

Chromosomal distribution of genes for resistance to seven potyviruses in *Pisum sativum*

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Pisum sativum L. has been reported to be infected by a number of potyviruses, including bean common mosaic (NL8 strain) (BCMV-NL8), bean yellow mosaic (BYMV), clover yellow vein (CYVV), lettuce mosaic (LMV), pea mosaic (PMV), pea seed-borne mosaic (PSbMV), peanut mottle (PMoV), turnip mosaic (TuMV), watermelon mosaic 2 (WMV-2), and a newly characterized virus, tentatively named white lupin mosaic (WLMV). The genes for resistance to seven of these viruses are: *bcm* for BCMV-NL8 (15); *cyv-1* and *cyv-2* for CYVV (13); *mo* for BYMV and WMV-2 (22, 25); *pmv* for PMV (14); *sbm-1*, *sbm-2*, *sbm-3*, and *sbm-4* for strains of PSbMV (5, 17, 18), and *wlv* for WLMV (19). Some of these genes are strain-specific as are those for PSbMV. Others appear to be duplicate entities: *cyv-1* and *cyv-2* for any isolate of CYVV (13), and *sbm-2* and *sbm-3* for the lentil strain of PSbMV (17).

Table 1. List of known genes for resistance to potyviruses affecting *Pisum sativum*

Gene	Linkage Group	Virus and strain	Author and Year	Reference
<i>bcm</i>	2	Bean common mosaic (NL-8)	Provvidenti, 1991	15
<i>cyv-1</i> *	2	Clover yellow vein	Provvidenti, 1987	13
<i>cyv-2</i> *	6	Clover yellow vein	Provvidenti, 1987	13
<i>mo</i>	2	Bean yellow mosaic	Yen & Fry, 1950	25
<i>mo</i>	2	Watermelon mosaic 2	Schroeder & Provvidenti, 1971	22
<i>pmv</i>	2	Pea mosaic	Provvidenti, 1990	14
<i>sbm-1</i>	6	Pea seedborne mosaic (Standard)	Hagedorn & Gritton, 1973	5
<i>sbm-2</i> *	2	Pea seedborne mosaic (Lentil)	Provvidenti & Alconero, 1988	17
<i>sbm-3</i> *	6	Pea seedborne mosaic (Lentil)	Provvidenti & Alconero, 1988	17
<i>sbm-4</i>	6	Pea seedborne mosaic (P4)	Provvidenti & Alconero, 1988	18
<i>wlv</i>	6	White lupin mosaic	Provvidenti & Hampton, 1990	19

* = Possibly duplicate entities.

There are a number of pea cultivars and landraces in which these potyvirus resistance genes appear closely linked and clustered on two chromosomes. The first of these clusters is on chromosome 2 and comprises: *bcm*, *cyv-1*, *mo*, *pmv*, and *sbm-2* (13, 14, 17, 20, 21). The second cluster is on chromosome 6 and includes: *sbm-1*, *sbm-3*, *sbm-4*, *cyv-2*, and *wlv* (16, 20, 21).

The seven potyviruses have been reported to be directly or indirectly serologically related (7), but they differ in many essential features, such as host range, symptoms incited in pea and other hosts, lack of cross protection and low sequence homology in RNA/cDNA hybridizations (2). The three pathotypes of PSbMV are serologically related, but cause different symptoms on susceptible pea genotypes (1). Genetic factors for resistance to potyviruses in pea are all single recessive, conferring a very high level of resistance or immunity. They are also temperature insensitive, except for *mo*, which in the heterozygous condition can behave as dominant (< 20°C) or fully recessive (> 27°C) (14, 22, 23).

Genetic markers are available for these two gene clusters. The locus for the isozyme phosphoglucomutase (*Pgm-p*), and *K* (keel) are in close proximity to *mo* and consequently, to the other genes of the first cluster. For the second cluster, *wlo* (waxless) has been successfully used as a marker (4, 6, 8, 10, 24)

From a breeding point of view, the clustering of resistance genes represents an ideal situation, because during breeding for multiresistance, viral testing can be reduced to a minimum. However depending upon the resistant germplasm involved, testing for more than one virus is strongly recommended (17). The cluster present on chromosome 2 seems to occur in American and European cultivars known to be resistant to BYMV (13, 14) The cluster on chromosome 6 is found mainly in a number of foreign introductions from Ethiopia and India (16, 20). However, it is difficult to generalize, because there are lines from India and other countries carrying the first gene cluster (unpublished).

When it was determined that cultivars resistant to BYMV were also resistant to other viruses, the prevailing opinion was toward the pleiotropic effect of the *mo* locus. However, the discovery of a line from China (PI 391630) which is resistant to BYMV and WMV-2, but susceptible to the other viruses, suggested the possibility of specific, but closely linked genes (13). Since then, direct and indirect evidence has given more credence to the second hypothesis. For example: PI 269818 is resistant to BYMV, PSbMV-L and PSbMV-ST, but is susceptible to the other viruses; PI 193835 and 347492 are resistant to the three strains of PSbMV, and CYVV, but susceptible to BYMV, PMV, BCMV-NL8 and WMV-2; PI 193835 is resistant to WLMV, but PI 347492 is susceptible to this virus; PI 193836 is resistant to PSbMV-ST, but susceptible to the other two strains of the same virus; PI 337420 is resistant to PSbMV-L and susceptible to the other two strains; and PI 347329 is resistant to PSbMV-L and PSbMV-P4, but susceptible to PSbMV-ST (13, 15, 16, 17, 18, unpublished). These and a few other cases indicate that resistance is conditioned by specific genetic factors, but the pleiotropic effect of some of these genes cannot be completely excluded until tests reveal more differential lines.

The origin of gene clustering is open to debate. Demarly (3) speculated that these clusters or 'linkats' may be ancestral entities that, in the course of evolution, gave rise to duplicates that eventually moved to other loci. In our cases, it could be also hypothesized that loci for resistance to potyviruses originated as independent mutations in different linkage groups and through successive translocations converged on chromosome 2 or 6.

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