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

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Table of Contents

	Page
Summary	xv
Statement	xix
Acknowledgements	xx
Chapter 1 General Introduction	1
1.1 Description of CMV	1
1.2 CMV variation and classification of strains	
1.2.1 Processes by which variation arises	2
1.2.2 Classification and detection of CMV strains	4
1.3 Types of natural transmission	
1.3.1 Aphid transmission	5
1.3.2 Seed transmission	8
1.4 Role of weed and ornamental plants in the ecology of CMV	11
1.5 Control of CMV	
1.5.1 Eradication or geographical isolation from sources of inoculum	13
1.5.2 Control by preventing aphid spread	14
1.5.3 Plant resistance to virus infection	16
1.6 Scope of the thesis	19
Chapter 2 General materials and methods	22

2.1	CMV isolate	22
2.2	Storage of CMV	22
2.3	Maintenance of the CMV isolate in the glasshouse	22
2.4	Inoculation and biological indexing	22
2.5	Serological testing	23
2.6	Management of field trials	24
2.7	Seed source	25
2.8	Plant protection	26
2.9	Introduction of infected lupin seedlings into field plots	27
Chapter 3 Description of the CMV epidemic in <i>L. angustifolius</i>		28
3.1	Introduction	28
3.2	Materials and methods	
3.2.1	The 1987 field trial	29
3.2.1.1	Design	29
3.2.1.2	Establishment of colonies of <i>A. craccivora</i> in treatment VV	29
3.2.1.3	Surveying for virus infection	30
3.2.2	The 1988 field trial	30
3.2.2.1	Design	30

3.2.2.2	Establishment of the oats	31
3.2.2.3	Initiation of colonies of <i>R. padi</i> on the oats in treatment VOA	31
3.2.2.4	Insecticide treatment of the oats in treatment VO	31
3.2.2.5	Sampling of the oats for aphids	32
3.2.2.6	Surveying for virus infection	32
3.2.3	The 1989 field trial	32
3.2.3.1	Design	32
3.2.3.2	Surveying for virus infection	33
3.2.3.3	Use of trap plants	33
3.2.3.4	Analysis of the spatial pattern of infected plants	33
3.3	Results	
3.3.1	Comparison of diagnosis by ELISA and by symptoms	34
3.3.2	Analysis of the temporal development of the epidemic	
3.3.2.1	Epidemic development in relation to crop growth	35
3.3.2.2	Effects of treatments VV and V on development of the 1987 epidemic	36
3.3.2.3	Effects of treatment VOA, VO and V on development of the 1988 epidemic	36
3.3.2.4	Epidemic development in the 1989 trial and the use of trap plants to measure infection pressure	37
3.3.3	Analysis of the spatial development of the epidemics	
3.3.3.1	Analysis of the spatial distribution of infected plants in treatment C of the 1987 field trial	38
3.3.3.2	Analysis of the spatial distribution of infected plants in the 1988 field trial	39
3.3.3.3	Analysis of the spatial distribution of infected plants in the 1989 field trial	40

3.4	Discussion	
3.4.1	Diagnosis of CMV infection of field plants using symptoms	41
3.4.2	Effect of treatment, 1987 field trial	41
3.4.3	Effect of treatment, 1988 field trial	42
3.4.4	Temporal progress of the epidemics	43
3.4.5	Spatial progress of the epidemics	44
Chapter 4 Vector Studies		46
4.1	Introduction	46
4.2	Materials and Methods	
4.2.1	Descriptions of aphid traps	47
4.2.2	Collection, storage and identification of aphids	48
4.2.3	Monitoring of aphid flights	48
4.2.4	Daily flight patterns	48
4.2.5	Aphid colonisation	49
4.2.6	Aphid transmission experiments	49
4.2.7	Correlation between aphid flights and field spread of CMV	50
4.3	Results	
4.3.1	Aphid species trapped in the yellow pans and their seasonal flight patterns	50
4.3.2	Daily flight patterns of aphids	52
4.3.3	A comparison of the species composition of the aphid catches from yellow pans and suction traps	53
4.3.4	Aphids trapped in green tile traps in 1989	55
4.3.5	Aphid colonisation of the lupins	55
4.3.6	Aphid transmission of CMV	57

