Adaptation to exercise for maximal aerobic capacity, submaximal aerobic efficiency, and cardiovascular adjustments: does the addition of heat stress induce greater improvements than exercise alone?

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Abstract

Background: Exercising in a hot environment often feels harder, and puts a greater amount of strain on the body than exercise in cooler temperatures. The extra strain caused by the heat has been utilised and explained extensively in the previous literature, by which training in the heat, and the concurrent physiological adaptations that arise (heat acclimation), has been shown to improve exercise performance in hot environments. It appears that the effect that heat acclimation can have on exercise performance in temperate environments, as opposed to hot, has been relatively overlooked in the literature. The physiological adaptations associated with the extra strain whilst exercising in the heat may not only induce performance benefits in temperate environments, but may also lead to positive resting cardiovascular adjustments.

Aim: The aim of this study was to determine what effect exercising with additional heat stress (35°C) has on maximal and submaximal aerobic capacity/performance in a moderate environment (18°C). The physiological adaptations obtained with exercise and additional heat stress was investigated, along with the impact they have on resting cardiovascular measures.

Methodology: In a randomised, matched control group study, eighteen moderately active males participated in a maximal and submaximal aerobic test, followed by an 11-day training protocol (five consecutive days, one day rest, six consecutive days) consisting of 60 minutes of incline walking each day on a treadmill at 50% of their $\dot{V}O_2$max in either a hot (35°C, 45% RH) or moderate (18°C, 53% RH) environment. Within four ± one day of completing the training protocol, the maximal and submaximal aerobic tests were repeated. Maximal aerobic capacity was measured in the maximal test; with submaximal $\dot{V}O_2$, heart rate and lactate measured to indicate changes during exercise. Core temperature, heart rate, plasma volume, forearm blood flow, whole body sweat rate, local sweat rate, and perceptual measures were taken throughout the 60 minutes of walking over the 11-day training period, in combination with resting heart rate and blood pressure measures to determine cardiovascular adjustments.

Results: Exercise, with or without heat stress improved maximal aerobic capacity by 7.0 ± 0.9 $mL \cdot kg^{-1} \cdot min^{-1}$ ($p < 0.001$), although, additional heat stress did not improve maximal aerobic capacity above exercise alone. The exercise protocol, irrespective of whether in a hot or moderate environment, lowered submaximal heart rate ($p = 0.008$) and relative $\dot{V}O_2$ ($p < 0.001$), but had no effect on submaximal blood lactate. The 11-day training protocol lowered resting heart rate ($p < 0.001$), reduced core temperature ($p = 0.039$), increased forearm blood flow ($p = 0.046$), and lowered perceived exertion ($p < 0.001$) for both groups. Additionally, the heat group had increased whole

body sweat rate ($p = 0.01$), and improved thermal comfort ($p = 0.024$). The exercise, regardless of environment, appeared to induce resting cardiovascular adjustments, although statistical significance was not reached.

Conclusions: Eleven days of exercise at 50% of $\dot{V}O_2max$, regardless of environment, can improve maximal and submaximal performance in a moderate environment and induce positive cardiovascular adjustments. Eleven days of exercise in 35°C can induce heat acclimation, illustrated through an increase in whole body sweat rate, and a reduction in exercising heart rate, core temperature and perceived exertion.

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List of Abbreviations

A

ANOVA	Analysis of variance

B

BLa	Blood lactate
BMI	Body mass index
BSA	Body surface area
$b \cdot min^{-1}$	Beats per minute

C

CBF	Cerebral blood flow
cm	Centimetres
CO_2	Carbon dioxide

E

ECF	Extracellular fluid
EHS	Exertional heat stroke

F

FI	Fixed-intenstiy

H

Hb	Haemoglobin
Hct	Haematocrit
HPL	Human Performance Laboratory
HSP	Heat shock protein

I

ISF	Interstitial fluid

K

kg	Kilograms
$kg \cdot m^{-2}$	Kilogram per square metre
km	Kilometres
$km \cdot h^{-1}$	Kilometres per hour

L

L	Litres
$L \cdot min^{-1}$	Litres per minute
$L \cdot h^{-1}$	Litres per hour
LSR	Local sweat rate

M

m	Metres
$m \cdot s^{-1}$	Metres per second
$MCAv_{mean}$	Middle cerebral artery mean blood velocity
mL	Millilitres
$mL \cdot kg^{-1} \cdot min^{-1}$	Millilitres per kilogram per minute
min	Minute
mm	Millimetres
mm Hg	Millimetres of mercury
$mmol \cdot L^{-1}$	Millimoles per litre
µL	Microlitres

N

Na^+/K^+ ATPase	Sodium-potassium adenosine triphosphatase
NE	Norepinephrine
NO	Nitric oxide

O

O_2	Oxygen

P

PV	Plasma volume

R

RER	Respiratory exchange ratio
RH	Relative humidity
RPE	Rating of perceived exertion

S

SD	Standard deviation
SE	Standard error
SkBF	Skin blood flow
SP	Self-paced
STPD	Standard temperature and pressure, dry
SWR	Whole body sweat rate

T

T_c	Core temperature
ThC	Thermal comfort
ThS	Thermal sensation
T_{sk}	Skin temperature
$\overline{T}_{sk}$	Mean skin temperature

U

USG	Urine specific gravity

V

V_E	Minute ventilation
$\dot{V}O_2$	Oxygen consumption
$\dot{V}O_2max$	Maximal oxygen uptake
VOP	Venous occlusion plethysmography

W

WBGT	Wet-bulb globe temperature

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Chapter One: Introduction

Large variations in environmental temperatures occur, but the human body has the ability to maintain its core temperature (T_c) within a narrow range. The body's ability to uphold a safe T_c ensures that homeostasis is maintained, thus allowing individuals to operate as effectively as possible. However, there are certain situations where T_c can be compromised that require additional (often behavioural) adjustments to prevent dangerous internal temperatures being reached. Exercise (especially high-intensity) in a hot environment is an example of this, and whilst it can be potentially dangerous, various researchers have found that repeated exposure to the heat in combination with a lower intensity of exercise can induce various positive adaptations to help protect an individual to subsequent heat exposures.

Exercising in a hot environment often feels harder, and puts a greater amount of strain on the body than exercise in cooler temperatures. The extra strain caused by the heat has been explained extensively in the literature, with various heat acclimation strategies being applied to induce physiological changes, which allow individuals to become 'heat adapted'. Exercise alone can induce positive physiological adaptations, but in combination with a hot environment, the human body develops cardiovascular, haematological, sudomotor, and metabolic adaptations over time that protects the body against further heat exposure. Additionally, these physiological adaptations also benefit future exercise performance in hot environmental conditions. For example, leading up to a race/event in a warm location some athletes will train in a hot climate, or artificially-induced hot environment. This allows the athlete to minimise future performance decrements when competing, as they have developed positive physiological adaptations to combat the negative effects of such an environment.

Previous literature has comprehensively concluded that heat acclimation provides performance improvements in a hot environment, which are directly related to the physiological adaptations that are induced. But, the impact that heat acclimation can have on exercise performance in a moderate/cool environment has received little attention. Similar to the "live high-train low" altitude theory developed by Levine and Stray-Gundersen (1997) where adaptation to a hypoxic environment induced performance improvements at low altitudes, the physiological adaptations achieved via heat acclimation may provide some benefits to exercise in a moderate/cool environment, and concurrently improve resting cardiovascular measures. Of the small number of associated studies, the main focus has been on highly trained athletes or soldiers, whilst utilising

cycling or walking as an exercise modality. Therefore, the impact that heat acclimation can have on running performance for a recreationally active individual in a moderate environment is yet to be quantified, however this holds great relevance to a larger proportion of the population, and is transferable to a large number of sports.

The aim of this thesis is to provide an extensive contemporary literature review that will identify reliable knowledge from which sensible hypotheses will be tested to investigate the impact of additional heat stress on maximal and submaximal aerobic performance, and resting cardiovascular adjustments in a moderate environment. The literature review (**Chapter Two**) will begin by explaining the concepts of human temperature regulation, leading into thermoregulation during exercise. Whilst this area of research is extensive, it is vital to the understanding of heat stress and acclimation; the literature review will aim to summarise the main concepts from the vast range of associated literature. The review will then begin to focus on the addition of heat stress and how this further impacts temperature regulation and performance, finally leading into heat acclimation. From the identified knowledge gained from the review of the literature, the aim and the hypotheses will be explained in **Chapter Three**. The methodology, equipment, and procedures will be documented in **Chapter Four** and the results of the study will be analysed and summarised in **Chapter Five**. **Chapter Six** will provide a comprehensive discussion that culminates in a general conclusion with possible directions for future research (**Chapter Seven**).

Chapter Two: Literature Review

2.1 Human Thermoregulation

The human body interacts with the environment to regulate internal temperature. Whilst the body generates heat through a number of different mechanisms, various modalities allow this heat to be exchanged from the body to the environment, in order to maintain an appropriate internal temperature. Section 2.1 of the literature review will aim to explain human thermoregulation through heat exchange modalities and parameters.

Human thermoregulation involves the maintenance of core body temperature within a narrow range (around 37°C), which is suitable for optimal functioning within the body. Previous literature has referred to a T_c of 37°C as a 'set point', a temperature by which thermoregulatory mechanisms are employed to uphold and is regulated for homeostasis and maintaining the body's internal environment (Campbell, 2008). Various models have been developed around this 'set point' in order to explain its effect on thermoregulatory responses, but more recently it has been proposed that the 'set point' may not be a point per se, but instead a T_c range, denoted the 'thermo-effector threshold zone' (Mekjavic & Eiken, 2006). Campbell (2008) states that, in general, humans tend to function with a T_c between 36.1°C and 37.8°C, which is able to be maintained over a wide range of ambient temperatures; a sedentary naked man is able to retain his T_c inside this range between ambient temperatures of 12°C and 60°C (Campbell, 2008). The hypothalamus is one of the most important structures involved in the regulation of body temperature and the prevention of T_c deviation outside of this threshold range (Werner, 1980; Wendt *et al.*, 2007; Campbell, 2008). Specifically, the posterior hypothalamus is involved in conserving heat within the body, whereas the preoptic-anterior hypothalamus initiates specific heat loss mechanisms (Campbell, 2008). Temperature regulation is an example of a "closed loop" system of regulation; the hypothalamus receives information on body temperature from various thermoreceptors; located in both the central and peripheral regions of the body (Cabanac, 1975; Gleeson, 1998). The central sensors are found in the hypothalamus, spinal cord, abdominal viscera, and the great veins, and are sensitive to temperatures between 30°C and 42°C (Campbell, 2008). The peripheral sensors are located in the skin and respond to both hot and cold temperatures, relaying the messages back to the appropriate part of the hypothalamus (Campbell, 2008). The hypothalamus acts as a coordinating centre to compare and integrate the information delivered from the central and peripheral thermoreceptors,

and consequently promote heat loss or gain from the body via various effector mechanisms (Boulant, 2000; Wendt *et al.*, 2007) (illustrated in Figure 2.1). The effector mechanisms are made up of both autonomic and behavioural responses; a mixture of conscious changes arising in the cerebral cortex and sub-conscious alterations often achieved via the sympathetic nervous system (Campbell, 2008).

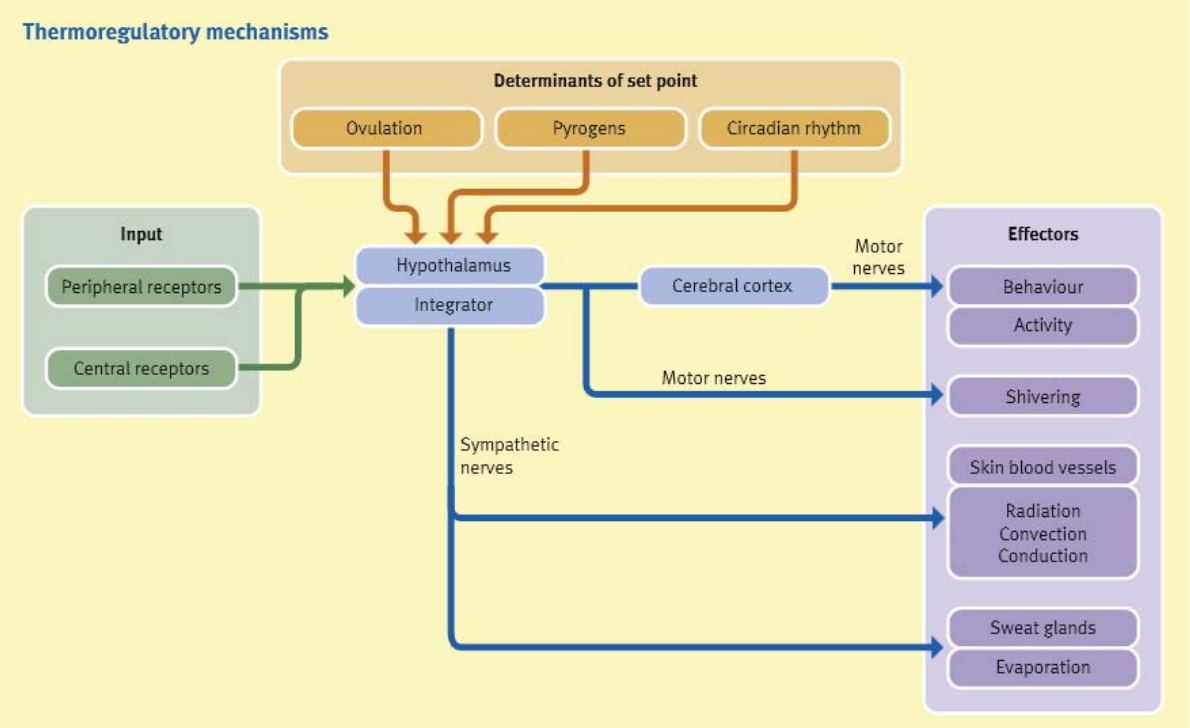

Figure 2.1. Thermoregulation within the body and the mechanisms for control (Campbell, 2008).

2.1.1 Heat Exchange Modalities

Heat can be exchanged between the human body and the environment which results in either heat loss or heat gain by the body, the majority of which is determined by the environmental conditions. This heat exchange can occur via four different mechanisms: radiation (+/-), convection (+/-), conduction (+/-) and evaporation (-). These mechanisms can be further divided into two different groups: dry heat exchange (radiative, conductive and convective mechanisms), which is reliant on a temperature gradient within the body, or between the body and the environment; and wet heat exchange (evaporative heat exchange).

Radiation: Radiation can be either the loss or gain of heat from the environment in the form of infrared heat rays. The transfer of heat via radiation always occurs from a hotter body to a cooler one, so for heat loss to occur via this mechanism, a large temperature gradient is required between

the skin and the air (Campbell, 2008). Therefore, as air temperature rises, radiation can be a mechanism of heat gain and does not contribute much to heat loss. In saying that, radiation can account for almost 60% of heat loss in a moderate environment, in a sedentary individual (Wendt *et al.*, 2007).

Conduction: Conduction is the transfer of heat between two objects that are in direct contact (Campbell, 2008). This process can occur within an individual, as well as between an individual and the environment, but a temperature gradient between the two is also required. Conduction can be a means of heat loss or gain through the transfer of heat from the body to an object (or vice versa). The relative contribution of conduction to heat loss is small, and in a sedentary state accounts for only 3% of whole body heat loss in a moderate environment (Wendt *et al.*, 2007).

Convection: Convection is the transfer of heat through a moving gas or liquid (Wendt *et al.*, 2007) and its contribution to total body heat loss is usually around 10-20% in a moderate environment. But, heat loss via convection will increase with greater air movement, i.e. increased wind velocity (Campbell, 2008). Just as heat loss via convection can be increased, it can also be hindered by clothing which limits the air flow over the skin (Campbell, 2008). For convection to occur, similar to radiation, a temperature gradient is required between the skin and the air, so at higher temperatures heat loss via convection is reduced (Wendt *et al.*, 2007).

Evaporation: Evaporation of a liquid from a surface requires energy (heat) to transform it from a liquid to a gas; with the energy for this conversion being sourced from the surrounding skin, resulting in heat loss from the body (Campbell, 2008). Evaporative heat loss consists of both insensible water loss and sweating. Insensible water loss refers to water loss from the body that goes undetected by the individual, and consists of water lost during breathing (respiratory evaporative heat loss) and water lost via diffusion through the skin (Commission, 2001). The majority of evaporative heat loss occurs via the mechanism of sweating, which can be detected by the individual involved. Heat loss via evaporation depends on the differences in the water pressures between the skin and air, rather than the skin and air temperature gradient (Blatteis, 1998). Water pressure at the skin needs to be higher than the environment for heat loss via evaporation to occur (Blatteis, 1998).

The contribution of each of the heat exchange mechanisms to heat loss or gain is subject to change and is dependent upon a wide range of environmental factors, in combination with physical activity levels.

2.1.1.1 Heat Balance

Given the nature of the human body and the underlying aim of temperature regulation to maintain T_c within an 'appropriate' range, the maintenance of heat balance is essential. Heat balance can be defined as when heat loss (to the environment) from the body is equal to heat gain (production), thereby resulting in neither a rise or fall in T_c outside of the usual daily 1°C fluctuations (Commission, 2001). Heat is produced in the body via a number of different mechanisms:

Basal Metabolism: Involves the minimum heat production that is required for the maintenance of the human body for general function. Basal metabolism can account for a large amount of heat production; the amount produced by each person is dependent on their body size, age and sex (Campbell, 2008).

Dietary-induced Thermogenesis: Following the intake of food, heat production can increase by 10-15%, especially with the consumption of large amounts of protein (Campbell, 2008).

Non-shivering Thermogenesis: This mechanism of heat production is not significant within the majority of humans, with the exception of infants. Infants (as well as animals that hibernate during winter) have a large proportion of brown adipose tissue in their body; which is brown due to the high mitochondrial content. This mitochondria is referred to as 'uncoupled', meaning that their stimulation within the brown adipose tissues results in the production of heat instead of ATP (Campbell, 2008).

Hormonal: Various hormones, for example catecholamines, have an influence over heat production via their stimulation of metabolic pathways (Campbell, 2008). The basal metabolism of the body is controlled by the thyroid.

Muscle Contraction: This is divided into two categories – voluntary (behavioural) and involuntary (shivering) (Campbell, 2008). At rest, muscle contraction has a very small contribution to heat production within the body, but when moving and exercising, this mechanism can lead to huge increases in heat production, which can pose problems for the maintenance of heat balance.

For the human body to maintain heat balance, the heat exchange mechanisms must result in enough heat loss to offset the heat production. The heat balance equation illustrates the interaction of the heat loss and heat gain mechanisms, therefore determining the resultant heat loss or heat gain by the body:

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

Where:

S = Heat storage

M = Metabolism

W = Work rate (muscle contraction)

R = Radiative heat transfer

C = Convective heat transfer

K = Conductive heat transfer

E = Evaporative heat transfer

The value of S will indicate whether the body is in a state of heat balance or not; if S is positive then heat production outweighs heat loss, and heat is being stored (and there will be a resultant rise in T_c). If S is negative then more heat is being dissipated to the environment than heat being produced, and T_c will decrease. Whereas, if S is equal to zero, heat loss is equal to heat production and heat balance exists (Commission, 2001). The contribution of both the heat production and heat exchange mechanisms is highly dependent upon the environmental conditions as well as the activity levels of an individual.

2.1.2 Heat Exchange Parameters

The environment that an individual is exposed to has a huge influence on their ability to maintain heat balance. Various parameters affect heat exchange and balance. These include: dry bulb (ambient) temperature, radiant temperature, air velocity, humidity, body surface area (BSA), and clothing.

Ambient temperature: This is the average temperature of the environment surrounding an individual, and will fluctuate naturally on a daily basis (Commission, 2001). Ambient temperature is what most people refer to in everyday use (for example; "it is 25°C outside today"), and often environments are assessed primarily in regards to the ambient temperature, but the importance of the other parameters should not be overlooked (Parsons, 2002).

Radiant Temperature: Radiative heat exchange involves the heat loss or gain from the environment to the body in the form of infra-red rays. The influence that radiant temperature has on heat exchange is in addition to the influence of air temperature, and is often referred to as either mean

radiant temperature (provides an overall average value) or plane radiant temperature (provides information associated with the direction of radiant exchange) (Parsons, 2002). This type of exchange occurs between the body and all surrounding environments, with one of the major interactions being solar radiation, the interaction of the sun and the human body (Parsons, 2002).

Air Velocity: Air velocity provides a description of the movement of air around an object, and will directly affect the heat exchange modalities of convection and evaporation; an increase in air velocity will result in greater heat loss from the body to the environment via both of these modalities. Air velocity in combination with air (ambient) temperature affects the rate at which vapour or warm air is taken away from the body (Parsons, 2002). The influence of air velocity on temperature regulation has been illustrated by Epstein and Moran (2006) who described that an alteration in wind speed of 0.1 $m \cdot s^{-1}$ is equivalent to a change of 0.5 °C in ambient temperature (up to a 1.5 °C change).

Humidity: Humidity is the partial pressure of water vapour in the air, and the difference between the partial vapour pressures of the skin and the environment is the main influence and driving force for heat loss from the body (Parsons, 2002). Humidity in the environment is even more important when exercising, as evaporation becomes the major mechanism for heat exchange to the environment. Humidity can be described in two different ways: Relative humidity (RH), which Kerslake (1972) defined as 'the ratio of the prevailing partial pressure of water vapour to the saturated water vapour pressure at the prevailing temperature' (p. 11); and absolute humidity, which describes the mass concentration or density of water vapour (units being mass per unit volume of moist air – $kg \cdot m^{-3}$).

Body Surface Area: This parameter represents the surface area of the body that is available for heat exchange, and is estimated by a simplified equation by Du Bois and Du Bois (1916) if both height and weight are known:

$$A_D = 0.202 \times W^{0.425} \times H^{0.725}$$

Where:
A_D = Dubois surface area (m^2)
W = Weight of the body (kg)
H = Height of the body (m)

Individuals of differing heights and weights will have different surface area values and will have different heat transfer coefficients; those with larger body surface areas can be expected to have greater potential for heat loss (Parsons, 2002).

Clothing: The amount of clothing worn can greatly affect one's ability to maintain heat balance in both cool and warm environments. Clothing restricts both insensible and sensible heat loss (Parsons, 2002). The evaporative rate of the sweat is limited as the amount of clothing worn increases (Nagata, 1978), and can be restricted by up to 78% (compared to when nude) (Havenith *et al.*, 1999).

Metabolic Heat Production: The body converts chemical potential energy that is in the form of carbohydrates, fats and proteins into cellular work and also heat; following the first law of thermodynamics (energy is converted from one form to another). During exercise, metabolic heat production increases considerably and this is defined by the equation M ± W; or essentially, the difference between the total heat produced and the total work being performed by the body (Parsons, 2002). The increase in metabolic heat production in various cases is either voluntary or involuntary. For example, often in cooler environments shivering is initiated via autonomic processes which increase the metabolic heat production involuntarily as a protective mechanism. Behavioural changes that are initiated voluntarily by the individual will either increase or decrease metabolic heat production (depending if ceasing or initiating movement/exercise) and are often associated with stimuli from the environmental factors.

2.1.3 Section Summary

The human body produces heat through a number of different mechanisms, and the subsequent heat is exchanged with the environment in order to reduce heat storage, maintaining thermoregulation. Four different modalities of heat exchange exist, but environmental conditions (temperature, RH etc.) greatly affect the body's ability to dissipate this heat. Overall, the human body is very efficient at keeping inside the narrow temperature range. It is only when multiple adverse influences are combined, i.e. high-intensity exercise in a hot-humid environment, that central body temperature can be compromised. Usually the human body will employ either voluntary or involuntary protector mechanisms before this dangerous level is reached.

2.2 Temperature Regulation at Rest

Temperature regulation is controlled by both conscious (behavioural) and subconscious (autonomic) means. Both are extremely important in the protection of T_c, with responses arising from inputs via sensors in the skin and core body regions. Section 2.2 of the literature review will aim to explain the different processes involved in autonomic temperature regulation (skin blood flow, sweating, and shivering), as well as the conscious behavioural changes and their contribution to thermoregulation.

2.2.1 Autonomic Regulation

Autonomic regulation of body temperature is a vital process for survival. The hypothalamus is the commanding centre for autonomic temperature regulation, whereby it receives information from various areas around the body (namely core and skin) and acts to integrate this information to produce the appropriate action. Both T_c and mean skin temperature ($\overline{T}_{sk}$) are important in determining the autonomic responses; and whilst some studies have suggested that T_c has more of an impact, illustrated by the proposed $T_c/\overline{T}_{sk}$ contribution ratio of 2:1 to 4:1 for autonomic responses (Frank *et al.*, 1999), other research has concluded through well-developed and executed studies that autonomic responses are a result of the summation of the internal and $\overline{T}_{sk}$ contributions, which contribute linearly to the responses produced (Cheung *et al.,* 1995; Nadel *et al.,* 1971). These processes are not under conscious control of the individual involved, and Figure 2.2 illustrates the variety, and complexity, of different responses that can occur depending on the environmental conditions and the subsequent pathway involved (Romanovsky, 2007).

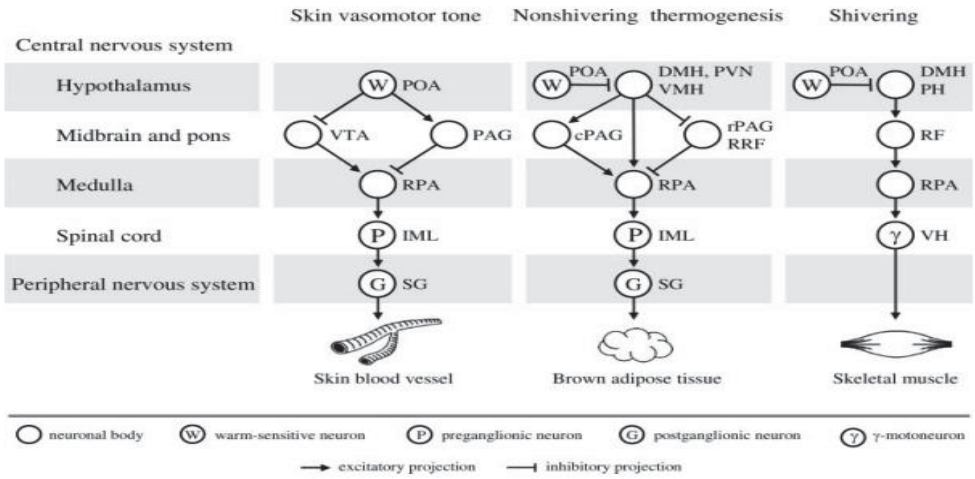

Figure 2.2. Efferent neuronal pathways for control of skin vasomotor tone, nonshivering thermogenesis, and shivering in the rat (Romanovsky, 2007). DMH, dorsomedial hypothalamus; IML, intermediolateral column; rPAG, rostral PAG; PH, posterior hypothalamus; PVN, paraventricular nucleus; RF, reticular formation; RPA, raphe/peripyramidal area; RRF, retrorubral field; SG, sympathetic ganglia; VH, ventral horn; VMH, ventromedial hypothalmus; VTA, ventral tegmental area.

A change in vasomotor tone is one of the first and least energy taxing autonomic responses that occurs during thermoregulation, with constriction of the peripheral blood vessels helping to reduce heat loss; vasodilation (expansion of the blood vessels) acting as a method of increasing heat loss (Cabanac, 1975). Peripheral vasodilation results in greater blood flow to the skin, which aids heat loss via the mechanisms described in the previous section, one of most effective being evaporative water loss via sweating. When vasoconstriction of the blood vessels is not sufficient to maintain thermoregulation (i.e. in cold environments), the efferent pathway to skeletal muscle is activated and shivering thermogenesis results. These various autonomic responses will be discussed below in more detail.

2.2.1.1 Skin Blood Flow

Blood flow to the skin is controlled via the sympathetic nervous system and can be altered as a result of information relayed from the hypothalamus (Rowell, 1977). As discussed above, blood flow to the skin will either increase or decrease depending on the information passed on from T_c and $\overline{T}_{sk}$, which causes alterations in one or both of the branches of the sympathetic nervous system via the adrenergic vasoconstrictor system, or the cholinergic vasodilator system (Minson *et al.*, 2001). Skin vasodilation is a defence mechanism against the heat, whereas skin vasoconstriction is a defence mechanism against the cold (Cabanac, 1975). When heat or cold stress is not present, skin blood flow (SkBF) at rest is around 250 $mL \cdot min^{-1}$, which can be increased substantially, as shown during hyperthermia where SkBF can reach as high at 6-8 $L \cdot min^{-1}$ (Charkoudian, 2003). The skin is second only to the skeletal muscle in the extent of vasodilation that can be achieved (González-Alonso *et al.*, 2008) and under heat stress the skin can account for 50-70% of the cardiac output (as opposed to only 5-10% at rest in a moderate environment) (Rowell, 1977). With such a mass distribution of blood to one area of the body, it is important that other process are not disrupted as a result – this is achieved through an increase in overall cardiac output during such cases (Charkoudian, 2003), and at times vasoconstriction occurring in the splanchnic region and a resultant redistribution of blood (Rowell *et al.*, 1966). Vasodilation and vasoconstriction of blood vessels in the peripheral regions of the body can be achieved via both reflex and local control/changes.

Reflex Control of Skin Blood Flow

The changes in SkBF that occur via the sympathetic nervous system are achieved via changes in the activity of either the vasodilator system, the vasoconstrictor system, or at times both (Charkoudian, 2003). This arrangement is termed reflex control and will affect the vasomotor tone of the majority of the body (rather than a localised area). Both T_c and $\overline{T}_{sk}$ are involved in the reflex control; at

moderate internal temperatures the vasoconstrictor system is always active (even with small environmental temperature changes) in order to maintain this functional T_c (Charkoudian, 2003). Whereas, under the influence of heat stress and elevated T_c, the role of the vasoconstrictor system is largely outweighed by the active vasodilator system which accounts for 80-95% of the resultant elevated SkBF (Kellogg *et al.*, 1998). Unlike the vasoconstrictor, the vasodilator system is not tonically active; only recruited at times of need i.e. elevated T_c. The active vasodilator system has been suggested to be facilitated via the sympathetic cholinergic nerves (Charkoudian, 2003); along with nitric oxide (NO), which has been proposed to have a potential mechanistic role in this active vasodilation (Kellogg *et al.*, 1998). Interestingly, Kellogg *et al.* (1998) concluded that NO is not required for the main primary control of active vasodilation but is mandatory for full expression of cutaneous vasodilation.

