Climate change and phoma stem canker

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Summary

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.

Introduction

Climate change is affecting plants in natural and agricultural ecosystems throughout the world (Stern, 2007). However, little work has been done to model effects of predicted 21st 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), which threaten food security if they affect staple crops (Chakraborty *et al.*, 2000) and can damage landscapes if they affect amenity species (Bergot *et al.*, 2004). Climatic factors, especially temperature and rainfall, can affect the severity of human, animal and plant disease epidemics (Fitt *et al.*, 2006a). Therefore weather-based forecasts have been developed to guide control strategies for many important diseases worldwide (Garrett *et al.*, 2006). We now have the opportunity to link weather-based plant disease forecasts with recent climate change models, to predict the possible 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). For example, some of the modelling approaches taken have matched existing climates in one region to climates predicted for another or used combinations of simulation models for crop growth and disease development. Before 1999, no work had used predicted climate variables generated by new, more sophisticated General Circulation Models (GCM); most studies had relied on predictions of fixed changes in temperature and rainfall (Coakley *et al.*, 1999). Recently, GCM 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 data sets including both regional and seasonal variation that are sufficiently extensive to allow both model development and validation.

Phoma stem canker

Phoma stem canker (blackleg, *Leptosphaeria maculans*) is an internationally important disease of wild and cultivated brassicas; each growing season it causes yield losses of millions of tonnes in brassica oilseed and vegetable crops in Europe, North America, Australia and Africa (Fitt *et al.*, 2006b). It has spread across North America and eastern Europe in the last 20 years and now threatens 10M ha of highly susceptible oilseed and vegetable brassicas in China, mostly grown by subsistence farmers. Temperature and rainfall affect not only 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 (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 effects of predicted climate change on the range and severity of plant disease epidemics, weather-based models predicting development of phoma stem canker epidemics were combined with climate change models to generate scenarios for the future severity of epidemics in the UK.

Phoma epidemic model development and validation

A weather-based model to describe 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 1992/93 and 2001/02 at a wide range of UK sites, each with 1-3 cultivars (Table 1). The development of a phoma stem canker epidemic was considered in three stages (Figure 1). In the first stage, the date when phoma leaf spot epidemics start in autumn was predicted from the preceding summer weather data (Evans *et al.*, 2007). Since phoma stem canker is a monocyclic disease (one cycle per growing season), the date in autumn when leaf spotting starts is a crucial factor affecting the severity of phoma stem canker epidemics on stems the following summer (West *et al.*, 2001). In the second stage, the date when phoma canker starts to develop on stems in spring was predicted from the start of phoma leaf spotting and accumulated thermal time (°C-days) (Evans *et al.*, 2007), which affects the rate of growth of the pathogen along leaf petioles to the stem in autumn/winter (Sun *et al.*, 2000). This work suggested that the accumulated thermal time needed before canker onset occurred was dependent on the resistance of the winter oilseed rape cultivar to *L. maculans*, so 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 (Figure 1) was predicted from the start of stem cankers in spring, using accumulated thermal time (°C-days), which affects colonisation of stem tissues by the pathogen (Evans *et al.*, 2007). 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 data sets).

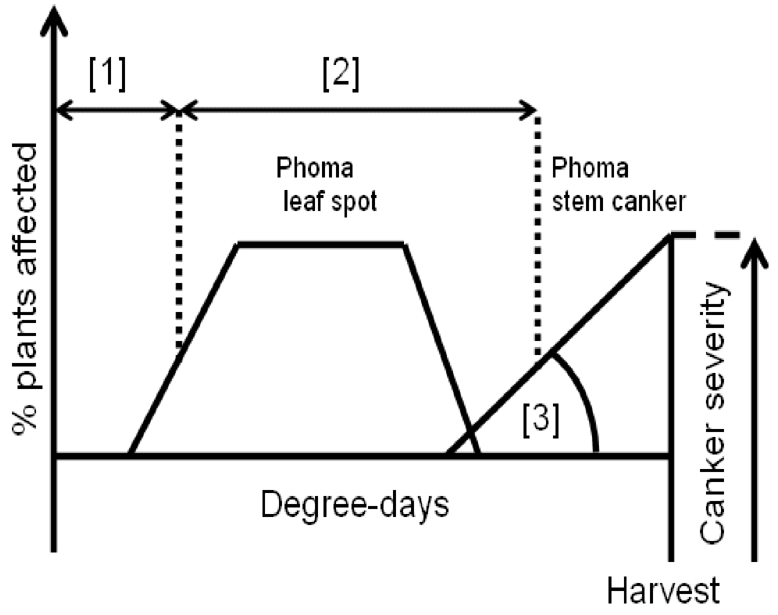

Figure 1. Three phase phoma leaf spot/canker epidemic model, showing [1] date 10% plants affected, [2] date of canker onset in spring and [3] canker severity at harvest.

