Introduction

The demand on the circulatory system to supply adequate oxygenated blood to the working muscle during exercise is further challenged in heat stress condition (Gonzalez-Alanso, 2008). The circulatory system will have to meet the dual demand of supplying adequate oxygenated blood to the working muscle and thermoregulatory demand of increased skin blood flow when exercising in heat stress condition (Thompson, 2006; Gonzalez-Alanso *et al.*, 2008). These combined demands for blood flow may result in competition for available cardiac output, which ultimately influence exercise performance.

During prolonged exercise, a time dependent drift in the cardiovascular (CV) system described by Rowell as "cardiovascular drift" (Rowell, 1986) has been well documented (Wingo et al., 2012; Periard et al., 2010; Thompson, 2006; Fritzsche *et al.*, 1999 and Arngrimsson *et al.*, 2003). According to Rowell (1986), CV drift is "a downward drift in central venous pressure, stroke volume, pulmonary and systemic arterial pressure and thoracic or central blood volume, while at the same time a rise in heart rate maintains nearly constant cardiac output." The classic hypothesis provided by Rowell (1986) for the occurrence of CV drift centres on the proposition that there is a progressive increase in cutaneous blood flow as body temperature rises. Additional heat will be transferred from the body core to the skin for dissipation via overall conduction that is made possible by an increased cutaneous blood flow. Although an increase in cutaneous blood flow is essential for the thermoregulatory system, it however, poses a threat to the cardiovascular system. This is due to the reduced ventricular filling, end diastolic volume and stroke volume as cardiac output is being diverted through to the compliant skin circulation (Rowell, 1986; Gonzalez-Alanso *et al.*, 2008; Johnson, 2010). Thus, under a heat stress environment, a continuing increase in heart rate to offset the decline in stroke volume in order to maintain the required cardiac output (Q) has been extensively reported (Thompson, 2006; Gonzalez-Alanso *et al.*, 2008; Johnson, 2010; Ely *et al.*, 2010; Kenefick *et al.*, 2010).

Environmental conditions have been shown to affect the magnitude of CV drift. Nadel *et al.* (1979) systematically increased ambient temperature and observed a greater decline in SV in a 36^0C compared with a 26^0C and 20^0C environment. It is now well established that warm to hot environmental conditions can have a profound effect on eliciting a cardiovascular drift. To the author's knowledge, the magnitude of CV drift during prolonged high intensity exercise in humid conditions has not been well established. To date, limited research to show that more humid

conditions cause a significant elevation in core temperature and heart rate during low to moderate (40-50% $\dot{V}O_{2\,max}$) intensity of exercise (Kamon & Belding, 1971; Nielsen *et al.,* 1997). However, no systematic investigation has been undertaken during prolonged intense exercise. The purpose of this study was to examine impact of a systematic increase of humidity on the cardiovascular drift during intense prolonged running exercise.

Method

Participants

Eleven well-trained non-heat-acclimated male athletes who regularly participated in middle and long distance running events and volunteered as research participants (mean ± SD: Age = 30.1 ± 4yr; height = 180.3 ± 5.8 cm; weight = 71.6 ± 5.9 kg; $\dot{V}O_{2\,max}$= 61.31 ± 5.53 mL/kg/min). Participants were briefed of the experimental design and testing protocols and provided informed written consent to participate. The experimental protocol was approved by The University of Sydney Human Ethics Committee (Ref. No: 99/05/46) and conformed to the current Declaration of Helsinki guidelines.

Environmental testing conditions

All experiments were conducted in a purpose-built Climate Chamber. The present study simulated three hot-humid conditions of a high humidity (HH=71%rh), neutral humidity (NH=43%rh) and low humidity (LH=26.2% rh) with the ambient temperature held at a constant 31^0C within each of the trials. The Climate Chamber pre-set conditions (dry bulb temperature, wet bulb temperature and air velocity) were checked with actual measurements taken prior to the start of each experiment.

