THE EFFECT OF PEA SEED-BORNE MOSAIC VIRUS ON YIELD OF PEAS.

G.E. Ovenden
Crop Research Division, DSIR, Christchurch
and
J.W. Ashby
Plant Diseases Division, DSIR, Christchurch

ABSTRACT

Field responses of a number of garden and field pea cultivars to pea seed-borne mosaic virus (PSbMV) were studied in Canterbury, New Zealand during the 1980/81 season. The plants were inoculated with the virus 2 and 4 weeks after emergence. Only transitory symptoms were observed in the field. No infection was detected in cultivars resistant to bean yellow mosaic virus (BYMV). A significantly lower yield of inoculated plots was detected only in the field pea Pamaro. A comparison of efficiency of seed and leaf tests for measuring seed transmission of the virus is reported. The difference in infection levels which were achieved are likely to be higher than the levels to be expected in well managed commercial crops and it is considered that PSbMV is unimportant in its effect on yield of pea crops in New Zealand.

Additional Key Words: cultivars, methods.

INTRODUCTION

Pea seed-borne mosaic virus (PSbMV), because of its seed-borne nature and the international exchange of pea seed over many years, is probably present in all countries where peas are grown (Hampton and Mink, 1975). The effects may be transitory or mild and many infections are not detected. It was first detected in New Zealand in 1978 by Fry and Young (1980).

Despite world-wide concern over possible losses caused by this virus and the institution of quarantine measures by some countries, we are aware of only two studies investigating the effect of the virus on crop yields (Chiko and Zimmer, 1978; Kraft and Hampton, 1980). These established some significant yield losses and technical problems of experimental work with this virus. We therefore decided to study the effect of PSbMV on several New Zealand field and garden pea cultivars.

PSbMV exists as a number of strains which differ in their reactions on a differential set of pea cultivars (Hampton *et al.*, 1981). The isolate used in the present crop loss assessment trial was of the strain most frequently isolated in New Zealand. The strain differs from many of the strains isolated elsewhere in being unable to infect cultivars homozygous for *mo*, a recessive gene (Ashby, unpublished results) which confers resistance to the pea mosaic strain of bean yellow mosaic virus (BYMV) (Yen and Fry, 1956).

MATERIALS AND METHODS

Yield trial

During the 1980/81 season, 17 pea cultivars were grown at Methven, Canterbury. In this area, 400 m above mean sea level, aphid populations are generally lower than elsewhere on the Canterbury Plains. Both BYMV-susceptible and BYMV-resistant cultivars were included to provide direct comparisons of their yields in the presence of PSbMV; and also to ascertain whether the BYMV-resistant cultivars were resistant to PSbMV under field conditions, since previous test had all been done in the glasshouse (Ashby, unpublished results). The susceptible cultivars were the garden peas — Princess, Victory Freezer, and Heron; and the field peas — Pamaro, Rovar, Maro and Birte. The cultivars presumed resistant were the garden peas C39, Kuru, Pania, Patea, Puke, Puget, Small Sieve Freezer, and Tere and the field peas — Huka and Whero.

Captan treated seed was sown in three-row plots, 6 m long, with 17 cm between rows and 1 m between plots. A randomised block design was used, 3 complete replicates being uninoculated and 3 replicates inoculated. A virus resistant breeding pea (SC 4) was sown in one row buffers between replicates.

A systemic insecticide, Metasystox at 700 ml/280 l/ha, was applied at 10-14 day intervals until flowering in an attempt to prevent virus spread by aphids.

A line of Pamaro with 24% of infected seed was sown in a growth chamber at 25 °C and 16h photoperiod (subsequently referred to as high light/high temperature treatment) and were used as inoculum. Inoculum was freshly prepared for each plot by grinding infected plants in Yarwood's (1972) buffer (0.5% K^2HPO^4 + 0.5% bentonite) plus a small amount of 600 mesh carborundum, and was then rubbed onto two leaves of each plant in the inoculated plots 2 and 4 weeks after emergence. Buffer and carborundum alone was rubbed on the leaves of uninoculated control plots.

Dry seed was harvested using a Vogel plot thresher. Seed yields and 1000 seed weights, after air cleaning, were determined for each plot.

Virus testing

Because of the transient nature of field symptoms and the impracticability of testing each plant for infection, the relative amount of virus in the plots was estimated by comparing virus levels in the parent seed with those in seed harvested at the end of the trial.

The levels of PSbMV infection in parent and progeny seed were determined by seed testing. 200-seed samples were taken from each of 17 parent seed lines and from progeny seed of each of the 102 plots. Each sample was divided into 20 lots of 10 seeds, soaked, ground and applied to dark-treated plants of *Chenopodium amaranticolor* Coste & Reyn, as described by Mink and Parsons (1978). These indicator plants were scored at 12 and 24 days after inoculation and the probable percentage infection was estimated using the method of Gibbs and Gower (1960).

