Data in the matrix can be frequencies of various protein bands (0–1) if pattern variation exists within groups, or can be binary (0,1) to represent presence or absence of bands if groups are represented by individuals or invariant patterns. The program calculates three estimates of similarity: those of Gelfand, Ferguson, and Marczewski and Steinhaus.[2,3,5] It also converts similarity indexes into distances using the simple relationship $D = 1 - S$.

Gelfand devised separate formulae for continuous and dichotomous (binary) data.[3] For continuous data, as is the case when there is intragroup variation,

$$S = \frac{1}{1 + \sum_{t=1}^{k} |X_t - Y_t|^2},$$

where $X_t$ and $Y_t$ represent the frequencies of the $t$th band in groups $X$ and $Y$, respectively, and $k$ is the total number of protein bands observed. For dichotomous data,

$$S = 1 - \frac{1}{k} \sum_{t=1}^{k} |X_t - Y_t|.$$

For an example of the use of Gelfand's statistic, see Cline et al.[1]

Ferguson's estimate is intended for dichotomous data,[2]

$$S = \frac{w}{m},$$

where $w$ = number of bands of common mobility, and $m$ = maximum number of bands in an individual. The program automatically converts nondichotomous data to dichotomous when calculating this and the next statistic. For an example of the use of Ferguson's statistic, see Munuswamy.[6]

The Marczewski and Steinhaus statistic also uses dichotomous data,[5]

$$S = \frac{w}{a + b - w},$$

where $w$ = number of bands of common mobility, $a$ = number of bands in unit A, and $b$ = number of bands in unit B. For an example, see Sywula and Bartkowiak.[9]

A listing of the program (which is 235 lines long) will be provided by the senior author upon request. Alternatively, the program will be written (DOS 3.3 format) onto a 5.25-in. soft sectored floppy disk if one is provided.

From the Department of Biology, University of Alabama at Birmingham. Address reprint requests to Dr. Angus, Department of Biology, University of Alabama at Birmingham, Birmingham, AL 35294.

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# The Morphogenesis and Inheritance of an Open Carpel Mutant *cd*1 in Pigeonpea

**K. B. Saxena, A. K. Singh, and D. G. Faris**

An open carpel mutant, identified in the pigeonpea (*Cajanus cajan* (L.) Millsp.) cultivar ICPL 24 was found to have a genetically controlled developmental defect. Inheritance studies in the $F_1$, $F_2$, and $BC_1$ generations indicated that this abnormality was due to a single recessive gene mutation. The symbol *cd*1 is proposed for the gene.

Morphogenesis in flowering plants is under genetic control.[1] The present note reports the nature and inheritance of a defective mutant that affects the normal development of the carpel or ovary of pigeonpea (*Cajanus cajan* (L.) Millsp.).

## Materials and Methods

An open carpel mutant was identified in the pigeonpea cultivar ICPL 24, in 1982, and was studied morphologically and histologically. Normal-looking flower buds of this mutant produce flowers with an open carpel similar to that found in primitive plants such as gymnosperms. This defective mutation restricts the development of the placenta and ovule, thus resulting in an open pod without seeds. In 1983, this mutant was successfully crossed as the male parent with the cultivars ICPL 1 and C 322. The reciprocals did not set any pods. The $F_1$ and $F_2$ populations were observed for carpel development in both crosses, whereas the testcross progenies were studied only in the cross involving ICPL 1. This mutant could only be maintained by taking seeds from heterozygous plants or by vegetative propagation.

For anatomical studies, the buds were fixed in 3% glutaraldehyde in 0.1 M sodium phosphate buffer (pH 7.2) for 6 h, and then transferred to 2% osmium tetroxide for 3.5 h. Dehydration was carried out in acetone, and infiltration was done overnight in Spurr's low-viscosity epoxy resin. The samples were embedded by polymerizing the resin at 70°C for 48 h. Sections were cut using a glass knife, then stained in 0.5% toluidine blue, and examined under the light microscope.

## Results and Discussion

### Development of the carpel and ovules

A carpel is a highly modified ovule-bearing leaf. Normally, a carpel primordium arises as a horseshoe-shaped or circular mound that grows upward into a sac-like structure with fused margins. The corresponding stages of development in a normal plant and the mutant have been summarized in Figure 1. In legumes the ovule primordium appears as a small protuberance on the adaxial margin of the carpel. This primordium grows into an ovule on the marginal placenta of the ovary as illustrated in Figure 1 (a, b, c). In the mutant, initial development of the carpel primordium is normal, except that the margins of this

