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# Geographic variation in severity of phoma stem canker and *Leptosphaeria maculans/ L. biglobosa* populations on UK winter oilseed rape (*Brassica napus*)

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**Abstract** Phoma stem canker, caused by *Leptosphaeria maculans* and *L. biglobosa*, is the most important disease of oilseed rape in Europe. Differences between *L. maculans* and *L. biglobosa* in their life-cycles enable the two species to co-exist on oilseed rape crops over a cropping season. This review considers the factors affecting geographic variation in the severity of phoma stem canker epidemics and in the structure of the population of the pathogens in the UK, where the most severe epidemics occur in the south of England and cankers do not develop in Scotland. It is concluded that this variation is directly related to differences in climate, since weather-based models show that stem canker severity increases with increasing winter/spring temperature and temperatures are greater in the south of the UK. It may also be related to differences in pathogen populations, since surveys showed that the proportion of the more damaging *L. maculans* in stem cankers was greatest in southern England, with most *L. biglobosa* in northern England. Regional variation in agronomic practices such as cultivar choice and fungicide use may also indirectly influence phoma stem canker severity. Differences in cultivar choice result in differences in *L. maculans* race structure, which may influence the severity of epidemics. Differences in fungicide use may also influence pathogen populations, since *L. maculans* and *L. biglobosa* differ in their sensitivities to different azole fungicides. These factors are discussed in relation to strategies for sustainable production of oilseed rape by adaptation to threats posed by climate change.

**Keywords** Adaptation to climate change · Coexisting pathogen species · Host-pathogen-environment interactions · Integrated disease management · Invasive species · Sustainable agriculture

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## Introduction

Phoma stem canker is the most economically important disease of winter oilseed rape in Europe, causing annual losses exceeding €300 M at a price of €300 $t^{-1}$ (Fitt et al. 2006a). The disease is caused by two related pathogen species, namely *Leptosphaeria maculans* and *L. biglobosa* (anamorph *Phoma lingam*). In the UK, there are regional differences in the extent to which winter oilseed rape crops are damaged by phoma stem canker; there was a greater incidence (Fig. 1; www.cropmonitor.co.uk) and severity (Evans et al. 2008) of the disease in southern England than in northern England on commercial winter oilseed rape crops that had received fungicide sprays. Severity is positively related to incidence of phoma stem canker (Zhou et al. 1999). Further north, in Scotland,

although phoma leaf spotting occurs on oilseed rape crops, phoma stem cankers do not develop because winter/spring temperatures are lower than in England (Evans et al. 2008). It is not clear whether regional variation in the severity of phoma stem canker epidemics is due wholly to differences in weather factors (especially temperature and rainfall) or to differences in the pathogen population structure and agronomic factors.

This review will discuss the factors that influence severity of phoma stem canker epidemics on UK winter oilseed rape, including the distribution of the two pathogens associated with the disease. Whilst the detailed data reviewed will be from the UK, mostly from England where the disease is most severe, they will be discussed in an international context, since phoma stem canker is a disease of worldwide importance. Firstly, the review will consider the life-cycles of *L. maculans* and *L. biglobosa*, which coexist on oilseed rape in the UK, Europe and North America. Then it will discuss the influence of geographic variation in weather, pathogen populations and agronomic factors on the regional variation in severity of epidemics.

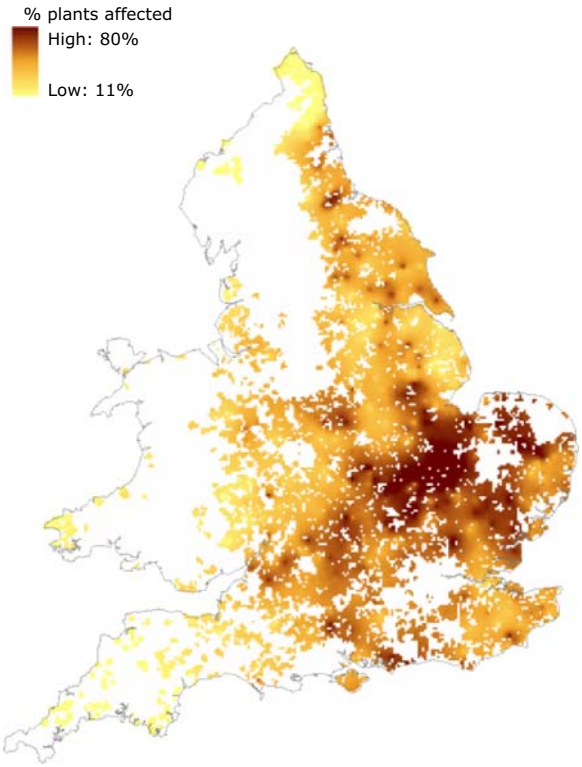


**Fig. 1** Regional variation in incidence (% plants affected) of phoma stem canker on commercial winter oilseed rape crops (several different cultivars) that had generally received fungicide sprays, 1997–2006. The map was generated by interpolating survey data, collected in Defra winter oilseed rape disease surveys of England and Wales, by kriging interpolation. To restrict the map to areas where winter oilseed rape is grown, data were plotted onto a grid mask of the winter oilseed cropping area in England and Wales (www.cropmonitor.co.uk). This shows that winter oilseed rape is mostly grown in the eastern half of the country. Stem cankers are not currently observed on winter oilseed rape crops in Scotland. As there is a positive relationship between the incidence and severity of phoma stem canker in the UK (Zhou et al. 1999), this map also indicates regional differences in the severity of phoma stem canker epidemics. Used with permission

