Preparing for Changes in Plant Disease Due to Climate Change

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Abstract: Climate change will change patterns of disease through changes in host distribution and phenology, changes in plant-associated microflora and direct biological effects on rapidly evolving pathogens. Short-term forecast models coupled with weather generated from climate simulations may be a basis for projection; however, they will often fail to capture long-term trends effectively. Verification of predictions is a major difficulty; the most convincing method would be to "back-forecast" observed historical changes. Unfortunately, we lack of empirical data over long time-spans; most of what is known concerns invasions, in which climate is not the main driving factor. In one case where long-term prevalence can be deduced, climate had little to do with change. Resilience to surprises should be the most important policy aim.

Keywords: climate change; population dynamics; plant disease forecasting; modelling; mutation

Background

Whatever humans do, plant diseases will change. New crop diseases will appear; old ones will change their relative importance, evolve to overcome host resistance and fungicides, or inexplicably cease to be a problem. There is no doubt that even if climate remained static this would still be true, and many serious and unexpected changes would occur. So in asking about the consequences of climate change for plant disease, we should be asking about system properties rather than specific predictions. We may reasonably ask "will the rate at which diseases acquire the ability to infect new hosts change in the new climate we anticipate?", but it is probably foolish (and hard!) to ask "will *Puccinia coronata* jump to infect wheat?" In this paper, I try to discuss the problems of trying to predict these changes in system properties, and the intermediate problem of trying to predict changes in disease if only climate and host distribution changed. The paper is not a review, and examples are cited to make specific points, not in any way as a comprehensive survey; inevitably, therefore, many valuable contributions to the literature are not cited.

In discussing the consequences of climate change, we are talking about consequences of subtle trends associated with a particular time-scale. More concretely, the temperature within a day could vary by 20°C, and the average temperature between seasons in a continental climate by 30°C, yet the concern over climate change is about decadal averages varying by as little as 2°C; or more accurately, over the distributional changes in short-term weather with quite slight changes in mean values (e.g. Scherm 1994). So the question of how these changes will affect plant disease is one about how to translate predictions of this physical time-series into predictions about one component of an ecosystem. The task is made more difficult because the organisms we are interested in are not the dominant biomass component of the ecosystem and are profoundly dependent on other organisms, small and large. At least in the short term, the problem is also made more difficult because the life-cycles of fungi and bacteria are intimately related to precipitation. Precipitation patterns are both harder to model

and harder to verify, so that the most critical aspect of climate prediction is also one of the least reliable (Solomon *et al.* 2007, Chapter 8).

The community of species within an ecosystem is determined by soil, climate, management and the accidents of history and evolution. Within this context, plant pathogens are one of the more visible microbial components, and crops one of the more intensively managed components. Like any other species, a plant pathogen, simply because it is a current component of an ecosystem, can be deduced to have a balance between periods of population increase and decrease that is sufficiently stable to avoid extinction. Infection by an individual cohort of propagules is likely to be influenced by weather conditions which are transitory on scales of hours. However, these weather fluctuations will not translate into similar fluctuations in population, because they will be smoothed by the spread in time-scale of the population processes within the population, which will be of the order of the generation time. An added level of complexity is introduced by the need to consider the time-scale of the host population dynamics. This is often substantially longer than the time-scale associated with the pathogen, which therefore tracks the host dynamics; in this case smoothing will typically be on the time-scale set by the host. Typical asexual generational time-scales will be days to weeks; sexual time-scales of the order of years. Evolutionary changes depend on the strength of the selective force operating: for breakdown of a major-gene resistance the time-scale is a few years, while for changes in response to slighter fitness differences, the time-scales can be anything up to the point where chance takes over (that is, when selective changes are much smaller than chance changes, change due to selection becomes unpredictable. Chance processes will dominate if selective changes are weaker than about $1\sqrt{N_e}$, where N_e is the effective population size-likely to be roughly the size of the population in the off-season. This is associated with a time-scale of about $\sqrt{N_e}$). Because the generational time-scales are quite short, and the reproductive rate of a pathogen may be greatly influenced by weather on time-scales of hours, rapid natural selective changes must be the norm in pathogen populations, if variability is present. However, the direction of the selective forces acting will change frequently and rapidly, so that, as with population dynamics, long-term trends are the average around a very noisy mean. We can see a time-series of abundance of disease as the outcome of a filter acting on the physical and biological time-series underlying it, the output of the filter being the disease time-series (Table 1, Figure. 1).

