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ORIGINAL ARTICLE

Influence of relative humidity on prolonged exercise capacity in a warm environment

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Abstract This study examined the influence of relative humidity on endurance exercise performance in a warm environment. Eight male volunteers performed four cycle exercise trials at 70% maximum oxygen uptake until volitional exhaustion in an environmental chamber maintained at 30.2 ± 0.2°C. Volunteers were tested under four relative humidity (rh) conditions: 24, 40, 60 and 80%. Core and weighted mean skin temperature, heart rate, skin blood flow, and cutaneous vascular conductance were recorded at rest and at regular intervals during exercise. Mean ± SD time to exhaustion was 68 ± 19, 60 ± 17, 54 ± 17, and 46 ± 14 min at 24, 40, 60, and 80% rh, respectively ($P < 0.001$); exercise time was significantly less at 60% ($P = 0.013$) and 80% ($P = 0.005$) rh than recorded at 24% rh. There were no differences in core temperature ($P = 0.480$) and heart rate ($P = 0.097$) between trials. Core temperature at exhaustion was 39.0 ± 0.3°C at 24, 40, and 60% rh and 39.1 ± 0.3°C at 80% rh ($P = 0.159$). Mean skin temperature at the point of exhaustion was higher at 80% rh than at 24% rh ($P < 0.001$). Total sweat loss was similar between trials ($P = 0.345$), but sweating rate was higher at 60 and 80% rh than at 24% rh ($P < 0.001$). The results suggest that exercise capacity at moderate intensity in a warm environment is progressively impaired as the relative humidity increases.

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Introduction

It has long been known that endurance exercise performance is impaired in hot environments. Dill et al. (1931) showed that performance at 34°C was less than at 12°C when the relative humidity (rh) was approximately constant at 50%. The humidity and the rate of air movement have also been recognised as important factors, and several studies on the effects of environmental conditions on thermal comfort and work output in industrial settings were carried out in the first half of the 20th century (Robinson et al. 1945). Later studies have systematically examined the influence of ambient temperature on prolonged exercise performance in a laboratory environment (Galloway and Maughan 1997; Parkin et al. 1999; Tatterson et al. 2000). In addition, studies have reported a progressive slowing of competitive race performance (Ely et al. 2007; McCann and Adams 1997) as wet-bulb globe temperature (WBGT) increases. Although the importance of humidity for effective thermoregulation during exercise, especially in warm environments, has been recognised in these studies, there has been no systematic investigation to confirm and quantify this effect. While Armstrong (2000) reported that steady state core temperature during exercise would increase in proportion to the relative humidity at ambient temperatures in excess of 16°C, this was based on a "hypothetical athlete" rather than experimental data. During exercise in warm environments, a primary limitation to endurance capacity may be related to the rise in body core temperature producing a direct effect on the central nervous system rather than to any metabolic, cardiovascular or

respiratory limitation (Nielsen et al. 1993). As the temperature gradient between the skin and the air is small under these conditions, the capacity to dissipate heat through radiation and convection is limited. Consequently, the body becomes increasingly reliant on evaporation to regulate body temperature, with heat lost due to the latent heat of vaporisation of water from the respiratory tract and the skin surface. When humidity is low and the skin temperature high, sweat secreted onto bare skin is readily evaporated and heat can be transferred at high rates from the body to the environment. Conversely, when the humidity of the environment is high, the rate at which sweat evaporates from the skin is lower than it would be under dry conditions. This is likely to result in a marked rise in core temperature despite ongoing sweat losses.

Niwa and Nakayama (1978) confirmed the elevation of core temperature in exercise of moderate intensity when the humidity was high and also reported an increased rate of sweat secretion and a decreased evaporation at high humidity. They did not, however, make any measure of exercise performance, and their study involved only three subjects. Backx et al. (2000) reported no effect on the performance of a high-intensity exercise test (3 × 30 s sprints) in what they described as normal (22°C, 30% rh), wet (30°C, 85% rh), and hot (40°C, 40% rh) environments, but this is perhaps not surprising given the exercise test employed. The few studies on the effects of humidity on exercise performance have been focussed on the implications for those with exercise-induced asthma or other respiratory problems. Maximum oxygen uptake (VO_{2max}) and peak running speed were lower in subjects with exercise-induced bronchoconstriction during an exercise test at 20°C when the ambient humidity was 40% than at high (95%) humidity at the same temperature (Stensrud et al. 2006). In the subjects who participated in these studies, however, there is a ventilatory limitation to exercise performance that is exacerbated by dryness of the airways, but this does not appear to apply in the absence of asthma.

The specific influence of variations in relative humidity on performance, and the physiological response to exercise, remains largely unexplored. Differences in humidity have been factored into studies investigating the relationship between environmental conditions and competitive race performance in athletes, where WBGT was used as an index of heat stress (Ely et al. 2007; McCann and Adams 1997). WBGT is a widely accepted index for the assessment of heat stress and provides a composite measure of ambient temperature, humidity and solar load. Since physical activity can take place in both warm/dry and warm/humid environments, it is important to be able to quantify the influence of these environmental extremes on work capacity. The aim of this study was therefore to examine the effects of variations in humidity on performance and the physiological response to exercise, in a warm environment when all other factors remain constant.

Methods

Subjects

Eight healthy males (mean ± SD age 26 ± 4 years; height 1.80 ± 0.03 m; body mass 72.0 ± 7.0 kg; VO_{2max} 4.38 ± 0.65 L/min) participated in this investigation. All subjects were active, but none was accustomed to exercise in a warm environment at the time of the study. Prior to volunteering all participants received written information regarding the nature and purpose of the study. Following an opportunity to ask any questions, a written statement of consent was signed. The protocol employed was approved by the local Ethical Advisory Committee (Ref no: R10-P55).

