1º Simpósio Latino Americano de Canola
19 a 21 de agosto de 2014
Passo Fundo, RS, Brasil

BLACKLEG RESISTANCE IN CANOLA, ITS BREAKDOWN AND LATEST STRATEGIES BEING ADOPTED IN AUSTRALIA TO MANAGE THE DISEASE.

Andrew Easton

Crop Research Lead – Brassicas. Pacific Seeds, Toowoomba Qld Australia.

ABSTRACT

Blackleg, caused by the fungus *Leptosphaeria maculans,* is the most serious disease of Canola in Australia. The disease is spread by ascospores released from fruiting bodies on old crop stubble in autumn and winter. The pathogen grows within the vascular tissue where it gradually causes necrosis within the stem at the base of the plant, leading plants to wilt and to die from flowering onwards. High volumes of Canola stubble present laying on soil surface lead to extremely large populations of genetically diverse wind-borne ascospores. If the same resistant Canola variety is grown for several years in succession, the pathogen population is able to adapt to the selection pressure and overcome genetic resistance. Growing Canola immediately adjacent to last year's crop creates the highest ascospore load. Two types of genetic resistance have been identified, which are categorised as either seedling or adult plant resistance. Seedling resistance is conferred by single dominant genes. Adult plant resistance is conferred by numerous genes which behave quantitatively. The 2014 Canola Blackleg Management Guide instructs growers how to monitor Blackleg severity and details environmental and management factors influencing Blackleg risk. Seedling resistance genes in released varieties are listed. Variety rotation is recommended if growers are in a high risk region or if they have been growing the same cultivar in close proximity for at least three years and have been observing increasing blackleg severity.

INTRODUCTION

Canola production began in Australia in the late 1960s with varieties introduced from Canada. These varieties turned out to be completely susceptible to Blackleg caused by the fungus *Leptosphaeria maculans* and by 1972 severe epidemics had almost destroyed the fledgling industry (Howlett, Ballinger & Barbetti, 1999). It wasn't until the release of locally developed resistant varieties in the mid 1980s that the crop area again began significantly increasing, to reach its current area of approximately 2.5 million ha.

Today, even with the availability of highly resistant varieties, Blackleg is still the most serious disease of Canola in Australia. The severity of blackleg has risen in recent years due to increased Canola area and intensity of production. Although not common, yield losses of 50 per cent and greater have been recorded in some seasons with up to 90 per cent yield loss occurring in cases where *L. maculans* has overcome major blackleg resistance genes within

certain varieties (Marcroft and Bluett, 2013). Although high levels of genetic resistance are available, reliance on genetic resistance alone is not enough to ensure protection of crops from yield loss or long term sustainability of Canola production.

Blackleg epidemics in South American Canola regions have not yet been as severe as experienced in Australia, but the climatic conditions and the expanding area of Canola production creates the potential for significant crop losses due to Blackleg. This paper reviews factors affecting Blackleg severity and latest management strategies, many of which can be readily applied to in South America.

BLACKLEG DISEASE LIFE CYCLE

The fungus survives over summer as a saprophyte on Canola stubble after harvest (**Figure 1**). During the autumn, sexual spores (ascospores) are produced on fruiting bodies (pseudothecia). Ascospores are distributed by wind up to eight kilometres and their release is generally highest during May to August, which coincides with the sowing and early growth period of Canola in Australia. Ascospores land on young Canola plants, germinate and invade the plant through the stomata. Approximately two to four weeks later, necrotic lesions develop on cotyledons and leaves at the site of initial infection. Severe infection can lead to plant death at the seedling stage.

If the plant survives the seedling stage, the pathogen continues to grow within the vascular tissue. In less resistant plants it gradually causes necrosis within the stem at the base of the plant. Flow of water and nutrients within the plant becomes restricted and from the flowering stage onwards plants begin to wilt and die.