Table 1. Time-scales associated with various sources of change in plant disease

Source	Time-scale	Evidence
Generation time	days-seasons	basic epidemiology: eg at minimum potato blight 4–7 days; probably no upper limit other than host lifetime
Invasion of a new avr type	season	breakdowns of rust resistance in wheat, blight resistance in potato
Waiting time for successful mutation in average genotype	5 seasons	boom-bust cycles in lettuce, wheat, potato
Equilibration of a soil or seed-borne disease following crop range expansion or change in agronomy	5 seasons	take-all decline; increases in trash disease after introducing minimal cultivation
Spatial expansion over a new range (including jumps)	decade	sigatoka, soy-bean rust, coffee rust, rhizomania
Change in range of host due to shifting climatic limits	multi-decade	predicted rate of climate change (0.2–0.4°C/decade)
Change in disease dominance due to changing probability distribution of weather alone	multi-decade	predicted rate of climate change
Qualitative host-shift	millennia?	depth of branching phylogenetic trees; but there are tens of thousands of sources so the rate observed looking at all crops together may be important

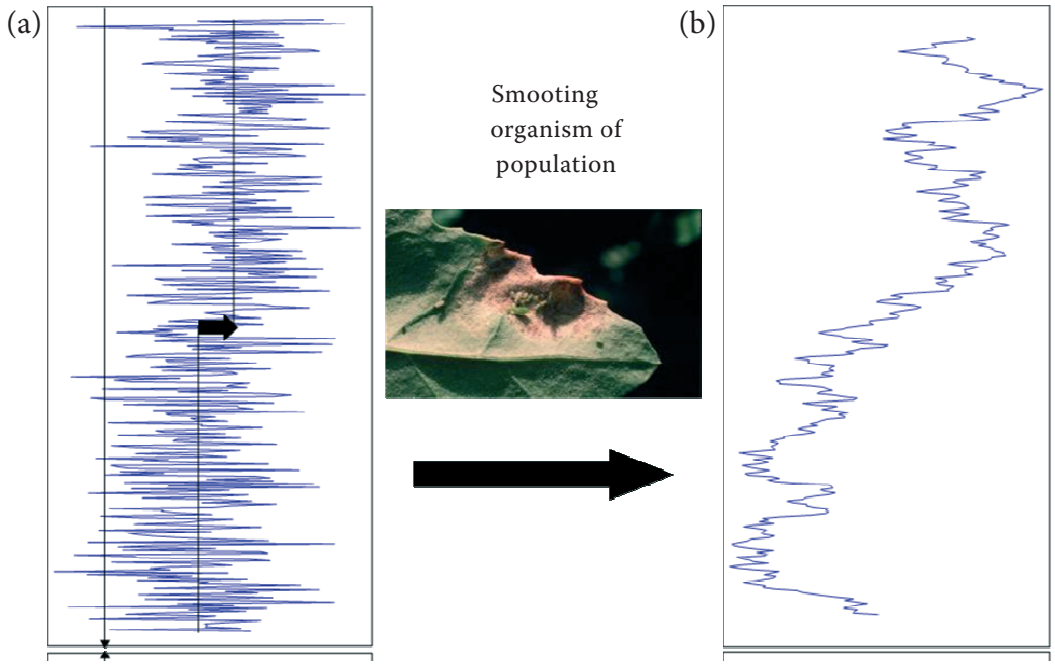

Figure 1. The effect of the time-scales over which biological responses by populations are integrated is to "filter" rapid changes from a time series. (a) A rapidly fluctuating series with a step-change in mean much smaller than the overall variability. The effect of this step change is to greatly alter the frequency of extreme events (line A); (b) The response as seen in a population or proxy for population such as disease incidence

