Skin temperature modifies the impact of hypohydration on aerobic performance

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Kenefick RW, Cheuvront SN, Palombo LJ, Ely BR, Sawka MN.
Skin temperature modifies the impact of hypohydration on aerobic
performance. *J Appl Physiol* 109: 79–86, 2010. First published April
8, 2010; doi:10.1152/japplphysiol.00135.2010.—This study deter-
mined the effects of hypohydration on aerobic performance in com-
pensable [evaporative cooling requirement (E_{req}) < maximal evapo-
rative cooling (E_{max})] conditions of 10°C [7°C wet bulb globe
temperature (WBGT)], 20°C (16°C WBGT), 30°C (22°C WBGT),
and 40°C (27°C WBGT) ambient temperature (T_a). Our hypothesis
was that 4% hypohydration would impair aerobic performance to a
greater extent with increasing heat stress. Thirty-two men [22 ± 4 yr
old, 45 ± 8 ml·kg^{-1}·min^{-1} peak O_2 uptake ($\dot{V}O_{2peak}$)] were divided
into four matched cohorts ($n = 8$) and tested at one of four T_a in
euhydrated (EU) and hypohydrated (HYPO, −4% body mass) con-
ditions. Subjects completed 30 min of preload exercise (cycle ergome-
ter, 50% $\dot{V}O_{2peak}$) followed by a 15 min self-paced time trial. Time-
trial performance (total work, change from EU) was −3% ($P = 0.1$),
−5% ($P = 0.06$), −12% ($P < 0.05$), and −23% ($P < 0.05$) in 10°C,
20°C, 30°C, and 40°C T_a, respectively. During preload exercise, skin
temperature (T_{sk}) increased by ~4°C per 10°C T_a, while core (rectal)
temperature (T_{re}) values were similar within EU and HYPO condi-
tions across all T_a. A significant relationship ($P < 0.05$, $r = 0.61$) was
found between T_{sk} and the percent decrement in time-trial perfor-
mance. During preload exercise, hypohydration generally blunted the
increases in cardiac output and blood pressure while reducing blood
volume over time in 30°C and 40°C T_a. Our conclusions are as
follows: *1*) hypohydration degrades aerobic performance to a greater
extent with increasing heat stress; *2*) when T_{sk} is >29°C, 4% hypo-
hydration degrades aerobic performance by ~1.6% for each addi-
tional 1°C T_{sk}; and *3*) cardiovascular strain from high skin blood flow
requirements combined with blood volume reductions induced by
hypohydration is an important contributor to impaired performance.

dehydration; total work; graded ambient temperature; cutaneous blood
flow; mean arterial pressure

HYPOHYDRATION (>2% body mass) degrades aerobic perfor-
mance in temperate and warm-hot conditions (34, 37); how-
ever, there is little knowledge of the relative impact of different
environmental conditions on aerobic performance at a given
level of hypohydration. Cheuvront and colleagues (3) demon-
strated that 3% hypohydration did not alter aerobic perfor-
mance (time trial) in cold (2°C) conditions but reduced aerobic
performance in temperate (20°C) conditions. Several reviews
speculate (6, 35) that hypohydration might degrade aerobic
performance more in warm-hot than temperate conditions. If
this is true, then it is also possible that hypohydration might
degrade aerobic performance more in hot than warm condi-
tions. While this is plausible, the impact of hypohydration on

aerobic exercise performance along a continuum of air tem-
peratures has not been experimentally evaluated.

Exposure to warm environments (10) and the wearing of
protective clothing (5, 12) will elevate skin temperature (T_{sk}).
In euhydrated (EU) subjects, T_{sk} elevations from 31°C to 36°C
(with no change in core temperature) degrade aerobic perfor-
mance (8). Impaired aerobic performance was likely caused by
the redistribution of blood from central to peripheral (skin)
circulation (32), resulting from elevated skin blood flow/
volume (28, 38). The preservation of high skin blood flow/
volume challenges the ability of the cardiovascular system to
sustain the required cardiac output (CO) (28, 33). Hypohydra-
tion also accentuates cardiovascular strain by reducing blood
volume and alters thermoregulation to induce greater hyper-
thermia (14, 26–28). Together, the combined stressors of
hypohydration and high T_{sk} can limit the ability of the cardio-
vascular system to support aerobic metabolism (14–16, 27,
36), which would result in a higher fractional utilization of
maximal O_2 uptake ($\dot{V}O_2$) for any given power output (1, 30):
the greater the heat stress [air temperature (T_a), ratio of re-
quired evaporative cooling to maximum evaporative cooling
(E_{req}/E_{max})], the higher the expected skin blood flow response
and, consequently, the greater the possible impact of hypohy-
dration on aerobic exercise performance.

This study determined the effects of hypohydration (~4%
body mass loss) on aerobic performance in cool [10°C T_a, 0.20
E_{req}/E_{max}, 7°C wet bulb globe temperature (WBGT)], temper-
ate (20°C T_a, 0.45 E_{req}/E_{max}, 16°C WBGT), warm (30°C T_a,
0.54 E_{req}/E_{max}, 22°C WBGT), and hot (40°C T_a, 0.66 $E_{req}/$
E_{max}, 27°C WBGT) conditions. We hypothesized that 4%
hypohydration would impair aerobic exercise performance
more with increasing heat stress. We employed compensable
environmental conditions to elevate T_{sk} in a graded manner
while minimizing core temperature differences between envi-
ronments (9, 35).

METHODS

Subjects

Thirty-two men (22 ± 4.2 yr old, 1.80 ± 0.045 m height, 85.4 ±
10.84 kg body wt) volunteered to participate in the study, which was
approved by appropriate institutional review boards. Before partici-
pation, each subject attended briefings informing them of the purpose
of the experiment and possible risks and completed a written informed
consent document. Investigators adhered to policies for protection of
human subjects as prescribed in US Army Regulations 70-25 and US
Army Medical Research and Materiel Command Regulation 70-25.
The research was conducted in adherence with the provisions of Code
45 of Federal Regulations Part 46.

Preliminary Testing and Familiarization

Two weeks of preliminary testing and familiarization preceded the
experimental trials. On the initial day of preliminary testing, an

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electronic scale (model WSI-600, Mettler Toledo, Toledo, OH) was used to determine the body mass of each subject, and peak $\dot{V}O_2$ ($\dot{V}O_{2peak}$) was measured. $\dot{V}O_{2peak}$ and peak power (in W) were measured using an incremental protocol on an electronically braked cycle ergometer (Excalibur Sport, Lode, Groningen, The Netherlands) and a computer-based metabolic system with continuous gas exchange measurements (Parvo Medics, Sandy, UT). The cycle ergometer was used in the hyperbolic (pedal rate-independent) mode for $\dot{V}O_{2peak}$ testing while the subjects maintained a constant cadence of 60 ± 5 rpm to exhaustion. Briefly, the subjects began exercise at 40 W, and power output increased by 20 W every minute until they reached volitional exhaustion. During $\dot{V}O_{2peak}$ testing, heart rate (HR; Polar a_3, Polar Electro, Polar Accurex II, Polar Instruments, Woodbury, NY), $\dot{V}O_2$, CO_2 production, and minute ventilation were measured continuously.

