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REVIEW ARTICLE

Epidemiology and management of *Leptosphaeria maculans* (phoma stem canker) on oilseed rape in Australia, Canada and Europe

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Phoma stem canker (blackleg), caused by *Leptosphaeria maculans*, is an important disease on oilseed rape (canola, rapeseed, *Brassica napus*, *Brassica juncea*, *Brassica rapa*) causing seedling death, lodging or early senescence in Australia, Canada and Europe, but not in China. The two forms of *L. maculans* (A group and B group) that occur on oilseed rape are now considered to be separate species. The epidemiology and severity of phoma stem canker differs between continents due to differences in the pathogen population structure, oilseed rape species and cultivars grown, climate and agricultural practices. Epidemics are most severe in Australia, where only the A group occurs, and can be damaging in Canada and western Europe, where both A and B groups occur, although their proportions vary within regions and throughout the year. Epidemics are slight in China, where the A group has not been found. Dry climates (Australia, western Canada) lengthen the persistence of infected debris and may synchronize the release of airborne ascospores (after rain) with seedling emergence. *L. maculans* spreads from cotyledon and leaf infections down petioles to reach the stem, with infections on cotyledons and leaves early in the season producing the most damaging stem cankers at the stem base (crown). Development of both crown cankers and phoma stem lesions higher up stems is most rapid in regions with high temperatures from flowering to harvest, such as Australia and Canada. Breeding for resistance (genetic, disease escape or tolerance), stubble management, crop rotation and fungicide seed treatments are important strategies for control of phoma stem canker in all areas. Fungicide spray treatments are justified only in regions such as western Europe where high yields are obtained, and accurate forecasts of epidemic severity are needed to optimize their use.

Keywords: A group and B group, blackleg, canola, crown canker, phoma leaf spot, *Phoma lingam*

Introduction

Leptosphaeria maculans (anamorph *Phoma lingam*) causes phoma stem canker (blackleg) on oilseed rape (canola, rapeseed, *Brassica napus oleifera*, *B. juncea, B. rapa*) in many areas of the world. The disease is of major economic importance in the main oilseed rape growing areas of Australia, Canada and Europe, although these areas have different cultivars, growing seasons, climates and agricultural practices (Table 1). The most severe epidemics occur in Australia, where the disease curtailed development of the emerging oilseed rape industry in the early 1970s (Bokor *et al*., 1975). Severe epidemics have also influenced production of oilseed rape in Canada and Europe (Gugel & Petrie, 1992). However, the disease is rare in Scotland and in the large area of oilseed rape grown in Asia, mostly in India (as *B. juncea* and *B. rapa*) and China (as *B. napus*). Within Australia, Canada or Europe, the severity of epidemics differs greatly between seasons, between regions and between crops. Where the disease occurs, total destruction of the crop due to seedling death is rare and usually yield losses at harvest are $< 10\%$, although they can reach 30–50% (Hall *et al*., 1993; Zhou *et al*., 1999; Barbetti & Khangura, 1999).

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Table 1 Management features of oilseed rape[a] in relation to climate in different regions where severe phoma stem canker occurs

	Australia	Canada	Europe
Oilseed rape type	> 95% *B. napus* (spring-type grown over winter) < 5% *B. juncea*	West: spring *B. napus* (some *B. rapa*) Ontario: spring (some winter) *B. napus*	West: winter *B. napus* East: *B. napus* (60% winter, 40% spring)
Winter cvs			
Sowing	Not grown	Mid-August/early Sept	Late August
Harvest	–	Mid-July	July
Season (days)	–	330	315
Mean temp. (°C)[b]	–	7	7–11
Spring cvs			
Sowing	May/June (winter)	Late April/mid May	April
Harvest	October–December	Mid-August	August
Season (days)	150–200	94–108	120
Mean temp. (°C)[b]	10–16	15–17	13–16
Typical yield (t/ha)	West 1·1 t/ha South-east 1·6 t/ha	West 1·5 t/ha Ontario 2·2 t/ha	West 4·0 t/ha East 1·5 t/ha
Crop management	Four-year rotation and stubble management	Four-year rotation, no tillage is popular	Four-year rotation, deep ploughing of stubble
Cultivar resistance[c]	Most cultivars are 4–7 on 1–9 scale (1, highly susceptible; 9, immune)	Most cultivars are 1–3 on 0–5 scale (0, immune; 5, highly susceptible)	Most UK cultivars 5–7 on 1–9 scale (1, highly susceptible; 9, immune)
Seed treatment	Iprodione (rhizoctonia)	Carbathin, thiram or iprodione	Thiram or iprodione
Other fungicide use	Flutriafol coating to fertilizer granules at sowing	Propiconazole sometimes applied to young plants	Protectant fungicides as foliar sprays[d]
Temperature (°C)[e] min/max	West 6–10/22–30 South-east 4–10/20–28	West −19/20 Ontario −8/22	West 1/21 East −10/25
Rainfall (mm)[f]	West 250–600 South-east 250–1000	West 250–750 Ontario 500–1000	West 200–750 East 250–600

[a]Also known as canola (Australia, Canada) or rapeseed.
[b]Mean of temperatures > 0°C during growing season.
[c]9 is most resistant on Australian and UK (National Institute of Agricultural Botany) scales.
[d]Difenoconazole plus carbendazim or flusilazole plus carbendazim.
[e]Minimum mean and maximum mean daily temperatures.
[f]Annual total rainfall.

L. maculans is able to cause phoma stem canker on different winter and spring cultivars of oilseed rape grown under a wide range of climates and despite different agricultural practices. There is some confusion in the terminology used to describe the different stages of the disease, with the same terms used to describe different symptoms and different terms used to describe the same symptoms on different continents. Nevertheless, there may be more than one disease on oilseed rape, since *L. maculans* has been shown to be highly variable; *L. maculans* has been divided into two groups termed highly virulent and weakly virulent, virulent and avirulent, aggressive and nonaggressive, Tox^{+} and Tox^{0}, or A group and B group. These groups may infect the same host and have similar looking spores but differences occur in culture (Cunningham, 1927), genetics (Taylor *et al*., 1991), metabolite production (Balesdent *et al*., 1992), leaf symptoms (Brun *et al*., 1997; Ansan-Melayah *et al*., 1997; Thürwächter *et al*., 1999) and stem symptoms (Johnson & Lewis, 1994). There is no evidence of sexual matings between A and B groups of *L. maculans* and pseudothecia of the A and B groups differ morphologically (Gabrielson, 1983; Petrie & Lewis, 1985; Somda *et al*., 1997; Farahani & Zinkernagel, 1997). Consequently, *L. maculans* is now considered by many to be a complex of at least two different species (Taylor *et al*., 1991; Williams, 1992; Jedryczka *et al*., 1999a; Williams & Fitt, 1999). The A group has been further divided into different pathogenicity groups (Koch *et al*., 1991; Mengistu *et al*., 1991; Badawy *et al*., 1992; Gall *et al*., 1995; Ansan-Melayah *et al*., 1997; Balesdent *et al*., 1998) and other groups exist, named on the basis of the plant species from which they were isolated. The proportion of A and B groups varies from region to region, with one or other group predominant in some areas and a mixture of the two groups elsewhere. However, there appear to be many common features of the epidemiology of the

disease associated with different pathogen populations in different climates and under different agricultural practices.

The different methods needed to manage the disease in different countries reflect these differences in cultural practices, climate and *L. maculans* pathogen populations, as well as differences in the economics of oilseed rape production. Methods for management of phoma stem canker include growing resistant cultivars, crop rotation, stubble management and the use of fungicides. In regions such as western Europe, high yields (e.g. 4–5 tonnes/ha in England) have traditionally merited the use of fungicide sprays, whereas such sprays cannot be justified economically in climates where yields are lower. This review discusses the differences in the epidemiology and management of *L. maculans* on oilseed rape in different regions of the world, in relation to differences in pathogen populations, cultivars grown, climate and cultural practices.

