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Influence of osmotic stress on thermal perception and thermoregulation in heat is different between sedentary and trained men

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HIGHLIGHTS

- The effect of hyperosmolality on thermal perception was evaluated.
- The effect was compared between sedentary and endurance-trained men.
- Hypertonic saline infusion attenuated thermal sensation in heat in sedentary men.
- The infusion also attenuated autonomic thermoregulatory responses in sedentary men.
- Autonomic thermoregulatory responses were inhibited in endurance-trained men.

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ABSTRACT

Hyperosmolality in extracellular fluid in humans attenuates autonomic thermoregulation in heat, such as sweating and blood flow in the skin. However, exercise training minimizes the attenuation. The aim of the present study was to clarify the influence of hyperosmolality on thermal perception and to assess the training effect of exercise. Ten sedentary (SED) and 10 endurance-trained (TR) healthy young men were infused with 0.9% (normal saline [NS]) or 3% NaCl (hypertonic saline [HS]) for 120 min on two separate days. After infusion for 20 min, heat stimulus to the skin of the whole body was produced by a gradual increase in hot water-perfused suit temperature (33 °C, 36 °C, and 39 °C), which was first used in the normothermic condition and then in the mild hyperthermic condition (0.5–0.6 °C increase in esophageal temperature) and controlled by immersion of the lower legs in a water bath at 34.5 °C and 42 °C, respectively. Thermal sensation and comfort were rated at the time of each thermal condition. Plasma osmolality increased by ~10 mosmL/kg·H_2O in the HS trial. In the mild hyperthermic condition, increases in sweat rate and cutaneous vascular conductance were lower in the HS than in the NS trial in both the SED and TR groups ($p < 0.05$). In the SED group, thermal sensation in the mild hyperthermic condition was lower in the HS than in the NS trial ($p < 0.05$); there was no significant difference between the trials in the TR group. These results might indicate that hyperosmolality attenuates thermal sensation with heat and that exercise training eliminates the attenuation.

1. Introduction

Prolonged and heavy exercise in heat is likely to induce hypohydration due to increased sweating and insufficient water intake. In turn, the hypohydration impairs autonomic thermoregulation such as skin vasodilation and sweating, which results in hyperthermia [1–3]. The impaired autonomic thermoregulatory responses are important for fluid preservation in the body and/or maintaining greater central blood volume. The underlying mechanism is considered to include the two factors of reduced blood volume and increased plasma osmolality (hyperosmolality) [1–3].

In humans, hypohydration also decreases the thermal sensation of heat [4]. An uncomfortable thermal feeling is considered the motive initiating behavioral thermoregulatory responses, such as escaping from the heat, removing clothes, and turning on an air conditioner [5]. Furthermore, we previously reported that plasma hyperosmolality decreases heat-escape behaviors in mice [6]. Thus, plasma osmolality could be a factor modulating behavioral thermoregulatory responses in humans. However, no experimental evidence is currently available.

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In humans, acclimation to heat is induced by regular exercise in heat [7], which involves both central and peripheral mechanisms. For example, the core temperature threshold for skin vasodilation and sweating decreases, and hypertrophy or hyperplasia of the sweat glands is observed. Exercise training helps to maintain autonomic thermoregulatory responses to heat even in the presence of plasma hyperosmolality [8] and might decrease the plasma osmolality threshold for the autonomic responses. However, the effect of exercise training on the thermal perception (thermal sensation and comfort) of heat is not known, especially with plasma hyperosmolality.

In the present study, we tested the following three hypotheses: i) plasma hyperosmolality, achieved via intravenous infusion of hypertonic saline, attenuates the thermal perception of heat; ii) exercise training prevents the impairment of the autonomic thermoregulatory responses by decreasing the sensitivity to plasma osmolality; and iii) in the presence of elevated plasma osmolality, exercise training modulates the thermal perception of heat.

2. Materials and methods

2.1. Subjects

Twenty healthy men were divided into two groups: sedentary group (SED, $n = 10$) who had not exercised on a regular basis for >3 years and trained (TR, $n = 10$) group who engaged in regular (≥4 days/week) and strenuous running for >3 years. The subjects in the TR group were middle or long distance runners. Because daily training results in a greater improvement in sweating for distance runners than for sprinters [9–11], heat acclimation would be much greater in the middle or long distance runners.

The groups were matched for age (mean ± standard deviation, 23.3 ± 3.3 and 21.9 ± 2.0 years, respectively), height (170.0 ± 3.1 and 170.8 ± 5.8 cm, respectively), body weight (60.9 ± 7.8 and 61.2 ± 7.3 kg, respectively), and body surface area (1.70 ± 0.11 and 1.71 ± 0.11 cm^2, respectively). Subjects were informed of the experimental procedures and potential risks and provided signed consent. The experimental procedure was approved by the Human Research Ethics Committee of Waseda University. The study was also conducted in accordance with the guidelines of the Helsinki Declaration.

2.2. Study design

Each subject visited the laboratory on three separate days, with at least 1-week intervals. On the first day, peak oxygen consumption ($\dot{V}O_2$) was measured. On the second and third days, normal (NS) or hypertonic (HS) saline trials were randomly conducted.

