Menstrual Status and Thermoregulatory Responses of Active Adolescents During Exercise in a Cold Environment

by

Melora Cunliffe

Faculty of Applied Health Sciences
Brock University

A thesis submitted in partial fulfilment
of the degree Masters of Science

PART I: Review of Literature

Menstrual Status and Thermoregulatory Responses of Active Adolescents During Exercise in a Cold Environment. (2002), Brock University.

ABSTRACT

This study examined the interactions between the reproductive status and the thermoregulatory responses during exercise in the cold in girls involved in competitive sports. Four girls with established menstrual cycles comprised the eumenorrheic menarcheal group (EM) and 5 non-menstruating girls comprised the pre-menarcheal group (PM). During the first visit maximal oxygen consumption, height, weight and percent body fat (%BF) were measured. The second visit involved: a determination of metabolic rate in thermoneutrality (21°C) involving 10-min rest and 20-min cycling (30% of $\dot{V}O_2$ max), and a cold stress test (5°C, 40% humidity, <0.3 m/s air velocity) involving 20-min rest and 40-min cycling (30% of $\dot{V}O_2$ max.). Subjects in the EM group were tested twice in the chamber during the follicular and luteal phases. Pre-menarcheal subjects were found to have significantly ($p<0.05$) lower core temperatures during the final stages of cold exposure. Overall, body fat was not significantly correlated with core temperature in the cold, however there was a significant surface-to-mass ratio difference between the groups. While in the follicular phase, EM girls had a higher core temperature during cold exposure. Therefore, reproductive hormonal status seems to be an important factor in terms of cold tolerance in females during adolescence.

ACKNOWLEDGEMENTS

I wish to acknowledge my advisor Dr. Nota Klentrou and my graduate committee Dr. Sandra Peters and Dr. Mike Plyley for their continued support and guidance. I also wish to extend thanks Dr. Oded Bar-or and Dr. Bogdan Wilk at the Children's Exercise and Nutrition Centre at Chedoke Hospital in Hamilton. I am forever grateful to Greig Inglis and Jill Slack for their unfaltering assistance throughout the entire project. A final note of appreciation to my parents, grandmother and best friend for their caring encouragement over the last two years.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

LIST OF APPENDICES

LIST OF ABBREVIATIONS

Core temperature	T_{re}
Eumenorrheic menstruating	EM
Eumenorrheic menstruating follicular phase	EM-Follicular
Eumenorrheic menstruating luteal phase	EM-Luteal
Exercise in cold session	EX. C
Exercise in neutral session	EX. N
Follicle stimulating hormone	FSH
Forearm blood flow	FBF
Gonadotrophic releasing hormone	GnRH
Heart rate at maximal oxygen consumption	HR@$\dot{V}O_2$ max
Luteinizing hormone	LH
Metabolic rate	MR
Pre-menstrual	PM
Rate of perceived effort	RPE
Rest in neutral session	Rest N
Rest in cold session	Rest C
Skin temperatures	T_{sk}
Maximal oxygen consumption	$\dot{V}O_2$ max

Chapter 1

Thermoregulation in a Cold Environment

The cold environment places extreme stress on the human thermoregulatory system. Prolonged exposure to a cold stress environment results in the body's inability to successfully regulate body temperature, resulting in a severe and potentially dangerous decrease in body core temperature. If the thermoregulatory system is not capable of maintaining core temperature, a condition known as hypothermia develops.

According to Paolone and Paolone (1995), maintaining core temperature in the cold requires an equilibrium between heat loss and heat production. Mechanisms for heat loss include radiation, conduction, convection and evaporation; heat gain is from metabolic heat or environmental heat. Interestingly, studies examining the thermoregulatory responses of males and females in a cold environment suggest that men and women adapt physiologically to the cold stress in a different ways. Wagner et al. (1974) observed that men exposed to cold had an initial reaction of vasoconstriction, followed by a rapid fall in skin temperature Yakimenko (1991) concluded that during repeated work bouts in the cold, males adapt to a cold stress by increasing heat production. Silami-Garcia and Haymes (1989), in their examination of female responses to repeated short-term cold air exposure (10°C) reported that resting females respond with a delayed onset of shivering and a decrease in heat production upon acclimatization. It was concluded that the female body is able to adapt to repeated exposures to cold stress through specific mechanisms aimed at maintaining body heat (Silami-Garcia and Haymes, 1989). These

mechanisms include delayed onset of shivering times with no thermoregulatory heat produced (Silami-Garcia and Haymes, 1989). Sato et al. (1988) demonstrated that young women were superior to young men in cold tolerance during 1h of 12°C exposure.

1.1 Metabolic Response

In a cold environment, an increase in heat production is required in order to maintain core temperature. This occurs through an increase in metabolic rate (MR). Horvath et al. (1955) found that men, who were exposed to a cold environment, prevented a decrease of rectal temperature by increasing metabolic rate and decreasing skin temperature. Oxygen consumption was found to increase three fold in the cold environment (Horvath et al. 1955).

The metabolic responses to exercise in the cold reveal age and sex related differences. Wagner et al. (1974) demonstrated that during rest, boys increased their MR, while older men exhibited only a small change in MR. Smolander et al. (1992) reported similar results in that prepubescent boys reacted to cold exposure with an increased MR and a greater reduction in peripheral skin temperature than adult males. Frank et al. (2000) observed when comparing younger and older individuals that older men had a larger gain for total body oxygen consumption.

Gender differences have also been documented in the research. Pettit et al (1999) found that women have a significantly lower respiratory exchange ratio (RER) than men during rest in 5°C environment; they also reported that neither body fat or catecholamine responses to the cold stress accounted for the metabolic differences to the cold stress. On the other hand, Doubt (1991) has stated that catecholamine levels are higher during exercise in the cold, and Gallow et al.

(1984) suggested that during exercise, the MR adaptations are more dominant than morphological differences, while during rest the adaptive response appears to be the opposite.

Previous research also suggests that the menstrual status of the young athlete will affect MR, and therefore ultimately heat production during exercise in the cold. Graham (1988) reported differences in MR between women of differing menstrual status. It was found that men and eumenorrheic women increased their MR within 10 to 20 minutes of cold exposure, while amenorrheic women had a delayed response increasing their MR after one hour.

1.2 Exercising in the cold

Exercise in the cold adds a new dimension to the understanding of thermoregulatory mechanisms. Working muscles create heat for the body; however, low intensity exercise provides minimal heat production, with measurable thermoregulatory response to both the exercise and the cold stress. Kruk et al. (1990) reported that during exercise in the cold, oxygen consumption increased proportionally to intensity. It was also observed that oxygen consumption was significantly higher at 5°C than at 24°C. Falk et al. (1994) observed age-related differences during exercise in the cold, with older men exhibiting a greater decrease in core temperatures than seen in younger men.

Gender differences are also revealed during exercise in the cold. Stevens et al. (1987) observed lowered mean skin temperatures (T_{sk}), primarily in women compared to men. However, women demonstrated no differences in heart rate, stroke volume or oxygen consumption at 5°C and 21°C. Walsh & Graham (1986), Graham (1988) and Graham & Lougheed (1985) found similar results. In addition, Graham (1988) found that core temperature

(T_{re}) was similar between genders during the first hour of cold exposure. Graham & Lougheed (1985) reported that women had a greater core-skin temperature gradient because they maintained their core temperature with similar heat losses compared to men.

1.3 Individual Differences

The literature concerning the human thermoregulatory responses often focuses on the male body. However, several individual differences have been observed between genders. Gender differences have been studied in different environments. Walsh and Graham (1986) found that women generally have a lower mean skin temperature during rest and exercise compared to men. Graham (1988) suggested that, by keeping skin temperatures lower, the female thermoregulatory system allows core temperature to be maintained even in a cold environment. This is demonstrated in Stevens et al. (1987) research that observed lowered mean skin temperatures.

Age differences have also been found in body temperature regulation. Wagner et al. (1974) found that thermoregulatory mechanisms in cold temperatures were affected by age. Younger males reacted to a cold stress by increasing their metabolic rates and minimizing peripheral heat loss compared to older men. Wagner and Horvath (1985) reported that older men were more susceptible to cold ambient temperatures than younger people since they were unable to prevent a further rapid decline in their initially low core temperatures. Despite insulation from body fat, the older women in the same study maintained constant core temperatures at a greater metabolic cost than men or young women. Greater decreases in core temperature and larger heat debts characterized the older subjects in Falk et al. (1994) who found age related differences in men during rest and exercise.

During core temperature (T_{re}) cooling, Frank et al. (2000) observed no significant increase in epinephrine concentrations in both younger and older groups. This suggested that the sympathoneural rather than adrenomedullary system within the hypothalamus was primarily responsible for body temperature control during core cooling intravenously with cold fluid (40ml/kg,4°C) (Frank et al., 2000).

Smolander et al. (1992) examined thermoregulation in the cold with regard to differences in age and size. In comparing differences in body temperature between men and boys during rest and exercise, it was found that age dictated how these two populations maintained their body temperature. Men had smaller surface to mass ratios, which kept them warmer, while boys demonstrated an enhanced metabolic response to cold and a greater reduction in skin temperature in their extremities.

Hostile thermal environments (both hot and cold) create challenges for coaches and athletes. A cold stress environment places a considerable physiological strain on the body, and exercise during cold exposure significantly alters these responses by changing how the body is able to produce heat. Although research in this field has provided some answers regarding thermoregulation during exercise, definitive conclusions regarding exercise in the cold are lacking, especially for females. While age, sex and menstrual status have all been reported to influence thermoregulation of females during exposure to a cold stress, there is insufficient published research concerning the thermoregulatory responses during exercise of the young, active females to a cold stress.

