Alcohol lowers the vasoconstriction threshold in humans without affecting core cooling rate during mild cold exposure

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Abstract. Elevated blood alcohol levels are often seen in hypothermia and hyperthermia related deaths, leading to the belief that alcohol renders humans poikilothermic. We examined the core temperature ($T_{co}$) thresholds for sweating, vasoconstriction and shivering as well as core cooling rates of seven subjects immersed in 28 °C water. On two separate days, subjects exercised on an underwater cycle ergometer to elevate $T_{co}$ above the sweating threshold. They then rested and cooled until they shivered vigorously. Subjects drank orange juice ($7 \text{ ml kg}^{-1}$) prior to immersion during the control trial and $1 \text{ ml kg}^{-1}$ absolute ethanol, added to orange juice in a 1:6 ratio, during the alcohol trial. Mean blood alcohol concentration (breath analysis) was $0.097 \pm 0.010 \text{ g}\%$ at the start of cooling and $0.077 \pm 0.008 \text{ g}\%$ at the end of the cooling period. Alcohol lowered the vasoconstriction threshold by $0.32 \pm 0.2 \text{ °C}$ and elevated finger tip blood flow, but had no effect on thresholds for sweating and shivering or core cooling rate. Considering these minor effects it is unlikely that moderate alcohol consumption predisposes individuals to hypothermia or hyperthermia via impaired thermoregulation, but rather likely due to behavioral factors.

Key words: ethanol - shivering - sweating - temperature regulation - vasodilation.

Introduction

It is a common belief that alcohol renders humans poikilothermic. Supporting evidence for this belief comes from studies showing that alcohol prevents effective thermoregulation during heat stress in animals (Kalanl and Lâ, 1984), and also reduces the ability of humans to cope with the cold by increasing heat loss through vasodilation (Fellows et al., 1984) and decreasing shivering and therefore, heat production (Graham and Baulk, 1980). Empirical evidence also suggests that alcohol impairs normal thermoregulatory processes in humans as elevated blood alcohol levels are often seen in hypothermia and hyperthermia related deaths (Kortelainen, 1987). Because these clinical studies are retrospective, it is not known whether the effect of alcohol was through impaired thermoregulation, cardiovascular compromise or even behavioral factors.

There are several limitations to the previous work on the effects of alcohol on human thermoregulation. 1) Although retrospective surveys implicate alcohol in hypothermia and hyperthermia related deaths, the effect of alcohol could be entirely behavioral. 2) Little information is available on the effects of alcohol during heat challenge. 3) The core temperature thresholds for warm and cold thermoregulatory responses following alcohol ingestion have not been examined. 4) Finally, most studies have used skin temperature as an indirect indicator of peripheral vessel tone and conclude that the vasodilatory effect of alcohol is overridden by the vasoconstriction produced by the cold stress (Andersen et al., 1963; Fox et al., 1979; Graham and Baulk, 1980; Martin and Cooper, 1978; Martin et al., 1977; Vangaard, 1978). Therefore, more reliable measures of blood flow should be used during milder cold exposure.

The purpose of this study was to determine the effects of alcohol on human thermoregulatory response thresholds and core cooling rates during mild cold stress under the condition of constant skin temperature. Accordingly, on two separate days, subjects exercised on an underwater cycle ergometer to elevate esophageal temperature ($T_{es}$) above the sweating threshold. They then rested and cooled until they shivered vigorously. Subjects drank orange juice ($7 \text{ ml kg}^{-1}$) prior to immersion during the control trial and $1 \text{ ml kg}^{-1}$ absolute ethanol in an equal volume of orange juice during the alcohol trial. We hypothesized that alcohol increases the core temperature threshold for sweating, decreases the thresholds for vasoconstriction and shivering and accelerates core cooling during mild cold stress.

Methods

With approval from our Faculty Human Ethics Committee, 7 healthy subjects (2 female) participated after giving their written, informed consent. Subjects were (mean ± SD) 23.3 ± 3.6 years old, 1.74 ± 0.05 m tall, weighed 69.7 ± 3.8 kg and had a sum of 4 skinfolds of 48.1 ± 11.6 mm. Female subjects were tested during the follicular phase (days 1-9) of their menstrual cycle.

Esophageal, forearm ($T_{fa}$) and finger tip ($T_{fi}$) temperatures, and heart rate were monitored continuously. Sweat rate (adjusted for the skin surface area under the capsule) was measured using a ventilated capsule (~5 x 3.5 cm) placed on the forehead.

Fingertip blood flow was assessed using a Perfusion Index derived from an Ohmeda Biox 3700 Pulse Oximeter (Ohmeda, Louisville, CO) with a clamp-type oximeter probe placed on the middle digit. The oximeter was modified and a program developed by Ohmeda Inc. to compute a perfusion index. This method correlates well with blood flow as measured by volume plethysmography. Oxygen consumption ($VO_2$) was determined by an open circuit method. Blood alcohol concentration (BAC)
was determined via breath analysis using a hand-held Alcosensor IV (Intoximeters Inc., St. Louis, MO) which had been previously validated against actual blood concentrations.