## Phoma stem canker; two pathogens and two diseases?

Of the two pathogens associated with phoma stem canker, *L. maculans* is generally regarded as more damaging since it is particularly associated with the severe cankers that occur at the stem base and result in crop lodging, early senescence and considerable yield loss (West et al. 2001, 2002b; Fitt et al. 2006a). By contrast, *L. biglobosa* is generally considered less damaging since it is especially associated with lesions on the upper stem confined to the outer cortex. Both *L. maculans* and *L. biglobosa* have a worldwide distribution (Fitt et al. 2006a) and *L. biglobosa* has six different subclades (Vincenot et al. 2008). These pathogens can spread on seed of *Brassica napus, B. oleracea*, *B. rapa* and other brassicas. A mixed population of *L. maculans* and *L. biglobosa* occurs in several European countries, including France (West et al. 2002b), Germany (Kuswinanti et al. 1999), Scandinavia (Kuusk et al. 2002) and the UK (Fitt et al. 2006a). There is evidence suggesting that *L. maculans* is spreading from western to eastern Europe. In Poland, where the population was almost entirely *L. biglobosa* until the mid-1990s, *L. maculans* had become widespread on oilseed rape in western regions by 2002 (Karolewski et al. 2002). The global spread of *L. maculans* poses a threat to oilseed rape production in China (Fitt et al. 2008) which has the largest area of oilseed brassicas worldwide. Infections by *L. biglobosa* are considered to be less damaging than those by *L. maculans*. However, in Poland *L. biglobosa* is known to cause considerable yield losses. Investigations with cultures

of *L. biglobosa* isolates from Poland, Canada, France and Germany (Pedras and Biesenthal 2000; Pedras et al. 2007) suggested that Polish isolates may be more pathogenic because they produce polanrazines and phomapyrones, whereas the other *L. biglobosa* isolates do not.

However, *L. maculans* and *L. biglobosa* have similar life-cycles on winter (autumn-sown) oilseed rape in Europe (Fig. 2). Ascospores released in autumn from mature pseudothecia on crop debris from the previous season serve as the main inoculum initiating phoma stem canker epidemics each growing season (West et al. 2001; Huang et al. 2007). Germ-tubes from ascospores penetrate the leaf through stomata and wounds (Huang et al. 2003b). The first symptoms of infection are phoma leaf lesions in the autumn; *L. maculans* produces pale grey lesions with many pycnidia whilst lesions produced by *L. biglobosa* are smaller, have a dark margin with a light brown centre and contain few, if any, pycnidia (Brun et al. 1997; Biddulph et al. 1999; Toscano-Underwood et al. 2001). Both *L. maculans* and *L. biglobosa* spread asymptomatically from the leaf lamina along tissues of the petiole to the stem (Eckert et al. 2005; Fitt et al. 2006b; Huang et al. 2006). In the stem, the pathogens invade and eventually kill host cell tissue, resulting in the formation of phoma stem cankers or upper stem lesions (West et al. 2001; Fitt et al. 2006a). The more damaging *Leptosphaeria maculans* is the predominant species in basal stem cankers and colonises the stem cortex, wood and pith tissues whilst the less damaging *L. biglobosa* is mainly confined to the stem cortex (West et al. 2002b; Van de Wouw et al. 2008). In controlled environment experiments, using an *L. maculans* isolate expressing green fluorescent protein (GFP), Sprague et al. (2007) showed that *L. maculans*

