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Impact of climate change on diseases in sustainable arable crop systems: CLIMDIS

by

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1. ABSTRACT

Climate change is predicted to lead to milder, wetter winters and hotter, drier summers for the UK. Crops will advance growth with current wheat cultivars flowering 2-weeks earlier and harvested three weeks earlier than traditionally. 'Mediterranean-types' of cereals could advance these growth stages by another 2 weeks. Elevated CO_2 concentrations will increase crop productivity, as long as diseases and pests are controlled. Crops will probably move slightly to the north, with potential new crops such as maize and sunflower grown in the south of England and many diseases will change in importance only slightly. However, the risk of newly introduced diseases establishing is increased by climate change so it is important to maintain or increase statutory crop monitoring, quarantine and surveillance and to ensure that crop genotype collections keep as much diversity as possible for future breeding programmes.

More extreme weather may make certain diseases (e.g. rusts and powdery mildews) more sporadic and encourage those that develop quickly in warm conditions. Insect vectored virus and phytoplasma diseases will increase due to greater vector activity. Many contradictory effects of climate change mean that some detailed knowledge of each pathogen's life cycle and ideally climate-based disease-progress models are needed. We predict that rusts and powdery mildews will become more severe after mild weather in winter and early spring (assuming some dry days will allow dispersal) but less severe after particularly hot, dry weather in summer. Summer droughts may favour other pathogens that sporulate on debris due to reduced activity by molluscs and other invertebrates. A knowledge gap exists in understanding pathogen survival and the timing of spore release to infect subsequent crops as different responses to the climate by the pathogen and crop could lead to more or less infection. More research is also needed to understand impacts of climate change on soil microbes, particularly those that mitigate root diseases such as take-all. Generally warmer conditions will increase severity of autumn- and winter-infecting root and stem rots, while spring-infecting root and stem rots will advance with earlier crop growth and so not change in relative severity. However, yield losses from these diseases will also increase due to greater and earlier transpiration stress caused by heat or drought. Effects of increased CO_2 concentrations on plant pathogens also require further research. Increased CO_2 will lead to denser crop canopies, which will encourage a range of foliar diseases. Due to milder winters that will advance both crop growth and disease epidemics, T0 sprays could increase in importance. Leaf production in mid-late spring may also become so rapid that the timings of T1 and T2 sprays (relative to growth stage) will need revision in order to achieve optimal protection. Introductions of new pathogens ('unknown unknowns'), changes in farm practices including new crops grown, complexities of climate change projections and the biotic responses to this make prediction of the future impact of climate change on plant diseases relatively uncertain. It is therefore also important to create funding mechanisms that can allow a rapid response to research new diseases. Climate change offers the opportunity to increase crop productivity and diversify cropping systems, and

emphasises the need to produce arable crops with a low carbon-footprint, while maintaining a secure and stable food supply.

ABBREVIATIONS

DON: Deoxynivalenol (mycotoxin)
EID: emerging infectious disease
IPM: integrated pest management
NAPPFAST: NCSU (North Carolina State University) APHIS (Animal and Plant Health Inspection Service) Plant Pest Forecasting System
OSR: oilseed rape
STICS: Simulateur mulTIdiscplinaire pour les Cultures Standard
UKCIP: UK Climate Impacts Programme

2. SUMMARY

2.1. Project aims and background

Europe is likely to experience milder wetter winters, hotter drier summers and more extreme weather events. As a result, arable cropping systems face new or increased threats from pests and diseases. It is difficult to predict impacts of climate change on crops and their diseases as many interactions are complex and non-linear. Associated with climate change, increases in atmospheric CO_2 are also likely to affect both crops and diseases. It is not all bad news. Climate change can be an opportunity and, managed correctly, crop productivity can increase with new arable crops and various tender vegetable and fruit crops potentially able to be grown outdoors on a wide scale. This project aims to inform industry and government about likely disease threats to UK arable crops in the future as a result of climate change. Although climate change is a gradual and long-term phenomenon, it is necessary to identify potential threats and, if necessary, conduct new research into them in the immediate future to optimise surveillance and disease control schemes, develop new crop protection methods and select cultivars with disease resistance. Breeding of elite cultivars with resistance to a new disease, or development of new crop protection products often takes over ten years. There is still considerable flexibility in arable crop systems to avoid or overcome any new disease problems as they arise, compared to systems such as orchards and forests, and arable farmers usually have added flexibility of being able to apply a crop protection product, or even to cut their losses and grow a different crop in the following season. Both disease-resistant crops and moderate use of crop protection products, when needed as part of Integrated Pest Management (IPM), are desirable in order to increase yields and such disease control methods actually decrease the carbon footprint of producing each tonne of crop-yield. Efficient crop production releases surplus land for alternative crops such as biofuel crops with a consequential increase in biodiversity and a reduction in green-house gas emissions associated with food production compared to low-input systems. This review considers fungal, bacterial and viral diseases of current key arable crops in the UK (wheat, oilseed rape, barley and sugar beet) and predicts likely changes to specific diseases in these groups, including new potential diseases, to inform industry and government, particularly to highlight R&D priorities. Predictions are based on the current climate projections of the UK Met office & United Kingdom Climate Impacts Programme (UKCIP), which are varied and subject to modification.

2.2. Factors influencing crop disease epidemics

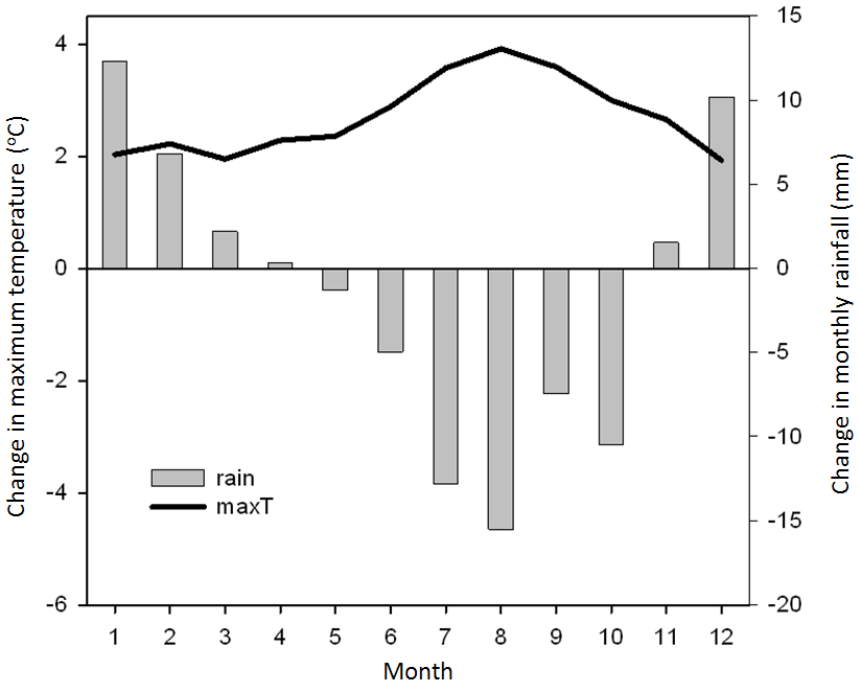
Plant disease occurs when three factors combine: a susceptible host, sufficient effective pathogen inoculum and suitable environmental conditions. Farmers are able to reduce plant disease by using a range of integrated crop protection practices that affect these three factors, such as tillage to bury crop residues (on which new spores are often produced) and crop rotation to separate new

crops from debris of previous crops, which both serve to reduce the amount of pathogen inoculum. Choice of cultivars that are resistant to certain pathogens affects host susceptibility, while the main environmental factor altered by the farmer's actions is application of crop protection products, such as fungicides to protect the crop at particular growth stages. Changes in disease pressure may occur due to altered sowing date but for broad-acre arable crops, the farmer has no control over the weather, which is the main environmental factor influencing arable crop diseases.

In particular, the weather can directly affect plant diseases by influencing the location and seasonal timing of release of spores that start disease epidemics. Changes to the weather affect the coincidence of inoculum (usually spores) and sensitive crop growth stages. Weather also affects the success of infection because suitable infection conditions are needed (most fungal plant pathogens require wetness or high humidity for infection). Temperature can increase or decrease the effectiveness of specific components of host resistance and the speed of disease development (pathogen growth and reproduction). Weather also affects pathogen survival (frost kills some pathogens while hot dry weather can reduce populations of those pathogens that only live on growing green leaves). 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 is a host to *Fusarium graminearum*, which also affects wheat. 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. Areas growing particular crops are likely to move slightly to the north as the climate warms while at the southern edge of their distribution, change to diseases and crop productivity may result in some crops becoming unprofitable and other crops may be introduced.

2.3. General Predictions

The climate of the UK is predicted to be milder over winter and particularly warmer in the summer but also much wetter over the winter and drier in the summer (Summary Figure 1) and there will also be more extreme weather events.



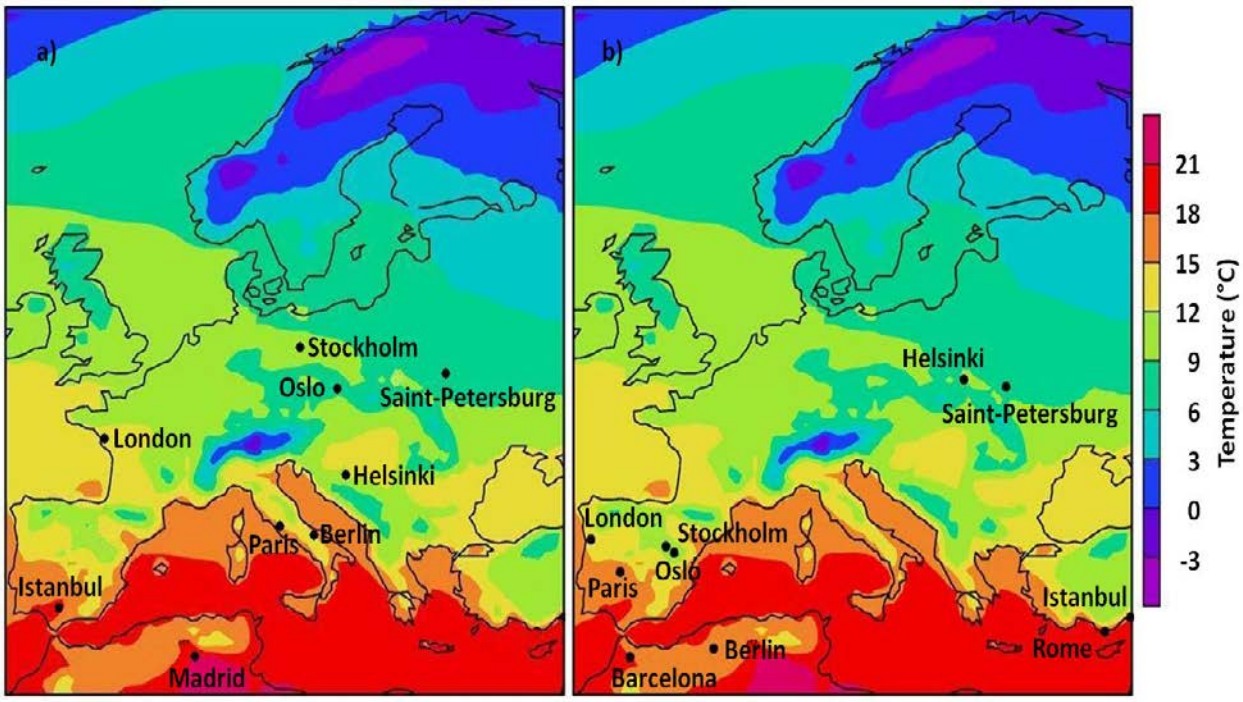
Summary Figure 1. Predicted change in temperature and rainfall forecast for Rothamsted, Hertfordshire, England, based on the HadRM3 scenario for the 2050s, modified from Semenov (2009) supplementary material.

Increased inoculum production per infection, increased pathogen aggressivity (altered host resistance) and/or increased infection success of pathogens (particularly those with many cycles of infection per season) could increase the rate of disease epidemics and final disease severity. Enhanced survival of pathogens may occur due to less crop debris being eaten by slugs, snails and other invertebrates over the summer, or by increased winter survival due to less-severe frost. This elevated survival will make disease epidemics appear earlier in the growing season. Additionally, changes to both crop and pathogen development in response to the climate may cause inoculum (disease propagule) production and susceptible crop growth stages to coincide more (which will increase disease incidence) or less (which will decrease disease – also known as 'disease escape'). Milder winters should therefore increase survival and epidemic rate of a range of foliar pathogens and cause spring epidemics to start earlier (rusts, mildews, leaf spots, etc). In contrast, very warm, dry and long summers should reduce over-summer survival of pathogens that need living green leaves (rusts). So rusts could become very sporadic because weather may be very favourable for a few years leading to a lot of disease, but the pathogen could decline to negligible levels for a few years following one or more years when weather patterns in the summer were unfavourable. Disease predictions considering climate change are usually not easy without mathematical models because often a climate-change scenario may promote one component of a disease epidemic but reduce another component of disease development. For example, with stem canker of oilseed rape, dry summers will delay ascospore release (suggesting a delayed or reduced epidemic) but milder winters will increase pathogen growth in the plant (suggesting

increased disease development). Detailed research is therefore needed to understand what the outcome of these contradictory factors is likely to be – in the case above, the increased pathogen growth in the winter and spring outweighs the effect of delayed disease onset in the autumn and so final disease severity is predicted to increase.

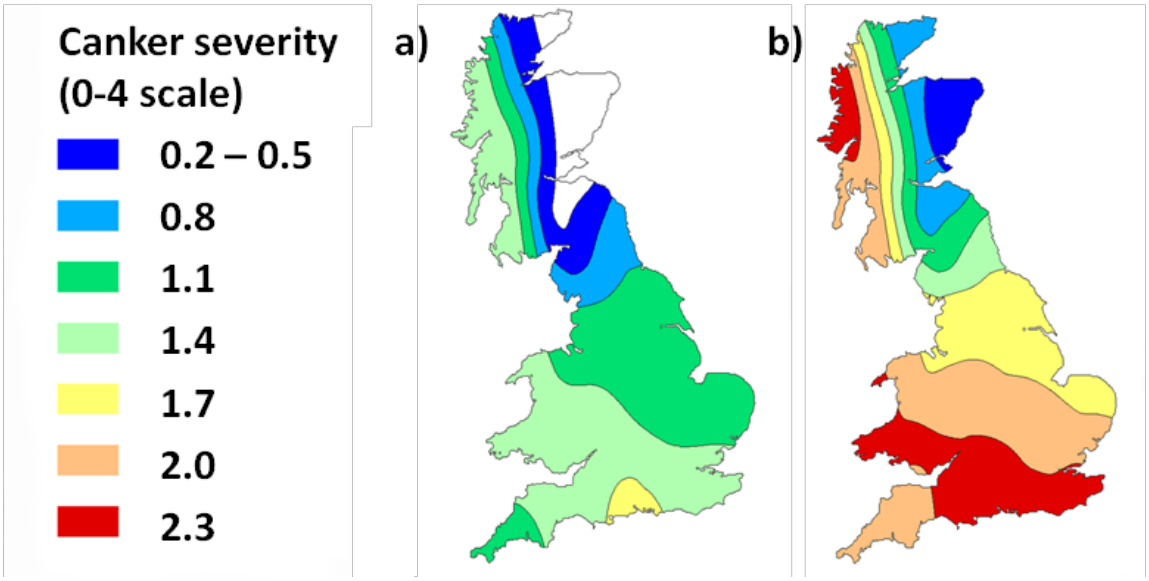
Introduction of a disease to a new location is still the main reason for new diseases occurring. Changes to climate should normally cause relatively small and gradual shifts in the range of existing diseases from the previous range. However, diseases that are newly introduced to an area usually spread faster than changes to the climate would suggest because the new pathogen is usually interacting with a host population that has developed little resistance to that pathogen. Establishment of the new disease can occur only if the crop system and climate is favourable for the pathogen to complete its life cycle (i.e. reproduce). Propagules of many new or rare pathogens frequently arrive in the UK, often in the wind or on imported plant products but they often do not establish because the climate is unfavorable. A key aspect of climate change is that it could enable a new range of introduced diseases to establish for the first time, rather than die out.

It is possible to use climate-matching software to map locations where key conditions needed by a pathogen are met in order to identify locations where increased surveillance is advised and mitigating control measures researched. An alternative approach is to identify locations that currently have a climate that is very similar to that projected for another location (say the UK) and to observe what crops and crop diseases occur there. However, climates currently similar to that predicted for the UK occur in very few locations because of the unique effect of our maritime situation and the gulf-stream (Summary Figure 2). Locations in south-west France (around Poitiers and Nantes) are most similar to the climate predicted for the UK in the 2050s. Septoria and brown rust are the two main wheat diseases there. In addition to wheat, maize and sunflower are also important crops of that region.



