The Stress and Strain of Triathlon Racing in Different Thermal Environments

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ABSTRACT

Athletic endurance competitions in hot environments have become more relevant in professional sports. The inception for this thesis was to understand why a group of elite endurance triathletes repeatedly failed to perform in hot environments. The subsequent aims were to explore the physiological basis of elite endurance performance in the heat, with a focus on cerebrovascular responses because they seemed relevant and yet remained relatively less well explored.

Four sequential studies addressed the thesis: 1) observe and measure athletes' physiology and race behaviour, 2) measure physiological responses from short-duration, maximal-intensity exercise in controlled temperate (TEMP) vs hot

(HOT) environments, 3) measure physiological responses from a simulated race performance in those controlled environments, and 4) develop and assess a heat-conditioning intervention to improve performance and reduce physiological limitations. Study One observed elite triathletes competing over 2010-11 in four races (two temperate, two hot environments). Run pace in the first kilometre was considerably slower and deteriorated more in hot environments. Cardiovascular responses were similar before, during and immediately after racing; indicating that athletes competed at the physiological limit of their cardiovascular systems.

Based on the field observations, Study Two investigated maximal exercise performances over 10 s and 5 min in TEMP and HOT environments because this duration is sufficient for performance to be predominantly aerobic but short enough to limit core (incl. brain) temperature and gastro-intestinal (GI) system influence. Cycling in HOT produced 11% more power over 10 s but 3.4% less power over 5 min, and was associated with increased actual and perceptual thermal strain and lower prefrontal cortex oxygen saturation.

Given the thermal strain and restricted physiological function observed within 5 min in Study Two, Study Three investigated elite athletes' performance and physiological profile of a triathlon race simulation (60-min SIM) in TEMP and HOT environments. Power profile was stochastic over 2-min periods throughout, but controlled externally for the first 40 min (FIXED). In HOT, power output was ~20% lower for the final 20-min self-selected paced efforts and all athletes experienced greater physiological strain during FIXED. To maintain homeostasis athletes may have reduced their power output during the self-selected section in response to prefrontal cortex oxygen desaturation, deteriorated affective state, leg muscle oxygen desaturation and blood flow redistribution that negatively influenced anticipatory pacing.

Having now quantified the negative performance and physiological impact of HOT on an under-prepared athlete group, Study Four was a controlled, short-term heat acclimation to determine its physiological and ergogenic effectiveness in TEMP and HOT environments for elite athletes. Overall, heat acclimation had small and unclear influences on performance, cerebrovascular, cardiovascular,

and psycho-physical responses to exercise in either TEMP or HOT conditions relative to those after control conditioning. The heat acclimation protocol may have been ineffective, or alternatively participants' training status may have constrained further adaptation.

The thesis demonstrated that elite and highly-trained athletes competing in HOT environments produced more power for singular sprints but less power for 5-min efforts, 60-min race simulations and run pace in triathlon races; contributing to poor performance or volitional exhaustion. Physiological factors that influenced performance in HOT included greater thermal, cardiovascular, cerebrovascular and affective strain, which generally were not improved by a 10-d heat-acclimation regime.

ACKNOWLEDGEMENTS

Throw roses into the abyss and say: “Here is my thanks to the monster who didn’t succeed in swallowing me alive.”

Nietzsche

To the participants in this study: my sincerest thanks for the blood, sweat and energy you gave so freely.

To Sam Lucas: for being the sunshine among the rainclouds of confusion.

And to Jim Cotter: My hero, my mentor, my friend.

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LIST OF ABBREVIATIONS

5-min TT	Five-minute cycling time trial at maximal effort
60-min SIM	Performance trial designed to simulate a 40-km draft-legal cycling leg in a triathlon
BF_{Skin}	Skin blood flow
BP	Blood pressure
Brain HHb	Brain deoxygenated haemoglobin concentration measured at the prefrontal cortex
Brain O_2Hb	Brain oxygenated haemoglobin concentration measured at the prefrontal cortex
Brain tHb	Brain total haemoglobin concentration measured at the prefrontal cortex
Brain TOI	Brain tissue oxygen index measured at the prefrontal cortex
BV	Blood volume
CO Dilution	The modified carbon monoxide rebreathing procedure (Schmidt and Prommer, 2005)
CON	Control conditioning, specifically the 10 d control conditioning protocol utilized in this study
CVR_{hyper}	Cerebrovascular reactivity to a hypercapnic (5% CO_2) stimulus
CVR_{hypo}	Cerebrovascular reactivity to a hypocapnic (hyperventilation) stimulus
EDV	End-diastolic volume
f	Breathing frequency
[Glu]	Blood glucose concentration
$[H^+]$	Hydrogen ion concentration
HA	Heat acclimation, specifically the 10 d heat acclimation conditioning protocol utilized in this study
Hb	Haemoglobin
HCO_3^-	Bicarbonate
Hct	Haematocrit

HR	Heart rate
GI System	Gastro-intestinal system
[La]	Blood lactate concentration
Leg HHb	Skeletal muscle deoxygenated haemoglobin concentration measured at the left vastus lateralis
Leg O_2Hb	Skeletal muscle oxygenated haemoglobin concentration measured at the left vastus lateralis
Leg tHb	Skeletal muscle total haemoglobin concentration measured at the left vastus lateralis
Leg TOI	Skeletal muscle tissue oxygen index measured at the left vastus lateralis
MABP	Mean arterial blood pressure
MCAv	Middle cerebral artery blood flow velocity
NIRS	Near-infrared spectroscopy
$PaCO_2$	Partial pressure of arterial carbon dioxide
PaO_2	Partial pressure of oxygen
$P_{ET}CO_2$	Partial pressure of end-tidal carbon dioxide
PH_2O	Water vapour pressure
PRE	60-min SIM performance trial before any intervention
PV	Plasma volume
$\dot{Q}$	Cardiac output
RCV	Red cell volume
RPE	Rating of perceived exertion
RH	Relative Humidity
SV	Stroke volume
T_{Brain}	Brain temperature
T_C	Core temperature

TCD	Transcranial Doppler ultrasound
T_{mus}	Muscle temperature
T_{Skin}	Skin temperature
USG	Urine specific gravity
v_a	Air velocity
$\dot{V}_E$	Minute ventilation
$\acute{V}O_2$	Volume of oxygen consumption
$\dot{V}O_2$ max	Maximum volume of oxygen consumption (maximal aerobic power)
V_t	Tidal volume
VT1	First ventilatory threshold
VT2	Second ventilatory threshold
W_{max}	Maximum sustained power output (calculated from lactate step test)

1 INTRODUCTION

1.1 Overview

Effects of hot environments on human performance remain only partly understood, as do the underlying causes. Since the pioneering studies conducted during World War One (Adolph and Fulton, 1924), we have known that the combined stress from exercise and a hot environment can detrimentally affect sustained exercise performance and potentially health. Humans, like all organisms, respond physiologically to stress through a cyclic process of fatigue and adaptation (Selye, 1950). For humans competing in endurance events, exercise intensity is the most stressful factor that aggravates fatigue. Metabolic heat stress is exacerbated in environments that lessen or reverse the gradients for eliminating this heat.

1.2 The scope and limitations of existing knowledge

Evidence from several laboratory studies and fewer field studies has shown that sustained and intense exercise performance is impaired by hot or humid environments (e.g., Bannister and Cotes, 1959; Pugh et al., 1967). Ambient temperatures higher than 25 ˚C were identified as detrimental to performance as early as the 1958 Empire Games (Bannister and Cotes, 1959), and elaborated more recently (Guy et al., 2015). The relationships between human thermoregulation and performance in endurance sports events have been studied in a variety of events, including marathon running (Ely et al., 2007; Maron et al., 1977; Pugh et al., 1967), half-marathon running (Byrne et al., 2006; Lee et al., 2010), 8000-m track running (Ely et al., 2009), cycling (Racinais et al., 2015), open-water swimming (Bradford et al., 2015), and Ironman triathlon (Laursen et al., 2009, 2006). Team sports, game sports (e.g., tennis), and sports less dependent on endurance power and capacity have also measured pre- and post-exercise core temperature and shown potentially exercise-limiting temperatures (e.g., Bergeron et al., 2006; Duffield et al., 2009; Shirreffs, 2010) and/or heat-induced reductions in high-intensity efforts (Nassis et al., 2015; Racinais et al., 2012).

Considerable research effort has gone into testing human thermoregulation and exercise capabilities in the well-controlled thermophysical conditions of an environmental chamber. Beneficially this research has produced accurate physiological data from tightly-controlled exercise and environmental stressors. However, data produced from controlled laboratory studies may lack validity when applied to race performances where the environmental and motivational conditions are inherently different (e.g, convective cooling generated by cycling at 30-50 km/h; (Faria et al., 2005a; Saunders et al., 2005) and the performance task is highly variable (e.g., the tactical pace changes during a 10,000 m track running race at a World Championships final or an event on topographically changing terrain; Mora-Rodriguez et al., 2008). Few study designs have attempted to transfer such race-specific factors from the field to the laboratory. Even fewer have investigated highly-trained and elite-level athletes. All such factors (e.g., intensity profile, endurance fitness, motivation, airflow) are known to alter thermophysical effects of exercise, so their collective influences may be far from trivial.

1.3 The Problem (and Thesis)

Exercise and heat are stressors that influence human performance and physiological responses (see Figure 2.1) through a process of fatigue and adaptation. Fatigue is a multifactorial response that may be defined by failure to maintain the required or expected force or power output (Allen et al., 2008; Edwards, 1983; Fitts, 1994), and that requires central and peripheral control to maintain homeostasis (Amann, 2011). All organisms (including humans) have the capacity to adapt if the stimuli are not excessive (e.g., fatal; Selye, 1950; Selye and Fortier, 1950), the normal regulatory mechanisms are not overwhelmed (Amann, 2011; Edwards, 1983; Noakes and Gibson, 2004; Noakes et al., 2005), and that recovery time is sufficient (Allen et al., 2008; Mosso, 1904). Humans adapt exceptionally well to heat compared with other physiological stressors including cold, high altitude, high pressure, high gravitational force and low calorie intake (Edholm, 1966). An aerobically-exercising human converts up to ~20% of total energy into external work; the remainder is converted to heat internally (Andersen and Saltin, 1985). Fortunately humans are exceptionally powerful at offloading heat – especially via evaporation of the water of sweat

from the skin (Nadel, 1988). The environment acts as a heat sink until ambient air temperature and humidity approach or exceed skin temperature and vapour pressure, respectively. However, the combination of exercise that generates more heat than a hot or humid environment can absorb causes uncompensable heat stress (Périard et al., 2011a; Wright et al., 2011). The effect of the environment on performance among elite athletes in the field is not high, in contrast to the detrimental effect evident in less trained individuals in the field (Ely et al., 2007) and lab-based studies of hot compared with temperate environments (Galloway and Maughan, 1997; Nybo, 2008; Schlader et al., 2010a; Tatterson et al., 2000). If individuals are highly motivated and/or resist strong physiological feedback (see Figure 6.1) they stop exercising due to heat exhaustion, or, in extreme cases they may exercise to heat stroke (Bouchama and Knochel, 2002; Casa et al., 2012; Gilat et al., 1963; Hanson and Zimmerman, 1979).

What causes highly-trained and motivated athletes to stop or fail to finish a race in hot conditions? For example, four out of ten New Zealand triathletes failed to finish the 2011 Mooloolaba ITU World Cup, an event at which the PhD candidate was also functioning as the New Zealand team Sport Scientist. Some field research and a much larger volume of laboratory research had investigated these questions of human capability and physiology during intense exercise in heat-stressful environments. Some factors were already clear; for example, the competition to supply blood to multiple tissues with a finite cardiovascular capacity. However, other physiological and psychophysiological restrictions were – and largely still are - unresolved. In view of these knowledge limitations, this thesis had three sequential purposes to: 1) observe and measure athletes' physiology and exercise behaviour during racing, 2) measure the physiological responses from a simulated race performance and environment, versus a short-duration, maximal-intensity exercise bout in a controlled laboratory environment, and 3) develop and assess a heat-conditioning intervention to improve performance, reduce physiological limitations, and examine cerebrovascular responses.

The thesis is organised into data chapters addressing its three purposes (Table 1.1, Figure 1.1). Chapter three was an observational field study designed to measure performance outcomes and physiological responses for elite triathletes competing in international races in different environments. The purpose was to quantify the performance-limiting factors in temperate versus hot environments because we understand the performance and physiological effects from exercising in the heat, however, we don't necessarily understand how the race environment (performance and physiology) affect the athletes. Athletes reported experiencing dyspnoea despite high ventilation, abnormal weakness and fatigue, and/or an overwhelming sensation of being inescapably hot. Given that both performance and physiology were hindered when racing in a hot environment we constructed laboratory studies to separate and analyse these two factors under temperate compared with hot environments.

Table 1.1: Physiological and physical factors that contribute to endurance competition examined in each thesis chapter. ✗: not measured or examined, ?: measured and examined in relation to performance and other factors, ✓: real-world condition that directly influenced performance, *: simulated to replicate real-world environments, §: adaptation that may influence other factors.

	Chapter 3.0 Triathlon Race Performance	Chapter 4.0 10-s Sprint & 5-Minute Time Trial	Chapter 5.0 60-Minute Race Simulation	Chapter 6.0 Heat Acclimation & Race Simulation
Physiological Factors				
Energy	✗	✗	?	✗
Thermal	?	✗	?	?
Perceptions	✗	?	?	?§
Cardiovascular	?	?	?	?§
Cerebrovascular	✗	?	?	?§
Physical Factors				
Airflow	✓	✓*	✓*	✓*
Ambient Temperature & Humidity	✓	✓*	✓*	✓*
Motivation	✓	✗	✗	✗

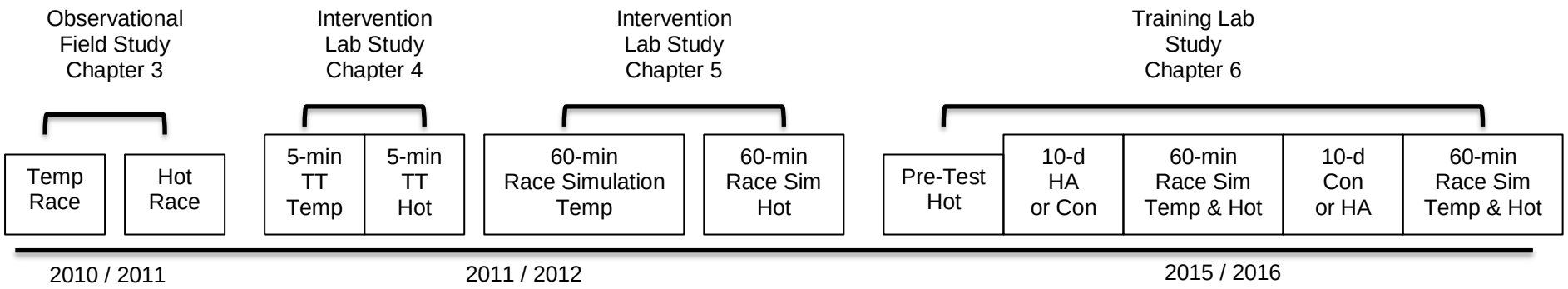

Figure 1.1: Timeline for each chapter and study that formed the thesis.

Chapter five examined performance changes and cardiovascular strain in controlled temperate versus hot environments during intense exercise effort that was long enough for performance to be predominantly aerobic but short enough to limit any potential influence from core temperature, brain metabolism, and the gastrointestinal (GI) system as limiting factors. It is well known that heat has a detrimental performance effect and that hyperthermia hinders brain function (e.g., motor output); however, the magnitude of effects on performance and brain physiology have seldom been investigated in concert. The hypothesis was that a hot environment would facilitate explosive (10-s) sprint power output but impair intense endurance (five-minute) performance in conjunction with lower cerebral oxygenation and blood flow.

The next chapter (six) investigated the performance and environmental factors that caused physiological strain sufficient to ultimately exceed athletes' capacities to complete a simulated triathlon cycle race in a hot environment. The purpose was to simulate the race performance and environments while measuring central and peripheral physiological variables to identify what mechanisms influenced performance over time. The hypothesis was that a 60-minute triathlon cycle race simulation that replicated the performance (oscillating power outputs including fixed pace and self-paced sections) and environmental (temperature, humidity, and wind speed) factors would have detrimental performance, cerebrovascular, cardiovascular and psychophysical impacts, including exhaustion.

Finally, we sought to determine if an appropriate (realistic and widely-effective) intervention could improve performance outcomes and the central physiological factors that had been the main focus of chapter six. The most effective option was considered to be heat acclimation, an intervention that had beneficial performance effects (Buchheit et al., 2011; Hue et al., 2007; Lorenzo et al., 2010; Racinais et al., 2014) but largely unknown effects on cerebrovascular function. In 2010 (at the inception of this thesis), hyperthermia was known to significantly impact cerebrovascular function (Nielsen and Nybo, 2003; Nybo et al., 2002a, 2002b; Rasmussen et al., 2004, 2010a), but adaptive effects of heat were largely unknown. [Since then, two studies have investigated how heat acclimation

influences cerebrovascular function rest (Fujii et al., 2015) and exercise (Karlsen et al., 2015a).] The study in chapter seven was therefore designed to determine whether heat acclimation improved performance power and had brain-related physiological and perceptual benefits in hot environments. These effects of heat acclimation were also tested in a temperate environment because ergogenic effects of heat acclimation for performance in hot environments has considerable scientific support (Chalmers et al., 2014; Garrett et al., 2012a, 2009, 2012b; Lorenzo et al., 2010; Nielsen et al., 1993; Racinais et al., 2015; Sawka et al., 1985), but had barely been studied in temperate environments and even now remains contentious (Minson and Cotter, 2016a, 2016b, Nybo and Lundby, 2016a, 2016b). The hypothesis was that short-term heat acclimation training would cause central (cerebrovascular) and thermoregulatory adaptations that would improve performance and reduce physiological stress in simulated race protocols for hot *and* temperate environments.

The concluding chapter contains recommendations for sport scientists conducting field research and laboratory studies and provides summary guidelines about exercise performance in temperate and hot environments, based on the research undertaken in this thesis. Implications and extended hypotheses are suggested to encourage technological development for field measurements and to stimulate future research into environmental exercise physiology and its application to field and laboratory studies.

2 LITERATURE REVIEW

2.1 Chapter Introduction

From 2009 until 2012 the New Zealand elite triathlon team was selected from race performances in New Zealand (Oceania Championships, Wellington) and Australia (ITU World Cup, Mooloolaba). Despite strong performances by established and potential elite athletes in New Zealand, many performed poorly or failed to finish the race in Australia. At the 2010 ITU World Cup in Mooloolaba, 15 New Zealand triathletes started. The best performance was 10th. Eight athletes required medical assistance and of those athletes five did not finish the race. Most athletes were unable to cope with the heat stress, citing heavy fatigue and breathing problems, especially during the 10-km run section. These outcomes stimulated the specific research interest, literature review, and consequent research questions.

The purpose of this review is to examine exercise and the environment as stressors to elite and highly-trained athletes using an aerobic training model extended from Coyle (1999) (Figure 2.1) and the general adaptation syndrome (Selye, 1950) to summarise current knowledge. The thesis was designed to examine novel questions posited in Chapter One, so this chapter details elite triathlon racing, physiological responses to short-duration, maximal-effort exercise and triathlon race simulations in different environments, and physiological adaptations to heat acclimation to augment performance in hot *and* temperate environments.

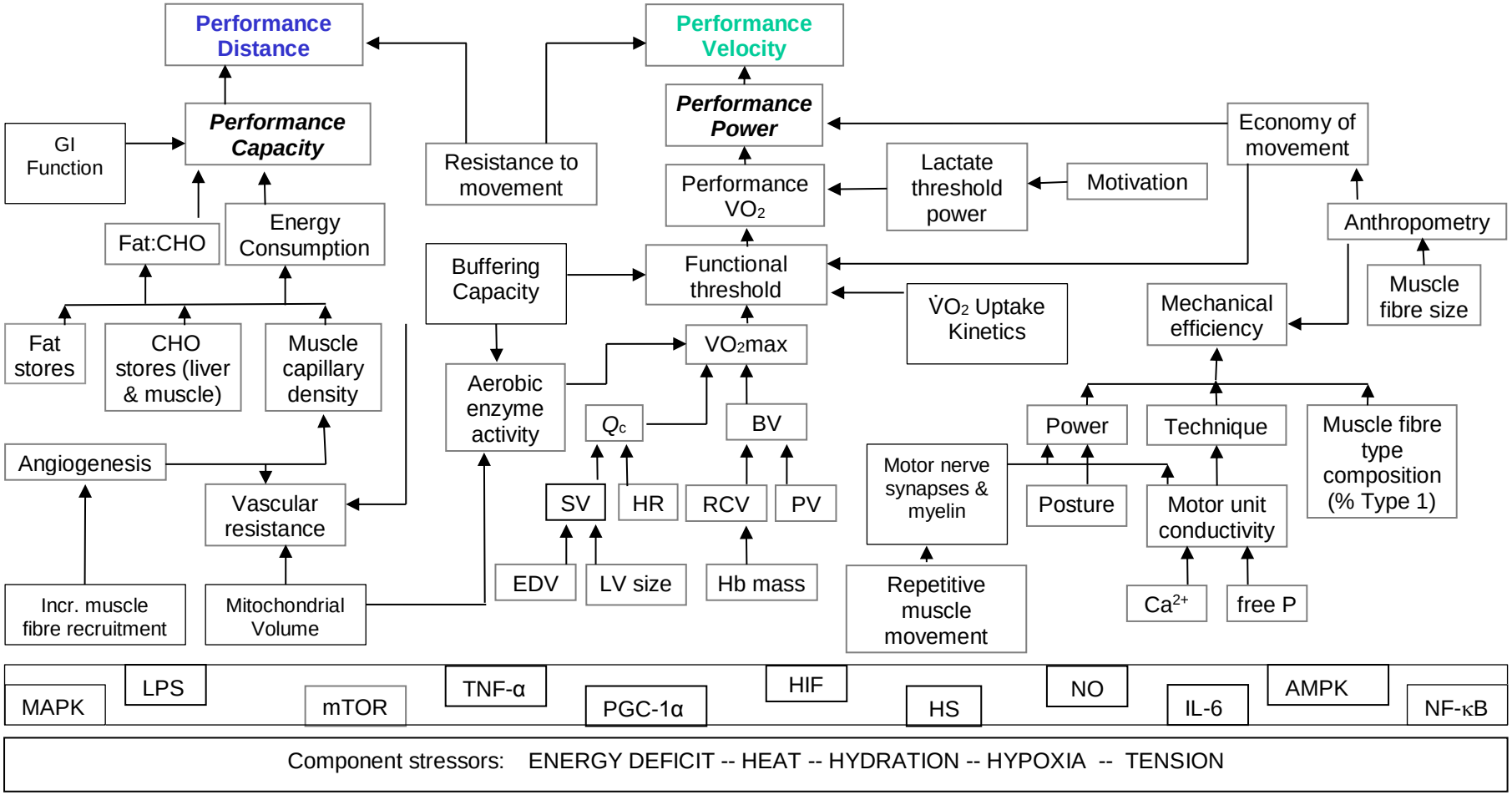

Figure 2.1: The aerobic performance model. Capacity is on the left, power in the middle, and structure on the right.

2.2 Elite Triathlon Racing

Elite triathlon competitions occur in diverse environments over various distances. This thesis focussed on the ITU World Series races over the draft-legal Olympic distance (1500 m swim, 40 km cycle, 10 km run). Successful performance is characterised by the ability to maintain high intensity [equivalent to second ventilatory threshold (VT2) or maximum lactate steady state (MLSS)] for long periods with exceptional movement economy (Sleivert and Rowlands, 2012) *and* the ability to perform short maximal efforts in three distinct disciplines. The strategy allows triathletes to stay near the front of the race while gaining the advantage from drafting (Bentley et al., 2008). Drafting reduces energy costs by up to 30% (Hausswirth and Brisswalter, 2012), and is most effective in swimming (Chatard et al., 1998; Peeling and Landers, 2009) and cycling (Faria et al., 2005a; Hausswirth et al., 1999) – less so in running, but at speeds > 6 $m \cdot s^{-1}$ (21.6 $km \cdot h^{-1}$) drafting reduced $\dot{V}O_2$ by 6.5% (Pugh, 1970, 1971) and improved 3000-m run performance (Zouhal et al., 2015).

2.2.1 Triathlon Swimming

From a mass start triathletes swim ~300 m to the first buoy. Swim position does not change substantially after the first buoy turn (Le Meur et al., 2009) because 1) it is easier to swim in another athlete's wash than overtake them (Chatard et al., 1998), and 2) performance in the subsequent cycle and run sections is superior when triathletes do not have to swim at maximum intensity – provided they are in the front group (Peeling et al., 2005). This potentially determines the overall race outcome (Vleck et al., 2006, 2008). Strategically, athletes must sprint from the starting gun to the first buoy, then reduce intensity through the rest of the swim leg slightly to hold their position without using anaerobic metabolism excessively and repeatedly. The thermal load during swimming depends primarily on exercise intensity, water temperature and whether a wetsuit is used (Kerr et al., 1998; Kreider et al., 1988a; Peeling and Landers, 2007; Trappe et al., 1995). Studies that have attempted to replicate triathlon race simulations have demonstrated end core temperatures at 37.7 ˚C (Kreider et al., 1988a) and 38.4 ˚C (Peeling and Landers, 2007) for 750-800 m distances, however, the

participants from these studies were swimming slower (1:22-1:23/100 m) than elite male (1:08-1:14/100 m) and female (1:16-1:23/100 m) triathletes. Presumably athletes swimming up to 18% faster over twice the distance would experience a similar if not greater core temperature increase, but this would be dependent on water temperature as water is ~25 times more conductive than air and thus also has much higher convective heat transfer (Nadel et al., 1974; Ramires et al., 1995), while sweating is ineffective.

2.2.2 Triathlon Cycling

Triathletes complete the transition to the non-drafting cycling leg (~30-90 s) with the largest time component being the run from the water's edge to the bike. Equipment innovations have economised the change from swimming to cycling down to seconds, even when wearing a wetsuit. Physiologically this is an exceptionally stressful section of the race due to the change in posture from prone exercise supported by hydrostatic pressure (swimming) to upright weight-bearing exercise (running) that cause a considerable spike in heart rate (HR) and drop in mean arterial pressure (Candidate's unpublished observations). The first lap of the cycle is usually the fastest (Le Meur et al., 2009) as athletes utilise the beneficial drafting from a peloton (Chatard and Wilson, 2003; Hausswirth et al., 1999) and try to maintain their position in the front group, or try to catch up to it (Vleck et al., 2006). After the first lap heart rate and power output progressively decreased with each lap (Bernard et al., 2009; Le Meur et al., 2009). The multi-lap and drafting components of the cycle leg require multiple stochastic power outputs. Power profiles for five elite male triathletes across seven international races demonstrated over 30 supramaximal efforts above 600 W with 18% of total time above 100% maximum aerobic power (Etxebarria et al., 2014a). Furthermore, the frequent and large cycling power variations produced by triathletes in drafting races produced a higher workload than cycling in non-drafting races at constant power (Etxebarria et al., 2014a).

2.2.3 Triathlon Running

The second transition follows cycling, and is the shortest section of the race. Running after cycling in a triathlon race may be especially challenging due to

poor blood flow redistribution (Kreider et al., 1988b), leg muscle fatigue (Hausswirth et al., 1997), ventilatory muscle fatigue (Hue et al., 1997), higher core temperature (Kreider et al., 1988a), reduced pulmonary compliance and hypoxaemia (Caillaud et al., 1995) and elevated heart rate caused by dehydration (Guezennec et al., 1996; Hausswirth et al., 1996, 1997). Similar to the swim and bike legs, triathletes also employ a positive pacing strategy on the run (Hausswirth et al., 2009; Le Meur et al., 2011; Vleck et al., 2008), punctuated by speed changes observed in middle distance running (Foster et al., 1994). The physiological demands combined with the negligible assistance from drafting on the run section (women ~17 $km \cdot h^{-1}$, men ~19 $km \cdot h^{-1}$) and aggressive pacing early in the run generates substantial fatigue and forces athletes to self-pace at the limit of their physiological capability and motivational psychology to achieve their best race outcome (Etxebarria et al., 2013). Unsurprisingly, performance in the run section has the highest correlation with finishing position (Vleck et al., 2008).

2.2.4 Triathlon Race Conditions

2.2.4.1 Field Environment

Triathlon is a summer sport with competitions typically in temperate and warm conditions (Candidate's unpublished observations; range: 11 to 35 ˚C), however it is races in cool and hot environments that cause the greatest thermal strain for athletes from hypothermia (e.g., 2008 ITU World Championships, Vancouver, Canada; 2011 ITU World Series, London, UK) and hyperthermia (e.g., 2012 ITU World Series, Yokohama, Japan; 2014 ITU World Series, Chicago, USA). Environmental conditions may be highly variable day-to-day. On the first day of racing at the 2011 ITU World Championships in Beijing, China the under-23 men competed in 27.8 ˚C ambient temperature, 26% relative humidity, and 35.5 ˚C black bulb temperature. The next day the under-23 women competed at the same time of day in 15.7 ˚C ambient temperature, 87-100% relative humidity (intermittent rain) and 15.7 black bulb temperature. In both races athletic performance was impeded by the environmental conditions, with 16 of 67 men and 4 of 37 women unable to finish the race (DNF). Not all the DNF results were caused by the conditions, but the environment did influence the race outcome for

many athletes – though notably more for the race in hot conditions. Anecdotally, many elite triathletes have noted that their performance has been negatively affected, and their perception of workload increased when racing in temperatures >25 ˚C. This has been replicated in marathon runners whose performance times were 2-3% slower when wet bulb globe temperature (WBGT) was greater than 20 ˚C (Ely et al., 2007), and in elite triathlon racing where total time varied by 2% in hot conditions compared with 1.6% variation in normal conditions for non-drafting races (Paton and Hopkins, 2005). Competitive performance in hot compared with temperate environments is examined in detail in Chapter 3 for an elite group of New Zealand triathletes who experienced detrimental performance outcomes in ambient race conditions that were not demonstrably adverse (WBGT: 20.9 ˚C).

Athletes preparing for competition in cold environments cannot perform effective acclimatisation or acclimation because humans do not adapt physiologically to the cold (Brazaitis et al., 2014; Tipton et al., 2013). Instead behaviour is crucial. Triathletes competing in cool and cold environments have used additional clothing to effectively minimise heat loss, especially during the cycling section where heat loss is substantial from relative air velocity and the exposed skin surface area. Conversely, preparation for competition in warm and especially hot environments can be highly effective through physiological adaptation, known as heat acclimation (training in a simulated environment) or acclimatisation (training in the environment itself). Heat acclimation/acclimatisation may confer adaptations including: higher plasma volume (Senay et al., 1976), lower resting core temperature (Buono et al., 1998; Nielsen et al., 1993), improved movement economy (Sawka et al., 1983a; Young et al., 1985), enhanced sweat rate with a lower osmolality (Cheung and McLellan, 1998; Nielsen et al., 1993), muscle glycogen sparing (Febbraio et al., 1994a; Garrett et al., 2012a; Young et al., 1985), lower blood lactate concentration ([La]) at any given exercise intensity (Nielsen et al., 1993; Young et al., 1985), improved vascular function (Lorenzo and Minson, 2010), and reduced thermal strain and effort perception (Cheung and McLellan, 1998; Cotter et al., 2001; Racinais et al., 2015). Many of these adaptations are already present in trained individuals (Taylor, 2000), but can be developed further with the added, exogenous heat stress. Cool environments

can be adequately dealt with by behaviour and clothing modifications whereas hot environments permit few behavioural but multiple physiological adaptations in an endurance racing context.

2.2.4.2 Race Simulations

Study designs with capable participants and well-designed protocols produce race simulation results and conclusions that can be accurately transferred to real-world competition. Triathlon race simulations that have included swimming have investigated swim suits (Peeling and Landers, 2007), the performance, core and skin temperature comparisons between wetsuit and non-wetsuit swimming (Kerr et al., 1998; Trappe et al., 1995), or as a pre-load to the cycle and run legs (Hausswirth et al., 1996; Kreider et al., 1988a; Laursen et al., 2000). Studies conducted in swimming pools lack the unique water conditions and pacing typical to triathlon swimming that requires a mass start and ability to swim effectively in a large group of other athletes using drafting strategies to reduce metabolic load and improve performance (Bassett et al., 1991; Chatard et al., 1998). The swim speeds were also too slow to transfer the findings to elite performance: 1:20 to 1:35 per 100 m (Laursen et al., 2000; Peeling and Landers, 2007; Peeling et al., 2005; Trappe et al., 1995) for an Olympic distance race (1500 m swim). In international triathlon events the slowest elite men swim 1:11-1:15 per 100 m and the slowest women swim 1:18-1:22 per 100 m at VT2, which may be as high as ~90% $\dot{V}O_2$ max (Candidate's unpublished observations). Core temperature increased 0.72 ˚C during an 800-yard non-wetsuit swim in 23 ˚C water (Kreider et al., 1988a) and 0.8 ˚C after 20-minutes (~1250 m) of race pace swimming in 33 ˚C water (Bradford et al., 2015). Unfortunately participants in both studies swam slower than 1.25 $m.s^{-1}$ (1:20 per 100 m). Elite triathletes produce substantially more speed and have superior abilities to offload heat through lower body fat composition and potentially higher surface area to mass ratios. Additionally their aerobic training adaptations also improve thermoregulation (Taylor and Cotter, 2006). Core temperature has been assessed to compare wetsuit versus non-wetsuit swimming, but the results have limited application to elite athletes because the water and ambient air temperatures exceeded the

maximum for wetsuit use in ITU elite racing (Kerr et al., 1998; Trappe et al., 1995), at least before 2017.

Swim intensity may have a strong impact on average cycling power and race outcome (Peeling and Landers, 2009) because swim performance does not necessarily win a race, but a poor swim may remove an athlete from contention. A 3000-m time trial at 1:35 per 100 m pace in an outdoor 50-m pool had no impact on power output for a 3-hour bike time trial (Laursen et al., 2000). However, swimming at maximum effort impaired average cycling power and overall race time compared with swimming at 90% or 80% of maximum effort in a sprint triathlon race (Peeling et al., 2005). Further, triathletes cycling after swimming in a triathlon simulation produced significantly less power than the same cycling task without a swim pre-load (Delextrat et al., 2005a; Kreider et al., 1988a). Athletes who could draft during the swim experienced less drag, lower swim intensity, improved cycling biomechanics and reduced fatigue at the start of the bike (Delextrat et al., 2005b); the benefits were similar if they wore a wetsuit during the swim (Delextrat et al., 2003).

Like swimming, cycling has been examined as a pre-load to the run (Bernard et al., 2003, 2007; Chan et al., 2008; Etxebarria et al., 2013; Galy et al., 2005; Garside and Doran, 2001; Guezennec et al., 1996; Hausswirth et al., 1996, 1997, 2001, Hue et al., 1997, 1999; Miura et al., 1999; Suriano et al., 2007; Vercruyssen et al., 2002, 2005). Drafting during the cycle leg had a strongly beneficial performance effect on the subsequent run section (Hausswirth et al., 1999, 2001). The standard protocol for the bike section of race simulations has been time trials at 70-80% $\dot{V}O_2$ max (Bernard et al., 2003; Brisswalter et al., 2000; Chan et al., 2008; Galy et al., 2003, 2005; Garside and Doran, 2001; Gonzalez-Haro et al., 2005; Hue et al., 1997; Kreider et al., 1988a; Peeling et al., 2005; Vercruyssen et al., 2002, 2005). However, the performance demands for triathlon racing at elite level require explosive power at varied cadences combined with the ability to recover quickly while riding in small or large groups, which is a more severe workload than cycling at constant power (Etxebarria et al., 2013, 2014a). Research that has investigated cadence, body position, frame design, environmental conditions, drafting, and pacing strategies to optimise

cycle and subsequent run performance may not be applicable to elite triathletes unless they are competing in non-drafting events. Additionally, triathlon simulation studies that have investigated performance and physiological responses do not mention airflow in their methodology, or have used commercial fans that deliver 1-2 $m.s^{-1}$ of airflow to a small frontal surface area for cycling and running.

Triathlon run performance is considerably influenced by specific exercise intensity and mode during pre-load. Variable cycling power generated greater physiological and perceptual stress responses compared with constant load, which consequently hindered run performance (Etxebarria et al., 2013). This was determined by collecting power data from Olympic-distance ITU triathlon races (Etxebarria et al., 2014a) to create a race-specific cycle simulation with a variable power profile (Etxebarria et al., 2014b) that demonstrated ~2% slower self-paced run speed after cycling (Etxebarria et al., 2013). Furthermore, sprint distance triathlon running (5-km distance) was 4.4% slower immediately after completing the simulated (variable) compared with constant load protocol (Bernard et al., 2007). Running in a triathlon was more demanding to the cardiovascular system, thermoregulation, and movement economy compared with the same running task with no pre-load of swimming and/or cycling when environmental conditions were replicated by running on an outdoor track (Bernard et al., 2007; Guezennec et al., 1996; Hausswirth et al., 2009), and not indoors on a treadmill (Chan et al., 2008; Kreider et al., 1988a). Unfortunately other race simulations for sprint and Olympic distances have been conducted at exercise intensities that were substantially lower than race effort and with insufficient airflow to replicate the appropriate thermoregulatory demand (Chan et al., 2008; Kreider et al., 1988a; Miura et al., 1999). In summary, race simulation studies involving highly-trained triathletes that accounted for the mode, intensity and environmental stressors demonstrated accurate findings that were applicable to real world performances.

2.2.4.3 Published Race Data

Race data have been collected from elite athletes during international races where ecological validity and motivation were very high for the athletes, but

comprehensive information was difficult to collect for the investigators. Bernard et al. (2009) measured ten French triathletes competing at the 2006 Beijing ITU World Cup (the same course as the 2008 Olympics) using power and heart rate during the cycle leg of the race to compare with their individual physiological thresholds. Both power and heart rate were highly variable, with 51% of the power distributed below the first ventilatory threshold and 17% above their maximal aerobic power (the highest power they could maintain for one min). Triathletes employed a positive pacing strategy, producing the greatest power, heart rate, and speed at the start; which significantly decreased throughout the bike leg. Etxebarria et al. (2014a) also demonstrated highly variable cycling power profiles during racing. Other studies have investigated pace changes in each discipline and between sexes in ITU Triathlon World Cup races, characterising the unique performance demands. Triathletes adopted a positive pacing strategy in each discipline (swim, bike, run; Le Meur et al., 2009) so that race position (especially for swimming and running) determined their position at the end of the discipline and overall in the race (Le Meur et al., 2009; Vleck et al., 2006, 2008). However, the optimal pacing strategy for each discipline and distance remain unclear (Wu et al., 2014).

2.3 Stress

Stress is defined as the factors imposed on the body that impact on homeostasis (i.e., induce strain) and can generate adaptation (deVries, 1966). Homeostasis encapsulates the physiological regulation of at least seven variables critical to survival: 1) plasma osmolality (especially sodium ion concentration), 2) carbon dioxide (CO_2) partial pressure and pH (the bicarbonate equation, maintaining the balance of CO_2 and H^+), 3) oxygen (O_2) partial pressure (especially haemoglobin mass and ventilation adequacy), 4) mean arterial blood pressure, 5) cell volume (intracellular fluid), 6) energy substrates (especially glucose metabolism), and 7) core temperature or perhaps mean body temperature. The multifactorial response to stress means that non-specific damage and defence drive a general adaptation syndrome, however, specific physiological reactions (to each stressor) are conditioning factors that stimulate the way general adaptation occurs (Selye, 1950). Further, exposure to a stressor may not cause the same

damage or activate the same cellular defence mechanisms due to factors independent to the stressor such as environment and energy status. This also explains why one individual exhibits a reaction and another does not when exposed to the same stress (Selye, 1950). Agonist and antagonist responses act on the target organ to initially stabilise, then adjust its response to the injury (Figure 2.2). The General Adaptation Syndrome is effective for short-stress responses, however, the stress hormones (adrenocorticotropic hormone, catecholamines, glucocorticoids) cause systemic complications when they are released for a prolonged period (Selye, 1950).

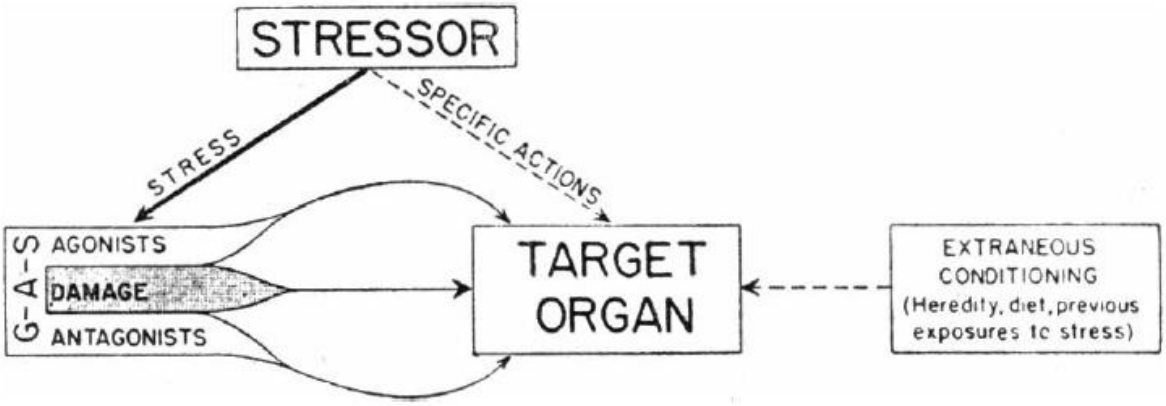

Figure 2.2: The general adaptation syndrome and the roles of damage and action at the target organ(s) in response to stress (Selye, 1950). Reproduced with permission.

Exercise is stressful and thus also provides protection against stress, fortifying the resistance stage in Selye's model (Michael, 1957). The stressors proposed in the aerobic performance model (Figure 2.2) are integral within exercise itself, and each one appears to play a role in exercise fatigue as well as in physiological adaptations. Too much stress may cause maladaptation. The discrete stressors include energy deficit, heat, hydration, hypoxia, and mechanical tension.

Stress caused by excessive exercise coupled with inadequate rest and recovery may cause inflammation in skeletal muscles. The inflammatory response is determined by exercise intensity, volume, muscle mass utilised, an individual's endurance fitness and dietary factors. The inflammatory cytokines themselves increase in differing quantities after strenuous exercise: interleukin 1 beta (IL-1β;

1-fold), tumour necrosis factor alpha (TNF-α; 2-fold), and interleukin 6 (IL-6; up to 128-fold; Febbraio and Pedersen, 2005; Pedersen and Febbraio, 2008; Robson-Ansley et al., 2009; Steinacker et al., 2004). Initial inflammation may be localised, and continued stress may develop into chronic and systemic inflammation (Pedersen and Febbraio, 2008; Pedersen and Hoffman-Goetz, 2000). Systemic inflammation may contribute to excessive fatigue because the stress imposed on the body (energy deficit, heat, hypoxia, hydration [hydrostatic and osmotic pressures], and mechanical tension) causes excessive energetic, structural, neuroendocrine or immunologic strain. That is, strain responses including altered metabolism, cellular transport, cellular repair and protein synthesis, act to adapt to the imposed stress (Pedersen and Febbraio, 2008; Pedersen and Hoffman-Goetz, 2000; Steinacker et al., 2004), but when these processes are overwhelmed adaptation is impeded or prevented. The inflammatory cytokine theory of overtraining (Smith 2000) is based on this construct, and posits IL-6 as a key mediator of such maladaptation. However, IL-6 alone has not been a definitive overtraining marker in field studies with elite athletes. This may be because the hormonal milieu is too complex to identify a single marker.

2.4 Fatigue

Fatigue experienced during exercise has been defined as a failure to maintain the required or expected force or power output (Edwards, 1983). The mechanisms may be centrally driven via factors such as increased serotonin in different brain regions (Davis and Bailey, 1997), or peripheral because the surface membrane, excitation-contraction coupling, or the metabolic state of the individual muscle cells may be disturbed (Allen et al., 2008; Fitts, 1994). Noakes et al. (2005) hypothesised that physical activity is controlled by a central governor in the brain, which determines the work rate for the anticipated exercise time period and termination point to maintain homeostasis, particularly for multiple stressors such as exercise combined with hypoxia or heat (Kayser, 2003). One landmark study in gaining understanding of the links between peripheral and central mechanisms of fatigue is that by Amann and colleagues (2011). These authors examined the fatigue and homeostasis responses to severe exercise,

demonstrating that central and peripheral systems communicate. They did so by blocking the sensory (but not motor) pathways during a 5-km cycling time trial using an opioid analgesic (fentanyl) compared with a placebo. Performance in the first half of the trial was superior with the analgesic, followed by rapid deterioration in the second half, presumably because homeostasis could not be maintained. Performance deterioration may have occurred because the central nervous system (CNS) could not upregulate homeostatic control processes including ventilation and blood flow, and peripherally because individuals exceeded their individual threshold for peripheral locomotor muscle fatigue – a level that an individual approaches at volitional exhaustion or the end of a time trial, but never exceeds (Amann, 2011). Further, the CNS and peripheral muscle may maintain a reserve capacity to avoid catastrophic fatigue (as defined by Edwards, 1983: muscular contraction disrupted by biochemical factors). Instead, at exhaustion exercise may be terminated voluntarily or intensity reduced to complete the task (Noakes et al., 2005). Peripheral fatigue may be the primary limitation to exercise performance for short and severely intense exercise when muscle homeostasis is disrupted (McKenna et al., 2008), whereas central fatigue may be the major limitation for low intensity exercise combined with high environmental stress (Nybo and Nielsen, 2001a). Finally, Hargreaves (2008) has posited (with considerable support) that exercise performance and fatigue are complex multifactorial mechanisms that require an eloquent integrative physiological model to understand. Conjecture remains between many authors about a complete definition for fatigue. The central and peripheral fatigue mechanisms that contribute to exercise termination and maintaining homeostasis are discussed extensively elsewhere (Amann, 2008; Ekblom, 2009a, 2009b, 2009c; Gandevia, 2001; Hargreaves, 2008; Noakes and Marino, 2009b, 2009a, 2009c; Shephard, 2009).

Volitional exhaustion is not the ultimate limit of muscle capacity (Amann, 2011), and catastrophic fatigue is rare, as is evident in several respects. First, adenosine triphosphate (ATP) concentrations remain above 50% of resting concentration during fatiguing exercise (Noakes and Gibson, 2004). Second, at volitional exhaustion from prolonged exercise, less than 30% of the available motor units were recruited in recreational athletes (Gibson et al., 2001; Kay et al., 2001).

Third, individuals exhausted by 10 min of cycling could produce sprint power output 300% higher immediately following volitional exhaustion (Marcora and Staiano, 2010). And finally, the CNS adjusts exercise intensity, metabolism and performance power by altering the number of motor units recruited throughout a task (Noakes et al., 2005). It takes an exceptional circumstance (such as the experiments described by Amann et al., 2011) to cause catastrophic fatigue. Volitional exhaustion may be strongly correlated to perceived effort (Marcora and Staiano, 2010). The ultimate limitation may in fact be the athlete's will to continue, because: 1) the effort required to continue exceeded the greatest effort they were willing to perform (potential motivation) or, 2) the effort required was perceived as beyond their ability (Wright, 1998). Exceptional circumstances may drive highly physically- and mentally-trained individuals to exceed their individual threshold for fatigue and lead to catastrophe in an event that imposes multiple stressors and has powerful personal motivation (e.g., World Championships, Olympic Games). Examples of physiologically catastrophic responses that force an athlete to terminate exercise include rhabdomyolysis (Knochel, 1990), heat exhaustion or heat stroke (discussed below; Bouchama and Knochel, 2002; Lim and Mackinnon, 2006).

2.5 Exercise Duration, Determinants, and Limitations

Exercise conveys multiple stressors to the body simultaneously that, when repeated, has beneficial outcomes including protection from oxidative stress, enhanced insulin-responses in skeletal muscle, and improved glucose uptake (Hawley and Lessard, 2008). A single exposure to high-intensity treadmill running can increase growth hormone secretion (Kraemer et al., 2004), while chronic exercise bouts stimulated inflammation at multiple organs and systems and initiated activity in the growth hormone - insulin growth factor - insulin axis (Roemmich, 2005). The exercise frequency, intensity, duration, and mode determine physiological responses from the cellular to systemic level.

Exercise to exhaustion at fixed intensity and self-paced exercise protocols are negatively affected by heat (Cheung and McLellan, 1998; Galloway and Maughan, 1997; Gonzalez-Alonso et al., 1999; Marino et al., 2004; Nielsen et al.,

1990; Nybo and Nielsen, 2001a; Tatterson et al., 2000; Tucker et al., 2004). However, fixed intensity exercise protocols evaluate exhaustion from constant metabolic demand that an individual cannot regulate, whereas self-paced intensity evaluates how an individual manages fatigue whilst maintaining the required performance (Schlader et al., 2011a). Crucially, the results from fixed-intensity protocols transfer poorly to real-world competition, most notably because participants are unable to perform a maximal effort end-spurt at the finish that characterises endurance performances. Fixed-intensity protocols push individuals to the maximum capacity of physiological strain that may be attained immediately before thermoregulation or other physiological regulators are compromised (Schlader et al., 2011a), which has considerable advantage in a laboratory setting where robust measurements can track the limits of an individual's physiological capacity. By contrast, self-paced intensity is regulated at a physiological strain level lower than at termination of fixed-intensity exercise and accurately describes how an individual responds physiologically to the race pace and environment (Byrne et al., 2006; Lee et al., 2010). Pragmatically, both fixed- and self-paced intensities should be included in the experimental design to gather a comprehensive dataset about the interplay between performance and physiological strain. When individuals are allowed to self-pace in the laboratory or real-world competition they use perceived exertion and anticipation to thermoregulate and manage central fatigue to attain optimal performance without over-reaching to an intensity that causes task failure (Lee et al., 2010; Schlader et al., 2010b; Tucker, 2009; Tucker et al., 2006a). In competition individuals also have the capacity to produce a maximal effort end-spurt that may be critical to performance outcome (Foster et al., 1994; Tucker et al., 2006a).

2.5.1 Short-Duration Maximal-Intensity Exercise

Sprint power is crucial for prolonged aerobic endurance events to respond to course topography, competitive tactics, or the final sprint finish that require maximal anaerobic effort (Fukuba and Whipp, 1999). An all-out effort over several seconds is sufficient to consume all the ATP stored in human skeletal muscle and cause rigor, but is protected against by ATP resynthesis via the anaerobic alactic, anaerobic glycolytic, and aerobic systems, at their sequentially

lower rates (Gaitanos et al., 1993; Newsholme and Start, 1973). The type of effort an individual performs may be defined by the predominant energy systems and the physiological responses from that effort. A sprint effort requires all-out exercise for a duration that can almost be sustained to the finish (≤ 10 s) (Girard et al., 2011) and utilises predominantly (~90%) the anaerobic alactic system (ATP and creatine phosphate). Stressors including macronutrient energy deficit, hypoxia, and dehydration generally do not influence single sprint performance of ~10-s duration. Further, elevated body temperature from exposure to a hot environment and/or vigorous warm-up may enhance sprint performance by way of higher nerve conductivity and muscular tension (Asmussen and Bøje, 1945; Ball et al., 1999).

Beyond 10 s the alactic contribution declines as the anaerobic glycolytic-lactic system predominates. By two min of maximal effort the aerobic system is dominant (Astrand and Rodahl, 1977). Maximum aerobic power ($\dot{V}O_2$ max) is defined as the highest rate that oxygen (O_2) can be consumed and utilised by the body during severe exercise, and requires longer than two min, but shorter than 15 min due to the fatigue developed by such high intensity (Bassett and Howley, 2000; slightly modified from the classic studies by Hill and Lupton, 1923; Hill et al., 1924). It is limited largely by the cardiorespiratory system's ability to transport O_2 to the muscles, a theory posited by Hill et al. (1924) and confirmed with laboratory testing (Astrand, 1952; Costill et al., 1973; Mitchell et al., 1958; Rowell, 1986; Saltin and Strange, 1992; Taylor et al., 1955).

Nybo et al. (2001) and Gonzalez-Alonso and Calbet (2003) confirmed Hill's theory and demonstrated that elevated core temperature or at least skin temperature further reduced $\dot{V}O_2$ max for maximal effort cycling. Gonzalez-Alonso and Calbet (2003) identified that ambient heat stress pushed the cardiovascular system to its absolute regulatory limit, lowering $\dot{V}O_2$ max from 4.72 to 4.28 $L \cdot min^{-1}$ and time to exhaustion from 7.63 to 5.45 min. Greater reductions in leg blood flow and mean arterial blood pressure decreased skeletal muscle blood flow, O_2 delivery and uptake because O_2 extraction by active limbs was already maximised (at ~90%). At exhaustion there were significant declines in leg muscle blood flow and hence muscle O_2 delivery. However, muscle energy

stores were not depleted (Gonzalez-Alonso and Calbet, 2003). The study design effectively controlled the thermal load to the core and skin with a water perfused suit that covered the upper body. Further, the experiments demonstrated that ambient heat stress increased cardiovascular strain and decreased oxygen delivery to muscles that accelerated fatigue. However, wearing a suit obstructed heat offload and may have altered skin blood flow (BF_{Skin}), core temperature, and skin temperature in a way that lacked ecological validity.

Maximal exercise efforts of approximately five min duration are limited by the cardiovascular system, more so in a hot environment (Gonzalez-Alonso and Calbet, 2003; Nybo et al., 2001). However, core temperature, cerebrovascular, and gastrointestinal (GI) function seem unlikely to contribute to impaired performance because severe exercise intensity that causes substantial increases in minute ventilation ($\dot{V}_E$), decreases in the partial pressure of carbon dioxide ($PaCO_2$; Imray et al., 2005; Nielsen, 1999), and blood flow redistribution (Rowell, 1974) occur no matter what the environmental conditions. The exercise intensity is the primary stressor; it is only when duration becomes prolonged enough to significantly raise core and brain temperature and accrue GI heating and ischaemia that they may contribute to environment-related exercise fatigue.

2.5.2 Prolonged High-Intensity Exercise

Prolonged exercise is more reliant on systemic integration to maintain homeostasis and contribute to aerobic endurance performance by managing one or multiple stressors during prolonged and maximal endurance exercise. However, fatigue inevitably accumulates as exercise duration increases (Hill, 1925). Effects of heat on fatigue are explained separately (Section 2.6), primarily because it formed the crux of this thesis for the reasons explained in Chapter 1, i.e., fitness is essentially already maximised in elite (tri)athlete performance, but environmental adaptation isn't necessarily optimal.

2.5.2.1 Determinants of Aerobic Performance

Performance velocity and performance distance are inversely related and govern aerobic performance (see Figure 2.1). Performance $\dot{V}O_2$ directly influences

performance power and velocity, and it is determined by four factors critical to maximal-effort prolonged aerobic exercise: $\dot{V}O_2$ max, lactate threshold, movement economy, and oxygen uptake kinetics (Coyle, 1999; Whipp et al., 1982). Performance $\dot{V}O_2$ is produced by the heart's ability to produce a high cardiac output ($\dot{Q}$) combined with elevated total haemoglobin (Hb) mass, muscle blood flow, and transfer of O_2 at the lung and muscle (Bassett and Howley, 2000; Dempsey, 1986; Mitchell et al., 1958; Rowell, 1986; Saltin and Strange, 1992). The training adaptations specific to enhancing performance $\dot{V}O_2$ include increased capillary density, mitochondrial density, stroke volume, and blood volume (BV; Costill et al., 1976). The performance distance (and therefore time) become critical because events requiring longer than ~10 min of dynamic exercise are performed at aerobically-submaximal intensities: 5-km running just below 100% $\dot{V}O_2$ max and marathon running at 75-85% $\dot{V}O_2$ max in highly-trained runners (Bassett and Howley, 2000; Costill et al., 1973).

Anaerobic threshold, equivalent to the VT2 and the maximum lactate steady state before an exponential increase in blood lactate concentration, represents the highest steady-state intensity that a well-trained individual can maintain for up to 120 min (Holloszy and Coyle, 1984; Holloszy et al., 1977). It is a highly trainable threshold developed by an increased oxidative capacity in the skeletal muscle (Holloszy and Coyle, 1984; Holloszy et al., 1977) that can increase from 60% of $\dot{V}O_2$ max in untrained to 75-85% $\dot{V}O_2$ max in well-trained athletes (Farrell et al., 1993).

Oxygen uptake kinetics limit performance $\dot{V}O_2$ at two intensities. The first is at exercise onset when tidal volume and breathing frequency rapidly upregulate to help to deliver sufficient oxygen to match metabolic demand (Eldridge, 1994). The second performance limitation occurs at intensities above anaerobic threshold when the mechanical effort of breathing combined with exercise drives the total $\dot{V}O_2$ closer to $\dot{V}O_2$ max (Hill and Smith, 1999; Poole et al., 1988; Whipp, 1994). The upward $\dot{V}O_2$ drift observed during constant power/pace exercise has been described as the $\dot{V}O_2$ slow component (Jones and Carter, 2000). The mechanisms that underpin the $\dot{V}O_2$ slow component are not completely

understood (Whipp, 1994) and training may (unpublished observation in Jones and Carter, 2000) or may not (Davis et al., 1979) alter it.

Movement economy is the energy or oxygen cost (expressed per unit distance) of moving at a given velocity (Bassett and Howley, 2000; Conley and Krahenbuhl; Costill et al., 1973; Joyner, 1991). It is highly variable between individuals, with 20-30% differences for running (Coyle, 1995), and 30-40% for cycling (Farrell et al., 1993; Joyner, 1991). Movement economy can also considerably increase for highly-trained and elite athletes. An eleven-year longitudinal case study for a marathon world record holder observed a 15% improvement in running economy from 205 $mL \cdot kg^{-1} \cdot km^{-1}$ in 1992 to 175 $mL \cdot kg^{-1} \cdot km^{-1}$ in 2003 (Jones, 2006). Economy in highly-trained cyclists was superior for those with a higher proportion of type I (slow twitch) muscle fibres in the vastus lateralis (Coyle et al., 1992). Further, self-selected cycling cadence improved economy in athletes with higher proportions of myosin heavy chain I in type I fibres (Hansen et al., 2002).

2.5.2.2 Limitations to Aerobic Performance: Performance Capacity

Performance distance is directly impacted by performance capacity, often defined as endogenous glycogen depletion (Christensen and Hansen, 1939; Coyle et al., 1983, 1986; Davies and Thompson, 1979), which is ultimately determined by an individual's initial glycogen content, vasculature, GI system, substrate utilisation, O_2 delivery, mitochondrial density and mitochondrial enzyme activity to sustain exercise intensity (Figure 2.1; Coyle, 1999). The primary intervention that enhances performance capacity is adequate nutritional intake and exercise training, while multiple factors limit it, particularly glycolysis.

2.5.2.3 Limitations to Aerobic Performance: Exercise-Induced Arterial Hypoxaemia

Exercise-induced arterial hypoxaemia (EIAH) is arterial oxygen desaturation during intense exercise; it significantly restricts aerobic performance capacity in trained athletes with high $\dot{V}O_2$ max (Harms et al., 1998a, 2000a; Williams et al., 1986). Arterial desaturation caused by EIAH is defined as a 3-4% decrease below resting concentration, and classified as mild (93-95%), moderate (88-

93%), and severe (< 88%; Dempsey and Wagner, 1999). The mechanisms that cause EIAH and the negative impact it has on $\dot{V}O_2$ max and performance remains unclear, but may be influenced by the physiological need to limit the work load that locomotor muscles can generate (Dempsey and Wagner, 1999). Further, airway size and lung structure may also constrain maximal exercise in highly trained female athletes (Guenette and Sheel, 2007; Harms et al., 1998a, 2000a). The EIAH response significantly increases locomotor muscle fatigue because not enough oxygen can be delivered to the lungs and then transported to contracting skeletal muscles (Amann et al., 2006; Kayser et al., 1994). Maximal efforts at low altitude (580 m) substantially reduce $\dot{V}O_2$ max for trained cyclists (Gore et al., 1996), indicating that O_2 loading onto haemoglobin may be a limiting factor in intense exercise; a theory supported by studies that have used hyperoxic gas mixtures and enhanced haemoglobin concentration to alleviate EIAH (Amann et al., 2006; Knight et al., 1993; Thomson et al., 1982). Performing maximal or prolonged strenuous exercise in a hot environment increases oxygen consumption (Febbraio et al., 1994a; Nielsen et al., 1990; Young et al., 1985), presumably exacerbating EIAH. Few studies have investigated whether heat has a significant influence on $\dot{V}O_2$ max or performance for short-duration maximal-effort exercise or prolonged endurance exercise in a temperate compared with hot environments.

