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The Effect of Physical Training on Body Heat Regulation

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THE EFFECT OF PHYSICAL TRAINING ON BODY HEAT REGULATION

By

JILL STAPLETON
B.Sc., Campbell University, 2006

THESIS

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ABSTRACT

Purpose: We evaluated the effects of an eight-week aerobic exercise training program in previously sedentary individuals on whole-body heat balance. Whole-body evaporative ($\dot{H}_E$) and dry ($\dot{H}_D$) heat loss as well as changes in body heat content (ΔH_b) were measured using simultaneous direct whole-body and indirect calorimetry. It was hypothesized that following the 8-week exercise training program, a more rapid increase in the rate of whole-body heat loss would occur during exercise resulting in a decrease in the change of body heat content by the end of exercise. It was further hypothesized that the rate of decay of whole-body heat loss during recovery would be greater following the exercise training program.

Methods: Ten previously sedentary young adults (7 males, 3 females) underwent an 8-week exercise program. Participants exercised at a university based facilities 4-5 times per week, 30-90 minutes per session, supervised by personnel. Prior to, and after the 8-week training program, subjects underwent an incremental treadmill test to measure their maximal aerobic capacity ($\dot{V}O_{2max}$). On a subsequent day, they performed 60-90 minutes of cycling at a constant rate of heat production (~450 W) followed by 60 minutes of recovery, in a calorimeter at 30°C and 15% relative humidity. Core temperature [esophageal; (T_{es}), rectal (T_{re}) and aural canal (T_{au})], mean skin temperature, skin blood flow (SkBF), local sweat rate (LSR), mean arterial pressure (MAP), and heart rate (HR) were measure at baseline and at 2 min, 5 min, 8 min, 12 min, 15 min, 30 min, 45 min, 60 min, and 90 min intervals for the exercise and post-exercise recovery periods.

Results: No significant difference in the rate of total heat loss ($\dot{H}_L = \dot{H}_E + \dot{H}_D$) was observed during exercise. As a result, the average ΔH_b was similar for the pre- (+441±89 kJ) and post-training (+430±118 kJ) 60-min exercise bout ($p=0.385$). Although the absolute changes in T_{es} ($p=0.060$), T_{re} ($p \leq 0.05$) and T_{au} ($p \leq 0.05$) were lower at rest post-training, no differences in the relative change from baseline was measured during exercise. Local sweat rate and SkBF were elevated during exercise however the relative changes from baseline were similar pre- to post-training. A 12% increase in $\dot{V}O_{2max}$ was measured after the 8-week training program ($p \leq 0.05$). This was paralleled by a decrease in heart rate throughout exercise ($p=0.004$).

Conclusion: Although physical training resulted in improvements in cardiorespiratory function as evidenced by increases in $\dot{V}O_{2max}$ and reduced HR response during exercise, these adaptations did not result in an improvement in the capacity for heat dissipation during exercise.

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GLOSSARY

$\dot{M}$ - Rate of metabolic work load (W)

$\dot{W}$ - Rate of external workload (W)

$(\dot{M} - \dot{W})$ – Rate of metabolic heat production (W)

$\dot{H}_L$ – Rate of total heat loss (W)

$\dot{H}_E$ – Rate of evaporative heat loss (W)

$\dot{H}_D$ – Rate of dry heat loss (W)

$\dot{S}$ - Rate of heat storage (W)

ΔH_b – Change in body heat content (kJ)

$\dot{V}O_{2max}$ – Rate of maximal oxygen consumption (mL/kg/min)

T_{es} – Esophageal temperature (°C)

T_{re} - Rectal temperature (°C)

T_{au} – Aural canal temperature (°C)

T_{sk} – Mean skin temperature (°C)

LSR – Local sweat rate ($mg/cm^2 \cdot min$)

SkBF – Skin blood flow

CVC - Cutaneous vascular conductance

MAP – Mean arterial pressure (mmHg)

HR – Heart rate (beats/min)

PART ONE:

EMPIRICAL AND THEORETICAL CONSIDERATIONS

Chapter 1

INTRODUCTION

1.0 Heat Balance

The capacity of the body to regulate core temperature around 37°C is crucial in overall human functions. Human thermoregulation is the process by which the amount of heat being produced is equilibrated with the amount of heat being lost. Heat production, thermogenesis, in the body is the resultant of metabolic processes, both voluntary and involuntary in nature. Involuntary mechanisms include muscle contraction, nervous activity processes and nonshivering thermogenesis which are often influenced by hormones such as thyroxine and epinephrine (Makjavic & Eiken, 2006). Voluntary mechanisms include muscular activity during exercise and physical work. Exercise requires a rapid increase in the generation of adenosine triphosphate (ATP), which occurs by a series of metabolic processes to provide energy for the working muscles. However, these processes are rather inefficient. Depending upon the movement and fuel selection, muscle mechanical inefficiency can be approximately 80% which causes the energy produced to be transferred as heat (Hadad *et al.*, 2004). Therefore, heat produced is directly proportional to exercise intensity and duration. It is for this reason that the net heat stored is dependent on the capacity of the body to dissipate.

Energy transfer in the body occurs internally by means of conduction and externally by means of radiation, conduction and evaporation, which are aided by convection (Nielsen, 1998). Radiation is energy in the form of waves. Energy is transferred from a higher energy state to a lower energy state. Conduction is when two objects are placed together and a transfer of energy occurs. Net energy transfer will always be in the direction of

high temperature to low temperature. An example would be blood flowing past internal organs. There will be a transfer of energy in the form of heat from the warm organs to the cooler blood. Then after, the warm blood will flow by the cooler surface of the skin where a subsequent transfer of energy in the form of heat will take place. Then, the heat will then be released to the environment aided by convection. Evaporation is another means of energy transfer and is the most important method of heat loss during exercise. Evaporation is dependent upon water vapour pressure. Relative humidity of the ambient environment, convective flow around the body, and the amount of skin area exposed to the surrounding environment will also determine the amount of sweat being evaporated from the surface of the skin (Sawka et al., 2000). With increasing air temperature, humidity and (or) surface insulation, the ability for heat dissipation, and therefore whole-body temperature regulation, is compromised thereby placing the individual at greater risk for heat illness.

1.1 Physical Training

Regular bouts of exercise elicit many physiological responses. Some of the responses include an increase in maximal aerobic power, decreased heart rate, and decreased skin and core temperatures (Daussin *et al.,* 2007; Kubukeli *et al.*, 2002). Also, chronic exercise will decrease the core temperature at which the onset of local skin blood flow and sweating will occur (Johnson, 1998). Chronic exercise training has been shown to induce changes in thermoregulatory function. Such adaptations include: 1) an increase in local skin blood flow; and, 2) an increase in local sweat rate (Nielsen *et al.,* 1998). However, it remains unclear if these responses are due to central- or peripheral-mediated influences.

Studies suggest that the sensitivity of the thermal reflex for skin vasodilation is enhanced (i.e., greater increase in skin blood flow per unit rise in core temperature) after regular bouts of aerobic exercise training (Roberts *et al.*, 1977). Further, the increase in blood volume associated with regular exercise training, results in an increase in the relative distribution of blood flow to the skin (Powers & Howley, 2004). This increase enhances the capacity for heat dissipation without compromising muscle blood flow and therefore delivery of oxygen and nutrients needed to sustain muscle contraction during exercise.

Studies suggest that the reduction in the onset threshold at the initiation of sweating with exercise training is the result of a centrally mediated adaptation while others report that changes in sudomotor activity may be peripherally mediated (Fortney & Vroman, 1985; Nadel, 1979) likely due to changes in local responsiveness. Whether centrally or peripherally mediated, an increase in sweating results in changes in heat dissipation. Since one's ability to evaporate sweat in a given environment is a major determinant of the level of heat stress risk and injury, a training-induced increase in sweating may have a profound effect in reducing this risk.

1.2 Statement of Problem

Traditionally, research on the effects of physical training on thermoregulatory control during heat exposure has primarily been based on the evaluation of local heat loss responses and core temperatures. However, recent reports suggest that local heat loss responses do not illustrate how much whole-body heat transfer is altered. Also, core and skin temperature measurements do not accurately represent the magnitude of residual

body heat storage. This is due to the fact that when estimating the change in body heat content through thermometry, the specific heat capacity for all the different tissues (ie, muscle, fat, organs and skin) are not accounted for. As such, the estimation of whole-body heat storage using these skin and core temperature changes during exercise and (or) exposure to extreme ambient conditions is inaccurate and may subsequently lead to erroneous conclusions regarding the effects of physical training on thermoregulatory control mechanisms and whole-body heat balance.

1.3 Purpose

It is generally accepted that the only way to accurately estimate the rates of whole-body evaporative and dry heat loss as well as ΔH_b in humans is by performing simultaneous minute-by-minute measurements of the individual heat balance components by whole-body calorimetry. As such, the rate of metabolic heat production is determined based on the stoichiometric relationship of the products and reactants of oxidative metabolism as well as respiratory gas analysis. The rate of net heat loss from the body is determined from the direct measurement of the rates of sensible (radiation and conduction, aided by convection) and insensible (evaporation from the skin and pulmonary surface) heat loss using a direct calorimeter. Therefore, the purpose of the following study was to determine the effects of an 8-week exercise-training program on the rates of whole-body heat loss as well as the ΔH_b during and following a moderate intensity exercise under constant ambient conditions of negative net heat exchange.

1.4 Hypotheses:

It was hypothesized that following the 8-week exercise training, a more rapid increase in the rate of whole-body heat loss would occur during exercise resulting in a decrease in the change of body heat content by the end of exercise. It was further hypothesized that the rate of decay of whole-body heat loss during recovery would be greater relative to the rate of whole-body heat loss prior to the start of training, due to the decreased change in body heat content, resulting ultimately in a decrease in the residual change of body heat content by the end of recovery.

1.5 Rationale

To date, there have been no studies quantifying the adaptations of an eight-week physical training program, in previously sedentary individuals, on its effect on whole body heat balance. By studying the physiological responses elicited by the human body to promote the dissipation of heat from the body and directly measuring the subsequent heat exchange to the environment via whole-body direct calorimetry before and following training, we will acquire valuable new insights on the influence of exercise-induced adaptations on thermoregulatory function from a whole-body perspective to the development of thermal stress in man. Also, we will be able to determine if the local responses reflect changes to whole-body heat balance.

1.6 Delimitations and Limitations

Subjects recruited to participate in this study were sedentary as determined by being physically inactive less than two times per week for the previous six months. The starting

$\dot{V}O_{2max}$ was < 55ml/kg/min for males and < 50ml/kg/min for females. They were placed on an eight-week exercise training program in order to bring out an increase in $\dot{V}O_{2max}$ by 10-15%. Subjects were between the ages of 18-28, non-smoking, non-pregnant, and otherwise healthy. Therefore, results cannot apply to children and the elderly.

Blood flow was measured only on the forearm and therefore conclusions cannot be made about muscle blood flow. Local sweat rate was only taken from the upper back and therefore conclusions cannot be applied to whole-body sweating. This experiment will not study the possible menstrual cycle effects within women. All female subjects were in their mid-follicular phase of the menstrual cycle during the two experimental trials.

Chapter 2

REVIEW OF LITERATURE

2.0 Calorimetry

The determination of the change in body heat content is fundamental to assessing the exposure of the human body to any heat stress that may result in thermal imbalance. In theory, the measurement of body heat exchange using simultaneous measures of total heat production and loss is one method whereby ΔH_b can be determined. Therefore by definition the difference between metabolic heat production using the stoichiometric relationship of the products and reactants of oxidative metabolism (indirect calorimetry) and the total heat lost from the body (direct calorimetry) can be used to measure the rate of change in body heat storage and ΔH_b. Due to the limited accessibility of direct calorimeters, thermometric models are often employed to determine ΔH_b.