Tracking host changes

To begin at the longest time-scale: host plant distributions will change in response to climate, and diseases will gradually follow. This allows some of the more robust predictions we may be able to make. For example, following the huge expansion in area cropped to oilseed rape, leading to large areas with the crop grown 1 in 4 or 5 years, increase in *Sclerotinia* is predictable; it is arguable that since the initial level was low and the pathogen monocyclic, the build-up is taking many years but without counter-measures will become extremely severe (compare Turner *et al.* 2002; Gladders *et al.* 2008). For the wild plants we have in most parts of the world, the plants we have are those which are able to alter their distributions over the time-scales involved in the glaciations and deglaciations of the last million years, of a century or so (Comes & Kadereit 1998). These range-expansion and contraction processes are faster than evolutionary emergence of new species, so communities have been assembled and torn apart many times. However, time-scales were probably long enough to allow pathogens to track the ranges of their hosts so that long-term escape from established diseases was probably rare. For crops, of course, enormous range-changes are possible over decadal time-scales. An indication of the kind of adaptation that commerce will drive is the current planting of champagne-type grapes in southern England by some of the major French champagne companies, who expect, or fear, that the ideal zone for growing grapes for this type of wine will move north. At the same time, genotypic changes will occur in crops with rapid breeding cycles, including most vegetable and arable crops; this is likely to alter the spectrum of diseases on these crops as selective changes responding to physiological adaptations have unexpected side-effects. (For example, introduction of semi-dwarfing genes to wheat accidentally implied environmental susceptibility to *Mycosphaerella graminicola* (Simon *et al.* 2005)). Alongside changes in crops, which may be put together in novel combinations, and changes in cropping patterns, which are probably more important, will go changes in soil microflora and in the unmanaged vegetation adjacent to cropped areas. These changes in soil and phylloplane microflora, alternate hosts, crop genotype, crop species and cropping patterns will drive not only changes in pathogen population dynamics,

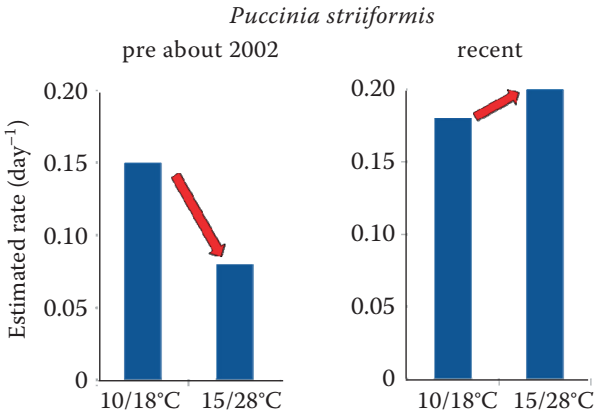

Figure 2. Evolutionary adaptation to climate by a pathogen. Estimates of intrinsic growth rate r are based on the data in Milus *et al.* (2009), using the approximate formula $r = \log(R)/p$, where R is total spore production and p generation time, assuming sporulation patterns do not differ greatly and that the generation time is approximated by the latent period (Segarra *et al.* 2001)

but also evolutionary changes in the pathogens. These changes, of course, are constrained by the space of variation which the integrated genotype of the pathogen can explore, which is limited in ways we are at present largely unable to define. The recent emergence of warm-adapted yellow rust of wheat (Milus *et al.* 2009), however, shows that the historical record of the environment limiting a disease may be an unreliable guide to the environments it can adapt to (Figure 2).

Predicting and testing predictions

Therefore, the problem of preparing for change in plant disease due to changing climate is inevitably complex, and specific prediction with any useful degree of skill more than a short time ahead is unlikely to be possible. (Skill here is used in the meteorologists' sense: in this case, better than a prediction based only on the long-term average.) This is because the uncertainties in predicting all the factors influencing disease are likely to spread out the initial distribution of uncertainty in climate prediction until the distribution is almost uninformative (cf. Scherm 2004).

If we wish to forecast changes in plant disease in relation to climate, we need to test our forecasts. The problem is that there is no point in forecasting if we must wait a half-century to see which elements of our forecast were true. We have some confidence in climate projections because, running them for past periods, they match past records to a better or worse extent. Such records include substantial spans of instrumental records in some countries, and then various proxy data available from ice and ocean sediment cores, from tree-rings, lake deposits, and the annual variation in growth of animals. With huge labour, such records, with varying degrees of certainty, now extend back for millenia or thousands of millennia (Solomon *et al.* 2007, Chapter 5; Mann *et al.* 2008). The problem is to find similar records of plant disease which can be used to test forecasts of plant disease models.

The long-term dynamics of plant disease has been little studied, because the focus of most pathologists has been on the provision of advice to and tools for farmers and breeders to deal with urgent problems. Many surveys of disease are either done once only, use incompatible or impressionistic measurements, or are not fully published. Other runs of data concerning breeders and chemical trials are published only sporadically and have not been collated. This is an urgent co-operative task, which will involve working out ways to relate measurements made in different ways, and even of symptoms now described differently.