Subjects wore T-shirts and shorts during preliminary testing, familiarization sessions, and all experimental testing. During the 2 wk of preliminary testing, subjects performed four familiarization sessions to reduce training and learning effects. Each familiarization session took place in a 22°C, 20–30% relative humidity environment and consisted of 30 min of steady-state preload (cycle ergometer exercise at 50% $\dot{V}O_{2peak}$) followed by a short rest break and then a 15-min aerobic performance time trial. Gas exchange data measured during the first and second steady-state familiarization sessions were used to confirm the workload needed to elicit 50% $\dot{V}O_{2peak}$.

For 5 consecutive days during the 2nd wk of familiarization, the subjects consumed 2 liters of sports drink after 1800. On each subsequent morning, the subjects provided a first morning urine sample, and a blood sample (<5 ml) was taken to measure plasma osmolality (P_{osmol}). In addition, nude body mass was measured before breakfast and after voiding. These measures of body mass, P_{osmol}, and urine specific gravity (USG) were then used to provide feedback to the subjects regarding their hydration state to help ensure that they would be well hydrated on the day of experimental testing. On the day of testing, subjects were considered euhydrated (EU) with a combination of any two of the following: USG <1.02, nude body mass within 1% of the 5-day average, or P_{osmol} <290 mmol/kgH$_2$O (34).

After the familiarization session, the 32 subjects were divided into 4 cohorts ($n = 8$) matched for $\dot{V}O_{2peak}$ and body mass (Table 1). Each cohort then performed experimental testing in one of four environments (10°C, 20°C, 30°C, and 40°C T_a) while in an EU, as well as a hypohydrated (HYPO, −4% body mass), state. EU and HYPO trials were randomly assigned and were separated by 1 wk. Given that familiarization and testing for each subject in all four environments in EU and HYPO states would take ~10 wk, we were concerned with changes in acclimatization status, fitness, and general health. In our experience, significant subject attrition was also likely to occur. Therefore, we elected to employ a valid mixed-model design, whereby subjects in each of four independent groups performed both EU and HYPO trials in a single environmental condition. This mixed-model design enabled each subject to complete familiarization and testing in 4 wk. Descriptive characteristics of each of the four subject cohorts are presented in Table 1.

Experimental Testing

Hypohydration and heat exposure. On the morning of each experimental testing trial, nude body mass, USG, and P_{osmol} were measured for comparison against the individual 5-day averages of the preceding week. Subjects consumed a standardized breakfast of 552 kcal (16 g fat, 94 g carbohydrate, and 8 g protein) and 250 ml of water. They were then instrumented for measurements of HR and rectal temperature (T_{re}) before entering the environmental chamber set at 50°C, ~20% relative humidity, and 1.6 m/s airflow. T_{re} was obtained from a telemetric temperature sensor (VitalSense Jonah Ingestible Capsule, Minimitter, Bend, OR), representative of true T_{re}, inserted 8–10 cm (length of gloved index finger) beyond the anal sphincter. This approach used simultaneously against a conventional rectal probe yields excellent agreement, with differences ≤0.05°C, and has been used in prior investigations (8, 23).

During the EU and HYPO sessions, subjects walked on a treadmill at 3 mph at 3.5% grade for 30 min, followed by 30 min of seated rest. This exercise-rest cycle continued for 3 h. The purpose of light walking exercise was to increase core temperature and initiate sweating, so that a 4% reduction in body mass could be achieved before experimental testing, a model that allows examination of how hypohydration impacts performance in the absence of other confounding factors (35). Throughout the heat exposure, if T_{re} reached 39.5°C, walking was discontinued and the subjects sat in the chamber for the remaining duration of that 30-min walking phase. Sweat loss volume was determined from changes in body mass measured every 30 min. In the EU trials, sweat volume and body mass losses were considered equivalent, so the subjects drank 1 ml of 0.05% NaCl-water solution to replace every 1 g of mass that was lost. After heat exposure, the subjects were allowed a 90-min break, during which they showered and rested. The purpose of this break was to allow body temperatures to return to preheat exposure levels, and this amount of time is sufficient, such that subsequent exercise performance will not be affected by prior heat exposure (23). After the 90-min break, nude body mass was again measured, and this value was compared with the value obtained before heat exposure. If body mass did not equal the value obtained before heat exposure in the EU condition, additional fluid was provided. In the HYPO condition, $(\Delta BM/PreBM) \times 100 = \%HYPO$, where BM is body mass.

Preload and performance time trial. Experimental testing consisted of 30 min of a preload (50% $\dot{V}O_{2peak}$) followed by a break (~10 min for instrumentation adjustments) and a self-paced 15-min performance time trial. The 30-min preload followed by a 15-min time trial in this study was selected, because *1*) preload exercise allows steady-state measures of physiological strain, *2*) the 15-min time trial has ecological validity for comparison with the similar exercise duration and energy system requirements (11) of the US Army 2-mile run, *3*) the 15-min time trial is a reliable performance test modality (21), and *4*) the 15-min time-trial performance test allowed for cross-validity with other studies from our laboratory (8, 23), as it limits several confounding factors on performance.

Table 1. *Subject characteristics for each cohort*

	T_a			
	10°C	20°C	30°C	40°C
Age, yr	23 ± 5 (18–32)	23 ± 3 (19–27)	24 ± 6 (18–35)	22 ± 3 (19–26)
Height, cm	180.0 ± 0.1 (173–185)	179.0 ± 0.1 (170–190)	179.0 ± 0.1 (172–187)	182.0 ± 0.1 (180–190)
Mass, kg	87.3 ± 13.7 (66–108)	79.8 ± 4.9 (74–90)	85.4 ± 14.5 (74–111)	89.1 ± 6.7 (79–95)
Peak O$_2$ uptake, ml·kg^{-1}·min^{-1}	43.6 ± 4.1 (37–48)	45.3 ± 4.6 (38–52)	46.3 ± 5.2 (38–51)	43.7 ± 7.0 (33–54)
Maximum peak O$_2$ uptake, W	313 ± 35 (262–359	300 ± 20 (258–316)	318 ± 24 (284–351)	314 ± 28 (260–340)
%Fat	14.6 ± 3.6 (10.7–20.4)	14.5 ± 2.8 (10.5–17.9)	14.0 ± 4.0 (9.2–20.6)	15.0 ± 4.0 (8.8–21.4)
BSA, m^2	2.1 ± 0.2 (1.8–2.3)	2.0 ± 0.1 (1.9–2.1)	2.0 ± 0.2 (1.9–2.3)	2.1 ± 0.1 (2.1–2.2)

Values are means ± SD, with range in parentheses ($n = 8$). T_a, ambient (air) temperature; BSA, body surface area.

