Novel Applications of Heat Training to Enhance Performance in Elite Athletes

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ATTESTATION OF AUTHORSHIP

I hereby declare that this submission is my own work and that, to the best of my knowledge and belief, it contains no material previously published or written by another person (except where explicitly defined), nor material which to a substantial extent has been submitted for the award of any other degree or diploma of university or institution of higher learning.

Julia Casadio

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At the young age of three years old, I witnessed the Winter Olympics Games in my home town of Calgary, Canada. It was those Games that inspired me to pursue excellence and to work hard at all I do in hopes that one day I too could be an Olympian. Physiology would have it that I wouldn't realise this dream as an athlete. However, through this thesis, and the many resulting opportunities, I was able to pursue my passions, which in some way feels like I reached my Olympic dream, just via an alternate route. This has only been possible due to the many amazing people in my life, to whom I owe many thanks.

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In closing, my hope is that as athletes continue to pursue the physically impossible through hard work and advances in sport science, more individuals (especially our youth) will be inspired to pursue a life of excellence, just as I was as a small child, fascinated and inspired by watching my Olympic hero's give their all.

Do you not know that those who run in a race all run, but only one receives the prize? Run in such a way that you may win (1 Cor 9:24).

ETHICAL APPROVAL

Ethical approval for all research within this thesis was obtained through the Auckland University of Technology's Ethic Committee (AUTEC). Each study and corresponding AUTEC Ethics Approval Number are outlined below.

- Study 1 (Chapter 3) – Ethics Approval Number 15/382

- Study 2 (Chapter 4) – Ethics Approval Number 15/379

- Study 3 (Chapter 5) – Ethics Approval Number 14/130

- Study 4 (Chapter 6) – Ethics Approval Number 14/397

CO-AUTHORED WORKS

Work Published (Chapters 3, 7 and 8)

Casadio JR, Kilding AE, Siegel R, Cotter, JD Laursen PB, (2016). Periodizing heat acclimation in elite Laser sailors preparing for a World Championship event in hot conditions. *Temperature* 3 (3): 437-443. (Casadio 80%, Kilding 5%, Siegel 5%, Cotter 5% Laursen 5%)

Casadio JR, Storey AG, Merien, F, Kilding, AE, Cotter, JD, Laursen, PB (2017). Acute effects of heated resistance training in highly-trained female and male power athletes. *European Journal of Applied Physiology.* In Press. (Casadio 80%, Storey 7.5%, Merien 2.5%, Kilding 2.5%, Cotter, 2.5%, Laursen 5%)

Casadio JR, Kilding AE, Cotter, JD, Laursen PB (2016). From lab to real world: Heat acclimation considerations for elite athletes. *Sports Medicine*, 1-10. (Casadio 80%, Kilding 5%, Cotter, 5%, Laursen 10%)

Casadio JR & Laursen PB (2016). Comments to CrossTalk 27: Heat acclimation and performance in the elite: Considering the individual. *Journal of Physiology*, 000 (0): 1-6. (Casadio 85%, Laursen 15%)

CONFIDENTIAL MATERIAL

Studies 2 (Chapter 3) and 3 (Chapter 4) of this thesis, along with the overall discussion (Chapter 9) are subject to embargo until May 2018. Cycling New Zealand believe the findings of this work represent a competitive advantage to the New Zealand track cycling team and the campaign to the Gold Coast Commonwealth Games in 2018.

ABSTRACT

The use of training in the heat to enhance performance has become a 'hot topic' in the world of elite sport given that a number of high profile events are held in hot environments, and emerging evidence suggests that training in the heat has the potential to be ergogenic. Short-term heat acclimation (HA) is a commonly used strategy to prepare athletes for competition in hot environments, but may also improve endurance performance in temperate conditions, enhance muscle strength and hypertrophy, and has been shown to increase sprint and power performance acutely. The majority of research used to establish our understanding of the acute physiological effects of heat and HA to date has been completed in untrained or moderately-trained individuals, with little work completed in well-trained or elite athletes. Thus, investigation into practical integration of HA or heat training into an elite athlete's already busy training schedule, without interfering with key training sessions and competitions, is needed. Finally, most studies to date have tended to focus on performance measures relevant to long-duration, endurance exercise, and a greater understanding of how HA or heat training affects sprint performance is needed. Therefore, the primary aim of this thesis was to examine how the application of heat, through HA or heat training, might enhance physical performance. A secondary aim was to develop practical strategies that could be easily incorporated into the training programmes of elite athletes, including those competing in sprint and short duration power events.

To address these aims, four studies were conducted. Study 1 examined the retention of HA adaptations and re-acclimation (RA) responses during a periodised short-term HA protocol in elite sailors preparing for a World Championship competition

Abstract

in hot conditions. Study 2 examined the effects of a sprint cycling-specific heat training camp on sprint performance and physiological responses in elite sprint track cyclists in temperate conditions. As partial HA adaptations were shown in Study 2, the next study (Study 3) examined the acute effects of heat, as well as the effects of 5 d of sprint- and resistance training-based HA on repeated-sprint performance in elite BMX athletes. Based on the methods used in Studies 2 and 3, Study 4 aimed to determine the effects of a strength- and power-based resistance training session in the heat on thermal strain, neuromuscular function and hormonal responses in highly-trained power athletes.

Collectively, the studies contained within this thesis showed that HA and heat training can be strategically integrated into the complex training schedules of elite athletes and may effectively enhance physiological adaptations that support improved performance. The new findings included: 1) RA may 'top-up' thermoregulatory adaptations following initial HA and a 2-week period without heat exposure, 2) sprint performance is temporarily impaired following short-term heat training, despite partial HA and improved cardiovascular function, 3) BMX repeated-sprint performance is enhanced in the heat, notwithstanding increased thermal strain, and further HA improvements, and 4) power and anabolic hormone responses are elevated following a strength- and power-based resistance training session in the heat.

CHAPTER ONE: INTRODUCTION

1.1 Background

Several pinnacle sporting events are held in locations where ambient temperature and/or humidity levels are high. Some of these high profile events include, most Summer Olympic Games, the Tour de France, the FIFA World Cup, as well as several annual World Cups and World Championships. Given that physical exertion in unaccustomed heat-stress can impose considerable physiological and thermal strain, leading to impaired performance (Racinais et al., 2012; Sunderland & Nevill, 2005; Tatterson, Hahn, Martini, & Febbraio, 2000), and possibly exertional heat injury (EHI) (Binkley, Beckett, Casa, Kleiner, & Plummer, 2002), ensuring that athletes are prepared to perform optimally in hot environments is of high importance. To combat heat strain, and the consequential heat-induced performance decrements, heat acclimation (HA) is generally prescribed. Heat acclimation (artificial heat exposure, indoors), or heat acclimatisation (natural heat exposure, outdoors), involves multiple exercise-training sessions in hot conditions, that induce physiological adaptations which attenuate heat-induced performance impairments to protect against heat stress and EHI (Périard, Racinais, & Sawka, 2015; Taylor, 2014; Tyler, Reeve, Hodges, & Cheung, 2016). In addition, emerging evidence suggests that training in the heat itself has the potential to be ergogenic versus equivalent training performed in temperate conditions (Corbett, Neal, Lunt, & Tipton, 2014). If confirmed, such training could be used as a targeted strategy to maximise training adaptations and enhance performance throughout a season (Chalmers, Esterman, Eston, Bowering, & Norton, 2014).

It is well established that competing in hot and humid environments imposes significant thermal and physiological strain, and that the consequences of hyperthermia

(core temperature > 38.5°C (Bligh & Johnson, 1973)), will ultimately diminish an athlete's performance in the unacclimated state (Armstrong & Dziados, 1986; Sawka, Young, Cadarette, Levine, & Pandolf, 1985). Heat-induced performance decrements can range from 6-16% in trained athletes during endurance and team sport events (Racinais et al., 2012; Sunderland & Nevill, 2005; Tatterson et al., 2000), while ~10% reductions in sprint performance have been shown in athletes that are hyperthermic (Drust, Rasmussen, Mohr, Nielsen, & Nybo, 2005; King, Costill, Fink, Hargreaves, & Fielding, 1985). Heat-induced performance impairments are caused by a number of physiological and thermoregulatory adjustments, including increases in body temperature, augmented cardiovascular strain, and a greater rate of fuel utilisation, which together lead to progressive reductions in work output (Galloway & Maughan, 1997; King et al., 1985; Périard, Cramer, Chapman, Caillaud, & Thompson, 2011). A full review of the physiological and thermoregulatory responses to heat stress and subsequent performance decrements is found in Chapter 2 of this thesis.

Heat acclimation can mitigate the abovementioned performance impairments through physiological and thermoregulatory adaptations that reduce the effects of heat stress (Castle, Mackenzie, Maxwell, Webborn, & Watt, 2011; Garrett, Creasy, Rehrer, Patterson, & Cotter, 2011; Lorenzo, Halliwill, Sawka, & Minson, 2010). The classical heat-acclimated state is characterised by decreased resting and exercise core temperatures, lowered skin temperature, increased plasma volume and sweat rate and decreased heart rate for a given workload, all of which combine to attenuate performance impairments caused by hot conditions in the unacclimated state (Armstrong & Maresh, 1991). Taken together, the enhanced thermoregulatory function

improves exercise economy (Armstrong & Maresh, 1991) and improves prolonged (Tatterson et al., 2000), high-intensity intermittent (Sunderland, Morris, & Nevill, 2008) and sprint performance (Castle et al., 2011) in the heat.

Recent consensus recommendations offer coaches and sport scientists an understanding of the key concepts needed to prescribe HA for individual and team sport athletes (Chalmers et al., 2014; Racinais et al., 2015). Best practice guidelines state that HA should be comprised of daily ~ 60 min training sessions in hot conditions for a minimum of 1 week, and ideally performed over 2 weeks to achieve further thermoregulatory and performance benefits. The HA protocol should mimic the event demands while inducing high sweat rates and increased body (skin and core) temperature (Racinais et al., 2015). While such recommendations are based on decades of evidence, limitations and barriers to adhering to such guidelines arise when practitioner and coach attempt to integrate these principles within the complex training schedule and priorities of an elite athlete. As a result, when the literature is scrutinised amongst the context of the elite athlete, a number of limitations are apparent. First, much of the research used to establish our understanding of HA to date has been completed in untrained or moderately-trained individuals, with little work completed in well trained or elite athletes, whom may already have partial heat-related adaptation due to their well-trained status (Cheung & McLellan, 1998). Second, as the majority of HA studies are laboratory based, investigation into how best to practically integrate HA into an elite athlete's already busy training schedule, is needed. As a result, more applied research is required to understand the use of more event or sport-specific HA, the optimal timing of HA implementations, how it might be periodised throughout a competitive season, as well as the effects of HA on concurrent training priorities and

recovery. Last, practical HA solutions are required for highly-trained athletes who do not always have accessibility to a climate chamber or hot environments.

To date, most literature in the area of HA using trained athletes, has focused on endurance sports, as training in the heat promotes cardiovascular adaptations, specifically an enhanced cardiac efficiency through increased plasma and stroke volume and reductions in heart rate (Nielsen et al., 1993). Little attention, however, has been given to the effects of heat acclimation on sprint events lasting < 2 min. In lab settings, sprint performance has been shown to improve in the heat when single and repeated efforts are performed (Ball, Burrows, & Sargeant, 1999; Girard, Bishop, & Racinais, 2013; Sargeant, 1987). This enhanced performance may be attributed to increases in muscle temperature that elicit higher peak power outputs and cadence (Sargeant, 1987). However, with hyperthermia, impairments in single (King et al., 1985) and repeated sprint performance have been shown, despite an increased muscle temperature (Drust et al., 2005). As one of the direct effects of HA is the reduction of core temperature during exercise in the heat (Adams, Fox, Grimby, Kidd, & Wolff, 1960), the use of HA for sprint athletes could be highly beneficial to performance during competitions where there are multiple rounds or long durations spent in hot ambient environments. Furthermore, the use of heat training as a tool to enhance training adaptations and performance also has potential. As an example, work has shown that long-term exposure in the heat can increase muscle hypertrophy and maximal strength (Goto et al., 2011; Goto et al., 2007). Indeed, mechanistic studies have shown that heat stimulates the Akt/mammalian target of rapamycin (mTOR) signalling pathway, a key regulator of protein synthesis and hypertrophy (Kakigi et al., 2011; Yoshihara et al., 2013). Thus heat, applied in various ways, could provide an ergogenic effect when combined with

resistance training, which is an integral component of any sprint athlete's training programme (Haff & Nimphius, 2012).

1.2 Rationale and Thesis Aims

At the time that this thesis was being proposed, High Performance Sport New Zealand (HPSNZ) had targeted the area of heat and performance as a research focus, in preparation for the 2016 Summer Olympic Games, in Rio de Janeiro, Brazil. The conditions in Rio were expected to be hot (~23-35°C, 40-70% relative humidity (RH)), in comparison with August winter conditions in New Zealand (~13°C, 70% RH) (Casadio, 2013). It was therefore crucial to develop and optimise HA protocols that were both effective and practical, so that New Zealand athletes were advantaged heading into the Games. This HPSNZ initiative, along with the background and limitations found in literature, formed the objectives of this body of work. Therefore, the overarching aim of this thesis was to optimise the use of heat, through HA or heat training, to enhance physical performance through practical strategies that could be easily incorporated into the training programmes of elite athletes. To achieve this aim, the thesis was divided into five chapters with the following specific objectives:

i.   To examine the retention and re-acclimation responses during a periodised short-term HA protocol prior to departing for competition in a hot environment.

ii. To investigate the effects of heat training on sprint performance and thermoregulation, and determine if heat training causes beneficial adaptations in sprint athletes.

iii. To determine, first, the effects of acute heat stress on sprint performance, and second, the effects of sprint- and resistance training HA on thermoregulation and sprint performance in the heat in sprint athletes.

iv. To understand the acute effects of resistance exercise performed in hot versus temperate conditions on neuromuscular function, hormone responses and thermal strain in power athletes.

v. To review alternative methods of HA and heat exposure, explore how HA might be integrated into an athlete's existing training program, and highlight specific athlete considerations for practitioners.

vi. To consider the effects of HA as an ergogenic tool for individual athletes, rather than assuming each athlete will follow a given mean response shown in literature.

1.3   *Thesis Organisation*

This doctoral thesis was intended to inform how HA and heat training can be used to enhance performance in elite athletes through practical strategies that could easily be integrated into an athlete's training programme. The thesis is presented in six main sections that have focused on HA and heat training and the effects on thermoregulation and athletic performance (Figure 1.1). These sections include an introduction, a literature review, a main body (four studies, one current opinion and one invited comment), an overall discussion and finishing with a conclusion, references, and finally an appendix section. Two chapters, included in the main body of this thesis, were embargoed at the request of Cycling New Zealand. Other specific chapters were written

for publication in targeted peer-reviewed journals. Therefore, these chapters were formatted based on publication guidelines and word limits for the respective journals they have been submitted to. However, for the consistency of referencing, all citations have been presented in American Psychological Association (APA) format using a single reference section at end of this thesis.

The literature review of this thesis (Chapter 2) examines the effects of heat and HA on performance in trained athletes. First, the physiological and thermoregulatory effects of heat stress are described, followed by the implications on performance in trained athletes. Second, classical heat acclimation adaptations are outlined, along with their effects on performance in endurance, sprint and team sport athletes. Last, the potential for heat to be used as a training tool, to enhance training adaptation and performance is examined. Based on the current state of literature, the limitations and apparent questions pertaining to how we might best go about using heat training and HA with elite athletes have been emphasised.

The thesis body consists of three HA or heat training interventions, one experimental trial, one current opinion and one invited comment. Three of these chapters were published (or submitted) prior to the completion of this thesis. These chapters hold the common theme of using HA or heat training in applied sport settings, with highly-trained or elite athletes, although, the athlete type or sport does vary between experimental studies.

The final chapter of the thesis (Chapter 9) consists of an overall discussion, which highlights the key findings of each chapter, how they interrelate, and addresses the limitations within these findings. There is also a section which outlines the practical

applications of this thesis, given the applied intention of the research is focused towards elite athletes. Finally, future research directions are explored. Overall, this thesis addressed questions related to how heat training and HA strategies might be used to maximise physiological adaptations and performance outcomes for elite athletes.
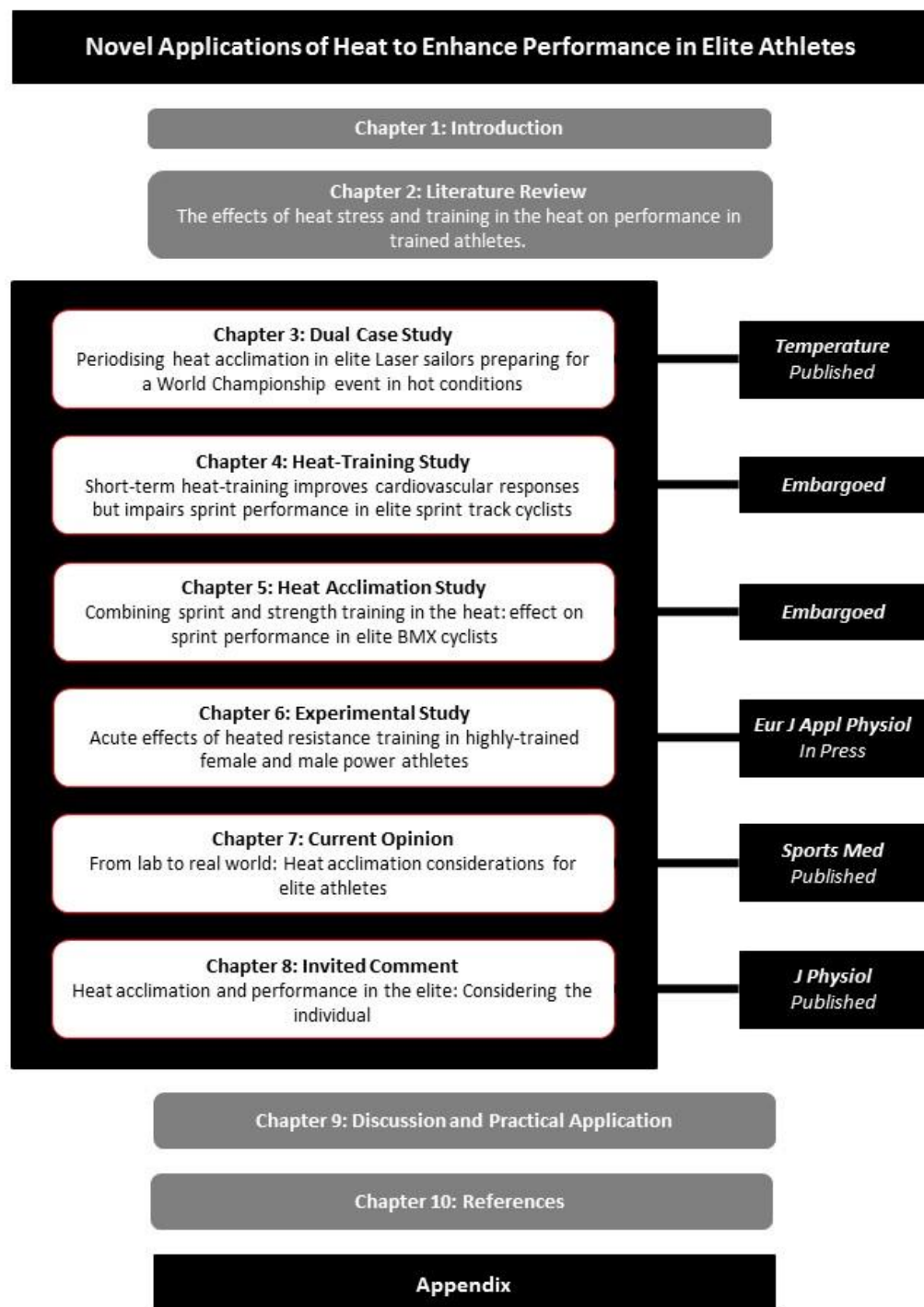
Figure 1.1 Overview of the thesis structure.

1.4 Overview of Studies

A summary of each chapter included in the main body of this thesis is provided below:

Chapter 3, Study 1

This dual case study involved two elite Laser sailors who used periodised HA to prepare for their World Championship regatta in Muscat, Oman (~27-30ºC, 40-60% RH). The aim of this study was to examine the retention of HA adaptations and re-acclimation responses during a periodised short-term HA protocol prior to the sailor's departure to the hot competition venue.

Chapter 4, Study 2

This study changed focus from endurance training-based HA, and shifted towards examining the use of heat training as a tool to enhance sprint performance. The specific aims of this study were to determine the effects of a sprint specific short-term heat training protocol on sprint performance in temperate conditions, as well as assess cardiovascular responses in elite sprint track cyclists.

Chapter 5, Study 3

Having used sprint- and resistance-based heat training in Study 2, I then wanted to examine whether this form of training would induce HA adaptations in BMX athletes, who were complaining of heat stress when competing at hot venues. The aims of this study were to examine the effects of acute heat stress on BMX sprint performance, and determine the effects of sprint- and strength-based HA, on

thermoregulatory factors and repeated sprint performance in the heat in elite BMX athletes.

Chapter 6, Study 4

Following studies 2 and 3, which used heated-resistance training, I wanted to gain a better understanding of the acute effects of a strength- and power-based resistance exercise session in the heat compared with normal temperate conditions. The aim of this study was to examine the acute effects of resistance exercise performed in hot versus temperate conditions on neuromuscular function, hormone responses and thermal strain in elite female and male power athletes.

Chapter 7, Current Opinion

Through my work designing HA protocols for elite athletes preparing for competitions in hot conditions, I found that best practice HA guidelines were not often attainable within the confines of highly demanding physical preparation and travel requirements of an elite athlete. The aims of the current opinion piece were to review alternative methods of HA and heat exposure, explore the gaps in literature for understanding how HA might be integrated into an athlete's existing training program, and highlight specific athlete considerations for practitioners.

Chapter 8, Invited Comment

Through my work in studies 1-4, varying responses to heat and HA were evident in both thermoregulatory and performance measures, highlighting that individual characteristics and sensitivities must be examined and understood. In this invited Cross

Talk Debate comment, published in the *Journal of Physiology*, I argue that the mean group responses to HA often shown in research, should not necessarily draw inference that such responses will occur in all individual athletes.

1.5 Significance of Thesis

The results of this thesis holds significance for athletes preparing to compete at pinnacle events held in hot locations. At the onset of this thesis, New Zealand National Sports Organisations, with support from HPSNZ, had their eyes set on the 2016 Rio Summer Olympic Games, where the average weather conditions in August are much hotter compared with New Zealand's winter months. Through addressing questions related to how heat training and HA could be used to enhance performance in elite athletes, practical applied strategies were developed to be used by various sports within their respective campaigns, preparing for the Games. Given that many pinnacle events are held in the Northern Hemisphere, during the warmest summer months, athletes living and training in the Southern Hemisphere will always have the added challenge of travelling from winter months; thus, heat training and HA may be even more important for such athletes. Lastly, the use of heat training to enhance training adaptation and performance has the potential to positively impact a number of sports.

CHAPTER TWO: HEAT STRESS &

HEAT ACCLIMATION:

PHYSIOLOGICAL RESPONSES &

EFFECTS ON PERFORMANCE IN

TRAINED INDIVIDUALS

LITERATURE REVIEW

2.1 Introduction

The aim of this section is to provide background literature on heat stress and heat acclimation (HA) and their effects on performance in trained athletes. First, the physiological and thermoregulatory adjustments caused from training and competing in the heat will be reviewed here, along with the resulting impact on performance. Second, a summary of traditionally understood HA adaptive responses and the different methods of HA will be explained, as well as the effects of HA on endurance, team sport and neuromuscular performance in trained populations. Finally, the ergogenic potential of heat and HA for various sport performance will be explored. This review of previous research will form the basis for a series of studies that will comprise this thesis.

2.2 Acute Heat Stress

At rest, humans generate heat when foodstuffs are chemically broken down through various enzymatic reactions into adenosine triphosphate (ATP), which is then used as cellular energy (Curtin & Woledge, 1978). In fact, ~70-90% of metabolism goes towards heat release (Gaesser & Brooks, 1975). Through hypothalamic homeostasis control, core temperature is maintained within a small range (36.5-37.5^{o}C), as heat gain (metabolism and the environment (conduction, convection, radiation)), and heat loss (evaporation and the environment) are balanced (Brotherhood, 2008; Romanovsky, 2007). At rest, ambient temperatures ranging from 15-25°C can elicit heat gain from the environment, depending on physical and environmental characteristics (Kingma, Frijns, & van Marken, 2011). During exercise, heat gain can even begin in cooler conditions. For example, endurance performance has been shown

to be maximised at ~10°C, while even seemingly temperate conditions of 20°C can cause greater heat gain and reductions in endurance performance (González-Alonso et al., 1999). During exercise in hot conditions, core temperature gradually rises due to both increases in metabolic work rate and reduced heat loss from the surrounding environment (Galloway & Maughan, 1997). In order to maintain thermal homeostasis, blood flow is redistributed from the core to the skin to allow for heat dissipation through evaporative cooling (Brengelmann, Johnson, Hermansen, & Rowell, 1977; Kenney & Johnson, 1992). This shift in blood flow distribution, combined with a heat-induced increase in sympathetic nervous system activity (Arngrímsson, Stewart, Borrani, Skinner, & Cureton, 2003), causes an increase in heart rate to maintain cardiac output to working musculature (Périard et al., 2011), as well as an increased rate of glycolysis (Edwards et al., 1972), thus resulting in an augmented energy cost for a given exercise intensity (Wingo, Lafrenz, Ganio, Edwards, & Cureton, 2005). This alone has important performance implications, as power production at lactate threshold, a key aerobic performance determinant (Bassett & Howley, 2000), has been shown to be significantly reduced in the heat compared with temperate conditions (de Barros et al., 2011). As exercise duration is extended, the core-to-skin temperature gradient narrows, causing further increases in heart rate and subsequent reductions in stroke volume and $\dot{V}O_2$max (Wingo et al., 2005). This occurrence is collectively termed cardiovascular strain (Cheuvront, Kenefick, Montain, & Sawka, 2010), and is reflected by an exponential decline in high-intensity aerobic performance (Périard et al., 2011). During prolonged aerobic exercise at low- to moderate-intensity, early onset of fatigue or exhaustion has been shown to occur at core temperatures of ~ 40°C (González-Alonso et al., 1999) and is often considered as a 'critical' core temperature at which volitional

hyperthermic-induced exhaustion occurs (Nielsen et al., 1993), sometimes referred to as central fatigue. Components of central fatigue include reductions in cerebral blood flow and/or reduced motor unit recruitment when environmental heat stress causes hyperthermia (Nybo, 2008). Heat-induced central fatigue also explains impairments in voluntary muscle activation and maximal voluntary contraction (Morrison, Sleivert, & Cheung, 2004; Thomas, Cheung, Elder, & Sleivert, 2006), as well as repeated sprint performance (Drust et al., 2005). From a perceptual standpoint, athletes training or competing in the heat will feel hotter (thermal sensation) and uncomfortable (thermal discomfort) (Gagge, Stolwijk, & Hardy, 1967), and their perceived effort (rating of perceived exertion, RPE) for a given workload will increase (Galloway & Maughan, 1997). While no single mechanism is solely responsible, the collective response of acute heat-induced hyperthermia is a reduction in exercise performance.

2.3 Heat Stress and Performance

Given the abovementioned summary of the thermal and physiological strain of training or competing in hot and humid environments, it is not surprising that the consequences of hyperthermia (core temperature > 38.5ºC (Bligh & Johnson, 1973)) will ultimately diminish an athlete's performance in the unacclimated state (Armstrong & Dziados, 1986; Sawka et al., 1985). The effects of heat stress on exercise performance has been examined thoroughly (de Barros et al., 2011; Galloway & Maughan, 1997; Nielsen et al., 1993; Périard, Caillaud, & Thompson, 2012; Périard et al., 2011; Shvartz et al., 1977; Tatterson et al., 2000), with the extent of hyperthermic-induced performance impairment being largely dependent on the duration and intensity

of exercise (Nybo, 2008). In trained populations, studies have shown significant heat-induced impairments in endurance (Galloway & Maughan, 1997; Périard et al., 2011; Tatterson et al., 2000), team sport (Nybo et al., 2013; Racinais et al., 2012), and sprint performance (Falk, Radom-Isaac, et al., 1998), compared with performance in temperate conditions. Table 2.1 provides a summary of research findings conducted in moderate- to highly-trained individuals across varied sport classifications. Amongst these studies, few include highly-trained subjects, in which heat-induced performance decrements would have the greatest consequences in terms of performance pressure on the world stage (Hopkins, Hawley, & Burke, 1999).

Endurance performance

The effects of heat stress on endurance performance has been most researched, with early studies in the area that focused on long-duration exercise in heat stress in occupational settings (Nybo, Rasmussen, & Sawka, 2014). Research has shown that endurance capacity, as shown through time to exhaustion (TTE) trials, is impaired in hot conditions (Galloway & Maughan, 1997; González-Alonso et al., 1999; Périard et al., 2012; Périard et al., 2011). In moderate- to highly-trained males, aerobic performance trials in the heat (30-32ºC) have resulted in endurance performance decrements ranging from 2.4-6.5% (Galloway & Maughan, 1997; Tatterson et al., 2000), which may be attributed to increases in body temperature, sweat rate, heart rate and RPE (Galloway & Maughan, 1997) (Table 2.1). Such performance decrements might be further explained through increases in skin blood flow and subsequent reductions in stroke volume and peak $\dot{V}O_2$, shown during maximal sustained TT efforts in hot conditions (Périard et al., 2011). As hyperthermic-induced performance impairment is largely dependent on the

Table 2.1 Effects of heat stress on physiological and performance variables in moderately- to highly-trained participants.

Study	Participants	Conditions	Mode	Performance test(s)	Physiological responses (HOT vs. CON)	Performance (HOT vs. CON)
Endurance performance						
Galloway et al. 1997	n = 8 males Mod-trained	4ºC, 10ºC, 20ºC, 30ºC	Cycling	TTE @ 70% $\dot{V}O_2$peak	↑ T_c, T_{sk}, HR, SR, RPE (30ºC vs. other conditions)	36% ↑ TTE (30ºC vs. 20ºC) (equivalent to ~2.4% ↓ TT performance)
Tatterson et al. 2000	n = 11 males Highly-trained	CON = 23ºC, 60% RH; HOT = 32ºC, 60% RH	Cycling	30 min TT	↑ T_{sk}, SR, HR	6.5% ↓ MP0
Periard et al. 2011	n = 8 males Well-trained	CON = 20ºC; HOT = 32ºC	Cycling	40 km TT	↑ T_c, T_{sk}, skin BF, ↓ SV, MAP, peak $\dot{V}O_2$	7.5% ↑ TT

Team sport performance

Hayes et al. 2014	n = 11 males Mod-trained	CON = 21ºC, 49% RH; HOT_1 = 34ºC, 87% RH; HOT_2 = 40ºC, 33% RH	IS	IS = 20 x 10 s rest, 5 s max sprint, 105 s AR	↑ T_c, T_{sk}, HR, RPE, TS, ↔ $\dot{V}O_2$, Bla, (HOT_1 and HOT_2 vs. CON)	↔ PPO and work done (sprints 1-14), ↓ PPO and work done (sprints 15-20)
Morris et al. 2000	n = 16 females Mod-trained	CON = 23ºC, 43% RH; HOT = 36ºC, 48% RH	IS	Part A = LIST Part B = 60 s @ 100% $\dot{V}O_2$max, 6 s RI to exhaustion	↑ T_c, HR, SR, BG, RPE, ↔ Bla, PV	2.2% ↑ 15-m sprint speed, 25% ↓ distance run
Nybo et al. 2013	n = 17 males Well-trained	CON = 21ºC; HOT = 43ºC	Football	Football match; pre and post RS = 3 x 30 m (25 s AR), MVC, Yo-Yo IR1	↑ T_c, T_m, ↓ CK, myoglobin, ↔ muscle glycogen, Bla, BM loss	7% ↓ total distance covered, 26% ↓ total high-intensity running, ↔ RS, MVC, Yo-Yo IR1 post-match
Racinais et al. 2012	n = 19 males Well-trained	CON = 21ºC; HOT = 43ºC	Football	GPS measurements during a football match	None	6.0% ↓ total distance covered, 16.4% ↓ total high-intensity running
Sunderland et al. 2005	n = 9 females Well-trained	CON = 19ºC, 51% RH; HOT = 30ºC, 38% RH	IS, Skills	LIST, field hockey skill test	↑ T_c, RPE, BG, serum aldosterone, SR, ↔ serum cortisol, BLa, PV	6% ↓ field hockey skill performance, 7.5% ↑ 15-m sprint time, ↔ total distance run

Neuromuscular performance

Backx et al. 2000	n = 8 males Mod-trained	CON = 22°C, 30% RH; HOT$_1$ = 30°C, 85% RH; HOT$_2$ = 40°C, 40% RH	RS	3 x 30 s max sprints (30 s AR) pre and post 60 min PR	↑ Osmolality (HOT$_2$ vs. HOT$_1$ and CON), ↔ BLa, HR, $\dot{V}O_2$, $\dot{V}CO_2$	↔ MPO, PPO (HOT$_1$ and HOT$_2$ vs. CON)
Falk et al. 1998	n = 11 males Mod-trained	CON = 22°C, 40% RH; HOT = 35°C, 30% RH	RS	Set 1 (5 x 15 s max sprints; 30 s RI), 60 min passive recovery, Set 2	↔ BLa Na$^+$, Cl$^-$, K$^+$, ↔ PV, $\dot{V}O_2$, HR, ↑ T$_c$ (~0.3^{o}C), ↑ T$_{sk}$ (~4°C)	Set 1 = 8.4% ↑ MPO, Set 2 = 4.1% ↓ MPO
Girard et al. 2014	n = 12 males Well-trained	CON = 22°C, 70% RH; HOT = 36°C, 35% RH	RS, SS, Power	RS = 3 x 15 m (15-s RI), SS = 15 m max sprint, VJ	None	↔ RS, SS, VJ
Hedley et al. 2002	n = 12 males Mod-trained	CON = 23°C, 65% RH; HOT = 65-75°C, 15% RH	Power, Strength	30 min passive rest in CON or HOT; 1 RMs (leg and bench press), VJ, reps to failure at 75% RM (leg and bench press)	↑ HR (58 bpm), SBP (23 mmHg), TDC (6 AU), ↓ DBP (16 mmHg)	↔ bench press 1 RM, ↓ (-4%) leg press 1 RM, ↑ (3%) VJ height, ↓ reps to failure (leg press = -29%, bench press = -16%)
Linnane et al. 2004	n = 11 males Mod-trained	CON = passive rest, ex in 22°C; HOT = HWI (43°C), ex in 44°C	SS	30 min HWI, 2 x 30 s max sprint, 4 min RI	↑ T$_c$ (1°C), BG, Bla	Sprint 1 = ↑ PPO (7%), MPO (6%), Sprint 2 = ↔ PPO, ↓ MPO (3%)

Mohr et al. 2013	n = 19 males Highly-trained	CON = 12ºC; HOT = 30ºC	Power	5 CMJs every 5 s pre and post soccer match	69% ↑ SR	6% ↓ mean CMJ performance, ↔ peak CMJ performance

AR = active recovery, BF = blood flow, BG = blood glucose, Bla = blood lactate, BM = body mass, CK = creatine kinase, CMJ = counter movement jump, CON = control condition, DBP = diastolic blood pressure, GPS = global positioning system, HR = heart rate, HOT = hot condition, HWI = hot water immersion, IS = intermittent sprinting, LIST = Loughborough Intermittent Shuttle Test, MAP = mean arterial pressure, Mod = moderately, MPO = mean power output, MVC = maximal voluntary contraction, PPO = peak power output, PR = passive recovery, PV = plasma volume, Q = cardiac output, RH = relative humidity, RI = recovery interval, RM = repetition maximum, RPE = rating of perceived exertion, RS = repeated sprints, SBP = systolic blood pressure, SR = sweat rate, SS = single sprint, SV = stroke volume, T_c = core temperature, TDC = thermal discomfort, T_m = muscle temperature, T_{sk} = skin temperature, TT = time trial, TTE = time to exhaustion, VJ = vertical jump, $\dot{V}O_2$ = oxygen uptake, $\dot{V}O_2$max = maximal oxygen uptake, Yo-Yo IR1 = intermittent recovery test level 1, ↑= statistically significant or meaningful increase, ↓ = statistically significant or meaningful decrease, ↔ = no statistical or meaningful difference or change between HOT vs. CON.

duration and intensity of exercise (Nybo, 2008), the majority of endurance events would be negatively impacted if an athlete is not prepared for the heat.

Team sport performance

In trained team sport athletes, both match play and determinants of team sport performance are reduced in the heat. During semi-professional football matches in the heat, total distance run and high-intensity running performance have been shown to be reduced by 6-7% and 16-26% respectively, compared with a football match in temperate conditions (Nybo et al., 2013; Racinais et al., 2012). Determinants of team sport performance, including single sprint speed, intermittent sprint ability and game-specific motor-skills (Bishop & Girard, 2013), have also been examined in team sport athletes. Both single sprint performance (-2%) (Morris, Nevill, & Williams, 2000; Sunderland & Nevill, 2005) and game-specific skills (-6%) (Sunderland & Nevill, 2005) have been shown to be impaired during team sport-type training sessions in the heat (30-36ºC, 48-51% RH). The effects of heat on intermittent sprinting (short maximal sprint efforts, followed by 60-300s recovery durations (Girard, Mendez-Villanueva, & Bishop, 2011)) is less clear, with studies showing large decrements (-25%) (Morris et al., 2000), minimal decrements (-4%) (Hayes, Castle, Ross, & Maxwell, 2014), or no effects of additional heat (Sunderland & Nevill, 2005). These discrepancies may be due to variations in total heat load and the intermittent-sprinting protocol used, thus varying the degree of heat strain acquired by subjects.

Neuromuscular performance

From an applied sport perspective, neuromuscular performance encompasses sprinting, strength and power, which are determinants of performance for speed and

power events (Haff & Nimphius, 2012). In laboratory settings, sprint performance has been shown to improve in the heat when single (Sargeant, 1987) and repeated efforts (Ball et al., 1999; Girard et al., 2013) are performed in untrained subjects. This enhanced performance may be attributed to increases in muscle temperature that elicit higher peak power outputs and cadence, thus enhancing all aspects of the power-velocity curve (Sargeant, 1987). Few studies, however, have examined the effects of heat stress on single sprint performance in trained individuals. In moderately-trained subjects, Falk et al. (1998) and Linnane et al (2004) showed that acute heat stress enhances power production (+7-8%) during an initial sprint, however, power of a second sprint was impaired (-3 to -4%) following short-term (4-min) (Linnane, Bracken, Brooks, Cox, & Ball, 2004) and long-term (60-min) (Falk, Radom-Isaac, et al., 1998), recovery intervals in the heat. These results are in line with findings in untrained subjects, where performance through single and repeated sprints have been shown to increase in hot conditions in concert with increases in body temperature (Ball et al., 1999; Girard et al., 2013; Sargeant, 1987). However, performance can also be markedly impaired with the onset of hyperthermia (>38.5°C) (Drust et al., 2005; King et al., 1985). Similarly, repeated sprinting (multiple short duration maximal sprint efforts, followed by < 60 s recovery periods (Girard et al., 2011) seems to be enhanced in the heat, if core temperature remains low (Girard et al., 2013), and impaired with hyperthermia (Drust et al., 2005) in untrained subjects. Conversely, others have shown that heat has no effect on repeated sprint ability in well-trained athletes, despite high core temperature values (39.4°C) (Girard, Christian, Racinais, & Périard, 2014; Périard et al., 2014). Therefore, it is unknown to what degree hyperthermia induces decrements in sprint performance in trained athletes.

In line with heat-induced enhancements in single sprint performance, power production, as measured by vertical jump height, has also been shown to improve (3%) following passive heating (Hedley, Climstein, & Hansen, 2002) in moderately-trained individuals. In contrast, following a football match in the heat (30°C), counter movement jump (CMJ) power was shown to decline, although core temperature was not assessed to determine if the drop in performance was related to hyperthermia (Mohr & Krustrup, 2013).

