

Best Management Practices for Blackleg Disease of Canola

Kutcher, H.R., W.G.D. Fernando, T.K. Turkington and D.L. McLaren

Summary

Blackleg disease of canola in western Canada has been effectively managed for the past 20 years through a combination of resistant varieties, adequate crop rotation intervals and good agronomics. Increased canola production in western Canada, in large measure through more frequent cropping in the rotation, has increased the risk that resistance to blackleg in current varieties may be defeated. Loss of resistance in Canada and in other countries has been reported in varieties dependent on single resistance genes, indicating selection pressure for virulent races of the pathogen. Sustainable canola production in western Canada will require adoption of innovative blackleg control strategies by growers.

Introduction

Blackleg disease of canola (*Brassica napus* L., *B. rapa* L. and *B. juncea* L.), is caused by a complex of at least two species of *Leptosphaeria*: *L. maculans* (Desmaz.) Ces. & de Not. (anamorph *Phoma lingam* (Tode:Fr.) Desmaz.), and *L. biglobosa* Shoemaker and Brun. *Leptosphaeria maculans* is the more damaging of the two and was reported from almost all canola and oilseed rape growing regions of the world except China¹. These species were previously referred to as highly and weakly virulent forms of a single pathogen (*L. maculans*), until the latter was determined to be *L. biglobosa*^{2,3,4}.

Blackleg has been a production challenge for western Canadian canola growers since the mid-1970's, when the disease was observed to cause serious impairment of yield and quality in north-eastern Saskatchewan⁵. Prior to this the disease was present, but was not considered a significant production issue⁶. In the 1980's yield losses up to 50% were reported in individual canola fields⁷. Early disease control recommendations included use of clean (certified) seed and seed treatment, tillage to bury infected residue of previous canola crops, control of volunteers and susceptible weed species, rotations separating canola crops in the same field by at least three other crops (four-year rotation) and to locate canola crops away from adjacent fields containing infected stubble of previous canola crops⁸.

With the introduction of resistant varieties in the early 1990's⁹ and adherence to rotations of four years or longer (canola once every four years on a particular field) blackleg has been well controlled for most canola growers in western Canada. These measures reduced yield and quality losses associated with the disease but did not eliminate the pathogen. From the late 1990's, evidence was obtained for changes in the virulence of the pathogen^{10,11,12,13}, and in recent years producers have on occasion reported high disease severity in previously resistant varieties.

Future management of blackleg in western Canada will require integration of a combination of management practices. Given relative commodity price considerations, it is likely that canola will remain a frequent component of most western Canadian crop rotations for the foreseeable future. The adoption of the two year, wheat-canola cropping system in many parts of the prairies is particularly concerning because of the increased risk associated with the development of new races of the pathogen. Sustainable management requires crop scouting, adequate rotation of canola with other crop species, isolating canola production from previously infected fields by a recommended distance and

efficient weed and volunteer control. The use of certified seed and seed treatment is relatively high by canola growers in western Canada, which is important because these practices help to slow the spread across the prairies of new races of the pathogen. This paper reviews the current management of blackleg disease of canola in western Canada and suggests areas of research that may lead to new or improved strategies to control the disease.

Western Canadian blackleg management tactics

Scouting. Crop scouting and correct diagnosis of all crop production problems is crucial for effective canola cultivation and is critical for blackleg control. From knowledge of the incidence or severity of blackleg in a field, growers or agronomists can evaluate the effectiveness of current control strategies, and if needed change or recommend new management tactics.

Blackleg can be a challenge to diagnose because symptoms result from two species: symptoms of *L. biglobosa* are generally superficial and not believed to have a great impact on yield¹², while *L. maculans* may cause severe, yield limiting blackleg symptoms. Also, blackleg can be confused with other diseases, such as grey stem (*Mycosphaerella capsellae* A.J. Inman & A. Sivanesan), which does not cause significant yield loss¹⁴, and is obvious on canola stems late in the season, particularly after swathing (Figure 1).



Figure 1. Symptoms of blackleg caused by *Leptosphaeria maculans* on the left and those of grey stem (*Mycosphaerella capsellae*) on the right. Samples taken approximately 7 days after swathing from the same field. Photo credit: H.R. Kutcher.

Scouting to identify blackleg symptoms can be conducted throughout the growing season. Prior to seeding, in the early spring or even the previous autumn, a field can be scouted for remnants of the last canola crop grown on the field. If substantial residue from the previous canola crop remains, and blackleg symptoms were observed in that crop, there is a risk that the pathogen is present in the field. This can be confirmed in the spring when basal stem and upper root

pieces are inspected for the presence of pseudothecia and pycnidia (Figure 2), the fruiting bodies that produce ascospores and pycnidiospores, respectively. Fields on which canola has been grown in the previous 2-3 years will likely contain significant quantities of canola residue that may harbour the pathogen until this residue fully decomposes.



