Plant Pathology (2011) **60,** 44–53 Doi: 10.1111/j.1365-3059.2010.02413.x

REVIEW

Impacts of changing air composition on severity of arable crop disease epidemics

B. D. L. Fitt[a]*, B. A. Fraaije[a], P. Chandramohan[b] and M. W. Shaw[b]

[a]*Rothamsted Research, Harpenden AL5 2JQ; and* [b]*School of Biological Sciences, University of Reading, Lyle Tower, Whiteknights, Reading RG6 6AS, UK*

This review assesses the impacts, both direct and indirect, of man-made changes to the composition of the air over a 200 year period on the severity of arable crop disease epidemics. The review focuses on two well-studied UK arable crops, wheat and oilseed rape, relating these examples to worldwide food security. In wheat, impacts of changes in concentrations of SO_2 in air on two septoria diseases are discussed using data obtained from historical crop samples and unpublished experimental work. Changes in SO_2 seem to alter septoria disease spectra both through direct effects on infection processes and through indirect effects on soil S status. Work on the oilseed rape diseases phoma stem canker and light leaf spot illustrates indirect impacts of increasing concentrations of greenhouse gases, mediated through climate change. It is projected that, by the 2050s, if diseases are not controlled, climate change will increase yields in Scotland but halve yields in southern England. These projections are discussed in relation to strategies for adaptation to environmental change. Since many strategies take 10–15 years to implement, it is important to take appropriate decisions soon. Furthermore, it is essential to make appropriate investment in collation of long-term data, modelling and experimental work to guide such decision-making by industry and government, as a contribution to worldwide food security.

Keywords: climate change, crop growth models, food security, gaseous pollutants, global warming, weather-based crop disease forecasts

Introduction

Ever since humans started to cultivate crops thousands of years ago, food security has been threatened by diseases that attack those crops when the host plant is susceptible, the pathogen virulent and the environment favourable for disease (Stukenbrock & McDonald, 2008). One component of that environment is the air in which crops grow, but it is only over the last 250 years, since the start of the industrial revolution, that human activities have greatly affected the gaseous composition of the air around crops (Chakraborty & Newton, 2011). These changes to the gaseous composition of the air have included increases in concentrations of pollutants (e.g. SO_2; Anonymous, 2001) and greenhouse gases (e.g. CO_2; De Klein *et al.*, 2006; Jackson *et al.*, 2007). These categories are not mutually exclusive since some greenhouse gases (e.g. N_2O) may also be pollutants (Jackson *et al.*, 2007). There may be direct impacts of changes in air composition on crop diseases, such as impacts on host–pathogen interactions through effects on the host, the pathogen or the interaction (Eastburn *et al.*, 2011). However, there may also be indirect impacts mediated by changes in climate resulting from increasing concentrations of greenhouse gases (Pachauri & Reisinger, 2007); these changes have been accelerating in the last 20 years (Semenov, 2009; Fig. 1). Such changes in climate are a route by which changes in air composition can affect severity of crop disease epidemics (Chakraborty, 2005; Garrett *et al.*, 2006; Evans *et al.*, 2008; Butterworth *et al.*, 2010). This is likely to increase food security problems caused by crop diseases (Anderson *et al.*, 2004; Garrett *et al.*, 2006; Gregory *et al.*, 2009).

It is essential to understand these direct and indirect impacts of changing air composition since they may exacerbate food insecurity (Chakraborty & Newton, 2011). Currently, more than 1 billion people (one-sixth of world population) do not on average have enough to eat (FAO, 2009). In normal circumstances and most parts of the world, this has little to do with losses to pests and diseases and much to do with access to input resources and power within society (Sen, 1999). Food security is now being

*E-mail: bruce.fitt@bbsrc.ac.uk

Plant Pathology

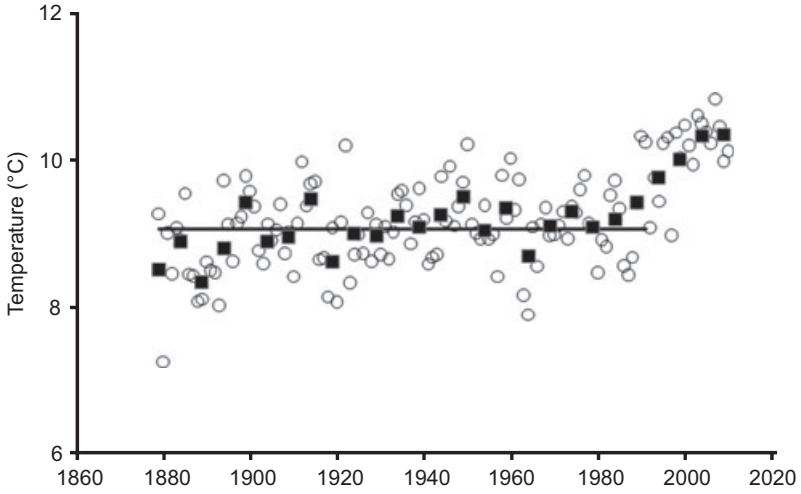

Figure 1 Change in annual mean (○) and 5-year mean (■) air temperatures at Rothamsted, Harpenden, UK, in the period from 1878 to 2009. Horizontal line indicates the mean air temperature for the period 1878–1990. Adapted from a figure at http://www.rothamsted.ac.uk/.

further affected by increased demand for biofuel crops through competition for resources (e.g. water, fertilizers and available culturable land). However, for subsistence farmers crop losses caused by pests and diseases mean that there is less food to eat and for non-producing consumers they mean that food costs are increased (Flood, 2010). In the longer term, pressures on land and resources are greater if outputs are reduced by pests, diseases and weed competition. Some estimates for four of the largest crops put actual worldwide losses to diseases, despite use of crop protection strategies, as high as 55 kg per person per season (Table 1, assuming a world population of 6·5 billion). The benefits of disease control strategies already in place for these four crops (difference between potential losses and actual losses), especially use of crop cultivars with improved resistance to diseases and of fungicides, have been estimated at more than 15 kg per person per season on average (Oerke, 2006; Table 1). However, because altering one aspect of a system alters the whole system, there is large uncertainty in these estimates.

