Check for updates

Threats to global food security from emerging fungal and oomycete crop pathogens

Helen N. Fones [1] ✉, Daniel P. Bebber [1], Thomas M. Chaloner[1], William T. Kay [1], Gero Steinberg[1,2] and Sarah J. Gurr [1,2] ✉

Emerging fungal and oomycete pathogens infect staple calorie crops and economically important commodity crops, thereby posing a significant risk to global food security. Our current agricultural systems — with emphasis on intensive monoculture practices — and globalized markets drive the emergence and spread of new pathogens and problematic traits, such as fungicide resistance. Climate change further promotes the emergence of pathogens on new crops and in new places. Here we review the factors affecting the introduction and spread of pathogens and current disease control strategies, illustrating these with the historic example of the Irish potato famine and contemporary examples of soybean rust, wheat blast and blotch, banana wilt and cassava root rot. Our Review looks to the future, summarizing what we see as the main challenges and knowledge gaps, and highlighting the direction that research must take to face the challenge of emerging crop pathogens.

Emerging pathogens can be defined as those that cause a new disease, greatly increase incidence of a disease, introduce a disease to a new geographical location or infect a new host[1–3]. There are a number of ways in which diseases emerge; the pathogen can be introduced to a new area where its host is present by physical transportation[1] or it may be introduced more gradually through the development of newly permissive abiotic factors, such as climate[4] or atmospheric composition[5,6]. Conversely, the host may be introduced to an area where the pathogen is already present. A pathogen can also acquire genes through mutation, hybridization or horizontal gene transfer. These genes may confer virulence, enabling infection of a new host, or resistance to disease control measures[3,7,8]. The spread and establishment of pathogens to and within new locations can also be affected by changes in land use and related landscape-scale ecological factors, such as connectivity. These factors in turn are often dependent on policy decisions that affect choices in cultural practice[9]. Finally, a range of biotic and abiotic factors might combine to facilitate a step-change in the incidence of disease[3,4]. Emerging pathogens affecting cultivated plants can be identified from all groups of pests and pathogens but, of these, fungi and oomycetes cause the most crop devastation[2].

Examples of emerging pathogens threatening staple and commodity crops

Examples of crop decimation include the Irish potato famine and the ongoing problem of Panama disease in banana crops. The Irish potato famine led to the death of around one million people and the displacement of a similar number[10]. The pathogen responsible for late blight of potato, *Phytophthora infestans*[11], spread from its centre of origin in Mexico through North America and then to Europe in 1845 (refs. [12–14]). The introduction of *P. infestans* to Europe in 1845 fits the definition of an emerging pathogen: although its host, the potato, was introduced to the region 300 years previously, Europe was a new geographical location for the pathogen. The potatoes grown in Ireland at the time carried no resistance (R) genes against the emerging strain of *P. infestans*[9]. That strain was a single genotype of *P. infestans*[14,15] but today multiple strains are seen worldwide,

having been recognized in Europe, North America, China and Australia. Severe epidemics have been reported where incidence correlates not with weather, but with instances of high genetic diversity in the local pathogen population — this oomycete is once again an emerging (or re-emerging) pathogen[16].

P. infestans can spread clonally as it did in Ireland in 1845 (ref. [17]), and to this day it exists as a highly successful clonal pathogen in regions such as India[16]. However, asexual spread relies on the presence of the host crop. The asexual spores are short-lived and cannot survive between growing seasons, except on infected tissues such as stored tubers or volunteer plants. By contrast, the sexually produced oospores survive much longer[18]. Further, increased diversity due to sexual reproduction endows the *P. infestans* population with significant evolutionary potential, making the breakdown of host resistance or the development of fungicide resistance more likely. In Northern Europe, up to 75% of isolates may be unique due to high levels of outcrossing. Both mating types are also established in North America and Asia, although there is little evidence of any but ephemeral sexual populations in the United States, where the parental clones appear to outcompete most of their offspring while occasionally generating problematic new strains via crosses[16,19]. In China, which now dominates global potato production, a clonal lineage of *P. infestans* from Russia triggered the spread of the disease. However, as in Europe, populations of both mating types quickly became established[16].

Diseases such as potato blight are most serious when the crop affected is a staple food on which a population relies, as was the case in the Irish potato famine. When considering the potential impact of emerging crop pathogens on food security, a key concern is that, worldwide, we are heavily dependent on a limited number of crops for calories[20,21]. Defining which are the 'most important' crops is not, however, entirely straightforward. Rice production ranks highest in terms of provision of calories per capita global population per day, followed by wheat, sugarcane, maize, soybean and potatoes[22]. Global yield data indicate that these five crops are also grown in the greatest quantities, although the rank order is shifted when considering tonnes of yield, with sugarcane and maize occupying the

¹Biosciences, University of Exeter, Exeter, UK. ²Department of Biosciences, Utrecht University, Utrecht, the Netherlands. ✉e-mail: h.n.eyles@exeter.ac.uk; s.j.gurr@exeter.ac.uk

Table 1 | Major global calorie and commodity crops

Region	Global	Africa	North America	Central and South America	Asia	Europe	Oceania
Crop rank							
1	Rice[a,b]	Cassava[b]	Maize[a]	Soybean[a,b]	Rice[a,b]	Wheat[a,b]	Wheat[a,b]
2	Wheat[a,b]	Banana and plantain[b]	Soybean[a,b]	Sugarcane[a]	Wheat[a,b]	Grape	Cotton
3	Sugarcane	Yam	Tomato	Maize[a]	Maize[a]	Potato[a,b]	Barley
4	Maize[a]	Rice[a,b]	Almond	Coffee	Sugarcane[a]	Maize[a]	Grape
5	Soybean[a]	Wheat[a,b]	Wheat[a,b]	Banana[b]	Potato[a,b]	Tomato	Rapeseed
6	Potato[b]	Tomato	Cotton	Rice[a]	Tomato	Olive	Sugarcane[a]
7	Palm oil[a]	Date	Rapeseed	Grape	Cotton	Barley	Apple

For each global sub-region, the highest value crops are shown (ranked using USD per annum). Those not among the top global calorie crops can be considered the most important commodity crops for each region. [a]Top global calorie crops. [b]Crops affected by emerging fungal pathogens discussed in this Review.

top two positions. One of the reasons for this difference is that not all crop yields contribute calories equally to the human diet. For example, some crops are grown as animal feed, and others as biofuels or fibre products. Cassidy et al.[23] reported that if crop contributions to animal feed as well as food is considered, then the top five crops by calories eaten are maize, wheat, rice, oil palm and soybean. Of these, wheat, rice and soybean are currently under threat from newly emerged fungal pathogens and will be discussed further.

Food security, however, is not achieved solely by growing sufficient staple crops to produce the required calories per capita global population. Large portions of the world's population are dependent on global trade and economic systems to access food grown by others. As a result, food security at a national level can be dependent on economic productivity that is generated, for example, by commodity or cash crop production. Sale and export of commodities is therefore pivotal for the food security of many communities. Defining the most important commodity crops is far more challenging than defining key calorie crops, as this will vary according to trade agreements, global economic factors and the intersection of these factors with local economies[24]. As such, consideration of the most important or highest value commodities on a global scale is not conceptually consistent. Instead, we examined the data of the Food and Agriculture Organization of the United Nations (FAO) on the value of crops on a per-continent basis (in USD; data from 2016). Excluding crops already identified as important staples, cassava and bananas/plantains are the top commodity crops in Africa; coffee and bananas in Central and South America; tomatoes and cotton in Asia; grapes and tomatoes in Europe; barley and grapes in Oceania and tomatoes and almonds in North America (Table 1). Many of these global commodity crops are also threatened by emerging pathogens.

Fusarium wilt of banana, also known as Panama disease, is both a historical example of the power of emerging fungal pathogens to devastate crops and a highly topical threat to a modern globally traded commodity. Caused by the fungus *Fusarium oxysporum* f. sp. *cubense*, the disease is likely to have originated in Southeast Asia but was first reported in Australia in the 1870s (ref. [25]). The disease spread globally during the early twentieth century[26], hastened by several conspiring factors. First, a single banana cultivar, Gros Michel, was cultivated worldwide for its export properties[26]. Second, as sterile triploids[27], bananas are propagated via rhizomes and it has been suggested that symptomless rhizomes or their associated soil may carry infection to new banana plantations. Third, *F. oxysporum* f. sp. *cubense* produces resilient chlamydospores that can survive in soil for up to 30 years (ref. [3]), meaning that infected land cannot be used to re-plant bananas. Together, these factors led to the eradication of Gros Michel as a commercial crop and threatened

the availability of bananas for export. This jeopardized those livelihoods reliant on banana sales[28]. Although a new banana cultivar, Cavendish, provided a reprieve for exporters, it is also grown as a global monoculture and today is threatened by a recently emerged variant of *F. oxysporum* f. sp. *cubense* known as tropical race 4 (TR4). TR4 is highly virulent towards Cavendish and has been spreading inexorably through the world's banana-growing regions in recent years, entering South America in 2019 and forcing Colombia, one of the world's top five largest exporters of banana, to declare a national state of emergency[29].

Agroecosystems as cradles for crop pathogen emergence

The twentieth-century Green Revolution increased global agriculture productivity[30], achieved through the introduction of chemical fertilizers and pesticides alongside dwarf and disease-resistant crop cultivars. Gains in yields have maximized global production and profits[31] and helped to rebalance the Malthusian equation in humanity's favour by feeding a growing population[32,33]. However, intensive agriculture has contributed to losses in biodiversity, and increased water and fossil fuel dependency[34]. Nitrogen inputs degrade fragile ecosystems and add nitrogenous gases to an atmosphere already burdened by greenhouse gas emissions[5]. In our current agroecosystems, crop disease protection is predominantly afforded by single-target-site antifungals and cultivars carrying single major R genes. Such protection is proving ephemeral: monocultures provoke the emergence of new fungal strains that are resistant to widely used fungicides and able to overcome host resistance, providing ideal feeding and breeding grounds for crop pathogens[2].