Upon propagation of the vasoconstrictor nerves controlling cutaneous vasoconstriction, norepinephrine (NE) is released and binds to α1- and α2-adrenergic receptors located on the postsynaptic membrane (Wilson *et al.*, 2002). The binding of NE to the adrenergic receptors initiates the reduction in blood flow to the periphery of the body. NE release increases with whole body cooling and decreases in times of heat stress, with the potential decrease in adrenergic uptake of NE combining to result in the reduced activity of the vasoconstrictor system (Hodges & Johnson, 2009).

Local Control

Alterations in local temperature of the skin can also contribute to changes in SkBF, alongside the changes in T_c and $\overline{T}_{sk}$. The response to local heating of the skin occurs in two independent phases, as illustrated by Figure 2.3. The initial phase involves a rapid increase in SkBF followed by a decrease, and then a slow increase up to a certain point whereby it plateaus, usually around 25-30 minutes of local warming (Charkoudian, 2003). Phase 1 of the local heating response occurs independent of the adrenergic system required for the reflex control of SkBF and is proposed to be mediated primarily by an axon reflex mechanism (Minson *et al.*, 2001). Phase 2, where cutaneous blood flow rises to a plateau is mediated by NO, independent of the first axon reflex mechanism. This has been illustrated by Minson *et al.* (2001) where phase 2 was inhibited by the inhibition of nitric oxide synthase, but not by the blockage of the nerves involved in the axon reflex.

Along with local heating, local cooling of the skin can cause dramatic changes in the vasculature, with vasoconstriction reducing SkBF to very low levels upon the appropriate stimulus (Kellogg *et al.*, 1999). The vasoconstriction response to local cooling is not dependent, and does not require an

intact central nervous system, instead it is reliant on the local activation of adrenergic nerves (Charkoudian, 2003).

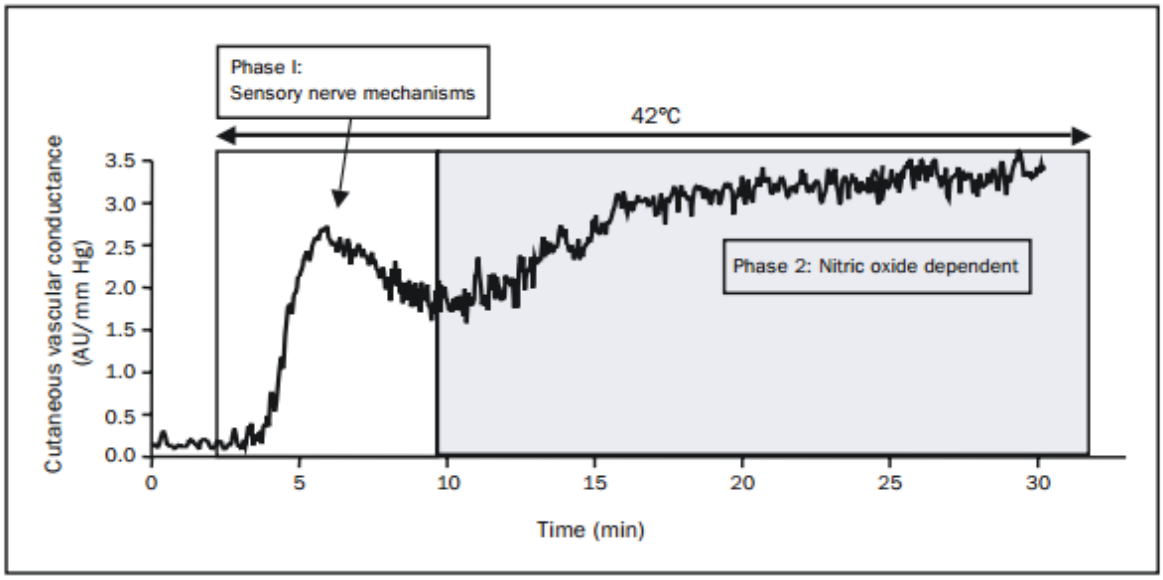

Figure 2.3. The typical bi-phasic cutaneous vasodilation during 30 minutes of local warming at 42°C (Charkoudian, 2003).

The reflex and local control of SkBF discussed provides a brief overview of what occurs during heating and cooling of the skin and internal temperatures, but the cutaneous blood flow can also be greatly affected by outside influences (for example, exercise), which will be discussed in more detail in following sections.

2.2.1.2 Eccrine Sweating

Eccrine sweating involves the expulsion of a liquid from sweat ducts onto the skin for evaporation. Sweating is a method of heat loss; when the sweat is evaporated, energy is released which can result in lowered $\overline{T}_{sk}$ and T_c (Wilke *et al.*, 2007). Sweating becomes increasingly important for heat loss in hot environmental conditions when other methods of heat dissipation from the skin (radiation, conduction) have become means of heat storage. Sweat that is secreted onto the skin is a substance comprising of 99% water; other components including sodium, chloride, potassium, calcium, magnesium, lactate, ammonia, amino acids, urea and bicarbonate, alongside several proteins and peptides (Wilke *et al.*, 2007). The composition of eccrine sweat will be different for each person, depending on hydration, exercise, health status, and region of the body (Wilke *et al.*, 2007).

The human body contains two types of sweat glands – eccrine sweat glands and apocrine sweat glands. Apocrine glands are located only in the 'hairy' body areas (axillia, mammary, pineal and genital regions) and exist from birth but only become active from puberty (Wilke *et al.*, 2007). The

apocrine glands are responsible for sweat expulsion as a result of emotional stimuli which is not classed as thermoregulatory sweating, as is eccrine sweating (Shibasaki *et al.*, 2006). Eccrine glands are distributed over the whole body with the number of sweat glands varying for each individual - ranging from 1.6 to 5.0 million in total (Wilke *et al.*, 2007). The density of these eccrine glands is non-uniform across the body, the greatest density found on the forehead (Shibasaki *et al.*, 2006) and an average density of 200 sweat glands per square centimetre (Wilke *et al.*, 2007). Eccrine sweat glands are made up of a secretory coil located in the lower dermis of the skin which is connected to a duct that extends through the dermal layer and opens on the skin surface (Shibasaki *et al.*, 2006). Before travelling through the sweat gland, the liquid is referred to as a 'precursor' fluid, or 'primary sweat', which is very similar to plasma, without the proteins (Bulmer & Forwell, 1956; Shibasaki *et al.*, 2006; Wilke *et al.*, 2007). The precursor fluid is driven through the sweat gland via an electrochemical gradient, though the formation of this gradient is still relatively unclear (Wilke *et al.*, 2007). As the fluid travels through the gland, the duct acts to reabsorb ions, primarily sodium and chloride, from the precursor sweat, driven by a Na^+/K^- ATPase pump relocating the sodium from the duct into the interstitial fluid (ISF) (Wilke *et al.*, 2007). This results in the sweat that is produced and excreted on the skin's surface being hypotonic in relation to plasma, minimising ion losses from the body (Shibasaki *et al.*, 2006).

The proportion of ions found in sweat will be dependent on the sweat rate of the individual. Sweat rate is determined not only by the output of sweat being produced from each individual gland, but also by the density of the active sweat glands (Kondo *et al.*, 2001). Different circumstances will account for increases in sweat rate, i.e. during heat stress and exercise it would be expected that sweat rate would increase. In such conditions, a huge amount of sweat can be produced; with the highest reported sweat rate recorded at >3 $L \cdot h^{-1}$ (average maximum sweat rate for exercising humans is ~1.4 $L \cdot h^{-1}$) (Shibasaki *et al.,* 2006). Extremely high rates of sweat loss cannot be maintained for long periods of time, due to both water and ion losses from the body. As the sweat rate increases, the ability of the duct to reabsorb ions decreases due to the reabsorption mechanisms becoming overwhelmed by the amount of sweat present in the duct (Shibasaki *et al.*, 2006). The ion and fluid losses will be required to be replaced during and/or after exercise to prevent detrimental effects to the functioning of the body.

Sweating is regulated by the central nervous system, primarily the hypothalamus, with acetylcholine released from the cholinergic sudomotor nerves which then binds to the eccrine sweat glands to initiate and moderate sweat rate (Shibasaki *et al.*, 2006). Much like the previously discussed thermoregulatory factors, sweat rate is driven by changes in T_c and $\overline{T}_{sk}$. Shibasaki *et al.* (2006) depict

the contribution of both T_c and $\overline{T}_{sk}$ (Figure 2.4); as T_c increases, initially there is no increase in sweat rate, up until a 'threshold' point where sweat rate rises steadily with the rise in T_c. The threshold for the initiation of sweating can be shifted by $\overline{T}_{sk}$, whereby increased skin temperature will cause sweating to start earlier, and decreased $\overline{T}_{sk}$ will prolong the initiation of sweating. This concept has most likely been experienced by almost every individual; when walking around on a hot summer's day, sweating will be initiated a lot earlier compared to the same activity on a cooler day, due to the combination of a faster rise in T_c, and higher $\overline{T}_{sk}$. Alongside the central control of sweating, peripheral influences can also impact sweat rate, where an increase in the local skin temperature (T_{sk}) increases sweat rate, while local cooling reduces the sweat rate, but the mechanisms behind these changes is still relatively unclear (Shibasaki *et al.*, 2006; Wilke *et al.*, 2007). In conjunction with the most important influences on sweating discussed (T_c and $\overline{T}_{sk}$), there are also outside stimuli that can affect thermoregulatory sweating, such as sex, physical fitness, menstrual cycle, circadian rhythm and also environmental factors (ie. humidity).

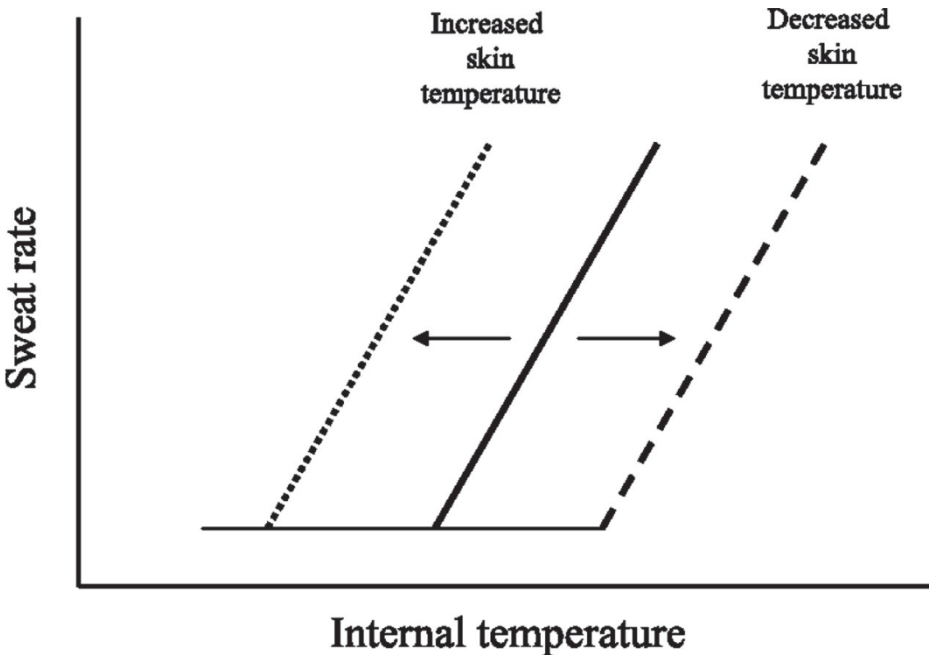

Figure 2.4. The interactions and contributions of core temperature and skin temperature to sweat rate (Shibasaki *et al.*, 2006).

2.2.1.3 Shivering

Shivering is a response that occurs in a cold environment and is employed after the initial response of cutaneous vasoconstriction. It involves involuntary contractions of the muscles, which occur very quickly and generate heat. Much like sweating, shivering is initiated through changes in either T_c or

$\overline{T}_{sk}$; with either a rapid drop in $\overline{T}_{sk}$ or a decrease in T_c to a shivering 'threshold' point (Armstrong, 2000). Prior to the onset of shivering, the hypothalamus receives information from afferent cold receptors located in the internal organs, skin, and spinal cord, and this information is integrated and relayed on from the hypothalamus. In this case, the efferent pathway is via the motor nerves, which are associated with skeletal muscle and the associated shivering response.

2.2.2 Behavioural Regulation

In more recent years behavioural regulation of body temperature has become a popular topic, with its importance for long-term temperature regulation of the body becoming increasingly apparent. Thermoregulatory behaviour has been defined as a behaviour that establishes a preferred condition for heat exchange between an organism and its environment (Commission, 2001), and is used as a conscious 'protective mechanism' against a substantial rise or fall in T_c. When possible, behavioural responses are often employed to prevent the autonomic responses discussed above, as these can be potentially energy and/or water consuming (Schlader *et al.*, 2010). As opposed to the autonomic responses which have a limited capacity, behavioural regulation is virtually unlimited, making it crucial at rest, as well as during exercise (when possible, i.e. during self-paced exercise). Some examples of behavioural regulation include turning on/off a heater, changing body position, seeking shade.

Thermal comfort (ThC) is a term that is often referred to in association with thermo-behavioural regulation and is a subjective term to describe the level of indifference with the surrounding environment (Schlader *et al.*, 2010). Thermal discomfort appears to be the main driving force behind the initiation of behavioural responses, but the contribution that the skin and core temperatures have to ThC has not been well defined in the previous literature. Various studies have proposed that T_c alone is responsible for the initiation of behavioural changes (Cabanac *et al.*, 1971; Cabanac *et al.*, 1972), whereas others have concluded that $\overline{T}_{sk}$ is more important in relation to determining ThC and therefore behaviour (Schlader *et al.*, 2009; Schlader *et al.*, 2010). The lack of agreement from the aforementioned studies is likely due to the methodological differences, which makes it difficult to directly compare between them. When there is behavioural freedom (no restrictions as to what environment to stay in, and/or exercise intensity), $\overline{T}_{sk}$ is viewed as being the most important thermal drive to changes in ThC and behaviour, due to the fact that behaviour is employed prior to changes in T_c (Schlader *et al.*, 2009). However, various methodologies have been used, which made it difficult to make a valid comparison. For example, Schlader *et al.* (2009) used rectal thermometers as a measure of T_c, however other studies have used oesophageal temperature (Cabanac *et al.*, 1972;

Flouris, 2011), which has previously been reported as having a greater sensitivity to detecting changes in T_c (Cranston *et al.*, 1954). Contrary to merely focussing on either T_c or $\overline{T}_{sk}$ as the primary thermal inputs in the development of ThC, Flouris (2011) concluded that thermal discomfort is actually affected by the thermal status of the entire body, and established through a novel method of quantifying thermoregulatory responses that the behaviour employed is done to help maintain a threshold mean body temperature, instead of combatting changes in individual areas of the body (Flouris & Cheung, 2009). Thermoregulatory behaviour is employed during times of thermal discomfort in both the cold and heat, but interestingly, the behavioural control initiated as a response of thermal discomfort appears to be more precise when in the heat, than in the cold (Schlader *et al.*, 2009).

Due to the methodological differences outlined from the previous research, it is currently difficult to make any conclusive agreement about thermoregulatory behaviour. However, behavioural responses at rest help to prevent changes in T_c and therefore prevent the initiation of autonomic/energy consuming processes.

2.2.3 Section Summary

The literature to-date surrounding autonomic responses in the regulation of internal temperature is sound. Increased peripheral vasodilation and skin blood flow primarily contribute to heat dissipation in a warm environment, with the sweating response initiated if further heat removal is required via evaporation. In a cold environment, cutaneous vasoconstriction ensues, followed by shivering, to preserve and then generate heat, respectively. Unlike the autonomic responses, behavioural regulation of temperature is a more recent area of research. Unfortunately, due to methodological differences, conclusions surrounding the extent of the contributions of behavioural alterations to thermoregulation are difficult to make. Therefore, this is an area requiring future research; it is as important as the autonomic processes in the regulation of internal temperature, but there is a lack of equivalency in the extent of the literature on both topics.

2.3 Temperature Regulation during Exercise

Whilst regulation of body temperature at rest in most environments is a relatively simple concept to understand, temperature regulation while exercising, and even more so with the addition of heat stress, is a more complicated process. During physical activity, metabolic heat production increases considerably (can increase by up to 10- to 20- fold), and because the human body is not entirely efficient , about 70-80% of the energy expended gets converted to heat (Powers & Howley, 2009). It is this increase in heat production within the body, in combination with competition between organs for cardiovascular adjustments, which can create problems within the human for regulating body temperature, and subsequently impact on performance. Section 2.3 of the literature review will aim to quantify the physiological responses that are involved in temperature regulation during exercise alone, and during exercise combined with heat stress. Furthermore, there will be a brief review of the literature associated with fixed-intensity and self-paced performance, alongside maximal aerobic capacity and how these are impacted by a hot environment. Finally, strategies developed to minimise the performance decrements will be reviewed.

2.3.1 Physiological Responses

Metabolic heat production is dependent on the intensity of exercise; as exercise intensity increases, the increased muscle contractions contribute to the higher metabolic heat production (Lim *et al.*, 2008; Powers & Howley, 2009); illustrated by 1) and 2) in Figure 2.5. The higher metabolic heat production during exercise will inevitably cause an increase in T_c to some extent, but this is not indicative of a 'loss' in thermoregulation of the body; instead thermoregulation can still be achieved during exercise - as observed when there is a plateau in the rise of T_c. It is this plateau that illustrates that the body is in thermal balance and heat loss is equal to heat gain. Thermal balance is seen to be disrupted if T_c continues to rise without a characteristic plateau, typical in higher intensity exercise and in hot environments, and can lead to hyperthermia (Powers & Howley, 2009).

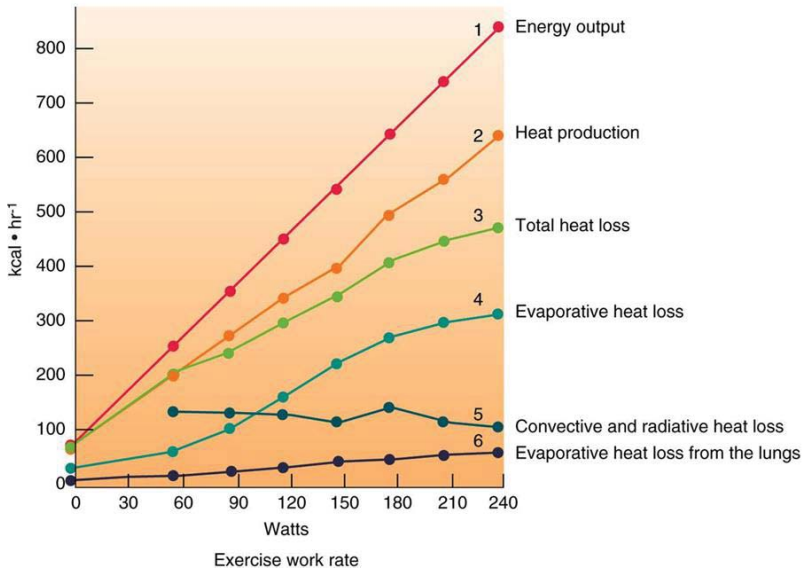

Figure 2.5. Heat exchange at rest and during cycling exercise at increasing rates of work (Powers & Howley, 2009).

During exercise, evaporation of sweat from the skin is the main mechanism of heat removal from the body and accounts for the majority of heat loss (Powers & Howley, 2009). Figure 2.6 illustrates the contributions of not only evaporation, but also convection and radiation to heat loss during exercise in a cool environment. Both convective and radiative heat loss mechanisms play small roles in heat dissipation during exercise (though also dependent on environmental conditions), regardless of the intensity; as opposed to evaporative heat loss which increases as a function of work rate (Powers & Howley, 2009). Sweating, and the evaporation of sweat from the skin, requires increased SkBF during exercise, which calls for redistribution of cardiac output. This can result in some competition between the skin and the muscle for blood flow (Rowell, 1974). During exercise, both thermoregulatory and non-thermoregulatory responses are accountable for the distribution of the cardiac output – skeletal muscle requires a significant portion of blood flow to maintain the metabolic demands of exercise, as does the skin to dissipate heat in order to prevent hyperthermia and fatigue (Wendt *et al.*, 2007). In such cases, cardiac output is often redirected from areas of less importance (at that instance), such as splanchnic and renal tissue, illustrated by vasoconstriction occurring in these areas at the onset of exercise (Rowell *et al.*, 1966). Often, the redistribution of part of the cardiac output is not satisfactory, and to maintain the required level of muscle blood flow as well as compensating for the increasing demands for blood flow to the skin, cardiac output must increase (Levick, 2010). The increase in cardiac output is most likely accounted for by an elevated heart rate, as cutaneous vasodilation reduces cardiac filling pressure and subsequently stroke volume tends to decrease during higher intensity, prolonged exercise (Levick, 2010).

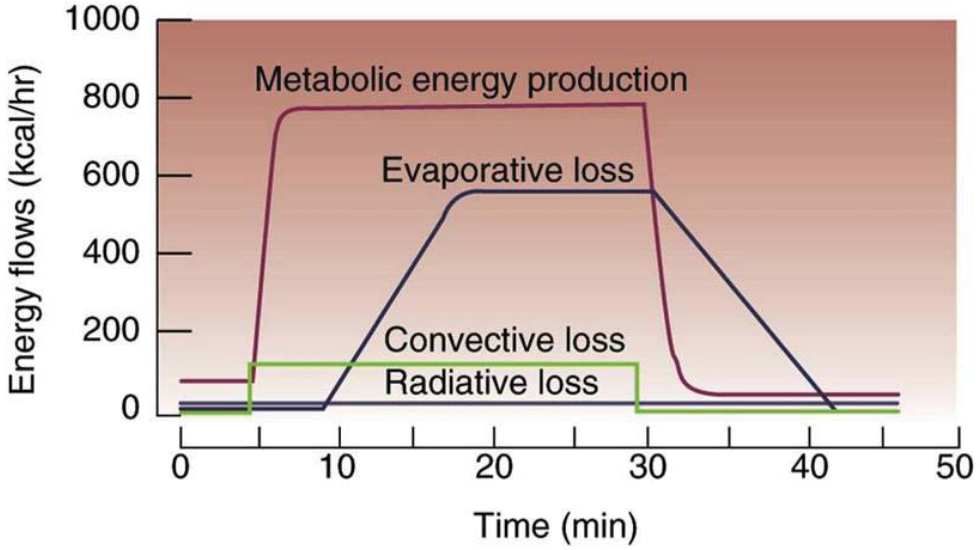

Figure 2.6. Alterations to metabolic heat production and evaporative, convective, and radiative heat loss over the course of a 25 minute submaximal exercise bout in a cool environment (Powers & Howley, 2009).

Unlike at rest, levels of SkBF are limited during exercise, likely due to the previously discussed conflicting demands for cardiac output. Figure 2.7 illustrates the SkBF response at both rest and during exercise. Exercise not only delays the cutaneous vasodilation response compared to rest (A. and B. in Figure 2.7), but there is also a limit placed on the extent of blood flow to the skin that can be achieved as T_c approaches 38°C (C. in Figure 2.7).

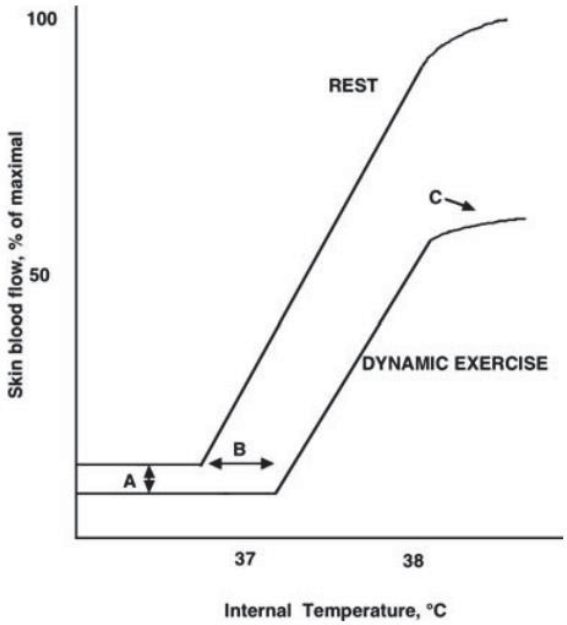

Figure 2.7. Skin blood flow at rest and during dynamic exercise (González-Alonso et al., 2008). A = vasoconstrictor response at the start of exercise; B = Increased threshold core temperature for the onset of vasodilation with exercise; C = Plateau of skin blood flow during exercise, at levels below maximum (core temperature ~38°C).

The delayed response of the skin to increase blood flow observed at the onset of exercise is due to conflicting demands between the cutaneous vasoconstrictor and vasodilator influences. The immediate cutaneous vasoconstriction that occurs has been shown to be mediated by increased active vasoconstrictor tone and subsequently delaying the active vasodilator response (Kellogg *et al.*, 1991; Wendt *et al.*, 2007). The reduced upper limit on the degree of cutaneous vasodilation that can be achieved during exercise is due to the restricted activity of the active vasodilator system; a consequence of the conflicting demands of cardiac output distribution (González-Alonso *et al.*, 2008). The muscle tends to take priority over demands for blood flow during dynamic exercise, so during higher intensity activities, SkBF will be restricted and internal body temperature will suffer as a result. Therefore, it is likely that an increased T_c and the consequential hyperthermia that may ensue will be a factor in fatigue, rather than restricted supply of metabolites to the muscle (González-Alonso *et al.*, 2008).

The importance of blood flow to the skin during exercise is essential for the subsequent heat dissipation that occurs, primarily via evaporation of sweat from the skin surface. Sweating is initiated in response to combined contributions from $\overline{T}_{sk}$ and T_c, and the rate of sweating will depend upon a variety of factors such as physical fitness, and environmental conditions. (Wilke *et al.*, 2007). When exercising at a moderate intensity in cooler environments, sweating and the heat loss accompanied by the evaporation of sweat from the skin would be sufficient to establish thermal balance within the body. But, as the intensity of exercise increases, or there are changes in environmental conditions, thermoregulation and maintaining thermal balance becomes a lot more challenging.

2.3.2 Exogenous Heat Stress

The addition of heat stress to an exercising individual can pose large challenges to the maintenance of thermoregulation and can, in some instances, be potentially dangerous to the individual's health and well-being. Exercising in a hot environment challenges the body through restricting heat dissipation and causing a variety of physiological responses that will be discussed overleaf. These changes that occur during exercise in a hot environment can impact greatly on performance, therefore athletes should look to strategies to minimise these performance deficits if exercise in this type of environment is unavoidable.

2.3.2.1 Physiological Responses

As the ambient tempeature rises, the body is put under heat stress and is less likely to be able to develop a thermal steady-state, especially during exercise. This is characteristic of an 'uncompensable' environment and the continuation of exercise will inevitably lead to an increase in T_c. Figure 2.8 illustrates the contribution of convection, radiation and evaporation to total heat dissipation from the body over a wide-range of ambient temperatures. As ambient temperature increases, the contribution from convection and radiation to heat loss rapidly decreases to the point where evaporative heat loss (via increased sweating) is the only means of heat removal from the body. At these temperatures, convection and radiation can acutally become mechanisms of heat gain, as the skin-to-air temperature gradient required for heat loss has been abolished and instead heat is transferred down the temperature gradient to the body (Powers & Howley, 2009). The increase in evaporative heat loss at these temperatures can compensate for this change at low exercise intensities, but as the intensity of exercise increases, metabolic heat production will be too high and heat removal will not be sufficient to maintain a thermal steady state, leading to an increase in T_c. This concept is illustrated in Figure 2.9, where Armstrong (2000) shows that when exercise intensity increases, in combination with high air temperatures, the less the liklihood is of being able to maintain a steady-state T_c, which disrupts thermal balance.

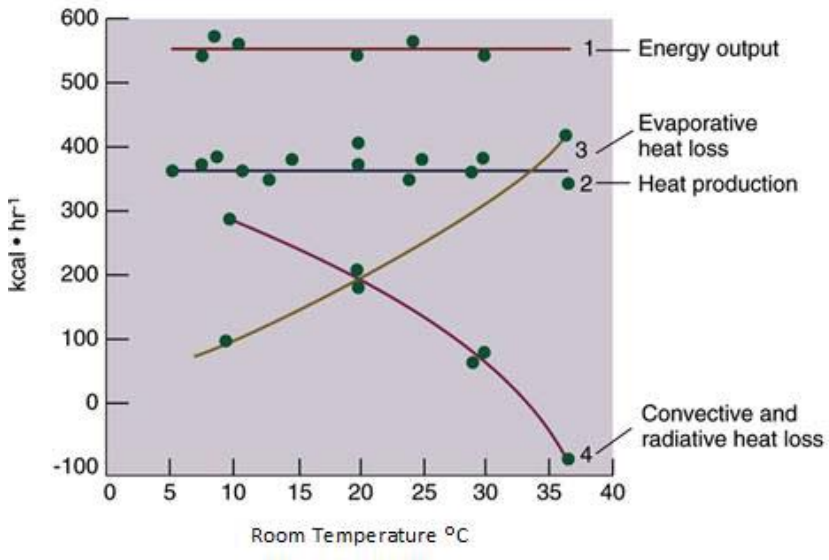

Figure 2.8. Heat production and contributions of evaporation, convection, and radiation to heat loss during exercise at a range of environmental temperatures (Powers & Howley, 2009).

