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# SOME ANTIOXIDANTS INHIBIT EMS-MUTAGENESIS IN *DROSOPHILA* GERM CELLS

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The effects of two antioxidants (AO) 2,6-dimethyl-3,5-diethoxycarbonil-4 (Na carboxylate)-1,4-dihydropyridine (DHP) and glutation (GP) were compared under different conditions:

(1) the treatment of adult males or
(2) their larvae;
(3) the treatment of females.

Then adult males were exposed to ethyl methanesulfonate (EMS). Germ cell mutability was estimated by sex linked recessive lethals (SLRLs) that are due to intra-locus alterations. Besides, the embryonic (EL) and postembryonic (PEL) lethals caused by chromosome breaks were scored

When adult males being fed by AOs, these compounds didn't influence EMS mutagenic and clastogenic effects in both test-systems. Larval pre-treatment with AO reduced the chromosome break level as well as the frequency of SLRLs. For example, DHP (90 mM) decreased the EMS induced embryonic lethality from 20.4 to 16.8% under 7-8 day-storage of spermatozoa and this AO reduced the EMS-induced SLRL frequency by more than 50% in sperm cells without storage. The protective effect of GP (10 mM) was found in both test systems: this AO reduced EMS mutagenicity by ~20% in SLRL test (z=2.29* by Cochran) and by~30% in chromosome break test after storage (z=2.67** by Cochran). Significant reduction of the chromosome breakage and SLRL rates was usually observed when spermatozoa exposed to EMS were stored in normal females treated with AOs. The lack of maternal

repair systems inhibited the sensitivity of females mei-9 and mei-41 to AO protective action.

Thus, the data obtained suggest DHP and GP action is mediated by defense systems. The antioxidants tested seems to affect repair pathways involved in chemical mutagenesis in Drosophila melanogaster.

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