Experimental protocol

All subjects completed an initial maximal exercise test, one familiarisation trial and four experimental trials. The preliminary trial consisted of incremental cycle exercise to volitional exhaustion and was used to determine VO_{2max} and the power output required to elicit 70% VO_{2max}. A familiarisation trial was undertaken to ensure the subjects were accustomed to the procedures employed during the investigation and to minimise learning or anxiety effects. This trial was performed in an environment maintained at 30°C and 80% rh and was identical to the experimental trials in all respects. Experimental trials were undertaken in a climatic chamber with the temperature at 30.2 ± 0.2°C, air velocity 1 m/s, and the relative humidity was maintained at either 24 ± 3, 40 ± 1, 60 ± 1 or 80 ± 1%. Consequently, the calculated WBGT index was 19.6, 23.2, 26.0 and 28.4°C, respectively. Experimental trials were completed in a counter-balanced, randomised order and were separated by at least 7 days to minimise the development of heat acclimation. Subjects were instructed to record dietary intake and physical activity during the 24 h before the first trial and to replicate this on the day prior to the subsequent experimental trials. No exercise or alcohol consumption was permitted in the 24 h before each trial.

Subjects entered the laboratory in the morning after an overnight fast, other than the ingestion of 500 mL of plain water 90 min before the start of the trial. Upon arrival a urine sample was collected and nude body mass was measured to the nearest 10 g (Adam AFW-120 K, Milton Keynes, UK). Subjects then inserted a rectal thermistor (Grant Squirrel SQ800, Cambridgeshire, UK) 10 cm beyond the anal sphincter for the measurement of core

temperature. Surface skin temperature probes (Grant Squirrel SQ800, Cambridgeshire, UK) were attached to four sites (chest, upper arm, thigh, and calf) to enable the calculation of weighted mean skin temperature (Tsk; Ramanathan 1964). Total body heat content was calculated as described by Webb (1995), assuming the specific heat of the body to be 3.47 kJ/°C/kg. These data were used to determine the rate of heat storage during each trial. A heart rate telemetry band (Polar beat, Kempele, Finland) was positioned and a skin blood flow probe (VMS-LDF Laser Doppler monitor, Moor Instruments, Devon, UK) was also secured on the medial aspect of right forearm, approximately 5 cm from the radial styloid process. Subjects were dressed in cycling shorts, socks and shoes for all trials.

Subjects then rested in a seated position for 15 min in a comfortable environment (22–23°C) with one hand immersed in warm water (42°C) for 10 min to allow arterialised venous blood to be drawn at rest. A 21 g cannula was introduced into a superficial forearm vein of the opposite arm to the skin blood flow probe, to enable repeated blood sampling. The cannula was flushed with a small volume of heparinized saline after each collection. During this period, resting temperature and heart rate were recorded at 5-min interval. A 60-s measurement of skin blood flow was made after 10 min of seated rest and blood pressure (BP) was measured using a manual sphygmomanometer (Accoson, Essex, UK). A venous blood sample was then drawn.

Subjects entered the climatic chamber and began cycle exercise at a power output corresponding to 70% $\dot{V}O_{2max}$. Exercise continued to volitional exhaustion, defined as the point at which volunteers were unable to maintain a pedal cadence of 60 rev/min despite verbal encouragement. Expired air for the measurement of oxygen uptake and respiratory exchange ratio was collected over a 2-min period for every 15 min: these data were used to calculate the rates of fat and carbohydrate (CHO) oxidation. Core and skin temperatures as well as heart rate were recorded at 5-min interval during exercise. Ratings of perceived exertion (Borg 1982) and perceived thermal stress (Hardy 1970) were assessed every 10 min. Forearm skin blood flow and BP were measured at 10-min interval until the 30th minute of exercise and then at the point of exhaustion. Cutaneous vascular conductance (CVC) was calculated as the ratio of skin blood flow (arbitrary units) to mean arterial pressure, and normalized relative to the peak CVC attained by the volunteer during each trial. Mean arterial pressure was calculated as (systolic BP − diastolic BP)/3 + diastolic BP. Venous blood samples were drawn after 15 and 30 min of exercise and at the point of exhaustion. Following exercise, subjects returned to a comfortable environment, the probes and cannula were removed and nude body mass was re-measured to allow the estimation of sweat losses (Maughan et al. 2007).

Blood collection and analysis

Five millilitre blood samples collected throughout the experimental protocol were drawn into dry syringes, with 1 mL dispensed into tubes containing K_2EDTA and the remaining 4 mL into plain tubes. Duplicate 100 μL aliquots of EDTA-treated whole blood were rapidly deproteinised in 1 mL of ice-cold 0.3 N perchloric acid. These were centrifuged, and the resulting supernatant was used for the determination of blood glucose (God-PAP, Randox, Co. Antrim, UK). Haemoglobin (cyanmethemoglobin method) and haematocrit (microcentrifugation) values were used to estimate percentage changes in blood, plasma, and red cell volumes relative to the resting sample (Dill and Costill. 1974). The 4 mL aliquot was left to clot at room temperature for 60 min before being centrifuged to yield serum. This was stored at 4°C for the analysis of sodium and potassium concentrations (Corning 410C, New York, USA) and osmolality (Gonotec Osmomat 030, YSI, Farnborough, UK). All analyses were carried out in duplicate, except for the haematocrit measurements, which were made in triplicate.

