The Journal of Heredity 73:239–240. 1982.

Resistance to papaya ringspot virus in *Cucumis metuliferus* and its relationship to resistance to watermelon mosaic virus 1

R. Provvidenti and D. Gonsalves

Abstract: In cross and backcross populations of PI 292190—an accession of *Cucumis metuliferus* (Naud.) Mey. resistant to papaya ringspot virus (PRVS)—with a susceptible line (Acc. 2459) of the same species, resistance to this virus was conferred by a single dominant gene. Clones of F_2 and testcross plants inoculated with PRSV or watermelon mosaic virus 1 (WMV-1) reacted identically, suggesting that the factor for resistance to PRSV is closely linked to *Wmv*, or may be the same factor. PRSV and WMV-1 are known to be closely related serologically. Acc. 2459 of *C. metuliferus* ('horned cucumber' or 'jelly melon') is a valuable host for the propagation of isolates of PRSV.

PAPAYA RINGSPOT VIRUS (PRVS) occurs commonly wherever papaya (*Carica papaya* L.) is cultivated[8]. This potyvirus is transmitted efficiently by several species of aphids[4] and usually spreads rapidly with destructive consequences[3,5,6,10,12,13]. PRSV also is able to infect several cucurbit species and some isolates incite symptoms resembling those caused by watermelon mosaic virus 1 (WMV-1). Although a close serological relationship between PRSV and WMV-1 has been demonstrated[2,9], WMV-1 does not infect papaya.

Recently, we found that genotypes of *Cucumis metuliferus* (Naud.) Mey., *C. melo* L., *C. sativus* L., *Lagenaria siceraria* (Mol.) Standl., and *Cucurbita ecuadorensis* Cuttler and Whitaker, which are resistant to WMV-1, also are resistant to PRVS isolates from Hawaii and Florida (unpublished data). Previously, Provvidenti and Robinson[7] reported that in *C. metuliferus* (commonly known as the 'horned cucumber' or 'jelly melon') resistance to WMV-1 is governed by a single dominant gene (*Wmv*). The purpose of this investigation was to elucidate the mode of inheritance of resistance to PRSV in this species and to determine whether the factors conferring resistance to PRSV and WMV-1 are linked.

Materials and Methods

The genetic background of the *C. metuliferus* germplasm utilized in this study was similar to that previously used to determine the inheritance of resistance to WMV-1[7]. Plants of Acc. 2459, susceptible to PRSV and WMV-1, were crossed with those of PI 292190, a line resistant to both viruses. Uniformity in germination was achieved by placing seed on moist blotters in plastic boxes, which were initially incubated at 5°C for 3–5 days and then to 30°C. Plants of F_1, F_2, and those of reciprocal backcross populations were mechanically inoculated when they had reached the two-leaf stage. To assure infection in all the susceptible genotypes, plants were reinoculated at the four-leaf stage. Inoculum was derived from *Cucurbita pepo* L. cv. Seneca Zucchini, or plants of Acc. 2459 systemically infected with the Hawaiian isolate PRSV-HA[2]. The same hosts were used as sources of inoculum for isolate NY69-49 of WMV-1[7]. Stock cultures of PRSV-HA and two other isolates PRSV-HB (Hawaii) and PRSV-Fla (Florida) were maintained in *C. papaya* cv. Solo.

Clones of F_2 and testcross plants were used to determine linkage between PRSV and WMV-1 resistance factors. Thus, each plant of these populations was separately tested with PRSV or WMV-1.

Enzyme-linked immunosorbent assay (ELISA) was employed in determining infectivity of PRSV or WMV-1 in inoculated plants[2]. An antiserum to PRSV-HA had been prepared by Gonsalves and Ishii[2], and that to WMV-1 was supplied by D. E. Purcifull (University of Florida). The work was conducted in an insect-free greenhouse maintained at 27°C.

Results

Plants of PI 292190 inoculated with PRSV-HA were free of local and systemic infection. Those of Acc. 2459, after an incubation period of 7–10 days, developed veinal chlorosis, mottle, reduction of leaf size and short internodes. Severely affected plants tended to wilt and died prematurely. Plants of Acc. 2459 responded with a very prominent mosaic to infection caused by PRSV-HB, and with a mild or moderate mottle to PRSV-Fla. Conversely, both PRSV-HB and PRSV-Fla incited only a few systemic chlorotic spots on Seneca Zucchini squash and after a longer incubation period (15–20 days). Isolate PRSV-HA caused more prominent symptoms in this same host and the incubation period was 12–15 days.