Epidemiology of *L. maculans* in different continents

Initiation of epidemics

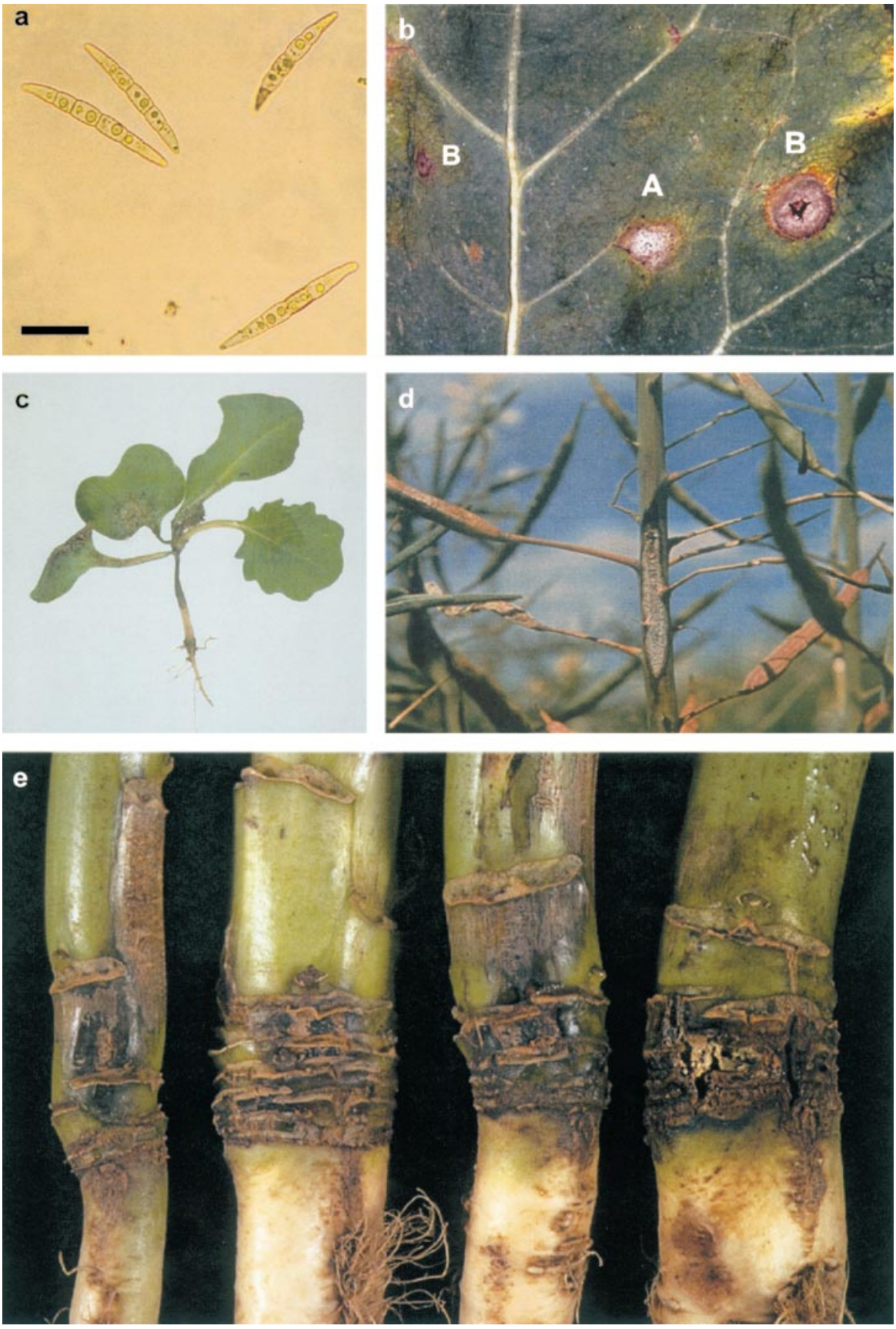
Phoma stem canker is usually a monocyclic disease in Australia, Canada and Europe, with epidemics generally initiated by airborne ascospores (Fig. 1a; McGee, 1974; Bokor *et al*., 1975; Hall, 1992; Mahuku *et al*., 1997). However, infections can also arise from infected seed (Jacobsen & Williams, 1971; Wood & Barbetti, 1977a; Kharbanda & Stevens, 1993), from infected stubble by direct contact and by rain-splashed conidia (Hall, 1992; Thürwächter *et al*., 1999). Alternative cruciferous hosts of *L. maculans* may also be possible sources of inoculum (Hall, 1992). The most common primary inoculum, ascospores, is released over an extended period from pseudothecia formed on woody remains of infected plants (Hall, 1992; Mahuku *et al*., 1997). Ascospore release occurs after wetting by rain (Pérès *et al*., 1997) and even dew (McGee, 1977; Kruger & Wittern, 1985). The period of ascospore release varies from region to region (Table 2) but usually coincides with the presence of young, susceptible plants. In Australia, ascospores are first released from debris in May in response to winter rainfall, which is also required for seedling emergence (McGee, 1974, 1977; Bokor *et al*., 1975). In Ontario, Canada, ascospores may be released from debris of recently harvested crops from September to November, when they can infect seedlings of new winter oilseed rape crops (Rempel & Hall, 1993). In western Canada, by contrast, ascospores are released from debris from May to August after the long, cold winter, when they can infect leaves of the new spring oilseed rape crops (McGee & Petrie, 1979; Kharbanda, 1993). In western Europe, ascospores are released from debris of the previous season's winter oilseed rape crop from late September onwards throughout the autumn/winter period, although the timing of maxima in ascospore release differs from season to season (Gladders & Musa, 1980; Pérès *et al*., 1997; Thürwächter *et al*., 1999). In eastern Europe, there may be some ascospore release from September to November, followed by the main period of release in the spring after the cold winter (Jedryczka *et al*., 1999b). Figure 1 shows the stages in the disease cycle of *L. maculans*.

Cotyledon and leaf infection

Both ascospores and conidia adhere to the cotyledons and leaves of new crops and germinate in humid or wet conditions to produce hyphae that cause infection. Infection is predominantly via stomatal pores (Fig. 2, Hammond *et al*., 1985; Chen & Howlett, 1996) but also via wounds. Although *L. maculans* produces a wide range of cell-wall degrading enzymes, no cutinase was detected by Annis & Goodwin (1996). Hall (1992) reported that ascospores germinated within 4 h at 4–28°C in laboratory experiments, suggesting that infection is normally limited by wetness rather than by temperature. This was confirmed by Biddulph *et al*. (1999a), who found that 4 h was the minimum wetness period required to produce leaf lesions by ascospores of both A and B group *L. maculans and* that most lesions were produced following a leaf wetness duration of 48 h at 12–20°C. Compatible leaf infections by A group ascospores produced leaf lesions (phoma leaf spot) after 5 days at 20°C and after 2 weeks at 8°C; these controlled environment results were validated by observing progress of phoma leaf spot epidemics in relation to weather in UK winter oilseed rape crops in several seasons (Biddulph *et al*. (1999b). However, the incubation period between infection and leaf lesion formation may differ between cultivars and between leaves at different positions (Poisson & Pérès, 1999).