Statistical analysis

Data are presented as means ± standard deviation (SD) unless otherwise stated. To evaluate differences in exercise capacity, a one-way repeated measures ANOVA was employed. Cohen's *d* effect sizes (ES) for the differences in exercise time were also determined. Data collected over time were analysed using a two-factor (time-by-trial) repeated measures ANOVA. Pair-wise differences between trials were evaluated using one-way ANOVAs with a Bonferroni adjustment applied for multiple comparisons. Relationships between variables, including changes in core/skin temperature and perceived exertion/thermal stress, were examined using a mixed-effects model (Lange et al. 1989). Statistical significance was accepted at $P < 0.05$. To improve the clarity of figures, data are displayed up to the last time point where all volunteers were still exercising and group standard deviations are displayed. Based on the results of a previous investigation (Wilson and Maughan 1992), we estimated a 90% probability of detecting a difference in time to exhaustion of 7.2 min with a sample size of 8 subjects.

Results

Pre-exercise body mass was not different between trials ($P = 0.533$) and there was also no difference between trials in the pre-exercise urine osmolality ($P = 0.806$), blood glucose ($P = 0.168$) or core temperature ($P = 0.749$), suggesting that subjects began each trial in a similar physiological state. There was no apparent order

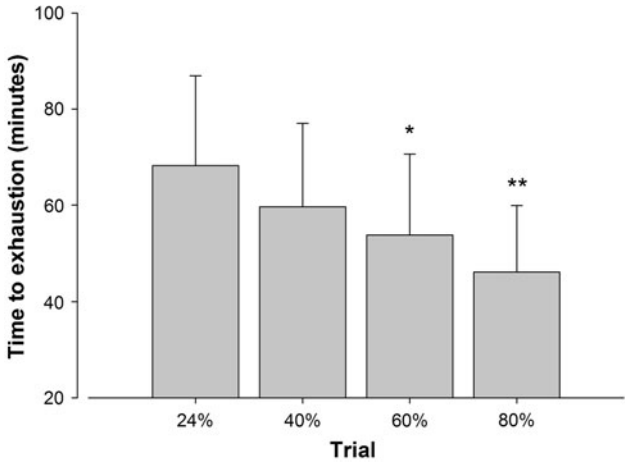

Fig. 1 Effect of the relative humidity of the environment on cycling time to exhaustion. *$P < 0.05$ and **$P < 0.01$ denote a significant difference from the 24% trial

effect across the four experimental trials ($P = 0.767$). A clear effect of the humidity of the environment on cycling time to exhaustion was observed ($P < 0.001$; Fig. 1). Further exploration of the data showed that performance relative to that at 24% rh was not significantly different at 40% rh (by 8.6 ± 8.6 min; $P = 0.152$; ES 0.49) but was reduced at 60% (by 14.5 ± 8.6 min; $P = 0.013$; ES 0.86) and at 80% (by 22.1 ± 11.0 min; $P = 0.005$; ES 1.61).

Figure 2 shows the responses of core temperature (upper panel) and skin temperature (lower panel). There was no effect of environment on core temperature during exercise ($P = 0.480$). In spite of the longer exercise times at lower humidity, the core temperature at the end of exercise was not different between trials (39.0 ± 0.3°C at 24, 40 and 60% rh and 39.1 ± 0.3°C at 80% rh, $P = 0.159$). The rate of rise in core temperature was significantly greater in the 80% trial than in the 24% trial (24% 0.3 ± 0.1°C/10 min; 40% 0.4 ± 0.1°C/10 min; 60% 0.4 ± 0.1°C/10 min; 80% 0.5 ± 0.1°C/10 min; $P < 0.001$). In addition, the calculated rate of heat storage during exercise was 8 ± 2, 9 ± 3, 10 ± 3 and 12 ± 3 kJ/min in the 24, 40, 60 and 80% trials, respectively ($P < 0.001$). The Tsk was affected by the humidity of the environment ($P < 0.001$). Tsk was not significantly higher at 40% rh ($P = 1.000$) or at 60% ($P = 0.065$) than at 24%, but was higher at 80% ($P = 0.003$) than at 24%. After the first 20–30 min of exercise, there was little or no change in skin temperature, though the core temperature continued to rise to the end of exercise. Tsk at the point of exhaustion was 33.6 ± 0.7, 33.8 ± 0.7, 34.3 ± 0.6 and 34.8 ± 0.5°C in the 24, 40, 60 and 80% trials, respectively, ($P < 0.001$).

Oxygen uptake and respiratory exchange ratio were not different between treatments, and there was no difference between trials in the calculated rates of fat and carbohydrate oxidation. Total CHO utilisation was decreased at higher