Regardless of the problems of financial measurement, the food security problems associated with crop diseases are especially severe in the developing world, where even local destruction of crops can lead to starvation for farming families (Strange & Scott, 2005; Forbes & Simon, 2007). Thus, there is a need to stabilize food production by managing crop diseases more consistently. More effective disease management to decrease average losses and stabilize variation in losses allows farmers to use inputs more efficiently. The advantage of protection against diseases in stabilizing yields is likely to be much greater than that of decreasing the average loss attributed to the disease, because the area planted to give a reliable output is decreased by the maximum loss the grower is concerned about, not the average loss. Therefore, if environmental change associated with anthropogenic effects on the composition of the air increases losses from crop diseases, and other aspects of the worldwide or regional economy are unchanged, more people will suffer food shortages. However, strategies for adaptation to the changing environment, such as development of new fungicides or new crop

Table 1 Crop protection and food security worldwide[a], illustrated by rice, wheat, maize and potato, harvest years 2001–2003. A comparison between potential losses if no crop protection measures were used to control diseases (caused by fungal, bacterial and virus pathogens) and actual estimated losses, adapted from Oerke (2006)

	Attainable production[b]	Potential crop losses (without crop protection)		Actual crop losses (with crop protection)	
	M t	%[c]	M t	%[c]	M t
Rice	933	15	140	12	112
Wheat	785	18	141	13	102
Maize	891	12	107	11	98
Potato	518	9	47	8	41

[a]Losses were estimated separately for 19 different regions of the world and mean values were calculated per unit of production (intensity of cropping differed between regions).
[b]Attainable yield is defined as the site-specific maximum yield and is generally much less than the theoretical yield potential.
[c]Expressed as a percentage of the attainable yield.

cultivars that will be resistant to diseases in the changed environment both take 10–15 years. Thus, decisions need to be taken now to plan for future (Barnes *et al.*, 2010). The aim of this review is to examine how environmental change associated with both direct and indirect impacts of human-caused changes in air composition may impact on severity of crop disease epidemics. Two UK arable crops are used as examples, because they are well studied; nonetheless, general conclusions can be tentatively drawn from these local examples. The review will not discuss impacts of atmospheric changes on diseases of forest trees (reviewed by Sturrock *et al.*, 2011), or amenity shrubs such as roses.

Direct impacts of changing air composition on severity of crop disease epidemics

Changes in the gaseous composition of the air may directly affect the severity of crop disease epidemics through effects on the host, the pathogen or the host–pathogen interaction (Eastburn *et al.*, 2011). The severity of epidemics may be decreased, for example, when increased concentrations of CO_2 increase healthy crop growth (Semenov, 2009). By contrast, the severity of epidemics may be increased, for example, when increased concentrations of O_3 damage tissue and favour development of necrotrophic pathogens such as *Botrytis cinerea* (cause of grey mould on many hosts) (Eastburn *et al.*, 2011). Since the impacts on plant diseases of changes in concentrations in air of CO_2 or O_3 are reviewed comprehensively by Eastburn *et al.* (2011), they will not be discussed further in this review. Whilst there are interactions between concentrations of N_2O and O_3 (Chipperfield, 2009), there has been little work on direct effects of N_2O on the severity of disease epidemics.

A pollutant of particular interest is SO_2, because it has changed greatly in abundance over the last two centuries. In the early industrializing areas of northern Europe, it increased during the 19th and 20th centuries. By the late 20th century, damage to sensitive ecosystems was obvious, the sources were quite concentrated and the technology existed to remove it from power-station and industrial exhausts. Over approximately two decades, the concentration in the atmosphere over western Europe decreased to little more than the background concentration arising from marine sources. The preserved wheat samples from the 170-year Broadbalk experiment in continuous winter wheat growing at Rothamsted Research, Harpenden, UK, (Fig. 2a) provided a unique insight into the changes in prevalence of a pathogen on a major crop when it was found that DNA characteristic of the two septoria pathogens, *Mycosphaerella graminicola* (*Septoria tritici*) (speckled leaf blotch, Fig. 2c) and *Phaeosphaeria nodorum* (*Stagonospora nodorum*) (glume blotch, leaf blotch, Fig. 2d), could be successfully amplified from both grain and straw samples (Bearchell *et al.*, 2005; Shaw *et al.*, 2008). Since *P. nodorum* (Fig. 2b) is also seed-borne, whereas *M. graminicola* is not, the changes in their relative importance influence strategies to decrease disease severity and yield loss (Shaw *et al.*, 2008). The two pathogens occupy a similar niche, requiring leaf wetness for infection and attacking the same tissue, so they are likely to compete. Changes in their abundance may reflect changes in the competitive balance between them (Nolan *et al.*, 1999; Chandramohan, 2010).

These pathogens were known from national surveys of wheat diseases to have reversed their relative importance in England and Wales during the period 1975–2005. The point-location DNA concentrations from the Broadbalk experiment were well correlated with the national survey data over this period, suggesting that the change in their relative importance occurred in the 1980s (King, 1977; Polley & Thomas, 1991; Hardwick *et al.*, 2001) and that the Broadbalk data reflected wider scale changes. The long-term Broadbalk series showed that *M. graminicola* had been common in the mid-19th century but very rare during the first three-quarters of the 20th century, whilst *P. nodorum* had an approximately opposite pattern in both grain and straw. Over the 170-year time-period covered, the pattern in the ratio of the two pathogens was extremely closely correlated with that of SO_2 emissions over England and Wales (Fig. 3). This could, of course, be coincidence, but the changes were poorly correlated with environmental (spring rain, winter temperature) or agronomic (harvest method, sowing and harvest dates, fungicide and other pesticide use, cultivar height) factors, or even with cultivar changes. Other pollutants (polychlorinated biphenyls [PCBs], polyaromatic hydrocarbons [PAHs] and inferred quantities of ozone and nitrogen oxides) were also uncorrelated with the changes in pathogen populations. By contrast, each pathogen individually, or their sum, was well correlated with spring rainfall and more weakly correlated with some other environmental factors. This suggests that SO_2 emissions were responsible for the balance between two pathogens, with similar responses to other environmental features.

