CHAPTER

PLANT PATHOGENS AS INDICATORS OF CLIMATE CHANGE

21

K.A. Garrett[1,2], M. Nita[3], E.D. De Wolf[2], P.D. Esker[4], L. Gomez-Montano[2], A.H. Sparks[5]

[1]*Institute for Sustainable Food Systems and Plant Pathology Department, University of Florida, Gainesville, FL, USA;* [2]*Department of Plant Pathology, Kansas State University, Manhattan, KS, USA;* [3]*Department of Plant Pathology, Physiology, and Weed Science, AHS Jr. AREC, Virginia Polytechnic Institute and State University, Winchester, VA, USA;* [4]*Escuela de Agronomía, Universidad de Costa Rica, San Pedro Montes de Oca, San José, Costa Rica;* [5]*International Rice Research Institute (IRRI), Los Baños, Laguna, Philippines*

CHAPTER OUTLINE

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, and 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 potential evidence of climate change fingerprints.

The battle to protect plant health is ongoing, and plant disease management is essential for our continued ability to feed a growing human population. The Great Famine in Ireland is one striking example of the impact of plant disease: in 1846–1851 around 1 million Irish people died during an extremely destructive epidemic of potato late blight [2]. Plant diseases continue to cause serious problems in global food production. Approximately 800 million people do not have adequate food and 10%–16% of global food production is lost to plant disease [3,4]. Not only does plant disease affect

Climate Change. http://dx.doi.org/10.1016/B978-0-444-63524-2.00021-X

human food production but it also impacts natural systems [5]. 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 dominate tree species and triggered major impacts on forest systems [6]. Furthermore, the genetic diversity of many plant pathogens greatly increases the risk of a large-scale epidemic, as evidenced by the threat posed by the emergence of race Ug99 of the wheat stem rust fungus [7].

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, each with potentially different mechanisms of response under climate change. As such there is great need for more quantitative information related to each type of important disease [8]. For example, viruses may be present in hosts while symptom expression is dependent on temperature [9]; thus, even the difficulty of detection of pathogens varies with climate. Fungal pathogens are often strongly dependent on humidity or dew [10], so changes in these environmental factors are likely to shift disease risk. Managing plant pathogens is also made more complicated due to genetic variation within pathogen populations [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 [11]. Many plant pathogens have a high reproduction potential and pathogen populations may increase rapidly when weather conditions are favourable for disease development [12,13]. The 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. There is also a complex set of interactions among microorganisms, especially those that make up soil biodiversity [14–16], making it challenging to define the temporal and spatial scales required to adequately study disease responses to climate [17].

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 [18]. This has motivated a large body of research addressing the effects of weather or climate on plant disease [18,19]. 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 [12,13]. All three 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 each reduce disease intensity. The synchronous interaction among host, pathogen and environment governs disease development. These interactions can be conceptualized 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 realized the importance of the disease cycle, its component events and the apparent relationships with environment, the quantification of these interactions did not begin in earnest until the 1950s [18]. The following decades of research have 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 [18].

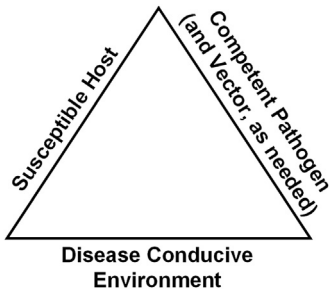

FIGURE 1

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.

The quantification of the relationship between the disease cycle of a given plant disease and weather is also the foundation of many predictive 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 fertilization or irrigation. A prediction of 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. Refs [20,21]).

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. [22] have used models of the impact of weather variables on the risk of infection by the oomycete *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 or adapted for application in climate change scenario analyses (e.g. Refs [23,24]).

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) [10] 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 maximize their chance of infection, the life cycle of pathogen populations must be in sync with host development. Here we discuss a few examples where host phenology is key for disease development.

