uOttawa

L'Université canadienne
Canada's university

FACULTÉ DES ÉTUDES SUPÉRIEURES ET POSTOCTORALES

u Ottawa

L'Université canadienne
Canada's university

FACULTY OF GRADUATE AND POSDOCTORAL STUDIES

Bruno Lemire
AUTEUR DE LA THÈSE / AUTHOR OF THESIS

M.Sc. (Human Kinetics)
GRADE / DEGREE

School of Human Kinetics
FACULTÉ, ÉCOLE, DÉPARTEMENT / FACULTY, SCHOOL, DEPARTMENT

Cold Water Immersion After Exercise-induced Hyperthermia

TITRE DE LA THÈSE / TITLE OF THESIS

G. Kenny
DIRECTEUR (DIRECTRICE) DE LA THÈSE / THESIS SUPERVISOR

CO-DIRECTEUR (CO-DIRECTRICE) DE LA THÈSE / THESIS CO-SUPERVISOR

EXAMINATEURS (EXAMINATRICES) DE LA THÈSE / THESIS EXAMINERS

E. Doucet

P. Imbeault

Gary W. Slater
Le Doyen de la Faculté des études supérieures et postdoctorales / Dean of the Faculty of Graduate and Postdoctoral Studies

Cold Water Immersion After Exercise-Induced Hyperthermia

Thesis

By
Bruno Lemire (M.Sc. candidate)

Submitted to the faculty of graduate and Postdoctoral Studies
in partial fulfillment of the requirements for the degree of
Master's of Science in Human Kinetics

Your file Votre référence
ISBN: 978-0-494-48473-9
Our file Notre référence
ISBN: 978-0-494-48473-9

ACKNOWLEDGEMENTS

I wish to thank the following individuals for their support and guidance in the realisation of this thesis as well as for making my years at the graduate level a memorable experience:

Dr. Pascal Imbeault et Dr. Éric Doucet – Merci d'avoir accepté la supervision de mon comité et de m'avoir bien dirigé vers l'achèvement de ce projet de recherche.

Dr. Ollie Jay – Thank you for your continuous support, great insights and informal teaching which helped advance my knowledge in the field of environmental physiology.

Daniel Gagnon (M.Sc.) – Merci de m'avoir gardé sur le droit chemin et de m'avoir donné de très bons conseils sur tous les projets que nous avons réalisé ensemble.

Dr. Glen Kenny – Thank you giving me the possibility to pursue my studies at the graduate level and for your unprecedented support throughout all aspects of the completion of my thesis.

I would like to thank all those who participated in the study that served as the basis for my thesis, as well as all my fellow graduate students for their technical support while running the experimental trials.

Finalement, j'aimerais remercier ma copine, la future Dre. Charbonneau et mes parents, Pierre et Micheline pour leur constant support tout au long de cette aventure.

ABSTRACT

Cold water immersion (CWI) is the most effective known cooling treatment against exercise-induced hyperthermia. However, sex differences related to body composition (i.e. body fat, muscle mass, surface area, etc.) may affect core cooling rates in hyperthermic males and females.

Purpose:

To determine sex related differences in core cooling rates during CWI after exercise-induced hyperthermia.

Methods:

Ten male (M) and nine female (F) participants matched for body surface area to mass ratio took part in this study. Participants exercised at 65% $\dot{V}O_{2max}$ at an ambient temperature of 40°C until rectal temperature (T_{re}) increased to 39.5°C. Following exercise, subjects were immersed in a 2°C circulated water bath until T_{re} decreased to 37.5 °C.

Results:

Females had a significantly greater core cooling rate compared to males. This was paralleled by a lower skin temperature and a shorter time to reach the exit criterion.

Conclusion:

We conclude that previously hyperthermic females have a 1.7 times greater T_{re} cooling rate compared to males. We attribute this difference to a smaller lean body mass (expressed by the body-surface-area-to-lean-body-mass ratio) in females compared to males.

TABLE of CONTENTS

PART ONE: Theoretical considerations

CHAPTER ONE

INTRODUCTION

1.0 Introduction

Participation in various sports, as well as military operations and industrial work, can put individuals at risk of suffering from heat stroke and other heat-related injuries, especially when exposed to high ambient temperatures (36, 115). Other than high ambient air temperatures, such environments often present sources of high radiant heat, high humidity and direct physical contact with hot objects. When these are coupled with the increased metabolic heat production from engaging in physical demands as well as the additional effects of semi-permeable or impermeable protective clothing, fatigue, dehydration, sleep deprivation or various pathological conditions, the body's physiological responses (i.e. skin vasodilation and sweating) are not sufficient to achieve thermal balance (98). This results in an elevation of core body temperature which influences the health and performance of these individuals. This can lead to an increased risk of accidents and/or heat-related illnesses, disability and even death (11, 14).

During physical work, the rate of heat production increases above resting levels because of heat produced as a by-product of skeletal muscle contraction (60, 89). As exercise continues, the increase in convective heat transfer between muscles and blood tissues causes a sustained rise in core temperature. Thus, core temperature rises during dynamic exercise subsequent to a sustained increase in muscle heat storage, while the magnitude of increase is dependent on the intensity of exercise (63).

Heat loss mechanisms including increases in skin blood flow and sweating are activated following the increase in core temperature. This results in an increased rate of whole-body heat loss. Once the rate of heat loss is sufficiently elevated to dissipate heat at the rate at which it is being produced, thermal balance is restored and core temperature reaches a steady-state level. However, as noted above, a progressive rise in core temperature will occur as the level of metabolic heat production exceeds the heat dissipating capacity of the thermoregulatory system. This will lead to an uncompensated heat gain and an increase in body heat storage (86).

At a core temperature of ~38°C, significant physical discomfort occurs; at 38.5°C, clinical hyperthermia begins, evidenced by dizziness and confusion, leading to a decrease in mental alertness. Beyond a core temperature of ~39°C the risk of physical collapse is prevalent (87). Heat stroke occurs at core temperatures greater than ~41°C (4, 90). In this case, symptoms include decreases in blood pressure, vomiting, convulsions, unconsciousness, delirium and failure in central nervous thermoregulatory control (83). The denaturing of body proteins occur at a core temperature of ~42°C with death soon following (90). The extent of tissue damage and physiological malfunctions is not only related to the degree of hyperthermia but also to the duration of this state (96, 101). Given this relationship, it is important to investigate treatments for exertional heat-related illnesses.

Treatment against Exercise-Induced Hyperthermia

An effective treatment for exercise-induced hyperthermia should decrease core temperatures as fast as possible (20, 102). Some studies suggest that enhancing evaporative cooling with the use of water and air sprays is the most effective method of reducing high core body temperature (102, 113), while other studies support the use of cold water immersion (CWI) due to the high thermal conductivity of water (5, 20, 22, 26, 83, 93). Proulx et al. (2003) showed the highest cooling rate (0.35°C/min) in laboratory settings using 2°C water and this was therefore perceived to be the most effective treatment in eliminating exercise induced-hyperthermia in young, healthy active adults. Others like Armstrong et al. (1996) also showed that CWI provided faster core cooling rates (0.20°C/min) compared to cool air exposure (0.11°C/min) in healthy runners. The argument against CWI is that it causes rapid skin vasoconstriction and intense shivering, thereby attenuating core cooling rates (68, 75). Casa et al. (2007) has recently argued against this stating that the conductivity of water (630.5 $mW/m^2/°K$) is so great compared to air (26.2 $mW/m^2/°K$), that heat loss through conduction will override the metabolic heat production and peripheral vasoconstriction during CWI. In addition, he concluded that the advantages of CWI (i.e. rapid reduction of core temperatures) far outweigh its possible disadvantages (i.e. patient discomfort, hygiene) and is currently viewed as 'the gold standard' treatment for heat stressed individuals (20). However, studies examining CWI as a treatment for exercise-induced hyperthermia have shown a large variance in core cooling rates (5, 23, 26, 35, 93). Thus, it is possible that physiological and anthropometrical

variations (i.e. sex, body fat, lean body mass, surface area, etc.) affect the outcome of this treatment.

Factors affecting heat loss during CWI

Body adiposity

Studies show that body fat acts as an insulator attenuating the fall in core body temperature during cold exposure in normothermic individuals (28, 44, 91, 95, 107). In vivo, adipose tissue is a very poor conductor, as it contains less water and has a thermal conductivity of ~35% less than blood and 50% less than skeletal muscle (28). Differences in distribution of fat mass have been shown to reduce the rate of heat loss in swimmers immersed in 25.2°C water (111). In light of the fact that females typically have a greater adiposity (97), they are likely to have a thermal advantage at rest during CWI (i.e. slower core cooling rates) (107). However, data from our laboratory report that a difference in ~10% body fat did not provide any advantage (i.e. greater core cooling rate) to a low fat (12.9%) group compared to a high fat (22.3%) group, when immersed in cold water (8°C) after being rendered hyperthermic by exercise in the heat (73). This occurrence is due to 1) an increased thermal gradient between the core, the skin and the water caused by hyperthermia increases the potential for greater heat loss bypassing the subcutaneous fat and 2) a post-exercise status (i.e. elevated blood flows) that accentuates heat loss during a subsequent CWI. This in fact minimizes the effect of adipose tissue thickness on core cooling rates in previously hyperthermic individuals.

Body Surface Area to Mass Ratio

In addition to the adipose tissue thickness, the body surface area to mass ratio (A_D/M) has also been shown to have an effect on core cooling rates during CWI (2, 76, 78). If we consider that the exchange of heat is greatly dependent on the surface area and mass across which heat is transferred and that females typically have a greater A_D/M; it would then be expected that they cool at quicker rates compared to males when exposed to the same cold stress (76). Although this result is said to be offset by an increase adipose tissue thickness (76), Hayward et al. (1975) observed that females at rest in cold water with a 52% larger skinfold thickness than males, had a similar rate of core cooling due to a greater A_D/M. In addition, Kollias et al. (1974) concluded that individuals with a greater A_D/M showed an increase in metabolic rate and a greater decrease in core temperature compared to those with a smaller A_D/M. These studies clearly show that A_D/M should be considered as an important variable when comparing sexes during cold water immersion. A previous study that used CWI on hyperthermic individuals did not significantly conclude that A_D/M played a role in affecting core cooling rates (92). It is of importance to note that this aforementioned study was not designed for this purpose, thus showing a narrow range of A_D/M in their participants (92).

Lean Body Mass

It has become apparent that muscle mass has a substantial effect on whole body insulation during cold stress (1). At rest in cold water, muscle has been shown to contribute substantially to the body's insulatory capacity while acting as a static layer of protecting tissue (1, 28, 109). Park et al. (1984) showed that muscle tissue accounts for 75% of the total body insulation at rest in cool water (28 °C to 32°C) with the remaining insulative

capacity attributed to skin and adipose tissue. The importance of muscle as an insulator was also shown previously by Veicsteinas et al. (1982) when he compared the local tissue insulation in contralateral limbs during rest and exercise. He concluded that the resting limb had a much greater local tissue insulation due to the lower blood flow and skin vasoconstriction compared with the exercising limb (109).

Blood Flow and Muscle Temperature

Differences in thermal status and tissue blood distribution in a hyperthermic state may reduce the relative influence of tissue insulation on core cooling rates during the immersion period. In an exercise-induced hyperthermic state, the core temperatures and skin-to-water thermal gradients are elevated, increasing the potential for non-evaporative heat loss. Furthermore, post-exercise pooling of blood in the previously active limbs has been associated with elevated deep muscle temperatures from pre-exercise baseline levels for up to 30-min post-exercise (61). These elevations in muscle temperature combined with high levels of skin blood perfusion during the early stages of exertional heat stress recovery (33) could lead to a greater overall core cooling rate during a subsequent CWI (100). In addition, a recent study by Kenny and Jay (2007) showed a greater and more prolonged elevation in core temperature in active and inactive muscle temperatures in females following dynamic exercise (61). This was explained by a greater decrease in mean arterial blood pressure caused by a greater blood pooling in the previous active limbs in females. Thus, this would indicate a greater thermal gradient between the peripheral muscles and the cold water during a subsequent immersion, providing a greater drive for heat loss in females. Regardless of the mechanism for these reported responses, the sex-

related differences in muscle temperature and blood pooling following exercise might influence core cooling rates during a subsequent CWI.

Recommendations for CWI on previously hyperthermic individuals

Previous studies investigating core cooling rates in hyperthermic individuals using CWI have elucidated possible problems relating to the 'afterdrop' in core temperatures after the cessation of the immersion (20, 43, 94). In order to overcome this, a recent study by Proulx et al. (2006), presented a specific recommendation for the duration of the CWI (94). They reported that whole-body cooling should be stopped when the patients` rectal temperature (T_{re}) decreases to 38.6°C when immersed in water less than 10°C. This exit T_{re} recommendation would eliminate close to 100% of the heat gained by the previous exercise without causing a risk of hypothermia. While providing good insight on how to manage cooling treatments, this recommendation has not yet been tested in a clinical setting. The results have been extrapolated from longer CWI trials. The testing of those recommendations is vital in order to provide 'guidelines' to health professionals who deal with heat stress individuals.

1.1 Rationale

As shown in past studies, there are significant differences between sexes in muscle temperatures following exercise. Muscle temperature remains elevated for a prolonged period post-exercise in females as compared to males while suggesting excessive pooling of blood in the lower limbs. This in combination with some key anthropometrical differences (i.e. smaller muscle mass, greater surface area) would cause a more rapid

cooling of the periphery and subsequently the core in females especially when exposed to very cold water (2°C).

Previous studies indicate the need to establish a consensus on the safe cooling limits of hyperthermic individuals during CWI. Proulx et al. (2006) have provided the most recent contribution to this field. However, these guidelines need to be tested in order to provide health professional with a solid basis for the use of these recommendations.

1.2 Hypotheses

First, we will investigate the hypothesis that whole-body core cooling rates will be greater in previously hyperthermic females as compared to males during a 2°C water immersion.

Second, we will evaluate the hypothesis that an exit rectal temperature of 38.6°C will prevent previously hyperthermic individuals to overshoot and fall into a hypothermic state (i.e. $T_{re} < 36°C$).

1.3 Objectives

The primary objective of this study is to investigate the core cooling efficacy of cold water immersion in males and females rendered hyperthermic with exercise in the heat.

This research project will also provide new insight on the latest cooling recommendations to health professionals who may deal with those exposed to exercise-induced hyperthermia.

1.5 Relevance

This study will add to our knowledge on the sex specific variables that impact the rate of core cooling in individuals that are rendered hyperthermic.

Also, this study will serve as a tool to accept or refute the established theoretical safe cooling limits for cold water immersion in individuals suffering from exercise-induced hyperthermia.

1.6 Limitations

The present study requires that the subjects exercise at ~65% of their maximal aerobic capacity. Some subjects might not be able to sustain this intensity due to the heat stress. In addition, the percent humidity will be taken as a marker but will not be controlled as a variable during the exercise. Also, the transfer time between the thermal chamber and the cold bath might affect the first minute cooling rate, but this process will be done as quickly as possible.

1.7 Delimitations

There will be an imposed restriction as to the degree of hyperthermia brought about by the exercise. Participants will be limited to a target core temperature, (i.e. as defined by a T_{re} of 39.5°C); therefore, the results of this study will be limited to individuals suffering from mild hyperthermia as compared to severe hyperthermia (i.e. as defined by a T_{re} above 40°C). Subjects recruited for the study will be aged between 18 and 35 years and physically active. Therefore, the results of this study will not apply to children, the elderly or to a sedentary or obese population.

CHAPTER TWO

REVIEW OF LITERATURE

2.1 Thermoregulation

2.1.1 Thermoregulation of the human body

The control of core body temperature is largely dependent upon the external heat exchange between the body surface and the ambient environment and the internal heat exchange between tissues and different compartments of the body (112). On average, the body temperature which is the temperature of organs and other internal tissues is around 37°C. Core body temperature can fluctuate within this range depending on numerous factors including circadian rhythms, menstrual cycle, exercise, endocrine disorders and infections (3). Maintaining homeostasis in regards to body temperature is one of the most complex systems in the human body. Many physiological adjustments are made in order to regulate body temperature. The anterior hypothalamus controls temperature regulation mechanisms through the autonomic nervous system (10). Both 'cold' and 'warm' thermosensitive neurons are found in the hypothalamus, brain stem and spinal cord (25, 34, 46). The information that the preoptic region of the anterior hypothalamus (PO/AH) receives is coming from different thermoreceptors located in different areas in the body. Blood temperature is measured by the hypothalamus itself, while skin temperature is assessed by thermoreceptors just under the skin.

There are also deep thermoreceptors that are located close to the visceral organs, spinal cord and major veins in the body (12, 16). It is important to note that the neurons affected by heat will increase their signalling when heated and the effectors will act in a hierarchy fashion in order to attend to the disturbance in body temperature (10). For

example, at rest, exposure to a heat stress (high ambient temperature) activates the heat loss mechanisms as measured by an increase in skin vasodilation and sweating. Similarly, exposure to a cold stress will activate peripheral cold sensitive neurons resulting in vasoconstriction of skin vasculature and a subsequent increase in shivering activity (10).

