

Effect of hot climates on thermoregulation and perceived effort during fixed intensity sub-maximal exercise.

Introduction

As many Sporting events and vocational commitments such as military training and wildland fire suppression are frequently scheduled in geographical areas characterized by hot environments (Ruddock *et al.*, 2017; Hailes *et al.*, 2016; Rodríguez *et al.*, 2020; Sousa *et al.*, 2019), gaining further understanding of the thermoregulatory responses to hot environments is of great interest (Ferguson, McCooy and Mosher, 2005).

The core temperature of the human body is maintained at a relatively consistent temperature range 35-39°C through the dynamic balance between endogenous heat production and heat dissipation to the surrounding environment (Cramer and Jay, 2016). At resting thermoneutral conditions, the body core temperature is set at 37°C and any fluctuation above or below this threshold temperature initiates a thermoregulatory response from the hypothalamus using sensory feedback from thermoreceptors within the peripheral and central nervous system (Wendt, van Loon and van Marken Lichtenbelt, 2007). Furthermore, Flouris and Schlader (2015) explain as a result of the inefficient metabolic transfer during cellular respiration, the vast majority of food's chemical energy is converted to heat (>75%), with only a small amount used as mechanical energy for external work.

During exercise the release of energy as heat and the concomitant rise in body core temperature evoke numerous physiological mechanisms of heat loss that are activated to prevent an excessive rise in body core temperature (Wendt, van Loon and van Marken Lichtenbelt, 2007), these bodily adjustments vary in regard to the duration, types and intensity of exercise, training level and environmental conditions (Silva *et al.*, 2019; Coso *et al.*, 2011). The excess heat produced in the working muscle through exercise causes an increase in the muscle-to-core temperature gradient that initiates the transfer of heat via conduction and convection from the muscle to the blood, and then body core (Ruddock *et al.*, 2017; Flouris and Schlader, 2015). Subsequently, to prevent a continuous rise in body core temperature (hyperthermia) Wendt, van Loon and van Marken Lichtenbelt (2007) explain, heat is transported from the core to the skin at a rate determined by the temperature gradient between these two areas. Once the metabolic heat is transferred to the skin it can be lost to the environment to limit heat storage and increases in body temperature through radiation, conduction, convection and evaporation (Cramer and Jay, 2016; Wendt, van Loon and van Marken Lichtenbelt, 2007). However, Aleksander *et al.* (2010) suggest when ambient temperature exceeds the skin temperature, mechanisms such as convection and radiation are impossible, and the only way heat can be eliminated is through sweat production and its evaporation. Wong *et al.* (2014) explain, evaporation is the process whereby heat is transferred from sweat on the surface of the skin and converted into water vapor and is considered the major mechanism for heat loss during exercise in the heat. Furthermore, Inbar *et al.* (2004) reveal, 80% of heat loss is achieved through evaporation with approximately 2.4 MJ of heat energy liberated per litre of sweat vaporized (Hayes *et al.*, 2014). Having said this, when ambient temperature exceeds 36°C, or the relative humidity is high the gradient between the skin and the air is reduced, preventing heat balance and therefore increasing the risk of muscle cramps, heat stroke/exhaustion and impaired exercise performance (Ruddock *et al.* 2017; Flouris and Schlader, 2015; Ross *et al.*, 2013).

Although the exact mechanisms of the afferent sensory feedback and the subsequent down-regulation of central drive associated with fatigue in the heat are not fully understood, the most likely cause is thermal strain (Cuttell, Kiri and Tyler, 2016; Abbiss *et al.*, 2010), with numerous studies

suggesting exercise performance is impaired when a “critical” core body temperature in the range of 39.5–40.5°C is reached (Ross *et al.*, 2013; Rodríguez *et al.*, 2020; Xu, Zhao and Gao, 2022). Abbiss *et al.* (2010) examined the influence of environmental temperature on cycling performance and showed core temperature was significantly greater in the hot (34°C) than in the cold (10°C) trial ($P < 0.001$) which led to reductions in power output possibly caused by an anticipatory reduction in muscle activation to prevent the attainment of critical core body temperature. However, in contrast, Brett *et al.* (2009) showed in a study involving well trained runners that core temperature ($>40^{\circ}\text{C}$) was not associated with a degradation in performance in an 8-km time trial in both cool (13°C) and warm (27°C) conditions, with no differences in running velocities (282 m/min vs 279 m/min; $P = 0.82$).