2.1 Exercise Training

Many physiological adaptations occur during exercise training. These include an increase in: aerobic capacity ($\dot{V}O_{2max}$), onset threshold for blood lactate accumulation, myoglobin content, hemoglobin content, size and number of mitochondria, oxidative and glycolytic enzymatic activity, capillary density, and oxidative capacity of fast-twitch fibres (Dussain *et al.,* 2008; Hawley, 2002; Powers & Howley, 2004; Young *et al.,* 1993; Ziemba, 2003). Some reports suggest that such improvements in cardiorespiratory fitness are associated with an increase in the ability for heat dissipation (Armstrong & Pandolf, 1988; Gisolfi, 1973; Gisolfi & Robinson, 1969). For example, researchers concluded that prolonged training is associated with improvements in aerobic capacity and may be linked with

significant improvements in exercise-heat tolerance independent of hydration or acclimation status (Cheung & McLellan, 1998). Some of the major thermoregulatory adaptations that are thought to occur with regular aerobic training and which elicit a reduction in core temperature during exercise in the heat include: an increased skin blood flow, increased sweat rate, and a lower heart rate both at maximal intensity and during rest (Cheung *et al.*, 2000; Watt *et al.*, 2000).

A number of studies have evaluated the effects of prolonged bed rest to simulate de-training on thermoregulatory responses (Ertl *et al., 2000;* Lee *et al.,* 2002). These studies show impairments in the heat loss responses of skin blood flow and sweat rate measured during exercise performed in the heat. This response is thought to be mediated by central mechanisms influencing the thermoregulatory efferent output (Lee *et al.*, 2002). These findings highlight the important influences that repeated bouts of exercise training may have on thermoregulatory function, more specifically the capacity for heat loss, and therefore the regulation of core temperature.

It is well known that highly trained individuals have a lower core temperature at rest and during exercise at a given work rate. Shvartz *et al.* (1974) trained five young men for 12 days at 85% of their pre-determined $\dot{V}O_{2max}$ in 21.5°C. These subjects showed a decreased heart rate as well as a reduction in rectal temperature, skin temperature, sweat rate, and conductance at rest and during exercise. Lower rectal temperatures during exercise resulted from the decrease in metabolism and in resting rectal temperature (Shvartz *et al.,* 1974). The subjects increased their $\dot{V}O_{2max}$ by 16% which meant that on

the final day of exercise they we no longer working at the same metabolic rate as the first day. Since the thermal load lower on the last day compared to the first day of training, the responses from the thermoregulatory system will be decreased.

In recent work, Shields *et al.* (2004) evaluated the effects of a 12-week moderate intensity physical training program (n=8) on the ability to dissipate heat in a warm environment in previously sedentary individuals. Responses were compared with a no-exercise control group (n=4). Prior to and following the training program all twelve subjects underwent a heat stress test which involved exercising for 45 minutes at 45% of their pre-determined $\dot{V}O_{2max}$ in a temperature controlled chamber maintained at 32°C and 32% relative humidity. Measurements included heart rate, rate of oxygen consumption, esophageal temperature, sweat threshold, average skin temperature, and skin blood flow. After the twelve-week training program, subjects underwent a second heat stress test. The trained group experienced an average increase in $\dot{V}O_{2max}$ of 13% which was paralleled by a reduction in submaximal heart rate (as determined for the same absolute work rate), and an increase in exercise cycle time. No significant difference in either esophageal temperature response from baseline or in the onset temperature at which sweating began was observed between groups or with the training intervention. While they measured an increase in the level of skin blood flow, it is unlikely that this had any significant influence on whole-body heat loss as evidence by a similar esophageal temperature response.

Given that the exercise sessions were not controlled in terms of the volume and (or) intensity and type of exercise (continuous versus discontinuous) performed, it is possible that this may have confounded their results. For example, a higher intensity exercise performed for a prolonged period would have induced a greater thermal load. When performed over repeated sessions this could elicit a greater thermal adaptation than as compared to performing lower intensity exercise for shorter periods (Avellini *et al.*, 1982). Gisolfi (1973) suggested that improvements in thermoregulatory function are likely manifested when an exercise-training stimulus is sufficient to elicit improvements in $\dot{V}O_{2max}$ by at least fifteen percent. These improvements in $\dot{V}O_{2max}$ may be associated with high intensity training and therefore greater increases in core temperature. This will elicit the thermoregulatory system to respond more often and chronic adaptations will occur to ensure the individual remain in heat balance during exercise.

2.2 Skin Blood Flow

An increase in blood flow to the skin during exercise is important to maintaining a thermal homeostasis. Okazaki *et al.* (2002) assessed the effects of aerobic training and resistance training on skin vasodilation and sweating on older adults. They observed a decrease in the temperature threshold for the onset of skin vasodilation and sweating suggesting an increase in the capacity for heat dissipation. However, it has been suggested that increases in blood volume (primarily mediated by an expansion in plasma volume) induced by aerobic training is an important stimuli in enhancing skin blood flow (Takeno *et al.*, 2001). Their findings suggest an exercise-induced increase in blood volume may be an important determinant in the regulation of core temperature as it relates to concurrent changes observed in skin blood flow.

Roberts *et al.* (1977) examined the concurrent effects of physical training and acclimation on thermoregulatory responses. Ten subjects underwent ten days of moderate intensity (75% $\dot{V}O_{2max}$) exercise performed in ambient conditions of 25°C. This period was immediately followed by 10 days of light intensity exercise (50% $\dot{V}O_{2max}$) performed in hot conditions (air temperature of 35°C). Exercise session lasted one hour each day. Prior to, at mid-point, and at the end of 20-day period, subjects underwent an exercise test (12-15min exercise @ 60-70% $\dot{V}O_{2max}$ performed in an ambient temperature of 25°C) to evaluate thermoregulatory responses. There was a decrease in esophageal temperature at which the initiation of skin vasodilation and sweating observed. A further downward shift in the onset thresholds was observed following the combined exercise and heat exposure phase of the study (referred to in this study as the acclimation phase) (Roberts *et al.,* 1977). These findings demonstrate that while exercise training elicits improvement in thermoregulatory function the response is also dependent on the thermal load. In light of the fact that subjects performed exercise during the 10-day period of the 'acclimation' phase, it is difficult to ascertain if the response was indeed due the acclimation alone, exercise training or a combination of both.

2.3 Sweat Rate

As noted above, sweating activity is influenced by physical training. However, there remains controversy as to whether an increase in sweat rate is mainly due to an increased thermosensitivity mediated primarily by peripheral input or the result of a centrally mediated decrease in the onset threshold for sweating (Johnson, 1998). Frye & Kamon

(1981) showed acclimation to dry heat [dry-bulb temperature (Tdb)/wet-bulb temperature (Twb) = 48/25 °C] in individuals with similar cardiorespiratory capacities improved sweating sensitivity, but did not affect the core temperature at which onset of sweating occurred in trained individuals. Nadel *et al.* (1974) showed that the increase in sweating following physical training is achieved via a peripheral mechanism resulting from increased cholinergic sensitivity (Nadel *et al.,* 1974). Some studies suggest that exercise induced adaptations of the eccrine gland function (i.e., rate of sweat output, volume of sweat output, etc.) itself may be a factor leading to enhanced sweating post-training (Buono & Sjohlm, 1998).

Henane *et al.* (1977) conducted a study to examine the effects of a 3-month exercise training program on sweating activity in previously sedentary individuals. Responses were compared with highly trained individuals who were engaged in a regular self-monitored training routine. A 28% increase in sweat output was measured in the previously sedentary individuals albeit no increase in the onset threshold of sweating was observed. However, when compared to the highly trained athletes, the absolute increase in sweat output remained lower than that measured for the highly trained individuals. These findings demonstrate that while physical training provides an important stimulus to increase thermolytic activity as measured by an increase in sweat rate, chronic adaptations with long-term training may elicit additional improvements in thermoregulatory function.

As discussed above, Roberts *et al.* (1977) showed improvements in sweating response with one-hour exercise sessions of moderate intensity exercise performed over 10-consecutive days. Their findings suggest that short-duration exercise training is sufficient to elicit modest improvements in the capacity for heat loss. The increase in sweating activity was also dependent on the thermal load as demonstrated by the greater decrease in the onset threshold for sweating observed when exercise training was performed in the heat (Roberts *et al.,* 1977).

Shvartz *et al.* (1973) examined three different acclimatization protocols which involved performing exercise while the subjects were exposed to: 1) dry heat (air temperature of 50°C and 28°C wet bulb), 2) wet heat (air temperature of 37°C and 20% RH, and 3) temperature in non heat stress conditions (ambient temperature of 23°C and 50% RH). The subjects in each group exercised between 60-90 min for 6 consecutive days. The two heat exposure groups performed moderate intensity exercise, whereas the non-heat stress group performed intense exercise. All groups showed an increase in sweat rate over the 6 day training period. However, the greatest increase was seen in the hot dry group and the smallest increase was seen in the group which performed their exercise in a non heat stress condition. Both the study by Shvartz *et al.* (1973) and Roberts *et al.* (1977) highlights the possible confounding influences that physical training and (or) acclimation may have on thermoregulatory function. Although physical training consistently has been shown to improve sweating activity, it remains unclear how improvements in sweating (or skin blood flow) are truly related to physical training per se. Some researchers consider that physical training itself is in effect a form of acclimation.

Styrdom *et al.* (1966) suggested that exercise training will result in partial acclimation. However, they noted that exercise training alone cannot elicit the improvements in thermoregulatory functions typically observed with heat acclimation. Studies testing the effects of sweat rate due to physical fitness have mainly compared trained to untrained individuals and have them work at the same percentage of their $\dot{V}O_{2max}$. However, each participant will then be working at a different thermal load and therefore will have different responses in sweating due to the differences in thermal input rather than their level of fitness.

2.5 Heat Tolerance

Cheung & McLellan (1998) studied the separate and combined effects of cardiorespiratory fitness, short term acclimation and hydration status on tolerance during uncompensable heat stress. Men who were moderately trained (<50 mL/kg/min) and highly trained (>55 mL/kg/min) were tested before and after 2 weeks of daily heat acclimation (1-h treadmill exercise at 40°C, 30% relative humidity, while wearing a nuclear, biological, and chemical protective clothing). Heat acclimation increased sweat rate and decreased skin and rectal temperatures in the highly fit subjects but had no effect on tolerance time. Moderately fit subjects increased sweat rate but did not alter heart rate, rectal temperature or tolerance time. It was concluded that exercise-heat tolerance during uncompensable heat-stress environment is not influenced by short-term heat acclimation but is significantly improved by long-term physical training.

McLellan (2001) noted that when protective clothing is worn, evaporative heat loss is restricted. Hence, it is not valid to assume that the higher sweat rates associated with

improvements in aerobic fitness will increase heat tolerance. An initial study compared thermoregulatory and cardiovascular responses to both compensable and uncompensable heat stress before and after 8 weeks of endurance training in previously sedentary males. Despite a 15% improvement in $\dot{V}O_{2peak}$, reduced heart rate response and a decrease in rectal temperature while wearing combat clothing, no changes were noted when subjects wore a protective clothing ensemble. Tolerance times remained unchanged at approximately 50 min. A subsequent short-term training model that used daily 1-h exercise sessions for 2 weeks also failed to show any benefit when the protective clothing was worn in the heat. Cross-sectional comparisons between groups of high and low aerobic fitness, however, have revealed that a high aerobic fitness is associated with extended tolerance time when the protective clothing is worn. The longer tolerance time is a function of both a lower starting rectal temperature and a higher rectal temperature tolerated at exhaustion. Improvements in cardiovascular function with long-term training may allow higher core temperatures to be reached prior to exhaustion. Conversely, elevations in core temperature that occur with normal training sessions may familiarize the more fit subjects to the discomforts of exercise in the heat. They suggest that other factors such as differences in body fatness may account for a faster increase in core temperature at a given metabolic rate for less fit individuals.