Phoma leaf spots

Leaf lesions or phoma leaf spots (Fig. 1b; Table 3) vary in appearance depending on host resistance, *L. maculans* group and the stage of lesion development, but appear to develop similarly in Australia, Canada and Europe. Lesions caused by the A group of *L. maculans* first appear as pale green spots, which enlarge to 1–2 cm in diameter, often turning pale brown and containing tiny dark specks, pycnidia, that produce conidia. Eventually the centre of the lesion may break or fall out completely. Smaller, darker leaf spots with few or no pycnidia tend to be caused by the B group of *L. maculans* (Brun *et al*., 1997; Ansan-Melayah *et al*., 1997). In Australia, since seedling emergence often coincides with ascospore release, phoma leaf spots may be seen on young plants (Barbetti & Khangura, 2000). In the autumn distinctive leaf spots may be seen on winter oilseed rape in Ontario, Canada (Hall *et al*., 1993), and western Europe (West *et al*., 1999) but few indistinct spots are seen in eastern Europe (Jedryczka


a
b
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A
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e

Table 2 Stages in the life cycle of *Leptosphaeria maculans* in different regions where severe phoma stem canker epidemics occur

	Australia	Canada	Europe
Period of ascospore release[a]	Late April to end of August	West: May to August Ontario: September to November, May to August	West: September to April East: September to November, April
Seedling blight (blackleg)[b]	Sporadic outbreaks destroy crops (mainly in the west) (July)	Occasionally	Extremely rare
Phoma leaf lesions[c] Spots	Leaf spots throughout the growing season	West: leaf spots at flowering (June, July) Ontario: leaf spots on young winter oilseed rape (October to December)	West: distinctive leaf spots on young plants, October to April East: little leaf spotting
Phoma stem canker[d]			
Crown canker	Most severe phase of disease; can occur at any growth stage	Develops in preharvest period (August)	West: most severe phase of disease (May to July) East: rare?
Phoma stem lesions[e]	Observed on stems during and post flowering (September to November)	Develop in preharvest period	Generally more severe in east than west Europe (June/July)
Survival on debris (years)[f]	West: 3–4 South-east: 1–3	Several	< 2

References

[a]Australia, Bokor *et al.* (1975), McGee (1974, 1977) (Barbetti M.J., unpublished); Canada, McGee & Petrie (1979), Kharbanda (1993), Rempel & Hall (1993); Europe, Pérès *et al.* (1997), Thürwächter *et al.* (1999), Gladders & Symonds (1995), Jedryczka *et al.* (1999b).
[b]Australia, Barbetti & Khangura (1999); Canada, Kharbanda (1993); Europe, Paul & Rawlinson (1992).
[c]Australia, Barbetti & Khangura (2000); Canada, Hall *et al.* (1993), Mahuku *et al.* (1996); Europe, Paul & Rawlinson (1992), West *et al.* (1999), Jedryczka *et al.* (1999c).
[d]Australia, Barbetti & Khangura (2000); Canada, Hall *et al.* (1993), Mahuku *et al.* (1996); Europe, Paul & Rawlinson (1992), West *et al.* (1999), Jedryczka *et al.* (1999c).
[e]Lesions on stem more than 5 cm above the crown.[f]Australia, Bokor *et al.* (1975), Barbetti & Khangura (1997); Canada, Hall (1992); Europe, West *et al.* (1999).

et al., 1999c). New spots continue to appear on leaves throughout the winter in western Europe, but not in eastern Europe or Canada, where the winter is much colder. In western Canada, leaf spots are observed on leaves of spring oilseed rape in June and July (Kharbanda, 1993).

Wood & Barbetti (1977b) reported that only one or two ascospores were needed to initiate a lesion in optimal temperature and wetness conditions. By contrast, in controlled environment experiments, conidia were able to infect only wounded leaves, petioles and stems (Hammond, 1985) and were unlikely to infect unwounded leaves unless applied at very high concentrations to older leaves (Vanniasingham & Gilligan, 1989). Secondary infections caused by conidia are rare in Canada and Europe but more common in Australia (Alabouvette *et al.*, 1974; Barbetti, 1976; Wood & Barbetti, 1977b; Gladders & Musa, 1980; Thürwächter *et al.*, 1999). In Western Australia, conidia can spread the disease up to 1 m from original foci (Barbetti, 1976) in crops of very susceptible cultivars (Barbetti, 1975a). This may reflect the greater aggressiveness of Australian isolates (Kucher *et al.*, 1993) but secondary infections are not generally considered to affect yield (Hall, 1992).

Initial symptoms of seed-borne infection are seen on cotyledons as distinct circular lesions with large numbers of pycnidia at growth stage (GS) 1·0–1·4 (Sylvester-Bradley & Makepeace, 1985). The fungus is able to grow from cotyledon lesions (Bokor *et al.*, 1975) and leaf lesions (Hammond *et al.*, 1985; Hammond & Lewis, 1986a) biotrophically in the lamella and petiole of the leaf to colonize the hypocotyl and stem (Hammond & Lewis, 1987). However, the fungus may be isolated from leaves without lesions (Hammond

Figure 1 Stages in the life-cycle of *Leptosphaeria maculans* on oilseed rape. (a) A group ascospores from the UK (scale bar = 20 μm); (b) typical pale leaf lesions with pycnidia caused by the A group (A) and darker lesions, with few pycnidia, caused by the B group (B) on cv. 'Lipton' in the UK (February 2000); (c) cotyledon and hypocotyl infection on a young plant (cv. 'Karoo') in Western Australia (July 1998) (courtesy of R. Khangura, Agriculture Western Australia); (d) phoma stem lesion on the upper stem of cv. 'Tower' (October 1979) (Agriculture Western Australia); and (e) crown cankers of different severities on cv. 'Lipton' (June 1999) in the UK.

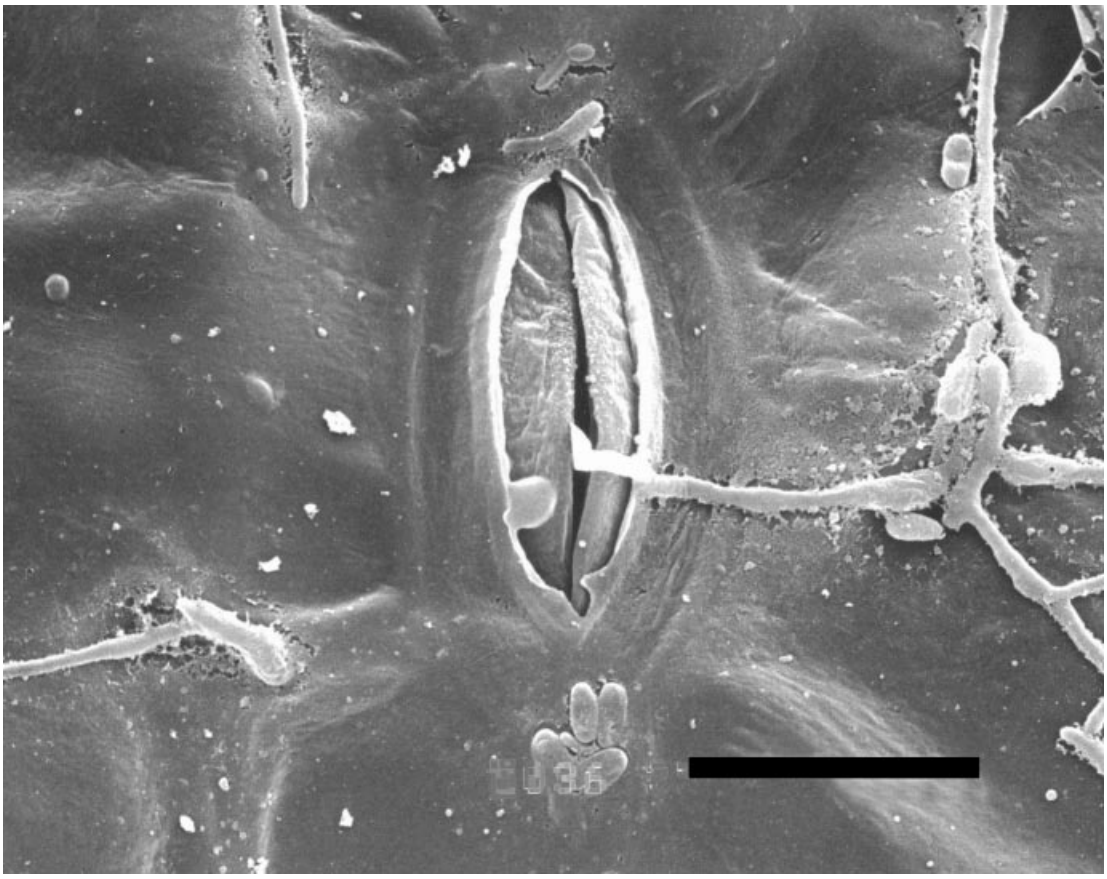

Figure 2 Scanning electron micrograph showing hyphae from conidia of A group *L. maculans* invading an oilseed rape leaf via a stomatal pore (scale bar = 20 μm). Courtesy of Alberta Research Council, Vegreville, Alberta, Canada.