2.1.2 Core body temperature regulations

Previous work of Hammel et al. (1963) and Hardy (1963) suggested that signal processing within the preoptic hypothalamus for the regulation of core body temperature was based upon a controller with an adjustable set-point (39-41). The body is constantly looking for a state of equilibrium at around a set point temperature and is always compared to that temperature for future neural effector responses (40, 42). The body will use neurosensoring mechanisms from aforementioned neural activity pathways to adjust the body temperature according to this set point temperature. Deviations from this set-point result in the activation of autonomic effector responses (i.e. vasomotor responses, sweating, shivering).

A modification of the set-point theory of temperature regulation was introduced by Mekjavic et al. (1991) who reported separate core body temperature thresholds for sweating and shivering, thus supporting the concept of a "null-zone" or interthreshold range of temperature over which the hypothalamus is insensitive to variations in its own temperature or input from other thermal receptors (81). A range of core body temperatures over which the body's thermoregulatory mechanisms remain inactive, supports the notion of an interthreshold zone rather than a set-point control of core body temperature regulation. These findings are consistent with earlier human (9) and animal (51) studies.

For a more comprehensive discussion of this topic, the reader is referred to the review by Mekjavic and Eiken (2006) (80).

A number of investigators have proposed that it is core body temperature that is regulated, not a central reference or set-point temperature, by the hypothalamus. As stated by Webb (1995) this is equivalent to saying that the body regulates its heat content and by that heat regulation is activated (112). Houdas and Guieu (1973) and Webb (1995) proposed a model in which whole body heat content is the controlled variable, with core body temperature merely following the dynamic regulation of heat and simply behaving as an indicator of the level of body heat content (48, 112). According to this theory, the themoregulatory control system acts more as a servo mechanism of thermal exchange rather than as a regulator of core temperature.

While there currently exist disagreement amongst physiologists regarding the theory of core body temperature regulation, the heat loss mechanisms by which the human body changes in core body temperature in the face of a thermal challenge are well established in the scientific community.

2.1.3 Heat exchange mechanisms

Many mechanisms help the human body to exchange heat with the environment. Conduction, which is the exchange of heat through direct contact of two surfaces, is seldom used in a sport or working setting to loose or gain heat. Convection on the other hand plays an important role in cooling during exercise. It is the transfer of heat between surface via gas or liquid. This process is regulated by the gradient between the skin and the peripheral blood vessels. When air is forced over the skin (forced air convection), the increase in air flow over the skin surface causes the warm air just above the skin to move and to be

replaced by cooler air which creates a better gradient for heat loss between the skin and the blood vessels. Evaporation is the most important mode of cooling during heat stress and is the transfer of heat from the evaporation of liquid from the skin. During exercise in a hot and dry environment, evaporation will account for 98% of the body cooling efficiency (17). Lastly, radiation refers to the movement of heat between the body and the environment via electromagnetic waves (56).The body will absorb these radiation waves such has sunrays. In summary, the interaction of the man with the thermal environment can be conceptually demonstrated using the human heat balance equation (7):

$$M - W = (K + C + R + E_{SK}) + (C_{RES} + E_{RES}) + \mathbf{S}$$

Where: M = rate of metabolic heat production

W = rate of mechanical work (effectively = 0)

K = rate of conductive heat loss

C = rate of convective heat loss from the skin

R = rate of radiative heat loss from the skin

E_{SK} = rate of evaporative heat loss from the skin

C_{RES} = rate of convective heat loss from respiration

E_{RES} = rate of evaporative heat loss from respiration

S = rate of body heat store (all units $W \cdot m^{-2}$)

2.1.4 Exercise and thermoregulation

During all types of exercise the body's ability to thermoregulate is challenged. Heat is produced as a bi-product of metabolism (metabolism = all of the reactions that occur in

the human body). However, the human body is only 25% efficient; therefore we lose approximately 75% of energy as heat (34). During exercise, the rate of heat production increases above resting levels because heat is produced as a secondary outcome of skeletal muscle contraction (99, 112). As the heat production continues, the increase in convective heat transfer between muscle and blood causes a sustained rise in core temperature. Thus, core temperature rises during dynamic exercise subsequent to a sustained increase in heat storage. The magnitude of increase in heat storage is dependent on the intensity of exercise, environmental conditions and physical characteristics (66).

In the absence of any heat dissipating mechanisms, the high rate of heat production during exercise could result in a core body temperature elevation of ~1°C every 5 minutes (84). In addition, cellular metabolism is increased by 13% for every 1°C increase in core body temperature and would result in developing a heat illness (56).

In response to the increase in core temperature, the heat loss response mechanisms of increased skin blood flow (skin vasodilation) and sweating are activated resulting in an increase in the rate of whole-body heat loss. The increase in skin blood flow is associated with the increase metabolic demand of the exercise bout. This phenomenon is also supported by a redistribution of blood flow from the renal and splanchnic circulations towards the region of working muscles (18, 21, 98). This enhanced in skin blood flow thereby enhances peripheral convective heat exchange and therefore surface heat loss. In addition, an increased cardiac output is needed to keep blood pressure to values sufficiently elevated so as to maintain both skin and muscle tissue perfusion. In parallel to the increase in skin blood flow in the early stages of exercise, sweat rate also increases, albeit the mechanism of control differs greatly. Heat loss by evaporation occurs through two channels

of insensible heat dissipation: loses through the skin (perspiration) and lung surfaces (respiration) (31). The skin is cooled when sweat absorbs heat from the body and evaporates. It's the evaporation of sweat that has cooling power. However, this heat dissipating mechanism occurs when the water vapour concentration in the ambient air is favourable in order to create a gradient between the skin and the environment.

Once the rate of heat loss is sufficiently elevated to dissipate heat at the rate at which it is being produced, thermal balance is restored such that core temperature stops rising and reaches a steady-state equilibrium level (67). When doing physical work in the heat, the response to a thermal stress is proportional to the displacement of core body temperature as defined by an integrated afferent input from both core body temperature and mean skin temperature as described above. At any given skin temperature, the heat loss response activity of sweating and skin blood flow is proportional to core body temperature (85). In contrast, increasing mean skin temperature (i.e., exercise in high ambient air temperature conditions) tends to lower the threshold of core body temperature at which the onset of sweating and/or skin blood flow occurs and increases the response at any given core temperature (65, 67). In thermoneutral conditions and during a low to moderate submaximal exercise effort, the rate of heat loss effectively matches the rate of heat production such that the core body temperature becomes stable, albeit at an elevated level. In a cooler ambient air conditions, the thermal gradient favours heat exchange with the surrounding environment. In contrast, when ambient air temperatures exceed skin temperature, the gradient is reversed.

Previous research showed that during physical work with substantial combined metabolic and environmental heat stress, the thermal load is sufficiently elevated to result

in a further increase in body core temperature (74). For example, when moderate intensity work is performed at ambient temperatures greater than 25°C, the evaporative capacity of the environment is insufficient to offset the metabolic heat load gained from the physical work resulting in a further elevation of body core temperature (85). During the summer, physical work is often complicated by environmental conditions. As noted above, when air temperature is greater than body temperature, the body absorbs heat from the environment. In these ambient conditions, evaporation provides the major defense against overheating. In fact, evaporative heat loss account for 98% of total heat loss in a hot and relatively low humidity setting (6). However, it is to note that the total sweat vaporized depends on the amount of skin exposed, air currents, temperature and humidity. Humidity is the most important determinant because evaporation is slowed when water vapor pressure in the air increases. Although you may be sweating more on a humid day, less sweat is evaporated. The body is then unable to effectively cool itself. Thus, exercise in the hot and humid conditions imposes a significant thermal challenge on the body. Typically, when exercise is performed under these conditions, the sweat simply falls from the body and does not provide any cooling power, rather it is simply wasted (114). This situation would therefore create an open window for suffering a heat illness.

2.2 Sex-Differences in thermoregulation

2.2.1 Sex differences in thermoregulation during exercise

Studies have looked at sex differences in thermoregulation during exercise. Some found that females sweat less than males (27, 55); others found that females tend to have greater heart rates for the same workload (104) and that females have much lower aerobic capacity than males (55). Moreover, females have a greater core temperature during

exercise (55) and they have a various degree of fluctuations in core temperatures during the luteal phase of their menstrual cycle (103). These aforementioned differences combined with differences in body composition may predispose females to a greater risk of an exertional heat-related injury. In general, females have a greater body fat percentage and a greater surface area to mass ratio than their male counterparts (88). Havenith et al. (1990) reported that body fat percentage and surface area to mass ratio were the most important physiological characteristics that contributes to an elevated body heat storage and therefore an elevated rectal temperature during exercise (45). It is important to note that when females and males are match for aerobic fitness, body fat percentage, body composition and body surface area to mass ratio, it is widely accepted that no difference still persist exist between genders, albeit this comparison is rarely done.

2.2.2 Post-exercise sex differences

Recent studies have established a link between the post-exercise decrease in blood pressure and the attenuation in heat loss response (53, 59, 63) the effect been more pronounced in females (62). A reduction in mean arterial pressure (MAP) after exercise was first described by Leonard Hill in 1898 (47), and subsequent studies have documented what is now considered the phenomenon of post-exercise hypotension (38, 58). During post-exercise recovery, the systemic vascular conductance is increased by about 30% (37). This in turn creates a rise in blood flow through the vasodilated regions, which contributes to an augmentation in venous blood pooling in previously active muscles (24, 57). It should be noted that the magnitude of the reduction in MAP is greater with the orthostatic influence of upright or standing posture than when the subjects are supine (38, 58). The "muscle pump", being non-functional during inactive recovery, also plays an important role

in the reduction of MAP. This increased blood volume in the vascular beds in conjunction with the loss of plasma volume associated with exercise (sweating), leads to a reduction in central venous pressure and cardiac filling pressure. The combined effects of persistent neural and vascular adjustments as well as the upright seated posture with dependent lower extremities and no muscle pump results in significant pooling of blood (38, 69). By this chain of events, an elevated cardiac output can be seen because of a maintained stroke volume and an elevated heart rate. Thus, post-exercise hypotension results from a sustained drop in vascular resistance that is not completely offset by a rise in cardiac output (37).

As noted above, many recent studies have provided evidence that the magnitude of heat loss responses after exercise appears to be correlated to a reduction in MAP that occurs after dynamic exercise (50, 52, 53). A reduction in MAP increases the threshold for sweating and increase the vasodilation threshold, which are both important factors affecting the rate of heat loss after exercise (64). It is also shown that post-exercise hypotension is associated with a prolonged elevation in esophageal temperature, which is an important measure of core temperature (63). In addition, post-exercise MAP has been shown to be significantly lower in females then males after an exercise bout (15, 54, 63). Kenny and Jay (2006) found that females had a greater MAP reduction up to 70 minutes after 15 minutes of exercise at 60% of VO_{2max} (61). In parallel to the greater reduction in MAP in females as compared to males, females demonstrate a greater and more prolonged elevation of core and active and inactive muscle temperature during and following exercise (59). This occurred in conjunction with a reduction of heat loss responses from skin blood flow and

sweating in females. Thus, we can conclude that females have a greater cardiovascular and thermal strain following dynamic exercise as compared to their males' counterparts.

2.3 Cooling methods after exercise-induced hyperthermia

2.3.1 Treatments for exercise-induced hyperthermia

In a recent review by Smith (2005) examining the efficacy of different cooling modalities, he demonstrated that the primary emphasis related to the treatment of hyperthermia should be to reduce the core temperature as quickly as possible. It has been shown that the major determinant of outcome in heatstroke is the duration of hyperthermia (102). Many studies have found CWI to be the best treatment for exercised-induced hyperthermia (5, 22, 26, 83). This modality is also the treatment of choice for many health centers such as the US Marine corps training base at Parris Island (32). CWI involves immersing the whole body (head out) in cold water. The best cooling times and cooling rates have been achieved with this technique (19). A study by Armstrong (1996), showed rates of core cooling of 0.20°C/min with CWI compared with rates of 0.11°C/min with cold air exposure (24.4°C) (5).

The high thermal conductivity of water (25 times greater than air) makes it an ideal choice to maximize core cooling (79, 102). It has been argued that whole-body cooling using a cold water bath would elicit a strong vasoconstrictor response of the skin thereby attenuating the rate of core cooling (20). Moreover, prolonged exposure to the cold water would result in an increase in heat production associated with the increase in shivering (20). However, this does not appear to be the case. Rather Proulx et al. (2003), showed that the rates of core cooling when cold water (2°C) was used to cool hyperthermic individuals, were much greater as compared to warmer water temperatures (93). In

addition, the majority of the individuals participating in that study were cooled before shivering even commenced, which suggests that even if skin vasoconstriction occurred, the effect is negligible in comparison to the rapid rate at which core cooling occurred (93).

2.3.2 Safe cooling limits for hyperthermic individuals

However, a major concern associated with CWI in the treatment for exercise-induced hyperthermia is the possible risk of overcooling the individuals beyond their normal baseline resting core temperatures. Studies show that when individuals are cooled to a core temperature of 37.5°C, core temperature continues to decrease even when the participants are out of the water (93, 94). In fact the 'afterdrop' is significantly large so as to cause hypothermia (82). Previous recommendations aimed at establishing an exit rectal temperature include: Moran et al. (2003) indicates between 38.0 to 37.5°C (82); Shapiro and Seidman (1990) and Harker and Gibson (1995) state 38.5°C should be the cut off point (43, 101) and finally Knochel (1974) recommends 38.9°C (70). More recent recommendations were published by Proulx et al.(2006) (94). They suggest that hyperthermic individuals who are cooled in water under 10°C, should be taken out of the water at a rectal temperature of 38.6°C (94). In contrast, for individuals who are cooled in a water bath above 10°C, they suggest that they should be removed when a rectal temperature of 37.8°C is achieved (94). However, these recommendations were interpolated from data where the participants were cooled until 37.5°C. It remains unclear if the recommendations are sufficient to protect these individuals. To date, no study has been conducted to evaluate and assess these specific guidelines under experimental conditions.

2.3.3 Sex differences in cooling mechanisms

Adult females tend to have a greater A_D/M compared to males, due primarily to their smaller body mass (105). The ratio has a significant influence on core cooling rates (13, 71, 76). When this ratio is larger, it facilitates the heat loss of an individual who is exposed to a cool environment. In a study by McArdle et al. (1984), the differences in thermoregulation between males and females during CWI (20°C) at rest was due partly to the sensitivity of the thermogenic responses and the A_D/M between the sexes (77). The core cooling rates were similar in both sexes with the same percent body fat; but this was true only for the individuals who had similar A_D/M. He concluded that under the same conditions of cold exposure and percent body fatness at rest, individuals that had a greater A_D/M cooled at a faster rate (77). In study by Tikuisis et al. (2000), it was concluded that the responses during water immersion at 18° C were identical between sexes when the percent body fat and the A_D/M were taken into account (106).

A study by Toner et al. (1986) showed the thermal differences between normothermic males and females could be attributed to body composition and total mass. They compared individuals with similar skinfold thickness and percentage of body fat but differing in A_D/M and total body mass. Results show that the tissue insulation was lower in individuals with a smaller mass, suggesting that the increases in tissue insulation in the larger individuals was due to a greater volume of muscle tissue and a larger muscle mass in the limbs at rest in cold water (108).

It is noteworthy that these aforementioned studies dealt with individuals who were not previously exercising and therefore the post-exercise and thermal status differences could not have played a role in the cooling processes. However, a study that used CWI to

cool off hyperthermic individuals did not significantly concluded that the percentage of body fat nor that the A_D/M had a significant influence on core cooling rates (93). Further, no significant relations were attributed to percent body fat (r^2=0.003) and A_D/M (r^2=0.03) in relation to the rate of core cooling (93), but this could easily be explain by the similar population used in this study.

2.4 Heat related injuries

2.4.1 Predisposing factors for heat illnesses

Risk factors for the onset of suffering a heat illness can be classified into two categories; internal (related to the body) or external (environmental) factors (49). Endogenous factors include any medications as well as the medical conditions of an individual (sickle cell trait, dehydration, recent febrile illness, sleep deprivation, sunburn, obesity, etc) (8). Exogenous factors can be high ambient temperature, humidity, excessive clothing or equipment, and the metabolic heat production of the individual (8).

Dehydration is known to be an important factor for the development of a heat illness (18). This usually happens after lack of fluid intake and/or extreme fluid loss (sweating). Proper hydration can reduce the incidence of heat illnesses (110).

The populations that are at a greater risk of suffering any kind of heat illnesses are the elderly, children, and those with medical conditions that may inhibit their thermoregulatory ability (49). In fact, heat loss responses in this vulnerable group is greatly reduced when there are demanded to perform in a high ambient temperature (98).

Many high active individuals share a common theme when the causes of heat illness are investigated. In the athlete population, heat illness is often related more to the endogenous heat production during high intensity activity level than to ambient

temperature as a primary source of heat (72). In the army, 50% of reported heat illnesses seen in the first 6 months of service (30). A high level of motivation combined with poor physical fitness is often linked to the onset of a heat illness. In fact, in the previously mentioned study, 60% of the soldiers with exertional heat stroke were overweight, which confirms that physical fitness and body composition are also very important factors to consider when dealing with people of a variety of body adiposity in hot environment (49).

2.4.2 Heat illnesses

Heat illnesses occur mainly because of lack of recognition of signs and symptoms. Typically, people force themselves to exercise in the heat because of perseverance and even because they due not want to be perceived as lazy (101). Survival rates are now 90 to 100% if heat illnesses are recognised early, but in the 1900's, survival was around 20% (29). Many forms of heat illnesses can occur; heat cramps, heat exhaustion, heat syncope and heat stroke.

