Mechanisms of Arsenic-Induced Atherosclerosis

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Arsenic is one of the natural compositions of our earth's crust that can present many lethal effects to our cardiovascular system. In fact, many individuals especially in third world and developing countries are constantly exposed to arsenic toxicity on a daily basis through the food they eat, the water they drink, and simply just the air they breathe. The purpose of this project was to further analyze the effect arsenic has on endoplasmic reticulum stress and how it can cause modifications in generated protein responses, leading to elevated risks of hypertension and atherosclerosis. Thus, it was hypothesized that with brief exposures to inorganic arsenic, it will induce endoplasmic reticulum (ER) stress and programmed cell death, leading to higher risks of cardiovascular diseases. In this experiment, human umbilical vein endothelial cells (HUVEC) and murine aortic endothelial cells (MAEC) were exposed to low concentrations of sodium arsenite. The Real-time PCR machine and flow cytometry machine were then used to deduce unfolded protein responses (UPR) and programmed cell death. Afterwards, HUVEC cells were treated with phenyl butyric acid (PBA), which helps attenuate arsenic-induced activation of ER stress. With significantly higher UPR, it increases risks of atherosclerosis. Results showed that with small concentrations of sodium arsenite, there was an increase in endothelial aggravation and cell death. Ultimately, HUVEC cells treated with PBA showed significant decreases in fold change, lessening the risk of cardiovascular disease.

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