Wheat production exemplifies this situation — vast areas are planted with one of a few 'elite' varieties, with high agricultural inputs facilitating tight planting densities. Temperate-grown wheat is vulnerable to infection by many pathogens. The most serious of these, Septoria tritici blotch (STB), costs UK growers alone around €240 million per year in yield losses[35]. STB is caused by the fungus *Zymoseptoria tritici*. Infections are thought to begin with wind-blown sexual spores. When a *Z. tritici* population is established, however, polycyclic asexual sporulation occurs. The disease can spread rapidly within the leaf canopy, reaching 10^{10}–10^{11} spores per hectare over a growing season[35]. The fast spread of isolates through a field means local variation increases rapidly[36], and most plants/fields are infected with multiple strains[37]. In the field, *Z. tritici* undergoes regular sexual reproduction[38], allowing for high rates of recombination[39]. Mutation rates are also high while the loss of alleles through drift is low[40,41]. Moreover, isolates of this fungus carry a complement of essential chromosomes and up to eight 'accessory' chromosomes that are easily lost and gained[39,42,43]. Fungal populations are also maintained by year-round presence of host
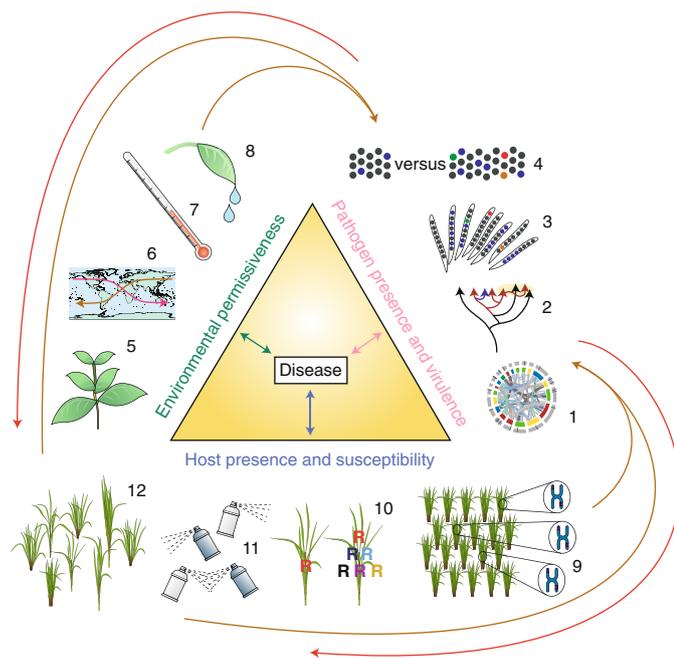
Fig. 1 | Anthropogenic effects that impact the disease triangle. Many factors affect the three facets of the disease triangle, and many of these are themselves influenced by agricultural practices and systems. In particular, pathogen presence and virulence are affected by factors endogenous to the pathogen and variables associated with environmental permissiveness and host susceptibility (see Fig. 4). Endogenous pathogen features include genome structure (1), reproductive systems and capacity for horizontal gene transfer (2), genetic variation within the population (3) and effective population size (4). These combine to determine the pathogen's evolutionary potential. Of the features, population size and variation are most obviously influenced by factors on the other sides of the triangle (yellow arrows). The availability of alternative hosts (5) and the ability to travel (6) by either natural or anthropogenic means are important in determining whether a pathogen is present and how large its population is. Climate factors, such as temperature (7) or humidity (8), can also affect population size, reproduction speed and mutation rates. Meanwhile, large genetically uniform crop monocultures (9) also affect the pathogen's population size and genetic variability. Agricultural practices such as choosing single or pyramids of R genes (10) in crop cultivars, use of single-target-site fungicides or fungicide mixtures (11) or growing cultivar mixes (12) also feed into pathogen evolution. In turn, these factors must be adapted by growers to respond to changing pathogen virulence or resistance to control measures (red arrows). Thus, we see an arms race between man and pathogen.

tissue, particularly in temperate zones[44,45]. *Z. tritici* thus displays all the hallmarks of high evolutionary potential[2]: large effective populations, frequent sexual reproduction, high variability and high recombination and mutation rates. This facilitates the emergence of new traits that allow *Z. tritici* to thrive on new host varieties, under new climatic conditions or in the face of heavy fungicide usage[37,46]. Single-target-site fungicides[47] are currently the major method of control for *Z. tritici*, accounting for around 70% of the European fungicide market[48]. This provides strong selection for resistance — a clear example of the tension between methods used in modern agriculture and the evolutionary pressures they create for new pathogen emergence. This tension provokes an arms race between humanity's crop protection interventions and the emergence of new pathogen variants, rather than between pathogen and host (Fig. 1).

Soybean is also extensively grown in monoculture. Similar to wheat, soybean yields are challenged by a plethora of pests and pathogens. Among these, soybean rust (SBR), caused by the fungus *Phakopsora pachyrhizi*, is the most destructive. Losses of up to 80% are common where conditions are disease conducive[49,50]. As with *Z. tritici*, the severity and spread of SBR can partially be attributed to the fact that the asexual spores are produced prolifically, causing repeated infection cycles[3]. Similar to wheat, soybean is grown at high planting density, which promotes such polycyclic infection by reducing the need for spore dispersal[3].

Fungicide use has provided the mainstay of SBR disease control. This strategy is expensive[51] and can be short-lived as pathogen spread and proliferation, favoured by changing climatic conditions, provoke emergence of resistant or tolerant strains[50]. Thus, STB and SBR show similarities. Both are fast-cycling polycyclic pathogens, infecting the host many times per growing season, and each maintains large populations with high genetic variability. Both threaten to break down control strategies through the enormous evolutionary potential facilitated by monoculture cropping systems.

A different and perhaps more dramatic example of modern-day crop pathogen emergence is the case of wheat blast. This disease is caused by a wheat-specific lineage of the fungus *Pyricularia oryzae*, known as *Pyricularia oryzae* pathotype Triticum, which is closely related to the pathotype *oryzae*, the cause of rice blast[52,53]. This disease causes enormous crop losses[54] and has a history of host hopping, having probably moved from small-grain millet to rice at the dawn of agriculture[55]. Wheat blast originated in 1985 in Brazil, the largest rice-producing country outside of Asia[56]. In Brazil, rice is grown in monoculture and is subject to severe blast epidemics. Sexual reproduction, an ancestral trait, is often lost during clonal spread of *P. oryzae*[57]. Both mating types are present in Brazil, leading to speculation that sexual reproduction may occur among strains, promoted by conditions similar to those at the pathogen's centre of origin[57]. If sexual reproduction is present as well as other mechanisms of genetic exchange, such as parasexuality, it may have facilitated the frequent 'host shifts' that have allowed *P. oryzae* to infect new, supposedly resistant, rice cultivars[58]. There is evidence that wheat-infecting lineages of *P. oryzae* have arisen more than once; an isolate found to be infecting wheat in the United States in 2011 is thought to have arisen from an isolate pathogenic on perennial ryegrass, and is separate from the Brazilian isolates[53]. It has been demonstrated that the jump onto wheat occurred separately in *P. oryzae* lineages that are pathogenic on ryegrass and oats as a result of the transfer of non-functional alleles of avirulence genes *PWT3* and *PWT4*, which are recognized by wheat R genes[59]. The transfer of this virulence-conferring allele evidences historical gene flow between the *P. oryzae* lineages infecting crops and those pathogenic on invasive grass and pasture grass species[56,60]. Cultivation of wheat varieties lacking the cognate R gene for *PWT3* and *PWT4* appears to have allowed *P. oryzae* populations to become established on wheat, while co-cultivation of wheat carrying those R genes provided selection pressure for the non-functional alleles[59]. The proximity of wheat crops to wild hosts for *P. oryzae* therefore appears to have played a key role in the emergence of the new pathogen. As with STB and SBR, high evolutionary potential, achieved through large anthropogenic pathogen populations, can be said to underpin the emergence of *P. oryzae* on new rice cultivars and new host grasses and cereals.

Trade and transport as drivers of pathogen emergence

The spread of wheat blast to the wheat-producing areas of Asia, particularly Bangladesh, has been well-documented. Here, wheat blast has caused crop losses of up to 50% (refs. [61,62]). Such dramatic damage is a common feature of pathogen emergence in a new geographical area, with historical and current examples easy to find among both natural and agricultural plants. Examples include chestnut blight, Dutch Elm disease and ash dieback, which each destroyed >90% of the host species in the locations to which they
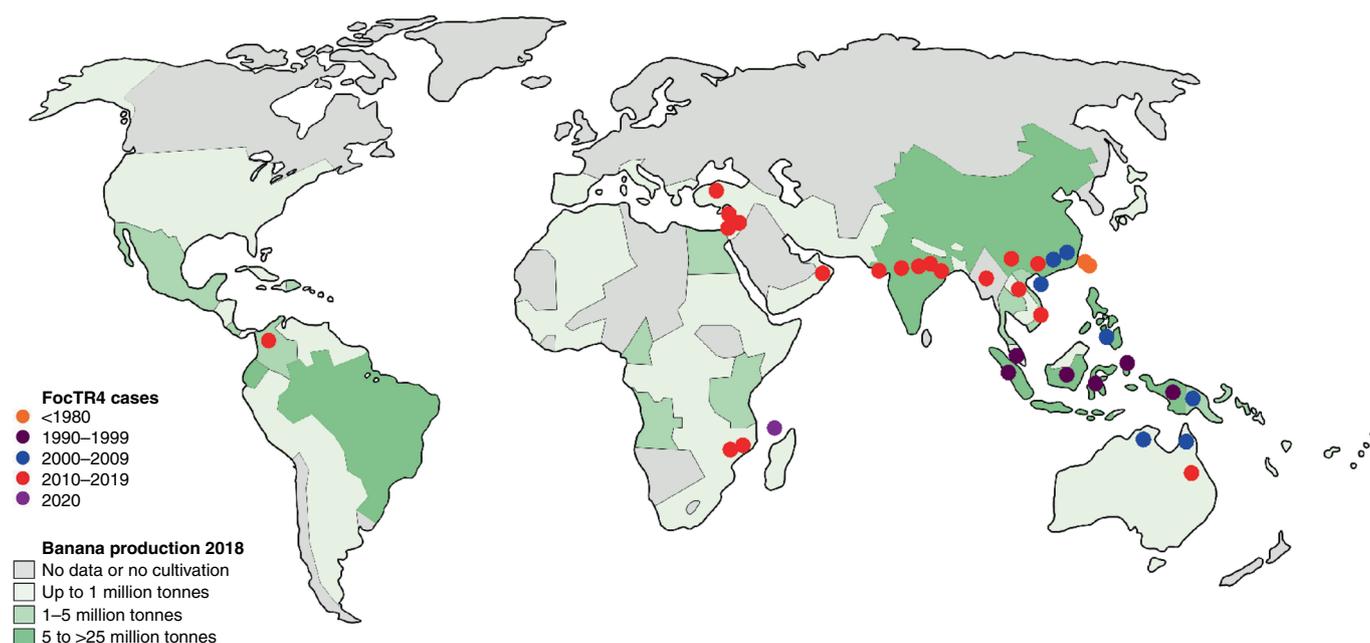
Fig. 2 | Spread of *F. oxysporum* f. sp. *cubense* TR4 throughout global banana-growing regions. TR4 was first reported in East Asia, followed by Southeast Asia and Australia, moving westwards to arrive in South America in 2019 and thus spreading throughout the world's Cavendish-banana-growing regions. TR4 cases, abstracted from refs. [80-91], are colour coded by decade and overlaid onto FAO banana production data[22].

