Lifelong inorganic arsenic compounds consumption affected blood pressure in rats

Hui-Ting Yang, Hsin-Ju Chou, Bor-Chen Han and Shih-Yi Huang
Yang HT;Chou HJ;Han BC;Huang SY

Abstract

Chronic arsenic exposure is a known risk factor for cardiovascular disease and has a strong correlation with hypertension. Oxidative stress may be one of the major contributors to arsenic-induced hypertension. To investigate the antioxidative and CYP systems through which inorganic arsenic compounds may contribute to blood pressure elevation in rats, we administered 50 ppm arsenic (as arsenite and arsenate) in drinking water to Wistar rats for 200 successive days. Systolic blood pressure was determined every 20 days, and blood samples and tissues were collected at each time point for biological analysis. Compared to the control group, weight gain in the arsenic-exposed animals was slightly but significantly lower, whereas the relative weights of the various tissues was higher. Blood pressure was elevated until day 80 in both arsenic groups followed by a time-dependent change in the antioxidative enzyme system. The hypertensive effect remained until day 200 for arsenite when the change by arsenate was minimized. Patterns of antioxidative enzyme change differed between arsenite and arsenate. However, the most common marker of hypertension, the angiotensin-converting enzyme, showed no significant change in either arsenic group. CYP4A was highly expressed in both arsenic groups, particularly in the arsenite group. These results indicate that low but chronic arsenic exposure might cause elevated blood pressure and antioxidative interference. Furthermore, CYP4A might be more important than ACE in contributing to arsenic-induced hypertension.