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Abstract Hyperosmotic hypovolemia impairs vasoconstriction during sedentary cold exposure. The purpose of this study was to determine whether hypohydration alters thermoregulation and cardiovascular responses to exercise in cold air. On four occasions, eight males [35.1 (2.7) years, 175.5 (3.1) cm, 73.3 (2.6) kg, 57.2 (2.6) ml kg⁻¹ min⁻¹ maximal oxygen uptake (VO₂max), 19.6 (2.4)% fat] walked, in t-shirt, shorts, and shoes, at 50% V_{O₂}max, for 60 min in either a 4°C (Cold) or a 25°C (Temperate) environment in both hypohydrated state (HYPO, -4% body mass) and euhydrated state (EU). During exercise, cold stress, rectal temperature (T_{re}), mean weighted skin temperature, heart rate (HR), cardiac output (CO), and stroke volume (SV) were measured every 20 min. Mean weighted skin temperature values were not different between HYPO and EU but were lower (P < 0.05) in Cold versus Temperate trials. T_{re}, was not different (P > 0.05) between HYPO-Cold and EU--Cold. CO and SV were not different within hydration states and were not different between Cold and Temperate trials (P < 0.05). HR was not different between HYPO--Cold and EU--Cold. These data demonstrate that moderate intensity exercise in the cold while hypohydrated does not alter metabolic heat production, skin temperatures and heat loss, nor does it increase thermoregulatory and cardiovascular strain.

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Abstract Hyperosmotic hypovolemia impairs vasoconstriction during sedentary cold exposure. The purpose of this study was to determine whether hypohydration alters thermoregulation and cardiovascular responses to exercise in cold air. On four occasions, eight males [35.1 (2.7) years, 175.5 (3.1) cm, 73.3 (2.6) kg, 57.2 (2.6) ml kg⁻¹ min⁻¹ maximal oxygen uptake ($\dot{V}O_{2max}$), 19.6 (2.4)% fat] walked, in t-shirt, shorts, and shoes, at 50% $\dot{V}O_{2max}$ for 60 min in either a 4°C (Cold) or a 25°C (Temperate) environment in both hypohydrated state (HYPO, -4% body mass) and euhydrated state (EU). During exercise-cold stress, rectal temperature (T_{re}), mean weighted skin temperature, heart rate (HR), cardiac output (CO), and stroke volume (SV) were measured every 20 min. Mean weighted skin temperature values were not different between HYPO and EU but were lower ($P < 0.05$) in Cold versus Temperate trials. T_{re} was not different ($P > 0.05$) between HYPO-Cold and EU-Cold. CO and SV were not different within hydration states and were not different between Cold and Temperate trials ($P < 0.05$). HR was not different between HYPO-Cold and EU-Cold. These data demonstrate that moderate intensity exercise in the cold while hypohydrated does not alter metabolic heat production, skin temperatures and heat loss, nor does it increase thermoregulatory and cardiovascular strain.

Keywords Environment · Heat production · Hypovolemia · Vasoconstriction

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Introduction

Cold exposure can cause substantial fluid loss causing individuals to dehydrate by 2–5% of body mass (O'Brien et al. 1996). Reasons for this include cold-induced diuresis, reduced voluntary fluid intake, reduced thirst sensitivity, poor access to water, and sweating caused by a combination of exercise and winter clothing (Freund and Sawka 1996; O'Brien et al. 1998; Ramathan 1964; Wyant and Caron 1983). These fluid losses can cause either an isosmotic (cold-induced diuresis) or a hyperosmotic (sweat loss) hypohydration. Previously, O'Brien et al. (1998) have shown that hyperosmotic hypovolemia blunts vasoconstriction during sedentary cold exposure. Furthermore, Gonzalez-Alonso et al. (2000) found forearm and cutaneous vascular conductance to be 28–67% higher (not statistically significant) during short-duration, strenuous exercise in 8°C air when dehydrated by 4.2%. If similar changes were observed during moderate exercise intensities of longer duration in an environment with greater cooling potential, hypohydration could cause higher peripheral skin temperatures and greater heat loss to the environment, subsequently leading to lower core temperatures.

