Bleomycin And Trifluoperazine Induced Sex-Linked Recessive Lethal Mutations In The Different Stages Of Spermatogenesis In Drosophila melanogaster

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ABSTRACT

The antitumor agent bleomycin and the calmodulin inhibitor trifluoperazine were evaluated for their mutagenic potential by using Drosophila melanogaster sex-linked recessive lethal assay. Wild type type Oregon-k males were injected with 0.2 µL of the proper concentrations of the drugs. Complete and mosaic lethals were scored by the muller-5 method in five successive broods representing the different stages of spermatogenesis.

Trifluoperazine (TFP), was found highly toxic to Drosophila males, its toxicity reached 30% at 1mM and 100% at 10mM. Slightly toxic concentration of trifluoperazine (0.1mM) was used in this study and was not mutagenic to Drosophila. The injection of young males with 0.1 µg/ml bleomycin (BLM) lead to the induction of both complete and mosaic sex-linked lethals. The increase in the complete lethal frequencies was significant in the dividing stages of spermatogenesis; spermatocytes, and late and early spermatogonia, but not in either post meiotic stages; sperms and spermatids. The increase in production of sex-linked mosaic lethals was however significant only in the spermatocytes. The premeiotic stages were generally more sensitive to the bleomycin effect than the postmeiotic ones. Therefore, bleomycin is more effective on the dividing stages than the non dividing stages of spermatogenesis. The toxic effect of bleomycin on Drosophila males increased with time following the injection. The survival rate reached
zero% after six days of injection at 5 μg/ml BLM and higher concentrations of bleomycin.

The non mutagenic concentration of trifluoperazine (0.1mM) potentiated the mutagenic effect of bleomycin in *Drosophila melanogaster*. The induction of complete and mosaic lethals was increased by 1.5 and 1.25 fold respectively. The number of mutated clusters was increased by 1.6 fold and the size of the mutated sector in F1 females gonads was increased by 1.15 fold. We suggested that the potentiation of TFP to the bleomycin effect, could be due to the inhibition of a calmodulin - dependant DNA damage/repair system.