Preliminary procedures

Prior to taking part in this study each participant underwent a familiarization phase to demonstrate the exercise protocol, equipment and measurement procedures used in the experiment. This was followed with preliminary testing session where anthropometric measures including height, body weight, body composition and body surface area was determined. Body surface area was calculated using the method of DuBois and DuBois (1916). Percent body fat was measured using the hydrodensiotometry underwater weighing technique based from the equation of Siri (1961). Each participant then performed a running economy test and a maximal exercise test to determine maximum oxygen uptake ($\dot{V}O_{2\,max}$) conducted in a thermoneutral environment (21^0C, 40% relative humidity). The running economy test required each participant to run at four submaximal steady state velocities of 10, 12, 14 and 16

kilometers/hour (km/hr) respectively over four minute stages at each velocity. Respiratory gas exchange and heart rate measurements were recorded over the final minute of each stage. On completion of the submaximal steady state running, participants engaged in an active recovery phase, where they walked at a speed of 5 km/hr for five minutes. This was followed by an exercise test to determine $\dot{V}O_{2\,max}$. In this test, each participant ran at a constant speed of 12 km/hr with the treadmill gradient increased 2% every two minutes until volitional fatigue. Oxygen consumption and heart rate over the final 30 seconds of exercise were recorded as the $\dot{V}O_{2\,max}$ and maximal heart rate (HR_{max}). A linear regression line was plotted for submaximal steady state $\dot{V}O_2$ and treadmill velocity to determine the running speed of 70% $\dot{V}O_{2\,max}$ for each subsequent testing session.

Experimental procedures
Participants were randomly assigned to complete 60 minutes submaximal running exercise at an intensity of 70% $\dot{V}O_{2\,max}$ on a motor driven treadmill at across three different humidity levels of LH, MH and HH, respectively. Each test session was separated by 3-5 days to minimize acclimation effects as well to provide adequate recovery to the participants. Prior to the visit to the laboratory for testing, each participant was instructed to refrain from heavy exercise and alcohol consumption the day before testing and to avoid caffeine consumption for 12 hours before the test. They were also requested to maintain a similar dietary intake the day before each of the one-hour exercise tests. To ensure that the participants were euhydrated at the onset of exercise, they were asked to ingest an amount of water (6 ml of water per kg lean body mass) at two-hours intervals (excluding when asleep) a day before the test and the morning of testing. Participants were also asked to maintain their physical activity level in the same way throughout the experiment period.

On the day of testing, upon arrival at the laboratory (~60 minutes prior to the testing session), participants emptied their bladder, changed into their running shorts and inserted a disposable rectal probe 12 cm beyond the anal sphincter which would remained in place throughout the entire test. During each trial subjects were only allowed to wear running shorts, socks and running shoes. Participants then sat resting in a thermoneutral room (20^0C and 40%rh) for the remaining of the time while being instrumented with a heart rate monitor (Polar Heart Rate) and skin temperature thermistors at four different sites (left shoulder, left chest, right mid-thigh and right mid-shin).

Measurements

Temperature measurements

Rectal temperature (T_{re}) was measured and recorded on a portable data logger (T-logger; University of Sydney) as an index of core temperature by a thermistor probe (YSI 400 series; Mallinckrodt Medical, Kansas City, MO) inserted 12 cm beyond the anal sphincter. Skin temperature was measured at four different sites using thermistor probes (YSI 409 Series). Rectal and skin temperatures were sampled at rest and at 10 minutes intervals during exercise. Mean skin temperature ($\overline{T}_{sk}$) was calculated using the equation of Ramanathan (1964). The rate of change (increase or decrease) in both T_{re} and $\overline{T}_{sk}$ during exercise from the initial resting level was expressed as delta T_{re} (ΔT_{re}) and delta $\overline{T}_{sk}$ ($\Delta \overline{T}_{sk}$), respectively at 10 minutes intervals. Both rectal and skin thermistor probes used in the study were calibrated before and after the study in a water bath with temperature ranging from 15^0C to 50^0C with a calculated accuracy of ±0.05 and ±0.01, respectively.