Pycnidia develop on the leaf lesions and produce asexual pycnidiospores. The pycnidiospores are spread during periods of rainfall and only move short distances by water splash movement. If periods of showery weather occur late in the crop development, 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. Stem cankering caused by early ascospore infection is the major cause of yield loss associated with Blackleg.

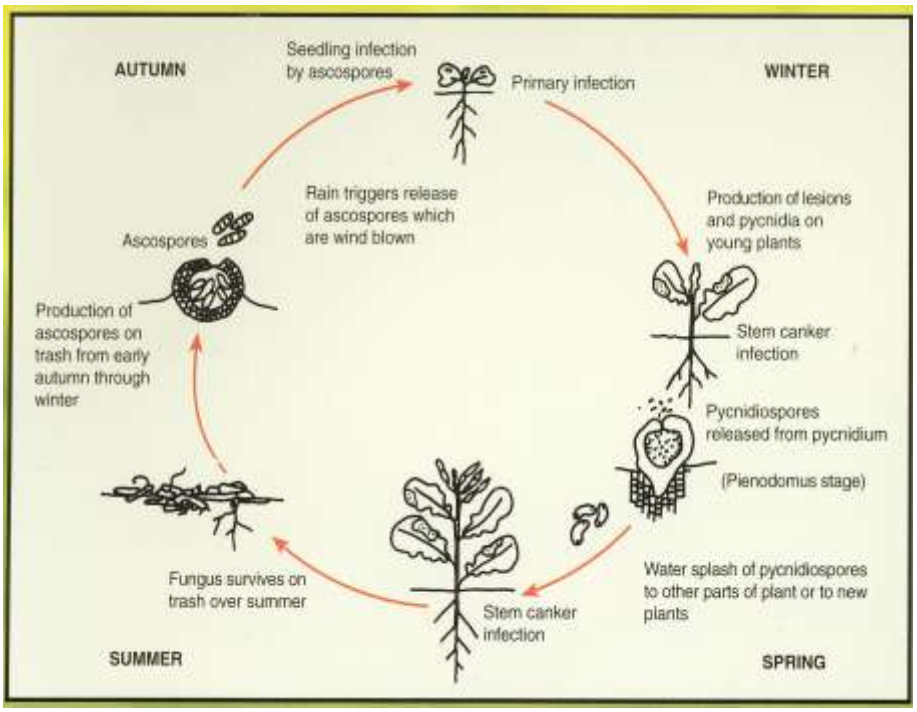

Figure 1. The Blackleg life cycle on Canola (Marcroft, 2007).

The Mediterranean-type environment in southern Australia favours disease carryover on residues, encourages epidemics of residue-borne pathogens, aligns ascospore showers with seedling emergence and maximises the disease impact on yield, as the moisture supply dwindles at the end of the season (Howlett, Ballinger & Barbetti, 1999).

In subtropical Canola production areas of South America, where there is high summer rainfall, stubble decomposition is likely to occur more quickly. The lower autumn and winter rainfall will likely lead to Blackleg incidence and severity being lower.

FACTORS AFFECTING DISEASE SEVERITY

Previous Canola Cropping History

Sexual reproduction occurs during the ascospore production phase and every ascospore potentially has a different combination of virulence genes. This, in combination with the high volumes of Canola stubble present, leads to extremely large populations of genetically diverse wind-borne recombinant ascospores. If the same resistant Canola variety is grown for several years in succession, the pathogen population is able to adapt to the selection pressure and overcome the genetic resistance. The frequency of virulent isolates increases leading to the breakdown of resistance (Howlett, 2014).

Stubble Volume

As previously discussed, the main source of infection is from ascospores produced on Canola stubble. The volume of stubble present therefore strongly influences the potential disease pressure. Stubble breaks down over time and because the volume of stubble is greatest in the first year after harvest it is always the most important source of inoculum (**Figure 2**).