spread[3]. Such severe epidemics are often connected to the fact that the pathogen has moved: new locations may be free from competitors and/or viruses that affect the pathogen itself. They may have a more suitable climate, allowing longer periods of peak pathogen virulence or better survival between host growing seasons. Most importantly, however, new geographical locations have naive hosts that have not co-evolved with the pathogen, and that do not recognize cues to mount an anti-pathogen response. The chance of host naivety is greatly increased when the rate of pathogen migration is accelerated beyond the rate at which host defence responses evolve. This can happen naturally with some pathogens travelling rapidly and over long distances in the atmosphere, as seen in *P. pachyrhizi*[49]. The most common method of rapid pathogen relocation is, however, human activity[3].

Host plants and plant products are traded around the globe, often transporting the associated pathogens. Despite the clear messages of history, humans continue to repeat the same errors and to contribute to global pathogen pollution — Dutch elm disease, for example, was spread worldwide through log trade. The UK spread of ash dieback on saplings imported from the Netherlands was not prevented by lessons learned from Dutch elm disease[63,64]. Both this failing and subsequent attempts to control ash dieback and determine best practice in managing infected woodlands exposed the importance of policy and cultural practice in the control and understanding of emerging diseases. The current response to, and management of, ash dieback is predicated on the results of large-scale citizen-science-based surveys (for example, AshTag[65]). This uses social science approaches to engage with diverse stakeholders (including foresters, conservationists, urban developers and seedling nurseries, among others) and to develop pragmatic policy approaches combining public safety, disease damage limitation and the retention of appropriately chosen potentially resistant trees[66,67]. Similar collaborative approaches are likely to be beneficial in understanding and containing other emerging pathogens[68].

Anthropogenic pathogen pollution

Anthropogenic pathogen pollution is responsible for pathogen emergence by facilitating their introduction to conducive growing conditions and naive hosts, as well as their escape from natural enemies. Arguably, however, the next step is even more concerning: introduced pathogens often exchange genetic material with local relatives, including those adapted to local hosts. This is facilitated by the breakdown of allopatric barriers to gene flow and is common via horizontal gene transfer processes, such as the exchange of dispensable chromosomes[69]. This means that new aggressive pathogens can emerge rapidly from non-virulent isolates introduced into a new location.

Cassava (*Manihot esculenta*), as an important commodity crop in India, China and Southern America, illustrates the role of trade and transport in the emergence of crop pathogens. It is grown both as a local food crop and for bioethanol production[70]. Cassava is propagated vegetatively using stem cuttings, for which the exchange between growers carries the risk of transmitting diseases to new locations[71]. Of particular note here is transmission of the oomycete *Phytophthora palmivora*, the cause of tuber rot. This pathogen renders the crop worthless as tubers become brown, water-soaked and foul-smelling[72]. However, early infection of the stem is asymptomatic and thus the crop appears to be healthy, and infected cuttings may be exchanged or traded between growers. *Phytophthora* spp. were recognized as the cause of cassava disease in Africa in the 1950s (ref. [73]), but *P. palmivora* was first noted as the causal agent of cassava tuber rot in India's Tamil Nadu region in 1997 (ref. [74]). The pathogen subsequently spread through Tamil Nadu and Kerala, where it appeared as an emerging aggressive pathogen and was responsible for up to 70% yield losses[71]. *P. palmivora* cassava tuber rot was identified in China in 2010, attesting to its ongoing global expansion[70]. This worldwide spread has probably been facilitated by the fact that *P. palmivora* has a broad host range and is capable of infecting tropical crop species such as cocoa, pineapple, coconut, rubber and durian, among others[75]. Such host generalism heightens
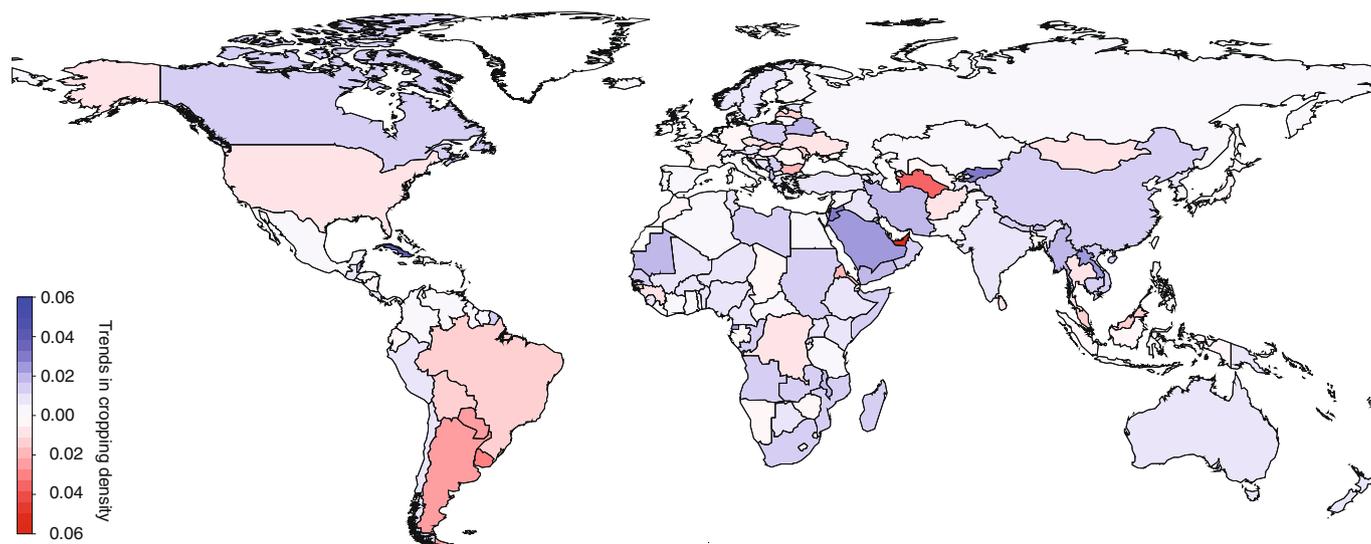
Fig. 3 | National changes in cropping diversity. Changes in Shannon's diversity index (*H*) for 108 crops in 202 global countries and territories from 1980–2017. Colours indicate the trend in *H* per year in each country as either increasing (blue) or decreasing (red) over the analysed period. Harvested crop areas were scaled by the maximum area for a crop in a particular country across all years, to remove the effects of variation in planting area across countries. *H* was then estimated for each individual year, allowing us to compare trends in cropping diversity within countries across years. Cropping diversity increased for 70% of countries from 1980 onwards (median trend: 0.0048 yr^{-1}; interquartile range: −0.00085–0.01006 yr^{-1}), indicating increases in the variety of crops being grown and the evenness of their relative abundances within countries. We found no statistical correlations between trends in cropping diversity and wealth (correlation with log-transformed per capita gross domestic product; Pearson's $r = -0.07$; d.f. = 200; $P = 0.31$) or production levels (correlation with total crop production quantity; Pearson's $r = -0.026$; d.f. = 198; $P = 0.71$).

Table 2 | Greatest gains and losses in crop production area

Losses			Gains		
Country	**Crop**	**Area (Mha)**	**Country**	**Crop**	**Area (Mha)**
India	Sorghum	−9.48	Brazil	Soybean	19.3
USA	Wheat	−8.16	China	Maize	18.99
India	Millet	−6.83	Argentina	Soybean	15.71
China	Wheat	−4.68	India	Soybean	9.66
Canada	Wheat	−3.84	USA	Soybean	7.01
Brazil	Rice	−3.30	USA	Maize	6.95
China	Sweet potato	−3.27	India	Wheat	6.83
USA	Sorghum	−2.82	Brazil	Sugarcane	6.40
USA	Oat	−2.81	Canada	Rapeseed	5.53

Changes in harvested crop areas were calculated for 108 crops in 202 global countries and territories from 1980–2017 using data from the FAOSTAT database. The crops with the greatest national changes in production area over this period are shown.

the risk of the pathogen moving to new areas. Indeed, *P. palmivora* as the causal agent of bud rot, for example, is also a serious and emerging threat to oil palm in Colombia[76].

