Offered Review

ANTHRACNOSE OF LUPINS CAUSED BY *COLLETOTRICHUM LUPINI*: A RECENT DISEASE AND A SUCCESSFUL WORLDWIDE PATHOGEN

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SUMMARY

Lupins are grain legume crops cultivated in several parts of the world, with important roles in the agricultural and natural ecosystems. Recently lupin breeding faced a new and important challenge, a destructive seed- and airborne disease affecting stems and pods, named anthracnose. The current disease outbreak began in the 1980s and rapidly spread worldwide, affecting apparently all lupin species. The pathogen belongs to *Colletotrichum lupini*, a member of the acutatum species complex, and contrasts with other members of the latter by its host specificity and by its apparent clonality. However, in a matter of a few decades this pathogen managed to cause severe epidemics in lupin crops from diverse species (both of Mediterranean and North and South American origins) throughout the world, whether in humid or in dry climates, frequently causing high yield losses and in many cases leading farmers to replace lupin with other crops. Although several lupin crops rely on rich genetic resources, it proved very difficult to find effective resistance sources. Recent successes in this matter (backed by advances in genome sequencing of some lupin species) are still conditioned by the very narrow range of resistance genes available for breeders, risking a possible overcoming of such resistances if the pathogen finds itself means to create diversity that enables it to overcome resistance. To this end, advances in *Colletotrichum* genomics, with the forthcoming sequencing of the genome of *C. lupini*, are of great importance to understand the genetic nature of *C. lupini* host specificity and reproduction strategies.

Key words: *Colletotrichum lupini*, *Lupinus* spp., anthracnose, lupins, *Colletotrichum acutatum sensu lato*

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HOST: *LUPINUS* spp.

Several species of the genus *Lupinus* are important agricultural crops in different parts of the world. Other species are grown as ornamentals or as pioneer plants in soil recovery/stabilisation programmes. In the wild, they are important components of ecosystems in a wide range of soils and climates, from the tropics to the Arctic Circle, mainly on both shores of the Mediterranean basin and in East Africa, and throughout most of the American continent. While the Old World species are few and well delimited, the American species are numerous and frequently intercrossing and morphologically overlapping (Cowling *et al.*, 1998). Lupins are important crops due to their high seed protein content, their adaptability to low fertility soils, their beneficial effect on crops in rotation (the *Lupinus* spp.-*Bradyrhyzobium* are among the most efficient nitrogen fixers in agricultural crops; Fernandéz-Pascual *et al.*, 2007) and the multiplicity of cropusages (seed production, dry and green forage, and grazing). The chemical composition of lupin seeds (e.g. white lupin cultivar Magnus) is very similar to soybean (35% proteins and 0.5% starch), while pea has 25% protein and 50% starch. Some lupin species also reach high levels, up to 18%, of oil content. Lupins are appreciated in organic farming systems as resourceful tools employed with the objective to attain self-sufficiency regarding nitrogen supply, namely for animal feed, but also as N suppliers to subsequent cereals and horticultural crops. Due to their deep root systems, they are also very important in mobilising nutrients from deeper soil levels that shallower roots of other crops cannot reach. They are also relevant in reducing the population levels of pests and diseases of other crops. Lupins have been grown for centuries in the Mediterranean area (*L. albus* L., white lupin; *L. luteus* L., yellow lupin; and *L. angustifolius* L., narrow leaf lupin) and in the Andes (*L. mutabilis* Sweet; tarwi or Andean lupin) as food source. White lupin is nowadays a popular snack in several Mediterranean and Latin American countries, while tarwi persists as an important protein source since pre-Colombian times in the diet of people in several Andean regions (Váldez, 1980). In the 19th and 20th centuries, the development of low alkaloid varieties

enabled the cultivation of lupins for feed and the expansion of cropping areas, namely of yellow lupin in Central and North-Eastern Europe, of white lupin in Central and Western Europe, the Americas and South Africa, and of narrow leaf lupin in Australia where, at its peak in the late 1990s, lupin cultivated area surpassed 1,000,000 ha (Cowling *et al*., 1998).

