Origins and spread of plant fungal and oomycete disease outbreaks

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Abstract: Infectious disease outbreaks are common in plant, animal, and human populations. However, aside from a few outbreaks that have developed into epidemics and pandemics, the factors that contribute to most outbreaks remain largely unknown. This is especially the case for plant fungal and oomycete disease outbreaks. Because all epidemics and pandemics start as localized outbreaks which subsequently spread to broader geographic regions, to effectively prevent and control plant disease epidemics and pandemics, it's critical to understand the factors that contribute to the initial outbreaks. In this review, the definition and detection of disease outbreaks are introduced and described. This was followed by brief descriptions of the main factors contributing to plant infectious diseases outbreaks, including host plants, pathogens, and their associated environmental factors. One fungal and one oomycete pathogen are then used as examples to provide brief overview of two agricultural host-pathogen systems and to highlight how molecular tools have helped revealing the origins and spread of pathogens causing outbreaks that have resulted in pandemics. With accelerating changes from anthropogenic activities, including climate change, plant disease outbreaks will likely increase. The paper finishes by discussing how we should prepare ourselves to better prevent and manage future outbreaks.

Key words: infectious disease; host plant diversity; pathogen diversity; environmental factor; molecular marker; epidemiology; anthropogenic effect

植物真菌和卵菌病暴发的起源和传播

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摘要：传染病暴发在植物、动物和人群中很常见。除了少数已发展为流行病和大流行病外，在很大程度上大多数传染病暴发的原因仍未知，植物真菌和卵菌病暴发尤其如此。所有流行病和大流行病都是从局部暴发开始，然后蔓延到更广泛的地理区域，因此了解其初始暴发的原因对于有效预防和控制植物病害流行病和大流行病至关重要。该文首先描述疾病暴发的定义和检测，随后简要描述导致植物传染病暴发的主要原因，包括寄主植物、病原体及其相关的环境因素，以一种真菌和一种卵菌病原体为例简要概述宿主病原体系统，并强调分子工具在帮助揭示病原体的起源和传播及其暴发及大流行方面的作用。由于人为活动及气候的加速变化，植物病害暴发的可能性越来越大，最后提出应该如何应对其暴发。

关键词：传染病；寄主植物多样性；病原体多样性；环境因素；分子标记；流行病学；人为影响

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Plants and pathogens have co-existed for a long time (McNew, 1960). Plant microbial pathogens are highly diverse and include viruses, viroids, bacteria, and eukaryotic microbes such as fungi and oomycetes (McNew, 1960; Agrios & Beckerman, 2011; Savary et al., 2019). Savary et al. (2019) estimated that the mean global yield losses due to pathogens and pests annually were 21% for wheat, 30% for rice, 22% for maize, 17% for potato, and 21% for soybean. Among them, fungal and oomycete pathogens are especially difficult to control and together they have caused many disease outbreaks to trees and agricultural crops. Fungal and oomycete diseases such as downy mildew, rice blast, early blight, head blight, stem rust, stem rot, root rot, *Fusarium* wilt, and late blight have devastated major crops such as tomato, rice, wheat, maize, soybean, banana, and potato in many agricultural fields and caused significant economic losses to communities around the world (Fisher et al., 2012; Asibi et al., 2019; FAO et al., 2021). It was predicted that if severe fungal and oomycete disease epidemics occurred to five major agricultural crops (wheat, rice, maize, potato, and soybean) simultaneously, the global food supply would be able to feed less than 40% of the world's population (Fisher et al., 2012). In addition to impacting pre-harvest crop plants, toxigenic fungi (i.e., fungi that produce mycotoxins) can also contaminate food post-harvest which when consumed, can cause serious health effects, ranging from acute poisoning to long-term effect such cancer (Chen et al., 2020; 2021; FAO, 2021).

Compounded with increasing human population size and changes in our consumption pattern, plant pathogens are challenging our agricultural systems' ability to sustainably provide food and other products and services for humans (FAO et al., 2021). Indeed, it is estimated that a growing population and rising incomes will lead to an increase in demand for agricultural products by 50% by 2050 (FAO et al., 2021). Due to their potential for rapid reproduction through sporulation and their abilities to disperse widely across large geographic regions, plant fungal and oomycete pathogens represent a major global threat to food security (Fisher et al., 2012; Corredor-Moreno & Saunders, 2020; Fang et al., 2020). Unfortunately, the highest crop losses and the biggest increases in demands will likely occur in developing regions where infrastructure is often insufficiently prepared to deal with food shortages and distributions (Zhang et al., 2022). Consequently, there is an increasing attention to fungal and oomycete diseases of plants at all levels of governments, from local to regional, national, and the global levels.

Most plants and agricultural crops throughout the world suffer endemic fungal and/or oomycete diseases (McNew, 1960; Agrios & Beckerman, 2011; Hu et al., 2022). Most of the time, the prevalence and severity of these endemic diseases are relatively low, at least in the natural environments where the crop plants originated and/or where the specific crops have been grown for a long time to develop locally adapted genotypes. Globally, the distributions of plant fungal and oomycete pathogens and the diseases that they cause are not uniform among geographic regions (Agrios & Beckerman, 2011) and as such, quarantine measures have been developed in most regions and countries to prevent intentional and unintentional import of plant pathogens by travel and trade (Zhao et al., 2021). When a pathogen disperses from its endemic region to a different region where the pathogen was initially absent, a disease outbreak can occur. In addition, within disease-endemic regions, the causative pathogen, the host plant(s), and/or the environment can all change over time, causing local outbreaks which can spread to neighboring regions and even to national and international levels. Understanding the patterns of disease prevalence and severity over time at various geographic scales can help identify major factors contributing to local disease outbreaks, epidemics, and pandemics, which in turn can help minimize the detrimental effects of such diseases (Corredor-Moreno & Saunders, 2020; Jones, 2021; Hu et al., 2022).

