Table 1. Production of tertiary trisomics with different genotypes

Cross no.	Genotype ♀♀	Genotype ♂♂	Total progeny	Offspring trisomics: +T43H/++/(Rb1)++[a]	Offspring trisomics: T^{hp}+/+T43H/(Rb1)++[a]	Offspring trisomics: T^{hp}+/T^{hp}+/+T43H
1	T^{hp}+/+T43H	+/+	27	—	8	—
2	T^{hp}+/+T43H	+T^{hp}/Rb1+	35	—	5	2
3	+T^{hp}+/+T43H/Rb1++	+T^{hp}/Rb1+	13	—	3	—
4	+/+	+T^{hp}+/++T43H/Rb7++	12	—	6	—
5	+T^{hp}/Rb1+	+/+/T43H	38	11	—	—
6	T43H/+	+T^{hp}/Rb1+	9	2	2	—

[a] In crosses 2, 3, 5, and 6 = Rb1++; in crosses 1 and 4 = +++.

Table 2. Viability of mice that received different doses of the paternal and maternal alleles of the *Tme* gene covered by the T^{hp} deletion

Cross no.	Gametes: Maternal	Gametes: Paternal	Offspring: Genotype	Offspring: No. of maternal and paternal alleles[a]	Offspring: Viability[b]
1	T^{hp}	+	T^{hp}/+	1 Tme^{P}	lethal
2	+	T^{hp}	T^{hp}/+	1 Tme^{M}	viable
3	T^{hp}/+	+	T^{hp}/+/+	1 Tme^{M}, Tme^{P}	viable (16)
4	+	T^{hp}/+	T^{hp}/+/+	1 Tme^{M}, Tme^{P}	viable (6)
5	T^{hp}/+	T^{hp}	T^{hp}/T^{hp}/+	1 Tme^{M}	viable (2)
6	T^{hp}	+/+	T^{hp}/+/+	2 Tme^{P}	lethal
7	+/+	T^{hp}	T^{hp}/+/+	2 Tme^{M}	viable (2)

[a] Tme^{M} = the maternal and Tme^{P} = the paternal allele of the *Tme* gene.

[b] The number of trisomics produced is in parentheses.

gene located in the same region of the chromosome 17 (Ruvinsky and Agulnik, 1990). Further work at the molecular level would provide information about the mechanisms involved in the suppression of the activity of the paternal *Tme* gene and thereby would allow experimental manipulation of its properties.

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Inheritance of Resistance to the NL-8 Strain of Bean Common Mosaic Virus in *Pisum sativum*

R. Provvidenti

The NL-8 strain of bean common mosaic virus (BCMV-NL8) can infect *Pisum sativum* systemically without causing appreciable symptoms. The presence of the virus in susceptible genotypes can be ascertained by enzyme-linked immunosorbent assays (ELISA) or indicator hosts. Resistance to BCMV-NL8 was found in domestic cultivars known to be resistant to bean yellow mosaic virus (BYMV) and five other viruses. In crosses involving the multiresistant cultivar Bonneville and two susceptible lines, resistance to BCMV-NL8 was conferred by a single recessive gene, to which the symbol *bcm* is tentatively assigned. This gene is closely linked to a cluster of other resistance genes present on chromosome 2, conferring resistance to BYMV, clover yellow vein virus, pea mosaic virus, lentil strain of pea seed-borne mosaic virus, and watermelon mosaic virus 2. BCMV-NL8 was not detected in seeds of seven pea lines systemically infected with this virus.