The effects of heat stress on maximal strength in trained populations have also shown varied findings. Hedley et al. (2002) showed reductions in maximal strength (-4%) and strength endurance (-16 to -29%), following passive heating (65-75°C, 15%RH), in resistance-trained men (Hedley et al., 2002). Conversely, Nybo et al (2013) showed no change in maximal voluntary contraction following a football match in 43°C in semi-professional players (Nybo et al., 2013). Overall, the effects of heat on muscle force production from the literature is equivocal. Some studies have shown enhanced short-term maximal force production following passive or dynamic heat exposure (Ball et al., 1999; Sargeant, 1987), while others have shown no effect with (Cheung & Sleivert, 2004; Ftaiti, Grélot, Coudreuse, & Nicol, 2001) or without hyperthermia (Girard, Brocherie, & Bishop, 2015). During submaximal sustained contractions, hyperthermia has been shown to impair central neuromuscular activation, reducing isometric endurance capacity (Morrison et al., 2004). Further research is warranted to determine the effects of heat stress on maximal strength and strength endurance in trained populations.

2.4 Heat Acclimation

Physiological adaptations

Heat acclimation can mitigate the performance impairments, discussed in the previous section, for athletes competing in hot and humid environments (Bergeron, Bahr, Bartsch, et al., 2012). Heat acclimation (artificial heat exposure, indoors), or heat acclimatisation (natural heat exposure, outdoors), involves multiple exercise-training sessions in hot conditions, that induce physiological adaptations that attenuate heat-induced performance impairments to protect against heat stress and EHI (Périard et al., 2015; Taylor, 2014; Tyler et al., 2016). It has been shown that HA over several days can induce physiological adaptations that can significantly reduce the effects of thermal strain and restore performance in the heat (Castle et al., 2011; Garrett, Creasy, et al., 2011; Lorenzo et al., 2010). The classical heat-acclimated state is characterised by an expanded thermoregulatory range consisting of an increased capacity to dissipate heat, enhanced protection from thermal injury, and improved efficiency and endurance in the heat (Horowitz, 2001). The expanse in thermoregulatory function is due to several physiological adaptations, including a decrease in core temperature (Adams et al., 1960; Nielsen et al., 1993), skin temperature (Cheung & McLellan, 1998) and heart rate (Nielsen et al., 1993), which is related to a drop in sympathetic nervous system activity through decreases in circulating norepinephrine levels (Hodge, Jones, Martinez, & Buono, 2013). In addition, HA enhances evaporative cooling through an earlier onset of sweating (Roberts, Wenger, Stolwijk, & Nadel, 1977), more dilute sweat and an increase in sweat rate (Racinais et al., 2012), which is partially attributed to a concurrent plasma volume expansion shown with HA (Garrett, Goosens, Rehrer, Patterson, &

Cotter, 2009; Garrett, Rehrer, & Patterson, 2011; Racinais et al., 2012). Blood plasma volume has been shown to increase from 4-13% with 5-to-10 day exposure periods (Garrett et al., 2009; Garrett, Rehrer, et al., 2011; Nielsen et al., 1993; Racinais et al., 2012) and likely accounts for most of the HA-induced increases in both cardiac output and stroke volume (Nielsen et al., 1993; Rowell, Kraning, Kennedy, & Evans, 1967). In terms of metabolic effects, the rate of fuel utilisation is adjusted through a lowered reliance on glycolysis and enhanced lipolysis following HA compared with an initial heat exposure (Febbraio et al., 1994). Subjectively, RPE, thermal sensation and thermal discomfort are also reduced with HA (Casadio, Kilding, Siegel, Cotter, & Laursen, 2016; Castle et al., 2011; Kelly, Gastin, Dwyer, Sostaric, & Snow, 2016; Neal, Corbett, Massey, & Tipton, 2015; Sunderland et al., 2008), and athletes tend to feel as though they are able to cope with hot and unpleasant conditions. Finally, HA expands the thermoregulatory range at a cellular level through increasing the expression (Gibson et al., 2015) and serum concentration (Magalhães et al., 2010; Yamada, Amorim, Moseley, Robergs, & Schneider, 2007) of heat shock proteins. Heat shock proteins are upregulated by both heat stress and exercise, offering cytoprotection from thermal injury (Moseley, 1997). Taken together, the enhanced thermoregulatory function enhances exercise economy (Armstrong & Maresh, 1991) and improves endurance (Tatterson et al., 2000), intermittent running (Sunderland et al., 2008) and sprint performance (Castle et al., 2011) in the heat, in trained individuals.

Methods

Multiple factors must be considered prior to designing an HA protocol for an athlete or team. Variables to specify include exercise mode, intensity, and duration,

heat load, number of days, daily vs. intermittent exposure, as well as the timing of HA prior to competition in the heat. The methods found in literature used to induce HA adaptations in trained athletes have varied in number of days (4-14 d), exercise duration (~30-90 min), exercise format (low- to moderate-intensity steady-state exercise vs. high-intensity intermittent exercise vs. controlled hyperthermia) and intensity (45-80% $\dot{V}O_2max$). Sufficient adaptations to promote performance improvements in the heat have been shown in both short-term (4-7 d) (Buchheit, Voss, Nybo, Mohr, & Racinais, 2011; Chen, Tsai, Lin, Lee, & Liang, 2013; Garrett et al., 2009; Sunderland et al., 2008) and moderate-term (8-14 d) HA protocols (Buchheit et al., 2013; Castle et al., 2011; Lorenzo et al., 2010; Racinais et al., 2012). Short-term protocols are commonly implemented in applied sport settings due to the ease of fitting the intervention into a single training week, which means less time disrupting normal training (Chalmers et al., 2014). However, several short-term HA studies have shown only partial HA adaptations, failing to reduce core temperature (Petersen et al., 2010; Racinais et al., 2012; Wingfield, Gale, Minett, Marino, & Skein, 2016). The reduction in core temperature following HA may be a key marker indicating protection in an athlete from performance impairments (Tyler, Sunderland, & Cheung, 2015) which may lower thermal injury risk during competition in the heat (Binkley et al., 2002). Recent reviews have demonstrated that moderate- to long-term ($\geq$ 15 d) HA is necessary for full thermoregulatory adaptations to occur and offers greater performance improvements in the heat (Guy, Deakin, Edwards, Miller, & Pyne, 2015; Tyler et al., 2016). While short-term HA may be easier to integrate into the schedule of an elite athlete, considerations should be given to the event type, the expected conditions, and the individual athlete(s) before a HA protocol length is chosen.

Based on differences shown between short-term and longer HA periods, it is clear that the extent of acclimation is determined largely by the volume of exercise, the degree of thermal load and the number of heat exposures (Garrett, Rehrer, et al., 2011). Consecutive days of HA has been shown to be superior compared with scheduling HA sessions intermittently, as the accumulation of daily HA results in a greater net gain of adaptations (Gill & Sleivert, 2001). With daily HA sessions, the time course of most adaptations appears to be 7 days, as thermoregulatory adjustments have been shown to plateau beyond this point (Weller, Linnane, Jonkman, & Daanen, 2007). Cardiovascular adaptations, including reduced heart rate (Hodge et al., 2013) and plasma volume expansion (Pandolf, Burse, & Goldman, 1977) occur more readily in just 3-4 days. Indeed, the rate of adaptation has been shown to be fitness dependant, with highly-trained athletes exhibiting many HA benefits in 4-6 days, while untrained participants have been shown to take 7-9 days (Pandolf et al., 1977). Retention of adaptations has been shown for 1 week following HA, with gradual decay occurring after 2-4 weeks, depending on HA length (Garrett et al., 2009). Re-acclimation may occur with fewer sessions (Weller et al., 2007; Wyndham & Jacobs, 1957). As highly-trained individuals may have slower rates of HA decay (Pandolf et al., 1977), and because periodic exposure to heat following HA can allow for the retention of HA over several weeks (Moseley, 1997), it may be worth periodising HA intermittently throughout a competitive season. Furthermore, a 'thermal memory' or 'thermal plasticity' concept may exist, where rapid re-acclimation is possible in those who have used HA routinely (Horowitz, 2001). Overall, the periodisation of HA poses complex questions around how best to schedule HA into an athlete's training and competition calendar. Such questions and considerations are extensively explored within a current

opinion piece (chapter 7) later in this thesis, which reviews HA alternatives, examines gaps in literature around understanding the real world application of HA for athletes, and highlights athlete considerations for practitioners.

2.5 Heat Acclimation and Performance

Although the adaptive potential for HA is reduced with increased training status (Cheung & McLellan, 1998), this does not negate the fact that thermoregulatory enhancement improves performance in the heat, irrespective of whether participants are untrained (Weller et al., 2007), or highly-trained (Garrett, Creasy, Rehrer, Patterson, & Cotter, 2012). Indeed, numerous studies have shown physiological and performance improvements in trained athletes (Buchheit et al., 2013; Buchheit et al., 2011; Castle et al., 2011; Chen et al., 2013; Garrett et al., 2009; Garrett, Rehrer, et al., 2011; Lorenzo et al., 2010). Specifically, HA has been shown to improve endurance performance (Garrett et al., 2012; Garrett et al., 2009; Lorenzo et al., 2010), team sport intermittent running performance (Buchheit et al., 2013; Buchheit et al., 2011; Sunderland et al., 2008) and repeated sprint performance (Brade, Dawson, & Wallman, 2013; Castle et al., 2011) in moderate- to highly-trained participants. Table 2.2 reviews studies conducted with athletes or focusing on specific-sport events, where a heat stress test, performance measure or both were performed to assess the effects of HA on performance in trained populations.

Endurance performance

Several studies (Table 2.2) have examined the effects of short- and moderate-term HA, in moderately-trained to elite level athletes, showing thermoregulatory range expansion and enhanced endurance performance in the heat (Chen et al., 2013; Garrett et al., 2012; Garrett et al., 2009; Karlsen, Nybo, et al., 2015; Lorenzo et al., 2010; Nielsen et al., 1993; Nielsen, Strange, Christensen, Warberg, & Saltin, 1997; Voltaire et al., 2002). Across these studies, different modes of HA have been used, including steady-state

Table 2.2 Effects of heat acclimation on physiological and performance variables across different sport categories in trained participants.

Study	Participants	Sport	HA protocol	Method	Performance measure(s)	Physiological responses (Post vs. Pre HA)	Performance (Post vs. Pre HA))
Endurance performance							
Casadio et al. 2016	n = 2 males Elite	Sailing	5 d, 60 min @ 60-70% $\dot{V}O_2$max; T_a, 35°C, 60% RH	SSE	Submaximal cycling	↓ T_c (0.5°C), HR (14 bpm), TDC (1 AU), RPE (2 AU), ↔ PV	↑ MPO (%)
Chen et al. 2014	n = 7 males Mod-trained	Racquet sports	5 d; 25-45 min @ ~ VT; T_a, 38°C, 52% RH	SSE	GXT	↓ HR, ↑ SR, ↔ T_{sk}	↑ GXT (7%)
Febbraio et al. 1994	n = 13 males Well-trained	Cycling	7 d; 90 min @ 50% $\dot{V}O_2$max; T_a, 40°C, 20% RH	SSE	None	↓ T_c (0.4°C), T_m (0.5°C), HR (11 bpm), RER (0.04), Bla (~1 mmol·L^{-1}), BG (~1 mmol·L^{-1}), ↔$\dot{V}O_2$	None
Garrett et al. 2009	n = 10 males Mod-trained	Cycling	5 d; 90 min @ T_c (38.5°C); T_a, 39.5°C, 60% RH	CHT	GXT	↓ T_c (0.3°C), HR (13 bpm), ↑ PV (4.2%)	↑ GXT duration (14%)
Garrett et al. 2012	n = 8 males Highly-trained	Rowing	5 d; 90 min @ T_c (38.5°C); T_a, 39.5°C, 60% RH	CHT	2 km rowing TT	↓ T_c (0.3°C), HR (14 bpm), ↑PV (4.5%), SR (0.9%)	↓ TT duration (1%)

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Gill et al. 2001	n = 14 Mod-trained	Rowing	10 d (Consecutive or intermittent); 30 min @ 70% $\dot{V}O_2$peak; T_a, 38ºC, 70% RH	SSE	None	Consecutive, ↓ T_c (1.0ºC), HR (13 bpm), RPE (5 AU) ↔ SR; Intermittent, ↓ T_c (0.6ºC), ↔ HR, RPE, SR	None
Houmard et al. 1990	n = 9 males Mod-trained	Running	9 d; LIHA, 60 min @ 50%, or MIHA, 30 min @ 75% $\dot{V}O_2$peak; T_a, 40ºC, 27% RH	SSE	None	LIHA and MIHA = ↓ T_c (0.5ºC), HR (10-14 bpm), $\dot{V}O_2$, ↔ T_{sk}, PV, SR	None
Karlsen et al. 2015	n = 9 males Well-trained	Cycling	14 d; ~60-160 min @ varying intensities; ~34ºC, 18% RH	Routine training	43 km TT	↑ SR (20%), PV (3.7%), ↔ HR	↓ TT duration (14%)
Lorenzo et al. 2010	n = 12 males Well-trained	Cycling	10 d; 90 min @ 50% $\dot{V}O_2$max; 40ºC, 30% RH	SSE	$\dot{V}O_2$max, 60 min TT	↓ T_c (0.5ºC), T_{sk} (1.1ºC) HR (15 bpm), ↑ PV (6.5%), SR (22%), ↔ RPE, Bla	↑ $\dot{V}O_2$max (8%), ↑ TT work done (8%)
Neal et al. 2015	n = 10 males Well-trained	Cycling	5 d; 90 min @ T_c (38.6ºC); T_a, 40ºC, 50% RH	CHT	20 km TT, GXT (22ºC, 60% RH)	↓ T_c (0.3ºC), HR (7 bpm), RPE, ↑ SR (7%), ↔ PV (1.2%)	↑ PPO (6 W) and LT (16 W) during GXT, ↔ TT duration
Nielsen et al. 1993	n = 13 males Well-trained	Cycling	9-12 d; TTE @ 60% $\dot{V}O_2$max; T_a, 40ºC, 10% RH	TTE	TTE @ 60% $\dot{V}O_2$max	↓ T_c, T_{sk}, HR, ↑ PV, Q, SV SR	↑ TTE duration (66%; equivalent to ~ 4% TT duration)

| Nielsen et al. 1997 | n = 13 males Well-trained | Endurance sports | 8-13 d; TTE @ 150 W; T_a, 35°C, 87% RH | TTE | TTE @ 45% $\dot{V}O_2max$ | ↓ T_c (0.3°C), T_{sk} (0.2°C), HR (12 bpm), ↑ PV (9%), SR (26%) | ↑ TTE duration (16%; equivalent to ~ 1% TT duration) |
| Voltaire et al. 2002 | n = 9 males Elite | Triathlon | 14 d; Routine training (16 h·wk^{-1}); T_a, 33°C, 87% RH | Routine training | GXT | ↓ T_c (0.4°C), HR (25 bpm), ↑ SR (55%) | ↑ MAV (4%) |

Team sport performance

Buchheit et al. 2011	n = 15 males Well-trained	Football	7 d; Routine training; T_a, 40°C, 27% RH	Routine training	Yo-Yo IR1 (22°C)	↓ HRex, HRV, ↑ PV (7%), ↔ HRR, RPE	↑ Yo-Yo IR1 running distance (7%)
Kelly et al. 2016	n = 7 males per group Mod-trained	Australian football	5 d (over a 9 d period); 27 min HIIT; T_a, 39°C, 34% RH	HIIT	None	↓ Bla, HR, RPE, TDC, ↔ T_c, T_{sk}	None
Petersen et al. 2010	n = 6 per group Mod-trained	Cricket	4 d; 30-45 min RS protocol; T_a, 30°C, 60% RH	HIT	Cricket Run Test (24°C, 48% RH)	↔ T_c, T_{sk}, SR, ↓HR, TDC, TS	↔ Run test total duration

Racinais et al. 2012	n = 19 males Well-trained	Football	6 d; Routine training; T_a, 38-43°C, 12-30% RH	Routine training	Football match GPS (CON = 21°C, Post HA = 43°C)	$\leftrightarrow T_c$, T_{sk}, HR, ↑ SR (36%), PV (4%)	↓ Match running distance (6%), ↓ high-intensity running (16%) (Post HA vs. CON)
Racinais et al. 2014	n = 19 males Elite	Australian football	14 d; Routine training; T_a, 31-33°C, 34-50% RH	Routine training	Yo-Yo IR2 (23°C), Drills (32°C)	↓T_{sk} (0.4°C), HR (11 bpm), TS (1 AU), ↑ SR (36%), PV (4%), $\leftrightarrow T_c$, SR	↑ Yo-Yo IR2 running distance (~44%), ↑ drills distance (5%)
Sunderland et al. 2005	n = 9 females Well-trained	Field hockey	4 d; 30-45 min IS; T_a, 30°C, 27% RH	IS	LIST, 15 m SS	↓T_c, HR, TDC, $\leftrightarrow$ PV, SR, RPE	↓ LIST distance run (36%), $\leftrightarrow$ 15-m SS

Neuromuscular performance

Brade et al. 2013	n = 10 males Mod-trained	Team sports	5 d (over a 10 d period); ~30-50 min HIIT; T_a, 35°C, 60% RH	HIIT	RS	↓ T_c (0.3°C), T_{sk} (0.2°C), ↑ SR (42%), $\leftrightarrow$ HR, RPE, TS	↑ Work done (5%)
Castle et al. 2011	n = 8 Mod-trained	Team sports	10 d; 60 min @ 50% $\dot{V}O_2$peak; T_a, 33°C, 52% RH	SSE	IS	↓ T_c (0.4°C), HR (18 bpm), RPE (2 AU), TS (1 AU), ↑ plasma HSP, $\leftrightarrow$ PV	↑ PPO (2%)

Wingfield et al. 2016	n = 10 males (per group) Mod-trained	Team sports	5 d; HITHA = 30 min HIT; LIHA = 90 min @ 40% $\dot{V}O_2$peak; T_a, 33°C, 60% RH	HIHA vs. LIHA	VJ, CMJ, MVC, RS, 20 km TT	HITHA = ↓HR, TS, ↔ T_c, T_{sk}; LIHA = ↓T_c, T_{sk}, HR, ↔ TS	HITHA = ↔ VJ, CMJ, MVC, RS, TT; LIHA = ↓ TT duration (6%), MVC (7%), ↔ VJ, CMJ, RS

BG = blood glucose, Bla = blood lactate, BM = body mass, CHT = controlled hyperthermia, CMJ = counter movement jump, CON = control condition, GPS = global positioning system, HA = heat acclimation, HIHA = high-intensity heat acclimation, HIIT = high-intensity interval training, HR = heart rate, HRex = heart rate exercise, HRR = heart rate recovery, HSP = heat shock protein, HST = hest stress test, HOT = hot condition, IS = intermittent sprinting, LIHA = low-intensity heat acclimation, LIST = Loughborough Intermittent Shuttle Test, MAV = maximum aerobic velocity, MIHA = moderate-intensity heat acclimation, Mod = moderately, MPO = mean power output, MVC = maximal voluntary contraction, PPO = peak power output, PV = plasma volume, Q = cardiac output, RH = relative humidity, RPE = rating of perceived exertion, RS = repeated sprints, SR = sweat rate, SS = single sprint, SSE = steady-state exercise, SV = stroke volume, Tc = core temperature, TDC = thermal discomfort, Tm = muscle temperature, Tsk = skin temperature, TT = time trial, TTE = time to exhaustion, VJ = vertical jump, $\dot{V}O2$ = oxygen uptake, $\dot{V}O2$max = maximal oxygen uptake, $\dot{V}O2$peak = peak oxygen uptake, VT = ventilatory threshold, Yo-Yo IR1 = intermittent recovery test level 1, Yo-Yo IR2 = intermittent recovery test level 2, ↑= statistically significant or meaningful increase, ↓ = statistically significant or meaningful decrease, ↔ = no statistical or meaningful difference or change between Post vs. Pre HA.

exercise (Casadio et al., 2016; Chen et al., 2013; Febbraio et al., 1994; Gill & Sleivert, 2001; Houmard et al., 1990; Lorenzo et al., 2010), the controlled hyperthermia technique (Garrett et al., 2012; Garrett et al., 2009), and routine training sessions, consisting of both steady-state and high-intensity interval training (Karlsen, Racinais, et al., 2015; Voltaire et al., 2002). Heat acclimation for endurance events typically consists of 60-90-min heat training sessions at 50-70% $\dot{V}O_2$max, which is sufficient to induce reductions in core temperature (0.3-0.5ºC), along with other classical HA adaptations (Casadio et al., 2016; Febbraio et al., 1994; Garrett et al., 2012; Garrett et al., 2009; Lorenzo et al., 2010; Neal et al., 2015; Nielsen et al., 1997; Voltaire et al., 2002). Enhanced endurance performance has been shown in improve in graded exercise tests (4-14%) (Chen et al., 2013; Garrett et al., 2009; Lorenzo et al., 2010; Voltaire et al., 2002), time to exhaustion trials (16-66%) (Nielsen et al., 1993; Nielsen et al., 1997), and submaximal work output (~9%) (Casadio et al., 2016) in the heat. More relevant to performance in real world endurance events, time trial performance is also improved following short (1-11%) (Garrett et al., 2012; Karlsen, Nybo, et al., 2015) and moderate-term (14%) HA (Karlsen, Nybo, et al., 2015). Overall, HA is clearly a worthwhile preparatory tool for endurance sport athletes aiming to perform at events in hot environments.

Team sports performance

Within the last decade, more research has focused on HA for team sports performance as evidence emerged that heat stress does indeed impair determinants of match-play performance (Morris et al., 2000; Sunderland et al., 2008), and a growing number of pinnacle events are scheduled in hot locations, such as the 2020 Summer

Olympic Games in Tokyo. Heat acclimation for team sports generally consists of routine training camps (Buchheit et al., 2011; Racinais et al., 2014; Racinais et al., 2012), or game-like simulations through intermittent sprint (Sunderland et al., 2008) and high-intensity interval training (Kelly et al., 2016; Petersen et al., 2010) performed in hot conditions. When short-term HA is used with moderate- to well-trained male athletes, partial HA adaptations are evident through reductions in heart rate, thermal perception and RPE (Kelly et al., 2016; Petersen et al., 2010), as well as increases in plasma volume and sweat rate (Racinais et al., 2012). However, such 4-6 d protocols might not be sufficient for lowering core temperature (Kelly et al., 2016; Petersen et al., 2010; Racinais et al., 2012). Conversely, Sunderland et al. (2005) showed that 4 d of intermittent sprint HA resulted in reduced core temperature at the end of their sprint protocol in female athletes. Racinais et al. (2014) examined the effects of a 2-wk training camp in the heat (31-33°C, 34-50% RH) in professional Australian Rules Football players, and showed no change in core temperature during a heat stress test, despite HA eliciting other expected thermoregulatory adaptations (Racinais et al., 2014). In terms of performance, HA has been shown to improve team sport skills (5%) (Racinais et al., 2014) and intermittent running tests of aerobic capacity (7%) (Buchheit et al., 2011) in hot conditions. In a game setting, Racinais et al (2012) showed that following HA, impairments in match running distance (-6%) and high-intensity running (-16%) were still evident in the heat (43°C) compared with a game played in temperate conditions (21°C). While evidence suggests that HA improves aerobic-based test performance (Chalmers et al., 2014), more work is required to optimise HA protocols for team sport athletes so performance is enhanced in hot conditions.

Neuromuscular performance

To date, minimal attention has been given to the effects of heat acclimation on neuromuscular performance, including sprint ability, power production and strength. Heat-induced hyperthermia has been shown to reduce measures of sprint (Falk, Radom-Isaac, et al., 1998), power (Mohr & Krustrup, 2013) and strength (Hedley et al., 2002) performance in trained individuals. Of the few studies that have examined the effects of HA on neuromuscular function, two have shown that sprint performance is enhanced (2-5%) following HA (Brade et al., 2013; Castle et al., 2011) in moderately-trained team sport athletes. The studies varied in HA protocols (Table 2.2) but both resulted in reduced core temperature (0.3-0.4^{o}C), with varied responses shown for adjustments in cardiovascular function and thermal perception (Brade et al., 2013; Castle et al., 2011). It appears that only one study to date has examined the effects of two different modes (low vs. high-intensity) of short-term HA on neuromuscular function in moderately-trained team sports players (Wingfield et al., 2016). The low-intensity HA protocol impaired maximal strength (-7%) during a maximal voluntary contraction, with no effects of HA on power or sprint performance. Conversely, the high-intensity HA protocol showed no change in strength, power, or sprint performance. Given the low number of studies examining the effects of HA on neuromuscular function in trained populations, more work is needed.

2.6 Heat as a Training Tool

Endurance performance

Like altitude or hypoxic training, the environmental stress-induced adaptations of HA, may be used as an ergogenic tool to elicit enhanced performance in temperate conditions (Corbett et al., 2014). Lorenzo examined the effects of 10-d HA (90 min cycling at ~50% $\dot{V}O_2$max power output) on $\dot{V}O_2$max and lactate threshold in highly-trained cyclists in both hot (40°C) and temperate (13°C) conditions. The HA protocol successfully induced thermoregulatory range expansion through an increased core-skin temperature gradient, reduced skin temperature and increased both plasma volume and maximum cardiac output. $\dot{V}O_2$max was increased in both hot (8%) and temperate (6%) conditions, while power output at lactate threshold increased to a similar level (5%) in both environments (Lorenzo et al., 2010). Similar endurance performance benefits of HA have been shown through training camps in hot conditions (Buchheit et al., 2013; Buchheit et al., 2011), and with passive heating through sauna bathing (Scoon, Hopkins, Mayhew, & Cotter, 2007), thus expanding the use of HA to ergogenic status rather than simply heat preparation. Common throughout all these studies documenting the use of HA as a training tool, is a marked increase in plasma volume (6.5-7%) and thus, enhanced cardiac efficiency (Sawka, Convertino, Eichner, Schnieder, & Young, 2000), which could explain the aerobic performance improvements observed. In contrast, others have shown that HA does not improve endurance performance in cool conditions (Chen et al., 2013; Houmard et al., 1990; Karlsen, Racinais, et al., 2015; Keiser et al., 2015). It has been argued (Nybo & Lundby, 2016) that many studies showing an ergogenic effect of HA fail to include control groups (Buchheit et al., 2011; Racinais et

al., 2014), or that training intensity is not always matched between control and heat training groups (Lorenzo et al., 2010). Due to the equivocal findings in the literature, more work is needed to determine the ergogenic potential of HA for endurance performance.

Sprint and power performance

Heat as a training tool to improve sprint and power performance may also have applications in applied sport settings, through its effects on enhancing sprint ability and resistance training adaptations (Figure 2.1). As described in Section 2.3, acute heat stress has been shown to improve sprint and power performance (Falk, Radom-Isaac, et al., 1998; Hedley et al., 2002), in the absence of hyperthermia (Drust et al., 2005). Heat-induced performance enhancements shown may be explained by increases in muscle temperature, greater ATP turnover, and faster muscle nerve conduction velocity (Farina, Arendt-Nielsen, & Graven-Nielsen, 2005; Gray, De Vito, Nimmo, Farina, & Ferguson, 2006). Indeed, for every 1°C rise in muscle temperature, a 4% increase in peak power production can be expected (Sargeant, 1987). This relationship does not always translate to enhanced sprint performance, however, with some studies showing no effect on single sprint (Girard et al., 2014) and repeated sprint (Backx, McNaughton, Crickmore, Palmer, & Carlisle, 2000; Girard et al., 2014) performance in moderate- to well-trained individuals. However, this concept has been utilised as a pre-competition strategy, where passive heating, or heat retention, has been used to attenuate the muscle temperature drop between a warm-up and actual competition, with beneficial performance effects shown in cycling (Faulkner et al., 2012), swimming (McGowan, Thompson, Pyne, Raglin, & Rattray, 2016), skeleton (Cook, Holdcroft, Drawer, &

Kilduff, 2013), and rugby (Kilduff, West, Williams, & Cook, 2013). A better understanding of passive heating and sprinting

HEAT

Acute Heat Stress

↑ thermal strain

↑ muscle temperature
↑ conduction velocity

↑ peak power
↑ peak force

↑ Akt/mTOR signalling
↑ anabolic hormone response

Pre-heating for sprint events?
Enhanced sprint-training quality?

Heat Acclimation

↓ thermal strain

↑ CSA
↑ strength

↑ Strength/power
performance?
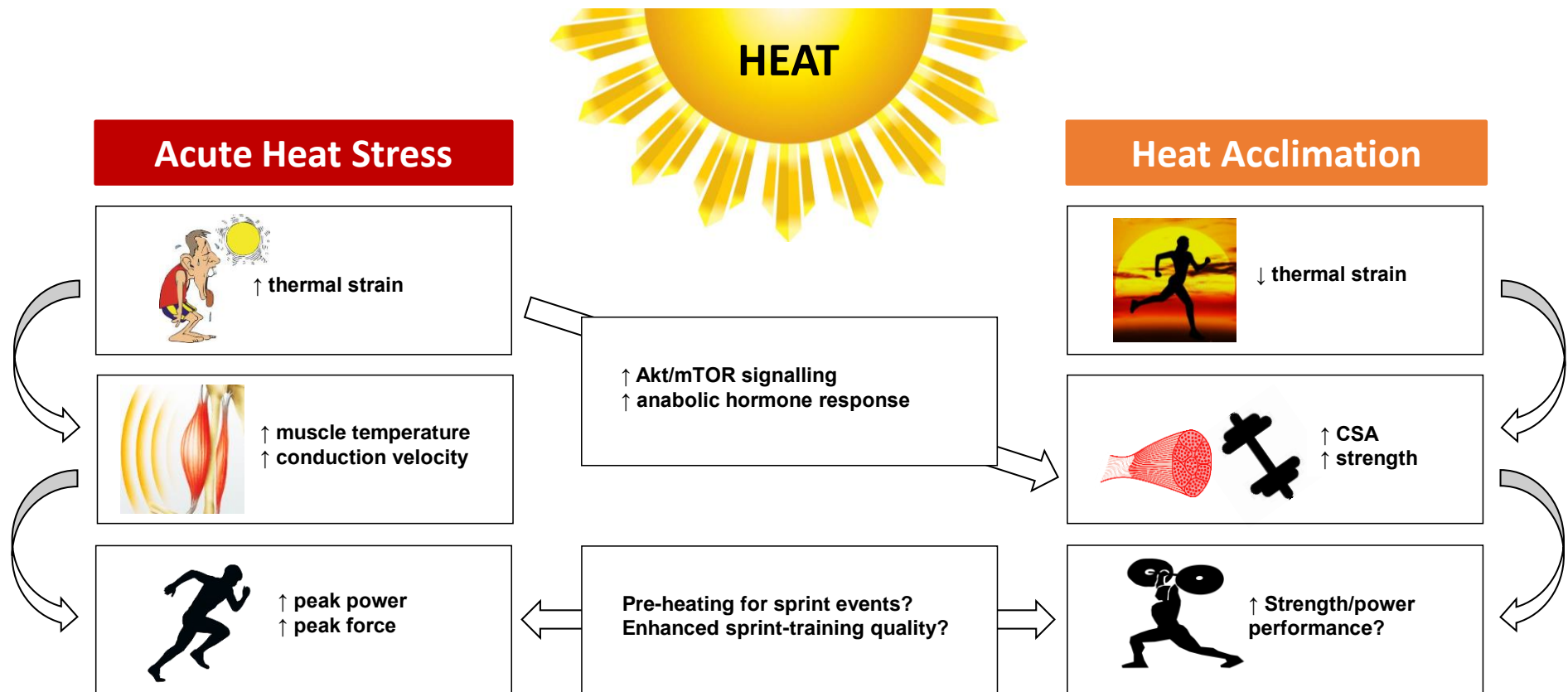
Figure 2.1 Potential applications of acute heat stress and chronic heat acclimation for sprint and power events. Akt/mTOR = Akt/mammalian target of rapamycin, CSA = cross-sectional area.

effects in the heat is needed to determine how best to use heat with elite athletes to enhance sprint and power performance.

Recent research has emerged suggesting that heat stress has an ergogenic potential for enhancing resistance training adaptations. Laboratory-based studies have shown that applying localised heat to skeletal muscle, with or without resistance training, can augment maximal force and muscle cross sectional area, in healthy untrained males (Goto et al., 2011; Goto et al., 2007). Rodent models have shown that heat stimulates the Akt/mTOR signalling pathway, a key regulator of protein synthesis and hypertrophy, in a temperature-dependant manner (Yoshihara et al., 2013), which may hint at possible mechanisms. In humans, heat-induced increases in hypertrophy signalling have been shown when heat is applied to a limb undergoing resistance exercise, compared with the unheated exercising limb (Kakigi et al., 2011). Given that resistance training is an integral component of any sprint or power athlete's training regime to enhance maximal strength, rate of force development and power production (Haff & Nimphius, 2012), it would seem that adding heat to resistance training for strength or power athletes may enhance desired neuromuscular training adaptions.

2.7 Conclusion

The effects of heat stress and HA has been extensively researched in untrained and recreationally-trained individuals, while a growing body of work is beginning to amount towards understanding these effects in highly-trained athletes. Heat stress can impair endurance, team sport and sprint performance in trained individuals, while HA appears to enhance performance in hot conditions. The current state of literature emphasises the effects of heat and HA in endurance and team sport events, while less attention has been

given to aspects of neuromuscular function, including sprint ability, power, and strength. A wide range of HA methods have been used with trained athletes and it is evident that careful consideration should be put into designing a HA protocol, including the timing and number of days, as well as the session type, intensity, duration, and frequency. In addition, an understanding of how to best integrate HA into the complex world of an elite athlete's training schedule is currently lacking. Finally, the application of heat or HA as a training tool, for both endurance and sprint and/or power event athletes, shows promise, although more work is needed to improve understanding so as to develop guidelines that appropriately advise practitioners.

CHAPTER THREE: PERIODISING HEAT ACCLIMATION IN ELITE LASER SAILORS PREPARING FOR A WORLD CHAMPIONSHIP EVENT IN HOT CONDITIONS

Chapter Three

3.1 Abstract

Purpose: To examine the retention and re-acclimation responses during a periodised heat acclimation (HA) protocol in elite sailors preparing for the 2013 World Championships in Muscat, Oman (~27-30°C, 40-60% RH). ***Methods:*** Two elite male Laser class sailors completed 5 consecutive days of HA (60 min per day in 35°C, 60% RH). Heat response tests (HRT) were performed on day 1 and 5 of HA, then 1 (decay 1, D1) and 2 (D2) weeks following HA. Participants were then re-acclimated (RA) for 2 days, within the next week, before a final HRT ~72 h post-RA. Rectal temperature, plasma volume, heart rate, sweat rate, as well as thermal discomfort and rating of perceived exertion were measured during each HRT. ***Results:*** Rectal temperature decreased with HA (-0.46 ± 0.05^{o}C), while individual responses following D1, D2 and RA varied. Heart rate (-14 ± 7 bpm), thermal discomfort (-0.6 ± 0.1 AU) and rating of perceived exertion (-1.8 ± 0.6 AU) decreased across HA, and adaptations were retained by D2. Plasma volume steadily increased over the decay period (D2 = +8.0 ± 1.3%) and after RA (+15.5 ± 1.1%) compared with baseline. RA resulted in further thermoregulatory improvements in each athlete, although individual adjustments varied. ***Conclusion:*** Heat strain was reduced in elite Laser sailors following HA and most thermoregulatory adaptations were retained for 2 weeks afterwards. RA may 'top up' adaptations after 2 weeks of HA decay.

3.2 Introduction

Short-term heat acclimation is a commonly used strategy to prepare athletes for competition in hot environments and typically involves 4-7 days of aerobic training in artificially hot conditions (30-40°C) (Garrett et al., 2012). The physiological adaptations seen with heat acclimation (HA) include decreased resting and exercise core temperatures, lowered skin temperature, increased plasma volume and sweat rate and a decreased heart rate for a given workload, all of which attenuate performance impairments that are caused by hot conditions in the unacclimated state (Armstrong & Maresh, 1991). An acute training session in the heat causes considerable physiological strain (Périard et al., 2011) which, accumulated over several days during HA, could misalign with training priorities during an athlete's taper phase leading in to a major competition. While some suggest incorporating HA to a precompetition taper (Chalmers et al., 2014; Périard et al., 2015), the added heat-induced stress may require a reduction in training intensity (Pyne, Mujika, & Reilly, 2009), which could hinder taper objectives where athletes often reduce training volume and increase or maintain training intensity (Mujika & Padilla, 2003). Moreover, training in hot conditions may cause an unplanned rise in internal training load (Crowcroft et al., 2015). Therefore, examining alternate application models of HA periodisation in athletes preparing for competitions in the heat is needed.

The time course of heat acclimation and re-acclimation is not yet clear in trained populations (Chalmers et al., 2014). Studies with untrained participants have shown that re-acclimation following moderate-term HA (10-12 d) may occur after only a short number of heat exposures (1-4 d) (Weller et al., 2007; Wyndham & Jacobs, 1957).

Highly-trained individuals may have even faster rates of HA accrual and slower rates of HA decay (Pandolf et al., 1977). Furthermore, it has been suggested that periodical exposure to heat following HA can allow for the retention of HA over several weeks (Moseley, 1997), however, supporting data in athletes is lacking. The aim of this study was to examine the retention of HA adaptations and re-acclimation responses during a periodised short-term HA protocol in elite sailors preparing for the 2013 Laser World Championships in Muscat, Oman (~27-30°C, 40-60% RH).

3.3 Methods

Participants

Two male elite Laser class sailors, who were members of the New Zealand Sailing Team, volunteered to participate in this descriptive study. The athletes (mean ± SD: age 22.5 ± 0.7 years, height 185.5 ± 3.5 cm, weight 81.4 ± 0.7 kg, maximal oxygen consumption ($\dot{V}O_2$max) 60.4 ± 5.5 ml·kg^{-1}min^{-1} at 420 ± 10 W) provided written informed consent for the study, which was ethically approved through the Auckland University of Technology Research Ethics Committee.

Experimental design

The study involved the observation of responses over a periodised 4-week training mesocycle, which included 3 heavy-load training weeks followed by 1 recovery week (Figure 3.1). Training volume and duration were recorded using an online monitoring system to determine weekly training load via the training stress score (TSS;

Day	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27
Phase	HA							D1							D2							RA					
Sailing																											
Resistance																											
Endurance																											
Heat	HRT	HA	HA	HA	HRT						HRT								HRT			RA	RA			HRT	
TSS (AU)	830 ± 9							857 ± 35							923 ± 21							661 ± 30					
Duration (h)	18.6 ± 1.1							19.7 ± 0.4							21.5 ± 1.1							14.8 ± 1.1					

Figure 3.1 Overview of the periodised heat acclimation (HA) protocol used for 2 elite Laser sailors preparing for the World Championships in Oman. A 4-week mesocycle included 3 heavy-build weeks and 1 recovery week (TSS = training stress score). HA (35°C, 60% RH) occurred during the first week with a heat response test (HRT) on day 1 and 5 of HA. HRTs were repeated during the two following weeks of decay (D1 and D2), and 3 d post re-acclimation (RA).

TrainingPeaks, Boulder, Colorado). HA consisted of 5 consecutive days cycling in hot conditions (35°C, 60% RH) for 45-60 min at a power output to elicit a heart rate response corresponding to 60-70% $\dot{V}O_2$max. To assess the thermoregulatory response to HA, heat response tests (HRT) were performed on day 1 and 5 of HA, as well as 1 (decay 1, D1) and 2 weeks (D2) post HA, and finally 3 d post RA. The study took place during Auckland's winter where the daily ambient temperature was 12-18°C. During the 2-week decay period, all training sessions apart from the weekly HRT were performed indoors (19-20°C) or outdoors in cooler conditions.

Heat response test

All HRTs were conducted in the afternoon at the same time of day as HA sessions (15:00), while resting blood samples were taken at 08:00 prior to any routine morning training sessions. Each HRT consisted of 30-min walking (5.5 km·h^{-1} and 10% incline) in an environmental chamber (35°C, 60% RH; Design Environmental, Wales, UK). Rectal temperature, measured to 0.01°C, was continuously recorded with a data logger (Squirrel SQ2020, Cambridge, UK) connected to a probe (Thermistor 400, Mon-a-Therm, Mansfield MA, USA) and self-inserted ~12 cm past the anal sphincter. Heart rate was recorded every 15 s, while rating of perceived exertion (RPE) and thermal discomfort were recorded at 5-min intervals. Sweat rate was estimated using the change in total body mass (shorts only, body dried; Weightec, Albany, NZ) that occurred during the HRT and was measured to 0.01 kg. Participants then remained in the heat chamber and completed 45 min of submaximal cycling on an ergometer (SRM Indoortrainer, Jülich, Germany), clamped to an RPE of 13, without heart rate or power output feedback. Power output was recorded every 0.5 s, and heart rate as described

above. Power to heart rate ratio was calculated to determine power output changes relative to the cardiovascular response. The combination of the HRT followed by submaximal cycling was considered as a HA exposure on days 1 and 5.