In this manuscript, I review the potential changes that can lead to sudden and significant increases in disease prevalence, with a focus on diseases caused by plant fungal and oomycete pathogens. I start by introducing what outbreaks are defined, classified, and

investigated. This is then followed by describing the major causes of disease outbreaks, including the host plant, the pathogen, and their associated environmental factors. I then summarize two well-known examples of plant fungal and oomycete disease outbreaks with a focus on how genetic markers have helped in tracking pathogen evolution and understanding their genetic changes contributing to those two disease outbreaks. With increasing anthropogenic effects, the frequencies of plant disease outbreaks will likely increase. Vigilant monitoring and associated research will be essential to prepare us for future disease outbreaks.

1 What Is An Outbreak

In human disease investigations, an outbreak is defined as the occurrence in a community or geographic region of cases of an illness with a frequency significantly higher than normally expected. It's commonly recognized that the number of cases indicating presence of an outbreak will vary among diseases due to differences in causative agents, size and type of exposed population, prior exposure (or a lack of exposure) to the disease, and the time frame and place of disease occurrence in the community. Specifically, an outbreak refers to an increased frequency of a defined disease relative to the usual frequency in the same area, among the same population, at the same season of the year (Chin, 2000). Thousands of infectious disease outbreaks have been reported in humans during the last two centuries. The common ones include cholera, Ebola, human immunodeficiency virus (HIV), influenza, malaria, measles, plague, severe acute respiratory syndrome (SARS), salmonella, smallpox, various foodborne outbreaks, and the current coronavirus disease-2019 (COVID-19).

The human disease outbreak definition can be similarly applied to define plant infectious disease outbreaks as sudden increases in disease prevalence, relative to the typical prevalence of the disease in a geographic region for a host plant population over a specific period of time. Plant infectious disease outbreaks may be very localized, limited in both space and time and to a specific host population. Alternatively, outbreaks can persist and become a new norm in the local host population and potentially spread to regional, national, continental, and/or global levels. While less reported and largely obscured from the public view, plant infectious disease outbreaks likely occur at a similar or even higher frequency than those in animals and humans (Fisher et al., 2012; Corredor-Moreno & Saunders 2020; Jones, 2021). Aside from crop plants, infectious disease outbreaks for other organisms can also significantly impact plant productivity and food security, such as those that impact the commercially grown mushrooms (Romaine & Schlagnhaufer, 1989), and pollinators such as honeybees (FAO, 2018) and bats (Forsythe et al., 2021).

Based on the above definition, to formally call a plant infectious disease as an "outbreak", three criteria must be met. Specifically, the host plants must show disease symptom(s) from infection caused by the specific disease agent; the disease and disease agent must be correctly diagnosed; and the disease prevalence must be shown as significantly higher than those from previous years in the same geographic region and in the same host plant species. Failure to meet any of the three criteria could result in a missed or wrong diagnosis of the outbreak. For example, first, if the disease symptom has few or no unique features to distinguish it from other diseases caused by different infectious agents, the disease prevalence may not be properly assessed and described (Ma et al., 2022). Second, there may not be any effective diagnostic tool that's accurate and sensitive enough to detect the causal agent. Often, even if effective diagnostic tools are available to the research community, they may not be easily accessible to the farmers and agronomists at the local level in many parts of the world. Such a barrier could lead to missed or wrong diagnosis and contributing to potentially missing a critical window for effective control and management of the outbreak. Third, if no records had been kept for the specific geographic region over previous years to establish a baseline for comparison, an accurate determination of the change in disease prevalence could not be determined.

Based on the spatial and temporal distributions

of infections, infectious disease outbreaks are commonly classified into two major types: (i) propagated outbreak, and (ii) common source outbreak (Chin, 2000). As the name indicated, a propagated outbreak of a plant infectious disease may have one or few plants in the field to start developing symptoms but overtime, the infection and disease symptoms would spread to adjacent plants, and from these secondarily infected host plants to other plants and so on. In contrast, a common source outbreak refers to infections on many different plants from a common source. One pattern consistent with a common source outbreak is that all plants in a field have disease symptoms all at the same time, e. g., due to contamination of the planted seeds or cuttings and/or the entire field of soil was contaminated by the same pathogen. However, a common source outbreak could also be resulted from one single pathogen source such as one diseased plant that continuously release pathogen propagules to infect other plants in the same and adjacent fields. For many plant diseases, especially those on perennial trees, a disease outbreak may include components of both types where each infected plant (including the originally infected plants, and secondary and tertiary infected ones etc.) can all continuously release pathogen propagules over an extended period of time to infect susceptible host plants. The different types of plant disease outbreaks would show different spatial and temporal patterns of disease symptoms among plants within and between crop fields.