Table 3 Proposed terminology for describing symptoms caused by *Leptosphaeria maculans* at different stages of oilseed rape crop growth in different regions where severe phoma stem canker epidemics occur, with alternative terms used

Plant tissue/ growth stage (GS)[a]	Proposed term	Symptoms[b]	Alternative terms
Leaves seedlings (1,0) young plants (1,1–1,12) stem extension (2,0) flower buds (3,0–4,9)	Phoma leaf spot	Large, pale brown necrotic lesions with pycnidia; smaller dark lesions	Leaf lesions
Stems	Phoma stem canker	Lesions on any part of stem	
seedlings (1,0)	Blackleg	Black/brown lesions on hypocotyl, may kill plant	Hypocotyl rot, seedling blight
flowering (4,0–5,2)	Blackleg	Black/brown lesions on green stem bases	Canker, stem base canker
Pod development (5,3–5,9) Seed development (6,1–6,9)	Crown canker	Dry rot which develops from lesions at stem base	Basal canker, collar rot, stem canker
	Phoma stem lesions	Pale brown lesions (dark margin) on stem above base (with pycnidia)	Upper stem lesions, blackleg, stem canker
Pods	Phoma pod spot	Pale brown lesions (dark margin)	

[a]Sylvester-Bradley & Makepeace (1985).
[b]Photographs of most of these symptoms can be seen in Fig. 1 or in Paul & Rawlinson (1992).

et al., 1985; Hall, 1992), which may explain why the correlation between incidences of phoma leaf spot lesions and basal stem cankers has sometimes been poor (Schramm & Hoffmann, 1991; West *et al*. 2000a).

Hypocotyl and stem infection

Hypocotyl infection produces a constriction in the stem above the ground and below the first leaves (Fig. 1c). The black/brown blackleg lesions on hypocotyls can cause a severe seedling blight and in Western Australia up to 70% of seedlings have been killed in individual crops (Barbetti & Khangura, 1999). Even up to the six-leaf stage, blackleg lesions may completely sever the tender stem base of seedlings in Australia (Barbetti & Khangura, 1999) and Canada (Kharbanda, 1993) but such attacks are extremely rare in Europe (Paul & Rawlinson, 1992). Blackleg seems an appropriate term with which to describe these black/brown lesions, when they form at the base of green hypocotyls of seedlings (Fig. 1c) or on green stems of older plants during flowering (Fig. 1e) (Table 3).

Blackleg lesions at the stem base (crown or root collar) often have a distinct dark brown or purple margin, and are typically associated with leaf scars; such lesions originate from lesions on leaves early in the season (i.e. in the autumn in western Europe: Hammond *et al*., 1985). During the pod development and seed ripening stage, these lesions may spread or coalesce and crack open to form the dry rots or cankers (Paul & Rawlinson, 1992) that generally form the damaging phase of epidemics. The terminology used to describe these stem base disease symptoms includes terms such as blackleg, crown canker, canker, collar rot and basal canker (Table 3); these symptoms on the dry, woody stem base seem most appropriately called 'crown canker' since they are no longer black. In Australia, these crown cankers often appear relatively early and can become very severe (Bokor *et al*., 1975). In Canada, in recent seasons severe crown canker epidemics have increased in incidence and have gradually spread westwards (Kharbanda, 1993). In Europe, severe epidemics occurred in the 1970s, were uncommon in the 1980s but have increased in incidence in the 1990s (Gladders *et al*., 1998). The rarity of severe epidemics in the early 1980s was probably because less severe cankers developed on new, more resistant cultivars such as Jet Neuf and Rafal (Gladders & Symonds, 1995; Gladders *et al*., 1998), although Humpherson-Jones (1986) noted that large numbers of pseudothecia were still produced on their stubble residues. Severe crown canker epidemics are common in western Europe but these stem base symptoms are rare in eastern Europe (Table 2, Jedryczka *et al*., 1999c).

From leaves infected later in the season (in late winter or spring in the UK), *L. maculans* spreads from phoma leaf spot lesions to produce lesions on upper parts of stems (Hammond *et al*., 1985). These lesions have been referred to as upper stem lesions, blackleg or stem canker, but 'phoma stem lesions' seems a more appropriate term because they are not generally black or cankerous. Furthermore, use of this term would distinguish them from sclerotinia stem rot, which can occur in the same region of the stem. Such phoma stem lesions appear at relatively early growth stages (e.g. during flowering, Fig. 1d) in Australia and can reduce yield in Australia (Barbetti & Khangura, 2000), Canada (Hall *et al*., 1993) and Europe (Zhou *et al*., 1999), especially in Poland (Jedryczka *et al*., 1999c). The term phoma stem lesions is proposed to describe symptoms on the stem >5 cm above the crown (where crown cankers occur) with the term phoma stem canker to cover all symptoms on stems (blackleg, crown canker and phoma stem lesions). Both crown cankers and phoma stem lesions may girdle the stem, causing premature ripening of the pods due to disrupted water transport (Davies, 1986), and in severe cases the stem is weakened enough to cause lodging and death of the plant. Late infections on the pods (siliques) are rare but phoma pod spots can cause premature ripening, splitting and decreased yield (Petrie & Vanterpool, 1974). More importantly, pod infections can spread to the seed inside (Wood & Barbetti, 1977a; Kharbanda & Stevens, 1993).

Survival and disease spread

After harvest, the senescent stem tissues are rapidly colonized by *L. maculans* and pycnidia are produced abundantly. Additionally, conidia are able to colonize stubble saprophytically and may increase inoculum levels and subsequent numbers of pseudothecia. Pseudothecia develop in these residues and, when mature, ascospores are released over an extended period of time. Ascospores from infested residue were considered a risk to crops several kilometres away in Australia (Bokor *et al*., 1975), Canada (Petrie, 1978) and Europe (Gladders & Musa, 1980). However, a separation of only 2 km is now recommended in Western Australia, reflecting the current use of more resistant cultivars and the practical difficulties of avoiding proximity to stubble-based inoculum (Barbetti & Khangura, 1999). The greatest risk of infection is within 500 m of the source with spore numbers decreasing with distance due to deposition and dilution, as indicated by experiments in Australia (Barbetti *et al*. 2000). Ascospores can remain viable for about 6 weeks (Paul & Rawlinson, 1992) and it is probable that a small number of ascospores travel considerable distances.

Although rare, seed-borne infection can be very important in spreading the disease to completely new areas. *L. maculans* can be found in crucifer seed as dormant mycelium within the seed coat or even within the embryo (Jacobsen & Williams, 1971). In experiments in Canada the incidence of infected (artificially contaminated) oilseed rape seed at sowing was correlated with the incidence of plants with phoma stem canker and the incidence of infected seed at harvest of

that crop by Hall *et al.* (1996), who reported the incidence of infected seed per sample in Canada to be < 5%, while in Western Australia, Wood & Barbetti (1977a) found that the incidence of seed infection was 0·1–0·2% in some seasons, but considered it unimportant in the presence of extensive areas of stubble-based inoculum. Infected seed of other brassica crops such as mustard (*B. juncea*) may also help to spread the disease (Gugel & Petrie, 1992).

Factors affecting severity of *L. maculans* epidemics

The differences between Australia, Canada and Europe in the epidemiology and severity of phoma stem canker epidemics reflect differences between continents in the population structure of *L. maculans*, the climate, cultivars grown, cultural and control practices, and the interactions between these factors.

Structure of *L. maculans* populations in different regions

The population structure of *L. maculans* appears to be an important factor affecting the severity of epidemics in different regions (Table 4). Survey results suggest that the population is entirely A group in Australia, where the epidemics are often very severe (Ballinger & Salisbury, 1996; Chen *et al.*, 1996). Western Australian isolates were found to be more pathogenic than isolates from western Canada (Kucher *et al.*, 1993), and Purwantara *et al.* (2000) also suggest that Australian isolates are more aggressive than those from other regions. In western Canada, phoma stem lesions caused by the B group are considered to appear late in the season during pod maturation, when they are superficial and cause little damage (Kharbanda, 1993). The increasing severity of epidemics in Canada appears to be associated with the increasing proportion of the A group. Epidemics are often more severe in western Europe, where the A group is usually predominant, than in eastern Europe, where the B group is predominant.