Each subject participated in a control and an alcohol trial in an equally balanced order, each on a separate day. Baseline data were collected for a period of 20 min. Subjects then ingested a drink 15 min prior to immersion. The drink for the control trial was orange juice. For the alcohol trial, 1 ml/kg absolute ethanol was added to orange juice in a 1:6 ratio. The subjects then sat on an underwater ergometer immersed in 28 °C water. They then exercised for 25 min (at 50% of their previously determined maximum workload) to elevate their core temperature and thus initiate sweating (Mekjavic et al., 1991). They then cooled passively, allowing determination of core temperature thresholds for sweating, vasoconstriction and shivering, at a constant skin temperature, as well as the Tes cooling rate.

A top up drink of 1/4 the original volumes was administered during exercise. This produced post-exercise blood alcohol concentrations ranging from 0.071 to 0.110 g% with a mean BAC of 0.097 ± 0.01 g%. Mean BAC at the end of the cooling period was 0.077 ± 0.01 g%. In all cases, Tes was elevated past the sweating threshold. Immediately following the exercise period, subjects rested their left forearm on a shelf just above water level and the pulse oximeter probe was attached to the middle finger to monitor finger tip blood flow. Subjects remained seated on the ergometer in the circulated water and cooled passively until Tes dropped below the thresholds for sweating, vasoconstriction and finally, shivering.

Statistical Analysis. The sweating threshold was defined as the Tes at which sweat rate stabilized at the baseline level (~50 g·m⁻²·hr⁻¹) (Kurz et al., 1993). The threshold for vasoconstriction was defined when fingertip blood flow reached a minimum (Kurz et al., 1993). The shivering threshold was indicated by a sustained elevation in VO₂ above the baseline level (Mekjavic et al., 1991).

Paired t-tests were used to test for significant differences in thermoregulatory response thresholds and the Tes cooling rates between the control and alcohol conditions (α=0.05). Mean data for VO₂, sweat rate, perfusion index, heart rate and minute ventilation (VE) were plotted for the two conditions and analyzed by 2 way ANOVA with a blocked repeated-measures design to determine differences between or within conditions (α=0.05). The Fisher PLSD test was used for post-hoc analysis of significant differences.

Results

Exercise elevated Tes by 0.66 ± 0.15 °C and 0.47 ± 0.14 °C during the control and alcohol trials, respectively (N.S.). The post-exercise Tes cooling rate was similar during control (1.1 ± 0.4 °C·hr⁻¹) and alcohol (0.94 ± 0.4 °C·hr⁻¹) trials.

VO₂ followed a nearly identical post-exercise pattern in control and alcohol conditions, rapidly falling from 1545 ± 221 (control) and 1499 ± 351 (alcohol) ml·min⁻¹ and stabilizing at resting levels of 457 ± 130 (control) and 434 ± 95 (alcohol) ml·min⁻¹ within 10 min. In both trials, VO₂ subsequently increased slowly with time as core temperature decreased. VE followed a pattern similar to VO₂ falling from 39.2 ± 6.4 l·min⁻¹ to 15.0 ± 2.5 l·min⁻¹ within 10 min post-exercise during control (the mean value from 10 to 58.5 min post-exercise was 13.7 ± 1.0 l·min⁻¹). During the alcohol trial VE fell from 37.5 ± 6.4 l·min⁻¹ to 14.1 ± 3.2 l·min⁻¹ within 10 min post-exercise (the mean value from 10 to 58.5 min post-exercise was 13.1 ± 0.7 l·min⁻¹).

Sweating also followed a similar post-exercise pattern in both conditions decreasing from 505 ± 149 g·m⁻²·hr⁻¹ (control) and 500 ± 106 g·m⁻²·hr⁻¹ (alcohol) to threshold values (i.e., 50 g·m⁻²·hr⁻¹) within 20 min. Finger tip blood flow fell gradually during cooling with perfusion index values more than doubled during the alcohol trials (P<0.05).

Thermoregulatory response thresholds are reported as the change in core temperature relative to the baseline resting level (Mekjavic et al., 1991). There were no significant differences between control and alcohol trials for sweating thresholds (ΔTes = 0.12 ± 0.26 °C vs. 0.07 ± 0.15 °C above baseline, respectively) or shivering thresholds (ΔTes = 0.37 ± 0.28 °C vs. 0.48 ± 0.34 °C below baseline, respectively) as there was great variability in these responses. However, alcohol lowered the vasoconstriction threshold in all subjects, significantly reducing the mean vasoconstriction threshold from a ΔTes of 0.00 ± 0.30 °C for control to -0.32 ± 0.26 °C below baseline with alcohol (p<0.05) (Fig. 1).

Discussion

This is the first study to examine the effects of alcohol on both warm and cold thermoregulatory response thresholds and core cooling rates in humans exposed to a mild cold stress (28 °C water). Our data suggest that the main effect of alcohol ingestion during mild cold stress is a downward shift in the core temperature threshold for vasoconstriction of -0.3 °C. As a result, the perfusion index was higher during the cooling period in the alcohol condition. Most studies have failed to demonstrate a significant vasodilatory effect of alcohol during mild (Martin and Cooper, 1978), moderate (Andersen et al., 1963; Martin et al., 1977) or severe (Fox et al., 1979) cold stress. These studies generally suggest that alcohol does not affect skin blood flow because the primary vasodilatory effect of alcohol is overridden by the vasoconstriction response to cold stress. A major limitation of these studies is that skin temperature (of submersed skin)