Body heat storage, sweating and skin blood flow responses following cold and warm water ingestion during exercise

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THESIS

Submitted to the Faculty of Graduate and Postdoctoral Studies in partial fulfillment of the requirements for the degree of Masters of Science in Human Kinetics

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ACKNOWLEDGEMENTS

I wish to thank the following people who have contributed to the conception and completion of this thesis, and provided support throughout my years at the University of Ottawa.

Foremost, I would like to thank my supervisor, Dr. Ollie Jay, whose wisdom and demeanor has given me the opportunity to pursue an uninhibited academic career, while providing countless hours of unprecedented support, teaching and insight.

Dr. Glen Kenny and Dr. François Haman – I am indebted to the valuable technical and theoretical guidance and would like to thank you for the generous teaching and extensive support while serving as active committee members.

Colleagues of the Thermal Ergonomics Laboratory – Matt Cramer, Yannick Molgat, Nathan Morris, Tomasz Deren and lastly Nicole Lesperance, who contributed with many long hours of data collection, thanks for your continuous support and making my graduate work an enjoyable experience.

Participants – Without the participants, who were probed and prodded, this project would have never been. Thank you.

Finally I would like to thank my family and friends – Mom, Dad, Andrew, Megan and Allie – for their continuous generosity and support.

ABSTRACT

Ingestion of cold (<10°C) compared to warm (>37°C) fluid has been suggested to attenuate heat storage levels during exercise. However, modulations in sweat output may yield differences in evaporative heat loss that are greater than differences in heat transfer with the ingested fluid. The purpose of the thesis was to evaluate thermoregulatory control and human heat balance, and compare thermometrically derived values of heat storage with those derived from partitional calorimetry following water ingestion of varying temperature during exercise. We found that water ingestion of 50°C compared to 1.5°C decreases heat storage in thermoneutral environments, and further exacerbates the error of thermometric heat storage estimations. Differences in heat storage were attributed exclusively to disproportionate reductions in whole-body and local sweat output and thus evaporative heat loss potential. Ingested fluid temperature only minimally altered skin blood flow and did not influence dry heat exchange with the ambient environment.

TABLE OF CONTENTS

ACKNOWLEDGEMENTS 1

ABSTRACT 2

PART ONE: EMPIRICAL AND THEORETICAL CONSIDERATIONS 4

CHAPTER 1 5

INTRODUCTION

1.0 INTRODUCTION 5
1.1 OBJECTIVES 11
1.2 HYPOTHESES 11
1.1 DELIMITATIONS AND LIMITATIONS 12

CHAPTER 2 13

REVIEW OF LITERATURE

2.0 BIOPHYSICS OF HUMAN HEAT EXCHANGE 13
    2.1 Conceptual heat balance 13
    2.2 Metabolic heat production 13
    2.3 Sensible heat exchange with the environment 14
    2.4 Evaporative heat loss at the skin 18
    2.5 Respiratory heat loss 19
    2.6 Heat exchange with ingested fluid 19
2.1.0 THERMORECEPTION 20
    2.1.1 Control theories 20
    2.1.2 Thermosensors 23
2.1.0 THERMOREGULATORY RESPONSES 26
    2.1.1 Sweating 26
    2.1.2 Cutaneous vasodilation 29
2.2.0 FACTORS INFLUENCING HEAT LOSS DURING EXERCISE 31
    2.2.1 Hydration 31
    2.2.2 Sweating efficiency 32

PART TWO: METHODS AND RESULTS OF THE THESIS 35

4.0 ARTICLE 1
Body heat storage during physical activity is greater with cold versus warm water ingestion under conditions that permit full sweat evaporation
(Submission to Acta Physiologica) 36
4.1 ARTICLE 2
Sudomotor and vasomotor activity following serial cooling and heat of thermosensors in the esophagus and stomach (Extended abstract submission to the CSEP annual conference) 73

PART THREE: DISCUSSION AND CONCLUSION OF THE THESIS 79

5.0 DISCUSSION 80
5.1 CONCLUSION AND FUTURE RESEARCH CONSIDERATIONS 82

PART FOUR: REFERENCES 84

APPENDIX 92

PART ONE:

EMPIRICAL AND THEORETICAL CONSIDERATIONS

CHAPTER 1
INTRODUCTION

1.0 Introduction

The ability of humans to cope with continually changing thermal environments are such that deep (core) body temperature can usually be maintained within a few tenths of a degree Celsius. However, during physical activity, 80 to 98% of the metabolic energy generated from within the body is released in the form of heat. In doing so, strenuous physical activity carries an inherent risk of substantially elevating core temperature and potentiating the risk of heat related illnesses (Nadel 1977). Physical activity performed in hot and humid environmental conditions, exacerbates this risk. A rise in core temperature of only 1 to 2°C from resting (of ~37°C) evokes the risk for heat exhaustion, characterized by painful muscle contractions, dizziness, malaise, fatigue, nausea, vomiting or headaches (ICON 2004). Further rises in core temperature thereafter elicit a risk of heat stroke, where upon distinct central nervous-system functioning is altered, resulting in confusion, ataxia, irritability, coma or even death (ICON 2004; Howe and Boden 2007).

Life threatening levels of hyperthermia are a particular concern for many physically demanding occupational settings, military operations, sporting events, and for at risk individuals – e.g. the elderly (Bouchama and Knochel 2002; Kenny et al. 2009). Notably, the United States Bureau of Labor Statistics reported from 2003 to 2005 that an average of 31 workers per year die from exposure to “environmental heat” (BLS 2003-2005), and although not reported, the number of workers who are hospitalized from heat related illnesses are presumably far greater (Nunneley and Reardon 2002). In the military, from 1971 to 2000, 3971 US Army personal were hospitalized for heat illness (Amoroso et al. 1999), and in the sporting environment, hyperthermia is the third leading cause of death in athletes behind cardiac disorders and head and neck trauma (Howe and Boden 2007). Moreover, in the general population, the health effects of "heat waves" carry a heavy burden. For example, prolonged bouts of high temperatures coupled with high humidity in Western Europe during the summer of 2003 accounted for ~30,000 deaths (Kosatsky 2005). Marked hyperthermia (i.e. core temperatures higher than 39°C) is therefore a concern for all populations.

To help mitigate the occurrence of exertional heat related illnesses and deaths, current American College of Sports Medicine (ACSM) guidelines recommend that replacement of body fluids through beverage ingestion be equal to a level that limits body mass reductions by no more than 2% from baseline (Sawka et al. 2007). As sweat rate, skin blood flow, and cardiovascular strain are collectively dependent upon proper hydration status, the considerations for such guidelines are central for health safety (Sawka et al. 2001). In conjunction with recommendations stipulated by the ACSM, recent literature (Wimer et al. 1997; Lee and Shirreffs 2007; Lee et al. 2008a; Lee et al. 2008b; Siegel et al. 2010; Stanley et al. 2010; Burdon et al. 2010a; Burdon et al. 2010b) recommends the ingestion of a cold (i.e. ≤10°C) fluid in order to help attenuate core temperature elevations during exercise. Conceptually, cold drink ingestion will act as a *heat sink* within the stomach and absorb residual heat from the body.

The practice of cooling the body through cold fluid ingestion stems from work by Pinson and Adolph in 1942 (Pinson and Adolph 1942) who found that, when compared to the application of ice packs, cold water showers or ice water irrigation of the rectum, the best method for creating a *"distributed heat deficit"* in the body was to ingest ice water (Pinson and Adolph 1942). This method has recently led to the use of cold fluid (or even ice-slurry) ingestion to pre-cool before exercise in the heat (Ross et al. 2011), and, as aforementioned an effective means to help mitigate the rise in core temperature during exercise (Lee and Shirreffs 2007; Lee et al. 2008a; Siegel et al. 2009; Burdon et al. 2010b). The collective body of recent literature concludes that ingesting cold compared to warm fluid during exercise attenuates levels of heat storage (Burdon et al. 2010a); however, an uncertainty in this notion is presented due to two limiting problems.

Firstly, an inherent problem may reside within the estimations of heat storage using the conventional proportional weightings of skin and rectal temperature. Indeed, separate body compartments during exercise (e.g. muscle compared to esophageal) can differ in temperature by up to almost 3.0°C (Kenny et al. 2003); therefore, a single internal temperature measurement site is not representative of mean core body temperature. In turn, the conventional two compartmental estimation of heat storage during exercise is often erroneous (Jay et al. 2007). Furthermore, local cooling of tissue surrounding the rectum following cold beverage ingestion may exacerbate errors in estimating heat storage using thermometry. Specifically, the relatively low blood flow in the stomach and visceral tissue during exercise (in comparison to other parts of the body, e.g. working muscles) (Flamm et al. 1990), would confine most of the heat transfer between the fluid and body in the stomach. It therefore stands to reason that a proportion of the heat transfer will eventually bypass the insulating properties of the tissue surrounding the rectum and subsequently elicit a residual reduction in rectal temperature. Given the above rationale, exacerbations in the inaccuracy of using rectal temperature to estimate whole-body heat storage (Jay et al. 2007) will become increasingly relevant during cold beverage ingestion. A calorimetric analysis of heat storage is therefore merited to provide a better estimation of whole-body heat storage following beverage ingestion of varying temperatures.

Secondly, a closer analysis of the existing literature reveals that cold (i.e. below 10°C) compared to warm (i.e. above 38°C) beverage ingestion is also followed by significant reductions in whole body sweat loss (Wimer et al. 1997; Lee et al. 2008a). It follows that, if sweating efficiency is maintained (i.e. the majority of sweat secreted is evaporated); cold drink ingestion would reduce the potential for evaporative heat loss. If the reduction in evaporative heat loss is greater than the heat transfer with the ingested fluid, warm vs. cold beverages may in fact lead to a reduced net heat storage. This discrepancy is retrospectively observed in data by Lee et al. (2008a), who reported a 260 ml reduction in whole body sweat loss during 90-min of fixed intensity cycling (in a 25.3°C and 60% RH environment) while ingesting four aliquots of 400 mL fluid at 10°C compared to 50°C. Assuming 100% evaporation, the 260 mL difference in sweat output equates to an evaporative heat loss difference of 631 kJ greater with 50°C compared to 10°C fluid ingestion. In turn, the difference in heat transfer to / from the ingested fluid only equates to 268 kJ more with 10°C fluid compared to 50°C. Therefore, this analysis shows that 10°C compared to of 50°C fluid ingestion elicited an end-exercise heat storage that was in fact ~363 kJ greater. Wimer et al. (1997) and Lee et al. (2007; 2008a) also report significantly lower mean skin temperatures following cold compared to warm beverages. It therefore appears that differences in heat loss with beverage temperature may stem beyond disproportionate modulations in sweat output. Specifically, reductions in skin temperature following cold beverages reduces the rate of dry heat exchange via conduction, convection and radiation in a given environment (Parsons 2003).

An evident critique of the above rationale may lie within the assumption that 100% of secreted sweat is evaporated from the skin, thereby contributing entirely to whole-body heat loss. Sweating efficiency (η_{sw}), defined as the percentage of sweat produced that evaporates from the skin, is maintained above 100% with combinations of climate and exercise that elicit a skin wettedness (*w*) (Gagge 1937) of 74% or below (Candas et al. 1979; Candas et al. 1979a). An exponential decline in η_{sw} occurs when *w* exceeds 74% (Candas et al. 1979; Candas et al. 1979a). Given that *w* is by definition the ratio of evaporated sweat and maximal evaporation possible in a given environment (Gagge 1937), the greater sweat output observed by Lee et al. (2008a) following 50°C compared to 10°C beverages may be associated with differences in η_{sw}. It is thus possible (albeit unlikely given the temperate environmental conditions and moderate exercise intensity) that the reduction in sweat output with cold beverage ingestion is indebted to large differences in η_{sw} (Lee et al. 2008a) and congruent to the evaporation required to achieve heat balance. Indeed, when conditions are such that sweating efficiency is not maintained (i.e. during intense exercise in a hot and humid environment), changes in sweat output are not paralleled by proportional changes in evaporative heat loss. Therefore cold vs. warm beverage ingestion during intense exercise in hot and humid conditions (e.g. (Lee et al. 2008b; Burdon et al. 2010b; Ross et al. 2011)) may very well reduce heat storage. However, given the current sweating data, it appears that this claim is highly dependent upon the environmental conditions, and thus η_{sw}. Therefore, in order to ascertain differences in net heat loss following beverage ingestion at varying temperatures, in addition to estimating heat storage through calorimetry rather than thermometry, future studies must be performed where dripped sweat is measured, or in conditions that maintain η_{sw} at ~100%.

The potential misconceptions associated with whole-body heat storage following beverage ingestion of varying temperature, likely stems from the lack of heat balance data. However, it is also apparent that there is a lack of literature on the physiological heat loss responses (i.e. skin blood flow and sweating activity) following beverage ingestion of varying temperatures. During exercise, heat activated thermoreceptor activity provide the primary dose-dependent stimulus for sudomotor and vasomotor activity (Romanovsky 2007). Conventionally, whole-body temperature reception is conceptualized as a proportional *shell* and *core* model, wherein the influence of changes in deep core temperature upon heat loss responses are far greater than changes in shell temperature (Shibasaki et al. 2006). However, the above representation of thermal status and subsequent indication for heat loss responses must certainly change with local cooling, which holds true following beverage ingestion of varying temperature. Indeed, *deep peripheral thermosensors* that (among other areas) reside in the esophagus and abdominal tissue (Rawson and Quick 1972; Riedel 1976; Gupta et al. 1979) have been shown to elicit strong activation following cold or hot stimuli. It follows that the mechanistic explanation for the potential disproportionate modulations in sweating following beverage ingestion of varying temperature may owe exclusively to thermoreceptor activity in the abdomen and esophagus. If so, a transient and distributed measurement of sweating following beverage ingestion of varying temperatures may shed light upon this mechanism. Furthermore, a concomitant analysis of skin blood flow would demonstrate separate roles that thermosensors in the esophagus and abdomen may have on vasomotor, in comparison to sudomotor activity following beverage ingestion.

1.1 Objectives

1) To assess changes in human heat balance components and subsequent heat storage following water ingestion of 1.5°C, 10°C, 37°C and 50°C (a range of temperatures frequently employed in previous research, e.g. Lee et al. 2008) during exercise in a temperate environment;

2) To compare the heat storage values derived by partitional calorimetry with those derived by conventional thermometric (i.e. using proportional weightings of core and skin temperature) estimations, and;

3) To concomitantly characterize the localized and transient responses of skin blood flow and sweating.

1.2 Hypotheses

1) Water ingestion of 50°C and 37°C compared to 10°C and 1.5°C will elicit lower levels of heat storage by virtue of disproportionate modulations in evaporative cooling potential compared to the heat transfer between the body and ingested water.

2) Water ingesting of 1.5°C and 10°C will exacerbate the error of thermometric heat storage estimations due to local reductions of tissue temperature surrounding the rectum.

3) Water ingestion of 1.5°C, 10°C and 50°C will elicit acute and transient temperature-dependent changes in sweating and skin blood circulatory activity, evidencing influential activity from thermosensors in the esophagus and abdomen.

1.3 Delimitations and Limitations

The data can only directly apply to healthy males aged 18 to 39 yrs. To avoid known thermoregulatory complications associated with menstrual cycle (Charkoudian and Joyner 2004), females will be excluded from the study. Measurement errors may lie within differences of estimating whole-body heat storage using partitional compared to direct calorimetry. However, accuracy in whole-body sweat loss measurements, and subsequently evaporative heat loss, will be maximized by correcting for mass lost through respiration (water and metabolic mass losses) and saliva, and accuracy in our sensible heat loss estimations will be assured by measuring skin and air temperature within ±0.1°C while assuring that ambient air temperature, humidity and air velocity are within ±0.5°C, ±5% and ±0.1 $m \cdot s^{-1}$ respectively for each condition within participant trials. The validity of the data will therefore be primarily dependent upon the accuracy of calculations, rather than the measurements *per se*.