Plants of F_1 behaved as the resistant parent and no virus infection was detected in inoculated and uninoculated leaves. Plants of the F_2 generation segregated in a ratio of 3 resistant to 1 susceptible. Resistant plants were free of local and systemic infection, whereas susceptible plants exhibited symptoms identical to that of the susceptible parent. The progeny of the backcross to the resistant parent was all resistant. Plants of the testcross segregated in a ratio of nearly 1 resistant to 1 susceptible. From the data presented in Table I, it is evident that resistance is conferred by a single, completely dominant gene.

The segregation of clones of F_2 and testcross populations, which had been inoculated with PRSV or WMV-1, is reported in Table II. Plants that were resistant to PRSV also were resistant to WMV-1; conversely, those that were susceptible to PRSV also were susceptible to WMV-1. The segregation pattern for both populations approximated the expected ratios.

Discussion

This study has revealed that resistance to PRSV in *C. metuliferus* is monogenically dominant. Data from clones of F_2 and testcross plants indicate a close linkage between the factor for resistance to PRSV and *Wmv*, the gene for resistance to WMV-1[7]. However, no gene symbol has been assigned to the factor for PRVS resistance because it is possible that *Wmv* may be responsible for the resistance to both viruses. PRSV and WMV-1 share a number of common features: 1) they belong to the potyvirus group[8]; 2) they are very closely related serologically[2,9]; 3) they share a common, although not identical host range[9,13]; and 4) in several species, plants resistant to

Table I. Segregation in F_1, F_2, and reciprocal backcross populations of *Cucumis metuliferus* for resistance to papaya ringspot virus

Populations	No. plants resistant	No. plants susceptible	Expected ratio	Goodness-of-fit (*P*)
PI 292190	75	0		
Acc. 2459	0	70		
(Acc. 2459 × PI 292190) F_1	43	0		
(Acc. 2459 × PI 292190) F_2	194	68	3:1	0.72
(Acc. 2459 × PI 292190) F_1 × PI 292190	135	0		
(Acc. 2459 × PI 29219) F_1 × Acc. 2459	71	62	1:1	0.45

The authors are members of the Department of Plant Pathology, New York State Agricultural Experiment Station, Cornell University, Geneva, New York 14456.

Table II. Reaction of clones of F_2 and testcross populations of *Cucumis metuliferus* to papaya ringspot virus (PRSV) and watermelon mosaic virus 1 (WMV-1)

Populations	Virus	No. plants resistant*	No. plants susceptible†	Expected ratio	Goodness-of-fit (*P*)
(Acc. 2459 × PI 292190) F_2	PRSV	63	23	3:1	0.69
	WMV-1	63	23		
(Acc. 2459 × PI 292190) F_1 × Acc. 2459	PRSV	39	34	1:1	0.57
	WMV-1	39	34		

* Plants resistant to PRSV also were resistant to WMV-1
† Plants susceptible to PRSV also were susceptible to WMV-1

WMV-1 also are resistant to PRSV. A few years ago, Schroeder and Provvidenti[11] demonstrated that in *Pisum sativum* L. resistance to bean yellow mosaic virus (BYMV) and to watermelon mosaic virus 2 (WMV-2) is conditioned by the same gene (*mo*). Both BYMV and WMV-2 are potyviruses, have common hosts, and are serologically related[1]. Further work on the comparison of RNA nucleotide sequence homologies of PRSV and WMV-1 will eventually determine how closely related these two viruses are, and whether they should be considered strains of the same virus.

Wang et al.[13] reported that PI 292190 was susceptible to an isolate of PRSV from Taiwan. The difference in reaction could be attributed to a different strain of the virus, mixture of viruses, or a mislabeled seed lot. The sensitivity of *C. Metuliferus* Acc. 2459 to infection with isolates of PRVS, makes this a valuable line for propagation of this virus.