In temperate and hot climates, hypohydration increases cardiovascular and thermoregulatory strain and reduces performance (Gonzalez-Alonso et al. 1995; Montain and Coyle 1992; Rogers et al. 1964). This performance decrease might also have implications in cold climates. If hypohydration also increases cardiovascular strain during exercise-cold stress, individuals may become fatigued earlier and will either stop exercise or decrease their exercise intensity. If environmental conditions are such that large radiative and convective heat losses will occur (e.g., cold-windy or cold-wet), stopping exercise or significantly lowering the exercise intensity could lead to large declines in core temperature, increasing risk for hypothermia.

Given the scenario presented above, hypohydration is likely to occur in individuals such as athletes, laborers,

and military personnel who work and exercise for extended periods in cold environments at moderate exercise intensities. However, to our knowledge the impact of hydration state on thermoregulatory and cardiovascular responses during moderate exercise-cold stress has not been examined thoroughly. The purpose of this study was to determine whether hypohydration would alter thermoregulatory and cardiovascular responses during subsequent exercise-cold stress. We hypothesized that: (1) hyperosmotic hypohydration would impair the vasoconstrictor response during exercise-cold exposure resulting in higher skin temperatures; and (2) similar to responses in a hot environment, hyperosmotic hypohydration would increase cardiovascular strain during subsequent exercise in the cold.

Methods

Subjects

Eight, endurance-trained men, not heat- or cold-acclimated, volunteered to participate in this investigation. Physical characteristics [mean (SEM)] were: age 35.1 (2.7) years; height 175.5 (3.1) cm; body mass 73.3 (2.6) kg; maximal oxygen uptake ($\dot{V}O_{2\max}$) 57.2 (2.6) ml kg⁻¹ min⁻¹; percentage body fat 19.6 (2.4)%; body mass index 23.8 (0.9) kg m⁻². Each subject completed a written informed consent document and a medical history questionnaire after being informed of the purpose of the experiment and possible risks. The Committee on the Use of Human Subjects in Research at the University of New Hampshire approved all procedures. All experiments were carried out in accordance to state and federal guidelines.

Preliminary measures

$\dot{V}O_{2\max}$ was determined on a motor-driven treadmill using a continuous protocol as previously described (Costill and Fox 1969). The hydrostatic weighing technique described by Katch and Katch (1980) was utilized to determine body density. Body fat was calculated from the formula of Brozek et al. (1967). To correct hydrostatic weighing values for residual volume, vital capacity was measured via spirometry, and residual lung volume was estimated from these values (Wilmore 1980).

Experimental design

Subjects performed four experimental exercise-testing sessions. Each session occurred in one of two states of hydration, hypohydrated state (HYPO) or euhydrated state (EU), and in one of two

environmental conditions, 4°C, 74% relative humidity (Cold) or 25°C, 38.5% relative humidity (Temperate). On the day previous to each experimental exercise-testing session, subjects performed an exercise-induced dehydration protocol in 37°C air, to reduce body mass by ~3% (Table 1). In order to keep exercise on the day prior to the EU experimental trials consistent, subjects performed the dehydration protocol but matched sweat losses with water replacement during the protocol. On the following morning, subjects performed experimental trials of 60 min of walking exercise at ~50% $\dot{V}O_{2\max}$ when either EU or HYPO in a Cold or a Temperate environment. Subjects walked in t-shirts, shorts, and shoes for both the dehydration protocol and experimental exercise session. Subjects were asked to maintain similar diets during the 3 days before each experimental trial and to refrain from activity other than the dehydration protocol 24 h prior to experimental testing.

Exercise-induced dehydration

Upon arrival at the laboratory subjects provided a urine sample for determination of urine specific gravity (USG; Spartan Refractometer, model A 300 CL, Japan) and body mass (General Electric Systems, model GE510, Cape Coral, Fla.) was measured. A USG of 1.023 (0.006) (Armstrong et al. 1994) was used to verify that the subject was adequately hydrated. Subjects were then fitted with a heart rate (HR) monitor (Polar Accurex II, Polar CIC, Port Washington, N.Y.) and a flexible thermistor (Yellow Springs Instruments, series 401, Yellow Springs, Ohio) was inserted 10 cm beyond the anal sphincter to monitor rectal temperature (T_{re}). The mean ambient temperature and percent relative humidity in the environmental chamber (Harris Environmental Systems, Andover, Mass.) were 37 (0.1)°C and 65 (2.0)%, respectively. Airflow (6.1 m s⁻¹, 13.6 mph), generated by two fans, was directed at the subject to enhance evaporative sweat loss.

