# Effect of Chemical Mutagens on Fiber Fineness of Egyptian Cotton

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## ABSTRACT

*This research aimed at studying the effect of triethanolamine (TEA) (0.02%) and dimethylsulphate (DMS) (0.025%) on fiber fineness, in the two Egyptian cotton varieties: Menoufi and Giza 74, and in the $F_1$, $F_2$, $Bc_1$, and $Bc_2$ of their hybrid. The study was carried out on the $M_1$ and $M_2$ of the two chemical mutagens. The fieldwork continued for three successive seasons. Both TEA and DMS treatments increased the means of fiber fineness in most of the six populations for $M_1$ and $M_2$ generations. Highly significant values were obtained in $F_2$ in $M_1$ and $M_2$ for the TEA treatment, while a highly significant value showed in the $F_2$ of $M_1$ only for the DMS treatment. Significant and highly significant inbreeding depression and epistasis, except $E_2$ in $M_1$,were found for the TEA treatment. Conversely, insignificant heterosis, inbreeding depression and epistasis were obtained for the DMS treatment and the control. The genetic variance was only due to the additive effect in the control. In the TEA treatment, all the genetic variance was due to dominance effects for both mutant generations, while in the DMS treatment all the genetic variance was additive variance in $M_1$, and additive variance was larger than dominance variance in $M_2$ for the same treatment. Broad and narrow sense heritability values and genetic advance on selection decreased after both treatments except broad sense heritability of $M_2$ in the DMS treatments*.

## Introduction

Mutagenic agents are accepted to be effective in inducing genetic changes in a treated population. This study was on the effect of two chemical mutagens, triethanolamine (TEA) and dimethylsulphate (DMS), on the means, coefficient of variation, heterosis, inbreeding depression, epistasis, genetic variance, heritability and expected genetic advance from selection for fiber fineness and fiber maturity, expressed as micronaire reading, in two Egyptian cotton varieties, Menoufi and Giza 74, as well as the $F_1$, $F_2$, $Bc_1$, and $Bc_2$ of their hybrid. The study was on all genetic materials in the first and second mutant generations and control.

Many investigators have studied the effect of mutagens on economic characters. Mukymov *et al.* (1971) studied the effect of some mutagens on two cotton varieties and found that the effect varied from one mutagen to another and from one concentration to another for each variety for the same mutagen. They found also that one variety showed lower sensitivity for the mutagens, and showed results equal to or less than the control for some characters. The second variety responded to mutagens increment and some traits were higher than the control. Abd-el-Hamid (1972) found in the $M_1$, that the variance and coefficients of variation were markedly increased in the population after treatment with gamma rays. The exposures produced slightly but significantly decreased fiber strength in the $M_1$ without affecting fiber fineness. Fursov *et al.* (1972) obtained some mutant lines in *G. hirsutum* and *G. barbadense* after treatment with some mutagens. These lines possessed good values for staple length, strength and fineness, and were used in breeding programs. Fotiadis (1973) studied the effect of gamma irradiation on the means, variances and covariances of several quantitative traits. Means for fiber fineness was significantly increased by the treatment as was genetic variance. Askerbeili (1976) found some mutants in cotton after treatment with ethyleneimine. These mutants had large bolls, good-quality fiber and compact plants. Daminov (1976) treated seeds of *G. barbadense* and *G. hirutum* with dimethylsulphate, and was able to select early, wilt-resistant, large bolled mutants with improved fiber quality. Okaz (1978) studied the effect of ethylmethanesulphate on two varieties of Egyptian cotton as well as the $F_1$, $Bc_1$, $Bc_2$ and $F_2$ of their hybrid. The treatment increased additive variance and both narrow sense heritability and response to selection of the $F_2$ population. El-Gohary *et al.*. (1981) studied the effect of ethylmethanosulphate on the Egyptian cotton variety Giza 45. They noticed that the mutant was similar to Giza 45 with respect to halo length and fiber length and fineness. Tagiev (1991) treated seeds of the *G. hirsutum* varieties with N-dimethyl-N-nitrosourea and 1,4-bisidazoacetylbutane. He found that most of the mutations noted in the $M_2$ were inherited in the $M_3$. They included improved fiber quality. He also reported that the mutation frequency rose with an increase in mutagen concentration.