In reporting the occurrence of the NL-8 strain of bean common mosaic virus (BCMV-NL8) in bean fields of western New York State in 1984, we noted that this strain is able to infect a number of pea cultivars without inciting appreciable symptoms (Provvidenti et al. 1984). BCMV-NL8, with the other African strains (BCMV-NL5 and BCMV-NL3) is classified as "temperature-independent necrosis-inducing" because it can cause a lethal necrosis in bean plants that possess the hypersensitive *II* gene (Drijfhout 1978; Drijfhout and Bos 1977). When compared with the American strains (US1, US2, and others), the African strains constitute also a rather distinct serological group (Serogroup A) (Wang et al. 1982). A high level of resistance to BCMV-NL8 was located in the domestic pea cultivars Bonneville, Perfected Freezer, Wando, Venus, and others known to be resistant to bean yellow mosaic virus (BYMV), clover yellow vein virus (CYVV), pea mosaic virus (PMV), the lentil strain of pea seed-borne mosaic virus (PSbMV-L), and watermelon mosaic virus 2 (WMV-2) (Provvidenti et al. 1984). Previous research had indicated that resistance to these potyviruses is conferred by specific but tightly linked genes clustered on chromosome 2: *mo* for BYMV and WMV-2 (Schroeder and Provvidenti 1970; Yen and Fry 1956), *cyv* for CYVV (Provvidenti 1987), *pmv* for PMV (Provvidenti 1990), and *sbm-2* for PSbMV-L (Provvidenti and Alconero 1988). The purpose of this study was to elucidate the inheritance of resistance to BCMV-NL8 in *Pisum sativum* L. and to determine whether the gene for resistance to BCMV-NL8 is linked to the other genes of that cluster.

Materials and Methods

Genetic populations (F_1, F_2, F_3, and backcrosses) had derived from crosses between the cultivar Bonneville × Ranger

and Bonneville × PI 391630. Bonneville is resistant to BYMV, CYVV, PMV, PSbMV, WMV-2, and BCMV-NL8, whereas Ranger is susceptible to these viruses. Conversely, PI 391630 is resistant to BYMV and WMV-2 but susceptible to the other viruses. I used F_3 families of the cross Bonneville × Ranger for linkage determination. I routinely maintained cultures of BYMV, CYVV, PMV, and PSbMV-L in Ranger pea but propagated BCMV-NL8 in Black Turtle 2 bean. All these viruses and antisera to them were available from previous studies (Provvidenti 1987; Provvidenti 1990; Provvidenti and Alconero 1988; Provvidenti et al. 1984). I prepared inocula by macerating young symptomatic leaves in a 0.05 M phosphate buffer (pH 8.5) and rubbed extracts on leaves of test plants that had been dusted with 400-mesh Carborundum. To minimize escapes, all plants received two consecutive inoculations, the first occurring when plants had reached the two-leaf stage and the second when the third leaf was fully expanded. Each test included controls consisting of inoculated and uninoculated plants of resistant and susceptible parents. Since BCMV-NL8 did not cause any appreciable symptoms on susceptible genotypes, I ascertained infectivity by direct enzyme-linked immunosorbent assays (ELISA) and/or by inoculations to Black Turtle 1 bean. This host responds to BCMV-NL8 with local necrotic lesions, systemic apical necrosis and premature death (Provvidenti et al. 1984). A Serotype A broad-spectrum monoclonal antibody (McAB#197-1) was obtained from G. I. Mink (Washington State University). Results were recorded by a Microelisa Auto Reader (MR 700, Dynatech Laboratories, Inc.). I considered optical density values (410 nm wavelength) more than twice the value of healthy controls to be significant. Considering that BCMV-NL8 is seed transmitted in *Phaseolus vulgaris* (Drijfhout and Bos 1977; Provvidenti et al. 1984), I conducted tests to determine whether this strain might also be seedborne in *P. sativum*. I harvested seeds from systemically infected plants of Ranger, PI 174519, PI 244054, PI 269818, PI 391630, PI 347462, and PI 347492, and then stored them for 1 year (at 4°C and 40% relative humidity). All pea plants were grown in an insect-free greenhouse at 28°C to 30°C.