Lupins are grown also as ornamentals (*Lupinus polyphyllus* Lindl. and hybrids, Russell lupin) due to their brilliant flowering racemes and are among the most popular ornamental plants in temperate climate countries (Elmer *et al*., 2001). Ecologically, lupins are noticeable as pioneer plants (Gladstones, 1998). Plants of *L. lepidus* Doug. ex Lindl. var. *lobbii* (Gray ex S. Wats.) Hitchc. were among the first natural colonisers of lava flows from Mount St. Helens volcanic eruption in 1980 in the USA (Bishop, 2002). Several examples of employment of lupins to soil reclamation/stabilisation arise from different parts of the world, including the use of tree lupin (*L. arboreus* Sims) for sand dune fixation in New Zealand (Dick, 1994), of Alaska lupin (*L. nootkatensis* Donn.) for recovery of barren soils (due to grazing, deforestation, harsh climate and/or recent volcanic activity) in Iceland (Magnusson, 1999) or of yellow lupin for the stabilisation of road-side slopes in Portugal.

The rusticity of lupins is often a limitation for the expansion of its cultivation, as they are frequently regarded as better suited for marginal cropping areas, while higher revenue crops are preferred in prime locations. This in part results from the relatively recent history of lupin breeding. For instance, while pod walls represent from 33% (in white lupin and in narrow leaf lupin) to 46% (in tarwi and yellow lupin) of the whole dry fruit, they represent only 13% in peas (Cowling *et al*., 1998). Similarly, while the hull represents 25% of the total seed weight in narrow leaf and yellow lupins, it represents only 9% in soybean (Hondelmann, 1984). While still dealing with early steps of domestication and development into a crop (including the search for low alkaloid content or even indehiscent genotypes) lupin breeding faced a new challenge in the last few decades, lupin anthracnose, a disease that soon proved to affect any lupin crop in nearly every part of the world.

HISTORY AND IMPORTANCE OF LUPIN ANTHRACNOSE

Anthracnose was first diagnosed on lupins in 1939 by J.L. Weimer in narrow leaf lupin in the USA, and attributed to *Colletotrichum gloeosporioides* (Penz.) Penz. & Sacc. (Weimer, 1943). Two previous references were mentioned in this work (Brazil 1912 and USA 1929), but the causal agent could not be confirmed.

Following an increase in narrow leaf lupin cultivation in South-eastern USA, an increase in disease incidence and severity was also observed. Disease resistance screening tests thus conducted led to the identification of resistance in Portuguese wild germplasm (Weimer, 1952). This was shown to be a dominant monogenic resistance (Forbes and Wells, 1961) that is temperature-dependent (Wells and Forbes, 1967): no disease occurs below 18ºC, even in susceptible lines, while even resistant lines become susceptible above 28ºC.

Later the disease reappeared with international importance in the 1970s and 80s in Europe and South America in white lupin and tarwi (Gondran *et al*., 1986; Baier and Linhares, 1991), leading the French phytopathologist and breeder Jean Gondran to forecast that this would become the most important disease of white lupin (Gondran, 1984). This turned out to be true soon after particularly in wet climates, namely for white lupins in France (Gondran, 1991), Russell lupin in the UK (Reed *et al*., 1996), strongly restricting lupin crop in Austria, France, Germany, Poland, Russia and Ukraine (Gondran *et al*., 1999), as well as in Brazil, Canada, Chile and Peru (Paulitz, 1995; von Baer and Hashagen, 1996), New Zealand (Dick, 1994), Australia (Sweetingham *et al*., 1995) and South Africa (van der Mey, 1996). The growing importance of the disease in less humid climates became evident when anthracnose emerged as major threat to the lupin industry in Australia in 1994 (Cowling *et al*., 2000a). Anthracnose became a serious disease of lupins worldwide, causing significant yield losses as high as 100% and a major limiting factor for lupin production (Nirenberg *et al*., 2002; Thomas and Sweetingham, 2004; Thomas *et al*., 2008b; Lotter and Berger, 2005; Semaškienė *et al*., 2008; Riegel *et al*., 2010). In Australia, the disease has greatest impact in the northern agricultural area of Western Australia, WA, where anthracnose susceptible sandplain lupins (*L. cosentinii* Guss.) occur spontaneously and are a reservoir for the pathogen (Thomas, 2003; Shea *et al*., 2008), rendering useless the initial eradication attempts (Bennett *et al*., 2013).