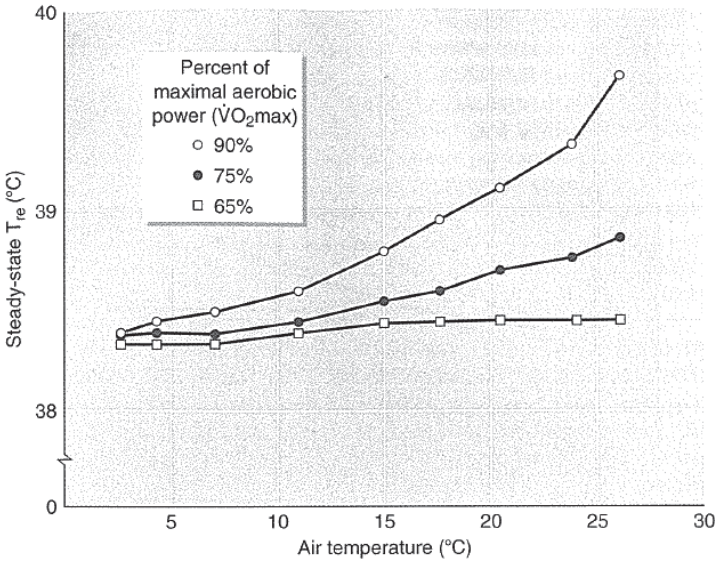

Figure 2.9. Core temperature over a range of differing air temperatures at three different exercise intensities (Armstrong, 2000).

For evaporative heat loss to increase during times of heat stress, blood flow to the skin must be elevated. As described in section 2.3, exercise in itself can cause problems associated with blood flow distribution and competition between the skin and the muscle for contribution of the cardiac output. This problem is exacerbated in hot environmental conditions, where a greater amount of blood flow is required to the skin for heat dissipation, whilst the active muscles still have a high demand for blood flow for oxygen (O_2) delivery (González-Alonso *et al.*, 2008). Reduced blood flow to either of these areas of the body would inevitably lead to performance decrements during exercise; reduced blood flow to the muscles limiting the duration and/or intensity of the exercise, and reduced blood flow to the skin limiting heat disposal, with potential hyperthermia developing over time (González-Alonso *et al.*, 2008). The body has limited capacity to alleviate this problem through the vasomotor reflexes redistributing blood flow from inactive tissue to the active muscles; but this will only lessen a small part of the competition issue (Wendt *et al.*, 2007). The idea of an increase in cardiac output is another method that has been theorised to relieve the blood flow competition issues during exercise in the heat (Rowell *et al.*, 1966). Heart rate is markedly higher during exercise in a hot environment compared to the same intensity of exercise in cooler climates and therefore could contribute to increasing cardiac output (Galloway & Maughan, 1997). Whilst this might be the case in the early stages of exercise in the heat, unfortunately stroke volume is restricted with reduced central blood volume and hyperthermia in hot environments, where cardiac output cannot be increased; instead cardiac output can be severely decreased by hot environmental conditions. Therefore, the higher the intensity of exercise, the greater the decrement (Williams *et al.*, 1962; Rowell *et al.*, 1966). These changes are illustrated in Figure 2.10, where the addition of heat stress to exercise inevitably leads to an increase in heart rate as well as other features of cardiovascular strain.

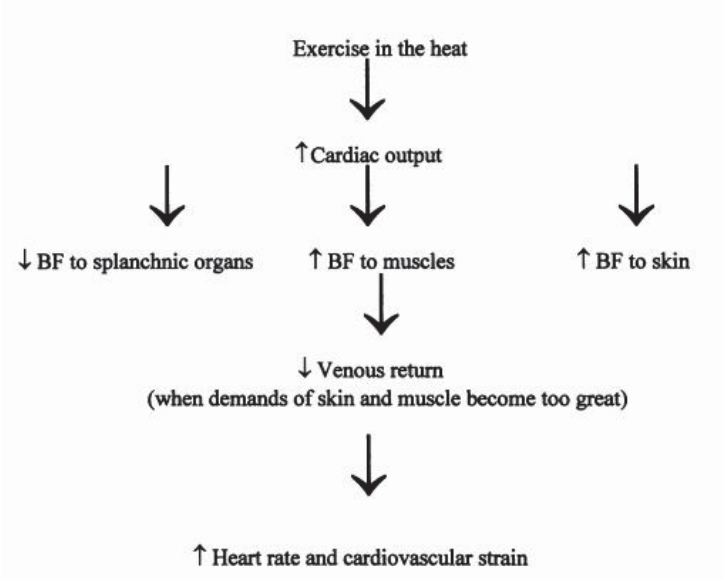

Figure 2.10. Physiological changes during exercise in the heat (Casa, 1999).

It appears that there is no system in place to completely alleviate the competition for blood flow between the skin and the active muscles during exercise in high ambient temperatures. According to González-Alonso *et al.* (2008) skeletal muscle commands a significant portion when competing for cardiac output. This is not entirely surprising considering the limit placed on SkBF during exercise compared to rest (Figure 2.7). The same initial cutaneous vasoconstriction and increased threshold for vasodilation is observed during exercise in the heat, illustrating skeletal muscle's dominance over the blood flow distribution (Kellogg Jr *et al.*, 1991). Although active muscle outweighs the skin for cardiac output distribution, the skin's requirements for blood flow during exercise in the heat are still higher than exercise in cooler ambient temperatures. In such cases it could be that muscle may in fact also fall victim to inadequate blood flow, which may play a part in impaired performance often observed in high ambient temperatures. There have been varying results in studies that have investigated muscle metabolism and blood flow in the heat. Fink *et al.* (1975) compared cycling exercise in the heat (41°C) and cold (9°C) to determine differences in muscle metabolism between the two environmental conditions. They found that in the hot environment, participants had a higher amount of glycogen breakdown than in the cool. From this, they proposed that blood flow to active muscles was reduced in the heat, limiting glucose delivery, therefore leading to greater glycogen breakdown for a fuel source (Fink *et al.*, 1975). Alternatively, Savard *et al.* (1988) and Nielsen *et al.* (1990) both designed studies investigating muscle blood flow during heat stress and both came to the conclusion that blood distribution to the working muscles was not reduced during exercise in hot environments. Savard *et al.* (1988) was the first study to measure muscle blood flow

directly via a thermodilution technique, whereas past studies had been using alternate methods to estimate muscle blood flow. Nielsen *et al.* (1990) found that there was no difference in O_2 uptake in the legs during treadmill walking in both hot (40°C) and moderate (18-20°C) environments. As opposed to Fink *et al.* (1975), they found no increase in glycogen utilisation in the gastrocnemius in the hot environment, as well as no increase in release of lactate nor glucose or free fatty acid uptake in the exercising legs (Nielsen *et al.*, 1990). Whilst there are conflicting results between these three studies, the differences in methodologies need to be considered. The studies by Nielsen *et al.* (1990) and Savard *et al.* (1988) involved exercise that was performed at 60-70% and 50-60% of $\dot{V}O_2$max, respectively; whereas the cycle exercise in the study by Fink *et al.* (1975) was completed at 70-85% of $\dot{V}O_2$max, which is considerably higher. More importantly, all three studies employed alternative modalities of exercise (walking, one-legged knee extensions, and cycling); the extent of muscle mass being utilised will affect whole body $\dot{V}O_2$. Therefore, it makes it difficult to directly compare the three studies, and it may be that muscle blood flow begins to be restricted as exercise intensity as the corresponding metabolic heat production increases. Fortunately, a more recent study has been completed measuring haemodynamics during cycling exercise to exhaustion at differing levels of heat stress/strain. González-Alonso and Calbet (2003) concluded through their research that in severe heat, decrements to muscle blood flow occur, as a result of reductions in cardiac output and mean arterial pressure. The reduction in blood flow to the working muscles hinders the amount of O_2 delivery, in turn reducing VO_2max in such conditions (González-Alonso & Calbet, 2003).

Whilst the muscle and skin are often the two areas of the body that are commonly discussed in regards to distribution of blood flow during exercise in the heat, one very important organ (the brain), cannot be overlooked, and could potentially play its own part in reduced performance/fatigue in the heat. Exercise, especially prolonged exercise, in the heat has been shown to impact both brain blood flow and brain temperature (Nielsen & Nybo, 2003). Nybo and Nielsen (2001b) found that during exercise in the heat, middle cerebral artery mean blood velocity (MCA V_{mean}) was reduced, indicating reduced blood supply to the brain in this condition. In another study, Nybo *et al.* (2002) found that as for MCA V_{mean}, global cerebral blood flow (CBF) was also reduced during the hot trial compared to the control, and that whole-brain blood flow was 18% lower at the end of exercise in the heat. These observed reductions in CBF and MCA V_{mean} have been proposed to be a result of a reduction in the arterial CO_2 tension, induced by hyperthermia during exercise in the heat (Nielsen & Nybo, 2003). Hyperthermia leads to hyperventilation not only during exercise but also at rest, and it is the change in arterial CO_2 tension that occurs as a result, that has been proposed to lead to the reduction in CBF. Although reduced CBF and MCA V_{mean} may be detrimental when exercising in the heat, the actual consequences of these reductions are not entirely known.

Even though there is apparent reduced blood flow to the brain, the metabolic consumption of O_2 is not reduced, indicating that O_2 delivery does not appear to be affected by the proposed reduction in blood flow (Nielsen & Nybo, 2003). CBF also acts as a means of heat removal from the brain, both at rest and during exercise and is especially important during exercise when T_c rises. As a result of compromised CBF during heat stress, heat removal from the brain via the jugular venous blood was reduced (Nielsen & Nybo, 2003). But, Nielsen and Nybo (2003) conclude that even if CBF was not impaired during exercise in the heat, the temperature gradient between the arterial blood and the cerebral tissue would be narrowed, which in turn would reduce the capacity for the arterial blood to remove the same amount of heat from the cerebral tissue. Therefore, it seems as if during exercise in the heat and times of hyperthermia, some degree of cerebral heat storage is inevitable. It may be this increase in brain temperature that plays a part in a concept called 'central fatigue' and reduced nerve activity leading to earlier fatigue during exercise in the heat (Nielsen & Nybo, 2003).

With the competition for blood flow and the reduced capacity of the body to dissipate heat, comes an increase in T_c during exercise in hot environmental conditions. The increase in T_c is dependent upon factors described above, but if the rise in internal temperature is considerable (often observed during high intensity, prolonged exercise in the heat) it can be potentially dangerous to the individual through the development of heat illness. There are different types of heat illness including heat exhaustion, exertional heat cramps, and the most severe, exertional heat stroke (EHS). Heat exhaustion is the most common form of heat illness and involves the inability to continue exercise and may or may not be associated with collapse (Coris *et al.*, 2004). Dehydration and a high body mass index (BMI) are major risk factors, so appropriate hydration in strenuous events and/or hot-humid environments is highly recommended (Armstrong *et al.*, 2007). Upon heat exhaustion, the individual will appear sweaty, pale and ashen and may be experiencing other symptoms which include headaches, weakness, dizziness, chills, nausea, vomiting, diarrhoea, irritability and decreased muscle co-ordination (Armstrong *et al.*, 2007). Heat exhaustion is likely to occur due to both peripheral and central factors, with exertional hyperthermia often being a predetermining element (exertional hyperthermia = T_c >40°C) (Armstrong *et al.*, 2007). The link between both the central and peripheral mechanisms suggest that heat exhaustion acts as a safety mechanism that helps to prevent additional exercise being undertaken in potentially dangerous conditions that could lead to the progression to EHS.

EHS is defined as 'hyperthermia associated with central nervous system disturbances and multiple organ system failure' (Armstrong *et al*., 2007, p. 558) Essentially, the lack of heat removal from the body leads to the rise of T_c to temperatures that are high enough to disrupt organ structure and

functioning. Usually heat exhaustion will occur prior to this point to help prevent the development of EHS, but some individuals are able to "override" the central and peripheral messages to cease exercise and, therefore, push themselves past that point, potentially resulting in EHS (Armstrong *et al.*, 2007). The major risk factors for the development of EHS are the lack of heat acclimation and low fitness levels (Armstrong *et al.*, 2007). EHS can result in death, and survival is largely dependent on immediate recognition of the condition. The symptoms of EHS are specific to the progression of the condition, but can include: disorientation, confusion, dizziness, irrational/unusual behaviour, headaches, inability to walk, loss of balance and muscle function, collapse, hyperventilation, vomiting, diarrhoea, delirium, seizure and coma (Armstrong *et al.*, 2007).

Exertional heat cramps are painful muscle spasms that can occur exclusively or in combination with other heat illness conditions. Exertional heat cramps often occur during periods of prolonged exercise where there is a great deal of body water loss through sweating. It has been proposed that the sodium (Na^+) that is lost in combination with the high sweat rate is replaced with a hypertonic fluid and it is this replacement that is the primary cause of the cramps (Armstrong *et al.*, 2007).

It is clear that exercise in the heat can cause dramatic physiological changes within the body and put it under a great deal of extra stress. Therefore, individuals need to be aware of the changes occurring in their body if exercising (especially at high intensities) in hot environmental conditions and to recognise possible symptoms of heat illness in order to prevent situations that could lead to potential harm.

2.3.2.2 Effect on Performance

Exercise performance in the heat has been researched extensively, with the general consensus that aerobic performance is impaired in hot environmental conditions (Galloway & Maughan, 1997; Martin & Buoncristiani, 1999; Parkin *et al.*, 1999; Tucker *et al.*, 2004; Ely *et al.*, 2007). The metabolic heat production by the athlete during exercise must be very closely matched by heat dissipation from the body if the individual is hoping to perform to the best of their ability (Casa, 1999). Unfortunately, in the heat this becomes nearly impossible, and this type of environment is classified as 'unconpensable'; indicative of the lack of ability to maintain a thermal steady state (Cheung *et al.*, 2000). When exercising in an uncompensable environment, thermoregulation is compromised and the consequential rise in T_c has been previously reported as either the direct cause of, or associated with, decrements in exercise performance (González-Alonso *et al.*, 1999). It was originally believed that exercise capacity was limited in hot environmental conditions as a result of cardiovascular

constraints and the negative associations of a decreased stroke volume due to a large redistribution of blood to the periphery, in combination with reduced blood volume as a consequence of large sweat losses (Rowell *et al.*, 1966; Sawka *et al.*, 1985; Galloway & Maughan, 1997; González-Alonso *et al.*, 1999). Over time, differing theories have been proposed, with a combination of two main strategies being used to assess the impact of heat stress on performance capacity in the heat. Various laboratory studies have employed fixed-intensity (FI) exercise in the heat until voluntary exhaustion to assess performance capacity. When working at a FI in the heat, there is no characteristic plateau of T_c, as heat balance is unable to be established (illustrated by A. in Figure 2.11; Schlader *et al.,* 2010) . The use of this methodology led to the focus on a potential 'critical core temperature' – a T_c upon when reached exhaustion will ensue (MacDougall *et al.*, 1974; Nielsen *et al.*, 1993; Galloway & Maughan, 1997; Cheung & McLellan, 1998; Parkin *et al.*, 1999; González-Alonso *et al.*, 2008). The second strategy used was self-paced (SP) exercise in the heat, which involves completing a set distance/work with the ability to alter the intensity they were working at. Studies employing this strategy found that performance was decremented through the voluntary reduction in exercise intensity by the participant (Tatterson *et al.*, 2000; Marino, 2004; Tucker *et al.*, 2004; Ely *et al.*, 2010; Schlader *et al.*, 2010; Schlader *et al.*, 2011a; Schlader *et al.*, 2011b). This type of exercise ensures that participants never reached this proposed 'critical core temperature', as illustrated by B. in Figure 2.11; instead reducing their exercise intensity (and therefore metabolic heat production) prior to this point. The observation of thermoregulatory responses during SP exercise (in combination with investigation of other mammal's temperature regulation) enabled the development of an 'anticipatory regulation' theory by Marino (2004), whereby the body is proposed to be a complex system of feedforward and feedback interactions that ultimately leads to the protection of T_c.

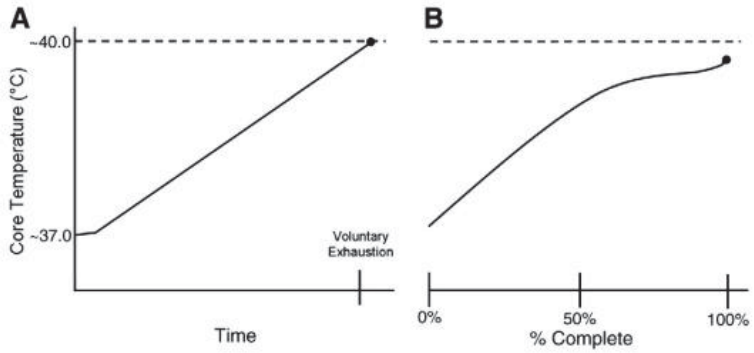

Figure 2.11. Core temperature responses during fixed-intensity exercise (A), and self-paced exercise (B) in a hot environment (Schlader *et al.*, 2010).

Both of these theories suggest that there is a mechanism within the human body protecting itself against the development of fatally high core temperatures, with advantages and disadvantages

associated with each theory. The applicability of the theories that have been developed from the laboratory setting must be addressed, as core temperatures higher than the proposed 'critical core temperature' have been observed in field studies, during prolonged, high intensity events (i.e. a marathon) (Robinson, 1963; Maron *et al.*, 1977; Ely *et al.*, 2009).

2.3.2.2.1 Fixed-Intensity Exercise

FI exercise involves working at a set intensity until exhaustion, and this method can be used to compare performance by assessments of time to exhaustion. Nielsen *et al.* (1993) were first to "coin" the potential 'critical core temperature', during a heat acclimation study. Eight participants cycled at 60% of $\dot{V}O_2max$ until exhaustion for 9-12 days in a hot-dry (40°C, 10% RH) environment, where it was observed that exhaustion occurred every day at a T_c of 39.7 ± 0.15°C, with no concurrent reduction in cardiac output at that point. They concluded that it was the high temperature per se, rather than circulatory alterations that was the most important factor in the cessation of exercise and decreasing the performance ability in the heat. Similar findings were produced by various other studies, where FI exercise performance and time to exhaustion was reduced in a hot environment, and exhaustion always occurred around a similar level of T_c (~40.1°C), regardless of starting T_c (Galloway & Maughan, 1997; Parkin *et al.*, 1999; González-Alonso & Calbet, 2003). The general consensus throughout the literature investigating the impact of FI exercise in the heat is that performance is hindered by hot environmental temperatures, and although there has been a proposed 'critical core temperature range' whereby exhaustion occurs, it is the combination of the high T_c and the secondary physiological factors that occur as a result (discussed above) that leads to a reduced exercise capacity (MacDougall *et al.*, 1974; González-Alonso *et al.*, 1999; Cheuvront *et al.*, 2010).

The reduction in performance has been shown to be due not only to a physiological effect, but also a neuromuscular aspect. Nybo and Nielsen (2001a) reported a reduction in maximal voluntary contraction force in knee extension and hand grip exercises after hyperthermia was induced via cycling in a hot environment, as opposed to after cycling in a cool environment. A passive heating study was used to elicit hyperthermia, that resulted in a reduction in maximal voluntary contraction and central activation of the knee extensor muscles during an isometric maximal voluntary knee extension (Morrison *et al.*, 2004). Therefore, it is probable that voluntary exhaustion occurs around a certain level of T_c and/or muscle temperature as a protective means, where it protects the body against serious damage associated with higher core temperatures.

2.3.2.2.2 Self-Paced Exercise

SP exercise involves the exerciser working at their own speed to complete a set distance or amount of work, and allows investigators to identify the impact of heat stress on performance through the observations of pacing and comparisons of time to complete the work. The individual has the ability to increase the compensability of the environment by reducing their exercise intensity, thereby reducing the amount of metabolic heat production (Schlader *et al.*, 2011a). There is a variety of field and laboratory studies associated with this type of exercise; marathon performance over a range of ambient temperatures being a popular field of investigation. Similar to FI exercise, SP performance has been shown to be reduced in hot environmental conditions; both in field and laboratory settings (Tatterson *et al.*, 2000; Marino, 2004; Tucker *et al.*, 2004; Ely *et al.*, 2007; Ely *et al.*, 2010; Schlader *et al.*, 2011a; Schlader *et al.*, 2011b). The reduction in performance is in association with the individual reducing their metabolic heat production, thereby improving their internal heat balance; observed with the fact that the 'critical core temperature' is almost never reached in SP laboratory studies. Tucker *et al.* (2004) proposed the idea that the reduction in performance during SP exercise in the heat is an anticipatory response to ensure thermoregulation is maintained and exhaustion does not occur. This reinforces the current thinking that the body receives information about the environment and integrates this to produce appropriate alterations to exercise intensity prior to any noticeable physiological responses to the hot environment.

$\overline{T}_{sk}$ (in combination with T_c) has been proposed to play a large role in the development of thermoregulatory behaviours during SP exercise. It acts as a signal receptor, relaying its information through to the CNS where it is integrated and the appropriate responses are elicited (Schlader *et al.*, 2010). The response that is produced during SP exercise is of a behavioural nature, as opposed to FI exercise where changes are more physiological in nature. The behavioural changes and the reduction in exercise intensity during exercise in the heat have been proposed to be both subconsciously (Tucker *et al.*, 2004; Noakes *et al.*, 2005) and consciously (Maughan, 2010; Schlader *et al.*, 2011b) controlled. Therefore, it appears that the human body uses the information relayed through the appropriate receptors to elicit behavioural responses consciously and/or subconsciously to reduce metabolic heat production, thereby thermoregulation is maintained but performance decreases.

The reduction in performance has not only been observed in highly-controlled laboratory studies, but also in outdoor events. Various studies have reported that marathon performance is reduced with increasing ambient temperature and/or wet-bulb globe temperature (WBGT) (Martin &

Buoncristiani, 1999; Ely *et al.*, 2007), as illustrated in Figure 2.12. Whilst laboratory studies are useful when the aim is to control certain factors, they often do not take into consideration some aspects of the environment, such as air flow, solar radiation, nor the impact of competition that mass participation events encourage. Some individuals have the ability to push themselves and their bodies to great lengths to do their best during events, and this has been observed in studies when individuals have finished events with core temperatures of >40°C without any sign of heat illness (Robinson, 1963; Maron *et al.*, 1977). Therefore, careful consideration is required when comparing laboratory-produced results to field-based events.

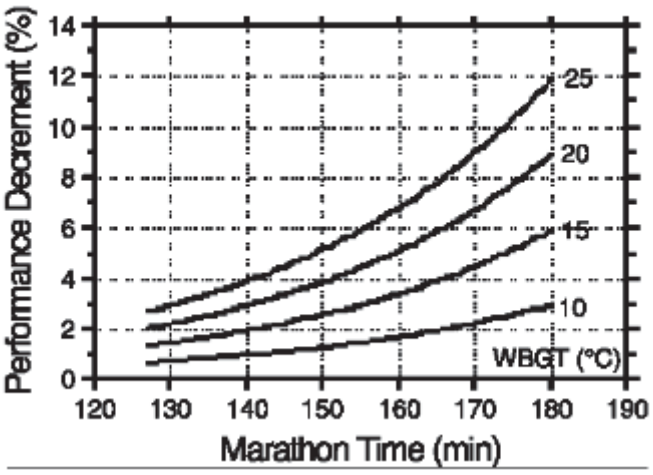

Figure 2.12. Estimated percentage decrement in marathon finishing time with increasing Wet-Bulb Globe Temperature (WBGT) (Ely et al., 2007)**.**

2.3.2.2.3 $\dot{V}O_2max$ Performance

$\dot{V}O_2$max is the maximal O_2 uptake during exercise, and is also often referred to as maximal aerobic capacity (Seeherman *et al.*, 1981). The impact that heat stress has on $\dot{V}O_2$max performance has varied between research studies. Some have stated that performance was reduced (Sawka *et al.*, 1985; Nybo *et al.*, 2001; González-Alonso & Calbet, 2003), while others have noted no change (Williams *et al.*, 1962; Pirnay *et al.*, 1970). The reduction in $\dot{V}O_2$max has been proposed to be associated with a decline in cardiac output and mean arterial pressure, which in turn reduces the blood flow, O_2 delivery and uptake at the working muscles (Sawka *et al.*, 1985; González-Alonso & Calbet, 2003). Due to the fact that a $\dot{V}O_2$max test is often a short bout of exercise, substrate depletion and dehydration can be ruled out as factors associated with the reduction in performance. Different methodologies may explain the discrepancies in the research results. For example, Williams *et al.* (1962) conducted a study with a small sample size (n=3), that were already

acclimatised to severe heat, which may be a reason that no decrement to performance was noted. Further, Pirnay *et al.* (1970) stated that the impact that a hot environment has on $\dot{V}O_2max$ was dependent upon an individual's initial T_c. Various researchers have employed exercise bouts prior to a $\dot{V}O_2max$ test, some of which resulted in significant increases in T_c, to the point of hyperthermia (Craig & Cummings, 1966; Klausen *et al.*, 1967). Pirnay *et al.* (1970) concluded that a high initial T_c (>40°C) will reduce maximal O_2 uptake in subsequent maximal aerobic tests.

The general trend emerging through the literature is that heat stress and hot environmental conditions will decrease performance, more so in prolonged, high-intensity events where other factors such as dehydration can further compound the issue. The majority of the previous literature has tended to focus on cycling as the exercise modality (especially the laboratory studies), but the results produced can be assumed to be transferable to other forms of land-based aerobic exercise (as opposed to water), as it appears to be the intensity and the duration of exercise that is important, irrespective of the modality.

2.3.2.3 Strategies to Minimise Performance Decrements

Various strategies have been used to try and minimise performance decrements in hot environmental conditions, including acclimation to the heat, pre-cooling/cooling and fluid ingestion. Unfortunately, while they might hold valid benefits to performance, not all of these strategies are feasible for the majority of individuals, due to equipment and time restrictions. This being the case, fluid ingestion and the maintenance of hydration status is the cheapest and most convenient method to employ when aiming to minimise performance decrements in the heat.

2.3.2.3.1 Cooling and Pre-Cooling

Considering that T_c has been identified as playing a major role in the reduced performance that has been observed in hot environmental conditions, the idea of cooling the body during, or prior to, exercise has been developed as a method to combat performance decrements.

Pre-cooling involves reducing T_c and/or $\overline{T}_{sk}$ prior to an exercise bout with the proposed mechanism behind the idea being that thermally induced fatigue is delayed due to the increased margin for metabolic heat production before reaching the 'critical core temperature' range. Whilst this appears to be a valid theory, the results produced in the literature have been equivocal; this is due to differences in methods of pre-cooling, modalities of exercise, and also ambient temperature range. Various methods of pre-cooling have been used, including water immersion (Bergh & Ekblom, 1979;

Schmidt & Brück, 1981; Hessemer *et al.*, 1984; Olschewski & Bruck, 1988; Kruk *et al.*, 1991; Lee & Haymes, 1995; Booth *et al.*, 1997; González-Alonso *et al.*, 1999; Kay *et al.*, 1999; Marsh & Sleivert, 1999; Booth *et al.*, 2001), cold air exposure (Schmidt & Brück, 1981; Hessemer *et al.*, 1984; Olschewski & Bruck, 1988; Kruk *et al.*, 1991; Lee & Haymes, 1995), ice/cooling vests (Duffield *et al.*, 2003; Cheung & Robinson, 2004; Hornery *et al.*, 2005; Duffield & Marino, 2007; Ückert & Joch, 2007; Bogerd *et al.*, 2010), cold water consumption (Lee *et al.*, 2008; Ihsan *et al.*, 2010), and cooling packs (Myler *et al.*, 1989; Duffield *et al.*, 2009; Minett *et al.*, 2011).

Whilst pre-cooling is a method that has been employed by a number of researchers and athletes to combat the negative impacts of a hot environment, it is an area of research that is beyond the scope of this thesis. Comprehensive reviews have been made by Marino (2002) and Wegmann *et al.* (2012), concluding that although the results from the previous literature are fairly equivocal, pre-cooling appears to have a positive impact on subsequent performance in warm/hot environmental conditions and endurance/prolonged exercise.

2.3.2.3.2 Hydration

Exercise in the heat results in higher fluid losses through increased sweating, therefore rehydration is particularly important, especially during longer duration endurance exercise. If rehydration is not employed, the progressive dehydration can be associated with physiological changes that may affect performance, such as increased T_c and heart rate, and reduced stroke volume and cardiac output (Hargreaves *et al.*, 1996). A review by Kay and Marino (2000) concluded that dehydration hinders subsequent performance, even more so when performing in the heat. Although, since this review hydration guidelines have been challenged, after Wall *et al.* (2013) developed a method to blind their participants to their hydration status, and did not observe any performance decrements with up to 3% of body mass lost due to fluid losses. The equivocal results from the various studies indicate that the current recommendations for fluid replacement need to be revisited. It is possible that fluid ingestion may hold further benefits for exercise performance in a laboratory setting, but is not as transferrable to 'real-life' sporting situations.

2.3.2.3.3 Heat Acclimation

Repeated exposure to a hot environment has the potential to induce various physiological adaptations within the human body that can reduce the thermoregulatory strain during subsequent exposures, meaning an individual can withstand adverse temperatures for longer. When exercise is combined with repeated heat exposure for an appropriate length of time, the concurrent physiological adaptations that develop are characteristic of a heat acclimated individual, one which

can maintain not only a sedentary state in the heat for longer, but also exercise. This idea of heat acclimation will be discussed in greater detail in the following section.

Heat acclimation is the most effective technique to reduce thermal strain, and if feasible, should be the method employed by athletes in the lead up to an event. Nonetheless, all individuals should aim to maintain an appropriate hydration status before and during exercise in the heat, even if the hydration effect is more psychological, rather than physiological in nature.

2.3.3 Section Summary

Exercise increases heat production in the body by a considerable extent, and when combined with a hot environment, some problems begin to arise as a result of the lack of thermal balance. Exercise in the heat and the concurrent heat storage that occurs has been investigated through both FI and SP performance parameters, concurrently with how much of an effect the heat has on $\dot{V}O_2max$ performance. In general, a hot environment has been associated with observed performance decrements, namely in prolonged/endurance events, yet the impact on $\dot{V}O_2max$ performance is relatively unclear and appears to depend on the level of T_c prior to the maximal exercise bout. The majority of the associated literature in the above section has utilised cycling as the exercise modality, and while it has been assumed that the results are transferable to other forms of aerobic exercise (excluding swimming), additional research into alternative modalities would be beneficial to confirm this. Strategies to deter the performance decrements have been developed, including pre-cooling of the body, hydration status, and heat acclimation. Heat acclimation undoubtedly leads to the greatest protection against subsequent performance decrements in the heat. Whilst heat acclimation involves a more prolonged period of development than pre-cooling and hydration, if an athlete has the resources to induce acclimation prior to an event in a hot environment, it will be the best option in the hope of reducing performance decrements.