Before experimental testing, subjects were fitted with an HR monitor and T_{sk} thermistors (Yellow Springs Instrument, Yellow Springs, OH) on the left chest, arm, calf, and thigh. Subjects then entered the environmental chamber set at 10°C (0.20 E_{req}/E_{max}, 7°C WBGT), 20°C (0.45 E_{req}/E_{max}, 16°C WBGT), 30°C (0.54 E_{req}/E_{max}, 22°C WBGT), or 40°C (0.66 E_{req}/E_{max}, 27°C WBGT) T_a and sat for ~30 min to equilibrate to the testing conditions. After resting equilibration, a 10-ml blood draw (controlling for arm position) was taken, subjects' fully instrumented body mass was measured, and they sat for an additional 10 min while preexercise measurements were recorded.

Preload exercise was achieved using the hyperbolic cycle ergometer mode and a 50% $\dot{V}O_{2peak}$ steady-state exercise intensity. Although exercise intensity was independent of pedal cadence, 60 ± 5 rpm was encouraged (25). Similarly, the cycle ergometer was switched to the linear mode for time-trial testing, where the linear factor was individualized to elicit a 50% $\dot{V}O_{2peak}$ power output for each subject at a cadence of 60 rpm (25). Selection of a linear factor that would elicit 50% $\dot{V}O_{2peak}$ at 60 rpm provided ample room for self-paced improvement up to maximal sustainable workloads, which were estimated at ~100 rpm from $\dot{V}O_{2peak}$ testing and expected to occur during the 15-min performance time trial. Drinking was not permitted during exercise, but subjects were weighed and rehydrated to within 1% of the instrumented body mass during the ~10-min break that followed the 30-min steady-state ride. During the break, skin thermistors, blood pressure (BP) cuff, and Finometer equipment were removed to eliminate interference with the subject during the 15-min performance time trial. Pedal cadence and workload were blinded, so that only elapsed time was known during the time trial, and no external motivation was provided. Elapsed time was given at standardized times of 5, 10, 12, 13, and 14 min, 30 s, and the final 10 s. Exercise HR and T_{re} were measured remotely every minute to limit distractions.

Physiological Measurements

During the 30-min preload, T_{sk} was monitored continuously using a data acquisition system, and mean weighted T_{sk} was calculated according to Ramanathan (31a). E_{req}/E_{max} was calculated to describe the range of compensable environments as an index (10). HR, T_{re}, and ratings of perceived exertion (RPE) were measured before exercise and every 5 min. Measurements of BP (by automated cuff; Cycle model, SunTech Medical, Morrisville, NC) and CO (Finometer, Finapres Medical Systems, Amsterdam, The Netherlands) were obtained before and at ~20 min of exercise. CO was derived from the BP waveform using the model flow method, incorporating age, sex, height, and weight (Beat-Scope 1.0 software, BMEye, Amsterdam, The Netherlands) (20). This method is a valid estimate of change in CO (19). Our laboratory has experience with this methodology (2) and has validated it against acetylene rebreathing during submaximal exercise. Percent change in CO at 20 min of steady-state preload exercise was calculated relative to preexercise measures. Mean arterial pressure (MAP) was calculated as follows: (systolic BP − diastolic BP) ∗ 0.33 + diastolic BP. Total peripheral resistance (TPR) was calculated using the following equation: TPR = MAP ÷ CO. TPR was expressed as percent change at 20 min of steady-state relative to preexercise. An estimate of whole body skin blood flow was made using the T_{re} and T_{sk} measurements at 20 min by the equation described by Rowell (32): $\dot{Q}_{sk} = 1/C \times h/(T_{re} - T_{sk})$, where C is specific heat of blood (≈ 0.87 kcal·°C^{-1}·l^{-1}), h is heat production ($\dot{V}O_2$, in l/min), and $\dot{Q}_{sk}$ is skin blood flow.

Analysis of Blood Samples

Before exposure to heat (dehydration protocol), a catheter was placed in an antecubital vein for blood sampling. The first 10-ml blood sample (before heat exposure) was taken after 20 min of seated rest to allow for equilibration. Subsequent samples were taken before and after 30-min preload while subjects were in a seated position. Whole blood was transferred to tubes containing lithium heparin, and samples of whole blood were taken for analysis of lactate, hemoglobin, and hematocrit (iSTAT 1 Analyzer, West Windsor, NJ). Whole blood was then centrifuged for 10 min at 4°C, and plasma was separated into aliquots for measurement of P_{osmol} in triplicate by freezing-point depression (model 210 Micro-Osmometer, Fiske, Norwood, MA). Percent change in blood volume (%ΔBV) was calculated using the equation of Dill and Costill (7) from appropriate hematocrit and hemoglobin values using the preexercise time point.

Statistical Analysis

Aerobic time-trial performance was the primary outcome of interest in this study. The practical importance of hypohydration on performance within environments was examined by using the mean and 95% confidence limits of the true effect for the percent change in performance to include comparison against an a priori zone of indifference [ZOI; percent coefficient of variation (CV) during practice trials] (3). Analysis of variance was used to separate within- and between-subject variation and provide a more elegant derivation of the %CV for each. Performance was measured as the total work performed (kJ) during a 15-min time trial, expressed as percent change from the EU condition. A power analysis selecting conventional alpha (0.05) and beta (0.20) values indicated that eight subjects within each T_a cohort would provide ample power to detect a meaningful difference ($\geq$5% change) in time-trial performance (24). This estimate was made using the mean total work (200 kJ) and the within-subjects CV (5.0%) calculated from practice trials of negligible difference (Grubb's test) during 2 wk of time-trial practice. The desire to detect an effect >1.0 times the CV was chosen on the basis of the likelihood of experimental perturbations producing unique performance infidelity (18), thus increasing performance variability. As performance heterogeneity was substantial between subjects (CV = 22%), despite similar phenotypic profiles and matching for $\dot{V}O_{2peak}$ (Table 1), we chose to make statistical comparisons between hydration states (EU vs. HYPO) only within groups (temperature) using a two-tailed, paired *t*-test. The interaction between hydration and environment on performance was based on the relationship of T_a to T_{sk} (10) and characterized by regression analysis of T_{sk} (*x*) on performance (*y*) across conditions (GraphPad, version 4.0). Statistical significance was accepted at the 95% probability level. Graphical data are presented with unidirectional error bars for clarity of presentation. All data are presented as means ± SD, except where indicated.

RESULTS

Hydration State

All subjects in the four cohorts completed all aspects of testing. All subjects were EU prior to heat exposure: their body mass was within 1% of the 5-day average, USG was <1.020, and P_{osmol} was <290 mmol/kgH$_2$O (34). In the HYPO trials, exposure to heat significantly elevated P_{osmol} (297–302 mmol/kgH$_2$O) and resulted in a −4.1%, −4.2%, −4.0%, and −4.1% reduction in body mass in 10°C, 20°C, 30°C, and 40°C trials, respectively. Hypohydration induced a −2.5% ΔBV (range −1.3% to −3.4% and −6.5% in plasma volume) at rest. To maintain euhydration during heat exposure for the subsequent EU trials, subjects drank 3.7 ± 0.6 liters of 0.05% NaCl-water solution (P_{osmol} = 284–287 mmol/kgH$_2$O).