2 Contributors to Disease Outbreaks

The occurrence of infectious disease depends on three interacting factors: the host, the pathogen, and the environments in which the host and the pathogen reside and interact. For a plant infection to occur, there must be a susceptible host plant, a pathogen virulent to the host plant, and an environment that favors the infection and growth of the pathogen inside the host plant. In situations where the diseases are endemic, the host-pathogen relationship at the population level may have reached a relative equilibrium whereby in the specific environment, only a subpopulation of the host plants are susceptible to a specific pathogen infection while the population size of the host plant remains relatively stable over time. For an endemic disease to become an outbreak, changes in at least one of the three factors (i.e., the host plant population, the pathogen population, and/or their environmental conditions) are needed to disrupt the equilibrium. In the sections below, I briefly describe how changes can occur in each of the three factors to contribute to disease outbreaks.

2.1 Host plants

For any infection to occur, a susceptible host must be present in the population. When a much larger number of susceptible hosts than before suddenly become available, an outbreak can occur in the population. The increase in susceptible hosts could happen through multiple ways. For example, in modern agricultural practices, one very common change has been from planting a genetically diverse crop plants to monoculture where only a single genotype of crop plants is grown in one or many adjacent fields (Dita et al., 2018; Jones, 2021; Zhang & Chen, 2022). If a pathogen virulent to the specific host plant genotype is present in the fields, all crop plants in the fields would be susceptible to infection, potentially leading to an outbreak. Even if the monoculture is resistant to one or a few locally adapted pathogen genotypes (but not all of them), an outbreak can still occur. Similarly, if the monoculture host genotype was brought in from outside to a region, one or a few of the endemic pathogen genotypes could cause infections and result in disease outbreaks.

Aside from the genotype(s) of host plant, the susceptibility of host plants to specific pathogens depends on other factors, including the lifestyle of the pathogens (Agrios & Beckerman, 2011). For example, some infectious agents are primary or obligate pathogens such as agents for downy mildews, powdery mildews, and rusts. They depend on specific host plants to reproduce and often, they can infect and cause disease in many individuals within a host plant species. On the other hand, many pathogens are secondary or opportunistic pathogens. They can grow and reproduce outside of their host plants but only infect certain individuals in the population with compromised de-

fense mechanisms. When the susceptible host population increases in frequency, infections by opportunistic pathogens would also increase. For example, in humans, infections by the HIV compromise the host immune system and render the HIV-infected patients highly susceptible to a broad range of mucosal and systemic infections by many fungal species such as *Aspergillus fumigatus*, *Candida albicans*, *Pneumocystis jirovecii*, *Cryptococcus neoformans*, and *Talaromyces* (*Penicillium*) *marneffei*, resulting in many human fungal disease outbreaks across the globe since the 1980s (Fisher et al., 2012). Increases in the proportion of susceptible plants to microbial infections (including fungal and oomycete infections) could be brought about by both intrinsic factors of the plants and extrinsic factors such as insect damages, climate change, and/or anthropogenic influences (Jones & Barbetti, 2012; Jones, 2021; Miedaner & Juroszek, 2021).

2.2 Fungal and oomycete pathogens

Similar to natural host plant populations, fungal (Manawasinghe et al., 2019; Maryani et al., 2019; Zhang & Chen, 2022) and oomycete (Goodwin et al., 1994; Grünwald et al., 2001) pathogen populations are often genetically highly diverse in their native environments. The genetic diversity within populations of a pathogen species often reflects their variations in infectivity, antimicrobial susceptibility, and virulence among pathogen strains, with some strains being more infectious, more resistant to certain antimicrobial agents, and/or more virulent than others to certain host genotypes but less so to different host genotypes (Manawasinghe et al., 2021). A similar phenomenon is frequently observed at above-species level for both pathogens and host plants where some pathogen species have broad host ranges and can infect many species of host plants while others have narrow host ranges and are obligately dependent on certain host species (Agrios & Beckerman, 2011; Newman & Derbyshire, 2020; Ma et al., 2022). Examples of fungal pathogens with broad host ranges include the species/species complexes of *Alternaria alternata*, *Colletotrichum gloeosporioides*, *Fusarium oxysporum*, and *Sclerotinia sclerotiorum* (Li et al., 2016; Fang et al., 2020). For example, *S. sclerotiorum* can cause watery soft-rot diseases in over 400 species in 75 families of plants (Boland & Hall, 1994). On the other hand, necrotrophic fungal pathogens such as *Parastagonospora nodorum*, *Phyllosticta citriasiana*, *Diaporthe citri*, and *Pyrenophora tritici-repentis* cause diseases in a relatively narrow range of host species (Newman & Derbyshire, 2020; Xiong et al., 2021; Zeng et al., 2021). The differences in host range (at both within and above-species levels) and in infectiousness to different host species/genotypes mean that an increase in any susceptible hosts could result in outbreaks. The differences in infectivity and virulence among pathogen strains and in disease susceptibilities among host plants are often related to the genome features of both the pathogens (Adhikari et al., 2013; Yu et al., 2020; Raman et al., 2021) and host plants. Those differences include the type and number of effectors and virulence genes in pathogens, and the genetic defense mechanisms in host plants such as those involved in regulating host plant's productions of reactive oxygen species and antimicrobial compounds etc (Newman & Derbyshire, 2020; Pang et al., 2021; Guo et al., 2022).

