grown banana in the world today.

Although there is great genetic diversity in the genus *Musa* in its center of origin, the story of the global distribution of bananas also illustrates that only a tiny fraction of that genus is used in modern-day commercial banana production. Currently, by far the most popular banana variety is Cavendish, which was a clone obtained by British colonial botanists in southern China in 1828. Today it is the single most popular product on the world’s supermarket shelves. Throughout the world all the Cavendish banana we eat (100 billion/year) are clonally descended from a sterile mutant selected by a Stone Age farmer. Well over a thousand sterile banana varieties have been selected over a long period of time in Southeast Asia, as can be seen in the large diversity of seedless fruit being grown and eaten in its ancestral home. However, outside the center of origin banana cultivation suffers from an extreme lack of genetic diversity. The whole export banana trade (15%) and a large fraction (25%) of local consumption in the tropics consist of the Cavendish variety. In addition to nearly 40% of the world banana production consisting of a single variety, another 20% consists of closely related varieties sharing a very similar genetic background. No other crop is as homogeneous as the banana while being grown in large plantations around the tropics, with the world export market based on a single banana variety. This lack of diversity creates a high level of risk, called genetic vulnerability, from disease.

GENETIC VULNERABILITY

Until about the end of the 19th century, farmers throughout the world used to select the best plants from their farm to collect seed or suckers for the next season as planting material. This process gave rise to large numbers of so-called landraces of many of our cultivated crops. Although productivity was low, yield stability was high because of the growth of large numbers of diverse varieties well adapted to local environmental conditions that yielded well in a wide range of environmental conditions. The advent of plant breeding and modern farming techniques in the 20th century changed all this. Farming communities today are no longer involved in the selection of their seed but instead buy it from seed

companies that use hybridization techniques to combine favorable traits of a crop into commercial varieties. For some crops, such as maize, hybrids are produced to use the heterosis effect (Sprague 1983). The advent of plant breeding in the 20th century, combined with the globalization of seed production and improved cultivation techniques, immensely increased crop productivity but also led to the unintended erosion of genetic diversity (Day-Rubenstein et al. 2005; National Research Council 1972; Smolders 2006). Increased genetic uniformity has given rise to what is called genetic vulnerability, defined as “the risk of large-scale crop failure from diseases or abiotic factors” (National Research Council 1972). The level of genetic vulnerability depends on the area under cultivation by a single variety, the global connectedness of an industry, and the presence and rate of spread of diseases and pests. Having established the background to the high level of genetic uniformity in banana cultivation in most parts of the world, and in the Cavendish-based export markets in particular, we discuss the emergence, spread, and impact of diseases on the Cavendish monoculture production system.

MAJOR DISEASES OF BANANA

Having globally >40% of a crop consisting of a single variety and a global export industry relying on that single banana variety creates a high level of vulnerability to diseases that are able to disseminate rapidly and have a high impact on productivity. The tropical zone in Southeast Asia where bananas originated and are grown has one of the highest levels of biodiversity in the world. This diversity not only is evident in animals and plants but is also reflected in the number of microorganisms able to cause disease. The number of known banana diseases has been reported to be >180, of which 10 cause severe disease (Wellman 1968). Some of these pathogens have coevolved with the banana in its center of origin in Southeast Asia, and others are so-called new encounter pathogens that evolved elsewhere in the world and are able to cause disease but have not coevolved with bananas in its center of origin (Table 1). Not only has the spread and large-scale cultivation of bananas around the world led to the occurrence of more banana diseases, but the increased global movement of both coevolved and newly encountered pathogens has

significantly increased the overall disease pressure on the widely grown Cavendish variety.

ORIGIN OF EMERGING BANANA DISEASES

The genus *Musa* evolved in Southeast Asia, and with it a large number of pathogens have coevolved. Coevolution is an ongoing process that removes the highly susceptible individuals from a host plant population and thus favors the more resistant ones to survive and genetically contribute to the next generation. The genus *Musa* is diverse, and interspecific and intraspecific hybridization have increased this diversity even more. Pathogens need to continually adapt to changes in the host plant to retain their ability to infect and colonize their host populations with ever-increasing levels of resistance. This gives rise to an ongoing arms race between the plant and the pathogen. Although we know the center of origin of most *Musa* subgroups (Perrier et al. 2011) the center of origin for banana plant pathogens is much harder to pinpoint. To some degree this difficulty is caused by elevated levels of resistance in the host plant due to pathogen pressure leading to a limited visible impact of the pathogens in the center of origin. The movement of plants and pathogens by humans over the last few millennia has further complicated the picture. With regard to banana there is evidence that Fusarium wilt, yellow Sigatoka, black leaf streak, banana bunchy top virus, freckle, and banana blood disease originate from the center of origin of *Musa* species in Southeast Asia.

When bananas were cultivated outside the center of origin on other continents, pathogens emerged with the ability to colonize this newly introduced host and caused what are commonly known as new encounter diseases (Drenth and Guest 2016). Examples are Moko, caused by several different strains of *Ralstonia solanacearum* in two different places in Latin America (Buddenhagen 1961), and Xanthomonas wilt, caused by *Xanthomonas vasicola* pv. *musacearum*, previously known as *X. campestris* pv. *musacearum* in Africa (Blomme et al. 2017; Studholme et al. 2020). Often these pathogens originate from related host species such as *Heliconia* for Moko in Central America (Sequeira and Averre 1961) and *Ensete* from Ethiopia in the case of Xanthomonas wilt (Yirgou and Bradbury 1974). The pan-tropical distribution and cultivation of bananas has

TABLE 1
Origin and current distribution of the nine most important banana diseases