The level of infection in parent seed was also determined by a growing-on test in which 200-seed samples were grown in pots (10 seeds/pot), in a growth chamber under high light/high temperature conditions. As infected plants were seen they were recorded and removed. Doubtful infections were checked by inoculation to *C. amaranticolor*. Two leaves per plant were removed from plants remaining at the end of the tests, combined into 20-leaf samples and inoculated to *C. amaranticolor* to check for symptomless infections of the peas.

RESULTS

Levels of PSbMV in parent and progeny seed

PSbMV was not detected in any of the parent seed lines of cultivars resistant to BYMV. In the field, no symptoms were observed in these cultivars when inoculated with PSbMV, and no virus was detected in the seed obtained from inoculated plants.

The results of seed tests to determine the levels of seed infection in parent and progeny seed of the BYMV-susceptible cultivars and the results of growing-on tests of parent seed are given in Table 1. PSbMV was not detected in any plants which did not show symptoms in the growing-on test. All parent lines of susceptible cultivars were infected with PSbMV, with levels ranging from 4.9% to 26.5% (growing-on test). Mild transient vein-clearing symptoms were observed in the field in some susceptible cultivars about 10 days after the first inoculation but by flowering time no symptoms of infection were evident. In all cultivars, the level of PSbMV infection in seed from inoculated plots was two to three times greater than the level in seed from uninoculated controls. The levels in seed from uninoculated controls were greater than that in parent lines of Victory Freezer, Pamaro, Maro and Birte and less than that in parent lines of Princess and Rovar.

Plot yields and 1000-seed weights

There were no significant differences in yield or 1000-seed weights between inoculated or uninoculated plots of resistant cultivars. The plot yields and 1000-seed weights of the susceptible cultivars are given in Table 2. Yields of 5 cultivars and seed weights of 6 were reduced by inoculation but these reductions were not significant. The reduction in yield, estimated from comparing growing-on and seed test results, was 11.6%. The only significant individual difference was in Pamaro in which yield from inoculated plots was reduced by 33%. Infection generally had little effect on seed size, except on Maro. The resistant cultivars as a group did not yield significantly more than the susceptible cultivars.

TABLE 1: Levels of PSbMV infection in parent and progeny seed of BYMV-susceptible cultivars as determined in seed tests and in growing-on tests.

Cultivar	Percentage Infection: Parent Seed		Progeny of Inoculated Plants		Progeny of Control Plants	
	Growing-on Test	Seed test	Estimate*	Seed test	Estimate*	Seed test
Heron	14.6	-	-	2.8	-	0.5
Princess	7.0	3.5	8.0	4.0	3.6	1.8
Victory Freezer	14.0	1.1	57.3	4.5	22.9	1.8
Birte	4.9	0.5	86.2	8.8	44.1	4.5
Maro	11.5	0.0	35.6	3.1	10.3	0.9
Pamaro	26.5	4.2	44.2	7.0	32.8	5.2
Rovar	11.7	3.5	9.4	2.8	3.0	0.9
Mean	12.9	2.1	40.1	4.7	19.4	2.2

- Insufficient seed available for test

* Estimate from comparison of growing-on and seed test results for parent seed.

TABLE 2: Plot yields and 1000 seed weights in BYMV susceptible cultivars inoculated with PSbMV compared with uninoculated controls.

Cultivar	Plot Yield (kg)		1000 Seed Weight (g)	
	Inoculated	Control	Inoculated	Control
Heron	3.27	2.85	194.7	191.3
Princess	5.58	8.05	247.7	254.0
Victory Freezer	6.82	6.80	248.3	257.3
Birte	6.77	8.60	257.0	264.0
Maro	3.66	3.85	273.7	299.0
Pamaro	6.17	9.23	200.0	216.3
Rovar	9.73	10.38	262.3	272.3
Standard errors of response to inoculation	1.38		10.36	

DISCUSSION

The lack of symptoms and seed infection in BYMV-resistant cultivars demonstrated that they were resistant to the strain of PSbMV used and that no other strains were detectable in the seed lines. Most strains of PSbMV tested in other countries are able to infect BYMV-resistant peas

and the reason why such strains appear to be rare in New Zealand is not known.

The detection of PSbMV by growing plants under high light/high temperature conditions revealed much higher levels of seed infection than predicted from seed tests. In many cultivars, however, the detection of PSbMV by observation in plants grown under optimal conditions for symptom expression required a considerable amount of experience and, for practical routine purposes, the seed test is adequate for determining moderate to high levels of infection.

