



Fig. 1. *D. toxica* (a) Conidial germination and infection - bar 10µm (b) sub-cuticular coralloid hyphae (c) susceptible infection after 14 days - bar 100µm (d) resistance after 14 days - bar 10µm

Disease: Phomopsis stem blight; lupinosis of sheep and other grazing animals

Pathogen: *Diaporthe toxica* anamorph *Phomopsis* sp.

Classification: K: Fungi P: Ascomycota C: Sordariomycetes O: Diaporthales F: Diaporthaceae

Diaporthe toxica (anamorph *Phomopsis* sp., formerly *P. leptostromiformis*) is a fungal endophyte and occasional pathogen of *Lupinus* species. When senescent lupin stubble absorbs moisture from rain or high humidity, the fungus grows saprophytically and produces toxic secondary metabolites known as phomopsins. Lupin grain can also be infected. Ingesting infected lupins exposes animals to these toxins, leading to lupinosis, a condition that manifests either as severe acute liver disease or as a chronic liver dysfunction syndrome. Although *D. toxica* can sometimes cause stem blight on green lupin seedlings and plants, such symptoms are rare in Australia.

History and Biology:

Lupins have been cultivated and consumed by humans and animals for thousands of years. However, many lupin species contain bitter alkaloids that historically required processing to make them suitable for human consumption.

Bitter, high-alkaloid narrow-leafed lupins were first introduced into Northern Europe around 1850 and quickly became the foundation of the Saxony Merino industry. In contrast, sweet, low-alkaloid lupin varieties were developed through breeding programs in Western Australia during the 1960s and 1970s.

Despite their value as a rotation crop, occasional human fatalities and major outbreaks of toxicity in animals have limited their broader adoption in recent years.

The discovery and naming of *Diaporthe toxica* brought closure to more than a century of investigation into the cause of lupinosis, dating back to the first major epidemic in Germany in 1872.

The infection process and life cycle of *Diaporthe toxica* in both resistant and susceptible lupins has now been fully characterised. Initial colonization occurs shortly after seedling emergence, when the fungus

establishes endophytic, subcuticular coralloid hyphae. These structures remain quiescent throughout the growing season, only resuming activity after plant maturity and senescence.

In resistant lupin varieties, the development of these coralloid latent structures is profoundly suppressed. This represents the first documented case of resistance targeting a saprophytically competent latent infection.

Early resistance breeding involved lengthy field trials and animal toxin tests. Understanding both the causal agent and the underlying resistance mechanism enabled the creation of rapid screening assays and molecular selection tools, accelerating the breeding of resistant lupin lines. As a result, all lupin varieties currently grown in Australia possess some degree of resistance

Impact and Management:

Lupinosis is no longer considered a disease of major significance for livestock producers in Australia. With sound grazing management, the risk of lupinosis remains low, while the agronomic benefits of lupins continue to be substantial.

A further benefit of resistant lupin varieties is their suitability for human consumption. Lupin grain can now be incorporated into a range of animal feeds, and lupin flour is currently sold for human use in Australia.

Further Reading:

Williamson et al 1994 *Diaporthe toxica* the cause of lupinosis in sheep. Mycological Research 98:1364-68

Williamson et al 1990 The development of subcuticular latent infection structures by *P. lept.* on lupins. Phytopathology 80:1039-41

<https://www.dpi.nsw.gov.au/animals-and-livestock/sheep/health/other/lupinosis-phomopsis>

<https://www.dpiird.wa.gov.au/businesses/pests-weeds-and-diseases/animal-pests-diseases/Lupinosis/>

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