COLLETOTRICHUM LUPINI AS PART OF THE *C. ACUTATUM* SPECIES COMPLEX

Lupins anthracnose was first attributed to *Glomerella cingulata* (Stonem.) Spauld. & v. Schrenk., the teleomorph of the fungus *Colletotrichum gloeosporioides* (Penz.) Penz. & Sacc (Weimer, 1943). Later, the causal agent of lupin anthracnose was attributed to *C. acutatum* Simmonds ex Simmonds (Sreenivasaprasad *et al*., 1994; Gondran and Pacault, 1997; Lardner *et al*., 1999). Lately morphological, cultural and molecular (ITS and RAPDs) data suggested that *Colletotrichum* isolates from lupin anthracnose could be distinguished from *C. gloeosporioides* and *C. acutatum* and should be classified as a new species *Colletotrichum lupini* (Bondar) Nirenberg, Feiler & Hagedorn, with two variants *C. lupini* var. *lupini* and *C. lupini* var. *setosum* (Nirenberg *et al*., 2002; Lotter and Berger, 2005). Authors described morphological and physiological differences

between the two varieties: strains of var. *lupini* produced more asexual spores in the aerial mycelium, grow slightly slower on PDA and had a lower optimum growth temperature compared to strains described as var. *setosum*. Furthermore, var. *lupini* isolates usually formed concentric growth rings in culture and rarely differentiate setae, while var. *setosum* did not form any rings in artificial medium and differentiate setae consistently (Nirenberg *et al*., 2002). Recent taxonomic re-assessment of the *C. acutatum* species complex (Damm *et al*., 2012) validated *C. lupini* as a distinct species under the binomial *C. lupini* (Bondar) Damm, P.F. Cannon & Crous based on a multigene analysis (with five nucleotide sequences providing unequivocal distinction from other species, including the rDNA-ITS region and part of the glyceraldehyde-3-phosphate dehydrogenase, chitin synthase 1, histone 3 and beta-tubulin 2 genes), but did not confirm all the morphological differences and did not recognise the two varieties described before.

In the original description of the lupin anthracnose pathogen, Weimer (1943) described both *C. gloeosporioides* acervuli and *G. cingulata* perithecia from lesions in stems of diseased plants in the USA. Not long after, in Europe, the teleomorph was also obtained, in culture, from the anamorph isolated in the field (Oliveira, 1955). However, neither teleomorphs (*G. cingulata* or *G. acutata* Guerber & Correll) were recorded thereafter (Gondran and Pacault, 1997; Sweetingham *et al*., 1998b).

Studies focused on genetic characterization of *Colletotrichum* isolates associated with anthracnose of lupins worldwide showed the absence or low interspecific diversity (Yang and Sweetingham, 1998; Talhinhas *et al*., 2002). In the Azores islands, *C. lupini* and *C. acutatum* were both found associated with lupin anthracnose symptoms, however, it was suggested that *C. acutatum* colonization on lupin was a saprophytic colonization occurring only occasionally (Nirenberg *et al*., 2002). The identification of two different vegetative compatibility groups, one of which corresponding to North American isolates with lower virulence suggested that such isolates might be the direct descendants of the 1940-50s North American outbreak, but the teleomorph was not observed (Shivas *et al*., 1998). Similarly, two distinct populations were detected associated with lupin anthracnose in Portugal, but with one of them comprising a small number of low virulence isolates that likely represent cross-infections from other hosts into lupin plants (Talhinhas *et al*., 2002). In southern Chile different crops were sampled and only *C. lupini* was detected as the causal agent of anthracnose. In Chile high intraspecific genetic diversity was detected with random amplified polymorphic DNA (RAPD) markers suggesting the hypothesis of a South American origin of *C. lupini* (Riegel *et al*., 2010). In Ecuador, based on ITS sequences, most of anthracnose pathogens in tarwi form a uniform group, distinct from *C. lupini* (Falconí *et al*., 2013), showing high similarity (99.8%) with other species. However, due to the low resolution of ITS within *C. acutatum* species complex