Like all organisms, fungal and oomycete pathogen populations are not static but can undergo a diversity of genetic changes such as mutation, asexual recombination (Xu, 2021), sexual recombination (Xiong et al., 2021; Zhang & Chen, 2022), hybridization (Samarasinghe et al., 2020), horizontal gene and horizontal chromosome transfers (Steensels et al., 2021). Such genetic changes can lead to the formation of new species and new genotypes, some of which may have increased virulence, expanded host ranges for both among populations within host species and among host species (Bhattacharya, 2017; Fernandez & Orth, 2018; Lewis et al., 2018). Similarly, host plant populations can also change. However, due to the much shorter generation time and faster rate of reproduction of fungal and oomycete pathogens than their host plants, fungal and oomycete pathogen genotypes capable of causing disease outbreaks will continue to emerge in the future, at a faster rate than the generation of crop plant genotypes (Zhang & Xu, 2018). In addition, due to the ability of most fungal and oomy-

cete pathogens to produce abundant spores that can easily disperse over a range of distances, new pathogen genotypes originated in one place can exert continuous pressure on host plant populations in other places, potentially causing outbreaks(Hu et al., 2022; Ma et al., 2022).

2.3 Environmental factors

Without a conducive environmental condition, a pathogen can't cause disease or disease outbreaks in plant hosts. Environmental factors such as nutrient availability, a permissive temperature, and sufficient water are essential for the growth and reproduction of many pathogens outside of plant hosts (Xu, 2006; Agrios & Beckerman, 2011). In addition, factors such as wind, rain fall, insect pests, and human activities can all impact the dispersals of pathogens from short to long distances (Li et al., 2016; Fang et al., 2020; Zeng et al., 2021). Furthermore, a variety of environmental factors can influence the interactions between pathogens and host plant tissues during pathogenesis such as appressoria formation, pathogen growth within hosts, and host defense (Chethana et al., 2021a, b). Those in situ interaction factors include water availability at site of infection, temperature, antifungal drugs, CO_2 concentrations, biotic and abiotic damages to the plants, and the endospheric and rhizospheric microbiomes of plants etc (Li et al., 2021; Pang et al., 2021). Too much or too little water in the fields, extreme high and low temperatures, crop damages by strong winds and insects, and changes in the normal microbiota in crop fields can all lead to increased susceptibility of host plants to pathogens (Zhu et al., 2010; Velásquez et al., 2018; Li et al., 2021). Furthermore, with global warming and increasing severe weathers due to climate change, pathogens are adapting and expanding their geographic and ecological niches and new infections and outbreaks will likely become increasingly frequent (Jones & Barbetti, 2012; Velásquez et al. 2018; Zhang & Xu, 2018). Lastly, agricultural fungicides are increasingly used in crop fields, especially in greenhouse settings, imposing strong selective pressures on fungal pathogens and facilitating the emergence and spread of drug-resistant fungi (Hwang et al., 2014; Diao et al., 2015; Zhou et al., 2021).

3 Molecular Markers Help Reveal Pathogen Histories and Outbreaks

As described in the above sections, infectious disease outbreaks in plants can be caused by changes in one or more of the three interacting factors: the host, the pathogen, and the environment in which the host and the pathogen reside. In this section, I outline how pathogen genotypes are identified and how such genotype information can be used to track the evolutionary histories of pathogens causing outbreaks.

Since the mid-1960s, a variety of molecular techniques have been developed to analyze the genotypes of microbial pathogens. Those techniques include multilocus enzyme electrophoresis (MLEE), electrophoretic karyotyping (EK), random amplified polymorphic DNA (RAPD), restriction fragment length polymorphism (RFLP), PCR-fingerprinting, amplified fragment length polymorphism (AFLP), multilocus microsatellite typing (MLMT), single copy gene-specific PCR, single gene sequencing, multilocus sequence typing (MLST), matrix-assisted laser desorption/ionization time of flight mass spectrometry (MALDI/TOF), and whole-genome sequencing typing (WGST). The underlying principles as well as the pros and cons for each of these methods for genotyping microbial strains were recently reviewed by Hong et al. (2021). Together, while controversies remain, these strain genotyping techniques have helped reveal a diversity of population genetic and epidemiological patterns and resolved many outstanding issues for plant fungal and oomycete pathogens, including: (i) the broad phylogenetic diversity of plant fungal and oomycete pathogens (Fang et al., 2020; Xu, 2020; Manawasinghe et al., 2021), (ii) the centers of origins for many pathogens as well as their gene flow and dispersal patterns (Goodwin et al., 1994; Goss et al., 2014; Maryani et al., 2019), (iii) the distributions of genetic variation among geographic and ecological populations of individual pathogen species such as *Colletotrichum truncatum* (Diao et al., 2015), *Colletotrichum fructicola* (Li et al., 2016), *Fusarium oxyspo-*

rum (Fang et al., 2020), *Diaporthe citri* (Xiong et al., 2021), and *Phyllosticta citriasiana* (Zeng et al., 2021), (iv) hybridizations and horizontal gene transfers among divergent lineages within species as well as between closely related species (Bhattacharya, 2017; Czislowski et al., 2018; Steensels et al., 2021), and (v) specific genes and genetic mutations impacting pathogen virulence (Fernandez & Orth, 2018; Corredor-Moreno & Saunders, 2020; Raman et al., 2021). Due to the fast mutation rate of microsatellite loci and the broad genome coverage from whole-genome sequencing, MLMT and WGST have emerged as increasingly important molecular markers for studying fungal pathogen epidemiology and evolution. For example, these two types of markers have become the most frequently used for addressing issues related to how crop domestication, agricultural practices, and anthropogenic activities such as trades of agricultural goods may have impacted the evolution of plant fungal and oomycete pathogens (Diao et al., 2015; Raman et al., 2021; Xiong et al., 2021).