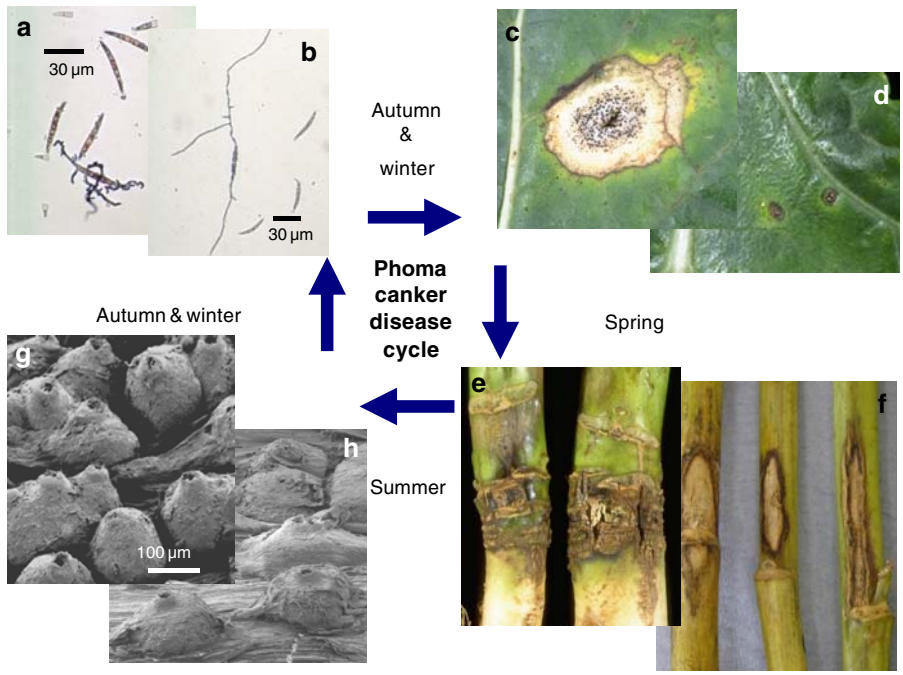


**Fig. 2** Life-cycles of *Leptosphaeria maculans* and *L. biglobosa* on winter oilseed rape in the UK; ascospores of *L. maculans* and *L. biglobosa*, released mainly during autumn and winter after rain and dispersed by wind, germinate (ascospores were stained with trypan blue, hyphae from *L. maculans* ascospores grow tortuously (**a**); hyphae from *L. biglobosa* ascospores are less-branched (**b**)) in wetness or high humidity on leaves and penetrate via stomata or wounds; leaf infections appear as phoma leaf spots during the autumn and winter (large, pale, *L. maculans* (**c**); small, dark, *L. biglobosa* (**d**))—both pathogens grow in vascular tissue from the leaf to the stem; visible stem cankers appear in the spring—stem-base (crown) cankers (**e**, produced from rosette leaf infections) are mostly caused by *L. maculans*, upper stem lesions (**f**) are produced by both species—symptom development and impact on yield differs with most yield loss caused by *L. maculans*; pathogens survive over summer on stem debris, reproducing both asexually (by pycnidia) and sexually by ascomata, which require moisture to mature. *Leptosphaeria maculans* ascomata (**g**) mature and release ascospores earlier than those of *L. biglobosa* (**h**)

is able to infect *B. napus* roots by growing within xylem vessels from the stem into the roots (at the onset of flowering). In addition, they found that *L. maculans* can penetrate the roots through sites of lateral root emergence, when inoculum is applied directly onto them. In the UK, both *L. maculans* and *L. biglobosa* have been isolated from upper stem lesions; however, *L. biglobosa* is more frequently associated with upper stem lesions than with basal stem cankers (West et al. 2002b).

The differences between *L. maculans* and *L. biglobosa* in different stages of their life-cycles enable the two species to co-exist on winter oilseed rape in the UK (Fig. 2). Results of both controlled environment and field experiments provide evidence that there are biological and epidemiological differences between *L. maculans* and *L. biglobosa* (Table 1), which enable the two species to occupy slightly different ecological niches so that they can co-exist on winter oilseed rape over cropping seasons. Differences between *L. biglobosa* and *L. maculans* in temperature optima for pseudothecial maturation lead to differences in time of initiation of epidemics by ascospore release (Toscano-Underwood et al. 2003; Huang et al. 2005), with most *L. maculans* ascospores released early in the cropping season (in autumn/winter), whereas most *L. biglobosa* ascospores are released later (in winter/spring). The differences between *L. biglobosa* and *L. maculans* in timing of ascospore release at the start of the cropping season in autumn result in differences in positioning of lesions on stems at the end of the season before harvest, with the more damaging stem-base cankers associated with *L. maculans* and less damaging upper stem lesions associated with *L. biglobosa* (Table 2).

Studies on the comparative survival of *L. maculans* and *L. biglobosa* on stem debris over the intercrop period in summer demonstrate that *L. maculans* survives longer than *L. biglobosa* on buried debris (Huang et al. 2003b). Both *L. maculans* and *L. biglobosa* survive longer on unburied debris than on buried debris and *L. biglobosa* becomes increasingly predominant on unburied debris over a period of one year. In Australia, *L. maculans* survives for 10–12 months on buried debris (Naseri et al. 2008); most of the *L. maculans* DNA was detected in the upper 5 cm of the soil 1 year after oilseed rape had been grown, with less detected 2 years and negligible amounts 3 or more years after cropping (Sosnowski et al. 2006).

Since pseudothecia of both *L. maculans* and *L. biglobosa* are produced on stem debris, factors affecting their survival on debris during the intercrop period affect the ratio of inoculum of the two species at the start of the new cropping season. There is evidence that the proportions of the two species vary both within and between UK cropping seasons (Table 2). In the autumn, the majority (70–90%) of phoma leaf spots are caused by *L. maculans*, with the proportion of phoma leaf spots caused by *L. biglobosa* slightly increasing by spring but differring between cropping seasons. Before harvest, *L. maculans* is mainly associated with stem-base cankers while *L. biglobosa* is mainly associated with upper stem lesions; these differences are maintained during the production of ascopores 3 months after harvest (Table 2). There is a need to understand how populations of *L. maculans* and *L. biglobosa* vary geographically in the UK in relation to geographical variation in the severity of phoma stem canker epidemics.