2.6 Heat

This section addresses humans exercising in hot environments, and their responses to heat and exercise as combined stressors. Heat is produced by chemical reactions driving adenosine (ATP) resynthesis (Kushmerick, 2011), which produce > 75% energy as heat, and < 25% as movement (Coyle et al., 1992). Exercise is therefore the primary heat stress in that its metabolic heat production increases core temperature (Nielsen, 1938; Robinson, 1963). Heat stress increases proportionally with exercise intensity (Nielsen and Nielsen, 1962; Saltin et al., 1972) and the physiological and physical potential for offloading that heat. Dynamic exercise that activates most of the body's skeletal muscle (e.g., cycling, running) produces the greatest thermal stress. When such exercise is intense or intense and sustained, heat production may exceed the

body's capacity to release heat to the environment; a common situation for 10-km to marathon-distance running races and triathlon races. Heat is offloaded via conduction, convection, radiation, and - most effectively during competitive terrestrial exercise - evaporation to the environment (Stitt, 1993). Evaporation from insensible heat loss via the upper airways through increased minute ventilation is driven by a higher respiratory frequency (The Commission for Thermal Physiology of the International Union of Physiological Sciences (IUPS Thermal Commission), 1987), a highly effective heat offload mechanism used by some animals (panting), but less relevant for humans. Instead humans have a powerful ability to evaporate water from the skin as a consequence of sweating (Buono and Sjoholm, 1988; Nadel, 1978; Roberts et al., 1977; Saltin et al., 1972). This process depends on the vapour pressure gradient between the skin and environment. However, hyperthermia-induced fatigue develops when metabolic heat production exceeds the capacity for heat loss (Nybo, 2008; Robinson et al., 1945). The accumulated metabolic heat increases body tissue temperatures that elevate heart rate and minute ventilation (Adams et al., 1975; Buono and Sjoholm, 1988; Nelson et al., 1947) and inhibits motor output (Gonzalez-Alonso et al., 1999; Morrison et al., 2004; Walters et al., 2000). Thermoregulation almost always prevents such a situation.

2.6.1 Thermoregulation

Thermoregulation is defined as controlled heat production and dissipation to ensure thermal homeostasis is maintained or re-attained. The normal resting core temperature in humans is 37 ˚C (Gisolfi and Mora, 2000), regulated within a narrow range of ~3 ˚C despite varied ambient environments and metabolic heat generation. It is influenced by many non-thermal variables including blood pressure, glucose, hydration, age, disease, sex hormones, and competitive motivation (Crandall et al., 2010; Grucza et al., 1993). Such tight regulation is maintained by the hypothalamus (Cheung and Sleivert, 2004; Hammel et al., 1963; Nakayama et al., 1961) that contributes with the cerebral cortex to execute behavioural thermoregulation (e.g., altering exercise intensity, changing or removing clothes), and physiological regulation (e.g., sweating and skin blood flow; Nybo et al., 2011; Sawka et al., 1996). Behavioural regulation is effective

because it minimises physiological strain (especially cardiovascular, core temperature, metabolic) and ensures the exercise task is completed successfully, though at a lower performance intensity in ambient heat stress (Marino, 2004; Tatterson et al., 2000; Tucker et al., 2004). Physiological thermoregulation is influenced by fluctuations in core and skin temperatures (Johnson and Park, 1979). The core is more sensitive than the skin to small temperature alterations caused by autonomic and metabolic responses (Frank et al., 1999), but skin temperature has a larger thermal range to influence regulation (Charkoudian, 2010).

Laboratory studies investigating the thermoregulatory responses to exercising in a hot environment should be considered in the context of the simulated conditions and exercise task. Firstly, exercise tasks are typically constant-load cycling over a fixed duration, walking or running completed as self-paced time trials that reveal fatigue accumulation, or fixed-pace time to exhaustion trials that measure exhaustion (Schlader et al., 2011a). As mentioned above, exercise and competition performance power require variable effort to respond to changes in topography, competitors, climatic or course conditions; changing the physiological demands (Bernard et al., 2007; Etxebarria et al., 2014a; Junge et al., 2016; Mora-Rodriguez et al., 2008). Secondly, skin temperature and blood flow responses to exercise in temperate compared with hot environments for running and especially cycling generally do not simulate the appropriate airflow and thermoregulatory response (Brown and Banister, 1985; Junge et al., 2016). Body temperature, heat storage and ratings of perceived exertion (RPE) were significantly higher when airflow was 0 $km \cdot h^{-1}$ and 10 $km \cdot h^{-1}$ (similar to most laboratory protocols) compared with 33.3 $km \cdot h^{-1}$ and 50.1 $km \cdot h^{-1}$ (similar to real-world cycling speed for highly trained athletes) that facilitated more effective convective and evaporative heat offload (Saunders et al., 2005). Experimental protocols that have used insufficient airflow also appear to have overestimated the detrimental performance effect of dehydration (Convertino et al., 1996; Galloway and Maughan, 1997; Sawka et al., 1992). At real-world airflow velocities, high fluid intakes (80% of sweat losses) had no effect on thermoregulation (Saunders et al., 2005). Thus, findings from typical lab-based studies of thermoregulation during exercise in different environments should be

interpreted cautiously when the conclusions and implications do not match the exercise and environmental contexts.

2.6.1.1 Skin Temperature and Skin Blood Flow

Skin temperature (T_{Skin}) is influenced by the ambient temperature, skin blood flow (BF_{Skin}) and sweat evaporation (Brunt et al., 2016; Kenney and Johnson, 1992; Sawka and Wenger, 1988; Sawka et al., 2011a). Conversely, elevated skin temperature directly alters skin vascular tone (Johnson, 1986, 1986; Johnson and Proppe, 2010; Kenney and Johnson, 1992) and aids heat loss (Sawka et al., 2011a). Altered skin blood flow facilitates heat removal from the core to the environment (Charkoudian, 2010; Rowell, 1977a). The physiological temperature gradient (difference from core to skin) dissipates heat from the body core to the skin surface through conduction, convection, radiation, and (most effectively) evaporation via sweat (Rowell, 1986; Sawka et al., 2011a). The cooler blood is then transferred back to the body core, where it minimises core temperature elevation during exercise (Charkoudian, 2010).

When skin temperature (T_{Skin}) is < 30 ˚C skin blood flow is maximally vasoconstricted (Veicsteinas et al., 1982). As T_{Skin} exceeds 30 ˚C vasoconstriction wanes and vasodilation increases blood flow (Charkoudian, 2010; Wingo et al., 2009). High T_{Skin} may intensify perceived exertion (Maw et al., 1993; Pivarnik et al., 1988) and contribute substantially to negative feedback signalling that moderates exercise intensity (Jay and Kenny, 2009; Schlader et al., 2010b). In summary, a higher skin blood flow increases T_{Skin}, a lower skin blood flow equilibrates T_{Skin} with the ambient temperature, and sweat evaporation removes heat at an equal rate to the higher skin blood flow that warms the skin (Nybo et al., 2011). Practically, for individuals exercising in an environment < 35 ˚C, sweat evaporation will lower T_{Skin} below ambient temperature (Sawka et al., 1983a; Shapiro et al., 1980). Combined exercise and heat stress may limit skin blood flow, impeding the cardiovascular system and diminishing exercise intensity (Cotter et al., 2001). During intense exercise in the heat the increased skin and muscle blood flow combined with diminished central venous pressure

may compromise blood distribution, thermoregulation, and performance power (Crandall and González-Alonso, 2010).

2.6.1.2 Core Temperature

Core temperatures (T_C) are commonly measured at the oesophagus and rectum, but are not representative of uniform T_C throughout the body because temperatures vary due to different rates of heat production, perfusion and proximity to the surface (Sawka et al., 1996). As metabolic rate increases, T_C rises correspondingly (Nielsen and Nielsen, 1962; Saltin et al., 1972). Core temperature becomes excessive when metabolic rate exceeds thermoregulatory heat offload for a sufficient time, causing uncompensable heat stress and rapid performance decrement (Galloway and Maughan, 1997; Gonzalez-Alonso et al., 1999; Nielsen and Nielsen, 1962). Even in situations where T_C is elevated and thermoregulation is effective, performance may still be reduced (Ely et al., 2010). However, an individual's ability to attenuate T_C rising during exercise will experience cumulative physiological advantages including reduced respiratory and energetic demands combined with reduced thermal, mood and exertion perceptions that have direct performance benefits.

Controlled laboratory studies have found that the actual T_C reached during exercise is almost identical between exposures (Gonzalez-Alonso et al., 1999; Nielsen et al., 1993; Nybo and Nielsen, 2001a) when exercising at a fixed intensity, such that the only behavioural option is to stop. Gonzalez-Alonso et al. (1999) performed a series of studies for participants cycling at 60% $\dot{V}O_2$ max in 40 °C conditions, but starting at three different baseline oesophageal temperatures (T_{es}; 35.9, 37.4, 38.2 °C). Despite the three separate conditions the participants fatigued at the same level of hyperthermia (T_{es}: 40.1-40.2 °C) and cardiovascular strain (heart rate: 196-198 beats·min^{-1}, cardiac output: 19.9-20.8 L·min^{-1}). This generated the critical core temperature hypothesis. Further, despite different rates of heat storage participants reached volitional exhaustion at similar T_C (40.1-40.3 °C) and muscle temperature (40.7-40.9 °C). Conversely T_{Skin} – while substantially higher - was significantly different (38.4 ± 0.4 °C high vs. 35.6 ± 0.2 °C low heat storage). The authors concluded that high internal

body temperature causes fatigue in trained athletes during prolonged exercise in environmental conditions where heat dissipation capacity is limited; therefore, the time to reach exhaustion in hot environments was directly related to the rate of heat storage (Gonzalez-Alonso et al., 1999).

However, the critical core temperature hypothesis has been challenged by field and laboratory studies [*and discounted by Gonzalez-Alonso himself recently* (Nybo and González-Alonso, 2015)]. In-competition, field investigations have demonstrated T_C exceeding 40 ºC during endurance running (Lee et al., 2010; Pugh et al., 1967). Further, field studies have also demonstrated that participants with T_C exceeding 40 ºC while competing in races have maintained running speed *and* accelerated at the finish (Cheuvront et al., 2010; Ely et al., 2009, 2010; Kenefick et al., 2010; Lee et al., 2010; Tucker et al., 2006a). Laboratory studies that have terminated exercise for ethical reasons (T_C > 39.5 °C) were compromised because the participants were capable to continue exercise despite their elevated T_C (Cheung & McLellan, 1998; Périard et al., 2011). Laboratory studies that have had ethical clearance to passively (i.e., no exercise) heat participants to T_C > 39.5 ºC have indicated that the T_C upper limit is variable between individuals and dependent on the measurement site (Bynum et al., 1978; Pettigrew et al., 1974). Three features distinguish laboratory-based studies from field research: 1) laboratory studies produce near-maximal thermal strain *and* cardiovascular strain (>95% maximum heart rate) (Cheung and Sleivert, 2004); 2) laboratory studies frequently prescribe a fixed work rate either to exhaustion or as a time trial, which prevents the participant from altering their heat production rate and responses to the exercise and environmental stress, and; 3) laboratory studies have an ethical end-point for T_C (usually 39.5 or 40.0 ˚C) that they cannot exceed, forcing individuals to stop who have the capability to continue (Cheung and McLellan, 1998; Gonzalez-Alonso et al., 1999; Nielsen et al., 1990; Nybo and Nielsen, 2001a). Studies that have employed a self-selected intensity have found that performance was not limited by attaining a critical temperature, but by the rate of heat storage and anticipated exercise duration (Marino, 2004; Marino et al., 2004; Tucker et al., 2004, 2006b). The critical core temperature hypothesis and the anticipatory fatigue hypothesis may

be complementary thermoregulatory safety mechanisms, and should receive future research attention incorporating fixed and self-selected protocols (Cheung, 2007).

Exercising humans voluntarily stop at individually different T_C, as discussed above, but all mammals have a physiological T_C upper limit. A T_C > 43 ˚C will denature cells and unfold cytoskeletal and cellular proteins causing tissue necrosis (Craig and Schlesinger, 1985; Moseley, 1997). Heat shock proteins may protect cells when T_C < 43 ˚C, however, T_C > 43 ˚C will cause systemic inflammation, disseminated intravascular coagulation, cell necrosis, multiple organ failure, and ultimately death (Bouchama and Knochel, 2002; Bouchama et al., 1993; Hales and Sakurada, 1998; Moseley and Gisolfi, 1993). Cell damage and organ tissue damage from exposure to temperatures between 43-45 ˚C caused death for rat tissue *in vitro* (Burger and Fuhrman, 1964) and live rats *in vivo* (Frankel, 1959; Nadel et al., 1987).

2.6.1.3 Exercise-Induced Hyperthermia

Hyperthermia occurs when core temperature is above its normal set range and there is a sustained imbalance between heat load and the capability to dissipate heat (The Commission for Thermal Physiology of the International Union of Physiological Sciences (IUPS Thermal Commission), 1987). Exercise-induced hyperthermia has been observed during 60 min or longer sub-maximal exercise at 40-80% $\dot{V}O_2$ max (Gonzalez-Alonso et al., 1999; Kay et al., 2001; Nybo et al., 2001; Tucker et al., 2004, 2006b; Watson et al., 2005). During exercise it significantly impairs sustained or repeated-effort skeletal muscle power output by affecting the cardiovascular and central nervous systems (Drust et al., 2005), with a progressive reduction in $\dot{V}O_2$ max as ambient temperature increased (Arngrímsson et al., 2003).

2.6.1.4 Heat Stroke

Heat stroke has been described as a process activated by hyperthermia and facilitated by endotoxaemia (Bouchama and Knochel, 2002; Bouchama et al., 1993; Hanson and Zimmerman, 1979). Heat stroke symptoms include: T_C > 40

˚C, inflammation, CNS dysfunction, and multiple organ failure. This may include compromised cardiovascular function and hot, dry skin (Lim and Mackinnon, 2006). When T_C reaches 41 ˚C oxidative phosphorylation in mitochondria is inhibited in the liver and brain (Reinhardt et al., 1973). At T_C > 42 ˚C organ tissue and cells are severely degraded: enzymes denature, lipids and proteins become unstable, mitochondria are damaged and cell membranes liquefy (Evans and Bowler, 1973; Lim and Mackinnon, 2006). Once T_C > 43.5 ˚C heat stroke will occur regardless of endotoxaemia due to cell necrosis and protein degradation in tissue (Lim and Mackinnon, 2006). Approximately 25% of people will experience hypotension (Bouchama et al., 1991) with marked hypocapnia (P_aCO_2 < 20 mmHg; Knochel and Reed, 1994).

Heat stroke is usually observed in individuals who reach T_C 40-43 ˚C (DuBose et al., 2003; Hanson and Zimmerman, 1979; Shapiro and Seidman, 1990), but heat stroke has been identified as low as 38 ˚C (Richards and Richards, 1987). To complicate diagnosis further many healthy and coherent soldiers and endurance runners achieve T_C between 40.6-41.3 ˚C (Gilat et al., 1963; Maron et al., 1977; Pugh et al., 1967; Roberts, 1989), as do cancer patients treated with whole-body hyperthermia for 1 – 8 h (Bynum et al., 1978). These inconsistencies for heat stroke definition and pathology were addressed by Lim and MacKinnon (2006), who proposed an alternative hypothesis that described heat stroke as a dual pathway stimulated primarily by endotoxaemia and secondarily by hyperthermia. Further, they postulated that individuals who could tolerate greater inflammation could exercise to higher T_C before heat stroke occurred. In a competitive endurance setting influenced by a hot environment an individual's responses to a heat stressful environment may depend on their tolerance to inflammation and T_C elevation.

2.6.2 Cardiovascular Responses to Heat

Multiple studies have shown that heat stress substantially reduced $\dot{V}O_2$ max and physical work capacity primarily caused by the cardiovascular mechanisms that influence O_2 delivery (Gonzalez-Alonso and Calbet, 2003; Rowell et al., 1966). Core temperature during exercise is strongly influenced by cardiovascular function, especially during dynamic exercise above 80% $\dot{V}O_2$ max, when skin

blood flow and peripheral fatigue increase and aerobic energy metabolism is reduced in skeletal muscle (Gonzalez-Alonso and Calbet, 2003; Gonzalez-Alonso et al., 2008; Hargreaves and Febbraio, 1998; Nybo et al., 2001).

When T_{Skin} and by association skin blood flow increase to offload heat efficiently (Rowell, 1986; Sawka et al., 2011a) the blood flow redistribution decreases central blood volume, end-diastolic volume and stroke volume (SV; Rowell, 1986; Rowell et al., 1966). The cardiac output can be maintained by elevating heart rate during rest or moderate exercise (Nybo and Nielsen, 2001b; Rowell et al., 1969a), but a higher heart rate reduces diastolic filling time, end diastolic volume and SV (Stöhr et al., 2011). Heart rate cannot compensate for the reduced SV when exercise intensity is moderate to severe for a prolonged duration because diastolic filling becomes insufficient as it approaches maximum capacity (Gonzalez-Alonso and Calbet, 2003; Nadel et al., 1979; Rowell et al., 1966). Consequently cardiac output diminishes, O_2 delivery decreases, and performance deteriorates (Gonzalez-Alonso and Calbet, 2003; Nybo et al., 2001; Rowell et al., 1966). Heat stress reduces SV by a combination of related factors: increased heart rate (Chou et al., 2018) (Chou et al., 2018), dehydration, lower blood volume, and increased venous compliance and blood flow redistribution to skin and skeletal muscle (Nybo et al., 2011).

Both heart rate and cardiac contractility are enhanced by sympathetic nervous system activity during exercise, and by heat stress (Gonzalez-Alonso et al., 1999, 2008). Trained endurance athletes can maintain SV when exercising in temperate environments by enhanced cardiac filling pressure (Gledhill et al., 1994; González-Alonso et al., 2000). However, in hot environments a higher heart rate decreases diastolic filling time and a lower central blood volume reduces cardiac filling pressure (Fritzsche et al., 1999). Heart rate also increases linearly when T_{Skin} < 32 ˚C, but exponentially above 35 ˚C (Cheuvront et al., 2003). Further, heating skin from ~32 to 38 ˚C increases heart rate by 40-50 beats·min^{-1} (Rowell et al., 1969b).

Endurance trained or heat acclimated men performing prolonged exercise in a hot environment where fluid intake was restricted and airflow was low had

substantially lower $\dot{Q}$ when dehydrated compared with when they were euhydrated (Montain and Coyle, 1992; Montain et al., 1995). However, the findings of Montain et al. (1992, 1995) and a large number of other studies (e.g., González-Alonso et al., 1997; Kenefick and Sawka, 2007; Sawka et al., 1984) that have correctly linked dehydration to cardiovascular strain may not accurately transfer those same findings to endurance events. Firstly, the performance task (constant load compared with variable power/pace race simulation) and environmental conditions (insufficient airflow) in the laboratory did not match the real-world competition demands (Goulet, 2011). Secondly, the experimental design did not examine the effect of hydration alone, but also carbohydrate feeding status as part of the rehydration (Montain & Coyle 1992), which would have also reduced the neuroendocrine response. Well-trained endurance athletes have higher blood volumes (Convertino, 1991; Jones and Carter, 2000), which may offer some protection for lower cardiac output (Gonzalez-Alonso, 1998; González-Alonso et al., 1998). However, trained cyclist's performance power declined at the same rate as cardiovascular variables (SV, cardiac output, mean arterial blood pressure) in hot compared with temperate environments with low (10 $km.h^{-1}$) airflow (Périard et al., 2011b), demonstrating that cardiovascular drift was exacerbated by heat stress. Furthermore, cardiovascular drift may also be influenced by the increased skin blood flow (Hartley, 1977). Cardiovascular, T_C, and skin blood flow changes are inter-related and change in response to exercise and heat stress.

2.6.3 Respiratory Responses to Heat

2.6.3.1 Hyperventilation

Exercise stimulates the CNS to increase minute ventilation to match intensity, ensuring adequate O_2 delivery to contracting muscles (Astrand and Rodahl, 1977; Wasserman et al., 1973). So when individuals exercise at high intensity hyperpnoea is a normal response to supply the required oxygen. Ventilation responses are driven by feedforward (e.g., exercise) as well as feedback mechanisms (e.g., airway, lungs, systemic, humoral, muscle neural sources). If an individual produces metabolic heat at a rate that causes hyperthermia while exercising, minute ventilation increases at a much greater rate (Cabanac and

White, 1995) due to the combination of two stressors (i.e., hyperthermia-induced hyperventilation). High ventilation rates cause CO_2 washout, decreased $PaCO_2$, and decreased hydrogen ion concentration ($[H^+]$; (Martin et al., 1979; Nybo and Nielsen, 2001b; White, 2006). Hyperthermia-induced hyperventilation is highly variable, depending on each individual's adaptability to thermoregulation and training (Dempsey et al., 1975; Hayashi et al., 2006, 2009; Sawka et al., 1980). The mechanisms that drive hyperthermia-induced hyperventilation remain unclear. Possible theories have identified one or more of the following: greater CO_2 sensitivity at the chemoreceptors; increased muscle temperature that alters muscle metaboreflexes; a more sensitive positive feed-forward response to exercise intensity, and greater thermal sensitivity of the respiratory system (Asmussen et al., 1965; Martin et al., 1979; Nybo and Nielsen, 2001b, 2001c).

2.6.3.2 Respiratory Fatigue

During submaximal exercise cardiac output is high enough to maintain blood flow for both contracting locomotive skeletal muscle (Nielsen et al., 1990), and though unproven, ventilatory skeletal muscle. High or severely intense exercise increases respiratory work and eventually respiratory muscle fatigue from sustained high minute ventilation rates (> 150 $L \cdot min^{-1}$; (Harms et al., 1998b). Furthermore, exercising at $\dot{V}O_2$ max requires up to 14-16% $\dot{Q}$ and 20-24% of $\dot{V}O_2$ to maintain rapid breathing frequency and deep tidal volumes (Harms et al., 1998b). High respiratory work rates activate the sympathetic nervous system to reduce skeletal muscle blood flow to ensure the respiratory system receives sufficient blood supply, which exacerbates whole-body fatigue and sympathetic nervous system activation via the pressor reflex (Harms et al., 1998b; Romer and Polkey, 2008). Ventilation frequency also influences perception because hyperventilation is associated with greater effort and discomfort (Romer and Polkey, 2008).

2.6.3.3 CO_2 and $[H^+]$

Ventilation is sensitive to small increases in $PaCO_2$ but relatively insensitive to the partial pressure of oxygen (PaO_2) fluctuations detected at the chemoreceptors during respiration (West, 1977). The hyperventilation that

causes CO_2 washout, reduces $PaCO_2$, and decreases $[H^+]$ also mediates vasoconstriction of the cerebrovasculature (Equation 2.1). This vasoconstriction diminishes brain stem blood flow (hypocapnic hyperventilation) and impedes brain O_2 delivery, increasing metabolite accumulation and central fatigue (Raichle and Plum, 1972; Secher et al., 2008). However, similar changes in $[H^+]$ and CO_2 do not hinder skeletal muscle function or peripheral responses (Fitts, 2010).

Equation 2.1: Bicarbonate $H_2O + CO_2 \longleftrightarrow H_2CO_3 \longleftrightarrow H^+ + HCO_3^-$

2.6.4 Cerebrovascular Responses to Heat

2.6.4.1 Central Fatigue

Exercise-induced hyperthermia may be the primary stimulus that limits central motor drive and therefore exercise performance. Nielsen and co-workers demonstrated that core temperature and heart rate responses were very high in hot environments despite similar leg muscle blood flow, [La], and glycogen levels in both cool and hot environments (Nielsen et al., 1990, 1993). In another study from this group, cycling for 50-60 min in a hot (40 ˚C) environment caused volitional exhaustion and reduced maximal voluntary contraction immediately afterwards, however, electrical stimulation evoked the same contraction force as the control condition, demonstrating that hyperthermia caused central fatigue and decreased motor output to skeletal muscles (Nybo and Nielsen, 2001a). The same outcomes were observed for maximal voluntary hand-grip contractions after cycling to volitional exhaustion, showing that a muscle group that experienced no peripheral fatigue was impeded by central fatigue (Nybo and Nielsen, 2001a). A similar study design was used to show that the CNS had an anticipatory mechanism that limited cycling power and contraction force when electrically stimulated in hot (35 ˚C) compared with cool (15 ˚C) environments; the researchers pointed out that crucially the anticipation began before T_C was significantly different (Tucker et al., 2004). The anticipation response may depend on the rate of heat storage to maintain homeostasis rather than when

physiological systems reach their upper limits or approach catastrophe (Tucker et al., 2006b). The evidence that the CNS uses heat storage as a feed forward mechanism to regulate effort has been questioned and remains contentious (Jay, 2009; Jay and Kenny, 2009; Marino, 2009). Central fatigue and thermoregulation may also be influenced by changes in serotonin, noradrenaline and dopamine concentrations or ratios (Feldberg, 1969), but human studies that have investigated the effects of oral branched chain amino acid and quercetin consumption on performance in a hot environment were ineffective in three out of four studies (Cheuvront et al., 2004, 2009; Mittleman et al., 1998; Watson et al., 2004). Central nervous system activation was reduced by endurance exercise that caused hypoglycaemia in humans (Nybo, 2003) and brain hyperthermia in goats (Caputa et al., 1986). While the mechanisms that cause central fatigue are still debated, the fact that central fatigue is caused by a high T_C has been found consistently in these and other studies (e.g., Morrison et al., 2004; Thomas et al., 2006; Todd et al., 2005), including more direct data from animal studies (Walters et al., 2000). During competitive exercise in a hot environment brain temperature and brain metabolism are elevated by T_C increasing brain glucose consumption (Rasmussen et al., 2010b; Yablonskiy et al., 2000).

2.6.4.2 Brain Blood Flow

Brain blood flow has been measured using different techniques and anatomical locations. Transcranial Doppler (TCD) ultrasound measures brain blood flow at the middle cerebral artery (Jorgensen, 1994; Willie et al., 2011). The Kety-Schmidt technique measures it at the internal jugular vein (Ide and Secher, 2000); whereas positron emission tomography (PET; Paus et al., 1998) and magnetic resonance imaging (MRI; Fontes et al., 2013) can measure the whole brain or region specific tissue perfusion. Of these approaches, TCD and the Kety-Schmidt technique have been most commonly used to determine changes in brain blood flow during exercise in the heat. Studies applying these brain blood flow/velocity measurement methodologies have shown that brain blood flow decreases for hyperthermic individuals exercising in a hot environment, whereas blood flow remains constant when performing the same exercise intensity in a

temperate environment (Nybo and Nielsen, 2001b; Nybo et al., 2002a; Périard and Racinais, 2015). Further, Nybo and Nielsen (2001b) showed that sub-maximal cycling (~57% $\dot{V}O_2$ max for 50 mins) combined with uncompensable heat stress (40 ˚C), compared with a temperate environment (18˚C), provoked hyperthermia (40.0 vs. 37.8 ˚C), hyperventilation (83 vs. ~60 $L \cdot min^{-1}$) and hypocapnia (36.0 vs. 40.9 mm Hg), resulting in a markedly lower brain blood flow (-3.8 vs +13.6 $cm \cdot s^{-1}$) for endurance-trained men performing in the heat.

Exercise-induced hyperthermia reduces brain blood flow by three mechanisms that usually occur in unison for an individual competing in a hot environment. Firstly, hyperventilatory hypocapnia has the strongest effect (Nelson et al., 2011) by increasing vascular tone in the cerebral arterioles (Ide and Secher, 2000; Kety and Schmidt, 1948; Lassen, 1959; Rasmussen et al., 2005, 2007). Secondly, brain blood flow may be decreased because cardiac output is also required to supply contracting skeletal and respiratory muscle to maintain exercise intensity and skin blood flow to offload heat (Ide et al., 1998; Secher et al., 2008). Thirdly, blood flow redistribution caused by exercise (skeletal and respiratory muscle) and thermoregulatory needs (increased flow to skin) lowers total peripheral resistance and mean arterial blood pressure to pressures that challenge cerebral autoregulation (Ide and Secher, 2000; Paulson et al., 1990). Cerebral autoregulation is defined as the myogenic process that alters vascular tone to respond to changes in intrapleural pressure that maintain relatively constant brain blood flow when mean arterial blood pressure changes (Ide et al., 1998; Paulson et al., 1990). The reduced blood pressure and/or brain blood flow may be interpreted by the CNS as anticipating circulatory failure, causing early fatigue and reducing motor output (Thompson, 2006) that degrades performance power. Exercise and heat stress exacerbated hyperventilation, hypocapnia, and hyperthermia to reduce brain blood flow by 26% (Périard and Racinais, 2015) and 18% (Nybo et al., 2002a).

2.6.4.3 Brain Oxygenation

Reduced brain blood flow may restrict O_2 delivery, facilitating low mitochondrial PO_2 and possibly central fatigue (Nybo and Rasmussen, 2007; Rasmussen et

al., 2007). Mitochondrial PO_2 increases during moderate intensity exercise but decreases during strenuous exercise because hyperventilation-induced hypocapnia causes a reflex decrease in brain blood flow, and the higher exercise intensity elevates brain metabolism and O_2 consumption (Rasmussen et al., 2010a). These factors combine to lower brain oxygenation and reduce maximal exercise capacity (Rasmussen et al., 2010a; Seifert et al., 2009a). In support of lower brain oxygenation limiting exercise performance, reduced handgrip strength has been correlated with lower frontal lobe oxygenation (Rasmussen et al., 2007), inferring that motor performance may be influenced by (prefrontal) cerebral oxygenation. To compensate for reduced brain blood flow, brain oxygen extraction rate increases modestly (7%; Nybo et al., 2002b) or substantially (47%; Nybo et al., 2002a). The cardiovascular and respiratory responses to exercise-induced hyperthermia and hyperventilatory hypocapnia collectively and substantially influences blood flow to the brain (and thus brain oxygenation) and likely worsens central fatigue and exercise performance. However, hypoxic environments are immediately more detrimental to performance power output (21%), whereas hot environments become progressively detrimental over time (12%) when compared with cool normoxic environments (Périard and Racinais, 2016). Furthermore, central fatigue hindered motor output function via diminished brain oxygenation (brain mitochondrial PO_2) and brain blood flow during maximal exercise that induced hyperthermia, hyperventilation, and hypocapnia (Rasmussen et al., 2010b).

2.6.4.4 Brain Temperature

The brain's blood flow, the body core temperature, and metabolic heat production within the brain itself are the primary factors that influence brain temperature (T_{Brain}; Nybo and Secher, 2011a; Nybo et al., 2002b, 2011). The reduced brain blood flow during strenuous exercise in hot environments from hyperthermia combined with hyperventilatory hypocapnia restricts heat removal via lower venous blood flow and therefore causes greater heat storage in the brain (Nielsen and Nybo, 2003). At present human T_{Brain} cannot be measured directly, but may be inferred from blood temperature of the internal jugular vein (Nybo et al., 2002b), which indicates T_{Brain} is 0.2-0.3 ˚C higher than T_C (Yablonskiy et al.,

2000). Experiments with exercising goats also imply that high T_{Brain} may be detrimental to central fatigue and motor output for exercising humans (Caputa et al., 1986). Crucially, central fatigue may be a negative feedback mechanism that limits motor output to prevent prolonged and excessive T_C and T_{Brain} (Nielsen and Nybo, 2003) that may lead to heat stroke (Stefanini and Spicer, 1971). No specific brain cooling mechanism has been found in humans that mimics the effective panting response in several species of heat-dwelling animals (Caputa et al., 1991; Elkhawad, 1992; McConaghy et al., 1995). A selective brain cooling response via hyperpnoea has been hypothesised for humans to facilitate heat loss from the upper airways and reduce brain temperature. However, the theory was based on measuring tympanic temperature (membrane over the inner ear canal) as a surrogate to brain temperature because it receives the same blood supply from the carotico-tympanic artery and external carotid artery (Cabanac and Caputa, 1979; White and Cabanac, 1996). The hypothesis remains contentious (Crandall et al., 2011; Nybo and Secher, 2011a, 2011b, 2011c; White et al., 2011a, 2011b, 2011c).

2.6.5 Peripheral Responses to Heat

Skeletal muscle temperature increases substantially during exercise (González-Alonso et al., 1999; Jay et al., 2007) enhancing nerve conduction, action potential transmission, calcium ion release from the sarcoplasmic reticulum, and cross-bridge cycling. The rate of force production and rate of relaxation between contractions improves due to the temperature quotient (Q_{10}) effect; a 10 ˚C muscle temperature increase doubles biochemical processes, metabolic reactivity, skeletal muscle contraction speed and power (Nybo, 2011). Examples of the Q_{10} effect have been observed during superior single sprint performances (Asmussen and Bøje, 1945; Ball et al., 1999; Duffield et al., 2009; Faulkner et al., 2013). Muscle function and $\dot{V}O_2$ are not affected at temperatures up to 41 ˚C (Gonzalez-Alonso et al., 1999), nor does local muscle temperature influence central fatigue (Thomas et al., 2006). The higher skeletal muscle temperatures increase metabolism, utilize more glycogen, produce more [La] (Febbraio et al., 1994a, 1996; Schmulewitsch, 1868), and consume more O_2 (Dimri et al., 1980). Skeletal muscle metabolism may be elevated even further by heat stress when

dehydration or blood flow redistribution reduce muscle blood flow (González-Alonso et al., 1998). Despite greater metabolite accumulation and reduced blood flow from hyperthermia during exercise, skeletal muscle has not been associated with volitional exhaustion or disturbed homeostasis: a conclusion that may require further examination (Amann, 2008). Instead the CNS limits motor output for a maximum voluntary contraction, but when electrically stimulated the same muscle group can produce a greater contraction, indicating that the CNS and not peripheral muscle is the limiting system (Nybo and Nielsen, 2001a). Peripheral feedback from skeletal muscle is essential during prolonged endurance exercise to regulate blood flow and respiration that maintain consistent O_2 delivery (Amann, 2011). Afferent peripheral feedback is also important to fine tune the pacing strategy and central fatigue that may alter motor output (Amann, 2012; Amann et al., 2011). In theory, skeletal muscle hyperthermia may have input into central fatigue, but this remains unproven (Nybo et al., 2011).

2.6.6 Psycho-Physical Responses to Heat

Exceptional physical performances have been observed for individuals powerfully motivated to succeed and who have refined their athletic skill through years of training. Such individuals are able to over-ride their normal volitional exhaustion responses to fatigue and maintain central motor output for a brief duration (Amann and Dempsey, 2008; Hargreaves, 2008). However, the metabolic and thermal consequences from maintaining exercise intensity for more than a brief period cause exertional heat exhaustion, especially in hot environments (Schlader et al., 2011a). Exertional heat exhaustion that mimics the performance stress from competitive races in hot environments is very difficult to replicate in the laboratory because the individual must be motivated to push themselves beyond the normal boundaries of volitional exhaustion. Studies that investigate psychological and physiological responses to heat must account for the participant's motivation that may influence an individual's tolerance to volitional exhaustion.

Thermal perceptions contribute to one's motivation to continue exercising in a hot environment (Cotter et al., 2001; Schlader et al., 2011b). Thermal discomfort

is strongly associated with high T_{Skin} (Gagge and Gonzalez, 1973; Gonzalez et al., 1973; Hardy, 1961). The T_{Skin}, thermal discomfort, and thermal sensation at the start of self-paced exercise substantially influenced exercise intensity at the beginning, and critically (from a performance perspective) for the entire exercise task (Schlader et al., 2011c). Interventions that produce a perceived cool sensation in a hot environment have had positive physiological and performance effects without any influence on T_C (Stevens et al., 2016). Further, strong feelings of thermal discomfort may influence CNS pathways and central fatigue (Meeusen and Roelands, 2010; Schlader et al., 2011c). It has been established that the relationship between T_{Skin}, thermal perceptions and motivation provide important feedback that influences exercise performance in hot environments. The central feedback mechanisms themselves remain debated, including: anticipatory pacing (Tucker, 2009; Tucker et al., 2004, 2006b), a central governor model (Noakes et al., 2001) and the brain integrating each input on its merits (Hargreaves, 2008).

2.6.7 Heat and the Menstrual Cycle

For female athletes the menstrual cycle degrades endurance exercise performance when it is supressed by heavy training and cyclically during the mid-luteal phase for short-duration, high-intensity and endurance exercise (Julian et al., 2017; Lei et al., 2018; Shaklina et al., 2016), although any such effect has also been shown to be negligible, e.g., less than is caused by humidity (Lei et al., 2017). Endurance exercise in hot environments is complicated further for female athletes because resting T_C typically rises by as much as 0.6 ˚C during exercise in the mid-luteal phase due to elevated progesterone and oestrogen (Hessemer and Bruck, 1985; Kolka and Stephenson; Pivarnik et al., 1992). Other physiological responses including heart rate, oxygen consumption, sweat rate, minute ventilation and forearm blood flow may not be affected by menstrual phase (Hirata et al., 1985; Horvath and Drinkwater, 1982; Wells and Horvath, 1974). However, several studies have demonstrated that thermoregulatory and cardiovascular strain may be significantly higher in the mid-luteal phase (Hessemer and Bruck, 1985; Janse et al., 2012; Pivarnik et al., 1992). Furthermore, exercise performance and exercise tolerance were reduced at the

mid-luteal (compared with follicular) phase, leading the researchers to recommend that women adjust their menstrual cycle to ensure they competed during the mid-follicular phase in endurance events with uncompensable heat stress (Janse et al., 2012; Tenaglia et al., 1999). The effect of the oral contraceptive pill (OCP) on cardiovascular strain, thermoregulation and performance has shown that the OCP reduces the different sweat response between active and sugar pill phases (Grucza et al., 1993). Pragmatically, female athletes may require more time to adapt to heat acclimation and acclimatisation than male athletes, and should aim for a greater number of days in their preparation to ensure thermoregulatory and cardiovascular adaptation has occurred (Mee et al., 2015).

2.7 Heat Acclimation

Human physiology adapts exceptionally well to heat stress. Adaptation to chronic hyperthermia was described by Lind (1768) and studied extensively in the 20th century for military (Bean and Eichna, 1943; Hellon et al., 1956; Pandolf et al., 1977) and mining purposes (Dresoti, 1935; Horvath and Shelley, 1946; Strydom et al., 1966; Weiner, 1950; Wyndham, 1967; Wyndham and Jacobs, 1957). Heat acclimation/acclimatisation as an ergogenic aid for athletes received scarce research attention until a recent flurry of studies identified positive performance benefits in hot (Buchheit et al., 2011; Garrett et al., 2009; Lorenzo et al., 2010; Neal et al., 2016) and temperate environments (Hue et al., 2007; Lorenzo et al., 2010; Scoon et al., 2007). Conversely, several well-constructed experiments have failed to replicate similar positive performance and physiological outcomes in a temperate environment (Karlsen et al., 2015a; Keiser et al., 2015a; Neal et al., 2016). Heat acclimation has been achieved by exposure to hot-dry or hot-humid conditions for 7-12 consecutive days in a simulated and controlled indoor environment, with most adaptations achieved to a large extent within 4-6 days (Garrett et al., 2012a, 2014; Nielsen, 1994; Pandolf, 1998). The number of consecutive heat exposures has been defined as short (< 7 days), medium (8-14 days) and long-term heat acclimation (>15 days; Garrett et al., 2011). Heat acclimation was induced by exercise at a constant workload (Lorenzo et al.,

2010; Nadel et al., 1974; Nielsen et al., 1993, 1997; Robinson et al., 1943; Sawka et al., 1985; White et al., 2016) or controlled hyperthermia (Fox et al., 1963, 1967; Garrett et al., 2009; Henane and Valatx, 1973; Neal et al., 2016; Patterson et al., 2004a). Heat acclimation adaptations respond most favourably to low intensity (< 70% $\dot{V}O_2$ max) and durations longer than 30 minutes, preferably 90 minutes (Kelly et al., 2016; Tyler et al., 2016; Wingfield et al., 2016). Passive heat exposures have also been used to effectively acclimate individuals (Beaudin et al., 2009; Brazaitis and Skurvydas, 2010). Heat exposures should be on consecutive days to induce acclimation (Gill and Sleivert, 2001), though individuals have acclimated successfully with regular breaks up to 72 hours but no more than 7 days (Barnett and Maughan, 1993). Heat acclimation adaptations can be maintained for longer durations by exercise and physical fitness, and by protocols that require higher frequency and more consistent heat exposures (Pandolf et al., 1977). Physiological adaptations that require the least time to induce (e.g., heart rate) decay the fastest compared with thermoregulation and cellular remodelling (Bennett, 1997; Pandolf et al., 1977; Williams et al., 1967).

Heat acclimatisation requires similar exposures from the natural (though uncontrolled) environment, and elicits the same adaptations (Wenger, 1998). Individuals best adapted to heat stress are long-term residents of a hot climate (Edholm et al., 1963). Acclimatisation confers all the performance (Buchheit et al., 2011; Hue et al., 2007) and physiological (Nielsen et al., 1997; Périard et al., 2016; Sawka et al., 1985) benefits of heat acclimation. Furthermore, acclimatisation may be superior to laboratory-based acclimation when airflow velocity does not replicate convective heat loss (Brown and Banister, 1985; Cheuvront et al., 2004; Saunders et al., 2005) and radiant heat load from the sun and road surface do not replicate radiant heat stress (Junge et al., 2016; Taylor and Cotter, 2006).

2.7.1 Heat Acclimation Adaptations

Heat acclimation may be ergogenic by enhancing metabolic efficiency, plasma volume, evaporative cooling, thermal endurance, glycogen sparing, cardiac efficiency combined with reduced heart rate and Tc (Horowitz, 2002; Knochel,

1990; Levi et al., 1993; Levy et al., 1997; Périard et al., 2015; Sawka et al., 1996; Senay et al., 1976). The strength of heat acclimation adaptations is determined by the intensity (ambient temperature and water vapour pressure), duration, frequency, and total number of exposures (Junge et al., 2016; Sawka et al., 1996; Taylor and Cotter, 2006). Adaptations may also be strongest at the same time of day that heat acclimation has occurred, especially central thermoregulatory control effects (Shido et al., 1999).

2.7.1.1 Increased Sweat Response

The sweating response adapts at the central and peripheral level. Centrally, the threshold that stimulates the sweating response begins at a lower T_C (Nadel et al., 1974; Roberts et al., 1977). Highly trained endurance athletes may also have a greater T_C range that allows them to begin exercise at a lower T_C and reach exhaustion at a higher T_C (Cheung and McLellan, 1998; Gonzalez-Alonso et al., 1999; Mora-Rodriguez et al., 2010; Pugh et al., 1967; Selkirk and McLellan, 2001). Peripherally, the renin-angiotensin-aldosterone axis conserves salt in the kidney and the sweat glands change the sweat rate, concentration and sensitivity to produce a higher volume of more dilute sweat for evaporation from the skin (Adams et al., 1975; Buono and Sjoholm, 1988; Gonzalez et al., 1974; Nielsen et al., 1997). Sweat rate is augmented via greater cholinergic sensitivity, eccrine gland size and sweat glands that can produce higher sweat rates combined with lower sweat hidromeiosis (Fox et al., 1963; Ogawa et al., 1982; Sato and Sato, 1983; Sato et al., 1990). Thus, sweat composition becomes more dilute via increased electrolyte reabsorption and lower sodium concentration (Allan and Wilson, 1971; Dill et al., 1938; Ogawa et al., 1982) via sodium reabsorbed from the ascending tubule (Allan and Wilson, 1971; Ogawa et al., 1982; Sato and Dobson, 1970). The superior sweat rate and volume conferred by heat acclimation reduces T_{Skin} and skin blood flow, distributing more blood volume to the central circulation (Eichna et al., 1950; Rowell et al., 1967). It is important to note that airflow during heat acclimation should be removed to maximally stimulate T_C elevation and the sweating response (Junge et al., 2016).

2.7.1.2 Enhanced Cardiovascular Adaptations

Plasma volume (PV) expansion increases cardiac filling pressure and end diastolic volume to improve cardiovascular stability in hot environments (Senay et al., 1976), and reduces blood temperature to lower skin blood flow and maintain a higher central blood volume (Sawka et al., 2011a). These adaptations convey substantial performance benefits (Berger et al., 2006; Luetkemeier and Thomas, 1994; Scoon et al., 2007). However, PV expansion may cause haemodilution that compromises O_2 delivery, and provides no beneficial blood volume enhancement for highly trained endurance athletes (Coyle et al., 1990; Hopper et al., 1988; Warburton et al., 1999). Research has demonstrated that substantial (4-15%) PV expansion can be driven by 3-4 consecutive days of heat exposure (individual ranges were 3-27%; (Nielsen et al., 1993; Patterson et al., 2004a; Senay et al., 1976). Plasma volume expansion is dependent on hydration, T_{Skin}, the number of exposures, whether the acclimation protocol required resting or exercising (Harrison, 1985; Sawka et al., 1983b), aerobic fitness (Convertino, 1991; Fellmann, 2012; Sawka et al., 2000), and exercise mode (Convertino, 1991; Harrison, 1985). Once PV expansion has occurred it may be retained for at least 7 days (Garrett et al., 2009) and up to 22 days (Patterson et al., 2004a).

When an individual competes in a dehydrated state the positive adaptations they have from aerobic endurance fitness and heat acclimation may be neutralised (Buskirk et al., 1958; Sawka et al., 1983b) because dehydration is detrimental to thermoregulation and cardiovascular responses (Morimoto, 1990; Sawka and Coyle, 1999). In training, however, dehydration should conceivably be stimulated as a stressor to aid heat acclimation during exercise because it may amplify cardiovascular adaptation, PV expansion, fluid retention, and electrolyte retention (Akerman et al., 2016; Garrett et al., 2011; Périard et al., 2015; Taylor and Cotter, 2006).

The most plastic and beneficial adaptations to heat acclimation for humans are enhanced blood volume and cardiovascular capacity (Convertino, 1991; Gillen et al., 1991; Heinicke et al., 2001; Lorenzo et al., 2010; Périard et al., 2015;

Sawka et al., 1996; Taylor, 2011). Heart rate is usually higher during the first heat exposure and decreases with each subsequent bout (Nielsen et al., 1993, 1997; Rowell et al., 1967; Wyndham et al., 1968). The reduction in heart rate across days may be explained by CNS adaptation that decreases sympathetic nervous system activity (Berlyne et al., 1974) and enhanced blood volume (Convertino, 1991; Sawka et al., 2000). Research has reported that heat acclimation has variable effects on stroke volume; either increasing it (Nielsen et al., 1993; Rowell et al., 1967; Wyndham et al., 1968) or not (Nielsen et al., 1997; Wyndham, 1951). Nielsen et al. (1993) demonstrated that heat acclimation (9-12 days, 40 ˚C, 10% RH) significantly increased cardiac output but a similar protocol (Nielsen et al., 1997; 8-13 days, 35 ˚C, 87% RH) made no significant change to cardiac output. One potential explanation for these differential effects may be that the higher ambient water vapour pressure of the second study may reduce the gradient between the environment and the skin, requiring substantial sweat response and skin blood flow adaptations that obscure measurable cardiac output adaptations. At present the differences in cardiovascular adaptations between dry and humid heat remain unanswered.

2.7.1.3 Superior Metabolic Efficiency

Metabolic efficiency has been shown to improve at any given work load after heat acclimation. Specifically, oxygen consumption (Beaudin et al., 2009; Keiser et al., 2015a; Sawka et al., 1983a), muscle glycogen utilization (King et al., 1985; Kirwan et al., 1987), blood [La] and muscle [La] (Febbraio et al., 1994a; Neal et al., 2016) were lower at submaximal intensities, and power output at lactate threshold was 5% higher in hot *and* cool environments (Lorenzo et al., 2010). However, the glycogen sparing benefits may be slightly beneficial and effective only in cool environments (Young et al., 1985). Further, study design and participant familiarity may influence physiological responses that indicate changes to metabolic efficiency.

2.7.1.4 Thermotolerance

Thermotolerance and heat acclimation may be mutually beneficial physiological mechanisms because both are controlled by the heat shock response to improve

an organism's ability to successfully overcome adverse (and potentially lethal) heat strain (Kuennen et al., 2011). Thermotolerance is stimulated by non-lethal heat exposure(s) that trigger heat shock protein (HSP) synthesis, cellular adaptations, and upregulated genetic expression that ensure an organism can survive an otherwise lethal heat stress (Guy et al., 2016; Horowitz, 1998; Kodesh et al., 2011; Moseley, 1997). The HSPs respond rapidly to stress, especially heat and exercise (Locke, 1997), over a period of hours to days by binding to newly formed proteins to provide protection or to denatured proteins to initiate repair after exposure to heat, hypoxia, energy deficit, fever or ischaemia (Feder and Hofmann, 1999; Horowitz, 2002; Kregel, 2002). Furthermore, heat acclimation has no negative influence on immune system function (Guy et al., 2016). Cardiovascular and plasma volume expansion adaptations to heat acclimation take less than five days, while cellular adaptation (including HSP's) may require up to 15 days of repeated exposures (Sandström et al., 2008).

2.7.1.5 Improved Thermal Comfort

Thermal comfort is influenced by T_C and T_{Skin} (Cabanac et al., 1972; Flouris and Cheung, 2009), especially T_{Skin} in response to heat stress that drives behaviour change to prevent increased T_C (Schlader et al., 2009). Participants usually feel greater discomfort when exercising in the heat compared with temperate and cool environments (Maw et al., 1993), but heat acclimation has reduced thermal strain for trained and untrained individuals during treadmill walking in an environment chamber (40 ˚C, 30% RH) for ~120 and ~150 min (Aoyagi et al., 1998). When individuals are thermally comfortable they exercise for longer and at higher work rates (Schlader et al., 2009), so heat acclimation may facilitate superior performance by reducing psychophysical and thermal strain (Cotter et al., 2001).

2.7.2 Dry and Humid Heat Acclimation

The humidity during heat acclimation may stimulate different physiological mechanisms and produce different relative responses. Early research indicated dry heat acclimation was superior (Bean and Eichna, 1943; Henshel et al., 1943; Robinson et al., 1943) because hot-humid environments had higher ambient and

skin water vapour pressure caused by greater sweat rates, which reduced the gradient between the skin-to-environment. In humid environments the skin-to-environment gradient can be improved by a higher T_{Skin} (caused by greater skin blood flow) to transfer heat to the skin and greater water vapour pressure (greater sweat coverage on skin). Both factors require greater skin blood flow with negligible circulatory strain. Sweat glands become more efficient at higher sweat rates by improving evaporation from the skin (Fox et al., 1964). An additional theory that humid heat acclimation redistributes more sweat to the limbs to increase evaporative heat offload (Shvartz et al., 1979) remains inconclusive, and may be better explained by variable sweat rates in different body regions (Patterson et al., 2004b) and blood flow redistribution to increase skin perfusion in the limbs (Chiesa et al., 2016). The induction, performance benefits, and decay of dry compared with humid heat acclimation remain unresolved in many areas. Athletes are advised to prepare for competition by simulating the environmental conditions and training at self-paced intensity to improve heat dissipation capability. Such preparation allows athletes to produce greater exercise intensity because individuals can endure higher metabolic heat production (Keiser et al., 2015a; Lorenzo et al., 2010; Racinais et al., 2015).

2.7.3 Short- and Long-Term Heat Acclimation

Repeated, acute heat exposures trigger short-term heat acclimation (STHA) adaptations (Figure 2.3) that significantly enhance cardiovascular adaptations (Piwonka and Robinson, 1967) but not the sweating response (Cotter et al., 1997; Patterson et al., 2004b) within five days. The physiological drive to establish the STHA adaptations may compromise homeostasis because the process requires increased ATP utilisation and therefore reduced metabolic efficiency to diminish the noradrenaline inotropic response, increase β-adrenergic activation, and increase phosphorylation (Horowitz, 1998, 2002). Repeated, chronic exposures from months or years working or residing in a hot environment produce a long-term heat acclimation (LTHA): an optimal adaptive state to heat that increases the efficiency of signalling pathways and metabolic processes. This allows an organism to maintain homeostasis during acclimation to a hot environment through a continuous process of re-programmed gene

expression (Bennett, 1997). Additional cell protection is provided by HSP 72. The LTHA process achieves the same outcomes as STHA (increased Ca^{2+} signalling and increased cardiac contractility), but with reduced ATP utilisation via an increased phospholamban (calcium regulatory protein) concentration, increased noradrenaline inotropic response, decreased β-adrenergic activation, and relative decrease in phosphorylation (Horowitz, 2002).

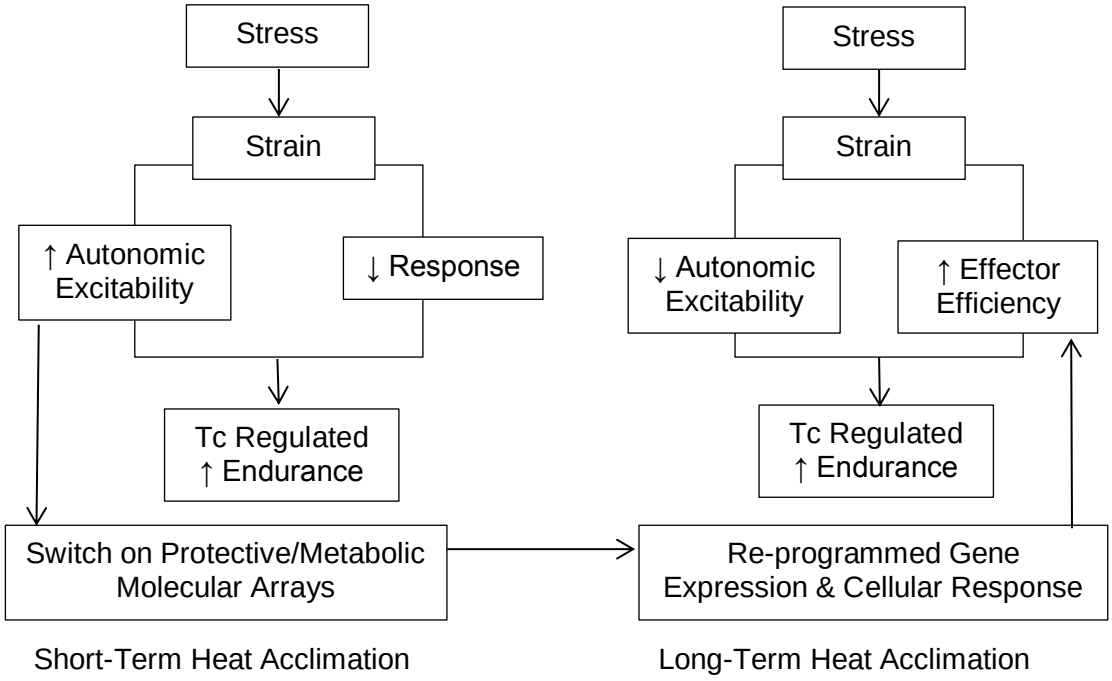

Figure 2.3: Physiological adaptation response to heat stress over an acute (short-term heat acclimation) or chronic (long-term heat acclimation) exposure to a hot environment (Horowitz, 2002). Reproduced with permission.

2.7.4 Heat Acclimation as a Performance Enhancer

Humans have used heat acclimation and acclimatisation to improve military, occupational, and sporting performance. Endurance training facilitates superior aerobic fitness, heat acclimation adaptations, and protection from heat injury compared with untrained individuals (Armstrong and Pandolf, 1988; Gardner et al., 1996; Taylor and Cotter, 2006), but cannot comprehensively replicate heat acclimation / acclimatisation because the central (elevated T_C) and peripheral (volume and composition) sweat rate responses must be powerfully stimulated (Armstrong and Pandolf, 1988; Henane et al., 1977). Heat acclimation has improved athletic performance for moderately trained athletes and untrained

soldiers performing ramp protocols to volitional exhaustion or $\dot{V}O_2$ max (Garrett et al., 2009; Sawka et al., 1985). Garrett et al. (2009) demonstrated that five days of heat acclimation increased time to exhaustion by 106 s for a continuous cycling ramp protocol seven days later. Sawka et al. (1985) showed that heat acclimation significantly increased both $\dot{V}O_2$ max and peak power output (W_{max}) for cycling in hot and cool environments (Hot: 4.2%, 1.9%; Cool: 3.5%, 3.8%, respectively). However, Sawka et al. (1985) also showed that $\dot{V}O_2$ max for heat acclimated soldiers exercising in a hot environment (49 ˚C) was still significantly lower compared with the same soldiers exercising in a temperate environment (21 ˚C). They also speculated that the 50% $\dot{V}O_2$ max training intensity during heat acclimation probably had a training effect because they had not accounted for the cumulative effect of exercise and heat as stressors. The study was not balanced by a control group and the beneficial, statistically significant improvements in W_{max}, $\dot{V}O_2$ max and heart rate were largely ignored by the authors.

Lorenzo et al. (2010) demonstrated significant performance improvement in temperate environments after heat acclimation or acclimatisation. In hot (38 ˚C, 30% RH) and cool (13 ˚C, 30% RH) environments, respectively, heat acclimation significantly increased $\dot{V}O_2$ max (8.2%, 4.9%), average cycling power output (7.5%, 5.3%), and cycling time trial performance (8%, 6%) (Lorenzo et al., 2010). Furthermore, post-run heat acclimation using 30 min of passive sauna bathing a day for three weeks improved run time to exhaustion by 32% (Scoon et al., 2007), though the thermal environment for the performance tests was not reported and an eight-day swimming acclimatisation training camp (30 ˚C, 80% RH, pool temperature 30 ˚C) improved 400 m freestyle times in temperate water (27 ˚C) 30 days later though the performance improvements were probably not related to superior sweat responses or cardiovascular adaptations (Convertino, 1991; Harrison, 1985). However, subsequent studies have demonstrated no significant cycling TT performance improvement in temperate environments for trained athletes after heat acclimation over 20 km (Neal et al., 2016), 30-min (Keiser et al., 2015a), or heat acclimatization over 43.4 km (Karlsen et al., 2015a).

The performance and physiological benefits elucidated by Lorenzo et al. (2010) should be read with caution because training intensity during heat acclimation and control training was fixed at 50% $\dot{V}O_2$ max. Firstly, exercise intensity may have caused a training effect. Sawka et al. (1985) identified an unexpected training effect from heat acclimation that they attributed to exercise intensity (120 mins, 9 consecutive days, 49 ˚C, 20% RH, treadmill walking at 40-50% of $\dot{V}O_2$ max measured in a temperate environment). The 40-50% $\dot{V}O_2$ max intensity was set below the level required to induce a training effect in temperate environments according to Pollock et al. (1973), but the combined heat and exercise stress was sufficient to improve physiological adaptation and performance. Secondly, heat stress and exercise stress have an additive effect on genetic up-regulation, in combination they are an additional stressor in and of itself that drives physiological adaptation (Kodesh et al., 2011). Training stress is difficult to quantify especially when multiple stressors affect the individual. Typically exercise intensity is reduced to cope with a greater number (e.g., heat plus dehydration) or intensity (e.g., 2000 m altitude vs sea level) of stressors (Schlader et al., 2011d, 2011b). Humans are particularly good at quantifying their responses to stressors during exercise using perceived exertion (Borg, 1982; Crewe et al., 2008). Setting an identical (though individualised) workload in different heat stress environments induces different strain and therefore physiological responses (Selye and Fortier, 1950). Lorenzo et al. (2010) ensured a positive performance outcome because the physiological strain and subsequent adaptation were greater, the only possible performance decrement was excessive heat and exercise stress causing sickness (or worse). Thirdly, heat acclimation protocols that prescribe a constant load (Lorenzo et al., 2010; Nadel et al., 1974; Nielsen et al., 1993, 1997; Robinson et al., 1943; Sawka et al., 1985) or controlled hyperthermia (Fox et al., 1963, 1967; Garrett et al., 2009; Henane and Valatx, 1973; Patterson et al., 2004a) protocol do not replicate the way athletes volitionally heat acclimate or acclimatise (Candidate's unpublished observations). Instead, athletes use perceived exertion to control their effort, ensuring they finish the exercise task while maintaining homeostasis. The recent published studies from Karlsen et al. (2015), Keiser et al. (2015), and Neal et al. (2016) cast doubt on the ergogenic effect of heat acclimation for temperate performance. The ergogenic potential of heat acclimation has also been

discussed thoroughly in a Point-Counter-Point Analysis (Minson and Cotter, 2016a, 2016b; Nybo and Lundby, 2015a, 2015b).

2.8 Conclusion

Human physiological responses to fatigue, exercise, and heat have been researched extensively, but few have examined the physiological mechanisms that underpin fatigue, adaptation and performance over different performance durations for elite and highly trained endurance athletes using study designs that replicate the variable performance demands and environmental challenges of real-world competition (Mora-Rodriguez et al., 2008). Two studies have observed the performance and physiological responses of elite triathletes during draft-legal racing in the field, however, neither investigated thermoregulatory responses (Bernard et al., 2009; Etxebarria et al., 2014a). Etxebarria et al. (2013, 2014) noted that the cycle section of the triathlon was punctuated by repeated supramaximal efforts at variable intensity, and that the large variability in power output would amplify any differences in physiological responses and performance outcomes for individual athletes. Further, the athletes with the highest power output for a 40-km time trial may not necessarily be the most successful in a draft-legal cycle leg and 10-km run (Etxebarria et al., 2013). Three studies (Hue et al., 2007; Lorenzo et al., 2010; Scoon et al., 2007) have demonstrated physiological adaptation and superior performance in hot and temperate competition environments from a structured heat acclimation intervention balanced by a control group. However, at least three subsequent studies have shown no performance benefit in a temperate environment (Karlsen et al., 2015a; Keiser et al., 2015a; Neal et al., 2016), which has generated considerable discussion (Minson and Cotter, 2016a, 2016b; Nybo and Lundby, 2015a, 2015b). Important areas that deserve further investigation are the acute and adaptive central responses from highly-trained endurance athletes to heat. The triathlon race observations that prompted this thesis noted that some individuals were more prone to hyperthermic hypocapnia. The experiments sought to identify the performance and physiological deterioration, and proposed that a heat acclimation intervention could assist highly-trained endurance athletes. Building upon the literature and longitudinal observations, this thesis

had three purposes: 1) observe and measure elite triathletes' physiology and exercise behaviour during racing; 2) measure the physiological responses of highly-trained endurance athletes from a simulated race performance and environment, versus a short-duration, maximal-intensity exercise bout in a controlled laboratory environment, and 3) develop and assess a heat-conditioning intervention to reduce physiological limitations and improve performance.

