

A Novel Treatment Approach to Mitigate Adverse Cardiovascular Effects of Burn Pit Exposure

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Anthropogenic open burning is known to release hazardous chemicals and has been associated with an increased prevalence of cardiopulmonary issues. For example, studies show that veterans who served at bases with burn pits were more likely to develop asthma, hypertension, and cardiovascular complications than individuals not exposed to the toxic air. This project studies the adverse effects of simulated burn pit exposure on mammalian cardiovascular cells and proposes a novel treatment approach. Exposure of H9C2 rat cardiomyocyte and HUVEC (Human Umbilical Vein Endothelial) cells to burn pit smoke constituents (BPC) (2,3,7,8-Tetrachlorodibenzodioxin,(TCDD), benzopyrene (BaP), fine atmospheric particulate matter<4 micrometer) for 24 hours exerts cytotoxicity in concentrations ranging from at 5 to 25 microgram/cm² in both the single and repeated BPC exposure. Cell viability was unaffected at BPC 5 microgram/cm² during a single exposure experimental setup compared to control cells. BPC exposure also induced cell death and caused a significant decrease in cell proliferation. Western blot analysis showed a significant increase in phospho-Stress Activated Protein Kinase (SAPK) levels and a significant increase in the number of phospho-gammaH2AX positive foci upon BPC exposure, indicating that BPC induces oxidative stress leading to DNA damage in these cells. Treatment with Cobinamide, a new Vitamin B12-derived antioxidant, is shown to protect cardiomyocytes and endothelial cells from BPC exposure-induced cardiac cytotoxicity. These results point to Cobinamide as a novel therapeutic drug to treat cardiovascular complications arising from burn pit smoke exposure-induced oxidative stress.

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