4 Tracking the Origins and Dispersal of Outbreaks

Population genetic and molecular epidemiological studies have revealed the patterns of genetic variation for many plant fungal and oomycete pathogens at various spatial and temporal scales. These studies have helped identify the centers of diversity for a number of fungal and oomycete pathogens and revealed how the pathogens have spread from such centers to other geographic regions and ecological niches, causing outbreaks in different populations and species of host plants. To robustly infer the center of pathogen diversity and the origin of an outbreak, extensive sample collections of all affected geographic regions and ecological niches such as soil and different host plant species are needed (Chin, 2000; Xu, 2006; Manawasinghe et al., 2018). The pathogen samples are then analyzed using highly polymorphic genetic markers (such as MLMT, MLST, and WGST) to identify strain genotypes and calculate the frequencies of alleles and multilocus genotypes within individual geographic and/or ecological niche populations. Based on allele and genotype frequencies within and among the sampled populations, and in the case of MLST, the gene genealogical histories, the putative center(s) of pathogen diversity and origin(s) of individual outbreaks can be inferred.

The centers of pathogen diversities typically have several population genetic signatures, including: (i) a high allelic richness, (ii) a high gene diversity, (iii) a high genotypic diversity, (iv) a high proportion of strains capable of sexual reproduction, and/or (v) abundant evidence for linkage equilibrium and sexual recombinant (Goodwin et al., 1994; Xu, 2006; Goss et al. 2014). In contrast, a recently derived population would have a low allelic richness, a low gene diversity, a low genotypic diversity, a small proportion of strains capable of sexual reproduction, and limited to no evidence of recombination. The frequencies of individual alleles and individual genotypes, including rare alleles and multilocus genotype-sharing, are often used to infer the amount of gene flow among geographic and ecological populations. Furthermore, coupled with sampling date information and spatial distribution of pathogen strains and genotypes, the directions and timing of pathogen spread from one host plant species to another, from one geographic region to another, and the potential mechanisms for such spread, could all be inferred (Goodwin et al., 1994; Goss et al., 2014; Maryani et al., 2019).

Below I use two examples, one fungal and one oomycete, to illustrate the complexity of disease outbreaks, how molecular markers have helped in tracking the history of these two outbreaks and pandemics, and what we can learn from them.

4.1 *Fusarium* wilt in banana

Banana (*Musa* spp.) is a major agricultural crop and the biggest traded fruit in the world (FAO, 2021). Banana cultivars can be divided into two major types: the dessert type (e.g., the Gros Michel banana and the Cavendish banana), and the cooking type (e.g., plantains). Bananas are predominantly produced in humid tropical and subtropical regions in Africa, Asia, Latin America, and Oceana. The largest banana producers for domestic consumption are India and China (FAO,

2021). The leading banana export countries are Ecuador and the Philippines while the top three leading import regions/countries are the European Union, the US, and China, in that order (FAO, 2021).

Banana is a perennial herb that was first domesticated about 7 000 to 10 000 years ago. Genetic analyses have revealed that the main center of banana genetic diversity is around Malaysia and Indonesia (Simmonds, 1962). However, the center(s) of banana domestication is still uncertain but likely in South and Southeast Asia (Heslop-Harrison & Schwarzacher, 2007). Similar to many other agricultural crops, commercial trade and human migration have played a major role in banana's widespread cultivation in the world. At present, the most widely grown cultivars have seedless fruits. These seedless cultivars are primarily triploid, derived from crosses between two diploid wild species that produce seeds, *Musa acuminata* and *M. balbisiana* (Heslop-Harrison & Schwarzacher, 2007).

Since the late 1800s, *Fusarium* wilt caused by *Fusarium oxysporum* f. sp. *cubense* (*Foc*) has been a serious threat to the banana industry (Moore et al., 1995; Dita et al., 2018). Results so far have indicated that *Foc* is a highly variable pathogen, comprising at least 24 vegetative compatibility groups belonging to multiple divergent lineages (Ploetz & Correll, 1988; Mostert et al., 2017). Variability in pathogenicity among strains within *Foc* to different banana cultivars has led to its subdivision into three races (1, 2 and 4). One population of *F. oxysporum* causing *Fusarium* wilt in *Heliconia* spp. was initially described as *Foc* race 3, but it is no longer considered as part of *Foc* (Ploetz, 2006). Race 1 is only virulent to the Gros Michel banana, while race 2 is virulent to Bluggoe and other closely related cooking bananas (Moore et al., 1995). However, race 4 is pathogenic to all known cultivated bananas, including the Cavendish banana and those that are susceptible to race 1 and race 2 (Su et al., 1986). Race 4 includes two sub-groups, the tropical group (TR4) and the subtropical groups (STR4), which infects bananas in subtropical climates in South Africa, Australia, Taiwan, and the Canary Islands (Groenewald et al., 2006; Ploetz, 2006). In contrast, TR4 infects bananas in the tropical regions of South Asia, Southeast Asia, the Middle East, South America, and Australia (Groenewald et al., 2006). *Foc* is soilborne and capable of producing spores that can remain viable in soil for a long time. As a result, any field with *Foc* contamination can't be used to grow susceptible cultivars for most farms. During infection, the fungus forms appressoria and penetrates banana roots which subsequently spreads to the vasculature, releases toxins, and causes the plant to secrete gelatinous substances that eventually leads to wilting, collapse, and death of the whole plant (Dita et al., 2018; Raman et al., 2021). Multiple *Fusarium* wilt outbreaks have been reported on banana in different regions throughout the world, collectively resulting in two pandemics, the first from the late 19th century to the mid-20th century and the second from the 1960s to still ongoing at present (Moore et al., 1995; Zheng et al., 2018; Raman et al., 2021).

