

BEAN YELLOW MOSAIC VIRUS IN LUPINS: STRAINS, LOSSES, EPIDEMIOLOGY AND CONTROL

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ABSTRACT

Bean yellow mosaic virus (BYMV) causes a serious yield limiting disease in all domesticated lupin species. Strains of BYMV that are seed-borne in *L. luteus* and *L. albus* and contaminate seed stocks, occur widely in Europe and North America, but are not established in Australia. In *L. angustifolius*, two types of BYMV strains are distinguished, necrotic and non-necrotic, both of which cause substantial yield losses. When spreading from adjacent pastures to *L. angustifolius* crops, the former has a largely monocyclic and the latter a polycyclic pattern of spread. The non-necrotic type spreads faster and causes greater clustering of infected plants than necrotic BYMV. Understanding the principle factors driving BYMV epidemics in south-west Australia and field experiments evaluating the effectiveness of individual control measures were important in devising Integrated Disease Management (IDM) approaches that minimise seed yield losses in *L. angustifolius* crops. The IDM package produced is effective against necrotic and non-necrotic types of BYMV and consists almost entirely of cultural measures. Partial resistance to initial infection by aphid vectors occurs in different lupin species. The systemic necrotic reaction in *L. angustifolius* is controlled by at least one hypersensitivity gene. Genetic engineering for BYMV resistance in *L. angustifolius* failed to provide transgenic plants with effective resistance. Effective quarantine is important in preventing establishment in Australia of strains that are seed-borne in lupins.

KEYWORDS

lupin, virus, disease, strains, epidemiology, control measures

INTRODUCTION

Virus diseases cause major yield losses in lupin crops worldwide, especially those caused by *Bean yellow mosaic virus* (BYMV) and *Cucumber mosaic virus* (CMV), both of which are transmitted non-persistently by aphids. BYMV causes damaging systemic diseases in *L. angustifolius* (narrow-leafed

lupin), *L. albus* (white lupin), *L. luteus* (yellow lupin) and *L. mutabilis* (pearl lupin), and also in *L. cosentinii* (sandplain lupin) and three other species currently being domesticated, *L. atlanticus*, *L. digitatus* and *L. pilosus*. CMV does so in *L. angustifolius*, *L. luteus* and *L. mutabilis*, but not in the other five species (Jones and McLean, 1989; Jones, 2001). In the south-west Australian grainbelt, *L. angustifolius* is the lupin species most widely grown. *L. albus* and *L. luteus* are also planted as crops, and *L. cosentinii* (sandplain lupin) for forage in pastures (Gladstones, 1998). Important vector aphids include *Myzus persicae* (green peach aphid), *Acyrtosiphon kondoi* (blue green aphid) and *Aphis craccivora* (cowpea aphid) which colonise *L. angustifolius*, and non-lupin colonising species, such as *Rhopalosiphum padi* (oat aphid) and *Lipaphis erysime* (turnip aphid) (Jones and McLean, 1989; Jones, 2001).

Early research on both viruses in lupins, especially in *L. luteus* and *L. albus*, in different parts of the world was reviewed by Jones and McLean (1989). Jones (2001) reviewed later research on virus diseases in *L. angustifolius*. This article focuses mainly on research on BYMV in lupins done after these reviews were published.

VIRUS STRAINS

Strains of BYMV occurring in Europe and North America are seed-borne in *L. luteus* and *L. albus*, but not in *L. angustifolius*. Where seed transmission occurs in lupins, sowing infected seed stocks constitutes the principal source of this virus (Jones and McLean, 1989). However, in the south-west Australian grainbelt, the BYMV strains found so far are not seed-borne in lupins but spread to lupin crops from annual pastures dominated by *Trifolium subterraneum* (subterranean clover) (Jones, 2001; Cheng *et al.* 2002; Jones *et al.* 2003). The original, more common types of BYMV strains cause severe necrotic symptoms killing the *L. angustifolius* plants that become infected (Jones and McLean, 1989; Jones, 2001). The less abundant non-necrotic strains do not kill *L. angustifolius* plants but cause mottle and stunting. Infected plants remain in crops and act as secondary sources for acquisition and further spread by aphid vectors throughout the growing season (Cheng and Jones, 2000; Cheng *et al.* 2002;

Thackray *et al.* 2002; Jones, 2005). Using a cultivar that develops the typical necrotic reaction with all necrotic strains and breeding line 90L423-07-13 that develops non-necrotic reactions with most of them as differentials, distinct strain groups of BYMV can be distinguished based on reactions in *L. angustifolius*. Species of lupins other than *L. angustifolius* cannot be used as differentials as necrotic strains of BYMV fail to induce necrotic reactions in them (Cheng and Jones, 2000).