## Variation in weather

One factor that clearly influences the geographical variation in severity of phoma stem canker is the weather. There is considerable geographical variation in temperature and rainfall; in the south, temperatures are higher and rainfall is lower than in the north (Fig. 3). Temperature and rainfall affect the date of onset of ascospore release and subsequent timing of phoma leaf spotting in autumn (Fig. 4; Huang et al. 2007). The effect of regional variation in summer temperature and rainfall (taken from harvest in July until sowing in late August/early September) on the date of onset of phoma leaf spotting has been demonstrated using a weather-based disease forecasting model (Evans et al. 2008). The model predicted an earlier onset of symptoms in southern England than in northern England (Fig. 5; www.rothamsted.bbsrc.ac.uk/ppi/phoma/). As the endophytic growth of *L. maculans* and *L. biglobosa* along the petiole to the plant stem is a function of accumulated temperature expressed as degree-days (Sun et al. 2001; Evans et al. 2008), regional variation in the onset of phoma leaf spot in the autumn results in

**Table 1** Differences between *Leptosphaeria maculans* and *L. biglobosa* in their life-cycles on winter oilseed rape, enabling them to coexist over a crop/intercrop season in Europe*

| Experiment | Parameter | *L. maculans* | *L. biglobosa* | Reference |
|---|---|---|---|---|
| Pseudothecial maturation | Maturation | Earlier at 5–10°C (same at 15–20°C) | Later at 5–10°C | a |
| | Position on debris | On surface at stem base | Under surface of upper stem (neck exposed) | |
| Ascospore release | Period of release | Early autumn/winter | Late autumn/winter/spring | b |
| Ascospore germination | Start of germination | Later at 5–20°C | Earlier at 5–20°C | c |
| | Germ tube length | Shorter | Longer | |
| | Hyphal growth | Curved | Almost linear | |
| Penetration of leaf tissues | Mode of entry | Through stomata | Through stomata | d |
| Symptoms | Leaf lesion | Large beige lesions with many pycnidia | Small dark lesions with few/no pycnidia | e |
| | Growth along petiole | Slower (0.7 mm $day^{-1}$ at 15°C) | Faster (1 mm $day^{-1}$ at 15°C) | |
| | Position of lesions on stem | Mainly stem base | Mainly upper stem | |
| | Internal growth in stem tissues | Extensively colonises all stem base (cortex, pith) and tap root tissues | Mostly restricted to cortex of stem base, occurs in pith in upper stems | |
| Survival | Survival on buried debris | Survives longer (1 year) on stem bases | Survives <2 months on upper stems | f |
| | Survival on unburied debris | Survives 1 year | Survives 1 year (becomes predominant) | |

*Modified and extended from Table 2 in Fitt et al. (2006b)

[a] Toscano-Underwood et al. (2003).

[b] Huang et al. (2005); West et al. (2002a).

[c] Huang et al. (2003b); Huang et al. (2001).

[d] Huang et al. (2003b); Hammond et al. (1985).

[e] Fitt et al. (2006b); Toscano-Underwood et al. (2003); West et al. (2002b); Brun et al. (1997)

[f] Huang et al. (2003a); West et al. (2002a).

regional variation in the severity of phoma stem canker epidemics. However, the most important effect of temperature on canker severity relates to the thermal time accumulated during winter and early spring, while the pathogens grow asymptomatically in the stem tissues (Evans et al. 2008). Thus, differences in winter temperature may be the main reason why cankers appear earlier in spring in the south than the north of England and currently not at all in Scotland, despite the presence of leaf spot lesions there. The earlier appearance of cankers combined with warmer weather in spring while cankers are developing explains why cankers are more severe in the south.

There is also evidence that temperature affects operation of resistance against *L. maculans,* both in leaves (race-specific genes such as *Rlm6,* Huang et al. 2006) and in stem tissues (quantitative resistance, Huang et al. 2009), so that the resistance is rendered ineffective as temperature increases. For example, results of both controlled environment experiments and field experiments in France have shown that temperature also affects stem canker severity. In the field experiments, stem canker severity was less severe on cultivars with quantitative resistance than on a cultivar without quantitative resistance in the season when temperatures were lower. However, in the season when temperatures were greater, there was no difference between the cultivars with or without quantitative resistance. These results suggest that less effective host resistance at higher temperatures may contribute to the greater severity of stem canker in southern England.

**Table 2** Seasonal co-existence of *Leptosphaeria maculans* and *L. biglobosa* on winter oilseed rape crops at the phoma leaf spot and phoma stem base canker/ upper stem lesion stages during the cropping season and on stem debris in the intercrop period, for five cropping seasons at Rothamsted, UK

| | | | Proportion (%) of *Lm* or *Lb* in population | | | | | |
|---|---|---|---|---|---|---|---|---|
| | | | 1999/2000 | 2000/2001 | 2001/2002 | 2002/2003 | 2003/2004 | Average |
| Leaf lesion[a] | Autumn | *Lm* | 69.2 | 72.7 | 54.9 | 72.5 | 81.7 | 70.9 |
| | | *Lb* | 30.8 | 27.3 | 45.1 | 19.5 | 18.3 | 29.1 |
| | Spring | *Lm* | 72.9 | 71.8 | 49.2 | 83.3 | 72.2 | 69.9 |
| | | *Lb* | 27.1 | 28.2 | 50.8 | 16.7 | 27.8 | 30.1 |
| Stem before harvest[b] | Stem base | *Lm* | –[d] | 78.5 | 77.3 | 58.5 | 92.5 | 76.7 |
| | | *Lb* | – | 21.5 | 22.7 | 41.5 | 7.5 | 23.3 |
| | Upper stem | *Lm* | – | 58.9 | 25.9 | 35.7 | 83.3 | 50.9 |
| | | *Lb* | – | 41.1 | 74.1 | 64.3 | 16.7 | 49.1 |
| Debris after harvest[c] | Autumn | *Lm* | 79.3 | 77.6 | 59 | – | – | 72.0 |
| | | *Lb* | 20.7 | 22.4 | 41 | – | – | 28.0 |
| | Spring | *Lm* | 64.5 | 52.7 | 46.8 | – | – | 54.7 |
| | | *Lb* | 35.5 | 47.3 | 53.2 | – | – | 45.3 |

