THE EFFECT OF AN ACCLIMATION PROGRAM ON EXERCISING IN THE HEAT FOR A FIT FEMALE POPULATION

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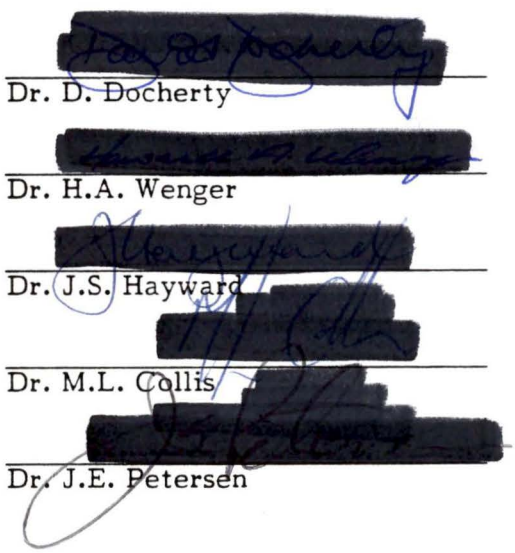
ABSTRACT

The purpose of this study was to determine if a heat acclimation program enhanced the tolerance of fit, young females to work in a heat stress environment. Twenty female subjects were randomly assigned into three different groups. One group acted as a Control (C), while the other two groups exercised one hour/day for 8 days under conditions of either thermal stress (39-40°C, 45%rh) (Heat/Exercise - HE), or the normal ambient environment (15°C, 68% rh) (Exercise - E). Both of the treatment groups maintained euhydration for the duration of the study. The C group were allowed to drink water ad libitum (ad lib). Pre and Post $\dot{V}O_2max$ assessment demonstrated that there was no change in aerobic power for any of the groups throughout the study. In addition, there was no significant difference between the groups in aerobic power ($\bar{x}$ $\dot{V}O_2max$= 52.5 $ml \cdot kg^{-1} \cdot min^{-1}$, SD= 3.9 $ml \cdot kg^{-1} \cdot min^{-1}$). All three groups performed a Pre and Post Thermal Stress Test (TST) at which time heart rate, power output, hematocrit, change in plasma volume, rectal and skin temperature measurements were recorded. The ad lib fluid consumption of the C group resulted in an estimated 900 and 500 ml loss of water in the Pre and Post TST respectively. This corresponds to a 1.4 and 1% dehydration. There was no difference between the groups on any of the thermal indicators, suggesting that the degree of dehydration attained by the C subjects did not result in any greater thermal strain in either

the Pre or Post TST than the euhydrated treatment groups. The heat acclimation program failed to enhance the thermal tolerance of the HE group as demonstrated by the lack of significant differences in the Pre and Post TST results. This suggests that for groups of this fitness level (who are euhydrated), exercising in the heat has little impact on the ability to withstand the thermal stress during exercise in a hot environment. Five of the 20 subjects were unable to complete either the Pre or Post TST. This is a clear representation of thermal intolerance. Examination of the individual data failed to distinguish any one factor which may have contributed to the obvious thermal distress.

Examiners:



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A special thanks to Joan who sparked my interest in pursuing my education; and to Kath who was involved from start to finish - thanks are not enough!

DEDICATION

To my family for all their love, support and encouragement: To Deb who was my inspiration, and to Sand who was without a doubt my favourite subject.

Chapter 1

INTRODUCTION

The capacity for humans to continue prolonged activity is significantly reduced in a hot environment (Fortney et al., 1985; Haymes & Wells, 1986; Nielson et al., 1981; Pirnay et al., 1977; Sawka et al., 1984). The detrimental effects of heat stress on endurance and power occur as a result of the dehydration and hyperthermia associated with exercise in the heat (Brooks & Fahey, 1985; Flynn et al., 1987; Harrison, 1985; Lamb, 1986; Murray, 1987; Noakes et al., 1986; Wenger, 1986; Wheeler et al., 1986).

Individuals exercising in a hot climate are subjected to a relatively greater thermal strain than those working under cool conditions. A double heat load results from the metabolic heat generated through the process of energy production, and the solar heat from the environment (Fortney et al., 1985; Haymes & Wells, 1986; Stolwijk et al., 1977; Wenger, 1986). Thus, the maintenance of internal thermoregulatory homeostasis during exercise in the heat requires that a proportionately greater amount of heat must be dissipated than with cooler ambient temperatures.

Sweating provides a very efficient means of heat loss in a hot, dry climate. Fit individuals have been estimated to lose 1.5-3 litres (L) of fluid in one hour during exercise in heat (Brooks & Fahey, 1985). Since, the human body contains 5-8 L of blood for the average 70 kg male and approximately half of this volume is

fluid, the loss of fluid through sweating creates a problem. A one percent (or more) decrease in body weight as a result of dehydration has been associated with decrements in work capacity and muscle strength (Brooks & Fahey, 1985).

The ability to continue exercise in a heat stress situation depends not only on an adequate circulation of blood to the working muscles, but a sufficient flow of blood from the core to the periphery for heat dissipation (Fortney et al., 1985; Wenger, 1986). Ultimately, the capacity to supply the active muscles and skin with blood depends on the volume available for distribution.

It has been suggested that the thermoregulatory and cardiovascular response to exercise and heat stress is predominantly influenced by fitness level. However, acclimation state and the level of hydration also exert a significant impact on the development of thermal tolerance to exercise in the heat (Buskirk, 1977; Fortney et al., 1985; Haymes & Wells, 1986; Taylor, 1986).

The review by Haymes and Wells (1986) emphasizes the importance of fluid replacement during exercise in a hot environment. They conclude that the maintenance of euhydration (normal body fluid levels, Sawka et al., 1984b) will help to alleviate cardiovascular and thermoregulatory strain. However, they also note that exercise tolerance under thermal stress conditions is affected by the degree of acclimation. To diminish the deleterious effects of exercising in the heat (enhanced cardiovascular and thermoregulatory strain, accelerated onset of fatigue and decrements in performance) athletes should be acclimatized in order to perform optimally (Brooks & Fahey, 1985; Fox, Bowers & Foss, 1988; Haymes & Wells, 1986).

The cardiovascular and thermoregulatory adaptations of individuals who acclimatized by exercising in a hot environment have been well documented. Among these modifications was an increased plasma volume (hypervolemia) (Buskirk, 1977; Fortney et al., 1985; Harrison, 1985; Mitchell et al., 1985; Sawka et al., 1983b; Senay & Pivarnik, 1985). It has been implied that the chronic expansion of plasma volume is the mechanism which allows an individual to continue exercise with less risk of heat strain.

Plasma volume can be increased through acclimatization in 6-8 days. Senay & Pivarnik (1985) consider hypervolemia to be the most important adaptation that occurs during acclimatization. However, Nadel (1977), Nadel et al. (1980), and Sawka et al. (1983a) found that maintenance of normal body fluid levels were as effective as hypervolemia in dealing with heat stress. Maintenance of normal body fluid levels was also found to be as effective as an acclimation program in the ability to complete a 90 minute walk in a hot, dry environment of 45°C and 20% relative humidity (Sawka et al., 1983a).

However, the previous studies have not simultaneously controlled for hydration state or the possible changes in fitness as a result of the acclimation program. Therefore, it is difficult to attribute the improvement in the ability to deal with thermal stress to acclimation, a better fitness level, or hydration state.

The objective of the present study is to monitor exercise, heat acclimation and hydration state in an attempt to establish whether or not heat acclimation is required for optimal performance during exercise in the heat for a fit population of females.

PURPOSE OF THE STUDY

The purpose of the study was to determine if a heat acclimation program enhanced the tolerance of fit, euhydrated females to work in a heat stress environment.

RESEARCH QUESTIONS

1. Is there a difference in the thermoregulatory capabilities between euhydrated subjects who exercised in a hot environment and euhydrated subjects who exercised in normal ambient conditions?

2. Is there a difference in the thermoregulatory capabilities between euhydrated subjects who exercised in normal ambient conditions and subjects who did not exercise and drank water ad lib?

3. Is there a difference in the thermoregulatory capabilities between euhydrated subjects who exercised in a hot environment and subjects who did not exercise and drank water ad lib?

Operational Definitions

Thermoregulation: The mechanism through which humans are able to maintain a relatively constant internal temperature (37 ± .5°C for humans) despite widely fluctuating environmental temperatures (Haymes & Wells, 1986).

Hemoconcentration: The progressive increase in concentration of intravascular constituents arising from continuous loss of fluid (plasma, water) from the intravascular space (Harrison, 1985). Hematocrit will be used to reflect

changes in plasma volume. An increase in the percentage hematocrit suggests a decrease in plasma volume (hemoconcentration).

Hemodilution: The progressive decrease in concentration of intravascular constituents arising from continuous gain of interstitial fluid by the intravascular space (Harrison, 1985). A decrease in the percentage hematocrit suggests an increase in plasma volume (hemodilution).

Hypohydration: Body fluid deficit (Sawka et al., 1984b) indicated by a decrease in body weight initiated by the loss of body fluids via sweat during the testing and training sessions.

Hyperhydration: Body fluid excess (Sawka et al., 1984b) indicated by an increase in body weight as a result of excessive water consumption during the testing and training sessions.

Euhydration: Normal body fluid content (Sawka et al., 1984b). The maintenance of body weight through fluid replacement during the testing and training sessions by the two treatment groups.

Dehydration: Dynamic loss of body fluids or transition from euhydration to hypohydration (Sawka et al., 1984b) indicated by a decrease in body weight during the testing and training sessions.

Voluntary Dehydration: Incomplete fluid replacement ad libitum. The thirst mechanism is not an adequate index of body water requirements. Thirst occurs after a 2 % water deficit, but does not increase in intensity with increasing hypohydration (Sawka et al., 1984b). The Control group were expected to undergo voluntary dehydration as water intake was ad libitum.

Conscious Hydration: Both of the treatment groups (Heat/Exercise and Exercise) were consuming specified amounts of water in order to maintain body weight during both the testing and training sessions.

Hyperthermia: Condition where heat gain exceeds heat loss resulting in a rise in core temperature above normal. The normal range is approximately 37 °C. Temperatures of 40 °C or above are considered hyperthermic (Mitchell et al., 1985).

Acclimation: The term applied to the compensatory changes which occur in the laboratory where the subjects are maintained under controlled conditions (Hoar, 1975). For the purposes of this study, **heat acclimation** will refer to the double stimulus of exercise (cycle at 65% $\dot{V}O_2$max) and heat (39-40 °C, 40-46% rh) one hour/day for 8 days performed by the subjects in the Heat/Exercise group.

Exercise: Refers to the group of subjects (Exercise) who cycled at 65% $\dot{V}O_2$max, one hour/day for 8 days in ambient conditions (15.5°C, 68% rh).

Acclimatization: Refers to the physiological adjustments which occur in response to stimuli which occur naturally in the environment. It is the sum of adjustments which follow repeated and prolonged exposure to natural environmental changes (Hoar, 1975).

Limitiations of the Study

1. Voluntary dehydration is the incomplete replacement of fluid ad libitum. Awareness of the importance of hydration may initiate fluid replenishment prior to the sensation of thirst. As such, the Control group may not sucessfully demonstrate voluntary dehydration which may confound the results.

2. The lack of diet control and hydration techniques at times other than testing might also affect the results of both the treatment groups and the Control group.

3. The 8 day heat/exercise stimulus might be inadequate to elicit an acclimation effect (as indicated by an increase in resting plasma volume and a decrease in core temperature and heart rate for a given load) due to the high level of fitness of the subjects.

4. Blood samples were drawn from the finger via a finger prick which during intense exercise in the heat may create problems if central and peripheral competition arises, and peripheral vasoconstriction occurs.

Chapter 2

RESEARCH METHODS

SUBJECTS

Twenty females volunteered to participate in the study, signed the Informed Consent and were randomly assigned into three groups. One group acted as

a) control (C), while the other two groups exercised for 8 days on a cycle ergometer under;

b) normal environmental conditions (15.5°C, 68% relative humidity - rh) (Exercise - E), or

c) in a hot, dry environment (39-40 °C, 46% rh) (Heat/Exercise - HE). The subject characteristics are presented in Table 1.

Control subjects (n=8) participated in two Thermal Stress tests (TST), prior to and following a three week control period. No information about dehydration or rehydration was provided for the C group. Although water was available for both testing sessions, fluid intake was ad libitum. As such, most of the subjects in this group demonstrated some degree of voluntary dehydration.

During the three week control period, the subjects were asked to maintain their normal, daily physical activity. The assessment of $\dot{V}O_2max$ (pre and post) illustrated whether or not the fitness level of this group changed over the control period.

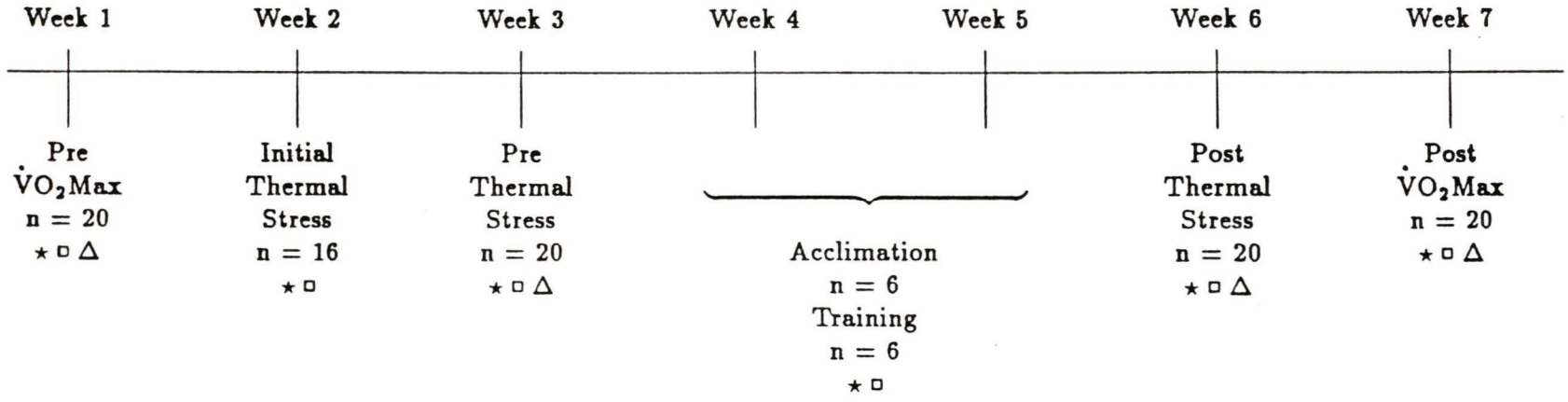
The two treatment groups HE (n=6) and E (n=6) completed both the Pre and Post TST and $\dot{V}O_2max$ tests. In addition, these subjects completed an Initial TST. The Initial TST served as a guideline to determine the degree of dehydration each subject reached while exercising in a hot, dry environment (40°C, 30% rh). The decrease in body weight due to fluid loss, reflected the amount of water intake required to maintain normal body fluid levels. This information was utilized for the Pre and Post TST and the training protocol.