This generalist strategy is also seen in the cassava pathogen *Macrophomina phaseolina*. The fungus causes charcoal rot disease on over 500 host species. It was first recognized as a globally pervasive pathogen in the 1990s, but difficulties in its identification mean that its presence probably predates the first reports of the pathogen[3,77]. It has recently become increasingly problematic on soybean crops in the USA[77]. While this pathogen emerged as a threat to cassava production in Benin and Nigeria in the late 1990s (ref. [78]), charcoal rot disease was reported on cassava for the first time in Brazil in 2017 (ref. [79]). The risk of spread to new geographical areas by trade and transport is greatly increased by the generalist nature of *Macrophomina*, which infects multiple wild species in addition

to its many crop hosts, and is an opportunist pathogen of animals. Further, such wide host ranges reduce the efficacy of international phytosanitary controls on trade.

Specialist pathogens can also be spread anthropogenically. Wheat blast, for example, is believed to have entered Bangladesh in seeds imported from Brazil[68]. *F. oxysporum* f. sp. *cubense* TR4 infects only bananas and plantains, and not all varieties of these, yet it has rapidly spread throughout the Cavendish-dominated banana-growing world. This mirrors the behaviour of a related strain, race 1 on Gros Michel[80]. TR4 emerged in Taiwan around 1960, rapidly spreading to Indonesia and Malaysia, and then to Australia, China and the Philippines by 2010. Its spread accelerated from 2010 onwards, reaching Africa, the Middle East, India and Pakistan, as well as more areas of Southeast Asia. It has now appeared in South America, reaching the banana plantations of Colombia in 2019 (refs. [80–91]) (Fig. 2). Possible transmission routes include the movement of infected rhizomes or spore transportation on the clothing or boots of visitors. Although *F. oxysporum* formae speciales are — like *P. oryzae* pathotypes — true specialists, they nevertheless have the capacity for gene flow with native relatives. Moreover, as with *Z. tritici*, *Fusarium* species carry dispensable chromosomes[91]; there is evidence that these chromosomes can be transmitted by horizontal transfer, vectoring the exchange of virulence genes and facilitating infection of new hosts. Thus, native *Fusarium* could provide a genetic bridge between hosts, just as Brazilian *P. oryzae* pathotypes infecting wild grasses bridged the way for a *Pyricularia* wheat pathogen to emerge[53]. This further illustrates the diverse mechanism underpinning evolutionary potential in fungal plant pathogens.

The impact of climate change on pathogen movement

Despite the importance of anthropogenic pathogen movement, it should be noted that crop pathogens, similar to any other organism, migrate when the opportunity to colonize a new suitable area arises or when changing conditions provide a pressure to move. This natural pathogen movement can play a significant role in
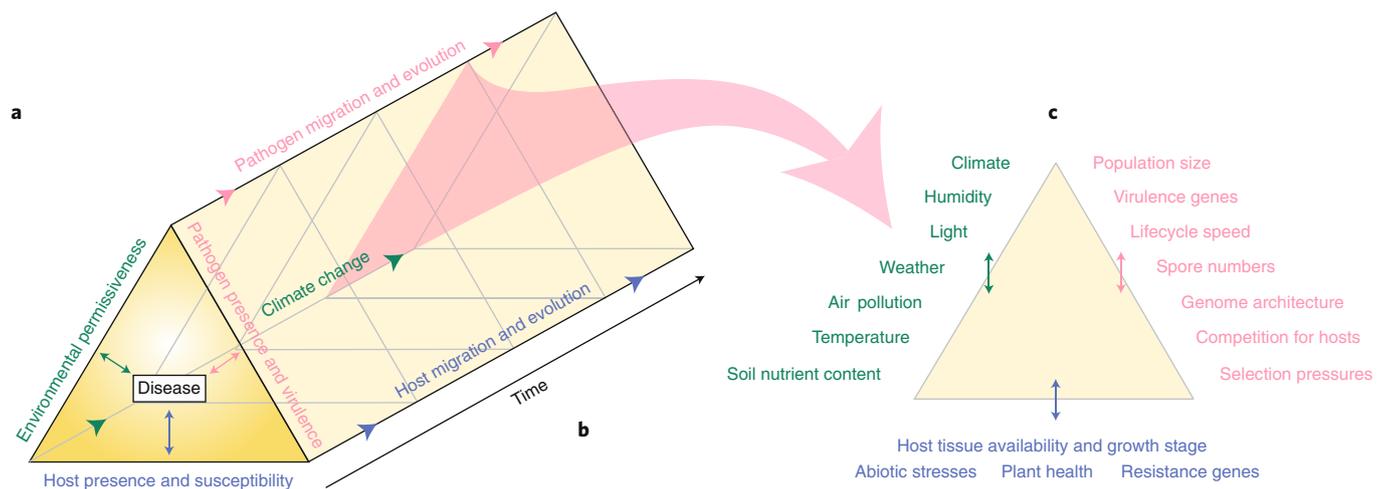
Fig. 4 | Parameterizing the disease triangle in evolutionary time. a, Disease occurrence requires the interaction of a virulent pathogen with a susceptible host under permissive conditions, forming the 'disease triangle'. **b**, As the parameters that underlie susceptibility, virulence and permissiveness change over time, it is helpful to reframe the triangle as a triangular prism, with time on the fourth axis. **c**, At any given moment in time, a triangular slice can be recovered and examined to determine the parameters on each face of the disease triangle. Parameters important for host virulence include genome and lifecycle traits that contribute to a pathogen's evolutionary potential over time, and thus to virulence in a given moment. On the host side, host tissue availability, architecture and developmental stage, overall plant health and nutrition, and R-gene complements are all important. Finally, environmental parameters determining disease outcomes include soil and air quality, weather factors, light, temperature and water availability. All these parameters interact with the features of agroecosystems that promote disease, as well as any control measures taken (see Fig. 1).

pathogen dispersal, as in the spread of ash dieback throughout continental Europe by wind-blown ascospores[92]. However, even natural movement is driven anthropogenically through the fluxes in abiotic conditions brought about by climate change. Bebber et al.[93] have demonstrated that pathogens are moving polewards as the climate warms. Human activity is not only changing the climate, but also the host availability for crop pathogens as agriculture itself responds to changing temperatures and weather patterns. This affects what crops are grown and where, and what cropping systems are used — all of which has knock-on effects for crop pathogens. An example of this already discussed is the increase in wheat production in Bangladesh, now threatened by wheat blast as a result of both anthropogenic pathogen transport and the climate changing to be more conducive to blast disease[94]. Climate change intersects with agricultural changes initiated in the Green Revolution, and the pressures created by globalization. Simplification of cropping patterns by increasing field sizes and reducing diversity to create efficient monocultures is a trend in modern, mechanized agriculture that dominates the developed world[95,96].

As global trade volume increases, we would expect farmers to specialize in crops that can be produced most efficiently to compete in global markets, leading to a reduction in cropping diversity within countries. Any trend toward reduced cropping diversity has been considered a threat to food security, but little quantitative analysis has been carried out concerning the existence of such trends. An exception to this is the work of Khoury et al.[97], who investigated trends in the composition of national food supplies for the period 1961–2009. These authors found that the global food supply has indeed become more homogeneous, with greater reliance on a few cereal and oil crops, such as soybean, oil palm and wheat[97]. On the other hand, while the increase in these staples has led to a reduction in the calories provided by crops such as sorghum, cassava and millet, these crops have not been entirely displaced by the global staples. This means that on a local level, food supplies have in fact become more diverse[97]. Similar patterns were identified even more recently[98]. We therefore performed further analysis to determine whether the same patterns are true for crops grown, as well as crops eaten. We analysed cropping diversity trends across 1980–2017 for

108 crops (that is, removing spices, fibre crops and crop groups from the FAOSTAT database) in 202 global countries and territories. Harvested crop areas were scaled by the maximum area for a crop in a particular country across all years, to remove the effects of variation in planting area across countries. Shannon's diversity index was then estimated for each year, allowing us to compare trends in cropping diversity within countries across years. We found that cropping diversity increased for 70% of countries from 1980 onwards (Fig. 3). The data provide no suggestion that wealthy or large countries are changing production diversity differently to poorer or smaller nations. The ten largest gains and losses between the 1980 and 2010 production areas occurred among the world's largest producers. Sorghum, wheat and millet production areas declined, while the soybean production area in particular grew dramatically (Table 2).

For wheat, the decline in crop area is reflected in Table 2, with a total of 16.68 Mha of wheat-growing area lost across the United States, Canada and China. However, wheat also appears among the largest gains of crop area in Table 2 (6.83 Mha gained in India). This implies a worldwide relocation of wheat-growing areas, supporting the idea that trade dictates where global staples such as wheat are grown. In combination with the increased reliance on wheat in global food supply shown previously[97], a net reduction in wheat-growing area indicates that the new growing locations may be more efficient for wheat production on a per-hectare basis. From the perspective of economic risks incurred by production losses affecting particular crops, the increase in national diversity suggests that cropping practices within countries are becoming more resilient. Meanwhile, the increased homogeneity in food supplies worldwide shows that trade has increased the interdependence of all countries. An example of the challenges that may be associated with such interdependence is the emerging pathogen, wheat blast. Increased global trade in wheat, both as part of the food supply and as seed stock for global wheat production, brings with it particular risks because this pathogen is seed-borne. This provides a strong argument for protecting local cropping diversity while maintaining the stability of global staple crop production, including in the face of emerging pathogens that may be spread via global trade for those staples crops, as a necessary two-pronged approach to global food security. The availability of

large new areas of individual crops increases the potential range of several devastating crop diseases, particularly SBR. As *P. pachyrhizi* uredospores can travel in the boundary layer of the atmosphere, even across continents (for example, via hurricane Ivan in 2004 (ref. [99])), this pathogen can follow the crop wherever it is introduced. *P. pachyrhizi*, although to a lesser extent than a true generalist, benefits from a surprisingly large host range that encompasses over 150 species within the Fabaceae[100]. Of these species, the most common alternative SBR host is the invasive legume kudzu, which serves as a spatial and temporal 'green bridge' in its disease cycle. Where the increase in soybean cropping area occurs in countries practising intensive agriculture, *P. pachyrhizi* will benefit from the evolutionary cradle that this provides to rapidly adapt to new challenges and environments, such as those presented by a differing climate.