Sweating, RPE and Thermal Comfort measurements: Body mass changes were evaluated within the first and last 30 minutes of submaximal exercise to determine sweat rate with correction for respiratory moisture loss, urine production, sweat trapped in clothing corrected for moisture loss due to the exchange of O_2 and CO_2 as described by Nadel *et al.* (1979). RPE were recorded using the Borg 20 points scale (Borg, 1982). A thermal comfort (TC) scale developed by the American Society of Heating, Refrigeration and Air-Conditioning (1966) (ASHRAE) was used for the participants to provide a participative indication of their perception or sensations of thermal comfort or otherwise. Both RPE and TC ratings were sampled during exercise at 10 minutes intervals. Tissue heat conductance (K) was derived from the method of Davies (1979) based on the following formula; $K = H_{sk} / ((T_{re} - \overline{T}_{sk}) * A_D)^{-1} (W.m^{2.0}C^{-1})$ where H_{sk} is the heat dissipated from the skin (E + R + C), T_{re} is rectal temperature, T_{sk} is mean skin temperature and A_D is the DuBois body surface area in m^{-2}.

Cardiorespiratory measurements

Expired respiratory gas were obtained using the Douglas bag method and analysed for fractions of Oxygen (O_2) and carbon dioxide (CO_2) concentration using gas sensors and analysers (O_2 analyser, Ametek S-3A/I and CO_2 analysers, Ametek CD-3A, Applied Electochemisrty Ametek Inc., Thermox Instruments Division, USA). The gas analysers were calibrated with known calibration gases prior to each analysis. Cardiac output ($\dot{Q}$) was determined every ten minutes by the CO_2 rebreathing method (Collier, 1956). Heart rate (HR) was continuously

monitored and recorded at every ten minutes intervals. Stroke volume (SV) was calculated using the Fick equation. A good test-retest repeatability was demonstrated for the measurements of $\dot{Q}$ during a pilot study involving a graded submaximal exercise with an interclass correlation coefficient (ICC) was 0.996.

Statistical Analysis

All data are presented as means ± SD, unless stated otherwise. A two-way (time x trial) repeated measure analysis of variance (ANOVA) was used to test the significance between and within treatments between the three humidity levels. A Huynh-Feldt correction was applied to adjust the degrees of freedom when the test of sphericity was significant ($p < 0.05$). The level of significance was set at a p-value of less than 0.05. An analysis of power using conventional (0.05) and (0.20) parameters indicated that eleven participants would provide sufficient power to detect meaningful differences in all of the variables tested (Tran, 1997).