2.4 Heat Acclimation

Performing repeated bouts of aerobic exercise in hot environmental conditions creates additional stress on the body, but can also induce certain physiological adaptations that can be beneficial to exercise performance and cardiovascular health. The development of the physiological adaptations reduces the strain of the individual exercising in the heat and illustrates a state of heat adaption. When this course of development occurs over a period of time in a natural environment, it is labelled heat acclimatisation; whereas when artificial modalities are employed (i.e. a climatic chamber) to induce these adaptations, it is termed heat acclimation (Armstrong & Maresh, 1991). Both acclimatisation and acclimation result in very similar physiological adaptations; to avoid confusion for the reader, both methods will be referred to as heat acclimation in this review. The aim of heat acclimation is to reduce the impact that the additional heat stress has on the exercising individual through the physiological adaptations that develop. The physiological adaptations are of a cardiovascular, sudomotor, blood flow, metabolic and cellular nature and the specific adaptations will be explained in more detail in the following section. The consequential reduced strain has the potential to lead to improved subsequent performance in the heat, and these effects may also benefit exercise in cooler environments (i.e. 10-20°C). Unfortunately, to-date the literature associated with the impact that heat acclimation has on performance in cooler environments is very limited, making conclusions on the topic difficult to make. Section 2.4 of the literature review will review the induction of heat acclimation and report the literature associated with the effects heat acclimation can have on performance in warm/hot environments, and also revise the small pool of research related to performance in cooler temperatures.

2.4.1 Experimental Protocols

Heat acclimation protocols in a laboratory setting enable the researchers to control the environmental temperatures and RH. Previous heat acclimation studies have assessed a wide range of environmental conditions, thus making direct comparisons between studies difficult. The period of the heat acclimation protocol can be divided into short-, medium-, and long-term periods. The degree of acclimation that is induced in an exercising individual is often influenced by the protocol factors such as, the length of the protocol; the modality/intensity/duration of exercise; the environmental conditions (i.e. the heat stress employed); and the method used to achieve acclimation. Additionally, not all individuals will react and acclimate in the same way; some will acclimate to the heat quickly (high responders), whilst others will take a lot longer, or may never develop the expected physiological adaptations (low responders) (Taylor & Cotter, 2006). If

repeated heat exposure is not maintained, the advantageous physiological adaptations obtained from heat acclimation will begin to return to pre-acclimation levels with full reversal complete after about three weeks of no exposure (Garrett *et al.*, 2011).

2.4.1.1 Environmental Conditions

Whilst a range of ambient temperatures has been employed by researchers to induce heat acclimation in various participants, the two main environments utilised have been divided into either hot-humid or hot-dry. Both types of environments have the potential to induce heat acclimation, if elevated T_c combined with sweating occur in the exercising individual (the two factors essential in the development of increased heat tolerance) (Wendt *et al.*, 2007). In general, hot-dry environmental conditions will allow participants to work in hotter ambient temperatures compared to a hot-humid environment, where the increased water-vapour in the air significantly reduces the compensability of such an environment (Armstrong, 2000). Exercise in either environment can induce heat acclimation, although each will elicit differences in physiological responses (e.g. higher sweat rate in a hot-humid environment compared to hot-dry). Therefore, it appears that the adaptations obtained from acclimation to one environment are not directly transferable to the other, and will not provide complete protection to exercise in these different environmental conditions (Goldman *et al.*, 1964; Taylor & Cotter, 2006). Both environments have their benefits and limitations associated with heat acclimation. Acclimation to a hot-dry environment tends to result in larger decreases in $\overline{T}_{sk}$, heart rate and T_c, combined with either a smaller increase, or unaffected sweat rate (Eichna *et al.*, 1950; Piwonka & Robinson, 1967; Gisolfi & Robinson, 1969; Shvartz *et al.*, 1973). Acclimation to a hot-humid environment appears to result in a decrease in T_c, large increases in sweat rate, with only minor alterations to heart rate, $\overline{T}_{sk}$ and metabolism (Garden *et al.*, 1966; Weinman *et al.*, 1967; Dawson, 1988). The increased ability to cool the body via evaporative cooling in a hot-dry environment has been proposed to be a favourable aspect of this type of heat acclimation. The consensus from the previous research indicates that acclimation specificity exists, therefore when aiming to improve tolerance in a hot-dry environment, heat acclimation should be carried out in similar conditions; likewise for hot-humid environments (Goldman *et al.*, 1964; Taylor, 2000). Heat acclimation from a hot-dry environment will not provide great protection against hot-humid conditions (and vice versa), nonetheless some adaptations will be gained (Shvartz *et al.*, 1973; Dawson, 1994).

2.4.1.2 Exercise Protocol Duration, Intensity and Modality

There have been a variety of protocol durations employed to induce heat acclimation in exercising individuals, with authors categorising the durations into three main groups: Short (< 7days), medium (8-14 days) and long term (>15 days) acclimation periods (Garrett *et al.*, 2011). The majority of the literature (especially earlier on) has employed medium-long term protocols, with a greater focus on short-term acclimation appearing to become popular in more recent years. Armstrong and Maresh (1991) have suggested that a heat acclimation protocol should be 10-14 days, which is based on the idea that complete acclimation to heat stress is usually achieved by 7-14 days (depending on the individual) (Pandolf, 1998). But, 75% of acclimation can be achieved by about 4-6 days, including an improved cardiovascular capacity which occurs in the first 7 days of the acclimation period (Pandolf, 1998; Garrett *et al.*, 2011). Taking this into consideration, some authors have concluded that short-term acclimation protocols may be the most beneficial, specifically for athletes, as a shorter protocol will lead to less disruption to usual training, be a cheaper option, and will still induce the cardiovascular alterations (Garrett *et al.*, 2009; Garrett *et al.*, 2011). A medium-long term acclimation protocol may lead to full acclimation, but is more time consuming for the individual and can lead to greater levels of dehydration as a result of an increased sweat rate (Garrett *et al.*, 2011). Most heat acclimation studies have employed a protocol consisting of consecutive days exercising and unfortunately there is only limited research assessing the development of acclimation using a non-consecutive day protocol. Gill and Sleivert (2001) found that a consecutive 10 day acclimation protocol resulted in greater acclimation than a non-consecutive day regime, with the authors reporting lower T_c, $\overline{T}_{sk}$, heart rate and RPE with the consecutive-day training. It was hypothesised that the days off between heat exposures caused some of the physiological adaptations to be lost (Gill & Sleivert, 2001). In contrast, Fein *et al.* (1975) reported that consecutive training was not necessary for the development of heat acclimation, thus illustrating that training every 3 days for a 30 day period induced the same level of heat acclimation as a 10 day consecutive protocol.

A variety of intensities and durations of exercise have been employed in the associated previous research, and it has been advised that optimal heat acclimation can only be achieved when interval training or continuous exercise is performed at an exercise intensity greater than 50% $\dot{V}O_2$max (Armstrong & Maresh, 1991; Pandolf, 1998). Lind and Bass (1962) originally suggested that an exercise bout of 100 minutes was the optimal for the development of heat acclimation, but this idea has further been challenged by other authors. Houmard *et al.* (1990) had 9 trained runners complete two 7-day heat acclimation protocols – one consisting of 60 minutes of exercise at 50% $\dot{V}O_2$max, and the other 30-35 minutes at 75% $\dot{V}O_2$max. They found that heat acclimation was established with

both methods of training, and there was no significant difference in adaptations or heat tolerance between the two protocols. Sunderland *et al.* (2008) achieved heat acclimation in their experimental group through four 30-35 minute bouts using the Loughborough Intermittent Shuttle Test, performed on non-consecutive days. The results from these studies show that shorter duration exercise in combination with higher exercise intensities has the same capability to induce heat acclimation compared to long duration and lower intensity exercise bouts. It may be that intermittent/interval exercise is a more powerful stimulus for the development of the adaptations that accompany heat acclimation, as higher intensity intermittent exercise has been proven to provide a greater amount of thermal strain than continuous exercise when working in the heat (Nevill *et al.*, 1995).

To elicit heat acclimation the most popular exercise mode has been cycling (Nielsen *et al.*, 1993; Cotter *et al.*, 1997; Nielsen *et al.*, 1997; Patterson *et al.*, 2004; Garrett *et al.*, 2009; Lorenzo *et al.*, 2010), followed by either walking or running (Sawka *et al.*, 1985; Sunderland *et al.*, 2008) and swimming (Hue *et al.*, 2007). Swimming in a hot environment has the ability to induce heat acclimation as illustrated by Hue *et al.* (2007), but the extent of adaptation has been proposed to be hindered slightly due to the lack of evaporative potential in water, limiting sweat rate and cutaneous blood flow (McMurray & Horvath, 1979). It appears that the intensity of the exercise has greater importance, rather than the specific exercise itself (with the exception of swimming) that will lead to the development of heat acclimation. Ideally, it would make sense that the modality of exercise used should be as specific as possible to the modality of exercise that the participant is competing in, but sometimes this is not always possible/ideal (i.e. some athletes may prefer a lower impact, non-weight-bearing exercise modality if they are training for long hours running). As long as the heat acclimation protocol is an appropriate length (between 5-14 days), and the intensity and duration of exercise (in combination with high ambient temperature) are high/long enough to establish an elevation in T_c and a stimulation of sweating, increased tolerance to the heat should ensue – irrespective of the modality of exercise used.

2.4.1.3 Methods of Acclimation

Since the establishment of the fact that an elevated T_c and stimulation of sweating are the two factors essential for the development of heat acclimation, various ways of achieving this have been employed by the previous research. The use of sweat clothing (i.e. tracksuits, waterproof clothing, and vapour barrier suits) has been examined by various studies. Sweat clothing creates a humid-heat microclimate, but the potential for this method to improve heat tolerance remains ambiguous due to the inconclusive results (Dawson, 1994). Allan and Haisman (1964) and Crowdy and Haisman

(1965) both found that exercising in a sweat suit produced physiological adaptations associated with heat acclimation and increased heat tolerance, but these changes were not significantly different to those achieved by their control groups exercising without the sweat suit. Marcus (1972) and Gisolfi *et al.* (1977) both conducted studies where they concluded that exercise in sweat clothing significantly improved heat tolerance through increased sweat rates and reductions in T_c, but neither studies used control groups, therefore the adaptations could not be put down to primarily the use of the sweat suits, over and above the physical activity itself. A study by Dawson and Pyke (1990) investigated the effects of wearing sweat clothing during intermittent exercise throughout hockey trainings. They concluded that the use of a sweat suit over the training itself only produced a small (and non-significant) benefit. The equivocal results from the previous literature illustrate that while sweat suits may be a cheaper and more practical method of heat acclimation, they may not be the optimal technique. But, in cases where athletes have no other choice leading up to an event in a hot environment, training in sweat clothing is more favourable than attempting no form of artificial acclimation (Dawson, 1994).

The use of a climatic chamber has been the method of choice for inducing heat acclimation in the previous laboratory research, because of its positive results. While using a climatic chamber appears to be the best method to induce artificial acclimation in an individual, it is a more expensive option, with reduced practicality for a lot of sports due to the limited chamber space. Nonetheless, there is vast research associated with methods of acclimation using this apparatus. Passive heating has also been employed to sedentary individuals through the use of a climatic chamber to cause an increase in T_c. While this method is much less effective than those that incorporate exercise, a small degree of heat acclimation can result from just passively heating an individual for a period of time (Taylor, 2000; Taylor & Cotter, 2006). Performing exercise bouts in the heat is the quickest and most effective way to induce heat acclimation in an individual, and there have been three main methods applied to achieve this: i) constant work-rate methods; ii) SP exercise; and iii) controlled-hyperthermia. The constant work-rate method was developed from work with the military as a means of evaluating performance, and involves the participant working at the same set intensity each day over the course of the acclimation protocol (Greenleaf, 1970). Regardless of its popularity, the constant work-rate method has some limitations: the absolute intensity will result in a variety of physiological strain across different participants; also because the intensity is kept constant across the entire heat acclimation protocol, the thermal strain will decline as heat acclimation begins to develop (Taylor & Cotter, 2006; Garrett *et al.*, 2011). This is illustrated in Figure 2.13 (A), where by day 12 the T_c response is a lot lower than day 1. The SP exercise method has participants completing a certain distance or amount of work in their own time each day for the heat acclimation protocol.

This technique allows the participant to adjust their pace according to their thermal and physiological strain and poses greater practical relevance to actual race events, but it is difficult to standardise (Taylor & Cotter, 2006; Garrett *et al.*, 2011). The third and final method is controlled-hyperthermia, which uses a process whereby the participant performs exercise which increases the core body temperature to a certain point at which it is 'clamped', above the sweating threshold (Taylor & Cotter, 2006). This method means that T_c is maintained at this same threshold across all days of the heat acclimation protocol, therefore imposing the same amount of strain on the exercising individual every day (Taylor, 2000). Figure 2.13 (B) illustrates this method of heat acclimation, showing that on day 12 of the protocol, the thermal strain is the same as it was on day 1 (indicated by the same degree of T_c rise). This equal strain has led authors to conclude that the controlled-hyperthermia method may be the optimal strategy to induce heat acclimation, and is the least dangerous (Taylor, 2000). But, as with the others, the controlled-hyperthermia method has some limitations, such that the clamped T_c is targeting the thermal strain of the individual and not their athletic performance. Because intermittent exercise is the type that is most often associated with this process, it is one that is not directly transferable to most competition events, therefore is deemed to be a training technique (Taylor & Cotter, 2006). All three methods have their benefits and limitations, but all have the potential to induce heat acclimation when combined with a hot environment over a period of time. The choice of what technique to use is dependent on what the researcher/athlete's goals are; hence they can choose the most applicable method to producing the desired outcome.

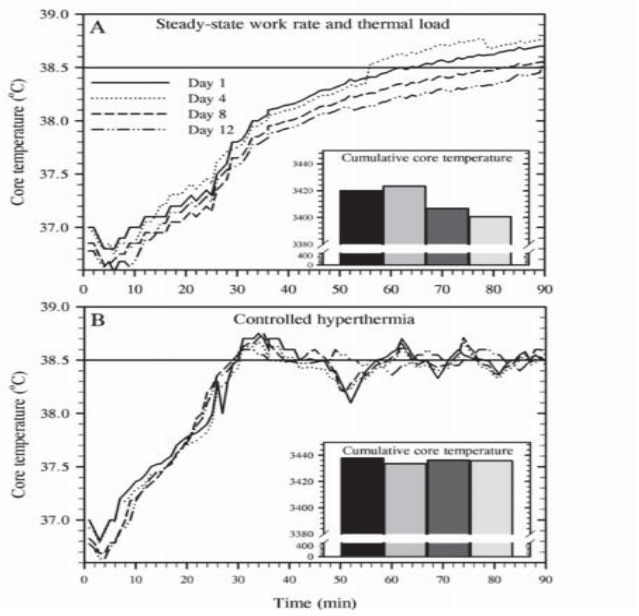

Figure 2.13. Core temperature responses on days 1, 4, 8, and 12 of a 12-day constant work-rate heat acclimation regime (A); or a controlled-hyperthermia heat acclimation regime (B) (Taylor & Cotter, 2006)

2.4.2 Physiological Adaptations to Heat Acclimation

Repeated bouts of exercise in the heat have the potential to induce certain physiological adaptations in the body, which reduces the thermoregulatory strain, improving an individual's ability to maintain a higher intensity of exercise in the same environmental conditions. The physiological adaptations range from cardiovascular, haematological, sudomotor, cellular, and metabolic backgrounds; the time taken to develop is highlighted in Figure 2.14. It generally takes between 7 and 14 days for full heat acclimation to be induced, depending on the individual, their responsiveness, and the strength/type of the heat stimulus.

Adaptation	Days of heat acclimatisation													
	1	2	3	4	5	6	7	8	9	10	11	12	13	14
Heart rate decrease			———	———	———	———								
Plasma volume expansion			———	———	———	———								
Rectal temperature decrease					———	———	———	———						
Perceived exertion decrease			———	———	———	———								
Sweat Na^+ and Cl^- concentration decrease[a]					———	———	———	———	———	———				
Sweat rate increase								———	———	———	———	———	———	———
Renal Na^+ and Cl^- concentration decrease			———	———	———	———	———	———						

a While consuming a low NaCl diet.

Figure 2.14. Amount of time to develop the physiological adaptations associated with the development of heat acclimation (Armstrong & Maresh, 1991).

The physiological adaptations that are characteristic of heat acclimation are not exclusively limited to/associated with training in a hot environment. In fact, some of the adaptations can be achieved via just exercise itself. To illustrate this, Table 2.1 shows the physiological responses that occur after a 14-day training program in a cool-dry, versus a hot-dry environment. The exercise itself has the potential to elicit adaptations that will improve cardiovascular stability, thereby improving body temperature regulation (Taylor & Cotter, 2006). The 'exercise-acclimated' individual is deemed as being in a state of partial-acclimation; exercise, in combination with a hot environment, to elicit a T_c rise and sweating is essential to develop full heat acclimation. Individuals who begin a heat acclimation program with a high basal aerobic fitness tend to adapt at a faster rate than those who begin at lower aerobic fitness levels (Pandolf *et al.*, 1977).

Table 2.1. The physiological adaptations obtained during passive heating, exercise in a hot environment, and exercise in a cool environment (Armstrong & Maresh, 1991).

Physiological responses	Heat acclimatisation without exercise (passive)	Strenuous exercise protocols	
		cool-dry environment	hot-dry environment
Lower core temperature at the onset of sweating	++	+	++
Increased heat loss via radiation & convection (skin blood flow)	++	++	++
Increased plasma volume	+	+	++
Decreased heart rate	0	++	++
Decreased core body temperature	++	+	++
Decreased skin temperature	+	+	+
Altered metabolic fuel utilisation	0	++	++
Increased sympathetic nervous system outflow (efferent)	+	++	++
Increased $\dot{V}O_{2max}$	0	++	++
Improved exercise economy	0	0	+
Adaptation to exercise in a cool environment	0	++	++
Adaptation to exercise in a hot environment	+	+	++

Symbols: 0 = minimal effect; + = moderate effect; ++ = major effect.

2.4.2.1 Cardiovascular and Haematological

Both cardiovascular and haematological changes are the first adaptations to occur during a heat acclimation protocol, characterised by an expansion of the plasma/blood volume, which is often concurrent with an increased stroke volume, and a reduction in heart rate. Whether the plasma volume (PV) expansion is the primary foundation for the decreased heart rate and increased stroke volume (therefore making them causally related) is still up for debate (Garrett *et al.*, 2009). Exercise alone has the potential to result in PV expansion and cardiovascular alterations. Convertino *et al.* (1980) conducted a study to determine the relative contributions of exercise induced T_c rise, and heat-induced sedentary T_c rise to the PV expansion. Both groups induced the same T_c rise via either exercise or heat, and the results showed that the exercise group had a 12% PV expansion, and the heat group induced an expansion of 4.9%. The authors concluded that 40% of the PV expansion is caused by a rise in the T_c, whilst the remaining 60% is connected to exercise-induced factors. Therefore, a combination of exercise in a hot environment would be expected to induce PV expansion to a greater extent than just heat exposure, or exercise alone.

Barcroft *et al.* (1922) was the first to observe that blood volume tended to increase as a response to repeated exposure to a hot environment, and since then numerous researchers have been investigating the associated mechanisms, concluding that it is the expanded PV that induces this change. The amount of PV expansion that occurs with the development of heat acclimation depends

on a number of different factors: the fitness status of the individual; the time of the day; level of activity (resting or exercising); and hydration status, where the level of PV expansion induced can range between 0 and 30% (Sawka *et al.*, 2000). The mechanism behind the PV expansion was originally hypothesised as the combination of a greater influx of protein from the ISF into the vascular volume, and less protein being taken back into the ISF (Senay *et al.*, 1976). Therefore, if a greater protein content is present inside the vascular volume, osmotic pressure will draw water into this area, at the expense of the ISF (Nielsen *et al.*, 1993). But this proposed mechanism has since been challenged, as Patterson *et al.* (2004) discovered a simultaneous expansion of not only PV and the extracellular fluid (ECF), but also the ISF; indicating that PV expansion may in fact not be occurring at the expense of the ISF. It has since been proposed that electrolyte retention and the ECF expansion may have more of an influence on PV expansion (Wendt *et al.*, 2007), and that the increasing plasma protein content may possibly have more of an effect in the later stages of heat acclimation (Patterson *et al.*, 2004). The dissonance in the mechanism of PV expansion may, in part, be a result of the fact that different methods of inducing heat acclimation were employed by the research groups. Patterson *et al.* (2004) used the controlled-hyperthermia method, which was hypothesised to be associated with a greater expansion of the entire ECF than other methods of heat acclimation (Taylor, 2000).

Whilst PV expansion occurs in the early stages of heat acclimation, it has been thought to gradually reduce towards baseline levels during longer-duration heat acclimation protocols (Senay *et al.*, 1976; Patterson *et al.*, 2004). This proposed reduction may, once again, depend on the method being used to induce heat acclimation, as Patterson *et al.* (2004) illustrated that PV expansion could be maintained for up to 22 days, using the controlled-hyperthermia method. PV expansion increases overall blood volume (hypervolaemia) which has its own benefits, although unfortunately, the PV expansion is not concurrent with increases in haemoglobin or red blood cell volume (Fortney & Senay, 1979; Convertino *et al.*, 1980; Harrison, 1985). Various studies have linked the PV expansion and concurrent hypervolaemia with improved cardiovascular functioning during exercise in the heat, in the form of increased stroke volume, and in turn, reduced heart rate (Wyndham *et al.*, 1968; Senay *et al.*, 1976; Sawka *et al.*, 1983a; Nielsen *et al.*, 1993). A reduced heart rate provides great benefits to exercise performance; the cardiovascular system is more efficient and able to meet the thermoregulatory, and circulatory demands of the body during exercise in the heat with fewer beats (Chalmers *et al.*, 2014). Although decreased heart rate and increased stroke volume have been observed in a number of studies, in association with PV expansion, they have not always all been observed together. For example, Nielsen *et al.* (1997) noted a PV expansion and decrease in heart rate, but no increase in stroke volume with heat acclimation to a hot-humid environment. The lack

of interaction between all three factors (PV, stroke volume and heart rate) in various studies has led to their association being challenged - with authors suggesting that there may be some centrally mediated changes with heat acclimation, leading to various cardiovascular alterations (Levi *et al.*, 1993; Garrett *et al.*, 2009; Chalmers *et al.*, 2014). Unfortunately, the proposed centrally mediated changes have not been investigated on humans; instead animal models have been used. Horowitz *et al.* (1986) and Levi *et al.* (1993) worked with rats to discover that heat acclimation improved central mechanisms, characterised by enhanced cardiac mechanics of the heart (compliance, and energy sparing in the form of preservation of high-energy phosphorous compounds), which in turn may be associated with a heat acclimation induced reduction in heart rate.

It seems plausible that more than one mechanism is responsible for heat acclimation induced changes in cardiovascular aspects; PV expansion, in combination with enhanced venous tone, establishes an increased stroke volume, alongside improved cardiac mechanics, leading to an overall more efficient cardiovascular system. Regardless of whether the two are directly linked, the cardiovascular and haematological adaptations reduce not only the cardiovascular strain, but also the thermoregulatory strain of exercising in the heat by permitting an increased blood flow from the core to the periphery, allowing a greater dissipation of heat from the body (Taylor, 2000). These changes are not only beneficial during exercise in the heat, but the adaptations are likely to be transferable to exercise in a cooler environment, having the potential to reduce strain and improve exercise performance. Increased stroke volume, and improved cardiac mechanics is indicative of improved cardiovascular capacity and would therefore be expected to benefit an individual regardless of whether exercising or sedentary. Therefore, it would be expected that resting heart rate would be lower, and there would be improved regulation of blood pressure, where both factors may be associated with overall improved aerobic fitness and cardiovascular health (Taylor & Cotter, 2006).

2.4.2.2 Sudomotor

Physical exercise and heat acclimation have the potential to improve the sudomotor functioning in the body, characterised by elevated sweat rate (Fox *et al.*, 1964; Shvartz *et al.*, 1979; Candas *et al.*, 1983), reduced T_c threshold for sweating (Nadel *et al.*, 1974; Shvartz *et al.*, 1979; Nielsen *et al.*, 1993), and an increased sweat sensitivity to T_c changes (Henane *et al.*, 1977; Libert *et al.*, 1983). Whilst both exercise and heat acclimation improve the function of sweating during exercise, heat exposure in combination with exercise holds the greatest potential for improvement. Figure 2.15 gives an overview of the impact of both physical activity and heat acclimation on sweat rate and the central drive required to initiate sweating. A) illustrates an un-trained, unacclimated individual; B)

illustrates a trained, aerobically fit individual that now has an enhanced sweating mechanism through peripheral effects, in that there is an increased sensitivity to the central sweating drive; finally, C) demonstrates a trained, and acclimated individual who not only has developed the peripheral adaptations, but there has also been a reduction in the point of central sweating drive (Nadel *et al.*, 1974). Heat exposure in combination with exercise has the potential to induce the greatest improvement in sudomotor adaptations than either aspect alone. An individual who is trained and heat acclimated has the ability to dissipate heat and withstand a hot environment to a greater extent.

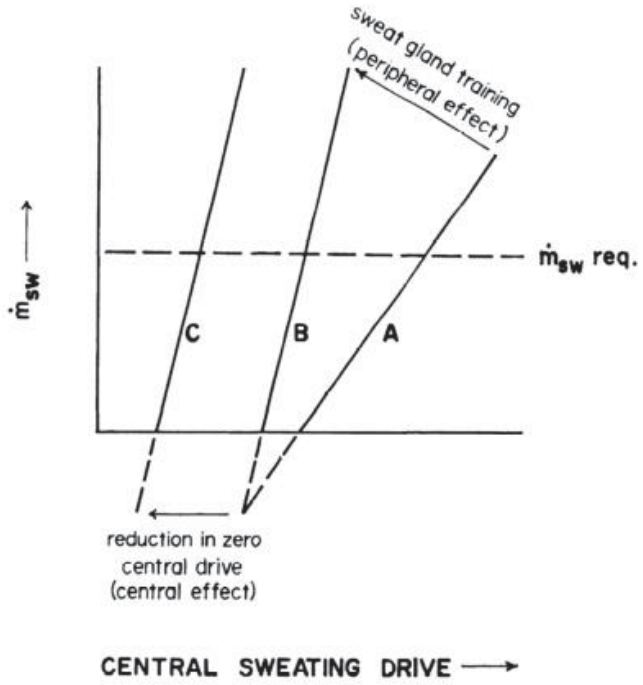

Figure 2.15. Changes to sweat rate as a result of physical activity and heat acclimation (Nadel *et al.*, 1974).

Sweating adaptations are present from the second day of a heat acclimation protocol, but generally take between 7-14 days to fully develop (Corbett *et al.*, 2014). The extent and nature of sudomotor adaptations depend on the type of environment that the individual is being heat acclimated to (i.e. hot-humid, or hot-dry). Exercise in a hot-humid environment tends to lead to progressively increasing sweat rates throughout the exercise bout, whereas exercise in a hot-dry environment does not tend to have this characteristic increase in sweat rate (Armstrong & Maresh, 1991). Sweat rate can increase up to two-fold following a heat acclimation protocol (Taylor, 2000); a result of both peripheral and central factors (as highlighted in Figure 2.15); depending on the method of acclimation employed, an appropriate protocol can improve an individual's evaporative potential by

up to 11% (highest with the controlled-hyperthermia methodology) (Poirier *et al.*, 2014). Alongside the increased sweat sensitivity and earlier onset of sweating for a given T_c, heat acclimation can also increase the size of the sweat gland, and sweat gland secretion (Peter & Wyndham, 1966; Sato & Sato, 1983; Sato *et al.*, 1990), but will not increase the number of sweat glands active on the body (Sargent II *et al.*, 1965; Peter & Wyndham, 1966; Inoue *et al.*, 1999). Not only is the rate of sweat production increased after heat acclimation by the above adaptations, but the sweat being produced is also more dilute – due to the sweat gland ducts having a greater potential for reabsorbing sodium and chloride from the primary sweat (Allan *et al.*, 1971; Taylor, 2000). Whilst an increase in sweat rate is a beneficial adaptation to help improve thermoregulation when exercising in the heat, this will only benefit individuals who are exercising in an environment that allows a greater extent of evaporative heat loss – i.e. when exercising in a hot-humid environment, or in sweat clothing, an increased sweat rate will hold little benefit to the exercising individual, and will in fact lead to wastage of body water due to the lack of evaporative potential of the environment (Nielsen *et al.*, 1997).

Although the majority of sudomotor adaptations tend not to develop until after 7 days of heat exposure, various authors have investigated the impact of short-term heat acclimation protocols on sweating rates and alterations (Chen & Elizondo, 1974; Cotter *et al.*, 1997; Chalmers *et al.*, 2014). These studies show that some adaptations and benefits to sweat rate are achieved within the 7 days of a short-term heat acclimation protocol; with Cotter *et al.* (1997) stating that their short-term heat acclimation induced only central alterations to sudomotor capacity by reducing the sweating threshold, but did not have any effect on peripheral alterations via changing sweat sensitivity or sweat rate. Chalmers *et al.* (2014) noted similar findings; the onset of sweating was established at a lower level of T_c (by 0.15-0.29°C), leading to improved evaporative cooling and thermoregulation after the short-term heat acclimation protocol. This suggests that peripheral sudomotor adaptations will occur in the later stages of a longer heat acclimation protocol, establishing complete heat acclimation.

2.4.2.3 Blood Flow

The cardiovascular adaptations that arise with development of heat acclimation also play a role in establishing elevations in blood flow to certain areas (namely the skin). The increased blood flow improves heat dissipation from the body, reducing T_c and the thermoregulatory strain of a hot environment (Wyndham *et al.*, 1968). Whilst blood flow to the skin has been shown to increase, relative to T_c, with both short-term (Yamazaki & Hamasaki, 2003; Fujii *et al.*, 2012), as well as medium- to long-term heat acclimation protocols (Roberts *et al.*, 1977; Takeno *et al.*, 2001), blood

flow to the active muscles appears to be unchanged (Kirwan *et al.*, 1987; Nielsen *et al.*, 1993; Nielsen *et al.*, 1997). The increase in SkBF at a given T_c is observed in combination with a reduction in the cutaneous vasodilatory threshold (Nielsen *et al.*, 1997; Buono *et al.*, 1998; Fujii *et al.*, 2012). But due to the fact that T_c is often lower for the same intensity of exercise after acclimation, heat adaptation actually results in reduced SkBF at absolute intensities, when not directly compared to the level of T_c (Chalmers *et al.*, 2014).

