we describe, monocentric loops and dicentric loops, can also contribute to chromosomal abnormalities at anaphases I and II. Figure 4 shows the consequences at anaphases I and II of cross-overs at selected points inside and outside a monocentric loop. These cross-overs may occur in one or a combination of the following sites: 1) between the external centromere and the loop; 2) inside the loop and between the centromeres; and 3) within the loop and distal to both centromeres. The following features may occur at anaphase I (see Figure 4): 1) single bridge with one fragment, 2) chromosome loop with one fragment, 3) double bridge with two fragments, or 4) "V bridge" with two fragments. Most of these features arising from monocentric loops are similar to those arising in maize from cross-overs involving paracentric loops, as studied by McClintock.[10] However, the "V bridge" arises as a result of cross-overs at positions inside and outside the loop (Figure 4). When these cross-overs occur, a chromosome is formed that possesses three centromeres: two sister and one nonsister. At anaphase I, the sister centromeres of this chromosome would tend to move to the same spindle pole while the nonsister centromere would move to the opposite pole. Anaphase II features arising from cross-overs in monocentric inversion loops are similar to those described by McClintock for paracentric loops (Figure 4). The chromosome loop formation at anaphase I would probably give rise to a bridge at anaphase II, as in McClintock's study.[10] V bridges have not been observed in meiotic cells of pigeonpea × *Atylosia* hybrids, nor have we found such bridges described in the literature.

Dicentric inversion loops can also lead to bridges at anaphase I. If a cross-over occurs between the centromeres, a chromatin bridge and fragment can be formed at anaphase I. This situation differs from that in pericentric loops, which also have both centromeres within the loop. Cross-overs in pericentric loops should lead to the formation of genetically imbalanced gametes rather than chromatin bridges.

The occurrence of inversion loops has important implications for pigeonpea improvement programs. Wild relatives of economic plants may possess valuable genetic traits for improvement of the cultivated species. However, the usefulness of this wild germ plasm depends on the interchange of genetic material between homoeologous chromosomes. Inversion loops can reduce the effectiveness of crossing-over between pairing chromosomes by reducing the level of synapsis of the pairing chromosomes and through sterility of cross-over products.[18] It should be stressed, however, that the chromosomes of pigeonpea and *Atylosia* do pair to form bivalents, so that gene interchange occurs even though some sterility results owing to the presence of inversions. The close pairing of homoeologous chromosomes on noninverted chromosome segments at pachytene indicates a high likelihood of gene transfer from the wild species to the pigeonpea.

From the Department of Agronomy, Waite Agricultural Research Institute, University of Adelaide, Glen Osmond, South Australia (Dundas); the Department of Agriculture, University of Queensland, St. Lucia (Britten); Queensland Agricultural College, Lawes via Gatton (Byth); and NewDor Consultancy Pty. Limited, Chippendale, New South Wales (Gordon), Australia. This work was supported in part by the International Crops Research Institute for the Semi-Arid Tropics (ICRISAT) and the Australian Centre for International Agricultural Research (ACIAR). Dr. Dundas thanks the Australian Government for a Commonwealth Postgraduate Research Award. The authors thank Mr. I. B. Staples, Queensland Department of Primary Industries, Mareeba, for the *Atylosia* seed. Address reprint requests to Dr. Dundas, Department of Agronomy, Waite Agricultural Research Institute, Glen Osmond, S.A., 5064, Australia.

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Inheritance of Resistance to Pea Mosaic Virus in *Pisum sativum*

R. Provvidenti

The high level of resistance to pea mosaic virus in the pea cultivar Bonneville is conferred by a single recessive gene. This factor, tentatively designated *pmv*, is closely linked to *mo*, *cyv*, and *sbm-2*, which confer resistance to bean yellow mosaic virus, clover yellow mosaic virus, and the lentil strain of pea seed-borne mosaic virus, respectively. These four genes are part of a cluster situated in linkage group 2. In the heterozygous condition *mo* was influenced by temperature, but *cyv*, *pmv*, and *sbm-2* were not.

Pea mosaic virus (PMV) is a member of the potyvirus group, which includes the following viruses also able to infect pea (*Pisum sativum* L.): bean yellow mosaic (BYMV), clover yellow vein (CYVV), lettuce mosaic (LMV), pea seed-borne mosaic (PSbMV), peanut mottle (PMoV), turnip mosaic (TuMV), and watermelon mosaic 2 (WMV-2).[7] PMV can be distinguished from the others by serology, cDNA, host range, and the bright yellow mosaic on susceptible pea genotypes.[1,4,6,9,11]

PMV was first noted by Doolittle and Jones[5] and subsequently characterized by Pierce[11] and others.[3,10,19,23] From their findings, it was evident that pea cultivars resistant to BYMV were also resistant to PMV.[4,11,20,23] This dual viral resistance in pea was subsequently reported by a number of other workers.[1,2,5,16] In 1956, Yen and Fry[22] disclosed that a single recessive gene (*mo*) was responsible for resistance to a

