Adh-1, A MARKER LOCUS FOR RESISTANCE TO PEA ENATION MOSAIC VIRUS

Weeden, N. F. and R. Provvidenti NYS Agricultural Experiment Station, Geneva, New York.

Resistance to pea enation mosaic virus (PEMV) is controlled by a single dominant gene, En (2). The gene lies about 8 recombinant units from tac on chromosome 3 (1). Several isozyme loci have been mapped to this region of the chromosome, and on the basis of relative map distances to various marker loci and clarity of phenotype, the locus Adh-1 appeared to be the best candidate for an isozyme marker for En (3).

Adh-1 encodes the monomers of the more anodal alcohol dehydrogenase isozyme in anaerobically treated seed or root tissue (4). A rare faster-migrating ADH-1 allozyme has been identified in the cultivar Alaska and line A683-168, both of which were obtained from G. A. Marx. These lines were both homozygous for en. They were crossed with the PEMV-resistant lines B880-221 and A76-46, respectively (also obtained from Dr. Marx). Three populations derived from these crosses were analyzed for ADH-1 phenotype and for reaction to PEMV: an Alaska x B880-221 F_2, an A79-46 x A683-168 F_2, and an F_3 from the Alaska x B880-221 cross. The F_3 consisted of the progeny from 3 doubly heterozygous plants from the Alaska x B880-221 F_2.

The data for segregation at the two loci are presented in Table 1. The F_3 data shown represent the combined data for the three progenies, but each progeny also showed normal segregation ratios at each locus. Table 2 presents the joint segregation analysis for the two loci. The 2-6% recombination frequency is low enough to allow Adh-1 to be a very useful marker for resistance to PEMV. As has been demonstrated previously (4), ADH-1 allozymes can be observed in seed tissue, permitting analysis of this isozyme before germination, if desired. The allele coding for the fast allozyme in Alaska and A683-168, because of its rarity in the cultivated germplasm, should make an excellent marker for En. We are currently developing homozygous lines that have this Adh-1 allele coupled with the resistance gene.

1. Marx, G. A., N. F. Weeden and R. Provvidenti. 1985. PNL 17:57-60.
2. Schroeder, W. T. and D. W. Barton. 1958. Phytopathology 48:628-632.
3. Weeden, N. F., G. A. Marx, and E. Pagowska. 1985. PNL 17:75-76.
4. Weeden, N. F. and E. Pagowska. 1985. PNL 17:79-80.

Table 1. Segregation at En, Adh-1, and Lap-1 in the F2 and F3 progeny examined.

Locus	No. Plants with designated Phenotype* fast/+	H	slow/-	Expected ratio	Goodness of fit (P)
Alaska x B880-221 (F2)					
En	37		15	3:1	0.53
Adh-1	14	26	12	1:2:1	0.91
Lap-1	7	25	15	1:2:1	0.29
A76-46 x A683-168 (F2)					
En	19	—	10	3:1	0.24
Adh-1	11	16	2	1:2:1	0.09
Lap-1	8	11	7	1:2: 1	0.95
Alaska x B880-221 (F3)					
	45	—	13	3:1	0.64
Adh-1	12	26	20	1:2:1	0.25

•Designations: dominant phenotype or homozygous fast = +; heterozygous = H; recessive phenotype or homozygous slow = -.

Table 2. Joint segregation of En and Adh-1 .

Progeny	No. Plants with designated Phenotype* +/+	+/H	+/-	-/+	-/H	-/-	Recomb. Fraction	S.E.
Alaska x B880-221 (F2)	0	25	12	14	1	0	2	2
A76-46 x A683-160 (F2)	1	16	2	10	0	0	3	3
Alaska x B880-221 (F3)	1	24	20	11	2	0	6	3

•Designations as described in Table 1.

*****