[a] Data for percentage of lesions caused by *L. maculans* (*Lm*) or *L .biglobosa* (*Lb*) were obtained by assessment of lesion appearance on leaves of 30 plants (20 plants in 1999/2000) and confirmed by isolation. Plants did not receive any fungicide spray. Data for 2002/2003 and 2003/2004 were from Eckert (2005).

[b] Data for percentage of stem-base canker or upper stem lesions caused by *L. maculans* or *L. biglobosa* were obtained by isolation.

[c] Data for percentage of *L. maculans* and *L. biglobosa* ascospores released from stem debris were obtained by single ascospore culture sub-sampled from ascospores ejected from pseudothecia on stem debris.

[d] No data collected.

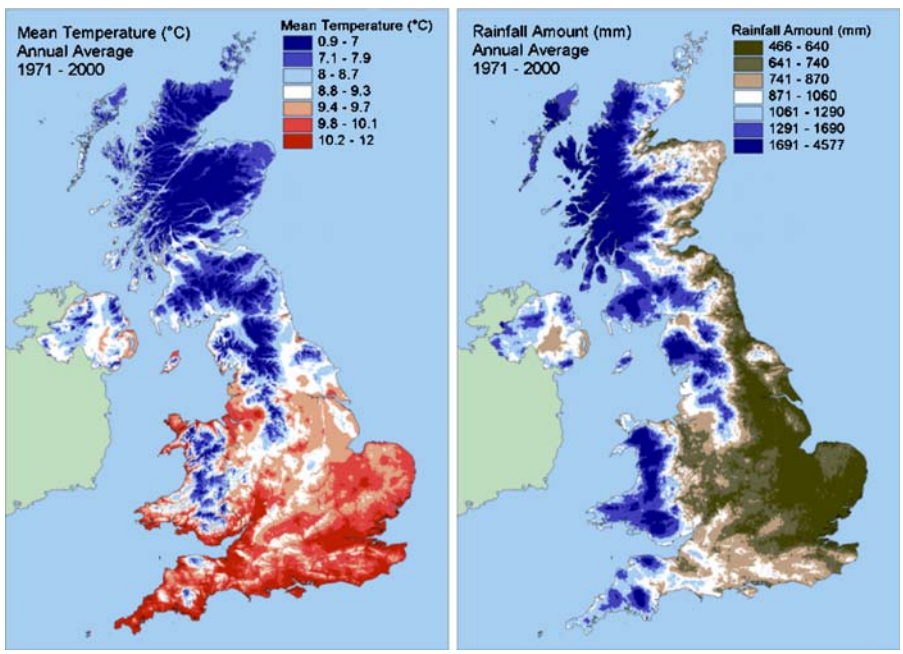


**Fig. 3** Mean annual temperature (°C) and mean annual rainfall (mm) for the UK for the period 1971–2000. © Crown copyright 2000, the Meteorological Office. Used with permission