A further explanation for impaired performance during prolonged exercise in the heat is cardiovascular strain caused by the physiological phenomenon known as “cardiovascular drift” (Polsinelli, Rodio and Federico, 2021). Cardiovascular drift (CVD) occurs after 10–15 minutes of continuous exercise and consists of a progressive increase in heart rate (HR) and a concomitant decrease in stroke volume (SV) and mean arterial pressure, while cardiac output is maintained (Wingo and Cureton, 2006; Stone *et al.*, 2021). The decrease in SV associated with CVD has been attributed to peripheral displacement of blood volume as a result of the circulatory conflict between vasoconstrictor and vasodilator systems involved in the redistribution of blood flow to the skin and active skeletal muscle during exercise (Sousa *et al.*, 2019). In addition, Polsinelli, Rodio and Federico (2021) suggest the increased cardiovascular demands are associated with a gradual increase in core temperature exacerbated by factors such as dehydration through reduced blood volume that is suggested to decrease maximal oxygen uptake ($\text{VO}_{2\text{max}}$) by as much as 5 to 12% (Wingo and Cureton, 2006; Stone *et al.*, 2021). A study by Wingo *et al.* (2005) revealed CVD during 45 min of exercise in the heat (35°C) was associated with decreased $\text{VO}_{2\text{max}}$, as a 12% increase in HR (151 vs 169 bpm, $P < 0.05$) and 16% decrease in SV (120 vs 101 ml/beat (-1), $P < 0.05$) were accompanied by a 19% decrease in $\text{VO}_{2\text{max}}$ (4.4 vs 3.6 L/min (-1), $P < 0.05$) despite attainment of a higher maximal HR ($P < 0.05$). With this said, Wingo and Cureton (2006) revealed that the reductions in $\text{VO}_{2\text{max}}$ associated with CVD during exercise in a hot environment is attenuated with body cooling via fan airflow as $\text{VO}_{2\text{max}}$ and SV decreased by 18% and 12% respectively, with HR increasing by 16% ($P < 0.05$) with no cooling, whereas, with fan airflow $\text{VO}_{2\text{max}}$ fell less (5.7%, $P < 0.05$), HR rose less (4%, $P < 0.05$) and SV decreased less (3%, $P < 0.05$). These contradictory findings may be indicative of the rating of perceived exertion (RPE), a subjective estimate of the intensity or difficulty of a physical task, generally measured using the Borg scale (Deshayes, Pancrate and Goulet, 2022). It is suggested that thermal perception plays a pivotal role in thermoregulation an RPE may be the ultimate modulator of behavior during exercise in the heat (Flouris and Schlader, 2015).

In light of this, the purpose of this study was to determine the effect of a hot environment on physiological responses and perception of effort during fixed intensity, sub-maximal exercise. The study hypothesized a hot environment would impact greater on thermoregulatory and cardiovascular systems and therefore a higher perceived effort.

Method

All lab equipment reference details are provided in **Appendix 1**.

Participants

Eleven students studying Sport and Exercise science at UWTSD were asked to participate in the current study. Participants included ($n = 6$ male and 5 female), group characteristics can be found in **Table 1**.

All participants were physically active to the extent that they met daily physical activity recommendations (ACSM, 2018, p. 4) and had no prior injuries within the last 12 months. All participants provided written and verbal informed consent before any preliminary or experimental trials and Ethical approval was obtained from the University Ethics Board. The participants were required to abstain from caffeine and alcohol 12 h before each trial, and to avoid exhaustive exercise during the 24 h period prior to trials.