Model validation

The weather-based model was then validated with data from 21 winter oilseed rape field experiments done under different weather conditions in growing seasons between 2004/05 and 2006/07 at a wide range of UK sites, each with 1-17 cultivars. Linear regressions were done of observed values against predicted values for start of phoma leaf spotting (autumn), start of phoma stem canker (spring) and severity of phoma stem canker (pre-harvest). The values of the % variance accounted for were used as measures of the goodness of fit of the model. When observed start of phoma leaf spotting was regressed against the date predicted by the model, 33% of the variance was accounted for. Although this is low, phoma leaf spot predictions were within 7 days of the predicted date and are therefore of considerable value to those looking at crops and making spray decisions. Regression of observed against predicted start of stem canker accounted for 21% of the variance. Regression of observed against predicted pre-harvest severity of phoma stem canker accounted for 36% of the variance, using data from the field experiments. Again, although the variance accounted for the latter two relationships are not particularly high, this reflects the long timescale of the prediction, the long intervals between field assessments and the use of 'average' weather.

Modelling climate change

To predict the effects of climate change on the range and severity of phoma stem canker epidemics, we used UKCIP02 climate change predictions for 2020 and 2050 (Hulme *et al.*, 2002), by comparison with a baseline period (1960-90), under high (HI) and low (LO) carbon emissions scenarios as defined by global IPCC emissions scenarios (Nakicenovic, 2000). 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° x 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 data sets for both 2020 and 2050 for each emission scenario, based on UKCIP02 projections and daily output from the HadRM3 climate model. We calculated weather generator parameter sets, representing the baseline climate, by using spatial interpolation of parameters, previously derived from observed daily weather for 1960-90 for 15 selected sites that were evenly distributed over the UK (Semenov & Brooks, 1999). To adjust 1960-90 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 temperature 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 for the phoma stem canker model to predict effects of climate change on the start of phoma leaf spotting in autumn, start of stem canker in spring and severity of stem canker at harvest. The results of simulations at 15 sites were spatially interpolated over the UK. 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 1960-90 period (Figure 2). Since the start of leaf spotting is dependent on both temperature and rainfall (Huang *et al.*, 2005; West *et al.*, 2001), the effects of increasing summer temperature were counteracted by effects of decreasing summer rainfall.

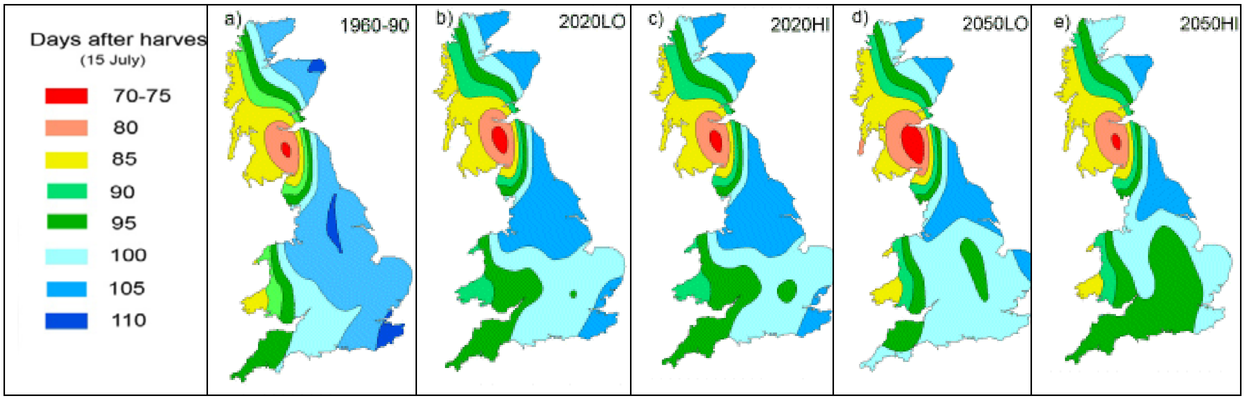

Figure 2. Predicted number of days after harvest in summer (15 July) until the date in autumn when 10% of winter oilseed rape plants are affected with phoma leaf spot (*Leptosphaeria maculans*) for a) baseline 1960-90, b) 2020LO, c) 2020HI, d) 2050LO and e) 2050HI climates under low and high emission scenarios (Evans *et al.*, 2007).

