

## SOME FEATURES OF EGYPTIAN COTTON AFTER CHEMICAL MUTAGENS TREATMENT

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### Abstract

The main objective of the present work is to study the effect of Monoethanolamine (MEA) (0.04%) as a chemical mutagen on some economic traits. Data showed that (MEA) treatment increased significantly means of boll weight and seed index of the first parent, fiber strength of the second parent and both parents for lint index. Significant positive inbreeding depression was observed for boll weight, seed index, lint index and fiber strength. Epistatic effects were positive and significant in  $E_1$  and  $E_2$  for number of bolls per plant and seed cotton yield.

Genetic variances were due only to dominance variance after treatment for boll weight and fiber fineness. On the other hand additive variances were greater than dominance variances by using (MEA) treatment for each of seed cotton yield per plant, lint percentage, number of bolls per plant and fiber strength. While dominance variance was greater than additive variance for lint percentage.

Heritability values and the expected genetic advance upon selection increased for number of bolls per plant, lint percentage and fiber strength.

### Introduction

The study was initiated to investigate the effectiveness of chemical mutagen in cotton seed to induce variability in genetic material. The induction of mutation as a source of genetic variability may be used to help the plant breeder, to have wide variability, since the success of breeding program is dependent upon sufficient genetic variability among the genotypes to permit effective selection. The following research was to investigate the effectiveness of Monoethanolamine (MEA) in inducing genetic variability in some quantitative characters in generations derived from treated cotton seed of intraspecific cross compared with that derived from untreated seed.

Some investigators studied the effect of chemical mutagens on cotton. Ibragimov and Koval'chuk (1973) obtained many valuable mutants having high boll weight, long fiber and short growth period. El-Gohari (1975) treated two Egyptian varieties with (EMS). The means were slightly increased in plant height, boll weight, lint percentage, number of fruiting branches, micronaire reading and

pressely index. Egamberdiev and Daminov (1976) select forms with increased boll weight and fiber length after ethyleneimine treatment. Okaz (1978) reported that significant increase in quantitative variation was detected, by Ethylmethane sulphate, for boll weight and seed index. Heritability and response to selection were higher than control for seed index and less for seed cotton yield per plant. Tagiev (1979) treated seed cotton by 1, 4-bisdiazoacetylbutane and N-nitrose-N-dimethyl urea, he obtained early forms with compact habit, increased number of fruiting branches, increased number of bolls and high boll weight. Luckett (1989) treated seedlings with colchicine in lanolin four days after emergence. No morphological mutations were obtained in the  $M_2$  or  $M_3$ , but considerable heritable variation for quantitative characters was detected. Several families were significantly different from the parents for one or more characters. Kurtgel (1991) the mutations were produced by treating seeds with chemical mutagen NP 83 and 1, 4-bisdiazoacetylbutane differed from their source varieties in habit and leaf blade shape and exceeded them in most yield related quantitative traits. Tagiev (1991) treated seeds of cotton with N-dimethyl-N-nitrosourea and 1, 4-bisdiazoacetylbutane. He found increased for number of bolls, boll weight and long fiber. He added most of the mutations noted in the  $M_2$  were inherited in the  $M_3$ .

### Materials and Methods

The materials used in this study included two cotton varieties which belong to the species *G. barbadense* L. These two varieties were the Egyptian varieties Giza 70 is classified an extra long staple and Giza 81 is along staple. The field work continued for three successive seasons. In the first season, a single cross between the two varieties was made to produce enough  $F_1$  hybrid seeds. Seeds of parents as well as  $F_1$  seeds were sown in the second season.  $F_1$  plants were selfed to obtain  $F_2$  progeny. In the meantime,  $F_1$  plants were backcrossed to both parents. During the same growing season, both parents were crossed to obtain additional  $F_1$  hybrid seeds. The parents were maintained by artificial self-pollination. In the third season, the six genetic population seeds ( $P_1$ ,  $F_1$ ,  $F_2$ ,  $BC_1$ ,  $BC_2$  and  $P_2$ ) from the last season were divided into two groups, the first one was treated with Monoethanolamine (MEA) (0.04%) by soaking the seeds for 24 hours before planting and the second without treatment. The two groups were grown in randomized complete block design with four replication.