CHAPTER 2
REVIEW OF LITERATURE

2.0 Biophysics of Human Heat Exchange with the Environment

2.1 Conceptual Heat Balance

The biophysics of human heat exchange is classically described through the conceptual human heat balance equation (Gagge 1941; Parsons 2003) as follows:

$$(M - W) - (\pm K \pm C \pm R) + (C_{res} + E_{res}) + E_{sk} = \pm S$$

In its simplest form, human heat balance can be divided into two components, 1) heat production and 2) heat loss. The sum of the two results in heat storage (S) (which can either be negative or positive). Calorimetrically, the estimations of heat storage are accomplished through the calculations of M, W, K, C, R, C_{res}, E_{res}, and E_{sk}, detailed below;

2.2 Metabolic heat production (M-W): is the result of metabolic energy expenditure (M), minus the mechanical work performed (W). Humans generate energy from ingested food (comprising of carbohydrates, fats, protein and in some cases alcohol) and inspired oxygen (Guyton 1969). Energy is thus generated by *burning* proteins, alcohol, carbohydrates or fats in oxygen. The latter two, carbohydrates and fats, when combined with oxygen, are (in absolute terms) the primary fuel for metabolic energy production. Since the amount of oxygen required to metabolize fats and carbohydrates is constant (Weir 1949; Brooks et al. 2005), measuring the rate of inspired and expired oxygen and carbon dioxide can be used to quantify metabolic energy expenditure (indirect calorimetry), as follows (Nishi 1981):

$$M = VO_2 \frac{\left(\frac{RER-0.7}{0.3}e_c\right)+\left(\frac{1-RER}{0.3}e_f\right)}{60\times BSA}\times 1000 \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots \mathrm{W{\cdot}m^2} \ldots\ldots\ldots (1)$$

Where; VO_2 is the amount of consumed oxygen in L/min, e_c is the caloric equivalent per liter of oxygen for the oxidation of carbohydrates (21.13 kJ). e_f is the caloric equivalent per liter of oxygen for the oxidation of fat (19.62 kJ). *RER* is the ratio of expired CO_2 over consumed O_2 and *BSA* is the body surface area of the individual in m^2, calculated using the equations from Dubois and Dubois (1916).

While heat generated within the body is primarily dependent upon total metabolic energy expenditure, it is also largely dependent upon the ability to utilize metabolic energy towards external work (*W*), i.e. force • distance. Therefore, at a given VO_2, internal heat production is dictated upon the efficiency of a task - e.g. novice swimmers have a mechanical efficiency of 2 to 4 % (Di Prampero et al. 1974) whereas the average cyclist has a mechanical efficiency of 15 to 20% (Moseley et al. 2004).

2.3 Sensible heat exchange with the environment (K, C, R): comprises the combination of conduction (K), convection (*C*), and radiation (R). The components of the sensible heat exchange with the environment follow the second law of thermodynamics – i.e. heat energy will spontaneously flow from higher to lower temperature regions in an attempt for equilibrium (Adkins 1968). Conduction (*K*) refers to heat transfer by direct contact between the body and a air or liquid, C refers to the flow of heat between the body and a medium by movement (e.g. air movement) and R refers to the flow of electromagnetic waves (where the largest radiant source is indisputably emitted from the sun but can arise from local sources such as room heaters). Collectively, the body can either lose or gain heat through the sensible heat exchange avenues.

The estimations of conductive heat transfer (K) depends upon; a) the cross sectional area between the two mediums in contact, b) the temperature difference between the two mediums and lastly, c) the *thermal conductivity*, of the medium; also known as the *conductivity constant* or *conduction coefficient* (*k)* measured in $W \bullet m^{-1} \bullet {}^{\circ}K^{-1}$. When these variables are known, conduction (K, or Q in the below equation) can be calculated using equation 2 (Parsons 2003)

$$Q = -\,kA\,\frac{T_2 - T_1}{d} \;\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots W \bullet m^{-2} \ldots\ldots\ldots (2)$$

Where: Q equals the rate of heat transfer by conduction (K as depicted in the conceptual heat balance equation), k equals the thermal conductivity of the medium (in $W \bullet m^{-}1 \bullet K^{-1}$); A equals the cross-sectional area normal to conductive direction (in m^2); $T_2 - T_1$ equals the temperature difference across the medium (in degrees Kelvin) and d equals the distance between the points at temperatures T_2 and T_1 (in m). In many circumstances conduction can be considered negligible as it requires direct contact with a solid surface, which, often holds little potential for heat transfer (Parsons 2003) – e.g. shoe soles have insulating properties that minimize conductive heat transfer with the ground while standing.

The estimations of convective heat transfer (*C*) depends upon; a) the velocity and temperature of the two surfaces in contact (e.g. air particles and skin), b) the thermal resistance of any clothing worn and lastly, c) the effective surface area in contact with the outside medium (which is primarily dependent upon posture and clothing). The rate of convective heat exchange at the skin from the surrounding air can be calculated using equation 3 (Parsons 2003).

$$C = f_{cl} h_c (t_{cl} - t_a) \text{ ……………………………………………W•m}^{-2}\text{……….(3)}$$

Where; f_{cl} is the clothing area coefficient factor; h_c is the convective heat transfer coefficient (in $\mathrm{Wm^{-2}K^{-1}}$ - dependent upon a person's physical position in space and the resultant relative velocity of the surrounding air); t_{cl} is the mean temperature of the clothed body (°C); and t_a is the surrounding air temperature (°C).

Radiation (*R*) can be estimated by replacing h_c in equation 3 by h_r, meaning the radiative heat transfer coefficient in $\mathrm{W{\bullet}m^{-2}{\bullet}K^{-1}}$, calculated using equation 4 (Parsons 2003).

$$h_r = 4\varepsilon\sigma \frac{A_r}{BSA}\left[273.2 + \frac{(t_{cl} + t_r)}{2}\right]^3 \text{ ………………………………W•m}^{-2}\text{K}^{-1}\text{……….(4)}$$

Where; ε is the area weighted emissivity of the clothing body surface; σ is the Stefan-Boltzmann constant, $5.67 \times 10^{-8}\ \mathrm{Wm^{-2}K^{-4}}$; A_r/BSA is the effective radiative area of body (in $\mathrm{m^2}$ - dependent upon the physical position of the person in space) and t_r is the mean radiant temperature (in °C), which when indoors, is assumed to be equal to t_a. Therefore, when indoors (and in the absence of external radiant warmers) radiation can be calculated using equation 5; (Parsons 2003).

$$R = f_{cl} h_r \left(t_{cl} - t_a \right) \ldots\ldots\ldots\ldots\ldots\ldots \mathrm{W{\bullet}m^{-2}} \ldots\ldots (5)$$

The heat transfer through convection and radiation can also be calculated using a combined heat transfer coefficient, or operative temperature (t_o). This calculation is used when clothing insulation cannot be considered negligible. The operative temperature can be calculated using equation 6 (Parsons 2003).

$$t_o = \frac{(h_r t_r + h_c t_a)}{(h_r + h_c)} \ldots\ldots\ldots\ldots\ldots\ldots {}^{\circ}\mathrm{C} \ldots\ldots (6)$$

The combined heat exchange of convection and radiation can thus be calculated using equation 7 (Parsons 2003).

$$C + R = \frac{(t_{sk} - t_o)}{\left(R_{cl} + \frac{1}{f_{cl} h} \right)} \ldots\ldots\ldots\ldots\ldots\ldots \mathrm{W{\bullet}m^{-2}} \ldots\ldots (7)$$

Where; R_{cl} equals the thermal resistance of clothing (in $\mathrm{m^{-2}{\bullet}K{\bullet}W^{-1}}$) and T_{sk} equals mean skin temperature.

2.4 Evaporative heat loss at the skin (E_{sk}): refers to the evaporation of sweat or liquid from the skin surface. Each gram of evaporated sweat (at 30°C) from the skin releases 2.426 kJ of heat (Wenger 1972). Therefore, if 100% of secreted sweat is evaporated, evaporative heat loss can be calculated as follows;

$$E_{sk} = \frac{2426\,(WBSL)}{(t)}\,(BSA^{-1}) \ldots\ldots\ldots\ldots \text{ W•m}^{-2} \ldots\ldots (8)$$

Where; *WBSL* is whole body sweat loss; *t* is time (in s) and BSA is body surface area (m^2).

When 100% sweating cannot be assumed, the theoretical levels of sweating efficiency can be estimated by the ratio of the evaporation required to achieve heat balance (E_{req}), to the maximal evaporation possible in a given environment (E_{max}) (refer to section 2.3.2). Values of E_{req} can be calculated by the metabolic heat production, minus the heat lost through respiration and dry avenues at the skin. Values of E_{max} can be calculated by letting *w* equal 1.00 in equation 9 (ASHRAE 1989).

$$E_{max} = \frac{w(P_{sk,s} - P_a)}{\left[R_{e,cl} + \frac{1}{f_{cl}h_e}\right]} \ldots\ldots\ldots\ldots \text{W•m}^{-2} \ldots\ldots (9)$$

Where: *w* equals skin wettedness (from 0.06 to 1.00, where 1.00 equals 100% skin wettedness); $P_{sk,s}$ equals saturated water vapor pressure at the skin (in kPa – calculated using Antoine’s equation (Parsons 2003)); P_a equals water vapor pressure in the ambient air (in kPa); $R_{e,cl}$ equals the evaporative heat transfer resistance of clothing (in m^2 kPa W^{-1}); and h_e equals the evaporative heat transfer coefficient (in Wm^{-2} kPa^{-1}) – equal to $16.5h_c$ for air velocity >0.2 $m•s^{-1}$ and 3.1 for air velocity <0.2 $m•s^{-1}$ (Parsons 2003).

2.5 Respiratory heat loss ($+C_{res}$ $+E_{res}$): occurs through the avenues of convective and evaporation. Convective heat loss (C_{res}) arises when cool inhaled air is heated to body temperature in the lungs and upper respiratory tract, which is subsequently expired into the environment. The evaporative component (E_{res}) arises while inhaled air is warmed and saturated with water, which is liberates into the environment during expiration. Respiratory heat loss thus depends upon the physical properties of the inspired air (i.e. temperature and vapor pressure) and a person's rate of respiration. Heat loss through respiration can be estimated from equation 7 (ASHRAE 1989).

$$C_{res} + E_{res} = [0.0014H(34 - t_a) + 0.0173H(5.87 - P_a)] \ldots\ldots\ldots \text{W•m}^{-2} \ldots\ldots\ldots (10)$$

Where: H is the metabolic heat production in $W•m^{-2}$; P_a is the partial pressure of water vapor of ambient air in kPa and t_a is the ambient air temperature in ºC.

2.6 Heat exchange with ingested fluid (H_{fluid}): is a component that requires consideration following fluid ingestion of a temperature that is different to body temperature. Heat transfer will occur until the surrounding body tissue and fluid temperature reach equilibrium (in correspondence with the second law of thermodynamics). The extent of heat transfer depends upon the temperature difference between body tissue and ingested fluid, the volume of ingested fluid, and the specific heat capacity of fluid. Heat transfer with ingested fluid can be calculated as follows (derived from equation 2);

$$H_{fluid} = (SpHc(Fluid))(T_{es} - T_{fluid}) \ldots\ldots\ldots\ldots \text{J} \ldots\ldots (11)$$

Where; *SpHc* is the specific heat capacity of fluid (e.g. water: 4.184 $\text{J}\bullet\text{g}^{-1}\bullet\text{C}^{-1}$), *fluid* is the amount of ingested fluid (in grams; for water, one gram is equal to 1 mL), T_{es} is esophageal temperature immediately before the fluid ingestion, and T_{fluid} is the temperature of the ingested fluid.

2.1.0 Thermoreception

2.1.1 Control Theories

In order to maintain and regulate a relatively constant body core temperature, humans require constant dynamic regulations of body tissue temperature. In doing so, the body interacts with its thermal environments, with the end goal of yielding a dynamic balance. The resultant product is a core body temperature that, under resting conditions, does not vary far from 37.0°C. At the level of whole-body thermal control, the preoptic/anterior hypothalamus (PO/AH), located immediately above the brain stem and forming the ventral part of the diencephalon, has indisputably been given the role of central integrator (Romanovsky 2007). As such, the PO/AH coordinates inputs from warm and cold temperature sensors (thermoreceptors) deriving from the deep (core) tissue, the skin and the central nervous system (CNS). The exact mechanisms underpinning thermal control however, remain an ongoing field of study.

Classically, the regulation of core body temperature was thought to behave much the same as the conventional home furnace, with the thermostat adjusted to a "*set point"* of ~37.0°C (Hardy 1961; Hammel et al. 1963; Hammel 1968). Central to this theory is the notion of a unified system of three working components; the sensor, the integrator and lastly, the effector. The integration of these components thus act according to a closed-loop feedback system (Hammel 1968), constantly monitoring and adjusting core temperature around it's set point. Applied to the human body, temperature sensitive neurons play the role of *sensors*, sending afferent stimuli to the hypothalamus – *the integrator*, which in turn, elicit thermolytic or thermogenic responses (through manipulations of blood flow, shivering, piloerection or sweating), via the *effectors*. A simplified schematic of the closed-loop feedback system is shown in Figure 1.

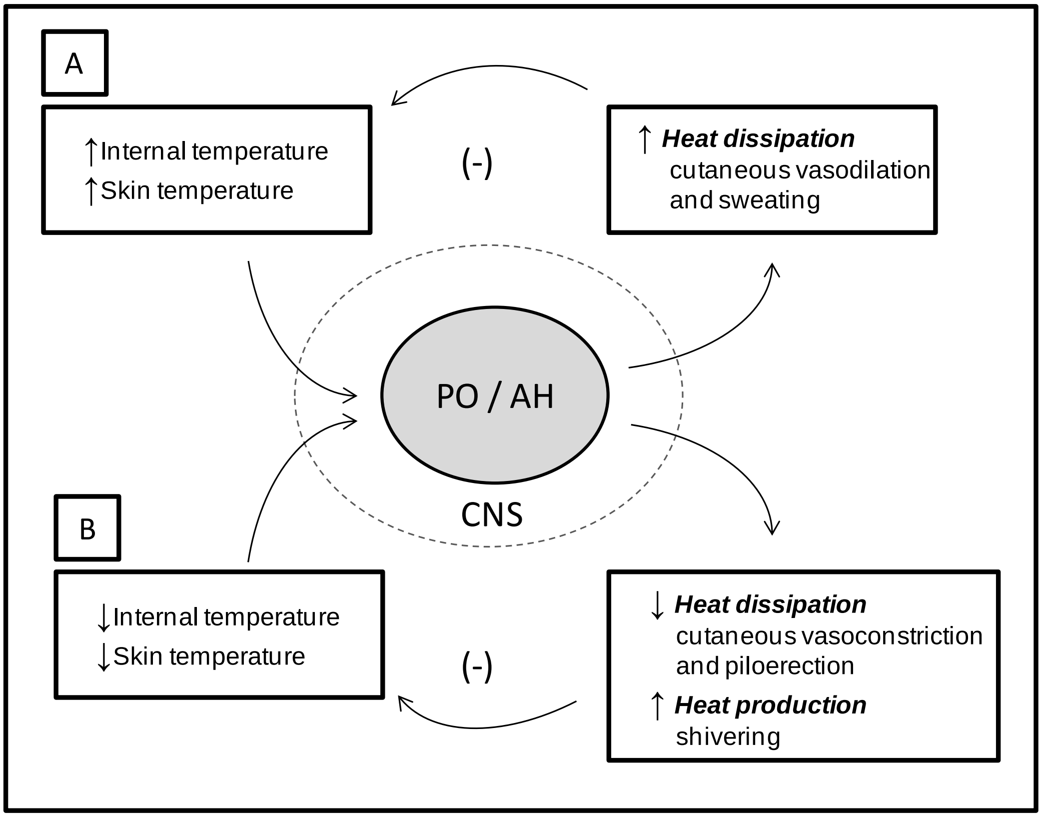

Figure 1. *Human thermoregulatory negative feedback loop. Temperature sensitive neurons activated across the body provide afferent signals to the hypothalamus (PO/AH), in turn generating efferent thermoregulatory responses. A: Elevations in internal and/or skin temperature initiate an increased heat loss through cutaneous vasodilation and sweating. B: Decreases in skin and/or internal temperature initiates heat gain and conservation through shivering and cutaneous vasoconstriction and piloerections respectively (Modified from Charkoudian,2003 (2003))*

Notwithstanding the convenient simplicity, recent literature has provided provisions to the set point theory, while some argue that the term *set point* when referring to human thermoregulation should be abandoned altogether (Werner 2009). Mekjavic et al. (1991) and later revisited by Bligh (2006), suggest that, rather than defending a set point, the regulation of core temperature is determined by a *"null zone"*. In other words, core temperature will vary by ~0.5°C from ~37.0°C before marked thermoregulatory responses are initiated. The implication of the null zone theory states that core temperature is regulated by an interplay of upper (i.e. warming) and lower (i.e. cooling) bound sensors, which each carry separate response coefficients; rather than through the comparison of signals with a constant set point. Further alternative control theories include 1) a dynamic thermosensitive balance of heat flow regulation (Webb 1997), and 2) "*comparator neurons*" instead of sensors (Kobayashi et al. 2006).

