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Yield and quality of *Cucurbita maxima* increase with delayed infection by papaya ringspot virus type W

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Abstract

When four-week-old *Cucurbita maxima* cv. Queensland Blue plants with 5 to 6 expanded leaves were inoculated mechanically with papaya ringspot virus type W, the average yield per plant was only 3.4 kg. Inoculation five weeks later, when female flowering had commenced, resulted in a yield of 9.5 kg/plant, approximately the same as uninoculated plants (8.9 kg/plant). In a second experiment, plants which were infected early and expressed symptoms as 16-day-old plants yielded 2.3 kg/plant, while plants which were infected about 10 weeks later and expressed symptoms as 86-day-old plants yielded 10.9 kg/plant. As well as reducing yield by 62 to 79%, early infection reduced quality by distorting the fruit. The time (T) symptoms were first observed, the node at which a fruit was produced (N) and the severity of symptoms on the fruit (F) were related by $F=-0.097T-0.118N+0.0021TN+8.436$.

It is suggested that losses could be reduced by delaying infection, by using reflective mulch or by promoting rapid early growth before spread of the virus becomes active or by the use of resistant cultivars.

INTRODUCTION

Pumpkins (*Cucurbita maxima* Duch. and *Cucurbita moschata* Duch. ex Poir) are important culinary vegetables in Australia. During 1982–83 approximately 37 800 t, valued at $8.4m, were produced in Queensland (Anon. 1984). Papaya ringspot virus type W (PRSV-W=watermelon mosaic virus type 1) is considered a most serious disease which reduces yield and deforms fruit of *C. maxima* cv. Queensland Blue, a major cultivar grown in Queensland (Greber 1978). However, for this cultivar the reduction in yield and fruit quality caused by PRSV-W has not been quantified.

Differential reductions in components of yield and quality have occurred in other crops following infections at different stages of growth (Hiruki 1977). Negligible reductions in yield and quality are expected when infection is delayed until after maximum plant growth, fruit set and development (Komm and Agrios 1978; Halliwell *et al.* 1979; Thomas 1980). Greber (1978), (R. S. Greber, pers. comm. 1986) reported that fruit of plants of *C. maxima* cv. Queensland Blue grown in the greenhouse were most severely deformed when fruit set occurred about three weeks after infection by PRSV-W. By contrast fruit were almost normal when fruit set occurred about two months after infection. I investigated the effect of virus infection on fruit number and weight and the effects of time of infection in the field on yield and quality of *C. maxima.*

MATERIALS AND METHODS

Field experiments were carried out using *C. maxima* cv. Queensland Blue Large Strain (Anon. 1975) at Gatton Research Station (100 km west of Brisbane) during spring 1981, and using selfed progeny of *C. maxima* cv. Queensland Blue Selected Strain (Anon. 1975) at Redlands Horticultural Research Station (30 km south of Brisbane) during autumn 1982.

The Gatton experiment was a randomised complete block design with three replications of three treatments. Seedlings in a peat–vermiculite mix (Nahrung 1984) were transplanted to the field on 9 September 1981, nine days after incubation. Treatments were:

- Mechanical inoculation four weeks after transplanting seedlings when plants had 5 to 6 expanded leaves;
- Mechanical inoculation five weeks later when female flowering had commenced; and
- Uninoculated control.

Each plot had five plants spaced 2 m×5 m. Inoculum was prepared from freshly harvested leaves of *C. maxima* cv. West Australia Grey known to be infected with PRSV-W. Leaves were ground with 0.1 M sodium potassium phosphate buffer, pH 7.1, containing 0.1% sodium sulphite. At time of treatment two young expanded leaves of cv. Queensland Blue were dusted with 600 mesh carborundum and manually inoculated with infective sap extracts. Yields were taken 16 weeks after transplanting seedlings. Three plants which showed symptoms of virus infection prior to inoculation were deleted from the analysis. The virus in the eight leaf samples taken at final harvest was identified as PRSV-W by host range tests and serology (Greber 1978).