### Statistical Procedure

The following parameters were estimated for each of the six populations in each treatment:

- i. The means ( $\bar{x}$ ), the variance ( $S^2$ ) and the coefficient of variation (c.v.).
- ii. Heterosis, was expressed as the deviation of the  $F_1$  mean from the mid parent value.

- iii. Inbreeding depression, was calculated as the deviation of the  $F_2$  generation mean from the  $F_1$  mean.
- iv. Test of epistasis: the equations used were:
- $$E_1 = \bar{F}_2 - 1/2\bar{F}_1 - 1/4\bar{P}_1 - 1/4\bar{P}_2$$
- $$E_2 = \bar{BC}_1 + \bar{BC}_2 - \bar{F}_1 - 1/2\bar{P}_1 - 1/2\bar{P}_2$$
- v. Partitioning of the total phenotypic variance into its components:

The variances within each of the six populations were calculated. Mather (1949) used the six variances to estimate the environmental variance (E), genetic variance (G) and its components, additive (D) and dominance (H) variances. The three non segregating population  $P_1$ ,  $P_2$  and  $F_1$  were used to determine the environmental variance (E):

$$E = \sqrt[3]{VP_1 \cdot VP_2 \cdot VF_1}$$

The variance of the segregating populations  $F_2$ ,  $BC_1$  and  $BC_2$  were expressed as:

$$VF_2 = 1/2 D + 1/4 H + E$$

$$VBC_1 = 1/4 D + 1/4 H + E$$

$$VBC_2 = 1/4 D + 1/4 H + E$$

from the above equations the additive variance ( $1/2D$ ) and dominance variance ( $1/4 H$ ) were estimated.

- (vi). Heritability estimates ( $h^2$ ):

$$h^2 \text{ (broad sense)} = \frac{1/2 D + 1/4 H}{1/2 D + 1/4 H + E}$$

$$h^2 \text{ (narrow sense)} = \frac{1/2 D}{1/2 D + 1/4 H + E}$$

- (vii) Expected genetic advance upon selection ( $G_s$ ): for the highest 5 percent of the  $F_2$  plants is as follows:

$$G_s = (K) \times (\sigma_A) \times (h^2) \text{ (Allard, 1960).}$$

## **Results and Discussion**

### **Number of Bolls per Plant**

The t-test showed insignificant differences for all six populations under study except, showed significant decrease in the means of ( $P_2$  and  $F_1$ ).

Heterosis value was positive and significant in untreated control. However, it was insignificant after treatment. Inbreeding depression was insignificant in the untreated. While it was significant and negative after (MEA) treatment. In untreated it showed insignificant negative ( $E_1$ ) and ( $E_2$ ). After (MEA) treatment highly significant values were obtained for ( $E_1$ ) and ( $E_2$ ).

In untreated, the results obtained from partitioning of the genetic variance showed positive estimates for both additive genetic variance and dominance genetic variance. It is clear that the greater portion of the total genetic variance is due to

dominance genetic variance. In (MEA) additive variance (Table 4) (Okaz, 1978).

Heritability in broad and narrow senses were 64.71% and 28.46% in untreated, meanwhile it were 56.83% and 52.40% in (MEA) treatment. This high value for the heritability in narrow sense for treatment indicated that a considerable part of variation in the  $F_2$  was genetic, and that environment had little effect on this trait. Accordingly, selection in  $F_2$  population for number of bolls per plant will be fairly effective. At the same time the expected genetic advance upon selection was 18.35% and 31.44% in untreated and (MEA) treatment.

### **Boll Weight**

The means of all genetic materials for this trait were not sensitive to (MEA), except in the ( $P_1$ ), which showed significant difference with an increase in boll weight. The coefficient of variation was nearly equal for treatment and control (Table 1). The results obtained were in complete agreement with those obtained by Ibragimov and Kiovalchuck (1973), El-Gohari (1975), Egamberdiev and Daminov (1976), Okaz (1978) and Tagiev (1991).

