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MEETING ABSTRACT Open Access

Cutaneous vascular & sudomotor responses to heat-stress in smokers & non-smokers

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Introduction

As approximately one billion people worldwide are chronic smokers [1] it is important to determine smokers' thermoregulatory responses to heat-stress. Although local maximal vasodilation may be attenuated in smokers [2], skin blood flow responses during whole-body heat stress are unknown. Moreover, it is unknown if sweat rate is altered in smokers; theoretically the binding of nicotine to nicotinic acetylcholine receptors [2] may initiate an earlier onset of sweating during whole-body heat stress compared to non-smokers [3]. The purpose of this study was to compare cutaneous vascular and sudomotor responses to whole-body passive heat-stress between smokers and non-smokers.

Methods

Nine male chronic smokers [SMK; 10 (6) cigarettes/day for 11.8 (9.5) y; 26 (8) y; 177.7 (6.6) cm; 80.6 ± 21.1 kg] and 13 male non-smokers [N-SMK; 28 (9) y; 177.6 (6.8) cm; 77.2 (8.2) kg] were matched for age, height, body mass, and exercise habits (all $p > 0.05$). Subjects were passively heated via water-perfused suits until gastrointestinal temperature (T_{gi}) increased 1.5 °C. Local sweat rate (LSR) via ventilated capsule and cutaneous vasomotor activity (CVC) via Laser Doppler on the forearm were continuously recorded; blood pressure, heart rate, sweat gland activation (SGA), sweat gland output (SGO), T_{gi}, and mean-weighted skin temperature (T_{sk}) were taken at baseline and each 0.5 °C T_{gi} increase. LSR and CVC onsets and sensitivities were calculated with mean body temperature (T_b) = 0.9*T_{gi} + 0.1*T_{sk} [4].

Table 1 Mean (SD) CVC and LSR parameters on the forearm for SMK and N-SMK during passive heat stress

	Measurement	Smokers	Non-smokers
CVC	CVC onset (ΔT_b from baseline, °C)	0.31 (0.12)	0.61 (0.21)*
	CVC plateau (% of max)	68.4 (27.4)	68.4 (21.6)
	CVC sensitivity (Δ%max per °C ΔT_b)	82.5 (46.2)	58.9 (23.3)
LSR	LSR onset (ΔT_b from baseline, °C)	0.35 (0.14)	0.52 (0.19)*
	LSR plateau ($mg \cdot cm^{-2} \cdot min^{-1}$)	0.79 (0.26)	1.00 (0.13)*
	LSR sensitivity ($\Delta mg \cdot cm^{-2} \cdot min^{-1}$ per °C ΔT_b)	0.60 (0.40)	0.63 (0.21)

*Significant difference between groups ($p < 0.05$).

Results

No differences existed between SMK and N-SMK for T_{gi}, T_{sk}, T_b, heart rate, mean arterial pressure, LSR, CVC, and SGA with each 0.5 °C T_{gi} increase (all $p > 0.05$). Overall, SGO tended to be lower in SMK than N-SMK [SMK = 5.94 (3.49) vs. N-SMK = 8.94 (3.99) $\mu g \cdot gland^{-1} \cdot min^{-1}$; $p = 0.08$].

Discussion

Smokers' CVC and LSR onsets occurred at an earlier T_b than non-smokers, possibly because heat stress enhances nicotine kinetics (*i.e.* binding of nicotine to nicotinic acetylcholine receptors; [2,3]). The lower LSR at plateau during whole-body heating might indicate a thermoregulatory impairment in young smokers, and is likely a result of decreased sweat gland output and not activation.

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Conclusion

Compared to non-smokers, smokers had an earlier onset but similar sensitivity (i.e. increase in response per increase in T_b) for sweating/cutaneous vasodilation. These data suggest that overall, most young chronic smokers' thermoregulatory responses to whole-body passive heat stress are not impaired.

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