YIELD LOSSES

Because necrotic BYMV kills infected *L. angustifolius* plants while plants infected with non-necrotic BYMV remain as virus sources for subsequent spread, the latter spread faster than the former within the same lupin stands infecting more plants and so cause greater overall yield losses despite inducing milder symptoms. When the effects of virus infection were examined in individual lupin plants infected with necrotic BYMV, they were killed by early infection so there was little or no seed production. With late infection, seed yield and seed number were decreased by at least 80% and 74% respectively (Jones *et al.* 2003). At low plant densities, magnitude of overall grain yield loss due to infection with necrotic BYMV tended to be the same as final infection incidence. However, as plant density increased in partially infected stands, seed yield loss decreased due to compensatory

growth of healthy plants and the requirement for more incoming viruliferous aphids to establish the same proportion of infected plants (Jones, 2001). With non-necrotic BYMV, seed yield and seed number diminished with increasing duration of plant infection, these decreases ranging from 48 to 99% and 35 to 98% for late to early infection, respectively. In 5.6 x 15 m plots into which different numbers of clover plants infected with non-necrotic BYMV were introduced, subsequent incidence of infection depended on the magnitude of the initial virus source and yield was decreased by 21–24%, 31–43% and 64–66% with 4, 8 or 16 foci/plot, respectively. With both types of BYMV, yield loss in infected plants was mainly due to failure to produce any seed or to fewer seeds being produced but smaller seed size also contributed (Jones *et al.* 2003). The results showed that both have considerable yield limiting potential.

EPIDEMIOLOGY

A sound understanding of the factors that drive BYMV epidemics in lupin crops is required before an effective Integrated Disease Management (IDM) package can be developed. Jones (2001) described the principle primary and secondary factors driving epidemics of both types of BYMV strains in *L. angustifolius* crops in south-west Australia. Table 1 provides a summary of these factors, what determines each of them and how they influence virus spread.

Table 1. Principle factors favouring epidemics of BYMV in *L. angustifolius* crops.

Factors	Determined by	Consequence
Primary		
Primary virus source external	Whether seed-transmission occurs in annual clovers in adjacent pasture.	Decreasing infection gradient from edge of lupin crop next to external pasture source resulting in greater yield losses at crop edge than internally.
Virus source substantial	Large numbers of seed-infected source plants within adjacent pasture. Requires high proportion of infected clover seeds and soil moisture for seedling establishment.	Greater virus spread to lupin crop. Epidemic spreads faster and reaches higher final incidence causing greater yield losses.
Early arrival of aphids	Presence of substantial 'green ramp' in 3–4 months preceding growing season. Requires rainfall in February to April. Aphids build up on weeds, volunteer crop plants and pasture plants before growing season starts.	Early aphid flights to lupin crops. Epidemic takes off early, and reaches higher final incidence causing greater yield losses.
Virus spread largely monocyclic or polycyclic	Necrotic BYMV kills lupin plants removing sources for virus acquisition by aphids within crop (largely monocyclic). Plants infected with non-necrotic BYMV persist as virus acquisition sources (polycyclic).	Faster spread and higher final incidences with polycyclic pattern causing greater yield losses (non-necrotic BYMV). Slower spread with smaller final incidence and overall yield losses with monocyclic pattern (necrotic BYMV).

Secondary		
Abundance of aphids during growing period	High mean winter temperatures and low frequency of cold fronts causing heavy rainfall and high winds favour aphid population growth and greater numbers (both colonising and non-colonising aphids).	More aphids mean increased spread from infected to healthy lupin plants. Virus spread, final incidence and yield losses enhanced.
Absence of groundcover	Absence of weeds and stubble residues from preceding crop.	Bare earth around lupin plants increases aphid landing rates, so increasing spread, final incidence and yield losses.
Sparse stands and poor canopy development	Late planting, deep sowing, low seeding rate, poor plant establishment, wide row spacing, fertiliser inadequate or not placed below seed.	Infected plants not shaded out by healthy neighbours and aphid landings increased. Virus spread, final incidence, yield losses enhanced.
Heavy grazing of neighbouring pasture	High stocking rates and continuous grazing.	BYMV-infected clover source plants not shaded out by healthy neighbours within pasture. Virus spread to lupins, final incidence and resulting yield losses enhanced.
Prolonged growing season	Good finishing rains allow crop to remain green and continue growing into late spring.	Virus spread continues increasing longer. Final virus incidence greater resulting in greater yield losses.

Table is a modified version of Table 1 in Jones (2001).