Some pathogens depend on flower tissues as a point of entry to the host. For example, the fungus *Botrytis cinerea*, which causes grey mould of strawberry and other fruits (producing a grey fuzz-balled strawberry, which you may have seen at a grocery store or in your refrigerator), infects the strawberry at the time of flowering [25]. The fungus stays in flower parts until the sugar level of the berry

increases, and then causes grey mould disease. Another example is Fusarium head or ear blight, also called scab of wheat and barley, which causes large yield losses, reductions in grain quality and contamination with mycotoxins [26,27]. Mycotoxins are toxic substances produced by the fungi, which can be more important than simple yield reductions, such that climate change effects on mycotoxin production are an important concern in themselves [28]. Several fungal species cause Fusarium head blight, where *Fusarium graminearum* (teleomorph: *Gibberella zeae*) is responsible for the most aggressive form. The anthesis (flowering) period seems to be the critical time for infection [27,29]. An important bacterial disease of apple and pears, called fire blight, also utilizes flowers as a major point of entry [30]. The causal agent (*Erwinia amylovora*) can be disseminated by pollinating insects such as bees and moves into the tree through flowers, causing 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* [31] and stripe rust (caused by *Puccinia striiformis* f. sp. *tritici*) resistance gene *Yr39* [32]. 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* (causing powdery mildew) [33], or *Guignardia bidwellii* (causing black rot) [34] or the oomycete pathogen *Plasmopara viticola* (causing downy mildew) [35] are less successful at infecting plants.

Host development patterns may be altered with changes in climate. 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 while 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 utilized along with inoculum production [36] because tissues become less susceptible as the rate of tissue expansion decreases.

Pathogen dispersal is another aspect of epidemiology that can be influenced by climate change. Pathogens such as rust fungi often overwinter in warmer regions and migrate annually via wind to cooler regions during crop production [37,38]. Pathogens and other microbes can be spread in dust storms [39]. As climate shifts, so may the patterns of wind dispersal of pathogens. For pathogens dispersed by insects, new patterns of insect movement and encounters with potential new vectors as ranges shift may also alter epidemics [40]. Combined with potential changes in cropland area due to climate and other global change factors, the resulting new patterns of invasion, reinvasion and saturation may result in epidemic evolution with new patterns of risk [41,42]. The ability of managers to track geographically the new management requirements will be a key issue in determining disease outcomes because there are important lags in responding to new needs through methods such as crop breeding for resistance, development of new pesticides and simply communicating effectively about management [43–50].

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).

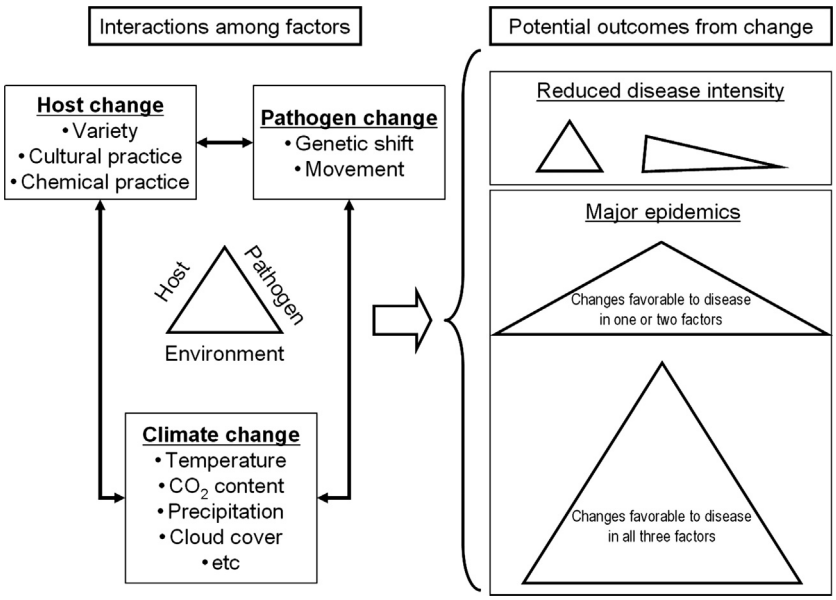

FIGURE 2

Interactions among components of the disease triangle and potential outcomes. Amount of disease (incidence, severity, etc.) or 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.

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 [51]. 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 [52]. 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 hybridize to produce new pathogens [53,54].

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 successfully represent future climate

scenarios, plant disease will respond. The second type of evidence is for changes in observed patterns of plant disease in agricultural or wild-land 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 characterized in terms of the scale of the effect being considered [55,56]. For many well-studied pathogens and vectors, the temperature ranges that support single infection events or survival are fairly well characterized. 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 for studying transcriptomes make it possible to study weather effects on plant gene expression in the field, including genes that may be important for disease resistance. Drawing conclusions about larger-scale processes from plot-level experiments may be challenging, however, since additional forms of interactions are important at larger scales [44].

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 meadows Roy et al. [57] 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 for a number of plant diseases, but results can be challenging to categorize [58–60]. Compared to studies in experimental chambers, free-air CO_2 enrichment (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 [61], so the outcome for any given host–pathogen interaction is not readily predictable a priori. Elevated ozone levels can also affect plant disease risk (reviewed in Chakraborty et al. [59]).

