Vaping to Vascular Damage: The Role of E-Cigarettes on Vascular Function

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Background: Electronic cigarettes (E-cigs) have exponentially increased in popularity and usage due to the perception that e-cigs are safe, their usefulness as a cigarette-smoking cessation tool, and the appeal of heavily marketed flavors. There is little known about the long-term effects of E-cig vapor exposure, particularly in the context of vascular dysfunction. We hypothesize that the long-term use of E-cig vapor decreased aortic function, and increase aortic stiffness.

Methods: Data were obtained from C57BL/6 female mice exposed to 3R4F reference cigarette (N=5-7), cappuccino flavored E-vapor (18 mg/ml nicotine, N=7), or filtered air (N=7-8) for 4 h/day, 5 d/wk for 8 months. Food and water were administered ad libitum. Before and after the exposure, in-vivo aortic stiffness (pulse wave velocity) was measured using B-mode and Doppler ultrasound by obtain blood flow signals at the aortic arch and before the carotid bifurcation from a single image, which were gated to the EKG. At the end of the exposure the thoracic aorta was dissected, sectioned into rings and mounted onto an ex-vivo wire tension myograph system. Force transduction was used to measure the changes in aortic tension in response to methacholine, or sodium nitroprusside.

Results: Aortic stiffness increased (0.45 ± 0.20 m/s) in the air-exposed group, reflecting the normal aging process. However, an accelerated age-associated aortic stiffness was noted in the cigarette (1.28 ± 0.27 m/s) and E-cig (1.14 ± 0.24 m/s) groups (ANOVA, $p<0.05$). In animals exposed to filtered air, the maximal aortic relaxation achieved to methacholine was 90%, compared to 60% and 70% in the cigarette and E-cig groups, respectively ($p<0.05$). No differences were noted in sodium nitroprusside dilation between groups.

Conclusion: Our data suggests that 8 months of E-cig significantly accelerated the age-associated increase in aortic stiffness, and significantly impaired aortic endothelial-dependent but not endothelial-independent dilation. These data show the E-cigs induce similar vascular dysfunction to cigarette smoke exposure suggesting that E-cig have similar risk to develop accelerated cardiovascular aging and disease.

Funding support by WVU-Marshall Health Cooperative Initiative Award; with additional support from NIH P20GM103434 (West Virginia IDeA Network for Biomedical Research Excellence), NIH P20GM104942 (West Virginia Clinical Translational Science Institute) and National Institutes of Health Grants 1P20 GM-109098, RR-2865AR, and P20 RR-016477.