Chapter 25

Plant Pathogens as Indicators of Climate Change

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1. INTRODUCTION

Plant disease risk is strongly influenced by environmental conditions [1]. While some animal hosts may provide their pathogens with a consistent range of body temperatures, plant pathogens are generally much more exposed to the elements. Plant disease will tend to respond to climate change, though a number of interactions take place among host, pathogen, potential vectors. In some cases, the actions of land managers may also complicate interpretation of climate change effects. In this chapter, we present a brief introduction to plant disease and a synthesis of research in plant pathology related to climate change. We discuss the types of evidence for climate change impacts ('climate change fingerprints') that might be observed in plant disease systems and evaluate what evidence of climate change fingerprints currently exists.

The battle against plant disease is not a new one, and plant disease management is essential for our continued ability to feed a growing human population. The Great Irish Hunger is one striking example of the impact of plant disease: in 1845 more than a quarter million Irish people starved as the result of an epidemic of potato late blight [2]. Plant diseases continue to cause serious problems in global food production. Currently more than 800 million people do not have adequate food and at least 10% of global food production

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is lost to plant disease [3]. Not only does plant disease affect human food production, it also impacts natural systems [4]. Introduced diseases such as chestnut blight in the Eastern US, and more recently the increasing occurrence of sudden oak death, have resulted in the rapid decline of dominant tree species and triggered major impacts on forest systems [5].

Plant pathogen groups include fungi, prokaryotes (bacteria and mycoplasmas), oomycetes, viruses and viroids, nematodes, parasitic plants and protozoa. The very different life histories of this diverse group of organisms and their different interactions with host plants produce a wide range of responses to environmental and climatic drivers. For example, viruses may be present in hosts while symptom expression is dependent on temperature [6]; thus, even the difficulty of detection of these pathogens varies with climate. Fungal pathogens are often strongly dependent on humidity or dew for plant infection [7], so changes in these environmental factors are likely to shift disease risk. Genetic variation in pathogen populations often makes plant disease management more complicated when pathogens overcome host disease resistance [3]. Pathogen species may quickly develop resistance to pesticides or adapt to overcome plant disease resistance, and may also adapt to environmental changes, where the rate of adaptation depends on the type of pathogen [8]. Pathogen populations may explode when weather conditions are favourable for disease development [9,10]. The potentially rapid onset of disease makes it difficult to anticipate the best timing of management measures, especially in areas with high levels of interannual variability in climatic conditions.

2. CLIMATIC VARIABLES AND PLANT DISEASE

Understanding the factors that trigger the development of plant disease epidemics is essential if we are to create and implement effective strategies for disease management [11]. This has motivated a large body of research addressing the effects of climate on plant disease [11,12]. Plant disease occurrence is generally driven by three factors: a susceptible host, the presence of a competent pathogen (and vector if needed) and conducive environment [9,10]. All three of these factors must be in place, at least to some degree, for disease to occur (Fig. 1). A host resistant to local pathogen genotypes or unfavourable weather for pathogen infection will lessen disease intensity. The synchronous interaction between host, pathogen and environment governs disease development. These interactions can be conceptualised as a continuous sequence of cycles of biological events including dormancy, reproduction, dispersal and pathogenesis [1]. In plant pathology this sequence of events is commonly referred to as a disease cycle. Although plant pathologists have long realised the importance of the disease cycle and its component events and the apparent relationships with environment, the quantification of these interactions did not begin in earnest until the 1950s [11]. The past five decades of research have

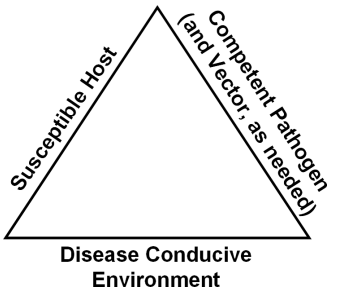

FIGURE 1 Plant disease results from the interaction of host, pathogen and environment. Climatic features such as temperature, humidity and leaf surface wetness are important drivers of disease, and inappropriate levels of these features for a particular disease may be the limiting factor in disease risk.

established a vast body of literature documenting the impact of temperature, rainfall amounts and frequency and humidity, on the various components of the disease cycle [11].

