Range and severity of a plant disease increased by global warming

Neal Evans[1],*, Andreas Baierl[1,2], Mikhail A. Semenov[1], Peter Gladders[3] and Bruce D. L. Fitt[1]

[1]*Rothamsted Research, Harpenden, Hertfordshire AL5 2JQ, UK*
[2]*Department of Statistics and Decision Support Systems, University of Vienna, Universitaetsstrasse 5/9, 1010 Vienna, Austria*
[3]*ADAS Boxworth, Battlegate Road, Boxworth, Cambridge CB3 8NN, UK*

Climate change affects plants in natural and agricultural ecosystems throughout the world but little work has been done on the effects of climate change on plant disease epidemics. To illustrate such effects, a weather-based disease forecasting model was combined with a climate change model predicting UK temperature and rainfall under high- and low-carbon emissions for the 2020s and 2050s. Multi-site data collected over a 15-year period were used to develop and validate a weather-based model forecasting severity of phoma stem canker epidemics on oilseed rape across the UK. This was combined with climate change scenarios to predict that epidemics will not only increase in severity but also spread northwards by the 2020s. These results provide a stimulus to develop models to predict the effects of climate change on other plant diseases, especially in delicately balanced agricultural or natural ecosystems. Such predictions can be used to guide policy and practice in adapting to effects of climate change on food security and wildlife.

Keywords: climate change scenarios; *Leptosphaeria maculans*; phoma stem canker; plant disease epidemiology; weather-based disease forecasts

1. INTRODUCTION

Climate change is affecting plants in natural and agricultural ecosystems throughout the world (Stern 2007). However, little work has been done to model the effects of predicted twenty-first-century climate change on plant disease epidemics (Garrett *et al.* 2006). Changing weather (e.g. temperature, rainfall) can induce severe plant disease epidemics (Coakley *et al.* 1999; Chakraborty 2005), which threaten food security if they affect staple crops (Luo *et al.* 1995; Chakraborty *et al.* 2000; Anderson *et al.* 2004) and can damage landscapes if they affect amenity species (Brasier & Scott 1994; Bergot *et al.* 2004). Severity of human, animal and plant disease epidemics is greatly affected by climatic factors, especially temperature and rainfall (Wint *et al.* 2002; Fitt *et al.* 2006*a*; Thomson *et al.* 2006; Bosch *et al.* 2007). Therefore, weather-based forecasts have been developed to guide control strategies for many important diseases worldwide (Wint *et al.* 2002; Garrett *et al.* 2006; Thomson *et al.* 2006). There is now an opportunity to link weather-based plant disease forecasts with recent climate change models, to predict the effects of climate change scenarios on the distribution and severity of plant disease epidemics.

Much discussion on the impact of climate change on plant disease epidemics has used qualitative, rule-based reasoning, which cannot easily accommodate the complex host–pathogen–environment interactions involved (Coakley *et al.* 1999; Anderson *et al.* 2004). Modelling approaches have included those matching existing climates in one region with climates predicted for another (Brasier & Scott 1994) or combinations of simulation models for crop growth and disease development (Luo *et al.* 1995). Before 1999, no work had used predicted climate variables generated by new more sophisticated general circulation models (GCMs); most studies had relied on predictions of fixed changes in temperature and rainfall (Coakley *et al.* 1999). Recently, GCMs have been used to predict the increase in range of *Phytophthora cinnamomi* (Bergot *et al.* 2004). Whereas empirical modelling has been used to produce disease epidemic models for combining with climate change predictions (Chakraborty *et al.* 1998; Coakley *et al.* 1999), few models have been based on datasets including both regional and seasonal variations that are sufficiently extensive to allow both model development and validation.