For this relationship to be causal, there has to be a link between national emissions and local effects. This is provided by the relatively long residence time of SO_2 in the atmosphere (half-time 3 days; Garland, 1977), coupled with emission and mixing at relatively high altitudes (approximately 1 km) and therefore extended horizontal scales. In general, sites more remote from sources receive more sulphur deposition in precipitation, whilst dry deposition by absorption into soil and plant tissues is more important closer to sources (Whelpdale, 1992). The combined effects of long residence times and the greater importance of rainfall deposition at more remote sites are to smooth the deposition pattern. It is therefore not implausible that the sulphate deposition at Rothamsted was representative of the England and Wales average (Zhao *et al.*, 2003). Both wet and dry sulphur deposition could cause plant tissue damage, acidity of the water within which spore germination takes place and improved sulphur nutrition of the crop.

Recent experimental work (Chandramohan, 2010) suggests that the effect is causal but indirect and that it interacts with other factors. At its worst, SO_2 in the

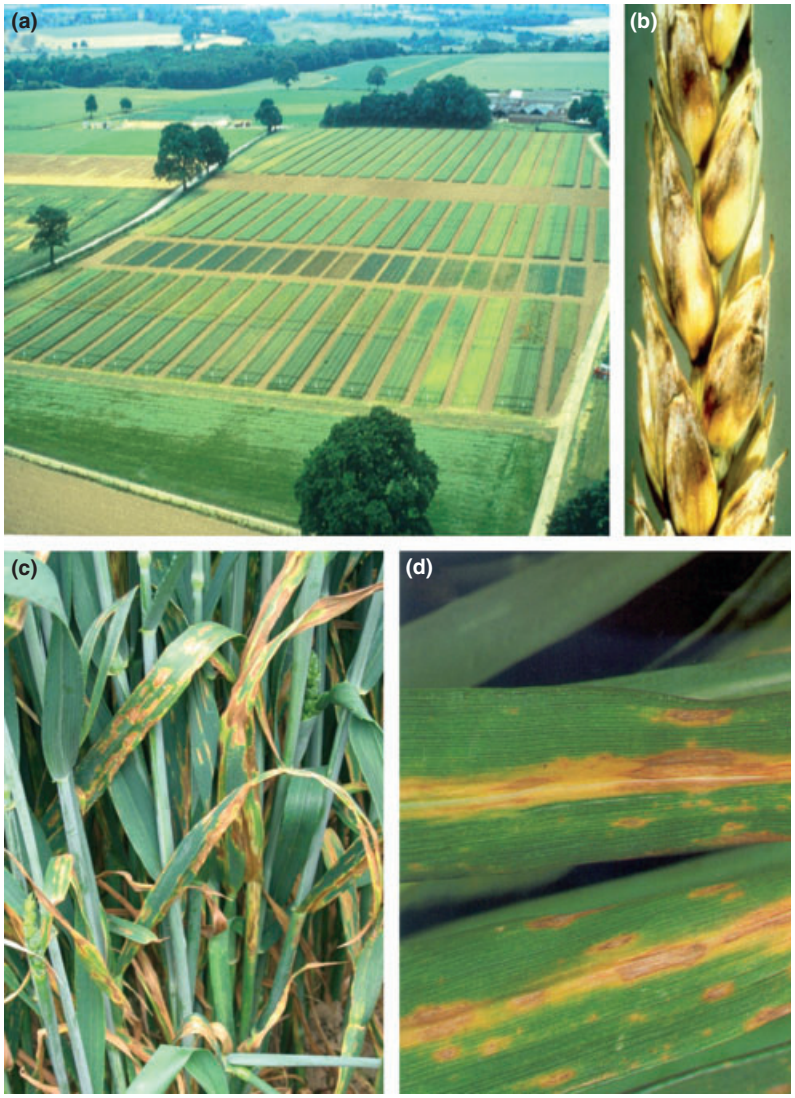

Figure 2 The Broadbalk winter wheat experiment at Rothamsted, Harpenden, UK, which started in 1843, photographed in June. (a) Plots going across the experiment received different fertilizer treatments and in each each strip of plots winter wheat was in a different crop rotation. Long-term changes in relative abundance of the pathogens responsible for septoria leaf blotch diseases, namely *Phaeosphaeria nodorum* (*Stagonospora nodorum*) (d) and *Mycosphaerella graminicola* (*Septoria tritici*) (c) were studied by quantifying DNA of these pathogens from samples winter wheat leaves/stems taken at harvest each year and stored in the Broadbalk archive (Bearchell *et al.*, 2005). Similarly, abundance of *P. nodorum* (glume blotch) (b) in grain samples was studied using quantitative PCR (Shaw *et al.*, 2008).

atmosphere led to rain falling with a pH as low as 4. There appeared to be very little direct effect of SO_3^{2-} concentrations leading to pH values between 7 and 4 on germination or growth of either *M. graminicola* or *P. nodorum*. However, high SO_3^{2-} concentrations in the water in which spores were suspended for artificial inoculation favoured infection of wheat leaves by spores of *P. nodorum* more than they favoured infection by spores of *M. graminicola*. Furthermore, glasshouse experiments in which wheat grown with different nutrient solution treatments was inoculated with the pathogens suggest that, although low sulphate concentration in soil favours both pathogens, it benefits *M. graminicola* much more. Given that the two pathogens must compete for resources, since they both infect the same leaves under similar environmental conditions (Shaw *et al.*, 2008), relatively moderate changes in host susceptibility mediated by changes in airborne S input to the soil could change the balance between the two pathogens. Although there are interactions with wheat cultivar and only a small number of isolates of each pathogen were tested by Chandramohan (2010), these results do suggest a mechanism by which SO_2 concentrations in the air could influence the range of diseases affecting a crop, and the historical data suggest that they have done so. They also illustrate the difficulties in making predictions of future changes in disease

