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Aldehydes in Cigarette Smoke Impair Vascular Endothelial Function

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Introduction: Exposure to tobacco and marijuana smoke impairs vascular endothelial function. While the particulate phase of smoke is heavily implicated, the role of volatile constituents is unclear. Smoke contains aldehydes, which are known to cause endothelial dysfunction. We explored whether two aldehydes found in smoke, acrolein and acetaldehyde, can induce endothelial dysfunction.

Hypothesis: Aldehydes in smoke impair endothelial function.

Methods: We exposed 4 groups of anesthetized rats to 3 ppm acrolein and 10-11.5 ppm acetaldehyde gases (concentrations relevant to levels in secondhand smoke), Marlboro Red cigarette sidestream smoke at modest levels ($600 \mu\text{g}/\text{m}^3$ PM_{2.5}) as a positive control, and clean air through the gas generation system as a negative control. Exposure was continuous for 10 minutes. Endothelial function (flow-mediated dilation; FMD) was quantified pre- and post-exposure by measuring femoral artery diameter with ultrasound before and after 5 min of transient ischemia and expressed as % vasodilation.

Results: Impairment of FMD was observed for acrolein (10.8 ± 1.7 (SD)% vs. $5.8 \pm 2.9\%$, $p=.001$), acetaldehyde ($8.8 \pm 2.0\%$ vs. $6.0 \pm 2.5\%$, $p=.001$), and cigarette smoke ($9.4 \pm 2.9\%$ vs. $5.8 \pm 2.0\%$, $p=.002$), but not for air ($7.9 \pm 2.0\%$ vs. $9 \pm 3.2\%$, $p=.44$) (figure; each colored line denotes a rat pre- and post-exposure; bars denote means).

Conclusions: Acrolein and acetaldehyde at levels found in secondhand smoke impair endothelial function. Our results suggest that despite a potential role of particles, volatile aldehydes may mediate part of the endothelial dysfunction caused by exposure to smoke.