Figure 2. Pseudothecia and or pycnidia of *Leptosphaeria maculans* on residue of canola, cv. 'Westar'. Photo credit: D.J. Cross.

During the seedling stage, careful inspection can identify lesions caused by *L. maculans* on the cotyledons and early true leaves of canola (Figure 3). Such lesions indicate the presence of the pathogen and indicate that the variety being grown may not carry specific genes for resistance to the races of *L. maculans* present. However, this does not necessarily indicate that an epidemic (epiphytotic) of the disease will develop. Lesions caused by *L. maculans* may also be observed on the lower leaves of plants at the mid to late flowering stage of canola (Figure 3).



Figure 3. Lesions caused by *Leptosphaeria maculans* on *Brassica napus* varieties at the seedling stage (left) and on a lower leaf at the mid-flowering stage (right) of the crop. Photo credit: H.R. Kutcher.

The most appropriate time to identify and quantify the severity of blackleg infection in a crop is just prior to swathing, i.e., the ripening stage, growth stages 5.2 to 5.4 of the Harper and Berkenkamp scale¹⁵ or stages 80 to 87 of the BBCH scale¹⁶. At this time lesions may be found on upper plant parts including pods (Figure 4), although these are not as common as basal cankers. Lesions on the base of the stem may be large cankers with black borders accompanied by pycnidia on severely infected plants (Figures 5 and 6), or more subtle lesions with some discoloration but little pycnidia formation (Figure 7). Researchers often clip the basal stems and observe the cross-sectional area of the stem for an indication of the severity of infection (Figures 8 and 9).



Figure 4. Blackleg symptoms on a canola pod. Photo credit: H.R. Kutcher.

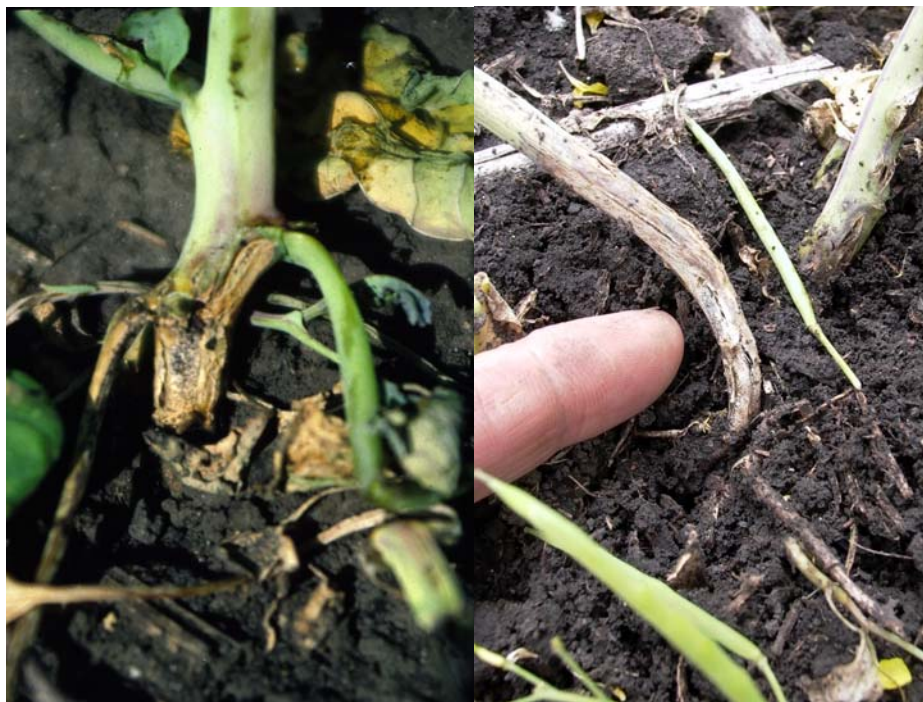


Figure 5. Severe symptoms of blackleg caused by *Leptosphaeria maculans* on variety ‘Westar’. Photo credit: H.R. Kutcher.



Figure 6. Canker with pycnids of *Leptosphaeria maculans* on unidentified variety of *Brassica napus*. Photo credit: H.R. Kutcher.



Figure 7. Less severely infected plants from an unidentified variety. Photo credit: H.R. Kutcher.



Figure 8. Blackleg symptoms on basal stems of canola plants showing blackening within the cross-sectional area of the plant stem.
Photo credit: H.R. Kutcher.



Figure 9. Clipped canola stems exhibiting varying degrees of severity of infection by *Leptosphaeria maculans*. Photo credit: H.R. Kutcher.