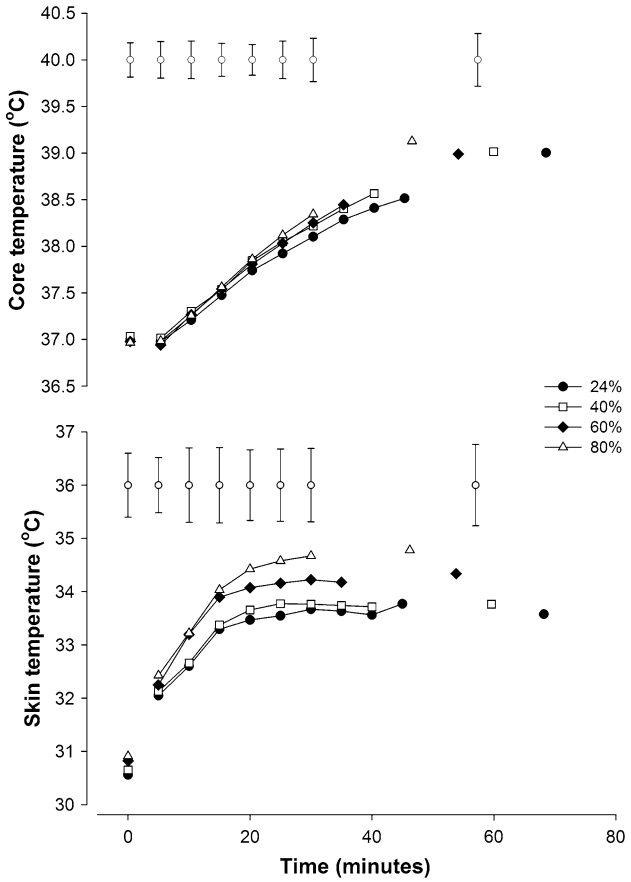

Fig. 2 Responses of core temperature (*upper panel*) and weighted mean skin temperature (*lower panel*) to exercise in environments with different relative humidity. Data are presented up to the last time point where all volunteers were still exercising in each trial along with the value recorded at exhaustion (mean and group standard deviation)

environmental humidity, but this is due to the reduction in exercise time, with 246 ± 63, 219 ± 57, 204 ± 64 and 177 ± 24 g of CHO oxidised in the 24, 40, 60 and 80% trials, respectively ($P = 0.003$). There was no significant difference in fat oxidation rates apparent between trials ($P = 0.583$). Exercise heart rate (Fig. 3) was not different between conditions, although there was a tendency for higher values with increasing relative humidity ($P = 0.097$). Skin blood flow ($P = 0.796$) was also not significantly affected by the relative humidity of the environment (Fig. 4, upper panel). CVC (Fig. 4, lower panel) increased over time ($P < 0.001$) but was not different between conditions ($P = 0.454$). The calculated sweat rate was significantly influenced by the environment ($P < 0.001$; Fig. 5a), but the fraction of the sweat that evaporated was not measured. Sweating rate was not different between the 24% (22 ± 4 mL/min) and 40% (23 ± 6 mL/min) trials ($P = 0.706$), but was higher at 60% (27 ± 5 mL/min; $P = 0.002$) and at 80% (30 ± 6 mL/min; $P = 0.003$) than at 24%. Total sweat loss during exercise period was not different between trials (24% 1.49 ± 0.46 L; 40%

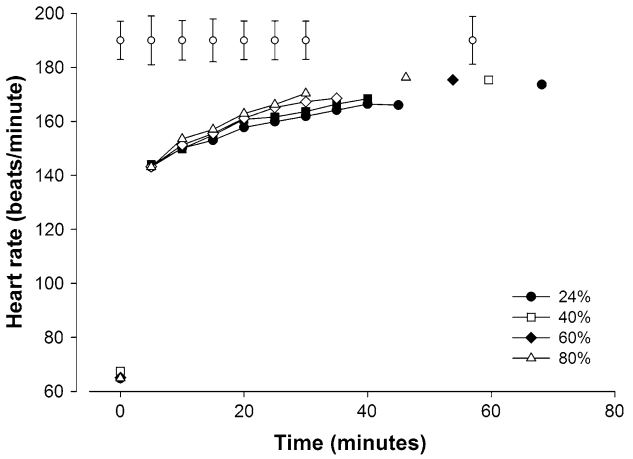

Fig. 3 Heart rate response to exercise in environments with different relative humidity. Data are presented up to the last time point where all volunteers were still exercising in each trial along with the value recorded at exhaustion (mean and group standard deviation)

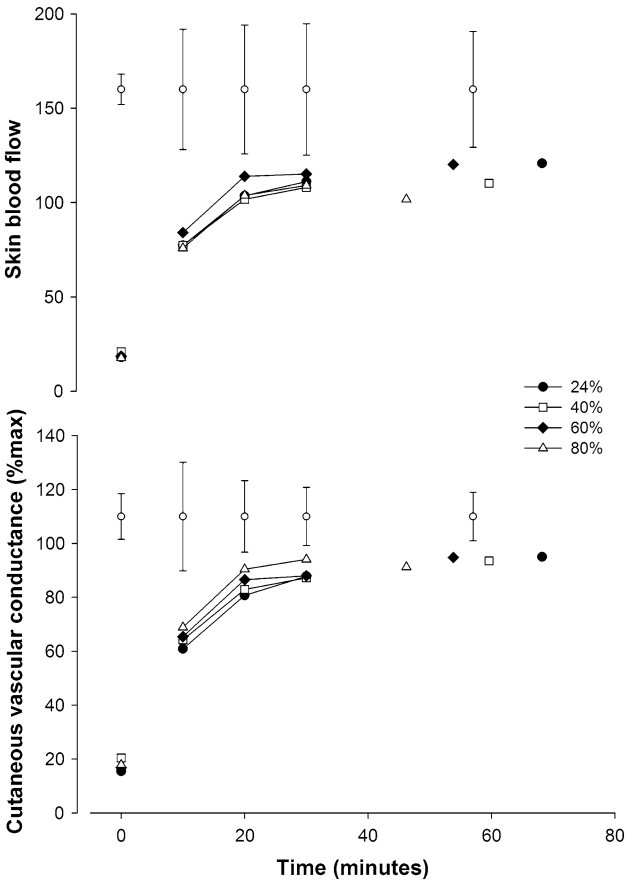

Fig. 4 Changes in skin blood flow (*upper panel*) and cutaneous vascular conductance (*lower panel*) in response to exercise. Data are presented up to the last time point where all volunteers were still exercising in each trial along with the value recorded at exhaustion (mean and group standard deviation)