Results

Temperature responses

Rectal temperature at the completion of exercise was systematically higher with rising humidity. T_{re} at the completion of 60 minutes exercise was significantly higher in the HH compared with the NH and LH environment ($39.5^0C \pm 0.5$ vs. $39.1^0C \pm 0.5$ and $38.9^0C \pm 0.5$, respectively, $p < 0.05$). Figure 1 demonstrated a greater rate of rise in ΔT_{re} during exercise in the HH condition with a significant difference detected from 40 minutes of exercise onwards. Similarly, $\overline{T}_{sk}$ at the end of exercise was significantly higher in the HH as compared with the NH and LH environment ($33.21^0C \pm 0.42$ vs. $32.43^0C \pm 0.39$ and $31.72^0C \pm 0.47$, respectively, $p < 0.05$). $\overline{T}_{sk}$ remained elevated during exercise in the HH condition and was significantly higher compared to the LH (Figure 2). Within each humidity conditions, $\overline{T}_{sk}$ remained stable within the last 30 minutes of exercise as no significant differences were detected (Figure 2).
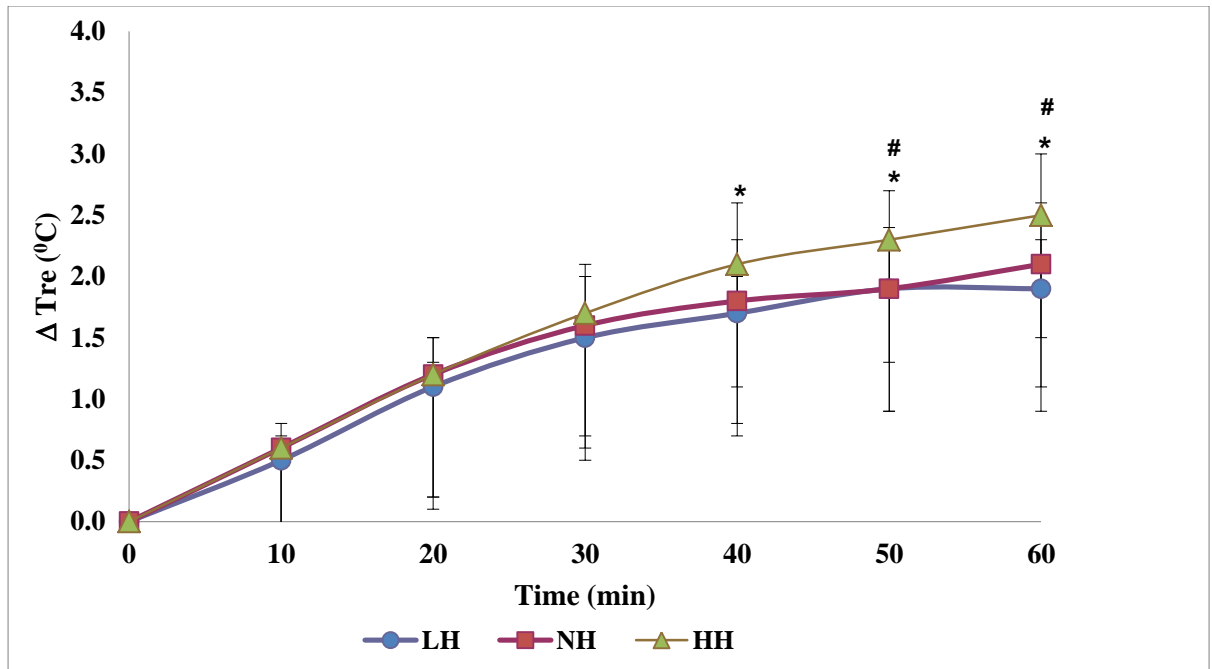
* significantly higher in the HH condition than the LH condition ($p < 0.05$)
# significantly higher in the NH condition than the LH condition ($p < 0.05$)

Figure 1: Changes in rectal temperature (ΔT_{re}) during exercise across the different levels of relative humidity ($n=11$).
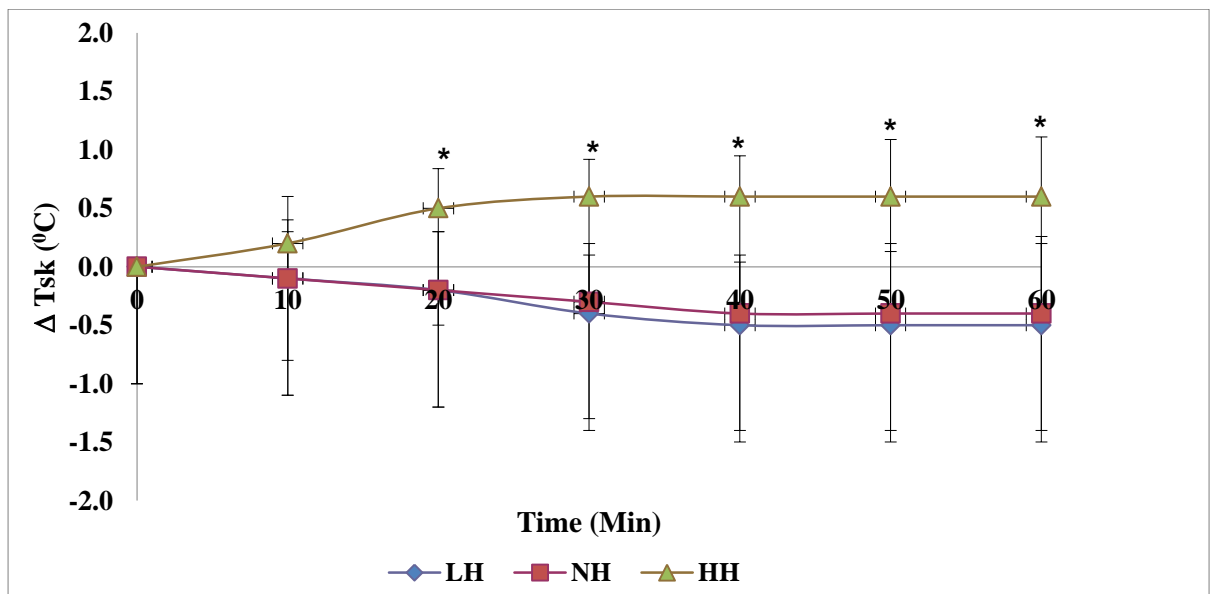
* significantly higher in the HH condition than the LH condition ($p < 0.05$)