An example of the use of long-term records comes from two series of measurements of wheat diseases in the UK. First, since 1970 the government of England and Wales has made a systematic survey of disease in the wheat crop, with a break of only two years (King 1977; Polley & Thomas 1991; Hardwick *et al.* 2001; Food and Environment Research Agency 2009). (That break illustrates the difficulty with this kind of work: it arose because the government of the time, believing that the public sector was too large, wished to cut all expenditure without obvious justification as a public good in the short term; where science funding is project-oriented on cycles of a few years, it is similarly very hard indeed for a data collection function to compete with a hypothesis-oriented project proposal, especially where, for the data to be continuous and useful, the collection project must win such a competition every three years.) Second, every year since 1845, wheat crops have been grown in the same field at Rothamsted Experiment Station in southern England, under a set of similar nutrient treatments. Each year samples of soil, straw and grain were dried and stored. In the early 2000s it proved possible to amplify specific

regions of DNA from both wheat and associated organisms in these archived samples (Bearchell *et al.* 2005). The survey data and these measurements make it possible to examine long-term changes in several pathogens.

Two pathogens of particular interest are those causing the so-called septoria diseases, *Mycosphaerella graminicola* and *Phaeosphaeria nodorum*. Both diseases require wet weather to multiply. *M. graminicola* currently produces abundant ascospores and so spreads from crop to crop, while *P. nodorum* is seed-borne. *P. nodorum* is perhaps adapted to somewhat higher temperatures than *M. graminicola,* and has a shorter minimum latent period. Thus, a simple environment based forecast would suggest that the prevalence of *P. nodorum* should have increased during the 1990s and 2000s, as average summer temperatures rose; little change before then would have been predicted. However, varietal factors and crop-crop spread change this picture. Varieties susceptible to *M. graminicola* were introduced during the 1970s, the wheat area increased, and seed fungicide treatment became the norm. All three factors should have led to an increase in *M. graminicola*. Looking further back, however, there should have been relatively little change during the 19th century, when both varieties and technology were stable.

Bearchell *et al*. (2005) correlated the England and Wales survey data with the measurements from archived samples at Rothamsted. The two data-sets agreed reasonably well. It is therefore reasonable to look back in the Rothamsted data into the 19th century (Bearchell *et al.* 2005; Shaw *et al.* 2008). Rather than the stability expected, there was a decline in *M. graminicola*, and a rise in *P. nodorum* through the latter 19th century and first three-quarters of the 20th century, before a dramatic and sustained rise in prevalence of *M. graminicola* in the last 30 years. Furthermore, there was an extraordinarily tight correlation with sulphate pollution throughout the period of the data series. Although this is not necessarily causal, it does illustrate the key problem of predicting disease: the factors controlling the systems are not fully understood (Shaw 2008). This is obvious enough to sound trivial, so it is worth emphasising that both diseases are intensively studied, and infect wheat, one of the most intensively studied plants. While there are no doubt still surprises lurking in climatic processes, the fundamental physics of climate is far better understood than the population biology of plant pathogens, and this difference needs to be emphasised when "best available" predictions are offered to discussions on policy.

There are two currently favoured methods for making predictions as to how geographic ranges and average severity of a disease may change. The more widely used assumes that climate and host currently limit the pathogen, and attempt to match the current geographic range with suitable climatic measures. Future range is then matched to predictions of future climate (Desprez-Loustau *et al.* 2007; Steffek *et al.* 2007); this is widely used in predicting the possible limits to expansion of newly invasive diseases (Venette & Cohen 2006). It is obviously valid only if the geographic range is approximately stable and therefore due to climatic limits rather than history; it is also vulnerable to stochastic evolutionary events such as the changes in yellow rust of wheat referred to above (Figure 2). The second approach applies if we believe that we know the basic factors limiting the abundance of a pathogen and understand quantitatively their relation to weather. It may then be appropriate to use a weather-based prediction system (Seem 2004; Semenov 2007) to predict future abundance. Garrett *et al*. (2006) argue that this reductive, process-based approach is practical, though challenging, for many pathosystems. For example, Evans *et al*. (2008) used an analysis of key features of the monocyclic pathogen *Leptosphaeria maculans*, coupled to a weather generator parameterised by predicted future climate, to predict the future levels of the pathogen (Figure 3). There are two aspects of this work which are of interest in the present context. First, there is no obvious way to test the predictions in advance of the period to which they refer. Second, despite the effort exerted, the problem is in certain senses atypically easy, because most of the key features of the models used were based on temperature sums and relations between abundance and functions of weather variables which were direct rather than via an exponential. The use of temperature sums smooths a good deal of variability; the non-exponential relationships arise because the pathogen is not polycyclic within a year. Many diseases of great economic importance – stem rust of wheat, for example – are highly polycyclic, and small errors in understanding relationships with the biotic or abiotic environment could produce very large errors in predictions. It is not obvious that these