In experiments in Europe, pathogenicity to oilseed rape stems differed between A- and B group isolates (Johnson & Lewis, 1994). On susceptible cultivars, A group isolates caused damaging brown, cortical lesions but B group isolates penetrated the leaf gap to enter the stem pith, rarely causing externally visible phoma stem cankers (Hammond & Lewis, 1986b, 1987; Johnson & Lewis, 1994). However, as stems senesce and turn brown, abundant pycnidia of both groups are produced on their surfaces. Furthermore, whereas the A group is generally associated with damaging crown cankers at the base of plants, the B group seems to be more prevalent on upper parts of the stems (Johnson & Lewis, 1994; Thürwächter *et al.*, 1999; Wang, 1999). Nevertheless, severe phoma stem lesion epidemics have been associated with the B group in Poland (Jedryczka *et al.*, 1999c).

There is a need for caution when interpreting survey data, because different methods are used in different surveys with samples often taken at different times in the season. Earlier surveys usually relied on the different pigment production in media to distinguish A group and B group isolates, whereas recent surveys have used molecular methods, such as PCR (e.g. Sosnowski *et al.*, 1999; Penaud *et al.*, 1999a), or immunological methods (e.g. an immunologically based kit released in 1998: Blackleg Alert, Brooks Diagnostics Ltd, Crop Centre South, PO Box 1701, Brooks AB, T1R 1C5, Canada). Nevertheless, the survey data provide good evidence that a predominance of the A group is associated with the occurrence of severe crown canker epidemics, because the A group is generally considered to be more damaging than the B group (West *et al.*, 1999).

Where *L. maculans* populations are a mixture of A group and B group, the proportions of the different isolates appear to be influenced by a complex interaction between pathogen, host, climatic and cultural factors. Even within a region, there can be large differences in the population structure of the pathogen. In recent surveys in France, isolates collected from a southern site were 95% A group, and those from the east were 62% B group, while other areas had roughly equal proportions of the two groups (Penaud *et al.*, 1999a), although it is not clear whether these proportions differ from season to season. In the UK, past surveys have suggested not only that there are large regional variations in the population structure (Humpherson-Jones & Ainsworth, 1983) but that the proportion of B group isolates was greater in the 1980s (Humpherson-Jones, 1986) than it is now (J. S. West, unpublished results), although the earlier survey depended on samples taken relatively late in the season when there are normally more B group lesions present. In Canada, the proportion of the A group, and the severity of phoma stem canker epidemics, has gradually increased in western oilseed rape growing provinces in the last decade, despite efforts to prevent this (Table 4).

The complex interaction between populations of the A group and B group is demonstrated by the changes in population structure that occur during the course of a growing season. In France, a greater proportion of A group isolates was collected in the autumn than at harvest (Penaud *et al.*, 1999a). In the UK, pseudothecia have been observed earliest on the stem bases at positions of severe crown cankers and pseudothecia at sites of phoma stem lesions were produced later (Gladders & Musa, 1980; Hammond, 1985). Petrie (1995a) also found that ascospores were produced earlier from oilseed rape stems with basal cankers than from stems with superficial lesions from the same field. Predominantly, A group isolates have been obtained from stem bases while A and B group isolates have been obtained equally from upper stem parts in the UK, France and Germany (J. S. West, unpublished results; J. Schmit, INRA Versailles, France, personal communication; Thürwächter *et al.*, 1999). Therefore, there is

Table 4 Proportions of A and B groups of *Leptosphaeria maculans* in different regions

Region	Population (A:B group)	Methods
Australia[a]	A group*	Isolation from leaf and stem lesions (several hundred isolates, identified by PCR[h] and by electrophoretic karyotyping)
Western Canada[b]	Formerly B group was predominant. A group, first reported in Saskatchewan in 1975 and in Alberta in 1983, now widespread in most areas	Annual disease surveys since 1965; detailed A:B group surveys (*c.* 2000 crops per year) in Alberta since 1988; isolation from leaf and stem lesions, June to Sept; identification culturally, recently by ELISA[g]
Ontario, Canada[c]	70% A group; 30% B group from leaf lesions. Phoma stem lesions all A group	1993 survey (two fields) on leaves (June), stems (harvest in August) and debris (October); identification by PCR (total samples approximately 200)
Western Europe[d]	Mixture of A and B groups, with A group now predominant in most areas; during a season percentage of B group increases	Isolation from leaf (Oct. to April), and stem lesions (June/July) and debris after harvest (July/August) 1986, mostly since 1995; identification culturally or by PCR (several thousand isolates)
Eastern Europe[e]	B group becomes more prevalent further east. Polish population almost exclusively B group	Isolation from stem lesions and debris since 1983; identification culturally or by PCR[h] (several hundred isolates)
China[f]	Only B group (small number of samples)	Isolation of ascospores from stem debris in 1999 (18 isolates)

References

[a]B. Howlett (School of Botany, University of Melbourne, Victoria, Australia, personal communication)· [*There is an unconfirmed report of two B group isolates from Australia by Sosnowski *et al*. (1999)].

[b]Alberta, Evans *et al*. (1995), Harrison *et al*. (1995), P. D. Kharbanda (unpublished); Manitoba, Platford (1996); Saskatchewan, McGee & Petrie (1979), Petrie (1995b); Kaminski *et al*. (1996).

[c]Mahuku *et al*. (1996).

[d]France, Ansan-Melayah *et al*. (1997), Penaud *et al*. (1999a); Germany, Kuswinanti *et al*. (1999); UK, Humpherson-Jones (1986), Jedryczka *et al*. (1999c), West *et al*. (unpublished).

[e]Jedryczka *et al*. (1999c).

[f]West *et al*. (2000b).

[g]ELISA, enzyme-linked immunosorbent assay.

[h]PCR, polymerase chain reaction.

potential for the population structure of the pathogen to change during the season as pseudothecia on different parts of stem residues mature. This might explain why 95% of ascospores released exceptionally early (May) in Saskatchewan, Canada were of the A group (McGee & Petrie, 1979), and in the UK leaf lesions caused by the B group were observed to occur slightly later than A group leaf lesions (Johnson & Lewis, 1994). Furthermore, the proportion of the B group was large in seed from the UK, which must have been infected near the end of the season (Wang, 1999).

It is likely that the balance between A group and B group may change between seasons in addition to during seasons. If seed infection is predominantly by the B group, then the A group is less likely to be introduced to new areas, although this can still occur. If upper stems, with their greater proportion of B group inoculum, are removed during harvesting, then the proportion of A group inoculum from the stem base will increase after harvest. There is a need for clearly defined, accurate surveys to examine in detail the changes in proportions of A and B groups, both within and between seasons, and in different locations. The incidence of phoma stem infection caused by the B group can be underestimated because infection may be confined to the pith without causing external symptoms and symptomless leaf infections also occur (Hammond *et al*., 1985; Hall, 1992).

Modern techniques enable disease surveys to investigate the population structure within the A group. This could help with the choice of cultivars by assessing which pathogenicity groups are present in an area. Pathogenicity groups of the A group are numbered 1–4, based on symptom scores on inoculated cotyledons of three cultivars (Westar, Glacier and Quinta), where four possible outcomes usually occur (Rimmer & van den Berg, 1992). Care is needed in the interpretation of pathogenicity group experiments because variability within batches of seed of the three cultivars used, plus the effect of temperature or other environmental conditions on symptom expression, may lead to discrepancies. The A group population has different ratios of pathogenicity groups in different regions. In Ontario, Canada, Mahuku *et al*. (1997) found PG4 (80%), PG3 (11%) and possibly a new group (PG5, 9%) of isolates giving an intermediate reaction on all three cultivars. In contrast, western Canadian isolates were mainly PG2 (Mengistu *et al*., 1991). In France, over 90% of isolates were PG3; the remainder were mostly PG4 and a few were PG2 (Ansan-Melayah *et al*.,

1997; Penaud *et al*., 1999a). In Australia, PG2, PG3 and PG4 are present (Mengistu *et al*., 1991). Surveys could provide valuable information about the dynamics of *L. maculans* populations, at the level of pathogenicity group, in response to the cultivation of a predominant cultivar and different control practices used in each region.