Table 1. *Demographics*

Age, years	29	8.38
Body mass, kg	72.5	9.8
Height, cm	173.6	8.2

Values are means $\pm$ SD

Research design

A randomized cross-over research design was used in which all participants were randomly assigned and tested under two experimental conditions (1) COLD, performed outside at a temperature of 7.3°C, 78% relative humidity, to replicate conditions in the UK and (2) HOT, performed in an environmental chamber maintained at 31.4°C, 47% relative humidity. All trials were separated by 7 days to control for fatigue and the training effect, whilst being conducted at the same time of day to control for circadian fluctuations in body temperature, other biological variables, and performance. For HOT trial, exercise was terminated if participants felt unable to continue or they reached the institutional ethics committee–approved core temperature for termination of exercise in the heat (39.7°C).

Preliminary measurements

Following anthropometric measurements of height (Seca 213) and weight (Seca 875) participants performed a graded exercise test (GXT), on an electronically braked cycle ergometer (Wattbike atom x), to determine $\text{VO}_{2\text{max}}$ and peak power output (PPO). The GXT began at a pre-determined intensity to accommodate the heterogeneity of the group and increased by 25 W every 2 min until subjects reached volitional exhaustion or could no longer maintain 40 rpm. Oxygen uptake and other gas exchange measures were obtained via open-circuit spirometry (Coretex Metalyzer 3B) that was calibrated prior to testing (Cranlea 995/2619 (20% O₂, 8% CO₂)) and averaged every 30 s, with HR recorded continuously (Polar H10). RPE was obtained at the end of every 2-min stage during the GXT.

$\text{VO}_{2\text{max}}$ was taken as the highest 30-s mean value attained prior to exhaustion in the test. The criteria used to determine $\text{VO}_{2\text{max}}$ were, (1) a plateau produced in the VO_2 curve with increases lower than (1.5 mL·kg⁻¹·min⁻¹) between 30 s intervals; (2) RER above 1.10; and (3) a heart rate equal to or

greater than the theoretical maximum. PPO was calculated as the final stage completed during the GXT.

Experimental trials

On each test day, prior to entering the designated test environments participants were fitted with an HR monitor (Polar H10) and core temperature (T_{cr}) was taken using tympanic thermometer (Innovation Radiant Th809) as when not possible to measure T_{cr} directly, alternative easily accessible sites can be used, such as the external auditory canal (Polsinelli, Rodio and Federico, 2021). Subsequently, participants were weighed semi-nude (underwear) on weighing scales (Seca 875) before and after completion of exercise, and total sweat rate was calculated as the difference in body mass. No fluid was permitted during trials.

After all resting measures were obtained, subjects entered the designated test environment and began cycling at a power output corresponding to 60% PPO for 30 min. During the submaximal exercise, T_{cr}, HR, RPE (Borg scale) and VO₂ were measured and recorded at 10, 20 and 30 minutes. Respiratory gases were collected using a Douglas bag (Douglas 100litre with 2100 series 3 way stop cock) for an exact period of time (60s) prior to subsequent analysis. Further details regarding the exact protocol can be found in **Appendix 2**.

Statistical analysis

All data was uploaded and presented in Microsoft Excel before being transferred and analysed with IBM SPSS statistics, version 28.0. All data was reported as mean $\pm$ SD, and a paired samples T-Test was used to compare significant differences between the experimental conditions, with a α level of $P < 0.05$ used as the criterion for statistical significance. Effect sizes were estimated using partial eta squared where 0.01 represented a small effect size, 0.06 a moderate effect size, and 0.14 a large effect size.

Results

Core temperature (Tcr)

Figure 1. shows there was an increase in Tcr in both conditions from 10 - 30 minutes by $1.30 \pm 0.78^{\circ}\text{C}$ in HOT, whereas COLD only increased by $0.59 \pm 1.05^{\circ}\text{C}$. Tcr was significantly greater at 10, 20 and 30 minutes in HOT ($P = 0.004$, $P = 0.001$, $P = 0.001$) with a significant difference in core temperature change observed between minute 10 and 30 in HOT [$M = 1.30$, $SD = 0.77$] compared to COLD ($M = 0.59$, $SD = 1.04$; $t = 2.679$, $P = .023$). The magnitude of the differences in the means was large (eta squared = 0.41). There were no significant differences in the two groups at 0 min ($P = 0.767$).