Disease	Pathogen	Coevolved in center of origin	Place of origin	Current distribution
Fusarium wilt race 1	<i>Fusarium oxysporum</i> f. sp. <i>cubense</i> race 1	Yes	Southeast Asia	Pantropical
Fusarium wilt tropical race 4	<i>F. oxysporum</i> f. sp. <i>cubense</i> tropical race 4 (<i>F. odoratissimum</i>)	Yes	Southeast Asia	Asia, Australia, Africa, Middle East, Europe, Colombia
Yellow Sigatoka	<i>Pseudocercospora musae</i>	Yes	Southeast Asia	Pantropical
Black leaf streak	<i>Pseudocercospora fijiensis</i>	Yes	Southeast Asia	Pantropical, (excluding Australia)
Freckle	<i>Phyllosticta cavendishii</i>	Yes	Southeast Asia	Southeast Asia (excluding Australia)
Banana bunchy top	<i>Banana bunchy top virus</i>	Yes	Southeast Asia	Asia, Australia, Africa
Blood disease	<i>Ralstonia syzygii</i> subsp. <i>celebesensis</i>	Yes	South Sulawesi	Indonesia, Peninsular Malaysia
Moko	<i>Ralstonia solanacearum</i>	No	Costa Rica, Amazonia	Central and South America, the Philippines, Malaysia
Xanthomonas wilt	<i>Xanthomonas vasicola</i> pv. <i>musacearum</i>	No	Ethiopia	Central Africa

thus further expanded the number of coevolved and new encounter plant diseases. The lack of any genetic diversification in commercial plantations has increased the risk that if a new disease emerges on Cavendish, it only needs to spread to threaten >40% of the global banana production. It is this increase in spread of pathogens around the world over the last few decades, caused in part by increases in global travel and trade, that is threatening the production of the Cavendish banana.

GLOBAL SPREAD OF PATHOGENS

The impact of disease, from the center of origin or new encounter pathogens, rises rapidly if pathogens are able to spread globally. The increase in travel and trade in the 20th and 21st centuries has given rise to the unprecedented spread of pathogens at a global scale. Examples include *Fusarium* wilt race 1 and *Fusarium* wilt tropical race 4, yellow Sigatoka, black leaf streak, banana bunchy top virus, Moko, and banana blood disease. In the following sections the emergence and global spread of these major banana pathogens are documented.

Fusarium wilt. An early indication of the dangers of genetic vulnerability of the modern monoculture banana production system was the decimation of the variety Gros Michel in the Latin American banana export industry beginning in the 1920s. Gros Michel was obtained by French explorer, navigator, and naturalist Nicolas

Baudin in Canton (Guangzhou) in China around 1790 and transported to Martinique (Hammond 2019). This material was subsequently brought to Jamaica by French botanist Jean François Pouyat in 1835. This single Gros Michel (AAA) clone is believed to be the foundation on which the entire banana export industry in the Americas was built. From the 1880s onward growing markets, railways, and refrigerated steamships led to a rapidly expanding industry in Central and South America based on this single banana clone. The danger of relying on a single clone came to light when *Fusarium* wilt race 1 (aka Panama wilt) (Fig. 1) was accidentally introduced from Asia to the newly developed megaplantations in Latin America. *Fusarium* wilt was first noted in Panama and Costa Rica in the 1890s (McKenney 1910; Stover 1962a) and subsequently Jamaica in 1903, Suriname in 1906, Trinidad and Tobago in 1907, Cuba in 1908, Guatemala in 1910, Colombia in 1929, and Venezuela in 1930. Although Bancroft (1876) first reported the disease from Australia, the first detailed account of the pathogen and the disease was published by Ashby (1913). Once *Fusarium* wilt established a foothold in Latin America it spread quickly and devastated the large plantations consisting of the highly susceptible variety Gros Michel in Central and South America over a few decades (Marquardt 2001; Soluri 2006). A large research effort and huge disease control measures, including the flooding of thousands of hectares of infested land, had large ecological and environmental impacts but did not result in effective control. Efforts to introduce genetic resistance through plant breeding into a Gros Michel background started but were not successful, and the only option at hand was relocation to uninfested land. The need for clean land, the shifting cultivation, and the spread of *Fusarium* wilt resulted in high-level political interference and corruption, giving rise to the term “banana republic” (Koeppel 2018; Marquardt 2001). The *Fusarium* wilt epidemic brought the Gros Michel-based export industry to its knees and has been estimated to have cost the equivalent of >US\$2.7 billion today (Ploetz 2005). The only viable way to save the banana export industry was to move away from the Gros Michel banana variety and replace it with another variety. This new variety, called Cavendish, was discovered by British colonial botanists in southern China in 1826 and brought to Chatsworth House in the Midlands, Derbyshire, in the United Kingdom in 1829. Here it was grown successfully in a glasshouse by Joseph Paxton, a keen gardener of the duke who coined the name “Cavendish” after his employer, William Spencer Cavendish, the sixth duke of Devonshire. Shoots from these plants were sent to various British colonies around the world and formed the basis of the banana industry after the loss of Gros Michel. In the 1950s and 1960s Gros Michel plantations were replaced with monoculture Cavendish plantations. Although it lacked taste and transportability, its complete resistance to *Fusarium* wilt race 1 was the defining factor, making it the most widely grown banana variety in the world today. Industry changed its supply chain and introduced boxes for transport, and consumers adopted the new variety in the absence of Gros Michel.

Fusarium wilt was first reported from Africa in 1924 in Gros Michel plantations in Nigeria, Sierra Leone, and Cameroon in West Africa and the Canary Islands. From there it spread to Ghana in 1937 and Guinea in 1939. On the other side of the African continent it was first identified in Mauritius in 1930, Madagascar in 1961, and Ethiopia and Mozambique in 1970 (Anonymous 1977). Another independent introduction may have taken place in Tanzania in 1951, followed by Kenya (1952) and Uganda (1953) (Stover 1962a). It was subsequently reported in Burundi in 1980 and the Democratic Republic of Congo in 1985 (Gatsinzi and Sebasigari 1989). The global spread of *Fusarium* wilt race 1 over time is graphically represented in Figure 2. The situation in Central and South America and Africa shows that once *Fusarium* wilt invades a new continent and gets established, further secondary invasions take place, giving rise to local spread. This phenomenon, in which initial invasions to a new area are followed by numerous secondary

FIGURE 1

Fusarium wilt race 1 affected Gros Michel in Costa Rica (photo by Rafael Mena).

invasions originating from the source population, is called the bridgehead effect (Lombaert et al. 2010). *Fusarium* wilt race 1 is now widespread across Central and South America and Africa, and there are few places left where race 1 is not present. When the banana export industry switched from monoculture Gros Michel to monoculture Cavendish, the *Fusarium* wilt problem disappeared into the background. However, the intrinsic vulnerability of growing a single clone on a global scale was not addressed, and no diversification took place. This led one of the leading scientists at the time, Dr. Harry Stover, to state that the banana industries 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 (Stover 1986).