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 through new experiments and new high-throughput sequencing approaches [14,15,62], 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?

The number of factors that interact to result in plant disease complicates such an analysis [17]. 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 patterns, the argument for climate change as an important driver is strongest when (1) the pathogen is known to have been present throughout the area during the period in question, (2) the genetic composition of the pathogen and host populations has apparently not shifted to change resistance dynamics, (3) management of the system has not changed in a way that could explain the changes in disease pattern, (4) 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 (5) 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, scenario analyses of the potential impact of climate change are limited for many diseases by the lack of needed data and models. Real evidence for the impact of climate change on plant disease could come from verification of the accuracy of scenario analyses. This would require long-term records of disease intensity for the regions projected to be impacted and 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 [63]. 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 [64,65]. New analyses, using databases indicating where diseases have been observed [66], provide stimulating new assessments of potential poleward movement of diseases and insect pests in recent decades [67]. Use of these types of data and other crowd-sourcing approaches opens many new possibilities for analysis but also requires grappling with the limits to interpretation when there are generally not observations of absence and there are different sampling approaches in different regions [68–71].

Interannual variation in climatic conditions can have important effects on disease risk. For wheat stripe rust (caused by *Puccinia 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 [72]. If climate change alters the frequency and/or the intensity of El Niño events [73] 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 [72]. The effects of patterns of weather fluctuation and extremes may produce new disease risk scenarios, including new management challenges [74,75].

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. & 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 [76]. In the UK, the abundance of two different wheat pathogens shifted in close correlation

with patterns of SO_2 pollution during the 1900s [48,77,78]. For potato light blight, Zwankhuizen and Zadoks [79] have analyzed 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°C 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 [79]. Baker et al. [80] evaluated late blight risk in central North America and found that the trends in climatic conditions should result in increased risk. Hannukkala et al. [81] 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. Diplodia (or Sphaeropsis) shoot blight of pines emerged in France, with epidemics probably driven in part by more frequent conducive temperature and precipitation in recent years [82].

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 [83], including forests in British Columbia with more than 14 million ha of lodgepole pine [84]. Due to a lack of natural or human mediated disturbances, lodgepole pine has been increasing in abundance in British Columbia since the 1900s [84,85]. 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 and native to western North American forests [86]. This beetle can infest many pine species, and lodgepole pine is a preferred host [83,85]. The distribution range has not been limited by availability of the host but by the temperature range required for beetle survival through the winter [83,87]. The beetle causes physiological damage on 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 [83,86]. Dead pines are not marketable and also can facilitate the spread of wild fire [88]. 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 [85], but, probably due to low winter temperatures, outbreak events were not common. However, the frequency of outbreaks appeared to be increasing, and 8 million hectares in British Columbia were affected in 2004 [85,88]. Carroll et al. [87] evaluated the shift in infestation range and concluded that the trend toward warmer temperatures more suitable for the beetle was part of the reason for this series of outbreaks. Further, in a study by Mock et al. [85], 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 [89], including lodgepole pines. The disease is associated with mild temperature ranges (18°C is the optimum temperature for sporulation [90]) and rain events with 10 or

more hours of wetness [89,91]. It causes extensive defoliation, mortality and a reduced growth rate in pine [89,92]. 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 [92]. A study by Woods et al. [92] evaluated the relationship between these disease outbreaks and (1) regional climate change and (2) long-term climate records (utilizing 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. Furthermore, a recent study by Welsh et al. [93] indicated that trends in minimum temperatures in August and increasing precipitation in April could be linked to the spread of the disease in British Columbia. On the other hand, in some locations, up to 40% of forest stands became dominated by lodgepole pine due to plantation development [92]. Thus, a combination of increased rain events and the abundance of the favoured host was the likely cause of increased disease occurrence.

More cases of Dothistroma needle blight have been reported in European countries as well [94,95]. However, when pathogen genetic diversity in Estonia, Finland and the Czech Republic was evaluated [96], similarly high levels of genetic diversity were found in all three populations. Therefore, there was no strong evidence of northward introduction of the pathogen. Since Dothistroma needle blight was present in Russia in the 1950s [97], and was considered to be a minor disease in France in the 1960s–1980s, this pathogen could have been present in these counties for a long time prior to recent outbreaks in the 1990s [94], indicating more evidence for a role of climate change.