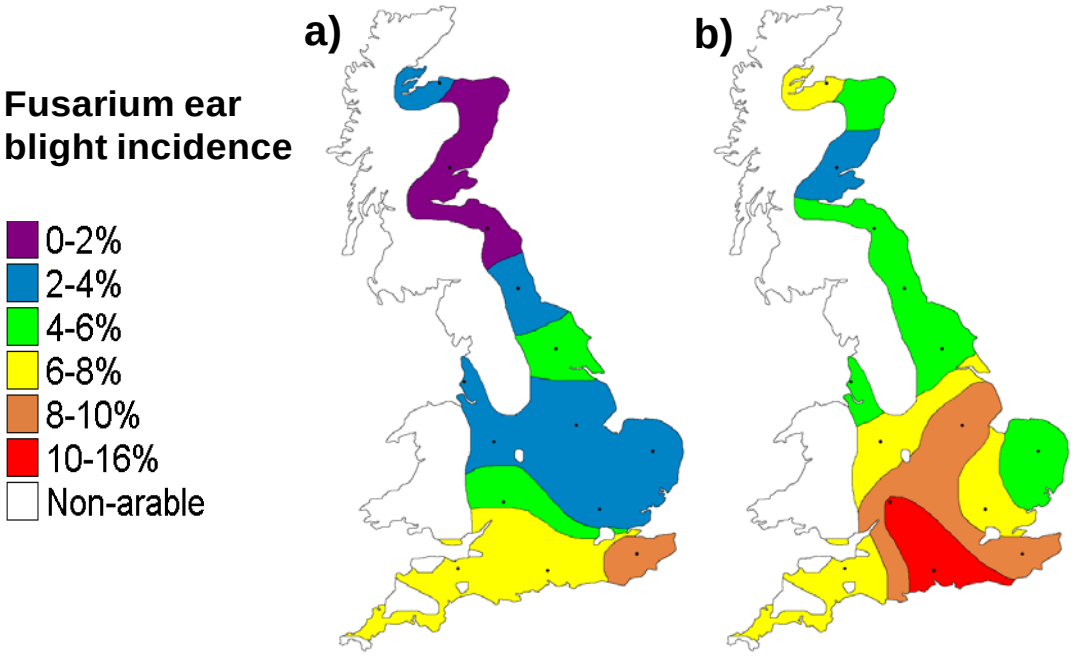
Summary Figure 2. Map showing the background mean temperature ranges of Europe (colours) for the period 1961-1990 and location of cities in places that have their predicted temperature patterns for the end of the 21st century according to two climate models; ARPEGE (a), and the HadRM3H (b) in an 'A2' global warming scenario (based on continued relatively high CO_2 emissions). Reproduced with permission from Kopf *et al.* (2008).

To help in this project's broad-ranging evaluation of possible diseases of arable crops, a detailed study of the likely response of both crop and crop-disease to the projected climate of the UK was made for phoma stem canker of oilseed rape and fusarium ear blight (specifically part of this disease complex caused by *Fusarium graminearum*) of wheat. Both studies tested predictions of a crop-growth model for oilseed rape and wheat, respectively. Climate change was found to advance flowering and harvest dates of both crops. Oilseed rape will flower approximately 3–4 weeks earlier than at present and a combination of both favorable and unfavourable effects of changed-climate on stem canker is predicted to lead to an overall increase in disease severity and a shift northwards in its range into eastern Scotland (Summary Figure 3; although not severely enough to affect yield in Scotland).



Summary Figure 3. Impacts of climate change on severity of phoma stem canker (*Leptosphaeria maculans*) at harvest of winter oilseed rape crops (mean of resistant and susceptible cultivars) for (a) baseline 1961–1990, (b) 2050s climates (mean of low and high emission scenarios); stem canker severity on a 0–4 scale (0, no disease; 4, plant dead); areas where crops are currently unaffected by the stem canker disease are marked white. Maps adapted from Butterworth *et al.* (2010).

Wheat flowering will be around 2 weeks earlier by the 2050s (4 weeks earlier if we switch to using 'Mediteranean-type' cultivars) and harvest will be 3 weeks earlier (or 5 weeks). Despite a shorter grain-filling period, yields in the absence of disease should be at least as good, if not greater than now because of the positive effect of greater CO_2 concentrations. Consideration of these altered growth stages is important because without it we would conclude that the incidence of fusarium ear blight will reduce substantially due to a decrease in occurrence of suitable wet conditions for infection occurring in early-mid June (the current flowering date over much of the UK arable area, slightly later in northern parts). However the predicted advanced flowering date means that suitable infection conditions will be only slightly less frequent than at present (see rainfall trend in Summary Figure 1). In the case of *F. graminearum*, warmer spring weather will increase spore production and additional spore release from maize debris is likely to lead to an overall increase in fusarium ear blight on wheat (Summary Figure 4). This is an example of an indirect effect of climate change on a crop disease.



Summary Figure 4. Maps showing the projected average fusarium ear blight incidence (% plants affected) generated by a fusarium ear blight model and based on advanced anthesis dates for two weather scenarios; baseline (a) and 2050s high emission scenario (b). The baseline scenario is based on weather from 1960–1990. The maps were produced by spatial interpolation between the 14 sites (adapted from Madgwick *et al*. 2011).

2.4. Effects of climate change on fungal crop diseases

Considering our findings from the two diseases studied in detail (phoma stem canker and fusarium ear blight), we furthermore conducted a broad-ranging review, categorising diseases into different ecotypes based on their survival, dispersal, epidemic type and plant tissue infected. As an exemplar for the first ecotype, we investigated the rain-splashed, polycyclic (many infection cycles per season) foliar fungal disease, septoria leaf blotch (*Mycosphaerella graminicola*) as this is currently the most important wheat disease in the UK. Septoria leaf blotch epidemics are started by air-dispersed ascospores (sexually-produced spores) in late summer and autumn but this is followed by many cycles of rain-splashed conidia (asexually-produced spores) and usually a second batch of ascospores in the spring. In summary of the literature, the disease is favored by dry summers, which increase inoculum survival, mild winters, which allow more infection and disease cycles and wet springs which promote rain-splashed dispersal and infection onto the most important leaf layers.

Moderately increased disease is likely for the following diseases that are similar ecotypes to septoria leaf blotch (*Mycosphaerella graminicola*). These are: leaf blotch or scald (*Rhynchosporium secalis*) and ramularia leaf spot (*Ramularia collo-cygni*) of barley; tan spot (*Pyrenophora tritici-repentis*) of wheat, and alternaria dark pod spot of oilseed rape (*Alternaria brassicae*). Most of

these 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 if longer intercrop periods promote disease escape due to ascospore release ahead of emergence of the following crop. In other cases, drier summer conditions may reduce the breakdown of crop debris (reduced activity of detritivorous invertebrates) and therefore increase inoculum availability, which may also be better synchronised with crop emergence. Two exceptions in this group are *Pyrenopeziza brassicae*, which causes light leaf spot of OSR and is favoured by cool temperatures so a slight decrease has been predicted. The second is cercospora leaf spot of beet (*Cercospora beticola*), which unlike the others diseases in this group, causes most leaf infection in the summer months, which are predicted to be drier while the disease is encouraged by hot and wet or humid weather. Current research gaps for this ecotype are primarily in understanding over-summer survival and timing of spore release to infect the following crop.

For the ecotype including dry/air-dispersed biotrophic (have to live on a living green plant) foliar fungal pathogens, such as brown rust (*Puccinia triticina*) of wheat and powdery mildew (*Blumeria graminis*), it is likely that epidemics will continue since crop growth stages will advance to earlier in the year. However, epidemics may be more sporadic particularly following droughts in the previous summer, because inoculum will decrease if grasses and cereal volunteers suffer drought conditions in the summer. Epidemics become severe when dry clear weather in spring allows sporulation and dispersal and these days are typically followed by dew films at night, which allow infection. This weather combination is not likely to change in frequency very much. 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 mildew) mean that epidemics of at least one or other will be sustained well into the grain filling period (brown rust is favoured by warmer conditions compared to yellow rust). Generally better winter survival will lead to earlier epidemics and possibly more spring sunshine hours and more plant biomass will also increase sporulation, particularly of yellow rust. It is therefore likely that there will be a moderate increase in these diseases on average but with large differences from year to year due to the effect of droughts on over-summer survival. Black stem rust (*Puccinia graminis* f. sp. *tritici*) may occur late in the season and has potential to develop to significant severities if the Ug99 race, which tolerates lower temperatures, becomes widespread in southern Europe. Currently this race has spread within Africa and the Middle East and has recently reached South Africa.

Rusts and powdery mildews of barley should follow a similar pattern to that predicted for the corresponding wheat diseases. The earlier maturation of barley will mean that growth stages up to flowering will, on average, experience milder, wetter weather, while flowering, grain-filling and harvest should be in progressively drier, warmer conditions than at present. There is scope for

summer droughts to reduce the impact of these pathogens in the following growing season. Similarly, crown rust (*Puccinia coronata*) of oats is likely to become more severe following mild winters and warm spring weather.

For the ecotype including various ear/flower infecting fungi, there is very little change expected except for fusarium ear blight, caused by *F. graminearum*, which can increase due to the effect of more maize (another host) cultivation. There is a need to maintain quarantine to prevent karnal bunt (*Tilletia indica*) from establishing here.

We divided the ecotype of monocyclic (single cycle of infection per growing season) root and stem fungal diseases into those with autumn spore release, those with spring spore release and those that are soil-borne. Conclusions for the autumn-releasing pathogens were based on conclusions for phoma stem canker (*Leptosphaeria maculans*). Severe epidemics of this group of diseases are favoured by mild, wet autumn, winter and spring weather. High evapo-transpiration stress in the summer before harvest may also exacerbate the yield-loss per unit of disease as the upper parts of the plant will lose water faster than it can be supplied through diseased root and stem tissue, leading to plant stress and earlier senescence. An analogous cereal disease is eyespot (*Oculimacula acuformis* and *Oculimacula yallundae; Helgardia acuformis* and *Helgardia herpotrichoides*).

Monocyclic diseases caused by pathogens that release spores in spring are likely to remain sporadic. Less change in effect of disease is likely for spring-infecting pathogens as their spore release is likely to advance along with crop growth stage and harvest will also advance. The potential for disease to develop severely depends on temperature preferences of individual species. Closer rotations and inclusion of other hosts (beans, peas and various vegetables) also increases risk for pathogens with many hosts such as *Sclerotinia sclerotiorum* (sclerotinia stem rot). For *S. sclerotiorum*, spore release should advance after milder winters to stay synchronised with flowering; hence there is little change in weather-related risk predicted.

For soil-borne pathogens, such as *Verticillium longisporum* of oilseed rape and take-all (*Gaeumannomyces graminis var. tritici*) of wheat, disease development and yield loss is also exacerbated by dry and hot conditions in late spring/early summer. Closer rotations will encourage this type of disease and warmer spring-summer weather should increase risk. Foot rot of cereals, caused by *Cochliobolus sativus,* is also likely to increase as this pathogen is favoured by warmer conditions than traditionally experienced in the UK. However, antagonism and competition by other soil-inhabiting microbes is important for control of these diseases but a knowledge gap exists in understanding the impact of climate change on soil microbes generally. Little climate-related

change is expected for root-invading foliar pathogens such as cephalosporium leaf stripe (*Hymenella cerealis*).

2.5. Effects of climate change on viruses, bacteria and phytoplasmas

Generally longer periods of migration and feeding activity of vectors and potential for new insect 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. Already, mild winters have been associated with an increase in BYDV in cereals and virus diseases of sugar beet. In the southern edge of geographic crop areas, warmer soils will affect soil-borne viruses as vectors will potentially be able to infect crops at earlier growth stages and will have greater impact on development and yield. Symptoms and yield-loss may also be exacerbated by heat and drought. Bacterial diseases are likely to remain rare in arable crops but phytoplasmas, which are usually insect vectored, will follow the same trend as that predicted for insect-vectored virus diseases – becoming more severe.

2.6. Conclusions and implications for disease control options

Many diseases will on average change in importance only slightly because regions of production of particular crops will tend to move northwards. However, more extreme or variable weather may make certain diseases (e.g. rusts and powdery mildew) more sporadic. The sporadic nature of epidemics of these obligate foliar pathogens is likely to be due to greater winter survival in mild winters, which will enhance epidemics, while dramatic reductions in pathogen populations will follow severe summer droughts, which will kill ‘green bridge’ volunteers and wild grasses. Epidemics of these obligate pathogens will therefore depend on combinations of favourable and unfavourable summer and winter weather over more than one season.

Summer droughts will not affect those pathogens that sporulate on dead plant tissue, i.e. that survive saprophytically (living on the nutrients in the dead host material), and these may even be enhanced by reduced destruction of crop residues (by molluscs and other invertebrates) in dry summer weather, leading to increased inoculum production in the autumn. Generally a knowledge gap exists in understanding pathogen survival and the timing of release of spores to infect subsequent crops as different responses to the climate by the pathogen and crop could lead to more or less infection (less or more disease-escape).

Where crops remain in their original crop areas or particularly at the southern parts of their distribution, generally warmer conditions will exacerbate diseases such as stem and root rots (stem canker of OSR, eyespot and take-all of wheat) due to increased thermal time. Insect vectored virus

and phytoplasma diseases will also become more important due to greater vector activity. Additionally, increased transpiration stress, heat or drought stress will speed-up symptom development and increase yield losses per unit of disease for many stem and root rots and some foliar diseases.

Increased CO_2 concentrations will lead to denser crop canopies, which will encourage a range of foliar diseases (rusts, powdery and downy mildews, and leaf blotch or spots). Increased CO_2 will also have various positive and negative direct effects on plant pathogens (systems studied so far have tended to show higher fecundity but longer latent periods) and further research could investigate combined effects of climate change and enhanced CO_2 on plant diseases.

Due to changes in crop canopy densities and milder winters that will advance both crop growth and disease epidemics, T0 sprays could increase in importance. Leaf production in mid-late spring may also become so rapid that the timings of T1 and T2 sprays (relative to growth stage) will need revision in order to achieve optimal protection. New crops such as maize could increase the incidence of wheat pathogens such as *Fusarium graminearum*. Sunflower may be introduced to southernmost England and this new crop may escape crop-specific diseases at first but will still be prone to generalists such as *Botrytis cinerea* and *Sclerotinia sclerotiorum*, the latter of which has capacity to return large numbers of sclerotia to the soil from each infected flower-head or stem. A knowledge gap exists in understanding the impact of climate change on soil microbes and particularly the effects of increased winter rainfall and decreased summer rainfall on them. Introductions of new pathogens, changes in farm practices including new crops grown, complexities of climate change projections (jet-stream changes may make it colder!), and the biotic responses to this, makes prediction of the future impact of climate change on plant diseases relatively uncertain. It is therefore also important to create funding mechanisms that can allow a rapid response to research new diseases and to maintain crop monitoring, quarantine and surveillance activities.

3. TECHNICAL DETAIL

3.1. Background

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. It is now broadly accepted that climate change is occurring, and that Europe is likely to experience warmer, wetter winters, hotter drier summers and more extreme weather events (Stern, 2007; Anon, 2005a; Semenov 2009; Fig 1). The OSI Foresight report considering future threats from animal and plant disease epidemics stressed the need for agriculture to develop optimal disease management strategies under predicted climate change scenarios (Anon, 2006). Arable cropping systems face new or increased threats from pests and diseases. Climate is the main environmental influence on plant diseases and affects disease distributions, although other factors such as changes to the host crop distribution, intensity of cropping, and various farming practices can also greatly affect disease severity. Little work has been done to study how impacts of climate change on crops and their diseases interact to affect productivity and this is difficult to predict because interactions are complex and non-linear. Furthermore, an elevated concentration of atmospheric CO_2, in addition to causing climate change, is also likely to directly affect crops and crop diseases (Coakley *et al.* 1999; Gregory, 2008).

Climate change can be an opportunity and, managed correctly, crop productivity can increase with new arable crops and various tender vegetable and fruit crops potentially able to be grown outdoors on a wide scale. The potential for joint Industry/Defra research to help farmers respond to threats and opportunities from climate change was highlighted at the 2007 NFU conference (Miliband, 2007). These threats and opportunities were further discussed at a recent HGCA R&D conference (Anon, 2008a). This project aims to inform industry and government about likely disease threats to UK arable crops in the future as a result of climate change. Although climate change is a gradual and long-term phenomenon, it is necessary to identify potential threats and conduct new research into them in the immediate future to optimise surveillance and disease control schemes, develop new crop protection methods and select cultivars with disease resistance.

There is increasing emphasis on breeding crop cultivars with durable resistance to major pathogens but this can take 10–25 years (Angus & 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. Diseases, as one of the main production constraints for farmers, regularly require control by fungicides. Agrochemical companies are concerned about what new diseases they will face in future, when fewer approved chemical control options will exist and resistance to available fungicides may be a greater problem.

It is desirable to use fungicides only when needed as part of Integrated Pest Management (IPM). However, fungicides have a relatively low carbon footprint (Berry *et al*. 2008; Mahmuti *et al*. 2009), and their use 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). 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. Decreased yields as a result of disease would otherwise mean that crops have to be grown on larger areas [releasing CO_2 that is sequestered in established grassland and increasing nitrogen use; Gregory, 2008], thereby impeding strategies to mitigate climate change. Efficient crop production releases surplus land for both wildlife and biofuel crops with a consequential reduction in green-house gas emissions associated with food production compared to low-input systems.

This review considers fungal, bacterial and virus diseases of current key arable crops in the UK (wheat, oilseed rape, barley and sugar beet). It discusses integration of work on simulating future climate scenarios, based on UKCIP projections, application of UK and overseas weather-based crop growth (Jamieson *et al.,* 1998) and disease progress models (Evans *et al*., 2008; Fitt *et al*., 2006a; Salam *et al*., 2007) and use of long-term datasets of weather and key diseases for a wide range of sites in the UK (e.g. CropMonitor data). It aims to provide a better understanding of future risks from crop disease (e.g. assessments of which diseases might increase or decrease) to guide future R&D such as cultivar resistance breeding or development of fungicides for new target pathogens. The report also provides guidance on disease management options in response to changed crop growth and disease epidemics and a potential decrease in the number of approved, effective fungicides in the UK (EC No 1107/2009 repeal of directive 91/414/EEC; Clarke *et al*., 2008). The project also aims to provide guidance to development of government policy for adaptation to climate change. Predictions are based on the current climate projections of the UK met office (UKCIP), which are varied and subject to modification.