Gisolfi (1973) compared exercise tolerance times and core temperatures before and after an 11 week training program [30 min/day, 5 days/week in a cool (21°C) temperature-controlled environments] in previously sedentary individuals. Subjects attempted a 100-min walk on a level treadmill at 5.6 km/hr in dry heat (48.9°C). Pre-training walk

performance times were 74 min with a maximum rectal temperature of 39.2°C. Post-training performance times increased significantly to 95 min and rectal temperature did not exceed 38.8°C. Using the ratio of end-point rectal temperature/performance time ratio as an index of heat tolerance, they showed that 8 weeks of interval training in a cool environment produced 50% of the total adjustment resulting from heat acclimation.

The literature involving the effects of physical training on thermoregulatory mechanisms is variable. Some studies suggest that training alone will produce improvements to thermoregulatory function such as an increase in sweat rate and skin blood flow, or a decrease in the temperature at which these mechanisms are activated. However, there are inconsistencies in the findings due to the variable training programs, the level of fitness in which trained and untrained subjects are compared to, as well as the methods of comparing thermoregulatory functions. The greatest concern is when researchers have their participants work at the same percentage of $\dot{V}O_{2max}$ for a given period during a thermal stress test or experimental trial in which the thermoregulatory mechanisms are evaluated. This will cause each individual to work at a different rate of metabolic heat production, therefore producing varied responses to the thermoregulatory system. Therefore, it is unclear as to what the effects of physical training alone will have on the thermoregulatory system when a consistent rate of metabolic heat production is employed during the experimental trial before and after a training program.

PART TWO:

METHODS AND RESULTS OF THE THESIS

Chapter 3

METHODOLOGY

3.0 Participants

Following approval of the experimental protocol from the University of Ottawa Research Ethics Committee and obtaining written informed consent, 10 healthy, non-smoking normotensive participants (7 males, 3 females) volunteered to participate in this study. All participants were healthy; non-smoking; non-heat acclimatized, by not being exposed to any extreme temperatures in the past month, individuals who had not previously suffered any heat-related injuries. Also, they were not currently engaged in any sort of training program for more than thirty minutes, two days per week. Mean ± SD characteristics of these participants were: age, 20 ± 4 years; mass, 72.61 ± 15.34 kg; body fat, 24.04 ± 6.43%; body surface area, 1.84 ± 0.22 m^2; maximum oxygen consumption ($\dot{V}O_{2max}$), 47.73 ± 4.74 mL/kg/min. Female subjects had not taken medications except monophasic oral contraceptive, which provided 30 to 35 μg of ethinyl estrogen and low-dose progestin for 21 days and placebo for seven days. To control for hormonal effects, female subjects were tested during the early follicular phase of their menstrual cycle.

3.1 Instrumentation

3.1.1 Whole-Body Calorimetry

The modified Snellen direct air calorimeter was employed for the purpose of measuring the rate of evaporative ($\dot{H}_E$) and dry heat loss ($\dot{H}_D$), yielding an accuracy of ± 2.3 W for the measurement of rate of total heat loss ($\dot{H}L$). A full peer-reviewed technical

description of the fundamental principles and performance characteristics of the Snellen whole-body calorimeter is available (Reardon *et al.,* 2006).

In summary, the calorimeter incorporated a semi-recumbent constant load eddy current cycle ergometer. The ergometer pedals are located inside of the calorimeter and mechanically linked by chains to the resistance control unit, regulating rate of external work ($\dot{W}$) at a pre-determined level, outside of the calorimeter so that any heat generated by the unit did not enter the calorimeter. The calorimeter was housed within a climatic chamber slightly pressurized (+8.25 mmHg) to nullify potential air leakage through the calorimeter walls. Differential air temperature and humidity were measured over the calorimeter by sampling the influent and effluent air. The water content was measured using precision dew point thermometry (RH Systems model 373H, Albuquerque, NM, USA), while the air temperature was measured using RTD high precision thermistors (± 0.002°C, Black Stack model 1560, Hart Electronics, UT, USA). Air mass flow through the calorimeter was estimated by differential thermometry over a known heat source (2 x 750 W heating elements) placed in the effluent air stream. Differential temperature over the heater is measured using a third aforementioned high precision thermistor placed down-stream from the heater. Air mass flow rate (kg air/min) is continuously measured during each trial. Data from the calorimeter was collected continuously at 8 s intervals throughout the trials. The real time data was displayed and recorded on a personal computer (Dell OPTIPLEX GX270) with LabVIEW software (Version 7.0, National Instruments, TX, USA).

$\dot{H}_E$ was calculated from the calorimetry data every minute using the following equation:

$$\dot{H}_E = \frac{(Massflow \bullet (Humidity_{in} - Humidity_{out}) \bullet 2427)}{60} \text{.....Watts.....} (1)$$

Where: Mass flow is the rate of flow of air mass (kg air/min); ($Humidity_{out}$ – $Humidity_{in}$) is the calorimeter inflow-outflow difference in absolute humidity (g water/ kg air); and 2427 is the latent heat of vaporization of sweat (J/g sweat) at 30°C (Wenger, 1972).

$\dot{H}_D$ from radiation, convection and conduction was calculated from the calorimetry data every minute using the following equation:

$$\dot{H}_D = \frac{(Massflow \bullet (Temperature_{in} - Temperature_{out}) \bullet 1005)}{60} \text{.....Watts.....} (2)$$

Where: Mass flow is the rate of flow of air mass (kg air/min); ($Temperature_{out}$ – $Temperature_{in}$) is the calorimeter inflow-outflow difference in air temperature (K), and 1005 is the specific heat of air (J•/(kg air • K)).

A 6 L fluted mixing box housed within the calorimeter was utilized for the concurrent measurement of metabolic energy expenditure ($\dot{M}$). The indirect calorimetry open circuit technique used expired gas samples drawn from the mixing box. Expired gas was analyzed for oxygen (O_2) (error of ± 0.01%) and carbon dioxide (CO_2) (error of ± 0.02%) concentrations using electrochemical gas analyzers (AMETEK model S-3A/1 and CD 3A, Applied Electrochemistry, Pittsburgh, PA, USA). Expired air was recycled back into the calorimeter chamber to account for respiratory dry and evaporative heat loss. Prior to

each session gas mixtures of 4% CO_2, 17% O_2, balance nitrogen were used to calibrate the gas analyzers and a 3 L syringe was used to calibrate the turbine ventilometer (error ± 3%, typically <1%). Rate of metabolic energy expenditure ($\dot{M}$) was calculated from minute-average values for $\dot{V}O_2$ and the respiratory exchange ratio (RER) using the following equation (Nishi, 1981).

$$\dot{M} \equiv \left(\dot{V}O_2 \bullet \left[\frac{RER - 0.7}{0.3} e_c + \frac{1 - RER}{0.3} e_f \right] \right) \quad \text{..... (3)}$$

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

The calorimeter data was then used to calculate rate of body heat storage ($\dot{S}$) and change in body heat content (ΔH_b) using the following equations:

$$\dot{S} = (\dot{M} - [\dot{H}_E + \dot{H}_D] - \dot{W}) \quad \text{.....Watts..... (4)}$$

$$\Delta H_b \text{ @ time (t)} = \int_{t=0}^{t} (\dot{M} - [\dot{H}_E + \dot{H}_D] - \dot{W})\, dt \quad \text{.....kilojoules..... (5)}$$

Where: $\dot{M}$ is rate of metabolic heat production; $\dot{H}_E$ is rate of evaporative heat loss; $\dot{H}_D$ is rate of dry heat loss; and $\dot{W}$ is rate of external work (all units in W).

3.1.2 Thermometry

Esophageal temperature (T_{es}) was measured by placing a paediatric thermocouple probe of approximately 2 mm in diameter (Mon-a-therm Nasopharyngeal Temperature Probe,

Mallinckrodt Medical, St-Louis, MO, USA) through the participant's nostril while they were asked to sip water through a straw. The location of the probe tip in the esophagus was estimated to be at the level of the eighth and ninth thoracic vertebrae (Mekjavic IB, Rempel ME, 2006). Rectal temperature (T_{re}) was measured using a paediatric 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. Aural canal temperature (T_{au}) was measured with a tympanic thermocouple. The tip of the sensor was inserted into the aural canal until it reached the tympanic membrane, and then withdrawn slightly. It was held into this position with cotton, and then the ear was covered with tape to isolate any temperature imbalance from the outside environment.

Mean skin temperature ($\bar{T}_{sk}$) was calculated using 4 skin temperatures weighted to the regional proportions as determined by Hardy and DuBois: trap 20%, chest 30%, quadriceps 30%, and back calf 20%.

Temperature data were collected using a HP Agilent data acquisition module (model 3497A) at a sampling rate of 15 s and simultaneously displayed and recorded in spreadsheet format on a personal computer (IBM ThinkCentre M50) with LabVIEW software (Version 7.0, National Instruments, TX, USA).

3.1.3 Local Thermal Measurements

Sweat rate was measured using a 5.0-cm_2 ventilated capsule placed over the medial inferior aspect of the trapezius muscle. Anhydrous compressed air was passed through the capsule and over the skin surface (Brooks 5850, mass flow controller, Emerson electric, Hetfield, PA). The vapor density of the effluent air was calculated from the

relative humidity and temperature measured using the Omega HX93 humidity and temperature sensor (Omega Engineering, Stanford, CT). Sweat rate was defined as the product of the difference in water content between effluent and influent air and the flow rate. The flow rate through the capsule was 0.7 L/min. The sweat rate value was adjusted for skin surface area under the capsule (expressed in $mg/cm^2 \cdot min$).

SkBF was estimated using laser-Doppler velocimetry (PeriFlux System 5000, main control unit; PF5010 LDPM, function unit; Perimed, Stockholm, Sweden) at the right midanterior forearm. The laser-Doppler flow probe (PR 401 angled probe, Perimed) was taped to cleaned skin, in an area that did not appear by visual inspection to be overly vascular and from which consistent readings were noted (Mack, 1998). Cutaneous vascular conductance (CVC) was calculated as the ratio of laser-Doppler flow to MAP. At the end of the experiment, local skin temperature at the skin site was raised to 42°C until peak CVC was measured (~30 min) (Taylor *et al.,* 1984). A heating element (PF 5020 temperature unit, Perimed) housed the laser-Doppler flow probe, and it was then activated to elevate local skin temperature to 42°C. Peak CVC was determined as a sustained elevated plateau in local SkBF. CVC data are presented as a percentage of maximal CVC as determined by local heating. SkBF measures were recorded from the left midanterior forearm such that the arm was level with the heart.

3.1.4 Heart Rate and Blood Pressure Measurements

Heart rate was monitored by a strap placed around the chest (Polar Vantage heart rate monitor). A heart rate monitor watch recorded the data which was then transferred to a portable memory stick. Heart rate was measured to help determine the individual's

workload. Blood pressure was also be measured at fifteen minutes intervals throughout the trial and at five minute intervals for the first fifteen minutes into recovery.

3.1.5 Subjective Measurement

The Borg scale and thermal sensation scales were also used to aide in assessing the participants comfort and work intensity. The Borg scale ranged between 6 – 20, where 6 meant no exertion at all and 20 meant maximal exertion. The thermal sensation scale ranged from 0 – 7, where 0 meant neutral and 7 meant extremely hot. These scales were used as perceived exerction as well as safetly measures.

3.2 Experimental Design

Subjects who volunteered were required to attend 4 supervised, aerobic exercise training sessions per week over an 8-week period at the University of Ottawa training center. The exercise training program took place during the winter months (December to April) in order to eliminate any effects of heat acclimation from the environment during summer months. All participants were asked to participate in 2 experimental test sessions conducted prior to the start of the training program, and at the end of the 8-week training session. All experimental sessions were performed at least 48 hours following the last exercise session to ensure complete recovery.