Figure 2: Changes in mean skin temperature ($\Delta \overline{T}_{sk}$) during exercise across the different levels of relative humidity ($n=11$).

The rate of sweating during the first 30 minutes of exercise was not significantly different across the LH, NH and HH conditions, espective (20.8± 4.2 g.min^{-1}, 20.4± 4.6 g.min^{-1}, and 19.4± 3.9 g.min^{-1}). Similarly, the sweating rate within the last 30 minutes of exercise in the HH and NH was not significantly different compared with the LH conditions (27.6± 4.9 g.min^{-1}, 26.5± 4.5 g.min^{-1} vs. 28.3± 5.5 g.min^{-1}, respectively).

RPE and Thermal Comfort Responses
The RPE response at the end of exercise as exercise was perceived as "hard" to perform in the HH compared to "somewhat hard" to perform in LH environment (60 minutes RPE = 15 ± 2 vs. 13 ± 3, HH vs. LH, respectively, $p < 0.05$). This response was consistent when comparing between the NH and LH environment (60 minutes RPE = 14 ± 2 vs. 13 ± 3, NH vs. LH, respectively, $p < 0.05$).

Table 1: Cardiorespiratory measurements during exercise in the Low, Neutral and High Humidity Levels ($n = 11$).

Relative Humidity	Time (Minutes)	VO₂ (L.min^{-1})	Q (L.min^{-1})	HR (bpm)	% Maximal HR	SV (ml.min^{-1})	a-VO₂ diff (ml/100ml^{-1})
Low Humidity (LH)	10	3.02 ± 0.25	20.7 ± 1.4	144 ± 13	78 ± 4	144.6 ± 13.2	14.5 ± 0.5
	30	3.05 ± 0.27	20.7 ± 1.8	153 ± 15	83 ± 5	135.8 ± 15.1	14.8 ± 0.9
	60	3.11 ± 0.30	20.3 ± 2.0	160 ± 15	86 ± 5	127.0 ± 14.1	15.4 ± 0.9
Neutral Humidity (NH)	10	3.10 ± 0.21	21.2 ± 1.5	149 ± 13	80 ± 4	143.1 ± 10.9	14.6 ± 0.3
	30	3.12 ± 0.26	20.8 ± 2.2	156 ± 14	84 ± 5	133.3 ± 12.2	15.0 ± 0.7
	60	3.22 ± 0.25	19.9 ± 2.2	166*± 16	90 ± 5	119.7 ± 10.2	16.3 ± 1.2
High Humidity (HH)	10	3.11 ± 0.27	20.8 ± 1.4	149 ± 12	80 ± 4	140.6 ± 11.6	14.9 ± 0.8
	30	3.08 ± 0.32	20.6 ± 2.3	159 ± 14	86 ± 5	129.9 ± 12.9	15.0 ± 1.0
	60	3.17 ± 0.31	20.0 ± 2.5	170*± 13	92* ± 5	117.2*± 10.1	16.0 ± 1.4

Data are shown as mean ± SD, * significantly ($p < 0.05$) different from the LH condition

The calculated tissue conductance (K), which is an indicator of skin blood flow, was significantly higher in the HH and NH as compared with the LH environment (60 minutes K = 154.1 ± 13.6 W.m^{-2} and 146.6 ± 19.3 W.m^{-2} vs. 138.3 ± 17.2 W.m^{-2}, respectively $p < 0.05$).