The experiment at Redlands was a randomised complete block design in six replications with seven treatments:

- An uninoculated control; and
- Mechanical inoculation at 8, 15, 29, 43, 57 and 71 days after incubation of the seed which began on 8 March 1982.

There were three plants per plot with 3 m spacings. Seedlings were transplanted to the field 11 days after incubation began. Cotyledons or 1 to 2 young leaves of *C. maxima* were dusted with 600 mesh carborundum abrasive and manually inoculated with PRSV-W at time of treatment (Table 1). At each time of inoculation the stage of development of 1 to 3 plants was recorded.

Table 1. Inoculation schedule for cv. Queensland Blue plants with papaya ringspot virus type W indicating treatment number, time of inoculation, stage of development of plant and tissue inoculated in the Redlands experiment

Treatment	Date inoculated	Time from seeding (days)	Tissue inoculated	Stage of development of plant	
				No of nodes±SE	No of leaves > 15 mm
1	16 Mar	8	cotyledon	6	0
2	23 Mar	15	leaf 2	10.5±0.5	2
3	6 Apr	29	leaf 5	24.5±0.5*	7
4	20 Apr	43	leaf 13	35.5±4.5†	19
5	4 May	57	young leaf	n.d.‡§	n.d.
6	18 May	71	young leaf	n.d.	n.d.
7	control		nil	n.d.	n.d.

* Female bud developing at node number 14.7±1.7.

† Ovary 7.2+0.9 mm in diameter.

‡ n.d. = not determined.

§ Female flower open.

Infectivity of inoculum was assessed by inoculating a sample of cucumber *Cucumis sativus* L. cv. Supermarket seedlings in the glasshouse. Inoculum was prepared from young leaves of infected plants of cucumber ground with three parts (w/v) of sodium potassium phosphate buffer 0.1 M, pH 7.0, containing 0.1% sodium sulphite. Inoculum was filtered through muslin.

On the day before the 43rd, 57th and 71st day treatments (Table 1), all symptomless plants to be inoculated in these treatments were indexed for virus using cucumber seedlings. A sample of approximately 3 g of a young leaf was ground in phosphate buffer (1:3 w/v) and the sap extract used to inoculate the cotyledons of five or six cucumber seedlings. Similarly, on the 57th day previously inoculated but symptomless plants from other treatments were indexed. The strain of virus present in some of the symptomless plants was identified using differential hosts (Greber 1978). Alate aphid populations were monitored weekly using five yellow pan traps (300×250 mm) spread throughout the field.

The severity of leaf symptoms on each plant was assessed seven times, at 16, 28, 42, 57, 71, 86 and 108 days after incubation of the seed. At 134 days after incubation each fruit was weighed and severity of viral symptoms was scored as 0 (no symptoms), 1 (minor distortion), 2 or 3 (substantial distortion but marketable depending on severity), 4 (severe distortion and unmarketable), 5 (very severely affected). The nodes from which fruit were harvested, and on the main stem those from which female buds or young fruit had aborted, were also recorded, up to the 43rd node from the cotyledonary scar.

Data from individual plants in the Redlands experiment were subjected to regression analysis, using the time at which leaf symptoms were first recorded as the independent variable. This independent variable was assumed to be closely related to the time of infection. As symptoms were assessed only once every 14 days low coefficients of determination could be expected in these regression relationships. Analysis of variance of these data was considered to be inappropriate because comparison of the treatments applied was confounded by natural spread, inefficient inoculation, and missing values created by the deletion of 48 plants with abnormal development unrelated to PRSV-W (R. S. Greber, pers. comm. 1982).

RESULTS

In the Gatton experiment, plants inoculated at the 5 to 6 leaf stage four weeks after transplanting produced less weight of fruit (3.4 kg/plant) than either those inoculated five weeks later (9.5 kg/plant) when female flowering had commenced or the uninoculated control (8.9 kg/plant) LSD (P=0.05)=4.8 kg/plant.

