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Early maturing mutants of chickpea (*Cicer arietinum*) induced by chemical mutagens

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ABSTRACT

An experiment was conducted to evaluate the extent of induced genetic variability for two quantitative traits, viz. days to flowering and maturity by three chemical mutagens namely methylmethane sulphonate (MMS), hydrazine hydrate (HZ) and sodium azide (SA) in M_4 generation of chickpea (*Cicer arietinum* L.). The mean days to flowering and maturity were significantly reduced in all the treatments with the exception of 0.01% of SA. MMS treatments were found to be more effective in reducing the flowering and maturity period than HZ and SA. The genotypic coefficient of variation (GCV), heritability (h^2) and genetic advance increased manifold in the treated population indicating that these traits are controlled by additive genetic variance. Magnitude of GCV, h^2 and GA were recorded to be higher with 0.2% of MMS treatment followed by 0.03% of HZ and SA. Enhancement in range of genetic variability for days to flowering and maturity in M_4 generation is indicative of wider scope of selection.

Key words: Chemical mutagens, Chickpea, Early flowering and maturity, Genetic variability

Chickpea (*Cicer arietinum* L.) is an annual grain legume used for human consumption in India. It is a winter season crop which requires cool climate for its growth and high temperature for maturity. Chickpea has one of the highest nutritional compositions of any dry edible legume and does not contain any specific major anti-nutritional factors (ICRISAT 2005). Chickpea is primarily used for human consumption and only a small proportion is used for livestock feed. Since chickpeas are high in fibre and protein and have a low glycemic index, they help in controlling human body weight. Including approximately 175 ml of chickpeas in daily diet can help in lowering low-density lipoprotein cholesterol levels, which reduces the risk of fatal cardiac diseases.

Due to the narrow genetic base, conventional breeding methods did not contribute much to the improvement of chickpea (Toker *et al*. 2011, Wani *et al.* 2012). The application of mutation techniques has generated a vast amount of genetic variability and has played a significant role in plant breeding and genetic studies (Micke 1979, Khadke and Kothekar 2011, Wani 2017). The widespread use of induced mutants in plant breeding programmes throughout the world has led to the official release of more than 3100 mutant crop varieties (http://mvgs.iaea.org). A large number of these varieties (including cereals, pulses, oil, root and tuber crops, and ornamentals) have been released in developing countries, resulting in enormous positive economic impacts.

Genetic variability is the most essential pre-requisite for any successful crop improvement programme as it provides the spectrum of variants for effective selection. Since spontaneous mutations occur at very low frequency, artificial inductions of mutations facilitate the development of improved varieties at a faster rate (Badigannavar and Murty 2007, Verma *et al.* 2014, Wani and Kozgar 2016). Mutagenesis provide a handy tool to enhance the natural mutational rate by enlarging the existing genetic variability and increasing the scope for obtaining desirable early flowering and maturing lines which could lead to an ideal high yielding and early maturing chickpea crop.

MATERIALS AND METHODS

Uniform and healthy seeds of chickpea var. BG 256 were presoaked in distilled water for 9 hr, prior to treatment with chemical mutagens, viz. 0.1% to 0.4% of methylmethane sulphonate (MMS) and 0.01% to 0.04% of hydrazine hydrate (HZ) and sodium azide (SA) respectively for 6 hr. The healthy, non-dormant and untreated seeds soaked in distilled water for 15 hr were sown as control. The solutions of MMS and HZ were prepared in phosphate buffer of pH 7, whereas SA solution was prepared in phosphate buffer adjusted to pH 3. Chemically treated seeds were thoroughly washed in running tap water to eradicate the residual mutagens from the seed surface.