Following the preliminary testing, these subjects participated in an 8 day exercise program (cycle at 65% $\dot{V}O_2max$) in either the heat or normal ambient conditions. Environmental Conditions (temperature and humidity) for both exercise groups are presented in Table 4, Appendix D. Figure 1 outlines the exercise/testing schedule for the three groups.

Note.

During the testing and exercise, attention was directed towards fluid replacement. The amount ingested per subject was determined in the Initial TST. The maintainenance of euhydration was monitored by body weight. Table 3 illustrates the average amount of fluid lost per group per test.

Figure 1. Experimental Protocol Timeline



★ Heat/Exercise - cycle 65% $\dot{V}O_2$max, 39° - 40°C, 46% rh, 1 hr/day, 8 days (n = 6).

▫ Exercise - cycle 65% $\dot{V}O_2$max, 15.5°C, 68% rh, 1 hr/day, 8 days (n = 6).

Δ Control (n = 8).

EXPERIMENTAL PROCEDURES

$\dot{V}O_2$max Protocol

$\dot{V}O_2$max was assessed prior to and following the three to four week control/exercise period. $\dot{V}O_2$max was performed on a Monarch Cycle Ergometer (model 868), using the continuous testing CASS protocol as outlined by MacDougall et al., 1982 (Table 5, Appendix E). The expired air was collected by a Two-Way Rudolph valve and channelled into the Beckman Metabolic Cart (model 1). Subsequent analysis occurred every 30 seconds and values of expired air ($\dot{V}E$), oxygen consumption ($\dot{V}O_2$) and the respiratory exchange ratio (R) were recorded. The heart rate was monitored every minute by a digital read out from the Sport Tester Heart Rate Monitor (model PE 3000).

$\dot{V}O_2$max was reached when one of the following criteria were met; a plateau or decrease in O_2 consumption with increasing workloads (<100 ml per min increase), an R value which exceeded 1.1, maximal heart rate was reached, or the subject stopped due to fatigue.

$\dot{V}O_2$max was obtained from the Beckman Cart printout based on one of the four criteria for determining $\dot{V}O_2$max. $\dot{V}O_2$max was expressed as an absolute value ($L \cdot min^{-1}$), and relative value ($ml \cdot kg^{-1} \cdot min^{-1}$). Power Output (watts) at $\dot{V}O_2$max was also determined as an index of aerobic performance.

Thermal Stress Protocol

Initial Thermal Stress Test: estimation of fluid intake required to maintain euhydration levels

Each subject cycled for one hour in an environment set at 39°C (SD± 1°C) and 44% rh (SD± 5). Temperature and humidity were determined using a Sling Psychrometer (model 11-666) and Relative Humidity Tables (Sybron Corp.). After a five minute warm-up (60 rpm, 1 kp), the load was increased to a predetermined load of 65% $\dot{V}O_2max$. The intensity of exercise was monitored using heart rate. As the heat load increased (a combination of exercise and environmental stress) workload on the cycle ergometer progressively decreased in order to maintain heart rate at 65% $\dot{V}O_2max$.

Heart rate and core temperature (rectal) were monitored every five minutes throughout the 60 minute cycle. In addition, pre and post nude weights were recorded, as well as the amount of water ingested.

Termination of the test occurred after 60 minutes or if core temperature reached 39.5°C, core temperature increased 1.5°C above resting values, heart rate reached 95% maximum, or the subject experienced discomfort or dizziness and wanted to stop.

The difference between pre and post test nude weight was determined. Additionally, fluid intake during the test was noted and subtracted from the final weight to estimate the amount of fluid lost during the test (1 L water = 1 kg). The total amount of weight lost, was used as an approximation for the amount of fluid intake required to maintain euhydrated levels.

Pre/Post Thermal Stress Test

These tests were similar to the Initial Thermal Stress Test with the following exceptions:

1) In addition to core temperature readings (Fisher General Purpose Temperature Probe), skin thermistors (Sensor Medic Skin Electrodes, model SBT-5) were placed on the arm, chest, and thigh and skin temperatures were recorded using a Thermalert Monitor (model TH-69).

2) Blood samples were collected five minutes prior to exercise, after 30 minutes of exercise, and immediately following exercise. One final blood sample was drawn 20 minutes into recovery. After completion of the test, the subject left the heat chamber and sat quietly at room temperature until the final blood collection.

Note.

The C group was provided with water and drank ad libitum, whereas, the HE and E groups were required to drink enough water to maintain body weight throughout the tests. Table 3 illustrates the amount of weight lost and water ingested during the TST's.

Exercise Protocol

The exercise and hydration procedures were identical for both groups. However, the conditions (temperature and humidity) under which these groups worked were different. The E group cycled at ATPS which over the two week period was an average of 15.5°C, and 68% rh. Heat stress was applied to the other group (HE) for the two week period. The mean temperature and humidity was 40°C, and 46% rh respectively.

Both groups were instructed to cycle for one hour at an intensity required to elicit a heart rate (HR) of 65% $\dot{V}O_2max$. The load and revolutions (revs) could vary as long as HR was maintained. It should be noted that the environment in the

heat chamber exerted a significant external heat strain and as such power outputs for the HE group were lower than the E group at a given heart rate. Table 6, Appendix F outlines mean HR, load and revs for each group over the exercise period.

The subjects cycled 8 days in either the heat stress environment or ATPS. The exercise schedule allowed for one to two days rest after three to four days of cycling (as outlined by Senay & Pivarnik, 1985). In addition, each subject had a minimum of 24 hours of rest before completing the final TST.

Blood Sampling Protocol

Blood samples were collected five minutes prior to, during (30 mins), and immediately following exercise. A final sample was drawn 20 mins into recovery. The blood was collected from the finger (finger prick method using a sterilized Auto-lancet as outlined by Allen et al., 1977) into a heparinized microhematocrit capillary tube.

The blood samples were subsequently analyzed for hematocrit (Hct) in order to evaluate any changes which might have occurred with plasma volume. Hct was measured using the Microhematocrit Centrifuge with the Adams Readacrit. No correction was made for trapped plasma. Hct readings were in percentages.

The change in plasma volume was determined with a nomogram developed by van Beaumont (1972) using Hct (Appendix G).

STATISTICAL ANALYSIS

The data were analyzed using a repeated-measures multivariate analysis of variance **(MANOVA)** to determine if there was any difference between the groups in their ability to deal with heat stress and, to investigate the within group differences as a result of the applied treatment. Subsequent post hoc analysis was applied where significance ($p < 0.05$) occurred.

The MANOVA was a **3 X 2 X 6** (group X session X dependent variable) design.

Dependent Variables:

The dependent variables were physiological measures which indicated the degree of thermal strain: work (TW), heart rate (HR), hematocrit (Hct), change in plasma volume (PV), rectal temperature (T_R), and mean skin temperature ($T_{\overline{SK}}$).

Independent Variables:

The independent variables were:

a) Group - Heat/Exercise (HE), Exercise (E) and Control (C) and

b) Session - Pre Thermal Stress Test and Post Thermal Stress Test.

Chapter 3

RESULTS

$\dot{V}O_2max$ and body weight prior to and following the Thermal Stress Tests (TST) are presented in Table 1.

A multiple analysis of variance (MANOVA) revealed that there was no main effect for group or session on $\dot{V}O_2max$ indicating that the fitness level between the subjects was similar and that this fitness level did not change significantly over the course of the study.

A repeated-measures MANOVA was conducted on the 6 dependent variables; total work, heart rate, hematocrit, change in plasma volume, rectal and mean skin temperature in a 3 (group) by 2 (session) by 6 (dependent variable) design to determine if the applied treatment (heat acclimation and the maintenance of euhydration) affects the ability to thermoregulate during exercise in the heat. Subsequent post hoc analysis was performed when significance ($p< 0.05$) occurred.

It should be noted that 5 of the 20 subjects were unable to complete the full hour of exercise during either the Pre or Post Thermal Stress Test (1 subject from the E group in the Post TST, and 4 subjects from the C group; 3 in the Pre TST and 1 in the Post TST). Consequently, the MANOVA was applied to the first 45 mins of exercise only to maintain the subject population (n=20). The final 15 mins of exercise is a descriptive analysis based on the means of the 15 individuals who completed the test. The results of the statistical analysis were as follows:

Table 1: $\dot{V}O_2max$ and Body Weight for the Pre and Post Thermal Stress Tests ($\bar{x} \pm SD$).

		Heat/Exercise n=6		Exercise n=6		Control n=8	
		Pre	Post	Pre	Post	Pre	Post
$\dot{V}O_2max$	$\bar{x}$	52.6	55.6	51.4	54.2	50.1	51.2
$(ml \cdot kg^{-1} \cdot min^{-1})$	SD	3.7	4.4	4.0	3.1	4.7	4.0
$\dot{V}O_2max$	$\bar{x}$	3.1	3.3	3.0	3.1	3.2	3.3
$(L \cdot min^{-1})$	SD	0.1	0.1	0.3	0.3	0.5	0.4
Body Weight	$\bar{x}$	58.2	57.8	57.9	57.9	62.9	62.9
(kg)	SD	3.3	3.1	4.6	4.2	5.0	5.3

Work (TW). TW (the total amount of work done on the cycle ergometer during each TST) is presented in Table 2. Figure 2 illustrates work per unit time (power output in watts). There was no significant difference between groups for TW in the Pre or Post Thermal Stress Test (TST). Additionally, there was no significant difference in TW values within each group between sessions. The first half of the test was characterized by an increase in power output from 44 to 113 watts within 15 min of exercise. However, in order to maintain HR at 65% of $\dot{V}O_2max$, power output was progressively decreased for the duration of the exercise.

Heart Rate (HR). HR values (means and standard deviation presented in Table 2 and Figure 3) were similar for all of the groups on the Pre and Post TST. This was expected since workload was adjusted according to HR. Significant increases in HR were noted within 20 min of exercise, $F(3,114)= 984.50$, $p< 0.001$, after which time HR values were maintained at approximately 160 bpm for the remaining 40 min (see Table 7, Appendix H).

Change in Plasma Volume (PV). PV values are presented in Table 3 and figure 4. No difference between pre and post PV values was demonstrated by any of the groups. However, a between group difference was found for the C and E group in the Pre TST. Post hoc analysis revealed that this significance occurred during the recovery PV recordings $F(2,23)= 4.76$, $p< 0.01$, with the E group having a lower (-8.8%) PV than the C group (-3.6%). No significant effect for time was found (Table 10, Appendix H).

Hematocrit (Hct). Mean Hct values are presented in Table 3 and Figure 5. No significant difference was found between the groups in Hct readings for either the

Table 2: Pre and Post Thermal Stress Test Values for Heart Rate, Work and Rectal and Mean Skin Temperature

		Heat/Exercise		Exercise		control	
		Pre	Post	Pre	Post	Pre	Post
Heart Rate	$\bar{x}$	158	158	161	158	163	156
(BPM)	SD	3.3	1.9	1.1	2.3	0.8	3.9
Rectal Temp.	$\bar{x}$	38.1	38.0	38.0	38.1	38.3	38.5
Final (°C)	SD	.20	.26	.29	.25	.28	.36
Skin Temp.	$\bar{x}$	36.8	36.6	37.4	36.9	37.8	37.2
Final (°C)	SD	.82	1.1	.51	.35	.56	.45
Work (X 10^3)	$\bar{x}$	56.3	56.7	53.8	54.8	51.3	50.4
(joules)	SD	4.7	4.5	6.6	4.6	6.9	8.7

Figure 2: Power Output for the Pre and Post Thermal Stress Tests for the Heat/Exercise (HE), Exercise (E) and Control (C) Group