3.2. Factors influencing crop disease epidemics

Plant disease occurs when three factors combine: a susceptible host, sufficient effective pathogen inoculum and suitable environmental conditions. Farmers are able to reduce plant disease by using a range of integrated crop protection practices that affect these three factors, such as tillage to bury crop residues (on which new spores are often produced) and crop rotation to separate new crops from debris of previous crops, which both serve to reduce the amount of pathogen inoculum. Choice of cultivars that are resistant to certain pathogens affects host susceptibility, while the main environmental factor altered by the farmer's actions is application of crop protection products, such

as fungicides to protect the crop at particular growth stages. Changes in disease pressure may also occur due to altered sowing date and although some outdoor vegetable crops may be protected with plastic sheeting, for broad-acre arable crops, the farmer has no 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 is a host to *Fusarium graminearum*, which also affects wheat (discussed in section 3.2). Maize production is likely to increase in the UK 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.

In addition to altered climate, changes in atmospheric gases could encourage diseases since ozone and CO_2 can reduce resistance expression (Gregory *et al*., 2009) and elevated CO_2 can increase pathogen fecundity, leading to enhanced rates of pathogen evolution (Chakraborty & Datta, 2003; Coakley *et al*., 1999). In contrast, increased CO_2 was reported to increase the latent period (duration between infection and sporulation), which would reduce epidemic rates and increased CO_2 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_2 on plant disease epidemics using free-air CO_2 enrichment (FACE) systems is needed (S Chakraborty, pers. comm. CSIRO Plant Industry, Australia). If this report had been commissioned in the 1970s, few would have predicted a dramatic reduction in *Septoria nodorum* (*Parastagonospora nodorum*) on wheat and a similarly dramatic increase in *Zymoseptoria tritici* (*Mycosphaerella graminicola*), yet this occurred, due not to climate change but to other environmental changes, principally a reduction in atmospheric SO_2 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

season) as hypothesised in Figure 1. Increased inoculum production per infection, increased pathogen aggressivity (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 over 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).

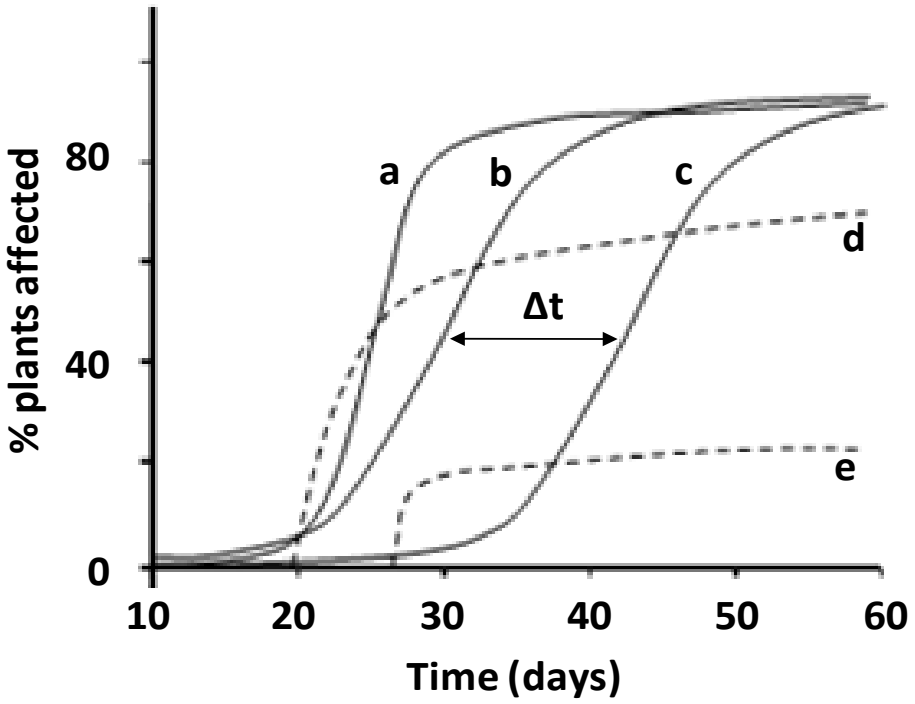


Figure 1. Progress of disease epidemics with time after inoculation for hypothetical diseases (a) polycyclic disease with rapid rate of spread, (b) polycyclic disease with same rate of spread as (c) but founding inoculum availability advanced by time period ∆t, (c) polycyclic disease, (d) monocyclic 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.

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 one of the important drivers 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, but are relatively unimportant for plant EIDs that are caused by viruses, where changes in vector populations are the most important influence after pathogen introduction. Interestingly, although agricultural changes (intensification, diversification, changed practices e.g. min-till) 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 an evolutionary boundary, such as 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 enable plants and pathogens to survive outside their historic ranges, Harvell *et al*. (2002) predicted an increase in the number of invasive pathogens. For example, range expansion of grey leaf blight of maize, caused by the fungus *Cercospora zeae-maydis*, was first noticed during the 1970s, and in the past two decades, has become the major cause of maize yield loss in the USA. Brown & Hovmøller (2002) described instances where introduction of infected plant material (followed by local dispersal of spores) and long-distance airborne dispersal of spores had spread diseases to new continents. If key climatic conditions for survival and establishment of a disease are known, it is possible to use climate-matching tools such as NAPPFAST (Magarey *et al.*, 2007), BIOCLIM (Busby, 1991), HABITAT (Walker & Cocks, 1991) or CLIMEX (Sutherst & Maywald, 1985) to map locations where those conditions are met in order to identify locations where increased surveillance is advised and mitigating control measures researched.

Evaluation of possible future plant disease threats is made more difficult by the high level of uncertainty about future technological developments and socioeconomic factors that will influence future agricultural practices in general (Coakley et al., 1999). Climate change can indirectly affect crop diseases by altered crop rotations, different farming practices, different crop types cultivated (winter v spring). Recent work has demonstrated that changes in cropping practice from spring to autumn-sown crops, such as for linseed or barley can have large effects on diseases; e.g. pasmo (*Mycosphaerella linicola*) became very severe on winter linseed but not on spring linseed (Perryman *et al*. 2009) and rhynchosporium leaf blotch has been shown to be very severe on winter barley compared to spring barley (pers. comm. S.D. Atkins, TWC Services, Oxford). These differences between winter and spring crops may occur because spring crops escape exposure to most primary inoculum or have fewer disease cycles in their shorter growing season.

3.3. UK climate change projections

The weather in the UK is relatively variable with large differences in monthly rainfall typically occurring during a year, despite the long term average monthly rainfall at any given location being similar each month. North and East Scotland are the coolest places in the UK with mean annual temperatures around 7°C (in the 1980s), while south-central England, south-east England and East Anglia are the warmest with mean annual temperatures around 10°C (in the 1980s). UKCIP projections of future weather vary depending on which of many climate change scenarios are used but the general consensus is that the UK is predicted to have a warmer climate (e.g. +2°C in the

winter to +4°C in the summer), with slightly wetter winters and drier summers (Fig. 2c). In the period 1961 to 1995, there was already a substantial increase in winter rainfall (total and number of rain-days) and a substantial decrease in spring and summer rainfall over large areas of the UK (Maraun *et al*., 2008). The UK is also projected to experience much more intense weather events, such as storms and more extreme weather events will occur globally (Coakley *et al*., 1999). A way to visualise projections of future climate change is by matching locations of European cities to locations that currently experience their respective predicted climates (Fig. 2 a,b; Kopf *et al.*, 2008), e.g. by 2080 London will experience the weather currently experienced in western France or even north Portugal, according to two different climate models. Although there is incontrovertible evidence that the average global temperature is increasing, there are many differing projections of future climate for the UK. For example, some alternative scenarios based on changes to the jet-stream have been suggested, which would result in colder winters and cool, wet summers for the UK (Greene & Monger, 2012). This review therefore attempts to describe responses of pathogens to general weather scenarios and recommendations of likely changes in epidemics of specific diseases are based on the UK Met Office (Hadley Centre, UKCIP) climate projections. Recently revised climate change predictions are now available from http://www.metoffice.gov.uk/research/hadleycentre/.

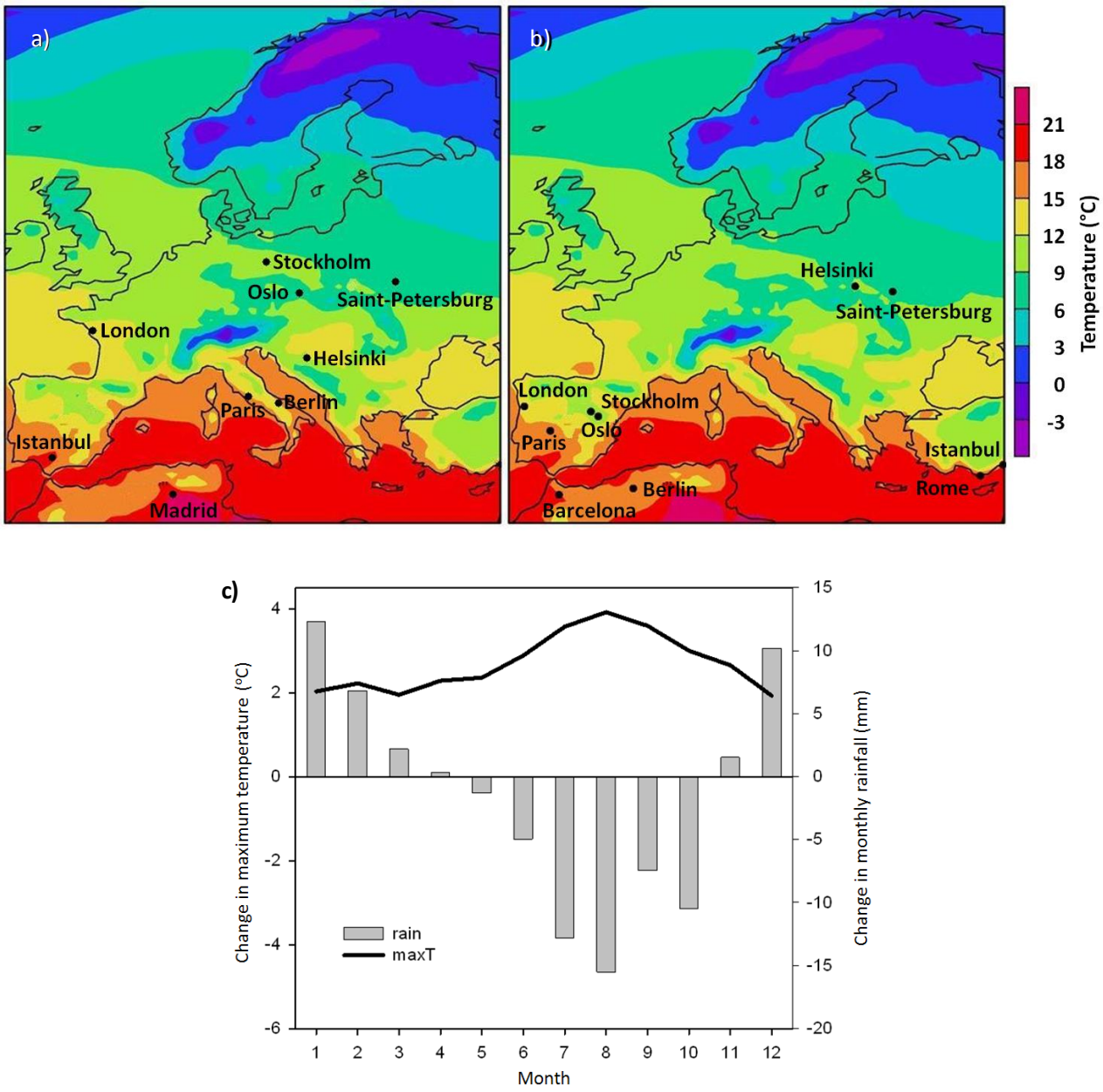


Figure 2. Representations of climates European cities are projected to experience by the end of the 21st century according to two possible climate change models: ARPEGE (a), and the HadRM3H (b) model runs in an A2 global warming scenario, which is a scenario based on increasing population, increasing CO_2 emissions and consequently projects higher temperature increases than most other scenarios considered. Both predictions are displayed on a background of mean temperature from the HadRM3H run for the period 1961–1990 for easier visual comparison and a basic impression of relative temperatures for the two periods (i.e. European cities are located in those places that currently have their predicted temperatures). Reproduced with permission from Kopf *et al.* (2008). c) Predicted change in temperature and rainfall forecast for Rothamsted, Hertfordshire, UK, based on the HadRM3 scenario for the 2050s, modified from Semenov, (2009) supplementary material.

3.4. Effects of climate change on crop growth and yield

The UKCIP projected weather would advance the date of onset of wheat anthesis (by approximately 2 weeks by the 2050s; Fig. 3) and maturity for harvest (by 3 weeks) (Semenov, 2009). 'Mediterranean-type' wheat cultivars, which respond to different environmental cues determining the time of flowering, typically flower 2 weeks earlier than current UK cultivars. Adoption of this kind of cultivar in the UK to avoid heat stress at flowering could advance the time of flowering by at least another week to mid-May in southern England. Oilseed rape, which currently flowers in mid-April to May in central England and slightly later in Scotland, would flower up to three weeks earlier following mild winter weather.

In addition to altered temperature, an associated increase in atmospheric CO_2 concentrations is predicted to increase crop productivity (Gregory, 2008; Goudriaan & Zadoks, 1995). Increased crop canopy size and density and consequentially increased canopy humidity was suggested to promote a range of foliar pathogens (Manning & von Tiedemann, 1995), although reduced density of stomata, predicted as a result of elevated CO_2 concentration (Bettarini *et al*., 1998) may offset this increase for pathogens that infect via stomata rather than directly. Modelling predicts that enhanced atmospheric CO_2 will offset the earlier harvest date so that wheat yields will increase by 10-17.5% in England and Wales by the 2050s (based on cvs Avalon and Mercia) (Semenov, 2009). Similarly oilseed rape yield (in the absence of disease) is predicted to increase by 10% in England and up to 15% in Scotland (Butterworth *et al*., 2010).

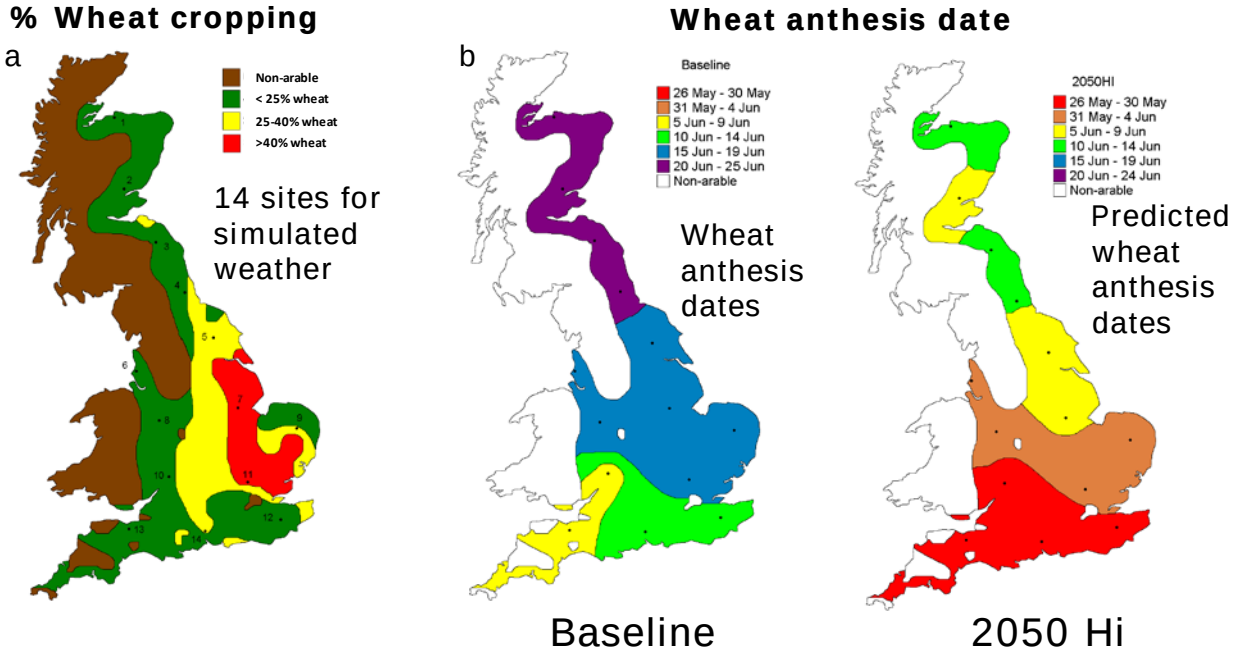


Figure 3. Maps of Great Britain showing; (a) prevalence of wheat cropping (<25% (■), 25-40% (■) and >40% (■) of the area) and terrain unsuitable for arable agriculture (■). The map also shows 14 met-station sites within the arable area used to give representative weather in different regions (details in Madgwick et

al., 2011 supplementary information). Wheat and arable area information were from www.hgca.com/cerealsmap/version9.swf. ; (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 2050Hi climate scenarios. The maps were produced by spatial interpolation between the 14 sites.

3.5. Effects of climate change on fungal crop diseases

Coakley *et al*. (1999) and Harvell *et al.* (2002) predicted some general effects of climate change on plant pathogens. Milder winters and particularly higher night-time temperatures will enable increased winter survival of plant pathogens. Generally warmer temperatures in winter and throughout the growing season will accelerate vector and pathogen life cycles, increasing sporulation and infection efficiency of fungal foliar pathogens. A review of climate change effects on plant disease by Garrett *et al*. (2006) highlighted potential effects at different scales due to factors such as elevated temperatures (which can reduce host resistance), changes in precipitation (which influences infection conditions) and increased storm events (which influences dispersal of many pathogens). The review considered changes in crop growth in relation to increasing or decreasing disease severity (e.g. through changes in occurrence of infection conditions through altered canopy density or changes to host susceptibility). In addition, changes in crop growth or yield potential will occur and are likely to affect strategies of disease control and other crop production methods.