4.3.6	Aphid activity in relation to virus spread	57
4.4	Discussion	
4.4.1	Seasonal patterns of aphid flights	58
4.4.2	Daily patterns of aphid flights	59
4.4.3	Comparison of aphid trapping methods	61
4.4.4	Aphid colonisation	62
4.4.5	Transmissibility of CMV-BSA by different aphid species	62
4.4.6	Relationship between aphid flights and field spread of CMV	63
Chapter 5 Modelling of epidemic progress		64
5.1	Introduction	64
5.2	Methods	
5.2.1	Frequently used symbols and their definitions	65
5.2.2	Modelling increases in incidence of infection as a function of numbers of vectors	66
5.2.3	Modelling temporal progress of the epidemic	68
5.2.4	Modelling infection gradients	69
5.2.5	Transformation of y when $y = 0$ or 1	70
5.2.6	General criteria for the selection of the most appropriate model	70
5.3	Results and Discussion	
5.3.1	Modelling epidemic progress in 1987 as a function of the cumulative number of <i>M. persicae</i> , <i>R. padi</i> , <i>A. craccivora</i> , <i>D. aucupariae</i> and <i>B. rumexicolens</i>	70
5.3.2	Modelling epidemic progress in 1987 as a function of the cumulative number of <i>R. padi</i>	72

5.3.3	Biological interpretation of vector model 4	72
5.3.4	Modelling of the infection gradients observed in 1988	73
5.3.5	Biological interpretation of the gradient models	74
Chapter 6 Seed transmission of CMV and the effect of CMV infection on lupin productivity		76
6.1	Introduction	76
6.2	Materials and methods	
6.2.1	Seed source	77
6.2.2	Virus source	78
6.2.3	Tests for seed transmission of CMV	78
6.2.3.1	Testing of seed by ELISA	78
6.2.3.2	Assay of germinated seedlings for virus (seedling assay)	78
6.2.4	Screening for seed transmission of CMV in commercial seedlots	79
6.2.5	Field experiments	
6.2.5.1	1988 experiment: effect of time of infection on rate of seed transmission of CMV	79
6.2.5.2	1989 experiment: relationship between the age of the plant at the time of inoculation and seed weight, dry matter production and rate of transmission of CMV in the seed	80
6.2.6	The distribution of infected seed on the lupin plant	80
6.2.7	Distribution of CMV in the seed	81
6.2.8	Relationship between seed weight and recovery of the virus from the seed	81
6.2.9	Comparison of the growth rate of seedlings infected via seed with those inoculated at the cotyledon stage	81
6.2.10	Survival of CMV through seed transmission	82

6.3	Results	
6.3.1	Seed transmission in commercial seedlots	82
6.3.2	CMV transmission in seed from plants infected during the 1988 field trial	83
6.3.3.	Relationship between plant age at the time of inoculation, symptom severity and seed and dry matter yields	84
6.3.4	Effect of plant age at the time of inoculation on rate of seed transmission	85
6.3.5	Distribution of infected seeds on the plant	86
6.3.6	Distribution of infectivity in seed parts	87
6.3.7	Effect of CMV infection on seed weight	88
6.3.8	Rate of growth of seedlings relative to the time of infection	88
6.3.9	Increase in seedborne CMV through sequential generations	88
6.4	Discussion	89
Chapter 7 General discussion		94
7.1	Sources of inoculum	94
7.2	Secondary spread by aphids	94
7.3	Patterns of aphid flights	97
7.4	Modelling disease progress as a function of vector numbers	99
7.5	Modelling spatial progression	100
7.6	Persistence of CMV between lupin generations through seed	101

