EVIDENCE OF A CLUSTER OF LINKED GENES FOR RESISTANCE TO PEA SEEDBORNE MOSAIC VIRUS AND CLOVER YELLOW VEIN VIRUS ON CHROMOSOME 6

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Previous research had established that there are at least three distinct strains or pathotypes of pea seedborne mosaic virus (PSbMV) and that resistance to each of these strains is controlled by a specific recessive gene (1,7). Thus, <u>sbm-1</u> is for resistance to the standard strain (PSbMV-ST); <u>sbm-2</u> and <u>sbm-3</u>, independently of each other, confer resistance to the lentil strain (PSbMV-L); and <u>sbm-4</u> confers resistance to PSbMV-P4 (3,10,11). The gene <u>sbm-1</u>, being closely linked to <u>wlo</u> (waxless adaxial surface of leaflets), was positioned by Gritton and Hagedorn on chromosome 6 (4). Provvidenti and Alconero demonstrated a close linkage between <u>sbm-2</u> and <u>mo</u> (10), for resistance to bean yellow mosaic virus (BYMV) (14) and watermelon mosaic virus 2 (WMV-2) (13), on chromosome 2 (5).

Tests by Provvidenti and Alconero (12) revealed that, of 40 plant introductions (P.I.'s) of Pisum sativum found to be resistant to PSbMV, 29 lines were resistant to PSbMV-ST, PSbMV-L, and PSbMV-P4. This association of resistance factors suggested that the genes sbm-1, sbm-3, and sbm-4 may be linked (12). The availability of eight PSbMV-resistant breeding lines developed by Muehlbauer (6) of the USDA-ARS and the Washington Agricultural Research Center, offered the opportunity of testing this hypothesis. These USDA lines had originated from backcrossing four times PSbMV-susceptible cultivars Tracer, Garfield, Alaska 4683, Campbells Scotch, Latah, Alaska, and Dark Skin Perfection with line WIS7105 carrying the sbm-1 gene. This factor had been derived from PI193586 and PI193835 (2), which later were shown also to possess sbm-3 and sbm-4, and one of the genes (cyv-2) for resistance to clover yellow vein virus (CYVV) (8). However, both P.I.'s were found to be susceptible to BYMV, pea mosaic virus (PMV), the NL8 strain of bean common mosaic (BCMV-NL8), and WMV-2 (R. Provvidenti, unpublished).

Twenty plants of each USDA line and the pertinent recurrent parent were tested with each of the following pathotypes or viruses: PSbMV-ST, PSbMV-L, PSbMV-P4, BYMV, CYVV, PMV, BCMV-NL8, and WMV-2. All plants were mechanically inoculated when they had reached the two-leaf stage and to avoid escapes, they were reinoculated later on the third and fourth leaves. Test plants were maintained in an insect-free greenhouse kept at 25-30°C.

From Table 1, it is evident that: 1) all the USDA lines were found to be homozygous resistant to PSbMV-ST, PSbMV-L, and PSbMV-P4; 2) all lines were either homozygous or heterozygous for resistance to CYVV; and 3) line X78006 was also resistant to BYMV, PMV, BCMV-NL8, and WMV-2. Considering that these USDA lines were never tested with PSbMV-L or PSbMV-P4 during their development, the retention of pertinent resistance genes can only be attributed to a very close linkage involving <u>sbm-1</u>, <u>sbm-3</u>, and <u>sbm-4</u> on chromosome 6. Similarly, the retention of resistance to CYVV after four backcrosses and selfing suggests that <u>cyv-2</u> may be linked to the genes for resistance to PSbMV.