Various methods are possible to assess likely effects of climate change on crop diseases. These are: to use (i) detailed modelling of each individual crop-pathogen-projected climate system, (ii) inoculated outdoor and controlled environment experiments (iii) comparison of disease occurrence in locations of the world with similar climates to that projected for the UK, (iv) expert knowledge, survey data and weather-related crop disease models reported in the literature. Available resources did not permit approach (i) to be made in this project for all crop-disease combinations, but two systems (stem canker of oilseed rape, and fusarium ear blight of wheat) were investigated in detail in order to assess how changes to the timing of crop development could affect disease progress. A summary of the main findings of these two investigations is reported below (section 3.5.1), with full details in Butterworth *et al*. (2010) (canker) and Madgwick *et al*. (2011) (fusarium ear blight). Although only two diseases were studied in detail, the findings improved assessment of a full range of different diseases using approach (iv). Similarly, available resources would not permit approach (ii) for all diseases of all arable crops, with the exception of new research on cercospora leaf spot of beet, caused by *Cercospora beticola*, reported below (Appendix 1). Approach (iii) (comparative climates) found a very limited number of possible locations with climates similar to that projected for the UK (see below, section 3.5.2). The study concentrated on approach (iv) in particular by comparing traits of different diseases against those of nine key fungal diseases (Table 1) that were reviewed using published weather-based disease progress models,

published information on pathogen biology, disease distribution maps, expert opinion and disease data collected by partners in the project.

3.5.1. Detailed modelling approach combining crop/disease models with climate change scenarios

Two example diseases that are important in the UK, namely phoma stem canker (West *et al*. 2001; Fitt *et al*. 2006b) of oilseed rape and fusarium ear blight (head blight, or scab) (Brown *et al.*, 2010; West *et al.* submitted; Parry *et al*., 1995) of wheat were investigated in detail to quantify risks that climate change will increase disease severity or range.

Weather based disease forecast and climate change models were combined to predict effects of climate change on both these diseases, using multi-site disease/weather data over several growing seasons for model development and validation (Evans *et al.,* 2008; 2010; Butterworth *et al.*, 2010; Madgwick *et al*., 2011). For canker of oilseed rape, a weather-based oilseed rape growth model (STICS, Brisson *et al*., 2003) and disease forecasting models were combined with 30 runs (30 years of daily weather data based on projected climate) (Semenov, 2009), per chosen date and climate-change scenario, to produce quantitative risk assessments (Butterworth *et al.*, 2010). Similarly incidence of light leaf spot of oilseed rape, caused by *Pyrenopeziza brassicae*, was modelled under future climate projections and yield-loss relationships for both canker and light leaf spot diseases were used to produce an economic analysis (Evans *et al.*, 2010). For fusarium ear blight of wheat, a similar method used the SIRIUS wheat growth model (Jamieson *et al*., 1998; Jamieson & Semenov, 2000) to predict dates of key growth stages (anthesis and harvest) for different arable-crop growing locations of the UK using projected climate data. This provided an estimation of a revised anthesis date around which a weather-based epidemiological model was used to predict disease risk for each location using projected climate data per chosen date and climate-change scenario (Madgwick *et al*., 2011).

3.5.2. Comparative climates

One approach to investigate likely effects of climate change is to examine crops and crop diseases found currently in locations in the world with climates that are currently the same as that projected for the UK. However, very few such locations occur due to the UK's maritime climate and particularly the unique effect of the gulf-stream, which keeps the UK and other parts of north-western Europe approximately 12°C warmer than typically expected for the latitude (50°-60°), yet under the influence of relatively long summer day-length and short winter day-length. Climates that most closely match that projected for the UK in 2050 are currently found in south-western coastal France (e.g. Poitiers & Nantes) and Southern New South Wales in SE Australia (Bombala). Eastern central Argentina had the right temperature but was far too dry and other latitudes

investigated in north America had continental climates (hotter in the summer, colder in winter). Of locations with suitable climates, differences in topography, soils and agricultural systems (Bombala's primary agriculture is grazing and forestry) mean that only the French locations are of any relevance. That region has a mixture of mostly calcareous soils and grows less oilseed rape but more maize than in the UK, along with wheat and sunflower. The maize cultivation may explain a greater prevalence of *Fusarium graminearum* on wheat crops in that location (see section below). The region around Poitiers and Nantes in France has septoria leaf blotch and brown rust as the main diseases of wheat (David Gouache, pers. comm., ARVALIS Institut du végétal, France). Climate-matching tools are available for a more detailed investigation of climates similar to that predicted for the UK (Magarey *et al.*, 2007; Busby, 1991; Walker & Cocks, 1991; Sutherst & Maywald, 1985).

3.5.3. Traits based approach

To assess risks that climate change will increase severity or range for current or new diseases of major UK arable crops, changes in weather factors that affect diseases were identified (Fig 2c; i.e. temperature and rainfall for the seasons, winter, spring, summer and autumn). Duration of wetness periods at key times, and frequency of frost and severe frost (below -5°C) events were also considered. Likely responses of the nine key fungal diseases (Table 1), based on different ecotypes and of particular importance in the UK, were evaluated by review of the literature, which included interpretation of published weather-based disease models against predicted climate, disease distribution maps and review of each pathogen's biology and epidemiology. In some cases, the projected climate was considered to promote one aspect of a pathogen's life cycle but reduce another aspect. For example, with 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). Findings from studying two diseases in detail provided improved resolution about which 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 of untreated susceptible crops was predicted to increase (see below; Butterworth *et al*., 2010).

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 existing diseases and potential new diseases (currently present on the crop in other climates) as similar eco-types to one of the key nine fungal diseases.

Information about weather-crop growth interactions produced as part of the detailed study of canker of oilseed rape and fusarium ear blight of wheat was used to define important climate change effects on crop growth (e.g. timings of key growth stages), which was used in the broad-ranging review [i.e. effects of both altered crop growth stages and projected weather were assessed for each disease]. Additionally, the adaptability of each pathogen species was evaluated, based on factors such as the mode of reproduction, method of dissemination, number of infection cycles per season, population size and number of hosts (McDonald & Linde, 2002).

Although these evaluations were substantiated against crop disease data from different locations and seasons, consideration was also made to the occurrence of several seasons of weather of the type predicted for the future, which has occurred rarely if at all. 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).

Table 1. Summary of evaluation of nine key fungal diseases

Pathogen	Host	Disease	Epidemic type	Key epidemiological features		
				Dissemination	Infection and adaptability	Prediction
Mycosphaerella graminicola	Wheat	Septoria leaf blotch	Polycyclic	Airborne ascospores then rain-splashed conidia(autumn-spring)	Dry summers increase inoculum survival. Mild winter temp and wet springs favour severe epidemics. Highly adaptable	*Slight increase*
Rhynchosporium secalis	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	*Little change*
Puccinia triticina	Wheat	Brown rust	Polycyclic	Airborne uredospores (spring)	Mild winters and dry springs favour severe epidemics (e.g. 2007). High adaptability	*Sporadic*
Blumeria graminis	Wheat	Powdery mildew	Polycyclic	Airborne conidia	Mild winters and warm, humid springs favour severe epidemics. High adaptability	*Sporadic*
Fusarium graminearum	Wheat	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	*Slight increase*
Leptosphaeria maculans	OSR	Phoma stem canker	Monocyclic	Airborne in UK	Warm winters favour severe epidemics. Currently good cv resistance available. High adaptability	*Moderate increase*
Verticillium longisporum	OSR	Verticillium	Monocyclic	Soil-borne mycelium Spores long-lived	Infection occurs in autumn; disease develops only when there is a hot, dry spring (e.g. 2007) Moderate adaptability	*Slight increase*
Sclerotinia sclerotiorum	OSR, 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	*Little change*
Cercospora beticola	Sugar beet	Cercospora	Polycyclic	Seed-borne (spring) Rain-splashed conidia	Favoured by warm, wet spring weather Moderate adaptability. Dry summers will reduce epidemics slightly. High adaptability	*No change unless irrigation used*

3.6. Results

3.6.1. Detailed modelling approach combining crop/disease models with climate change scenarios

Weather-based disease forecasting models were combined with a climate change model predicting UK temperature and rainfall under high and low carbon emissions for the 2020s and 2050s. UKCIP02-based simulated weather data (Semenov, 2007) were used as inputs for the weather-based disease forecast of stem canker of oilseed rape (Evans *et al*., 2008). Multi-site data collected over a 15-year period were used to develop and validate the 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 2050s (Fig 4b). Changes in incidence of light leaf spot are also shown (Fig. 4d); the incidence is predicted to reduce slightly over the UK to levels that would not affect yield in the south (Evans *et al.*, 2010). To investigate crop-disease-climate interactions, UKCIP02 scenarios predicting UK temperature and rainfall under high- and low-CO_2 emission scenarios for the 2020s and 2050s were combined with a crop simulation model predicting yield of fungicide-treated winter oilseed rape (STICS; Brisson *et al.*, 2003) and with a weather-based regression model predicting severity of phoma stem canker epidemics (Evans *et al*., 2008). The combination of climate scenarios and crop model predicted that climate change will increase 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 England (Butterworth *et al.*, 2010; Fig 4f). However, in fungicide untreated crops of moderate disease susceptibility, the combination of climate scenarios, crop growth, disease development and yield loss models predicted that climate change will increase yield losses from phoma stem canker to up to 50% (1.5 t/ha) in southern England (not shown; Butterworth *et al*., 2010). The size of losses was predicted to be greater for winter oilseed rape cultivars that are susceptible than for those that are resistant to the phoma stem canker pathogen *Leptosphaeria maculans*.

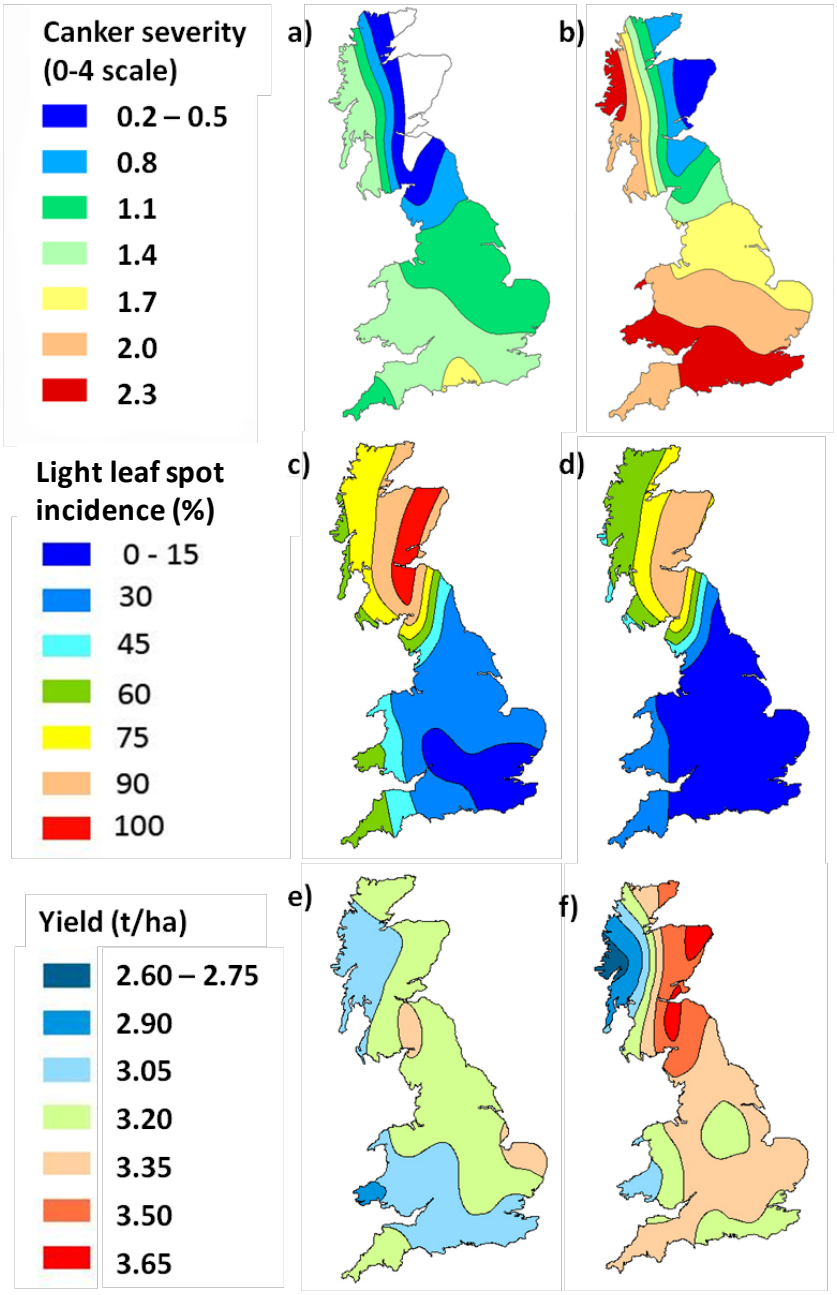


Figure 4. Impacts of climate change on severity of phoma stem canker, incidence of light leaf spot and yield of oilseed rape treated with fungicide to control diseases. Predicted severity of phoma stem canker (*Leptosphaeria maculans*) at harvest of winter oilseed rape crops (mean of resistant and susceptible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

Weather data generated for different parts of the UK under five climate change scenarios were used as input for models to predict oilseed rape yields and yield losses from the two most important diseases, phoma stem canker and light leaf spot and analyse the economic implications (Evans *et al*., 2010). With effective disease control against stem canker (the primary disease in England) and light leaf spot (primarily in Scotland), the value of the crop was predicted to increase by £13M in England and £2.5M in Scotland by the 2050s under a high CO_2 emissions scenario. Although phoma stem canker is predicted to increase in severity and range with climate change, the incidence of light leaf spot will decrease in both Scotland and England (Fig 4d). Due to the predicted increase in canker and reduction in light leaf spot, combined losses from both phoma stem canker and light leaf spot in fungicide untreated crops were predicted to increase by up to 40% (worth £50M) in southern England and some regions of Scotland by the 2050s under the high emission scenarios. However, with effective crop protection in most crops, predicted increases in yield potential should result in a net increase in yield in the UK. Such predictions illustrate the unexpected, contrasting impacts of aspects of climate change on crop-disease interactions in agricultural systems in different regions.

Fusarium ear blight was also investigated in detail using a similar approach to that for stem canker (above). The incidence of fusarium ear blight was related to rainfall during anthesis and temperature during the preceding 6 weeks. It was projected that, with climate change, wheat anthesis dates will be approximately two weeks earlier than at present. As a result, the rain-related risk of infection at anthesis did not decrease, as would have been predicted if anthesis had remained in mid-June (see Fig 2c, rainfall is projected to be almost unchanged in May but substantially reduced in June). Due to wetter and warmer conditions in spring, the model predicted a slight increase in severity of fusarium ear blight epidemics by the 2050s, particularly in southern England (Fig. 5; Madgwick *et al*., 2011). This predicted slight increase reflects purely the weather-related risk. Increased indirect cultivation, which is likely to substantially increase production of inoculum of *F. graminearum* is an additional indirect climate-related factor and as discussed by West *et al*. (2012) is likely to cause a much greater increase in severity of fusarium ear blight.

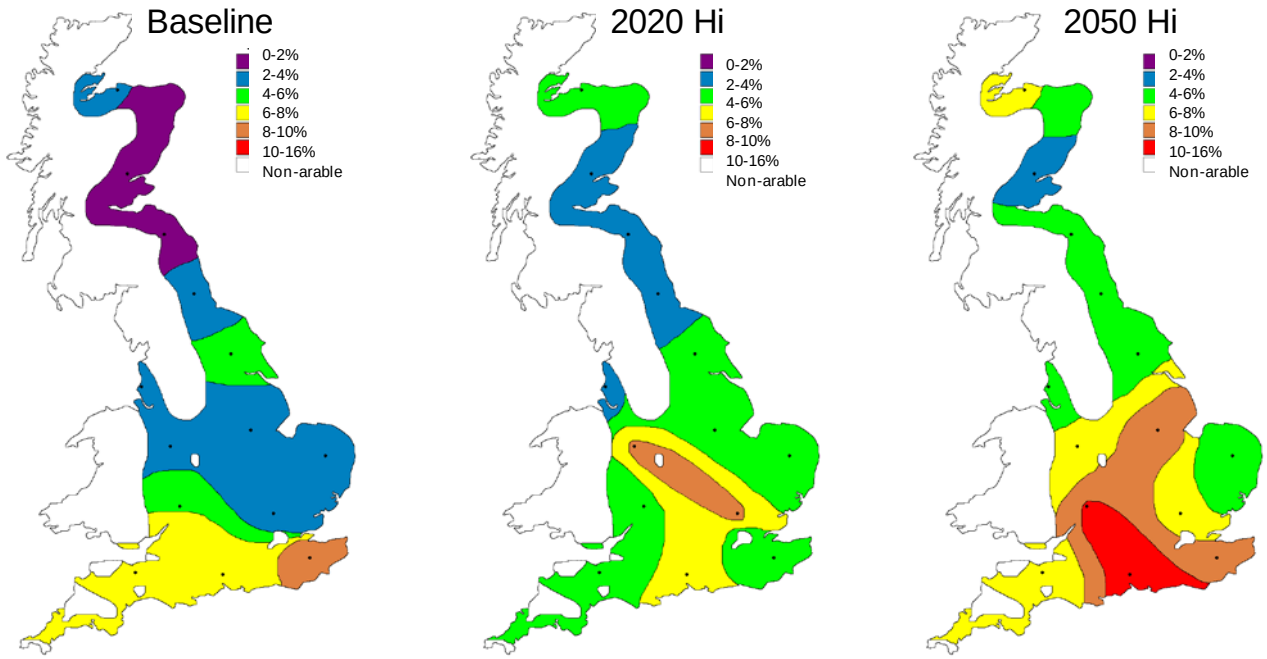


Figure 5. Maps showing the projected average fusarium ear blight incidence (% plants affected) generated by a fusarium ear blight model and based on advanced anthesis dates for three weather scenarios; baseline, 2020 high emission scenario (2020HI), and 2050 high emission scenario (2050HI). The baseline scenario is based on weather from 1960–1990. The maps were produced by spatial interpolation between the 14 sites. Adapted from Madgwick *et al*. (2011).