The dehydration protocol consisted of alternating treadmill walking [1.6 (0.1) m s⁻¹; 8.6 (0.5)% gradient; Quinton, Seattle, Wash.] and cycle ergometry [117 (9) W; Model 818E, Monark, Sweden] at a 25 min:5 min ratio (exercise:rest) for each modality to induce a ~3% change in body mass. The percent $\dot{V}O_{2\max}$ for the four dehydration trials ranged from 47–51%. Body mass was measured during each rest interval. Urine was collected throughout the dehydration stage and was included as part of the body mass loss. The mean exercise time for the dehydration protocol was 78 (6) min. After completion of the dehydration protocol subjects left the laboratory for the night. To induce an additional 1% loss in body mass overnight during HYPO treatments, subjects were instructed to consume only dry foods and refrain from fluid intake. Subjects were permitted to have typical intake of food and fluid overnight during EU treatments.

Experimental exercise testing

For all subjects the first exercise-testing session was performed in one of the HYPO states (HYPO-Temperate or HYPO-Cold). This was carried out to establish the amount of time to achieve a 4%

Table 1 Selected variables pre- and post-dehydration and pre- and post-exercise in cold or temperate environments [$n=8$; mean (SEM)]. EU-Cold Euhydrated-cold, HYPO-Cold hypohydrated-cold, EU-Temperate euhydrated-temperate, HYPO-Temperate hypohydrated-temperate, USG urine specific gravity

	HYPO-Cold	EU-Cold	HYPO-Temperate	EU-Temperate
Initial body mass (kg)	74.5 (1.7)	73.9 (1.8)	74.2 (1.9)	74.1 (1.8)
Pre-exercise body mass change (%) ^a	-4.1 (0.2)	0.5 (0.3)	-3.5 (0.3)	-0.2 (0.2)
Post-exercise body mass change (%) ^a	-4.7 (0.3)	0.0 (0.2)	-4.7 (0.4)	-1.1 (0.4)
Pre-dehydration USG	1.001 (0.003)	1.017 (0.004)	1.012 (0.003)	1.011 (0.005)
Pre-exercise USG	1.028 (0.017)*	1.018 (0.003)	1.030 (0.001)*	1.014 (0.003)

*Significant difference ($P<0.05$) from pre-exercise EU-Cold and EU-Temperate values

^aChange from pre-dehydration weight

body mass loss for each subject. This time was then held constant for the following three trials in order to control for the effect of the prior day's exercise on the experimental exercise trial. The remaining three trials were then performed at random. Upon arrival at the laboratory between 0600 and 0700 hours, subjects provided a urine sample for determination of USG and body mass was measured. Subjects were then fitted with a heart rate monitor, a flexible thermistor to monitor T_{re} , and skin thermistors (Yellow Springs Instruments, series 401) were placed on the upper arm, chest, upper thigh, and calf of the subject's left side for measurement of mean weighted skin temperatures (Ramanathan 1964). The subject then entered the environmental chamber where the conditions were set for either the Cold or Temperate testing session and immediately began the experimental exercise session. Subjects walked on a treadmill for 60 min at a fixed workload that would elicit 50% $\dot{V}O_{2max}$ determined when they were in a EU state [$1.6 (0.1) \text{ m s}^{-1}$; $8.6 (0.5)\%$ gradient]. Airflow was 6.1 m s^{-1} (13.6 mph), generated by two fans directed at the subject to increase the cold stress.

Physiological measures

During all dehydration bouts, the subject's heart rate and T_{re} were measured every 8 min. To ensure subject safety, if heart rates exceeded $180 \text{ beats min}^{-1}$ for 5 min and/or T_{re} was $>39.5^\circ\text{C}$ the dehydration protocol was terminated. Oxygen consumption ($\dot{V}O_2$, liters per minute) and respiratory exchange ratio (R) were measured once every 8 min during the dehydration protocol for a period of 5–8 min. Expired gas samples were analyzed using an on-line metabolic system (SensorMedics 2900; Yorba Linda, Calif.).

