Hypohydration effect on finger skin temperature and blood flow during cold-water finger immersion

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O'Brien, Catherine, and Scott J. Montain. Hypohydration effect on finger skin temperature and blood flow during cold-water finger immersion. J Appl Physiol 94: 598–603, 2003. First published October 11, 2002; 10.1152/japplphysiol.00678.2002.—This study was conducted to determine whether hypohydration (Hy) alters blood flow, skin temperature, or cold-induced vasodilation (CIVD) during peripheral cooling. Fourteen subjects sat in a thermoneutral environment (27°C) during 15-min warm-water (42°C) and 30-min cold-water (4°C) finger immersion (FI) while euhydrated (Eu) and, again, during Hy. Hy (−4% body weight) was induced before FI by exercise-heat exposure (38°C, 30% relative humidity) with no fluid replacement, whereas during Eu, fluid intake maintained body weight. Finger pad blood flow (as measured by laser-Doppler flux (LDF)) and nail bed (Tnb) pad (Tpad), and core (Tc) temperatures were measured. LDF decreased similarly during Eu and Hy (32 ± 10 and 33 ± 13% of peak during warm-water immersion). Mean Tnb and Tpad were similar between Eu (7.1 ± 1.0 and 11.5 ± 1.6°C) and Hy (7.4 ± 1.3 and 12.6 ± 2.1°C). CIVD parameters (e.g., nadir, onset time, apex) were similar between trials, except Tpad nadir was higher during Hy (10.4 ± 3.8°C) than during Eu (7.9 ± 1.6°C), which was attributed to higher Tc in six subjects during Hy (37.5 ± 0.2°C), compared with during Eu (37.1 ± 0.1°C). The results of this study provide no evidence that Hy alters finger blood flow, skin temperature, or CIVD during peripheral cooling.

Only a few studies have examined the effect of Hy on peripheral responses to cold, and the results are inconclusive. In one study, although an experimental group had lower mean finger temperatures during cold air exposure during Hy (−4.6% body weight), compared with during euhydration (Eu), finger temperatures were not fully restored after subsequent rehydration (to −1.9% of Eu body weight) (13). In another study, although the authors suggest that finger temperatures during cold exposure were lower with Hy (−3.5% body weight), the data actually demonstrate increased finger temperatures in the control group, with no change in finger temperatures in the experimental group (14). Our laboratory previously reported higher mean skin temperatures during a 2-h whole body cold-air (7°C) exposure when subjects were tested during Hy (−4% of body weight), compared with during Eu, but no differences in finger skin temperatures were observed (10). Whole body cold exposure is a strong stimulus for peripheral vasoconstriction, which could obscure any differences in digit temperatures that might have occurred with Hy in that study.

Cold-exposed extremities typically respond with a strong vasoconstriction, followed after several minutes by cold-induced vasodilation (CIVD), which increases digit temperature (8). This vasodilation persists for several minutes until vasoconstriction again reduces digit blood flow and skin temperature. Thereafter, another period of vasodilation may occur, producing a characteristic cyclic pattern of increasing and decreasing blood flow and skin temperature, known as the “hunting reflex” (8). Strong CIVD and higher mean finger temperatures may have a protective role against peripheral cold injury (19), and they have been associated with reduced pain during finger cooling (7). The pattern of response may be affected by physiological changes, such as whole body heating, which produces higher nadir and apex temperatures and earlier onset of CIVD (3); whole body cooling, during which CIVD is dramatically blunted (3); or peripheral acclimation, which generally produces higher nadir temperatures and earlier onset of CIVD (1). Previous studies on hydration reported only mean finger temperatures.
during cold exposure (13, 14), which does not indicate whether the pattern of CIVD was altered because of hydration state.

The effect of Hy on peripheral temperature responses during cold exposure, and whether Hy may increase susceptibility to cold injury, has not been demonstrated. The purpose of the present study was to compare finger blood flow and temperature response to local cold-water immersion in subjects under both Eu and Hy conditions, while controlling for some of the confounding factors evident from previous studies. A secondary aim of this study was to characterize the CIVD response during Eu and Hy. We hypothesized that Hy would not significantly alter finger blood flow, skin temperature, or CIVD parameters during peripheral cold exposure.