3.6.2. Traits-based approach: rain-splashed, polycyclic foliar fungal diseases

Septoria leaf blotch (*Mycosphaerella graminicola*) is the most important disease of wheat in the UK. The majority of UK wheat cultivars lack fully effective resistance to this disease and so control relies mainly on fungicides. However, currently there is complete resistance to MBC and QoI fungicides, and reduced sensitivity to azoles in pathogen populations (Cools & Fraaije, 2008) [for current guidelines on fungicide choice, see: http://www.hgca.com/crop-management/disease-management/fungicide-performance.aspx]. *M. graminicola* is a foliar pathogen and initiates disease by airborne ascospores in late summer and autumn but this is followed by many cycles of rain-splashed conidia and usually a second batch of ascospores in the spring. The disease distribution map available from www.cropmonitor.co.uk shows that the greatest severity is in the west of England and Wales, which is associated with increased rainfall. In summary of the literature, the disease is favored by dry summers, which increase inoculum survival (Shaw *et al.*, 2008), and wet springs which promote rain-splashed dispersal and infection (Gladders *et al*.,2001; Pietravalle *et al*., 2003; Shaw *et al*., 2008). The latent period is estimated to be 250 to 300 degree-days (Lovell *et al*., 2004). The literature reveals some differences of opinion over the effects of winter temperature on septoria epidemics – some studies suggest that cool winters encourage disease as the fungus has a lower base temperature (-2.5°C) than the plant so there is more pathogen growth and sporulation per unit of leaf production (Lovell *et al.*, 2004). Others suggest

that infection success (of spores) is promoted by milder winter weather (>7°C) and this seems to override the importance of a small amount of pathogen growth at very low temperatures (Pietravalle *et al*., 2003; te Beest, *et al*., 2009). It seems clear that these factors are likely to lead to an increase in this disease on leaves at the base of the plant over winter and early spring.

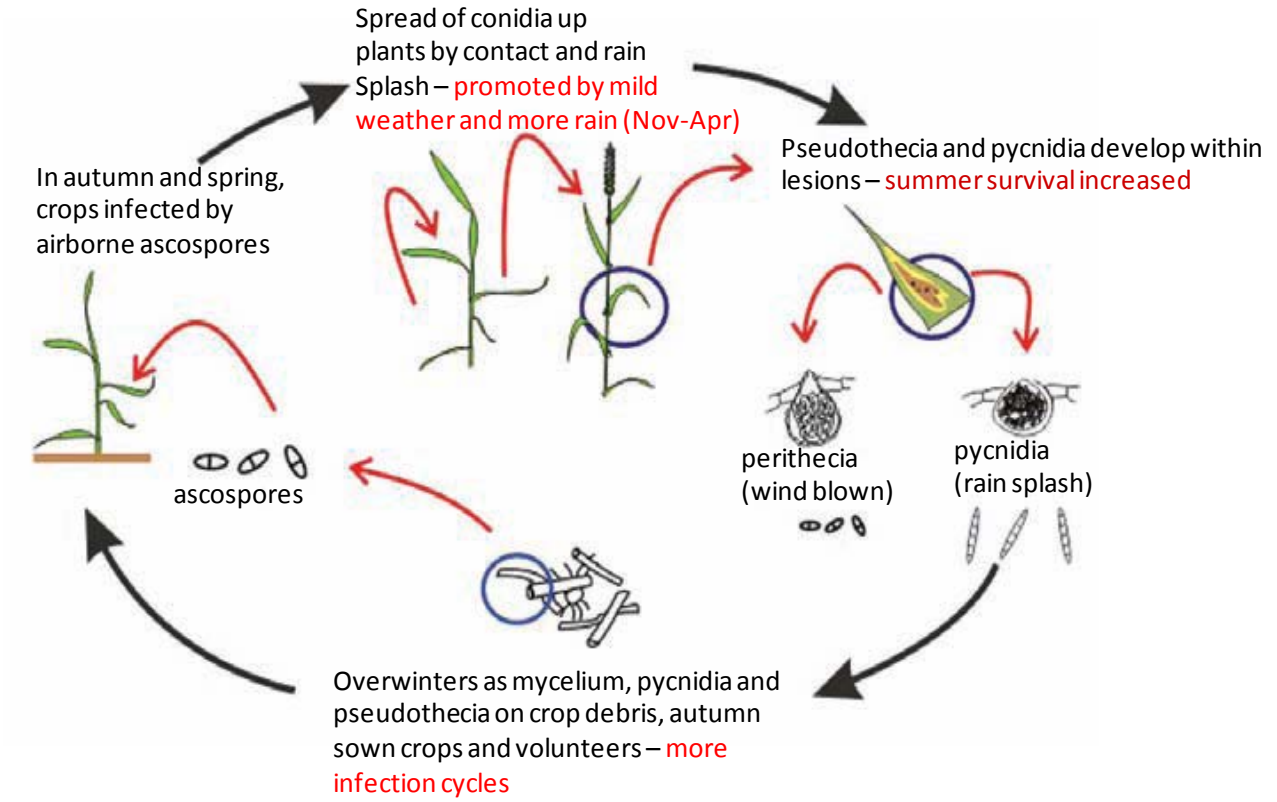


Figure 6. Summary of effects of climate change on stages in the life cycle of septoria leaf blotch (*Mycosphaerella graminicola*), adapted from the encyclopaedia of cereal diseases (Anon. 2008b)

Infection of the uppermost leaves, particularly the flag leaf, in April and May (currently) leads to yield losses. Due to the predicted advancement of growth stages (reported above) into a period when monthly rainfall is projected to be approximately the same or more than at present (April to early May, Fig 1c), we predict that infection conditions for upper leaves will be similar to those now. As there will be an enhanced capacity for inoculum production from basal leaves, a moderate increase in disease is predicted (Fig. 6). In addition, reduced slug and snail activity over summer (Willis *et al*., 2006) is likely to dramatically increase pathogen survival and inoculum production in the autumn.

Other pathogens that fit this ecotype are described briefly below.

Pyrenophora tritici-repentis (*Drechslera tritici-repentis*) causes tan spot of wheat and was the most important disease of wheat in Australia from 1999–2008 (Murray & Brennan, 2009). The pathogen is analogous to *M. graminicola* or *P. nodorum* but with a higher temperature preference. The

disease is favored by minimal tillage and warm wet weather after GS32 (currently mid-April in central England) so climate-related risk is likely to increase slightly.

On barley, leaf blotch or scald (*Rhynchosporium secalis*) is a similar ecotype to *M. graminicola* in many respects, primarily because it is a polycyclic foliar pathogen with rain-splashed spore dispersal but it lacks a capacity for widespread dispersal as there seems to be no truly airborne / sexual stage known. This may explain why it appears to be slower to adapt (e.g. resistance to fungicides) than *M. graminicola*. The pathogen also has a preference for cooler conditions than *M. graminicola* and has a long and potentially extensive symptomless phase. It may therefore not increase, or even decline slightly as also predicted for *Pyrenopeziza bras*sicae (which also has a preference for cool temperatures, see below). *Rhynchosporium secalis* survives on seed, debris and volunteers. The fact that leaf scald is not a problem on spring crops suggests that inoculum build-up over late autumn, winter and early spring is important. Like septoria (above), advancement of crop growth stages (earlier for barley than wheat in any case) means that greater rainfall predicted for mid-spring will favour this disease slightly but its preference for cool conditions may result in little change (on winter barley, very little on spring barley).

Halo spot (*Selenophoma donacis*) of barley tends to infect plants late in the season and is encouraged by wet weather, often associated with rhyncosporium leaf scald. Although infection is usually late in the season, with weather starting to become drier from May (Fig. 2c), wetter and milder weather in winter and early spring is likely to promote this disease.

Pyrenophora teres f. *teres* (*Drechslera teres*) causes net blotch of barley (*Pyrenophora teres* f. *maculate* produces small dark spots rather than the network of dark lines). This is particularly encouraged by minimal tillage, which allows better survival and spore release. Generally warmer wetter winters will encourage this disease as most infections are caused by rain-splashed conidia.

Pyrenopeziza brassicae causes light leaf spot of OSR. Detailed modelling has predicted a slight decrease, based on an existing weather-based disease model (Welham *et al*., 2004) and tested with simulated future climate data based on various climate change scenarios (Evans *et al*., 2010). Although *P. brassicae* is favoured by cooler, wet climates and is distributed primarily in the north and west of Britain (www.cropmonitor.co.uk; Gilles *et al*. 2000; Welham *et al.* 2004; Boys *et al*. 2007), it is otherwise similar to *M. graminicola* in having ascospores as primary inoculum that start epidemics in the autumn, followed by multiple cycles of rain-splashed conidia. Currently in the UK the airborne ascospores are released from apothecia on debris of the previous crop in the autumn and early winter (Oct-Dec) and again from debris of the current season's crop in March-June (McCartney & Lacey, 1990). A difference in over-summer survival compared to *M. graminicola* and

its preference for cool conditions is thought to be the reason why a slight decrease in importance is predicted for *P. brassicae* (Evans *et al*. 2010)

Alternaria dark leaf and pod spot of oilseed rape (*Alternaria brassicae*) has greatest disease severity distributed in the south of England where, like *Cercospora beticola* (below) the disease is favoured by warm, humid weather. A slight increase is therefore predicted.

Pseudocercosporella capsellae causes white leaf spot of oilseed rape and occurs in wet areas of north-west Europe, but is currently of low importance here and in other countries. However there is potential for a slight increase, particularly as rotations of OSR are shortened. Currently this pathogen appears to be a relatively weak foliar pathogen and causes little effect on stems and pods of OSR in the UK, and even where stem and pod infections occur, it is not reported to cause any significant yield reduction.

Cercospora leaf spot (CLS) of sugar beet has been regarded as a minor disease in the UK, although in 2006 there was an increase in the number of cases reported. It is potentially devastating under warm, wet/showery conditions (up to 40% yield loss) and is favoured by high temperatures (27-32°C) and high relative humidity (above 60%). Conidia are disseminated not only by rain-splash, but also by wind, irrigation, insects and mites. Research on the biology of the causal agent, *Cercospora beticola,* particularly yield loss relationships in UK conditions, is reported as Work-Package 2 of this project (see Appendix 1). It's requirement for high temperatures (> 25°C with optimum *ca* 30°C with high leaf wetness), has previously confined Cercospora leaf spot to be a problem in Mediterranean and continental climates, with the potential to defoliate crops within two weeks if not controlled. Vereijssen *et al*. (2007) recently compared strategies for control of cercospora leaf spot of sugar beet based either on disease severity thresholds or environmental (weather) conditions. They found that fungicide treatments reduced disease severity by up to 50% and resulted in significantly higher relative sugar yields than the unsprayed treatment. Fungicide treatments based on weather conditions delayed the increase in disease incidence, and were applied 1 week before treatments based on the first action threshold. This resulted in significantly greater financial returns in 2001 and in 2003 and 2004, leading to the conclusion that sugar beet growers in the Netherlands should move towards using supervised control approaches for CLS in sugar beet, although they reported that further testing of the weather-based system will be necessary. Unlike the winter and spring-active polycyclic foliar pathogens considered here, *C. beticola* is an exception because it infects and damages leaves of beet later in the season (June-September) compared to the cereal and OSR pathogens and this period is predicted to be much drier. It could remain a problem during summer in irrigated crops and whenever dew periods allow infection.

Conclusion for rain-splashed, polycyclic foliar fungal diseases

Most of these diseases are predicted to increase in severity due to more epidemic cycles, greater plant biomass and denser canopies, and wetter conditions for most of the vegetative crop growth period. Some, however, may reduce slightly if longer intercrop periods promote disease escape due to ascospore release ahead of emergence of the following crop. In other cases, drier summer conditions may reduce the breakdown of crop debris (reduced activity of detritivorous invertebrates) and therefore increase inoculum availability, which may also be better synchronised with crop emergence. Due to advancement of plant growth stages, drier weather, predicted to be from early May onwards (Fig. 2c) is unlikely to lead to a reduction in this type of disease on wheat and barley because all the upper leaves will have been produced by then. Cercospora of beet may be an exception, since it infects and damages leaves of beet later in the season (June–September), a period that is predicted to be much drier.

3.6.3. Traits-based approach: dry/air-dispersed biotrophic foliar fungal pathogens

Brown rust (*Puccinia triticina*) is a foliar pathogen of wheat and initiates disease by many cycles of airborne uredospores, particularly in the spring. The disease distribution map available from www.cropmonitor.co.uk shows the greatest severity in the south and east of England and Wales, which is associated with relatively warm and dry conditions. Dry springs favour severe epidemics as more spores are produced and dispersed, particularly because dry conditions in spring are usually associated with dew at night, which allows infection. A study by Roche *et al*. (2008) modelled effects of climate change in four regions of France. The study assumed that inoculum was not limiting and simulated daily weather events throughout annual periods to investigate effects of changes in wetness periods under climate scenarios on infection success and disease. They concluded that there was no particular trend discernible concerning the infection rates due to 'opposite evolutions of two major variables: temperature and surface wetness duration'. They also predicted that yield loss in diseased crops would not change from the current situation because they predicted a slight decrease in healthy crop yield combined with a slight reduction in brown rust disease. Assuming this study confirms little change in infection success, potential changes in the amount of inoculum, which was not included in the study by Roche *et al*. (2008), suggests that there could be an increase in epidemics following mild winters and also a decrease in epidemics in seasons following particularly hot, dry summers. Enhanced epidemics may be associated with a predicted increase in plant biomass due to more CO_2, which will also promote sporulation and milder winters will increase survival on volunteers and new crops (Fig. 7). However, hot, dry weather in the summer, leading to drought conditions may decrease over-summer survival on volunteers and wild grasses. In the south of France, severe summer droughts have been associated with reduced brown rust epidemics for the following one or two years and this is presumed to be due to a major reduction in the *P. triticina* population as volunteers and grass

hosts senesce due to water-stress (David Gouache, pers. comm.). Brown rust in the UK may therefore also become sporadic with potential for elevated severity of epidemics following a wet summer and mild winter but reduced amounts if the previous summer(s) had a severe drought.

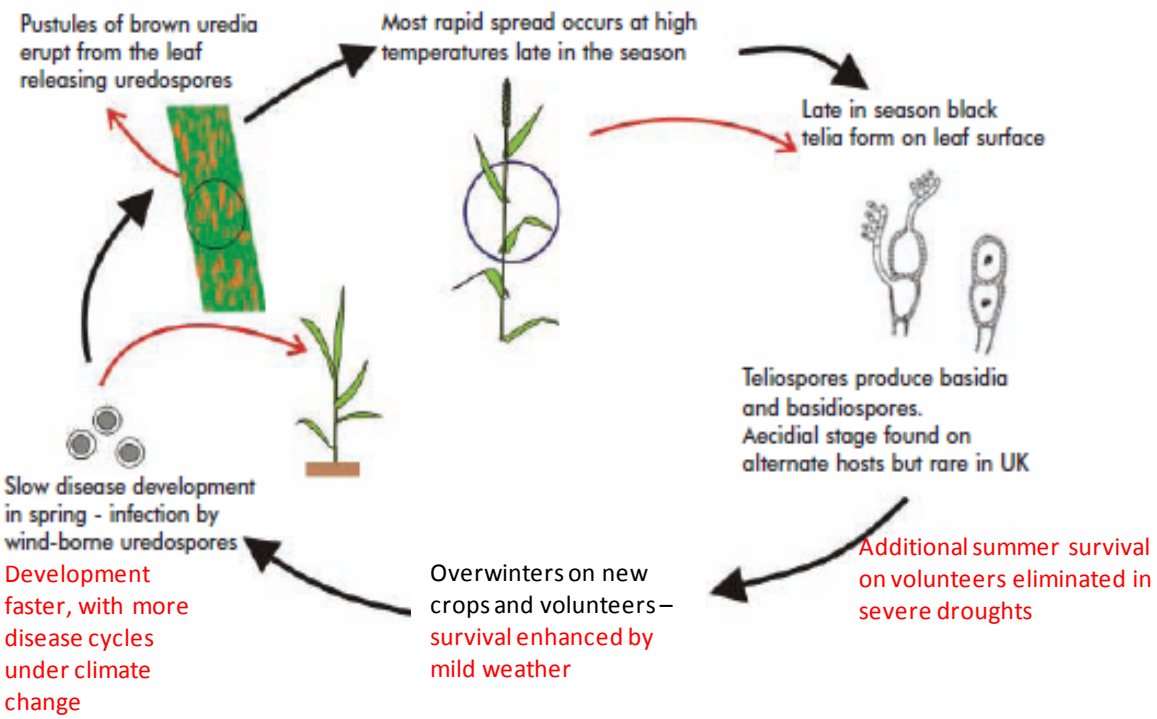


Figure 7. Summary of effects of climate change on stages in the life cycle of brown rust (*Puccinia triticina*) adapted from the encyclopaedia of cereal diseases (Anon. 2008b)

The majority of UK wheat cultivars are susceptible to yellow rust (*Puccinia striiformis*). The disease is globally more important than black stem rust and is increasing in Europe and the USA, possibly due to new strains emerging that can tolerate higher temperatures (Milus *et al.* 2009). Winter survival and conditions favouring spore production are key drivers of epidemics (van den Berg & van den Bosch, 2007; te Beest *et al*., 2008). The pathogen is reported to survive temperatures as low as -5°C, but we have successfully infected plants in lab conditions using spores rubbed directly from pustules on dry leaves that were kept frozen for five months at -20°C (JS West, unpublished). As with brown rust, epidemics advance in dry clear spring days (spore dispersal during daytime, infection in dew films at night). Epidemics are predicted to start earlier (March) and continue longer, particularly for strains that can tolerate temperatures as high as 25°C.