Time-Trial Performance

Table 2 provides the physiological and perceptual strain at the completion of each time trial. HR and RPE were not different ($P < 0.05$) between EU and HYPO trials. T_{re} values were greater ($P < 0.05$) during HYPO than EU trials within each T_a.

Table 2. *HR, T_{re}, and RPE at completion (15 min) of the performance time trial*

	T_a			
	10°C	20°C	30°C	40°C
T_{re}, °C				
EU	37.7 ± 0.4	37.9 ± 0.2	37.9 ± 0.3	38.0 ± 0.3
HYPO	38.4 ± 0.2*	38.6 ± 0.2*	38.7 ± 0.4*	38.9 ± 0.4*
HR, beats/min				
EU	184 ± 9	185 ± 7	185 ± 8	181 ± 13
HYPO	187 ± 9	188 ± 7	186 ± 13	183 ± 11
RPE				
EU	17 ± 3	18 ± 2	19 ± 1	19 ± 1
HYPO	18 ± 2	18 ± 2	19 ± 1	19 ± 1

Values are means ± SD. T_{re}, rectal (core) temperature; HR, heart rate; RPE, rating of perceived exertion; EU, euhydrated; HYPO, hypohydrated. *Significantly different ($P < 0.05$) from EU within an environment.

Figure 1 provides the individual time-trial performance data for each environment as plotted against the line of identity. Time-trial performance (total work) values were 199 ± 37 and 194 ± 36 kJ during EU and HYPO trials, respectively, in 10°C ($P = 0.1$); 198 ± 22 and 179 ± 20 kJ during EU and HYPO, respectively, in 20°C ($P = 0.06$); 198 ± 21 and 174 ± 30 kJ during EU and HYPO, respectively, in 30°C ($P < 0.05$); and 157 ± 19 and 122 ± 37 kJ during EU and HYPO, respectively, in 40°C ($P < 0.05$).

Figure 2 plots the 95% confidence interval (CI), individual percent change in total work values from EU, mean values, and within-subjects CV or ZOI (±5%, based on within-subject
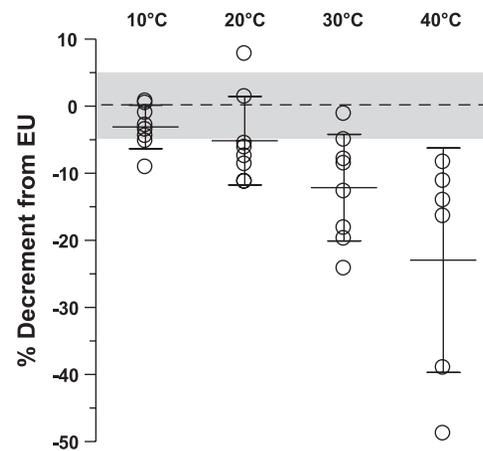

Fig. 2. Percent decrement in total work performance relative to EU trial for all subjects ($n = 32$) in 10°C, 20°C, 30°C, and 40°C T_a. Data are means; horizontal bars are 95% CI. Shaded area represents coefficient of variation (±5%) based on performance variability measured during 2-wk familiarization sessions.

performance variation measured during the 4 familiarization sessions) in 10°C, 20°C, 30°C, and 40°C T_a. The percent change in total work (and 95% CI) from EU was −3.0% (−5.8 to −0.4) at 10°C, −5% (−10.6 to 0.40) at 20°C, −12% (−18.8 to −5.5) at 30°C, and −23% (−40.5 to −5.4) at 40°C (Fig. 2). At 10°C, seven of eight values were within the ZOI. At 20°C, six of eight subjects were below the ZOI, with the mean falling on the edge; thus we considered the combination
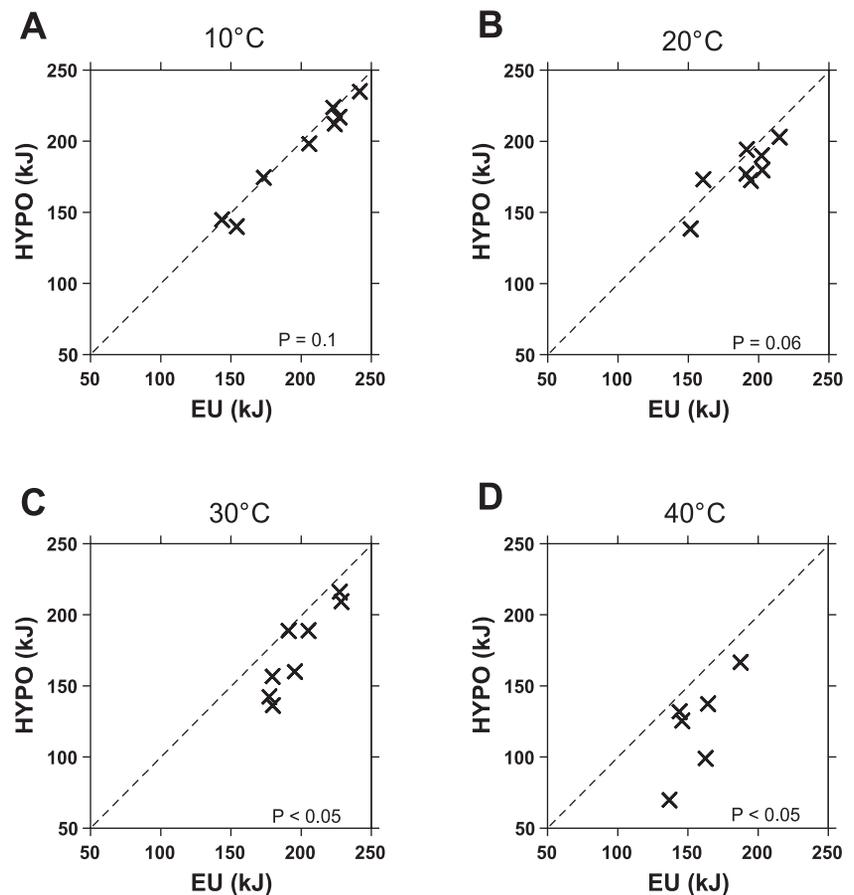

Fig. 1. Individual time-trial performance data and mean ± SD values for each environment [euhydration (EU) and hypohydration (HYPO)] plotted against the line of identity and inset histogram of total work (kJ) in 10°C (*A*), 20°C (*B*), 30°C (*C*) and 40°C (*D*) ambient temperature (T_a).
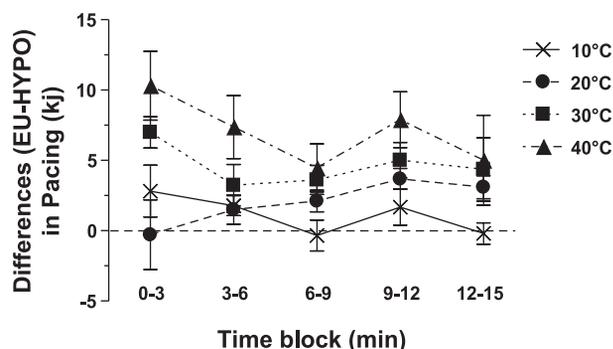
Fig. 3. Difference in pacing (EU-HYPO; 3-min work blocks) across the performance time trial in 10°C, 20°C, 30°C, and 40°C T_a.

of hypohydration and exposure to 20°C to be of practical importance. At 30°C, seven of eight subjects and the mean were below the ZOI; at 40°C, all subjects and the mean were below the ZOI during the HYPO trial.