The first outbreak of *Fusarium* wilt of banana (also called the 'Panama disease') was initially described in Australia in 1874 (Moore et al., 1995; Dita et al., 2018). The pathogen was latter named *Foc* race 1. This outbreak subsequently spread to other geographic regions, causing a pandemic and destroying the well-established Gros Michel banana that was grown in monoculture plantations throughout Asia, Africa, and the Americas until the mid-1900s (Moore et al., 1995). This outbreak and the resulting pandemic facilitated to the development of *Foc* race 1- resistant Cavendish banana. Unfortunately, the *Fusarium* wilt disease re-emerged in Southeast Asia, caused by a new race of *Foc* TR4 which was first reported in Taiwan island in 1967 (Su et al., 1986). However, the disease in Taiwan was suspected to have been introduced from Indonesia through infected plants. Since then, *Foc* TR4 has been reported from various regions in at least 17 countries in Southeast Asia, South Asia, southern and southeastern provinces in China, the Middle East, Africa, and South America (Zheng et al., 2018; Maymon et al., 2020). The South American strains from northern Colombia was named a new species called *F. odoratissimum* (Maryani et al., 2019), though there was a recent rejection to the new

species proposal (Torres Bedoya et al., 2021). For consistency with the majority literature so far, this review refers this group of strains as *Foc* TR4 or *Foc* STR4. Th expanding distribution of *Fusarium* wilt caused by *Foc* TR4 has raised severe concerns among scientists and policy makers throughout the world (FAO, 2021). At present, the Cavendish banana represents an estimated 70% of the world's banana cultivation and all of them are known to be susceptible to *Foc* TR4 infection (Dita et al., 2018). Indeed, no banana cultivar completely resistant to *Foc* TR4 is known and the control measure against the spread of *Foc* TR4 has been largely based on quarantine, sanitation, application of chemical fungicides and biocontrol agents, modifications of soil features, crop rotations, and selecting and planting cultivars that are less susceptible to *Foc* (Dita et al., 2018). Though such measures have been successful in slowing down its spread, *Foc* TR4 and STR4 has been nonetheless steadily expanding its geographic range, threatening the global banana production.

Several studies have examined genetic variations among *Foc* strains from different geographic regions and from different banana cultivars. These results have contributed to an emerging pattern of *Foc* origin and evolution. Based on AFLP analyses of 40 strains from 11 countries, Groenewald et al. (2006) revealed no consistent clustering of strains based on either host banana cultivar or geographic regions. Due to the small sample size from individual regions/countries and the drawbacks of AFLP markers, the authors failed to identify clear signals for center(s) of diversity for the pathogen. However, based on DNA sequence polymorphisms at three genetic loci for a larger sample size (200 isolates) collected from 40 different local banana varieties at 15 provinces on six islands in Indonesia and by comparing them with those from other geographic regions, Maryani et al. (2019) concluded that the center of *Foc* diversity and the origins of the *Foc* races were most likely in Indonesia. Indeed, significant genetic variation of *Foc* strains was found within most individual local banana varieties and within most locations in Indonesia. In contrast, outside of Indonesia, genotyping studies based on vegetative compatibility, PCR fingerprinting, and whole-genome sequencing have revealed that the strains of *Foc* TR4 and *Foc* STR4 from several geographic regions (e.g., the Middle East, the Greater Mekong Region, and north Colombia) were generally less variable, consistent with single introductions to most individual regions (Zheng et al., 2018; Maymon et al., 2020; Raman et al., 2021). Collectively, these results suggest that Indonesia is the most likely center of origin for *Foc*, including *Foc* TR4 (Maryani et al., 2019).

Despite the presence of strong genetic evidence supporting the Indonesian origin of *Fusarium* wilt, it should be noted that further sampling and analyses of *Fusarium* strains from Malaysia and the surrounding Southeast Asian regions are required before a definitive conclusion about the exact locations of where *Foc* R1, *Foc* R2, *Foc* TR4 and *Foc* STR4 originated. Furthermore, as demonstrated based on whole-genome sequence analyses, during clonal expansion of *Foc* TR4 strains, mutations have been accumulating. For example, analyses of the genome-wide single nucleotide polymorphisms separated the global samples of *Foc* TR4 into three predominantly geographic location-based groups: (i) a Middle Eastern clade; (ii) a South and Southeast Asian clade, and (iii) a South American clade (Zheng et al., 2018; Maymon et al., 2020; Raman et al., 2021). Interestingly, whole genome sequence comparisons among strains belonging to *Foc* R1, *Foc* TR4, and *Foc* STR4 in India and elsewhere revealed abundant race-specific genes in these genomes and significant variations in the number of virulence genes and gene families, including those that are secreted in xylem (Czislowski et al., 2018; Raman et al., 2021). Such rapid genetic changes during the spread of *Foc* TR4 strains are significant causes for concerns.