The quantification of the relationship between the disease cycle of a given plant disease and weather is also the foundation of many prediction models that can be used to advise growers days or weeks before the onset of an increase in disease incidence or severity [1]. Such prediction tools can allow a grower to respond in a timely and efficient manner by adjusting crop management practices. Given enough time to respond, a disease prediction might allow a grower to alter the cultivar they select for planting, the date on which the crop is sown, or the scheduling of cultural practices such as fertilisation or irrigation. A prediction of a low disease risk may also result in reduced pesticide use with positive economic and environmental outcomes. Larger scale predictions of disease risk, such as the typical risk for regions or countries based on climatic conditions, can be used to form policy and priorities for research (e.g. [13]).

Interestingly, the quantification of these relationships and application of this information as part of disease prediction models has also facilitated the simulation of potential impacts of climate change. For example, Bergot et al. [14] have used models of the impact of weather variables on the risk of infection by *Phytophthora cinnamomi* to predict the future distribution of disease caused by this pathogen in Europe under climate change scenarios. As more detailed climate change predictions are more readily available, many plant disease forecasting systems may be applied in this context.

Some relationships between climate and disease risk are obvious, such as some pathogens' inability to infect without sufficient surface moisture (i.e. dew or rain droplets) [7] or other pathogens' or vectors' inability to overwinter when temperatures go below a critical level. Other effects of climate may be more subtle. For example, a given pathogen may only be able to infect its host(s) when the plants are in certain developmental stages. This also means

that in order to maximise their chance of infection, the life cycle of pathogen populations must be in sync with host development. Since climate change can influence the rate of both host and pathogen development, it could affect the development and impact of plant diseases. Here, we discuss a few examples where host phenology is the key to disease development.

Some pathogens depend on flower tissues as a point of entry to the host. For example, *Botrytis cinerea*, which causes gray mold of strawberry and other fruits (producing a gray fuzz-balled strawberry, which you may have seen at a grocery store or in your refrigerator), infects strawberry at the time of flowering [15]. It stays in flower parts until the sugar level of the berry increases, and then causes gray mold disease. Another example is Fusarium head blight of wheat and barley, which causes large yield losses, reductions in grain quality and contamination with mycotoxins (toxic substances created by the fungi) [16,17]. Several fungal species including *Fusarium graminearum* (teleomorph: *Gibberella zeae*) cause this disease, and anthesis (flowering) period seems to be the critical time for infection [17,18]. An important bacterial disease of apple and pears, called fire blight, also utilises flowers as a major point of entry [19]. The causal agent (*Erwinia amylovora*) can be disseminated by pollinating insects such as bees and moves into flowers to cause rapid wilting of branch tips.

Certain hosts become more resistant after a particular developmental stage, some exhibiting a trait referred to as adult plant resistance. There are many examples of genes that follow this pattern in wheat, including leaf rust (caused by the fungus *Puccinia triticina*) resistance genes *Lr13* and *Lr34* [20] and stripe rust (caused by *Puccinia striiformis* f. sp. *tritici*) resistance gene *Yr39* [21]. These genes are activated by a combination of wheat developmental stage and temperature changes. In grape, there are many cases of ontogenic (or age-related) resistance against pathogens. Once grape fruit tissue matures, certain fungal pathogens such as *Erysiphe necator* (formerly *Uncinula necator*, causing powdery mildew) [22], or *Guignardia bidwellii* (causing black rot) [23], or the oomycete pathogen *Plasmopara viticola* (causing downy mildew) [2] are less successful at infecting plants.

With changes in climate, host development patterns may be altered. For the examples above, the timing and duration of flowering in wheat are a function of the average daily temperature. Heavy rain and/or strong wind events can shorten flowering duration in strawberry and apple through flower damage. Some pathogen species may be able to maintain their synchrony with target host tissue, and others may become out of sync. Thus, there are some efforts to modify disease prediction systems to accommodate potential impacts from climate change. For example, in efforts to predict the risk of apple scab (caused by the fungus *Venturia inaequalis*), the concept of ontogenic resistance was utilised along with inoculum production [24] because tissues become less susceptible as the rate of tissue expansion decreases.