During experimental exercise testing, HR, blood pressure, T_{re} , mean weighted T_{sk} , cardiac output (CO), stroke volume (SV), and $\dot{V}O_2$ were measured pre-exercise (25°C , 38.5% relative humidity) and at minutes 20, 40, and 60 during exercise. CO was measured via the Collier CO_2 -rebreathing technique (Wilmore et al. 1982). SV was calculated as CO/HR . Metabolic heat production was calculated as described by Gagge and Gonzalez (1996).

Statistical analysis

Repeated measures analysis of variance (trial $\times$ time) was used to compare differences among trials. A Newman-Keuls post hoc analysis was employed to determine significant differences within and between conditions. The 0.05 level of significance was selected. All data are presented as means (SEM).

Results

Hydration state

The dehydration protocol and overnight fluid restriction achieved a -3.5% (range -3.1% to -4.8%) and -4.1% (range -3.1% to -5.2%) loss in body mass before the HYPO-Temperate and HYPO-Cold exercise sessions, respectively. In addition, pre-exercise USG was significantly greater in HYPO trials compared to EU trials (Table 1). During the dehydration protocol on the days prior to EU trials, sweat loss was matched with fluid intake in order to maintain a state of euhydration. Subjects drank 1,200 (0.1) ml and 1,300 (0.1) ml of H_2O for the EU-Cold and EU-Temperate sessions, respectively. Pre-dehydration to pre-exercise body mass changes before the EU-Cold and EU-Temperate exercise sessions were 0.5 (0.3)% and -0.2 (0.2)%, respectively. EU-Cold

and EU-Temperate pre-exercise USG values were not different from pre-dehydration values (Table 1).

Thermoregulatory responses

Cold trials (4°C air)

Metabolic heat production (watts per square meter) was similar between HYPO-Cold [317 (20)] and EU-Cold [306 (24)]. Mean skin temperature values were not different between EU and HYPO trials (Fig. 1A). T_{re} were not different ($P > 0.05$) at each corresponding time point between the HYPO-Cold and EU-Cold experimental trials (Fig. 1B). Also whole-body sweat rates (liters per hour) were not different between HYPO-Cold [0.40 (0.06)] and EU-Cold [0.41 (0.12)].

Temperate trials (25°C air)

Metabolic heat production was similar between HYPO-Temperate [300 (26)] and EU-Temperate [279 (21)] trials. Mean skin temperatures were the same between HYPO and EU trials during exercise in 25°C air