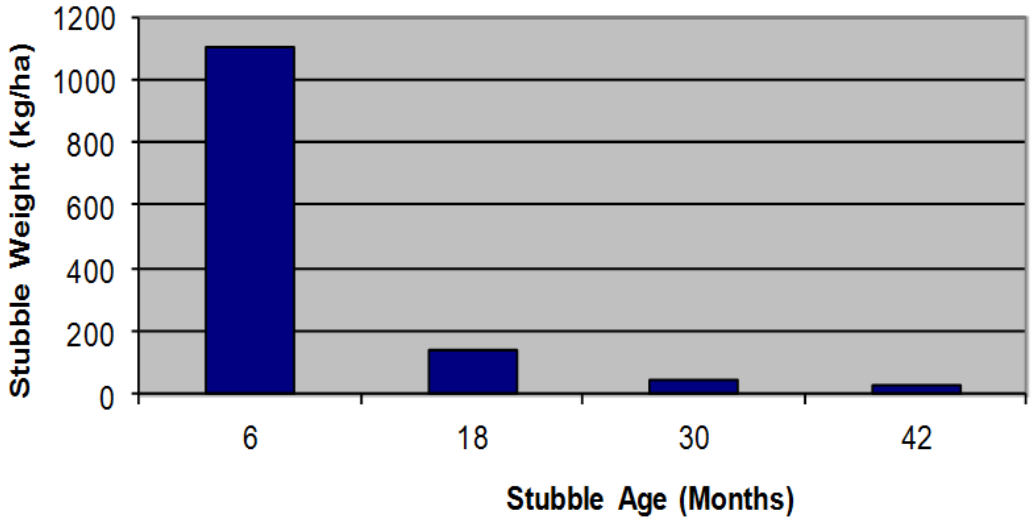

Figure 2. The effect of time on Canola stubble weight (Marcroft, 2002).

Distance from Previous Year's Crop

Ascospores are wind borne and previous studies have shown they can travel for distances of up to eight kilometres (Howlett, Ballinger & Barbetti 1999). Marcroft (2002) assessed the level of Blackleg infection at varying distances from the previous year's stubble using the CSII (Cut Stem Incidence Index) method (**Figure 3**). The CSII is determined by cutting stems at ground level at physiological maturity and assessing the percentage of the stem infected. Disease severity was highest immediately next to stubble from the previous year's crop, decreasing rapidly for 50m, then more gradually until 500m where it reached a plateau. The implication is that growing Canola immediately adjacent to last year's crop will create the highest ascospore load on the new crop. But there is no effect from increasing distance beyond 500m.

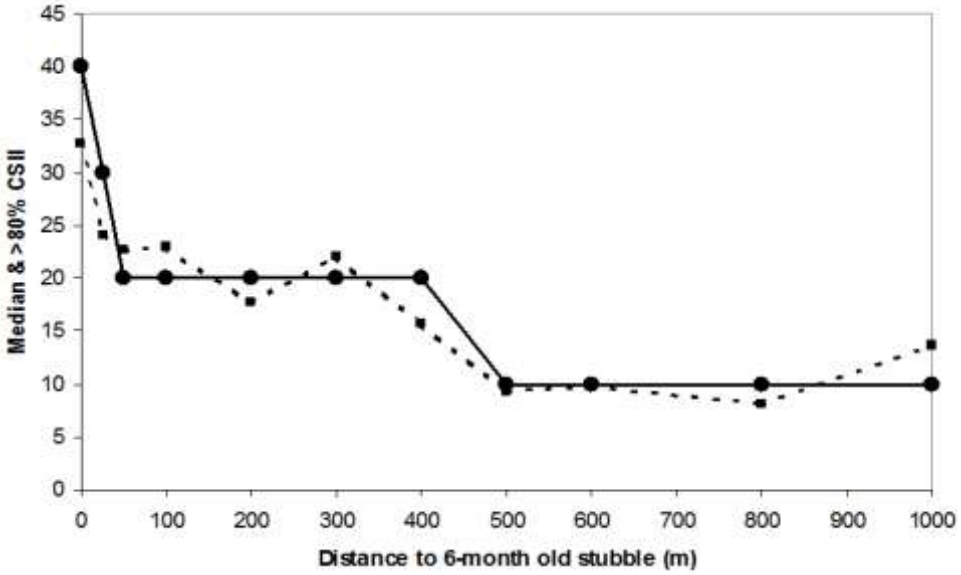

Figure 3. The effect of distance from stubble on Blackleg severity (Marcroft, 2002).