Other confounding factors, such as damage to canola stems by hail (Figure 10), or damage to lower stems caused by drift of phenoxy herbicides (Figure 11) from adjacent cereal fields, provide an opportunity for various pathogenic, and later saprophytic fungal species, including *L. maculans* to enter the plant. At swathing, root maggot infection may be apparent and blackleg is often found in association with wounding of the plant by root maggot (Figure 12).

Importantly, blackleg symptoms recognized in association with wounds caused by abiotic factors or other pest species should not be taken to mean that the genetic resistance inherent in a variety has broken down, or even that blackleg is necessarily the primary cause of damage to the plant and subsequent yield and quality losses.

Scouting the field for blackleg symptoms just prior to, or during swathing, does not allow remedial action to be taken to deal with the disease in that crop year. However, this information is valuable for future years to plan and assess blackleg control strategies. Fields that are highly infected indicate that action should be taken to reduce the risk of blackleg. Highly infected fields indicate that the rotation may be too short, which results in the build up of large amounts of infected residue. It may also indicate that the resistance carried by the variety or varieties grown has broken down and different varieties should be selected in future years.



Figure 10. Hail damaged stems and upper stem from which *Leptosphaeria maculans*, as well as other fungal species were isolated. Photo credit: H.R. Kutcher.



Figure 11. Phenoxy herbicide damaged canola plants that may be at increased risk of infection by *Leptosphaeria maculans*. Photo credit: H.R. Kutcher.



Figure 12. Blackleg symptoms in association with root maggot damaged canola plants. Photo credit: H.R. Kutcher.

Resistance. Resistance to *L. maculans* in *Brassica* spp. is of two types: quantitative or adult plant resistance and qualitative or race-specific resistance¹⁷. Quantitative resistance is expressed at the adult plant stage as reduced development of necrotic tissue at the stem base compared to that found in susceptible varieties, and is believed to be due to many genes, each with a relatively small effect. Qualitative resistance in *Brassica* spp. is usually effective at the site of initial infection on the cotyledons and leaves and is controlled by race-specific resistance genes. A resistant response is presumed to result from recognition of the corresponding *L. maculans* avirulence genes in a gene-for-gene manner¹⁸. In Canada, all commonly grown varieties of *B. napus* canola and high erucic acid rapeseed carry moderate to high resistance to *L. maculans*, although the type of resistance or the specific genes for resistance present in a variety are not generally known^{19,20}.

Deployment of genetic resistance in canola varieties against *L. maculans* has been a successful control strategy in western Canada and in many other canola or oilseed rape growing regions of the world. A number of instances where resistance has failed however, indicates that exclusive reliance on this strategy is not sustainable^{21,22,23}. Reports in the literature identify as many as 14 specific resistance genes to *L. maculans* in *Brassica* spp.²⁴, some of which are carried by varieties of *B. napus* from France, Australia and Canada^{25,26}. Unfortunately, surveys in Europe^{27,28}, Canada²⁹ and analysis of Australian isolates of the pathogen³⁰ have identified races of *L. maculans* with the ability to overcome most of these resistance genes.

An understanding of the frequency of individual avirulence alleles present in the pathogen population can provide insights into the corresponding resistance genes present in commercially grown varieties, including an indication of their potential durability for blackleg control. Races of *L. maculans* can be used to identify specific resistance genes in varieties or breeding material of *B. napus* and other *Brassica* spp. Knowledge of host resistance genes and pathogen

avirulence genes is beneficial to the development of blackleg disease resistance strategies that might prolong the effectiveness of specific resistance genes.

Cultivation of varieties that employ resistance from various sources over the landscape (spatial diversity) as well as on a particular field (temporal diversity) reduces the risk of selection of virulent races of the pathogen, compared with cultivation of a single variety or a number of varieties of canola, all carrying the same gene(s) for resistance. For example, the most appropriate canola varieties could be chosen for production in each of the various regions of the prairies, or even provide a basis for on-farm rotation of varieties. Similarly, knowledge of resistance genes in varieties and of the pathogen population structure might provide a basis for the use of variety mixtures. Each variety would carry different specific resistance genes. The rationale is that resistance due to a number of different specific resistance genes will slow the evolution of pathogen virulence on any single resistance gene in the host³¹. More acceptable to growers and industry might be use of multilines (near-isogenic lines), identical for all traits, but varying for specific resistance genes to *L. maculans*. Compared with variety mixtures, multilines would be uniform for other plant characteristics, such as maturity and the quality characteristics of the harvested product.