The debate regarding thermal control in the body is no doubt ongoing (Werner 2009). However, the most recent and seemingly accepted theory, a product from the dismissal of the set point, is the "*balanced point theory"* (Romanovsky 2007) or equally the "*proportional control theory"* (Werner 2009). Both the balanced point theory and proportional control theory state that temperature regulation consists of a process of independent *thermoeffector* loops, rather than a unified system, without a comparative set point core temperature. Body temperature is thereby controlled by a "multi-sensor, multi-processor, multi-effector proportional feedback control system" (Werner 2009).

2.1.2 Thermosensors

Temperature sensing neurons (thermosensors) are typically categorized into two groups; 1) those that are located centrally (e.g. in the brain and spinal cord – central thermosensors) and 2) those that are located peripherally (e.g. in the skin and mouth – peripheral thermosensors). Thermosensors can further be separated into two more groups; 1) those that respond to warm stimuli and 2) those that respond to cold stimuli (Romanovsky 2007). According to Romanovsky (2007), the characteristics, location and abundance of thermosensors are driven by two facts. First, resting body temperature of

~37.0°C (a product of the resting balanced point of thermosensor feedback loops) is "asymmetrical", that is, only a few degrees away from a critically high body temperature, but much further (i.e. almost 10°C) away from a critically low body temperature. The body's asymmetrical resting body temperature yields a thermosensor makeup that is predominantly warm sensitive. The second fact, is that humans are endothermic, that is, generate heat from inside the body (this becomes particularly relevant during exercise as internally generated heat can increase by more than ten times). Therefore, relative to peripheral thermosensors, the centrally located ones are predominantly warm sensitive.

Central thermosensors, conventionally defined by their existence in the brain and spinal cord, are considered the most influential for driving thermoregulatory responses (more so in the brain than spinal cord (Nakamura 2011)). The driving mechanisms behind the functional contributions of thermosensors in the hypothalamus however, are unclear. It is classically assumed that warm and cold thermosensors in the hypothalamus function reciprocally, whereby the inactivation of warm is paralleled by the inhibition of cold thermosensors, or vice versa (Bligh 2006). It is also thought that cold (i.e. shivering) and warm (i.e. sweating) defense mechanisms require the activation of cold or warm sensitive thermosensors respectively (Bligh 2006). This theory is supported by the fact that the abundance of thermosensors located in the hypothalamus are warm sensitive, in agreement with the asymmetrical nature of resting core body temperature. However, work by Zhang et al. (Zhang et al. 1995) and Chen et al. (Chen et al. 1998) suggest that the role of warm thermosensors are far more influential than cold, irrespective of the relative amount. Their theory is that the inhibition or activation of warm sensitive thermosensors can initiate cold and warm defense mechanisms respectively, independently from activity of cold sensitive thermosensors. That is, the inhibition of warm sensitive thermosensors can initiate shivering responses in the absence of cold sensitive thermosensor activation. In keeping with the predominant role of warm thermosensors in the hypothalamus, many seem to exhibit a continual tonic activation, with a spontaneous membrane depolarization (Zhao and Boulant 2005; Wechselberger et al. 2006). The logical explanation for the tonic activation is likely in response to the body’s basal metabolic heat production, which generates a tonic vasomotor response (section 2.2.2). It is therefore to no surprise that these cells are often referred to as the pacemakers of thermoreception (Romanovsky 2007).

In contrast to thermosensors found in the brain and spinal cord, the majority of those found in the periphery are cold sensitive. Peripheral thermosensors are typically referred to as those that reside in the cutaneous tissue (skin) (Parsons 2003). Cutaneous thermosensors are very active during rapid changes in temperature however reduce their activity following steady state thermal stimuli (Nakamura 2011) (the extent of this is easily recognized by the first minute of jumping into a swimming pool). However, there also exists a group of peripheral thermosensors that, in contrast to cutaneous receptors, respond to deep tissue temperature. These thermosensors are found in the esophagus, stomach, large-abdominal veins and blood vessels (Rawson and Quick 1972; Riedel 1976; Gupta et al. 1979; Romanovsky 2007). The influence that these thermosensors have on thermoregulatory control remain unclear (Nakamura 2011). It may be possible that, like central thermosensors, abdominal thermosensors display an influential contribution to thermal control, and mediate thermo-effector responses more than those located in the cutaneous tissue. This notion is supported by early work by Rawson and Quick (Rawson and Quick 1972), who demonstrated that locally warming and cooling the intra-abdominal tissue in the ewe elicits strong thermo-effector responses (i.e. panting and shivering respectively), without changes in hypothalamic temperature. If thermosensors in the abdomen and esophagus respond similarly to those located in the central nervous system, direct cooling or heating (e.g. through cold or warm fluid ingestion), would elicit a thermo-effector response that is misrepresentative of heat balance requirements. This in turn may explain the potential disproportionate modulations in sweat output following beverage ingestion of varying temperatures (see Chap. 1).

2.2.0 Thermoregulatory Responses

2.2.1 Sweating

From the age of two, the average human body carries 1.6 to 4.0 million sweat glands (Kuno 1956). Of those, the most abundant (located across nearly the entire skin surface) are eccrine sweat glands, and provide the primary means for evaporative heat loss during thermal stress (Kondo et al. 1998). Sweat gland output is mediated through the sympathetic pathway (primarily with cholinergic activation), the control of which however, is dependent both on thermal and non-thermal stimuli. Non-thermal sweating is mediated by central commands, mechanoreceptors, metaboreceptors, baroreceptors, osmoreceptors, chemoreceptors and mental stimuli. Partitioning the distributed and proportional influences of these factors upon sudomotor activity remains an ongoing field of study (Kenny and Journeay 2010).

Notwithstanding the influences of non-thermal mediators, during exercise, gross sudomotor activity (sweating) is primarily dependent upon thermal inputs (Shibasaki and Crandall 2010) and ultimately evaporative needs to achieve heat balance. Our understanding of thermally mediated changes in sweating stems from work by Adolph, in (1923) and Kuno, in (1956), who proposed separate but linking ideas. Adolph suggested that sweat rate was proportional to the outside environmental temperature, thereby asserting that changes in skin temperature provide the main stimulus for sweat output. In contrast, 33 years later, Kuno suggested that sweat rate is far more dependent upon internal temperature, with little dependence on temperature of the periphery. Years later, subsequent articles have sought to assess the interplay of skin temperature and body core temperature on sweat rate (Nadel et al. 1971; Nadel 1979; Shibasaki et al. 2006). The collective product is the now accepted notion that both peripheral and internal tissue temperatures contribute to changes in sweat rate. Figure 2 provides an illustration of the relationship of core and skin temperature on sweat rate.

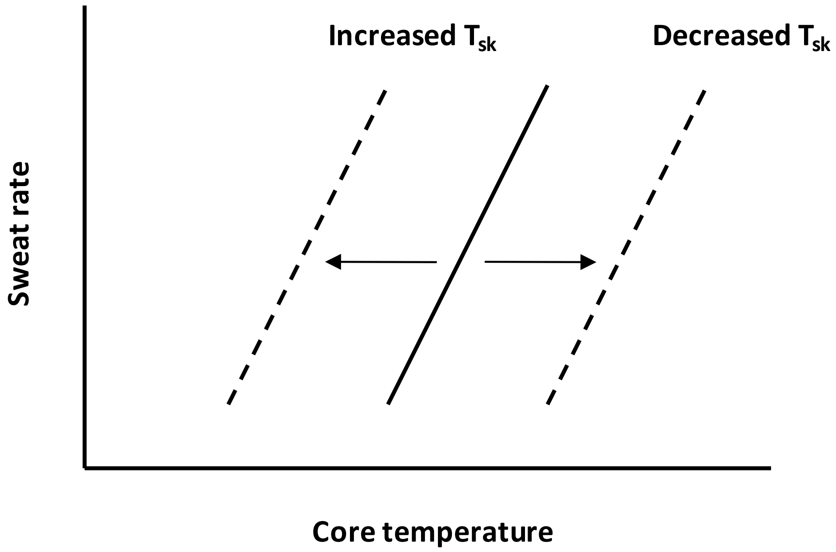

***Figure 2**. The relationship of mean skin temperature (T_{sk}) and core temperature on sweat rate; the onset sweating threshold at a given internal temperature is modified by skin temperature. Following the onset threshold sweat rate increases linearly with internal temperature. (Modified from Shibasaki et al., (2006)).*

Only a few papers have investigated the effects of drink temperature upon sweating activity (Wimer et al. 1997; Lee et al. 2006; Lee and Shirreffs 2007; Lee et al. 2008a) and all report significant reduction in sweat loss with cold compared to warm beverages. As previously mentioned and retrospectively calculated (Chap. 1), assuming 100% sweating efficiency, the differences in sweat output (and thus potential for evaporative heat loss) seem to be disproportionate to the sensible heat transfer with the ingested fluid. Localized transient measurements of sweat rate may clarify why the sweating responses are not congruent with evaporative needs for heat balance. For example, a hot (e.g. above 45°C) beverage may elicit a psychological stress response, subsequently generating non-thermal sweating activity located predominantly on the forehead (McGregor 1952; Cramer et al. 2011) and other areas of glabrous skin (hairless) (Kuno 1956). In order to test this notion, transient responses in sweating activity must be simultaneously measured in three or more areas, covering both non-glabrous and glabrous skin tissue.

2.2.2 Cutaneous vasodilation

In thermoneutral (~25°C) environments and while at rest, the control of body temperature falls almost entirely on the manipulations of skin blood flow through maintaining an appropriate skin to air temperature gradient (Savage and Brengelmann 1996). Indeed, a constant *vasomotor tone* (Pergola et al. 1994) elicits levels of dry (convective, radiative and conductive) heat loss, that nearly match resting levels of metabolic heat production (the remaining metabolically generated heat is dissipated through evaporative heat loss at the skin from transepidermal water diffusion and respiratory heat loss). At rest, the rate of skin blood flow is approximately 250 mL/min, however during exercise and marked hyperthermia, levels can rise several fold, peaking at about 6 to 8 L/min (Nagashima 2006). Similar to sudomotor activity, the control of peripheral blood flow is mediated through a combination of thermal- and non-thermal inputs (Charkoudian 2010). However, during exercise (in the absence of dehydration), levels of cutaneous blood flow are mainly determined by thermal influences and thus requirements for heat loss. Indeed, dry heat loss at the skin becomes particularly relevant in humid conditions, where heat loss through evaporation is impaired (section 2.3.2). The mechanistic actions of cutaneous blood flow primarily stems from the constriction or dilation of peripheral blood vessel. This is accompanied by opening or closing of arterio-venous anastomoses (AVA) beneath the skin capillaries. Vasodilation and opening of AVA results in a greater skin blood flow, thereby increasing the rate of dry heat loss, while a vasoconstriction and AVA closing results in a reduction of skin blood flow, thus decreasing the rate of dry heat exchange and conserving heat within the body (Parsons 2003).

Following a rise in core and skin temperature, the reflex control of cutaneous blood circulation is primarily mediated through cholinergic activated sympathetic vasodilators (80% to 90%), which are otherwise dormant in resting thermoneutral conditions (Charkoudian 2003; Charkoudian 2010). In contrast, cholinergic activated sympathetic vasoconstrictors provide the *tonic vasomotor tone* at rest in thermoneutral environments. The withdrawal of which is responsible for the remaining 10% to 20% of cutaneous blood flow during heat stress (Pergola et al. 1994). Local skin temperature will also contribute to the control of cutaneous blood flow, independently of sympathetic whole-body reflexes. Acute heating of skin tissue will cause an initial rise in cutaneous blood flow through activation of local sensory nerves, which later (within ~10 min) continues to rise via nitric oxide stimulation (Charkoudian 2003).

To date, only one study, by Wimer et al. (1997), has investigated the influence of drink temperature upon modulations in peripheral blood flow during exercise. The authors noted that skin blood flow was reduced following cold (0.5°C) compared to warm (38°C) fluid ingestion, with concomitant reduction in skin temperature, and thus rate of radiative and convective heat loss. What remains to be seen is whether significant elevations in skin blood flow follow the ingestion of fluid warmer than body temperature (e.g. 50°C) compared to cold (<10°C) or even fluid at body temperature, and whether these changes translate into differences in sensible heat loss

2.3.0 Factors influencing heat loss during exercise

2.3.1 Hydration

Fluid within the body exists in both intracellular and extracellular spaces where, due to the semi-permeability construct of cell membranes, a relatively easy redistribution occurs between compartments. However, the shifts of fluid between the spaces are, in large part, determined by hydrostatic and osmotic pressures (Hall et al. 1996). Therefore, when a person becomes dehydrated, a marked decrease in blood plasma (hypovelemia) is apparent in addition to a state of hyperosmotic (increased solutes) fluids. This state mitigates the osmotic pressure for fluid movement, and in turn, inhibits fluid availability to the sweat gland (Sawka et al. 1985; Wendt et al. 2007). The hypovolemic condition also presents less blood to be circulated to the periphery. Furthermore, in vitro studies examining the effects of dehydration on thermal control show that when hypothalamic thermosensitive neurons are placed in a hyperosmotic medium, the firing rates decrease by nearly a half relative to when they are placed in a balanced medium (Silva and Boulant 1984; Nakashima et al. 1985). As such, dehydration will collectively alter blood flow, sweat rate and cardiovascular strain (Sawka 1992), to the detriment of thermoregulatory control.

While a loss of 1 to 2% body mass has been linked to sizeable decreases in exercise performance (Sawka 1992), by definition, a loss of fluid that equates to 2% of body weight or more, classifies dehydration (ICON 2004). Following profuse sweating, fluid losses can approach and in some cases exceed 6 to 9% (Pugh et al. 1967; Knechtle et al. 2005) thus generating severe levels of dehydration. As such, an inherent risk of dehydration accompanies exercise in the heat, particularly when fluid replacement is limited. It is therefore crucial that proper guidelines and recommendations are established to mitigate health related problems associated with dehydration (Sawka et al. 2007). Accurate sweat loss predictive equations are also required to determine the amount of fluid necessary to carry during endurance, occupational or military operations. To date, considerations of beverage temperature have been neglected in guidelines and predictive equations (Shapiro et al. 1982; Gonzalez et al. 2009). Existing literature has shown that the temperature of beverage probably has little influence upon absorption rate (Leiper et al. 2001). However, if exercise is prolonged, changes in sweat output associated with varying beverage temperature may yield differences in hydration status. Furthermore, in condition where sweating efficiency is compromised, the potential blunting ability of cold beverages may mitigate inefficient sweat losses, thereby conserving hydration status.

2.3.2 Sweating efficiency

The ability to dissipate heat largely depends upon the ambient environment. Indeed, environmental conditions favorable for heat loss (i.e. air temperature: $\leq$ 20°C, relative humidity: $\leq$ 40 % and air movement: $\geq$ 1.5 m/s) rarely conjure a state of uncompensable heat stress (Binkley et al. 2002). However, humid conditions with low air velocity, considerably reduces the potential for evaporative heat loss. The evaporative efficiency of sweating (η_{sw}), defined as the ratio between the amount of sweat evaporated from the skin and the total amount of excreted sweat (Candas et al. 1979) is, in large part, dependent upon the external vapor pressure and surrounding air movement. As such, by virtue of diverse ambient conditions, given levels of sweat output may yield large differences in evaporative heat loss, whereby η_{sw} alone can dramatically alter the rate of heat loss and subsequent body core temperature elevations. However, notwithstanding the driving forces that ambient conditions have upon η_{sw}, some physiological adaptations have been shown to mitigate inefficient sweat losses, namely; elevations in skin temperature and, more so, a distributed sweat output across a greater body surface area (Candas et al. 1979a; Candas et al. 1979b). Current literature suggests a greater skin temperature following warm compared to cold drinks (Burdon et al. 2010a), however, the effects of drink temperature upon the distribution of sweating is unknown.