The aim of the present study was to examine the thermoregulatory response of 13-18 year old athletic females during prolonged exposure to a cold stress environment, and to assess the role that menstrual status might have in altering the thermoregulatory response.

Chapter 2

Menstrual Cycle and Thermoregulation

The menstrual cycle is a component in the complex reproduction system, and is comprised of three different phases. The cycle begins on day 1 of the follicular phase, which is the onset on menses. In the first phase of the cycle, the ovarian follicles begin to mature as a result of follicle stimulating hormone (FSH). FSH stimulates the growth and development of the follicle in the ovary. At the beginning of the phase, estrogens and progesterone levels are low (Masters et al., 1995). During the mid-follicular phase an increase in estrogen output from the ovaries is followed by a surge of luteinizing hormone (LH). Ovulation occurs within hours of these surges. Ovulation is the second phase of menstruation and occurs during mid-cycle. The final phase of the cycle is the luteal phase, which is characterized by the output of large amounts of estrogen and progesterone (Vander et al. 2000).

Estrogen is composed of four separate hormones, with estradiol being the most abundant and most commonly measured (Bunt, 1990) of the four. Normal levels of estradiol in the follicular phase should be elevated prior to ovulation, with a second peak in the luteal phase. Progesterone levels also fluctuate pre- and post-ovulation, and exhibit a marked elevation after ovulation.

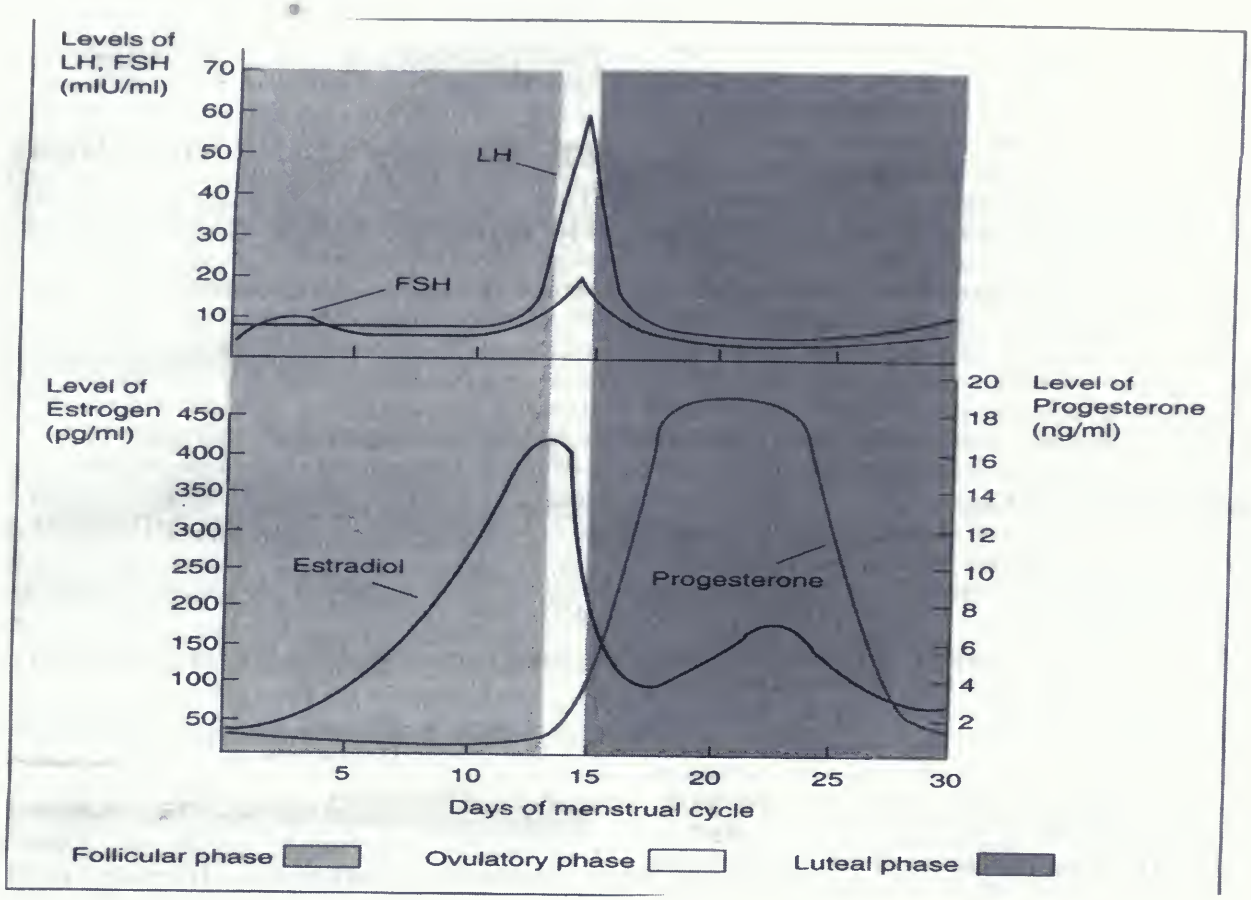

Figure 1.1: Hormones of the Menstrual Cycle (from Masters et al., 1995).

The endogenous hormones of the menstrual cycle are known to influence the female thermoregulatory system. The interplay between the reproductive and thermoregulatory systems is mediated by the hypothalamus (Graham, 1989). Boulant (1996) and Hammel (1968) illustrated the connection between hypothalamic temperature and skin and core temperature in the control of the thermoregulatory responses. Both estrogen and progesterone influence temperature regulation in women. These established cyclic fluctuations in endogenous hormones

are correlated to alterations in the thermoregulatory responses that have been documented in females.

2.1 Menstrual status of young active females

Recently, there has been an increased interest in the physiological responses of female athletes. These observations have focused on research concerning the interaction of exercise on the female menstrual cycle.

Abnormal menstrual responses are widely recognized in female athletes. Oligomenorrhea, amenorrhea, delayed menarche and shortened luteal phase have been observed in female athletes in a variety of sports, and in response to intense training. The combination of a high-energy output and low caloric intake often results in low body weight and low body fat. In some sports, such as running, gymnastics, wrestling, rowing and swimming, an emphasis is placed on lean, compact, and efficient bodies. Burrows and Bird (2000) examined exercise related menstrual disturbances, and concluded that no single factor was alone responsible in the initiation of abnormal cycles, but that it was a combination of several factors. However, as the LH pulse was suppressed in these athletes, the authors suspected that factors influencing LH were important in the etiology of an abnormal cycle (Burrows & Bird, 2000). Loucks (1990) found a reduced excretion of progesterone metabolites and a reduced length of the luteal phase with a longer follicular phase during intense training cycles athletes compared to sedentary women.

Morris and Wark (2001) measured hormone levels in female athletes with regular menstrual cycles and reported that many cycles were annovulatory. Morris and Wark (2001)

observed that 35% and 75% of schoolgirls and lightweight rowers had annovulatory cycles. Buchanan et al. (1987) found that changes in endocrine function associated with irregular menstruation have been reported to range from 1% to 60% among female athletes. Vuorento and Huhtaniemi (1992) reported similar data for athletes aged 14-15 years, in which 33% of the cycles were found to be annovulatory.

Along with the menstrual dysfunction, hypothalamic disruptions have also been reported. Lowered T_4 (thyroxin) levels in amenorrheic athletes compared to sedentary eumenorrheic controls; these finding were observed (Harber et al. 1998), (Loucks et al. 1992) Loucks and Callister (1993), in short-term studies with women aged 20-30, associated these altered concentrations of thyroid hormones with reduced energy availability where energy expenditure exceeds energy intake. Further evidence for thyroid involvement came from a study of young Japanese women complaining of unusual coldness who exhibited lower serum levels of T_4 and lower MR for both the control (29.5°C) and the colder session (23.5°C) (Nagasnma et al. (2002).

2.2 Thermoregulation and menstrual cycle

The influence of training on the female athlete's endocrine system and its interaction with thermoregulation has not been fully explored. The connection between these two systems is examined through the thermoregulatory responses of the female athlete with differing menstrual phases and/or menstrual status during rest and exercise.

Frascarolo et al. (1990) established that women have higher internal body temperatures during the luteal phase of the menstrual cycle. During investigations of the mechanisms that maintain the higher T_{re}, it was found that a heat balance was achieved during both phases, with

neither heat production nor heat loss being altered between the various phases of the cycle (Frascarolo et al., 1990). It has been suggested that during the luteal phase there is a re-setting of the basal body temperature, causing an increase in the thermoregulatory set point after ovulation.

In the research literature, it has been suggested that the higher temperature in the luteal phase sets all thermoregulatory responses at higher thresholds compared to the follicular phase. Hessemer and Bruck (1985a), concluded that skin and core temperatures, as well as heart rate, were higher during the luteal phase. The same authors, in a second article, also concluded that the thresholds for sweating, shivering and cutaneous vasodilators increased in the luteal phase (Hessemer & Bruck, 1985b). Kolka and Stephenson (1997) observed greater forearm blood flow (FBF) and heart rate in mid-luteal phase. The increase in FBF was thought to be caused by the endogenous reproductive hormones of the female body. When exercise was performed on days 2, 8, 14, 20, 26 of menstrual cycle, the onset and degree of forearm sweating occurred at higher temperatures during the luteal phase (Kolka & Stephenson, 1997). Gonzalez and Blanchard (1998) found lower heat debts and longer tolerance times for women during decreasing temperatures during the luteal phase. Illustrating that the women better able to thermoregulate during the luteal phase in cooler environments. The effect of post-ovulatory elevations in endogenous hormones on core temperature, skin temperature, heart rate, sweating and shivering rates is reflective in the change in the thermoregulatory set point in females during the luteal phase.