Fig. 1. Anthracnose symptoms in a white lupin plant, evidencing the typical twisting of stems and necrosis in a petiole.

no final identification could be performed. Species belonging to clade 1 of the *C. acutatum* species complex (sensu Damm *et al*., 2012) have not been an easy task for taxonomists as all the strains unable to be assigned to a specific species do fit in this clade. Probably all Clade 1 members originate in South America as this geographic origin shows the highest diversity within the clade and more genetic groups and species are recently being described from this area (Crous *et al*., 2015; Bragança *et al*., 2016).

PATHOLOGY

Lupin anthracnose symptoms are similar in all lupin species (Sweetingham *et al*., 1998b), and are characterised by a typical twisting of stems, petioles and pods, with necrosis with orange masses of conidia (Fig. 1). Seeds in infected pods become brown and wrinkled. If able to germinate, the plantlet exhibits necrosis in cotyledons or in the hypocotyl (Gondran and Pacault, 1997).

The disease can be seed transmitted (Decker, 1947), and this is the main source of inoculum dispersal around the world (Sweetingham *et al*., 1998b; Semaškienė *et al*., 2008). Initially, the pathogen is spread by infected seed and can survive on infected crop stubble. Seedlings emerging from infected seed can have dark sunken lesions on hypocotyls, cotyledons, petioles or stem. Lesions are generally oval shaped, pink to beige and up to 2 cm long and cause the stem twisting (Semaškienė *et al*., 2008). In the centre of lesions are produced abundant orange condial masses that are dispersed within a crop by rain splash (Talhinhas *et al*., 2002; Thomas and Sweetingham, 2004). High humidity and temperatures over 10°C (4 h at 24°C or 16 h at 12°C) are sufficient for conidia germination and the onset of the disease. After penetration, the infection evolves rapidly, requiring eight days at 24°C (16 days at 12°C) until spore production and release (Sweetingham *et al*., 1998c). Primary seed infections as low as 0.01-0.1% can cause very severe infections depending on the agro-ecological

conditions (Thomas and Sweetingham, 2004; Sreenivasaprasad and Talhinhas, 2005). Infected crop can be an important source of inoculum, particularly in dry summer conditions, with no decomposition of organic matter (Sweetingham *et al*., 1998b), making crop rotation an important tool to minimise disease risk. Insects have also been implied in long distance inoculum dispersal within Australia (Bennett *et al*., 2013).

Other plants may act as inoculum sources for lupin crops and vice-versa. Lupins have been infected with *Colletotrichum* species from other hosts, such as *C. nymphaeae* (Pass.) Aa (Talhinhas *et al*., 2002), *C. fragariae* Brooks and *C. trifolii* Bain & Essary (Welty, 1984), and *C. gloeosporioides* f. sp. *aeschynomene* Daniel *et al.* (Weidemann *et al*., 1988), highlighting the importance of lupins as inoculum reservoirs for anthracnose pathogens of other hosts. On the other hand, and on what concerns lupin anthracnose, *Colletotrichum* isolates obtained from lupins were also pathogenic to peaches (Weimer and Dunegan, 1949), apples (Oliveira, 1955), tamarillo (Falconí *et al*., 2013) and strawberry (Baroncelli *et al*., 2015a), highlighting that other hosts may act as inoculum reservoirs for lupin anthracnose. Although the *Colletotrichum* genus, and the *C. acutatum* species complex in particular, are regarded as polyphagous, the lupin anthracnose pathogen have proved to form a well-defined, homogeneous and host-specific group within the *C. acutatum* species complex, unlike other heterogeneous and polyphagous groups in that species complex (Nirenberg *et al*., 2002; Sreenivasaprasad and Talhinhas, 2005; Baroncelli *et al*., 2015b). This host preference of lupin anthracnose pathogens was however atypical in the generally polyphagous *Colletotrichum* scene, and it was only after several concordant reports that it became clear that *C. lupini* is preferentially the lupin anthracnose pathogen. This host preference is quantitative rather than qualitative, with other species capable of infecting lupins and with *C. lupini* capable of infecting other hosts, but with maximum virulence of *C. lupini* occurring on lupins (Gondran and Pacault, 1997; Falconí *et al*., 2013). Nevertheless, not always lab tests reflect the situation in nature; for example most of *C. acutatum* species can infect strawberry in controlled environment, but only a few species are commonly detected on this host in nature (Baroncelli *et al*., 2015a).

