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The effects of environmental temperature on high-intensity interval training

Jason R. Boynton
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The Effects of Environmental Temperature on High-Intensity Interval Training

This thesis is presented for the award of
Doctor of Philosophy (Sport Science)

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School of Medical and Health Sciences

2020

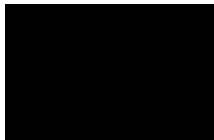
DECLARATION

I certify that this thesis does not, to the best of my knowledge and belief:

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ACKNOWLEDGEMENTS

The long ride...

I will begin this section with an obligatory sigh of relief expressed in my native Midwestern tongue: *uff da!*

So, I get the impression from talking with, and reading acknowledgements of other PhD candidates, people put a fair amount of time into this. To be quite honest my mind hasn't really mulled this over much because it really doesn't feel like I'm coming to the end of a journey. It really does feel like tomorrow will be just another day in Australia as a PhD candidate.

Anyhow, I don't have a lot of time at the moment, so I'm just going to shoot from the hip.

And without further ado, I give thanks to those who rode along with me...

Supervisory Panel

Associate Professor Chris Abbiss: Many thanks to you for your patience, empathy, and guidance. I've always felt very lucky to have received the opportunity to work with you during my PhD. It was an enriching and humbling experience, without a doubt. I think my only regret was we weren't able to get out on more rides while I was here. Thanks for introducing me to the power of the word 'indeed' when writing scientifically (I use it correctly probably about 76% of the time now) and showing me how to take off knee warmers while riding at the same time. Anyhow, thanks again for taking a chance on an awkward guy from the Midwest. Please remain a standup human being.

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many great conversations early on in my PhD. It was a great addition to the overall experience. Watching you on your career journey has been very inspiring. Thanks for being there with all of your practical and professional advice! It's certainly been appreciated.

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Fellow Academics

Dr. Oliver Barley: Mate, I don't know what this journey would have been like without you there. You are at the apex of a good friend and a good colleague. And who would have thought the MMA guy and cyclist would have gotten along so well? For me, I think it's because I always know what your underlying values are, so I always have a good idea where you stand on most things. I could probably say a lot more, but I have to save some space and time for other people. Honestly, if you play back in your head the speech your dad gave you at your wedding, I'd say I'm solidly in alignment with that.

Georgios Mavropalias and Ricardo Mesquita: My Mediterranean Scatlords. So many burritos. Let me say that again. Soooo many burritos. I don't know what the future holds, with the coronapocalypse and all, but whether we Mad Mex it or Mad Max it, hopefully our paths cross often. Thanks for being good friends and colleagues!

Dr. Kester Choo: My comrade-in-chamber. Thanks for all of your practical guidance and patience as I broadened my resume from an exercise physiologist to an environmental exercise physiologist

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Nadija Vrdoljak: You were the yang for the grad students' yin. You brought order to our chaos. Thanks for all of your help during my research. I will remember fondly our office conversations and colorful rants.

Family and Friends

Mom: Well, here I am at the end of another degree. Lucky for you, I can't really continue formally with my education without making a move down or laterally. So, this is where I thank you for all of it. You have given so much so that I could follow my dreams. I sincerely thank you for all of your support over these many years. Much love!

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The end of the ride...

And I guess that is it. I reckon the biggest gauge of success for this experience is I realize how little I actually know, despite learning much. But if there is one thing I know for certain, it's that Jeffrey Epstein did not commit suicide. Cheers!

ABSTRACT

Endurance athletes typically spend the large majority of training (> 70%) at low intensities (i.e. below lactate threshold) coupled with short and intermittent bouts of high-intensity exercise or interval training (HIIT). Despite HIIT being a relatively small part of training in terms of duration, it has a substantial effect on the adaptations to endurance training. While it is well-established endurance exercise performance is affected in both hot and cold environmental conditions, the effect ambient temperature (T_A ; frequently referred to as environmental temperature) has on HIIT as performed by an endurance athlete population is not well understood. Therefore, the overall purpose of this thesis was to investigate the effects T_A has on HIIT in an endurance trained population. Specifically, this thesis aimed to increase the understanding of how T_A acutely affects performance and physiological responses during high-intensity intervals (Study 1); how repeated exposure to T_A manipulates physiological responses during high-intensity intervals (Study 2), and how T_A affects performance outcomes of a HIIT intervention (Study 3).

In Study 1, eleven well-trained cyclists completed 4 interval sessions at 5°C, 13°C, 22°C, and 35°C (55 ± 13% RH) in a randomised order. Each session involved 5 x 4-minute intervals interspersed with 5 minutes of recovery. During the intervals, power output, core temperature (T_C), oxygen consumption (VO_2), and heart rate (HR) were recorded. It was hypothesized that the 13°C condition would have the highest mean power output compared to the other T_A conditions. However, mean session power output for 13°C (366 ± 32 W) was not significantly different than 5°C (363 ± 32 W), 22°C (364 ± 36 W), or 35°C (352 ± 31 W). Power output was lower in the 5th interval of the 35°C condition, compared with all other T_A . T_C was higher in 22°C compared with both 5°C and 13°C ($P = .001$). VO_2 was not different across T_A . HR was higher in the 4th and 5th intervals of 35°C compared with 5°C and 13°C. It was concluded well-trained cyclists performing

maximal high-intensity aerobic intervals can achieve near optimal power output over a broader range of T_A than previous literature may indicate.

Study 1 indicated T_A had acute effects on performance and physiological responses during high-intensity aerobic intervals, especially in terms of cardiovascular stress. However, whether acute cardiorespiratory and thermoregulatory responses during high-intensity intervals change as a result of repeated T_A exposures (i.e. during HIIT) was unknown. In Study 2, 20 trained cyclists and triathletes completed a 4-week (8 session) HIIT intervention in either cool (13°C) or hot (35°C) conditions. The HIIT intervention utilized the interval protocol from Study 1 and recorded cardiopulmonary and thermoregulatory measures during the first (INT1) and last (INT8) sessions. It was observed that time spent at or near maximal oxygen consumption ($\text{VO}_{2\text{max}}$) during HIIT was greater in 13°C (877 ± 297 seconds) than 35°C (421 ± 395 seconds), but did not change for either T_A condition between INT1 and INT8. HR was not significantly different between 13°C (164 ± 9 bpm) and 35°C HIIT (164 ± 12 bpm). T_C significantly decreased in 35°C HIIT between INT1 and INT8. These results potentially indicate the relationship between time spent at or near $\text{VO}_{2\text{max}}$ and cardiovascular strain during HIIT is influenced by T_A . Additionally, HIIT performed intermittently ($\sim 2\times$ per week) at 35°C resulted in demonstrated evidence for heat acclimation in endurance athletes.

Study 1 and Study 2 provided findings for performance, cardiorespiratory, and thermoregulatory responses during acute high-intensity interval sessions and after repeated exposure to T_A . In particular, differences in time spent at or near $\text{VO}_{2\text{max}}$ between 13°C and 35°C HIIT, and changes in thermoregulatory responses over the course of a HIIT intervention both have the potential to affect endurance performance outcomes and coinciding physiological responses. In order to investigate this, Study 3 evaluated submaximal warm-ups and 20 km time-trials in temperate

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The results of this thesis demonstrate T_A acutely affects performance, and cardiorespiratory and thermoregulatory responses during high-intensity intervals; repeated exposures to T_A during HIIT can stimulate changes in thermoregulatory responses; and T_A exposure during HIIT has limited effect on temperate endurance performance, yet affects coinciding cardiorespiratory, thermoregulatory, and subjective responses. These findings will assist coaches and athletes to make better informed decisions relating to HIIT prescription and acclimating endurance athletes to T_A .

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LIST OF PUBLICATIONS

The following chapter has been published in an academic journal:

Chapter Three: Study 1

Boynton, J.R., Danner, F., Menaspà, P., Peiffer, J.J., & Abbiss, C.R. (2019). Effects of environmental temperature on high-intensity intervals in well-trained cyclists. *International Journal of Sports Physiology and Performance*, 14(10): 1401-1407. Impact factor: [3.979].

(Appendix A)

Additional outcomes arising from PhD research

CONFERENCE PROCEEDINGS

Boynton, J.R., Danner, F., Menaspà, P., Peiffer, J.J., & Abbiss, C.R. *Effects of ambient temperature on high-intensity intervals in well-trained cyclists*. 23rd Annual ECSS Congress, Dublin, Ireland 2018.

(Appendix B)

1 CHAPTER ONE:
INTRODUCTION

1.1 Background

Endurance exercise performance is reduced in both hot^{1,2} and cold²⁻⁴ environmental conditions. This inverse U-shaped relationship implies that there is an optimal ambient temperature (T_A ; frequently referred to as environmental temperature) in which to perform an endurance task². According to the literature this temperature is in the range of 10-17°C^{2,5-7} and dependent on the mode of exercise, duration of exercise, and rate of convection^{8,9}. Yet, much of the research examining the influence of T_A on exercise capacity and the quantification of optimal environmental conditions has examined steady-state efforts at or around lactate threshold^{2,5-7}. Paradoxically, elite endurance athletes typically spend little time at these intensities during training and certain competitions^{10,11}. This is especially the case for elite/professional road cyclists¹². Indeed, such athletes typically spend the large majority of training (> 70%) at low intensities (i.e. below lactate threshold) coupled with short and intermittent bouts of high-intensity exercise or training^{10,11}. The influence of T_A on both performance and subsequent training adaptations at intensities observed in endurance athletes are not well understood.

High-intensity exercise results in an increased metabolic rate¹³ and therefore increases metabolic heat production^{14,15}, possibly compromising acute interval performance. Indeed, research by Drust et al.¹⁶ demonstrated that power output during repeated sprint efforts performed after 40 minutes of intermittent exercise in the heat (40°C) was decreased compared to a control condition (~20°C). Additionally, variations in an athlete's velocity throughout an interval training session will influence convective heat loss. It is therefore plausible that the effects of T_A on acute high-intensity interval performance would differ to prolonged steady-state exercise. It is also important to note that previous literature examining endurance exercise under a range of T_A does not usually

consider particular conditions that have an effect on endurance performance (e.g. a standardized warm-up, replication of realistic convective heat loss through high wind velocities^{8,17}, and training status). Galloway & Maughan² examined time-to-exhaustion under a range of T_A , however the mean maximal oxygen consumption (VO_{2max}) for these subjects was below what the literature would consider “well-trained” endurance athletes^{18,19}. This is important because trained individuals can exercise at higher exercise intensities¹⁸, eliciting higher metabolic heat production^{14,15} than untrained individuals²⁰. Indeed, heat appears to have a greater negative effect on the performance of top finishing marathon competitors (1st to 25th)⁵. However, it is not currently understood if the performance of high-intensity intervals conducted by trained endurance athletes is optimised in a particular range of T_A similar to that observed in the literature (10-17°C^{2,5-7}) involving steady-state exercise performed by recreationally active individuals.

Despite high-intensity exercise being a relatively small part of training in terms of duration^{10,11}, it has a large effect on the adaptations to endurance training^{21,22}. Structured high-intensity exercise with alternating periods of work and rest is recognized as high-intensity interval training (HIIT). It has been suggested that aerobic adaptations during HIIT sessions are stimulated most effectively when time spent at or near VO_{2max} is maximised²³⁻²⁵. However, during high-intensity exercise in hyperthermia, a reduction of blood flow to working muscles occurs, which in turn reduces VO_{2max} ^{26,27}. It is therefore plausible that endurance athletes regularly performing HIIT under hot conditions might experience detriments in training intensity and subsequent adaptations^{28,29}. Conversely, exercise in cooler conditions increase core temperature (T_C) to skin temperature (T_{sk}) gradient, reducing skin blood flow¹⁷, increasing venous return, and therefore enhance the proportion of cardiac output delivered to the active muscle²⁶. Indeed, increased muscle blood flow to the exercising muscles during single-leg cycling training³⁰ has been shown to stimulate a

superior oxidative potential and metabolic profile of the skeletal muscle compared to double-leg cycling³¹. Yet, it could also be argued that environmental conditions would have little influence on adaptations to HIIT interventions since cardiovascular strain may be similar under varying T_A conditions²⁶. Clearly, more research is required to determine how T_A affects adaptations to HIIT, and subsequent endurance performance outcomes.

As previously alluded to, performance of trained endurance athlete is negatively affected by hot T_A ³². However, repeated exposure to heat stimulates physiological adaptations (i.e. heat acclimatisation/heat acclimation (HA)) that improve performance of athletes exercising in hot conditions^{33–35}. Additionally, it has been demonstrated that HA, under certain circumstances, improves endurance performance in temperate conditions^{36–39}. Adaptations to repeated heat exposure that may provide ergogenic benefits in temperate conditions include: an increase in red-cell volume^{40,41}, increase in plasma volume⁴², enhanced skin blood flow response³⁸, decreased T_{C} ⁴³ (during rest and submaximal exercise), T_{Sk} ³⁸, and increased $T_C:T_{Sk}$ gradient³⁸. Adaptations to HA that affect endurance performance typically occur after repeated exposure to hot temperatures over 4 to 14 days with less than 2 days between exposures⁴⁴. Conversely, HIIT sessions are generally scheduled 2 days or more apart to allow for sufficient recovery and avoid maladaptation^{45,46}. Indeed, intermittent T_A exposures during HIIT could potentially influence cardiorespiratory adaptations and (or) performance outcomes, but be too infrequent to stimulate acclimation in trained endurance athletes. Currently, the effect of repeated heat exposures during HIIT and subsequent temperate performance is not well understood.

1.2 Purpose and Significance

The overall purpose of this thesis was to investigate the effects of T_A on high-intensity interval session performance and physiological responses, and HIIT outcomes in an endurance trained

population. The novelties of this thesis were the investigations to examine: T_A optimisation of high-intensity intervals; the effects of repeated hot and cool T_A exposure on the physiological responses during high-intensity intervals; and the effects of hot and cool HIIT on endurance athlete performance in temperate conditions. The findings from this thesis will assist coaches and athletes to make appropriate and informed decisions around training prescription (e.g. the environmental conditions to perform HIIT under). In particular, this thesis will give insight into training adaptations that occur after HIIT performed in different T_A , as well as T_A acclimation in endurance athletes performing repeated bouts of high-intensity exercise.

1.3 Research Aims

This thesis aimed to determine the effects T_A on high-intensity interval training. Focuses included an examination of the acute effects of T_A on high-intensity intervals, the effects of repeated exposure to T_A on physiological responses to high-intensity intervals, and the effects of hot and cool HIIT interventions performance outcomes.

The specific aims of each research study were:

1.3.1 Study 1 (Chapter 3)

To determine the acute influence of T_A (5°C, 13°C, 22°C, & 35°C) on performance and physiological responses during a maximal high-intensity interval session performed by well-trained cyclists.

1.3.2 Study 2 (Chapter 4)

To examine the effects of repeated exposures (8 sessions over 4 weeks) to cool (13°C) and hot (35°C) environmental conditions on cardiorespiratory and thermoregulatory responses during a maximal high-intensity interval session performed by trained cyclists.

1.3.3 Study 3 (Chapter 5)

To investigate the effects of 4 weeks of HIIT (8 sessions) in cool (13°C) and hot (35°C) environmental conditions on time-trial performance in a temperate environment (22°C) in trained cyclists.

1.4 Research Questions and Hypotheses

1.4.1 Study 1

1. What is the influence of T_A (5°C, 13°C, 22°C, & 35°C) on performance (e.g. power output) and physiological responses (e.g. oxygen consumption and body temperature), and subjective responses (e.g. rate of perceived exertion and thermal sensation) during a maximal high-intensity interval session (5 x 4 min) performed by well-trained cyclists?

- It was hypothesized that intervals performed at 13°C will result in the highest average power output during high-intensity intervals compared to other T_A (5°C, 22°C, & 35°C).
- It was hypothesized that intervals performed in cool (13°C) and temperate (22°C) T_A will result in an increase in $\dot{V}O_2$ & power output; a decrease in T_{C} , T_{sk} , sweat rate; & RPE will remain the same compared to intervals performed in hot T_A (35°C).

1.4.2 Study 2

1. Are there cardiorespiratory (e.g. heart rate and oxygen consumption), thermoregulatory (e.g. body temperature), subjective (e.g. rate of perceived exertion and thermal sensation), and power output differences between the first and last interval bouts of a 4-week intervention (8 total sessions) performed at two different temperature conditions (13°C & 35°C)?

- It was hypothesized that the last interval bout of a 4-week intervention of both temperature conditions (13°C & 35°C) would exhibit an increase in VO_2 and power output, while RPE and T_{C} would remain the same, when compared to the first interval bout.
- It was hypothesized that the increase in VO_2 and power output observed in the last interval bout of a 4-week intervention would be higher in the 13°C condition compared to the 35°C condition.

2. Do physiological markers of heat acclimation (e.g. body temperature & sweat rate) in trained cyclists differ between the first and last interval bouts of a 4-week intervention (8 total sessions) performed under two different temperature conditions (13°C & 35°C)?

- It was hypothesized there would be no change in markers of HA (body temperature measurements & sweat rate) following a 4-week interval intervention at 35°C & 13°C.

1.4.3 Study 3

1. Does 4 weeks of HIIT (8 sessions) in cool environmental conditions (13°C) improve performance and physiological responses of trained cyclists during a 20 km cycling time-trial (22°C), when compared to HIIT conducted in hot environmental conditions (35°C)?

- It was hypothesized that compared to interval training in the heat (35°C), interval training conducted in cool T_{A} (13°C) would result in increased average power during a simulated 20 km TT (22°C).

2. Does 4 weeks of HIIT (8 sessions) in cool environmental conditions (13°C) improve physiological responses (e.g. heart rate & oxygen consumption) and gross efficiency of trained

cyclists during a fixed-intensity submaximal test (22°C), when compared to HIIT conducted in hot environmental conditions (35°C)?

- It was hypothesized that compared to interval training in the heat (35°C), interval training conducted in cool T_A (13°C) would result in a lower heart rate, decreased $\dot{V}O_2$, and increased gross efficiency during a submaximal test (22°C).

1.5 Definition of Terms

ANOVA: Analysis of variance

GME: Gross mechanical efficiency

GXT: Graded exercise test

HA: Heat acclimation/Heat acclimatization

HIIT: High-intensity interval training

HIIT₁₃: 13°C high-intensity interval training group

HIIT₃₅: 35°C high-intensity interval training group

HR: Heart rate

HRR: Heart rate recovery

INT1: Interval session one

INT8: Interval session eight

O₂: Diatomic oxygen

PPO: Peak power output

RER: Respiratory exchange ratio

RH: Relative humidity

RPE: Rate of perceived exertion

$T > 90\% \text{VO}_{2\text{max}}$: Time above ninety percent maximal oxygen consumption

T_A : Ambient temperature (also referred to as “environmental temperature”)

T_{arm} : Arm skin temperature

T_B : Body temperature

T_C : Core temperature

$T_C:T_{\text{Sk}}$ gradient: Core temperature to skin temperature gradient

T_{leg} : Calf skin temperature

T_{thigh} : Thigh skin temperature

TS : Thermal sensation

T_{Sk} : Skin temperature

TT : Time-trial

$TT1$: Pre-intervention time-trial

$TT2$: Post-intervention time-trial

VE : Ventilation

VE/VO_2 : Ventilatory equivalent for oxygen consumption

VE/VCO_2 : Ventilatory equivalent for expired carbon dioxide

VO_2 : Volume of oxygen consumed

$\text{VO}_{2\text{max}}$: Maximal oxygen consumption

$\text{VO}_{2\text{peak}}$: Peak oxygen consumption

η_p^2 : Partial eta squared

2 CHAPTER TWO: REVIEW OF THE LITERATURE

ENVIRONMENTAL TEMPERATURE CONSIDERATIONS FOR HIGH-INTENSITY INTERVAL TRAINING

This review of literature provides background information relevant to the studies of this PhD thesis. The primary focus of this chapter was to introduce research that relates to environmental temperature's effects on high-intensity interval training on endurance athletes.

2.1 Introduction

Previous research has demonstrated that endurance athletes train in ‘polarized’ or ‘pyramid’ manners^{10,11}. These training approaches incorporate large volumes of training spent exercising at low intensities coupled with bouts of high-intensity exercise^{10,11}. When high-intensity exercise is structured into repeated work periods (i.e. greater than maximal lactate steady-state) interspersed with periods of recovery (i.e. light exercise or complete rest) within a training session, it is known as high-intensity interval training (HIIT)⁴⁷. HIIT is associated with physiological adaptations that increase function and performance. Maximal oxygen consumption ($\text{VO}_{2\text{max}}$) and ventilatory thresholds are examples of important physiological adaptations related to endurance performance that are improved by HIIT^{22,48}. Henceforth, given the importance of HIIT for endurance athletes, and its relatively small training volume, it can be argued that this type of training is an important research focus for improving overall athlete endurance performance.

A growing body of literature has assessed the effects of ambient temperature (T_A ; also referred to frequently as environmental temperature) on endurance exercise acutely^{2,7,26,49} and in the context of a training intervention^{38,50,51}. This research has allowed for the development of several methods to improve endurance performance, especially during particular thermal stresses. However, studies investigating T_A and exercise predominantly focus on steady-state exercise at submaximal intensities^{2,7,52,53}, or single bouts of high-intensity exercise^{26,27}. To date, little research has focused on T_A 's acute effects on high-intensity interval sessions or its effects in a training context^{54,55}, especially with endurance athletes. As such, the purpose of the following literature review is to provide relevant background information on HIIT (Section 2.2) and thermoregulation in endurance athletes (Section 2.3), as well as explore the extent T_A potentially affects HIIT performed by this population (Section 2.4).

2.2 High-Intensity Interval Training

High-intensity interval training has been utilized for over a century and is an effective method for improving athletic performance^{25,47,56}. In order to be effective high-intensity intervals should stress the physiological systems most associated with improving the desired performance outcome⁵⁷. To achieve this outcome the intensity and duration of the work and rest intervals for interval sessions are manipulated (Section 2.2.3)⁵⁷. Given the importance of the aerobic system in determining endurance sport performance^{18,58,59}, high-intensity interval sessions prescribed for endurance athletes often focus on improving this system^{23,47,57}. Therefore, the primary focus of this review will be aerobic HIIT utilized with the intention of increasing endurance performance.

Currently a plethora of literature has examined HIIT as a modality to induce physiological adaptations that improve function and performance^{48,56}. These studies have examined HIIT with an assortment of populations including sedentary, diseased, recreationally fit, and well-trained individuals. In the current review we focus on research conducted on well-trained endurance athletes. These individuals are characterized by their notable ability to acutely perform high levels of work for extended periods of time¹⁸. This performance ability is associated with a number of physiological attributes not observed in other populations (e.g. high $\text{VO}_{2\text{max}}$, lactate thresholds, and economy of motion)^{18,19}.

2.2.1 Acute Responses During High-Intensity Intervals

It has been argued that in order for high-intensity intervals to be effective at stimulating aerobic adaptations in endurance athletes, work interval intensities should be higher than the power output (or run velocity) associated with the maximal lactate steady-state⁴⁷. Whereby, during sessions a pattern of increases and decreases in metabolic rate and associated physiological responses occur that are characteristic to HIIT (Figure 2.1)⁶⁰⁻⁶². Cipryan et al.⁶¹ compared the physiological

response of highly-trained endurance athletes during high-intensity intervals (4x 3 minutes at 100% velocity at $\text{VO}_{2\text{max}}$) to that of matched workload and duration steady-state exercise. These researchers demonstrated higher peak values for heart rate (HR), VO_2 , and blood lactate during the high-intensity interval session, compared with steady-state work⁶¹. Stepto et al.⁶² demonstrated similar trends for greater HR, VO_2 , and blood lactate during work intervals of an aerobic high-intensity interval protocol (8x 5 minutes at 82.5% of peak power output; PPO). These researchers also observed decreases in muscle glycogen and muscle pH, and increases in muscle lactate and muscle temperature⁶². Given the high exercise intensity and corresponding physiological responses achievable during high-intensity intervals, it can be assumed that as maximal cardiac output and stroke volume⁶³ is approached, large motor units are recruited⁶⁴, muscle blood flow increases²⁶, and body temperature is elevated¹⁴. These acute responses of the oxygen transport and utilization systems during high-intensity intervals are believed to stimulate much of the physiological adaptations responsible for increasing endurance performance^{23,47,57}.

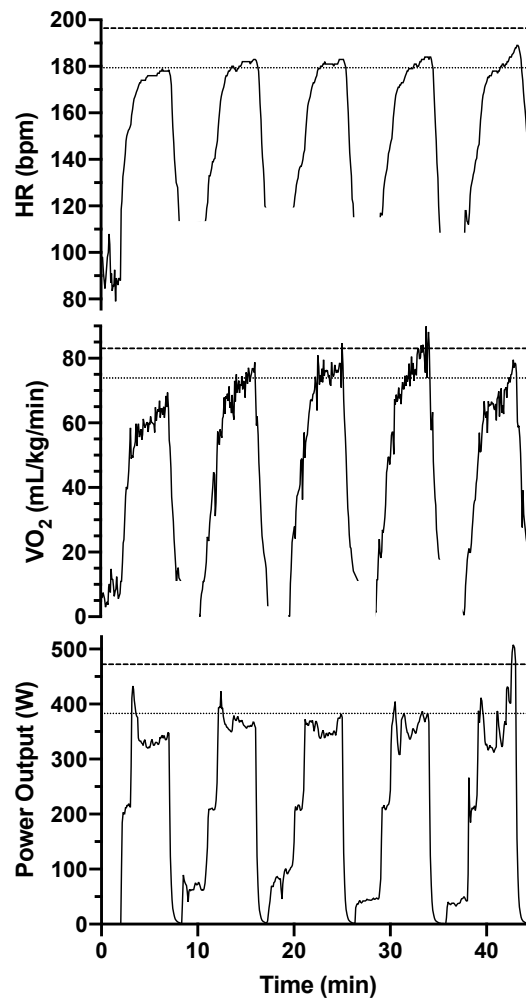


Figure 2.1 Heart rate (HR), oxygen consumption (VO₂), and power output for a single high-intensity interval session (5 x 4 minute work intervals with 5 minutes of rest between). Prior to each work interval the participant rode at 50% their peak power output for 1 minute. The session was self-paced as a maximal effort with the intention of achieving the highest average power output possible across all work intervals. Bold dashed lines represent maximal values from a graded exercise test for the individual. Fine dashed lines represent 90% of maximal values.

2.2.2 Physiological and Performance Outcomes of HIIT

High-intensity interval training offers substantial benefits for endurance athletes in terms of performance and physiological improvements^{25,56}. Improved endurance performance is typically related to enhancements in athletes' VO_{2max}, 'threshold', and (or), efficiency^{25,59}. Physiological

adaptations stimulated by HIIT that improve endurance performance can be categorized as ‘central’ or ‘peripheral’, and generally refer to improvements at the cardiovascular or musculoskeletal levels, respectively. HIIT is noted for its ability to increase $\text{VO}_{2\text{max}}$, as is characterized by a meta-analysis of HIIT and $\text{VO}_{2\text{max}}$ improvements by Wen et al.⁵⁶. Improvements in $\text{VO}_{2\text{max}}$, as a result of HIIT, are thought to primarily occur as a result of improvements in central limiters (i.e. cardiac output)^{65,66}. Cardiac output during exercise at $\text{VO}_{2\text{max}}$ is a function of maximal HR and stroke volume. Since it is established that maximal HR cannot be increased with training⁶⁷, improvements to cardiac output (and therefore $\text{VO}_{2\text{max}}$) typically arise through improved stroke volume⁶⁸. Improvements in stroke volume can occur via cardiac remodeling^{69–71}, and increased venous return due to elevated blood volume⁷² (i.e. increased plasma⁷³ and red blood cell volume). Indeed, Mahjoub et al.⁷⁰ observed that cardiac remodeling and reduced mean arterial pressure coincided with an increase in $\text{VO}_{2\text{max}}$ and PPO in endurance-trained men after a 6-week (18 session) HIIT intervention. This demonstrates the important effects HIIT has on improving central adaptations in endurance athletes.

While $\text{VO}_{2\text{max}}$ is undoubtedly a key factor of endurance athlete exercise capacity, ‘threshold’ (i.e. the highest exercise intensity an individual can perform for a substantial duration) is a demonstrably better predictor of overall endurance performance^{58,59}. This component of endurance performance is strongly associated with peripheral aspects such as high proportions of type I muscle fibers⁷⁴ and high mitochondrial function and content⁵⁸. These elements of the skeletal muscle allow for an increase in fat oxidation during prolonged (i.e. aerobic) exercise⁷⁵, allowing for finite glycogen stores to be spared, possibly attenuating fatigue. Mitochondria are important to this process as they oxidize fat as a fuel source and produce adenosine triphosphate (ATP) aerobically (i.e. in the presence of oxygen). Currently, evidence for HIIT increasing skeletal

muscle mitochondrial adaptations primarily exists for untrained and recreationally fit individuals⁷⁶⁻⁷⁸. Additionally, in the case of mitochondrial content, evidence supports training intensity may not be as important as training volume⁷⁹. Kohn et al.⁸⁰ observed an increase in peak running velocity in trained endurance runners after 6 weeks of HIIT (12 sessions), yet no change in muscle oxidative capacity. However, the researchers did not observe an increase in VO_{2max} either, potentially suggesting the intervention did not provide an appropriate stimulus for aerobic adaptations in this population⁸⁰. While the effect HIIT has on mitochondrial adaptations in well-trained endurance athletes is still up for debate, it is likely to influence other peripheral adaptations that can improve performance⁸¹.

2.2.3 Time at or Near VO_{2max}

Research suggests that the amount of time spent at or near VO_{2max} during an interval session is an important measure for the effectiveness of the session to elicit an aerobic training response^{24,47,57}. Time at or near VO_{2max} during an interval session is typically manipulated by changing work and rest interval intensity and duration^{24,57}, but approaches to pacing have also been demonstrated to be important^{82,83}. High-intensity intervals prescribed for a long time at or near VO_{2max} often incorporate a relatively long work interval (i.e. 3-4 minutes) with similar rest (i.e. >3-4 minutes)⁵⁷. However, various studies have also demonstrated a long time at or near VO_{2max} utilizing intervals with shorter work and rest durations (i.e. 30-90 second work intervals and 15-180 second rests) clustered into series^{84,85}.

The suggestion that time at or near VO_{2max} during HIIT is an important stimulus for increasing endurance performance is primarily theoretical^{24,25,47,57} with few intervention studies⁸⁴ investigating this concept. Indeed, little is known about this measure during HIIT and its actual relationship with physiological and performance outcomes⁸⁴. Turnes et al.⁸⁴, in recreational

cyclists, compared two work matched HIIT interventions with different time at $\text{VO}_{2\text{max}}$ and observed greater increases in $\text{VO}_{2\text{max}}$ and lactate threshold for the group training at a higher time at $\text{VO}_{2\text{max}}$. However, this study did not observe a difference in performance outcomes (i.e. maximal aerobic power and critical power) between groups, nor was a correlation between time at $\text{VO}_{2\text{max}}$ during the HIIT intervention and improvements in $\text{VO}_{2\text{max}}$ demonstrated⁸⁴. Similarly, Denadai et al.⁸⁶ observed superior improvements in velocity at $\text{VO}_{2\text{max}}$ ($\text{vVO}_{2\text{max}}$), running economy, and 1500-meter run performance after 8 HIIT sessions at 100% $\text{vVO}_{2\text{max}}$, compared to 95% $\text{vVO}_{2\text{max}}$. Yet, they observed similar improvements in lactate threshold and 5 km run time⁸⁶. Olsen et al.⁸⁷ compared two HIIT interventions performed at 92% $\text{vVO}_{2\text{max}}$ and 100% $\text{vVO}_{2\text{max}}$ by trained army recruits and observed similar improvements in $\text{VO}_{2\text{max}}$, ventilation threshold, and 3200-meter run time between groups. As a caveat, whilst Olsen et al.⁸⁷ and Denadai et al.⁸⁶ performed HIIT at different running intensities relative to $\text{vVO}_{2\text{max}}$, but it is not certain if these different HIIT protocols consisted of substantially different time at or near $\text{VO}_{2\text{max}}$ during the interval sessions, as this measure was not recorded. However, these studies do speak to the overall complexity of aerobic interval intensity and subsequent HIIT outcomes.

2.3 Thermoregulation During Exercise

Temperature and exercise have a complex relationship. At the most basic level temperature has an effect on the rate of biological chemical reactions (i.e. metabolism)⁸⁸. An increased temperature can improve enzyme kinetics and therefore cell function⁸⁸. Indeed, endotherms (e.g. humans) have evolved intrinsic processes of thermoregulation in order to maintain a body temperature above that of the temperature often found in their immediate environment. However, when body temperature is too hot enzymes can denature and their ability to catalyze cellular reactions decreases or ceases. During exercise, approximately 75% of the energy released from metabolized fuel sources in the

working muscle is lost to the local tissue in the form of heat⁸⁹. This metabolic heat production is greatly affected by exercise intensity and duration^{14,15}. In order to dissipate excess heat energy from muscle metabolism, and avoid detrimental increases in body temperature, blood flow is redirected from the body core to the skin facilitating heat loss to the environment⁹⁰.

Heat exchange between exercising individuals and the environment occurs through four major pathways during performance: convection, conduction, evaporation, and radiation. Convection and conduction involve transferring heat between matter (solids or fluids) that come in contact with the athlete. The rate of this heat transfer is relative to the type of fluid (water has a conductivity 27 times greater than air) and the speed of the fluid as it passes across the skin⁹¹. These factors are important to swimmers and triathletes who train and compete in water as well as runners and cyclists who, while potentially exercising at the same intensity, experience different rates of airflow across their bodies. Evaporative heat exchange involves heat loss to the environment via a liquid to vapor phase change of water. This method of heat exchange offers the advantage that it is effective even when T_A is too high to elicit an effective gradient for heat loss, yet involves an increased risk for dehydration and cardiovascular strain⁹². Radiative heat exchange for the endurance athlete mostly involves solar energy, either from direct sunlight (e.g. a sunny day) or indirectly from reflection (e.g. from snow) or from objects warmed by the sun (e.g. hot pavement). However, heat loss via radiation from the skin should not be overlooked.