3 ELITE TRIATHLON RACE PERFORMANCES IN TEMPERATE & COOL ENVIRONMENTS

3.1 Chapter Introduction

This chapter presents an observational field study conducted within an elite national triathlon team racing in different environments in New Zealand and Australia. The intent was to observe racing performance and potential critical factors within such performance in temperate and hot environments, and then apply these findings subsequently to a series of laboratory protocols aimed at understanding the physiological mechanisms that underpin performances in the field (see chapters four and five). In many cases, the same athletes observed and measured in the field were participants in the subsequent laboratory testing. The athletes were members of the New Zealand Triathlon High Performance squad in the under 19 (U19) and 23 (U23) years of age, and senior elite competitive categories competing in International Triathlon Union (ITU) events over the Olympic distance (1.5 km swim, 40 km bike, 10 km run). Physiological and performance race data were measured and recorded from athletes in New Zealand and Australia during 2010 and 2011.

3.2 Abstract

Information about elite triathlon performance and physiological responses in different environments is sparse. This study aimed to obtain field data from international races. Fourteen elite Olympic distance triathletes (6 female, 8 male) in the New Zealand High Performance Triathlon squad were recruited to participate (age: 24 ± 4 years; height: 175.7 ± 8.7 cm; mass 65.5 ± 9.3 kg; $\dot{V}O_2$ peak: 60 ± 5 $mL \cdot kg^{-1} \cdot min^{-1}$). Triathletes competed in four races in 2010-11: Two ITU Oceania Championships in Wellington, New Zealand (TEMP 1 & 2), and two ITU World Cup races in Mooloolaba, Australia (HOT 1 & 2). Performance data were collated using electronic race splits supplemented with stopwatch splits. Wet bulb globe temperature (WBGT) was recorded at the start of the swim and run stages. Physiological data were measured continuously for heart rate (HR) and before and immediately after the race for core temperature (T_C; gastro-intestinal pill) and finger oxygen saturation (SpO_2). Data are reported using descriptive statistics [mean (SD)] and inferential statistics (Hopkins, 2006) in which minimum functionally-important differences were calculated as > 0.5% for performance (Paton and Hopkins, 2005). WBGT was 7.7 ˚C higher in HOT compared with TEMP. As WBGT increased, 10-km run time increased [slope: 16.7 (13.9) $s \cdot ˚C^{-1}$, 90% CI: 9.1 to 24.3 $s \cdot ˚C^{-1}$, P = 0.097]. Run time was 9.8% slower in HOT 1 compared with TEMP 1 [TEMP 1: 34:42 (3:05) vs HOT 1: 38:08 (4:21) min:s; 90% CI: 1:45 to 4:49 min:s, 100% most likely harmful, P = 0.01] and 2.9% slower in HOT 2 compared with TEMP 2 [TEMP 2: 34:41 (2:42) vs HOT 2: 35:41 (3:08) min:s; 90% CI: -0:27 to 2:06 min:s, 81% likely harmful, P = 0.25]. T_C increased in TEMP [Pre: 36.40 (0.64) vs Post: 37.95 (0.27) ˚C; 90% CI: 0.71 to 2.38 ˚C; P = 0.02] and HOT [Pre: 37.10 (1.20) vs 38.85 (1.14) ˚C; 90% CI: 0.60 to 2.89 ˚C; P = 0.006] and was similar between environments pre- and post-race. HR in the final 10 min of the race was also similar. SpO_2 post-race was lower in TEMP [Pre: 98.0 (1.0) vs Post: 95.0 (2.2)%; 90% CI: -4.1 to -2.0%; P = 0.01] and HOT [Pre: 98.1 (0.3) vs 87.3 (5.8)%; 90% CI: -13.3 to -8.3%; P = 0.09], with HOT lower than TEMP (90% CI: -10.3 to -5.0%; P = 0.06). Triathletes ran positive splits (i.e., run time slowed) in TEMP and HOT, but pace in the first kilometre was considerably slower, and deteriorated more in HOT. In summary, performance deteriorated more in hot conditions but with similar HR, indicating that cardiovascular capacity may be a key mediator of performance.

3.3 Introduction

Elite triathletes represent a highly-trained athletic group with few performance and physiological data available. Race performance data from specific

international events for elite triathletes have been published (Le Meur et al., 2011; Vleck et al., 2006, 2008), however, these studies did not link performance (stress) with the physiological responses (strain) experienced by triathletes. Previous studies that have linked performance and physiology typically use race simulations with sub-elite athletes (Delextrat et al., 2005a; Hausswirth et al., 2009; Kreider et al., 1988a; Laursen et al., 2000; Peeling and Landers, 2007; Peeling et al., 2005). The simulations addressed important physiological questions unique to triathlon performance such as pacing, thermoregulation, and race strategy; but unfortunately were not conducted in a race-specific setting. A study that describes performance of elite triathletes and their concomitant physiological responses in that race context is required to understand the sport better and improve coaching and sport science interventions.

A key variable to triathlon performance and physiology is the thermal burden of sustained, intense exercise in environments that lessen dry or evaporative heat loss. Most ITU triathlons are held in temperate environments [15-22˚C, 20-50% relative humidity (RH), 0.34-1.32 kPa water vapour pressure (PH_2O)], but temperatures may range from 11 to 35 ˚C (unpublished observations of the candidate's, by virtue of measuring conditions at ITU races). Run performance has been shown to slow considerably with increased environmental temperature during marathons (Ely et al., 2007; Maughan, 2010), an event that has a similar duration to Olympic distance triathlon (~2 hours). A triathlon race simulation study performed in the laboratory showed participants reached the highest T_C during the run (Kreider et al., 1988a) and field studies from Ironman triathlon showed that the highest T_C (39.4 ˚C) was reached ~10 km into the run (Laursen et al., 2006), indicating that running is the most thermally stressful race discipline (notwithstanding also being last). However, no study has measured T_C for elite triathletes while racing in ITU events, nor have T_C measures been compared for races in temperate and warm environments.

Strategically, triathletes race using a positive pacing strategy; i.e., starting the swim, bike and run sections as fast as possible, then gradually slowing down (Abbiss and Laursen, 2008; Vleck et al., 2006, 2008). Research has also focussed on the detrimental effect of swimming on cycling (Peeling et al., 2005),

and cycling to running performance, in laboratory simulations (Galy et al., 2003; Hausswirth et al., 1999). However, there is no information that describes the physiological consequences of a positive pacing strategy in elite triathlon racing. Laboratory race simulations have demonstrated greater $\dot{V}O_2$, $\dot{V}_E$ and HR strain in each subsequent discipline caused by physiological drift across fixed-pace and time trial protocols (Hausswirth et al., 1999; Kreider et al., 1988a; Peeling et al., 2005). Conversely, variables such as T_C and arterial saturation in the field are unknown and may differ from those in laboratory studies for reasons described in Chapters 1 and 2 (e.g., unrealistic air flow). Furthermore, the pacing strategies used by triathletes and their physiological responses to different environments in competition are poorly understood, with considerable conjecture among coaches and sport scientists about the appropriate methods to train triathletes for optimal performance.

In light of the above, the purpose of this study was to measure performance outcomes and physiological responses for elite triathletes competing in international races in different thermal environments. Although this was to a large extent an observational study, we hypothesised that performance would be reduced despite similar cardiovascular and thermoregulatory strain when triathletes competed in warm (> 25 ˚C) or humid (e.g., PH_2O > 2 kPa) conditions.

3.4 Method

3.4.1 Participants

Ethical approval was granted by the University of Otago Human Ethics Committee (09/068). Six female and eight male elite triathletes from New Zealand's High Performance Triathlon squad were recruited to participate in the field studies (age: 24 ± 4 years; height: 175.7 ± 8.7 cm; mass 65.5 ± 9.3 kg; $\dot{V}O_2$ peak: 60 ± 5 $mL \cdot kg^{-1} \cdot min^{-1}$). All athletes had competed for New Zealand at the World Championships within the 2010-2012 competitive seasons in U23 or senior elite categories. Athletes completed annual medical screening procedures and signed informed consent forms prior to the first race. The rationale, methods and intended use of the research were outlined to all athletes prior to field testing. Five athletes pulled out of the study due to injury or extenuating circumstances unrelated to the study.

3.4.2 Experimental Design

The design was a non-blinded crossover study with a double control trial (i.e., two races in temperate conditions). Race performance and physiological responses were measured for four separate races: two in Wellington, New Zealand on Saturday the 13th of March 2010, and Saturday the 12th of March 2011, the other two in Mooloolaba, Queensland, Australia on Saturday the 27th of March 2010, and Saturday the 26th of March 2011. Race performances, physiological responses and environmental conditions were compared for the Wellington races in 2010 and 2011 to determine intra-individual reliability. Data from Wellington were compared with Mooloolaba to compare performance, physiological, and environmental differences.

3.4.3 Race Measures

Eight hours prior to the race start athletes swallowed an ingestible radio telemetry core temperature pill (HQ Inc, Palmetto, FL) to ensure the pill passed through the stomach to the intestine for pre- and post-race measurements. Ninety minutes prior to race start, athletes donned a heart rate chest strap (Suunto Memory Belt, Vaanta, Finland) and resting measures for core temperature (T_C), heart rate (HR), and peripheral oxygen saturation (GE TuffSat Pulse Oximeter, General Electric, Fairfield, CT) were recorded. Athletes cycled ~5 min to the race

60-80 min before the start. Another pre-race measure was taken 15 min before race start after athletes had completed their pre-race warm-up and preparation routines. This was the last time coaches or support staff could interact with the athletes until they had finished the race. Immediately after athletes crossed the finish line, HR, SpO_2 and T_C were measured in the finishing chute (within ~20 s of finishing). Athletes who did not finish (DNF) the race were included in the performance, but not physiological data analysis.

3.4.4 Environmental Measures

Environmental data, including ambient air temperature, relative humidity, air velocity and wet bulb globe temperature (WBGT), were measured with a portable weather station (Kestrel 5500 Weather Meter, Nielsen-Kellerman, Boothwyn, PA). Environmental data were collected on the run course 1 km (measured distance) from the start of the run leg under the nearest source of shade to avoid the effect of radiant heat from direct sunlight on the equipment.

3.4.5 Race Performance

Race performance data were collected for swim, bike, run and transition splits from timing mats and ankle bracelets (ChampionChip, Haarlem, Netherlands). The number of competitors (30-75) and the comparable ability of the athletes in each discipline created large groups and therefore drafting. Only run time was used to compare performances in this study. The swim sections of the two races were very different: the Wellington race was a wetsuit swim in the harbour from dive start and pontoon exit, the Mooloolaba race was a non-wetsuit surf swim from a beach start and exit. The cycle was influenced by the different temperature and therefore air density environments: Wellington was considerably cooler – creating greater air density and subsequently slower cycling times (Davies, 1980; Karlsen et al., 2015a; Nybo, 2010; Racinais et al., 2015). Additionally the beneficial drafting influence (Bentley et al., 2008; Faria et al., 2005b; Hausswirth et al., 2001) could not be factored into an accurate performance analysis. Conversely run performance was least influenced by drafting, the courses were relatively similar, and the run leg was the last and most critical race section. Different run course distances were compared by calculating the corrected 10-km time across races. Air velocity and elevation changes in the run course profile were not corrected for comparisons between event locations. The elevation

change on the Wellington run course was ± 1 m each lap for four laps, in Mooloolaba the elevation change was ± 15 m each lap for 4 laps (www.triathlon.org). Splits for the total 10-km time and for each lap were transcribed from the official race results or collected manually on the run course using a stopwatch synchronised with the race start.

3.4.6 Data Analyses

The 10-km run times were combined for inferential analyses for women and men to improve statistical power but performance data from different years was not combined. Run pace is presented separately for male and female athletes in figures, then compared between different environments, to help illustrate pace changes accurately. Physiological data were pooled to improve the statistical analysis power.

3.4.7 Statistical Analyses

The impact of heat stress (environment WBGT) on performance (10-km run time) and physiological responses (T_C, SpO_2, HR) were analysed by simple linear regression and reported as the slope [mean (SD)], 90% confidence interval, P-value (significance set at $P < 0.1$), and Z-score (probability that the slope was 0). 10-km run times were compared between different environments (TEMP 1: Wellington 2010, TEMP 2: Wellington 2011, HOT 1: Mooloolaba 2010, HOT 2: Mooloolaba 2011) by inferential statistics using a post-only crossover as the mean (± SD) with 90% confidence interval for the difference, the percentage difference between means, the percentage likelihood that racing in a hot environment was beneficial, trivial or harmful to performance (Hopkins, 2006) based on a smallest functional performance difference > 0.5% (Paton and Hopkins, 2005), and paired t-test ($P < 0.1$). Run times were compared by temperature (TEMP vs HOT) each year and also across different years (TEMP 1 vs. TEMP 2; HOT 1 vs HOT 2). The effect of run performance time on physiological responses were analysed by correlation using Pearson's r as the slope [mean (SD)], 90% confidence limits and P-value. Physiological data were pooled for TEMP (TEMP 1 combined with TEMP 2) and HOT data (HOT 1 combined with HOT 2) and compared using paired t-tests presented as mean (SD) with 90% confidence interval for the difference and paired t-tests ($P < 0.1$). Data that were not normally distributed were log transformed before analyses.

3.5 Results

3.5.1 Environmental Conditions

The environmental conditions were similar between the triathlon races in Wellington, New Zealand in March 2010 and 2011. However, conditions were substantially hotter and more humid in Mooloolaba, Queensland, Australia in March 2010 and 2011 compared with New Zealand (Table 3.1). As WBGT increased 10-km run time also increased [slope: 16.7 (13.9) s·˚C^{-1}; 90% CI: 9.1 to 24.3 s·˚C^{-1}, P = 0.097; Z: 0.00003; Figure 3.1].

Table 3.1: Environmental conditions recorded at the start of the run leg in Wellington, New Zealand on Saturday 13th March 2010 (TEMP 1), Saturday 12th March 2011 (TEMP 2) for the women's and men's ITU Oceania Championships, and Mooloolaba, Queensland, Australia on Saturday 27th March 2010 (HOT 1), Saturday 26th March 2011 (HOT 2) for the women's and men's ITU Mooloolaba World Cup triathlon races.

	TEMP 1		TEMP 2		HOT 1		HOT 2	
	Women	Men	Women	Men	Women	Men	Women	Men
Time recorded	12:41pm	12:26 pm	2:47 pm	2:30 pm	11:31 am	3:15 pm	2:45 pm	4:37 pm
Ambient Temperature (˚C)	16.8	16.8	17.7	19.5	27.1	26.6	22.1	25.4
Relative Humidity (%)	57	57	75	67	69	62	95	68
Water Vapour Pressure (kPa)	1.06	1.06	1.52	1.52	2.47	2.16	2.53	2.21
Barometric Pressure (hPa)	1015	1015	1029	1029	1018	1016	1018	1012
WBGT (˚C)	13.5	13.5	15.2	16.1	24.2	23.0	21.3	20.8

TEMP 1: Women: n = 6; Men: n = 7.
TEMP 2: Women: n = 4; Men: n = 7.
HOT 1: Women: n = 6; Men n = 10.
HOT 2: Women: n = 3; Men: n = 5.
2 Women and 3 Men completed all 4 races.

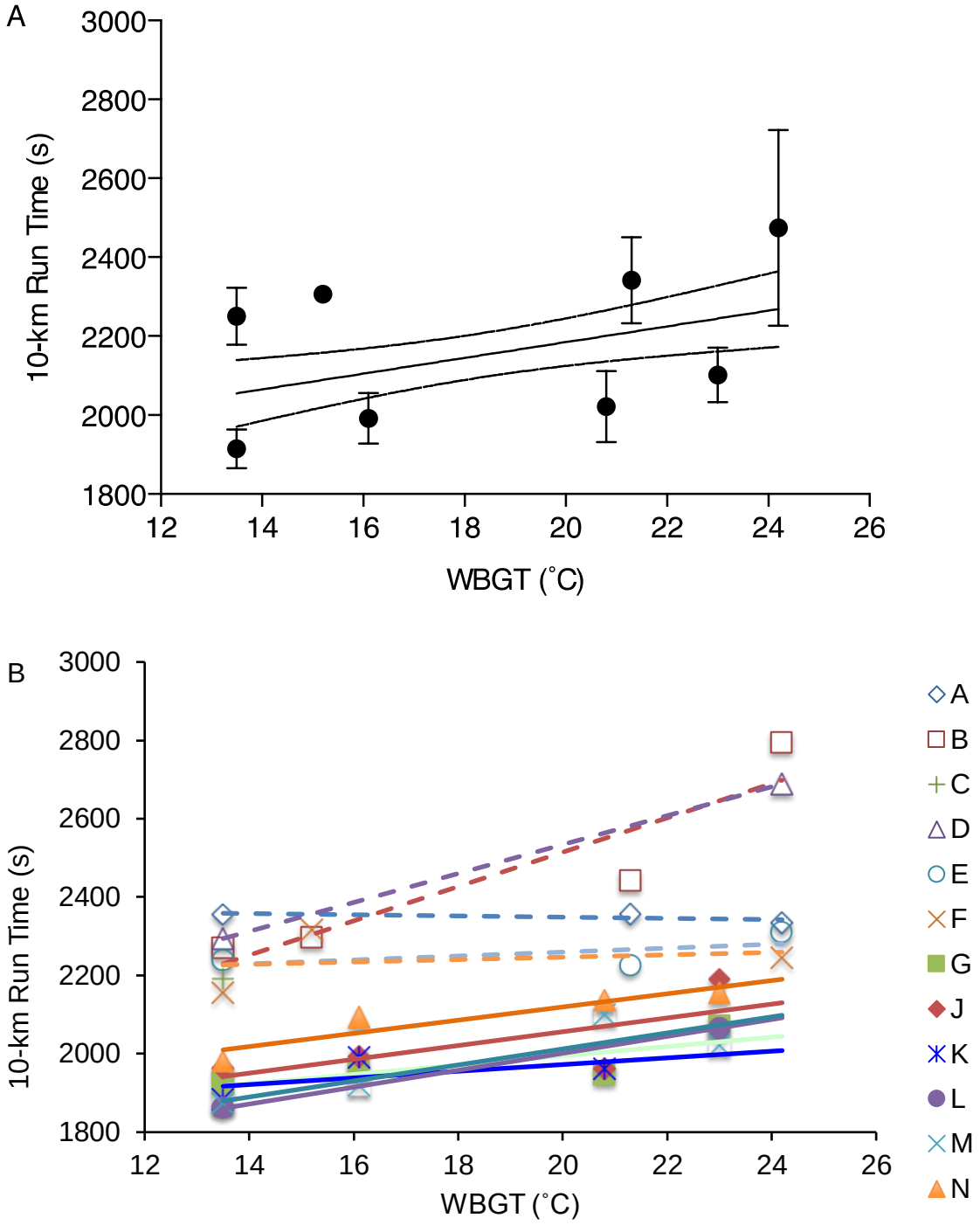

Figure 3.1: 10-km run time (s) in different WBGT environments (˚C) for the pooled cohort of male and female elite triathletes (n = 10). Figure A: Grouped data for 10-km run time in each environment (mean ± SD) and averaged trend line with 90% confidence limits; Figure B: Individual data (trend lines). Data for females are shown using dashed lines, and males as solid lines. NB: Run times from participants H and I were not included because they did not finish.

3.5.2 10-km Run Performance

Five out of twelve athletes completed all four races, with eleven athletes completing a TEMP and HOT race in the same year. All athletes completed at least one TEMP race (Wellington). In the HOT races (Mooloolaba) one athlete did not start (injury) and three did not finish (heat exhaustion). The 40-km cycle times were faster in HOT in 2010 by ~5% between TEMP 1 and HOT 1 and in 2011 by 2.5% between TEMP 2 and HOT 2 for men and women (Table 3.2). The 10-km run times in 2010 were 9.8% (10.1%) faster in TEMP 1 compared with HOT 1 conditions [34:42 (3:05) vs. 38:08 (4:21); 90% CI: 1:45 to 4:49 min:s, 100% most likely harmful, P = 0.01]. In 2011, run times were 2.9% (8.3%) faster when athletes raced in TEMP 2 compared with HOT 2 [34:41 (2:42) vs. 35:41 (3:08) min:s; 90% CI: -0:27 to 2:06 min:s, 81% likely harmful in HOT, P = 0.25]. Athletes slowed down considerably in HOT races, completing the last 2-3 km with the slowest split time for any section of the run (Figure 3.2, Figure 3.3).

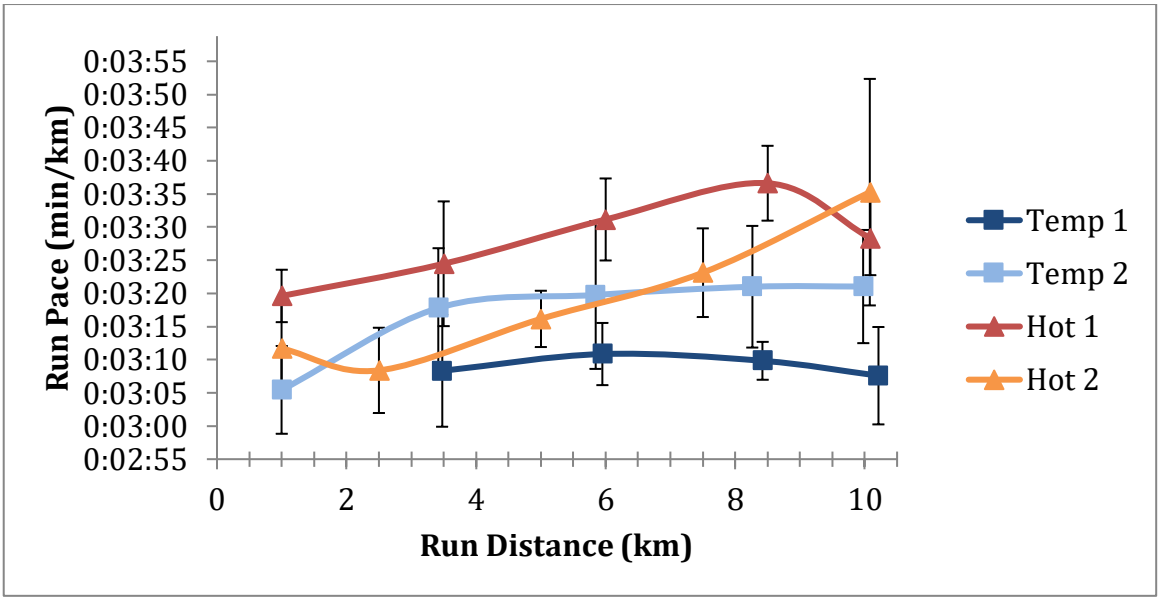

Figure 3.2: Run pace measured at different distance markers on the course for elite male triathletes racing in temperate (TEMP 1 & 2) and hot (HOT) environmental conditions [data presented are mean (SD), n = 6].

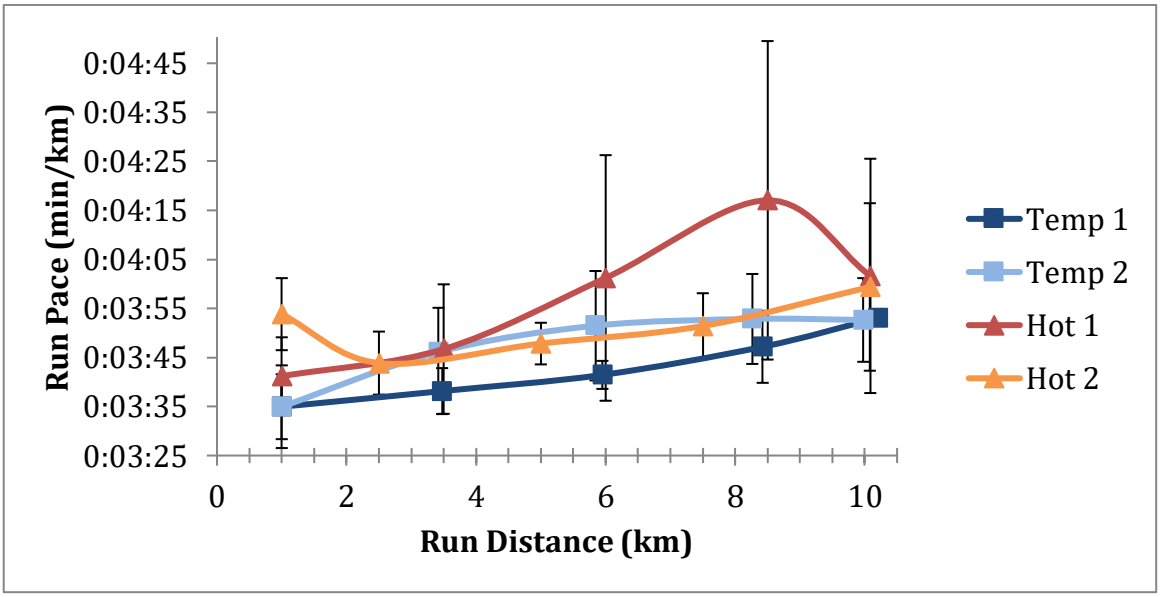

Figure 3.3: Run pace data measured at different distance markers on the course for elite female triathletes racing in temperate (TEMP 1 & 2) and hot (HOT 1 & 2) environmental conditions [data presented as mean (SD), n = 4].

Table 3.2: Average athlete split times (h:min:s) for each section of the triathlon events (Women: n = 4; Men: n = 6). In TEMP races swim to bike transition (T1) was included in the swim time and bike to run transition (T2) was included in the bike time.

		Swim	T1	Bike	T2	Run
Men	TEMP 1	0:18:05	n/a	1:04:10	n/a	0:32:25
	TEMP 2	0:18:31	n/a	1:02:15	n/a	0:33:01
	HOT 1	0:17:53	0:00:20	1:00:40	0:00:17	0:34:57
	HOT 2	0:18:22	0:01:05	1:01:06	0:00:16	0:34:16
	HOT - TEMP	-0:00:10		-0:02:20		0:01:54
Women	TEMP 1	0:20:54	n/a	1:11:40	n/a	0:38:19
	TEMP 2	0:20:07	n/a	1:10:24	n/a	0:38:40
	HOT 1	0:20:01	0:00:18	1:08:45	0:00:18	0:40:36
	HOT 2	0:20:24	0:01:11	1:08:13	0:00:19	0:40:00
	HOT - TEMP	-0:00:18		-0:02:33		0:01:48

3.5.3 Physiological Responses

There were no correlations between run time and physiological responses. Core temperature increased by a similar extent during the races in TEMP (90% CI: 0.71 to 2.38 ˚C, P = 0.02) and HOT (90% CI: 0.60 to 2.89 ˚C; P = 0.006) and was similar between environments after pre-race warm-up [36.40 (0.64) vs. 37.10 (1.20); 90% CI: -0.29 to 1.69 ˚C; P = 0.50], and post-race [37.95 (0.27) vs. 38.85 (1.14); 90% CI: -0.13 to 1.93 ˚C; P = 0.30). Two athletes experienced heat exhaustion and stopped running

Heart rate during the last 10 min of the run was similar between TEMP and HOT [179 (7) vs. 177 (9); 90% CI: 1 to 9 beats·min^{-1}]. Two participants experienced heat exhaustion; their HR's were discounted from the analysis (Figure 3.4).

Finger oxygen saturation (SpO_2) was similar after pre-race warm-up for TEMP compared with HOT [98 (1) vs. 98 (0.3); 90% CI: -0.6 to -0.8%; P = 0.50], and was higher at the start compared with end of the race in TEMP [98 (1) vs. 95 (2); 90% CI: -4 to -2%; P = 0.01] and HOT [98 (0.3) vs 87 (6); 90% CI: -13 to -8%; P = 0.09], with HOT lower than TEMP immediately post-race (90% CI -10 to -5%; P = 0.06).

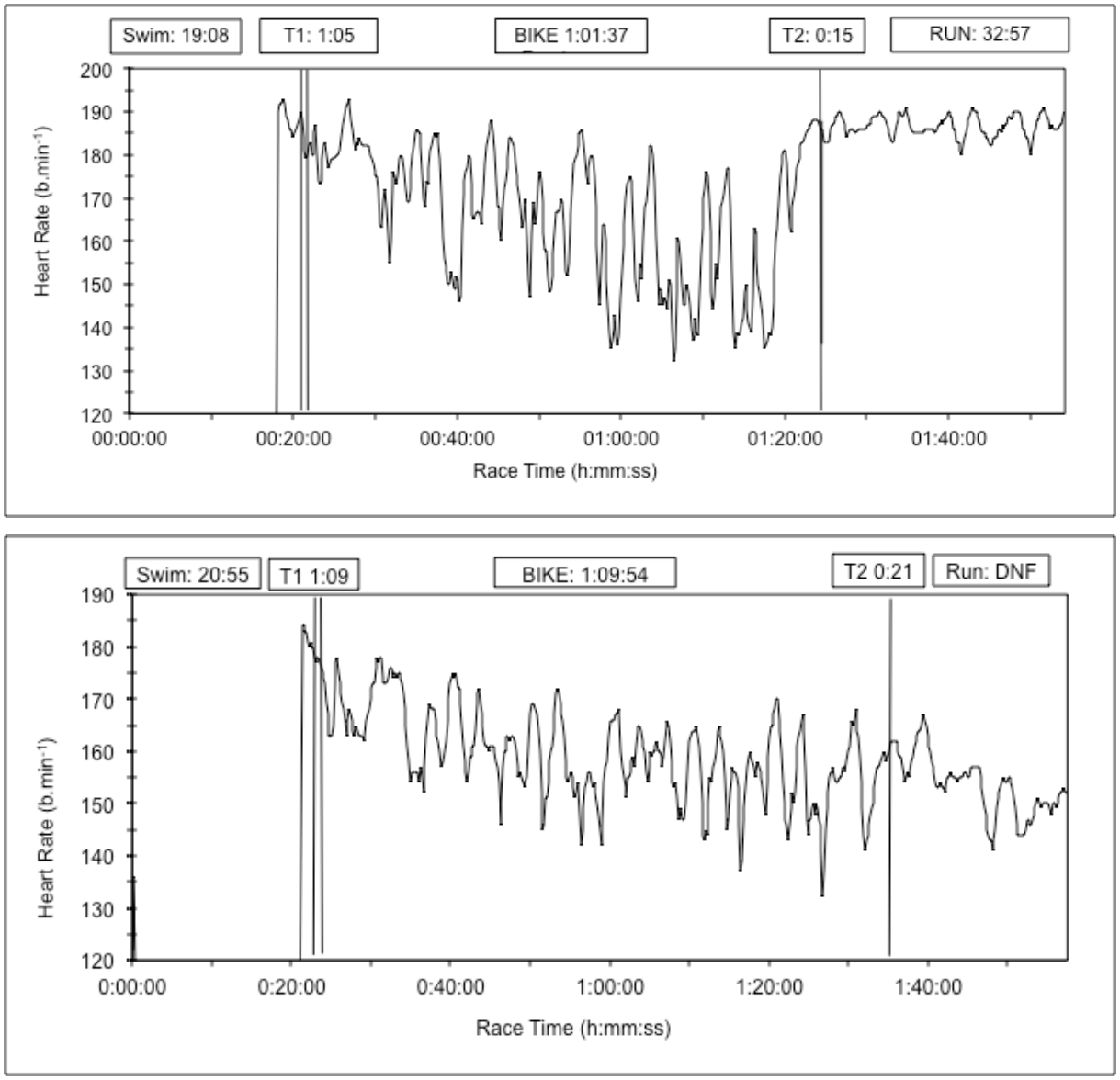

Figure 3.4: Race heart rate data from HOT 2 showing; Top: an athlete who successfully completed the race, and Bottom: an athlete who experienced heat exhaustion during the run and did not finish (DNF) the race. NB 1: the data are from the same race, but not the same athlete. NB 2: Heart rate during the run in A is a normal profile. Heart rate in B was considerably lower and continued to decrease. NB 3: Sea water causes considerable interference with the heart rate monitor signal, heart rate recording only started when athletes stood and started running out of the water.

3.6 Discussion

This appears to be the first study that has linked performance data from elite triathletes to their physiological responses to racing in different thermal environments. Run performance in humid heat was considerably slower than in a temperate environment, and was associated with higher thermal strain (as reflected by higher final T_C) and potentially limiting cardiovascular strain. Specifically, heart rate in the last 10 minutes of the race was matched between HOT and TEMP at a presumably-maximum sustainable level, with different running velocities, while oxygen delivery was impaired in perhaps two ways. First, arterial saturation was lower (likely due to the higher Tc and/or acidity of pulmonary venous blood) and perfusion of exercising muscle may have been more compromised in the warmer and more humid conditions with oxygen extraction already maximised (Gonzalez-Alonso and Calbet, 2003). The WBGT was ~7.6 ˚C higher in HOT, sufficient to hinder race performance and physiological responses.

3.6.1 Slower 10-km Run Performance

Average 10-km run time was slower in a HOT environment probably because athletes ran to an anticipatory pacing strategy according to their thermal perceptions (Schlader et al., 2011d, 2011c; Tucker, 2009). In TEMP environments athletes started the run as fast as possible and slowed throughout the race until the last 1-3 km, where they maintained their pace, similar to data published by Abbiss and Laursen (2008), Le Meur et al (2011) and Vleck et al. (2008). However, in HOT 2, the first kilometre was not the fastest (Figure 3.2; Figure 3.3). Many athletes ran slower in HOT than TEMP at the start of the run, then between two to five kilometres athletes attained their fastest speeds for the run stage. In the final five kilometres pace deteriorated further despite similar HR, indicating that physiological strain was similar to or greater than TEMP conditions.

The relatively slow first kilometre of the run in HOT conditions may have been caused by prolonged hyperthermia from high-intensity swimming (~20 minutes) and cycling (~60 minutes). The swim-bike section of the race could be considered a fixed-pace protocol, because the substantial benefits from drafting

during the swim (Chatard et al., 1998; Peeling and Landers, 2009) and bike (Hausswirth et al., 1999) forced athletes to alter their intensity to whatever was required to stay in a drafting group. Insufficient heart rate and power data were collected to make accurate comparisons for the swim and bike legs between races, further, the swim and cycle distances and demands (e.g., topography, currents) were different, but it is reasonable to assume that exercise intensity was similar throughout all races because the triathletes were motivated to race as hard as possible. Furthermore, at the same power output a cyclist covers any given distance faster in a warmer environment due to the lower air density (Davies, 1980; Karlsen et al., 2015b; Nybo, 2010; Racinais et al., 2015).

The run section from 2-5 km was the fastest part of the race in hot conditions for most (69%) male and female triathletes. Athletes may have voluntarily (or involuntarily) self-paced by decreasing their run pace in the first kilometre to limit physiological strain and restore thermoregulation (Adams et al., 1975; Byrne et al., 2006; Kenefick et al., 2007). Peripheral fatigue may have been lower at the same point in the race because athletes had not been capable of racing at their normal intensity (Nielsen et al., 1993; Nybo and Nielsen, 2001a). Additionally, central fatigue mechanisms that limited run pace were alleviated by thermoregulation, allowing athletes to feel better and run faster.

In the same run section two athletes experienced heat exhaustion and stopped running. Both cited breathing problems, overwhelming heat, and deteriorating run speed despite a strong desire to continue racing. They ran at their competitors' pace rather than their capabilities, creating considerable fatigue that likely exceeded thermoregulatory capacity (Nielsen et al., 1993; Sawka et al., 1996), limited motor output (Nybo and Nielsen, 2001a; Tucker et al., 2004), and induced heat exhaustion (Armstrong et al., 1996; Schlader et al., 2011a).

Run pace declined by a greater magnitude over time in the hot environment, reaching its nadir in the final 2.5-km lap in HOT 2, however, in HOT 1 all athletes could run faster in the final lap. Pace may have deteriorated to match cardiovascular capacity depending on the thermal stress athletes experienced in each environment. By contrast, run pace in the last lap improved in temperate

conditions (except women in TEMP 1). Athletes achieved superior pacing strategies in TEMP 1, TEMP 2, and HOT 1, something they were incapable of doing in the HOT 2, possibly because they could not accurately anticipate the physiological inputs and rate of physiological change relative to the magnitude of exercise left to complete (Tucker, 2009). Further, Hausswirth and colleagues have reported that triathletes who ran the first kilometre of an Olympic distance race simulation too fast – even in a temperate environment – ran slowest in the last 2-3 km, achieved the worst total run time and substantially increased their metabolic, ventilatory, and central fatigue responses (Brengelmann et al., 1977; Gonzalez-Alonso et al., 1999; Johnson and Park, 1979; Rowell, 1977b). The triathlon-specific performance limitations highlighted by Hausswirth et al. (2009) were exacerbated by exercising in the heat in this study, specifically: 1) metabolic load is increased by blood flow redistribution to skeletal muscle and skin (Brengelmann et al., 1977; Gonzalez-Alonso et al., 1999; Johnson and Park, 1979; Rowell, 1977b); 2) greater respiratory muscle fatigue caused by prior cycling exercise (Boussana et al., 2001; Galy et al., 2003) is aggravated by hyperthermic hyperventilation (Fujii et al., 2008, 2015) that may consume 20-24% of $\dot{V}O_2$ when exercising at maximal intensity (Harms et al., 1998b), and 3) central fatigue reduces motor unit recruitment (Tucker, 2009), which is further impaired by heat (Tucker et al., 2004).

3.6.2 Physiological Responses

Post-race T_C was non-significantly higher in HOT, possibly because athletes paced themselves more conservatively in hot environments. Additionally the sample size for each group was small (TEMP: n = 4, HOT: n = 5), making comparisons between environments unclear. Most athletes were measured within ~20 s, though some athletes took up to 90 s before an accurate reading could be taken. Despite the delay the T_C would not have substantially altered, accurately reflecting T_C for the athletes at the end of the race. Athletes were instructed by their coaches to alter their pre-race routines and warm-up for hot conditions by arriving at the race venue later, performing a shorter warm-up of only swimming, and using wet towels and ice slushies to keep themselves cool prior to race start. The pre-race interventions may have beneficially influenced T_C, which was non-significantly higher in hot conditions. By contrast, athletes

completed longer land and water-based warm-ups for races in temperate conditions with the specific aim to raise T_C.

The T_C varied considerably between individuals due to different heat storage capacities. Triathletes are typically lean with a large surface:mass ratio that allows them to offload heat effectively (Sleivert and Rowlands, 2012). However, triathletes who carry more skeletal muscle (typically tall muscular athletes) may generate and store more heat during races in hot *and* temperate environments. For athletes that generate and store more heat, temperate conditions enable larger temperature gradients from the core to the skin and the skin to the environment via lower ambient air temperature and skin blood flow that allows greater thermal transfer; reducing T_C and heat stress that can inhibit performance (Cuddy et al., 2014). The lower vapour pressure also facilitates heat transfer by allowing greater rates of evaporative heat loss, and additionally helps by reducing the sweating requirement and reducing skin temperature. Lower T_{Skin} also reduces cardiovascular strain, and thus potentially also metabolic strain (Cotter et al., 2001; Gonzalez-Alonso et al., 1999; Kozlowski et al., 1985).

While T_C increased substantially from start to finish in TEMP and HOT environments, and was substantially higher in HOT immediately post-race, HR recorded in the last 10 minutes of each race were almost identical. Heart rate data were not analysed for the swim section because the signal dropped out for many athletes as soon as they dived into the water. During the bike section, insufficient power and heart rate data were collected to make accurate comparisons for workload or physiological strain. Athletes also drafted in pelotons at a pattern and intensity they could rarely control. Triathletes paced the start of the run well in temperate, but poorly in hot conditions, again making comparisons difficult between environments. The last 10 minutes of the run were compared because it represented the section of the race that was at self-paced and best-effort intensity for each individual.

The similar HR's measured for the last 10 minutes of the run infers that the triathletes experienced the same cardiovascular strain irrespective of environments. The cardiovascular system may have been the primary limiting

factor. Firstly, sweat loss during the race may not have matched fluid intake, causing dehydration and hypovolemia that reduced end diastolic and stroke volume (González-Alonso, 1998; González-Alonso et al., 2000). Secondly, high skin blood flow to transfer heat from the core to the periphery and environment combined with high skeletal muscle blood flow may have limited end diastolic volume (Johnson, 1992; Johnson and Park, 1979). The HR responses from these triathletes concurs with research that showed a large performance decrease in a hot environment combined with similar HR responses for elite road cyclists that performed 30-minute time trials in temperate and hot laboratory environments (Tatterson et al., 2000). The triathletes may have different responses because they were highly-motivated elite athletes competing in international events, racing at the physiological limit of their cardiovascular systems in all races; therefore, it seems reasonable to conclude that they simply ran slower in hot conditions, with maximised cardiovascular response.

Contrary to the HR response, SpO_2 measured at the finger immediately post-race decreased considerably more in hot compared with temperate environments. Finger SpO_2 was taken to infer changes in arterial and to a lesser extent - muscle oxygenation. The lower SpO2 observed in hot environments may have influenced slower run pace via lower oxygen delivery from blood to muscle, combined with lower leg skeletal muscle blood flow and higher skin blood flow. Laboratory-based research has shown muscle oxygenation is significantly reduced by high T_C and heat stress for moderately-trained athletes exercising in a hot, low airflow environment (Nielsen et al., 1990; Pearson et al., 2011), and brain oxygenation decreases (despite elevated O_2 extraction) for highly-trained athletes exercising to exhaustion in a similar environment (Gonzalez-Alonso et al., 2004), which was the likely limiting factor to exercise completion or performance (Rasmussen et al., 2010b). Though finger SpO_2 has considerable limitations and applications to the physiological responses discussed above, it was the only feasible measure for elite athletes racing in a highly-motivated setting, and may provide valuable information on oxygen availability for aerobic power when considered alongside the Tc data at the end of an elite triathlon race in different environments.

3.6.3 Limitations

Of the twelve athletes who started the study, five completed all four races. The attrition rate was especially high for the races in the hot and humid environments, where three athletes did not finish (one athlete DNF both HOT races) and six did not start due to injuries sustained prior to the race or because they did not meet Triathlon New Zealand selection criteria. The study included nearly all the triathletes in the New Zealand elite team competing in the Oceania Championships in 2010 and 2011 and the Mooloolaba World Cup in 2010 and 2011; we could not have recruited more participants because additional athletes would be sub-elite and/or not competing in the environmental conditions we were investigating.

The small sample size was also confounded heavily by methodological problems and weather outcomes. On multiple occasions the radiotelemetry system (pills or receiver/logger) designed to measure T_C failed to show a reading, returned clearly erroneous measurements, or passed too quickly through the GI system. Heart rate chest straps usually provided accurate data, but often slipped when athletes dived into the water at the start of the race, especially in hot conditions when wetsuits were not worn. Conversely, the heart rate chest straps caused considerable discomfort on the run if they were too tight, creating a real or perceived restriction to the athlete's ability to breathe. Pre-race and immediate post-race measures could not be obtained for every athlete at every race because access was often restricted in the start and finish areas. This was especially problematic in the finish chute area immediately after triathletes crossed the line. Athletes (quite rightly) did not want to look for or move to the investigator at the end of a gruelling and - at times - galling race result.

The measurement devices used in this study substantially limited the accuracy and reliability, and thus availability, of physiological data. Future studies involving elite endurance athletes competing in adverse environments have considerable potential to improve our understanding of the limits of human physiology once monitoring technology has become more reliable and user-friendly. In 2010 and 2011 the difficulty measuring pre- and post-race variables emphasised that it was impossible to take accurate manual measurements without considerable

technological improvements that store or transmit data throughout the race. Global positioning system devices have improved in the last 10 years. In 2006, the GPSports SPI10 was the size and weight of three iPhone 6's stacked on top of each other, and worn in a custom-made backpack. In 2016 companies produced water proof (Garmin™, TomTom™) wrist watches capable of measuring GPS and heart rate at the wrist (Fitbit, TomTom), then recording and storing the data by wireless transfer to a smart phone. By contrast, T_C data were, and still are, recorded by a radio frequency telemetry pill that transmits to a receiver the size of a large pocket calculator (HQ Inc) or a 4 cm x 6 cm disc (BMedical); neither proved to be water proof in field testing undertaken by our laboratory group in this and parallel studies. Swimming goggles (Instabeat™) and cycling helmets (LifeBEAM™) have been designed to use infra-red light to measure heart rate at the temple and forehead respectively, but not capillary oxygen saturation. At present, no single technology exists that can measure heart rate, core temperature, peripheral oxygenation, prefrontal cortex oxygenation, run/swim pace, bike power, and swim/bike/run cadence, nor are relevant such devices lightweight, small, waterproof and robust with effective storage and transmitting capabilities – though there are combinations of different devices with ANT+ technology that could achieve these aims.

3.6.4 Implications and Recommendations for Future Research

3.6.4.1 Field Studies

Elite triathletes raced poorly in a hot environment. But faulty and cumbersome equipment considerably limited measurement frequency and accuracy. Further, injury, de-selection and the adverse environment itself diminished the sample size and statistical power.

This appears to be the first study that has attempted to examine the performance and physiological responses from elite athletes competing in international competitions in temperate compared with hot environments. Future research on factors limiting (tri)athletes' performance in the heat should include field studies investigating performance pace and power during elite draft-legal international triathlon competitions, and linked directly to physiological responses. Such

studies will gather insightful information about how human physiology responds to the combined influences of competition and environmental stress, and provide objective information on how to prepare and train elite triathletes for racing in hot or humid environments. Field studies that quantify cardiovascular strain and determine if it is the primary limiting factor to performance - alongside actual and perceived thermal strain - during triathlon racing are critical. Only two field studies have measured triathlon-specific cycling and running performances with physiological responses, showing that a variable power output during the cycle leg causes significantly poorer running times for a sprint triathlon compared with a fixed power or self-paced time trial (Bernard et al., 2007; Etxebarria et al., 2013, 2014a).

Race simulations and laboratory studies offer an incomplete picture, usually because the purpose and methods do not align with the specific race demands or data. One laboratory-based study has compared physiological responses from single disciplines (e.g., swim, bike or run) at a fixed pace with a triathlon race simulation, demonstrating that T_C, HR, cardiac output ($\dot{Q}$), and mean arterial blood pressure (MABP) were significantly higher for the triathlon (Kreider et al., 1988a). To date, technological innovation has developed measurement tools that illuminate only part of the physiological picture; no device has been developed that is adequate and acceptable for triathlon (e.g., can be comfortably worn by triathletes, whilst being robust, light, unobtrusive and yet comprehensively inform triathlon coaches and sport scientists).

3.6.4.2 Laboratory Studies

Valuable information about the performance outcomes and physiological responses unique to triathlon may be advanced by laboratory studies that replicate valid race demands and environments *combined with* comprehensive physiological and psychophysical measurements. The advantages are the control, accuracy, and measurement resolution, and hence reliability, achieved in a climate-controlled laboratory.

A laboratory study specific to draft-legal elite triathlon racing must include fixed-pace and separate self-paced sections in the performance protocol. Drafting in a group is highly advantageous in the swimming and cycling legs of the race (Bentley et al., 2008; Chatard et al., 1998; Hausswirth et al., 1999; Peeling and Landers, 2009) and triathletes will exercise at the intensity required to stay in this group (Peeling et al., 2005; Vleck et al., 2006), as discussed above. However, the physical benefits of drafting are considerably less during the run because run speed must be over 20 km/h to gain a measurable advantage (Pugh, 1970, 1971; Zouhal et al., 2015), and total run time is considerably slower when triathletes run the first kilometre too fast (Hausswirth et al., 2009). Practically, triathletes race at a stochastic intensity reactive to their competitors for the swim and bike legs. The run leg may also be at a variable pace, though athletes usually run at a proactive self-selected intensity to ensure they finish the race in the fastest possible time. A laboratory protocol must be developed to simulate these performance demands and environmental constraints. However, the exercise mode must be cycling because (unlike swimming and running) an effective performance protocol cannot be designed that incorporates authentic self-paced maximal efforts and stochastic pacing strategies. Further, it is not practical to accurately measure the physiological responses to exercise, particularly central physiological responses including brain blood flow and prefrontal cortex haemodynamics. The stress created by appropriate laboratory protocol and ambient thermal settings (i.e., high air velocity within controlled temperate versus hot & humid conditions) should be confirmed by the strain responses.

Pertinent questions arising from this initial field-based study include:

- How do skin and core temperature change in relation to each other before and during a race in different environments?
- How large is fluid loss and how does it – along with skin temperature - influence blood pressure, stroke volume and cardiac output?
- Is heart rate reserve the primary performance limitation or are other (unmeasured) responses important in limiting performance (e.g., concomitant reductions in brain blood flow and oxygenation)?
- Which physiological responses precede heat exhaustion?

- Can these potentially limiting factors be improved by heat conditioning (acclimation or acclimatisation) to improve pacing and performance in hot environments (or indeed in temperate environments given the equivalently limited heart rate reserve in both environments)?

Transferring the findings from this field study to a laboratory simulation where multiple measures can be collected and controlled could provide a more comprehensive picture of the physiological responses triathletes experience that forces some to alter their pacing strategy and others to experience heat exhaustion.

3.7 Conclusion

This appears to be the first study reporting performance and physiological responses in international triathletes racing in different thermal environments. Run performance was substantially slower and SpO_2 was lower in a hot environment. Heart rate and T_C were similar between environments, indicating that the capacity of the cardiovascular system may be a key mediator of performance. Run pace in the first kilometre was considerably slower, and deteriorated more, during the run in the hot and humid environment, indicating that triathletes struggled to pace themselves appropriately and that central fatigue may have also been a performance limitation. Triathletes competing in hot conditions arrived at the race venue later, performed a shorter warm-up of only swimming, and used wet towels and ice slushies to keep themselves cool prior to race start. The pre-race interventions may have beneficially influenced T_C, which was non-significantly higher in hot conditions. More field research is required to describe elite triathlon racing comprehensively. The findings from observational field studies must be transposed and expanded to a controlled laboratory study to investigate the interplay between changes in central and peripheral responses during maximal intensity exercise in different thermal environments.

4.1.1 Participants

Athletes participating in the laboratory studies (Chapters 5-7) had competed at the elite or age-group national championships in triathlon or cycling in the past 12 months. Eleven had represented New Zealand internationally at the world championships in elite fields or performed with distinction (top 10) as age-group athletes at a world championship event. The participants were accustomed to high-volume endurance training throughout the year and completed swim, bike and run training, which was recorded in log books by their coaches while they were taking part in these studies, and obtained for the purposes of the studies. None of the athletes had completed a structured heat acclimation programme prior to the studies, nor were the athletes acclimatised to living in hot or tropical environments. An information sheet was provided to participants to ensure they understood the exercise and measurement procedures. A medical screening questionnaire was completed by participants prior to exercise testing. They had no history of cardiovascular, cerebrovascular, or respiratory disease. Participants were encouraged to consume a high carbohydrate meal, abstain from caffeine, and ensure they drank sufficiently so they were hydrated prior to each trial. Female participants completed the experiments during the follicular phase of their menstrual cycle. The laboratory studies were approved by the University of Otago Human Ethics Committee (09/090).

4.1.2 Experimental Design

The studies utilised a repeated-measures design in TEMP (18 °C, 40% RH, 0.83 kPa PH_2O, 4.0 $m \cdot s^{-1}$ v_a) and HOT (33°C, 60% RH, 3.02 kPa PH_2O, 4.0 $m \cdot s^{-1}$ v_a) laboratory conditions at the same time of day. TEMP trials always preceded HOT trials to avoid any plasma volume expansion generated by strenuous exercise in a hot environment that may have improved (or impaired) aerobic performance (Lorenzo et al., 2010; Sawka et al., 1996). Participants in this study exercised regularly, so the exercise stimulus alone was considered insufficient to enhance plasma volume (Coyle et al., 1990). However, they were not regularly exposed to exogenous heat stress. Test order was therefore based on the physiologically least to most stressful. The order was also necessary to limit the time for the

studies to be completed (6 compared with 11 days) to accommodate the needs of athletes.

Cycling was the exercise mode selected for all laboratory testing. The field study (Chapter 3) concluded that the run section of a triathlon race was both the most physiologically stressful and crucial to the race outcome. Acknowledging this conclusion treadmill running in the environment chamber was investigated and piloted. However, treadmill running was not compatible with accurate brain blood flow measurement during the pilot trials. Additionally, many of the physiological measurements could only be obtained or accurately measured if the athlete stopped running – a study design counter-productive to assessing the exercise stressor. Given that brain blood flow was an integral measure in the laboratory study aims and that accurate measurements required the athletes to stop running an alternative exercise mode was selected. Cycling allowed continuous data collection without interrupting the exercise stimulus. It was also possible to design a cycling protocol that matched the specific race demands (Etxebarria et al., 2014a) that had both fixed pace and self-paced elements. This was piloted with five elite triathletes to determine an appropriate exercise protocol whilst collecting the cardiovascular, respiratory, cerebrovascular, and psycho-physical perceptions.

4.1.3 Preliminary Procedures

Athletes completed an incremental $\dot{V}O_2$ step test in temperate conditions (20 ºC, 40% RH, 0.93 kPa PH_2O, 4.0 $m \cdot s^{-1}$ v) starting at 100 W (females) and 150 W (males) and incremented by 25 W at four-minute intervals. At rest and in the final 60 s of each stage respiratory gas volumes and concentrations (CPET, Cosmed, Italy), heart rate (Polar T31, Kempele, Finland), [La] (LactatePro, Arkray, Japan), SpO_2 (Nonin ipod, Plymouth, MN) and RPE (Borg, 1982) were measured to identify first ventilatory threshold (VT1), VT2, and $\dot{V}O_2$ peak. Strong verbal encouragement was provided to cycle to exhaustion. On a subsequent visit body composition was assessed (ISAK) to estimate lean body mass and each participant completed a familiarisation trial to set up the cycle ergometer dimensions, understand the gear shifting procedure, and race simulation protocol.

Haemoglobin mass was measured using the modified carbon monoxide rebreathing technique (CO dilution; Schmidt and Prommer, 2005) to calculate total blood volume (BV), red cell volume (RCV) and plasma volume (PV). Participants were weighed on arrival and sat for 20-30 min to stabilise PV before their baseline breath concentration of carbon monoxide was measured (Dräger Pac 5500, Lübeck, Germany). Arterialised capillary blood was drawn from a vasodilated earlobe into six 75-µL capillary tubes and measured for Hb and carboxyhaemoglobin concentrations (OSM3, Radiometer, Copenhagen). The ~50 µL of remaining blood in each tube was then centrifuged at 1520 g for five minutes and measured for haematocrit (Hct) using a custom-made Vernier-calliper microhaematocrit reader. Following a full expiration, participants then inhaled a measured bolus of 100% carbon monoxide (1.0 $mL \cdot kg^{-1}$ males, 0.7 $mL \cdot kg^{-1}$ females) introduced from a graduated syringe into 100% oxygen, retained their maximal lung volume for 10 s, then continued breathing on the closed-circuit spirometer for 110 s before another full expiration and removal from the circuit. Carbon dioxide was scrubbed via a soda-lime filter. Two minutes after completing the rebreathing, the blood sampling and analyses were repeated. Residual carbon monoxide concentrations in the breath and closed circuit were measured two minutes later. Haematological volumes were calculated using the equations of Schmidt & Prommer (2005).

Familiarisation trials for the maximal effort 10 s sprint, five-minute time trial (5-min TT), and 60-minute cycling race simulation (60-min SIM) were conducted to prepare athletes for the protocols. For 60-min SIM protocol familiarisation was 1:50 (min:s) paced efforts and 10 s sprints repeated 10 times, as completing the full protocol would have provided no greater benefit and unnecessarily fatigued the athletes. Participants' individual power profiles were calculated from the incremental $\dot{V}O_2$ step test. First, participants completed 1:50 (min:s) at paced effort (equivalent to VT1) and 10 s at 200% W_{max} (FIXED) repeated five times for a total of 10 min. Following a 60-s break to change the cycling protocol participants practiced using the gearing system to produce a self-paced effort as close as possible to VT1 and a maximal effort 10 s sprint (FREE) repeated five

times for 10 minutes. The testing equipment and protocols were explained to ensure athletes understood 60-min SIM.

4.1.4 Pre-test Procedures

Upon arrival, hydration status, urine specific gravity, and nude body mass were measured. If required participants were given 500 mL of sports drink (Powerade™: 30 g carbohydrate, 210 mg sodium). Each athlete fitted a rectal thermistor and HR monitor, then sat quietly for 10 min while baseline measures including HR, T_C, BP, [La], BF_{skin}, and psychophysical perceptions were collected. Athletes then moved into the climate-controlled environment chamber and sat for 30 min in either TEMP or HOT conditions. During this period athletes were instrumented with NIRS, transcranial Doppler, and respiratory gas analysis equipment, then baseline resting measures were recorded for five minutes.

4.1.4.1 Venous Occlusion Plethysmography

Following baseline measures participants stood for a resting forearm skin blood flow measurement using venous occlusion plethysmography to obtain three stable readings.

4.1.4.2 Exercising CO_2 Challenge

Participants mounted a cycle ergometer and were fitted with a mouth piece and nose clip. A pneumotachometer (PNT 3813, Hans Rudolph, Kansas City, MO) and Y-shaped two-way non-rebreathing valve were connected to the mouth piece. The Y-valve drew gas from either room air or a Douglas bag containing 5% CO_2 (21% O_2, Nitrogen balanced) to measure changes in the partial pressure of carbon dioxide ($P_{ET}CO_2$; hypercapnia). Three minutes of baseline data were collected at rest, followed by three minutes cycling at 30% W_{max} breathing room air, then the valve was switched to the 5% CO_2 mixture for three minutes while cycling intensity was maintained. The Y-valve was removed and participants resumed breathing room air immediately afterwards. When athletes had recovered their normal breathing frequency and $P_{ET}CO_2$ they completed a 10-min warm-up at 30% W_{max} and self-selected cadence. The fan was left off for the warm-up period to ensure participants did not shiver in the TEMP environment.

4.1.4.3 Resting CO_2 Challenge

The resting CO_2 challenge included both hypercapnia and hypocapnia to determine a composite cerebrovascular reactivity. Participants sat quietly while a pneumotachometer (PNT 3813, Hans Rudolph, Kansas City, MO) was connected to a Y-shaped, two-way non-rebreathing valve (2700 series, Hans Rudolph, Kansas City, MO) drawing from either room air or a Douglas bag (Harvard Apparatus, Holliston MA) containing 5% CO_2 and attached to the face mask to measure changes in $P_{ET}CO_2$. Three minutes' baseline data were collected breathing room air, then the valve was switched to the Douglas bag containing 5% CO_2 for three minutes. The Y-valve was removed and participants recovered for two minutes breathing room air. They were then instructed to alter their breathing frequency (f) and tidal volume (V_t) to achieve a $P_{ET}CO_2$ of 30 mm Hg and then 20 mm Hg for 2 minutes each. Feedback from a computer screen and verbal cues were provided to help participants achieve the appropriate partial pressures.

4.1.5 Post-test Procedures

Exercise was terminated at volitional exhaustion or at the completion of the trial. Post-test forearm blood flow was measured using venous occlusion plethysmography. The NIRS, Doppler, and respiratory gas analysis equipment were removed from the athlete and they were assisted from the environmental chamber. Participants sat quietly in the anteroom (~20ºC, 40% RH) while physiological (HR, T_C, T_{skin}) and perceptual measures were recorded every five minutes until core temperature returned to pre-exercise temperature and heart rate was <100 beats·min^{-1}. Finally, athletes provided a urine sample for hydration analysis and were weighed to determine change in body mass.

4.1.6 Measurements

4.1.6.1 Cycling Performance

Race simulations were performed on a cycle ergometer (Velotron, Racermate, Seattle, WA) calibrated to within 1% of the factory standard before each trial to ensure the magnetic resistance system was consistent between different environmental conditions. Power output was recorded at 10 Hz for every trial.

The ergometer seat height, seat-handlebar length, and seat-bottom bracket distance were adjusted to the dimensions of each athlete's bike used in racing. During race simulations airflow was directed into participants' frontal plane by a fan (4.0 $m \cdot s^{-1}$ wind speed over a 50-cm diameter; Imasu, Japan).

4.1.6.2 Body Temperature

Core temperature was measured every 30 s from a rectal thermistor connected to a data logger (OM Daqpro 5300, Omega, Stamford, CT) and downloaded to Microsoft Excel® for analysis. Manual recordings were collected at the start of the 10-s sprint and at every minute of the 5-min TT. Skin temperature was measured at four sites (forehead, mid-axilla, forearm, finger) with an infrared thermometer (Digitech, Auckland, New Zealand) held 2 cm from the skin. The thermometer was calibrated against a known temperature of water (30 °C at 2 cm distance). Manual recordings were taken pre-trial, after 30-min sitting in the environment chamber, immediately before, and in the last 30 s of the 5-min TT.

The ingestible radio telemetry core temperature pills (HQ Inc, Palmetto, FL) were not used in the laboratory trials. The primary reason was poor measurement reliability, but additionally the thermistors (OM Daqpro 5300, Omega, Stamford, CT) were re-usable, cost-effective, and reliable. Skin temperature was measured by an infra-red thermometer rather than skin thermistors because covering the skin may have altered the thermal responses of the skin (e.g., heat evaporation by sweat).