METHODS

Fourteen subjects (12 men, 2 women) completed this study, which was approved by the Institute Scientific and Human Use Review Committees. The subjects' characteristics were as follows: age, 22 ± 4 yr; height, 170 ± 10 cm; body surface area-to-mass ratio, 0.0249 ± 0.0019 m²/kg; body fat, 21 ± 7%; and peak oxygen uptake, 44.0 ± 5.1 ml/kg. Written, informed consent was obtained from each subject who volunteered to participate after being informed of the purpose, experimental procedures, and known risks of the study. Investigators adhered to Army Regulation 70-25 and US Army Medical Research and Materiel Command Regulation 70-25 on the Use of Volunteers in Research. The protocol used a repeated-measures design, such that each subject participated in both Eu and Hy tests. The order of these tests was randomized among subjects, with six subjects completing Eu first and eight completing Hy first. Two subjects who began the study did not complete the testing.

Test conditions. Each subject reported to the laboratory at 0700 on each of two occasions spaced a week apart. Subjects were instructed to refrain from alcohol intake for 24 h and caffeine and tobacco usage for 12 h before any testing. They were also instructed to drink two glasses of water before going to sleep the night before testing and to eat a standardized breakfast (typically a bagel and a granola bar) with two glasses of fluid on waking. The subject's body weight was measured on reporting to the laboratory, and this body weight was used to determine the target weight for each trial, i.e., maintaining the same body weight for Eu or 4% body weight loss for Hy. From 0700 to 1000, the subject completed three 50-min bouts of moderate-intensity exercise (10-min rest) in a hot climate (38°C, 30% relative humidity). The same exercise protocol was followed in both trials. During Eu, subjects drank sufficient volume of a flavored, noncaloric, salted (−0.04% NaCl) beverage to replace fluid loss due to exercise, whereas during Hy, fluid loss was not replaced. In the event that fluid loss during exercise was less than the 4% target weight loss for Hy, passive heat exposure in a dry sauna (−65°C) was used to elicit additional fluid loss. Seven subjects completed one or two 15-min passive heat exposures during Hy. Core temperature and heart rate were monitored during exercise-heat stress and passive heat exposures for safety. Volunteers were temporarily removed from the hot climates if their temperature exceeded 39.0°C or heart rate exceeded 90% of age-predicted maximum. After achieving the desired hydration level, the subjects showered, ate a standardized meal (300–400 kcal) (2 oz turkey on 2 pieces of bread, 100 ml fruit juice, and a single Popsicle) at ~1100, and then rested in a temperate climate (25°C) until completing the cold-water finger immersion test at ~1300. This recovery period was intended to restore basal core temperature and allow equilibration of body fluid spaces. During the rest period, body weight was periodically measured, and fluid intake was adjusted to maintain the target body weight. Typically, fluid intake was minimal during Hy, whereas during Eu subjects drank sufficient fluid to compensate for both sweat loss during exercise and urine loss during the rest period. After completing the CIVD test during Hy, subjects were rehydrated with a sports drink.

Cold-water finger immersion. Ambient conditions during finger immersion were designed to be thermoneutral (27°C, 50% relative humidity). Subjects wore standardized clothing consisting of shorts, T-shirt, shoes, and socks. They lay semisupine and rested quietly during the experiments. Thermocouples were attached to four skin sites (calf, thigh, chest, and forearm) for calculation of mean weighted skin temperature. A laser-Doppler probe (MicroFlo DSP, Oxford Optronics, Oxford, UK) with integrated thermistor was attached by using double sticky tape to the pad of the middle finger of the immersion (dominant) hand for measurement of skin blood flow and pad temperature. A wire thermocouple was attached with tape (−1 cm²) along the nail bed of the same finger for measurement of nail bed skin temperature. A finger blood pressure cuff (Finapres model 2300, Ohmeda) was attached to the index finger of the contralateral hand. Both hands were positioned at the approximate level of the heart. A harness was placed around the volunteer's chest for recording heart rate with a heart rate watch (Polar NV, Polar Electro Oy, Kempele, Finland).

Data collection began as the subject immersed the middle finger to the middle phalanx in warm (42°C) water. This temperature was chosen to abolish vasoconstriction (11) and ensure a standardized finger temperature for all volunteers on all tests before cold-water finger immersion. After 15 min in warm water, the subject transferred the finger to a cold-water (4°C) bath for 30 min. At the end of the immersion period, the volunteer withdrew the finger, instrumentation was removed, and a blood sample was obtained by venipuncture for measurement of hemoglobin, hematocrit, and plasma osmolality. Temperatures, skin blood flow, blood pressure, and heart rate were recorded every 6 s.