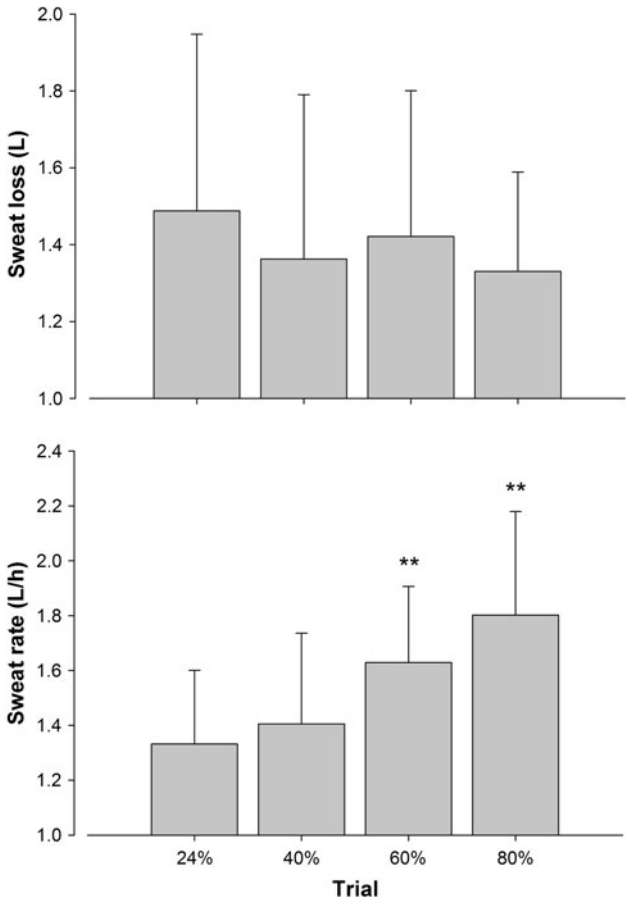

Fig. 5 Total sweat loss (*top*) and the calculated sweat rate (*bottom*) in each experimental trial. $^{**}P < 0.01$ denotes a significant difference from the 24% trial

1.36 ± 0.43 L; 60% 1.42 ± 0.38 L; 80% 1.33 ± 0.26 L; $P = 0.345$; Fig. 5b).

Both the subjective RPE ($P = 0.001$) and the rating of thermal stress ($P < 0.001$) were affected by the humidity of the environment (Table 1). RPE was not significantly different at 40% rh ($P = 0.384$) or at 60% ($P = 0.065$) from that measured at 24%, but was higher at 80% ($P = 0.008$) than at 24%. Thermal stress was not higher at 40% rh than at 24% ($P = 0.085$), but was higher at 60% ($P = 0.003$) and at 80% ($P < 0.001$) than at 24%. A mixed-effects model was employed to examine the relationship between skin and core temperature with perceived feelings of exertion and thermal stress. There was no significant association apparent between core or skin temperature after 20 or 30 min of exercise, perceived feelings of exertion or thermal stress reported at the same time points and the time to exhaustion attained.

Blood volume ($P = 0.009$) and plasma volume ($P = 0.006$) decreased over time, but the humidity of the environment did not affect these changes. Serum osmolality ($P = 0.012$), serum sodium concentration ($P = 0.002$) and serum potassium concentration ($P = 0.038$) all increased

Table 1 Rating of perceived exertion (RPE) and perceived thermal stress during exercise in different relative humidity

	Pre-ex	10	20	30	40	Exh
Rating of perceived exertion (RPE)						
24%	–	12 ± 2	14 ± 2	15 ± 2	16 ± 2	20 ± 0
40%	–	13 ± 1	15 ± 1	16 ± 1	17 ± 1	20 ± 1
60%	–	14 ± 1	15 ± 1	16 ± 1	–	20 ± 0
80%	–	14 ± 1	15 ± 2	17 ± 2	–	20 ± 0
Perceived thermal stress (0 neutral; 10 unbearable heat)						
24%	0 ± 1	4 ± 1	4 ± 1	5 ± 1	5 ± 1	6 ± 1
40%	0 ± 2	4 ± 1	5 ± 1	5 ± 1	6 ± 1	8 ± 1
60%	0 ± 1	4 ± 1	5 ± 1	6 ± 1	–	8 ± 1
80%	0 ± 1	5 ± 1	6 ± 1	8 ± 1	–	9 ± 1

Table 2 The effect exercise and environmental relative humidity on serum electrolyte concentration, serum osmolality, blood glucose concentration and percentage change in plasma volume

	Pre-ex	15 min	30 min	Exh
Serum sodium (mmol/L)				
24%	142 ± 2	144 ± 2	145 ± 2	147 ± 1
40%	142 ± 1	144 ± 3	144 ± 2	146 ± 2
60%	142 ± 1	143 ± 1	145 ± 2	147 ± 2
80%	141 ± 1	144 ± 2	144 ± 1	146 ± 1
Serum potassium (mmol/L)				
24%	4.6 ± 0.5	5.3 ± 0.7	5.3 ± 0.6	5.3 ± 0.5
40%	4.7 ± 0.3	5.2 ± 0.3	5.5 ± 0.4	5.6 ± 0.4
60%	4.7 ± 0.3	5.1 ± 0.2	5.4 ± 0.3	5.6 ± 0.3
80%	4.6 ± 0.3	5.3 ± 0.4	5.6 ± 0.3	5.6 ± 0.3
Serum osmolality (mosmol/kg)				
24%	287 ± 2	296 ± 2	297 ± 2	299 ± 2
40%	287 ± 2	295 ± 2	297 ± 2	298 ± 3
60%	287 ± 3	294 ± 4	296 ± 3	297 ± 1
80%	287 ± 2	296 ± 2	296 ± 1	297 ± 1
Blood glucose (mmol/L)				
24%	4.1 ± 0.2	3.7 ± 0.2	3.9 ± 0.3	5.0 ± 0.4
40%	4.1 ± 0.1	3.9 ± 0.2	4.1 ± 0.1	5.0 ± 0.5
60%	4.2 ± 0.1	3.9 ± 0.4	4.3 ± 0.5	5.2 ± 0.5
80%	4.3 ± 0.2	4.1 ± 0.3	4.6 ± 0.3	5.3 ± 0.9
Δ Plasma volume (%)				
24%	0.0 ± 0.0	11.9 ± 2.0	12.9 ± 2.2	12.9 ± 2.8
40%	0.0 ± 0.0	12.4 ± 2.7	13.1 ± 2.0	12.5 ± 2.0
60%	0.0 ± 0.0	11.2 ± 2.8	11.9 ± 3.8	13.1 ± 2.8
80%	0.0 ± 0.0	12.3 ± 2.2	13.0 ± 2.4	13.4 ± 3.0