Givoni (1963) was the first to show reductions in sweating efficiency, concluding that the percentage of evaporated sweat begins to decline as soon as the evaporative rate required to maintain heat balance (E_{req}) reaches 20% of the evaporation possible in a given environment (E_{max}). At the same time Kerslake (1963) demonstrated that on a vertical cylinder, 100% wettedness is observed when at least 40% of the excreted sweat drips off. However, in contrast to Givoni (1963), Candas et al, (1979a) found that the decline in efficiency begins in humans when the evaporative rate reaches approximately 74% of E_{max} (as opposed to 20%) (Figure 3).

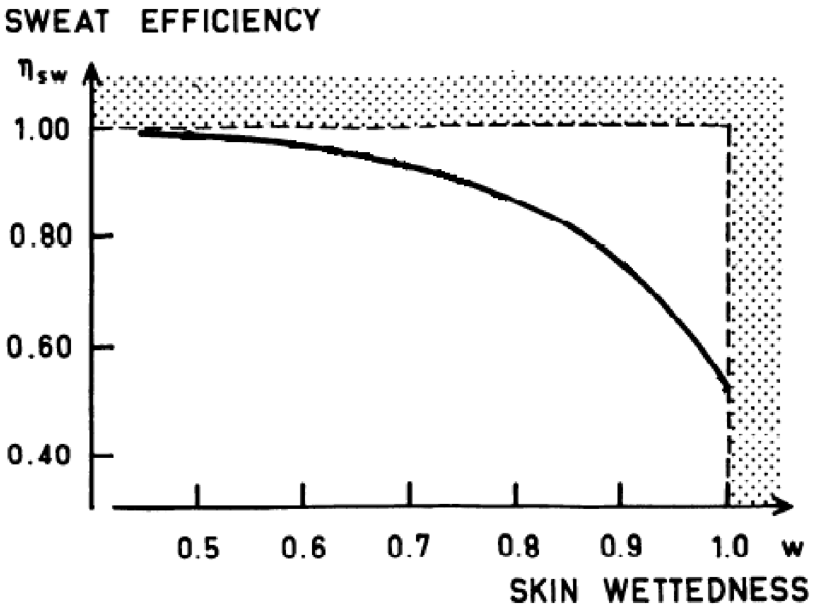

Figure 3. *Exponential relationship between sweating efficiency and skin wettedness. Sweating efficiency is dramatically reduced when skin wettedness approaches its maximum. Modified from Candas et al. (Candas et al. 1979b).*

Using the theoretical considerations presented by Candas et al. (1979a), in order to assure 100% η_{sw}, conditions must be set so that E_{req} is less than 74% of E_{max}. This ratio also provides a basis for describing whole body sweat rates (Shapiro et al. 1982; Gonzalez et al. 2009; Bain et al. 2011). (Retrospective calculations of data reported by Lee et al (2008a), indicate values for E_{req} that range between 74% and 85% of E_{max}.)

PART TWO:

METHODS AND RESULTS OF THE THESIS

Body heat storage during physical activity is lower with hot fluid ingestion under conditions that permit full evaporation

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SUBMITTED TO ACTA PHYSIOLOGICA – APH-2011-12-0330

ABSTRACT

Aim: To assess whether, under conditions permitting full evaporation, body heat storage during physical activity measured by partitional calorimetry would be lower with warm relative to cold fluid ingestion due to a disproportionate increase in evaporative heat loss potential relative to internal heat transfer with the ingested fluid. ***Methods*****:** Nine males cycled at 50% VO_{2max} for 75-min at 23.6±0.6°C and 23±11% RH while consuming water of either 1.5°C, 10°C, 37°C or 50°C in four 3.2 $mL•kg^{-1}$ boluses. The water was administered 5-min before, and 15, 30 and 45-min following, the onset of exercise. ***Results*****:** No differences in metabolic heat production, sensible or respiratory heat losses (all $P>0.05$) were observed between fluid temperatures. However, while the increased internal heat loss with cold fluid ingestion was paralleled by similar reductions in evaporative heat loss potential at the skin (E_{sk}) with 10°C ($P=0.08$) and 1.5°C ($P=0.55$) fluid; the increased heat transfer with warm (50°C) fluid ingestion was accompanied by a significantly greater E_{sk} ($P=0.04$). The resultant calorimetric heat storage was lower with 50°C water ingestion in comparison to 1.5°C, 10°C and 37°C (all $P<0.05$). In contrast, heat storage derived conventionally using thermometry yielded higher values following 50°C fluid ingestion compared to 1.5°C ($P=0.025$). ***Conclusion*****:** Under conditions permitting full sweat evaporation, body heat storage is lower with warm water ingestion, likely due to disproportionate modulations in sweat output arising from warm-sensitive thermosensors in the esophagus/stomach. Local temperature changes of the rectum following fluid ingestion exacerbate the previously identified error of thermometric heat storage estimations.

KEYWORDS: Drink temperature, Fluid replacement, Evaporation, Heat balance, Sweat output

INTRODUCTION

The evaporation of sweat at the skin provides the greatest potential for physiological heat dissipation; however, poor hydration maintenance can have detrimental impacts upon sweat output, and subsequently evaporative heat loss (Sawka et al., 2001). Fluid guidelines and succeeding active measures of fluid replacement for both the general public and athletes are therefore necessary to reduce the risk of heat-related illnesses, particularly during physical activity (Sawka et al., 2007).

An often overlooked component to fluid replacement is the potentially large and direct influence that fluid temperature may have on body heat storage. Indeed, a recent amendment to fluid replacement guidelines suggests that ingested fluids be cooled (i.e. below 10°C) (Burdon et al., 2010a). These recommendations comply with recent literature reporting reductions in body heat storage following cold compared to warm fluid ingestion (Lee et al., 2008a, Lee et al., 2008b, Lee and Shirreffs, 2007, Siegel et al. 2010) during physical activity in the heat. However, all of these studies have assessed the influence of ingested fluid temperature on body heat storage using a weighted two-compartment thermometric model of “core” and “shell” represented by changes in rectal and mean skin temperature respectively; and this method has been conclusively proven to be erroneous (Jay et al., 2007a, Jay et al., 2007b, Sawka and Castellani, 2007). Furthermore, cold fluid residing in the stomach may cool the tissue surrounding the rectum thereby exacerbating the thermometric error of whole-body heat storage estimations even further.

An analysis of previous data indicates that warm fluid ingestion may be paralleled by greater than expected increases in sweat output and thus evaporative heat loss when compared to the ingestion of a cold fluid (Wimer et al., 1997, Lee et al., 2008a). For example, Lee et al. (2008a) observed a 260 ml increase in whole-body sweat loss during 90-min of fixed intensity cycling while ingesting a 50°C compared to a 10°C fluid. Such an increase in sweat output would equate to a net heat loss potential (a combination of heat lost/gained from the ingested fluid and evaporative heat loss through sweating) that is ~363 kJ greater with the ingestion of 50°C fluid than a 10°C fluid. Under environmental conditions that permit full evaporation, this disproportionate increase in insensible heat loss relative to the internal transfer of heat energy between a warm ingested fluid and the body, should in fact lead to a lower body heat storage in comparison to the ingestion of a cold fluid.

However, a potential confounding variable with the above heat balance analysis may be differences in sweating efficiency (i.e. the proportion of secreted sweat that evaporates). According to work by Candas et al. (Candas et al., 1979), sweating efficiency begins to decline when the rate of evaporation at the skin required for heat balance (E_{req}) reaches ~70% of the maximal evaporation possible in the environment (E_{max}). Average data from Lee et al. (2008a) show that E_{req} ranged between ~85% and ~90% of E_{max} which, as previously recognized by the authors (Lee et al., 2008a), may have compromised sweating efficiency. Therefore, in order to ascertain the true influence of fluid temperature on body heat storage, experimental conditions (i.e. combination of climate and environment) must be set to permit full evaporation (i.e. E_{req} < 70% of E_{max}). From a public health perspective, understanding human heat balance following the ingestion of fluids of different temperatures under such conditions is ecologically valid since it is improbable that non-athletes among the general population are regularly exposed to combinations of climate and activity that yield large decrements in sweating efficiency even during the summer months. Furthermore, the consumption of warm beverages either before or during light-to-moderate physical activity is commonplace in many working environments and among many members of the general public.

The purpose of this study was to use partitional calorimetry to 1) assess whole-body heat storage during moderate intensity physical activity under conditions that permit full sweat evaporation (i.e. $E_{req} < 70\% \ E_{max}$) while ingesting fluid (water) of different temperatures and 2) compare these heat storage data to those simultaneously estimated using the conventional two-compartment thermometric model of “core” and “shell” employed in all other previous studies. It was hypothesized that in opposition to the previous literature 1) a lower net body heat storage would be measured calorimetrically following the ingestion of a warm (50°C) relative to a cold (1.5°C) fluid due to a disproportionate increase in the potential for evaporative heat loss at the skin, relative to the internal heat transfer with a warm ingested fluid; and 2) compared to calorimetrically derived values, the ingestion of a cold fluid would exacerbate the error of thermometric estimations of body heat storage.

MATERIALS AND METHODS

Participants

Nine non-heat acclimated male Caucasians (mean age: 22±2 yr, body mass; 80.5±9.7 kg, VO_{2peak}: 53.4±3.6 $mL \bullet min^{-1} \bullet kg^{-1}$) volunteered for four experimental sessions and one preliminary session. Participants refrained from exercise the day prior to, and the morning before all experimental sessions and were also instructed to maintain a consistent routine (e.g. sleep schedules) during that time. No caffeine or alcohol was consumed 48 hrs before testing. A food log was kept the night and morning before the first session, which was subsequently used as a staple diet for all remaining sessions. Before each session, urine samples were provided and analyzed for urine specific gravity (Reichert TS 400, Depew, NY) to assure the same levels of hydration before experimentation. During the preliminary session, total body mass, height, and peak oxygen consumption were measured. Peak oxygen consumption (VO_{2peak}) was measured using an upright cycle ergometer protocol consisting of a two minute warm up at 40 W followed by cycling at 100 W for the third minute with a 20 W increase every minute thereafter until physical exhaustion. This protocol was based upon recommendations from the Canadian Society of Exercise Physiology (CSEP, 1986). All participants were screened for cardiovascular and metabolic health disorders before consenting to the study. The study was approved by the University of Ottawa Research Ethics Committee and conforms to the Declaration of Helsinki.

Experimental Protocol

Participants were instructed to cycle for 75 minutes at 50% of their maximal oxygen uptake on an upright cycle ergometer (Kettler Ergoracer 7988-899, Ense, Germany) for four experimental conditions. The conditions were separated by ingesting tap water of either 1.5°C, 10°C, 37°C or 50°C, administered in a counter-balanced order, and separated by at least 48 hours but no more than one week. During each session water was ingested in four 3.2 $mL•kg^{-1}$ boluses (equating to an average of 942 ± 0.07 mL), the first at 5-min before exercise, and the following three after 15, 30 and 45-min of exercise. The volume of water was chosen to standardize for body mass while providing similar drink volumes as previous literature (Lee and Shirreffs, 2007). The water temperature at 37ºC and 50ºC was maintained using a hydrostatic controlled water bath (Polyscience – DA05A, Niles, IL, USA). Water at 1.5ºC was maintained by keeping ice-water in a thermos. Water at 10°C was maintained using a standard office water cooler (Crystal Mountain – MOEF2WTW18C, Edmonton, AB, Canada). The temperature of the water before each ingestion was measured using a glass thermometer with an accuracy of ± 0.1°C and recorded for subsequent sensible heat transfer calculations. Fluid temperatures did not vary more than ± 0.5°C from 1.5°C, 10°C, 37°C or 50°C for any participant. The ambient air temperature and relative humidity was similar between all participants (23.6±0.6°C, 23±11% RH) and within participant sessions (±0.4ºC and ± 5% RH). A mechanical fan placed 1.25 m in front of the participants produced a mean whole body air velocity of 0.75 $m•s^{-1}$, measured using a hot wire anemometer (Omega Engineering, Stamford, CT, USA). Participants were semi-nude with clothing standardized between all sessions consisting of only light running shorts, socks and shoes (0.1 clo). This specific combination of exercise intensity, environmental conditions and clothing was selected to ensure that the rate of evaporation occurring at the skin (E_{sk}) did not exceed 70% of the maximum rate of evaporation possible in the ambient environment (E_{max}), and thus maintain sweating efficiency at ~100% (Candas et al., 1979). All within-subject experimental sessions were completed at the same time of the day.

Instrumentation

Thermometry: 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. Esophageal temperature (T_{es}) was measured by placing a pediatric thermocouple probe through the participant's nostril into the esophagus. The location of the probe tip in the esophagus was estimated to be at the level of the eighth and ninth thoracic vertebrae reflecting the region of the left ventricle and aorta (Mekjavic and Rempel, 1990). Skin temperature (T_{sk}) was measured at 8 points over the right side of the body using 0.3-mm diameter T-type (copper/constantan) thermocouples integrated into heat flow sensors (Concept Engineering, Old Saybrook, CT, USA). The probes were attached using double-sided adhesive discs and surgical tape (Transpore, 3M, London, ON, Canada). Mean skin temperature was weighted using the following regional proportions: forehead 7%, chest 17.5%, hand 5%, thigh 19%, scapula 17.5%, calf 20%, shoulder 7%, triceps 7% (ISO-9886, 1992) All thermometry data were collected using a National Instruments data acquisition module (model NI cDAQ-9172) at a sampling rate of 5 s. Data were simultaneously displayed and recorded in spreadsheet format on a personal computer (Dell Inspirion 545) with LabVIEW 2009 software (National Instruments, TX, USA).

Whole-body sweat loss (*WBSL*): was measured by placing a platform scale (Combics 2, Sartorius, Missiauga, ON, Canada) next to the cycle ergometer and taking the difference of weight measurements before and after exercise to the nearest 2 g. An average of three separate measurements was used both before and after exercise. The subjects were weighed fully instrumented exactly 10-min before the onset of exercise and within 1-min of completing exercise. The instrumented wires were secured at exactly the same point to a cart which was placed in the same location upon pre- and post-weighing. Saliva that accumulated in the spit valve and ingested water were weighed and subsequently accounted for when calculating changes in body mass. Corrections were further made for evaporative and metabolic mass loss using Eq. 6 and 7 respectively.

Indirect calorimetry: measurements were performed throughout exercise using a Vmax® Encore Metabolic Cart (CareFusion, San Diego, CA). Subjects were equipped with a mouth piece and nose clip and were instructed to breathe normally through the mouth piece. The mouth piece was removed only briefly while the participants consumed the boluses of water.

Calculations - Partitional calorimetry

Calorimetric estimations of heat storage (S_{cal}) were calculated using an adapted form of the conceptual heat balance equation (Eq. 1) (Parsons, 2003) by including the heat transfer with the ingested water (H_{water}).

$$S = (\mathrm{M} - W) - (C + R) - (C_{res} + E_{res}) - E_{sk} - H_{water} \ldots\ldots\ldots \mathrm{kJ} \ldots\ldots\ldots (1)$$

Where;

Metabolic energy expenditure (*M*): was calculated from minute-average values for oxygen consumption (VO_2) in Liters/min, and the respiratory exchange ratio (*RER*) using Eq. 2 (Nishi, 1981).

$$M = VO_2 \frac{\left(\frac{RER-0.7}{0.3} e_c\right) + \left(\frac{1-RER}{0.3} e_f\right)}{60 \times BSA} \times 1000 \ldots\ldots\ldots \text{W•m}^{-2} \ldots\ldots\ldots (2)$$

Where e_c is the caloric equivalent per litre of oxygen for the oxidation of carbohydrates (21.13 kJ); e_f is the caloric equivalent per litre of oxygen for the oxidation of fat (19.62 kJ); and *BSA* is body surface area - calculated using Dubois and Dubois (DuBois and Dubois, 1916).

External work (*W*): was regulated and measured directly using the upright cycle ergometer (Kettler Ergoracer 7988-899, Ense, Germany).