4.1.7 Respiratory Gas Exchange

Expired breath volumes and gas concentrations were assessed using a gas analysis system (CPET, Cosmed, Italy), a Pneumotach (Hans Rudolph 7200, Kansas City, MO) and analysers for oxygen and carbon dioxide (AEI Technologies, Pittsburgh, PA). The pneumotach was calibrated with a 3-L syringe (Hans Rudolph, Kansas City, MO). Gas concentrations were calibrated using a two-point calibration against known concentrations of oxygen (room air: 20.95%, medical grade gas 15.03%) and carbon dioxide (room air 0.03%, medical grade gas 5.02%).

Breathing frequency (f), tidal volume (V_t), $\dot{V}_E$, $\dot{V}O_2$), carbon dioxide production ($\dot{V}CO_2$), respiratory exchange ratio (RER), $P_{ET}O_2$, $P_{ET}CO_2$, HR, SpO_2, brain blood flow velocity, brain and muscle haemodynamic data were recorded at 200 Hz to an analogue-to-digital 16-channel Powerlab (ADInstruments, Sydney, Australia) system and displayed in real time on a PC via commercially available software (LabChart 7, ADInstruments). Respiratory data were recorded breath-by-breath from the Cosmed and displayed in real time on a PC (PFT Suite, Cosmed, Rome, Italy), then downloaded to Microsoft Excel® for analysis.

Following baseline measures the Douglas bag (Harvard Apparatus, Holliston MA) containing 5% CO_2 mixed with room air was attached to the Y-valve (2700 series, Hans Rudolph, Kansas City, MO) and inspired by participants; the expired air was directed through the gas analysers described above. Participants cycled for five minutes on the ergometer at 30% W_{max} while breathing from the Douglas bag. Participants resumed breathing room air immediately afterwards.

4.1.7.1 Cardiovascular Variables

Blood pressure was measured manually using a sphygmomanometer and stethoscope by locating the brachial artery at the antecubital fossa and manipulating the cuff pressure using the auscultatory method to find the 1st (systolic) and 5th (diastolic) Korotkoff sounds. Skin blood flow was measured at the forearm using venous occlusion plethysmography. For this, the participant's hand was raised to shoulder height and supported by a researcher while the left

forearm was wrapped with an Indium-Gallium strain gauge (Hokanson, Bellevue, WA) that was 2 cm shorter than forearm circumference. A pressure cuff was wrapped around the left upper arm and connected to a gas cylinder controlled by a precision-valve regulator (School of Physical Education, University of Otago, New Zealand) set to deliver 50 mm Hg pressure when activated by a switch. Three consecutive measurements were collected on LabChart software over 10-s cycles.

4.1.7.2 Near Infra-Red Spectroscopy (NIRS)

Prefrontal cortical and vastus lateralis muscle oxygenation and Hb volume were measured at the right forehead and left thigh using NIRS [Near Infra-Red Spectroscopy; NIRO 200, Hamamatsu Photonics, Japan]. The thigh position was measured as 15 cm above the proximal aspect of the patella and 4 cm lateral to the femoral mid-line, as done by others (Subudhi et al., 2007). The technique has been described previously (Al-Rawi et al., 2001) and applied in this laboratory (Faull et al., 2015; Thomas et al., 2017). The NIRS system measures the absolute concentration (μmol·cm) changes in oxyhameoglobin (O_2Hb), deoxyhaemoglobin (HHb), and total haemoglobin (tHb) based on the modified Beer-Lambert law (Al-Rawi et al., 2001). All three variables have been reported because the information each describes is important not just as a standalone measure, but in relation to the other variables (Ekkekakis, 2009). Tissue oxygenation index (TOI) was calculated as a percentage by the NIRS system ((O_2Hb/tHB) x 100) from the light attenuation gradient between the light emitter to the two photodiodes in the detection probe (via the spatially resolved spectroscopy method). The probes were enclosed in a plastic cover that separated the emitter and detector ~4 cm, held in place by double sided tape and covered by cloth tape to shield external light and provide extra fixation. Measurements were sampled at 6 Hz and analysed as the change from resting baseline values (Subudhi et al., 2009).

4.1.7.3 Brain Blood Flow

Brain blood flow velocity was measured through the middle cerebral artery (MCAv) by a transcranial Doppler ultrasound system (DWL, Compumedics Ltd, Germany) using established search techniques (Willie et al., 2011). The probe was prepared with conductive gel (Aquasonic 100, Fairfield, NJ) to optimise the signal quality, and held in place by an adjustable head band (Spencer Technologies, USA). Photographs were used to standardise Doppler position, angle, sampling depth (30-58mm) and gain (30-50) between sessions (including for data reported in later chapters).

4.1.7.4 Perceptions

Mood status was assessed using psychophysical scales including the thermal comfort and thermal sensation scales (Appendix 11.2 and 11.3; Gagge et al., 1986), and RPE (Appendix 11.5; Borg, 1982). Thermal discomfort was assessed by the cue: “How comfortable do you feel with the temperature of your body?” Thermal sensation was assessed using the cue: “How does the temperature of your body feel?”

4.1.7.5 Hydration Status

Hydration status was measured by collecting a small urine sample and comparing the colour in a clear plastic cup with a numbered colour hydration scale (Appendix 11.5). Urine specific gravity was determined using an analogue refractometer (Atago, Uricon, Tokyo, Japan). Nude body mass was measured to estimate fluid and glycogen loss (Pugh et al., 1967). Participants with a hydration status higher than 3 (Appendix 11.5) and USG higher than 1.020 were given 500 mL of sports drink (Powerade™, CocaCola, Atlanta, GA).

4.1.8 Data Analyses

4.1.8.1 Calculations

Peak power output (W_{max}) for each participant was calculated from the $\dot{V}O_2$ step test from the power output in the final stage completed plus the time achieved and power output in the uncompleted stage ().

$$W_{max} = (P_{Final\ Stage} + T_{Incomplete\ Stage}) \div (T_{Stage} \times P_{Stage\ Increment})$$

P: Power (W)

T: Time (s)

Equation 4.1: Peak power output (W_{max}) calculation from incremental step cycling test

Power output was calculated for each participant as power per kilogram of fat free mass ($W \cdot kg^{-1}$ FFM). Fat free mass was calculated by collating the sum of eight skinfolds in each participant, then calculating an estimated body fat percentage based on the International Society of Kinanthropometry (ISAK) measurement protocols and calculations (; Yuhasz 1962). The percentage was multiplied by body mass, then subtracted from body mass.

$$\text{Estimated \% Fat} = 0.1051 \times \Sigma 6 \text{ skinfolds} + 2.585 \text{ (male)}$$
$$0.1548 \times \Sigma 6 \text{ skinfolds} + 3.58 \text{ (female)}$$

$\Sigma 6$ skinfolds: tricep, subscapular, supraspinale, abdominal, thigh, calf

$$\text{FFM} = \text{Body mass} - \text{Body mass} \times (\text{Estimated \% Fat} \div 100)$$

$$W.kg^{-1}\ \text{FFM} = \text{Power (W)} \div \text{FFM (kg)}$$

Equation 4.2: Fat-free body mass calculation from sum of skinfolds assessment.

5 EFFECT OF AMBIENT HEAT STRESS ON INTENSE CYCLING PERFORMANCE AND PHYSIOLOGY

5.1 Chapter Introduction

This chapter investigated how temperate (TEMP) compared with a hot environment (HOT) influenced explosive (10 s) and short maximal effort (5-min TT) cycling performances. The performance durations and exercise mode in a laboratory environment were a shift away from observing elite triathletes while racing, with good reasons. Firstly, the performance durations were considerably reduced to mitigate factors that normally limit performance and physiological responses in prolonged endurance exercise, including: T_C, brain metabolite accumulation and GI function. Secondly, the exclusive exercise mode was cycling because (unlike swimming and running) it was possible to accurately measure the physiological responses to exercise and design exercise protocols that were authentic self-paced maximal efforts. Thirdly, testing was conducted in the laboratory environment to ensure the experimental protocol was repeatable. Despite the artificial constraints inherent to a laboratory setting such as poor ecological validity and individual motivation, every attempt was made to make the exercise tasks realistic and challenging. The experiments investigated the cardiovascular strain from exercising in a hot environment whilst minimising confounding factors including prolonged core and brain hyperthermia, GI disturbance and elevated brain metabolism. The results and discussion sections in Chapters 5-7 lead with performance power and body temperature outcomes, based on the premise that the other physiological responses follow. However, a very good argument can be made to lead with any other section, especially the brain, which is also a major focus of this series of studies. However, the research questions for this thesis are all designed to interrogate performance outcomes.

5.2 Abstract

This study investigated factors that may contribute to performance in temperate (TEMP) and hot (HOT) conditions. Highly-trained triathletes performed 10-s sprint and 5-min cycling time trials (5-min TT) to assess performance power and physiology. Twelve triathletes (8 male and 4 female, 24 ± 5 years, 176.1 ± 8.1 cm, 68.4 ± 8.4 kg, 58.6 ± 3.6 $mL \cdot kg^{-1} \cdot min^{-1}$ $\dot{V}O_2$ peak) performed a step-incremental cycling test. Two days later each performed 1-2 10-s maximal sprints and a 5-min TT in TEMP [18 °C, 40% relative humidity (RH), 0.83 kPa water vapour pressure (PH_2O), 4.0 $m \cdot s^{-1}$ wind velocity (v_a),] and two days later in HOT (33 °C, 60% RH, 3.02 kPa PH_2O, 4.0 $m \cdot s^{-1}$ v_a). Average and peak brain blood flow velocities were measured at the right middle cerebral artery using transcranial Doppler ultrasound, while near-infra-red spectroscopy was used to measure cerebral (right prefrontal cortex) and skeletal muscle (vastus lateralis) oxygenation (TOI) and volume of oxy- (O_2Hb), deoxy- (HHb) and total haemoglobin (tHb). Ventilation and the partial pressure of end-tidal carbon dioxide ($P_{ET}CO_2$) were measured using gas analysis, SpO_2 by pulse oximetry, and T_C using rectal thermistor and perceptions using scales. Peak values were recorded for 10-s sprints, while data were averaged for the Start (0-30 s), Middle (1-4 min), and End (4:30-5:00) of the 5-min TT. Data [mean (SD),] from TEMP vs. HOT conditions were compared using two-way repeated measure (RM) ANOVA. Power output for 10-s sprints was 11% higher in HOT compared with TEMP ($P = 0.02$), but was 3.4% lower in 5-min TT ($P = 0.01$). Brain HHb was greater in HOT at the Start and Middle ($P < 0.001$), while TOI was 3-4% lower in HOT than TEMP throughout (all $P \leq 0.002$). Leg O_2Hb was lower in HOT at Middle and End ($P < 0.01$), and [La] was 2.5 $mmol \cdot L^{-1}$ greater after the HOT 5-min TT ($P = 0.003$). Participants felt warmer ($P < 0.001$) and warming-up perceived to be harder ($P = 0.004$) before 5-min TT, and felt more heat and associated discomfort afterward ($P = 0.004$). Conclusions: In HOT, sprint power was greater whereas 5-min TT was less, in conjunction with lower prefrontal cortex oxygen saturation and more discomfort, perhaps encouraging a conservative pacing strategy. Yet, metabolic perturbations were still larger in HOT, presumably due to the additional cardiovascular strain with more blood flow redistribution to the skin.

5.3 Introduction

This chapter describes the performance and physiological responses to an all-out 10-s maximal effort and 5-min TT in TEMP [18 °C, 40% relative humidity (RH), 0.83 kPa water vapour pressure (PH_2O), 4.0 $m \cdot s^{-1}$ air velocity (v_a)], and two days later in HOT (33 °C, 60% RH, 3.02 kPa PH_2O 4.0 $m \cdot s^{-1}$ v_a). The durations were selected to determine performance differences between the anaerobic alactic system (10-s effort) and anaerobic lactic and maximal aerobic power ($\dot{V}O_2$ max) systems in the aforementioned environmental conditions. The environments were selected based on weather data collected from the ITU Oceania Championships in Wellington, New Zealand (TEMP) and the ITU World Cup in Mooloolaba, Queensland, Australia (HOT) from 2008 to 2011.

Competitive endurance racing that elicits high-intensity and maximal efforts in environments ranging from temperate to hot drives hyperthermia (Byrne et al., 2006; Lafrenz et al., 2008; Lee et al., 2010; Rowell et al., 1969a) and has a cumulative and negative influence on athletic performance and physiology (Crandall and González-Alonso, 2010; Gonzalez-Alonso and Calbet, 2003; Gonzalez-Alonso et al., 2004, 2008; González-Alonso, 1998; Nybo, 2010; Nybo and Nielsen, 2001b, 2001c; Nybo and Rasmussen, 2007; Nybo and Secher, 2004a; Nybo et al., 2002b; Rasmussen et al., 2010a). The brain is obviously central to exercise performance, and it is powerfully influenced by hyperthermia that significantly reduces responses such as brain blood flow and oxygenation that maintain homeostasis (Crandall and González-Alonso, 2010; González-Alonso, 1998; Gonzalez-Alonso and Calbet, 2003; Gonzalez-Alonso et al., 2004, 2008; Nybo, 2010; Nybo and Nielsen, 2001b, 2001c; Nybo and Rasmussen, 2007; Nybo and Secher, 2004a; Nybo et al., 2002b; Rasmussen et al., 2010a). Hyperthermia also impacts on cardiovascular function, via reduced oxygen delivery to muscle, decreased aerobic energy metabolism in skeletal muscles, increased peripheral fatigue and increased skin blood flow (Gonzalez-Alonso and Calbet, 2003; Gonzalez-Alonso et al., 2008; Hargreaves and Febbraio, 1998; Nybo et al., 2001). Athletes may experience performance deterioration in a hot environment when they feel thermal discomfort even if hyperthermia does not occur (Gagge and Gonzalez, 1973; Gonzalez et al., 1973; Hardy, 1961).

Feeling hot before or during competition may negatively influence performance (Schlader et al., 2011c) and greater thermal discomfort may influence anticipatory pacing (Altareki et al., 2008; Tucker, 2009; Tucker et al., 2004, 2006b), CNS pathways, and central fatigue (Meeusen and Roelands, 2010; Schlader et al., 2011c). Field data collected in Chapter 3 from triathlon racing demonstrated that run pace deteriorated in hot environments with similar cardiovascular strain to temperate environments. However, short-duration high-intensity exercise tasks may influence athletic performance and physiology differently between environments.

Short-duration, high-intensity exercise above VT2 is a critical performance factor in endurance events to respond to course topography, competitive tactics, or the final sprint finish that requires maximal anaerobic effort (Fukuba and Whipp, 1999). Additionally, higher muscle temperature induced by a warm-up or passive heat exposure improves mean power output over 12-30s by 9-15% (Asmussen and Bøje, 1945; Ball et al., 1999; Faulkner et al., 2013; Kilduff et al., 2013), but impairs $\dot{V}O_2$ max over 5-8 min by 9% (Gonzalez-Alonso and Calbet, 2003) and performance time by 1.9% (Altareki et al., 2008). Normal race preparation in a warm environment may be sufficient to improve sprint performance (lasting ≤ 10 s) compared with the same preparation in a temperate environment, although this remains untested. Further, while the cardiovascular and peripheral responses to short-duration maximal-intensity exercise (~5 min) have been investigated (Gonzalez-Alonso and Calbet, 2003), cerebrovascular responses have not.

Against this background, this study aims to quantify performance changes in real-world race simulations alongside physiological responses, focussing on – but not limited to – brain blood flow and oxygenation. It is well known that: 1) heat has a detrimental performance effect, and 2) hyperthermia stresses cerebral homeostasis; however, the magnitude of effects on performance and brain physiology have not been investigated in concert. It was hypothesised that a) 10-s sprint power output would be superior for athletes cycling in a hot – relative to temperate environment, and b) 5-min maximal cycling performance in a hot

environment would elicit lower cerebral oxygenation, brain blood flow, and power output when compared to the same performance in a temperate environment.

5.4 Method

5.4.1 Participants

Eight males and four females (24 ± 5 years, 176.1 ± 8.1 cm, 68.4 ± 8.4 kg, sum of 8 skinfolds: 62 ± 13 mm, 58.6 ± 3.6 $mL \cdot kg^{-1} \cdot min^{-1}$ $\dot{V}O_2$ peak) gave informed consent to participate.

5.4.2 Experimental Design

This study consisted of a lactate test, blood volume assessment, 10 s sprint and 5-min TT in TEMP (18 °C, 40% RH, 0.83 kPa PH_2O, 4.0 $m \cdot s^{-1}$ v_a) and a second 10-s sprint and 5-min TT in HOT (33°C, 60% RH, 3.02 kPa PH_2O, 4.0 $m \cdot s^{-1}$ v_a) on consecutive days (Figure 5.1).

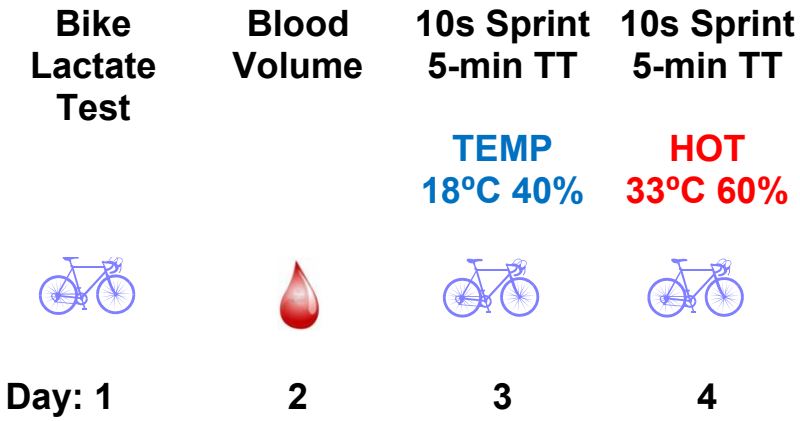

Figure 5.1: Timeline for physiological and performance tests for 10-s sprints and 5-min TT in TEMP and HOT environments

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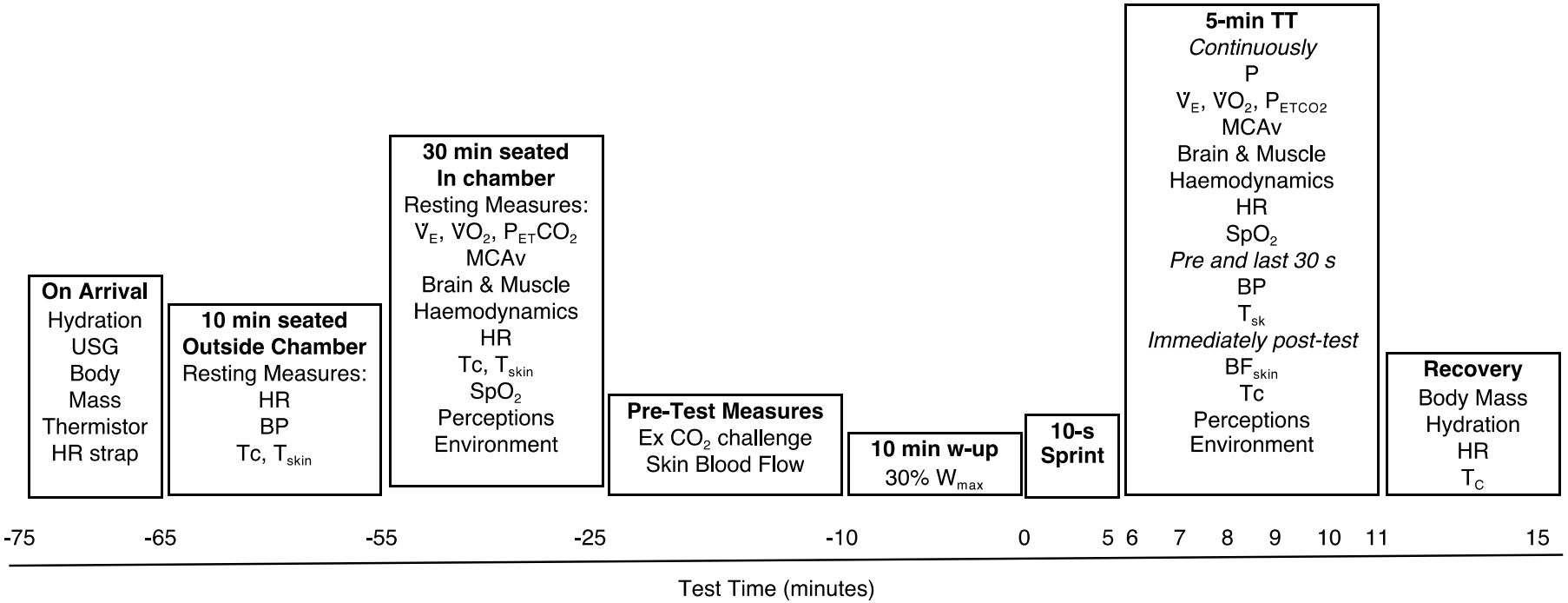
5.4.3 Procedures

5.4.3.1 Pre-Test Procedures

Participants completed the preliminary procedures followed by venous occlusion plethysmography and exercising CO_2 challenge pre-test procedures.

5.4.3.2 Test Procedures

Athletes began the test with a 10-s sprint. They were given a countdown and instructed to keep their cadence high (>100 rpm) and gave verbal feedback when they wanted the Velotron gear increased. Athletes were allowed to perform a second 10-s sprint if they were unsatisfied with their first effort. Following the sprint they completed a 5-min recovery at 30% W_{max}. As soon as each athlete indicated they were ready for the 5-min TT a 1-min count down was initiated. During this period a computer screen providing feedback was covered so only elapsed time was visible. A fan was placed in front of the ergometer to direct airflow onto participants at 4 $m \cdot s^{-1}$ over a 50 cm diameter. Power, HR, ventilation variables, NIRS-derived brain and muscle haemodynamics, and MCAv were recorded continuously (e.g., beat-to-beat/breath-by-breath). Core temperature was recorded every 30 s. During the time trial HR, T_C, and SpO_2 were manually recorded every minute. Immediately before, and in the last 30 s of the time trial, T_{Skin} and BP were measured manually. Psychophysical perceptions and environmental conditions including temperature, humidity, and barometric pressure were recorded immediately before and after (Figure 5.2). Finally, participants completed the normal post-test procedures.



Instrumentation

Thermistor (rectal core temperature), HR Strap (heart rate), near infra-red spectroscopy (brain & muscle haemodynamics), transcranial Doppler ultrasound (MCAv)

Continuous measurements

Core temperature (Tc), heart rate (HR), minute ventilation ($\dot{V}_E$), oxygen consumption ($\dot{V}O_2$), partial pressure end-tidal CO_2 ($P_{ET}CO_2$), brain blood flow velocity (MCAv), NIRS-derived brain and muscle oxyhaemoglobin content, deoxyhaemoglobin content, tissue oxygenation index (brain & muscle heamodynamics), arterial O_2 saturation of Hb (S_pO_2) and power output (P).

Manual, intermittent measurements:

Hydration, urine specific gravity (USG), blood pressure (BP), skin temperature (T_{skin}), skin blood flow (BF_{skin}), exercise, feeling, thermal discomfort, thermal sensation perceptions (perceptions), environment

Figure 5.2: Experimental design for 10-s sprints and 5-min cycling time trials

5.4.4 Data Analyses

5.4.4.1 Calculations

Power and lean mass were calculated for each athlete. Matlab software (Mathworks, Chatswood, Australia) was used to filter data from LabChart to remove missing and outlier values (outside 2 SD) and calculate average, peak and ranges for each data channel. All data were combined into Microsoft Excel® and partitioned into baseline rest, exercising CO_2 challenge, 10-s sprint and 5-min TT data blocks. For 10-s sprints, data were analysed as the change from the start to the end of the sprint in TEMP and HOT. For 5-min TT, data were grouped into three distinctive time blocks: 0:00-0:30 min, 1:00-4:00 min, 4:30-5:00 min for TEMP and HOT. The NIRS-derived brain and muscle haemodynamics, and MCAv data were determined by the change in micromolar concentration/velocity from baseline measures.

5.4.4.2 Statistics

Comparisons for power were compared for 10-s sprints between TEMP and HOT using paired student t-tests. Comparisons for power, physiological and psychophysical variables were compared for 5-min TT using two-way repeated measures ANOVA. Time effects were compared for each separate time block by Tukey's post-hoc analysis. Temperature effects were compared between TEMP and HOT environments by Holm-Sidak post-hoc analysis. Mean brain and leg HbO_2, HHb, tHb, TOI, and MCAv were correlated against power output at the Start, Middle and End of the 5-min TT by Linear Regression modelling. Type-1 errors were controlled using either Tukey or Sidak multiple comparison tests. Non-parametric data were log transformed and data that were not normalised by log transformation were analysed by Wilcoxon's signed rank test. Data are presented as mean (SD) with 95% CI, statistical significance accepted as $P < 0.05$. The data figures shown in Chapters 4-6 typically represent individual responses between conditions (on different days) rather than across time within a condition (i.e., within a day), because the condition comparison is usually most important for the research question(s).

5.5 Results

5.5.1 Cycling Power

5.5.1.1 10-s Sprints

Sprint power were an average of 11% [0.99 (0.52) W·kg^{-1} FFM] higher in HOT (Figure 5.3A).

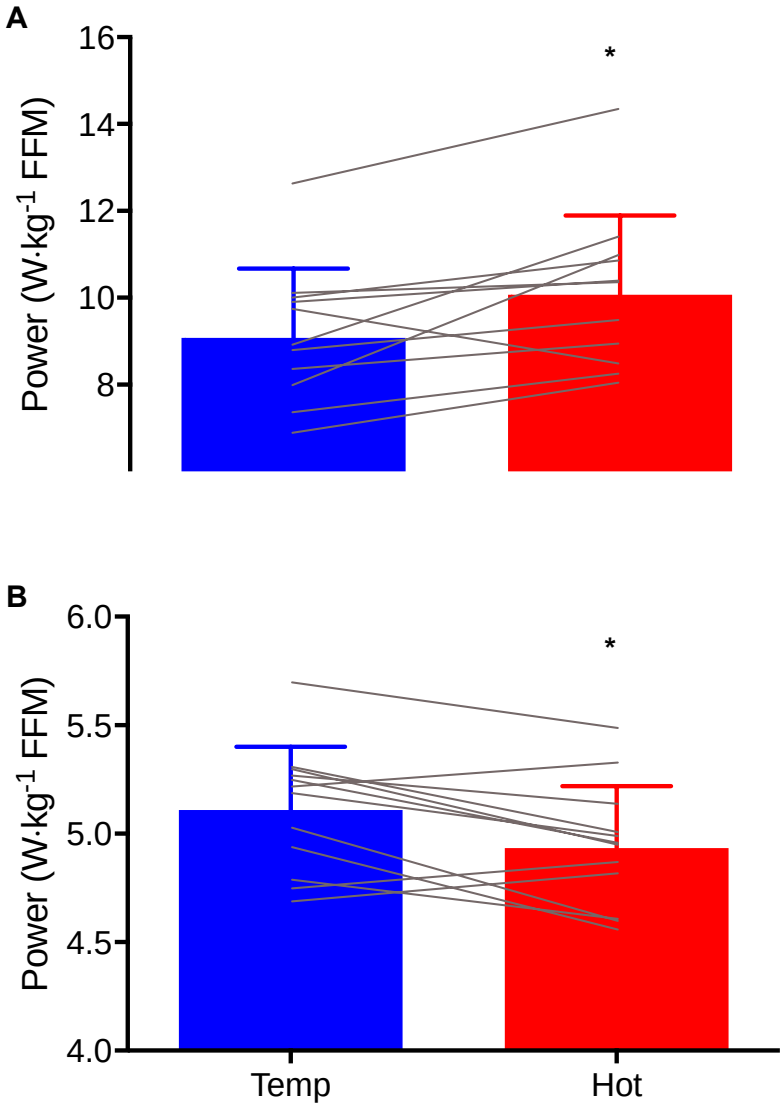


Figure 5.3: Power output for maximal effort 10-s seated sprint cycling (A) and 5-min time trial (B) in temperate (TEMP: 18 ˚C, 40% RH, 0.83 kPa PH_2O, 4.0 m·s^{-1} v) and hot (HOT: 33 ˚C, 60% RH, 3.02 kPa PH_2O, 4.0 m·s^{-1} v) environments. Grouped mean (+SD) in coloured blocks, individual participants in grey lines, * P < 0.05).

5.5.1.2 Five-Minute Time Trials

Averaged across the 5 min, mean power was 3.4% [0.17 (0.19) W·kg^{-1} FFM] lower in HOT (CI: 0.06 to 0.29, P = 0.007, Figure 5.3B). The pacing profile tended to differ between conditions (Interaction effect: P = 0.09), presumably reflecting a tendency to be lower in HOT at the Start and Middle but not at the End. Power also differed across the TT; being similar from the Start to the Middle (P > 0.99) then showing an end spurt effect by ~8% (i.e., by 0.46 W·kg^{-1} FFM, CI: 0.13 to 0.76, P = 0.005; Time effect: P = 0.002; Figure 5.4).

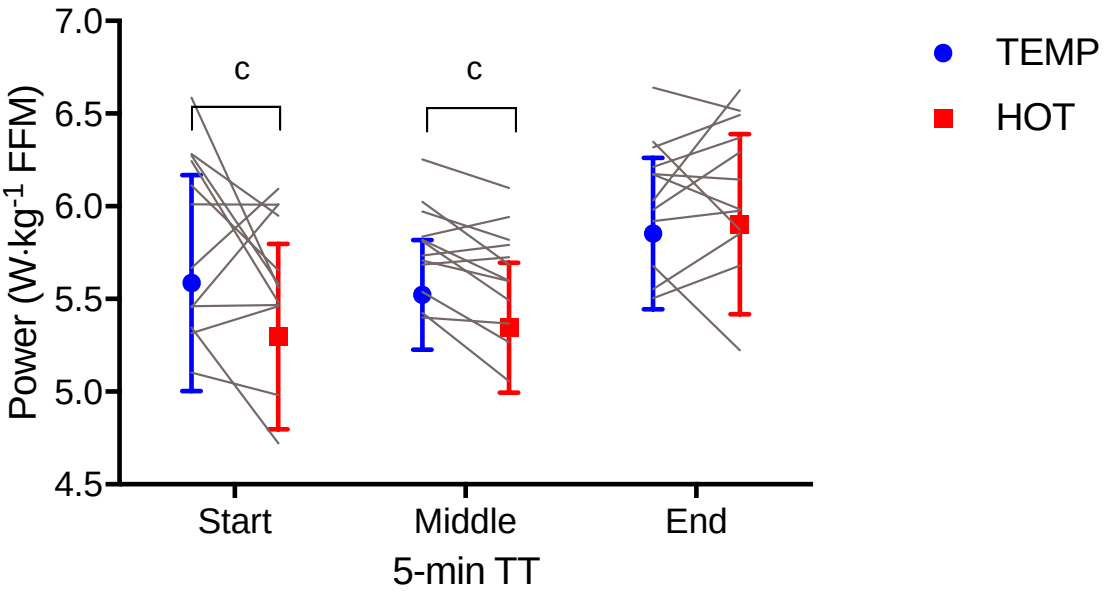


Figure 5.4: Power output for the Start (0:00-0:30), Middle (1:00-4:00), and End (4:30-5:00) periods of the 5-min time trial in TEMP and HOT. Data are displayed as grouped means with SD, and individual participants in grey lines. Time effect: P < 0.05 for Start and Middle compared with ^{c} End. For the data in this and most figures in chapters 5-7, the individual responses are between conditions (on different days) rather than across time within a condition, because the condition comparison is usually most important for the research question(s).

5.5.2 Body Temperature

5.5.2.1 Core Temperature

Core temperature (T_C) increased from Pre to Post in both environments, and showed a tendency to increase more in the HOT environment (Time effect: P < 0.001, Interaction effect: P = 0.16, Figure 5.5]. Specifically, T_C increased by 0.75 ˚C (0.55 to 0.94, P < 0.001) in TEMP and 0.91 ˚C (0.72 to 1.10, P < 0.001) in HOT, and exceeded 38.3 ˚C in two participants.

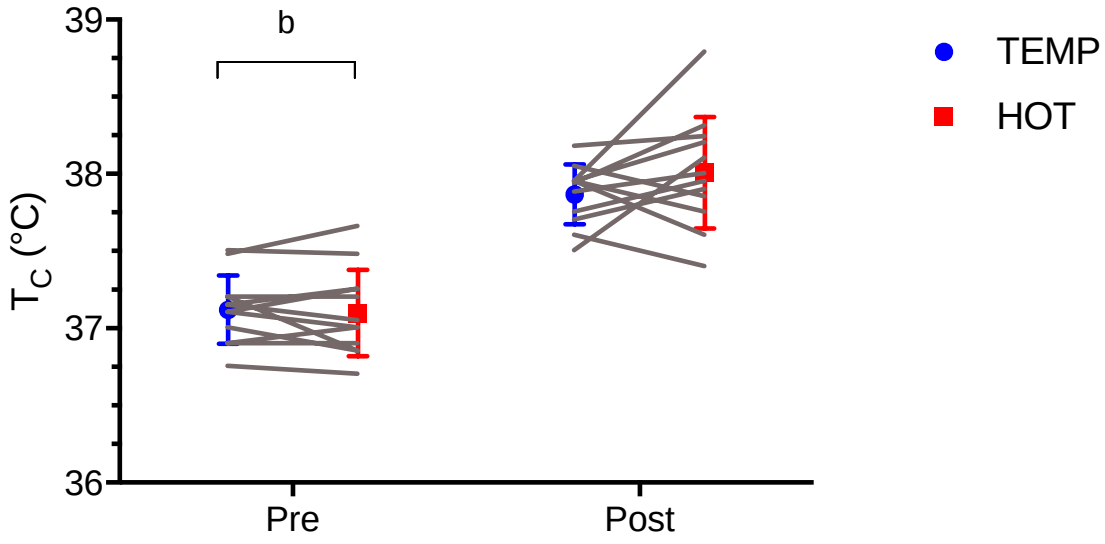


Figure 5.5: Core temperature changes (T_C) in TEMP and HOT environments from pre-test after 30 min rest in the environment (Pre) and immediately post-test (Post). Data are displayed as grouped means with SD, and individual participants in grey lines. Time effect: [b] P < 0.001 Pre compared with Post.

5.5.2.2 Skin Temperature

Forehead skin temperature (T_{Head}) was consistently lower in TEMP than HOT by ~4.6 (1.7) ˚C (Temperature effect: $P < 0.001$; Interaction effect: $P = 0.48$; Figure 5.6A). T_{Head} decreased by ~2.9 (1.7) ˚C across both exercise conditions (Time effect: $P = 0.02$).

Mid-axilla skin temperature (T_{Axilla}) decreased from Pre to Post in both environments, but by a greater amount in TEMP that was mediated by the larger T_{Arm} reduction across exercise in TEMP (Temperature effect: $P < 0.001$; Interaction effect: $P = 0.02$; Figure 5.6B). Specifically, T_{Axilla} was cooler in TEMP than HOT by 4.1 (1.3) ˚C at Pre and by 7.0 (2.7) ˚C at Post. T_{Axilla} decreased across exercise by 6.0 (2.5) ˚C in TEMP compared with 2.8 (1.7) ˚C in HOT environments (Time effect: $P < 0.001$).

Forearm skin temperature (T_{Arm}) decreased from Pre to Post in both environments, but by a greater amount in TEMP mediated by the larger T_{Arm} reduction across exercise in TEMP (Temperature effect: $P < 0.001$; Interaction effect: $P = 0.02$; Figure 5.6C). Specifically, T_{Arm} was cooler in TEMP than HOT by 6.7 (1.0) ˚C at Pre and 8.2 (2.4) ˚C at Post. T_{Arm} decreased across exercise by 4.3 (1.0) ˚C in TEMP compared with 2.7 (2.0) ˚C in HOT environments (Time effect: $P \leq 0.001$).

Finger skin temperature (T_{Finger}) was ~10 (3.3) ˚C lower in TEMP than HOT (Temperature effect: $P < 0.001$, Interaction effect: $P = 0.56$; Figure 5.6D). The T_{Finger} decreased ~2.5 (2.2) ˚C across exercise (Time effect: $P = 0.01$).

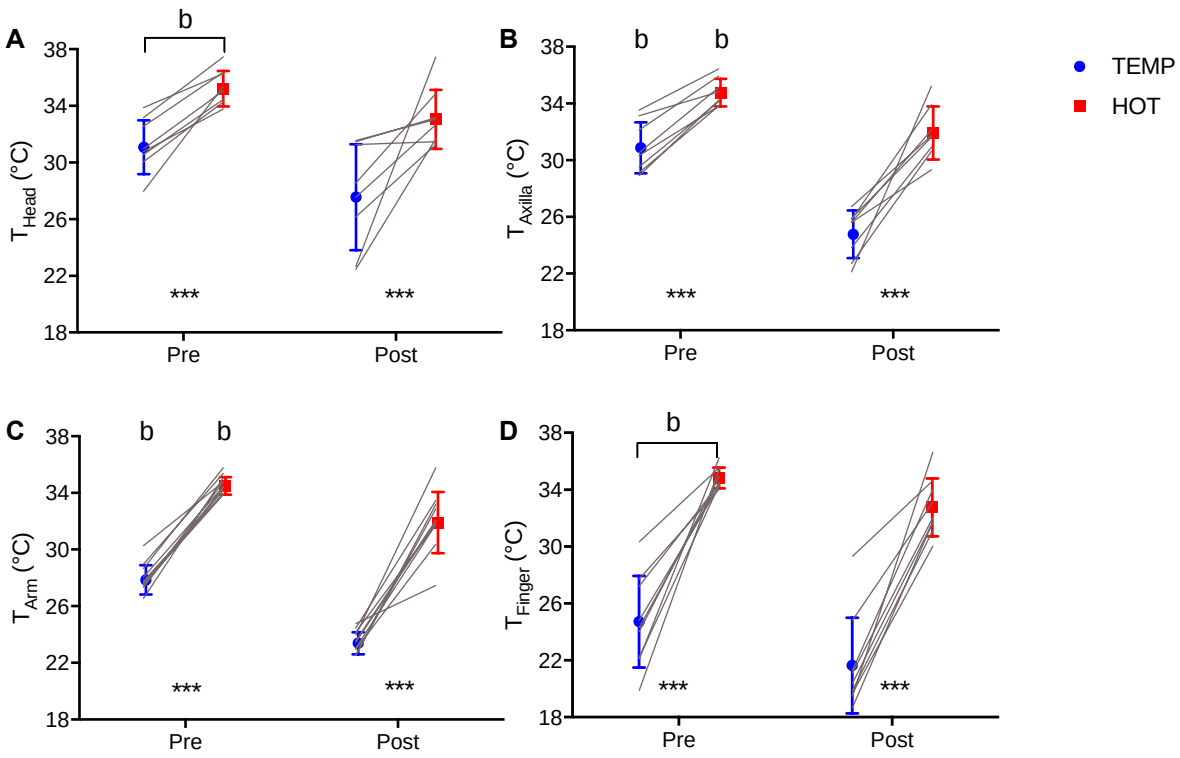


Figure 5.6: Skin temperature at the forehead (T_{Head}, A), mid-axilla (T_{Axilla}, B), forearm (T_{Arm}, C), and finger (T_{Finger}, D) in TEMP and HOT from pre-test resting in the environment for 30 min (Pre) and immediately post-test (Post). Data are displayed as grouped means with SD, and individual participants in grey lines. Time effect: $P < 0.05$ Pre compared with ^{b} Post; Temperature effect: *** $P < 0.001$.

5.5.3 Respiratory Responses

5.5.3.1 Minute Ventilation

Minute ventilation ($\dot{V}_E$) increased from the Start to the Middle by ~53 L·min^{-1} and Middle to the End by ~16 L·min^{-1} in both environments, (Time effect: P < 0.001; Interaction effect: P=0.52), and was similar between environments (Temperature effect: P = 0.51; Figure 5.7).

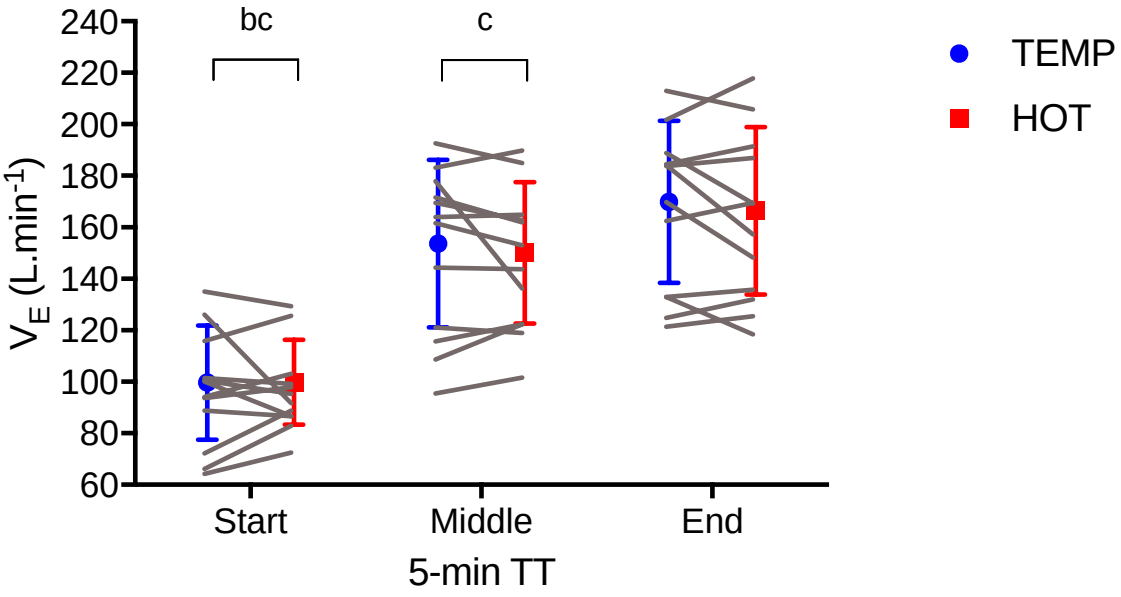


Figure 5.7: Ventilation ($\dot{V}_E$) for the Start (0:00-0:30), Middle (1:00-4:00) and End (4:30-5:00) periods of the time trial in TEMP and HOT. Data are displayed as grouped means with SD, and individual participants in grey lines. Time effect: P < 0.05 compared with [b] Middle and [c] End.

5.5.3.2 Breathing Frequency

Breathing frequency (f) increased from the Start to the Middle by ~12 breaths·min^{-1} and Middle to the End by ~10 breaths·min^{-1} in both environments (Time effect: P < 0.001; Interaction effect: 0.47), and was similar between environments (Temperature effect: P = 0.24; Figure 5.8).

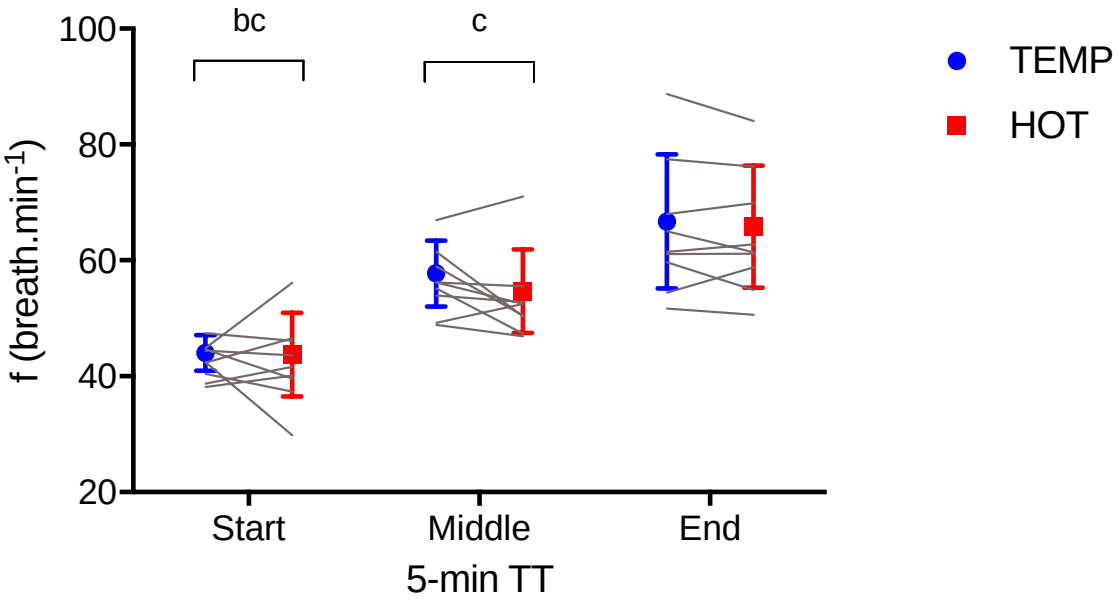


Figure 5.8: Breathing frequency (f) for the Start (0:00-0:30), Middle (1:00-4:00) and End (4:30-5:00) periods of the time trial in TEMP and HOT. Data are displayed as grouped means with SD, and individual participants in grey lines. Time effect: P < 0.05 compared with [b] Middle and [c] End.

5.5.3.3 Partial Pressure of End-Tidal Carbon Dioxide

The $P_{ET}CO_2$ was considerably lower at the End of 5-min TT, decreasing from the Start to the End and Middle to the End in both environments (by ~3 and ~2 mmHg respectively; Time effect: P < 0.001; Interaction effect: 0.26). There was a tendency for $P_{ET}CO_2$ to be lower in HOT (Temperature effect: P = 0.14; Figure 5.9).

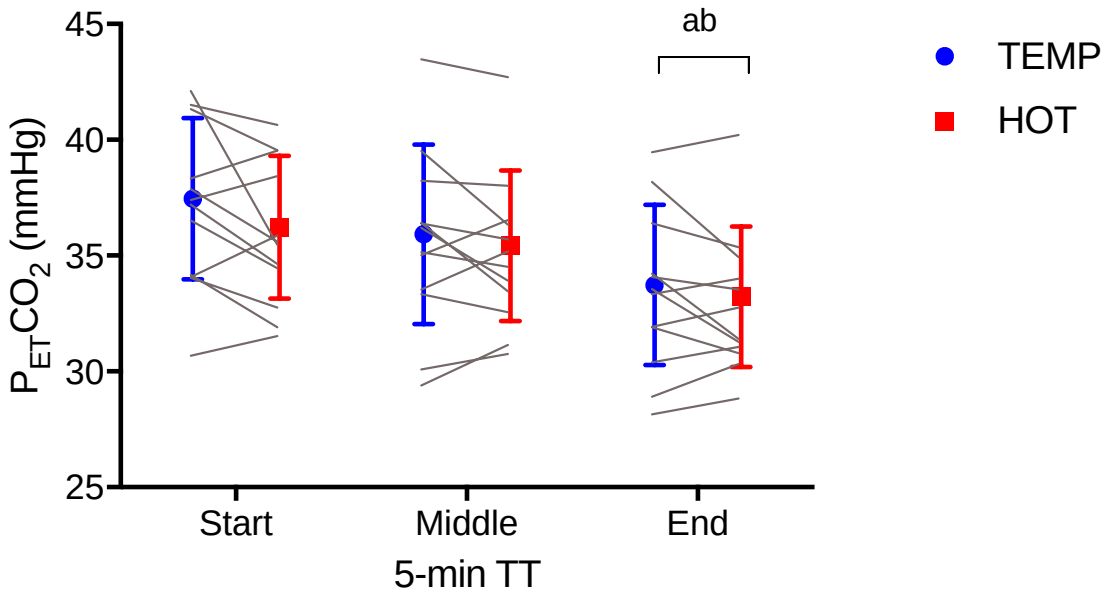


Figure 5.9: Partial pressure of end-tidal carbon dioxide ($P_{ET}CO_2$) for the Start (0:00-0:30), Middle (1:00-4:00) and End (4:30-5:00) periods of the time trial in TEMP and HOT. Data are displayed as means with SD, and individual participants in grey lines. Time effect: P < 0.05 at End compared with [a] Start and [b] Middle.

5.5.3.4 Exercising CO_2 Challenge

Refer to Results section 6.5.3.4

5.5.4 Cardiovascular Responses

5.5.4.1 Blood Pressure

Systolic blood pressure (SBP) increased from Pre to Post in both environments by ~39 mm Hg (Time effect: P = 0.001, Interaction effect: P = 0.83, Figure 5.10A], whereas DBP tended to decrease across exercise (Time effect: P = 0.11; Interaction effect: P = 0.28; Figure 5.10B). Both SBP and DBP were similar between environments (P > 0.70).

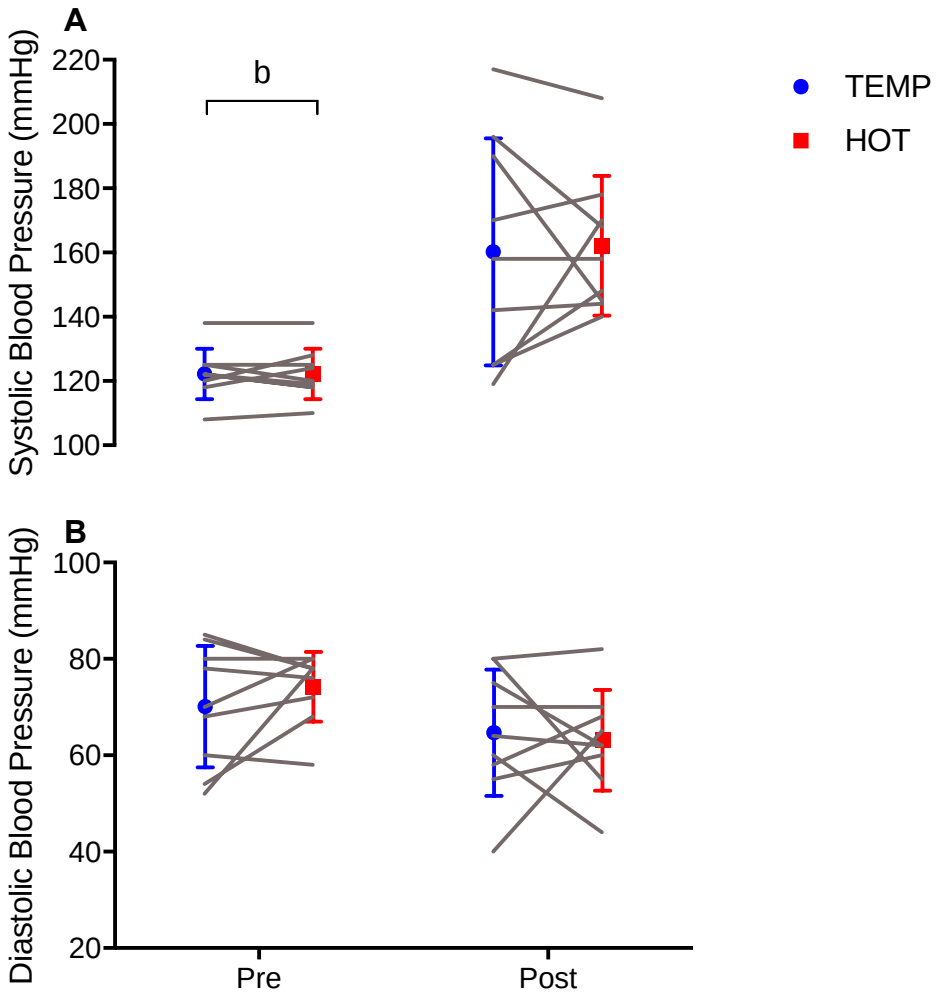


Figure 5.10: Systolic (A) and diastolic (B) blood pressure in TEMP and HOT from pre-test after resting 30 min in the environment (Pre) and immediately post-test (Post). Data are displayed as means with SD, and individual participants in grey lines. Time effect: P < 0.05 compared with [b] Post.

5.5.4.2 Heart Rate

Heart rate increased from the Start to the Middle (by ~18 beats·min^{-1}, CI: 14 to 22, P< 0.001) and Middle to the End (by ~4 beats·min^{-1}, CI: 1 to 8, P = 0.03) in both environments, but was similar between TEMP and HOT (Time effect: P < 0.001; Interaction effect: P = 0.17; Temperature effect P = 0.46; Figure 5.11).

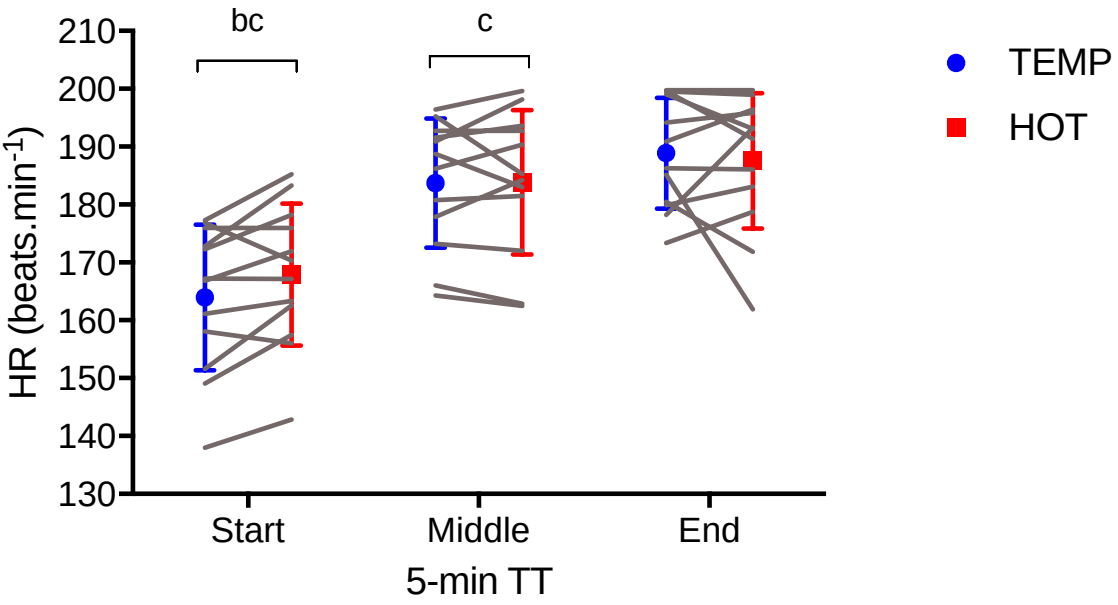


Figure 5.11: Heart rate (HR) for the start (0:00-0:30), middle (1:00-4:00) and end (4:30-5:00) periods of the time trial in TEMP and HOT. Data are displayed as means with SD, and individual participants in grey lines. Time effect: P < 0.05 compared with [b] Middle and [c] End.

5.5.4.3 Oxygen Pulse

Oxygen pulse ($\dot{V}O_2$ ÷ HR, O_2 pulse) increased from the Start to the Middle (by ~2.5 mL·beat^{-1}, CI: 1.3 to 3.7, P < 0.001) but was similar from the Middle to the End (P = 0.70) and between environments (Time effect: P < 0.001; Temperature effect: P = 0.21; Interaction effect: P = 0.84; Figure 5.12).

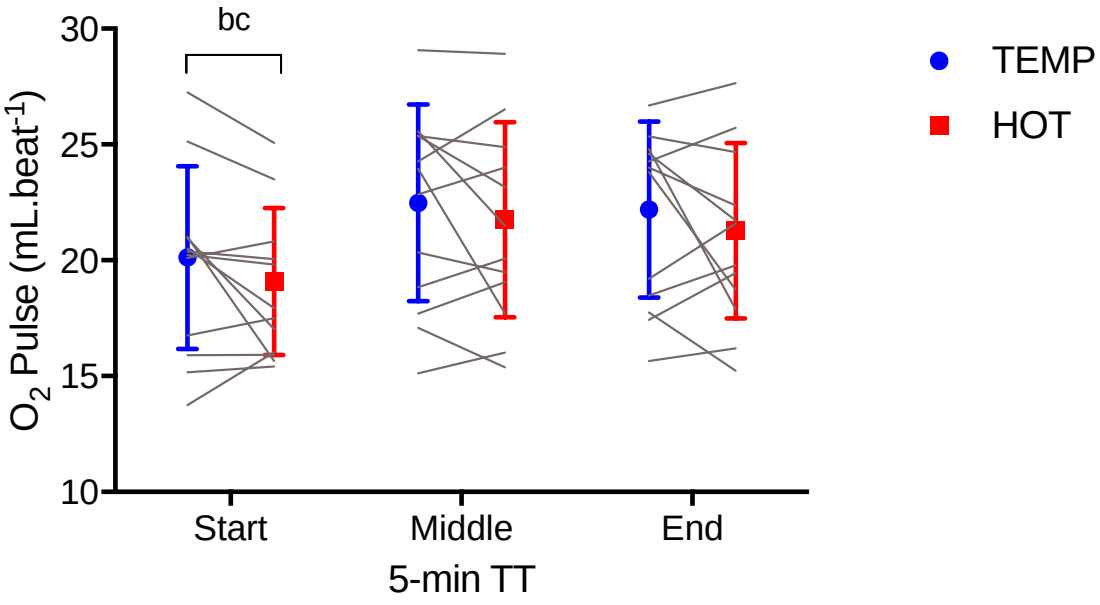


Figure 5.12: Oxygen pulse (O_2 Pulse) for the start (0:00-0:30), middle (1:00-4:00) and end (4:30-5:00) periods of the time trial in TEMP and HOT. Data are displayed as means with SD, and individual participants in grey lines. Time effect: P < 0.05 compared with [b] Middle and [c] End.

5.5.4.4 Skin Blood Flow

Forearm skin blood flow (BF_{Skin}) increased similarly across exercise in both environments by ~2.1 a.u ($P < 0.001$), but was consistently ~4.6 a.u. lower in TEMP than in HOT (Temperature effect: $P < 0.001$; Interaction effect: $P = 0.43$; Figure 5.13).

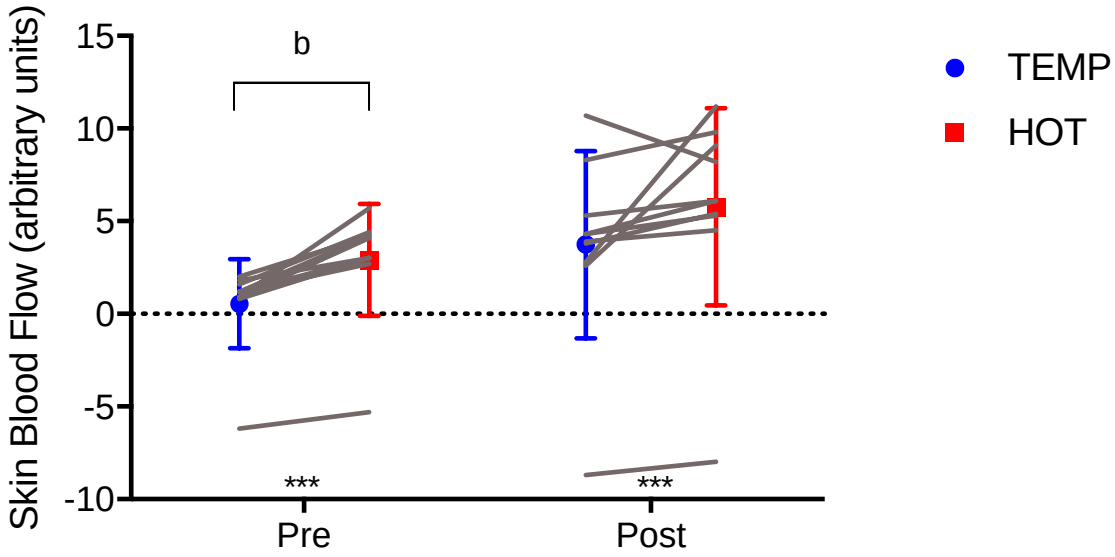


Figure 5.13: Skin blood flow at the forearm in TEMP and HOT from pre-test after 30 min resting in the environment (Pre) and immediately post-test (Post). Data are displayed as means with SD, and individual participants in grey lines. Time effect: $P < 0.05$ compared with [b] Post; Temperature effect: *** $P < 0.001$.

5.5.5 Brain (prefrontal cortex) Haemodynamics

The change in oxygenated haemoglobin (from rest) measured at the prefrontal cortex (Brain O_2Hb) showed a tendency to decrease in HOT (Temperature effect: $P = 0.06$; Figure 5.14A). Brain O_2Hb was similar across the 5-min TT for both environments (Time effect: $P=0.85$; Interaction effect: $P = 0.77$).

The change in brain deoxygenated haemoglobin from rest (Brain HHb) increased from the Start to the End in both environments, but by a greater amount in TEMP (Interaction effect: $P < 0.001$). Specifically, Brain HHb increased by 162 μM·cm (CI: 142 to 181, $P < 0.001$) in TEMP compared with 111 μM.cm (CI: 92 to 131, $P < 0.001$) in HOT, while Brain HHb increased in the HOT environment (compared

to TEMP) at the Start (by 61 μM·cm, P < 0.001) and Middle (by 43 μM·cm, P < 0.001) time points (Figure 5.14B).

The change in brain total haemoglobin from rest (Brain tHb) increased 118 μM·cm (CI: 35 to 200, p = 0.005) at the End of 5-min TT compared with the Start (Time effect: P < 0.006; Interaction effect: P = 0.20). Brain tHb tended to decrease in HOT compared with TEMP environments across all time points (Temperature effect: P = 0.13; Figure 5.14C).

Brain tissue oxygen index (Brain TOI) decreased similarly in both environments (Interaction effect: P = 0.78), but was lower (relative to rest) in HOT compared to TEMP (Temperature effect: P = 0.05). Specifically, Brain TOI decreased from the Start to the Middle (by 4%, CI: -6 to -1, P = 0.007) and from the Start to the End (by 6%, CI: -9 to -3, P < 0.001) of 5-min TT in both environments, but decreased 4% (CI: -6 to -1, P = 0.002), 3% (CI: -5 to -1, P = 0.01) and 3% (CI: -5 to -0.4, P = 0.02) in the HOT compared to TEMP environment at the Start, Middle and End time points, respectively (Figure 5.14D).