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 [84,85]. Widely available and genetically similar hosts often increase plant disease risk, and these factors may also explain at least part of the change in risk observed for lodgepole pine. Also, the rapid expansion of a host may encourage the movement of nursery trees without obvious symptoms, even those potentially infected. Movement of planting materials may help to explain the high genetic diversity among isolates from European countries [96].

Another important disease that has exhibited recent changes in its pattern of occurrence is wheat stripe rust (or yellow rust, caused by the fungus *Puccinia 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 [98,99]. The disease can cause 100% yield loss at a local scale [99], 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 [100], and the temperature range for infection was measured as between 2°C and 15°C with an optimum temperature of 7°C to 8°C [101,102]. Spores could be produced between 0°C and 24.5°C [100]. This pathogen species was not well adapted for higher temperature conditions, and disease development declined at temperatures above 20°C [101–103], while spores produced at 30°C were shown to be nonviable [100].

However, more recent populations of *P. striiformis* f. sp. *tritici* were adapted to warmer temperature ranges [104]. Isolates from the 1970s to 2003 and newer (post-2000) isolates had a significantly 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 [105] 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 [105]. 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 in Pullman, WA, indicated that pre-2000 isolates were not commonly collected in surveys after 2000 [105]. Thus, it seems that post-2000 isolates took the place of pre-2000 isolate types. 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 virulent (able to cause disease) on wheat plants with resistance genes *Yr8* and *Yr9*, while these resistance genes worked very well against pre-2000 isolates [98,105]. 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.

Pierce's disease (PD) of grape is caused by a gram-negative, xylem-limited, fastidious bacterium, *Xylella fastidiosa* (Wells) [106,107]. *X. fastidiosa* is vectored by sharpshooter leafhoppers (Hemiptera: Cicadellidae), such as glassy-winged sharpshooter, *Homalodisca vitripennis* (Germar), which has been identified as a major PD vector in California and southeastern regions of the US [108–111]. PD symptoms include interveinal chlorosis, marginal necrosis, uneven lignification of shoots or 'green islands', and leaf abscission that results in characteristic 'matchstick petioles' [112]. Diseased vines suffer decline, yield loss, and even death. With severe disease on a susceptible cultivar, a vine will be killed within a few years [109].

The geographic distribution of PD is concentrated in California and spans from Texas and Florida up to Virginia (VA) in the US [113]. Anas et al. found that presence of PD was limited by daily minimum temperatures below −9.4°C for four or more days or −12.2°C for two days [114] because low temperatures can reduce or kill bacterial populations in the vine [115]. Therefore, areas north of central VA had not been considered a high-risk area for PD when risk was based on the 25-year average between 1972 and 1997 [114,116]. However, when the data from 1997 to 2005 were used, the very high-risk area moved northward considerably to include almost the entire state of VA. The same group conducted a survey in North Carolina and Georgia, and PD was identified in 82% and 75% of surveyed vineyards, respectively. Moreover, a 2006 VA vineyard survey found 70% of surveyed vines PD-positive [117]. However, since the recent reintroduction of wine grapes in the Eastern US resulted in rapid expansion of vineyards since the 1980s, it is not clear to what extent the trend of northward disease detection is due to climate-facilitated disease spread versus simply coinciding with the northward expansion of vineyards.

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 is sought for climate change based on 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 synthesized [65,67], and climate patterns exhibit greater changes over a longer period, the impacts of climate change on plant disease are likely to become clearer.

ACKNOWLEDGEMENTS

We appreciate support by the CGIAR Consortium Research Program for Roots, Tubers and Bananas (RTB) and the CGIAR Research Program on Climate Change, Agriculture and Food Security (CCAFS), by USDA NC RIPM Grant 2010-34103-20964 and USDA APHIS Grant 11-8453-1483-CA, by the US National Science Foundation (NSF) through Grant DEB-0516046 and NSF Grant EF-0525712 as part of the joint NSF-National Institutes of Health (NIH) Ecology of Infectious Disease program, by the US Agency for International Development (USAID) to the OIRED at Virginia Tech for the SANREM CRSP under Award No. EPP-A-00-04-00013-00 and for the IPM CRSP under Award No. EPP-A-00-04-00016-00, and by the Kansas Agricultural Experiment Station. The views expressed herein can in no way be taken to reflect the official opinion of these agencies.