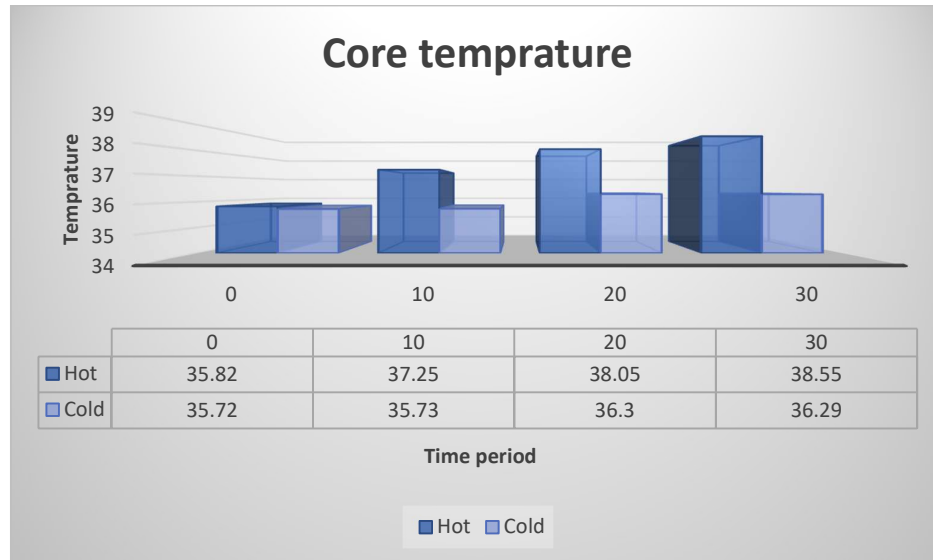


Figure 1.

Heart rate

HR increased in both conditions from 10 - 30 minutes by 15.55 ± 7.62 bpm (8.7%) and 5.27 ± 7.76 bpm (3.3%) in HOT and COLD respectively. **Figure 2.** shows HR was significantly greater at 10, 20 and 30 minute time points in HOT ($P = 0.010$, $P = 0.001$, $P = 0.001$), with a significant difference in test result observed between minute 10 and 30 in HOT [$M = 15.54$, $SD = 7.62$] compared to COLD ($M = 5.27$, $SD = 7.76$; $t = 3.163$, $P = .010$). The magnitude of the differences in the means was large (eta squared = 0.50).

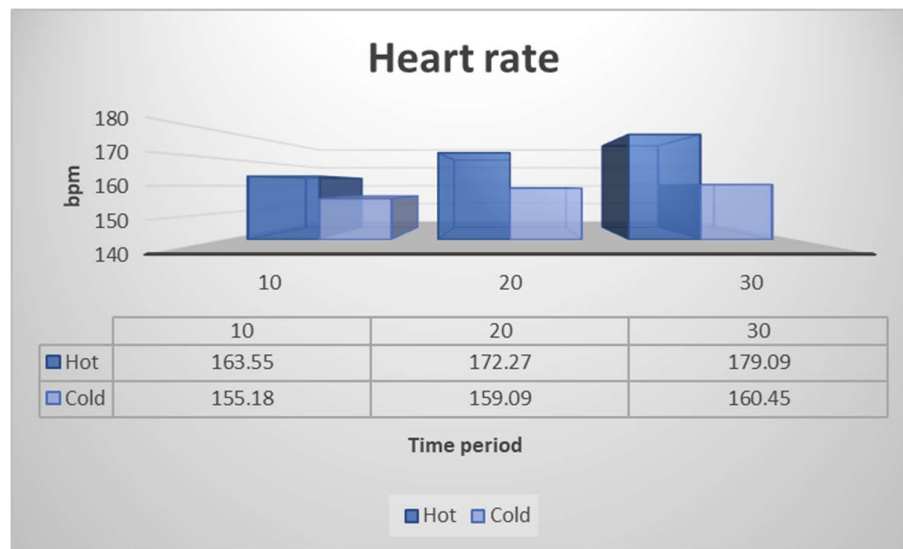


Figure 2.