Heat energy moves down a gradient (i.e. from hot to cold), and the rate of this transfer increases as the gradient increases. Therefore, the environmental temperature (T_A) that exercise is performed in can have significant effects on exercise performance^{2,6,7,49}. However, the magnitude in which T_A is an additional stress during exercise is determined by its ability to affect an organism's internal heat balance⁹¹. Whereby, the heat balance of an organism is determined by factoring metabolic

heat production (\pm external work performed) and heat exchange to the environment (Equation 2.1)⁹². These factors are influenced by both behavioral and physiological responses of the organism.

$$S = M \pm W \pm R \pm C \pm K - E$$

Equation 2.1 Equation for body heat balance. S= rate of body heat storage, M= rate of metabolic heat production, W= external work performed, R= radiative heat exchange, C= convective heat exchange, K= conductive heat exchange, E= evaporative heat loss.

2.3.1 Thermoregulation and the Endurance Athlete

Endurance athlete thermoregulation is altered due to their physiological^{20,93–95} and anthropometric⁹⁶ characteristics. Their ability to perform a high capacity of work for an extended period^{18,19} of time leads to increased metabolic heat production^{14,15} and high need for heat removal. Fortunately, the high cardiovascular ability^{95,97}, improved sweat response⁹⁸, small body sizes⁹⁹, large skin surface to volume ratios, and low percentage of body fat⁹⁶ of endurance athletes allow for an increased capacity to lose heat to their environment during exercise, compared to sedentary or untrained individuals. Conversely, the anthropometric characteristics noted here may give cause for concern for endurance athletes located in cold environments, particularly when not exercising (e.g. just prior to cross country skiing or cyclocross races)¹⁰⁰.

Other than physiological responses, organisms also regulate their heat balance via behavioral means. However, during training and competitive events pacing requirements can supersede thermoregulation needs and limit behavioral options for endurance athletes to influence their heat balance in terms of increasing or decreasing exercise intensity. For endurance athletes performing HIIT in the heat various pre-cooling and cooling techniques may provide additional thermal comfort and increase work interval performance¹⁰¹. In the case of endurance athletes performing

HIIT in cold conditions, the option of wearing warmer clothing to maintain heat balance is available¹⁰². However, caution must be given when performing high-intensity intervals in the cold, as the resulting post session fatigue and sweat drenched clothing is likely to affect the ability to maintain core body temperature as water (i.e. sweat) has a very high specific heat capacity¹⁰³.

2.4 Environmental Temperature and High-Intensity Interval Training

The following section explores the effects T_A has on HIIT in endurance athletes. This examination is divided into three subcategories: the acute effects T_A has on high-intensity intervals (Section 2.4.1), repeat exposure to T_A during HIIT (Section 2.4.2), and the effect T_A has HIIT outcomes (i.e. performance and coinciding physiology; Section 2.4.3). Currently, literature investigating the effects of T_A on HIIT and subsequent outcomes is sparse, particularly in endurance athletes⁵⁵. Inferences regarding the effects of T_A on high-intensity intervals are primarily based on previous literature investigating the effects of T_A during submaximal steady-state exercise, and (or) single bouts of maximal exercise. It is then deduced that acute physiological differences during high-intensity intervals at different T_A would affect performance and physiological outcomes of HIIT. Given this level of speculation, we are sometimes left with contradictory, yet similarly plausible hypotheses for the effects T_A has on HIIT. For this reason, we explore studies specific to the topic in more depth.

2.4.1 Acute Effects of Environmental Temperature on High-Intensity Intervals

2.4.1.1 Environmental Temperature and Optimal Performance

Endurance exercise performance is reduced in both hot^{1,2} and cold^{2,53} environmental conditions. This results in an inverse U-shaped relationship and implies that there is an optimal T_A in which to perform an endurance task². Researchers have sought to determine this T_A employing both

retrospective analyses of competitive endurance events^{5,6,104}, and laboratory studies^{2,7,102,105,106}. Utilizing these different methods it has been demonstrated that optimal T_A for endurance performance is within a range of 10-17°C^{2,5-7}. Hence, it should not be surprising T_A in this range are often utilized as “control” or “thermoneutral” conditions in laboratory studies investigating thermoregulation during exercise, particularly in training interventions^{38,55}.

To date, much of the current literature examining the influence of T_A on endurance performance has investigated steady-state efforts at or around threshold^{2,7,32,49,52,53,105}, with little to no studies investigating the effect of a range of T_A on high-intensity exercise. As mentioned in Section 2.1, elite endurance athletes typically spend little time exercising at threshold during training^{10,11}, and certain endurance competition¹² (e.g. road cycling events). Indeed, such athletes typically spend the large majority of training at low intensities (i.e. below lactate threshold) coupled with short and intermittent bouts of high-intensity exercise or training¹¹. Additionally, variations in an athlete’s velocity over the course of a high-intensity interval session would notably change convective heat loss, and therefore skin temperature (T_{sk}) and T_{sk} to core temperature (T_c) gradient. As a result, the effects T_A has on high-intensity interval performance may differ to what has been observed in prolonged steady-state exercise. Henceforth, further research investigating the influence of T_A on performance and physiological responses at intensities observed in endurance training is desirable.

2.4.1.2 High-Intensity Intervals in the Heat

The negative effect of heat on endurance performance is well-documented^{2,26,107}. As alluded to in Section 2.3, a number of environmental (e.g. humidity¹⁰⁸, rate of convection⁸, solar radiation¹⁰⁹) and individual (e.g. anthropometry⁹⁹, fitness⁹⁷, acclimation¹) factors affect the specific reduction in performance for a given scenario, and should be considered when applying laboratory findings

to real-world applications. Indeed, laboratory time to exhaustion tests have demonstrated ~45% reductions in test duration in heat compared to cooler conditions^{2,110}. Similarly, a review of environmental influences on self-paced cycling exercise reported an average reduction in mean power of 15% in hot conditions ($>30^{\circ}\text{C}$), compared with cooler controls¹⁰⁷. However, the literature is sparse when it comes to research investigating the acute effects of heat on high-intensity interval training.

Increased T_A during exercise hinders heat loss by decreasing the temperature gradient between the skin and the environment, subsequently increasing T_C ². Increases in T_{Sk} , or the combination of high T_{Sk} and high T_C (i.e. $T_C:T_{Sk}$ gradient), correspond with reductions in $\text{VO}_{2\text{max}}$ ¹¹¹, and consequently reduced performance^{26,112,113}. Indeed, Périard and Racinais¹¹² demonstrated a reduction in peak oxygen consumption ($\text{VO}_{2\text{peak}}$) during self-paced cycling exercise in hot (35°C) and cool (18°C) conditions. However, reductions in $\text{VO}_{2\text{peak}}$ and relative $\%\text{VO}_{2\text{peak}}$ were greater in the heat and corresponded with greater reductions in power output¹¹². This impairment in performance is argued to be primarily due to cardiovascular limitations and subsequent attenuation of oxygen delivery to exercising muscles¹¹³. In prolonged self-paced steady-state exercise in the heat, reductions in performance and VO_2 coincide with decreased mean arterial pressure, stroke volume, and an increase in HR compared to thermoneutral conditions⁵². This increase in cardiovascular strain was believed to be caused by an increase in skin blood flow as the body attempts to thermoregulate under increased T_A ⁵². Decreases in performance and similar evidence for cardiovascular strain have been observed during maximal exercise (i.e. $\text{VO}_{2\text{max}}$ elicited in 3-5 minutes) during hyperthermia^{26,27}. However, during exercise at this intensity and duration, skin blood flow was not different to thermoneutral conditions²⁶. Nevertheless, limitations on blood flow likely provide the greatest limitation to endurance performance¹¹³ and pacing⁵² in the heat. In

addition to increasing skin blood flow, sweating and hyperthermia-induced hyperventilation^{114,115} (i.e. human panting) occur during exercise in hot conditions to further facilitate the removal of heat waste from the body during exercise. Paradoxically, these responses also correspond with increased cardiovascular strain and (or) risk of dehydration for a given workload and can be detrimental to endurance exercise performance^{27,92,112}.

Based on previous research examining maximal high-intensity exercise^{26,27}, prolonged steady-state^{2,105,107}, and intermittent¹⁶ exercise in the heat, it can be hypothesized high-intensity interval session performance (i.e. power output) would be negatively affected under hot conditions. Of note, similarities in performance and physiological responses between interval sessions and steady-state exercise may correlate with the duration of heat exposure. It can be argued the initial effect heat has on high-intensity intermittent exercise is dependent primarily on the ratio of the energy systems utilized during the session. Whereby, the lower the aerobic contribution¹³ (i.e. reliant on the cardiovascular system), the less likely heat will decrease performance^{13,116–119}. Indeed, participants performing repeat anaerobic efforts (e.g. 2x 30-second cycling sprints separated by 4 minutes of recovery) produced greater mean power output in hot conditions versus control conditions^{116,117}. Yet, despite similar durations of heat exposure, hyperthermic individuals conducting a single maximal aerobic effort (time to exhaustion 5-10 minutes) have demonstrated decreased performance (and corresponding $\text{VO}_{2\text{max}}$)^{26,27}. However, this relationship between energy utilization and the performance capacity of high-intensity exercise in the heat appears to subside after prolonged exercise in the hot conditions¹⁶. Indeed, Drust et al.¹⁶ demonstrated that power output during 5x 15 second sprint efforts decreased following 40 minutes of intermittent exercise in the heat (40°C), when compared to a control condition (~20°C). These authors suggested, based on a lack of evidence for increases in fatigue related metabolites, this impaired

performance may have resulted from high T_C influencing function of the central nervous system¹⁶. This implies the mechanisms for fatigue in the heat are likely to differ between early and later stages of a high-intensity interval session.

Considering endurance athletes would mostly utilize aerobic high-intensity intervals, it is likely performance of their intervals in the heat would be associated with increased cardiovascular strain and subsequent decreases in acute exercise capacity. Cardiovascular strain is hypothesized to be induced by increased T_{sk} in the heat^{120,121}, resulting in increased skin blood flow and (or) increased tachycardia relative to exercise intensity^{26,52,122,123}. For a given interval session in hot conditions, increases in HR, and decreases in stroke volume, cardiac output, muscle blood flow, and VO_2 could be expected. Additionally, attenuated VO_2 during the session would likely negatively affect the time at or near VO_{2max} for a given interval protocol, and potentially affect HIIT outcomes (discussed further in Section 2.4.3.2). Currently, further research is needed to investigate cardiovascular and thermoregulatory responses during aerobic high-intensity intervals in the heat.

2.4.1.3 High-Intensity Intervals in Cold

Extreme cold T_A negatively effects exercise capacity^{2,3,53,103}. Galloway & Maughan², demonstrated a 13% decrease in cycling time to exhaustion in 4°C (~81 minutes at 70% VO_{2max}) versus 11°C (~94 minutes), and similar time to exhaustion as 21°C (~81 minutes). Similarly, Ferguson et al.⁵³ demonstrated that mild hypothermia (i.e. reduced T_C) in 0°C decreased time-trial power output by 5%, in trained cyclists, compared to a thermoneutral control (23°C). Both of these studies demonstrate aerobic exercise performance can be impaired by cold conditions.

The detrimental relationship between cold T_A and prolonged exercise is likely attenuated by a number of factors. For example, Sandsund et al.¹⁰² demonstrated optimal endurance performance

at -4 and 1°C while wearing cross-country skiing clothing. Parkin et al.¹⁰⁵, in a similar study design to Galloway & Maughan², demonstrated a longer time to exhaustion in 3°C compared to 20°C and 40°C. The discrepancies in time to exhaustion results between Parkin et al.¹⁰⁵ and Galloway & Maughan² are unclear, but may have been due to ‘endurance-trained’ individuals (i.e. high metabolism and heat production during exercise) in the former¹⁰⁵ study and ‘healthy’ participants in the latter². Additionally, a warm-up prior to exercising in the cold could benefit performance¹²⁴. Indeed, Spitz et al.¹²⁵ observed no difference in time-trial performance between temperate (24°C) and cold (5°C) conditions after a standardized 15-minute warm-up. Contrary to this, Morrissey et al.¹²⁶ concluded that avoiding a warm-up prior to exercising in the cold (0°C) might improve endurance performance (i.e. lactate threshold and time to exhaustion at 120% at PPO). These contradictory findings create doubt for the value of a warm-up prior to exercise in the cold. However, given the ecological validity of a time-trial (i.e. Spitz et al.¹²⁵) versus a lactate threshold test (which would intrinsically include a warm-up; Morrissey et al.¹²⁶), and evidence demonstrating time-trial performance is reduced during mild hypothermia (i.e. reduced T_C)⁵³, a warm-up prior to exercise in the cold is likely valuable.

Reductions in exercise capacity in the cold are likely due to multiple physiological factors. Contrary to the relationship between HR and steady-state workload in the heat, when exercising in the cold HR can actually decrease for a given workload¹²⁷. Whereby, reduced blood flow to the skin (via vasoconstriction) increases venous return to the heart and therefore increases stroke volume¹²⁷. Muscle metabolism and (or) neuromuscular limiters are likely the primary causes of reduced performance in the cold¹²⁸. Indeed, the ability of a skeletal muscle to produce force decreases as its temperature decreases⁴. This outcome is most likely caused by a decrease in muscle velocity. Indeed, while Ferguson et al.⁵³ observed decreased time-trial power output in mild

hypothermia, a reduction in cadence was also present. An additional skeletomuscular influence on aerobic performance in the cold ($< 0^{\circ}\text{C}$), compared to temperate conditions, is the increase of carbohydrate¹²⁹ and decrease of lipid oxidation for energy sources¹³⁰. These physiological responses, along with others, may contribute to reduced performance during high-intensity interval sessions.

2.4.2 Repeat Exposure to Environmental Temperature during HIIT

2.4.2.1 Repeat Exposure to Environmental Stress as an Ergogenic Aid

Exercise and T_A (i.e. hot and cold) are both stressors on the body. Repeated exposure to these stressors, at suitable doses and frequency, stimulate physiological adaptations to aid in the body's response to future stress⁹¹. Interestingly, various physiological adaptations that occur as a response to training also occur after repeated exposures to hot³⁶ or cold T_A ^{131,132}. These adaptations to thermal stress can occur after natural (acclimatization) or artificial (acclimation) exposures to T_A stress. For the purposes of this review adaptations to thermal stress will be referred to generally as 'acclimation' unless the differentiation between natural and artificial exposure is deemed important.

The addition of an environmental stressor to an endurance athlete's training scheme for the purpose of increasing performance is not a new concept, as can be seen with multiple decades of hypoxic training employed for this purpose¹³³. The utilization of an environmental stressor as an ergogenic aid can be categorized by the primary desired outcome. Either, 1) the intent is to acclimate an athlete to an environmental condition they will be exposed to during competition, and therefore decrease the negative effects of that environmental stress; or 2) environmental stress is purposely added to training stress for the desired outcome of increasing performance under normal environmental conditions (e.g. temperate and normobaric). Currently, the literature appears to

indicate better evidence for the former purpose than the latter^{37,134,135}. Additionally, of note, there is an increasing body of literature investigating both cross-tolerance effects of adaptations to environmental stress (e.g. heat acclimation before competing in hypoxia)^{136–138}, and incorporating multiple environmental stressors into a single training regimen^{55,139}.

2.4.2.2 Heat Acclimation and HIIT

As mentioned in Section 2.4.1.2, aerobic exercise capacity is reduced in hot conditions. Repeated exposure to heat can stimulate physiological adaptations to improve exercise performance in hot conditions^{28,44}. These adaptations include, but are not limited to: increased exercise performance and capacity, reduced T_C (at rest and during exercise), improved sweat response⁹⁸, reduced skin temperature, improved skin blood flow, and improved cardiovascular ability³⁸ (e.g. reduced HR and increased stroke volume)⁴⁴. In order to stimulate these adaptations thermal impulses (i.e. changes in body temperature with respect to duration) sufficient in load and frequency are required. However, given a uniform thermal load, adaptations to heat stress progress over unique time courses, with key cardiovascular (e.g. plasma volume and reduced HR) and body temperature (i.e. T_C and T_{Sk}) adaptations reaching peak levels sooner than those of sweat response and exercise capacity improvements¹⁴⁰.

Exposures to heat with the intent of acclimation are most likely beneficial if they are conducted under conditions similar to what the individual is expected to perform in²⁹. For the endurance athlete, this implies heat acclimation (HA) with the intent of improving exercise performance in the heat (e.g. competitions in hot environments) should consist of heat exposure while exercising (versus passive exposure). Heat exposure while performing aerobic exercise offers the additional benefit of increasing thermal impulse for a given duration in a hot environment, as increases in T_C would occur sooner than if the individual was inactive (i.e. passive HA). Indeed, HA protocols

incorporating a range of exercise intensities from low to high have been shown to stimulate physiological adaptations to heat^{50,141,142}. However, given the importance of HIIT to an endurance athlete, and its small relative training volume, it is crucial to consider the effects that heat exposure during HIIT could have on performance outcomes⁵⁰. Incorporating heat exposure with HIIT could be advantageous as it would allow for HA to conform better to the ecological needs of training and could allow for an increased thermal impulse per acclimation session⁵⁰.

Sessions for HA protocols are typically conducted on consecutive days or less than two days apart⁴⁴. However, while HIIT on consecutive days (i.e. block training) has been demonstrated to improve performance in well-trained endurance athletes¹⁴³, HIIT sessions are generally scheduled 2 or more days apart to allow for sufficient recovery and avoid non-functional overreaching^{45,46}. The difference in duration between HA and HIIT bouts is a potential obstacle for combining these two modalities. Indeed, a primary concern with the addition of heat to training at high intensities is that it would elevate internal training load and increase the propensity for non-functional overreaching⁵⁰. Schmit et al.⁵⁰ demonstrated a decrease in 35°C time-trial performance after 5 consecutive days of high-intensity exercise in the heat that was not observed in either a low intensity HA or control group (temperate conditions and normal training). Similarly, Reeve et al.¹⁴⁴ demonstrated decreased time to exhaustion (i.e. exercise capacity) with well-trained endurance athletes performing cycling exercise at 35°C after 5 consecutive days of HIIT in the heat. These studies suggest performing high-intensity exercise during consecutive day heat exposure actually inhibits subsequent performance outcomes in the heat. A potential solution for incorporating HIIT into a HA protocol might be to allow for greater rest between bouts (i.e. at least 48 hours). However, intermittent-day bouts of HIIT that allow for enough recovery from the exercise training load may not allow for sufficient thermal impulse to stimulate the necessary adaptations to heat⁴⁴.

Yet, even if intermittent-day bouts of hot HIIT cannot provide enough stimulation for substantial HA to manifest, hot HIIT could still be utilized for the purpose of preventing HA decay after the conclusion of a traditional HA protocol^{145,146}. Whereby, maintaining heat acclimation takes less exposure to heat than generating it¹⁴⁵. Currently, more research examining HIIT in HA protocols is needed.

2.4.2.3 Repeat Cold Exposures and HIIT

Repeat exposures to cold is associated with cold acclimation^{51,147}. In contrast to the general pattern of HA¹⁴⁰, cold acclimation has multiple patterns of adaptation¹⁴⁸. Additionally, exercising during cold exposures is suggested to attenuate the acclimation process, as the increase in T_C that occurs during exercise prevents the required whole-body T_C drop¹⁴⁹. Indeed, Launay et al.¹⁵⁰ observed no thermoregulatory changes during rest at 1°C after 4 weeks of HIIT (20 sessions) in 1°C compared to a resting cold exposure group that demonstrated a drop in resting T_C in 1°C. Thus, repeated cold exposure during HIIT would have little effect on thermoregulatory responses. However, Lorenzo et al.³⁸ demonstrated an increase in T_{sk} and skin blood flow, and a decrease in $T_C:T_{sk}$ gradient during exercise after 10-days of submaximal exercise (2 x 45 minutes at 50% PPO) in 10°C. Similarly, Geurts et al.⁵¹ observed no differences in local cold acclimation of the hand (10 sessions of cold water immersion), when simultaneously exercising or remaining at rest. These findings support the conclusion that localized thermoregulatory adaptations could occur after performing HIIT in cold conditions. In addition, these adaptations could be beneficial to endurance athletes under certain circumstances¹⁵¹. Increased T_{sk} in conjunction with other cold adaptations may be useful for increasing athlete tolerance¹⁵² and performance¹⁵³ while exercising in the cold. Indeed, repeated bouts of cool intervals could provide beneficial adaptations for unacclimatized endurance

athletes traveling to cold environments¹⁵¹. Currently, more research is needed on the effects of repeat exposures to cold during HIIT.

2.4.2.4 Repeat Bout Effects of Environmental Temperature on High-Intensity Intervals

Environmental temperature has an acute effect on cardiorespiratory and thermoregulatory responses during high-intensity and prolonged steady-state exercise^{1-3,49,52}. While it can be hypothesized that acute physiological responses occurring during high-intensity interval sessions contrast for different T_A , the benefits of HIIT manifest in trained populations after repeated bouts. It can be argued that differences in acute cardiorespiratory stress during high-intensity intervals, observed at different T_A , could potentially change after repeat sessions at that given T_A . This change in physiological stress over the course of a HIIT intervention could potentially diminish or enhance training outcomes. Currently, to our knowledge there is little or no literature that has investigated this topic. Understanding how cardiorespiratory physiology and thermoregulation during high-intensity intervals change after repeated exposures to different T_A could give beneficial insight into optimizing HIIT prescription.

2.4.3 Effects of Environmental Temperature on HIIT Outcomes

2.4.3.1 Potential Benefits of Performing HIIT in Optimal Environmental Temperatures

As described in Section 2.4.1.2, high-intensity intervals performed in the heat would likely reduce time at or near VO_{2max} , caused by a reduction in cardiac output and a subsequent reduction in muscle blood flow. If the duration of time at or near VO_{2max} during high-intensity intervals directly relates to improved endurance performance and physiological outcomes (e.g. increased VO_{2max} and lactate threshold)^{24,84}, then HIIT in hot T_A would likely be detrimental to endurance performance. Indeed, Gifford et al.¹⁵⁴ demonstrated that VO_{2max} in trained endurance athletes was limited by O_2 supply to the working muscles (i.e. cardiac output). This implies that in order to

stimulate peripheral aerobic adaptations in trained endurance athletes during HIIT, blood flow to the working muscles should be maximised. Performing HIIT in cooler environmental conditions would increase $T_C:T_{sk}$ gradient, reducing skin blood flow⁵², increasing venous return, and enhance the proportion of cardiac output¹²⁷ delivered to the active muscle²⁶. Reinforcing this premise are results indicating increased muscle blood flow to the exercising muscles during single-leg cycling training³⁰ stimulate a superior oxidative potential and metabolic profile of the skeletal muscle compared to double-leg cycling³¹. Conversely, it could be argued that T_A has little influence on HIIT outcomes. Increases in cardiovascular stress and T_C correspond with increased exercise intensity during HIIT¹¹² and are likely to maintain similar trends in different T_A conditions. These physiological responses possibly stimulate adaptations to HIIT as much or more than time at or near VO_{2max} , facilitating similar training outcomes.

2.4.3.2 Potential Benefits of Performing HIIT in Hot Environmental Temperatures

Heat acclimation as an ergogenic aid in temperate conditions has been a topic of much research^{35,36} and debate^{37,135}. While it is debatable whether hot HIIT could provide sufficient thermal impulse to stimulate HA, while at the same time not inducing overreach (discussed above in Section 2.4.2.2), this mechanism as a potential influence on endurance performance is still worth exploring. Indeed, adaptations that occur after repeated heat exposure such as: increased red-cell volume^{40,41}, increased plasma volume⁴², enhanced skin blood flow response³⁸, decreased T_C ⁴³ (during rest and submaximal exercise), decreased skin temperature (T_{sk})³⁸, increased $T_C:T_{sk}$ gradient³⁸, and improved sweat response⁹⁸, could all provide ergogenic benefits in during exercise in temperate conditions.

To date, experimental evidence for HA as an ergogenic aid in temperate conditions has typically been demonstrated in studies without control groups^{1,39,155–157}, or in matched group experiments

where training intensities were determined from graded exercise tests in control T_A conditions (i.e. relative training workloads between groups)^{38,55,158}. Under these circumstances, cardiovascular load would be higher during training in the heat compared to training in cooler control conditions^{2,26}. Indeed, after a 3-week relative workload training intervention (9 total sessions: 6 HIIT and 3 steady-state) in either hot or cool conditions, McCleave et al.⁵⁵ observed superior 3-km run performance improvements after training in the heat. Similarly, Souza-Silva et al.⁵⁴ demonstrated increased PPO after a 4-week (12 sessions) relative workload HIIT intervention in hot conditions (35°C), compared to a control group performing HIIT in temperate conditions (22°C). These studies demonstrate that hot HIIT performed at relative workloads have similar performance outcomes to HA studies utilizing relative workloads. Contrary to these findings, HA studies where cardiovascular strain was similar between hot and cool training conditions have observed no difference in performance outcomes in temperate T_A ^{35,159}. Collectively, these results provide evidence that additional cardiovascular load caused by training in the heat is potentially a key component of stimulating adaptations that increase endurance performance in temperate conditions. Unfortunately, to our knowledge no HIIT intervention study has utilized hot and cool T_A and attempted to match cardiovascular strain between conditions. Self-paced intervals performed at a maximal effort may provide similar cardiovascular load between hot and cool interventions, and subsequently result in similar performance outcomes.

Previous research has demonstrated HIIT can increase mitochondrial content^{66,77}, which is an important factor in increasing endurance performance⁷⁴. However, during high-intensity exercise muscle acidosis increases. Paradoxically there is evidence to claim mitochondrial biogenesis is attenuated in lower pH environments¹⁶⁰. Performing high-intensity intervals in the heat could reduce the negative effect muscle acidosis has on mitochondrial biogenesis. Whereby, when

humans perform intense and (or) prolonged exercise in the heat they will commence hyperthermia-induced hyperventilation (i.e. human panting) to assist thermoregulation^{114,115}. This increase in ventilation results in an increase in gas exchange, and consequently a reduction in arterial CO₂ pressure and increase in blood pH (i.e. blood alkalosis)^{114,115}. To our knowledge no research has investigated the effects of hot HIIT and (or) reduced blood pH on mitochondrial function and biogenesis. However, a number of studies have demonstrated relationships between mitochondria and temperature^{161,162}, including Hafen et al.¹⁶¹, which demonstrated repeated deep tissue heating of skeletal muscle improved mitochondrial respiratory capacity in healthy sedentary humans. Additionally, declines in mitochondrial oxidative phosphorylation efficiency associated with hyperthermia are attenuated in healthy men after HIIT¹⁶². These studies illustrate the relationship between heat, mitochondria, and HIIT are unclear, yet worthy of further research, especially in terms of improving endurance athlete performance.

2.5 Conclusions

High-intensity interval training is an important component of the overall training scheme for endurance athletes. The influence T_A has on exercise performance and physiology is well documented. Therefore, high-intensity interval cardiorespiration, thermoregulation, and performance would likely be affected by T_A , and could influence HIIT outcomes (e.g. endurance performance) after repeated exposures. However, to date, the literature investigating the acute effects of T_A on high-intensity intervals is sparse. Current, hypotheses regarding the effects of T_A on high-intensity intervals are primarily based on previous literature investigating T_A effects during submaximal steady-state exercise, and (or) single bouts of maximal exercise. It can be reasoned that hot conditions would decrease cardiac output, muscle blood flow, VO₂, and performance during high-intensity intervals, whilst training in cooler conditions (i.e. 10-17°C)

would manifest a comparative increase in these measures. It can potentially be asserted these acute physiological differences during high-intensity intervals at different T_A would affect performance and physiological outcomes of HIIT. However, it is difficult to draw strong conclusions on the outcomes of HIIT in different T_A as the current literature on this topic is also limited. Current hypotheses regarding the effects of T_A on HIIT are primarily based on HA research and HIIT studies with different acute physiological responses (i.e. time at or near VO_{2max}) between interventions. HA research utilizing high-intensity intervals during heat exposure has provided evidence that consecutive day HIIT in the heat can decrease performance, potentially due to non-functional overreach. Additionally, HA research highlights the importance of considering differences in cardiovascular load between T_A conditions when assessing and predicting training intervention outcomes. HIIT research comparing interventions with different time at or near VO_{2max} or training intensities has highlighted these factors can induce different performance and physiological outcomes. However, establishing a clear pattern between different stimuluses during HIIT and subsequent outcomes is currently difficult. Additionally, assuming similar physiological differences occur between thermoneutral interventions with different protocols and interventions at different T_A (e.g. time at or near VO_{2max} reduced from work interval differences versus reduced by the effects of heat) would result in the necessarily similar outcomes is problematic. In conclusion, given the importance of HIIT, the established effects T_A has on exercise, and the current deficit of conclusive literature describing the interaction of these two elements, further research is warranted in this area of study.

3 CHAPTER THREE:

STUDY 1

EFFECT OF ENVIRONMENTAL TEMPERATURE ON HIGH-INTENSITY INTERVALS IN WELL-TRAINED CYCLISTS

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(Appendix A)

3.1 Abstract

Purpose: To examine the effect of environmental temperature (T_A) on performance and physiological responses (e.g. body temperature and cardiopulmonary measures) during a high-intensity aerobic interval session. It was hypothesized that power output would be highest in the 13°C condition and lower in the 5°C, 22°C, and 35°C conditions. **Methods:** Eleven well-trained cyclists randomly completed 4 interval sessions at 5°C, 13°C, 22°C, and 35°C ($55 \pm 13\%$ RH), each involving 5 x 4-minute intervals interspersed with 5 minutes of recovery. During the intervals, power output, core temperature (T_C), skin temperature, VO_2 , and heart rate (HR) were recorded. **Results:** Mean session power output for 13°C (366 ± 32 W) was not higher than 5°C (363 ± 32 W, $P = 1.00$, Hedges' $g = 0.085$), 22°C (364 ± 36 W, $P = 1.00$, $g = 0.061$), or 35°C (352 ± 31 W, $P = .129$, $g = 0.441$). The 5th interval of the 35°C condition had a lower power output compared with all other T_A . T_C was higher in 22°C compared with both 5°C and 13°C ($P = .001$). VO_2 was not significantly different across T_A ($P = .187$). HR was higher in the 4th and 5th intervals of 35°C compared with 5°C and 13°C. **Conclusions:** This study demonstrates that whilst mean power outputs for intervals are similar across T_A , hot T_A ($\geq 35^\circ\text{C}$) reduces interval power output later in a training session. In conclusion, well-trained cyclists performing maximal high-intensity aerobic intervals can achieve near optimal power output over a broader range of T_A than previous literature would indicate.

3.2 Introduction

Endurance exercise performance is reduced substantially in hot^{1,2} and cold^{2,3} environmental conditions. There is an inverse U-shaped relationship between endurance performance and environmental temperature (T_A) implying that there are optimal conditions in which to perform an endurance task^{2,6}. According to previous literature this temperature is in the range of 10 to 17 °C^{2,5-7} but is dependent on the mode of exercise, duration of exercise, and rate of convection⁸. Yet, much of the research quantifying optimal T_A for exercise has involved relatively steady-state tasks at or around lactate threshold^{2,5-7}. Paradoxically, elite endurance athletes typically spend very little time at these intensities during training¹¹. This is especially the case for elite/professional road cyclists¹². Indeed, such athletes typically spend the large majority of training at low intensities (i.e. below lactate threshold) coupled with short and intermittent bouts of high-intensity exercise or training (HIIT)¹⁶³. The influence of T_A on performance and physiological response to such exercise is not well understood.

Despite HIIT being a relatively small part of training volume¹¹, it can induce rapid and large adaptations important to endurance performance²¹. Such high-intensity exercise results in an increased metabolic rate¹³, high metabolic heat production¹⁴, possibly compromising performance. Indeed, Drust et al.¹⁶ demonstrated that power output during repeated sprint efforts performed following 40 minutes of intermittent exercise in the heat (40°C) were decreased when compared to the control condition (~20°C). Additionally, due to variations in an athlete's velocity, convective heat loss will also notably change throughout an interval training session. It is therefore plausible that the effects T_A has on high-intensity interval performance differ to what has been observed in prolonged steady-state exercise.

Previous literature examining endurance exercise under a range of T_A has not considered conditions that have an effect on cycling performance in a well-trained cyclist population. Galloway & Maughan² examined the time-to-exhaustion under a range of T_A , but of note, the mean maximal oxygen consumption (VO_{2max}) for these subjects (~ 56 mL/kg/min) was below what the literature would consider ‘well-trained’ endurance athletes^{18,19}. This is important since trained individuals can exercise at higher exercise intensities¹⁸ eliciting higher metabolic heat production¹⁴ than untrained individuals. In addition, the aforementioned studies examining the effects of T_A on endurance performance have not included important elements known to affect cycling performance; such as a standardized warm-up¹⁶⁴ and replication of realistic convective heat loss through high wind velocities^{8,17}.

From this synopsis it can be recognized that a better understanding of the effects T_A has during high-intensity intervals is needed. Therefore, the overall purpose of this study was to determine the effects of T_A on physiological responses (e.g. body temperature, and cardiopulmonary measures) and performance (i.e. power output) during intervals. To our knowledge this was the first investigation seeking to explore the effect a range of T_A has on high-intensity intervals. It was hypothesized that power output and VO_2 would decrease in the 5°C, 22°C, and 35°C conditions compared with the 13°C condition.