Four hundred seeds for every treatment and control were sown in the field in a complete randomized block design (CRBD) to raise M_1 generation. The distance between the

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seeds in a row and between the rows was kept as 30 cm and 60 cm respectively. Seeds harvested from individual M_1 plants were sown as M_2 families in three replicates in the field. For raising M_3 generation, such 10 M_2 progenies were selected which showed significant deviation in mean values in the negative direction from the mean values of control for days to flowering and maturity. Progenies of each M_3 selection were again grown as families to raise M_4 generation.

Data collected for days to flowering (number of days taken by the plant from date of sowing up to the date of opening of first flower bud) and days to maturity (number of days taken by the plant from the date of sowing up to the date of harvesting) of the mutants isolated in M_4 generation were subjected to statistical analysis as indicated below in order to assess the extent of induced variation. The significance of difference between the means of treated and control population was tested by using least significant difference (LSD) estimated from the error mean square and tabulated 't' value at 5% level of significance.

The mean ($\overline{X}$) was computed by taking the sum of a number of values (X_1, X_2 …….. X_n) and dividing by the total number of values (N) involved, thus;

$$\overline{X} = \frac{X_1 + X_2 + ...X_n}{N}$$

$$\overline{X} = \frac{\Sigma X_n}{N}$$

where, X_1, X_2, …….. X_n = Observations,

N = Total number of observations involved.

Standard error (SE)

$$S\,E = \frac{S.D.\ of\ sample}{\sqrt{N}}$$

where, SD = Standard deviation, N = Number of observations.

Analysis of variance was done according to Singh and Chaudhary (1985) to find out the variance between the families and within the families. The components of variance considered were: (i) Within–family variation in the control and in the treated material which was an estimate of environmental variation. (ii) between–families variation which was an estimate of the between families genetic variation.

The genotypic variance ($\sigma^2 g$) was estimated by the following formula:

$$\sigma^2 g = \frac{(MS_{Bf} - MS_e)}{N}$$

where, MS_{Bf} and MS_e = Mean sum of squares for between families and within families or error, respectively; N = Number of replications.

Genotypic coefficient of variation (GCV)

$$GCV\,(\%) = \frac{\sqrt{\sigma^2 g}}{\overline{X}} \times 100$$

Phenotypic variance ($\sigma^2 p$) was estimated by summing the estimated genotypic variance ($\sigma^2 g$) and the environmental variance (MS_e or $\sigma^2 e$)

$\sigma^2 p = \sigma^2 g + {}^2 e$

Phenotypic coefficient of variation (PCV)

$$PCV\,(\%) = \frac{\sqrt{\sigma^2 p}}{\times} \times 100$$

Heritability is the ratio of genotypic variance to the total phenotypic variance. The broad-sense heritability (h^2) was estimated by the formula suggested by Johnson *et al*. (1955).

$$h^2\,(\%) = \frac{\sigma^2 g}{\sigma^2 t} \times 100$$

where, $\sigma^2 g$ = Induced genotypic variance, $\sigma^2 t$ = Total phenotypic variance ($\sigma^2 t = \sigma^2 g + {}^2 e$) calculated from the treated population.

The estimates of genetic advance (GA) with 1% selection intensity were based on the formula given below:

$GA = k.\ \sigma p.\ h^2$

where, h^2 = Broad sense heritability, σp = Phenotypic standard deviation of the mean performance of treated population, K = 2.64, constant for 1% selection intensity.

$$GA\,(\%\ of\ \overline{X}) = \frac{GA}{\overline{X}} \times 100$$

In order to compare the means of various treatments, least significant difference (LSD) was applied and computed as follows:

Step-1. Construction of data table

Step-2. Correction Factor (CF)

$$CF = \frac{(Grand\ total)^2}{t.r.}$$

or

$$CF = \frac{(Wx)^2}{t.r.}$$

where, t = number of treatments, r = number of replicates, Wx = grand total.