In untreated a highly significant positive heterotic effect and inbreeding depression effect was revealed. Epistatic effects ( $E_1$ ) and ( $E_2$ ) were negative and highly significant and significant, respectively. (MEA) treatment, showed insignificant heterotic effect and highly significant inbreeding depression effect. Furthermore, a highly significant negative effect ( $E_1$ ) was obtained, whereas ( $E_2$ ) was insignificant.

The untreated showed that all genetic variance due to dominance effect of genes. The (MEA) treatment showed positive dominance genetic variance estimate of (0.05), whereas the additive genetic variance was a negative value which was usually regarded as estimate of zero. This meant that all genetic variances in this treatment were dominance variance (Table 4).

Estimates of broad sense heritability were 30.0 and 22.22 percent for control and (MEA) treatment, respectively. On the other hand heritability estimates in treatment was zero in narrow sense. Accordingly the expected genetic advance upon selection as estimated zero.

### **Seed Cotton Yield per Plant**

Treatment showed highly significant decrease in the mean of the second parent and only significant decrease in the  $F_1$  generation. The coefficient of variation was higher than in control.

In the untreated, this trait showed highly significant positive heterotic effect. Whereas insignificant heterotic effect was revealed after treatment. Estimates inbreeding depression were not significant in untreated and (MEA) treatment. Both epistasis ( $E_1$  and  $E_2$ ) were insignificant in untreated.

On the other hand, these values were positive and highly significant and significant, respectively after (MEA) treatment.

Estimates of additive and dominance genetic variances were positive in untreated. The same trends were observed after (MEA) treatment. Therefore, the greater portion of the total genetic variance is due to additive variance in untreated and (MEA) treatment.

In untreated estimates of broad and narrow senses, heritability were 63.12 and 52.08 percent, respectively. After (MEA) treatment, estimates of broad and narrow senses heritability were 55.22 and 41.19 percent, respectively. Accordingly, selection in  $F_2$  population for seed cotton yield per plant will be fairly effective. The expected genetic advance from selecting the desired five percent of individual plants are shown in Table (5), it was 34.41 in untreated and 24.93 in (MEA) treatment (Okaz, 1978).

#### **Lint Percentage**

From Table (1), the means of this trait showed non-significant differences for all genetic materials under study with nearly equal values after treatment compared with control. The coefficient of variability for each genetic materials was nearly similar.

From the values obtained for heterosis, inbreeding depression and epistasis, it appears that all values showed insignificant.

Estimates of dominance genetic variance was negative in untreated. Accordingly the genetic variance for untreated was due additive genetic variance. In (MEA) treatment, values of both additive and dominance genetic variances were positive. These values indicate also that the greater portion of the total genetic variance was the additive genetic variance in (MEA) treatment.

The heritability estimates (Table 5) for this trait in (MEA) treatment indicated that heritability was higher than that of untreated in both broad and narrow senses.

#### **Seed Index**

Results in Table (1) showed that the first parent (Giza 70) showed sensitivity for the treatment. Highly significant increase in the mean was found after MEA treatment (Luckett, 1989). The coefficient of variation was nearly similar for each genetic material.

The data indicated insignificant heterotic effects in untreated and (MEA) treatment. Inbreeding depression was insignificant in untreated, while it was positive and highly significant for (MEA) treatment. In untreated both epistasis values ( $E_1$  and  $E_2$ ) were insignificant, while after (MEA) treatment it was negative and highly significant for ( $E_1$ ) and insignificant for ( $E_2$ ), (Table 2).

The additive genetic variance was negative values and therefore estimated zero in untreated. All genetic variance was considered therefore to be due to dominance effect of genes in untreated. Estimates of genetic variance was zero in (MEA) treatment.

The heritability estimates in the (MEA) treatment was lower than that of untreated in broad sense. Meanwhile, heritability estimate in treatment under study was zero in narrow sense. Accordingly the expected genetic advance upon selection was estimated zero (Okaz, 1978).

