

# Comparative biology of different plant pathogens to estimate effects of climate change on crop diseases in Europe

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## Abstract

This review describes environmental factors that influence severity of crop disease epidemics, especially in the UK and north-west Europe, in order to assess the effects of climate change on crop growth and yield and severity of disease epidemics. While work on some diseases, such as phoma stem canker of oilseed rape and fusarium ear blight of wheat, that combine crop growth, disease development and climate change models is described in detail, Climate-change projections and predictions of the resulting biotic responses to them are complex to predict and detailed models linking climate, crop growth and disease development are not available for many crop-pathogen systems. This review uses a novel approach of comparing pathogen biology according to 'ecotype' (a categorization based on aspects such as epidemic type, dissemination method and infection biology), guided by detailed disease progress models where available to identify potential future research priorities for disease control. Consequences of projected climate change was assessed for factors driving elements of disease cycles of fungal pathogens (nine important pathogens are assessed in detail), viruses, bacteria and phytoplasmas. Other diseases classified according to 'ecotypes' were reviewed and likely changes in their severity used to guide comparable diseases about which less information is available. Both direct and indirect effects of climate change are discussed, with an emphasis on examples from the UK, and considered in the context of other factors that influence diseases and particularly emergence of new diseases, such as changes to farm practices and introductions of exotic material and effects of other environment changes such as elevated CO<sub>2</sub>. Good crop disease control will

1 contribute to climate change mitigation by decreasing greenhouse gas emissions from agriculture while  
2 sustaining production. Strategies for adaptation to climate change are needed to maintain disease control and  
3 crop yields in north-west Europe.  
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5 **Keywords:** Climate change adaptation, CO<sub>2</sub> emissions, food insecurity, plant pathogens, epidemics,  
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7 invasive species  
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## 10 11 12 **Introduction** 13

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16 Climate change affects plants in natural and agricultural ecosystems throughout the world and has the  
17 potential to affect food security and stability of food supply, either by directly affecting crop productivity or  
18 by indirect effects such as exacerbating threats of pests and diseases (Beddington 2010; Garrett et al. 2006).  
19 Little work has been done on the effects of climate change on plant disease epidemics. It is now broadly  
20 accepted that climate change is occurring, and that many parts of the world will experience warmer  
21 conditions and more extreme weather events. There will also be substantial changes in precipitation with  
22 increased precipitation in the far northern, far southern and most equatorial latitudes, but drier in most other  
23 locations. Additionally, and the average annual precipitation projections do not show that in some locations,  
24 there may be seasonal changes in precipitation (Stern 2007; Semenov 2009). For example, north-western  
25 Europe is projected to experience wetter winters but drier summers with little change in annual average  
26 rainfall. Western, northern and central Europe will have increased winter rainfall, while this will be reduced  
27 in southern Europe but projections for summer are for substantially reduced rainfall in southern and central  
28 Europe and slightly reduced rainfall in northern Europe (Anon. 2007). Generally, Europe will become  
29 warmer (Fig. 1) but this too is a generalisation with seasonal and regional differences. Eastern Europe will  
30 experience the greatest warming effect in winter leading to milder winters, and Western and Southern  
31 Europe will experience the greatest summer warming, leading to hotter summers (Anon. 2007). Many of  
32 these projections average multiple simulation runs that individually indicate a wider range of weather  
33 extremes than at present due to altered circulation patterns (Anon. 2007). The UK government Office for  
34 Science (OSI Foresight) report considering future threats from animal and plant disease epidemics stressed  
35 the need for agriculture to develop optimal disease management strategies under predicted climate change  
36 scenarios (Anon. 2006). Arable cropping systems face new or increased threats from pests and diseases.  
37 Weather is the main environmental influence on plant diseases and affects disease distribution, although  
38 other factors such as changes to the host crop distribution, intensity of cropping and farming practices can  
39 also greatly affect disease severity. Little work has been done to study how impacts of climate change on  
40 crops and their diseases interact to affect productivity; this is difficult to predict because interactions are  
41 complex and non-linear. Furthermore, the elevated concentration of atmospheric CO<sub>2</sub> that is a cause of  
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climate change is also likely to directly affect crops and plant pathogens that cause crop diseases (Coakley et al. 1999; Gregory 2008; Fitt et al. 2011).

\*\*\*\*Fig 1 near here \*\*\*\*

Although climate change threatens food security in many regions of the world including southern Europe (Beddington 2010; Stern 2007). (Beddington 2010; Stern 2007), it presents an opportunity, if managed correctly, to increase crop productivity in northern Europe (Barnes et al. 2010), with new arable crops and new tender vegetable and fruit crops potentially able to be grown outdoors on a wide scale. Climate change is a gradual and long-term phenomenon but it is necessary to identify potential threats and conduct new research into them to optimise surveillance and disease control schemes, develop new crop protection methods and select varieties with disease resistance able to operate in warmer climates.

Climate change globally may exacerbate the threat to food security posed by crop diseases, (Stukenbrock and McDonald 2008), which currently are estimated to cause losses of 16% of crop production worldwide (Oerke 2006). Such losses are particularly serious for subsistence farmers growing crops in marginal environments, particularly in Africa and Asia, who cannot afford to use crop protection chemicals and are most threatened by climate change (Schmidhuber and Tubiello 2007). To benefit from increased theoretical yield potential of crops due to climate change in north-western Europe (Butterworth et al. 2010), to reduce the carbon footprint of food production (Berry et al. 2008; Mahmuti et al. 2009; Hughes et al. 2011, Carlton et al. 2011) and to maintain yields in areas where climate will reduce yield potential, it will be necessary to enhance crop protection to avoid losses due to pests and diseases.

There is increasing emphasis on breeding crop varieties with durable resistance to major pathogens but this can take 10-25 years (Angus and Fenwick 2008). Despite this, arable crops still have a relatively high level of flexibility to avoid or overcome any new disease problems as they arise, compared to systems such as orchards and forests (Shaw and Osborne 2011). Diseases, as one of the main production constraints for farmers, require consideration for control by a range of methods such as cultural practices, more resistant varieties and crop protection products. Farmers and agrochemical companies face a challenge of knowing what new diseases they will face in future, when EU legislation means that fewer approved chemical control options will exist and resistance to available fungicides may be a greater problem (Cools and Fraaije, 2008).

It is desirable to use fungicides only when they are needed as part of Integrated Pest Management (IPM). However, when used correctly, fungicides have a relatively low carbon footprint in their use but substantially increase yields (Berry et al. 2008; Mahmuti et al. 2009; Hughes et al. 2011, Carlton et al. 2011). The use of fungicides is likely to increase in order to maintain yields if recommendations are adopted to reduce the environmental impact of arable food production by reducing nitrogen applications, i.e. by enhancing disease control while fertilizer application is reduced (Gregory 2008; Paveley et al. 2008).

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Recommendations are needed to help target both disease resistance breeding programmes and development of fungicides against future disease threats and to optimise fungicide application timings under altered crop growth patterns. Decreased yields as a result of disease would otherwise mean that crops have to be grown on larger areas [releasing CO<sub>2</sub> that is sequestered in established grassland and increasing nitrogen use; Gregory 2008, Carlton et al. 2011], thereby impeding strategies to mitigate climate change. Efficient crop production releases surplus land for both wildlife and biofuel crops, with a consequential reduction in greenhouse gas emissions associated with food production compared to low-input systems. This review aims to provide a better understanding of direct and indirect effects of climate change on crop diseases in Europe to help direct future research.