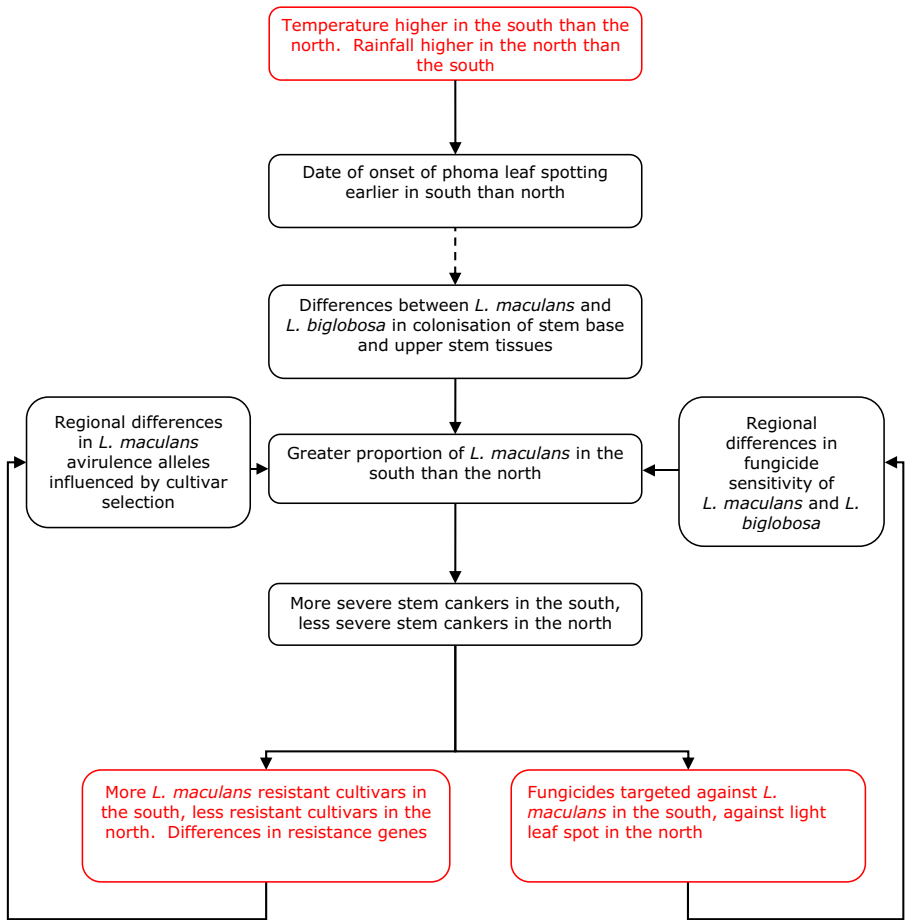


**Fig. 4** Relationships between weather (e.g. temperature, rainfall) and agronomic (e.g. cultivar choice, fungicide use) factors (shown in red) affecting differences in populations of *L. maculans* and *L. biglobosa* and differences in the severity of phoma stem canker epidemics between the north and south of the UK (in black). This illustrates the direct effects of differences in weather (especially temperature) on both populations of the pathogens and severity of epidemics, and the indirect effects of agronomic factors such as cultivar choice and fungicide use on severity of epidemics through their effects on pathogen populations. There is a dashed line between boxes starting 'date of onset' and 'differences between' because there is no causal relationship implied; both contribute to the differences in proportions of the two species between north and south

In European countries such as France, phoma stem canker epidemics are more severe than in England; this may be due to the warmer temperatures in continental Europe (Fitt et al. 2006a). Weather factors also affect severity of phoma stem canker epidemics in Australia where oilseed rape is grown over the winter period. Several weather factors favour severe stem canker epidemics in Western Australia (Howlett et al. 2001; West et al. 2001). Hot summer temperatures favour the survival of *L. maculans* in crop debris on the soil surface during the inter-crop period. The emergence of seedlings and ascospore release both coincide with the first periods of rain in the autumn. After emergence, strong winds wound seedlings and in areas with sandy soils sand blasting can occur, making plants more susceptible to infection. Mild winter temperatures favour the development of severe cankers. In addition, very damaging stem cankers are observed in areas with medium or low rainfall, where drought stress occurs at the end of the growing season. The severity of canker epidemics in France and Australia suggests that epidemics will increase in severity in southern England under predicted climate change, with warmer winters and dryer summers (Evans et al. 2008).

## Variation in *L. maculans/L. biglobosa* populations

Another factor that may affect the geographical variation in severity of phoma stem canker epidemics is variation in populations of the pathogens. The occurrence of *L. maculans* and *L. biglobosa* was surveyed in England in 2001–2003 (Liu 2007) and 2006 (Stonard 2008), using methods based on plate cultures and/or quantitative PCR. Results from visual assessment of cultures and end-point PCR reported by Liu (2007) suggested that there is regional variation

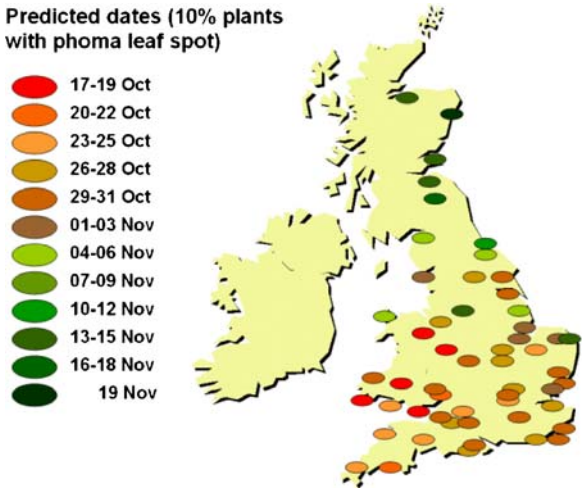


**Fig. 5** Regional variation in date in autumn when incidence of phoma leaf spot reaches 10% plants affected. The predicted dates when incidence of phoma leaf spot reaches 10% are given for the 2006/2007 winter oilseed rape growing season. The predictions were obtained with a regression model (Evans et al. 2008) and are based on the weather (temperature/rainfall) that affects the maturation of *L. maculans* ascospores in crop debris and infection of leaves of the new oilseed rape crop over the period July to September 2006. These dates are predicted each season and are available online (www.rothamsted.bbsrc.ac.uk/ppi/phoma/)

in the distribution of *L. maculans* and *L. biglobosa* in England, with a greater incidence of *L. biglobosa* in the north (Fig. 4). Quantitative PCR was used to determine the amounts of DNA of *L. maculans* and/or *L. biglobosa* in the phoma stem cankers assessed. When the results of these studies were geostatistically mapped, the proportion of *L. maculans* DNA in phoma stem cankers was greatest in southern England whilst that of *L. biglobosa* was greatest in northern England (Stonard 2008; Stonard et al. 2009). The observed pattern of regional variation matched patterns of predictions, made using a weather-based disease forecasting model, of the time between onset of phoma leaf spot in autumn (predicted using temperature and rainfall data from the harvest of the previous crop to sowing of the new crop) and onset of phoma stem canker in spring (predicted as the date when a threshold period, expressed in degree-days, had elapsed after the onset of phoma leaf spotting was reached) (Evans et al. 2008). This suggests that temperature may affect the proportions of *L. maculans* and *L. biglobosa* in basal stem cankers, with a greater proportion of *L. maculans* associated with warmer temperatures.

