Impaired Endothelium-Dependent Dilation After Pulmonary Residual Oil Fly Ash (ROFA) Exposure in the Systemic Microcirculation

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Acute exposure to airborne pollutants such as solid particulate matter (PM) increases the risk of cardiovascular dysfunction, but the mechanisms by which PM evokes systemic effects remain to be proven. The purpose of this study was to determine if pulmonary exposure to a PM surrogate such as ROFA affects endothelium-dependent arteriolar dilation. Rats were intratracheally instilled with saline or ROFA at 2, 1 and 0.25 mg/rat 24 hrs prior to experiments. In vivo microscopy of the spinotrapezius muscle was used to study arteriolar responses to the Ca^{2+} ionophore A23187. A23187 was ejected via pressurized micropipette at 5, 10, 20 and 40 psi into the arteriolar lumen (resting and passive diameters = 46±5 and 110±3 μm). Bronchoalveolar lavage (BAL) identified pulmonary inflammation and damage. To determine if ROFA exposure affected arteriolar nitric oxide (NO) sensitivity, sodium nitroprusside (SNP) was iontophoretically applied at 5, 10 and 20 nAmp to arterioles of rats exposed to 0.25 mg ROFA. In saline treated rats, A23187 dilated arterioles to 9±2, 26±5, 46±7 and 72±7% of maximum. In ROFA treated rats, A23187 responses were significantly attenuated except at 5 psi. BAL fluid analysis revealed pulmonary inflammation and damage after 2 and 1 mg ROFA, (but not 0.25 mg) as evidenced by significantly higher PMN cell counts, albumin and LDH activity. SNP dilated arterioles in saline and ROFA exposed rats equally to 26±5, 43±2 and 83±3% of maximum. These results indicate that pulmonary ROFA exposure impairs endothelium-dependent arteriolar dilation. Moreover, because 0.25 mg ROFA caused neither pulmonary damage nor inflammation, it appears that ROFA exposure can impair systemic microvascular function independently of a pulmonary effect.

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ABSTRACTS
PART II

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