Graded air temperatures were employed to produce systematic elevations in T_{sk}. During the preload bout, T_{sk} increased ~4.0°C with each 10°C T_a. A regression analysis was performed between those T_{sk} values and time-trial performance across all four environments. A significant relationship ($r = 0.61$, $P < 0.05$) was found between T_{sk} and EU-HYPO performance decrement, such that when T_{sk} is >29°C, each subsequent 1°C T_{sk} elevation yielded ~1.6% decline in total work.

Figure 3 provides pacing data (EU vs. HYPO) to examine how total work was accumulated (pacing) during the performance trials. The difference in pacing (EU vs. HYPO) was graphed in 3-min blocks (Fig. 3) (8). Visual examination suggests that pacing strategy was comparable in all environ-

ments. Overall, pacing was slower in HYPO than EU trials. In warmer (30°C and 40°C) trials, pacing was initially reduced and remained so throughout the rest of the time trial.

Preload Exercise Responses

$\dot{V}O_2$ (2.0 l/min) was not affected by exercise duration or environmental condition. The steady-state exercise bout was employed to induce heat strain and associated physiological responses prior to initiation of the performance test. Table 3 provides the physiological data at rest (preexercise) and at 30 min of preload exercise. T_{sk} values were not different ($P > 0.05$) between EU and HYPO at rest or exercise in any environment. T_{re} values were greater ($P < 0.05$) at rest and 30 min of exercise in the EU than HYPO trials within each T_a. The T_{re}-T_{sk} gradient was not different ($P > 0.05$) between EU and HYPO trials at rest or at 30 min of exercise within each T_a, except at 40°C, where the HYPO value was greater than the EU value (2.2 ± 0.4°C vs. 1.7 ± 0.2°C). HR was greater ($P < 0.05$) in the HYPO than EU trial at rest and exercise within each environment. %ΔBV from rest (0 min) was greater in the HYPO trial in each T_a, but this difference was significant only at 40°C ($P < 0.05$, HYPO > EU). Whole body sweat rates were greater ($P < 0.05$) in the EU than HYPO trial at 20°C, 30°C, and 40°C but were not different ($P > 0.05$) at 10°C. Whole body skin blood flow estimates were not different between EU and HYPO trials in the 10°C, 20°C, or 30°C environment but were lower ($P < 0.05$) in HYPO than EU trials at 40°C.

Figure 4 shows the percent change in CO, MAP, and TPR from rest to 20 min of preload exercise in each environmental condition. Absolute CO values at 20 min were 17.3 ± 0.8 and 17.8 ± 1.3 l/min in EU and HYPO trials, respectively, at 10°C; 16.2 ± 1.1 and 16.0 ± 1.6 l/min in EU and HYPO trials,

Table 3. *T_{sk}, T_{re}, T_{re}-T_{sk} gradient, HR, percent change in blood volume, whole body SR, and estimated whole body skin blood flow at pre- and post-steady-state preload*

	T_a							
	10°C		20°C		30°C		40°C	
	0 min	30 min	0 min	30 min	0 min	30 min	0 min	30 min
T_{sk}, °C								
EU	24.8 ± 0.8	25.0 ± 0.8	28.7 ± 0.6	29.0 ± 0.7	32.5 ± 0.2	32.5 ± 0.6	35.6 ± 0.4	35.9 ± 0.4
HYPO	25.0 ± 1.0	24.7 ± 0.6	29.3 ± 0.8	29.3 ± 0.8	32.8 ± 0.2	32.8 ± 0.4	36.1 ± 0.3	36.3 ± 0.6
T_{re}, °C								
EU	37.2 ± 0.2	37.4 ± 0.5	37.1 ± 0.3	37.7 ± 0.2	37.1 ± 0.3	37.6 ± 0.2	37.2 ± 0.2	37.8 ± 0.3
HYPO	37.7 ± 0.1*	38.2 ± 0.3*	37.7 ± 0.2*	38.2 ± 0.2*	37.5 ± 0.3*	38.2 ± 0.3*	37.9 ± 0.2*	38.7 ± 0.3*
T_{re}-T_{sk}, °C								
EU	12.4 ± 0.8	13.0 ± 0.5	8.4 ± 0.5	8.7 ± 0.7	4.6 ± 0.4	5.1 ± 0.7	1.6 ± 0.2	2.0 ± 0.4
HYPO	12.7 ± 1.0	13.5 ± 0.8	8.4 ± 0.9	8.9 ± 0.9	4.8 ± 0.3	5.4 ± 0.6	1.9 ± 0.3	2.6 ± 0.4*
HR, beats/min								
EU	78 ± 15	127 ± 12	66 ± 11	132 ± 7	76 ± 7	135 ± 99	95 ± 11	155 ± 21
HYPO	87 ± 15*	141 ± 20*	86 ± 14*	152 ± 11*	97 ± 17*	160 ± 15*	119 ± 20*	171 ± 18*
%ΔBV								
EU		−0.1 ± 1.9		−2.7 ± 2.0		−4.2 ± 2.5		−6.4 ± 1.8
HYPO		−2.1 ± 2.3		−3.6 ± 1.9		−6.2 ± 2.5		−9.1 ± 1.5*
Whole body SR, l/h								
EU		0.3 ± 0.2		0.6 ± 0.2		0.9 ± 0.1		1.4 ± 0.3
HYPO		0.2 ± 0.1		0.4 ± 0.1*		0.7 ± 0.2*		1.1 ± 0.2*
Estimated SkBF, l/min								
EU		0.9 ± 0.1		1.2 ± 0.1		2.3 ± 0.3		7.0 ± 1.0
HYPO		0.9 ± 0.1		1.2 ± 0.2		2.1 ± 0.4		5.6 ± 0.9*