There is no doubt that weather influences plant disease; that relationship is fundamental to the modelling of plant disease epidemiology. Thus, it is fairly straightforward to predict that where climate change leads to weather events that are more favourable for disease, there will be increased disease pressure. But the relationship between climate change and associated weather events, and resulting changes in disease development will generally not be a simple one-to-one relationship (Fig. 2). The impacts will tend to be most dramatic when climatic conditions shift above a threshold for pathogen reproduction, are amplified through interactions, or result in positive feedback loops that decrease the utility of disease management strategies [25]. For example, the Karnal bunt pathogen, *Tilletia indica*, which reduces wheat quality, will tend to have lower reproductive rates per capita when populations are low because individuals of different mating types must encounter each other for reproductive success [26]. If climatic conditions change to favour pathogen reproduction, the pathogen will be released from this constraint and show a larger response to the change than would otherwise have been anticipated. The trend toward greater global movement of humans and materials also produces new types of interactions as pathogens are introduced to new areas and may hybridise to produce new pathogens [27,28].

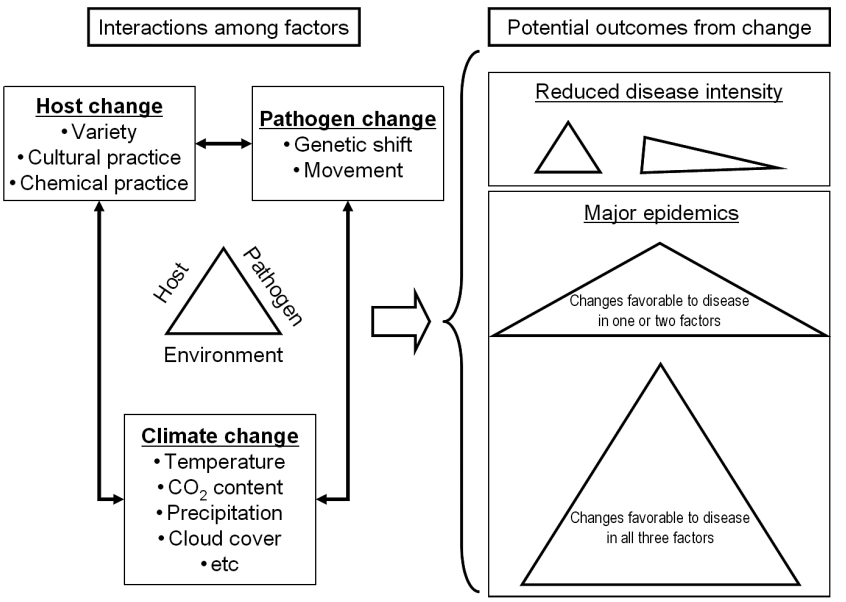

FIGURE 2 Interactions among components of the disease triangle and potential outcomes. Amount of disease [quantity (incidence, severity, etc.) or quality (risk)] is indicated by the area of the triangle. Changes in host, pathogen and climate can increase or decrease the amount of disease as a result of their interactions.

3. EVIDENCE THAT SIMULATED CLIMATE CHANGE AFFECTS PLANT DISEASE IN EXPERIMENTS

Next we consider two types of evidence for effects of changes in climate on plant disease. The first is evidence that simulated climate change affects plant disease in experimental settings. The effect of simulated climate change has been studied in experiments with altered heat treatments, altered precipitation treatments and carbon enrichment treatments. Where there are apparent effects from these treatments, this implies that, to the extent that the simulations do effectively represent future climate scenarios, plant disease will respond. The second type of evidence is for changes in patterns of plant disease in agricultural or wildland systems that can be attributed to climate change with some level of confidence, discussed in Section 4. In this case, the changes in plant disease might be taken as fingerprints of climate change. We also discuss what types of plant disease scenarios might qualify as fingerprints of climate change in this sense.

The range of possibilities for climate change simulations can be characterised in terms of the scale of the effect being considered [29]. For many well-studied pathogens and vectors, the temperature ranges that support single infection events or survival are fairly well characterised. The effects of plant water stress and relief from water stress on disease risk have also been studied in controlled experiments for some pathogens, and may be quite relevant to scenarios where patterns of drought occurrence are changing. Advances in the development of technologies such as microarrays make it possible to study drought effects on plant gene expression in the field, including genes that may be important for disease resistance [30]. Drawing conclusions about larger-scale processes from plot-level experiments may be challenging, however, since additional forms of interactions are important at larger scales.