Heat cramps are characterised by painful spasm of the skeletal muscles. The cause is often a combination of dehydration and muscle fatigue (29, 83).

Heat syncope is often referred to as an 'orthostatic syncopal episode' that usually happen after the cessation of exercise. Causes are attributed to low blood volume and postexercise vasodilation (29).

On the other hand, heat exhaustion appears to be associated with a cardiovascular system shut-down that results in a reduction in muscle and skin perfusion thereby reducing the rate of core body temperature increase and onset of muscle fatigue. This is the most common form of heat distress seen among athlete (5, 17). The causes are attributed to water

depletion or salt depletion, which occurs only after a long exposure to a hot and humid environment (5).

Heat stroke is known as the most severe form of heat illnesses. Two types of heat stroke can occur in individuals: the classical heat stroke and the exertional heat stroke (101). The first is a common disorder seen in children and the elderly population during heat waves. This heat stroke is purely from an exogenous heat load (heat stroke can occur from heat exposure without exercise) (29). The exertional heat stroke, on the other hand, appears very suddenly. Heat loads are exogenous and endogenous, which cause a serious elevation in body temperature. This event can cause some damage to the organs and tissues in the body (5). Body temperature could be as high as 43° Celsius and can be accompanied by a decrease in blood pressure, vomiting, convulsions, unconsciousness and delirium if conscious (83).

Therefore, the primary objective of this study is to investigate the core cooling efficacy of cold water immersion in males and females rendered hyperthermic with exercise in the heat. This research project will also provide new insight on the latest cooling recommendations to health professionals who may deal with those exposed to exercise-induced hyperthermia.

PART TWO: Methods and Results of Thesis

3.0 *Article 1*

Sex-Related Differences in Cooling Rates after Exercise-Induced Hyperthermia

Sex-Related Differences in Cooling Rates after Exercise-Induced Hyperthermia

Bruno Lemire, Daniel Gagnon, Ollie Jay and Glen Kenny

Centre for Human and Environmental Physiology Research, School of Human Kinetics, Faculty of Health Sciences, University of Ottawa, Ottawa, Ontario, Canada, K1N 6N5 and

Running head: Hyperthermia and cold water immersion

Address for correspondence:

Dr. G.P. Kenny
University of Ottawa
School of Human Kinetics
125 University,
Montpetit Hall
Room 367
Ottawa, Ontario, Canada
K1N 6N5
(613) 562-5800 ext. 4282
(613) 562-5149 (fax)
E-mail: gkenny@uottawa.ca

ABSTRACT

This study evaluated sex related differences in core cooling rates (as measured by rectal, esophageal and aural canal temperatures) during cold water immersion (CWI) after exercise-induced hyperthermia. Ten male (M) and nine female (F) participants matched for body surface-area-to-mass ratio (A_D/M) took part in this study. Participants exercised at 65% $\dot{V}O_{2max}$ at an ambient temperature of 40°C until rectal temperature (T_{re}) increased to 39.5°C. Following exercise, participants were immersed in a 2.0°C circulated water bath until T_{re} decreased to 37.5 °C. Significant differences were found in the overall core cooling rate for T_{re} (0.22°C/min ± 0.07 for F vs. 0.12°C/min ± 0.03 for M, $p = 0.001$) and in the overall immersion times (10.89 min ± 4.49 for F, and 18.13 min ± 4.47 for M, $p = 0.003$). Furthermore, a lower mean skin temperature ($p < 0.001$) at each minute during the CWI was recorded for females compared to males. However, no differences were found between the groups in the rate of heat loss during the CWI ($p = 0.180$). In addition, no differences were seen in metabolic heat production between groups until the mean end immersion time for females (~ 11 minutes) ($p > 0.05$), after which the males showed an increase in metabolic heat production until their mean end immersion time (~ 18 minutes). We conclude that previously hyperthermic females have a 1.7 time greater core cooling rate compared to males. We attribute this difference to a smaller lean body mass.

Keywords: sex difference; cold water immersion; core temperature; exercise-induced hyperthermia

INTRODUCTION

Exertional heat stress occurs with the combination of physical exertion and of high ambient temperatures and relative humidity. This can result in core temperatures above of 39.5°C or higher (32). The elevated core temperature associated with exertional heat stress can cause loss of consciousness, severe tissue damage, organ failure and even death if not recognized and treated immediately (16). The severity of the illness is directly related to the duration and level of core temperature elevation (27). Therefore, rapid cooling of hyperthermic individuals to a 'normal' core temperature should be the main goal of any treatment strategy (10, 13, 25, 27). It is now generally accepted that cold water immersion (CWI) is the 'gold standard' for the treatment of exertional heat stress (10). However, there is large variance in core cooling rates within and between studies that examined whole-body cooling with CWI (1, 4, 9, 25). Factors such as sex, body size, shape and composition can affect physiological outcomes when exposed to cold water. For example, females typically have a greater percentage of body fat, a smaller lean body mass and a larger body-surface-area-to-mass ratio (A_D/M) compared to their male counterparts (6, 20), which has resulted in inconsistency in cold stress responses between normothermic males and females (3, 18, 20, 21). However, it is unknown if these physical differences will result in different core cooling rates between sexes following exercise-induced hyperthermia.

First, the exchange of heat is greatly dependent on the surface area and the mass across which it is exchanged. As such, the A_D/M of an individual has been shown to be an important factor influencing core cooling rates (3). Since females have a greater A_D/M, it could be expected that they would cool at a quicker rate compared to males when exposed to a given cold stress. In fact, normothermic females with a similar percentage of body fat

but a greater A_D/M compared to males were reported to cool faster in cool water (20 to 28°C)(20).

Second, in normothermic individuals, cold-induced vasoconstriction combined with high levels of tissue insulation (i.e. subcutaneous fat) is inversely related to the rate of core temperature decline for a given cold stress (5, 26). However, subcutaneous fat does not affect core cooling rates in previously hyperthermic individuals (19). The post-exercise thermal status associated with exercise-induced hyperthermia (i.e. high core and skin temperatures as well as elevated blood perfusion) resulted in a lack of influence of body adiposity on subsequent core cooling rates (19). It was therefore concluded that a greater level of adipose tissue (~10%) does not affect core cooling rates in individuals that are under 25% of body fat. However, the remaining lean body mass may have a greater influence on core cooling rates.

Third, a study by Toner et al. (1986) showed that thermal differences between normothermic males and females during CWI could be attributed to body composition and total mass. Results show that tissue insulation was lower in individuals with a smaller body mass, suggesting that the increases in tissue insulation in the larger individuals were due to a greater volume of muscle tissue and a larger muscle mass in the limbs at rest in cold water (30). Moreover, lean body mass is often correlated to an increased in metabolic responses in cold water (2, 8, 20), which could attenuate the rate of core temperature decay during CWI. Since males typically have a greater amount of lean body mass, their metabolic response during cold exposure is greater than females when exposed to similar cold stress (14).

The purpose of this study was to evaluate possible sex-related differences in core cooling rates, as measured by rectal (T_{re}), esophageal (T_{es}) and aural canal (T_{ac}) temperatures during CWI (2°C) following exercise-induced hyperthermia. To eliminate some possible confounding influences due to differences in anthropometrical characteristics between sexes, participants were matched for A_D/M. We evaluated the hypothesis that core cooling rates in previously hyperthermic females would be greater compared to males due to their smaller lean body mass.

METHODS

Participants

Following approval of the experimental protocol from the University of Ottawa Research Ethics Committee, nineteen healthy and physically active participants volunteered and gave written consent to participate in this study. Participants were matched for A_D/M and divided into two groups; male (M, n = 10) and female (F, n = 9). Five to seven days before the experiments, maximal oxygen consumption ($\dot{V}O_{2max}$) was measured as well as body density using the hydrostatic weighing technique. Body fat percentage was then calculated using the Siri equation (7). Maximal oxygen consumption ($\dot{V}O_{2max}$) was measured during a progressive treadmill running protocol. The $\dot{V}O_{2max}$ data were used to select the submaximal workload for the experimental exercise phase of the study. The A_D/M of each participant was calculated from the measurements of weight and height (12). Females were tested in the follicular phase to avoid any disruptions in thermoregulatory response (24). The physical characteristics of the subjects are presented in Table 1.

Instrumentation

Esophageal temperature (T_{es}) was measured by placing a pediatric thermocouple probe of approximately 2 mm in diameter (Mon-a-therm Nasopharyngeal Temperature Probe, Mallinckrodt Medical, St-Louis, MO, USA) through the participant's nostril while they were asked to sip water through a straw. The location of the probe tip in the esophagus was estimated to be corresponding to the level of the eighth and ninth thoracic vertebrae (22). Aural canal temperature (T_{ac}) was measured using a tympanic thermocouple probe (Mon-a-therm Tympanic, Mallinckrodt Medical, St. Louis, MO, USA) placed in the aural canal until resting against the tympanic membrane (determined by the participant reporting an audible scratching sound), following which it was withdrawn slightly. The tympanic probe was held in position and isolated from the external environment with cotton, and ear protectors. Rectal temperature (T_{re}) was measured using a pediatric thermocouple probe (Mon-a-therm General Purpose Temperature Probe, Mallinckrodt Medical, St-Louis, MO, USA) inserted to a minimum of 12 cm past the anal sphincter. Skin temperature was monitored at 12 sites by Type T thermocouples integrated into heat-flow sensors (Concept Engineering, Old Saybrook, CT). These are commercially available heat flow disks that can read both the heat flux and skin temperature. The skin temperature is measured from the thermocouple that is integrated to the disk. The area-weighted mean skin temperature ($\bar{T}_{sk}$) and mean heat loss ($\bar{H}_{sk}$) were calculated by assigning the following regional percentages: head 6%, bicep 9%, forearm 6%, hand 2%, chest 9.5%, abdomen 9.5%, upper back 9.5%, lower back 9.5%, quadricep 10%, hamstring 10%, front calf 9.5%, back calf 9.5% (8). The

$\bar{T}_{sk}$ as well as the $\bar{H}_{sk}$ was thus calculated using the following equation: $\bar{T}_{sk}$ = (0.06 x $T_{forehead}$) + (0.09 x T_{bicep}) + (0.06 x Tf_{orearm}) + (0.02 x T_{hand}) + (0.095 x T_{chest}) + (0.095 x $T_{abdomen}$) + (0.095 x $T_{upper\ back}$) + (0.095 x $T_{lower\ back}$) + (0.10 x $T_{quadricep}$) + (0.10 x $T_{hamstring}$) + (0.095 x $T_{front\ calf}$) + (0.095 x $T_{back\ calf}$). During the CWI, average skin temperature for the sites immersed in water was calculated by assigning the following regional percentages ($\bar{T}_{sk\text{-}im}$): upper back 12%, lower back 12.5%, abdomen 12.5%, bicep 9.5%, forearm 9.5%, hand 2%, quadricep 12%, hamstring 12%, front calf 9%, back calf 9% (8, 12). Since the head and the chest were not entirely immersed in water for every subject, they were therefore not used to calculate $\bar{T}_{sk\text{-}im}$. Temperatures were collected and digitized (IBM ThinkCentre M50) with LabVIEW software (Version 7.0, National Instruments, TX, USA) at 5-s intervals, displayed graphically on the computer screen and recorded in spreadsheet format on a hard disk (IBM ThinkCentre M50) with LabVIEW software (Version 7.0, National Instruments, TX, USA). Oxygen consumption ($\dot{V}O_2$) was continuously recorded during the immersion period with the portable Jaeger – Oxycon mobile system (VIASYS healthcare, Hoechberg, Germany). Millilitres of O_2 were displayed on the laptop computer screen and recorded every minutes on an excel spreadsheet. Rate of metabolic heat production ($\dot{M}$) was calculated from minute-average values for $\dot{V}O_2$ and the respiratory exchange ratio (RER) during the CWI using the following equation (23):

$$\dot{M} \equiv \left(\dot{V}O_2 \bullet \left[\frac{RER - 0.7}{0.3} e_c + \frac{1 - RER}{0.3} e_f \right] \right) \text{.....Watts.....}$$

.....Where: e_c is the caloric equivalent per liter of oxygen for the oxidation of

carbohydrates (21.13 kJ), and e_f is the caloric equivalent per liter of oxygen for the oxidation of fat (19.62 kJ).

Experimental protocol

Each participant took part in one experimental trial. All trials were performed at the same time of day to avoid circadian variation in skin and core temperatures (31). Participants were asked to fast at least 3 h prior to experimentation, and were given instructions on what the last meal should be (i.e. two brown toasts, butter and orange juice). Water ingestion was permitted *ad libitum* during this time to ensure good hydration level. Upon arrival at the laboratory, subjects clothed in shorts, and athletic shoes were fitted with the appropriate instruments.

After instrumentation, the participants remained resting in the upright seated posture for 15 minutes at an ambient air temperature of 25.0°C and a relative humidity of 35.0%. They then entered a temperature controlled chamber regulated at an ambient temperature of 40.0°C where they ran on a treadmill at 65% of their predetermined $\dot{V}O_{2max}$ until their T_{re} reached 39.5°C or until volitional fatigue. Volitional fatigue was defined as an inability to maintain the required exercise intensity and/or a verbal manifestation from the participant to stop. Participants were then transferred (~1.5 min) and immersed up to the clavicles in a circulated water bath maintained at 2.0°C. Prior to entering the circulated water bath, participants were fitted with neoprene mitts and socks to reduce discomfort. Participants remained in the water until T_{re} reached 37.5°C.

Statistical Analyses

Physical characteristics between sexes were compared using independent sample T-tests. For each trial, baseline resting and end-exercise temperatures, exercise duration, cooling times, time to reach (in minutes) core temperature value of 39°C, 38.5°C. 38°C and 37.5°C, as well as the overall core cooling rates (start to end of immersion) were calculated for T_{re}, T_{es} and T_{ac}. Area-weighted mean $\overline{H}_{sk}$, $\overline{T}_{sk}$, $\overline{T}_{sk\text{-}im}$ were also calculated. In addition, $\dot{M}$ was calculated during the cold water immersion. Statistical analysis was carried out on the data for the two groups with independent sample T-tests. Further, a two-way mixed model analysis of variance was used to analyze $\overline{H}_{sk}$, $\overline{T}_{sk\text{-}im,}$ and $\dot{M}$ data during the immersion period using the repeatable factor of time (levels: 0, 1, 2, 3, 4, 5 –min (until the first participant was out of the water)) and the non-repeatable factor of sex [levels: male (M) and female (F)]. In addition, correlations between all participants` (i.e. males and females) overall T_{re} cooling rates the body-surface-area-to-lean-body-mass ratio (A_D/LBM). The level of significance was set at 0.05 and alpha level was adjusted during multiple comparisons so as to maintain the rate of Type I error at 5% during the Holm-Bonferroni post hoc analysis. All analyses were performed using the statistical software package SPSS 15.0 for Windows (SPSS Inc. Chicago, IL, USA).

RESULTS

Physical Characteristics

There were significant differences in body fat percent ($p < 0.001$), lean body mass (LBM) ($p < 0.001$), surface-area (A_D) ($p = 0.008$), surface-area-to-lean-body mass-ratio

(A_D/LBM) ($p < 0.001$) and relative $\dot{V}O_{2max}$ ($p < 0.001$) between groups. However, subjects were matched for A_D/M and therefore, participants had similar A_D/M ($p = 0.337$) (Table 1).

Exercise Period

There were no significant differences between groups in baseline T_{re} ($p = 0.482$), T_{es} ($p = 0.638$), and T_{ac} ($p = 0.686$). Exercise time taken to reach the experimental withdrawal criterion (T_{re} = 39.5°C) was not different between groups (35.21 ± 7.48 min for F, and 34.17 ± 7.29 min for M, $p = 0.763$). All subjects met this criterion. Exercise resulted in an increase from baseline in T_{re} of 2.24 ± 0.23°C and 2.15 ± 0.28°C ($p = 0.461$) for the F and the M groups respectively. Furthermore, T_{re} ($p = 0.898$), T_{es} ($p = 0.361$), T_{ac} ($p = 0.909$) were similar between groups at the end of exercise. Finally, $\bar{T}_{sk}$ ($p = 0.505$) at the end of exercise was similar between groups (Table 2).

Water Immersion

Before the start of immersion, T_{re} ($p = 0.575$), T_{es} ($p = 0.171$), T_{ac} ($p = 0.631$) and $\bar{T}_{sk}$ ($p = 0.200$) did not differ between groups. Immersion times were significantly different between groups (10.89 ± 4.49 min for F, and 18.13 ± 4.47 min for M, $p = 0.003$). Time to reach a T_{re} of 39°C ($p = 0.178$) and 38.5°C ($p = 0.097$) were similar between groups but the times to reach a T_{re} of 38°C ($p = 0.035$) and 37.5°C ($p = 0.003$) were significantly longer in males (Fig.1). In addition, significant differences occurred in the overall core cooling rates for T_{re} ($p = 0.001$) and T_{ac} ($p = 0.025$), while the overall core cooling rates for T_{es} ($p = 0.067$) tended ($0.05 < p < 0.10$) to show a difference between males and females (Table 3). Decreases in $\bar{T}_{sk\text{-}im}$ at the beginning of immersion became gradually less as a function of

time ($p < 0.001$) and were significantly different between males and females ($p = 0.001$) from the first minute to the end of the mean immersion period for females (Fig 2). Similarly, decreases in $\overline{H}_{sk}$ at the beginning of immersion became gradually less as a function of time ($p < 0.001$) but were not significantly different between males and females ($p = 0.180$) for the duration of the immersion period (Fig. 2). Furthermore, decreases in $\dot{M}$ became less as a function of time ($p < 0.001$) and were not different ($p = 0.307$) until the mean end immersion time for females. It is to note that $\dot{M}$ was elevated gradually in males after the 10^{th} minute of immersion (Fig 3).