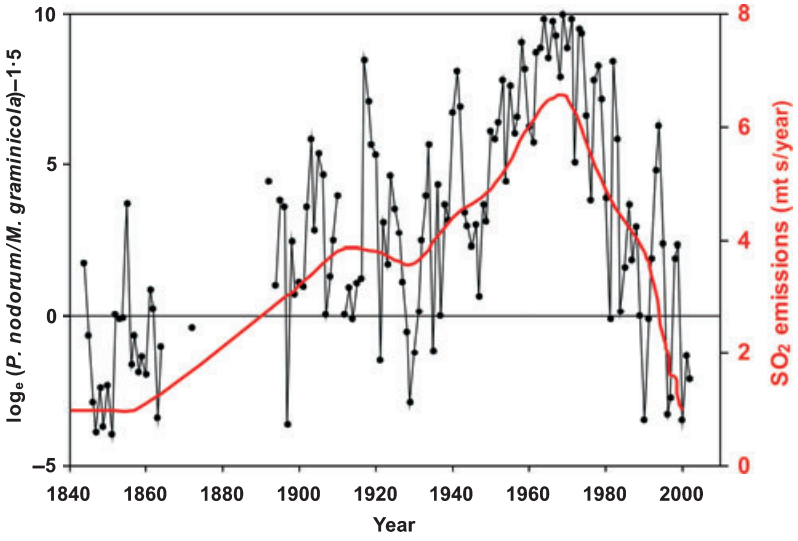

Figure 3 Relationship between ratio of *Phaeosphaeria nodorum* to *Mycosphaerella graminicola* DNA in samples from the Broadbalk experiment, Rothamsted, Harpenden, UK, 1844–2003 (●) and smoothed estimates of UK atmospheric SO_2 emissions (Mt S per year, red line). For linear regression of 10-year average values of the pathogen ratio on SO_2 emissions (not illustrated) $r = 0{\cdot}96$, $P < 0{\cdot}001$. Adapted from a figure in Bearchell *et al.* (2005).

patterns, because of the complexity of interactions possible in the system.

Whilst the effect of high SO_3^{2-} concentrations in the water on infection of wheat leaves by spores of *P. nodorum* was clear (Chandramohan, 2010), it is difficult to interpret it in terms of plant tissue damage. *Phaeosphaeria nodorum* is usually described as a more necrotrophic pathogen, favoured by host tissue damage, whilst *M. graminicola* is thought of as hemibiotrophic (Eyal, 1999), so by analogy with other hemibiotrophs to be disfavoured by SO_3^{2-}. Thus, one would expect high SO_3^{2-}concentrations to favour *P. nodorum,* as seen in the historical data. However, the complexity of the situation and the difficulty in making a useful prediction from an historical observation is emphasized by the effects being different on different wheat cultivars; although the relative severity of damage caused by the two pathogens was changed consistently, in one case both pathogens were favoured by high SO_3^{2-} concentration, in another both were disadvantaged.

Indirect impacts of increasing greenhouse gases, through climate change, on severity of crop disease epidemics

It is necessary to assess the impact of climate change on crop yield losses caused by diseases, to guide industry and government planning for adaptation to climate change. Such an assessment requires outputs from quantitative models of crop–disease–climate interactions. However, much work on effects of climate change on crops and their diseases has been qualitative (e.g. Anderson *et al.*, 2004; Ghini *et al.*, 2007; Dumalasová & Bartoš, 2009) and there have been few attempts to produce combined crop–disease–climate models (Luo *et al.*, 1995). The efforts that have been made are hindered by the relatively complex epidemiology of many pathogens and great sensitivity of final disease severity to small environmental changes. Furthermore, the relationships in most weather-based models developed for use in disease forecasting are often not adequate for projection of the disease into future climates (Bourgeois *et al.*, 2004; Shaw, 2009).

In some cases, however, the disease cycle is favourable for an integrated model of the relationship between climate and disease to be feasible. This can be illustrated by work with oilseed crops (*Brassica napus*) and the two most important diseases of oilseed rape in the UK. Oilseed rape is grown worldwide to provide oil and protein for human or animal food and for use in fuel. Severe epidemics of phoma stem canker (blackleg) due to *Leptosphaeria maculans*) in Europe, North America and Australia cause losses of more than £1000M per cropping season at the current UK price of £300 per tonne. The disease is spreading across the world and potentially threatens crops in Africa, India and China (Fitt *et al.*, 2006, 2008). Light leaf spot (*Pyrenopeziza brassicae*) also causes losses in northern Europe, including the UK (Gilles *et al.*, 2000; Boys *et al.*, 2007). In the UK, phoma stem canker is currently more important in southern England and light leaf spot more important in northern England and Scotland (http://www.cropmonitor.co.uk; Fitt *et al.*, 1998). It is predicted that climate change will, other factors remaining unchanged, increase the range and severity of phoma stem canker epidemics (Evans *et al.*, 2008; Butterworth *et al.*, 2010).

UK temperature and rainfall values projected under high and low CO_2 emissions for the 2020s and 2050s were input into weather-based models for forecasting

severity of phoma stem canker epidemics on oilseed rape across the UK (Evans *et al.*, 2008). This projection suggested that epidemics will not only increase in severity but also spread northwards by the 2050s (Fig. 4a,b). However, similar work projected that climate change will decrease the incidence of light leaf spot, even in Scotland (Evans *et al.*, 2010; Fig. 4c,d). To investigate crop–disease–climate interactions, UKCIP02 scenarios projecting UK temperature and rainfall under high- and low-CO_2 emission scenarios for the 2020s and 2050s were combined with a crop simulation model predicting yield of fungicide-treated winter oilseed rape (STICS; Brisson *et al.*, 2003) to predict that climate change will increase yield of winter oilseed rape crops treated with fungicide to control diseases in Scotland by up to 0·5 t ha^{-1} (15%) (Butterworth *et al.*, 2010; Fig. 4e,f). When a model predicting yield loss from phoma stem canker epidemics was also included, by contrast, the projection was that in southern England climate change will increase yield losses from phoma stem canker epidemics to up to 50% (1·5 t ha^{-1}) unless the disease is controlled, so greatly decreasing yields of untreated winter oilseed rape. Such projections illustrate the contrasting impacts of climate change on crop diseases in different regions. These results provide a stimulus to develop models to project effects of climate change on other crop diseases. Such projections can be used to guide policy and farming practice in adapting to effects of climate change on food security.

Continuing with the UK oilseed rape disease example, it is possible to analyse the classes of crop protection actions available for farmers to use within their strategies for adaptation to climate change (Barnes *et al.*, 2010). An initial response to increases in disease-related yield loss is to use a more effective fungicide regime in the autumn (Gladders *et al.*, 2006; Fig. 5). Farmers may improve spray timing by using web-based disease forecasts, such as the light leaf spot and phoma leaf spot forecasts developed at Rothamsted (Welham *et al.*, 2004; Evans *et al.*, 2008; http://www.rothamsted.ac.uk/leafspot/). However, this alone will not offer a long-term solution to disease problems, because of decreasing effectiveness of fungicide with evolution of fungicide resistance in pathogen populations (Hollomon & Brent, 2009) and withdrawal of fungicides in response to EU legislation (Mahmuti *et al.*, 2009). Another disease control tactic is for the farmer to choose to extend rotations and/or introduce novel crops within the rotation. A 4-year break between oilseed rape crops may decrease yield losses from phoma stem canker (West *et al.*, 2001). If appropriate, this can be adopted rapidly within a single season, after harvest of the previous crop and before the new growing season in the autumn.