Figure 3. shows there was an increase in RPE during both conditions from 10 - 30 minutes by 4.45 ± 1.86 and 2.18 ± 1.47 in HOT and COLD respectively, with RPE significantly greater at minute 20 and 30 ($P = 0.014$, $P = 0.002$) in HOT, although non-significant at minute 10 ($P = 0.091$). Furthermore, there was a significant difference in test result observed between minute 10 and 30 in hot [$M = 4.4545$, $SD = 1.86$] compared to COLD ($M = 2.18$, $SD = 1.47$; $t = 3.180$, $P = .010$). The magnitude of the differences in the means was large (eta squared = 0.50).

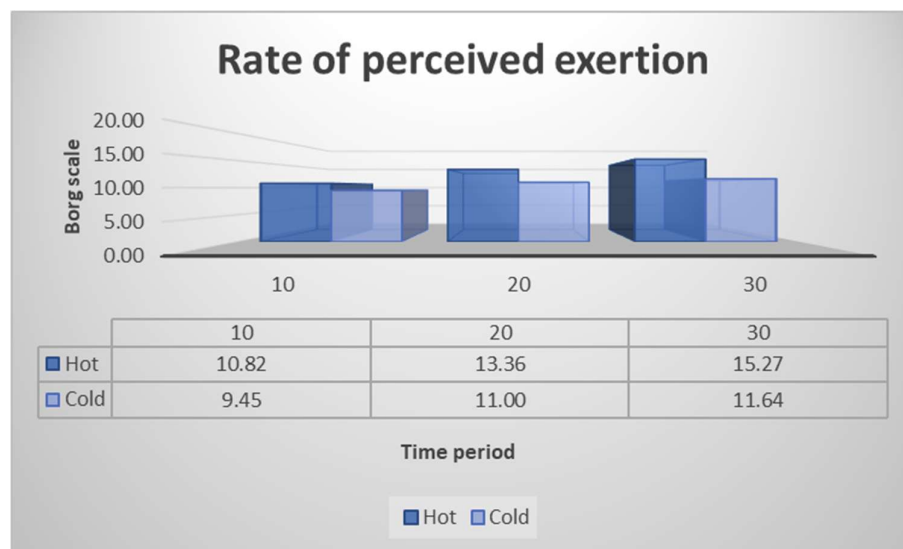


Figure 3.

VO_2 increased in both conditions from 10 - 30 minutes by 4.69 ± 6.82 mL/kg/min and 1.79 ± 2.52 mL/kg/min during HOT and COLD respectively (**Figure 4**). VO_2 was greater at all 3 time points measured in HOT although only minute 30 was significant ($P = 0.030$), with minute 10 and 20 non-significant ($P = 0.622$, $P = 0.659$). Furthermore, there was a significant difference in the test result observed between minute 10 and 30 in HOT [$M = 4.69$, $SD = 6.82$] compared to COLD ($M = 1.79$, $SD =$

2.52; $t = 1.289$, $P = .226$]. The magnitude of the differences in the means was large (eta squared = 0.50).