Finally, when pooling data from both sexes, the T_{re} overall cooling rates correlated significantly with the A_D/LBM ($r = 0.70$, $p = 0.001$) (Fig.4).

DISCUSSION

The main finding of this study was that previously hyperthermic females had greater T_{re}, T_{es} and T_{au} cooling rates and a shorter time to reach a T_{re} of 38°C and 37.5°C. Despite the fact that both sexes were matched for A_D/M and that the female group had a ~10% greater body fat percentage, females cooled ~ 1.7 times faster than males even tough both sexes had a similar heat loss response. However, females did have a smaller amount of lean body mass and a smaller body surface area to lean body mass ratio. Although possible physiological differences may also account for these sex-related differences, our results suggest that physical differences related to lean body mass are mainly responsible for the differences observed in core cooling rates.

The main avenue for heat transfer during CWI is through conduction. The heat transfers through layers of muscle, adipose tissue and skin to the environment. However, this transfer depends greatly on the surface area across which heat is exchanged. In this manner, the relative amount of heat loss and heat produced are based on the surface area of the body relative to its total mass (i.e. the surface-area-to-mass ratio - A_D/M -). Typically, females have a larger A_D/M, and therefore, would be expected to cool at quicker rates than normothermic males (20). Sloan and Keatinge (1973) have shown that the rates of core cooling of young males and females immersed in 20°C water were mainly accounted for by the discrepancy between sexes in A_D/M (26). Our data suggest that the A_D/M does not affect the core cooling rates of hyperthermic individuals, since the females cooled faster than males despite having the same A_D/M.

Females also had a greater core cooling rate despite having a 10% greater body fat compared to males. Although the greater percentage of body fat could possibly explain the lower skin temperature for females during the CWI (15), this also suggests that greater levels of adipose tissue insulation had little effect in reducing the rate of heat transfer in previously hyperthermic females. This is in accordance with Lemire et al. (2008) that showed the non-effect of body adiposity on heat loss in previously hyperthermic individuals (19). They concluded that the post-exercise thermal status associated with exercise-induced hyperthermia lead to the lack of influence of body adiposity on subsequent core cooling rates in hyperthermic individuals (19).

McArdle et al. (1984) found the metabolic response of males and females to be similar during immersion at rest over the first 1°C drop in core temperature. However, they showed that males have a greater metabolic response than females when core

temperature decreased by more than 1°C (20). In the present study, our data also show no differences in metabolic response between males and females during the first part of the CWI (i.e. until T_{re} reached 38.5°C). The females exited the cold water around the 11th minute mark (i.e. T_{re} was 37.5°C for the females), which corresponded to the increase in metabolic heat production of the males which hadn't reach a T_{re} of 37.5°C. This suggests that hyperthermic males respond with an increased metabolic heat production at a greater core temperature compared to females during CWI. It has been suggested that males might be more sensitive to decreases in skin temperature than females during CWI which would result in shivering at a greater core temperature compared to females (14, 15). In addition, this could explain in part why the immersion time for the males was much longer since greater metabolic heat production lowers the net heat loss during CWI and therefore reduce the core cooling rates.

Looking at sex differences in reaction to cold stress, it has been suggested that mass and muscle mass is significantly related to the rate of core cooling rates and to increases in metabolic rate (3). Rather, differences in response to cold stress between sexes have been suggested to be caused by a larger lean body mass in males; and that if the lean body mass is taken into account, the sex-related differences in thermogenic response should be greatly reduced (3). As such, it would be logical to take into account the surface area across which the heat is dissipated and the factor that reduces the sex-related differences in thermal response to cold stress; the body-surface-area-to-lean-body-mass ratio (A_D/LBM). For example, we examined a female and a male from each group that had almost identical A_D/LBM (322 cm^2/kg of *LBM* for the M, and 323 cm^2/kg of *LBM* for the F). Both the male and females had a similar response (0.09°C/min for the F, and 0.11°C/min for the M)

during the CWI, which resulted in similar times of immersion (21.50 minutes for the F, vs. 19.50 minutes for the M).

To better examine the relation between our T_{re} cooling rates and the A_D/LBM, we correlated both variables independently of sex (see Fig. 5) and we observed a good correlation (r = 0.70). Tikuisis et al. (2000) previously showed similar results between core cooling rates (ΔT_{re}) and surface area (m^2) to volume (m^3) ratio, showing a correlation of r = 0.42 (29), albeit our result show a better correlation. Taking into consideration that our data does not reflect a full range of possible A_D/LBM, we suggest that this ratio can possibly explain core cooling variations in hyperthermic individuals, but future studies are invited to deal with a wide spectrum of A_D/LBM within the same sex to further elucidate this point.

Physiological considerations

A study by Kenny and Jay (2007) showed a greater and more prolonged elevation in core temperature in active and inactive muscle temperatures in females following dynamic exercise (17). This was explained by a greater decrease in mean arterial blood pressure caused by an increase in blood pooling in the previous active limbs in females. This hyperemia elucidated a greater peripheral muscle temperature, providing possibly a greater drive for heat loss in females. Although the physical differences between sexes are of primary concern to explain the differences in core cooling rates, these reported physiological sex-related differences in muscle temperature and blood pooling following exercise might influence core cooling rates during a subsequent CWI and should not be discarded in future studies.

Practical considerations

Proulx et al. (2003) reported an average T_{re} cooling rate of 0.35°C/min in 2°C water and 0.19°C/min in 8°C water, while Clements et al. (2002) reported T_{re} cooling rates of 0.15°C/min in 5°C water in previously hyperthermic individuals. Our results show similar average T_{re} cooling rates of 0.22°C/min and 0.12°C/min for females and males respectively. The average of our results (0.17°C/min) is comparable with previous studies The core cooling rates for the males and females in this study agree with the current guideline stating that an effective treatment for exertional heat stress should provide a T_{re} cooling rate of at least 0.10°C/min (9, 28). This suggests that no changes to the proposed standards are required for males and females. However, it should be considered that immersion times will differ between sexes.

CONCLUSION

Previously rendered hyperthermic females showed a ~ 1.7 times faster core cooling compared to males when immersed in a 2°C circulated water bath. This was evidence by greater core cooling rates, lower skin temperatures and shorter times to reach predetermined core temperatures. We suggest that the A_D/LBM might be a factor, independent of sex, contributing to the differences in the core cooling rates in previously hyperthermic males and females. Further investigations on this topic are needed to confirm this possible interaction.

ACKNOWLEDGEMENTS

This research was supported by the Natural Sciences and Engineering Research Council (Grant # RGPIN-298159-2004, held by Dr. Glen P. Kenny). Dr. Glen P. Kenny was supported by a University of Ottawa Research Chair Award. The authors wish to thank all the participants who volunteered in the study.

REFERENCES

1. Al-Aska AK, Abu-Aisha H, Yaqub B, Al-Harthi SS, and Sallam A. Simplified cooling bed for heatstroke. *Lancet* 1: 381, 1987.
2. Anderson GS. Human morphology and temperature regulation. *International journal of biometeorology* 43: 99-109, 1999.
3. Anderson GS, Ward R, and Mekjavic IB. Gender differences in physiological reactions to thermal stress. *European journal of applied physiology and occupational physiology* 71: 95-101, 1995.
4. Armstrong LE, Crago AE, Adams R, Roberts WO, and Maresh CM. Whole-body cooling of hyperthermic runners: comparison of two field therapies. *The American journal of emergency medicine* 14: 355-358, 1996.
5. Baker PT, and Daniels F, Jr. Relationship between skinfold thickness and body cooling for two hours at 15 degrees C. *J Appl Physiol* 8: 409-416, 1956.
6. Behnke AR, and Wilmore JH. *Evaluation and Regulation of Body Build and Composition.* NJ: Prentice-Hall, 1974.
7. Brooks GA, Fahey TD, White TP, and Baldwin KM. *Exercise Physiology: Human bioenergetics and its application.* Mountain View, CA: Mayfield, 2000.
8. Cannon P, and Keatinge WR. The metabolic rate and heat loss of fat and thin men in heat balance in cold and warm water. *The Journal of physiology* 154: 329-344, 1960.
9. Casa DJ, Armstrong LE, Ganio MS, and Yeargin SW. Exertional heat stroke in competitive athletes. *Current sports medicine reports* 4: 309-317, 2005.
10. Casa DJ, McDermott BP, Lee EC, Yeargin SW, Armstrong LE, and Maresh CM. Cold water immersion: the gold standard for exertional heatstroke treatment. *Exercise and sport sciences reviews* 35: 141-149, 2007.
11. Clements JM, Casa DJ, Knight J, McClung JM, Blake AS, Meenen PM, Gilmer AM, and Caldwell KA. Ice-Water Immersion and Cold-Water Immersion Provide Similar Cooling Rates in Runners With Exercise-Induced Hyperthermia. *J Athl Train* 37: 146-150, 2002.
12. Dubois D, and Dubois E. Clinical calorimetry. A formula to estimate the approximate surface area if height and weight be known. *Arch Int Med* 17: 863-871, 1916.
13. Gaffin SL, Gardner JW, and Flinn SD. Cooling methods for heatstroke victims. *Annals of internal medicine* 132: 678, 2000.
14. Graham TE. Thermal, metabolic, and cardiovascular changes in men and women during cold stress. *Medicine and science in sports and exercise* 20: S185-192, 1988.

15. Graham TE, Viswanathan M, Van Dijk JP, Bonen A, and George JC. Thermal and metabolic responses to cold by men and by eumenorrheic and amenorrheic women. *J Appl Physiol* 67: 282-290, 1989.
16. Howe AS, and Boden BP. Heat-related illness in athletes. *The American journal of sports medicine* 35: 1384-1395, 2007.
17. Kenny GP, Jay O, Zaleski WM, Reardon ML, Sigal RJ, Journeay WS, and Reardon FD. Postexercise hypotension causes a prolonged perturbation in esophageal and active muscle temperature recovery. *American journal of physiology* 291: R580-588, 2006.
18. Kollias J, Barlett L, Bergsteinova V, Skinner J, Buskirk ER, and Nicholas W. Metabolic and thermal responses of women during cooling in water. *J Appl Physiol* 36: 577-580, 1974.
19. Lemire B, Gagnon D, Jay O, Dorman L, Ducharme MB, and Kenny GP. Influence of adiposity on cooling efficiency in hyperthermic individuals. *European journal of applied physiology* 104: 67-74, 2008.
20. McArdle WD, Magel JR, Gergley TJ, Spina RJ, and Toner MM. Thermal adjustment to cold-water exposure in resting men and women. *J Appl Physiol* 56: 1565-1571, 1984.
21. McArdle WD, Toner MM, Magel JR, Spina RJ, and Pandolf KB. Thermal responses of men and women during cold-water immersion: influence of exercise intensity. *European journal of applied physiology and occupational physiology* 65: 265-270, 1992.
22. Mekjavic IB, and Rempel ME. Determination of esophageal probe insertion length based on standing and sitting height. *Journal of Applied Physiology* 69: 376, 1990.
23. Nishi Y. *Measurement of thermal balance in man. In: Bioengineering, thermal physiology and comfort.* New York, NY: 1981.
24. Pivarnik JM, Marichal CJ, Spillman T, and Morrow JR, Jr. Menstrual cycle phase affects temperature regulation during endurance exercise. *J Appl Physiol* 72: 543-548, 1992.
25. Proulx CI, Ducharme MB, and Kenny GP. Effect of water temperature on cooling efficiency during hyperthermia in humans. *J Appl Physiol* 94: 1317-1323, 2003.
26. Sloan RE, and Keatinge WR. Cooling rates of young people swimming in cold water. *J Appl Physiol* 35: 371-375, 1973.
27. Smith JE. Cooling methods used in the treatment of exertional heat illness. *British journal of sports medicine* 39: 503-507; discussion 507, 2005.
28. Tek D, and Olshaker JS. Heat illness. *Emergency medicine clinics of North America* 10: 299-310, 1992.
29. Tikuisis P, Jacobs I, Moroz D, Vallerand AL, and Martineau L. Comparison of thermoregulatory responses between men and women immersed in cold water. *J Appl Physiol* 89: 1403-1411, 2000.
30. Toner MM, Sawka MN, Foley ME, and Pandolf KB. Effects of body mass and morphology on thermal responses in water. *J Appl Physiol* 60: 521-525, 1986.
31. Webb P. Daily activity and body temperature. *European journal of applied physiology and occupational physiology* 66: 174-177, 1993.
32. Yaqub B, and Al Deeb S. Heat strokes: aetiopathogenesis, neurological characteristics, treatment and outcome. *Journal of the neurological sciences* 156: 144-151, 1998.

Table 1. Mean physical characteristics for the female (F, n = 9) and male (M, n = 10) groups.

Groups	Age (yr)	Height (m)	Weight (kg)	Body fat (%)	LBM (kg)	A_D/M ($cm^2 \cdot kg^{-1}$)	A_D/LBM ($cm^2 \cdot kg^{-1}$)	$\dot{V}O_{2max}$ ($mlO_2 \cdot kg^{-1} \cdot min^{-1}$)
F	24 ± 3	165.4 ± 6.8*	64.2 ± 9.3*	23.9 ± 6.1*	48.4 ± 4.5*	268 ± 19	353 ± 17*	51.0 ± 5.3*
M	25 ± 4	177.5 ± 5.8	73.6 ± 9.4	13.8 ± 2.9	63.4 ±7.2	261± 16	302 ± 15	65.2 ± 6.4

Values are mean ± SD. LBM, Lean Body Mass; A_D/M, body surface area to mass ratio; A_D/LBM, body surface area to lean body mass ratio; $\dot{V}O_{2max}$, maximal oxygen consumption.

*Significantly different from males, $p \leq 0.05$.

Table 2. Mean Results Exercise Period for the female (n = 9) and male (n = 10) groups.

	Females		Males	
	Baseline Temperature (°C)	**End Exercise Temperature (°C)**	**Baseline Temperature (°C)**	**End Exercise Temperature (°C)**
T_{re}	37.32 ± 0.22	39.56 ± 0.07	37.42 ± 0.27	39.57 ± 0.06
T_{es}	37.21 ± 0.17	40.07 ± 0.53	37.27 ± 0.34	39.85 ± 0.36
T_{au}	36.82 ± 0.19	39.59 ± 0.46	36.75 ± 0.46	39.62 ± 0.55
T_{sk}	32.40 ± 0.66	37.50 ± 1.67	31.85 ± 2.03	36.92 ± 1.46

Values are mean ± SD. T_{re}, rectal temperature; T_{es}, esophageal temperature; T_{au}, aural canal temperature; T_{sk}, mean skin temperature.

Table 3. Mean Core Cooling Rates for the female (n = 9) and male (n = 10) groups.