Results

Plants of Bonneville, Ranger, and PI 391630 inoculated with BCMV-NL8 failed to develop any visible symptoms. ELISA and recovery tests confirmed that Bonneville is highly resistant to local and systemic infection, whereas Ranger and PI 391630 were systemically infected (susceptible). Infected plants of these two lines, when compared with uninoculated controls, did not show any significant difference in growth, leaf and plant size, or pod production. F_1 plants of Bonneville × Ranger and Bonneville × PI 391630 were symptomless carries of the virus. In F_2 populations of these two crosses, segregation was near an expected ratio of 3 susceptible : 1 resistant. The progenies of the backcross to the resistant parent segregated in the ratio 1 resistant : 1 susceptible, whereas the progenies of the backcrosses to the two susceptible parents were all susceptible. These results (Table 1) fit the hypothesis of a single recessive gene, to which the symbol *bcm* (bean common mosaic) is tentatively assigned.

Linkage Tests

Preliminary studies had shown that symptomless but systemically BCMV-NL8-infected plants could be reinfected easily with BYMV, CYVV, PMV, PSbMV-L, or WMV-2, reacting with prominent systemic symptoms. This lack of cross protection was exploited for linkage tests between *bcm* and *mo*. I reinoculated F_2 plants of Bonneville × Ranger, Bonneville × PI 391630 and BC (F_1 × Bonneville) that had been inoculated with BCMV-NL8 and were found to segregate for resistant and susceptible individuals (Table 1), with BYMV on the last three fully expanded leaves. Prominent systemic symptoms caused by the challenging virus were visible on all BCMV-NL8 susceptible plants after an incubation period of 6–10 days. Conversely, BCMV-NL8 resistant plants failed to develop any symptoms, and assays confirmed that they were not infected with BYMV. This identical segregation for resistance and susceptibility in the F_2 and BC populations strongly suggested that *bcm* and *mo* are closely linked. These two genes, moreover, are distinct entities because Bonneville is resistant to both BCMV-NL8 and BYMV, whereas PI 391630 is resistant to BYMV but susceptible to BCMV-NL8. Previously, it had been demonstrated that the *mo* gene is located on chromosome 2 (Marx and Provvidenti 1979) and that Bonneville and PI 391630 possess this factor (Provvidenti 1987).

To determine linkage between *bcm* and *cyv, pmv,* and *sbm-2,* I randomly divided 12 to 16 F_3 plants of 84 Ranger × Bonneville F_3 families into four groups. I inoculated each group with one of the following viruses: BCMV-NL8, CYVV, PMV, and PSbMV-L. The data reported in Table 2 show that families resistant or susceptible to BCMV-NL8 were also resistant or susceptible, respectively, to the other three viruses. Families segregating for BCMV-NL8 also segregated for CYVV, PMV, and PSbMV.

Seed Transmission Tests

I assayed plants of Ranger, PI 174519, PI 244054, PI 269818, PI 391630, PI 347462, and PI 347492, that had derived from seeds of BCMV-NL8-infected plants for seedborne infection in the 12-leaf stage. When I used both ELISA and Black Turtle 1 bean as the indicator host, none of the 312 plants of the seven lines was found to be infected with BCMV-NL8.

Discussion

NL-8 is the only strain of BCMV able to infect *P. sativum*. The testing of hundreds of pea lines of domestic and foreign origin did not reveal any genotype that showed conspicuous foliar symptoms or stunting after inoculation with this strain (Provvidenti R, unpublished data). Hence, BCMV-NL8 appears to be of no economical importance for the pea crop and should not constitute a threat unless circumstances change. In that event, BCMV-NL8 can be controlled easily by adopting resistant cultivars.

While my tests have shown that *bcm* is a distinct entity from *mo,* there was no way to demonstrate that it differs from *cyv-1, pmv,* or *sbm-2,* to which it is closely linked. Although we tested a number of domestic pea lines, in no case did we find resistance to BCMV-NL8 alone; it was always associated with those for resistance to CYVV, PMV, and PSbMV-L. However, there are some plant introductions from Ethiopia and India (e.g., PI 193835 and PI 347492) that are resistant to CYVV and PSbMV-L but susceptible to BCMV-NL8. This resistance is located on chromosome 6 and it is conferred by *cyv-2* and *smb-3,* which appear to be duplicate entities of those found on chromosome 2 of domestic cultivars (Provvidenti 1987; Provvidenti and Alconero 1988; Provvidenti and Muehlbauer 1990). Hence, there is indirect evidence that resistance to BCMV-NL8 does not depend on the genes for resistance to CYVV or PSbMV-L. A further search among plant introductions could reveal different pea lines that possess specific genes for each of these potyviruses. All the potyviruses