#### **Lint Index**

Table (1) shows the means of this trait in different generation, which were nearly equal. From the same table, there were insignificant differences between means of untreated and treatment, except the two parents after treatment, which were significantly increased (Luckett, 1989). The coefficient of variation for each genetic material under study were nearly equal.

In untreated and (MEA) treatment, showed no heterotic effects. Inbreeding depression was significant and positive in untreated, however it was highly significant and positive after (MEA) treatment. The untreated showed insignificant values for ( $E_1$  and  $E_2$ ). After (MEA) treatment highly significant negative value was obtained for ( $E_1$ ) and negative and insignificant value for ( $E_2$ ) (Table 2).

In untreated, the results obtained from partitioning the genetic variance, showed that all genetic variance was due to additive variance. In (MEA) treatment, values of additive and dominance variance were positive. The values of additive and dominance variances were 0.03 and 0.05 respectively. Accordingly dominance genetic variance is the main component of the genetic variance in this treatment.

The heritability estimates of lint index are shown in Table 5. It was lower in (MEA) treatment than that of untreated in broad and narrow senses. The value of response to selection (Table 5) was lower in (MEA) treatment than that of untreated.

#### **Fiber Strength**

Monoethanolamine treatment showed significant increase in the mean of the second parent (Giza 81). Highly significant and significant decreases in the means were obtained in the first parent (Giza 70) and the first back cross, respectively. The coefficient of variation was nearly similar for all populations, except in  $F_2$  generation and the second back-cross, which showed high values after (MEA) treatment.

The untreated showed a highly significant negative heterotic effect, while inbreeding depression was insignificant. The (MEA) treatment showed highly significant negative heterosis, while inbreeding depression was significant and negative. From the values obtained from epistasis, it appears that all values showed insignificant epistasis results.

In untreated, the results showed negative estimate of additive genetic variance and therefore was estimated as zero, while dominance variance was positive. This means that all genetic variance in untreated is due to dominance variance. The (MEA) treatment showed positive estimates for both additive genetic variance and dominant genetic variance 0.74 and 0.03, respectively. It is clear that the greater portion of the total genetic variances due to additive genetic variance in this treatment.

The heritability estimates in (MEA) treatment was higher than that of untreated in broad and narrow senses. The value of response to selection were higher in treatment than that in the untreated, with low values.

### **Fiber Fineness**

The means of all populations for this trait were not sensitive to mutagen, giving no significant differences. The coefficient of variability for each group was nearly equal, except in F<sub>1</sub> generation, (Table 1).

From the values obtained for heterosis, inbreeding depression and epistasis, it appears that all values showed insignificant response (Table 2).

Data showed negative estimates for additive genetic variance in untreated and (MEA) treatment. Accordingly the genetic variance for untreated and treatment was due to dominance genetic variance.

The heritability estimates are shown in Table 5. In (MEA) treatment, it was lower than that of untreated in broad sense. Estimates of narrow sense heritability, were zfor untreated and (MEA) treatment. Accordingly, response to selection estimates was zero for untreated and the treatment (Table 5).

### **References**

Allard, R.W. (1960). Principles of plant Breeding. John Wiley & Sons, Inc. New York. London. 485 pp.

Egamberdev, A. and M. Daminov (1976). The mutagenic effectiveness of ethyleneimine n the gaseous state in the treatment of cotton seeds. in Genet: I. Seleksiya Khlopchatnika. Tashkent. Uzbek. SSR, Fan. pp. 65-73 (RU), From Referativnyi zhurnal 11. 55. 90. (c.f. Pl. Breed. Abst., 48: (2503), 1978).

El-Gohari, A.A. (1975). Induced variability of agronomic and fiber quality characters in two Egyptian cotton varieties by the chemical mutagen EMS. Ph.D. Thesis, Fac. of Agric., Cairo Univ., Egypt.

Ibragimov, Sh. I. and R. I. Koval'chuk (1973). Cotton Mutagensis. Institute Seleksii I Semenovodstva Dhlpchatnika. T Tashkent, Uzbek SSR, 1973 pp. 122-150.

