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Toxicities of Isoprothiolane on the Development of *Xenopus laevis*

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Toxic effects of Isoprothiolane in *Xenopus laevis* embryogenesis were investigated. Embryos were exposed to various concentrations of Isoprothiolane up to 160 μM. The total lethal concentration for *Xenopus* embryos by Isoprothiolane was 160 μM. The exposure to Isoprothiolane resulted in 9 different types of external malformations. In histopathological study, various dysplasias were observed in eyes, heart, gut, liver, somatic muscle, and pronephric ducts. For the investigation of tissue-specific toxic effects, an animal cap assay was performed. As a result, the differentiation of blood cells was inhibited by Isoprothiolane. Electron micrographs of tested embryos showed severe degeneration of photoreceptor cell, but it showed weak degeneration of muscle and mitochondria.

Key Words: Isoprothiolane, *Xenopus laevis,* Toxic effects, Malformation

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The kinase Activity of *N*-Acetylglucosamine Kinase is not Essential for the Role of the Enzyme in the Development of Neuronal Dendrites

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N-Acetylglucosamine kinase (GlcNAc kinase or NAGK; EC 2.7.1.59) catalyzes the phosphorylation of GlcNAc to GlcNAc 6-phosphate (GlcNAc-6-P), a component utilized in uridine diphosphate (UDP)-GlcNAc biosynthesis and in energy metabolism. Previously, it is shown that NAGK is highly expressed in neuronal soma and dendrites, and that knockdown of NAGK mRNA by small interference RNA (RNAi) resulted in degeneration of dendrites but not axons, indicating that NAGK plays essential roles in the development and/or maintenance of neuronal dendrites. The purpose of this study is to address a question on whether NAGK plays structural or enzymatic roles in the development and/or maintenance of neuronal dendrites. For this aim, four point mutants at the kinase active site and two deletion mutants were constructed, transfected rat hippocampal neurons, and observed the morphology of transfected neurons. When the point mutants (N36A, D107A, C131S and C143S) of NAGK activity site was overexpressed in the EGFP-tagged form, the number of dendritic branches increased very significantly. Overexpression of the large domain-deleted NAGK in the EGFP-tagged form, EGFP-NAGKs, resulted in dendritic degeneration. However, the overexpression of the small domain-deleted NAGK was neutral to dendrite development. These results show that the kinase activity of NAGK is not essential in the development and/or maintenance of neuronal dendrites, and it seems that the small domain of NAGK play important structural roles than the large one in this aspect.

Key words: Dendrites; GlcNAc; Hippocampal cell; N-acetylglucosamine kinase; NAGK