

Effects of Acute Vaporized Nicotine in Non-Tobacco Users at Rest and during Exercise

JUAN MEJIA, JEREMY GARCIA, WILLIAM H COOKE, CAROLINE RICKARDS, and DONOVAN L FOGT

Integrative Cardiopulmonary and Autonomic Performance Laboratories; Department of Kinesiology, Health, and Nutrition; University of Texas at San Antonio; San Antonio, TX

Category: Undergraduate

Mentors: Donovan L Focht, donovan.focht@utsa.edu; William H Cooke, william.cooke@utsa.edu

ABSTRACT

Smokers, and even non-smokers, may utilize vaporized nicotine delivered by electronic cigarette (EC) due to the perception that EC are “healthier” than traditional tobacco cigarettes. The effects of vaporized nicotine delivered by EC on resting blood pressure (BP) and metabolic rate (RMR), or BP and aerobic power during exercise have not been studied. This investigation tested the effects of acute vaporized nicotine inhalation by EC on resting BP and RMR and cycle exercise BP, metabolic responses, and aerobic power in young, normotensive non-smokers. Using a double-blind design, 20 subjects (10 female; 23.1±2.5 years, 1.69±0.1 m, 70.6±14.9 kg; 22.1±11.0% body fat) self-reporting as healthy and non-smoking participated. All subjects participated in two randomized trials: placebo (0 mg nicotine) or nicotine (18 mg nicotine). Participants inhaled from EC once every 30 s for 10 min (20 inhalations total) during each trial. RMR was assessed 40 min later by indirect calorimetry followed by an incremental cycle test. Participants' pre-inhalation SBP, DBP, and HR were also not significantly different between conditions or from those averaged over the last 5 min of the indirect calorimetry protocol. Cotinine, a stable nicotine metabolite, was assessed on post-inhalation (i.e., 10 min) urine samples. The cotinine concentration ranges, as scored using the semi-quantitative urine analysis kit strips, were significantly higher ($p < 0.001$) after nicotine inhalation (30-100 ng • ml⁻¹) compared to placebo (0-10 ng • ml⁻¹). RMR was assessed ~40 min after the last EC inhalation. RMR ($p = 0.39$), VO₂ ($p = 0.5$), RQ ($p = 0.15$), and HR ($p = 0.47$) were not significantly different between the placebo and nicotine trials. Compared to the placebo trial, nicotine use resulted in a 3.7 mmHg lower resting SBP ($p = 0.04$) but a 3.0 mmHg higher DBP ($p = 0.04$). VO_{2peak} was not different between the nicotine trial (2.3±0.8 L • min⁻¹) and placebo trial (2.3±0.7 L • min⁻¹) trials ($p = 0.77$). No statistically distinguishable difference was observed for W_{peak} between nicotine (201.0±53.8 W) and placebo (204.8±57.8 W) ($p = 0.29$). There was a main effect of time over the cycle test for VO₂, energy expenditure, RQ, and HR but no between treatment effects. A main treatment effect was identified for DBP, which was higher following nicotine compared to placebo at all time points during the test ($p = 0.05$). No time by treatment interaction was identified for any variable during exercise. Exercise DBP_{peak} after nicotine (79.4±7.6) was significantly higher ($p = 0.02$) than placebo (74.9±8.3 mmHg). Peak SBP was not different between trials ($p = 0.14$). Our results show that acute vaporized nicotine inhalation via EC increases resting and exercise DBP but does not affect RMR or cycle aerobic power in young, normotensive non-smokers.