Field experiments that incorporate simulations of changes in temperature and/or precipitation are becoming increasingly common in both agricultural and natural systems, often associated with long-term study systems such as the US National Science Foundation's Long-term Ecological Research sites. For example, in Montane prairie Roy et al. [31] studied the impact of heating treatments on a suite of plant diseases. They found that higher temperatures favoured some diseases but not others. This type of 'winners and losers' scenario is likely to be common as more systems are evaluated; the overall level of disease under climate change may be buffered in some environments as some diseases become less common and others become more common.

The impact of elevated CO_2 on plant disease has been evaluated in the context of several free-air CO_2 enrichment (FACE) experiments (reviewed in Ref. [32]). Compared to studies in experimental chambers, FACE experiments allow more realistic evaluations of the effects of elevated CO_2 levels in agricultural fields or natural systems such as forests. Higher CO_2 levels may favour disease through denser more humid plant canopies and increased pathogen

reproduction but may reduce disease risk by enhancing host disease resistance [33], so the outcome for any given host-pathogen interaction is not readily predictable. Elevated ozone levels can also affect plant disease risk (reviewed in Ref. [32]).

In addition to the more direct influences of the abiotic environment on plant disease, climate change may also affect plant disease through its impact on other microbes that interact with pathogens. While certain microbes affect plant pathogens strongly enough to be used as biocontrol agents, a number of microbial interactions probably also have more subtle effects. As the effect of climate change on microbial communities is better understood [34], this additional form of environmental interaction can be included in models of climate and disease risk.

4. EVIDENCE THAT PLANT DISEASE PATTERNS HAVE CHANGED DUE TO CLIMATE CHANGE

If patterns of plant disease in an area have shifted at the same time that changes in climate are observed, when can this correlation be taken as evidence of climate change impacts on disease? Such an analysis is complicated by the number of factors that interact to result in plant disease. For example, if a disease becomes important in an area in which it was not important in the past, there are several possible explanations. The pathogen populations may have changed so that they can more readily infect and damage hosts. The pathogen species or particular vectors of the pathogen may be newly introduced to the area. In agricultural systems, host populations may have changed as managers have selected new cultivars based on criteria other than resistance to the disease in question. Management of the abiotic environment may have changed, such as changes in how commonly fields are tilled (tillage often reduces disease pressure), or changes in planting dates (which may result in more or less host exposure to pathogens). To rule out such competing explanations for changes in plant disease pattern, the argument for climate change as an important driver is strongest when (a) the pathogen is known to have been present throughout the area during the period in question, (b) the genetic composition of the pathogen and host populations has apparently not shifted to change resistance dynamics, (c) management of the system has not changed in a way that could explain the changes in disease pattern, (d) the climatic requirements of the pathogen and/or vector are well-understood and better match the climate during the period of greater disease pressure and (e) the change in disease pattern has been observed long enough to establish a convincing trend beyond possible background variation.

Even though the impact of changes in temperature, humidity and precipitation patterns has been quantified, the simulations of the potential impact of climate change remain just that, simulations. By their very nature these simulations depend on the best available projections of meteorological models.

Real evidence for the impact of climate change on plant disease could come from verification of the accuracy of these projections. This would require long-term records of disease intensity for the regions where impacts are projected and for control regions. Long-term monitoring of pathogens and other plant-associated microbes is necessary in general to understand their ecology, and to develop predictions of their impact on plant pathology [35]. The lack of availability of long-term data about disease dynamics in natural systems, and even in agricultural systems, limits opportunities for analysis of climate change effects on plant disease [36,37].

Interannual variation in climatic conditions can have important effects on disease risk. For wheat stripe rust (caused by *P. striiformis* Westend. f. sp. *tritici* Eriks.) in the US Pacific Northwest, disease severity was lower in El Niño years than in non-El Niño years [38]. If climate change alters the frequency and/or the intensity of El Niño events [39] or other extreme weather events, it will also alter patterns of disease risk; knowledge of the associations between disease and climate cycles is needed to inform predictions about plant disease epidemics under climate change [38].

