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Sub-cytotoxic Levels of Heavy Metals Induce Pro-inflammatory Signaling in the Aortic Endothelium without Impairing Flow-Mediated Dilation in Rats

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Abstract:

Introduction: Acute exposure to tobacco or marijuana secondhand smoke (SHS) causes endothelial dysfunction. The identity of specific SHS constituents that cause vascular toxicity is unclear. Heavy metals are present in SHS and at elevated levels in the blood of smokers, and may mediate acute endothelial dysfunction through reactive oxygen species formation and decreasing NO bioavailability. We assessed the effects of exposure to cadmium, lead, mercury, and arsenic at levels present in the blood of human smokers on flow-mediated dilation (FMD) as a measure of endothelial function in rats. We also evaluated the effects of heavy metal exposure on intimal structure, protein localization pattern, and inflammatory gene expression in the aortic endothelium. Hypothesis: Sub-cytotoxic levels of heavy metals impair FMD, alter intimal structure, and induce pro-inflammatory signaling in endothelium. Methods: We injected rats (n=8/group) with heavy metal cocktail or vehicle intravenously and quantitated pre- and post-exposure FMDs measures defined as percent vasodilation of femoral artery after transient ischemia. We performed en face aorta immunostaining and assessed endothelial cell axis alignment, cell length ratio, and localization pattern of PECAM-1, VE-cadherin, and vimentin. We also quantified gene expression of key endothelial proteins in aorta homogenates. Results: FMD was not impaired in the heavy metal group (8.8±3.6(SD)% vs. 12.9±8.0%, p=.31 or controls (7.5±2.7% vs. 8.8±5.8%, p=.63). No

significant difference in cell length ratio and endothelial x and y axes of alignment were detected between groups (p>.8) and localization of PECAM-1, VE-cadherin, and vimentin in the aorta endothelium remained unaltered following heavy metal injection. However, expression of PECAM-1 and VE-cadherin was significantly lower in the heavy metal-treated rats, while VCAM-1 gene expression was significantly higher (p<.05). Conclusion: Acute exposure to sub-cytotoxic levels of heavy metals can induce pro-inflammatory signaling in endothelium, which could potentially lead to vascular injury. However, FMD and endothelial structure remain unchanged by heavy metal exposure.

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