Better information on specific resistance genes in the host and avirulence genes in the pathogen would also be used in breeding programs aimed at blackleg resistance. An effective specific resistance gene might be incorporated into many contemporary varieties, but substituted for another when the pathogen population is observed to shift significantly to isolates which can overcome the original resistance gene. In addition, new sources of specific resistance carried by *Brassica* spp. may be identified using combinations of races that overcome all currently characterized specific resistance genes. It should be possible to combine specific gene resistance genes (gene pyramiding) in a single variety or combine specific resistance genes with a source of quantitative resistance. Marker assisted selection could speed up the identification of key resistance genes in order to transfer or pyramid genes into varieties³². Effective use of specific resistance, either alone or in a background of quantitative resistance, will require that the pathogen population be monitored at regular intervals. An understanding of the population structure will identify races with the ability to overcome current specific resistance genes before a significant proportion of the crop becomes vulnerable.

Agronomic practices. It is possible under western Canadian conditions for *L. maculans* to survive for a number of years on the residue of an infected canola crop. It is on this residue that the sexual stage of *L. maculans* can occur and give rise to wind borne sexual spores, the ascospores. The production of ascospores allows for possible generation of new pathogen races, and because ascospores are airborne, for their long-distance dispersal. Reducing the impact of ascospore movement between fields may be accomplished by maintaining an effective buffer distance between future canola fields and fields containing infected residue. In the management of blackleg disease, it is recommended that canola crops in western Canadian should be planted at least 50 to 100 m from each other³³. However, in Australia it has been shown that canola plants grown within 400 metres of a field containing infected residue are at increased risk of infection compared with those grown at a distance of greater than 400 metres from the source of infection³⁴. Adhering to a buffer distance is a strongly recommended practice in Australia, but does not appear to be a significant consideration of canola producers in western Canada. A better understanding of appropriate buffer distances and their benefits under western Canadian conditions could improve adoption by growers to help avoid significant infection in the future.

Crop rotation is a fundamental control strategy for many plant pests, especially diseases. The major benefit of a crop rotation is to impact the growth, survival and reproduction of the causal organism and thereby reduce the pathogen population to a level low enough that significant crop damage does not occur. Growing canola in a four-year rotation, i.e., one canola crop on the same field only once every four years, has been a recommended strategy to deal with various diseases of canola, particularly blackleg¹⁴. Studies in Manitoba indicated that a four-year rotation, coupled with conventional tillage provided the greatest reduction in blackleg disease severity³⁵ and reduced the number of ascospores and pycnidiospores of *L. maculans* compared to more intensive canola rotations³⁶. These studies indicated that an appropriate combination of rotation and tillage can lower the amount of airborne inoculum and reduce the level of *L. maculans* infection.

By contrast, other research has suggested that tillage and burial of infested canola residues do not necessarily decrease the risk of blackleg^{37,38,39}. Traditionally, tillage has been recommended to bury infested residue and thereby reduce the amount of inoculum of the pathogen at the soil surface. However, during the mid 1970's to the late 1980's when blackleg prevalence and severity increased dramatically in Saskatchewan, Manitoba, and east-central Alberta, typical practices for seedbed preparation would have been characterized as aggressive conventional tillage, and summerfallowing was still common. These general practices did not prevent the spread and establishment of blackleg in the prairie region. Overall, tillage likely will have a lesser impact on blackleg risk compared with the rotation intervals used, variety grown and seasonal weather conditions. A four-year crop rotation will not eradicate *L. maculans* because the pathogen can survive on crop residues that may persist for longer periods⁵, and on volunteer canola plants or related weed species¹⁴. Effective control of weeds and volunteer canola is an important blackleg management tool, but is often compromised by short canola rotations.

Under continuous monoculture or rotations too short to allow infested canola residue to fully decompose, *L. maculans* can complete and repeat its lifecycle, resulting in its perpetuation and multiplication. Growing a number of crop species in a rotation, such as wheat, barley, field pea, flax and forages⁴⁰, and alternating the varieties of each crop grown⁴¹, as these may carry different sources of resistance, interrupts the lifecycle of the pathogen. Recent high canola crop prices relative to other crops have encouraged the frequent production of canola in the rotation, despite the fact that research indicates that greater crop diversification results in increased overall productivity^{42,43}. This trend was facilitated by the development of herbicide tolerant canola with a high level of resistance to blackleg. Increasing the time interval between canola crops has been shown to effectively reduce the severity of blackleg infection in both susceptible and resistant varieties of canola when compared to continuous production of canola⁴³, Figure 13. The reduction in disease severity was not as great when a resistant variety was cultivated, although it is still significantly less under a four-year rotation compared to continuous canola production. Over time, repeated use of short rotations results in increased amounts of *L. maculans* infested canola residue, and selection for pathogen races that can overcome the resistance genes in the varieties grown. Since the pathogen is harbored on previous canola residue, a rotation of four-years or more allows much of this residue to decompose before canola is grown again. The use of a sufficiently long rotation to allow infested canola residue to degrade is crucial to maintain the resistance present in canola varieties.