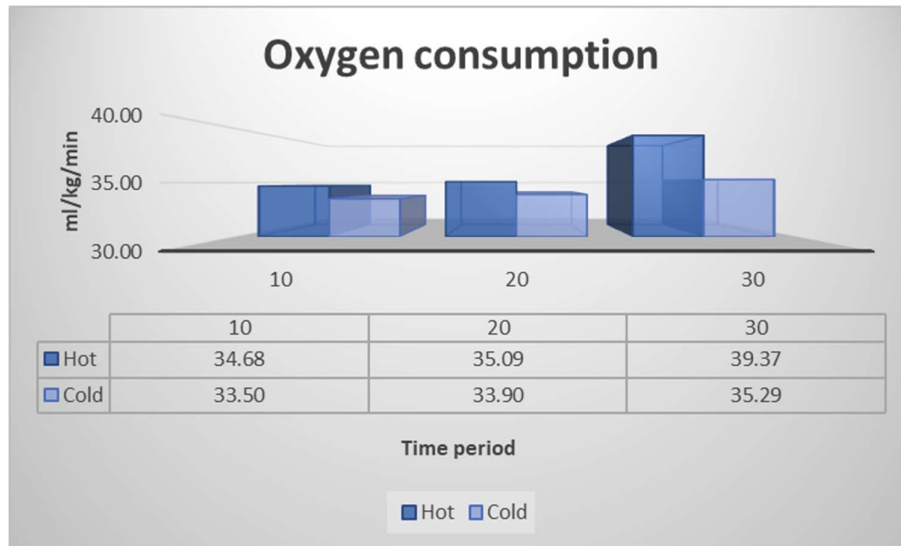


Figure 4.

Body mass

Figure 5. shows body mass decreased in both conditions from pre to post test with a reduction of 0.68 ± 0.49 kg and 0.29 ± 0.14 kg in HOT and COLD respectively. A significant difference in test results was observed between hot [$M = 0.68$, $SD = 0.49$] and cold [$M = 0.29$, $SD = 0.144$; $t = 3.173$, $P = 0.10$]. The magnitude of the differences in the means was very large (eta squared = 0.50).

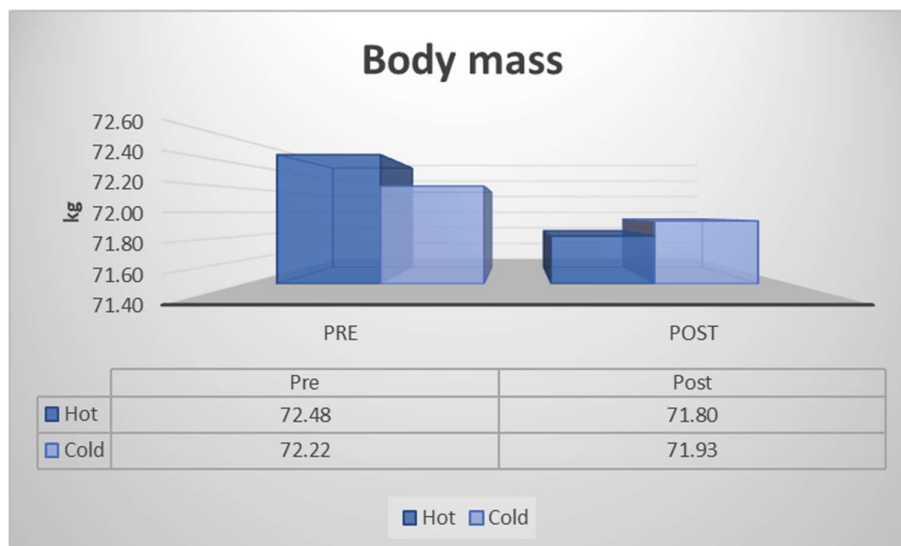


Figure 5.

Discussion

The aim of this study was to assess the physiological and perceptual responses to sub-maximal exercise in different environments. As expected the results agreed with the hypothesis in regard to a greater demand on the thermoregulatory and cardiovascular systems during the HOT trial with a higher perceived effort.

The main finding of the current study was that T_{re} and HR increased significantly more during HOT at all measured time points compared to COLD ($P = 0.004$, $P = 0.001$, $P = 0.001$) ($P = 0.010$, $P = 0.001$, $P = 0.001$), while $\dot{V}O_2$ was only significantly different at minute 30 ($P = 0.030$). This would suggest that during the early stages of exercise (<20m) T_{re} may be the more important thermoregulatory factor that effects performance in the heat. The findings support Xu, Zhao and Gao (2022) who revealed elite sailors time for completing a 6 km rowing test was significantly longer ($P = 0.0014$) during hot conditions (31.8°C) compared to warm (23°C) as a result of increased heat storage at minute 5 and 15 ($P = 0.0036$; $P = 0.0018$) narrowing the core to skin temperature. Having said this, the concept of a “critical” core temperature of 40°C being the determining factor of fatigue in the heat (Brett *et al.*, 2009) was not tested in the current study as 38.55 ± 0.86 was the highest T_{re} recorded. Additionally, the findings of the present study revealed CVD did occur after 10 minutes of exercise with a progressive increase in HR of 15.54 bpm (8.7%) during HOT, compared to a relatively small increase of 5.27 bpm (3.3%) seen in COLD. These results are similar to the 9% increase in HR that Stone *et al.* (2021) reported, however slightly less than the 12% increase in HR reported by Wingo *et al.* (2005), although it must be mentioned that during that study test duration was 50% longer (45min) and the environment was 11.5% hotter (35 °C), suggesting that the severity of temperature and duration of exercise both play a pivotal role in CVD.