Another action assisting adaptation to the altered environment may be to sow seed of cultivars that are more resistant against the pathogens favoured by the changed environment. The effectiveness of this resistance can be expected to improve if disease epidemics gradually become more severe, since disease resistance will then have a greater weighting in the selection criteria used by individual breeders and national testing procedures, such as the UK HGCA Recommended Lists (http://

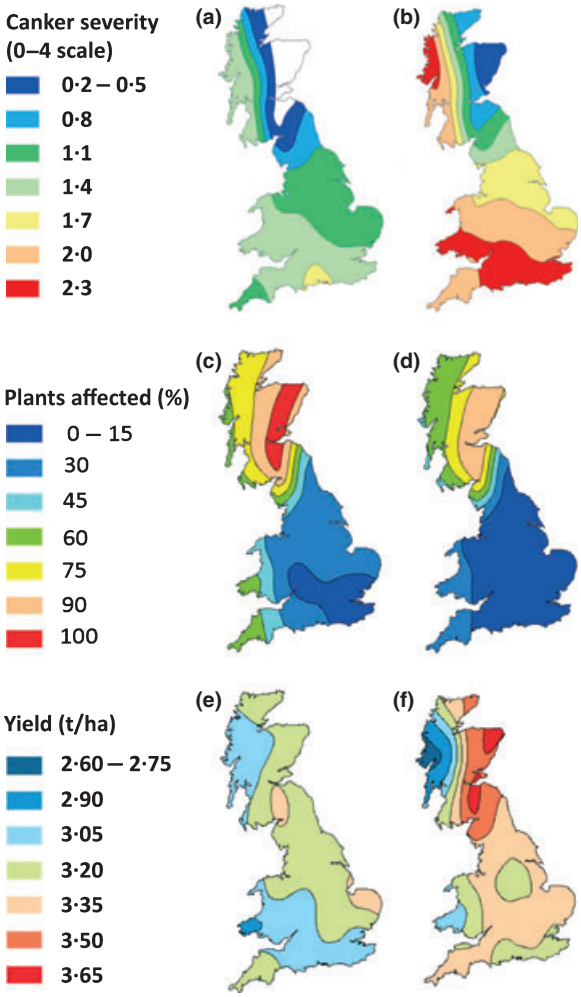

Figure 4 Impacts of climate change on severity of phoma stem canker, incidence of light leaf spot and yield of oilseed rape treated with fungicide to control diseases. Predicted severity of phoma stem canker (*Leptosphaeria maculans*) at harvest of winter oilseed rape crops (mean of resistant and susceptible cultivars) for (a) baseline 1961–1990, (b) 2050s climate (mean of low- and high-emission scenarios); stem canker severity on a 0–4 scale (0, no disease; 4, plant dead; Zhou *et al.*, 1999); areas where crops are unaffected by the stem canker disease are marked white. Predicted incidence (percentage plants affected) of light leaf spot (*Pyrenopeziza brassicae*) at green flower bud (GS 3,3) of UK winter oilseed rape crops (mean of resistant and susceptible cultivars) for (c) baseline 1961–1990 and (d) 2050s high-emissions climate scenarios. Predicted yield (t ha^{-1}) of winter oilseed rape (treated with fungicide to control diseases) for (e) baseline 1961–1990, and (f) 2050s high-emissions climate scenarios using the STICS crop growth model. Predicted values are interpolated from predictions for 14 sites across the UK. Winter oilseed rape crops are generally grown in the eastern halves of England and Scotland; less fertile and mountainous areas in the west are unsuitable for arable crops. Adapted from figures in Evans *et al.* (2008, 2010) and Butterworth *et al.* (2010).

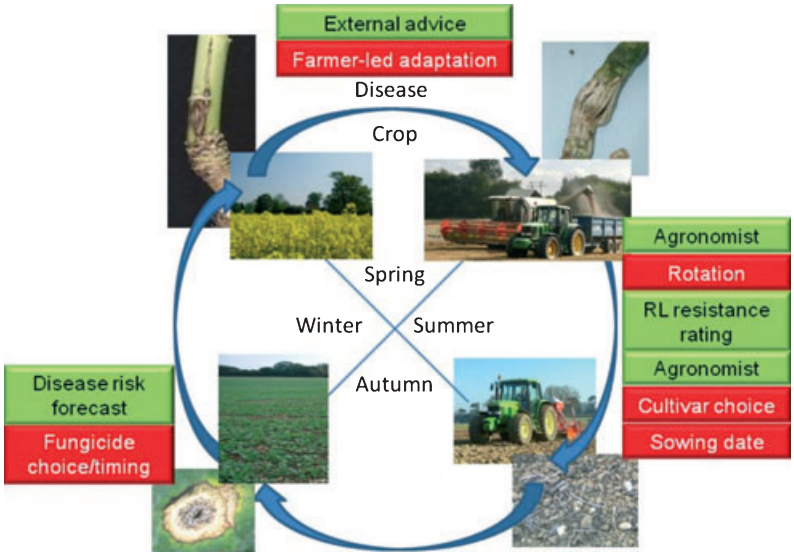

Figure 5 Seasonal development of winter oilseed rape in the UK in relation to progress of phoma stem canker (*Leptosphaeria maculans*) epidemics and short-term farmer-led adaptation strategies. Crops are sown in late summer (August/September) and emerge within 10 days when there is sufficient soil moisture. Stem extension occurs in late winter (February/March) and is followed by flowering in spring (April/May) with harvest in summer (July). Phoma stem canker epidemics are started by airborne ascospores produced on diseased crop debris in autumn/winter (October–December) with phoma leaf spot developing 10–30 days after spore release (depending on temperature). *Leptosphaeria maculans* grows along leaf petioles to reach the stem where early cankers may be seen in spring (April/May); these may become severe by harvest and cause considerable yield loss. Farmer-led short-term adaptation strategies include choice of rotation (e.g. increasing interval between successive oilseed rape crops), choice of cultivar (e.g. selection of cultivars with greater resistance to *L. maculans*) and choice of sowing date (e.g. early sowing favours disease) before the start of the growing season. In autumn, farmers can decide on fungicide, fungicide timing and frequency (to maximize control of phoma stem canker). External advice is available from agronomists, HGCA recommended lists (RL resistance rating), forecasting schemes (e.g. http://www.rothamsted.ac.uk/leafspot) and agrochemical company representatives. Adapted from a figure in Barnes *et al.* (2010).