3.3 Methods

3.3.1 Participants

Eleven male cyclists (age: 34.1 ± 9.8 y, height: 181.3 ± 5.0 cm, mass: 75.78 ± 8.01 kg, sum of 8 skinfolds: 65.3 ± 19.1 mm, VO_{2max} : 69.62 ± 6.74 mL/kg/min) were recruited for this study (Appendix C). Participants were classified as well-trained based on the study of De Pauw, et al¹⁹. Participants reported to have been training for competitive cycling for 1 to 22 y (10.4 ± 6.6 y), at

an average of 15 ± 5 h per week for 4-7 days (5.5 ± 1.0 days) per week for the 6 weeks prior to participation. All participants had had previous experience with structured interval training. The University Human Research Ethics Committee approved the study prior to commencement. All participants signed a written informed consent prior to data collection.

3.3.2 Study Design

3.3.2.1 Overview

Participants completed 5 testing sessions that were each separated by at least 48 h. In the initial session participants performed a graded exercise test. The remaining 4 sessions involved participants performing repeated high-intensity intervals in 4 different T_A (5°C , 13°C , 22°C , & 35°C at 55% relative humidity; Appendix D), were performed in a randomized order, and were completed within a maximum time span of 14 days. Prior to interval and testing sessions subjects were asked to refrain from strenuous exercise in the 36 h prior, from eating large meals in the 3 h prior, and from consuming non-habitual caffeine in the 24 h prior. Eating and drinking were self-monitored and recorded in a log prior to the 1st interval session and subjects were asked to replicate prior to subsequent trials. It was recommended to participants that they consume food and drink similar to what they would prior to a competitive cycling event. Height was measured by a stadiometer and weight was measured on a calibrated scale (GWB Mettler ID1 Multi-Range, Toledo, Columbus, OH, USA). Body composition in terms of sum of skinfolds (Harpender skinfold caliper, Baty International, West Sussex, U.K.) were assessed through an 8-site skinfold test¹⁶⁵. On interval testing session days subjects were asked to complete a recovery-stress questionnaire for athletes (RESTQ) prior to exercise. Nude body weight was taken prior to and following the interval session protocol. Interval sessions were performed at the same time of day for each participant to avoid circadian body temperature variations¹⁶⁶. Participants were instructed

to wear the same athletic clothing to each interval session and cycling jerseys remained zipped up in all T_A .

3.3.2.2 *Graded Exercise Test*

The graded exercise test was performed on an electromagnetically braked cycling ergometer (Velotron, Racermate, Redmond, WA, U.S.A.) adjusted to mimic the participant's own bike. During this test participants began cycling at 100 W for 5 minutes and then 200 W for a subsequent 5 minutes after which power output increased 50 W per minute until volitional fatigue. Expired air was collected continuously and averaged over 5-second intervals (Parvomedics, Sandy, UT, USA). VO_{2max} was determined as the average of the 6 highest consecutive 5-second intervals (30 seconds). Peak power output (PPO) was determined as the peak power value corresponding with the time of volition during the graded exercise test¹⁸. After the completion of the graded exercise test participants were familiarized to the interval session (see below) on the Velotron cycling ergometer by riding 2 interval/rest cycles.

3.3.2.3 *Interval Session*

Cycling exercise was performed on an electromagnetically braked cycling ergometer (Velotron, Racermate, Redmond, Washington). Sessions consisted of a 10-minute warm-up at 50% PPO at room temperature ($\sim 22^\circ\text{C}$). Following the warm-up and a 5-minute transition period, participants performed 5 4-minute intervals with 5 minutes of recovery between each interval inside an environmental chamber. This protocol was chosen as a standard aerobic interval session that could elicit a considerable time spent above 90% VO_{2max} ($T > 90\% VO_{2max}$)⁵⁷. The recovery period consisted of 1 minute at rest, 3 minutes pedaling at volition, followed by 1 minute at 50% PPO. A 75 cm diameter fan was placed ~ 75 cm from the participants face as they sat on the cycle ergometer and provided a simulated wind at a speed of ~ 28 km/h (measured at the face; LM-8000

Anemometer, Lutron Electronic Enterprise CO., LTD, Taipei, Taiwan) during the duration of each interval. Athletes were instructed to pace the intervals to achieve the highest achievable combined intensity (i.e. average power output) for the 5 efforts while being blinded to their power output and speed. During the last interval participants were verbally encouraged to produce a maximal effort to help ensure the session overall was paced at their highest achievable intensity. Whole body perceived exertion (RPE)¹⁶⁷ and thermal sensation (TS)¹⁶⁸ were recorded prior to the 1st interval and at the end of each interval using numerical rating scales. Heart rate (HR; Polar Precision Performance SW5.20, Polar Electro, Kempele, Finland) and power output (Velotron software) were monitored throughout the trials (0.2 Hz and 1 Hz, respectively) and averaged over each individual interval. Peak (highest 1-second power output) and average power output for the intervals was determined using cycling power analytics software (GoldenCheetah, version 3.4, 2016). Heart rate recovery (HRR) was calculated as the difference between the maximum HR per interval and the minimum HR in the subsequent 1-minute rest¹⁶⁹. Rectal temperature (T_C) and skin temperature (T_{sk}) were measured continuously throughout the session (Squirrel SQ2020 Data Logger, Grant Instruments, Shepreth Cambridgeshire, UK). T_C was measured using a disposable rectal thermometer (Monatherm Thermistor 400 Series, Mallinckrodt Medical, St. Luis, MO, USA) inserted ~10 cm past the anal sphincter. Pre-interval session T_C was measured for 30 seconds prior to starting exercise in the 1st interval to ensure consistency across conditions. Skin temperature was measured via 4 thermistor (YTS Temperature, 400 Series, Dayton, Ohio) placed on the chest (T_{chest}), bicep (T_{arm}), thigh (T_{thigh}), and calf (T_{leg}) and mean skin temperature (T_{sk}) was calculated¹⁷⁰ using the following formula:

$$T_{sk} = 0.3 (T_{chest} + T_{arm}) + 0.2 (T_{thigh} + T_{leg})$$

T_C and T_{Sk} measurements were used to calculate mean body temperature (T_B) and the $T_C:T_{Sk}$ gradient. T_B was calculated using the formulas from Colin 1971¹⁷¹ for neutral (5°C, 13°C, & 22°C) and hot (35°C) conditions, respectively:

$$T_B = 0.66 \times T_C + 0.34 \times T_{Sk}$$

$$T_B = 0.79 \times T_C + 0.21 \times T_{Sk}$$

VO_2 , expired carbon dioxide (VCO_2), respiratory exchange ratio (RER), and ventilation (VE) were continuously collected via a Parvomedic metabolic cart (averaged to 5 seconds) except when athletes were allowed to drink ad libitum during the 3-minute period of pedalling at volition in the rest interval. The highest VO_{2max} observed across an individual's 5 testing sessions was utilized as their descriptive VO_{2max} and used to calculate $T > 90\% VO_{2max}$. Sweat loss was calculated by subtracting the nude bodyweight after exercise from the nude bodyweight prior to exercise & weight of water consumed during exercise. Water consumed during exercise was determined by measuring pre and post water bottle weights.

3.3.3 Statistical Analysis

Values are reported as mean \pm standard deviation. Sample number is indicated where a full sample ($n= 11$) was not obtained due to sampling error. A two-way repeated measure ANOVA was used to compare between interval and temperature conditions for the measurements of power output, HR, RPE, TS, VO_2 , RER, VE, VE/VO_2 , VE/VCO_2 , RESTQ, and thermoregulatory responses (T_C , T_{Sk} , $T_C:T_{Sk}$ gradient, T_B). A one-way repeated measure ANOVA was used to compare change in pre-interval session T_C , $T > 90\% VO_{2max}$, body mass changes, water consumption, water loss, and percentage body mass lost. Significance was determined at a confidence level of 95% ($P < .050$). When sphericity was violated a Greenhouse-Geisser correction was utilized. Where significant

differences were observed, Bonferroni corrections for multiple comparisons were used to locate where differences existed. Cohen's d effect sizes with a Hedges' g correction (g) were calculated via a statistical spreadsheet (Jared DeFife, PhD, Emory University, 2009). Data was analysed using SPSS software version 24.0 (IBM, Armonk, New York).

3.4 Results

T_A and relative humidity (RH) for the trials were: $5.01 \pm 0.70^\circ\text{C}$ at $63 \pm 4\%$ RH; $13.13 \pm 0.56^\circ\text{C}$ at $62 \pm 6\%$ RH; $22.04 \pm 0.37^\circ\text{C}$ at $58 \pm 12\%$ RH; and $34.52 \pm 0.95^\circ\text{C}$ at $38 \pm 7\%$ RH. The experimental trials are referred to as 5°C , 13°C , 22°C , and 35°C for clarity of presentation.

3.4.1 Interval Performance

A significantly greater power output was observed in 22°C (364 ± 36 W; $P = .039$, $g = 0.354$) compared with 35°C (352 ± 31 W). The power output for 13°C (366 ± 32 W) was not significantly higher compared with 5°C (363 ± 32 W, $P = .59$, $g = 0.085$), 22°C ($P = 1.00$, $g = 0.061$), and 35°C ($P = .129$, $g = 0.441$). No significant difference was observed across intervals for power output, however significant interval and T_A interactions were observed (Figure 3.1; $P < .001$). Peak power output was significantly different between intervals ($P = .002$) with the 5th interval (final; 570 ± 19 W) significantly higher than the 2nd (517 ± 19 W; $P = .008$), 3rd (410 ± 15 W; $P = .001$), and 4th (409 ± 15 W; $P < .001$) intervals. Cadence was not significantly different across the intervals or T_A , although a significant interaction ($P = .001$) was observed between T_A conditions (Figure 3.1).

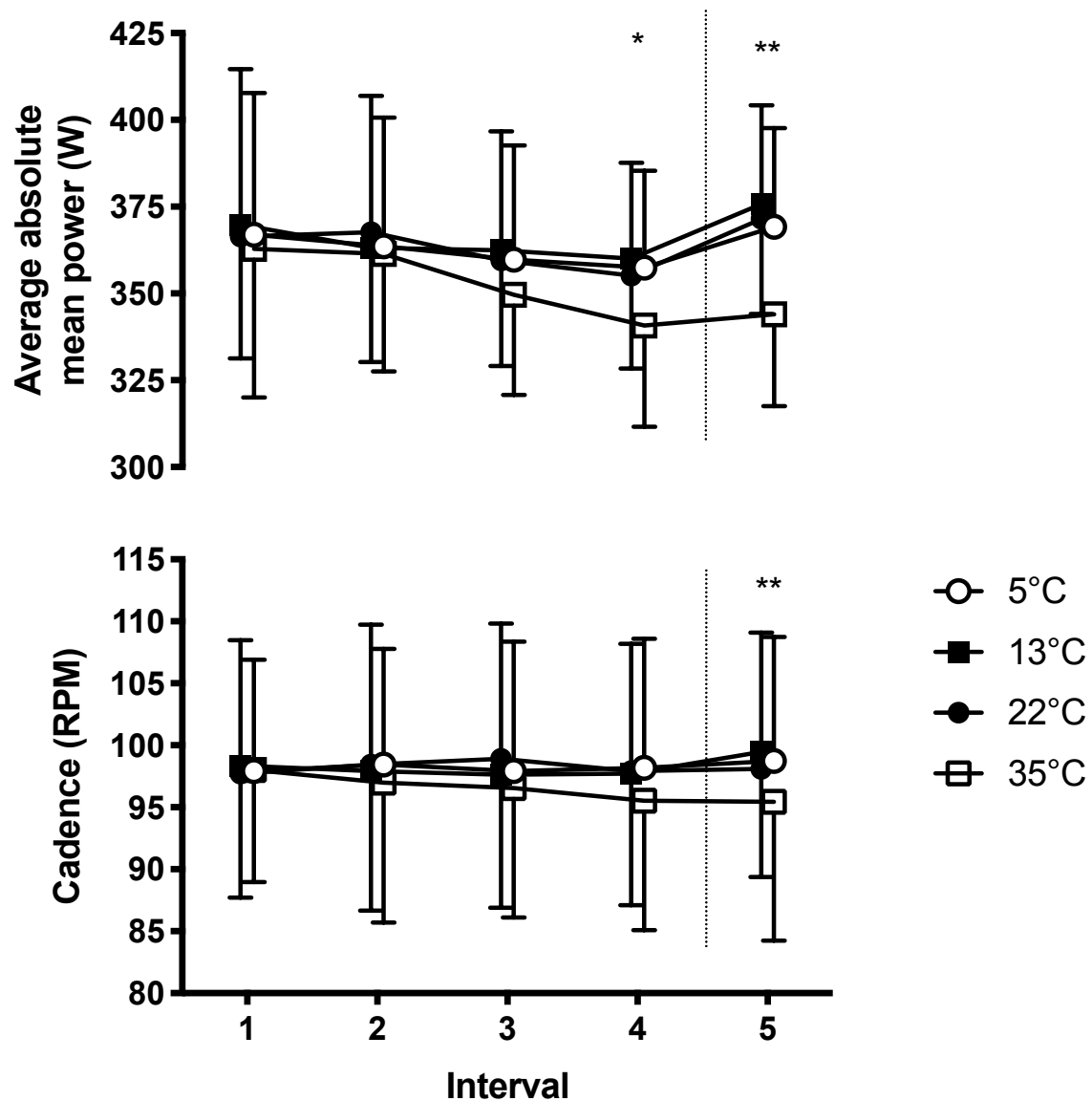


Figure 3.1 Absolute mean power and cadence during 5 high-intensity intervals at 5°C, 13°C, 22°C, & 35°C. Participants were verbally encouraged to perform a maximal effort for the 5th interval. *Significant difference between 35°C vs 5°C & 13°C. **Significant difference between 35°C vs 5°C, 13°C, & 22°C. Values are mean \pm SD for eleven subjects.

3.4.2 Respiratory Measures

No significant differences were observed across T_A for relative $\dot{V}O_2$ ($n=8$, $P=.187$). Relative $\dot{V}O_2$ was lower in the 1st interval versus intervals 2, 3, and 5 ($P=.04$, $P=.038$, $P=.016$, respectively), and similarly interval 4 versus 5 ($P=.026$). RER was not significantly different across T_A ($P=.084$; Figure 3.2), but was significantly different across intervals ($P<.001$). $T>90\% \dot{V}O_{2max}$ was significantly different across T_A ($P=.021$), but post hoc analysis did not reveal a difference ($5^\circ\text{C}=184 \pm 210$ seconds, $15 \pm 18\%$; $13^\circ\text{C}=179 \pm 150$ seconds, $15 \pm 12\%$; $22^\circ\text{C}=260 \pm 194$ seconds, $22 \pm 16\%$; $35^\circ\text{C}=418 \pm 270$ seconds, $35 \pm 23\%$). The percent differences for $T>90\% \dot{V}O_{2max}$ 35°C versus 13°C and 22°C were 80% ($P=.236$, $g=1.035$) and 47% ($P=.514$, $g=0.635$), respectively. VE ($n=8$) in 35°C (135.08 ± 17.70 L/min) was not significantly different ($P=.230$) compared to 22°C (128.35 ± 13.90 L/min, $g=0.40$), 13°C (126.05 ± 17.15 L/min, $g=0.49$), or 5°C (126.67 ± 10.62 L/min, $g=0.55$), nor was a significant interaction found ($P=.921$). VE/ $\dot{V}O_2$ ($n=8$; 5°C 30.71 ± 1.58 ; 13°C 30.70 ± 1.69 ; 22°C 30.85 ± 2.06 ; 35°C 31.62 ± 2.49) was not significantly different across T_A ($P=.649$) and no significant interaction was found ($P=.094$). VE/ $\dot{V}CO_2$ ($n=8$; 5°C 31.23 ± 0.45 ; 13°C 31.14 ± 0.50 ; 22°C 30.85 ± 0.71 ; 35°C 31.46 ± 1.20) was not significantly different across T_A ($P=.309$), yet a significant interaction was found ($P<.001$), however but post hoc analysis did not reveal a difference.

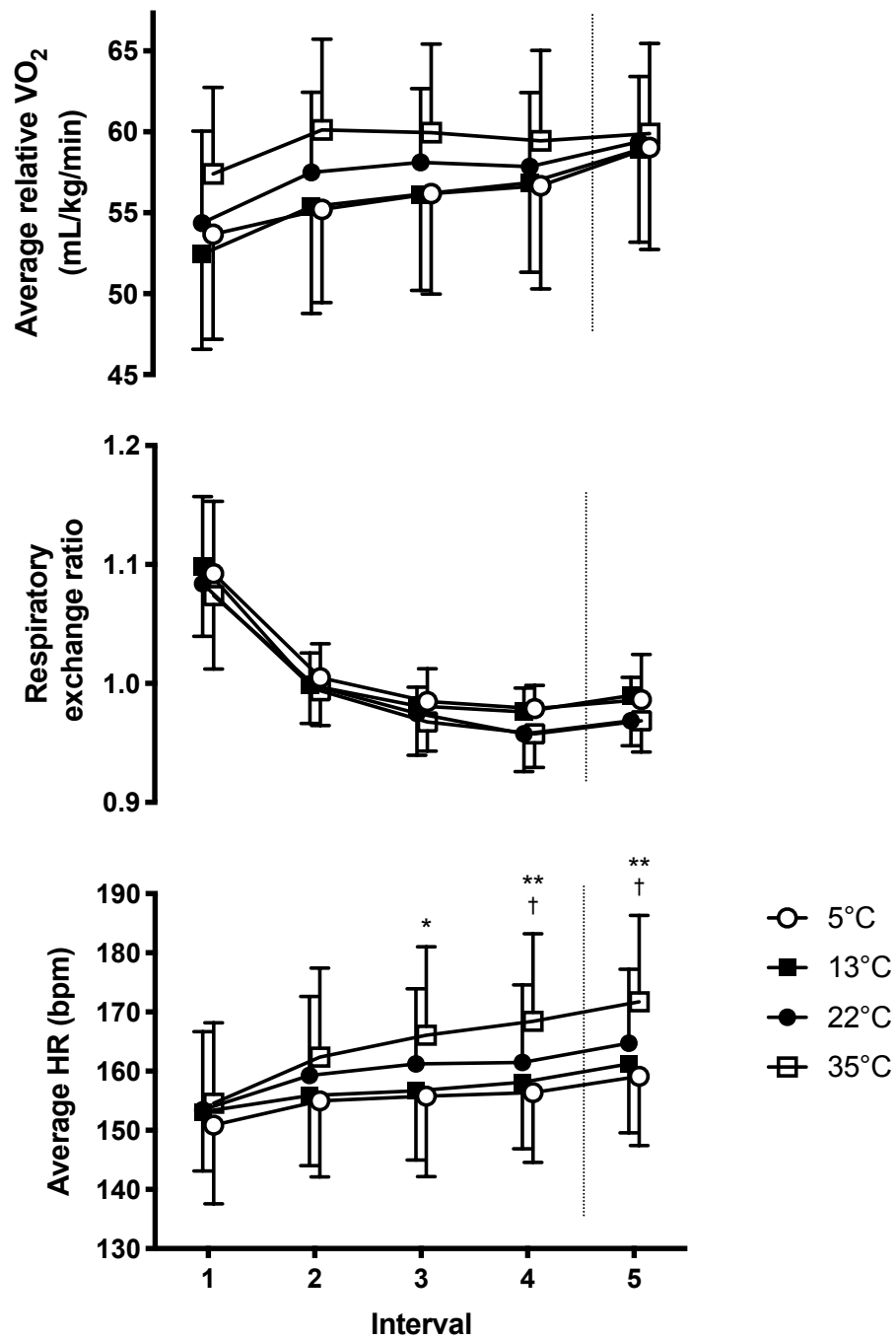


Figure 3.2 Average relative oxygen consumption (VO_2 , $n=8$), respiratory exchange ratio ($n=8$), and average heart rate (HR, $n=8$) during 5 high-intensity intervals at 5°C, 13°C, 22°C, & 35°C. Participants were verbally encouraged to perform a maximal effort for the 5th interval. *Significant difference between 22°C vs 35°C. **Significant differences between 5°C vs 22°C & 35°C temperatures. †Significant difference between 13°C vs 35°C. Values are mean \pm SD.

3.4.3 Body Temperature

All thermoregulatory measures were significantly different across all T_A conditions (T_C $n=9$, $P=.001$; T_{Sk} $n=8$, $P<.001$; $T_C:T_{Sk}$ gradient $n=7$, $P<.001$; T_B $n=7$, $P<.001$). Post hoc analysis found T_C was significantly higher in 22°C ($38.25 \pm 0.17^\circ\text{C}$) versus 5°C ($38.01 \pm 0.17^\circ\text{C}$, $P=.001$, $g=1.38$) and 13°C ($38.11 \pm 0.15^\circ\text{C}$, $P=.001$, $g=0.843$). T_C was lowest in the 5°C compared with 13°C ($P=.195$, $g=0.631$), 22°C, and 35°C ($38.18 \pm 0.19^\circ\text{C}$, $P=.260$, $g=0.957$) T_A conditions ($P<.001$, Figure 3.3). T_{Sk} and T_B were greatest in 35°C and lowest in 5°C. Inversely, $T_C:T_{Sk}$ gradient values were highest in 5°C and progressively decreased in hotter T_A . T_C and $T_C:T_{Sk}$ gradient significantly increased over the intervals ($P<.001$; $P<.001$, respectively), while T_{Sk} decreased through the session ($P<.001$). T_B was not significantly different across intervals ($P=.193$). Significant interactions were observed in T_{Sk} ($P<.001$), $T_C:T_{Sk}$ gradient ($P<.001$), and T_B ($P<.001$; Figure 3.3). Pre-interval session T_C was not significantly different across T_A ($37.55 \pm 0.14^\circ\text{C}$, $P=.715$), but was significantly higher than baseline ($37.31 \pm 0.18^\circ\text{C}$, $P<.001$).

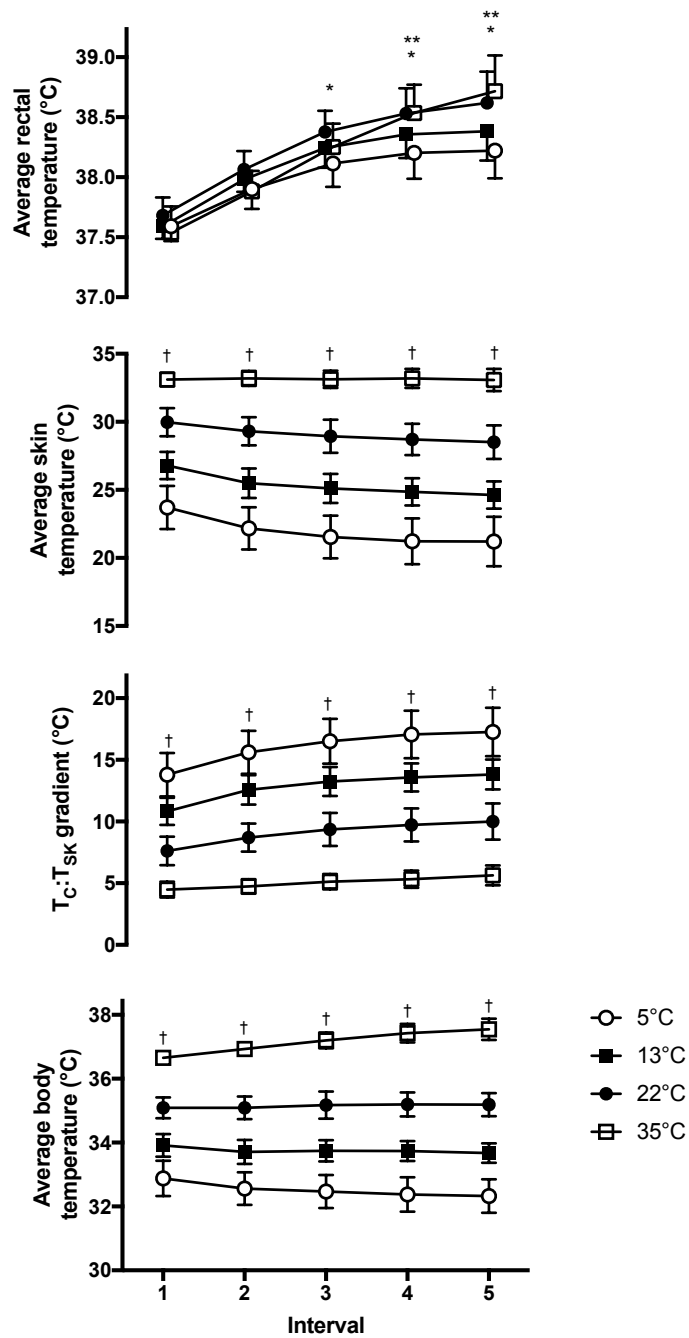


Figure 3.3 Average rectal temperature ($n=9$), average skin temperature ($n=8$), skin temperature to rectal temperature ($T_c:T_{sk}$) gradient ($n=7$), and body temperature ($n=7$) during 5 high-intensity intervals at 5°C, 13°C, 22°C, & 35°C. *Significant difference between 22°C vs 5°C & 13°C. **Significant difference between 5°C vs 13°C & 35°C. †Significant differences between all temperatures. Values are mean \pm SD.

3.4.4 Heart Rate

Average HR for the intervals (n= 8) was significantly different across T_A and intervals ($P= .001$, $P< .001$, respectively). Post hoc analysis did not reveal significance differences in HR across T_A , yet there was a trend for 35°C to be higher than 5°C ($P= .052$, $g= 0.714$). Heart rate increased significantly with each subsequent interval except between intervals 2 and 3 (Figure 3.2). Maximum HR for the intervals (n= 8) was significantly different across both the intervals and T_A ($P< .001$, $P= .001$, respectively), but no significant interaction between intervals and T_A was observed ($P= .073$). Significant main effects were observed for HRR across T_A and intervals, and trended towards significance in the interaction ($P< .001$, $P< .001$, and $P= .051$, respectively). HRR for the 35°C condition was found to be significantly lower than the 13°C condition ($P= .013$). Post hoc analysis revealed that the HRR for the 5th interval was significantly lower than the 2nd and 4th intervals ($P= .026$, $P= .035$, respectively). The values for the HRR of the 5th (final) interval were: 5°C= 48 ± 22 bpm, 13°C= 47 ± 22 bpm, 22°C= 40 ± 18 bpm, 35°C= 29 ± 14 bpm.

3.4.5 Mass and Water Loss

There was no significant difference in the overall change in participants' body mass between T_A ($P= .054$). Water consumption was significantly different across T_A conditions ($P< .001$) with significant differences found between all T_A except 22°C and 35°C (Table 3.1). Water loss in terms of absolute and percent of body mass was significantly different across T_A conditions ($P< .001$, $P< .001$, respectively; Table 3.1).

Table 3.1 Subjects' change in body weight (BW), fluid intake, sweat lost, and percentage BW lost during 5 high-intensity intervals at 5°C, 13°C, 22°C, & 35°C.

	5°C	13°C	22°C	35°C
Change in BW (kg)	0.28 ± 0.21	0.44 ± 0.25	0.57 ± .31	0.77 ± .74
Fluid intake (kg)	0.23 ± 0.14*	0.35 ± .14*	0.51 ± .23**	0.73 ± .37**
Sweat lost (kg)	0.51 ± 0.15*	0.79 ± .20*	1.08 ± .19*	1.50 ± .51*
BW lost (%)	0.67 ± 0.18*	1.03 ± .22*	1.41 ± .13*	1.96 ± 0.57*

*Significant difference between all other temperature conditions; ** significantly different to 5°C and 13°C. Values are mean ± SD for eleven subjects.

3.4.6 Subjective Responses

RESTQ results were not significantly different across interval sessions ($n = 10$, $P = .803$). RPE ($n = 10$) was significantly different across temperature conditions, specifically the 13°C being lower than the 35°C ($P = .041$). RPE increased with each successive interval ($P < .001$) with significant differences across all intervals. A significant interaction between intervals and T_A was found ($P = .037$) in RPE (Figure 3.4). Thermal sensation was significantly different across T_A , intervals, and the interaction between the two factors ($P < .001$, $P = .005$, $P < .001$, respectively). Significant interactions for thermal sensation are presented in Figure 3.4.

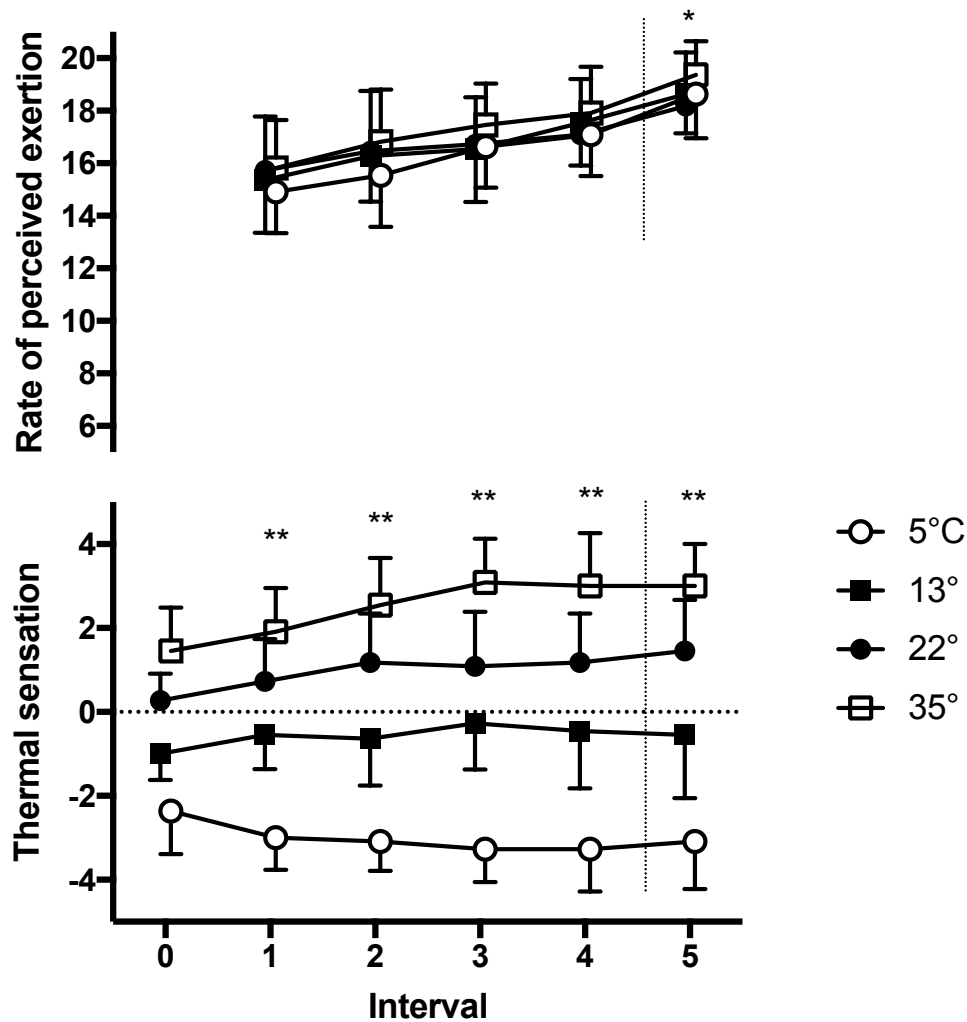


Figure 3.4 Rate of perceived exertion (n= 11) and thermal sensation (n=11) during 5 high-intensity intervals at 5°C, 13°C, 22°C, & 35°C. Participants were verbally encouraged to perform a maximal effort for the 5th interval. Baseline values for thermal sensation were not included in the statistical analysis. *Significant difference between 13°C vs 35°C. **Significant difference between all temperatures. Values are mean \pm SD.

3.5 Discussion

The purpose of this study was to determine performance and physiological responses during intervals performed under a range of T_A . The main findings from this study are that: i) power output was lower in the last interval of the 35°C condition compared with all other T_A , yet no significant interactions were observed between 5°C, 13°C, and 22°C conditions (Figure 3.1); ii)

T_C increased significantly over the course of the intervals but was significantly lower overall in 5°C and 13°C compared with 22°C; iii) no significant difference in VO_2 was observed between T_A conditions, whilst HR for intervals in 35°C were higher than intervals in 5°C, 13°C, and 22°C (Figure 3.2).

It is well accepted that endurance exercise performance is compromised in cold^{2,6} and hot T_A ^{2,6,7}. Within the current study lower power output was observed in 35°C compared with 5°C and 13°C during the 4th interval and by the 5th interval power output in 35°C was significantly lower than all the other T_A (Figure 3.1). This reduction in performance in the heat is consistent with the research examining steady-state exercise, intermittent exercise, and sprint intervals, but to our knowledge, had yet to be demonstrated in aerobic high-intensity intervals⁵⁷. Reduction in exercise performance in the heat is typically associated with hyperthermia-induced alterations in blood flow and particularly compromised blood flow, increased cardiovascular strain, and reductions in VO_2 ^{26,52}. Indeed, Périard et al.⁵² observed an increase in skin blood flow, decrease in cardiac output, and decrease in VO_2 during a 40 km time-trial in hot conditions (35°C). Interestingly, VO_2 within the present study was not different across any of the T_A conditions, despite the decrease in performance within the hottest condition. The reasons for this are unclear. No significant differences in VE (i.e. a source of cardiopulmonary oxygen demand) were observed across T_A , yet the ES between 35°C and the other T_A conditions were notable (22°C $g = 0.40$, 13°C $g = 0.49$, 5°C $g = 0.55$). Additional research is needed to fully understand if this difference in VE could potentially explain maintenance of systemic VO_2 while observing a decrease in power output.

