

Clinical and Medical Research and Studies

**Research Article** 

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# Fetal Exposure to Arsenic Results in the Development of Multiple Risk **Factors for the Development of Metabolic Syndrome**

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## Abstract

Exposure to arsenic is a major concern in the United States and worldwide, since this metalloid has been associated with a number of ailments, including cardiovascular and metabolic diseases. Environmental exposures to toxicants throughout fetal development have been shown to play a critical role as triggers of adult disease.

Keywords: Arsenic; metabolic; fetal; hypercholesterolemia; hyperglycemia; NAFLD

## Introduction

Exposure to environmental arsenic is a major concern in the United States and worldwide. It is estimated that hundreds of millions of people are exposed to arsenic through drinking water on a daily basis [1]. The highest concentrations seen in drinking water are found in the endemic Blackfoot disease regions of Taiwan where mean concentrations of arsenic in water are around 700 µg/L. Affected countries include Bangladesh, with over 30 million people exposed to concentrations between 0.1  $\mu g/L$  and 864  $\mu g/L,$ India, with 40 million people exposed to concentrations exceeding 1000  $\mu$ g/L, as well as China (1.5 million people) and the United States (2.5 million people) exposed to concentrations between 1  $\mu$ g/L and 100  $\mu$ g/L of arsenic in the drinking water [2-5]. Importantly, exposure to arsenic has been associated with an array of diseases ranging from multiple forms of cancer, to developmental and reproductive effects, as well as cardiovascular and metabolic disorders [6-8].

## **Methods**

Swiss Webster pregnant mice were purchased from Harlan (Harlan Laboratories Inc, WI, USA). Treatment groups were initiated at embryonic day 6 (E6) by exposing the dams to either 100 parts per billion (ppb) sodium arsenite (NaAsO2, Sigma, St. Louis, MO, USA) or 100 ppb sodium chloride (NaCl, VWR, Aurora, CO, USA) through drinking water, and were maintained on their respective treatments until birth. Mice were housed in sterile microisolator cages and provided diet (2019 Teklad Global 19% Protein Extruded Rodent Diet, Harlan Laboratories Inc, WI, USA) and water ad libitum. Offspring from arsenic exposed dams (n=2) consisted of 3 females and 7 males (n=10), whereas offspring from control dams (n=2) consisted of 8 females and 2 males (n=10). At weaning age (day 21), mice were separated bv treatment and gender.

## Results

Given the associations between cardiovascular, metabolic disorders and obesity, we evaluated growth changes during the 36-week period after birth. Female pups exposed to arsenic showed a statistically significant decrease in weight at two weeks of age, when compared to control females (p<0.05) While this difference in weight was apparent at 2 weeks of age, female mice recovered quickly, and by 4 weeks of age, there was no difference between IU arsenic-exposed and control mice.

## Discussion

Chronic arsenic exposure has been well studied and evaluated in the context of adult disease; however, little is known about the contribution of fetal arsenic exposure to disease onset and progression. Several studies established a strong association between chronic arsenic exposure and an increased prevalence of hypertension, atherosclerosis, and ischemic heart disease-related mortalities. Importantly, these epidemiological studies have shown that the relationship between cardiovascular disease and arsenic exposure is not only dependent on dose, but also on duration of exposure. Seldom have studies evaluated the contribution of developmental exposures. However, Smith's group provided strong evidence for the association between arsenic exposure through drinking water, and a sustained increased risk in circulatory disease-related mortalities, years after exposure.

## Conclusions

In summary, we demonstrate that fetal exposure to arsenic results in the development of multiple risk factors for the development of metabolic syndrome, which are also significant contributors to cardiovascularrelated mortalities. The presence of several components of metabolic syndrome in mice suggests that fetal exposures to arsenic may contribute to dysregulation of regulatory pathways important for glucose and lipid homeostasis. Mechanisms of arsenic-mediated metabolic disruption go beyond the scope of this manuscript, and are discussed in great reviews



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however, preliminary findings from our lab indicate that arsenic may be affecting the function of key enzymes involved in energy metabolism and the tricarboxylic acid (TCA) cycle. Given the pivotal role that the liver plays in glucose and lipid homeostasis, it is likely that disruption of a central bioenergetics pathway such as the TCA cycle could contribute to the effects observed in this study.

## **Competing Interests**

The authors declare that they have no competing interests.

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