Values are means ± SD. T_{sk}, skin temperature; %ΔBV, percent change in blood volume; SR, sweat rate; SkBF, skin blood flow. *Significantly different ($P < 0.05$) from EU within an environment.
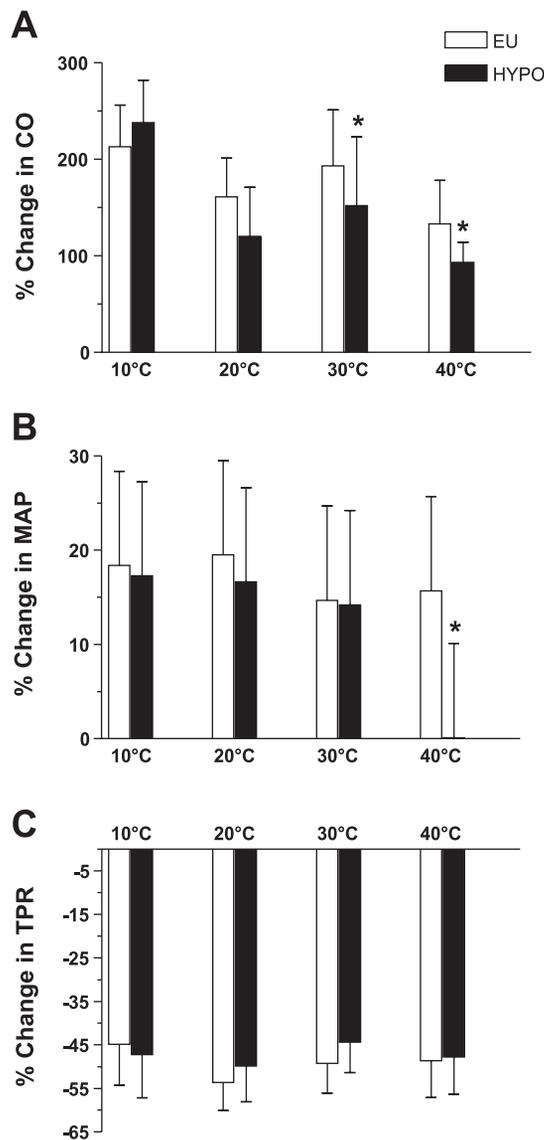
Fig. 4. Percent change in cardiac output (CO, A), mean arterial pressure (MAP, B), and total peripheral resistance (TPR, C) from before to 20 min of steady-state exercise for EU and HYPO conditions in 10°C, 20°C, 30°C, and 40°C T_a. *Significant difference ($P < 0.05$) from EU.

respectively, at 20°C; 16.1 ± 1.7 and 15.7 l/min in EU and HYPO trials, respectively, at 30°C; and 16.8 ± 1.6 and 14.6 ± 1.7 l/min in EU and HYPO trials, respectively, at 40°C. In the HYPO trials, the percent change in CO from rest was smaller ($P < 0.05$) at 30°C and 40°C (Fig. 4A). Absolute MAP values were not different ($P > 0.05$) at 10°C (115 ± 8 vs. 109 ± 12 mmHg), 20°C (108 ± 11 vs. 102 ± 12 mmHg), and 30°C (98 ± 12 vs. 99 ± 10 mmHg) between EU and HYPO trials, respectively, but were lower ($P < 0.05$) in HYPO than EU trials (83 ± 11 vs. 95 ± 10 mmHg) only in the 40°C environment. Similarly, the percent change in MAP from rest to 20 min of exercise was not different ($P > 0.05$) between EU and HYPO trials at 10°C, 20°C, and 30°C but was less ($P < 0.05$) at 40°C in the HYPO trials (Fig. 4B). The percent change in TPR between EU and HYPO trials was not different ($P > 0.05$) in any environment (Fig. 4C).

DISCUSSION

Our study is the first to use incremental heat-stress conditions to systematically evaluate the impact of hypohydration on aerobic exercise performance. Our data demonstrate that 1) hypohydration degrades aerobic performance to a greater extent with increasing heat stress, 2) at $T_{sk} > 29$°C, 4% hypohydration degrades aerobic performance by ~1.6% for each additional 1°C T_{sk}, and 3) cardiovascular strain from high skin blood flow requirements combined with blood volume reductions induced by hypohydration are important contributors to impaired performance. This means that athletes, military personnel, and occupational workers who experience skin warming (ambient conditions, wearing protective clothing or body armor) are more susceptible to negative effects of hypohydration on performance. Therefore, rehydration should be stressed more with increasing heat stress conditions, not simply because of greater sweat rates (13) and potential for incurring a water deficit (34), but because hyperthermia (particularly warm skin) accentuates the performance decrement from a given water deficit. Our approach of employing graded compensable heat stress avoided potential confounders of alternative approaches and allowed for the examination of the role of T_{sk} in aerobic exercise performance.

Our data clearly demonstrate that hypohydration degrades aerobic performance to a greater extent with increasing heat stress (>20°C). Time-trial performance (Fig. 1) during hypohydration (compared with euhydration) was -3% ($P = 0.1$), -5% ($P = 0.05$), -12% ($P < 0.05$), and -23% ($P < 0.05$) at 10°C, 20°C, 30°C, and 40°C T_a, respectively. In addition, our analysis of performance test variability (Fig. 2) provided additional support. The variability of our performance test was ±5% (Fig. 2). At 10°C, the 95% CI for the performance decrement was -5.8 to 0.07, which lies almost entirely within the variability of the test itself. Because the magnitude of change is nearly contained within the test variability, we do not consider the decrement in performance during the HYPO trial at 10°C to be of meaningful or practical importance. Although performance at 20°C was also not significantly impaired (95% CI crosses zero), one performance value clearly skewed this result (Fig. 2) but was retained in the analysis, because the relatively small cohort ($n = 8$) did not justify outlier classification. More importantly, the mean cohort value and the performance of six of eight subjects is outside the CV; thus we considered the combination of hypohydration and exposure to 20°C to be of practical importance. Our cool and temperate condition findings are similar to those of Cheuvront et al. (3), who used a similar hypohydration protocol but a slightly longer performance task (30 min). They reported a -7.6% decline in time-trial performance during hypohydration at 20°C ($P < 0.05$) and a -2.7% decline at 2°C ($P > 0.05$). In our study and the study of Cheuvront et al., T_{sk} at 10°C and 2°C was low (~25 and 20°C, respectively) and was similar in 20°C (~29°C). Subjects could not be blinded to hypohydration or exposure to extreme temperatures (10°C, 30°C, and 40°C); thus the psychological or hedonic impact of these factors cannot be discounted. However, during the performance time trial, HR and RPE were not different between EU and HYPO trials, which would indicate that the subjects worked to the maximum of their capability, despite environment or hydration state.

The 20°C condition (16°C WBGT, 0.45 E_{req}/E_{max}) was the approximate threshold at which hypohydration begins to degrade aerobic exercise performance. During the 20°C T_a experiments, the higher T_{sk} (~29°C) and resulting elevated whole body skin blood flow combined with hypohydration to reduce central blood volume and degrade aerobic exercise performance. As T_{sk} increased (~33 and ~36°C), there was a greater decrement in performance with the HYPO trial. The higher T_{sk} is associated with greater skin blood flow requirements and increased cutaneous venous compliance (33), acting to shift blood volume from central to peripheral circulation. Figure 2 demonstrates that, at 30°C and 40°C, the mean decrement in performance and 95% CI progressively lie well outside the CV, thus supporting our results that the hypohydration-mediated degradation in aerobic exercise performance is amplified with increasing T_{sk}.