Contrary to the hypothesis of this study, no reduction in performance in the coldest condition of 5°C was observed, implying that 5°C was not cold enough to alter homeostatic control to a point performance was compromised (e.g. reduced aerobic power³). The reason cold did not negatively

affect performance in this study, while it has been demonstrated to have this effect in past studies, could potentially be explained by multiple factors including: the standardized neutral environment warm-up (10 minutes at 50% PPO) prior to exercise; and high metabolic heat caused by both the high-intensity of the interval session¹⁴ and the high power output (and therefore heat production) of the well-trained cyclist participants¹⁸. Indeed, T_C in the 5°C intervals was lower than the T_C in the 13°C and 22°C conditions (Figure 3.3), yet no difference in power output across these conditions were observed (Figure 3.1); implying either a minimal temperature (T_C or muscle) to perform intense exercise had been attained¹²⁴, or possibly the duration of the attenuated T_C was not long enough to negatively affect performance.

3.5.1 Practical Applications

Interval training is an essential component of competitive endurance training²¹, and it has been suggested that an important aspect of maximizing adaptations to aerobic interval training is to optimize the time spent at or near VO_{2max} ^{23,57}. Indeed while the relative VO_2 for the intervals in this study were not significantly different across T_A , the time spent above 90% VO_{2max} was greater in 35°C versus 13°C and 22°C with 80% ($P = .236$, $g = 1.035$) and 47% ($P = .514$, $g = 0.635$) differences, respectively. However, it is important to note that it is not currently well understood whether the major aerobic adaptations from interval training are due to systemic increases in VO_2 or the concurrent increases in muscle blood flow (i.e. power output). Given that time spent above 90% VO_{2max} was greater at 35°C in this study, and a counterargument can be made that muscle blood flow would be greater for intervals performed in cool conditions, more research is needed to determine what T_A is best for well-trained endurance athletes performing HIIT.

3.5.2 Conclusion

Our data indicate that hot environmental temperature ($>35^{\circ}\text{C}$) has a negative effect on interval power output during the latter intervals of a 40-minute interval session, while power output for intervals in environmental temperatures as low as 5°C are not affected when performed by well-trained cyclists after a standard neutral temperature warm-up. The decrease in power output and increase in HR during the interval sessions in the 35°C condition was in agreement with data previously collected from our lab during self-paced time-trial performance in the heat (32°C)⁷. No decrement in power output during the 5°C intervals sessions occurred despite an attenuated rise in core temperature similar to that observed in previous studies stating cold environmental temperatures (5°C) had negative effects on endurance performance². More research is needed before temperature can be effectively considered as an ergogenic factor during high-intensity interval training.

3.6 Acknowledgements

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4 CHAPTER FOUR:

STUDY 2

EFFECT OF REPEATED HOT AND COOL TEMPERATURE EXPOSURE ON HIGH-INTENSITY INTERVALS IN TRAINED CYCLISTS

Link: Findings from Chapter 3 indicated that environmental temperature has an acute effect on performance and physiological responses during high-intensity interval sessions performed by well-trained cyclists, especially in terms of cardiorespiratory stress. This study (Chapter 4) sought to understand the effects repeated exposure to hot (35°C) and cool (13°C) environmental temperatures had on physiological responses during high-intensity interval sessions.

4.1 Abstract

Purpose: To examine changes in cardiopulmonary and thermoregulatory responses during maximal high-intensity intervals after being repeatedly performed in either hot (35°C) or cool (13°C) environmental temperature (T_A). **Methods:** Twenty trained cyclists and triathletes completed a 4-week (8 session) high-intensity interval training (HIIT) intervention at a T_A of either 13°C (HIIT₁₃) or 35°C (HIIT₃₅). Interval training sessions were separated by at least 36 hours and consisted of five 4-minute intervals with 5 minutes of recovery between each. Cardiopulmonary and thermoregulatory data were collected during the first (INT1) and last (INT8) interval sessions. **Results:** $T > 90\%$ VO_{2max} was significantly greater in HIIT₁₃ (877 ± 297 seconds; $P = .007$, $g = 1.278$), compared with HIIT₃₅ (421 ± 395 seconds) during the intervention, but did not change for either group between INT1 and INT8. Heart rate was not significantly different between HIIT₁₃ (164 ± 9 bpm) and HIIT₃₅ (164 ± 12 bpm; $P = .989$, $g = 0.006$). T_C significantly decreased in HIIT₃₅ between INT1 and INT8 ($P = .030$, $g = -0.544$; HIIT₁₃ $P = .801$, $g = -0.104$). **Conclusions:** These results indicate the relationship between time spent at or near VO_{2max} and cardiovascular strain during HIIT is influenced by T_A . This relationship is important as it gives insight into training prescription and adaptations to HIIT. Additionally, 35°C HIIT intermittently prescribed (~2x per week) demonstrated some evidence for heat acclimation in endurance athletes.

4.2 Introduction

High-intensity interval training (HIIT) is well known for its role in increasing performance and stimulating beneficial physiological adaptations for exercise, especially among endurance athlete populations^{21,22,172,173}. Physiological benefits from HIIT that are important to performance include increased maximal oxygen consumption^{22,172} ($\text{VO}_{2\text{max}}$) and ventilatory thresholds^{21,22}. These adaptations are associated with repeated stress on the oxygen transport and utilization systems⁵⁷ which occurs during HIIT; whereby maximal cardiac output is approached⁶³, large motor units are recruited⁶⁴, and muscle blood flow is subsequently increased. It has been suggested that aerobic adaptations during HIIT sessions are stimulated most effectively when time spent at or near $\text{VO}_{2\text{max}}$ is maximised^{23–25}. To improve cardiopulmonary function, HIIT sessions are prescribed with characteristic durations for interval (~4-minutes) and rest periods (>3-4 minutes)⁵⁷. Additionally, maximizing time spent at or near $\text{VO}_{2\text{max}}$ during an interval session requires considerations for work interval intensity. Whereby, pacing intervals at the highest achievable combined intensity possible (i.e. maximal effort) results in a higher time at or near $\text{VO}_{2\text{max}}$, versus work intervals at prescribed intensities⁸².

During a high-intensity training session heat may increase skin blood flow⁵², possibly compromising cardiac output⁵², and decreasing oxygen delivery to working muscles^{26,27}. Conversely, performing high-intensity intervals in cooler conditions increases core temperature (T_C) to skin temperature (T_{sk}) gradient¹⁷⁴, reducing skin blood flow²⁶, increasing venous return, and therefore potentially enhancing the proportion of cardiac output delivered to the working muscles²⁶. Recently, Boynton et al. 2019¹⁷⁴ (Chapter 3) observed lower power output and heart rate (HR) during intervals performed in 35°C as compared with 13°C, and a higher T_C compared to 5°C. Whilst the findings of this study gave insight into the acute physiological responses

occurring during high-intensity intervals at different T_A , the benefits from HIIT manifest after repeated bouts. It can be argued that differences in acute cardiorespiratory stress during high-intensity intervals, observed at different T_A , could potentially change uniquely after repeat sessions at that given T_A . This change in physiological stress over the course of a HIIT intervention could potentially diminish or enhance training outcomes. An understanding of how cardiorespiratory physiology during intervals changes after repeated exposures to different T_A , and how these changes differ between T_A , could give beneficial insight into optimizing HIIT prescription.

It is well-established that exercise performance in hot conditions improves after sufficient repeated exposures to heat^{33,34,175}. Physiological adaptations that occur in response to repeated sessions of exercising in the heat include increased sweat loss, reduced HR, T_{C} , T_{Sk} , & thermal sensation (TS)⁴⁴, and a reduced T_C threshold for hyperventilation¹⁷⁶. Recently, Reeve et al. 2019¹⁴⁴ demonstrated adaptations to heat with a coinciding decrease in cycling performance after well-trained endurance athletes performed HIIT (12 x 1-minute intervals at 100% peak power output (PPO)) in hot (35°C) conditions over 5 consecutive days. However, this study's protocol contrasts with HIIT prescribed specifically to increase endurance performance, which generally incorporates a minimum of 48 hours between bouts to allow for sufficient recovery in order to avoid non-functional overreaching^{45,46}. Conversely, it has been argued that exercising during repeated cold exposures attenuates cold acclimation¹⁴⁹, as the increased T_C from exercise prevents the required whole-body T_C drop¹⁴⁷. Additionally, intermittent T_A exposures (e.g. hot or cool) during HIIT could influence cardiorespiratory adaptations but be too infrequent to stimulate thermoregulatory adaptations in trained endurance athletes. Currently, the magnitude of thermoregulatory adaptations that occur after repeated hot and cool intermittent-day interval sessions is not well understood.

The purpose of the this study was to investigate changes in cardiorespiratory (e.g. VO_2 , time at or near $\text{VO}_{2\text{max}}$, ventilation, and HR) and thermoregulatory (e.g. T_C & T_{sk}) measures at two separate T_A (13°C & 35°C) between the first and last interval sessions of a 4-week (8 session) intervention in trained cyclists. It was hypothesized that training would result in an increase in VO_2 and time spent at or near $\text{VO}_{2\text{max}}$ during the intervals in both conditions, while whole body perceived exertion (RPE) and thermoregulatory responses would not change. Additionally, it was hypothesized that the increase in VO_2 observed in the last interval bout of the intervention would be higher in the 13°C condition compared to the 35°C condition.

4.3 Methods

4.3.1 Participants

Twenty-one male and female trained cyclists and triathletes (De Pauw et al. 2013¹⁹) were recruited from the Perth, Western Australia region for this study (Appendix E). Twenty participants completed the protocol and were included in the final analysis. One participant did not complete the study protocol. Participants were assigned to one of two groups that were matched for $\text{VO}_{2\text{max}}$, PPO, and biological sex (Table 4.1). All participants signed a written informed consent prior to data collection. The Edith Cowan University Human Research Ethics Committee approved the study prior to commencement.

4.3.2 Study Overview

During the initial testing session all participants height, weight, and body composition were assessed before completing a graded exercise test (GXT; described Section 4.3.2.1) to determine $\text{VO}_{2\text{max}}$ and PPO. Height was measured by a stadiometer and weight was measured on a calibrated scale (GWB Mettler ID1 Multi-Range, Toledo, Columbus, OH, USA). Body composition in terms of sum of skinfolds (Harpenden skinfold calliper, Baty International, West Sussex, U.K.) were

assessed through an 8-site skinfold test¹⁶⁵. Participants partook in a 4-week HIIT intervention at a T_A of either 13°C (HIIT₁₃) or 35°C (HIIT₃₅; Appendix D). The intervention consisted of a total of 8 cycling high-intensity interval sessions, where performance and physiological data (described below) were collected during the first and last interval sessions (INT1 & INT8, respectively). Interval sessions were separated by at least 36 hours and consisted of 5 x 4-minute intervals with 5 minutes of recovery between each¹⁷⁴. Athletes were instructed to pace the intervals to achieve the highest achievable combined intensity (i.e. average power output) for the 5 efforts while being blinded to their power output and speed. During the last interval participants were verbally encouraged to produce a maximal effort to help ensure the session overall was paced at their highest achievable intensity. This interval session protocol was chosen for its demonstrated time above 90% VO_{2max} ($T > 90\% VO_{2max}$)^{23–25,174}. Interval training sessions 2 to 7 were performed on a WattBike (WattBike Pro, WattBike, West Bridgford, Nottingham, UK) adjusted to mimic the participant's own bike. During these interval training sessions (sessions 2 to 7) a 75 cm diameter fan was placed ~75 cm from the participants face as they sat on the cycle ergometer and provided a simulated wind at a speed of ~17 km/hour (measured at the face; LM-8000 Anemometer, Lutron Electronic Enterprise CO., LTD, Taipei, Taiwan) for the duration of the individual intervals. Participants were instructed not to perform any structured HIIT outside of the laboratory during the course of the intervention. Participant's training volume in terms of duration (hours) of aerobic exercise (e.g. cycling, running, & swimming) completed 6 weeks prior to and during the interval intervention was acquired via the participant's personal training logs. Previous HIIT history and years as a cyclist were reported by participants via a questionnaire. The GXT, INT1, and INT8 sessions were performed on an electromagnetically-braked cycling ergometer (Velotron, Racermate, Redmond, WA, U.S.A.). Prior to testing sessions subjects were asked to refrain from

strenuous exercise in the 36 hours prior, eating large meals in the 3 hours prior, and from consuming non-habitual caffeine in the 24 hours prior. Participants were instructed to eat and drink as they would before a competitive event and record their consumption in a log prior to the first testing session. For consistency participants were then asked to replicate their dietary consumption 24 hours prior to INT1 and INT8.

Table 4.1 Baseline physical, performance, and training characteristics of participants in 13°C (HIIT₁₃) and 35°C (HIIT₃₅) high-intensity interval training groups.

	HIIT ₁₃	HIIT ₃₅	<i>p-value</i>
	n= 10	n= 10	
	m= 7, f= 3	m= 7, f= 3	
	cy= 7 , tri= 3	cy= 5, tri= 4, du= 1	
Age (y)	33 ± 6	36 ± 12	.526
Body Mass (kg)	71.5 ± 13.5	70.1 ± 11.4	.800
Height (cm)	177 ± 10	175 ± 8	.610
Sum of 8 (mm)	92 ± 46	85 ± 36	.725
PPO (W)	352 ± 59	346 ± 63	.810
PPO (W/kg)	4.97 ± 0.64	4.94 ± 0.74	.915
VO _{2max} (L/min)	4.06 ± 1.00	4.01 ± 0.77	.710
VO _{2max} (mL/kg/min)	56.9 ± 11.2	57.3 ± 9.5	.921
Time as a cyclist (y)	10.2 ± 7.3	14.4 ± 16.9	.505
Utilize HIIT in program (n=)	6	6	NA
Training hours per week (h)	10.93 ± 4.25	10.28 ± 2.95*	0.706

Data presented as group mean ± SD. Sum of 8 = total 8 sites of skinfolds, VO_{2max} = maximal oxygen consumption, Time as cyclist = time participant reported that they had been riding recreationally (i.e. total time cycling), Utilize HIIT in program = number of participants per group that had utilized high-intensity interval training in the past, Train hours per week = average hours per week participants trained per week (6 weeks prior to intervention start + training during the intervention), m = male, f = female, cy = cyclists, tri = triathletes, du = duathletes. *n= 9

4.3.2.1 Graded Exercise Test

The GXT was performed on a cycling ergometer adjusted to mimic the participant's own bike. Measurements were recorded and utilized in subsequent sessions. During this test participants began cycling at 100 W (male) or 50 W (female) for 3 minutes, after which power increased 20 W per minute until volitional fatigue. Expired air was collected continuously and averaged over 5-second intervals (Parvomedics, Sandy, UT, USA). $\text{VO}_{2\text{max}}$ was determined as the average of the 6 highest consecutive 5-second intervals (30 seconds) and was used to calculate $T > 90\% \text{VO}_{2\text{max}}$ during INT1 and INT8. PPO was determined using the power value corresponding with the time of volition (cadence below 60 rpm) during the graded exercise test¹⁸, and 50% PPO was calculated using this value.

4.3.2.2 Interval Testing

Sessions consisted of a 10-minute warm-up at 50% PPO at room temperature ($\sim 24^{\circ}\text{C}$). Following the warm-up and a 5-minute transition period, participants performed 5 x 4-minute intervals with 5 minutes of recovery between each interval inside an environmental chamber. The recovery period consisted of 1 minute at rest, 3 minutes pedaling at volition, followed by 1 minute at 50% PPO. A 75 cm diameter fan was placed ~ 75 cm from the participants face as they sat on the cycle ergometer and provided a simulated wind at a speed of ~ 28 km/h during the duration of each interval. Whole body RPE¹⁶⁷ and TS¹⁶⁸ were recorded prior to the 1st interval and at the end of each interval using numerical rating scales. HR (Polar Precision Performance SW5.20, Polar Electro, Kempele, Finland) and power output (Velotron software, Racermate, Redmond, WA, U.S.A.) were monitored throughout the trials (0.2 Hz and 1 Hz, respectively) and averaged over each individual interval. Average power output for the intervals was determined using cycling power analytics software (GoldenCheetah, version 3.4, 2016). Rectal temperature (T_{C}) and T_{Sk}

were measured continuously throughout the session (Squirrel SQ2020 Data Logger, Grant Instruments, Shepreth Cambridgeshire, UK). Fluctuations in temperature data greater than 0.10°C/second are physiologically improbable and likely the result of faulty temperature probes and thus were removed from analysis. T_C was measured using a disposable rectal thermometer (Monatherm Thermistor 400 Series, Mallinckrodt Medical, St. Luis, MO, USA) inserted ~10 cm past the anal sphincter. T_{Sk} was measured via 4 thermistor (YTS Temperature, 400 Series, Dayton, Ohio) placed on the chest (T_{chest}), bicep (T_{arm}), thigh (T_{thigh}), and calf (T_{leg}) and mean T_{Sk} was calculated¹⁷⁰ using the following formula:

$$T_{Sk} = 0.3 (T_{chest} + T_{arm}) + 0.2 (T_{thigh} + T_{leg})$$

T_C and T_{Sk} measurements were used to calculate mean body temperature (T_B) and the $T_C:T_{Sk}$ gradient. T_B was calculated using the formulas from Colin 1971¹⁷¹ for neutral (13°C & 22°C) and hot (35°C) conditions, respectively:

$$T_B = 0.66 \times T_C + 0.34 \times T_{Sk}$$

$$T_B = 0.79 \times T_C + 0.21 \times T_{Sk}$$

VO_2 , expired carbon dioxide (VCO_2), respiratory exchange ratio (RER), ventilation (VE) and respiratory rate were continuously collected via a Parvomedic metabolic cart (averaged to 5 seconds) except when athletes were allowed to drink ad libitum during the 3-minute period of pedalling at volition in the rest interval. Sweat loss was calculated by subtracting the nude bodyweight after exercise from the nude bodyweight prior to exercise & weight of water consumed during exercise. Water consumed during exercise was determined by measuring pre and post water bottle weights.

4.3.3 Statistical Analysis

Values in text are reported as mean \pm standard deviation. Sample number is indicated where a full sample ($n=10$) was not obtained due to sampling error. A 2-tailed t-test was utilized to match performance, anthropometric, and physiological attributes of the two groups. A two-way repeated measure ANOVA was used to compare measures between group and intervals session, and within subject measures recorded during individual intervals across both tests. Significance was determined at a confidence level of 95% ($P < .050$). When sphericity was violated a Greenhouse-Geisser correction was utilized. Where significant differences were observed, Bonferroni corrections for multiple comparisons were used to locate where differences existed. Cohen's d with a Hedges' g correction (g) and partial eta squared (η_p^2) were reported as effect sizes. Hedges' g was calculated via a statistical spreadsheet (Jared DeFife, PhD, Emory University, 2009). SPSS software version 25.0 (IBM, Armonk, New York) was utilized to calculate η_p^2 and analyze data.

4.4 Results

Anthropometric and physiological characteristics were not different between experimental groups (Table 4.1). The training hours for HIIT₁₃ and HIIT₃₅ during the intervention were 12.73 ± 4.96 hours/week and 11.10 ± 4.83 hours/week ($n=9$), respectively ($P=.479$). Environmental chamber conditions for the interval testing sessions for the two groups were $12.92 \pm 0.29^\circ\text{C}$, $63 \pm 5\%$ RH and $34.90 \pm 0.53^\circ\text{C}$, $47 \pm 4\%$ RH (RH $P < .001$). Significant differences for T_A conditions were noted between INT1 and INT8 for HIIT₁₃ ($12.82 \pm 0.26^\circ\text{C}$ vs. $13.01 \pm 0.31^\circ\text{C}$, $P=.041$) and HIIT₃₅ ($34.67 \pm 0.49^\circ\text{C}$ vs. $35.12 \pm 0.49^\circ\text{C}$, $P=.014$). Room temperature for the standardized warm-up was $23.65 \pm 0.74^\circ\text{C}$ (RH was not recorded).

4.4.1 Interval Performance Measures

Power output was not significantly different between HIIT₁₃ (309 ± 53 W) and HIIT₃₅ (273 ± 53 W; $P = .146$, $g = 0.673$). Power output was not different between INT1 and INT8 in either HIIT₁₃ (INT1 = 310 ± 57 , INT8 = 308 ± 53 ; $P = .594$, $g = -0.027$) or HIIT₃₅ (INT1 = 270 ± 51 , INT8 = 276 ± 55 ; $P = .372$, $g = 0.111$; Figure 4.1). No differences in cadence or RPE were observed between HIIT₁₃ and HIIT₃₅, and INT1 and INT8 (Figure 4.1).

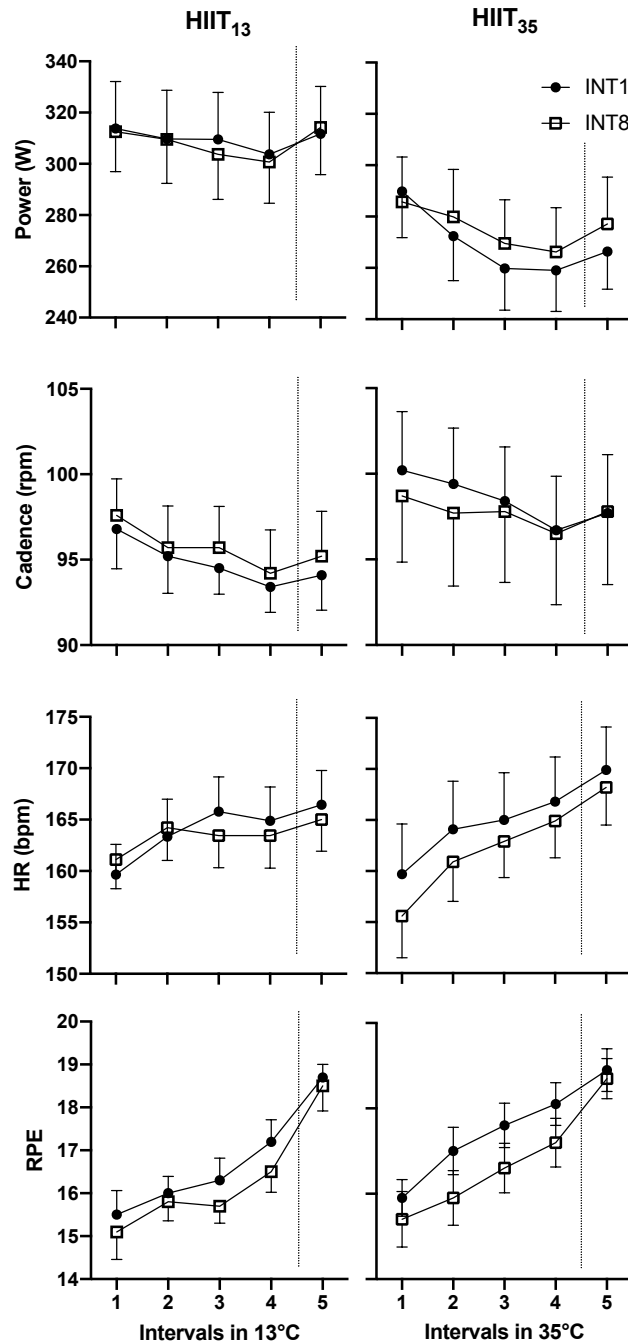


Figure 4.1 Power output, cadence, heart rate (HR), and rate of perceived exertion (RPE) measures during the first (INT1) and last (INT8) session of a 4-week (8 sessions) high-intensity interval training intervention performed by 2 matched groups of trained cyclists in either 13°C (HIIT₁₃) or 35°C (HIIT₃₅). Interval sessions consisted of 5x 4-minute intervals separated by 5 minutes of rest. During the last interval participants were verbally encouraged to produce a maximal effort to help ensure the session overall was paced at their highest achievable intensity. Testing conditions were 40% relative humidity with a fan providing a simulated wind at a speed of ~28 km/hour during individual intervals. Values are mean ± SEM, n= 10 per group except for HR in HIIT₁₃ (n= 9). rpm indicates revolutions per minute; bpm, beats per minute.

4.4.2 Cardiorespiratory Measures

HR was not significantly different between HIIT₁₃ ($n=9$, 164 ± 9 bpm) and HIIT₃₅ (164 ± 12 bpm; $P=.989$, $g=0.006$; Figure 4.1). Similarly, HR was not different between INT1 and INT8 for HIIT₁₃ ($P=.706$, $g=-0.064$) and HIIT₃₅ ($P=.221$, $g=-0.196$; Figure 4.1).

Time above 90% VO_{2max} was significantly greater in HIIT₁₃ (877 ± 297 seconds; $P=.007$, $g=1.278$), compared with HIIT₃₅ (421 ± 395 seconds; Figure 4.2). However, no difference in $T > 90\%$ VO_{2max} was observed between INT1 and INT8 in either HIIT₁₃ ($P=.105$, $g=-0.249$) or HIIT₃₅ ($P=.689$, $g=-0.105$). VO_2 (L/min; HIIT₁₃ $n=9$; $P=.163$, $g=0.640$), VCO_2 (HIIT₁₃ $n=9$), RER (HIIT₁₃ $n=9$), and VE (HIIT₁₃ $n=9$; $P=.651$, $g=0.197$; Figure 4.3) during the interval sessions were not significantly different between HIIT₁₃ and HIIT₃₅. VO_2 and VE displayed no significant interaction between groups and INT1 and INT8 ($P=.814$, $\eta_p^2=0.003$; $P=.865$, $\eta_p^2=0.002$; Figure 4.3). Ventilatory equivalents for oxygen (VE/ VO_2 ; HIIT₁₃ $n=9$) and carbon dioxide (VE/ VCO_2 ; HIIT₁₃ $n=9$) were significantly higher in HIIT₃₅, compared with HIIT₁₃ ($P=.001$, $P<.001$, respectively; Figure 4.3), but no interactions ($P=.926$, $\eta_p^2=0.001$; $P=.687$, $\eta_p^2=0.048$) were observed with INT1 and INT8. Effect sizes for changes in VE, VE/ VO_2 , and VE/ VCO_2 between INT1 and INT8 were $g=-0.186$, $g=-0.060$, $g=-0.223$, for HIIT₁₃ and $g=-0.202$, $g=-0.069$, $g=-0.465$ in HIIT₃₅, respectively. Respiratory rate (HIIT₁₃ $n=9$) was not significantly different between groups ($P=.414$), but did significantly decrease during INT8 for both groups ($P=.014$).

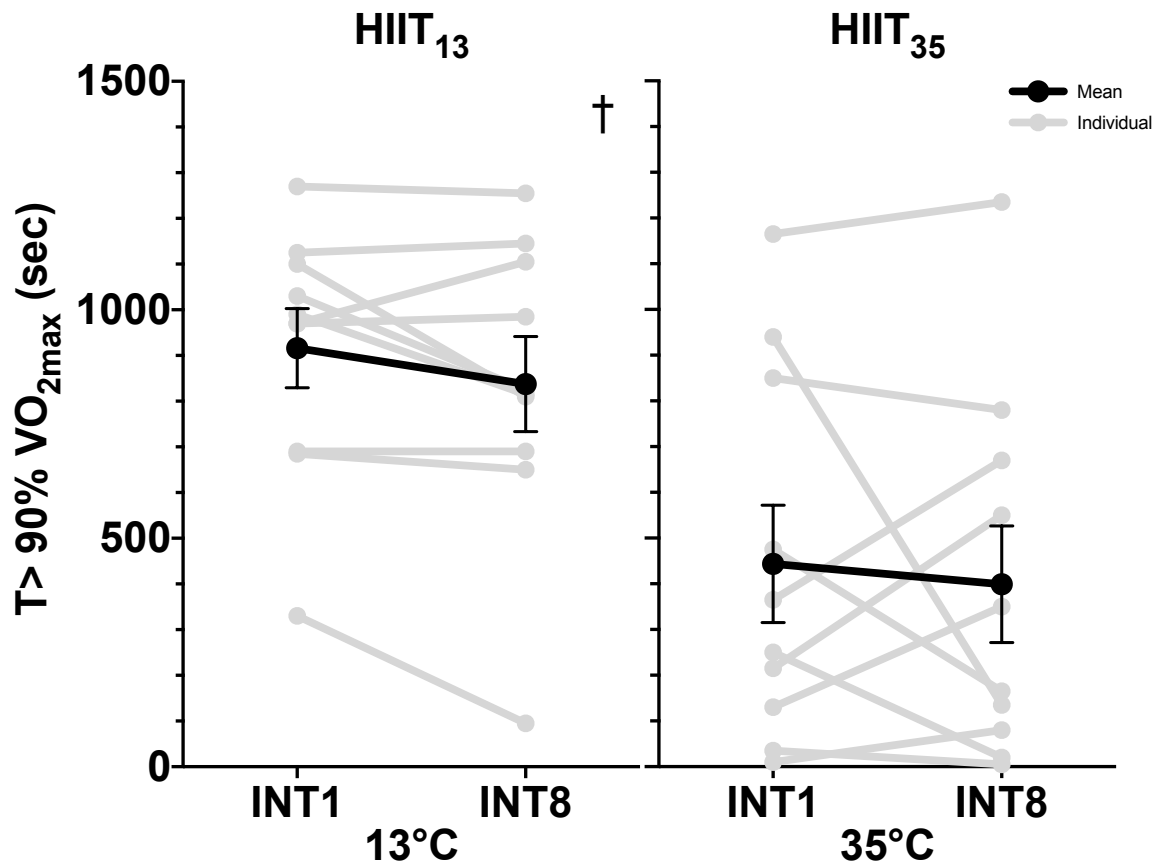


Figure 4.2 Mean \pm SEM and individual change in time above 90% VO_{2max} ($T > 90\% \text{ VO}_{2\text{max}}$) between the first (INT1) and last (INT8) session of a 4-week (8 sessions) high-intensity interval training intervention performed by 2 matched groups of trained cyclists in either 13°C (HIIT₁₃, $n = 10$) or 35°C (HIIT₃₅, $n = 10$). † $T > 90\% \text{ VO}_{2\text{max}}$ was significantly different between HIIT₁₃ (877 ± 297 sec; $P = .007$, $g = 1.278$) and HIIT₃₅ (421 ± 395 sec). No significant changes were observed for either group between INT1 and INT8

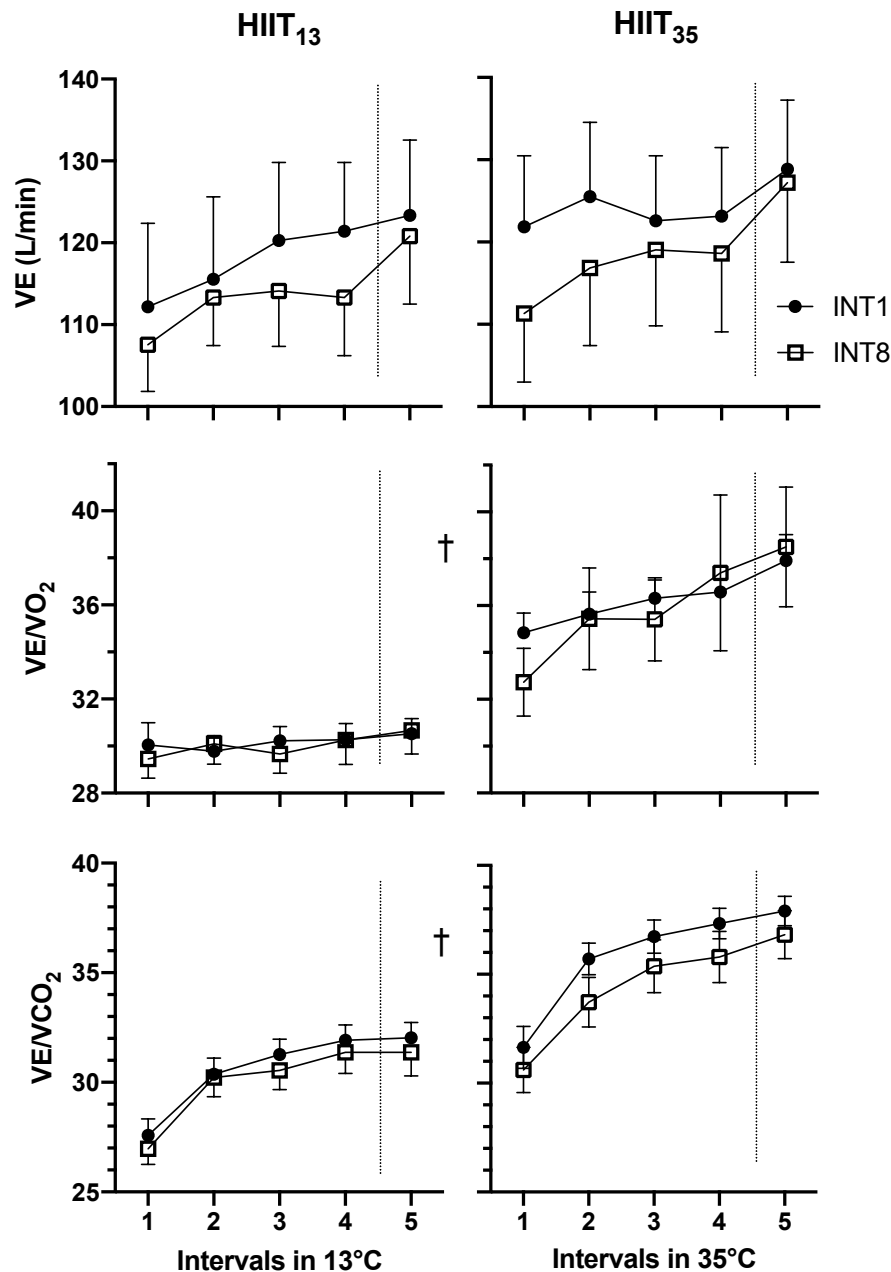


Figure 4.3 Ventilation (VE) related measures during the first (INT1) and last (INT8) session of a 4-week (8 sessions) high-intensity interval training intervention performed by 2 matched groups of trained cyclists in either 13°C (HIIT₁₃) or 35°C (HIIT₃₅). Interval sessions consisted of 5x 4-minute intervals separated by 5 minutes of rest. During the last interval participants were verbally encouraged to produce a maximal effort to help ensure the session overall was paced at their highest achievable intensity. Testing conditions were 40% relative humidity with a fan providing a simulated wind at a speed of ~28 km/hour during individual intervals. Values are mean \pm SEM. † Ventilatory equivalents for oxygen (VE/VO₂) and carbon dioxide (VE/VCO₂) were significantly different between HIIT₁₃ and HIIT₃₅.