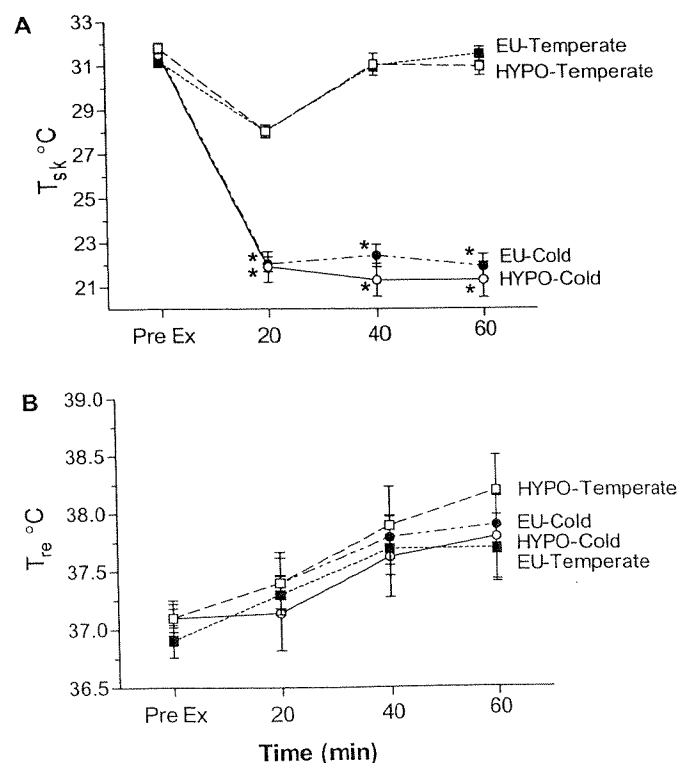


Fig. 1 Responses of mean weighted skin temperature (T_{sk}) (A) and rectal temperature (T_{re}) (B) as a function of exercise time during euhydrated-cold (EU-Cold; ●), hypohydrated-cold (HYPO-Cold; ○), euhydrated-temperate (EU-Temperate ■), and hypohydrated-temperate (HYPO-Temperate; □) trials. Values are means (SEM). *EU-Cold and HYPO-Cold significantly different from corresponding EU-Temperate and HYPO-Temperate time points, $P < 0.05$.

(Fig. 1A). T_{re} values were also not significantly different between trials in 25°C air although T_{re} was 0.6°C higher in HYPO at the completion of 60 min of exercise (Fig. 1B). Whole-body sweat rates were not different between HYPO-Temperate [0.90 (0.12)] and EU-Temperate [0.70 (0.24)].

Cardiovascular responses

Cold trials (4°C air)

SV during exercise ranged from 125–135 ml during exercise in cold air but there were no significant differences ($P > 0.05$) between HYPO-Cold and EU-Cold (Fig. 2A). HR values were also the same between hydration trials (Fig. 2B). Therefore, CO was not different between HYPO-Cold and EU-Cold (Fig. 2C).

Temperate trials (25°C air)

There were no significant differences between HYPO-Temperate and EU-Temperate for SV, although SV tended to be ~10 ml lower in the HYPO versus the EU trial at the end of exercise (Fig. 2A). HR was ~20 beats min^{-1} higher ($P < 0.05$) in the HYPO-Temperate trial versus EU-Temperate (Fig. 2B). Even with these differences CO was not statistically different between HYPO-Temperate and EU-Temperate trials (Fig. 2C).

Cold air (4°C) versus temperate air (27°C) trials

As expected, skin temperatures during exercise were lower during exercise in 4°C air versus 27°C. However, core temperature responses were not different during exercise in different ambient conditions (Fig. 1). Differences in cardiovascular responses were observed between 4°C and 27°C trials (Fig. 2). SV and CO in both the EU and HYPO conditions were significantly higher during exercise in 4°C air versus 27°C air.

Discussion

This experiment examined the effects of hypertonic hypohydration on thermoregulatory and cardiovascular changes during exercise-cold stress. Hypertonic, rather than isotonic, hypohydration was chosen because this type of dehydration is most often encountered in cold environments due to sweat loss. This type of dehydration has also been observed to cause changes in thermoregulatory effector responses during sedentary cold exposure (O'Brien et al. 1998). In addition to this, a 4% level of hypohydration was chosen because effects on thermoregulation in the cold have been observed at this level. While we did not measure plasma osmolality or changes in plasma or blood volume, previous studies