Measurements and statistics. Mean skin temperature was calculated by using the formula 0.3(temperature + triceps) + 0.2(temperature + calf) (12). Mean arterial pressure (MAP) was calculated as ½(systolic − diastolic) + diastolic pressure. Cutaneous vascular conductance (CVC; in mV/mmHg) was calculated as the ratio of laser-Doppler flux (LDF; in mV) to MAP (in mmHg) (11). Both LDF and CVC are expressed in absolute values and as the percent change from the peak value during warm-water immersion, after smoothing of the data with a 10-point running average to reduce variability. Percent change in blood volume was predicted from changes in hemoglobin (4).

The impact of Hy was assessed with repeated-measures ANOVA. Mean LDF, CVC, and nail bed and pad temperatures are presented for the last 25 min of cold-water immersion, which removes the initial rapid fall in blood flow and finger temperature on cold-water immersion and allows comparison to previous studies. To examine CIVD, finger temperature data were smoothed by using a three-point running average to reduce noise, and a change in temperature of at least 0.5°C was chosen to represent an occurrence of CIVD. CIVD parameters include temperature at the first nadir during cold-water immersion, time to reach that temperature, temperature at the next apex, and the time to reach
that temperature. Significant difference was set at $P < 0.05$.
Data are presented as means ± SD.

RESULTS

During Eu, despite efforts to fully replace fluid losses due to exercise, 13 subjects did not restore their initial body weight, and Eu body weight decreased 0.8 ± 0.8% ($P < 0.05$) from 76.0 ± 14.4 to 75.4 ± 14.6 kg by the time of the CIVD test. During Hy, body weight decreased 4.0 ± 0.7% ($P < 0.05$) from 76.3 ± 14.6 to 73.3 ± 14.1 kg at the time of the CIVD test. Plasma osmolality, hemoglobin, and hematocrit were all higher ($P < 0.05$) during Hy (298 ± 5 mosmol/kgH$_2$O, 154 ± 14 g/l, and 45.1 ± 3.7%, respectively) than during Eu (284 ± 6 mosmol/kgH$_2$O, 149 ± 10 g/l, and 43.4 ± 3.0%, respectively). Blood volume decreased by 3.4 ± 4.7% from Eu to Hy. Heart rate was higher ($P < 0.05$) during Hy (81 ± 12 beats/min) than during Eu (73 ± 10 beats/min), but it did not change with cold-water immersion. MAP was higher during cold-water immersion (94 ± 12 mmHg) compared with warm-water immersion (90 ± 10 mmHg), but it did not differ between the two trials. Temperature and blood flow data are shown in Table 1. Core temperature was higher ($P < 0.05$) during Hy than during Eu. There was no difference between trials for peak LDF (Eu: 264.0 ± 92.9 mV; Hy: 248.5 ± 80.9 mV) or peak CVC (Eu: 3.0 ± 1.0 mV/mmHg; Hy: 2.9 ± 1.1 mV/mmHg) during warm-water immersion or in mean percent reduction during cold-water immersion (see Table 1). Minimum values during cold-water immersion, expressed as percentage of warm-water peak values, were similar between trials for LDF (Eu: 6 ± 4%; Hy: 6 ± 7%) and CVC (Eu: 5 ± 3%; Hy: 6 ± 7%), as were maximum values for LDF (Eu: 67 ± 14%; Hy: 67 ± 20%) and CVC (Eu: 67 ± 13%; Hy: 64 ± 16%). All subjects exhibited CIVD, and the overall difference between lowest and highest temperatures achieved during cold-water immersion did not differ between Eu and Hy in either the nail bed (3.7 ± 0.9 and 4.0 ± 1.4°C, respectively) or pad (6.4 ± 1.7 and 7.4 ± 1.9°C, respectively). Finger pad nadir temperature was higher ($P < 0.05$) in Hy compared with Eu. No other CIVD parameters differed between trials, including nail bed nadir temperature, onset time of CIVD, apex temperatures, or apex time.