3.2.1 Preliminary information session

Both the preliminary session and the experimental test sessions took place in the Human Bioenergetics and Environmental Physiology Laboratory located on the main campus at the University of Ottawa. The time involvement was approximately 45 min to 1 hour for

the preliminary session. During the preliminary session, all experimental procedures, equipment, and measuring devices were reviewed with the subject. Subjects were then asked to complete a health-screening form called the Physical Activity Readiness Questionnaire (Par-Q). This questionnaire is a standard questionnaire that has been developed by the Canadian Society for Exercise Physiology. It was used to assist the researcher to evaluate the subject's general physical health and level of physical activity. Also, the American Heart Association/American College of Sports Medicine (AHA/ACSM) Health/Fitness Facility Pre-participation Screening Questionnaire was completed. At the end of the preliminary session, the subject had the opportunity to read the Background and Informed consent document. If the subject agreed to participate in the study they signed the informed consent.

The completion of some basic measurements including height, weight, and body density, as determined by hydrostatic weighing, were taken. The value for body density was then placed into the Siri formula and the percent body fat was calculated. They were then asked to perform the first of two maximal incremental exercise-tests on a treadmill. Typically, this test lasted no longer than 12 minutes. The measurement of oxygen consumption during the test was used to determine their maximal aerobic capacity and the heart rate was monitored continuously using a polar heart rate monitor. Information obtained from this test was also be used to calculate the work intensity that was used during the exercise training sessions and the workload performed during the experimental test sessions.

3.1.2 Training program

All participants were provided with a gym membership to the University of Ottawa Fitness Centre, if they did not already have one. The membership fee was provided by the study funding to remove economic barriers to participation. During the 8-week training program, subjects were required to attend 4-5 supervised exercise-training sessions per week. The duration of the training session increased progressively from 30 minutes at the start of the training program to a maximum of 90 minutes at the end of the 8-week program (including warm-up and cool-down time). The program was designed to increase the participant's aerobic exercise capacity and it was tailored to meet each individual's personal fitness level and rate of progression. An example of the training program can be seen in the Appendix. Adjustments in exercise intensity were made periodically and progressively as needed. Attendance was verified through constant supervision and exercise logs. The training program represented a mixture of different exercise training techniques including continuous steady state exercise, interval training, Fartlek training, and resistance circuit training. The researcher supervised all training sessions, with the assistance of volunteers from the School of Human Kinetics who are certified as Certified Fitness Consultants from the Canadian Society for Exercise Physiology.

Continuous steady state training was performed either on a treadmill, stationary bike or elliptical machine. Interval training involved running intermittently for a pre-determined distance or duration, with recovery breaks between sets. The intensity of exercise was higher (between 75-90% of the subject's pre-determined heart rate reserve) than that used for the continuous exercise (between 55-75% of the subject's pre-determined heart rate

reserve). Most forms of fartlek running are time intervals in a ladder fashion. An example would be running for 1 min, 2 min, 3 min, 4 min, 5 min, 4 min, 3 min, 2 min, 1 min at a greater intensity than continuous running and using the same amount of time for recovery (Bompa, 1999). This is a useful type of training to stress the aerobic system and break up the monotony of continuous training (Bowerman & Freeman, 1991). Circuit training was performed using stationary weight equipment and free weights, which targeted different muscle groups. Participants were required to lift a light to moderate weight for three sets of 12-15 repetitions. There was approximately a 15-second rest in between each set. Then they proceed to the next exercise station and so on to complete the circuit. The training program was designed by the researcher and changes were made on a daily basis for each individual.

3.1.3 Experimental test sessions

All experimental test sessions in the calorimeter were conducted at the same time of the day to negate any differences in their circadian rhythm. The final trial took place at least 48 hours following the last exercise training session. All participants were asked to refrain from exercise for 24 hours prior to the start of each calorimeter trial. On each day, care was taken to avoid major thermal stimuli or a substantial increase in metabolic rate between awakening and the start of the experiment. Following instrumentation, participants consumed 500 mL of water to ensure ample hydration at the beginning of the trial. They then entered the calorimeter set at 30.0°C and began a 60-min habituation period in a semi-recumbent position. Participants then began cycling on the semi-recumbent constant-load cycle ergometer at a constant rate of metabolic heat production ($\dot{M}$-$\dot{W}$) equalling 450 watts. The rate of metabolic heat production during the

experimental test sessions remained constant for each individual. Exercise stopped after either ninety minutes of continuous cycling or volitional fatigue, whichever occurred first. Typically, the exercise session lasted for ninety minutes. In addition to this, participants will be free to terminate exercise at any point, for any reason they feel necessary, including symptoms of heat illness (e.g. nausea, dizziness or muscle weakness). Participants then performed a 60-minute resting recovery in the semi-recumbent position. Subjective measures (thermal and perceived exertion) were taken at 15-min intervals throughout the experimental session from beginning of rest to the end of recovery. Blood pressure was taken electronically by the participant at fifteen minute intervals throughout the trial and at five minute intervals for the first fifteen minutes after cessation of exercise.

All female participants were required to complete a questionnaire relating to their menstrual cycle prior to the experimental test sessions in the calorimeter. Sex hormones vary significant during the menstrual cycle and are known to significantly influence heat loss responses. Standard published procedures in this field of research require the determination of the phase (follicular or luteal) of the menstrual cycle at the time of the experimental session. All female participants were tested during their follicular phase since it has been shown that body temperature is normally regulated during this phase.

3.3 Statistical Analysis

A two-way analysis of variance (ANOVA) with repeated measures was performed. The independent variables are the state of training (i.e., before and after the eight-week training program) and time (baseline, 2, 5, 8, 12, 15, 30, 45, 60, 90 min during exercise

and recovery). The following dependant variables were analyzed as a function of increases from baseline rest: rate metabolic heat production, rate of evaporative heat loss, rate of dry heat loss, rate of total heat loss, core temperatures, mean skin temperature, sweat rate, forearm skin blood flow, mean arterial pressure, and heart rate during the trial in the calorimeter. Paired sample t-tests were used to perform post hoc comparisons between time and state of training and to compare changes in body heat content, participant characteristics, as well as pre-exercise values for core temperatures and heart rate.

The level of significance was set at 0.05 and alpha level was adjusted during multiple comparisons so as to maintain the rate of Type I error at 5% during the Bonferroni post hoc analysis ($p \leq 0.05n^{-1}$; where n = number of comparisons). All analyses were performed using the statistical software package SPSS 15.0 for Windows (SPSS Inc. Chicago, IL, USA).

Chapter 4

RESULTS

4.0 Cardiorespiratory Fitness and Body Composition

Treadmill maximal oxygen consumption ($\dot{V}O_{2max}$) increased significantly over the eight-week training period ($p \leq 0.05$). $\dot{V}O_{2max}$ on the recumbent bike also increased significantly ($p \leq 0.05$). There was a trend ($0.05 > p < 0.1$) for body fat percentage to be decreased post-training compared to pre-training ($p=0.069$). Body mass was also slightly decreased post-training compared to pre-training ($p=0.074$). Lean body mass increased slightly ($p=0.083$) post-training compared to pre-training. (Table 1).

4.1 Calorimetry Data

Metabolic heat production increased at the onset of exercise and remained elevated throughout exercise ($p \leq 0.05$) and was similar during the experimental test session between the pre- and post-training period ($p=0.401$). The rate of total heat loss rose significantly with time during exercise ($p \leq 0.05$), but was not significantly different before or after training during exercise ($p=0.087$). On the other hand, the rate of total heat loss declined with time at the end of exercise ($p \leq 0.05$) and at a significantly greater rate during recovery ($p=0.037$) between 12 ($p=0.003$) and 15 ($p=0.020$) minutes post-training compared to pre-training (Fig. 1A).

Evaporative heat loss increased significantly with time ($p \leq 0.05$), however, there was no main effect with training on the rate of evaporative heat loss during exercise ($p=0.124$). During the sixty minute recovery period, the rate of evaporative heat loss decreased with

time ($p \leq 0.05$), and the rate of decay was greater post-training ($p=0.018$) at minute 12 ($p=0.002$) (Fig 1B). The rate of dry heat loss did not change during exercise due to time ($p=0.362$) or training ($p=0.108$) from pre- to post-training. However, during recovery, the rate of dry heat loss changed with time ($p=0.593$), but was not different with training ($p=0.593$) (Fig. 1B).

The rate of heat storage increased with time during exercise ($p \leq 0.05$), but there was no main effect of training on the rate of heat storage ($p=0.874$). Similarly, the rate of heat storage decreased with time during recovery ($p \leq 0.05$), but there was no difference in the rate of heat storage during the recovery due to training ($p=0.140$). (Fig. 2). There was no change in body heat content during exercise, as measured by calorimetry, from pre- +441±28 kJ to post-training +430±38 kJ ($p=0.385$) (Fig. 3A). Also, during recovery there was no difference in the change in body heat content between pre- and post-training ($p=0.808$). (Fig. 3B), which resulted in no difference in the residual body heat content after the 60-min recovery period between pre- and post-training (Fig. 3C).

4.2 Thermometry

There was a trend for esophageal temperature to be lower at baseline post-training when compared to pre-training (36.95±0.24°C, 37.10±0.28°C) ($p=0.060$) (Fig. 4). Esophageal temperature increased with time during exercise ($p \leq 0.05$), and there was a trend for esophageal temperature to be lower after training during exercise ($p=0.0.68$). During recovery, esophageal temperature decreased with time ($p \leq 0.05$), and there was a trend for lower temperature after training ($p=0.0.076$). When adjusted from baseline, there was no

difference in esophageal temperature from pre- to post-training during exercise (p=0.290) or recovery (p=0.279) (Fig. 5A).

Rectal temperature was significantly lower at baseline after training (36.84±0.36°C) when compared with pre-training (37.05±0.38°C) ($p \leq 0.05$) (Fig. 4). Rectal temperature increased with time during exercise ($p \leq 0.05$), and was significantly lower during exercise after training (p=0.056). Rectal temperature was lower during exercise at 30 – 90 minutes of exercise. During recovery, rectal temperature decreased with time ($p \leq 0.05$), and was significantly lower after training during the entire recovery period (p=0.001). However, when adjusted from changes in baseline, there was no significant different in rectal temperature during exercise (p=0.914) or during recovery (0.356) (Fig. 5B).

Tympanic temperature was significantly lower at baseline after training when compared with pre-training (36.56±0.25°C, 36.73±0.30°C) ($p \leq 0.05$) (Fig. 4). Tympanic temperature increased with time during exercise ($p \leq 0.05$) and was significantly lower during exercise from 15 to 90 minutes (p=0.031). During recovery, tympanic temperature decreased with time ($p \leq 0.05$) and there was a trend for it to lower after training at 2-5 minutes and 15 minutes (p=0.069). When adjusted from baseline, there was no difference in tympanic temperature during exercise (p=0.356) or recovery (p=0.665) (Fig. 5C).

Mean skin temperature was not significantly different at baseline post- compared to pre-training (34.13±0.51, 34.32±0.53), (p=0.255). Mean skin temperature increased with time during exercise ($p \leq 0.05$), but there was no main effect with training (p=0.444). During

recovery, mean skin temperature decreased with time ($p \leq 0.05$), but there was no effect with training ($p=0.934$). (Fig. 5D).

4.3 Local Thermal Responses

Local sweat rate (LSR) increased with time during exercise ($p \leq 0.05$), but there was no main effect with training ($p=0.108$). During recovery, LSR decreased with time ($p \leq 0.05$), but there was no main effect with training ($p=0.352$). (Fig. 6A).

Percentage of maximum cutaneous vascular conductance (%CVC) increased with time during exercise ($p \leq 0.05$), but there was no main effect with training ($p=0.910$). During recovery, %CVC decreased with time ($p \leq 0.05$), but there was no main effect with training ($p=0.276$). (Fig 6B).