Powdery mildew (*Blumeria graminis*) is distributed mainly in the north and west of England and Wales (www.cropmonitor.co.uk). Windy weather in December to February was associated with epidemics but temperature, RH and rain in April-June were best predictors of severity (te Beest *et al*., 2008). Powdery mildew may be less affected by summer drought than the rusts as it can survive as cleistothecia, which release ascospores, but epidemics will still be reduced by

particularly hot summers. Based on this, epidemics should become slightly more severe and sporadic.

Black stem rust (*Puccinia graminis*) is a rare visitor to the UK 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. The alternate host, *Berberis*, appears to be too sparsely distributed in the UK to allow the sexual stage to persist (Shaw & Osborne, 2011). The optimum temperature is >20°C, so warm air-streams from southern Europe in late spring could provide both inoculum and infection conditions. However, temperatures below 15°C normally inhibit development of the disease so it rarely occurs under UK conditions. Race Ug99, however, has a lower temperature optimum and since it has recently spread from central to South 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).

For the present, epidemics are likely to remain rare and occur too late in the season to be a problem in the UK in most years but parts of southern Europe are under threat and the UK could be affected if the Ug99 race establishes in southern Europe.

Rusts and powdery mildews of barley should follow a similar pattern to that predicted for the corresponding wheat diseases. The earlier maturation of barley will mean that growth stages up to flowering will, on average, experience milder, wetter weather, while flowering, grain-filling and harvest should be in progressively drier, warmer conditions than at present. There is scope for summer droughts to reduce the impact of these pathogens in the following growing season. Similarly, crown rust (*Puccinia coronata*) of oats is likely to become more severe following mild winters and warm spring weather.

Downy mildew of oilseed rape (*Hyaloperonospora parasitica;* actually an oomycete rather than a fungus) also infects other brassicas and some related crucifers and is favoured by temperatures around 15°C (Smith *et al*., 1988). It is usually of minor importance, occurring on older basal leaves. Little change is predicted.

Powdery mildew of oilseed rape (*Erysiphe cruciferarum*) can be extensive on leaves of oilseed rape plants, although it has little effect on yield. However, it can also occur on pods in relatively dry conditions and has potential to advance the onset of senescence. A moderate increase (on pods in May and June) can be expected on susceptible cultivars.

Conclusion for dry/air-dispersed biotrophic foliar fungal pathogens

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 drought conditions). Epidemics become severe when dry clear weather in spring allows sporulation and dispersal and these days are typically followed by dew films at night, which allows infection. This weather combination is not likely to change in frequency very much. 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 mildew) mean that epidemics of at least one or other will be sustained well into the grain filling period. Generally better winter survival will lead to earlier epidemics and possibly more spring sunshine hours and more plant biomass will also increase sporulation, particularly of yellow rust. It is therefore likely that there will be a moderate increase in these diseases on average but with large differences from year to year.

3.6.4. Traits-based approach: upper leaf and, or ear/flower infecting fungi

Fusarium ear blight (Figure 8; West *et al*., 2012; Parry et al., 1995) is caused by a complex of species of the genus *Fusarium*, *Giberella* and *Microdochium*.

Species of main concern in the UK are DON toxin producers *F. culmorum* and *F. graminearum.* There is a trend of increasing incidence in UK but it is not clear that this is weather-related as changes to cultivars, tillage practices and an increase in maize cultivation may also be responsible.

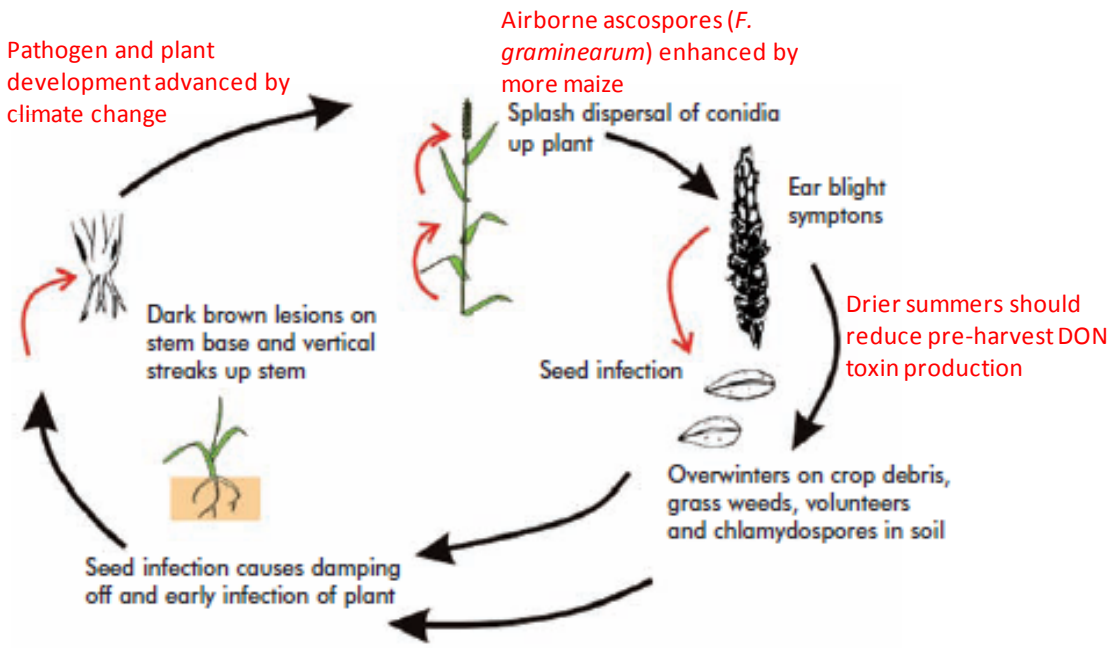


Figure 8. Summary of effects of climate change on stages in the life cycle of fusarium ear blight, *Fusarium spp.* and *Microdochium nivale* adapted from the encyclopaedia of cereal diseases (Anon. 2008b)

No UK wheat cultivar is fully resistant. In relation to the ear blight disease, wheat is only susceptible for a short period during flowering and disease risk is increased by warm springs and rainfall just before and during anthesis. Due to the anthesis of wheat being predicted to advance into late May, infection conditions at flowering are likely to remain similar to those at present. However, increased inoculum production, provided by infected maize debris is likely to cause at least a slight to moderate increase in this disease (on average) but with large seasonal differences. Some species of *Fusarium* and *Microdochium* also cause infections of the roots, stem base or basal (winter) leaves (see 4. below).

*Parastagonospora nodorum (*Leaf and glume blotch) is increased by spring rainfall on leaves but should remain of low importance unless SO_2 levels increase (Shaw *et al*., 2008).

*Tilletia tritici (*Bunt; stinking smut) is wind- and soil-borne, monocyclic and analogous to *F. graminearum* (but without the maize inoculum effect). It should remain rare but a slight increase may occur after dry summers.

*Tilletia controversa (*Dwarf bunt) is not in the UK currently and is prevented by seed treatments. Its spores in soil survive longer than those of *T. tritici*. It needs cool winter weather before infection so the weather-related risk shouldn't change.

Urocystis agropyri (Flag smut of wheat) is included here as it can be seed-borne and is similar to bunt except that it mainly sporulates on leaves. It is not present in UK but occurred in Essex in 1998 (Anon 2008b) and could return as warm, dry summers will increase risk.

*Ustilago nuda (*Loose smut) is favoured by dry conditions at flowering, which means that little change in disease risk can be expected. In practice it is likely to remain rare as it is easily detected in seed-crops.

Claviceps purpurea (Ergot) is favoured by wet conditions just before and at flowering so a slight increase in risk is possible but in practice likely to remain rare.

Alternaria spp. and *Cladosporium spp.* (Black point) are favoured by wet weather after flowering should the risk should decrease. Since these fungi are ubiquitous, they will always be a problem in wet summers.

A recently emerging ear disease of wheat is wheat blast (*Magnaporthe sp.*) which caused a severe epidemic in parts of Brazil in 2009. It seems to have crossed from wild grasses (pers. comm. Alex

Amaral, Embrapa, Brazil). Hot temperatures coupled with high humidity seem to be required for epidemics so this is unlikely to affect the UK but it may be a problem in other regions if it spreads from Brazil.

Karnal bunt, caused by *Tilletia* (Neovossia) *indica,*infects wheat, durum, rye and triticale. The risk of establishment was estimated by Sansford *et al*. (2008) in part by applying a published karnal bunt disease model to the UK (and other parts of Europe). They showed that cool weather, rainfall and high humidity during the '*heading*' period (from just before anthesis, ~ May and June) were favourable for infection and disease development in many places of Europe.

Ramularia leaf spot (*Ramularia collo-cygni*) of barley is primarily a foliar disease but it is included here as it is also seed-borne. The disease appears to have a long cryptic or asymptomatic phase like *Rhyncosporium secalis* or *Pyrenopeziza brassicae* but symptoms appear on older plants and are particularly exacerbated by heat, light and/or temperature stress. Spores are airborne rather than splash-dispersed but it is included here due to similarities with other members of this group. It seems likely that this disease will also increase.

Leaf stripe (*Pyrenophora graminea* [*Drechslera graminea*]) of barley is most obvious as a leaf disease, causing leaf stripe but air-dispersed conidia infect ears and the pathogen is primarily seed-borne. Little change in climate-related disease risk is expected.

Mycosphaerella brassicicola prefers cool, moist regions of the world and infects oilseed rape and other brassicas to cause ringspot. Infection is primarily by ascospores, released after rain (Smith *et a*l., 1988) and also by infected seed. Little change is expected as growth stages will advance to be earlier in the spring.

Conclusion for upper leaf and, or ear/flower infecting fungi

Due to drier conditions in late spring and summer but an advancement of crop growth stages, we expect little change in these diseases. An exception is *F. graminearum* which may increase due to an indirect effect of increased maize cultivation, increasing the pathogen population and Ramularia leaf spot may also be exacerbated by heat stress.

3.6.5. Traits-based approach: monocyclic root and stem fungal diseases

Monocyclic root and stem fungal diseases (autumn spore release)

Based on conclusions for phoma stem canker (*Leptosphaeria maculans*) (Fig. 9; and see detailed modelling description above), severe epidemics of this group of diseases are favoured by mild, wet autumn, winter and spring weather. High evapo-transpiration stress in the summer, before harvest, may also exacerbate the yield-loss relationship per unit of disease.

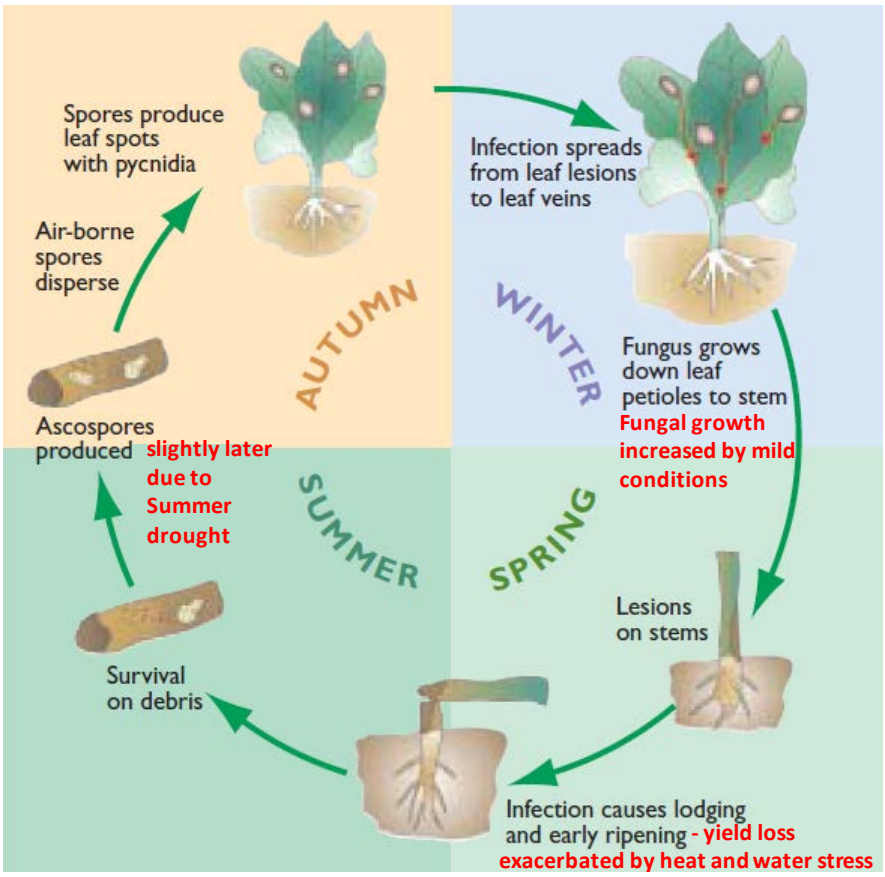


Figure 9. Summary of effects of climate change on stages in the life cycle of phoma stem canker, *Leptosphaeria maculans* adapted from the HGCA publication: Oilseed rape - a grower's guide (Anon, 2005b)

An analogous wheat disease is eyespot (*Oculimacula acuformis* and *Oculimacula yallundae; Helgardia acuformis* and *Helgardia herpotrichoides*) as this is also monocyclic, with autumn-winter infections of the stem base initiating infections. The final disease severity depends on thermal time through winter and spring (Bock *et al*. 2009) so it should increase moderately in severity. Foot rot of cereals, caused by *Cochliobolus sativus* is similar to *Fusarium*, but is included here since damage is primarily by the root and stem-base infection. It is reported to occur in warmer countries than the UK so is also likely to become more severe in the UK.

Monocyclic root and stem fungal diseases (spring spore release)

Kauserud *et al*. (2010) reported that between 1960 and 2007, there was a trend towards spring-fruiting 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 range of vegetables, is likely to release spores in synchrony with earlier flowering of crops like oilseed rape. For Sclerotinia stem rot, the severity of epidemics is greatest in the west of England and Wales (http://www.cropmonitor.co.uk/). Epidemics are monocyclic and sporadic since ascospore release, petal fall and rain or high RH need to coincide. The disease is likely to remain sporadic. Closer

rotations and inclusion of other susceptible hosts (beans, peas, sunflower [potentially a new crop under climate change] and various vegetables) also increases risk. Spore release should advance after milder winters to stay synchronised with flowering but disease progress may be faster in warmer spring conditions so infection efficiency will increase (disease escape decrease) slightly, hence there is little change in weather-related risk (Fig. 10).

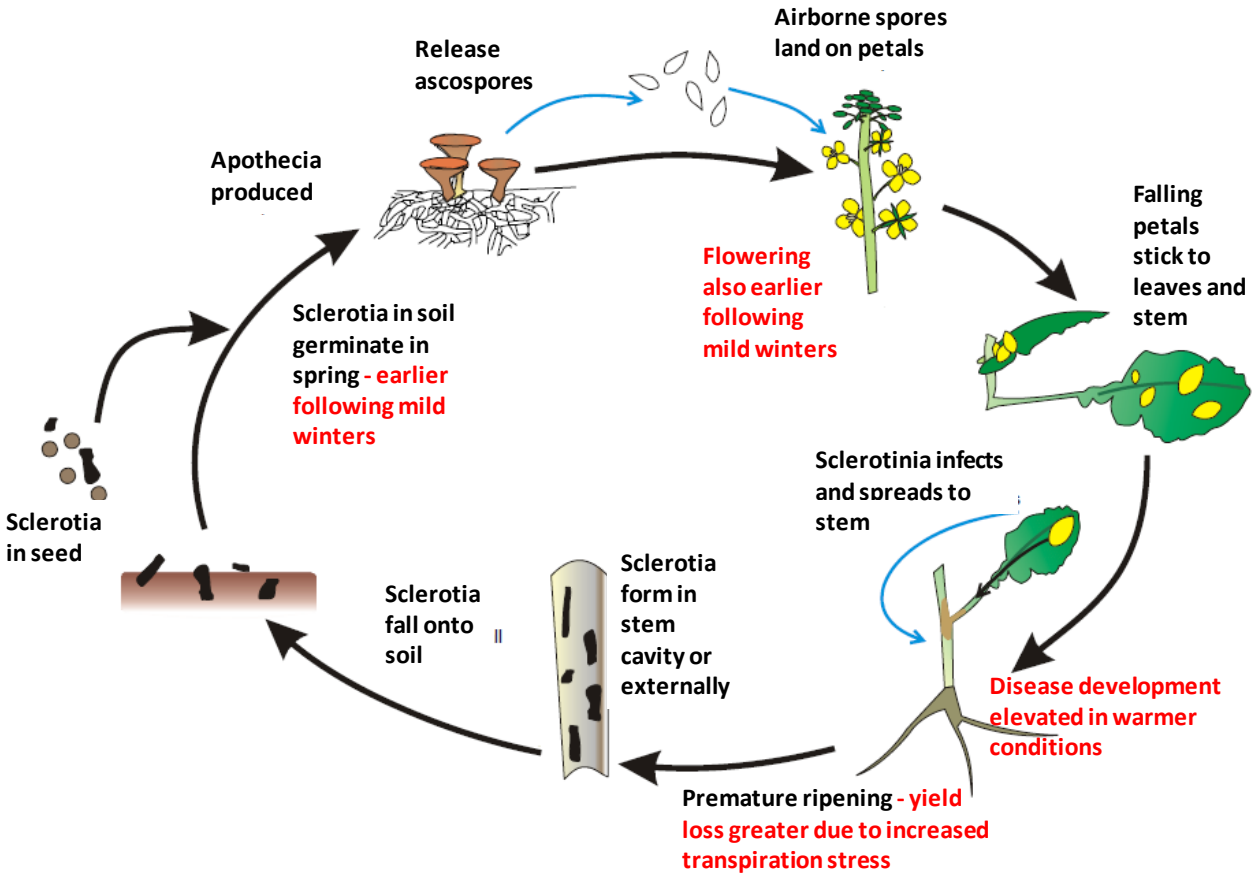


Figure 10. Summary of effects of climate change on stages in the life cycle of Sclerotinia stem rot (*S. sclerotiorum*) adapted from the HGCA publication: Gladders *et al*. (2008)

Monocyclic root and stem fungal diseases (soil borne)

Verticillium longisporum causes verticilium wilt of oilseed rape. Infection occurs in the autumn; but disease develops to a noticeable extent only when there is a hot, dry spring (e.g. as in 2007; Gladders 2009). The prevalence of this disease may be currently under-estimated in the UK. Yield loss is exacerbated by dry and hot conditions in late spring/early summer. Closer rotations will encourage the disease and warmer spring-summer weather should increase risk.