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 in 1960-90 (Figure 3). 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 on Figure, currently unaffected by phoma stem canker). Furthermore, the predicted severities of phoma stem canker at harvest for 2020 and 2050 were much greater than in 1960-1990 with the UK maximum mean severity predicted to increase from 1.7 (1960-90) to 2.0 (2020) and 2.3 (2050) on the 0-4 scale for a harvest date of 15 July (Figure 4). 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 1). Effects of climate change on the range and severity of the disease can already be observed by comparing values for periods 1960-90 and 1975-2005 at Rothamsted, UK where the simulated start of canker is 10 days earlier in 1975-2005 than 1960-90 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 (Sun *et al.*, 2000), although effects are less on resistant than susceptible cultivars (Table 1).

Table 1. 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, for winter oilseed rape crops affected with *Leptosphaeria maculans* for 2020 and 2050 climates under low (LO) and high (HI) emissions scenarios.

Cultivar resistance (rating[1])	Emission scenario				
	Baseline	2020 LO	2020 HI	2050 LO	2050 HI
	Date of first phoma stem canker (days after 15 July)				
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 July (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[2]		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

[1] resistance rating on a 1-9 scale (www.hgca.com)

[2] day of the year, obtained from 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

Implications for policy makers

These results demonstrate how predicted global warming can increase the range and severity of plant diseases of worldwide importance within the next 20 years. Effects of climate change may be on the pathogen, the host or the host-pathogen interaction (Coakley *et al.*, 1999; Garrett *et al.*, 2006; Huang *et al.*, 2006; Huang *et al.*, 2005). Long-term effects of man-made environmental change on plant diseases may be masked by short-term seasonal fluctuations (Bearchell *et al.*, 2005; Fitt *et al.*, 2006c). However, 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 (Chakraborty *et al.*, 2000). These models can guide policy and practice to counter such emerging threats to delicately balanced natural and agricultural ecosystems. 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 recognise that climate change effects on complex host-pathogen-environment interaction may also decrease severity of epidemics (Chakraborty *et al.*, 1998).

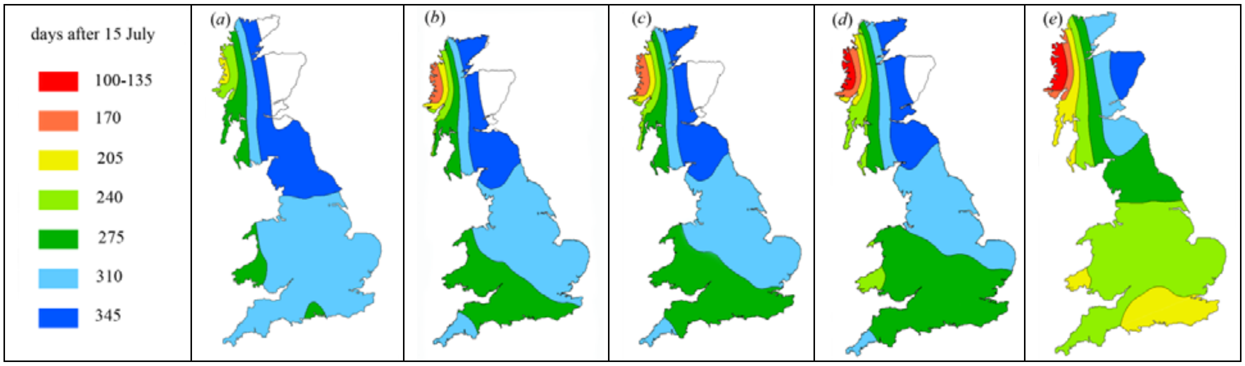

Figure 3. Predicted start of phoma stem canker (*Leptosphaeria maculans*) in spring. Predicted number of days after 15 July until the date in spring when the first phoma stem canker is observed on winter oilseed rape crops (resistant and susceptible cultivars) affected with *Leptosphaeria maculans* for a) baseline 1960-90, b) 2020LO, c) 2020HI, d) 2050LO and e) 2050HI climates under low and high emission scenarios (Evans *et al.*, 2007). Areas unaffected by the disease are white.