Climate

The relationship between climatic factors, such as temperature and rainfall (Table 1), and differences between continents in the epidemiology and severity of phoma stem canker epidemics is complex, with different factors affecting survival of inoculum, maturation of pseudothecia, timing of ascospore release, infection conditions and host resistance. Climatic factors affect the persistence of pseudothecia on crop debris because the rate of degradation of debris is affected by wetness and soil temperature, with survival favoured by dry summers or cold winters. Residues remain an inoculum source for up to 4 years in western Australia, due to the hot, dry summers (Barbetti & Khangura, 1997), and for several years in western Canada, with its very cold winters and dry hot summers (Petrie, 1986). In contrast, the mild, wet climate in the UK promotes rapid decomposition of debris, which generally degrades within 2 years (West *et al*., 1999). In south-east Australia, oilseed rape residues declined in volume by 90% within 1 year and produced fewer ascospores in the second year than in the first year after harvest (Table 2).

Pseudothecial maturation depends on both temperature and wetness, with an optimum at 14–15°C (Petrie, 1994; Poisson, 1997; Pérès *et al*., 1999). In western Australia, mature pseudothecia do not usually form until the autumn/winter following harvest the previous spring because their maturation stops in the dry, hot Mediterranean-type summers. Pseudothecia have been observed as early as harvest time in North America and Europe (Hershman & Perkins, 1995; J. S. West, unpublished results). However, dry weather over the summer period in many areas delays further maturation and spore release. In Ontario, pseudothecia have been seen on stubble of the current year's crop in September, only one month after harvest, releasing ascospores from late September to infect the new winter crop (Rempel & Hall, 1993). Ascospore release in western Canada is delayed by subzero winter temperatures so that pseudothecia usually form 9–10 months after harvest on standing stubble (Kharbanda, 1993).

The timing of spore release (Table 2), and hence the onset of phoma leaf spot in Europe, appears to be associated with temperature and summer and autumn rainfall, which affect both pseudothecial maturation and ascospore release (Gladders & Symonds, 1995; LePage & Penaud, 1995; Pérès & Poisson, 1997; West *et al*., 1999). The first release of ascospores in France occurred 16–19 rain-days after harvest, when the average temperature decreased to 14°C (Pérès & Poisson, 1997). The first ascospore release in France is generally earlier (in September) than in the UK (in October: Biddulph *et al*., 1999a, b), perhaps because the mean temperature during this period is greater in France. Pérès & Poisson (1997) used a combination of the date of first spore capture and weather conditions (amount of rain, number of rain-days, temperature and number of hours of relative humidity above 90%) to produce a preliminary model to advise on spray timing at different sites. In dry Mediterranean-type climates, such as southern France and Western Australia, spore release is well synchronized with rain events, whereas in wetter areas, such as the UK, the disease, although mainly monocyclic, is initiated by ascospore release over a long period, lasting several months.

The timing of initial leaf infections, in relation to the growth stage of the plant, may affect both the success of infection and the subsequent phoma stem canker severity. McGee & Petrie (1979) found that the first six leaves appear to be more susceptible to infections than those produced later. Poisson & Pérès (1999) found that symptoms appeared more quickly (after 19 days) on leaf six than on leaves four or two (35 days), giving the impression that the earlier leaves are more resistant especially since 'resistant' cultivars showed symptoms later than susceptible ones. However, when isolations were attempted from symptomless leaves, the fungus was found on leaf two after only 2 days, on leaf four after 6 days and on leaf six after 14 days, suggesting that the early leaves were more susceptible but had not produced lesions readily. Additionally, Badawy *et al*. (1991) found that on ageing host tissue, symptom expression, even by the B group, was more severe than on younger tissue. These differences in symptom expression could explain why correlations between leaf spotting and crown canker severity are not always apparent.

There is evidence from Australia (Barbetti, 1975a; Bokor *et al*., 1975; Barbetti & Khangura, 1997), Canada (McGee & Petrie, 1979), France (Brunin & Lacoste, 1970; Poisson & Pérès, 1999), Germany (Badawy *et al*., 1992) and the UK (Hammond & Lewis, 1986a) that the most severe crown cankers develop from cotyledon and leaf spots on young plants early in the growing season, whether this is in the autumn (Europe, Ontario), spring (Canada) or winter (Australia). Thus, the severe epidemics may occur in Australia because the winter rainfall that is required for seed germination and emergence of oilseed rape seedlings also serves to synchronize release of ascospores of *L. maculans* with emergence (Table 2; Barbetti & Khangura, 1997). The exact growth stage up to which infection leads to severe cankers appears to vary with region and type of oilseed rape grown. McGee & Petrie (1979) found that inoculation after the six-leaf stage led to canker development too late to cause severe yield loss, while Hammond & Lewis (1986a) found that in the UK, the most damaging cankers are formed from

leaf infections occurring before the onset of rapid stem extension and mainly on leaves 3–10. In Europe and Ontario, late ascospore releases (late autumn and winter) cause infections of later, larger leaves leading to stem infections when the temperature is low and when the stem base has become tougher. Little damage is done to the stem in the cold winter conditions and most phoma stem canker lesions are not apparent until temperatures increase in the spring. Zhou *et al*. (1999) showed that in the UK, the earlier crown canker symptoms appeared the greater the yield loss, and that in older plants, after the flowering stage, new crown cankers seldom developed while existing cankers became more severe. This is because stem infections are associated with leaf scars (Hammond *et al*., 1985), so any infections that reach the stem base do so from leaf infections during the rosette stage and will produce symptoms that continue to develop. Infections of later leaves will only lead to lesions further up the stem rather than additional crown cankers.

Symptom severity on cotyledons, leaves and stems appears to be increased by high temperatures. On plants inoculated at the cotyledon growth stage, crown canker was more severe at temperatures above 12°C than at lower temperatures (Barbetti, 1975b; McGee, 1977). Cotyledons inoculated with A group isolates at 18°C produced incompatible reactions but at 27°C the reaction was compatible (Badawy *et al*., 1992). This supports the suggestion by Boudart (1982) that resistance genes of juvenile plants may be temperature sensitive, producing more disease at 24°C than at 14°C. On plants inoculated at the green bud stage, phoma stem canker symptoms were less severe at 12°C than at 18°C (McGee & Petrie, 1979). The most severe epidemics, in Australia, are associated with a climate in which temperatures are typically 25–30°C during the period when phoma stem canker lesions are developing. Severe epidemics can also occur in Canada and eastern Europe, where crops experience high summer temperatures. Although such severe epidemics do not occur on oilseed rape in China, despite the high summer temperatures, this could be due to the scarcity or absence of the A group (West *et al*., 2000b). Additionally, the severity of phoma stem canker epidemics decreases in the UK with distance northwards, and the disease is rare on oilseed rape in Scotland, possibly because host resistance operates well at the low summer temperatures found there. However, higher temperatures may also increase phoma stem canker severity because water movement restrictions, caused by stem infection, could prevent increased transpiration rates, leading to water-stress. This would result in the plant senescing early and could explain why drought-induced water-stress, occurring around harvest, exacerbates the yield loss caused by crown canker in western Australia (Barbetti & Khangura, 1997). Water stress induced by application of a desiccant to facilitate harvesting of a crop can also cause *L. maculans* to spread rapidly in stems as they senesce.

Physical damage can increase infection; wind damage at the early seedling stage, sometimes combined with resultant sand-blasting, can increase susceptibility to infection in western Australia (Barbetti & Khangura, 1999) and damage caused by hail in Canada was thought to enhance disease symptoms (P. Kharbanda, personal communication). These climatic and population factors, which influence the severity of epidemics, need to be considered when devising the best strategy for managing phoma stem canker epidemics in Australia, Canada or Europe.

Implications for disease management

Differences in the epidemiology of phoma stem canker and economics of oilseed production between different regions have considerable implications for the management of the disease by cultural, chemical and other methods.