Colletotrichum species utilize diverse strategies for invading host tissues (Bailey and Jeger, 1992). Although the mechanisms developed by *Colletotrichum* species appear analogous in pre-penetration, there are differences between species in the later mechanisms such as spore adhesion, melanisation and cutinisation in penetration of the plant cuticle by the appressoria. Although *C. acutatum* commonly produces necrotic symptoms, the interaction with hosts seems to have a longer biotrophic phase if compared to other species such as *C. higginsianum* and *C. gloeosporioides* (Freeman *et al*., 2001; Wharton and Diéguez-Uribeondo, 2004). In most cases *C. acutatum* persists and competes weakly as a saprophyte (Eastburn and Gubler, 1990). Nevertheless, *C. acutatum* appear to be an important endophyte as it has been isolated from many plants and has not always been associated with disease. For example, this fungus may colonize plants as an epiphyte or endophyte on hosts and non-hosts system, without producing symptoms (Freeman *et al*., 2000). Generally speaking *C. acutatum* is able to develop four different types of interaction with the host: biotrophic infection, necrotrophic infection, hemibiotrophic infection with infection vesicle and broad primary hyphae within host cell and hemibiotrophic and subcuticular, intra - and intercellular development. Unlike other systems very little is known about lupin anthracnose disease cycle and the interaction between *C. lupini* and its host. In this view, the availability of strains marked with reporter genes, such as fluorescent ones, could represent an useful tool to study the development of this pathogen within its host, as made for other plant/pathogen systems (Sarrocco *et al*., 2007), and transformation methodologies for such have been optimised for members of the acutatum species complex (Talhinhas *et al*., 2008).

Lupin anthracnose incidence depends on various environmental factors as warm temperatures, rainfall and wind. Rainfall required for liberation, dispersal and germination of spores, appears to be a dominant factor influencing the different levels of anthracnose infection on lupins. Stem tissue immediately below the growing point is most susceptible to infection and older stem tissues are more resistant (Thomas and Sweetingham, 2004). Both temperature and wind speed affect development of anthracnose epidemics. Susceptible cultivars are unlikely to be suitable in high rainfall areas due to the high risk associated with small levels of anthracnose inoculum. Dingle *et al*. (2002) developed AnthracnoseTracer, a spatiotemporal model for simulating the spread of lupin anthracnose, showing that in a susceptible lupin cultivar with a 0.01% initial seed infection, anthracnose would cause *ca.* 15% loss under favourable environmental conditions. In high rainfall areas, anthracnose resistance will be a major determinant of cultivar selection and seed infection will have a serious effects, particularly in less resistant cultivars (Thomas and Sweetingham, 2004).

CROP PROTECTION

The employment of inoculum-free seeds and the conduction of crop rotations enabling breaking the disease cycle are two fundamental factors to minimise the risk of anthracnose occurrence. A one-year break is considered sufficient to break inoculum viability in lupin debris (Sweetingham *et al*., 1998b; Shea *et al*., 1999). Inoculum-free seeds are preferentially obtained in regions environmentally unfavourable for the disease, from plots under long crop rotation and under tight phytosanitary control of the seeds produced.

