DISEASES OF CANOLA

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SUMMARY

- Blackleg is the major disease of canola. The Mediterranean-type environment in canola growing regions of Australia is conducive to disease spread.
- Blackleg isolates appear to be more virulent than those in other countries and Australian canola varieties are the most blackleg-resistant spring varieties in the world.
- Other diseases are observed on canola in Australia, but with the exception of damping off and *Sclerotinia* stem rot, they seldom have an impact on yield.

INTRODUCTION

In Australia, as in other countries, blackleg caused by the fungus *Leptosphaeria maculans* is the major disease of canola, *Brassica napus*. In some years symptoms caused by other fungi are apparent; for example, *Sclerotinia* stem rot and *Rhizoctonia* seedling wilt, whilst there is very little incidence of bacterial diseases. This chapter discusses current research on canola diseases in Australia, particularly blackleg, including its epidemiology, pathogen variability and control strategies.

BLACKLEG DISEASE

Rapeseed production began in Australia in the late 1960s with varieties introduced from Canada. These varieties (eg. Target, Oro, Span), however, proved to be highly susceptible to blackleg and by 1972, severe blackleg disease epidemics across Australia posed a major threat to the industry. As a result, the area sown to rapeseed plummeted and did not increase significantly for a decade. In Western Australia the area planted increased dramatically before the epidemic (from 120 hectares in 1969 to 49,000 ha in 1972); whilst in 1973 and 1974 the area planted was 3,200 and 3000 ha, respectively. Since then the introduction of blackleg-resistant canola varieties and improved management practices have led to the resurgence of the industry nationwide. However, with the dramatic increase in area sown to canola recently, there are concerns about the threat of blackleg to further expansion of the canola industry in Australia.

The main source of infection in Australia arises from sexual spores (ascospores) produced in fruiting bodies (pseudothecia) on infested canola stubble during summer. Ascospores are distributed by wind up to eight kilometres distant and their release is generally highest during May to August which coincides with the sowing period in Australia. Severe infection can lead to seedling death, but cankering may occur at any plant growth stage. Stem cankering is the major cause of yield loss associated with blackleg. Lesions may also form on pods allowing the fungus to spread to the seed. Seed infection appears to be of minor importance in causing epidemics, but may be responsible for introducing the disease to new areas. Certified seed in Australia is not routinely tested for blackleg infestation. The Mediterranean-type environment in Australia favours disease carryover on residues,

encourages epidemics of residue-borne pathogens, aligns ascospore showers to seedling emergence and maximises the disease impact on yield as the moisture supply dwindles at the end of the season.

Disease control

As in other countries, growing resistant varieties is the best control strategy for blackleg in Australia. Farmers are also encouraged to plant canola on the same area a maximum of only once every four years. However, the recent excellent financial returns on canola has resulted in some farmers opting for tighter rotations often whereby canola is planted every second year in a wheat-canola rotation. This is of major concern, particularly given the increased area of canola sown and the resultant large amount of infected stubble. Although planting canola as far away as possible from previous canola crops reduces the risk of ascospore infection, this practice is now less effective due to the increased area of canola production; for instance in Western Australia there are now 1 million ha of infected residues in canola production areas. Destruction of stubble by burning or burying is a common practice to reduce carry-over of *L. maculans*. The most effective chemical control of blackleg has been achieved with the fungicide flutriafol (Impact-in-Furrow), a flowable formulation for blending with fertiliser. This chemical is being currently used on a wide scale in Western Australia to supplement varietal resistance in areas prone to severe blackleg infection. A wide range of potential alternative fungicidal seed treatments, foliar sprays and fertiliser treatments are being evaluated by Khangura and Barbetti. Other control measures under development include genetically engineering canola varieties with anti-fungal proteins. This strategy is being pursued by Kazan and Manners (Queensland) and Spangenberg (Victoria).

Pathogenic variability of *L. maculans*

Worldwide there are two strains of *L. maculans*, aggressive and non-aggressive, based on their ability to cause stem cankers on canola. These strains appear to be different species that look similar under the microscope. Only the aggressive isolates are present in Australia and they appear to be more virulent than those from other countries. This is indicated by the finding that canola varieties with good resistance in their countries of origin, are susceptible in Australian blackleg nurseries where canola stubble was present from the previous year's crop. Furthermore Australian isolates have been identified that, as well as attacking canola, attack Indian mustard (*B. juncea*), a species that previously has been resistant to blackleg. Such isolates are not common in other countries. This increased virulence of Australian blackleg isolates and severity of disease compared to other countries may be because canola is grown in temperate climates in Australia favourable to outcrossing of the fungus. Outcrossing leads to a large number of ascospores as primary inoculum for next years crop and a high degree of sexual recombination, which may enable the fungus to adapt to selection pressures and eventually to overcome disease resistance genes in canola. In Canada and Europe, harsher environmental conditions such as snow cover delay the maturity and release of sexual spores from stubble, which means that less outcrossing and subsequent recombination occurs. Also infection of new crops often occurs too late to damage the crop significantly.

Both race-specific and non-specific resistance to blackleg have been identified by Ballinger and Salisbury by *Brassica* differentials in response to infection by Australian field isolates. Comparison of race specificity designations is difficult due to a lack of standardisation of differentials, virulence testing procedures and the heterogenous nature of lines used as differentials. To avoid heterogeneity of lines, doubled haploid differentials developed by Smithard and Wratten are being tested with blackleg isolates by Weichel and Vanderaa. In contrast to the situation with *B. napus*, the specificity of Australian isolates on Indian mustard (*B. juncea*) is clearcut; isolates can be divided into two groups - those that form stem and cotyledonary lesions on all lines, and those that do not. All isolates tested in these experiments caused lesions on cotyledons of *B. napus* cultivars Westar, Glacier and Quinta, suggesting that they are in Pathogenicity Group 4, in contrast to isolates in other countries which belong to Pathogenicity Groups 2, 3 and 4.