	transmission	
7.7	The mechanism of seed transmission	103
7.8	Recommendations for control	106
7.9	Conclusions	110
	Appendices	111
A 1	Description of the CMV epidemic in <i>L. angustifolius</i>	
A 1.1	Temporal progress of the epidemic in the 1987 field trial (diagnosis by symptoms)	111
A 1.2	Analysis of variance to test for differences in incidence of infection between treatments VV, V and C of the 1987 field trial	111
A 1.3	Temporal progress of the epidemic in treatment C of the 1987 field trial (diagnosis by ELISA)	112
A 1.4	Temporal progress of the epidemic in the 1988 field trial (diagnosis by ELISA)	112
A 1.5	Spatial pattern of infected plants in the 1988 field trial on September 7	
A 1.5.1	Incidence of infection in rows at varying distance from the linear source of inoculum	113
A 1.5.2	Incidence of infection in columns at varying distance from the edge of the plot	113
A 1.6	Analyses of variance to test for differences in incidence of infection between treatments, between rows at varying distance from the linear source of inoculum, and between halves of the plot	
A 1.6.1	Analysis using data from treatments VOA, VO, V and C	114
A 1.6.2	Analysis using data from treatments VOA, VO and V	114

A 1.6.3	Analysis using data from treatment C	114
A 1.7	Analyses of variance to test for differences in incidence of infection between treatments and between columns at different distances from the edge of the plot	
A 1.7.1	Analysis using data from treatments VOA, VO, V and C	115
A 1.7.2	Analysis using data from treatment C	115
A 2	Vector studies	
A 2.1	Numbers of aphids trapped in the yellow pans in 1987	116
A 2.2	Numbers of aphids trapped in the yellow pans in 1988	117
A 2.3	Numbers of aphids trapped in the yellow pans in 1989	118
A 2.4	Aphid species trapped in the yellow pans in 1987	119
A 2.5	Aphid species trapped in the yellow pans in 1988	120
A 2.6	Aphid species trapped in the yellow pans in 1989	121
A 2.7	Numbers of aphids collected in the suction traps in 1989	122
A 2.8	Hourly trap collections of aphids on September 5, 1989	123
A 2.9	Hourly trap collections of aphids on September 7, 1989	124
A 2.10	Hourly trap collections of aphids on September 8, 1989	125
A 2.11	Hourly trap collections of aphids on September 20, 1989	126
A 2.12	Hourly trap collections of aphids on September 21, 1989	127
A 2.13	Hourly trap collections of aphids on September 29, 1989	128
A 2.14	Temperature and wind conditions in September, 1989	129
A 3	Modelling of the epidemic	130
A 3.1	Regression analyses to fit the linear forms of the vector models to the epidemic observed in treatment C of the 1987 field trial	
A 3.1.1	Vector species - <i>A. craccivora</i> , <i>B. rumexicolens</i> , <i>D. aucupariae</i> , <i>M. persicae</i> and <i>R. padi</i>	
A 3.1.1.1	Vector model 1	130

A 3.1.1.2	Vector model 2	130
A 3.1.1.3	Vector model 3	130
A 3.1.1.4	Vector model 4	131
A 3.1.2	Vector species - <i>R. padi</i>	
A 3.1.2.1	Vector model 1	131
A 3.1.2.2	Vector model 2	131
A 3.1.2.3	Vector model 3	131
A 3.1.2.4	Vector model 4	132
A 3.2	Regression analyses to compare the epidemic observed in the 1987 field trial with that predicted by the vector models	
A 3.2.1	Vector species - <i>A. craccivora</i> , <i>B. rumexicolens</i> , <i>D. aucupariae</i> , <i>M. persicae</i> and <i>R. padi</i>	
A 3.2.1.1	Vector model 1	132
A 3.2.1.2	Vector model 2	132
A 3.2.1.3	Vector model 3	133
A 3.2.1.4	Vector model 4	133
A 3.2.2	Vector species - <i>R. padi</i>	
A 3.2.2.1	Vector model 1	133
A 3.2.2.2	Vector model 2	134
A 3.2.2.3	Vector model 3	134
A 3.2.2.4	Vector model 4	134
A 3.3	Regression analyses to fit the linear forms of the gradient models to the infection gradients observed in the 1988 field trial on September 7	
A 3.3.1	Gradient model 1	
A 3.3.1.1	Distinct lines fitted to gradient data from treatments VOA, VO and V	135
A 3.3.1.2	Parallel lines fitted to gradient data from treatments VOA, VO and V	135
A 3.3.1.3	One coincident line fitted to gradient data from treatments VOA, VO and V	135