Blood sample

On each testing day, athletes were instructed to consume 500 ml of water before arriving for their morning blood sample, at which point they sat for 20 min to allow plasma stability. Resting blood samples were collected via venepuncture in K2EDTA vacutettes (Greiner Bio-One, Mon-a-Therm Kremsmunster, Austria). The samples were immediately refrigerated and analysed in triplicate within 1 h for red blood cell count, haematocrit and haemoglobin concentration (AcT 5diff, Beckman Coulter, Miami FL, USA). Estimated change in plasma volume (PV) with HA was calculated using the Dill and Costill equations (Dill & Costill, 1974).

Statistical analysis

All physiological and perceptual data collected during the 30 min HRTs and individual mean power output during 45 min of cycling post HRT are presented as the means ± SDs for each athlete (A and B). The average weekly training duration, for both athletes combined, is presented as mean ± SD (n= 2). The differences in power output, physiological and subjective measurements were analysed using a magnitude-based inference approach (Hopkins, Marshall, Batterham, & Hanin, 2009). This method is used to indicate the possible benefit or harm of each trial. The smallest worthwhile change (SWC) in rectal temperature and plasma volume changes were based off the typical error of published reliability data (Garrett et al., 2009), while the SWC for heart

rate, RPE and cycling power output were calculated from multiple baseline measurements used to determine the coefficient of variation within an athlete. Changes in sweat rate were determined using a SWC score (6.1%) based on results from a similar HA protocol conducted at our laboratory (J Casadio, 2013; unpublished data). Thereafter, chances of change scores within an individual due to HA, decay and RA were determined using published spreadsheets (xPrecisionSubject.xls) found at sportsci.org (Hopkins, 2004). Thresholds for quantitative chances to assess whether a change was *clinically increased*, *clinically decreased* or *clinically trivial* were described as follows: <1%, *almost certainly not*; 1-5%, *very unlikely*; 5-25%, *unlikely*; 25-75%, *possible*; 75-95%, *likely*; 95-99%, *very likely*; >99% *almost certainly* (Hopkins et al., 2009). When an effect was > 5% for both benefit and harm, the true value of the difference was described as unclear. Pearson's product moment correlation analysis was used to assess the association between PV change and submaximal power output. The magnitude of the correlation (r) between these measures was defined as: <0.1, *trivial*; 0.1–0.3, *small*; >0.3–0.5, *moderate*; >0.5–0.7, *large*; >0.7–0.9, *very large*; and >0.9–1.0, *almost perfect* (Hopkins et al., 2009).

3.4 Results

Initial HA adaptations were similar in both athletes (Figure 3.2), with reductions in mean HRT rectal temperature (Athlete A = 71% *possible*; Athlete B = 83% *likely* decrease), heart rate (Athlete A = 94% *likely;* Athlete B = *74% possible* decrease), and RPE (Athlete A = 100% *almost certain;* Athlete B = 94% *likely* decrease) compared

with baseline. Physiological and perceptual HA adaptations were retained at D1 (*trivial* change, D1 vs. HA), and D2 (*trivial* change, D2 vs. HA), with the exception of mean
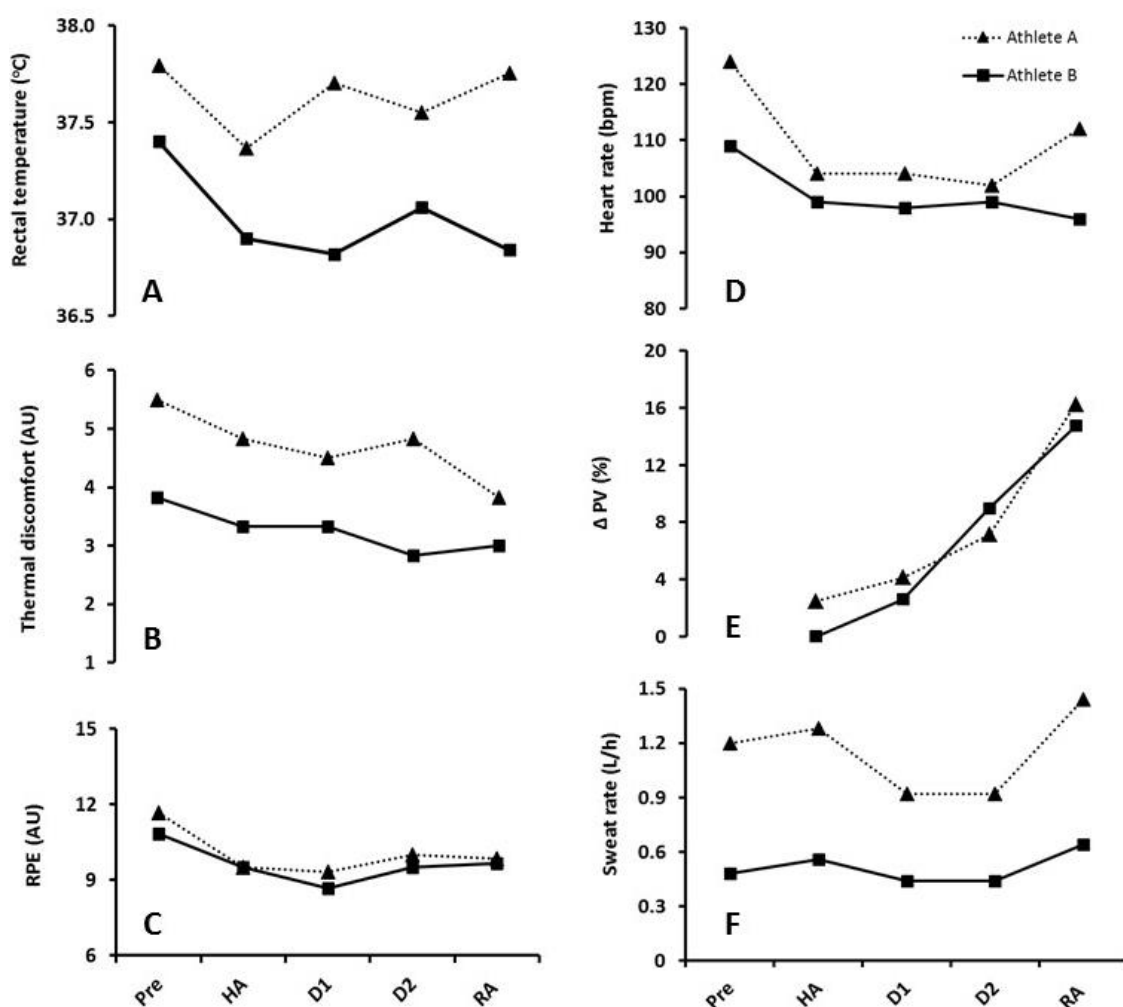
Figure 3.2 Mean physiological and psychophysical changes (rectal temperature, A; thermal discomfort, B; rating of perceived exertion (RPE), C; heart rate, D; plasma volume change (PV), E; and sweat rate, F) during a heat response test in hot conditions (35oC, 60% RH) from before (pre), to after heat acclimation (HA), and after 1 and 2 weeks decay (D1, D2) and 2 d re-acclimation (RA) in two elite Laser sailors (Athlete A & Athlete B).

rectal temperature in Athlete A (55% *possible* increase, D1 vs. HA; 66% *possible* increase, RA vs. HA). Compared with baseline values, PV increased substantially at each time point (99% *very likely* to 100% *almost certain*) following HA in Athlete B, and following D1 in Athlete A. Average power output during submaximal exercise increased meaningfully above baseline following RA (*very likely* increase Athlete A = 97%; Athlete B = 98%; Table 3.1). Mean heart rate during submaximal cycling decreased in both athletes (*likely* to *almost certain*), following HA, decay and RA compared with baseline values. Compared with baseline values, Athlete A showed *trivial* changes with HA and RA, and a 69% *possible* decrease in sweat rate during the two week decay period (D1 and D2 = 0.28 L·h^{-1}) (Figure 3.2). Following RA, Athlete A showed a 95% *very likely* increase (0.52 L·h^{-1}) in sweat rate compared with D2. Sweat rate changes for Athlete B were 94% *very likely* to 100% *almost certainly trivial* at all-time points compared with pre HA values.

Table 3.1 Individual mean power output, heart rate (HR) and power to HR ratio (Power: HR) during 45 min cycling at a clamped RPE of 13 in hot conditions (35ºC, 60% RH) in elite male Laser sailors (n = 2) before (Pre), after heat acclimation (HA), after 1 (D1) and 2 (D2) weeks decay and after re-acclimation (RA).

	Power (W)		HR (bpm)		Power : HR ($W \cdot b^{-1}$)	
	Athlete A	Athlete B	Athlete A	Athlete B	A	B
Pre	211 ± 17	216 ± 27	156 ± 14	143 ± 10	1.37 ± 0.21	1.52 ± 0.24
HA	206 ± 8	210 ± 9	135 ± 6****	133 ± 8**	1.53 ± 0.08	1.59 ± 0.14
D1	188 ± 30	215 ± 17	142 ± 4***	135 ± 5**	1.33 ± 0.23	1.59 ± 0.16
D2	207 ± 11	210 ± 11	136 ± 11****	134 ± 9**	1.54 ± 0.12	1.58 ± 0.16
RA	230 ± 20***	237 ± 4***	138 ± 6****	135 ± 4**	1.68 ± 0.16	1.86 ± 0.16

Pre vs. following HRT time points: ****almost certain* increase (power output) or decrease (HR), ****very likely* increase or decrease, ***likely* increase or decrease.

3.5 Discussion

The key observations made from this descriptive study were that following HA, most thermoregulatory adaptation markers appeared retained following 2 weeks of training in thermoneutral conditions within the athlete's normal training environment, and that only 2 days of RA returned several of these markers to near the level attained after 5 days of HA. Although there were individual variances during decay and RA phases, results provide practitioners with a useful model to consider to enable both HA and essential training elements within an athlete's training programme.

HA induced the expected thermoregulatory and cardiovascular adaptations, including reduced rectal temperature (Athlete A = -0.42^{o}C; Athlete B = -0.50^{o}C), heart rate (Athlete A = -20 bpm; Athlete B = -10 bpm), RPE (Athlete A = -2.2 AU; Athlete B = -1.3 AU) and thermal discomfort (Athlete A = -0.67 AU; Athlete B = -0.50 AU) (Figure 3.2). These results compare with Garrett et al. (2012) who used a similar 5 d HA protocol in a highly-trained population (Rowers; $\dot{V}O_2$max = 65 ± 3 ml·kg^{-1}·min^{-1} at 400 W) and showed reductions in heart rate (-14 bpm), core temperature (-0.30^{o}C), and 2000-m rowing ergometer time (-1.5%) in the heat. In the current study, with the exception of rectal temperature, most HA adaptations (heart rate, RPE, thermal discomfort) were retained in both athletes at 1 and 2 weeks following HA. A lower rectal temperature was retained over 2 weeks in Athlete B, but not Athlete A, highlighting the individual nature of HA responses (Figure 2A)). In both athletes, sweat rate adjustments were shown to be *trivial*, which is in line with other short-term HA studies (Brade et al., 2013; Chen et al., 2013; Cotter, Patterson, & Taylor, 1997;

Petersen et al., 2010), and serves as a reminder to practitioners that sudomotor adaptations require moderate-term HA (8-14 d) (Armstrong & Maresh, 1991).

The retention of most HA adaptations shown in the current study are similar to other studies showing that many thermoregulatory adaptations can be retained for up to 2-3 weeks in active to highly-trained men (Pandolf et al., 1977; Weller et al., 2007). Our findings differ, however, from those of Garrett et al. (2009), who showed decayed adaptations at 2 weeks post-HA in moderately-trained men (Garrett et al., 2009). Two consecutive days of RA showed further thermoregulatory improvements with additional reductions in thermal discomfort, as well as rectal temperature and heart rate (Athlete B only). Plasma volume continued to increase with RA in both athletes, while sweat rate was enhanced in Athlete A. The ability to re-adapt or make further gains with only 2 d has been shown previously (Pandolf et al., 1977; Weller et al., 2007), but never in highly-trained athletes and supports the concept that heat acclimated individuals may acquire thermal 'memory' as evident through rapid RA (Horowitz, 2001).

This study also showed comparable rises in both PV and submaximal power output in the heat over the 4 week training block. While a substantial increase in PV was not shown immediately following HA (Athlete A = +2.5%; Athlete B = 0.0%), meaningful PV expansion was shown as training volume continually increased at D2 (Athlete A = +7.1%; Athlete B = +9.0%; 100% *almost certain* increase) and following RA (Athlete A = +16.2%; Athlete B = +14.7%; 100% *almost certain* increase), and may be attributed to a sustained high-volume training load (TSS = 860-920 AU) during the HA decay period. Power output during submaximal cycling (~ aerobic threshold) was similar (within 5-12 W) following HA and during the decay period, but increased in

both athletes (+20 W; *very likely* increase) following RA during the reduced volume week prior to departure (Table 3.1). While the relationship between PV expansion and aerobic performance in the literature is unclear (Corbett et al., 2014), our findings showed a *very large* (r = 0.70) relationship between PV change and submaximal power output. While supercompensation following heavy-build weeks and a subsequent download week cannot be ignored (Fry, Morton, & Keast, 1992), this increase was larger than what is normally seen in these athletes during a focused sailing block, where aerobic gains are not expected, thus highlighting the potential ergogenic 'boost' that frequent HA sessions could produce within an athlete's season (Chalmers et al., 2014).

Practical applications

This descriptive study demonstrates that HA can be scheduled up to 3 weeks before departing for competition in a hot environment, when accompanied with 'top up' RA sessions. Adding environmental stress to training is an additive physiological stressor, and could cause an increase in internal training load, potentially leading to overreaching (Crowcroft et al., 2015; Taylor & Cotter, 2006). The approach shown in the present study could allow athletes and coaches to periodise the timing of HA so as to reduce interference with more critical training goals. Specifically, we suggest that implementing HA 2-3 weeks prior to competing in a hot environment, along with 2 additional RA sessions, may preserve precompetition taper quality while restoring HA adaptations, thus serving as an alternative and possibly more appropriate HA strategy for elite athletes. Finally, while adaptations followed a similar time course between the two athletes in this study, individual variances during the decay and RA period were noted, highlighting the importance of considering individual athlete responses.

3.6 Conclusion

Short-term HA decreased heat strain in two elite Laser sailors through decreased rectal temperature, heart rate, RPE and thermal discomfort. Most thermoregulatory adaptations were retained over the 2 subsequent weeks, without HA, except for rectal temperature in one athlete. Additional RA sessions provided further thermoregulatory enhancement with varied responses between the two athletes. The results of this study suggest that RA may be used to 'top up' heat tolerance adaptations after 2 weeks of decay from HA. Further investigation involving greater subject samples is needed to confirm these observations.

CHAPTER FOUR: HEAT TRAINING IMPROVES CARDIOVASCULAR RESPONSES BUT IMPAIRS SPRINT PERFORMANCE IN ELITE SPRINT TRACK CYCLISTS

4.1 Abstract

Purpose: To investigate the effects of short-term heat training on sprint performance and cardiovascular function in elite sprint track cyclists. *Methods:* Five elite male sprint cyclists (23.8 ± 2.0 years; peak power output = 22.8 ± 1.3 W·kg^{-1}) completed a 5 d training camp in hot conditions (30-35°C, 40-60% relative humidity (RH)) consisting of sprint-interval training and resistance training. Sprint performance was assessed in temperate conditions (~20°C, 50% RH) on day 1 and 5 of heat training, and 72 h post. Cardiovascular responses were assessed (~20°C, 50% RH) pre and 24 h post heat training and included a submaximal cycling test and resting plasma volume measurement. During the first (heat training day 1 = HT1) and last (HT5) resistance training session thermoregulatory measurements were recorded. *Results:* There was a small *very likely* decrease in sprint peak power output [-4.3% (90% CL -6.1, -2.5)] and a small *possible* decrease in optimal cadence [-2.0 rpm (-5.1, 1.2)] 24 h post heat training. Core temperature during HT5 was lower (-0.29-0.36°C from 10-40 min; small *possible* decrease) during exercise but there was no effect of heat training on skin temperature or sweat rate compared with baseline values. Following heat training, heart rate during submaximal exercise decreased [-10 bpm (-18, -2); large *very likely*] and recovery heart rate increased [11 bpm (9, 14); large *most likely*], compared with baseline values. There was a small *likely* increase in plasma volume [6.3% (2.7-10.0)] immediate post 5 d HT, compared with baseline values. *Conclusion:* Our findings indicate that markers of cardiovascular stability improved with short-term heat training, and that sprint performance was supressed at 24 h but rebounded at 72 h post.

4.2 Introduction

Increased body temperature is associated with enhanced performance acutely during single (Sargeant, 1987) and multiple sprint efforts (Ball et al., 1999; Girard et al., 2013). Acute heat-induced sprint enhancement may be explained by increases in muscle temperature (Ball et al., 1999; Sargeant, 1987) leading to enhanced skeletal muscle contractility (Gray et al., 2006). Increased muscle temperature results in faster muscle fibre nerve conduction velocity (Farina et al., 2005; Gray et al., 2006) as well as augmented adenosine triphosphate (ATP) turnover via increased phosphocreatine (PCr) phosphorylation and glycolytic rates (Gray et al., 2006). Combined, such alterations in muscle contractile and metabolic function enhance both force-velocity and power-velocity relationships, thus explaining acute heat-induced sprint performance improvements (Sargeant, 1987). These combined relationships offer an alluring ergogenic prospect acutely.

The chronic effects of training in the heat on sprint performance is less understood. Traditionally, multi-day heat exposure, or heat acclimation (HA), has been more commonly implemented with endurance and team sport athletes as a means to reduce thermoregulatory strain and attenuate heat-induced performance impairments (Armstrong & Maresh, 1991). More recently, heat exposure been suggested to have ergogenic potential through its enhanced cardiovascular function and reduced physiological strain apparent in temperate conditions (Corbett et al., 2014). Heat acclimation (artificial heat), or heat acclimatisation (outdoors), involves successive training sessions, typically moderate steady-state exercise (30-90 min) in hot conditions (typically 30-40ºC) (Racinais et al., 2015). Short-term HA protocols (4-7 d) are routinely applied with athletes, resulting in reductions in body temperature (skin and

core temperature), heart rate and thermal perception, as well as increases in plasma volume (Chalmers et al., 2014; Garrett, Rehrer, et al., 2011). Together, these physiological adaptations attenuate performance impairments caused by hot conditions in the unacclimated state (Racinais et al., 2012; Sunderland & Nevill, 2005). Although several recent reviews detail the usefulness of HA implementation with endurance athletes and team sport athletes preparing to compete in the heat (Chalmers et al., 2014; Périard et al., 2015; Racinais et al., 2015; Tyler et al., 2016), research examining the effects of sprint-training in hot conditions is lacking. Of the limited work available, three studies have shown that 4-to-5 d of intermittent high-intensity training (30-50 min per session) in hot conditions (~30-35ºC, 30-60% relative humidity (RH)) had no effect on sprint running performance in team sport athletes in hot (Sunderland et al., 2008) or temperate (Petersen et al., 2010; Philp, Buchheit, Kitic, Minson, & Fell, 2016) conditions. Although the heat exposure times were short in both trials, thermoregulatory adaptions occurred, as evidenced through reductions in core temperature (Sunderland et al., 2008), heart rate and thermal discomfort (Petersen et al., 2010; Sunderland et al., 2008). Conversely, 10-d of traditional steady-state exercise (60-min cycling at 50% $\dot{V}O_2$peak) in hot conditions (~33ºC, 52% RH) showed improvements in sprint performance during a 40-min intermittent sprint protocol in moderately-trained males (Castle et al., 2011). Given the small body of research examining the effects of chronic heat exposure on sprint performance, it remains unclear whether the sprint-training stimulus might be augmented through chronic heat exposure (increased muscle temperature and enhanced muscle contractility), thus leading to improved power production in temperate conditions.

The cardiovascular adaptations induced by HA represent another potential benefit for sprint athletes who would be unlikely to perform endurance-type training. Within 3-4 days of initiating heat exposure, cardiovascular stability improves through increases in plasma volume and reductions in heart rate (Périard et al., 2015), and is similar to the response shown following endurance training (Green, Jones, Hughson, Painter, & Farrance, 1987; Green, Jones, & Painter, 1990). Such adaptations have been linked with increases in $\dot{V}O_2$max following HA (Lorenzo et al., 2010), and may lead to improved exercise economy (Sawka, Pandolf, Avellini, & Shapiro, 1983), as well as enhanced recovery following high-intensity and maximal sprint-training (Tomlin & Wenger, 2001). Such adaptations may also lead to faster post-exercise $\dot{V}O_2$ recovery (Hamilton, Nevill, Brooks, & Williams, 1991) and associated increased rates of ATP/PCr resynthesis (Hultman, Bergström, & Anderson, 1967). Combined, these aerobic adaptations may speed the rate of recovery following or between maximal sprint efforts, which may be important for performance. For instance, international-level sprint track cycling involves several events lasting ~10 s – 150 s, including the Individual Sprint, Team Sprint and Keirin (*UCI Cycling Regulations*, 2016). Each event involves maximal efforts requiring high levels of anaerobic contribution (Craig & Norton, 2001; Jeukendrup, Craig, & Hawley, 2000) and are repeated over multiple heats during a single event session (~2-3 h). Hence, enhanced heat-induced cardiovascular function may allow for faster recovery between sprint event heats. Therefore, the aim of this study was to determine the effects of sprint-specific short-term heat training on sprint performance and cardiovascular responses in elite sprint track cyclists in temperate conditions.

4.3 Methods

Participants

Five male, sprint track cyclists (age = 23.8 ± 2.0 years; height = 183.6 ± 4.2 cm; body mass = 90.7 ± 8.8 kg; peak power output = 22.8 ± 1.3 W·kg^{-1}) from the New Zealand National Team participated in this study during a pre-season training camp. Of the 5 cyclists, 3 were World Champions, 1 was an Olympic medallist and the other was a medallist at a World Championships, all of whom were not acclimated to hot environments. Participants provided written informed consent and ethics approval was attained through the Auckland University of Technology Research Ethics Committee.

Experimental design

The design of this study was a pre-post, single group trial. Participants completed a team training camp, in which all training sessions were completed in hot conditions (30-35°C, 40-60% RH) over 5 days. Training consisted of 3 resistance training sessions (Monday, Wednesday, and Friday) in a heated gym (30°C, ~40% RH) while wearing long-sleeved tops and full-length pants. On alternate days, (Tuesday, Thursday), participants performed sprint-interval training sessions in a climate chamber (35°C, 60%; Design Environmental, Wales, UK). Sprint performance was assessed prior to the first heat training (HT) session on day 1, as well as 24 and 72 h following the final HT session. Rest and recovery was encouraged during the 48 h period between post HT inertial sprint tests. Cardiovascular adaptations were assessed before HT and 24 h post HT, and consisted of a submaximal cycling test and resting plasma volume measurement. During the first and last resistance training session (HT1 and HT5), thermoregulatory measurements were assessed, while any changes in peak power were

measured via counter movement jump (CMJ), before and after these resistance training sessions (Figure 4.1).
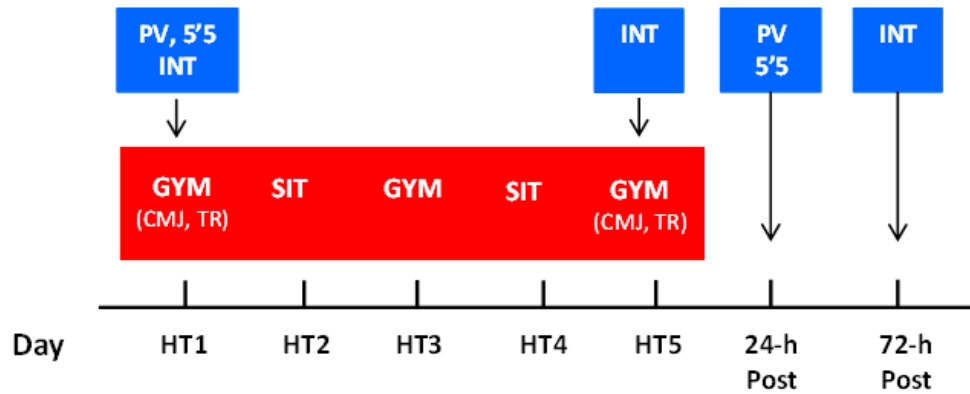
Figure 4.1 Experimental overview. Five days of heat training (HT; red panel) consisting of resistance training (GYM) and sprint-interval training (SIT). The retention of inertial (INT) peak power output, submaximal exercise response (5'5) and plasma volume (PV) were assessed pre and post heat training. CMJ = counter movement jump; TR = thermoregulatory measures.

Performance measurements

Sprint performance was assessed during 2 consecutive inertial sprints, whereby power was measured using the inertial load method using torque readings applied to a fly wheel over a range of pedalling frequencies (Martin, Wagner, & Coyle, 1997). Briefly, cyclists started from rest in a seated position and were instructed to accelerate maximally for 3-4 s with verbal encouragement. Peak power output, optimal cadence (pedalling frequency at peak power production) and time to peak power were recorded for each sprint using a custom-built inertial ergometer (Goldmine, Auckland, New Zealand). For this group of athletes, the inertial sprint was performed every 2 weeks as part of their routine monitoring and results were used as an indication of expected

performance outcomes. To assess the acute effect of resistance training in the heat on power production, CMJs were performed before and after select resistance training sessions, in which 3 separate jumps were performed on a force plate (Innovation Design, Auckland, New Zealand; sampling frequency 500 Hz), separated by ~ 30 s. Cyclists were instructed to place their hands on their hips, squat down so knees were bent to 90°, then immediately jump vertically as high as possible, landing back on the force plate with both feet at the same time. The highest peak power of the 3 trials was recorded (Force Board Software, Auckland, New Zealand).

Physiological measurements

Blood samples were collected via venepuncture in K2EDTA vacuettes (Greiner Bio-One, Kremsmunster, Austria). For the resting blood samples, the cyclists were instructed to consume 500 ml of water before arrival at the laboratory (08:00), at which point they sat for 20 min to allow plasma stability. The samples were immediately refrigerated and analysed in triplicate within 1 h for red blood cell count, haematocrit (Hct) and haemoglobin (Hb) concentration (AcT 5diff, Beckman Coulter, Miami FL, USA). Estimated change in plasma volume with heat training was calculated using the Dill and Costill equations (Dill & Costill, 1974).

A 5'5 test was performed in temperate conditions (20°C, 50% RH) to assess submaximal exercise heart rate (HR_{ex}) and heart rate recovery (HRR) (Buchheit, Al Haddad, Laursen, & Ahmaidi, 2009). Briefly, cyclists cycled for 5-min at a predetermined power output aimed to elicit an exercise intensity slightly below anaerobic threshold. At 5-min, HR_{ex} was recorded (RS800cxr, Polar Electro, Kempele, Finland) and athletes sat passively in a chair for a further 5-min, at which point HRR was recorded.

Thermoregulatory measurements to assess adjustments during heated resistance training included core temperature, skin temperature, and sweat rate. Core temperature was recorded at 5-min intervals with a data logger (CorTemp, HQInc, Palmetto, Florida), which read temperature from an ingested telemetric capsule (CorTemp Sensor, HQInc, Palmetto, Florida), swallowed by the subjects 6 h before testing. The capsules were factory calibration with assured accuracy to $\pm\ 0.1$°C. Skin temperature was measured at 10-s intervals using wireless telemetric sensors (Maxim DS1921G, San Jose, California) affixed to the chest and front mid-thigh on the right side of the body. Sweat rate was estimated using the change in total body mass measured to 0.01 kg (undergarments only, towel dried; Weightec, Albany, NZ) that occurred during the resistance training session.

Heat training

All resistance training sessions occurred at 14:00 and began with a 15-min warm up (skipping, mobility exercises and dynamic stretches). During HT1 and HT5, cyclists performed one strength (back squat) and four power (cleans, box jumps, speed pull ups and narrow power press-ups; all body weight) exercises. For strength exercises, the work set intensity was ~ 85% 1-repetition maximum, and 3-4 work sets of 3-5 reps were completed, with ~ 3-min rest between sets. The total work load during HT1 and HT5 was held constant. During HT3 the exercises differed, however, the training focus (strength and power), as well as the reps, sets and intensity were similar. Sprint-interval training during HT2 and HT4 sessions, were performed on each athlete's personal road bicycle fixed to a stationary trainer (LeMond, Minneapolis, Minnesota) and consisted of

routine training elements involving repeated maximal efforts (1-2 sets, 2-5 reps, 20-30-s). All cycling HT sessions were ~ 60 min in duration.

Statistical analysis

All data are reported as means ± standard deviations (SD), and mean (90% confidence limits (CL)) as appropriate. The differences in measurements were analysed using a magnitude-based inference approach (Hopkins et al., 2009). This method is used to indicate the magnitude and probability of substantial increases or decreases of each variable in a given condition. For sweat rate, plasma volume and heart rate indices, the magnitude of the changes between trials were expressed as standardised differences (Cohen effect sizes, ES). The criteria used for interpreting the magnitude of the ES for these three variables were: < 0.2, trivial; > 0.2, small; > 0.6, moderate; > 1.2, large; > 2.0, very large; and > 4.0, extremely large (Hopkins et al., 2009). The smallest worthwhile change (SWC) was calculated based on previous testing data from 13 occasions over a 5 month period and was expressed as the coefficient of variation (CV) (Hopkins, 2000) for inertial peak power output (2.2%) and optimal cadence (1.5%), and CMJ peak power (3.5%). Magnitude thresholds were used to determine the SWC for core temperature, in which the possible range of change was transformed into a full scale of deflection (FSD) (Hopkins, 2010). In brief, the range was made from 0-100% and magnitude thresholds were defined as 10%, 30%, 50%, 70% and 90% for small, moderate, large, very large and extremely large changes. Effect sizes, with uncertainty of the estimates shown as 90% CL, were determined using published spreadsheets found at sportsci.org (Hopkins et al., 2009). Quantitative chances of measurements affecting performance were assessed qualitatively as follows: <1%, *most unlikely* or

almost certainly not; 1-5%, *very unlikely*; 5-25%, *unlikely*; 25-75%, *possible*; 75-95%,

likely; 95-99%, *very likely*; >99% *most likely* or *almost certainly* (Hopkins et al., 2009).

When an effect was > 5% for both benefit and harm, the true value of the difference was

described as unclear.

4.4 Results

Performance outcomes

Compared with mean normal values, there was a small *very likely* decrease [-

4.3% (-6.1, -2.5)] in inertial sprint peak power output immediately 24 h post heat

training and a *possibly* trivial change [0.2% (-3.4, 3.9)] 72 h post (Figure 4.2). There

was a small *possible* decrease in optimal cadence [-2.0 (-5.1, 1.2)] 24 h post HT, while

the effects of 72 h post were trivial [-0.8, (-3.5, 2.0)]. For mean CMJ peak power there

was a small *likely* increase [4.5% (-1.2, 10.5)] post HT1 compared with pre HT1 values,

while the change following HT5 was unclear [-0.2% (-9, 9.5)] (Figure 4.3).

Thermoregulatory responses

Core temperature was similar at rest during the first [HT1 = 37.57 ± 0.25°C] and

final [HT5 = 37.52 ± 0.23°C] heat training trials. Exercising core temperature was

possibly lower during the middle portion of the resistance training session during HT5

compared with HT1 (Figure 4.6). The change in skin temperature in HT5 compared

with HT1 was *unclear* [0.05°C (-0. 26, 0.37)], as was sweat rate [0.11 L·h^{-1} (-0.25,

0.47); ES = 0.31].

Cardiovascular responses

Following 5 d HT, there was a large *very likely* decrease in HR_{ex} [-10 bpm (-18, -2); ES = -1.61] and large *most likely* improvement in HRR [11 bpm (9, 14); ES = 1.26] compared with baseline values (Figure 4.4). Plasma volume increased [6.3% (2.7, 10.0); small *likely*] following 5 d of heat training, compared with baseline values.
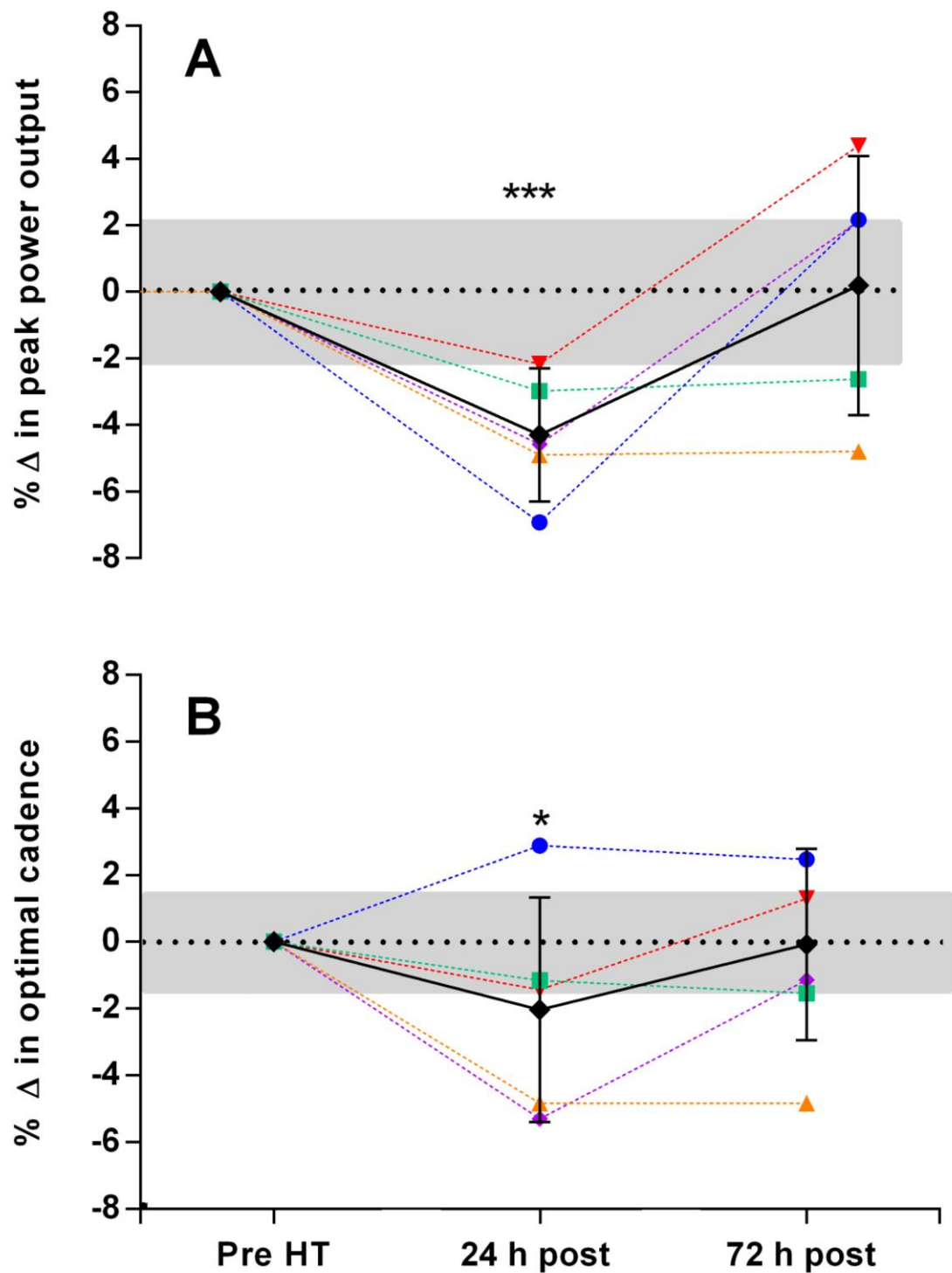
Figure 4.2 Change (%) in A) inertial peak power output and B) optimal cadence from baseline values (pre heat training = pre HT). The solid black line represents mean ± SD. The shaded area represents the smallest worthwhile change (CV%) in peak power (2.2%) and cadence (1.5%). The dashed coloured lines represent individual results. Time point vs. Pre HT: *possible decrease, ***very likely decrease.
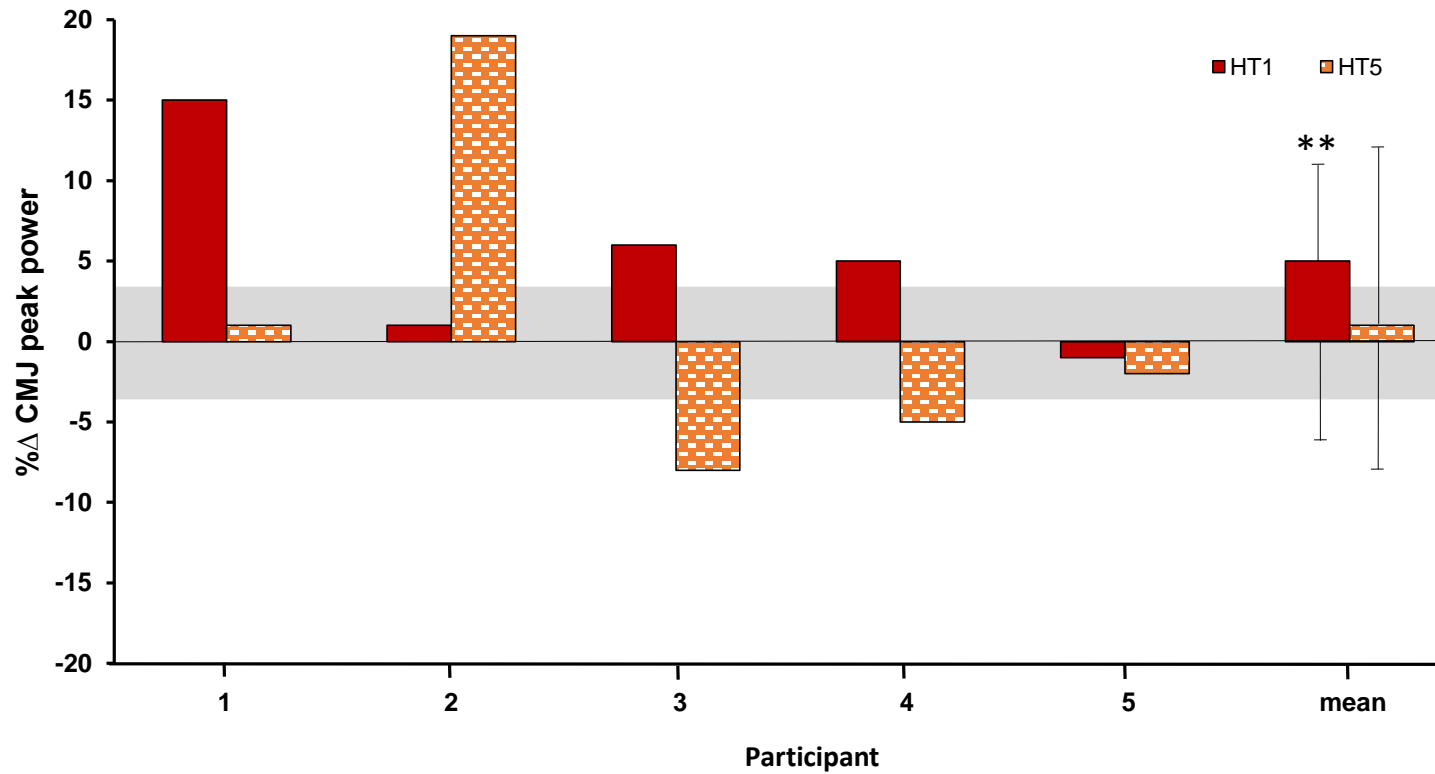
Figure 4.3 Individual and mean ± SD percent changes (end vs. start of the heat training session (HT)) in counter movement jump (CMJ) peak power during sessions one (HT1) and five (HT5) in hot conditions (30ºC, 40% RH). The shaded area represents smallest worthwhile change (3.5%). End vs. Start of heat training session: ***likely* increase.
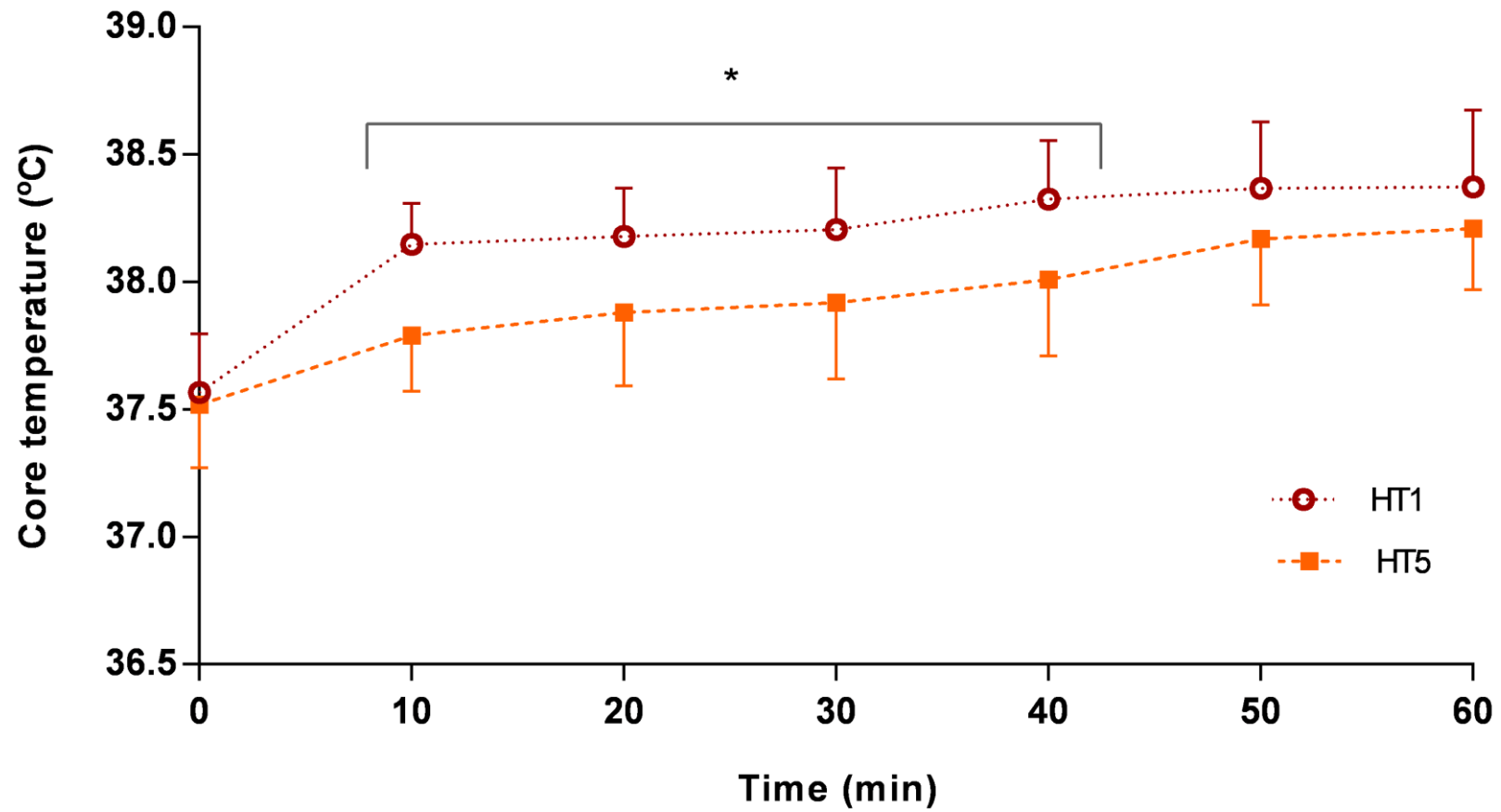
Figure 4.4 Mean core temperature (ºC) during 60-min of resistance training in hot conditions (30ºC, 40% RH) on the first (HT1) and final (HT5) day of a heat training camp with elite male sprint track cyclists (n = 5)). HT5 vs. HT1: *possible* decrease.
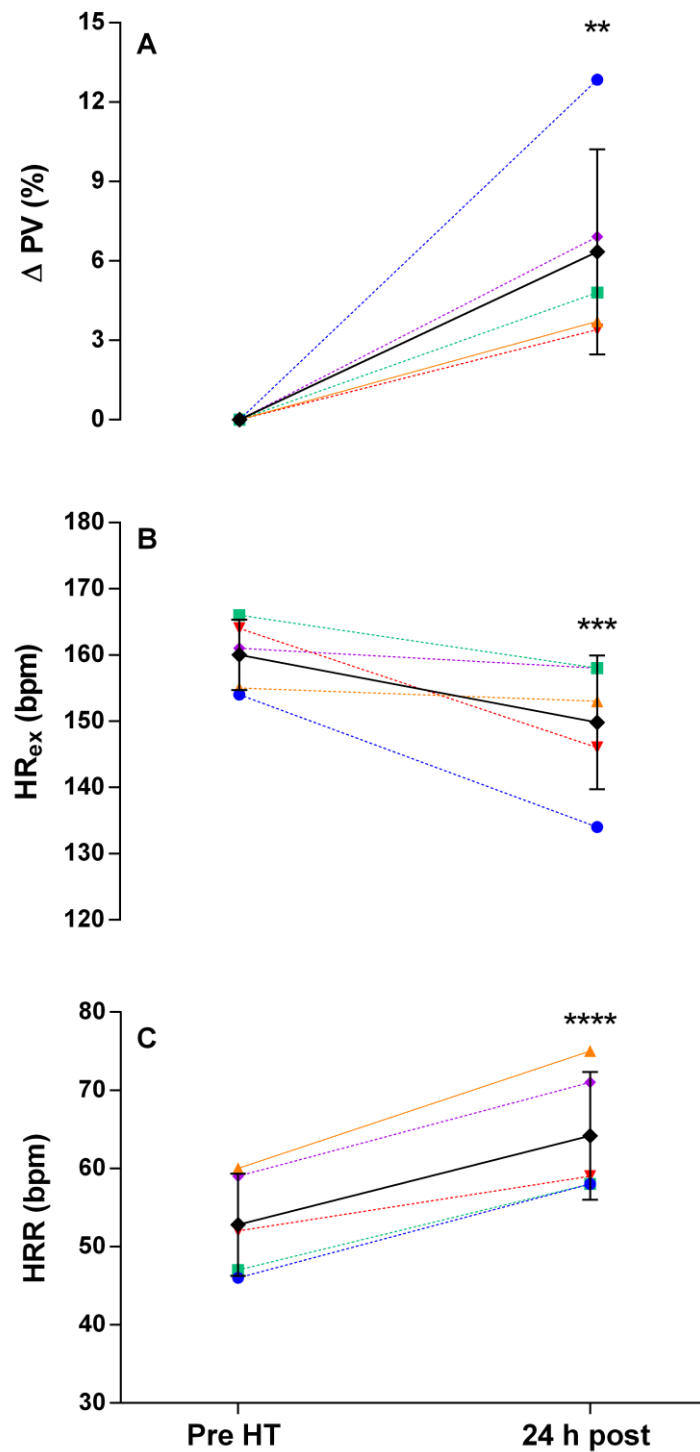
Figure 4.5 Individual and mean ± SD values for A) heart rate during submaximal exercise (HR$_{ex}$), B) heart rate recovery (HRR), C) percent change in plasma volume (PV) pre and 24 h post heat training (30-35°C, 40-60% RH). 24 h post vs. Pre HT: *******likely* change (increase or decrease), ****very likely* change, *****most likely* change.

