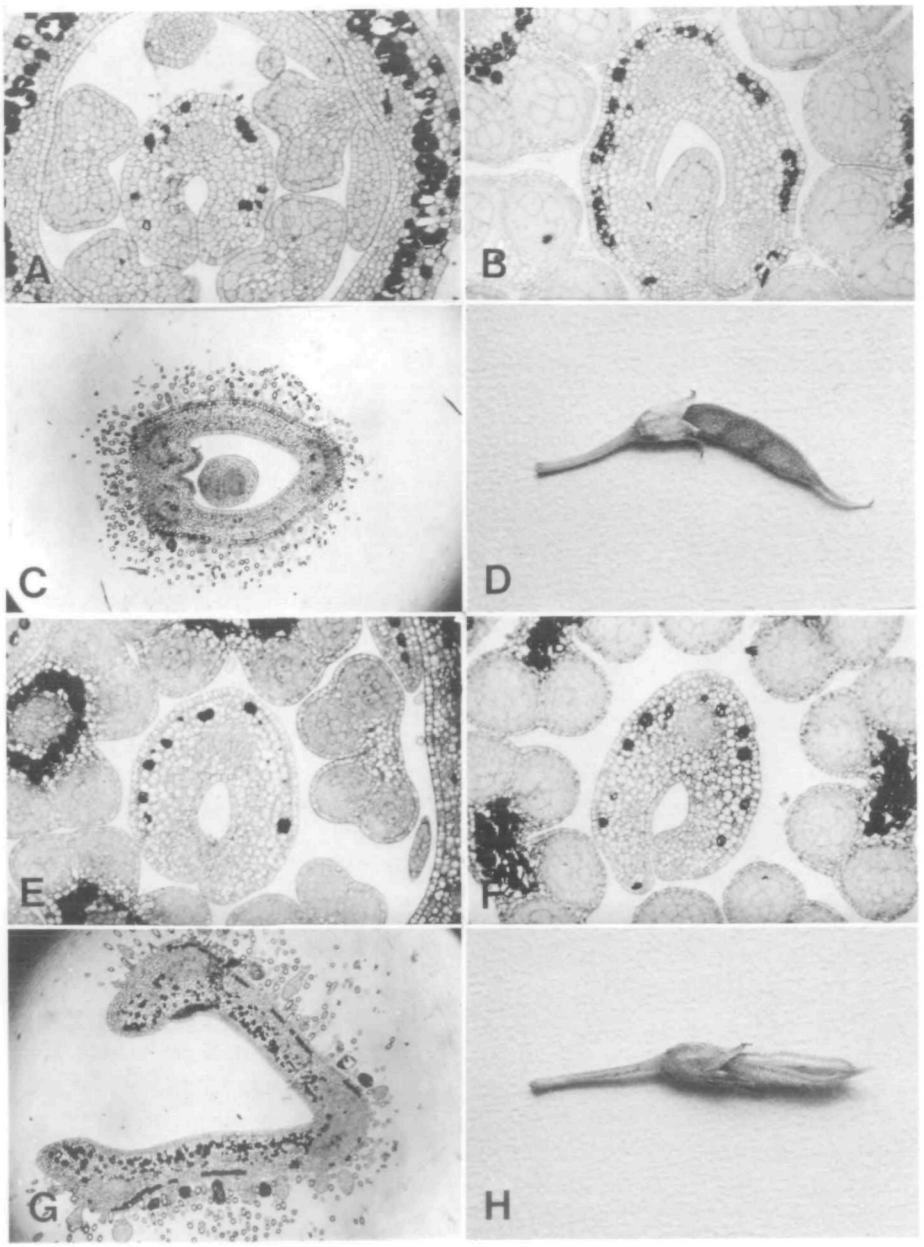

Figure 1. (a–d) The stages of development of the carpel in a normal pigeonpea flower. (e–h) The corresponding stages of development of the carpel in a flower of the defective mutant.

horseshoe-shaped primordium are obliquely placed (Figure 1e) and do not fuse (Figure 1f). This abnormality hampers the normal development of the ovule primordium and results in gradual degeneration of the ovule. Finally, the carpel falls open because of the nondevelopment of a ventral suture and does not form any ovules (Figure 1g).

Genetics

The F_1, F_2, and BC_1 populations could be easily classified into normal and mutant types. All F_1 plants in both crosses produced normal carpels and ovules, thus indicating the recessive nature of the mutant gene. The F_2 populations of both crosses segregated into normal and mutant types fitting into the expected monogenic ratio of 3:1 (Table 1). These data suggest that the open carpel mutant identified in cultivar ICPL 24 is controlled by a single recessive gene. This monogenic segregation ratio was confirmed in a testcross involving the parent ICPL 1 (Table 1). The testcross population segregated into 33 normal to 30 mutant plants, fitting well the expected ratio of 1:1.

Such defective mutants have no economic value but can serve as an effective tool in understanding the morphogenesis and development of the carpel and ovary. This represents a case of evolutionary retrogression where the carpel has reverted back to a leaf-like structure. This mutant also provides further evidence for the evolutionary hypothesis that the flower is a modified leaf. The symbol *cdl* is proposed for this gene.

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Inheritance of Resistance to a Third Pathotype of Pea Seed-Borne Mosaic Virus in *Pisum sativum*

R. Provvidenti and R. Alconero

Resistance to a newly recognized pathotype of pea seed-borne mosaic virus, PSbMV-P4, was found in PI 347492, an accession of *Pisum sativum* from India. In cross and backcross populations between PI 347492 and the susceptible cultivars Bonneville, Ranger, and PI 269816, resistance was determined to be monogenically recessive. The symbol *sbm-4* is proposed for the gene conferring resistance to this pathotype of PSbMV.