Cardiorespiratory responses

Cardiac output was maintained during exercise with no significant difference detected across the different conditions. At the end of exercise, HR expressed as percent of HR_{max} was significantly higher in the HH and NH environment compared with the LH environment (92% $\pm$ 5 and 90% $\pm$ 5 vs. 86% $\pm$ 5, respectively, $p < 0.05$). A greater magnitude in CV drift was observed in the HH condition where HR drifted upwards by 21 beats.min^{-1} within the last 50 minutes of exercise (from 10 to 60 minutes) following the first ten minutes of exercise where a steady state HR was initially observed. The upward drift in HR in the NH and LH was 17 beats.min^{-1} and 16 beats.min^{-1}, respectively. Data presented in Table 1 demonstrated the different rate of rise in HR across three different humidity conditions. Regression analysis of HR measures at 60 minutes of exercise indicated a strong correlation between the increase in HR and rising in T_{re} ($r^2 = 0.88$). Stroke volume was systematically reduced during exercise and was significantly lower at the end of exercise in the HH compared with the LH environment (117.2 $\pm$ 10.1 ml.min^{-1} vs. 127.0 $\pm$ 14.1 ml.min^{-1}, $p < 0.05$). The rate of oxygen extraction reflected from the a-$\dot{V}O_2$ was not significantly affected across the three different conditions.

Discussion

The purpose of the present study was to evaluate the impact of rising humidity level on cardiovascular strain during prolonged intense exercise. To our knowledge, limited numbers of studies that examined the impact of humidity on cardiovascular strain involving well-trained distance runners. The main finding from this experiment was an increase in circulatory stress during exercise in the higher humidity. At the end of 60 minutes exercise, HR was 92% of HR_{max} in the HH as compared to 90% and 86% of HR_{max} in the NH and LH, respectively. The increase in HR during exercise in the present study was correlated with an increase in thermoregulatory stress ($r^2 = 0.88$). Our current observation was consistent with the trend previously reported of an elevated HR during exercise in conjunction with rising heat stress level (Arngrimsson *et al.*, 2003; Periard *et al.*, 2011). This observation further support the earlier propositions of an increase in cardiovascular stress is linked to increase in thermal strain (Arngrimsson *et al.*, 2003; Cheuvront *et al.*, 2010; Periard *et al.*, 2011), which was reflected from the rise in both T_{re} and $\overline{T}_{sk}$ during exercise under rising humidity conditions. In higher humidity conditions, the rise in both T_{re} and $\overline{T}_{sk}$ narrows the thermal gradient between the core and the skin. This in turn has been recently shown to cause greater displacement of blood from the working muscles to the skin

thus resulting in greater circulatory strain and reduced exercise performance (Ely *et al.*, 2010; Kenefick *et al.*, 2010).

Both T_{re} and $\overline{T}_{sk}$ have important sensory inputs with regards to the control of skin blood flow (SkBF). It has been suggested that the reflex increase in SkBF to a 1^0C elevation in skin temperature was 1% to 20% of that associated with an equal elevation in core temperature (Gonzalez-Alanso *et al.*, 2008; Johnson, 2010). Elevations in skin temperature to 40^0C or more have been suggested to produce a 5 to 10 fold increase in SkBF while local cooling of the skin can reduce skin blood flow (Rowell, 1986). In his earlier finding Rowell *et al.* (1969) observed significant decrease in SV, central blood volume, mean arterial pressure, right arterial mean pressure and total peripheral resistance and a significant increase in HR when $\overline{T}_{sk}$ was raised via a water perfusion suit ($\overline{T}_{sk} =$ 32.9^0C to 38.3^0C, $\Delta\overline{T}_{sk} = 5.4^0$C) along with relatively small increases in right atrial temperature (Ta = 37.6^0C to 38.4^0C, ΔTa = 0.8^0C). These cardiovascular responses were then reversed after cooling the skin surface to 26.9^0C and decreasing blood temperature to 37.8^0C.