4.5 Discussion

To our knowledge, this study is the first to report the acute and cumulative effects of short-term heat training on sprint performance, thermoregulation and cardiovascular responses in elite sprint track cyclists. The main finding was that sprint performance, as measured through peak power output and optimal cadence, showed a short-term impairment following 5 d of heat training, which was restored at 72 h post. In addition, sprint specific heat training induced signs of HA through reduced exercising core temperature as well as enhanced cardiovascular function. Although this study was limited by the lack of a control group and its small sample size, these findings are meaningful given they represent the entire population size of the elite high performance male sprint track cyclists in the country.

Performance outcomes

In the present study, short-term heat training showed an initial drop in sprint performance during an inertial sprint test in temperate conditions, which was later restored with 2 d of recovery. The sprint test was performed in temperate conditions to examine the effects of chronic heat exposure (5 d) on sprint performance rather than the acute effects of heat, which have previously been shown to enhance peak power during maximal sprint efforts (Ball et al., 1999; Sargeant, 1987). Research by Castle et al. (2011) indicated that chronic heat training, or HA, might enhance sprint performance, although the performance test was conducted in hot conditions where HA adaptations would have likely aided the improved power output shown (Castle et al., 2011). It may be that the ergogenic effects of heat for sprinting rely on acute increases in muscle temperature and associated muscle contractility enhancement (Gray et al., 2006), rather

than inducing short-term adaptations. Previous work has shown that chronic heat exposure has direct effects on skeletal muscle adaptation and performance (Goto et al., 2011; Goto et al., 2007), although, such studies involved elevating muscle temperature (38.5-39.0°C) over a substantial period (40-h over a 10 week period) (Goto et al., 2007). In comparison, skin temperature in the present study was elevated to ~34-35°C towards the end of each 60-min heat training session and was therefore unlikely to have caused the muscle temperature increases shown in previous heat-induced skeletal muscle adaptation studies (Goto et al., 2011; Goto et al., 2007).

Overall, the training volume during the heat training camp was low for these athletes compared with normal fluctuations in their training volume. Therefore, the reduction in inertial sprint peak power and optimal cadence was unexpected (Figure 4.2). Reductions in inertial sprint performance compared with normal variations have been shown to reflect fatigue (MacIntosh, Svedahl, & Kim, 2004). In addition, CMJ performance before and after HT1 and HT5 resistance training sessions, indicates that neuromuscular fatigue may have accumulated from consecutive days training in the heat in some of the cyclists. Following HT1, CMJ peak power was higher than before the heated resistance training session (Figure 4.3), and is supported by Hedley et al. (2002) who showed a 3.1% improvement in vertical jump height following 30 min of passive heating in a sauna (Hedley et al., 2002). Conversely, CMJ peak power decreased in 3 of the cyclists following HT5 on the final day of heat training, and may suggest a degree of heat-induced neuromuscular fatigue. Heat-induced neuromuscular fatigue has been shown both acutely through reduced repeated jump performance (Mohr & Krustrup, 2013) and chronically during sustained maximal voluntary contractions following heat acclimatisation (Brazaitis & Skurvydas, 2010). Although subjective fatigue was not

recorded during this study, the cyclists reported high levels of fatigue given that the training volume during the heat training camp was low compared with typical routine training, where 2 training sessions a day would be more representative of their typical training load. Therefore, if heat acutely enhances muscle contractility during training that demands high levels of neuromuscular function (maximal sprinting, strength and power exercises), it could be speculated that this higher level stimulus may in fact cause greater central nervous system fatigue in the short-term. Interestingly, 3 of 5 cyclists showed a rebound effect for both inertial peak power output and optimal cadence (Figure 4.2), thus showing that the effects of heat-induced fatigue on neuromuscular function may have varied time courses of recovery for different individuals. More work is required to understand the chronic effects of heat on sprint performance.

Thermoregulatory response

Sprint-specific heat training over 5 d caused a decrease in core temperature during resistance training exercise in hot conditions, with no effects on skin temperature or sweat rate. To our knowledge, no other studies have examined the thermoregulatory response during a strength- and power-based resistance training session, let alone the effects of consecutive heat training sessions. While resting core temperature was unaffected by sprint-specific heat training, exercising core temperature was reduced during 10-40 min of the final heat training session (Figure 4.4), which is supported by others who have shown that 4 d of high-intensity intermittent running in the heat elicited reductions in exercising but not resting core temperature (Sunderland et al., 2008). Conversely, Peterson et al. (2010) showed no observable effect of 4 d high-intensity interval training (30-45 min per session) in hot conditions on core temperature.

A recent meta-analysis has shown that short-term HA may not be sufficient for lowering core temperature compared with moderate and long-term protocols (Tyler et al., 2016). However, the aim of the current study was to elicit short-term HA PV expansion (Périard et al., 2015), as well as enhance the training stimulus for sprinting and resistance training sessions in the heat. The mean values for core temperature during the first heat session ranged from between ~38.0-38.4°C, while the final session reached only ~38.0°C after 40 min into the 60-min session. The heat stress during these resistance training sessions would therefore not be considered high. To see greater thermoregulatory adaptations, it is thought that time ($\geq$ 60 min) at an elevated core temperature (~38.5°C) is a key factor (Houmard et al., 1990). The resistance training session examined in the present study may not have increased core temperature substantially due to the low work volume (3-5 reps) and long-duration rest period (2-3 min between sets) characteristics of strength- and power-based exercise protocols (Stone, O'Bryant, Garhammer, McMillan, & Rozenek, 1982). Anecdotally, the cyclists perceived that heat training enhanced the quality of the sprint- and resistance training sessions. One cyclist inadvertently performed a personal best for one of the resistance training exercises, and was surprised by how easily he could lift loads that may otherwise seem heavier in the normal temperate gym conditions.

Cardiovascular response

Sprint and resistance training in the heat elicited cardiovascular adaptations through plasma volume expansion and improved heart rate indices during submaximal exercise in temperate conditions. In trained populations, exercise in the heat over 4-7 d has been shown to increase plasma volume (~4-7%) by performing steady-state endurance-

training (~4%) (Garrett et al., 2009; Garrett et al., 2014) and team sports-specific training in the heat (~4-7%) (Buchheit et al., 2011; Racinais et al., 2012), as well as with post-exercise sauna bathing (~18%) (Stanley, Halliday, D'Auria, Buchheit, & Leicht, 2015). Our study is the first to show similar increases in plasma volume (6.3%) with low-volume sprint and resistance training in the heat. Plasma volume expansion is associated with improvements in HR_{ex} (Convertino, 1991) and HRR (Buchheit, Laursen, Al Haddad, & Ahmaidi, 2009). In the present study, HR_{ex} during submaximal cycling and HRR improved by -6.3% and +21.5% respectively. These changes are similar to those found following 9 weeks of repeated sprint and high-intensity intermittent training (Buchheit et al., 2008), and a 7 d football camp in the hot conditions (Buchheit et al., 2011). Conversely, 10 d of post-exercise sauna bathing in well-trained endurance cyclists showed impairments in both HR_{ex} (+5.5%) and HRR (-15.6%), which might have been a result of a self-regulated reduction in training volume shown during the sauna intervention (Stanley et al., 2015). Our study involved little-to-no endurance-type exercise performed during each training session, so that cardiovascular adaptations were likely heat-, rather than endurance-training induced, which is comparable with results from others using passive heating techniques (Scoon et al., 2007; Stanley et al., 2015; Zurawlew, Walsh, Fortes, & Potter, 2015). While improved aerobic fitness is associated with faster recovery rates following maximal sprint bouts (Tomlin & Wenger, 2001), this was not directly measured in the present study and therefore we can only speculate that heat-induced cardiovascular adaptations may be beneficial for recovery in sprint track cyclists.

Perspectives

The future application of heat training camps for sprint athletes requires further investigation. Although our findings showed beneficial cardiovascular adaptations, thus implying improved fitness, sprint performance was temporarily impaired. Consecutive days of sprint-specific heat training may have caused neuromuscular fatigue in this elite athlete population given that the training stimulus during sessions may have been elevated by improved muscle contractility, as well as the added physiological heat strain. Given that heat training elicited an increase in perceived training quality, it may be that intermittent days of heat training or targeting specific training sessions may enhance the training stimulus without the accumulation of heat-induced fatigue over several days. Although it is interesting that HR_{ex} and HRR following submaximal exercise in temperate conditions were improved, the translation to sprint track cycling performance needs to be better understood. First, does this gain in cardiovascular stability elicit improved sprint performance for longer duration efforts involving greater aerobic contribution (3rd cyclist of the Team Sprint, Keirin and Kilometre Time Trial)? Second, does this improved submaximal fitness translate to enhanced recovery between heats in competition? Although the results herein are interesting, extensive work is needed before heat training as an ergogenic strategy should be used by sprint athletes.

4.6 Conclusion

Short-term heat training over 5 days decreased sprint performance at 24 h post as measured through peak power output and optimal cadence in elite male sprint track cyclists. Sprint performance was restored at 72 h post. In addition, sprint-specific heat

training induced signs of HA through reduced exercising core temperature, as well as enhanced cardiovascular function. While short-term heat training does not appear to improve single sprint performance, future research should explore the ergogenic potential of heat training in sprint athletes.

CHAPTER FIVE: COMBINING SPRINT AND RESISTANCE TRAINING IN THE HEAT: EFFECT ON SPRINT PERFORMANCE IN ELITE BMX CYCLISTS

5.1 Abstract

Purpose: To investigate the effects of acute heat and sport-specific short-term heat acclimation on repeated sprint performance in elite bicycle motocross (BMX) cyclists. *Methods:* Seven elite BMX cyclists (4 males: mean ± SD; 25 ± 3 yr; relative peak power output; PPO = 23.9 ± 0.4 W·kg^{-1}; 3 females = 20 ± 4 yr; relative PPO = 17.3 ± 1.5 W·kg^{-1}) completed 5 d of BMX specific HA (60 min per day, 35°C, 50% RH). Effects of heat and HA were assessed using a heat response test (HRT) and repeated sprint test (3 sets of 3 x 10 s on/off sprints; 10 min recovery between sets). An initial assessment day was completed in temperate conditions (CON = 20°C, 50% RH), with subsequent test days completed in hot conditions (HOT = 35°C, 50% RH), before and after HA (Post HA). *Results:* Mean PPO was enhanced in HOT (small *likely* increase = +8.1 ± 9.2%), despite evidence for higher thermal strain (*very likely* increases in skin temperature (+3.3 ± 1.0°C), heart rate (+17 ± 4 bpm), *likely* increases in sweat rate (+0.3 ± 0.2 L·h^{-1}), *possible* increases in thermal discomfort (+1.2 ± 1.1 AU) and sensation (+0.6 ± 1.1 AU). After 5 d HA, a further improvement in PPO occurred (small *likely* increase, +6.4 ± 6.8%) and thermal discomfort had decreased (-1.4 ± 2.2 AU). No beneficial changes in core (-0.1 ± 0.1°C), skin temperature (-0.4 ± 1.1°C), heart rate (-3 ± 13 bpm) or sweat rate (+0.2 ± 0.5 L·h^{-1}) were observed during exercise. *Conclusion:* Our findings suggest that acute heat stress increases thermal strain but does not impair repeated sprint performance in elite BMX cyclists. However, BMX-specific HA may improve a cyclist's thermal perception and increase sweat rate during training and competing in hot environments.

5.2 Introduction

Successful bicycle motocross (BMX) performance requires an initial 'all out' sprint down a steep start ramp (15-20% incline), accompanied by skills and tactics that require the rider to manoeuvre against competitors over a technical track (~300-400 m) in the fastest time possible (Debraux & Bertucci, 2011a; Louis et al., 2012). Despite its short duration (~40 s), elite-level competition requires a high level of energy provision from both aerobic and anaerobic systems (Louis et al., 2012). In international competition, BMX cyclists can race up to 6 times per day, with as little as 20-30 min separating races.

Several pinnacle BMX events are held in locations associated with typically hot ambient temperatures, including the quadrennial Olympic Summer Games. High-speed BMX racing is also dangerous, and thus requires protective clothing and equipment not conducive to facilitating heat removal from athletes. Such sport attire can increase physiological strain, reduces evaporative cooling and increases the rate of rise in core temperature during exercise in hot conditions (Armstrong et al., 2010). While heat acclimation (HA) is a commonly used strategy to improve endurance performance in hot environments, it is unknown whether a HA strategy would be of benefit to performance for BMX athletes competing in hot conditions.

Improved sprint performance (increased mean and peak power output) has been shown in athletes acutely exposed to heat during single (Sargeant, 1987) and repeated sprints (Ball et al., 1999; Girard et al., 2013). This enhanced performance effect has been attributed to increases in muscle temperature that elicits higher peak power output and pedal rate, thus enhancing all aspects of the power-velocity curve (Sargeant, 1987). However, these findings are limited to short heat exposure periods (< 30 min), untrained

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participants, and unhindered evaporative cooling rates, all of which do not occur within the BMX racing environment. Indeed, contrary to the abovementioned studies, 5 x 15-s maximal sprint performance following 40 min exercise was significantly reduced (-10%) with heat exposure in untrained men (Drust et al., 2005). Furthermore, long duration heat exposure (6 h) has been shown to decrease 45 s sprint performance to the same degree (-10%) in untrained men (King et al., 1985). Thus, long-duration exposure to the heat may compromise BMX sprint performance.

High core temperatures inhibit motor output, a process sometimes referred to as central fatigue, resulting in reduced muscle activation and decreased sprint performance (Drust et al., 2005). As one of the direct effects of HA is a reduction in core temperature during exercise in the heat (Adams et al., 1960), the use of HA for sprint athletes could be beneficial to performance during competitions involving multiple rounds in hot environments. Of the limited research available on HA in athletes performing in sprint activities, it has been shown that long-term exposure (4 months) in the heat can improve neuromuscular capacity through increased maximal force production and explosive power performance of the leg extensors (Rintamäki et al., 2012). Furthermore, 10 d of HA was shown to decrease exercising core temperature (-0.4°C) and increase peak power output (2%) during repeated sprints (Castle et al., 2011). In light of these findings, it is possible that HA might be a useful strategy for improving sprint performance in BMX competitions held in hot environments.

Typical short-term HA procedures consist of long duration (45-90 min), moderate-intensity endurance training sessions, and might interfere with planned BMX training sessions that predominantly involve resistance training, sprint training and skill development (Debraux & Bertucci, 2011a, 2011b). However, studies have shown that

sport-specific training can be performed in the heat to simultaneously induce HA without jeopardizing skill-based work (Buchheit et al., 2013; Buchheit et al., 2011). Therefore, the aims of this study were to 1) examine the effects of acute heat stress on BMX sprint performance, and 2) determine the effects of sprint and strength HA, on thermoregulatory factors and repeated sprint performance in the heat in elite BMX athletes.

5.3 Methods

Participants

Seven elite BMX cyclists (age = 23 ± 4 years; body mass = 78 ± 10 kg; height = 179 ± 7 cm), including 4 males (peak power output = 23.9 ± 0.4 $W \cdot kg^{-1}$) and 3 females (peak power output = 17.3 ± 1.5 $W \cdot kg^{-1}$) from the New Zealand National team participated in this study during a pre-season training camp. Oral contraceptives were being taken by 2 of the female athletes and the 3rd reported regular menstrual cycles. Participants were instructed to maintain a normal diet for the duration of the study, as well as to refrain from strenuous exercise (24 h), alcohol (12 h) and caffeine (3 h) prior to any testing. Participants provided written informed consent prior to study commencement, and ethics approval for the study was obtained through the Auckland University of Technology Research Ethics Committee.

Experimental design

The design of this study was a pre-post, single group, controlled trial. Participants completed a heat response test (HRT) and repeated sprint test, first in temperate (20°C, 50% RH), then in hot conditions (35°C, 50% RH) (Ward Chandler,

Auckland NZ). These initial trials determined the heat-induced decrement in repeated sprint performance and also served as the baseline measurements prior to commencing 5 d of sprint and strength HA. This mixed-methods HA protocol consisted of 3 resistance training sessions (Monday, Wednesday and Friday) in a heated gym (30°C, ~40% RH) while wearing long sleeves and pants (Figure 5.1). Resistance training sessions consisted of 3 strength (2 lower body and 1 upper body; 4 x 4 reps at 85% 1 RM; 2-3 min recovery interval) and 3 power exercises (1 whole body and 2 lower body; 3 x 3-5 reps at body weight; 2-3 min recovery interval). On alternate days, (Tuesday, Thursday), participants performed sprint-interval training sessions in a climate chamber (35°C, 50%; Design Environmental, Wales, UK). A final HRT and repeated sprint performance test was completed ~ 72 h post HA after a weekend where recovery was emphasised (active recovery, good nutrition, sleep). Throughout the study, all training occurred in the usual team environment and controlled high performance laboratory settings. Participants were provided with dietary guidelines and asked to recall their dietary food intake for the 24 h period prior to the first testing session, and to replicate this diet in the 24 h period prior to subsequent trials. Food and fluid intake during the training intervention was also monitored and recorded.
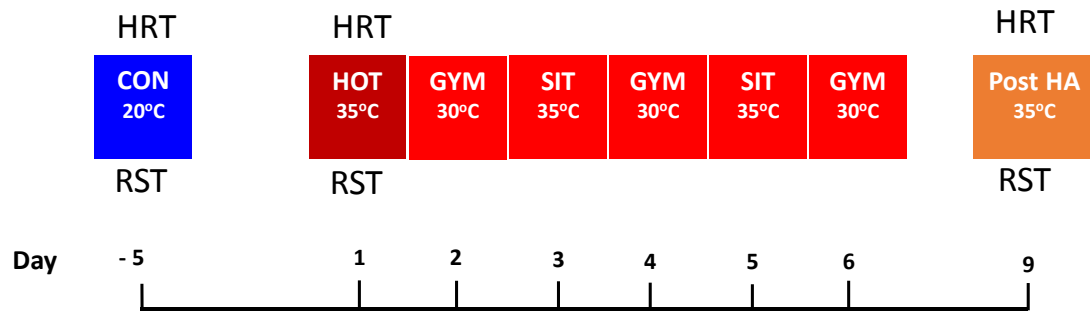
Figure 5.1 Experimental overview. A heat response test (HRT) and repeated sprint test (RST) were performed in both temperate (20ºC, 50% RH) and hot conditions (35ºC, 50% RH), before 5 days of heat acclimation (HA) during gym and sprint interval training (SIT) sessions in the heat. A final HRT and RST was performed in the heat ~ 72 h after the final HA exposure.

HRT and repeated sprint performance tests

The HRT and repeated sprint performance test were designed to mimic aspects of the athlete's routine training. All testing sessions were conducted in the afternoon at the same time of day (13:00). Upon arrival at the laboratory, a resting venous blood sample was drawn and participants were instrumented before entering the climate chamber, where they adjusted to the conditions for 5 min. The HRT was comprised of 20 min of cycling (SRM Indoortrainer, Jülich, Germany) at a power output of 2 W·kg^{-1} of body mass. Rectal temperature (T_{re}) was recorded continuously with a data logger (Squirrel SQ2020, Cambridge, UK) connected to a probe (Thermistor 400, Mon-a-Therm, Mansfield MA, USA) self-inserted ~12 cm past the anal sphincter. Skin temperature was measured at 10 s intervals using wireless telemetric sensors (Maxim DS1921G, San Jose, California) placed on the chest and mid-thigh on the right side of the body. Heart rate and power output were recorded every 15 and 0.5 s, respectively, while thermal discomfort (1-10 scale) and sensation (1-14 scale) (Gagge et al., 1967) were recorded at 5 min intervals. Sweat rate was estimated using the change in total

body mass (undergarments only, towel dried; Weightec, Albany, NZ) that occurred during the HRT. Participants then completed a single inertial sprint, whereby power was measured using the inertial load method using torque readings applied to a fly wheel over a range of pedalling frequencies (Martin, Wagner, & Coyle, 1997). Briefly, cyclists were started from rest in a standing BMX position and were instructed to accelerate maximally for 3-4 s with verbal encouragement. Peak power output, optimal cadence (pedalling frequency at peak power production) and time to peak power were recorded for each sprint using a custom-built inertial ergometer (Goldmine, Auckland, New Zealand). For this group of athletes, the inertial sprint was performed every 2-4 weeks as part of their routine monitoring and results were used as an indication of expected performance outcomes. The typical error for the inertial test was 40 W (2.4% coefficient of variation; CV) for peak power output and 4 rpm (2.8% CV) for optimal cadence. Participants then rested for 5 min before starting the repeated sprint performance test on the same ergometer, which consisted of 3 sets of 3 x 10 s of maximal sprinting, each sprint separated by 10 s of passive recovery, with 10-min breaks between sets. Full BMX protective clothing and helmets were worn for all repeated sprint tests in both temperate and hot conditions to simulate race-like conditions.

Blood samples were collected via venepuncture in K2EDTA vacutettes (Greiner Bio-One, Kremsmunster, Austria). For the resting blood sample, participants were instructed to consume 500 mL of water before arrival at the laboratory, at which point they sat for 20 min to allow plasma stability. The samples were immediately refrigerated and analysed in triplicate within 1 h for red blood cell count, haematocrit (Hct) and haemoglobin (Hb) concentration (AcT 5diff, Beckman Coulter, Miami FL,

USA). Estimated change in plasma volume (PV) with HA was calculated using the Dill and Costill equations (Dill & Costill, 1974).

Statistical analysis

All data were reported as means ± standard deviations (SD), and mean (90% confidence limits; CL) as appropriate. The differences in performance, physiological and subjective measurements were analysed using a magnitude-based inference approach (Hopkins et al., 2009). This method is used to indicate the possible benefit or harm of each condition. For optimal cadence and sweat rate, the magnitude of differences in the changes between trials were expressed as standardised differences (Cohen effect sizes, ES). The criteria used for interpreting the magnitude of the ES for these two variables were: < 0.2, trivial; 0.2-0.5, small; 0.5-0.8, moderate; and > 0.8, large (Hopkins et al., 2009). The smallest worthwhile change (SWC) for peak power output (3.4%; 56 W) was calculated as $\sqrt{2}$ (CV of typical error), which is the bottom limit of useful change for performance (Gore, 2004). A novel approach for magnitude thresholds was used to determine the SWC for thermoregulatory variables, in which the possible range of change was transformed into a full scale of deflection (FSD) (Hopkins, 2010). In brief, each range was made from 0-100% and magnitude thresholds were defined as 10%, 30%, 50%, 70% and 90% for small, moderate, large, very large and extremely large changes. Effect sizes, with uncertainty of the estimates shown as 90% CL, were determined using published spreadsheets (xParrallelGroupsTrial.xls) from sportsci.org (Hopkins et al., 2009). Quantitative chances of HA measurements affecting performance were assessed qualitatively as follows: <1%, *most unlikely*; 1-5%, *very unlikely*; 5-25%, *unlikely*; 25-75%, *possible*; 75-95%, *likely*; 95-99%, *very*

likely; >99% *most likely* (Hopkins et al., 2009). When an effect was > 5% for both benefit and harm, the true value of the difference was described as unclear.

5.4 Results

Performance outcomes

Performance-related responses, percentage changes in performance, percentage chances, and qualitative assessments comparing conditions (CON vs. HOT; HOT vs. Post HA) are shown in Table 5.1. Average peak power output during the inertial sprint and maximal sprint sets are shown in Figure 5.2. The individual and average overall peak power output for the trials relative to the normative data from temperate conditions are presented in Figure 5.3.
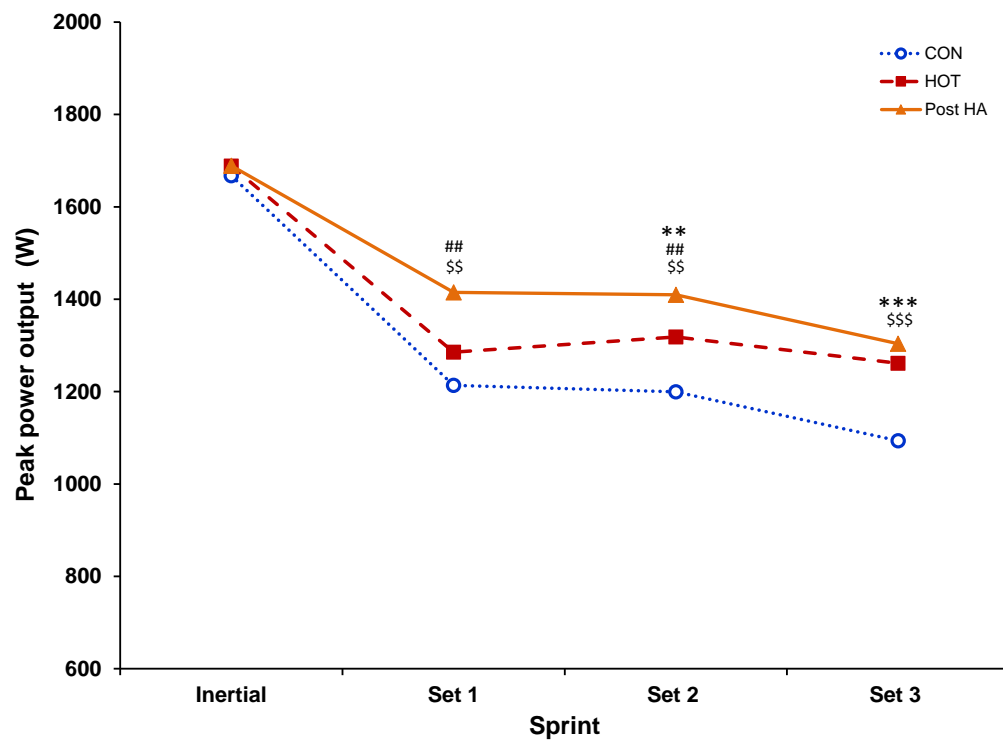
Figure 5.2 Mean values for peak power output (watts; W) during the 3-s inertial sprint and the 3 sets of maximal sprinting during the CON, HOT and Post heat acclimation (HA) trials (see text for details). CON vs. HOT: **possible* increase, ***likely* increase, ****very likely* increase; HOT vs. Post HA: #*possible* increase, ##*likely* increase, ###*very likely* increase; CON vs. Post HA: $*possible* increase, $$*likely* increase, $$$*very likely* increase.
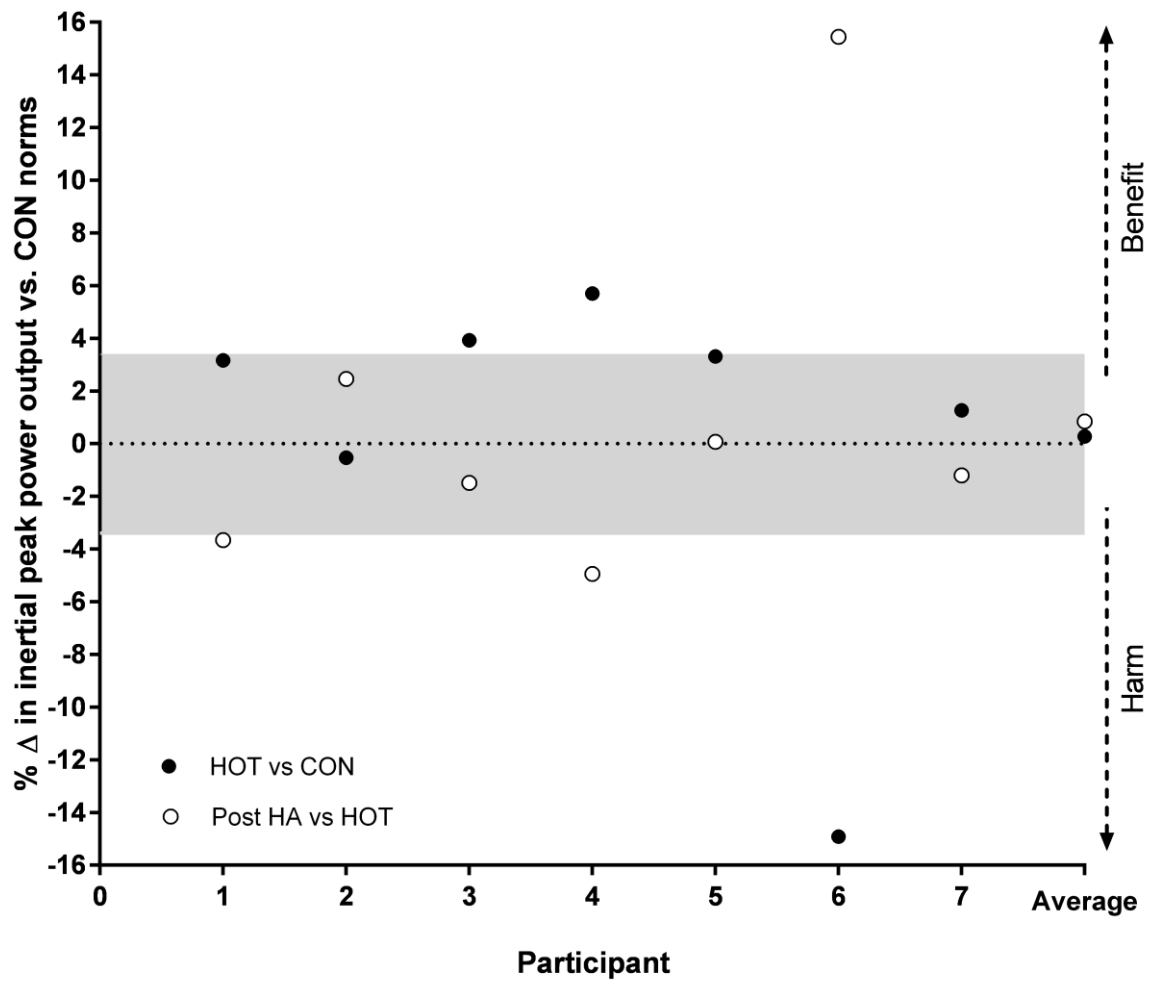
Figure 5.3 The individual and average responses in inertial sprint peak power output in hot conditions (HOT) versus temperate norms (Norms) (black, closed circles) and HOT versus post-heat acclimation (Post HA) (white, open circles). The grey shaded bar represents the smallest worthwhile change (3.4%) in power output.

Table 5.1 Overall performance and thermoregulatory responses (mean ± SD), mean differences (mean ± SD; ± 90% CL) and qualitative assessments during the sprinting sets for all trials.

Outcome measure	CON	HOT	Post HA	SWC	HOT vs. CON		Post HA vs. HOT	
					Difference	Qualititative	Difference	Qualititative
Peak power output (W)	1229 ± 220	1328 ± 284	1407 ± 352	56	99 ± 113 ; ± 83	*Small* ↑**	79 ± 83 ; ± 61	*Small* ↑##
Optimal cadence (rpm)	151 ± 21	145 ± 19	132 ± 16	4.3	-6.4 ± 8.4 ; ± 6.2	*Small* ↓*	-13.0 ± 7.7 ; ± 5.7	*Small* ↓###
Thermal discomfort (AU)	3.8 ± 1.3	4.9 ± 1.6	3.6 ± 1.4	0.9	1.2 ± 1.1 ; ± 0.8	*Small* ↑*	-1.4 ± 2.2 ; ± 1.8	*Small* ↓##
Thermal sensation (AU)	9.1 ± 0.9	9.7 ± 1.2	9.4 ± 1.0	0.7	0.6 ± 1.1 ; ± 0.8	*Small* ↑*	-0.3 ± 2.2 ; ± 1.8	*Trivial*##
Rectal temperature (^{o}C)	38.43 ± 0.05	38.54 ± 0.15	38.47 ± 0.12	0.3	0.11 ± 0.14 ; ± 0.10	*Trivial***	-0.07 ± 0.11 ; ± 0.08	*Trivial*####
Skin temperature (^{o}C)	32.49 ± 0.87	35.76 ± 0.71	35.34 ± 0.54	0.8	3.27 ± 1.01 ; ± 0.74	*Mod* ↑***	-0.42 ± 1.06 ; ± 0.87	*Trivial*##
Heart rate (bpm)	117 ± 14	134 ± 13	131 ± 12	13	17 ± 4 ; ± 3	*Small* ↑***	-3 ± 13 ; ± 11	*Trivial*####
Sweat rate (L·h^{-1})	0.49 ± 0.13	0.78 ± 0.26	0.99 ± 0.32	0.03	0.29 ± 0.23 ; ± 0.17	*Large* ↑**	0.21 ± 0.45 ; ± 0.33	*Unclear*

↓ = decrease, ↑ = increase, AU = arbitrary unit, bpm = beats per minute, CON = control condition (20ºC, 50% relative humidity (RH)), HA = heat acclimation, HOT = hot condition (35ºC, 50% RH), rpm = revolutions per minute, W = watts. CON vs. HOT: **possible* change, ***likely* change, ****very likely* change; HOT vs. Post HA: #*possible* change, ##*likely* change, ###*very likely* change.

