Cypermethrin-induced DNA damage in organs and tissues of the mouse : Evidence from the comet assay

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Abstract:

Cypermethrin is the most widely used Type II pyrethroid pesticide because of its high effectiveness against target species and its low mammalian toxicity reported so far. It is a fast-acting neurotoxin and is known to cause free radical-mediated tissue damage. The present study investigates the genotoxic effects of cypermethrin in multiple organs (brain, kidney, liver, spleen) and tissues (bone marrow, lymphocytes) of the mouse, using the alkaline comet assay. Male Swiss albino mice were given 12.5, 25, 50, 100, 200 mg/kg BW of cypermethrin intraperitoneally, daily for 5 consecutive days. A statistically significant (p < 0.05) dose-dependent increase in DNA damage was observed in all the organs assessed, as evident from the comet-assay parameters, viz., Olive tail moment (OTM; arbitrary unit), tail DNA (%) and tail length (?m). Brain showed maximum DNA damage followed by spleen > kidney > bone marrow > liver > lymphocytes, as evident by the OTM. Our data demonstrate that cypermethrin induces systemic genotoxicity in mammals as it causes DNA damage in vital organs like brain, liver, kidney, apart from that in the hematopoietic system.

Evaluation of in vivo genotoxicity of cypermethrin in Drosophila melanogaster using the alkaline Comet assay

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The single cell gel electrophoresis (SCGE) assay, also known as the Comet assay, is one of the most promising genotoxicity tests developed in recent years to measure and analyse DNA damage in single cells. The present study was undertaken to assess the in vivo genotoxicity of the synthetic pyrethroid cypermethrin in brain ganglia and anterior mid gut of Drosophila melanogaster. Freshly emerged first instar larvae (22 ± 2 h) were placed in different concentrations of cypermethrin (0.0004, 0.0008, 0.002, 0.2 and 0.5 p.p.m.) mixed in standard Drosophila food and allowed to grow. At 96 \pm 2 h, brain ganglia and anterior midgut from control and treated larvae were dissected out, single cell suspensions were prepared and a Comet assay was performed. Our results revealed a significant dose-dependent increase in DNA damage in the cells of brain ganglia and anterior midgut of D.melanogaster exposed to cypermethrin as compared with controls (P < 0.05 at 0.002 p.p.m.; P < 0.001 at 0.2 and 0.5 p.p.m.). The present study shows in vivo genotoxicity of cypermethrin even at very low concentrations, which proves D.melanogaster as a model for in vivo genotoxicity assessment using the Comet assay.

Effects of pesticides on human peripheral lymphocytes in vitro: induction of DNA damage

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Abstract:

Because of the widespread use of pesticides for domestic and industrial applications the evaluation of their genotoxic effects is of major concern to public health. Although various experimental data have provided evidence that pesticides can possess genotoxic properties in animals and in in vitro test systems after acute and chronic exposure, the information on the genotoxic effects of some of pesticides is limited and inconsistent. In the present study, the genotoxic potential of commonly used pesticides (i.e., dimethoate and methyl parathion from the organophosphate class, propoxur and pirimicarb from carbamates, and cypermethrin and permethrin from pyrethroids) have been evaluated. The genotoxic effects of these substances were examined using the single cell gel electrophoresis (comet) assay in freshly isolated human peripheral lymphocytes. The cells were incubated with 10, 50, 100 and 200 $\mu\text{g/ml}$ concentrations of the test substances for 0.5 h at 37°C and DNA damage was compared with that obtained in lymphocytes from the same donor not treated with substances. Hydrogen peroxide, 100 μ M, was used as a positive control. Within the concentration ranges studied, no significant cytotoxic effects were observed. Dimethoate and methyl parathion at 100 and 200 μ g/ml; propoxur at 50, 100 and 200 μ g/ml, and pirimicarb, cypermethrin and permethrin at 200 μ g/ml significantly increased DNA damage in human lymphocytes.