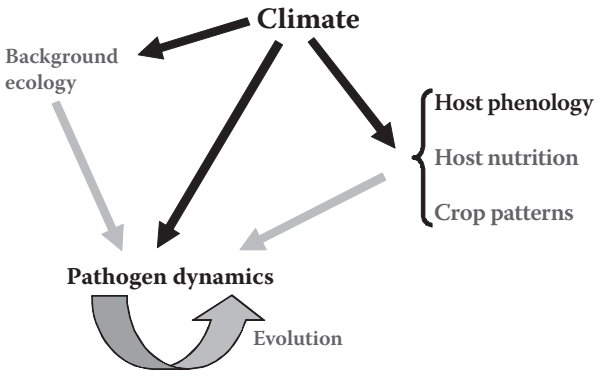

Figure 3. A simplified conceptual diagram of (black) the processes which are usually taken into account in a forecasting model and which can therefore be used to predict changes in disease abundance under an altered climate (grey) a minimum set of processes which are implicit in such a model but which may change as climate and consequently host communities change

limitations in extrapolating from small-scale, short time processes to much longer time-scales will be easy to overcome.

Approaches to predicting long-term prevalence that start from the basic processes involved require that we have a good understanding of these. At a minimum, they should make possible good short-term forecasting. Bourgeois *et al.* (2004) examined a number of pathosystems for which forecasts were available in Canada, concluding that none were reliable enough in a single season to use for long-term prediction with any reasonable confidence. Probably the most-studied plant disease is potato blight caused by *Phytophthora infestans*, and if any system is suitable for longer-term forecasting, this should be. The comparison made by Taylor *et al.* (2003) of the performance of various forecast models against historical data is sobering: under UK conditions, forecasts were very poor indeed in at least one year in six; in fact, while the forecasts do have some skill, they function to assist spraying only when continually updated with current observations. Without a clear understanding of what is missing in these short-term forecast models, they can hardly be used as the basis for longer-term climatic predictions. In fact, it seems quite possible that what is missing is evolutionary change, which has clearly happened rapidly in the last decades (e.g. Fry *et al.* 1992) – possibly after a long period of stasis. The rapid change in this case may have been made possible by the variability released through sex (e.g. Lehtinen *et al.* 2008). However, given a pathogen species, a list of hosts and a list of environments, we have few clues as to the constraints to variation there are in that pathogen to respond to selection to infect a new host or utilise a new environment. Even with a detailed molecular understanding of how an organism currently works, it still seems unlikely we shall ever be able to predict exactly what phenotype space an organism is potentially able to evolve into.

Changes in vector-borne virus diseases may be the easiest to predict, because vector activity and range is often related to simple medium-term climatic variables such as temperature sums (Lewis & Sturgeon 1978). However, most systems deployed to guide pesticide use rely on trapping methods both to initiate the calendar within which the temperature sums will be used and to determine the severity in a particular year. Furthermore, recent very serious problems with vector borne diseases seem to involve genetic changes in the pathogen or vector coupled with movement in trade or expansion of the area occupied by a crop, rather than any changes in climate such as improved or altered over-wintering (Colvin *et al.* 2004; Bove 2006).

Genetic change and community change

The last point may be a general one: changes in climate will produce changes in crop, weed and wild plant distributions, and to changes in animal ranges which will bring together pathogens and hosts in novel combinations or at novel times of the year. It seems increasingly probable that this will bring unexpected host shifts either through the pathogen "discovering" a new host which is vulnerable with only one or two mutational changes or through the acquisition of novel genetic material within the species or horizontally (Bove 2006; Stukenbrock & McDonald 2008). When such shifts happen, of course, they do so into host genetic landscapes which have not been selected for resistance, and the results may be very spectacular. Molecular biology, hopefully, as it increases our understanding of the interplay of host defence and pathogen counter-attack will clarify what is more and less likely, and provide an expanding set of examples of how host shifts may occur, and the routes of genetic innovation which make them possible. However, as it seems increasingly unlikely that a complete catalogue of

the organisms on the earth is attainable, it seems even less likely that a complete catalogue could be compiled, not just of what each does but of what it could do given arbitrary changes or exchanges. So one fairly secure prediction is that climate change will increase the rate at which unpleasant surprises appear.

