Blackleg of canola: A Trojan horse

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Blackleg, also known as stem canker or phoma stem canker, is the most notable disease in canola, both locally and internationally. It is present in all growing regions of the Western Cape, where almost all of South Africa's canola is produced. The disease can present symptoms on most parts of the plant, with common symptoms including leaf and pod lesions (*Figure 1*), as well as stem and crown cankers (*Figure 2*).

Serious yield losses are primarily the result of crown cankers later in the season, which, interestingly, are caused by leaf infection early in the season. Blackleg disease therefore acts as a Trojan horse. These damaging cankers restrict the transport of nutrients and water between the roots and upper canopy, and in severe cases, cause the plant to topple over and die.

Causal agents

Blackleg is caused by two closely related fungal species, *Leptosphaeria maculans* and *Leptosphaeria biglobosa*. The former is more virulent and responsible for upper and lower stem cankers, as well as the damaging crown cankers.

L. biglobosa causes cankers on the upper part of the stem, which is usually less damaging. The reason why *L. maculans* causes more severe damage is due to its ability to produce a toxin called Sirodesmin PL, which strengthens the pathogen that causes blackleg. Sirodesmin PL is also responsible for the yellow halo that is seen around lesions caused by *L. maculans* on canola leaves (*Figure 1A*).

Lesions caused by *L. biglobosa* are smaller, contain fewer fruiting bodies and have a thin, dark border. Both species are widely distributed in canola production areas around the world, with variation in their predominance and distribution.

No previous surveys on canola in the Western Cape exist to determine which of the two species are more prevalent. This is currently being addressed in a joint research project between the Stellenbosch University and the Western Cape Department of Agriculture. To date, only *L. maculans* has been confirmed on canola in the Western Cape.

L. maculans is known to be the dominant of the two species in Australia, from where most of our seed is imported. It is therefore possible that *L. biglobosa* may not be present nor be a significant contributor to blackleg disease in the Western Cape. Further research later in the season will confirm whether this is the case, since *L. biglobosa* is known to release spores later during the season.

Figure 1: Necrotic lesions containing fruiting bodies on leaves (A) and pods (B). (Photographs: Gert van Coller [A], Piet Lombard [B])

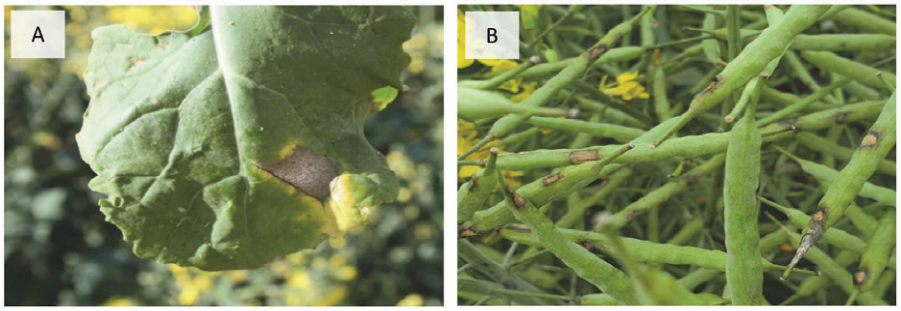


Figure 2: Cankers on canola crowns (A and B) and stems (C). (Photographs: Gert van Coller)