2.3 Menstrual cycle interactions with thermoregulation during exercise

In order to fully investigate the menstrual cycle and its affects on thermoregulation, it is important to examine differences in energy metabolism during rest and exercise. Webb (1986) concluded that there was an increase in energy expenditure during the post-ovulatory phase. Women were studied over a 24-hour period, and it was suggested that progesterone caused an elevated 24-hour expenditure in the luteal phase menstrual cycle (Webb, 1986). Unfortunately, this study involved only a limited number of subjects, and no control group. Forman et al. (1987) observed no correlation between the degrees of elevation of progesterone and the amplitude of the rise in body temperature.

No significant differences in metabolic responses to short-term moderate and high intensity exercise across the menstrual cycle have been reported (Galiliven et al., 1997) Stephenson et al. (1982) concluded that the menstrual cycle does not affect overall energy production over a range of metabolic intensities. Kim and Tokura (1995) reported an increased metabolic rate during exercise in the cold in the luteal phase. Matsuo et al. (1999) found a greater fat oxidation and post-exercise oxygen consumption, as well as a greater energy expenditure, during the luteal phase. While these reports would suggest an interaction between fuel utilization and the hormones of the menstrual cycle, De Souza et al. (1990) concluded that neither menstrual cycle phase (follicular or luteal), nor menstrual status, alters or limits exercise performance in female's athletes during either maximal or sub-maximal exercise. This is agreement with Melanson et al. (1996) who observed no difference in energy expenditure or substrate oxidation in young women during the follicular and luteal phases of the menstrual cycle.

2.3.1 Exercise in the heat

Past research has examined the effect of menstrual function on the thermoregulation of females during exercise in the heat. Horvath and Drinkwater (1982) measured four eumenorrheic women during the three stages of their menstrual cycle in a hot environment. It was concluded that minor cyclic alterations in the physiological systems are apparent at rest, but are masked by the demands of activity and the environment. The authors observed no differences in oxygen consumption, heart rate or blood pressure between phases (Horvath & Drinkwater, 1982). Frye et al. (1982) concluded that in a hot dry environment, acclimatized thermoregulatory function did not differ between gender and pre- and post-ovulation when fitness levels were the same. In contrast, Tengalia et al. (1999) found temperatures were elevated during the luteal phase in exercising women, resulting in decreased tolerance times in a hot environment with better thermoregulatory function in the follicular phase.

2.3.2 Exercise in thermoneutrality

Jurkowski et al. (1978) examined the effect of estradiol, progesterone, LH, and FSH during light, heavy and exhaustive exercise during the menstrual cycle. It was observed that exercise as a stimulus elevated plasma estradiol, progesterone and FSH, but not LH. The increases in these endogenous hormones exhibited elevations that were more marked in the luteal. During exercise, FSH increased at all intensities in the follicular phase, and LH appeared unaffected by the cycle phase. Prolonged exercise on a treadmill at mild to moderate intensities has been examined by Montagnani et al. (1992), testing occurred following the LH peak in the subject's cycles. Decreases in peripheral plasma levels of estradiol and progesterone occurred during exercise.

Pivarnik et al. (1992) examined temperature regulation during steady state endurance exercise during mid-luteal and mid-follicular phase. Maximal oxygen consumption, sweat loss and T_{sk} were not affected by cycle phase; however, heart rate and T_{re} were found to be higher during the luteal phase. While Hirata et al. (1986) found no significant differences between the two phases in FBF, oxygen consumption, carbon dioxide production, or heart rate during rest or exercise.

2.3.3 Exercise in the cold

Menstrual phase-related differences in thermoregulation during exercise have been found to be masked during cold exposure. Glickman-Weiss et al. (2000) reported no phase-related differences during a 90-minute cold exposure by menstruating women.

Graham et al. (1989) investigated menstrual status and cold exposure. Subjects were tested four times: twice at rest during a 60-minute exposure at 5°C and 22°C, and twice during exercise at the same temperatures. In the cold environment, eumenorrheic and amenorrheic subjects did not exhibit any differences in maximal oxygen consumption or percent body fat. Ammenorrheic women showed no increase in metabolism during exercise, while eumenorrheic women took 20 minutes to demonstrate an increased metabolic rate. Ammenorrheic women exhibited a low skin and core temperature, and a lower resting metabolism, in both normal and cold environments. Graham (1988, unpublished data) found that men and eumenorrheic women displayed an increased metabolic rate within 10 and 20 minutes of cold exposure, while amenorrheic women showed no increase in MR after one hour. Viswanathan et al. (1987) investigated changes in the endogenous opiods involved in the modulation of gonadotrophic releasing hormone (GnRH). Differing GnRH responses were observed between women during various phases of the menstrual cycle. While eumenorrheic women exhibited increased GnRH levels during rest and

exhaustive exercise in the cold, amenorrheic women did not show any evidence of an increase in GnRH.

Limited data are available concerning thermoregulation and the menstrual cycle in cold environments in amenorrheic women, and even less information has been reported for adolescents.

Chapter 3

Models to Study the Impact of Ovarian Hormones

As ovarian hormones have been documented to influence thermoregulatory responses, this chapter will examine two types of synthetic hormones and their affect on thermoregulation. The first model examines oral contraceptive use and the second hormone replacement therapy in older women.

The oral contraceptive pill is used by millions of women to control their menstrual cycle. Oral contraceptive pills are an accepted way to manage menstrual function through synthetically altering the female menstrual cycle. Understanding the effect that these exogenous hormones play in regulating body function could lead to information concerning the role of endogenous hormones in the body.

There are two types of oral contraceptives pills; the mini pill which contains only a low dose of progesterone, and the combination pill (Masters et al., 1995). This type of oral contraceptive pill is the most commonly used; it is a combination pill that provides both synthetic estrogens and progesterone. Together these exogenous hormones influences how the body regulates temperature.

Combination pills provide both estrogen and progesterone synthetically to the woman's body during the first half of the menstrual cycle and lends to a decreased output of FSH and LH by the pituitary gland (Master et al., 1995). The fluctuations caused by oral contraceptives in the thermoregulatory system can be observed at rest and during exercise and will be examined further in this chapter. Separately, synthetic progesterone and estrogen both have lasting effects on the female body, but varying effects on the female thermoregulatory system as evidenced by

increasing or decreasing core body temperatures. The changes observed in body core temperature and oral contraceptive use skim the surface of awareness for the female thermoregulatory system hormonal control. This chapter will review research concerned with different environments and states of activity to illustrate the many intertwined components of female thermoregulation and the use of exogenous reproductive hormones.

Pre-menoupausal and post-menopausal women use hormone replacement therapy in order to decrease menopausal effects on their bodies. These effects include decreased risk of osteoporosis and heart disease, while hormone replacement therapy has also been linked to increased risk of breast cancer and cancer of the uterus (Master et al. 1995). In regards to thermoregulation during menopause circulating levels of estrogen and progesterone begin to fall. The response to these decreased women begin hormone replacement therapy or estrogen replacement therapy; the synthetic impact of these hormones will be examined later in this chapter.

3.1 Oral contraceptives and body temperature regulation

Studies examining the effect of oral contraceptives use on core body temperature regulation involve the use of non-oral contraceptive users as controls. Baker et al. (2001) investigated core temperature in women in the active and placebo phases of oral contraceptives and compared them to women in their mid-follicular and mid-luteal phases. Body temperature was raised throughout the 24 hour testing period in the oral contraceptives users during both the active and placebo phase (Baker et al 2001a; Baker et al 2001b). Placebo phase is the final week of the oral contraceptive cycle that does not contain synthetic hormones., while the non oral contraceptives

users exhibited a higher temperature only in the luteal phase. These results illustrate the prolonged action of synthetically produced reproductive hormones on temperature regulation in oral contraceptive users. The raised temperature supports the findings that progesterone or progestin facilitates a rise in body temperature, for both oral contraceptives users and non-oral contraceptives users. Roger and Baker (1997) investigated the effects of synthetic estrogen and progesterone on thermoregulation in exercising women. It was concluded that the progesterone component of the pill had a dominant effect of thermoregulation and increased body temperature during both rest and exercise (Roger & Baker, 1997).

Stachenfeld et al. (2000) administered acute doses of progesterone- or estrogen-based hormones and examined women during passive heating at 35°C at rest and during exercise. It was observed that unopposed progesterone increased the esophageal temperature in women, and if progesterone was combined with estrogen a reversed effect was observed (Stachenfeld et al., 2000). Stephenson et al. (1982) confirmed earlier findings that estrogen decreases the thermoregulatory operating point. Taken together, these findings highlight that both progesterone and estrogen have significant effects on the female thermoregulatory system. However, the mechanisms by which progesterone and estrogen influence temperature regulation are unclear. Nakayma and Suzuki (1975) found that in female rats, progesterone inhibits warm-sensitive neuronal activity, which may inhibit heat loss mechanisms, resulting in increased body temperature. Conversely, estrogens inhibit cold and stimulate warm sensitive neurons, and therefore should inhibit heat-retaining mechanisms.