For instance, as the production of inoculum-free seeds of *L. angustifolius* in Australia became more and more difficult (Thomas *et al*., 2008b), the detection and quantification of inoculum in seed lots became necessary, as well as the establishment of infection thresholds. For lupin anthracnose, infection threshold is defined as the seed infection level conducing to a 5% yield loss. Qualitative molecular tests are implemented for susceptible cultivars, where infection thresholds are as low as 0.01%. For less susceptible cultivars, the incubation of 1000 seed lots is sufficient for quantifying infection rates. In fact, the infection threshold depends mainly on the cultivar resistance level and on the local average annual rainfall (Table 1) (Thomas and Sweetingham, 2000; Thomas *et al*., 2008b; Semaškienė *et al*., 2008).

It is well established that long term seed storage causes substantial reductions in seed infection levels (Weimer, 1952). In fact, inoculum viability was totally lost after 6 months storage at 30ºC of *L. albus* and *L. angustifolius* seeds with infection rates of up to 70% (Thomas and Sweetingham, 2000). At 20ºC, under otherwise identical conditions, the reduction of inoculum viability is of 50%. Seed infection was significantly reduced by exposure to 60ºC for 8 days or to 80ºC for one day, with minor effect on germination (less than 15% reduction) (Thomas and Adcock, 2004).

Chemical disinfection of seeds can be achieved using carbendazim with 8-Hydroxyquinoline copper (II) or with iprodione without compromising germination rate or *Bradyrhizobium* sp. infection (Gondran and Pacault, 1997), or with thiram, 1 g/kg seed (Thomas and Sweetingham, 2000). Following a 21-fungicides test, thiram was registered as a seed treatment in Western Australia for control of anthracnose seed transmission in lupins (Thomas and Sweetingham, 2003). Similar disinfection levels can be obtained with a mixture of fludioxonil (75 mg/kg de seed), cyprodinil (75 mg/kg) and tebuconazole (30 mg/kg) (Römer *et al*., 2000).

Seed disinfection controls primary inoculum but does not act in secondary infections. For such, under field conditions, the preventive application of a mixture of cyproconazole (80 g ha^{-1}) and chlorothalonil (750 g ha^{-1}) has shown good effects (Gondran and Pacault, 1997), although such treatments were considered economically unrewarding (Lindner *et al*., 2000; Römer *et al*., 2000).

In field conditions, azoxystrobin 0.8 l/ha or metaconazol (0.8 l/ha) and cyproconazol + azoxystrobin 1 l/ha have a good efficacy on anthracnose (Arvalis, 2014).

In Australia, when applied one day prior to infection, azoxystrobin, chlorothalonil, mancozeb and copper oxychloride fungicides were highly effective, leading to yield gains in narrow leaf lupin with different degrees of susceptibility, suggesting that the application of foliar fungicides for anthracnose control is potentially a viable management option for lupin production in high anthracnose risk areas of Western Australia (Thomas *et al*., 2008a).

LUPINUS RESISTANCE TO ANTHRACNOSE

The use of resistant cultivars is generally regarded as a more economically and environmentally desirable option of plant protection as compared to chemical control and lupins have several characteristics that favour this option, including the fact that they are annual crop plants (smaller breeding cycles than perennial crops), have rich and generally accessible germplasm pools (in the nature and in germplasm banks; Talhinhas *et al*., 2003, 2006; Berger *et al*., 2013) and are minor crops (rendering the costs of specific fungicide development, testing, homologation and maintenance in catalogue uneconomical).

Plant resistance to anthracnose varies significantly between lupin species and between cultivars within species. When anthracnose first became an important disease in *L. angustifolius*, resistance was identified in Portuguese wild germplasm and introduced in cultivars. No resistance was identified in *L. albus* germplasm accessions tested by that time (Weimer, 1943).