The change in brain blood flow velocity (from rest) measured at the middle cerebral artery (MCAv) increased at the End of 5-min TT compared with the Start (by 4 $cm \cdot s^{-1}$, CI: 0.05 to 9, P = 0.05) (Figure 5.14E) - equivalent to 4% of resting values, and this increase was similar between environments (Interaction effect: P = 0.83; Temperature effect: P = 0.71). In addition, there was a positive correlation between higher cycling power output and greater MCAv at the End in TEMP; i.e., 24 $cm \cdot s^{-1}$ for every 1 $W \cdot kg^{-1}$ FFM difference ($Y = 24x - 128$; R^2 = 0.64, CI: 0.38 to 0.95, P = 0.003; Figure 5.15A); however there was no relation between MCAv and power output in the HOT environment (P = 0.75; Figure 5.15B).

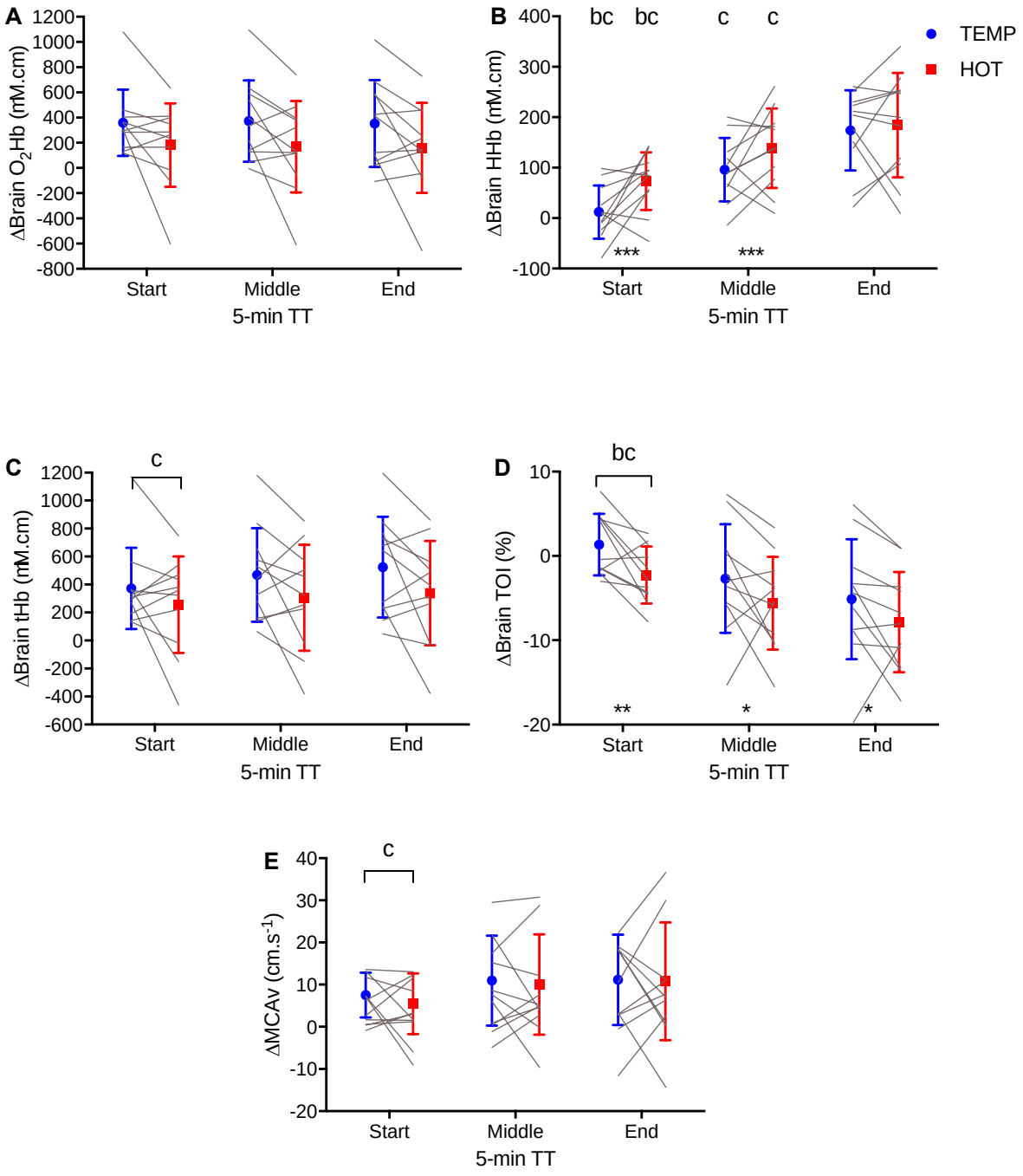


Figure 5.14: Change in oxygenated haemoglobin measured at the prefrontal cortex (A: Brain O_2Hb), deoxygenated haemoglobin (B: Brain HHb), total haemoglobin (C: Brain tHb), haemoglobin tissue oxygen index (D: Brain TOI), brain blood flow velocity measured at the middle cerebral artery (E: MCAv) for the Start (0:00-0:30), Middle (1:00-4:00) and End (4:30-5:00) periods of the time trial in TEMP and HOT. Data are displayed as means with SD, and individual participants in grey lines. Time effect: $P < 0.05$ compared with [b] Middle and [c] End; Temperature effect: ** $P < 0.01$, *** $P < 0.001$.

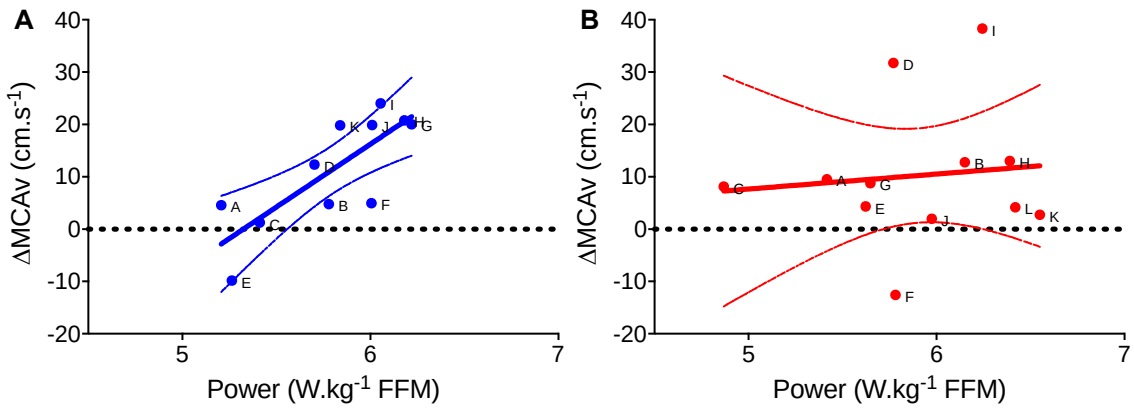


Figure 5.15: Average change in brain blood flow velocity at the middle cerebral artery (MCAv) compared with average power output in the last 30 s (End) of 5-min TT in TEMP (A) and HOT (B). Data are displayed as a linear regression (solid line) with 95% confidence interval (dotted line) and individual values in temperate (blue) and hot (red) environments.

5.5.6 Peripheral Haemodynamics

The change in oxygenated haemoglobin (from rest) measured at the left leg vastus lateralis muscle (Leg O_2Hb) was lower than resting levels throughout 5-min TT, but while this remained constant in the TEMP environment across each time point ($P > 0.26$), Leg O_2Hb decreased further in the HOT environment (Interaction effect: $P = 0.006$, Figure 5.16A). Leg O_2Hb decreased in HOT from the Start to End (by 82 μM.cm, CI: -141 to -24, $P = 0.006$) and tended to decrease in HOT from the Middle to the End of 5-min TT ($P = 0.08$). Further, while Leg O_2Hb was similar in both environments at the Start, it decreased in the HOT environment at the Middle (by 88 μM·cm, CI: -148 to -28, $P = 0.004$) and End (by 152 μM·cm, CI: -212 to -92, $P < 0.001$) time points compared to the TEMP environment.

The change in leg deoxygenated haemoglobin from rest (Leg HHb) increased from the Start to the Middle (by 61 μM.cm, CI: 16 to 106, $P = 0.007$) and increased from the Start to the End of 5-min TT (by 57 μM.cm, CI: 12 to 102, $P = 0.01$) in both environments (Time effect: $P = 0.004$; Interaction effect: $P = 0.43$). Leg HHb was lower in HOT compared with TEMP (Temperature effect: $P = 0.05$; Figure 5.16B).

The change in leg total haemoglobin from rest (Leg tHb) across the 5-min TT was different between the conditions (Interaction effect: $P < 0.001$). Specifically, in the TEMP condition, Leg tHb increased 93 μM.cm (CI: 58 to 129, $P < 0.001$) from the Start to the Middle and remained elevated at the End, whereas for the HOT condition Leg tHb was similar between Start and Middle and then decreased 40 μM.cm (CI: -74 to -4, $P = 0.03$) from the Middle to End. Further, Leg tHb was consistently lower in HOT compared to TEMP for all time points (Start: by -66 μM.cm; Middle: by -132 μM.cm; End by -193 μM.cm; all $P < 0.001$; Figure 5.16C).

Leg tissue oxygen index (Leg TOI) was lower than resting levels throughout the 5-min TT. Leg TOI showed a tendency in HOT to decrease from the Start to End of the 5-min TT, whereas it was relatively constant in the TEMP environment across all time points (Interaction effect: $P = 0.08$; Time effect: $P = 0.25$; Temperature effect: $P = 0.68$; Figure 5.16D).

Finger capillary oxygen saturation decreased from 97% to 94% across 5-min TT (Time effect: $P < 0.001$; Interaction effect: $P = 0.68$; Figure 5.16E). Specifically, SpO_2 decreased 1% from the Start to the Middle (CI: 0.08 to 2, $P = 0.03$) and a further 1% from the Middle to the End (CI: 0.2 to 2.0, $P = 0.02$). The SpO_2 tended to be lower in HOT compared with TEMP environments (Temperature effect: $P = 0.19$).

Blood lactate concentration increased from Pre to Post, with a greater increase in HOT compared with TEMP environments (Interaction effect: $P = 0.01$; Figure 5.16F). Specifically, [La] increased from Pre to Post in TEMP by 7.2 $mmol \cdot L^{-1}$ (CI: 5.7 to 8.7, $P < 0.001$) and in HOT by 9.7 $mmol \cdot L^{-1}$ (CI: 8.2 to 11.2, $P < 0.001$). The [La] was similar between environments at Pre ($P > 0.99$) but was 2.5 $mmol \cdot L^{-1}$ higher in HOT at Post (CI: 1.1 to 4.0, $P = 0.003$).

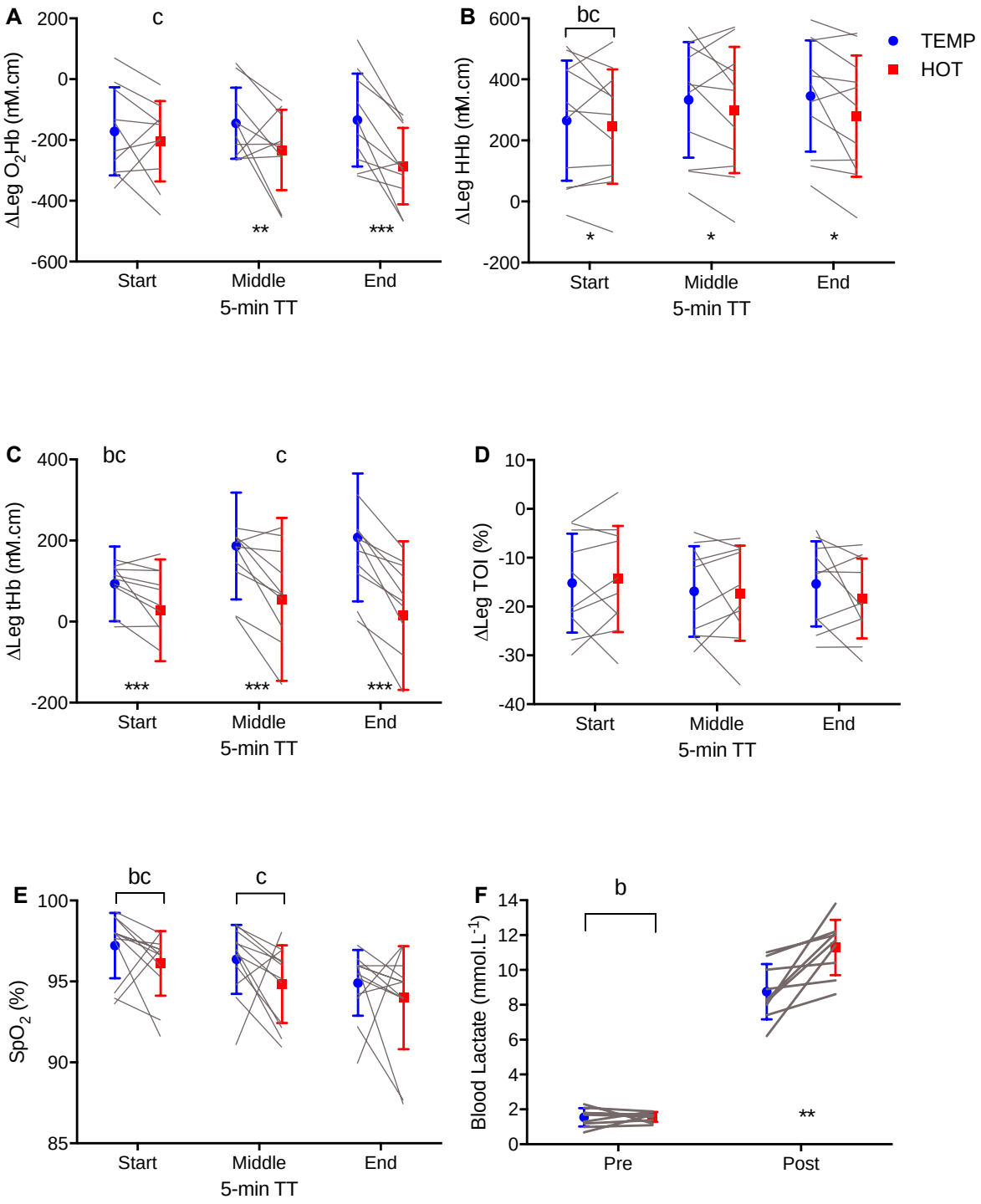


Figure 5.16: Change in oxygenated haemoglobin from resting values measured at the left leg vastus lateralis muscle (A: Leg O_2Hb), deoxygenated haemoglobin (B: Leg HHb), total haemoglobin (C: Leg tHb), haemoglobin tissue oxygen index (D: Leg TOI), percent finger capillary oxygen saturation (E: SpO_2) for the Start (0:00-0:30), Middle (1:00-4:00) and End (4:30-5:00), and blood lactate concentration change (**F**) measured Pre and Post the time trial in TEMP and HOT environments. Data are displayed as means with SD, and individual participants in grey lines. Time effect: $P < 0.05$ compared with [b] Middle, [c] End; Temperature effect: ** $P < 0.01$, *** $P < 0.001$.

5.5.7 Psycho-Physical Responses

Thermal discomfort increased from Pre to Post only in HOT (Interaction effect: P = 0.03; Figure 5.17A), whereas thermal sensation increased in both environments, but by a greater amount in TEMP (Time effect: P < 0.001; Interaction effect: P = 0.03; Figure 5.17B). RPE was higher at Pre in HOT and increased to the same rating at Post (Time effect: P < 0.001; Interaction effect: P = 0.004; Figure 5.17C).

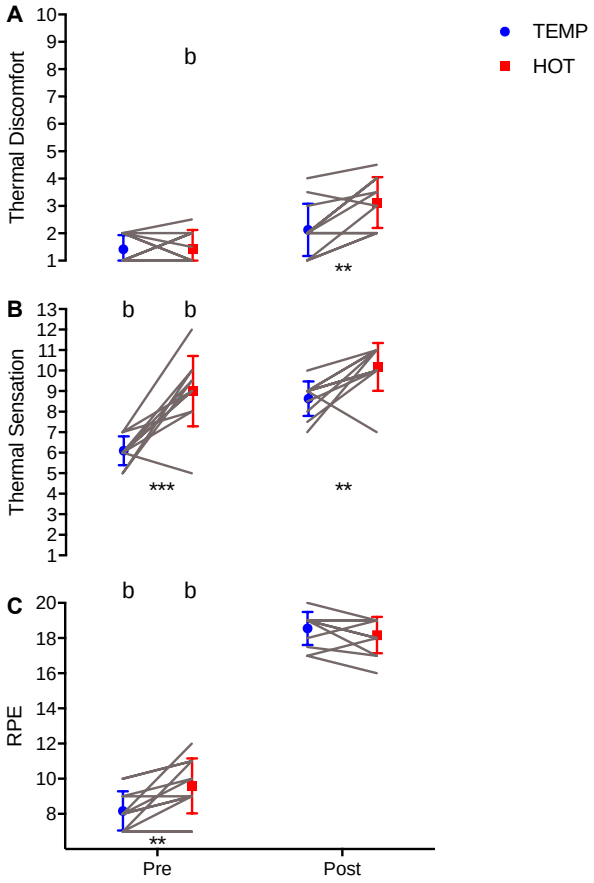


Figure 5.17: Thermal discomfort (A), thermal sensation (B), and perceived exertion (C) in temperate (TEMP) and hot (HOT) environments from warm-up (Pre) and at the end of the 5-min cycling time trial (Post). Thermal Discomfort scale: 1 = comfortable, 3 = slightly uncomfortable, 5 = uncomfortable, 7 = very uncomfortable, 9 = extremely uncomfortable; thermal sensation scale: 1 = Unbearably cold, 3 = very cold, 6 = slightly cool, 7 = neutral, 8 = slightly warm, 9 = warm, 10 = hot, 13 = unbearably hot. RPE scale: 9 = light, 11 = fairly light, 13 = somewhat hard, 15 = hard, 17 = very hard, 19 = very, very hard (Borg, 1982). Data are displayed as means with SD, and individual participants in grey lines. Time effect: P < 0.05 compared with [a] Pre, [b] Post; Temperature effect: ** P < 0.01, *** P < 0.001.

5.5.8 Fluid Loss

Fluid loss, approximated as gross loss of body mass from pre-trial to immediately post-trial was 2.4-fold greater in HOT (Figure 5.18).

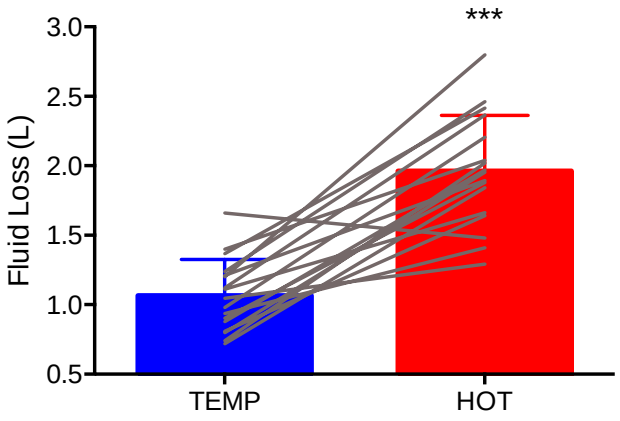


Figure 5.18: Fluid loss for each participant in TEMP and HOT compared between resting baseline and post-trial body weight accounting for fluid consumed and excreted. Data are displayed as mean + SD, with individual data overlaid in grey. Temperature effect: *** P < 0.001.

5.5.9 Blood Volume

Refer to Chapter 6.5.9 Results section.

5.6 Discussion

The aim of this chapter was to quantify performance power during race simulations in temperate and hot environments whilst measuring physiological responses, with particular focus on brain blood flow and oxygenation. The major findings were: 1) performance power was 11% higher in HOT for 10-s sprints; 2) average performance power was 3.4% lower in HOT for 5-min TT; 3) thermal strain was higher in HOT, as evidenced by the tendency toward higher (but not critically high) T_C and higher T_{Skin} and BF_{Skin} that may have reduced centrally available blood volume; 4) brain oxygen saturation at the prefrontal cortex decreased across 5-min TT and was consistently 3-4% lower in HOT while RPE and perceived temperature were higher at the outset, either or all of which may have influenced anticipatory pacing strategy and thermal perceptions.

5.6.1 Cycling Performance Power

Cycling power output for the 10-s sprints was 11% higher in HOT, corresponding with higher T_{Skin} (Figure 5.6) supporting the premise that elevated muscle temperature improved power production (Figure 5.3; Asmussen and Bøje, 1945; Ball et al., 1999; Faulkner et al., 2013; Kilduff et al., 2013). Average power output for the 5-min TT was 3.4% lower in HOT (Figure 5.3), with participants tending to produce lower power at the Start and Middle sections of the time trial (albeit not statistically significant for each phase); nevertheless, participants were able to produce more power at the End in both conditions (Figure 5.4). These findings indicate that participants were able to pace themselves effectively, saving sufficient physiological reserve to produce a powerful end-spurt (Corbett et al., 2012; Stone et al., 2011) regardless of the environmental conditions. It would seem that the HOT environment tended to influence a conservative pacing strategy (Schlader et al., 2011d, 2011c) and reduced mean performance power or work capacity in HOT with lower average power for 4-6 min TT or work trials (Figure 5.4; Altareki et al., 2008; Gonzalez-Alonso and Calbet, 2003). However, Leg TOI was lower in conjunction with higher [La] and RPE at Pre, which may indicate that despite reduced power output, glycolysis could have been higher at Pre. Similar metabolite accumulation and increased glycolysis were observed in

cyclists performing prolonged efforts in warm compared with temperate environments (Febbraio et al., 1994b) and in cyclists performing anaerobic efforts for two minutes with pre-warmed muscles (Febbraio et al., 1996).

Core temperature increased during the 5-min TT in both environments with two participants exceeding 38.3 °C (Figure 5.5). Fluid loss was higher in HOT, but did not exceed 2-3% total body weight loss that could significantly reduce performance power or negatively influence physiological responses (Cheuvront et al., 2010; González-Alonso et al., 1997; Nybo et al., 2001; Sawka et al., 1992; Trangmar et al., 2014a). Additionally, HR and SBP increased across the 5-min TT, but DBP was maintained in both environments (Figure 5.10, Figure 5.11). Nybo et al. (2001) reported that a T_C exceeding 38.5 °C hindered performance (5-8 min cycling at fixed power output) through altered metabolic and $\dot{V}O_2$ responses, however, the study required participants to wear water-perfused suits that would have restricted evaporative cooling and convective air flow, an experimental design that does not normally replicate the environment for competitive triathlon or cycling (Altareki et al., 2008; Saunders et al., 2005). Conversely, Altareki et al. (2008) demonstrated reduced average cycling power output concurrent with elevated T_C and T_{Skin} for a 4-km TT (~6:30) in 35 °C, 60% RH, ν_a: 5.6 $m \cdot s^{-1}$, and attributed the 1.9% performance time decrement to an anticipatory pacing strategy informed by thermal perceptions rather than failure of one or more physiological systems.

The participants in the present study produced greater average power in 5-min TT for TEMP compared with HOT. Superior average power in TEMP was facilitated in part by increased oxygen delivery to contracting skeletal muscle across 5-min TT, as indicated by the NIRS measurements (Figure 5.16A,C). Leg TOI was consistently ~15% lower than resting values across 5-min TT in TEMP and tended to decrease from -14 to -18% in HOT, indicating that the muscle was substantially desaturated from the Start in both environments and was maintained at the muscles physiological limit across the 5 min TT (Figure 5.16D). In HOT, however, reduced performance power was influenced by blood flow redistribution to the skin [4.6-fold greater at the forearm (Figure 5.13; Johnson,

1992; Johnson and Park, 1979; Rowell, 1974, 1977, 1986)]. Pre-test T_{Skin} measured from participants sitting quietly in the environment was substantially higher in HOT at every measurement site. One consequence of this was that heat storage would have been higher at the onset of exercise (Figure 5.6), which may have influenced power output during the time trial (Figure 5.4) – reinforcing the relationship between T_{Skin} and anticipatory pacing (Altareki et al., 2008; Schlader et al., 2011c). Though the heat load generated in HOT may have influenced the athlete's pacing strategy and performance it did not influence respiratory responses. Maximal exercise in both environments increased ventilation and breathing frequency steadily, while $P_{ET}CO_2$ decreased steadily as the 5-min TT progressed, indicating that respiratory responses were influenced by workload but less so by environment over time (Figure 5.7, Figure 5.8, Figure 5.9).

Pre-test T_{Skin} was >34 °C at every measurement site (Figure 5.6) and BF_{Skin} was substantially higher in HOT before exercise started (Figure 5.13), indicating that blood flow was redistributed away from contracting skeletal muscle to the skin (Gonzalez-Alonso and Calbet, 2003; Rowell, 1986; Sawka et al., 2011a). Additionally, Leg tHb [a measure that represents muscle blood volume (Subudhi et al., 2007)] was lower in HOT throughout the 5-min TT indicating that blood flow redistribution was substantial (Figure 5.15C). The convective cooling power provided by the fan (4 $m \cdot s^{-1}$) substantially reduced T_{Skin} in both environments (Figure 5.6).

The higher T_{Skin} in HOT limited the size of the thermal gradient between core to skin (TEMP: -13.6 °C, HOT: -5.6 °C) and skin to air (TEMP: -6.3 °C, HOT: 0.6 °C), which was already compromised by additional heat storage prior to starting the 5-min TT and exacerbated by the exercise task itself (Charkoudian, 2010; Cheuvront et al., 2010; Cuddy et al., 2014; Schlader et al., 2011c; Tucker et al., 2006b). However, participants maintained thermoregulation throughout 5-min TT despite higher T_{Skin} and BF_{Skin}, showing no significant difference in T_C between environments and only one participant reaching T_C > 38.5 °C. Conversely,

greater T_{Skin} may have reduced blood volume delivered to the muscle, inferred by lower total haemoglobin throughout 5-min TT (Figure 5.16C).

5.6.2 Brain (prefrontal cortex) Haemodynamics

Brain TOI desaturated by 6% across 5-min TT and was consistently lower in HOT. Such a large drop in Brain TOI may indicate that the brain was desaturating to the limit that athletes could maintain before syncope occurred, considering that brain desaturation of 5-7% has been found to coincide with orthostatic intolerance (Thomas et al., 2010). Further, syncope was observed in one athlete who fainted immediately after 5-min TT in TEMP. While the prefrontal cortex does not control the motor cortex it does influence fatigue, affective state, decision making, and planning (Gandevia, 2001; Gibson and Noakes, 2004; Krawczyk, 2002). The reduced oxygen saturation at the prefrontal cortex in HOT may have influenced athletes psycho-physical perceptions and anticipatory pacing before the 5-min TT started because thermal sensation and perceived exertion were higher pre-test (Figure 5.17B,C). Brain TOI started 4% lower in HOT, possibly because there was a greater proportion of deoxyhaemoglobin at the frontal cortex from the Start in HOT that consistently increased across 5-min TT (Figure 5.14B) despite a relatively stable oxyhaemoglobin and total haemoglobin at the frontal cortex (Figure 5.14A,C). At the Middle and End of 5-min TT the lower prefrontal cortex oxygen saturation most likely contributed to greater perceived thermal discomfort and sensation post-test (Figure 5.17A,B).

Participants blood flow through the middle cerebral artery throughout 5-min TT was stable in TEMP and increased 4% in HOT, which was not sufficient to maintain prefrontal cortex oxygen saturation, indicating that brain blood flow and brain oxygenation may not respond uniformly (e.g., decreased brain blood flow does not infer reduced prefrontal cortex oxygenation) during exhaustive exercise (Keiser et al., 2015b; Nybo et al., 2002a, 2011; Rasmussen et al., 2004). In the last 30 s of the 5-min TT in TEMP participants with higher MCAv produced more power (Figure 5.15A). This result, though speculative, may indicate that individuals with higher brain blood flow during exercise in a temperate environment are able to produce more power in a maximum voluntary effort and

implies that performance outcomes (i.e., the result decided by a sprint finish) could be related to brain blood flow. Importantly, brain blood flow responses may indicate how the environment or an individual's physiological responses to the environment influence subsequent performance capability. Support for this speculation may be that typically 1) brain blood flow and power decrease proportionally during high-intensity exercise when hyperthermia restricts blood distribution (Nybo and Nielsen, 2001b; Nybo et al., 2002a), 2) brain blood flow is higher in trained than untrained individuals (Ainslie et al., 2008; Murrell et al., 2013), and 3) decreased brain blood flow corresponds with lower P_aCO_2 (Nybo and Nielsen, 2001b; Nybo et al., 2002a; Secher et al., 2008), and in the present study the substantially lower $P_{ET}CO_2$ at the End of 5-min TT did not correspond with a change in brain blood flow (Figure 5.14E).

5.7 Conclusion

In HOT, sprint power was 11% higher whereas 5-min TT was 3.4% lower. The lower prefrontal cortex oxygen saturation and greater thermal discomfort may have encouraged a conservative pacing strategy. Yet, metabolic perturbations were still larger in HOT, possibly because cardiovascular strain was increased by the greater skin blood flow redistribution.

6 PERFORMANCE AND PHYSIOLOGY OF ELITE AND HIGHLY-TRAINED TRIATHLETES AND CYCLISTS DURING SIMULATED RACING IN TEMPERATE AND HOT ENVIRONMENTS

6.1 Chapter Introduction

This chapter documents the performance, physiological and psycho-physical strain experienced by unacclimated athletes to a simulated race with specific course and environmental protocols that matched the experiences of elite athletes in a race. Again, cycling was used because it was possible to accurately measure the physiological responses to race simulations. Twelve of the seventeen participants that performed the 10-s sprint and 5-min TT protocols participated in this study, completing those 5-min TTs in TEMP followed by HOT trial on successive days, then resting for 1 to 2 days before the 60-min SIM in TEMP then HOT environments reported in this chapter. The question central to this chapter was what profile of physiological strain appeared to influence endurance performance in these respective environments and when did they occur during a race simulation.

6.2 Abstract

New Zealand elite triathletes have performed poorly during international competitions when ambient temperatures exceeded 30°C. The aim of this study was to investigate endurance performance decrement in hot environments. Seventeen athletes (13 male and 4 female, 24 ± 7 years, 178.3 ± 9.5 cm, 60.9 ± 9.8 $mL \cdot kg^{-1} \cdot min^{-1}$ V̇O2 peak) performed a step-incremental cycling test to determine ventilatory thresholds, lactate thresholds, Wmax, and $\dot{V}O_2$ peak. Three days later each performed a 60-min SIM in an environmental chamber in TEMP [18.1 °C, 58% RH, 1.18 kPa PH_2O, 4.0 $m \cdot s^{-1}$ v_a), and two days later in HOT (33.1 °C, 62.1% RH, 3.10 kPa PH_2O 4.0 $m \cdot s^{-1}$ v_a). Exercise was a fixed intensity profile (FIXED) for 40 min [1:50 (min:s) at VT1 power (paced effort), then 0:10 at 200% Wmax (sprint) repeated 20 times] immediately followed by self-selected effort (FREE) for 20 min (1:50 paced effort then 0:10 maximum effort sprint, repeated 10 times), on an electromagnetically-braked ergometer. Data [mean (SD), 95% CI] from TEMP vs. HOT conditions were compared using two-way RM ANOVA and paired t-tests. Power during FREE paced efforts was 19-26% lower in HOT ($P < 0.001$), and the last FREE sprint was 15% lower in HOT ($P < 0.001$ vs. last sprint in TEMP). T_C increased more substantially and was greater in HOT ($P = 0.03$). T_{Skin} was 5-10 °C higher in HOT ($P < 0.001$). $P_{ET}CO_2$ across 60-min SIM decreased in both environments ($P < 0.001$) and was ~2 mmHg lower in HOT ($P < 0.05$). Cerebrovascular reactivity (CVR) was higher in HOT compared with TEMP during resting hypercapnia ($P = 0.01$). Brain TOI decreased 6% in HOT ($P < 0.001$), Leg TOI decreased 5% in HOT ($P = 0.005$). Participants felt more uncomfortable, hotter, and perceived they were exercising harder in HOT (all $P < 0.001$). Conclusion: Participants experienced greater physiological and psycho-physical strain during FIXED HOT that caused volitional exhaustion in nine participants. Athletes reduced self-paced effort and sprint power during FREE to a greater extent in HOT that may have been influenced by lower prefrontal cortex oxygen saturation and a deterioration in affective state.

6.3 Introduction

This chapter describes the performance and physiological responses to 60-min cycling in TEMP (18 °C, 40% RH, 1.18 kPa PH_2O, 4.0 $m \cdot s^{-1}$ v_a), and two days later to HOT (33 °C, 60% RH, 3.10 kPa PH_2O 4.0 $m \cdot s^{-1}$ v_a). The simulation was designed to mimic repeated short maximal efforts interspersed with aerobic-intensity paced efforts that punctuate a 40-km draft-legal Olympic distance triathlon in Wellington, New Zealand (TEMP) and Mooloolaba Australia (HOT). Prolonged exercise with stochastic changes in performance power in thermally neutral or stressful environments are common performance parameters for endurance athletes and have received considerable scholarly interest (Abbiss et al., 2010; Armstrong et al., 1985; Bergeron et al., 2012; Cheuvront et al., 2010; Ely et al., 2007; Mohr et al., 2012; Nassis et al., 2015; Nielsen, 1996). However, few studies have examined performance under thermally neutral or temperate environments and warm or hot environments (Kerr et al., 1998; Kreider et al., 1988a; Laursen et al., 2006; Mountjoy et al., 2012; Stevens et al., 2013). One study has collected performance *and* physiological data from a race (Bernard et al., 2009), and no published research to our knowledge has investigated thermal strain during an exercise protocol designed to simulate the cycle leg of triathlon racing. Considerable performance decrement has been observed when WBGT > 20 ˚C for marathon (Ely et al., 2007) and was demonstrated for triathlon in Chapter 3 of this thesis, yet little is known about the physiological mechanisms that underpin triathlon race performances.

Chapter 3 identified that while racing in a hot environment triathletes identified heavy fatigue and breathing problems that slowed or stopped them, which may be related to a combination of hyperthermia, hyperventilation, hypocapnia, hypotension and associated pre-syncopal symptoms. The physiological symptoms are multi-factorial involving inter-dependent central nervous and cardiovascular responses via increased T_C, T_{Brain}, T_{Skin} and energetic strain in the brain and skeletal muscle (Figure 6.1). The CNS may be inhibited by exercise greater than 30 minutes at 50-80% $\dot{V}O_2$ max in heat-stressful environments that increase T_C and T_{Brain} (T_{Brain} > T_C by 0.2-0.3 ˚C; Nybo et al., 2002a; Yablonskiy et al., 2000). Elevated T_{Brain} may reduce motor cortex activation that drives voluntary skeletal muscle contraction, causes peripheral fatigue, and inhibits motivation (Gonzalez-Alonso et al., 1999; Nielsen et al., 1993; Nybo and Nielsen,

2001a; Nybo and Secher, 2004b; Nybo et al., 2002b; Walters et al., 2000). Elevated T_{Skin} (and therefore muscle temperature) also contribute to decreased force output and increased central fatigue (Lloyd et al., 2015). The cardiovascular system may be maximally strained in hot and humid environments when exercise intensity was ≥ 80% $\dot{V}O_2$ max, an intensity that elite triathletes maintain for parts of the swim and bike, and all of the run section (unpublished observations). The high cardiovascular strain, T_C, and T_{Skin} increases BF_{Skin} while concurrently decreasing oxygen delivery to skeletal muscle, maximal oxygen extraction, and cardiac output ($\dot{Q}$; Gonzalez-Alonso and Calbet, 2003; González-Alonso et al., 1997; Gonzalez-Alonso et al., 2008; Hargreaves and Febbraio, 1998; Nybo, 2008; Nybo et al., 2001). Ventilation during exercise is driven higher by hyperthermia, causing hyperventilatory hypocapnia (Adolph and Fulton, 1924; Gaudio and Abramson, 1968; Martin et al., 1979; Nybo and Nielsen, 2001b; White, 2006). Such high ventilation has been associated with central fatigue and cerebral alterations (Morrison et al., 2004; Nielsen et al., 1993; Nybo and Nielsen, 2001a; Thomas et al., 2006; Todd et al., 2005). However, muscle metabolism and peripheral fatigue have not been shown to be adversely hindered (González-Alonso et al., 1998; Nielsen et al., 1990), presumably at least partly because of the experimental designs because at some point these must also constitute fatigue mediators if pacing is constrained appropriately.

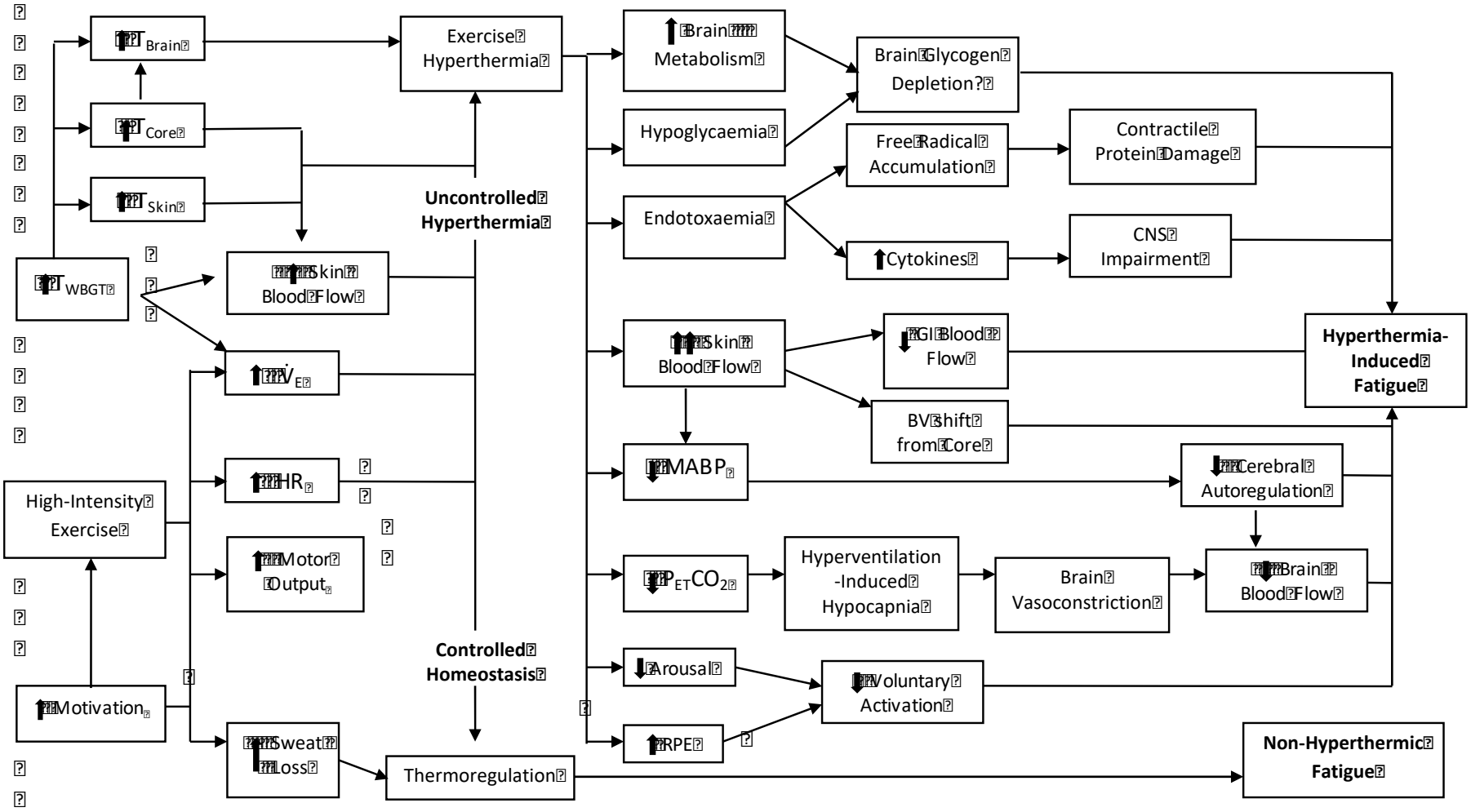


Figure 6.1: Mechanisms that contribute to thermoregulation and hyperthermia-induced fatigue (adapted from Cheung and Sleivert 2004).

Hyperthermia, hyperventilation, hypocapnia, and syncope also have a cumulative adverse effect on brain oxygenation and blood flow. Hypocapnia (lower [$PaCO_2$]) causes the cerebral arteries to vasoconstrict and reduce brain blood flow that normally washes out [H^+], [CO_2], and heat from the brain. Chemoreceptors in the brain stem detect increased metabolite concentration, triggering increased ventilation that normally removes metabolites effectively. However, when hyperthermic, the multiple signals from the exercise itself, psychological motivation, and thermoreception act to exacerbate hyperthermia, hyperventilation, hypocapnia, and syncope (Figure 6.1; Ide and Secher, 2000; Jorgensen et al., 2000). Hyperthermia may lower brain oxygenation via decreased brain blood flow and increase its oxygen requirement (~7%: Nybo et al., 2002a; 43%: Rasmussen et al., 2010a). But normal motor function is retained until brain oxygenation decreases to the physiological threshold that may cause syncope (Lieshout et al., 2003; Roberts, 1989) and presumably reduced motor output. Furthermore, dehydration reduces the available $\dot{Q}$ required to maintain cerebral perfusion pressure for a hyperthermic individual (Cheuvront et al., 2010; Gonzalez-Alonso, 1998, 1998; González-Alonso et al., 1997; Montain and Coyle, 1992). The factors that may compromise $\dot{Q}$ include: BF_{Skin} to maintain thermoregulation (Brengelmann et al., 1977; Johnson and Park, 1979; Rowell, 1977b, 1977a), ventilatory muscle to maintain $\dot{V}_E$ (Harms et al., 1998b; Romer and Polkey, 2008), and locomotor-related skeletal muscle to maintain performance (Figure 6.1; Gonzalez-Alonso, 1998).

Laboratory studies have indicated that individuals reach exhaustion at a critical T_C (Gonzalez-Alonso et al., 1999; Nielsen et al., 1993). However, highly variable T_C at the end of field studies and race simulations have been reported (Nybo and González-Alonso, 2015; Schlader et al., 2011a). Furthermore, runners have achieved T_C > 40°C during competitive races (Byrne et al., 2006; Maron et al., 1977; Robinson et al., 1945). The core temperatures and performance outcomes observed in the field or self-paced protocols indicate individuals may use anticipatory pacing (Abbiss et al., 2010; Tatterson et al., 2000; Tucker, 2009; Tucker et al., 2004, 2006b), potentially determined by a central governor (Noakes et al., 2001) or brain integration (Hargreaves, 2008). Pacing strategy may also be influenced by skeletal muscle that regulates blood flow, respiration, O_2

delivery, and pacing strategy (Amann, 2012; Amann et al., 2011). Such factors may (Amann, 2011) or may not (Nybo et al., 2011) influence central fatigue. Skeletal muscle blood flow and oxygen delivery were not limited by hyperthermia during submaximal exercise (Febbraio et al., 1994b; Nybo, 2008; Nybo and Nielsen, 2001a), or brief maximal skeletal muscle contractions (Nielsen et al., 1993), but were lower for sustained isometric muscle contractions (Nybo and Nielsen, 2001a; Nybo et al., 2001).

The present study was undertaken to provide insight into the extent and profile of such physiological strain for highly trained athletes during a more realistic race simulation under ambient heat stress. Specifically, the purpose and contribution of this study was to utilise the stochastic exercise profile that characterises performance demands from competing in an elite-level, draft legal 40-km cycle leg of a triathlon matched with the environmental stress imposed by racing in New Zealand and Australian climates. The central and peripheral physiological variables were measured over time in different environments to identify which factors influence performance and the time course over which they occur. The hypothesis was that a 60-min triathlon cycle race simulation that replicated the performance work profile and thermal environment race factors would elicit more strain on cerebrovascular, cardiovascular, and psychophysical factors in the heat, which would then confer impaired tolerance and performance.

6.4 Method

6.4.1 Participants

Thirteen males and four females (24 ± 7 years, 178.3 ± 9.5 cm, 70.6 ± 10.9 kg, 60.9 ± 9.8 mL.kg^{-1}·min^{-1} $\dot{V}O_2$ peak) gave informed consent to participate.

6.4.2 Experimental Design

This study consisted of a lactate test and blood volume assessment, then three days later a rest day before the 60-min SIM in TEMP (18.2 ± 0.4 ˚C, 55.6 ± 10.3% RH, 0.83 kPa PH_2O, 4.0 m·s^{-1} v_a), and another rest day before the 60-min SIM in HOT (33.0 ± 0.2 ˚C, 62.3 ± 2.2% RH, 3.02 kPa PH_2O, 4.0 m·s^{-1} v_a) then a final blood volume assessment on day nine (Figure 6.2).

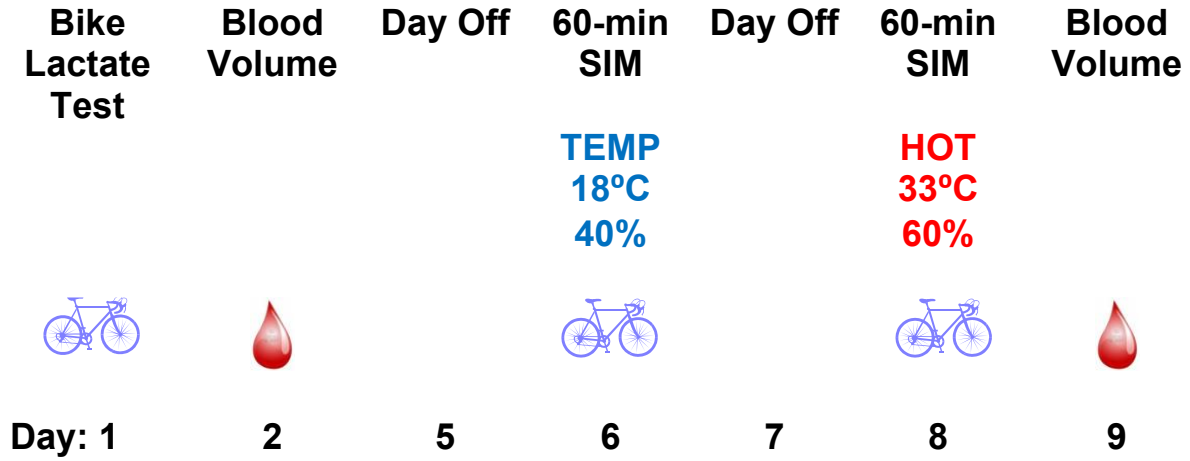


Figure 6.2: Timeline for physiological and performance tests for 60-min SIM in TEMP and HOT environments. Note that days 3-4 excluded from this figure were reported in Chapter 5.

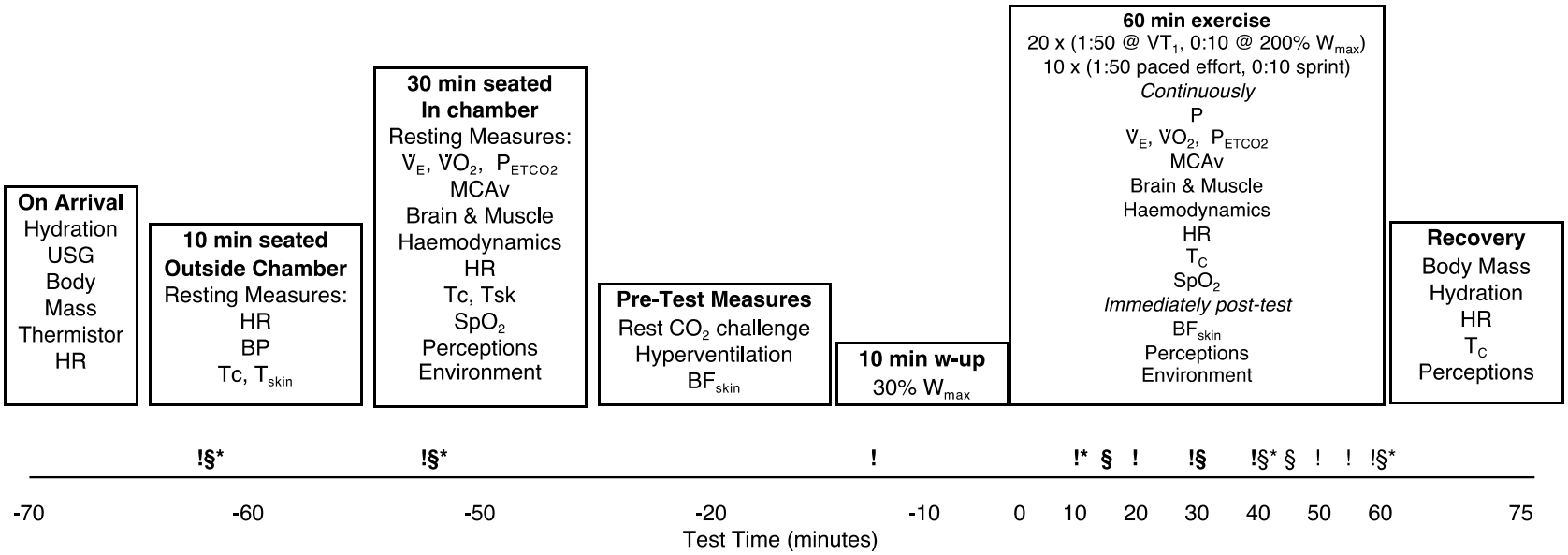
6.4.3 Procedures

6.4.3.1 Pre-Test Procedures

Participants completed the preliminary procedures followed by venous occlusion plethysmography and resting CO_2 challenge pre-test procedures.

6.4.3.2 Test Procedures

Athletes were instructed to treat the trial with the same preparation and intensity as a race. A fan was placed in front of the ergometer to direct 4.0 $m.s^{-1}$ airflow onto participants. The 60-min SIM protocol was split into two distinct phases; a 40-min prescribed phase and then a 20-min self-paced phase. First, participants completed 1:50 (min:s) at paced effort (VT1) and 10 s at 200% W_{max} (both calculated from the cycle step test) repeated 20 times continuously for a total of 40 min (FIXED). A 60-s break to change the cycling protocol was immediately followed by 1:50 at self-selected intensity and 10 s maximal effort repeated 10 times continuously for 20 min (FREE). During FREE, participants were strongly encouraged to maintain the same intensity during paced efforts and exceed 200% W_{max} in sprint efforts. Power, HR, respiratory variables, NIRS-derived brain and muscle haemodynamics, and brain blood flow velocity were recorded continuously (i.e., beat-to-beat/breath-by-breath). Core temperature was recorded every 30 s. Every five minutes manual records were taken for HR, T_C, and SpO_2. Every 10 min T_{Skin} and psychophysical perceptions were recorded. Blood pressure was measured during FIXED at 10 min and 40 min (or the conclusion of the FIXED protocol), and then again immediately after FREE (at 60 min or volitional exhaustion) (Figure 6.3). Finally, participants completed the normal post-test procedures.



Instrumentation

Thermistor (rectal core temperature), HR Strap (heart rate), near infra-red spectroscopy (brain & muscle haemodynamics), transcranial Doppler ultrasound (MCAv)

Continuous measurements

Core temperature (Tc), heart rate (HR), minute ventilation ($\dot{V}_E$), oxygen consumption ($\dot{V}O_2$), partial pressure end-tidal CO_2 ($P_{ET}CO_2$), brain blood flow velocity (MCAv), brain and muscle oxyhaemoglobin content, deoxyhaemoglobin content, tissue oxygenation index (brain & muscle heamodynamics), arterial O_2 saturation of Hb (S_pO_2) and power output (P).

Manual, intermittent measurements:

Hydration, urine specific gravity (USG), blood pressure (BP), skin temperature (T_{skin}), skin blood flow (BF_{skin}), exercise, feeling, thermal discomfort, thermal sensation perceptions (perceptions), environment

! Exercise and thermal perceptions, T_{skin}

§ Blood lactate concentration, environment

* Arterial blood pressure

Figure 6.3: Experimental design for the 60-min triathlon race simulation

6.4.4 Data Analyses

6.4.4.1 Calculations

Power and lean mass were calculated for each athlete. Matlab software (Mathworks, Chatswood, Australia) was used to filter data from LabChart to remove missing and outlier values (outside 2 SD) and calculate average, peak and ranges for each data channel. All data were combined into Microsoft Excel® and partitioned into baseline rest, resting CO_2 challenge, and race simulation data blocks. The race simulation data were partitioned further into 1:50 paced efforts and 10 s sprints for TEMP or HOT environments (60 blocks for each participant across the FIXED and FREE phases). The brain and muscle haemodynamics, and brain blood flow velocity data were determined by the change in micromolar concentration/velocity from baseline measures.

6.4.4.2 Statistical Analyses

Comparisons for power, physiological and psychophysical variables for each block were calculated using two-way RM ANOVA and post-hoc comparisons were made using Tukey or Holm-Sidak analyses. Comparisons were reported as 95% confidence intervals (CI) with the associated P value. Time effects were compared separately for paced efforts and sprints at specific time points for fixed-pace and free-pace sections. For FIXED: resting baseline (Rest), two minutes (First), ten minutes (Mid) and the last fixed block completed (Last) were compared. For FREE: the last fixed-pace effort ($FIXED_{Last}$) was compared with the first two minutes (First), the middle block that was usually, but not always, at 10 minutes (Mid), the penultimate block (Penult), and the last block (Last) in FREE. Note that blocks in the FREE section were based on how much the participant performed (e.g., a participant that completed 12 min of FREE would be analysed as Mid: 6 min, Penult: 10 min, Last: 12 min) instead of set time points. Temperature effects were compared between TEMP and HOT environments separately for paced efforts and sprints at the same time points using paired t-tests. Type-1 errors were controlled using Tukey post-hoc pairwise comparisons. Non-parametric data were log transformed, and data not normalised by log transformation were analysed by Wilcoxon's signed rank test.

6.5 Results

6.5.1 Cycling Race Simulation Power

By design, the work intensities used in FIXED were at a level that were individualised and difficult to complete at least in HOT before switching to FREE. All participants completed the TEMP trials, however, 9 out of 17 participants were unable to complete the full 40 minutes of the FIXED section of the HOT trial and 2 participants did not complete 60-min SIM because T_C reached 40.0ºC (Figure 6.4).

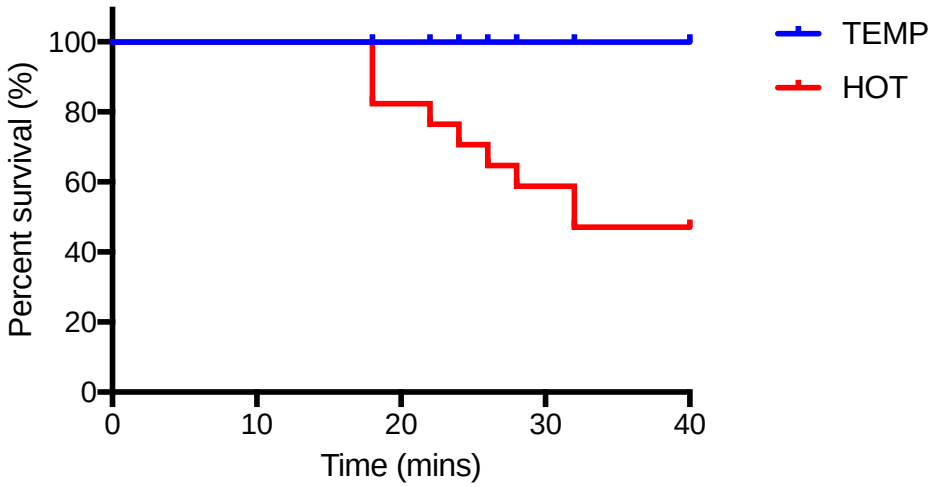


Figure 6.4: Survival analysis during the first 40 min at FIXED power outputs. In HOT nine participants changed to the FREE (self-paced) protocols at time points earlier than 40 minutes. All participants then completed the 20-min FREE protocol.

Paced Efforts: Paced-effort cycling power was consistent during FIXED and similar in TEMP and HOT (Interaction effect P = 0.35, Time effect: P = 0.38, Temperature effect: P = 0.44), while during FREE power was similar in TEMP (compared to FIXED) whereas it decreased in HOT (Interaction effect: P = 0.001; Figure 6.5). Specifically, paced-effort power was similar in TEMP for $FIXED_{Last}$ compared with all FREE time points (P > 0.11), but decreased by 24-32% in HOT from $FIXED_{Last}$ to all FREE time points (by 0.85 to 1.14 W·kg FFM; all P < 0.001). Paced-effort power was 19-26% lower in HOT than TEMP at all FREE time points (by 0.84 to 0.62 W·kg FFM; all P < 0.001).

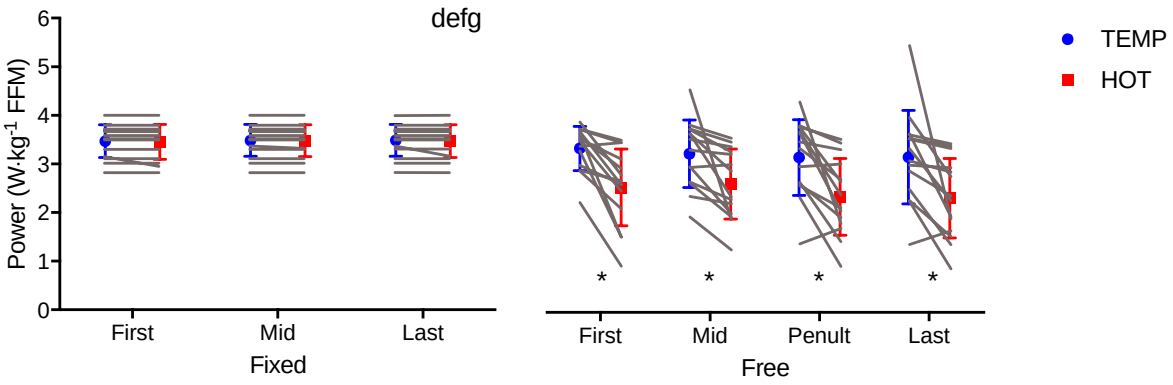


Figure 6.5: Power outputs for paced efforts at the start (First), middle (Mid), penultimate (Penult), and final (Last) 1:50 every 2:00 for FIXED (pre-set power) and FREE (self-selected power) during the 60-min race simulation. Mean ± SD in coloured symbols, individual responses in grey lines. Time effect: P < 0.05 at: [d] $FREE_{First}$, [e] $FREE_{Mid}$, [f] $FREE_{Penult}$, [g] $FREE_{Last}$; Temperature effect: *P < 0.05.

Sprints: Sprint power during FIXED was similar in TEMP and HOT (Interaction effect P = 0.83; Time effect: P = 0.38; Temperature effect: P = 0.44; Figure 6.6). During FREE, power increased in TEMP (compared to $FIXED_{Last}$) whereas it remained similar in HOT such that sprint power in HOT was consistently lower than TEMP during the FREE phase (Interaction effect: P = 0.21; Time effect: P= 0.009; Temperature effect: P < 0.001; Figure 6.6). Specifically, sprint power increased 25% from $FIXED_{Last}$ to $FREE_{Last}$ (by 1.84 W·kg FFM; CI: 0.48 to 3.21, P = 0.003). Sprint power was 15% lower at $FREE_{Last}$ (by 1.49 W·kg FFM; CI: -2.73 to -0.25, P = 0.01) in HOT compared to TEMP.

In summary, during the FREE phase (compared with FIXED) paced effort was maintained and sprint power was greater in TEMP, but paced effort was reduced and sprint power was maintained in HOT.

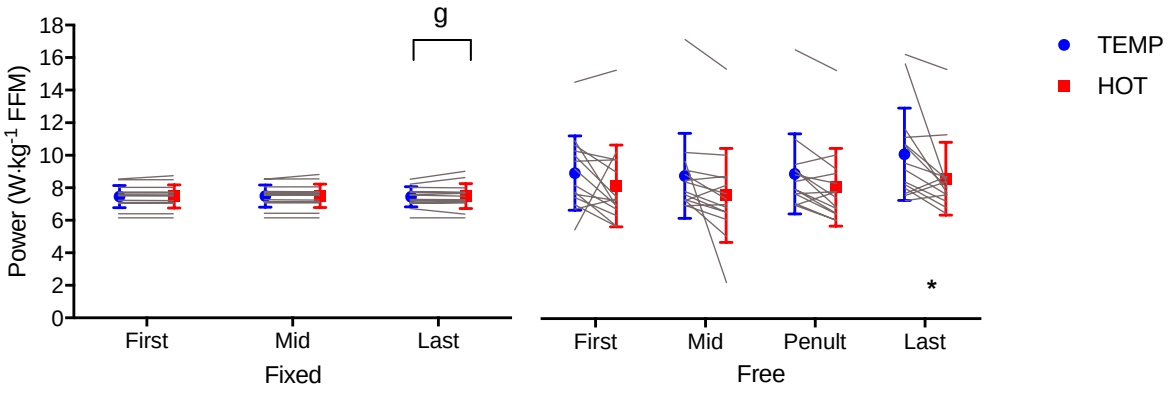


Figure 6.6: Power outputs for sprints at the start (First), middle (Mid), penultimate (Penult), and final (Last) last 0:10 every 2:00 for FIXED (pre-set power) and FREE (self-selected power) during cycling simulation. Mean ± SD in coloured symbols, individual responses in grey lines. Time effect: P < 0.05 at: $^{g}FREE_{Last}$; Temperature effect: *P < 0.05.