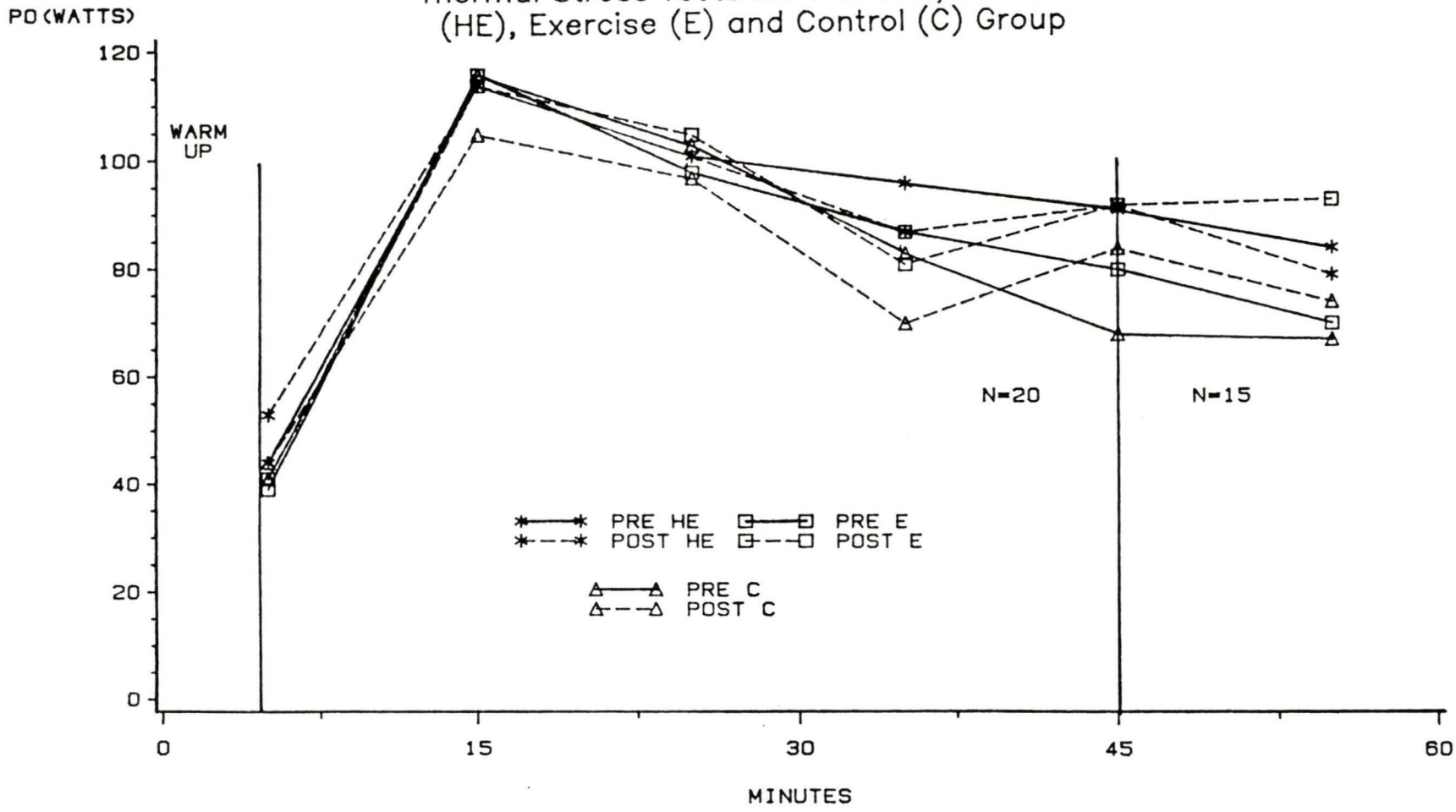
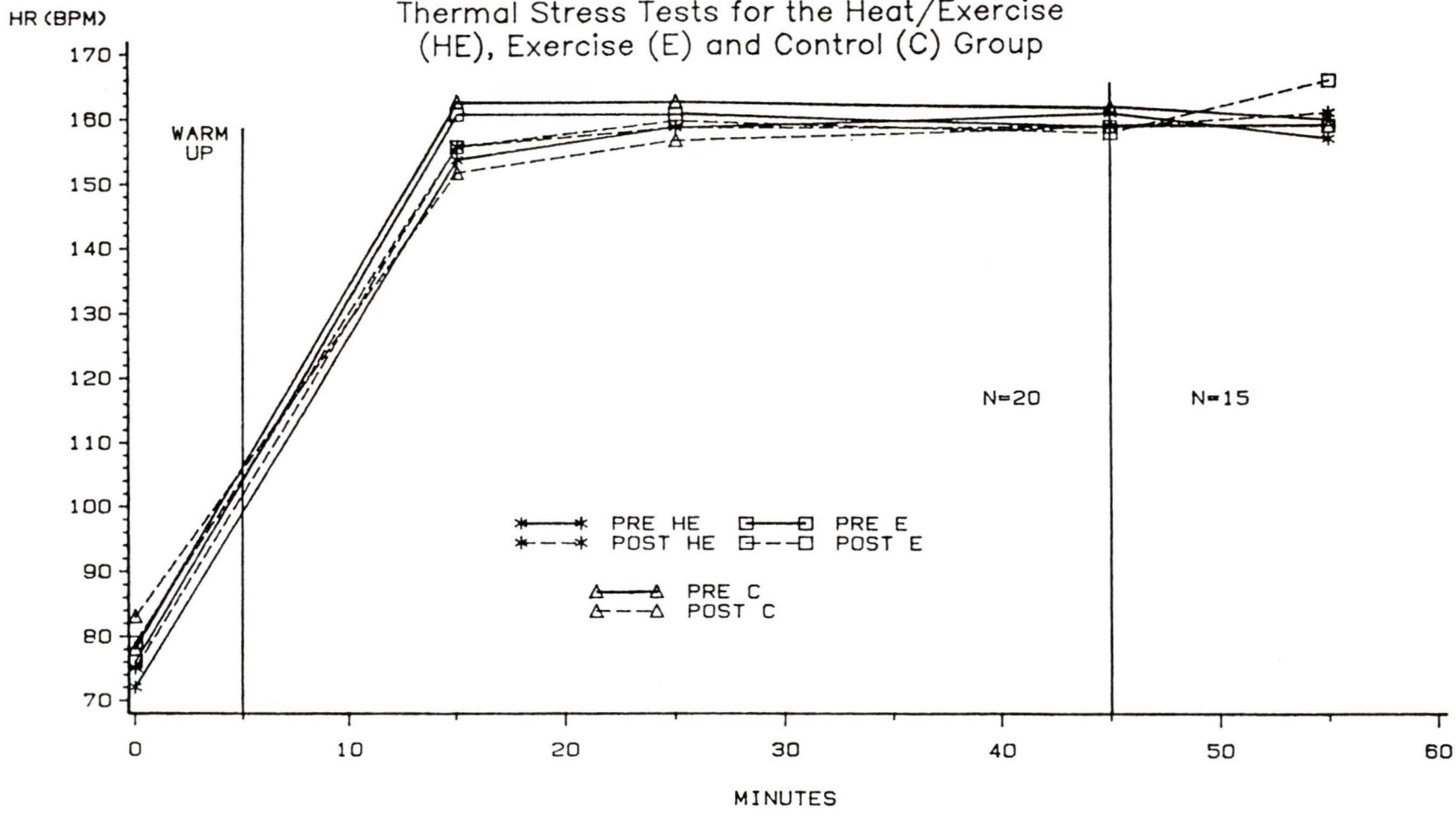


Figure 3: Heart Rate for the Pre and Post Thermal Stress Tests for the Heat/Exercise (HE), Exercise (E) and Control (C) Group



Pre or Post TST. Additionally, there was no difference between pre and post values for any of the groups. Fluctuations in Hct were noted for both the Pre and Post TST for all groups with increases from 40.2% to 42.0%, occurring within 30 min of exercise (Table 11, Appendix H). The subsequent decline in Hct during recovery (40.9%) suggests that blood volume had returned to normal.

Rectal Temperature (T_R) (Table 2, Figure 6). There was no significant difference in T_R between groups during the Pre TST, or within each group between sessions. However, the HE group had significantly lower T_R values than the C group during the Post TST, $F(2,17)= 6.64$, $p< 0.01$. This difference was apparent for all T_R readings (including recordings taken at the start). T_R increased gradually during the test with significant differences from the starting value of 36.7 °C (Time 0) found as early as 30 min into exercise. Final T_R reached 38.3 °C. All of the groups demonstrated significant increases in T_R during 45 mins of exercise in both the pre and post test $F(3,108)= 519.63$, $p< 0.001$ (Table 8, Appendix H). Of the 15 subjects who completed the full hour of exercise, T_R responses were shown to plateau as demonstrated by the non-significant increase from 45-60 mins of exercise. This indicates that these subjects were thermoregulating.

Mean Skin Temperature ($T_{\overline{SK}}$) (Table 2, Figure 7). T_{SK} increased gradually over time with a levelling off (approximately 36-37 °C) during the final 30 min of exercise. Significant increases in $T_{\overline{SK}}$ were demonstrated by the E and C groups, $F(3,108)= 142.70$, $p< 0.001$ during the Pre and Post TST. No significance was found between the sessions, but the HE group had significantly lower $T_{\overline{SK}}$ values during the Pre TST than the C group at every recording including the start $F(2,16)= 6.20$, $p< 0.01$ (Table 9, Appendix H).

Table 3: The Difference Between Resting Values of Hematocrit and Change in Plasma Volume and Body Weight, Fluid Intake and the Percentage Dehydration during the Pre and Post Thermal Stress Test

		Heat/Exercise		Exercise		Control	
		Pre	Post	Pre	Post	Pre	Post
Hematocrit	$\bar{x}$	40.0	38.5	40.5	39.9	41.0	39.5
(%)	SD	3.7	1.8	1.8	1.1	2.4	1.9
Change in Mean Hematocrit (%)		-1.5		-0.6		-1.5	
Change in Mean Plasma Volume (%)		+5.5		+2.0		+5.5	
Body Weight (kg)	$\bar{x}$	-.13	-.23	+.30	+.10	-.90	-.50
Fluid Intake (ml)	$\bar{x}$	1325	1129	1339	1073	413	559
Percentage Dehydration		< 1	< 1	< 1	< 1	1.4	1.0

Note. Hematocrit measures were taken prior to each TST. These values were used to reflect changes in resting plasma volume as a result of the heat acclimation protocol.

Figure 4: The Change in Plasma Volume for the Pre and Post Thermal Stress Tests for the Heat/Exercise (HE), Exercise (E) and Control (C) Group

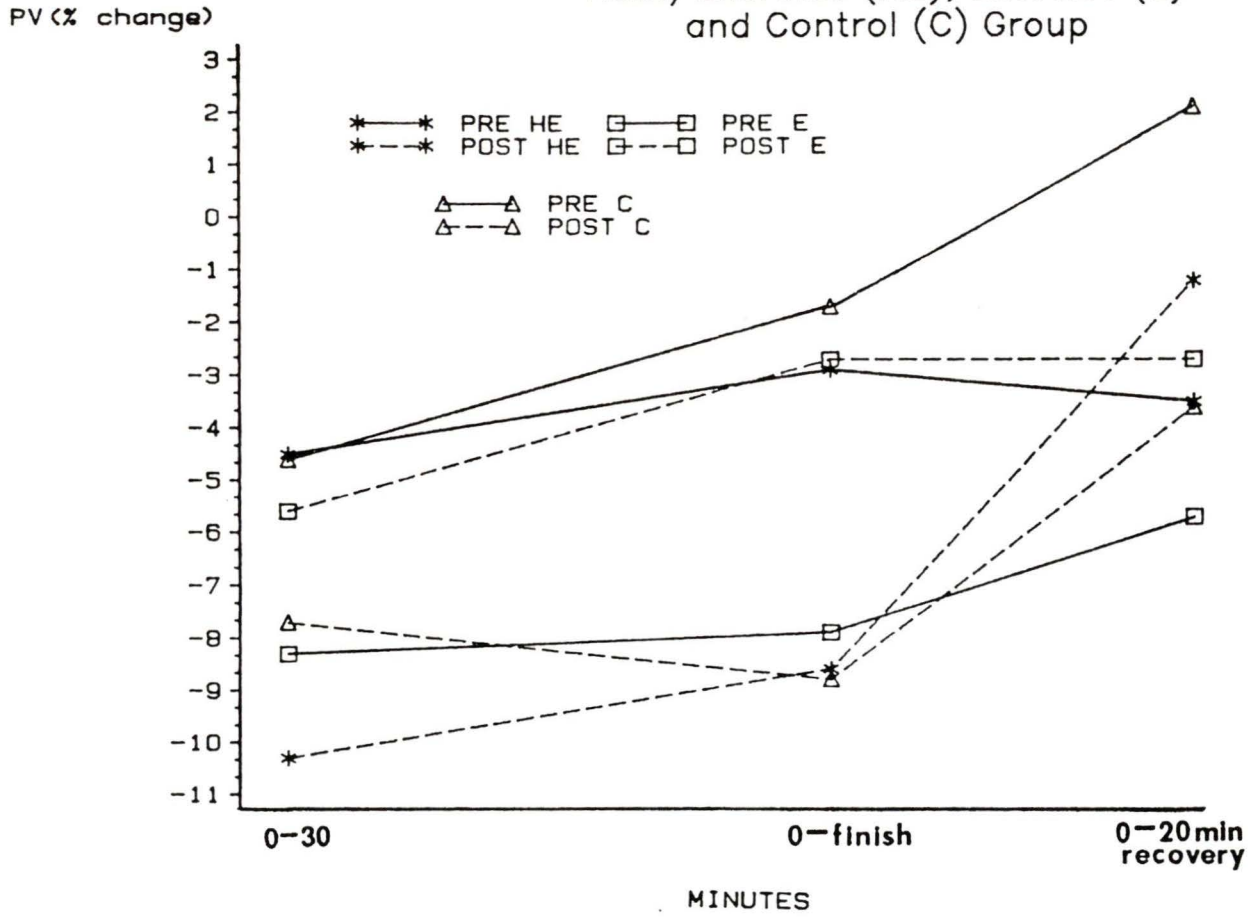


Figure 5: Hematocrit for the Pre and Post Thermal Stress Tests for the Heat/Exercise (HE), Exercise (E) and Control (C) Group