Some general historical analyses of the relationship between disease and environmental factors have been developed. For example, the first annual appearance of wheat stem rust (caused by *Puccinia graminis* Pers.:Pers. f. sp. *tritici* Eriks. and E. Henn.) was compared for cool (1968–1977) and warm (1993–2002) periods in the US Great Plains, but a significant difference in arrival date was not observed [40]. In the UK, the abundance of two different wheat pathogens shifted in close correlation with patterns of SO_2 pollution during the 1900s [41,42]. For potato light blight, Zwankhuizen and Zadoks [43] have analysed epidemics in the Netherlands from 1950 to 1996 using agronomic and meteorological variables as predictors of disease severity. They found that some factors were associated with enhanced disease, such as greater numbers of days with precipitation, greater numbers of days with temperatures between 10 and 27 °C, and a relative humidity >90% during the growing season. Temperatures above 27 °C and higher levels of global radiation in the Netherlands appeared to reduce disease risk [43]. Baker et al. [44] evaluated late blight risk in central North America and found that the trends in climatic conditions should result in increased risk. Hannukkala et al. [45] evaluated late blight incidence and first appearance in Finland 1933–2002, concluding that there was higher risk in more recent years. The comparison of years is complicated in this case by changes in the pathogen population and management practices. Increases in fungicide use were consistent with increased disease risk; records of pesticide use or other management change are one potential form of evidence for climate change impacts.

Pathogens and insect pests of lodgepole pine (*Pinus contorta*) have been well-studied and offer an interesting example of a potential climate change fingerprint. Lodgepole pine is the most widely distributed pine species in natural (unmanaged) forests in western North America [46], including forests

in British Columbia where there are more than 14 million ha of lodgepole pine [47]. Due to a lack of natural or human mediated disturbances, lodgepole pine has been increasing in abundance in British Columbia since the 1900s [47,48]. Recently, there have been increased cases of decline of lodgepole pines in these forests and researchers are evaluating the potential effects of climate change on these events.

Mountain pine beetle (*Dendroctonus ponderosae*) is a bark beetle native to western North American forests [49]. This beetle can infest many pine species, and lodgepole pine is a preferred host [46,48]. The distribution range has not been limited by availability of the host but by the temperature range required for beetle survival through the winter [46,50]. The beetle causes physiological damage to the host trees by creating tunnels (insect galleries) underneath the bark, and in addition, microorganisms, such as the blue-stain fungi complex, can take advantage of these wounds to cause secondary infestation that may further reduce plant health [46,49]. Dead pines are not marketable and also can facilitate the spread of wild fire [51]. Beetle populations can be very low for many decades, but when there is an outbreak, a large area of susceptible hosts may be killed. The beetle has been known to be native to British Columbia [48], but, probably due to low winter temperatures, outbreak events were not common. However, there have been a series of outbreaks in recent years, and 8 million hectares in British Columbia were affected in 2004 [48,51]. Carroll et al. [50] evaluated the shift in infestation range and concluded that the trend toward warmer temperatures more suitable for the beetle is part of the reason for this series of outbreaks. Further, in a study by Mock et al [48], genetic markers did not reveal any significant differences among beetle genotypes from inside and outside of British Columbia, indicating the beetle population had not changed. Thus, other factors including climate change are likely to be the reason why there have been more outbreaks in northern areas.

Dothistroma needle blight is a fungal disease (causal agent *Dothistroma septosporum*) of a variety of pine species worldwide [52], including lodgepole pines. The disease is associated with mild temperature ranges (18 °C is the optimum temperature for sporulation [53]) and rain events [52,54], and causes extensive defoliation, mortality and a reduced growth rate in pine [52,55]. As with the mountain pine beetle, Dothistroma needle blight has been found in British Columbia in the past, but damage due to this disease was relatively minor. However, the number of cases and intensity of epidemics in this region has increased since the late 1990s [55]. A study by Woods et al. [55] evaluated the relationship between these disease outbreaks and (i) regional climate change and (ii) long-term climate records (utilising the Pacific Decadal Oscillation, PDO, as an indicator variable). Although they did not find a substantial increase in regional temperature nor a significant correlation between PDO and directional increase of precipitation or temperature, increased mean summer precipitation in the study area was observed. The authors also found that

in some locations, up to 40% of forest stands became dominated by lodgepole pine due to plantation development, and they hypothesised that a combination of increased rain events and the abundance of the favoured host were the probable cause of increased disease occurrence.