Step-3. Total sum of squares (SSQT)

This is the sum of squares of all the values in the table, minus the correction factor

$SSQT = [(A_1)^2 + (B_1)^2 + \ldots. (E_1)^2] - CF$

or $SSQT = Wz - CF$

Step-4. Sum of squares of treatments (SSQt)

$$SSQt = \frac{(Y_1)^2 + (Y_2)^2 + ...(Y_5)^2}{r} - CF$$

$$SSQt = \frac{Wy}{r} - CF$$

where, r = number of replicates.

Step-5. Sum of squares of replicates (SSQr)

$$SSQr = \frac{(X_1)^2 + (X_2)^2 + (X_3)^2}{t} - CF$$

or

Step 1 The data were compiled such that each treatment occupies a column and their replicates were arranged in rows

Rows (Replicates)	Column (Treatments) number					Total of rows (Replicates) (Σ)	Squares of total of rows
	T_1	T_2	T_3	T_4	T_5		
R_1	A_1	B_1	C_1	D_1	E_1	A_1+..E_1=X_1	$(X_1)^2$
R_2	A_2	B_2	C_2	D_2	E_2	A_2+..E_2=X_2	$(X_2)^2$
R_3	A_3	B_3	C_3	D_3	E_3	A_3+..E_3 = X_3	$(X_3)^2$
Total of column (Σ)	A_1+..A_3 = Y_1	B_1+..B_3 = Y_2	C_1+..C_3 = Y_3	D_1+..D_3 = Y_4	E_1+..E_3 = Y_5	$(X_1)^2$+..$(X_3)^2$	= Wr
						(Grand Total) Y_1+..Y_5	= Wx
						X_1+..X_3	
Squares of total of columns $(\Sigma)^2$	$(Y_1)^2$	$(Y_2)^2$	$(Y_3)^2$	$(Y_4)^2$	$(Y_5)^2$	$(Y_1)^2$+..$(Y_5)^2$	= Wy
Sum of square of total of columns (Σ^2)	$(A_1)^2$+..$(A_3)^2$ = Z_1	$(B_1)^2$+..$(B_3)^2$ = Z_2	$(C_1)^2$+.. $(C_3)^2$ = Z_3	$(D_1)^2$+..$(D_3)^2$ = Z_4	$(E_1)^2$+.. $(E_3)^2$ = Z_5		
						Z_1+..Z_5	= Wz

$$SSQr = \frac{Wr}{t} - CF$$

where, t = number of treatments.

Step-6. Sum of squares of error (SSQ_e)

SSQ_e = SSQT – (SSQt – SSQr)

Step-7. Estimated variance of error (MS_e)

$$MS_e = \frac{SSQ_e}{(t-1)(r-1)}$$

Step-8. Least significant difference

$$\text{LSD at 5\%} = \frac{\sqrt{\frac{2MS_e}{r}}}{r} \times (t - \text{Value at 5\% level})$$

If the difference between any two treatment means exceeds the LSD values obtained at 5% level, the difference between the two means is taken to be significant.

RESULTS AND DISCUSSION

Mutation breeding offers scope for achieving in many instances what cannot be accomplished through backcrossing and selection. The advantage of mutation breeding is that it can be applied for changing the specific characters in otherwise good varieties by incorporating some useful variations in comparatively shorter period of time than the conventional breeding methods. So induced mutations supplement plant breeding and confer specific improvement on a variety without significantly altering its otherwise acceptable phenotype.

In the present study, data on genetic variability induced by MMS, HZ and SA for days to flowering and maturity studied in M_4 generation are presented in Tables 1-2. A glance at the tables indicates that ample variation was induced by mutagenic treatments for these two traits. Mean number of days to flowering was reduced significantly in all the mutagenic treatments as compared to control except 0.01% SA. The mean days to flowering was reduced by five days with the treatment of 0.2% MMS (control mean=78.40; treatment mean=73.39). Though the mutagenic concentrations were increased in linear order, yet the values of genetic parameters did not show any relationship with the mutagen dose. The highest phenotypic (11.79%) and genotypic (7.78%) variability, heritability (51.76%) and genetic advance (11.98%) were recorded with 0.2% of MMS treatment (Table 1).