Phoma stem canker (blackleg, *Leptosphaeria maculans*) is an internationally important disease of wild and cultivated brassicas; during each growing season, it causes yield losses of millions of tonnes of brassica oilseed and vegetable crops in Europe, North America, Australia and Africa (Fitt *et al.* 2006*b*). It has spread across North America and eastern Europe in the

Table 1. Parameters/terms used in developing phoma stem canker models.

parameter/variable	term/abbreviation	unit	explanation
15 Jul (day 196)	start of optimized prediction period	day of the year	selected by model optimization (approx. date of harvest)
26 Sep (day 289)	end of optimized prediction period	day of the year	selected by model optimization (approx. date for establishment of new crop, sown late Aug/early Sep)
R_{sum}	total rainfall	mm	total rainfall for the period 15 Jul–26 Sep
T_{max}	mean maximum temperature	°C	mean maximum daily temperature for the period 15 Jul–26 Sep
Dl	date, start of leaf spotting[a]	day of the year[b]	date (autumn) when 10% of plants in crop affected by phoma leaf spotting
Dc	date, start of stem canker[a]	day of the year[b]	date (spring) when 10% of plants in crop affected by phoma stem canker
Dh	date of pre-harvest sample	day of the year[b]	date in the period between Dc and harvest
Sc	severity of stem canker[a]	0–4 scale	severity of phoma stem canker on date Dh
$T' = \sum_{i=a}^{i=b} T_i$	thermal time	°C-days	accumulated °C-days above 0°C between date a and date b; to estimate Dc and Sc, multiplied by a constant depending on cultivar resistance
	climate		mean weather over a 30-year period (comparisons made with the period 1960–1990)
	HadCM3		HadCM3 is a coupled atmosphere–ocean global climate model with a spatial resolution of 300 km developed at the Hadley Centre
	HadRM3		HadRM3 is a regional climate model with a spatial resolution of 50 km developed at the Hadley Centre
	LO		low-emission scenario (less than 1100 Gt carbon per annum)
	HI		high-emission scenario (1450–1800 Gt carbon per annum)
	LARS-WG		stochastic weather generator, used to generate climate change scenarios

[a] For these parameters, the values predicted by the model (p) are compared with the observed values (o).
[b] Dates used are assessed as 'days after 15 July'.

last 20 years and now threatens 10 Mha of highly susceptible oilseed and vegetable brassicas in China, mostly grown by subsistence farmers. Temperature and rainfall affect not only the development of the pathogen (Huang *et al.* 2005) but also the resistance response of the host (Huang *et al.* 2006). Globally, the most severe epidemics occur in oilseed rape (*Brassica napus*) growing areas of Australia, with their Mediterranean climate, where susceptible crops can be destroyed by the disease (Howlett *et al.* 2001; Sprague *et al.* 2006). However, much of the world's oilseed rape crop is grown in cooler climates. In the UK, the most severe phoma stem canker epidemics occur in southern England; in Scotland, where the climate is colder, phoma leaf spotting does occur but damaging phoma stem cankers do not subsequently develop. To illustrate the effects of predicted climate change on the range and severity of plant disease epidemics, weather-based models predicting the development of phoma stem canker epidemics were combined with climate change models to generate scenarios for the future severity of epidemics in the UK.