6.5.2 Body Temperature

6.5.2.1 Core Temperature

Core temperature (T_C) tended to be higher in HOT at Rest (P = 0.11) compared to TEMP. Tc increased during FIXED in both TEMP and HOT, but by a greater amount in HOT (Interaction effect: P = 0.03; Figure 6.7). Specifically, T_C in TEMP increased by 1.50 ˚C (CI: 1.34 to 1.67, P < 0.001) from Rest to $FIXED_{Last}$, while T_C in HOT increased by 1.71 ˚C (CI: 1.54 to 1.88, P < 0.001) from Rest to $FIXED_{Last}$. In addition, Tc was between 0.2 and 0.35 ˚C higher in HOT at all FIXED time points compared to TEMP (P ≤ 0.005). During the FREE phase, Tc remained similar to $FIXED_{Last}$ during TEMP (P > 0.50), but increased further in HOT environments (e.g., by 0.29 ˚C from $FIXED_{Last}$ to $FREE_{Penult;}$ P < 0.001; Figure 6.7). T_C remained consistently higher in HOT at all FREE time points relative to TEMP (all P < 0.001).

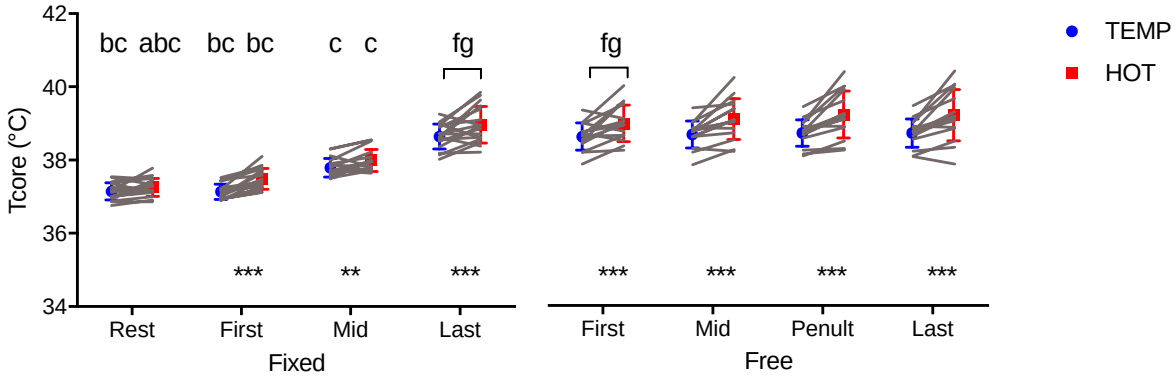


Figure 6.7: Core temperature (rectal) for paced efforts at rest (Rest), and then the first, middle and end for FIXED followed by the first, middle, penultimate and last for FREE during cycling simulation. Mean ± SD in coloured symbols, individual responses in grey lines. Time effect: P < 0.05 at: [a] $FIXED_{First}$, [b] $FIXED_{Mid}$, [c] $FIXED_{Last}$, [f] $FREE_{Penult}$, [g] $FREE_{Last}$; Temperature effect: *P < 0.05.

6.5.2.2 Skin Temperature

Forehead (T_{Head}), mid-axilla (T_{Axilla}), forearm (T_{Arm}) , and finger (T_{Finger}) skin temperatures decreased from pre-test to exercise, and by a greater amount in TEMP (Interaction effect: all $P < 0.001$; Figure 6.8A-D). In TEMP T_{Head} decreased from pre-test in the range of 5.1 - 6.9 ˚C across 60-min SIM (all $P < 0.001$), whereas in HOT it decreased by only 2.5 - 2.9 ˚C ($P < 0.001$). The T_{Head} was higher in HOT at pre-test by 4.2 ˚C, and this difference was increased further at 60 min (to 8.2 ˚C, both $P < 0.001$).

The T_{Axilla} decreased by 5.6 - 7.4 ˚C across TEMP ($P < 0.001$), and by 2.4 - 2.6 ˚C across HOT ($P < 0.001$; Interaction effect: $P < 0.001$). Thus, while T_{Axilla} was already 4.5 ˚C higher in HOT than in TEMP at pre-test, the difference doubled across the 60-min SIM (to 9.3 ˚C, $P < 0.001$).

The T_{Arm} decreased by 5.0 - 5.6 ˚C across TEMP ($P < 0.001$), and by 1.6 – 2.4 ˚C across HOT ($P \leq 0.02$; Interaction effect: $P < 0.001$). Thus, while T_{Arm} was already 6.3 ˚C higher in HOT than TEMP at pre-test, the difference increased further across the 60-min SIM (to 10.7 ˚C, $P < 0.001$).

The T_{Finger} decreased by 5.4 ˚C from pre-test to 10 min ($P < 0.001$), then increased by 4.0 - 5.9 ˚C from 10 min compared to 20 to 60 min across TEMP ($P < 0.001$), and decreased by 2.3 to 2.6 ˚C from pre-test compared to 10 and 20 min for HOT ($P \leq 0.03$; Interaction effect: $P < 0.001$). Thus, while T_{Finger} was 9.3 ˚C higher in HOT than TEMP at pre-test, the difference decreased across the 60-min SIM (to 7.8 ˚C, $P < 0.001$).

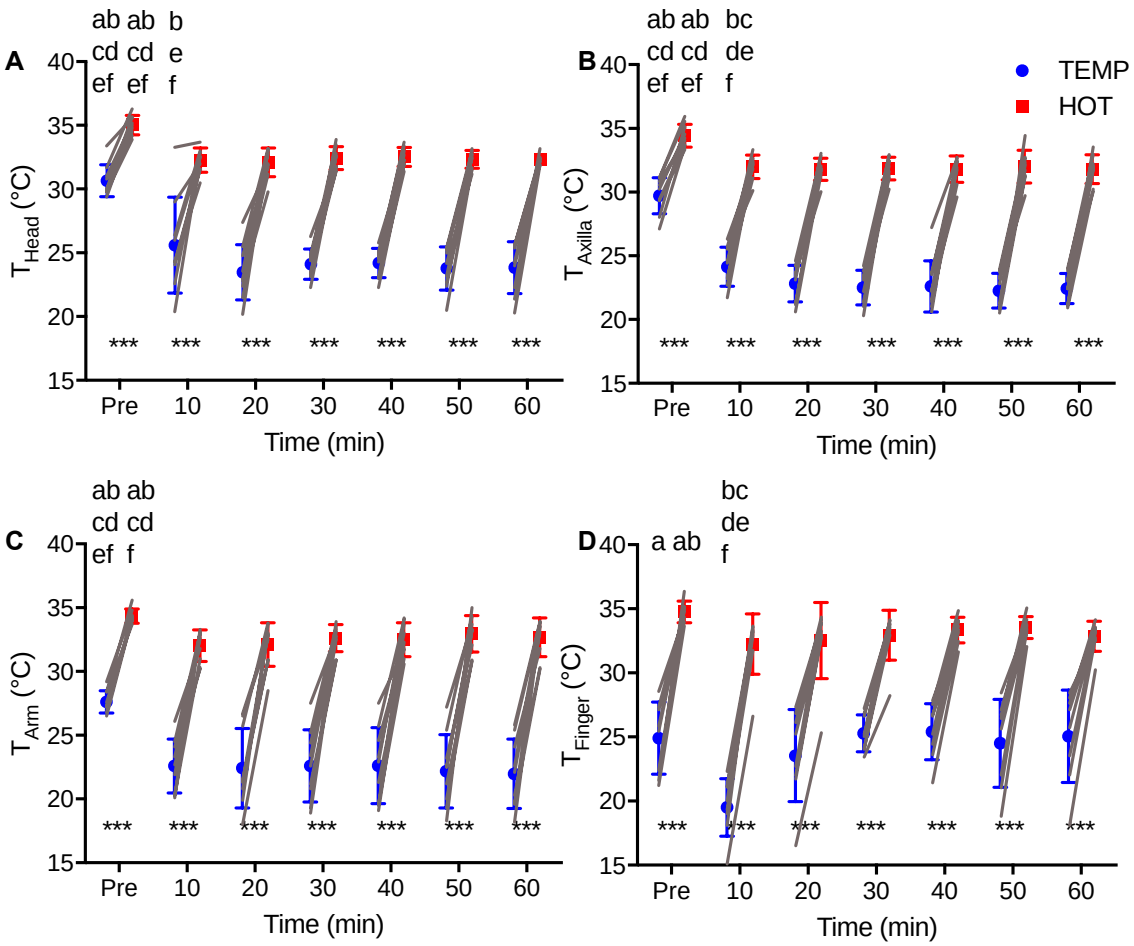


Figure 6.8: Skin temperature at the forehead (A), mid-axilla (B), forearm (C), and finger (D) in TEMP and HOT from pre-test resting in the environment for 30 min (Pre) and at 10-, 20-, 30-, 40-, 50-, and 60-min of the race simulation. Mean ± SD in coloured symbols, individual responses in grey lines. Time effect: $P < 0.05$ at: [a] 10 min, [b] 20 min, [c] 30 min, [d] 40 min, [e] 50 min, [f] 60 min, Temperature effect: ***$P < 0.001$.

6.5.3 Respiratory Responses

6.5.3.1 Minute Ventilation

Minute ventilation during FIXED increased over time and tended to be higher in HOT (Interaction effect: P = 0.07; Time effect: P < 0.001; Temperature effect: P < 0.001; Figure 6.9). Specifically, $\dot{V}_E$ increased 43% from $FIXED_{First}$ to $FIXED_{Last}$ (P < 0.001) and was consistently higher in HOT at $FIXED_{First}$, $FIXED_{Mid}$, and $FIXED_{Last}$ (by ~10%, Temperature effect: P < 0.001).

In contrast to FIXED, $\dot{V}_E$ was lower in HOT compared to TEMP by ~10% from $FREE_{Mid}$ to $FREE_{Last}$ (P ≤ 0.03). In both environments $\dot{V}_E$ decreased from $FIXED_{Last}$ to $FREE_{First}$, then across FREE $\dot{V}_E$ increased in TEMP by 15% but was similar in HOT (Interaction effect: P < 0.001; Figure 6.9).

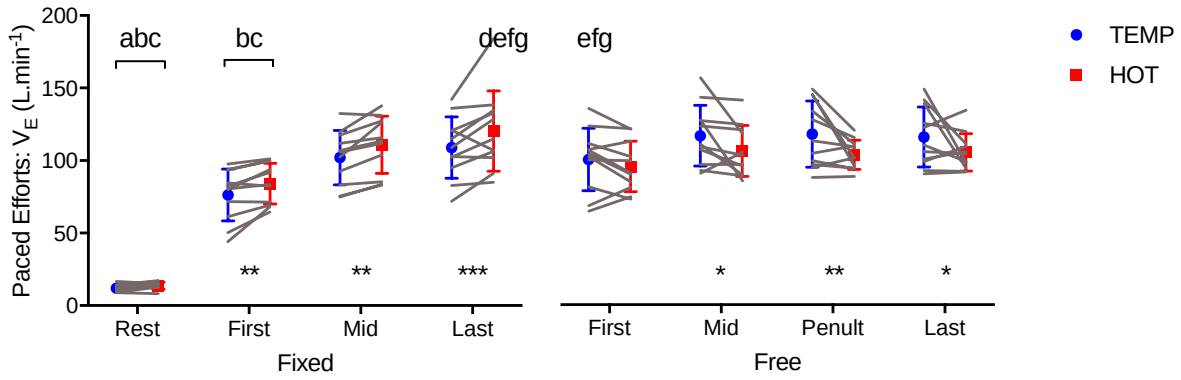


Figure 6.9: Ventilation for paced efforts at rest (Rest), and then the first, middle and end for FIXED followed by the first, middle, penultimate and last for FREE during the race simulation. Mean ± SD in coloured symbols, individual responses in grey lines. Time effect: P < 0.05 at: [a] $FIXED_{First}$, [b] $FIXED_{Mid}$, [c] $FIXED_{Last}$, [d] $FREE_{First}$, [e] $FREE_{Mid}$, [f] $FREE_{Penult}$, [g] $FREE_{Last}$; Temperature effect: *P < 0.05, **P < 0.01, ***P < 0.001.

6.5.3.2 Partial Pressure of End-Tidal Carbon Dioxide

The $P_{ET}CO_2$ increased from Rest to $FIXED_{First}$, then decreased steadily during FIXED in TEMP and HOT, but to a greater extent in HOT (Interaction effect: P = 0.04; Figure 6.10). Further, $P_{ET}CO_2$ tended to be lower in HOT at Rest (P = 0.09) and was 1.3 (1.6) mm Hg, 2.0 (1.9) mm Hg, and 3.1 (2.3) mm Hg lower at $FIXED_{First}$, $FIXED_{Mid}$ and $FIXED_{Last}$, respectively (all P < 0.05).

The $P_{ET}CO_2$ decreased across FREE in both environments and was consistently lower in HOT (Interaction effect: P = 0.05; Figure 6.10). Specifically, $P_{ET}CO_2$ decreased from $FIXED_{Last}$ to $FREE_{Last}$ in TEMP (by 3.1 mmHg, P < 0.001) and HOT (by 2 mmHg, P < 0.001). The $P_{ET}CO_2$ in HOT was 2.7 (1.4) mmHg, 1.5 (1.7) mmHg, 1.3 (2.0) mmHg, and 2.0 (2.0) mmHg lower at $FREE_{First}$, $FREE_{Mid}$, $FREE_{Penult}$, and $FREE_{Last}$, respectively (all P ≤ 0.006).

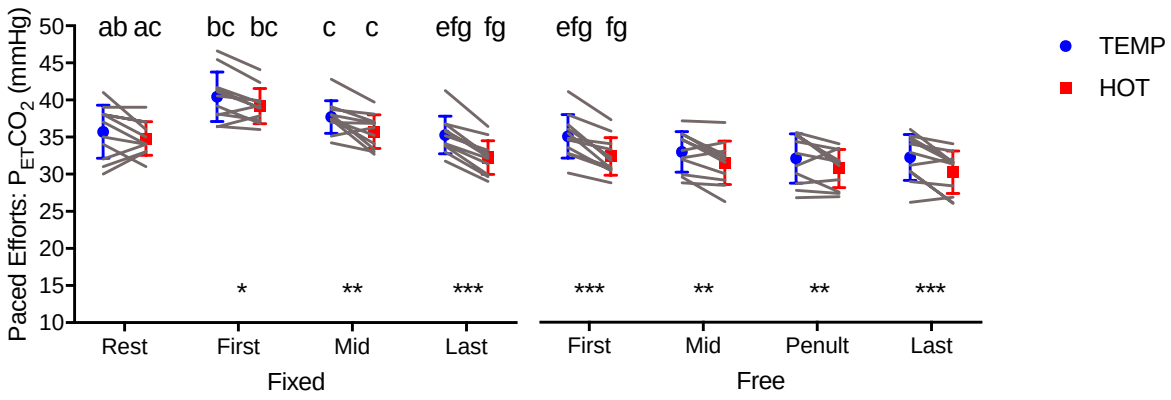


Figure 6.10: Partial pressure of end-tidal carbon dioxide for paced efforts at rest (Rest), and then the first, middle and end for FIXED followed by the first, middle, penultimate and last for FREE during the race simulation. Mean ± SD in coloured symbols, individual responses in grey lines. Time effect: P < 0.05 at: [a] $FIXED_{First}$, [b] $FIXED_{Mid}$, [c] $FIXED_{Last}$, [e] $FREE_{Mid}$, [f] $FREE_{Penult}$, [g] $FREE_{Last}$; Temperature effect: *P < 0.05, **P < 0.01, ***P < 0.001.

6.5.3.3 Cerebrovascular Reactivity

Cerebrovascular reactivity (CVR) was similar between TEMP and HOT when individuals underwent the hypocapnia CVR challenge, while hypercapnia CVR was 43% lower resting in HOT environments (P = 0.01). Interestingly, this temperature effect was not evident during the exercising hypercapnia CVR test completed prior to the 5 min TT protocol (P = 0.12; Figure 6.11, Figure 6.12).

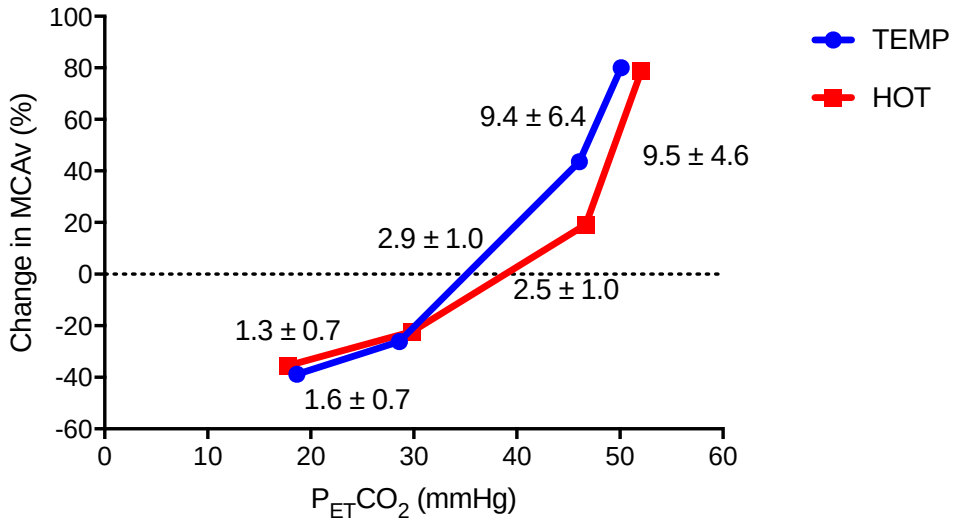


Figure 6.11: Brain blood flow velocity in response to the change in partial pressures of end-tidal carbon dioxide after 3 min: 1) voluntary hyperventilation at rest to 20 mm Hg, 2) voluntary hyperventilation at rest to 30 mm Hg, 3) breathing a 5% CO_2 gas mixture during rest, and 4) breathing a 5% CO_2 gas mixture whilst exercising at 30% peak power output. Group cerebrovascular reactivity (%MCAv / mm Hg, mean ± SD) for each slope (hypocapnia, hypercapnia) in temperate and hot environments.

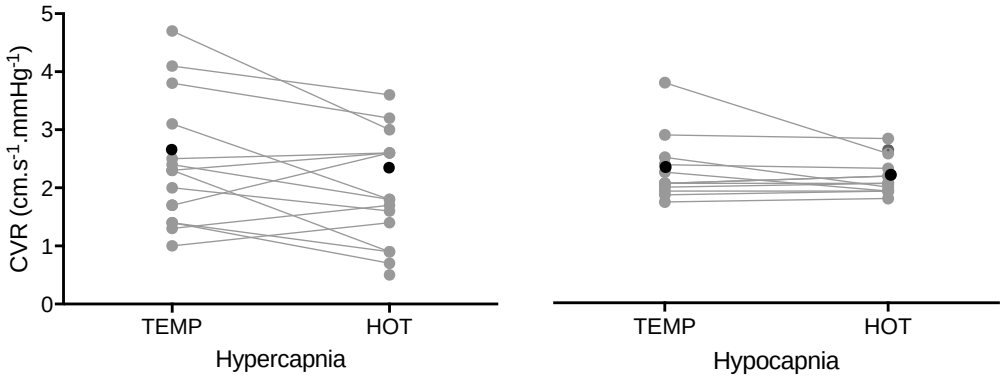


Figure 6.12: Cerebrovascular reactivity (CVR) in temperate (TEMP) and hot (HOT) environments during hypocapnia (hyperventilating) and hypercapnia (breathing 5% CO_2). Black dots for grouped mean, grey dots and lines for individual responses.

6.5.4 Cardiovascular Responses

6.5.4.1 Blood Pressure

Systolic blood pressure (SBP) increased from resting measures in both TEMP and HOT environments, then decreased across the 60-min SIM, but by a greater extent in HOT (Interaction effect: P < 0.001; Figure 6.13). Specifically, SBP in both environments increased by ~30 mmHg from Rest to $FIXED_{Mid}$ (P < 0.001), but by the end of the 60 min ($FREE_{Last}$) SBP in the HOT environment was similar to resting values (P = 0.79) and was 22 mmHg lower than TEMP (P < 0.001).

Diastolic blood pressure (DBP) decreased from Rest to the end of 60-min SIM in both TEMP and HOT environments, but by a greater extent in HOT (Interaction effect: P = 0.06; Time effect: < 0.001; Temperature effect: P < 0.001; Figure 6.13). Specifically, DBP in both environments decreased from Rest to $FREE_{Last}$ (by 14 mmHg, P = 0.001) and was lower in HOT at $FIXED_{Last}$ (by 16 mmHg, P = 0.002) and $FREE_{Last}$ (by 13 mmHg, P= 0.01).

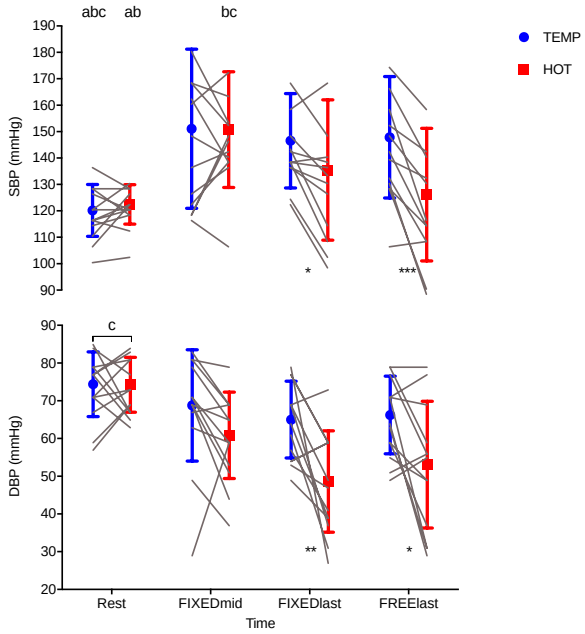


Figure 6.13: Systolic and diastolic blood pressure at rest (Rest), $FIXED_{Mid}$, $FIXED_{Last}$, and $FREE_{Last}$ during the race simulation. Mean ± SD in coloured symbols, individual responses in grey lines. Time effect: P < 0.05 compared with: [a] $FIXED_{Mid}$, [b] $FIXED_{Last}$, [c] $FREE_{Last}$; Temperature effect: *P < 0.05, **P < 0.01, ***P < 0.001.

6.5.4.2 Heart Rate

Heart rate increased in both environments and was higher in HOT compared with TEMP across FIXED (Interaction effect: P = 0.26; Time effect: < 0.001;

Temperature effect: P = 0.005; Figure 6.14) and FREE (Interaction effect: P < 0.001). Specifically, HR increased across FIXED (by 31 beats·min^{-1}, P < 0.001) and was higher in HOT (by ~ 7 beats·min^{-1}, P ≤ 0.06). The HR across FREE increased in TEMP from $FREE_{First}$ to $FREE_{Penult}$ (by ~5 beats·min^{-1}, P ≤ 0.02) while HR in HOT decreased from $FIXED_{Last}$ to $FREE_{First}$ (by 8 beats·min^{-1}, P < 0.001), but was similar across FREE.

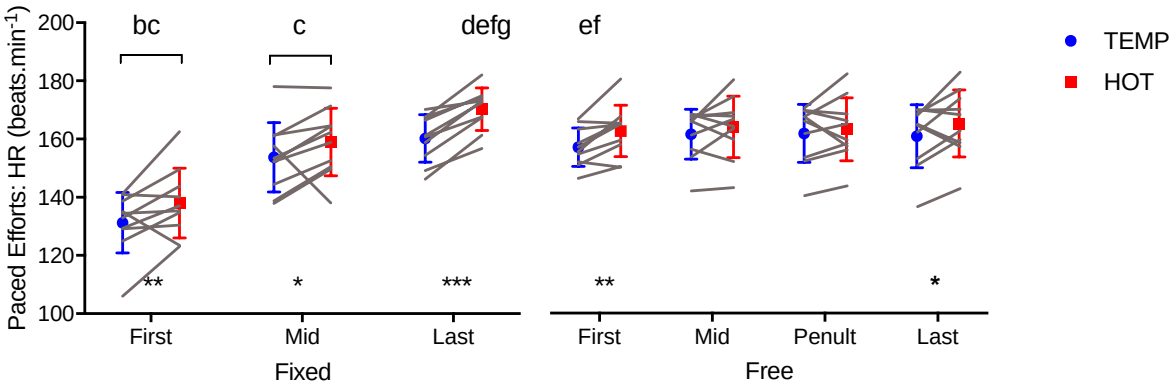


Figure 6.14: Heart rate for paced efforts at the first, middle and last for FIXED followed by the first, middle, penultimate and last for FREE during the race simulation. Mean ± SD in coloured symbols, individual responses in grey lines. Time effect: P < 0.05 at: [b] $FIXED_{Mid}$, [c] $FIXED_{Last}$, [d] $FREE_{First}$, [e] $FREE_{Mid}$, [f] $FREE_{Penult}$, [g] $FREE_{Last}$; Temperature effect: *P < 0.05, **P < 0.01, ***P < 0.001.

6.5.4.3 Oxygen Pulse

Oxygen pulse, the volume of oxygen consumed per cardiac cycle [O_2 pulse; relative aerobic power ($\dot{V}O_2$) divided by heart rate] tended to increase across FIXED and tended to be lower in HOT (Interaction effect: P = 0.89; Time effect: P = 0.07; Temperature effect: P = 0.13; Figure 6.15). Oxygen pulse decreased across FREE and was consistently lower in HOT (Interaction effect: P = 0.18; Time effect: P = 0.04; Temperature effect: P = 0.05; Figure 6.15). Specifically, oxygen pulse was lower in HOT by 13-22% between environments across FREE (P ≤ 0.03).

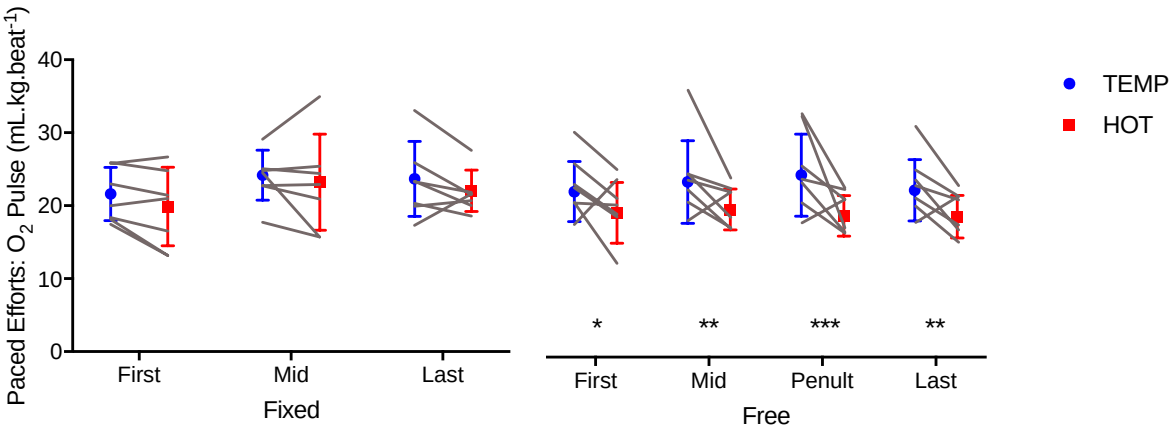


Figure 6.15: Oxygen pulse for paced efforts at the first, middle and last for FIXED followed by the first, middle, penultimate and last for FREE during the race simulation. Mean ± SD in coloured symbols, individual responses in grey lines. Temperature effect: *P < 0.05, **P < 0.01, ***P < 0.001.

6.5.4.4 Skin Blood Flow

Forearm skin blood flow (BF_{Skin}) increased ~3.0 a.u. from pre to post 60-min SIM and was ~2.2 a.u. higher in HOT (Interaction effect: P = 0.76; Time effect: P = 0.006; Temperature effect: P = 0.003; Figure 6.16).

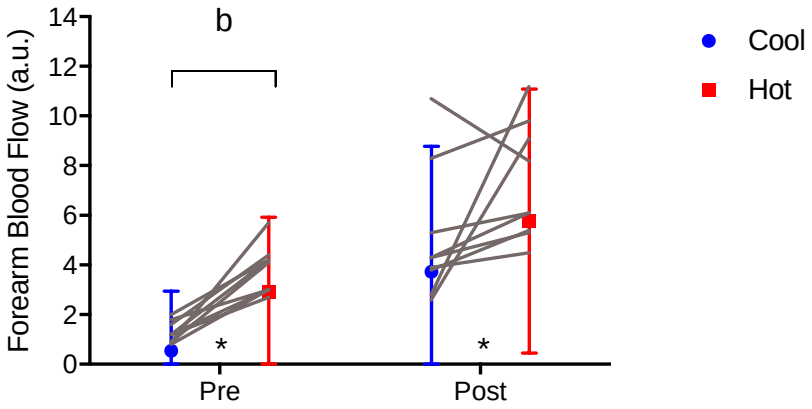


Figure 6.16: Forearm skin blood flow in temperate and hot environments from pre- (Pre) after resting in the environment for 30 min to post-test (Post) immediately after the race simulation. Mean ± SD in coloured symbols, individual responses in grey lines . Time effect: [b] P < 0.05; Temperature effect: * P < 0.05.

6.5.5 Brain (prefrontal cortex) Haemodynamics

The change in oxygenated haemoglobin (from rest) measured at the prefrontal cortex (Brain O_2Hb) increased across FIXED and was higher in HOT at the first effort but lower (on average) by the middle and end efforts (Interaction effect: P < 0.001; Figure 6.17A). Specifically, Brain O_2Hb increased from $FIXED_{First}$ to $FIXED_{Mid}$ in TEMP (by 389 μM·cm, P < 0.001) and HOT (by 201μM·cm, P < 0.001) but was was higher at $FIXED_{First}$ in HOT (by 120 μM·cm, P = 0.004) then similar from $FIXED_{Mid}$ to $FIXED_{Last}$ in both environments (P > 0.62). The Brain O_2Hb was similar across FREE and between environments (Interaction effect: P = 0.54; Time effect: P = 0.77; Temperature effect: P = 0.99).

Deoxygenated haemoglobin (Brain HHb) increased across FIXED and was higher in HOT at the middle and last efforts (Interaction effect: P = 0.004; Figure 6.17B). Specifically, Brain HHb increased in TEMP from $FIXED_{First}$ to $FIXED_{Mid}$ (by 32 μM·cm, P = 0.03) and in HOT from $FIXED_{First}$ to $FIXED_{Last}$ (by 106 μM·cm, P < 0.001). The Brain HHb was higher in HOT compared with TEMP at $FIXED_{Mid}$ (by 43 μM·cm, P = 0.003) and $FIXED_{Last}$ (by 59 μM·cm, P < 0.001). The Brain

HHb increased across FREE and was higher in HOT (Interaction effect: P = 0.45; Time effect: P = 0.04; Temperature effect: P = 0.03). Specifically, Brain HHb was ~50 μM·cm (P < 0.003) higher in HOT at all FREE time points and tended to increase across FREE (pooled across environments, P = 0.07).

Total haemoglobin (Brain tHb) increased across FIXED and tended to be higher in HOT (Interaction effect: P = 0.16; Time effect: P < 0.001; Temperature effect: P = 0.17; Figure 6.17C). Specifically, Brain tHb (pooled across environments) increased from $FIXED_{First}$ to $FIXED_{Last}$ (by 442 μM·cm, P < 0.001). The Brain tHb was similar from $FIXED_{Last}$ to $FREE_{First}$, all FREE time points, and between environments (Interaction effect: P = 0.52; Time effect: P = 0.98; Temperature effect: P = 0.27).

Brain tissue oxygen index (Brain TOI) across FIXED was similar in TEMP and decreased in HOT (Interaction effect: P = 0.005; Figure 6.17D). Specifically, while Brain TOI remained relatively constant in TEMP (P > 0.44), it decreased from $FIXED_{First}$ to $FIXED_{Last}$ (by 5%, P < 0.001) and was lower in HOT only at $FIXED_{First}$ (by 3%, P = 0.008). During FREE Brain TOI was similar across all time points and between environments (Interaction effect: P = 0.20; Time effect: P = 0.92; Temperature effect: P = 0.86), but did rebound in HOT from $FIXED_{Last}$ to $FREE_{First}$ by 2%.

The change in brain blood flow velocity (from rest) measured at the middle cerebral artery (MCAv) tended (P = 0.13) to decrease across FIXED for both environments, but not differentially so (Interaction effect: P = 0.28; Temperature effect: P = 0.59; Figure 6.17E). The MCAv was similar from $FIXED_{Last}$ to $FREE_{First}$ (P > 0.99) and between environments (P = 0.59), but tended to decrease across FREE (Interaction effect: P = 0.16; Time effect: P = 0.10).

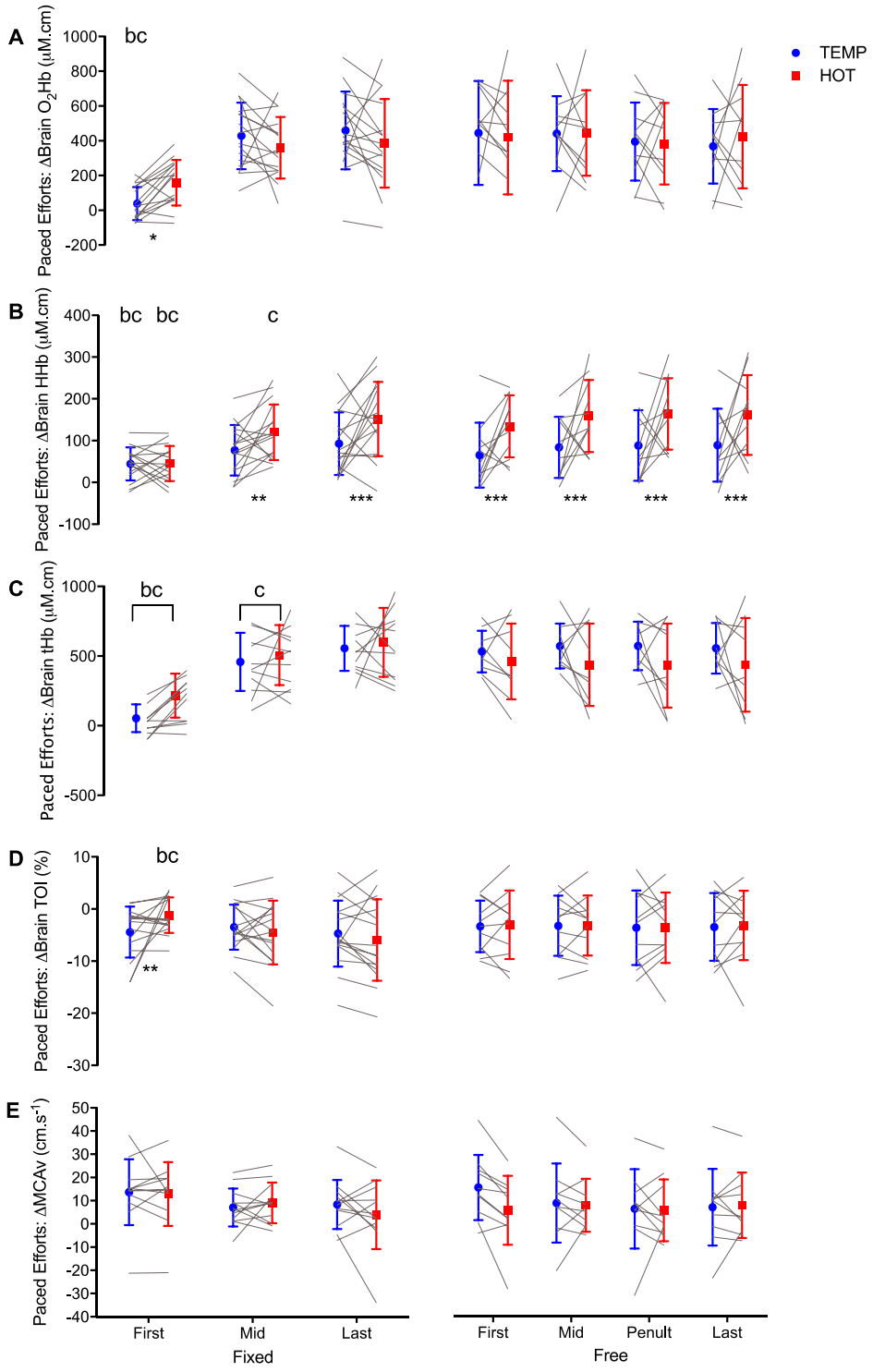


Figure 6.17: Brain oxygenated haemoglobin (A), deoxygenated haemoglobin (B), total haemoglobin (C), brain tissue oxygen saturation (D) measured at the prefrontal cortex and at the middle cerebral artery (E) at the first, middle, penultimate, and last Paced Efforts (1:50 every 2:00) for FIXED (pre-set power) and FREE (self-selected power) during cycling simulation. Mean ± SD in coloured symbols, individual responses in grey lines. Time effect: $P < 0.05$ at: [b] $FIXED_{Mid}$, [c] $FIXED_{Last}$; Temperature effect: *$P < 0.05$, **$P < 0.01$, ***$P < 0.001$.

6.5.6 Peripheral Haemodynamics

The change in oxygenated haemoglobin (from rest) measured at the left leg vastus lateralis muscle (Leg O_2Hb) increased in TEMP and decreased in HOT across FIXED, remaining consistently lower in HOT (Interaction effect: $P < 0.001$; Figure 6.18A). Specifically, Leg O_2Hb increased in TEMP from $FIXED_{First}$ to $FIXED_{Mid}$ (by 178 μM·cm, $P < 0.001$), but was similar in HOT across all FIXED time points ($P > 0.92$); with lower Leg O_2Hb in HOT at $FIXED_{Mid}$ (by 120 μM·cm, $P < 0.001$) and $FIXED_{Last}$ (by 125 μM·cm, $P < 0.001$). The Leg O_2Hb increased across FREE and was similar between environments (Interaction effect: $P = 0.11$; Time effect: $P < 0.001$; Temperature effect: $P = 0.24$). Specifically, Leg O_2Hb increased from $FIXED_{Last}$ to $FREE_{First}$ (by 73 μM·cm, $P = 0.01$) then was similar across FREE ($P = 0.18$).

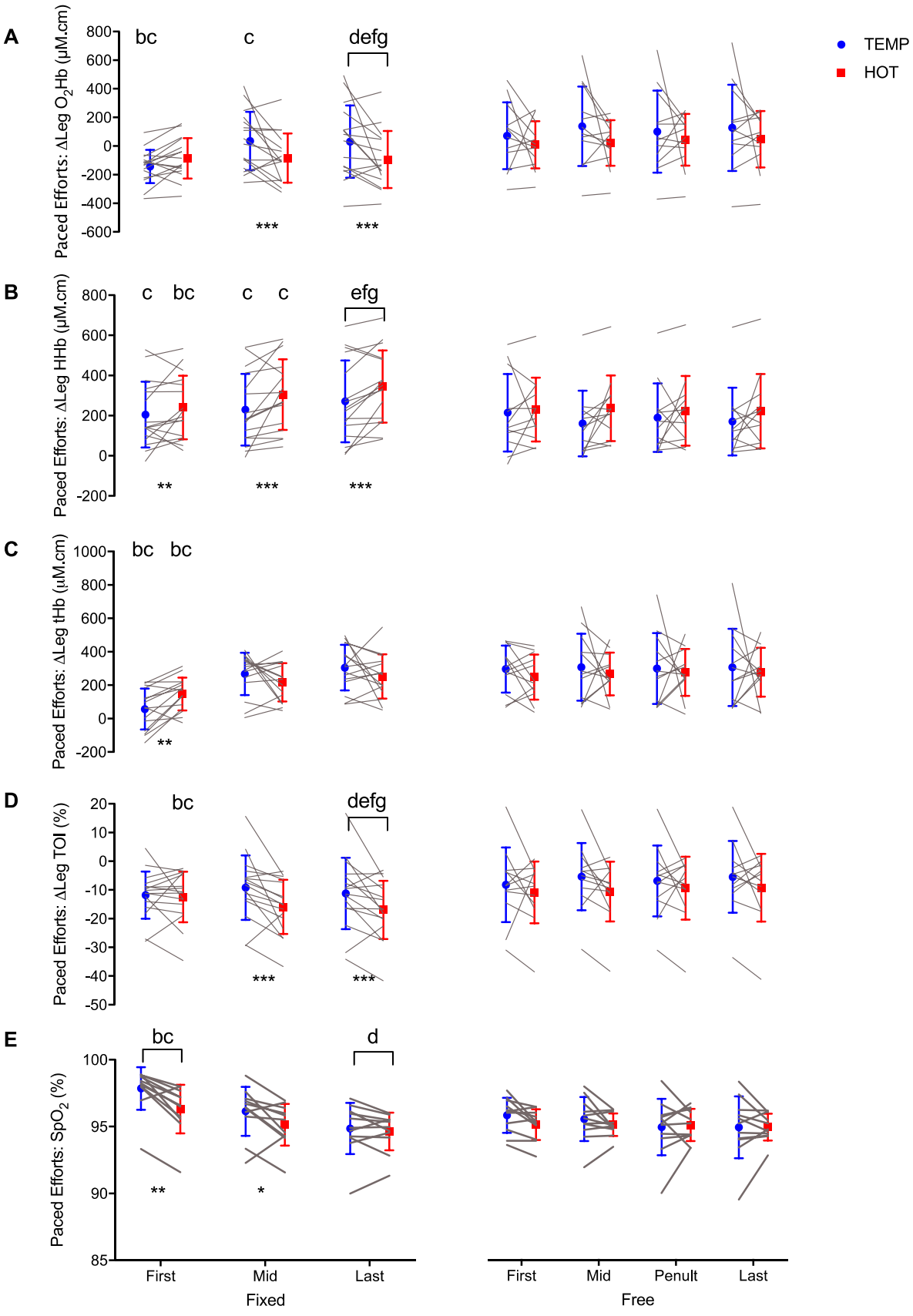
Deoxygenated haemoglobin (Leg HHb) increased across FIXED and was consistently higher in HOT (Interaction effect: $P = 0.04$; Figure 6.18B). Specifically, Leg HHb increased in TEMP from $FIXED_{Mid}$ to $FIXED_{Last}$ (by 66 μM·cm, $P = 0.004$), increased in HOT from $FIXED_{First}$ to $FIXED_{Last}$ (105 μM·cm, $P < 0.001$), and was higher in HOT at $FIXED_{First}$, $FIXED_{Mid}$, and $FIXED_{Last}$ (by 35 μM·cm, $P = 0.005$; by 75 μM·cm, $P < 0.001$; by 74 μM·cm, $P < 0.001$ respectively). The Leg HHb tended to decrease from $FIXED_{Last}$ to $FREE_{First}$ ($P = 0.10$) but was similar across FREE (pooled across environments) and tended to be higher in HOT (Interaction effect: $P = 0.59$; Time effect: $P = 0.003$; Temperature effect: $P = 0.12$).

Total haemoglobin (Leg tHb) increased across FIXED and was initially higher, then lower in HOT environments (Interaction effect: $P < 0.001$; Figure 6.18C). Specifically, Leg tHb increased from $FIXED_{First}$ to $FIXED_{Mid}$ in TEMP (by 210 μM·cm, $P < 0.001$), and HOT (by 70 μM·cm, $P = 0.03$), and was higher in HOT at $FIXED_{First}$ (by 90 μM·cm, $P = 0.004$), but tended to be lower at $FIXED_{Mid}$ and $FIXED_{Last}$ ($P = 0.09$). The Leg tHb tended to increase across FREE (pooled across environments) and was similar between TEMP and HOT (Interaction effect: $P = 0.70$, Time effect: $P = 0.16$, Temperature effect: 0.49).

Leg tissue oxygen index (Leg TOI) across FIXED was similar in TEMP and decreased in HOT, and was consistently lower in HOT (Interaction effect: P = 0.006; Figure 6.18D). In HOT Leg TOI decreased from $FIXED_{First}$ to $FIXED_{Last}$ (by 4%, P = 0.04), and was lower at $FIXED_{Mid}$ and $FIXED_{Last}$ (by 7%, P < 0.001; by 6%, P < 0.001 respectively). The Leg TOI increased from $FIXED_{Last}$ to $FREE_{First}$ in both environments (by 3%, P = 0.03) then was similar across FREE (for pooled environments), and was similar between TEMP compared with HOT (Interaction effect: P = 0.27; Time effect: P < 0.001; Temperature effect: P = 0.20).

Finger capillary oxygen saturation (SpO_2) decreased across FIXED and was consistently lower in HOT, tending to decrease more over time in HOT (Interaction effect: P = 0.09; Time effect: P < 0.001; Temperature effect: P < 0.001; Figure 6.18E). Specifically, SpO_2 decreased from $FIXED_{First}$ to $FIXED_{Last}$ (by 2%, P < 0.001) and was lower in HOT at $FIXED_{First}$ and $FIXED_{Mid}$ (by 2%, P = 0.003 and 1%, P = 0.04 respectively). The SpO_2 tended to increase from $FIXED_{Last}$ to $FREE_{First}$ (P = 0.06), was similar across FREE (pooled between environments), and similar when TEMP was compared with HOT (Time effect: P = 0.05; Interaction effect: P = 0.22; Temperature effect: P = 0.44).

Blood lactate concentration ([La]) increased from Pre to $FIXED_{Mid}$ (by 3.6 $mmol \cdot L^{-1}$, P = 0.001), was similar during FIXED (P > 0.99), then increased during FREE (by 2.9 $mmol \cdot L^{-1}$, P = 0.01) and tended to be higher in HOT at each time point (Interaction effect: P = 0.24; Time effect: P < 0.001; Temperature effect: P = 0.15; Figure 6.18F**).**


A
B
C
D
E
TEMP
HOT
Paced Efforts: ΔLeg O2Hb (μM.cm)
Paced Efforts: ΔLeg HHb (μM.cm)
Paced Efforts: ΔLeg tHb (μM.cm)
Paced Efforts: ΔLeg TOI (%)
Paced Efforts: SpO2 (%)
First
Mid
Last
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Fixed
Free

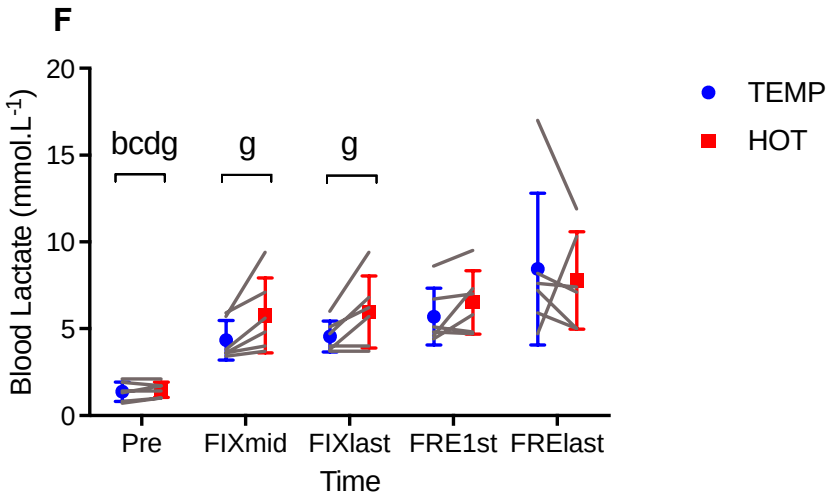


Figure 6.18: Leg oxygenated haemoglobin (A), deoxygenated haemoglobin (B), total haemoglobin (C), leg tissue oxygen index (D) and finger capillary oxygen saturation (E) measured at the first, middle, penultimate, and last Paced Efforts (1:50 every 2:00) for FIXED (pre-set power) and FREE (self-selected power) during cycling simulation. Blood lactate (F) measured before exercise (Pre) during FIXED (FIX) and FREE (FRE) during cycling simulation. Mean ± SD in coloured symbols, individual responses in grey lines. Time effect: $P < 0.05$ at: [a] FIXED First, [b] FIXED Mid, [c] FIXED Last, [d] FREE First, [e] FREE Mid, [f] FREE Penult, [g] FREE Last; Temperature effect: *$P < 0.05$, **$P < 0.001$, ***$P < 0.001$.

6.5.7 Psycho-Physical Responses

Participants began both trials in thermal comfort and reported greater discomfort as the race simulation progressed, especially in HOT (Interaction effect: $P < 0.001$; Figure 6.19A). Specifically, thermal discomfort across FIXED in a TEMP environment was similar ($P > 0.36$) but increased in HOT from 'comfortable' at Rest to 'slightly uncomfortable' at $FIXED_{Last}$ ($P < 0.001$). Thermal discomfort was similar at Rest and Pre, then was more uncomfortable in HOT at $FIXED_{Mid}$ and $FIXED_{Last}$ ($P < 0.001$ in both respectively). Thermal discomfort did not increase further from $FIXED_{Last}$ and across FREE, but participants continued to feel greater discomfort in HOT (Interaction effect: $P = 0.61$; Time effect: $P = 0.69$; Temperature effect: $P < 0.001$).

Participants felt cooler from Rest to Pre then warmer during exercise in TEMP, while they felt considerably warmer across 60-min TT in HOT (Interaction effect: $P < 0.001$; Figure 6.19B). Specifically, thermal sensation in TEMP decreased from 'neutral' at Rest to 'slightly cool' at Pre ($P= 0.002$) then increased to 'neutral' at $FIXED_{Mid}$ ($P = 0.004$) to 'slightly warm' at $FIXED_{Last}$ ($P = 0.01$) and remained 'slightly warm' throughout FREE ($P > 0.68$). By comparison, thermal sensation in HOT increased from 'neutral' at Rest to 'warm' at Pre ($P = 0.02$) to 'hot' at $FIXED_{Mid}$ ($P = 0.05$) to 'very hot' at $FIXED_{Last}$ ($P = 0.03$), remaining 'very hot' throughout FREE ($P > 0.56$). Thermal sensation was higher in HOT at Pre, $FIXED_{Mid}$, $FIXED_{Last}$, $FREE_{First}$ and $FREE_{Last}$ (Temperature effect: $P < 0.001$).

Participants perceived they were exercising progressively harder across 60-min SIM and that they were exercising harder in HOT environments, with a tendency for RPE to be highest at the end of 60-min SIM in HOT (Time effect: $P < 0.001$; Temperature effect: $P < 0.001$; Interaction effect: $P = 0.08$; Figure 6.19C). Specifically, in TEMP RPE increased from 'somewhat hard' at $FIXED_{Mid}$ to 'hard' at $FIXED_{Last}$ ($P < 0.001$), then remained at this rating across all $FREE_{Mid}$ time points ($P > 0.58$). In HOT, RPE increased from 'hard' to 'very hard' at $FIXED_{Last}$, then remained at this rating across all FREE time points ($P > 0.42$).

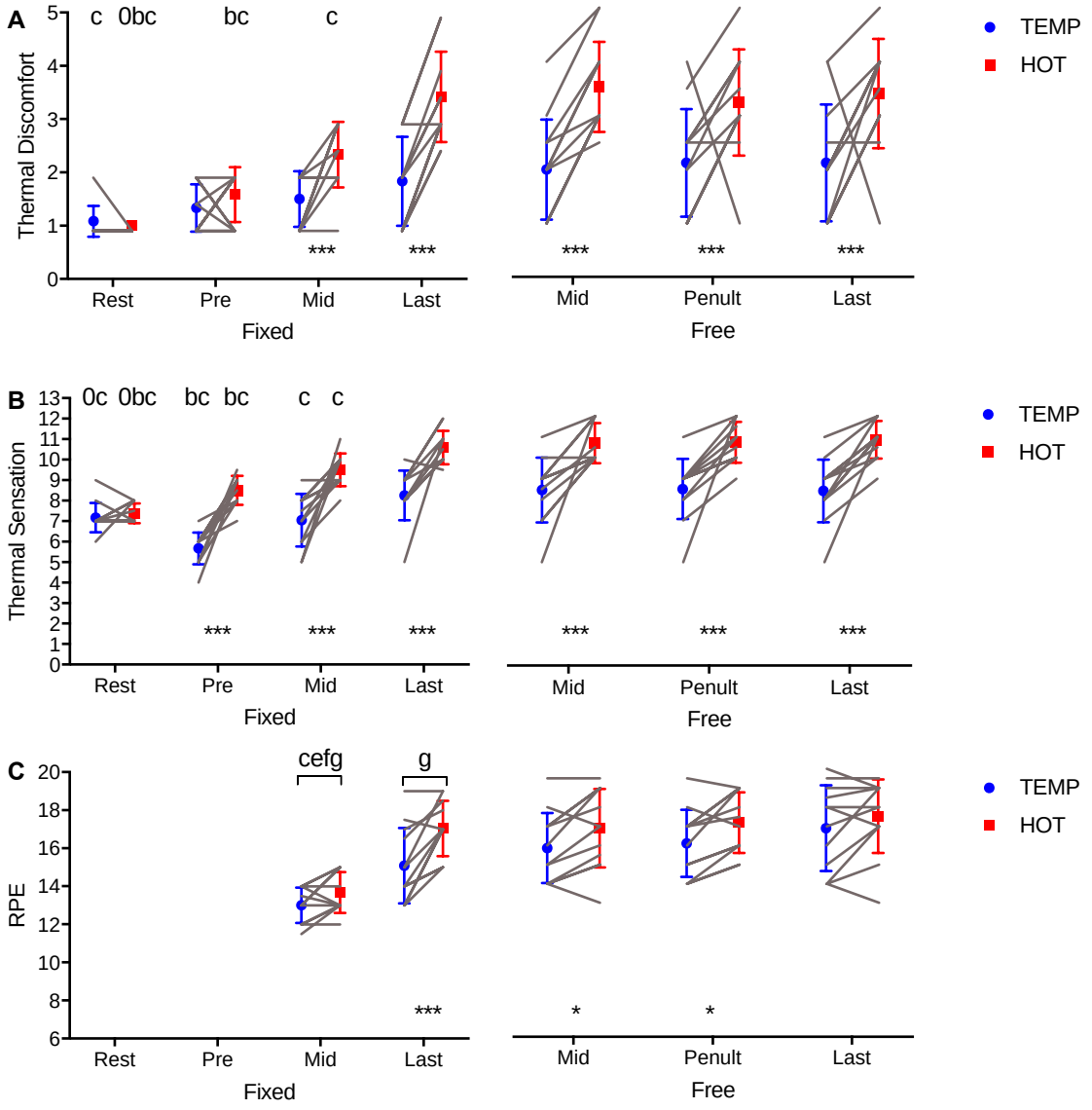


Figure 6.19: Thermal discomfort (A), thermal sensation (B), and exertion (C) at rest (Rest), after 30 minutes resting in the environment (Pre), then at the middle and end of FIXED and at the middle, penultimate and last effort of FREE for the cycling simulation. Mean ± SD in coloured symbols, individual responses in grey lines. Time effect: $P < 0.05$ at: ^{0} Pre, ^{b} $FIXED_{Mid}$, ^{c} $FIXED_{Last}$, ^{e} $FREE_{Mid}$, ^{f} $FREE_{Penultimate}$, and ^{g} $FREE_{Last}$; Temperature effect: *$P < 0.05$, **$P < 0.01$, ***$P < 0.001$.

6.5.8 Fluid Loss

Fluid loss approximated as gross loss of body mass from pre- to post-trial was 84% greater in HOT ($P < 0.001$; Figure 6.20).

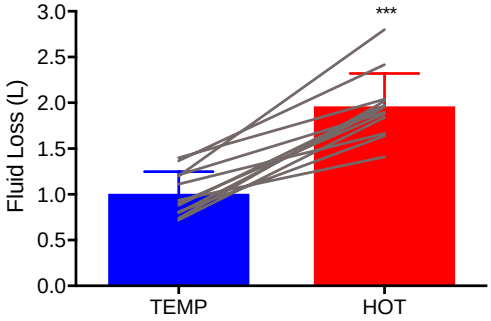


Figure 6.20: Fluid loss from the start to the end of the cycling simulation in temperate and hot environments. Mean + SD in coloured symbols, individual responses in grey lines, time effect: ***$P < 0.001$.

6.5.9 Blood Volume Responses

Across the 6 days of the 10 s sprints, 5-min TT and 60-min SIM in TEMP and HOT environments, BV increased 2.7% ($P = 0.03$) and PV increased 6.4% ($P < 0.001$), with no change in Hb mass ($P = 0.99$) or RCV ($P = 0.43$; Figure 6.21).

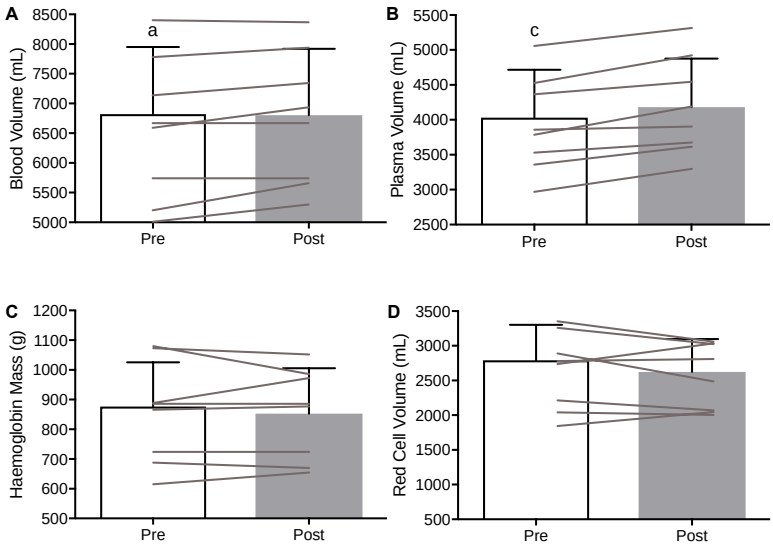


Figure 6.21: Blood volume (A), plasma volume (B), haemoglobin mass (C), and red cell volume (D) measured the day before (Pre) and day after (Post) all five-minute time trials and 60-minute race simulations in temperate and hot environments. Data displayed as mean + SD, individual participants in grey lines. Time effect: [a] $P < 0.05$, [c] $P < 0.001$.

6.6 Discussion

The purpose of this study was to simulate the specific race performance demands from competing in an elite-level, draft legal 40-km cycle leg of a triathlon race. The environmental stress imposed by racing in New Zealand and Australian climates were simulated while measuring changes in central and peripheral physiological variables over time to identify which factors influence performance over time. Performance power was significantly reduced in HOT. Immediately before the 60-min SIM in HOT, T_{Skin} and BF_{Skin} were significantly higher pre-test causing blood flow redistribution and greater cardiovascular strain (reduced Leg TOI, SpO_2, MABP, elevated HR). When exercise commenced participants experienced greater respiratory strain (higher $\dot{V}_E$, reduced $P_{ET}CO_2$), higher T_C, and significant oxygen desaturation at the prefrontal cortex and skeletal muscle that – collectively - was severe enough to stop 9 out of 17 participants from finishing the fixed pace section. During the self-paced section oxygen desaturation combined with higher T_C likely had a negative influence on affective state and anticipatory pacing that further degraded power output. Surprisingly however, brain blood flow velocity at the middle cerebral artery was maintained in both environments despite hyperventilation-induced hypocapnia, and reduced MABP.

6.6.1 Cycling Race Simulation Power

Power output for paced efforts was similar between environments during FIXED, a result that was expected by design. Participants also experienced significant hyperventilation in both environments during FIXED, marked by decreased $P_{ET}CO_2$ (Figure 6.10), which would have caused decreased $PaCO_2$ and decreased $[H^+]$ that indicate CO_2 washout (Martin et al., 1979; Nybo and Nielsen, 2001b; White, 2006). Muscle-mediated feedback to ventilatory drive should also be considered as a powerful contributing factor given the stochastic nature of the exercise and the higher cardiovascular strain. This aspect was not measured in these studies and consequently, cannot be discounted.

All participants could complete the race simulation in TEMP with T_C peaking at < 38.7 ˚C (the last FIXED sprint) and plateaued thereafter. During the FREE

section participants were reminded to maintain the same paced effort power and sprint as hard as possible to simulate the effort required to achieve their best result. This was achieved in TEMP with statistically similar paced-effort power and greater sprints at each time point, with the highest power output achieved in the last sprint. The $\dot{V}_E$ for paced efforts was similar until $FREE_{Mid}$ and $FREE_{Last}$ where it reached its highest rate (also higher than HOT), perhaps indicating that participants could support higher $\dot{V}_E$ in TEMP because there was less cardiovascular competition with other vascular beds (Nielsen et al., 1990). Oxygen pulse was maintained in TEMP, which infers that athletes could maintain SV via enhanced cardiac filling pressure (Gledhill et al., 1994; González-Alonso et al., 2000).

Athletes also felt better in TEMP. After 40 minutes ($FIXED_{Last}$) participants felt slightly uncomfortable, slightly warm, and exercising hard; these perceptions described a lower psycho-physical strain compared with HOT, and these perceptions were maintained throughout self-paced exercise. The exception was RPE, which increased to above very hard by the end of 60-min SIM and was similar to HOT (as expected from maximal effort exercise). Athletes also produced significantly more power in the last compared with the penultimate sprint, while none of the measured physiological variables showed significant differences. This end-spurt effect was observed in an intermittent running test (Catalano, 1973; Skein et al., 2011), and was effective provided recovery was sufficient (Glynn et al., 2008). However, the end-spurt did not occur in HOT for this study, or for cyclists performing intermittent sprints with short recoveries under hyperthermia-induced fatigue (Drust et al., 2005).