Because core temperature influences finger temperature during cold exposure (3), the data were further examined by comparing the responses of six subjects whose core temperatures did not differ (±0.2°C, $P > 0.05$) between trials with those of six subjects whose core temperatures were higher (≥0.3°C, $P < 0.05$) in Hy compared with Eu. Two subjects were missing core temperature data on one trial; therefore, they were not included in this portion of the data analysis. The data for these groups are shown in Table 1. No differences were observed in any variables for the group whose core temperatures were similar in the two trials. The group with higher core temperatures in Hy had higher ($P < 0.05$) nail bed and pad temperatures. In the nail bed, these higher temperatures were associated with higher ($P < 0.05$) apex temperature, whereas in the pad, the higher temperatures were associated with higher ($P < 0.05$) nadir temperatures. Figure 1 shows finger temperatures and CVC averaged for the six subjects in each of these groups.

DISCUSSION

This study examined finger blood flow, skin temperature, and CIVD response during peripheral cold exposure in subjects who completed both Eu and Hy trials. The results provide no evidence that Hy reduces peripheral blood flow or skin temperature during cold-water finger immersion, nor was the pattern of the

| Table 1. Temperatures and blood flow during cold-water finger immersion |
|--------------------------|--------------------------|--------------------------|
|                          | Eu (n = 14)              | Similar Core Temperature (n = 6) | Different Core Temperature (n = 6) |
|                          | Eu                      | Hy                       | Eu                      | Hy                       |
| Core temperature, °C     | 37.0 ± 0.3†             | 37.2 ± 0.3†             | 36.9 ± 0.3             | 37.0 ± 0.3             | 37.1 ± 0.1†             | 37.5 ± 0.2†             |
| Mean skin temperature, °C| 34.3 ± 0.7              | 34.5 ± 0.8              | 34.0 ± 0.7             | 34.0 ± 0.9             | 34.7 ± 0.4             | 35.1 ± 0.3             |
| Finger nail bed          |                         |                         |                         |                         |                         |                         |
| Nadir temperature, °C    | 5.0 ± 0.7               | 6.0 ± 0.8               | 4.7 ± 0.3              | 5.1 ± 1.3              | 5.2 ± 0.8              | 6.8 ± 2.0              |
| Onset of CIVD, min       | 4.3 ± 0.8               | 3.6 ± 1.7               | 4.6 ± 0.6              | 4.3 ± 2.0              | 4.3 ± 0.9              | 2.9 ± 1.4              |
| Apex temperature, °C     | 8.3 ± 0.8               | 8.7 ± 1.3               | 8.0 ± 0.7              | 7.8 ± 1.2              | 8.2 ± 0.6†             | 9.6 ± 1.1†             |
| Time of apex temperature, min | 9.5 ± 2.5             | 7.6 ± 3.9               | 9.0 ± 0.8              | 9.1 ± 4.6              | 9.9 ± 3.8              | 5.1 ± 2.6              |
| Mean finger temperature, °C | 7.1 ± 1.0             | 7.4 ± 1.3               | 6.8 ± 0.8              | 6.2 ± 0.6              | 7.0 ± 0.9†             | 8.3 ± 1.0†             |
| Finger pad               |                         |                         |                         |                         |                         |                         |
| Nadir temperature, °C    | 7.9 ± 1.6†             | 10.4 ± 3.8†             | 7.3 ± 1.0              | 8.6 ± 4.1              | 8.2 ± 0.5†             | 12.0 ± 2.9†             |
| Onset of CIVD, min       | 5.3 ± 0.9               | 5.2 ± 1.2               | 5.1 ± 0.6              | 5.4 ± 0.8              | 5.7 ± 1.0              | 5.2 ± 1.7              |
| Apex temperature, °C     | 13.7 ± 1.7             | 15.4 ± 2.2              | 13.7 ± 1.3             | 14.3 ± 2.7             | 13.7 ± 1.6             | 13.9 ± 1.5             |
| Time of apex temperature, min | 10.7 ± 2.2             | 9.6 ± 1.4               | 9.3 ± 0.9              | 9.9 ± 0.9              | 11.9 ± 2.5             | 9.2 ± 1.0              |
| Mean finger temperature, °C | 11.5 ± 1.6             | 12.6 ± 2.1              | 11.3 ± 1.8             | 10.9 ± 1.5             | 11.5 ± 0.9†             | 13.5 ± 1.4†             |
| Laser-Doppler flux, %warm-water peak | 32 ± 10             | 33 ± 13                  | 32 ± 11                 | 30 ± 14                 | 31 ± 11                 | 38 ± 13                 |
| Cutaneous vascular conductance, %warm-water peak | 30 ± 9             | 32 ± 12                  | 30 ± 10                 | 27 ± 12                 | 28 ± 9                  | 37 ± 11                 |

Values are means ± SD; n, no. of subjects. CIVD, cold-induced vasodilation; Eu, euhydration; Hy, hypohydration. *Mean during last 25-min cold-water immersion. †Statistical difference between Eu and Hy, $P < 0.05$. 