2. PHOMA STEM CANKER MODEL DEVELOPMENT AND VALIDATION

A weather-based model to describe the development of phoma stem canker epidemics was constructed, using disease and weather data from 40 winter oilseed rape field experiments done under different weather conditions in growing seasons between 1992–1993 and 2001–2002 at a wide range of UK sites, each with one to three cultivars (electronic supplementary material, table 1). The development of a phoma stem canker epidemic was considered in three stages. In the first stage, the date when phoma leaf spot epidemics start in autumn was predicted from the preceding summer weather data. Since phoma stem canker is a monocyclic disease (one cycle per growing season), the date in autumn when leaf spotting starts (Dl; table 1; figure 1*d*) is a crucial factor affecting the severity of phoma stem canker epidemics on stems the following summer (West *et al.* 2001). The date when phoma leaf spotting starts in autumn (Dl) was estimated from weekly records of disease incidence (percentage of plants affected) between the end of September and mid-December on 25 randomly selected plants per crop (40 datasets for different cultivars in different seasons, untreated with fungicides), using linear interpolation between the two assessment dates with disease incidences that were above and below the 10% incidence threshold. Multiple linear regression was used to examine the relationship between the start of phoma leaf spot in autumn (Dl) and the temperature and rainfall during the intercrop period between the harvest of the previous crop and the establishment of the new crop, because maturation in pseudothecia (fruiting bodies; figure 1*a*) on crop debris
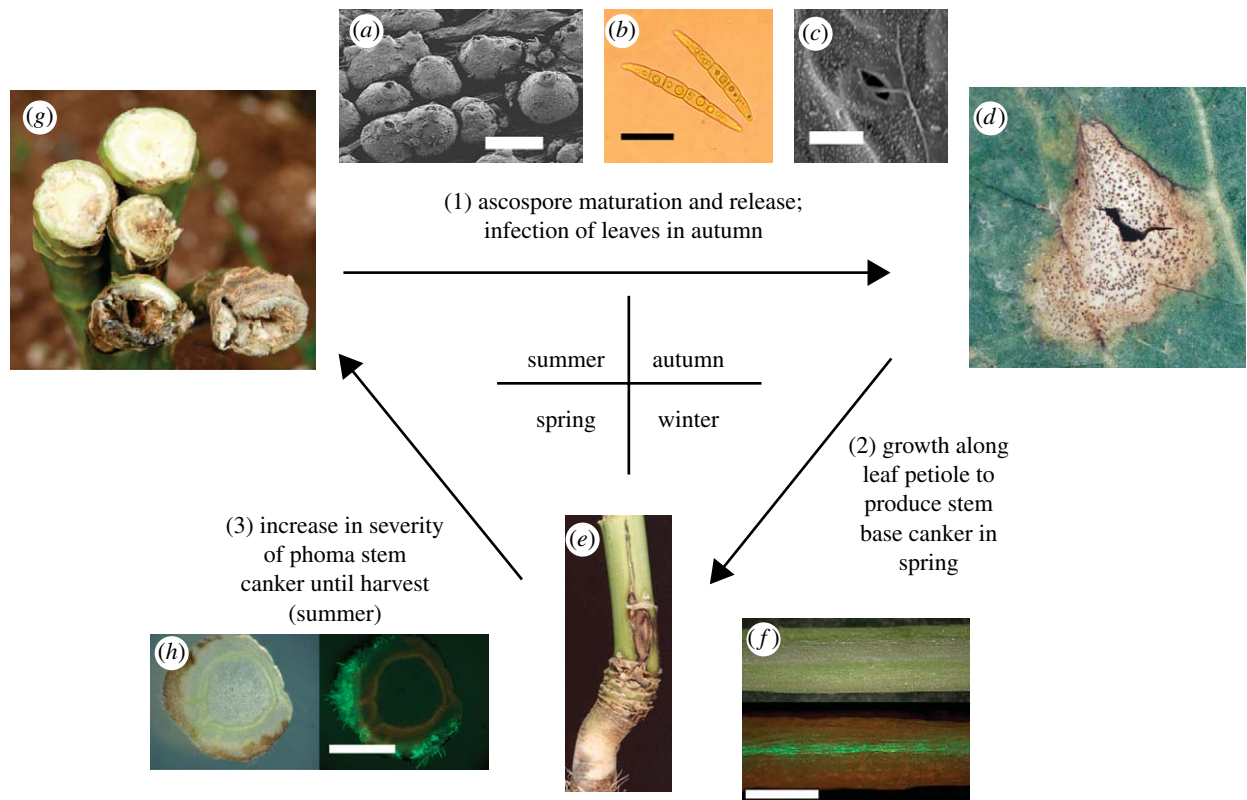
Figure 1. Stages in modelling phoma stem canker epidemics on winter oilseed rape in the UK. (1) Predicting the date in autumn when 10% of plants have (*d*) phoma leaf spotting (*Dl*), from data for temperature and rainfall which affect maturation of (*a*) *L. maculans* pseudothecia (scale bar, 300 μm) on affected oilseed rape stems after harvest to produce (*b*) ascospores (scale bar, 10 μm) that infect leaves through (*c*) stomata (scale bar, 10 μm). (2) Predicting the first date in spring when 10% of plants have (*e*) phoma stem canker lesions (*Dc*), from autumn/winter temperature data and cultivar resistance which affect the rate of pathogen growth along the leaf petiole ((*f*) shown with GFP-labelled *L. maculans* growing along the centre of symptomless petiole; scale bar, 5 mm). (3) Predicting increase in severity of phoma stem canker lesions until (*g*) harvest (*Sc*), from spring/summer temperature data and cultivar resistance rating which affect growth through oilseed rape stem tissues ((*h*) shown with GFP-labelled *L. maculans* growing in stem cortex; scale bar, 5 mm). Images (*f*) and (*h*) each show the same tissue viewed under white light or with a GFP filter.