2.4.2.4 Metabolism

Whilst blood flow to the active muscles appears to be unchanged following a heat acclimation protocol, alterations in substrate usage have been observed in some (but not all) studies. Various authors have concluded that the increased carbohydrate utilisation that is usually observed during exercise in the heat is partly supressed as an adaptation of heat acclimation (King *et al.*, 1985; Kirwan *et al.*, 1987; Febbraio, 2001). The reduced carbohydrate usage has also been reported in combination with less muscle and lactate accumulation, which could potentially be a factor in improved exercise performance after heat-acclimation (Young *et al.*, 1985; Kirwan *et al.*, 1987). In contrast to the reports of King *et al.* (1985) and Kirwan *et al.* (1987) who noted significant reductions in glycogen usage, studies by Nielsen and Colleagues (1993, 1997) did not observe any alterations in substrate utilisation as a result of heat acclimation. Though, once again, methodological differences between studies may play a large role in the discrepancies that have been reported. For example, both King *et al.* (1985) and Kirwan *et al.* (1987) had their participants exercise at higher intensities than Nielsen *et al.*, (1993, 1997). Not only does heat acclimation appear to reduce carbohydrate usage, it also has the potential to reduce metabolic rate, and consequently heat production (Houmard *et al.*, 1990). Various authors have reported a lower O_2 uptake ($\dot{V}O_2$) at a given exercise intensity after heat acclimation, demonstrating an increased metabolic efficiency and a decrease in O_2 demand (Shvartz *et al.*, 1972; Sawka *et al.*, 1983b; Young *et al.*, 1985; Houmard *et al.*, 1990). Improved metabolic efficiency benefits the exercising individual, especially in the heat, with reduced metabolic heat production helping establish less of a thermoregulatory and cardiovascular strain in such environments (Chalmers *et al.*, 2014).

2.4.2.5 Cellular

Exposure to hot environments induces alterations at a cellular level. Heat shock proteins (HSP) are expressed as a result of such exposure, and subsequently help to protect the body against further heat stress (Kregel, 2002). Whilst heat shock proteins are associated with the development of thermotolerance (tolerance to temperatures that were previously lethal), the HSP70 family of heat

shock proteins have also been associated with the development of heat acclimation (Moseley, 1997; Kregel, 2002). The HSP70 family is the more temperature sensitive of the heat shock proteins, and while this topic is beyond the scope of this thesis, Moseley (1997) and Kregel (2002) provide good evidence in their review articles that HSP70 is induced upon heat exposure, and the expression of this protein leads to increased tolerance to the heat and the ability to work/exercise at higher environmental temperatures.

2.4.2.6 Trained vs. Untrained Participants

An individual who is endurance trained has often developed certain physiological adaptations (as highlighted briefly above), including improved cardiovascular capacity, and some sweating modifications; positioning them in a state of 'partial heat acclimation' (Pandolf *et al.*, 1977; Taylor, 2000). Although some of the adaptations are achieved via the physical activity aspect, full heat acclimation and the associated range of physiological characteristics cannot be achieved without exercise in combination with a hot environment (Taylor & Cotter, 2006). Individuals who begin a heat acclimation regime with this partial acclimation status tend to develop all of the adaptations and fully acclimate at a faster rate than those with lower aerobic fitness levels. Pandolf *et al.* (1977) indicated that full acclimation could be complete in as little as four days for a very aerobically fit individual, and stated that there is an inverse relationship between the aerobic fitness of an individual and the length of time it takes for full acclimation to the heat. In combination with the shorter development time, aerobically fit individuals also retain heat acclimation adaptations for longer during the time course of decay; this is likely due to the fact that the underlying high aerobic fitness levels will still be casting positive adaptations (Armstrong & Maresh, 1991; Pandolf, 1998). Moreover, it appears that individuals of a lower aerobic fitness level going into a heat acclimation protocol will benefit to a greater extent than an individual with a high basal aerobic fitness level - larger decreases in heart rate and T_c will be expected as a result of heat acclimation (Shvartz *et al.*, 1977). Aerobically fit individuals begin a heat acclimation protocol with a greater extent of adaptations shared between physical activity and acclimation, thereby leaving less of a potential to develop new ones compared to lesser aerobically fit individuals (Taylor & Cotter, 2006; Garrett *et al.*, 2011). Heat acclimation holds benefits for all fitness levels, with the extent of physiological adaptations obtained from the actual heat acclimation protocol dependent on what adaptations were pre-existing within the individual.

2.4.2.7 Decay of Physiological Adaptations

Unfortunately, the physiological adaptations obtained during heat acclimation do not persist for long after exercise in combination with exposure to the heat is ceased. The literature surrounding the decay of heat acclimation is scant compared to the induction, and is equivocal due to many confounding variables that have an effect on how fast the adaptations from a heat acclimation protocol are both induced, and decay – including type of acclimation (has been proposed that dry-heat acclimation lasts longer than humid-heat (Pandolf, 1998)), fitness level, activity during decay period, and length of heat acclimation protocol (Garrett *et al.*, 2011). In general, it has been concluded by various authors that adaptations have largely returned to normal levels 3 weeks after the cessation of a heat acclimation protocol (Adam *et al.*, 1960; Williams *et al.*, 1967; Armstrong & Maresh, 1991; Garrett *et al.*, 2011), and that for every two days with no heat exposure, one day of heat acclimation is lost (Givoni & Goldman, 1972). Table 2.2 illustrates three early studies completed associated with the decay of heat acclimation, and demonstrates some of the discrepancies that are found within the literature. Pandolf *et al.* (1977) noted only small reductions in the heat acclimation adaptations over the 18-day decay period, but this may be due to the fact that the participants involved had a high natural state of heat acclimation and aerobic fitness levels before starting the protocol, therefore would be expected to decay at a slower rate (Armstrong & Maresh, 1991).

Table 2.2. Time-course for the decay of heat acclimation from three different studies (Armstrong & Maresh, 1991).

Technique or measurement	Loss of heat acclimatisation (%) during periods of no heat exposure				Reference
	6-7 days	12-14 days	18-21 days	28 days	
Heart rate	50	80	100		Williams et al. (1967)
Rectal temperature	25	40	50		
Heart rate	23	20	29		Pandolf et al. (1977)
Rectal temperature	13	18	4		
Heat tolerance	Substantial			100	Adams et al. (1960)

The first physiological adaptations that begin to decay are those that are first induced (i.e. the cardiovascular adaptations); those taking the longest to develop (sudomotor adaptations) decaying at the slowest rate (Williams *et al.*, 1967; Pandolf *et al.*, 1977; Armstrong & Maresh, 1991; Pandolf, 1998; Garrett *et al.*, 2009; Garrett *et al.*, 2011). Interestingly, it has been found that the PV and cardiovascular adaptations that have been previously proposed to be linked during induction do not mimic each other during the decay of acclimation. It is this lack of correlation between the two that suggests other factors (i.e. central mechanisms), and not solely hypervolemia, may be associated with heat acclimation induced cardiovascular adaptations (Garrett *et al.*, 2009).

The rapid rate of decay of the adaptations from a heat acclimation protocol needs to be taken into consideration by athletes, especially those who are using heat-training in the hope of developing optimal adaptations leading up to an event in a hot climate, such that re-introduction of heat exposure will be beneficial to maintain the physiological adaptations. Therefore, it would be recommended that an athlete partake in a heat acclimation protocol no longer than three weeks out from their event.

The physiological adaptations that arise from completing an appropriate level of physical activity in a hot environment over a chosen period of time, lead to a greater amount of heat (originating in the muscles and core) to be dissipated from the body via the skin; lowering the T_c of the body for the level same environmental and exercise stress, and enabling a person to work for harder/longer in this heat (Taylor, 2000). The physiological responses developed, and reduced thermoregulatory strain encountered, by the heat acclimated individual have benefits to everyday health, in that they possess more efficient cardiovascular, metabolic and thermoregulatory systems, and are thereby able to deal with not only heat, but everyday stresses more competently than prior to heat acclimation.

2.4.3 Effects on Exercise Performance/Aerobic Capacity

2.4.3.1 Performance in Hot Environments

The physiological adaptations that are obtained through repeated exposure to the heat have the potential to positively impact performance in the heat, as cardiovascular capacity has been improved, and thermoregulatory strain for the same environmental conditions has been reduced. Table 2.3 illustrates the studies that have been completed so far, with the focus on how heat acclimation impacts performance parameters, and is divided into short-term and moderate/long-term heat acclimation protocols. In general, the consensus from the literature is that heat acclimation will improve performance in a hot environment. Unfortunately, the large range of environmental conditions, exercise modalities, methods of heat acclimation, durations of heat acclimation, and performance parameters employed by the previous research make it difficult to clarify how much of an extent heat acclimation can have on performance. The large variability amongst the studies is illustrated in the 'performance change' column, which ranges from no change up to almost a 70% increase in $\dot{V}O_2$max performance. One of the biggest limitations amongst the studies highlighted in Table 2.3 is the lack of control groups. Without a control group in the study, the performance cannot be concluded as to be a result of heat acclimation itself, as it may be just the exercise improving performance, irrespective of the heat. As previously discussed, a period of

exercise at a suitable intensity has the potential to induce some of the adaptations observed with repeated heat exposure; to conclude that performance improvements are a result of the combination of exercise and heat, a control group is essential. The studies that have employed exercising control groups have noted performance improvements in the heat acclimation groups, with no enhancements to performance registered in the control groups (Nielsen *et al.*, 1993; Sunderland *et al.*, 2008; Lorenzo *et al.*, 2010). Therefore, these studies were able to conclude that it was specifically the heat acclimation protocol itself, not just the exercise, which led to the improved exercise performance in the heat, post-heat acclimation.

The physiological adaptations that arise from the development of heat acclimation hold the potential to improve subsequent exercise in hot environmental conditions. This is proven in the associated literature, leading to the conclusion that some form of heat acclimation prior to events (specifically endurance) in hot environmental conditions can be beneficial to optimising performance.

Table 2.3. Previous studies investigating the effect of short- to long-term heat acclimation protocols on performance in a hot environment (30-50°C).

Study	HA Participants	Control Participants	HA Protocol	Exercise Modality	Performance Environment	Performance Measure	Performance Change (%)
Cotter *et al.* (1997)	8 physically active	NA	6d; 70min; CH 40°C, 60% RH	Cycling	40°C, 60% RH	WC in 70min	↔
Patterson *et al.* (2004)	12 physically active	NA	7d; 90min; CH 40°C, 60% RH	Cycling	40°C, 60% RH	WC in three 30min bouts @ ~30% PPO	↑ 8%
Sunderland *et al.* (2008)[C]	6 well-trained game players	5 well-trained game players	4d; 30-45min; LIST 30°C, 24% RH	Running (LIST)	30.5°C, 27% RH	Distance run TE (LIST)	↑ 33% HG; ↔ CG
Garrett *et al.* (2009)	10 relatively trained	NA	5d; 90min; CH 40°C, 60% RH	Cycling	35°C, 60% RH	90min SS @ 40% PPO + IET TE ($\dot{V}O_2$max)	↑ 14%
Garrett *et al.* (2012)	8 highly-trained rowers	NA	5d; 90min; CH 40°C, 60% RH	Rowing (Perf) Cycling (HA)	35°C, 60% RH	2km TT	↑ 1.5%
Sawka *et al.* (1985)	13 soldiers	NA	9d; 120min; 40-50% $\dot{V}O_2$max; 49°C, 20% RH	Walking	49°C, 20% RH	$\dot{V}O_2$max (cycling) + PPO	↑ 4% ($\dot{V}O_2$ max) ↑ 2% (PPO)
Nielsen *et al.* (1993)[C]	8 endurance trained	5 endurance trained	9-12d; 60% $\dot{V}O_2$max TE 40°C, 10% RH	Cycling	40°C, 10% RH	60% $\dot{V}O_2$max TTE	↑ 66% HG; ↔ CG
Nielsen *et al.* (1997)	12 endurance trained	NA	8-13d; 45% $\dot{V}O_2$max TE 35°C, 87% RH	Cycling	35°C, 87% RH	45% $\dot{V}O_2$max TTE	↑ 15.5
Cheung and McLellan (1998)	7 untrained; 8 trained	NA	10d; 60min, 4.8km·h^{-1}, 3-7% grade NBC; 40°C, 30% RH	Walking	40°C, 30% RH	60min; 3.5km·h^{-1}, 0% grade NBC	↔
Lorenzo *et al.* (2010)[C]	12 highly-trained endurance cyclists	8 highly-trained endurance cyclists	10d; 90min; 50% $\dot{V}O_2$max 38°C, 30% RH	Cycling	38°C, 30% RH	MAPT + 60min TT	↑ 8% HG; ↔ CG (MAPT) ↑ 8%; ↔ CG (TT)

[C], control group present; d, day; CH, controlled-hyperthermia; RH, relative humidity; $\dot{V}O_2$max, maximal oxygen consumption; TE, to exhaustion; NBC, nuclear, biological and chemical protective clothing; LIST, Loughborough Intermittent Shuttle Test; Perf , performance; HA, heat acclimation; WC, work completed; PPO, peak power output; IET, intermittent exercise test; TTE, time to exhaustion; TT, time trial; MAPT, maximal aerobic power test; HG, heat group; CG, control group; ↑, performance increase; ↓, performance decrease; ↔, no change in performance.

2.4.3.2 Performance in Moderate/Cool Environments

Whilst the majority of the previous literature has focussed on how heat acclimation can impact performance in hot environmental conditions, there has been a small selection of studies that have investigated the impact of heat acclimation on performance in moderate/cool environments. The notion that adaptation to the heat could have beneficial effects outside of that type of environment has been related to the "live high-train low" altitude training concept developed by Levine and Stray-Gundersen (1997). These authors illustrated that adaptations obtained from exposure to a certain environment (i.e. hypoxia) could improve performance in an alternative environment (low-altitudes). Whilst this principal involves training at a low altitude, but living at a high-altitude to improve performance at sea-level (almost opposite to the concept of the current thesis), the idea that certain physiological adaptations obtained from one environment can be transferrable to an alternative still stands. Comparing the current research to the hypoxia theory, it would be equivalent to a live-low, train-high initiative where an athlete may utilise high altitude training to help improve performance at lower-altitudes. This concept could be transferable to heat acclimation and performance in cooler locations as physiological adaptations present with acclimation - improved cardiovascular capacity, and increased and more efficient sweating - are such that benefits to performance in any environment after heat acclimation could be expected. The applicability of this concept to sporting events is present, as many team sports involve pre-season training in a climate either hotter or cooler than when they will be performing. For example, soccer is a sport played in winter, however pre-season training occurs in the warmer months, which could be utilised to improve subsequent performance in the cold. This type of training could benefit all individuals, both highly trained and less aerobically fit, through faster improvements to their current fitness levels. Interestingly, the focus on heat acclimation and its impact on performance in moderate/cool environments has been neglected in the previous literature, with some of the previous associated research outlined in Table 2.4. The previous research has employed a wide range of exercise modalities, participants, durations of heat acclimation, as well as environmental temperatures and performance parameters; making it very difficult to compare directly between them. So far, all of the previous reported literature, apart from the study by Morrison *et al.* (2002), has observed an increase in performance post-heat acclimation; the improvements ranging from 4 - 44%. The studies by Lorenzo *et al.* (2010), Scoon *et al.* (2007), and Morrison *et al.* (2004) have utilised randomised cross-over studies or used a matched control group to enable direct conclusions to be made about the impact that the heat stress has on performance. Lorenzo *et al.* (2010) used a matched control group that completed the same training as the heat acclimation group, in cool environmental conditions (13°C, 30% RH). The results from this study showed that the heat acclimation improved

performance to a greater extent, noting advances in both maximal aerobic power, and time trial performance; compared to no improvements observed in the control group. The authors were able to conclude that the enhanced performance was due to the heat acclimation protocol, not just the physical training. The authors proposed that the improved maximal aerobic power observed could be a result of the heat acclimation adaptation of expanded PV, in combination with cardiovascular adaptations; increased ventricular compliance, and an increased cardiac output (Lorenzo *et al.*, 2010).

Although the majority of the studies have not included a control group, it appears that heat acclimation has the potential to improve performance in moderate/cool environments, but this is an area that needs a lot more focus on in order to clarify the extent that this improvement may be. Considering that all studies, except one (Shvartz *et al.*, 1977), have used participants that are either professional athletes (Buchheit *et al.*, 2013), trained athletes (Morrison *et al.,* 2002; Hue *et al.,* 2007; Scoon *et al.,* 2007; Buchheit *et al.,* 2011) or aerobically fit soldiers (Sawka *et al.*, 1985), future studies should aim to clarify the impact that heat acclimation can have on performance in moderate/cool environments for less aerobically fit individuals. This will give an indication of the impact that heat acclimation has on performance in a cooler environment for the general exercising population. Shvartz *et al.* (1977) reported greater improvements in performance after heat acclimation in unfit, compared to aerobically fit participants, but unfortunately due to the differences in aerobic fitness levels between the control group (45.3 $mL \cdot kg^{-1} \cdot min^{-1}$) and the unfit group (35.7 $mL \cdot kg^{-1} \cdot min^{-1}$) it is difficult to clarify how much of the improvement is due to the heat acclimation itself, over and above the physical exercise. Scoon *et al.* (2007) is the only study to have investigated the impact of heat acclimation on running performance in a moderate environment, but unlike the majority of previous research, this study induced heat acclimation through passive heating post-training rather than through exercising methods. Running is involved in a large proportion of different sports and by individuals for everyday fitness; therefore, verifying the impact that heat acclimation (induced through training in a hot environment) can have on running in a moderate environment could be an area of research that would apply to most of the population.

Table 2.4. Previous studies investigating the effect of heat acclimation protocols on performance in a cool/moderate environment (10-25°C).

Study	HA Participants	Control Participants	HA Protocol	Exercise Modality	Performance Environment	Performance Measure	Performance Change (%)
Shvartz *et al.* (1977)[C]	7 trained; 7 untrained; 7 unfit	5 relatively trained	8d; 180min; 41W; 39.4°C db, 30.3°C wb	Bench stepping	23°C	$\dot{V}O_2$max (running)	↔ (trained) ↑ 13% (untrained) ↑ 23% (unfit) ↔ (control)
Sawka *et al.* (1985)	13 soldiers	NA	9d; 120min; 40-50% $\dot{V}O_2$max; 49°C, 20% RH	Walking	21°C, 30% RH	$\dot{V}O_2$max (cycling) + PPO	↑ 4% ($\dot{V}O_2$max) ↑ 4% (PPO)
Morrison *et al.* (2004)	9 highly-trained cyclists	9 highly-trained cyclists	7d; 90min; SS; 37°C, 50% RH	Cycling	20°C, 50% RH	40km TT	↔
Hue *et al.* (2007)	16 trained swimmers	NA	8d; 14 sessions in 30°C water; ~30°C, 80% RH	Swimming	27.1°C water	400m TT – 10 & 30 days post-exposure	↑ 10% (at 30days)
Scoon *et al.* (2007)	6 trained runners	6 trained runners	21d; 12 post-training sauna sessions; ~90°C	Running	Not stated	Run TE @ max 5km speed	↑ 2% endurance performance
Lorenzo *et al.* (2010)[C]	12 highly-trained endurance cyclists	8 highly-trained endurance cyclists	10d; 90min; 50% $\dot{V}O_2$max 38°C, 30% RH	Cycling	13°C, 30% RH	MAPT + 60min TT	↑ 5% HG; ↔ CG (MAPT) ↑ 6% HG; ↔ CG (TT)
Buchheit *et al.* (2011)	15 trained soccer players	NA	7d training camp; 60-95min; 50-83% $\dot{V}O_2$max; ~40°C, 27% RH	Soccer training	22°C, 50% RH	Distance run TE (Yo-Yo IET)	↑ 7%
Buchheit *et al.* (2013)	17 professional Australian Rules football players	NA	14d training camp; 10 skill sessions (~70min; 29-33°C, 37-50% RH); 15h IHE; ~13h interval/strength (~23°C, 55% RH)	Football skills, interval/strength training	22-23°C	Distance run TE (Yo-Yo IET)	↑ 44%
** Bradford *et al.* (2015)	8 trained swimmers	8 trained swimmers	7d; 60min; 33°C water	Swimming	28°C (swimming) 29°C (air)	Distance swum/cycled (20min)	↔
** Karlsen *et al.* (2015)	9 trained cyclists	9 trained cyclists	14d; normal training; 35°C	Cycling	~5-13°C	$\dot{V}O_2$max Outdoor 43.4 TT	↔ ($\dot{V}O_2$max) ↔ (TT)

[C], control group present; NA, not applicable; d, day; -W, Watts; db, dry-bulb; wb, wet-bulb; $\dot{V}O_2$max, maximal oxygen consumption; RH, relative humidity; SS, self-selected; IHE, incidental heat exposure; IET, intermittent exercise test; TTE, time to exhaustion; PPO, peak power output; TT, time trial; TE, to exhaustion; MAPT, maximal aerobic power test; HG, heat group; CG, control group; ↑, performance increase; ↓, performance decrease; ↔, no change in performance.

** Studies were published after the submission of this thesis.

2.4.4 Section Summary

There is a vast range of literature associated with temperature regulation (at rest and during exercise), heat acclimation and the associated physiological adaptations that arise, leading into how these positive changes within the body can affect subsequent performance. The physiological changes have been well documented amongst the literature, with the impact that heat acclimation has on performance in the heat appearing to be positive, but is slightly more ambiguous due to the large variation in methodologies, environments, participants, in combination with the lack of matched control groups. Research associated with how heat acclimation affects performance in moderate/cool environments has been largely neglected in the previous literature; the studies that have been completed thus far have not developed any concrete conclusions. The physiological adaptations that develop with repeated heat exposure such as lowered T_c, improved cardiovascular efficiency, and PV expansion are adaptations that are not only specific to improving thermoregulation and performance in a hot environment. Such physiological changes would be expected to have benefits to any subsequent exercise performed, regardless of the environmental temperature. Therefore, this is an important area of future research and is the reason behind the decision to investigate this topic; if the results are positive, this form of training, and the adaptations gained from exercise in a thermally stressing environment, could be put to practical use, not only by athletes to gain an edge over fellow competitors, but also by the general population as a way to establish the positive adaptations that will improve aerobic fitness and resting cardiovascular measures concurrently.

Chapter Three: Research Aim and Hypotheses

The research conducted in this thesis was built from undertaking a comprehensive review of the current literature, which is summarised in **Chapter Two**; it has been identified that the effects of heat acclimation on performance in a moderate environment requires further investigation. **Chapter Two** provided insights of how heat acclimation and the associated physiological adaptations can help to reduce the thermoregulatory strain of a hot environment during subsequent exposures for an exercising human. The idea that these physiological alterations may also have the potential to enhance performance in moderate/cool environments has gained some popularity, although differing exercise modalities and equivocal findings have made it difficult to draw definite conclusions. Therefore, the aim of this study was to determine what effect exercising with additional heat stress (35°C) has on maximal and submaximal aerobic capacity/performance of a moderately active individual, in a moderate environment (18°C), in combination with physiological adaptations obtained and their impact on resting cardiovascular measures.

The primary hypothesis for the current study is outlined below:

1. Exercise, with or without additional heat stress will significantly improve maximal aerobic capacity; further, exercise in combination with added heat stress will significantly enhance performance to a greater extent than exercise alone.

A number of secondary hypotheses were proposed, including:

2. Exercise in combination with heat stress will significantly improve submaximal aerobic efficiency to a greater extent than exercise alone, illustrated by reduced heart rate, blood lactate, and $\dot{V}O_2$ for the same intensity of exercise.
3. Exercise in combination with heat stress will significantly enhance resting cardiovascular measures greater than will exercise alone, indicated by lowering resting heart rate and blood pressure.
4. Exercise in combination with heat stress will induce heat acclimation, illustrated by a lowering of T_c, exercising heart rate and perceived exertion, expanding PV, and increasing sweat rate.

Chapter Four: Methodology

4.1 Experimental Overview

Eighteen healthy and moderately active males completed an 11-day exercise protocol, walking for an hour at 50% of their $\dot{V}O_2max$ in either hot (35°C, 45% RH) or moderate (18°C, 53% RH) environmental conditions. Figure 4.1 gives a general overview of the protocol; participants completed a running maximal aerobic exercise test and submaximal exercise test on separate days in the week leading up to- and following- the 11-day training protocol. All exercise sessions took place at the same time of day and the 11 exercise days were divided into two blocks of five and six consecutive days, with one day rest between.

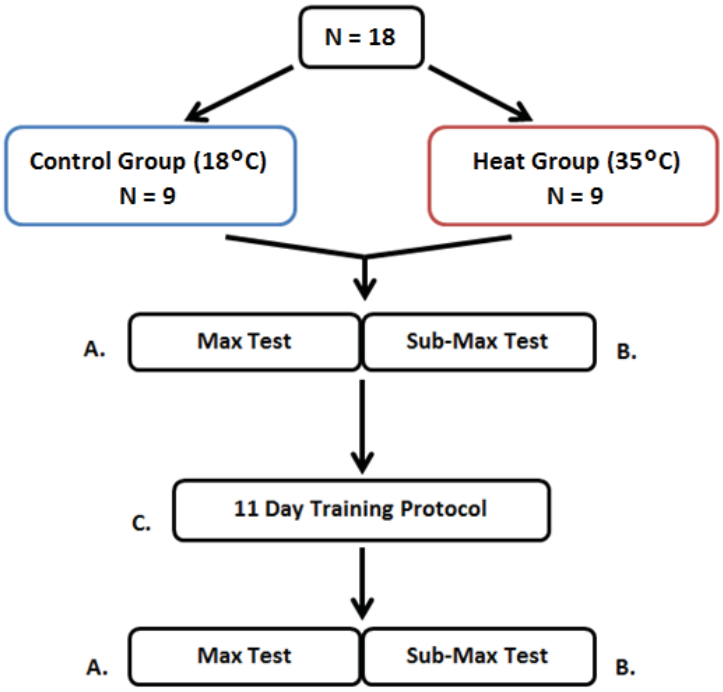

Figure 4.1. General overview of the testing procedure completed over a four week period for the control group (n=9) and the heat group (n=9). A = Pre- and post-training maximal aerobic capacity tests; B = Pre- and post-training submaximal aerobic tests; C = 11-day training protocol.

4.1.1 Participants

Before participation, each participant was fully informed of the experimental procedures and possible risks before giving informed, written consent. This protocol was approved by the Massey

University Human Ethics Committee. Eighteen healthy and moderately active males volunteered to participate in this study. The participants' characteristics were (mean ± SE) age 24.9 ± 1.9 years; height 179.0 ± 2.6 cm; weight 83.6 ± 5.3 kg; $\dot{V}O_2$max 56.8 ± 2.3 mL·kg^{-1}·min^{-1}. The eighteen participants were divided into two groups of nine (control and heat) that were matched for height, weight, body surface area (BSA), percent body fat, and maximal aerobic capacity (Table 4.1).

Table 4.1. Physical characteristics and initial aerobic performance of the control and heat groups

Characteristics	Control Group (n=9)	Heat Group (n=9)	P Value
Age (years)	26 ± 2	23 ± 2	0.309
Height (cm)	177 ± 2	181 ± 3	0.309
Weight (kg)	84 ± 6	83 ± 5	0.893
BMI (kg·m^{-2})	27 ± 2	25 ± 1	0.473
Body surface area (m^2)	2.0 ± 0.1	2.0 ± 0.1	0.832
% Body fat	17 ± 2.	14 ± 2	0.444
$\dot{V}O_2$max (mL·kg^{-1}·min^{-1})	58 ± 2	56 ± 2	0.523
$\dot{V}O_2$max (L·min^{-1})	4.8 ± 0.2	4.6 ± 0.2	0.485

Vales are shown as means ± SE. Reported values of maximal oxygen uptake ($\dot{V}O_2$max) were from a $\dot{V}O_2$max test completed in a moderate environment (18°C, 52% RH). BMI, Body mass index.

4.1.2 Pre- and Post-Training Sessions

In the week prior to the beginning of the 11-day training protocol, participants reported to the Human Performance Laboratory (HPL) for two sessions on two separate days (A. and B. on Figure 4.1). The first session (Figure 4.2) involved the determination of anthropometric measurements (height, weight and body composition), resting heart rate and blood pressure, followed by an incremental running exercise test (Bruce protocol) on a wide-bodied treadmill (Payne, Australia) to determine maximal aerobic capacity. The Bruce Protocol is an incremental maximal walking/running treadmill test, which involved the participants completing 3 minute stages, to exhaustion. The first stage started at 2.74 km·h^{-1} at an incline of 10%, the speed increased by 1.28 km·h^{-1} and grade by 2% every 3 minutes, up until the 15th minute where after the speed increased by 0.81 km· h^{-1} and grade by 2%. Heart rate was recorded at each stage and participants exercised to volitional exhaustion; expired gas was collected in the final 2-3 minutes of the test to determine $\dot{V}O_2$max. The second preliminary session (Figure 4.3) involved a submaximal protocol where participants completed 5 minute stages on a commercial treadmill (TRUE, St Louis) at 40, 50, 60, and 70% of their $\dot{V}O_2$max, calculated from the prior maximal aerobic capacity test. Resting blood lactate (BLa) and heart rate were recorded initially; with expired gas, BLa, heart rate and running speed collected and recorded in the final minute of every stage of the submaximal test. Subsequent to end of the test, a rest period of 20 minutes was provided, followed by a short familiarisation period walking on the treadmill to mimic the speed and incline that the participant was walking at during their training

period. A one minute expired respiratory gas sample was collected to verify the intensity of exercise. The period of walking lasted 20 minutes and provided the individual with an opportunity to get acquainted with the speed and incline that they were walking at throughout the 11-day training protocol. The two preliminary sessions were performed in a moderate environment (18°C, 52% RH).

Within 4 ± 1 days of completing the 11-day training protocol, participants reported to the HPL on two separate occasions to complete the maximal and submaximal tests, consisting of exactly the same protocols (and the exact same treadmill speeds for the submaximal test) that were undertaken in the pre-training sessions, excluding the familiarisation.