3.2 Oral contraceptives and heat exposure

During light exercise, Tengalia et al. (1999) examined heat strain and the duration of physical work tolerance in female oral contraceptives users and non-users during uncompensable heat stress. The women exercised intermittently wearing nuclear and chemical protective clothing in a 40°C chamber. It was observed that the female oral contraceptives users had a more uniform response to heat strain during their full cycle, while in the non oral contraceptives users, temperatures were elevated during the luteal phase, resulting in decreased tolerance times. The authors concluded that although there is a more uniform response with oral contraceptive use there appeared to be no influence on temperature regulation during uncompensable heat stress (Tengalia et al., 1999). Grucza et al. (1993) reached a similar conclusion, that the oral contraceptives users exhibited reduced fluctuations in their thermoregulatory responses, making it appear that their thermoregulatory responses were more consistent throughout the menstrual cycle. Therefore it was concluded that synthetic hormones during exercise influence the body by fostering a more uniform response throughout the entire menstrual cycle.

Although synthetic hormones influence thermoregulation, the menstrual cycle still exhibits a strong effect on the female body. Grucza et al. (1993) examined the extent to which oral contraceptives could modify the thermoregulatory responses to exercise. Both controls subjects and the oral contraceptive users exhibited an upward shift in core temperature during the luteal phase. These results indicate the strong effect that the menstrual cycle has over female thermoregulation both in the users and non-users of oral contraceptives.

3.3 Oral contraceptives and cold exposure

Oral contraceptive use was found reduced the menstrual phase-related difference in the thermoregulatory response to cold exposure in women who were tested during days 9-10 of their "quasi-follicular" phase and days 22-23 of "quasi-luteal" phase (Grucza et al., 1997). A 15-minute rest period at room temperature was followed by 30 minutes of sitting at a temperature of 3.4°C. The authors concluded that cold exposure during the quasi-follicular phase constituted a greater physiological strain than that during the quasi-luteal phase of menstruation of university age students (Grucza et al., 1997). This was indicated by an increased heart rate, respiratory frequency and oxygen uptake in the follicular phase.

Clearly, exogenous reproductive hormones affect temperature regulation in females. While endogenous hormones also affect temperature regulation, there is limited data on how low levels of what affects the female athlete.

3.4 Hormones Replacement Therapy

The purpose of hormones replacement therapy is to increase plasma levels of estrogen or progesterone. This administration of hormones alters the thermoregulatory responses of these older women. Estrogen replacement therapy has been found to decrease the occurrence of hot flashes by core temperature sweating threshold in women (Freedman & Blacker, 2001). This affect occurred independently of core and skin temperatures, as they were not changed (Freedman & Blacker, 2001). In a hot environment Brooks et al., 1997 found that chronic estrogen replacement acts centrally to decrease core temperature and lower the effect mechanism

initiation by active vasodilatation. It was also concluded that exogenous progestin block the effect of synthetic estrogens. Effects of exogenous hormones on temperature control in both younger and older women highlight the differences that an altered menstrual status may have on thermoregulatory responses (Charkoudian and Johnson, 1999; Freedman and Blacker 2002).

PART II: Research Paper

Chapter 4

Introduction

Essential to survival is the ability to adjust to changing environments. The body has evolved complex mechanisms to thermoregulate effectively. Thermoregulatory responses to various environmental conditions, such as hot air, cold air, hot humid air, and cold-water environments, have been the subject of extensive research in recent decades. Evidence suggests that there are various thermoregulatory responses between ages and genders, have also been found (Falk et al., 1994; Smolander et al., 1992; Wagner et al., 1974). According to Sloan and Keatinge (1973) in their investigation of gender differences in the cold, boys and younger swimmers had more rapid cooling rates than girls or older subjects, due to lower subcutaneous fat levels. Thus demonstrating thermoregulatory responses in the cold are linked to sex as well as maturation.

Wagner et al. (1974) found resting young male subjects reacted rapidly to cold stress through vaso-constriction and maintained core temperature, while older men were not able to maintain core temperature. Similarly, Smolander et al. (1992) examined boys and adults during rest and exercise, and found that the boys maintained core temperatures as effectively as the adults. In contrast, Falk et al. (1994) found that during exercise in the cold, core temperature decreased in older men more than in younger men.

However, gender differences were less pronounced since Graham (1983) and Walsh et al. (1986) found similar core temperatures between men and women in a cold environment. Research concerning girls thermoregulatory response to any environment is extremely limited,

therefore this investigation was designed to improve the knowledge concerning thermoregulation in the cold in female athletes.

The thermoregulatory responses of females in either the hot or the cold reveals phase-related differences in thermoregulatory response. Since both estrogen and progesterone influence temperature regulation in women, the fluctuations in these hormones during menstruation should elicit altered thermoregulatory responses (Graham, 1989). Progesterone is believed to initiate a temperature increase during luteal phase at rest in a warm environment, and higher core temperatures facilitated this during this phase (Frascarolo et al., 1990). Tenaglia et al. (1999) concluded that tolerance times in a hot environment during light exercise were significantly longer during the follicular phase compared to the mid-luteal for non-oral contraceptive users. These findings suggest that the phase-related differences in core temperature in neutral and hot environments are facilitated by estrogen and progesterone. Estrogen during the luteal phase decreases from its peak at ovulation followed by a second small increase in the mid-luteal phase. Progesterone peaks but reaches a plateau for the majority of the luteal phase. Although several studies have examined differing reproductive status' and thermoregulation very few authors investigated in a cold environment or with women of differing gynaecological age and hormonal status. It is yet unclear in the research how the underdeveloped ovarian hormones cycle in young women affects thermoregulatory responses in a cold environment. This study compared responses in both a neutral and cold environment.

The synthetic hormones found in the birth control pill have provided investigators with an ability to manipulate the menstrual cycle. This has also led to several key conclusions concerning how the hormones influence thermoregulation in the female body. Grucza et al. (1993) found that women who used oral contraceptives exhibited a reduced difference in

thermoregulatory response, making the thermoregulatory response more consistent throughout the menstrual cycle. Due to the uniform dosages of oral contraceptives, the hormonal spikes of estrogen, LH and progesterone do not occur. Hormone replacement therapy is a second model of menstrual cycle hormone replacement, which examines synthetic hormone affect on thermoregulatory responses

Pivarnik et al. (1992) examined thermoregulation during endurance exercise, and noted that one of the subjects was amenorrheic. It was observed that the temperature of the amenorrheic woman was not higher during rest or exercise in what should have been the luteal phase, suggesting that estrogen and progesterone influence thermoregulatory response. It is yet unclear whether non-menarcheal girls will demonstrate a similar affect in temperature regulation during the cold exposure.

In addition to the phase-related differences in core temperature of young women, developmental and abnormal menstrual status should also be examined. The abnormal menstrual cycle responses to intense training are an important factor in sport performance. High-energy output and low caloric intake results in low body weight and low levels of body fat. This is often associated with a delay in menarche, and a late onset of menstruation. Vuorento and Huhtaniemi (1992) reported data for girls aged 14-15 years, in which 33% of the cycles were found to be annovulatory.

There has been limited research on how the menstrual status effects on thermoregulation in a cold environment. Although some research suggests that menstrual cycle has no effect during rest in a cool environment. Little research has been done in young women during exercise (Graham 1983), Cunningham et al. (1978). In an attempt to add to the understanding of the effect of thermoregulation in female athletes, the present study aimed to investigate the

thermoregulatory responses between active young non-menarcheal and active young eumenorrheic athletes during exercise in the cold at various phases of the menstrual cycle. In the present study we expect to observe a decreased core temperature in pre-menstrual subject in the cold compared to eumenorrheic girls. Secondly, similar to adult women it suspected that young menstruating girls would have no thermoregulatory differences in the cold between their menstrual cycle phases. The specific objectives involve:

- Comparing the thermoregulatory response in the cold between eumenorrheic menarcheal and non-menarcheal active adolescents.
- Detecting possible fluctuations in the thermoregulatory response in the cold during different phases of the menstrual cycle in early post-menarcheal years.
- Examine the role that hormone level has on thermoregulatory responses to exercise in the cold.

Chapter 5

Methodology

5.1 Subjects

Nine active female adolescents matched for body type volunteered to participate in the research. Inclusions criteria were as follows:

- 13-18 years of age
- Artistic and rhythmic gymnasts, wrestlers, rowers and dancer;
- Minimum training volume 3 times per week for a minimum of 60 minutes

During a briefing meeting, the researcher provided oral and written information regarding procedures and possible risks involved in the experiments to all subjects and their parents/guardians. Institutionally approved informed written consent was obtained from all subjects' parents/guardians. The study was approved by the Ethics Board of both Brock and McMaster Universities.

All subjects were requested to complete the menstrual status questionnaire (Appendix 1) in order to be assigned to one of the following experimental groups:

- Eumenorrheic Menarcheal (EM, n=4): menarcheal girls with established menstrual cycles ≥ 9 cycles/year (Harber et al. 1998)
- Pre-Menarcheal (PM, n=5): non-menstruating girls.

Subject physical and baseline characteristics are presented in Table 6.1

5.2 Experimental design

After being assigned to one of the groups, each subject was contacted in order to schedule their visits to the Children's Exercise and Nutrition Centre, Chedoke Hospital. During the subject's first visit to the centre, they were familiarized with the study procedures. Anthropometric measurements (height, weight, relative body fat) were calculated for normalization of data. An aerobic capacity test of open-air spirometry was performed to determine maximal oxygen consumption ($\dot{V}O_2$ max).