Sensible heat loss at the skin (*C* + *R*): The combined rate of convective (*C*) and radiative (*R*) heat exchange at the skin was calculated using Eq. 3 and 4 respectively (Parsons, 2003):

$$C = f_{cl} h_c (t_{cl} - t_a) \ldots\ldots\ldots \text{W•m}^{-2} \ldots\ldots\ldots (3)$$

$$R = f_{cl} h_r (t_{cl} - t_r) \ldots\ldots\ldots \text{W•m}^{-2} \ldots\ldots\ldots (4)$$

Where; f_{cl} is clothing area factor (since participants were very lightly clothed, this was assumed to be equal to BSA). t_{cl} is mean temperature of the clothed body in ºC (assumed to be equal to T_{sk}). Thermal resistance of clothing was assumed to be negligible since participants were semi-nude. h_c is the convective heat transfer coefficient in $Wm^{-2}K^{-1}$ and was estimated to be $8.3v^{0.6}$ (Parsons, 2003), where v equals air velocity in m•s^{-1}. h_r is the linear radiative heat transfer coefficient in $Wm^{-2}K^{-1}$. h_r was calculated using Eq. 5 (Parsons, 2003):

$$h_r = 4\varepsilon\sigma\frac{A_r}{BSA}\left[273.2 + \frac{(t_{cl} + t_r)}{2}\right]^3 \text{.........W•m}^{-2}\text{K}^{-1} \text{..(5)}$$

Where; ε is the area weighted emissivity of the clothing body surface (assumed to be 1.0); σ is the Stefan-Boltzmann constant, 5.67×10^{-8} $Wm^{-2}K^{-4}$; A_r/BSA is the effective radiative area of body in m^2 (assumed to be 0.71 since the participants were seated(Fanger, 1970)); t_r is mean radiant temperature in ºC and assumed to be equal to t_a.

<u>Evaporative heat loss at the skin (E_{sk})</u>: was calculated from whole body sweat loss measurements where each mL of evaporated sweat required the release of 2.426 kJ (Wenger, 1972). Corrections for respiratory and metabolic mass losses were accomplished by subtracting mass loss calculated through Eq. 6 and 7 (Kerslake, 1972).

$$\text{Evaporative mass loss from respiration} = E_{res}\left(\frac{t}{2426}\right) \text{..........(g)........................(6)}$$

Where: E_{res} is the evaporative loss through respiration (in W), calculated using the second half of Eq. 8 and multiplying by body surface area; and t is the time of exercise (s)

Metabolic mass loss = $t\left(\frac{VO_2(44(RER-32))}{22.4}\right)$……….(g)...(7)

Where: VO_2 is the rate of oxygen consumption (L•min^{-1}), *RER* is the respiratory exchange ratio; and *t* is the time of exercise (min)

Heat loss from respiration (C_{res} + E_{res}): The combined rate of convective and evaporative respiratory heat loss was calculated using Eq. 8 (Parsons, 2003).

$C_{res} + E_{res} = [(0.0014(M-W)(34-t_a)) + (0.0173(M-W)(5.87-P_a))]$……….W•m^{-2}..(8)

Where; $M - W$ is the rate of metabolic heat production (in W•m^{-2}), P_a is the ambient vapour pressure and t_a is the ambient air temperature.

Heat loss from body to ingested fluid (H_{fluid}): was calculated using Eq. 9.

$H_{fluid} = \frac{(T_{core} - T_{fluid}) \times C_{p(fluid)} \times mass_{fluid}}{1000}$ ……….kJ...........…………………..….(9)

Where: T_{core} is rectal temperature; T_{fluid} is ingested fluid temperature; $C_{p(fluid)}$ is the specific heat capacity of the ingested fluid (e.g. 4.184 J/g/°C if ingested fluid is water); $mass_{fluid}$ is the mass of ingested fluid (estimated using pre and post measurement of cup) in grams.

Calculations – Thermometry

Change in mean body temperature (ΔT_b): was calculated using the traditional two-compartmental model (Hardy and DuBois, 1938, Stolwijk and Hardy, 1966) using changes in skin and core temperature, Eq. 10;

$\Delta T_b = X \bullet \Delta T_{core} + (1 - X \bullet \Delta T_{sk})$.........°C..(10)

Where; X represents the proportional influence of core temperature on ΔT_b, chosen to be 0.8, based upon literature from Hardy and Dubois (1938) and Stolwijk and Hardy (1966) for thermoneutral environments; ΔT_{core} represents the change in T_{re} or T_{es} and ΔT_{sk} represents the change in mean skin temperature.

Thermometric estimations of heat storage (S_{therm}): were derived using the average specific heat capacity of human tissue (Geddes and Baker, 1967), body mass and ΔT_b, Eq. 11;

$S_{therm} = 3.47 \bullet mass \bullet \Delta T_b$.........kJ..(11)

Where; *mass* equals the participants body mass in kg, and ΔT_b is in °C.

Thermometric estimations of heat storage using the change in mean body temperature derived using ΔT_{es} as the ΔT_{core} component are depicted as S_{therm_Tes}, whereas the change in mean body temperature derived using ΔT_{re} as the ΔT_{core} component are depicted as S_{therm_Tre}.

Statistical Analysis

All data are expressed as a mean with standard deviation (±SD). All calorimetric components calculated in $W \bullet m^{-2}$ were multiplied by body surface area and exercise time (in seconds) which was then divided by 1000 to yield values in kJ. The change in evaporative heat loss at the skin (E_{sk}) relative to 37°C fluid ingestion following 1.5°C, 10°C and 50°C fluid ingestion was compared to the change in the evaporative requirement for heat balance (E_{req}) relative to 37°C fluid ingestion following 1.5°C, 10°C and 50°C fluid ingestion, using a one-way repeated measures ANOVA. E_{req} was calculated by subtracting all non-evaporative heat losses from metabolic heat production, i.e. (M-W) – (C+R) – (C_{res}+E_{res}) - H_{fluid}. Pre-planned simple directional contrasts were used to compare WBSL, change in T_{re}, T_{es}, T_{sk} S_{cal}, S_{therm_Tre} and S_{therm_Tes} data at each fluid temperature to the warmest fluid temperature (50°C). A two-way repeated measures ANOVA was used to analyze heat storage values estimated using thermometry and partitional calorimetry by employing the factors of method (2 levels: thermometry and calorimetry) and fluid temperature (4 levels: 1.5°C, 10°C, 37°C and 50°C). These analyses were performed separately for S_{therm_Tre} and S_{therm_Tes}. The probability of making a type I error in all tests was maintained at 5% using a Holm-Bonferroni correction. All analyses were performed using the statistical software package SPSS 18.0 for Windows (SPSS Inc. Chicago, IL, USA).

The estimation error of thermometrically derived heat storage values (S_{therm}) relative to heat storage values measured using partitional calorimetry (S_{cal}) were also assessed by calculating the mean percentage error at each drink temperature (i.e. 1.5°C, 10°C, 37°C and 50°C) with 95% confidence intervals. The percent error was defined using:

$$\%Error = \frac{(S_{therm} - S_{cal})}{S_{cal}} \times 100 \;\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots \text{(12)}$$

RESULTS

Whole-body sweat losses (WBSL)

Differences in WBSL are depicted in Figure 1. With the ingestion of 50°C fluid, WBSL was significantly greater than with the ingestion of 1.5°C ($P<0.001$), 10°C ($P=0.001$) and 37°C ($P=0.029$) fluid.

Heat Balance Parameters

The present study was designed to attain a combination of activity, climate and clothing permitting full sweat evaporation from the skin. Table 1 details the mean rate of evaporation at the skin (E_{sk}), the maximum rate of evaporation possible in the ambient environment (E_{max}) and the mean skin wettedness (w) estimated by determining the ratio of E_{sk} relative to E_{max}. While w became greater with increasing ingested fluid temperature due to a greater E_{sk}, w remained well below the level of skin wettedness at which marked decrements in sweating efficiency have been found to occur (i.e. ~0.70).

Heat storage components with partitional calorimetry

Table 2 depicts the components of the partitional calorimetry calculations. No significant differences were observed between fluid temperatures for metabolic heat production, dry heat loss at the skin ($C+R$) or respiratory heat loss (C_{res} + E_{res}) (all $P>0.05$). As expected, internal heat transfer from the body to the ingested fluid (H_{fluid}) was significantly greater with decreasing fluid temperature ($P<0.001$), and negative with a 50°C fluid (i.e. heat transfer occurred in the opposite direction - from the fluid to the body).

However, the change in the evaporative requirement for heat balance (E_{req}) with the ingestion of fluid at different temperatures was not proportional to the change in the potential for evaporative heat loss at the skin (E_{sk}) (Figure 2). Relative to the thermoneutral fluid temperature of 37°C, the reduction in E_{req} (due to a greater H_{fluid}) was similar to the reduction in E_{sk} with 10°C ($P=0.081$) and 1.5°C ($P=0.549$) fluid ingestion; however the increase in E_{sk} with 50°C fluid ingestion was significantly greater than the increase in E_{req} (due to a negative H_{fluid}) ($P=0.039$).

The total body heat storage measured calorimetrically (S_{cal}) at each fluid temperature is presented in Figure 3. Compared to 50°C fluid ingestion, body heat storage was significantly greater ($P<0.05$) with 37°C, 10°C and 1.5°C water ingestion. However, fluid ingestion at 37°C and 10°C did not elicit lower levels of heat storage compared to 1.5°C ($P=0.372$ and $P=0.902$ respectively).

Core and Skin Temperatures

Absolute pre- and end-exercise values for T_{re}, T_{es} and T_{sk} are given in Table 3. Compared to 50°C, the change in T_{re} from pre-exercise to end-exercise was significantly smaller following 1.5°C ($P=0.011$) and 10°C ($P=0.008$) fluid ingestion, but not following 37°C ($P=0.695$) fluid ingestion. On the other hand, no differences were observed between fluid temperatures for changes in T_{es} or T_{sk} during exercise (all $P>0.05$).

Comparison between heat storage estimations with thermometry and calorimetry (S_{cal})

Heat storage estimations were significantly lower using the T_{re} thermometry model (S_{therm_Tre}) relative to partitional calorimetry (S_{cal}) ($P=0.039$). In addition, a significant interaction was observed between method and fluid temperature ($P=0.002$).

In fact, the influence of fluid temperature on heat storage values was the opposite using thermometry in comparison to calorimetry (Figure 3). While as previously mentioned, heat storage values were significantly lower with increasing fluid temperature when measured using partitional calorimetry; heat storage values estimated using the T_{re} thermometry model were greater with increasing fluid temperature. In comparison to 50°C, S_{therm_Tre} was greater following ingestion of 1.5°C ($P=0.025$) and 10°C ($P=0.023$), but not 37°C ($P=0.747$)

Relative to S_{cal}, heat storage estimations were again significantly lower using the T_{es} thermometry model (S_{therm_Tes}) ($P=0.003$), and similarly to the T_{re} thermometry model a significant interaction was observed between method and fluid temperature ($P=0.037$). However, heat storage estimations using the T_{es} thermometry model were not significantly different between any fluid temperatures (all $P>0.05$) (Figure 3).

Relative to S_{cal}, mean percentage error (with upper and lower bound 95% confidence intervals in parentheses) of S_{therm_Tre} were between -42.7% (-32.8, -52.5) for 1.5°C fluid, -43.6% (-31.7, -55.5) for 10°C fluid, -27.4% (-20.4, -34.4) for 37°C fluid and +6.6.% (+20.7, -7.6) for 50°C fluid. Mean percentage error of S_{therm_Tes} were between -51.1% (-42.6, -59.6) for 1.5°C fluid, -52.1% (-40.8, -63.5) for 10°C fluid, -44.9% (-32.3, -57.5) for 37°C fluid and -23.4% (-10.1, -36.6) for 50°C fluid.

DISCUSSION

By using partitional calorimetry, the present study demonstrates that during exercise in an environment that permits full sweat evaporation, the ingestion of 50°C water appears to in fact yield a lower body heat storage compared to the ingestion of colder water. These differences were attributed exclusively to disproportionate modulations in sweat output and thus the potential for evaporative cooling; i.e., the increase in evaporative potential with 50°C fluid ingestion was greater than the heat absorbed by the body from the warm ingested water (Figure 2). Furthermore, a comparison of thermometric and calorimetric estimations of heat storage (Figure 3) reveals that discrepancies between the two methods become increasingly exacerbated as ingested fluid temperature declines. Specifically, when estimated with thermometry using rectal temperature to represent the “core” component of the two-compartment model, estimations of heat storage decreased with declining fluid temperature; however when heat storage was measured calorimetrically the exact opposite was observed, whereby heat storage decreased with increasing fluid temperature.

The partitional calorimetric data in the present study is in direct opposition to previous reports of a lower body heat storage with the ingestion of cold water (Lee et al., 2008b, Lee et al., 2008a, Lee and Shirreffs, 2007, Siegel et al.). On the other hand, the thermometrically derived estimations of body heat storage using T_{re} are similar to previous observations. Existing literature using whole-body direct calorimetry has conclusively shown that thermometric estimations of heat storage using either T_{re} or T_{es} as a representative of the “core” compartment systematically under estimate whole body heat storage (Jay et al., 2007a, Jay et al., 2007b, Sawka and Castellani, 2007). In the present study, the opposite influence of ingested water temperature on heat storage estimations with the T_{re} thermometry model relative to partitional calorimetry likely stems from local temperature changes of the tissue surrounding the rectal temperature measurement site. Indeed, during physical activity the stomach and viscera receive proportionately lower blood flow compared to the working muscles and other parts of the body (Flamm et al., 1990). Therefore, a large proportion of the heat transfer with the water will be confined to the viscera and surrounding tissue as the limited blood flow would potentially mitigate distribution throughout the body. This in turn would have elicited a residual effect on T_{re}, thereby generating smaller changes in core temperature during physical activity with the ingestion of colder fluids despite a greater whole-body heat storage. By virtue of the abovementioned rationale, changes in tissue temperature of regions excluding the gut and surrounding tissue (e.g. the arms and legs) must have theoretically been higher following 1.5°C compared to 50°C fluid ingestion. It is therefore likely that had exercise been prolonged, the local heat sink in the gut would have been eradicated by the greater heat storage in the peripheral tissue and subsequently lead to measurably greater increases in rectal temperature with 1.5°C relative to 50°C fluid ingestion. Partial support for the above rationale may be found in our esophageal temperature data which demonstrates a nearly identical change during exercise (Figure 2) and subsequent estimations of heat storage (S_{therm_Tes}) (Figure 3) following 50°C and 1.5°C fluid ingestion. This is likely a result of the higher blood circulation near the esophagus, which was able to partially offset the acute local temperature change during water ingestion.

To our knowledge, only one study (Wimer et al., 1997) has employed a calorimetric approach to estimate heat storage following fluid ingestion of varying temperatures. Similarly to the present study, they observed a greater heat storage following ingestion of fluids at 0.5°C, compared to 26°C and 38°C. Furthermore, they also found that 0.5°C fluids yielded the greatest attenuation in the rise of core temperature. Therefore, both studies collectively support the notion that a thermometric analysis following cold drink ingestion is a poor indicator of heat storage. However, the data from Wimer et al. (1997) are limited to water temperatures equal to or below body temperature, and their experimental conditions, which were warmer and more humid than the present study, may have induced some decrements in sweating efficiency which were almost certainly avoided, by design, in the present study. It follows that the present data show that disproportionate modulations in sweat output are far more evident with ingested fluids above body temperature (i.e. 50°C) than below (i.e. 10°C and 1.5°C). Specifically 50°C fluid elicited a heat storage that was on average 95 ± 38 kJ lower than following the ingestion of water at body temperature (37°C) (calorimetric data). Whereas 1.5°C and 10°C fluid ingestion elicited a heat storage that was, in comparison to 37°C fluid ingestion, greater by 29 ± 30 kJ and 35 ± 53 kJ respectively (Figure 3). Therefore, it seems that the mechanism underpinning heat storage differences is more attributable to disproportionate elevations in sweat output following hot fluid ingestion rather than disproportionate decreases following the ingestion of fluids cooler than body temperature.

The explanation for the disproportionate modulation in sudomotor activity likely lies within the unique characteristics of temperature sensing neurons in the stomach, esophagus and large intra-abdominal veins. It has been previously demonstrated that both warm (Rawson and Quick, 1972, Riedel, 1976) and cold sensitive (Gupta et al., 1979) thermosensors reside in the abdomen, providing thermoreceptor input through splanchnic and vagus nerve afferent fibers in response to deep tissue temperature. Although the abdominal thermosensors are considered peripheral by definition (Nakamura, 2011, Romanovsky, 2007), our WBSL data indicate that they display a hierarchical contribution to thermal control, and likely influence thermo-effector responses more than those located in the cutaneous tissue. This notion is supported by early work by Rawson and Quick (1972), who demonstrated that locally warming or cooling the intra-abdominal tissue in the ewe elicits strong thermo-effector responses (i.e. panting and shivering respectively), without changes in hypothalamic temperature. Therefore the disproportionate increase in sweat output following 50°C fluid ingestion is probably a result from over compensatory thermosensor activity derived exclusively from the local thermal environment in the abdomen, which was not representative of a distributed internal body temperature.