### **Environmental factors influencing crop disease epidemics**

Plant disease occurs when three factors combine: a susceptible host, sufficient effective pathogen inoculum and suitable environmental conditions. Globally, farmers are able to reduce inoculum of plant pathogens by using a range of integrated crop protection practices, such as crop debris management (by removal, grazing, burning or burial by tillage) , paddy-field creation, crop rotation, intercropping and companion planting to reduce inoculum production or separate crops from sources of inoculum including insect vectors. Choice of varieties that are resistant to certain pathogens affects host susceptibility, while the main agronomic factor altered by the farmer's actions is application of crop protection products, such as fungicides, to protect the crop at particular growth stages. However, although some outdoor vegetable crops may be protected with plastic sheeting, for broad-acre arable crops, the farmer has no direct control over the weather, which is the main environmental factor influencing arable crop disease. Changes in the weather are likely, therefore, to result in changes in the occurrence and severity of crop diseases.

In particular, the weather can directly affect plant diseases by influencing spatial and temporal dispersal of propagules, synchrony of pathogen propagules with sensitive crop growth stages, frequency of suitable infection conditions (most fungal plant pathogens require wetness or high humidity for infection), host resistance (some resistance genes are temperature sensitive), speed of disease development (pathogen growth and for polycyclic pathogens – number of disease cycles) and pathogen survival (frost periods, length of intercrop period, etc), which affects whether the disease is epidemic following importation of propagules from elsewhere, endemic or absent). Climate change may also have indirect effects due to the inclusion in arable rotations of alternative crops that can act as hosts for certain pathogens, e.g. maize, a host to *Fusarium graminearum*, which also affects wheat, as maize is likely to increase in crop area in western Europe due to (i) use of cultivars that are adapted to cooler climates than those where maize was traditionally grown, (ii) climate change and (iii) demand for animal feed and biofuel (West et al. 2011).

In addition to altering climate, changes in atmospheric gas concentrations can encourage diseases since increasing ozone and CO<sub>2</sub> can reduce resistance expression (Gregory et al. 2009) and elevated CO<sub>2</sub> can increase pathogen fecundity, leading to enhanced rates of pathogen evolution (Chakraborty and Datta 2003;

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Coakley et al. 1999). In contrast, increased CO<sub>2</sub> was reported to increase pathogen latent periods (duration between infection and sporulation), which would reduce epidemic rates. Increased CO<sub>2</sub> was also reported to increase resistance of barley to *Blumeria graminis (hordei)* (Chakroborty et al. 1998; Coakley et al. 1999). Further research on the effects of increased CO<sub>2</sub> on plant disease epidemics using free-air CO<sub>2</sub> enrichment (FACE) systems is needed (Luck et al., 2011). In the 1970s, few would have predicted a considerable reduction in the incidence of *Septoria nodorum (Stagnospora nodorum)* on wheat and a similarly rapid increase in the incidence of *Septoria tritici (Mycosphaerella graminicola)* in Europe, yet this occurred, due not to climate change but to other environmental changes, principally a reduction in atmospheric SO<sub>2</sub> concentrations (Shaw et al. 2008). Environment- and particularly climate-change, has been predicted to lead to an altered geographic distribution of both crop hosts and their pathogens as well as changes in host pathogen interactions and yield-loss relationships (Coakley et al. 1999).

These environmental changes are likely to affect both polycyclic (pathogens with many cycles of infection per season) and monocyclic pathogens (pathogens with a single period of infection per cropping season; Fig. 2). Increased inoculum production per infection, increased pathogen aggressiveness (or altered host resistance) and, or increased infection success of polycyclic pathogens is likely to produce an epidemic described by curve (a) i.e. an increased epidemic rate. Enhanced survival of inoculum e.g. reduced degradation and grazing of crop debris in the intercrop period (summer), or increased winter survival of foliar pathogens, is likely to result in curve (b) compared to the baseline hypothetical polycyclic disease epidemic curve (c). In contrast, changes in crop and pathogen development may cause inoculum production and susceptible crop growth stages to coincide more (curve, d) or less (curve, e).

\*\*\*\*Fig 2 near here\*\*\*\*\*

Consideration of climate change effects on crop diseases and particularly newly emerging infectious diseases (EIDs), should be put into context alongside a brief review of other factors that influence the emergence of new diseases. According to Anderson et al. (2004), introduction is the most, or in one case, second most important driver for emergence of new diseases in different pathogen groups (fungi, bacteria, virus and phytoplasmas). Weather conditions were found to be a major influencing factor for bacterial and fungal plant EIDs, and although direct effects of climate were reported as relatively unimportant for plant EIDs that are caused by viruses, changes in vector populations (which are often due to climate) were reported as the most important influence after pathogen introduction. Interestingly, although agricultural changes (intensification, diversification, changed practices e.g. introduction of minimal-tillage) were identified as important driving factors of plant EIDs caused by fungi and viruses, they were not mentioned as drivers of bacterial diseases. Anderson et al. (2004) introduced the term ‘pathogen pollution’ to describe the anthropogenic movement of pathogens resulting in a pathogen crossing a boundary that previously provided geographical or ecological separation. As a result, there may be heightened impact of introduced pathogens on naïve susceptible host populations. Given the predicted continued increase in global air travel and trade volume, the number of introduced emerging diseases is also likely to increase. Because climate change will

1 enable plants and pathogens to survive outside their historic ranges, Harvell et al. (2002) predicted an  
2 increase in the number of invasive pathogens. For example, range expansion of grey leaf blight of maize,  
3 caused by the fungus *Cercospora zea-maydis*, was first noted during the 1970s, and subsequently became  
4 the major cause of maize yield loss in the USA. Brown and Hovmøller (2002) described instances where  
5 introduction of infected plant material (followed by local dispersal of spores) and long-distance airborne  
6 dispersal of spores had spread diseases to new continents. If key climatic conditions for survival and  
7 establishment of a disease are known, it is possible to use climate-matching tools such as NAPPFAST  
8 (Magarey et al. 2007), BIOCLIM (Busby 1991), HABITAT (Walker and Cocks 1991) or CLIMEX (Sutherst  
9 and Maywald 1985) to map locations where those conditions are met in order to identify locations where  
10 increased surveillance is advised and mitigating control measures researched. For example, Karnal bunt,  
11 caused by *Tilletia* (Neovossia) *indica*, which infects wheat, rye and triticale, is favoured by cool weather,  
12 rainfall and high humidity at the time of wheat ear emergence. The risk of establishment in Europe was  
13 estimated by Sansford et al. (2008) in part by applying a published karnal bunt disease model; they showed  
14 that conditions during the ear emergence or heading period (from just before anthesis, ~ May and June) were  
15 favourable for infection and disease development in many places in Europe.  
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## 25 **Effects of climate change on crop growth and yield in north-western Europe**