A 3.3.2	Gradient model 2	
A 3.3.2.1	Distinct lines fitted to gradient data from treatments VOA, VO and V	136
A 3.3.2.2	Parallel lines fitted to gradient data from treatments VOA, VO and V	136
A 3.3.2.3	One coincident line fitted to gradient data from treatments VOA, VO and V	136
A 3.3.3	Gradient model 3	
A 3.3.3.1	Distinct lines fitted to gradient data from treatments VOA, VO and V	137
A 3.3.3.2	Parallel lines fitted to gradient data from treatments VOA, VO and V	137
A 3.3.3.3	One coincident line fitted to gradient data from treatments VOA, VO and V	137
A 3.3.4	Gradient model 4	
A 3.3.4.1	Distinct lines fitted to gradient data from treatments VOA, VO and V	138
A 3.3.4.2	Parallel lines fitted to gradient data from treatments VOA, VO and V	138
A 3.3.4.3	One coincident line fitted to gradient data from treatments VOA, VO and V	138
A 3.4	Regression analyses to compare the infection gradients observed in the 1988 field trial with those predicted by the models	
A 3.4.1	Gradient model 1	139
A 3.4.2	Gradient model 2	139
A 3.4.3	Gradient model 3	139
A 3.4.4	Gradient model 4	139
A 3.5	Residual plots for the linear forms of the vector models fitted to data from the 1987 field trial (vector species - <i>A. craccivora</i> , <i>B. rumexicolens</i> , <i>D. aucupariae</i> , <i>M. persicae</i> and <i>R. padi</i>)	140
A 3.6	Residual plots for the linear forms of the vector models fitted to data from the 1987 field trial (vector species - <i>R. padi</i>)	141
A 3.7	Residual plots for the linear forms of the gradient models fitted to data from the 1988 field trial	142

A 4	Seed transmission of CMV and the effect of CMV infection on lupin productivity	143
A 4.1	Analysis of variance to test for differences between the seedling assay and testing of seed by ELISA to determine seed transmission rates	143
A 4.2	Effect of age at the time of inoculation on seed and dry matter productivity	
A 4.2.1	Seed and dry matter yields	143
A 4.2.2	Analyses of variance to test for differences in seed yields between treatments	
A 4.2.2.1	Analysis using data from treatments 1-5	143
A 4.2.2.2	Analysis using data from treatments 3-5	144
A 4.2.3	Analyses of variance to test for differences in dry matter yields between treatments	
A 4.2.3.1	Analysis using data from treatments 1-5	144
A 4.2.3.2	Analysis using data from treatments 3-5	144
A 4.3	Effect of plant age at the time of inoculation on seed viability (1989 field experiment)	
A 4.3.1	Germination rates	144
A 4.3.2	Analysis of variance to test for differences in germination rates between treatments 2, 3 and 4	145
A 4.4	Effect of plant age at the time of inoculation on rate of seed transmission of CMV (1989 field experiment)	
A 4.4.1	Rates of seed transmission of CMV	145
A 4.4.2	Analysis of variance to test for differences in rates of seed transmission between treatments 2, 3 and 4	145
A 4.5	Relationship between seed weight and infection of that seed	
A 4.5.1	Weights of infected and uninfected seed	146
A 4.5.2	Analysis of variance to test for differences in weight between infected and uninfected seeds	146

References

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 probability 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 transmission 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 occurring 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.