REFERENCES

[1] E.D. De Wolf, S.A. Isard, Annu. Rev. Phytopathol. 45 (2007) 203–220.
[2] C. Kinealy, A Death-Dealing Famine: The Great Hunger in Ireland, Pluto Press, 1997.
[3] R.N. Strange, P.R. Scott, Annu. Rev. Phytopathol. 43 (2005) 83–116.
[4] S. Chakraborty, A.C. Newton, Plant Pathol. 60 (2011) 2–14.
[5] J.J. Burdon, P.H. Thrall, L. Ericson, Annu. Rev. Phytopathol. 44 (2006) 19–39.
[6] T. Emiko Condeso, R.K. Meentemeyer, J. Ecol. 95 (2007) 364–375.
[7] R.P. Singh, D.P. Hodson, J. Huerta-Espino, Y. Jin, S. Bhavani, P. Njau, S. Herrera-Foessel, P.K. Singh, S. Singh, V. Govindan, Annu. Rev. Phytopathol. 49 (2011) 465–481.
[8] J. Luck, M. Spackman, A. Freeman, P. Trebicki, W. Griffiths, K. Finlay, S. Chakraborty, Plant Pathol. 60 (2011) 113–121.
[9] J. DeBokx, P. Piron, Potato Res. 20 (1977) 207–213.
[10] L. Huber, T.J. Gllespie, Annu. Rev. Phytopathol. 30 (1992) 553–577.
[11] B.A. McDonald, C. Linde, Ann. Rev. Phytopathol. 40 (2002) 349–379.
[12] G.N. Agrios, Plant Pathology, Academic Press, San Diego, 2005.
[13] J.E. Van Der Plank, Plant Diseases: Epidemics and Control, Academic Press, New York and London, 1963.
[14] K.A. Garrett, A. Jumpponen, C. Toomajian, L. Gomez-Montano, Can. J. Plant Pathol. 34 (2012) 349–361.
[15] S. Chakraborty, I.B. Pangga, M.M. Roper, Global Change Biol. 18 (2012) 2111–2125.
[16] S.G. Pritchard, Plant Pathol. 60 (2011) 82–99.
[17] K.A. Garrett, G.A. Forbes, S. Savary, P. Skelsey, A.H. Sparks, C. Valdivia, A.H.C. van Bruggen, L. Willocquet, A. Djurle, E. Duveiller, H. Eckersten, S. Pande, C.V. Cruz, J. Yuen, Plant Pathol. 60 (2011) 15–30.