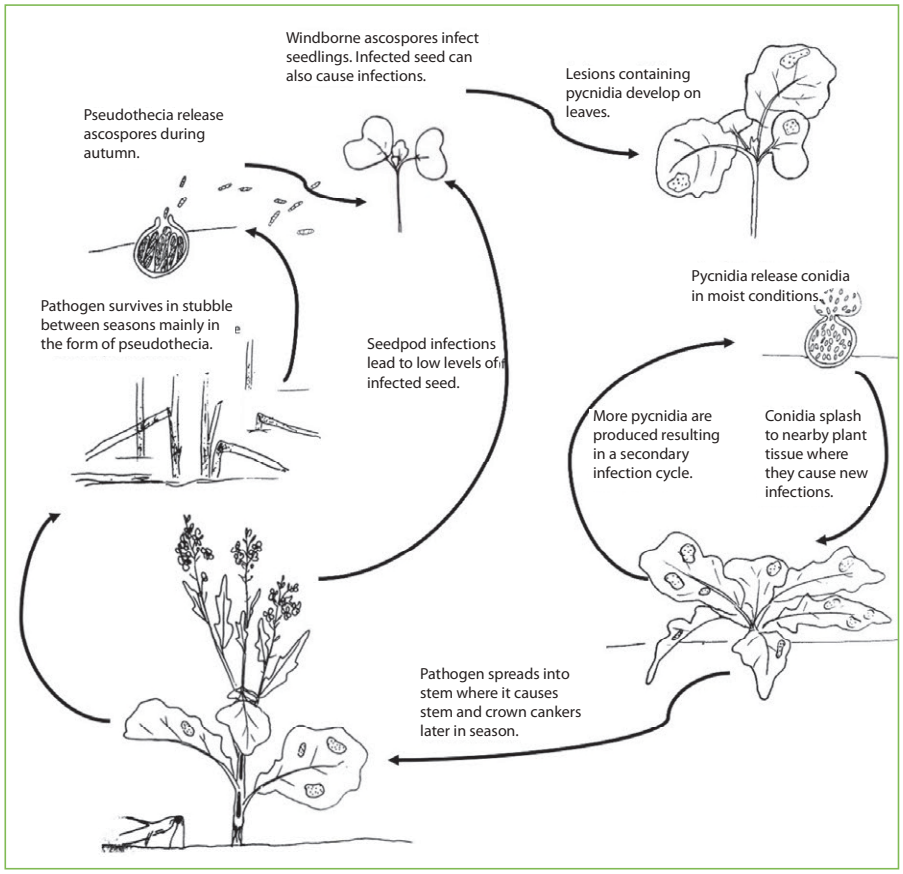


Disease cycle

The disease cycle of blackleg is presented in *Figure 3*. The blackleg pathogens survive on canola stubble left in the field between growing seasons. During this period, sexual reproduction can occur by which genetic material is exchanged between individuals of the pathogen. This process leads to the formation of sexual fruiting bodies (pseudothecia) which are visible as small black dots on the stubble (*Figure 4*).

Pseudothecia begins to release sexual spores (ascospores) in wet conditions during autumn, shortly after canola has been sown in the new season. Airborne ascospores can be transported by wind over vast distances to infect surrounding canola fields.

Figure 3: The disease cycle of blackleg.



Ascospores landing on the leaves and cotyledons of canola seedlings cause characteristic leaf spots. Within these leaf spots, asexual fruiting bodies (pycnidia) become visible as small black dots after a few days (*Figure 1*). Pycnidia releases asexual spores (conidia) when wet, which can splash along with raindrops to cause new infections on nearby plants, leaves and seed pods.

Leaf infection caused by ascospores up to the five-leaf stage is largely responsible for the damaging crown cankers later in the season. From infected leaves, the pathogen grows through the petiole into the developing stem and crown region, and remains dormant without presenting any symptoms. Later in the season, however, this relationship changes when the pathogen becomes necrotic, and causes stem and crown cankers.

These cankers usually become visible from full flowering onwards. From the crown area, the pathogen can grow into the taproot and cause root rot. After harvest, the pathogen survives on infected canola stubble, which allows it to subsist throughout the summer and cause disease during the following growing season.

Figure 4: Pseudothecia on canola stubble. (Photograph: Gert van Coller)



Management challenges

Various management strategies have been implemented over the years to mitigate losses caused by blackleg, the most significant being longer crop rotations (four years), the development of resistant cultivars, and chemical control with fungicide applications. However, due to the nature of its life cycle, blackleg remains difficult to manage.

Challenges pertaining to the management of blackleg include the following:

- Ascospores can travel long distances, which means that the disease can easily spread from infected stubble to nearby canola fields.
- Blackleg pathogens are seedborne. Canola pods infected by *L. maculans* can lead to seed infection, resulting in seedling infection and subsequently cankering in adult plants. Infected seed is a major source of pathogens spreading to new areas.
- The pathogen population consists of many races. The resistant genes present in canola cultivars mean that certain races of the pathogen are effectively controlled, while others are not. Over time, these uncontrolled races can multiply and overcome a particular cultivar's resistance, resulting in substantial losses.
- Sexual reproduction introduces new genetic variants into the local pathogen population every season. This has implications for management practices as it can lead to fungicide resistance and/or overcoming host resistance.

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