For both mountain pine beetle and Dothistroma needle blight, it is reasonable to assume that climate has influenced pathogen and pest behaviour; however, at the same time, there has been a substantial increase in the abundance of the host (lodgepole pine) in British Columbia [47,48]. Widely available and genetically similar hosts generally increase plant disease risk [56], and these factors may also explain at least part of the change in risk observed for lodgepole pine.

Another important disease that has exhibited recent changes in its pattern of occurrence is wheat stripe rust (or yellow rust, caused by the fungus *P. striiformis* f. sp. *tritici*). This disease decreased and then increased in importance in the US during the past century. Stripe rust was economically important in the 1930s–1960s, but the development of resistant wheat varieties successfully reduced the number of epidemic events. However, several epidemic events have been observed since 2000 [57,58]. The disease can cause 100% yield loss at a local scale [58], and epidemics in 2003 in the US resulted in losses estimated to total $300 million. Are these changes related to climate change?

Historically, *P. striiformis* f. sp. *tritici* was known to be active at relatively lower temperature ranges. Under favourable conditions (i.e. with dew or free water on plant surfaces), its spores can germinate at 0 °C [59], and the temperature range for infection was measured as between 2 and 15 °C with an optimum temperature of 7–8 °C [60,61]. And it could produce spores between 0 and 24.5 °C [59]. This pathogen species was not well adapted for higher temperature conditions and disease development declined at temperatures above 20 °C [60–62], while spores produced at 30 °C were shown to be nonviable [59].

However, more recent populations of *P. striiformis* f. sp. *tritici* were adapted to warmer temperature ranges [63]. Isolates from the 1970s to 2003 were compared, and newer (post-2000) isolates had a significantly ($P < 0.05$) higher germination rate and shorter latent period (period between infection and production of spores) than older isolates when they were incubated at 18 °C, whereas isolate effects were not different when incubation took place at 12 °C. In a follow-up study, Markell and Milus [64] examined isolates from the 1960s to 2004 with genetic markers and morphological comparisons, and found that isolates collected pre- and post-2000 could be classified into two different groups. Although within a population group less than nine polymorphic markers were identified, when pre-and post-2000 populations were compared there were 110 polymorphic markers [64]. The large difference between pre- and post-2000 groups led the authors to conclude that post-2000 isolates were introduced from outside of the US, rather than resulting from mutations in pre-2000 isolates.

Results from annual race surveys conducted by the United States Agricultural Research Service of Pullman, WA, indicated that pre-2000 isolates were not commonly collected in surveys after 2000 [64]. Thus, it seems that post-2000 isolates took the place of pre-2000 isolates. The question remains whether the success of post-2000 isolates is due to the change in climatic conditions (i.e. increase in overall temperature) or something else. Since post-2000 isolates were better adapted to a warmer temperature range, climate change might have played a role in selection for the new isolates, but there is another important factor for post-2000 isolates. All post-2000 isolates examined were able to cause disease on wheat plants with resistance genes *Yr8* and *Yr9*, while these resistance genes were effective at preventing disease for pre-2000 isolates [57,64]. There are other wheat varieties that are resistant to post-2000 isolates, but these varieties were less commonly grown since they were not effective against older isolates. Thus, the ability of new isolates to overcome these resistance genes was most likely the major factor behind the drastic change in populations of *P. striiformis* f. sp. *tritici* and recent epidemic events.

In summary, there is no doubt that plant disease responds to weather and that changes in weather events due to climate change are likely to shift the frequency and intensity of disease epidemics. Simulated climate change experiments reveal changes in plant disease intensity and the profile of plant diseases. When evidence for climate change is sought in observed changes in plant disease patterns, conclusions are less clear. Since the search for fingerprints of climate change is correlative by nature, there may always be alternative predictors for the changes, but this seems particularly true for plant disease. It is a typical biological irony that, while plant disease risk may be particularly sensitive to climatic variables and climatic shifts, plant disease may also be particularly difficult to use as an indicator of climate change because of the many interactions that take place to result in disease. However, as more data sets are collected and synthesised [37], and climate patterns exhibit greater changes over a longer period, the impacts of climate change on plant disease are likely to become clearer.

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