Reduction in flowering time accompanied by increase in variability indicated that the variability has been induced in desired direction and would offer the possibility for selecting early flowering mutants in such treatment. The decrease in mean values for days to flowering is presumably due to the predominant incidence of micro-mutations for this trait. Kaul (1980) suggested that the mutation of two dominant genes to their recessive forms makes for an early flowering in peas. Gumber and Sarvjeet (1996) provided preliminary evidence that the two duplicate genes in homozygous recessive state cause early floweing. The early flowering mutants were also reported by Kharkwal (2000), Solanki (2005), Manjaya and Nandanwar (2007), Arulbalachandran and Mullainathan (2009) and Girja *et al.* (2013) in chickpea, lentil, soybean, urdbean and cowpea respectively.

Mean days to maturity was also reduced significantly in most of the mutagenic treatments. Maturity period was more reduced with 0.2% of MMS treatment (control mean=117.55; treatment mean=112.42) followed by 0.03% of HZ (control mean=117.55; treatment mean=113.10) and SA (control mean=117.55; treatment mean=113.54). The genetic parameters like phenotypic and genotypic coefficients of variation, heritability and genetic advance increased considerably in all the mutagen treatments. MMS treatment at 0.2% resulted in larger genetic variation for days to maturity (Table 2). Late or early maturity has substantial agronomic significance as these mutants suit for the specific

Table 1 Estimates of mean values ($\overline{X}$), shift in $\overline{X}$ and genetic parameters for days to flowering in M_4 generation of chickpea var. BG 256

Treatment	Mean ± S E	Shift in $\overline{X}$	PCV (%)	GCV (%)	h^2 (%)	GA (% of $\overline{X}$)
Control	78.40 ± 0.53		3.55	1.87	17.87	2.75
0.1% MMS	76.60 ± 0.75	- 1.80	8.25	5.54	49.99	10.63
0.2% MMS	73.39 ± 0.87	- 5.01	11.79	7.78	51.76	11.98
0.3% MMS	74.20 ± 0.91	- 4.20	9.53	7.53	48.23	10.23
0.4% MMS	75.50 ± 0.77	- 2.90	7.87	5.43	46.78	9.94
LSD (P=0.05)		0.88				
0.01% HZ	75.20 ± 0.73	-3.20	7.25	4.43	45.25	7.76
0.02% HZ	76.20 ± 0.92	- 2.20	9.76	5.25	43.73	8.83
0.03% HZ	74.50 ± 0.87	- 3.90	10.25	5.77	46.25	10.98
0.04% HZ LSD (P=0.05)	75.10 ± 0.82	- 3.30	9.76	6.76	41.54	10.07
0.01% SA	78.70 ± 0.78	1.02 + 0.30	7.52	4.65	41.23	7.53
0.02% SA	76.78 ± 0.83	- 1.62	9.42	5.27	44.32	8.21
0.03% SA	74.70 ± 0.89	- 3.70	10.07	5.72	45.51	10.82
0.04% SA	75.45 ± 0.74	- 2.95	9.21	6.19	42.23	9.91
LSD (P=0.05)		0.99				

LSD=Least significant difference; PCV=Phenotypic coefficient of variation; GCV=Genotypic coefficient of variation; h^2=Heritability; GA=Genetic advance.