of *L. maculans* ascospores (figure 1*b*) and infection of oilseed rape leaves through stomata (figure 1*c*) are dependent on both temperature and wetness. The dates for the start and end of this period, selected by model optimization over a range of dates to give the optimal regression fit, were 15 July and 26 September, respectively, and the dates in the growing season were then expressed as days after 15 July. These dates are assumed to represent the current average dates of harvest in summer (15 July, when the maturation of *L. maculans* ascospores starts) and the establishment of the new crop in autumn (26 September, when new leaves are available to be infected). Location and year (growing season) were included as random effects to account for the hierarchical nature of the data. The analysis of variance was used to investigate differences between cultivars in the start of phoma leaf spotting.

In the second stage, the date when phoma canker starts to develop on stems in spring (*Dc*; figure 1*e*) was predicted from the start of phoma leaf spotting (*Dl*$_\mathrm{p}$) and accumulated thermal time (°C-days) from *Dl*$_\mathrm{p}$, which affects the rate of progress of the pathogen along leaf petioles to the stem in autumn/winter (figure 1*f*; Sun *et al.* 2000). The date when phoma stem canker starts in spring (*Dc*) was estimated from weekly records of stem canker incidence (percentage of plants affected) in March, April

and May (18 datasets). This work suggested that the thermal time accumulated between the two dates (*Dl* and *Dc*) was dependent on the resistance of the winter oilseed rape cultivar to *L. maculans*, so the dates of first appearance of phoma stem canker were estimated separately for cultivars with high-(6–9) and low (1–5)-resistance ratings (www.hgca.com).

In the third stage, the increase in severity of phoma stem canker (*Sc*, 0–4 severity scale; figure 1*g*) was predicted from the start of stem cankers in spring (*Dc*$_\mathrm{p}$), using accumulated thermal time (°C-days) from *Dc*$_\mathrm{p}$, which affects colonization of stem tissues by the pathogen (figure 1*h*). The increase in severity of phoma stem canker in the period before harvest (mean of all plants sampled) was assessed on a 0–4 scale (0, healthy; 4, plant dead; Zhou *et al.* 1999) on plants regularly sampled from untreated crops in the period March to July (32 datasets). The severity of phoma stem canker on the pre-harvest date of sampling (*Sc*) was predicted from *Dc* as a function of thermal time and cultivar resistance to *L. maculans* (Sun *et al.* 2000) by applying a model and treating each experiment as a random effect. Analyses were done with the statistical software GENSTAT (Anon. 2006).