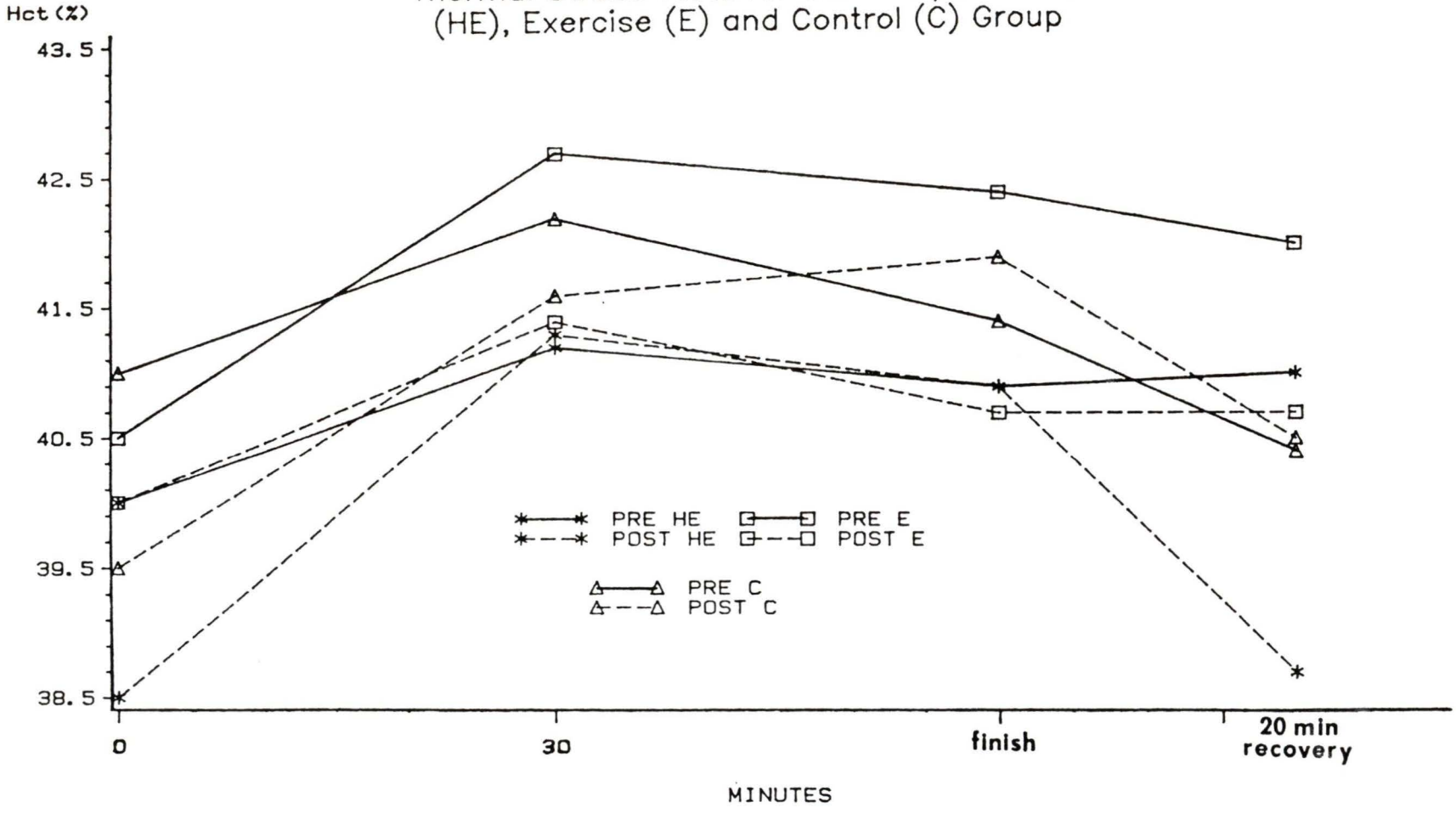


Figure 6: Rectal Temperature for the Pre and Post Thermal Stress Tests for the Heat/Exercise (HE), Exercise (E) and Control (C) Group

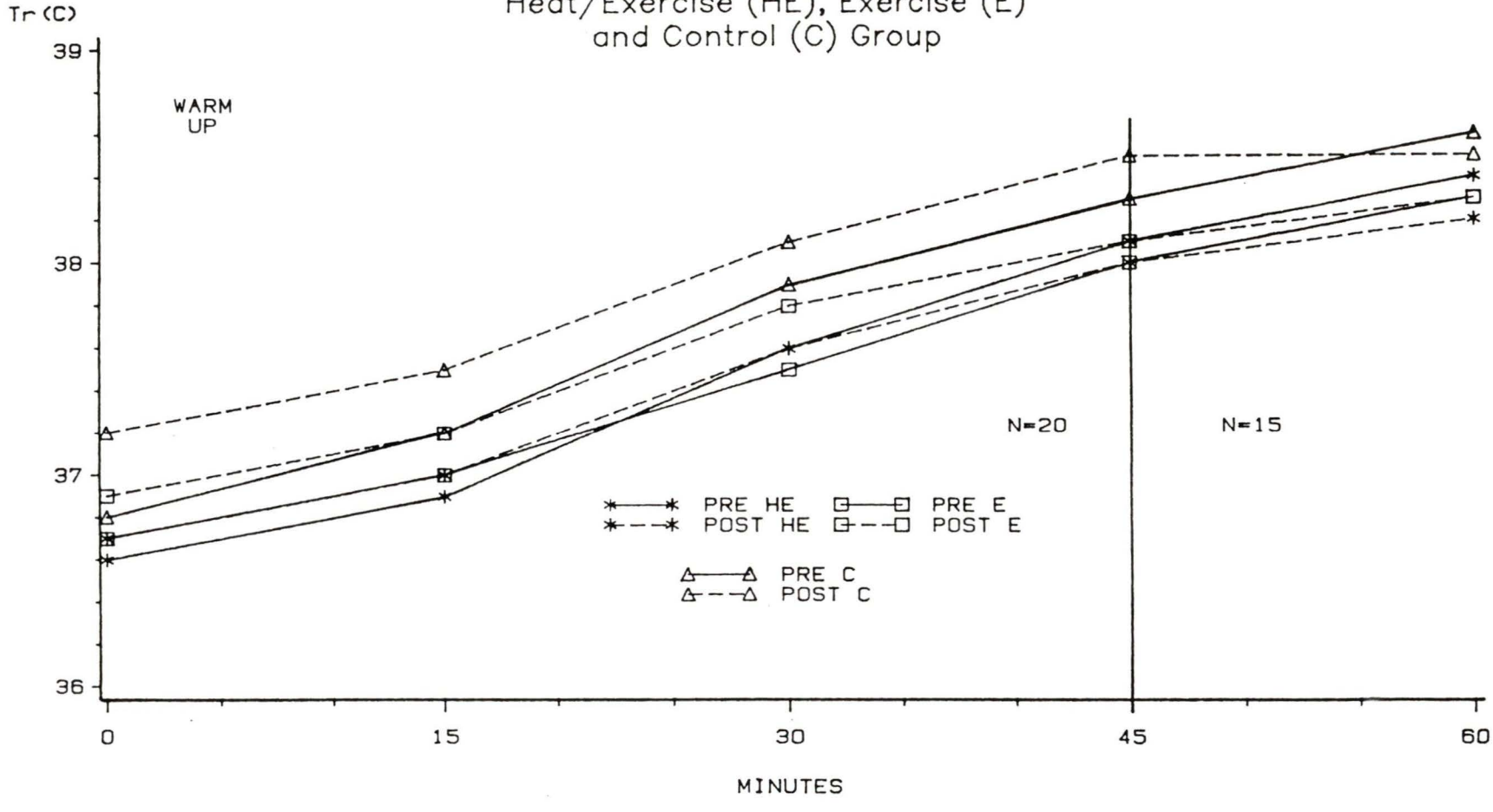
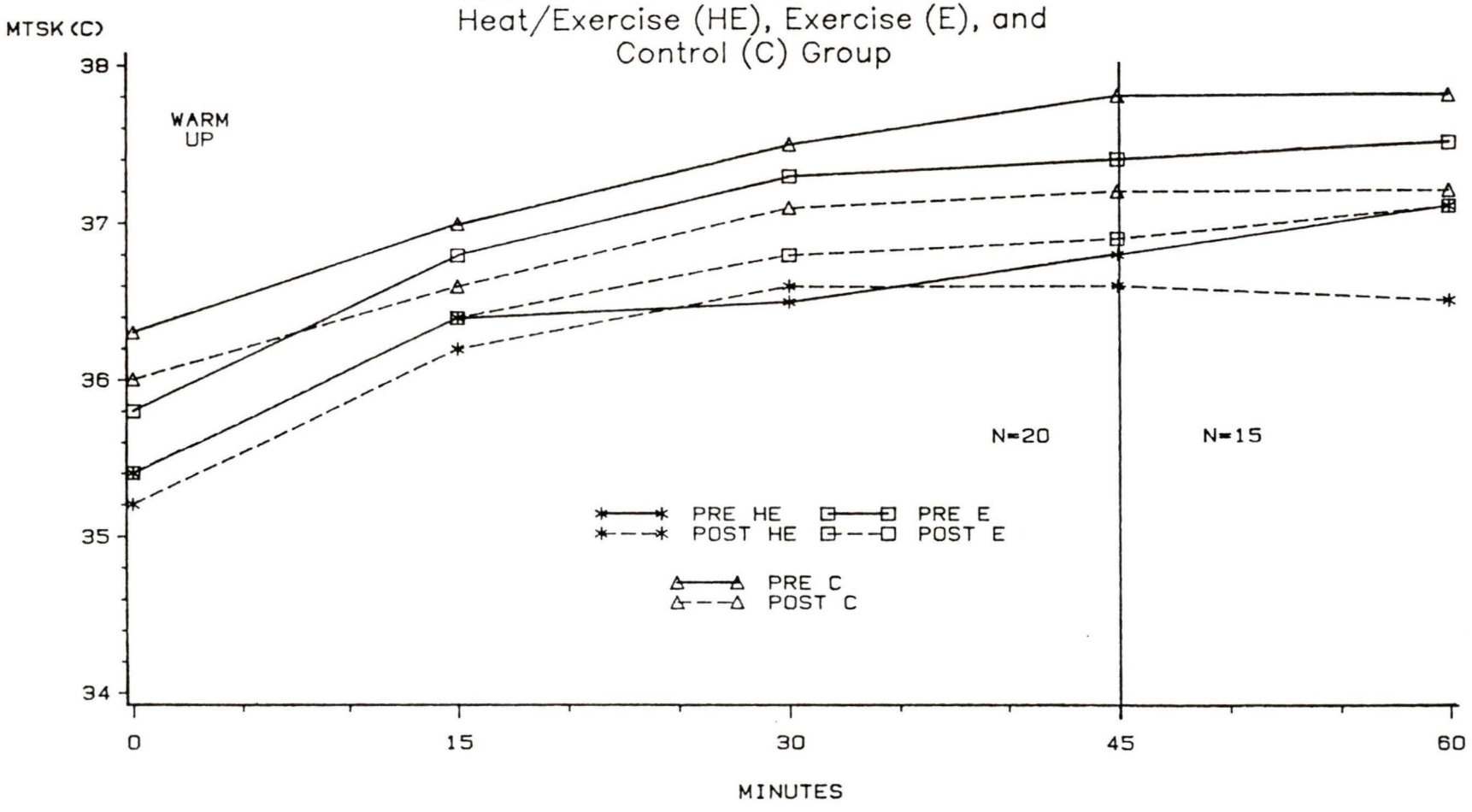


Figure 7: Mean Skin Temperature for the Pre and Post Thermal Stress Tests for the Heat/Exercise (HE), Exercise (E), and Control (C) Group



Chapter 4

DISCUSSION

Previous research has shown that individuals exercising in a hot environment are subjected to a relatively greater thermal strain than those exercising in cooler temperatures (Fortney et al., 1985; Haymes & Wells, 1986; Sawka et al., 1984). Prolonged activity under conditions of heat stress may eventually lead to dehydration. The loss of body fluids, via sweat, has been associated with a decrease in plasma volume and a concomitant increase in cardiovascular and thermoregulatory strain. Inadequate body fluid hinders heat dissipation, metabolic processes within the active muscles, and decreases central blood volume, the combination of which will ultimately interfere with the ability to continue exercising in the heat (Fortney et al., 1984; Haymes & Wells, 1986; Lamb, 1986). This situation is known as thermal intolerance and becomes a critical consideration for individuals exercising in elevated ambient temperatures.

The ability to deal with thermal strain can be affected by fitness level, the state of heat acclimation and hydration status (Buskirk, 1977; Fortney et al., 1985; Haymes & Wells, 1986). Aerobic fitness is considered to be the single most important factor in the determination of thermal tolerance. In general, a higher aerobic power ($\dot{V}O_2max$) is associated with lower core temperatures (T_R) and heart rate (HR), and higher sweat rates and physical working capacity in the heat (Senay & Pivarnik, 1985).

The objective of this study was to determine the effect of a heat acclimation program on the thermoregulatory capabilities of fit females during exercise in the heat. Because fitness level has a significant impact on performance in the heat, any change in aerobic fitness may alter the ability to perform in the heat, thereby confounding the results. Assessment of $\dot{V}O_2max$ prior to and following the testing and exercise protocol monitored any change in aerobic power.

The subjects in the study had a relatively high aerobic fitness level ($\bar{x}$ $\dot{V}O_2max$ = 52.5 $ml \cdot kg^{-1} \cdot min^{-1}$) compared to the average 20-29 year old Canadian female (predictive; $\bar{x}$ $\dot{V}O_2max$ = 35 $ml \cdot kg^{-1} \cdot min^{-1}$, at the 50th percentile, Canada Fitness Survey, 1981). In fact, the $\dot{V}O_2max$ values obtained from the subjects are well above the predicted "excellent" category of 40 $ml \cdot kg^{-1} \cdot min^{-1}$ from the same survey. The lack of a significant difference in $\dot{V}O_2max$ values between the groups on the pre test indicated that the subjects were of similar fitness levels prior to the study. In addition, Post Thermal Stress Test (TST) $\dot{V}O_2max$ measures were comparable to Pre TST values which indicated that the protocol did not significantly effect aerobic power. Therefore, any changes in the ability to perform under the conditions of heat and exercise in the study, would not be attributable to variations in fitness level.

The results will be discussed in terms of between group comparisons in the Pre TST, the effect of the heat/exercise protocol (ie. pre to post session comparisons within each group), and finally, between group analysis during the Post TST. During the Pre TST the two treatment groups were required to maintain body fluid levels through fluid replenishment. The underlying premise was that the preservation of body fluid levels will sustain normovolemia and as such prevent

the development of the cardiovascular and thermoregulatory strain associated with dehydration.