4.4 Heart Rate and Blood Pressure Responses

Mean arterial pressure (MAP) increased with time during exercise ($p \leq 0.05$). MAP was significantly lower at baseline post-training 82±8.03 mmHg compared to pre-training 87±11.69 mmHg ($p=0.011$). Although MAP was lower at baseline and throughout exercise after training there was no significant difference during exercise ($p=0.111$) or recovery ($p=0.288$) pre- to post-training. During recovery MAP decreased with time ($p \leq 0.05$). (Fig. 7A).

Pre-exercise values for heart rate were lower after training (73±9.19 bpm) when compared with before training (83±6.12 bpm) ($p=0.004$). Heart rate increased with time during exercise ($p \leq 0.05$), and was significantly lower after training during exercise from 8 to 90 minutes ($p=0.005$). During recovery, heart rate decreased with time ($p \leq 0.05$), and

was significantly lower after training during the entire recovery period ($p=0.001$) (Fig. 7B).

4.5 Subjective Measurements

The rating of perceived exertion during exercise was significantly reduced from a value of 15 on the Borg Scale to 13 at the end of exercise due to training ($p=0.016$). The rating of thermal sensation during exercise was also lower following the training period.

Chapter 5

DISCUSSION

In the present study, we show a rapid increase in the rate of heat production to the pre-determined values of 450 Watts in both pre- and post-training exercise trials. The exponential increase in the rate of heat loss lagged significantly behind that for the increase in the rate of heat production, resulting in net body heat storage, which was similar between pre- and post-training. Similar increases in the rate of total heat loss during exercise were observed despite significant improvements in cardiorespiratory fitness, as measured by an increase in $\dot{V}O_{2max}$ by 12%. The main findings of this study are that an eight-week physical training program in previously sedentary individuals resulted in improvements in cardiorespiratory fitness and body composition, but did not result in improvements in whole-body heat loss during exercise. This can be explained by the individuals working at the same rate of metabolic heat production during the pre- and post-training experimental test sessions.

At rest, a trained individual will typically have a lower core temperature corresponding to a lower metabolic rate and heart rate (Gisolfi, 1973; McLellan, 2001; Okazaki *et al.*, 2002; Shvartz *et al.*, 1973; Shvartz, 1974; Styrdom *et al.*, 1966). In the present study, we observed a decrease in regional body core temperature during rest of 0.15±0.06°C for esophageal, 0.2±0.02°C for rectal, and 0.22±0.05°C for tympanic. In parallel, we observed a reduction in resting heart rate, but no change in metabolic rate. Shvartz *et al.* (1974) observed a decrease in resting rectal temperature by 0.2°C following a 12 day

exercise protocol @ 85% $\dot{V}O_{2max}$, 1 hr/day, at 21.5°C. A similar decrease of 0.17°C was seen by Kampmann *et al.* (2008) after a 12-day exercise training protocol involving 4x25 min treadmill walking at 4 km/hr (ambient air temperature of 25°C). They also reported a lower resting heart rate with the greatest decreases seen in the first four days (Shvartz *et al,* 1974). It is unclear however, if these improvements in thermal and cardiovascular responses during rest were associated with improvements in heat tolerance during exercise.

Studies show that highly trained individuals are able to tolerate exercise in the heat for longer periods of time compared to an untrained individual. The increase in heat tolerance is thought to be the result of improvements in thermoregulatory functions as measured by a reduction in the level of core temperature paralleled by an enhanced skin blood flow and sweating response. These changes combined with the improvements in cardiovascular function (i.e., reduced heart rate response for a given workload) is thought to result in improvement to heat tolerance (Baum *et al.,* 1976; Cheung & McLellan, 1998; Gisolfi, 1973; Henane *et al.*, 1977; Shvartz *et al.*, 1974). In the present study, we observed lower maximal exercise core temperatures by 0.3°C in both esophageal and rectal, and 0.24°C in tympanic. However, when we adjusted for the lower starting temperature, there was no difference in the relative increase from baseline pre- to post-training. Of note, however, we did observe a decrease in maximal exercise heart rate during the trial, by 20±8 bpm. Consistent with our research, Shvartz *et al.* (1974) found a decrease in the maximal absolute rectal temperature increase during exercise by a magnitude of 0.4°C. Gisolfi & Robinson (1969) showed that after a 6-week intense

training program (i.e., 1 hr per day, 5 days per week, ambient air temperature of 21°C) peak exercise rectal temperature was reduced by 0.9°C. This was paralleled by a reduction in the peak exercise heart rate by 24 bpm. Nadel *et al.,* (1974) showed a decrease in heart rate by 6 bpm during a heat stress test involving 15 min cycling at 60-70% $\dot{V}O_{2max}$ (ambient air temperature of 23.5°C) performed following 10 consecutive days of moderate intensity exercise (i.e., 70-80% $\dot{V}O_{2max}$ in ambient air conditions of 22°C). An even further decrease in heart rate by 11 bpm was measured after a subsequent 10 days of training performed at a lower intensity (i.e., 50% $\dot{V}O_{2max}$ in the heat; ambient air temperature of 45°C dry heat or 36°C wet heat).

Numerous studies have reported an increase in sweating sensitivity and/or a decrease in the temperature at which the onset for sweating initiates following exercise training. When comparing endurance trained versus untrained individuals, peripheral sweat rate has been shown to be higher in the trained individuals (Buono *et al.,* 1988; Selkirk & McLellan, 2001). Previously sedentary individuals have also been shown to increase their sweating sensitivity, shown through a greater sweat output at a given core temperature, during a physical exercise program (Gisolfi & Robinson, 1969; Henane, 1977). Repeated bouts of exercise increase an individual's core temperature initiating the sweating response. Adaptations to sweating occur for an individual to become more responsive to a thermal stimulus. The sweat gland starts to produce sweat at a lower core temperature as well as more sweat will be produced. Exercise training has also been shown to decrease the threshold for the onset of sweating. Roberts *et al.* (1977) observed a decrease in

internal temperature at the onset of sweating by 0.1°C after one-hour exercise sessions of moderate intensity exercise performed over 10-consecutive days. This was further reduced by 0.12°C with an additional 10 days of exercise training in the heat. Studies have reported a significantly higher skin blood flow in highly fit individuals as compared to sedentary individuals (Ho *et al.,* 1997; Tankersely *et al.,* 1991). Roberts *et al.* (1977) reported a shift in the threshold for skin vasodilation towards a lower core temperature by 0.21°C after exercise training and even lower by 0.24°C after exercise training in the heat. In the present study, we observed a decrease in the esophageal temperature to the onset of sweating and skin blood flow by 0.16°C and 0.17°C, respectively. However, no corresponding increase in whole-body heat loss was observed.

During post-exercise recovery, elevations in core temperature persist despite the rapid decrease in metabolic heat production. Thoden *et al.* (1994) reported a prolonged and sustained elevation in post-exercise esophageal temperature of ~0.5°C above baseline resting values for the duration of a 65-min recovery period. In the present study by the end of recovery, we observed elevations in core temperature from baseline values pre-training of 0.1°C, 0.35°C, and 0.15°C at esophageal, rectal, and aural canal locations, respectively. Post-training core temperatures were still elevated at the end of 60-min recovery from baseline values (0.12°C, 0.28°C and 0.23°C esophageal, rectal and aural canal, respectively) as well. Heart rate remained elevated 60-min into recovery pre-training by 17 bpm and post-training by 19 bpm, but the absolute values were lower post-training than pre-training. The rates of decay of total heat loss and dry heat loss were

significantly greater during recovery post-training than pre-training, which was reflected in a larger body heat content post-training at the end of recovery.

Exercise training is known to elicit physiological adaptations that can improve both cardiorespiratory and thermoregulatory function. In the present study, we quantified the effects of physical exercise by measuring the rates of whole-body evaporative and dry heat loss as well as ΔH_b by performing simultaneous minute-by-minute measurements of the individual heat balance components by whole-body calorimetry. Although we observed a decrease in resting and maximal exercise core temperature, no changes in whole-body heat loss response was observed.

Our present findings suggest that improvements in physical fitness do not result in improvements in whole-body heat loss. This finding is due to the fact that the participants were exercising at the same rate of metabolic heat production during the pre- and post-training experimental test sessions. Previous studies had participants working at the same percentage of their $\dot{V}O_{2max}$, which will ultimately have the individuals working at different heat loads. Therefore, they will exhibit differences in the thermal responses, which will not necessarily be due to different levels of fitness. It needs to be understood that when comparing the benefit of exercise training on thermoregulatory mechanisms, each participant needs to be given the same thermal input to quantify differences in thermal output responses.

PART THREE:

CONCLUSION AND PERSPECTIVES

OF THE THESES

Chapter 6

CONCLUSIONS AND PERSPECTIVES

6.0 Conclusion

In summary, an eight-week exercise training program has many health and cardiovascular benefits to individuals, but is not an appropriate stimulus to elicit a significant response in whole-body heat loss and ultimately a change in body heat content. Since it has been reported that training during heat exposure will have a greater effect on local sweat rate and skin blood flow than just training alone in a non-heat stressed environment (Kampmann *et al.,* 2008; Nadel *et al.,* 1974; Shvartz *et al.,* 1973; Styrdom *et al.,* 1966), further studies are needed to determine the effects of passive heating as well as exercise in the heat on whole-body heat loss. Whole-body direct calorimetry is the gold standard in quantifying the rate of whole-body heat loss and changes in body heat content. Therefore, assumptions based on changes to local responses can only be verified if whole-body direct calorimetry is used simultaneously and the participants are working at the same rate of metabolic heat production.

6.1 Perspectives

Our findings will have practical implications to individuals facing situations under heat stress. Athletes who are preparing for competition in a warm climate must train in warm conditions prior to competition to ensure acclimation occurs. In addition, industrial workers, whose work environment places them under heat stress, are also at risk for heat illness and should consider training in a warm environment to better prepare themselves for their daily job requirements. It is important for individuals to find methods which eliminate thermal discomfort during work in the heat. It can be suggested that physical

conditioning will reduce some of the thermal strain, bur training alone will not be sufficient enough to have an effect on achieving heat balance at a quicker rate.