[18] C.L. Campbell, L.V. Madden, Introduction to Plant Disease Epidemiology, John Wiley & Sons, NewYork, 1990.
[19] L.V. Madden, G. Hughes, F. van den Bosch, The Study of Plant Disease Epidemics, APS press, St. Paul, MN, 2007.
[20] R.J. Hijmans, G.A. Forbes, T.S. Walker, Plant Pathol. 49 (2000) 697–705.
[21] A.H. Sparks, G.A. Forbes, R.J. Hijmans, K.A. Garrett, Global Change Biol. 20 (2014) 3621–3631.
[22] M. Bergot, E. Cloppet, V. Pearnaud, M. Deque, B. Marcais, M.-L. Desprez-Loustau, Global Change Biol. 10 (2004) 1539–1552.
[23] A.H. Sparks, G.A. Forbes, R.J. Hijmans, K.A. Garrett, Ecosphere 2 (2011) art90.
[24] R. Ghini, W. Bettiol, E. Hamada, Plant Pathol. 60 (2011) 122–132.
[25] D.R. Cooley, W.F. Wilcox, J. Kovach, S.G. Schloemann, Plant Dis. 80 (1996) 228–237.
[26] M. McMullen, R. Jones, D. Gallenberg, Plant Dis. 81 (1997) 1340–1348.
[27] J.C. Sutton, Can. J. Plant Pathol. 4 (1982) 195–209.
[28] N. Magan, A. Medina, D. Aldred, Plant Pathol. 60 (2011) 150–163.
[29] A.L. Anderson, Phytopathology 38 (1948) 595–611.
[30] S.V. Beer, D.C. Opgenorth, Phytopathology 66 (1976) 317–322.
[31] J.A. Kolmer, Annu. Rev. Phytopathol. 34 (1996) 435–455.
[32] F. Lin, X. Chen, Theor. Appl. Genet. 114 (2007) 1277–1287.
[33] A. Ficke, D.M. Gadoury, R.C. Seem, Phytopathology 92 (2002) 671–675.
[34] L.E. Hoffman, W.F. Wilcox, D.M. Gadoury, R.C. Seem, D.G. Riegel, Phytopathology 94 (2004) 641–650.
[35] M.M. Kennelly, D.M. Gadoury, W.F. Wilcox, P.A. Magarey, R.C. Seem, Phytopathology 95 (2005) 1445–1452.
[36] D.M. Gadoury, R.C. Seem, A. Stensvand, New York Fruit Quarterly 2 (1995) 5–8.
[37] X. Li, P.D. Esker, Z. Pan, A.P. Dias, L. Xue, X.B. Yang, Plant Dis. 94 (2010) 796–806.
[38] J.K.M. Brown, M.S. Hovmoller, Science 297 (2002) 537–541.
[39] C. Gonzalez-Martin, N. Teigell-Perez, B. Valladares, D.W. Griffin, The global dispersion of pathogenic microorganisms by dust storms and its relevance to agriculture, in: S. Donald (Ed.), Advances in Agronomy, Academic Press, 2014, pp. 1–41.
[40] R.A.C. Jones, M.J. Barbetti, CAB Rev. 7 (2012) 1–33.
[41] K.A. Garrett, S. Thomas-Sharma, G.A. Forbes, J. Hernandez Nopsa, Climate change and plant pathogen invasions, in: L. Ziska, J. Dukes (Eds.), Invasive Species and Climate Change, CABI Publishing, 2014, pp. 22–44.
[42] M.R. Sanatkar, C. Scoglio, B. Natarajan, S. Isard, K.A. Garrett, Phytopathology 105 (2015) 947–955.
[43] M. Pautasso, T.F. Doring, M. Garbelotto, L. Pellis, M.J. Jeger, Eur. J. Plant Pathol. 133 (2012) 295–313.
[44] M. Pautasso, K. Dehnen-Schmutz, O. Holdenrieder, S. Pietravalle, N. Salama, M.J. Jeger, E. Lange, S. Hehl-Lange, Biol. Rev. 85 (2010) 729–755.
[45] K.A. Garrett, Eur. J. Plant Pathol. 133 (2012) 75–88.
[46] M.W. Shaw, T.M. Osborne, Plant Pathol. 60 (2011) 31–43.
[47] R.N. Sturrock, S.J. Frankel, A.V. Brown, P.E. Hennon, J.T. Kliejunas, K.J. Lewis, J.J. Worrall, A.J. Woods, Plant Pathol. 60 (2011) 133–149.
[48] B.D.L. Fitt, B.A. Fraaije, P. Chandramohan, M.W. Shaw, Plant Pathol. 60 (2011) 44–53.
[49] S. Savary, A. Nelson, A.H. Sparks, L. Willocquet, E. Duveiller, G. Mahuku, G.A. Forbes, K.A. Garrett, D. Hodson, J. Padgham, S. Pande, M. Sharma, J. Yuen, A. Djurle, Plant Dis. 95 (2011) 1204–1216.
[50] R.R. McAllister, C.J. Robinson, K. Maclean, A.M. Guerrero, K. Collins, B.M. Taylor, P.J. De Barro, Ecol. Soc. 20 (2015) 67.
[51] K.A. Garrett, Climate Change and Plant Disease Risk, Global Climate Change and Extreme Weather Events: Understanding the Contributions to Infectious Disease Emergence, National Academies Press, Washington, DC, 2008, 143–155.