Table 1. Segregation ratios of cross and backcross populations of *Pisum sativum* lines resistant and susceptible to pea mosaic virus (PMV)

Genotype	No. plants		Expected ratio	Goodness of fit (probability)
	Resistant	Susceptible		
Bonneville	45	0		
Ranger	0	51		
PI 391630	0	42		
Bonneville × Ranger				
F_1	0	18		
F_2	39	129	1:3	.61
BC (F_1 × Bonneville)	30	37	1:1	.41
BC (F_1 × Ranger)	0	41		
Bonneville × PI 391630				
F_1	0	27		
F_2	23	83	1:3	.45
BC (F_1 × Bonneville)	40	49	1:1	.35
BC (F_1 × PI 391630)	0	37		

pea mosaic virus occurring in New Zealand. A similar mode of inheritance was demonstrated by Johnson and Hagedorn[8] for an isolate of BYMV from Wisconsin. In 1964, Barton et al.,[2] using pea clones of segregating F_2 populations, concluded that resistance to PMV and BYMV was conditioned by *mo*. Other researchers also have reported that cultivars resistant to BYMV and PMV are resistant to CYVV, PSbMV-L, WMV-2, and the NL-8 strain of bean common mosaic virus (BCMV-NL8),[1,13-15,18] suggesting a common genetic factor for multiresistance. However, the discovery of a pea line from China (PI 391630) that was resistant to BYMV but susceptible to the other viruses implied that resistance may be governed by distinct genes.[13]

Recent findings have established that in the BYMV-resistant cultivar Bonneville, resistance to CYVV and PSbMV-L is governed by the recessive genes *cyv* and *sbm-2*, respectively.[13,14] These genes are closely linked to *mo*, which also confers resistance to WMV-2 and is located on chromosome 2.[10,18] My aim was to elucidate the inheritance of resistance to PMV in Bonneville and demonstrate that genes for resistance to PMV, BYMV, CYVV, and PSbMV are tightly linked.

Materials and Methods

Genetic populations were derived from crosses between the cultivars Bonneville × Ranger and Bonneville × PI 391630. Bonneville is resistant to BYMV, PMV, CYVV, PSbMV-L, BCMV-NL8, and WMV-2, whereas PI 391630 is resistant only to BYMV.[13-16,18] The cultivar Ranger is susceptible to these and other viruses. F_1 plants of Bonneville × Ranger also were used to determine the effect of temperature on the expression of symptoms incited by the aforementioned viruses.[19] F_3 families of the same cross were employed for linkage determination. Isolates of PMV, BYMV, CYVV, and PSbMV, available from previous studies,[13-16,18] were maintained in Ranger pea. Inocula were prepared by grinding infected pea tissue in a 0.05 M sodium phosphate buffer, pH 8.5, and then rubbing the extracts on leaves dusted with Carborundum. The first inoculation was accomplished when test plants had reached the two-leaf stage; then they were reinoculated on the third fully expanded leaf. Two consecutive inoculations minimized escapes among susceptible genotypes. Each test included inoculated and uninoculated parental plants. Resistance or susceptibility of test plants was confirmed by direct enzyme-linked immunosorbent assay (ELISA) using antisera available from previous studies.[13-16] Results were recorded by a Microelisa Auto Reader (MR 700, Dynatech Laboratories, Inc.). Optical density values (410 nm wavelength) more than twice the value of healthy controls were considered significant. Unless otherwise noted, all test plants were maintained at 28°C to 30°C in an insect-free greenhouse. The effect of temperature on the heterozygous plants inoculated with PMV, BYMV, CYVV, or PSbMV-L was determined in two growth chambers kept at 18°C and 28°C.

Results

Inheritance Studies

Bonneville plants inoculated with PMV failed to develop local or systemic symptoms, and ELISA confirmed their high level of resistance or immunity to this virus. Conversely, Ranger and PI 391630 plants displayed the typical brilliant yellow mosaic associated with PMV infection. However, the incubation period was 4 to 6 days for Ranger and 10 to 12 days for PI 391630. F_1 plants of Bonneville × Ranger and Bonneville × PI 391630 crosses were susceptible and exhibited symptoms identical to those displayed by susceptible parents. The incubation period also was similar to that of the susceptible parents. In F_2 populations of the Bonneville × Ranger and Bonneville × PI 391630 crosses, segregation was close to the ratio of 3 susceptible : 1 resistant, and further evidence of recessiveness was obtained with reciprocal backcrosses. Plants of Bonneville × F_1(Bonneville × Ranger) and Bonneville × F_1(Bonneville × PI 391630) segregated in a ratio of 1 resistant : 1 susceptible. Conversely, for the backcrosses in which Ranger and PI 391630 were the recurrent parents, all plants were susceptible. The data shown in Table 1 clearly demonstrate that the high level of resistance to PMV in Bonneville is conferred by a single recessive gene.