Recently genetic variation of Australian isolates has been examined using Amplified Fragment Length Polymorphic (AFLP) markers by Purwantara and Barrins. Each isolate had a unique genetic profile (fingerprint) and the isolates showed a high degree of genetic diversity, reflecting the fact that the fungus outcrosses prolifically in the field. Chromosome size of blackleg isolates was also analysed and, like the AFLP fingerprints, was unique for each isolate. A preliminary genetic map of the fungus has been prepared using AFLP markers to examine F_1 progeny of a cross between an isolate that attacks *B. juncea* and one that cannot. A locus conferring the ability to attack *B. juncea* is being mapped by Cozijnsen and Popa. The development of this map means that individual genes can now be pinpointed to *L. maculans* chromosomes.

The population structure and virulence of blackleg isolates across Australia is currently being examined using molecular markers by Barrins, Purwantara and Popa. Such knowledge is essential for plant breeders to develop more rational strategies with disease resistance genes. Hierarchical sampling of field populations is currently underway whereby genetic diversity is being compared between pycnidia within a lesion, lesions on a leaf, leaves in a paddock and plants in different canola growing regions of Australia.

Blackleg disease resistance genes

The main sources of blackleg resistance used in Australian public breeding programs are Japanese spring varieties and French winter varieties. Australian canola varieties are the most blackleg-resistant spring varieties in the world, but as yet little is known about the genetic control of resistance in these lines. Alternative forms of resistance are being sought by Australian researchers and breeders, including in B genome species (Marcroft), wild crucifer species (Salisbury and Howlett) and using anti-fungal proteins (Kazan and Manners, Spangenberg).

OTHER CANOLA DISEASES

The next two most important diseases in Australia are *Sclerotinia* stem rot and damping off. Other diseases such as white rust, *Alternaria* black spot and white leaf spot only rarely cause yield losses.

Sclerotinia stem rot

In some countries *Sclerotinia* stem rot caused by the fungus *Sclerotinia sclerotiorum* is the most important disease of canola. It is widespread in Australia, but generally not severe. Hind, Ash and Murray are looking at the feasibility of forecasting *Sclerotinia* stem rot by surveying petal infection. In a survey of canola in NSW in the 1998 growing season, they found that up to 80% of petals of canola were infected in individual crops, whilst at the end of the season 30% of stems of the same plants were infected. The effect of fungicides and the impact of stubble retention of the previous crop on sclerotial survival and germination are also being evaluated.

Damping-off

Since 1994 there has been a significant increase in the extent of hypocotyl rot and damping-off associated with establishment of canola seedlings in Western Australia. *Rhizoctonia* spp. were associated with these conditions. Isolates of ZG5 (Anastomosis Group 2-1) and ZG1-1 (Anastomosis Group 8) were highly pathogenic on canola, delayed seedling emergence and caused severe hypocotyl or root rot, respectively. Isolates of ZG5 also induced post emergence damping-off. Pathogenicity tests on a range of rotational crops and two weed species revealed that ZG5 was highly pathogenic on *B. juncea*, mildly pathogenic on narrow-leafed lupin and clover, but failed to infect any of the cereal hosts tested. In contrast, all the crops tested were highly susceptible to ZG1-1, except *B. juncea* which was only moderately susceptible. Damping-off is also caused by other common soil inhabitants such as *Pythium* spp. and *Fusarium* spp., for which no control measures are currently available. However, useful control of *Rhizoctonia* can be obtained using an iprodione fungicidal seed treatment. This work is being carried out by Khangura and Barbetti.

Alternaria spot

Leaf infection by *Alternaria* commonly occurs on canola grown in medium to high rainfall areas in Australia. This can lead to stem and pod infection during favourable conditions in spring. Pod infection may also result in infected seed, which, when sown without an appropriate seed dressing, causes seedling death.

Downy mildew

Downy mildew, caused by *Peronospora parasitica,* occurs sporadically and until recently rarely caused any yield loss. However, in 1998, severe downy mildew was widespread in Western Australia on seedlings, and in some areas appeared to retard seedling growth and vigour severely. Control measures have not been warranted in Western Australia prior to 1998 but potential control measures will now be investigated.

Viral diseases

A survey of 150 canola crops in Western Australia by Jones during 1998 revealed the presence of Beet Western Yellows Virus in more than two thirds of the crops

surveyed and Cauliflower Mosaic Virus in one third of the crops. Aphids spread these viruses. In some crops more than 60% of plants were infected. During July of this year Beet Western Yellows Virus has been also detected in New South Wales. The impact and importance of these two viruses have yet to be determined fully in Australia. However, overseas they cause both seed yield losses and diminished oil content.

FUTURE DIRECTIONS

The ability to control diseases, particularly blackleg, is crucial for the long term success of the Australian canola industry. Strategies that will continue to be important for disease control include improved host resistance, stubble management, four year rotations, cheaper and more effective fungicide treatments. Breeding for resistance will benefit from a better understanding of the host-pathogen interactions between *Brassica* species and *L. maculans*. Knowledge of the number and nature of genes involved in resistance in current lines, and the development of techniques such as molecular markers, will enhance the effectiveness of selection for blackleg resistance. This will be facilitated by the development of doubled haploid lines with uniform reaction to blackleg. Likewise, a better understanding is required of the pathogenic variability within *L. maculans* and its propensity for change.

FURTHER READING

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