4.4.3 Thermoregulatory Measures

Rectal temperature was not significantly different between HIIT₁₃ (n= 6) and HIIT₃₅ (n= 9; $P= .229$). An interaction for T_C was absent between groups and INT1 and INT8 ($P= .490$, $\eta_p^2= 0.044$). T_C was significantly lower in INT8, compared with INT1, in HIIT₃₅ ($P= .030$, $g= -0.544$; HIIT₁₃ $P= .801$, $g= -0.104$; Figure 4.4). T_{Sk} (HIIT₃₅ n= 8) and T_B (HIIT₁₃ n= 6, HIIT₃₅ n= 7) were higher and $T_C:T_{Sk}$ gradient (HIIT₁₃ n= 6, HIIT₃₅ n= 7) lower in HIIT₃₅, compared with HIIT₁₃. T_{Sk} , T_B , and $T_C:T_{Sk}$ gradient displayed significant interactions between groups and INT1 and INT8 ($P= .010$, $\eta_p^2= 0.345$; $P= .002$, $\eta_p^2= 0.589$; $P= .037$, $\eta_p^2= 0.339$; respectively; Figure 4.4). T_{Sk} ($P= .007$, $g= 0.821$), and T_B ($P= .014$, $g= 0.933$) significantly increased, whilst $T_C:T_{Sk}$ gradient significantly decreased ($P= .047$, $g= -1.208$) from INT1 to INT8 in HIIT₁₃. These measures were not significantly different between INT1 and INT8 in HIIT₃₅ ($g= -0.236$, $g= -0.460$, $g= 0.132$, respectively; Figure 4.4). TS was significantly higher in HIIT₃₅ compared with HIIT₁₃ ($P< .001$). TS in HIIT₃₅ significantly decreased from INT1 to INT8 ($P= .010$, $g= -0.591$; HIIT₁₃ $P= .120$, $g= 0.245$).

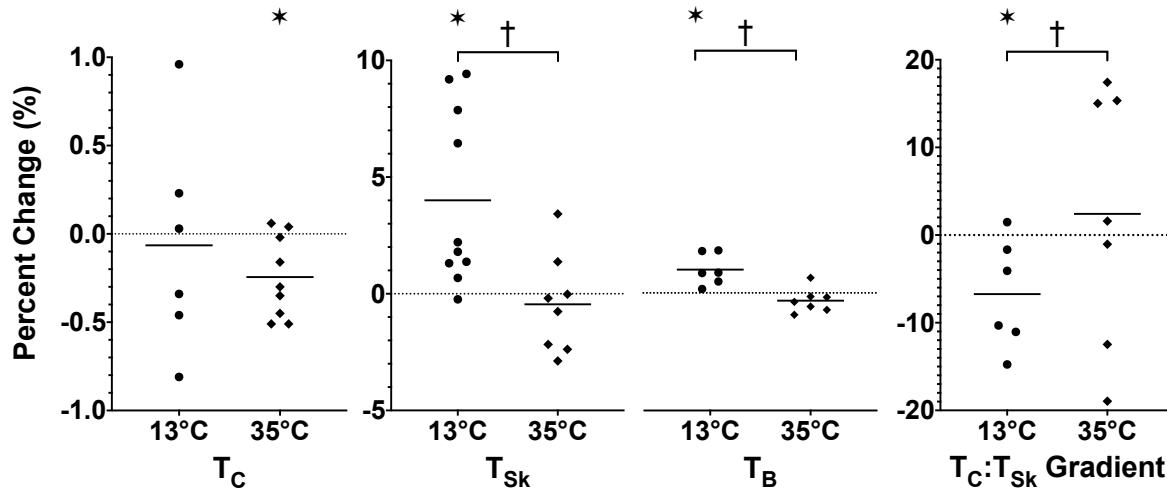


Figure 4.4 Percent change for thermoregulatory measures between the first and last session of a 4-week (8 sessions) high-intensity interval training intervention performed by 2 matched groups of trained cyclists in either 13°C (HIIT₁₃) or 35°C (HIIT₃₅). Test conditions were set to 40% relative humidity with a fan providing a simulated wind at a speed of ~28 km/hour during individual intervals. Black lines within groups represent means, n= 10 per group unless otherwise noted. core temperature (T_C), HIIT₁₃ n= 6, HIIT₃₅ n= 9; skin temperature (T_{sk}), HIIT₃₅ n= 8; body temperature (T_B), HIIT₁₃ n= 6, HIIT₃₅ n= 7; T_C:T_{sk} gradient, HIIT₁₃ n= 6, HIIT₃₅ n= 7; *Significant within subject changes. †Significant interaction between groups.

4.4.4 Mass and Water Loss

No significant change in body mass was observed between INT1 and INT8 for either group. Sweat loss did not change significantly for HIIT₁₃ ($g = 0.012$) or HIIT₃₅ ($g = 0.049$) between INT1 and INT8.

4.5 Discussion

The purpose of this study was to examine changes in physiological responses during maximal high-intensity intervals after being repeatedly performed in either hot (35°C) or cool (13°C) T_A. Herein, T > 90% VO_{2max} was significantly lower in HIIT₃₅ than HIIT₁₃ during interval training sessions, however T > 90% VO_{2max} did not change as a result of HIIT in either group (Figure 4.2). Average HR and T_C for the work intervals were not different between HIIT₃₅ and HIIT₁₃. Evidence for hyperthermia-induced hyperventilation in HIIT₃₅ was observed, but did not change after

repeated bouts. Additionally, hot HIIT is potentially a viable method for inducing heat acclimation (HA) in endurance athletes.

It has been suggested that aerobic adaptations during HIIT are stimulated most effectively when time spent at or near $\text{VO}_{2\text{max}}$ (i.e. $T > 90\% \text{VO}_{2\text{max}}$) is maximised^{23–25}, which additionally corresponds with high cardiovascular strain¹⁷⁴ (Chapter 3). In the HIIT sessions for the current study, $T > 90\% \text{VO}_{2\text{max}}$ was lower in 35°C (HIIT₃₅), compared with 13°C (HIIT₁₃; Figure 4.2), whilst HR was not different between conditions (Figure 4.1). These results are interesting as they indicate the relationship between time spent at or near $\text{VO}_{2\text{max}}$ and cardiovascular strain during HIIT is influenced by T_A . This relationship is understandable given previous research has demonstrated VO_2 and HR disassociate during hyperthermia in both high-intensity²⁶ and steady-state exercise⁵². However, employing this literature to determine the precise underlying mechanism for the relationship between VO_2 and HR during high-intensity intervals is problematic. Research examining self-paced prolonged exercise in the heat (i.e. similar in duration to a high-intensity interval session) has indicated that increased skin blood flow and corresponding decreases in cardiac output may be a mechanism for reduced VO_2 ⁵². Conversely, during maximal intensity exercise in hyperthermia (i.e. similar intensity to high-intensity intervals), reductions in VO_2 have been shown to be associated with reduced cardiac output without increases in skin blood flow²⁶. Nonetheless, the relationship between T_A and time spent at or near $\text{VO}_{2\text{max}}$ is important as it potentially gives insight into training prescription and adaptations to HIIT. A high cardiovascular strain relative to VO_2 during hot HIIT could provide improved stimulation for central cardiovascular adaptations. However, it is currently unclear if HIIT in different T_A would have an effect on aerobic performance adaptations, and is a noteworthy question for future research. Regardless, these findings indicate that time spent at or near $\text{VO}_{2\text{max}}$ differs between hot and cool

conditions, and provide justification that aerobic responses (i.e. training adaptations) and subsequent performance outcomes may differ despite intervals being performed maximally in both conditions.

Previous research has demonstrated increased ventilation occurs when humans exercise in the heat (i.e. hyperthermia-induced hyperventilation)^{114,115}. To our knowledge the current study was the first to observe evidence for hyperventilation (i.e. increased VE/VO_2 and VE/VCO_2 ; Figure 4.3) during high-intensity intervals. Previously, it has been suggested that hyperthermia-induced hyperventilation is influenced by T_C during exercise and T_{Sk} during rest¹¹⁵. However, in the current study hyperventilation was present during HIIT₃₅ while T_C was not different between hot and cool interval conditions. This relationship indicates that T_{Sk} , T_B , and (or) $T_C:T_{Sk}$ gradient may play important roles in inducing hyperventilation during high-intensity intervals in the heat. The differences in ventilation observed within the present study, despite similar T_C may be due to the intermittent nature of high-intensity intervals, and (or) the coinciding changes in convection (i.e. windspeed) that occurred between work and rest intervals.

Evidence has indicated that HA¹⁷⁶ and increased aerobic capacity¹⁷⁷ may affect hyperthermia-induced hyperventilation. However, after repeated bouts, a decrease in T_C in HIIT₃₅ (i.e. an indicator of HA; Figure 4.4) did not correspond with significant changes in VE/VO_2 and (or) VE/VCO_2 (Figure 4.3). For HIIT₃₅, only a decrease in VE approaching significance during the first interval of INT8 was observed (Figure 4.3). Of note, respiratory rates decreased in both HIIT₁₃ and HIIT₃₅, indicating a possible training effect from HIIT. This may partially explain similar effect sizes in HIIT₁₃ and HIIT₃₅ for VE between INT1 and INT8. It should also be noted that significant differences for chamber T_A were noted between INT1 and INT8 for HIIT₁₃ and HIIT₃₅. However, the magnitude of changes in T_A for HIIT₁₃ ($0.18 \pm 0.24^\circ\text{C}$, $1.4 \pm 0.9\%$) and HIIT₃₅ (0.44

$\pm 0.46^{\circ}\text{C}$, $1.3 \pm 1.3\%$) were both notably small. The influence of such minor differences in environmental temperature between INT1 and INT8 are therefore unlikely to be responsible for the lack of effect observed in ventilation as a result of the HIIT intervention. Clearly, further research is needed in order to better understand the association between ventilation and thermoregulation, during HIIT at different T_A .

Repeated exposure to heat during exercise can lead to decreases in T_C , T_{sk} , TS , and HR , and increases in $T_C:T_{sk}$ gradient and sweat loss, which help to improve endurance performance (i.e. increases power output) in the heat^{44,140}. As a result, hot HIIT is potentially a viable method for inducing HA in endurance athletes. A recent meta-analysis by Tyler et al.⁴⁴ summarized the results of heat adaptations to heat for 96 HA studies. Effect sizes from the current study for T_C , TS , and sweat loss, were within the ranges presented in the meta-analysis, and effects for decreased HR and T_{sk} were greater than the presented ranges. However, the effect size for increased performance (i.e. power output) was lower in the current study than what was presented in the meta-analysis. Previous research investigating HA in conjunction with high-intensity exercise observed decreases in performance for trained endurance athletes^{50,144}. Importantly, these previous studies utilized HA protocols with consecutive day sessions, which in turn likely resulted in cumulative fatigue, insufficient recovery, and overreaching^{50,144}. The current study utilized heat with HIIT and accounted for recovery periods between sessions to avoid overreaching and (or) overtraining⁴⁷. However, it should be noted this study was not specifically designed to measure performance outcomes in the heat after hot HIIT (i.e. with a submaximal test or time-trial in the heat^{38,44}). Future research may be needed to better assess the effectiveness of hot HIIT as a method to HA trained endurance athletes.

It has been argued that exercising in cold T_A mitigates adaptations to the condition due to the absence of whole-body temperature drop^{149,150,178}. However, in the current study, thermoregulatory adaptations (i.e. increased T_{Sk} , & T_B , and reduced $T_C:T_{Sk}$ gradient; Figure 4.4) occurred after repeated local cooling of the skin during exercise at 13°C, even when acute T_C during HIIT₁₃ was comparable to HIIT₃₅. These results for T_{Sk} have been observed in another study utilizing a protocol with consecutive day cool T_A exposures³⁸, but to our knowledge this type of adaptation has not been observed in an intermittent-day protocol spanning as long as four weeks. These findings are important as increased T_{Sk} , in conjunction with other cold adaptations, may be useful for increasing athlete tolerance¹⁵² and performance¹⁵³ while exercising in the cold and (or) cool T_A . Repeated bouts of cool intervals could provide beneficial adaptations for unacclimatized endurance athletes traveling to compete in cold environments. Conversely, increases in T_{Sk} , after relatively few exposures to cool T_A , could potentially affect aerobic performance in the heat, as it has been argued that high T_{Sk} ¹²¹ and low $T_C:T_{Sk}$ ¹⁷⁹ gradient impair such performance. Currently, the effects of thermoregulatory adaptations following repeated exposures to cool T_A on subsequent endurance performances in hot and cool conditions is not well understood.

4.5.1 Conclusions

Results from the current study indicate time spent at or near VO_{2max} was lower during 35°C HIIT than 13°C HIIT, whilst average HR for the work intervals was not different between conditions. This implies a potential dissociation between HR and the time spent at or near VO_{2max} during HIIT in hot conditions. These findings are potentially important researching the efficacy of time spent at or near VO_{2max} during HIIT as a measure for inducing adaptations for endurance performance. Evidence for hyperthermia-induced hyperventilation was present during hot HIIT, however changes in ventilation after repeated bouts in hot and cool environmental temperatures were

unclear. Findings from the current study indicate that hot HIIT (7 sessions) intermittently prescribed (~2x per week) can be potentially utilized for heat acclimation in endurance athletes. Repeated bouts of HIIT in cool environmental temperatures resulted in increases in skin & body temperature, and a decrease in core temperature to skin temperature gradient during high-intensity exercise in cool conditions.

5 CHAPTER FIVE:

STUDY 3

HOT AND COOL HIGH-INTENSITY INTERVAL TRAINING: PERFORMANCE INCREASES WITH DIFFERENT ADAPTATIONS

Link: Chapter 4 demonstrated that: 1) greater time was spent at or near $\text{VO}_{2\text{max}}$ during HIIT at 13°C versus 35°C, and 2) changes in thermoregulation response to repeated HIIT are dependent on environmental temperature. Both of these findings have the potential to affect endurance performance outcomes and subsequent physiological adaptations. Therefore, this study (Chapter 5) sought to explore power output and physiological responses during a 20 km time-trial in temperate conditions after the HIIT intervention in Study 2 (Chapter 4).

5.1 Abstract

Purpose: To determine the effect hot and cool high-intensity interval training (HIIT) interventions have on endurance performance and physiological responses in temperate conditions. **Methods:** Twenty trained cyclists and triathletes completed a 4-week (8 session) HIIT intervention at an environmental temperature (T_A) of either 13°C (HIIT₁₃) or 35°C (HIIT₃₅). Interval sessions consisted of five maximal 4-minute intervals with 5 minutes of recovery between each. Participants completed submaximal warm-ups and 20 km time-trials (TT) in temperate conditions (22°C) before (TT1) and after (TT2) the HIIT intervention. Gross mechanical efficiency (GME) was measured during the warm-up (at 50% peak power output), whilst power and heart rate (HR) were measured during the 20 km TT; rate of perceived exertion (RPE) and body temperature (T_B) were measured through the warm-up and TT. **Results:** 20 km TT power output increased in both groups between TT1 and TT2 (HIIT₁₃ $P = .023$, $g = 0.163$; HIIT₃₅ $P = .003$, $g = 0.336$), with no significant interactions or differences between groups noted for power ($P = .115$, $\eta_p^2 = 0.132$; $P = .421$, respectively), HR ($P = .747$, $\eta_p^2 = 0.006$; $P = .535$), or RPE ($P = .126$, $\eta_p^2 = 0.126$; $P = .658$). However, within subject increases for HR and RPE during the 20 km TT were noted in HIIT₁₃ ($P = .025$, $g = 0.398$; $P = .013$; respectively), but not in HIIT₃₅ ($P = .342$, $g = 0.183$; $P = .405$). GME approached a significant decrease ($P = .051$, $g = -0.744$) in HIIT₁₃. A significant interaction in T_B was observed between groups and TT1 and TT2 during both the 20 km TT ($P = .042$, $\eta_p^2 = 0.263$) and submaximal warm-up ($P = .008$, $\eta_p^2 = 0.382$). **Conclusions:** These results indicate that 20 km TT performance at 22°C is increased after 4 weeks of HIIT in both 13°C and 35°C, with no differences between groups. However, changes in cardiorespiratory, thermoregulatory, and subjective responses during temperate endurance exercise are dependent on the T_A HIIT is performed in.

5.2 Introduction

High-intensity interval training (HIIT) stresses the oxygen transport and utilization systems⁵⁷, whereby maximal cardiac output is approached⁶³ (central) and large motor units are recruited (peripheral)⁶⁴. This stress stimulates adaptations important for maximizing endurance performance (e.g. increased maximal oxygen consumption ($\text{VO}_{2\text{max}}$), ventilatory thresholds, and anaerobic capacity^{22,66}). It has been suggested that aerobic adaptations during HIIT are stimulated most effectively when time spent at or near $\text{VO}_{2\text{max}}$ is maximised^{23–25}. Indeed, given the relatively short time commitment and the important benefits of HIIT, optimizing the conditions under which this training is performed could potentially enhance adaptation and endurance performance in a practical and cost-benefit manner.

During exercise in hot T_A , an increase in skin blood flow occurs⁵² in order to increase dissipation of metabolic heat waste to the environment. It is believed this shunting of blood to the skin reduces muscle blood flow causing an increase in HR for a given workload^{52,180}. Conversely, performing high-intensity intervals in cooler conditions increases core temperature (T_C) to skin temperature (T_{Sk}) gradient¹⁷⁴, reducing skin blood flow^{17,180}, increasing venous return, and therefore potentially enhancing the proportion of cardiac output delivered to the working muscles¹⁷. HIIT in the heat may therefore induce central adaptations, but compromise peripheral adaptations compared to HIIT performed in cool T_A . Indeed, a study by Gifford et al.¹⁵⁴ demonstrated that $\text{VO}_{2\text{max}}$ in trained endurance athletes was limited by O_2 supply to the working muscles (i.e. cardiac output), implying that in order to maximize key peripheral aerobic adaptations in trained endurance athletes with HIIT, blood flow to the working muscles should be maximized. Supporting the premise that greater muscle blood flow may enhance performance outcomes from HIIT (e.g. power output during a

time-trial; TT) are results indicating increased amounts of muscle blood flow to the exercising muscles during single-leg cycling training³⁰ stimulated a superior oxidative potential and metabolic profile of the skeletal muscle compared to double-leg cycling³¹. Interestingly, Chapter 4 demonstrated a significantly higher amount of time spent at or near $\text{VO}_{2\text{max}}$ during intervals performed at 13°C versus 35°C, indicating a potential for superior aerobic adaptations from HIIT performed in cool conditions. However, it is not well understood if differences in peripheral oxygen transport and utilization associated with hot and cool HIIT would affect subsequent endurance performance outcomes.

Endurance training performed in hot conditions has been demonstrated to increase performance in temperate conditions³⁶. This performance benefit is believed to be from heat acclimation (HA) adaptations (e.g. increases in plasma & improved sweat response) that attenuate thermal limiters and their negative effects on performance. Lorenzo et al³⁸. observed greater increases in power output and $\text{VO}_{2\text{max}}$ under temperate conditions with highly trained endurance athletes following 10 days in 40°C conditions at a submaximal intensity (2x 45 minutes at 50% $\text{VO}_{2\text{max}}$), compared with a control group. Conversely, a study of similar design conducted by Keiser et al³⁵. demonstrated no change in time-trial performance or $\text{VO}_{2\text{max}}$ when cardiovascular strain between groups (18°C versus 38°C) during training was similar. Collectively, these results provide evidence that additional cardiovascular load caused by training in the heat is potentially a key component of adaptations that increases endurance performance in temperate conditions. It is plausible that the incorporation of heat stress with HIIT could induce greater cardiovascular adaptations than HIIT in cooler conditions leading to improved performance. Indeed, multiple studies have been conducted that have integrated heat with HIIT^{50,54,55,144}. However, to our knowledge, no studies investigating the effects of T_A on HIIT have incorporated trained endurance

athletes performing maximal high-intensity intervals that were selected specifically for their time at or near $\text{VO}_{2\text{max}}$.

From this synopsis it can be recognized that a number of contradictory and complementary physiological factors can potentially affect the performance outcomes following HIIT in either hot or cool conditions. Therefore, the overall purpose of this study was to determine the effect 4 weeks of HIIT performed at 13°C or 35°C would have on endurance performance and physiological responses in temperate conditions (22°C). It was postulated that the benefits of more time spent at or near $\text{VO}_{2\text{max}}$ during HIIT at 13°C would exceed the potential benefits of HA during HIIT at 35°C. Subsequently, it was hypothesized that HIIT performed in 13°C would result in an increase in power output during a simulated 20 km TT and gross efficiency during a submaximal test that would exceed the outcomes of HIIT performed in 35°C.

5.3 Methods

5.3.1 Participants

Twenty-one male and female trained cyclists and triathletes (De Pauw et al. 2013¹⁹) were recruited from the Perth, Western Australia region for this study. Twenty were included for final analysis as a single participant did not complete the study protocol due to schedule conflicts (detailed description of participants can be found in Chapter 4). Participants were assigned to one of two groups that were matched for $\text{VO}_{2\text{max}}$, peak power output (PPO), 20 km TT performance, and biological sex (Table 5.1). A 2 tailed t-test showed no significant differences in the characteristics of the two experimental groups. Seven males and three females were included in each group. All participants signed a written informed consent prior to data collection. The Edith Cowan University Human Research Ethics Committee approved the study prior to commencement.

5.3.2 *Study Overview*

This study examined the effects the HIIT interventions from Chapter 4 had on endurance performance and physiological responses in temperate conditions. During the initial testing session all participants height, weight and body composition were assessed before completing a graded exercise test (GXT) to determine $\text{VO}_{2\text{max}}$ and PPO (described in Chapter 4). Participants partook in a 4-week HIIT intervention at a T_A of either 13°C (HIIT₁₃) or 35°C (HIIT₃₅; described in Chapter 4). The high-intensity interval session protocol was chosen for its demonstrated time above $\text{VO}_{2\text{max}}$ ($T > 90\% \text{VO}_{2\text{max}}$)^{24,174}. Prior to the intervention participants completed two simulated TTs: a familiarization and pre-test (TT1). These test sessions were separated from each other and the first interval session by at least 48 hours. A final TT (TT2) was conducted 7-9 days after the last interval session (Figure 5.1). Participants were instructed to prepare themselves as if approaching a competitive event the days between the intervention end and TT2. Test sessions (GXT, familiarization TT, TT1, & TT2) were performed on an electromagnetically-braked cycling ergometer (Velotron, Racermate, Redmond, WA, U.S.A.). Prior to all testing sessions subjects were asked to refrain from strenuous exercise in the 36 hours prior, eating large meals in the 3 hours prior, and consuming non-habitual caffeine in the 24 hours prior. Participants were instructed to eat and drink as they would before a competitive event and record their consumption in a log prior to the first testing session in order to replicate their dietary consumption 24 hours prior to subsequent sessions.

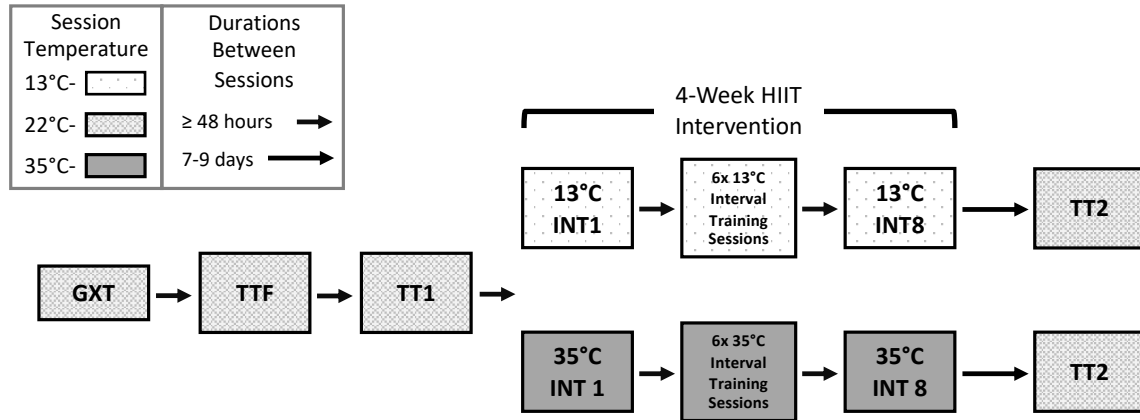


Figure 5.1 Experimental design. GXT indicates graded exercise test; TTF, familiarization time-trial; TT1, pre intervention time-trial; INT1, 1st interval testing session; INT8, last interval session and 2nd interval testing session; TT2, post intervention time-trial.

5.3.2.1 Time-trial Sessions

During the TT sessions participants performed a 3-minute resting baseline, a 10-minute standardized submaximal warm-up, 5 minutes of rest, and then a flat 20 km TT (Figure 5.2). During the warm-up participants cycled at 50% PPO followed by 75% PPO for 5-minutes. Environmental conditions for the TT sessions were set at 22°C and 40% relative humidity. A fan provided a simulated wind at a speed of ~28 km/hour during the 20 km TT. Participants started the 20 km TT a virtual gearing of 56x17, but were allowed to self-select cadence and gearing once the TT began. Participants were instructed to pace the 20 km TT at the highest average power output possible with the goal of traveling the virtual distance in the least amount of time. Participants were blinded to their power output, HR, speed, and time lapsed while completed distance was displayed. Participants were verbally encouraged to perform at their best and allowed to consume water ad libitum during the course of the 20 km TT. Power output (Velotron software; recorded at 1 Hz) was analysed using cycling power analytics software (GoldenCheetah, version

3.4, 2016). Power output, HR, whole body perceived exertion¹⁶⁷ (RPE), thermal sensation¹⁶⁸ (TS), and participant temperature measures (described below) were recorded and analysed for every 5 km of the 20 km TT. During the warm-up, respiratory, participant temperature, and HR measures were continuously collected and analysed for the last 30 seconds of each stage (50% PPO & 75% PPO). RPE and TS were recorded at 4.5 and 9.5 minutes into the submaximal warm-up. Gross mechanical efficiency (GME) was calculated¹⁸ for the 50% PPO segment of the submaximal warm-up. Respiratory, participant temperature, and HR were recorded continuously during the pre-test resting baseline. Resting HR was determined as the lowest 30-second average during the 3-minute rest. TS was also recorded during the rest prior to warm-up.

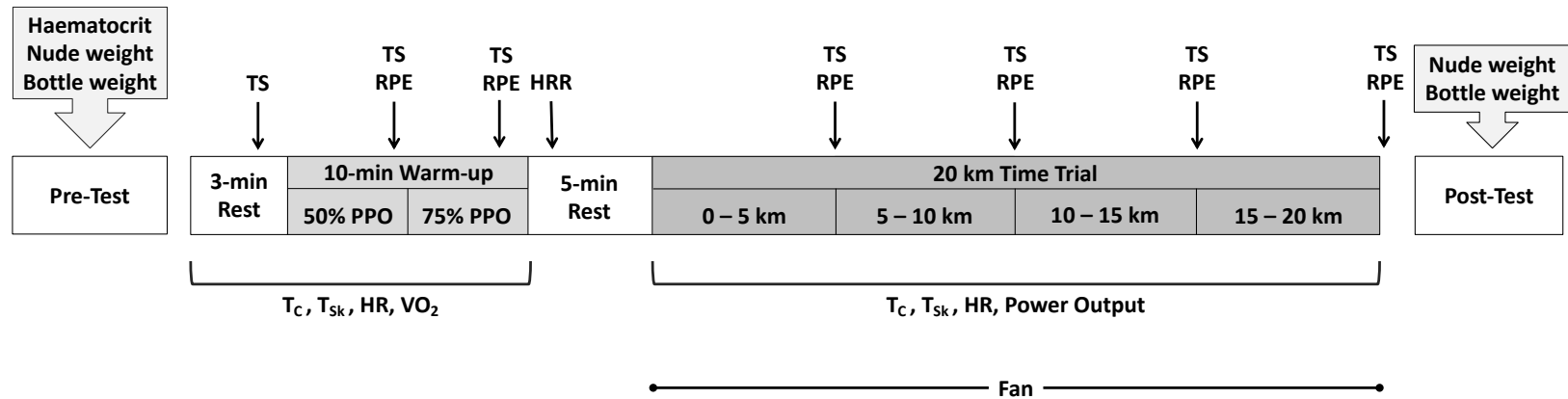


Figure 5.2 Time-trial testing protocol. PPO indicates peak power output; TS, thermal sensation; RPE, rate of perceived exertion; HRR, heart rate recovery; T_c , core temperature; T_{sk} , skin temperature; HR, heart rate; VO_2 , oxygen consumption.

RPE and TS were recorded using visual numerical scales. HR was recorded (0.2 Hz) and averaged over 5-second intervals. In order to minimize data loss, HR was measured via Polar (primary method, Polar Precision Performance SW5.20, Polar Electro, Kempele, Finland), Parvomedics (1 session), and Garmin (2 sessions; Edge 810, Garmin Ltd., Olathe, KS, USA) systems. Linear modelling was used to replace 36 HR data points lost (180 seconds) during the steady-state of 1 20 km TT. Heart rate recovery (HRR) was measured at the end of the warm-up and calculated as the difference between the HR at the end of warm-up and the HR at the end of the subsequent 1-minute rest¹⁶⁹. T_C and T_{Sk} were measured continuously throughout TT1 and TT2 (Squirrel SQ2020 Data Logger, Grant Instruments, Shepreth Cambridgeshire, UK), but not during the familiarization TT. T_C was measured using a disposable rectal thermometer (Monatherm Thermistor 400 Series, Mallinckrodt Medical, St. Luis, MO, USA) inserted ~10 cm past the anal sphincter. T_{Sk} was measured via 4 thermistors (YTS Temperature, 400 Series, Dayton, Ohio) placed on the chest (T_{chest}), bicep (T_{arm}), thigh (T_{thigh}), and calf (T_{leg}) and mean T_{Sk} and was calculated¹⁷⁰ using the following formula:

$$T_{Sk} = 0.3 (T_{chest} + T_{arm}) + 0.2 (T_{thigh} + T_{leg})$$

T_C and T_{Sk} measurements were used to calculate mean body temperature (T_B) and the $T_C:T_{Sk}$ gradient. T_B was calculated using the formulas from Colin 1971¹⁷¹ for neutral (13°C & 22°C) and hot (35°C) conditions, respectively:

$$T_B = 0.66 \times T_C + 0.34 \times T_{Sk}$$

$$T_B = 0.79 \times T_C + 0.21 \times T_{Sk}$$

Test sessions measuring T_C and T_{sk} temperature were performed at the same time of day for each participant to avoid circadian body temperature variations¹⁶⁶. Fluctuations in temperature data of greater than 0.10°C/second were removed.

Hematocrit measurements were taken as a double baseline prior to the intervention (separated by a minimum of 48 hours and averaged together for analysis) and the TT2 before exercise testing. Finger prick blood for hematocrits was collected via capillary (100 Capilette, 955 05 32 02, Selzer Labor Technik, Mannheim-Heidelberg-Karlsruhe, Germany), spun at 3500 rpm for 5 minutes (Centrifuge MPW-212, MPW Medical Instruments, Thebarton, South Australia), and assessed by the same tester on all participants.

5.3.3 *Statistics*

Values are reported as mean \pm standard deviation. Sample number is indicated where a full sample ($n=10$) was not obtained for a group due to sampling error. A two-way repeated measure ANOVA was used to compare all between group measures and within subject measures recorded during the TTs. A 2 tailed paired t-test was used to compare within subject measures recorded at rest and during the warm-up. Significance was determined at a confidence level of 95% ($P < .050$). When sphericity was violated a Greenhouse-Geisser correction was utilized. Where significant differences were observed, Bonferroni corrections for multiple comparisons were used to locate where differences existed. Cohen's d with a Hedges' g correction (g) and partial eta squared (η_p^2) were reported as effect sizes. Hedges' g was calculated via a statistical spreadsheet (Jared DeFife, PhD, Emory University, 2009). SPSS software version 25.0 (IBM, Armonk, New York) was utilized to calculate η_p^2 and analyze data.

5.4 Results

The training hours for HIIT₁₃ and HIIT₃₅ during the 4-week HIIT intervention were 12.73 ± 4.96 hours/week and 11.10 ± 4.83 hours/week ($n = 9$), respectively ($P = .479$). TT sessions were conducted at $22.33 \pm 0.29^\circ\text{C}$, $56 \pm 7\%$ RH.

5.4.1 20 km Time-trial

No significant differences between groups or interactions were noted for power output ($P = .421$; $P = .115$, $\eta_p^2 = 0.132$, respectively). However, power output significantly increased from TT1 to TT2 in both groups (HIIT₁₃, $3.3 \pm 3.4\%$, $P = .023$, $g = 0.163$; HIIT₃₅, $7.3 \pm 6.3\%$, $P = .003$, $g = 0.336$; Figure 5.3). Significant interactions for power output between TT1 and TT2 and 5 km TT split were also noted in both HIIT₁₃ ($P = .005$) and HIIT₃₅ ($P = .050$; Figure 5.4). No significant differences between groups or interactions were noted for HR (HIIT₁₃ $n = 9$, $P = .535$; $P = .747$, $\eta_p^2 = 0.006$), or RPE ($P = .658$; $P = .126$, $\eta_p^2 = 0.126$). HR and RPE increased from TT1 to TT2 in HIIT₁₃ ($P = .025$, $g = 0.398$; $P = .013$; respectively), but not in HIIT₃₅ ($P = .342$, $g = 0.183$; $P = .405$; Figure 5.4). For additional 20 km trial-trial results see Appendix F.