Climate change at the rate projected for this century will mean that many plants find themselves in climatic zones which are no longer ideal; in consequence, they will be stressed, and disease resistance may be compromised. However, as a factor organising a plant community this particular mode of change seems likely to be less important than competition between plant species, in which disease would simply be another factor altering the dominance relations of the species growing in a particular area. The problem has two facets. One is aesthetic: the community of plants now best-suited to a changed climate will differ from that we are accustomed to. The other is to do with rate: the rate of climate change (and especially of change in rainfall patterns) is liable to be faster than the time-scale associated with turn-over in both forest (because trees take a long time to mature) and dryland vegetation (because growth is water-limited and plants must invest heavily in structural and secondary compounds and so grow slowly).

Research and advisory priorities

Evidently, research on the likely patterns of change in plant disease attributable to predicted climate change is important. Advances throughout the entire field of pathology will be helpful, but analysing the questions specifically posed by climate change points to some curiously neglected areas. One already mentioned is the need for better historical understanding of disease and better collation of the scattered data which exists. Another is the need for much better (quantitative) understanding of crop-crop transfer and off-season survival, without which long-term predictions of average prevalence will be almost meaningless. But our substantive advances in this area will not really be distinguishable from advances in general understanding of the ecology of plant disease. Advice to growers, and the politicians regulating markets in land food and commodities, must emphasise the need for systems to be resilient and adaptive to the unexpected. Planning must obviously include "best guesses" and clear predictions but we must convey the sources of uncertainty effectively.

References

Bearchell S.J., Fraaije B.A., Shaw M.W., Fitt B.D.L. (2005): Wheat archive links long-term fungal pathogen population dynamics to air pollution. Proceedings of the National Academy of Sciences, USA, **102**: 5438–5442.

Bourgeois G., Bourque A., Deaudelin G. (2004): Modelling the impact of climate change on disease incidence: a bioclimatic challenge. Canadian Journal of Plant Pathology, **26**: 284–290.

Bove J.M. (2006): Huanglongbing: a destructive, newly-emerging, century-old disease of citrus. Journal of Plant Pathology, **88**: 7–37.

Colvin J., Omongo C.A., Maruthi M.N., Otim-Nape G.W., Thresh J.M. (2004): Dual begomovirus infections and high *Bemisia tabaci* populations: two factors driving the spread of a cassava mosaic disease pandemic. Plant Pathology, **53**: 577–584.

Comes H.P., Kadereit J.W. (1998): The effect of Quaternary climatic changes on plant distribution and evolution. Trends in Plant Sciences, **3**: 432–438.

Desprez-Loustau M.L., Robin C., Reynaud G., Deque M., Badeau V., Piou D., Husson C., Marcais B. (2007): Simulating the effects of a climate change scenario on the geographical range and activity of forest pathogenic fungi. Canadian Journal of Plant Pathology, **29**: 101–120.

Evans N., Baierl A., Semenov M.A., Gladders P., Fitt B.D.L. (2008): Range and severity of a plant disease increased by global warming. Journal of the Royal Society Interface, **5**: 525–531.

Food and Environment Research Agency (2009): Survey of commercially grown wheat. Available at http://www.cropmonitor.co.uk/wwheat/surveys/wwheat.cfm (Accessed 14 July 2009)

Fry W.E., Goodwin S.B., Matuszak J.M., Spielman L.J., Milgroom M.G. (1992): Population genetics and intercontinental migrations of *Phytophthora infestans*. Annual Review of Phytopathology, **30**: 107–129.

Garrett K.A., Dendy S.P., Frank E.E., Rouse M.N., Travers S.E. (2006): Climate change effects on plant disease: genomes to ecosystems. Annual Review of Phytopathology, **44**: 489–509.

Gladders P., Ginsburg D., Smith J.A. (2008): Sclerotinia in oilseed rape – a review of the 2007 epidemic in England. HGCA Project Report No. 433.