Fusarium wilt TR4. Disease symptoms akin to *Fusarium* wilt in Cavendish were first identified in Taiwan in 1967 (Su et al. 1977, 1986). The pathogen spread rapidly and had a major impact

on the banana industry in Taiwan (Su et al. 1977). After some initial confusion the strain was designated as VCG 01213 (Ploetz 2015). This same strain was also the one that devastated newly established Cavendish plantations in Western Indonesia and peninsular Malaysia in early 1992 and was given the designation Foc-tropical race 4 (TR4) (Pegg et al. 1994). More recently, TR4 was found to be genetically distant from other species causing *Fusarium* wilt in bananas and therefore described as a new species, *Fusarium odoratissimum* (Maryani et al. 2019). The presence of this new race made the production of Cavendish increasingly difficult in affected areas (Buddenhagen 2009). *Fusarium* wilt spreads easily through the movement of infected plant material, surface and irrigation water, and soil attached to shoes, clothes, tools, and machinery. *Fusarium* wilt survives in the soil and on asymptomatic hosts for long periods of time, between 20 and 40 years (Buddenhagen 2009; Stover 1962b). After decades of research to manage *Fusarium* wilt race 1 and more recently TR4 in bananas, no cost-effective options other than exclusion and resistance have been identified.

It is believed that plant material transported from Taiwan to China brought the pathogen to mainland Asia in 1996. This invasion provided the bridgehead from which TR4 spread widely in China from 1997 onward. At present >100,000 ha has already been affected by TR4 in China (Figs. 3 and 4) and major shifts in production to new areas further west, not yet affected by *Fusarium* wilt, have taken place (Drenth 2019). Because limited land is suitable for banana cultivation in China, production has also shifted to neighboring countries in the south. Lack of awareness of the disease and ways to prevent its further spread resulted in the disease being introduced to neighboring countries. Recent surveys have shown that the particular strain of TR4 found in China has spread further and is now found in the neighboring countries Laos, Myanmar, and Vietnam (Zheng et al. 2018).

FIGURE 2

The cumulative number of countries affected by *Fusarium* wilt race 1 (Data obtained from Table 3.1 in Ploetz 2019).

FIGURE 3

Cavendish bananas affected by *Fusarium* wilt TR4 in China.

Foc TR4 was identified in 1997 in northern Australia, where it destroyed a small local industry. Interstate quarantine was put in place to reduce the risk of spread to the main banana production area in North Queensland. This quarantine was effective, but after 18 years TR4 was identified in the main production area in Tully in North Queensland in 2015. A large containment activity, which involved buying the first infested farm and taking it out of production and putting several biosecurity measures in place in the affected area, has seen the pathogen confined to five farms in a small area at present.

Foc TR4 was identified in the Philippines in 2005 but may have been present there several years before and confused with Moko, because the first report outlined the presence of many cases indicative of prior spread in the country (Molina et al. 2009). The lack of effective biosecurity and the promotion of untested and ineffective containment measures, coupled with the reliance on less susceptible varieties lacking complete resistance, which have the potential to increase the inoculum load, has seen the pathogen spread rapidly and widely in the Mindanao area and threatens the long-term

productive capacity of the large export industry, as well as small-holder production of bananas in the Philippines.

Detailed analysis of the strains causing disease revealed that the invasion of TR4 in Pakistan is linked to the strain prevalent in the Philippines (Zheng et al. 2018). Recently, TR4 has been reported in four different states in India and is believed to have been present since 2015 (Damodaran et al. 2019; Kulkarni 2018; Thangavelu et al. 2019). Unfortunately, no data or biological materials have been made available to determine the identity of the strains present there. India is by far the largest banana producer in the world, and the spread of this disease will have devastating consequences for their banana production and potentially for food security.

Another example of the bridgehead effect concerns the intercontinental spread followed by local spread after Foc TR4 was found in Lebanon in 2012 and a genetically similar strain was found in Jordan in 2013 (Zheng et al. 2018). TR4 was also reported in neighboring Israel in 2016, and a recent single-nucleotide polymorphism analysis revealed that the isolates from Israel, Jordan, and Lebanon show a high level of genetic similarity, suggesting a common source (Maymon et al. 2020).

Recently TR4 has also taken a foothold in Africa, detected in a Cavendish plantation in Mozambique (Butler 2013). Although it has spread to other farms in Mozambique, no information is available about strain identity or further spread on the African continent. The susceptibility of the Eastern African Highland banana and plantains to TR4 has been assessed, with most cultivars showing good levels of resistance (Molina et al. 2016).

In June 2019 the Colombian government confirmed the presence of TR4 on four farms in the Guajira region in northern Colombia (García-Bastidas et al. 2020; Stokstad 2019). Although several quarantine measures were implemented, the number of infested farms in that region more than doubled to 10 within a year. In April 2021 an outbreak of TR4 was confirmed on a small farm in Querecotillo in the Chira Valley in northern Peru, where several thousand small-scale farmers produce organic bananas (FAO 2021). If no rigorous efforts are undertaken to contain the pathogen in these initial outbreaks, it is likely that the disease will spread rapidly through central and South America along similar lines that *Fusarium* wilt R1 spread almost a century earlier.

Data on the global expansion clearly indicate that the spread of *Fusarium* wilt TR4 to other countries is accelerating, as illustrated in Figure 5. The estimated cost of the global damage caused by the *Fusarium* wilt R1 outbreak is about US\$2.7 billion in today's currency (Ploetz 2015). The global banana industry has grown several fold in the last 50 years, with a global economic value of bananas of >US\$52 billion per year. The total impact of TR4 on the global banana industry is estimated at US\$10 billion (Drenth 2019). The high level of susceptibility combined with the lack of effective

FIGURE 4

Internal symptoms of the pseudostem of Cavendish affected by *Fusarium* wilt TR4.