To fully understand the threat that climate change poses to global agriculture, deep knowledge of a pathogen's evolutionary capacity regarding its tolerance of temperature, drought and other climatic factors is required. For instance, the ease or otherwise with which pathogen populations evolve new responses to temperature stress, and the diversity of temperature responses of phytopathogen communities more generally, are open questions at present. While we know that various abiotic[5] and biotic[101] factors interact with temperature in terms of their effects on pathogen spread and host resistance, these interactions are frequently complex, generating non-linear effects that can be hard to predict[101]. Further, we do not know whether specialism in one dimension of niche space correlates with specialism in others. A host-specific pathogen that has specific requirements for other parameters, such as temperature or light, may be less of a threat under climate change than an abiotically flexible but host-specific pathogen that can tolerate changes in host growing conditions, or a pathogen that can infect a range of host plants while being intolerant of abiotic changes.

Knowing the enemy

Disease control strategies should be guided by an intimate understanding of the lifecycle and genetics of a given pathogen, coupled with knowledge of the host resistance status and environmental parameters that propel a pathogen to cause disease. Pathogen, host and environment form the corners of the disease triangle, a long-established concept in plant pathology[102,103]. However, the disease triangle remains largely descriptive. Robust parameterization on a per-pathogen basis, with the inclusion of a temporal component, may enable a paradigm for infection prediction (Fig. 4). None of the elements represented are meaningful in isolation. For example, neither host resistance nor pathogen virulence can be separated from abiotic factors, and the environment cannot be meaningfully integrated into a model in which the pathogen or host's biology is treated as a black box. To illustrate the necessity of a holistic consideration of the disease triangle, we return to the wheat pathogen *Z. tritici*, causal agent of STB disease.

In 2019, Chaloner et al.[104] parametrized a new mechanistic model for predicting STB. This model was driven with experimentally derived data for the temperature- and/or wetness-determined germination, growth and death of *Z. tritici* at each stage of its lifecycle[104]. This was made possible by advances in our understanding of the *Z. tritici* lifecycle[44], which allowed more accurate definition of the periods during which the pathogen would be exposed to drying or temperature stresses. The model resolved regions of varying disease risk but was not able to predict observed annual disease, despite the advances made. This highlights the need for greater understanding of the interplay between multiple environmental, host and pathogen factors. Population-level genomics and/or metagenomics, for example, might be needed to quantify the evolutionary potential of the pathogen population and — in combination with knowledge of cultivars grown and fungicides used — may provide a first step towards understanding the risk of infection by newly emerged,

virulent or resistant isolates. Studies of the population metagenome of the human gut microbiome have shown that genomic markers can predict microbial diversity in a host[105]. Studies of the population genetics of the 2013–2016 Ebola outbreak, which allowed transmission routes to be studied and the effectiveness of policy interventions estimated[106], have shown both the potential and the pitfalls of such an approach. Incorporating genomics into our understanding of the epidemiology of emerging plant pathogens will not be trivial but is likely to provide valuable data to inform policy and protect crops. Indeed, the research response to the emergence of *P. oryzae* Triticum in Bangladesh, in which researchers created and shared field pathogenomic data during the outbreak, provides a striking illustration of what might be possible if scientists and other stakeholders work together on such projects[68].

The second step, also non-trivial, would be the definition of environmental factors that alter disease risk. The interaction of light, ultraviolet, moisture, humidity and temperature at each lifecycle stage should be understood for a fully mechanistic model describing a single growing season. We must also understand how those factors will vary under climate change, and how the pathogen will respond to those changes. To highlight regions at risk of pathogen invasion as the climate changes, for example, we need to define the pathogen's temperature niche[107]. For *Z. tritici*, steady progress has been made in this research area. First, temperature-dependent STB disease development curves were defined[108]. Next, the impact of temperature fluctuation on STB disease development was investigated[109] and in-planta thermal performance curves were developed for three strains of *Z. tritici*[110]. These efforts provided the first insights into the pathogen's temperature tolerance, and how its tolerance changes in response to disease. Boixel et al.[111] advanced this knowledge by including multiple *Z. tritici* strains in their quantification of temperature-dependent in-planta STB disease progression, illuminating the variability in these traits. However, we still have considerable gaps in our knowledge. First, we have limited knowledge regarding the shape of pathogen temperature responses, including resultant non-linearity in temperature effects, as have been reported for crops, especially within and between pathogen populations[112]. For many pathogens, we are further hampered by the paucity of published data on temperature response variation through the pathogen lifecycle[113,114], and resultant effects on overall disease severity and virulence.

In addition, high levels of variation in temperature responses within *Z. tritici* populations in a single field have been reported, with evidence also found for local thermal adaptation at a global scale[111,115]. To add to this complexity, wheat-growing regions display seasonality; hence, the selective pressures imposed by temperature on *Z. tritici* vary during an epidemic[116,117]. In *Z. tritici*, it has also been reported that higher temperatures increase genomic instability[118]. Under climate change, increasing temperatures are expected[119]. This may create greater pathogen genetic variation, increasing the ability of *Z. tritici* to evolve new virulence mechanisms and to overcome other barriers, such as fungicide application and inbred host resistance. Assuming the pathogen's responses to be uniform or creating models driven on relatively low-resolution weather data will not yield effective disease prediction. A robustly parameterized disease triangle, with a temporal element to allow for pathogen evolution (Fig. 4), would provide an ideal framework for disease modelling. It would then be at the researcher's discretion to determine which elements could best be exempted to achieve a model of sufficient simplicity.

Problems, challenges and the future of disease control

The uniformity of modern intensive agroecosystems challenges sustainability in three specific ways. First, the loss of crop diversity has provoked emergence of new virulent races capable of overcoming inbred R genes, as seen, for example, in the breakdown of the

wheat yellow-rust resistance conferred by the R gene *Yr17* (ref. [120]). Second, overreliance on particular fungicides has led to rapid emergence of new strains of fungi, such as triazole-resistant *Z. tritici* in UK wheat. Third, the loss of semi-natural landscape features, such as field margins, and depletion of natural resources have led to ecological deterioration[121].

The most valuable natural weapon against phytopathogens is a plant's own immunity. Most plants show 'non-host' resistance to most pathogens, and this is so effective that host shifts and jumps, such as those discussed above, are exceptional. The mechanisms by which plants achieve this have been reviewed in detail elsewhere[122,123]. We exploit plant immunity through breeding but, to date, we have focused such strategies on integrating a few R genes of major effect into crops[124]. Detailed discussion of the strategies used can be found in reviews by other authors[124] but in brief, breeding[125] is slow, taking 10–20 years between resistance-related marker discovery and release of new crop cultivars[126]. Faster times (around 2 years) can be achieved through transgene cloning or gene editing, although these approaches face public and political distrust, so that no transgenic antifungal plants have yet been made commercially available. As pathogen evolution in agroecosystems is rapid, R-gene-mediated resistance is rapidly overcome[127]. Greater durability in disease control can be achieved by stacking or pyramiding R genes[128], and this remains an area of active research effort. Other forms of host resistance, not dependent on R genes and already commonly used in some crops, should also be further investigated. An example of the power of such approaches can be seen in wheat rust resistance. From the 1970s to the early 2000s, resistance to stem rust (*Puccinia graminis*) in wheat was conferred in many cases by a single gene, *Sr31*. This resistance was broken by a newly emergent strain of the fungi, Ug99, which arose in Uganda in 1999 (ref. [129]). Subsequently, breeding efforts for rust resistance in wheat have focused on so-called adult partial resistance (APR) genes, which act in combination to give a 'slow-rusting' phenotype that is proving a durable form of resistance[130,131]. Such approaches, especially when combined with open-source science[68] and an internationalist not-for-profit approach to crop disease-resistance breeding, may provide our best hope of breeding durable resistance. These methods are currently espoused by global crop-breeding networks, and institutions such as the International Rice Research Institute (IRRI) and International Maize and Wheat Improvement Center (CIMMYT)[132,133].

Fungicides are another major component of our arsenal against crop pathogens. However, in striking parallel to plant R genes, approximately 77% of the world market comprises single-target-site fungicides. Their worth in 2018 was approximately US$15 billion, with their market value growing at a constant annual growth rate (CAGR) of 5.8% (ref. [134]). There are six main classes of such fungicides: morpholines and azoles (which target the fungal membrane component ergosterol); benzimidazoles (which interfere with the fungal cytoskeleton); strobilurins and succinate dehydrogenase inhibitors (SDHIs; which inhibit the mitochondrial respiration electron transport chains); and anilinopyrimidines (target mitochondrial signalling pathways). Of these, the azoles, strobilurins and SDHIs dominate, accounting for approximately 60% of the global fungicide market[134,135]. However, fungicide resistance has emerged against all major classes of single-target-site fungicides in several major crop pathogens. The mechanisms dominating such emergence are target-site mutations leading to conformational changes or overexpression of target proteins, and upregulation of efflux pumps to obviate drug accumulation[136].

We therefore need new antifungals — ideally, new chemistries with either broad-spectrum antifungal activity or the ability to boost plant defences, as these mechanisms generate the lowest risk for the emergence of fungicide resistance. New antifungals must also be environmentally benign. One such chemistry, a mono-alkyl lipophilic cation (MALC), was recently described; its potential as a fungicide merits further investigation[137]. In reality, however, fewer new antifungals are being introduced to the European market each year, as the cost of discovery, development and registration soars. Cost estimates for each new chemistry were around US$286 million in 2014, with registration costs alone accounting for more than one-third of the spend[138].

