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Dioxin exposure is an environmental risk factor for ischemic heart disease

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Abstract

Epidemiologic studies have linked dioxin exposure to increased mortality caused by ischemic heart disease. To test the hypothesis that dioxin exposure may constitute an environmental risk factor for atherosclerosis, we exposed C57BL/6J mice to 5 microg/kg of dioxin daily for 3 d, and measured various molecular and physiological markers of heart disease. Dioxin treatment led to an increase in the urinary excretion of vasoactive eicosanoids and an elevation in the mean tail-cuff blood pressure. In addition, dioxin exposure led to an increase in triglycerides, but not in high-density lipoproteins, in both Apoe(+/+) mice and in hyperlipidemic Apoe(-/- mice. Dioxin exposure also led to an increase in low-density lipoproteins in Apoe(-/-) mice. After treatment, dioxin was associated with low-density lipoprotein particles, which might serve as a vehicle to deliver the compound to atherosclerotic plaques. Dioxin treatment of vascular smooth-muscle cells taken from C57Bl/6J mice resulted in the deregulation of several genes involved in cell proliferation and apoptosis. Subchronic treatment of Apoe(-/-) mice with dioxin (150 ng/kg, three times weekly) for 7 or 26 wk caused a trend toward earlier onset and greater severity of atherosclerotic lesions compared to those of vehicle treated mice. These results suggest that dioxin may increase the incidence of ischemic heart disease by exacerbating its severity.

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