The HOT environment elevated skin blood flow before the cycle simulation started (Figure 6.16). Once participants began cycling metabolic heat production raised T_{Skin} to temperatures with much smaller thermal gradients (T_{Axilla}, T_{Arm} > 31˚C; T_{Head}, T_{Finger} > 32 ˚C; environment = 33 ˚C; Figure 6.8). The combined stress from the environment and cycling simulation drove T_C > 39 ˚C (Figure 6.7) and increased skin temperature and skin blood flow (Figure 6.8, Figure 6.16), suggesting that fatigue was driven in part by hyperthermia (Figure 6.1; Abbiss et al., 2010; Nybo and Nielsen, 2001a; Nybo and Secher, 2004; Tatterson et al.,

2000). Redistributing central and skeletal muscle blood volume to the skin to reduce blood temperature to the core and brain may have improved participants' ability to thermoregulate, but increased cardiovascular strain. This was observed by higher heart rate and lower systolic and diastolic blood pressure that most likely decreased central blood volume, EDV and SV (Figure 6.13, Figure 6.14; Caputa et al., 1986; Chou et al., 2018; Rowell, 1986; Rowell et al., 1966; Sawka and Coyle, 1999; Sawka et al., 2011b).

Participants also dehydrated from the start to finish of 60-min SIM by >2% body weight, sufficient to significantly impair aerobic performance (Cheuvront et al., 2003; Sawka, 1992; Sawka et al., 2007) and increase cardiovascular strain. Dehydration may also have reduced end diastolic filling indicated by diminished DBP and reduced stroke volume (González-Alonso et al., 1997, 2000; Nybo et al., 2001). The CNS may have interpreted the increased cardiovascular strain and lower blood pressure as the initial phase of circulatory failure: causing early fatigue, reducing motor output, and degrading performance power (Thompson, 2006).

Greater cardiovascular and respiratory strain, including lower blood pressure and $P_{ET}CO_2$, presumably contributed to the 5% decrease in Brain TOI throughout FIXED HOT (Figure 6.17) to similar levels observed at the end of 5-min TT (6%; Figure 5.14) and to levels that caused syncope in young, healthy adults (Thomas et al., 2010). Further, leg muscle oxygen saturation decreased from -12% to -17% during FIXED, desaturating to almost the same concentration as the end of 5-min TT in HOT (-18%) and indicating that the muscle may have approached or reached its physiological limit as early as $FIXED_{Mid}$ (-16%). The deterioration observed in peripheral haemodynamics combined with blood flow redistribution to the skin infer insufficient oxygen delivery and greater oxygen consumption at the muscle that was exacerbated as high intensity exercise progressed to maximal intensity (Gonzalez-Alonso and Calbet, 2003). As a result, 9 of the 17 participants could not complete the race simulation in HOT. Most participants switched from FIXED to FREE between 18 to 32 min. The high failure rate (53%) equated to the real-world race situation where an athlete would be dropped from the bunch and achieve a sub-standard overall performance in the race.

Similarly, a landmark study required participants to cycle at severe intensity while the sensory (but not motor) pathways were blocked (Amann, 2011). Performance deteriorated rapidly because the CNS could not upregulate homeostatic control processes including ventilation and blood flow, and peripherally because individuals attained peripheral locomotor muscle fatigue (Amann, 2011, 2012; Amann et al., 2011). The highly-trained participants in the present study had likely approached similar physiological limits at $FIXED_{Mid}$ compared to the end of 5-min TT (Chapter 5), indicating they were very close to volitional exhaustion. At $FIXED_{Mid}$ in HOT the prefrontal cortex had desaturated -4% (Figure 6.17D), which presumably contributed among multiple negative influences on participants' affective state (Figure 6.19A,B). The prefrontal cortex is a brain region that contributes to decision-making capacity (Krawczyk, 2002), so reduced oxygen saturation may have compromised decision making and pacing for the participants.

Failure to complete the full race simulation by 53% of athletes equated to a DNF and reflected the race outcomes in HOT (Mooloolaba ITU World Cup; described in Chapter 3, with 60% DNF). Participants who experienced volitional exhaustion during FIXED coped during FREE by producing efforts that were significantly inferior to the same time point in TEMP to maintain thermoregulation (Figure 6.5, Figure 6.6; Gonzalez-Alonso et al., 1999; Schlader et al., 2010b). Changing to FREE allowed participants to alter their pacing strategy but did not mitigate T_C or the rate of heat storage, which continued to increase until the end of the race simulation (group average reaching 39.2 ˚C; Figure 6.7; Saunders et al., 2005; Tucker et al., 2006b). The T_{Brain} and T_{mus} would presumably have been even hotter (T_{Brain} 0.2-0.3 ˚C higher than T_C; Nybo et al., 2002b; Yablonskiy et al., 2000). This demonstrated that when highly trained athletes reached exhaustion driven by T_C and blood flow redistribution they could self-pace to restore homeostasis, possibly because the CNS and peripheral muscle maintained a reserve capacity to avoid catastrophic failure at exhaustion when exercise was terminated voluntarily ($FIXED_{Last}$) or at the end of 60-min SIM (Noakes et al., 2005).

Athletes experienced greater thermal strain during FREE HOT, which was generated by near-maximal efforts in a warm and humid environment. Prolonged thermal strain may have exacerbated hyperventilation and increased respiratory strain (Figure 6.9; Cabanac and White, 1995). Both $\dot{V}_E$ and $P_{ET}CO_2$ were lower in HOT compared with TEMP at every time point in FREE, demonstrating that participants were in a constant hypocapnic and hyperventilatory state (decreased $PaCO_2$ and $[H^+]$) even though they could choose how hard to exercise. $\dot{V}_E$ was also significantly lower in HOT from $FIXED_{Last}$ compared with all FREE efforts, an unexpected outcome as $\dot{V}_E$ typically increases with exercise duration (Harms et al., 1998b, 2000b). Higher $\dot{V}_E$ typically corresponds with elevated HR (observed in TEMP), instead $\dot{V}_E$ was lower and HR was higher at the same time point. This may indicate that athletes could not sustain sufficient ventilation and skeletal muscle contraction during self-paced exercise to maintain performance power (Harms et al., 1998b). The observed hypocapnic hyperventilatory responses may have contributed to central fatigue by increasing brain metabolite accumulation (Raichle and Plum, 1972; Secher et al., 2008).

The elevated heart rates observed throughout FREE in HOT compared with TEMP may have been crucial to maintain cardiac output, oxygen delivery and skin blood flow (Cheuvront et al., 2003; Nybo and Nielsen, 2001b; Rowell et al., 1969b, 1969a). Oxygen extraction at the brain may have been heightened throughout FREE, possibly in response to higher T_{Brain} and metabolic rate (though neither variable was directly measured). This was observed by consistently higher Brain HHb with no difference between environments (Figure 6.17B). The reduced power output coupled with increased T_C observed during FREE HOT matches similar studies that have found elevated T_C, T_{Brain}, and reduced CNS output (Caputa et al., 1986; Morrison et al., 2004; Nybo and Nielsen, 2001a; Walters et al., 2000), and studies that have demonstrated involuntary anticipatory pacing to avoid catastrophic heat stress (Marino, 2004; Tucker et al., 2004).

Thermal strain and hyperventilation in FREE HOT also influenced perceptual strain. Participants felt uncomfortable, very hot, and perceived they were exercising very hard from $FIXED_{Last}$ until the end of the race simulation. Feelings

of thermal discomfort are strongly associated with high T_{Skin} (Figure 6.8; Gagge and Gonzalez, 1973; Gonzalez et al., 1973; Hardy, 1961) and central fatigue (Meeusen and Roelands, 2010; Schlader et al., 2011c). Furthermore, elevated T_{Skin}, thermal discomfort, and thermal sensation at the start of self-paced exercise (similar to this study) substantially influenced exercise intensity at the beginning and throughout the entire exercise task (Schlader et al., 2011c). Participant motivation was a major concern to the validity of this study because the race simulation forced individuals to be uncomfortable, very hot, exercising very hard, and (for 9/17 participants in HOT trials) incapable of finishing the fixed intensity section. However, participants could pace themselves to reach statistically similar RPE at the end of the race simulation.

Despite greater thermal, respiratory and perceptual strain during FREE HOT other physiological responses rebounded somewhat. Brain TOI increased from 6% to 3% below baseline levels (Figure 6.17D) and Brain tHb was maintained (Figure 6.13). Consistent brain oxygenation throughout self-paced exercise may be attributed to higher cerebral efficiency conferred by consistent endurance training in this participant group (Seifert et al., 2009b). Leg HbO_2, Leg tHb, Leg TOI, and finger capillary saturation also rebounded slightly. However, greater thermal strain during FREE tended to increase [La], inferring a higher metabolic rate driven by greater sympathetic activation, skeletal muscle temperature, and glycogen turnover (Figure 6.18F; Figure 6.5).

The final T_C in TEMP and HOT was higher in these experiments than the T_C recorded in elite athletes immediately after finishing racing, albeit with different and possibly less accurate equipment (Chapter 3). The primary reason may have been that airflow used in the laboratory (4 $m \cdot s^{-1}$, 50 cm diameter) did not match the convective cooling power experienced in the real world environment (Saunders et al., 2005). This study was unique in assessing performance and T_C responses based on both fixed and self-paced exercise protocols (Cheung, 2007) to examine the theories proposing critical core temperature (fixed intensity; Gonzalez-Alonso et al., 1999) and anticipatory regulation (self-selected intensity; Marino, 2004; Tucker et al., 2004). This study indicates that anticipatory pacing was very important to successfully completing the performance task (Abbiss et

al., 2010; Tatterson et al., 2000; Tucker, 2009; Tucker et al., 2004, 2006b), and concurs with Schalder et al. (2011a) that both an individualised critical T_C during fixed pace protocols and the ability to use anticipation while performing self-paced protocols maintained thermoregulation.

In summary, the combined CNS and cardiovascular strain may have generated both central *and* peripheral fatigue (Nybo, 2008) causing some participants to terminate exercise early through volitional exhaustion (n = 7) or because they reached the ethical limit for T_C (40 ˚C, n = 2, within the last few minutes of 60-min SIM). Most cardiovascular responses indicated that competition for perfusion was more pronounced in HOT and thus presumably contributed to worsen metabolic strain in active muscles, which was indicated to some extent during elite triathlon racing in warm conditions.

6.6.2 Brain Haemodynamics

Brain HHb in this study was significantly higher in HOT compared with TEMP and increased from Rest to $FIXED_{Last}$ by 97% in TEMP and 107% in HOT, as measured by NIRS. By comparison, Nybo et al (2002b) reported a 7% increase during a 65-min hyperthermia cycling protocol and Rasmussen et al. (2010b) reported a 43% increase from a 60-min hyperthermia cycling protocol, though both studies used a different technique (Kety-Schmidt). Although NIRS is perhaps susceptible to artefact by including BF_{Skin} within the sampled field, that would not confound these results because it would have the opposite effect on Brain HHb.

The increased Brain tHb during FIXED for both environments did not correlate with the large changes in MCAv (Figure 6.17C,E), a result that was also observed during high intensity exercise in a hypoxic environment (Subudhi et al., 2008). Brain blood flow velocity followed a different pattern to Brain O_2Hb, Brain HHb, and Brain tHb, showing a tendency ($P \leq 0.13$) to decrease across FIXED by 54% and across FREE by 82%. The brain blood flow decreased from the start to the end of 60-min SIM irrespective of environment (Figure 6.17E), which was unexpected for three reasons. Firstly, minute ventilation increased while $P_{ET}CO_2$

decreased and was significantly lower in HOT for both responses (Figure 6.9, Figure 6.10) indicating significant hyperventilatory hypocapnia that may have constricted vascular tone in cerebral arterioles in HOT (Ide and Secher, 2000; Kety and Schmidt, 1948; Rasmussen et al., 2005, 2007). However, the lower $\dot{V}_E$ during FREE in a HOT environment may have reduced hyperventilatory hypocapnia severity to a level that athletes could maintain while exercising. Secondly, cardiac output probably decreased during FREE in HOT, evidenced by significantly lower SBP (Figure 6.13), consistently higher HR (Figure 6.14), and higher skin blood flow that would divert a greater blood flow proportion away from the brain (Ide et al., 1998; Secher et al., 2008). And thirdly, MABP was lower throughout FREE, demonstrating that lower cardiac output and blood flow redistribution may have combined to substantially challenge cerebral autoregulation (Ide and Secher, 2000; Paulson et al., 1990). Despite these physiological challenges brain blood flow was maintained with minimal disturbance in HOT, possibly by cerebral vascular vasodilation (not measured), cardiac output and blood pressure responses sufficient to maintain the pressure gradient between MABP and intracranial pressure, and by increasing brain O_2, glucose, and [La] uptake (not measured). Crucially, cerebrovascular reactivity to hypocapnia (or hypercapnia) was not significantly different between TEMP and HOT, at least during low-intensity exericse in each environment (Figure 6.11, Figure 6.12). Brain blood flow was not a performance limitation in this study, and decreased brain blood flow did not influence prefrontal cortex oxygen saturation, results that contradict some research (Nybo and Rasmussen, 2007; Rasmussen et al., 2007, 2010b; Secher et al., 2008), but concur with studies that demonstrate brain blood flow restoration with supplemental CO_2 had no performance benefit (Keiser et al., 2015b).

6.6.3 Limitations

The performance task was a cycling simulation of a triathlon race, utilising stable power profiles in an artificially controlled laboratory environment. Though every effort was made to simulate the cycling leg of a draft-legal triathlon race this was not possible. The dynamic nature of other competitors and the participants own responses could not be replicated. Participant motivation to push themselves to the same intensities they experience in a race were also difficult to replicate.

However, participants reached and maintained very high RPE scale responses, while nine participants experienced volitional exhaustion during 60-SIM during FIXED. These responses validated the pilot study with five elite triathletes that demonstrated the exercise protocol was strenuous and specific to those experienced in a competitive race, and that recovery between successive performance tests was sufficient. Though every effort was made to simulate the convective airflow that participants experienced during the cycle leg wind speed was limited to 4 $m \cdot s^{-1}$ (15 $km \cdot h^{-1}$) compared with the ~8-14 $m \cdot s^{-1}$ (~30-50 $km \cdot h^{-1}$) that triathletes experience. The upper limit for T_C was also restricted to 40 ºC in this study for ethical reasons. Immediately after finishing a race elite triathletes have reached T_C > 40 ºC (unpublished observations). However, the impact of this limitation were minor as the two participants whose T_C reached 40 ºC had less than five min left in the 60-min SIM.

6.6.4 Conclusion

Participants experienced greater physiological and psycho-physical strain during FIXED in a HOT environment that caused volitional exhaustion in nine participants and heavy fatigue that substantially challenged homeostasis in all participants. Athletes reduced self-paced effort and sprint power during FREE to a greater extent in HOT. Most cardiovascular responses indicated that competition for perfusion was larger in HOT and may have contributed to greater metabolic strain similar to the 5-min TT in HOT and indicated during elite triathlon racing in HOT. This was influenced by elevated T_C, lower prefrontal cortex oxygen saturation, and a deterioration in affective state during FIXED HOT that forced some participants to stop (volitional exhaustion) and all participants to pace themselves conservatively during FREE HOT to maintain homeostasis.

7 DOES HEAT ACCLIMATION IMPROVE PERFORMANCE AND CEREBROVASCULAR FUNCTION IN TEMPERATE AND HOT ENVIRONMENTS?

7.1 Chapter Introduction

This chapter extends chapters three, five, and six by incorporating a heat acclimation protocol into the daily training routine of endurance athletes. In chapter three an observational field study of elite triathletes racing in different environments demonstrated that performance deteriorated considerably, physiological strain was greater, and athletes' ability to pace effectively was impaired in hot compared with temperate environments. Chapter five compared maximal, short aerobic time trials in temperate and hot laboratory environments to determine how strongly cardiovascular strain impaired athletic performance in the heat. Chapter six tested highly-trained athletes' performances using a race simulation that mimicked the cycling demands of an ITU triathlon race in a temperate and hot environment to examine the thermoregulatory and cerebrovascular performance limitations. The present chapter was designed as a training intervention study using heat acclimation to prepare highly-trained athletes for competition in different environments. Specifically, we examined whether heat acclimation could attenuate heat-induced cerebrovascular strain and whether this had any impact on heat-related impairment of endurance exercise performance. Therefore, we replicated the stress intensity and profile of simulated triathlon (cycle section) racing, in both temperate and hot conditions before and after heat acclimation or a control intervention, and monitored cardiovascular, cerebrovascular, thermoregulatory and perceptual strain and performance. A secondary purpose was to examine whether heat acclimation could enhance such performance in temperate (18 °C) conditions, given that (i) this issue remains equivocal, and (ii) almost no studies have undertaken controlled-trial comparisons of matched *relative* exercise loads (e.g., matched %HRR or RPE) during heat versus temperate conditioning, i.e., which is what an athlete or worker would do in most real-life scenarios.

7.2 Abstract

The purpose of this study was to compare heat acclimation (HA) with a counter-balanced control (CON) protocol for performance power and physiological responses to a draft-legal triathlon cycling race simulation, focussing on cerebrovascular strain. Seven endurance-trained male triathletes and cyclists [23 (9) years, 183 (9) cm, 76 (13) kg, 65 (14) $mL \cdot kg^{-1} \cdot min^{-1}$ $\dot{V}O_2$ peak) performed a step-incremental cycling test to determine ventilatory thresholds, lactate thresholds, Wmax, and $\dot{V}O2$ peak. Participants performed 2 pre-intervention trials (PRE) in TEMP (18 ˚C, 56% RH, 1.2 kPa PH_2O, 4.0 $m \cdot s^{-1}$ v_a) and HOT (33 ˚C, 62% RH, 3.1 kPa PH_2O, 4.0 $m \cdot s^{-1}$ v_a) then 60-70 min/d of conditioning for 10 d in an environment chamber as HA [38 ˚C, 59% RH, bath temperature 41 ˚C] and CON [20 ˚C, 49% RH, bath temperature 31 ˚C] followed by a 3-wk washout, then crossed over. Participants lay in a bath for ~12 (HA) or ~16 (CON) min, then transitioned to an ergometer to cycle at self-paced intensity for the remaining time to achieve matched relative environmental and work intensity stress. BV was measured 1 d before and after each intervention. 60-min SIM was the same protocol described in Chapter 6. Data were compared between environment (TEMP, HOT) and conditioning intervention (PRE, CON, HA) or between conditioning intervention and time in each environment using two-way RM ANOVA and paired t-tests. During 10-d conditioning, participants completed 66% more work in CON ($P = 0.004$) but experienced greater thermal strain and perceived exertion in HA conditioning ($P < 0.001$). Performance in 60-min SIM tended to improve after HA in that (i) 5/7 participants completed HOT compared with 2/7 in PRE and 3/7 after CON, (ii) power in HOT averaged ~120% higher for FREE paced efforts after HA compared to 55% higher after CON ($P = 0.04$), and (iii) power in TEMP averaged ~20% higher for FREE sprints after HA compared to 7% higher in CON ($P = 0.006$). HA compared with PRE and CON conditioning reduced T_{Head} during TEMP ($P = 0.02$), and HOT ($P = 0.04$), but T_C was not reliably lowered. Brain HHb in TEMP was similar at $FIXED_{First}$, then consistently lower after HA compared with PRE and CON conditioning ($P = 0.01$). HA improved thermal sensation at Rest in TEMP and improved feeling, comfort and thermal sensation at $FIXED_{Mid}$ in HOT ($P = 0.01$). Conclusions: Ten days of HA reduced thermal and affective strain indicated by reduced T_{Head} in TEMP and HOT combined with improved affective state.

7.3 Introduction

Structured heat acclimation has facilitated adaptation in endurance-trained athletes, improving thermoregulation and performance while reducing thermal strain in warm and hot environments (Garrett et al., 2011; Karlsen et al., 2015b; Keiser et al., 2015a; Périard et al., 2015; Racinais et al., 2012; Zurawlew et al., 2016). Further, heat acclimation may (Hue et al., 2007; Lorenzo et al., 2010; Scoon et al., 2007) or may not (Karlsen et al., 2015a; Keiser et al., 2015a) augment performance in temperate environments (Corbett et al., 2014; Minson and Cotter, 2016a, 2016b, Nybo and Lundby, 2016a, 2016b). Considerable information has accumulated about the adaptations generated by heat acclimation. Individuals adapt physiologically with decreased exercising T_C (Sawka et al., 2001), T_{Skin} (Roberts et al., 1977), HR (Sawka et al., 1985), [La] (Febbraio et al., 1994a; Lorenzo et al., 2010; Young et al., 1985), and muscle glycogen utilisation (King et al., 1985; Kirwan et al., 1987). Other physiological adaptations include increased sweat rate (Dill et al., 1938; Fox et al., 1964; Nielsen et al., 1997), BV (Sawka and Coyle, 1999), PV (Nielsen et al., 1993; Patterson et al., 2014; Senay et al., 1976), heat shock proteins (Amorim et al., 2015; Kuennen et al., 2011; Marshall et al., 2007), SBP and DBP (Sawka et al., 2011a). Furthermore, physiological efficiency improves, including metabolic efficiency (Horowitz, 2011; Kodesh and Horowitz, 2010), $\dot{V}O_{2max}$ (Lorenzo et al., 2010; Sawka et al., 1985) vascular sensitivity (Lorenzo and Minson, 2010), and thermotolerance (Moseley, 1997; Sandström et al., 2008). Psycho-physically, individuals report better feelings of thermal sensation and lower perceived effort (Saat et al., 2005; Zurawlew et al., 2015). One physiological aspect that has been neglected is the effect of heat acclimation on heat-induced impairment of cerebrovascular function during exercise in HOT or TEMP environments, which was an important aspect of the present study.

Exercise in hot environments powerfully influences cerebral autoregulation, brain blood flow and brain oxygenation (Chapter 6; Ide and Secher, 2000; Nybo and Rasmussen, 2007; Paulson et al., 1990; Rasmussen et al., 2007, 2010; Secher et al., 2008). Two counter-balanced heat acclimation studies demonstrated superior brain blood flow velocity measured at the middle cerebral artery after heat acclimation but showed either no difference in mean arterial blood pressure between groups at rest (Fujii et al., 2015) or significantly greater mean arterial

blood pressure for heat acclimated participants during a 30-min cycling time trial in 38 ˚C (Keiser et al., 2015a). Despite higher brain blood flow after heat acclimation at rest or during exercise, brain oxygenation and performance during exercise in a hot environment were not improved for participants after heat acclimation compared with control conditioning (Keiser et al., 2015a). Conversely, brain blood flow may be less important than brain oxygen extraction to maintain brain metabolism (Trangmar et al., 2014b) and performance in a hot environment (Chapter 6); a physiological response that requires further investigation.

Performance testing after heat acclimation or acclimatisation has typically used time trials to determine performance (Garrett et al., 2011; Karlsen et al., 2015a; Keiser et al., 2015a; Lorenzo et al., 2010). Time trials have validity for athletes competing in events where they have autonomy for their pacing strategy to achieve the highest average power (e.g., individual time trial races, pool swimming, rowing, sprint kayaking), but it does not account for athletes competing in events that require stochastic power defined by best average effort punctuated with maximal efforts to tactically win the race (e.g., track running, road cycling, open water swimming). Heat influences power output in accordance with volume of exposure, enhancing isolated sprint efforts (Asmussen and Bøje, 1945; Ball et al., 1999; Faulkner et al., 2013) but hindering prolonged endurance efforts (Kayser, 2003; Nybo and Nielsen, 2001a; Nybo et al., 2011; Tucker et al., 2004, 2006b). Endurance athletes also compete in outdoor environments with highly variable conditions, and may inadvertently or deliberately seek to obtain physiological benefits from heat acclimation preparation for performance on a cooler race day. Given the time and effort invested by athletes and sports teams in heat preparation it is pertinent to investigate the magnitude and timing of performance and physiological benefits that may be transferred to temperate and cool environments.

Heat acclimation research has used mostly fixed intensity protocols based on workload, %$\dot{V}O_2$, HR, or RPE for at least 90 minutes, that often raises and then maintains core body temperature at ~38.5 ˚C in an environmentally controlled room for 5 to 14 consecutive days (Fox et al., 1964; Garrett et al., 2012b; Lorenzo

et al., 2010). However, athletes train at self-paced intensity, especially during the initial training sessions in the heat. One published study has documented self-paced heat acclimation (Armstrong et al., 1986), a protocol that may allow athletes to control the cumulative stress from exercise and heat to optimise adaptation and anticipatory pacing (Racinais et al., 2015; Tucker, 2009). Furthermore, a more accessible heat acclimation protocol needs to be refined to prepare elite and highly-trained endurance athletes living and training in temperate environments for competition using resources they already have (e.g., bathroom, bath tub, indoor trainer, bike). Many athletes require improvised heat acclimation to prepare to compete in hot environments because laboratory-based acclimation and acclimatisation are not financially or practically feasible, irrespective of which protocol is more or less effective (Buchheit et al., 2011; Racinais et al., 2015).

The purpose of this study was to use heat acclimation in conjunction with athletes' normal training to prepare them for physiologically-monitored cycling race simulation performances in temperate and hot environments. Participants completed heat acclimation or control conditioning protocols in a cross-over design to elucidate the physiological (especially cerebrovascular) adaptations and performance effects. We hypothesised that a 60-min triathlon cycle race simulation that replicated the performance (stochastic power outputs including fixed pace and self-paced sections) and environmental (temperature, humidity, and wind speed) race factors would have detrimental performance, cerebrovascular, cardiovascular, and psychophysical impacts, including heat-related exhaustion for participants before and after control conditioning, but that heat acclimation would lessen these effects in temperate and especially hot environments.

7.4 Methods

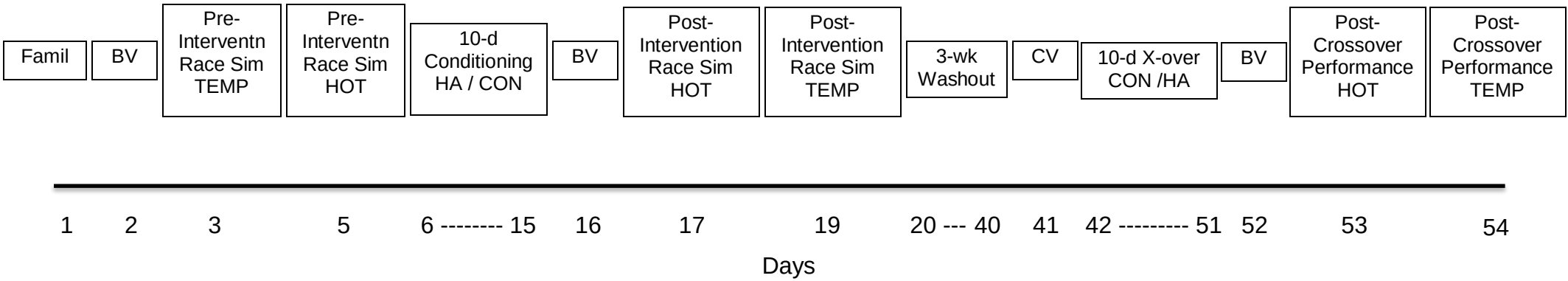
7.4.1 Participants

Seven endurance-trained male triathletes and cyclists [23 (9) years, 183.1 (9.4) cm, 75.9 (12.5) kg, 65 (14) $mL.kg^{-1}.min^{-1}$ $\dot{V}O_2$ peak] gave informed consent to participate.

7.4.2 Experimental Design

The study was a repeated-measures design of seven 60-min SIM trials: one familiarisation, two PRE trials (TEMP and HOT), then TEMP and HOT trials after 10-d regimes of a control intervention (CON) or heat acclimation (HA), undertaken in a randomised crossover. A three-week washout followed HA, before CON. The design is illustrated in Figure 7.1.

PRE trials were completed in TEMP (18.0 ± 0.1 ˚C, 56 ± 8% RH, 1.18 PH_2O, 4.0 $m{\cdot}s^{-1}$ v_a; day three) and HOT (33.1 ± 0.1 ˚C, 62 ± 2% RH, 3.10 kPa PH_2O, 4.0 $m{\cdot}s^{-1}$ v_a; day five). Thereafter participants were allocated to HA or CON interventions and completed conditioning for 60-70 minutes at self-paced intensity for ten consecutive days in an environment chamber to ensure HA and CON conditioning were matched for relative work intensity. Exercise intensity during conditioning was self-paced, with one exception: in TEMP power output was limited below VT1 to ensure the training load was comparable. Blood volume was assessed after each intervention by CO dilution (days 16 and 52). Two days after each intervention (days 17 and 53) athletes completed a performance test in HOT, had a rest day, then completed a performance test in TEMP (days 19 and 54). A three-week washout period followed (days 20 to 40) before athletes completed a cerebrovascular challenge (day 41) and began the crossover intervention the next day. Following the crossover intervention the same post-intervention tests were completed in the same order (Figure 7.1).



Famil: Familiarisation trial and $\dot{V}O_2$ peak test
BV: Blood volume measured by CO Rebreathing technique
Race Sim: 60-minute triathlon race simulation in an environment chamber
HA: Heat acclimation protocol – 10-minute bath (~41 ˚C) followed by cycling for 50-70 minutes (38 ˚C, 60% RH, 0 $m.s^{-1}$ wind speed)
CON: Control protocol – 10-minute bath (~31˚C) followed by cycling for 50-70 minutes (20˚C, 40% RH, 0 $m.s^{-1}$ wind speed)
CV: Active and passive cerebrovascular CO_2 challenge (33 ˚C, 60% RH, 0 $m.s^{-1}$ wind speed)

Figure 7.1: Experimental timeline for each athlete.

7.4.3 Procedures

7.4.3.1 Preliminary Procedures

Participants completed a familiarisation trial and $\dot{V}O_2$ step-incremental test in TEMP on day one. Blood volume was assessed on day two.

7.4.3.2 Pre-Test Procedures

Participants completed the preliminary procedures followed by the resting CO_2 challenge and exercising CO_2 challenge pre-test procedures..

7.4.3.3 Test Procedures

Participants completed the test and post-test procedures described in Chapter 6.

7.4.3.4 Heat Acclimation and Control Conditioning

The athletes completed either heat acclimation or control conditioning training sessions for 10 consecutive days (Figure 7.2).

In heat acclimation, athletes wore a heart rate monitor and inserted a thermistor, then entered the environment chamber (38.2 ± 0.3 ˚C, 59 ± 14% RH). Conditioning started with a passive heat load, where they lay in a bath filled with 40.8 ± 1.1 ˚C water to the top of the shoulders and covered by foam mats for insulation. Participants stayed until volitional exhaustion (12:36 ± 4:00 m:ss) then recovered on a chair while T_C, T_{Skin}, HR, BP, thermal comfort, thermal sensation, and RPE were measured. Thereafter they put on cycling shoes and began exercising at a self-selected intensity on a cycle ergometer. Athletes were asked to cycle at any power output they felt was sustainable for the conditioning bout. The duration was at least 60 min for the initial training sessions, extending to 70 min, then 80 min for the last 2-3 sessions to graduate the heat acclimation stimulus. Training session time was matched within each participant between conditioning protocols. If they could not cycle participants sat on a chair, and in some cases lay on the floor until they were able to cycle again. Athletes were encouraged to refrain from drinking but could consume water or sports drink *ad*

libitum. Every five min HR and either bath temperature or cycling power output were recorded, T_C was also recorded from the rectal thermistor in six participants and oesophageal thermistor for one participant. Skin temperature was measured before entering the chamber, immediately after leaving the bath, and at 15-min intervals during exercise, using an infrared thermometer (Braun, Kronberg, Germany). Thermal comfort, thermal sensation, ambient temperature and relative humidity were measured every 15 min throughout. Blood pressure was measured manually before entering the chamber, after leaving the bath, and at the end of the trial in the chamber. Athletes were required to spend at least 60 min and no more than 80 min in the environment chamber each day.

The control conditioning bouts followed the same protocols and measurements as heat acclimation. The only differences were the bath temperature (31.4 ± 1.7 ˚C) and environment chamber settings (20.1 ± 1.1 ˚C, 49 ± 7% RH). Athletes stayed in the bath for 16:12 ± 2:42 m:ss as volitional exhaustion was not anticipated/realistic. They were also asked to maintain cycling power at a self-selected intensity below VT1, with the aim of matching the relative intensity anticipated during HA conditioning, limit the training stimulus created by the workload, and reduce the thermal stimulus driven by the workload without convective cooling.

7.4.4 Measurements

The same measurements and equipment were used as Section 4.4.4, however, the metabolic cart that measured breath-by-breath respiration and finger capillary oxygen saturation were removed as all relevant variables were recorded using respective ventilation and gas analysers (AEI, Pittsburgh, PA) connected to an A-D converter (Powerlab, ADInstruments) interfaced with Chart software (ADInstruments). Feeling scale (Hardy and Rejeski, 1989) was included in the psycho-physical assessment prompted by the question “How do you feel about exercise?”.

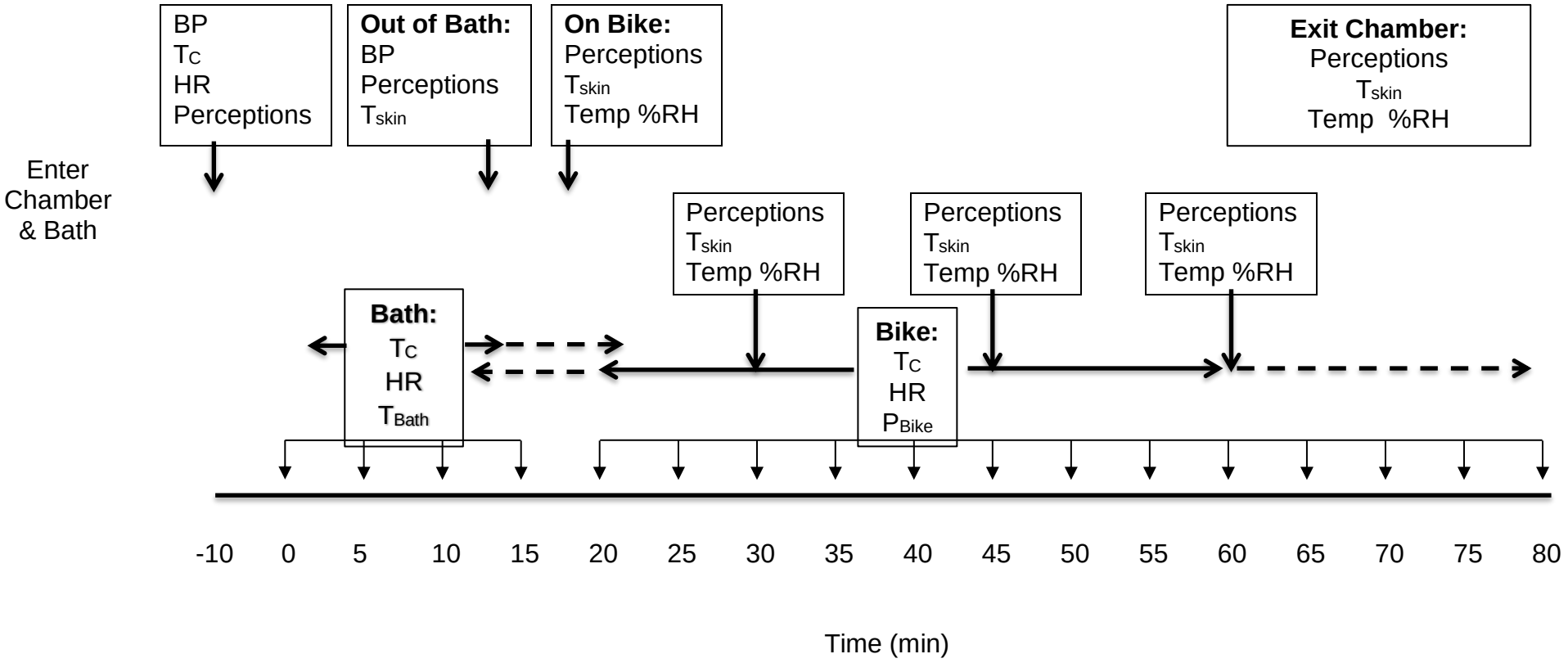


Figure 7.2: Timeline for conditioning bouts during heat acclimation and control.

7.4.5 Data Analyses

7.4.5.1 Calculations

Calculations and data filtering followed the same procedures described in Chapter 6.

7.4.5.2 Statistical Analyses

Participant responses to 10 d of CON and HA conditioning were analysed across condition by one-way RM ANOVA with the Geisser-Greenhouse correction, and type-1 errors were controlled using Tukey post-hoc pairwise comparisons. Cycling completion for 60-min SIM was compared between conditioning interventions (PRE, CON, HA) and environments (TEMP, HOT) using Survival Analysis. Total work, cerebrovascular reactivity, and BV changes were compared between conditioning interventions and environments using two-way RM ANOVA utilising the same methods detailed in Section 6.4.5.1. Cycling power, body temperatures, $P_{ET}CO_2$, BP, HR, cerebrovascular responses and psycho-physical perceptions were analysed using two-way RM ANOVA. Tukey or Holm-Sidak pairwise post-hoc comparisons or paired t-tests were used to compare conditioning intervention (PRE, CON, HA) with different time points ($FIXED_{First}$, $FIXED_{Last}$, $FREE_{First}$, and $FREE_{Last}$) separately in TEMP and HOT environments. Based on the survival analysis, comparisons between variables were made at the same time point (e.g., a participant who completed 18 min of FIXED would be analysed as $FIXED_{Last}$ at 18 min in TEMP and HOT for each condition).

7.5 Results

7.5.1 Control and Heat Acclimation Conditioning

Conditioning sessions were of similar duration in CON and HA (~60 min, P = 0.76). Total work completed during conditioning trials in CON was 66% higher than in HA [324 (72) vs. 195 (81) kJ, CI: -68 to -190, P = 0.004] but were similar in total duration [101 (8), vs. 99.5 (7.7) h, P = 0.11). Conversely, T_C was lower in conditioning for CON than HA by 0.94 °C (CI: 0.56 to 1.32, P = 0.02) and T_{Skin} was 6.7 °C lower (CI: 6.0 to 7.4, P < 0.001). Participants also felt better (CI: 2 to 5, P = 0.005), less discomfort (CI: -5 to -3, P = 0.001), and cooler (CI: -3 to -2, P < 0.001), tending to perceive that exercise intensity was lower (CI: -6 to 0.1, P = 0.06) despite greater total work output (Figure 7.3). Cycling economy was similar between conditioning protocols for $\dot{V}O_2$ and oxygen pulse at VT1 and VT2. The mean SBP within a conditioning bout tended to increase from the first to last conditioning sessions during CON (P = 0.09) but not HA (P = 0.44). Participants tended to feel cooler at the last compared with the first HA conditioning session (Start: Hot to Warm; End: Very Hot to Hot; P = 0.09).

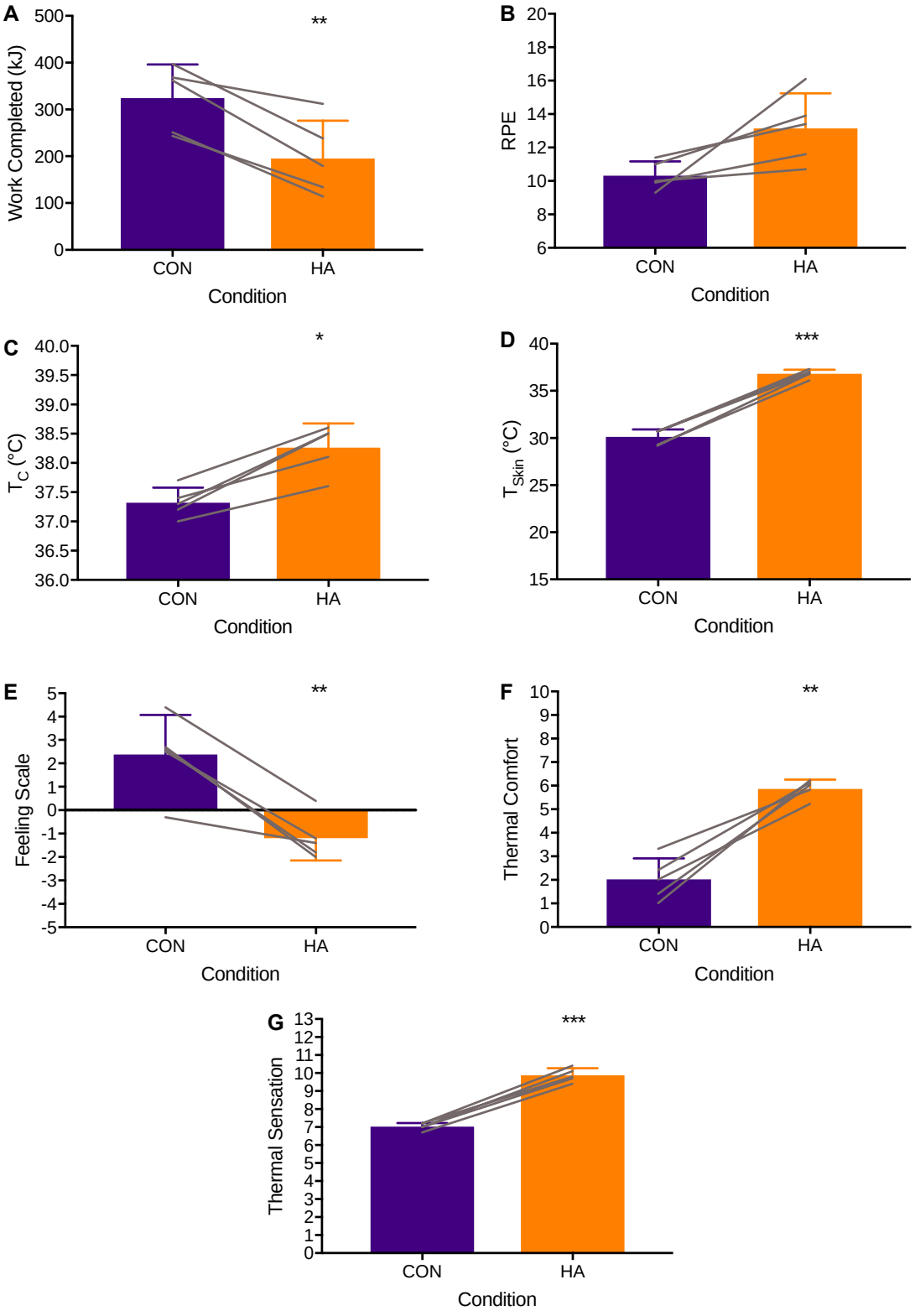


Figure 7.3: Total work completed (A), ratings of perceived exertion (B), core temperature (C), average skin temperature (D), Feeling Scale (E), Thermal Comfort (F), and Thermal Sensation (G) during 10 d control conditioning (purple), or heat acclimation (orange). Temperature effect: * $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$.

7.5.2 Cycling Race Simulation Power

7.5.2.1 Survival Analysis

All seven participants undertook the TEMP and HOT trials for PRE and HA; one participant was unable to complete CON due to sickness during conditioning. By design, the work intensities used for the FIXED section of the race simulation (60-min SIM) were individualised and difficult to complete - at least in HOT - before switching to FREE. All participants successfully completed the full 40 minutes of the FIXED section of the TEMP trials for PRE, CON, and HA. In the HOT trials, five participants could not complete FIXED for PRE, four participants could not complete FIXED for CON, and two participants could not complete FIXED for HA (Figure 7.4).

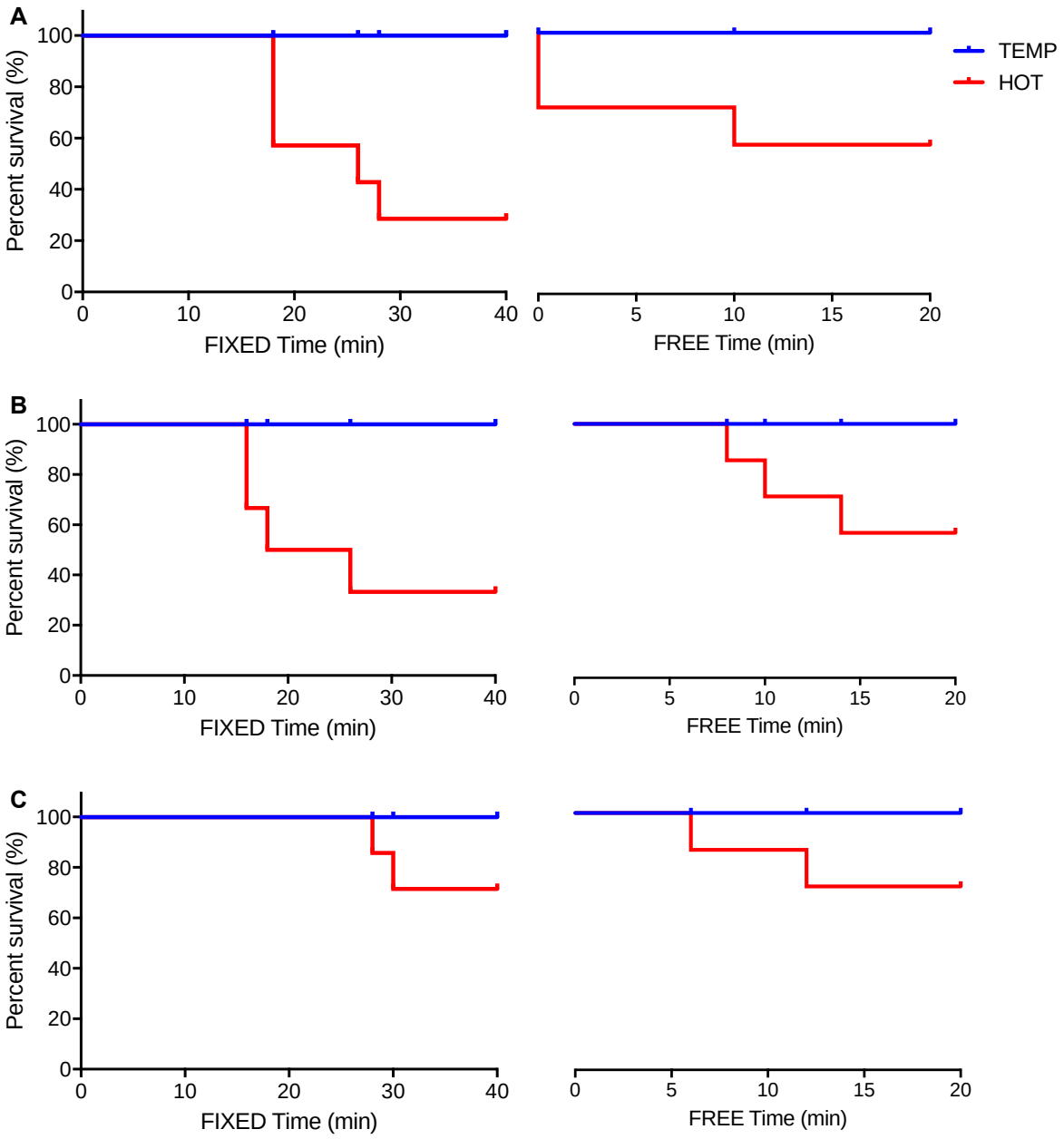


Figure 7.4: Survival analysis during 40 min at FIXED and 20 min at FREE power outputs for pre-intervention trials (A), post-control conditioning intervention (B), and post-heat acclimation intervention (C). The full protocol was unable to be completed in HOT by five of the seven participants in PRE, four in CON and two in HA.

7.5.2.2 Total Work

Less work was completed in HOT (Temperature effect: P = 0.03, Interaction effect: P = 0.18; Figure 7.5), tending to be less after PRE (by 38%, P = 0.008) and CON (by 37%, P = 0.008), but similar after HA conditioning (by 14% less, P = 0.31).

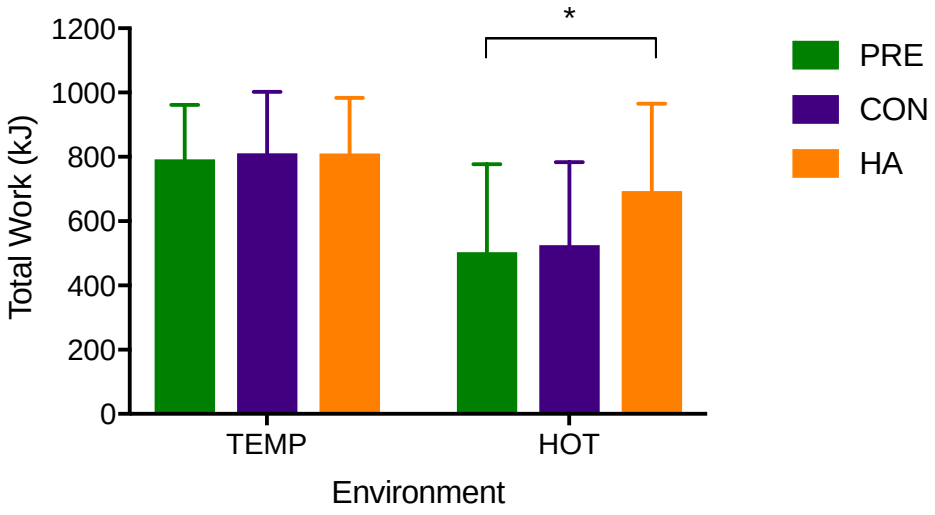


Figure 7.5: Total work completed in cycling simulations in temperate or hot environments for each condition: pre-intervention (green), after 10 d control conditioning (purple), and after 10 d heat acclimation (orange). Temperature effect: *P < 0.05.

7.5.2.3 Race Simulation Performance After Heat Acclimation

Temperate Environments: Paced-effort cycling power was not reliably higher after HA than CON conditioning or relative to PRE (Condition effect: P = 0.16; Interaction effect: 0.27; Figure 7.6A), and declined across 60-min SIM for all conditioning protocols during FREE paced efforts (by 15%, 0.47 $W \cdot kg^{-1}$ FFM, P = 0.01).

Sprint cycling power became progressively greater after HA conditioning compared with PRE and CON (Interaction effect: P = 0.04; Figure 7.6B). Sprint power was 19% higher at $FREE_{Last}$ for HA compared with PRE and CON (2.11 and 2.20 $W \cdot kg^{-1}$ FFM, P ≤ 0.008) and increased from $FIXED_{First}$ to $FREE_{Last}$ by 66% (PRE), 71% (CON) and 101% (HA) (4.47 to 6.71 $W \cdot kg^{-1}$ FFM, P < 0.001).

Hot Environments: Paced-effort power was greater after HA conditioning compared with PRE and CON at $FREE_{First}$, and greater by similar amounts after HA and CON conditioning at $FREE_{Last}$ (Interaction effect: P = 0.006; Figure 7.6A). Paced efforts were higher after HA compared with PRE and CON at $FREE_{First}$ [by 170% and 90% (1.69 and 1.28 $W \cdot kg^{-1}$ FFM) respectively, P ≤ 0.001]. Compared to FIXED, paced-effort power declined during FREE paced efforts (by 45%, 1.39 $W \cdot kg^{-1}$ FFM, P = 0.01).

Sprint cycling power was not altered by CON or HA conditioning, and was similar throughout FIXED and FREE (Interaction effect: P = 0.59; Condition effect: 0.53; Time effect: 0.48; Figure 7.6B).

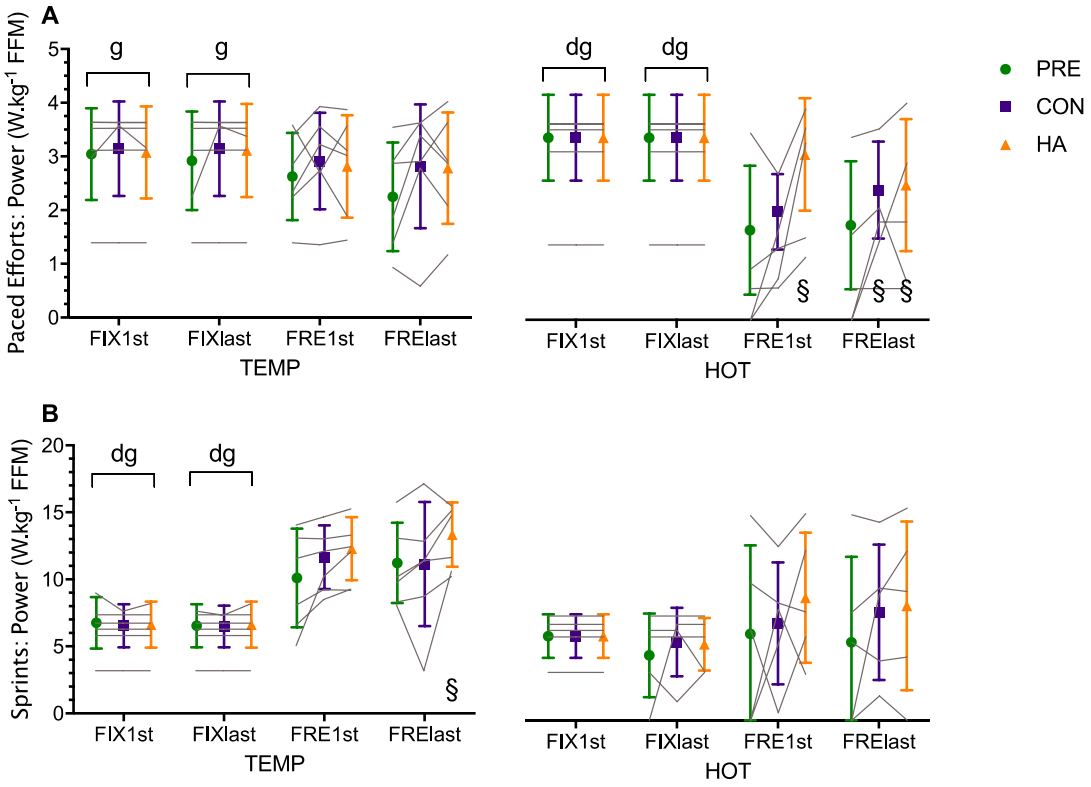


Figure 7.6: Power output at first 2-min at pre-set power output (FIX1st), last 2 min at pre-set power output (FIXlast), first 2min at self-selected power output (FRE1st), and last 2 min at self-selected power output (FRElast) in temperate and hot environments during the race simulation before or following control or heat acclimation conditioning. Note that the FIXlast is mostly earlier during exercise in HOT, especially for PRE and CON (see Figure 7.4). Mean ± SD in coloured symbols, individual responses in grey lines . Time effect: P < 0.05 at: [d] FRE1st, [g] FRElast; Condition effect: [§] P < 0.05 PRE compared to CON or HA.

7.5.3 Body Temperatures

7.5.3.1 Core Temperature

Core temperature at rest or during exercise was not reliably lowered by HA relative to CON conditioning or PRE, in either test environment (Interaction effect: $P > 0.61$; Condition effect: $P > 0.21$; Figure 7.7). The T_C increased during FIXED in TEMP and HOT (both by 1.3 °C, $P < 0.001$) but was unchanged during FREE ($P > 0.99$).

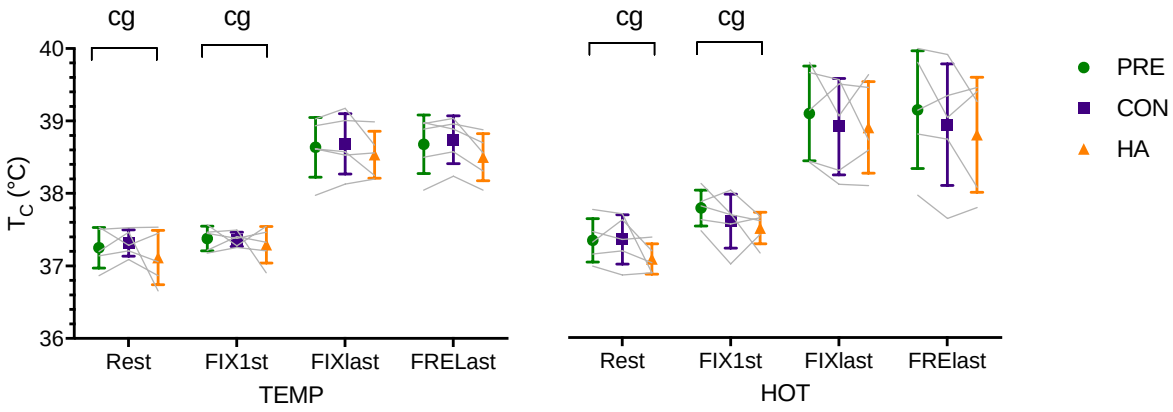


Figure 7.7: Core temperature at rest, first 2-min at pre-set power output (FIX1st), last 2 min at pre-set power output (FIXlast), first 2min at self-selected power output (FRE1st), and last 2 min at self-selected power output (FRElast) in temperate and hot environments during the race simulation before or following control or heat acclimation conditioning. Mean ± SD in coloured symbols, individual responses in grey lines . Time effect: $P < 0.05$ at: ^{c} FIXlast, ^{g} FRElast.

Participants who could not maintain FIXED power output experienced the highest rate of change in T_C. During the FREE section, T_C plateaued for participants in TEMP and HOT environments. Participants also had T_C limits that they consistently reached at volitional exhaustion, i.e., if they stopped before 60 min (Figure 7.8).

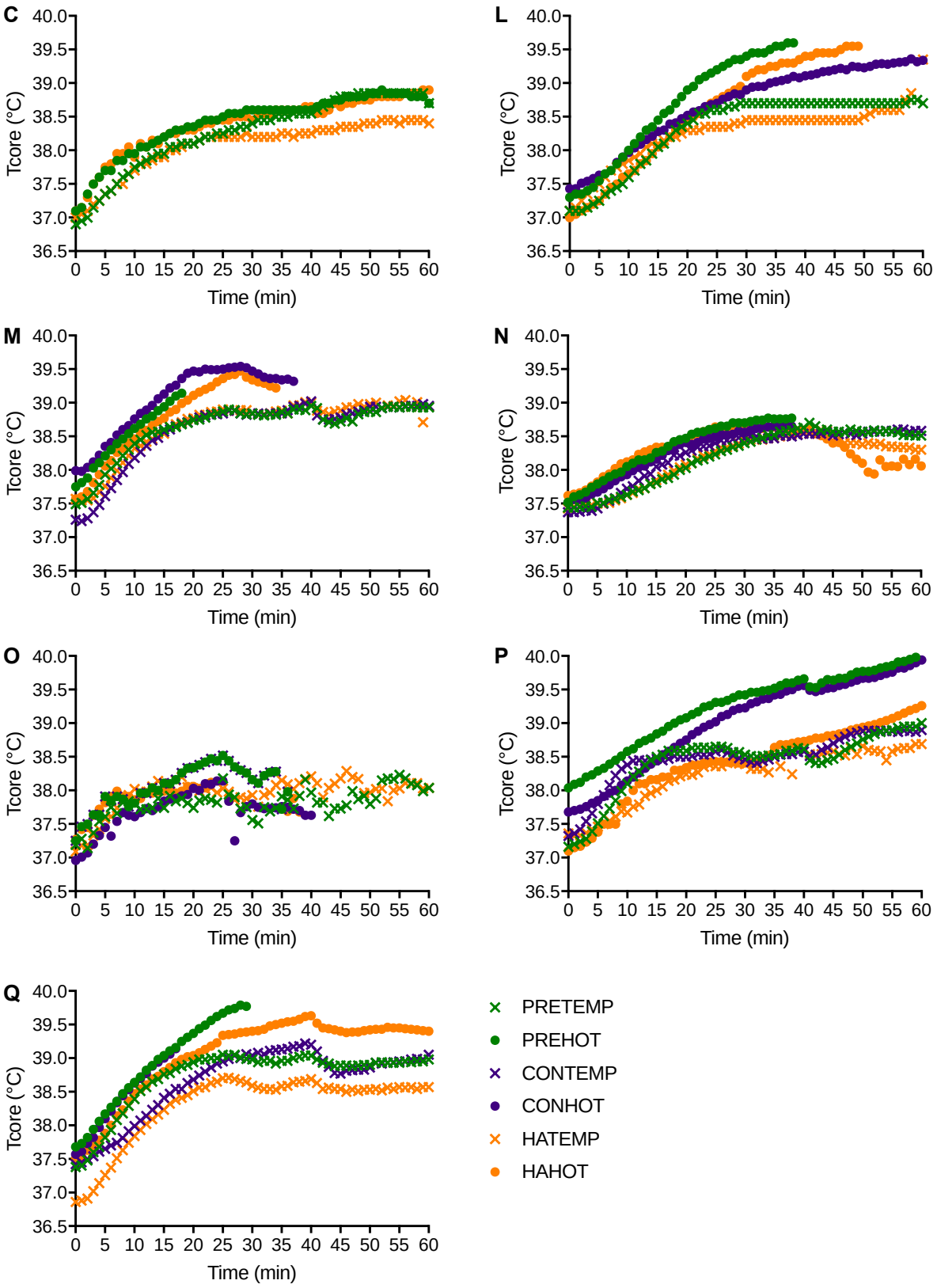


Figure 7.8: Individual core temperature responses over the duration of the race simulation for each condition in temperate or hot environments. Letters C to Q denote each participant.