**Table 1. Segregation pattern for open carpel mutant in crosses involving the mutant and two pigeonpea cultivars**

| Cross | Generation | No. of plants | | Ratio tested | $\chi^2$ | Probability |
|---|---|---|---|---|---|---|
| | | Normal | Mutant | | | |
| ICPL 1 × mutant | $F_2$ | 845 | 265 | 3:1 | 0.73 | 0.3–0.5 |
| C 322 × mutant | $F_2$ | 440 | 138 | 3:1 | 0.38 | 0.5–0.7 |
| (ICPL 1 × mutant) × mutant | $BC_1$ | 33 | 30 | 1:1 | 0.13 | 0.7–0.9 |

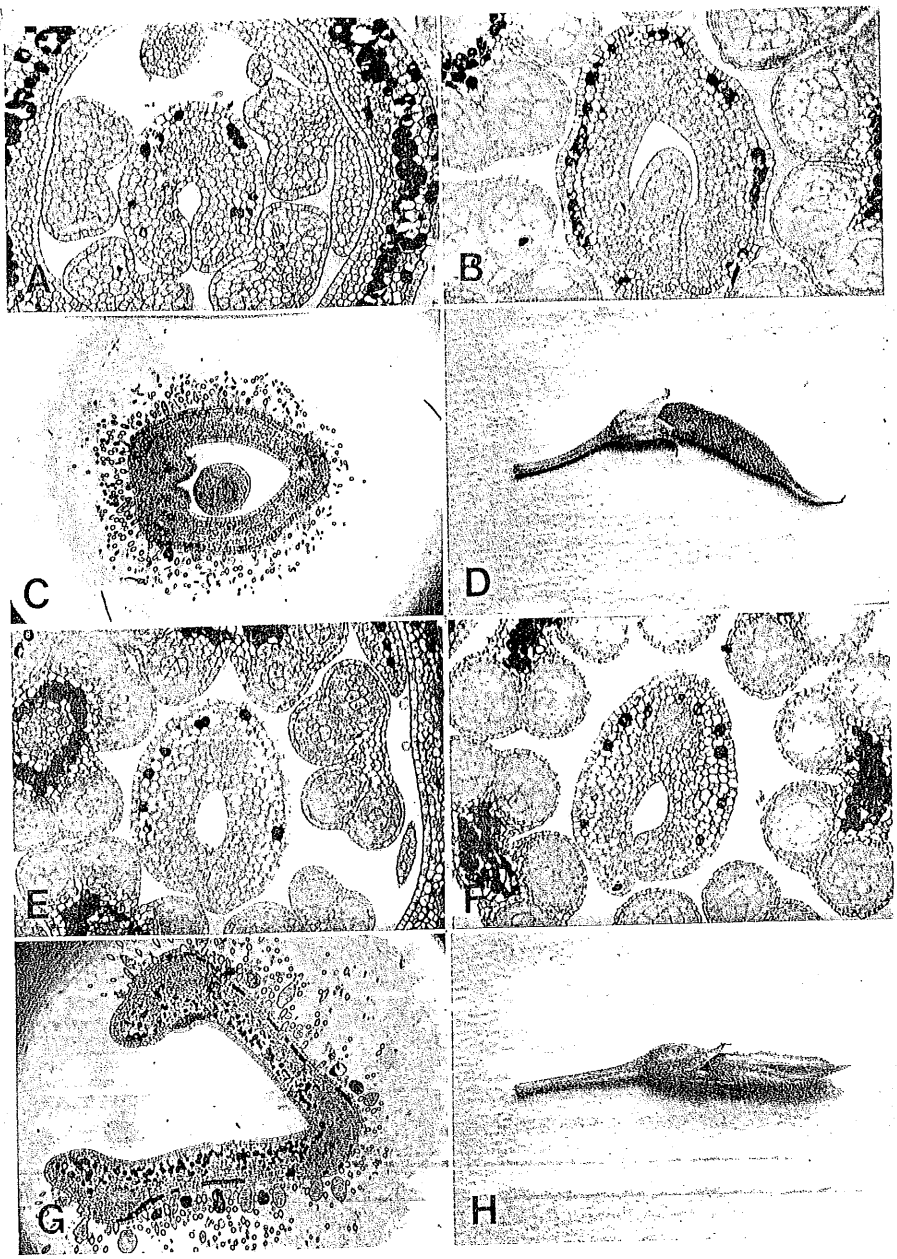


**Figure 1.** (a–d) The stages of development of the carpel in a normal pigeonpea flower. (e–h) The corresponding stages of development of the carpel in a flower of the defective mutant.

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.

From the Legumes Program at the International Crops Research Institute for the Semi-Arid Tropics, Patancheru, India. This research is ICRISAT Journal Article No. 612. Address reprint requests to Dr. Saxena, Asian Grain Legumes Program at the International Crops Research Institute for the Semi-Arid Tropics, Patancheru P.O., A.P., 502 324, India.

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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,