By design, the combination of environmental conditions and metabolic heat production should have theoretically maintained sweating efficiency close to 100%. According to Candas et al. (1979) skin wettedness, described as the ratio of sweat produced to the maximal evaporation possible in a given environment, when ~0.74 or less, should result in ~100% evaporation. Retrospective calculations revealed that mean skin wettedness throughout 1.5°C, 10°C, 37°C and 50°C water ingestion in the present study were well below this critical level (Table 1). We are therefore confident that most if not all measured sweat loss was evaporated from the skin and heat loss was not over estimated in our calculations (this was supported by observing no dripped sweat from the participants). However, had conditions been such that sweating efficiency was not maintained, changes in sweat output would not have been paralleled by proportional changes in evaporative heat loss. It follows that the present data are only applicable to conditions permitting 100% evaporation. Therefore, had a similar calorimetric analysis been performed during exercise in a hot and humid environment (while controlling for dripped sweat) the results may have been different, however more work is required to conclusively determine differences in heat storage as a result from changes in sweating efficiency. As such, this study does not necessarily refute the recent sequence of literature (e.g. (Burdon et al., 2010b, Lee et al., 2008b, Ross et al., 2011)) claiming a reduced heat strain with cold drink ingestion during or before exercise in hot/humid conditions, rather; we provide a provision by suggesting that the validity of this claim is dependent upon the combination of environmental conditions and exercise intensity.

In conditions that are unfavourable for full evaporation, our findings in fact indirectly support cold drink ingesting by demonstrating that colder fluids would minimize inefficient sweat losses, and may subsequently aid hydration status. Indeed, our WBSL data show that changing ingested fluid temperature from 50°C to 1.5°C elicits a reduction in sweat loss of ~20% (Figure 1). Had exercise been prolonged beyond 75-min, or conducted in an already dehydrated state it follows that hydration status would have likely been better maintained with the cold water. Therefore, in addition to describing differences in heat storage, our data can be potentially extended to fluid replacement guidelines, specifically under occupational and military settings that are currently based on existing sweat loss prediction equations and do not include the considerations of ingested fluid temperature (Gonzalez et al., 2009, Shapiro et al., 1982). Furthermore, the apparent non linearity of the sweating responses in relation to changes in fluid temperature (Figure 1) indicates that if ingesting fluids warmer than body temperature is not viable, or conditions are not favourable for full evaporation, colder fluids (i.e. ~10°C) may better help attenuate rising levels of heat storage, as differences in evaporative potential are apparently much smaller with declining temperatures below 10°C (Table 2).

The present study did not use direct calorimetry to measure whole-body heat loss; therefore estimations of heat storage are dependent upon the accuracy of the measurements used for the partitional calorimetric analyses. However, the percentage error in using thermometry vs. partitional calorimetry in the 37°C condition, is comparable to that found in existing literature that employed direct calorimetry (i.e. a mean error of 27% in the present study relative to a mean error of ~30% previously reported with direct calorimetry during exercise in a 24°C environment) (Jay et al., 2007b). The accuracy of our methods were further promoted by providing corrections for metabolic, respiratory and salivary mass loss from whole-body mass losses and by fixing the experimental conditions so that sweating efficiency was maintained at ~100%, thereby minimizing the potential for over estimations of evaporative heat loss. However, had evaporative heat loss been overestimated, the discrepancy between thermometric and calorimetric estimations of heat storage would have been even greater, thereby providing further evidence for local temperature changes of the tissue surrounding the rectal measurement site exacerbating the error of thermometric heat storage estimations. Furthermore, accurate estimations of sensible heat loss at the skin were assured by measuring skin and air temperature within ± 0.1°C and by maintaining air velocity constant between all conditions, which was verified with an accuracy of ± 0.1 $m \cdot s^{-1}$ with hot wire anemometry. Future research should investigate whether measureable increases in the rise of core temperature following cold compared to hot fluid ingestion can be observed by conventional core temperature measurements in the rectum and esophagus when physical activity is extended to ~2 hours or beyond. Moreover, additional work is required to conclusively tease out changes in hydration and heat storage following fluid ingestion of varying temperature, particularly under environmental conditions that elicit varying degrees of sweating efficiency. Finally, future analyses following fluid ingestion of varying temperature during physical activity should focus on examining the transient responses of skin blood flow and local sweat rates, which may help elucidate the underlying mechanisms for the apparent reductions in net heat loss with cold drink ingestion.

In conclusion, under conditions permitting full sweat evaporation, a partitional calorimetric analysis demonstrated that ingesting fluid of 50°C elicited a significantly lower body heat storage during moderate intensity physical activity compared to 1.5°C, 10°C and 37°C fluid ingestion. Differences in heat storage were attributed to disproportionate modulations in sweat output, whereby increases in evaporative potential with 50°C fluid ingestion were greater than the internal heat transfer occurring between the body and fluid. Furthermore, using calorimetrically-derived heat storage values as a reference, the present study demonstrated that the previously indentified error of thermometric heat storage estimations are exacerbated with the ingestion of cold fluid, thus describing the discrepancy between the influence of fluid temperature on body heat storage during physical activity determined calorimetrically in the present study, and thermometrically in all other previous studies.

ACKNOWLEDGEMENTS

This research was supported by a Natural Science and Engineering Research Council (NSERC) of Canada Discovery Grant (Grant Holder – Ollie Jay). Mr. Bain was supported by a University of Ottawa Master's Scholarship and an Ontario Graduate Scholarship. The authors would like to thank the volunteering participants and the members of the Thermal Ergonomics Laboratory who contributed to data collection.

Conflicts of Interest

None.

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Table 1. Skin wettedness (*w*) as estimated by the ratio of evaporation at the skin (E_{sk}) to the maximal evaporation possible (E_{max})

Fluid Temperature	1.5°C	10°C	37°C	50°C
E_{sk} ($W \bullet m^{-2}$)	133 ± 18	140 ± 28	151 ± 20	163 ± 18
E_{max} ($W \bullet m^{-2}$)	288 ± 27	295 ± 26	287 ± 30	295 ± 26
E_{sk}:E_{max} (w)	0.46 ± 0.08	0.47 ± 0.07	0.53 ± 0.05	0.55 ± 0.06

Values are means ± SD

Table 2. Cumulative values for each component of human heat balance measured using partitional calorimetry after 75-min of exercise at 50% VO_{2max} with the ingestion of 1.5°C, 10°C, 37°C and 50°C water

Fluid Temperature	1.5°C	10°C	37°C	50°C
M-W	2664 ± 211	2676 ± 333	2654 ± 255	2601 ± 199
C+R	770 ± 76	754 ± 55	771 ± 57	760 ± 56
$C_{res} + E_{res}$	279 ± 25	283 ± 42	275 ± 35	272 ± 26
H_{water}	141 ± 11*	104 ± 8*	3 ± 1*	-49 ± 4
E_{sk}	1130 ± 159*	1184 ± 274*	1287 ±164 *	1396 ± 180

Values are means ± SD in kilojoules (kJ). Asterisk (*) denotes a significant difference relative to 50°C water ingestion

Table 3. Core and skin temperature prior to and at the end of 75-min of exercise at 50% VO_{2max} with the ingestion of 1.5°C, 10°C, 37°C and 50°C water

Fluid Temperature	1.5°C	10°C	37°C	50°C
Pre-exercise T_{re}	37.2 ± 0.2	37.1 ± 0.2	37.1 ± 0.2	37.1 ± 0.3
End-exercise T_{re}	38.0 ± 0.3*	37.9 ± 0.3*	38.0 ± 0.2	38.1 ± 0.2
Pre-exercise T_{es}	37.0 ± 0.2	36.9 ± 0.3	36.9 ± 0.2	36.9 ± 0.2
End-exercise T_{es}	37.6 ± 0.3	37.6 ± 0.3	37.6 ± 0.4	37.5 ± 0.2
Pre-exercise T_{sk}	31.2 ± 0.5	31.3 ± 0.4	31.1 ± 0.3	31.2 ± 0.6
End-exercise T_{sk}	32.0 ± 0.3	31.9 ± 0.4	31.9 ± 0.5	31.9 ± 0.4

Values are means ± SD in degrees Celsius (°C). Asterisk (*) denotes a significant difference in the change from pre-exercise to end-exercise relative to 50°C water ingestion

LEGENDS TO FIGURES

Figure 1. Whole body sweat loss for ingested water of 1.5°C, 10°C, 37°C and 50°C. Asterisks (*) denote significant differences from 50°C.

Figure 2. Changes in evaporation from the skin (E_{sk}) and the evaporative requirements for heat balance (E_{req}) with 1.5°C, 10°C and 50°C fluid ingestion in comparison to 37°C fluid ingestion after 75-min at 50% of VO_{2max}. Asterisks (*) denote significant differences between E_{req} and E_{sk}.

Figure 3. The influence of ingested water temperature upon body heat storage estimated using the conventional 2-compartment thermometry model of "core" and "shell" and partitional calorimetry following 75-min of exercise at 50% VO_{2max} with the ingestion of 1.5°C, 10°C, 37°C and 50°C water. Asterisks (*) denote significant difference between indicated groups.

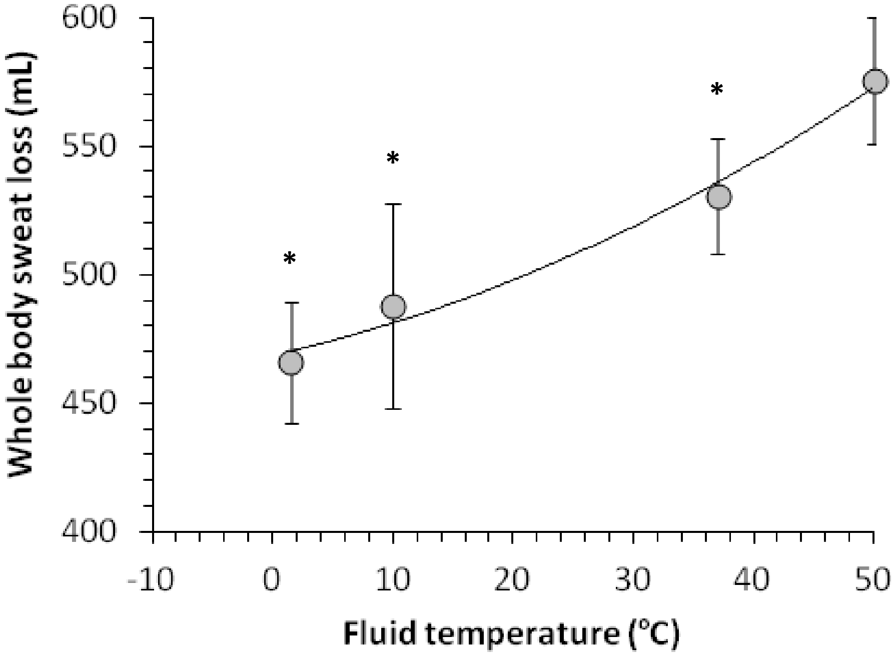
Whole body sweat loss (mL)
600
550
500
450
400
-10
0
10
20
30
40
50
Fluid temperature (°C)
*
*
*

Figure 1.

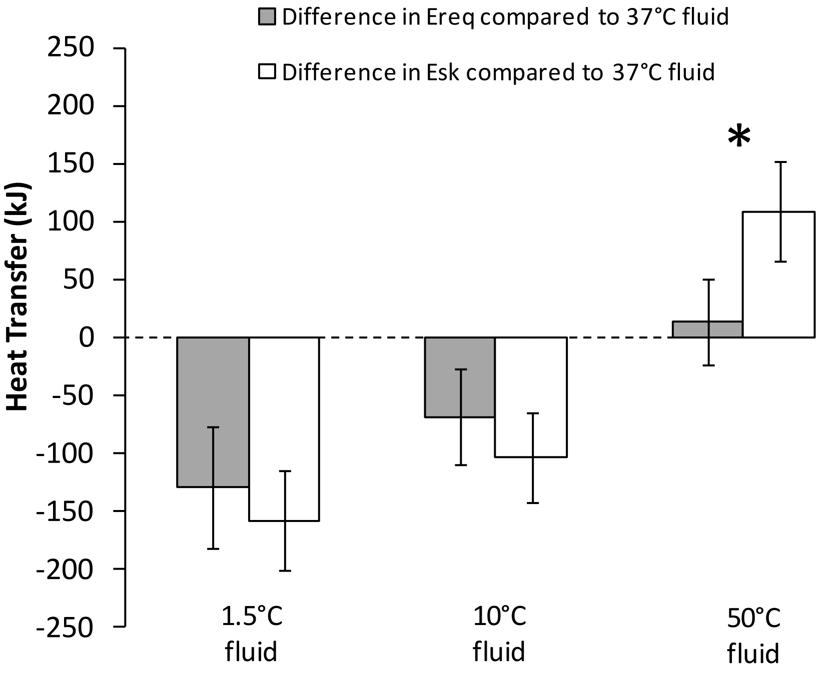
Difference in Ereq compared to 37°C fluid
Difference in Esk compared to 37°C fluid
Heat Transfer (kJ)
250
200
150
100
50
0
-50
-100
-150
-200
-250
*
1.5°C fluid
10°C fluid
50°C fluid

Figure 2.

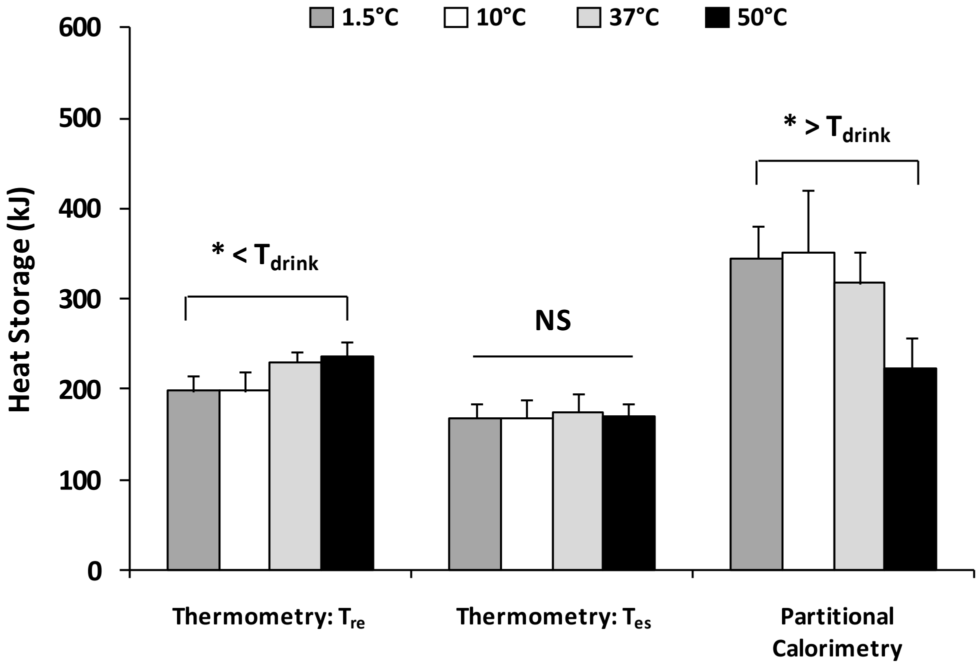
1.5°C
10°C
37°C
50°C
Heat Storage (kJ)
600
500
400
300
200
100
0
* < T_{drink}
NS
* > T_{drink}
Thermometry: T_{re}
Thermometry: T_{es}
Partitional Calorimetry

Figure 3.

Sudomotor and vasomotor activity following serial cooling and heating of thermosensors in the esophagus and stomach

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(Extended abstract submission to the Canadian Society for Exercise Physiology (CSEP)
2011 annual conference, Oct. 19 – 22nd, Quebec City, Canada)

Accepted for oral presentation

Introduction

During exercise, heat activated thermoreceptor activity provide the primary dose-dependent stimulus for sudomotor (sweating) and vasomotor (peripheral blood flow) activity, and subsequently enhanced heat dissipation. Whole body temperature reception is conventionally conceptualized as a proportional *shell* and *core* model, wherein the influence of changes in deep core temperature upon heat loss responses is roughly nine times greater than the influence of changes in shell temperature (Romanovsky 2007). Indeed, brain and deep tissue temperature has much less room to vary than temperature of the skin. However, the relative contribution of different groups of thermoreceptors, particularly those located in the stomach and esophagus, to changes in sudomotor and vasomotor activity, remain vague (Romanovsky 2007).