7.5.3.2 *Forehead Skin Temperature*

In TEMP, T_{Head} was substantially lower after HA conditioning compared with PRE and CON at $FIXED_{Mid}$ (by 2.0 and 2.9 °C respectively, $P < 0.03$), and decreased across 60-min SIM (PRE: 4.1 °C, CON: 5.3 °C, HA: 6.5 °C, $P < 0.001$; Interaction effect: $P = 0.02$). In HOT, T_{Head} was lower after HA conditioning compared with PRE and CON at $FIXED_{Last}$ and $FREE_{Last}$ (by ~1.3 °C, $P \leq 0.03$) and tended to decrease across 60-min SIM after HA conditioning (Interaction effect: $P = 0.09$; Time effect: $P = 0.12$; Figure 7.9A).

7.5.3.3 Mid-Axilla Skin Temperature

In TEMP, T_{Axilla} was increased or decreased by HA and CON conditioning compared with PRE at different time points, but was similar for CON compared with HA across 60-min SIM (Interaction effect: $P = 0.006$; Figure 7.9B). At rest T_{Axilla} was higher after HA (by 1.5 °C, $P = 0.02$) and CON (by 2.1 °C, $P = 0.001$), remained higher at $FIXED_{Mid}$ after CON (by 1.3 °C, $P = 0.04$), and was lower at $FIXED_{Last}$ after HA (by 1.3 °C, $P = 0.05$). In HOT, T_{Axilla} was lower after HA conditioning than PRE and CON (Condition effect: $P = 0.05$, Interaction effect: $P = 0.25$; Figure 7.9B) at $FIXED_{Last}$ and $FREE_{Last}$ (by ~2.0 °C, $P < 0.03$), and for PRE, CON and HA decreased from Rest to $FIXED_{Mid}$ (by ~3.0 °C, $P < 0.01$) but was similar during FREE ($P > 0.99$).

7.5.3.4 Forearm Skin Temperature

The T_{Arm} tended to show the same effects as for T_{Axilla}, but these were not significant (TEMP: Interaction effect: $P = 0.08$, Condition effect: $P = 0.08$; HOT: Condition effect: $P = 0.07$, Interaction effect: $P = 0.99$; Figure 7.9C).

7.5.3.5 Finger Skin Temperature

In TEMP T_{Finger} at Rest increased after HA conditioning compared with PRE (by 2.7 °C, $P = 0.02$) but was not different between CON and HA conditioning, and decreased from Rest to $FIXED_{Mid}$ (by 5.3 °C, $P = 0.001$) but *increased* from $FIXED_{Mid}$ to $FREE_{Last}$ (by 3.4 °C, $P = 0.03$; Interaction effect: $P = 0.03$; Figure 7.9D). In HOT T_{Finger} decreased from Rest to $FIXED_{Mid}$ (by 3.2 °C, $P = 0.02$) and was not influenced by CON or HA conditioning (Interaction effect: $P = 0.87$, Condition effect: $P = 0.33$).

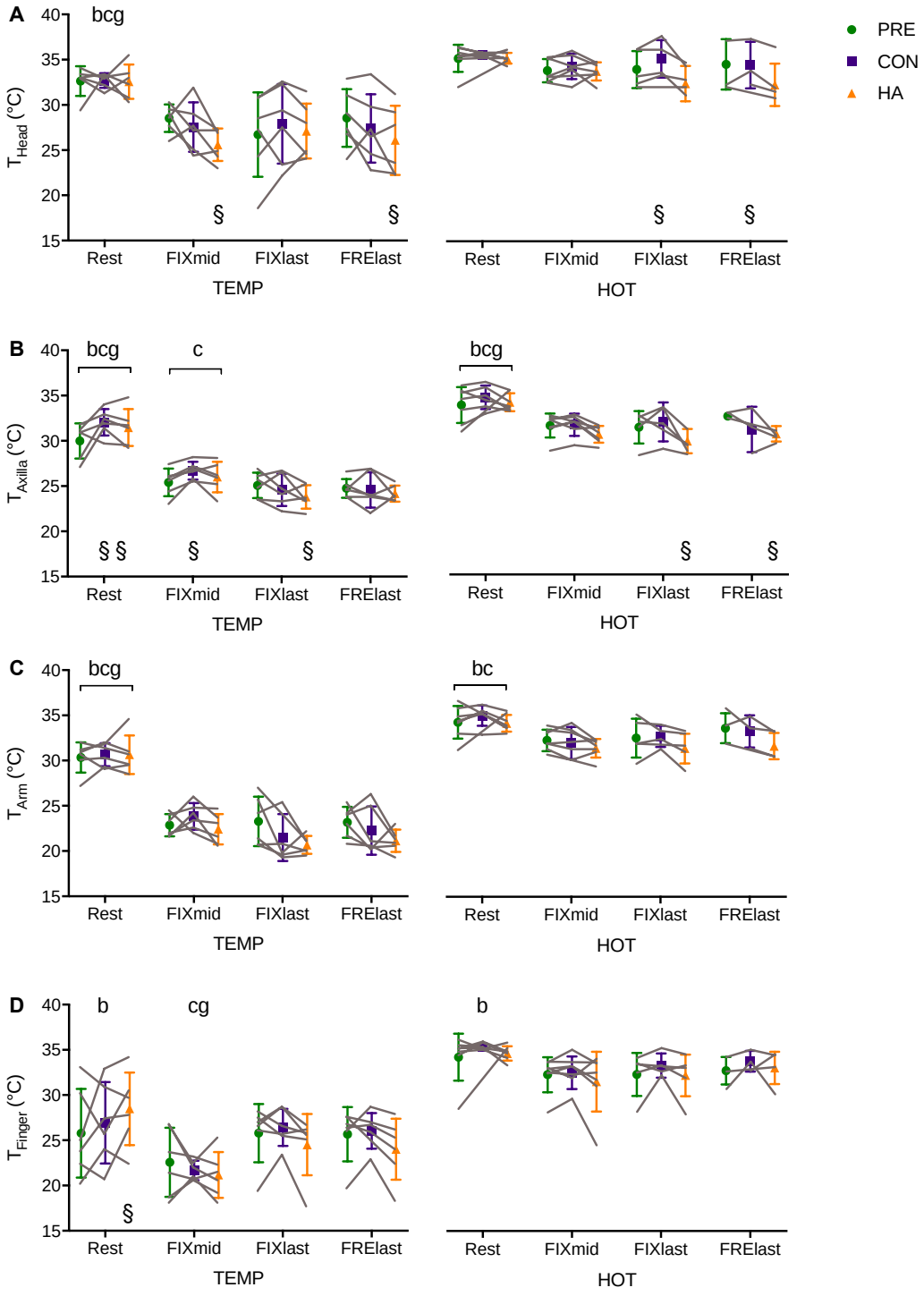


Figure 7.9: Skin temperature measured at the forehead (T_{Head}), mid-axilla (T_{Axilla}), forearm (T_{Arm}) and index finger (T_{Finger}) on the right side of the body at rest (Rest), 10 min at pre-set power output (FIX_{mid}), last 2 min at pre-set power output (FIX_{last}), and last 2 min at self-selected power output (FRE_{last}) in temperate (TEMP) and hot (HOT) environments during the race simulation before (PRE), or following either control conditioning (CON) or heat acclimation conditioning (HA). Mean ± SD in coloured symbols, individual responses in grey lines. Time effect: $P < 0.05$ at: ^{0} REST, ^{b} FIX_{mid}, ^{c} FIX_{last}, ^{g} FRE_{last}; Condition effect: $^{\S}P < 0.05$ for PRE compared with CON or HA.

7.5.4 Respiratory Responses

7.5.4.1 Partial Pressure of End-tidal Carbon Dioxide

In TEMP, $P_{ET}CO_2$ increased from Rest to $FIXED_{First}$ (by 4 mm Hg, P = 0.03) then decreased from $FIXED_{First}$ to $FREE_{Last}$ (by 13 mm Hg, P < 0.001) but was similar between conditions (Time effect: P < 0.001; Interaction effect: P = 0.38; Condition effect: P = 0.38; Figure 7.10).

In HOT, $P_{ET}CO_2$ increased from Rest to $FIXED_{First}$ (by 5 mmHg, P = 0.03), decreased across FIXED (by 12 mm Hg, P < 0.001), then was similar across FREE (P > 0.85), while CON tended to be lower than PRE at each time point (Time effect: P < 0.001; Interaction effect: P = 0.17; Condition effect: P = 0.40; Figure 7.10).

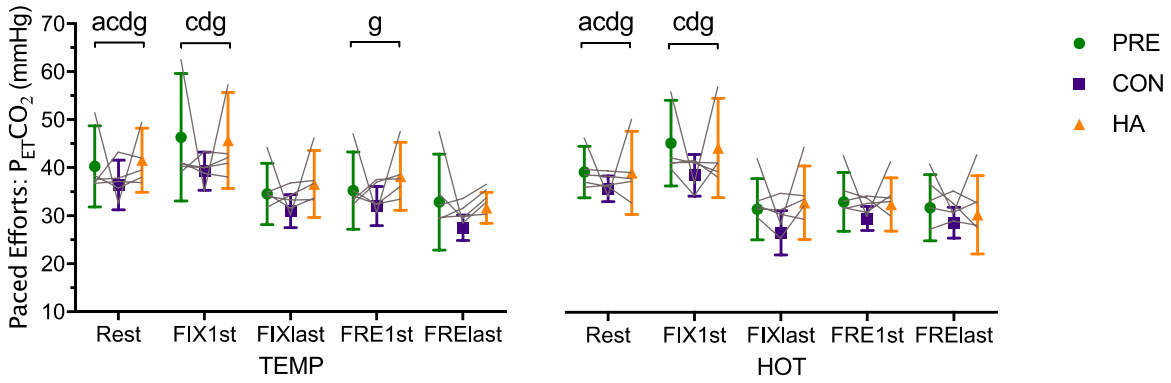


Figure 7.10: Partial pressure of end-tidal carbon dioxide at rest, first 2-min at pre-set power output (FIX_{1st}), last 2-min at pre-set power output (FIX_{last}), first 2-min at self-selected power ($FREE_{1st}$), and last 2-min at self-selected power output (FRE_{last}) in temperate and hot environments during cycling simulation before, or following control or heat acclimation conditioning. Mean ± SD in coloured symbols, individual responses in grey lines. Time effect: P < 0.05 at: ^{0} REST, ^{a} FIX_{1st}, ^{c} FIX_{last}, ^{d} FRE_{1st} ^{g} FRE_{last}.

7.5.5 Cardiovascular Responses

7.5.5.1 Blood Pressure

In TEMP and HOT, SBP and DBP were not influenced by CON or HA conditioning (Interaction effect: P > 0.34; Figure 7.11). In TEMP, the exercise-induced increase in SBP (by 20 mmHg, P = 0.02) tended to produce higher pressure after CON (Condition effect: P = 0.08), while DBP was similar across time for the 60-min exercise (Time effect: P = 0.60). In HOT, SBP increased by 21 mm Hg (P < 0.001) while DBP decreased by 10 mm Hg (P = 0.01) from Rest to $FIXED_{Mid}$. Data were not analysed beyond $FIXED_{Mid}$ because three only participants completed 60-min SIM after CON conditioning in HOT environments.

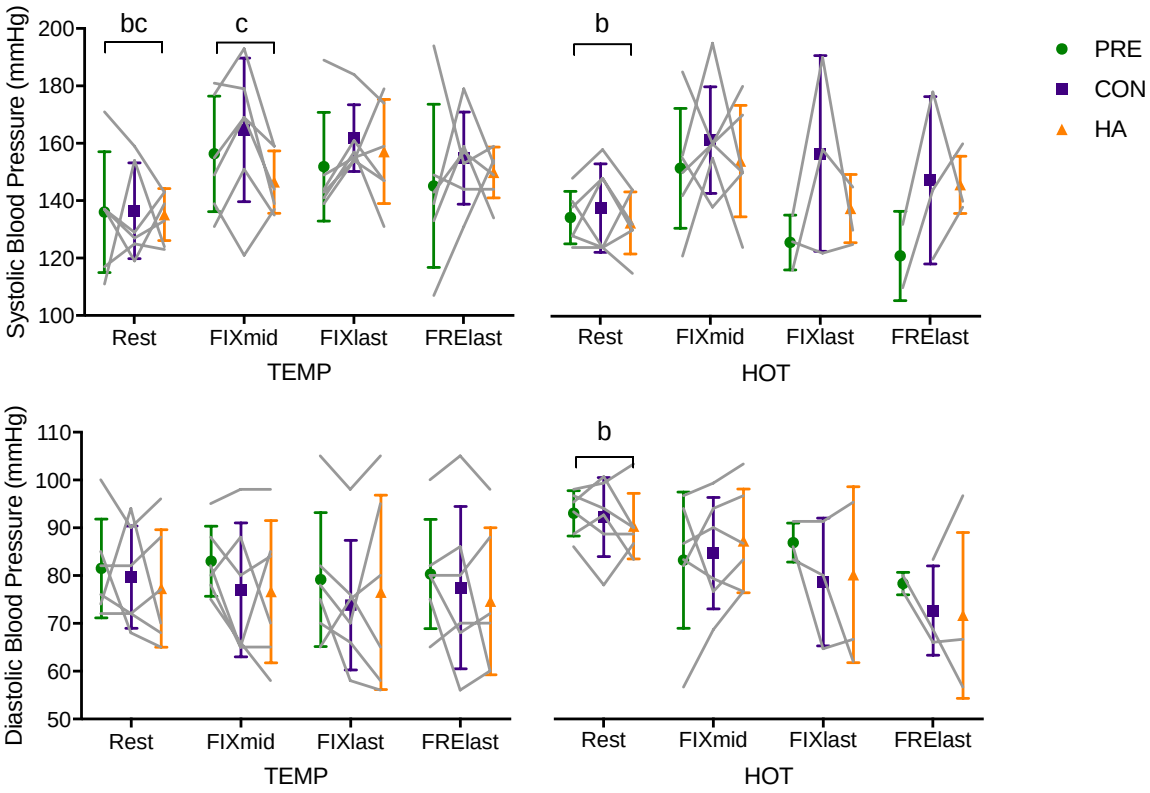


Figure 7.11: Systolic and diastolic blood pressure at rest, at 10-min pre-set power output (FIX_{Mid}), last 2-min at pre-set power output (FIX_{last}), and last 2-min at self-selected power output (FRE_{last}) in temperate and hot environments during cycling simulation before, or following control or heat acclimation conditioning. Analysis was not run in HOT for FIX_{last} and FRE_{last} as too many participants did not progress beyone FIX_{mid} (see Figure 7.4). Mean ± SD in coloured symbols, individual responses in grey lines. Time effect: P < 0.05 at: [b] FIX_{Mid}, [c] FIX_{last}.

7.5.5.2 Heart Rate

In TEMP HR was higher after CON conditioning and lower after HA conditioning compared with PRE (Interaction effect: P = 0.17; Condition effect: P = 0.01, Figure 7.12). Specifically, HR was lower after HA by 18 beats·min^{-1} at FIXED$_{First}$ (P < 0.001) and 11 beats·min^{-1} at FREE$_{First}$ (P = 0.04), but was higher after CON by 12 beats·min^{-1} at FREE$_{Last}$ (P = 0.03). Within each condition, HR drifted up by ~23 beats·min^{-1} across FIXED (P = 0.02) and thereafter remained similar during FREE (P > 0.99). Whereas, in HOT, HR drifted by 36 beats·min^{-1}across FIXED (P = 0.003) but tended to decrease across FREE (P = 0.18) and was similar for CON and HA compared with PRE (Interaction effect: P = 0.53; Condition effect: P = 0.44).

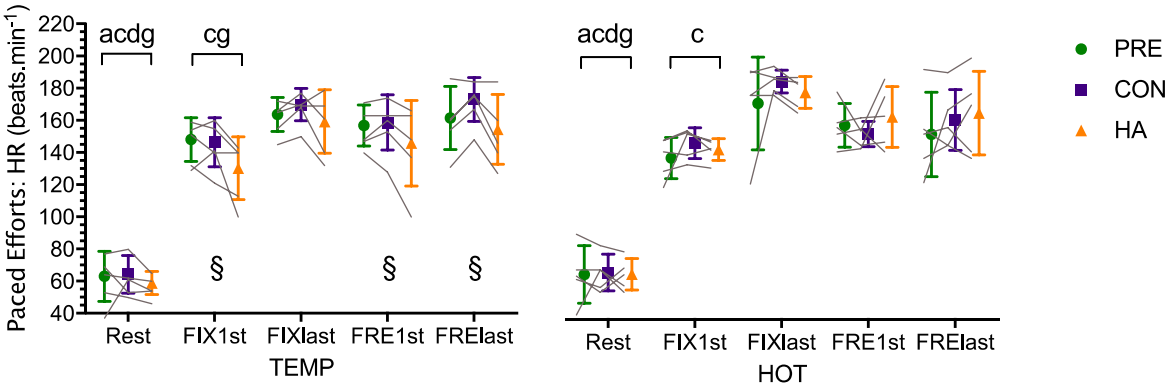


Figure 7.12: Heart rate at rest, first 2-min at pre-set power output (FIX$_{1st}$), last 2-min at pre-set power output (FIX$_{last}$), first 2-min at self-selected power (FREE$_{1st}$), and last 2-min at self-selected power output (FRE$_{last}$) in temperate and hot environments during cycling simulation before, or following control or heat acclimation conditioning. Mean ± SD in coloured symbols, individual responses in grey lines. Time effect: P < 0.05 at: [a] FIX$_{1st}$, [c] FIX$_{last}$, [d] FRE$_{1st}$ [g] FRE$_{last}$; Condition effect: [§]P < 0.05 for PRE compared with CON and HA.

7.5.6 Cerebrovascular Responses

7.5.6.1 Cerebrovascular Reactivity

Cerebrovascular hypercapnic reactivity (CVR_{hyper}) was differentially affected by the conditioning protocols (Interaction effect: P = 0.04, Figure 7.13A). Specifically, CVR_{hyper} was higher for TEMP compared with HOT at PRE (by 0.85 $cm \cdot s^{-1} \cdot mmHg^{-1}$, P = 0.03). Also, in TEMP, CVR_{hyper} was higher at PRE compared with HA (by 1.1 $cm \cdot s^{-1} \cdot mmHg^{-1}$, P = 0.01), tended to be lower after CON compared with PRE (P = 0.14), and was similar for HA compared with CON (P = 0.27). Whereas, In HOT CVR_{hyper} was not significantly altered from PRE values (P > 0.44; Figure 7.13A). Cerebrovascular hypocapnic reactivity (CVR_{hypo}) was similar between TEMP and HOT for both conditioning interventions compared to PRE (P > 0.50, Figure 7.13B).

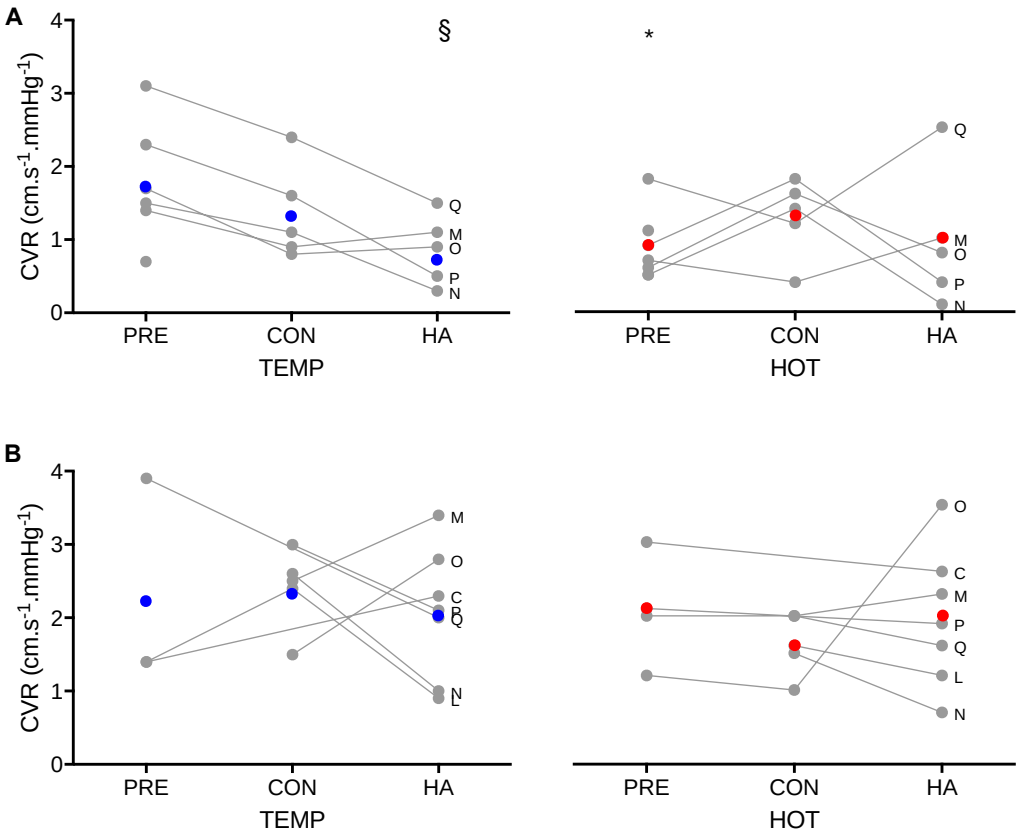


Figure 7.13: Cerebrovascular reactivity during hypercapnia (**A**) and hypocapnia (**B**) in temperate and hot environments before cycling simulation for no conditioning, control or heat acclimation conditioning. Mean in coloured symbols, individual responses in grey lines. Condition effect: §P < 0.05 for PRE compared with HA in TEMP; Time effect: * P < 0.05 for TEMP compared with HOT after PRE.

7.5.6.2 Brain Oxygenated Haemoglobin

In TEMP, the exercise-induced change in oxygenated haemoglobin volume (from rest) measured over the prefrontal cortex (Brain O_2Hb) increased more substantially after CON conditioning across 60-min SIM (Interaction effect: P = 0.05; Figure 7.14A). The Brain O_2Hb was higher after CON compared with PRE and HA at $FIXED_{Last}$, and $FREE_{First}$ (by ~136 μM·cm, P < 0.03) and increased across FIXED (by 398 μM·cm $FIX_{Last\ -\ First}$, P < 0.001) then remained similar across FREE (up ~490 μM·cm vs rest, P = 0.89). In HOT, Brain O_2Hb increased across FIXED (by 314 μM·cm $FIX_{Last\ -\ First}$, P < 0.001), then remained similar across FREE (up ~390 μM·cm vs. rest, P = 0.53). Conditioning did not affect the exercise-induced increase (Condition effect: P = 0.22, Interaction effect: P = 0.64; Figure 7.14A).

7.5.6.3 Brain Deoxygenated Haemoglobin

In TEMP, Brain HHb was similar between conditions at $FIXED_{First}$ but was lower following HA conditioning compared with PRE and CON at each time point thereafter (Interaction effect: P = 0.01; Figure 7.14B). Specifically, Brain HHb was lower after HA compared with PRE and CON at $FIXED_{Last}$, $FREE_{First}$, and $FREE_{Last}$ (by ~88 μM·cm, P ≤ 0.01). In HOT, Brain HHb increased across FIXED (by 111 μM·cm $FIX_{Last\ -\ First}$, P < 0.001) then remained similar across FREE (up ~110 μM·cm vs. Rest, P = 0.31); but was unchanged by CON and HA conditioning (Condition effect: P = 0.42, Interaction effect: P = 0.76; Figure 7.14B).

7.5.6.4 Brain Total Haemoglobin

In TEMP, Brain tHb increased after CON and reduced after HA conditioning across the FREE section of 60-min SIM (Interaction effect: P = 0.05; Figure 7.14C). Specifically, the Brain tHb was lower after HA compared with PRE and CON at $FREE_{First}$ (by ~200 μM·cm, P = 0.04) and higher after CON at $FREE_{Last}$ (by ~170 μM·cm, P = 0.04), increasing across FIXED (PRE: 357 μM·cm, CON: 559 μM·cm , HA: 374 μM·cm $FIX_{Last\ -\ First}$, P < 0.001 respectively) then maintaining similar concentrations across FREE (by ~580 μM·cm above resting, P = 0.91). In HOT, Brain tHb increased across FIXED (by 406 μM·cm $FIX_{Last\ -}$

$_{First}$, P < 0.001) then maintained similar concentrations across FREE (~480 μM·cm vs. rest, P = 0.88) but was unchanged by CON and HA conditioning (Condition effect: P = 0.26, Interaction effect: P = 0.61; Figure 7.14C).

7.5.6.5 Brain Tissue Oxygen Index

In TEMP, Brain TOI tended to decrease across the 60-min SIM and this response was unchanged by CON and HA conditioning (Time effect: P = 0.09; Condition effect: P = 0.55, Interaction effect: P = 0.53; Figure 7.14D). In HOT, Brain TOI tended to increase across 60-min SIM (P = 0.06) and this was also unchanged by CON and HA conditioning (Time effect: P = 0.06; Condition effect: P = 0.20, Interaction effect: P = 0.70).

7.5.6.6 Brain Blood Flow

In TEMP, the change in brain blood flow velocity (from rest) measured at the middle cerebral artery (MCAv) decreased across FIXED (by -7 $cm \cdot s^{-1}$, P < 0.001) then was similar across FREE (~6 $cm \cdot s^{-1}$, P = 0.46), and this time profile was unchanged by CON or HA conditioning (Condition effect: P = 0.80, Interaction effect: P = 0.29; Figure 7.14E). In HOT, accurate MCAv data were collected from only three participants, preventing statistical analysis for between conditioning interventions at all time points.

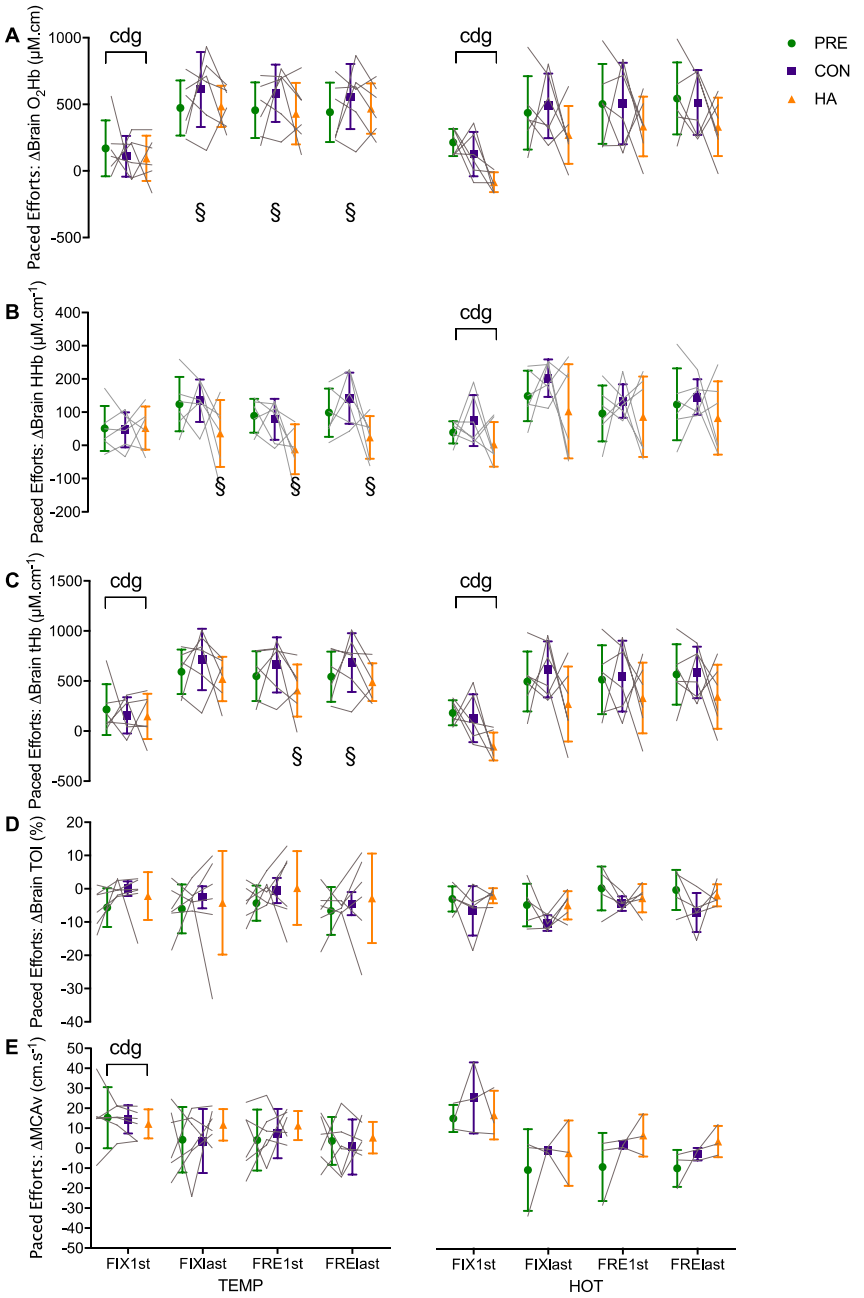


Figure 7.14: Brain oxygenated haemoglobin (A), deoxygenated haemoglobin (B), total haemoglobin (C), brain tissue oxygen index (D) and brain blood flow velocity (E) at first 2-min preset power output (FIX1st), last 2 min at preset power output (FIXlast), first 2 min at self-selected power output (FRE1st), and last 2 min at self-selected power output (FRElast) in temperate and hot environments during cycling simulation before or following control or heat acclimation conditioning. Analyses were not run for MCAv in HOT due to insufficient data. Mean ± SD in coloured symbols, individual responses in grey lines. Time effect: $P < 0.05$ at: [a] FIX1st, [c] FIXlast, [d] FRE1st, [g] FRElast. Interaction effect: [§]$P < 0.05$ for HA compared with CON and PRE.

7.5.7 Psycho-Physical Perceptions

7.5.7.1 Feeling Scale

In TEMP, participants' positive mood to exercise declined from Rest to $FIXED_{Last}$ (from 'Good' to 'Fairly Good', P = 0.03) and were similar during FREE (~'Fairly Good', P = 0.65) but were not altered by CON or HA conditioning (Interaction effect: P = 0.68; Condition effect: P = 0.17; Figure 7.15A). In HOT, participants felt more positive about exercise after HA compared with PRE, but felt similarly after HA compared with CON (Condition effect: P = 0.01; Interaction effect: P = 0.17; Figure 7.15A), feeling 'Fairly Good' after HA compared with 'Neutral' after PRE at $FIXED_{Mid}$ (P = 0.002). Positive mood to exercise declined from Rest to $FIXED_{Mid}$ (from 'Good' to 'Neutral', P = 0.002). Psycho-physical perceptions were not statistically analysed beyond $FIXED_{Mid}$ because insufficient (three) participants completed 60-min SIM after CON conditioning.

7.5.7.2 Thermal Discomfort

In TEMP participants' thermal discomfort increased from Rest to $FIXED_{Last}$ (from 'Comfortable' to 'Slightly Uncomfortable', P = 0.006) and was similar during FREE ('Slightly Uncomfortable', P = 0.92), which was not altered by CON or HA conditioning (Condition effect: P = 0.18; Interaction effect: P = 0.21; Figure 7.15B). In HOT, thermal discomfort deteriorated from Rest to $FIXED_{Mid}$ ('Comfortable' to 'Uncomfortable', P < 0.001), but was not altered by CON or HA conditioning (Condition effect: P = 0.20; Interaction effect: P = 0.15).

7.5.7.3 Thermal Sensation

In TEMP, participants felt cooler after HA (Condition effect: P = 0.04; Interaction effect: P = 0.34; Figure 7.15C), feeling 'Cool' after HA compared with 'Neutral' after CON conditioning at $FIXED_{Mid,}$ (P = 0.02) but similar thereafter (P > 0.46). In HOT, participants felt warmer from Rest to $FIXED_{Mid}$ (from 'Slightly Warm' to 'Warm', P < 0.001) and tended to feel cooler after HA conditioning compared with CON conditioning (Condition effect: P = 0.08; Interaction effect: P = 0.25).

7.5.7.4 Rating of Perceived Exertion

In TEMP, participants perceived exercise (RPE) was easier after CON and HA conditioning compared with PRE at different time points (Interaction effect: $P = 0.003$; Figure 7.15D). During warm-up RPE was lower after HA (8 vs 9, $P = 0.02$), but at $FIXED_{Last}$ it was lower after CON (14 vs 16, $P < 0.001$). In HOT, RPE increased from Warm-up to $FIXED_{Mid}$ (from 9 to 14, $P < 0.001$) but was not altered by CON or HA conditioning (Condition effect: $P = 0.38$; Interaction effect: $P = 0.90$).

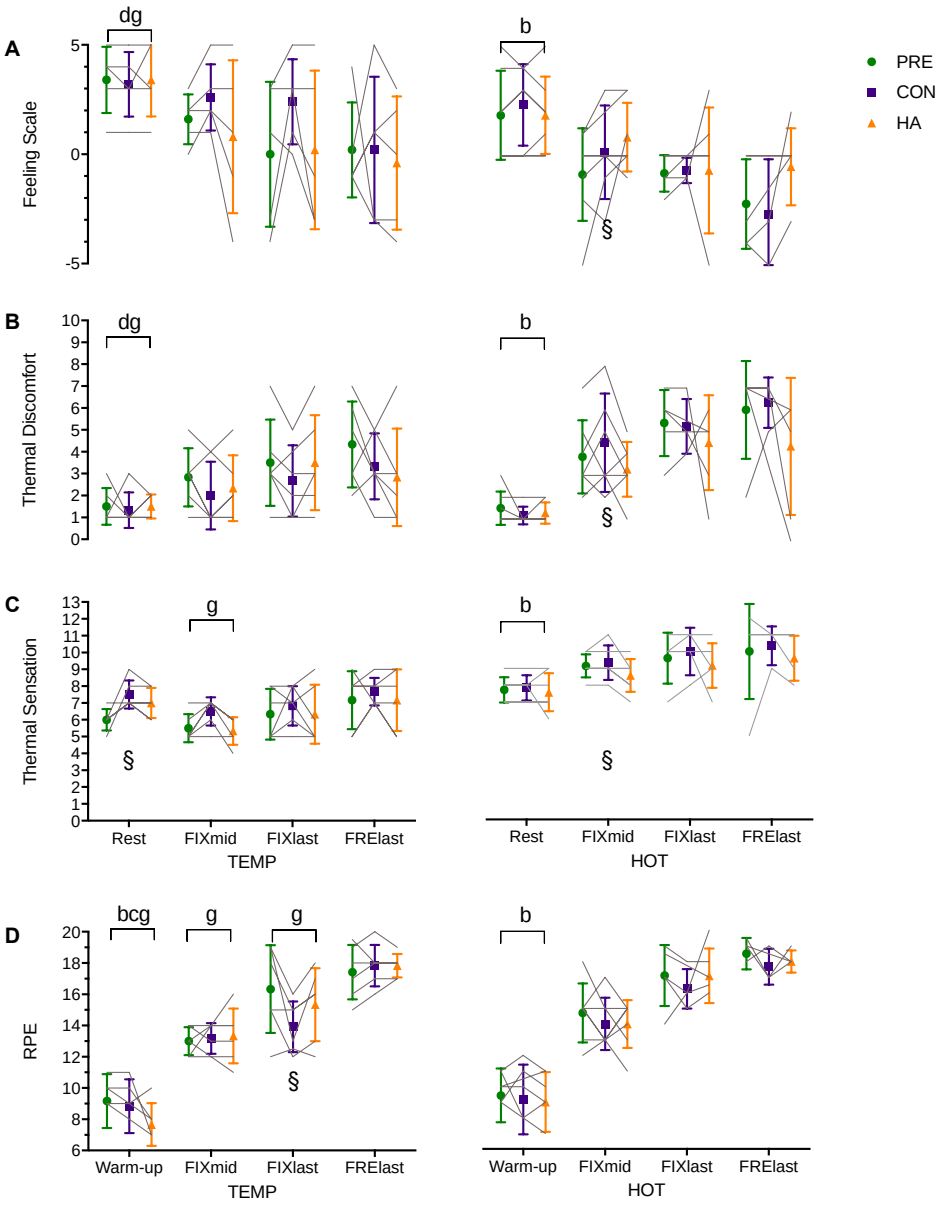


Figure 7.15: Feeling (**A**), thermal discomfort (**B**), thermal sensation (**C**), and perceived exertion (**D**) at rest, Warm-up, 10-min pre-set power output (FIX_{mid}), last 2-min at pre-set power output (FIX_{last}), and last 2-min at self-selected power output (FRE_{last}) in temperate and hot environments during cycling simulation before or following control or heat acclimation conditioning. Analyses was not run for FIX_{last} or FRE_{last} in HOT due to insufficient data. Mean ± SD in coloured symbols, individual responses in grey lines. Time effect: $P < 0.05$ at: ^{0}Rest, ^{b} FIX_{mid}, ^{c} FIX_{last}, ^{g} FRE_{last}; Condition effect: $^{\S}P < 0.05$ for PRE compared with CON or HA.

7.5.8 Fluid and Blood Volume Changes

7.5.8.1 Fluid Loss

Fluid loss, approximated as gross loss of body mass, was similar between conditioning protocols (P > 0.40) and greater in HOT across 60-min SIM (TEMP: 1.5%, HOT: 2.3-2.7%).

7.5.8.2 Blood Volume Changes

The HA conditioning did not significantly alter BV, PV, RCV or Hb mass compared with CON (P > 0.25, Figure 7.16A-D). From the first to last CON conditioning sessions Hct decreased [46.6 (2.69) vs. 44.8 (2.5), CI: -3.6 to -0.0, P = 0.05] but were similar for HA (P = 0.91).

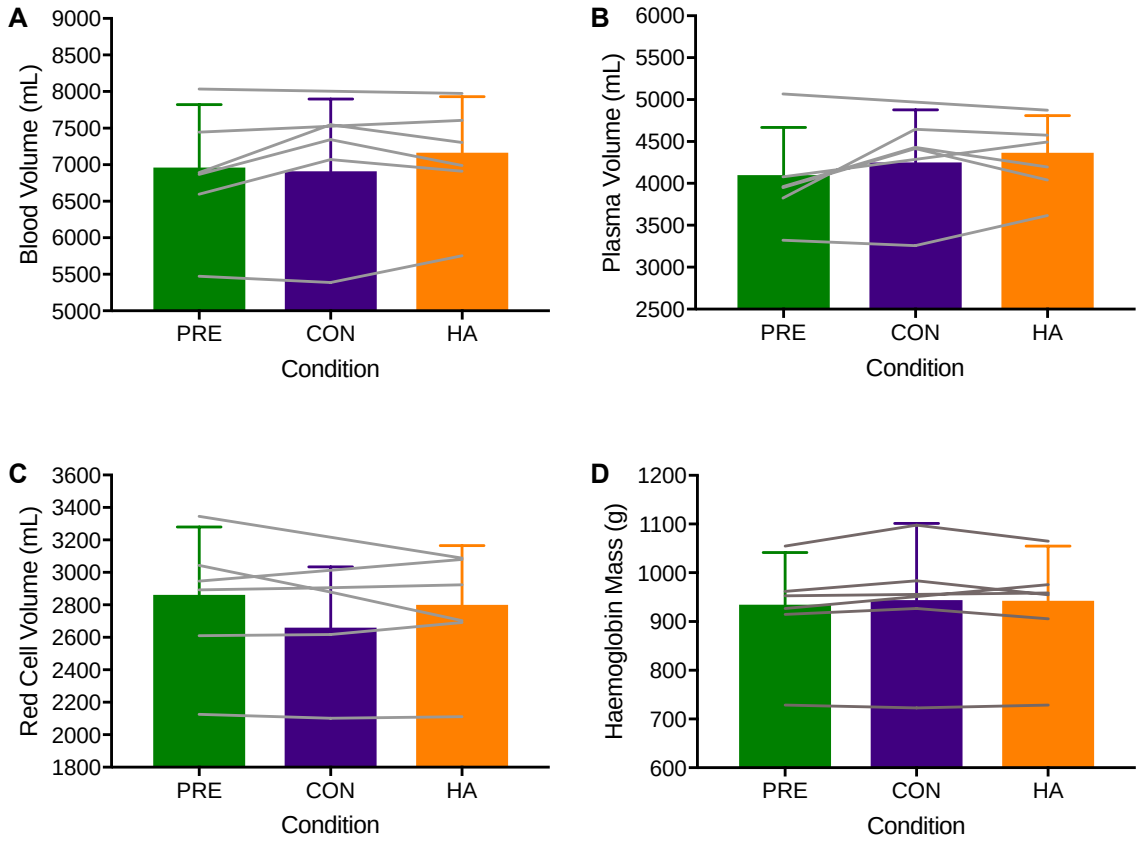


Figure 7.16: Blood volume, plasma volume, red cell volume, and haemoglobin mass measured before (green), then before and after control conditioning (purple) and heat acclimation (orange).

7.6 Discussion

The main findings from this study were that 10-d of heat acclimation reduced T_{Head} in TEMP and HOT, and improved athlete's mood to exercise in HOT. Heat acclimation also tended to improve 60-min SIM completion and total work in TEMP and HOT. The performance, cerebrovascular, cardiovascular, and psycho-physical responses during 60-min SIM were small and unclear in TEMP, while most physiological adaptations were not observed in HOT – an outcome that was not hypothesised. The heat acclimation protocol may not have induced the physiological adaptations required to significantly enhance 60-min SIM performance. Alternatively, the high endurance training status of the participants may have limited the ergogenic power of heat acclimation.

7.6.1 Heat Acclimation and Control Conditioning

Participants heat acclimated for ~10 h (38.2 ˚C, 59% RH) that improved affective state and thermal sensation. The HA conditioning for 10 d did not cause some of the significant adaptations that may indicate self-paced heat acclimation (Armstrong et al., 1986) or acclimatisation (Karlsen et al., 2015a), including: lowered resting HR, reduced exercising T_{Skin}, increased fluid loss, improved cerebrovascular reactivity, or greater MCAv during rest or exercise. Athletes also produced less total work during HA conditioning, not because they lacked motivation, but more likely because exercise intensity was self-paced allowing them to control (and so potentially optimise) the physiological strain arising from the exercise intensity plus environment. Average core temperatures during HA conditioning bouts were 38.3 °C, similar to the T_C strain advocated to induce heat acclimation (Fox et al., 1967; Garrett et al., 2009; Patterson et al., 2004a) and 1.0 °C higher than during CON conditioning bouts (Figure 7.3). The HA conditioning sessions were exceptionally hard, with some participants experiencing volitional exhaustion and presyncope. Self-pacing during the conditioning sessions allowed participants to exercise for a duration and intensity dependent on their anticipatory pacing strategy (Racinais et al., 2015; Tucker, 2009). This may have hidden physiological and psycho-physical changes that would normally be observed from a fixed pace protocol (e.g., Keiser et al., 2015a; Lorenzo et al., 2010; Nielsen et al., 1993, 1997) or controlled hyperthermia (Fox

et al., 1967; Garrett et al., 2009; Patterson et al., 2004a). Alternatively, it may indicate that the heat acclimation protocol was ineffective for this participant group.

7.6.2 Cycling Race Simulation

The 60-min SIM cycling performances were improved by HA conditioning in TEMP and HOT when considered in terms of survival, i.e., with five of seven participants completing 60-min SIM in HOT after HA compared with two completing in PRE and three in CON (Figure 7.4). Changes in cycling paced-effort and sprint power were most important during self-paced exercise (FREE). After heat acclimation participants produced greater sprint power in TEMP by 19% at $FREE_{Last}$, and greater paced effort power in HOT by 169% at $FREE_{First}$ and 81% at $FREE_{Last}$. However, CON *and* HA conditioning improved cycling paced-effort and sprint power by similar magnitudes in TEMP and HOT at all other FREE time points, demonstrating that HA may not have been ergogenic.

As expected, participants felt progressively bad, uncomfortable, and hot as 60-min SIM evolved, irrespective of conditioning protocol, because the 60-min SIM required maximal efforts (Figure 7.15A-C). The RPE was similar at the end of 60-min SIM (Figure 7.15D; Condition effect: $P > 0.25$) demonstrating that participants perceived they worked at equivalent intensity in different environments. However, thermal perception may have been improved by heat acclimation because participants felt better at $FIXED_{Mid}$ in HOT and felt cooler at the same time point in TEMP, implying that thermal perception was improved that may have contributed to greater performance power at the start of FREE (Schlader et al., 2011d). Furthermore, T_{Head} and T_{Ax} were substantially lower in TEMP and HOT (Figure 7.9), indicating that participants may have offloaded more heat via sweating (Cuddy et al., 2014; Sawka and Coyle, 1999).

After heat acclimation, power output was greater at the final sprint concomitant with reduced cardiovascular strain (lower HR), and lower Brain HHb in a temperate environment at the end of 60-min SIM. These observations may indicate that heat acclimation augmented intrinsic cardiovascular efficiency

(Horowitz, 2002; Lorenzo et al., 2010) and brain oxygen utilisation and metabolic efficiency (Horowitz, 2011; Kodesh and Horowitz, 2010). Heat acclimation may also have had a small beneficial influence on brain haemodynamics at the prefrontal cortex, improving anticipatory pacing, decision making and affective responses. Two controlled-trial studies have demonstrated that heat acclimation had a beneficial performance effect in temperate environments (Lorenzo et al., 2010; Scoon et al., 2007), while six have demonstrated no consistent performance benefits (Crowcroft et al., 2015; Karlsen et al., 2015a; Keiser et al., 2015a; Morrison et al., 2002; Neal et al., 2016; Zurawlew et al., 2016). Critically, all studies used time trials to determine performance, which has validity for athletes competing in events where they have autonomy for their pacing strategy and aim to optimise their effort over the performance distance by producing the highest average power possible. However, it does not account for athletes competing in events that require stochastic power defined by best average effort punctuated by maximal efforts (open water swimming, road, track and mountain bike cycling, road, track and trail running, draft-legal and off-road triathlon, football, field hockey). The present study showed no significant difference for total work completed in TEMP after HA compared with PRE and CON conditioning protocols, but it demonstrated that athletes produced greater sprint efforts at the end of 60-min SIM (Figure 7.6).

Prior to heat acclimation conditioning, participants were unable to constrain their rate of heat storage during FIXED, which substantially inhibited survival and performance (Figure 7.4; Figure 7.8) in the HOT environment. After HA conditioning athletes tended to feel cooler during 60-min SIM in HOT, presumably facilitated by lower T_{Skin} that may have contributed to greater self-paced power at the start of FREE (Schlader et al., 2011c, 2011b; Tucker et al., 2004, 2006b). Athletes also felt more positive during FIXED and were able to complete more of the FIXED section and successfully thermoregulate during FREE (Schlader et al., 2011d; Tucker, 2009), potentially via augmented heat loss at their individual core temperature tolerance (plateau in T_C). The delayed rate of change in T_C for four participants (Figure 7.8) may represent individuals who can produce more explosive and sustained power than their competitors. Thus, these same athletes would typically experience the highest metabolic rate and

therefore the greatest rate of change in T_C (Nielsen, 1938; Robinson, 1963) and performance deterioration when unacclimated. Therefore, these athletes potentially stand to gain more meaningful performance benefits from heat acclimation.

Heat acclimation improved respiratory responses (higher $P_{ET}CO_2$) at the start of self-paced efforts ($FREE_{First}$) for 60-min SIM in HOT when T_C and thermal strain were at or near their peak but this did not significantly influence cardiovascular or cerebrovascular responses. Blood pressure responses were similar between conditioning protocols, with changes over time and environments within 60-min SIMs closely resembling those of Chapter 6 (Figure 6.13), albeit self-paced exercise in HOT lacked sufficient statistical power to make accurate comparisons (n = 3; Figure 7.11). The CVR_{hypo} response was similar between environments, indicating that CON and HA conditioning did not alter brain vasculature responses to reduced $PaCO_2$ or alterations in brain $[H^+]$ driven by hyperventilatory hypocapnia (Ainslie and Duffin, 2009; Ogoh et al., 2008). Improved cerebrovascular tolerance at higher core temperatures has been observed in untrained individuals after repeated warm water immersion training (Bailey et al., 2016) and in highly-trained cyclists after ten days of heat acclimation (13% MCAv increase; (Keiser et al., 2015a). However, highly trained cyclists demonstrated no significant change in cycling performance or Brain HbO_2 content (Keiser et al., 2015a), indicating that highly trained athletes may require longer heat acclimation training protocols.

When performance was analysed collectively (e.g., Survival Analysis: Figure 7.4; Total Work: Figure 7.5; Cycling Power: Figure 7.6) it indicated that HA conditioning showed only a tendency to be ergogenic for 60-min SIM in HOT. Paced efforts tended to be more powerful at the start of FREE in HOT, which influenced greater total work, though remained unclear due at least partly to limited sample size (n = 7). The ergogenic characteristics of heat acclimation may have been blunted by the participant group, who had substantial physiological adaptation from years of endurance training. Indeed, typical adaptations from heat acclimation including blood volume expansion and the associated increase in $\dot{V}O_2$ max and therefore performance enhancements may

have been limited (Karlsen et al., 2015a) because the athletes already had high blood volumes before the study began [PRE: 6960 (863) mL, 92 $mL \cdot kg^{-1}$; Figure 7.16].

7.6.3 Limitations

The study lacked statistical power when conditioning interventions were compared due to the high drop-out rate during 60-min SIM HOT trials after PRE and CON, potentially masking any beneficial or detrimental influence to performance or physiological responses that may have been present. This was especially problematic when exploring how heat acclimation influenced hyperthermic hyperventilatory hypocapnia because insufficient MCAv data were collected in HOT environments due to recording challenges during prolonged maximal exertion and the small number of participants who completed 60-min SIM in PRE and CON interventions. For central adaptations, cerebrovascular reactivity produced unexpected results: acute heat stress did not reduce CO_2 reactivity (Ogoh et al., 2014), and the response to hypercapnia was greater in TEMP than HOT environments at PRE when it should have responded similarly for PRE and CON. Whether this was a characteristic of the testing equipment, protocol or participants themselves was unclear. Some peripheral adaptations to heat acclimation were not measured in this study, including cellular thermotolerance conferred by heat shock protein (HSP) synthesis (Horowitz, 1998; Kodesh et al., 2011; Moseley, 1997). The frequency, intensity and volume of HA conditioning were expected to confer HSP and cellular adaptations typical of heat acclimation training (Amorim et al., 2015; Lee et al., 2015; Marshall et al., 2007; Sandström et al., 2008; Yamada et al., 2007). The training level and characteristics of these athletes may have also hindered the aims of this study because the best athletes may achieve only subtle performance and physiological improvements from heat acclimation, however, a 0.3-1% improvement may be crucial to success in elite level endurance sports (Hopkins, 2004). The athletes who stand to gain the most from heat acclimation training are those who are less aerobically powerful and/or less comfortable in hot environments. Finally, this study was unique because it allowed athletes to control physiological strain based on their affective state, a protocol that many

athletes intuitively follow when training in different environments. However, HA conditioning (hot-water bathing followed by self-paced cycling in a hot environment) may have lacked the stimulus required to improve 60-min SIM performance.

7.6.4 Conclusion

In conclusion heat acclimation for 10-d reduced T_{Head} in TEMP and HOT, and improved athlete's mood to exercise in HOT. Heat acclimation tended to improve 60-min SIM completion and total work in TEMP and HOT. The performance, cerebrovascular, cardiovascular, and psycho-physical responses during 60-min SIM were small and unclear in TEMP, while most physiological adaptations were not observed in HOT. The HA conditioning did not induce the performance enhancements or physiological adaptations that these participants required. Alternatively, the high endurance training status of the participants may have limited the ergogenic power of heat acclimation.

8 CONCLUSION

Conclusions, recommendations and limitations are based on the findings and research experiences gained from these studies.

8.1 Conclusions

- Elite triathletes racing in warm, humid environments:
    - completed the cycle leg in a faster time due to the lower air density
    - ran slower with higher T_C and potentially lower SpO_2;
    - had similar heart rates to temperate environments, indicating that the capacity of the cardiovascular system may have been a key performance limitation. The cardiovascular responses observed in competitive racing supported the laboratory findings despite the constrained exercise and environmental protocols;
    - ran slower in the first kilometre and deteriorated substantially thereafter possibly because they could not pace themselves appropriately and experienced considerable fatigue.
- Highly trained triathletes and cyclists produced 11% greater power for 10-s sprints when exercise was preceded by sitting quietly then warming up for 10 min in hot environments.
- Highly trained triathletes and cyclists performing a 5-min time cycling time trial in hot environments:
    - produced 3.4% less power
    - tended to produce less power at the Start and Middle of the time trial
    - experienced lower prefrontal cortex (brain) oxygen saturation and felt hotter (thermal discomfort) that may have encouraged a conservative pacing strategy;
    - experienced greater metabolic perturbations and blood flow redistribution to the skin.
- Highly-trained triathletes and cyclists performing a 60-min cycling race simulation in hot environments:

- Reached volitional exhaustion during the fixed pace and sprint section for 9/17 athletes;
- produced 19 to 26% less power during self-selected paced efforts compared with the same efforts in a temperate environment;
- produced 15% less power for the last sprint of the race simulation compared with the same effort in a temperate environment;
- experienced greater T_C and T_{Skin} during fixed and self-selected efforts;
- experienced greater physiological (respiratory, cardiovascular, central, peripheral, dehydration) strain;
- experienced greater psycho-physical (thermal discomfort, sensation, RPE) strain;
- experienced prefrontal cortex oxygen desaturation that may have influenced anticipatory pacing and affective state, in conjunction with leg muscle oxygen desaturation and blood flow redistribution during the fixed-pace section that presumably contributed to volitional exhaustion or severely challenged their ability to continue exercising;
- when athletes self-selected their paced efforts and sprints they could reduce (e.g., $\dot{V}_E$) or limit the rate of increase (e.g., T_C, HR, prefrontal cortex haemodynamics, peripheral haemodynamics) of physiological responses to avoid volitional exhaustion.

- Highly-trained triathletes and cyclists who completed 10 days of heat acclimation:
  - Improved their completion of 60-min SIM in a hot environment
  - improved the total work they completed in a hot environment;
  - did not experience a reliably beneficial power improvement compared with the control conditioning protocol;
  - experienced lower T_{Head} in temperate and hot environments;
  - felt more positive about exercising in a hot environment;
  - had lower heart rates in temperate environments and similar heart rates in hot environments concurrent with greater power output.

8.2 Recommendations

- The beneficial performance outcomes from increasing core and muscle temperature in a hot environment for sprinting events hinder short-duration maximal-effort performances of 5-min duration, and stochastic-profiled performances of 40 min duration are markedly impaired. The duration and specific competitions where the performance cross over occurs between environments requires further investigation, but will logically depend also on factors relating to the athlete and their circumstances (e.g., warm up).
- Acclimation should not be limited to training in an environment chamber. Athletes may include sauna bathing to volitional exhaustion immediately after endurance training (Scoon, Hopkins, Mayhew, & Cotter, 2007; unpublished observations), Bikram yoga, and immersing themselves in a hot bath either before (the present study: Chapter 6) or immediately after exercise (Zurawlew et al., 2015).
- Heat acclimation, like altitude training, may be ergogenic but involves considerable risk. The benefit from heat acclimation is that it is cheaper, can be effectively performed within the resources of an athlete's home, and can still be beneficial if environmental conditions are unseasonably cool, but meaningful erythropoiesis from such conditioning is doubtful.

8.3 Limitations

The intent to test highly-trained and elite triathletes and cyclists limited the number of participants and clarity of some results, particularly the field (Chapter 3) and heat acclimation (Chapter 7) experiments. Additionally the requirement to involve the greatest number of elite participants as possible created considerable time pressure on the experimental protocols for the 10 s, 5-min TT, and 60-min SIM (Chapters 5 and 6) experiments. Coaches and athletes were comfortable to allow a seven-day window away from their normal training routines for experimental testing – but no more. Thus the 10 s, 5-min TT, and 60-min SIM required shorter recovery times between experiments and a protocol that was structured from physiologically least to most-stressful to meet the time

restrictions. The potential confounding from a learning and/or order effect may have influenced the results.

The literature review made a point of highlighting airflow as an important environmental consideration that was not accounted for in some studies and strongly influenced thermoregulation in prolonged endurance cycling (Brown and Banister, 1985). The laboratory experiments delivered airflow at participants at 4 $m.s^{-1}$, generated using one of the largest fans that could be commercially obtained for the experiments. This airflow was realistic for running but not cycling. Cyclists move at 8-12 $m.s^{-1}$. Experiments that have simulated airflow similar to cycling have done so in a wind tunnel (Saunders et al., 2005), and demonstrated that. because of the exponential relation between airflow and heat transfer, 4 $m.s^{-1}$ provides similar physiological responses to those at higher velocities.

Running was identified in the field study as the most physiologically demanding section of the race. However, the laboratory studies focussed on cycling – not running. Pilot trials were conducted using treadmill running in the environment chamber because of the considerable evidence gathered in the field. Piloting in the laboratory demonstrated overwhelming physiological stress, reproducing the performance outcomes observed in the field. Furthermore, prior to 2012 one study had investigated how brain blood flow was influenced by treadmill running, concluding that running challenged blood pressure and cerebral autoregulation (Lyngeraa et al., 2013). Regrettably, few physiological measures could be accurately collected while athletes ran on the treadmill. Instead, cycling allowed greater quantity and quality of data collection, particularly brain blood flow (a major focus of the studies). For this reason cycling was selected as the exercise mode for laboratory experiments.

The potentially negative influence from the short recovery between experiments may have been offset by the participants' training status and therefore ability to recover from prolonged, very hard efforts that were part of their normal training.
Heat acclimation generated almost no measurable physiological adaptations. This was both unexpected and prevented a definitive conclusion that explored whether heat caused an ergogenic influence for highly-trained triathletes and

cyclists. Speculatively, the cause may have been the low participant numbers; a larger participant pool would have produced clearer results. However, increasing the participant numbers would have included participants with a lesser training status – potentially making the conclusions of uncertain relevance to highly-trained endurance athletes. Alternatively, the modified heat acclimation protocol that was designed to be more practical and effective may have been ineffective compared with a traditional design.

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10 APPENDICES

10.1 Appendix One: Feeling Scale

“How do you feel about exercise?”

+5 Very Good

+4

+3 Good

+2

+1 Fairly Good

0 Neutral

-1 Fairly Bad

-2

-3 Bad

-4

-5 Very Bad

10.2 Appendix Two: Thermal Discomfort Scale

“How comfortable do you feel with the temperature of your body?”

1.0 Comfortable

2.0

3.0 Slightly Uncomfortable

4.0

5.0 Uncomfortable

6.0

7.0 Very Uncomfortable

8.0

9.0 Extremely Uncomfortable

10.0

10.3 Appendix Three: Thermal Sensation Scale

“How does the temperature of your body feel?”

1.0 Unbearably Cold

2.0 Extremely Cold

3.0 Very Cold

4.0 Cold

5.0 Cool

6.0 Slightly Cool

7.0 Neutral

8.0 Slightly Warm

9.0 Warm

10.0 Hot

11.0 Very Hot

12.0 Extremely Hot

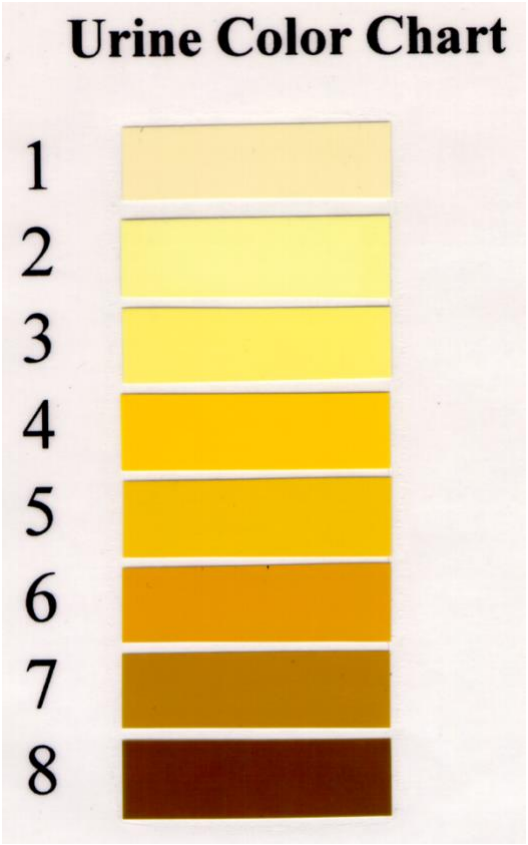
13.0 Unbearably Hot

10.4 Appendix Four: Rating of Perceived Exertion (Borg, 1982)

“How hard are you exercising”

6	**None**
7	**Extremely Light**
8	
9	**Very Light**
10	
11	**Light**
12	
13	**Somewhat Hard**
14	
15	**Hard**
16	
17	**Very Hard**
18	
19	**Extremely Hard**
20	**Maximal Exertion**

10.5 Appendix Five: Hydration Scale



- Do not judge urine colour within a few hours after taking vitamin supplements, as the unused vitamins may turn the urine a brighter shade of yellow.
- Check urine colour regularly at similar times of the day. Day-to-day differences in urine colour will be insignificant if meals, fluid consumption and training are consistent.
- The first specimen of urine produced for a test is always slightly more concentrated than subsequent specimens, therefore always check the second urine specimen for colour on the urine colour chart.
- Remember that frequency of urination is another good indicator of hydration status (*frequent* is better than *infrequent*).
- Seek to produce urine that is *'very pale yellow', 'pale yellow'* or *'straw coloured'* corresponding with numbers 1, 2 and 3 on the chart respectively.