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Alcohol 42 (2008) 207–212

ALCOHOL

Effects of alcohol on autonomic responses and thermal sensation during cold exposure in humans

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Received 29 September 2007; received in revised form 10 January 2008; accepted 17 January 2008

Abstract

We investigated the effects of alcohol on thermoregulatory responses and thermal sensations during cold exposure in humans. Eight healthy men (mean age 22.3 ± 0.7 year) participated in this study. Experiments were conducted twice for each subject at a room temperature of 18°C. After a 30-min resting period, the subject drank either 15% alcohol at a dose of 0.36 g/kg body weight (alcohol session) or an equal volume of distilled water (control session), and remained in a sitting position for another 60 min. Mean skin temperature continued to decrease and was similar in control and alcohol sessions. Metabolic rate was lower in the alcohol session, but the difference did not affect core temperature, which decreased in a similar manner in both alcohol and control sessions (from 36.9 ± 0.1°C to 36.6 ± 0.1°C). Whole body sensations of cold and thermal discomfort became successively stronger in the control session, whereas these sensations were both greatly diminished after drinking alcohol. In a previous study we performed in the heat, using a similar protocol, alcohol produced a definite, coordinated effect on all autonomic and sentient heat loss effectors. In the current study in the cold, as compared to responses in the heat, alcohol intake was followed by lesser alterations in autonomic effector responses, but increased changes in sensations of temperature and thermal discomfort. Overall, our results indicate that although alcohol influences thermoregulation in the cold as well as in the heat, detailed aspects of the influence are quite different.

Keywords: Alcohol; Cold exposure; Thermoregulation; Thermal sensation; Human

Introduction

The pharmacological and physiological effects of alcohol are very complex, as are its effects on the thermoregulatory systems of animals. In addition to possessing an unusually steep dose–response curve (Crawshaw et al., 1998), alcohol affects body temperature in two disparate ways. High alcohol doses and severe environmental conditions lead to nonspecific disruptive effects, with very hot and very cold environments producing hyperthermia and hypothermia, respectively (see Yoda et al., 2005). Less severe environmental conditions and moderate doses of ethanol lead to decreases in body temperature. This fall in body temperature has been demonstrated in mice, rats, and fish and is due to a decrease in the regulated body temperature (Gordon & Stead, 1986; Gordon et al., 1988; O'Connor et al., 1988, 1989). The temperature decrease appears to be an adaptive response, because the lethal dose of ethanol in mice is decreased by 21% following a 17°C decrease in body temperature (Malcom & Alkana, 1983).

In humans, the effects of alcohol on temperature regulation are particularly difficult to discern. The overall effect seems to be less than with other, smaller mammals (Johnston et al., 1996), and although many studies have been published, there is little commonality between alcohol dose, type and degree of thermal stress, and experimental methodology. Although initial studies on the effect of alcohol in a warm environment showed little effect on thermoregulation, they were performed in situations of extreme heat stress involving either 40°C water immersion (Allison & Reger, 1992) or exercise at 35°C (Desruelle et al., 1996).

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0741-8329/08/$ – see front matter
doi: 10.1016/j.alcohol.2008.01.006

By administering alcohol (0.36 g/kg) at 33°C, a more moderate condition, we were able to demonstrate a coordinated heat loss response. For period from 10 to 30 min after alcohol ingestion, sweat rate and skin blood flow increased significantly over control values, as did the perception of warmth. During this same period, core temperature fell significantly for the alcohol group. The perception of warmth increased in spite of a decrease in the mean skin temperature, due to increased sweating.

As was the situation in the heat, previous work with humans involving alcohol administration in a cold environment has produced enticing results. Although administration of moderate doses of alcohol to humans in the cold typically has minor effects on core body temperature (T_{core}), or thermal effector systems (Andersen et al., 1963; Fellows et al., 1984; Fox et al., 1979; Graham & Baulk, 1980; Johnston et al., 1996; Martin et al., 1977), it has been noted that perception of cold as well as the associated cold discomfort are clearly decreased following alcohol ingestion (Andersen et al., 1963; Fox et al., 1979; Graham & Baulk, 1980; Martin et al., 1977). This relationship between thermal sensation and physiological thermoregulatory responses is atypical and has led to disparate suggestions such as that although alcohol's behavioral effects could be used for minimizing the discomfort of sudden cold exposure (Martin et al., 1977), they also might be a major factor in the development of accidental hypothermia (Johnston et al., 1996).