FIGURE 5

The cumulative number of countries affected by *Fusarium* wilt TR4. Data from Promusa.org.

control options for TR4 means the Cavendish era is coming to an end and that new resistant germplasm is needed more than ever to secure the viability of the banana industry and increase the sustainability and the profitability of the production practices.

Freckle. Freckle causes leaf and fruit spotting on bananas. On leaves, these spots may appear as streaks because drops containing spores run over the leaves, leaving a trail of spores causing infection. Raised pycnidia form on the upper leaf surface (Fig. 6A), giving affected leaves a sandpaper feel in contrast to healthy leaves, which are smooth. The infected fruit are unsuitable for export (Fig. 6B), but some amount of freckle on the fruit can be tolerated in local markets with short supply chains.

The causal agent of freckle was long thought to be various strains of *Guignardia musae* (anamorph *Phyllosticta musarum*). Recent research has identified three different causal agents: *Phyllosticta maculata*, *Phyllosticta musarum*, and *Phyllosticta cavendishii* (Wong et al. 2012). *Phyllosticta maculata* is common in Southeast Asia and Oceania on banana AAB and ABB varieties. *Phyllosticta musarum* has been identified in India, Thailand, and China (Wu et al. 2014) on AAB and ABB banana varieties. *P. cavendishii* infects AAB varieties and many varieties belonging to the AAA genomic subgroup to which Cavendish belongs.

Freckle is common in Southeast Asia, Oceania, Pakistan, India, Sri Lanka, and Bangladesh, and although records of the disease in Africa and the Caribbean exist, it is unclear which species are involved. As far as we are aware the disease has not been identified in the Cavendish plantations in Central and South America.

Although freckle is not considered the most important plant pathogen of bananas, some Cavendish varieties are highly susceptible. In fact, in Taiwan the Pei Chiao banana variety, which is the basis of the somaclonal variants with reduced susceptibility to Fusarium wilt TR4, is highly susceptible. In Taiwan, where these varieties are widely grown, freckle has a higher impact than black leaf streak (Tsai et al. 1993). In the Philippines freckle is an important disease necessitating regular applications of fungicides to avoid fruit being downgraded for export (Corcolon and Raymundo 2008). Efforts to keep Cavendish free of this emerging disease prompted an extensive and ultimately successful *P. cavendishii* eradication campaign in northern Australia in 2013 (McMaster et al. 2020).

FIGURE 6

Freckle caused by *Phyllosticta cavendishii* on **A**, the leaf and on **B**, the fruit.

Sigatoka. There are three closely related diseases of banana caused by three different species of *Pseudocercospora*: yellow Sigatoka, caused by *Pseudocercospora musicola*; black leaf streak (i.e., black Sigatoka) (Crous et al. 2021), caused by *Pseudocercospora fijiensis*; and Eumusae leaf spot, caused by *Pseudocercospora eumusae*. These three leaf diseases have had major influences on banana production around the world at different points in time with regard to their spread, distribution, and impact.

Yellow Sigatoka. Yellow Sigatoka was first recorded on Java in Indonesia (Zimmermann 1902). The fungus infects the leaves and causes elongated lesions with chlorotic halos (Fig. 7), which reduce the photosynthetic leaf area. Initial small lesions scattered over the leaf coalesce, causing premature death of the leaves. This lowers the yield and initiates premature ripening of the fruit (Meredith 1970).

The disease first became a problem in the Cavendish plantations established in the Sigatoka Valley in Fiji (Philpott and Knowles 1913). It spread quickly and was found in Sri Lanka in 1919, the Philippines in 1921, and Australia in 1924. In the 1930s it spread to Central and South America (Fig. 8) and eastern Africa (Fig. 9). It was detected in India and Brazil in the 1940s, and by the 1960s it had reached the limits of its distribution, and it has been recorded in almost all banana growing countries (Jones 2003, 2019; Stover 1962a). Genetic analysis of the pathogen population supports the hypothesis that Southeast Asia is the center of origin (Hayden et al. 2003).

FIGURE 7

Yellow Sigatoka symptoms on Cavendish leaf.

FIGURE 8

The cumulative number of countries in Latin America and the Caribbean affected by yellow Sigatoka (data obtained from Table 1 in Jones 2003).

The intercontinental movement of plant material played a major role in the global dissemination of this pathogen. When yellow Sigatoka first entered the export banana plantations in Central and South America, it disrupted the production in several countries. Only through the use of approximately 15 fungicides applications per annum were plantation managers able to keep the disease under control (Meredith 1970). The plantation industry was able to resume trade at previous levels, but the cost of production increased significantly, in some cases by 14% (Cook et al. 2013a).

Black leaf streak. Black leaf streak is the most important foliar disease of bananas. In comparison with yellow Sigatoka, it is more aggressive (Fig. 10) and affects many more banana varieties (Guzmán et al. 2019). This has played out in the field to such a degree that black leaf streak has in almost all cases replaced yellow Sigatoka in areas where they both occur (Guzmán et al. 2019).

Black leaf streak was first noticed in the Sigatoka Valley in Fiji in 1963 (Rhodes 1964). Surveys for this new pathogen revealed that it was already widespread in Oceania and the Pacific (Johnston 1965; Stover 1978). Investigations by Stover revealed that the disease might have been present much earlier in Papua New Guinea and Taiwan (Stover 1978). In 1980, it invaded China, and within a decade it was found throughout the banana growing regions of China and Vietnam (Jones and Mourichon 1993). At present, the disease is widespread in Southeast Asia and has invaded Australia several times, with an incursion into the major production area around Tully in 2001. Here, black leaf streak was eradicated through a concerted effort of researchers, growers, and state and federal government agencies, which is the only time this pathogen has ever been eradicated from a banana production area (Henderson et al. 2006).

FIGURE 9

The cumulative number of countries in Africa affected by yellow Sigatoka (data obtained from Table 1 in Jones 2003).

FIGURE 10

Black leaf streak symptoms on Cavendish leaf.

Although the disease is present in the center of origin of the banana in Southeast Asia, the damage in smallholder production systems is lower than in monoculture Cavendish plantations. This difference may be due to higher levels of resistance in the varieties in combination with different and more diversified agronomical settings. In addition, problems such as premature ripening, which are disastrous for long-range export markets, have less impact on short-range domestic markets.