over time, but no treatment effects were apparent (Table 2). The blood glucose concentration did not change over time ($P = 0.214$) and no differences between trials were apparent ($P = 0.149$; Table 2).

Discussion

The results of the present study demonstrate that, for active males who were not specifically heat acclimated, exercise capacity at moderate intensity in a warm environment was progressively impaired as the relative humidity increased. Although this response was not statistically significant when the humidity increased from 24 to 40%, the mean time to fatigue was 8.6 ± 8.6 min less at 40% rh than recorded at 24%: this represents a mean reduction in exercise time of 12.3 ± 11.8%. There were more substantial, and statistically significant, reductions in exercise capacity at the higher levels of humidity (Fig. 1).

While differences in humidity have been factored into studies investigating the relationship between environmental conditions and competitive race performance in athletes (Ely et al. 2007; McCann and Adams 1997), the specific influence of relative humidity on performance and on the physiological response to exercise remains largely unexplored. In these investigations, WBGT, a widely accepted index for the assessment of heat stress, was employed to provide a composite measure of ambient temperature, humidity and solar load. An average WBGT was determined for each event, meaning that changes in ambient temperature, humidity or wind speed/direction occurring during the course of the race were not accounted for. It should also be recognised that, while WBGT is a widely employed tool, it has limitations when predicting the physiological response to a particular environmental condition and should be used with caution in practical settings (Parsons 2003): in humid environments, WBGT correlates well with skin temperature, but this relationship does not appear to hold for other physiological variables of interest, including rectal temperature, heart rate and sweat rate.

Studies of prolonged exercise undertaken in a temperate environment have suggested that the cause of fatigue is primarily related to the depletion of glycogen in the exercising muscles, coupled with a fall in circulating blood glucose levels (Saltin and Karlsson 1971). At higher ambient temperatures, there is a considerable decrease in exercise time to fatigue, but a substantial quantity of glycogen has been measured in the muscle at the point of fatigue, suggesting that CHO availability is not a limiting factor (Parkin et al. 1999). In the present study, there was a significant difference in the calculated total carbohydrate oxidation between trials, resulting from the marked reduction in exercise time as the environmental humidity increased. During the 24% trial 246 ± 63 g of CHO was oxidised, but this was reduced to 179 ± 24 g in the 80% trial, suggesting that adequate carbohydrate reserves should have been available at the point of fatigue at the higher levels of humidity. Despite evidence suggesting that substrate availability is not limiting during exercise in the heat, several studies have reported improvements in performance following the ingestion of a high CHO diet (Pitsiladis and Maughan 1999) and with the use of CHO supplements before and during exercise (Carter et al. 2003; Galloway and Maughan 2000). Notwithstanding these observations, CHO availability should not be limiting.

This suggests that there must be an alternative explanation for the termination of exercise in the heat, and the obvious candidates are related to the elevation in body temperature that occurs when strenuous exercise is performed in hot environments. Nielsen (1938) demonstrated that humans can regulate their core temperature during moderate intensity exercise, independent of the climatic conditions over a relatively wide range of temperatures (5–30°C). This observation was apparent in the present study: there was no statistically significant effect of environment on core temperature during exercise, and there was no difference in core temperature between trials at the point of fatigue in spite of the different exercise times. This latter observation suggests a faster rate of rise of core temperature when humidity was higher. There was also a faster rise in heat storage with higher humidity, with a 36 ± 9% difference between the 24 and 80% trials: given the same power output and the absence of any evidence of a difference in efficiency, this is consistent with a reduced rate of heat loss at the higher levels of humidity.

It has been suggested that there is a critical core temperature above which exercise performance cannot be sustained because of a reduced central neural drive to the exercising muscles. Nielsen et al. (1993) reported that fatigue in a cycling exercise test at 60% VO_{2max} in dry heat (40°C and 10% rh) occurred at the same core temperature (39.7 ± 0.2°C) after acclimation as before, though the exercise time was longer: they concluded that "Physical endurance in hot, dry environments appears to be limited by attainment of a critical level of core temperature, perhaps due to temperature reducing motivation". Cabanac et al. (1968) had demonstrated earlier, in rabbits, the existence of neurons in the brain stem whose firing rate was sensitive to the local temperature within the range of 32–42°C, so there is a mechanism in place for responding to brain temperature. In addition, Fuller et al. (1998) reported that rodents ceased exercise when brain temperature exceeded 40°C, with exercise time altered when the starting core temperature was manipulated by exposure to different environments. While the premise of a 'critical core temperature' has persisted for a number of years now, several studies have observed differences in the endpoint core temperature related to physical fitness, degree of heat acclimation or hydration status (Cheung 2010; Otani et al. 2006). The core temperatures reached at fatigue in the present study were not high—about 39°C, with the highest individual values of 39.5°C. This is lower that the values commonly observed after completion of marathon runs (Byrne et al. 2006; Pugh et al. 1967), but is very similar to the situation that is observed when restriction of evaporative heat loss is achieved by wearing of clothing that is impermeable to water vapour (Sawka et al. 2001). However, there was no difference in core temperature at fatigue between the high and low humidity trials. This may reflect the particular combination of ambient temperature and exercise intensity used in this study, but also the moderate fitness status of the subjects.