The limitation of our study was that there was no direct measurement of skin blood flow (SkBF) performed during exercise. Instead, we calculated tissue heat conductance (K), which has been described as indirect marker of SkBF (Brengelmann *et al.*, 1977). The higher K value in the more humid conditions would suggest a higher SkBF during exercise in order to meet the demand of thermoregulatory system. The rate of rise of SkBF during heat-stress exercise has been reported as fairly rapid at the onset of exercise with rising internal body temperature. However, the increase in SkBF becomes markedly reduced as internal body temperature reached around 38^0C. This phase is referred to as a "plateau" with minimal increase (50% of its maximal capacity) in SkBF is expected when internal body temperature reached around 38^0C (Gonzalez-Alanso *et al.*, 2008; Johnson, 2010). In the present study SkBF is proposed to have increased rapidly from the onset through to 30 minutes of exercise in the HH environment based on the elevated $\overline{T}_{sk}$ from the pre-exercise level. As exercise was prolonged for a further 30 minutes and T_{re} surpassed the 38^0C level, the rise in SkBF has been reduced and reflected in a plateau in $\overline{T}_{sk}$ within all of the three environmental conditions (for details refer to Figure 2).

Another interesting finding from the present study was the ability to maintain cardiac output during exercise across the three humidity levels, despite an indirect indication suggesting that SkBF was higher in the HH and NH environment compared with the LH environment. The cardiac

output response is consistent with the previous studies, and concluded that the cardiac output remained stable during prolonged exercise with increasing heat stress (Gonzalez-Alanso *et al.*, 2008; Johnson, 2010). Even at a higher level of relative humidity (81%), Nielsen *et al.* (1997) observed no alterations in cardiac output when participants terminated the exercise due to hyperthermia ($T_{re} = 39.9^0$C at exhaustion) after 45 minutes of exercise.

This study further demonstrates the effect of a decline in the evaporative capacity of the environment (E_{max}) on thermoregulatory system. Reduction in E_{max} directly restricts the rate of sweat evaporation and limits the amount of heat dissipated into the environment leading to increase area of skin wettedness (Alber-Wallerstrom *et al.*, 1985). Failure to increase sweating rate during exercise in the present study may suggest that some level of sweat suppression occurred with increasing level of skin wettedness in the higher humidity conditions. The increase area of skin wettedness has been shown to progressively suppress sweat output, despite an increased thermoregulatory stress (Candas *et al.*, 1979).

In addition, the observation from the present study supports the hypothesis that CV drift originates from the redistribution of central blood volume to the skin under a condition that combines a high level of exercise intensity and a low environmental capacity for heat dissipation (rising relative humidity level). The proposed increase in SkBF in the present study may have reduced the ventricular filling, end diastolic volume and stroke volume by diverting a higher portion of the cardiac output through the compliant skin circulation as described in earlier publications (Rowell, 1986). While a linear increase in HR across time is believed to compensate for the decline in SV thus, maintaining cardiac output.

In conclusion, the present study highlighted the difficulty to perform prolonged intense exercise in hot-humid conditions due to increased cardiovascular stress. This warrants further research on strategies to reduce the cardiovascular stress while exercising in hot-humid condition.

References

Alber-Wallerstrom, B. & Holmer, I. (1985). Efficiency of sweat evaporation in unacclimatized man working in a hot humid environment. *European Journal of Applied Physiology, 54*, 480-487.

Arngrimsson, S.A., Stewart, D.J., Borrani, F., Skinner, K.A., & Cureton, K.J. (2003). Relation of heart rate to percent VO_2 peak during submaximal exercise in the heat. *Journal of Applied Physiology, 94*, 1162-1168.

Brengelmann, G.L., Johnson, J.M., Hermansen, L., & Rowell, L.B. (1977). Altered control of skin blood flow during exercise at high internal temperatures. *Journal of Applied Physiology, 43*, 790-794.

Borg, G.A. (1982). Psychophysical bases of perceived exertion. *Medicine and Science in Sports and Exercise, 14*, 377-381.

Candas, V., Libert, J.P., and Vogt, J.J. (1979). Human skin wettedness and evaporative efficiency of sweating. *Journal of Applied Physiology,* 46: 522-528.