Kurtgel, Dyev, B.K. (1991). Effect of the action of mutagenic factors on cotton. In Geneticheskie Posledstiviya Zagryazneniya Okruzhayushcher sredi mutagennymi faktorami. Vsesoyuznoe koordinatsionnoe soveshchanic, (Samarkand), 8-10 oktyabrya, 1990. Moscow, Russia 108-109. (RU) From Referativnyi Zhurnal (1991) 5 Ya 3367.

Lukett, D.J. (1989). Colchicine mutagenesis is associated with substantial heritable variation in cotton. Cotton Res. Unit., CSIRO Div. Pl. Indust., Po Box 59, Narrabri, NSW 2650, Australia. Euphytica. 1989, 42: 1-2, 177-182; 16 ref.

Mather, K. (1949). Biometrical Genetics. Methuen and Co., Ltd., London 162 pp.

Okaz, A.M. (1978). Genetical studies on cotton. Ph.D. Thesis, Fac. Agric., Al-Azhar University.

Tagiev, A.A. (1979). Effect of chemical mutagens on variability in cotton. Insttutkhlopkovodstva, Kiroabad, Azerbaijan SSR. Moscow 1979, pp. 138-139. From Res. Z. (1980), 2. 65. 140.

Tagiev, A.A. (1991). Variation in cotton induced by chemical mutagens. In khimicheski I mutagenez I problemy seleksii. Moscow Russian, (1991), 214-217 (RU) From Referativnyi Zhurnal (1991) 9 Ya 3135.

Table 1. Statistical values in the populations under study in control and treatment and the tests of differences between means of the studied characters.

Population	Control			MEA Treatment		
	$\bar{x}$	S <sup>2</sup>	C.V.%	$\bar{x}$	S <sup>2</sup>	
C.V.%						
<b>No. of bolls</b>						
P <sub>1</sub>	21.3	25.5	23.71	22.0	25.50	2295
P <sub>2</sub>	27.3	17.57	15.35	18.2**	22.62	2613
F <sub>1</sub>	29.4	37.12	20.72	21.4**	28.71	2540
F <sub>2</sub>	27.2	72.34	31.27	26.4	59.14	2913
BC <sub>1</sub>	22.0	60.20	35.27	22.6	42.85	2896
BC <sub>2</sub>	29.0	63.89	27.56	30.2	44.4	2207
<b>Boll weight:</b>						
P <sub>1</sub>	2.8	0.06	8.75	3.1*	0.09	967
P <sub>2</sub>	3.2	0.10	9.88	3.4	0.08	832
F <sub>1</sub>	3.3	0.06	7.42	3.3	0.06	742
F <sub>2</sub>	2.9	0.10	10.90	3.0	0.09	1000
BC <sub>1</sub>	3.0	0.11	11.06	3.2	0.08	884
BC <sub>2</sub>	3.0	0.09	10.00	3.1	0.13	1163
<b>Seed cotton yield per plant</b>						
P <sub>1</sub>	59.3	125.20	18.87	67.2	305.93	2603
P <sub>2</sub>	85.5	242.73	18.22	62.2**	239.13	2486
F <sub>1</sub>	94.6	446.81	22.34	70.4*	309.82	2500
F <sub>2</sub>	79.3	646.86	32.07	79.7	532.76	2896
BC <sub>1</sub>	63.5	553.96	29.63	61.6	445.38	2947
BC <sub>2</sub>	86.8	602.85	28.29	91.4	397.51	2181
<b>Lint percentage:</b>						
P <sub>1</sub>	35.9	2.95	7.78	35.0	2.83	481
P <sub>2</sub>	38.0	2.52	4.18	39.3	2.15	373
F <sub>1</sub>	37.4	1.51	3.29	37.0	1.80	362
F <sub>2</sub>	36.8	2.81	4.56	37.0	3.46	503
BC <sub>1</sub>	35.6	2.27	4.23	35.1	3.70	543
BC <sub>2</sub>	38.0	1.68	3.41	38.1	2.13	883
<b>Seed index</b>						
P <sub>1</sub>	8.6	0.32	6.58	9.5**	0.34	614
P <sub>2</sub>	9.5	0.65	8.49	10.0	0.90	949
F <sub>1</sub>	9.2	0.20	4.86	9.6	0.29	561
F <sub>2</sub>	8.9	0.40	7.11	8.9	0.35	665
BC <sub>1</sub>	9.1	0.62	8.65	9.5	0.43	614
BC <sub>2</sub>	9.6	0.63	8.27	9.6	0.22	489
<b>Lint index</b>						
P <sub>1</sub>	4.7	0.20	9.52	5.1*	0.16	784
P <sub>2</sub>	5.8	0.23	8.27	6.4*	0.22	733
F <sub>1</sub>	5.5	0.12	6.30	5.6	0.06	437
F <sub>2</sub>	5.2	0.28	10.18	5.2	0.26	981
BC <sub>1</sub>	5.0	0.21	9.17	5.2	0.30	1053
BC <sub>2</sub>	5.9	0.25	8.47	5.9	0.19	739
<b>Fiber strength</b>						
P <sub>1</sub>	11.6	0.07	2.28	10.5**	0.16	381
P <sub>2</sub>	10.8	0.31	5.16	11.4*	0.31	488
F <sub>1</sub>	10.7	0.11	3.10	10.5	0.09	286
F <sub>2</sub>	10.8	0.23	4.44	10.9	0.90	870
BC <sub>1</sub>	11.1	0.53	6.56	10.5*	0.31	530
BC <sub>2</sub>	10.8	0.21	4.24	11.0	0.75	787
<b>Fiber fineness</b>						
P <sub>1</sub>	4.2	0.06	5.83	4.0	0.05	559
P <sub>2</sub>	4.7	0.14	7.96	4.9	0.07	540
F <sub>1</sub>	4.3	0.03	4.03	4.3	0.06	570
F <sub>2</sub>	4.3	0.08	6.58	4.3	0.07	615
BC <sub>1</sub>	4.3	0.12	8.06	4.2	0.11	790
BC <sub>2</sub>	4.4	0.06	5.57	4.5	0.06	544