Thermoregulatory responses

Average thermoregulatory responses over time, including thermal discomfort and sensation, rectal temperature, skin temperature and heart rate are shown in Figure 5.4. HA had a likely trivial effect on the change in pre-exercise plasma volume (+0.8 ± 4.3%). With the exception of rectal temperature, all thermoregulatory measures showed a meaningful increase in the HOT compared with the CON condition, with a decrease in thermal discomfort during the Post HA session. The details of the group changes are shown in Table 5.1, while sex differences are displayed in Table 5.2.
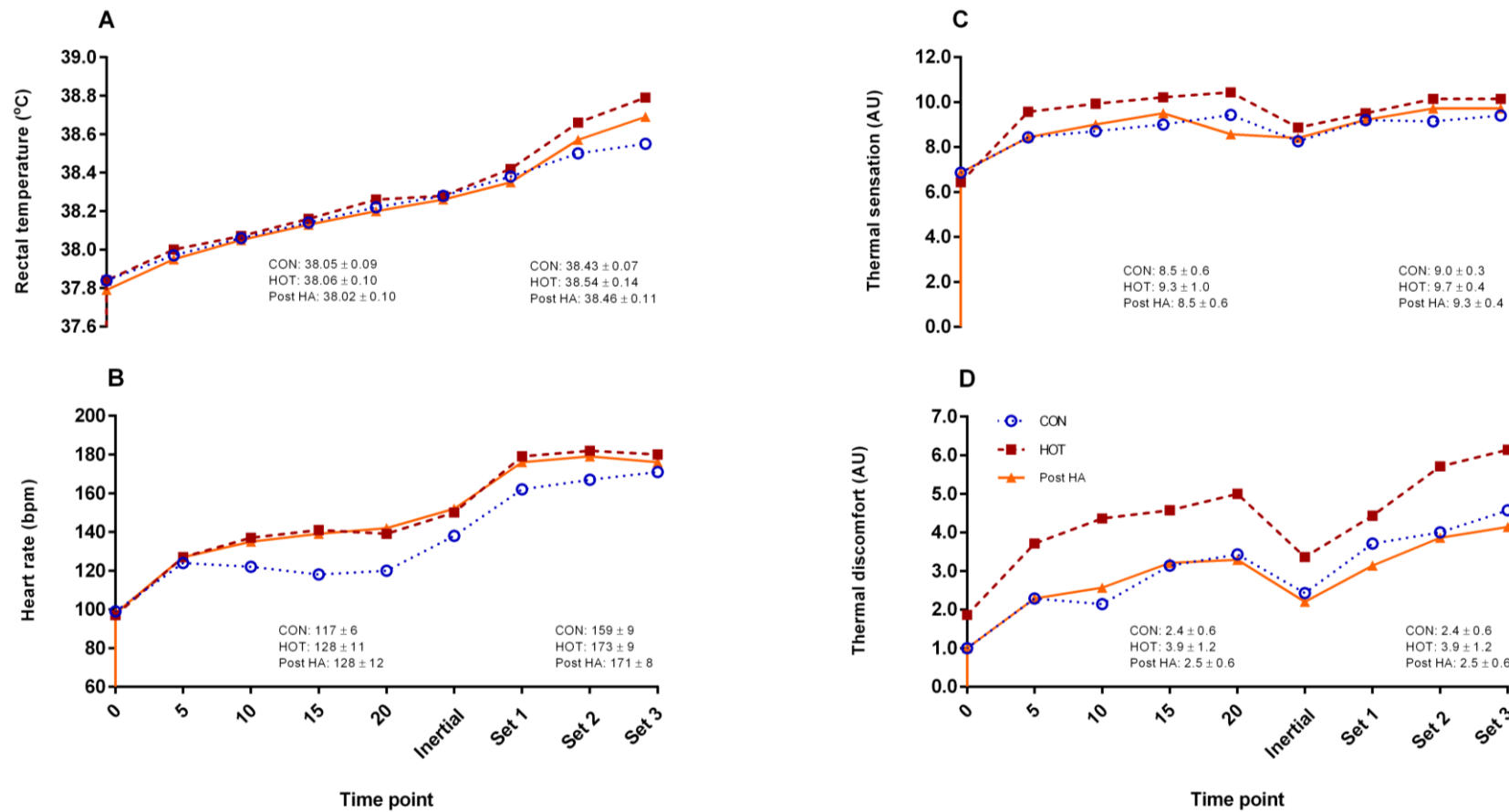
Figure 5.4 Measured response of A) rectal temperature, B) heart rate, C) thermal sensation, and D) thermal discomfort, during temperate (CON; open circles), pre (HOT; closed squares) and post heat acclimation (Post HA; closed triangles). Values are the mean response of each respective trial and measure. The overall mean values and 90% confidence limits are represented at the bottom left corner of each graph.

Table 5.2 Female and male thermoregulatory responses (mean ± SD) during the sprinting sets for all trials.

Outcome measure	Female			Male		
	CON	HOT	Post HA	CON	HOT	Post HA
Thermal discomfort (AU)	3.3 ± 0.7	4.9 ± 0.5**	3.0 ± 0.2###	2.6 ± 0.6	3.9 ± 1.5**	2.8 ± 1.0#
Thermal sensation (AU)	8.5 ± 0.8	9.6 ± 0.7**	9.2 ± 0.4	8.8 ± 0.6	9.4 ± 0.4	8.6 ± 0.1#
Rectal temperature (^{o}C)	38.50 ± 0.02	38.73 ± 0.08	38.71 ± 0.14	38.55 ± 0.04	38.83 ± 0.16	38.67 ± 0.19
Skin temperature (^{o}C)	33.11 ± 0.37	36.06 ± 0.39**	35.72 ± 0.46	32.24 ± 0.62	35.45 ± 0.71***	35.23 ± 0.45
Heart rate (bpm)	128 ± 11	140 ± 6*	141.3 ± 12	114 ± 17	134 ± 18***	130 ± 15
Sweat rate (L·h^{-1})	0.11 ± 0.08	0.23 ± 0.09***	0.37 ± 0.17	0.25 ± 0.12	0.26 ± 0.11	0.33 ± 0.05

AU = arbitrary unit, bpm = beats per minute, CON = control condition (20°C, 50% relative humidity (RH)), HA = heat acclimation, HOT = hot condition (35°C, 50% RH). CON vs. HOT: **possible* increase, ***likely* increase, ****very likely* increase; HOT vs. Post HA: #*possible* decrease, ###*very likely* decrease.

5.5 Discussion

This study is the first to examine both the effects of acute heat stress and sport specific HA on thermoregulatory and repeated sprint performance in elite BMX athletes. The main findings were that peak power output increased and thermal strain was higher in HOT compared with CON conditions. Post HA, a further increase in peak power output was observed, in parallel with improvements in thermal perception. However, no changes in physiological adaptations associated with HA occurred. Overall, both performance and physiological responses were highly individual. While acute heat stress does not appear to impair repeated sprint performance in elite BMX athletes, sport-specific HA might improve a sprint athlete's comfort and cooling ability during training and competing in hot environments.

Performance outcomes

The acute effect of heat on sprint cycling performance revealed a $+7.1 \pm 7.7$ % increase in overall peak power output (HOT vs CON; Table 5.1). While peak power output during the inertial sprint ($0.0 \pm 6.8\%$) and set 1 ($+5.8 \pm 12.5\%$) of the repeated sprint protocol were similar, there was a small *likely* and small *very likely* increase in set 2 ($+9.4 \pm 16.1\%$) and 3 ($+14.5 \pm 11.7\%$) performance, respectively, in the HOT versus CON trial (Figure 5.2). Previous studies have shown that the application of heat, either through passive warming or hot ambient conditions, increases single sprint peak power output by 11-25% in untrained individuals (Ball et al., 1999; Sargeant, 1987), while repeated sprint peak power output can improve by 3-8% (Falk, Radom-Isaac, et al., 1998; Girard et al., 2013), all in the absence of hyperthermia (core temperature $>39^{\circ}C$)

(Drust et al., 2005). Enhanced sprint performance in the heat might be explained by the velocity-dependant effect of muscle temperature on peak power output, with a ~4% increase in power output occurring for each $1^{\circ}C$ muscle temperature rise, as a result of the higher pedalling frequency rates (Sargeant, 1987). While muscle temperature was not measured in the present study, there was a moderate *very likely* increase in skin temperature in HOT compared with CON conditions (Table 5.1), although it is acknowledged that a change in skin temperature alone does not sufficiently predict changes in intramuscular temperature (Jutte, Merrick, Ingersoll, & Edwards, 2001).

The rate of pedalling frequency, as measured by peak and optimal cadence was higher during the first set of the repeated sprints, with *likely* trivial effects (Δ peak cadence = +1.2 ± 1.2 %) and small *possible* decreases (Δ optimal cadence = -4.2 ± 5.6%) during sets 2 and 3 in the HOT compared with the CON trial. Given that pedalling frequency and optimal cadence was increased in the HOT trial only during the first set, when power did not change, the velocity-dependant effect of heat on power output does not seem to explain the results of the present study. As the temperature dependence of the maximal shortening velocity of skeletal muscle is more pronounced in slow versus fast twitch muscle (Bottinelli, Canepari, Pellegrino, & Reggiani, 1996), it is possible that the effect of heat on cadence may be blunted in this elite sprint population, given that sprinters have a higher composition of fast twitch muscle fibres (Costill et al., 1976). Elevations in muscle temperature during exercise have also been shown to augment muscle sympathetic nerve activity (Ray & Gracey, 1997), nerve conduction velocity and skeletal muscle adenosine triphosphate turnover (Gray et al.,

2006), which may further explain the increased power output shown in the HOT condition.

After 5 d of HA, through performing resistance and sprint training sessions in the heat, overall mean peak power output increased a further 7.6 ± 8.0%, with small *likely* increases shown in repeated sprint sets 1 and 2, but not 3 (Figure 5.2). We are aware of only one other study that has examined the effects of HA on repeated sprint cycling performance in the heat (~ 33°C, 50% RH). In that study, Castle et al. (2011) measured cycling peak power output in moderately-trained male team sport players during 20 x 5 s 'all out' efforts, separated by 105 s recovery. Performance increased by ~ 2% following 10 d of traditional HA (60 min cycling at 50% V̇O₂peak) (Castle et al., 2011). However, given only 1 familiarisation session was performed, the small increase in peak power output shown could have been due to a learning effect, as two familiarisation sessions are recommended for non-cyclists performing repeated sprint cycling protocols (Mendez-Villanueva, Bishop, & Hamer, 2007). In the present study, our elite BMX cyclists were highly familiar with the sprint cycling and inertial sprint test protocols used, as they are tests used routinely during the athletes' regular high performance testing routine. Short-term (4 d), sport-specific HA has been examined also in trained female team sport athletes (Sunderland et al., 2008) using an intermittent running protocol (2-3 sets of 10 x 15 m sprints separated by variable speed shuttle running) in hot conditions (30°C, 24% RH). After just 4 HA sessions, running distance during the intermittent running test increased by 33%, despite no change in sprint performance. The large increase in running distance was attributed to an attenuation of thermal stress (decreased exercising core temperature and thermal discomfort),

compared with the unacclimated state. While comparisons are difficult due to the elite training status of the athletes and cycling performance test used in the current study, the mechanisms behind the higher peak power output in the Post HA trial are likely consistent with those mentioned previously regarding the effects of acute heat exposure on sprint performance (Girard et al., 2015).

Thermoregulatory response

The HOT condition increased thermal strain compared with CON, as shown by increases in skin temperature (+3.3 ± 1.0^{o}C; moderate, *very* large), heart rate (+17 ± 4 bpm; small, *very likely*), sweating rate (+0.29 ± 0.23 L·h^{-1}; large, *likely*), as well as thermal perception (small, *possible* increase) without changes in rectal temperature (Table 5.1). These results compare with others showing that repeated sprint efforts in the heat induce physiological alterations that enhance the capacity for heat dissipation (Castle et al., 2011; Girard et al., 2013). The overall rise in rectal temperature (+0.11 ± 0.14^{o}C) was *very likely* trivial, however the largest difference was seen at the end of exercise in HOT (38.79 ± 0.13^{o}C) compared with CON (38.55 ± 0.03^{o}C). Girard et al. (2013) and Castle et al. (2011) showed similar increases in peak core temperature (~38.30 - 38.80^{o}C) and peak power output (+1-3%) in repeated high-intensity exercise scenarios, supporting the notion that repeated mean sprint performance is enhanced (Ball et al., 1999; Girard et al., 2013) or does not change (Castle et al., 2011) in the absence of hyperthermia. Hyperthermia (core temperature > 39°C) may impair sprint power output, as shown by Durst et al. (2005), who examined intermittent sprint performance (15 s on, 15 s off) in hot conditions (40°C), and showed that core

temperature was elevated rapidly to 39.5°C during the 40 min protocol (Drust et al., 2005). It was reported that impairments were not explained by the accumulation of metabolic fatigue markers but instead possibly related to central nervous system adjustments in response to the high core temperature. While high core temperatures cause afferent feedback that induce inhibition of motor output and reductions in maximal voluntary muscle activation (Morrison et al., 2004; Thomas et al., 2006) end rectal temperate in the present study was slightly below hyperthermic levels that would be likely to impair sprint performance (Figure 4.4). Nevertheless, caution should still be advised for BMX athletes that train or compete in hot environments for extended periods (> 60 min) without prior HA, as core temperature could continue to rise towards hyperthermic values that have the potential to impair both athlete health and sprint performance.

Thermoregulatory adjustments from 5 d of sprint and resistance training in the heat resulted in minimal HA adaptations through a small *likely* decrease in thermal discomfort during all time points of the HRT and repeated sprints sets (Figure 5.4). While end exercising rectal temperature was slightly lower Post HA (-0.10 ± 0.14°C, Figure 5.4), this was not a statistically meaningful reduction. This outcome parallel's the lack of changes in skin temperature, heart rate, sweat rate and thermal sensation following HA (Table 5.1). These data align with other studies showing how short-term HA (4-7 d) has a minimal effect on skin or core temperature during exercise (Petersen et al., 2010; Racinais et al., 2012; Sunderland et al., 2008). Two of these studies involved multiple sets of intermittent sprint exercise (30-45 min) over four consecutive (Petersen et al., 2010) or intermittent (Sunderland et al., 2008) days of HA. In the present study,

BMX-specific resistance and sprint training were conducted in the heat. All sprints were < 10 s with long rest periods (5-10 min), while resistance training sessions were strength- and power-focused (low repetition and long rest duration between sets) (Stone et al., 1982), without endurance exercise training. Previous work in Chapter 4 of this thesis had monitored core temperature during similar sprint and resistance training sessions in elite sprint track athletes, showing that the core temperature rate of rise is slow, typically peaking at ~ 38.0-38.5°C over 60-75 min period. Given that HA adaptations are thought to be dependent on time spent exercising at a particular core temperature range in the heat (Houmard et al., 1990), it is possible that the anaerobic nature of the training and low volume of work performed, consistent with other sport-specific, short-term HA protocols (Petersen et al., 2010; Sunderland et al., 2008), diminished the core temperature rise during HA sessions and was not sufficient to elicit further thermoregulatory adjustments typically reported with traditional endurance-based HA (Horowitz, 2001).

Individual and sex differences

In elite sport, there is a high variation in the individual athlete response to training (Borresen & Lambert, 2009; Kraemer, Duncan, & Volek, 1998; Plews, Laursen, Stanley, Kilding, & Buchheit, 2013) and environmental stress (Chapman, Stray-Gundersen, & Levine, 1998; Racinais et al., 2012). Understanding these different responses in athletes is a critical piece of the programming puzzle that coaches and sports scientists require to implement successful training blocks that herald peak performance. Results showed that individual responses varied in both performance and

physiological measurements. For the inertial sprint, 3 of 7 athletes showed increases in peak power output that crossed the smallest worthwhile effect bandwidth, and also happened to be personal bests for them, while 2 athletes showed impairments in inertial peak power in the Post HA condition (Figure 5.3). From a physiological perspective, some athletes showed more complete HA adaptations while others showed minimal changes compared with the first HOT trial. For instance, athlete number 3 (male) showed improvements Post HA in nearly all cardiovascular and thermoregulatory variables that crossed the smallest worthwhile threshold, whereas athlete 6 (female) only showed a worthwhile improvement in thermal discomfort. Interestingly, she was the only athlete of 7 that showed a decrease (-47 W) in her mean peak power output Post HA. Our results and others (Racinais et al., 2012) reinforce the notion that individual responses can vary greatly from the average data, a concept vital to gain more understanding into the narrow-margin world of elite sport. As such, it is recommended that individual responses to both heat and/or HA should be assessed prior to international competition in hot environments so as to identify those that may be predisposed to performance impairment or enhancement (Epstein, 1990).

This relatively small population of athletes included both males and females. It is well established that there are sex differences to acute heat stress responses (Gagnon, Dorman, Jay, Hardcastle, & Kenny, 2009; Gagnon & Kenny, 2012; Moran, Shapiro, Laor, Izraeli, & Pandolf, 1999), and there is emerging evidence showing that some of these differences may translate into delayed HA adaptations in females (Mee, Gibson, Doust, & Maxwell, 2015). Specifically, heart rate and core temperature reductions in females may require long-term HA (> 10 d) to achieve the same reductions in

cardiovascular and thermoregulatory strain that are typically evident in males after short-term HA (< 8 d). After 5 d of HA in the current study, no meaningful effect was shown for core temperature or heart rate in the females or males compared with the initial HOT trial. However, the results in Table 5.2 indicate a small reduction in both parameters in the males (core temperature = -0.12°C; heart rate = -4 bpm) with no change in the females (core temperature = -0.02°C; heart rate = +1 bpm). Therefore, the duration and HA and degree of heat strain may not have been enough to elicit expected sex differences in response to HA.

Limitations

Given that this study was conducted with the entire country's population of elite BMX cyclists, rather than a sample, it was not possible to conduct a randomised control trial, so it is unknown what effect 5 d of resistance and sprint training would have had on the repeated sprint protocol used. Also, a learning effect was possible in the repeated sprint protocol used. While this group of athletes had performed a modified version of this type of training previously, the exact protocol was not performed in the 2 weeks prior to the CON trial. It is recommended that 1-2 familiarisation sessions are necessary to produce reliable peak power results for repeated sprint protocols in non-cyclists (McGawley & Bishop, 2006), but such effects are less apparent in highly-trained (Martin, Diedrich, & Coyle, 2000), and there is unlikely to be a learning effect between the HOT and Post HA trials.

4.5 Conclusion

In the current study, 60 min of low-intensity cycling followed by a repeated sprint protocol in the heat caused increased thermal strain without hyperthermia or other apparent risks of heat illness or injury, and repeated sprint performance was enhanced compared with CON. Short-term HA, through BMX specific resistance and sprint training, induced partial HA adaptations, indicating that HA prior to training camps or competitions with multiple rounds in hot environments could be beneficial for improving athlete comfort and cooling mechanisms through increased sweating rates. Finally, as HA induced a further increase in repeated sprint peak power, future research should explore the ergogenic potential of heat training in sprint athletes.

CHAPTER SIX: ACUTE EFFECTS OF HEATED EXERSICE TRAINING IN HIGHLY-TRAINED FEMALE AND MALE POWER ATHLETES

6.1 Abstract

Purpose: To determine the effects of heated resistance exercise on thermal strain, neuromuscular function and hormonal responses in power athletes. *Methods:* Sixteen (n= 8 female; 8 male) highly-trained power athletes completed a combined strength and power resistance exercise session in hot (HOT ~ 30°C) and temperate (CON ~ 20°C) conditions. Human growth hormone (hGH), cortisol and testosterone concentrations in plasma, peak power (counter-movement jump, CMJ) and peak force (isometric mid-thigh pull) were measured before and after each training session; thermoregulatory responses were monitored during training. *Results:* Skin temperature, thermal sensation and thermal discomfort were higher in HOT compared with CON. Sweat rate was higher in HOT for males only. Compared with CON, HOT had trivial effects on core temperature and heart rate. During HOT, there was a *possible* increase in upper-body power (medicine ball throw) in females [3.4% (90% CL -1.5, 8.6)] and males [(3.3% (-0.1, 6.9)], while lower-body power (vertical jump) was enhanced in males only [3.2% (-0.4, 6.9)]. Following HOT, CMJ peak power [4.4% (2.5; 6.3)] and strength [8.2% (3.1, 13.6)] were enhanced in female athletes, compared with CON, while effects in males were unclear. Plasma hGH concentration increased in females [83% (18; 183)] and males [107% (-21; 444)] in HOT compared with CON, whereas differential changes occurred for cortisol and testosterone. *Conclusion:* Heated resistance exercise enhanced power and increased plasma hGH concentration in female and males power athletes. Further research is required to assess the ergogenic potential of resistance training in the heat.

6.2 Introduction

Increases in body temperature can acutely enhance components of neuromuscular performance, including speed, power and force (Sargeant, 1987). In laboratory settings, increased sprint performance has been shown when single (Sargeant, 1987) and repeated efforts (Ball et al., 1999; Girard et al., 2013) are performed in hot versus temperate conditions. In applied settings, lower-body power output has been shown to increase following passive heating (sauna bathing) in resistance trained men (Hedley et al., 2002), while international track and field competition results from 1999-2011 show that 100 and 200 m sprint performance is improved by ~2% (moderate-to-large effect size) when events occurred in hot ($> 25^{o}C$) compared to cooler environments ($<25^{o}C$) (Guy et al., 2015). Clearly, expected rises in body temperature are associated with acute enhancement of sprint power output across many different settings.

The mechanisms explaining the enhanced sprint and/or power output in the heat may be due largely to the increased muscle temperature and associated neuromuscular contractility and metabolic properties (Gray et al., 2006). For example, Sargeant et al. (1987) showed that increasing muscle temperature to $39^{o}C$ elicited higher peak force and power output (~11%) and pedalling velocity (~13%) compared with lower muscle temperatures (~$37^{o}C$, $32^{o}C$, $29^{o}C$), thus enhancing all aspects of the power-velocity curve (Sargeant, 1987). Increased muscle temperature generally causes a more rapid neural depolarisation (Rutkove, Kothari, & Shefner, 1997), leading to faster muscle fibre nerve conduction velocity (Gray et al., 2006), in turn causing increased sarcoplasmic reticulum calcium release and faster cross-bridge cycling (Karatzaferi, Chinn, & Cooke, 2004). Elevated muscle temperature has also been shown to augment adenosine triphosphate (ATP) turnover via increased phosphocreatine (PCr)

phosphorylation and glycolytic rates (Gray et al., 2006). Combined, such alterations in muscle contractile and metabolic function enhance both force-velocity and power-velocity relationships.

Resistance training is an integral component of any sprint or power athlete's training regime and is used to enhance maximal strength, rate of force development and power production (Haff & Nimphius, 2012). Interestingly, laboratory-based studies have shown that the chronic application of localised heat to skeletal muscle, with or without resistance training, can augment maximal force and muscle cross sectional area (Goto et al., 2011; Goto et al., 2007). For instance, 10 weeks of elbow flexion-extension exercise (4 d per week; 3 x 30 reps at 30% repetition maximum (RM)) with applied heat (steam generating sheet), increased maximal isometric force production (18%) and biceps brachii muscle cross-sectional area (CSA, 8%), compared with the unheated contralateral arm in untrained men (Goto et al., 2007). While the underlying mechanisms are not fully understood, rodent models have shown that heat stimulates the Akt/mammalian target of rapamycin (mTOR) signalling pathway, a key regulator of protein synthesis and hypertrophy, in a temperature-dependant manner (Yoshihara et al., 2013). In humans, heat-induced increases in hypertrophy signalling have been shown when heat is applied to a limb undergoing resistance exercise, compared with the unheated exercising limb (Kakigi et al., 2011). Although not causally linked to hypertrophy, anabolic hormones, such as testosterone and human growth hormone (hGH), may be complementary measures that could offer important insight into the systemic effects of heat and resistance training on skeletal muscle regulation (Crewther, Cronin, & Keogh, 2006). Testosterone is elevated following resistance-exercise and may have an effect on the central nervous system to acutely increase strength (Kraemer

& Ratamess, 2005). Human growth hormone release is also augmented by resistance-exercise (Kraemer & Ratamess, 2005), and has been shown to increase following exercise in hot conditions (Brenner, Zamecnik, Shek, & Shephard, 1997; Ftaiti et al., 2008; Ööpik, Timpmann, Kreegipuu, Unt, & Tamm, 2014). Therefore, it is of interest to determine if resistance exercise in the heat might cause an additive effect on anabolic hormone concentration.

Combined, the aforementioned evidence implies that adding heat to resistance training for strength or power athletes may enhance desired neuromuscular training adaptions. To our knowledge, no previous studies have assessed its effects in a highly-trained population. Therefore, the purpose of this study was to examine the acute effects of resistance exercise performed in hot versus temperate conditions on neuromuscular function, hormone responses and thermal strain in elite female and male power athletes.

6.3 Methods

Participants

Sixteen (n = 8 female; 8 male) highly-trained power athletes participated in this study from sports including weightlifting, powerlifting, athletics (200 m, 400 m sprint; 400 m hurdles) and netball (Table 6.1). All athletes were competing at a level greater than or equal to the New Zealand National Championships in their sport (n=11 international, 6 national-level athletes), and had > 3 years of consistent (i.e. > 3 structured training sessions per week) resistance training experience. Oral contraceptives were used by 4 female athletes, while the others reported normal

menstrual cycles. Self-reporting determined that both training sessions occurred within the same menstrual phase (or quasi-phase for oral contraceptive users) for 7 of the female athletes (n = 6 follicular phase; n =1 luteal phase; n = 1 luteal then menses). Participants provided written informed consent prior to study commencement, and ethics approval for the study was attained through the Auckland University of Technology Research Ethics Committee.

Table 6.1 Participant characteristics and competition level.

Sex	Sport	Competitive level	Age (y)	Height (cm)	Weight (kg)
Female	Athletics	National (n=1)	20	163	59
	Netball	International (n=5)	19 ± 1	179 ± 5	76 ± 7
	Weightlifting	International (n=2)	23 ± 4	157 ± 3	76 ± 29
		Group mean	20 ± 2	172 ± 11	74 ± 13
Male	Athletics	International (n=2); National (n=1)	21 ± 3	183 ± 2	76 ± 5
	Powerlifting	National (n=2)	31 ± 2	186 ± 3	93 ± 4
	Weightlifting	International (n=1); National (n=2)	24 ± 1	172 ± 6	76 ± 9
		Group mean	25 ± 4	180 ± 8	80 ± 11

Experimental design

In a randomised crossover controlled trial, participants completed a combined strength and power resistance exercise session in hot (HOT ~ 30ºC, 40-60% RH) and

temperate (CON ~ 20°C, 40-60% RH) conditions. Each trial was conducted in the afternoon at the same time of day and were separated by 5-7 days. Athletes were familiar with all testing protocols and exercises used within the resistance exercise sessions as they were derived from routine training and testing protocols. Resistance exercises and workloads were held constant for both sessions and consisted of power cleans, parallel back squats, vertical jumps, bench press and seated medicine ball throws, including 3-4 incremental warm-up sets (50%, 65%, 75% 1 repetition maximum (RM)) and 3 work sets at 85-90% 1RM for weighted movements, including 2-3 min rest intervals between sets. Participants were instructed to maintain a normal diet for the duration of the study, and provided a 24 h diet recall journal prior to their first training session, which was replicated before the subsequent training session.
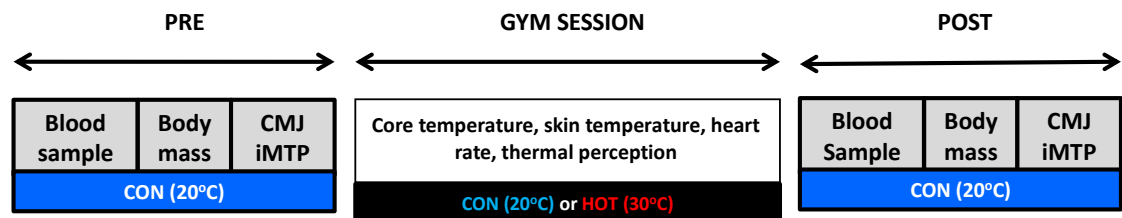
Figure 6.1 Experimental overview. Blood samples, body mass and neuromuscular function tests (CMJ = counter movement jump; iMTP = isometric mid-thigh pull) before and after a resistance exercise session in control (CON = 20°C) versus hot (HOT = 30°C) conditions.

Performance and physiological measurements

Upon arrival for each resistance exercise session, a resting blood sample was taken following a 10 min period of stasis. Following a standardised warm-up, participants performed counter movement jump (CMJ) tests to assess leg power, and isometric mid-thigh pulls (iMTP) to assess whole-body strength. All pre- and post-

testing measurements were carried out in temperate conditions (20°C, 50% RH). Briefly, three CMJs were performed on a force plate (PASCO, Roseville, California; sampling rate 100 Hz), separated by ~ 1 min. Athletes were asked to place their hands on their hips, squat down to ~ 90°, then immediately jump vertically as high as possible, landing back on the force plate with both feet at the same time. The maximal peak power and flight time were recorded (Goldmine, Auckland, New Zealand). For the iMTP, athletes stood on the force plate within a squat rack. A barbell was fixed and positioned in the squat rack so that participants could grasp the bar with straight arms at ~140° knee flexion (Haff et al., 2005). Subjects were instructed to pull up on the bar as quickly as possible while exerting a maximal effort for 5 s. Following 3 warm-up efforts (~60%, 80% maximal iMTP), a total of three trials were performed, separated by 3 min. Force-time curves were measured during each mid-thigh pull, and the highest peak force of the three trials was recorded (Goldmine, Auckland, New Zealand).

Participants then moved into a gym to perform the resistance exercise programme, where temperature was controlled with heat pumps and infrared heating panels (Infracomfort, Napier, New Zealand). Long-sleeved shirts and full-length pants were worn during the HOT condition to increase heat load. During the sessions, core temperature was measured continuously using an ingested telemetric pill (HQInc., Palmetto, Florida), which was ingested 6-h prior to each trial. Skin temperature was measured at 1 min intervals using wireless telemetric sensors (Maxim DS1921G, San Jose, California) placed over the right biceps and mid-thigh. Heart rate was recorded every 15 s, while thermal discomfort (modified scale; 1-10) and sensation (modified scale; 1-14) (Gagge et al., 1967) were recorded at 5-min intervals. For variables measured continuously during the gym sessions, mean session data were calculated.

Athletes were instructed to refrain from drinking fluids for the duration of the resistance exercise session. Sweat rate was estimated using the total body mass (Weightec, Albany, NZ, measured to 0.01 kg) lost during the resistance exercise session (shorts only; towel dried). Total load volume lifted was quantified by recording the number of sets, reps and resistance lifted during each exercise. For exercises requiring a barbell, mean (back squat and bench press) and peak (power clean) barbell velocity was measured using an accelerometer (Gym Aware, Canberra, Australia) and the average of all exercise sets was calculated. During each session seated medicine ball throws and vertical jumps were performed to assess upper- and lower-body power, respectively. To assess vertical jump height, athletes performed a body weight CMJ with a maximal reach. The highest of 4 trials, separated by 30-60 s recovery, was recorded. For the medicine ball throw athletes sat on the floor with their back upright against a wall and legs positioned straight in front with feet approximately shoulder width apart. Athletes were instructed to throw the medicine ball (males = 4 kg; females = 2 kg) as far as possible with both arms from chest height. The maximal distance of 4 trials, separated by 30-60 s recovery, was recorded. Upon completing each training session, participants exited the temperature-controlled gym, and a second blood sample was taken immediately, followed by post-CMJs and iMTPs. Approximately 30 min following resistance exercise, session quality (5 point likert scale) was recorded along with session rating of perceived exertion (sRPE) to assess overall training load (Lockie, Murphy, & de Jonge, 2011).

Plasma hormones

Blood samples were collected via venepuncture in K2EDTA vacutettes (Greiner Bio-One, Kremsmunster, Austria). The samples were immediately analysed in triplicate for red blood cell count, haematocrit and haemoglobin concentration (AcT 5diff, Beckman Coulter, Miami FL, USA), then centrifuged at $4^{\circ}C$ and 3000 rpm for 15 min. Plasma was then extracted and samples were frozen at $-20^{\circ}C$ until later analysis. Plasma testosterone, cortisol and hGH concentrations were analysed using specific diagnostics assays (coeffient of variation (CV) %; testosterone = 2.9%, hGH = 2.3%, cortisol = 1.4%) on a Roche Diagnostics Modular E170 analyser. Total duration of assay for each analyte was 18 min based on the electrochemiluminescence principle (ruthenium-conjugated monoclonal antibodies). Quantitative results were determined via an instrument-specific full point calibration curve. Hormone concentrations were corrected for changes in haematocrit and haemoglobin during the training session (Dill & Costill, 1974).

Statistical analysis

All data are reported as means ± standard deviations (SD) and mean (90% confidence limits (CL)) as appropriate. The differences in performance, physiological and subjective measurements were analysed using a magnitude-based inference approach (Hopkins et al., 2009). This method is used to indicate the possible benefit or harm of each condition. For plasma volume concentrations, sweat rate and training session volume variables, the magnitude of differences in the changes between trials were expressed as standardised differences (Cohen effect sizes, ES). The criteria used for interpreting the magnitude of the ES for these two variables were: < 0.2, trivial; 0.2-

0.5, small; 0.5-0.8, moderate; and > 0.8, large (Hopkins et al., 2009). For changes in plasma testosterone and cortisol, pre versus post comparisons are presented, whereas post-only comparisons were made for plasma hGH data due to the unstable and pulsatile nature of the hormone (Vance et al., 1985). The smallest worthwhile change (SWC) for vertical jump height (1.2%), medicine ball throw distance (2.3%), CMJ (peak power = 0.9%; flight time = 1.0%), iMTP (peak force = 1.7%) and barbell velocity variables (%1 RM and exercise dependant) were calculated based off of CVs from previously collected reliability data. A novel approach for magnitude thresholds was used to determine the SWC for thermoregulatory variables where the possible range of change was transformed into a full scale of deflection (FSD) (Hopkins, 2010). In brief, each range was made from 0-100% and magnitude thresholds were defined as 10%, 30%, 50%, 70% and 90% for small, moderate, large, very large and extremely large changes. Standardised mean differences, with uncertainty of the estimates shown as 90% CL, were determined using published spreadsheets (xPostOnlyCrossover.xls) found at sportsci.org (Hopkins et al., 2009). Quantitative chances of measurements affecting performance were assessed qualitatively as follows: <1%, *most unlikely*; 1-5%, *very unlikely*; 5-25%, *unlikely*; 25-75%, *possible*; 75-95%, *likely*; 95-99%, *very likely*; >99% *most likely* (Hopkins et al., 2009). When an effect was > 5% for both substantial increases and decreases, the true value of the difference was described as unclear. Pearson's product moment correlation analysis was used to assess the association between variables of interest. The magnitude of the correlation (r) between these measures was defined as: <0.1, trivial; 0.1–0.3, small; >0.3–0.5, moderate; >0.5–0.7, large; >0.7–0.9, very large; and >0.9–1.0, almost perfect (Hopkins et al., 2009).

6.4 Results

Training sessions

Six male athletes performed the iMTPs, while the remaining 2 avoided this test based on a coach's preference to take precautions following previous lower back injuries. There were no differences between conditions in total load lifted (kg), sRPE and session quality for female and male groups (Table 6.2).

Table 6.2 Summary of HOT (~30oc) and CON (~20oC) resistance exercise sessions.

	Sex	HOT	CON
Training load (kg)	Female	3913 ± 627	3905 ± 616
	Male	6987 ± 1653	7061 ± 1669
Training load (sRPE)	Female	356 ± 72	354 ± 97
	Male	587 ± 186	524 ± 132
sRPE (AU)	Female	5.1 ± 1.0	4.8 ± 1.3
	Male	6.9 ± 1.8	6.0 ± 1.3
Duration (min)	Female	70 ± 5 [##]	75 ± 7
	Male	85 ± 11	88 ± 12
Session quality (AU)	Female	4.6 ± 0.5	4.3 ± 0.7
	Male	4.0 ± 0.5	3.8 ± 0.9

sRPE = session rating of perceived exertion. [##]likely decrease HOT vs. CON.

Thermoregulatory response

The HOT relative to the CON condition showed trivial effects on average session core temperature [females = 0.03°C (-0.12, 0.18) *very likely*; males = 0.03°C (-0.04, 0.11) *most likely*] and average session heart rate [females = 0 bpm (-5, 5) *most likely*; males = 6 bpm (2, 79) *most likely*] (Figure 6.2). Skin temperature increased in females [3.05°C (2.75, 3.35), moderate *most likely*] and males [1.97°C (1.70, 2.24), small *most likely*] in the HOT compared with the CON condition. Sweat rate increased in the male athletes [ES = 0.6 (0.3, 0.9), moderate *very likely*] during HOT compared with CON, while an unclear effect was shown in females [ES = 0.3 (-0.7, 1.2)]. Thermal discomfort and sensation were higher during the HOT compared with the CON trial in both athlete groups [thermal discomfort; females = 1.4 AU (0.7, 2.1), small *likely*; males = 1.8 AU (1.0, 2.7), small *very likely*; thermal sensation; females = 1.2 AU (0.4, 1.9), small *likely*; males = 1.6 (1.0, 2.3), small *very likely*].
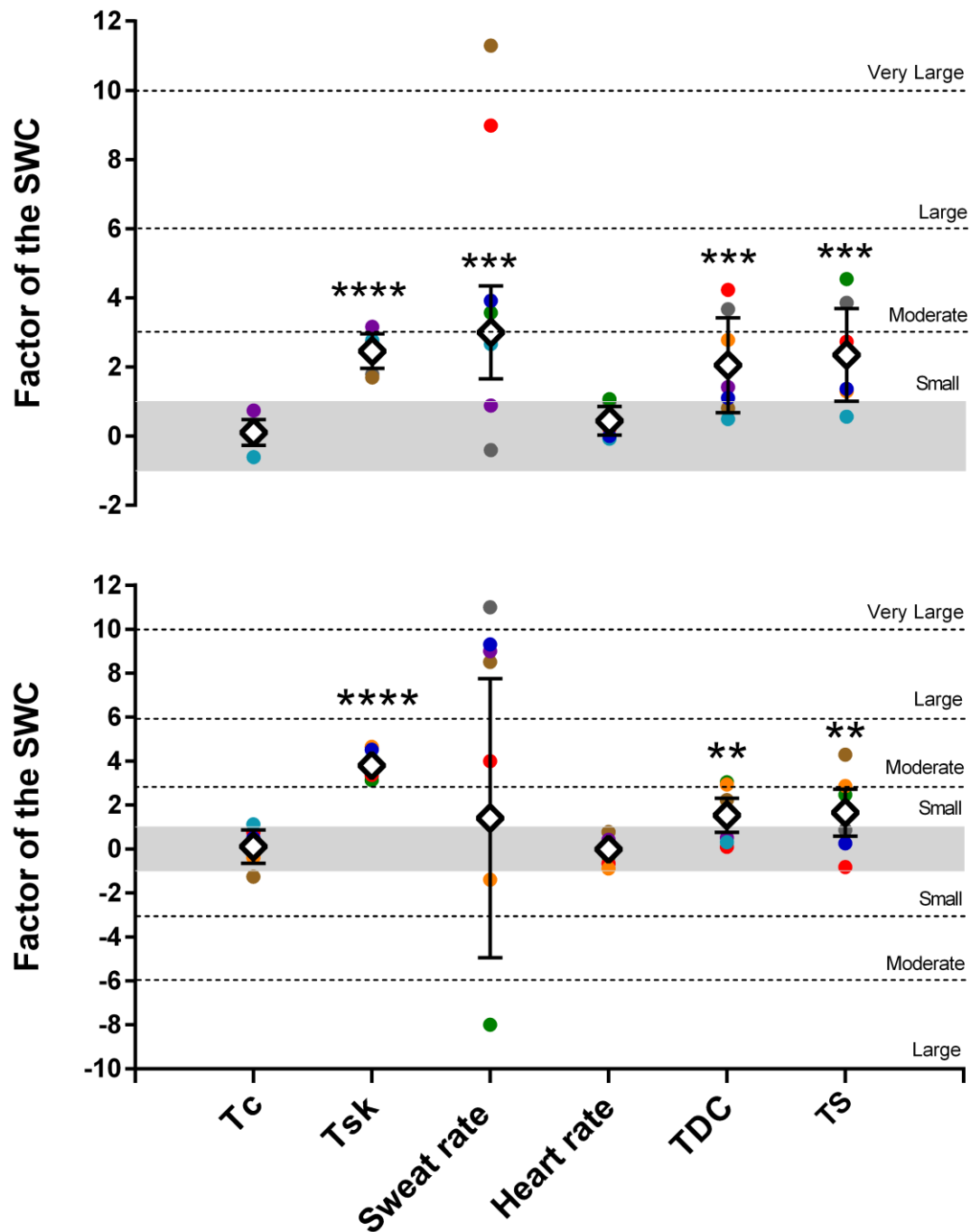
Figure 6.2 Changes (mean ± CL) in thermoregulatory markers including core temperature (T_c), skin temperature (T_{sk}), sweat rate, heart rate, thermal discomfort (TDC) and thermal sensation during resistance exercise in hot (HOT, ~30°C) vs. control (CON, ~20°C) conditions. Panel A includes male data and panel B includes female data. Closed circles represent individual data points. Changes are presented as the factor of the smallest worthwhile change (SWC; grey shaded area) and the magnitude of the effect is quantified as small (1x SWC), moderate (3x SWC), large (6x SWC) and very large (10x SWC). **likely* increase, ***very likely* increase, ****most likely* increase HOT vs. CON.

