DNA PROTECTIVE POTENTIAL OF PINITOL AGAINST ETHYL METHANESULFONATE INDUCED GENOTOXITY IN *Drosophila melanogaster*

Sanja Matić[1], Snežana Stanić[2]

[1]*University of Kragujevac, Institute for Information Technologies Kragujevac, Department of Science, Jovana Cvijića bb, 34000 Kragujevac, Serbia,*

[2]*University of Kragujevac, Faculty of Science, Department of Biology and Ecology, Radoja Domanovića 12, 34000 Kragujevac, Serbia*

Corresponding author: sanjamatic@kg.ac.rs

INTRODUCTION

Pinitol, a cyclic polyo, isolated from *Ceratonia siliqua* L., has been suggested to exert a wide range of biological activities, *i.e.*, antioxidant, antitumor, hepatoprotective, antibacterial, insulinomimetic, immunomodulator, and antiaging. Potentially beneficial effects of pinitol have been reported in treatments of osteoporosis and Alzheimerôs disease. Although several studies have supported potential health benefits from pinitol, genotoxic and antigenotoxic effects is still unknown. The aim of the present study was to determine *in vivo* genotoxic effect of pinitol on ethyl methanesulfonate (EMS)-induced DNA damage in germ cells of *Drosophila melanogaster*. In order to identify compounds that might protect DNA from damage, the antigenotoxic effects of pinitol against DNA damage induced with EMS were evaluated in *D. melanogaster* males using the sex-linked recessive lethal (SLRL) test.

METHOD

To assess the genotoxic effect three days old Canton S males were treated with pinitol in concentration of 100 ppm. In order to detect protective activity against DNA damage, *D. melanogaster* males were exposed to EMS in concentration of 0.75 ppm, 24 h prior to pinitol in the concentration of 100 ppm. The standard procedure for the detection of sex linked recessive lethal mutations on *D. melanogaster* was applied.

Frequencies of sex linked recessive lethal mutations in the *Drosophila melanogaster* after the treatment with EMS and the post-treatment with pinitol

Broods		Treatments								
		S[a]	EMS[b]	P[c]	P+EMS	$t_{S/EMS}$	$t_{S/P}$	$t_{S/EMS+P}$	$t_{EMS/P}$	$t_{EMS/EMS+P}$
I	No of crosses	92	104	166	172	8.3	2.25	0.26	11.6	3.4
	No of lethal	12	64	6	16	$p < 0.001$***	$p < 0.05$*	$p > 0.05$	$p < 0.001$***	$p < 0.001$***
	% of lethal	13.04	61.5	3.6	9.3					
II	No of crosses	96	90	150	124	6.7	0.6	1.2	6.8	5.5
	No of lethal	10	44	12	20	$p < 0.001$***	$p > 0.05$	$p > 0.05$	$p < 0.001$***	$p < 0.001$***
	% of lethal	10.4	48.9	8.0	16.1					
III	No of crosses	64	108	58	74	5.3	0.8	1.2	4.5	4.2
	No of lethal	6	44	8	12	$p < 0.001$***	$p > 0.05$	$p > 0.05$	$p < 0.001$***	$p < 0.001$***
	% of lethal	9.4	40.7	13.8	16.2					
I+II+III	No of crosses	252	302	374	370	13.3	1.7	0.8	13.4	10.9
	No of lethal	28	152	26	48	$p < 0.001$***	$p > 0.05$	$p > 0.05$	$p < 0.001$***	$p < 0.001$***
	% of lethal	11.1	50.3	6.9	12.9					

The results are analysed by the test for difference in proportions, by simultaneous comparison with the positive and negative controls. Triple asterix indicates significantly higher frequency compared to EMS as positive control or to sucrose as negative control.
[a]S; sucrose; negative control, 1%; [b]EMS; ethyl methanesulfonate, positive control, 0.75 ppm; [c]P; pinitol, 100 ppm.

RESULTS

EMC induced a statistically significant sex linked recessive lethal mutations in all three broods at a dose of 0.75 ppm. The treatment with pinitol in concentration of 100 ppm reduced the frequency of sex linked recessive lethal mutations in comparison with the negative control value. Compared with the sucrose, as the negative control, pinitol decreased ($p > 0.05$) the genotoxicity of EMS in postmeiotic germinative cell line ï at spermatozoids and spermatids, and in premeiotic line ï spermatocytes. The frequency of germinative mutations induced by EMS decreased with high significance ($p < 0.001$***) after post-treatments with pinitol.

CONCLUSIONS

The results indicated that pinitol exhibited a DNA protective potential against EMS and also it did not induce the genotoxic effect alone in tested concentration in *D. melanogaster* males using the sex-linked recessive lethal test.

ACKNOWLEDGEMENTS

This work was supported by the Serbian Ministry of Education, Science and Technological Development (Agreement No. 451-03-9/2021-14/200378 and Agreement No. 451-03-9/2021-14/200122).