2.3. Peak $\dot{V}O_2$ test

Peak $\dot{V}O_2$ was determined during incremental cycling exercise at 60 rpm to volitional exhaustion on an electrically braked cycle ergometer (Model 232C, Combi, Tokyo, Japan). After a 5-min warm-up at 50 W, the workload was increased by 20 W every 2 min until the subject could not no longer maintain the cadence. Heart rate (HR) was continuously monitored using electrocardiography (Life Scope BSM-2401, NIHON KOHDEN, Tokyo, Japan). $\dot{V}O_2$ and minute ventilation were measured every 30 s with open-circuit auto O_2 and CO_2 analyzers and a hot-wire flowmeter (model AE300S, Minato Medical Science, Osaka, Japan).

2.4. Experimental protocols

Subjects were asked to refrain from consuming food and beverages containing caffeine or alcohol from 8 pm before the experimental day. They were provided food (400-kcal biscuits; carbohydrate 41 g, fat 22.2 g, protein 8.2 g, Calorie Mate, Otsuka Pharmaceutical, Tokushima, Japan) and 500 mL water, which were consumed at home 4 h before the experiment, and were allowed to drink water until arrival at the laboratory. In the laboratory, the subjects drank another 500 mL water, voided completely, and entered an environmental chamber set at ambient temperature (27 °C) and 30% relative humidity. A 20-gauge Teflon catheter (Terumo, Tokyo, Japan) was placed in the left forearm vein for infusion and blood sampling.

Body heating was conducted via heating of the skin surface, with or without core heating. To heat the skin surface, subjects wore a water-perfusion suit that covered the entire body surface except for the face, hands, calves, and feet. For core heating, subjects sat on a chair, and both lower legs were immersed in a water bath. The water was filled up to the level of the lower edge of the patella bone. The temperatures of the perfused water in the suit (T_{suit}) and water bath (T_{bath}) were initially set at 33 °C and 34.5 °C, respectively.

Normal (0.9% NaCl; NS) or hypertonic (3.0%; HS) saline was infused through the venous catheter at a rate of 0.05 mL/min·kg body weight for 120 min (Fig. 1). The first 20 min was defined as the baseline period. T_{suit} was increased to 36 °C at 20–35 min and to 39 °C at 35–50 min; T_{bath} remained at 34.5 °C. At 50–55 min, the subjects removed their legs from the bath, and T_{bath} was set at 42 °C. Then, the subjects placed their lower legs in the bath again, and T_{suit} was maintained at 39 °C at 55–70 min to accelerate core heating. While maintaining T_{bath} at 42 °C, the heating of the skin surface was repeated; T_{suit} was decreased to 33 °C at 70–90 min, followed by 36 °C and 39 °C at 90–105 and 105–120 min, respectively.

2.5. Measurements

The sensitivity to plasma osmolality was estimated by changes in fluid-regulating hormones (arginine vasopressin [AVP], plasma renin activity [PRA], and angiotensin II [AngII]). Plasma concentrations of heat shock protein 70 (HSP 70) and interleukin 6 (IL-6) were measured to determine if exercise training reduces thermal stress [12–14].

To constantly measure esophageal temperature (T_{es}), a thin copper-constantan thermocouple was inserted via the nose through a 5-Fr feeding tube; the thermocouple was fixed at the level of the left atrium (the length was estimated as one-fourth of the subject's height from the nostril).

Skin temperature was monitored with thermocouples placed on the forehead, chest, abdomen, back, and lateral sides of the upper arm, forearm, thigh, calf, hand, and foot. The mean skin temperature of the ten sites (T_{skin}) was calculated using the area weighting formula by Hardy and DuBois [15].

Blood flow in the chest skin was estimated using laser Doppler flowmetry (LDF; ALF 21, Advance, Tokyo, Japan). LDF is expressed as the percent change from the averaged value of the baseline period [16]. The rate of sweat on the chest was evaluated using dew hygrometry (POS-02, Skinos Giken, Nagoya, Japan). The LDF probe and ventilated sweat capsule were placed under the suit. HR was monitored using electrocardiography, and blood pressure in the left brachial artery was measured using oscillometry every 5 min (STBP-780, Colin, Komaki, Japan). Mean arterial pressure (MAP) was calculated as (systolic pressure − diastolic pressure) / 3 + diastolic pressure. Cutaneous vascular conductance (CVC) was calculated as LDF/MAP and is expressed as the percent change from the baseline value.

Thermal perception, thirst, fatigue, and sensation of skin dampness were assessed every 5 min using a visual analogue scale consisting of a 100-mm line on paper; the subjects drew a vertical line that intersected the scale based on their perception. Because the duration of the transition of T_{bath} from 34.5 °C to 42 °C was slightly different in each experiment, the assessment was not performed during the 55–65-min period. Thermal perception is considered as both thermal sensation and comfort [17–19]. Thermal sensation is described as the subjective evaluation of a conscious feeling (i.e., warm or cold). Thermal comfort is described as a state of mind of satisfaction with the surrounding environment. Thermal sensation and comfort were separately