These regional differences in the proportions of the two species may contribute to the regional differences in the severity of epidemics, since *L. maculans* is the pathogen which causes severe phoma stem canker epidemics. The survey of proportions of DNA from *L. maculans* or *L. biglobosa* in total DNA extracted from phoma stem cankers showed that proportions of *L. maculans* were greatest in southern England (Fig. 4; Liu 2007; Stonard 2008) where phoma stem canker epidemics are most severe (Fig. 1). This association is supported by evidence from Canada, where *L. biglobosa* was once widespread, and the westward spread of *L. maculans* from Saskatchewan was accompanied by an increase in severity of phoma stem canker epidemics (West et al. 2001; Kutcher et al. 2007; Fitt et al. 2008). Therefore, regional variation in the proportions of *L. maculans* and *L. biglobosa* can influence the severity of phoma stem canker epidemics.

## Variation in agronomy

Differences between regions in agronomic practices may also contribute to the variation in severity of phoma stem canker epidemics. For example, winter oilseed rape is cultivated most intensively in the east of England and Scotland (www.hgca.com) because the terrain and soil type are often unsuitable in the west. Evidence that severity of phoma stem canker epidemics is affected by the intensity of oilseed rape cropping, obtained by modelling spread from affected crops arranged in different cropping intensities (Aubertot et al. 2006), is provided by the low severity of phoma stem canker epidemics in south-west England where there are few oilseed rape crops (Fig. 1) even though climatic conditions in this area (Fig. 3) are conducive to severe epidemics. The most severe phoma stem canker epidemics occur in south-east England (Fig. 1; www.cropmonitor.co.uk), where oilseed rape cropping is intense. In northern England and in Scotland, where the incidence of phoma stem canker is low, there is a high incidence of light leaf spot caused by *Pyrenopeziza brassicae* (anamorph *Cylindrosporium concentricum*) necessitating use of different cultivars and different fungicides for disease control (Fitt et al. 1998; Boys et al. 2007). Therefore, cultivars with a good phoma stem canker resistance rating are selected in southern England whilst those

with a good light leaf spot resistance rating are selected in northern England. For example cv. Cobra (light leaf spot resistance rating of 7 and phoma stem canker resistance rating of 4 (www.hgca.com)) was a popular choice only in northern England (S. Elcock, pers. comm.).

Since cultivar choice is likely to affect the proportions of *L. maculans* and *L. biglobosa* in a population because cultivars resistant to *L. maculans* may be susceptible to *L. biglobosa* and *vice-versa* (Fitt et al. 2006a), cultivar choice may also indirectly influence the severity of epidemics. However, there is little information about variation in cultivar resistance to *L. biglobosa* (Fig. 4). Nevertheless, there is much evidence that regional variation in cultivar choice affects the race structure of *L. maculans* populations. Race-specific (qualitative) genes for resistance to *L. maculans* are bred into different oilseed rape cultivars for control of phoma stem canker. To date, 13 *B. napus* race-specific genes for resistance to *L. maculans* (*Rlm1*-9, *RlmS* and *LepR1-3*) and 10 corresponding *L. maculans* avirulence alleles (*AvrLm1-9* and *AvrLmS*) have been identified (Ansan-Melayah et al. 1995; Balesdent et al. 2001, 2002, 2005, 2006; Delourme et al. 2004, 2006; Yu et al. 2005, 2008; Van de Wouw et al. 2009). *Leptosphaeria maculans* has a high evolutionary potential, according to the criteria of McDonald and Linde (2002), due to its possession of both sexual and asexual cycles, effective wind-dispersal of ascospores and rain-splashed conidia and its large population size. Therefore, regional variation in cultivar choice results in variation in *L. maculans* race structure (Fig. 4). For example, the large-scale cropping of oilseed rape with the *Rlm1* gene in France, with a rapid increase in the area of *Rlm1* cultivars from 1996 to 1999 (44% of total area grown), resulted in directional selection for the virulent allele *avrLm1* in the pathogen population, thereby rendering *Rlm1*-mediated resistance ineffective by the 2000/2001 cropping season (Rouxel et al. 2003).