Multiple linear regression, using the 40 datasets (electronic supplementary material, table 1), indicated that mean maximum daily temperature [T_max] and

total rainfall [R_{sum}] between 15 July and 26 September produced the best prediction of the start of the phoma leaf spotting epidemic (Dl_p) for each site and growing season. The mean maximum daily temperature discriminated well between different sites and the sum of rainfall discriminated well between different growing seasons (electronic supplementary material, figure 1). The variables crop sowing date, cultivar resistance to *L. maculans* (www.hgca.com) and proximity to a previous season's oilseed rape crop did not account for additional variation in these data. The relationship was described by the equation

$$Dl_p = 216.5 - 0.24R_{sum} - 4.55\,T_{max}. \qquad (2.1)$$

The 95% CI of the parameters (estimate $\pm$ interval; 216.5 ± 52.84, -0.24 ± 0.08 and -4.55 ± 2.36) show that both T_{max} and R_{sum} were significant ($p < 0.001$). When the observed start of phoma leaf spotting (Dl_o) was regressed against the date predicted by equation (2.1) (Dl_p), 63% of the variance was accounted for (electronic supplementary material, figure 2).

The prediction of the start of phoma stem canker in spring (Dc_p), using the start of phoma leaf spotting in the previous autumn (Dl_p) and a derived function of °C-days (T') from Dl_p, was improved by taking account of cultivar resistance to *L. maculans* (Sun *et al.* 2000). The mean thermal time ($\pm$ s.e.) was $1097(\pm 66.0)$ °C-days for susceptible cultivars (resistance rating 1–5; www.hgca.com, $A = 0$) and $1386(\pm 74.5)$ °C-days for resistant cultivars (resistance rating 6–9; $A = 1$), and Dc_p is the first day where the accumulated thermal time (°C-days) exceeds the threshold ($\min(x)$)

$$Dc_p = \min\left(x \in \{Dl_p, Dl_p+1, \ldots\} \mid \sum_{i=Dl_p}^{x} T_i \geq (1097 + 289A)\right),$$
$$(2.2)$$

where T_i is the temperature in °C on day i and $\sum_{i=Dl_p}^{x} T_i$ is the accumulated °C-days from start of leaf spotting (Dl_p) to day x ($= Dl_p$, $Dl_p + 1$, …).

The increasing severity of phoma stem canker in the period until harvest (Sc) was predicted as a function of °C-days since the start of stem canker, with different slopes for susceptible ($0.00135(\pm 0.00011)$) and resistant ($0.001(\pm 0.00017)$) cultivars. The equation to predict the canker severity on a specific date (e.g. last sample before harvest) was

$$Sc_p = \sum_{i=Dc_p}^{Dh} T_i(0.00135 - 0.00035A), \qquad (2.3)$$

where Sc_p is the mean stem canker severity of plants (0–4 scale; Zhou *et al.* 1999) and Dh is the pre-harvest sample date.

The weather-based model for describing the development of phoma stem canker epidemics was validated with data from 21 winter oilseed rape field experiments done under different weather conditions in growing seasons between 2004–2005 and 2006–2007 at a wide range of UK sites, each with 1–17 cultivars (electronic supplementary material, table 2). Linear regressions were done for the observed values against the predicted values for the start of phoma leaf spotting (autumn),

start of phoma stem canker (spring) and severity of phoma stem canker (pre-harvest). The values of the percentage of variance accounted for were used as measures of the goodness of fit of the model. When the observed start of phoma leaf spotting (Dl_o) was regressed against the date predicted by the model (Dl_p), 33% of the variance was accounted for (electronic supplementary material, figure 3a). Regression of observed (Dc_o) against predicted (Dc_p) start of stem canker accounted for 21% of the variance (electronic supplementary material, figure 3b). Regression of observed (Sc_o) against predicted (Sc_p) pre-harvest severity of phoma stem canker accounted for 36% of the variance, using data from the field experiments (electronic supplementary material, figure 3c).