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28 Climate change is likely to have direct effects on crop growth. According to UKCIP climate projections, the  
29 date of onset of wheat anthesis in the UK would advance (by approximately 2 weeks by the 2050s; Fig. 3)  
30 and the date of maturity for harvest will advance by 3 weeks (Semenov 2009). Similarly research predicts  
31 flowering to advance by 9 days and harvest date to advance by 10 days in north France in the near future  
32 (2020-2060) (Bancal and Gate 2011). ‘Mediterranean-type’ wheat varieties, which respond to different  
33 environmental cues determining the time of flowering, typically flower 2 weeks earlier than current northern  
34 European varieties. Adoption of this kind of cultivar to northern Europe to avoid heat stress at flowering  
35 could advance the time of flowering by at least another week, e.g. to mid-May in southern England. Oilseed  
36 rape, which currently flowers in mid-April to May in central England and slightly later in Scotland, would  
37 flower up to three weeks earlier following mild winter weather. In considering effects of climate change on  
38 crop diseases, it is important to incorporate the effects of climate change on crop growth, to avoid making  
39 over-simplified, unreliable predictions (Butterworth et al. 2010, Madgwick et al. 2011).  
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54 In addition to altered temperature, an associated increase in atmospheric CO<sub>2</sub> concentrations is  
55 predicted to increase crop productivity (Gregory 2008; Goudriaan and Zadoks 1995). Changes in crop  
56 phenotype is predicted to include reduced density of stomata and increased crop canopy size and canopy  
57 density. Consequentially increased canopy humidity was suggested to promote a range of foliar pathogens  
58 (Manning and von Tiedemann 1995), although the reduced density of stomata, a result of elevated CO<sub>2</sub>  
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1 concentration (Bettarini et al. 1998), may offset this increase for pathogens that infect via leaf stomata rather  
2 than directly through the cuticle. Modelling predicts that enhanced atmospheric CO<sub>2</sub> will offset the earlier  
3 harvest date so that wheat yields will increase by 10-17.5% in England and Wales by the 2050s (based on  
4 cvs Avalon and Mercia) (Semenov, 2009). Similarly oilseed rape yield (in the absence of disease) is  
5 predicted to increase by 10% in England and up to 15% in Scotland (Butterworth et al. 2010).  
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## 8 **Direct effects of climate change on fungal and oomycete crop diseases**

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12 Coakley et al. (1999) and Harvell et al. (2002) predicted some general effects of climate change on plant  
13 pathogens. In temperate locations, milder winters and particularly higher night-time temperatures will  
14 enable increased winter survival of plant pathogens. Generally warmer temperatures in winter and  
15 throughout the growing season will accelerate insect vector and pathogen life cycles, increasing virus and  
16 phytoplasma transmission and sporulation and infection efficiency of fungal foliar pathogens. A review of  
17 climate change effects on plant diseases by Garrett et al. (2006) highlighted potential effects at different  
18 scales due to factors such as elevated temperatures (which can reduce host resistance), changes in  
19 precipitation (which often influences infection conditions) and increased storm events (which influences  
20 dispersal of many pathogens). The review considered effects of changes in crop phenotype and maturity in  
21 relation to increasing or decreasing disease severity (e.g. changes in occurrence of infection conditions  
22 through altered canopy density or changes to host susceptibility). In addition, changes in crop growth or  
23 yield potential will occur and are likely to affect strategies for disease control and other crop production  
24 methods. Various methods are possible to assess likely effects of climate change on crop diseases. These  
25 are: to use (i) detailed modelling of each individual crop-pathogen-projected climate system, (ii) inoculated  
26 outdoor and controlled environment experiments, (iii) comparison of disease occurrence in locations of the  
27 world with similar climates to that projected for other locations, (iv) expert knowledge, survey data and  
28 weather-related crop disease models reported in the literature, which could be interpreted and applied to  
29 comparable systems that lack published models but were assessed to exhibit similar biology or 'ecotype' and  
30 described in detail later. There were very few other locations found to match the climate of north-western  
31 Europe due largely to topographical and maritime effects (i.e. iii, above) and it would take an enormous  
32 project to conduct new experiments on every plant-pathogen system (i.e. ii, above) so the approach used here  
33 was to review detailed combined models (i, above) where available to aid interpretation of information  
34 assessed in method (iv).  
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50 Detailed modelling approaches that combine future climate simulations, crop growth models and  
51 disease models have been developed for phoma stem canker of oilseed rape (Evans et al. 2008; 2010;  
52 Butterworth et al. 2010) for fusarium head blight of wheat (Madgwick et al. 2011), and recently for both  
53 septoria leaf spot and brown rust of wheat (Gouache et al. 2011). For canker of oilseed rape, a weather-  
54 influenced oilseed rape growth model (STICS, Brisson et al., 2003) and weather-based disease forecasting  
55 models were combined with 30 runs (30 years of daily weather data based on projected climate) (Semenov,  
56 2009) per chosen date and climate-change scenario, to produce quantitative risk assessments (Butterworth et  
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1 al., 2010). The combination of climate scenarios and crop model predicted that climate change will increase  
2 yield of fungicide-treated oilseed rape crops in Scotland by up to 0.5 t/ha (15%) and by 0.15 t/ha (5%) in  
3 England (Butterworth et al. 2010). However, in fungicide-untreated crops of moderate disease susceptibility,  
4 the combination of climate scenarios, crop growth, disease development and yield loss models predicted that  
5 climate change will increase yield losses from phoma stem canker to up to 50% (1.5 t/ha) in southern  
6 England (Butterworth et al. 2010). The size of losses was predicted to be greater for winter oilseed rape  
7 cultivars that are susceptible than for those that are resistant to the phoma stem canker pathogen  
8 *Leptosphaeria maculans*.  
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11 For fusarium ear blight of wheat (head blight or scab), a similar method used the SIRIUS wheat  
12 growth model (Jamieson et al. 1998; Jamieson and Semenov, 2000) to predict dates of key growth stages  
13 (anthesis and harvest) for different arable-crop growing locations of the UK using projected climate data.  
14 This provided an estimation of revised anthesis date around which a weather-based epidemiological model  
15 was used to predict disease risk for each location using projected climate data per chosen date and climate-  
16 change scenario (Madgwick et al. 2011). The incidence of fusarium ear blight was related to rainfall during  
17 anthesis and temperature during the preceding 6 weeks. It was projected that, with climate change, wheat  
18 anthesis dates will be approximately two weeks earlier than at present so the rain-related risk of infection at  
19 anthesis did not decrease, as would have been predicted if anthesis had remained in mid-June (rainfall for the  
20 UK is projected to be almost unchanged in May but substantially reduced in June). Due to wetter and  
21 warmer conditions in spring, the model predicted a slight increase in severity of fusarium ear blight  
22 epidemics by the 2050s, particularly in southern England (Madgwick et al. 2011). This predicted slight  
23 increase reflects purely the weather-related risk. Increased maize cultivation, which is likely to substantially  
24 increase production of inoculum of *F. graminearum*, is an additional indirect climate-related factor that is  
25 likely to cause a much greater increase in severity of fusarium ear blight (West et al. 2011).  
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27 Research to predict effects of climate change on the wheat disease, septoria leaf spot in France,  
28 concluded that predictions were difficult due to contradictory effects of mild weather promoting inoculum  
29 build-up over winter but drier weather reducing infection of final leaves in late-spring (Gouache et al. 2011).  
30 The early stages of disease are likely to be enhanced because the intercrop survival of the pathogen is  
31 favored by dry summers, (Shaw *et al.* 2008) and because infection success (of spores) is promoted by milder  
32 winter weather (>7°C) (Pietravalle *et al.* 2003; te Beest *et al.* 2009). It seems clear that these factors are  
33 likely to lead to an increase in this disease on leaves at the base of the plant over winter and early spring.  
34 However, Gouache et al (2011) concluded that in France, despite some advancement of wheat phenology,  
35 declining spring rainfall events will reduce spread of this disease onto the final leaf layers. It should be noted  
36 that in the event of a wet spring, there may be capacity for this disease to be more severe than would be  
37 expected currently. For the UK, according to the HadRM3 scenario for the 2050s, (Semenov, 2009), rainfall  
38 is predicted to reduce below the current (baseline 1960-1990 monthly average) only from May onwards,  
39 which will be only just before emergence of the flag leaf for crops advanced to flower in late May (Semenov,  
40 2009) so rather than a reduction in disease predicted in France there is expected to be little change in this  
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disease on upper leaves in the UK. This illustrates how differences in predicted rainfall patterns between neighbouring countries of Europe can be significant.