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CIVD response altered by hydration state. These results contrast with citations suggesting that Hy increases risk of peripheral cold injury (6, 18), a claim that remains unsubstantiated. Only two previous studies reported blunted finger temperatures during peripheral cold exposure in subjects during Hy (13, 14), but these data are inconclusive because the study designs did not provide adequate controls.

Roberts and Berberich (13) examined finger temperatures during cold-air (0°C) exposure of the hands in subjects during Eu, Hy (−4.6% body weight over 2.5 days through exercise and limited fluid intake), and rehydration (−1.9% of Eu body weight). These responses were compared with a control group that was tested on the same three occasions but remained in the Eu condition. Although during Hy the subjects had lower finger temperatures compared with during Eu, their finger temperatures remained lower on rehydration (13). Furthermore, the groups were dissimilar at the start, with the experimental group maintaining warmer finger temperatures during cold exposure than the control group on the first trial when all subjects were tested during Eu (13). Thus, in the remaining trials, finger temperatures in both groups were similar, despite differences in hydration state.

In another study, Roberts et al. (14) examined finger temperatures during hand exposure to cold air (0°C) in 18 subjects who exercised to lose ~2% body weight over 3 days and then lived in a cold environment (−23°C) for 5 days while receiving either normal fluid intake (3.0 l/day, n = 9) to restore body weight or low fluid intake (1.5 l/day, n = 9) to further decrease to ~3.5% body weight. At the end of the week, finger temperatures during cold exposure were higher in the normal fluid intake group but were unchanged in the low-water group (14). These results suggest a change in the Eu group, rather than demonstrating blunted finger temperatures due to Hy.

The mechanism commonly suggested for Hy to reduce finger temperature during cold exposure is that Hy causes decreased plasma volume, resulting in reduced peripheral blood flow and increased blood viscosity (6). However, plasma volume is typically well maintained during exercise dehydration (15). In the present study, where Hy was induced acutely, blood volume was estimated to decrease by ~3% during Hy, as calculated from blood samples drawn only at the time of the finger immersion test on both trials. Ideally, plasma volume changes would have been calculated from a baseline sample before exercise on each trial to account for any variation in hemoconcentration due to the first trial or other individual variability, but unfortunately we did not make that measurement. In the study of Roberts et al. (14), Tappan et al. (16) demonstrated an increase in plasma volume in Marines after they had lived and worked in a cold environment for several days with adequate fluid intake (3.0 l/day). If such an increase in plasma volume occurred in the control subjects of Roberts et al. (14), perhaps this allowed greater blood flow to the fingers that would not be observed in the Hy group. Unfortunately, no blood measurements were presented in that study to indicate whether any change in plasma volume occurred. Our laboratory’s previous study (10) also examined the effect on finger temperature response to cold during a diuretic-induced Hy, which causes a large decrease in plasma volume (~18%), but no differences in finger temperatures between trials were evident in that study. However, because whole body cold stress blunts CIVD (3), subtle changes in finger temperature response may have been masked. To date, no study has conclusively demonstrated an effect of Hy on peripheral temperature response to cold.
The present study was designed to control many of the variables that confound blood flow or temperature measurements. All subjects completed both trials, and each trial consisted of the same amount of exercise, so that the only variable between trials was hydration status. Testing began at the same time of day, and subjects ate the same breakfast and lunch on each trial. During the finger immersion test, subject posture was controlled by having subjects lie in a semisupine position on a gurney, with the hands supported at the approximate level of the heart. The subjects rested quietly in this posture for \( \sim 30 \) min before cold-water immersion. Finger immersion tests were conducted in a thermoneutral environment, and finger temperature was standardized by immersion in warm water before the cold-water immersion.