Neuromuscular function

Heated resistance exercise enhanced neuromuscular function in female athletes. Specifically, iMTP peak force was higher in HOT relative to CON [8.2% (3.1, 13.6) moderate, *very likely*], as was CMJ peak power [4.4% (2.5, 6.3) moderate, *most likely*] and flight time [3.1% (-0.3, 6.6) small, *likely*] in female athletes (Figure 6.3). These effects were unclear in male athletes [peak force = -0.3% (-4.3, 3.8); peak power = 2.3% (-3.4, 8.4); flight time = 0.9% (-1.1, 3)]. There was an unclear effect of the HOT compared with the CON condition on peak barbell velocity for all sets of power cleans in female athletes. Peak barbell velocity for power cleans was increased in set 1 [50% 1 RM = 2.0% (0.4, 3.7) small *possible* decrease] and set 4 [85% 1 RM = 6.8% (-0.6, 14.6) moderate *likely* increase] in the male athletes, while all other power clean sets were unclear in HOT compared with the CON trial. Mean barbell velocity during the HOT condition compared with the CON condition was slower for most sets of back squat (4.6-6.7%) and bench press (6.0-11.9%) in females, while most sets for males had unclear effects, except for a faster barbell velocity during set 1 of the back squat [50% 1 RM = -12.3% (-23.7, 0.9) moderate *likely* decrease] (Figure 6.4). There was a small *possible* increase in medicine ball throw distance during the HOT resistance exercise session [females = 3.4% (-1.5, 8.6); males = 3.3% (-0.1, 6.9)], compared with the CON conditions, while vertical jump height was lower [-2.1% (-4.1, 0.0) small *likely* decrease] in female, and higher [3.2% (-0.4, 6.9) small *likely* increase] in male athletes.
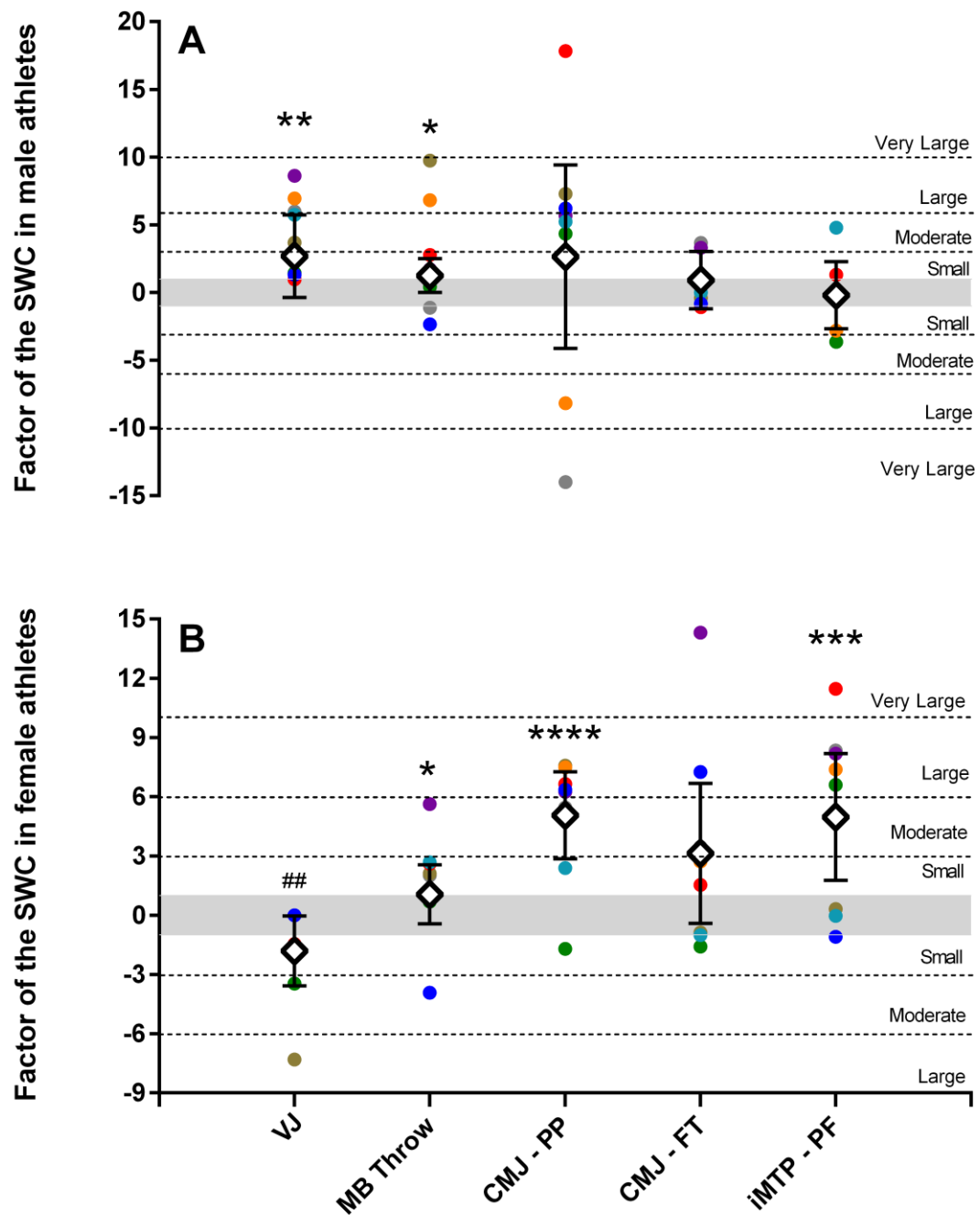
Figure 6.3. Changes (mean ± CL) in neuromuscular markers during (vertical jump height (VJ) and medicine ball (MB) throw distance), and following (counter movement jump (CMJ) peak power (PP) and flight time (FT), and isometric mid-thigh pull (iMTP) peak force (PF)) resistance exercise in hot (HOT, ~30ºC) vs. control (CON, ~20ºC) conditions. Panel A includes male data and B includes female data. Closed circles represent individual athlete data. Changes are presented as the factor of the smallest worthwhile change (SWC; grey shaded area) and the magnitude of the effect is quantified as small (1x SWC), moderate (3x SWC), large (6x SWC) and very large (10x SWC). *possible* increase, ****very likely* increase, *****most likely* increase HOT vs. CON. ##*likely* decrease HOT vs. CON.
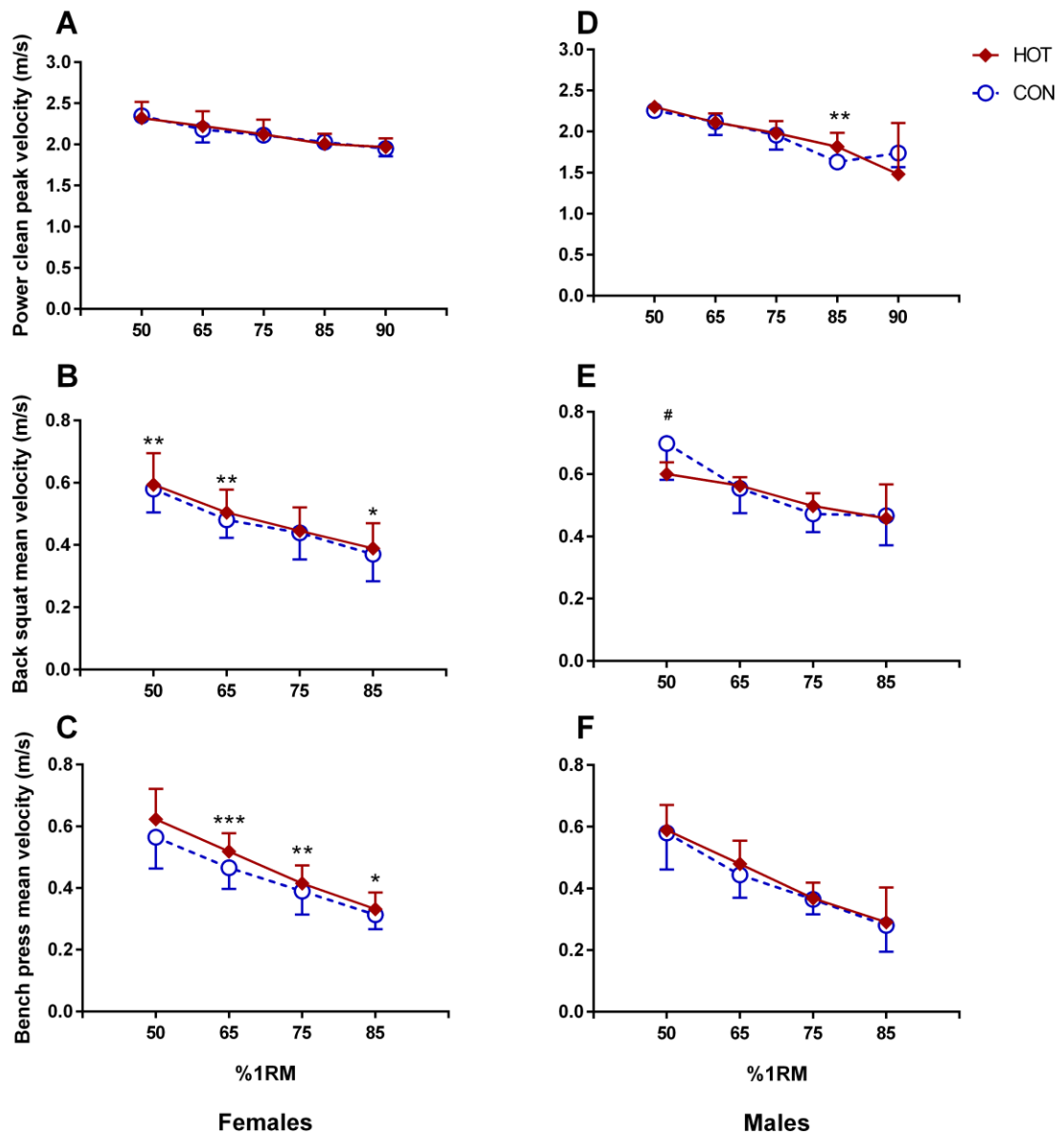
Figure 6.4 Peak and mean (± SD) barbell velocity during resistance exercises at increasing workloads (RM = repetition maximum) in hot (HOT, ~30ºC, solid line) and control (CON, ~20ºC, dashed line) conditions. Panel A, power clean peak velocity; B, female back squat mean velocity; C, female bench press mean velocity; D, male power clean peak velocity; E, male back squat mean velocity; F, male bench press mean velocity. **possible* increase, **likely increase, ***very likely increase, HOT vs. CON. #possible decrease, HOT vs. CON.

Hormone response

Plasma hGH concentration increased in both females [85% (20, 186) small *likely* increase] and males [107% (-21, 444) small *likely* increase], following the HOT compared with the CON condition (Figure 6.5). Differences in plasma testosterone concentration in the HOT compared with the CON condition were

likely trivial [1.7% (-10.8, 16.0)] for females and unclear [3.7% (-12.8, 23.3] for males, while cortisol concentration changes were unclear in both athlete groups [female = 17.1% (-17.8, 66.9); male = -4.7% (-28.7, 27.3)]. There were trivial [ES = 0.0 (-0.23, 0.23)] and unclear [ES = 0.22 (-0.43, 0.86)] changes in testosterone-to-cortisol ratio for female and male athletes respectively in the HOT compared with the CON condition.
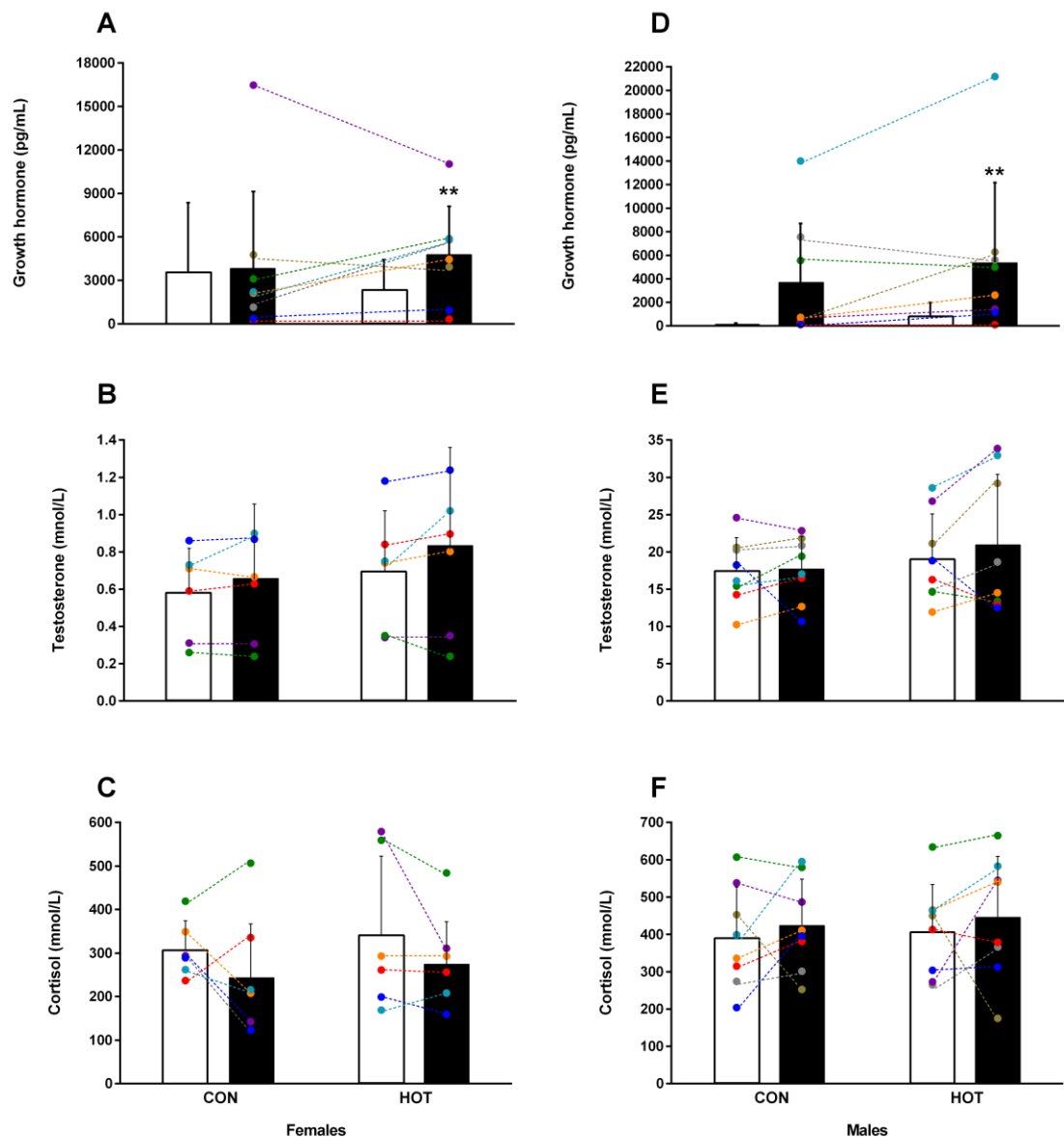
Figure 6.5 Plasma hormone concentration (mean ± SD) pre (white bars) and post (black bars) resistance exercise in hot (HOT, ~30ºC) vs. control (CON, ~20ºC) conditions. Grey shapes and dashed lines represent individual athlete data. Panel A, female human growth hormone (hGH); B, female testosterone; C, female cortisol; D, male hGH; E, male testosterone; F, male cortisol. Pre and post values were compared between trials for testosterone and cortisol and post-only values were compared for hGH. ***likely* increase HOT vs. CON.

6.5 Discussion

To our knowledge, this is the first study to examine the effects of heated resistance exercise on neuromuscular function, hormone response and thermoregulation in highly-trained power athletes. First, heated resistance exercise increased perceived temperature and discomfort along with skin but not core temperature (or heart rate) in both sexes, as well as sweat rate in males only. Second, heat increased upper-body power during, and peak force and power following, heated resistance exercise in the female athletes compared with control conditions, while male athletes showed enhanced upper- and lower-body power during the heated resistance exercise session but peak force and power were unaffected by the heated resistance exercise session. Third, we showed that heat augmented the anabolic hormone response through increases in plasma hGH concentration, however, heat had no effect on testosterone or cortisol concentration compared with control conditions. Combined, the data suggests that a strength- and power-based resistance exercise programme in the heat may be beneficial for enhancing some contributors to power and strength adaptation in female and male athletes.

Thermoregulatory adjustments

Performing a strength and power based resistance exercise session in the heat elevated thermal strain in power athletes as evidenced through increases in skin temperature and thermal perception in female and male athletes and sweat rate in males only (Figure 6.2). The strength and power-based resistance exercise session performed involved low exercise volume (3-5 reps) and long rest intervals (2-3 min), which meant that athletes were in a passive state for much of the session. As a result, differences in core temperature and heart rate between conditions were trivial. Studies investigating

the effects of heat on low-volume repeated sprint exercise have also shown core temperature elevations to ~ 37.5-38.0°C (Girard et al., 2013; Hoffman et al., 1996). In the current study, peak core temperature rose to similar values (38.0°C). Skin temperature was *most likely* increased in females (~34°C) and males (~35°C) in HOT vs CON, respectively, as shown by Girard et al. (15) where athletes displayed slightly higher skin temperature values (~ 36°C) during a 30 min repeated sprint cycling protocol (10 x 6 s maximal sprints; 5 min recovery; 5 x 6 s maximal sprints) in hot conditions (35°C, 40% RH). Sweat rate was enhanced in the HOT condition in male but not female athletes (Figure 6.2), and is consistent with evidence that attenuated sudomotor function is evident in females including higher sweating thresholds and lower total sweat rate (Gagnon & Kenny, 2012). However, it was noted that 6 of 8 female athletes showed moderate-to-very large increases in sweat rate in HOT, compared with CON, highlighting the individual response nature of heat stress (Racinais et al., 2012).

Neuromuscular function

The effects of heated resistance exercise revealed an increased upper- and lower-body power, with varying results between male and female athletes for force production and barbell velocity. During the HOT trial, vertical jump height was enhanced in males (3.2%) but not females (-2.1%), while seated medicine ball throw was increased in both athlete groups (females = 3.4%; males = 3.3%) (Figure 6.3). Enhanced power during the HOT resistance exercise results are in line with Hedley et al. (2002) who showed a 3.1% improvement in lower-body power through increased vertical jump height following 30 min of sauna bathing in resistance-trained men (Hedley et al., 2002). Following the resistance exercise session in the present study, female athletes showed

an improvement in CMJ peak power and flight time in HOT relative to CON conditions (Figure 6.3). The gain in CMJ peak power was also reflected in 6 of 8 male athletes (moderate to very large increases), however, a large variance was also shown due to 2 athletes having lower measurements in the heat, resulting in an unclear group effect. Although power increases were not shown in both power tests across sexes, the increases evident are in agreeance with others who have shown the ergogenic effect of heat on power production (Ball et al., 1999; Girard et al., 2013; Hedley et al., 2002; Sargeant, 1987) and might be explained by transient increases in muscle temperature leading to increased muscle fibre conduction velocity and a greater utilisation of anaerobic metabolism (Gray et al., 2006).

Heat-induced increases in power output are associated with increased contraction velocity (Ball et al., 1999; Sargeant, 1987). While velocity was not specifically assessed during the vertical jump or CMJ, we did assess barbell velocity during the main resistance exercises in each trial. Male athletes showed faster barbell velocity only during the initial set of back squats, while female athletes showed slower barbell velocity during several sets of back squat and bench press exercises (Figure 6.4). Therefore, our findings contradict those of others who have shown heat-induced increases in power production related to enhanced velocity of a given movement (Ball et al., 1999; Sargeant, 1987). The slower barbell velocity for female athletes was unexpected and may be related to augmentation of skin temperature, thermal sensation and thermal discomfort in HOT compared with CON conditions as elevated body temperature and thermal perception has been shown to cause feelings of discomfort and demotivation during exercise (Brück & Olschewski, 1987).

The effects of heat on muscle force production from the literature is equivocal. Some have shown enhanced short-term maximal force production following passive or dynamic heat exposure (Ball et al., 1999; Sargeant, 1987), while others have shown no effect with (Cheung & Sleivert, 2004; Ftaiti et al., 2001) or without hyperthermia (Girard et al., 2015). During submaximal sustained contractions, hyperthermia impairs central neuromuscular activation, reducing isometric endurance capacity (Morrison et al., 2004). In the present study, maximal isometric force was enhanced in HOT compared with CON conditions in female but not in male athletes (Figure 6.3). As with muscle power, heat-induced improvements in force may be related to increased muscle temperature and conduction velocity (Ball et al., 1999; Gray et al., 2006; Sargeant, 1987). While skin temperature was elevated (~2-3°C), it cannot be inferred that muscle temperature was also elevated. Moreover, the force gain shown in females in HOT versus CON conditions may be due to menstrual cycle-induced fluctuations in strength. During the study, 4 females (non-oral contraceptive users) performed both trials during the follicular phase, where strength levels have been shown to be up to ~10% higher due to increasing oestrogen and testosterone levels (Phillips, Sanderson, Birch, Bruce, & Woledge, 1996). Of these 4 females, only 1 showed an increase in peak force, while those taking oral contraceptives, who are not expected to have cyclic changes in strength (Phillips et al., 1996), did improve their peak force. Therefore, menstrual cycle changes cannot explain the female's improved force production in HOT compared with CON conditions. To summarise, upper- and lower-body power may be enhanced by resistance exercise in the heat, however more work is necessary to understand the effects of acute heat application on force and velocity characteristics of resistance exercise.

Hormone response

Human growth hormone is anabolic in nature, is involved in cell growth and regeneration and has been shown to increase following resistance exercise in males and females (Kraemer & Ratamess, 2005). Heated resistance exercise induced a similar response in female and male athletes with small *likely* increases in plasma hGH concentration compared with CON (Figure 6.5). The hGH response in the present study resembles the response shown in other studies (Brenner et al., 1997; Ftaiti et al., 2008) using low-to-moderate-intensity aerobic exercise in the heat. Thus, changes in hGH may be a result of both core temperature (Ööpik et al., 2014) and exercise intensity (Kraemer & Ratamess, 2005), as both stimuli concomitantly increase blood flow and transport of hGH to skeletal muscle, where it is thought to facilitate Akt phosphorylation involved in protein synthesis (Yoshihara et al., 2013). Although assumptions cannot be made that the elevated hGH shown herein are associated with enhanced protein synthesis signalling, others have shown that heated resistance exercise does indeed elevate Akt/mTOR signalling (Kakigi et al., 2011) and may aid in muscle hypertrophy and strength gains (Goto et al., 2007). Although exercise induced increases in hGH may be augmented with oral contraceptive use (Bernardes & Radomski, 1998) only 2 of 4 oral contraceptive users showed an increase in hGH, while all non-users showed an increase after HOT compared with CON. While lack of menstrual cycle control is a noted limitation in the present work, the randomised sessions would have minimised the effects of increasing oestrogen levels during the follicular phase, which is thought to augment hGH (Bernardes & Radomski, 1998).

Testosterone is an anabolic hormone that has influences on muscle function and behaviour, such as aggression (Harris, Rushton, Hampson, & Jackson, 1996). Although

the testosterone response to various forms of resistance exercise has been well documented (Kraemer & Ratamess, 2005), the effects of heat on systemic testosterone release during exercise is less understood. To our knowledge, only one study has examined this and showed that repeated maximal sprints (2 sets of 5 x 15 s Wingate sprints) in the heat (35°C) had no effect on testosterone concentration compared with control (22°C) conditions in active men (Hoffman et al., 1996). Similar to these findings, the present study showed trivial and unclear effects of heat on plasma testosterone concentration in female and male athletes respectively (Figure 6.5). When post-only values were assessed, a heat-induced small *likely* increase [19.3% (8.0, 31.7)] in testosterone emerged for the female athletes only. As testosterone is associated with aggression, the greater force produced in the iMTP following HOT may be explained by elevated post-exercise testosterone concentration and its effect on increasing vigour (Cardinale & Stone, 2006). In addition, testosterone has been shown to correlate (r = 0.61) with lower-body power (Cardinale & Stone, 2006). In comparison, our data revealed a moderate correlation (r = 0.54) between post-training testosterone concentration and CMJ peak power in HOT vs. CON in males only. When individual responses were assessed, positive and negative responders were evident, with 3 female and 4 male athletes showing meaningful physiological elevations in testosterone concentration in HOT compared with CON trials (Figure 6.5). Such results are not surprising given the high individual variation shown with both hormone response to resistance training (Beaven, Gill, & Cook, 2008) and thermoregulatory response to heat stress (Racinais et al., 2012). Anecdotally speaking, correspondence with a coach of some of the power athletes involved in this study revealed that those athletes who had typically performed poorly in the heat showed meaningful decreases in testosterone and

hGH concentration, as well as higher thermal and cardiovascular strain following HOT compared with CON conditions, while those that he had perceived to perform better in the heat showed an elevated anabolic hormone response. The contrasting individual responses highlight the possible need for some power athletes to undergo heat acclimation to ensure power performance is not impaired in the heat. Furthermore, while we cannot deem that post-exercise hormone response had a hypertrophic effect, such data may be useful when individualising training to optimise individual athlete adaptations during short-term training blocks (Beaven, Cook, & Gill, 2008).

Cortisol is a catabolic hormone released due to different forms of stress including but not limited to exercise and heat (Brenner et al., 1997). Our results showed an unclear effect of HOT compared with CON conditions on plasma cortisol concentration (Figure 6.5). This finding contradicts Brenner et al. (1997) who showed that heat increased cortisol during exercise compared with exercise in control conditions (23°C). Heat-induced increases in cortisol have been shown when core temperature exceeds 38°C and is accompanied by feelings of discomfort (Collins, Few, Forward, & Giec, 1969), which may explain the unclear effects across conditions on plasma cortisol concentration in the present study despite a concomitant minimal rise in core temperature. Furthermore, it has been shown that maximal strength training does not increase cortisol, while muscular hypertrophy and strength-endurance-based resistance exercise training appear to (Smilios, Pilianidis, Karamouzis, & Tokmakidis, 2003). This may help explain why cortisol was not raised in our maximal strength-based protocol.

Practical applications

The present study builds on evidence that heat can enhance power and provides a practical method of integrating heat into training for power sports. Until now, most research involving exercise in the heat has used environmental chambers, where chamber size may limit the modes of training that can be performed. A heated resistance training area may enhance the training stimulus during sport-specific power exercises for athletes in sports such as weightlifting, throws, jumping, and sprinting. The maximal strength and power exercises performed in the heat caused low levels of thermal strain, yet performance during the session was not compromised. Nevertheless, training sessions focusing on muscular hypertrophy or strength endurance are more likely to cause greater thermal heat stress and could reduce session quality. Finally, resistance training in the heat might be useful as an alternative to traditional exercise-based heat acclimation. Future research examining different types of resistance training in the heat and its long-term effects on performance and thermoregulatory adaptations are warranted.

6.6 Conclusion

In summary, a combined strength and power resistance exercise session in the heat caused enhanced power production and increased plasma hGH concentration in highly-trained power athletes. In addition, heated resistance exercise increased maximal force in female but not male athletes. While the hot conditions caused increased thermal strain through augmented skin temperature and thermal perception, core temperature

and heart rate were not affected. Further research is required to assess the ergogenic

potential of resistance training in the heat.

CHAPTER SEVEN: FROM LAB TO REAL WORLD: HEAT ACCLIMATION CONSIDERATIONS FOR ELITE ATHLETES

7.1 Abstract

As major sporting events are often held in hot environments, increased interest in ways of optimally heat acclimating athletes to maximise performance has emerged. Heat acclimation involves repeated exercise sessions in hot conditions that induce physiological and thermoregulatory adaptations that attenuate heat-induced performance impairments. Current evidence-based guidelines for heat acclimation are clear, but the application of these recommendations is not always aligned with the time commitments and training priorities of elite athletes. Alternative forms of heat acclimation investigated include hot water immersion and sauna bathing, yet uncertainty remains around the efficacy of these methods for reducing heat-induced performance impairments, as well as how this form of heat stress may add to an athlete's overall training load. An understanding of how to optimally prescribe and periodise heat acclimation based on the performance determinants of a given event is limited, as is knowledge of how heat acclimation may affect the quality of concurrent training sessions. Finally, differences in individual athlete responses to heat acclimation need to be considered. This article addresses alternative methods of heat acclimation and heat exposure, explores gaps in literature around understanding the real world application of heat acclimation for athletes, and highlights specific athlete considerations for practitioners.

7.2 Introduction

Several major sporting events are held each year in hot environments. Some of the largest high profile events, including most Summer Olympic Games, the Tour de France, the FIFA World Cup, as well as several annual World Cups and World Championships are held in the summer months when high temperatures are often expected. For athletes and support teams preparing for pinnacle events, executing performance to their maximum potential is of the utmost importance. Heat-induced performance decrements can range from 6-16% in trained athletes during endurance and team sport events (Racinais et al., 2012; Sunderland & Nevill, 2005; Tatterson et al., 2000), while lack of acclimation is a major risk factor for exertional heat injury (EHI) (Binkley et al., 2002). Heat acclimation (HA) or acclimatisation involves repeated exercise sessions in hot conditions (typically ~30-40°C, 20-60% relative humidity (RH) for athletic circumstances) either by artificial means (acclimation; heated room or chamber) or outdoors (acclimatisation; hot ambient temperature), and is a routine strategy employed to induce physiological adaptations that will attenuate heat-induced performance impairments and offer protection against heat stress and EHI (Périard et al., 2015; Taylor, 2014; Tyler et al., 2016). Recent consensus recommendations offer practitioners an understanding of the key concepts needed to prescribe HA for individual and team sport athletes (Chalmers et al., 2014; Racinais et al., 2015). Briefly, these best practice guidelines state that HA should be comprised of daily ~ 60 min training sessions in hot conditions for a minimum of 1 week, and ideally over 2 weeks to achieve further thermoregulatory and performance benefits. The HA protocol

should mimic the event demands while inducing high sweat rates and increased body (skin and core) temperature (Racinais et al., 2015).

A number of scenarios have been proposed in the literature to administer HA within an athlete's season to maximise performance in hot conditions, including pre- or in-season training camps to augment the training response, or as a taper tool when training volume is reduced and high-intensity quality is to be maintained (Chalmers et al., 2014; Racinais et al., 2015). While such recommendations are based on decades of evidence, limitations and barriers to adhering to such guidelines, within the context of elite athlete training needs, pose frequent conundrums for coaches and sport scientists. In short, best practice guidelines are often not entirely attainable within the confines of highly demanding physical preparation and travel, which are necessary requirements for the elite athlete. The purpose of this current opinion piece, therefore, is to review alternative methods of HA and heat exposure, explore the gaps in literature for understanding how HA might be integrated into an athlete's existing training program, and highlight specific athlete considerations for practitioners.

7.3 Alternative HA Methods

Sport-specific HA, simulating competition-like conditions, is now considered best practice HA for athletes (Racinais et al., 2015). Unfortunately, several barriers may prevent athletes from achieving HA in this way, especially for those athletes living in cold-to-temperate climates. HA protocol design challenges may include limited access to environmental chambers, constrained training modes not attainable within the small confines of most environmental chambers, high costs associated with international

travel to conduct heat camps, and potential interference that HA may have with higher-prioritised training phase objectives. Alluring alternative HA strategies for athletes, such as post-exercise hot water immersion and sauna bathing, have been shown to elicit the desired physiological adaptations (Scoon et al., 2007; Zurawlew et al., 2015), while overcoming the aforementioned barriers that traditional HA present. These HA alternatives and their reported effects are summarised in Table 7.1 and described briefly below.

Table 7.1 Passive heat acclimation strategies using hot water immersion (HWI) and post exercise sauna bathing (PES).

Study	Participants	Intervention	Protocol	Conditions	Physiological responses	Performance test
Bonner et al. 1976	n = 5 male; untrained	HWI (whole body)	13 x 60 min controlled hyperthermic HWI	HWI ~41°C; T_a = 40°C	↓ T_c, ↓ HR, ↑ sweat rate, ↑ PV (6.7%)	None
Brazaitis et al. 2010	(Exp) n = 13; (Con) n = 12 female & male; untrained	HWI (lower limb)	7 x 45 min HWI sessions over 2 wk; alternate days	HWI ~44°C; T_a = 23°C	0.30°C ↓ T_c, 12 bpm ↓ HR, 0.3 L·h^{-1} ↑ sweat rate, 1.0 AU ↓ PSI	↓ central & peripheral fatigue w/ hyperthermia; no Δ post HA
Shin et al. 2013	n = 9 male; untrained	HWI (lower limb)	10 x 30 min HWI sessions over 3 wk; alternate days	HWI ~42°C; T_a = 26°C	0.13°C ↓ T_c, 0.2 L·h^{-1} ↑ sweat rate,	None
Zurawlew et al. 2015	(Exp) n = 10; (Con) n = 7 male; active	HWI (whole body)	6 x 40 min HWI sessions over 3 wk; randomised between-subjects control trial	~40°C	0.36°C ↓ T_c, 6 bpm ↓ HR, ↔ sweat rate, 0.5 AU ↓ PSI	4.9% ↓ 5 km TT in 33°C
Scoon et al. 2007	n = 6 male; mod-trained	PES	~13 x 30 min PES sessions over 3 w; randomised crossover control trial	~90°C	7.1% ↑ PV, unclear 3.5% ↑ RCV	32% ↑ TTE (~2% ↓5 km TT)
Stanley et al. 2014	n = 7 male; well-trained	PES	10 x 30 min PES sessions; consecutive days	87°C 11% RH	17.8% ↑ PV, 15.6% ↓ HRR$_{60s}$	None reported

AU = arbitrary unit, Con = control group, Exp = experimental group, HA = heat acclimation, HR = heart rate, HRR60s = heart rate recovery at 60 s, mod = moderately, PSI = physiological strain index, PV = plasma volume, RCV = red cell volume, RH = relative humidity, T_a = ambient temperature, T_c = core temperature, T_{sk} = skin temperature. All reported increases (↑) or decreases (↓) were significant changes.

Hot water immersion

Hot water immersion applied in untrained men as a form of passive HA has been shown to enhance thermoregulation (Bonner, Harrison, Hall, & Edwards, 1976; Brazaitis & Skurvydas, 2010; Shin, Lee, Min, & Yang, 2013) and improve exercise performance in the heat (Zurawlew et al., 2015). In one study, seven 45-min hot water baths (44°C) completed over a 2-week period was shown to reduce thermal and cardiovascular strain, through reductions in core temperature (0.30^{o}C) and heart rate (12 bpm) (Brazaitis & Skurvydas, 2010); an effect comparable to what is typically shown after conventional short-term HA (Garrett et al., 2012). Likewise, Zurawlew et al. (Zurawlew et al., 2015) showed reductions in both resting (0.27^{o}C) and end-exercise (0.28^{o}C) core temperature, as well as a 4.9% improvement in 5-km run time trial (TT) performance in hot conditions (33°C) following six consecutive post-exercise hot water baths (40 min running at 65% maximal oxygen consumption ($\dot{V}O_2$max) in 18°C; 40 min bath in 40°C water). This performance improvement seems meaningful given that the coefficient of variation (CV) for 5 km TT performance is 2% (Laursen, Francis, Abbiss, Newton, & Nosaka, 2007). Given many training facilities have hot baths available to athletes for hydrotherapy purposes, hot water immersion holds potential as an accessible and time-efficient means of inducing HA. Further research with trained individuals is needed to understand the effects of hot water immersion compared with best practice HA methods.

Post-exercise sauna

The high thermal load (80-100°C, 10-20% RH) imposed by sauna bathing presents its use as a potentially practical HA approach. Sauna bathing in a rested state has been

shown to impose considerable heat stress, resulting in increased core and skin temperature, sweat rate and heart rate (Hannuksela & Ellahham, 2001). Consecutive days of sauna bathing in healthy untrained men has been shown to increase heat tolerance after only 3 d/exposures, as evidenced by reductions in core temperature (Leppäluoto, Tuominen, Väänänen, Karpakka, & Vuor, 1986). Sauna exposure immediately following a training session may enhance the thermoregulatory-adaptive response, as core temperature, considered a key contributor to HA-induced adaptations (Horowitz, 2001), has been shown to rise to a greater extent compared with sauna bathing without exercise (Ridge & Pyke, 1986). Furthermore, post-exercise heat stress may additively enhance endurance-training induced mitochondrial function, through increased citrate synthase enzyme activity (Tamura et al., 2014). To date, only two studies have reported on the use of post-exercise sauna bathing in trained athletes; however neither study examined the typical spectrum of HA-induced adaptations (Scoon et al., 2007; Stanley et al., 2015). Both studies used ~10-15 post-exercise sauna sessions and reported significant plasma volume expansion (7-17%) (Scoon et al., 2007; Stanley et al., 2015). Only one study showed a possible improvement in running performance, equivalent to a ~2% improvement in 5-km TT performance in temperate conditions (Scoon et al., 2007), and equal to the performance test CV (Laursen et al., 2007). Similar to hot water immersion, post-exercise sauna bathing may be more accessible than heat chambers for some athletes, and can be conveniently added to a training schedule with minimal disruption. While post-exercise sauna bathing is currently recommended by specialists as an HA alternative (Racinais et al., 2015), no studies have reported its efficacy to induce thermoregulatory adaptations and enhance performance in hot conditions.

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7.4 *Integration* with *Training*

Prescription and periodization

The majority of HA-induced adaptations (reduced body temperature, cardiovascular strain, perceived effort and discomfort) are actualised following short-term HA (4-7 d) (Garrett, Rehrer, et al., 2011), with further thermoregulatory (increased sweat rate) (Mee et al., 2015) and exercise capacity enhancement (Guy et al., 2015) requiring moderate-term HA (8-14 d) or even long-term HA ($\geq$ 15 d). Long-term HA, though often overlooked as a HA strategy for athletes, may have merit for athletes preparing to perform ultra-endurance events in the heat, as longer-term HA has been shown to enhance molecular and cellular adaptations leading to improved cardiac contractile efficiency during exercise in the heat, and a greater accumulation of heat shock proteins, albeit in untrained animal models (Horowitz, 2002). Direct comparisons of differing HA intervention length on performance outcomes would aid the development of HA dose recommendations based on competition length, thus providing an evidence-based menu that practitioners could refer to. For instance, long-term HA may be more optimal for endurance events over 2 h, where larger heat performance decrements are often evident (~3%) compared with middle-distance events (1%) (Guy et al., 2015), in which short-to moderate-term HA may be sufficient.

The appeal and ease of implementing short-term HA within an athlete's training schedule has driven research to determine the minimal dose of HA required to elicit thermal adaptations and enhance performance in the heat. Rapid HA with just 2 d of twice daily HA was recently shown to improve 3-km TT running performance (3.5%) in

hot conditions (30°C, 60% RH), in moderately-trained males (Willmott, Gibson, Hayes, & Maxwell, 2016). Five sessions of short-duration (27 min) high-intensity intermittent HA, over a 9 d normal training period, in Australian Rules Football players, reduced RPE, thermal discomfort and blood lactate during a submaximal heat stress test in hot conditions (~ 38°C, 30%RH) (Kelly et al., 2016). Core temperature, sweat rate and heart rate were unaffected by these low-volume short-term HA protocols, supporting the notion that HA strategies should include a minimal duration at which an athlete's core temperature is elevated (Houmard et al., 1990). However, caution should be warranted when attempting 'rapid' short-term HA strategies, as the protective effects (reduced core temperature and heart rate; increased sweat rate) that longer-term HA elicit appear to be lacking, and therefore these strategies may not sufficiently eliminate heat-induced performance impairments nor reduce an athlete's risk of EHI (Binkley et al., 2002; Guy et al., 2015).