Such burgeoning costs have led to exploration of additional strategies for crop disease management. Most obvious here is the use of combinatorial fungicide mixtures to mitigate emergence of resistance. In 2014, van den Bosch provided a theoretical and lab-based validation of the claim that addition of either a multi-site (such as chlorothalonil) or single-site mixing partner to a fungicide reduces the rate of selection for fungicide resistance[139], with recent work showing that simultaneous application of a mixture of fungicides generally outperforms temporal alternation[140]. Chlorothalonil merits a further mention, for it has been an important mainstay in wheat and barley fungicide programmes since it was launched in 1964. It currently carries a market value of US$205 million per annum, growing at a CAGR of 4.61%. Despite its usefulness, this chemistry was banned from use in Europe 2019 (ref. [141]), highlighting the difficulties facing the crop protection industry and, by extension, agriculture.

Conclusions

Emerging fungal and oomycete pathogens pose a significant risk to food security worldwide, largely due to the effects of agricultural systems and practices on pathogen evolution, including monoculture planting and reliance on easily out-evolved single-R-gene or single-target-site fungicide control measures. These factors are compounded by highly variable, outcrossing and often generalist pathogens that quickly out-manoeuvre us in the arms race that we have embarked on. The adaptability of phytopathogens does not bode well for an agricultural system that will probably be tested by climate change, as the pathogens seem likely to follow their hosts around the globe and to evolve to overcome associated stresses. Further, the movement of pathogens, whether in traded goods or as a result of climate-induced migration, is likely to lend the pathogens opportunities to emerge in new places or on new hosts, as seen throughout history. Our best weapon is to understand these pathogens fully. What are the details of their lifecycles, both on and off the plant? What selection pressures do they face from ourselves, their hosts and the environment, and what is their evolutionary potential in the face of these pressures? We then must develop models that account for all such factors acting on a given pathogen but that are still simple enough to be useful in disease prediction. Fundamental research efforts should thus focus on such questions, while impact should be sought by attempting to use the resulting knowledge to inform the development of new disease control strategies.

References

1. Anderson, P. K., Cunningham, A. A., Patel, N. G., Morales, F. J., Epstein, P. R. & Daszak, P. Emerging infectious diseases of plants: pathogen pollution, climate change and agrotechnology drivers. *Trends Ecol. Evol.* **19**, 535–544 (2004).
2. Fisher, M. C. et al. Emerging fungal threats to animal, plant and ecosystem health. *Nature* **484**, 186 (2012).
3. Fones, H. N., Fisher, M. C. & Gurr, S. J. Emerging fungal threats to plants and animals challenge agriculture and ecosystem resilience. *Microbiol. Spec.* https://doi.org/10.1128/microbiolspec.FUNK-0027-2016 (2017).
4. Bebber, D. P., Ramotowski, M. A. & Gurr, S. J. Crop pests and pathogens move polewards in a warming world. *Nat. Clim. Change* **3**, 985–988 (2013).
5. Fones, H. N. & Gurr, S. J. NOXious gases and the unpredictability of emerging plant pathogens under climate change. *BMC Biol.* **15**, 36 (2017).

6. Manning, W. J. & Tiedemann, A. V. Climate change: potential effects of increased atmospheric carbon dioxide (CO_2), ozone (O_3), and ultraviolet-B (UV-B) radiation on plant diseases. *Environ. Pollut.* **88**, 219–245 (1995).

7. Ahmed, S., de Labrouhe, D. T. & Delmotte, F. Emerging virulence arising from hybridisation facilitated by multiple introductions of the sunflower downy mildew pathogen *Plasmopara halstedii*. *Fungal Genet. Biol.* **49**, 847–855 (2012).

8. Stukenbrock, E. H. Evolution, selection and isolation: a genomic view of speciation in fungal plant pathogens. *New Phytol.* **199**, 895–907 (2013).

9. Meentemeyer, R. K., Haas, S. E. & Václavík, T. Landscape epidemiology of emerging infectious diseases in natural and human-altered ecosystems. *Ann. Rev. Phytopathol* **50**, 379–402 (2012).

10. Turner, R. S. After the famine: plant pathology, *Phytophthora infestans* and the late blight of potatoes, 1845–1960. *Hist. Stud. Phys. Biol. Sci.* **34**, 341–370 (2005).

11. Fry, W. *Phytophthora infestans*: the plant (and R gene) destroyer. *Molec. Plant Pathol* **9**, 385–402 (2008).

12. Ristaino, J. B., Groves, C. T. & Parra, G. R. PCR amplification of the Irish potato famine pathogen from historic specimens. *Nature* **411**, 695–697 (2001).

13. Ristaino, J. B. Tracking historic migrations of the Irish potato famine pathogen, *Phytophthora infestans*. *Microbes Infect.* **4**, 1369–1377 (2002).

14. Yoshida, K. et al. The rise and fall of the *Phytophthora infestans* lineage that triggered the Irish potato famine. *eLife* **2**, e00731 (2013).

15. Goodwin, S. B., Cohen, B. A. & Fry, W. E. Panglobal distribution of a single clonal lineage of the Irish potato famine fungus. *Proc. Natl Acad. Sci. USA* **91**, 11591–11595 (1994).

16. Fry, W. E. et al. Five reasons to consider *Phytophthora infestans* a reemerging pathogen. *Phytopathol.* **105**, 966–981 (2015).

17. Goodwin, S. B., Sujkowski, L. S. & Fry, W. E. Rapid evolution of pathogenicity within clonal lineages of the potato late blight disease fungus. *Phytopathol* **85**, 669–676 (1995).

18. Drenth, A., Janssen, E. M. & Govers, F. Formation and survival of oospores of *Phytophthora infestans* under natural conditions. *Plant Pathol.* **44**, 86–94 (1995).

19. Andersson, B., Sandstrom, M. & Stromberg, A. Indications of soil borne inoculum of *Phytophthora infestans*. *Potato Res.* **41**, 305–310 (1998).

20. Fischer, T., Byerlee, D. & Edmeades, G. *Crop Yields and Global Food Security* (ACIAR, 2014).

21. *The State of Food and Agriculture* No. 37 (FAO, 2006).

22. *FAOSTAT* (FAO, 2016); http://www.fao.org/faostat/en/#data

23. Cassidy, E. S., West, P. C., Gerber, J. S. & Foley, J. A. Redefining agricultural yields: from tonnes to people nourished per hectare. *Environ. Res. Lett.* **8**, 034015 (2013).

24. Burles, D. *Dimensions of Need: An Atlas of Food and Agriculture* (FAO, 1995).

25. Bancroft, J. Report of the board appointed to enquire into the cause of disease affecting livestock and plants. *Votes Proc.* **3**, 1011–1038 (1876).

26. Ploetz, R. C. Panama disease: a classic and destructive disease of banana. *Plant Health Prog.* https://doi.org/10.1094/PHP-2000-1204-01-HM (2000).

27. Hippolyte, I. et al. Foundation characteristics of edible Musa triploids revealed from allelic distribution of SSR markers. *Ann. Bot.* **109**, 937–951 (2012).

28. Ordonez., N. et al. Worse comes to worst: bananas and Panama disease – when plant and pathogen clones meet. *PLoS Pathog.* **11**, e1005197 (2015).

29. Galvis, S. Colombia confirms that dreaded fungus has hit its banana plantations. *Science* https://doi.org/10.1126/science.aaz1033 (2019).

30. Rajaram, S. Norman Borlaug: the man I worked with and knew. *Ann. Rev. Phytopathol.* **49**, 17–30 (2011).

31. Weiner, J. Applying plant ecological knowledge to increase agricultural sustainability. *J. Ecol.* **105**, 865–870 (2017).

32. Evenson, R. E. & Gollin, D. Assessing the impact of the Green Revolution, 1960 to 2000. *Science* **300**, 758–762 (2003).

33. Trewavas, A. Malthus foiled again and again. *Nature* **418**, 668–670 (2002).

34. Tilman, D., Cassman, K. G., Matson, P. A., Naylor, R. & Polasky, S. Agricultural sustainability and intensive production practices. *Nature* **418**, 671–677 (2002).

35. Fones, H. & Gurr, S. The impact of *Septoria tritici* blotch disease on wheat: an EU perspective. *Fungal Genet. Biol.* **79**, 3–7 (2015).

36. Linde, C. C., Zhan, J. & McDonald, B. A. Population structure of *Mycosphaerella graminicola*: from lesions to continents. *Phytopathology* **92**, 946–955 (2002).

37. McDonald, B. A. & Stukenbrock, E. H. Rapid emergence of pathogens in agro-ecosystems: global threats to agricultural sustainability and food security. *Phil. Trans. Royal Soc. B* **371**, 20160026 (2016).

38. Zhan, J., Pettway, R. E. & McDonald, B. A. The global genetic structure of the wheat pathogen *Mycosphaerella graminicola* is characterized by high nuclear diversity, low mitochondrial diversity, regular recombination, and gene flow. *Fungal Genet. Biol.* **38**, 286–297 (2003).

39. Möller, M. & Stukenbrock, E. H. Evolution and genome architecture in fungal plant pathogens. *Nat. Rev. Microbiol.* **15**, 756–771 (2017).

40. Plissonneau, C., Stürchler, A. & Croll, D. The evolution of orphan regions in genomes of a fungal pathogen of wheat. *mBio* **7**, e01231-16 (2016).

41. Stukenbrock, E. H. et al. The making of a new pathogen: insights from comparative population genomics of the domesticated wheat pathogen *Mycosphaerella graminicola* and its wild sister species. *Genome Res.* **21**, 2157–2166 (2011).

42. Croll, D., Zala, M. & McDonald, B. A. Breakage-fusion-bridge cycles and large insertions contribute to the rapid evolution of accessory chromosomes in a fungal pathogen. *PLoS Genet.* **9**, e1003567 (2013).

43. Wittenberg, A. H. et al. Meiosis drives extraordinary genome plasticity in the haploid fungal plant pathogen *Mycosphaerella graminicola*. *PLoS One* **4**, e5863 (2009).

