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Oral intake of hydrogen-rich water ameliorated chlorpyrifos-induced neurotoxicity in rats

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Abstract

Chronic exposure to low-levels of organophosphate (OP) compounds, such as chlorpyrifos (CPF), induces oxidative stress and could be related to neurological disorders. Hydrogen has been identified as a novel antioxidant which could selectively scavenge hydroxyl radicals. We explore whether intake of hydrogen-rich water (HRW) can protect Wistar rats from CPF-induced neurotoxicity. Rats were gavaged daily with 6.75mg/kg body weight (1/20 LD50) of CPF and given HRW by oral intake. Nissl staining and electron microscopy results indicated that HRW intake had protective effects on the CPF-induced damage of hippocampal neurons and neuronal mitochondria. Immunostaining results showed that the increased glial fibrillary acidic protein (GFAP) expression in astrocytes induced by CPF exposure can be ameliorated by HRW intake. Moreover, HRW intake also attenuated CPF-induced oxidative stress as evidenced by enhanced level of MDA, accompanied by an increase in GSH level and SOD and CAT activity. Acetylcholinesterase (AChE) activity tests showed significant decrease in brain AChE activity after CPF exposure, and this effect can be ameliorated by HRW intake. An in vitro study demonstrated that AChE activity was more intense in HRW than in normal water with or without chlorpyrifos-oxon (CPO), the metabolically-activated form of CPF. These observations suggest that HRW intake can protect rats from CPF-induced neurotoxicity, and the protective effects of hydrogen may be mediated by regulating the oxidant and antioxidant status of rats. Furthermore, this work defines a novel mechanism of biological activity of hydrogen by directly increasing the AChE activity.

Keywords: Acetylcholinesterase; Chlorpyrifos; Hydrogen; Neurotoxicity; Oxidative stress.

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