Canola Varietal Genetic Resistance

Two types of genetic resistance have been identified which are categorised as either seedling or adult plant resistance. Seedling resistance is conferred by single dominant genes which correspond to an avirulence gene in the pathogen. When the resistance gene is present in the Canola plant and the corresponding avirulence gene present in the pathogen, the plant shows a complete immune response with no development of leaf lesions or stem cankers. To date, approximately fourteen different seedling resistance genes have been identified and many have been deployed in commercial varieties. Varieties are available which have just one or combinations of seedling resistance genes.

Whilst seedling resistance genes are very effective; if varieties with the same resistance gene/s are grown in succession, the resistance can be overcome in as little as three years. Recent studies have shown there is a fitness cost associated with virulence in the pathogen and that rotation of single resistance genes can be used to manipulate the Blackleg population (Van de Wouw, 2014).

Adult plant resistance is conferred by numerous genes which behave quantitatively. Leaf lesions develop, but canker formation is reduced. Because the resistance is only partial, more cankering occurs when conditions are more favourable for the disease.

LATEST RECOMMENDATIONS

One of the most significant recent developments in Australian Canola management has been the publication of the 2014 Canola Blackleg Management Guide (Marcroft, 2014). The aim of the guide is to enable growers to develop sustainable Blackleg Management strategies tailored to their own individual farms.

A key component of the guide is instructing growers how to monitor the severity of Blackleg on their own farms. The guide then details the key environmental and management factors which influence the risk of Blackleg. Depending on the cropping history and management strategies, individual fields on the same farm may have different risks and require different future management strategies. Seedling resistance genes present in all commercially released varieties are listed. Rotating between varieties with different seedling resistance genes is recommended if one of two conditions exist; the grower is in a high risk region or they have been growing the same cultivar in close proximity for at least three years and observing increasing blackleg severity.

REFERENCES

Barbetti, M.; Khangura, R. **Fungal diseases of Canola in Western Australia.** Based on Bulletin 4406a [Reviewed June 2005]. Available at <http://archive.agric.wa.gov.au/PC_92073.html#blackleg>.

Howlett, B.; Ballinger, D.; Barbetti, M. **Canola in Australia: The first 30 Years.** 10th International Rapeseed Congress 1999. Available at <http://www.australianoilseeds.com/commodity_groups/Canola_association_of_australia/Canola_in_australia_-_the_first_30_years>.

Howlett, B. **How did Leptosphaeria maculans become such a successful pathogen?** 2014 Canola Pathology Workshop. Available at <http://www.australianoilseeds.com/__data/assets/pdf_file/0007/9952/2.3_BarbaraHowlett_UniMelb.pdf>.

Marcroft, S.; Bluett, C. **Blackleg of Canola.** Note Number: AG1352 Published: May 2008 Updated: July 2013. Available at <http://www.depi.vic.gov.au/agriculture-and-food/pests-diseases-and-weeds/plant-diseases/grains-pulses-and-cereals/blackleg-of-Canola>.

Marcroft, S.; 2002 Canola Pathology Workshop.

Marcroft, S. **2014 Blackleg management guide**. Fact Sheet Available at http://www.grdc.com.au/Resources/Factsheets/2014/04/Blackleg-Management-Guide-Fact-Sheet-Western-and-Southern-Regions.

Marcroft, S. and Stanley, M.; **Canola: The Ute Guide.** 2007.

Van de Wouw, A. **Virulence evolution, rotation and resistance stacking.** 2014 Canola Pathology Workshop. Available at <http://www.australianoilseeds.com/__data/assets/pdf_file/0007/9961/4.2b_AngelaVandeWouw_UniMelb.pdf>.