44. Fones, H. N., Eyles, C. J., Kay, W., Cowper, J. & Gurr, S. J. A role for random, humidity-dependent epiphytic growth prior to invasion of wheat by *Zymoseptoria tritici*. *Fungal Genet. Biol.* **106**, 51–60 (2017).

45. Suffert, F., Sache, I. & Lannou, C. Early stages of *Septoria tritici* blotch epidemics of winter wheat: build-up, overseasoning, and release of primary inoculum. *Plant Pathol.* **60**, 166–177 (2011).

46. Suffert, F., Ravigné, V. & Sache, I. Seasonal changes drive short-term selection for fitness traits in the wheat pathogen *Zymoseptoria tritici*. *Appl. Environ. Microbiol.* **81**, 6367–6379 (2015).

47. van den Berg, F., Paveley, N. D. & van den Bosch, F. Dose and number of applications that maximize fungicide effective life exemplified by *Zymoseptoria tritici* on wheat – a model analysis. *Plant Pathol.* **65**, 1380–1389 (2016).

48. Torriani, S. F. et al. *Zymoseptoria tritici*: a major threat to wheat production, integrated approaches to control. *Fungal Genet. Biol.* **79**, 8–12 (2015).

49. Li, X. et al. The uniqueness of the soybean rust pathosystem: an improved understanding of the risk in different regions of the world. *Plant Dis.* **94**, 796–806 (2010).

50. Rosa, C. R. E., Spehar, C. R. & Liu, J. Q. Asian soybean rust resistance: an overview. *J. Plant Pathol. Microbiol.* https://doi.org/10.4172/2157-7471.1000307 (2015).

51. Childs, S. P., Buck, J. W. & Li, Z. Breeding soybeans with resistance to soybean rust (*Phakopsora pachyrhizi*). *Plant Breeding* **137**, 250–261 (2018).

52. Islam, M. T. et al. Emergence of wheat blast in Bangladesh was caused by a South American lineage of *Magnaporthe oryzae*. *BMC Biol.* **14**, 84 (2016).

53. Valent, B. et al. *Pyricularia graminis-tritici* is not the correct species name for the wheat blast fungus: response to Ceresini et al. *Molec. Plant. Pathol.* **20**, 173–179 (2019).

54. Skamnioti, P. & Gurr, S. J. Against the grain: safeguarding rice from rice blast disease. *Trends Biotech.* **27**, 141–150 (2009).

55. Stukenbrock, E. H. & McDonald, B. A. The origins of plant pathogens in agro-ecosystems. *Annu. Rev. Phytopathol.* **46**, 75–100 (2008).

56. Ceresini, P. C. et al. Wheat blast: from its origins in South America to its emergence as a global threat. *Molec. Plant Pathol.* **20**, 155–172 (2019).

57. DÁvila, L. S., De Filippi, M. C. C. & Café-Filho, A. C. Both MAT1–1 and MAT 1–2 idiomorphs present in rice blast populations (*Magnaporthe oryzae*) collected in rice fields in northern Brazil. *New Dis. Rep.* **40**, 3 (2019).

58. Prabhu, A. S., Filippi, M. C., Silva, G. B., Lobo, V. L. S. & Morais, O. P. in *Advances in Genetics, Genomics and Control of Rice Blast Disease* (eds Wang, G.-L. & Valent, B.) 257–266 (Springer, 2009).

59. Inoue, Y. et al. Evolution of the wheat blast fungus through functional losses in a host specificity determinant. *Science* **357**, 80–83 (2017).

60. Castroagudin, V. et al. The wheat blast pathogen *Pyricularia graminis-tritici* has complex origins and a disease cycle spanning multiple grass hosts. Preprint at https://www.biorxiv.org/content/10.1101/203455v1 (2017).

61. Mottaleb, K. A. et al. Threat of wheat blast to South Asia's food security: an ex-ante analysis. *PLoS One* **13**, e0197555 (2018).

62. Brasier, C. M. & Kirk, S. A. Rapid emergence of hybrids between the two subspecies of *Ophiostoma novo-ulmi* with a high level of pathogenic fitness. *Plant Pathol.* **59**, 186–199 (2010).

63. Chavez, V. A., Parnell, S. & van den Bosch, F. V. D. Designing strategies for epidemic control in a tree nursery: the case of ash dieback in the UK. *Forests* **6**, 4135–4145 (2015).

64. Heuch, J. What lessons need to be learnt from the outbreak of ash dieback disease, *Chalara fraxinea* in the United Kingdom? *Arboricult. J.* **36**, 32–44 (2014).

65. *Living Ash Project Survey* (Living Ash Project); https://livingashproject.org.uk/survey

66. Skovsgaard, J. P. et al. Silvicultural strategies for *Fraxinus excelsior* in response to dieback caused by *Hymenoscyphus fraxineus*. *For. Intl J. For. Res.* **90**, 455–472 (2017).

67. *Managing Ash Dieback Case Studies* (Royal Forestry Society, Forestry Commission, 2019).

68. Kamoun, S., Talbot, N. J. & Islam, M. T. Plant health emergencies demand open science: tackling a cereal killer on the run. *PLoS Biol.* **17**, e3000302 (2019).

69. Raffaele, S. & Kamoun, S. Genome evolution in filamentous plant pathogens: why bigger can be better. *Nat. Rev. Microbiol.* **10**, 417–430 (2012).

70. Guo, H., Li, C. P., Shi, T., Fan, C. J. & Huang, G. X. First report of *Phytophthora palmivora* causing root rot of cassava in China. *Plant Dis.* **96**, 1072–1072 (2012).

71. Lebot, V. *Tropical Root and Tuber Crops: Cassava, Sweet Potato, Yams and Aroids* Vol. 17 (CABI, 2009).

72. Reddy, P. P. *Plant Protection in Tropical Root and Tuber Crops* (Springer, 2015).

73. Álvarez, E., Llano, G. & Mejía, J. F. *Cassava Diseases* (CIAT, 2012).

74. Johnson, I. & Palaniswami, A. *Phytophthora* tuber rot of cassava - a new record in India. *J. Mycol. Plant Pathol.* **29**, 323–332 (1999).

75. Maizatul-Suriza, M., Dickinson, M. & Idris, A. S. Molecular characterization of *Phytophthora palmivora* responsible for bud rot disease of oil palm in Colombia. *World. J. Microbiol. Biotech.* **35**, 44 (2019).

76. Torres, G. A., Sarria, G. A., Martinez, G., Varon, F., Drenth, A. & Guest, D. I. Bud rot caused by *Phytophthora palmivora*: a destructive emerging disease of oil palm. *Phytopathol* **106**, 320–329 (2016).

77. Kaur, S., Dhillon, G. S., Brar, S. K., Vallad, G. E., Chand, R. & Chauhan, V. B. Emerging phytopathogen *Macrophomina phaseolina*: biology, economic importance and current diagnostic trends. *Crit. Rev. Microbiol.* **38**, 136–151 (2012).

78. Msikita, W., James, B., Wilkinson, H. T. & Juba, J. H. First report of *Macrophomina phaseolina* causing pre-harvest cassava root rot in Benin and Nigeria. *Plant Dis.* **82**, 1402–1402 (1998).

79. de Queiroz Brito, A. C. et al. First report of *Macrophomina pseudophaseolina* causing stem dry rot in cassava in Brazil. *J. Plant Pathol.* **1**, 1 (2019).

80. Ploetz, R. C. Fusarium wilt of banana. *Phytopathol* **105**, 1512–1521 (2015).

81. *Tropical Race 4: Distribution* (Promusa); http://www.promusa.org/tiki-index.php?page=Tropical%20race%204%20-%20TR4#Distribution

82. Buddenhagen, I. Understanding strain diversity in *Fusarium oxysporum* f. sp. *cubense* and history of introduction of 'Tropical Race 4' to better manage banana production. *Acta Horticult* **828**, 193–204 (2009).

83. Davis, R. I., Moore, N. Y., Bentley, S., Gunua, T. G. & Rahamma, S. Further records of *Fusarium oxysporum* f. sp. *cubense* from New Guinea. *Austral. Plant Pathol.* **29**, 224 (2000).

84. Qi, Y. X., Zhang, X., Pu, J. J., Xie, Y. X., Zhang, H. Q. & Huang, S. L. Race 4 identification of *Fusarium oxysporum* f. sp. *cubense* from Cavendish cultivars in Hainan province, China. *Austral. Plant Dis.* **3**, 46–47 (2008).

85. Ploetz, R. et al. Tropical race 4 of Panama disease in the Middle East. *Phytoparasitica* **43**, 283–293 (2015).

86. Syed, R. N. et al. First report of panama wilt disease of banana caused by *Fusarium oxysporum* f. sp. *cubense* in Pakistan. *J. Plant Pathol.* **1**, 213 (2015).

87. Zheng, S. J., García-Bastidas, F. A., Li, X., Zeng, L. & Bai, T. New geographical insights of the latest expansion of *Fusarium oxysporum* f.sp. *cubense* tropical race 4 into the Greater Mekong subregion. *Front. Plant Sci.* **9**, 457 (2018).

88. O'Neill, W. T. et al. Detection of *Fusarium oxysporum* f. sp. *cubense* tropical race 4 strain in northern Queensland. *Austral. Plant Dis.* **11**, 33 (2016).

89. Maymon, M. et al. First report of *Fusarium oxysporum* f. sp. *cubense* tropical race 4 causing Fusarium wilt of Cavendish bananas in Israel. *Plant Dis.* **59**, 348 (2018).

90. Damodaran, T. et al. First report of *Fusarium wilt* in banana caused by *Fusarium oxysporum* f. sp. *cubense* tropical race 4 in India. *Plant Dis.* **103**, 367 (2019).

91. Coleman, J. J., Wasmann, C. C., Usami, T., White, G. J. & Temporini, E. D. Characterization of the gene encoding Pisatin Demethylase (FoPDA 1) in *Fusarium oxysporum*. *Mol. Plant Microbe Interact.* **24**, 1482–1491 (2011).