Enander (5) noted wide variability in baseline finger temperature despite a 60-min equilibration period at room temperature, and baseline finger temperature was negatively correlated to the rate of finger cooling during cold-air exposure. To avoid the confounding effects of such variability, we sought to standardize initial finger temperature by using warm-water immersion. Although this method increased the heat content of the finger, this was standardized across trials, and the heat gain during warm-water immersion would quickly be eliminated with immersion in cold water. The warm-water immersion had the additional advantage of providing a standard condition against which the relative change in blood flow during cold-water immersion could be compared. In the forearm, maximal vasodilation has been demonstrated to occur at a local skin temperature of 42°C (17); therefore, we anticipated that a similar blood flow response would occur during finger immersion in water at this temperature. Peak blood flow typically occurred within the first 5 min of warm-water immersion (Eu: 5.5 ± 3.7 min; Hy: 4.1 ± 2.7 min). Perhaps another method of standardizing initial finger temperature would be more appropriate, such as using a warm-water temperature to elicit maximal blood flow, followed by thermoneutral air exposure so that finger temperature and blood flow would reflect actual vasomotor tone. Further investigation is required to determine whether such a method would produce reproducible initial finger temperatures.

The inability to obtain absolute skin blood flow from laser-Doppler instruments makes it difficult to compare data across studies. Finger skin temperature has the advantage of reflecting changes in blood flow, while allowing comparison between studies that use the same measurement methods and locations. Skin temperature also has less variability; therefore, differences may be detected more easily. Furthermore, skin temperature may be more relevant for evaluating risk of cold injury, such as frostbite. Although skin temperature measurement methods are accurate and reliable, the type of instrumentation affects the response time. For example, in the present study, nail bed temperature was measured by using a type T (24 gauge) thermocouple wire, which had a very short time constant of \(< 2 \) s (the time required to reach 63.2% of an instantaneous temperature change). In contrast, pad temperature was measured by a thermistor (10 kΩ, negative temperature coefficient) embedded in an acetyl resin laser-Doppler probe (≈1.8-cm² surface coverage and \( \sim 0.7 \)-cm thickness). Although the thermistor itself has a time constant of \(< 1 \) s, we measured a time constant of \( \sim 40 \) s when moving the probe from the warm water to cold water, because of the heat storage of the acetyl resin. Once the resin has equilibrated at the temperature of the cold water, this effect would be minimized; however, a somewhat slower response time is observed with finger pad temperature, as can be seen in Fig. 1. The higher temperatures observed in the finger pad reflect the greater vascularity of this area, compared with the nail bed.

Although the 2-h recovery between exercise and the finger immersion test was intended to allow recovery of body temperatures, this was insufficient for one-half of the subjects, resulting in elevated core temperatures during Hy compared with during Eu. The subjects whose core temperatures were higher during Hy were those with lower body surface area-to-mass ratio (0.0236 ± 0.0013 m²/kg), thus greater heat storage, compared with those whose core temperature was similar on both trials (0.0259 ± 0.0018 m²/kg). Core temperature increased more during exercise during Hy compared with during Eu; therefore, the subjects with greater heat storage would require longer to return to normal core temperature. Because core temperature is known to influence CIVD parameters (3), any finger blood flow or temperature differences observed between Eu and Hy might be due to core temperature differences, rather than to Hy. In subjects whose core temperature was not different between trials, no change in blood flow or finger temperatures were observed with Hy (Fig. 1A), whereas elevated core temperature during the Hy trial did have an effect (Fig. 1B).

This study was designed to examine the effects of moderate Hy on finger skin blood flow and temperature responses to cold exposure. The degree of Hy induced in this study was similar to that commonly experienced by soldiers during cold-weather training exercises (9). The results of this study do not provide any evidence that Hy blunts finger temperatures during peripheral cold exposure. The possibility cannot be ruled out that a greater decrease in plasma volume may affect peripheral blood flow during cold exposure, such as would be induced by dehydration from diuretics or by the cold-induced diuresis that typically occurs with whole body cold exposure. Nevertheless, the findings of this study do not support the suggestion that Hy increases susceptibility to peripheral cold injury.

The views, opinions and/or findings contained in this publication are those of the author(s) and should not be construed as an official Department of the Army position, policy, or decision unless so designated by other documentation. The investigators have adhered to the policies for the protection of human subjects as prescribed in Army Regulation 70-25, and the research was conducted in adherence with the provisions of 45 CFR Part 46.
REFERENCES