During their following visits to the centre, subjects performed two tests in the climate chamber:

1) A test for the determination of metabolic rate in thermoneutrality (22.5°C). This test consisted of a 10-min rest period and a 20-min exercise period on a cycle ergometer at 65% of relative $\dot{V}O_2$ max. Following the test, each subject rested for 30-min in thermoneutrality.
2) The cold stress test under 5°C air temperature, 40% air humidity and <0.3 $m\text{-}s^{-1}$ air velocity. This test started with a 20-min rest period in a seated position followed by a 40-min cycling period at 30% of relative $\dot{V}O_2$ max. Total exposure time to cold was 60 min.

All tests were completed in the afternoon; a light meal was allowed approximately 2 hours before testing. During all tests, the subjects wore shorts, T-shirt, socks, and running shoes. Blood samples were collected before the neutral sessions and after the cold chamber sessions. All subjects were visually screened during cold exposure for reactions in the cold, including

Raynauds phenomenon. Subjects were able to consume approximately 50 ml of room temperature water during the 30-min rest period in a neutral environment; this was not normalized for body weight. Subjects in the PM groups were tested once. Subjects in the EM group were tested in the climate chamber twice: a) during the early follicular phase of their cycle (days 1-5), and b) during the mid luteal phase of their cycle (days 19-22).

5.3 Determination of menstrual status and phase

Determination of menstrual status was confirmed through the determination of serum hormonal levels. Menarcheal subjects reported their age at the onset of menarche as well as day 1 (i.e., first day of flow) of their last three cycles. They were then instructed to report day 1 of their next cycle. Testing sessions were scheduled to correspond to each of the desired phases. Progesterone and estradiol (17-β) were measured to verify the subjects' menstrual status and phase. For menstruating subjects, blood analysis was performed during days 1-5, 13-15 and 19-22 of menstrual cycle. For the non-menarcheal subjects, blood was drawn prior to their testing sessions.

5.4 Measurements

After a medical check-up, the subjects' $\dot{V}O_2$ max was directly measured on a Monark cycle ergometer at a cycling rate of 60 rpm by a SensorMedics Vmax 29 series metabolic cart. Based

on individual response power in watts was calculated at each 2-min stage. The criteria used to verify the achievement of $\dot{V}O_2$ max were:

a) a respiratory gas exchange ratio ≥ 1.1, and

b) a plateau of $\dot{V}O_2$ with increasing power output.

Skinfold thickness (sum of subcutaneous adipose tissue in mm) was assessed at five sites (subscapular and triceps) using Harpenden calipers. Relative body fat (%BF) was measured using bioelectrical impedance analysis (10/ARJL System/Clinton township.). Height (cm) was assessed using a Harpenden Stadiometer model 2109 and body mass (kg) was determined using a Mott Electro-scale, model UMC-600/ Brantford. Body surface area and body surface area-to-mass ratio were calculated using Dubois and Dubois (1916) equations. In order to estimate 30% power output oxygen consumption and power were plotted for linear regression analysis. 30 % of maximum oxygen consumption was found on the linear regression line and the corresponding power output was used for the climate chamber exposure.

Skin temperature ($^{\circ}C$) was monitored every 5-min using thermistors fixed at five sites (upper back, upper arm, forearm, finger and thigh). Mean skin temperature (T_{sk}) was calculated using Bonner et al. (1981).

$$TS = 0.03\ (T_{sub} + T_{upa}) + 0.2\ (T_{th} + T_{fr})$$

Heart rate (bpm) was monitored continuously through out the testing protocol by a Polar heart rate monitor 3000 system/Finland. Rectal temperatures (T_{re},$^{\circ}C$) were collected every 5-min by a thermistor (Yellow Springs Instrument, 400 series) inserted approximately 10 cm beyond

the anal sphincter. Heat production was determined from oxygen consumption and group mean respiratory exchange ratios and expressed as w/kg (watts/kilogram) and w/m^2 (watts/surface are)

The increase in the metabolic heat production was calculated from the $\dot{V}O_2$ measurements every 10-min during cold exposure, rest, and exercise. The thermoregulatory increase in $\dot{V}O_2$ was defined as the excess $\dot{V}O_2$ in the 5°C environment in comparison to the values obtained for comparable work rates in the thermoneutral environment. Heat conductance was calculated using heat production and rectal and skin temperature gradient and is expressed as w/m2/°C. The rate of perceived effort (RPE) was determined using the Borg 6-20 category scale (Borg, 1970). The thermal sensation rating of the body was determined using at 9-point scale (Appendix 2), with –4 being very cold, 0 being neutral and +4 being very hot (Fanger, 1972). Thermal comfort was assessed for the body using a 5-point scale (appendix 3), 0 being comfortable and 4 being very uncomfortable (Fanger, 1972).

Intravenous blood samples were taken from the subjects' arm. All blood samples were placed in a centrifuge for a 15-min period. The separated plasma was removed from the 10 ml sample tube and placed into to a 2 ml microtube. The samples were stored in a –20°C freezer until analysis. Estradiol (17-β) and progesterone were determined by immunoassays using the Immulite Analyser (Randolph, New Jersey).

5.5 Statistical Analysis

A one-way analysis of variance (ANOVA) was performed to assess group differences in physical and baseline characteristics. A one-way analysis of variance (ANOVA) was then performed to determine changes between status and phases for all thermoregulatory responses measured. Statistical significance was accepted at $p > 0.05$. When a significant F-ratio was obtained, a Bonferonni post-hoc test was further used to isolate differences among groups.

Chapter 6

Results

6.1 Subject characteristics

Table 6.1 summarizes the baseline data for the two groups. No significant differences were found in age, $\dot{V}O_2$ max, heart rate at $\dot{V}O_2$ max or percent body fat. Significant differences between the groups were observed in height, weight and surface-to-mass ratio at $p<0.01$ and in body surface area $p<0.05$.

Table 6.1: Physical and baseline characteristics for eumenorrheic menarcheal (EM;n=4), pre-menarcheal (PM;=5).

Variable	EM (mean ± SE)	PM (mean ± SE)	Range EM	Range PM
Age (y)	15 ± .7	13 ± 0.6	13-16	12-15
Height (cm)	169.5 ± 3.0	152.5 ± 2.83**	162.2-176	143.4-159
Weight (kg)	59 ± 2.0	40 ± 2**	54.9-62.3	34.5-47.1
Body Surface Area (m^2)	1.6 ± .04	1.31 ± .04*	1.59-1.7	1.46-1.3
Surface Area/Mass ($m^2 \cdot kg^{-1}$)	0.03 ± .01	0.03 ±.01**	0.03-0.03	0.03-0.03
Relative Body Fat	16.8 ± .1	14.5 ± 1	16.5-17	12-18
$\dot{V}O_2$ max ml kg^{-1} lean body mass min^{-1}	47 ± 1.5	48.3 ± 2.5	43.1-49.6	42.8-54.3
HR@$\dot{V}O_2$ max (bp min^{-1})	193.50 ± 4	199 ± 3.0	185-203	194-206

** $p<0.01$ PM significantly different from EM
* $p<0.05$ PM significantly different from EM

6.2 Rest and exercise in thermoneutral environment

No differences were observed in core temperature during rest or exercise. Neither menstrual phase nor menstrual status altered core temperature responses in the neutral environment. (Table 6.3). Mean skin temperature decreased in the neutral environment during both rest and exercise (Table 6.4). Menstrual status and menstrual phase did not affect mean skin temperature.

Heat production increased from rest to exercise in the neutral environment. Heat production per kilogram was significantly higher in PM subjects compared to EM girls only at the initial reading during rest ($p<0.05$)(Table 6.5). No differences were observed between menstrual phases. Table 6.6 depicts the increase in heart rate from exercise in the neutral session. In PM data subjects there was a trend towards higher heart rates when compared to both EM-Follicular and EM-Luteal. EM subjects in the luteal phase had slightly higher heart rates than EM-follicular subjects but this was not significant.

Thermal comfort and sensitivity were not significantly different between rest and exercise, or between status or phase (Table 6.7). Table 6.8 illustrates the similarities between the groups in rate of perceived exertion during exercise. Table 6.9 reveals heat conductance was similar for the subjects, with an expected increase during exercise.

Table 6.2: Core temperature in a neutral environment: (°C)

Time Interval	EM-Follicular (mean ± SE)	EM-Luteal (mean ± SE)	PM (mean ± SE)
Neutral 0 min (rest)	37.33 ± 0.13	37.16 ± 0.10	37.46 ± 0.02
Neutral 10 min (rest)	37.35 ± 0.16	37.2 ± 0.08	37.44 ± 0.07
Neutral 20 min (exercise)	37.35 ± 0.17	37.22 ± 0.09	37.34 ± 0.14
Neutral 30 min (exercise)	37.35 ± 0.10	37.38 ± 0.09	37.26 ± 0.22

Table 6. 3: Skin temperature in a neutral environment: (°C)

Time Interval	EM-Follicular (mean ± SE)	EM-Luteal (mean ± SE)	PM (mean ± SE)
Neutral 0 min (rest)	31.57 ± 0.60	32.06 ± 0.21	32.26 ± 0.46
Neutral 10min (rest)	30.69 ± 0.98	30.97 ± 0.21	30.51 ± 0.65
Neutral 20 min (exercise)	30.27 ± 0.80	30.59 ± 0.23	30.18 ± 0.58
Neutral 30 min (exercise)	30.41 ± 0.82	30.57 ±0 0.32	30.62 ± 0.40

Table 6.4: Heat production responses in a neutral environment: Watts (w), kilograms (kg), body surface area (m^2)

Time Interval	EM-Follicular (mean ± SE) w/m^2	EM-Luteal (mean ± SE) w/m^2	PM (mean ± SE) w/m^2	EM-Follicular (mean ± SE) w/kg	EM-Luteal (mean ± SE) w/kg	PM (mean ± SE) w/kg
Neutral (rest)	62.1 ± 3.7	54.7 ± 4.0	60.4 ± 3.0	1.7 ± .08	1.6 ± .1	2.0 ± .11*
Neutral (exercise)	172.8 ± 23.5	154.6 ± 7.5	153 ± 12	4.8 ± .6	4.4 ± .3	5.1 ± 4.1
Neutral (exercise)	163.8 ±21.8	144.1 ± 7.4	151. ±13.0	4.6 ± .5	4.1 ± .2	5.1 ± .45

(* $p<0.05$ PM significantly different to EM-Luteal).