A significant ($P < 0.05$) portion (37% of explained variance) of the aerobic performance decrements observed for HYPO subjects in this study was primarily due to high T_{sk}. Thus, despite inherent biological variation in performance between subjects (CV = 22%) under nearly ideal circumstances, in addition to the variation in performance due to experimental perturbations (19), T_{sk} still accounted more than one-third of the explained variance in time-trial performance across environments. It is important to note that confounders such as hyperthermia (core temperature elevation) and glycogen depletion were also carefully controlled; thus the decrements in performance observed with hypohydration represent the minimum that can be expected. Previously, we reported a larger performance reduction (~50%) with a test of longer duration in subjects who were previously glycogen depleted and hypohydration (−4%, accumulating to −5% body mass) in 40°C (4). The potential influence of very high ("critical") core temperature elevations (17) were minimized by the present design using compensable heat stress and short exercise bout. If glycogen depletion or core temperature elevations were not controlled, if a longer time trial were employed, or if the magnitude of fluid losses became even larger, performance decline would likely have been greater than that reported here (4).

Our data support recent studies (8, 9) demonstrating the importance of high T_{sk} on degrading aerobic performance in EU subjects. The sports medicine literature has recently focused on the concept of a critical core temperature of ~40°C as the primary mechanism of fatigue (17, 29–31). In the present study, T_{re} at the end of the performance time trial were <39.0°C (Table 2), which indicates that, in our experiments, high core temperature itself was likely not responsible for impaired aerobic exercise performance during the HYPO trial. Thus it appears that more attention needs to focus on the contributions of high T_{sk} on degrading aerobic performance (9). T_{sk} is a function of environmental temperature (10), and on exposure to hot environments, the T_{re}-to-T_{sk} gradient narrows and skin blood flow increases to maximize dry heat exchange with the environment. In the present study, with each 10°C increase in T_a, the T_{re}-T_{sk} gradient decreased by ~4.5°C (Table 3) and estimated skin blood flow increased in a stepwise manner, with the largest values at 40°C (7.0 and 5.6 l/min in EU and HYPO, respectively; Fig. 4). Thermoregulatory increases in skin blood flow to 6–8 l/min during severe hyperthermia (22, 40) and during moderately intense exercise reduce

stroke volume (SV), increase HR, and, ultimately, compromise CO (33). This cardiovascular strain is further exacerbated by hypohydration, which results in a blood (plasma) volume reduction and further reduces central venous pressure, venous return, SV, and CO below values reported in normally hydrated individuals (14, 15, 27, 28, 36). In fact, in our HYPO trials, we observed a significantly lower percent change (from rest) in CO at 30°C and 40°C (Fig. 4A) and a lower absolute and percent change in MAP at 40°C (Fig. 4B).

The combination of heat stress (30°C and 40°C) and hypohydration challenged any compensatory mechanisms to offset a reduction in cardiac filling to maintain CO (16). In the present study, we observed greater HR at 30 min of preload exercise at each T_a in HYPO trials. It appears that these HR increases were sufficient to offset SV reductions and, thus, sustain CO in HYPO trials at 10°C and 20°C but not at higher T_a and T_{sk}. These findings are similar to those of Gonzalez-Alonso et al. (16), who tested eight trained cyclists for 30 min at 72% of maximal $\dot{V}O_2$ in 8°C and 35°C while EU and with a 1.5%, 3.0%, and 4.2% body weight loss. When their subjects exercised in 35°C, SV and CO significantly declined at the higher levels of hypohydration while HR was significantly elevated. We also observed a significantly lower MAP and percent change in MAP from preexercise in 40°C HYPO trials, a finding that coincides with the largest decrement in aerobic exercise performance (Fig. 2). In our study, maintenance of MAP in the HYPO trials at 10–30°C agree with data reported by Gonzalez-Alonso et al. during exercise-heat stress and hypohydration; however, this was not the case during our 40°C trials. These factors likely combined in a higher fractional utilization of $\dot{V}O_{2peak}$ at any given power output (1), similar to the phenomenon observed at altitude (2), and contributed to the downregulation of pace in HYPO trials in progressively warmer T_a, when T_{sk} was >29°C (Fig. 3).

To determine how fatigue (indicated by total work) was accumulated during each time trial, we examined the pacing strategies (Fig. 3). While different subjects participated between each environmental condition, pacing was very similar for each condition, with a reduced pace in the 30°C and 40°C conditions. Ely et al. (8) observed the impact of heat exposure (40°C vs. 21°C) on pacing strategy during an identical 15-min performance time trial in EU subjects. They reported a 17% heat-related decline in aerobic performance due to an inability to maintain initial pace and a gradual slowing over time when normalizing pacing strategy within subjects. Tatterson et al. (39) examined pacing of elite cyclists during a 30-min time trial at 23°C and 32°C. They reported that power output significantly declined after 10 min of cycling in 32°C and remained lower for the duration of the time trial. Ours is the only study, to our knowledge, that has investigated pacing strategies in multiple environments or in response to hypohydration.

In conclusion, our data demonstrate that 1) hypohydration degrades aerobic performance to a greater extent with increasing heat stress, 2) at T_{sk} >29°C, 4% hypohydration degrades aerobic performance by ~1.6% for each additional 1°C T_{sk}, and 3) cardiovascular strain from high skin blood flow requirements combined with blood volume reductions induced by hypohydration, are important contributors to the observed performance degradations. These findings have important implications for athletic and occupational communities, where

hypohydration and warm skin can combine to limit work performance.

DISCLAIMER

The views, opinions, and/or findings in this report are those of the authors and should not be construed as official Department of the Army position, policy, or decision unless so designated by other official designation. All experiments were carried out in accordance to state and federal guidelines.

DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the authors.