Cultural practices

In all regions, stubble management and good crop rotation (4 years breaks between oilseed rape crops are usually recommended) decrease the risks of infection by ascospores released from colonized residue (Table 1). The greatest problems with infected residues occur in western Australia, where a greater use of minimum tillage following oilseed rape has increased the amount of infected residue remaining on the soil surface, particularly as the dry climate promotes its persistence. Closer rotations have also increased amounts of stubble residues nearby to subsequent oilseed rape crops (Barbetti & Khangura, 1999). This is associated with an increase in the area of oilseed rape cultivation in Australia from 175 000 ha in 1993 to 1·8 Mha in 1999 (Burton *et al*., 1999), with 35 000 ha in western Australia in 1993 increasing to 510 000 ha in 1998 (Barbetti & Khangura, 1999) and then to 920 000 ha in 1999. Currently, destruction of infected residues by raking, burning or burying is recommended and improved methods for the management of residues and enhancement their breakdown are being sought (Barbetti & Khangura, 1999; Barbetti & Khangura, 2000). The problem of stubble management is not so acute in Europe, where infected residues break down more quickly (Table 2). This factor may also explain why phoma stem canker is not considered to be a problem in China (West *et al*. 2000b) or India where labour intensive cultural practices, such as removal of the whole plant at harvest and subsequent flooding of fields for the following rice crop, help to destroy the inoculum. In Europe, deep ploughing to bury residues, followed by minimal tillage or direct drilling, is recommended but is difficult on heavy soils (Gladders & Musa, 1980). The ploughing of stubble is often delayed by farmers in order to allow seed, spilt at harvest, to germinate and produce seedlings, otherwise ploughed seed becomes dormant and poses a problem of

volunteer rape for several years. This leaves a very short period, of about 3 weeks, in which fields that have previously grown oilseed rape can be ploughed before the new crop of oilseed rape seedlings emerge. The use of herbicides to control volunteer rape in cereal crops and alternative hosts is also important (Gugel & Petrie, 1992).

In France, early sowing can allow the crop to have produced enough leaves, by the time ascospores are released, to evade infection at its most sensitive stage (LePage & Penaud, 1995). Conversely, in Australia delaying the sowing date was proposed so that seedlings would escape the period when most ascospores are present, because the number of ascospores in the air increased greatly to a maximum and then declined (McGee, 1977). However, in dry, Mediterranean-type climates there can be very severe yield losses associated with delayed sowing. More recently, in Western Australia, very early crop establishment, to minimize infection at the sensitive seedling stage, is showing promising results in some areas (R. K. Khangura & M. J. Barbetti, unpublished results).

Legislation has also been used in management of phoma stem canker. In Alberta, Canada, *L. maculans* was declared a pest under the Alberta Agriculture Pest Act in 1984, soon after the disease was first found there (in 1983). Under these Pest Act regulations, sowing or transporting infected seed was prohibited and farmers could not grow oilseed rape for 4 years in a field in which the disease had been found. Detection of seed-borne infection by the A group of *L. maculans* is now possible using monoclonal antibodies (Stace-Smith *et al*., 1993). Although the disease eventually spread throughout most of Alberta, the rate of spread was considerably decreased by this legislation.

Chemical control

Different combinations of fungicide seed treatments, soil fungicides or foliar fungicide sprays are used for control of phoma stem canker in different regions, depending on the epidemiology of the disease and the economics of the crop. In Canada, carbathin, thiram and iprodione are currently registered as seed treatments, and thiram and iprodione are used as seed treatment in parts of Europe (Table 1). Iprodione is also used in Australia for control of rhizoctonia hypocotyl rot and damping-off, although it is not specifically applied as a seed treatment against blackleg. However, flutriafol applied as a coating to fertilizer granules can protect young seedlings for a longer period than seed treatment alone, although serious losses from crown canker can still occur in crops grown with treated fertilizer granules, so it is advised that this practice should be integrated with other methods of disease management (Barbetti & Khangura, 1999). Generally it is recommended for use only in crops with high yield potential, where the level of inoculum is high (moderate to severe) and where cultivar resistance is low (susceptible or moderate adult plant resistance to crown canker).

Use of foliar fungicides in association with cultivars with little or no resistance has proved to be ineffective for phoma stem canker control in Australia (Brown *et al*., 1976). In Canada, propiconazole has sometimes been used as a foliar fungicide but it does not give complete control of phoma stem canker (Kharbanda *et al*., 1999). In western Europe, foliar sprays of difenoconazole alone or mixed with carbendazim, or flusilazole plus carbendazim, are often effective for the control of crown canker (Gladders *et al*., 1998). Furthermore, the greater yields obtained in western Europe can justify the use of more expensive fungicide spray treatments, whereas their use is uneconomic in areas where the yield is generally low due to shorter day-lengths and/or early harvesting caused by hot weather (e.g. Canada and Australia).

Many experiments have shown that the optimum time for application of foliar fungicides to control severe crown canker epidemics in western Europe is in the autumn, some 6 months before symptoms appear on stems, so there is a need for a forecasting scheme to predict the severity of epidemics at the time when decisions need to be taken (Gladders *et al*., 1998). Because current fungicides have low eradicant activity and are effective as protectants for only a limited period, as a result of degradation, leaf expansion and the production of new untreated leaves, it is important that fungicides are correctly timed (West *et al*. 2000a). Poisson & Pérès (1999) concluded that early leaves (leaves 2 and 4) did not readily produce lesions despite being infected, so fungicide spray decisions, based on the presence of leaf spotting on very young plants, may be too late to control the disease as the fungus could already have reached the stem by the time leaf lesions appear.

McGee & Petrie (1979) and Hammond & Lewis (1986a) indicate that fungicide use is not needed after a certain growth stage, when new leaf infections will not produce severe phoma stem cankers and result in yield loss. This key growth stage, marking a cut-off period for fungicide use, appears to vary with climate and host–pathogen interaction and has not been identified accurately. Consequently, farmers in the UK have often applied fungicides unnecessarily (Fitt *et al*., 1997). Methods for forecasting the severity of phoma stem canker epidemics in western Europe have been reviewed by West *et al*. (1999) and will only be discussed briefly. In the UK, the most widely used forecasts are based on the incidence of phoma leaf spotting on crops in the autumn, although such forecasts may not always allow growers sufficient time to control the disease before the fungus reaches the stem, when fungicides are ineffective. There are prospects for developing more accurate forecasts based on an understanding of the relationships between weather factors and ascospore maturation, release and infection, and for using immunological or molecular

techniques to detect airborne ascospore inoculum and symptomless leaf infection. In France, a system to improve fungicide spray timing based on epidemic risk and agronomic factors has been produced (Penaud *et al*., 1999b). This calculates the risk of infection using weather and biological factors (7 rain-days after sowing, maturation of pseudothecia or first detection of >20 ascospores per day). When the risk of infection is imminent, the decision to apply a fungicide is made, based on four agronomic factors: cultivar susceptibility, soil type, growth stage and plant vigour.

Host resistance and biological control

Breeding for resistance, at both seedling and adult plant stages, to both the A and B group isolates of *L. maculans* is a key requirement for sustainable management of phoma stem canker. Resistance reactions in adult plants, which impede the development of crown cankers or phoma stem lesions, are thought to have a different genetic basis to seedling resistance, which may impede initial spread in the leaf lamina or spread down the petiole to the hypocotyl or stem (Rimmer & van den Berg, 1992). The genetics of *L. maculans* pathogenicity and oilseed rape resistance responses have been reviewed previously (Rimmer & van den Berg, 1992; Williams, 1992; Balesdent *et al*., 2000) and are beyond the scope of this review. One area of confusion results from the fact that 'field resistance' to phoma stem canker observed in oilseed rape crops has several components. First, there is 'genetic resistance' to *L. maculans*, which is often assessed in juvenile plants only, by inoculating wounded cotyledons with conidia (e.g. Badawy *et al*., 1991). Secondly, there is also a 'disease escape' component, because plants that lose leaves with phoma leaf spots before the pathogen has reached the stem will escape the crown canker phase. Finally there is a 'disease tolerance' component, which may operate in combination with a 'genetic resistance' in the crown canker phase of epidemics, to decrease symptom development in plants. Tolerance factors could include physical toughness of the stem, inhibitory chemicals (not associated with defence reactions) and stem thickness, because thick, robust stems appear to tolerate infection more readily than thin, weak stems. All of these components of 'field resistance' may be influenced not only by host and pathogen (genetic) factors but also by cultural and environmental factors. For example, a crop with tough, thick stems may be produced by early sowing, a low plant density, moderate use of fertilizer, and a windy, cool climate; it may escape the worst of the epidemic and have increased tolerance in addition to its genetic resistance.