Core temperature (HIIT₃₅ $n = 8$), T_{sk} (HIIT₁₃ $n = 9$, HIIT₃₅ $n = 8$), and $T_{\text{C}}:T_{\text{sk}}$ gradient (HIIT₁₃ $n = 9$, HIIT₃₅ $n = 7$) were not significant between HIIT₁₃ and HIIT₃₅ during the 20 km TT, nor were any significant interactions noted ($P = .127$, $\eta_p^2 = 0.139$; $P = .241$, $\eta_p^2 = 0.090$; $P = .369$, $\eta_p^2 = 0.058$, respectively). A significant interaction in T_{B} (HIIT₁₃ $n = 9$, HIIT₃₅ $n = 7$) was observed between groups and TT1 and TT2 ($P = .042$, $\eta_p^2 = 0.263$; Figure 5.5). T_{S} was higher in both TT1 and TT2 during HIIT₃₅, compared with HIIT₁₃ ($P = .027$), while no significant interaction was noted ($P = .401$).

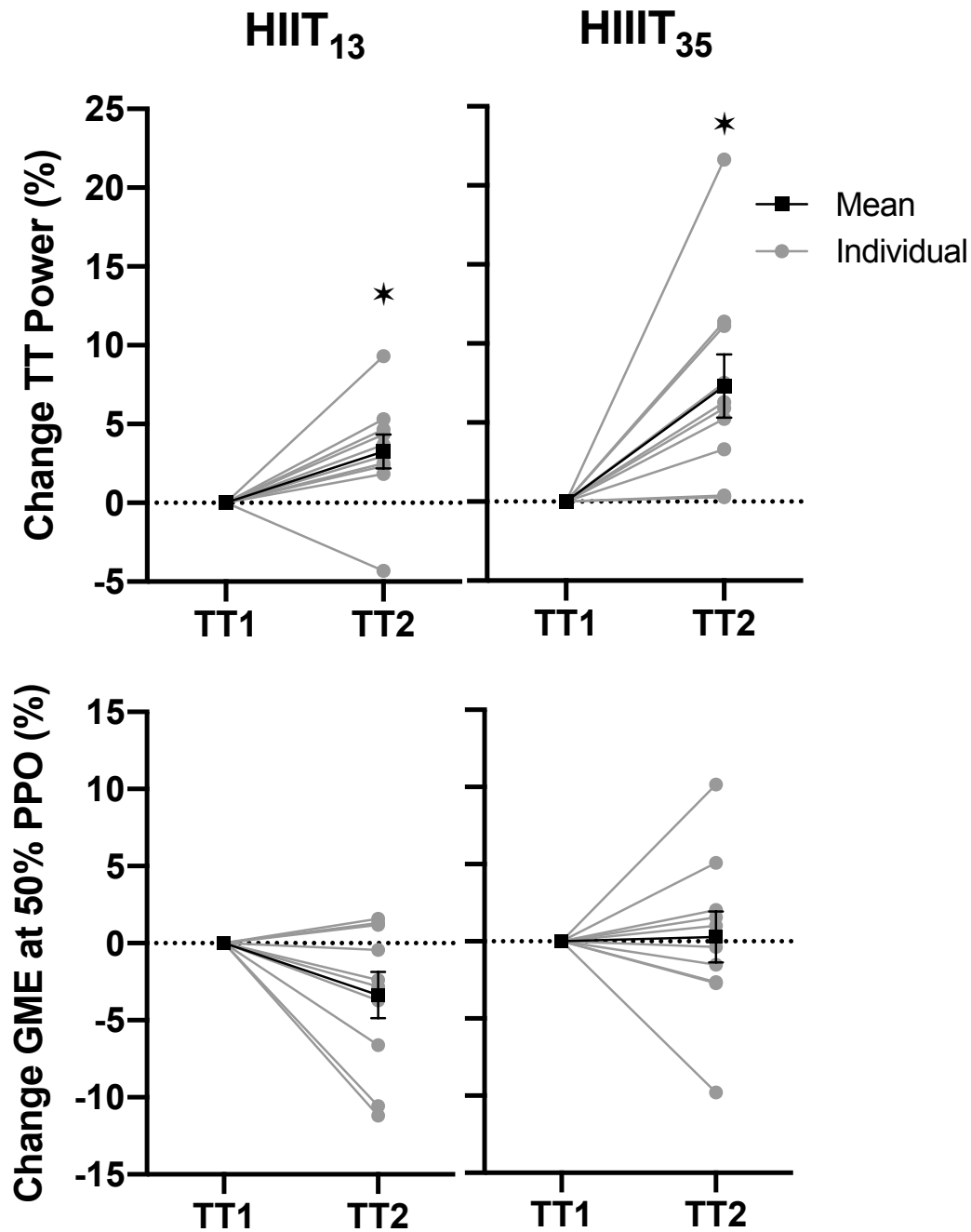


Figure 5.3 Percent change for power output during a 20 km time-trial (TT) and gross mechanical efficiency (GME) during a 50% peak power output (PPO) warm-up pre (TT1) and post (TT2) a 4-week (8 sessions) high-intensity interval training intervention performed in either 13°C (HIIT₁₃) or 35°C (HIIT₃₅). TT conditions were set to 22°C and 40% relative humidity with a fan providing a simulated wind at a speed of ~28 km/hour. The fan was not utilized during the warm-up. Mean values are mean \pm SEM, n= 10 per group *Significant within subject change. Percent change for GME within HIIT₁₃ approached significance (P= .051)

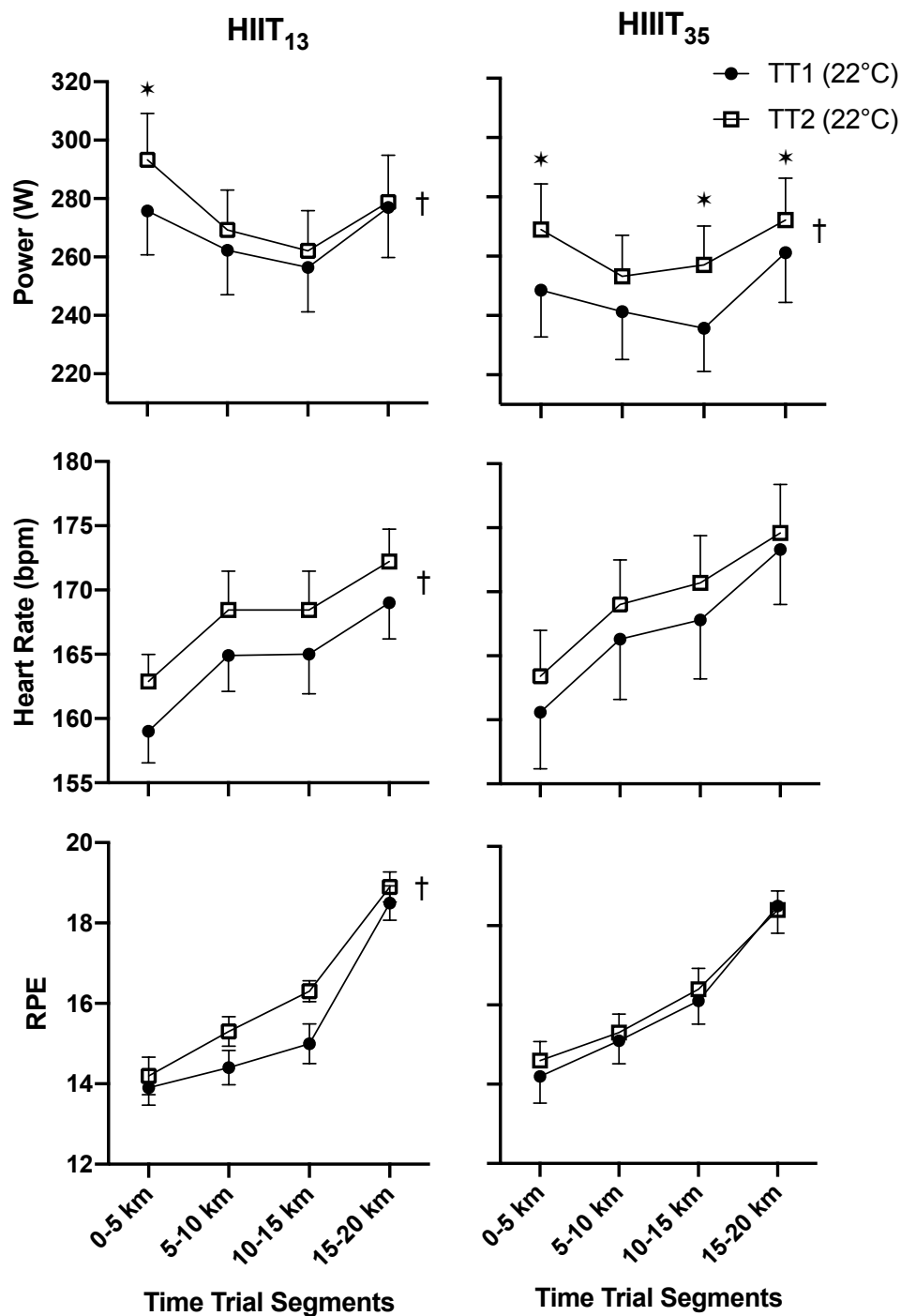


Figure 5.4 Power output, heart rate, & rate of perceived exertion (RPE) during 20 km time-trials (at 22°C) pre (TT1) and post (TT2) 4-weeks (8 sessions) of high-intensity interval training performed by 2 matched groups of trained cyclists in either 13°C (HIIT₁₃) or 35°C (HIIT₃₅). Testing conditions were set to 22°C and 40% relative humidity with a fan providing a simulated wind at a speed of ~28 km/hour. Values are mean \pm SEM, n= 10 per group except within heart rate in HIIT₁₃ (n= 9). *Significant differences between time-trial segments in TT1 versus TT2. bpm, beats per minute. †Significant within subject change.

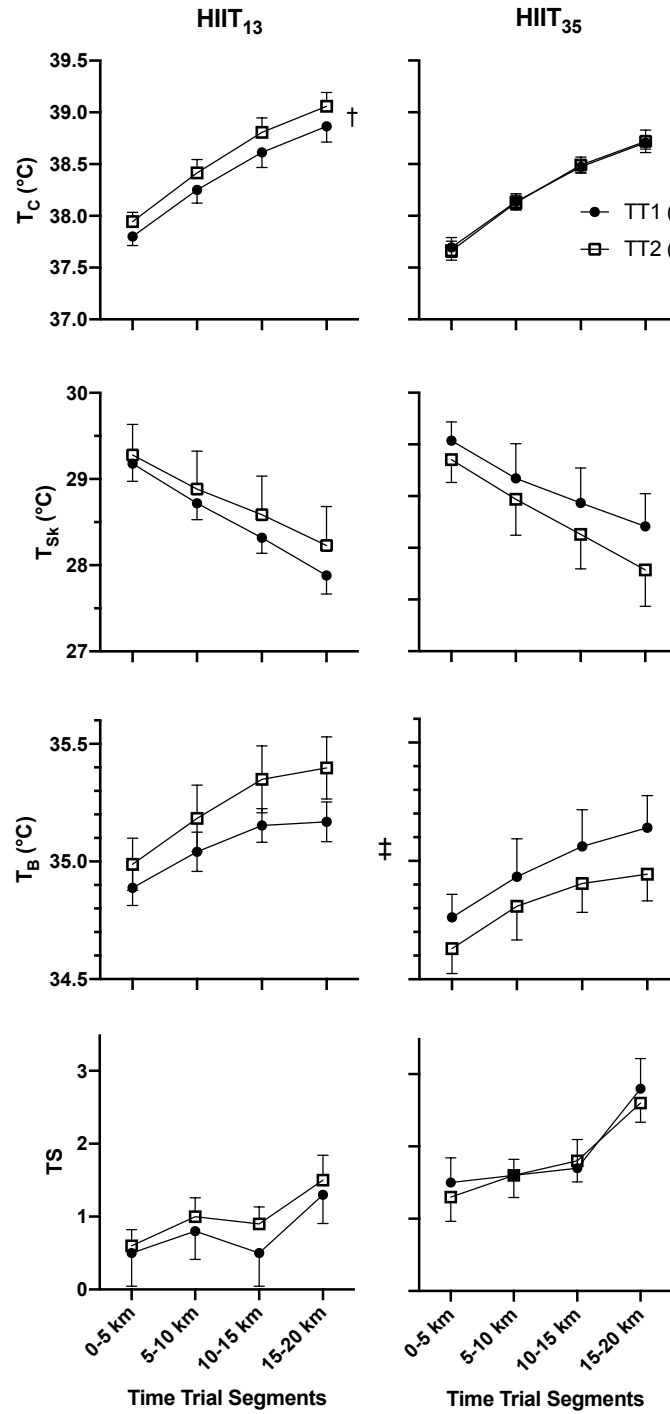


Figure 5.5 Thermoregulatory measures during 20 km time-trials pre (TT1) and post (TT2) 4-weeks (8 sessions) of high-intensity interval training performed by 2 matched groups of trained cyclists in either 13°C (HIIT₁₃) or 35°C (HIIT₃₅). Testing conditions were set to 22°C and 40% relative humidity with a fan providing a simulated wind at a speed of ~28 km/hour. Values are mean \pm SEM, $n = 10$ per group unless otherwise noted. Core temperature (T_c), HIIT₃₅ $n = 8$; skin temperature (T_{sk}), HIIT₁₃ $n = 9$, HIIT₃₅ $n = 8$; body temperature (T_b), HIIT₁₃ $n = 9$, HIIT₃₅ $n = 7$. TS indicates thermal sensation. No significant differences between time-trial segments in TT1 versus TT2 were noted for thermoregulatory measures. †Significant within subject change. ‡ Significant interaction between groups and TT1 versus TT2.

5.4.2 Submaximal Warm-up

No significant interactions or main effects were observed for cardiorespiratory, thermoregulatory, and subjective measures during the 75% PPO of the warm-up. GME was not different between HIIT₁₃ and HIIT₃₅ during 50% PPO ($P = .922$), and there was absence of interaction between the groups and TT1 and TT2 ($P = .125$, $\eta_p^2 = 0.126$; Figure 5.6). The decrease in GME approached significance in HIIT₁₃ ($P = .051$, $g = -0.744$; Figure 5.3). VO₂ significantly increased in HIIT₁₃ ($P = .041$, $g = 0.333$), but not in HIIT₃₅ ($P = .876$, $g = 0.020$). RPE was significantly higher in HIIT₃₅ compared to HIIT₁₃ during 50% PPO of both TT1 and TT2 warm-ups ($P = .005$). No significant changes in HRR recorded 1-minute after the submaximal warm-up were found.

Core temperature, T_{sk} (HIIT₁₃ $n = 9$, HIIT₃₅ $n = 8$), T_B (HIIT₁₃ $n = 9$, HIIT₃₅ $n = 8$), T_C:T_{sk} gradient (HIIT₁₃ $n = 9$, HIIT₃₅ $n = 8$), and TS during 50% PPO were not significantly different between groups. However, significant interactions were observed between groups and time-trials for T_{sk} ($P = .003$, $\eta_p^2 = 0.462$), T_B ($P = .008$, $\eta_p^2 = 0.382$), T_C:T_{sk} gradient ($P = .045$, $\eta_p^2 = 0.242$), and TS ($P = .009$, $\eta_p^2 = 0.323$; Figure 5.6). The interaction for T_C approached significance ($P = .062$, $\eta_p^2 = 0.181$) where the measure was lower in HIIT₃₅ relative to the HIIT₁₃ in TT2. Within subject temperature measures for the warm-up are displayed in Figure 5.6.

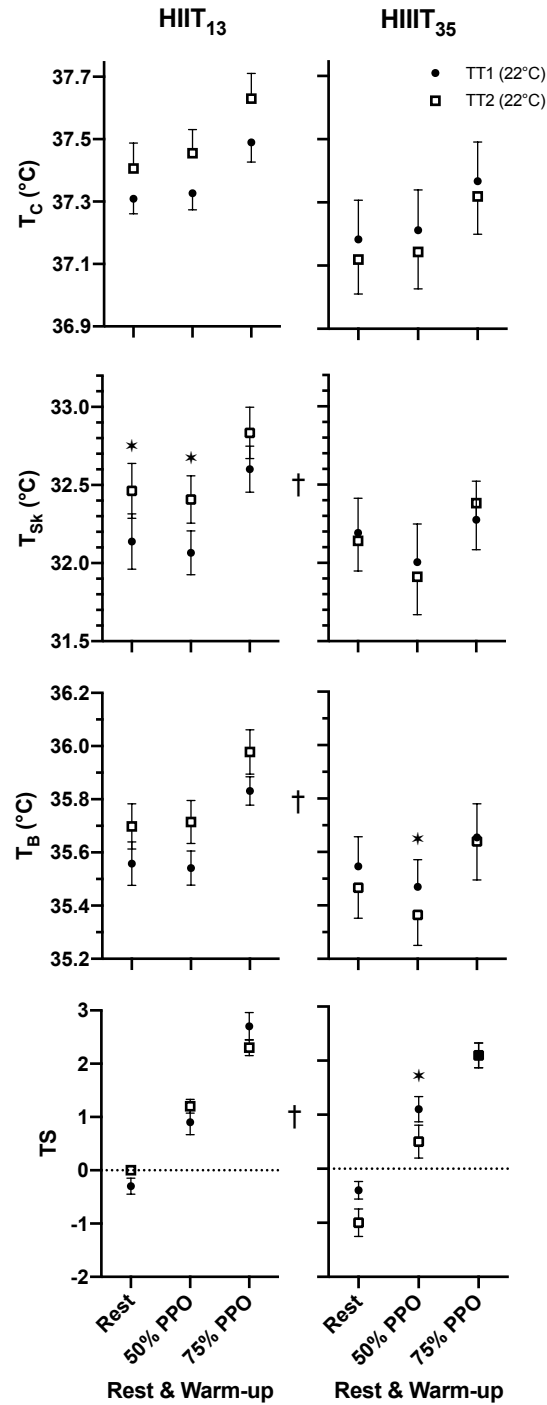


Figure 5.6 Thermoregulatory measures during rest and a submaximal warm-up pre (TT1) and post (TT2) 4-weeks (8 sessions) of high-intensity interval training performed by 2 matched groups of trained cyclists in either 13°C (HIIT₁₃) or 35°C (HIIT₃₅). The warm-up consisted of 5 min at 50% peak power output (PPO) followed by 5 min at 75% PPO. Testing conditions were set to 22°C and 40% relative humidity with no fan operating. Values are mean ± SEM, n= 10 per group unless otherwise noted. T_C indicates core temperature. Skin temperature (T_{sk}) and body temperature (T_B): rest HIIT₁₃ n= 9, HIIT₃₅ n= 9; 50% PPO HIIT₁₃ n= 9, HIIT₃₅ n= 8; 75% PPO HIIT₁₃ n= 9, HIIT₃₅ n= 8. TS indicates thermal sensation. *Significant within subject differences between TT1 versus TT2. † Significant interaction between groups and TT1 versus TT2 for rest and 50% PPO.

5.4.3 Resting Measures

Resting HR (13°C n= 9, 35°C n= 9) prior to the submaximal warm-up was not significantly different between groups ($P= .246$, $P= .353$, respectively), nor was a significant interaction noted ($P= .843$, $\eta_p^2= 0.002$; $P= .318$, $\eta_p^2= 0.062$). Effects for resting HR in HIIT₁₃ and HIIT₃₅ were $g= 0.350$ and $g= -0.028$, respectively.

Resting T_C was not significant between HIIT₁₃ and HIIT₃₅ ($P= .113$), nor was a significant interaction noted ($P= .140$, $\eta_p^2= 0.117$). Effects sizes for resting T_C between TT1 and TT2 in HIIT₁₃ and HIIT₃₅ were $g= 0.439$ and $g= -0.159$, respectively. Resting T_{Sk} , T_B , and $T_C:T_{Sk}$ (n= 9 for all respective measures in HIIT₁₃ & HIIT₃₅) were not different between groups ($P= .619$, $P= .373$, $P= .932$, respectively). Significant interactions were observed for resting TS, T_{Sk} , T_B , and $T_C:T_{Sk}$ gradient ($P= .017$, $\eta_p^2= 0.278$; $P= .008$, $\eta_p^2= 0.362$; $P= .027$, $\eta_p^2= 0.270$; $P= .049$, $\eta_p^2= 0.220$; respectively). T_{Sk} and T_B were higher in TT2 for HIIT₁₃ than HIIT₃₅ (Figure 5.6). Resting T_{Sk} increased (Figure 5.6) and $T_C:T_{Sk}$ decreased ($P= .005$, $g= -0.518$) in HIIT₁₃ significantly between TT1 and TT2.

5.4.4 Body Mass, Fluids, & Hematocrit

Body mass measured prior to testing was not significantly different between the TT1 and TT2 ($P= .194$), HIIT₁₃ and HIIT₃₅ ($P= .914$), nor was there a significant interaction ($P= .991$, $\eta_p^2 < 0.001$). There was an absence of significant differences for all water consumption and sweat loss analyses. Effects between TT1 and TT2 for sweat loss in HIIT₁₃ and HIIT₃₅ were $g= 0.301$ and $g= 0.146$, respectively. Hematocrit was not different between HIIT₁₃ and HIIT₃₅ and an interaction was absent between groups and TT1 and TT2. However, hematocrit levels for HIIT₁₃ significantly increased from $42 \pm 3\%$ to $44 \pm 3\%$ ($P= .008$, $g= 0.655$).

5.5 Discussion

The purpose of this study was to determine the effect of 4 weeks of HIIT performed at 13°C or 35°C on endurance performance and physiological responses in temperate conditions (22°C). The primary finding from this study was that, contrary to our hypothesis, the observed improvement in 20 km TT performance after HIIT was not significantly different between HIIT₁₃ and HIIT₃₅. Additionally, evidence for HA adaptations during temperate TT performance after HIIT in 35°C was unclear; and thermoregulatory adaptations to cool T_A after HIIT in 13°C potentially attenuate performance outcomes in temperate conditions.

It has been suggested that performance improvements from aerobic HIIT are most effective when time at or near VO_{2max} is maximised^{23–25}. In contrast to this claim, no difference in increased TT power output was observed between HIIT₁₃ and HIIT₃₅, despite a demonstrably lower $T > 90\%$ VO_{2max} in HIIT at 35°C (Chapter 4). Indeed, power output during the 20 km TT significantly increased for HIIT₃₅ (Figure 5.3) by an effect that was twice the size of what was observed in HIIT₁₃ (Figure 5.4). This occurred despite lower power output noted during HIIT at 35°C¹⁷⁴ (Chapter 3), which is thought to be associated with a lower oxygen delivery to the working muscles²⁶. Conversely, HR and T_C values during hot HIIT are similar to or higher¹⁷⁴ than values noted in cooler HIIT (Chapter 3 & 4). As a result, it appears from the current study, that cardiovascular strain and (or) T_C during HIIT at 13°C and 35°C (Chapter 4) may have stimulated the necessary adaptations for increased TT performance, despite $T > 90\%$ VO_{2max} differences during the intervention (Chapter 4). Consequently, cardiovascular and (or) thermal loads during HIIT may be as (or more) important as time at or near VO_{2max} for stimulating ergogenic adaptations. Indeed, it has been previously noted that a long time at or near VO_{2max} may not optimally stimulate physiological components limiting VO_{2max} in all cases^{24,57}. However, it is also

important to note that the difference in time spent at or near $\text{VO}_{2\text{max}}$ observed in this study may not be physiologically meaningful. For example, training at a running velocity of 90% $\text{VO}_{2\text{max}}$ versus 100% $\text{VO}_{2\text{max}}$ has demonstrated no differences in subsequent performance outcomes⁸⁷. Indeed, despite intervals in 35°C having a lower $T > 90\% \text{VO}_{2\text{max}}$ than in 13°C, the $T > 90\% \text{VO}_{2\text{max}}$ for HIIT₃₅ was still comparable to values observed in other HIIT studies^{181–184}.

Previous research has demonstrated that repeated bouts of training in the heat can increase endurance performance outcomes better than training in cool conditions^{38,55}. This research could provide a potential mechanism to explain the similar performance increases observed between groups, when time spent at or near $\text{VO}_{2\text{max}}$ was lower during HIIT in 35°C. However, HA as an ergogenic aid in temperate conditions is debated in the literature^{37,135}. Indeed, evidence for HA as an ergogenic aid in temperate conditions is typically demonstrated in experiments without control groups^{1,39,155–157}, or in matched group experiments under specific circumstances^{38,55,158} (discussed further below). In the current study, sufficient evidence was not available to confirm or dismiss the hypothesis that similar TT performance improvements in HIIT₁₃ and HIIT₃₅ were due to HA compensating for lower $T > 90\% \text{VO}_{2\text{max}}$ during 35°C HIIT. Previous research demonstrating temperate performance increases after heat exposure have suggested increased plasma volume and improved sweat response as potential mechanisms to explain this outcome^{36,38,55}. The current study did not measure plasma volume directly. However, a decrease in hematocrit nor an increase in participant mass (i.e. indicating a potential increase in plasma volume or total body water) were not observed between TT1 and TT2 in HIIT₃₅. Nonetheless, if an undetected increase in plasma volume occurred in the current study, the claim it could have increased performance in trained cyclists is questionable⁷³. Sweat loss between time-trials did not improve as a result of hot HIIT (indeed, the effect size for sweat loss was actually higher in HIIT₁₃ than HIIT₃₅). Additionally,

decreases in resting HR and T_C are established signs of HA⁴⁴, yet these measures did not decrease significantly in HIIT₃₅. Indeed, the effects sizes for resting HR and T_C changes were smaller than the range presented in a recent meta-analysis of HA adaptations⁴⁴. The only thermoregulatory measures that significantly improved in temperate testing after HIIT in 35°C was T_B (Figure 5.6) and TS during the 50% PPO portion of the warm-up (without a fan). Currently, more research is needed investigating mechanisms for HIIT performance increases and their relationship with T_A and time at or near VO_{2max} .

Previous research that has used a matched group design and demonstrating improvements in temperate and cool T_A endurance performance after training in the heat, have typically shown higher cardiovascular strain during training in the experimental group, compared with the control^{38,55,158}. Conversely, studies where cardiovascular strain was similar between hot and cool training conditions observed no difference in performance outcomes in temperate T_A ^{35,159}. In Chapter 4 of this thesis, no differences in average HR were observed between 13°C and 35°C HIIT. Subsequently, improvements in 20 km TT performance in the current study were not different between intervention groups. These findings provide additional evidence that greater performance gains observed in temperate conditions following training in the heat (compared with cool T_A) are not necessarily an outcome of increased thermal stress, but potentially the product of corresponding increases in cardiovascular load¹³⁵. However, this is not to imply hot and cool HIIT increased endurance performance with the same physiological adaptations. While HR may be similar (Chapter 4) or higher^{26,174} in hot versus cool conditions during high-intensity exercise, the underlying cardiophysiology (e.g. stroke volume) between the two conditions is likely to be different²⁶ and potentially stimulates different cardiovascular adaptations. However, more research is needed examining specific physiological adaptations to hot and cool HIIT.

It is plausible from the results of the present study that thermoregulatory adaptations after cool HIIT impaired endurance performance. Indeed, it has been suggested that submaximal aerobic performance in the heat is impaired by high T_{sk} ¹²¹ and low $T_c:T_{sk}$ gradient¹⁷⁹. Additionally, it has been demonstrated that increasing T_{sk} during submaximal steady-state exercise subsequently increases HR and T_c , and reduces stroke volume^{122,123}. Supporting this, in the current study significant increases in T_{sk} (Figure 5.6) and decreased $T_c:T_{sk}$ gradient occurred during the 50% portion of the warm-up (22°C with no fan) after 13°C HIIT that were not observed after 35°C HIIT. Concurrent with these thermoregulatory changes, a significant increase in VO_2 and a decrease in GME approaching significance was noted during the warm-up for HIIT₁₃ (Figure 5.3). However, while increased T_{sk} implies an influence on cardiovascular responses, it is also important to consider the potential effects cool HIIT could have on skeletomuscular adaptations, which could subsequently affect VO_2 and GME¹⁸⁵. Increased T_{sk} , T_c , and HR (i.e. cardiovascular strain) are associated with decreases in performance during self-paced steady-state exercise^{7,52}. In the current study T_c , HR, and RPE (i.e. cardiovascular, thermal, and subjective stresses) increased during the 20 km after 13°C HIIT, but not after 35°C HIIT (Figure 5.4 & Figure 5.5). Whilst no difference in performance improvements were noted between intervention conditions, the effect size and percentage for increased TT power output for HIIT₁₃ was smaller than what was observed for HIIT₃₅ (Figure 5.3). The above findings offer a caveat when considering cool T_A (i.e. T_A in the optimal performance range) for “control group” conditions when investigating the effects of heat exposure on exercise. However, more research investigating the effects a range of T_A have on thermoregulation and performance after repeated exposures during HIIT is needed before concluding cool HIIT is detrimental to performance.

5.5.1 Conclusions

Contrary to our initial hypothesis, the data herein indicates that 20 km TT performance at 22°C is increased after 4 weeks of HIIT in both 13°C and 35°C, with no differences between groups. This outcome is contrary to current reasoning, considering less time was spent at or near $\text{VO}_{2\text{max}}$ during HIIT in the heat. Additionally, little evidence was found for thermoregulatory adaptations in temperate T_A after hot HIIT that could potentially provide a mechanism for the similar performance gains between groups. These findings highlight the possibility that cardiovascular strain during HIIT, which was similar between intervention groups, is more important to performance outcomes than the time spent at or near $\text{VO}_{2\text{max}}$. Thermoregulatory changes in HIIT₁₃ were more apparent than in HIIT₃₅ and associated with a near significant decrease in gross mechanical efficiency for HIIT₁₃. More research into the potential benefits of heat stress on maximal self-paced HIIT is required.

6 CHAPTER SIX:

GENERAL DISCUSSION

Thesis Chronicle: The primary research purpose of this PhD project underwent noteworthy changes throughout its progression. The research aim of the initial proposal was to investigate world-class cyclists travelling from the warm/hot Southern Hemisphere to the cool/cold Northern Hemisphere (i.e. Europe) to compete in the early events of the professional road cycling schedule. This aim was prompted by anecdotal evidence from these riders and professional cycling organizations suggesting athletes travelling from hot to cold environments may struggle to cope with the rapid change in environmental conditions, possibly compromising training volume, intensity, and competition performance.

In order to examine the influence of dwelling and exercising in contrasting environments on athletes we proposed to assess a number of variables including: mood, illness, behavioral, biological markers for heat acclimatization, training load, and clothing microclimates. In this proposal it was hypothesized that a training intervention utilizing heat acclimation and high-intensity intervals at different environmental temperatures (either cool or hot), could potentially improve elite-level Southern Hemisphere athlete performance in a colder climate. Piloting for these studies included: i) the collection of data from elite athletes completing a questionnaire via Qualtrics; ii) temperature data collection utilizing iButtons during outdoor rides; iii) examination of a heat acclimation protocol in a well-trained cyclist with a portable sauna (Appendix G); and iv) the assessment of HIIT protocols to develop the testing and training sessions used within the present thesis. During this piloting it was apparent that greater detail on the relationships between environmental temperature and high-intensity interval training was required prior to confidently utilizing the proposed intervention with an elite athlete population. Indeed, research into how

environmental temperature affects HIIT performed by endurance trained athletes is lacking in the literature. Additionally, focusing on this area of research would impact a greater portion of the endurance athlete population than the small targeted cohort from the initial proposal. Henceforth, the proposed research within the PhD was refocused, prior to the commencement of the research, to its current aim of investigating the effects of environmental temperature on high-intensity interval training.

This change in research focus resulted in a number of novel findings, as well as a broadening of the PhD experience. Indeed, to our knowledge an investigation of high-intensity interval performance in a range of environmental temperature had not yet been conducted. Nor had there been research into endurance athlete performance outcomes after maximum effort high-intensity interval training in hot and cool environmental temperatures. Also, to our knowledge, the investigation of physiological responses during high-intensity intervals after repeated bouts (e.g. physiological changes first versus the last interval session of an intervention) found in this thesis is especially unique in HIIT research.

6.1 Thesis Summary

The primary aim of this thesis was to increase the understanding of how environmental temperature (T_A) affects high-intensity interval training in endurance athletes. Study 1 examined of the acute effects of T_A (5°C, 13°C, 22°C, & 35°C) on performance and physiological responses during a maximal high-intensity interval training session in well-trained cyclists. Studies following this examined the effects of repeated exposures to cool (13°C) and hot (35°C) environmental conditions on cardiorespiratory and thermoregulatory responses during a maximal high-intensity interval session (Study 2) and subsequent time-trial performance in a temperate environment (22°C; Study 3) in trained cyclists. The main findings of this thesis were: i) well-trained cyclists performing maximal high-intensity aerobic intervals can achieve near-optimal power output over a broader range of T_A (5°C, 13°C, & 22°C) than previous literature would indicate (Study 1, Chapter 3); ii) time spent at or near maximal oxygen consumption (VO_{2max}) was significantly lower during HIIT in 35°C compared with HIIT in 13°C (Study 2, Chapter 4); iii) after repeated bouts thermoregulatory responses during high-intensity intervals changed depending on T_A , yet changes in cardiorespiratory responses were negligible in both conditions (Study 2); and iv) temperate 20 km time-trial power output improved after 4-week (8 session) HIIT interventions in both 13°C and 35°C, with no difference in power output between conditions (Study 3, Chapter 5).