Cheuvront et al. (2010) have recently argued that fatigue in endurance exercise under conditions of thermal stress is

not mediated by mechanisms residing within the CNS, but can be accounted for by the increased cardiovascular strain. Interestingly, these authors have shifted focus from core temperature to skin temperature as the key factor in the fatigue process. During exercise in the heat, a large part of the available blood volume is directed to the skin; this can lead to a reduction in blood flow to the exercising muscles (González-Alonso et al. 2008) and also from the brain (Nybo and Nielsen 2001), thereby reducing oxygen delivery to and removal of heat from these tissues. This model suggests that a high skin temperature is necessary to maintain heat loss to the environment in conditions of thermal stress and that a high core temperature is therefore necessary to maintain the core-to-skin temperature gradient. It has long been recognised that skin temperature has an important role in thermal perception and in conscious decisions as to preferred environments (Cabanac et al. 1971; Cheung 2010). Schlader et al. (2009) have recently reported that skin temperature and thermal comfort were significant predictors of subjects' conscious decision to move from a cold environment to a hot environment or from a hot environment to a cold environment, but that rectal temperature was not an important factor. They also reported that this behavioural response was more precise when exposed to heat rather than to cold. Given these observations, it might be reasonable to expect a clear relationship between core or skin temperature at a particular time point during exercise and the corresponding individual perception of effort, but the present study failed to identify any clear association between measures of body temperature and perceived exertion. While these data do not support this hypothesis, the decision to seek respite from a high thermal stress may, perhaps, be seen as analogous to the decision to terminate exercise.

The selection of an appropriate test to measure exercise performance has generated considerable controversy in recent years. Constant power tests to volitional exhaustion have been employed to examine the influence of various interventions on performance. This method of testing has been criticised for a lack of ecological validity and poor test–retest reliability, but recent reports have highlighted similar errors of measurement when changes in performance are normalised across tests. Amann et al. (2008) demonstrated that time to exhaustion and time trial protocols display a similar sensitivity, brought about by larger effects on time to exhaustion in response to an intervention that are typically observed in time trial protocols compensating for the potentially larger test–retest variability. In the present study, an obvious limitation of time trial-type test would be the difficulty in comparing the effect of the intervention on physiological responses to exercise because of fluctuating power outputs.

In conclusion, the present study demonstrates a progressive reduction in exercise capacity in the heat as the environmental relative humidity rises. Early fatigue in the higher humidity trials was accompanied by a faster rate of rise in core temperature and a greater weighted mean skin temperature, with no differences in heart rate, skin blood flow or the metabolic response to exercise apparent. Ultimately, the decision to terminate exercise is a conscious one, dictated by the subjective perception of the balance between the desire to continue exercise and the desire to seek respite by stopping, as described by Cabanac (2006). The neurobiological response underpinning this decision is unclear at present, but evidence supporting the role of the brain in fatigue comes in part from the effect on exercise performance of pharmacological agents that affect brain serotoninergic (Wilson and Maughan 1992) and dopaminergic (Watson et al. 2005) function without significant metabolic or cardiovascular actions. In addition, although cells and tissues may be resistant to failure at the temperatures achieved during strenuous exercise in the heat, there is evidence of functional changes, including a progressive loss of blood brain barrier integrity (Kiyatkin and Sharma 2009) at relatively modest temperatures (>38°C). It seems likely that, as Bainbridge (1919) pointed out, the sensation of fatigue, while central in origin, is strongly influenced by signals arising in the periphery, including feedback from both thermal and non-thermal pathways.

References

Amann M, Hopkins WG, Marcora SM (2008) Similar sensitivity of time to exhaustion and time-trial time to changes in endurance. Med Sci Sports Exerc 40:574–578

Armstrong LE (2000) Performing in extreme environments. Human Kinetics, Champaign, p 21

Backx K, McNaughton L, Crickmore L, Palmer G, Carlisle A (2000) Effects of differing heat and humidity on the performance and recovery from multiple high intensity intermittent exercise bouts. Int J Sports Med 21:400–405

Bainbridge FA (1919) The physiology of muscular exercise. Longmans, Green and Co, London, p 177

Borg GA (1982) Psychophysical bases of perceived exertion. Med Sci Sports Exerc 14:377–381

Byrne C, Lee JW, Chew SN, Lim CL, Tan EM (2006) Continuous thermoregulatory response to mass-participation distance running in heat. Med Sci Sports Exerc 38:803–810

Cabanac M (2006) Sensory pleasure optimizes muscular work. Clin Invest Med 29:110–116

Cabanac M, Stolwijk JAJ, Hardy JD (1968) Effect of temperature and pyrogens on single-unit activity in the rabbit's brain stem. J Appl Physiol 24:645–652

Cabanac M, Massonet B, Belaiche R (1971) Preferred skin temperature as a function of internal and mean skin temperature. J Appl Physiol 33:699–703

Carter JM, Jeukendrup AE, Mundel T, Jones DA (2003) Carbohydrate supplementation improves moderate and high-intensity exercise in the heat. Pflugers Arch 446:211–219

Cheung SC (2010) Interconnections between thermal perception and exercise capacity in the heat. Scand J Med Sci Sports 20(Suppl 3):53–59