	Females Cooling rates (°C/min)	**Males Cooling rates (°C/min)**
$\mathbf{T_{re}}$	0.22 ± 0.07	0.12 ± 0.03*
$\mathbf{T_{es}}$	0.36 ± 0.17	0.22 ± 0.08^{t}
$\mathbf{T_{au}}$	0.32 ± 0.10	0.22 ± 0.06*

Values are mean ± SD. T_{re}, rectal temperature; T_{es}, esophageal temperature; T_{au}, aural canal temperature;
*Significantly different from females, $p \leq 0.05$
^{t} Trend to be significantly different , $0.05 \leq p \leq 0.10$

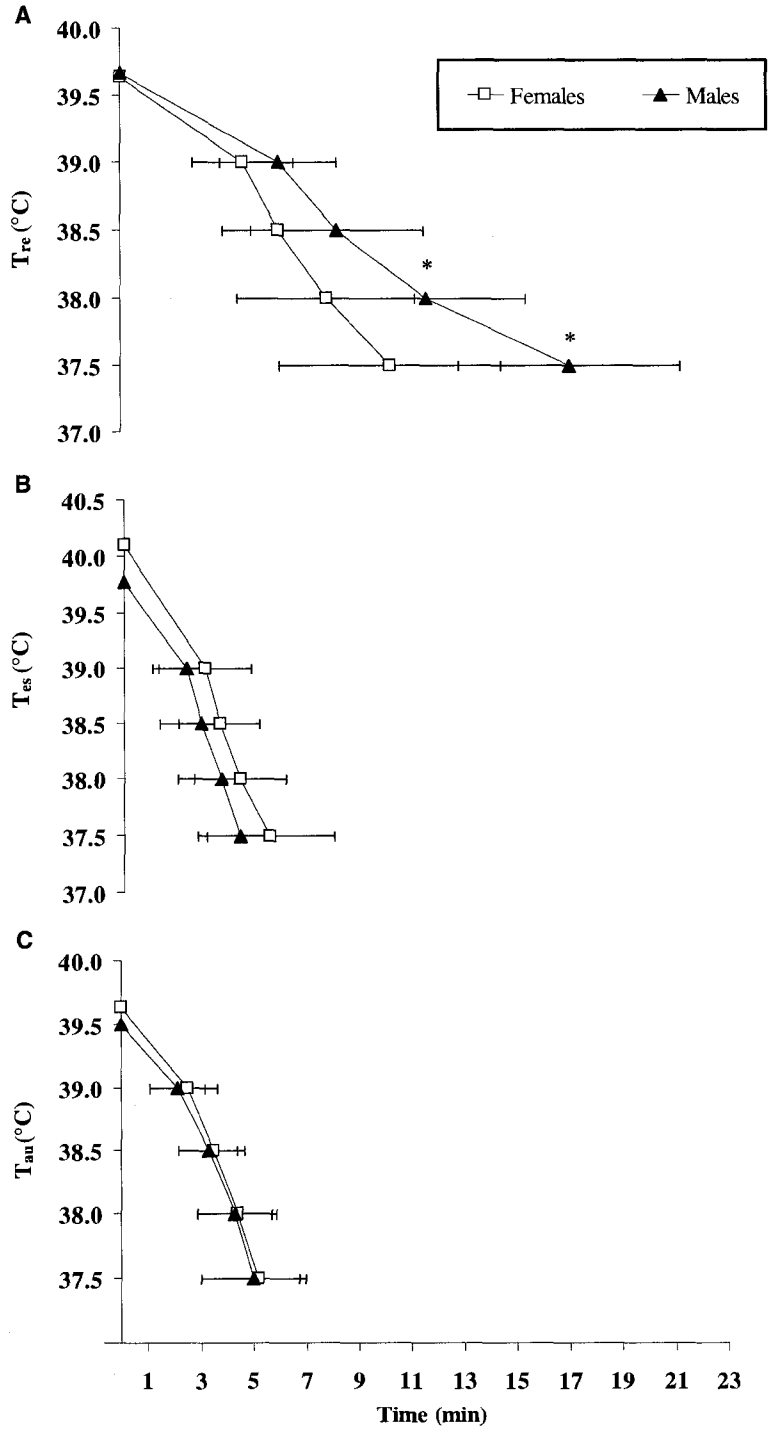

Figure 1. Time to reach ± SD, 39.5°C, 38.5°C and 37.5°C for rectal (T_{re}, panel A), esophageal (T_{es}, panel B) and aural canal (T_{au}, panel C) temperatures for the female (□) and male (▲) groups. Values are mean ± SD.
*Significantly different from females, $p \leq 0.05$

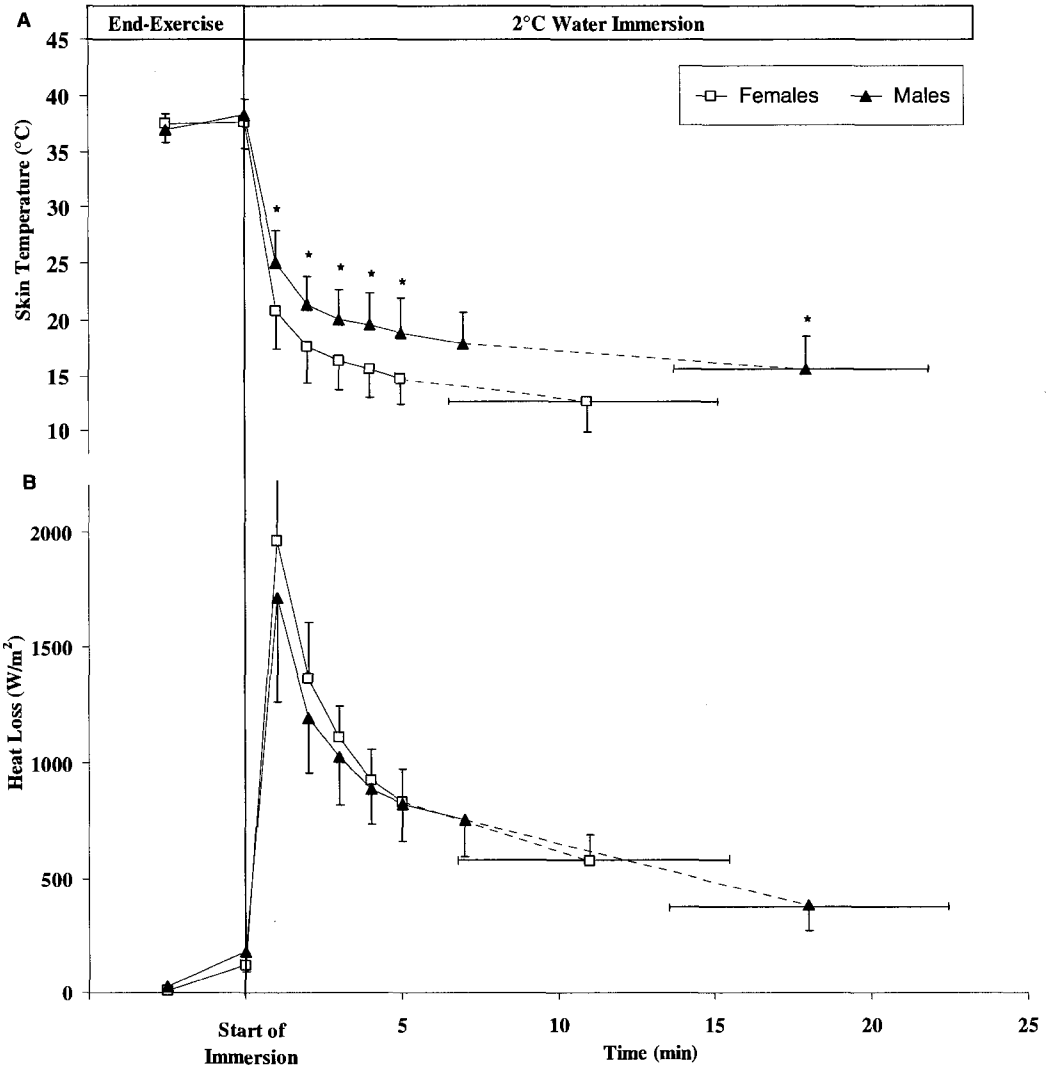

Figure 2. Mean ± SD of skin temperature (panel A) and heat loss (panel B) for the female (□) and male (▲) groups during a 2°C water immersion. Mean (± SD) data presented for the average end-immersion time.
*Significantly different from females, $p \leq 0.05$.

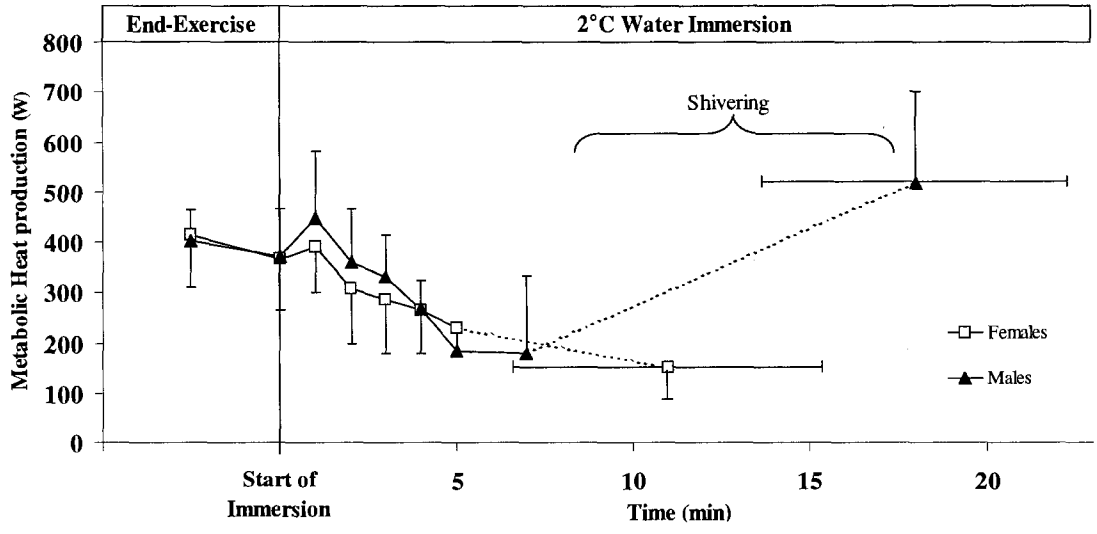

Figure 4. Mean ± SD of metabolic heat production during immersion in a 2°C circulated water bath female (□) and male (▲) groups during a 2°C water immersion. Mean (± SD) data presented for the average end-immersion time. Participants were removed from the water when their rectal temperature reached 37.5°C. Increasing metabolic production above 300 W at the 10th minute corresponded with the visual observation of moderate to intense shivering.

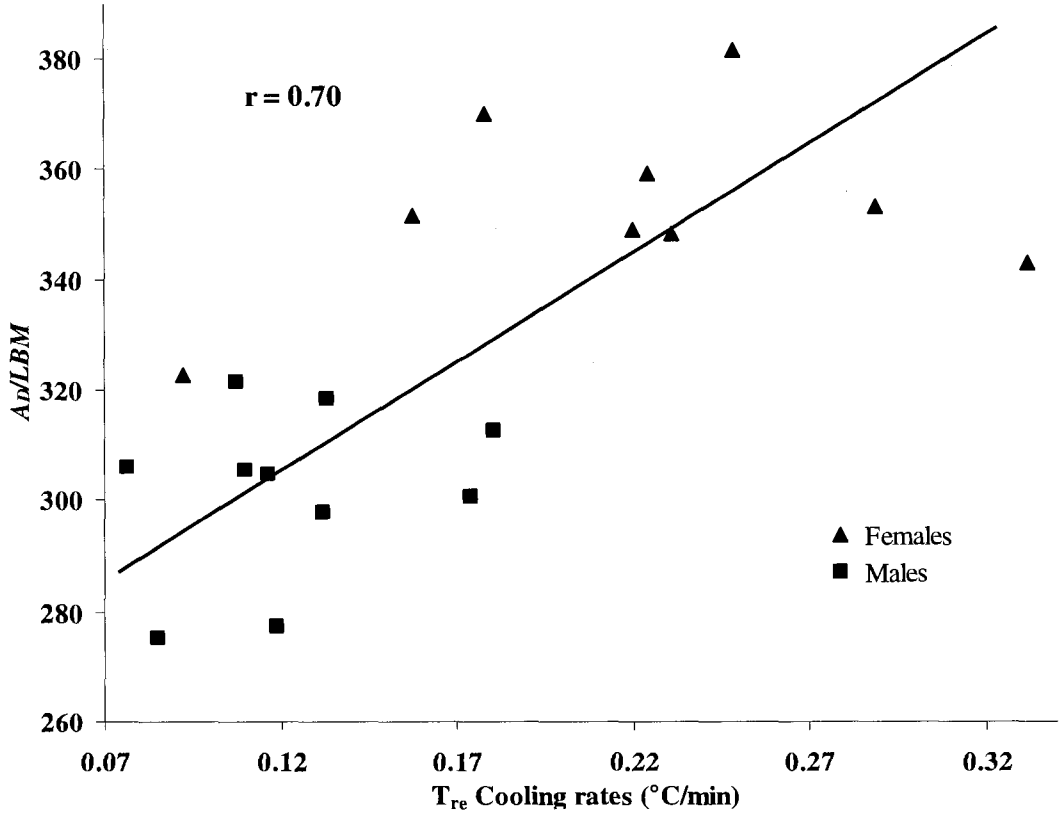

Figure 5. Correlation between A_D/LBM and T_{re} overall cooling rates.

3.1 Article 2

The Effectiveness of Safe-Cooling Limits of Hyperthermic Individuals

The Effectiveness of Safe-Cooling Limits of Hyperthermic Individuals

Bruno Lemire, Glen Kenny

Centre for Human and Environmental Physiology Research, School of Human Kinetics, Faculty of Health Sciences, University of Ottawa, Ottawa, Ontario, Canada, K1N 6N5 and

Running head: Hyperthermia and cold water immersion

Address for correspondence:

Dr. G.P. Kenny
University of Ottawa
School of Human Kinetics
125 University,
Montpetit Hall
Room 367
Ottawa, Ontario, Canada
K1N 6N5
(613) 562-5800 ext. 4282
(613) 562-5149 (fax)
E-mail: gkenny@uottawa.ca

ABSTRACT

This study evaluated the proposed guidelines from Proulx et al. (2006) interpolated data, that an exit rectal temperature (T_{re}) of 38.6°C would prevent any future 'afterdrop' in rectal and esophageal (T_{es}) temperature following a cold water immersion (2°C) in previously hyperthermic individuals. Ten (6 males and 4 females) participants took part in two similar experimental trials. Participants exercised at 65% of their VO_{2max} at an ambient temperature of 40.0°C until T_{re} increased to 39.5°C. Following exercise, participants were immersed in a 2.0°C circulated water bath until T_{re} decreased to 38.6°C in one trial and 37.5 °C in the second trial. Both trials elucidated similar T_{re} cooling rates ($p = 0.231$). Decreases in T_{re} during the recovery became gradually less as a function of time ($p < 0.001$) and was significantly greater in the 37.5 °C trial ($p < 0.001$). Also, decreases in T_{es} up to the 4th minute of recovery were not different between trials ($p = 0.084$). However, from the 5th minute to the 11th minute, T_{es} was significantly lower for the 37.5°C trial ($p = 0.031$). From the 13th minute to the end recovery time, T_{es} was not significantly different across groups ($p = 0.115$).

Conclusion: We conclude that an exit rectal temperature of 38.6°C is sufficient to induced rapid decrease in core temperature while preventing an excessive 'afterdrop' after the cold water immersion.

Keywords: cold water immersion; exercise-induced hyperthermia; core temperature; afterdrop.

INTRODUCTION

Cold water immersion (CWI) has emerged in the last few years as the modality of choice for the treatment of exertional heat stress (2, 28). The high conductive power of water (630.5 $mW/m^2/°K$) compared with air (air 26.2 $mW/m^2/°K$), in combination with the greater temperature gradient between the core and the water makes it an ideal environment to lose heat (2). Hence, immersion in cold water for an extended period of time may lead to serious complications related to hypothermia if no appropriate exit criteria are adhere to. Studies show that severe hypothermia can occur if hyperthermic individuals are overcooled (2, 9, 10, 16, 22). To date, there is no consensus on an appropriate exit core temperature.

For example, Casa et al. (2007) suggested that heat stressed individuals should be removed from the water when the rectal temperature (T_{re}) has reached 39.0°C (2). If T_{re} is not available, they suggest that cooling should cease after 15-20 minutes (2). Others suggest that exit T_{re} should be between 38.9°C to 37.5°C (9, 10, 16, 25). Recently, Proulx et al. (2006) showed that 75% of the heat gain during exercise is eliminated when T_{re} of a previously hyperthermic individual immersed in cold water is at 39.3°C (22). Based on the interpolation of their findings, they proposed that hyperthermic individuals should be taken out of the water at a T_{re} of 38.6°C. This was believed to result in dissipation of 100% of the heat produced by previous exercise (22). However, these recommendations have not been evaluated in an experimental setting.

Differences in the indices of core temperature used during CWI must be considered when developing appropriate guidelines. Esophageal and rectal temperatures are the most commonly used core temperature measurements; however they often show different outcomes (3, 17, 26). The core temperature measurement chosen will greatly influence the

diagnosis of the thermal status of an individual. For example, T_{es} is a good measure of the mixed central blood (26) and is highly reflective of the surrounding changing environment (3, 5, 17). T_{es} is reported to often be 1°C higher or lower then T_{re} depending on the environment and physical activity level (22). In contrast, T_{re} is a good measurement of the inner visceral area, however it is usually delayed and often affected by the postural stance of the individual (18). The guidelines proposed by Proulx et al. (2006) only considered rectal temperature. As such, it remains unclear how the exit temperature of 38.6°C rectal will affect the T_{es} during the recovery.

The primary purpose of this study was to compare the response of core cooling rates and 'afterdrop' during whole-body cooling following exercise-induced hyperthermia as a function of the proposed exit temperature of 38.6°C versus a typical exit temperature of 37.5°C. We evaluated the hypothesis that the exit rectal temperature of 38.6°C would prevent T_{es} and T_{re} from excessive afterdrop following CWI (2°C).

METHODS

Participants

Following approval of the experimental protocol from the University of Ottawa Research Ethics Committee, ten healthy (4 females, 6 males) and physically active participants volunteered and gave written consent to participate in this study. Five to seven days before the experiments, maximal oxygen consumption ($\dot{V}O_{2max}$) was measured as well as body fat percentage of each participant using hydrostatic weighing technique. $\dot{V}O_{2max}$ was measured during a progressive treadmill running protocol. The $\dot{V}O_{2max}$ data were used to select the submaximal workload for the experimental exercise phase of the study. The physical characteristics of the subjects are presented in Table 1.

Instrumentation

Esophageal temperature (T_{es}) was measured by placing a pediatric thermocouple probe of approximately 2 mm in diameter (Mon-a-therm Nasopharyngeal Temperature Probe, Mallinckrodt Medical, St-Louis, MO, USA) through the participant's nostril while they were asked to sip water through a straw. The location of the probe tip in the esophagus was estimated to be at the level of the eighth and ninth thoracic vertebrae (14).