www.hgca.com). If direct genetic modification techniques become accepted in European agriculture, it may be possible to increase resistance to specific pathogens more rapidly. If disease resistance is improved, crop growth will be increased and the optimal level of nitrogen fertilizer may be increased, taking into account yield and altered disease susceptibility (Berry *et al.*, 2010). However, increased use of nitrogen may increase input costs and there are demands to reduce nitrogen inputs to decrease greenhouse gas emissions and diffuse water pollution (Glendining *et al.*, 2009; Mahmuti *et al.*, 2009). Integrated management using combinations of these adaptation practices has been projected to be capable of increasing UK oilseed rape yield from an average of 3 to 6·5 t ha^{-1} under optimal conditions (Berry & Spink, 2006). Although the management of the whole system would be by farmers, several of the options mentioned would require sustained further investment in applied research and the effective transmission of the resulting knowledge to the industry (Gladders *et al.*, 2006). Aspects of this work are likely to require government investment, particularly where the outputs will not necessarily lead directly to increased profits for industry or where the industry structure is such that a 10- to 15-year time-scale for return on investment is not acceptable. Similar analyses may be applied to assess benefits of farmer-led adaptation to climate change for other crop diseases.

Discussion

The work reviewed provides evidence that man-made changes in the composition of the air have had considerable impact, either directly or indirectly, on the severity of arable crop disease epidemics and are likely to continue to have such impacts. Such impacts can include changes in the predominant disease present on a crop (Bearchell *et al.*, 2005; Shaw *et al.*, 2008), changes in the range or severity of epidemics (Evans *et al.*, 2008) or introduction of new diseases to a region (Shaw & Osborne, 2011). However, long-term data sets are needed to test the models used for projection, with both disease/pathogen data and gaseous composition/weather data (Jeger & Pautasso, 2008). Such data are illustrated by those from the Broadbalk archive (Bearchell *et al.*, 2005; Shaw *et al.*, 2008) or from field experiments done over a 15-year period (Evans *et al.*, 2008, 2010). Such work may be complemented by direct experimental work on non-climatic effects of changing air composition on crop plants, for example using a FACE system (Eastburn *et al.*, 2011). To make predictions for other major crop diseases, it is important to invest in collation of datasets from existing

sources of data and in modelling and new experimental work.

Modelling work to assess impacts of environmental change on severity of crop disease epidemics that combines weather-based disease models, crop models and climate scenarios (Luo *et al.*, 1995; Butterworth *et al.*, 2010) is a considerable improvement on the qualitative rule-based methods for predicting impacts of climate change on diseases that were used previously (Coakley *et al.*, 1999; Anderson *et al.*, 2004). Nevertheless, there will inevitably be uncertainty in such projections associated with uncertainty in projections of future weather (Semenov & Stratonovitch, 2010) and uncertainty in the disease and crop models. However, that is no reason not to make projections (Stern, 2007), provided that any assumptions made are clearly stated (Evans *et al.*, 2010), since they provide the best available basis for discussion of current decision making. Since strategies for adaptation to climate change, such as breeding new crop cultivars with resistance that can operate at increased temperatures (Huang *et al.*, 2006) or development of new fungicides, may take 10–15 years, informed comment about what diseases are likely to increase in importance is needed now to guide decision-making by industry and government. Projections in which values of explanatory variables (e.g. temperature, rainfall) remain within the range of those observed when collecting the experimental data must be more secure than those projections which extrapolate beyond this range; hence Evans *et al.* (2008) projected forward to the 2020s and 2050s, but not the 2080s, which would have taken relationships within the model outside the range of observed data.

This review demonstrates that environmental change associated with man-made changes in the composition of the air is likely to affect the severity of disease on arable crops (Gregory *et al.*, 2009). There is a need to assess the threat to food production from such changes and develop appropriate strategies to mitigate them or adapt agricultural methods before it is too late (Beddington, 2010; Smith & Olesen, 2010). It is particularly important to assess threats to food production in areas of the world most threatened by environmental change where there are already serious shortages of food, such as sub-Saharan Africa (Strange & Scott, 2005; Schmidhuber & Tubiello, 2007) but, in general, data are least available and experiments hardest to do in the poorest communities. However, it is also important to assess changes to food production in areas that may potentially benefit from environmental change, such as northern Europe (Butterworth *et al.*, 2010), since there is likely to be increasing pressure on such areas to produce more food to mitigate reduced production elsewhere (Stern, 2007; Mahmuti *et al.*, 2009).

Acknowledgements

We thank the UK Biotechnology and Biological Sciences Research Council (BBSRC; Centre for Bioenergy and Climate Change ISPG) and Department for Environment, Food and Rural Affairs (Defra, OREGIN), the Sustainable Arable LINK programme (PASSWORD, CORDISOR, CLIMDIS) and the Commonwealth Scholarships Commission for funding this research, and the British Society for Plant Pathology for supplementary funding. We are grateful to the many other colleagues who have contributed to the work reviewed, including Sarah Bearchell (Reading), Andrew Barnes, Dominic Moran (SAC), Peter Gladders (ADAS), Neal Evans, Michael Butterworth, Mikhail Semenov and Jon West (Rothamsted). We thank Paul Poulton and David Hughes for Fig. 1, Mikhail Semenov for Fig. 4, Neal Evans for Fig. 5 and Keith Goulding for comments on the paper.

References

Anderson PK, Cunningham AA, Patel NG, Morales FJ, Epstein PR, Daszak P, 2004. Emerging infectious diseases of plants: pathogen pollution, climate change and agrotechnology drivers. *Trends in Ecology & Evolution* **19**, 535–44.

Anonymous, 2001. *Transboundary Air Pollution: Acidification, Eutrophication and Ground Level Ozone in the UK*. London, UK: National Expert Group on Transboundary Air Pollution, Department for the Environment, Food and Rural Affairs.