Participants were asked to complete a food and activity diary for the day prior to, and the day of their maximal and submaximal sessions. This diary was replicated by them for their post-training maximal and submaximal sessions to minimise other variables influencing performance.

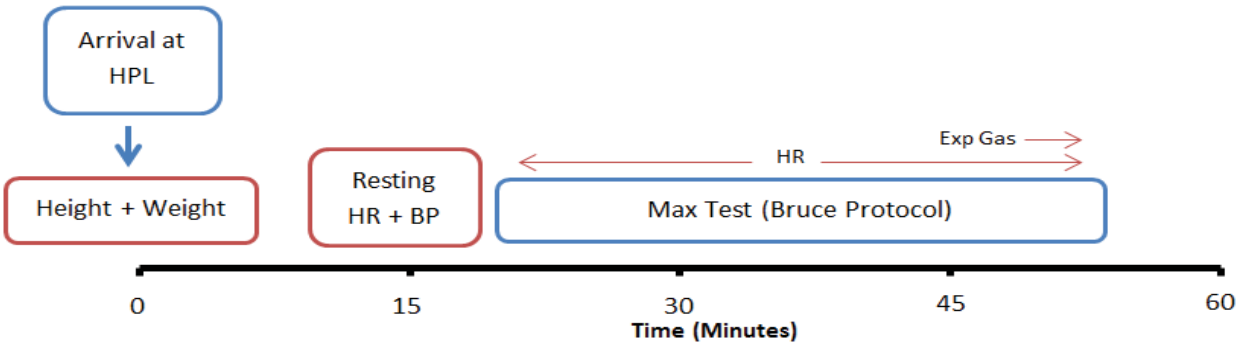

Figure 4.2. Overview of the maximal-test visit to the HPL in the week before and after training for the control group (n=9) and the heat group (n=9). HPL, Human Performance Lab; HR, heart rate; BP, blood pressure, Exp Gas, expired gas.

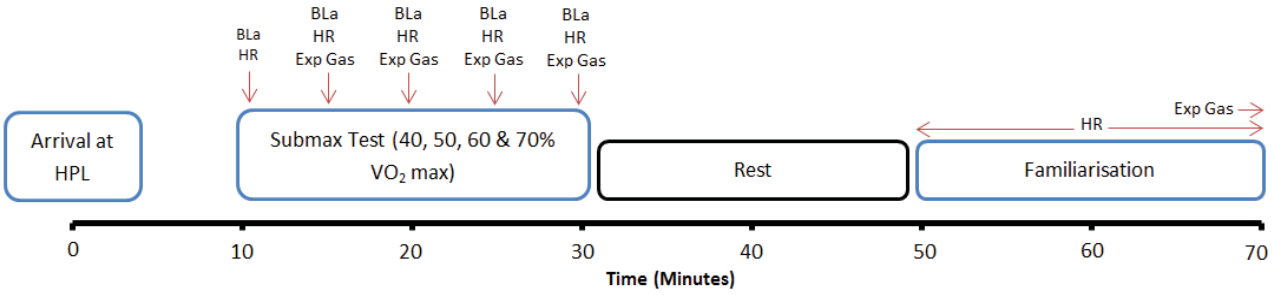

Figure 4.3. Overview of the submaximal test and familiarisation visits to the HPL in the week before and after training, for the control group (n=9) and the heat group (n=9). HPL, Human Performance Lab, BLa, blood lactate; HR, heart rate; Exp Gas, expired gas.

4.1.3 Training Protocol

The 11-day training protocol commenced within a week of completion of the maximal and submaximal sessions and is summarised in Figure 4.4. Every training day, participants reported to the HPL at the same time of day to minimise variables such as circadian T_c changes and previous activity. All participants arrived at the laboratory for all training sessions in a hydrated state, determined via a urine specific gravity (USG) measure, having refrained from physical activity and food intake in the two hours leading up to the session. A record of additional exercise that participants were taking part in outside of the daily training sessions was completed by each participant for the entirety of the 11-day training period. Participants were asked to wear the same combination of clothing to each training session – athletic shoes, shorts and the choice of either a t-shirt, singlet or topless (the decision of either a t-shirt, singlet, or topless was made in the first session and kept constant for each participant throughout the subsequent training sessions). Upon arrival, participants voided and provided a urine sample (analysis of USG) to determine hydration status. Following this, a nude weight was obtained, before self-insertion of a rectal thermistor 10 cm beyond the anal sphincter. A heart rate monitor was applied and participants entered the climatic chamber where resting T_c was recorded. Immediately prior to the initiation of exercise, perceptual measures of thermal sensation (ThS) and thermal comfort (ThC) were recorded. During the one hour of walking, participants were provided with water ad libitum and 1-2 $mL \cdot kg^{-1}$ body weight of an isotonic sports drink (Powerade, The Coca-Cola Company, NSW, Australia) every 15 minutes. Heart rate, T_c and environmental conditions (dry-bulb (ambient) temperature, wet-bulb temperature, and RH) were recorded every five minutes; perceptual measures of RPE, ThC and ThS were assessed every 15 minutes.

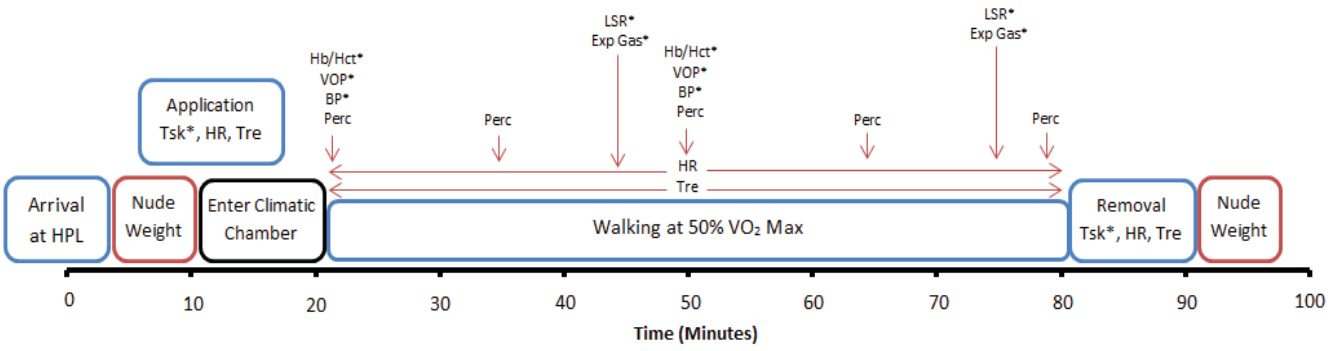

Figure 4.4. Overview of each of the training days in the 11-day training protocol for the control group (n=9) and the heat group (n=9). * Measures only taken on days 1, 5 and 11; HPL, Human Performance Laboratory; Tsk, skin temperature; HR, heart rate; Tre, rectal temperature; Hb, haemoglobin; Hct, haematocrit; BP, blood pressure; Perc, perceptual measures; LSR, local sweat rate; VOP, venous occlusion plethysmography; Exp Gas, expired gas.

In addition to heart rate, T_c and perceptual measures, additional recordings were taken on training days 1, 5 and 11. On these days, before entering the climatic chamber, T_{sk} thermistors were attached at the same time as the heart rate monitor. Upon entering the chamber, participants stood on the treadmill for five minutes before a strain gauge was applied to the forearm and inflatable cuff to the upper arm for measurement of resting forearm blood flow via the venous occlusion plethysmography (VOP) method. Pre-exercise blood pressure and haemoglobin (Hb) and haematocrit (Hct) readings were also obtained, after the participant had been standing for 10 minutes. At 25 minutes of exercise, a sweat patch was applied to the forearm for the determination of local sweat rate (LSR) via the technical absorbent method, and a one minute expired respiratory gas sample was collected in a Douglas bag for determination of $\dot{V}O_2$. At 30 minutes, the participant stopped walking and measures of VOP, and blood pressure were repeated before the continuation of exercise. At 55 minutes a second measure of both LSR and expired respiratory gas were collected.

Upon completion of the one hour exercise bout (every training session), participants removed the rectal thermistor and upon removing excess surface sweat, performed a final nude weight.

4.1.4 Justification of Methodology

The majority of the limited associated literature has focussed on the impact that heat acclimation has for highly-trained athletes; however there is little information for the general population. This fact led to the decision to recruit males who were recreationally/moderately active and are more representative of the majority of the population who exercise at least two-three times per week.

Initially, the heat stimulus, duration of protocol, exercise intensity and duration was based on Lorenzo *et al.* (2010), and with the addition of pilot work the intensity was set at 50% of $\dot{V}O_2$max, as it was thought that a higher intensity would be unrealistic for the participants to maintain for 60 minutes of exercise, given the level of their initial fitness. The decision was made to use a FI exercise protocol as opposed to SP or controlled-hyperthermia, which allowed the observation of physiological adaptations to the heat (e.g. reduced T_c, heart rate etc.) for the same exercise intensity. Contrastingly, Lorenzo *et al.* (2010) had participants complete 90 minutes of exercise in total on each day of the protocol (45 minutes, 10 minutes break, 45 minutes); we decided to reduce the duration for our study to 60 minutes, due to a combination of the lower fitness levels of the participants, and the weight-bearing modality of exercise (walking) that was employed.

The performance measures were assessed by investigating running performance, rather than cycling; indicative of the fact that running is involved in a greater number of sports and is a popular

modality of exercise for the general population, whilst taking into consideration the experience of the participants. Running involves frequent impact with the ground which can cause muscle damage; this was taken into consideration when designing the acclimation protocol. An increased chance of muscle soreness, combined with the fact that the participants were not trained runners, meant that walking at an incline on the treadmill for training was determined to be the best option; running would cause greater muscle damage, and increase the potential of participant drop-outs. The decision to have walking as the training modality did not completely eliminate the possible muscle damage, as walking still has large ground impact, and when walking uphill, some muscle damage may ensue. To reduce the number of possible participant drop-outs, participants were provided with one rest day after five days of the protocol, followed by a remaining six training days.

The heat stimulus from Lorenzo *et al.* (2010) was 38°C, combined with 30% RH. To ensure an appropriate T_c rise occurred during heat acclimation, pilot work was performed to compare exercise at 50% of $\dot{V}O_2$max for 60 minutes at 38°C and 35°C, in combination with low RH (hot-dry environment). The decision was made that 35°C, combined with an average RH of 45% was sufficient to bring about the appropriate changes in T_c whilst maximising participant adherence.

4.2 Experimental Procedures

4.2.1 Measurements

The participant's height and weight were measured using a stadiometer (Seca, Bonn, Germany; accurate to 0.1 cm) and body scale (Hiweigh Technologies Ltd, Shanghai, China; accurate to 0.02 kg), from which BMI (Keys *et al.*, 1972) and BSA (Du Bois & Du Bois, 1916) was estimated. Body composition was measured using a Bioelectrical Impedance Analyser (InBody230, Korea). Heart rate and blood pressure were monitored using a heart rate monitor (Polar FS1, Polar Electro, Finland) and a digital blood pressure monitor (Nissei, Japan), respectively. Expired gases were collected for 1 minute via standard Douglas bags. The expired gases were analysed for CO_2 and O_2 concentrations (AEI Technologies, USA) and volume (dry gas meter, Havard, UK), and values converted to STPD. Rates of O_2 uptake ($\dot{V}O_2$), CO_2 elimination (VCO_2), respiratory exchange ratio (RER), and expiratory minute ventilation (V_E) were calculated. Metabolic rate was calculated using the equation $\dot{V}O_2$ ($L \cdot min^{-1}$) x non-protein RER kcal equivalent and presented in $kcal \cdot min^{-1}$. The non-protein RER kcal equivalent was obtained from standard non-protein RER equivalent tables developed by Lusk (1924). BLa was measured via a small (0.3 µL) sample of blood from the fingertip via lactate strips (Lactate

Pro 2, ARKRAY, the Netherlands), which was analysed by BLa test meter (Lactate Pro 2, ARKRAY, the Netherlands). USG was measured via a small urine sample being placed on a Master Refractometer (Atago, Tokyo, Japan), which was calibrated on a daily basis using a distilled water sample.

Four skin temperature thermistors (iButtons, Maxim, San Jose, USA; accurate to 0.05°C) were secured in place with Transpore Surgical Tape (3M Healthcare, St. Paul, Minnesota, USA) to the chest, thigh, leg and arm on the left side of the body for determination of $\overline{T}_{sk}$ (Ramanathan, 1964).

VOP was used to measure forearm arterial blood flow via the method first described by Whitney (1953). A blood pressure cuff was placed just proximal to the elbow and inflated to between 50-60 mm Hg, occluding venous outflow but allowing sufficient arterial inflow to the limb. The cuff was inflated rapidly to this level via a custom compressor and pressure transducer system for 5-10 seconds and then rapidly deflated. The expansion of the forearm that occurs as a result of the restricted venous drainage was measured via a Mercury-in-Silastic double-strand gauge (Hokanson, WA, USA), which was placed around the widest part of the forearm and secured in place with surgical tape. The strain gauge voltage output data were acquired via an analogue-to-digital converter (PowerLab ML870; ADInstruments, Australia) and analysed using LabChart software (v7.3.3, ADInstruments). The initial rate of expansion (slope), representing arterial inflow, was manually selected in LabChart and determined by the least sum of squares and averaged over the three measurements taken. Forearm arterial blood flow was then calculated by the equation 200 x increase in forearm circumference ($mm \cdot min^{-1}$) / forearm circumference (mm) and presented in the units $mL \cdot 100\ mL\ tissue^{-1} \cdot min^{-1}$ (Whitney, 1953).

Hb levels were measured via a finger-prick blood sample collected via Hb 201^{+} microcuvettes (HemoCue AB, Angelholm, Sweden) and analysed in a Hb 201^{+} Analyser (HemoCue AB, Angelholm, Sweden). Percentage of Hct in the blood was calculated from the average of two capillary tube blood samples that were spun down in a Hct centrifuge (Micro MB, Thermo IEC) to separate Hct from plasma. The Hb and Hct measures were used to calculated PV change over the training period (between days 1-5, and days 1-11) via the method proposed by Dill and Costill (1974).

Whole body sweat rate (SWR) was estimated from the difference in nude body weight before and after training and corrected for consumption of liquid and urine output – using the following equation: ((Nude weight pre – Nude weight Post)+((Liquid consumed (mL)/1000)-(Urine output (mL)/1000)))/ Exercise duration. SWR values were presented as $L \cdot h^{-1}$.

LSR of the forearm was calculated from the application of a 6 cm^2 sweat patch for a five minute period using the technical absorbent method (Havenith *et al.*, 2008). The sweat patch was weighed

in a zip-lock bag before application and re-weighed immediately after being applied to the left forearm of the participant for exactly five minutes. The sweat patch was applied to the skin, covered with a bandage and taped in place so no movement of the patch was possible. LSR was calculated using the difference between post- and pre-application mass (where 1 g is assumed to be equivalent to 1 mL of sweat), divided by the surface area of the patch (6 cm^2) and the duration of the application (5 min) (Morris *et al.*, 2013). LSR values were presented as $mL \cdot min^{-1} \cdot cm^{-2}$

RPE were measured on the 15-grade Borg rating of perceived exertion scale (from 6 to 20; (Borg, 1982)). ThC and ThS were determined on four (from 1 – comfortable to 4 – very uncomfortable) and seven (from 1 – cold to 7 – hot) point scales, respectively (Gagge *et al.*, 1967).

4.3 Statistical Analysis

A two-way, repeated measure analysis of variance (ANOVA) was completed for pre- and post-training maximal test results. Two-way and three-way, repeated measure ANOVAs were applied to the pre- and post-training submaximal test data, and also applied within and between groups for the data collected from the 11-day training period. Where an interaction effect was observed, a pairwise post-hoc analysis was performed to determine specific differences, using the Bonferroni adjustment, which reduced the possibility for Type I error for multiple comparisons. Correlations were produced assess the relationships between changes in maximal test results to alterations in various training variables collected during the 11-day training period.

All data were assessed for sphericity and if needed, the Huynh-Feldt correction was used. All data were analysed using SPSS statistical software (Version 20, Chicago, IL, USA), with statistical significance set at $p < 0.05$. All data are reported as mean ± standard error of mean (SE) for eighteen participants, unless otherwise stated.

Chapter Five: Results

5.1 Maximal Aerobic Capacity Test

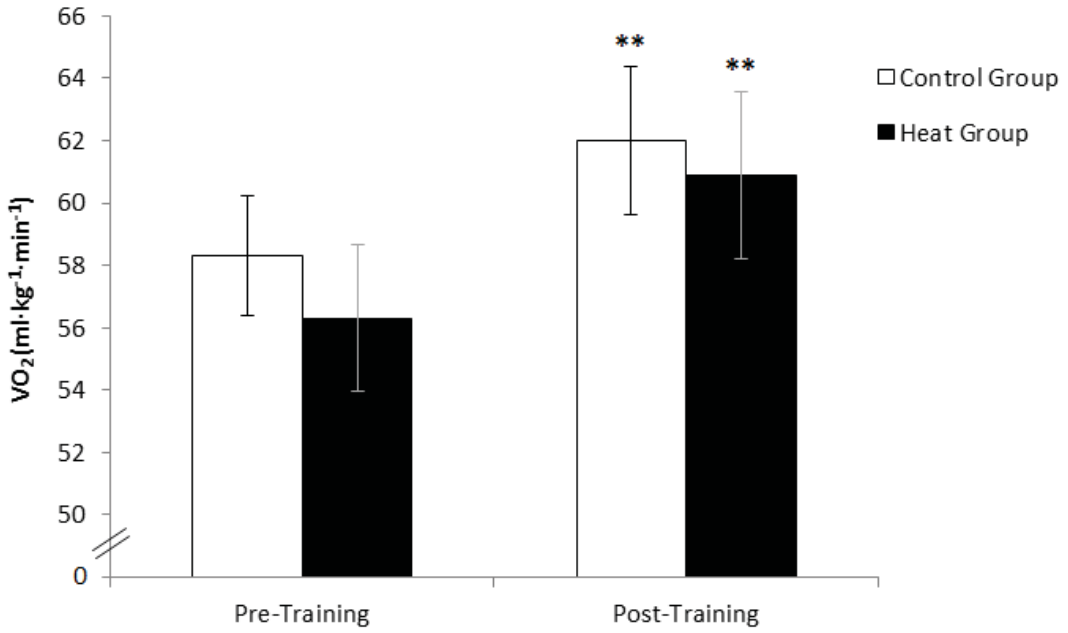

Figure 5.1. Effect of an 11-day training protocol on $\dot{V}O_2max$. Values are means ± SE for the heat group (n=9) and the control group (n=9).
** p < 0.001 vs. Pre-Training

Figure 5.1 illustrates that $\dot{V}O_2max$ performance improved with exercise in both moderate (58.3 ± 1.9 vs. 62.0 ± 2.4 mL·kg^{-1}·min^{-1}) and hot environments (56.3 ± 2.4 vs. 60.9 ± 2.7 mL·kg^{-1}·min^{-1}). ANOVA revealed a main effect of training ($p < 0.001$) with both groups significantly improving their $\dot{V}O_2max$ by (mean ± SE) 4.1 ± 0.5 mL·kg^{-1}·min^{-1} compared to their pre-training condition. The heat group had a greater percentage increase in $\dot{V}O_2max$ from to pre- to post-training, compared to the control group (7.9 ± 1.1% vs. 6.2 ± 1.3%, respectively), but this difference did not reach statistical significance ($p = 0.340$).

5.2 Submaximal Aerobic Test

Table 5.1. Percent $\dot{V}O_2max$ at the four submaximal stages during the pre- and post-training submaximal test for the heat and control groups.

	Control Group (n=9)		Heat Group (n=9)	
	% $\dot{V}O_2max$ (Pre-Training)	% $\dot{V}O_2max$ (Post-Training)**	% $\dot{V}O_2max$ (Pre-Training)	% $\dot{V}O_2max$ (Post-Training)**
6.6 ± 0.1 km·h^{-1}	40.0 ± 1.7	37.5 ± 1.8	38.0 ± 1.0	35.1 ± 1.4
7.7 ± 0.2 km·h^{-1}	54.8 ± 2.0	51.3 ± 2.6	47.7 ± 1.4	45.2 ± 1.7
8.6 ± 0.2 km·h^{-1}	62.5 ± 1.6	57.4 ± 2.6	57.9 ± 1.1	53.3 ± 1.4
9.6 ± 0.2 km·h^{-1}	70.1 ± 2.1	63.9 ± 2.9	64.2 ± 1.3	60.4 ± 1.0

Values are means ± SE. Pre-Training % $\dot{V}O_2max$ calculated from Pre-Training $\dot{V}O_2max$ values; Post-Training % $\dot{V}O_2max$ calculated from Post-Training $\dot{V}O_2max$ values.
** $p < 0.001$ vs. Pre-Training

Table 5.1 shows $\dot{V}O_2$ as a percentage of their maximum for the four submaximal stages of the protocol, both pre- and post-training. ANOVA revealed a main effect of time ($p < 0.001$) indicating that post-training, both groups were exercising at a lower percentage (3.6 ± 0.8%) of their $\dot{V}O_2max$ for the same absolute exercise intensity.

Table 5.2. Mean responses during the submaximal protocol pre- and post-training in the heat and control groups.

		Control Group (n=9)		Heat Group (n=9)	
		Pre-Training	Post-Training¥	Pre-Training	Post-Training¥
40% $\dot{V}O_2max$	HR (b·min^{-1})	116 ± 2	109 ± 3	115 ± 3	108 ± 4
	BLa (mmol·L^{-1})	1.6 ± 0.2	1.4 ± 0.1	1.8 ± 0.3	1.6 ± 0.2
	$\dot{V}O_2$ (mL·kg^{-1}·min^{-1})	23 ± 1	23 ± 1	21 ± 1	22 ± 2
50% $\dot{V}O_2max$	HR (b·min^{-1})	137 ± 3	131 ± 3	130 ± 3	125 ± 4
	BLa (mmol·L^{-1})	2.0 ± 0.2	1.5 ± 0.2 ǂ	2.2 ± 0.4	1.4 ± 0.0 ǂ
	$\dot{V}O_2$ (mL·kg^{-1}·min^{-1})	32 ± 1	32 ± 1	27 ± 2	28 ± 2
60% $\dot{V}O_2max$	HR (b·min^{-1})	149 ± 2	142 ± 3	145 ± 4	140 ± 4
	BLa (mmol·L^{-1})	2.5 ± 0.4	2.1 ± 0.3	1.6 ± 0.3	1.5 ± 0.2
	$\dot{V}O_2$ (mL·kg^{-1}·min^{-1})	36 ± 1	35 ± 1	32 ± 1	32 ± 2
70% $\dot{V}O_2max$	HR (b·min^{-1})	159 ± 3	153 ± 4	155 ± 4	149 ± 4
	BLa (mmol·L^{-1})	3.0 ± 0.6	2.9 ± 0.6	2.3 ± 0.5	1.8 ± 0.3
	$\dot{V}O_2$ (mL·kg^{-1}·min^{-1})	40 ± 1	39 ± 1	36 ± 2	37 ± 2

Values are means ± SE. HR, heart rate; BLa, blood lactate.
ǂ $p < 0.05$ vs. Pre-Training
¥ $p < 0.05$ vs. Pre-Training for HR values

Table 5.2 shows the results from the submaximal protocol for both groups pre- and post-training in moderate or hot environments. ANOVA was performed for heart rate and $\dot{V}O_2$ values, revealing a main effect of intensity ($p < 0.001$ for both factors); both heart rate and $\dot{V}O_2$ significantly increased in both groups as the intensity of exercise increased (from 40-50%, 50-60%, 60-70%). A main effect of training was revealed ($p = 0.008$) for heart rate, such that it was reduced by on average by 6 ± 2 $b \cdot min^{-1}$ for both groups post-training compared to pre-training values. BLa tended to rise as the intensity of exercise increased, but the difference between intensities was not significant ($p = 0.084$). The BLa data revealed a time*intensity interaction ($p = 0.017$), however upon post-hoc analyses, only a significant difference between pre- and post-training was identified at the intensity of 50% of $\dot{V}O_2max$.

5.3 Training Results

5.3.1 Resting and Cardiovascular Measures

Table 5.3. Mean differences between pre- and post-training for the heat and control groups.

	Control Group (n=9)		Heat Group (n=9)	
	Pre-Training	Post-Training	Pre-Training	Post-Training
Weight (kg)	84 ± 6	84 ± 6	83 ± 5	83 ± 5
Rest Heart Rate ($b \cdot min^{-1}$)	66 ± 4	62 ± 3	66 ± 2	64 ± 4
Max Heart Rate ($b \cdot min^{-1}$)	190 ± 2	191 ± 2	190 ± 2	193 ± 4
Rest SBP (mm Hg)	128 ± 4	126 ± 3	127 ± 4	124 ± 4
Rest DBP (mm Hg)	74 ± 3	71 ± 3	73 ± 2	71 ± 2

Values are means ± SE. SBP, systolic blood pressure; DBP, diastolic blood pressure.

Table 5.3 illustrates the changes in resting measures, namely cardiovascular adjustments, for the control and heat groups pre- and post-training. Both groups had a similar, small (0.5 ± 0.3 kg) weight loss associated with the 11-day training protocol, but this reduction was not significantly different to pre-training values ($p = 0.130$). Both groups observed lower resting systolic and diastolic blood pressure, along with resting heart rate post-training, but these reductions did not reach a level of significance ($p = 0.262$; $p = 0.103$; $p = 0.084$, respectively).

5.3.2 Heart Rate

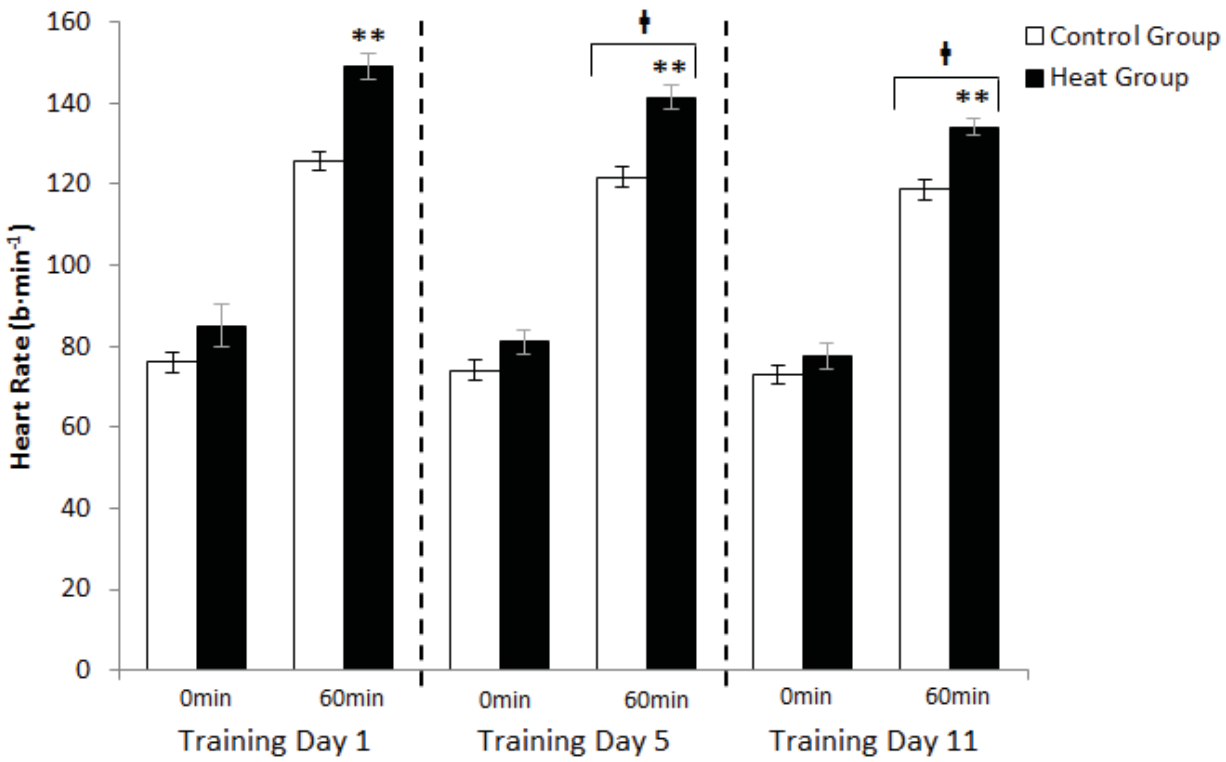

Figure 5.2. Heart rate (mean ± SE) at 0 min and 60 min of the 60 min exercise bouts on day 1, 5 and 11 of the 11-day training protocol for the heat group (n=9) and the control group (n=9).
** $p < 0.001$ vs. Control Group
ǂ $p < 0.05$ vs. Day 1

Figure 5.2 illustrates heart rate at rest (0 min) and at 60 minutes of exercise on the 1st, 5th and 11th day of the 11-day training protocol. There was no significant difference in heart rate at 0 min between the two groups on all three training days ($p = 0.094$). Main effects revealed a time ($p < 0.001$), training day ($p < 0.001$), time*group ($p = 0.009$), and time*training day ($p = 0.031$) interactions; indicating that irrespective of training day, the heat group had a significantly higher heart rate at 60 minutes by 20 ± 4 $b \cdot min^{-1}$; and heart rate at 60 minutes for both groups was significantly lower on day 5 and day 11, compared to day 1, by 6 ± 1 $b \cdot min^{-1}$ and 11 ± 1 $b \cdot min^{-1}$, respectively.