As shown above, changes in both the pathogen and the host plants have contributed to *Fusarium* wilt outbreaks in bananas. The advances in our understanding of the origin(s) and spread of pathogen genotypes based on pathogen genome information have spurred the development of targeted diagnostic markers for specific races and genotypes (Czislowski et al., 2018;

Dita et al., 2018). The markers and our knowledge about the epidemiology have established a solid foundation for critical monitoring of pathogen dispersal among geographic regions and assessment of the effectiveness of various control measures to prevent the spread of *Fusarium* wilt. However, while progresses have been made, significant challenges remain. For example, aside from identifying an abundant genetic diversity among *Foc* strains from Indonesia, Maryani et al. (2019) also revealed other *Fusarium* varieties and species were causing *Fusarium* wilt to many local banana cultivars within Indonesia. Some of these *Fusarium* strains and species could evolve and become the agents of future banana disease outbreaks. Thus, significant effects should be put into understanding these new varieties and species of *Fusarium* to help prevent and control future *Fusarium* wilt outbreaks.

4.2 Potato late blight

Among all the plant oomycete disease outbreaks, the one associated with the Irish potato famine in the mid-1840s is probably the best known. To help understand this disease outbreak better, here I first describe the origin and dispersal history of the host potato plant, *Solanum tuberosum*. Archaeological and genetic evidence suggested that potato was domesticated about 9 000 years ago from a wild potato species native to the Lake Titicaca region of the Andean Plateau in west-central South America (Hawkes, 1990). Following the Spanish arrival in the Americas in 1542, as part of the Colombian Exchange, domesticated potato cultivars were brought from South America to Europe in the late 16th century (Nunn & Qian, 2010). The cultivars subsequently spread from Europe to other continents, with each of the regions containing relatively limited potato genetic diversity (Glendinning, 1983). Potato is now cultivated on all continents except Antarctica, with most productions in relatively cool climates, including temperate regions as well as at high altitudes in tropical and subtropical regions (FAO et al., 2021). Potato is currently ranked as the fourth most important staple food crop in the world (after corn, wheat, and rice) and plays a very important role in ensuring food security in both developing and developed countries (FAO et al., 2021; Li et al., 2021). Indeed, even after 180 years since its occurrence, the 1840s Irish potato famine is still frequently brought up as a reminder about the importance of potato to food security around the world.

The Irish potato famine was caused by the oomycete pathogen *Phytophthora infestans*. Despite much progress on potato breeding as well as sanitation measures and fungicide applications developed specifically against late blight, *P. infestans* still causes potato crop losses worth billions of dollars each year (Savary et al., 2019), including causing many local and regional outbreaks around the world, including in China (Hwang et al., 2014; Li et al., 2021). In addition, aside from potato, *P. infestans* can infect tomato (*S. lycopersicon*) and other species in the plant family Solanaceae (Platt, 1999; Deahl et al., 2006).

Over the years, many population genetic and epidemiological analyses of *P. infestans* have been conducted using a variety of molecular markers, from MLEE to MLST, MLMT, and whole-genome sequencing. In a landmark paper, Goodwin et al. (1994) analyzed over 300 isolates of *P. infestans* collected from 20 countries on five continents using three types of genetic markers: the mating type idiomorph, two allozyme loci, and DNA fingerprinting using a repetitive DNA fragment as probe in Southern hybridization. Their results suggested that a single clonal lineage dominated most geographic populations worldwide and that all the variations among strains within this global clonal lineage could be explained by mitotic recombination and/or by mutation. The low levels of gene diversity in most geographic populations, including those from West Europe, formed a stark contrast to the high-level genetic diversity in the central highlands of Toluca, central Mexico. The results led them to propose the hypothesis that there was an initial migration of *P. infestans* in the early 1840s from Mexico to the United States and that a single strain of the *A1* mating type was transported from the US to Europe, causing the Irish potato famine, and which subsequently also spread to many regions around the world. Later analyses largely supported their hypothesis but revealed additional regions with high levels of population genetic diversity for *P. infestans* in Central

America and parts of South America, including evidence for the presence of both mating types (required for sexual mating and recombination), high allelic richness, and significant allelic sequence divergence at multiple loci in the genome (Deahl et al., 2006; Goss et al., 2014).

Though the original population of *P. infestans* causing the Irish potato famine belonged to a single clonal lineage, recent and ongoing localized outbreaks of potato late blight have been found to contain mixtures of strains with different genetic ancestries (Goodwin et al., 1994; Kalischuk et al., 2012; Hwang et al., 2014). For example, recent outbreaks in Canada and the US were found caused by pathogen populations containing both mating types A1 and A2, with the A2 mating type migrated out of Mexico more recently than that of the A1 mating type (in the early 1980s vs. the early 1840s) (Deahl et al., 2006; Kalischuk et al., 2012; Hwang et al., 2014). Anthropogenic activities were again likely responsible for the dispersal of the A2 mating type to many parts of the US and Canada. The co-existence of both mating types in proximity can result in sexual mating and recombination, contributing to increasing genetic diversity of the outbreak pathogen populations in the US and Canada.

