THE EPIDEMIOLOGY OF CUCUMBER MOSAIC VIRUS IN NARROW-LEAFED LUPINS (*LUPINUS ANGUSTIFOLIUS*) IN SOUTH AUSTRALIA

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Summary

(1) Epidemics of CMV in *L. angustifolius* were experimentally initiated in 1987, 1988 and 1989, to study factors affecting the rate of epidemic progress.

(2) Rapid virus spread occurred during spring, and coincided with the plant growth stages of flowering and pod fill.

(3) Field diagnosis of infection by symptoms and by detection of antigen by DAS ELISA was compared. Incidence of infection at crop maturity was underestimated by about 50 % when symptoms were used for diagnosis, due to the occurrence of symptomless infections.

(4) Lupins, which were either infected through seed or inoculated at the seedling stage, were shown to be important primary sources of inoculum. Clumps of infected plants formed following virus spread by aphids. Infection gradients arising from linear sources of inoculum were steep, with incidence of infection decreasing from 100 % to 20 % in a distance of 2.5 m. (5 plant rows). Secondary infection foci also developed from longer distance dispersal of inoculum.

(5) Yellow pan traps were used to monitor aphid flights during the lupin growing season in 1987, 1988 and 1989. *Myzus persicae*, *Lipaphis erysimi*, *Rhopalosiphum padi*, *Aphis craccivora* and *Brachycaudus rumexicolens* were trapped in largest numbers. For all species, most abundant flights were in the period between late August to October. *R. padi* and *M. persicae* were trapped regularly, though in low numbers, through winter.

(6) In 1989, the yellow pans were compared with suction traps, which were mounted at the height of the lupin canopy, and with green tile traps. The green tiles trapped inefficiently and no comparison could be made with the yellow pans and suction traps. Large numbers of *R. padi* and *M. persicae* were collected in the suction traps and these species were

therefore abundant in the boundary layer of the crop where they could alight on the lupins. Abundant flights of *L. erysimi* were detected using the yellow pans, but this species was rarely trapped in the suction traps. It was therefore considered that *L. erysimi* were not flying in the boundary layer of the lupin crop and were therefore not attempting to alight.

(7) The daily flight patterns of aphids on six days in spring, 1989, were monitored, and corresponding weather conditions also measured. The daily flight patterns of *M. persicae*, *R. padi* and *L. erysimi* were variable and affected by temperature and wind speed. Aphid flight was not detected below 10.6 C for *M. persicae*, 9.7 C for *R. padi* and 12.7 C for *L. erysimi*. High wind speeds reduced, but did not inhibit flight, as some aphids were trapped when wind speed was greater than 10 km/hour. The rapid detection of abundant aphid flights following a change in the weather to conditions that favour flight initiation, suggested that the aphid source was close (within 5 km.) to the field site.

(8) From glasshouse transmission tests, *M. persicae*, *R. padi*, *A. craccivora*, *B. rumexicolens*, *D. aucupariae* and *H. lactucae* were shown to be capable of transmitting a lupin isolate of CMV, but not *L. erysimi*, *Macrosiphum euphorbiae* and *Metapolophium dirhodum*.

(9) Field spread of CMV correlated with aphid flights, assuming a 2 week delay between inoculation and detection of systemic infection. *R. padi* was concluded to be an important vector as (a) virus spread in the 1987 field trial correlated with a flight of aphids composed primarily of *R. padi*, (b) *R. padi* was shown to be abundant in the boundary layer of the crop and was found alighting on the lupins and (c) *R. padi* was shown to be capable of transmitting CMV. There was no effect on epidemic progress of either initiating colonies of *A. craccivora* on introduced sources of inoculum, or initiating colonies of *R. padi* on oats, planted next to introduced sources of inoculum.

(10) Epidemic progress in the 1987 field trial was quantified using previously published models proposed to describe the functional relationship between disease increase and vector numbers. The interpretations of the best fitting model were (a) the growth rate of the epidemic increased as the number of alates entering the crop increased, (b) the probabilty of virus acquisition by the aphids increased as incidence of infection increased, as might occur during a polycyclic epidemic, and (c) the probability of transmission decreased as the epidemic progressed.

Infection gradients observed in the 1988 field trial were also quantified using previously published models. The interpretations of the better fitting models were that either most or all of the inoculum originated from the linear source of inoculum, and that inoculum was diluted with increasing distance from the source. Infection gradients with the shape observed, are considered to occur during a monocyclic epidemic, or at the beginning of a polycyclic epidemic. The infection gradients were, in fact, observed soon after the first spring flight of aphids.

(11) Commercially traded lupin seed from South Australia, Victoria and New South Wales, was tested for CMV transmission. Transmission rates ranged between 0 and 11.5 %. CMV transmission was found in seeds from the lupin cultivars 'Danja', 'Illyarrie', 'Warrah', 'Wandoo' and 'Yandee'. CMV transmssion was detected in 23 of the 51 seedlots tested.

(12) Seed transmission rates were dependent on the age of the plant at the time of inoculation. Highest rates of transmission (between 23 and 25 %) occurred when the plant became infected during vegetative growth. The rate of transmission progressively declined with later inoculations after the beginning of flowering. The probability that a seed became infected decreased the more developed the seed at the time of inoculation. Infectious CMV was recovered from the cotyledons and primordial radicle and plumule, suggesting that seed transmission resulted from infection of the embryonic tissues.

(13) Dry matter productivity was only affected when the plant became infected during vegetative growth. Seed productivity was still affected when the plant became infected during flowering. For lupins infected at the seedling stage, the reduction in seed yield was 99.7 % and the reduction in dry matter yield was 98.6 %. Seedlings that were infected through seed showed no greater tolerance to infection than those seedlings that were inoculated at the cotyledon stage.

(12) Largest numbers of infected seed were produced by plants which were inoculated at the beginning of flowering. Virus spread occuring at the beginning of flowering was shown mathematically to be optimal for virus persistence by seed transmission, as for all but the largest of epidemics, maximum seed transmission levels are predicted to occur when the plants are inoculated at this time. It was also shown that CMV could not persist by transmission in lupin seeds if no secondary spread by aphids occurred.

Seed transmission levels were observed to increase in one generation, even when secondary spread by aphids was small.