Surveys have been done to examine regional variation in Europe in *L. maculans* race structure as assessed by disease phenotypes on wounded brassica seedlings from a differential set of brassica lines with alleles *AvrLm1-AvrLm9*. In a 2006 survey of five oilseed rape-growing counties of England, genetic distance was lowest among isolates from sites that were geographically the closest (Stonard 2008), supporting the theory that *L. maculans* race structure is affected by the history of oilseed rape cropping and cultivar choice (Balesdent et al. 2005), since both oilseed rape cropping history and cultivar choice in England are likely to be more similar between nearby sites than those far apart. In France, a survey in the autumns of 2000 and 2001 reported variability in the frequency of given *Avr* alleles between 20 sites (Balesdent et al. 2006); the greatest frequency of isolates with the avirulent *AvrLm1* allele was in a region in which oilseed rape was not a major crop, whilst the smallest frequency was in a region in which oilseed rape was intensively cropped. In a Europe-wide survey done in the autumns of 2002 and 2003, different ratios of avirulence alleles were found in six sites selected from Germany, Sweden, England and Poland; however, the allele composition for the three most common races of the *L. maculans* populations was similar in different countries (Stachowiak et al. 2006). There is good evidence that European isolates of *L. biglobosa* all belong to the subclade *L. biglobosa* 'brassicae', although other subclades are found in other parts of the world (Vincenot et al. 2008). There is a need to examine the differential interactions between the subclades of *L. biglobosa* and genotypes of *B. napus*.

Regional variation in choice of fungicides in the UK may also influence populations of the canker pathogens and thus indirectly influence severity of epidemics (Fig. 4). In northern England and Scotland, fungicides effective against light leaf spot, such as tebuconazole or prothioconazole, are used (Boys et al. 2007). In southern England, fungicides such as flusilazole, difenaconazole and metconazole, together with prothioconazole, that are more effective against phoma stem canker are used (Gladders et al. 2006). The proportions of *L. maculans* and *L. biglobosa* in populations may be affected by this regional variation in fungicide application regime since *L. maculans* and *L. biglobosa* differ in their sensitivities to different azole fungicides (Eckert 2005) (Fig. 4). Regional variation in sensitivity to the azole fungicide flusilazole in *L. maculans* populations has been determined; isolates from northern England were found to be more sensitive than those from southern England (Hood et al. 2008). However, yield responses from these applications are achieved only when fungicides are used at the optimal time, since they have limited eradicant activity and their period of protectant

activity is short due to degradation, leaf expansion and production of new, untreated leaves (West et al. 1999, 2001, 2002a; Aubertot et al. 2006; Gladders et al. 2006). Throughout Europe, fungicide sprays are more effective in controlling phoma stem cankers when they are applied in the autumn rather than in the spring (West et al. 2002a; Kruse and Verreet 2005; Steed et al. 2007). The English national oilseed rape disease survey of commercial winter oilseed rape crops from 1990–2006 (www.cropmonitor.co.uk) showed that crops in southern England received a greater number of fungicide applications, especially in autumn, than those in northern England (S. Elcock, pers. comm.). This survey (Fig. 1) thus suggests that the differences in incidence and severity of phoma stem canker between the north and south of England would have been even greater in unsprayed crops, since more sprays with the most effective fungicides were applied to control phoma stem canker to crops in the south of England.

## Conclusions

This review provides good evidence that geographical differences in climate, especially temperature, have a dominant direct influence on the differences between northern and southern UK in the severity of phoma stem canker epidemics and in the proportions of *L. maculans* and *L. biglobosa* in the pathogen populations (Fig. 4). Variation in temperature and rainfall between the harvest of the previous season and the sowing of the new crop results in an earlier onset of phoma leaf spotting in the south than in the north (Fig. 5, Evans et al. 2008). Warmer temperatures in southern England result in an earlier onset of phoma stem cankers in the spring and, therefore, more severe stem cankers at harvest (Evans et al. 2008). Nevertheless, regional variation in the severity of phoma stem canker epidemics is also influenced indirectly by other interactions between the host, pathogen and abiotic factors. For example, the structure of pathogen populations is affected by variation in both climate and agronomic practices such as cultivar choice and fungicide use. To account for such interactions, Integrated Avirulence Management (IAM) has been used in France to enhance durability of specific genes for resistance to *L. maculans* by limiting the selection on *L. maculans* populations and reducing the size of such populations through a combination of cultural, physical, biological and chemical disease control practices (Aubertot et al. 2006). Such strategies could also be used to improve control of phoma stem canker in the UK, whilst minimising use of fungicides. To do this, there is a need to develop models that predict the effects of different combinations of control practices on severity of epidemics and to test these combinations experimentally.

There is a consensus that global warming is occurring and that it is linked to human activity (Hansen et al. 2005). Climate change is likely to have effects on individual plants and plant communities that will affect their interactions with pathogens (Garrett et al. 2006). Of concern is the prediction of an increase in phoma stem canker severity, especially in southern England, and a northwards spread, into Scotland, of phoma stem canker due to increased temperature associated with climate change (Evans et al. 2008). It is likely that this will be accompanied by an increase in the proportion of the more damaging *L. maculans* in pathogen populations. In the face of climate change, for production of oilseed rape in southern England to be sustainable, there is a need for a coordinated strategy, involving both government and industry, to improve control of phoma stem canker. For example, there is a need for improved resistance to *L. maculans* that can operate at increased temperatures and for exploiting the resistance that *L. biglobosa* and chemical defence activators can induce against *L. maculans* (Liu et al. 2006), especially since legislation passed by the European Parliament in January 2009 (Directive 91/414, http://ec.europa.eu/food/plant/protection/index_en.htm) may mean that fewer fungicides are available to control the disease. By understanding the factors affecting the severity of phoma stem canker epidemics, improvements can be made in the control of phoma stem canker as part of a strategy for adaptation to future challenges affecting UK oilseed rape cultivation.

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