The Control (C) group were not provided with any information regarding fluid replacement, and as such were reliant on the sensation of thirst as the stimulus to drink water. Based on the literature, the thirst sensation does not occur until the individual is at least 2% dehydrated (Sawka et al., 1984). It was assumed that the ad libitum water consumption of the C group would result in inadequate fluid replacement leaving these subjects comparatively hypohydrated. This is considered to be a disadvantage when dealing with heat stress (Fortney et al., 1984; Haymes & Wells, 1986).

The C group did in fact lose an estimated 900 ml of fluid through sweat during the Pre TST compared to a 130 and 300 ml loss by the Heat/Exercise (HE) and Exercise (E) groups respectively. This corresponds to a 1.4% dehydration. In addition, the fluid consumed by the C group (413 ml) was well below the intake values of the HE (1325 ml) and E (1339) groups. Fluid was made available to the C group, but intake was on a voluntary basis. The lack of water consumption suggests that, in agreement with Hecker and Wheeler (1984), Brooks and Fahey (1985), and Sawka et al., (1984) the 1.4% dehydration was not sufficient to stimulate the thirst sensation.

It would seem that the greater percentage dehydration, of the C group would result in a distinct disadvantage while exercising in the heat. Hecker and Wheeler (1984) suggest that thermoregulatory function may be impaired at 2% dehydration, but physical working capacity is not affected until a 3% decrease in body weight. Brooks & Fahey (1985) recommend that even as little as a 1% decrease will have

detrimental effects on performance. If thermal strain develops as a result of the heat/exercise treatment, it would be expected to become apparent predominantly in the C group. However, the degree of dehydration reached by the subjects in this study may have been inadequate to negatively affect the 45 min cycle at 65% $\dot{V}O_2max$ in the heat.

Thermal strain may be evident in either a reduced capacity to continue to generate force, or in uncontrolled elevations in core temperature. Presumably, if body fluid levels drop, plasma volume will be compromised resulting in increasingly greater thermoregulatory, cardiovascular and metabolic costs to maintain a given workload (MacDougall et al., 1974). However, there was no significant difference in the change in plasma volume (PV), T_R and work (TW) measures between the C group and the two treatment groups.

There was no significant difference in PV responses between the groups during the Pre TST. Although the C group had a higher percentage dehydration, this was not reflected in PV. It was expected that the HE and E groups would remain normovolemic throughout the test due to continual water intake. Furthermore, hypovolemia was the expected PV response of the C group as a result of inadequate fluid replacement. However, the pattern and magnitude of PV fluctuations throughout the test were similar between all the groups and could simply be a reflection of fluid shifts between compartments.

Shifts in body fluids, including plasma are the result of changing hydrostatic and osmotic pressures and occur in response to the dynamics of exercise (Buono et al., 1985; VanBeaumont, 1973). The relatively greater drop in body weight by the C group suggests that if PV was to be affected, it would be evident in this group.

This was not the case, and the lack of significant PV changes in the C group suggests that the 900 ml of lost fluid was supplied by sources other than the vascular system. Convertino et al. (1983) and Fortney et al. (1981) suggest that the loss of plasma volume is related to absolute blood volume. During hypovolemia, when it is necessary to maintain cardiac filling pressure, losses of plasma will be minimized. This is in agreement with Buono et al. (1985), Sawka et al. (1984), Myhre et al. (1985), and Van Beaumont (1973), who have suggested that the circulatory system strives to maintain homeostasis and as such, fluid for sweating is initially provided from the interstitial space. As dehydration progresses, an increasingly greater amount of fluid will be drawn from the intracellular compartment with fluid from the vasculature being supplied as a last resort. The level of dehydration attained by the C group was not severe enough to sufficiently compromise blood volume and as such no significant changes in PV occurred during exercise.

There was a significant difference in PV response during the 20 minute recovery period of the Pre TST between the E and C group (-5.9 and 2.1% respectively). These values suggest that the C group returned to normal PV values more readily than did the E group. The greater percentage dehydration of the C group may have provided a stronger stimulus (ie. greater hemoconcentration) to draw fluids back into the vasculature after the cessation of exercise. Because body weight was maintained for the E group, and the percentage dehydration was less than 1, the drive to return to resting PV values may not have been as great, and as such the return to normality may have been delayed.

A decrease in body fluid levels will also affect the ability to transfer heat from the core to the periphery for heat dissipation and as such body temperature measurements are effective indicators of thermal strain. Mean skin temperatures ($T_{\overline{SK}}$), although elevated above resting values, plateaued below the surrounding ambient temperature, suggesting that evapourative cooling was occurring. However, this method of heat loss appeared inadequate to dissipate the metabolic heat generated through exercise. As such, heat production was greater than heat loss and was reflected by the continual rise in T_R up to 45 mins of exercise. Had the duration of exercise in the present study been longer, it is possible that T_R would have reached a steady state. The progressive rise in T_R may simply reflect the fact that the subjects had not yet reached this steady state and does not necessarily indicate thermal intolerance. In fact, from 45-60 mins T_R values levelled off indicating that thermoregulation was occurring.

This is in contrast to the findings of Sawka et al. (1983a) whose subjects were unable to thermoregulate during a 90 min walk (45% $\dot{V}O_2max$) in the heat (45 °C, 20% rh) (Sawka et al., 1983a). Further evidence for a lack of thermoregulatory ability is presented by Armstrong et al. (1988), Nadel et al. (1980), and MacDougall et al. (1974). These studies indicate that there are some conditions under which humans appear to be incapable of regulating body temperature while exercising. Based on the non-significant increase in T_R for the final 15 mins of exercise, the subjects in the present study were able to regulate internal body temperature under the conditions of heat and exercise.

Peripheral shutdown of blood flow occurs in response to a significant reduction of PV. Hecker and Wheeler (1984) suggest that a 2% decrease in body

weight will impair thermoregulatory function. Progressive dehydration restricts body fluid levels such that the volume of distributable fluid available is insufficient to adequately maintain both central and peripheral blood flow. The maintenance of central blood volume is the predominant function and will occur at the expense of thermoregulation if body fluids fall below a critical level (possibly 2% as suggested by Hecker & Wheeler, 1984).

This was demonstrated to an extent by the pre $T_{\overline{SK}}$ values. When compared to the HE group, the C's had a significantly higher $T_{\overline{SK}}$. This suggests that there was less evapourative cooling occurring for the C group. This could have been an indication that because of the relatively greater degree of dehydration of this group, peripheral blood flow (and therefore heat transfer) may have been reduced, resulting in higher $T_{\overline{SK}}$ measures. It should be noted that this trend was not demonstrated in the Post TST and could be possibly due to the 150-300 ml increase in fluid consumption of the C group in the Post TST.

The higher $T_{\overline{SK}}$ in conjunction with the greater percentage dehydration would facilitate a more rapid increase in T_R. However, a higher T_R was not recorded for the C group in the Pre TST. Considering that PV values were similar between groups, it is logical to assume that T_R measures would be comparable. Additionally, even though the C's decreased body weight by .9 kg and fluid intake was minimal, their percentage dehydration did not seem to impair thermoregulation any more than those subjects who maintained body fluid levels (HE & E) under the conditions of this study. According to PV and T_R values, the groups were equally stressed.

The ability to continue exercise in the heat is another indicator of thermal strain and can be quantified by the total amount of work accomplished (TW). There is an inverse relationship between the degree of thermal strain and the ability to generate force (Armstrong et al., 1988; MacDougall et al., 1974). As the level of dehydration progresses, fluid movement within the body is restricted. This can be reflected by the rise in core temperature as previously mentioned, or by a decrease in metabolic function. This impairment of metabolic processes at the active muscle site may occur as a result of extreme muscle temperatures (Martin & Rodwell, 1981; Zubay, 1984), a lack of intracellular fluids (Lamb, 1986; Wenger & Reed, 1976), and/or neuromuscular dysfunction due to a change in osmolality (Bullock & Rosendahl, 1984; Emes & Nowak, 1983). According to Hecker and Wheeler (1984) reduced muscular endurance time occurs as early as a 3% decrease in body weight.

None of the groups reached this level of dehydration, but power output (PO) indicated that as thermal strain increased, the subjects were unable to maintain the initial cycling pace if HR was to be maintained at 65% $\dot{V}O_2$max. The first half of the test was characterized by higher power outputs than the second half of the test. Total work values between all of the groups were similar and is further indication that the 1.4% dehydration of the C's did not incur any greater thermal intolerance than the maintenance of euhydration. These findings are similar to the results of a study by Greenleaf and Castle (1971) who were unable to demonstrate differences in heart rate, core temperature and oxygen consumption during a 70 minute cycle at 49% $\dot{V}O_2$max, 24 °C. Similar to the present study, their subjects were 'hyperhydrated' (increased body weight by 1.2%), and 'ad lib' (decrease in body weight by 1.6%).

However, it should be noted that 4 out of the 8 C subjects were unable to complete the 60 mins of exercise (3 in the Pre TST and 1 in the Post TST). Of any of the thermal stress indicators, this was the most dramatic representation of thermal intolerance. One individual out of the 12 treatment subjects terminated exercise before the hour was up. This would suggest that although the physiological variables (PV, T_R & TW) were unable to demonstrate significant differences between the groups, the fact that half of the C's became dizzy, nauseous and were incapable of finishing the test signifies a greater magnitude of thermal strain.

Close examination of the individual data of these 5 subjects failed to produce any recognizable patterns which could link the failure to complete the test to any one variable. Initially, it was thought that these individuals would perhaps be less fit than the rest of the subject population. This was not the case. Fluid intakes, the decrease in body weight and the percentage dehydration were comparable to the C subjects who were able to complete the test. There appears to be no discernable trend for these subjects. Nothing in their individual data (Table 13, Appendix I), including PV, PO and T_R provides any possible explanation for their seemingly greater thermal strain (note: TW values were lower due to the early cessation ot the test). They were simply unable to continue to exercise under the conditions of heat and exercise. Similar results were reported by Myhre et al. (1982) who studied five runners participating in a marathon (20 °C). Two of the runners were unable to complete the run and a case study of these two individuals revealed that of the measured variables (heart rate, core temperature, body weight, plasma volume, hematocrit and hemoglobin), only hematocrit and

hemoglobin were slightly elevated above the values obtained by the runners who did complete the run. These differences were insufficient to account for the inability to finish the race.

Successful completion of the TST (by those subjects able to complete only one) did seem to distinguish one common underlying characteristic which may have contributed to their success. Once again, T_R, PV, PO, decrease in body weight and percentage dehydration were similar in both tests. However, for four of the subjects who completed the TST, fluid intake increased by 150-300ml. This slight increase seems to be the difference between completion of the test, or early termination due to nausea, dizziness and exhaustion.

It should be noted that the C subjects were expected to voluntarily dehydrate and although fluid consumption was well below the 2 treatment groups, these subjects did not reach a critical level of dehydration. Their intake levels were above what was expected based on the research of Greenleaf et al. (1983), and Sawka et al. (1984b). However, the majority of the subjects in the present study were either physical education students or fitness instructors, and the possibility exists that these subjects were subconsciously aware of the need to replenish fluids. Therefore, the thirst sensation may not have been the factor stimulating fluid intake.

Acclimation to heat has been shown to occur within 2-6 days of exposure to the stimulus and can be demonstrated by decreased HR, T_R and increased PO for a given workload, or an increase in PV under resting conditions (Buskirk, 1977; Convertino et al., 1980; Haymes & Wells, 1986; O'Toole et al., 1983). The HE group could have been expected to acclimate since they were exposed to the

stimuli of heat (39-40 °C) and exercise (65% $\dot{V}O_2max$) for 1 hour per day for 8 days (Senay & Pivarnik, 1985). However, according to the dependent variables of PV, T_R & TW the HE group did not demonstrate acclimation to the heat. There was no significant difference in pre to post test recordings of exercising or resting T_R, HR, PV and in the total amount of work accomplished for any of the groups. Discussion of the HE group in terms of acclimation would be inappropriate due to the lack of significant Pre to Post TST results.

A possible explanation for the absence of an acclimation effect could be associated with the fitness level of the subjects. The fitness level as previously mentioned was high compared to the average Canadian female of the same age. In addition, the subjects $\dot{V}O_2max$ values were generally higher than those of the subjects in the other studies (Sawka et al., 1984a, 1983b; O'Toole et al., 1983; Kirby & Convertino, 1986) whose values ranged from 45 to 53 $ml \cdot kg^{-1} \cdot min^{-1}$. The possibility exists that because of the high fitness level of the subjects in the present study, the heat/exercise stimulus was not of sufficient intensity to elicit any further changes.

Specifically, the PV of these very fit individuals may already have reached a critical hypervolemia strictly through training prior to the study, and that any inducement of further expansion would require a much greater stimulus. Theoretically, PV could have a genetic limit, whereby as the "ceiling" is approached, an increasingly greater stimulus is required to produce increasingly smaller responses. Specific to this study, the exercise and/or heat load could have been manipulated. To control for changes in fitness level, exercise intensity was intentionally inadequate (as demonstrated by the lack of change in $\dot{V}O_2max$

measures) which means than in order to elicit an acclimation response either the intensity or duration of heat stress may have to be increased.

Another possible explanation for the lack of an acclimation effect in the present study could be the result of an oversight in methodology of the previous research of Sawka et al., 1983b. Although fitness level was quantified prior to all testing/training protocols (male and Female $\bar{x}$ $\dot{V}O_2max$= 46 $ml \cdot kg^{-1} \cdot min^{-1}$), post $\dot{V}O_2max$ assessment was neglected. Because fitness level has such a significant impact on the development of thermal tolerance, this failure to monitor aerobic fitness has serious implications. The authors attributed changes in the physiological variables to be the result of manipulations in treatment (hydration level and/or heat acclimation). They neglected to consider the significance of a training program on fitness level and in turn the change fitness level would have on thermoregulatory parameters.

This is especially true for individuals with initially low $\dot{V}O_2max$ measures. The review by Wenger and Bell (1986) cited three different authors (Shepard, 1986; Sharkey, 1970; Wenger & MacNab, 1975) who were able to demonstrate that less stimulus (ie frequency, intensity and/or duration) is required to elicit greater improvements in cardiovascular fitness for individuals who begin at lower levels. Therefore, even if the training intensity was low, enhancements in cardiovascular conditioning may occur as a result of the low initial fitness level.