Human movement of plant material facilitated the entry of black leaf streak in a germplasm collection block of the United Fruit Company in Honduras in 1972 (Stover and Dickson 1976). From this bridgehead in Central America, it spread further through the monoculture Cavendish plantations of Latin America. Its spread and establishment were also supported by its ability to infect plantain crops, often grown in smaller plots as part of traditional cropping systems. From this initial invasion in 1972 the disease spread rapidly, and by 1981 it was present in most banana growing countries in Central America (Stover 1987). In 1981, it was found in northern Colombia, from which it spread to the main banana producing regions in South America, including Ecuador (1986), Venezuela (1991), Peru (1994), northern Brazil (1998), and the southern states of Brazil (2004). In the Caribbean, the disease invaded from two fronts (Carlier et al. 2021). Black leaf streak was reported in Cuba in 1990 (Vidal 1992), from which it spread east, ending up in Puerto Rico in 2004 (Irish et al. 2006; Ploetz 2004). The second invasion started in Trinidad and Tobago in 2003, from which it spread north, reaching Guadeloupe in 2012 (Hubert 2012). Hence, in 40 years black leaf streak disease had disseminated to all the banana growing countries in the Americas (Fig. 11). Although reported disease losses are low in well-managed plantations (Strobl and Mohan 2020), the cost to achieve this control is often >25% of the production costs of bananas (Stover and Simmonds 1987).

Black leaf streak is believed to have entered Africa on infected plant material after it was detected on horn plantain in Gabon in 1978 (Blomme et al. 2013; Frossard 1980). From this bridgehead, it spread rapidly to the Democratic Republic of Congo, Nigeria, and Ghana (Wilson and Buddenhagen 1986). In East Africa black leaf streak first appeared in coastal Mozambique in 1987 (Dabek and Waller 1990), and from there it spread to Tanzania and Kenya in 1988 (Kung'u et al. 1992), up to Ethiopia (Gurmu et al. 2017). Today, most banana growing countries in Africa are affected by black leaf streak (Fig. 12).

Eumusae leaf spot. The third leaf spot disease, Eumusae leaf spot, formerly known as Septoria leaf spot, has a less well-defined distribution (Crous and Mourichon 2002). The disease symptoms look similar to those of black leaf streak and are easily confused

FIGURE 11

The cumulative number of countries in Latin America and the Caribbean affected by black leaf streak (data obtained from Table 2.1 in Guzmán et al. 2019).

with this disease, and its fungal morphology is more closely aligned with *Pseudocercospora musae*, causing yellow Sigatoka. Eumusae leaf spot was first recognized in Nigeria, with the first record dating back to 1989 (Carlier et al. 2000). Subsequent reanalysis of isolates of banana leaf disease revealed that it was present in southern India, Sri Lanka, Vietnam, western Malaysia, Mauritius, and Thailand (Carlier et al. 2000). Because the disease symptoms look very similar to those of black leaf streak, it may be that the distribution of this pathogen is much wider, and only detailed molecular analysis of the fungal pathogen populations will reveal the differences (Arzanlou et al. 2007). The impact of this disease is similar to that of black leaf streak, but in some areas where it occurs, such as India, it has become the most dominant leaf disease, indicating its competitiveness compared with black leaf streak and yellow Sigatoka (Devi and Thangavelu 2014).

Banana bunchy top. The most important virus disease of banana is caused by *Banana bunchy top virus* (BBTV) (Kumar et al. 2015). The virus has a multicomponent genome with six different single-stranded DNA components and belongs to the genus *Babuvirus*, family *Nanoviridae* (Vetten et al. 2012). The virus particles are restricted to the phloem of infected plants (Magee 1939). A range of disease symptoms are associated with BBTV, including dark green streaks in a typical dot-dash pattern on the minor leaf veins, midribs, and petioles and chlorotic, upturned leaf margins. The most striking whole plant symptoms are the stunted and bunched appearance of short and narrow leaves at the top of an infected banana plant (Fig. 13), from which the disease gets its name, and the failure to produce fruit.

The banana aphid (*Pentalonia nigronervosa*) spreads the virus, and once a plant is infected, it remains infectious for life (Magee 1927). If not removed, infected banana plants remain a source of infection for spread of the virus to new plants. Therefore, management is based on regular inspection for symptoms and removal and destruction of the infected plants, including the corm, to prevent further dissemination. Another important component of disease management is the availability of virus-free planting material, either through tissue culture or sourced from an area not affected by the disease. In addition to aphids, movement of infected plant material is major cause of spread of the disease, especially long-distance movement. Banana varieties carrying the B genome (AAB and ABB) are often found to be less affected than cultivars only containing the A genome (AA and AAA), but no complete resistance has been reported (Thomas 2019). There are no reports of infection of wild diploid *Musa* species.

BBTV first rose to notoriety when it appeared in Fiji in 1879 (Darnell-Smith 1924) and caused serious production problems in

the newly established Cavendish production system from 1894 onward. It was next found in Egypt in 1901, then reported in Australia and Sri Lanka in 1913. After its introduction into Australia in 1913, the local banana industry nearly collapsed, and it was only through the research efforts of Magee (1927) that an effective management strategy was developed. Ongoing investment over the last century to conduct inspections and destroy infected plants has kept the virus contained in a localized area in northern New South Wales and southern Queensland, and the main production areas in North Queensland are still free of the pathogen.

BBTV isolates exist as two genetically distinct groups: the “South Pacific” group occurring in Australia, Africa, and South Asia and a more genetically diverse “Asian” group generally occurring in Southeast Asia (Karan et al. 1994). The “South Pacific” group escaped its ancestral home and spread intercontinentally to be recognized as the most devastating viral disease of banana (Dale 1987; Stainton et al. 2012, 2015).

Over the last century, the virus has spread more widely into Asia (Fig. 14), with recent incursions in Pakistan, Laos, Myanmar, and Thailand to such a degree that the virus is now present in 18 Asian

FIGURE 12

The cumulative number of countries in Africa affected by black leaf streak (data obtained from Table 2.1 in Guzmán et al. 2019).