Cheuvront, S.N., Kenefick, R.W., Montain, S.J., & Sawka, M.N. (2010). Mechanisms of aerobic performance impairment with heat stress and dehydration. *Journal of Applied Physiology*, 109:1989-1995.

Collier, C.R. (1956). Determination of mixed venous CO_2 tensions by rebreathing. *Journal of Applied Physiology, 9*, 25-29.

Davies, C.T. (1979). Influence of skin temperature on sweating and aerobic performance during severe work. *Journal of Applied Physiology, 47*, 770-777.

DuBois, D. & DuBois, E.F. (1916). A formula to estimate the approximate surface area if height and weight be known. *Archives of Internal Medicine, 17*, 863-871.

Ely, B.R. Cheuvront, S.N., Kenefick, R.W. & Sawka, M.N. (2010). Aerobic performance is degraded, despite modest hyperthermia, in hot environments. *Medicine and Science in Sports and Exercise,* 42(1): 135-141.

Fritzsche, R.G., Switzer, T.W., Hodgkinson, B.J. & Coyle, E.F. (1999). Stroke volume decline during prolonged exercise is influenced by the increase in heart rate. *Journal of Applied Physiology, 86*, 799-805.

Gonzalez-Alonso, J., Crandall, C.G., and Johnson, J.M (2008). The cardiovascular challenges of exercising in the heat. *Journal of Physiology*, 586 (1), 45-53.

Gonzalez-Alonso, J., Teller, C., Andersen, S., Jensen, F., Hyldig, T. & Nielsen, B. (1999). Influence of body temperature on the development of fatigue during prolonged exercise in the heat. *Journal of Applied Physiology, 86*, 1032-1039.

Johnson, J.M. (2010). Exercise in a hot environment: the skin circulation. *Scandinavian Journal of Medicine and Science in Sports*. 20(Suppl. 3): 29-39.

Kamon, E. & Belding, H.S. (1971). Heart rate and rectal temperature relationships during work in hot humid environments. *Journal of Applied Physiology, 31*, 472-477.

Kenefick, R.W., Cheuvront, S.N., Palombo, L.J., Ely, B.R. & Sawka, M.N. (2010). Skin temperatures modifies the impact of hypohydration on aerobic performance. *Journal of Applied Physiology,* 109, 79-86.

Nadel, E.R., Cafarelli, E., Roberts, M.F. & Wenger, C.B. (1979). Circulatory regulation during exercise in different ambient temperatures. *Journal of Applied Physiology, 46*, 430-437.

Nielsen, B., Strange, S., Christensen, N.J., Warberg, J. & Saltin, B. (1997). Acute and adaptive responses in humans to exercise in a warm, humid environment. *Pflugers Archiv, 434*, 49-56.

Periard, J.D., Caillaud, C. & Thompson, M. (2011). Central and peripheral fatigue during passive and exercise-induced hyperthermia. *Medicine and Science in Sports and Exercise,* 43(9), 1657-1665.

Ramanathan, N.L. (1964). A new weighting system for mean surface temperature of the human body. *Journal of Applied Physiology, 19*, 531-533.

Rowell, L.B., Brengelmann, G.L., Murray, J.A., Kraning II, K.K. & Kusumi, F. (1969). Human metabolic responses to hyperthermia during mild to maximal exercise. *Journal of Applied Physiology, 26*, 395-402.

Rowell, L.B. (1986). *Human Circulation: Regulation During Physical Stress.* New York: Oxford University Press.

Siri, W. E. (1961). Body composition from fluid space and density. In J. Brozek & A. Hanschel (Eds.), Techniques for measuring body composition (pp. 223-244). Washington, DC: National Academy of Science.

Thompson, M. W. (2006). "Cardiovascular drift and critical core temperature: Factors limiting endurance performance in the heat?" Journal of Exercice Science and Fitness **4**(1): 15-24.

Tran ZV. (1997). Estimating sample size in repeated-measures analysis of variance. *Measurement in Physical Education and Exercise Science, 1*:89–102.