PART FOUR:

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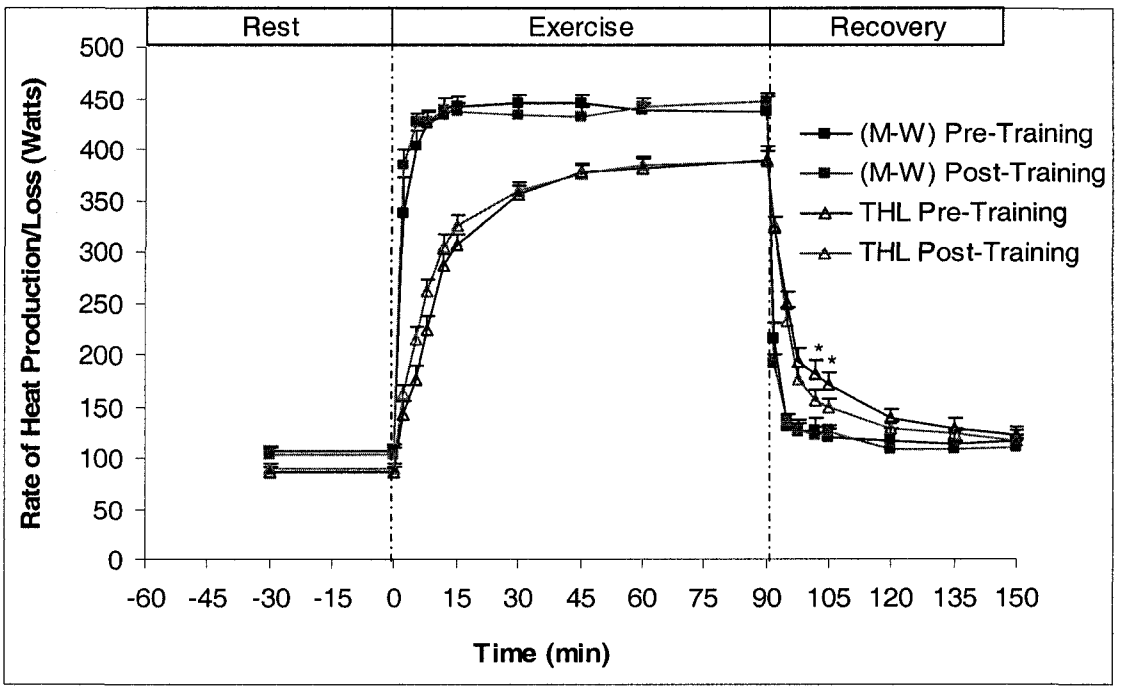
Table 1. Participant Characteristics Mean and SD

	Height (cm)	Weight (Kg)	BMI	VO2 (mL/min) treadmill	VO2 (mL/kg/min) treadmill	VO2 (mL/min) bike	% fat
Pre-Training	170.75	72.61	24.82	3466	47.73	2808.14	24.04
	9.30	15.34	3.68	787.15	4.74	651.09	6.43
Post-Training	170.75	71.86	24.52	3894	54.19	2937.67	22.45
	9.30	14.19	3.42	885.59	4.09	662.03	7.25

* represents $p < 0.05$

Figure 1.

A. Rate of metabolic heat production and total heat loss (W)



B. Rate of evaporative and dry heat loss (W)

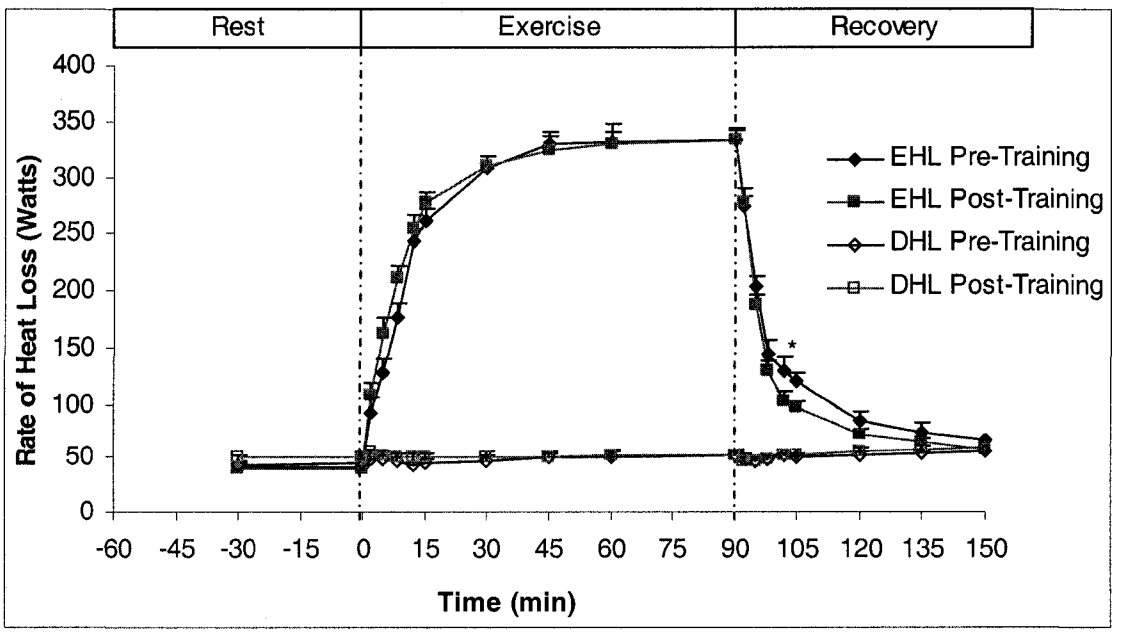


Figure 2.
Rate of Heat Storage (Watts)

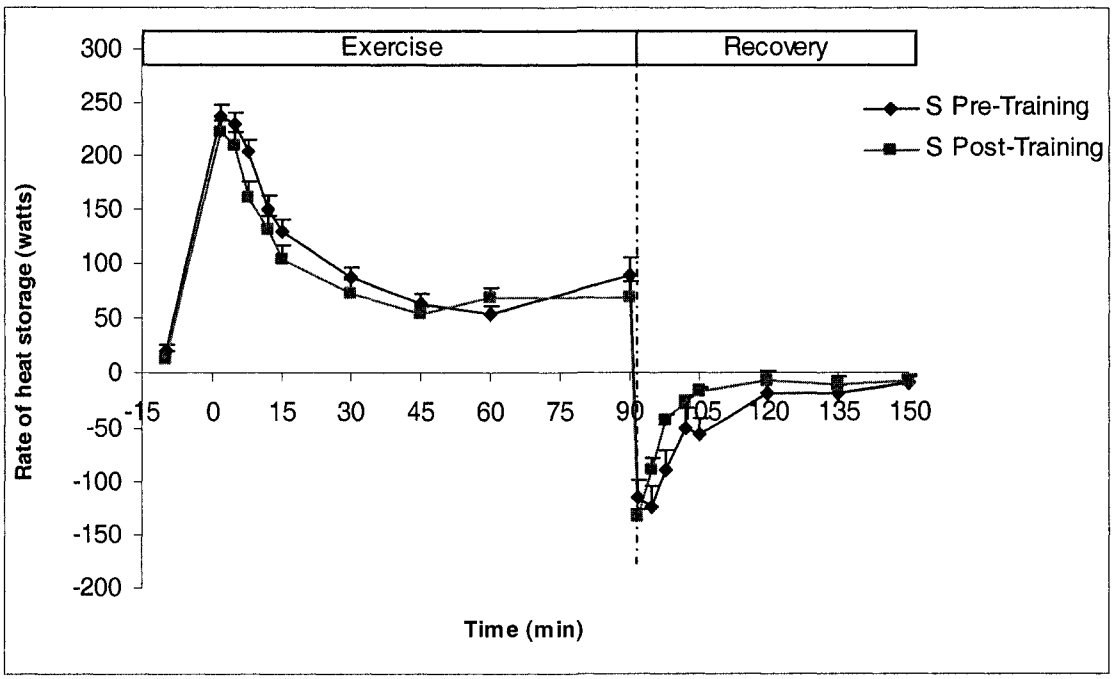
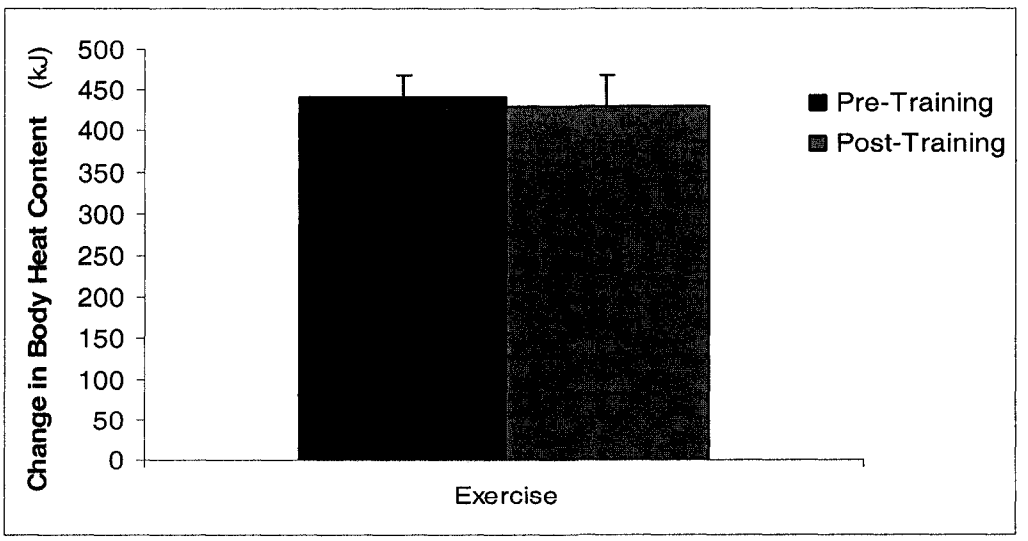
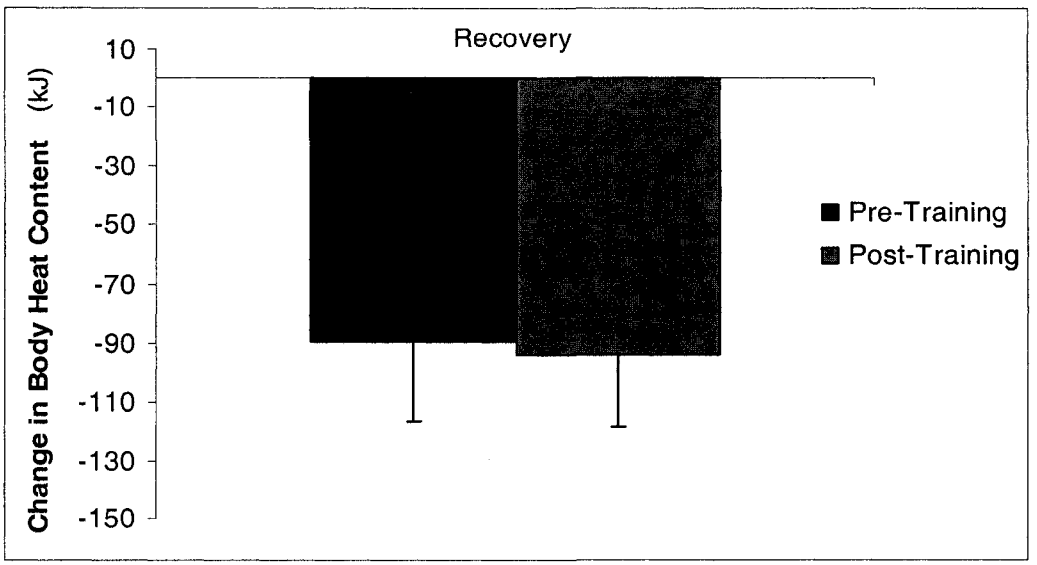


Figure 3.
A.
Change in body heat content during exercise



B.

Change in body heat content during recovery (kJ)



C.

Residual body heat content after 60-min recovery (kJ)

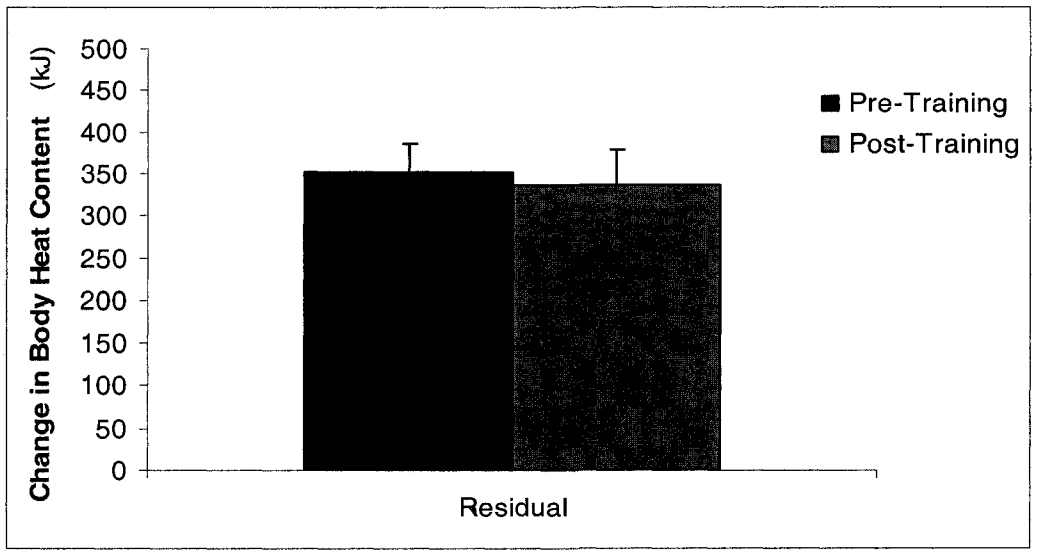


Figure 4.