In the case of brown rust, Gouache et al (2011) reported that in France, as for Septoria, a contradiction between slightly reduced wetness periods but slightly warmer conditions, which each diminish and promote infection respectively, necessitated a detailed modelling approach. They concluded that there would be little change in this disease with a possible slight reduction for late-sown crops at some sites and a slight increase in disease for early maturing varieties at some sites.

To assess risks that climate change will increase severity or range for current or new diseases of major arable crops in north-western Europe, likely responses to projected climate of nine key fungal diseases were evaluated by review of the literature, which included each pathogen's biology and epidemiology plus interpretation of published weather-based disease models against predicted climate and current disease distribution maps (Table 1). In some cases, as described above in Gouache et al (2011), the projected climate was considered to promote one aspect of a pathogen's life-cycle but reduce another aspect. As another example, stem canker of oilseed rape, caused by *Leptosphaeria maculans*, warmer, drier summers would delay the release of inoculum in the autumn (which would reduce final disease severity) but increased thermal time over winter and spring would increase pathogen development in the stem (which would increase final disease severity). The few published studies that have modelled in detail the combined effects of altered climate on both crop growth and disease development, provided improved resolution about which biological aspects were likely to override others. This was reinforced by examining past data from disease surveys and field experiments in different years or locations, and, or consultation with experts to aid the assessment. In the case of stem canker, the increased thermal time outweighed effects of delayed inoculum release and the disease severity was predicted to increase on untreated susceptible crops (Butterworth et al. 2010). In some cases, it was not possible to determine which of two contradictory factors would outweigh another and so a degree of uncertainty may be expressed or a qualification added as to what would occur under certain weather patterns.

Table 1 near here \*\*\*\*\*

Consideration of biological traits affecting the epidemiology of different diseases [e.g. epidemic type (mono- or polycyclic), dissemination method, infection condition requirements, latent period response to temperature) and the timing of key events such as sporulation or infection] was used to categorise the nine fungal diseases in Table 1 into seven 'ecotypes' (Table 2). Other existing diseases and potential new diseases (currently present on the crop in other climates) could then be categorised as similar to one of these eco-types. The main classifying factors used were: epidemic type, dissemination method combined with time of initial or primary infection (wind-dispersed after rain (ascospores), wind dispersed dry (e.g. spores of powdery mildews and rusts), splash dispersed (e.g. conidia of *Septoria tritici* (*Mycosphaerella graminicola*)), insect vectored, seed or soil-borne.

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Information about weather-crop growth interactions produced as part of the detailed study of canker of oilseed rape and fusarium of wheat (Butterworth et al. 2010; Madgwick et al. 2011) and more recent studies on septoria and brown rust (Bancal and Gate, 2011; Gouache et al. 2011) was used to define important climate change effects on crop growth (e.g. timings of key growth stages), [i.e. effects of both altered crop growth stages and projected weather were assessed for each disease]. Although these evaluations were substantiated against crop disease data from different locations and seasons, consideration was also made to the successive occurrence of several seasons of weather of the type predicted for the future. It is thought that several successive favourable seasons would allow build-up of inoculum to cause more disease than would occur in a single favourable season (Turner, 2008).

Application of this approach to other diseases of similar ecotypes in north-western Europe concluded that most rain-splashed, polycyclic foliar fungal diseases are predicted to increase in severity due to more epidemic cycles, greater plant biomass, denser canopies, and wetter conditions for most of the vegetative crop growth period. Some, however, may reduce slightly due to drier conditions at the end of the growing season (late spring and early summer) which will reduce severity on upper leaves of wheat for example but not on earlier maturing barley. *Cercospora* of beet (*Cercospora betticola*) is an exception, classed into a different ecotype (7), since it infects and damages leaves of beet much later in the year (June-September), a period that is predicted to be much drier throughout north-western Europe (Gladders et al. 2001; Pietravalle et al. 2003; Shaw et al. 2008; Lovell et al., 2004; te Beest, et al. 2009; Willis et al. 2006; Vereijssen et al. 2007). Disease will also be reduced if longer intercrop periods promote disease escape due to ascospore release before emergence of the following crop. In other cases, typically necrotrophs, which survive saprophytically, drier summer conditions may reduce the breakdown of crop debris (reduced activity of detritivorous invertebrates) and therefore increase inoculum availability, the release of which may also be synchronised with crop emergence to increase disease severity.

For dry/air-dispersed biotrophic foliar fungal pathogens (ecotype 2), since crop growth stages will advance to earlier in the year, it is likely that epidemics will continue but they may be more sporadic due to effects of droughts in the previous summer (inoculum may reduce if grasses and cereal volunteers suffer from drought conditions). Epidemics become severe when dry clear daytime weather in spring allows sporulation and dispersal and these conditions typically promote dew films at night, which allows infection. This weather combination is not likely to change in frequency very much during the key spring period, April-May in the UK, where rainfall is predicted to be similar to current (baseline) levels. By late spring in the UK, dew periods overnight are shorter but temperatures warmer and so different temperature preferences for infection by different rust species (and powdery mildews) mean that epidemics of at least one or other of them will be sustained well into the grain filling period. Generally for this ecotype, better winter survival will lead to earlier epidemics and possibly more late-spring sunshine hours and more plant biomass will also increase sporulation, particularly of *Puccinia striiformis* (yellow or stripe rust of wheat). It is therefore likely that there will generally be a moderate increase in severity of these diseases but with large differences from year to year (Roche et al. 2008; Milus et al. 2009; van den Berg and van den Bosch, 2007; te Beest et al. 2008; Shaw and Osborne, 2011; Smith et al. 1988). This includes an increased risk of black stem rust

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*(Puccinia graminis)*, which is currently a rare visitor to northern Europe and epidemics are caused by air-  
borne spores blown from south west Europe and north Africa, usually occurring too late to establish a  
damaging epidemic and inhibited by relatively cool weather. However, race Ug99, has a lower temperature  
optimum than other races and since it has recently spread from central to southern Africa, it is now exposed  
to air currents that are likely to spread it to new areas including the Middle East, south Asia and ultimately  
north America and Europe (Ronnie Coffman, Cornell University;  
[www.nature.com/news/2010/100526/full/news.2010.265.html](http://www.nature.com/news/2010/100526/full/news.2010.265.html)). For the present, epidemics are likely to  
remain rare and occur too late in the season to be a problem in northern Europe in most years but parts of  
southern Europe are under threat. Northern parts could be affected if the Ug99 race establishes in southern  
Europe.

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Little change is expected for upper leaf or ear/flower infecting fungi (ecotype 3), due to predicted  
drier conditions in late spring and summer but an advancement of crop growth stages. Two exceptions are *F.*  
*graminearum*, which may increase due to an indirect effect of increased maize cultivation, increasing the  
pathogen population and Ramularia leaf spot of barley (*Ramularia collo-cygni*), which may be exacerbated  
by heat stress (West et al. 2011; Parry et al. 1995; Shaw et al. 2008; Smith et al. 1988).

A common feature of monocyclic root and stem-infecting pathogens (ecotypes 4, 5 and 6) is that the  
effect of disease on yield is likely to be exacerbated by increased summer heat and drought stress on the host.  
Increased transpiration demand in hotter weather will mean that infected plants may suffer sufficient stress to  
induce senescence at lower disease severities than at present and hence, yield-loss relationships will change  
adversely per unit of disease. In addition, we predict an increase in disease development for autumn and  
winter-infecting root and stem pathogens (ecotype 4) due to increased thermal time (Evans et al., 2008;  
Butterworth et al. 2010). Kausarud et al. (2010) reported that between 1960 and 2007, there was a trend  
towards spring-fruited fungi releasing spores on average 18 days earlier over the study period. Most species  
studied were basidiomycetes but if similar responses occurred with ascomycetes, pathogens such as  
*Sclerotinia sclerotiorum*, which causes stem or white rot of oilseed rape and a wide range of vegetable crops,  
is likely to release spores in synchrony with earlier flowering of crops like oilseed rape. Hence no change is  
expected for spring-infecting root and stem pathogens (ecotype 5) as both pathogens and crop will advance  
in development. For soil-borne pathogens (ecotype 6), there is a great deal of uncertainty about the likely  
impact of climate change because little information is currently available and further research is suggested.