The proportion of virus-infected seed produced by a PSbMV-infected plant is extremely variable and depends mostly on the genetic constitution of the host (Stevenson and Hagedorn, 1973).

In the absence of any vector, the percentage of infected seed in a seed line should decrease with each generation. Although insecticide was regularly applied to the trial area, a few pea aphids (*Acyrthosiphon pisum* Harris) were observed in the plots between applications. Since the inoculated plots provided a high inoculum potential and the pea aphid is a very efficient vector of PSbMV (Ashby, unpublished results) it was anticipated that some natural spread of virus would occur. The results (Table 1) suggest that this may have happened since the level of seed infection in progeny of Victory Freezer, Pamaro, Maro and Birte from uninoculated control plots increased relative to the levels in parent seed. The decrease in levels of PSbMV in seed from uninoculated Princess and Rovar relative to the parent lines may indicate that seed transmission occurred at a comparatively lower rate, or that they are less palatable to aphids, than the other cultivars.

Even though there was seed-borne infection in the parent seed and possibly natural spread in the field, the infection achieved in inoculated plots was two to three times greater than that occurring in uninoculated controls. This estimate is based on the differences in levels of infection on seed from inoculated plots compared with that from uninoculated plots. It was shown from parent seed testing that the seed test underestimated the 'true' level of seed transmission as shown in growing-on tests. If these tests are used as a basis for estimation of the probable true rate of seed transmission in progeny seed (Table 2), the difference in level of transmission between inoculated and uninoculated plots is 20.7%. This suggests that the difference in level of infection between inoculated and uninoculated plots was at least 20.7% and almost certainly much higher. This difference in infection levels had little effect on yield or 1000 seed weights in most cultivars. Pamaro, the only cultivar in which yield was significantly affected, was the cultivar from which the virus isolate was obtained.

The problems encountered in this trial were similar to those encountered by Chiko and Zimmer (1978) and Kraft and Hampton (1980). These resulted from the seed-borne nature of the virus, the ease of natural spread and the lack of readily observable symptoms. Chiko and Zimmer (1978), by correcting for infection of controls, considered that PSbMV caused 11% yield reduction in one cultivar and 33% in another.

Kraft and Hampton (1980) found significant losses in yield in 3 processing pea cultivars out of 5 tested. Time of inoculation also had a significant effect on yield losses. In the present trial, a greater difference between infection levels of control and inoculated plots might have demonstrated significant yield decreases. However, as the differences in infection levels which were achieved were almost certainly higher than the levels expected in most commerical crops, it is concluded that PSbMV is unimportant in its effect on the yield of pea crops in New Zealand.

ACKNOWLEDGEMENTS

The assistance of J.D. Fletcher, A. Kyriakou, H. Stengs and P. Murphy with inoculation of plants is gratefully acknowledged. Miss E. Stevenson is thanked for her assistance with analysis of data.

REFERENCES

Chiko, A.W., Zimmer, R.C. 1978. Effect of pea seed-borne mosaic virus on two cultivars of field pea grown in Manitoba. *Canadian Journal of Plant Science 58*: 1073-1080.

Fry, P.R., Young, B.R. 1980. Pea seed-borne mosaic virus in New Zealand. *Australasian Plant Pathology 9*: 10-11.

Gibbs, A.J., Gower, J.C. 1960. The use of a multiple-transfer method in plant virus transmission studies — some statistical points arising from the analysis of results. *Annals Applied Biology 48*: 75-83.

Hampton, R.O., Mink, G.I. 1975. Pea seed-borne mosaic virus. *C.M.I./A.A.B. Descriptions of Plant Viruses* No. 146.

Hampton, R.O., Mink, G.I., Bos, L., Inouye, T., Musil, M., Hagedorn, D.J. 1981. Host differentation and serological homology of pea seed-borne mosaic virus isolates. *Netherlands Journal of Plant Pathology 87*: 1-10.

Kraft, J.M., Hampton, R.O. 1980. Crop losses from pea seed-borne mosaic virus in six processing pea cultivars. *Plant Disease 64*: 922-924.

Mink, G.I., Parsons, J.L. 1978. Detection of pea seed-borne mosaic virus in pea seed by direct-seed assay. *Plant Disease Reporter 62*: 249-253.

Stevenson, W.R., Hagedorn, D.J. 1973. Further studies on seed transmission of pea seed-borne virus in *Pisum sativum*. *Plant Disease Reporter 57*: 248-252.

Yarwood, C.E. 1972. Virus transmission from *Chenopodium amaranticolor*. *Plant Disease Reporter 56*: 1085-6.

Yen, D.E.N., Fry, P.R. 1956. The inheritance of immunity to pea mosaic virus. *Australian Journal of Agricultural Research 7*: 272-80.