Table 6.5: Heart rate in neutral a environment: Beats min^{-1}

Time Interval	EM-Follicular (mean ± SE)	EM-Luteal (mean ± SE)	PM (mean ± SE)
Neutral 0 min (rest)	78.00 ± 15.00	79.25 ± 6.02	90.20 ± 4.29
Neutral 10min (rest)	79.33 ± 7.62	82.50 ± 4.87	90.80 ± 7.11
Neutral 20 (exercise)	104.67 ± 4.18	106.25 ± 4.70	111.40 ± 2.04
Neutral 30 (exercise)	102.00 ± 3.61	107.00 ± 2.27	115.00 ± 3.91

Table 6.6: Thermal comfort and thermal sensitivity in a neutral environment.

Time Interval	EM-Follicular (mean ± SE)	EM-Luteal (mean ± SE)	PM (mean ± SE)
Thermal Comfort			
Neutral 0 min (rest)	2 ± 0.25	1 ± 0.25	1 ± 0.00
Neutral 10min (rest)	2 ± 0.00	1 ± 0.25	1 ± 0.20
Neutral 20 (exercise)	1 ± 0.33	1 ± 0.25	1 ± 0.24
Neutral 30 (exercise)	1 ± 0.33	1 ± 0.00	1 ± 0.40
Thermal Sensitivity			
Neutral 0 min (rest)	0.5 ± 0.50	-0.5 ± 0.29	0.3 ± 0.48
Neutral 10min (rest)	-0.7 ± 0.33	-0.5 ± 0.50	0.1 ± 0.56
Neutral 20 (exercise)	0.3 ± 0.33	0.3 ± 0.63	0.6 ± 0.40
Neutral 30 (exercise)	0.7 ± 0.33	0.8 ± 0.48	0.8 ± 0.37

Table 6.7: Rate of perceived exertion in neutral and cold environments.

Time Interval	EM-Follicular (mean ± SE)	EM-Luteal (mean ± SE)	PM (mean ± SE)
Neutral 20 min	8.00 ± 2.00	7.75 ± 1.03	8.20 ± 0.37
Neutral 30 min	8.33 ± 2.33	8.00 ± 1.15	9.00 ± 0.63
Cold 30 min	7.67 ± 1.67	7.50 ± 0.87	8.50 ± 0.96
Cold 40 min	7.00 ± 1.00	7.25 ± 0.75	9.00 ± 1.15
Cold 50 min	6.67 ± 0.67	6.75 ± 0.48	9.67 ± 1.76
Cold 60 min	6.33 ± 0.33	6.25 ± 0.25	9.67 ± 1.76

Table 6.8 Heat conductance in a neutral environment: Watts (w)/surface area (m^2)/skin-core temperature gradient (^{o}C)

Time Interval	EM-Follicular (mean ± SE)	EM-Luteal (mean ± SE)	PM (mean ± SE)
Neutral (rest)	9.4 ± .76	8.68 ± .60	9.21 ± 1.4
Neutral (exercise)	24 ± 2.8	23.6 ± 2.	21.6 ± 3.2
Neutral (exercise)	23.28 ± 1.4	21.2 ± 1.8	21.8 ± 2.2

6.3 Rest and exercise in cold environment

Figure 6.1 depicts core temperature during rest and exercise in the cold. There were no significant menstrual phase or status related difference during rest. However, during exercise EM subjects displayed a slight increase in $T_{re,}$ while during the same period PM subjects sharply

declined. There was a significant difference between EM-follicular and PM in T_{re} at the 60th min. of cold exposure ($p<0.05$). Although not significant there was a increase in T_{re} of EM-follicular compared to EM-luteal. Skin temperature in the cold rapidly decreased during the initial 10 min of cold exposure (Figure 6.2). Between 10 and 20 min T_{sk} a levelling off in all groups. There were no phase or status differences throughout the rest or exercise period.

Figure 6.3 displays the significantly increased heat production per kilogram in the PM girls compared to EM, during rest and exercise ($p<0.05$). Table 6.10 displays the heat production per surface area unit, PM girls recorded an increased response, but this did not reach significance. The EM group had a significant increase in heart rate from rest to exercise in the cold. The PM group's heart rate did not follow the same trend, it was initially high and maintained this elevation for the full exposure time (Table 6.11). The PM group was significantly different from EM-follicular at the beginning and after 20-min of rest in the cold ($p<0.05$). The difference was not apparent during exercise. There were no menstrual phase related differences in heart rate observed in the cold between in the EM group.

Table 6.12 depicts thermal comfort and thermal sensitivity in the cold. Exercise did not significantly alter perceived comfort or sensitivity for any of the groups. There were no significant differences in either thermal comfort or sensitivity based on status, a phase related trend revealed that the EM group felt more comfortable and less sensitive to the cold during the luteal phase. The RPE during the cold exposure decreased for the EM groups during both phases, and the PM subjects had an increased RPE during the same time period (Table 6.13). There were no menstrual phase related differences in RPE during the cold session. Table 6.14 displays heat conductance during rest and exercise in the cold, PM subject responses were significantly increased compared to EM-luteal ($p<0.05$).

The final table depicts the excess heat production from the neutral to cold environment during rest and exercise. All subject groups noted a significant increase in heat production during rest and exercise in the cold ($p<0.01$). The largest difference for heat production change was from the PM subjects.

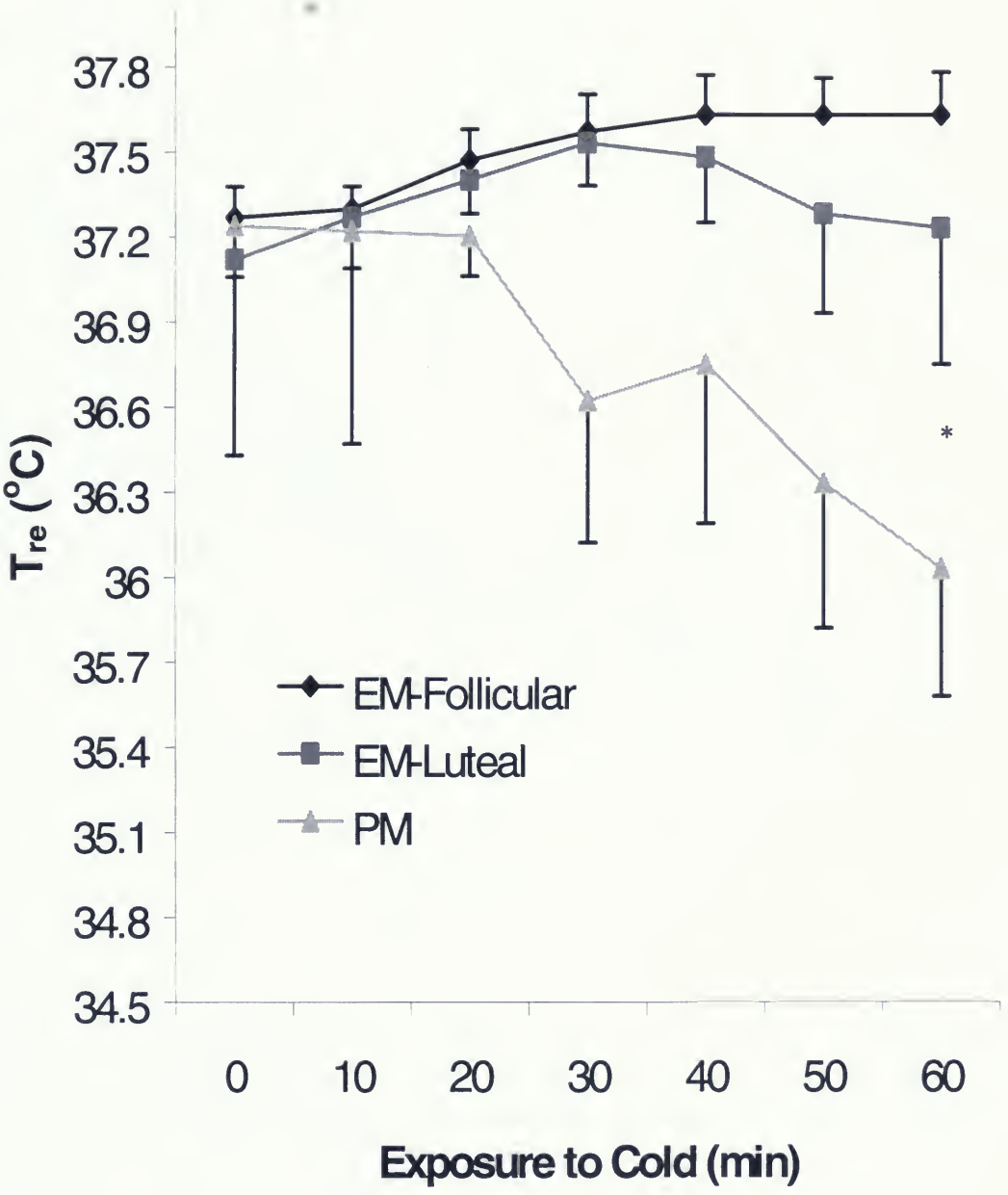

Figure 6.1: Rectal temperature in cold environment for Eumenorrheic menarcheal (EM) and pre-menarcheal (PM) subjects (* $p<0.05$ PM significantly different to EM-Follicular).