44

Table 1. Reaction of eight USDA pea lines and pertinent recurrent parents to bean yellow mosaic virus (BYMV), clover yellow vein virus (CYVV), pea mosaic virus (PMV), strains of pea seedborne mosaic virus (PSbMV-ST, PSbMV-L, and PSbMV-P4), the NL-8 strain of bean common mosaic virus (BCMV-NL8), and watermelon mosaic virus 2 (WMV-2)

	Chromosome 2						Chromosome 6			
Virus	BYMV	CYVV	PMV	PSbMV	BCMV	WMV-2	PSbMV	PSbMV	PSbMV	CYVV
				-L	-NL8		-ST	-L	-P4	
Gene	<u>mo</u> *	cyv	pmv	sbm-2	Δ	mo	<u>sbm-1</u> **	sbm-3	sbm-4	cyv-2
X78006	R	R	R	R	R	R	R	R	R	R
Dk Sk Perf.	R	R	R	R	R	R	S	S	S	S
X78122	S	S	S	S	S	S	R	R	R	R
Scout	S	S	S	S	S	S	S	S	S	S
X78123	S	S	S	S	S	S	R	R	R	Н
Tracer	S	S	S	S	S	S	S	S	S	S
X78124	S	S	S	S	S	S	R	R	R	Н
Garfield	S	S	S	S	S	S	S	S	S	S
X78125	S	S	S	S	S	S	R	R	R	R
Alaska 4683	S	S	S	S	S	S	S	S	S	S
X78126	S	S	S	S	S	S	R	R	R	R
Campb. Sctch	S	S	S	S	S	S	S	S	S	S
X78127	S	S	S	S	S	S	R	R	R	Н
Latah	S	S	S	S	S	S	S	S	S	S
X76128	S	S	S	S	S	S	R	R	R	Н
Alaska	S	S	S	S	S	S	S	S	S	S

R = Resistant (no symptoms, ELISA negative)
S = Susceptible
H = Segregating
* Previously located on chromosome 2
** Previously located on chromosome 6

 Δ no gene symbol has been assigned for resistance to BCMV-NL8

Resistance to BCMV-NL8, BYMV, CYVV, PMV, WMV-2 was derived from the recurrent parent Dark Skin Perfection. Available evidence indicates the presence on chromosome 2 of a cluster of tightly linked genes: <u>mo</u> (for resistance to BYMV and WMV-2), <u>cyv</u> (for CYVV), <u>pmv</u> (for PMV), <u>sbm-2</u> (for PSbMV-L), and a resistance factor for BCMV-NL8 (5,8-10). Due to the presence in both parents of nonallelic resistance genes for CYVV and PSbMV-L, line X78006 appears to possess two genes for resistance to CYVV (<u>cyv/cyv</u> and cyv-2/cyv-2), and two for PSbMV-L (sbm-2/sbm-2 and sbm-3/sbm-3).

Thus, available evidence indicates a cluster of four linked genes on chromosome 6, three for resistance to three strains of PSbMV (<u>sbm-1</u>, <u>sbm-3</u> and <u>sbm-4</u>) and one for resistance to CYVV (<u>cyv-2</u>). The gene <u>wlo</u> is a useful marker for this cluster.

Alconero, R., R. Provvidenti and D. Gonsalves. 1986. Plant Dis. 70:783-786.
 Hagedorn, D.J. and E.T. Gritton. 1971. Crop Sci. 11:945-946.
 Hagedorn, D.J. and E.T. Gritton. 1973. Phytopathology 63:1130-1133.
 Gritton, E.T. and D.J. Hagedorn. 1975. Crop Sci. 15:447-448.
 Marx, G.A. and R. Provvidenti. 1979. PNL 11:2.
 Muehlbauer, F.J. 1983. Crop Sci. 23:1019.
 Provvidenti, R. 1987. PNL 19:48-49.
 Provvidenti, R. 1978. J. Hered. 78:126-128.
 Provvidenti, R. 1990. J. Hered. 81: 141-143.
 Provvidenti, R. and R. Alconero. 1988. J. Hered. 79:45-47.
 Provvidenti, R. and R. Alconero. 1988. J. Hered. 79:76-77.
 Provvidenti, R. and R. Alconero. 1988. PNL 20:30-31.
 Schroeder, W.T. and R. Provvidenti. 1970. Phytopathology 43: 11-15.
 Yen, D.E. and P.R. Fry. 1956. Aust. J. Agr. Res. 7:272-280.