\*\* Significant at 0.05 and 0.01 levels, respectively.  
\*, \*\*MEA = Monoethanolamine.

Table 2. Heterosis, inbreeding depression and epistasis for all characters studied in untreated and treated population.

Treatment	Generation means			Heterosis %	Inbreeding depression %	Epistasis (E <sub>1</sub> )	Epistasis (E <sub>2</sub> )
	M.P.	F <sub>1</sub>	F <sub>2</sub>				
<b>No. of bolls per plant</b>							
Control	24.30	29.4	27.2	20.99*	7.48	0.35	-270
MEA treat.	20.10	21.4	26.4	6.47	-23.36*	5.65	1130
<b>Boll weight</b>							
Control	3.00	3.3	2.9	10.00**	12.12**	1.90**	-0.33*
MEA treat.	3.25	3.3	3.0	1.54	9.09**	-0.275**	-0.25
<b>Seed cotton yield per plant</b>							
Control	72.40	34.6	79.3	30.66**	16.17	-4.200	-1670
MEA treat.	64.70	70.4	79.7	8.81	-13.21	12.15**	1790
<b>Lint percentage</b>							
Control	37.00	37.4	36.8	1.08	1.60	-0.37	-075
MEA treat.	37.20	37.0	37.0	-0.54	0.00	-0.075	-055
<b>Seed index</b>							
Control	9.05	9.2	8.9	1.66	3.26	-0.22	045
MEA treat.	9.75	9.6	8.9	-1.54	7.29**	-0.775**	-025
<b>Lint index</b>							
Control	5.25	5.5	5.2	4.76	5.45*	-0.17	015
MEA treat.	5.75	5.6	5.2	-2.61	7.14**	-0.475**	-025
<b>Fiber strength</b>							
Control	11.20	10.7	10.8	-4.46**	-0.93	-0.15	000
MEA treat.	10.95	10.5	10.9	-4.11**	-3.81*	0.175	005
<b>Fiber fineness</b>							
Control	4.45	4.3	4.3	-3.47	0.00	-0.07	-005
MEA treat.	4.45	4.3	4.3	-3.37	0.00	-0.075	-005

\*, \*\* Significant at 0.05 and 0.01 levels, respectively.  
M.P. = Mid parent.