In Australia, there has been extensive work to identify new sources of improved seedling and adult plant resistance to the pathogen (Marcroft *et al*., 1999). Australian breeders have developed spring-type cultivars with good resistance to the A group of *L. maculans*, and this has been the reason for the re-establishment of an industry there (Salisbury *et al*., 1995) following the severe epidemics of the early 1970s (Bokor *et al*., 1975). However, the increased resistance of cultivars grown has led farmers in some areas to shorten rotations between oilseed rape crops which, combined with an increase in minimal tillage, has increased inoculum pressure, producing severe epidemics and yield losses. A system for advising growers about choice of cultivar based on the cultivar resistance rating and the risk of yield loss from phoma stem canker has been developed for western Australia (Barbetti & Khangura, 1999). The system considers different 'disease pressure' scenarios, based on proximity to infected debris of different ages, and shows the maximum potential yield losses for each scenario, giving growers the opportunity to assess their individual risks and to make informed choices from the options available to them (Table 5). In western Europe, the decrease in the severity of crown canker epidemics in the 1980s by comparison with the late 1970s was associated with the introduction of new cultivars with increased resistance to crown canker (Gladders & Symonds, 1995; Gladders *et al*., 1998). More recently, new winter oilseed rape cultivars capable of delaying mycelial penetration and crown canker formation are being developed in France (Pérès & Poisson, 1997).

Species of bird's nest fungus, *Cyathus striatus* and *C. olla*, have been investigated as biological control agents because they decrease the stubble food base on which *L. maculans* survives between oilseed rape crops and so can reduce the level of inoculum available to infect new crops (Tewari *et al*., 1997). Furthermore, a bacterium *Paenibacillus polymyxa* PKB1 has been found to produce two antifungal peptides that decrease the growth of *L. maculans* in culture, on leaves, on stems and on stubble (Kharbanda *et al*., 1999). Several fungicides and herbicides approved for use on oilseed rape in Canada did not affect this bacterium so it has potential for use as a direct biological pesticide. There is clearly also the prospect of genetic modification of oilseed rape to incorporate genes for antifungal proteins or resistance genes from other plants. Wang *et al*. (1999) showed that at least one pea (*Pisum sativum*) gene, constitutively expressed by the cauliflower mosaic virus 35S promoter, decreased phoma stem canker scores in inoculated plants.

Discussion

Differences in the epidemiology of *L. maculans* between Australia, Canada and Europe occur due to differences in the pathogen population, type of oilseed rape, cultivar, climate and timing of infection relative to the crop growth stage. Biological differences in the development of phoma stem canker suggest that there may be different diseases caused by the A and B groups of *L. maculans*. The two groups of *L. maculans* appear to be associated with different symptoms on both leaves

Table 5 Phoma stem canker risk assessment system (adapted from Barbetti & Khangura, 1999). Values are the estimated maximum potential yield losses in oilseed rape cultivars with different levels of adult crown canker resistance growing directly on ground with oilseed rape residues of different ages

Cultivar	Canker resistance score*	Potential yield loss (%) due to residue aged (years)			
		4	3	2	1
Hyola 42	4	10–15	25–35	60–70	100
Karoo	5	5–10	20–25	50–60	80–90
Oscar	6	0–5	10–15	40–50	70–90
Dunkeld	7	0	5–10	30–40	65–85

*Cultivars rated on a 1–9 scale: 1, extremely susceptible; 2, very susceptible; 3, susceptible; 4, moderately susceptible; 5, intermediate; 6, moderately resistant; 7, resistant; 8, highly resistant; 9, immune.

(Brun *et al*., 1997) and stems (Johnson & Lewis, 1994), which supports the genetic, biochemical and cultural evidence for the need to describe them as two separate species. The possibility that there may be different diseases has been confounded by the terminology used to describe the same symptoms in different countries. There is an urgent need to standardize the terminology, with the suggestions made (Table 3) providing a basis for discussion. Despite these epidemiological differences, there are similarities between the life cycles of the A and B groups of *L. maculans* in different regions, with epidemics initiated by airborne ascospores and pathogen spread from leaf to stem. Furthermore, in areas of Canada and Europe, the two groups appear to coexist (Table 4). While the importance of the A group in causing severe epidemics is clear, there is some confusion over the importance of the B group. Indeed, our understanding of how phoma stem canker severity (amount of infection on the diseased plants, rather than the average amount of disease on all plants) affects yield has not been fully quantified. It is not clear whether yield loss is due to reduced water movement alone or if phytotoxins contribute. In Canada, the B group is not considered to be a serious pathogen (Kharbanda, 1993), yet it has caused serious losses in Poland (Jedryczka *et al*., 1999c). The coexisting A and B group populations are dynamic, with changes both within and between seasons and between geographical locations. The groups may have evolved to exploit different niches, enabling them to cause disease on oilseed rape in the different climates and cropping systems found in Australia, Canada and Europe. A thorough understanding of the fluctuations in *L. maculans* population structure and the development of diseases caused by the A group and the B group is needed to improve strategies for management of phoma stem canker throughout the world.

In addition to finding improved seedling and adult plant resistance (Marcroft *et al*., 1999), knowledge of the virulence of pathogen populations in a region would improve the choice of cultivars to be grown (Balesdent & Rouxel, 1998; Balesdent *et al*., 1998). Furthermore, there may be a need for legislation, similar to that established in Alberta, Canada, to prevent the A group of *L. maculans* from spreading on contaminated seed into other new areas such as eastern Europe. Stubble management is also extremely important, mainly to decrease production of ascospores but also to avoid direct contact with new crops in areas where stubble residue persists. Ideally, the destruction of stem base residues would remove the most persistent source of inoculum and, because the A group is found more frequently at the stem base than the B group (Johnson & Lewis, 1994; West *et al*., 1999), the proportion of A group ascospores in the inoculum would decrease. In addition to inoculum management, it is important to use other cultural factors to exploit opportunities for disease escape and tolerance. Penaud *et al*. (1999b) indicated that crop vigour is important in assessing whether to apply fungicides. Therefore, it may be possible to combine both disease escape and tolerance to crown canker by early sowing and decreased planting densities both to avoid infections at the most sensitive stage and to produce thicker-stemmed, vigorous plants and thus to reduce fungicide use.

As the most severe crown cankers develop from phoma leaf spots on leaves at the start of the growing season, it is important to protect plants at this growth stage (Brunin & Lacoste, 1970; Bokor *et al*., 1975; McGee & Petrie, 1979; Hammond & Lewis, 1986a; Poisson & Pérès, 1999). However, a study by Poisson & Pérès (1999) indicated that fungicide applications in response to leaf spotting may be too late on young plants as the fungus will often reach the stem before leaf lesions appear. Improved fungicidal seed treatments could protect young plants over this crucial stage and would avoid the need for farmers to make foliar applications of fungicide. Alternatively, the method used in Australia, where fertilizer coated with fungicide is placed close to the seed at sowing to protect the young seedlings (Barbetti & Khangura, 1999), might be extended to other areas where severe phoma stem canker epidemics occur. Nevertheless, in the foreseeable future, control of phoma stem canker in western Europe is likely to rely on foliar fungicide applications. Forecasting schemes to optimize this spray timing to periods when pathogen inoculum is present and the crop is susceptible are under development in France (Penaud *et al*., 1999b) and the UK (Fitt *et al*., 1997; West *et al*., 1999) to decrease unnecessary fungicide use. Thus it may be possible to manage phoma stem canker

development and sustain oilseed rape production in Australia, Canada and Europe.

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