Together, analyses of the late blight outbreaks from both the 1840s in Ireland and the ongoing ones in North America and China illustrate the important roles of pathogen dispersal, mode of reproduction (both clonal expansion of specific pathogen genotypes and recombination), host plant population history, and human activities in plant disease outbreaks. Similar to what were found for *Fusarium* wilt in bananas, advances in our understanding of its genome and the origin and spread of *P. infestans* genotypes have also spurred the development of rapid diagnostic markers for this pathogen (Adhikari et al., 2013; Hwang et al., 2014; Ristaino et al., 2020). The markers and our knowledge about its epidemiology are allowing critical monitoring of disease dispersals among geographic regions and host species, including helping assess the effectiveness of various control measures to prevent late blight outbreaks.

5 Continuing Challenges and Potential Strategies

Changes in one or more of the three interacting factors (the pathogen, the host, and their interacting environments) can result in disease outbreaks and potentially cause widespread epidemics and pandemics. Unfortunately, such changes are happening and accelerating for all three factors. For example, the movements of crop plants from their domestication centers to distant regions are accelerating. Such movements would expose the crop plants to novel pathogens that the crop plants had never been exposed to before. Similarly, such crop plant movements could also bring pathogens from the center of domestication to new locations and impacting crops at these new locations, including potentially novel susceptible hosts. These movements are facilitated by rapid expansion of international trade in plants and plant products due to (i) trade globalization such as free trade or tariff reductions, (ii) more efficient transport by air, land, and sea, and (iii) increasingly relaxed regulations of plant quarantine measures to meet international trade agreement rules (IMF, 2000). For example, large conglomerates and multinational seed companies often produce seed crops in distant regions and countries with warmer climates at times of year when they cannot be grown in regions with cooler climates. Coupled with increasing monoculture for many crops and extreme weather conditions due to climate change, disease outbreaks will likely increase and become more difficult to predict and manage (Pedroncelli et al., 2019; Corredor-Moreno & Saunders, 2020; FAO et al., 2021).

Despite their economic importance, compared to the significant monitoring efforts for infectious diseases in animals and humans, relatively limited resources have been put into monitoring plant infectious diseases and disease outbreaks. Indeed, so far, most plant disease outbreaks were recognized only after the diseases have become widespread, resulting in epidemics and even pandemics (Bhattacharya, 2017; Asibi et al., 2019). At present, while many reports of plant infectious diseases can be found in the literature,

most such reports are in the form of "case report" and "first report" (Chen et al., 2021; Cui et al., 2021; Fang et al., 2021), missing one or a few key pieces of information for evaluations as potential outbreaks. In addition, to become acceptable for publication in scientific journals as "case report" and "first report", the disease must be caused either by a pathogen species not reported previously for the specific host plant species or by a known disease (i. e., a known host-pathogen system) but in a new geographic area (usually a country not previously reported). Rarely is information about the prevalence of the specific disease over specific spatial and temporal boundaries and how the prevalence compares with previous years available in such reports. Furthermore, most "case report" and "first report" publications became public at least one or several years after such diseases had occurred in the field. Consequently, the true prevalence of plant infectious disease outbreaks is not known and the usefulness of such reports for outbreak control and prevention is very limited. To enhance their values, a real-time disease monitoring network integrating molecular markers such as the ones currently being used for monitoring COVID-19 should be developed for plant disease surveillance. The molecular markers could be used to target specific fungal (Cheng et al., 2020; Ma et al., 2022; Zhang & Chen, 2022) or oomycete pathogens (Ristaino et al., 2020; Wang et al., 2020; 2021). Importantly, technologies such as remote sensing (RS), geographic information system (GIS), global positional system (GPS), and artificial intelligence (AI) are increasingly used for monitoring plant diseases and pests (Hu et al., 2022; Zhou et al., 2022). Such technologies and datasets will help identify the spatial and temporal patterns of disease prevalence at various geographic scales, revealing outbreaks as they happen in real-time, and enabling quick decision-making to deploy control measures to prevent potential epidemics and pandemics of such diseases.

For most major agricultural crops, one to several methods have been developed to help control fungal and oomycete infectious diseases. Those include the use of chemicals and agricultural fungicides, application of biocontrol agents, growing multiple cultivars with different resistance genes in the same field, removal of crop residues (especially the diseased ones) from crop fields, rotation of crops, minimizing seed contaminations, and limiting pathogen spread from one growth season to the next by avoiding double cropping etc. Unfortunately, resistances to agricultural fungicides have been increasingly reported among plant fungal and oomycete pathogens (Song et al., 2021).

Ultimately, to prevent and control disease outbreaks, we need comprehensive understandings of the global pathogen populations, the host plant populations including their susceptibilities to infections by various pathogens, and the broad environmental conditions that impact the growth, reproduction, dispersal, and transmission of individual pathogens. For fungal and oomycete pathogens, such understandings require continued surveys of the prevalence of diseases that they cause and of the spatial and temporal patterns of pathogen population genetic variations. Similarly, understanding the host plant genetic diversity and their disease susceptibility profiles could help guide the development of a series of pathogen-specific resistance cultivars (Huang et al., 2021). In addition, information about the susceptibilities of plant fungal and oomycete pathogens to agricultural fungicides should be integrated into management databases. Together with RS, GIS, GPS, and AI technologies, such data could help in developing realistic epidemiological models for each disease (or several diseases together) and forecasting the potential future distributions of the diseases and pathogen genotypes. Targeted monitoring for specific genotypes in individual jurisdictions and corresponding control strategies could then be more efficiently deployed. With real-time monitoring of disease prevalence using fast and accurate diagnostic markers in field conditions, plant disease outbreaks could be more readily detected and more efficiently managed.

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