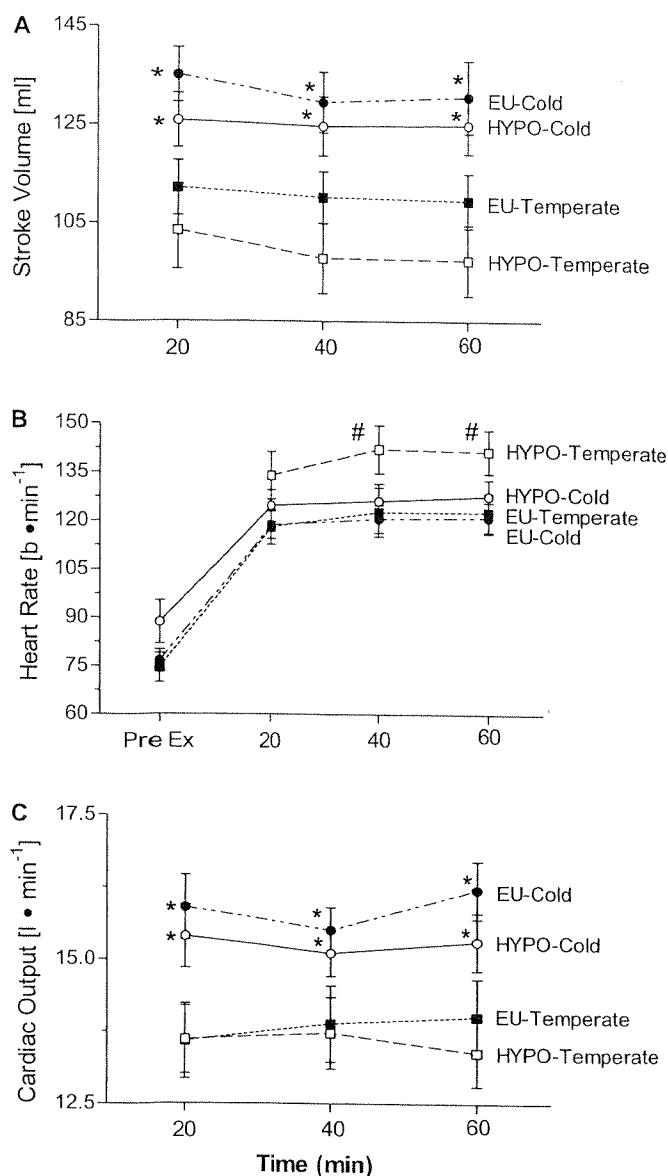


Fig. 2 Responses of stroke volume (A) heart rate (B), and cardiac output (C) as a function of exercise time during EU-Cold (●), HYPO-Cold (○), EU-Temperate (■), and HYPO-Temperate (□) trials. Values are means (SEM). *HYPO-Temperate and EU-Temperate significantly different from EU-Cold and HYPO-Cold, $P < 0.05$. #HYPO-Temperate significantly different at corresponding HYPO-Cold, EU-Temperate, and EU-Cold time points, $P < 0.05$.

that have employed similar dehydration protocols to achieve a 2–4% change in body mass in their subjects have reported that their subjects were in a state of hyperosmotic hypovolemia (Gonzalez-Alonso et al. 1995, 2000; Montain and Coyle 1992). Given the change in body mass and change in pre-exercise USG values achieved by the dehydration protocol, we believe a state of hyperosmotic hypovolemia was likely achieved in these subjects. Lastly, walking was chosen to simulate an exercise intensity (50% $\dot{V}O_{2\max}$) that can be sustained by many recreational athletes in a cold environment.

The primary finding of this study was that 4% hypohydration had no effect on thermoregulatory or cardiovascular responses during exercise in cold air. Our first hypothesis stated that, compared with euhydration, hypohydration would blunt peripheral vasoconstriction and, as a result, skin temperatures would be higher. This would create potential for greater heat loss to the environment and lead to lower core temperatures when in the cold. We based this hypothesis on the finding by O'Brien et al. (1998) who, using measures of skin and esophageal temperatures, observed impaired vasoconstriction due to hyperosmotic hypovolemia in subjects during sedentary cold exposure (7°C). Differences between our findings and those of O'Brien et al. (1998) may be due to the type of cold exposure. Subjects in the present study were exercising at 50% $\dot{V}O_{2\max}$ in a 4°C environment and generating metabolic heat via exercise, and not sitting quietly. Thus, any reductions in cold-induced vasomotor tone associated with hypohydration may have been over-ridden by metabolic heat production in the present study.

Our findings also contrast with those observed by Gonzalez-Alonso et al. (1995). They found increases (albeit not statistically significant) in cutaneous blood flow (38%), forearm vascular conductance (67%), cutaneous vascular conductance (28%), and mean skin temperature (0.6°C) when subjects were dehydrated by 4.2%, compared to euhydration. However, the changes observed in that study (Gonzalez-Alonso et al. 1995) may not be caused by hydration status per se, but may also be a function of the experimental design. Subjects in Gonzalez-Alonso et al. (1995) exercised for a minimum of 2 h before performing the experimental cold trials. Recently, Castellani et al. (1999, 2001) have reported that acute exercise increases peripheral heat loss and mean skin temperatures during subsequent cold exposure. Thus, the changes in blood flow indices in Gonzalez-Alonso et al. (2000) may be caused by the interaction of exercise and dehydration, whereas in the present study, prior exercise was not a confounding variable because the subjects dehydrated the previous day.