3. MODELLING THE EFFECTS OF CLIMATE CHANGE ON PHOMA STEM CANKER EPIDEMICS

To predict the effects of climate change on the range and severity of phoma stem canker epidemics, we used UKCIP02 climate change predictions for the years 2020 and 2050 (Hulme *et al.* 2002), by comparison with a baseline period (1960–1990), under high (HI)- and low (LO)-emission scenarios as defined by global IPCC emission scenarios (Nakicenovic 2000). The UKCIP02 climate change predictions for the year 2080 were available but not used since predicted average values are outside the current boundary values within which the phoma stem canker model was validated. The UKCIP02 scenarios are based on the HadCM3 global and HadRM3 regional climate models (Collins *et al.* 2001). The HadRM3 model has a horizontal resolution of $0.44° \times 0.44°$ (50 km). For each grid cell, UKCIP02 provides predicted changes in monthly climate variables. A weather generator (LARS-WG; Semenov & Barrow 1997) produced 70 yearly site-specific daily weather datasets for both 2020 and 2050 for each emission scenario, based on UKCIP02 projections and daily output from the HadRM3 climate model. For selected sites (15 locations evenly distributed over the UK), we calculated weather generator parameter sets, representing the baseline climate, using spatial interpolation of parameters, previously derived from the observed daily weather for 1960–1990 (Semenov & Brooks 1999). To adjust 1960–1990 parameters for climate change predictions, changes in mean and variability of climate variables for a site were required. The mean monthly changes in total rainfall, maximum and minimum temperatures were provided by UKCIP02. Changes in duration of monthly mean dry and wet series were calculated using HadRM3 daily rainfall output. New sets of parameters adjusted for changed climate were used to generate 70 yearly site-specific daily weather sets for each emission scenario for both 2020 and 2050 at each location.

These data were used as input in the phoma stem canker model to predict the effects of climate change on the start of phoma leaf spotting (Dl_p), start of stem canker (Dc_p) and severity of stem canker at harvest (Sc_p; initially assumed to be on 15 July). The results of simulations at 15 sites were spatially interpolated over
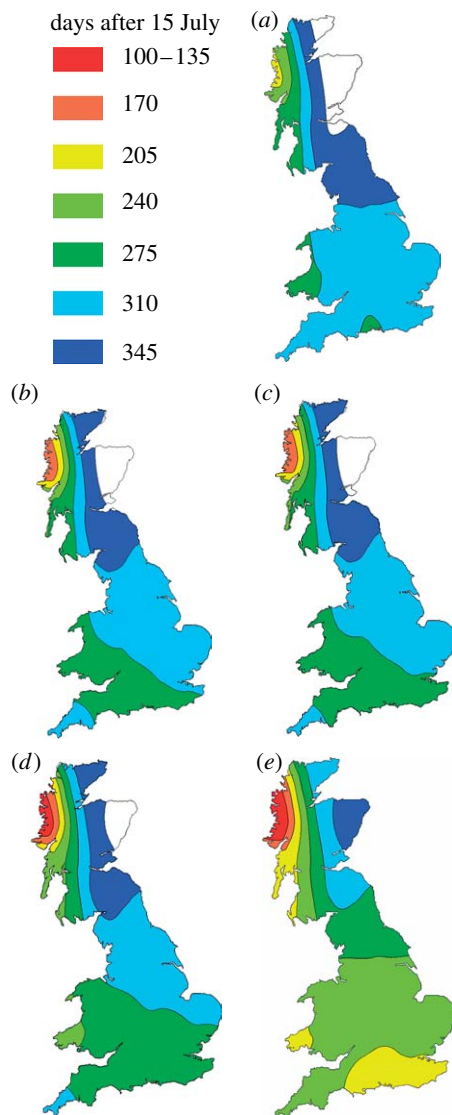
Figure 2. Predicted start of phoma stem canker (*L. maculans*) in spring. Predicted number of days after 15 July until the date in spring when the first phoma stem canker is observed on winter oilseed rape crops (mean of resistant and susceptible cultivars) affected with *L. maculans* for (*a*) baseline 1960–1990, (*b*) 2020LO, (*c*) 2020HI, (*d*) 2050LO and (*e*) 2050HI climates. Areas unaffected by the disease are white.