Baseline Core Temperatures

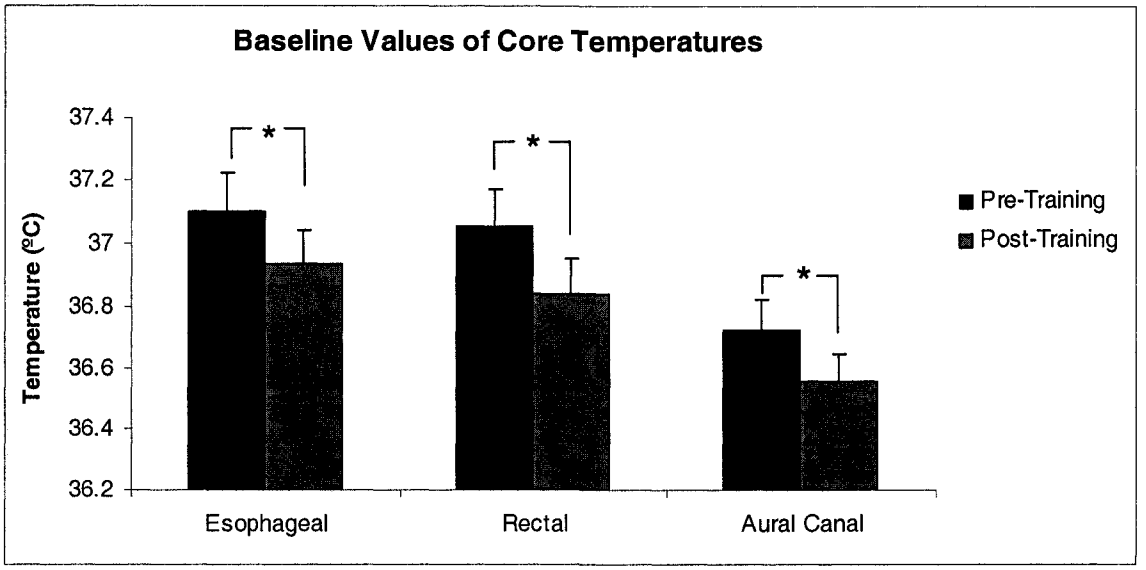
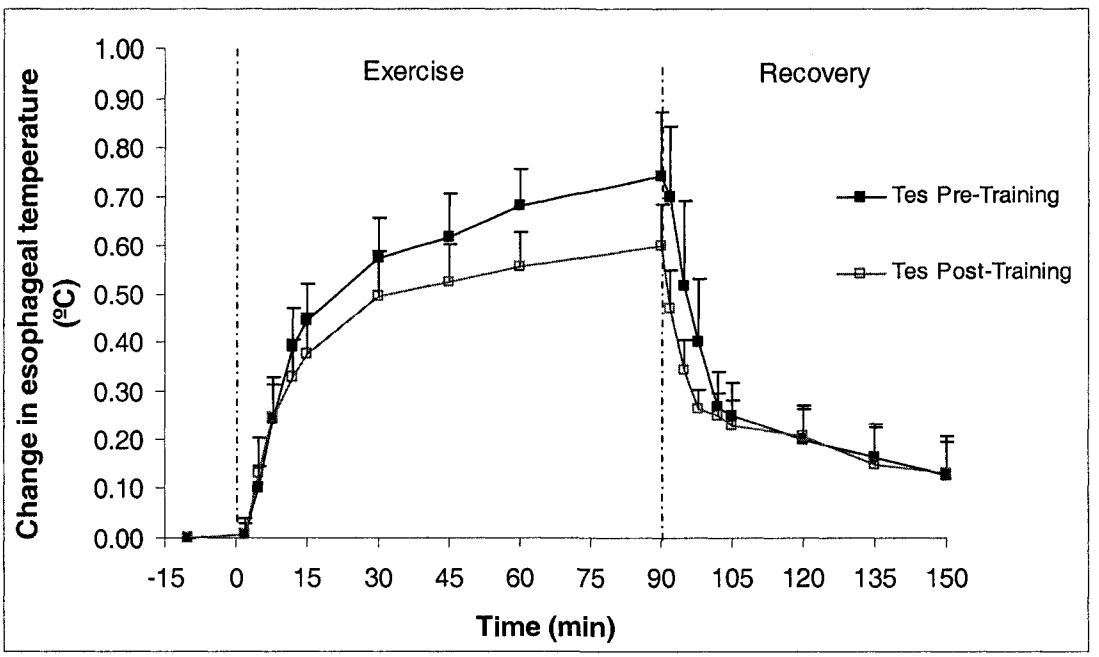
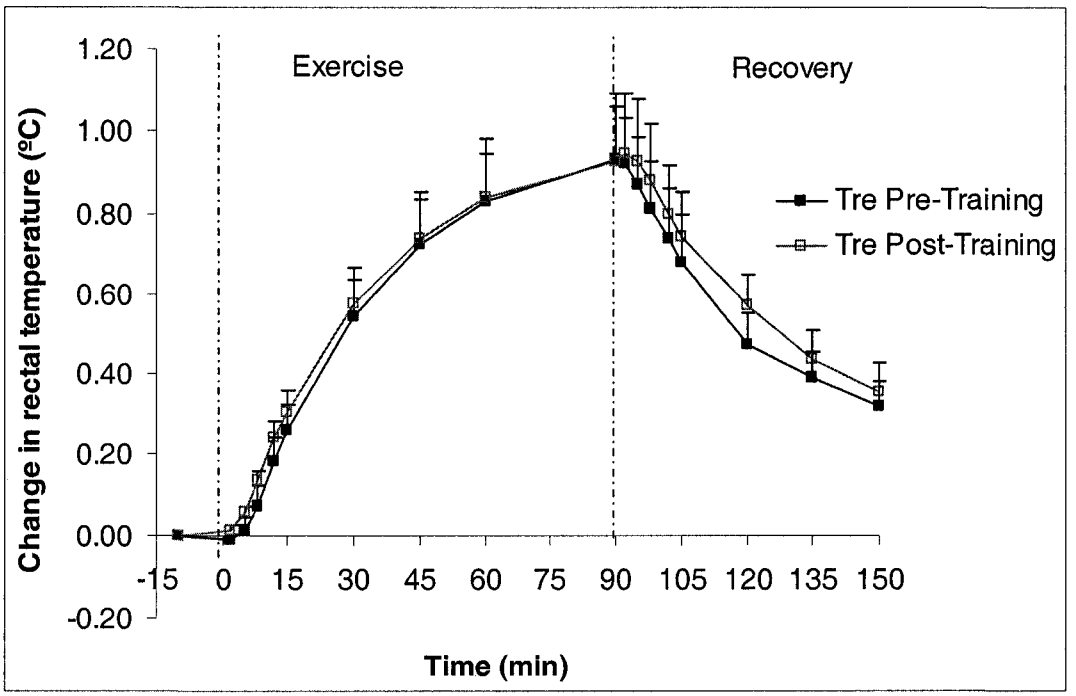


Figure 5.

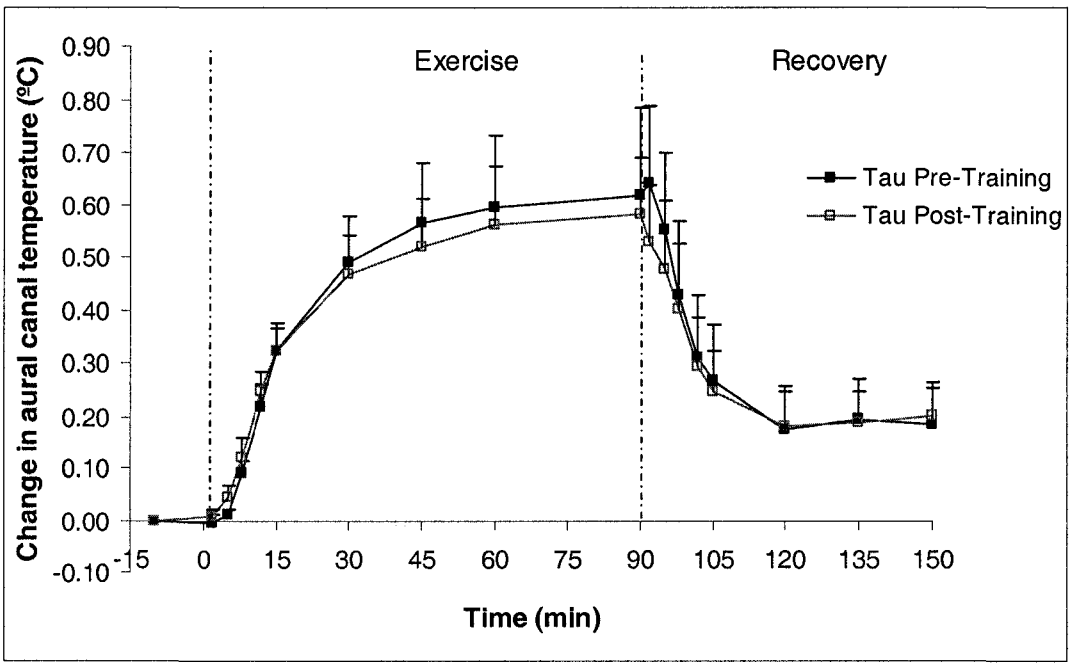
A. Esophageal Temperature change from baseline (ºC)