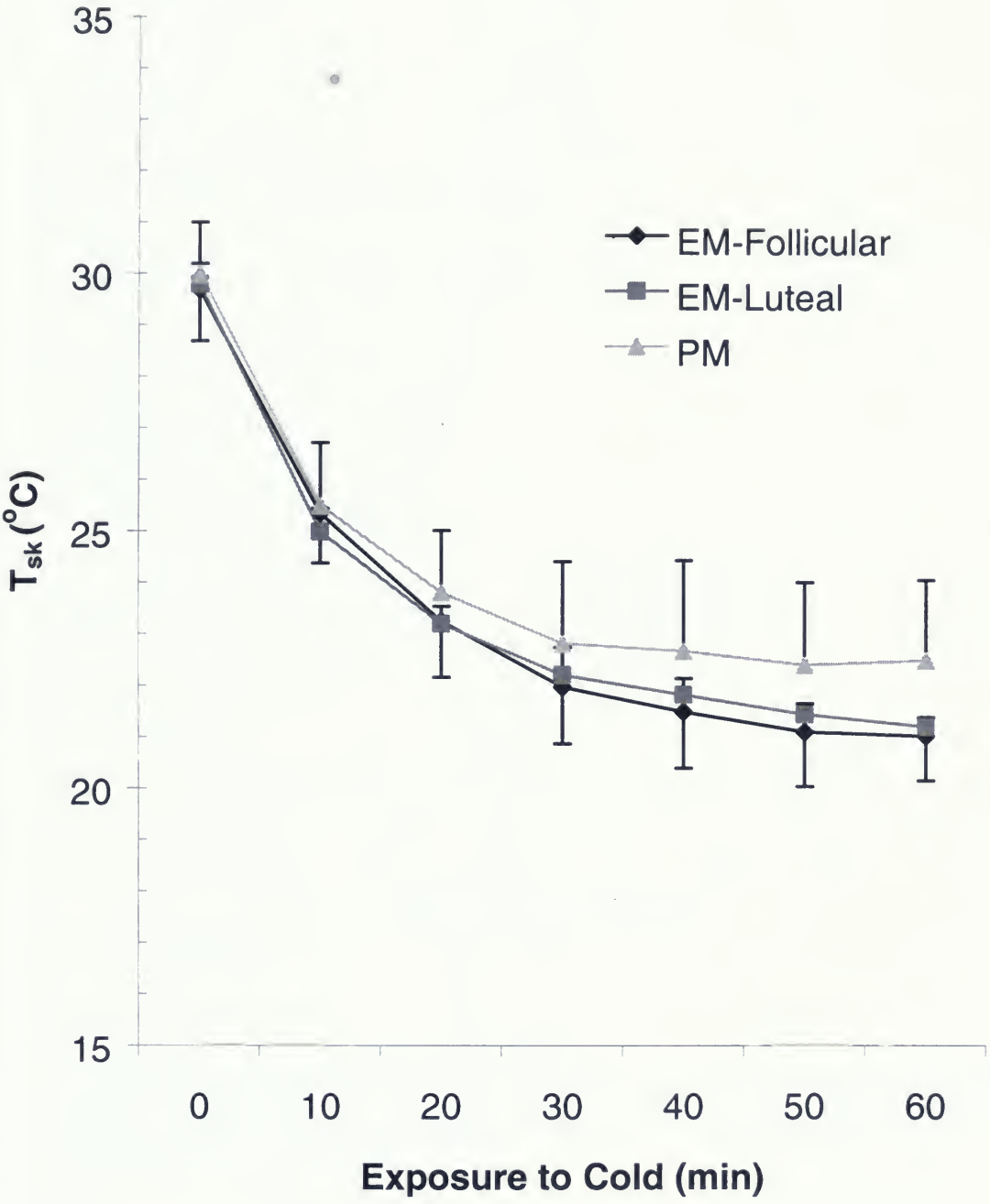

Figure 6.2: Skin temperature in cold environment for Eumenorrheic menarcheal (EM) and pre-menarcheal (PM) subjects.

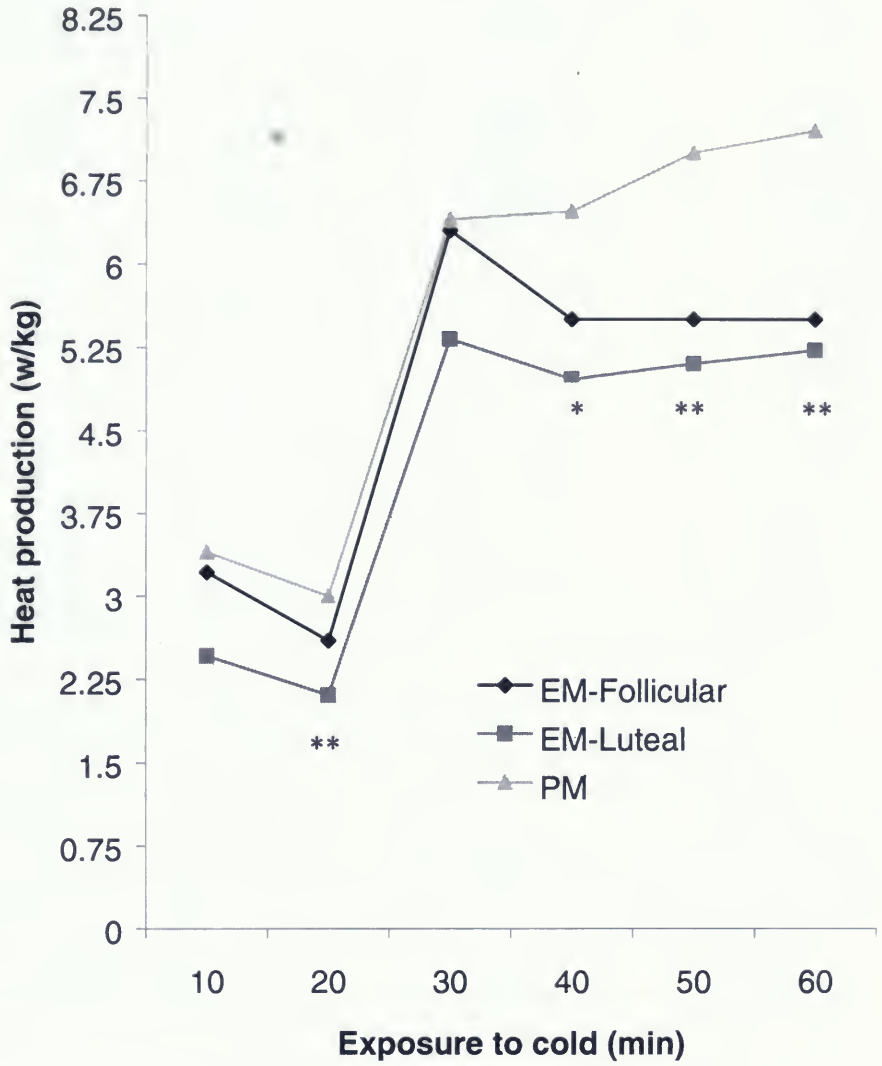

Figure 3: Heat production during cold sessions for eumenorrheic menarcheal (EM) and pre-menarheal (PM) subjects (* $p<0.05$ PM significantly different to EM-Luteal) ($p<0.01$ PM significantly different to EM-Luteal)

Table 6.9 Heat production responses in a cold environment: Watts (w), kilograms (kg), body surface area (m^2)

Time Interval	EM-Follicular (mean ± SE) w/m^2	EM-Luteal (mean ± SE) w/m^2	PM (mean ± SE) w/m^2
Cold (rest)	114.9 ± 11.1	86.1 ± 8.	102 ± 12.2
Cold (rest)	93.9 ± 10.2	74.2 ± 3.7	92.5 ± 6.04
Cold (exercise)	224.6 ± 11.6	186 ± 5.4	194.3 ±18.6
Cold (exercise)	197.1 ± 16.6	173 ± 6.1	192 ± 12
Cold (exercise)	197.8 ± 19.4	179 ± 8.3	208 ± 10.5
Cold (exercise)	197.5 ± 15.4	181.8 ± 11.8	213.5 ± 8.9

Table 6.10: Heart rate in cold environment. (Beats min^{-1})

Time Interval	EM-Follicular (mean ± SE)	EM-Luteal (mean ± SE)	PM (mean ± SE)
Cold 0 min (rest)	72.0 ± 7.21	77.8 ± 6.86	103.8 ± 8.60*
Cold 10 min (rest)	88.7 ± 6.98	82.8 ± 4.66	97.4 ± 4.27
Cold 20min (rest)	88.0 ± 4.93	89.3 ± 4.73	107.8 ± 4.59*
Cold 30 (exercise)	97.0 ± 5.57	98.5 ± 4.72	111.2 ± 3.84
Cold 40 (exercise)	97.7 ± 4.91	99.8 ± 4.66	108.8 ± 6.37
Cold 50 min (exercise)	99.7 ± 5.24	99.8 ± 4.97	110.8 ± 7.70
Cold 60 min (exercise)	100.7 ± 6.23	100.5 ± 4.66	102.0 ± 6.52