In summary, the Pre TST results demonstrated that there was no significant difference between groups (for the subjects who completed the test) in their ability to deal with thermal stress. Additionally, the pre and post test analysis revealed that their was no significant difference between sessions for any of the

groups. This indicates that the heat/exercise treatment was unsuccessful in producing an acclimation response in the HE group. The final analysis studied the between group differences in the Post TST.

The Post TST results were similar to the pre TST results except in core temperature recordings between the C and HE group. The C group demonstrated significantly higher T_R throughout the whole post test. This does not appear to be attributable to a better thermoregulatory capacity of the HE group, but rather due to the fact that the C group started with higher T_R (.2°C) and maintained this difference throughout the test. The increase in T_R (1.5°C) was identical for both groups.

The results of this study suggest that for females of this fitness level, training in the heat will have little impact on performance during exercise in the heat as long as euhydration is maintained.

Chapter 5

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APPENDIX A

LITERATURE REVIEW

Thermoregulation is the mechanism through which humans are able to maintain a constant internal body temperature (37 ± .5°C for humans), despite widely fluctuating environmental conditions (Haymes & Wells, 1986). Exercise under ideal conditions of cool air, low humidity, and moderate air flow generally pose no threat to the thermoregulatory mechanism.

The metabolic heat produced by the muscles during exercise is carried via venous drainage to the body core, elevating deep body temperature. Consequently, there is an internal heat load and a thermal gradient develops between the core and the periphery. This temperature gradient facilitates the transfer of deep body heat to the skin by the circulating blood, where it is dissipated to the environment by radiation, convection and/or the evapouration of sweat (Fortney et al., 1985; Haymes & Wells, 1986).

The hypothalamus is responsible for the maintenance of a homeostatic internal environment. Thermal input from the body core and skin receptors are integrated in the hypothalamus which stimulates various effector mechanisms to mediate the conservation or dissipation of body heat (Bligh, 1973; Fortney et al., 1985; Haymes & Wells, 1986).

The anterior hypothalamus monitors heat gain and is therefore important in the control and regulation of heat loss. Afferent information from both the skin and deep body core temperature receptors stimulate firing of the Hi Q10 neurons in the anterior hypothalamus. This information is transferred neurally to the effector mechanisms. Vasodilation and sweating are the two primary effector mechanisms controlled by the anterior hypothalamus (Fortney et al., 1985; Harrison, 1985; Haymes & Wells,1986; Mitchell et al., 1985; Wenger, 1986).

In order to maintain thermal equilibrium, the body must balance heat production by heat loss. Vasodilation in conjunction with radiation and convection and the evapouration of sweat are effective mechanisms to dissipate the internal heat produced during exercise. Therefore, in cooler environments, most humans are able to exercise for extended periods of time because they are able to thermoregulate efficiently.

THERMOREGULATION

Athletes exercising in a hot environment place a double heat stress on the body. Metabolic heat is produced from work and heat is absorbed from the environment. Increased ambient temperatures severely limit the amount of heat lost from the body to the environment since the core to periphery temperature gradient and the skin to ambient temperature gradient are decreased. The rate at which heat is dissipated is directly related to the severity of the temperature gradient. A small thermal gradient reduces heat flow (Fox & Mathews, 1981).

The lack of a temperature gradient restricts heat flow from the core to the periphery, and from the skin to the environment and as such impairs the thermoregulatory mechanism. This situation is conducive to the development of hyperthermia (Fortney et al., 1985; Haymes & Wells, 1986; Wenger, 1986). Mitchell (1985) defined hyperthermia as the rise in core temperature above normal values (greater than 40°C is considered hyperthermic) due to the inability to release body heat; that is, heat production is greater than heat loss.

Exercise involves adjustments of the circulatory system and body temperature to levels above resting. Changes in heart rate at the onset of exercise reflect

these circulatory adjustments. The load placed on the system during exercise in the heat is twofold. An adequate amount of blood flow must be distributed to the working muscles. Blood is also required to transfer heat from the core to the periphery (Harrison, 1985; Johnson et al., 1982; Rowell et al., 1977; Tanaka et al., 1979).

In order to maintain muscular activity during exercise, a large increase in blood flow is required to provide the oxygen (O_2) consumed and to remove the carbon dioxide (CO_2) and heat produced. The elevated blood flow at the active skeletal muscle site is the result of a neural vasodilator drive, controlled by the sympathetic nervous system.

In general, sympathetic activity is associated with the release of adrenergic transmittors (norepinephrine/ epinephrine - NE/E) resulting in vasoconstriction. However, the arterioles supplying the skeletal muscles are unique in that they are innervated by sympathetic cholinergic fibers which release acetylcholine (Ach) as the neurotransmittor. This is similar to the parasympathetic nervous system which also utilizes Ach. Therefore, in this specific instance, the sympathetic system appears to act like the parasympathetic system and causes vasodilation instead of vasoconstriction at the active muscle site (Tortora & Evans, 1986; Vander, Sherman & Luciano, 1975).

The dilation of peripheral blood vessels is brought about by the decrease in sympathetic (adrenergic) firing which inhibits cutaneous vasomotor tone. This enables blood to flow from the core to the periphery redirecting heat away from the essential internal organs. This mechanism, in conjunction with venodilation, extends the transient time of blood flow through the superficial tissues which

facilitates body cooling by the transfer of heat from the warm blood to the cool skin (Fortney et al., 1985, 1983; Haymes & Wells, 1986; Nadel, 1980; Rowell, 1977).

Although vasodilation is an effective means of regulating body temperature, there are two problems associated with it. Vasodilation extends the distribution of circulating blood over two to three times the normal area. During upright exercise maximal skin blood flow levels may reach 2-4 $L.min^{-1}$(Rowell, 1977). This causes a problem as the human body only contains a finite amount of blood (5-8 L in the average adult).

In order to maintain central blood pressure, compensation for the peripheral redirection of blood must be accomplished. As such, blood flow to the inactive muscles and visceral organs drops dramatically. This splanchnic and renal shunt allows redistribution of blood to the areas in need (Brengelmann et al., 1977; Harrison, 1985; Haymes & Wells, 1986; Senay & Pivarnik, 1985). Elevated cardiac output and renal and splanchnic redistribution of blood contribute to skin blood flow. This enhanced peripheral outflow facilitates body cooling (Harrison, 1985; Rowell, 1977).

Another consequence of vasodilation is an increase in heart rate in order to maintain cardiac output. Vaso/venodilation increases the transient time of blood flow through the superficial tissues. High ambient temperatures exert a local heating effect on the blood vessels enhancing dilation. As such, the veins can accomodate more fluid leading to venous pooling of blood (Harrison, 1985; Rowell, 1977).

This peripheral pooling of blood decreases the central blood volume, which reduces stroke volume. In order to maintain cardiac output, heart rate must increase. Therefore, when compared to heart rate values during exercise in cooler environmental conditions, heart rate is elevated for the same workload.

As previously mentioned, sweating is one of the main thermoregulatory mechanisms utilized to maintain thermal equilibrium especially in a hot, dry environment in which radiation, conduction and convection are restricted. Eccrine sweat glands are under direct control of the anterior hypothalamus. The hypothalamus activates the sweat glands via cholinergic sympathetic neurons. Stimulation of the sweat glands result in the secretion of fluid from the surrounding interstitium into the sweat duct (Fortney et al., 1985; Haymes & Wells, 1986).

Initially, sweat, which is an ultrafiltrate of plasma, is isotonic. As the fluid moves through the gland duct, NaCl is absorbed, leaving the emergent sweat hypotonic. This fluid is placed on the skin surface and will only be effective in heat loss if evapouration of the fluid occurs (Fortney et al., 1985; Haymes & Wells, 1986; Mountcastle, 1974; Myhre et al., 1985; Taylor, 1986)

Endurance events are associated with large amounts of water loss from the body via sweating. A hot environment intensifies the level of dehydration. The ability to maintain performance for a prolonged duration is largely dependent upon blood volume; as it decreases, the ability to continue work decreases. Both physical activity and temperature extremes can alter blood volume. (Mitchell et al., 1985).

THERMAL INTOLERANCE

A hot environment diminishes the human capacity for endurance events. The reduced exercise tolerance is in part due to the loss of body fluids. Many researchers suggest that the decrease of blood volume, brought on by dehydration, serves to enhance cardiovascular strain and heat storage (Candas et al., 1986; Fortney et al., 1984; Haymes & Wells, 1986; Lamb, 1986). Dehydration is exacerbated by sweating which is the primary route of heat dissipation during exercise in the heat.

The greater the intensity and duration of exercise, the greater the thermal load and consequently, the greater the perspiration rate. Elevated ambient temperatures exaggerate the thermal stress, so a greater amount of heat must be dissipated. Consequently, a proportionately greater amount of water is lost (Brooks & Fahey, 1985; Candas et al., 1986; Hecker & Wheeler, 1984; Lamb, 1987).

During exercise in the heat, sweat output often exceeds water intake causing hypohydration which results in a loss of total blood fluid . Compounding this problem is the retention of water at the active muscle site; both of these mechanisms cause the blood to become very concentrated (Fortney et al., 1983; Nielson, 1986).

The intracellular space is separated from the extracellular space by a water permeable cell membrane which allows fluid exchange between the compartments. The exchange of fluid will depend on osmotic and hydrostatic pressures. Water moves from areas of low ionic concentration to areas of high ionic concentration and thus because the blood has a high osmolarity, there is a trend for movement of water out of the extracellular fluid compartments, into the blood vessel. In most

exercise/heat situations water for sweating is provided by the extracellular compartment. (Buono et al., 1985; Sawka et al., 1984; Van Beaumont, 1973). It should be noted that fluid movements are strictly regulated by the demands placed upon the tissues at any given time and are subject to change as cell requirements vary.

In the situation of severe dehydration, changes in intracellular volume have been noted. Initially water is drawn from the extracellular space into the blood vessel to counteract the hemoconcentration. As the plasma osmolarity increases, (due to the continual loss of fluid via sweat), more water is absorbed from the extracellular compartment making it increasingly hyperosmotic (Myhre et al., 1985; Van Beaumont, 1973).

Eventually, the high osmolarity in the extracellular compartment will elicit fluid movement from the intracellular compartment to the extracellular space. The loss of fluid from the cell, as seen by a decrease in cell volume, indicates cell dehydration (Green et al., 1984; Harrison, 1985; Myhre et al., 1985; Sawka et al., 1984; Van Beaumont, 1973).

The opposite is true for the cells within the active muscle site. Unlike the tissues not directly involved in exercise which may become progressively dehydrated, cells at the active muscle site retain water compounding the problem of hypovolemia. A linear relationship has been found between lactate accumulation and increased water content in muscles after exhaustive exercise. The increased acidity within the muscle tissue draws water into the cells via osmosis and filtration (Lundvall et al., 1986; Myhre et al., 1985; Nielson et al., 1986).

A decrease in body fluid supresses the sweat response and in doing so inhibits the thermoregulatory response. This facilitates heat storage within the exercising muscle and body core. Elevations of body temperatures beyond 39-40°C are generally accompanied by an abrupt decline in biological processes (Armstrong et al., 1985; Costill et al., 1985; Martin & Rodwell, 1981; Segal et al., 1986; Zubay, 1984).

Dehydration is also associated with transportation problems. The decline in blood volume (as a result of body fluid loss), leads to increased blood viscosity and decreased blood pressure. Consequently, a restriction to blood flow has been incurred. This interferes with the delivery and removal of nutrients and waste products to and from the cell (Hecker & Wheeler, 1984; King et al., 1985; Lamb, 1986; Wenger & Reed, 1976).

Additionally, diminished body water interferes with the fluid and electrolyte balance, initiating a chain of events which are deleterious to normal physiological processes. Because sweat is hypotonic to plasma, pronounced sweating leads to a progressive hemoconcentration. Plasma and cellular hypertonicity affects cellular function, neuromuscular transmission and fluid movement (Bullock & Rosendahl, 1984; Emes & Nowak, 1983; Goldberg, 1981; Tortora & Evans, 1986). The lack of water is considered to be a significant factor mediating the onset of fatigue in a thermal stress situation (Brooks & Fahey, 1985; Costill et al., 1986; Green, 1987; Kozlowski et al., 1985; Lamb, 1986; Young et al., 1985).

CARDIOVASCULAR RESPONSE

Blood volume appears to be the primary stimulus of cardiovascular and circulatory responses. During prolonged exercise in the heat, total blood volume may be compromised. In addition, as the core temperature increases during exercise, larger portions of blood volume are distributed to cutaneous vessels, thus effectively reducing cardiac return and central blood volume (Fortney et al., 1985, 1983; MacDougall et al., 1974; Nadel, 1980).

Competition between thermoregulation (peripheral direction of blood flow) and central venous pressure (internal direction of blood flow) intensifies, and the body must decide which of the two mechanisms is more important. The necessity to maintain blood flow to the essential organs supercedes the thermoregulatory response, suggesting that the control of central venous pressure is the more predominant response (Brengelmann et al., 1977; Fortney et al., 1983; Sawka et al., 1983).

Hypovolemia is the stimulus which alters atrial baroreceptor activity and thus affects afferent input to the hypothalamus. A decrease in the atrial filling pressure stimulates vasoconstriction at the periphery. The decision of the body to maintain central blood volume (via peripheral vasoconstriction) has been demonstrated by the decreased forearm blood flow, and is known as the critical juncture (Armstrong et al., 1985; Brengelmann et al., 1977; Fortney et al., 1985, 1983; Harrison, 1985).

In addition, the loss of fluid results in an increased blood osmolality. Hypertonicity triggers hypothalamic osmoreceptors (Harrison, 1985). Hemoconcentration in conjunction with a lower blood volume acts to suppress the

sweating response thereby facilitating water conservation. The modification of the thermal response for both the control of skin blood flow and sweat gland activity precipitates internal heat storage (reflected by the rise in core temperature) (Convertino et al., 1983; Coyle et al., 1986; Fortney et al., 1985, 1983; Sawka et al., 1984).