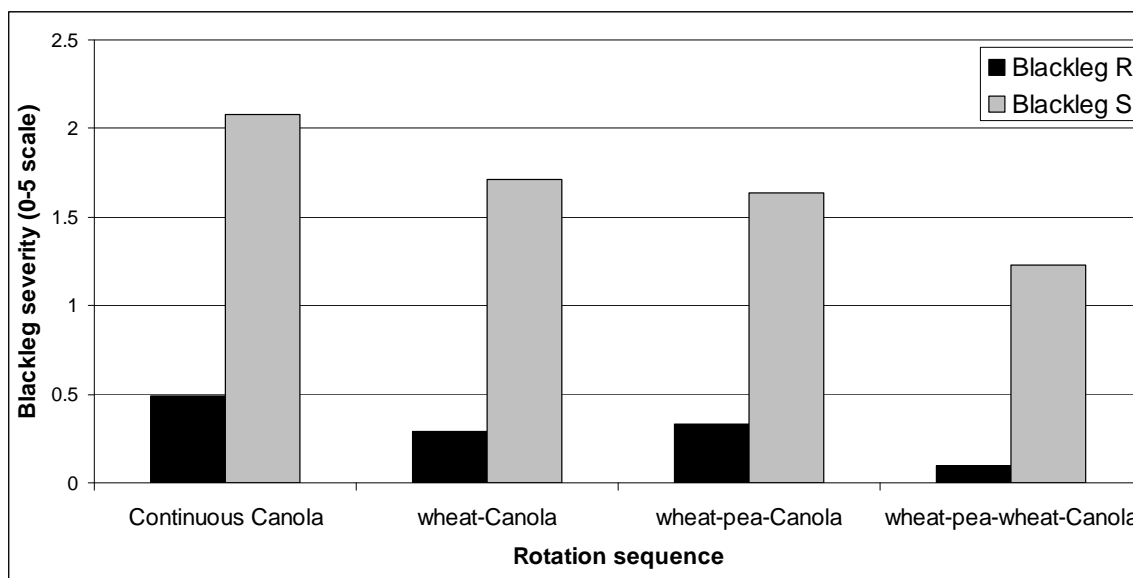


Figure 13. Severity of blackleg symptoms from rotation studies conducted at Melfort and Scott, SK from 2000 to 2006 (means calculated from 13 site years). Resistant (Invigor hybrids) and susceptible ('Westar') varieties were grown continuously or every 2nd, 3rd and 4th year with wheat and field pea as rotation crops.

Seeding certified, fungicide- and insecticide-treated seed has become the norm in western Canada, particularly since the adoption of herbicide tolerant and hybrid varieties. This is an excellent practice to prevent the spread of new virulent races of *L. maculans* across the prairies. Growers should also be aware of a relationship between high nitrogen use and the disease. There is some evidence to suggest high fertility increases the risk of the disease⁴⁴, making use of effective management practices even more important for crops under high fertility.

Foliar Fungicides. Blackleg control using a triazole fungicide (propiconazole, Tilt®, Bumper®, Pivot®, Propel®) was available to western Canadian canola growers in the early 1990's, the first strobilurin (azoxystrobin, Quadris®) in the early 2000's and a second (pyraclostrobin, Headline®) in 2010. These products have been shown to reduce symptoms of the disease and contribute to increased yield in blackleg susceptible canola varieties⁴³. However, with the combination of good blackleg resistance in canola varieties since the early 1990's and adherence to adequate rotation lengths, growers generally have not found an economic benefit from foliar fungicide use. This was verified by research on the use of fungicides to control blackleg, which did not indicate a yield benefit for resistant varieties under four-year rotations^{45,46}. However, anecdotal reports from some canola growers indicate that blackleg is again a significant production issue and growers have begun to use foliar fungicide as an additional control measure.

The difficulty of achieving a consistent response to fungicide, even in high risk situations (susceptible variety in short rotation) may be due to the long infectivity period in canola. Foliar infection by *L. maculans* can occur from the cotyledon stage to bolting, and even to flowering of the crop, resulting in significant stem cankering by maturity. A single application of fungicide may not be sufficient to protect the plant during this lengthy period. Further research should be conducted to investigate the usefulness of multiple applications of fungicide during the period of greatest risk. Early detection of airborne *L. maculans* spores might be of value in western Canada, as it has been in Poland, to develop an early warning system to determine the optimum timing of fungicide application⁴⁷. Seasonal and diurnal patterns of spore dispersal were associated with crop stage and weather factors in western Canada, which should be studied further to guide fungicide application³³. Weather-based models have been developed to predict the date of ascospore release by *L. maculans* in Australia⁴⁸. In western Canada, total rainfall per week was significantly correlated to disease severity on plants infected at the cotyledon, three-leaf, and six-leaf stages, while average maximum weekly temperature was correlated only with plants infected at the cotyledon and six-leaf stages⁴⁹. These results established a correlation between disease incidence and rainfall for plants infected at all three growth stages and a nonlinear model was developed to evaluate the combined effects of rainfall and temperature on disease severity for western Canada⁴⁹. Further research resulting in more years of data would aid in testing these models and modifying them to predict disease epidemics more precisely in western Canada.