Take-all (*Gaeumannomyces graminis* var. *tritici*) is the most damaging root disease of wheat in the UK. Options for control have been limited as few fungicides are effective and no cultivars are completely resistant. Good crop rotation and seed dressing with fungicides, plus use of more resistant cultivars is advised. Take-all is considered here as analogous to *Verticillium* since disease impact is worse in hot, dry spring/summer. In parts of Australia, increased rainfall due to climate

change was predicted to decrease this disease due to reduced survival (interactions with other microbes) (Chakraborty *et al*., 1998). However, effects of increased winter rainfall and decreased summer rainfall, predicted for the UK, are difficult to assess and further research is required. Currently, reduced rainfall in April-June appears to reduce disease severity and mild winters advance development (pers. comm..V. McMillan, Rothamsted Research). Predictions suggest more rain in April and less in June so on average there appears to be little change likely.

Thanatephorus cucumeris (*Rhizoctonia solani)* is a soil borne pathogen with a wide host-range including cereals and grasses (Rhizoctonia stunt), legumes, oilseed rape (brown girdling root rot), sugar beet and potato. Direct effects of climate change are difficult to estimate but as with examples above, root infection may make plants more susceptible to heat and drought stress.

Sharp eyespot (*Ceratobasidium cereal* [*Rhizoctonia cerealis*]) infects all UK cereal crops from autumn to spring and progress is favoured by temperatures around 9°C. Effects of summer droughts and wetter winters are not clear and further work is required.

Omphalina patch (*Omphalina pyxidata*) is a soil-borne disease of barley and wheat with the causal fungus surviving as sclerotia but producing fruiting bodies (small mushrooms) which release basidiospores in mid-late winter. It seems to be sporadic and that is likely to continue.

Snow mould (*Monographella nivalis* [*Microdochium nivale*]) is a soil and debris-borne disease that is particularly severe on barley after periods of snow cover and should therefore reduce in occurrence. However, it also has an ear-infecting stage (see 3, above).

Snow rot (*Typhula incarnate*) infects cereals, mainly barley, either by mycelium or airborne spores released from germinated sclerotia. As with snow mould and as the name suggests, it is more severe after periods of lying snow and should therefore reduce in occurrence.

Fusarium wilt (*Fusarium oxysporum*) has various forma speciales that are specialised to specific hosts. Most are favoured by high temperatures. *Fusarium oxysporum* f. sp *conglutinans* infects crucifers such as oilseed rape and is affected by high temperatures, causing apparently healthy plants to develop a yellows wilt in temperatures above 17°C (Smith et al., 1988). This is likely to increase.

Club root of oilseed rape (*Plasmodiophora brassicae*) is favoured by warm (20°C) and moist soils (Anon. 2001) so it is difficult to predict any change. However, control by can be achieved by raising soil pH to 7 and currently by resistant cultivars of oilseed rape.

Conclusion for monocyclic root and stem fungal diseases

A common feature of monocyclic root and stem-infecting pathogens 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 (due to increased thermal time), no change in spring-infecting root and stem pathogens (both pathogens and crop will advance in development) and for soil-borne pathogens, there is a great deal of uncertainty as to the likely impact of climate change due to little information being available at present – further research is suggested.

3.6.6. Effects of climate change on bacteria, phytoplasmas and viruses

Virus diseases

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 & Stork, 1995). Already, mild winters have been associated with an increase in BYDV in cereals and virus diseases of sugar beet (Harrington & Stork, 1995; Thomas, 1989; Fig 11).

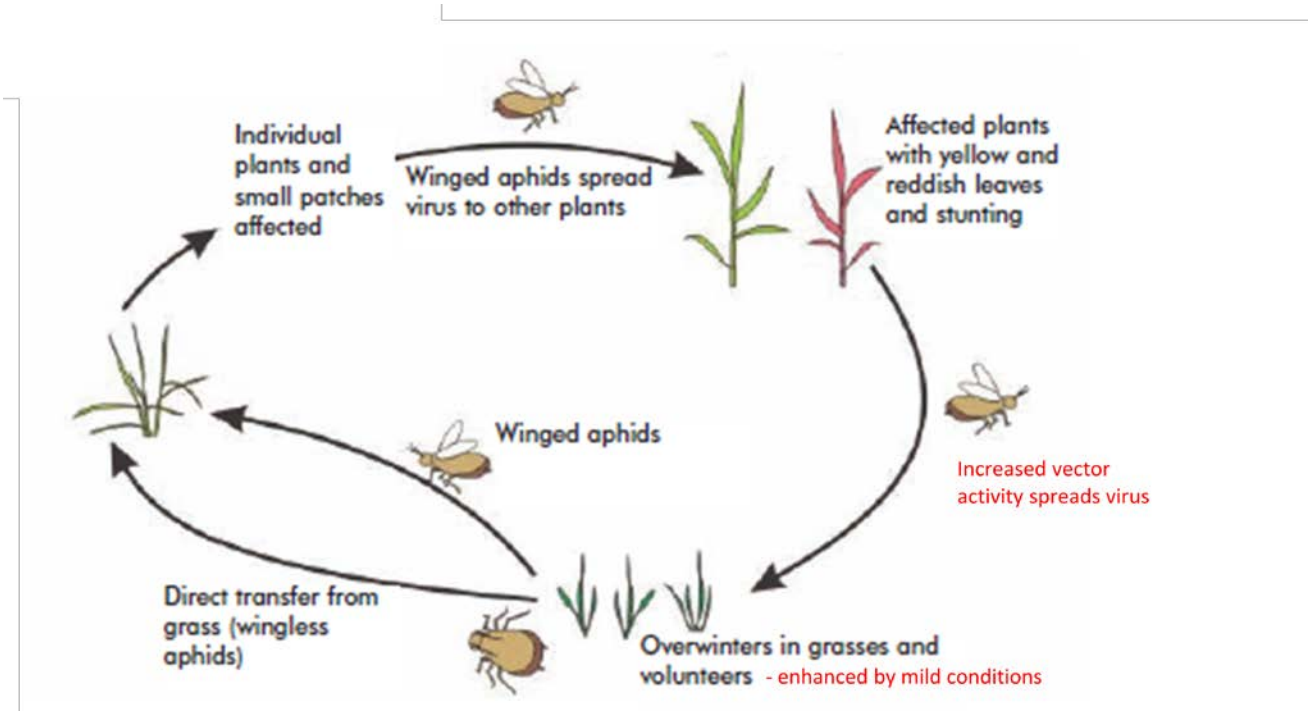


Figure 11. Summary of effects of climate change on stages in the disease-cycle of BYDV, vectored by aphids, adapted from the encyclopaedia of cereal diseases (Anon. 2008b)

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 white fly, 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 (see supplementary information B).

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

Bacteria and phytoplasma diseases

Currently bacteria are not a problem of UK arable crops but they can affect some vegetable or horticultural crops. *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 is seed/soil borne and rain-splashed with infection via hydathodes or wounds. It is probably under reported in the UK. *Pseudomonas syringae* pv. *maculicola* causes pod rot of oilseed rape but is rare or absent in the UK. Considering drier conditions are projected to occur from May, it is likely to remain rare.

Phytoplasmas, like virus diseases, are probably under reported in the UK. Many are vectored by insects and so there is potential for an increase in their importance as reported 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. Summary of effects of climate change on arable crop diseases

Disease type	Example	Prediction
Winter-spring foliar-infecting polycyclic rain-splashed fungus	*Mycosphaerella graminicola*	Slight increase (with a few exceptions – e.g. cool-preferring *P. brassicae*)
Dry air-dispersed polycyclic foliar fungus	*Puccinia triticina*	Sporadic – capacity for more severe and less severe seasons
Upper leaf and ear/flower infecting fungus	*Fusarium* spp	Little change except an increased risk for *F. graminearum*, flag smut, karnal bunt and

		Ramularia
Monocyclic root and stem-infecting fungus (above-ground autumn-winter infection)	*Leptosphearia maculans*	Increase in severity and yield loss per unit of disease
As above (above-ground spring infection)	*Sclerotinia sclerotiorum*	On average, little change in incidence or severity, possible increase in yield loss per unit of disease
As above (root infecting)	*Verticillium* sp.	Varied/unknown response with respect to disease severity, probable increase in yield loss per unit of disease
Insect vectored virus	BYDV	increase
Soil-borne virus	Wheat soilborne mosaic	Little change – depending on rainfall at location
Phytoplasma (insect vectored)	Aster yellows	Increase

3.6.7. Effect of climate change on control strategies

Changes in the efficacy of control strategies may occur due to factors such as a decrease in frequency of suitable spray conditions for autumn and winter spray applications and an increased likelihood of water-logging over winter, preventing the use of farm machinery. More rapid leaf production in autumn and spring would reduce the period of protection conferred by a fungicide spray as active chemicals on leaves are diluted by leaf expansion and as new, unsprayed leaves unfold. Additionally there are likely to be subtle changes in the rate of breakdown of applied agrochemicals under slightly warmer temperatures. The greatest changes are likely to be a need to respond to earlier disease epidemics, particularly those caused by polycyclic foliar pathogens, rather than relying on the currently accepted crop growth-stage regulated application dates (i.e. T1 and T2 applications to cereal crops). Due to changes in crop canopy densities and milder winters that will advance both crop growth and disease epidemics, T0 sprays could increase in importance. Leaf production of cereals in mid-late spring may also become so rapid that the timings of T1 and T2 sprays (relative to growth stage) will need revision in order to achieve cost-effective/optimal protection.

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 & Osborne (2011) put forward the plausible argument that to respond to unpredictable and potentially sudden changes in disease pressures, maintenance of publically 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 preserving accessions with traits currently thought to be

useful. Since some mechanisms of resistance are temperature sensitive, it has been suggested to assess the performance of breeding material and new cultivars under warmer conditions than found in the UK, e.g. in France (Evans *et al*. 2008)

On the subject of biological control or at least useful effects of naturally-occurring microbes, further research is needed to investigate impacts of environment change (both climate and atmospheric gases) on beneficial phyllosphere and rhizosphere microbes.

3.7. Conclusions and knowledge gaps for future R&D

Many known diseases will on average change in importance only very slightly as regions of production of particular crops will tend to move northwards. Despite this, some changes to disease risk are likely and are summarised in Table 2. Insect vectored virus and phytoplasma diseases are a group of pathogens that, in general, are likely to become more important due to greater vector activity. Despite little long-term average change in certain diseases, more extreme or variable weather may make some (e.g. rusts and powdery mildew) more sporadic. The sporadic nature of epidemics of these obligate foliar diseases is likely to be due to greater winter survival in mild winters, which will enhance epidemics while dramatic reductions in pathogen populations will follow severe summer droughts, which will kill 'green bridge' volunteers and wild grasses. Epidemics of these obligate pathogens will therefore depend on combinations of favourable and unfavourable summer and winter weather over more than one season. Summer droughts will not affect most necrotrophs, which survive saprophytically, and these may even be enhanced by reduced destruction of crop residues (by molluscs and other invertebrates) in dry summer weather, leading to increased inoculum production in the autumn – unless spore release occurs before emergence of the succeeding crop. An improved understanding of both crop cultivation and pathogen survival and maturation is therefore important for development of disease-progress models to predict effects of climate change. Adaptability of pathogens to climate change can be considered using the approach reported by McDonald & Linde (2002), based on relative amount of sexual to asexual reproduction, method of dispersal, number of disease cycles per season 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.

Where crops remain in their original geographical range, particularly at the southern parts of their distribution, generally warmer conditions (increased thermal time) will exacerbate those root and stem diseases that first infect hosts during the autumn and winter, such as stem canker of OSR, eyespot and take-all of wheat. In contrast, spring-infecting root and stem pathogens are not likely to change significantly as their development is likely to advance only as crop development also advances. However, increased transpiration stress, heat or drought stress is likely to increase yield

losses per unit of disease for many stem and root rots and any foliar diseases that promote water loss from leaves. Furthermore in certain cases, disease development may be worsened if warmer temperatures reduce components of disease resistance. 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. Breeders also need to consider that current host resistance may be ineffective at only slightly higher temperatures that occur now in other countries where the crops are grown (Huang *et al*., 2006).

Increased CO_2 concentrations will lead to denser crop canopies, which may slightly encourage a range of foliar diseases (rusts, powdery and downy mildews, and leaf blotch or spots) but in contrast a lower density of stomata may slightly reduce infection efficiency by those pathogens that infect via stomata. A current knowledge gap exists as to the effect of increased CO_2 concentrations on various aspects of pathogens' lifecycles. Increased CO_2 may have various positive and negative direct effects on plant pathogens (systems studied so far have tended to show higher fecundity but longer latent periods). Further research using FACE systems is needed to investigate combined effects of climate change and enhanced CO_2 on plant diseases (Eastburn *et al*. 2011).

Due to changes in crop canopy densities and milder winters that will advance both crop growth and disease epidemics, T0 sprays could increase in importance. Leaf production in mid-late spring may also become so rapid that the timings of T1 and T2 sprays (relative to growth stage) will need revision in order to achieve optimal protection. New crops such as maize could increase common wheat pathogens such as *Fusarium graminearum*. Sunflower may be introduced to southernmost England and this new crop may escape crop-specific diseases at first but will still be prone to generalists such as *Sclerotinia sclerotiorum* particularly where known field-crop hosts such as OSR, peas, and carrots are currently grown.

These general predictions about effects of climate change on epidemic severity and control methods for a wide-spectrum of arable crop diseases, based on direct effects of weather on the mode of dispersal, epidemic type and infection conditions, along with indirect effects such as changes to crop rotation, provide strategic guidance for adaptation to climate change to benefit growers, advisors, breeders, the AgChem industry and policymakers. However, despite our recommendations made here, introductions of new pathogens, changes in farm practices including new crops grown, complexities of climate change projections (jet-stream changes may make it colder!), and the biotic responses to this, makes these predictions of the future impact of climate change on plant diseases relatively uncertain. Therefore it is essential to maintain capability to monitor crops for diseases, identify new diseases and for future resistance-breeding, to draw on genotype collections of crop species that preserve as much genetic diversity as possible. Climate

change offers the opportunity of increasing crop productivity and diversifying cropping systems, and emphasises the need to produce arable crops with a low GHG footprint, while maintaining a secure and stable food supply.

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SUPPLEMENT A

List of maize diseases that also infect wheat

SUPPLEMENT B

Pathogen characteristics (wheat and OSR diseases)

APPENDIX 1: IMPACT OF *CERCOSPORA BETICOLA* UNDER UK CONDITIONS 2008-2011.

Introduction

Cercospora leaf spot, caused by *Cercospora beticola* Sacc., is normally associated with hot conditions in continental or Mediterranean countries (preferably over 25^{o}C with high leaf wetness) and has only been noticed on occasional isolated plants or in small patches in the UK in past years. Usually, this has occurred in hot summers and where irrigation has been used, as these are ideal conditions for its development. It has gradually been spreading northwards in Europe and started causing significant problems in Eastern Holland in the late 1990s. The disease can be devastating once established, since the small brown spots rapidly coalesce, leading to defoliation and severe yield losses. However, in 2006, probably as a result of the hot July and wet August weather, widespread cercospora was seen in a number of crops in the UK, particularly in September/October. Because of this late development it was unlikely to have caused much, if any loss of yield but vigilance will be necessary in future years. If summers in the UK continue to be warmer than average this disease may become endemic. Much work is being undertaken across Europe and the US to identify germplasm that is resistant to the disease, but currently fungicides remain the main option for controlling the disease. In the UK, the fungicides Punch C (flusilazole), Spyrale (difenoconazole and fenpropidin), Escolta (cyproconazole and trifloxystrobin) and Opera (epoxiconazole and pyraclostrobin) all have activity against cercospora. In countries like The Netherlands, Italy and the USA weather-based disease forecasts are used to warn growers of the risk of the disease each summer. Potentially, cercospora may well replace powdery mildew as the main foliar pathogen of sugar beet in the UK and future control strategies will have to take this into account.

Aim

To determine the economic importance of *Cercospora beticola* under UK conditions.

Methods

To determine the threshold for economic damage of cercospora leaf spot under UK conditions, artificially inoculated trials were conducted in 2008, 2009 and 2010 on a non-beet growing farm at the Woburn Estate, Rothamsted Research. Plots were sequentially inoculated with cercospora spore and mycelial macerate suspensions following standard procedures described in the next paragraph (as used by IfZ, Germany) usually in June, July and August, to simulate a primary infection incidence of 0, 1, 5 and 10% of plants per plot. Spreader plants were not used as this would not allow a controlled infection process. The trial was sprayed with quinoxyfen to control powdery mildew (this product has no activity against cercospora leaf spot). Symptoms were

monitored and the trials taken to harvest to determine the impact of cercospora on root and sugar yields and impurities.