Barnes AP, Wreford A, Butterworth MH *et al.*, 2010. Adaptation to increasing severity of phoma stem canker on winter oilseed rape in the UK under climate change. *Journal of Agricultural Science* **148**, 683–94.

Bearchell SJ, Fraaije BA, Shaw MW, Fitt BDL, 2005. Wheat archive links long-term fungal pathogen population dynamics to air pollution. *Proceedings of the National Academy of Sciences, USA* **102**, 5438–42.

Beddington J, 2010. Food security: contributions from science to a new and greener revolution. *Philosophical Transactions of the Royal Society B* **365**, 61–71.

Berry PM, Spink JH, 2006. A physiological analysis of oilseed rape yields: past and future. *Journal of Agricultural Science, Cambridge* **144**, 381–92.

Berry PM, Kindred DR, Olesen JE, Jorgensen LN, Paveley ND, 2010. Quantifying the effect of interactions between disease control, nitrogen supply and land use change on the greenhouse gas emissions associated with wheat production. *Plant Pathology* **59**, 753–63.

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**, 284–90.

Boys EF, Roques SE, Ashby AM *et al.*, 2007. Resistance to infection by stealth: *Brassica napus* (winter oilseed rape) and *Pyrenopeziza brassicae* (light leaf spot). *European Journal of Plant Pathology* **118**, 307–21.

Brisson N, Gary C, Justes E *et al.*, 2003. An overview of the crop model STICS. *European Journal of Agronomy* **18**, 309–32.

Butterworth MH, Semenov MA, Barnes A, Moran D, West JS, Fitt BDL, 2010. North-south divide; contrasting impacts of climate change on crop yields in Scotland and England. *Journal of the Royal Society Interface* **7**, 123–30.

Chakraborty S, 2005. Potential impact of climate change on plant–pathogen interactions. *Australasian Plant Pathology* **34**, 443–8.

Chakraborty S, Newton AC, 2011. Climate change, plant diseases and food security: an overview. *Plant Pathology* **60**, 2–14.

Chandramohan P, 2010. *Causal Relationships Between Sulphur Emissions and Pathogen Abundance*. Reading, UK: University of Reading, PhD thesis.

Chipperfield M, 2009. Atmospheric science: nitrous oxide delays ozone recovery. *Nature Geoscience* **2**, 742–3.

Coakley SM, Scherm H, Chakraborty S, 1999. Climate change and plant disease management. *Annual Review of Phytopathology* **37**, 399–426.

De Klein CAM, Novoa RSA, Ogle SM *et al.*, 2006. N_2O emissions from managed soils and CO_2 emissions from lime and urea application. In: Eggleston HS, Buendia L, Miwa K, Ngara T, Tanabe K, eds. *2006 IPCC Guidelines for National Greenhouse Gas Inventories. Volume 4. Agriculutre, Forestry and Other Land Use*, [http://www.ipcc-nggip.iges.or.jp/public/2006gl/pdf/4_Volume4/V4_11_Ch11_N2O&CO2.pdf].

Dumalasová V, Bartoš P, 2009. Will climatic changes enhance the risk of *Tilletia indica* in Europe? *Plant Protection Science* **45**, S38–40.

Eastburn DM, McElrone AJ, Bilgin DD, 2011. Influence of atmospheric and climatic change on plant-pathogen interactions. *Plant Pathology* **60**, 54–69.

Evans N, Baierl A, Semenov MA, Gladders P, Fitt BDL, 2008. Range and severity of a plant disease increased by global warming. *Journal of the Royal Society Interface* **5**, 525–31.

Evans N, Butterworth MH, Baierl A *et al.*, 2010. The impact of climate change on disease constraints on production of oilseed rape. *Food Security* **2**, 143–56.

Eyal Z, 1999. The *Septoria tritici* and *Stagonospora nodorum* blotch diseases of wheat. *European Journal of Plant Pathology* **105**, 629–41.

FAO (Food and Agriculture Organisation of the United Nations), 2009. *1.02 Billion People Hungry; One Sixth of Humanity Undernourished – More Than Ever Before* [http://www.fao.org/news/story/en/item/20568/icode/].

Fitt BDL, Doughty KJ, Gladders P, Steed JM, Sutherland KG, 1998. Diagnosis of light leaf spot (*Pyrenopeziza brassicae*) on winter oilseed rape (*Brassica napus*) in the UK. *Annals of Applied Biology* **133**, 155–66.

Fitt BDL, Brun H, Barbetti MJ, Rimmer SR, 2006. World-wide importance of phoma stem canker (*Leptosphaeria maculans* and *L. biglobosa*) on oilseed rape (*Brassica napus*). *European Journal of Plant Pathology* **114**, 3–15.

Fitt BDL, Hu BC, Li ZQ *et al.*, 2008. Strategies to prevent spread of *Leptosphaeria maculans* (phoma stem canker) onto oilseed rape crops in China; costs and benefits. *Plant Pathology* **57**, 652–64.

Flood J, 2010. The importance of plant health to food security. *Food Security* **2**, 215–31.

Forbes GA, Simon R, 2007. Implications for a warmer, wetter world on the late blight pathogen: how CIP efforts can reduce risk for low-input potato farmers. *Journal of SAT Agricultural Research* **4**, 1–34.

Garland JA, 1977. The dry deposition of sulphur to land and water surfaces. *Proceedings of the Royal Society of London, Series A* **354**, 245–68.

Garrett KA, Dendy SP, Frank EE, Rouse MN, Travers SE, 2006. Climate change effects on plant disease: genomes to ecosystems. *Annual Review of Phytopathology* **44**, 489–509.

Ghini R, Hamada E, Gonçalves RRV, Gasparotto L, Pereira JCR, 2007. Análise de risco das mudanças climáticas globais sobre a sigatoka-negra da bananeira no Brasil. *Fitopatologia Brasileira* **32**, 197–204.

Gilles T, Evans N, Fitt BDL, Jeger MJ, 2000. Epidemiology in relation to methods for forecasting light leaf spot (*Pyrenopeziza brassicae*) severity on winter oilseed rape (*Brassica napus*) in the UK. *European Journal of Plant Pathology* **106**, 593–605.

