Prx-3 is linked to *sbm,* the gene conferring resistance to seedborne mosaic virus

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Resistance to the standard strain of pea seedborne mosaic virus (PSbMV) is conferred by a recessive gene, *sbm-1*, located on chromosome 6 (1). This gene has been incorporated into a number of commercial pea varieties, but the continuing occurrence of serious PSbMV infestations in many grower's fields indicates that resistance to this virus remains an important goal for many breeding programs. In addition, several genes conferring resistance to other potyviruses are clustered near *sbm-1* (2). Any marker for *sbm-1* also could serve as a marker for these genes.

The precise position of the locus, *Sbm-1*, recently has been questioned (3). Originally it was believed to be located near *Wlo*; however, Skarzynska (3) presented data suggesting that it was much closer to *Pl* than to *Wlo.* In a small (n=51) F_2 population segregating at *Wlo, Gpi-c, Pl* and *Sbm-1*, we failed to detect linkage between the first two loci and *Sbm-1*, but observed moderate linkage (r = 9 cM) between *Pl* and the resistance gene (Table 1). Thus, our results confirm those of Skarzynska, although our data showed a significant ($P < 0.001$) excess of the recessive phenotype at *Pl* which resulted in a high standard error for our linkage value.

Unfortunately, *Pl* would be an awkward marker for PSbMV resistance because it is dominant whereas *sbm-1* is recessive. Furthermore *Pl* (black hilum) is expressed only in seed and could not be used as an early screen. These problems are not characteristic of isozyme loci, which typically display codominant expression and can be detected in seedling tissue. Several isozyme loci have been identified on chromosome 6 between *Wlo and Pl* (5). The location of *Sbm-1* near *Pl* suggested that *Prx-3* might be a practical marker for the former locus.

We investigated this possibility by performing joint segregation analysis for *Sbm-1* and *Prx-3* on an F_2 population derived from the cross PI347492 x 'Bonneville'. *Prx-3* genotypes were determined on 1-2 week old seedlings before inoculation. The distal 2-3 cm of primary root were removed and crushed in 0.5 ml 0.1 M Tris-HCl pH 8.0 (this extraction buffer can contain other additives such as sucrose, polyvinyl pyrrolidione, or Triton X-100, but reducing agents such as 2-mercaptoethanol should be avoided). The extract was placed on an 11% starch gel as described in (4), using the Tris citrate/lithium borate buffer system. Electrophoresis was performed at 50 mA (200-300 V) at 5°C for about 4 hours. The filter paper wicks used for sample application were removed after 10 minutes. The cathodal portion of the gel was stained for peroxidase activity using the assay described in (4) except that two drops of 3% H_2O_2 were used per 30 ml assay instead of one drop 30% H_2O_2. Mechanical inoculations with the standard strain (PSbMV-ST) were performed by rubbing the first two

fully expanded leaves with an inoculum that was derived from infected Bonneville pea. To minimize escapes, all plants were inoculated a second time using the third and fourth leaves. These tests were conducted in an insect free greenhouse maintained at 28-30°C.

The analysis revealed a close linkage between the isozyme locus and expression of the resistant phenotype (Table 1), indicating that *Prx-3* might be a practical marker locus for *Sbm-1*. The allele encoding the fast PRX-3 allozyme is linked to the resistance gene in PI347492 accession we used as our resistant parent. However, the Wisconsin 7105 and Wisconsin 7106 lines released by Gritton and Hagedorn, are homozygous for the 'slow' *Prx-3* allele. In addition, susceptible lines homozygous for the 'fast' allele (e.g. 'Ranger') have been identified. In order to use PRX-3 phenotype as a marker, the susceptible parent must have a different allozyme than the resistant parent. Our survey of commercial pea varieties indicates that the PRX-3 "fast" allozyme is much more common. Thus, resistant germplasm displaying the "slow" PRX-3 allozyme should usually be selected as the source of PSbMV resistance if marker assisted selection is to be employed.

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1. Gritton, E.T. and Hagedorn, D. 1975. Crop Science 15:447-448.
2. Provvidenti, R. and Muehlbauer, F.J. 1990. PNL 22:43-45.
3. Skarzynska, A. 1988. PNL 20:34-36.
4. Weeden, N.F. and Marx, G.A. 1987. J. Heredity 78:153-159.
5. Wolko, B. and Weeden, N.F. 1990. PNL 22:71-74.

Table 1. Joint segregation analysis of loci on chromosome 6

Cross	No. progeny with designated phenotype							Rec.	S.E.
Loci	+/S	+/R	H/S	H/R	–/S	–/R	χ^2	fract.	
779/88 x 227258									
Pl : Sbm-1	24	3	--	--	3	21	29	9	14
PI347492 x Bonneville									
Prx-3 : Sbm-1	2	13	19	0	15	0	40	4	3

+ = Dominant or homozygous fast, H = heterozygous, – = recessive or homozygous slow, S = susceptible to PSbMV, R = resistant to PSbMV.