FIGURE 13

Banana bunchy top symptoms.

countries (Stainton et al. 2015). Further spread in Oceania has also occurred, with the disease now present in 12 countries (Thomas 2019).

BBTV first entered Africa through Egypt in 1901 (Fahmy 1927), from which limited spread occurred. However, once a new incursion took place in the 1950s in Central Africa in the Democratic Republic of Congo (Wardlaw 1961) it established a bridgehead from which it continued to spread and now causes bunchy top disease in 15 African countries. Spread has increased lately, and since 2007 it has invaded Mozambique, Angola, Cameroon, Nigeria, Benin, Zambia, and South Africa, causing havoc across Africa, particularly in countries where agronomy services are limited. A recent incursion in Togo was detected early and eradicated (Kolombia et al. 2021). Thus, BBTV is now present in most countries from South Africa in the south, Nigeria in the west, and Egypt in the northeast on the African continent (Fig. 15). The only areas still free of the disease are Central and South America and the Caribbean (Thomas 2019).

Although BBTV can be controlled through regular inspection and removal of infected plants, the impact of this disease is devastating for smallholder farmers not used to regular replanting and having limited access to pathogen-free planting material. Larger commercially oriented growers replant more often and typically use tissue culture and nurseries that source plants from disease-free areas to limit the risk of establishment and spread of the virus.

BACTERIAL WILT DISEASES OF BANANA

Bananas are affected by several bacterial wilt diseases, of which the main ones are banana Blood disease, Moko, and *Xanthomonas* wilt. Typical symptoms include wilting of the leaves, internal vascular discoloration in the pseudostem and peduncle, and internal rot and discoloration of green fruit.

Blood disease. The first recorded major impact of bacterial wilt on bananas grown in monoculture appeared when the Dutch started growing the cooking banana Pisang Kepok in monoculture on several of the Selayar islands south of Sulawesi. After reports of disease problems around 1910, production collapsed on several islands, notably Kayudi and Tampea. This gave rise to a general investigation (Rijks 1916) followed by the detailed studies by Gäumann (1921, 1922, 1923). The bacterium was isolated and Koch's postulates performed, and the pathogen was given the name *Pseudomonas celebensis* (Gäumann 1923). Recent research in this group of *Ralstonia* plant pathogens renamed the causal agent *Ralstonia syzygii* subsp. *celebesensis* (Safni et al. 2014). Insight into the spread of the pathogen and details on disease etiology were described, and testing of nearly a hundred banana varieties revealed that there was limited resistance to the Blood disease bacterium.

FIGURE 14

The cumulative number of countries in Asia and the Pacific affected by *Banana bunchy top virus* (data obtained from Table 6.1 in Thomas 2019).

Subsequent investigations revealed that symptoms of blood disease include wilting of the leaves, internal vascular discoloration in the pseudostem and peduncle (Fig. 16), and internal rot and discoloration of green fruit (Fig. 17). The disease is caused by a bacterium that is present throughout the low-lying areas of southwestern Sulawesi, which led Gäumann to conclude that the disease did not originate from the Selayar islands itself but that it was endemic in southern Sulawesi, where it was common in wild bananas grown in virgin forests (Gäumann 1923). Locals reported occasional flare-ups of the disease. It seems logical to conclude that the pathogen was introduced to the Selayar island group about 30 years after a banana plantation industry was established there. From 1910 onward, banana production collapsed on the islands within a very short period of time, and efforts to revive the industry have not been successful because of recurrence of Blood disease. The dense plantations consisting of a single variety, in this case Kepok, facilitated explosive epidemics, which destroyed the industry in a short time. Based on Gäumann's findings, a quarantine order was put in place restricting the movement of bananas and banana planting material from this part of the world to the rest of Indonesia. For 60 years, the disease was contained in Selayar and southwestern Sulawesi.

This containment was effective until 1987, when an outbreak of the disease occurred in West Java (Eden-Green and Sastraatmadja 1990). From Java it continued to spread rapidly to other islands in Indonesia including Kalimantan, Sumatra, Sumbawa, Bali, Maluku, West Papua, and recently to peninsular Malaysia (Baharuddin et al. 1994; Davis et al. 2000; Kusumoto et al. 2004; Safni et al. 2014; Supriadi 2005; Teng et al. 2016). Recent detailed surveys report

FIGURE 15

The cumulative number of countries in Africa affected by *Banana bunchy top virus* (data obtained from Table 6.1 in Thomas 2019).

FIGURE 16

Internal discoloration of the peduncle caused by banana Blood disease.

and confirm the disease from most of the larger islands of the Indonesian archipelago (Ray et al. 2021). Because of its occurrence in Malaysia on mainland Asia, it is expected to spread throughout Asia if nothing is done to halt it. The lack of resistance in banana varieties implies labor-intensive and costly management practices to control this disease in banana plantations.

Moko. A bacterial wilt problem in bananas was first noted in Trinidad in the 1890s (Rorer 1911). Multiple banana varieties were affected, but a cooking banana called Bluggoe (ABB) (i.e., Moko) was particularly susceptible, and the disease was named when the pathogen devastated this variety.

Moko is caused by *Ralstonia solanacearum*, which is divided into strains, races, biovars, phylogroups, and sequevars. Strains able to cause Moko disease in bananas are in biovar 1 and race 2, phylo-type 2. Initial strain variation was noted, and the banana or B strain originated from wild *Heliconia* species present in areas cleared for banana plantations in Central America. The B strain is persistent in the soil and spreads with planting material and cultivation practices in plantations (Buddenhagen 1960), causing significant damage in Cavendish plantations (Fig. 18).

Another strain, called SFR (small fluid round), was first noted in Honduras on Bluggoe bananas introduced from Venezuela in the early 1960s (Blomme et al. 2017; Buddenhagen and Elsasser 1962; Sequeira 1998). The SFR strain produces large amounts of bacterial ooze from the inflorescence, which facilitate insect transmission, in contrast to the B strain, which has a more soilborne nature. These SFR strains are believed to have originated from the Amazon basin and have spread to many places in Central and South America and parts of the Caribbean (French and Sequeira 1970). Moko is clearly a new encounter disease with different strains that arose in different areas in the Americas, indicating that this is a polyphyletic

pathogen from *Heliconia* species and other hosts with pathogenicity toward the introduced bananas (Sequeira and Averre 1961).