Alongside physiological differences the current study revealed perceived effort increased during HOT and COLD conditions at all measured time points, however RPE was only significantly greater during HOT at 20 and 30 minutes ($P = 0.014$, $P = 0.002$). The findings support earlier work by Crewe, Tucker and Noakes (2008) who showed RPE increases linearly during exercise at a fixed intensity and that T_{cr} correlates significantly with RPE ($r = 0.92$). The timing of the difference in RPE coincided with increases in HR and may be indicative of CVD as Borg *et al.* (2018) suggest it might be expected that a (~ 10 b·min⁻¹) difference would be required to alter RPE, which was only shown in the present study at 20 and 30 minutes. Considering this, the results further enhance the role of thermal perception and the belief that RPE may be the ultimate modulator of behavior during exercise in the heat (Flouris and Schlader, 2015; Crewe, Tucker and Noakes, 2008).

Although the current study did not provide any evidence of decreased performance associated with a hot environment, it can be assumed based on the physiological responses and differences in perceived effort between groups that hot environments significantly impact exercise performance, and with major sporting events such as the Summer Olympic Games and the World Athletics Championships being held during the summer months (Miyazawa *et al.*, 2021), training approaches that augment the relevant adaptations that prepare individuals for maximal exercise in hot conditions such as heat acclimation and acclimatisation (HA) (Lorenzo *et al.*, 2010) are recommended. Rahimi *et al.* (2019) explain HA may alter critical physiological parameters by increasing plasma volume, reducing exercise HR, lowering T_{cr} and improving maximal cardiac output. Details of adaptations and time frame needed to impose are shown in **Appendix 3**. In addition, a recent meta-analysis by Waldron *et al.* (2021) revealed moderate-to-large improvements in $\dot{V}O_{2max}$ following HA in both thermoneutral and hot environments (CI 0.24–0.59, $P < 0.001$), (CI 0.22–1.27, $P = 0.005$). Alternatively, Ruddock *et al.* (2017) suggest practical cooling strategies such as

cold fluid, ice slurry ingestion and neck cooling may also improve performance in hot environments by benefiting thermal perception and RPE. Furthermore, the attenuation of RPE was also made possible with intermittent face cooling as RPE values were significantly lower when soft gel packs were applied to the faces of subjects performing 40 minutes of continuous cycling at 65% peak HR when compared to a control group (FC, 11.6 ± 2.2 points; CONT, 14.2 ± 1.3 points; $P < 0.01$) (Miyazawa *et al.*, 2021). Considering this, what appears evident throughout the literature is that limiting the number of physiological or emotional stressors to a minimum during exercise should ensure RPE is controlled (Deshayes, Pancrate and Goulet, 2022). Therefore, the current study suggests that individuals competing in sporting events or undertaking vocational commitments in hot environments should take appropriate measures to ensure they are prepared for exercise or physical activity in the heat. Additionally, any athlete looking to achieve a personal best performance may wish to choose an event scheduled in a cooler climate.

The present study acknowledges that as most sporting endeavours are performed at a self-paced intensity (Brett *et al.* 2009; Borg *et al.*, 2018) the results found within may not have practical application in real world settings. Further limitations of the study was a lack of data collection in a real environment, and that subjects were a rather heterogeneous population who were tested under specific environmental conditions. Thus, applying the current findings to a specific population and/or environment different from the one employed in this study warrants caution.