## Material and Methods

The material used were the parents, $F_1$, backcrosses to both parents, and $F_2$ population of an intraspecific cross between two Egyptian cotton varieties, Menoufi and Giza 74. Triethanolamine (0.02% concentration) and dimethylsulphate (0.025% concentration) were used as chemical mutagens.

The fieldwork continued for three successive seasons. In the first season, a single cross between the two parents was done to produce enough hybrid seeds. In the second season, the hybrid seeds and the two parents were divided into three parts; the first part was planted in order to obtain the $F_2$ generation through self fertilization. The second and third parts of hybrid seeds and the two parents were treated with the chemical mutagens by soaking the seeds for 24 hours before planting in triethanolamine solution with concentration 0.02% and dimethylsulphate solution with concentration 0.025%. In the same season, the two backcrosses between the untreated $F_1$ hybrid and the two parents were done, as well as the two backcrosses between the treated $F_1$ hybrid and the treated parents. The treated parents were also crossed to obtain the $F_1$ generation in the third season. The untreated parents were also crossed to obtain additional hybrid seeds. The parental cultivars were selfed to produce sufficient seeds of each parent to maintain their purity. In the third season, the genetic materials obtained from the second season were sown in randomly distributed rows.

The t-test was used to test the significance of the shifts of the treated population means from their corresponding untreated population means.

Heterosis was expressed as the deviation of the $F_1$ mean from the mid-parent value using the following equation: Heterosis deviation = $F_1 - 1/2 (P_1 + P_2)$.

Inbreeding depression was calculated as the deviation of the $F_2$ generation mean from the $F_1$ mean as follows: Inbreeding depression deviation = $F_1 - F_2$.

The first epistatic deviation of the $F_2$ generations ($E_1$) and the second epistatic deviation of the backcrosses ($E_2$) were calculated as follows:

$E_1 = F_2 - 1/2\ F_1 - 1/4\ P_1 - 1/4\ P_2$

$E_2 = B_1 + B_2 - F_1 - 1/2\ P_1 - 1/2\ P_2$

Mather's procedure (1949) was used to estimate environmental variance (E), genetic variance (G) and its components: additive (D) and dominance (H) variance. Heritability in broad and narrow senses was also measured. The expected genetic advance from selection was estimated according to Allard (1960).

## Results and Discussion

### *Means and coefficients of variation*

Table 1 shows that the mean of fiber fineness (micronaire reading) of the first parent Menoufi was 3.85 in the control with a range of 3.3-4.7. The average means of the two treatments in $M_1$ and $M_2$ were 3.66 and 3.51 for triethanolamine (A) and 3.87 and 3.89 for dimethylsulphate (B) with ranges of (2.4-4.3 and 2.7-4.3), and (3.2-4.3 and 3.4-4.3), respectively. The t-test revealed that there were no differences between both mutant generations for the two mutagens and control. The triethanolamine second mutant generation showed a highly significant decrease in the mean compared with the control. These results are in agreement with those reported by (Abd-El-Hamid, 1972; El-Gohari *et al.*, 1981; Fursov *et al.*, 1972 and Tagiev, 1991).

These results showed that the effect varied from one mutagen to the other. The two mutant generations showed higher C.V. percent in the TEA treatment than in the control. The first backcross means ($Bc_1$) were nearly equal except the second mutant generation for (B) showed significant differences between means of the treatment and the control. In $F_1$, the treatments (A and B) for the two mutant generations showed insignificant differences. The $F_2$ generation showed highly significant differences between the control and the two mutant generations for triethanolamine, However, for DMA only $M_1$ was highly significant. In the second backcross to $P_2$ highly significant decreased values were obtained between the control and the two treatments, except in $M_2A$ which had insignificant differences. The coefficient of variation showed higher values in all treatments than the control. The second parent ($P_2$) Giza 74 showed insignificant differences in the two treatments. The first mutant generation showed sensitivity for the two mutagens but with insignificant differences.

Table 2 shows that the values of the segregating generations in $M_2$ were higher than $M_1$ in the TEA treatment except in the $F_1$. In the DMS treatment the values of the segregating generations in $M_2$ were lower than the values in $M_1$ except the first backcross. On the other hand, Table 3 showed a highly significant increase over the control in $M_2$ for the second generation in the DMS treatment.