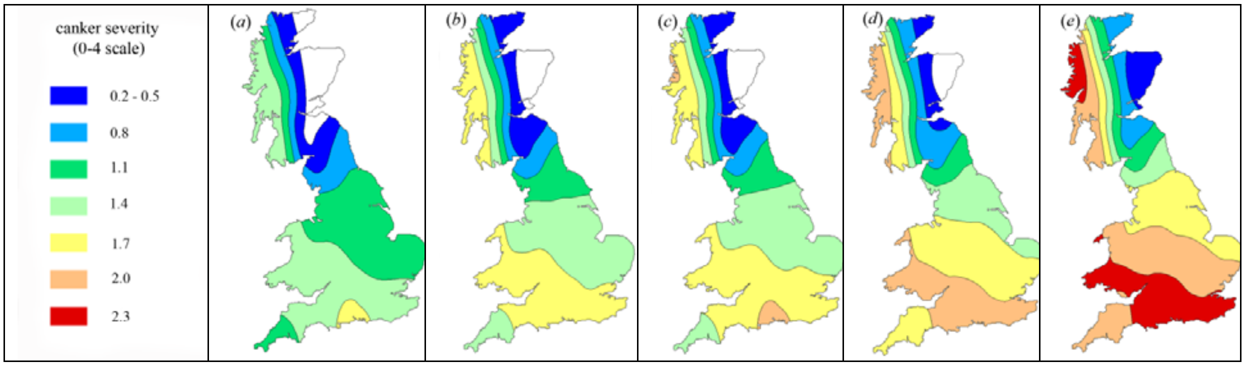

Figure 4. Predicted severity of phoma stem canker (*Leptosphaeria maculans*) at harvest (Sc) of winter oilseed rape crops (resistant and susceptible cultivars) for a baseline 1960-90, b 2020LO, c 2020HI, d 2050LO and e 2050HI climates under low and high emission scenarios (Evans *et al.*, 2007). Stem canker severity on a 0-4 scale (0, no disease; 4, plant dead) (Zhou *et al.*, 1999). Areas unaffected by the disease are white.

Implications for breeders

The evidence that climate change will increase the range and severity of phoma stem canker is supported by observations that phoma stem canker epidemics are currently most severe in oilseed rape growing regions with Mediterranean climates (e.g. in Australia or France; (Fitt *et al.*, 2006a; Sprague *et al.*, 2006). There is a need for new cultivars destined for the future UK market to be tested under such climates. However, it is important to remember that changes in temperature and rainfall affect not only development of the pathogen with respect to spore maturation and release (Huang *et al.*, 2005) but also the resistance response of the host. At increased temperatures, the resistance to *L. maculans* conferred by the oilseed rape major resistance gene *RLm6* was ineffective (Huang *et al.*, 2006).

Implications for growers

The development of the phoma epidemic model provides growers with a new tool to help target fungicide application for the control of phoma stem canker (Evans *et al.*, 2007). An Internet based forecast website has been published that provides growers and their advisors with predicted dates in the autumn when we expect 10% of plants to be affected with phoma leaf spot (http://www.rothamsted.ac.uk/ppi/phoma/), which coincides with the threshold when it is suggested a fungicide application might be considered (Steed *et al.*, 2007a). Predictions are made for 48 sites across the UK and, for example, for the 2007/08 growing season, the predictions ranged from 17 October to 7 November depending on site.

Predictions that phoma stem canker epidemics will become more severe and that we can expect a shift northwards in the range of the disease imply that control of the initial phoma leaf spotting will become even more important that it is currently. This also suggests that growers in the north of England and Scotland should consider phoma control in combination with control of light leaf spot (*Pyrenopeziza brassicae*).

The need for long-term data collection and crop growth modelling

These predictions about effects of climate change on range and severity of phoma stem canker were possible only because there are available both knowledge about the epidemiology of this monocyclic disease and extensive data sets on epidemic development and weather for a range of sites and growing seasons. These data sets 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.*, 2007b). A high priority over the next decade should be the collation of accurate disease and weather data and development of models to forecast effects of climate change on other plant diseases to provide the necessary foresight for strategic adaptation to climate change.

Conclusions

If climate change happens as predicted, there will be severe consequences for the UK agricultural industry (Stern, 2007). The modelling work reported in this paper describes the probable increase in severity and range of phoma stem canker, a disease which currently has the potential to cause annual UK losses of £40M (Fitt *et al.*, 2006a). The challenge for growers will be the early control of phoma leaf spotting, either by a well timed fungicide application guided by forecast predictions or the use of new cultivars which have been bred with enhanced levels of resistance against this pathogen.

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