Gladders P, Evans N, Marcroft SJ, Pinochet X, 2006. Dissemination of information about management strategies and changes in farming practices for the exploitation of resistance to *Leptosphaeria maculans* (phoma stem canker) in oilseed rape cultivars. *European Journal of Plant Pathology* **114**, 117–26.

Glendining MJ, Dailey AG, Williams AG, van Evert FK, Goulding KWT, Whitmore AP, 2009. Is it possible to increase the sustainability of arable and ruminant agriculture by reducing inputs? *Agricultural Systems* **99**, 117–25.

Gregory PJ, Johnson SN, Newton AC, Ingram JS, 2009. Integrating pests and pathogens into the climate change/food security debate. *Journal of Experimental Botany* **60**, 2827–38.

Hardwick NV, Jones DR, Slough JE, 2001. Factors affecting diseases of winter wheat in England and Wales, 1989–98. *Plant Pathology* **50**, 453–62.

Hollomon DW, Brent KJ, 2009. Combating plant diseases – the Darwin connection. *Pest Management Science* **65**, 1156–63.

Huang YJ, Evans N, Li ZQ *et al.*, 2006. Temperature and leaf wetness duration affect phenotypic expression of *Rlm6*-mediated resistance to *Leptosphaeria maculans* in *Brassica napus*. *New Phytologist* **170**, 129–41.

Jackson J, Li Y, Passant N, Thistlethwaite G, Thomson A, Cardenas L, 2007. *Greenhouse Gas Inventories for England, Scotland, Wales and Northern Ireland: 1990–2005*. Didcot, UK: AEA Environment and Technology [http://nora.nerc.ac.uk/2230/].

Jeger MJ, Pautasso M, 2008. Plant disease and global change – the importance of long-term data sets. *New Phytologist* **177**, 8–11.

King JE, 1977. Surveys of diseases of winter wheat in England and Wales 1970–75. *Plant Pathology* **26**, 8–20.

Luo Y, Tebeest DO, Teng PS, Fabellar NG, 1995. Simulation studies on risk analysis of rice leaf blast epidemics associated with global climate change in several Asian countries. *Journal of Biogeography* **22**, 673–8.

Mahmuti M, West JS, Watts J, Gladders P, Fitt BDL, 2009. Controlling crop disease contributes to both food security and climate change mitigation. *International Journal of Agricultural Sustainability* **7**, 189–202.

Nolan S, Cooke BM, Monahan FJ, 1999. Studies on the interaction between *Septoria tritici* and *Stagonospora nodorum* in wheat. *European Journal of Plant Pathology* **105**, 917–25.

Oerke EC, 2006. Crop losses to pests. *Journal of Agricultural Science* **144**, 31–43.

Pachauri RK, Reisinger A, 2007. *Climate Change 2007: Synthesis Report Contribution of Working Groups I, II and III to the Fourth Assessment Report of the Intergovernmental Panel on Climate Change*. Geneva, Switzerland: IPCC.

Polley RW, Thomas MR, 1991. Surveys of diseases of winter wheat in England and Wales, 1976–88. *Annals of Applied Biology* **119**, 1–20.

Schmidhuber J, Tubiello FN, 2007. Global food security under climate change. *Proceedings of the National Academy of Sciences, USA* **104**, 19703–8.

Semenov MA, 2009. Impacts of climate change on wheat in England and Wales. *Journal of the Royal Society Interface* **6**, 343–50.

Semenov MA, Stratonovitch P, 2010. The use of multi-model ensembles from global climate models for impact assessments of climate change. *Climate Research* **41**, 1–14.

Sen A, 1999. *Development as Freedom*. Oxford, UK: Oxford University Press.

Shaw MW, 2009. Preparing for changes in plant disease due to climate change. *Plant Protection Science* **45**, S3–10.

Shaw MW, Osborne TM, 2011. Geographic distribution of plant pathogens in response to climate change. *Plant Pathology* **60**, 31–43.

Shaw MW, Bearchell SJ, Fitt BDL, Fraaije BA, 2008. Long-term relationships between environment and abundance in wheat of *Phaeosphaeria nodorum* and *Mycosphaerella graminicola*. *New Phytologist* **177**, 229–38.

Smith P, Olesen JE, 2010. Synergies between the mitigation of, and adaptation to, climate change in agriculture. *Journal of Agricultural Science* **148**, 543–52.

Stern N, 2007. *The Economics of Climate Change: The Stern Review*. Cambridge, UK: Cambridge University Press.

Strange RN, Scott PR, 2005. Plant disease: a threat to global food security. *Annual Review of Phytopathology* **43**, 83–116.

Stukenbrock EH, McDonald BA, 2008. The origins of plant pathogens in agro-ecosystems. *Annual Review of Phytopathology* **46**, 75–100.

Sturrock RN, Frankel SJ, Brown AV *et al.*, 2011. Climate change and forest diseases. *Plant Pathology* **60**, 133–49.

Welham SJ, Turner JA, Gladders P, Fitt BDL, Evans N, Baierl A, 2004. Predicting light leaf spot (*Pyrenopeziza brassicae*) risk on winter oilseed rape (*Brassica napus*) in England and Wales, using survey, weather and crop information. *Plant Pathology* **53**, 713–24.

West JS, Kharbanda PD, Barbetti MJ, Fitt BDL, 2001. Epidemiology and management of *Leptosphaeria maculans* (phoma stem canker) on oilseed rape in Australia, Canada and Europe. *Plant Pathology* **50**, 10–27.

Whelpdale DM, 1992. An overview of the atmospheric sulphur cycle. In: Howarth RW, Stewart JWB, Ivanov MB, eds. *SCOPE 48 – Sulphur Cycling on the Continents: Wetlands, Terrestrial Ecosystems, and Associated Water Bodies*. New York, USA: Wiley, 5–26.

Zhao FJ, Knights JS, Hu ZY, McGrath SP, 2003. Stable sulfur isotope ratio indicates long-term changes in sulfur deposition in the Broadbalk experiment since 1845. *Journal of Environmental Quality* **32**, 33–9.

Zhou Y, Fitt BDL, Welham SJ, Gladders P, Sansford CE, West JS, 1999. Effects of severity and timing of stem canker (*Leptosphaeria maculans*) symptoms on yield of winter oilseed rape (*Brassica napus*) in the UK. *European Journal of Plant Pathology* **105**, 715–28.