The cardiovascular system is also affected by hypovolemia. The loss of blood volume reduces ventricular volume and is directly related to the decrease in stroke volume. In order to maintain cardiac output, the heart rate must increase. Therefore, the physiological responses to exercise in the heat (decreased blood volume and hemoconcentration), are increased heart rate and core temperature, and decreased skin blood flow and sweat rate. These mechanisms are responsible for water conservation and therefore the maintenance of normovolemia (normal blood volume) (Armstrong et al., 1985; Candas et al., 1986; Nielson et al., 1986; Sawka et al., 1984). The shutdown of thermoregulation results in uncontrolled elevations in internal body temperature which will rapidly bring about the cessation of exercise (Candas et al., 1986; Convertino et al., 1987; Haymes & Wells, 1986).

DEVELOPMENT OF THERMAL TOLERANCE

1) HEAT/TRAINING ACCLIMATIZATION

The dehydration and hyperthermia associated with exercising in a hot environment can be largely attributed to a loss of body fluids with a corresponding drop in plasma volume. Heat acclimatization and physical training have both proven to be effective mechanisms to increase plasma volume over time. The

mechanisms by which plasma volume increases differ slightly, but the end result is the same; less cardiovascular and thermoregulatory strain.

The acute response to either physical activity or exposure to heat is a drop in plasma volume. However, the combination of exercise and heat results in a much larger drop in plasma volume with a correspondingly exaggerated hemoconcentration over and above what is seen independently with heat or exercise. Conversely, the largest plasma volume expansions are demonstrated when individuals acclimatize by exercising in the heat (Green et al., 1984; Harrison, 1985; Senay & Pivarnik, 1985; Stolwijk et al., 1977). Independently heat stress and exercise are not as effective in producing the adjustments elicited by the double stimulus of training in the heat (Avellini et al., 1982; Haymes & Wells, 1986; Senay & Pivarnik, 1985; Stolwijk et al., 1977).

The mechanism through which plasma volume expansion occurs has been attributed to the ability of the vasculature to retain proteins (Avellini et al., 1982; Harrison, 1985; Senay & Pivarnik, 1985). The intravascular protein mass (IVPM) is responsible for the water-binding capacity within the intravascular space. A positive linear relationship exists between plasma volume and IVPM. Trained individuals demonstrate higher plasma volumes due to the significant elevation in IVPM (Rocker et al., 1975; Senay & Pivarnik, 1985).

During the first few minutes of prolonged submaximal work, Harrison (1985) found that the primary hemoconcentration corresponded with a slight reduction in plasma proteins. However, if no further hemoconcentration occured (ie. no heat stress applied), the IVP concentration could be maintained, if not slightly elevated.

The addition of thermal stress, will result in a significant loss of IVP. During exercise, the primary route of plasma protein loss is through the muscle capillaries. If the ambient temperature is elevated, cardiac output to the periphery increases, and proteins leave the vasculature through cutaneous capillaries in addition to the outward flow at the active muscle site (Harrison, 1985; Senay & Pivarnik, 1985). Therefore the secondary hemoconcentration seen with heat stress is accompanied by a progressive reduction in IVP. The cessation of exercise/heat attenuates the loss of plasma proteins from the vasculature (Harrison, 1985; Senay & Pivarnik, 1985).

Researchers have observed significant and progressive increases in plasma volume during the first six days of heat exposure. This plasma volume increase was accompanied by an increase in total circulating proteins (Avellini et al., 1982; Harrison, 1985; O'Toole et al., 1983; Sawka et al., 1983; Senay & Pivarnik, 1985).

The method of IVP augmentation following exercise in the heat has not been well established to date. Several possibilites have been discussed including; increased delivery of proteins from the interstitial space via the lymphatic system (Harrison, 1985; Myrhe et al., 1985); the delivery of proteins directly from the liver into the IV space (Harrison, 1985; Myhre et al., 1985); and passage of proteins from the splanchnic region into the sinusoids of the liver to be released when hepatic flow increases at the cessation of exercise (Harrison, 1985). Despite the fact that the mechanism of plasma volume expansion following exercise/heat training sessions has not been clearly elucidated, hypervolemia is a well-documented response (Avellini et al., 1982; Harrison, 1985; Myhre et al., 1985; Senay & Pivarnik, 1985).

Avellini et al. (1982) and Harrison (1985) suggest that the ability to increase intravascular proteins is not as efficient with training as it is with heat acclimatization. Convertino et al. (1980) and Senay & Pivarnik (1985) found the exact opposite effect, citing a larger increase in plasma volume with training as well as increased plasma protein concentration. Convertino (1980) suggested that the hypervolemia induced by training is accompanied by hyperproteinemia. Since water binds to the protein an increase in protein content would correspond with an increase in water content.

Harrison (1985) stated that although heat stress elevates lymph flow, exercise provides a stronger lymphatic stimulus due to the action of the muscle pump. As such, it would seem that exercise stimulates a greater return of protein to the circulation via the lymph system. This observation seems to contradict Harrison's hypothesis of a more efficient plasma protein replenishment with heat acclimatization.

Several researchers have cited that 40% of the hypervolemia induced by exercise training can be attributed to thermal stimulus. The remaining 60% appears to be contributed by additional factors related to exercise (Convertino et al., 1980; Green, 1984; O'Toole, 1983; Senay & Pivarnik, 1985). This suggests that exercise does indeed induce higher plasma volume responses than heat acclimatization.

The non-thermal factors which may contribute to the conservation of body fluids are: a faster, more efficient distribution of blood flow through body regions, with increased skin blood flow; increased stroke volume, decreased heart rate and thus maintenance of cardiac output with less effort; more efficient

uptake and utilization of oxygen at the muscular level; reduced energy cost for the same absolute work load; increased sweat rate at a lower core temperature, and; more efficient sweat gland activity (Avellini et al., 1982; Harrison, 1985; Haymes & Wells, 1985; Nadel et al., 1977).

The supercompensatory changes that occur in response to the double stimulus of heat and training, appear to be a result of the acute reductions in plasma volume and increases in osmolarity that are elicited from exposure to these stressors. The elevation in plasma protein content promotes an increased uptake and retention of the plasma during the recovery interval between acclimatization sessions (Green et al., 1984; McKeever et al., 1987). The significance of this increased fluid retention seen with heat acclimatization, training, and heat/training acclimatization means that there is a greater amount of fluid available for distribution and therefore circulatory and thermoregulatory homeostasis can be maintained.

The expansion of plasma volume may also be influenced by hormonal input, specifically the action of aldosterone and arginine vasopressin (AVP). Repeated elevations in renin and AVP activity may develop a chronic enhancement of water retention within the vasculature and other tissues (Avellini et al., 1982; Green et al., 1984; McKeever et al., 1987).

Both of these hormones are involved in the longterm maintenance of blood volume and are thought to be involved in the hypervolemic response elicited by training in the heat (Harrison, 1985). The method of augmentation is unclear, but Harrison proposes two possible methods: the more efficient retention of proteins (already discussed), leading to an oncotic expansion of plasma; or the increased

retention of electrolytes leading to an osmotic expansion. Senay & Pivarnik (1985) expand upon this concept, suggesting that elevations in plasma volume during training are the result of increased plasma proteins. However, heat acclimatization expands plasma volume through an increase in the ionic content within the vasculature. This suggests that the response of plasma volume to heat acclimatization may be substantially influenced by hormonal input. This is supported in part when the effect of heat acclimatization on sweat gland activity and the significant role that aldosterone and AVP play in sodium retention are considered (Kirby & Convertino, 1986; Senay & Pivarnik, 1985).

Kirby & Convertino (1986) have demonstrated significant decreases in sodium output by the sweat gland as an adaptation following prolonged exposure to thermal stress. In fact, heat acclimatization produced substantial reductions in sodium loss during exercise in the heat despite the elevated sweat rate. This may be the result of the increased capacity for sweat dilution (due to enhanced glandular reabsorption of sodium).

Through further investigation, Kirby & Convertino (1986) were able to establish a relationship between sodium sweat secretion, and aldosterone; a decrease in the concentration of sodium in sweat corresponds to a concomitant rise in circulating aldosterone. The greater sodium conservation responsiveness to a given level of aldosterone has been associated with chronic, intermittent elevations in core temperature. Therefore, sweat dilution may involve increased eccrine gland responsiveness to aldosterone.

The enhanced sodium retention found within the kidney and sweat gland serves to increase the hypertonicity within the vasculature and the extracellular fluid.

During recovery, the elevated ionic concentration would mediate a proportionately greater osmotic concentration gradient. Because water passively follows sodium, there would be a more pronounced drive by the tissues and the vasculature to absorb and retain water (Vander, Sherman & Luciano, 1975).

Heat acclimatization and physical training have been associated with additional changes in sweat gland activity. These modifications include; an elevated sweat rate, heightened sudomotor sensitivity, reduced sweat thresholds, and a reduction in the sweat onset time (Avellini et al., 1982; Harrison, 1985; O'Toole et al., 1983; Sawka et al., 1983; Taylor, 1986).

The elevated sweat outputs seen with heat/training acclimatization are thought to be a combination of central and peripheral adjustments. Physical training mediates changes in the rate of sweating. Thus for a given core temperature, the sweat rate is increased, which allows heat to be dissipated more rapidly (Senay & Pivarnik, 1985; Stolwijk et al., 1977; Taylor, 1986).

Conversely acclimatization to heat shifts the sweat threshold to a lower core temperature. As such, the onset of sweating begins earlier. This modification to heat exposure is accompanied by a concomitant elevation in skin blood flow (Senay & Pivarnik, 1985; Stolwijk et al., 1977; Taylor, 1986). Therefore, with training in the heat, the initiation of sweat will begin at a lower core temperature and proceed at a faster rate. Heightened sweat outputs may also be explained by: the elevation of sudomotor sensitivity, which may result in a proportionately greater sweat response to a given neural drive; inhibition of the hidromeiotic effect (sweat supression in the presence of skin wettedness), leading to a greater sweat output (Stolwijk et al., 1977; Taylor, 1986); a more even distribution of sweat over

the skin surface. The majority of sweat occurs in the trunk region of the unacclimatized individual. Heat acclimatization is accompanied by a greater sweat output in the limbs (Stolwijk et al., 1977); it has also been suggested that elevations in the sweat response may be due simply to gland training. Repeated exposures to heat may enhance eccrine efficiency, permitting greater flow, earlier onset, and apparently greater sensitivity (Taylor, 1986). Although the mechanisms of increased sweat outputs are not conclusive, enhanced plasma volume and sweat output responses are both effective mechanisms of body cooling. In fact, sweat outputs ranging from 1.5-4 $L.hr^{-1}$ have been cited with elevated plasma volumes (Avellini et al., 1982; Brooks & Fahey, 1985).

Senay and his colleagues (Harrison, 1986) state that hypervolemia is the single most important adjustment to heat/training acclimatization. The advantages which have been associated with plasma volume expansion are; increased sweat output, decreased heat storage and decreased core temperature at a given absolute steady state work load; decreased heart rate; increased stroke volume, and; increased maximal oxygen uptake (Convertino et al., 1980).

These conclusions were based on previous research which focused primarily on the comparison of hypervolemic versus hypovolemic individuals exercising in a hot environment. Considering the extensive documentation on the deleterious effects of dehydration and/or hypovolemia on subjects performing a task in the heat, it is not suprising that hypervolemia not only alleviates thermal and cardiovascular strain, but is considered to be the underlying stimulus facilitating thermal tolerance.

However, the recent trend in research manipulates blood volume levels, and investigates the possibility that normovolemia is as effective as hypervolemia in dealing with heat stress. Sawka et al. (1983) suggests that although hypervolemia may facilitate lowered heart rate responses, it does not improve thermoregulatory function. These conclusions were based on research comparing hypervolemic and normovolemic subjects. It was found, that despite the elevated plasma volume seen with acclimated hypervolemic individuals, both groups reacted similarily to the thermal strain. The increased plasma volume did not lower core temperature or improve performance for a given workload. In fact, if blood volume is maintained throughout a heat/training regime, there is no evidence to suggest that the body will undergo anymore undue stress than when blood volume is increased. Thermoregulatory and cardiovascular strain was evident only when blood volume was compromised and the individuals became hypovolemic (Sawka et al., 1983).

These results indicate that perhaps the critical element to be considered during exercise in the heat, is the maintenance of normal blood volume. If this is true, there is a distinct possibility that the improved performances seen with acclimated individuals exercising in the heat (Convertino et al., 1980; Harrison, 1986; Haymes & Wells, 1986; Nadel et al., 1977), are linked with factors other than plasma volume expansion.

2) HYDRATION LEVEL

Exercise dehydration can lower plasma volume by 16-20%. The consequent hypohydration has been shown to limit performance during exercise in the heat due to a lack of body fluids and the corresponding increase in thermal strain. This problem can be compounded by the process of voluntary dehydration (Candas et al., 1986; Convertino, 1987; Fortney et al., 1985; Haymes & Wells, 1986).

The thirst mechanism responds to a 2% water deficit which places the body in a state of dehydration before fluids may be consumed. As such, most athletes undergo voluntary dehydration and therefore suffer from inadequate fluid replacement (Sawka et al., 1984). This thermal strain may be alleviated by maintaining normal body fluid levels (euhydration).

Initially the research indicated that thermal tolerance derived from training in cool environmental conditions was inadequate for exposure to severe environmental heat stress. Gisolfi concluded that "distance runners in training for several years appear to be acclimatized for two hours of mild work in dry heat" (Buskirk, 1977).

Heat/training acclimatization is an effective means to increase plasma volume gradually over time (approximately six days - Senay & Pivarnik, 1985). Immediate plasma volume maintenance may be accomplished by hydration. The intake of fluid before, during and after an event has been shown to maintain plasma volume, and decrease the thermoregulatory and cardiovascular strain associated with dehydration (Convertino,1983; Haymes & Wells, 1986; Nadel, 1980, 1977).