5.3.3 Core Temperature

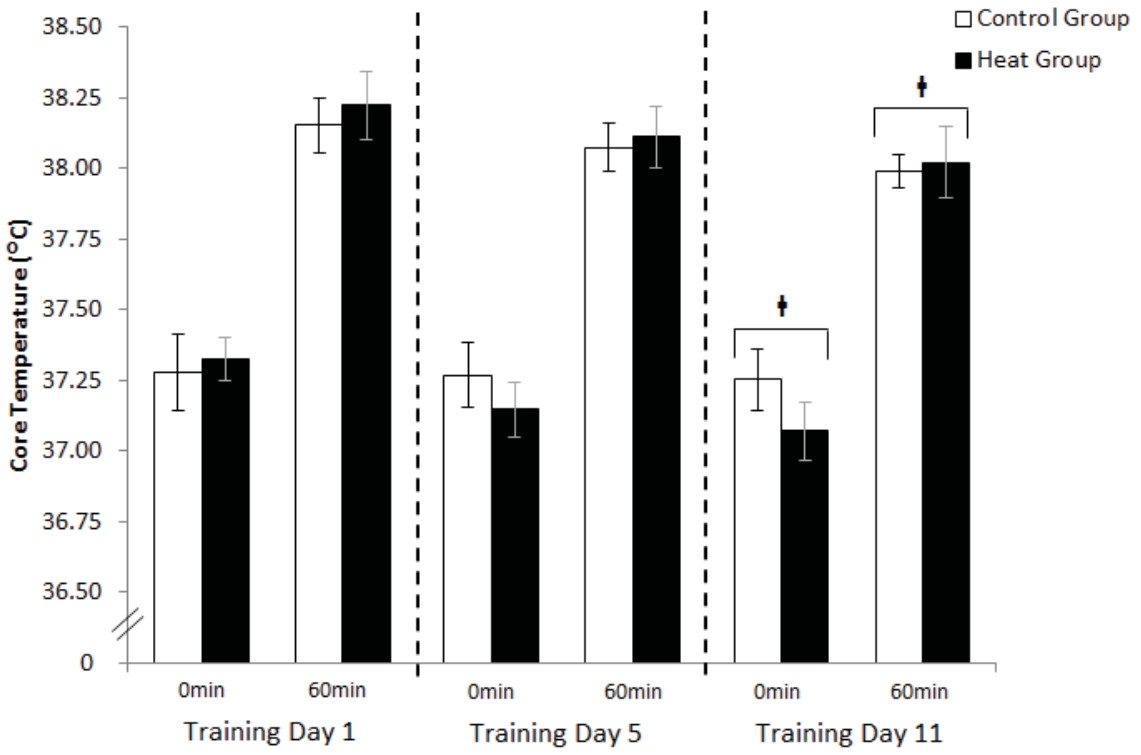

Figure 5.3. Core temperature (mean ± SE) at 0 min and 60 min of the 60 min exercise bouts on day 1, 5 and 11 of the 11-day training protocol for the heat group (n=9) and the control group (n=9).
ǂ $p < 0.05$ vs. Day 1

Figure 5.3 shows T_c at rest (0 min) and at 60 minutes of exercise on the 1st, 5th and 11th day of the 11-day training protocol. There were no significant differences in resting T_c between the two groups on day 1 of the training protocol ($p = 0.753$). ANOVA revealed main effects of time ($p < 0.001$), and training day ($p = 0.039$); with T_c significantly higher at 60 minutes than 0 min for both groups on all training days; and on day 11, T_c had significantly reduced compared to day 1 by 0.19 ± 0.05 °C, irrespective of time and group. There were no significant differences observed between the heat group and control group ($p = 0.754$).

5.3.4 Whole-body Sweat Rate

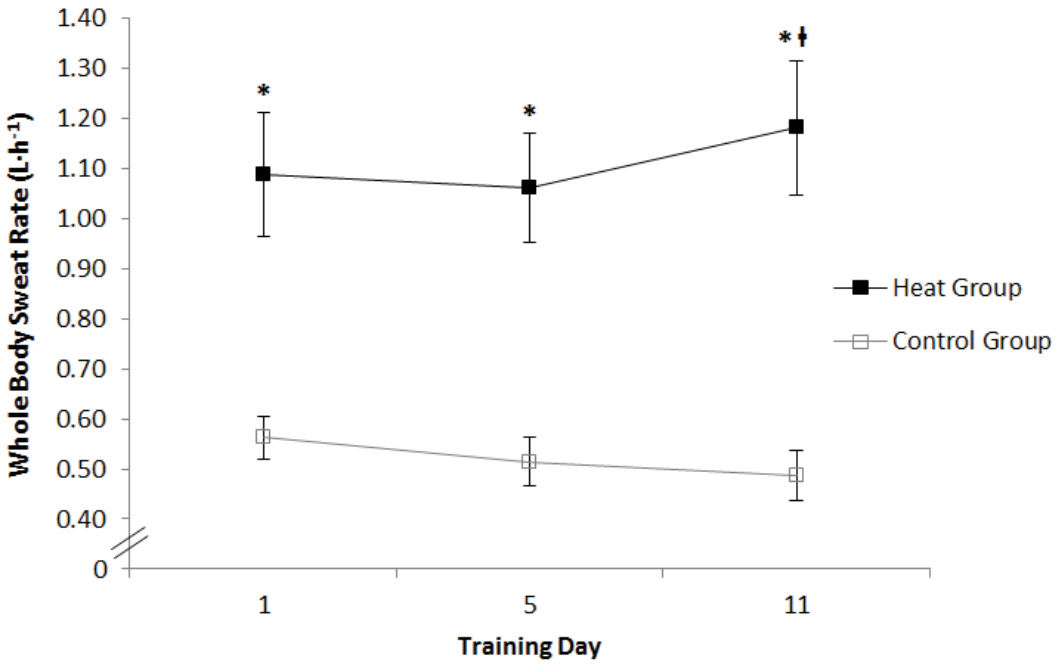

Figure 5.4. Whole body sweat rate (mean ± SE) on day 1, 5 and 11 of the 11-day training protocol for the heat group (n=9) and the control group (n=9).
* $p < 0.005$ vs. Control Group
ǂ $p < 0.05$ vs. Day 1

Figure 5.4 shows SWR on days 1, 5 and 11 of the 11-day training protocol. ANOVA revealed main effects of group ($p < 0.001$), and a group*training day ($p = 0.01$) interaction; indicating that SWR was significantly higher in the heat group (by on average 0.57 ± 0.12 $L \cdot h^{-1}$) across all three training days; and in the heat group, SWR significantly increased from day 1 to day 11 (1.05 ± 0.09 $L \cdot h^{-1}$ vs. 1.18 ± 0.10 $L \cdot h^{-1}$). There was no significant change in SWR over the training period observed in the control group ($p = 0.193$).

5.3.5 Plasma Volume Change

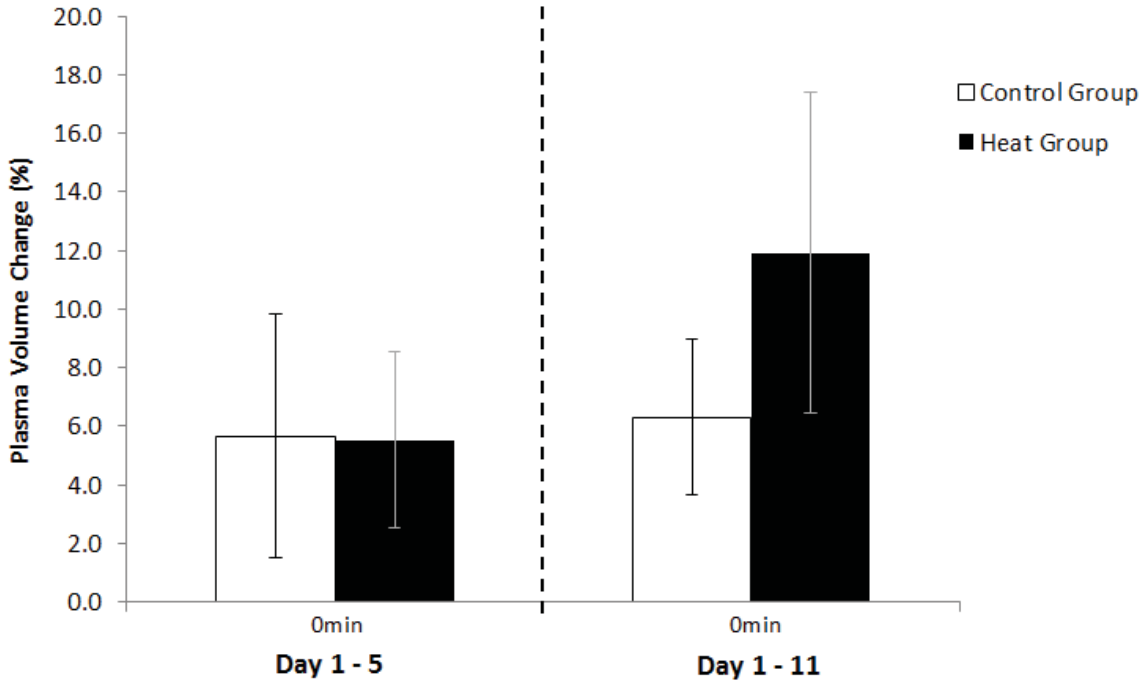

Figure 5.5. Plasma volume change (mean ± SE) at rest (0 min) between days 1 and 5, and days 1 and 11 of the training protocol for the heat group (n=9) and the control group (n=9).

Figure 5.5 illustrates the change in resting PV from day 1 to 5, and day 1 to 11 of the 11-day training protocol. Both groups observed PV expansion of ~5% from day 1 to 5, with the heat group displaying a greater degree of expansion from day 5 to 11, as opposed to the control group who only had a minor degree of expansion in the second half of the training protocol. However, ANOVA revealed no main effects, indicating that these observed mean changes in PV for both groups across the training period were not significant ($p = 0.200$).

5.3.6 Forearm Blood Flow

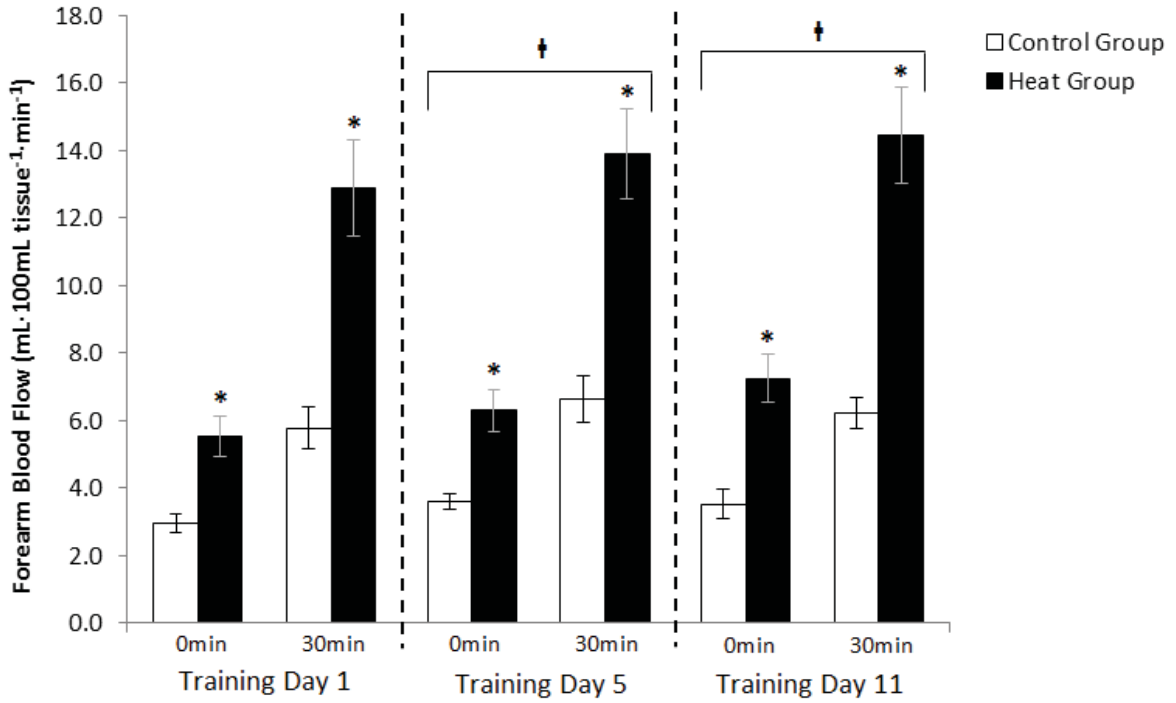

Figure 5.6. Forearm blood flow (mean ± SE) at 0 min and 30 min of the 60 minute exercise bouts on day 1, 5 and 11 of the 11-day training protocol for the heat group (n=9) and the control group (n=9).
***** $p < 0.005$ vs. Control Group
ǂ $p < 0.05$ vs. Day 1

Figure 5.6 shows forearm blood flow at rest (0 min) and exercise (30 min) on the 1st, 5th and 11th day of the 11-day training protocol. Main effects of group ($p < 0.001$), time ($p < 0.001$), time*group ($p = 0.001$), and training day ($p = 0.046$) were found. Therefore, exercise increased forearm blood flow compared to resting levels, by 5.3 ± 0.6 mL·100mL tissue^{-1}·min^{-1}, with the heat group having significantly higher forearm blood flow than the control group at both 0 min (6.2 ± 0.9 vs. 3.4 ± 0.4 mL·100mL tissue^{-1}·min^{-1}) and 60 min (13.9 ± 1.0 vs. 6.2 ± 0.4 mL·100mL tissue^{-1}·min^{-1}). Forearm blood flow, on average, increased from day 1 to 5, and day 1 to 11 for both groups by 0.8 ± 0.4 and 1.2 ± 0.5 mL·100mL tissue^{-1}·min^{-1}, respectively. The heat group appeared to have a greater increase in forearm blood flow across the 11-day training protocol, both at rest and exercise, but there was no training day*group interaction observed ($p = 0.279$).

5.3.7 Mean Skin Temperature

There was a main effect of group ($p < 0.001$), in that $\overline{T}_{sk}$ was higher in the heat group than the control for day 1 (35.6 ± 0.3 vs. 29.9 ± 0.3 °C), day 5 (35.3 ± 0.2 vs. 29.7 ± 0.2 °C), and day 11 (35.4 ± 0.3 vs. 29.1 ± 0.3 °C).

5.3.8 Blood Pressure

Systolic and diastolic blood pressure was measured at 0 min and 30 min on days 1, 5 and 11 of the 11-day training protocol (Table 5.4). Systolic blood pressure revealed a main effect of time ($p = 0.009$), significantly increasing with the onset of exercise, with no significant differences between the two groups ($p = 0.746$). Diastolic blood pressure changes over the training period revealed no main effects.

5.3.9 Haemoglobin and Haematocrit

Table 5.4 shows Hb and Hct measures taken at 0 min and 30 min on days 1, 5 and 11 of the 11-day training protocol. Analysis of Hb revealed no main effects, indicating that there were no changes to Hb concentration as a result of 11-days of exercise in either the heat or a moderate environment. There was a main effect of training day for Hct ($p = 0.028$), such that Hct decreased over the 11-day training protocol, characterised by a lower % of Hct measured in respect to plasma. Although it appears that the heat group had a greater reduction in Hct concentration than the control group, there was no group*training day interaction effect ($p = 0.103$), indicating no significant difference between the control and heat groups.

5.3.10 Local Sweat Rate

LSR was measured at 30 min and 60 min on days 1, 5 and 11 of the 11-day training protocol (Table 5.5). The heat group had a significantly higher LSR than the control group ($p = 0.003$), and ANOVA revealed a main effect of time ($p = 0.032$), in that LSR increased between 30min and 60min of exercise for both groups by (mean ± SE) 0.26 ± 0.11 $mL \cdot min^{-1} \cdot cm^{-2}$. There was no main effect of training day ($p = 0.884$), indicating that LSR did not significantly change for either group over the 11-day training period.

Table 5.4. Mean responses on day 1, 5, and 11 of the 11-day training protocol for measures taken at 0 min and 30 min in the heat and control groups.

	Control Group (n=9)						Heat Group (n=9)					
	0 min			30 min			0 min			30 min		
	Day 1	Day 5	Day 11	Day 1	Day 5	Day 11	Day 1	Day 5	Day 11	Day 1	Day 5	Day 11
SBP (mmHg)	127 ± 3	118 ± 2	124 ± 8	133 ± 5	123 ± 5	129 ± 2	128 ± 5	126 ± 4	123 ± 4	140 ± 5	134 ± 4	133 ± 3
DBP (mmHg)	73 ± 2	74 ± 2	75 ± 2	79 ± 4	79 ± 3	79 ± 2	81 ± 2	75 ± 3	76 ± 5	94 ± 6	79 ± 5	81 ± 8
Haemoglobin ($g \cdot dl^{-1}$)	14.9 ± 0.2	14.3 ± 0.3	14.2 ± 0.3	14.8 ± 0.4	14.4 ± 0.3	15.0 ± 0.3	15.9 ± 0.4	15.6 ± 0.4	15.0 ± 0.4	15.6 ± 0.3	15.4 ± 0.3	15.1 ± 0.2
Haematocrit (%)	45.5 ± 0.8	44.4 ± 0.7	45.7 ± 1.2	45.4 ± 1.0	44.7 ± 0.7	45.0 ± 1.3	47.5 ± 0.6	46.2 ± 0.7	45.5 ± 0.6	47.4 ± 0.8	46.0 ± 0.7	44.8 ± 0.7

Values are means ± SE. SBP, systolic blood pressure; DBP, diastolic blood pressure.

Table 5.5. Mean responses on day 1, 5, and 11 of the 11-day training protocol for measures taken at 30 min and 60 min in the heat and control groups.

	Control Group (n=9)						Heat Group (n=9)					
	30 min			60 min			30 min ✖			60 min ✖		
	Day 1	Day 5 ǂ	Day 11 ǂ	Day 1	Day 5 ǂ	Day 11 ǂ	Day 1	Day 5 ǂ ₴	Day 11 ǂ ₴	Day 1	Day 5 ǂ ₴	Day 11 ǂ ₴
LSR ($mL \cdot min^{-1} \cdot cm^{-2}$)	0.19 ± 0.03	0.22 ± 0.04	0.20 ± 0.04	0.27 ± 0.06	0.22 ± 0.04	0.25 ± 0.04	0.89 ± 0.15	0.85 ± 0.15	0.84 ± 0.12	1.28 ± 0.29	1.31 ± 0.24	1.38 ± 0.32
RPE	10.6 ± 0.4	10.1 ± 0.5	9.8 ± 0.5	11.2 ± 0.4	10.8 ± 0.5	10.3 ± 0.5	11.9 ± 0.5	11.2 ± 0.4	10.5 ± 0.6	12.3 ± 0.5	11.6 ± 0.5	11.2 ± 0.6
Thermal Comfort	1.6 ± 0.2	1.7 ± 0.2	1.6 ± 0.2	1.7 ± 0.2	1.7 ± 0.2	1.7 ± 0.2	2.1 ± 0.2	1.7 ± 0.3	1.8 ± 0.2	2.5 ± 0.3	2.1 ± 0.2	2.1 ± 0.2
Thermal Sensation	4.8 ± 0.3	4.9 ± 0.3	4.9 ± 0.2	5.3 ± 0.3	5.0 ± 0.2	5.0 ± 0.3	5.5 ± 0.3	5.2 ± 0.1	5.3 ± 0.1	5.5 ± 0.4	5.6 ± 0.1	5.7 ± 0.1
$\dot{V}O_2$ ($mL \cdot kg^{-1} \cdot min^{-1}$)	28.8 ± 1.2	28.1 ± 1.1	28.3 ± 1.0	28.5 ± 0.9	28.4 ± 0.9	28.4 ± 1.0	28.2 ± 1.2	28.2 ± 1.5	26.7 ± 1.5 ¥	29.0 ± 1.3	28.5 ± 1.4	28.0 ± 1.6
RER	0.84 ± 0.03	0.82 ± 0.02	0.81 ± 0.02	0.83 ± 0.03	0.81 ± 0.02	0.79 ± 0.02	0.84 ± 0.01	0.85 ± 0.02	0.82 ± 0.01	0.85 ± 0.02	0.83 ± 0.02	0.82 ± 0.01
Metabolic Rate ($kcal \cdot min^{-1}$)	11.3 ± 0.5	11.0 ± 0.5	11.3 ± 0.4	11.3 ± 0.5	11.2 ± 0.5	11.4 ± 0.4	11.3 ± 0.6	11.1 ± 0.6	10.4 ± 0.5	11.3 ± 0.7	11.2 ± 0.6	10.9 ± 0.5

Values are means ± SE. LSR, local sweat rate; RPE, rating of perceived exertion; RER, respiratory exchange ratio.
✖ $p < 0.05$ vs. Control Group for LSR and TS values
ǂ $p < 0.001$ vs. Day 1 for RPE values
₴ $p < 0.05$ vs. Day 1 for ThC values
¥ $p < 0.05$ vs. Day 5
+++

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5.3.11 Rating of Perceived Exertion

Table 5.5 shows RPE scores taken at 30 min and 60 min of exercise on the 1st, 5th and 11th day of the 11-day training protocol. Main effects of time ($p = 0.05$) and training day ($p < 0.001$) were revealed, such that RPE scores were significantly higher at 60 mins than at 30 min of exercise, for both groups; and on average (for both groups) ratings of perceived exertion were reduced from day 1 to 5 by 0.6 ± 0.2, and from day 1 to 11 by 1.1 ± 0.2 on the 15-point scale.

5.3.12 Thermal Comfort

The ThC scores recorded at 30 min and 60 min on day 1, 5 and 11 of the 11-day training protocol (Table 5.5) revealed a main effect of time ($p = 0.018$) and a group*training day interaction ($p = 0.024$). Therefore, ThC significantly decreased from 30 min to 60 min of exercise, for both groups; whereas ThC significantly increased over the 11-day training period for the heat group (irrespective of whether it was 30 min or 60 min). This was illustrated by a reduction in ThC scores from day 1 to 5 by 0.4 ± 0.1, and day 1 to 11 by 0.3 ± 0.1. ThC appears to have improved by day 5 of training in the heat, and is sustained through to day 11 of heat training.

5.3.13 Thermal Sensation

Table 5.5 illustrates the ThS scores taken at 30 min and 60 min on day 1, 5 and 11 of the 11-day training protocol. The heat group had, on average, a significantly higher ThS score than the control group (5.5 ± 0.2 vs. 4.9 ± 0.2) ($p = 0.043$), illustrating a greater feeling of heat/hotness than the control group.

5.3.14 $\dot{V}O_2$

$\dot{V}O_2$ measured at 30 min and 60 min on days 1, 5 and 11 of the 11-day training protocol (Table 5.5). There was no significant difference in $\dot{V}O_2$ between the heat and control group for $\dot{V}O_2$ at either 30 min or 60 min of exercise ($p = 0.700$). ANOVA revealed a time*training day*group interaction effect ($p = 0.003$), indicating a significant reduction in $\dot{V}O_2$ at 30 min of exercise for the heat group between training day 5 and training day 11.

5.3.15 Respiratory Exchange Ratio

Table 5.5 illustrates RER values calculated from respiratory gas analysis taken on day 1, 5 and 11 of the 11-day training protocol. There was no significant difference in RER values between the heat group and the control group ($p = 0.149$), and ANOVA revealed no main effects, indicating that the training period, either in the heat or a moderate environment had no impact on RER.

5.3.16 Metabolic Rate

Table 5.5 illustrates metabolic rate values calculated from $\dot{V}O_2$ and RER data taken on day 1, 5 and 11 of the 11-day training protocol. There was no significant difference in metabolic rate values between the heat group and control group ($p = 0.161$), and ANOVA revealed no main effects, indicating that the training period, either in the heat or a moderate environment had no impact on metabolic rate.

5.3.17 Correlations

Table 5.6 Correlations between % change in $\dot{V}O_2$max from pre- to post-training and alterations in heart rate, rectal temperature, plasma volume and forearm blood flow as a result of the 11-day training protocol

			$\dot{V}O_2$max % Change (Pre-Post Training)	
			Pearson Correlation	Sig (2-tailed)
% Change from Training Day 1 - 11	Rest	Heart rate	-0.025	0.923
		Rectal Temperature	0.171	0.504
		Plasma Volume	0.086	0.742
		Forearm Blood flow	-0.251	0.315
	Exercise	Heart Rate	-0.191	0.447
		Forearm Blood Flow	-0.036	0.886

Sig**,** significance.

Correlations for the training induced percent changes in the main variables (heart rate, rectal temperature, plasma volume, and forearm blood flow) were run against the main dependent variable - percent change in $\dot{V}O_2$ max from pre- to post- training. The correlation revealed no main correlations between any of the main training induced physiological changes and the changes to $\dot{V}O_2$ max performance.

Chapter Six: Discussion

This study was undertaken primarily to test the hypothesis that exercise, with or without heat stress will improve maximal aerobic capacity; further, that exercise in combination with heat stress will significantly enhance aerobic performance to a greater extent than exercise alone. The results confirmed the first half of the primary hypothesis, as both the control group and heat group improved their maximal aerobic capacity upon completion of the 11-day training protocol, although the second half of the hypothesis was rejected with no additional improvement observed as a result of the additional heat stress. A number of secondary hypotheses were tested: 1) exercise in combination with heat stress will significantly improve aerobic efficiency to a greater extent than exercise alone; 2) exercise in combination with heat stress will significantly enhance resting cardiovascular measures greater than exercise alone; 3) exercise in combination with heat stress will induce heat acclimation. Whilst exercise in combination with heat stress appeared to induce heat acclimation, the additional heat stress did not improve submaximal efficiency, or resting cardiovascular measures to a greater extent than exercise alone. These findings will be discussed in more detail below.

6.1 The impact of training and heat stress on maximal and sub-maximal aerobic performance

Additional heat stress did not improve $\dot{V}O_2$max performance above exercise alone.

The main hypothesis of this study was that 'exercise, with or without additional heat stress will significantly improve maximal aerobic capacity'. This is supported by the current findings; that an 11-day training protocol consisting of 60 minutes of walking on a treadmill at an intensity of 50% $\dot{V}O_2$max in both hot (35°C) and moderate (18°C) environments improved maximal aerobic capacity in a moderate environment by at least 6%. Whilst the heat group appeared to have greater aerobic performance improvements compared to the control group (8% vs. 6%), there was no significant statistical difference. Therefore, the second component of the main hypothesis that 'exercise in combination with added heat stress will significantly enhance performance to a greater extent than exercise alone' cannot be supported.

The finding of improved maximal aerobic performance in a moderate environment from heat stress is in agreement with the previous literature (Shvartz *et al.*, 1977; Sawka *et al.*, 1985; Lorenzo *et al.*,

2010; Buchheit *et al.*, 2011; Buchheit *et al.*, 2013). This specific finding also illustrates the importance of implementing a control group; previous studies have concluded that the improved performance in a cool/moderate environment was due to the additional heat stress and the associated physiological adaptations (Sawka *et al.*, 1985; Buchheit *et al.*, 2013). Whilst other studies indicated that undertaking a randomised controlled experimental design would provide a more accurate method to determining whether a cause-effect relationship actually exists between the addition of heat stress and the improved aerobic performance in a moderate environment (Buchheit *et al.*, 2011). As a result, the current study was able to confirm through implementation of a matched control group that the performance improvements (observed in both groups) may be associated with the exercise itself, rather than the additional heat stress. From the current literature only one other study (Lorenzo *et al.*, 2010) has employed a matched control group instead of using the same participants in a randomised cross-over design. Interestingly, these investigators did not detect an improved aerobic performance from the control group, although the training intensity was identical to the current study. It must be noted that there are methodological differences between the current study and that by Lorenzo *et al.* (2010); for example, they used cycling as the exercise modality, in comparison to walking/running in the current study. Additionally, highly trained cyclists were recruited that had a mean $\dot{V}O_2max$ of 66.9 $mL \cdot kg^{-1} \cdot min^{-1}$ compared to 56.8 $mL \cdot kg^{-1} \cdot min^{-1}$, with a greater range of aerobic fitness levels (from 47.6 to 71.4 $mL \cdot kg^{-1} \cdot min^{-1}$) in the current study. Having a large variation in basal aerobic fitness levels could be classed as a limitation of the current study, however, it could also be a reason for the variation in results observed between the current study and that by Lorenzo *et al.* (2010). When designing a training programme for optimal cardiovascular fitness gains, the initial fitness of the individual is one of the main factors to take into consideration (Shephard, 1968). Pollock *et al.* (1998) state that for an aerobically unfit individual (i.e. $\dot{V}O_2max$ <45 $mL \cdot kg^{-1} \cdot min^{-1}$), an exercise intensity of 40-50% of $\dot{V}O_2max$/heart rate reserve is sufficient to induce cardiovascular fitness benefits; the intensity required for such improvements for an individual commencing with a greater base level of aerobic fitness will be higher (>50% $\dot{V}O_2max$). Lorenzo *et al.* (2010) employed an intensity of 50% $\dot{V}O_2max$, which is likely too low for the trained cyclists to induce cardiovascular benefits. However, the hot environment produced additional cardiovascular stress, resulting in a higher relative work load, capable of inducing improvements in aerobic capacity.

Exercise, irrespective of whether in a hot or moderate *environment, improved submaximal aerobic efficiency, illustrated by lowered submaximal heart rate and relative* $\dot{V}O_2$*, but had no effect on blood lactate or absolute* $\dot{V}O_2$*.*

The exercise period led to a reduction in relative $\dot{V}O_2$ at all four stages of the submaximal protocol, but did not change absolute O_2 uptake. This finding was to be expected, as both groups had higher $\dot{V}O_2$max values following the 11-day training protocol, and whilst the absolute O_2 cost for the same treadmill speed was equal, the participants were now working at a lower percentage of their elevated $\dot{V}O_2$max. There was no significant difference observed between the control and heat group which is not in agreement with the findings associated with a number of heat acclimation studies. Various authors have stated that a heat adapted individual presents an increased metabolic efficiency, and a reduced O_2 demand for the same exercise intensity, as illustrated by a lower $\dot{V}O_2$ post-training (Shvartz *et al.*, 1972; Sawka *et al.*, 1983b; Young *et al.*, 1985; Houmard *et al.*, 1990). In the current study, there were no alterations in $\dot{V}O_2$ during the training, regardless of the addition of heat stress; therefore changes in absolute O_2 uptake during the four submaximal stages would not be expected.

The 11-day training protocol, both in hot and moderate environments, led to reductions in exercising heart rate at all four stages of the submaximal protocol. There was no significant difference observed between the control group and the heat group, indicating that the reduction in heart rate is likely a result of the exercise itself, rather than the additional heat stress. Whilst lowered exercising heart rate is one of the characteristic adaptations achieved and reported in prior heat acclimation studies and reviews (Wyndham *et al.*, 1968; Senay *et al.*, 1976; Sawka *et al.*, 1983a; Garrett *et al.*, 2009), it has often been associated with PV expansion; however, various participants in the current heat group did not experience any PV expansion (discussed further in section 6.2).