The periodisation of HA poses complex questions around how best to schedule HA into an athlete's training and competition calendar, where multiple events held in hot environments are likely throughout a competitive season. While more evidence is needed around its application in highly-trained populations, current literature has shown that adaptations following HA appear to decay after 2-4 weeks depending on HA length (Garrett et al., 2009), while re-acclimation may occur with fewer sessions (Weller et al., 2007; Wyndham & Jacobs, 1957). As highly-trained individuals may have slower rates of HA decay (Pandolf et al., 1977), and because periodic exposure to heat following HA can allow for the retention of HA over several weeks (Moseley, 1997), it may be worth periodising HA intermittently throughout a competitive season. Furthermore, a 'thermal memory' or 'thermal plasticity' concept may exist, where rapid re-acclimation

is possible in those who have used HA routinely (Horowitz, 2001). These concepts were recently applied in a case study where HA decay was perhaps lessened following 2 weeks of HA in an elite sailor (Casadio et al., 2016). Indeed, 2 d of consecutive re-acclimation provided further thermoregulatory enhancement (Casadio et al., 2016), and is supported by similar findings in occupational settings (Weller et al., 2007; Wyndham & Jacobs, 1957). Figure 7.1 offers an example of how HA might be implemented in the build-up phase for athletes departing for a competition in hot conditions. Introducing HA 2-3 weeks prior to travel, combined with re-acclimation sessions before departure, poses an attractive strategy to 'top up' heat tolerance adaptations following a period of HA decay, and may alleviate training disruptions caused by repeating subsequent HA periods. More work is needed to understand how to optimise the periodisation of HA within an athlete's annual training plan.

Day	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	
Phase				HA							Decay							Decay							RA			
Sailing																												
Resistance																												Depart
Endurance																												
Heat	HRT	HA	HA	HA	HRT							HRT							HRT			RA	RA			HRT		
Training Load			Moderate-High								High							High							Recovery			
Duration (h)			19								20							22							15			

Figure 7.1 Overview of the periodised heat acclimation (HA) protocol used for 2 elite Laser sailors preparing for the World Championships in Oman. A 4-week mesocycle included 3 heavy-build weeks and 1 recovery week (TSS = training stress score). HA (35°C, 65% relative humidity (RH)) occurred during the first week with a heat response test (HRT) on day 1 and 5 of HA. HRTs were repeated during the two following weeks of decay, and 3 d post re-acclimation (RA). Reproduced from Chapter 3.

Concurrent training considerations

The training programme structure of an elite athlete is often complex and multifaceted, with multiple layers of stress applied at various times, altogether aiming to develop the physical and mental resiliency needed for peak performance. When heat stress is added to training, coaches and sport scientists must consider the impact that HA will have on the athlete's overall state of stress. For example, heat stress on top of normal training stress is likely to impact upon an athlete's overall sympathovagal balance (Dranitsin, 2008; Epstein et al., 2010; Sollers III, Sanford, Nabors-Oberg, Anderson, & Thayer, 2002; Yamamoto, Iwamoto, Inoue, & Harada, 2007) and resulting hypothalamic-pituitary-adrenal axis response (Brenner et al., 1997). While recommendations have been made to adjust overall training intensity, volume and recovery practices during periods of HA (Guy et al., 2015), detail is lacking on exactly how practitioners should do so. Horowitz (2001) explains that heat stress is a potent stimulus affecting every cell of the body (Horowitz, 2001), translating to heat-induced augmentations in sympathetic nervous system activity (Arngrímsson et al., 2003), cardiac strain (Périard et al., 2011) and rate of fuel utilisation (Edwards et al., 1972; King et al., 1985), thus resulting in an increased energy cost for a given exercise intensity (Wingo et al., 2005). Such added stress has been shown to not only amplify internal training load (Crowcroft et al., 2015), but could potentially impair an athlete's ability to recover for subsequent training sessions (Minett et al., 2015). This scenario provokes questions as to how concurrent training should be structured around HA, which sessions heat should be added to, and how subsequent routine training sessions should be adjusted based on prior heat stress.

It seems logical to use sport-specific exercise modes for HA sessions that provide opportunities for athletes to simulate competition in hot conditions to fine tune pacing and cooling strategies prior to competition (Taylor & Cotter, 2006). However, a number of authors speculate that low- to moderate-intensity sessions may be performed best in the heat during HA periods, and that so called 'key' training sessions of higher-intensity might be performed in cool conditions, to avoid reduced training quality that could potentially lead to a diminished high-intensity stimulus and associated peripheral adaptations over an extended HA period (Guy et al., 2015; Taylor & Cotter, 2006). Conversely, key sessions may warrant their place in the heat chamber in sports where there is an anaerobic or power component. For example, a recent comparison of low- (90 min at 40% maximal aerobic power output (P_{max})) versus high-intensity (5 x 3 min at 70% P_{max}, 3 min at 30% P_{max}) HA resulted in performance enhancement that was somewhat specific to the type of HA undertaken (Wingfield et al., 2016). Specifically, low-intensity HA resulted in improved 20-km TT endurance performance (5.9%), while high-intensity HA showed improvements in anaerobic performance (early sprint peak power output, vertical jump, counter-movement jump), without performance changes in the 20-km TT (Wingfield et al., 2016). As with normal variations in training intensity, perhaps a combination of high- and low-to-moderate-intensity training in the heat would elicit a wider range of performance benefits in the heat for sports with multiple performance determinants. In addition, introducing high-intensity HA sessions may be best placed following 2-3 d of HA once initial adaptations to the heat have occurred, thus supporting the maintenance of intensity in hot conditions.

Athletes undergoing HA could be training up to 2-3 times per day, yet an understanding of how previous and subsequent routine training sessions surrounding

HA may affect an athlete's response is largely unexplored and possibly overlooked by practitioners. Exercise-induced muscle damage, through high-volume eccentric training, has been shown to increase core temperature (0.2-0.3°C) during a subsequent exercise bout and may be explained by an augmented inflammatory response (Fortes et al., 2013; Montain, Latzka, & Sawka, 2000). However, this eccentric-training induced increase in heat strain is diminished with repeated bouts of eccentric training (repeated bout effect) (Dolci et al., 2015). Practically speaking, if eccentric training is introduced during a period of HA, heat strain may be elevated during the initial days. Interestingly, heat strain itself imparts a prophylactic effect against muscle damage (Nosaka, Muthalib, Lavender, & Laursen, 2007), so it might be used as a tool in endurance athletes, i.e. heat applied in the period before introduction to eccentric resistance training to reduce muscle soreness. In addition, low-intensity short-term HA (5 d; 90 min of cycling at 40% of power at $\dot{V}O_2$max) has been shown to reduce mean and peak torque during a maximal voluntary contraction (Wingfield et al., 2016). This finding supports the concept that endurance-based HA might impair training quality and adaptive responses during routine concurrent resistance training. In contrast, some have shown that applying heat to skeletal muscle, with or without resistance training, can augment maximal force and muscle cross sectional area (CSA) over a 10-week period in untrained men (Goto et al., 2011; Goto et al., 2007). Mechanistic studies have shown that heat stimulates the Akt/mammalian target of rapamycin (mTOR) signalling pathway, a key regulator of protein synthesis and hypertrophy (Kakigi et al., 2011; Yoshihara et al., 2013). Thus heat, applied in various ways, could provide an ergogenic effect when combined with resistance training. In summary, practitioners should consider the effects of HA on concurrent resistance training sessions. Introducing

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eccentric training during a period of HA should be avoided when the maintenance of a specific heat strain level during HA is required. Long-duration endurance sessions in the heat may impair the quality of concurrent resistance training. Finally, heat may be used as a training tool to incur protection against muscle damage or to augment muscle strength and hypertrophy adaptations.

7.5 Athlete Considerations

As no two athletes are the same, the high variation in individual responses to training (Borresen & Lambert, 2009; Kraemer et al., 1998; Plews et al., 2013) and environmental stress (Chapman et al., 1998; Racinais et al., 2012) is unsurprising. Understanding how each athlete responds to added heat stress and acclimation through repeat heat response testing is therefore key and should be conducted well before any critical event in hot conditions (Bergeron, Bahr, Bärtsch, et al., 2012; Racinais et al., 2012). Heat response tests can be sport-specific, be comprised of a steady-state effort, and/or include a performance measure. The protocol should complement normal training so it can be easily repeated, possibly multiple times a season, to assess an athlete's level of acclimation. Simple measures, such as heart rate, sweat rate, thermal perception and rating of perceived exertion can be easily employed without specialised equipment, while core temperature and plasma volume (or at least their change) can add further valuable information if available. High inter-individual variability in responses to heat response testing can be expected, even within similar athlete cohorts, which may be explained by several factors, summarised in Table 7.2. These include sex differences (Gagnon & Kenny, 2011, 2012; Mee et al., 2015), differences in ethnicity (Horowitz,

2014; Ladell, 1964; Lyashko et al., 1994; Taylor, 2014; Ulmasov, Shammakov, Karaev, & Evgen'ev, 1992), athlete type (Amano, Koga, Inoue, Nishiyasu, & Kondo, 2013), training status (Banfi et al., 2006; Cheung & McLellan, 1998; Pandolf et al., 1977; Piwonka, Robinson, Gay, & Manalis, 1965), anthropometric characteristics (Chung & Pin, 1996; Havenith, 2001; Havenith, Luttikholt, & Vrijkotte, 1995; Hayward, Eckerson, & Dawson, 1986; Selkirk & McLellan, 2001), previous HA (Casadio et al., 2016; Pandolf et al., 1977; Weller et al., 2007; Wyndham & Jacobs, 1957), history of EHI (Armstrong, De Luca, & Hubbard, 1990; Johnson et al., 2013), as well as sleep quality and duration (Armstrong et al., 1990). For female athletes, thermoregulatory changes due to menstrual cycle phase (Avellini, Kamon, & Krajewski, 1980; Carpenter & Nunneley, 1988; Inoue et al., 2005; Kolka & Stephenson, 1989) and oral contraceptive use (Armstrong et al., 2005; Stachenfeld, Silva, & Keefe, 2000; Tenaglia, McLellan, & Klentrou, 1999) could cause false-negative or positive responses to heat response testing following HA, and should be noted prior to any thermoregulatory assessment. In addition, practitioners working with female athletes should consider that HA induction may require moderate-term HA (Avellini et al., 1980; Frye, Kamon, & Webb, 1982), as short-term HA may be less effective in females (Sunderland et al., 2008) compared with males (Mee et al., 2015). Paralympic athletes require special considerations when training and competing in the heat, especially those with spinal cord injuries whose sudomotor cooling capacity is limited (Castle et al., 2013; Price & Campbell, 2002; Sawka, Latzka, & Pandolf, 1989). Finally, there is merit in discerning the immune status of an athlete prior to HA commencement. Individuals presenting a fever or upper respiratory tract infection are predisposed to EHI (Cooper, 1994; Sithinamsuwan et al., 2009) and should not perform HA sessions for risk of further

harm to themselves and their fellow athletes. Although HA does not appear to alter the

immune response in healthy individuals (Guy et al., 2015; Yamada et al., 2007), athletes

with suspected immune suppression may avoid further inflammatory exacerbation by

avoiding HA sessions.

Table 7.2 Possible factors contributing to individual athlete responses to heat response testing and acclimation.

Factor	Influence on heat response testing	Influence on HA
Sex	Females have a higher threshold for sweating onset and reduced sweating output compared to males (Gagnon 2011, 2012).	Females may require MTHA to achieve reductions in T_c and HR seen in males with STHA (Mee 2015).
Ethnicity	Individuals from hot climates may have greater HSP content and enhanced sudomotor function (Lyashko 1994, Taylor 2014, Ulmasov 1992).	Faster HA kinetics in individuals from hot climates (Kraemer 1998).
Athlete type	Enhanced evaporative cooling in endurance versus sprint athletes (Amano 2013).	Specific athlete type differences unknown, however, can relate to $\dot{V}O_2max$ (see below).
Training status	Higher $\dot{V}O_2max$ relates to greater HSP content and exercise heat tolerance (Banfi 2006, Cheung 1998, Pandolf 1977).	Higher $\dot{V}O_2max$ relates to faster rates of HA induction and greater heat tolerance following HA (Cheung 1998, Pandolf 1977).
Body size	Larger body size results in lower body surface area to body mass ratio, and higher heat strain in body mass dependent exercise (Havenith et al. 1995, 2001).	Not specified in literature.
Body composition	Higher adiposity and mesomorphy results in a faster rise in T_c (Chung 1996, Hayward 1986, Selkirk 2001).	Higher adiposity is directly related to $\dot{V}O_2max$ (see Athlete type) (Cheung 1998).
HA history	Reduced heat strain in subsequent HRT with ~2-4 w of prior HA (Casadio 2016, Garrett 2009, Pandolf 1977, Weller 2007, Wyndham 1957).	HA may occur more rapidly with 2-4 sessions (Casadio 2016, Weller 2007, Wyndham 1957).
EHI history	Possible early test termination and higher end T_c (Armstrong 1990).	HA is possible; some may require repeated HA interventions until enhanced heat tolerance is shown (Armstrong 1990, Johnson 2013).
Menstrual cycle	Tc reduction (0.1-0.2°C) and increase (0.2-0.5°C) prior to and following ovulation, respectively (Carpenter 1988, Kolka 1989, Stachenfeld 2000).	Females can heat acclimate, regardless of menstrual cycle phase, following MTHA. STHA may be less effective (Mee 2015, Avellini 1980, Frye 1982, Sunderland 2008).
Oral contraceptives	Combined pill use causes higher T_c (0.5°C) and cyclical fluctuations may be dampened. Progesterone-only pills reduce T_c (0.6-0.7°C) (Stachenfeld 2000, Tenaglia 1999).	Oral contraceptive users can acclimate similarly to non-users (Armstrong 2005).
Spinal cord injury	Inability to sweat below the site of injury (Sawka 1989); reduced exercise intensity for a given rise in T_c with greater lower body heat storage (Price 2002).	Partial HA through reduced T_c and PV expansion, without changes in sweat rate (Castle 2013).
Immune status	Fever or URTIs may increase EHI risk (Sithinamsuwan 2009, Cooper 1994).	HA may not change immune markers but increases protection from EHI (Yamada 2007).

EHI = exertional heat injury, HA = heat acclimation, HR = heart rate, HRT = heat response test, HSP = heat shock protein, MTHA = moderate-term HA, STHA = short-term HA, T_c = core temperature, URTI = upper respiratory tract infection, $\dot{V}O_2max$ = maximal oxygen consumption, w = week.

7.6 Perspectives

The purpose of this current opinion piece was to address practical considerations for integrating HA within an athlete's programme and stimulate new research. When considering an HA approach several factors need to be reviewed in order to determine the appropriate heat load (temperature and humidity), mode, intensity, duration, frequency and periodization of adding heat to the existing training plan (Figure 7.2). Often the priorities of training and the confines of an athlete or team's schedule and location outweigh the ability to implement best practice HA. Advancing research offers potential solutions to overcome common barriers to HA. Alternatives to conventional HA through post-exercise hot water immersion and sauna bathing add little disruption to normal training and may be used to induce HA adaptations if sport-specific training cannot be performed in a hot environment. While some form of heat exposure prior to competition in the heat would be better than none at all, caution is nevertheless warranted when using alternative forms of HA, and thermoregulatory markers should be monitored to measure heat adaptation progress. Before the length and intensity of HA are chosen, practitioners should consider the event duration and demands and be aware that short-term HA may not induce the thermoregulatory and performance enhancement that longer-term HA provides. Implementing a re-acclimation strategy prior to an event in hot conditions may offer flexibility for athletes and their schedules when other key elements of training are more crucial. When adjusting routine training, the added stress imposed by HA should be considered, and the volume and intensity of other routine training in normal conditions may need to be reduced based on physiological and subjective athlete feedback during a heat camp. Finally, understanding individual

athlete responses to heat stress and HA will assist practitioners to tailor protocols to their individual needs.
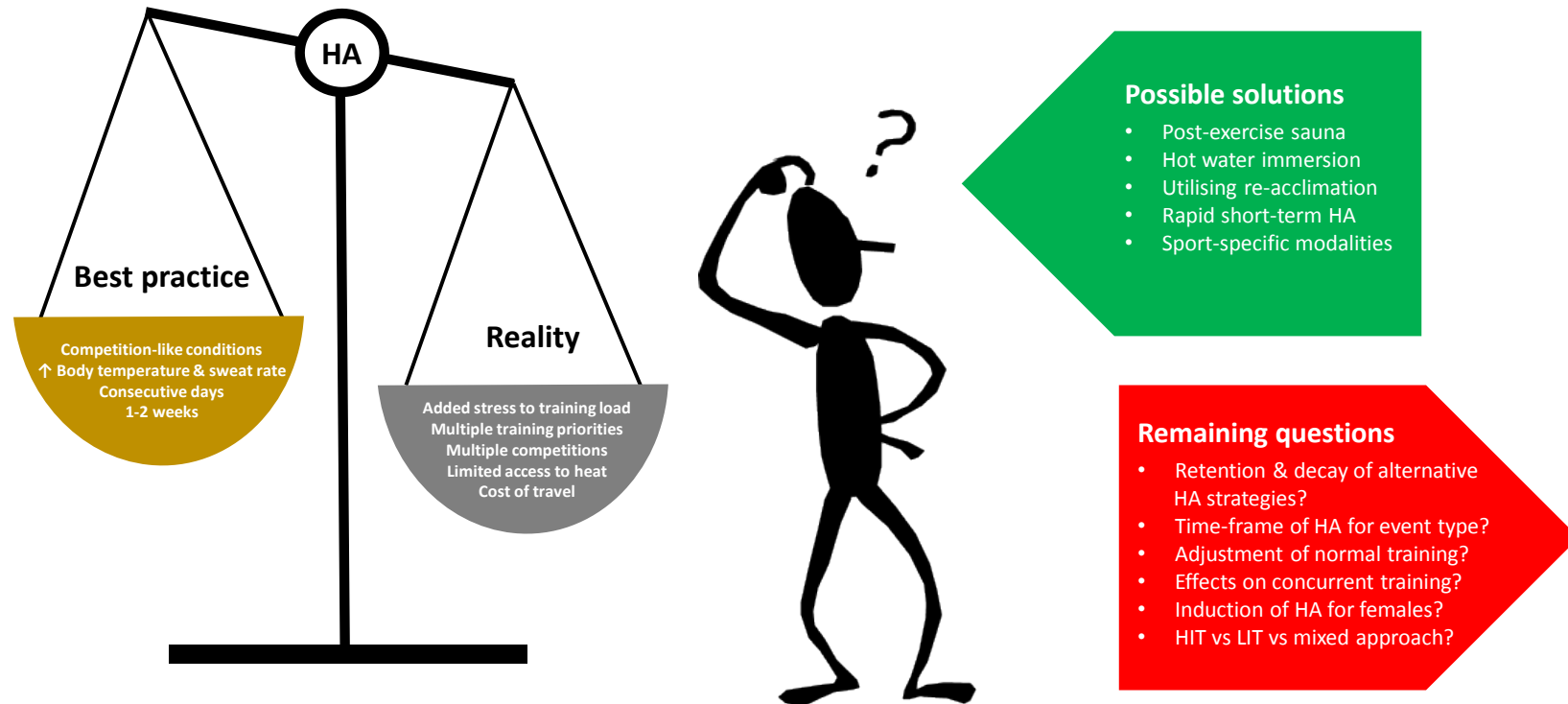
Figure 7.2 Integrating best practice heat acclimation (HA) recommendations within the constraints and complexities of an elite athlete's training and competition schedule. HIT = high-intensity training, LIT = low-intensity training, ↑ = increased.

7.7 Conclusion

Heat strain associated with exercise in hot conditions has a negative impact on exercise performance. HA improves thermoregulation, cardiovascular stability and attenuates heat-induced performance impairments. While the present HA guidelines provide a sound starting point for practitioners working with athletes, new research aimed at reducing the limitations and barriers of translating evidence-based guidelines to the real world of elite sport is needed. Alternative HA methods may be beneficial and easier to apply with athletes. An understanding of how HA may affect established training load would assist practitioners towards optimising the implementation of HA within the complex intricacies of an athlete's training programme. Finally, the unique individual athlete response to HA should be considered when attempting to achieve optimal performance outcomes in hot condition events.

CHAPTER EIGHT: HEAT ACCLIMATION AND PERFORMANCE IN THE ELITE: CONSIDERING THE INDIVIDUAL

8.1 Invited Comment: Journal of Physiology

In this CrossTalk debate, Nybo and Lundby (2015) dismiss the concept that the response to heat acclimation may be highly individual; that is, that there could be responders and non-responders. From our point of view and experience within the Olympic athlete environment, this assumption is an oversight. Indeed, the individual response to any form of planned training cannot be overlooked, especially when considering the narrow-margins of importance within the world of elite sport. The conclusion that HA does not enhance performance in temperate conditions may be supported by comparisons of the mean response in some studies (Nybo & Lundby, 2015), yet when individual responses are presented, clear responders and non-responders emerge (Neal et al., 2015; Racinais et al., 2012). For example, Racinais et al., (2012) reported large inter-individual differences in soccer players' response to HA (Δ plasma volume: -10 to +20%) and their ability to perform in the heat following HA (total distance run: -6 ± 6%; high-intensity running: -16 ± 21 %) (Racinais et al., 2012). As well, a closer look at the individual data presented by Neal et al., (Neal et al., 2015) reveal that ~40% of cyclists improved their VO_2max (+5-11%) and time trial power output (+3-5%) (Neal et al., 2015). As the list of physiological adaptations to HA with ergogenic potential is long (Corbett et al., 2014), more work is needed to understand the causal effects of HA-induced temperate performance enhancement in those athletes who do 'respond'. While caution is warranted in assessing true individual effects, repeating performance measures along with reporting of the measurement error and smallest worthwhile change, can assist to decipher uncertainties between normal within-subject variation 'noise', and meaningful important responses (Hopkins, 2015).

CHAPTER NINE: OVERALL DISCUSSION & CONCLUSION

9.1 Summary of findings

The use of training in the heat to enhance performance has become a 'hot topic' in the world of elite sport given that a number of high profile events are held in hot environments, and emerging evidence suggests that training in the heat has the potential to be ergogenic (Corbett et al., 2014). Indeed, short-term heat acclimation (HA) indoors (or heat acclimatisation outdoors), is a commonly used strategy to prepare athletes for competition in hot environments and typically involves 4-7 days of aerobic training in artificially hot conditions (30-40°C) (Garrett, Rehrer, et al., 2011). The physiological adaptations seen with HA include decreased resting and exercise core temperatures, lowered skin temperature, increased plasma volume and sweat rate and a decreased heart rate for a given workload, all of which attenuate performance impairments that are caused by hot conditions in the unacclimated state (Armstrong & Maresh, 1991). Heat exposure may increase endurance performance in temperate conditions (Buchheit et al., 2011; Lorenzo et al., 2010; Scoon et al., 2007), enhance muscle strength and hypertrophy (Goto et al., 2011; Goto et al., 2007), and has been shown to enhance sprint performance acutely (Ball et al., 1999; Girard et al., 2013; Sargeant, 1987). As every 0.1% improvement can make a difference in terms of podium finishes in elite sport (Hopkins et al., 1999), it could be advantageous to prescribe heat training protocols at strategically beneficial times throughout a competitive season.

The majority of research used to establish our understanding of HA to date has been completed in untrained or moderately-trained individuals, with little work completed in well trained or elite athletes, which may have partial heat-related adaptation already due to their well-trained status (Cheung & McLellan, 1998).

Furthermore, investigation into how best to practically integrate HA into an elite athlete's already busy training schedule, without interfering with key training sessions and competitions, was needed. Most HA studies tend to target performance measures in long-duration, endurance exercise, and a greater understanding of how HA affects sprint performance is needed. Last, practical HA solutions are required for highly-trained athletes who do not always have accessibility to a climate chamber or hot environments. Therefore, the overarching aim of this thesis was to optimise the use of heat, through HA or heat training, to enhance physical performance and develop practical strategies that could be easily incorporated into the training programmes of elite athletes. The findings of this thesis informed New Zealand National Sports Organisations, with support from High Performance Sport New Zealand (HPSNZ), on heat and HA strategies for their respective Rio Games' campaigns.

Given the limitations found in literature, and with partnership with research questions from sports funded by HPSNZ, this thesis set out to answer the following questions, using the studies presented in Chapters 3-8 of this thesis. The questions included:

Chapter 3: Can HA be periodised into an elite athlete's training schedule using a short-term HA protocol followed by a brief period of re-acclimation?

Chapter 4: What are the effects of heat training on sprint performance and thermoregulation, and can heat training induce beneficial cardiovascular adaptations in elite sprint track cyclists?

Chapter 5: What are the effects of acute heat stress on thermoregulation and sprint performance in highly-trained BMX cyclists? Given the heat stress imposed from BMX-type training in the heat, what are the effect of sprint and strength-based HA on performance and thermo-physiological measures?

Chapter 6: If heat training for power athletes has ergogenic potential (Chapters 4 and 5) what are the acute effects of resistance exercise performed in hot versus temperate conditions on neuromuscular function, hormone responses and thermal strain in highly-trained female and male power athletes?

Chapter 7: Current best practice guidelines for HA are often not entirely attainable within the confines of highly demanding physical preparation and travel of an elite athlete. What alternative methods of HA are effective for athletes and how might HA be integrated into an athlete's existing training program? In addition, what are some of the specific athlete considerations that practitioners should account for?

Chapter 8: If the mean response of a group following HA does not show performance improvements in temperate conditions, does this infer that HA should be discounted as an ergogenic tool for all individual athletes?

The following section discusses the findings from these chapters and how they interrelate.

Chapter 3: Periodised heat acclimation

In the first study (Chapter 3), I examined the retention of HA adaptations and re-acclimation responses during a periodised short-term HA protocol in elite sailors preparing for the 2013 Laser World Championships in Muscat, Oman (~27-30°C, 40-60% RH). The study involved the observation of responses over a periodised 4-week training mesocycle, which included 3 heavy-load training weeks followed by 1 recovery week. The HA and re-acclimation protocols were incorporated into the first and fourth week of the mesocycle respectively, with the aim of having the athletes prepared for competing in hot conditions, while reducing possible heat-induced fatigue prior to overseas long-haul travel. It was shown that short-term HA decreased heat strain in two elite Laser sailors through decreased rectal temperature, heart rate, RPE and thermal discomfort (Figure 3.2). Most thermoregulatory adaptations were retained over the 2 subsequent weeks, without HA, except for rectal temperature in one sailor. Previous research supports this time-course of HA retention, with evidence to suggest that about 2-weeks may be the period in which adaptations begin to reverse (Pandolf et al., 1977; Weller et al., 2007). This study was the first to show that additional re-acclimation sessions further thermoregulatory enhancements in an applied context with elite athletes. These findings are in line with others examining re-acclimation in occupational settings, showing that 1-4 d of re-acclimation may be used to 'top up' heat tolerance adaptations after a period of decay from HA (Weller et al., 2007).

Also in this study, I showed comparable rises in both PV and submaximal power output in the heat over the 4-week training block, which was a greater improvement than what is normally seen in these athletes during a focused sailing block, where

aerobic gains are not expected. While the relationship between PV expansion and aerobic performance in the literature is unclear (Corbett et al., 2014) and the potential for supercompensation following heavy-build weeks followed by a taper (Fry et al., 1992), the improvement in submaximal fitness highlights the potential ergogenic 'boost' that frequent HA sessions could elicit throughout an athlete's season (Chalmers et al., 2014).

Chapter 4: Heat training for sprint performance

The second study (Chapter 4), I changed focus from examining HA applications for endurance events, and shifted towards investigating heat training for sprint athletes. This study arose from discussions with Cycling New Zealand staff following previous successful performances that were shown by the men's sprint track team at World Cup events held in hot environments. These performances were also coupled with higher than normal inertial peak power outputs and optimal cadence values. To better understand how the men's team responded to the heat, an initial 5 d heat training camp was conducted in May 2013, in which 3 of the men's elite sprint track cycling squad performed their sprint-specific training sessions in hot conditions (35°C, 60%RH). The cyclists showed varied responses in inertial sprint performance (peak power output = -12.2 to +9.0%; smallest worthwhile change = 2.2%) during heat training, compared with temperate conditions. What was somewhat surprising was that sessions in the heat, where minimal work was performed (Warm up: 10 min passive heating, 10 min of low-intensity cycling; Main set: 1-3 reps of 2-3 x 20-30 s sprints, 10 min recovery interval), elicited signs of HA adaptation, including reduced exercising core temperature. While there may not be enough research to define the minimum dose of HA required to elicit

core temperature reductions, the current guidelines recommend that HA sessions last at least 60 min per day, as to induce increases in body temperature and sweat rate (Racinais et al., 2015). In addition, there were meaningful PV increases (~4%) in the 2 cyclists that showed improved sprint performance in the heat, whereas the cyclist with a seemingly heat-induced performance impairment (-12.2% decrease in inertial peak power output) showed a negative plasma volume response (-2%). Following this finding, Cycling New Zealand staff were eager to examine the performance and physiological effects of even more sport-specific heat training by conducting their resistance training sessions in the heat. We also wondered whether the PV expansion finding seen in some of the cyclists through heat training would translate to aerobic fitness adaptations, which may support heavy training volume and enhance recovery between maximal sprint efforts (Tomlin & Wenger, 2001). Based on these preliminary findings and questions, the next heat training camp (Chapter 4) was scheduled for September 2013.

In Chapter 4, I examined the effects of a sprint cycling-specific heat training camp on sprint performance in temperate conditions, as well as assessed thermoregulatory and cardiovascular responses in 5 male elite sprint track cyclists. This training camp consisted of 5 days of sprint-interval training and resistance training in hot conditions ($30\text{-}35^{\circ}\text{C}$, 40-60% RH), with sprint performance and cardiovascular responses to heat training assessed in temperate conditions ($\sim20^{\circ}\text{C}$, 50% RH). The main finding was that sprint performance, as measured through inertial sprint peak power output and optimal cadence, decreased immediately following 5 days of heat training. It may be that previously shown ergogenic effects of heat for sprinting rely on acute increases in muscle temperature (Ball et al., 1999; Sargeant, 1987) and associated

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muscle contractility enhancement (Farina et al., 2005; Gray et al., 2006), rather than short-term induced adaptations. Previous work has shown that long-term (40-h over 10 weeks) heat exposure, which sustains elevated muscle temperature (38.5-39.0°C), has direct effects on skeletal muscle adaptation and performance (Goto et al., 2011; Goto et al., 2007). Therefore, while the cyclists perceived that training quality was enhanced during sessions in the heat, short-term heat training did not appear to be a beneficial strategy for increasing peak power output in temperate conditions in this study.

In addition, sprint-specific heat training induced signs of HA through reduced exercising core temperature as well as enhanced cardiovascular function. A reduction in exercising core temperature on the 5th day of heat training (Figure 4.4) is supported by similar findings following high-intensity intermittent running in hot conditions (Sunderland et al., 2008). These results suggest that the use of combined sprint- and resistance training in the heat could act as an alternative to traditional steady-state exercise HA protocols, thus allowing for more sport-specific training and less training interference.

Last, this study revealed enhanced cardiovascular function, as shown through PV expansion and associated improvements in measured heart rate indices during submaximal exercise in temperate conditions. While others have found similar improvements in PV (Garrett et al., 2009; Garrett et al., 2014; Racinais et al., 2012) and heart rate indices (Buchheit et al., 2011) following short-term HA, this study differs from others in that little-to-no endurance type exercise was performed during each training session and that the cardiovascular adaptations shown were likely heat-, rather than endurance-training induced. While improved aerobic fitness is associated with

faster lactate removal and phosphocreatine resynthesis following maximal sprint bouts (Tomlin & Wenger, 2001), this was not directly measured in the present study and therefore we can only speculate that heat-induced cardiovascular adaptations may be beneficial for metabolic recovery in sprint track cyclists.

Chapter 5: Heat acclimation for sprint performance

Conveniently adding to the next study in my thesis, the New Zealand BMX team had been eager to investigate issues they were faced when competing overseas in hot conditions. Specifically, some athletes were finding training and competing in the heat uncomfortable and challenging. Given that sprint and resistance training in the heat (Chapter 4) induced partial HA adaptations, it was decided to run a sport-specific HA camp with the BMX team (Chapter 5). The first part of this study examined the effects of acute heat stress on BMX sprint performance compared with temperate conditions in the elite squad. Briefly, low-intensity cycling, followed by a repeated sprint test was performed in temperate (20°C, 50% RH) and hot (35°C, 50% RH), then again in the hot condition following 5 days of BMX-specific HA, to assess the effects of heat and HA respectively on BMX sprint performance. Acute heat exposure increased peak power output during the 2nd (9%) and 3rd (15%) sets of the repeated sprint protocol, compared with the control trial (Figure 5.2). These results are in line with others showing that heat-induced improvements in single sprint (11-25%) (Ball et al., 1999; Sargeant, 1987) and repeated sprint performance (3-8%) (Falk, Radom-lsaac, et al., 1998; Girard et al., 2013) in the absence of hyperthermia (core temperature >39°C) occur (Drust et al., 2005), and suggests that heat itself could enhance the stimulus of sprint training.

From a physiological standpoint, hot conditions induced greater thermal strain through increases in skin temperature, sweat rate, and heart rate, as well as thermal perception (Table 5.1), compared with control conditions. These results compare with others showing that repeated sprint efforts in the heat can induce physiological alterations that enhance the capacity for heat dissipation (Castle et al., 2011; Girard et al., 2013). Although the effects of heat on mean exercising core temperature were trivial, the range of end exercise values tended to be higher for the individual cyclists in the hot (38.5-38.6°C) versus control (38.6-38.9°C) conditions (Figure 5.4). Given that real world training and competition time-frames are often longer than the 60-min period examined in this study, it is possible that BMX cyclists could be subject to hyperthermia-induced impairments in sprint performance (Drust et al., 2005) and warrants the recommendation of HA or cooling strategies for this population of athletes prior to competing in hot conditions.

The second aim of this study was to determine the effects of HA, on thermoregulatory factors and repeated sprint performance in the heat. The heat training protocol employed closely resembled that described in Chapter 4, and consisted of three strength- and power-based resistance training sessions and two sprint-interval training sessions in the heat. The main difference between the HA protocols of Chapter 4 and 5 were that the BMX cyclists may have sustained a higher thermal load by wearing full protective clothing and helmets (Armstrong et al., 2010) during the sprint-training and heat stress testing trials. In addition, two extra heat training sessions were performed compared with the protocol in study 2. Despite the increase in both magnitude and days of heat exposure, only subtle adaptations to hot conditions were shown through decreases in thermal discomfort (Figure 5.4), with trivial effects shown for core

temperature, skin temperature, heart rate, sweat rate and thermal sensation following HA (Table 5.1). Although the modality of HA in this study was unique, the results align with other studies showing how short-term HA (4-7 d) has a minimal effect on skin and core temperature during exercise (Petersen et al., 2010; Sunderland et al., 2008). It is possible that the anaerobic nature of the training and low volume of work performed diminished the core temperature rise during HA sessions and was not sufficient to elicit further thermoregulatory adjustments typically reported with traditional endurance-based HA (Horowitz, 2001). This serves as an important reminder for practitioners that HA adaptations, which are key for reducing the risk of heat-related injury (Binkley et al., 2002), cannot be rushed, and may in fact be dependent on the time spent exercising at a particular core temperature range in the heat (Houmard et al., 1990). Others have also shown that short-term, sport-specific HA protocols fail to reduce core temperature (Petersen et al., 2010; Sunderland et al., 2008), which suggests that other methods of increasing heat strain within a session (clamped core temperature technique) or following (post-exercise sauna) may be required to achieve a sufficient level of HA through heated resistance training.

While HA adaptations were minimal, sprint performance increased a further 8% following HA (Figure 5.2) and is supported by previous research showing improved sprint performance (~2%) following moderate-term HA (Castle et al., 2011). Conversely, single sprint performance was impaired in temperate conditions (Chapter 4) 24 h following heat training in the sprint track cyclists. The altered HA protocol in Chapter 5 may account for these different findings, as 6 d of consecutive heat exposures were performed, followed by 48 h without heat with an emphasis on recovery, whereas in chapter four, 4 days of heat sessions were performed with the final heat stress test

conducted on the 5th day. As the BMX athletes in Chapter 5 had ~48 h between HA and the final heat stress test, this may have enhanced recovery from potential neuromuscular fatigue accumulation following consecutive days of training in the heat. This is supported by the rebound in sprint performance shown by 2 of 3 sprint track cyclists at 72 h versus 24 h post after 4 days of heat training (Figure 4.2). Given the meaningful increase in sprint performance in the heat, this study shows that HA is a worthwhile training strategy for elite BMX cyclists.

Chapter 6: Heated resistance exercise

From the findings in Chapters 4 and 5, it was decided that the concept of resistance training in the heat required further understanding. Strength- and power-based resistance training in the heat was believed to improve training quality and induce partial HA adaptations (Chapter 4), and may have been instrumental in enhancing sprint performance in the heat (Chapter 5). Therefore, Chapter 6 set out to 1) understand the level of thermal strain that occurred in power athletes performing strength- and power-based resistance exercise in the heat, and 2) determine the acute effects of heated resistance exercise on strength and power compared with normal (temperate) conditions. Performing strength and power exercises in the heat were shown to increase thermal strain through augmented skin temperature and thermal perception, while core temperature and heart rate were not affected. The lack of change in core temperature shown in the hot condition shed light into why previous studies of this thesis only showed minimal (Chapter 5) or partial (Chapter 4) HA adaptations. For heated resistance training to be used as an HA tool, different exercise protocols, such as hypertrophy, strength-endurance or circuit-type training where the training volume to

rest interval ratio is greater (Stone et al., 1982), might be better suited for augmenting thermal strain to a level that will induce desired thermoregulatory adjustments.

Although core temperature was unaffected during heated resistance exercise, I showed a beneficial effect of heat on power production, both during and following the resistance exercise session in highly-trained power athletes (Figure 6.1). Although power increases were not shown in both power tests (vertical jump and CMJ) across sexes, the increases shown are in agreeance with others who have shown the ergogenic effect of heat on power production (Ball et al., 1999; Girard et al., 2013; Hedley et al., 2002; Sargeant, 1987) and might be explained by transient increases in muscle temperature leading to increased muscle fibre conduction velocity and a greater utilisation of anaerobic metabolism (Gray et al., 2006). These results are further supported by an increase in CMJ peak power following an acute heated resistance exercise session with the sprint track cycling team (Chapter 4). Moreover, subjective session quality was higher (5-7%) in hot conditions (Table 6.2), although not meaningful, and again is in line with the feedback given from the sprint track cyclists (Chapter 4). These findings along with that of the augmented anabolic hormone responses shown (Figure 6.4), suggest that heated resistance exercise may enhance the training stimulus during sport-specific power exercise and perhaps be used as an ergogenic strategy during targeted training blocks for power athletes.

Chapter 7: Considerations for elite athletes

In addition to the learnings derived from studies in Chapters 3-6, I also facilitated several HA or heat training projects with other sports including Rugby Sevens, Paralympic Shooting and ultra-marathon running, which are not described in this thesis.

Chapter Nine

Through my collective experiences working in applied high-performance sport, it became evident that many of the recommendations that originated from controlled laboratory-based studies would not translate into practice when working with elite athletes and teams in real world circumstances. The need to "think outside the box" became essential in order to design HA protocols that maximised thermoregulatory adaptations while minimising interference with other key training objectives. As a result, the current opinion piece (Chapter 7) aimed to review HA considerations when working with elite athletes. First from this paper, HA protocol design challenges may include limited access to environmental chambers, constrained training modes not attainable within the small confines of most environmental chambers, high costs associated with international travel to conduct heat camps, and potential interference that HA might have with higher-prioritised training phase objectives. Table 7.1 reviews literature examining alternative HA protocols such as post-exercise sauna bathing and hot water immersion with evidence that the desired HA adaptations occur, and thus provides alternative options when traditional exercise-based HA protocols are not possible. Other potential alternatives, although not mentioned in the current opinion, include resistance training in the heat as described in Chapters 4-6.

Second, I explored the literature to understand how HA might be integrated into an athlete's existing training program while considering timing, intensity, training volume adjustments and concurrent training sessions. Literature clearly suggests that heat is a powerful stimulus and that the added thermal stress can increase training load (Crowcroft et al., 2015) and potentially impair an athletes ability to recover (Minett et al., 2015). Therefore, the timing of HA implementation may be key. As shown in Chapter 3, introducing HA 2-3 weeks prior to travel, combined with re-acclimation

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sessions before departure, poses an attractive strategy to 'top up' heat tolerance adaptations following a period of HA decay, and may alleviate training disruptions caused by repeating subsequent HA periods. In terms of training intensity and volume adjustment, as with normal variations in training intensity, perhaps a combination of high- and low-to-moderate-intensity training in the heat would elicit a wider range of performance benefits in the heat for sports with multiple performance determinants. In addition, introducing high-intensity HA sessions may be best placed following 2-3 d of HA once initial adaptations to the heat have occurred, thus supporting the maintenance of intensity in hot conditions. Finally, when considering the quality of concurrent resistance training, introducing eccentric training during a period of HA should be avoided when the maintenance of a specific heat strain level during HA is required. Indeed, long-duration endurance sessions in the heat may impair the quality of concurrent resistance training (Wingfield et al., 2016), and heat may be used as a training tool to incur protection against muscle damage (Nosaka et al., 2007) or to augment muscle strength and hypertrophy adaptations (Goto et al., 2011; Goto et al., 2007). Figure 7.2 summarises the balance between best practice HA and the reality that coaches and sports scientists must consider before implementing an HA protocol. Possible solutions for practitioners include alternative forms of HA (post-exercise sauna and hot water immersion), the use of re-acclimation, rapid short-term HA, and more sport-specific modalities such as resistance training in the heat (Chapters 4-6).