Conclusion

In summary, the present study shows that hot environments significantly increase the demands on the thermoregulatory and cardiovascular systems. These effects can, in turn, lead to the risk of conditions such as hyperthermia and dehydration, which can eventually impair and limit performance (Miyazawa *et al.*, 2021). The findings also suggest that RPE may be used to accurately predict the duration of exercise to exhaustion at a fixed work rate in different environmental conditions. It is therefore necessary to highlight the significance of this information to clinical sport coaches responsible for athletes during competition in hot environments, as well as senior personnel involved in the decision making process of organisations such as the military.

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Appendix

Appendix 1.

Item	Make & Model	Reference	Website
Stadiometer	Seca 213	(Seca, Hamburg, Germany)	https://www.seca.com
Seca Weighing Scale	Seca 875	(Seca, Hamburg, Germany)	https://www.seca.com
Wattbike Atom	Wattbike atom x	(Wattbike, Nottingham, UK)	https://www.wattbike.com
Metabolic Cart	Coretex Metalyzer 3B	(Leipzig, Germany)	https://cortex-medical.com
Calibration gas	Cranlea 995/2619 (20% O ₂ , 8% CO ₂)	(Cranlea, Birmingham, UK)	https://www.cranlea.co.uk
Heart rate chest strap	Polar H10	(Polar Electro, Warwick, UK)	https://www.polar.com/uk-en
Tympanic Thermometer	Innovation Radiant Th809	(Innovation, Radiant, Taiwan)	https://www.radiantek.com
Douglas Bag	Douglas 100litre with 2100 series 3 way stop cock	(Hans Rudolph, Kansas, USA)	https://www.rudolphkc.com

Appendix 2.

Douglas bag data collection

- It is imperative that data collection is precisely timed using the stopwatch.
- Respiratory gases are collected for an exact period of time.
- It is important that the mouthpiece and nose clip are worn by the participant PRIOR to the start of the data collection period (e.g., ask the participant to attach the nose clip and mouthpiece in the 30 seconds before the planned time for data collection).
- The data collection period should have a clear count down from the timekeeper e.g., 3, 2, 1, GO, in which the valve of the designated Douglas bag is opened/closed in line with the count down.
- Once this data collection has been completed, remove the mouthpiece and nose clip from the participant.
- It may be advisable to provide the participant with some blue roll to remove the mouthpiece.

Douglas bag data analysis

- Firstly, the gas concentration (%O₂, %CO₂) of each bag must be sampled.
- Analysing the gas concentration is completed for an exact period of time (60 seconds) and is precisely timed using the stopwatch.
- A 60 second sampling period equals the removal of 1.8 L of air.
- Hold the sample line of the Douglas bag firmly closed and THEN remove the clip/stopper from the end.
- Still holding this sample line closed, attach the sample line of the Douglas bag to the sample line of the Servomex gas analyser.

- The sampling period should have a clear count down from the timekeeper e.g., 3, 2, 1, GO, in which the sample line is opened/closed in line with the count down.
- Record the numbers on the Servomex within the last 15 seconds of the sampling period. If these are not recorded at this point, they will disappear when the sample line of the Servomex is put back into ambient air.
- At the end of the sampling period, hold the sample line of the Douglas bag firmly closed until the clip/stopper has been replaced.
- Secondly, the volume of gas within the bag must be measured.
- Attach the Douglas bag to the vacuum as previously.
- Make sure the vacuum monitor is reset to 0 before starting.
- Opening the valve and turning on the vacuum must be coincided for accurate measurement of volume. Again, an individual should countdown, e.g., 3, 2, 1, GO, when the vacuum should be turned on, and the valve of 1 bag opened.
- When the number is stable, note the value.
- Add 1.8 L to the value. This is to accommodate for the 1.8 L removed during the gas concentration sampling.

Appendix 3.

Adaptation	Days of heat acclimatisation
Decrease in heart rate during exercise	3-6
Plasma volume expansion	3-6
Decrease in sweat NA ⁺ and Cl ⁻ concentrations	5-10
Increase in sweat rate and sweat sensitivity	7-14
Increase in cutaneous vasodilation	7-14

(Wendt, van Loon and van Marken Lichtenbelt, 2007).