Linkage Studies

F_3 plants of 58 Bonneville × Ranger families were randomly divided into four groups and then mechanically inoculated with PMV, BYMV, CYVV, or PSbMV. As shown in Table 2, families that were resistant or susceptible to PMV were also resistant or susceptible to BYMV, CYVV, and PSbMV. Families that segregated for PMV also segregated for the other three viruses.

Effect of Temperature on Heterozygous Plants

F_1 plants of Bonneville × Ranger and both parents were divided in four groups of 12 plants each and subsequently inoculated with PMV, BYMV, CYVV, or PSbMV-L. Plants of each group were then randomly subdivided in two sets of six plants each and placed in two growth chambers maintained at 18°C and 28°C with 14 h illumination. At 28°C, systemic symptoms appeared within 5 days in all the F_1 and Ranger plants, whereas Bonneville remained resistant. After an incubation period of 8 to 12 days at 18°C, F_1 and Ranger plants inoculated with PMV, CYVV, and PSbMV developed systemic symptoms. Conversely, Bonneville and F_1 plants inoculated with BYMV remained symptomless. Ranger developed symptoms typical for all the other viruses. Assays, however, revealed that the F_1 plants were infected

Table 2. Reaction to bean yellow mosaic virus (BYMV), clover yellow vein virus (CYVV), pea mosaic virus (PMV), and the lentil strain of pea seed-borne mosaic virus (PSbMV-L) in 58 F_3 families of the cross Bonneville × Ranger

Virus	No. families[a] Resistant	Heterozygous (1 resistant: 3 susceptible)	Susceptible	Expected ratio	Goodness of fit (probability)
BYMV, CYVV, PMV, PSbMV-L	11	31	16	1:2:1	.52

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

with BYMV and that those of Bonneville were not.

Discussion

For many years PMV and BYMV were considered to be distinct entities mainly because of PMV's inability to infect cultivars of *Phaseolus vulgaris*.[4,6,11,18,23] In 1966, Schroeder and Provvidenti[16] demonstrated that a few bean cultivars were susceptible to PMV, developing symptoms similar to those caused by BYMV. Subsequently, they determined that a single dominant gene (*By*) was responsible for the lack of infection in other bean cultivars.[17] Serological tests also showed a certain relationship between these two viruses,[9] and so PMV was referred to as the pea mosaic strain of BYMV.[3] The recent work of Barnett et al.[1] with RNA/cDNA hybridization, however, has revealed a low sequence homology between PMV and BYMV. Quantitative ELISA also indicated a distant relationship between these two viruses.[1] In view of these latest findings and the existence of specific genetic factors for resistance to PMV in bean and pea, these two viruses once again should be considered distinct entities.

Yen and Fry[22] assigned the symbol *mo* to the gene for resistance to a pea mosaic virus occurring in New Zealand. However, the strain used in their study must be considered to be BYMV; it was similar to that characterized by Chamberlain[4] but differed in that it was able to infect French bean. Hence, I propose to retain the symbol *mo* for BYMV resistance. As mentioned above, PMV differs in many ways from CYVV and PSbMV, and it is safe to assume that specific genes confer resistance to each of these viruses. Consequently, I am tentatively assigning the symbol *pmv* to the recessive gene for resistance to the typical isolate of PMV used in this investigation.[16,17]

This study provides further evidence of a cluster of tightly linked genes (*mo, cyv, pmv,* and *sbm-2*) on chromosome 2 that are resistant to BYMV, CYVV, PMV, PSbMV-L, and WMV-2. The *mo* gene is known to govern resistance to two of these viruses, BYMV and WMV-2.[18] Available evidence also suggests the presence of another genetic factor in the same cluster that confers resistance to BCMV-NL8.[15] The proximity of these genes simplifies the development of new virus-resistant cultivars. This task can be easily expedited using the isozyme locus *Pgm-p* (Phosphoglucomutase) as a marker, as it was found to be located on chromosome 2, two recombinant units from *mo*.[21]

This and other studies[18,19] demonstrate that *mo* in the heterozygous condition is affected by temperature. When inoculated with BYMV or WMV-2, plants with the *mo/+* genotype appear to be resistant (no symptoms) at 18°C and susceptible (mosaic) at 28°C.[18,19] The advantage resulting from this "phenotypical dominance" was illustrated and exploited in two previous studies.[12,19] Conversely, plants possessing *pmv/+* as well as *cyv/+* and *sbm-2/+* genotypes are not affected by temperature when inoculated with the pertinent viruses. The response to temperature of *mo/+* plants is further proof that this gene differs from the others to which it is closely linked.

From the Department of Plant Pathology, Cornell University, New York State Agricultural Experiment Station, Geneva, New York. Address reprint requests to Dr. Provvidenti, Department of Plant Pathology, Cornell University, New York State Agricultural Experiment Station, Geneva, NY 14456.

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