Rectal temperature (T_{re}) was measured using a pediatric thermocouple probe (Mon-a-therm General Purpose Temperature Probe, Mallinckrodt Medical, St-Louis, MO, USA) inserted to a minimum of 12 cm past the sphincter.

Skin temperature was monitored at 12 sites by Type T thermocouples integrated into heat-flow sensors (Concept Engineering, Old Saybrook, CT). These were

commercially available heat flow disks that can read both the heat loss and the skin temperature. The skin temperature is measured from the thermocouple that is integrated to the disk. The area-weighted mean skin temperature ($\bar{T}_{sk}$) and mean heat loss ($\bar{H}_{sk}$) were calculated by assigning the following regional percentages: head 6%, bicep 9%, forearm 6%, hand 2%, chest 9.5%, abdomen 9.5%, upper back 9.5%, lower back 9.5%, quadricep 10%, hamstring 10%, front calf 9.5%, back calf 9.5% (8). The mean skin temperature (as well as the mean heat loss) was thus calculated using the following equation: T_{sk} = (0.06 x $T_{forehead}$) + (0.09 x T_{bicep}) + (0.06 x $T_{forearm}$) + (0.02 x T_{hand}) + (0.095 x T_{chest}) + (0.095 x $T_{abdomen}$) + (0.095 x $T_{upper\ back}$) + (0.095 x $T_{lower\ back}$) + (0.10 x $T_{quadricep}$) + (0.10 x $T_{hamstring}$) + (0.095 x $T_{front\ calf}$) + (0.095 x Tback calf). During the water immersion, the average skin temperature for the sites immersed in water was calculated by assigning the following regional percentages ($\bar{T}_{sk\text{-}im}$): upper back 12%, lower back 12.5%, abdomen 12.5%, bicep 9.5%, forearm 9.5%, hand 2%, quadricep 12%, hamstring 12%, front calf 9%, back calf 9% (8, 11). Since the head and the chest were not entirely immersed in water for every subject, they were therefore not used to calculate $\bar{T}_{sk\text{-}im}$. Temperatures were collected and digitized (IBM ThinkCentre M50) with LabVIEW software (Version 7.0, National Instruments, TX, USA) at 5-s intervals, displayed graphically on the computer screen and recorded in spreadsheet format on a hard (IBM ThinkCentre M50) with LabVIEW software (Version 7.0, National Instruments, TX, USA).

Oxygen consumption (VO_2) was continuously recorded during the immersion period with the portable Jaeger – Oxycon mobile system (VIASYS healthcare, Hoechberg, Germany). Millilitres of O_2 were displayed on the laptop computer screen and recorded

every minutes on an excel spreadsheet. Rate of metabolic heat production ($\dot{M}$) was calculated from minute-average values for $\dot{V}O_2$ and RER during the CWI using the following equation:

$$\dot{M} \equiv \left(\dot{V}O_2 \bullet \left[\frac{RER - 0.7}{0.3} e_c + \frac{1 - RER}{0.3} e_f \right] \right) \text{.....Watts.....} \quad (20).$$

Where: e_c is the caloric equivalent per liter of oxygen for the oxidation of carbohydrates (21.13 kJ), and e_f is the caloric equivalent per liter of oxygen for the oxidation of fat (19.62 kJ).

Experimental protocol

Each subject participated in two experimental trials. All trials were performed at the same time of day to avoid circadian variation in skin and core temperatures (29). Females were tested in the follicular phase to avoid any disruptions in baseline temperature and influence on exercise-induced hyperthermia (21). Participants were asked to fast at least 3 h prior to experimentation, and were given instructions on what the last meal should be (i.e. Two brown toast, butter and orange juice). Water ingestion was permitted *ad libitum* during this time to ensure good hydration level. Upon arrival at the laboratory, subjects clothed in shorts, and athletic shoes were fitted with the appropriate instruments.

After instrumentation, the participants remained resting in the upright seated posture for 15 minutes at an ambient air temperature of 25.0°C and a relative humidity of 18%. They then entered a temperature controlled chamber regulated at an ambient temperature of 40.0°C where they ran on a treadmill at 65% of their predetermined $\dot{V}O_{2max}$ until their rectal temperature (T_{re}) reached 39.5°C or until volitional fatigue. Volitional

fatigue was defined as an inability to maintain the required exercise intensity and/or a verbal manifestation from the participant to stop. Participants were then transferred (~1.5min) and immersed up to the clavicles in a circulated water bath maintained at 2.0°C. Prior to entering the circulated water bath, participants were fitted with neoprene mitts and socks to reduce discomfort. Participants remained in the water until T_{re} reached 37.5°C for one of the trials and 38.6 °C for the other. The order in which the trials were performed was decided randomly.

Statistical Analyses

For each trial, baseline resting and end-exercise temperatures, exercise duration, cooling times and overall cooling rates were calculated for T_{re}, T_{es} and T_{ac}. Area-weighted mean $\overline{H}_{sk}$, pre-cooling $\overline{T}_{sk}$, end-cooling $\overline{T}_{sk}$, pre-cooling $T_{sk\text{-}im}$ and end-cooling $T_{sk\text{-}im}$ were also calculated. Statistical analysis was carried out on the data for the two experimental trials with a paired sample T-tests. Furthermore, a two-way mixed model analysis of variance was used to analyze the $\dot{M}$, T_{re} and T_{es} during the recovery from the immersion period using the repeatable factor of time (levels: 1, 3, 5, 7, 9, 11, 13, 15, 17, 19, 21-min) and the non-repeatable factor of the T_{re} exit value (levels: T_{re} = 37.5°C and T_{re} = 38.6°C). The level of significance was set at 0.05 and alpha level was adjusted during multiple comparisons so as to maintain the rate of Type I error at 5% during the Holm-Bonferroni post hoc analysis. All analyses were performed using the statistical software package SPSS 15.0 for Windows (SPSS Inc. Chicago, IL, USA).

RESULTS

Exercise Period

There were no significant differences between conditions in baseline T_{re} ($p = 0.947$) and T_{es} ($p = 0.673$). Exercise time (38.8 ± 9.90 min, for the 38.6°C trial and 35.6 ± 7.43 min, for the 37.5°C trial) taken to reach the experimental withdrawal criterion (T_{re} = 39.5°C) was not different between trials ($p = 0.426$). All subjects met this criterion. Exercise resulted in an increase from baseline in T_{re} of 2.20 ± 0.29°C and 2.18 ± 0.26°C ($p = 0.860$) for the 37.5°C and the 38.6°C trials respectively. T_{re} (39.53 ± 0.04°C, for the 38.6°C trial and 39.53 ± 0.04°C, for the 37.5°C trial, $p = 0.922$) and T_{es} (39.89 ± 0.40°C, for the 38.6°C trial and 39.84 ± 0.30°C, for the 37.5°C trial, $p = 0.778$) were similar between trials at the end of exercise.

Water Immersion

After transfer to the immersion bath, T_{re} ($p = 0.090$) and T_{es} ($p = 0.900$) at the start of immersion did not differ between trials. Times of immersion were significantly different between trials (8.83 ± 2.61 minutes, for the 38.6°C trial and 16.55 ± 5.74 minutes, for the 37.5°C trial, $p < 0.01$). There were no significant differences in the overall cooling rates for T_{re} ($p = 0.231$) between the two trials, however the overall cooling rates for T_{es} ($p = 0.005$) were significantly different. Pre-immersion $\bar{T}_{sk\text{-}im}$ ($p = 0.533$) were similar but end-immersion $\bar{T}_{sk\text{-}im}$ ($p = 0.036$) were significantly different between trials (Table 2). End of immersion T_{re} (38.60 ± 0.05°C, for the 38.6°C trial and 37.55 ± 0.02°C, for the 37.5°C trial, $p < 0.001$) for both trials were obviously significantly different between trials, but the end immersion T_{es} (36.49 ± 1.14°C, for the 38.6°C trial and 36.31 ± 0.81°C, for the 37.5°C

trial, p = 0.689) were not different between trials. In addition $\overline{H}_{sk}$ were different between trials (922 ± 171 W/m^2, for the 38.6°C trial and 741 ± 177 W/m^2, for the 37.5°C trial, p = 0.003). Furthermore, the small increase in $\dot{M}$ at the beginning of recovery became gradually less as a function of time (p = 0.006) and was similar between trials (p =0.288) until the end immersion time of the shorter trial (i.e. 38.6°C) where the $\dot{M}$ slowly increase until their respective end immersion time for the longer trial (i.e. 37.5°C) (Fig.2).

Recovery

The lowest values recorded for T_{re} (37.19 ± 0.71°C, for the 38.6°C trial and 36.47 ± 0.70°C, for the 37.5°C trial, p = 0.016) during the recovery were different between trials, while the difference in T_{es} (36.38 ± 1.13°C, for the 38.6°C trial and 35.78 ± 0.98°C, for the 37.5°C trial, p = 0.072) were nearly significant. Decreases in T_{re} at the beginning of recovery became gradually less as a function of time (p < 0.001) and were significantly different between trials (p < 0.001). No interactions between the absolute values of T_{re} and time were present during the recovery (p = 0.348) (Fig.1). Decreases in T_{es} at the beginning of recovery until the 4th minute were not different between groups (p = 0.101). From the 5th minute to the 11th minute of recovery, T_{es} was significantly lower for the 37.5°C trial (p = 0.035), going under 36°C. From the 13th minute to the end recovery (21 minutes), T_{es} was not significantly different across groups (p = 0.115). Overall no interactions between the absolute values of T_{es} and time were present during the recovery (p = 0.154) (Fig.1).

DISCUSSION

Our findings show that the use of a T_{re} of 38.6°C as an exit criterion for the treatment of previously hyperthermic individuals in 2°C water protects against the possible development of hypothermia. This was evidenced by showing that T_{re} (above 37°C) and T_{es} (above 36°C) remained in a safe range during the recovery of the CWI.

Furthermore, similar levels T_{re} cooling rates were observed in both trials (0.12°C/min for exit T_{re} of 38.6°C, and 0.14°C/min for exit T_{re} of 37.5°C). This was to be expected since the participants were exposed to the same condition in both trials as only the exit T_{re} differed. However, the major difference between trials was the absolute temperature decrease. In fact, T_{re} only decreased on average 0.99°C when the exit temperature was 38.6°C and by 2.12°C when the exit temperature was 37.5°C.

One of the primary goals of any treatment of hyperthermic individuals is to lower core temperature as rapidly as possible. Proulx et al. (2003) showed that 2°C water immersion provided the best core cooling rate as compared to higher water temperatures (8°C, 14°C and 20°C). While it is now recommended that 2°C water is the optimal treatment modality for hyperthermic individuals, the prolonged immersion associated with an exit T_{re} of 37.5°C may result in a level of discomfort that some individuals may not be able to tolerate. In addition to reducing the immersion time and the level of physical discomfort associated with shivering (Fig.2), an exit T_{re} of 38.6°C results in a lesser decrease in core temperature that reduces the risk of subsequent hypothermia (Fig. 1).

At an exit T_{re} of 37.5°C, we show 'afterdrops' of 1.03°C to a T_{re} of 36.47°C, and of 0.32°C to a T_{es} of 35.78°C. Taking into account individuals' differences, we observed a T_{es} of 33.86°C, 35.49°C and 35.29°C in three of our participants. As a result, individuals were

at risk of developing clinical hyperthermia (T_{es} < 35°C). In fact, after 25 minutes of recovery, two participants had to be placed in a hot tub to be re-warmed. In contrast, we show 'afterdrops' of 1.41°C to a T_{re} of 37.19°C, and of 0.22°C to a T_{es} of 36.38°C when the exit T_{re} was of 38.6°C. T_{es} lowest levels were 35.20°C, 35.25°C and 35.26°C however none of the participants were noticeably distressed by these core temperatures. Thus, it would appear that the cold water induced similar afterdrop in both exit temperature but the residual affects are less pronounced since T_{re} was at significantly higher absolute levels when the exit T_{re} of 38.6°C was employed. Despite the fact that T_{es} was at low levels even when an exit temperature of 38.6°C was used, T_{re} remained close to the 'normal' range (~37°C). Hence, using a higher exit temperature as previously suggested by Proulx et al. (2006) provides the rapid cooling necessary to treat hyperthermic individuals while eliminating the risk of further thermal injury.

It is well known that after removal from cold exposure, core temperature can continue to decrease as much 3 to 6°C in humans depending on the time of exposure (7). However, the indices of core temperature used can greatly influence the conclusions drawn when comparing the "afterdrop". We and others have shown that indices of core temperature differ between one another in terms of temporal response and thermal transients (4, 6, 13, 15, 19, 23, 27). Since T_{es} is an indicator of aortic blood temperature (24) and T_{re} is mostly stable in quick changing environments and a good indicator of conductive heat loss through tissue (24), it would therefore be best to monitor both core temperature measurements during CWI. In the field, esophageal temperature is hard to manage; which is why rectal temperature is readily used (1, 2, 28). This been said, if rectal temperature is used to assess the thermal status of an individual, medical staff should be

aware of the different responses of each core temperature measurement during CWI to prevent hypothermia (Fig.1). These important temporal and absolute differences between the core temperature indices may be important to consider when administering treatment to heat stressed individuals.

CONCLUSION

While cold water immersion is the treatment modality of choice in clinical settings for hyperthermia, it is important to avoid overcooling. The afterdrop associated with prolonged whole body cooling is considered to have disastrous physiological consequences on the body. Therefore, we have provided the first study to evaluate safe cooling guidelines for cold water immersion. We conclude that the 'afterdrop' to low levels of core temperatures after CWI is preventable if individuals are taken out of the water at a T_{re} of 38.6°C.

REFERENCES

1. Armstrong LE, Crago AE, Adams R, Roberts WO, and Maresh CM. Whole-body cooling of hyperthermic runners: comparison of two field therapies. *The American journal of emergency medicine* 14: 355-358, 1996.
2. Casa DJ, McDermott BP, Lee EC, Yeargin SW, Armstrong LE, and Maresh CM. Cold water immersion: the gold standard for exertional heatstroke treatment. *Exercise and sport sciences reviews* 35: 141-149, 2007.
3. Cooper KE, and Kenyon JR. A comparison of temperatures measured in the rectum, oesophagus, and on the surface of the aorta during hypothermia in man. *The British journal of surgery* 44: 616-619, 1957.
4. Cooper KE, and Kenyon JR. A comparison of temperatures measured in the rectum, oesophagus, and on the surface of the aorta during hypothermia in man. *British Journal of Surgery* 44: 616-619, 1957.
5. Cranston WI, Gerbrandy J, and Snell ES. Oral, rectal and oesophageal temperatures and some factors affecting them in man. *The Journal of physiology* 126: 347-358, 1954.
6. Cranston WI, Gerbrandy J, and Snell ES. Oral, rectal and oesophageal temperatures and some factors affecting them in man. *Journal of Physiology* 126: 347-358, 1954.
7. Giesbrecht GG, and Bristow GK. Recent advances in hypothermia research. *Annals of the New York Academy of Sciences* 813: 663-675, 1997.
8. Hardy JD, and Dubois EF. The technique of measuring radiation and convection. . *J Nutr* 15: 461-475, 1938.
9. Harker J, and Gibson P. Heat-stroke: a review of rapid cooling techniques. *Intensive Crit Care Nurs* 11: 198-202, 1995.
10. Knochel JP. Environmental heat illness. An eclectic review. *Archives of internal medicine* 133: 841-864, 1974.
11. Layton RP, Mints WH, Jr., Annis JF, Rack MJ, and Webb P. Calorimetry with heat flux transducers: comparison with a suit calorimeter. *J Appl Physiol* 54: 1361-1367, 1983.
12. Lemire B, and Kenny GP. Sex-related differences in cooling rates after exercice-induced hyperthermia. *Thesis article* 2008.
13. Livingstone SD, Grayson J, Frim J, Allen CL, and Limmer RE. Effect of cold exposure on various sites of core temperature measurements. *Journal of Applied Physiology: Respiratory, Environmental & Exercise Physiology* 54: 1025, 1983.
14. Mekjavic IB, and Rempel ME. Determination of esophageal probe insertion length based on standing and sitting height. *J Appl Physiol* 69: 376-379, 1990.
15. Minard D, Copman L, and Dasler AR. Elevation of body temperature in health *Annals of the New York Academy of Sciences* 121: 12-25, 1964.
16. Moran DS, Heled Y, Shani Y, and Epstein Y. Hypothermia and local cold injuries in combat and non-combat situations--the Israeli experience. *Aviation, space, and environmental medicine* 74: 281-284, 2003.
17. Moran DS, and Mendal L. Core temperature measurement: methods and current insights. *Sports medicine (Auckland, NZ* 32: 879-885, 2002.
18. Newsham KR, Saunders JE, and Nordin ES. Comparison of rectal and tympanic thermometry during exercise. *Southern medical journal* 95: 804-810, 2002.
19. Newsham KR, Saunders JE, and Nordin ES. Comparison of rectal and tympanic thermometry during exercise. *Southern Medical Journal* 95: 804-810, 2002.