## 51 **Direct effects of climate change on viruses, bacteria and phytoplasmas**

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Generally longer periods of migration and feeding activity of vectors, caused by warmer conditions and  
longer growing seasons, will favour many insect-vectored virus diseases on a wide range of crops. An  
increased incidence of aphid-vectored viruses is predicted to occur, due to either increased winter survival of  
aphids or their earlier spring migration (Harrington and Stork, 1995). Already, mild winters have been

associated with increases in barley yellow dwarf virus (BYDV) in cereals and in virus diseases of sugar beet (Harrington and Stork, 1995).

New vectors or new crops may facilitate recombination of new virus diseases onto crops since many viruses are able to recombine to produce new types of virus. This process is likely to increase due to climate change, which will increase the range of different insect vectors, which may encounter viruses from different host plants for the first time. An example of this has occurred recently in Brazil due to the introduction of the B-biotype of whitefly (*Bemisia tabaci*), which facilitated the vectoring of viruses present in different native plants onto cultivated tomato crops in which they recombined to produce new virus diseases (Fernandes et al. 2008). New or increased use of existing crops such as maize and sunflower may increase the spread of viruses. Maize for example, is a host to a large number of viruses that can also cross-infect wheat, such as wheat spot mosaic and wheat streak mosaic and African cereal streak virus.

Warmer soils will affect soil-borne viruses because vectors will be able to infect crops at earlier growth stages and these diseases will have greater impact on development and yield. Symptoms and yield-loss may also be exacerbated by heat and drought.

Currently bacteria are of little importance in temperate arable crops but they can affect some vegetable or horticultural crops particularly in the south of Europe. *Xanthomonas spp.* (e.g. *X. campestris* on brassicas) affect oilseed rape in warm and wet European countries such as Portugal, causing non-vascular leaf spot or vascular black rot. This pathogen is seed and soil borne and rain-splashed with infection via hydathodes or wounds. It is probably under reported in many parts of Europe. *Pseudomonas syringae* pv. *maculicola* causes pod rot of oilseed rape but is rare and considering drier conditions are projected to occur from May, it is likely to remain rare.

Phytoplasmas, like virus diseases, are probably under reported. Many are vectored by insects and so there is potential for an increase in their importance due to climate change, similarly to our prediction for insect-vectored viruses. A 16SrI phytoplasma has been previously reported affecting winter oilseed rape in the Czech Republic (Bertaccini et al. 1998). An outbreak in Greece was reported and 16S rDNA sequence showed 100% identity with that of coneflower phyllody phytoplasma (EU333394) from the group 16SrI, ‘*Candidatus Phytoplasma asteris*’ (Maliogka et al. 2009).

\*\*\*\*\*Table 2 near here \*\*\*\*\*

## **Indirect effects of climate change on crop diseases**

Climate change can indirectly affect crop diseases through the adaptation strategies that it may induce, including altered crop rotations, different farming practices and different crop types cultivated (e.g. changes between winter and spring types) (Barnes et al. 2010). Recent work has demonstrated that changes in cropping practice from spring to autumn-sown crops, such as for linseed can have large effects on diseases;

1 e.g. the disease pasmo (*Mycosphaerella linicola*) became very severe on winter (autumn-sown) linseed  
2 when it was introduced into the UK although it had not been a problem on the spring linseed crops grown  
3 previously (Perryman et al. 2009). These differences between winter and spring crops may occur because  
4 spring crops escape exposure to most of the primary inoculum (often released in autumn) or have fewer  
5 disease cycles in their shorter growing season.  
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7 Climate change may indirectly affect the efficacy of control strategies due to factors such as a  
8 decrease in frequency of suitable spray conditions for autumn and winter spray applications and an increased  
9 likelihood of water-logging over winter, preventing the use of farm machinery. More rapid leaf production  
10 in autumn and spring would reduce the period of protection conferred by a fungicide spray as active  
11 chemicals on leaves are diluted by leaf expansion and as new, unsprayed leaves unfold. Additionally there  
12 are likely to be subtle changes in the rate of breakdown of applied agrochemicals under slightly warmer  
13 temperatures. The greatest changes are likely to be a need to respond to earlier disease epidemics,  
14 particularly those caused by polycyclic foliar pathogens, rather than relying on the currently accepted crop  
15 growth-stage regulated application dates. Due to changes in crop canopy densities and milder winters that  
16 will advance both crop growth and disease epidemics, late winter-early spring sprays could increase in  
17 importance. Leaf production of cereals in mid-late spring may also become so rapid that the timings of leaf  
18 three and flag leaf sprays will need revision in order to achieve cost-effective/optimal protection. Further  
19 research is needed to evaluate effects of climate and other environmental changes on biological control and  
20 useful effects of naturally-occurring microbes on phyllosphere and rizosphere pathogens.  
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22 Increased CO<sub>2</sub> concentrations will lead to denser crop canopies, which may slightly encourage a  
23 range of foliar diseases (rusts, powdery and downy mildews, and leaf blotch or spots) but in contrast, a lower  
24 density of stomata may slightly reduce infection efficiency by those pathogens that infect via stomata. A  
25 current knowledge gap exists as to the effect of increased CO<sub>2</sub> concentrations on various aspects of  
26 pathogens' lifecycles. Increased CO<sub>2</sub> may have various positive and negative direct effects on plant  
27 pathogens (systems studied so far have tended to show higher fecundity but longer latent periods). Further  
28 research using FACE systems is needed to investigate combined effects of climate change and enhanced CO<sub>2</sub>  
29 on plant diseases (Eastburn et al. 2011).  
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31 New crops (e.g. maize in north-western Europe) could increase common wheat pathogens such as  
32 *Fusarium graminearum*. Sunflower may be introduced to north-western Europe and this new crop may  
33 escape crop-specific diseases at first but will still be prone to generalists such as *Sclerotinia sclerotiorum*  
34 particularly where known field-crop hosts such as oilseed rape, peas, and carrots are currently grown. To  
35 avoid heat and drought stress, it may become more common in southern areas of Europe to switch from  
36 winter crops (i.e. sown in autumn and harvested the following summer) to grow frost tolerant spring varieties  
37 (i.e. not needing vernalisation) over the winter and harvested in late spring. This may also cause unexpected  
38 changes to disease epidemics. However, many current diseases may on average change in importance only  
39 very slightly in Europe as regions of production of particular crops move northwards.  
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## Concluding discussion