Mean BLa levels were lower at all submaximal exercise intensities post-training for both groups, but the difference only reached statistical significance at 50% of $\dot{V}O_2$max. Previous research states that aerobic training alone (Hurley *et al.*, 1984), as well as heat acclimation induces reductions in BLa concentrations for the same exercise intensity (Young *et al.*, 1985; Febbraio *et al.*, 1994). Whilst the data indicates that a significant reduction in BLa occurs in both groups, it only exists at 50% of $\dot{V}O_2$max. However, the remaining data does not completely negate the previous research, as there appears to be a trend (although non-significant) for lower levels of BLa after a period of training, irrespective of whether the exercise was completed in a hot, or moderate environment.

The mechanisms behind the maximal and submaximal aerobic performance improvements observed in the current study have been directly linked to the physiological adaptations that are obtained

from both exercise and additional heat stress (Shvartz *et al.*, 1972; Sawka *et al.*, 1985; Lorenzo *et al.*, 2010). Heat acclimation induces various physiological adaptations that enable the body to better withstand any further exposure to the hot environment, and these include reduced heart rate and T_c, expanded PV, increased sweat rate and sweat sensitivity (Armstrong & Maresh, 1991). Whilst these adaptations are characteristic of a fully heat acclimated individual, they are not all exclusively limited to exogenously-applied heat adaption, as endurance exercise itself can elicit a number of similar physiological responses; for instance PV and cardiovascular alterations can improve temperature regulation and enable an individual to be in a state of 'partial acclimation' (Pandolf *et al.*, 1977; Taylor & Cotter, 2006).

6.2 Physiological responses during the 11-day training protocol

Exercise, with or without additional heat stress, lowered exercising heart rate, although no significant expansion of plasma volume was observed, on average.

Heart rate, measured at 60 minutes of exercise during the 11-day training protocol was significantly lower by day 5, and even further-so by day 11, when compared to day 1. This is illustrated in Figure 5.2, and while there was no apparent statistical significant difference between the two groups, the heat group appeared to have a greater reduction in heart rate over the training protocol than the control group. This finding from the current study concurs with previous literature, as both heat acclimation and physical activity alone have been reported to induce reductions in heart rate (Armstrong & Maresh, 1991; Taylor & Cotter, 2006). Exercise independently would be expected to reduce heart rate over time, as endurance exercise leads to myocardial alterations that improve contractile pumping and filling properties (Blomqvist & Saltin, 1983). Heat acclimation is thought to have a further effect on the heart rate response through the heat-induced expansion of PV and a concurrent increase in stroke volume; although the extent of this association has not reached a consensus (Garrett *et al.*, 2009). Early work by Convertino *et al.* (1980) set out to define the contributions of an exercise-induced T_c rise, and sedentary heat-induced T_c rise to PV expansion, where they concluded that 40% of PV expansion was initiated by simply a T_c rise, whilst the remaining 60% was indicative of exercise-induced factors. Considering that the heat group in the current study was completing exercise in combination with heat exposure, the larger PV expansion by day 11 compared to the control group (Figure 5.5), was to be expected. Both groups had a mean PV expansion of at least 6%, but due to the large variation in levels of expansion amongst the participants, these changes did not reach statistical significance for either group. By training day 11, three participants from the heat group had not experienced any PV expansion, although all three

had considerable reductions in exercising heart rate, by at least 7 (and up to 16) b·min^{-1}. It seems unlikely that a reduction in exercising heart rate of 16 b·min^{-1} is primarily a result of the exercise itself, considering the mean reduction in heart rate by training day 11 for the control group was only 7 b·min^{-1}. This finding contradicts the proposed link between heat-induced PV expansion and associated reductions in heart rate. Horowitz *et al.* (1986) and Levi *et al.* (1993) worked with rats during heat acclimation, and discovered that the heat adaptations improved central mechanisms and enhanced cardiac mechanics of the heart. Unfortunately, research of this nature has not progressed to humans, but it is likely that PV expansion is not the only mechanism responsible for heat acclimation induced alterations in heart rate.

Exercise with additional heat stress increased whole body sweat rate.

Both physical activity and heat acclimation have the potential to improve sudomotor functioning, which is often (such as this case) characterised by an elevated sweat rate (Fox *et al.*, 1964; Shvartz *et al.*, 1979; Candas *et al.*, 1983). The heat group had an overall significant increase in SWR from training day 1 to training day 11. The onset/initiation of sweating is associated with thermal inputs/feedback from both skin and core temperatures; heat acclimation has been associated with a lowered T_c threshold for the onset of sweating during an exercise bout in the heat (Nadel *et al.*, 1974; Shvartz *et al.*, 1979; Nielsen *et al.*, 1993). In the current study, the timing/T_c at the onset of sweating was not measured, but this mechanism, in combination with the proposed increased size and secretion of a heat adapted individual's sweat gland is likely to be linked to the augmented SWR observed (Peter & Wyndham, 1966; Sato & Sato, 1983; Sato *et al.*, 1990). Not only does heat acclimation have the ability to improve the sweat rate, the excreted sweat is also more dilute, conserving greater amounts of sodium and chloride (Allan *et al.*, 1971; Taylor, 2000). Whilst this was not measured in the current study, it is likely that the heat group developed this adaptation, concurrently with their elevated sweat rate. An increase in SWR is a positive adaptation associated with heat acclimation, helping to reduce the thermoregulatory strain of a hot environment via improved evaporative heat loss.

Exercise, with or without additional heat stress, increased forearm blood flow.

The findings that both the control group and heat group have elevated forearm blood flow on training days 5 and 11, compared to 1, supports previous research concluding that both endurance exercise and heat acclimation have the ability to increase the cutaneous vasodilatory response for a given T_c (Roberts *et al.*, 1977). At rest, the heat group had a noticeable increase in forearm blood

flow across the training protocol. This would be expected, as the additional heat stress poses a thermoregulatory challenge (even at rest), whereas the control group prior to exercise in the moderate environment had very minimal levels of cutaneous vasodilation. Thus, any exercise-induced vasodilatory enhancements would not be expected to become apparent until they begin exercising and generating excess heat. Forearm blood flow is higher for the same intensity of exercise in the current study, enabling improved evaporative heat loss, and in a moderate/cool environment, also convective and radiative heat loss.

Exercise, with or without additional heat stress, reduced resting and exercising core temperature.

A reduction in core body temperature is an adaptation that arises from both endurance exercise and heat acclimation – the combination of exercise in a hot environment facilitating the greatest reductions (Armstrong & Maresh, 1991). By the last day of the training protocol, both the control group and the heat group had experienced reductions in resting and exercising T_c; although there was no significant difference between the two, the heat group appeared to have a larger decrease both at rest, and during exercise. A lowered T_c is indicative of reduced thermoregulatory strain for the same intensity/duration of exercise; a greater amount of heat must be dissipated from the body to reduce the heat storage (Parsons, 2002). Collectively, the physiological adaptations described above, obtained by the heat and/or control group, enable the body to minimise the amount of heat stored, reducing the T_c at all stages of the exercise bout – illustrating a reduced thermoregulatory strain and helping to enable an individual to exercise for longer or harder than they previously would have been able to withstand.

Perceived exertion reduced over the 11-day training protocol, with and without heat stress; additional heat stress also led to a concurrent improvement in thermal comfort.

Both the control group and the heat group had significantly lower ratings of perceived exertion on training day 5 and 11, compared to training day 1, for the same intensity of exercise. This finding is in agreement with the previous literature highlighting that reduced perceived exertion is prevalent with heat acclimation (Armstrong & Maresh, 1991; Aoyagi *et al.*, 1997) and improved cardiovascular fitness (Ekblom & Golobarg, 1971; Patton *et al.*, 1977). The reduced perceived exertion is indicative of the ideas discussed above and that both groups progressively found the exercise to have less cardiovascular strain, with the heat group experiencing less of a burden from the hot environment. Adaptations arising from the development of heat acclimation such as the increase in SWR and the concurrent lowered T_c are likely to have played a role in the observed increased ThC during the training protocol in the heat group – indicative of reduced thermal strain.

6.3 The impact of exercise and additional heat stress on resting cardiovascular adjustments

Exercise, with or without additional heat stress, reduced resting heart rate, and resting blood pressure measures, though none reached statistical significance.

In general, the research surrounding heat acclimation has mainly focussed on the physiological adaptations that arise and how these impact further exercise or performance (namely in hot environments). The physiological adaptations developed are positive changes that would be expected to benefit performance and improve resting cardiovascular measures. Whilst the results from our study did not reach statistical significance, both the exercise, and the additional heat stress appear to have positive impacts on cardiovascular health – reducing resting heart rate, as well as reducing both systolic and diastolic blood pressure measures. Although the mentioned measures were taken when the participants were in a rested state (having been seated for 15 minutes), the measures were taken prior to the maximal aerobic capacity test. In anticipation to this test, participants may have been anxious, misrepresenting their resting heart rate and blood pressure values. This possibly needs to be taken into consideration when observing the 'resting' measures, as it may have played a part in the lack of significance observed amongst the results.

6.4 Considerations/Limitations

This study design was a non-crossover, controlled investigation whereby eighteen moderately active males were randomly assigned to either a control group or a heat (experimental) group. The study was designed with reference to the paper by Lorenzo *et al.* (2010), who investigated heat acclimation and its associated benefits using a randomised, matched control group trial. The groups in the current study were successfully matched for basal fitness level, age, height, weight, BSA, and percent body fat – the successful matching of the two groups ensured that the results of the current study have been presented with confidence, and that any difference between the two groups was due to the additional heat stress. However, within each group there was a large range of basal levels of fitness. This may have contributed to the degree of acclimation that individuals in the heat group obtained – as the aerobically fit participants would have come into the study already in a state of 'partial acclimation', resulting in a faster rate of development of full acclimation. Even so, because the range was matched in both groups, this is unlikely to have affected the final outcome; although a closer range of basal fitness levels may have produced more powerful training results.

Whilst eighteen participants was the maximum number manageable within the time constraints, having a larger sample size may have allowed more relationships/trends to emerge from the data. If time wasn't a restraint, at least two, even three more groups could have been added to the existing control and heat – one group spending 60 minutes in the hot environment, in a sedentary state; another completing no exercise nor heat exposure altogether over the 11-day protocol; the third completing the 11-day protocol in the heat at an intensity that matches the cardiovascular strain of the group exercising in the moderate environment. These additional experimental groups would help to define what contribution the additional heat stress has on any performance improvements.

On average there was a four day delay between the end of the heat acclimation protocol and the post-training maximal and submaximal tests. On review, a smaller gap between the two would have helped to eliminate the doubt that one day of heat acclimation associated physiological adaptations may have been lost with a break this size.

The decision was made through pilot research that 35°C, combined with an average RH of 45% was sufficient to bring about the appropriate changes in T_c. In hindsight, it may have been beneficial to utilise a hotter environment, as most of the participants did not experience a T_c rise during training to the extent of those involved in the pilot research.

Various measurements, including SkBF, core body temperature, and sweat rate, have multiple methods of determination depending of what tools are available and/or suitable for each situation. In the current study the decision was made to use VOP to determine forearm blood flow, the technical absorbent method for LSR, and rectal temperature as a measure of T_c. Whilst it has been recognised that there are techniques to measure SkBF, T_c, and LSR that are both continuous and more sensitive than those employed in the current study (i.e. Laser Doppler for SkBF, ventilated capsule for LSR, oesophageal temperature for T_c) (Cork *et al.*, 1983; Johnson *et al.*, 1984; Morris *et al.*, 2013); the tools were either not available (Laser Doppler and ventilated capsule), or not appropriate for the current study (oesophageal temperature would have limited timing and quantity of liquid consumption, which may have jeopardised participant recruitment and compliance).

Lastly, in addition to the maximal and submaximal tests that were employed at the beginning and end of the training protocol, a time trial may have been a useful addition to measure performance. Whilst a $\dot{V}O_2$max test is an accurate method to obtain maximal aerobic capacity, a time trial whereby the participants were advised to complete a set distance (i.e. 5 km) as fast as possible, would provide a more practical and applicable approach to fitness.

6.5 Future research

The investigation into the impact that heat acclimation can have on performance in moderate/cool environments requires further research before any conclusions can be made. Given the appropriate levels of time and/or funding, future researchers should aim to employ two-three additional testing groups; this will enable the clarification of the contributions of exercise and the additional heat stress to any improvements noted in aerobic performance.

Considering the lack of well-controlled studies that have been completed it is recommended that differing fitness levels and sexes should be included in further work. Both the current study, and the majority of the previous research studies have recruited males as their participants, so future research could focus on the impact of heat acclimation on performance in females, and also whether the menstrual cycle has any effect on subsequent performance in moderate/cool environments would be worthy of consideration.

Short-term heat acclimation has been shown to be effective in improving performance in hot environments (Garrett *et al.*, 2011), so the application of a short-term protocol (<7 days), would be interesting to observe whether the physiological adaptations gained over the short period would be sufficient for improving performance in moderate/cool environments.

Finally, research surrounding the optimal combination of heat stimulus and intensity/duration of exercise would be beneficial to aiding optimal performance improvements from a heat acclimation protocol. The combination will likely vary for differing fitness levels and/or sex, but would provide invaluable information to athletes/individuals hoping to utilise this type of training as a means to improving their performance in moderate/cool environments.

Chapter Seven: Conclusions

The purpose of this study was to determine what impact additional heat stress has on resting cardiovascular measures and aerobic capacity in a moderate environment. In regards to the hypotheses outlined in **Chapter Three** it can be concluded that:

- Exercise with additional heat stress does not induce any further significant improvements in maximal aerobic capacity over and above those induced by the exercise alone.
- Training at an exercise intensity of 50% $\dot{V}O_2max$ for 11 days can improve maximal aerobic performance by at least 6% in moderately active males ($\dot{V}O_2$ ~56 $mL \cdot kg^{-1} \cdot min^{-1}$).
- Both exercise, and exercise in combination with additional heat stress reduce the cardiovascular strain and relative O_2 uptake of submaximal exercise to the same extent, although neither appear to improve BLa characteristics.
- Exercise, with and without additional heat stress, has the potential to improve resting cardiovascular measures to the same extent, illustrated by lower resting heart rate and blood pressure after training. Although, the reductions did not reach statistical significance in the current study.
- Eleven days of exercise in 35°C can induce heat acclimation, illustrated through an increase in SWR, reduction in T_c and exercising heart rate, and a reduction in RPE.

The results from this study illustrate that a moderately active individual can significantly improve their level of aerobic fitness through daily exercise at an intensity as low as 50% of their $\dot{V}O_2max$, irrespective of the type of environment. Completion of daily exercise at this intensity in a hot environment will develop positive physiological adaptations associated with heat acclimation which will help to reduce thermoregulatory strain during future exercise both in the heat, and in a moderate environment. The results produced can be applied to individuals hoping to improve their basal fitness level, as well as sports teams and athletes who may be preparing for sporting events in a hot environment. In combination with future research, the results from this study can potentially begin to set recommendations of heat acclimation training for improving performance in a moderate environment, for both athletes and moderately active individuals.

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Appendices

Participant Information Sheet

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PARTICIPANT INFORMATON SHEET

Project Title: Adaptation to exercise for maximal aerobic capacity, submaximal aerobic efficiency, and cardiovascular adjustments: does addition of heat stress induce greater improvements than exercise alone?

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You have been invited to participate in a study evaluating the effect of the addition of heat stress to exercise and the potential performance and health benefits that arise from this exercise adaptation. Participation in this study is on a voluntary basis and you have the right to pull out, or ask questions at any time. This study forms part of Melissa Black's MSc qualification.

Why are we doing this study?

Exercising in hot environments often feels harder, and puts a greater amount of strain on the body than exercise in cooler temperatures. The extra strain caused by the heat has been utilised and explained extensively in the previous literature, by which training in the heat (heat acclimation) has been shown to improve exercise performance in hot conditions. It appears that the effect that heat acclimation can have on exercise performance in temperate conditions, rather than hot, has been relatively overlooked in the literature. The extra strain whilst exercising in the heat may not only induce performance benefits in temperate conditions, it may pose benefits to overall cardiovascular

health also. The information obtained will enable a better understanding of the effect of heat acclimation on exercise capacity in temperate conditions, and positive adaptions to overall cardiovascular health. This study will potentially give an insight into a new method of training for individuals to see greater improvements in their fitness levels in a shorter length of time, whilst posing health benefits at the same time.

What is the aim of this study?

The aim of the project is to evaluate the effects of an 11-day heat acclimation protocol on running performance in temperate conditions and health adaptations that may arise.

If I agree to take part, what will I be asked to do?

If you agree to participate and meet our inclusion criteria you will be asked to come into the laboratory on a number of occasions. The sessions will take place under supervision in the Human Performance Laboratory (HPL), School of Sport and Exercise at Massey University and in total will take approximately 20 hours of your time.

Preliminary Visits:
You will have had this Information Sheet for at least one week, and we will answer any questions you may have about any of the experimental procedures/sessions. You will be provided with a food and activity diary which will require you to fill in your food/beverage intake for the first two preliminary visits and your physical activity throughout the entire study. During the first session we will ask for your informed, written, consent, which will be followed by a continuous incremental exercise test on a treadmill to determine peak aerobic capacity. You will be informed of what group you have been randomly selected into (either hot or temperate). In the subsequent session, you will be familiarised with the equipment used and the intensity and modality of exercise you will encounter and complete a short sub-maximal protocol.

Heat Acclimation Protocol:
Over a period of 12 days you will visit the laboratory eleven times (five consecutive days, one day rest, six consecutive days) where nude body weight will be obtained before completing 60mins of walking at 50% VO_2 max on the treadmill in either 35°C (hot group) or 18°C (temperate group). On day 1, 5 and 10 of the acclimation protocol we will take blood samples (through a finger prick), forearm blood flow and blood pressure before you begin exercising and immediately after. On these days samples of your expired air will be collected throughout, along with skin temperature which will be recorded by thermal iButtons attached by adhesive tape to the skin. Core temperature will be monitored throughout every session via rectal thermistors. Blood samples will be used for the determination of plasma volume and will not be stored. You will be provided with a carbohydrate/electrolyte drink throughout the sessions at 15 minute intervals, and any extra water required with be provided upon your request.

Post Acclimation Measures:
Within a week of completion of the heat acclimation protocol, you will be asked to visit the laboratory to complete the incremental exercise test on a treadmill to determine peak aerobic capacity as well as the sub-maximal protocol.

Following this test, if you agree to take part, visitation to the laboratory every 3-4 days (morning) for two weeks (total of 4 visits) would allow resting health measures to be taken – heart rate, blood pressure and plasma volume (via a finger prick blood sample). Breakfast will be provided for you in these sessions.

What are the risks?

Exercise:
You are likely to experience the fatigue associated with strenuous exercise, particularly during the peak aerobic capacity test. Nevertheless, as in any physical activity, there is a very small possibility of injuries that include, but are not restricted to, muscle, ligament or tendon damage, breathing irregularities and dizziness. However, all protocols are commonly performed in exercise physiology laboratories and potential risks to participants have been minimised.

Rectal Temperature:
When measuring rectal temperature there is a risk, however rare, of rectal perforation. To minimize this risk the rectal temperature thermistor is flexible which will allow for freedom of movement with limited discomfort. The thermistor itself has a protective vinyl covering to prevent wires from possibly tearing the colon. Prior to self-insertion the rectal thermistor will be coated with petroleum jelly which will not only act as a lubricant but will also be another means of protecting the inside of the colon. The rectal thermistor is disposable and therefore every thermistor will be sterile prior to insertion. You should feel no sensation of pain during insertion and removal of the rectal thermistor. Additionally, to ensure that discomfort is minimized, insertion of the rectal temperature probe will not take place if: 1) you have hemorrhoids, 2) you have recently under gone rectal surgery, or 3) you have diarrhea.

The guidelines for safe and successful insertion/removal of the rectal temperature thermistor are as follows:
- Wear sterile gloves to prevent any contamination with equipment and for personal hygiene.
- Lubricate the thermistor and the anus using the lubricant (petroleum jelly) provided.
- Insert thermistor slowly and carefully until the marked point (10cm) of the thermistor is reached.
- After each trial, remove thermistor by pulling the thermistor out slowly and carefully.

You will be provided with the rectal thermistor and lubricant for self-insertion prior to each session in the privacy of the laboratory changing rooms where you can void prior to insertion.

Heat Illness:
Core body temperature (as indicated by rectal temperature) is the primary variable that will be monitored continuously throughout the exercise bouts. Core body temperature is the primary means to differentiate between heat exhaustion (non-life threatening) and heat stroke (a potentially life threatening condition). Heat stroke occurs when core temperature exceeds 40°C and as such exercise will be discontinued if core temperature reaches 39.5°C, thus preventing it from rising to potentially dangerous levels. However, should this occur you will be cooled (via cold rags, a fan, etc.), be given fluids and will be monitored for the signs and symptoms of heat stroke. You will be provided with water throughout the trials to avoid dehydration, thereby lowering the chance of developing heat illness; we also recommended that you restrict your caffeine and alcohol intake to small/moderate amounts to help avoid dehydration.

What are the benefits?

For the time you invest in this study, you will receive a $100 voucher; this will cover your transport, diet and inconvenience costs due to taking part in this study. If you do not complete the study this will be paid on a pro-rata basis (i.e. payment will be based on the percent of the study completed). You will also have your aerobic fitness score given to you at the end of the study.

What are my rights?

- You can ask questions on any aspect of the project at any time, and we will do our best to answer them to your satisfaction.
- As a participant in the study you will provide information on the understanding that your name will not be used unless you give permission to the researcher.
- You have the right to view your own data at any stage and have it explained to you.
- You have the right to have any blood samples returned to you after they have been analyzed.
- You will also be given access to a summary of the project findings when it is concluded.
- You can withdraw from the project at any time, without giving any reason and without penalty.

What about compensation for injury?

If physical injury results from your participation in this study, you should visit a treatment provider to make a claim to ACC as soon as possible. ACC cover and entitlements are not automatic and your claim will be assessed by ACC in accordance with the Injury Prevention, Rehabilitation and Compensation Act 2001. If your claim is accepted, ACC must inform you of your entitlements, and must help you access those entitlements. Entitlements may include, but not be limited to, treatment costs, travel costs for rehabilitation, loss of earnings, and/or lump sum for permanent impairment. Compensation for mental trauma may also be included, but only if this is incurred as a result of physical injury. If your ACC claim is not accepted you should immediately contact Toby Mundel. Toby Mundel will initiate processes to ensure you receive compensation equivalent to that to which you would have been entitled had ACC accepted your claim.

Am I eligible?

Although voluntary, your participation will also be confirmed on criteria relating to health and safety; namely, in this study we are seeking healthy, moderately active (exercise a minimum of 3 days per week and have a VO_2max of 40-55 ml/kg/min) males. For health/safety reasons, you should **not** participate if any of the following apply to you:

- You have any known heart or cardiovascular condition or if a member of your family died below the age of fifty (50) as a result of a heart condition.
- You have (a) condition(s) that could be made worse by exercise.
- You have ever had an injury or any medical condition that you think may affect your ability to sense pain or discomfort.

- You have cultural or religious sensitivities about human body measurements.
- You have any other reason to consider that you are not in good health and of average, or better than average, fitness.
- You or a family member has a bleeding disorder.
- You have previously had to seek medical advice after exercising in hot conditions.
- You have hemorrhoids, diarrhea or have undergone rectal surgery.

Anything else I need to know?

You will be asked to wear suitable clothing and footwear that you feel comfortable running and walking in. Water will be provided throughout the testing procedure and showers are also available should you need them. We would also like you to fill out a food and physical activity diary and arrive at each session having fasted for between 2 and 4 hours.

All data obtained from this study will be kept strictly confidential. Data will be identified as code only. Results will be made available to you at the completion of the study.

If you are interested in taking part, contact the researchers at the start of this sheet.

This project has been reviewed and approved by the Massey University Human Ethics Committee: Southern A, Application 14/10. If you have any concerns about the conduct of this research, please contact Dr Brian Finch, Chair, Massey University Human Ethics Committee: Southern A, telephone 06 350 5799 x 84459, email humanethicsoutha@massey.ac.nz.

Health Screening Questionnaire

School of Sport and Exercise

Private Bag 11222

Palmerston North 4442

New Zealand

MASSEY UNIVERSITY

Pre-Exercise Health Screening Questionnaire

Name:_____

Address: _____

Phone: _____

Age: _____

This questionnaire has been designed to identify the small number of persons (15-69 years of age) for whom physical activity might be inappropriate. The questions are based upon the Physical Activity Readiness Questionnaire, originally devised by the British Columbia Dept of Health (Canada), as revised by Thomas *et al.* (1992) and Cardinal *et al.* (1996), with the added requirements of the Massey University Human Ethics Committee. The information provided by you on this form will be treated with the strictest confidentiality.

You should be aware that even amongst healthy persons who undertake regular physical activity there is a risk of sudden death during exercise. Though extremely rare, such cases can occur in people with an undiagnosed heart condition. If you have any reason to suspect that you may have a heart condition that will put you at risk during exercise, you should seek advice from a medical practitioner before undertaking an exercise test.

Please read the following questions carefully. If you have any difficulty, please advise the exercise specialist who is conducting the exercise test. Please answer all of the following questions by ticking only one box for each question:

1. Has your doctor ever said that you have a heart condition and that you should only do physical activity recommended by a doctor?

Yes ☐ No ☐

2. Do you feel a pain in your chest when you do physical activity?

Yes ☐ No ☐

3. In the past month have you had chest pain when you were not doing physical activity?

Yes ☐ No ☐

4. Do you lose your balance because of dizziness or do you ever lose consciousness?

Yes ☐ No ☐

5. Is your doctor currently prescribing drugs (for example, water pills) for your blood pressure or heart condition?

Yes ☐ No ☐

6. Do you have a bone or joint problem that could be made worse by vigorous exercise?

Yes ☐ No ☐

7. Do you know of any other reason why you should not do physical activity?

Yes ☐ No ☐

8. Have any immediate family had heart problems prior to the age of 50?

Yes ☐ No ☐

9. Have you been hospitalized recently (past two months)?

Yes ☐ No ☐

10. Do you have any infectious disease that may be transmitted in blood?

Yes ☐ No ☐

11. This test will include the taking of blood for biochemical measuring. Do you have any objection to this?

Yes ☐ No ☐

12. Do you, or anyone in your family, have a bleeding or clotting disorder?

Yes ☐ No ☐

13. Do you currently have hemorrhoids?

Yes ☐ No ☐

14. Do you currently have any other rectal-associated conditions (i.e. anal fissures)?

☐ ☐

Yes No

15. Have you recently undergone rectal surgery?

Yes ☐ No ☐

16. Do you currently have diarrhea?

Yes ☐ No ☐

17. Do you take any supplements regularly?

Yes ☐ No ☐

18. Do you exercise 3 or more days per week?

Yes ☐ No ☐

I have read, understood and completed this questionnaire.

Signature: _____ Date: _____

References

Thomas S, Reading J and Shephard RJ. (1992) Revision of the Physical Activity Readiness Questionnaire (PAR-Q). *Can J Sport Sci* 17(4): 338-345.

Cardinal BJ, Esters J and Cardinal MK. (1996) Evaluation of the revised physical activity readiness questionnaire in older adults. *Med Sci Sports Exerc* 28(4): 468-472

Consent Form

School of Sport and Exercise
Private Bag 11222
Palmerston North 4442
New Zealand
T 64 6 350 4336
F 64 6 350 5657

Adaptation to exercise for maximal aerobic capacity, submaximal aerobic efficiency, and cardiovascular adjustments: does addition of heat stress induce greater improvements than exercise alone?

PARTICIPANT CONSENT FORM

I have read the Information Sheet and have had the details of the study explained to me. My questions have been answered to my satisfaction, and I understand that I may ask further questions at any time.

I agree to participate in this study under the conditions set out in the Information Sheet.

Signature: …… **Date:** ……

Full Name – Printed: ……

Ethical Letter of Approval

MASSEY UNIVERSITY
TE KUNENGA KI PŪREHUROA

25 March 2014

Melissa Black
School of Sport & Exercise
PN621

Dear Melissa

Re: HEC: Southern A Application – 14/10
Adaptation to exercise for health and exercise capacity: Does addition of heat stress induce greater improvements than exercise alone?

Thank you for your letter dated 24 March 2014.

On behalf of the Massey University Human Ethics Committee: Southern A I am pleased to advise you that the ethics of your application are now approved. Approval is for three years. If this project has not been completed within three years from the date of this letter, reapproval must be requested.

If the nature, content, location, procedures or personnel of your approved application change, please advise the Secretary of the Committee.

Yours sincerely

Dr Brian Finch, Chair
Massey University Human Ethics Committee: Southern A

cc Dr Toby Mundel
School of Sport & Exercise
PN621

Dr Darryl Cochrane
School of Sport & Exercise
PN621

Prof Stephen Stannard, HoS
School of Sport & Exercise
PN621

Massey University Human Ethics Committee
Accredited by the Health Research Council

Research Ethics Office, Research and Enterprise

Massey University, Private Bag 11222, Palmerston North 4442, New Zealand T 06 3505573; 06 3505575 F 06 350 5622
E humanethics@massey.ac.nz; animalethics@massey.ac.nz; gtc@massey.ac.nz **www.massey.ac.nz**

Cultural Consideration for Ethical Approval

MASSEY UNIVERSITY
COLLEGE OF HEALTH
TE KURA HAUORA TANGATA

March 21, 2014

To whom it may concern,

I have met with Melissa Black and Dr. Toby Mundel to discuss possible ethical issues, particularly pertaining to Māori who may participate within their study ***"Adaptation to exercise for health and exercise capacity: does addition of heat stress induce greater improvements than exercise alone?"***.

Although risks to cultural integrity are minimal in this study in context with a kaupapa Māori framework and previous methods of research our group at Te Pumanawa Hauora have used in the lab-based setting, I have discussed with Melissa and Toby aspects of their study which may be perceived as harmful for Māori both culturally and psychologically. They are happy to consult myself and other Māori researchers as needed throughout the study.

If you require further information from me, please feel free to contact me.

Nāku noa nā,

Dr Isaac Warbrick

Ngāti Rangitihi, Ngāti Whakaue, Ngāti Te Ata

Research Fellow

Te Kunenga ki Pūrehuroa

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