### *Heterosis, inbreeding depression and epistasis*

Table 4 gives the tests of significance of these estimates. In the control, both heterotic and inbreeding depression effects were insignificant. In addition, $E_1$ and $E_2$ were not significant. The first $M_1A$ and second $M_2A$ mutant generations for the TEA treatment showed insignificant heterosis, significant and highly significant negative inbreeding depression (-3.23) and (-10.58), highly significant positive $E_1$ (0.1700) and (0.3525) for $M_1A$ and $M_2A$, and highly significant positive $E_2$ (0.3750) for $M_2A$. The DMS treatment showed insignificant heterotic effects, inbreeding depression effects and $E_1$ and $E_2$ values in first and second mutant generations ($M_1B$ and $M_2B$).

### *Partitioning of the total phenotypic variance into its components*

Estimates of additive genetic variance was positive in the control (0.1801), whereas dominance variance was negative (Table 5). Genetic variance was mainly considered to be due to the additive effect of genes in the control. The phenotypic variance gave negative additive genetic variance estimates of -0.1072 and -0.1523 for $M_1$and $M_2$ in the TEA treatment. Negative estimates of variance are usually regarded as estimates

of zero. This means that all genetic variance exhibited in connection with fiber fineness in $M_1$ and $M_2$ for the TEA treatment was dominance variance. On the other hand, the dominance genetic variance was negative (-0.0057) for the first mutant generation in (DMS) treatment. It is clear that all total genetic variance is due to additive genetic variance. In the second mutant generation of this treatment it is clear that the greater portion of the total genetic variance is due to additive genetic variance (Fotiadis and Miller, 1973 and Okaz, 1978).

***Heritability estimates***

Estimates of heritability in the control was 39.67 for broad and narrow senses (Table 6). In the TEA treatment these were -1.98 and 0 for $M_1$ and -43.94 and 0 for $M_2$. Heritability estimates were 2.03 percent in the broad and narrow senses for the $M_1$ (DMS) treatment. At the same time $M_2$ estimates were 53.70 and 37.06 percent, respectively, as shown in Table 6.

All genetic variance was considered to be due to the additive effect of genes in the control and $M_1$ for the DMS treatment since the dominance variance was negative and was therefore considered to be zero. On the other hand the values obtained for control were intermediate, whereas the mutant generations in the two treatments, except $M_2$ for DMS, are low. These results reflect the fact that environmental variance for this character is high.

***Expected genetic advance upon selection***

In Table 7, the expected genetic advance from selecting the top 5% of the population in $F_2$ for the control, ($G_s$) was 0.4111 or (11.26%). No advance is expected from selecting the top 5% of the $F_2$ population in the TEA treatment. This is because the estimates of heritability were zero in the TEA treatment. In the DMS treatment it was 0.0176 in $M_1$ (0.4531%) and 0.3471 in $M_2$ (9.38%). The low response to selection in these mutant generations shows that selection for this character is not fruitful.

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**Table 1. Tests of significance of differences between means of the studied characters.**