References

1. BRANDES, J. Identifizierung von gestreckten pflanzenpathologenen viren auf morphologischer Grundlage. *Mitteilungen Biol. Bundesanstalt Land Forstwirtschaften* 100:1-130. 1964.
2. GONSALVES, D. and M. ISHII. Purification and serology of papaya ringspot virus *Phytopathology* 70:1028-1032. 1980.
3. ISHII, M. Observations on the spread of papaya ringspot virus in Hawaii. *Pl. Dis. Reptr.* 56: 331-337. 1972.
4. JENSEN, D. D. Papaya ringspot virus and its insect vector relationships. *Phytopathology* 39: 212-220. 1949.
5. LASTRA, R. and E. QUINTERO. Papaya apical necrosis, a new disease associated with a rhabdovirus. *Pl. Dis. Reptr.* 65:439-440. 1981.
6. LIMA, J. A. A. and M. N. S. GOMES Identificacao de 'papaya ringspot virus' em Fortaleza, Ceara. *Fitossanidade* 1:56-59. 1975.
7. PROVVIDENTI, R. and R. W. ROBINSON. Inheritance of resistance to watermelon mosaic virus 1 in *Cucumis metuliferus*. *J. Hered.* 68.56-57. 1977.
8. PURCIFULL, D. E. Papaya ringspot virus. no. 84. *In* Description of Plant Viruses. Comm. Mycol. Inst. Assoc. Appl. Biol., Kew, Surrey, England. 1972.
9. —— and E. HIEBERT. Serological distinction of watermelon mosaic virus isolates. *Phytopathology* 69:112-116. 1979
10. SANCHEZ de LUQUE, C. and G. MARTINEZ LOPEZ. Reconocimiento del virus de la mancha anular de la papaya (*Carica papaya* L.) en Colombia. *Revista ICA* 11:205-220. 1976.
11. SCHROEDER, W. T. and R. PROVVIDENTI. A common gene for resistance to bean yellow mosaic virus and watermelon mosaic virus 2. in *Pisum sativum*. *Phytopathology* 61:846-848. 1971.
12. SUREKA, S. K., K. MATHUR, and D. D. SHUKLA. Virus diseases of Papaya (*Carica papaya*) in Udaipur. *Indian J. Mycol. Pl. Path.* 7:115-121. 1977.
13. WANG, H. L., C. C. WANG, R. J. CHIU, and M. SUN. A preliminary study of papaya ringspot virus in Taiwan. *Pl. Protect. Bull.* 20:133-140. 1978.

The Journal of Heredity 73:240-242. 1982.

Endosperm balance numbers among New Guinea-Indonesian *Impatiens* species

Toru Arisumi

ABSTRACT: The results of crosses among various plant accessions of *Impatiens* species from the New Guinea-Indonesian area support the endosperm balance number (EBN) hypothesis. With few exceptions, crosses among plants with equal EBN's succeeded and those between unequal EBN's failed. Existing cross data indicate that the present collection of New Guinea-Indonesian *Impatiens* is comprised of standard (2EBN) and tetraploid (4EBN) species.

THE ENDOSPERM BALANCE NUMBER (EBN) hypothesis was proposed by Johnston et al.[9] to explain endosperm development in interploidy-intraspecific and interspecific crosses. To test this hypothesis, a species to be used as a standard is arbitrarily assigned an EBN. Other species are assigned EBN's on the basis of their crossing behavior with the standard species. According to this hypothesis it is the EBN's that determine the effective ploidy in the endosperm, and for successful endosperm development the EBN's must be in a maternal:paternal ratio of 2:1. Johnston and Hanneman[10] used results of crosses among tuber-bearing *Solanum* species to support the EBN hypothesis. The authors[9,10] noted that other interactions also are important in determining endosperm development and that the 2:1 EBN ratio was a necessary but not sufficient condition for a successful cross.

The purpose of this study was to test the validity and predictive value of the EBN hypothesis using results of breeding experiments with *Impatiens* species from the New Guinea-Indonesian area. This group seemed to be good material for these tests because: 1) it was comprised of diverse species showing cytological as well as morphological differences; 2) the breeding behavior of most of the species and the colchicine-induced polyploids of their hybrids was known; and 3) some new plant accessions were available for testing the predictive value of the EBN's.

Materials and Methods

The species and hybrids used in the present tests and some that had been studied in the past are listed and described in Table I. The EBN's of these species and the references from which the EBN determinations were obtained are also listed in Table I with the eight new species listed at the end. Seven of these species were from the New Guinea-Indonesian area. One species, M28

The author is research plant geneticist with the Florist and Nursery Crops Laboratory, Horticultural Science Institute, USDA, ARS, Beltsville, MD 20705. He thanks Dr. Gilbert McCollum for his helpful suggestions and for reviewing the manuscript.