[52] K.A. Garrett, R.L. Bowden, Phytopathology 92 (2002) 1152–1159.
[53] P.K. Anderson, A.A. Cunningham, N.G. Patel, F.J. Morales, P.R. Epstein, P. Daszak, Trends Ecol. Evol. 19 (2004) 535–544.
[54] C.M. Brasier, BioScience 51 (2001) 123–133.
[55] K.A. Garrett, S.P. Dendy, E.E. Frank, M.N. Rouse, S.E. Travers, Annu. Rev. Phytopathol. 44 (2006) 489–509.
[56] S.M. Coakley, H. Scherm, S. Chakraborty, Annu. Rev. Phytopathol. 37 (1999) 399–426.
[57] B.A. Roy, S. Gusewell, J. Harte, Ecology 85 (2004) 2570–2580.
[58] D.M. Eastburn, A.J. McElrone, D.D. Bilgin, Plant Pathol. 60 (2011) 54–69.
[59] S. Chakraborty, J. Luck, G. Hollaway, A. Freeman, R. Norton, K.A. Garrett, K. Percy, A. Hopkins, C. Davis, D.F. Karnosky, CAB Rev. 3 (2008).
[60] I.B. Pangga, J. Hanan, S. Chakraborty, Plant Pathol. 60 (2011) 70–81.
[61] S. Chakraborty, Australas. Plant Pathol. 34 (2005) 443–448.
[62] M.P. Waldrop, M.K. Firestone, Microb. Ecol. 52 (2006) 716–724.
[63] C.D. Harvell, C.E. Mitchell, J.R. Ward, S. Altizer, A.P. Dobson, R.S. Ostfeld, M.D. Samuel, Science 296 (2002) 2158–2162.
[64] H. Scherm, Can. J. Plant Pathol. 26 (2004) 267–273.
[65] M.J. Jeger, M. Pautasso, New Phytol. 177 (2008) 8–11.
[66] N.M. Pasiecznik, I.M. Smith, G.W. Watson, A.A. Brunt, B. Ritchie, L.M.F. Charles, EPPO Bull. 35 (2005) 1–7.
[67] D.P. Bebber, M.A.T. Ramotowski, S.J. Gurr, Nat. Clim. Change 3 (2013) 985–988.
[68] H. Scherm, C.S. Thomas, K.A. Garrett, J.M. Olsen, Annu. Rev. Phytopathol. 52 (2014) 453–476.
[69] D.P. Bebber, T. Holmes, D. Smith, S.J. Gurr, New Phytol. 202 (2014) 901–910.
[70] K.A. Garrett, Nat. Clim. Change 3 (2013) 955–957.
[71] J.R. Rohr, A.P. Dobson, P.T.J. Johnson, A.M. Kilpatrick, S.H. Paull, T.R. Raffel, D. Ruiz-Moreno, M.B. Thomas, Trends Ecol. Evol. 26 (2011) 270–277.
[72] H. Scherm, X.B. Yang, Int. J. Biometeorol. 42 (1995) 28–33.
[73] A. Timmermann, J. Oberhuber, A. Bacher, M. Esch, M. Latif, E. Roeckner, Nature 398 (1999) 694–697.
[74] K.A. Garrett, A.D.M. Dobson, J. Kroschel, B. Natarajan, S. Orlandini, H.E.Z. Tonnang, C. Valdivia, Agric. For. Meteorol. 170 (2013) 216–227.
[75] C. Rosenzweig, A. Iglesias, X.B. Yang, P.R. Epstein, E. Chivian, Global Change Hum. Health 2 (2001) 90–104.
[76] H. Scherm, S.M. Coakley, Australas. Plant Pathol. 32 (2003) 157–165.
[77] S.J. Bearchell, B.A. Fraaije, M.W. Shaw, B.D.L. Fitt, Proc. Natl. Acad. Sci. U.S.A. 102 (2005) 5438–5442.
[78] M.W. Shaw, S.J. Bearchell, B.D.L. Fitt, B.A. Fraaije, New Phytol. 177 (2008) 229–238.
[79] M.J. Zwankhuizen, J.C. Zadoks, Plant Pathol. 51 (2002) 413–423.
[80] K.B. Baker, W.W. Kirk, J.M. Stein, J.A. Anderson, HortTechnology 15 (2005) 510–518.
[81] A.O. Hannukkala, T. Kaukoranta, A. Lehtinen, A. Rahkonen, Plant Pathol. 56 (2007) 167–176.
[82] B. Fabre, D. Piou, M.-L. Desprez-Loustau, B. Marçais, Global Change Biol. 17 (2011) 3218–3227.
[83] G.D. Amman, The role of the mountain pine beetle in lodgepole pine ecosystem: impact on succession, in: W.J. Wattson (Ed.), The Role of Arthropods in Forest Ecosystems, Springer-Verlag, New York, 1978.
[84] S.W. Taylor, A.L. Carroll, Disturbance, forest age, and mountain pine beetle outbreak dynamics in BC: a historicalperspective, in: T.L. Shore, J.E. Brooks, J.E. Stone (Eds.), Mountain Pine Beetle Symposium: Challenges and Solutions. (October 30–31, 2003), Natural Resources Canada, Canadian Forest Service, Pacific Forestry Centre, Information Report BC-X-399, Victoria, BC, Kelowna, British Columbia, 2004, pp. 41–51.
[85] K.E. Mock, B.J. Bentz, E.M. O'Neill, J.P. Chong, J. Orwin, M.E. Pfrender, Mol. Ecol. 16 (2007) 553–568.