The spatial and temporal dynamics of spread of necrotic and non-necrotic strains of BYMV in *L. angustifolius* stands were studied. Spread of non-necrotic BYMV was polycyclic regardless of whether the initial virus source was internal or external. In contrast, especially when it came initially from external sources, spread of necrotic BYMV was largely monocyclic. When both types spread from adjacent naturally infected pasture, necrotic BYMV was initially more abundant, but non-necrotic BYMV then spread faster reaching a greater final incidence. In experiments where both types of strains were introduced to large plots, whether virus sources were removed or left in place, non-necrotic BYMV always spread faster than necrotic BYMV. In plots without introduced infection foci, spread was also faster with non-necrotic BYMV. Spread of necrotic BYMV was more diffuse with more isolated symptomatic plants and small clusters than occurred with non-necrotic BYMV, spread of which was more comprehensive, reflecting near monocyclic and polycyclic patterns of spread respectively. When BYMV spread into commercial crops in the absence of a non-host barrier, there was a steep decline in its incidence with distance from the crop edge. However, when a 20 m-wide perimeter barrier of oats intervened between the two, there was only a shallow decline (Cheng *et al.* 2002; Thackray *et al.* 2002; Jones, 2005).

CONTROL

CULTURAL AND CHEMICAL MEASURES

Jones (2001) summarised a series of field experiments examining the effectiveness of different

cultural control measures against necrotic BYMV strains spreading from adjacent annual legume pastures into *L. angustifolius* stands. The following measures helped to suppress BYMV spread: early crop canopy cover, high seeding rates, narrow row spacing, stubble groundcover using minimum tillage, perimeter non-host barriers, and mixed cropping with a non-host (Jones, 2001). Other cultural control measures likely to be useful that were not evaluated in field experiments were avoiding use of fields with large perimeter:area ratios surrounded by pasture, sowing early maturing cultivars and maximising weed control (especially clovers) within the crop. Table 2 shows the mode of action of each of the cultural measures included within the IDM package against BYMV. As regards chemical control, applying insecticides to suppress virus spread is generally ineffective with non-persistently aphid-borne viruses like BYMV and this was also the case in a field experiment with BYMV and *L. angustifolius*. However, because of the greater role played by wingless aphids as virus vectors in pasture than annual crops, application of rapid 'knock down' insecticides (e.g. pyrethroids) to adjacent pasture might assist in suppressing the BYMV epidemic in clovers thereby limiting the virus source for spread to lupin crops (Jones, 2001). Control of BYMV in *L. angustifolius* crops therefore depends on an IDM package that employs a range of cultural measures with insecticide application to adjacent pastures the only chemical measure included. The overall package is designed to be 'user friendly', causing the farmer few additional labour demands and minimal extra expense or disruption to normal farming operations. By

combining control measures that operate in different ways, suppression of BYMV spread is maximised. However, the farmer does not necessarily need to use all of the component measures. Also, the forecasting model

for BYMV in lupins (Maling *et al.* 2008) can be used to predict the likelihood of severe BYMV epidemics and the need for extra control measures at sowing time.

Table 2. Components of integrated disease management strategy for BYMV in *L. angustifolius* crops.

Control measure	Mode of action
Promote early crop canopy development.	Shades over infection sources within crop (seed-infected and/or early infected plants) and diminishes aphid landing rates.
Sow at high seeding rates to generate high plant densities.	Minimises infection sources (seed-infected and/or early infected plants) and diminishes aphid landing rates. Dilutes numbers of infected plants.
Sow at narrow row spacing.	Narrow spacing diminishes aphid landing rates.
Maximise stubble groundcover using minimum tillage procedures that minimise soil cultivation.	Diminishes aphid landing rates until crop canopy develops.
Sow perimeter non-host barrier crop in between adjacent pasture and lupin crop.	Decreases virus spread into crop from external pasture source.
Mixed cropping with non-host.	Diminishes virus spread.
Avoid fields with large perimeter: area ratios adjacent to pastures in high virus risk regions.	Decreases ingress of virus into crop from external pasture source.
Sow early maturing cultivars.	Decreases final infection incidence reached, especially in prolonged growing seasons.
Maximise weed control.	Minimises potential weed virus infection sources within crop (especially clovers for BYMV).
Spray adjacent pasture with pyrethroid insecticide in high virus risk regions.	Suppresses virus spread within external pasture infection source by killing colonising aphids.

Table is a modified version of Table 3 in Jones (2001).

HOST RESISTANCE

The value of the systemic necrotic reaction in limiting yield losses by diminishing the rate of BYMV spread within *L. angustifolius* crops was demonstrated in field experiments that compared the rates of spread of necrotic and non-necrotic strains (Cheng *et al.* 2002). Cheng and Jones (2000) postulated that presence of two independently inherited strain-specific hypersensitivity genes might be responsible for the systemic necrotic reactions. Jones and Smith (2005) studied segregation for the necrotic (systemic hypersensitive) response to infection with a necrotic strain of BYMV in progeny plants from six crosses with *L. angustifolius*. The parents were two cultivars that always developed necrosis when BYMV-infected (Danja and Merrit) and two genotypes that always responded without necrosis (90L423-07-13 and P26697). In the four possible combinations of crosses between genotypes with necrotic and non-necrotic reactions, segregation for the necrotic response in F₂ progeny plants always fitted a 3:1 ratio (necrotic: non-necrotic). This provided evidence for the existence of one of the two hypersensitivity genes postulated, a dominant hypersensitivity gene named *Nbm*⁻¹. However, its expression seemed influenced by independently segregating modifier genes in the genetic background

since necrosis developed at widely different rates within affected F₂ progeny plants resulting in staggered killing.