1 To guide government and industry strategies for adaptation to climate change in the light of the food  
2 security debate, it is essential to consider effects on crop diseases (Boonekamp 2011). There is an urgent  
3 need to predict which diseases are likely to increase in importance, but this requires the construction of  
4 coupled crop-disease-weather interaction models (Butterworth et al. 2010, Madgwick et al. 2011; Gouache et  
5 al. 2011). The categorisation of diseases into different ‘ecotypes’ provides a simple way to assess which of a  
6 multitude of diseases may increase in importance (Tables 1 & 2), based on direct effects of weather on the  
7 dispersal, epidemic type and changes to the occurrence of weather conditions affecting disease epidemiology  
8 at key times, along with indirect effects such as changes to crop rotation. However, this approach provides  
9 an indication but should not be considered as a substitute for a more detailed assessment of the predicted  
10 effects of climate change on important crop diseases. The general approach of categorising ecotypes,  
11 suggests that many known diseases will on average change in importance only very slightly if at all in north-  
12 western Europe as regions of production of particular crops will tend to move northwards. However, where  
13 crops remain in their original geographical range, particularly at the southern parts of their distribution,  
14 generally warmer conditions (increased thermal time) will exacerbate insect vectored diseases (many virus  
15 and phytoplasmas) and those root and stem diseases that first infect hosts during the autumn and winter, such  
16 as stem canker of oilseed rape (*Leptosphaeria maculans*), and eyespot (*Oculimacula acufiformis* and  
17 *Oculimacula yallundae*; *Helgardia acufiformis* and *Helgardia herpotrichoides*) of wheat. In contrast, spring-  
18 infecting root and stem pathogens are not likely to change significantly as their development is likely to  
19 advance to mirror advances in crop development (Table 2). However for these stem and root-infecting  
20 diseases, there may still be a detrimental effect of climate change if increased transpiration stress later in the  
21 season combined with root and stem disease will induce earlier senescence in the crop, which will exacerbate  
22 yield losses per unit of disease. Increased transpiration stress, heat or drought stress is also likely to increase  
23 yield losses per unit of disease for foliar diseases that promote water loss from leaves.

24 General reviews cannot easily take into consideration all the complexities of specific crop-pathogen-  
25 weather interactions, which may be contradictory for different aspects of the disease cycle, nor variations in  
26 predicted climate, which due to an element of chaos, may be altered as new information arises or may  
27 change significantly over short distances. Additionally, more extreme or variable weather may make some  
28 diseases (e.g. rusts and powdery mildew) more sporadic. The sporadic nature of epidemics of dry-dispersed  
29 obligate foliar diseases (rusts and powdery mildew or cereals) for example is likely to be due to greater  
30 winter survival in mild winters, which will enhance epidemics while dramatic reductions in pathogen  
31 populations will follow severe summer droughts, which will kill ‘green bridge’ volunteers and wild grasses.  
32 Epidemics of these obligate pathogens will therefore depend on combinations of favourable and  
33 unfavourable summer and winter weather over more than one season. In contrast, summer droughts will not  
34 affect most necrotrophs, which survive saprophytically, and reduced destruction of crop residues in dry  
35 summer weather, may result in increased inoculum production in the autumn. An improved understanding of  
36 both crop cultivation and pathogen survival and maturation is therefore important for development of  
37 disease-progress models to predict effects of climate change.

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Adaptability of pathogens to climate change can be considered using the approach reported by McDonald and Linde (2002) and also as a key determinant of an organism's likely success under climate change, as discussed by Davis et al (2005). Adaptability of pathogen species is difficult to predict but will be enhanced by sexual, polycyclic and air-dispersed life-cycle stages. However, introductions of new pathogens adapted to new conditions (Anderson et al. 2004, Flood 2010), changes in farm practices including new crops grown (Barnes et al. 2010) and complexities of climate change projections and the biotic responses to them (Semenov and Stratonovitch 2010) makes these predictions of the future impact of climate change on plant diseases relatively uncertain. Therefore it is essential for government and industry to invest in future food security by maintaining capability to monitor crops for diseases and identify new diseases (Shaw and Osbourne 2011, Barnes et al. 2010).

Evaluation of possible future plant disease threats is made more difficult by the high level of uncertainty about future technological developments and socio-economic factors that will influence future agricultural practices in general (Coakley et al. 1999). For example, changes to legislation (EC No 1107/2009 repeal of directive 91/414/EEC) will decrease the number of approved, effective fungicides for use on crops in the EU (Clarke et al. 2008). Although research is underway to improve disease control with reduced chemical inputs as part of IPM (e.g. EU Pure project FP7/2007-2013. FP7-265865), other approaches include sustainable intensive agriculture, in which inputs are used to maximise crop yields in order to produce food on less land area with implications for reducing the carbon cost of food production (Hughes et al. 2011). Another possible technological development is the use of genetically modified crops, which could include traits to resist certain diseases. Use of this technology is currently restricted in Europe, to use as a research tool in carefully controlled lab conditions to understand disease resistance and it is not clear whether this situation will change in the immediate future. However other sophisticated breeding methods, such as marker-assisted breeding, could be used to quicken resistance breeding targeted to those diseases identified as likely to increase.

In developing strategies for adaptation to climate change, it will be particularly important to breed new varieties that are resistant to pathogens when the crops are grown at higher temperatures since in certain cases, warmer temperatures reduce components of disease resistance (Zhu et al. 2010). In particular, plant breeders and pre-breeding researchers need to be able to access collections of host genotypes with as much diversity as possible in order to allow a response to new diseases that may emerge. For disease control based on effective host resistance, a greater emphasis on monitoring crops nationally for resistance breakdown and potentially a mechanism to coordinate deployment of resistance sources may be needed to combat the elevated speed of adaptation by pathogen populations, particularly for polycyclic pathogens. Shaw and Osborne (2011) put forward the plausible argument that to respond to unpredictable and potentially sudden changes in severity of diseases, maintenance of publicly funded pre-breeding and research programmes is essential. They add that these research programmes should aim to maintain a wide genetic diversity for each crop species, rather than to concentrate on preserving accessions with traits currently thought to be useful.

The general predictions made in this review about effects of climate change on epidemic severity and control methods for a wide-spectrum of arable crop diseases in north-western Europe suggest that most

diseases will not alter substantially due to climate change alone, provided that good crop protection practices are followed. The predictions, for the first time, suggest classes of disease that are likely to increase in severity as a practical guide to aid adaptation to climate change by the research community, growers, advisors, breeders, the agrochemical industry and policymakers. Climate change in north-west Europe offers the opportunity of increasing crop productivity (Butterworth et al. 2010) and diversifying cropping systems, and emphasises the need to produce arable crops with minimum emissions of greenhouse gases, while maintaining a secure and stable food supply (Berry et al. 2008, Mahmuti et al. 2009, Carlton et al. this issue).