When the disease reappeared with major importance, in the 1980s, it attained higher severity in *L. albus* and *L. mutabilis*, although it also became a problem for *L. angustifolius* and *L. luteus* (Sweetingham *et al*., 1998b). *L. angustifolius* cultivars showing resistance to the first disease outbreak were not resistant against the second outbreak (Sweetingham *et al*., 1998a). Germplasm resistance tests were performed in different parts of the world, with France and Brazil pioneering such experiments (Gondran, 1991; Baier and Linhares, 1991). At this stage, international guidelines were established for performing germplasm

Table 1. Production breaks caused in *Lupinus albus*, *L. angustifolius* and *L. luteus* cultivars with an initial anthracnose seed infection level of 5% according to the average precipitation under Western Australian conditions upon seed treatment with thiram, 1 g kg^{-1} seed (Thomas and Sweetingham, 2000).

Relative resistance level	Species and cultivar	Precipitation (mm)		
		<325	**325-400**	**>400**
Resistant	*L. angustifolius* 'Wonga' and 'Tanjil'	2.0 %	1.0 %	0.4 %
Moderately resistant	*L. angustifolius* 'Kalya'	1.0 %	0.4 %	0.1 %
Moderately susceptible	*L. angustifolius* 'Merrit', 'Belara' and 'Gungurru'	0.5 %	0.2 %	0.05 %
Susceptible	*L. angustifolius* 'Myallie', 'Tallerack' and 'Quilinock'	0.2 %	0.05 %	0.02 %
Very susceptible	*L. luteus* 'Wodjil'	0.05 %	0.02 %	0.0 %
Extremely susceptible	*L. albus* 'Kiev Mutant'	0.02 %	0.0 %	0.0 %

screening tests, in order to render studies comparable (Gondran *et al*., 1999). As the 1994 outbreak settled in Western Australia, and even wild sand plain lupin proved susceptible, all available germplasm was screened against anthracnose in New Zealand over the summer of 1997 and 1998 in an endeavour to find a source of resistance to anthracnose (Adhikari *et al*., 2009). Accessions noted as less susceptible to anthracnose were: in *L. albus*, cultivars 'Rumbo' (von Baer and Hashagen, 1999), 'Victoria' (Gondran *et al*., 1999), 'Prima', 'Lolita' (Galdames and Peñaloza, 1999), 'Cruz Alta' (Baier and Linhares, 1991), 'LA128' (Gondran, 1991), 'Andromeda' (Adhikari *et al*., 2009), 'Amira' (Adhikari *et al*., 2013); in *L. angustifolius*, a unspecified line (Galdames and Peñaloza, 1999), the determined cultivar 'Borweta' (Semaškienė *et al*., 2008) and cultivars 'Wonga' and 'Tanjil' (Sweetingham *et al*., 1998a; Cowling *et al*., 2000b).

'Tanji' cultivar has been strongly used for breeding anthracnose-resistant varieties in Australia (You *et al*., 2005); in this specific case resistance was controlled by a single dominant gene *Lanr1* (You *et al*., 2005). In white lupin high level of resistance have been found in some Ethiopian landraces and its lines have been used to confer anthracnose resistance in breeding programmes in Western Australia (Adhikari *et al*., 2011a). In fact, landraces of *L. albus* var. *albus* have been preferred as germplasm sources for breeding programmes, rather than wild accessions of *L. albus* var. *graecus*. Recently, breeding efforts proved successful in combining anthracnose resistance, high yield and early flowering/no vernalisation required, a combination difficult to obtain because of low frequency of early flowering progenies and the quantitative nature of anthracnose resistance in white lupin (Yang *et al*., 2010; Adhikari *et al*., 2011a, 2013). Cultivars arising from such approaches, as 'Amira', are expected to contribute to relaunching the Western Autralian white lupin industry after its collapse in the 1990's (Adhikari *et al*., 2013). Analyses of the progenies of the most promising crosses, supported by detailed integrated genetic linkage maps (Phan *et al*., 2007; Vipin *et al*., 2013), suggest that breeding efforts may become faster and more accurate in the near future.