Concluding Remarks

Managing blackleg disease of canola in western Canada will require an integrated approach that combines some or all of the strategies discussed. Reliance on any one strategy alone, such as genetic resistance, is not likely to be a viable long-term strategy. The durability of specific resistance genes depends largely on the amount of the pathogen present in the environment, which is directly related to the amount of diseased *B. napus* residue. Combining genetic resistance with a sustainable crop rotation, isolation of canola from previously infected residue, control of volunteers and weeds and use of fungicide treated seed has, and should continue to provide control of the disease without resorting to foliar fungicides. However, foliar fungicides remain an option within the continuum of integrated disease management practices, if required. Control of blackleg achieved with fungicides might be improved through further research. In the long-term, research and extension efforts are needed to increase the attractiveness of other crop options for western Canadian farmers. Factors such as increased commodity prices for cereals and pulses relative to canola, lower production costs compared with canola and the nitrogen fixing ability of pulses may help to convince farmers to increase the frequency of non-canola crops in their rotations, thus lessening the risk of blackleg and potentially improving productivity and profitability in the canola phase of the rotation.

References

1. Fitt, B.D.L., H. Brun, M.J. Barbetti, and S.R. Rimmer. 2006. Worldwide importance of phoma stem canker (*Leptosphaeria maculans* and *L. biglobosa*) on oilseed rape (*Brassica napus*). *Eur. J. Plant Pathol.* 114:3-15.
2. Cunningham, G.H. 1927. Dry-rot of swedes and turnips: its cause and control. *N-Z. Dep. Agric. Bull.* 133.
3. Petrie, G.A. 1978. Occurrence of a highly virulent strain of blackleg (*Leptosphaeria maculans*) on rape in Saskatchewan (1975-77). *Can. Plant Dis. Surv.* 58:21-25.
4. Mendes-Pereira, E., M.H. Balesdent, H. Brun, and T. Rouxel. 2003. Molecular phylogeny of the *Leptosphaeria maculans*-*L. biglobosa* species complex. *Mycol. Res.* 107:1287-1304.
5. Petrie, G.A. 1995. Long-term survival and sporulation of *Leptosphaeria maculans* (blackleg) on naturally-infected rapeseed/canola stubble in Saskatchewan. *Can. Plant Dis. Surv.* 75:23-34.
6. Vanterpool, T.C. 1961. Rape diseases in Saskatchewan in 1961. *Can. Plant Dis. Surv.* 41:372-373.
7. Gugel, R.K., and G.A. Petrie. 1992. History, occurrence, impact, and control of blackleg of rapeseed. *Can. J. Plant Pathol.* 14:36-45.
8. Martens, J.W., W.L. Seaman, and T.G. Atkinson. 1988. Diseases of Field Crops in Canada. Pages 120-121. Canadian Phytopathological Society.
9. Anonymous. 1993. Varieties of grain crops for Saskatchewan 1993. Saskatchewan Agriculture and Food.
10. Chen, Y., and W.G.D. Fernando. 2006. Prevalence of pathogenicity groups of *Leptosphaeria maculans* in western Canada and North Dakota, USA. *Can. J. Plant Pathol.* 28:533-539.
11. Fernando, W.G.D., and Y. Chen. 2003. First Report on the presence of *Leptosphaeria maculans* Pathogenicity Group-3, causal agent of Blackleg of Canola in Manitoba. *Plant Dis.* 87:1268.