[86] W. Cranshaw, D. Leatherman, B. Jacobi, L. Mannex, Insects and Diseases of Woody Plants in the Central Rockies, Colorado State University Bulletin no. 506A, Colorado State University Cooperative Extension, Fort Collins, CO, 2000.
[87] A.L. Carroll, S.W. Taylor, J. Régnière, L. Safranyik, Effects of climate change on range expansion by the mountain pine beetle in British Columbia, in: T.L. Shore, J.E. Brooks, J.E. Stone (Eds.), Mountain Pine Beetle Symposium: Challenges and Solutions. (October 30–31, 2003), Natural Resources Canada, Canadian Forest Service, Pacific Forestry Centre, Information Report BC-X-399, Victoria, BC, Kelowna, British Columbia, 2004, pp. 223–232.
[88] Mountain pine beetle action plan 2006–2011. Ministry of forests and range – province of British Columbia.
[89] I.A.S. Gibson, Annu. Rev. Phytopathol. 10 (1972) 51–72.
[90] M.H. Ivory, Trans. Brit. Mycol. Soc 50 (1867) 563–572.
[91] D. Hocking, D.E. Etheridge, Ann. Appl. Biol. 59 (1967) 133–141.
[92] A. Woods, K.D. Coates, A. Hamann, BioScience 55 (2005) 761–769.
[93] C. Welsh, K.J. Lewis, A.J. Woods, Can. J. For. Res. 44 (2014) 212–219.
[94] D. Villebonne, F. Maugard, Rapid development of Dothistroma needle blight (*Scirrhia pini*) on Corsican pine (*Pinus nigra* subsp. *laricio*) in France, Le Département de la Santé des Forêts, Paris, France, 1999.
[95] J.A. Nowakowska, A. Tereba, T. Oszako, Folia For. Pol. Ser. A For. 56 (3) (2014) 157–159.
[96] R. Drenkhan, J. Hantula, M. Vuorinen, L. Jankovský, M.M. Müller, Eur. J. Plant Pathol. 136 (2013) 71–85.
[97] J.S. Murray, S. Batko, Forestry 35 (1962) 57–65.
[98] X. Chen, M. Moore, E.A. Milus, D.L. Long, R.F. Line, D. Marshall, L. Jackson, Plant Dis. 86 (2002) 39–46.
[99] X.M. Chen, Can. J. Plant Pathol. 27 (2005) 314–337.
[100] H. Tollenaar, B.R. Houston, Phytopathology 56 (1965) 787–790.
[101] E.L. Sharp, Phytopathology 55 (1965) 198–203.
[102] C. de Vallavieille-Pope, L. Huber, M. Leconte, H. Goyeau, Phytopathology 85 (1995) 409–415.
[103] M.V. Wiese (Ed.), Compendium of Wheat Diseases, APS Press, St. Paul, MN, 1987.
[104] E.A. Milus, E. Seyran, R. McNew, Plant Dis. 90 (2006) 847–852.
[105] S.G. Markell, E.A. Milus, Phytopathology 98 (2008) 632–639.
[106] J. Wells, B. Raju, H. Hung, W. Weisburg, L. Mandelco-Paul, D. Brenner, Int. J. Syst. Bacteriol. 37 (1987) 136–143.
[107] S. Chatterjee, R. Almeida, S. Lindow, Annu. Rev. Phytopathol. 46 (2008) 243–271.
[108] R. Hernandez-Martinez, K.A. de la Cerda, H.S. Costa, D.A. Cooksey, F.P. Wong, Phytopathology 97 (2007) 857–864.
[109] D.L. Hopkins, A.H. Purcell, Plant Dis. 86 (2002) 1056–1066.
[110] J.T. Sorensen, R.J. Gill, Pan-Pac. Entomol. 72 (1996) 160–161.
[111] W.C. Adlerz, D.L. Hopkins, J. Econ. Entomol. 72 (1979) 916–919.
[112] R.C. Pearson, A.C. Goheen, Compendium of Grape Diseases, American Phytopathological Society, St. Paul, Minnesota, 1988.
[113] R.P.P. Almeida, F.E. Nascimento, J. Chau, S.S. Prado, C.-W. Tsai, S.A. Lopes, J.R.S. Lopes, Appl. Environ. Microbiol. 74 (2008) 3690–3701.
[114] O. Anas, U.J. Harrison, P.M. Brannen, T.B. Sutton, Plant Health Prog. (2008), http://dx.doi.org/10.1094/PHP-2008-0718-01-RS.
[115] J.H. Lieth, M.M. Meyer, K.H. Yeo, B.C. Kirkpatrick, Phytopathology 101 (2011) 1492–1500.
[116] M.S. Hoddle, Crop Prot. 23 (2004) 691–699.
[117] A.K. Wallingford, S.A. Tolin, A.L. Myers, T.K. Wolf, D.G. Pfeiffer, Plant Health Prog. (2007), http://dx.doi.org/10.1094/PHP-2007-1004-01-BR.