Table 3. Tests of significance of the genetic variance among F<sub>2</sub> populations.

Treatment	VF <sub>2</sub>	VE	F-test
<b>No. of bolls per plant</b>			
Control	72.34	25.53	**
MEA treat.	59.14	25.53	**
<b>Boll weight</b>			
Control	0.10	0.07	**
MEA treat.	0.09	0.07	**
<b>Seed cotton yield per plant</b>			
Control	646.86	238.57	**
MEA treat.	532.76	238.57	**
<b>Lint percentage</b>			
Control	2.81	2.24	**
MEA treat.	3.46	2.24	**
<b>Seed index</b>			
Control	0.40	0.35	**
MEA treat.	0.35	0.35	*
<b>Lint index</b>			
Control	0.28	0.18	**
MEA treat.	0.26	0.18	**
<b>Fiber strength</b>			
Control	0.23	0.13	**
MEA treat.	0.90	0.13	**
<b>Fiber fineness</b>			
Control	0.08	0.06	**
MEA treat.	0.07	0.06	**

\*, \*\* Significant at 0.05 and 0.01 levels, respectively.

Table 4. Partitioning of phenotypic variance into its components.

Treatment	variance				
	Phenotypic	Genotypic	Additive	Dominance	Environmental
<b>No. of bolls per plant</b>					
Control	72.34	46.81	20.59	26.22	25.53
MEA treat.	59.14	33.61	30.99	2.62	25.53
<b>Boll weight</b>					
Control	0.10	0.03	0.00	0.03	0.07
MEA treat.	0.09	0.02	-0.03	0.05	0.07
<b>Seed cotton yield per plant</b>					
Control	646.86	408.29	336.91	71.38	238.57
MEA treat.	532.76	294.19	222.63	71.56	238.57
<b>Lint percentage</b>					
Control	2.81	0.57	1.67	-1.10	2.24
MEA treat.	3.46	1.22	1.09	0.13	2.24
<b>Seed index</b>					
Control	0.40	0.05	-0.45	0.50	0.35
MEA treat.	0.35	0.00	0.00	0.00	0.35
<b>Lint index</b>					
Control	0.28	0.10	0.10	0.00	0.18
MEA treat.	0.26	0.08	0.03	0.05	0.18
<b>Fiber strength</b>					
Control	0.23	0.10	-0.28	0.35	0.13
MEA treat.	0.90	0.77	0.74	0.03	0.13
<b>Fiber fineness</b>					
Control	0.08	0.02	-0.02	0.04	0.06
MEA treat.	0.07	0.01	-0.03	0.04	0.06

MEA = Monoethanolamine.

Table 5. Heritability in broad and narrow senses and genetic advance upon selection.

Treatment	heritability		Genetic advance	
	Broad	Narrow	GS	GS%
<b>No. of bolls per plant</b>				
Control	64.71	28.46	4.99	18.35
MEA treat.	56.83	52.40	8.30	31.44
<b>Boll weight</b>				
Control	30.00	0.00	-	-
MEA treat.	22.22	0.00	-	-
<b>Seed cotton yield per plant</b>				
Control	63.12	52.08	27.29	34.41
MEA treat.	55.22	41.79	19.87	24.93
<b>Lint percentage</b>				
Control	20.28	20.28	0.70	1.90
MEA treat.	35.26	31.50	1.21	3.27
<b>Seed index</b>				
Control	12.50	0.00	-	-
MEA treat.	0.00	0.00	-	-
<b>Lint index</b>				
Control	35.71	35.71	0.39	7.50
MEA treat.	30.77	11.54	0.12	2.31
<b>Fiber strength</b>				
Control	43.48	0.00	-	-
MEA treat.	85.56	82.22	1.61	14.77
<b>Fiber fineness</b>				
Control	25.00	0.00	-	-
MEA treat.	14.29	0.00	-	-

MEA = Monoethanolamine.