Table 2 Estimates of mean values ($\overline{X}$), shift in $\overline{X}$ and genetic parameters for days to maturity in M_4 generation of chickpea var. BG 256

Treatment	Mean ± S.E.	Shift in $\overline{X}$	PCV (%)	GCV (%)	h^2 (%)	GA (% of $\overline{X}$)
Control	117.55 ± 0.67	-	3.53	1.76	20.27	2.22
0.1% MMS	114.24 ± 0.86	- 3.31	6.76	4.42	48.48	8.98
0.2% MMS	112.42 ± 0.90	- 5.13	9.89	6.66	52.87	11.55
0.3% MMS	114.34 ± 0.87	- 3.21	7.20	4.92	49.30	9.96
0.4% MMS	115.07 ± 0.79	- 2.48	7.09	4.02	47.52	9.39
LSD (P=0.05)		0.82				
0.01% HZ	114.27 ± 0.77	- 3.28	5.33	4.33	46.43	7.72
0.02% HZ	114.04 ± 0.86	- 3.51	7.23	5.05	45.44	9.95
0.03% HZ	113.10 ± 0.90	- 4.45	8.14	5.73	50.54	10.74
0.04% HZ	114.65 ± 0.84	- 2.90	7.01	4.46	44.98	8.47
LSA (P=0.05)		0.94				
0.01% SA	117.76 ± 0.75	+ 0.21	5.54	4.22	45.63	7.29
0.02% SA	114.54 ± 0.83	- 3.01	7.28	5.14	44.98	8.99
0.03% SA	113.54 ± 0.88	- 4.01	8.07	5.52	49.66	10.41
0.04% SA	114.25 ± 0.80	- 3.30	7.32	4.29	45.35	9.02
LSD (P=0.05)		0.75				

LSD=Least significant difference; PCV=Phenotypic coefficient of variation; GCV=Genotypic coefficient of variation; h^2=Heritability; GA=Genetic advance.

requirements of the breeding strategy (Zakri and Jalani 1998). Being cultivated as winter season crop in northern India, chickpea faces chilling and freezing temperatures during vegetative and reproductive growth. Cold stress during reproductive growth is detrimental to the flowering and pod setting of chickpea. Early maturity would thus be ideal to avoid such severe cold and attain maximum production in chickpea. The early maturity in mutants also makes them more suitable for intercropping practices and could possess greater degree of disease tolerance and show wide stability when grown in different agro-climatic conditions. Shamsuzzaman and Shaikh (1991) in chickpea, Yaqoob and Rashid (2001) and Wani and Kozgar (2016) in mungbean also reported a significant reduction in days to maturity after mutagenic treatments. Early maturity may be attributed to the physiological, biochemical, enzymological and hormonal changes induced by the mutagens (Bolbhat *et al.* 2012).

There had been discrepancies among the opinions of the researchers as to which generations are actually appropriate for the selection of quantitative traits. Some opined that selection for quantitative traits should be delayed till M_3 or later generations following mutagenic treatments (Gaul 1964, Kumar 1972, Jana and Roy 1973, Tar'an *et al.* 2004). However, others have proposed that effective selection for polygenic traits can be done in early generations even in M_2 (Sneep 1977, Solanki and Sharma 2002, Sheeba *et al*. 2003). The quantitative traits studied in this communication showed a wide range of phenotypic variation. The magnitude of phenotypic variation however does not reveal the relative amounts of heritable (genetic) and non-heritable (non-genetic) components of variation. This was ascertained with the help of some genetic parameters such as genotypic coefficient of variation, heritability and genetic advance in percent of mean. The estimates of genotypic coefficient of variation and heritability of various quantitative traits are essential since they indicate the degree of stability to environmental fluctuations and the potential transmissibility of a trait from parent to off spring.

The heritability estimates for the traits under study was higher in treated population. The high estimates of heritability has been found to be useful from breeder's point of view as this would enable him to base his selection on phenotypic performance. Ibrahim and Sharaan (1974) also reported that the increase in heritability is an indication of effective selection. Heritability in conjunction with genetic advance is more helpful in predicting the effect of selection than the heritability alone (Johnson *et al.* 1955). This is because the heritability estimates are subjected to certain estimation errors (Lin *et al.* 1979) and genotype–environment interactions. High heritability coupled with high genetic advance indicates the predominance of additive gene action, and as a result, there is a better scope for improving the traits like days to flowering and maturity through selection practices.

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