Moko is also found in the Philippines, and this invasion is reported to be the result of the movement of plant material from Honduras (Fegan and Prior 2006). Further confirmed reports of the disease come from Malaysia (Zulperi and Sijam 2014). Moko is considered a serious disease of bananas, and its control is costly. It involves using pathogen-free planting material, avoiding infection through regular decontamination of tools in the field, and regular inspections to remove infected and surrounding plants from within and around plantations. Because the disease is also soilborne, a waiting period of 6 months is needed before replanting (Sequeira 1962).

Xanthomonas wilt. The other new encounter banana disease is Xanthomonas wilt, caused by the bacterium *X. campestris* pv. *musacearum*, recently renamed *X. vasicola* pv. *musacearum* (Studholme et al. 2020). This disease was first reported in Ethiopia on Ensete (*Ensete ventricosum*), also known as false or Ethiopian banana (Castellani 1939; Yirgou and Bradbury 1968), originating from high-rainfall forests on the eastern edge of the great African Plateau. It does not produce edible fruit, but the trunk and inflorescence stalk are pulverized and eaten.

It was found that the bacterium was also able to cause disease on commercially grown banana cultivars (Yirgou and Bradbury 1974). The causal agent remained geographically confined to Ethiopia until about 2000, when it was reported to cause disease in East African Highland bananas in Uganda (Tushemereirwe et al. 2003) and the Democratic Republic of Congo (Ndungo et al. 2004). The pathogen's geographic range increased rapidly over the next decade, and the disease has been reported to cause damage in Ethiopia, Uganda, the Democratic Republic of Congo, Rwanda, Burundi, Tanzania, and Kenya (Blomme et al. 2017; Carter et al. 2010; Tripathi and Tripathi 2009).

Like the other two bacterial wilt diseases of banana, Xanthomonas wilt is transmitted by insects that visit the male inflorescence, contaminated tools, and infected planting material. Because affected fruit may not show external symptoms, a high percentage is marketed, but it is not clear whether movement of infected fruit contributes to further spread of this disease (Blomme et al. 2017). Resistance to this pathogen has recently been identified in *M. acuminata* subsp. *zebrina* and in *M. balbisiana* (Nakato et al. 2019) in addition to banana varieties with botanical characteristics, such as dehiscent bracts or absence of the male bell, which may escape from infection.

FIGURE 17

Symptoms of banana Blood disease in Pisang Kepok fruit in Indonesia.

FIGURE 18

Symptoms of Moko disease in Cavendish fruit.

Removal of the male inflorescences at the right stage is an effective way to limit further dissemination in the field.

CONCLUSIONS

The emergence and dissemination of banana diseases has been without precedent over the last century. One major disease, *Fusarium* wilt race 1, resulted in the demise of the banana variety Gros Michel and the rise of Cavendish as the major banana variety grown for export. At present a new *Fusarium* wilt pathogen, *Fusarium* wilt tropical race 4, is spreading rapidly around the world and threatens cultivation of the Cavendish banana. Four major foliar diseases, yellow Sigatoka, black leaf streak, Eumusae leaf spot, and freckle, have spread between continents and significantly extended their geographic ranges, resulting in substantial increases in the cost of production due to the need for regular applications of fungicides. Banana bunchy top disease has spread intercontinentally and requires ongoing surveillance, removal of infected plants, and access to clean planting material to maintain production. Three major bacterial wilt diseases of banana have spread beyond their ancestral homes, causing significant disease losses and increased management costs. Two of these are new encounter diseases (Moko, originating in the Americas, and *Xanthomonas* wilt, originating from Africa), whereas Blood disease is believed to have coevolved with banana in Southeast Asia. Hence, in the span of a century Gäumann's statement that "in publications of tropical plant diseases until about 1910 banana diseases typically did not rate a mention" (Gäumann 1922) has been completely turned around. In fact, over the last century the rapid global spread of these pathogens has resulted in major and costly disease problems in banana, as outlined in this review and several monographs (Jones 2019, 2000; Stover 1972; Wardlaw 1935, 1961).

In his review Stover (1986) suggested strategies for disease control and research to lead the banana industry into the 21st century. This goal has clearly not materialized. In the 1980s black leaf streak control already made up 27% of production costs of Cavendish in Latin America (Stover and Simmonds 1987), and these costs are now >30% and still increasing. The large-scale monoculture plantation systems based on Gros Michel were highly susceptible to *Fusarium* wilt race 1. The land-grabbing strategy to maintain production between 1910 and 1950 caused societal and agricultural instability but kept banana production stable. Eventually, shifting to Cavendish varieties in the 1960s saved the banana export industry from total collapse. The prediction that a tropical race 4 *Fusarium* wilt pathogen in the tropics would introduce a new and similar "devastating era" (Stover 1986) is now upon us. The claim that a new desert banana variety resistant to *Fusarium* wilt TR4 and black leaf streak was urgently needed has not been taken seriously for years. Today a high price has to be paid for not following up on this plea. A new *Fusarium*, *Fusarium* wilt TR4 (*F. odoratissimum*), with the ability to attack Cavendish emerged from the center of origin of banana and has already spread widely in banana producing countries of the world (Maryani et al. 2019). It has been estimated that *Fusarium* wilt TR4 has already destroyed >100,000 ha of bananas in China alone (Drenth 2019). The recent invasion of this pathogen in Africa and Latin America will no doubt underscore the extreme genetic vulnerability of the banana industry relying on a single variety.