12. Keri, M., H.R. Kutcher, and S.R. Rimmer. 2001. Virulence of isolates of *Leptosphaeria maculans* from western Canada on *Brassica napus* differentials. *Can. J. Plant Pathol.* 23:199.
13. Kutcher, H.R., M. Keri, D.L. McLaren, and S.R. Rimmer, 2007. Pathogenicity of *Leptosphaeria maculans* in western Canada. *Can. J. Plant Pathol.* 29:388-393.
14. Bailey, K.L., B.D. Gossen, R.K. Gugel, and R.A.A. Morrall. 2003. Diseases of field crops in Canada. 3rd Ed. The Canadian Phytopathological Society, University Extension Press, University of Saskatchewan.
15. Harper, F.R., and B. Berkenkamp. 1975. Revised growth-stage key for *Brassica campestris* and *B. napus*. *Can. J. Plant Sci.* 55:657-658.
16. Lancashire, P.D., H. Bleiholder, T. Van den Boom, P. Langeluddeke, R. Strauss, E. Weber, and A. Witzemberger. 1991. A uniform decimal code for growth stage of crops and weeds. *Ann. Appl. Biol.* 119:561-601.
17. Delourme, R., A.M. Chèvre, H. Brun, T. Rouxel, M.H. Balesdent, J.S. Dias, P. Salisbury, M. Renard, and S.R. Rimmer. 2006. Major gene and polygenic resistance to *Leptosphaeria maculans* in oilseed rape (*Brassica napus*). *Eur. J. Plant Pathol.* 114:41-52.
18. Ansan-Melayah, D., M.H. Balesdent, R. Delourme, M.L. Pilet, X. Tanguy, M. Renard, and T. Rouxel. 1998. Genes for race-specific resistance against blackleg disease in *Brassica napus* L. *Plant Breeding* 117:373-378.
19. McVetty, P.B.E., W.G.D. Fernando, G. Li, M. Tahir, and C.D. Zelmer. 2010. Red River 1997 Roundup ReadyTM high erucic acid, low glucosinolate summer rape. *Can. J. Plant Sci.* 90:711-713.
20. Rimmer, S.R. 2006. Resistance genes to *Leptosphaeria maculans* in *Brassica napus*. *Can. J. Plant Pathol.* 28:S288-S297.
21. Brun H., S. Levivier, D. Ruer, I. Somda, A.M. Chèvre, and M. Renard. 2000. A field method for evaluating the potential durability of new resistance sources: application to the *Leptosphaeria maculans*/*Brassica napus* pathosystem. *Phytopathol.* 90:961-966.
22. Li, H., K. Sivasithamparam, and M.J. Barbetti. 2003. Breakdown of a *Brassica rapa* subsp. *sylvestris* single dominant blackleg resistance gene in *B. napus* rapeseed by *Leptosphaeria maculans* field isolates in Australia. *Plant Dis.* 87:752 (abstract).
23. Rouxel, T., A. Penaud, X. Pinochet, H. Brun, L. Gout, R. Delourme, J. Schmit, and M.H. Balesdent. 2003a. A 10-year survey of populations of *Leptosphaeria maculans* in France indicates a rapid adaptation towards the *Rlm1* resistance gene of oilseed rape. *Eur. J. Plant Pathol.* 109:871-881.
24. Kutcher, H.R., F. Yu, and H. Brun. 2010a. Improving blackleg disease management of *Brassica napus* from knowledge of genetic interactions with *Leptosphaeria maculans*. *Can. J. Plant Pathol.* 31:29-34.
25. Kutcher, H.R., S.R. Rimmer, M.H. Balesdent, T. Rouxel, and H. Brun. 2008. Detection of specific resistance genes against *Leptosphaeria maculans* in *Brassica napus*. *Can. J. Plant Pathol.* 30:386 (abstract).
26. Rouxel, T., E. Willner, L. Coudard, and M.H. Balesdent. 2003b. Screening and identification of resistance to *Leptosphaeria maculans* (stem canker) in *Brassica napus* accessions. *Euphytica* 133:219-231.