The purpose of the study was to characterize the transient localized sudomotor and vasomotor responses while ingesting water of different temperatures during moderate-intensity exercise. It was hypothesized that direct cooling/heating of thermosensors in the esophagus and stomach would elicit acute changes in sudomotor and vasomotor activity independently of deep body (core) and skin (shell) temperatures. The implications of these data will shed light on the framework of the thermoregulatory control system, specifically the distributed heat loss responses derived from independent contributions of thermoreceptors located in the esophagus and stomach. Furthermore, these findings can be extended to understanding heat balance requirements and fluid losses during cold drink ingestion (a recent recommendation for exercise in the heat (Burdon et al. 2010a)).

Methodology

Nine healthy males (aged 19 to 25 y) cycled on three occasions at 55% of their VO_{2peak} in a temperate environment (23.7±0.4°C and 20±7% RH) for 75-min. During each session, four 3.2 $mL•kg^{-1}$ boluses of water were ingested, the first at 5-min before exercise, and the following three after 15, 30 and 45-min of exercise. Within each experimental session the temperature of the ingested fluid was the same, but between sessions, beverages were fixed at either 1.5ºC, 37ºC or 50ºC. Rectal temperature (T_{re}), mean skin temperature (T_{sk}), local sweat rates (LSR) using ventilated capsules (on the upper back, forearm and forehead), skin blood flow using laser-Doppler velocimetry (SKBF) (upper back) and whole-body sweat loss (WBSL) were measured throughout each session.

Results

Skin blood flow during 50°C water ingestion was significantly greater than 1.5°C ($P=0.036$), but not 37°C ($P=0.796$). No significant decline in SKBF was found with 1.5°C compared to 37°C ($P=0.999$) (See Fig 1A). Mean LSR of the combined three sites was significantly greater with 50°C water ingestion compared to 1.5°C ($P=0.017$), but not 37°C ($P=0.120$). No significant decline in mean LSR was found with 1.5°C compared to 37°C ($P=0.604$) (See Fig. 1B). Whole-body sweat loss was significantly different between all three beverage temperatures (1.5°C: 622±68 mL, 37°C: 680±78 mL, 50°C: 726±84 mL) ($P<0.05$). The average mean body temperature (T_b) throughout exercise (i.e. ($0.9T_{re}+0.1T_{sk}$)) was not significantly different between any beverage temperature (1.5°C: 37.13±0.17°C, 37°C: 37.15±0.21°C, 50°C: 37.13±0.16°C) ($P>0.05$).

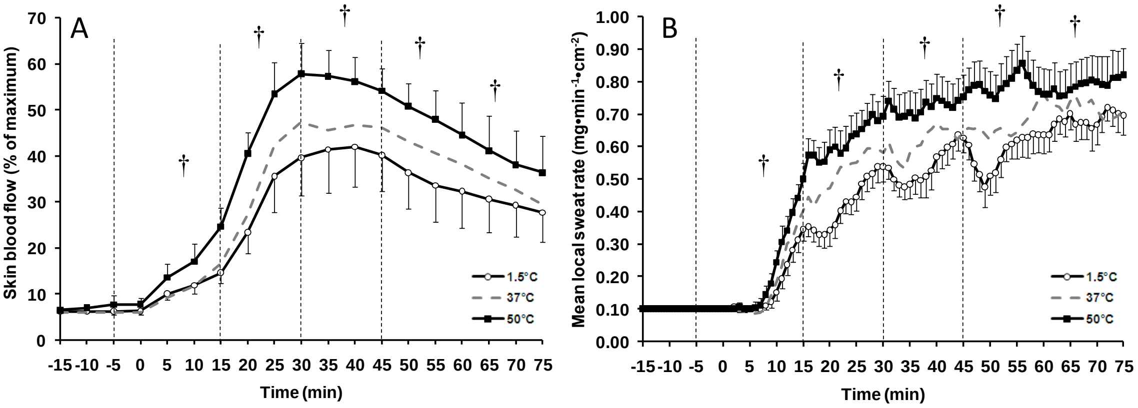

Figure 1. Upper back skin blood flow represented as a present of maximum (A) and mean local sweat rate of the forearm, upper back and forehead (B) throughout 75-min of exercise while ingesting water at 1.5°C *(open circles),* 37°C *(grey dashed line)* and 50°C *(closed squares)*. *Error bars* indicate standard error. *Dashed vertical lines* indicate the time of beverage ingestions. *Daggers* indicate a significant difference from 15-min averages between 1.5°C and 50°C

Discussion

The data show, for the first time, the transient sudomotor and vasomotor responses to local temperature changes in the esophagus and stomach with beverage ingestion (Fig. 1). These responses were independent of changes in T_b, suggesting that afferent signals arising from thermoreceptors in the esophagus and stomach were primarily responsible for modulating the observed changes in SKBF and LSR. The largest acute decline in sudomotor activity in response to cooling occurred after 45-min of exercise (Fig. 1), suggesting that the magnitude of responses are dependent upon the temperature gradient between the water and body (i.e. T_{re} was higher during the later stages of exercise), and thus rate of tissue cooling.

Whole-body sweat loss was 105 mL less in the 1.5°C compared to the 50°C condition. These data suggest that fluid guidelines should be adjusted according to the temperature of an ingested beverage. It remains to be seen whether the difference in evaporative heat loss was disproportionate to the difference in heat lost / gained through the ingestion of water at varying temperatures, however such findings most likely depend upon the environmental conditions and thus the degree of sweating efficiency (i.e. the fraction of evaporated sweat from the skin versus dripped sweat).

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PART THREE:

DISCUSSION AND CONCLUSION OF THE THESIS

4.0 Discussion

The purpose of the thesis was to examine thermoregulatory control and human heat balance while ingesting water of varying temperature immediately prior to, and during exercise. This was accomplished by measuring local sweat rate (on the forehead, upper back and forearm), whole body sweat loss, skin blood flow (on the upper back), core temperature (in the esophagus and rectum) and skin temperature, during 75-min of exercise while ingesting boluses of water of either; 1.5°C, 10°C, 37°C or 50°C, at 5-min before the onset of exercise, and 15, 30 and 45-min into exercise. A secondary objective was to compare thermometrically derived values of heat storage with those derived from partitional calorimetry.

The primary finding of the thesis demonstrates that, during exercise in an environment that permits full sweat evaporation, the ingestion of 50°C water, appears to in fact reduce whole body heat storage compared to ingestion of 1.5°C water. As hypothesized, this finding was attributed exclusively to disproportionate modulations in sweat output (and thus evaporative heat loss), whereby the differences in evaporative heat loss were greater than the differences in heat transfer with the ingested water. However, an important aspect of this finding lies within the confounding influence of the combination of environmental conditions and exercise intensity, and therefore sweating efficiency. It follows that this thesis does not necessarily refute the literature claiming a reduced heat strain with cold drink ingestion during exercise in hot/humid conditions, rather; it provides a provision by suggesting that the validity of this claim is dependent upon the degree of sweating efficiency. Specifically, with the aim of reducing heat storage, drinks should be consumed at temperatures of ~50°C when environments are such that permit full sweat evaporation, conversely, in conditions that do not permit full sweat evaporation, drinks should be cooled to temperatures of ≤10°C.

An explanation for the disproportionate modulations in sweat output is likely accredited to the unique characteristics of temperature sensing neurons in the stomach, esophagus and large intra-abdominal veins. The data show that locally warming or cooling the intra-abdominal tissue elicits a strong sudomotor response, which is the most pronounced following 45-min of exercise. This suggests that the magnitude of responses is dependent upon the temperature gradient between the water and body and thus rate of tissue cooling. The data further show, that the deep abdominal thermosensors exhibit a hierarchical contribution to thermal control, whereby local heating or cooling elicits a sudomotor response that is disproportionate to evaporative needs. Conversely, despite a main effect of drink temperature on skin blood flow, whereby 1.5°C water ingestion mitigated the averaged increase compared to 50°C water, acute changes (as observed with sudomotor activity) were not apparent in the skin blood flow measurements. Furthermore, the modest elevation in skin blood flow on the upper back following 50°C compared to 1.5°C water ingestion did not transfer to a greater mean skin temperature and therefore a greater rate of sensible heat loss.

A secondary finding of the thesis demonstrates that, upon comparison of the thermometric (weighted skin and rectal temperature) to calorimetric estimations of heat storage, discrepancies in the two methods become increasingly exacerbated as beverage temperature declines. Specifically, estimations of heat storage decrease with declining water temperature, but when heat storage is measured using partitional calorimetry the exact opposite is observed, whereby heat storage increases with decreasing water temperature. The reason for the large discrepancy in methods (greater than previously reported in existing literature) probably stems from local temperature changes of the tissue surrounding the rectal temperature measurement site. This subsequently explains why previous literature concluded that cold compared to warm drink ingestion reduces levels of heat storage, whereas we observed the opposite.

Lastly, this thesis demonstrates that changing beverage temperature from 50°C to 1.5°C elicits a reduction in sweat loss of ~20%. Furthermore, whole-body sweat loss declined exponentially with drink temperature, whereby differences in evaporative potential are apparently almost negligible with declining drink temperatures below 10°C. These findings should be incorporated into existing fluid replacement guidelines that currently do not consider the effects of beverage temperature. Indeed, differences in sweat loss, and thus hydration status would become increasingly relevant over an extended (i.e. > 2 hrs) period of time.

4.1 Conclusion and future research considerations

In summary, these findings provide an important amendment to the conventional thinking that cold fluid ingestion will, under all circumstances, reduce heat storage during physical activity. This study also supports evidence for influential thermosensor activity in the abdomen. The data can be applied to fluid replacement guidelines for occupational, military and sporting environments, and to the general public. Furthermore, these findings can be extended to sweat loss prediction equations, which currently do not include the considerations of beverage temperature.

Future research should focus upon extending exercise over a longer period of time (i.e. >2 hrs), to conclusively reveal whether the theoretically greater heat storage in the peripheral tissue would over time eradicate the local heat sink created by the ingested fluid, and therefore lead to measurably greater increases in the conventional measurements of core temperature (e.g. esophageal and rectal) with cold relative to hot fluid ingestion. Furthermore, extending the exercise session would test the notion that cold drinks may be able to minimize inefficient sweat losses in conditions that do not permit full sweat evaporation, and therefore help maintain hydration status. A similar analysis should also be performed during intermittent exercise, to test whether the temperature of beverages modifies post exercise heat loss.

PART FOUR:

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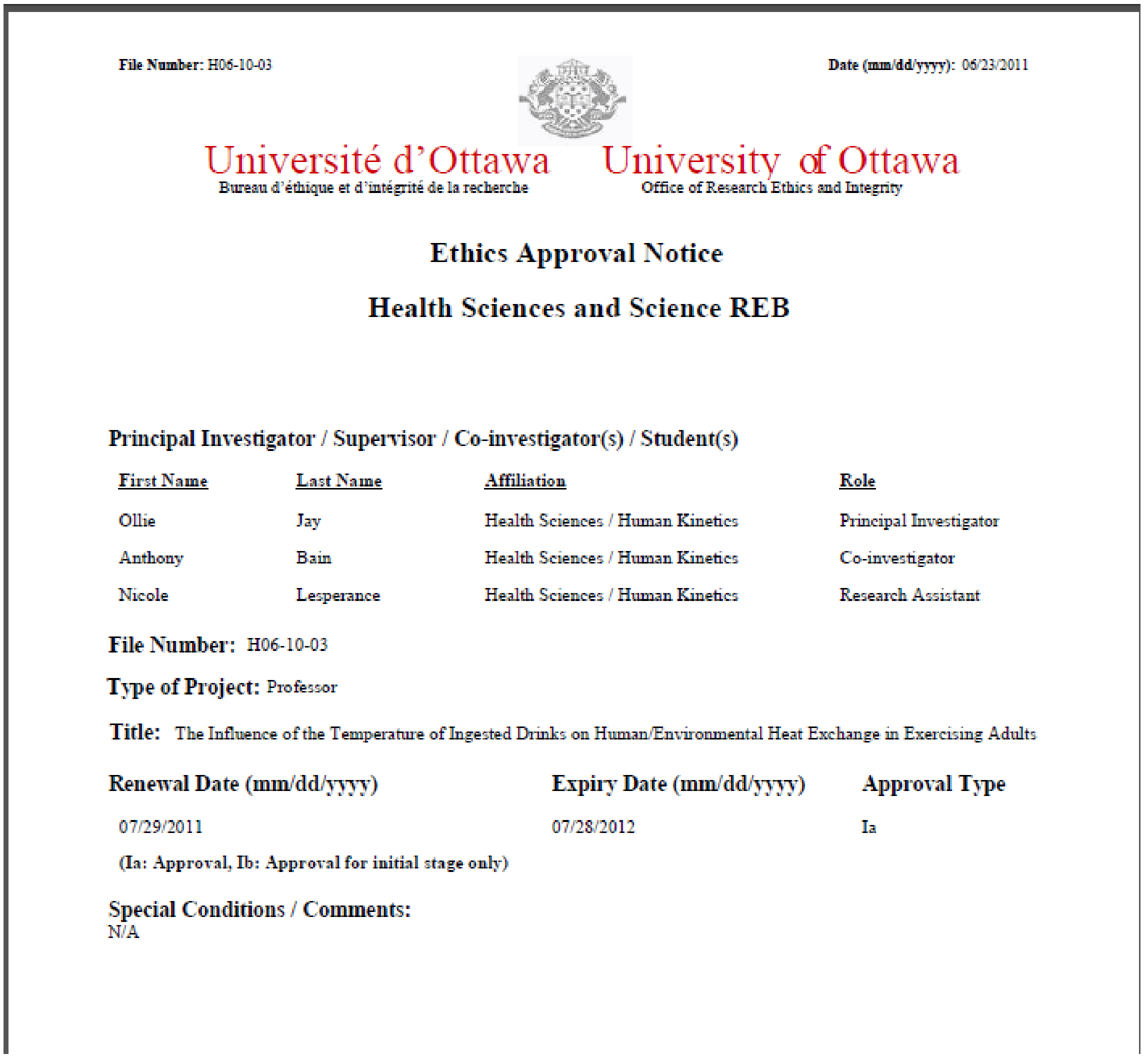
APPENDIX



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Date (mm/dd/yyyy): 06/23/2011

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Ethics Approval Notice

Health Sciences and Science REB

Principal Investigator / Supervisor / Co-investigator(s) / Student(s)

First Name	Last Name	Affiliation	Role
Ollie	Jay	Health Sciences / Human Kinetics	Principal Investigator
Anthony	Bain	Health Sciences / Human Kinetics	Co-investigator
Nicole	Lesperance	Health Sciences / Human Kinetics	Research Assistant

File Number: H06-10-03

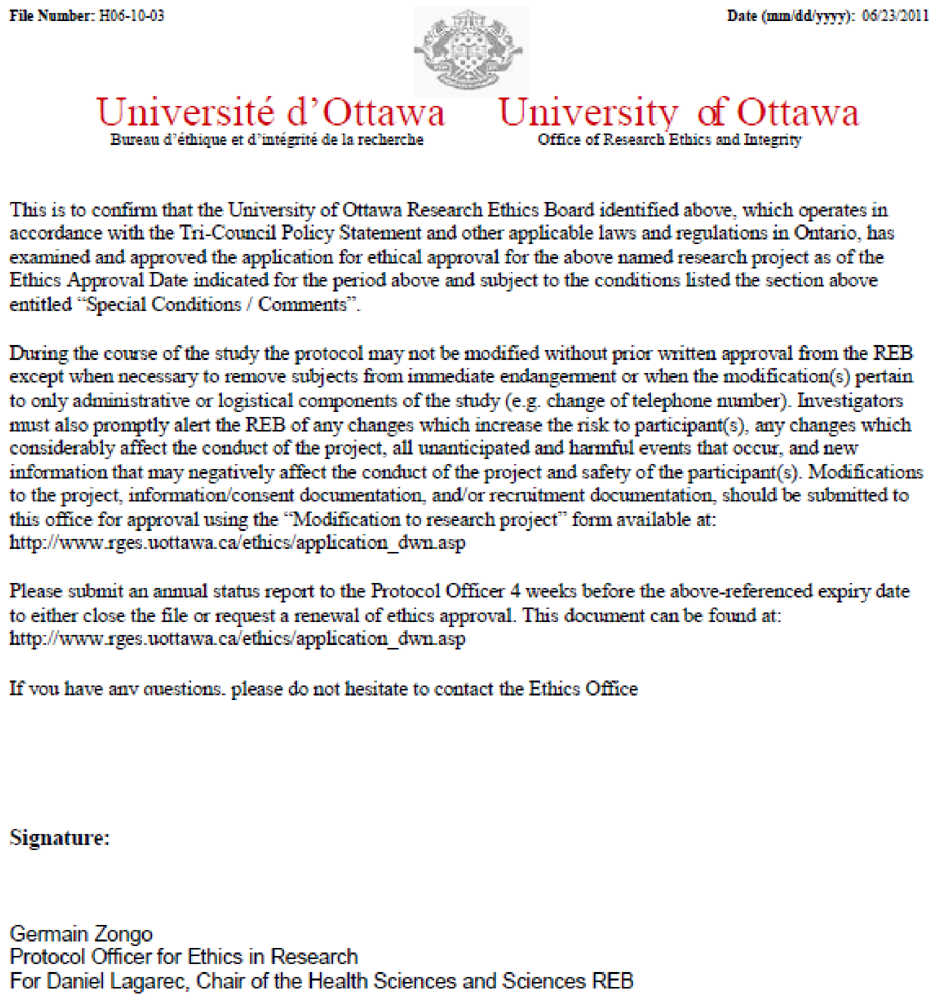
Type of Project: Professor

Title: The Influence of the Temperature of Ingested Drinks on Human/Environmental Heat Exchange in Exercising Adults

Renewal Date (mm/dd/yyyy)	Expiry Date (mm/dd/yyyy)	Approval Type
07/29/2011	07/28/2012	Ia

(Ia: Approval, Ib: Approval for initial stage only)

Special Conditions / Comments:
N/A



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Date (mm/dd/yyyy): 06/23/2011

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This is to confirm that the University of Ottawa Research Ethics Board identified above, which operates in accordance with the Tri-Council Policy Statement and other applicable laws and regulations in Ontario, has examined and approved the application for ethical approval for the above named research project as of the Ethics Approval Date indicated for the period above and subject to the conditions listed the section above entitled "Special Conditions / Comments".

