

EVALUATION OF NaF-INDUCED GENOTOXICITY AND OXIDATIVE STRESS AT TWO TROPHIC LEVELS: SWISS ALBINO MICE AND THE PLANT *ALLIUM CEPA*

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SUMMARY: Fluorosis is a serious public health problem affecting millions of people around the world. With the perception that fluoride (F) interacts with cellular systems, even at low concentrations, the present investigation evaluated sodium fluoride (NaF)-induced DNA damage and oxidative stress at low concentrations with both *in vitro* and *in vivo* models. The incubation of human peripheral blood mononuclear cells with various concentrations of NaF (0.1, 0.5, 1, 5, and 10 µg/mL) did not induce DNA damage but NaF-generated oxidative stress led to apoptotic cell death. Swiss albino mice exposed to NaF-containing drinking water (4, 12, and 20 mg F/L) for 30 consecutive days showed a significant increase in the frequency of micronuclei (MN) in polychromatic erythrocytes (PCEs) and structural chromosome aberrations in bone marrow cells. With the exception of spleen, DNA strand breaks were observed in multiple organs such as bone marrow, kidney, and liver. Furthermore, we observed an increase in lipid peroxidation and catalase activity with a simultaneous decrease in the reduced glutathione (GSH) level and the glutathione-S-transferase activity (GST) in the liver tissues of the mice exposed to NaF. When *Allium cepa*, a representative plant system, was treated with NaF (2.5, 5, and 10 mM) significant DNA damage and the generation of reactive oxygen species occurred at the low concentration. Taken together, our findings document that NaF has the potential to damage DNA and to enhance oxidative stress.

Keywords: *Allium cepa*; Chromosome aberrations; DNA strand breaks; Fluorosis; Mice; Mononuclear cells; Oxidative stress; Reactive oxygen species generation.

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