27. Balesdent, M.H., K. Louvard, X. Pinochet, and T. Rouxel. 2006. A large-scale survey of races of *Leptosphaeria maculans* occurring on oilseed rape in France. *Eur. J. Plant Pathol.* 114: 53-65.
28. Stachowiak, A., J. Olechnowicz, M. Jedryczka, T. Rouxel, M.H. Balesdent, I. Happstadius, P. Gladders, A. Latunde-Dada, and N. Evans. 2006. Frequency of avirulence alleles in field populations of *Leptosphaeria maculans* in Europe. *Eur. J. Plant Pathol.* 114:67-75.
29. Kutcher, H.R., M.H. Balesdent, S.R. Rimmer, T. Rouxel, A.M. Chevre, R. Delourme, and H. Brun. 2010b. Frequency of avirulence genes among isolates of *Leptosphaeria maculans* in western Canada. *Can. J. Plant Pathol.* 31:77-85.
30. Balesdent, M.H., M.J. Barbetti, H. Li, K. Sivasithamparam, L. Gout, and T. Rouxel. 2005. Analysis of *Leptosphaeria maculans* races structure in a world-wide collection of isolates. *Phytopathol.* 95:1061-1071.
31. Mundt, C.C. 2002. Use of multiline cultivars and cultivar mixtures for disease management. *Annu. Rev. Phytopathol.* 40:381-410.
32. Dusabanyagasani, M., and W.G.D. Fernando. 2008. Development of a SCAR marker to track canola resistance against blackleg caused by *Leptosphaeria maculans* pathogenicity group 3. *Plant Dis.* 92:903-908.
33. Guo, X.W., and W.G.D. Fernando. 2005. Seasonal and diurnal patterns of spore dispersal by *Leptosphaeria maculans* from canola stubble in relation to environmental conditions. *Plant Dis.* 89:97-104.
34. Marcroft, S.J., S.J. Sprague, S.J. Pymer, P.A. Salisbury, and B.J. Howlett. 2004. Crop isolation, not extended rotation length, reduces blackleg (*Leptosphaeria maculans*) severity of canola (*Brassica napus*) in south-eastern Australia. *Aust. J. Exper. Agr.* 44:601-606.
35. Guo, X.W., W.G.D. Fernando, and M.H. Entz. 2005. Effects of crop rotation and tillage on the blackleg disease of canola. *Can. J. Plant Pathol.* 27:53-57.
36. Guo, X.W., W.G.D. Fernando, and M.H. Entz. 2008. Dynamics of infection by *Leptosphaeria maculans* on canola as influenced by crop rotation and tillage. *Arch. Phyto. Pl. Protec.* 41:57-66.
37. Kutcher, H.R., and S.S. Malhi. 2010. Residue burning and tillage effects on diseases and yield of barley (*Hordeum vulgare*) and canola (*Brassica napus*). *Soil and Till. Res.* 109:153-160.
38. Lange, R.M, P.D. Kharbanda, and M.J. Ostaszewski. 1996. Effect of tillage and cultivar on several important canola diseases in north-western Alberta: preliminary results. In Proceedings of the Seventeenth Annual Meeting, Plant Pathology Society of Alberta, November 4-6, 1996, Lacombe, Alta. Edited by D.D. Orr. Plant Pathology Society of Alberta, Lacombe, AB.
39. Turkington, T.K., G.W. Clayton, H. Klein-Gebbinck, and D.L. Woods. 2000. Residue decomposition and blackleg of canola: influence of tillage practices. *Can. J. Plant Pathol.* 22:150-154.
40. Karlen, D.L., G.E. Varvel, D.G. Bullock, and R.M. Cruse. 1994. Crop rotations for the 21st century. *Advances in Agronomy* 53:1-45.
41. Turkington, T.K., K. Xi, J.P. Tewari, H.K. Lee, G.W. Clayton, and K.N. Harker. 2005. Cultivar rotation as a strategy to reduce leaf diseases under barley monoculture. *Can. J. Plant Pathol.* 27:283-290.
42. Johnston, A.M., H.R. Kutcher, and Bailey, K.L. 2005. Impact of crop sequence decisions in the Saskatchewan Parkland. *Can. J. Plant Sci.* 85:95-102.
43. Kutcher, H.R., and S.A. Brandt. 2008. Optimizing Canola Production: Pest Implications of Intensive Canola Rotations. Final Canola Agronomic Research Program Report to the Canola Council of Canada. 39 pp.
44. Kutcher, H.R., S.S. Malhi, and K.S. Gill. 2005. Topography and management of nitrogen and fungicide affects diseases and productivity of canola. *Agron. J.* 97:533-541.
45. Bailey, K. L., A. M. Johnston, H. R. Kutcher, B. D. Gossen, and R. A. A. Morrall. 2000. Managing crop losses from foliar diseases with fungicides, rotation, and tillage in the Saskatchewan Parkland. *Can. J. Plant Sci.* 80:169–175.
46. Kutcher, H.R., D. Ulrich, and S.A. Brandt. 2003. Plant disease implications of intensive crop rotations of canola and field pea in western Canada. In "Proceedings of the symposium, Dynamic Cropping Systems: Principles, Processes and Challenges" pages 273-276, August 4-7, 2003, Radisson Inn, Bismarck, ND.
47. Jedryczka, M., J. Kaczmarek, A. Dawidziuk, and A. Brachaczek. 2008. System for forecasting disease epidemics – aerobiological methods in Polish agriculture. *Asp. Appl. Biol.* 89:65-70.
48. Salam, M.U., B.D.L. Fitt, J.N. Aubertot, A.J. Diggle, Y.J. Huang, M.J. Barbetti, P. Gladders, M. Jedryczka, R.K. Khangura, N. Wratten, W.G.D. Fernando, A. Penaud, X. Pinochet, and K. Sivasithamparam. 2007. Two weather-based models for predicting the onset of seasonal release of ascospores of *Leptosphaeria maculans* or *L. biglobosa*. *Plant Pathol.* 56:412-423.
49. Ghanbarnia, K., W. G. D. Fernando, and G. Crow. 2009. Developing rainfall- and temperature-based models to describe infection of canola under field conditions caused by pycnidiospores of *Leptosphaeria maculans*. *Phytopathol.* 99:879-886.