6.1.1 *Acute Effects of T_A on High-Intensity Intervals*

Endurance exercise performance is reduced in hot^{1,2} and cold^{2,3} environmental conditions. As a result, there is an inverse U-shaped relationship between endurance performance and T_A , implying that there are optimal conditions in which to perform an endurance exercise task^{2,6}. According to

previous literature exploring this relationship on steady-state exercise (i.e. time-to-exhaustion² and self-paced time-trials^{7,52}) this range is 10-17°C^{2,5-7}. However, findings from Study 1 indicated well-trained cyclists performing maximal high-intensity aerobic intervals can achieve near optimal power output over a broader range of T_A . Whereby, power output for intervals performed in T_A as low as 5°C were not affected, and decreases for power output in the heat (35°C) were not observed until the latter intervals of the 40-minute session. Supporting these findings from Study 1, Study 2 found no difference in the main effect for power output between high-intensity intervals performed at 13°C and 35°C.

Maintenance of interval performance during the coldest condition of 5°C implies that 5°C (Study 1) indicates T_A was not cold enough to alter homeostatic control to a point performance was compromised (e.g. reduced aerobic power). Indeed, core temperature (T_C) in the 5°C intervals was lower than the T_C in the 13°C and 22°C conditions (Figure 3.3), yet no difference in power output across these conditions was observed (Figure 3.1). It is plausible the standardized thermoneutral (~22°C) warm-up (10 minutes at 50% peak power output; PPO) prior to exercise¹²⁵, high metabolic heat caused by both the high intensity of the interval session¹²⁶, and (or) the high power output (and therefore heat production) of the well-trained participants¹⁸ assisted in maintaining a body temperature that would not compromise performance. As such, it is plausible that either a minimal temperature (T_C or muscle) to perform intense exercise had been attained^{53,125}, or possibly the duration of the attenuated T_C was not long enough to negatively affect performance².

In Study 1, power output in 35°C was lower during the last interval (5th) compared to all other T_A (Figure 3.1), yet T_C in 35°C during that interval was only higher than 5°C (Figure 3.3). A potential explanation for this inconsistency between T_C and power output is that performance in the heat during high-intensity intervals is also affected by increased T_{Sk} and (or) decreased $T_C:T_{Sk}$ gradient

(Figure 3.3)^{121,179}. Indeed, it has been argued that submaximal aerobic performance in the heat is impaired by high T_{sk} ¹²¹ and low $T_c:T_{sk}$ gradient¹⁷⁹. The intermittent nature of high-intensity intervals (4-minute work intervals followed by 5-minutes of rest), and (or) the large changes in windspeed during the protocol for Study 1, could have disrupted the association of T_{sk} and T_c measures typically observed in steady-state exercise^{2,52}. This could have subsequently enhanced the effects high T_{sk} in the heat had on performance.

Study 1 and Study 2 observed effects of T_A on acute cardiorespiratory responses during high-intensity interval sessions that could potentially affect HIIT performance outcomes. In particular, the time spent at or near VO_{2max} is suggested as an important measure of stimulus during HIIT⁸⁴. In Study 2, this cardiorespiratory measure was found to be shorter in 35°C, compared with 13°C (Figure 4.2). These findings are similar to other studies that observed decreases in VO_2 during exercise in hot conditions^{2,26,52}. Based on the findings from Study 2, and previous literature^{24,84}, it could be hypothesized that cool HIIT would provide greater improvements in performance than hot HIIT. However, this hypothesis was not supported by the findings of this thesis (Study 3; discussed in Section 6.1.3).

Study 2 demonstrated HR during hot intervals is similar to, or higher than, HR observed in cooler intervals, whilst time at or near VO_{2max} is lower in hot intervals (Figure 4.2). In Study 1, heart rate (HR) during maximum effort high-intensity intervals was observed to be higher in 35°C during the last two intervals compared to 5°C and 13°C (Figure 3.2). In Study 2, the main effect for average HR during HIIT in 13°C and 35°C was not different (Figure 4.1). These cardiorespiratory results align with previous studies investigating high-intensity exercise^{26,27}, and prolonged self-paced steady-state exercise^{7,52} in hot versus thermoneutral conditions. It is plausible that the time spent at high cardiac loads (near maximal cardiac output, stroke volume and HR) is as important

as time spent at or near $\text{VO}_{2\text{max}}$, and can be elevated with exercise in the heat. However, research on the effects heat has on high-intensity interval cardiophysiology (e.g. cardiac output and stroke volume) is currently limited.

Hyperthermia-induced hyperventilation (i.e. human panting) is a thermoregulatory response demonstrated to occur in humans while performing steady-state exercise in the heat. To our knowledge Study 2 was the first study to find evidence for hyperthermia-induced hyperventilation during high-intensity intervals. Whereby, higher ventilatory equivalents for oxygen (VE/VO_2) and carbon dioxide (VE/VCO_2) were observed in 35°C , compared with 13°C (Figure 4.3). This increased ventilation, when exercising in the heat, results in increased gas exchange, and consequently a reduction in arterial CO_2 pressure and increase in blood pH (i.e. blood alkalosis)^{114,115}. It has been suggested that increased T_{C} induces hyperthermia-induced hyperventilation during exercise and rest, whilst T_{Sk} induces this response only while at rest¹¹⁵. However, in Study 2 T_{C} was not different between 13°C and 35°C HIIT, while T_{Sk} and $T_{\text{C}}:T_{\text{Sk}}$ gradient were higher in 35°C . This potentially indicates, similar to interval performance in the heat, ventilation is also affected by T_{Sk} . Again, this might be due to the intermittent nature of high-intensity intervals and (or) the concurrent changes in convective heat loss. However, changes in acid base balance during HIIT in the heat are currently unclear.

6.1.2 Effects of Repeated T_{A} Exposure on High-Intensity Intervals

Heat exposure and HIIT result in rapid adaptations, and as a result, physiological responses to high-intensity intervals are likely to be transient and change throughout a typical HIIT intervention. Therefore, the purpose of Study 2 was to investigate how bouts (8 sessions total) of HIIT influence thermoregulatory and cardiorespiratory responses to a particular interval session in hot (35°C) and cool (13°C) conditions.

Results from Study 2 indicated that hot HIIT is potentially a viable method for inducing some degree of HA in endurance athletes (Figure 4.4). The effect sizes for T_C , TS, and sweat loss, were within the ranges presented in a recent HA meta-analysis⁴⁴, whilst the effects for decreased HR and T_{Sk} were greater than the presented ranges. These findings were contradictory to our initial hypotheses, where it was thought that T_A exposures (i.e. thermal impulses) during a HIIT intervention would be too infrequent to stimulate changes in thermoregulatory responses⁴³. Nevertheless, these results have positive implications for the HA of endurance athletes (discussed in Section 6.2).

Study 2 provided valuable information for coaches and sport scientists regarding the stress (physiological and mechanical) and subjective responses during maximal effort high-intensity intervals after repeated bouts. It was demonstrated that changes in cardiorespiratory responses (Figure 4.1, Figure 4.2, Figure 4.3), maximal self-paced power output, and rate of perceived exertion (Figure 4.1), during intervals were negligible for both (35°C) and cool (13°C) conditions after 4 weeks of HIIT. These results for cardiorespiratory responses and power output are interesting as it was hypothesized these measures would change as the participants became fitter over the course of the HIIT intervention. Indeed, results from Study 3 demonstrated improvements in performance occurred after HIIT in both T_A conditions indicating fitness had increased over the course of the training intervention. While the above findings for Study 2 and Study 3 might appear contradictory, the cardiorespiratory and power output findings for Study 2 are nonetheless potentially valuable when prescribing HIIT to an endurance athlete population (Section 6.2).

6.1.3 The Effects of Hot and Cool HIIT on Endurance Performance and Physiology

As previously discussed (Section 6.1.1), time at or near VO_{2max} during HIIT is associated with beneficial performance and physiological outcomes^{24,84}. However, the findings from Study 3

challenge this suggested relationship. Indeed, Study 3 demonstrated 20 km time-trial performance improvements were not different between 35°C and 13°C HIIT conditions (Figure 5.3), whilst $T > 90\%$ $\text{VO}_{2\text{max}}$ during 35°C HIIT was shorter than 13°C HIIT (Figure 4.2). It is therefore feasible cardiovascular strain (i.e. HR; Figure 4.1) and (or) T_{C} during HIIT also provide ample stimulus for performance benefits, as these were similar between HIIT conditions.

Contrary to the similar improvements in 20 km time-trial performance observed between 35°C and 13°C HIIT, T_{A} does appear to influence physiological and subjective responses during temperate time-trials. Indeed, increases in HR, RPE (Figure 5.4), and T_{C} (Figure 5.5) during the 20 km time-trial were observed after 13°C HIIT, but not after 35°C HIIT. Additionally, a number of interactions between the two T_{A} conditions for thermoregulatory measures occurred during the 20 km time-trial (Figure 5.5) and submaximal warm-up (Figure 5.6) after the intervention. The effects these changes in physiological responses may have had on time-trial performance are unclear. However, it is worth noting the percent change for time-trial power output was lower after HIIT in 13°C ($3.3 \pm 3.4\%$) versus 35°C ($7.3 \pm 6.3\%$; Figure 5.3), and increases in power output were observed during different sections of the time-trial (Figure 5.4). Turnes et al.⁸⁴ observed similar performance and physiological outcomes after two HIIT interventions with different time at $\text{VO}_{2\text{max}}$ (T_{A} not reported). Where, there was an absence in difference for improved performance measures (i.e. critical power and mean maximal power) between interventions, yet $\text{VO}_{2\text{max}}$ and lactate threshold improvements (i.e. physiological differences) were greater after the HIIT with higher time at $\text{VO}_{2\text{max}}$ ⁸⁴. These results demonstrate HIIT interventions with different time at or near $\text{VO}_{2\text{max}}$ can induce similar performance improvements, yet simultaneously different physiological adaptations. However, the physiological measures taken post intervention in Study

3 and Turnes et al.⁸⁴ were different (i.e. Turnes et al.⁸⁴ did not measure thermoregulatory responses), and therefore the level of comparison that can be made between studies is limited.

6.2 Practical Applications

A number of findings of this thesis are novel and further increase our understanding of the effect T_A has on HIIT.

Within Study 1, thermal sensation was closest to neutral/comfortable in 13°C compared with other conditions (Figure 3.4). This is a potentially important note for the comfort of athletes who partake in HIIT indoors, as room temperature (~22°C) is notably higher than this. Indeed, owners/managers of ‘cycling gyms’ and spin studios may find that reducing the T_A of their venues improves participant thermal comfort.

In Study 2, evidence for HA was observed after performing HIIT in 35°C, demonstrating the potential for utilizing hot HIIT as a method for preparing endurance athletes for competitive events in the heat (Figure 4.4). While hot HIIT as an independent HA modality needs to be further explored, given thermoregulatory data from Study 1 (Figure 3.3) and Study 2 (Figure 4.4), the thermal impulse provided from a session should be sufficient enough to be incorporated with more established methods for HA. Additionally, Study 3 demonstrated performance improvements from hot HIIT were similar to cool HIIT (Figure 5.3 & Figure 5.4). This is especially useful knowledge when considering hot HIIT in conjunction with a HA strategy, but it is also a valuable assurance for athletes who may have to conduct HIIT in hot conditions due to unavoidable circumstances.

Study 2 demonstrated cardiorespiratory responses (i.e. stress) during high-intensity intervals did not change over the course of a 4-week HIIT intervention, when the intervals were performed at maximum effort (Figure 4.1, Figure 4.2, Figure 4.3). This implies cardiorespiratory stress (i.e.

stimulus) during such intervals does not change over this period of time and number of sessions. This is potentially important information for both researchers and coaches to consider when designing/prescribing HIIT interventions for athletes or research purposes.

6.3 Limitations

The findings of this thesis have important implications for mechanistic and practical applications; however, some limitations should be noted.

Many of the novel findings from this thesis relied on devices that recorded physiological and performance data from within the climate chamber when participants exercised (e.g. metabolic cart, temperature data logger, ergometer). The metabolic cart could be of particular concern as T_A for the chamber is a crucial metric for calculating respiratory measures. However, per the suggestion of the metabolic cart manufacturer, this device was recalibrated prior to testing in new chamber temperatures, and after sitting in the new temperature for at least 30 minutes. Additionally, this device was operated within the T_A range recommended by the manufacturer to ensure accuracy.

In the current thesis time spent at or near VO_{2max} was measured as time during the interval session spent above 90% VO_{2max} ($T > 90\% VO_{2max}$). However, $T > 90\% VO_{2max}$ was calculated differently between the two studies that measured it (Study 1 & Study 2), and this could be deemed a limiter. In Study 1 VO_{2max} was determined as the highest VO_{2max} observed across all 5 testing sessions (i.e. a graded exercise test and 4 intervals sessions at different T_A). This was deemed to be appropriate as it would increase the potential of eliciting a true VO_{2max} for the participants¹⁸⁶. Contrary to this method, Study 2 employed the method that it is traditionally utilized in the literature^{82,184}. Whereby, VO_{2max} was determined from the initial graded exercise test, and subsequently utilized

to calculate $T > 90\% \text{VO}_{2\text{max}}$ during the first (INT1) and last (INT8) interval sessions. The results for $T > 90\% \text{VO}_{2\text{max}}$ were different between Study 1 and Study 2. It could be asserted the difference in findings for this measure between the two studies was due to the different methods $T > 90\% \text{VO}_{2\text{max}}$ was calculated. However, a t-test demonstrates the interval $\text{VO}_{2\text{max}}$ (68.47 ± 5.75 mL/kg/min) and graded exercise test $\text{VO}_{2\text{max}}$ (66.25 ± 7.99 mL/kg/min) values from Study 1 were not significantly different ($p = .471$). Therefore, it is unlikely the difference in $T > 90\% \text{VO}_{2\text{max}}$ results between Study 1 and Study 2 are due to the different methods of calculation.

In the present thesis, participants were instructed to comply with a number of protocol requirements (e.g. maintain a uniform diet and performing maximal efforts during testing). Given these study circumstances there was an underlying risk participant noncompliance could potentially skew research outcomes. However, endurance athletes were selected for this thesis as a participant group, not only for their ecological validity, but also for their experience in performing and preparing for intense bouts of endurance exercise. In addition to instructions given to participants prior to the studies, participants were questioned prior to testing sessions about their readiness to perform (via a Recovery-Stress Questionnaire) and diet coming into the test.

It is advantageous to conduct exercise research in a laboratory space as the environment offers an advantage in terms of controlling experimental conditions, however this can reduce ecological validity, and subsequently affect application of the research in situ. In the current thesis, testing for intervals and 20 km time-trials was conducted in an environmental chamber (i.e. a laboratory environment), inviting a level of scepticism in regard to ecological validity. In order to improve this aspect a large fan providing a windspeed of ~ 28 km/hour was utilized during the 20 km time-trial and the work intervals of HIIT testing sessions. In order to mimic convection that would be experienced while riding outdoors.

In this thesis trained endurance athletes performed a HIIT intervention (Studies 2 & 3). In order to obtain and maintain the overall training volume required to be considered ‘trained’, participants conducted training prior to and during the intervention outside of the laboratory. Individuals were instructed to maintain a similar training volume throughout the intervention and match this volume to what they were training prior to the intervention. Additionally, participant training volume was recorded 6 weeks prior to and during the intervention and analysed to confirm there was no difference between groups. A potential limitation to this intervention study is training outside of the laboratory was not controlled to the level of being prescribed by researchers for the participants. However, the considerations herein for participant training outside of the study intervention are more robust than a considerable amount of past intervention studies.

During the course of data collection data was lost for various time points for multiple variables (e.g. H.R. & respiratory measures) in Studies 1, 2, & 3. Therefore, data and outcomes from these studies should be interpreted with caution and careful consideration.

6.4 Directions for Future Research

The results of this thesis increase the knowledge of the acute effects of T_A on high-intensity intervals, how repeated exposure to T_A affect the physiological responses during high-intensity intervals, and how hot and cool T_A affects performance related outcomes of a HIIT intervention. However, the findings herein generate new questions for future research.

Study 1 and Study 2 provided valuable insight into the acute effects T_A has on physiological responses during high-intensity intervals. However, a number of precise mechanisms involved in the cardiorespiratory and thermoregulatory responses we observed during intervals are still

unclear. Greater understanding of these mechanisms may provide additional insight into what influences the HIIT outcomes measured in Study 3.

This thesis primarily reported on physiological measures that occurred during the work intervals (versus the rest intervals). Exceptions to this were the measures of heart rate recovery, recorded one minute after each work interval, and time at or near $\text{VO}_{2\text{max}}$, which was analyzed for the entire interval session. Nevertheless, future research into the acute effects of T_A on high-intensity intervals should also take into consideration physiological data occurring during rests between intervals. This might be especially valuable when investigating the relation between thermoregulatory responses, hyperthermia-induced hyperventilation, and high-intensity intervals. Whereby, previous literature has proposed increased T_C induces hyperventilation in the heat while exercising and rest, whilst increased T_{sk} induces hyperventilation only when at rest¹¹⁵. Yet, high-intensity intervals often incorporate periods of both exercise and rest.

Study 2 found evidence for HA after 7 sessions of HIIT in 35°C, with sessions occurring ~2x per week (Figure 4.4). However, these results would be more meaningful in a practical sense had more traditional performance tests (e.g. $\text{VO}_{2\text{max}}$ test, time-trial, and lactate threshold test) been performed pre and post the intervention in hot conditions (Appendix H.2). Similarly, Study 2 found evidence for thermoregulatory changes after HIIT in 13°C (Figure 4.4). The inclusion of performance tests in cool/cold temperatures could help investigate potential improvements in exercise economy when exercising in the cold, while including performance tests in the heat could test whether thermoregulatory changes observed after cool HIIT (Figure 4.4) decrease performance in hot conditions.

Study 3 demonstrated HIIT in hot and cool conditions result in similar performance outcomes in a population of trained cyclists (Figure 5.3), providing a degree of confidence that HIIT can be conducted over a range of T_A and remain beneficial. The selection of 13°C and 35°C as intervention temperatures was heavily influenced by results from Study 1, physiologically based rationale, and methods from similar intervention studies (e.g. Lorenzo et al. 2010³⁸). However, it is very likely that cyclists do not perform HIIT in either of these T_A regularly in situ. Therefore, future research with this population should consider comparing hot and cool HIIT interventions to either T_A typically trained in by cyclists (see Section 6.5) or room temperature (~22°C).

This thesis utilized trained endurance athletes (i.e. cyclists and triathletes) as its research cohort, and the knowledge gained from this research is expected to benefit this population. However, the effects T_A ¹⁸⁷ have on HIIT may be beneficial to other populations and therefore should be explored. Indeed, HIIT are both known for their health benefits within sedentary, diseased, and general populations^{56,188}. Combining these interventions together may improve health outcomes, and (or) the time-to-benefit ratio for either intervention conducted alone. Additionally, hot HIIT might be a novel approach to HA military personnel maintaining high levels of aerobic fitness prior to deployment to hot environments.

6.5 Additional Considerations

This thesis investigated the effects of a range of T_A on performance, subjective, and physiological responses during acute exercise (5°C, 13°C, 22°C, 35°C) and after repeated exposure (13°C & 35°C). In science it is common to design experiments with an experimental and control group. However, when testing the effects of T_A on exercise, denoting an T_A as a “control” or “thermoneutral” may be inappropriate. While choosing a control T_A in the range of 10-17°C^{2,5-7} for acute studies investigating performance outcomes in hot or cold conditions is arguably

appropriate, this same argument made for interventions with repeated exposure to T_A during exercise becomes problematic. Study 2 demonstrated a number of interactions for thermoregulatory responses between conditions and the first and last interval sessions (Figure 4.4). Concurrent with these interactions were significant within subject changes in the 13°C condition, whilst an absence of within subject changes were observed in the 35°C condition (Figure 4.4). If 13°C was considered a “control” under these circumstances, it could be erroneously concluded that HIIT in 35°C causes these thermoregulatory responses to be maintained compared to *normal* conditions. However, anecdotal observations would have us believe that HIIT in 13°C in situ would be notably different than how it was performed in this thesis (i.e. athletes would wear more than just bibs and a jersey and warming up would occur in the same T_A that HIIT was performed in and for a longer duration). This also highlights the question of how often athletes would train in conditions as cool as 13°C when they could potentially train in warmer indoor conditions. Therefore, it may be best practice for applied studies investigating T_A and endurance exercise interventions to utilize a control condition determined by retrospectively analyzing the conditions athletes actually train in. Conversely, for basic science research investigating how T_A affects endurance exercise after repeated exposures, determining a “true” control T_A may be an impossible task, and best practice might involve denoting research groups with the actual T_A the intervention was performed in (as was done in this thesis). Nevertheless, the findings of this thesis demonstrate that T_A should be taken into consideration, recorded, and reported by researchers investigating training interventions, especially interventions involving HIIT.

6.6 Conclusions

In summary, this thesis examined how T_A acutely affects high-intensity intervals, how repeated exposure to hot and cool T_A manipulate the physiological responses to high-intensity intervals, and

how T_A affects performance outcomes of a high-intensity interval training intervention. This thesis concludes the following:

1. Power output during a high-intensity interval session performed in 35°C declines at the end of the session compared with high-intensity interval sessions performed in 5°C, 13°C, and 22°C conditions. These findings for power output do not appear to relate to T_C as previous research investigating the effects on T_A on steady-state exercise has indicated.
2. Time spent at or near VO_{2max} is lower during HIIT in 35°C versus HIIT in 13°C, however time spent at or near VO_{2max} does not appear to change in either temperature after 7 repeated bouts.
3. Thermoregulatory responses during high-intensity intervals are altered after 7 repeated bouts of HIIT in 13°C (i.e. increased skin temperature) and 35°C (i.e. reduced core temperature).
4. Ventilatory equivalents for oxygen consumption and expired carbon dioxide are higher for HIIT in 35°C compared with HIIT in 13°C, which provides evidence for hyperthermia-induced hyperventilation during high-intensity intervals in the heat.
5. Temperate 20 km time-trial power output improves after a 4-week (8 session) HIIT interventions in both 13°C and 35°C, with no difference in power output between conditions. However, cardiorespiratory and thermoregulatory responses during a 20 km time-trial and submaximal warm-up appear to depend on the T_A a HIIT intervention is performed in.

Overall, the findings of this thesis expand the scientific understanding of how T_A affects HIIT in an endurance trained population. Since HIIT has been demonstrated to play an important role in endurance athlete training, it is important that the interactions between HIIT and T_A are well

understood. These findings will hopefully help coaches and athletes make better informed decisions relating to HIIT prescription and acclimating endurance athletes to T_A .

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APPENDICES

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Appendix A Published Article (Study 1)

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Human Kinetics 
ORIGINAL INVESTIGATION

Effect of Environmental Temperature on High-Intensity Intervals in Well-Trained Cyclists

Jason R. Boynton, Fabian Danner, Paolo Menaspà, Jeremiah J. Peiffer, and Chris R. Abbiss

Purpose: To examine the effect of environmental temperature (T_A) on performance and physiological responses (eg, body temperature, cardiopulmonary measures) during a high-intensity aerobic interval session. It was hypothesized that power output would be highest in the 13°C condition and lower in the 5°C, 22°C, and 35°C conditions. **Methods:** Eleven well-trained cyclists randomly completed 4 interval sessions at 5°C, 13°C, 22°C, and 35°C (55% [13%] relative humidity), each involving five 4-min intervals interspersed with 5 min of recovery. During the intervals, power output, core temperature (T_C), skin temperature, $\dot{V}O_2$, and heart rate were recorded. **Results:** Mean session power output for 13°C (366 [32] W) was not higher than 5°C (363 [32] W; $P = 1.00$, effect size = 0.085), 22°C (364 [36] W; $P = 1.00$, effect size = 0.061), or 35°C (352 [31] W; $P = .129$, effect size = 0.441). The 5th interval of the 35°C condition had a lower power output compared with all other T_A . T_C was higher in 22°C compared with both 5°C and 13°C ($P = .001$). $\dot{V}O_2$ was not significantly different across T_A ($P = .187$). Heart rate was higher in the 4th and 5th intervals of 35°C compared with 5°C and 13°C. **Conclusions:** This study demonstrates that while mean power outputs for intervals are similar across T_A , hot T_A ($\geq 35^\circ\text{C}$) reduces interval power output later in a training session. Well-trained cyclists performing maximal high-intensity aerobic intervals can achieve near-optimal power output over a broader range of T_A than previous literature would indicate.

Keywords: endurance athletes, intermittent exercise, heart-rate recovery, sweat loss

Endurance exercise performance is reduced substantially in hot^{1,2} and cold^{2,3} environmental conditions. There is an inverse U-shaped relationship between endurance performance and environmental temperature (T_A), implying that there are optimal conditions in which to perform an endurance task.^{2,4} According to previous literature, this temperature is in the range of 10°C to 17°C,^{2,4-6} but is dependent on the mode of exercise, duration of exercise, and rate of convection.⁷ Yet, much of the research quantifying optimal T_A for exercise has involved relatively steady-state tasks at or around lactate threshold.^{2,4-6} Paradoxically, elite endurance athletes typically spend very little time at these intensities during training.⁸ This is especially the case for elite/professional road cyclists.⁹ Indeed, such athletes typically spend the large majority of training at low intensities (ie, below lactate threshold) coupled with short and intermittent bouts of high-intensity interval exercise or training (HIIT).¹⁰ The influence of T_A on performance and physiological response to such exercise is not well understood.

Despite HIIT being a relatively small part of training volume,⁸ it can induce rapid and large adaptations important to endurance performance.¹¹ Such high-intensity exercise results in an increased metabolic rate¹² and high metabolic heat production,¹³ possibly compromising performance. Indeed, Drust et al¹⁴ demonstrated that power output during repeated-sprint efforts performed following 40 minutes of intermittent exercise in the heat (40°C) was decreased when compared with the control condition (~20°C). In addition, due to variations in an athlete's velocity, convective heat loss will also notably change throughout an interval training

session. Therefore, it is plausible that the effects T_A has on high-intensity interval performance differ to what has been observed in prolonged steady-state exercise.

Previous literature examining endurance exercise under a range of T_A has not considered conditions that have an effect on cycling performance in a well-trained cyclist population. Galloway and Maughan² examined the time to exhaustion under a range of T_A , but of note, the mean maximal oxygen consumption ($\dot{V}O_{2\text{max}}$) for these subjects (~56 mL/kg/min) was below what the literature would consider "well-trained" endurance athletes.^{15,16} This is important because trained individuals can exercise at higher exercise intensities,¹⁵ eliciting higher metabolic heat production¹³ than untrained individuals. In addition, the aforementioned studies examining the effects of T_A on endurance performance have not included important elements known to affect cycling performance; such as a standardized warm-up¹⁷ and replication of realistic convective heat loss through high wind velocities.^{7,18}

From this synopsis, it can be recognized that a better understanding of the effects T_A has during high-intensity intervals is needed. Therefore, the overall purpose of this study was to determine the effects of T_A on physiological responses (eg, body temperature and cardiopulmonary measures) and performance (ie, power output) during intervals. To our knowledge, this was the first investigation seeking to explore the effect a range of T_A has on high-intensity intervals. It was hypothesized that power output and $\dot{V}O_2$ would decrease in the 5°C, 22°C, and 35°C conditions compared with the 13°C condition.

Methods

Participants

Eleven male cyclists (age 34.1 [9.8] y, height 181.3 [5.0] cm, mass 75.78 [8.01] kg, sum of 8 skinfolds 65.3 [19.1] mm, $\dot{V}O_{2\text{max}}$ 69.62

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Appendix B 23rd Annual ECSS Congress Poster (Study1)



EFFECT OF ENVIRONMENTAL TEMPERATURE ON HIGH INTENSITY INTERVALS IN WELL-TRAINED CYCLISTS

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Introduction

Performance of steady-state endurance exercise close to lactate threshold is reduced in substantially hot and cold environmental temperatures (T_A), demonstrating an inverse U-shaped relationship between T_A and endurance performance (Galloway and Maughan, 1997; Peiffer and Abbiss, 2011). Paradoxically, endurance athletes train a small duration at threshold intensity while maximal high-intensity aerobic intervals (interval training) are an important component of endurance training (Stöggl and Sperlich, 2015). This study examined the influence of a range of T_A on performance and physiological responses (e.g. body temperature and cardiopulmonary measures) during interval training. Similar to the findings of previous research (Galloway and Maughan, 1997; Peiffer and Abbiss, 2011), it was hypothesized that power output and oxygen consumption ($\dot{V}O_2$) would be highest in the 13°C condition and lower in the 5°C, 22°C, and 35°C conditions.

Methods

Eleven well-trained cyclists completed four interval sessions at 5°C, 13°C, 22°C, and 35°C (55% RH) in a randomized order. Interval sessions involved a standardized warm-up at a neutral T_A (22°C) and five self-paced 4-minute high intensity intervals interspersed with five minutes of recovery. Power output, $\dot{V}O_2$, core temperature (T_{re}), and heart rate (HR) were recorded during the sessions.

Results

Mean session power output for 13°C (366 ± 32 W) was not markedly higher than 5°C (365 ± 35 W, $P = 1.00$, $ES = .030$), 22°C (364 ± 36 W, $P = 1.00$, $ES = 0.061$), or 35°C (351 ± 31 W, $P = .129$, $ES = 0.441$). Power output was lower in the 5th interval of the 35°C condition compared with all other T_A , yet no significant interactions were observed between 5°C, 13°C, and 22°C conditions. $\dot{V}O_2$ was not significantly different across T_A ($P = .187$). T_{re} was higher in 22°C compared with both 5°C and 13°C ($P = .001$). HR in the 4th and 5th intervals were higher in 35°C compared with 5°C and 13°C.

Conclusions

This study demonstrates that whilst mean power outputs for intervals are similar across T_A , hot T_A ($\geq 35^\circ\text{C}$) had a negative effect on interval power output later in a training session (> 20 min). This study also shows power output for intervals in a T_A as low as 5°C is not affected when performed by well-trained cyclists. In conclusion, well-trained cyclists performing maximal high-intensity aerobic intervals after a standardized warm-up can achieve near optimal power output over a broader range of T_A than previous literature has indicated.

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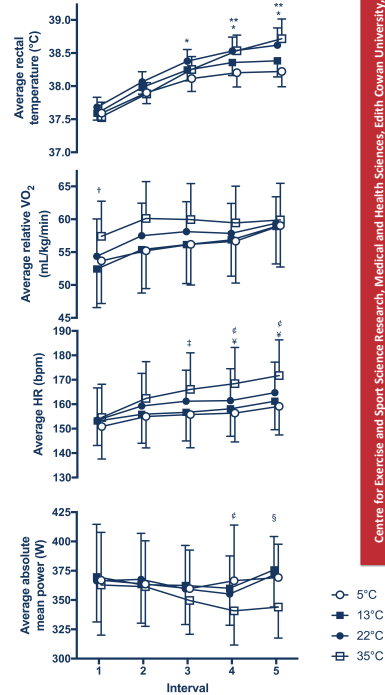


Figure significant differences ($p < .05$):

*22°C vs 5°C & 13°C; **5°C vs 13°C & 35°C; †13°C vs 22°C; ‡22°C vs 35°C; §5°C vs 22°C & 35°C; ¶13°C vs 35°C; §35°C vs 5°C, 13°C, & 22°C



EDITH COWAN

Appendix C Recruitment Flyer (Study 1)

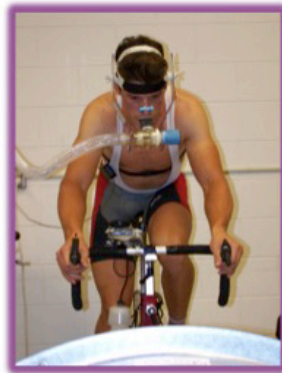


ATTENTION!
18- 40 year old trained cyclists
You are invited to participate in HIGH
PERFORMANCE research at Edith
Cowan University.

If you are a male cyclist who trains more than 8 hrs/wk you are welcomed to partake in an innovative performance research project. Your participation will greatly improve our knowledge of temperature's effects on endurance exercise.

SCHEDULE:

1x VO_{2max} testing session
4x coached high intensity interval training sessions
PLUS! Feedback on aerobic fitness!



Interested persons please contact:

Jason Boynton, MS, PhDc

Mobile: [REDACTED]

Email: j.boynton@ecu.edu.au

Appendix D Environmental Chamber





ATTENTION!

18-55 year old trained cyclists

You are invited to participate in HIGH PERFORMANCE research at Edith Cowan University

If you are a lean **male or female cyclist** 18-55 y/o who regularly trains more than 7 hrs/wk you are welcomed to partake in an innovative performance research project. Your participation will greatly improve our knowledge of temperature's effects on interval training.

SCHEDULE:

1x VO_{2max} session
3x indoor time trial testing sessions
8x high intensity interval training sessions (2/week)
PLUS! Feedback on aerobic fitness!



Interested persons please contact:
Jason Boynton, MS, PhDc
Mobile: [REDACTED]
Email: j.boynton@ecu.edu.au

Appendix F Best Pre Time-Trial versus Last Time Trial

Appendix Table F.1 Within subject changes for performance, cardiorespiratory, and subjective measures during a 20 km time-trial and submaximal warm-up (WU) for pre (TTB) and post (TT2) after 4-weeks (8 sessions) of high-intensity interval training in either 13°C (HIIT₁₃) or 35°C (HIIT₃₅).