Last, several unique athlete considerations, including their history and physical characteristics, should be understood by practitioners before implementing HA protocols, as individual characteristics often elicit different heat stress and HA responses. Possible factors contributing to individual athlete responses to heat response

testing and HA are described in Table 7.2, which may also serve as a guide for practitioners to consider when working with athletes in the heat. Included in these factors are sex differences to heat and HA, as well as considerations with respect to menstrual cycle and oral contraceptive use in female athletes. Sex differences to acute heat stress and HA were apparent from the studies in this thesis. In Chapter 5, female BMX cyclists showed no change in heart rate or core temperature, while males demonstrated some reductions post-HA (Table 5.2.). This could have been due to the menstrual cycle phase or to the fact that HA adaptations in females may require moderate-term HA (Mee et al., 2015). Chapter 6 showed reduced sudomotor function for female athletes in the heat, compared with males (Figure 6.1), as well as maximal strength increases following the heated resistance exercise session; a trivial effect was shown in the male athletes (Figure 6.2). Such findings highlight the importance of considering unique athlete characteristics, including sex, when aiming to optimise HA.

Chapter 8: Considering the individual

The final piece to this thesis included an invited comment (Chapter 8) to a Cross Talk Debate in *The Journal of Physiology.* I was asked my opinion as to whether existing evidence supports the presumption that HA itself has the capacity to enhance endurance performance in temperate conditions (Nybo & Lundby, 2015). Nybo and Lundby (2015) took the stance against this presumption. However, their comment failed to address the fact that HA and its multiple effects are highly individual; that is, that there could be responders and non-responders. From my point of view and experience within the Olympic athlete environment, this assumption was an oversight. In fact, when one looks beyond mean results, the story of clear positive, non and negative

responders emerge (Neal et al., 2015; Racinais et al., 2012). Such effects cannot be overlooked, especially when considering the narrow-margins of importance within the world of elite sport (Hopkins et al., 1999). Variable responses to heat were clearly evident throughout this thesis. Chapter 4 showed various responses to heat on CMJ power during resistance training (Figure 4.3) and sprint performance following heat training (Figure 4.2). In Chapter 5, three of seven athletes showed increases (3.5-5.8%) in inertial sprint peak power output that crossed the smallest worthwhile effect bandwidth (3.4%), including athlete personal best performances, while 2 athletes showed impairments (-3.8%, -4.9%) in inertial peak power in the Post HA condition (Figure 5.3). Given these results were compared with athlete norms, the coach viewed such responses as substantial. This assisted us further to identify athletes who may respond positively to sprint training in the heat acutely and others who may be susceptible to increased neuromuscular fatigue following HA. Finally, Chapter 6 showed high variability in individual athlete response in thermoregulatory (Figure 6.1), performance (Figure 6.2), and hormonal responses (Figure 6.4) to resistance exercise in the heat. Interestingly, one male athlete, whose performance was reduced in the heat (CMJ peak power = -12%), also showed reductions in anabolic hormone response (testosterone = -11%, GH = -26%). In contrast, another male athlete appeared to respond positively to the heat through increases in performance (CMJ peak power = 6%) and hormone response (testosterone = 48%, GH = 100%). Discussion with both athletes and their coach were also in alignment, with beliefs that athletes either struggled or performed better in the heat, respectively. Such examples support the concept that monitoring physiological markers such as plasma hormones, may assist to tailor training to enhance individual adaptation (Beaven, Cook, et al., 2008; Beaven,

Gill, et al., 2008). Taken together, these examples of individual responses highlight to coaches and practitioners the importance of understanding unique athlete characteristics and responses, rather than assuming that all athletes follow the mean response.

9.2   *Limitations of research*

Consideration of the studies and interpreted outcomes within this thesis requires attention to the following limitations:

i.   Although the aim of this thesis was to gain an understanding of how to optimise the use of heat to enhance performance, not all of the studies involved clear performance measures that were ecologically valid and that truly represented the sport-specific event demands (i.e., sailing, sprint track cycling, BMX racing). There are several constraints imposed when working with elite athlete populations, and in the case of the studies presented within, I did my best working alongside coaches to find logistical solutions that would help work towards answering my research questions without disturbing other important training objectives within the athlete's programmes (i.e. athletes not able to perform multiple maximal effort performance tests in one week due to other key training sessions; rigid travel schedules; reluctance to change multiple aspects of the athletes routine training, etc.).

ii.   Several of the studies included small sample sizes, which can increase uncertainty of effects due to sample variation (Atkinson, 2006). Although study sample sizes were indeed small in Chapters 3-5 (n = 2-7), these studies included the entire elite population of each respective sport within the New Zealand

national programmes. In this case, the results could be viewed as having come from the entire population rather than a sample of that population.

iii. The timing of heat as a stressor for studies involving female athletes presents another limitation to the present thesis, in that we were not always able to control for menstrual cycle phase. When thermoregulatory changes are assessed in females, trials should ideally be performed during the same menstrual cycle phase (follicular or luteal), rather than across the two phases, in order to avoid cyclical fluctuations in core temperature and thermoregulatory function (Carpenter & Nunneley, 1988; Stachenfeld et al., 2000). For the BMX HA camp (Chapter 5), there was only a single week of opportunity where the coach could bring all the National Team athletes to Auckland for testing and training; thus, all performance and thermoregulatory trials may not have been performed in the same phase. In Chapter 6 (heated resistance exercise), 7 of the female athletes completed both trials within the same phase (n = 6 follicular phase; n =1 luteal phase), while one athlete crossed phases (n = 1 luteal then menses). Furthermore, 4 females (non-oral contraceptive users) performed both trials during the follicular phase, where strength levels have been shown to be up to ~10% greater due to enhanced oestrogen and testosterone levels consistent with that phase (Phillips et al., 1996; Sarwar, Niclos, & Rutherford, 1996). Of these 4 females, only 1 showed an increase in peak force, while those taking oral contraceptives, who are not expected to have cyclic changes in strength (Phillips et al., 1996), did improve their peak force. Therefore, menstrual cycle changes cannot explain the female's improved force production in the hot compared with the temperate training conditions in Chapter 6.

iv. This thesis could be criticised for the lack of control groups in Chapters 3, 4 and 5. As previously mentioned, these studies included the entire elite population of athletes for their given event in the country. As there are multiple training requirements, the athletes are always progressing forward on a periodised plan. Thus, so called washout periods, or repeating protocols in control conditions is typically impractical for high performance athletes performing regular training in various forms to maintain a high chronic training load. Enforcing such procedures typically found within 'classic' thermophysiology research literature would be a detriment to the athlete's overall training objectives, and therefore unlikely to be attainable.

v. Although the thesis aimed to consider the effects of heat training in elite athletes, in Chapter 6, some of the athletes were only highly-trained and competing at national rather than the international level. This was deemed worthy to ensure that the male and female groups in this study were represented by adequate sample sizes. However, in terms of thermoregulatory effects to heat stress, the difference between highly-trained versus elite athletes appears minimal (Garrett et al., 2012).

9.3 *Practical Application and Significance*

The findings of this thesis have substantial practical application for those looking to integrate HA or heat training into an elite athlete's programme to enhance performance. Figure 9.1 details relevant HA protocol considerations for practitioners working with elite athletes. Based on the learnings herein, the following section offers an application guide for alternative and novel heat strategies in highly-trained and elite athletes, which was the overarching aim of this industry-based PhD. Several of these strategies were used by athletes from various sports preparing for the Rio Olympic Games, which were projected to be hot (~23-35°C, 40-70% relative humidity (RH)) compared with New Zealand (~13°C, 70% RH) (Casadio, 2013). An environmental report is found in Appendix J, which was conducted by myself and formed the basis of recommendations for National Sports Organisations preparing for the Rio Games.

Figure 9.1 Heat acclimation (HA) protocol considerations when working with elite athletes.

Periodising heat acclimation

Using the re-acclimation strategy outlined in Chapter 3, it was shown that HA can still be effectively implemented if you plan appropriately. In this example, I scheduled the heat sessions back approximately 3 weeks before departing for competition in a hot environment, and then implemented 'top up' re-acclimation sessions in amongst normal elite laser sailors training requirements. As adding environmental stress to training is an additive physiological stressor, and could cause an increase in internal training load, potentially leading to overreaching (Crowcroft et al., 2015; Taylor & Cotter, 2006), the approach shown in this thesis could allow athletes and coaches to periodise the timing of HA so as to reduce interference with more critical training goals. Specifically, I suggest that implementing HA 2-3 weeks prior to competing in a hot environment, along with 2 additional RA sessions (as just one example), may preserve precompetition taper quality while restoring HA adaptations, thus serving as an alternative and possibly more appropriate HA strategy for elite athletes. Prior to using a re-acclimation strategy in the build-up towards a major competition, it is important to understand individual athlete responses within a sport or team, as re-acclimation responses may vary.

Heat and sprint-training

Across Chapters 4-6, I showed the potential benefits of using heat training with sprint athletes. First, sprint and resistance training performed in the heat may induce beneficial cardiovascular adaptations without the need for endurance-type training. Improvements in submaximal aerobic fitness may translate to enhanced recovery between sprint efforts in training and competition. Furthermore, these findings suggest

that training in the heat for injured athletes could assist to attenuate the inevitable cardiovascular fitness loss. Second, repeated sprint performance may be enhanced in hot conditions, compared with temperate conditions, as well as following short-term HA. These findings suggest that heat training for sprint athletes may beneficial during targeted training blocks. Figure 2.1 (Literature Review) further eludes to potential mechanisms and practical uses of applying heat stress or heat training for sprint or power events. Although sprint performance has been shown to be enhanced in hot conditions (Ball et al., 1999; Girard et al., 2013; Sargeant, 1987), there is likely to be an individual response that may be reliant on the extent of muscle temperature rise. Last, caution should be given when using heat training with sprint athletes, as it may increase neuromuscular fatigue and reduce peak power output in temperate conditions in the 24 h period following.

Heated resistance training

The findings within this thesis extend on evidence showing that hot external environments that increase body temperature can enhance power production in athletes, and the methods shown herein provides a practical way of integrating heat into training programmes for power sport athletes. Until now, most research involving exercise in the heat has used environmental chambers, where chamber size may limit the modes of training that can be performed. A heated resistance training area may enhance the training stimulus during sport-specific power exercises for athletes in sports such as weightlifting, throws, jumping, and sprinting. Not only was power in these athletes shown to increase, but so was maximal power production and training quality perception. The strength- and power-based exercises performed in the heat (Chapters 4-

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6) caused low levels of thermal strain, yet performance during the sessions was not compromised. Nevertheless, I showed that resistance training in the heat induces partial HA adaptation through de

creases in body temperature and sweat rate. Therefore, heated resistance training may serve as an alternative to steady-state HA, and might appeal to team sports and those sports where resistance training forms an integral component of the programme.

Athlete considerations

Findings throughout this thesis provides further evidence for concept that there is high variation in individual athlete response to training and environmental stress. Understanding these different responses in athletes is a critical piece of the programming puzzle that coaches and sports scientists require to implement successful training blocks that herald peak performance. Prior to major competitions held in hot environments, knowing how each athlete responds to added heat stress and acclimation through repeat heat response testing is key, and should be conducted well in advance. Heat response tests can be sport-specific and ideally include a performance measure. The protocol should complement normal training so it can be easily repeated, possibly multiple times a season, to assess an athlete's level of acclimation. High inter-individual variability in response to heat response testing should be expected, even within similar athlete cohorts, which may be explained by several factors that are summarised in Table 7.2 of this thesis. Understanding individual athlete responses to heat stress, HA and/or heat training, will assist practitioners to tailor protocols to their individual needs and hopefully benefit performance outcomes.

9.4 Future research

This thesis focused on short-term, non-invasive heat training methods to optimise the use of heat to enhance performance. While the studies within this thesis form a starting point towards understanding this area, much is still to be determined. First, relatively little is known about how to periodise and manipulate the timing of HA throughout an athlete's typical annual (or quadrennial) training calendar, as either an acclimation or training tool, so as to maximise major event competition performance. Second, there is a lack of evidence of the effects of sprint and/or resistance training in the heat, on measures of sprint, power and strength performance. It is possible that a sustained elevation in muscle temperature may be a key signal/stimulus for eliciting acute power performance and potential chronic heat-induced augmentation in skeletal muscle adaptations. However, mechanistic studies are needed to examine this concept. Third, the effects of different modes of resistance training in the heat (E.g. strength, power, strength-endurance, hypertrophy, circuit training etc.) needs further investigation in order to determine both the acute and chronic effects of such training. Further insight will help to ascertain if heated resistance training holds ergogenic potential and HA applications. Finally, an understanding of how to integrate HA and heat training into routine training programmes, so as to optimise individual athlete's (positive, negative and non-responders) thermoregulatory and performance, responses will aide practitioners in designing suitable heat training and HA protocols for elite athletes.

9.5 Concluding remarks

The aim of this thesis was to improve understanding around how heat can be used as a tool to enhance performance in elite athletes, and from this, consider practical applied strategies. Based on its findings, combined with previous literature, HA and heat training strategies were developed and performed by multiple New Zealand National Sports Organisations (Yachting, Sprint Track Cycling, BMX, Weightlifting, Hockey, Rugby Sevens, Paralympic Shooting and Paralympic Track Cycling) during the 3-year period leading up to the Rio Olympic and Paralympic Games. It is hoped that the research presented herein has made a meaningful contribution to the wider international community in the area of heat acclimation and heat training and their application in elite sport. These strategies can be used by coaches and sport-scientists to optimise the use of heat with their athletes to enhance performance in both hot and temperate conditions. Such strategies may be considered further, as we project our thoughts towards preparing for the next Olympic Summer Games event in Tokyo 2020, which some are predicting will be the hottest Olympic Games on record to date.

CHAPTER TEN: REFERENCES

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CHAPTER ELEVEN: APPENDICES

LIST OF APPENDICES

Participant Information Sheet

22nd April, 2014

Project Title

Effects of sprint and strength heat acclimation training on sprint performance in highly-trained BMX cyclists

An invitation

Hi, my name is Julia Casadio and I am a PhD student at AUT, as well as a Performance Physiologist for High Performance Sport New Zealand (HPSNZ). Along with Adjunct Prof Paul Laursen and Assoc. Prof Andrew Kilding, I invite you to help with a project that examines the effects of sprint training in the heat before and after heat acclimation, in highly-trained BMX cyclists.

Purpose of this research?

Exercise in hot environments (≥ 27°C) results in an increase in core body temperature, which is known to impair exercise performance over a variety of sport events. Acclimatising the body to the temperatures it will perform in, through training in the heat (heat acclimation), prepares an individual to deal with the competition environment more efficiently. Ultimately, improving an athlete's ability to train and compete in hot and/or humid environments. As BMX athletes and coaches have experienced negative effects of the heat during competition, this research aims to develop heat management plans for individual athletes by answer the following:

1) How does each athlete respond and perform in the heat before heat acclimation?

2) How does each athlete respond and perform in the heat after heat acclimation?

How was I identified and why am I being invited to participate in this research?

As a BMX athlete you have been invited to be part of this research. However, you will not be able to take part in this research if:

✓ *You have any known heart or cardiovascular condition or if a member of your family died below the age of 50 as a result of a heart condition*
✓ *You have ever had an injury or medical condition that you think may affect your ability to sense pain or discomfort*
✓ *You have ever had persistent or regular back pain*
✓ *You are taking prescribed medication*

  ✓ *You have cultural or religious sensitivities about human body measurements*
  ✓ *You have any other reason to consider that you are not in good health and of average, or better than average fitness*
  ✓ *You are diabetic*

What will happen in this research?

If you are between 16-19 years old you will be required to sign an informed consent form, as well as have your parent/legal guardian sign a consent form, in order to participate in this research. All those 20 years or older will need to sign their own consent form. Thereafter, your involvement in this research will require you to do 3 heat response tests (HRT), with one conducted in normal conditions (20°C) and two in hot conditions (35°C), at the beginning and end of the heat training programme. During each HRT core temperature, skin temperature, heart rate, sweat loss, thermal comfort and sensation, rate of perceived exertion and plasma volume will be measured. Core temperature is measured using a small rectal probe which you will self-insert ~ 12 cm past the rectal sphincter. At the opposite end of the rectal probe there is a wire attachment that will sit outside of your cycling shorts/pants and will plug into a data logger which will record your core temperature while you are exercising in the hot or thermoneutral conditions. This is the gold standard technique for measuring core temperature in athletes when exercising. Plasma volume will be measured by taking a routine blood sample. The Performance Physiologists running these measurements are very experienced in running such tests and have the necessary qualifications. Your coach or parent/guardian may also be present at each testing session. Further details of the HRT's can be found below.

Heat Response Test:

- The HRT includes a resting blood sample, 20 min of easy spinning on a bike, a single 5 s inertial sprint, and a repeated sprint performance test (3 sets of supra-maximal sprinting with 3 x 10 s on/off). This will be done first in normal (20°C) conditions, and then twice in hot conditions (35°C), both before and after the heat camp. Once the blood sample is taken, the whole HRT session should take ~ 60 min.

- During the HRT we will measure core temperature, skin temperature, heart rate, rate of perceived exertion and thermal perception, as mentioned above.

Heat Training Programme:

- The heat training programme will consist of resistance training in a heated gym (30°C, 40% RH) while wearing long sleeves and pants to increase heat storage for 3 sessions (Monday, Wednesday and Friday). On alternate days (Tuesday, Thursday), you will perform a sprint-interval training session in a climate chamber (35°C, 60% RH).
- Each training session will be ~ 60 min, including a warm up and cool down, and will include exercises that you are currently performing in your training programme.

If you have any personal or cultural issues regarding the above procedures, please let the primary researcher now of these prior to the study so that these can be accommodated for.

What are the discomforts and risks?

The discomforts you may experience during this research are the same you would experience during your normal sprint training (heavy legs and breathing). You may also experience discomfort during the blood collection in the form of a small sting from the needle prick. The concept of the rectal probe may sound uncomfortable, however, once inserted many athletes report that they do not notice it while exercising. Furthermore, there are no risks of having core temperature measured this way. Finally, you may experience discomfort when exercising in the hot conditions due to elevated body temperature and sweat rate. There is no risk of heat injury as your core temperature and thermal perception will be closely monitored and you will be able to leave the heat chamber at any time.

What are the benefits?

You will benefit from being part of this study as you will learn your personal response to performing exercise in the heat, as this has been shown to be highly individual. You will also gain knowledge about how well you adapt to the heat. Such information can guide you and your coach (if permission is granted to share your results with them) in making you an individualised heat management plan. This experience and information will assist you when competitions are held in hot environments.

What compensation is available for injury or negligence?

In the unlikely event of a physical injury as a result of your participation in this study, rehabilitation and compensation for injury by accident may be available from the Accident Compensation Corporation, providing the incident details to satisfy the requirements of the law and the Corporation's regulations.

How will my privacy be protected?

All the data gained during this study will only be available to the researchers involved. If the data is published in the public domain your name as a subject will not be revealed and all subjects will remain anonymous.

What are the costs of participating in this research?

The cost of participating in this study will be the extra time for lab-based training sessions (~10 h), required to instrument you with sensors for heart rate and body temperature, as well as the blood sample.

Will I receive feedback on the results of this research?

At the end of the study, verbal feedback will be given to you and written feedback can be provided upon request. Your results from the study will only be shared with your coach if you grant us permission.

What do I do if I have concerns about this research?

Any concerns regarding the nature of this project should be notified in the first instance to the Project Supervisor:

Name: Prof Paul Laursen
E-mail: paul.laursen@hpsnz.org,nz

Concerns regarding the conduct of the research should be notified to the Executive Secretary, AUTEC, Kate O'Connor, *ethics@aut.ac.nz*, 921 9999 ext 6038.

Whom do I contact for further information about this research?

Researcher Contact Details:

Name: Julia Casadio; E-mail: julia.casadio@hpsnz.org.nz

Project Supervisor Contact Details:

Name: Prof Paul Laursen; E-mail: paul.laursen@hpsnz.org.nz

Approved by the Auckland University of Technology Ethics Committee on *12/6/14*, AUTEC Reference number *14/130*

Consent Form

Project Title:	Effects of sprint and strength heat acclimation training on sprint performance in highly-trained BMX athletes.
Project Supervisors:	Prof Paul Laursen, Assoc Prof Andy Kilding
Researcher:	Julia Casadio

- I have read and understood the information provided about this research project (Information Sheet dated 22 April, 2014).
        Yes/No

- I have had an opportunity to ask questions and to have them answered.   **Yes/No**

- I am in good health and am not currently suffering from any injury or illness which may impair my physical performance   **Yes/No**

- I agree to provide blood and samples and will inform the researchers before participation if I require my samples to be returned after analysis.   **Yes/No**

- I understand that I may withdraw myself or any information that I have provided for this project at any time prior to completion of data collection, without being disadvantaged in any way.

        Yes/No

- I agree to take part in this research.   **Yes/No**

- I agree to allow the use of my collected data to be used for research, including journal publications and post-graduate thesis
        Yes/No

- I agree to allow the use of my collected data to be shared with my coach.   **Yes/No**

- I understand my data collected will be de-identified prior to analysis and will be held for the purpose of research only (by the names researcher and supervisor) for a period of three years.   **Yes/No**

Participant signature: _____

Participant name: _____

Date: _____

If the participant is between 16-19 years old please also complete the Parental/Guardian Consent Forms.

Project Supervisor Contact Details:
Adjunct Prof Paul Laursen
High Performance Sport New Zealand
AUT|Millennium
17 Antares Place, Mairangi Bay, 0632
Mob: 021-303-153
Email: paul.laursen@hpsnz.org.nz

Approved by the Auckland University of Technology Ethics Committee Date 12/6/14

A U T E C
S E C R E T A R I A T

12 June 2014

Paul Laursen
Faculty of Health and Environmental Sciences

Dear Paul

Re Ethics Application: **14/130 Effects of sprint and strength heat acclimation training on sprint performance in highly-trained BMX cyclists.**

Thank you for providing evidence as requested, which satisfies the points raised by the Auckland University of Technology Ethics Committee (AUTEC).

Your ethics application has been approved for three years until 12 June 2017.

As part of the ethics approval process, you are required to submit the following to AUTEC:

- A brief annual progress report using form EA2, which is available online through http://www.aut.ac.nz/researchethics. When necessary this form may also be used to request an extension of the approval at least one month prior to its expiry on 12 June 2017;

- A brief report on the status of the project using form EA3, which is available online through http://www.aut.ac.nz/researchethics. This report is to be submitted either when the approval expires on 12 June 2017 or on completion of the project.

It is a condition of approval that AUTEC is notified of any adverse events or if the research does not commence. AUTEC approval needs to be sought for any alteration to the research, including any alteration of or addition to any documents that are provided to participants. You are responsible for ensuring that research undertaken under this approval occurs within the parameters outlined in the approved application.

AUTEC grants ethical approval only. If you require management approval from an institution or organisation for your research, then you will need to obtain this.

To enable us to provide you with efficient service, please use the application number and study title in all correspondence with us. If you have any enquiries about this application, or anything else, please do contact us at ethics@aut.ac.nz.

All the very best with your research,

Kate O'Connor
Executive Secretary
Auckland University of Technology Ethics Committee

Cc: Julia   Skleryk   julia.skleryk@hpsnz.org.nz

Chapter Eleven – Appendix D

Heated Gym

Picture of the heated resistance training space used in Chapers 4-6. The space was heated using a combination of heat pumps and infrared heated panels. An inductrial thermal curtain was hung to prevent heat loss from the gym into the surrounding sports hall.

Female athlete participating in the heated reistsance training study (Study 4).

Male athlete participating in Study 4. Long sleeve tops and pants were worn during the hot trial.

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PRIORITY REPORT

∂ OPEN ACCESS

Periodizing heat acclimation in elite Laser sailors preparing for a world championship event in hot conditions

Julia R. Casadio[a,b], Andrew E. Kilding[b], Rodney Siegel[c], James D. Cotter[d], and Paul B. Laursen[a,b]

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ABSTRACT

Purpose: To examine the retention and re-acclimation responses during a periodized heat acclimation (HA) protocol in elite sailors preparing for the 2013 World Championships in Muscat, Oman (~27–30°C, 40–60% RH). **Methods**: Two elite male Laser class sailors completed 5 consecutive days of HA (60 min per day in 35°C, 60% RH). Heat response tests (HRT) were performed on day 1 and 5 of HA, then 1 (decay 1, D1) and 2 (D2) weeks following HA. Participants were then re-acclimated (RA) for 2 days, within the next week, before a final HRT ~72 h post-RA. Rectal temperature, plasma volume, heart rate, sweat rate, as well as thermal discomfort and rating of perceived exertion were measured during each HRT. **Results**: Rectal temperature decreased with HA (0.46 ± 0.05°C), while individual responses following D1, D2 and RA varied. Heart rate (14 ± 7 bpm), thermal discomfort (0.6 ± 0.1 AU) and rating of perceived exertion (1.8 ± 0.6 AU) decreased across HA, and adaptations were retained by D2. Plasma volume steadily increased over the decay period (D2 = 8.0 ± 1.3%) and after RA (15.5 ± 1.1%) compared with baseline. RA resulted in further thermoregulatory improvements in each Athlete, although individual adjustments varied. **Conclusion**: Heat strain was reduced in elite Laser sailors following HA and most thermoregulatory adaptations were retained for 2 weeks afterwards. RA may 'top up' adaptations after 2 weeks of HA decay.

Introduction

Short-term heat acclimation is a commonly used strategy to prepare athletes for competition in hot environments and typically involves 4–7 days of aerobic training in artificially hot conditions (30–40°C).[1] The physiological adaptations seen with heat acclimation (HA) include decreased resting and exercise core temperatures, lowered skin temperature, increased plasma volume and sweat rate and a decreased heart rate for a given workload, all of which attenuate performance impairments that are caused by hot conditions in the unacclimated state.[2] An acute training session in the heat causes considerable physiological strain[3] which, accumulated over several days during HA, could misalign with training priorities during an athlete's taper phase leading in to a major competition. While some suggest incorporating HA to a precompetition taper,[4,5] the added heat-induced stress may require a reduction in training intensity,[6] which could hinder taper

objectives where athletes often reduce training volume and increase or maintain training intensity.[7] Moreover, training in hot conditions may cause an unplanned rise in internal training load.[8] Therefore, examining alternate application models of HA periodization in athletes preparing for competitions in the heat is needed.

The time course of heat acclimation and re-acclimation is not yet clear in trained populations.[4] Studies with untrained participants have shown that re-acclimation to moderate-term HA (8–14 d) may occur after only a short number of heat exposures (1–4 d).[9,10] Highly-trained individuals may have even faster rates of HA accrual and slower rates of HA decay.[11] Furthermore, it has been suggested that periodical exposure to heat following HA can allow for the retention of HA over several weeks,[12] however, supporting data in athletes is lacking. The aim of this study was to examine the retention of HA adaptations and re-acclimation responses during a periodized

Casadio JR, Kilding AE, Siegel R, Cotter, JD Laursen PB, (2016). Periodizing heat acclimation in elite Laser sailors preparing for a World Championship event in hot conditions. *Temperature* 3 (3): 437-443. (Casadio 80%, Kilding 5%, Siegel 5%, Cotter 5% Laursen 5%)

Heat acclimatization can improve performance in a cool condition in some specific conditions or populations only

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Minson & Cotter (2016) highlight that heat acclimation (HA) can be ergogenic in certain individuals, in part through the expansion of plasma volume. They also suggest that there may be responders and non-responders to HA and describe many unresolved/unexamined issues. This is important because correlations have been reported between plasma volume expansion and performance improvements in the heat only, whereas improvements in cool likely relate to different factors (Racinais *et al.* 2012, 2014). Indeed, it may be worth considering contextual factors such as training status (Nybo & Lundby, 2016). For example, HA may optimize physiological adaptations in team-sport athletes, while saving time for technical training (Buchheit *et al.* 2011, 2013). HA may also speed up pre-season adaptations in team-sport athletes, rather than increase maximal performance (Racinais *et al.* 2014). Hence, HA might be suitable to boost physiological adaptations for a given training load at a particular time within a season. HA could therefore be of particular interest for sub-elite or non-endurance athletes, for pre-season training and for experienced athletes requiring a new training stimulus (Racinais *et al.* 2014). Moreover, HA could represent a valuable tool for post-injury rehabilitation in order to optimize the metabolic-to-mechanical load ratio. Lastly, as noted in both viewpoints, there is no evidence that HA impairs cool weather performance; thus it should be implemented when the weather conditions are uncertain, as may be the case for the 2016 summer Olympics where conditions are likely to range from cool to tropical.

Competing interests

None declared.

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HA and performance in the elite: considering the individual

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In this CrossTalk debate, Nybo & Lundby (2016) dismiss the concept that the response to heat acclimation may be highly individual; that is, that there could be responders and non-responders. From our point of view and experience within the Olympic athlete environment, this assumption is an oversight. Indeed, the individual response to any form of planned training cannot be overlooked, especially when considering the narrow margins of importance within the world of elite sport. The conclusion that HA does not enhance performance in temperate conditions may be supported by comparisons of the mean response in some studies (Nybo & Lundby, 2016), yet when individual responses are presented, clear responders and non-responders emerge (Racinais *et al.* 2012; Neal *et al.* 2016). For example, Racinais *et al.* (2012) reported large inter-individual differences in soccer players' response to HA (Δ plasma volume: −10 to +20%) and their ability to perform in the heat following HA (total distance run: −6 ± 6%; high-intensity running: −16 ± 21%) (Racinais *et al.* 2012). As well, a closer look at the individual data presented by Neal *et al.* (2016) reveals that ~40% of cyclists improved their V_{O_2max} (+5–11%) and time trial power output (+3–5%) (Neal *et al.* 2016). As the list of physiological adaptations to HA with ergogenic potential is long (Corbett *et al.* 2014), more work is needed to understand the causal effects of HA-induced temperate performance enhancement in those athletes who do 'respond'. While caution is warranted in assessing true individual effects, repeating performance measures along with reporting of the measurement error and smallest worthwhile change can assist to decipher uncertainties between normal within-subject variation 'noise', and meaningful important responses (Hopkins, 2015).

Competing interests

None declared.

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ORIGINAL ARTICLE

Acute effects of heated resistance exercise in female and male power athletes

Julia R. Casadio[1,2] · Adam G. Storey[1,2] · Fabrice Merien[3] · Andrew E. Kilding[1] · James D. Cotter[4] · Paul B. Laursen[1,2]

Abstract

Purpose To determine the effects of heated resistance exercise on thermal strain, neuromuscular function and hormonal responses in power athletes.

Methods Sixteen ($n = 8$ female; 8 male) highly trained power athletes completed a combined strength and power resistance exercise session in hot (HOT ~30 °C) and temperate (CON ~20 °C) conditions. Human growth hormone (hGH), cortisol and testosterone concentrations in plasma, peak power (counter-movement jump, CMJ) and peak force (isometric mid-thigh pull) were measured before and after each training session; thermoregulatory responses were monitored during training.

Results Skin temperature, thermal sensation and thermal discomfort were higher in HOT compared with CON. Sweat rate was higher in HOT for males only. Compared with CON, HOT had trivial effects on core temperature and heart rate. During HOT, there was a possible increase in upper-body power (medicine ball throw) in females [3.4% (90% CL −1.5, 8.6)] and males [(3.3% (−0.1, 6.9)], while lower-body power (vertical jump) was enhanced in males only [3.2% (−0.4, 6.9)]. Following HOT, CMJ peak power [4.4% (2.5; 6.3)] and strength [8.2% (3.1, 13.6)] were enhanced in female athletes, compared with CON, while effects in males were unclear. Plasma hGH concentration increased in females [83% (18; 183)] and males [107% (−21; 444)] in HOT compared with CON, whereas differential changes occurred for cortisol and testosterone.

Conclusion Heated resistance exercise enhanced power and increased plasma hGH concentration in female and males power athletes. Further research is required to assess the ergogenic potential of resistance exercise in the heat.

Keywords Strength · Growth hormone · Testosterone · Cortisol

Abbreviations

AU	Arbitrary unit
ATP	Adenosine triphosphate
CMJ	Counter-movement jump
CON	Control condition
CSA	Cross sectional area
ES	Effect size
FSD	Full scale of deflection
hGH	Human growth hormone
iMTP	Isometric mid-thigh pull
mTOR	Mammalian target of rapamycin
PCr	Phosphocreatine
RH	Relative humidity
RM	Repition maximum
SD	Standard deviation

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Chapter Eleven – Appendix H

CrossMark

CURRENT OPINION

From Lab to Real World: Heat Acclimation Considerations for Elite Athletes

Julia R. Casadio[1,2] · Andrew E. Kilding[1] · James D. Cotter[3] · Paul B. Laursen[1,2]

Abstract As major sporting events are often held in hot environments, increased interest in ways of optimally heat acclimating athletes to maximise performance has emerged. Heat acclimation involves repeated exercise sessions in hot conditions that induce physiological and thermoregulatory adaptations that attenuate heat-induced performance impairments. Current evidence-based guidelines for heat acclimation are clear, but the application of these recommendations is not always aligned with the time commitments and training priorities of elite athletes. Alternative forms of heat acclimation investigated include hot water immersion and sauna bathing, yet uncertainty remains around the efficacy of these methods for reducing heat-induced performance impairments, as well as how this form of heat stress may add to an athlete's overall training load. An understanding of how to optimally prescribe and periodise heat acclimation based on the performance determinants of a given event is limited, as is knowledge of how heat acclimation may affect the quality of concurrent training sessions. Finally, differences in individual athlete responses to heat acclimation need to be considered. This article addresses alternative methods of heat acclimation and heat exposure, explores gaps in literature around understanding the real world application of heat acclimation for athletes, and highlights specific athlete considerations for practitioners.

Key Points

Post-exercise sauna bathing and/or hot water immersion may represent a practical means of implementing heat acclimation (HA) in athletes when barriers to traditional exercise-based HA are present.

To optimise HA, the timing of implementation, sport specificity and other concurrent training sessions should all be considered in order to maintain training quality and maximise performance in the heat.

Several unique athlete considerations, including their history and physical characteristics, should be understood by practitioners before implementing HA protocols, as individual characteristics often elicit different heat stress and HA responses.

1 Introduction

Several major sporting events are held each year in hot environments. Some of the largest high profile events, including most Summer Olympic Games, the Tour de France, the FIFA World Cup, as well as several annual World Cups and World Championships are held in the summer months when high temperatures are often expected. For athletes and support teams preparing for pinnacle events, executing performance to their maximum potential is of the utmost importance. Heat-induced performance decrements can range from 6 to 16% in trained athletes

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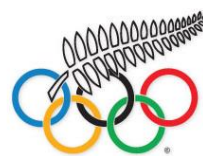
2016 Olympic and Paralympic Games Environmental Assessment Report

Rio de Janeiro, Brazil
October 2013

By: Julia Skleryk
Performance Physiologist, HPSNZ
PhD Scholar in Heat and Performance

POSITION DESCRIPTION

TITLE	Preparation and Recovery Support Role
TEAM	Olympic Games – Rio 2016
REPORTS TO	Preparation & Recovery Area Leader
DATE	November 2015

NZOC is now seeking a person with appropriate skills, high performance experience and personal attributes suited to working in a team.

A. Purpose of the Position

The key focus will be supporting the activities of the Preparation and Recovery area (alongside a team of two others) and providing support as required. They will assist with the delivery of the performance focused team environment.

B. Location

The role will be village-based and primarily operate from New Zealand Team's Preparation and Recovery Area in the Rio Olympics Village. At this time accommodation will likely be predominantly based out of village and access will be via day passes between 9am – 9pm each day.

C. Duties

As part of the Preparation and Recovery area team, provide support to New Zealand team athletes in Rio 2016. Duties include:

a) Work effectively together as part of the team to assist in the coordination of the preparation and recovery areas, including appropriate use of the area, maintenance, hygiene and safety, restocking of agreed supplies, timetabling and record keeping.
b) Support athletes in the correct recovery strategies within the Preparation and Recovery areas as requested by NSO support team's, Medical and Area Leader.
c) Support NSO support teams in the use, cleaning and recycling of water bottles, slurry machine and shakers and maintaining availability for use.
d) Provide support for monitoring and intervention strategies if requested by NSO support teams and medical.
e) Any other duties as required by the Area Leader, NZOC management and/or medical teams.

D. Essential Experience

- Currently working fulltime for HPSNZ Athlete Performance Support.
- Experience in a direct athlete delivery role.
- Knowledge and skills in preparation and recovery strategies.

E. Essential Personal Attributes

- A commitment to assist in the development of a performance-focused team environment.
- An ability to work as a team player.
- Ability to manage self, remain calm and work with others effectively for prolonged periods while being in a challenging and high pressure environment.
- Flexible and conscientious approach to work.
- Positive energy and uses initiative.
- Good communicator.

F. Time commitment

<u>Pre Games</u>

- Attendance at 2 Support Staff forums.
  - 6 & 7 May 2016
  - 17 & 18 June 2016
- Meetings as required with Preparation and Recovery Area Leader and/or NZOC Performance Nutritionist, for planning purposes.

<u>Games Time</u>

Attendance at Games during an agreed period between 19 July to 23 August 2016.

<u>Post Games</u>

Attendance at a 1 day debrief/review session.

G. Costs

- Time for the role will be covered by HPSNZ as it is incorporated into the candidate's existing role.
- Costs for Games related air travel, outfitting, accommodation and food will be met by the NZOC

Rio Olympic Team Recognition Medal

"The Olympic Games would not be possible without your involvement. Thank you for helping make the Olympic Games Rio de Janeiro 2016 such a success."

"Os Jogos Olímpicos não seriam possíveis sem a sua participação. Obrigado por nos ajudar a fazer dos Jogos Olímpicos Rio 2016 um sucesso."

Thomas Bach
President | Presidente
International Olympic Committee
Comitê Olímpico Internacional

"Thanks for helping make Brazil's first ever Olympic Games a memorable celebration. Your contribution to Rio 2016 Olympic Games has made a difference."

"Obrigado por ajudar a tornar os primeiros Jogos Olímpicos do Brasil uma celebração memorável. Sua contribuição aos Jogos Olímpicos Rio 2016 fez diferença."

Carlos Arthur Nuzman
President | Presidente
Rio 2016 Organising Committee for the Olympic and Paralympic Games
Comitê Organizador dos Jogos Olímpicos e Paralímpicos Rio 2016

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Faculty	Health	School/Dept	Sport and Recreation
Programme	Doctor of Philosophy	Date of submission for examination	16 February 2017
Research Output	Thesis ☒ Dissertation ☐ Exegesis ☐	Points Value	
Thesis Title	Novel applications of heat training to enhance performance in elite athletes		

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Primary Supervisor	Paul Laursen	Signature		Date	15/2/2017
Secondary Supervisor	Andrew Kilding	Signature		Date	15/2/2017

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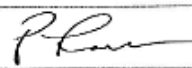**SPORT NEW ZEALAND**

EMBARGO AGREEMENT

13 February 2017

Craig Palmer
Cycling New Zealand
15 Hanlin Rd
Cambridge 3283
Waikato, New Zealand

Dear Craig Palmer:

This letter serves as a formal agreement between AUT doctoral student and High Performance Sport New Zealand employee, Julia Casadio, and Cycling New Zealand (CNZ) representative, Craig Palmer, whereby the content of Mrs. Casadio's thesis titled "Novel Applications of Heat Training to Enhance Performance in Elite Athletes" will be embargoed until May 2018.

Specifically, studies 2 (Chapter 3) and 3 (Chapter 4) of Mrs. Casadio's thesis, along with the overall discussion (Chapter 9) are subject to embargo until May 2018. Cycling New Zealand believe the findings of this work represent a competitive advantage to the New Zealand track cycling team and the campaign towards the Gold Coast Common Wealth Games in 2018.

Julia Casadio (PhD Candidate)	Signature		Date 13/2/17
Paul Laursen (Primary Supervisor)	Signature		Date 13/2/17
Craig Palmer (CNZ representative)	Signature		Date 15/2/17
	Signature		Date

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