Recently, Alconero et al characterized a newly recognized pathotype of pea seed-borne mosaic virus, PSbMV-P4.[1] This pathotype was recovered from PI 471128, an accession of *Pisum sativum* L. from India. Serologically, PSbMV-P4 was indistinguishable from PSbMV-ST and PSbMV-L, the other two known pathotypes of the virus, but it could be easily differentiated using resistant and susceptible pea genotypes.[1]

Previous studies demonstrated that resistance in the pea to PSbMV is viral-pathotype specific.[1,6] Consequently, for effective control of this virus, it is necessary to utilize all the available genes for resistance. However, successful breeding programs depend upon the understanding of the genetics of each resistance factor. The aim of this investigation was to elucidate the inheritance of resistance to PSbMV-P4,

Table 1. Reaction of resistant and susceptible pea lines and their crosses and backcrosses to pea seed-borne mosaic virus, pathotype PSbMV-P4

Pea lines and their crosses	No. of plants		Exp. ratio	Goodness-of-fit (probability)
	Resistant	Susceptible		
Bonneville	0	35		
Ranger	0	25		
PI 269818	0	65		
PI 347492	45	0		
(PI 347492 × Bonneville)				
F_1	0	16		
F_2	18	58	1:3	0.79
BC (F_1 × Bonneville)	0	45		
BC (F_1 × PI 347492)	19	22	1:1	0.65
(PI 347492 × Ranger)				
F_1	0	16		
F_2	23	69	1:3	1.00
BC (F_1 × Ranger)	0	37		
BC (F_1 × PI 347492)	22	26	1:1	0.58
(PI 269818 × PI 347492)				
F_1	0	16		
F_2	25	95	1:3	0.29
BC (F_1 × PI 269818)	0	63		
BC (F_1 × PI 347492)	26	33	1:1	0.52

previously found in PI 347492 (India), a line resistant to the other pathotypes of the virus.[1]

Materials and Methods

Genetic populations used in this study were derived from crosses and backcrosses between the resistant plants of PI 347492 with those of the susceptible cultivars Bonneville, Ranger, and PI 269818. A culture of PSbMV-P4, available from a previous study, was maintained in Bonneville.[1] This cultivar is known to be resistant to several viruses, including PSbMV-L1 and bean yellow mosaic virus (BYMV).[6] Inoculum was prepared by grinding infected pea leaves with a phosphate buffer (K^+) at pH 8.5. Test plants, previously dusted with 400-mesh Carborundum, were mechanically inoculated by rubbing the first and second fully expanded leaves. A week later, the same plants received a second inoculation on the third leaves. This dual inoculation minimized the number of plants escaping infection, thus enhancing the reliability of data. Test plants were considered to be resistant if they failed to develop systemic infection. This condition was confirmed by employing the enzyme-linked immunosorbent assay (ELISA) or recovery tests using Ranger pea. Antisera to isolates of PSbMV had been prepared during a previous study.[1] All plants were maintained in an insect-free greenhouse at 25–30°C.

Results

Plants of PI 347492 remained free of systemic infection upon inoculation with PSbMV-P4. Conversely, plants of Bonneville, Ranger, and PI 269818 reacted with green foliar mottle, but they lacked the characteristic downward curling of leaflets usually associated with PSbMV infection.[1,4,6] Affected plants were partially stunted, and seed production was reduced. All plants of F_1 (PI 347492 × Bonneville), (PI 347492 × Ranger), and (PI 269818 × PI 347492) were susceptible to PSbMV-P4, showing symptoms similar to those noted on susceptible parents. Plants of F_2 populations segregated in a ratio of one resistant to three susceptible (Table 1), indicating that resistance was monogenically recessive. Confirmation of this mode of inheritance was obtained from reciprocal backcross populations. The progeny of F_1 plants crossed with the resistant parent segregated in the expected ratio of one resistant to one susceptible. Conversely, plants from the backcross to the susceptible parent were all susceptible. Thus, resistance to PSbMV-P4 in PI 347492 was determined to be monogenic recessive and the symbol *sbm-4* is proposed for this gene.

Discussion

Previously, the symbols *sbm-1*, *sbm-2*, and *sbm-3* had been given to the single recessive genes conferring resistance to other pathotypes of PSbMV.[3,6] Gritton and Hagedorn found *sbm-1* to be situated in *Pisum* linkage group 6.[2] Provvidenti and Alconero determined that *sbm-2* is closely linked to *mo* (the gene for resistance to BYMV), which is located in *Pisum* linkage group 2.[5,6] Provvidenti and Alconero also determined that *sbm-2* and *sbm-3* condition resistance to the same pathotype PSBMV-L1, but they are inherited independently of each other and might be duplicate entities.[6] No information is presently available regarding the linkage group of *sbm-3*. The frequent association of *sbm-4* with *sbm-1* and *sbm-3* in a number of pea lines from India and Ethiopia suggests linkage.[1]

In nature, the host range of PSbMV is very restricted. Infected seeds of peas and lentils are the main sources of local and long distance spread of this virus. The virus has been reported to be present in the seeds of a number of pea and lentil lines, mostly of foreign introduction, and undoubtedly its presence could be significantly reduced by better management of the seed stocks of public and private institutions.[1,3,4]

A long-term and more effective solution to the problem created by PSbMV depends upon the development of resistant cultivars. Four single recessive genes are presently available for a high level of resistance to PSbMV. Considering that these genes confer resistance to specific pathotypes of the virus, breeding for resistance will necessitate the incorporation into the same cultivar three of the four resistance genes, because two of these, *sbm-2* and *sbm-3*, have the same function. A previous study disclosed that in addition to PI 347492, a number of other lines from India and Ethiopia are resistant to the three known pathotypes of the virus, representing valuable germ plasm in breeding for multiresistance.[1]

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