Figure 3. Predicted severity of phoma stem canker (*L. maculans*) at harvest (*Sc*) on winter oilseed rape crops (mean of resistant and susceptible cultivars) for (*a*) baseline 1960–1990, (*b*) 2020LO, (*c*) 2020HI, (*d*) 2050LO and (*e*) 2050HI climates. Stem canker severity on a 0–4 scale (0, no disease; 4, plant dead; Zhou *et al.* 1999). Areas unaffected by the disease are white.

the UK. As data were skewed, the median date rather than the mean date for the start of phoma leaf spotting was calculated for each scenario (the date was calculated as the number of days from 15 July). From this predicted date (Dl_p), the predicted start of phoma stem canker (Dc_p) and phoma stem canker severity at harvest (Sc_p) were calculated. The start of phoma leaf spotting in autumn predicted for 2020 and 2050 using the climate change model scenarios under high- or low-emission scenarios was only 5–10 or 10–15 days, respectively, earlier than in the 1960–1990 period (electronic supplementary material, figure 4). Since the start of leaf spotting is dependent on both temperature and rainfall (equation (2.1); West *et al.* 2001; Huang *et al.* 2005), the effects of increasing summer temperature were counteracted by the effects of decreasing summer rainfall.
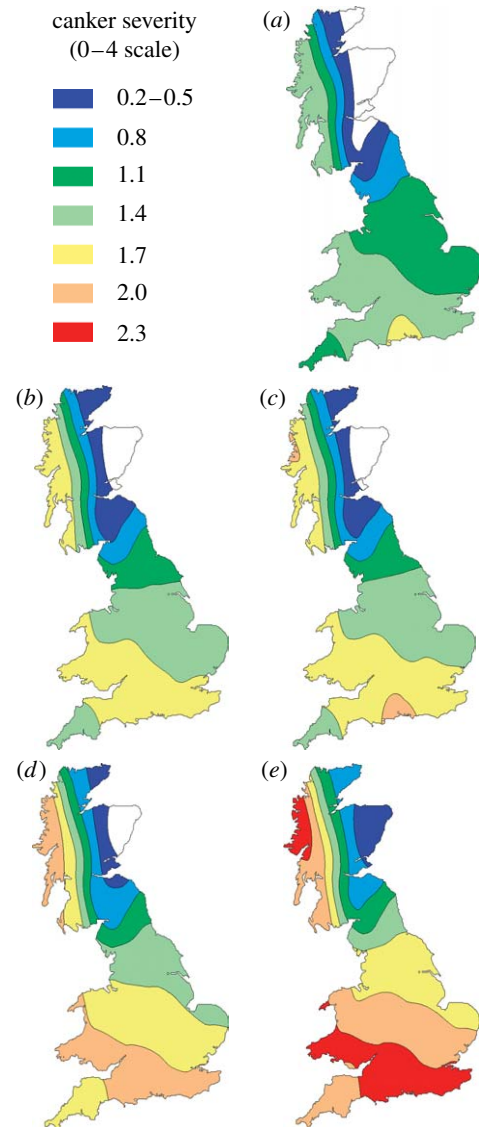
There was a large effect of predicted climate change on the start of phoma stem canker in spring, with predicted dates often 80 days earlier than during 1960–1990 (figure 2). The range of the damaging stem canker phase of epidemics was predicted to extend northwards from England into oilseed rape growing areas in eastern Scotland (white area in figure 2, currently unaffected by phoma stem canker). Furthermore, the predicted severities of phoma stem canker at harvest for 2020 and 2050 were much greater than during 1960–1990; the UK maximum mean severity increased from 1.7 (1960–1990) to 2.0 (2020) and 2.3 (2050) on the 0–4 scale for a harvest date of 15 July (figure 3). These increases in severity of epidemics still occurred if harvest dates were earlier, as predicted for sites in southern England, when harvest dates under these scenarios were estimated for Rothamsted from predicted wheat harvest dates (table 2). The effects of

Table 2. Effect of cultivar resistance on predicted number of days after 15 July until the date in spring when the first phoma stem canker is observed and the severity of stem canker at harvest (on 15 July or on predicted harvest date) at Rothamsted, Hertfordshire, UK for winter oilseed rape crops affected with *L. maculans* for 2020 and 2050 climates under low (LO)- and high (HI)-emission scenarios.