During the course of the study the protocol may not be modified without prior written approval from the REB except when necessary to remove subjects from immediate endangerment or when the modification(s) pertain to only administrative or logistical components of the study (e.g. change of telephone number). Investigators must also promptly alert the REB of any changes which increase the risk to participant(s), any changes which considerably affect the conduct of the project, all unanticipated and harmful events that occur, and new information that may negatively affect the conduct of the project and safety of the participant(s). Modifications to the project, information/consent documentation, and/or recruitment documentation, should be submitted to this office for approval using the "Modification to research project" form available at: http://www.rges.uottawa.ca/ethics/application_dwn.asp

Please submit an annual status report to the Protocol Officer 4 weeks before the above-referenced expiry date to either close the file or request a renewal of ethics approval. This document can be found at: http://www.rges.uottawa.ca/ethics/application_dwn.asp

If you have any questions, please do not hesitate to contact the Ethics Office

Signature:

Germain Zongo
Protocol Officer for Ethics in Research
For Daniel Lagarec, Chair of the Health Sciences and Sciences REB

Background information and consent form

The Influence of the Temperature of Ingested Drinks on Human/Environmental Heat Exchange in Exercising Adults

Principal Investigator:
Dr. Ollie Jay,
Assistant Professor,

Background

During exercise in the heat, the body employs thermoregulatory (autonomic) responses that attenuate rises in body core temperature. To facilitate these responses, behavioral techniques have been proposed and employed which pronounce heat loss and reduce thermal strain during exercise. One of the most common of those techniques is the ingestion of cold fluids. Recently, Lee and Shirreffs (2007) investigated the effects of fluid temperature on thermoregulatory responses (i.e. sweating, elevations in skin temperature and subsequent elevations in body core temperature) during exercise. The authors noted that the ingestion of 1 L of a 10°C fluid relative to 1 L of a 50°C fluid produced lower rectal and skin temperatures and a correspondingly lower sweat output. However, upon further scrutiny of their data, rational heat balance analysis actually suggests a greater heat storage with ingestion of 10ºC relative to the 50°C fluid, by virtue of a lower sweat output and skin temperature. This suggests that core temperature with the ingestion of cold fluids when measured away from the stomach (the primary heat exchange location with cold fluids) may actually be greater; and the reduction in sweat output with cold fluid ingestion is disproportionate relative to the sweat rate required to attain heat balance. The question therefore remains, will the ingestion of a cold fluid reduce all body temperature elevations during exercise through means of conduction (direct cooling between two mediums), or will the local stomach cooling of the ingested fluid produce thermoregulatory shifts opposing the body's need to eliminate heat which exceed the conductive cooling capacity of the fluid?

Purpose

The purpose of the present study is to examine the effect of drink temperature upon thermoregulatory control. Changes in core body temperature, heat loss responses (i.e. sweating, skin temperature, skin blood flow) and sweating efficiency (i.e. the amount of sweat actually evaporated from the body relative to the amount of sweat produced) will be measured during 90-min of exercise while ingesting four 250 ml aliquots of water at either 4°C, 37°C, or 50°C. It is hypothesized that cold fluid ingestion will attenuate the body's natural defences against heat gain (i.e. sweating and skin temperature) and as such, the ingestion of a warmer fluid relative to a cold will provide a more effective means for reducing body temperature elevations during exercise.

Subject profile

To be a participant you must be a healthy (no history of respiratory, metabolic, cardiovascular, blood pressure disease, or of diabetes and not currently on any medication related to these conditions) male adult, aged between 18 and 39 years. If you agree to participate in this study, you will be required to participate in one preliminary session and three experimental sessions to be conducted on different days and separated by a minimum of 48 hours.

Preliminary session

Both the preliminary session and the experimental sessions will take place in at the Thermal Ergonomics Laboratory (E028), Lees Ave campus at the University of Ottawa. The time involvement will be approximately 45 min to 1 hour for the preliminary session. During the preliminary session, we will review all procedures with you. In addition, you will be introduced to all of the equipment and measuring devices that we will be using for the experimental sessions. We will give you the opportunity to read the Background and Informed consent document. If you agree to participate in the study, we will ask you to sign the informed consent below and complete a *Physical Activity Readiness Questionnaire (Par-Q)* and an *American Heart Association/American College of Sports Medicine Health/Fitness Facility Pre-participation Screening Questionnaire*. These questionnaires are standard questionnaires that have been developed to help us evaluate your readiness for exercise and are also used to assist us evaluate your general physical health and level of physical activity. Thereafter, we will complete some basic measurements including height, mass. Following these measures, you will be asked to perform a maximum oxygen consumption test on a cycle ergometer where upon you will be required to exercise until exhaustion. This will consist of pedalling at a cadence of 80 rpm while the resistance is increased by 20 watts every minute until you can no longer maintain the required cadence (8-12 min). We will also assess your body composition by using underwater weighing. You will be asked to wear a bathing suit, enter the tank and situate yourself on the hanging chair. You will be asked to immerse yourself completely under water for 5 seconds. Once the measurement is completed you will be given a few minutes to relax after which you will be asked to perform the same steps again. Five trials will be done in order to obtain accurate results.

Experimental session

The study will consist of 3 experimental sessions. Each session will last approximately 4 to 5 hours. Upon arrival at the laboratory you will change into athletic clothing (shorts and shoes). The experimental sessions will begin with an instrumentation period. Once all the equipment and probes (see description below) are in place and functioning, you will be weighed and then enter the a climatic chamber regulated at ~30ºC and ~30% relative humidity; and remain seated at rest for baseline data to be collected for 30 minutes.

At the end of this period, you will be asked to cycle on an upright cycle ergonomer (Velotron) inside the climatic chamber for 90 minutes under the same environmental conditions as the baseline measurements. The exercise intensity for each session will be ~55% of your VO_{2max}.

During exercise you will be asked to ingest within 90 seconds a 250 ml aliquot of water directly before the onset of exercise, and after 15, 30 and 45-min of exercise. Within each experimental session the temperature of the ingested fluid will be the same; but between sessions the temperature of the fluid will be different (either 4ºC, 37ºC or 50ºC).

Once exercise stops, you will remain in the chamber for a further 30-min of recovery and then exit for a final weight measurement. No further fluid ingestion will be allowed during this recovery period.

In preparation for the experimental trials, you will be asked to abstain from alcohol, caffeine and severe or prolonged physical activities for 24 hours prior to all sessions. It is highly recommended that you avoid eating a heavy meal for at least two hours before the trial.

The following instruments will be used to monitor and record your physiological response during the experimental trial:

Metabolic data: In order to measure metabolic heat production you will be equipped with a mouth piece and nose clip and will breath through the mouth piece for the duration of exercise (Vmax® Encore Metabolic Cart).

Esophageal probe**:** In order to monitor central body temperature, the researcher will insert a flexible oesophageal temperature probe (2 mm in diameter) will be inserted through one of your nostrils, during which time you will be asked to swallow sips of water. The tip of the probe, once fully inserted in your esophagus (swallowing tube), will rest at the level of the heart. There can be mild discomfort and mild gagging reflex from swallowing the probe. However, this sensation soon passes (5-10 seconds). **Please note that the probe will be inserted by Dr. Ollie Jay or Mr. Anthony Bain, who has been legally authorized to do so and who have been approved by the University of Ottawa Office of Risk Management to have the necessary skills and training to perform these insertions properly.*

Rectal probe**:** You will be asked to insert a flexible probe though the anus into the rectum (10-12 cm). Proper instruction will be given to you on the placement of the rectal probe. A marker is placed on the rectal probe using sterile surgical tape. The subject inserts the probe until the tape reaches the anal surface. The insertion of the rectal probe may cause some mild discomfort and minor irritation; however, this sensation soon passes. This probe provides the researcher with an indication of the amount of heat stored in your body. You should be aware that there is some minimal risk associated with the insertion of a rectal probe. With the insertion, there is a risk of perforation of the rectum, and may cause some discomfort and minor irritation. However, proper instruction will be given to you on the placement of the rectal probe to ensure your safety and comfort. You will be responsible for the insertion of this probe.

***Tympanic probe*:** The researcher will insert a probe into your ear canal. The probe will be pushed gently until it touches the tympanic membrane. At this point, you will sense a slight discomfort and the probe will then be retracted slightly. The probe will be secured in its position by packing the ear with cotton balls held in place with surgical tape. The auditory canal temperature will be used as an index of brain and core temperature. **Please note that the probe will be inserted by Dr. Ollie Jay or Mr. Anthony Bain, who has been legally authorized to do so and who have been approved by the University of Ottawa Office of Risk Management to have the necessary skills and training to perform these insertions properly.*

***Skin temperature probes*:** Twelve skin probes will be taped to the skin surface (on the forehead, shoulder, chest, upper right back, abdomen, lower back, bicep, back of the hand, front of the thigh, back of the thigh, back of the calf and front of the calf) with hypoallergenic tape. These probes give an indication of skin temperature and heat loss from the skin surface. Some hair may need to be shaved (by the use of disposable razors) in order to secure the probes adequately to the skin surface. Some discomfort may be experienced upon removing the tape.

***Sweat capsule*:** Up to three small plastic capsules will be taped to the back of the shoulder (upper back), chest, forearm and/or forehead. This capsule picks up humidity from the skin and provides a measurement of local sweat rate.

***Technical absorbents*:** Local sweat rate will be measured using ~ 36 cm^2 patches of technical absorbent material (Technical Absorbents Ltd, Grimsby, UK) placed on the forehead and forearm of each individual for the final 5-min of exercise. The difference between pre and post application weights of the absorbent pads will be measured to indicate local sweat rate in $mg/cm^2/min$.

***Whole-body sweat rate*:** You will be weighed on a platform scale (Combics 2, Sartorius, Canada) immediately before the start of exercise and immediately after exercise has stopped.

Sweating efficiency: An oil filled pan will be placed underneath the bike. This pan will collect any sweat which not been evaporated into the air and that has dripped off the body and will be weighed before and after exercise.

***Skin blood flow*:** A flexible laser probe will measure skin blood flow non-invasively at the upper back. This measuring device does not result in any discomfort or residual medical effects.

***Heart rate*:** Heart rate will be monitored by a strap placed around the chest (Polar Vantage heart rate monitor).

Risks and discomforts

In the event of a health related emergency, our research staff is trained in CPR and we have emergency phones located in the laboratory for immediate contact with University emergency response (University Protection Office).

Physical activity: There are some minor physical risks associated with any form of exercise. There is essentially no major risk for young, healthy, active people while performing the submaximal exercises. Some effects of maximal exercise testing are nausea, dizziness, fainting, abnormal blood pressure, chest pain and leg cramps. For the maximal and experimental exercise sessions, the 'Guidelines for Graded Exercise Testing and Exercise Prescription' (by the American College of Sports Medicine) indicate that for men under 40 years of age, with no symptoms or risk factors for cardiovascular disease, the presence of a physician during the test is not required. The incidence of cardiac arrest during maximal exercise tests is 1 in 10000 tests. Participants may stop at any time during these tests. All tests will be conducted under standardized conditions for human exercise experiments as laid out by the Canadian Society for Exercise Physiology and the American College of Sports Medicine.

Temperature probes: Perforation of the esophagus or aural or nasal cavities, as well as the rectum can occur during insertion of the esophageal and rectal probes (potentially causing inflammation and infection). Perforation of the esophagus or oral or nasal cavities, as well as the rectum is very rare and no such incident has ever occurred in a laboratory the principal investigator has worked in. The risk of transmission of infectious disease is negligible as each subject has his own sterile probes that will be disposed of once all tests have been completed.

Elevation of core body temperature: There are certain risks that accompany a mark elevation in core temperature associated with exercise-induced dehydration. These include: headache, extreme weakness, dizziness, nausea, hyperventilation, hypotension, confusion, diarrhoea, vomiting and loss of consciousness. During all experimental protocols, you will be under close examination by the research assistant. Further, core body temperatures will be monitored continuously during the experimental trials, and exercise will be terminated if you reach 39.5°C esophageal temperature. Additionally, during the experimental protocols, a circulated cold water bath will be prepared and available if needed to rapidly cool you. If you become light headed or dizzy, exercise will be terminated and a mat will be readily available in an adjacent room maintained at a comfortable ambient temperature where you will be laid in the supine position, cooled with cold towels, and given a commercially available sports drink (Gatorade) in order to rehydrate and maintain blood sugar.

Headaches associated with ingesting cold water: You may experience a mild headache from the ingestion of the cold water (sphenopalatine ganglioneuralgia) which should pass shortly after ingestion (3 to 5-min).

An emergency first aid kit is readily available if needed for all laboratory session. A qualified person (Mr. Anthony Bain) will be on hand to administer treatment if required.

Benefits of Participating

While participating in this study you will gain knowledge of your aerobic fitness and body composition. You will also learn about the research process and the knowledge acquired during the experimental sessions may be shared upon request.

Anonymity and Confidentiality

All raw data will be stored using alphanumeric coding system as such, no one will be able to identify you as your name will not appear on these files. Data will be kept in Montpetit Hall, Room 372, in locked file cabinets and only the researchers directly involved in this study will have access to your data.

No records bearing your name will leave the institution. You are encouraged to request and discuss the results of the experimental trials at any time. The results of the preliminary session (aerobic fitness and body composition) will be available to you upon completion of the study.

The data collected in this study will be published in scientific journals. The data will kept for a period of 5 years post-publication and will subsequently be destroyed by the physical resources service of the University of Ottawa.

For the entire duration of the study, it is fully understood that you may refuse to participate or withdraw from the study at any time, without question. You may also withdraw from participating when you are in the thermal chamber or at any point during either the exercise or recovery period.

INFORMED CONSENT OF PARTICIPANT

Research involving human subject require written consent of the participants.

I, ___________________________, hereby volunteer to participate as a subject in the study entitled **"The Influence of the Temperature of Ingested Drinks on Human/Environmental Heat Exchange in Exercising Adults"**. I have read the information presented in the above background information and I had the opportunity to ask questions to the investigators. I understand that my participation in this study, or indeed any research, may involve risks that are currently unforeseen.

I recognize that there will be no direct benefit to me from my participation in this study (besides receiving an aerobic fitness and body composition evaluation).

(omitted personal information)

I have been given a copy of this Background Letter and Consent Form for me to keep.

Signature of participant: _________________________ Date: __________________

Signature of Researcher: _________________________ Date: __________________