Reflecting the more recent domestication history of narrow leaf lupin (as compared to white lupin), wild *L. angustifolius* genetic resources play an important role in narrow leaf lupin breeding (e.g., while wild accessions represent 65% of the *L. angustifolius* entries in the Australian Lupin Collection, they only account for 7% of *L. albus* entries; Berger *et al*., 2013). The major impact of anthracnose in the growing narrow leaf lupin industry in Western Australia in the 1990s exposed the vulnerability of the germplasm used in breeding, leading to the search for new sources of resistance from wild germplasm. Cultivars 'Tanjil' and 'Wonga' bear the dominant gene *Lanr1*, conferring resistance to anthracnose, and crosses between these resistance donors and susceptible lines with other interesting agronomic traits have been extensively analysed. The establishment of a co-dominant PCR-Based molecular marker (AnManM1), tightly linked to the *Larn1* (Yang *et al*., 2004), enabled the implementation of Marker Assisted Selection (Yang *et al*., 2007), an approach that was subsequently refined using Restriction-site Associated DNA (RAD) markers (Yang *et al*., 2012). The sequencing and annotation of the *L. angustifolius* genome enabled the identification of a TIR-NBS-LRR gene in cultivar 'Tanjil', co-segregating (0 cM) with gene *Lanr1* among 94 F8 recombinant inbred lines (RILs), and therefore a likely candidate to be *Lanr1* (Yang *et al*., 2013). Recently, an alternative source of resistance was identified based on European-bred germplasm (Fisher *et al*., 2015). Gene *LanrBo* is a dominant gene conferring resistance to anthracnose, for which flanking marker genes have been identified. Field trials suggest that the genetic background of the breeding line Bo7212, bearing gene *LanrBo*, is quite different from that of 'Tanjil' and other *Lanr1*-bearing lines, providing a more diversified type of response to anthracnose (Fisher *et al*., 2015). The current success on achieving effective resistance against anthracnose in narrow leaf lupin is however hampered by the reliance on a single resistance source. Although alternative sources of resistance, involving transgenic approaches, may provide a broader basis of resistant germplasm (Wijayanto *et al*., 2009), the current reliance on a limited number of genes and on qualitative resistance strengthen the need to discover alternative and durable (i.e., quantitative) sources of resistance.

In yellow lupin, resistance was identified in the Portuguese wild accession PI168539, enabling its introduction in several breeding lines which show partial resistance to the disease, providing enough protection for cultivation in Western Australia (Adhikari *et al*., 2011b). No true resistance has been so far identified in tarwi, although tolerance related with growth cycle patterns may be exploited to minimise disease severity (Falconí *et al*., 2015).

In summary, while germplasm screening and disease resistance breeding efforts are providing promises that the current lupin anthracnose crisis may be overcome, it is notable that a pathogen with such a narrow genetic diversity has been able to cause such a level of damage and economic loss. It should be of concern what might happen if the pathogen finds means of creating diversity and the challenges that this would likely pose to lupin industry.

COLLETOTRICHUM LUPINI NEW PERSPECTIVES

In addition to its economic impact, *Colletotrichum lupini* as part of the *C. acutatum* species complex is an interesting model for evolutionary investigations due to the diversity of host-determined specialization. Genome projects of *Colletotrichum* species have already opened a new era for the study of pathogenesis and evolution (O'Connell *et al*., 2012). Evidence of the evolutionary tendency of switching from a wide host range to a narrow host range suggests

the hypothesis of differences at a genome level such as: expansion of specific gene families, differences in gene content and in synteny. The first complete genome sequence from a member of the *C. acutatum* species complex has been released (Baroncelli *et al*., 2014) and more are expected soon (JGI CSP Plans: http://jgi.doe.gov/developing-colletotrichium-genomics-resources/) with a dataset that include *C. lupini* genome. The new knowledge generated and the resources are likely to provide impetus for comparative and functional genomics studies in *C. acutatum* species. Advances in next generation sequencing methods and bioinformatics tools for genome analyses provide significant scope to advance our knowledge of the genetic basis of host-microbe interactions and pathogen evolution in important system like *C. lupini/Lupinus* spp. A deep knowledge of the mechanisms underlying *C. lupini* speciation and host specialization are certainly of relevance for a better informed deployment of disease resistance breeding strategies.

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