Thus, there are both theoretical and practical reasons for further study of the effects of alcohol in a cold environment. Because we established that a clear dose and protocol for eliciting a consistent array of heat loss responses for all thermoeffector systems in a warm environment (Yoda et al., 2005), in this study we administered an identical dose of alcohol in an environment of moderate cold (18°C). We used the same protocol as in the experiments at 33°C. Our first hypothesis was that, because the dose and protocol were identical, alcohol ingestion would be followed by consistent decreases in heat production and in the sensation of cold discomfort, as well as increases in heat loss. Our second hypothesis was that changes in cold discomfort and cold sensation would not be directly related to alterations in skin temperature.

Materials and methods

Eight healthy, male Japanese subjects participated in the study. Subjects were screened by a personal history as well as by medical examination. All were occasional drinkers but no subjects had a current or past diagnosis of alcohol abuse or dependence. Some Japanese (5–10%) cannot drink alcohol at all, probably because of the lack of mitochondrial aldehyde dehydrogenase (ALDH2) activity (Harada et al., 1980; Shibuya et al., 1989). Such potential subjects were screened out of these experiments. The subjects gave informed consent for the experimental protocol, which was approved by the Human Research Ethics Committee in the School of Sport Sciences, Waseda University. Their mean age was 22.3 ± 0.7 (S.E.M.) years, body weight 67.0 ± 2.0 kg, and height 170.6 ± 1.5 cm.

Experiments were conducted twice for each subject. They fasted from 8:00 pm on the day before the experiment, but were allowed an isotonic drink the following morning. They arrived at the laboratory at 8:00 am and changed to shorts, after which they entered the environmental chamber (18°C with 50% relative humidity). The subjects then rested in a sitting position for 1 h during which time all measuring devices were applied. They rested for another 30 min as baseline data were obtained. Then, the subjects drank either 15 vol% (ml/100 ml) alcohol at a dose of 0.36 g/kg body weight (alcohol session) or an equal volume of distilled water (control session), and remained in a sitting position for another 60 min. Ethanol of 99.5% was used and given as a 15 vol% solution diluted with distillated water. The order of the two sessions was randomly chosen; there was a 2-day interval between experiments. To avoid affecting T_{core} or the temperature sensor (see below), both alcohol and water were ingested at 37°C. The alcohol solution was prepared immediately before drinking.

T_{core} was measured with a telemetry system (CoreTemp2000, HIT Technologies, Inc.). The transmitter pill was swallowed 90 min before the initiation of baseline data recording. Recordings of T_{core} were made each min and were presented as 10 min means. Heart rate (HR) was measured with the same device (CoreTemp2000, HIT Technologies, Inc.) and was similarly recorded. Skin temperatures at eight sites (forehead, chest, back, arm, hand, thigh, calf, and toe) were measured with copper–constantan thermocouples. Mean skin temperature (mean T_{sk}) was calculated from temperatures of the eight skin sites according to the area weighting formula of Hardy and DuBois (1938). Skin temperature was recorded every 10 s and averaged over 10 min. Indirect calorimetry was used to assess metabolic rate (MR). Oxygen and carbon dioxide concentrations of the expired gases were collected from a valved face mask and were analyzed every 1 min (AE-300, Minato Medical Science). Metabolic rate was calculated from the values of the nonprotein respiration quotient, the oxygen consumption rate, and the flow rate, and was expressed as kilocalories per body surface area (m^2) per hour.

Hensel (1981) delineates two kinds of sensory awareness to temperature—temperature sensation and thermal comfort. Temperature sensations provide an organism with an assessment of the thermal status of the immediate surround, whereas thermal comfort relates the thermal condition of the body to ''optimal'' conditions. When the body is near or at ''optimal'' the person will report comfort, but as the thermal condition of the body moves farther above or below the ''optimal'' level, greater feelings of discomfort will ensue. Both types of thermal awareness are likely to be involved in behavioral thermoregulatory responses, so we