In early studies in Europe, useful partial resistance to infection by aphid vectors was found in some genotypes of *L. albus* and *L. luteus*. Decreased ability to transmit BYMV through seed was also present in *L. luteus*. Resistance to initial infection and decreased seed transmission were sought in routine breeding for BYMV resistance in *L. luteus* at that time (reviewed by Jones and McLean, 1989). In Australia, routine field screening of *L. angustifolius* genotypes for partial resistance to initial infection with BYMV by aphids was undertaken for many years by the Australian national lupin breeding program, but was recently dropped. Unless a source of extreme resistance to BYMV is found that can be used in breeding, it is important to continue to ensure that all new *L. angustifolius* cultivars carry the necrotic trait (Jones, 2001).

GENETICALLY ENGINEERED RESISTANCE

Transgenic lupins containing a synthetic 'hairpin' replicase (NIb) gene for resistance to BYMV derived from an isolate that causes systemic necrosis in *L. angustifolius* (MI) were generated. Many transgenic *L. angustifolius* plants harbouring this transgene were

tested by inoculation with infective sap. In 2004, three independent transgenic lines at generations T2 and T3 were identified as potentially resistant to isolate MI. Progeny from these transgenic lines were tested in 2007 using five distinct BYMV isolates. Unfortunately, however, the resistance had been silenced as it was no longer expressed in infections by any isolate, including MI (S.J. Wylie, unpubl.).

QUARANTINE

For Australia, maintenance of quarantine measures to avoid establishment of strains that are seed-borne in lupin is critical. Such strains would be likely to induce disease problems similar to those caused in Europe and North America when lupin seed stocks are contaminated with seed-borne BYMV (Jones and McLean, 1989), especially if non-necrotic strains of BYMV that prove to be seed-borne in *L. angustifolius* were to become established.

LITERATURE CITED

- Cheng, Y. and R.A.C. Jones. 2000. Biological properties of necrotic and non-necrotic strains of bean yellow mosaic virus in cool season grain legumes. *Annals of Applied Biology* 136: 215–227.
- Cheng, Y., R.A.C. Jones and D.J. Thackray. 2002. Deploying strain specific hypersensitive resistance to diminish temporal virus spread. *Annals of Applied Biology* 140: 69–79.
- Gladstones, J.S. 1998. Distribution, origin, taxonomy, history and importance. *IN Lupins as Crop Plants: Biology, Production and Utilisation* (Eds J.S. Gladstones, C.A. Atkins, J. Hamblin) pp. 1–39 (CAB International: Oxford, UK).
- Jones, R.A.C. 2001. Developing integrated disease management strategies against non-persistently aphid-borne viruses: A model programme. *Integrated Pest Management Reviews* 6: 15–46.
- Jones, R.A.C. 2005. Patterns of spread of two non-persistently aphid-borne viruses in lupin stands under four different infection scenarios. *Annals of Applied Biology* 146: 337–350.
- Jones, R.A.C., B.A. Coutts and Y. Cheng. 2003. Yield limiting potential of necrotic and non-necrotic strains of *Bean yellow mosaic virus* in narrow-leaved lupin (*Lupinus angustifolius*). *Australian Journal of Agricultural Research* 54: 849–859.
- Jones, R.A.C. and G.D. McLean. 1989. Virus diseases of lupins. *Annals of Applied Biology* 114: 609–637.
- Jones, R.A.C. and L.J. Smith. 2005. Inheritance of hypersensitive resistance to *Bean yellow mosaic virus* in narrow-leaved lupin (*Lupinus angustifolius*). *Annals of Applied Biology* 146: 539–543.
- Maling, T., A.J. Diggle, D.J. Thackray, K. Siddique and R.A.C. Jones. 2008. Forecasting green-bridge diseases in the south-west of Western Australia: *Bean yellow mosaic virus* in lupins. *IN Proceedings of 12th International Lupin Conference*, Fremantle, Western Australia.
- Thackray, D.J., Smith, L.J., Cheng, Y., Perry, J.N. and R.A.C. Jones. 2002. Effect of strain-specific hypersensitive resistance on spatial patterns of virus spread. *Annals of Applied Biology* 141: 45–59.