Similar results were obtained in the Redlands experiment. The earlier the time when symptoms were first observed (T), as days from incubation of seed, the greater the reductions in total yield (Y in kg), number of fruit (M), average weight per fruit (W in kg).

$$Y=0.125T+0.460 \ (r=0.56, \ P<0.01),$$

$$M=0.057T+1.3 \ (r=0.54, \ P<0.01),$$

$$W=0.01\mathrm{T}+0.99 \ (r=0.5, \ P<0.01).$$

At late times of symptom expression severity of symptoms of fruit (F) also increased with the distance of the node (N) from the cotyledons. T, F and N were associated (Figure 1) by:

$$F=-0.097T-0.118N+0.0021TN+8.436 \ (R^2=0.32;\ P<0.01).$$

However, on the mainstem the proportion of flower buds which developed into harvested fruit (H) was independent of the time of infection.

$$H=0.0002T+0.427 \ (r=0.02,\ P>0.05)$$

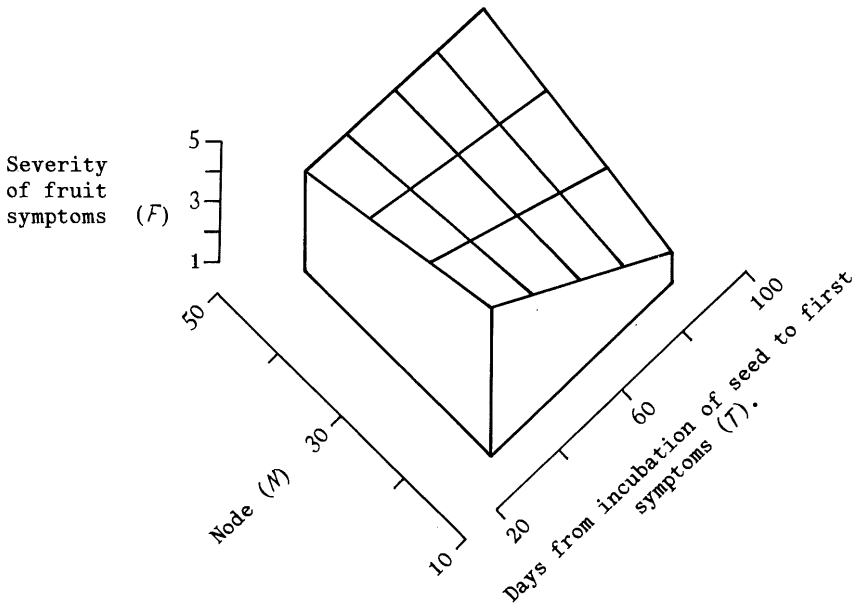

Figure 1. Relationship of the node at which fruit was produced (N), the time leaf symptoms were first recorded (T), and the severity of symptoms on fruit (F) produced on plants of *C. maxima* cv. Queensland Blue infected with PRSV-W.

DISCUSSION

The results of experiments at both Redlands and Gatton agree with the finding of Thomas (1980) in New Zealand that early infection with PRSV-W reduced yield of *C. pepo*, and further indicate that the earlier *C. maxima* cv. Queensland Blue is infected with PRSV-W the greater the reduction in yield. This reduction in yield is due to fewer and smaller fruit. These are probably both due to stunting of the plant following infection with the virus.

Greber (1978), (R. S. Greber, pers. comm. 1986) found that, in the greenhouse, fruit set about two months after infection were almost normal although those set about three weeks after infection were severely distorted. In the Redlands experiment fruit produced at distal nodes (measured from the cotyledon) following early infection were less deformed than those at proximal nodes. The reverse was true following late infection (Figure 1). With late infections, fruit at proximal nodes probably developed before viral infection of the plant and no fruit distortion would be expected. These results are consistent with those of Greber (1978).

The large losses in yield and quality following infection of cv. Queensland Blue with PRSV-W demonstrated in this experiment clearly indicate the need to adequately control

PRSV-W. Control might be achieved by strategies that delay infection relative to plant development. Potential strategies include earlier time of planting before spread of the virus becomes rapid, reflective mulches (McLean *et al.* 1982), resistant cultivars or perhaps high density plantings of early flowering cultivars. The use of tolerant or resistant cultivars may be the most economical solution in the long term.

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