20. Nishi Y. *Measurement of thermal balance in man. In: Bioengineering, thermal physiology and comfort.* New York, NY: 1981.
21. Pivarnik JM, Marichal CJ, Spillman T, and Morrow JR, Jr. Menstrual cycle phase affects temperature regulation during endurance exercise. *J Appl Physiol* 72: 543-548, 1992.
22. Proulx CI, Ducharme MB, and Kenny GP. Safe cooling limits from exercise-induced hyperthermia. *European journal of applied physiology* 96: 434-445, 2006.
23. Robinson J, Charlton J, Seal R, Spady D, and Joffres MR. Oesophageal, rectal, axillary, tympanic and pulmonary artery temperatures during cardiac surgery. *Canadian Journal of Anaesthesia* 45: 317-323, 1998.
24. Romet TT. Mechanism of afterdrop after cold water immersion. *J Appl Physiol* 65: 1535-1538, 1988.
25. Shapiro Y, and Seidman DS. Field and clinical observations of exertional heat stroke patients. *Medicine and science in sports and exercise* 22: 6-14, 1990.
26. Shiraki K, Konda N, and Sagawa S. Esophageal and tympanic temperature responses to core blood temperature changes during hyperthermia. *J Appl Physiol* 61: 98-102, 1986.
27. Shiraki K, Konda N, and Sagawa S. Esophageal and tympanic temperature responses to core blood temperature changes during hyperthermia. *Journal of applied physiology* 61: 98, 1986.
28. Smith JE. Cooling methods used in the treatment of exertional heat illness. *British journal of sports medicine* 39: 503-507; discussion 507, 2005.
29. Webb P. Daily activity and body temperature. *European journal of applied physiology and occupational physiology* 66: 174-177, 1993.

Table 1. Mean participant characteristics

Group	Age (yrs)	Height (cm)	Weight (kg)	Body Fat (%)	*LBM* (kg)	A_D/M ($cm^2 \cdot kg^{-1}$)	VO_{2max} ($mlO2 \cdot kg^{-1} \cdot min^{-1}$)
n = 10	22 ± 3	171.9 ± 10.0	67.8 ± 10.7	17.1 ± 4.5	56.3 ± 10.1	267 ± 16	59.3 ± 8.7

Values are mean ± SD

LBM, Lean Body Mass; A_D/M, body surface area to mass ratio; VO_{2max}, maximal oxygen consumption.

Table 2. Mean water immersion period results

Group	T_{re} Start of Immersion (°C)	T_{es} Start of Immersion (°C)	Start Skin Temperature (°C)	End Skin Temperature (°C)
37.5°C trial	39.66 ± 0.07	39.82 ± 0.34	36.52 ± 2.08	13.05 ± 2.78
38.6°C trial	39.59 ± 0.10	39.81 ± 0.43	36.00 ± 1.54	15.84 ± 2.78*

Values are mean ± SD for the 37.5°C trial and the 38.6°C trial. (n =10)
*Significantly different from the 37.5°C trial, $p \leq 0.05$.

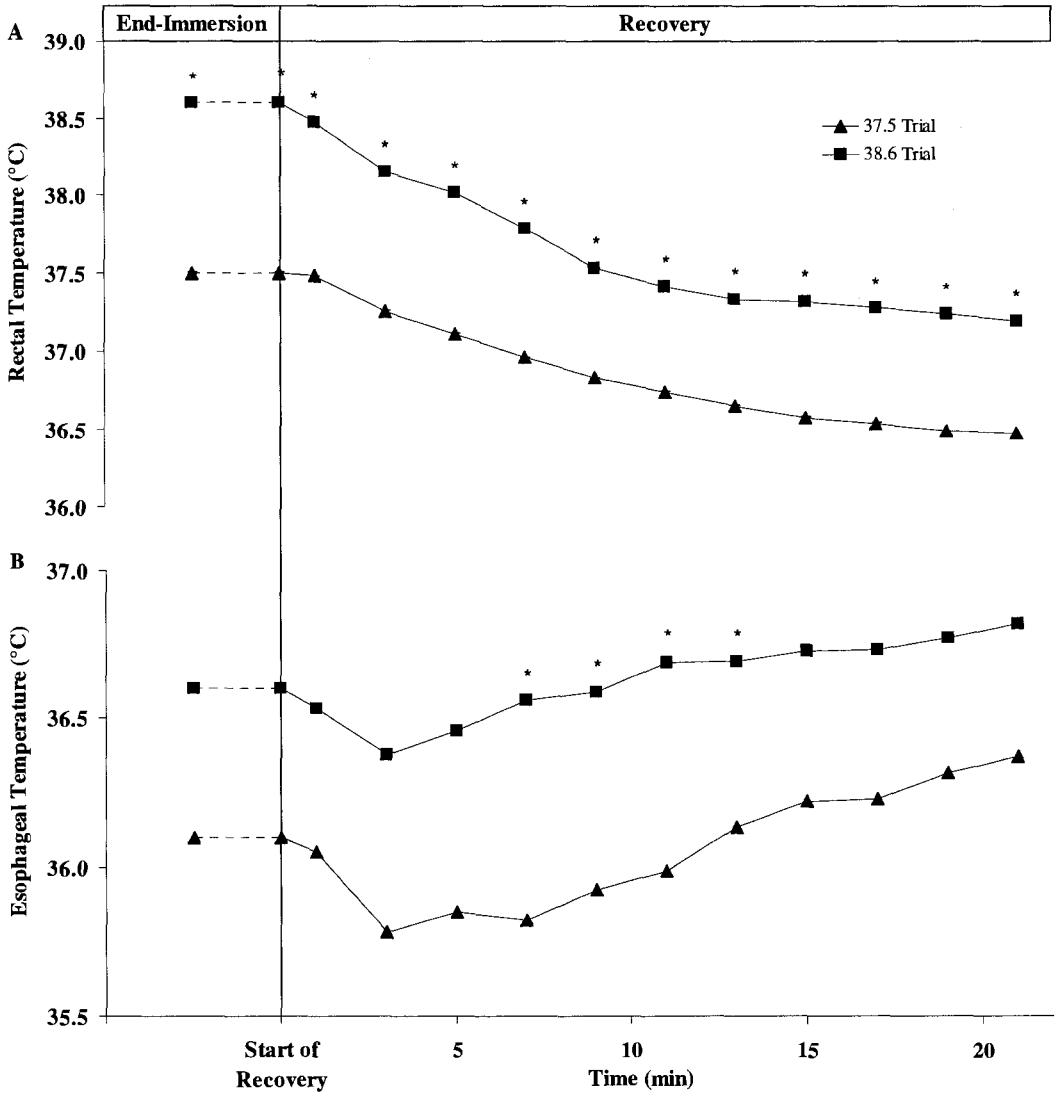

Figure 1. Mean (SE) of T_{re} (panel A) and T_{es} (panel B)during recovery from an immersion in a circulated 2°C water bath for the 37.5°C (▲) and the 38.6°C trial (■) groups.

*Significantly different from the 37.5°C trial, $p \leq 0.05$.

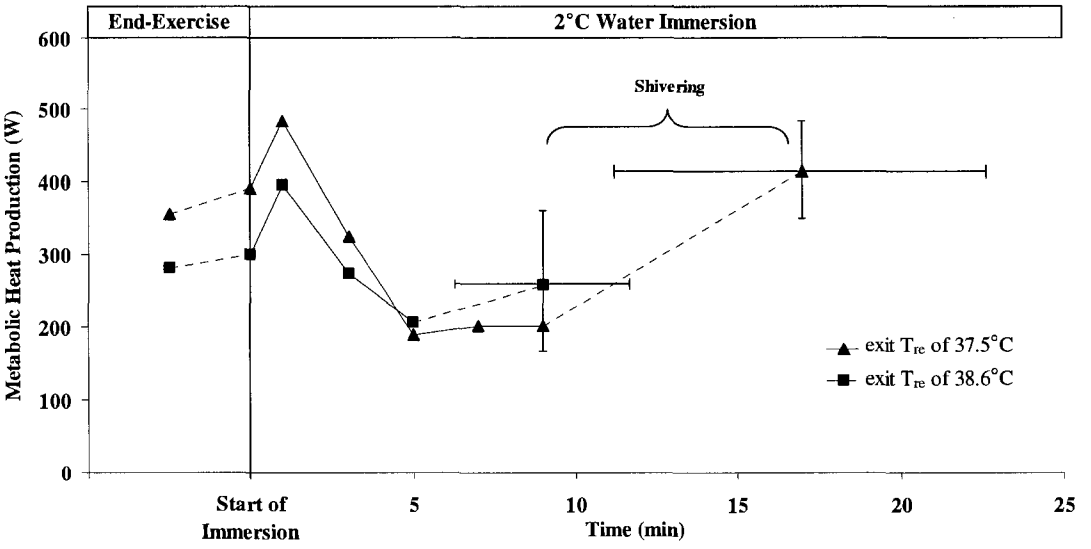

Figure 2. Mean (SE) of $\dot{M}$ during immersion in a 2°C circulated water bath for the 37.5°C (▲) and the 38.6°C trial (■) groups until the last common point. Mean (± SD) data presented for end mean immersion time periods as well as for the end mean metabolic heat production for both groups.

PART THREE: Discussion and Conclusion of Thesis

4.0 Discussion

The main findings of this study were that:

1) Females cooled ~ 1.7 times faster then males when immersed in a 2°C circulated water bath after being hyperthermic when matched for A_D/M. This was evidence by a faster overall core cooling rates, a greater average non-evaporative heat loss as well as a lower skin temperature in females during the CWI.

2) The exit rectal temperature of 38.6°C provided the previously hyperthermic individuals with fast cooling rates and a shorter immersion time, while preventing any major afterdrop in T_{re} and T_{es} (i.e. core temperature < 36°C). We can also conclude that the safe cooling recommendations provided by Proulx et al. (2006) were sufficient and should be considered as the reference for future CWI safety guidelines.

Upon close examination of our results, we show that the first ~10 minutes of immersion were similar for both males and females in terms of core cooling rates and this was paralleled by similar rates of metabolic heat production. Both groups show the usual 'cold shock response' (i.e. increases of 150% in ventilation) upon entry into the cold bath but all participants showed a decline of metabolic heat production after the first few minutes. The female participants had reached their exit rectal temperature criterion around the 11^{th} minute without any sign of increase in metabolic heat production. However, male participants showed a delay in their exit time due primarily to the fact that they showed a progressive increase in the rate of metabolic heat production. This resulted in a reduction in net non-evaporative heat loss. Hence males exited the water around the 18^{th} minute mark. Our responses paralleled those of McArdle et al. (1984), that showed that metabolic

responses between sexes to be similar during immersion over the first 1°C drop in core temperature, while males demonstrated a greater metabolic response when core temperature decreased for more then 1.0°C (76). We show that in the shorter immersion trial, increases in metabolic heat production is avoided and provided a fast core cooling rate. This is in accordance with Proulx et al. (2003) that showed hyperthermic individuals shivered (i.e. increased heart rate) only after the 10^{th} minute mark of immersion (93).

Anderson (1995) showed muscle mass to be significantly related to increases in metabolic rate and the rate of core cooling (2). He concluded that the differences in the responses to cold stress between sexes may be the cause of a larger lean body mass in males. He suggests that if lean body mass is taken into account, the sex-related differences in the thermogenic responses will be greatly reduced (2). If this holds true, then it would be logical to look at factors that take into account the surface area across which the heat is dissipated and the factors that diminish the sex-related differences in thermal response to cold stress; hence the surface-area-to-lean-body-mass ratio (A_D/LBM). No previous study has proposed this ratio as a major factor influencing the core cooling rates in previously hyperthermic individuals. For example, when comparing across sexes, we see that a female (JR) had a similar response to the CWI than our male participants (i.e. similar cooling rates, similar time of immersion). In fact, this female had a much smaller *LBM* than the average males (51.7 kg of *LBM* for the female (JR), and 63.7 kg of *LBM* for the males). The only factor that seemed to be similar across our female (JR) and the males physical characteristics is the A_D/LBM. Our female (JR) showed a A_D/LBM of 323 cm^2/kg of *LBM,* while the average for the males was 302 cm^2/kg of *LBM.* Taking into consideration that the average value for the A_D/LBM for the females was 357 cm^2/kg of *LBM,* this would put our

female (JR) closer to the male group than the female group. To further this point, a smaller male (RSC) had almost an identical A_D/LBM (322 cm^2/kg of *LBM*) to our female (JR). Not surprisingly, they have almost an identical time of immersion (21.50 minutes for the female (JR), vs. 19.50 minutes for the male (RSC).

A similar comparison was done by Tikuisis et al. (2000) between core cooling rates (ΔT_{re}) and surface area (m^2) to volume (m^3) ratio, showing a respectable correlation of $r = 0.42$ (106). The relation between our overall T_{re} cooling rates and the A_D/LBM, independent of sex, shows an even better correlation ($r = 0.70$). Therefore, we suggest that future studies examine a wider range of A_D/LBM in individuals treated with CWI after being rendered hyperthermic by exercise to elucidate a possible relationship between core cooling rates and the A_D/LBM.

A key factor for the successful treatment of individuals suffering from hyperthermia is to provide a fast cooling rate with few complications. We show that cooling individuals in a 2°C water at a T_{re} of 38.6°C prevented the core temperatures (i.e. rectal and esophageal) to drop to dangerous levels. Although these cooling guidelines were suggested earlier by Proulx et al. (2006), they were not yet assessed in a controlled environment. Hence this may explain why these recommendations have not been recognized to date (20). It is important for health professionals to take into account the various measurements and the different temporal variations between the different indices of core temperatures during CWI. For safety reasons, hyperthermic individuals should not be cooled when rectal temperature drops below 38.6°C when immersed in cold water (< 10°C). This research

project has provided evidence that these guidelines are effective for the prevention of hypothermia in previously hyperthermic individuals.

5.0 Conclusion

Based on the findings of our study, we can conclude that;

1) hyperthermic females cool faster than hyperthermic males due to anthropometrical differences. The proposed physical characteristics that influence core cooling rates in previously hyperthermic individuals are not the same as in normothermic individuals (i.e. body fat, A_D/M, total mass...). Taking into consideration that the heat from previous exercise is stored in the previously active and inactive muscles and that body fat does not seem to affect heat loss during CWI, we suggest that the A_D/LBM might be the main factor affecting heat loss in hyperthermic individuals, and;

2) the recommendations suggested by Proulx et al. (2006), stating an exit rectal temperature of 38.6°C should be respected in order to avoid severe 'afterdrop', appears to suitable for previously rendered hyperthermic individuals immersed in cold water.

PART FOUR: References

6.0 References

1. Anderson GS. Human morphology and temperature regulation. *International journal of biometeorology* 43: 99-109, 1999.
2. Anderson GS, Ward R, and Mekjavic IB. Gender differences in physiological reactions to thermal stress. *European journal of applied physiology and occupational physiology* 71: 95-101, 1995.
3. Armstrong L. *Exertionnal Heat Illnesses*. Champaign IL: Human Kinetics, 2003, p. 275.
4. Armstrong LE, Casa DJ, Millard-Stafford M, Moran DS, Pyne SW, and Roberts WO. American College of Sports Medicine position stand. Exertional heat illness during training and competition. *Medicine and science in sports and exercise* 39: 556-572, 2007.
5. Armstrong LE, Crago AE, Adams R, Roberts WO, and Maresh CM. Whole-body cooling of hyperthermic runners: comparison of two field therapies. *The American journal of emergency medicine* 14: 355-358, 1996.
6. Armstrong LE, Curtis WC, Hubbard RW, Francesconi RP, Moore R, and Askew EW. Symptomatic hyponatremia during prolonged exercise in heat. *Medicine and science in sports and exercise* 25: 543-549, 1993.
7. ASHRAE. Physiological principles, comfort and health Atlanta, USA: 1989.
8. Barrow MW, and Clark KA. Heat-related illnesses. *American family physician* 58: 749-756, 759, 1998.
9. Benzinger TH. Heat regulation: homeostasis of central temperature in man. *Physiological reviews* 49: 671-759, 1969.
10. Boulant JA. Role of the preoptic-anterior hypothalamus in thermoregulation and fever. *Clin Infect Dis* 31 Suppl 5: S157-161, 2000.
11. Brake DJ, and Bates GP. Fluid losses and hydration status of industrial workers under thermal stress working extended shifts. *Occup Environ Med* 60: 90-96, 2003.
12. Brooks F, White and Baldwin. *Exercise Physiology: Bienergetics and its applications*. New York: Kirschenbaum: Carla White, 2000, p. 851.
13. Burse RL. Sex differences in human thermoregulatory response to heat and cold stress. *Human factors* 21: 687-699, 1979.
14. Carter R, 3rd, Cheuvront SN, Williams JO, Kolka MA, Stephenson LA, Sawka MN, and Amoroso PJ. Epidemiology of hospitalizations and deaths from heat illness in soldiers. *Med Sci Sports Exerc* 37: 1338-1344, 2005.
15. Carter R, 3rd, Watenpaugh DE, and Smith ML. Gender differences in cardiovascular regulation during recovery from exercise. *J Appl Physiol* 91: 1902-1907, 2001.
16. Carter R, 3rd, Watenpaugh DE, Wasmund WL, Wasmund SL, and Smith ML. Muscle pump and central command during recovery from exercise in humans. *J Appl Physiol* 87: 1463-1469, 1999.
17. Casa DJ. Exercise in the Heat. I. Fundamentals of Thermal Physiology, Performance Implications, and Dehydration. *J Athl Train* 34: 246-252, 1999.
18. Casa DJ. Exercise in the Heat. II. Critical Concepts in Rehydration, Exertional Heat Illnesses, and Maximizing Athletic Performance. *J Athl Train* 34: 253-262, 1999.