B. Rectal Temperature change from baseline (°C)



C. Aural Canal Temperature change from baseline (°C)



D. Mean Skin Temperature (°C)

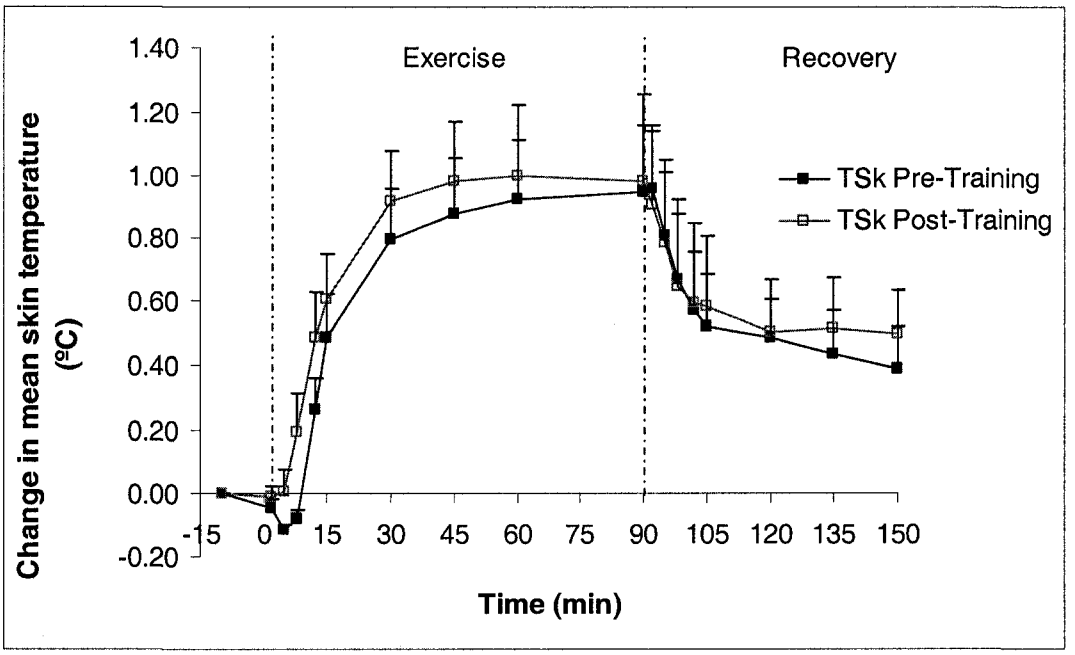
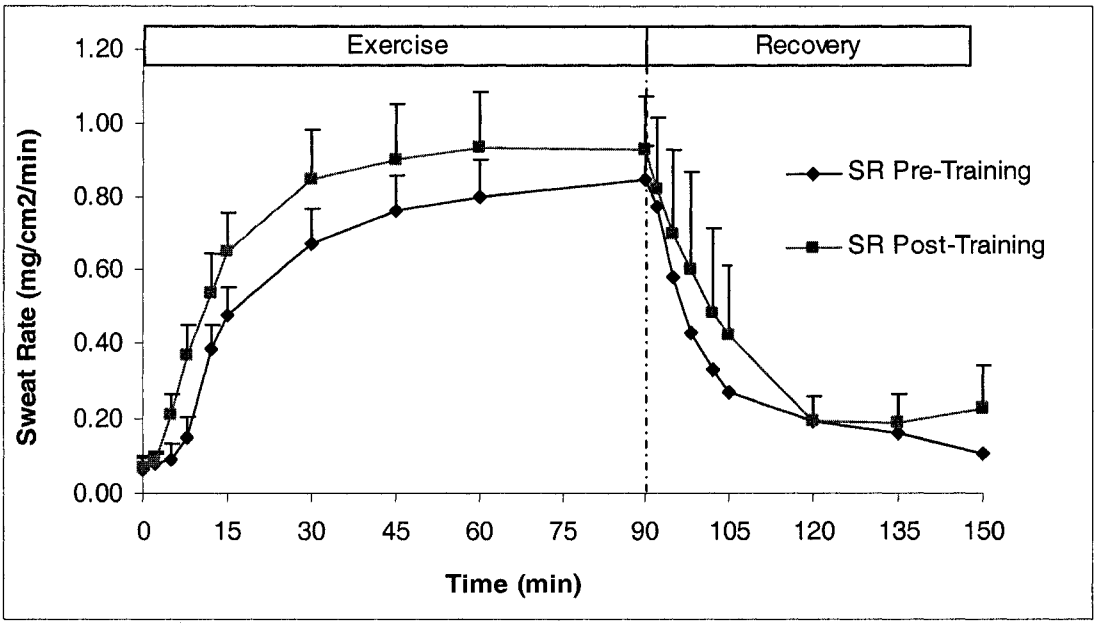


Figure 6.

A. Sweat Rate (mg/cm^2·min)



B. Percentage Cutaneous Vascular Conductance (%)

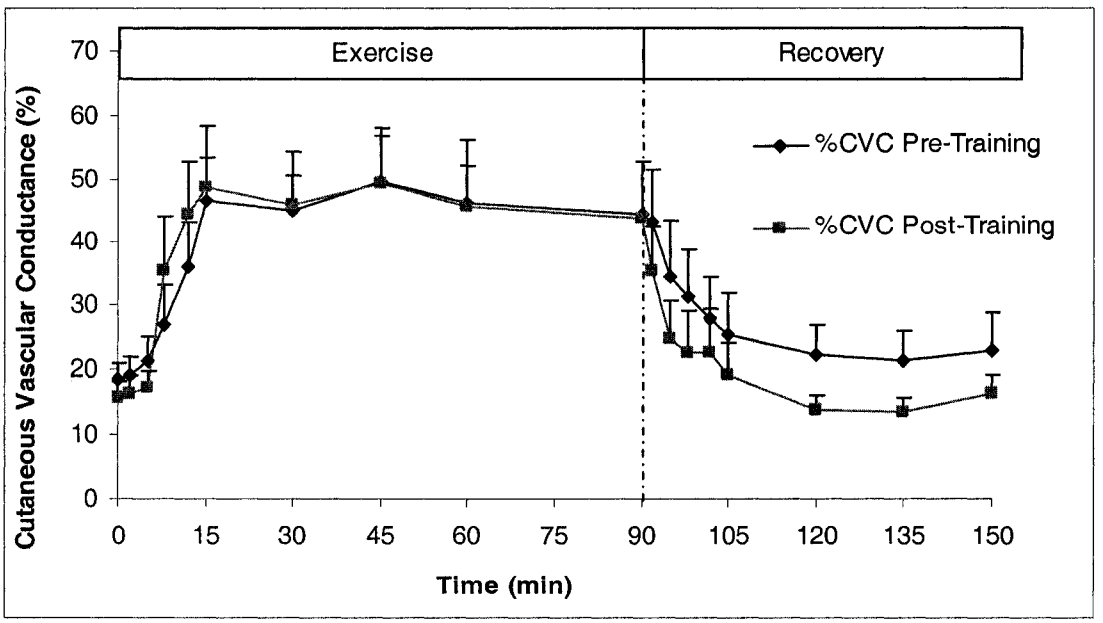
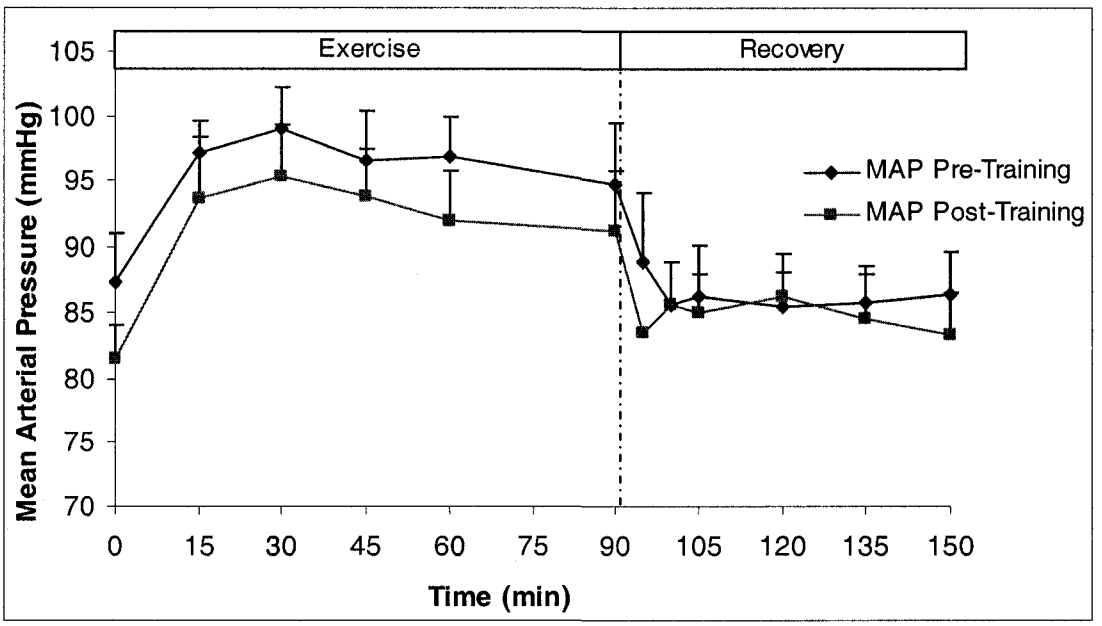
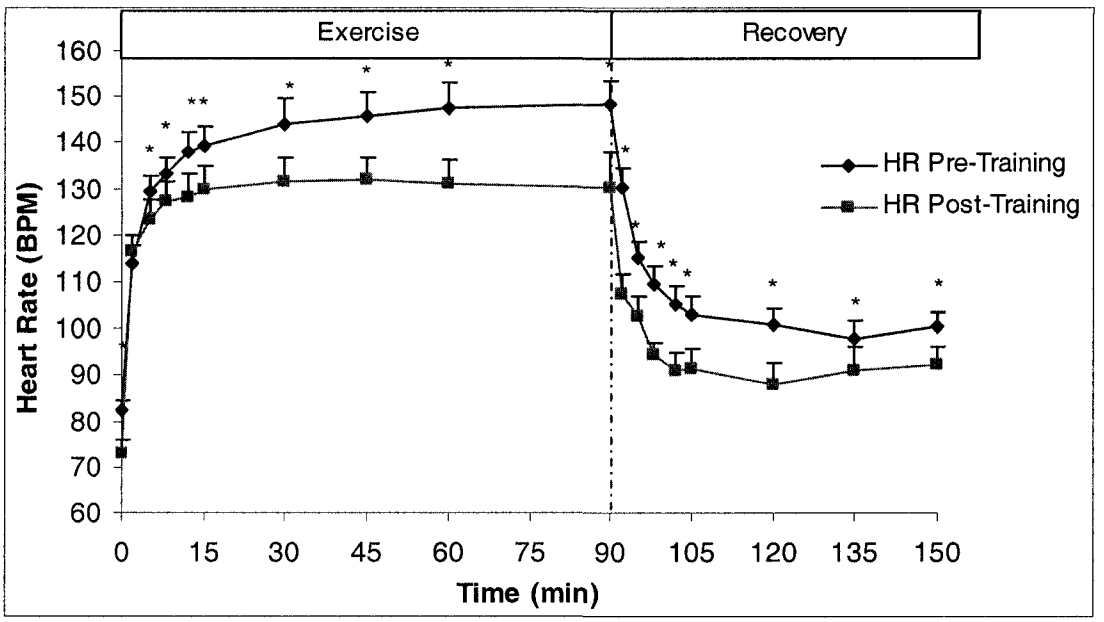


Figure 7.

A. Mean Arterial Pressure. (mmHg)



B. Heart Rate (bpm)



APPENDIX

Week 1 Experimental Test Session	Day Off	3(10 min run, 1 min walk) min. @ 40-50% HRR	30 min. @ 40-50% HRR	Day Off	35 min. @ 45-55% HRR	Day off
Week 2 40 min @ 45-55% HRR	10 min warm up 3x4 min @ 80% HRR 10 min cool down	35 min. @ 50-60% HRR	Day off	10 min WU Circuit 10 min CD	40 min. @ 50-60% HRR or day off	Day off
Week 3 45 min @ 45-60% HRR	10 min WU 5 min @ 80% HRR 5 min rec 10 min @ 80% HRR 10 min. CD	35 min. 55-65% HRR	Day off	45 min. @ 50-65% HRR	10 min WU Circuit 10 min CD or day off	Day off
Week 4 50 min @ 55-70% HRR	35 min. @ 50-65% HRR	Day off	40 min. 50-65% HRR	10 min WU 1,2,3,4,5, 4,3,2,1 min. 10 min CD	Day off	35 min. @ 55-70% HRR

Week 5 55 min run	35 min. @ 55-70% HRR	10 min WU Circuit 10 min CD	Day off	40 min. @ 55-70% HRR	10 min WU 4X30 sec 2x3 min 4x30 sec 10 min CD	Day off
Week 6 60 min @ 55-70% HRR	10 min WU 5(3 min @ 80-90% HRR) 10 min CD	40 min @ 55-65% HRR	Day off	10 min WU Circuit 10 min CD	45 min @ 60-75% HRR	Day off
Week 7 60-70 min @ 55-70% HRR	40 min. @ 65-80% HRR or day off	Day off	10 min WU Circuit 10 min CD	45 min. @ 60-85% HRR	Day off	10 min WU 4x5 min. @ 75-95% HRR 10 min CD
Week 8 30 min. @ 60-85% HRR	45 min @ 60-85% HRR	Day Off	10 min WU 3x1,2,3 min 10 min CD	Day off	VO_{2max} Test Body Comp. Test	Experimental Test Session