recorded on day 3. Wrigglesworth and Pover[5] also reported maximum damage in protein contents on the third day with 1000 R which agrees with the present study. Kwok and Chapman[6] reported that the protein synthesis decreased after irradiation which may be due to an impairment of carrier transport system of precursors in protein. Maisin[4] and Paul and Zimmerman[7] reported that radiation depressed the m-RNA synthesis thereby decreasing the protein anabolism. The observation of Mathur[8,9] indicated that depression in intestinal protein content is directly related to the DNA and RNA metabolism. After the maximum damage, recovery starts and normal value is reached earlier in lower dose groups and in the 1000 R group, normal value is not attained.

MPG-treated animals showed a similar pattern of changes, the maximum damage being observed at 24 hr but damage is less severe than in the control. It is clear from the fact that the protein contents in the experimental animals remained significantly higher than in the control. The recovery observed is faster as compared to control. The present findings agree with those of Romantsev and Blokhina[10] who reported that shortly after injection of aminothiols into animals, inhibition of radiosensitive biochemical precursors (DNA, RNA and Protein) was observed, which in turn, protected DNA, RNA and protein from radiation-induced lesions. Eldjarn *et al*[11] proposed that the cysteine-cysteamine group of radio-protectors acted by forming temporary mixed disulfides with -SH and -S-S- groups of proteins. Kollmann and Shapiro[12,13] and Kollmann *et al*[14] showed that GED protected the protein against radiation damage by the formation of mixed disulphide bond between GED and protein. It appears from the present findings that the drug MPG exerts its protective influence on the cells by the formation of mixed disulphide bond between the -SH compounds (protector) and protein. The formation of mixed disulphide bond, could result in restoration of the target irrespective of the radiation attack which may be *via* direct or indirect action and brings about an early recovery.

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FURTHER GENETIC ANALYSIS OF THE TRILOBATE LEAF MUTANTS IN MUNGBEAN (*VIGNA RADIATA* VAR *AUREUS* (L) WILCZEK)

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THE inheritance of trilobate leaf condition in mungbean cv K851 was reported earlier[1]. The mutant analysed in that study was induced through γ-irradiation (source ^{60}Co). This mutant plant (mutant-1) had all trilobate leaves in contrast to the standard plants which had only monolobate leaves. A spontaneous leaf mutant (mutant-2) with similar phenotype (figure 1) later appeared in the experimental population of the same cultivar. A true breeding stock of mutant-2 was prepared for further studies. In both the mutant stocks, penetrance was complete but expressivity was variable. Also, both the mutants showed anthocyanin pigmentation of stem, leaf petioles and leaf veins. The gene for anthocyanin pigmentation appeared to be tightly linked to the gene for trilobate leaf shape as the

Table 1 *Segregation of the trilobate leaf gene in mungbean.*

Progeny of	Segregation pattern (No of plants) Normal	Mutant	Total	Ratio	*P*
Mutant-2 selfed	0	129	129	0:1	—
F_1's					
Normal x Mutant-2	0	79	79	0:1	—
Mutant-2 x normal	0	91	91	0:1	—
F_2	47	182	229	1:3	0.10
F_1 × Normal (test cross)	89	107	196	1:1	0.20
F_1s					
Mutant-1 × mutant-2	0	92	92	0:1	—
Mutant-2 × mutant-1	0	77	77	0:1	—
F_2(F_1 plants selfed)	25	279	304	1:15	0.20
F_1 × normal	35	85	120	1:3	0.20

Figures 1–3. Typical leaves of the trilobate mutant-1, **1.** normal, **2.** and mutant-2, **3.** plants.

plants with trilobate leaves were always pigmented. The mutant-2 like the earlier mutant-1 gave a monogenic inheritance for the trilobate leaf shape gene (table 1). The present communication reports the results of inheritance studies conducted in both the mutants.

Reciprocal crosses were attempted between the plants from two mutant stocks. For this, the unopened flower buds were emasculated in the evening, immediately sprayed with 50 ppm aqueous solution of kinetin to minimise flower shedding and were pollinated the next morning. The results obtained from various crosses are presented in table 1. The F_1 plants were further allowed to self-pollinate. The F_2 progeny gave a 15 trilobate: 1 normal segregation indicating that the character is controlled by two genes Tlb_1 and Tlb_2 and the two mutant plants were having genotypes $Tlb_1\ Tlb_1\ tlb_2^+\ tlb_2^+$ and $tlb_1^+\ tlb_1^+\ Tlb_2\ Tlb_2$, so that the plants possessing either Tlb_1 or Tlb_2 or both had trilobate leaves. In the test cross, F_1 × normal, a 3 trilobate: 1 normal ratio, typical of a test cross in duplicate gene inheritance, was obtained. The quality of the fit for these ratios was tested by a X-square test and the *P* values are given in table 1.

From the results, it can be concluded that the leaf shape (trilobate vs normal) in mungbean is governed by at least two genes Tlb_1 and Tlb_2.

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INHERITANCE OF TEN INDUCED MUTANTS IN OKRA (*ABELMOSCHUS ESCULENTUS* (LINN) MOENCH.)

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ABELMOSCHUS ESCULENTUS (Linn) Moench commonly known as okra or bhindi is an important vegetable crop of the tropical and sub-tropical regions. There are only a few reports on spontaneous and induced mutations in this crop[1–5]. In studies aimed to explore the possibilities of increasing genetic variability in okra, 35 true breeding mutants were isolated