Concomitant with our finding that skin temperatures and metabolic heat productions were not affected by 4% hydration, we also did not find any differences in core temperature between the HYPO and EU trials during exercise in the cold. However, Gonzalez-Alonso et al. (2000) observed that esophageal temperatures were higher when subjects were dehydrated compared to EU. One reason may be that the exercise intensity employed in that study was higher (72% vs 50% $\dot{V}O_{2\max}$) and the ambient conditions were not as severe (8°C and 2 m s⁻¹ wind speed vs 4°C and 6.1 m s⁻¹). However, the rate of rise was the same between EU and HYPO trials in Gonzalez-Alonso et al. (2000) when taking into account the pre-exercise esophageal temperature value, similar to our findings. Thus hypohydration did not cause either increased or decreased core temperatures during exercise in the cold.

Cardiovascular responses were also not different between the HYPO and EU trials when exercising in the cold, i.e., we did not observe a lower SV and an elevated HR (increased cardiovascular strain) during the HYPO trial in the cold as has been previously observed in warm and hot environments (Gonzalez-Alonso et al. 1995; Montain and Coyle 1992). We had hypothesized that due to the hypohydration-mediated impairment of vasomotor control, cardiovascular responses during exercise in the cold would be more representative of hypohydration per se (decreased SV, increased HR) and not those typically observed during cold exposure (less cardiovascular strain due to increases in central volume). The mechanism for the observation that 4% hypohydration did not cause greater cardiovascular strain during exercise-cold stress is likely due to cold-induced vasoconstriction, which increased venous return to the heart, maintained central blood volume, and allowed a higher SV and RQ compensating for the 4% fluid loss. As shown in Fig. 2A and B, although the values were not significantly different, the mean values of HR were higher (8–9%) and SV was lower (9–10%) when subjects were dehydrated during exercise in the cold environment, compared with EU. These findings are similar to those of a previous study (Gonzalez-Alonso et al. 2000) that, moreover, found HR was lower (7–8%) and SV higher (11%) during exercise in the cold (8°C) compared with exercise in the heat (35°C) when 4–5% dehydrated. Despite similar levels of dehydration between the two studies, reasons for these similar findings are likely due to dissimilar mechanisms. Gonzalez-Alonso et al. (2000) attributed the elevated HR and lower SV during HYPO exercise in the heat to a greater skin blood flow than was observed in the cold; subjects in their study exercised at a greater exercise intensity (72% $\dot{V}O_{2\max}$) and in a hotter environment (35°C) than employed in the present study. This exercise intensity caused high metabolic heat productions, leading to greater heat storage and likely discriminated between fluid conditions and less so between environmental conditions. We employed an exercise intensity (50% $\dot{V}O_{2\max}$) that would be likely used by individuals who would be exercising in the cold for many hours and could not maintain high levels of cardiovascular strain for prolonged periods. It is possible that this exercise intensity in the present study did not discriminate between euhydration and hypohydration conditions in the cold, as it does in the heat.

Despite the findings that hypohydration does not increase thermoregulatory or cardiovascular strain, fluid intake during exercise in the cold is still important in order to maintain hydration. During prolonged or high-intensity exercise in the cold, heat production can still be greater than heat loss, leading to heat storage and subsequent increases in core temperature. This will be further exacerbated by hypohydration caused by cold associated decrease in sense of thirst and increased insensible water loss.

In summary, 4% hyperosmotic hypohydration does not increase thermoregulatory or cardiovascular strain

during moderate-intensity exercise in the cold. Endogenous metabolic heat production and peripheral heat loss were not altered by hypohydration and thus core temperature was similar between EU and HYPO states. Also, 4% hyperosmotic hypohydration did not increase HR and decrease SV during exercise-cold stress, responses typically observed during exercise in warm environments. Thus, moderate intensity exercise in the cold while HYPO does not incur the same physiological penalty that has been previously observed in a warm environment (i.e., greater cardiovascular strain and increased risk of hyperthermia).

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