19. Casa DJ, Armstrong LE, Ganio MS, and Yeargin SW. Exertional heat stroke in competitive athletes. *Current sports medicine reports* 4: 309-317, 2005.
20. Casa DJ, McDermott BP, Lee EC, Yeargin SW, Armstrong LE, and Maresh CM. Cold water immersion: the gold standard for exertional heatstroke treatment. *Exercise and sport sciences reviews* 35: 141-149, 2007.
21. Charkoudian N. Skin blood flow in adult human thermoregulation: how it works, when it does not, and why. *Mayo Clinic proceedings* 78: 603-612, 2003.
22. Clapp AJ, Bishop PA, Muir I, and Walker JL. Rapid cooling techniques in joggers experiencing heat strain. *Journal of science and medicine in sport / Sports Medicine Australia* 4: 160-167, 2001.
23. Clements JM, Casa DJ, Knight J, McClung JM, Blake AS, Meenen PM, Gilmer AM, and Caldwell KA. Ice-Water Immersion and Cold-Water Immersion Provide Similar Cooling Rates in Runners With Exercise-Induced Hyperthermia. *J Athl Train* 37: 146-150, 2002.
24. Coats AJ, Conway J, Isea JE, Pannarale G, Sleight P, and Somers VK. Systemic and forearm vascular resistance changes after upright bicycle exercise in man. *The Journal of physiology* 413: 289-298, 1989.
25. Cooper KE. Some historical perspectives on thermoregulation. *J Appl Physiol* 92: 1717-1724, 2002.
26. Costrini A. Emergency treatment of exertional heatstroke and comparison of whole body cooling techniques. *Medicine and science in sports and exercise* 22: 15-18, 1990.
27. Davies CT. Thermoregulation during exercise in relation to sex and age. *European journal of applied physiology and occupational physiology* 42: 71-79, 1979.
28. Ducharme MB, and Tikuisis P. In vivo thermal conductivity of the human forearm tissues. *J Appl Physiol* 70: 2682-2690, 1991.
29. Eichner ER. Treatment of suspected heat illness. *International journal of sports medicine* 19 Suppl 2: S150-153, 1998.
30. Epstein Y, Moran DS, Shapiro Y, Sohar E, and Shemer J. Exertional heat stroke: a case series. *Medicine and science in sports and exercise* 31: 224-228, 1999.
31. Fortney SM, and Vroman NB. Exercise, performance and temperature control: temperature regulation during exercise and implications for sports performance and training. *Sports medicine (Auckland, NZ* 2: 8-20, 1985.
32. Gaffin SL, Gardner JW, and Flinn SD. Cooling methods for heatstroke victims. *Annals of internal medicine* 132: 678, 2000.
33. Gagnon D, Jay O, Reardon FD, Journeay WS, and Kenny GP. Hyperthermia modifies the nonthermal contribution to postexercise heat loss response. *Med Sci Sports Exerc* 40: 513-522, 2008.
34. Gleeson M. Temperature regulation during exercise. *International journal of sports medicine* 19 Suppl 2: S96-99, 1998.
35. Hadad E, Rav-Acha M, Heled Y, Epstein Y, and Moran DS. Heat stroke : a review of cooling methods. *Sports medicine (Auckland, NZ* 34: 501-511, 2004.
36. Hales JR, Khogali M, Fawcett AA, and Mustafa MK. Circulatory changes associated with heat stroke: observations in an experimental animal model. *Clinical and experimental pharmacology & physiology* 14: 761-777, 1987.
37. Halliwill JR. Mechanisms and clinical implications of post-exercise hypotension in humans. *Exercise and sport sciences reviews* 29: 65-70, 2001.

38. Halliwill JR. Mechanisms and clinical implications of post-exercise hypotension in humans. *Exercise and Sport Sciences Reviews* 29: 65-70, 2001.
39. Hammel HT. Neurones and temperature regulation. AMRL-TR-65-232. *Amrl-Tr* 1-32, 1965.
40. Hammel HT. Regulation of internal body temperature. *Annual review of physiology* 30: 641-710, 1968.
41. Hammel HT, Jackson DC, Stolwijk JA, Hardy JD, and Stromme SB. Temperature Regulation by Hypothalamic Proportional Control with an Adjustable Set Point. *J Appl Physiol* 18: 1146-1154, 1963.
42. Hardy JD. Physiology of temperature regulation. *Physiological reviews* 41: 521-606, 1961.
43. Harker J, and Gibson P. Heat-stroke: a review of rapid cooling techniques. *Intensive Crit Care Nurs* 11: 198-202, 1995.
44. Hatfield HS, and Puch LG. Thermal conductivity of human fat and muscle. *Nature* 168: 918-919, 1951.
45. Havenith G, and van Middendorp H. The relative influence of physical fitness, acclimatization state, anthropometric measures and gender on individual reactions to heat stress. *European journal of applied physiology and occupational physiology* 61: 419-427, 1990.
46. Hellon RF. Thermal stimulation of hypothalamic neurones in unanaesthetized rabbits. *The Journal of physiology* 193: 381-395, 1967.
47. Hill L. Arterial pressure in man while sleeping, resting, working and bathing. *J Physiol* 22: XXVI-XXIV, 1898.
48. Houdas Y, and Guieu JD. [The thermoregulatory system in man: a controlled system or a servo system?]. *Archives des sciences physiologiques* 27: 311-338, 1973.
49. Howe AS, and Boden BP. Heat-related illness in athletes. *The American journal of sports medicine* 35: 1384-1395, 2007.
50. Jackson DN, and Kenny GP. Upright LBPP application attenuates elevated postexercise resting thresholds for cutaneous vasodilation and sweating. *J Appl Physiol* 95: 121-128, 2003.
51. Jessen C, and Ludwig O. Spinal cord and hypothalamus as core sensors of temperature in the conscious dog. II. Addition of signals. *Pflugers Arch* 324: 205-216, 1971.
52. Journeay WS, Reardon FD, Jean-Gilles S, Martin CR, and Kenny GP. Lower body positive and negative pressure alter thermal and hemodynamic responses after exercise. *Aviation, space, and environmental medicine* 75: 841-849, 2004.
53. Journeay WS, Reardon FD, Martin CR, and Kenny GP. Control of cutaneous vascular conductance and sweating during recovery from dynamic exercise in humans. *J Appl Physiol* 96: 2207-2212, 2004.
54. Journeay WS, Reardon FD, McInnis NH, and Kenny GP. Nonthermoregulatory control of cutaneous vascular conductance and sweating during recovery from dynamic exercise in women. *J Appl Physiol* 99: 1816-1821, 2005.
55. Kaciuba-Uscilko H, and Grucza R. Gender differences in thermoregulation. *Current opinion in clinical nutrition and metabolic care* 4: 533-536, 2001.
56. Keim SM, Guisto JA, and Sullivan JB, Jr. Environmental thermal stress. *Ann Agric Environ Med* 9: 1-15, 2002.

57. Kenney MJ, and Seals DR. Postexercise hypotension. Key features, mechanisms, and clinical significance. *Hypertension* 22: 653-664, 1993.
58. Kenney MJ, and Seals DR. Postexercise hypotension: key features, mechanisms, and clinical significance. *Hypertension* 22: 653-664, 1993.
59. Kenny GP, Jay O. Gender differences in postexercise esophageal and muscle tissue temperature response. *in press* 2006.
60. Kenny GP, and Jay O. Sex differences in postexercise esophageal and muscle tissue temperature response. *Am J Physiol Regul Integr Comp Physiol* 292: R1632-1640, 2007.
61. Kenny GP, Jay O, Zaleski WM, Reardon ML, Sigal RJ, Journeay WS, and Reardon FD. Postexercise hypotension causes a prolonged perturbation in esophageal and active muscle temperature recovery. *American journal of physiology* 291: R580-588, 2006.
62. Kenny GP, Murrin JE, Journeay WS, and Reardon FD. Differences in the postexercise threshold for cutaneous active vasodilation between men and women. *American journal of physiology* 290: R172-179, 2006.
63. Kenny GP, and Niedre PC. The effect of exercise intensity on the post-exercise esophageal temperature response. *European journal of applied physiology* 86: 342-346, 2002.
64. Kenny GP, Periard J, Journeay WS, Sigal RJ, and Reardon FD. Cutaneous active vasodilation in humans during passive heating postexercise. *J Appl Physiol* 95: 1025-1031, 2003.
65. Kenny GP, Reardon FD, Ducharme MB, Reardon ML, and Zaleski W. Tissue temperature transients in resting contra-lateral leg muscle tissue during isolated knee extension. *Can J Appl Physiol* 27: 535-550, 2002.
66. Kenny GP, Reardon FD, Giesbrecht GG, Jette M, and Thoden JS. The effect of ambient temperature and exercise intensity on post-exercise thermal homeostasis. *European journal of applied physiology and occupational physiology* 76: 109-115, 1997.
67. Kenny GP, Reardon FD, Zaleski W, Reardon ML, Haman F, and Ducharme MB. Muscle temperature transients before, during, and after exercise measured using an intramuscular multisensor probe. *J Appl Physiol* 94: 2350-2357, 2003.
68. Khogali M. *The Makkah body cooling unit. In: Heatstroke and temperature regulation.* Sydney, Australia: Academic Press, 1983.
69. Kilgour RD, Gariepy P, and Rehel R. Cardiovascular responses during recovery from exercise and thermal stress. *Aviat Space Environ Med* 64: 224-229, 1993.
70. Knochel JP. Environmental heat illness. An eclectic review. *Archives of internal medicine* 133: 841-864, 1974.
71. Kollias J, Barlett L, Bergsteinova V, Skinner J, Buskirk ER, and Nicholas W. Metabolic and thermal responses of women during cooling in water. *J Appl Physiol* 36: 577-580, 1974.
72. Lee-Chiong TL, Jr., and Stitt JT. Heatstroke and other heat-related illnesses. The maladies of summer. *Postgraduate medicine* 98: 26-28, 31-23, 36, 1995.
73. Lemire B, Gagnon D, Jay O, Dorman L, Reardon FD, Ducharme MB, and Kenny GP. Influence of adiposity on cooling efficiency in hyperthermic individuals. *European journal of applied physiology* In press: 2008.
74. Lind AR. A physiological criterion for setting thermal environmental limits for everyday work. *J Appl Physiol* 18: 51-56, 1963.

75. Magazanik A EY, Udassin R,Shapiro Y,Sohar E. Tap water, an efficient method for cooling heatstroke victims - a model in dogs. *Aviat Space Environ Med* 9 864-866, . 1980.
76. McArdle WD, Magel JR, Gergley TJ, Spina RJ, and Toner MM. Thermal adjustment to cold-water exposure in resting men and women. *J Appl Physiol* 56: 1565-1571, 1984.
77. McArdle WD, Magel JR, Spina RJ, Gergley TJ, and Toner MM. Thermal adjustment to cold-water exposure in exercising men and women. *J Appl Physiol* 56: 1572-1577, 1984.
78. McArdle WD, Toner MM, Magel JR, Spina RJ, and Pandolf KB. Thermal responses of men and women during cold-water immersion: influence of exercise intensity. *European journal of applied physiology and occupational physiology* 65: 265-270, 1992.
79. McDonald A, Goode RC, Livingstone SD, and Duffin J. Body cooling in human males by cold-water immersion after vigorous exercise. *Undersea biomedical research* 11: 81-90, 1984.
80. Mekjavic IB, and Eiken O. Contribution of thermal and nonthermal factors to the regulation of body temperature in humans. *J Appl Physiol* 100: 2065-2072, 2006.
81. Mekjavic IB, Sundberg CJ, and Linnarsson D. Core temperature "null zone". *J Appl Physiol* 71: 1289-1295, 1991.
82. Moran DS, Heled Y, Shani Y, and Epstein Y. Hypothermia and local cold injuries in combat and non-combat situations--the Israeli experience. *Aviation, space, and environmental medicine* 74: 281-284, 2003.
83. Murphy RJ. Heat illness in the athlete. *The American journal of sports medicine* 12: 258-261, 1984.
84. Nadel ER, Wenger CB, Roberts MF, Stolwijk JA, and Cafarelli E. Physiological defenses against hyperthermia of exercise. *Annals of the New York Academy of Sciences* 301: 98-109, 1977.
85. Neilsen M. Die Regulation der Kopertemperatur bei Muskelerbeit. *Skandinavian Archives of Physiology* 193-230, 1938.
86. Nielsen B, and Kaciuba-Uscilko H. *Temperature regulation in exercise*. Singapore: World Scientific, 1998, p. 127-143.
87. NIOSH. Occupational exposure to hot environments Wahington DC, USA: National Institute for Occupational Safety and Health, 1986.
88. Nunneley SA. Physiological responses of women to thermal stress: a review. *Medicine and science in sports* 10: 250-255, 1978.
89. Nybo L. Hyperthermia and fatigue. *J Appl Physiol* 104: 871-878, 2008.
90. Parsons KC. *Human Thermal Environments*. London: Taylor & Francis, 2003.
91. Petrofsky JS, and Lind AR. Insulative power of body fat on deep muscle temperatures and isometric endurance. *J Appl Physiol* 39: 639-642, 1975.
92. Proulx CE. Une évaluation de diverse température d'eau pour le refroidissement corporelle lors d'une hyperthermie induite par l'exercice. In: *Science de la Santé*. Ottawa: Univesité d'Ottawa, 2002, p. 178.
93. Proulx CI, Ducharme MB, and Kenny GP. Effect of water temperature on cooling efficiency during hyperthermia in humans. *J Appl Physiol* 94: 1317-1323, 2003.
94. Proulx CI, Ducharme MB, and Kenny GP. Safe cooling limits from exercise-induced hyperthermia. *European journal of applied physiology* 96: 434-445, 2006.

95. Pugh LG, Corbett JL, and Johnson RH. Rectal temperatures, weight losses, and sweat rates in marathon running. *J Appl Physiol* 23: 347-352, 1967.
96. Richards D, Richards R, Schofield PJ, Ross V, and Sutton JR. Management of heat exhaustion in Sydney's the Sun City-to-Surf run runners. *Med J Aust* 2: 457-461, 1979.
97. Ross WD WR. *Human proportionality and sexual dimorphism.* New York: Praeger; : Hall RL, ed. , 1982.
98. Rowell LB. Human cardiovascular adjustments to exercise and thermal stress. *Physiological reviews* 54: 75-159, 1974.
99. Saltin B, and Hermansen L. Esophageal, rectal, and muscle temperature during exercise. *J Appl Physiol* 21: 1757-1762, 1966.
100. Scott CG, Ducharme MB, Haman F, and Kenny GP. Warming by immersion or exercise affects initial cooling rate during subsequent cold water immersion. *Aviation, space, and environmental medicine* 75: 956-963, 2004.
101. Shapiro Y, and Seidman DS. Field and clinical observations of exertional heat stroke patients. *Medicine and science in sports and exercise* 22: 6-14, 1990.
102. Smith JE. Cooling methods used in the treatment of exertional heat illness. *British journal of sports medicine* 39: 503-507; discussion 507, 2005.
103. Stephenson LA, and Kolka MA. Menstrual cycle phase and time of day alter reference signal controlling arm blood flow and sweating. *The American journal of physiology* 249: R186-191, 1985.
104. Stephenson LA, and Kolka MA. Thermoregulation in women. *Exercise and sport sciences reviews* 21: 231-262, 1993.
105. Stocks JM, Taylor NA, Tipton MJ, and Greenleaf JE. Human physiological responses to cold exposure. *Aviation, space, and environmental medicine* 75: 444-457, 2004.
106. Tikuisis P, Jacobs I, Moroz D, Vallerand AL, and Martineau L. Comparison of thermoregulatory responses between men and women immersed in cold water. *J Appl Physiol* 89: 1403-1411, 2000.
107. Tipton M, Eglin C, Gennser M, and Golden F. Immersion deaths and deterioration in swimming performance in cold water. *Lancet* 354: 626-629, 1999.
108. Toner MM, Sawka MN, Foley ME, and Pandolf KB. Effects of body mass and morphology on thermal responses in water. *J Appl Physiol* 60: 521-525, 1986.
109. Veicsteinas A, Ferretti G, and Rennie DW. Superficial shell insulation in resting and exercising men in cold water. *J Appl Physiol* 52: 1557-1564, 1982.
110. Vepraskas C. Beat the heat: managing heat and hydration in marching band. *J Sch Nurs* 18: 237-243, 2002.
111. Wade CE, Dacanay S, and Smith RM. Regional heat loss in resting man during immersion in 25.2 degrees C water. *Aviation, space, and environmental medicine* 50: 590-593, 1979.
112. Webb P. The physiology of heat regulation. *Am J Physiol* 268: R838-850, 1995.
113. Weiner JS, and Khogali M. A physiological body-cooling unit for treatment of heat stroke. *Lancet* 1: 507-509, 1980.
114. Wyndham CH, Morrison JF, and Williams CG. Heat reactions of male and female Caucasians. *J Appl Physiol* 20: 357-364, 1965.

115. Yaqub B, and Al Deeb S. Heat strokes: aetiopathogenesis, neurological characteristics, treatment and outcome. *Journal of the neurological sciences* 156: 144-151, 1998.