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Acute effects of electronic hookah smoking on endothelial function, inflammation and oxidative stress

Authors:
M Rezk-Hanna¹, E Ricci¹, E Ilkharo¹, ML Brecht¹, ¹University of California Los Angeles - Los Angeles - United States of America,

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Background: Electronic nicotine delivery systems (ENDS) are a new rapidly growing global epidemic. More recently, electronic (e-) hookahs, have increased in popularity in the United States, with the greatest uptake by young female adults, who endorse marketing claims that these products are safer alternatives to traditional hookah tobacco smoking. Unlike other ENDS such as e-cigarettes, e-hookah bowls are used through traditional waterpipes, allowing the vapor—containing aerosolized nicotine, propylene glycol, glycerin, and flavorings—to pass through a water-filled basin, before it is inhaled through the user’s mouth. Contributing to e-hookah bowls’ popularity is the belief that e-hookah flavored smoke is detoxified as it passes through the water-filled basin, rendering e-hookah a safer tobacco alternative. However, an e-hookah bowl delivers flavored nicotine by creating a vapor of fine (<2.5 µm) and ultrafine particles (<0.1 µm) that could induce vascular toxicity.

Purpose: To test the acute effect of electronic hookah smoking on endothelial function, inflammation and oxidative stress.

Methods: In 17 healthy young adults who smoke hookah but not cigarettes (age 26±1 years, mean±SE; BMI 23.8±0.7 kg-m2), we measured brachial artery flow-mediated dilation (FMD) before and after a 30-minute e-hookah bowl smoking. To test for inflammatory mediation, pro-inflammatory cytokines hsCRP, TNF-a, and fibrinogen were collected before and after smoking. To test for oxidative stress mediation, on a separate day, the acute effect of e-hookah smoking on FMD was examined after intravenous infusion of Vitamin C, an effective antioxidant. Plasma nicotine levels were collected before and after the smoking session. The same measurements were performed before and after a subset of subjects (n=8) performed a sham-smoking control study.

Results: E-hookah smoking, which markedly increased plasma nicotine (plasma nicotine: +6.07±1.87, p=0.018) and mean arterial pressure (mean arterial pressure: +12±2 mm Hg, p<0.001), acutely decreased FMD from 8.04±0.68 to 6.14±0.52 %, p < 0.001, indicating impaired endothelial function. While fibrinogen and TNF-a levels increased from 225.31±7.41 to 236.77±9.79, p =0.026 and from 0.80±0.04 to 0.87±0.05, p=0.036, respectively, hsCRP did not change (P=ns). Vitamin C administration prevented the acute FMD impairment by e-hookah smoking (P=ns). All parameters were unchanged during sham-control studies.

Conclusions: In contrast to the widespread popular belief that e-hookah is safe, the data herein show that each e-hookah session constitutes a potent vascular toxin acutely impairing endothelial function and inducing an inflammatory state. That the acute impairment in FMD with electronic hookah is restored with administration of the potent antioxidant Vitamin C suggest that elevated vascular oxidative stress as a key mechanism involved. These new data provide evidence to counter claims that e-hookah is a safer tobacco alternative.