REFERENCES

1. **Arngrimsson SA, Stewart DJ, Borrani F, Skinner KA, Cureton KJ.** Relation of heart rate to percent $\dot{V}o_{2peak}$ during submaximal exercise in the heat. *J Appl Physiol* 94: 1162–1168, 2003.
2. **Beidleman BA, Muza SR, Fulco CS, Jones JE, Lammi E, Staab JE, Cymerman A.** Intermittent hypoxic exposure does not improve endurance performance at altitude. *Med Sci Sports Exerc* 41: 1317–1325, 2009.
3. **Cheuvront SN, Carter R, Castellani JW, Sawka MN.** Hypohydration impairs endurance exercise performance in temperate but not cold air. *J Appl Physiol* 99: 1972–1976, 2005.
4. **Cheuvront SN, Carter R 3rd, Kolka MA, Lieberman HR, Kellogg MD, Sawka MN.** Branched-chain amino acid supplementation and human performance when hypohydrated in the heat. *J Appl Physiol* 97: 1275–1282, 2004.
5. **Cheuvront SN, Goodman DA, Kenefick RW, Montain SJ, Sawka MN.** Impact of a protective vest and spacer garment on exercise-heat strain. *Eur J Appl Physiol* 102: 577–583, 2008.
6. **Coyle EF.** Fluid and fuel intake during exercise. *J Sports Sci* 22: 39–55, 2004.
7. **Dill DB, Costill DL.** Calculation of percentage changes in volumes of blood, plasma, and red cells in dehydration. *J Appl Physiol* 37: 247–248, 1974.
8. **Ely BR, Cheuvront SN, Kenefick RW, Sawka MN.** Aerobic performance is degraded, despite modest hyperthermia, in hot environments. *Med Sci Sports Exerc* 42: 135–141, 2010.
9. **Ely BR, Ely MR, Cheuvront SN, Kenefick RW, DeGroot DW, Montain SJ.** Evidence against a 40°C core temperature threshold for fatigue in humans. *J Appl Physiol* 107: 1519–1525, 2009.
10. **Gagge AP, Gonzalez RR.** Mechanisms of heat exchange: biophysics and physiology. In: *Handbook of Physiology. Environmental Physiology.* Bethesda, MD: Am. Physiol. Soc., 1996, sect. 4, p. 45–84.
11. **Gastin PB.** Energy system interaction and relative contribution during maximal exercise. *Sports Med* 31: 725–741, 2001.
12. **Godek SF, Godek JJ, Bartolozzi AR.** Thermal responses in football and cross-country athletes during their respective practices in a hot environment. *J Athl Train* 39: 235–240, 2004.
13. **Gonzalez RR, Cheuvront SN, Montain SJ, Goodman DA, Blanchard LA, Berglund LG, Sawka MN.** Expanded prediction equations of human sweat loss and water needs. *J Appl Physiol* 107: 379–388, 2009.
14. **Gonzalez-Alonso J, Calbet JA, Nielsen B.** Muscle blood flow is reduced with dehydration during prolonged exercise in humans. *J Physiol* 513: 895–905, 1998.
15. **Gonzalez-Alonso J, Mora-Rodriguez R, Below P, Coyle E.** Dehydration reduces cardiac output and increases systemic and cutaneous vascular resistance during exercise. *J Appl Physiol* 79: 1487–1496, 1995.
16. **Gonzalez-Alonso J, Mora-Rodriguez R, Coyle EF.** Stroke volume during exercise: interaction of environment and hydration. *Am J Physiol Heart Circ Physiol* 278: H321–H330, 2000.
17. **Gonzalez-Alonso J, Teller C, Andersen SL, Jensen FB, Hyldig T, Nielsen B.** Influence of body temperature on the development of fatigue during prolonged exercise in the heat. *J Appl Physiol* 86: 1032–1039, 1999.
18. **Hopkins WG.** Measures of reliability in sports medicine and science. *Sports Med* 30: 1–15, 2000.
19. **Imholz BP, Wieling W, van Montfrans GA, Wesseling KH.** Fifteen years experience with finger arterial pressure monitoring: assessment of the technology. *Cardiovasc Res* 38: 605–616, 1998.
20. **Jellema WT, Wesseling KH, Groeneveld AB, Stoutenbeek CP, Thijs LG, Van Lieshout JJ.** Continuous cardiac output in septic shock by simulating a model of the aortic input impedance: a comparison with bolus injection thermodilution. *Anesthesiology* 90: 1317–1328, 1999.
21. **Jeukendrup A, Saris WH, Brouns F, Kester AD.** A new validated endurance performance test. *Med Sci Sports Exerc* 28: 266–270, 1996.
22. **Johnson JM, Proppe DW.** Cardiovascular adjustments to heat stress. In: *Handbook of Physiology. Environmental Physiology.* Bethesda, MD: Am. Physiol. Soc., 1996, sect. 4, p. 215–243.
23. **Kenefick RW, Ely BR, Cheuvront SN, Palombo LJ, Goodman DA, Sawka MN.** Prior heat stress: effect on subsequent 15-min time trial performance in the heat. *Med Sci Sports Exerc* 41: 1311–1316, 2009.
24. **Lipsey MW.** *Design Sensitivity: Statistical Power for Experimental Research.* Newbury Park, CA: Sage, 1990.
25. **McNaughton L, Thomas D.** Effects of differing pedalling speeds on the power-duration relationship of high intensity cycle ergometry. *Int J Sports Med* 17: 287–292, 1996.
26. **Montain SJ, Coyle EF.** Influence of graded dehydration on hyperthermia and cardiovascular drift during exercise. *J Appl Physiol* 73: 1340–1350, 1992.
27. **Montain SJ, Sawka MN, Latzka WA, Valeri CR.** Thermal and cardiovascular strain from hypohydration: influence of exercise intensity. *Int J Sports Med* 19: 1–5, 1998.
28. **Nadel ER, Fortney SM, Wenger CB.** Effect of hydration state on circulatory and thermal regulations. *J Appl Physiol* 49: 715–721, 1980.
29. **Nybo L.** Hyperthermia and fatigue. *J Appl Physiol* 104: 871–878, 2008.
30. **Nybo L, Jensen T, Nielsen B, Gonzalez-Alonso J.** Effects of marked hyperthermia with and without dehydration on $\dot{V}o_2$ kinetics during intense exercise. *J Appl Physiol* 90: 1057–1064, 2001.
31. **Nybo L, Nielsen B.** Hyperthermia and central fatigue during prolonged exercise in humans. *J Appl Physiol* 91: 1055–1060, 2001.
31a. **Ramanathan NL.** A new weighting system for mean surface temperature of the human body. *J Appl Physiol* 19: 531–533, 1964.
32. **Rowell LB.** *Human Circulation: Regulation During Physical Stress.* New York: Oxford University Press, 1986, p. 363–406.
33. **Rowell LB, Marx HJ, Bruce RA, Conn RD, Kusumi F.** Reductions in cardiac output, central blood volume, and stroke volume with thermal stress in normal men during exercise. *J Clin Invest* 45: 1801–1816, 1966.
34. **Sawka MN, Burke LM, Eichner ER, Maughan RJ, Montain SJ, Stachenfeld NS.** American College of Sports Medicine position stand. Exercise and fluid replacement. *Med Sci Sports Exerc* 39: 377–390, 2007.
35. **Sawka MN, Francesconi RP, Young AJ, Pandolf KB.** Influence of hydration level and body fluids on exercise performance in the heat. *JAMA* 252: 1165–1169, 1984.
36. **Sawka MN, Knowlton RG, Critz JB.** Thermal and circulatory responses to repeated bouts of prolonged running. *Med Sci Sports* 11: 177–180, 1979.
37. **Sawka MN, Noakes TD.** Does dehydration impair exercise performance? *Med Sci Sports Exerc* 39: 1209–1217, 2007.
38. **Sawka MN, Wenger CB, Pandolf KB.** Thermoregulatory responses to acute exercise-heat stress and heat acclimation. In: *Handbook of Physiology. Environmental Physiology.* Bethesda, MD: Am. Physiol. Soc., 1996, sect. 4, p. 157–185.
39. **Tatterson AJ, Hahn AG, Martin DT, Febbraio MA.** Effects of heat stress on physiological responses and exercise performance in elite cyclists. *J Sci Med Sport* 3: 186–193, 2000.
40. **Taylor WF, Johnson JM, O'Leary D, Park MK.** Effect of high local temperature on reflex cutaneous vasodilation. *J Appl Physiol* 57: 191–196, 1984.