To inoculate sugar beet plants at each time point, forty V8 agar plates individually cultured with an agar plug of *Cercospora beticola* were produced. These plates were placed in a Sanyo incubator for 3 weeks at 30°C with 16 hours daylight. The colonies were then excised, weighed and made up to one litre with distilled water before being liquidised in a Waring blender for 90 seconds; the number of conidia was counted. The cercospora suspension was applied via a Hozelock hand sprayer (without filter) onto beet leaves in each trial.

In addition, British Sugar field staff examined plants in fields visited in August and September (*ca* 500 sugar beet fields each year) and recorded the presence of cercospora. Samples of leaves were sent to Broom's Barn for confirmation. This survey was based on a random but stratified sample of fields to provide a robust survey, fully representative of the fields sown to sugar beet each year.

Results

Impact of *Cercospora beticola* on sugar beet yield

Plots were inoculated with cercospora suspensions on three occasions each year (7 and 20 August and 4 September 2008; 30 June, 24 August and 3 September 2009 and 25 June, 30 July and 25 August 2010). Unfortunately, throughout the study period, the predominantly cool, damp summer conditions were not conducive for disease development or spread, although the inoculation procedure was successful as primary infections were established in most cases during the three year period (Fig. A1).

2008 – Although plants were inoculated with suspensions of cercospora throughout August and September, symptoms were only recorded on those plants infected at the beginning of August; no secondary spread occurred as conditions were not conducive for disease development (Fig. 2). Consequently, when the trial was harvested and analysed in November, cercospora had had no significant impact on yield, sugar concentration or juice impurities (Fig. 3).

2009 – Cercospora symptoms did develop from the early inoculations when temperatures reached almost 30^{o}C in late June/early July (Fig. 4). The symptoms were particularly noticeable in September from plots inoculated in June and a small yield decrease (primarily sugar reduction) was recorded when the trial was harvested and analysed in December (Fig. 5).

2010 – The disease only really infected and spread between plants following inoculation at the end of June (Fig 6). In autumn assessments, little disease was present in plots following July or August inoculations and, following harvesting in November, there was no significant impact on yield (Fig 7).

Figure 1. Symptoms of cercospora leaf spot, September 2009

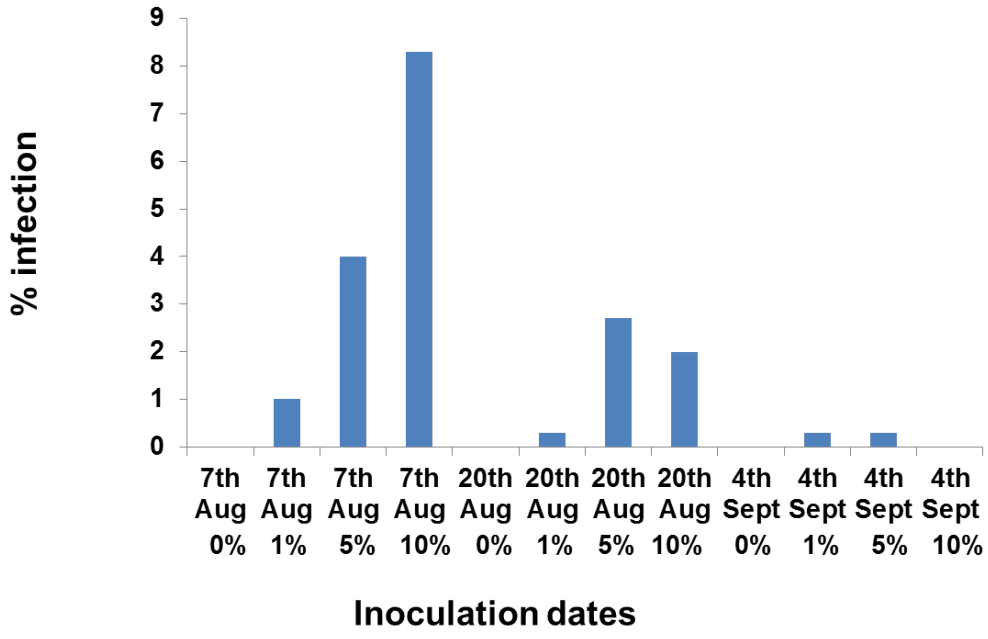

Figure 2. Incidence of cercospora leaf spot in inoculated plots, September 2008

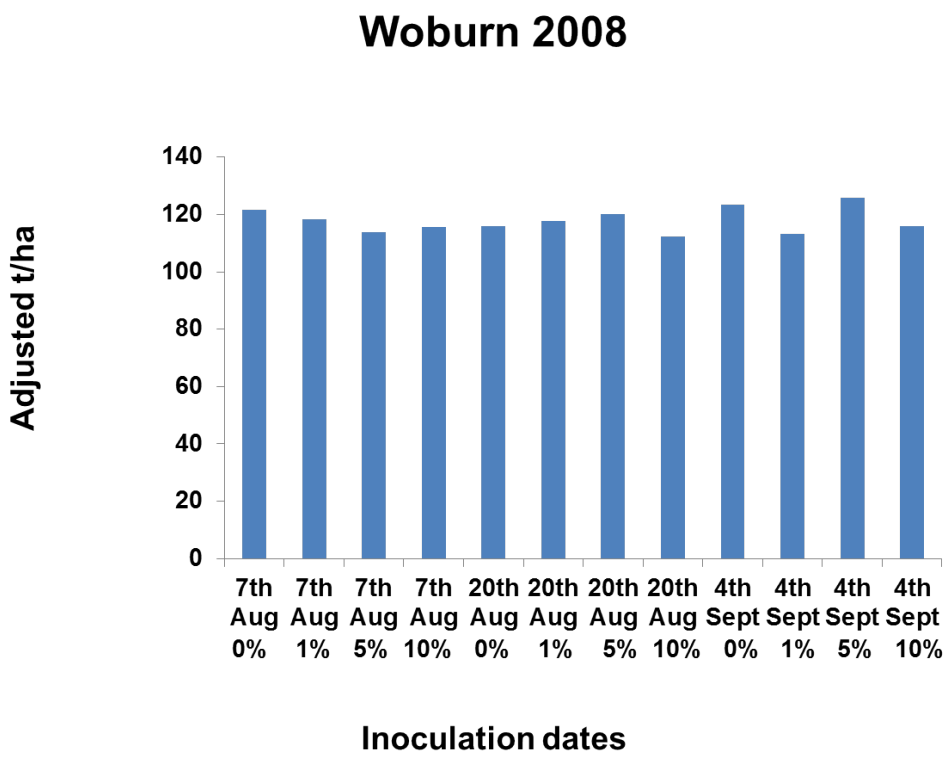

Fig. 3. Impact of cercospora infection on adjusted root yield 2008 (LSD = 9.01)

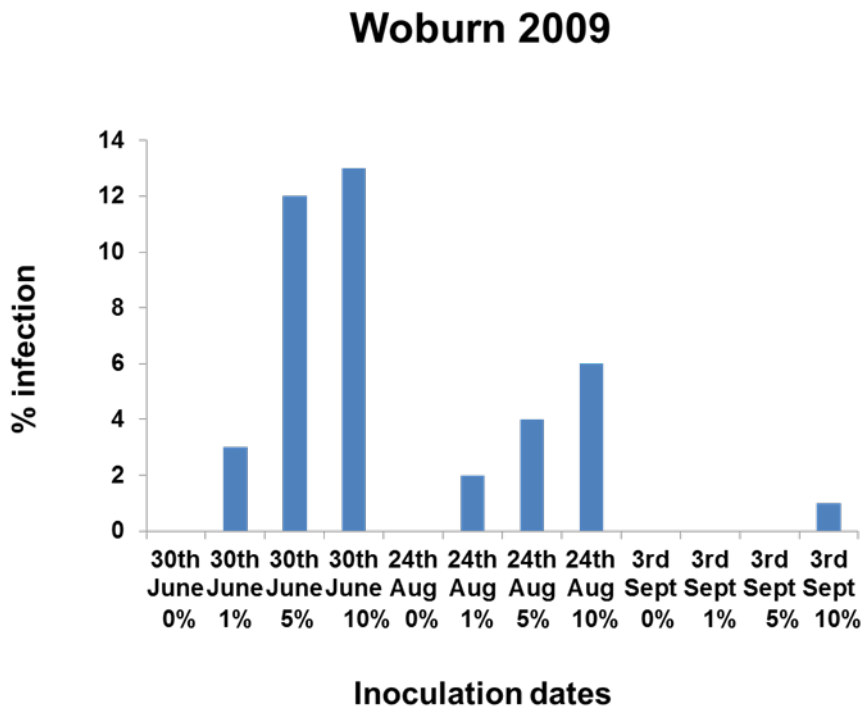

Fig. 4. Incidence of cercospora leaf spot in inoculated plots, September 2009

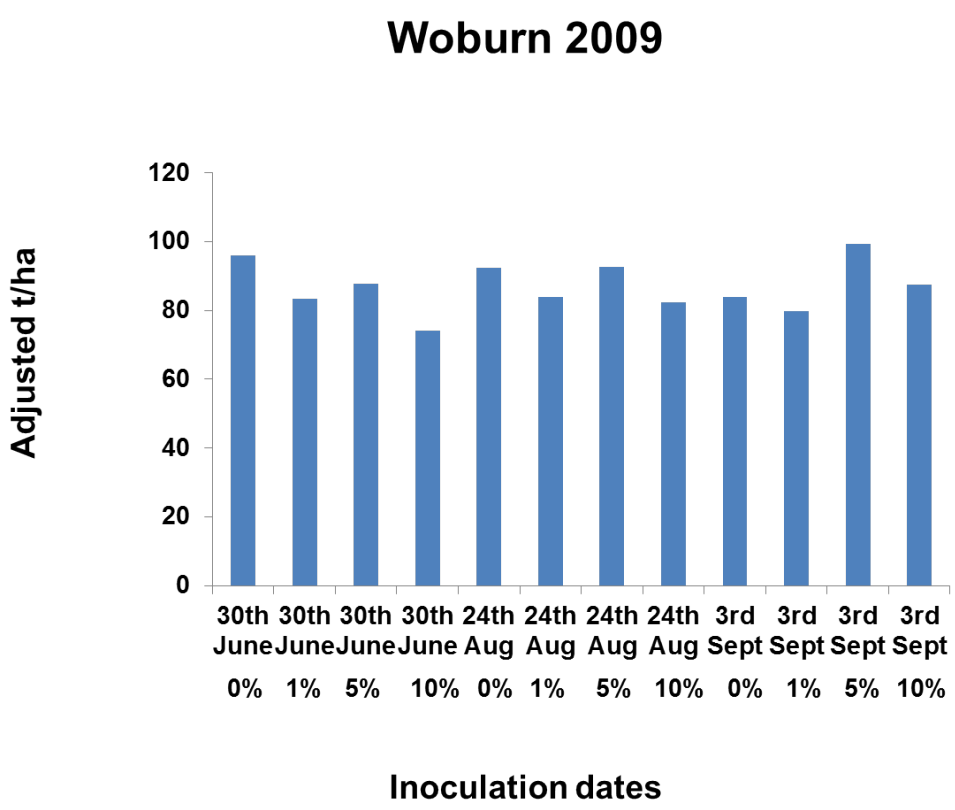

Fig. 5. Impact of cercospora infection on adjusted root yield 2009 (LSD = 25.84)

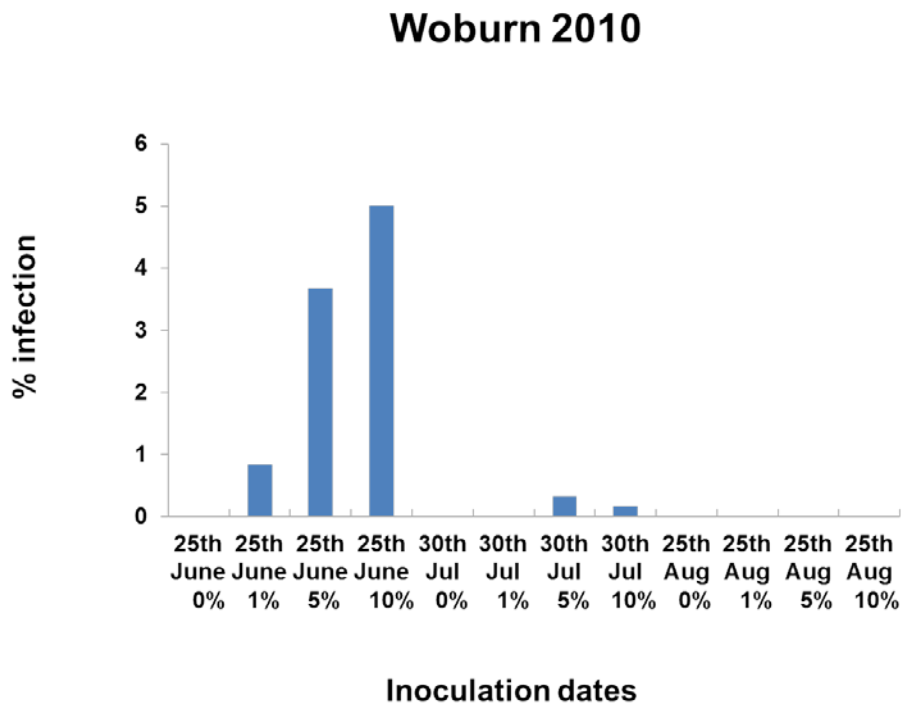

Fig. 6. Incidence of cercospora leaf spot in inoculated plots, September 2010

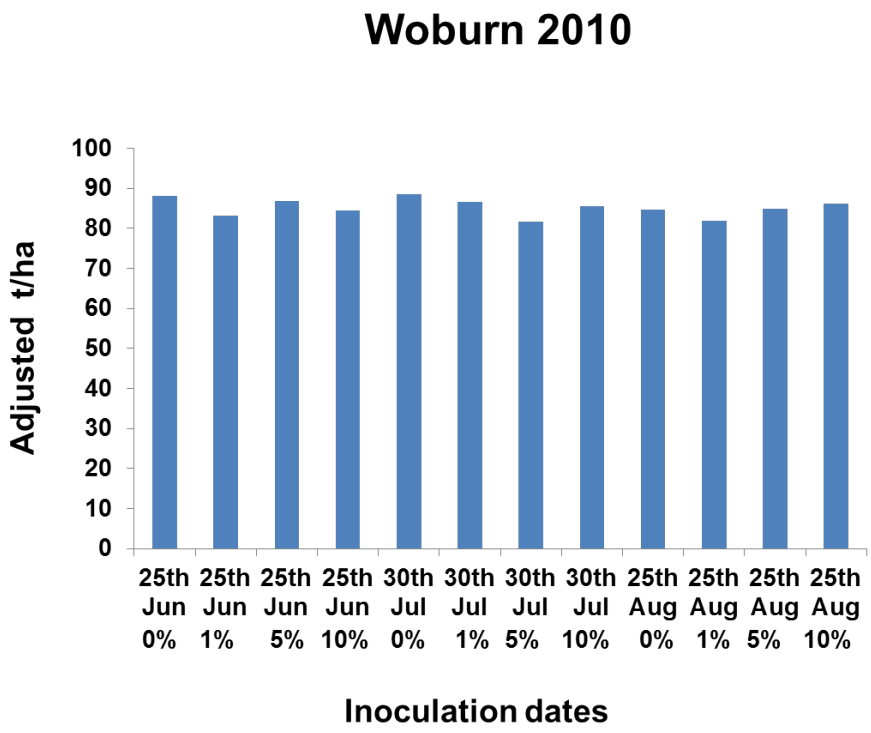

Fig. 7. Impact of cercospora infection on adjusted root yield 2010 (LSD = 7.62)

Survey data

2008

The weather throughout August ensured that conditions were not favourable for disease development. Consequently, only a very limited number of reports were received, primarily in East Anglia during September and October.

2009

The first field reports were not received until September, and in all cases only small patches (6–12 plants) were identified.

2010

The first sugar beet foliar disease to make an appearance in 2010 was cercospora leaf spot. The warm early summer conditions obviously favoured its development. However, in the commercial crop most cases of cercospora were reported in September, particularly in Norfolk, where small patches in fields could be found.

2011

Little cercospora leaf spot was recorded in 2011, although it did make an appearance in a small number of fields in September onwards that had received no fungicide treatments or only a single application. Such late sporadic infection would not have had an impact on yield, but UK isolates have been collected and sent to the USA (Dr Gary Secor and his team at North Dakota State

University) to determine their sensitivity to different active ingredients. We continue to await the findings of these studies.

Conclusions

Cercospora leaf spot was identified in UK sugar beet crops throughout the period of this study. However, symptoms were often confined to single plants or small patches within field and would not have had a significant impact on commercial yield. Fungicide applications would have also limited the impact of this disease. High temperatures and humidity play an important role in the epidemiology of this disease and throughout the period of this study these conditions did not predominate. Consequently, the yield trials, although successfully infected with the disease, did not demonstrate any significant yield penalties.

Therefore, cercospora leaf spot should continue to be regarded as a threat to the UK sugar beet industry, but will only have a significant impact on yield performance if climate enables the disease to establish and spread. None of the current sugar beet varieties are resistant to this disease at present, although the fungicides used to control powdery mildew and rust generally are effective against cercospora too.

Cercospora leaf spot will continue to monitored and future climate change predictions would lead to this disease becoming a greater issue in the future in this country. The sensitivity of isolates to fungicides will also be important data to gain baseline information for future control strategies and appropriate choice of fungicide active ingredients.