Cheuvront SN, Kenefick RW, Montain SJ, Sawka MN (2010) Mechanisms of aerobic performance impairment with heat stress and dehydration. J Appl Physiol 109:1989–1995

Dill DB, Costill DL (1974) Calculation of percentage changes in volumes of blood, plasma, and red cells in dehydration. J Appl Physiol 37:247–248

Dill DB, Edwards HT, Bauer PS, Levenson EJ (1931) Physical performance in relation to external temperature. Eur J Appl Physiol Occup Physiol 4:508–518

Ely MR, Cheuvront SN, Roberts WO, Montain SJ (2007) Impact of weather on marathon-running performance. Med Sci Sports Exerc 39:487–493

Fuller A, Carter RN, Mitchell D (1998) Brain and abdominal temperatures at fatigue in rats exercising in the heat. J Appl Physiol 84:877–883

Galloway SDR, Maughan RJ (1997) Effects of ambient temperature on the capacity to perform prolonged cycle exercise in man. Med Sci Sports Exerc 29:1240–1249

Galloway SDR, Maughan RJ (2000) The effects of substrate and fluid provision on thermoregulatory and metabolic responses to prolonged exercise in a hot environment. J Sports Sci 18:339–351

González-Alonso J, Crandall CG, Johnson JM (2008) The cardiovascular challenge of exercising in the heat. J Physiol 586:45–53

Hardy JD (1970) Thermal comfort: skin temperature and physiological thermoregulation. In: Hardy JD, Gagge AP, Stolwijk JAJ (eds) Physiological and behavioral temperature regulation. Springfield, III, Thomas, pp 856–873

Kiyatkin EA, Sharma HS (2009) Permeability of the blood-brain barrier depends on brain temperature. Neuroscience 161:926–939

Lange KL, Little RJA, Taylor JMG (1989) Robust statistical modeling using the t-distribution. J Am Stat Assoc 84:881–896

Maughan RJ, Shirreffs SM, Leiper JB (2007) Errors in the estimation of sweat loss and changes in hydration status from changes in body mass during exercise. J Sports Sci 25:797–804

McCann DJ, Adams WC (1997) Wet-bulb globe temperature index and performance in competitive distance runners. Med Sci Sports Exerc 29:955–961

Nielsen M (1938) Die regulation der korpertemperatur bei muskelarbeit. Skand Arch Physiol 79:193–230

Nielsen B, Hales JR, Strange S, Christensen NJ, Warberg J, Saltin B (1993) Human circulatory and thermoregulatory adaptations with heat acclimation and exercise in a hot, dry environment. J Physiol 460:467–485

Niwa K, Nakayama T (1978) Thermoregulation during exercise in high humidity environments (in Japanese with English abstract) Jpn. J Phys Fit Sport 27:11–18

Nybo L, Nielsen B (2001) Middle cerebral artery blood velocity is reduced with hyperthermia during prolonged exercise in humans. J Physiol 534:279–286

Otani H, Kaya M, Tsujita J, Hori K, Hori S (2006) Low levels of hypohydration and endurance capacity during heavy exercise in untrained individuals. J Therm Biol 31:186–193

Parkin JM, Carey MF, Zhao S, Febbraio MA (1999) Effect of ambient temperature on human skeletal muscle metabolism during fatiguing submaximal exercise. J Appl Physiol 86:902–908

Parsons KC (2003) Human thermal environments (second edition). Taylor and Francis, London, pp 284–286

Pitsiladis Y, Maughan RJ (1999) The effects of exercise and diet manipulation on the capacity to perform prolonged exercise in the heat and cold in trained humans. J Physiol 517:919–930

Pugh LGCE, Corbett JL, Johnson RH (1967) Rectal temperatures, weight losses and sweat rates in marathon running. J Appl Physiol 23:347–352

Ramanathan LM (1964) A new weighting system for mean surface temperature of the human body. J Appl Physiol 19:531–532

Robinson S, Turrell ES, Gerking SD (1945) Physiologically equivalent conditions of air temperature and humidity. Am J Physiol 143:21–32

Saltin B, Karlsson J (1971) Muscle glycogen utilization during work of different intensities. In: Pernow B, Saltin B (eds) Muscle metabolism during exercise. Plenum Press, New York, pp 289–300

Sawka MN, Latzka WA, Montain SJ, Cadarette BS, Kolka MA, Kraning KK, Gonzalez RR (2001) Physiologic tolerance to uncompensable heat: intermittent exercise, field vs. laboratory. Med Sci Sports Exerc 33:422–430

Schlader ZJ, Prange HD, Mickleborough TD, Stager JM (2009) Characteristics of the control of human thermoregulatory behavior. Physiol Behav 98:557–562

Stensrud T, Berntsen S, Carlsen KH (2006) Humidity influences exercise capacity in subjects with exercise-induced bronchoconstriction (EIB). Respir Med 100:1633–1641

Tatterson AJ, Hahn AG, Martin DT, Febbraio MA (2000) Effects of heat stress on physiological responses and exercise performance in elite cyclists. J Sci Med Sport 3:186–193

Watson P, Hasegawa H, Roelands B, Piacentini MF, Looverie R, Meeusen R (2005) Acute dopamine/noradrenaline reuptake inhibition enhances exercise performance in warm, but not temperate conditions. J Physiol 565:873–883

Webb P (1995) The physiology of heat regulation. Am J Physiol 268:R838–R850

Wilson WM, Maughan RJ (1992) A role for serotonin in the genesis of fatigue in man: administration of a 5-hydroxytryptamine reuptake inhibitor (Paroxetine) reduces the capacity to perform prolonged exercise. Exp Physiol 77:921–924