Not all banana diseases are present everywhere, so the ability to prevent the spread of plant diseases to new areas and continents is still urgently needed. However, despite all the evidence at hand after a century of invasions there is still a lack of consensus on what actions to take, if any, to prevent invasions and dissemination of banana pathogens. The recent sequence of incursions of *Fusarium* wilt TR4 in various countries and continents can be seen as an example of what ecologists call invasive species denialism (Russell and Blackburn 2017). This denialism in the context of invasive pathogens hampers efforts to eradicate or contain an invasive

pathogen. With regard to bananas, only Australia can claim to use a proactive approach, and there is now an established record of containing the BBTV to a defined region for more than a century, eradicating both black leaf streak (Henderson et al. 2006) and freckle (McMaster et al. 2020), and containing *Fusarium* wilt TR4 in a major banana production area. To achieve this, plant pathologists and industry stakeholders in Australia have continuously raised awareness of the potential impact of such diseases, provided data to estimate the exclusion benefits (Cook et al. 2012, 2013a, 2013b), and provided access to well-trained staff and facilities to quickly make a definitive diagnosis. A positive pathogen diagnosis must be followed by swift action related to eradication or containment, ideally based on prearranged cost sharing agreements to prevent delays due to bureaucratic indecisiveness and arguments about funding arrangements. In addition, it is paramount that industry, research scientists, affected local communities, and government agencies support such efforts to take action in a timely manner after an invasion occurs.

All the fungal, bacterial, and viral pathogens as outlined in Table 1 and discussed in this review have spread significantly over the last few decades, and all but banana Blood disease have spread intercontinentally through anthropogenic action. With the exception of *Fusarium* wilt race 1, Cavendish varieties are susceptible to all eight other major diseases, which makes cultivation of this variety into the future unsustainable. The cost of disease control is increasing, in addition to staggering environmental, health, and food safety concerns related to fungicide use. A familiar pattern is that banana pathogens emerge, mostly in the center of origin, and spread to other continents, generally through human activities. Once a pathogen establishes a bridgehead in a new continent it typically continues to rapidly spread from this initial invasion event to neighboring countries, as illustrated in Figures 2, 5, 8, 9, 11, 12, 14, and 15. The spread of banana diseases outlined in this review underscores that rarely is enough attention paid to initial small outbreaks of emerging diseases. Only at the early stages of an incursion is it possible to eradicate a disease, as demonstrated for black leaf streak and freckle in Australia (Henderson et al. 2006; McMaster et al. 2020).

A problem most plant pathologists face is that instead of focusing on long-term plant health, aimed at disease prevention, many (often externally funded) projects commence with a focus on short-term management strategies for pathogens that currently cause significant disease losses. Although this is important, we also need to direct attention to the prevention, spread, containment, or eradication of emerging diseases. All diseases start small, on a regional basis, and prevention strategies must be in place before the pathogen appears. A lack of leadership, willingness, and ability to act swiftly early on is often a costly mistake in the long run. The recent dissemination of *Fusarium* wilt TR4 to Africa and South America indicates that learnings from the disease epidemic caused by *Fusarium* wilt race 1, costing US\$2.7 billion in today's dollars (Ploetz 2005), have not been implemented effectively. The failure to proactively protect the banana industry from new incursions such as *Fusarium* wilt TR4, known to cause major disease problems on Cavendish plantations elsewhere in the world, will have serious consequences. Coupled with a lack of effective prevention, containment, or eradication methods, in almost all banana growing countries this will be a costly mistake for banana growers and will not only affect Cavendish production but also radiate to many smallholders growing other susceptible banana varieties.

It is of interest to note that most incursions in the banana industry occur on plantations, which makes the growers who spend most time in the field the first line of defense against invading pathogens. Early detection, before the pathogen has had time to spread, is of the utmost importance. These efforts must be supported by an organized framework, infrastructure, and awareness campaigns to prompt action. Contingency plans must be prepared in advance and

regularly updated, because time is of the utmost importance in responding to a pathogen invasion to maximize the window for eradication or containment. Unfortunately, we have also seen that circumstances during an invasion attract businessmen abusing the despair of growers and the lack of preparedness of government by offering ineffective quick fix solutions for sale. Therefore, it is important that contingency plans include strategies for eradication or containment that are rigorously tested, scientifically sound, and effective and can be implemented immediately after an invasion event.

The emergence and spread of plant diseases threatening crops are not unique to banana. In fact, the intercontinental and subsequent local spread of pathogens on almost all crops has become a global phenomenon because of increased travel, trade, and the speed of intercontinental transport (Bebber et al. 2014; Fisher et al. 2012; Savary et al. 2019). Although the threat from exotic pathogen invasions is similar between crops, the main difference lies in how one responds to them. A major difference between bananas and many other crops lies in the willingness, or lack thereof, to invest in long-term strategies to reduce the level of genetic vulnerability through genetic diversification. The initial global *Fusarium* wilt race 1 epidemic was overcome by use of the genetic resistance already present in the existing Cavendish variety. Because plant breeding played no role in this solution, subsequent investment in banana breeding has been low in relation to the size of the problem and the importance of bananas and plantains for economic and food security. Little investment was made in genetics and plant breeding or in preemptive or anticipatory breeding. Compared with many other major crops in the world, banana lags far behind. For example, diseases caused by pathogens such as rusts on cereals, which travel easily, have led to major long-term international cooperation for pathogen characterization, identification of resistance, and preemptive breeding approaches that have been successful (McIntosh 1992).

The history, spread, and impact of disease in banana as outlined in this review and the consequences of the *Fusarium* wilt race 1 epidemic in the last century should mobilize major efforts to identify and characterize resistance against the major plant diseases and to develop resistant, market-acceptable varieties. Instead, the focus of research in bananas has been on extending the lifespan of the highly susceptible Cavendish variety. This strategy focuses on *Fusarium* wilt TR4 and overlooks the demand for genetically based black leaf streak disease control, resulting in large numbers of fungicide applications in the wet tropics. Lack of genetic diversification and a sole focus on Cavendish because of its current economic importance have resulted in today's extreme level of genetic vulnerability and lack of currently available options for genetic diversification in the global banana industry. The underlying economic drivers have pushed the banana industry to a fivefold increase in size compared with the 1960s. But this giant monoculture has feeble legs and is about to fall over, unless appropriate action is taken to turn the fragile banana industry into a forward-looking, sustainable business serving both international and domestic markets. It is time for fundamental change driven by visionary strategies to significantly broaden the genetic base of banana production to improve the crop and its cultivation in a sustainable manner and enhance the livelihoods of all stakeholders.

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