If, as the literature now seems to indicate, the maintenance of plasma can be accomplished simply by the intake of water, and that normovolemia is just as successful as hypervolemia at temperature regulation, acclimation may be an unnecessary process in preparing individuals for exercise in the heat.

The literature establishes a strong relationship between improved performance during exercise in the heat and acclimatization (Convertino et al., 1980; Harrison, 1986; Haymes & Wells, 1986; Nadel et al., 1977). A further

association links this improvement to hypervolemia and a more efficient sweat response (Convertino et al., 1980; Haymes & Wells, 1986; Senay & Pivarnik, 1985). Harrison (1986) states that although the intravascular volume increases with heat acclimation and training, the physiological significance of hypervolemia in terms of enhancing exercise capacity remains unclear. As such, it would appear that further investigation is required in this area in an attempt to determine the role of acclimatization, hydration, and exercise in the development of thermal tolerance.

APPENDIX B

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APPENDIX C

INFORMED CONSENT & PAR-Q

In order to assess physiological functions the following laboratory tests will be performed.

Lab Initial _____ Subject Initial _____

Submaximal Cardio-vascular Function
You will exercise on a cycle ergometer up to 75% of predicted maximum heart rate (HR). The following indicated variables will be measured.

A)Thermoregulatory responses:
1) Rectal Temperature ___

2) Skin Temperature (3) ___

3) Heart Rate ___

B) Circulatory Responses
Blood samples will be assessed by peripheral sampling (finger prick) and analyzed for:
1) Hematocrit ___

2) Hemoglobin ___

C) Maximal Cardio-Respiratory Function
You will exercise on a cycle ergometer with progressively increasing loads to elicit maximal response in the following indicated variables:
1) Oxygen Consumption ___

2) Heart rate ___

3) Ventilation ___

Blood samples will be drawn with a minimal amount of discomfort. Strict protocol will be followed to ensure sterility of the subject, examiner, and equipment.

Lab Initial _____ Subject Initial _____

Tests will be administered by qualified personnell under the direct supervision of the investigator.

Lab Initial _____ Subject Initial _____

Tests results will be treated in a confidential manner and used only to describe group responses.

Lab Initial _____ Subject Initial _____

While it is highly unlikely that a subject should

be injured or taken ill during a test or training session. lab personnel are trained in emergency procedures and emergency equipment is on-site at all times.

_____ _____ All laboratory activity will be completed proximal to medical and/or paramedical assistance.

_____ _____ The maximal exercise loads imposed will not exceed those which might be expected of an individual during sports performance.

_____ _____ The heat stress imposed will not exceed that which might be expected in a normal summer environment.

I have read the above and agree to participate in this research project/fitness appraisal at my own risk. I regularly take part in strenuous physical activity at least as intense as these tests. I realize that I may expect a thorough explanation and/or demonstration of any procedures and that I may terminate participation at any time in any or all procedures of my own volition.

Having voluntarily assumed participation and the risks thereof, in the project, I hereby disclaim and release the University of Victoria, its agents, servants or employees, including all personnel involved in the research project/fitness appraisal from any and all liability that might otherwise arise as a result of my participation as a research subject in this study/or fitness appraisal.

NAME: _____ DATE: _____

SIGNATURE: _____

APPENDIX D

MEAN TEMPERATURE AND HUMIDITY DURING TESTING AND TRAINING

Table 4: Mean Temperature and Humidity for the Heat/Exercise, Exercise and Control Groups during Testing and Training Sessions

		Heat/Exercise		Exercise		control	
		Temp (°C)	Humidity (mmhg)	Temp (°C)	Humidity (mmhg)	Temp (°C)	Humidity (mmhg)
Initial TST	$\bar{x}$	39.1	44.0	38.8	43.9		
Pre TST	$\bar{x}$	38.3	48.5	38.8	51.4	40.0	45.8
Post TST	$\bar{x}$	38.0	46.9	38.8	46.9	39.4	42.6
Training Sessions	$\bar{x}$	40.0	46.0	15.4	68.0		

Note. TST stands for Thermal Stress Test

APPENDIX E

$\dot{V}O_2$MAX PROTOCOL

Table 5: The Loads on the Cycle Ergometer for the Continuous Maximal Oxygen Consumption Test (CASS Protocol - MacDougall et al., 1982).

Resistance (kp)	Duration (min)
1.0	2
1.5	2
2.0	2
2.5	2
3.0	2
3.5	2
4.0	2
4.5	2
5.0	2

Note. Revolutions per minute varied from 70-90.

APPENDIX F

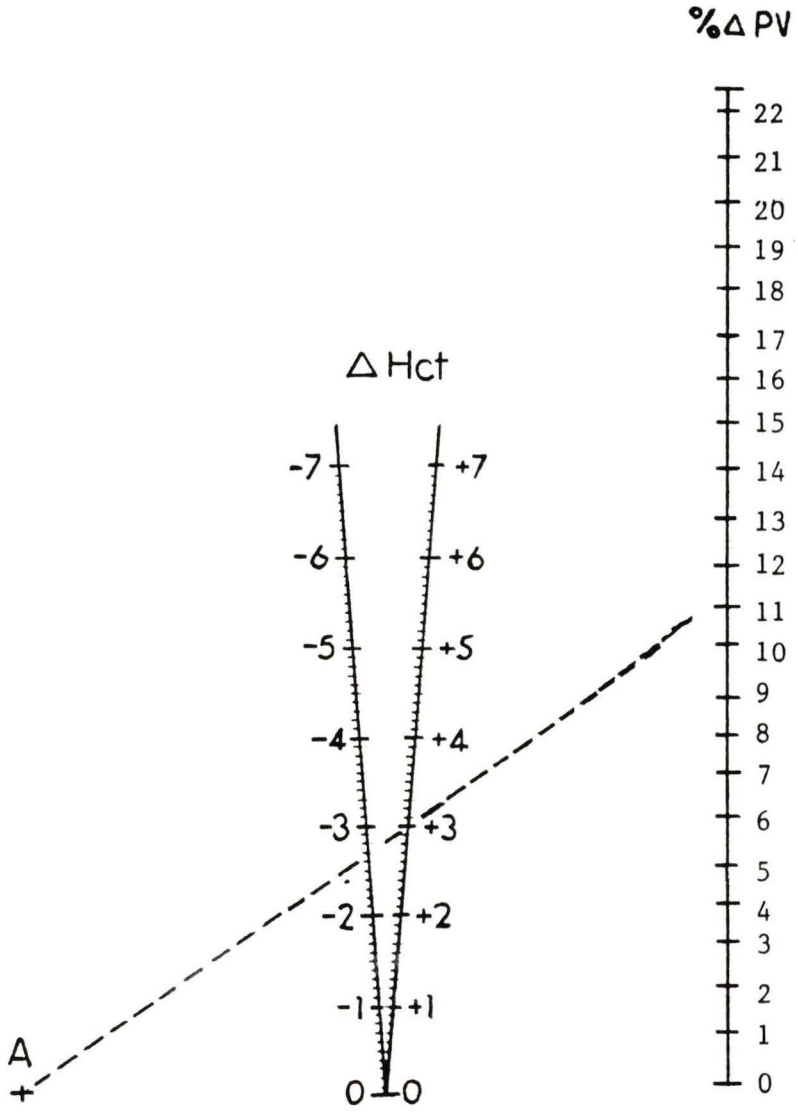
TRAINING INTENSITY FOR THE HEAT/EXERCISE AND EXERCISE GROUPS

Table 6: Intensity of Training for the Heat/Exercise and Exercise Groups During the Training Sessions

	Heat/Exercise	Exercise
Load (kpm)	1.25-1.75	1.75-2.25
Revs/min	50-70	40-60
Heart Rate (bpm)	155-165	160-165

APPENDIX G

NOMOGRAM TO CALCULATE PLASMA VOLUME FROM HEMATOCRIT



van Beaumont (1972)

APPENDIX H

MEANS AND STANDARD DEVIATION FOR HEART RATE, RECTAL TEMPERATURE, MEAN SKIN TEMPERATURE, PLASMA VOLUME, HEMATOCRIT AND POWER OUTPUT

Table 7: The Change in Heart Rate Over Time During the Pre and Post Thermal Stress Test

		Heat/Exercise		Exercise		Control	
		Pre	Post	Pre	Post	Pre	Post
HEART RATE (bpm)							
Start	$\bar{x}$	72	75	76	79	78	83
	SD	17	20	8	16	10	13
15 min	$\bar{x}$	154 *	156 *	161 *	156 *	163 *	152 *
	SD	9	14	8	13	9	14
30 min	$\bar{x}$	159 *	159 *	161 *	160 *	163 *	157 *
	SD	8	13	10	9	12	10
45 min	$\bar{x}$	161 *	159 *	159 *	158 *	162 *	159 *
	SD	5	10	8	9	8	14

* indicates a significant difference from starting values ($p < 0.05$).

Table 8: The Change in Rectal Temperature Over Time During the Pre and Post Thermal Stress Test

		Heat/Exercise		Exercise		Control	
		Pre	Post	Pre	Post	Pre	Post
RECTAL TEMPERATURE (°C)							
Start	$\bar{x}$	36.6	36.7	36.7	36.9	36.8	37.2
	SD	.17	.31	.39	.33	.34	.33
15 min	$\bar{x}$	36.9	37.0	37.0	37.2	37.2	37.5
	SD	.23	.24	.29	.23	.33	.32
30 min	$\bar{x}$	37.6 *#	37.6 *#	37.5 *	37.8 *#	37.9 *#	38.1 *#
	SD	.34	.28	.26	.16	.24	.35
45 min	$\bar{x}$	38.1 *#@	38.0 *#	38.0 *#	38.1 *#	38.3 *#@	38.5 *#@
	SD	.20	.26	.29	.25	.28	.36

* indicates a significant difference ($p < 0.05$) from starting values;
# from 15 min;
@ from 30 min.

Table 9: The Change in Mean Skin Temperature Over Time During the Pre and Post Thermal Stress Test

		Heat/Exercise		Exercise		Control	
		Pre	Post	Pre	Post	Pre	Post
MEAN SKIN TEMPERATURE (°C)							
Start	$\bar{x}$	35.4	35.2	35.8	35.4	36.3	36.0
	SD	.95	.97	.46	.97	.39	.74
15 min	$\bar{x}$	36.4	36.2	36.8 *	36.4	37.0	36.6
	SD	.31	.83	.26	.24	.40	.57
30 min	$\bar{x}$	36.5	36.6	37.3 *	36.8 *	37.5 *	37.1 *
	SD	.62	.85	.32	.31	.54	.48
45 min	$\bar{x}$	36.8	36.6	37.4 *	36.9 *	37.8 *#	37.2 *
	SD	.82	1.1	.51	.35	.56	.45

* indicates a significant difference ($p, 0.05$) from starting values;
# from 15 min.

Table 10: Means and Standard Deviation for Change in Plasma Volume during the Pre and Post Thermal Stress Tests

		Heat/Exercise		Exercise		Control	
		Pre	Post	Pre	Post	Pre	Post
PLASMA VOLUME (%)							
30 min	$\bar{x}$	-4.5	-10.3	-8.3	-5.6	-4.6	-7.7
	SD	2.9	6.4	.3	4.5	4.3	3.4
Finish	$\bar{x}$	-2.9	-8.6	-7.9	-2.7	-1.7	-8.8
	SD	3.9	7.4	5.0	3.8	6.1	3.9
Recovery	$\bar{x}$	-3.5	-1.2	-5.7	-2.7	+2.1	-3.6
(20 mins)	SD	3.1	8.4	4.4	3.6	4.8	3.2

* indicates a significant difference from Pre TST values for the C group ($p < 0.05$).

Table 11: Means and Standard Deviation for Hematocrit during the Pre and Post Thermal Stress Tests

		Heat/Exercise		Exercise		Control	
		Pre	Post	Pre	Post	Pre	Post
Hematocrit (%)							
Start	$\bar{x}$	40.0	38.5	40.5	40.0	41.0	39.5
	SD	3.7	1.8	1.8	1.1	2.4	1.9
30 min	$\bar{x}$	41.2	41.3	42.7	41.4	42.2	41.6
	SD	3.4	2.6	1.4	1.5	2.4	1.9
Finish	$\bar{x}$	40.9	40.9	42.4	40.7	41.4	41.9
	SD	3.1	3.3	0.9	0.9	2.9	2.3
Recovery	$\bar{x}$	41.0	38.7	42.0	40.7	40.4	40.5
(20 mins)	SD	3.5	3.7	1.3	1.0	2.9	2.4

Table 12: Means and Standard Deviation for Power Output during the Pre and Post Thermal Stress Tests

		Heat/Exercise		Exercise		Control	
		Pre	Post	Pre	Post	Pre	Post
Power Output (watts)							
5 min	$\bar{x}$	48	53	40	41	41	44
	SD	8	14	5	5	10	8
15 min	$\bar{x}$	114	114	116	114	116	105
	SD	16	20	19	9	15	14
25 min	$\bar{x}$	101	101	99	105	103	97
	SD	11	7	11	8	21	21
35 min	$\bar{x}$	96	87	87	81	83	70
	SD	12	6	9	19	14	22
45 min	$\bar{x}$	91	92	80	92	68	84
	SD	10	9	12	8	16	19

APPENDIX I

A CASE STUDY OF ONE SUBJECT UNABLE TO COMPLETE THE THERMAL STRESS TEST

Table 13: A Representative Case Study of one of the Five Subjects Unable to Complete Either the Pre or Post Thermal Stress Test

		Pre	Post
Subject A			
5 VO_2max ($ml \cdot kg^{-1} \cdot min^{-1}$)	$\bar{x}$	51.6	53.8
Weight Loss (kg)	$\bar{x}$	1.6	1.3
Fluid Intake (ml)	$\bar{x}$	150	475
T_R Start (°C)	$\bar{x}$	36.8	37.0
T_R Final (°C)	$\bar{x}$	38.3	38.5
% Dehydration	$\bar{x}$	3.0	2.5
Termination (mins)	$\bar{x}$	45	60

Note. Subject A represents the trends displayed by all of the subjects who were unable to complete either the Pre or Post Thermal Stress Test.

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