Test Section	Group	Measure	TTB	TT2	Change (absolute)	Change (%)	Effect Size	P
20 km Time-trial	HIIT ₁₃	Power (W)	271 ± 50	276 ± 46	5 ± 9	2.0 ± 3.0	0.092	.131
	HIIT ₃₅	Power (W)	252 ± 48	263 ± 44	11 ± 11	4.6 ± 4.6	0.221	.016*
	HIIT ₁₃	Power (W/kg)	3.84 ± 0.52	3.93 ± 0.48	0.10 ± 0.10	2.7 ± 2.7	0.184	.018*
	HIIT ₃₅	Power (W/kg)	3.60 ± 0.64	3.77 ± 0.58	0.17 ± 0.18	5.2 ± 5.3	0.265	.016*
	HIIT ₁₃	HR (bpm)	167 ± 11	168 ± 8	1 ± 5	0.5 ± 3.2	0.072	.691
	HIIT ₃₅	HR (bpm)	165 ± 13	164 ± 11	0 ± 10	-0.1 ± 3.5	-0.032	.832
	HIIT ₁₃	Average RPE	16 ± 1	16 ± 1	0 ± 1	-	-	.118
	HIIT ₃₅	Average RPE	16 ± 1	16 ± 1	0 ± 1	-	-	.287
	HIIT ₁₃	Average TS	1 ± 1	1 ± 1	0 ± 1	-	0.250	.401
	HIIT ₃₅	Average TS	2 ± 1	2 ± 1	-1 ± 1	-	-0.051	.775
	HIIT ₁₃	Cadence (rpm)	95 ± 6	96 ± 6	1 ± 4	0.7 ± 4.4	0.095	.658
	HIIT ₃₅	Cadence (rpm)	98 ± 10	95 ± 11	-3 ± 3	-3.0 ± 3.3	-0.267	.014*
	HIIT ₁₃	H ₂ O intake (kg)	0.13 ± 0.10	0.25 ± 0.34	0.12 ± 0.32	-	0.454	.266
	HIIT ₃₅	H ₂ O intake (kg)	0.23 ± 0.12	0.30 ± 0.20	0.07 ± 0.23	75 ± 202	0.384	.387
WU 75% PPO	HIIT ₁₃	VO ₂ (mL/kg/min)	50.4 ± 7.7	49.6 ± 6.0	-0.8 ± 3.4	-1.0 ± 6.8	-0.112	.468
	HIIT ₃₅	VO ₂ (mL/kg/min)	48.6 ± 6.4	48.3 ± 6.9	-0.3 ± 2.2	-0.7 ± 4.5	-0.043	.680
	HIIT ₁₃	HR (bpm)	162 ± 9	161 ± 8	-1 ± 5	-0.5 ± 2.8	-0.118	.515
	HIIT ₃₅	HR (bpm)	159 ± 15	157 ± 16	-2 ± 7	-1.3 ± 3.9	-0.129	.383
	HIIT ₁₃	RPE	13 ± 1	13 ± 1	0 ± 1	-	-0.242	.468
	HIIT ₃₅	RPE	14 ± 1	14 ± 1	0 ± 1	-	0.000	1.00
	HIIT ₁₃	TS	3 ± 1	2 ± 1	-1 ± 1	-	-0.851	.015*
	HIIT ₃₅	TS	2 ± 1	2 ± 1	0 ± 1	-	-0.271	.443
	HIIT ₁₃	Cadence (rpm)	91 ± 11	92 ± 5	1 ± 11	0.9 ± 12.4	0.112	.786
	HIIT ₃₅	Cadence (rpm)	87 ± 10	91 ± 12	4 ± 5	4.4 ± 5.6	0.487	.040*
WU 50% PPO	HIIT ₁₃	VO ₂ (mL/kg/min)	35.3 ± 4.3	35.9 ± 3.7	0.6 ± 2.3	2.1 ± 6.7	0.142	.428
	HIIT ₃₅	VO ₂ (mL/kg/min)	33.9 ± 3.8	34.3 ± 4.1	0.4 ± 1.3	1.2 ± 3.8	0.098	.340
	HIIT ₁₃	HR (bpm)	131 ± 12	132 ± 11	1 ± 4	0.5 ± 3.0	0.041	.725
	HIIT ₃₅	HR (bpm)	128 ± 14	127 ± 13	-1 ± 6	-0.6 ± 4.1	-0.066	.622
	HIIT ₁₃	RPE	11 ± 1	10 ± 1	0 ± 1	-	-	.394
	HIIT ₃₅	RPE	11 ± 0	11 ± 1	0 ± 1	-	-	.758
	HIIT ₁₃	TS	1 ± 1	1 ± 0	0 ± 1	-	-0.192	.591
	HIIT ₃₅	TS	1 ± 1	1 ± 1	-1 ± 1	-	-0.757	.010*
	HIIT ₁₃	Cadence (rpm)	88 ± 10	91 ± 4	2 ± 9	2.3 ± 11.0	0.281	.492
	HIIT ₃₅	Cadence (rpm)	82 ± 10	88 ± 8	6 ± 6	6.7 ± 6.8	0.625	.029*

TTB values were taken from the familiarization or pre-time trial (i.e. TT1) with the highest power output. The 10-minute warm-up consisted of two five-minute segments at 50% and 75% peak power output (PPO). Testing conditions were set to 22°C and 40% relative humidity. During the time-trial a fan providing a simulated wind at a speed of ~28 km/hour was provided. Average rate of perceived exertion (RPE) and thermal sensation (TS) were the average values taken every 5 km during the time-trial. n= 10 per group except for heart rate (HR) for HIIT₁₃ (n= 9) during the time-trial, and HR (n= 9) & cadence (n= 8) for HIIT₃₅ during the warm-up. VO₂, oxygen consumption. Values are mean ± SD, Hedge's g was calculated for the effect size. *Significant within subject changes between TTB and TT2(P< .05).

Appendix G Sauna Pilot Study

A SHORT-TERM SAUNA PROTOCOL FOR HEAT ACCLIMATING WELL-TRAINED CYCLISTS: A CASE AND PILOT STUDY

A G.1 Introduction

Repeated heat exposure can affect endurance performance in hot conditions with as few as 4-5 bouts¹. Post-exercise sauna bathing has been suggested as a method to heat acclimate (HA) endurance athletes²⁻⁴. Sauna sessions have been shown to stimulate an increase in heat tolerance in untrained individuals in as few as 3 days⁴. However, to date, only two studies have investigated sauna HA on trained athletes utilizing a sauna^{5,6}. Of these studies, both incorporated 10 or more days of sauna heat exposure, and neither examined the effects a sauna intervention had on exercising in the heat^{5,6}. It is important to consider that short-term passive HA in a sauna may not demonstrate the same performance benefits as short-term active HA protocols⁷. Whereby, it has been suggested that HA for athletes should occur under the conditions in which they compete (i.e. while exercising)⁷. Yet, being able to utilize a portable sauna for HA serves multiple advantages for researchers and athletes alike (e.g. inexpensive, easy to store and transport). Currently, research examining short-term HA in well-trained endurance athletes utilizing a portable sauna is lacking. However, prior to investigating this research topic in our laboratory, a number of devices (e.g. portable sauna), and methodological aspects (e.g. the duration spent in the sauna) needed to be evaluated.

Well-trained endurance athletes are categorized by high levels of performance (e.g. power output), physiological ability (e.g. maximal oxygen consumption), and training loads⁸. High training loads and the subsequent high amounts of stress on an endurance athlete are important to manage in order to optimize performance outcomes⁹. As a research population, endurance athletes are likely to have concerns about intervention efficacy, the potential costs of lost fitness due to overtraining, and subsequent performance outcomes. However, the present researcher did not have previous experience with HA in endurance athletes using a portable sauna, and subsequently questioned

their ability to address concerns about the protocol that would be specific to that population (e.g. acute discomfort; i.e. not general safety concerns). Additionally, given the lack of researcher experience with sauna HA, unforeseeable consequences and issues with the HA protocol and testing sessions were a concern. Therefore, it was determined a firsthand account by a member of the research team for the proposed research HA protocol would be valuable for addressing potential concerns of future participants and improve future study methodology.

From this brief synopsis it should be clear that a better understanding of the methodological approach for investigating short-term sauna HA interventions in a well-trained population is needed. Therefore, the purpose of this pilot study was to practically evaluate the devices and methods of the proposed HA research protocol, while also gaining first-hand researcher experience of short-term sauna HA.

A G.2 Methods

A G.2.1 Participant & Study Overview

A detailed methodology for this pilot study is provided herein, in large part, to assist future research into short-term sauna HA. One well-trained⁸ male cyclist and researcher from Edith Cowan University (Appendix Table G.1) completed two submaximal tests in an environmental chamber at 35°C before (ST1) and after (ST2) a sauna HA intervention. The sauna intervention consisted of 5 sessions (over 6 days) in a portable infrared sauna (Appendix A H.1). A familiarization HA session was conducted 7 days prior to ST1. During the intervention the researcher/participant attempted to maintain a training load typical to previous 6 weeks of training.

Nude bodyweight was measured before and after each submaximal test and sauna session on a calibrated scale (GWB Mettler ID1 Multi-Range, Toledo, Columbus, OH, USA). Nude

bodyweight was recorded 12 and 7 days before ST1 and 8 days after ST2. Sweat loss was calculated based on change in nude body mass during each session, accounting for water consumed. The participant was allowed to consume water ad libitum during all sessions. Water consumed during testing and sauna sessions was determined by measuring pre and post water bottle weights. Sum of 8 skinfolds (Harpender skinfold calliper, Baty International, West Sussex, U.K.) was assessed¹⁰.

A Table G.1 Baseline physical, performance, and training characteristics of the well-trained cyclist participant

Age (y)	40
Body Mass (kg)	69.12
Height (cm)	179
Sum of 8 (mm)	77.9
PPO (W)	422
PPO (W/kg)	6.1
VO _{2max} (L/min)	83.0
VO _{2max} (mL/kg/min)	5.7
Years of structured training (y)	12
Sum of 8 = total 8 sites of skinfolds, VO _{2max} = maximal oxygen consumption.	

A G.2.2 Submaximal Tests

60-minute submaximal tests were completed on an electromagnetically-braked cycling ergometer (Velotron, Racermate, Redmond, WA, U.S.A.) at the participant's 50% peak power output (PPO) in 35°C at 40% relative humidity. Prior to the test the participant rested for 3-minutes. Rectal temperature (T_C) and skin temperature (T_{sk}; Squirrel SQ2020 Data Logger, Grant Instruments,

Shepreth Cambridgeshire, UK), were continuously measured (1 Hz). T_C was measured using a disposable rectal thermometer (Monatherm Thermistor 400 Series, Mallinckrodt Medical, St. Luis, MO, USA) inserted ~10 cm past the anal sphincter. T_{Sk} was measured via 4 thermistors (YTS Temperature, 400 Series, Dayton, Ohio) placed on the chest (T_{chest}), bicep (T_{arm}), thigh (T_{thigh}), and calf (T_{leg}) and mean T_{Sk} and was calculated¹¹ using the following formula:

$$T_{Sk} = 0.3 (T_{chest} + T_{arm}) + 0.2 (T_{thigh} + T_{leg})$$

T_C and T_{Sk} measurements were used to calculate $T_C:T_{Sk}$ gradient. Resting measures for participant temperature were calculated as an average for the values 2 minutes prior to exercise. Participant temperature values were averaged over 5-minute intervals during exercise. Maximum T_C was determined as the highest 30-second average during the session. Sessions were performed at the same time of day to avoid circadian body temperature variations¹². Fluctuations in temperature data greater than 0.10°C/second are physiologically improbable and likely the result of faulty temperature probes and thus were removed from analysis. Heart rate (HR; Polar Precision Performance SW5.20, Polar Electro, Kempele, Finland) was recorded continuously (0.2 Hz and averaged over 5-second intervals). Resting HR was determined as the lowest 30-second average during the 3-minute rest. HR data during exercise was averaged over 5-minute intervals. Whole body perceived exertion¹³ (RPE) and thermal sensation (TS) were recorded using visual numerical scales every 5 minutes during exercise, in addition to TS being recorded during the rest prior to exercise. Prior to exercise finger prick blood for hematocrits was collected via capillary while the participant sat (100 Capilette, 955 05 32 02, Selzer Labor Technik, Mannheim-Heidelberg-Karlsruhe, Germany), spun at 3500 rpm for 5 minutes (Centrifuge MPW-212, MPW Medical Instruments, South Australia). Before submaximal testing the participant refrained from strenuous

exercise (36 hours prior), eating large meals in the (3 hours prior), and consuming non-habitual caffeine in the (24 hours prior).

A G.2.3 Sauna Sessions

Sauna sessions were conducted at $\sim 55^{\circ}\text{C}$ for a duration of ~ 35 minutes. The participant performed a warm-up at room temperature ($\sim 24^{\circ}\text{C}$) for 10 minutes at 50% PPO followed by 5 minutes at 75% PPO on a cycling ergometer (WattBike Pro, WattBike, West Bridgford, Nottingham, UK) in order to increase T_{C} prior to entering the portable sauna (Appendix A H.1). Prior to warm-up the participant rested for 3-minutes. T_{C} and HR were recorded continuously (described above). Resting values (for T_{C} & HR), average HR at 50% PPO, and maximum T_{C} were calculated (described above).

A G.2.4 Training Load

The workout duration data were acquired retrospectively via the participant's personal online training logs (TrainingPeaks, Boulder, United States) across 42 days prior to, during the pilot, and 7 days post. Additionally, training load, in terms of Training Stress ScoreTM (TSS), was analysed with TrainingPeaks' Performance Management ChartTM (PMC), to calculate Chronic Training LoadTM (a measure of fitness; CTL) and Training Stress BalanceTM (TSB). Training Stress BalanceTM, is considered a measure of athlete 'form' (or 'freshness') and is calculated with Performance Management ChartTM's measures of fatigue and fitness.

A G.2.5 Statistics

Results are presented as $n = 1$. Differences in reported values should not be interpreted as significant.

A G.3 Results

Results for measurements taken during this pilot study are presented for the purposes of evaluating methodology and hypothesis building. It is important that the findings reported here are interpreted as anecdotal. Differences in values should not be perceived as statistically significant. Additionally, figures and tables are provided as examples for how future data collected from this research protocol could be presented (i.e. data from multiple participants).

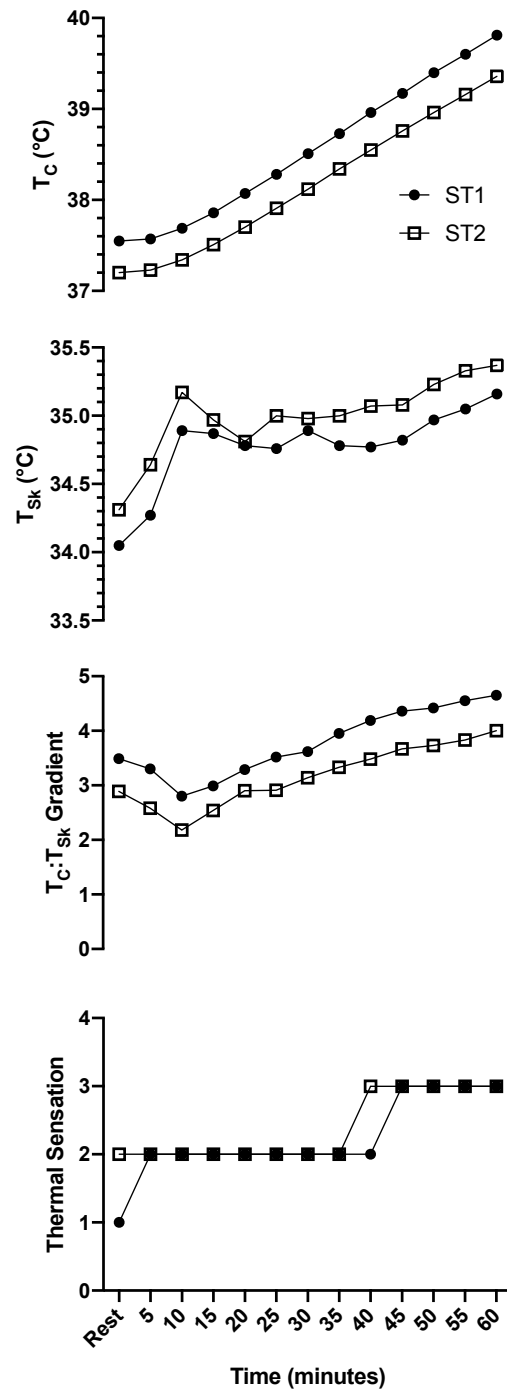
Temperature for the portable sauna was $56.7 \pm 3.7^{\circ}\text{C}$ and ranged from 51.8°C to 59.9°C ($n=4$ sessions). The participant spent an average of 34 ± 7 minutes in the sauna during each session (range 25 to 40 minutes). Maximum T_{C} reached during sauna sessions was $39.4 \pm 0.7^{\circ}\text{C}$ (range 38.4°C to 40.0°C).

A G.3.1 Submaximal Test

Chamber temperatures for the ST1 and ST2 were 34.8°C and 35.0°C , respectively. Average T_{C} during exercise decreased by 1.0% between ST1 (38.6°C) and ST2 (38.3°C ; Appendix Figure G.1). Similarly, the maximum T_{C} observed in ST1 (39.9°C) decreased by 1.1% in ST2. Conversely, average T_{Sk} during ST2 increased by 0.8% compared to ST1 (Appendix Figure G.1), and subsequently $T_{\text{C}}:T_{\text{Sk}}$ gradient decreased by 17.4% (Appendix Figure G.1). Average and maximum HR both decreased between ST1 (170 bpm & 186 bpm, respectively) and ST2 (161 bpm & 174 bpm; 5.5%, 6.5%; Appendix Figure G.2). Resting T_{C} and HR values decreased by 0.9% and 15.2%, respectively. Hematocrit values for ST1 and ST2 were 46.0% and 44.5%, respectively. Bodyweight and sweat loss values are presented in Appendix Table G.2. TS and RPE during submaximal sessions are shown in Appendix Figure G.1 and Appendix Figure G.2, respectively.

A Table G.2 Subject's change in bodyweight (BW), fluid intake, sweat lost, and percentage BW during a 60-minute test at 50% PPO pre and post 5 sauna heat acclimation sessions.

	ST1	ST2	% Change
Change in BW (kg)	1.93	0.98	-49.2
Fluid intake (kg)	0.30	0.44	46.7
Sweat lost (kg)	2.23	1.42	-35.8
BW lost (%)	3.20	2.06	-36.5
PPO = peak power output, ST1 = pre 60-minute submaximal test, ST2 = post 60-minute submaximal test			



A Figure G.1 Thermoregulatory measures for a well-trained cyclist ($n=1$) during a submaximal test (60 minutes at 50% peak power output) at 35°C pre (ST1) and post (ST2) a 5 sauna session heat acclimation protocol. T_c = core temperature, T_{sk} = skin temperature, TS = thermal sensation.

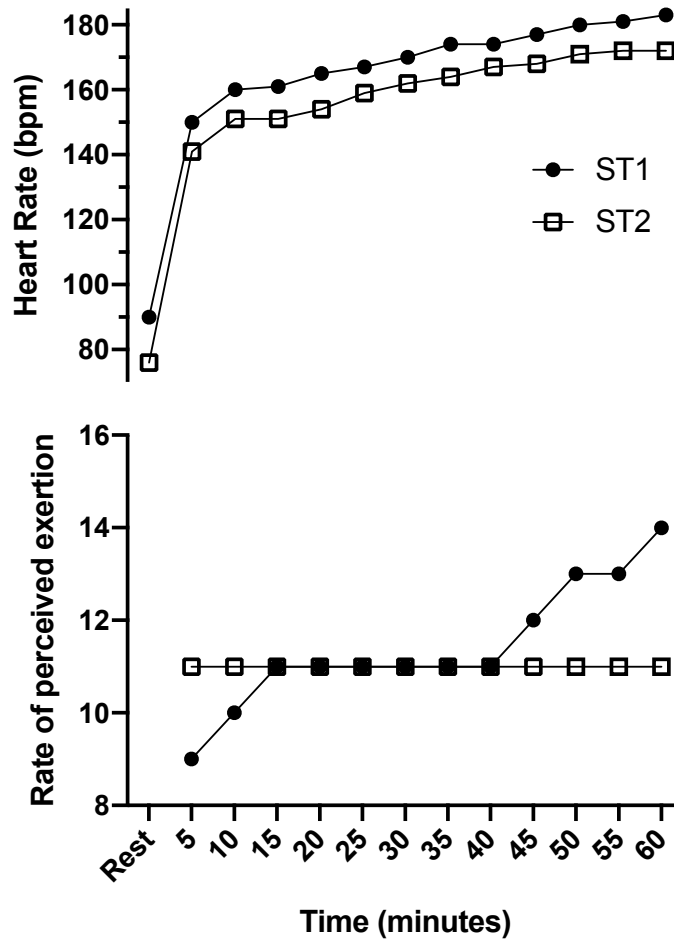
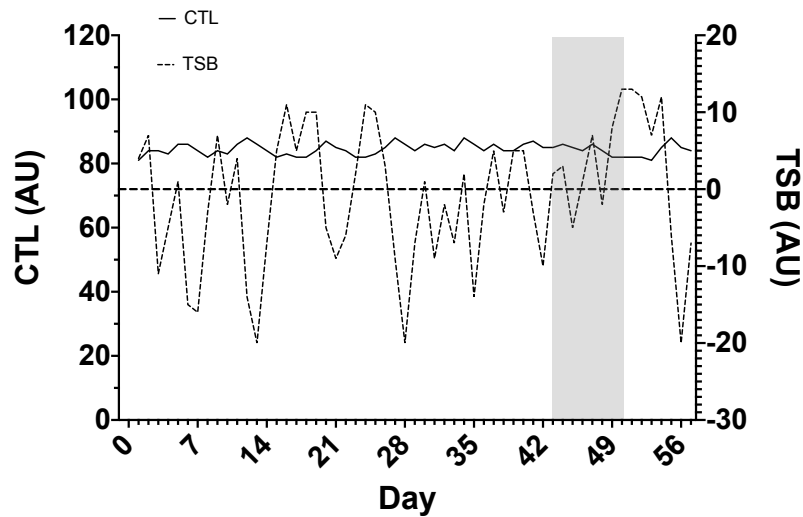


Figure G.1 Heart rate and rate of perceived exertion measures for a well-trained cyclist (n= 1) during a submaximal test (60 minutes at 50% peak power output) at 35°C pre (ST1) and post (ST2) a 5 sauna session heat acclimation protocol.

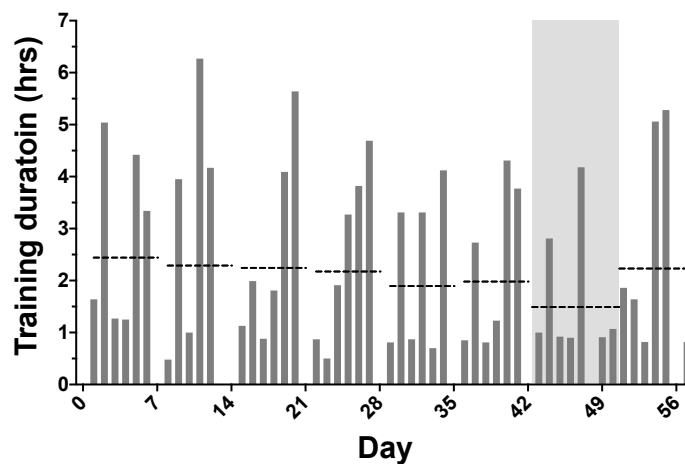
A G.3.2 Training and Intervention

The participant trained 2.15 hours/day for the 6 weeks prior to ST1, and 1.62 hours/day from ST1 to ST2 (Appendix Figure G.4). The participant's Training Stress Balance™ and Chronic Training Load™ values are presented in Appendix Figure G.3. Changes in resting and 50% PPO HR prior

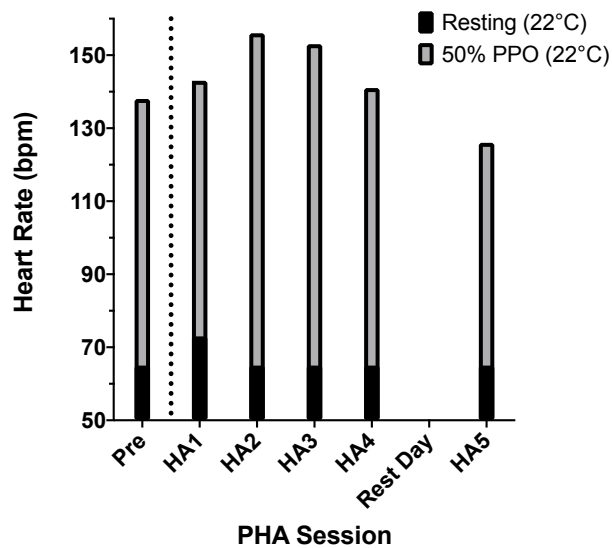
to sauna sessions are displayed in Appendix Figure G.5. Changes in bodyweight throughout the pilot study are shown in Appendix Figure G.6.



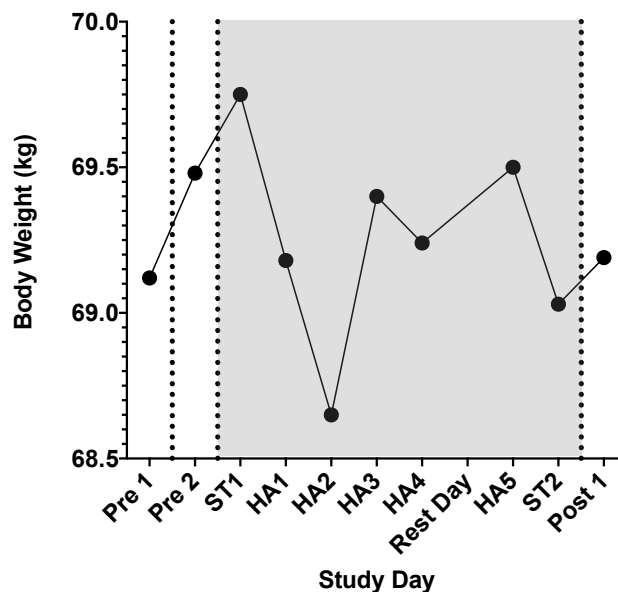
A Figure G.2 Chronic Training Load™ (CTL) and Training Stress Balance™ (TSB) for a well-trained cyclist (n= 1) prior to (42 days), during (8 days), and post (7 days) a passive heat acclimation pilot study (highlighted in grey). The study included a pre and post submaximal test and 5 sauna sessions (over 6 days). AU = arbitrary units.



A Figure G.3 Daily training duration data for a well-trained cyclist (n= 1) prior to (42 days), during (8 days), and post (7 days) a passive heat acclimation pilot study (highlighted in grey). The study included a pre and post submaximal test and 5 sauna sessions (over 6 days). Average daily loads for 7 day blocks and the pilot study are indicated with horizontal dotted lines.



A Figure G.4 Heart rate values at rest and data for a well-trained cyclist ($n=1$) prior to sauna heat acclimation (HA) sessions in a sauna. Pre heart rate values were taken during a HA familiarization session 8 days prior to HA1. A submaximal test (60 minutes at 50% peak power output) was conducted at 35°C the day before HA1.



A Figure G.5 Bodyweight for a well-trained cyclist ($n=1$) prior to, during, and post a sauna heat acclimation (HA) pilot study (highlighted in grey). The study included submaximal tests pre (ST1) and post (ST2) 5 sauna sessions (over 6 days). Pre 1 bodyweight was measured 12 days prior to ST1, Pre 2 was 7 days prior. Post 1 bodyweight was measured 8 days after ST2.

A G.4 Discussion

The purpose of this pilot study was to practically evaluate the devices and methods of the proposed sauna HA research protocol, while also gaining first-hand researcher experience of short-term sauna HA. The primary conclusion of this pilot study was the overall proposed protocol was not conducive to achieving the desired research outcome of the current thesis. Additionally, it was found that a number of the components of the research protocol needed to be better controlled/accounted for (e.g. sauna temperature and session duration) and evaluated (e.g. external and subjective training loads). These changes ideally ensure research outcomes that were robust and applicable to well-trained endurance athletes.

It was concluded by the researcher who participated in the piloting of this study protocol that its inclusion would not clearly benefit the findings of the current thesis, and including it would be potentially premature. Initially this HA protocol was intended to be conducted prior to the high-intensity interval interventions (HIIT) utilized in Study 2 (Chapter 4) as a method to induce HA adaptations (e.g. an increase in plasma volume⁶). It has been demonstrated that HA adaptations can increase endurance performance in temperate conditions¹⁴⁻¹⁶ under certain conditions. It was therefore initially hypothesized that HA prior to HIIT would potentially help maximize the benefits of intervals for endurance athletes. However, including the HA protocol prior the HIIT interventions in the overall study design with HIIT introduced an epistemological paradox. Currently, short-term sauna HA has not been demonstrated to induce the necessary adaptations to improve temperate endurance performance. Subsequently, given the overall HA/HIIT study design, this question of efficacy could not be confidently assessed until after research with the HIIT interventions was completed. In this scenario it could have been determined the HA protocol

had negligible effects on HA adaptations and performance outcomes. Therefore, the inclusion of this HA protocol could have needlessly impeded logistics of answering questions more relevant to the scope of the thesis (i.e. the effect of environmental temperature on HIIT). Additionally, as demonstrated by Study 2 and 3, the investigation of HIIT at different environmental temperatures provided novel results. Incorporating the current HA protocol prior the HIIT interventions could have skewed results (e.g. thermoregulatory findings in Study 2 and 3) and reduced the ecological validity of the overall HIIT intervention findings. Future research should assess the outcomes short-term HA in saunas prior to incorporating this methodology with HIIT in different environmental temperatures.

The researcher/participant experienced an unexpected increase in the magnitude of subjective fatigue during this pilot study that provided additional support for the decision to remove the HA protocol from the thesis. Importantly, the participant noted this increase in subjective fatigue did not coincide with their training volume (Appendix Figure G.4) or training load model (i.e. PMC; Appendix Figure G.3). However, it is not possible to confirm if this personal anecdote was a direct result of the HA protocol or other contributing factors (e.g. illness). Stanley et al.⁶ noted a decrease in participant training load during a sauna intervention in well-trained cyclists, but could not confirm if this was due to the additional stress from heat exposure. Nonetheless, it was reasoned if this magnitude of additional fatigue during the HA protocol was experienced by future research participants then substantial participant attrition could result, and consequently impede research progress for questions more relevant to the overall thesis. Additionally, this researcher/participant observation for increased fatigue provided information for improving future research methods and hypothesis generating relevant to the present protocol. It was realized the methods to track participant training load and fatigue were not sufficient enough to represent accurate scientific

findings. Indeed, given the unexpected magnitude for the increase in fatigue, the decision to collect participant training load and training modelling data was made post hoc. Future research into short-term sauna HA for endurance athletes should include valid objective and subjective methods of recording training load and training induced fatigue (e.g. Recovery-Stress Questionnaires).

This pilot study allowed for multiple methodological issues within the current HA protocol to be identified and addressed. In order to induce HA, the protocol of this study utilized a small portable commercially available sauna. HA with this device is potentially advantageous to athletes, as sessions can be conducted in a variety of locations (e.g. while traveling). Additionally, for research purposes, portable saunas offer a cost and space effective device for inducing HA. However, in the current pilot study it was noted that internal temperature of the sauna prior to use varied, and once the participant entered the device the internal temperature dropped considerably. Additionally, the duration of time the researcher/participant could tolerate in the sauna varied considerably, and did not seem to follow a trend (e.g. increase with each session). These issues potentially make it difficult to control the thermal impulse for HA sessions within and across individuals, and will have to be accounted for in future research. The current study recorded a number of subjective and physiological measures associated with adaptations to heat exposure (e.g. thermal sensation, HR, T_{C}) during rest and exercise in hot T_{A} , but future research with this protocol should consider including a measure for plasma volume. We are confident that sufficient measures were collected during the current protocol to assess HA in an experimental context (i.e. not in an $n=1$ context)¹⁷. However, the addition of a plasma volume measure would provide additional information for an adaptation that literature suggests is beneficial to performance in temperate conditions^{15,16}. Indeed, this measure could potentially provide valuable mechanistic data when incorporating the present HA protocol with other types of interventions (e.g. HIIT).

This HA protocol incorporated a 15-minute warm-up on a cycling ergometer which served the purpose of increasing T_C prior to the individual entering the sauna, and subsequently improving thermal impulse for the session. However, this pilot study offered the realization this warm-up could also potentially be utilized as a submaximal test and provide additional insight into participant fatigue throughout the HA protocol⁶. In the current protocol the participant performed the warm-up at 50% PPO (initial 10 minutes) and 75% PPO (last 5 minutes), and it was noted HR during the warm-up differed substantially during individual sessions (Appendix Figure G.5). Similar to the duration the participant spent in the sauna, HR did not display a linear trend as the protocol progressed. This signifies HR and other physiological measures during the warm-up may provide interesting findings in future research that could relate to fatigue, training load, and modelling fitness. However, contrary to the methods in the present pilot study, future research should collect power data during the warm-up to ensure consistency was maintained within and across individuals.

A G.5 Conclusion

The primary conclusion of this pilot study indicated the present research protocol was not overtly conducive to answering the primary questions of this thesis, and including it with HIIT interventions at different environmental temperature would have been potentially premature. Additionally, the present protocol offers potential as a viable research methodology for investigating HA in well-trained cyclists utilizing a portable sauna. However, a number of methodological issues (e.g. accounting for variations in sauna temperature and duration of heat exposure) should be addressed to ensure the occurrence of robust research outcomes. Future research on short-term heat acclimation utilizing portable saunas, in a protocol similar to what is presented here, could potentially provide valuable information to endurance athletes.

A G.6 References

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Appendix H Participant Heat Acclimation

A H.1 Sauna Heat Acclimation



A H.2 Submaximal Heat Acclimation Testing