92. Wylder, B., Biddle, M., King, K., Baden, R. & Webber, J. Evidence from mortality dating of Fraxinus excelsior indicates ash dieback (*Hymenoscyphus fraxineus*) was active in England in 2004–2005. *For. Int. J. For. Res* **91**, 434–443 (2018).

93. Bebber, D. P., Field, E., Gui, H., Mortimer, P., Holmes, T. & Gurr, S. J. Many unreported crop pests and pathogens are probably already present. *Glob. Change Biol.* **25**, 2703–2713 (2019).

94. Islam, M. T., Kim, K. H. & Choi, J. Wheat blast in Bangladesh: the current situation and future impacts. *Plant Pathol. J.* **35**, 1 (2019).

95. Lowder, S. K., Skoet, J. & Raney, T. The number, size, and distribution of farms, smallholder farms, and family farms worldwide. *World Dev.* **87**, 16–29 (2016).

96. Ricciardi, V., Ramankutty, N., Mehrabi, Z., Jarvis, L. & Chookolingo, B. How much of the world's food do smallholders produce? *Global Food Secur.* **17**, 64–72 (2018).

97. Khoury, C. K. et al. Increasing homogeneity in global food supplies and the implications for food security. *Proc. Natl Acad. Sci. USA* **111**, 4001–4006 (2014).

98. Bentham, J. et al. Multidimensional characterization of global food supply from 1961 to 2013. *Nat. Food* **1**, 70–75 (2020).

99. Schneider, R. W. et al. First report of soybean rust caused by *Phakopsora pachyrhizi* in the continental United States. *Plant Dis.* **89**, 774–774 (2005).

100. Slaminko, T. L., Miles, M. R., Frederick, R. D., Bonde, M. R. & Hartman, G. L. New legume hosts of *Phakopsora pachyrhizi* based on greenhouse evaluations. *Plant Dis.* **92**, 767–771 (2008).

101. Del Cid, C., Krugner, R., Zeilinger, A. R., Daugherty, M. P. & Almeida, R. P. Plant water stress and vector feeding preference mediate transmission efficiency of a plant pathogen. *Environ. Entomol* **47**, 1471–1478 (2018).

102. Agrios, G. N. *Plant Pathology* (Academic Press, 2005).

103. Scholthof, K. B. G. The disease triangle: pathogens, the environment and society. *Nat. Rev. Microbiol.* **5**, 152–156 (2007).

104. Chaloner, T. M., Fones, H. N., Varma, V., Bebber, D. P. & Gurr, S. J. A new mechanistic model of weather-dependent *Septoria tritici* blotch disease risk. *Phil. Trans. Roy. Soc. B* **374**, 20180266 (2019).

105. Zhernakova, A. et al. Population-based metagenomics analysis reveals markers for gut microbiome composition and diversity. *Science* **352**, 565–569 (2016).

106. Holmes, E. C., Dudas, G., Rambaut, A. & Andersen, K. G. The evolution of Ebola virus: unsights from the 2013–2016 epidemic. *Nature* **538**, 193–200 (2016).

107. Chaloner, T. M., Gurr, S. J. & Bebber, D. P. Geometry and evolution of the ecological niche in plant-associated microbes. *Nat. Commun.* (in the press).

108. Shaw, M. Effects of temperature, leaf wetness and cultivar on the latent period of *Mycosphaerella graminicola* on winter wheat. *Plant Pathol.* **39**, 255–268 (1990).

109. Bernard, F. *The Development of a Foliar Fungal Pathogen Does React to Temperature, but to which Temperature?* PhD thesis, AgroParisTech (2012).

110. Bernard, Frédéric, Sache, I., Suffert, F. & Chelle, M. The development of a foliar fungal pathogen does react to leaf temperature! *New Phytol.* **198**, 232–240 (2013).

111. Boixel, A. L., Delestre, G., Legeay, J., Chelle, M. & Suffert, F. Phenotyping thermal responses of yeasts and yeast-like microorganisms at the individual and population levels: proof-of-concept, development and application of an experimental framework to a plant pathogen. *Microbial Ecol.* **78**, 42–56 (2019).

112. Schlenker, W. & Roberts, M. J. Nonlinear temperature effects indicate severe damages to US crop yields under climate change. *Proc. Natl Acad. Sci. USA* **106**, 15594–15598 (2009).

113. Bebber, D. P., Castillo, Á. D. & Gurr, S. J. Modelling coffee leaf rust risk in Colombia with climate reanalysis data. *Phil. Trans. R. Soc. B* **371**, 20150458 (2016).

114. Lewis, C. M. et al. Potential for re-emergence of wheat stem rust in the United Kingdom. *Comm. Biol.* **1**, 13 (2018).

115. Croll, D. & McDonald, B. A. The genetic basis of local adaptation for pathogenic fungi in agricultural ecosystems. *Molec. Ecol.* **26**, 2027–2040 (2017).

116. Lovell, D. J., Hunter, T., Powers, S. J., Parker, S. R. & van den Bosch, F. Effect of temperature on latent period of septoria leaf blotch on winter wheat under outdoor conditions. *Plant Pathol.* **53**, 170–181 (2004).

117. Suffert, F. & Thompson, R. N. Some reasons why the latent period should not always be considered constant over the course of a plant disease epidemic. *Plant Pathol.* **67**, 1831–1840 (2018).

118. Möller, M., Habig, M., Freitag, M. & Stukenbrock, E. H. Extraordinary genome instability and widespread chromosome rearrangements during vegetative growth. *Genetics* **210**, 517–529 (2018).

119. IPCC *Climate Change 2014: Synthesis Report* (eds Core Writing Team, Pachauri, R. K. & Meyer L. A.) (IPCC, 2014).

120. Bayles, R., Flath, K., Hovmøller, M. & de Vallavieille-Pope, C. Breakdown of the *Yr17* resistance to yellow rust of wheat in northern Europe. *Agronomie.* **20**, 805–811 (2000).

121. He, D. C., Zhan, J. S. & Xie, L. H. Problems, challenges and future of plant disease management: from an ecological point of view. *J. Integrat. Agricult.* **15**, 705–715 (2016).

122. Lee, H. A. et al. Current understandings of plant nonhost resistance. *Molec. Plant Microbe Interact.* **30**, 5–15 (2017).

123. Jones, J. D. & Dangl, J. L. The plant immune system. *Nature* **444**, 323 (2006).

124. Gurr, S. J. & Rushton, P. J. Compatibility and disease and incompatibility and defence in plant–pathogen interactions. *Trends Biotechnol.* **6**, 275–282 (2005).

125. McDowell, J. M. & Woffenden, B. J. Plant disease resistance genes: recent insights and potential applications. *Trends Biotechnol.* **21**, 178–183 (2003).

126. Ashkani, S. et al. Molecular breeding strategy and challenges towards improvement of blast disease resistance in rice crop. *Front. Plant Sci.* **6**, 886 (2015).

127. Brown, J. K. Durable resistance of crops to disease: a Darwinian perspective. *Ann. Rev. Phytopathol* **53**, 513–539 (2015).

128. Fuchs, M. Pyramiding resistance-conferring gene sequences in crops. *Curr. Op. Virol.* **26**, 36–42 (2017).

129. Singh, R. P. et al. The emergence of Ug99 races of the stem rust fungus is a threat to world wheat production. *Annu. Rev. Phytopathol.* **49**, 465–481 (2011).

130. Singh, R. P., Huerta-Espino, J. & William, H. M. Genetics and breeding for durable resistance to leaf and stripe rusts in wheat. *Turk. J. Agric. Forest.* **29**, 121–127 (2005).

131. Rehman, M. U. et al. Adult plant resistance to stem rust (*Puccinia graminis* f. sp. *tritici*) in Pakistani advanced lines and wheat varieties. *Austral. J. Crop Sci* **12**, 1633–1639 (2018).

132. Garrett, K. A. et al. Resistance genes in global crop breeding networks. *Phytopathol.* **107**, 1268–1278 (2017).

133. Byerlee, D. & Dubin, H. J. Crop improvement in the CGIAR as a global success story of open access and international collaboration. *Internat. J. Comm* **4**, 452–480 (2009).

134. *Global Fungicides Market Research Report* (Globe Newswire, 2018).

135. Oliver, R. P. & Hewitt, H. G. *Fungicides in Crop Protection* (CABI, 2014).

136. Fisher, M. C., Hawkins, N. J., Sanglard, D. & Gurr, S. J. Worldwide emergence of resistance to antifungal drugs challenges human health and food security. *Science* **360**, 739–742 (2018).

137. Steinberg, G. et al. A lipophilic cation protects crops against fungal pathogens by multiple modes of action. *Nat. Commun.* **11**, 1608 (2020).

138. McDougall, P. *Evolution of the Crop Protection Industry Since 1960* (Informa, 2019).

139. Bosch, F. V. D., Oliver, R., van den Berg, F. & Paveley, N. Governing principles can guide fungicide-resistance management tactics. *Ann. Rev. Phytopathol.* **52**, 175–195 (2018).

140. Elderfield, J. A., Lopez-Ruiz, F. J., van den Bosch, F. & Cunniffe, N. J. Using epidemiological principles to explain fungicide resistance management tactics: Why do mixtures outperform alternations? *Phytopathol.* **108**, 803–817 (2018).

141. EU votes to withdraw chlorothalonil. *AgriTradeNews* (29 March 2019).

Author contributions
H.F. wrote the paper with S.G. H.F. made Figs. 1, 3 and 4 and Tables 1 and 2; G.S. made Fig. 2. D.B. carried out data analyses. W.K., T.C., D.B. and G.S. contributed to the writing of the paper. All authors proofread and approved the submitted work.

Competing interests
The authors declare no competing interests.

Additional information
Correspondence should be addressed to H.N.F. or S.J.G.

Reprints and permissions information is available at www.nature.com/reprints.

Publisher's note Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.