Hardwick N.V., Jones D.R., Slough J.E. (2001): Factors affecting diseases of winter wheat in England and Wales, 1989–1998. Plant Pathology, **50**: 453–462.

King J.E. (1977): Surveys of diseases of winter wheat in England and Wales 1970–1975. Plant Pathology, **26**: 8–20.

Lehtinen A., Hannukkala A., Andersson B., Hermansen A., Le V.H., Naerstad R., Brurberg M.B., Nielsen B.J., Hansen J.G., Yuen J. (2008): Phenotypic variation in Nordic populations of *Phytophthora infestans* in 2003. Plant Pathology, **57**: 227–234.

Lewis T., Sturgeon D.M. (1978): Early warning of egg hatching in Pea Moth (*Cydia-nigricana*). Annals of Applied Biology, **88**: 199–210.

Mann M.E., Zhang Z., Hughes M.K., Bradley R.S., Miller S.K., Rutherford S., Ni F. (2008): Proxy-based reconstructions of hemispheric and global surface temperature variations over the past two millennia. Proceedings of the National Academy of Sciences, USA, **105**: 13252–13257.

Milus E.A., Kristensen K., Hovmøller M.S. (2009): Evidence for increased aggressiveness in a recent widespread strain of *Puccinia striiformis* f.sp. *tritici* causing stripe rust of wheat. Phytopathology, **99**: 89–94.

Polley R.W., Thomas M.R. (1991): Surveys of diseases of winter wheat in England and Wales, 1976–1988. Annals of Applied Biology, **119**: 1–20.

Scherm H. (1994): Global warming and nonlinear growth: how important are changes in average temperature? Phytopathology, **84**: 1380–1384.

Scherm H. (2004): Climate change: can we predict the impacts on plant pathology and pest management? Canadian Journal of Plant Pathology, **26**: 267–273.

Seem R.C. (2004): Forecasting plant disease in a changing climate: a question of scale. Canadian Journal of Plant Pathology, **26**: 274–283.

Segarra J., Jeger M.J., Van den Bosch F. (2001): Epidemic dynamics and patterns of plant diseases. Phytopathology, **91**: 1001–1010.

Semenov M.A. (2007): Simulation of extreme weather events by a stochastic weather generator. Climate Research, **35**: 203–212.

Shaw M.W. (2008): The population dynamical consequences of density-dependence in fungal plant pathogens in stress in yeasts and filamentous fungi. In: Avery S. V., Stratford M., van West P. (eds): Stress in Yeasts and Filamentous Fungi, British Mycological Society Symposia, Series 27. Kluwer, Dordrecht.

Shaw M.W., Bearchell S.J., Fitt B.D.L., Fraaije B.A. (2008): Long-term relationships between environment and abundance in wheat of *Phaeosphaeria nodorum* and *Mycosphaerella graminicola*. New Phytologist, **177**: 229–238.

Simon M.R., Perello A.E., Cordo C.A., Larran S., Putten P.E.L., van der Struik P.C. (2005): Association between Septoria tritici blotch, plant height, and heading date in wheat. Agronomy Journal, **97**: 1072–1081.

Solomon S., Qin D., Manning M., Chen Z., Marquis M., Averyt K.M., Tignor M., Miller H.L. (eds) (2007): Climate Change 2007: The Physical Science Basis. Cambridge University Press, Cambridge.

Steffek R., Reisenzein H., Zeisner N. (2007): Analysis of the pest risk from Grapevine flavescence dorée phytoplasma to Austrian viticulture. EPPO Bulletin, **37**: 191–203.

Stukenbrock E.H., McDonald B.A. (2008): The origins of plant pathogens in agro-ecosystems. Annual Review of Phytopathology, **46**: 75–100.

Taylor M.C., Hardwick N.V., Bradshaw N.J., Hall A.M. (2003): Relative performance of five forecasting schemes for potato late blight (*Phytophthora infestans*). I. Accuracy of infection warnings and reduction of unnecessary, theoretical, fungicide applications. Crop Protection, **22**: 275–283.

Turner J.A., Elcock S.J., Walters K.F.A., Wright D.M., Gladders P. (2002): A review of pest and disease problems in winter oilseed rape in England and Wales. In: BCPC: Pests and Diseases. BCPC, Brighton: 555–562.

Venette R.C., Cohen S.D. (2006): Potential climatic suitability for establishment of *Phytophthora ramorum* within the contiguous United States. Forest Ecology and Management, **231**: 18–26.

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