cultivar resistance (rating[a])	emission scenario				
	baseline	2020 LO	2020 HI	2050 LO	2050 HI
date of first phoma stem canker (days after 15 Jul)					
resistant (6–9)	309	295	292	281	264
susceptible (1–5)	279	264	261	246	227
mean	296	275	278	257	242
phoma stem canker severity at harvest on 15 Jul (day 196; 0–4 scale)					
resistant (6–9)	1.14	1.43	1.49	1.71	2.04
susceptible (1–5)	1.55	1.83	1.88	2.11	2.44
mean	1.36	1.63	1.69	1.98	2.31
phoma stem canker severity at predicted harvest date (0–4 scale)					
predicted harvest date[b]		190	186	185	178
resistant (6–9)		1.27	1.22	1.44	1.56
susceptible (1–5)		1.68	1.63	1.83	1.97
mean		1.47	1.43	1.64	1.77

[a] Resistance rating on a 1–9 scale (www.hgca.com).
[b] Day of the year, obtained from the predicted harvest date for winter wheat under these climate change scenarios and difference in harvest dates between winter wheat and winter oilseed rape in °C-days.

climate change on the range and severity of the disease can already be observed by comparing the values for periods 1960–1990 and 1975–2005 at Rothamsted, UK; the simulated start of canker is 10 days earlier during 1975–2005 than during 1960–1990 and canker severity is 18% greater. These large effects on the start and severity of stem canker were related to predicted increase in temperature, especially in winter, which greatly influences this stage of epidemic development (equation (2.2); Sun *et al.* 2000), although the effects are less on resistant than on susceptible cultivars (table 2).

4. DISCUSSION

These results demonstrate how predicted global warming can increase the range and severity of plant diseases of worldwide importance within the next 20 years. The effects of climate change may be on the pathogen, the host or the host–pathogen interaction (Coakley *et al.* 1999; Huang *et al.* 2005, 2006; Garrett *et al.* 2006). The long-term effects of human-made environmental change on plant diseases may be masked by short-term seasonal fluctuations (Bearchell *et al.* 2005; Fitt *et al.* 2006c). To ignore such effects may result in devastating epidemics on staple food crops, with far-reaching socio-economic consequences, or on important plants in natural ecosystems, threatening wildlife (Luo *et al.* 1995; Chakraborty *et al.* 2000; Anderson *et al.* 2004).

The evidence that climate change will increase the range and severity of phoma stem canker is supported by the observations that phoma stem canker epidemics are currently most severe in oilseed rape growing regions with Mediterranean climates (e.g. in Australia or France; Howlett *et al.* 2001; Fitt *et al.* 2006a). There is a need for new cultivars destined for the future UK market to be tested under such climates. Whereas some predicted effects of climate change may be anticipated by qualitative reasoning (Coakley *et al.* 1999), others,

such as the small effect on the date of phoma leaf spotting in autumn, may not. It is important to recognize that climate change effects on complex host–pathogen–environment interaction may also decrease severity of epidemics (Chakraborty *et al.* 1998).

These predictions about the effects of climate change on range and severity of phoma stem canker were possible only because of the availability of both knowledge about the epidemiology of this monocyclic disease and datasets on epidemic development and weather for a range of sites and growing seasons. These datasets encompass a wide range of dates of establishment in autumn (late August to mid-October) and harvest dates in summer (early July to mid-August). To improve accuracy of predictions, there is a need to improve the model by further validation against data obtained in a wide range of climates, such as those predicted for the UK, and by incorporating into the model a weather-based crop growth model to describe the effects of climate change on crop growth that influence disease development (Steed *et al.* 2007). A high priority over the next decade should be the collation of accurate disease and weather data and development of models to forecast the effects of climate change on other plant diseases to provide the necessary foresight for strategic adaptation to climate change. These models can guide policy and practice to counter such emerging threats to delicately balance natural and agricultural ecosystems.

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