Table 1. Segregation ratios of cross and backcross populations of *Pisum sativum* lines resistant and susceptible to the NL-8 strain of bean common mosaic virus

Genotype	No. plants		Expected ratio	Goodness of fit (probability)
	Resistant	Susceptible		
Bonneville	35	0		
Ranger	0	40		
PI 391630	0	87		
Bonneville × Ranger				
F_1	0	25		
F_2	39	131	1:3	.55
BC (F_1 × Bonneville)	25	31	1:1	.44
BC (F_1 × Ranger)	0	71		
Bonneville × PI 391630				
F_1	0	25		
F_2	28	99	1:3	.46
BC (F_1 × Bonneville)	37	46	1:1	.33
BC (F_1 × PI 391630)	0	63		

that infect pea have been reported to be directly or indirectly serologically related (Hollings and Brunt 1981), but they differ in many essential features, such as host range, symptoms incited in pea and other hosts, lack of cross protection, and RNA/cDNA hybridization, which has revealed a low sequence homology among some of these viruses (Barnett et al. 1987). Given that BCMV-NL8, CYVV, PMV, and PSbMV are distinct entities, it is safe to assume that resistance to them is conferred by different genes. A previous study proved that BYMV and WMV-2 share *mo* as a common gene for resistance (Schroeder and Provvidenti 1970), but, for lack of evidence, it would be incorrect to attribute resistance to BCMV-NL8 to one of the other genes (*cyv, pmv,* or *sbm-2*). Thus, in this article I am tentatively suggesting the symbol *bcm* for the single recessive gene that governs resistance to BCMV-NL8.

This investigation completes my studies regarding an apparent cluster of five resistance genes (*bcm, cyv, mo, pmv,* and *sbm-2*) on chromosome 2 of *P. sativum.* The close proximity of these resistance factors offers substantial advantage in developing multiresistant cultivars. Viral testing can be easily limited to one of the six viruses involved: BCMV-NL8, BYMV, CYVV, PMV, PSbMV-L, and WMV-2. This task can be facilitated by using the gene *Pgm-p* for the isozyme phosphoglucomutase, a marker situated two recombinant units from *mo* (Weeden et al. 1984). This gene cluster is usually found in domestic pea cultivars and some foreign introductions resistant to BYMV, but it will be unwise to generalize and assume that all lines resistant to BYMV are also multiresistant. As was mentioned in this paper and reported in others, there are lines that are resistant to BYMV and WMV-2 but susceptible to the other four viruses (e.g., PI 391630 and PI 269818). Some lines are resistant to CYVV but susceptible to BYMV (e.g., PI 193586, PI 193835, PI 347464), and presumably further screening, particularly among lines from regions of great genetic diversity, may reveal valuable differentials for each virus. But, even when two pea lines are resistant to the same isolate of a given virus, it should not be assumed that the same genetic factor is involved. Previous studies reported that there are two independently inherited sets of genes for CYVV and PSbMV. Of these, *cyv-1* and *sbm-2* are on chromosome 2, whereas *cyv-2* and *sbm-3* appear to be located on chromosome 6 (Provvidenti 1987; Provvidenti and Alconero 1988; Provvidenti and Muehlbauer 1990).

Table 2. Reaction to the NL-8 strain of bean common mosaic virus (BCMV-NL8), clover yellow vein virus (CYVV), pea mosaic virus (PMV), and the lentil strain of pea seedborne mosaic virus (PSbMV) in 84 F_3 families of the cross Bonneville × Ranger

Virus	No. families[a]			Expected ratio	Goodness of fit (probability)
	Resistant	Heterozygous (1 resistant: 3 susceptible)	Susceptible		
BCMV-NL8 CYVV PMV PSbMV-L	17	47	20	1:2:1	.50

[a] For each family, 12 to 16 plants were tested with each virus.

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