| Generation | Treatment | m | X | $S^2$ | S | Sx | C.V.% | Range |
|---|---|---|---|---|---|---|---|---|
| $P_1$ | Control | 32 | 3.85 | 0.0832 | 0.2885 | 0.0510 | 7.49 | 3.3-4.7 |
| | $M_1A$ | 32 | 3.66 | 0.2346 | 0.4843 | 0.0856 | 13.23 | 2.4-4.3 |
| | $M_2A$ | 32 | 3.51** | 0.2020 | 0.4494 | 0.0794 | 12.80 | 2.7-4.3 |
| | $M_1B$ | 18 | 3.87 | 0.0715 | 0.2675 | 0.0631 | 6.91 | 3.2-4.3 |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | $M_2B$ | 16 | 3.89 | 0.0886 | 0.2977 | 0.0744 | 7.65 | 3.4-4.3 |
| $Bc_1$ | Control | 49 | 3.71 | 0.1762 | 0.4198 | 0.0600 | 11.31 | 2.7-4.5 |
| | $M_1A$ | 62 | 3.79 | 0.2535 | 0.5035 | 0.0640 | 13.28 | 2.9-4.7 |
| | $M_2A$ | 60 | 3.82 | 0.1409 | 0.3753 | 0.0484 | 9.83 | 2.7-4.7 |
| | $M_1B$ | 42 | 3.72 | 0.1765 | 0.4202 | 0.0648 | 11.29 | 2.9-4.3 |
| | $M_2B$ | 60 | 3.86* | 0.1248 | 0.3533 | 0.0456 | 9.15 | 3.3-4.4 |
| $F_1$ | Control | 56 | 3.67 | 0.2709 | 0.5205 | 0.0696 | 14.18 | 2.4-4.9 |
| | $M_1A$ | 77 | 3.71 | 0.1415 | 0.3762 | 0.0429 | 10.14 | 2.9-4.9 |
| | $M_2A$ | 64 | 3.59 | 0.2528 | 0.5028 | 0.0628 | 14.01 | 2.6-4.5 |
| | $M_1B$ | 57 | 3.84 | 0.2028 | 0.4503 | 0.0596 | 11.73 | 3.0-4.8 |
| | $M_2B$ | 53 | 3.77 | 0.2194 | 0.4684 | 0.0643 | 12.42 | 2.7-4.5 |
| $F_2$ | Control | 148 | 3.65 | 0.2531 | 0.5031 | 0.0414 | 13.78 | 2.7-4.7 |
| | $M_1A$ | 123 | 3.83** | 0.1821 | 0.4267 | 0.0385 | 11.14 | 2.9-5.3 |
| | $M_2A$ | 103 | 3.97** | 0.1452 | 0.3811 | 0.0375 | 9.60 | 2.5-4.9 |
| | $M_1B$ | 134 | 3.89** | 0.1777 | 0.4251 | 0.0365 | 10.84 | 2.7-4.8 |
| | $M_2B$ | 78 | 3.70 | 0.2067 | 0.4546 | 0.0515 | 12.29 | 2.9-4.9 |
| $Bc_2$ | Control | 66 | 3.93 | 0.1499 | 0.3872 | 0.0477 | 9.85 | 3.2-5.2 |
| | $M_1A$ | 58 | 3.74* | 0.2179 | 0.4668 | 0.0613 | 12.48 | 2.6-4.5 |
| | $M_2A$ | 60 | 3.79 | 0.3018 | 0.5494 | 0.0709 | 14.50 | 2.6-4.7 |
| | $M_1B$ | 45 | 3.72** | 0.1696 | 0.4118 | 0.0614 | 11.07 | 3.0-4.7 |
| | $M_2B$ | 25 | 3.63** | 0.2120 | 0.4604 | 0.0921 | 12.68 | 2.6-4.3 |
| $P_2$ | Control | 33 | 3.66 | 0.1581 | 0.3977 | 0.0693 | 10.86 | 2.9-4.3 |
| | $M_1A$ | 33 | 3.56 | 0.1925 | 0.4388 | 0.0764 | 12.32 | 3.1-4.5 |
| | $M_2A$ | 34 | 3.78 | 0.1788 | 0.4229 | 0.0725 | 11.19 | 3.1-4.6 |
| | $M_1B$ | 24 | 3.71 | 0.3643 | 0.6036 | 0.1232 | 16.27 | 2.7-4.7 |
| | $M_2B$ | 19 | 3.72 | 0.0451 | 0.2123 | 0.0487 | 5.71 | 3.3-4.1 |

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

$M_1$= First mutant generation A = Triethanolamine treatment.

$M_2$ = Second mutant generation. B = Dimethylsulphate treatment.

**Table 2. Tests of significance of differences between means of first and second mutant generation of TEA and DMS treatments.**

| | TEA | | DMS | |
|---|---|---|---|---|
| Generation | $M_1$ | $M_2$ | $M_1$ | $M_2$ |
| $P_1$ | 3.66 | 3.51 | 3.87 | 3.89 |
| $BC_1$ | 3.79 | 3.82 | 3.72 | 3.86 |
| $F_1$ | 3.71 | 3.59 | 3.84 | 3.77 |
| $F_2$ | 3.83 | 3.97** | 3.89 | 3.70** |
| $Bc_2$ | 3.74 | 3.79 | 3.72 | 3.63 |
| $P_2$ | 3.56 | 3.78 | 3.71 | 3.72 |

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

**Table 3. Tests of significance of the genetic variance among $F_2$ generations in control and both treatments.**

| | TEA | | | DMS | | |
|---|---|---|---|---|---|---|
| Generation | VF2 | VE | F-test | VF2 | VE | F-test |
| Control | 0.2531 | 0.1527 | 1.6575** | 0.2531 | 0.1527 | 1.6575** |
| $M_1$ | 0.1821 | 0.1857 | 0.9806 | 0.1777 | 0.1741 | 1.0207 |
| $M_2$ | 0.1452 | 0.2090 | 0.6947 | 0.2067 | 0.0957 | 2.1599** |