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## Figure Legends

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3 Figure 1. Maps showing the background mean temperature ranges of Europe (colours) for the period 1961-  
4 1990 and location of cities in places that have their predicted temperature patterns for the end of the 21st  
5 century according to two climate models; ARPEGE (a), and the HadRM3H (b) in an 'A2' global warming  
6 scenario (based on continued relatively high CO<sub>2</sub> emissions). Reproduced with permission from Kopf et al.  
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13 Figure 2. Progress of disease epidemics with time after inoculation for hypothetical diseases; (a) polycyclic  
14 disease with rapid rate of spread, (b) polycyclic disease with same rate of spread as (c) but founding  
15 inoculum availability advanced by time period  $\Delta t$ , (c) polycyclic disease, (d) monocyclic disease epidemic  
16 (coinciding with susceptible crop growth stage or suitable infection conditions), (e) monocyclic epidemic  
17 with inoculum availability delayed leading to a decrease in disease incidence due to disease escape.  
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23 Figure 3. Maps of Great Britain showing; (a) prevalence of wheat cropping (<25% (■), 25-40% (■) and  
24 >40% (■) of the area) and terrain unsuitable for arable agriculture (■). The map also shows 14 met-station  
25 sites within the arable area used to give representative weather in different regions (details in Madgwick et  
26 al., 2011 supplementary information). Wheat and arable area information were from  
27 [www.hgca.com/cerealsmap/version9.swf](http://www.hgca.com/cerealsmap/version9.swf) ; (b) Average dates of anthesis (growth stage 65), for winter wheat  
28 cv. Consort projected by the wheat growth model Sirius, for baseline (1960-1990) and 2050s High CO<sub>2</sub>  
29 emission scenarios. The maps were produced by spatial interpolation between the 14 sites (●).  
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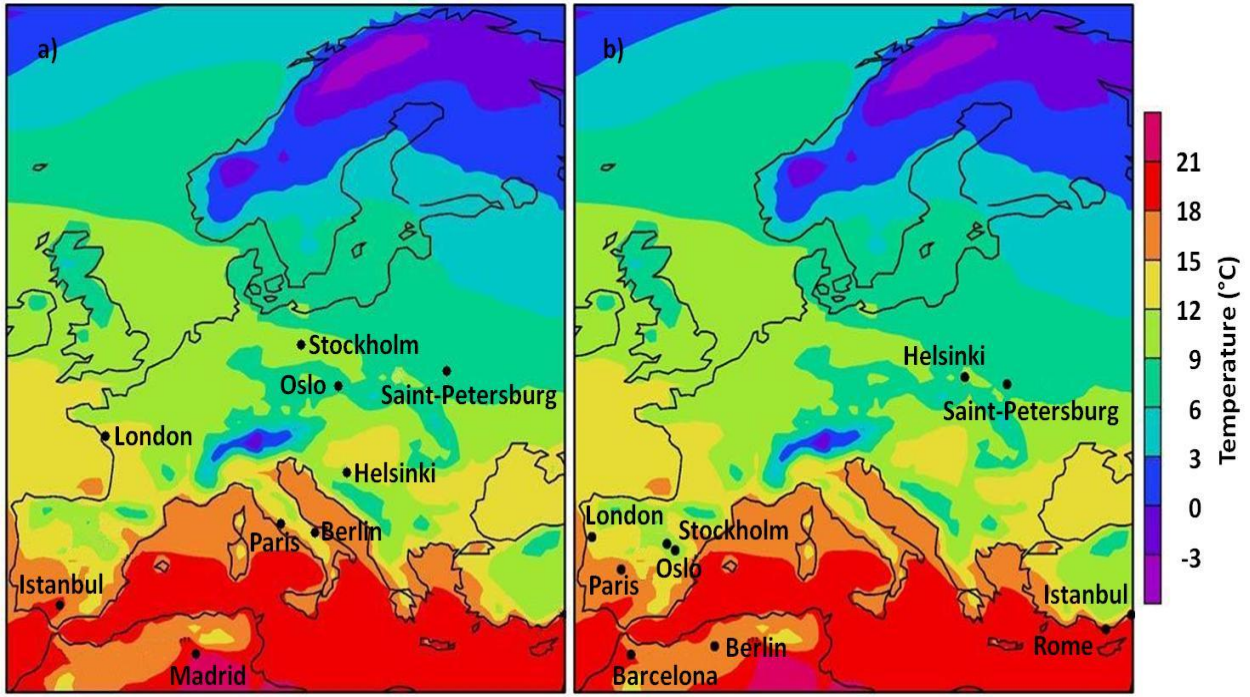


Figure 1. Maps showing the background mean temperature ranges of Europe (colours) for the period 1961-1990 and location of cities in places that have their predicted temperature patterns for the end of the 21st century according to two climate models; ARPEGE (a), and the HadRM3H (b) in an 'A2' global warming scenario (based on continued relatively high CO<sub>2</sub> emissions). Reproduced with permission from Kopf et al. (2008).

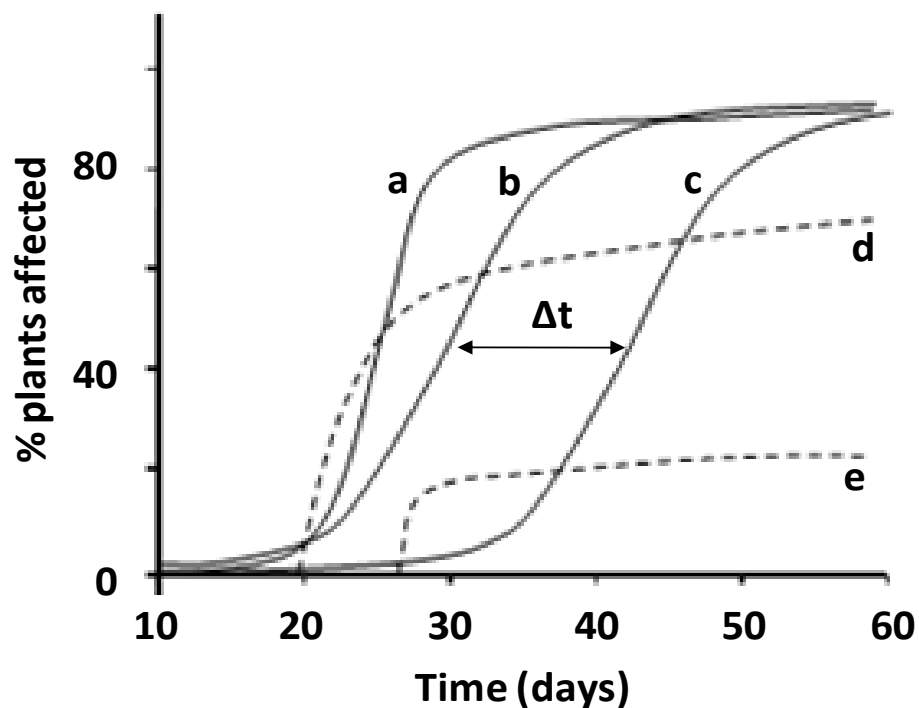
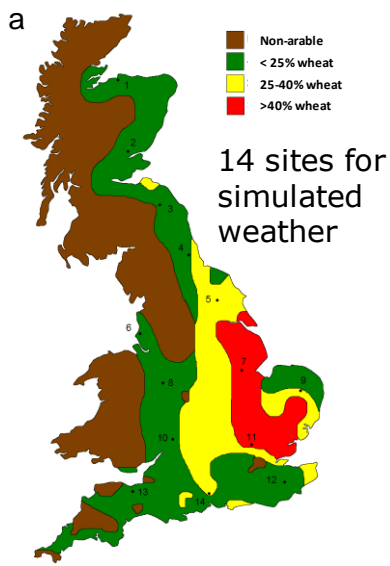


Figure 2. Progress of disease epidemics with time after inoculation for hypothetical diseases (a) polycyclic disease with rapid rate of spread, (b) polycyclic disease with same rate of spread as (c) but founding inoculum availability advanced by time period  $\Delta t$ , (c) polycyclic disease, (d) monocyclic disease epidemic (coinciding with susceptible crop growth stage or suitable infection conditions), (e) monocyclic epidemic with inoculum availability delayed leading to a decrease in disease incidence due to disease escape.



### % Wheat cropping



### Wheat anthesis date

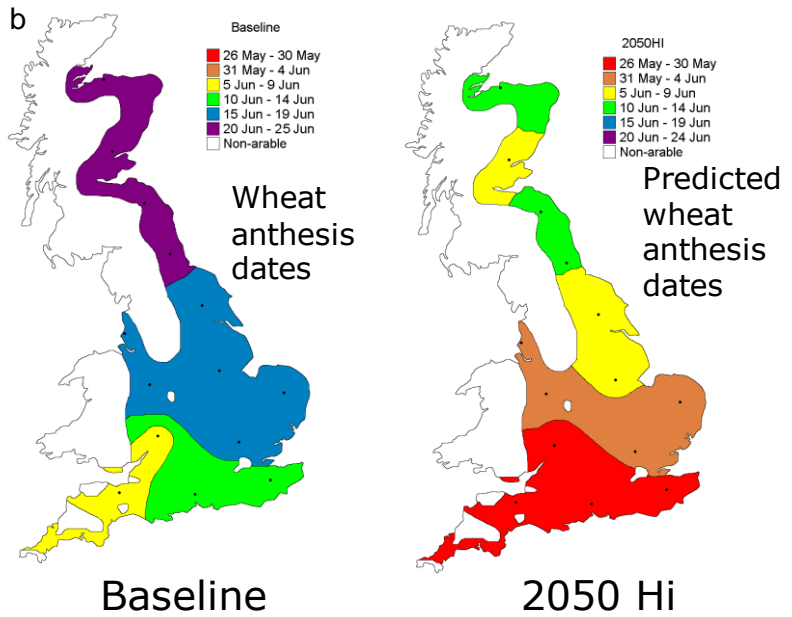


Figure 3. Maps of Great Britain showing;(a) prevalence of wheat cropping (<25% (■), 25-40% (■) and >40% (■) of the area) and terrain unsuitable for arable agriculture (■). The map also shows 14 met-station sites within the arable area used to give representative weather in different regions (details in Madgwick et al., 2011 supplementary information). Wheat and arable area information were from [www.hgca.com/cerealsmap/version9.swf](http://www.hgca.com/cerealsmap/version9.swf). ; (b) Average dates of anthesis (growth stage 65), for winter wheat cv. Consort projected by the wheat growth model Sirius, for baseline (1960-1990) and 2050s High CO<sub>2</sub> emission scenarios. The maps were produced by spatial interpolation between the 14 sites.

**Table 1 Evaluation of impacts of climate change on nine contrasting fungal diseases of north-western European broad-acre crops**

Pathogen (Ecotype No.)	Host(s)	Disease	Epidemic type	Key epidemiological features		
				Main dissemination	Infection and adaptability	Prediction
<i>Mycosphaerella graminicola</i> (1)	Wheat	Septoria leaf blotch	Polycyclic	Airborne ascospores then rain-splashed conidia (autumn-spring)	Dry summers increase inoculum survival. Mild, wet winters favour initial disease but drier late spring will reduce final severity. Highly adaptable	<i>Little change (UK)</i> <i>Slight decrease (France)</i>
<i>Rhynchosporium secalis</i> (1)	Barley	Leaf blotch or scald	Polycyclic	Either seed-borne or rain-splashed conidia (autumn/spring)	Favoured by wet spring weather. Preference for cool temperatures. Low adaptability	<i>Little change</i>
<i>Puccinia triticina</i> (2)	Wheat Barley?	Brown rust	Polycyclic	Airborne uredospores (mostly spring)	Mild winters and dry springs favour severe epidemics (e.g. 2007). High adaptability	<i>Sporadic</i>
<i>Blumeria graminis</i> (2)	Wheat Barley	Powdery mildew	Polycyclic	Airborne conidia (mostly spring)	Mild winters and warm, humid springs favour severe epidemics. High adaptability	<i>Sporadic</i>
<i>Fusarium graminearum</i> (3)	Wheat Maize	Ear blight	Effectively monocyclic	Splash dispersed conidia/air-borne ascospores (spring)	Warm spring and rain just before and during anthesis increases risk as does maize cultivation. High adaptability	<i>Slight increase</i>
<i>Leptosphaeria maculans</i> (4)	Oilseed rape	Phoma stem canker	Primarily Monocyclic	Airborne ascospores, rain-splashed conidia (autumn-winter)	Warm winters favour severe epidemics. Currently good cv resistance available. High adaptability	<i>Moderate increase</i>
<i>Sclerotinia sclerotiorum</i> (5)	Oilseed rape, legumes & vegetables	Stem rot	Monocyclic	Air-borne ascospores (spring)	Epidemic severe if ascospore release, petal fall and rainfall coincide. Closer rotations currently increasing risk. Moderate adaptability	<i>Little change</i>
<i>Verticillium longisporum</i> (6)	Oilseed rape	Verticillium	Monocyclic	Soil-borne mycelium Spores long-lived	Infection occurs in autumn; disease develops only when there is a hot, dry spring. Moderate adaptability	<i>Slight increase</i>

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<i>Cercospora beticola</i> (7)	Sugar beet	Cercospora	Polycyclic	Seed-borne (spring) Rain-splashed conidia (summer)	Favoured by warm, wet spring weather Moderate adaptability. Dry summers will reduce epidemics slightly. High adaptability	<i>Reduced but promoted by irrigation</i>
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**Table 2 Summary of predicted effects of climate change on arable crop diseases in north-western Europe classified into different ‘ecotypes’**

Disease type	Example(s)	Prediction
Winter-spring foliar-infecting polycyclic rain-splashed fungus (epidemics often initiated by air-borne ascospores at start of cropping season)	<i>Mycosphaerella graminicola</i> , <i>Rhynchosporium secalis</i> , <i>Selenophoma donacis</i> , <i>Pyrenophora tritici-repentis</i> ( <i>Drechslera tritici-repentis</i> ), <i>Pyrenophora teres</i> f. <i>teres</i> ( <i>Drechslera teres</i> ), <i>Alternaria brassicae</i> ,	little change (with a few exceptions – e.g. cool-preferring <i>Pyrenopeziza brassicae</i> will decrease)
As above but summer-infecting	<i>Cercospora betticola</i>	decrease due to drier summer conditions)
Dry air-dispersed polycyclic foliar fungus	<i>Puccinia triticina</i> , <i>Puccinia graminis</i> , <i>Puccinia striiformis</i> , <i>Blumeria graminis</i> , <i>Erysiphe cruciferarum</i> , <i>Bortytis</i> spp.	Sporadic – capacity for more severe and less severe seasons
Upper leaf and ear/flower infecting fungus	<i>Fusarium</i> & <i>Giberella</i> spp, <i>Claviceps purpurea</i> , <i>Phaeosphaeria nodorum</i> , <i>Tilletia tritici</i> , <i>Tilletia controversa</i> , <i>Ustilago nuda</i> , <i>Alternaria</i> spp., <i>Cladosporium</i> spp., <i>Pyrenophora graminea</i> [ <i>Drechslera graminea</i> ]	Little change except an increased risk for <i>F. graminearum</i> , <i>Urocystis agropyri</i> , <i>Tilletia</i> (Neovossia) <i>indica</i> and <i>Ramularia collo-cygni</i>
Monocyclic root and stem-infecting fungus (above-ground autumn-winter infection)	<i>Leptosphaeria maculans</i> , <i>Oculimacula yallundae</i> , <i>Cochliobolus sativus</i>	Increase in severity and yield loss per unit of disease
As above (above-ground spring infection)	<i>Sclerotinia sclerotiorum</i>	Little change in incidence or severity, slight increase in yield loss per unit of disease
As above (root infecting)	<i>Verticillium longisporum</i> , <i>Gaeumannomyces graminis</i> var. <i>tritici</i> , <i>Thanatephorus cucumeris</i> [ <i>Rhizoctonia solani</i> ], <i>Ceratobasidium cereal</i> [ <i>Rhizoctonia cerealis</i> ], <i>Omphalina pyxidata</i> , <i>Fusarium oxysporum</i> , <i>Plasmodiophora brassicae</i>	Varied/unknown response w.r.t. disease severity, probable increase in yield loss per unit of disease. <i>Monographella nivalis</i> [ <i>Microdochium nivale</i> ] and <i>Typhula incarnate</i> should reduce due to reduced winter snow cover.
Insect vectored virus	BYDV	increase
Soil-borne virus	Wheat soilborne mosaic Barley mosaic	Little change – depending on rainfall at location
Phytoplasma (insect vectored)	Aster yellows	increase