(* $p<0.05$ PM significantly different to EM-Follicular)

Table 6.11: Thermal comfort and sensitivity in a cold environment:

Time Interval	EM-Follicular (mean ± SE)	EM-Luteal (mean ± SE)	PM (mean ± SE)
Thermal Comfort			
Cold 0 min (rest)	4 ± 0.33	3 ± 0.25	3 ± 0.32
Cold 10 min (rest)	4 ± 0.00	4 ± 0.25	4 ± 0.20
Cold 20min (rest)	4 ± 0.00	3 ± 0.41	4 ± 0.20
Cold 30 (exercise)	4 ± 0.00	3 ± 0.41	3.5 ± 0.40
Cold 40 (exercise)	4 ± 0.33	3 ± 0.43	4 ± 0.25
Cold 50 min (exercise)	4 ± 0.33	3 ± 0.48	4 ± 0.25
Cold 60 min (exercise)	3.50 ± 0.29	3 ± 0.48	3.5 ± 0.29
Thermal Sensitivity			
Cold 0 min (rest)	-3.3 ± 0.33	-3.0 ± 0.41	-3.6 ± 0.24
Cold 10 min (rest)	-3.7 ± 0.33	-4.0 ± 0.00	-3.8 ± 0.20
Cold 20min (rest)	-3.7 ± 0.33	-3.5 ± 0.29	-3.8 ± 0.20
Cold 30 (exercise)	-3.7 ± 0.33	-3.0 ± 0.41	-3.6 ± 0.40
Cold 40 (exercise)	-3.7 ± 0.33	-2.8 ± 0.63	-3.5 ± 0.50
Cold 50 min (exercise)	-3.7 ± 0.33	-2.5 ± 0.65	-3.8 ± 0.25
Cold 60 min (exercise)	-2.3 ± 1.20	-2.5 ± 0.65	-3.8 ± 0.25

Table 6.12: Rate of perceived exertion.

Time Interval	EM-Follicular (mean ± SE)	EM-Luteal (mean ± SE)	PM (mean ± SE)
Neutral 20 min	8.00 ± 2.00	7.75 ± 1.03	8.20 ± 0.37
Neutral 30 min	8.33 ± 2.33	8.00 ± 1.15	9.00 ± 0.63
Cold 30 min	7.67 ± 1.67	7.50 ± 0.87	8.50 ± 0.96
Cold 40 min	7.00 ± 1.00	7.25 ± 0.75	9.00 ± 1.15
Cold 50 min	6.67 ± 0.67	6.75 ± 0.48	9.67 ± 1.76
Cold 60 min	6.33 ± 0.33	6.25 ± 0.25	9.67 ± 1.76

Table 6.13 Heat conductance in a cold environment: Watts (w)/surface area (m^2)/skin-core temperature gradient (^{o}C)

Time Interval	EM-Follicular (mean ± SE)	EM-Luteal (mean ± SE)	PM (mean ± SE)
Cold rest 10 min	9.96 ± 1.9	7.0 ± .56	9.0 ± 2.27
Cold rest 20 min	6.6 ± .48	5.22 ± .25	7.01 ± 1.1
Cold exercise 30 min	14.8 ± 2.1	12.13 ± .2	13.78 ± 1.85
Cold exercise 40 min	12.4 ± 1.0	11.01 ± .47	13.76 ± 1.48
Cold exercise 50 min	12.1 ± 1.2	11.38 ± .9	15.47 ± 1.71
Cold exercise 60 min	12.0 ± 1.1	11.42 ± 1.0	16.29 ± 1.4*

(* $p<0.01$ PM significantly different to EM-luteal).

Table 6.14: Heat production excess neutral to cold environment during rest and exercise: Watts (w)/kilogram (kg)

Time Interval	EM-Follicular (mean ± SE)	EM-Luteal (mean ± SE)	PM (mean ± SE)
Neutral –Cold* (rest)	.88 ± .33	.73 ± .08	1.22 ± .28
Neutral – Cold** (exercise)	1.9 ± .64	2.3 ± .31	3.2 ± .20

(* $p>0.05$ significant difference neutral-to cold all groups)

Chapter 7

Discussion & Conclusions

7.1 Effects of reproductive status

Several significant findings from this study illustrate the altered thermoregulatory responses of young girls of differing reproductive statuses in the cold. Despite higher heat production (w/kg) in the cold, PM girls demonstrated a rapid decline in T_{re}. Secondly, PM subjects exhibited an increase heat production response per unit of mass while having a higher heat conductance than EM girls.

Menstruating girls demonstrated significantly higher core temperatures than pre-menarcheal subjects following 60 minutes of cold exposure. The sharp decrease in core temperature of pre-menarcheal girls began after light exercise. These differences in core temperature did not correspond to body fat differences and were only displayed between the two groups during exercise.

Young boys sufficiently maintain their core temperature in the cold compared to adults through increased heat production; reported by Horvath (1958). Although both young boys and girls display increased heat production in the cold compared their older counterparts it is still unclear why the PM subjects core temperature continued to decline. It would appear that the heat production was insufficient to maintain core temperature in these girls.

In young women, Graham et al. (1989) found lower core temperatures in amenorrheic subjects compared to eumenorrheic controls, this was explained by the lack of a metabolic rate increase during the cold session. This data corresponded to earlier research by Graham (1988) who concluding metabolic responses of amenorrheic women were slower then menstruating women. Although a similar drop in core temperature was observed in the pre-menarcheal girls as Graham et al. (1989), there was an increased metabolic response in PM girls at the beginning of the cold session. Suggesting that metabolic responses are not the reason for the inability of the PM subject to maintain T_{re}.

Heat conductance was significantly higher for PM subjects in the cold during exercise. Despite the larger heat production the PM subjects displayed a larger skin/core temperature gradient. PM subjects produced more heat per unit of mass, however more heat was conducted towards the skin. This suggests a disrupted vasoconstrictor response in the PM subjects, and helps to explain the greater thermoregulatory in these subjects compared to EM subjects during the cold session.

Skin temperature did not differ with menstrual status in either the neutral or cold environment. Although a rapid decline was observed during the initial rest period in the cold, with the exercise stimulus T_{sk} stabilized at 30 – 40 minutes time interval. This occurred for all conditions and following 10-20 min of exercise. This would suggest that the exercise stimulus was sufficient to maintain skin temperature for all groups. These results are in agreement with Falk et al. (1994) who examined young boys and adults while exercising and found no significant difference in mean T_{sk} among the groups. However, Graham et al. (1989) observed lower skin temperatures in amenorrheic women compared to their eumenorrheic controls. Graham (1988) also concluded that women had lower T_{sk} to maintain their core temperature.

Therefore it would be expected that amenorrheic thermoregulatory responses would be similar to pre-menstrual subjects because of the lack menstrual cycle hormones. However, the pre-menarcheal subjects in the present study did not demonstrate the same response as women and amenorrheic women. This suggests PM subjects have altered thermoregulatory responses compared to menstruating and non-menstruating women. Suggesting that thermoregulatory responses not only differ with status but gynaecological age.

No significant differences were found during rest or exercise in the cold session between thermal comfort and thermal sensitivity. Eventhough the pre-menarcheal group had lower core temperatures in the cold; their perceived thermal comfort and perceived thermal sensation did not display this distress. Rate of perceived exertion was not altered by reproductive status during neutral or cold sessions. However, menstruating girls had a RPE that decreased during the session in the cold while PM girls indicated an opposite reaction. Although relative workloads were the same for both groups, as the exercise duration increased the harder PM girls felt they were working. RPE appears to be influenced by the temperature responses of the PM girls in the cold.

7.2 Effects of menstrual phase

The suggestion that menstrual cycle hormones are a component of the female thermoregulatory system is well supported in the literature. It has been suggested that the higher temperature in the luteal phase sets all thermoregulatory responses at higher thresholds compared to the follicular phase (Frascarolo et al. 1990; Hessemer and Bruck 1985a). More specially, Hessemer and Bruck (1985a) found that skin and core temperature, as well as heart rate, were higher during

the luteal phase. Frascarolo et al. (1990) established that women have higher internal body temperatures during the luteal phase of menstrual cycle. Investigating the mechanism that maintain the higher Tre, it was found that a hat balance was achieved during both phases, with neither heat production nor heat loss differed between the phase of the cycle (Frascarolo et al., 1990). It has been suggested that during the luteal phase there is a re-setting of the basal body temperature, causing an increase in the thermoregulatory set point after ovulation. In our study however, no menstrual phase differences were detected in core temperature in the neutral environment, therefore suggesting that the young eumenorrheic girls do not appear to have the same phase-related temperature fluctuations as older women. This may be explained by the differences in gynaecological age. Shortened luteal phase and lower progesterone levels have been reported in younger women compared to middle aged women (Bonen et al., 1981)

In the cold, menstruating girls demonstrated a similar core temperature at rest and during exercise in both phases of their menstrual cycle. This is in agreement with previous research in the cold (Graham, 1983; Cunningham et al., 1978:Glick-Weiss et al., 2000) reporting no significant differences in core temperature between menstrual phase in adult women. This however, is in contrast to Gonzalez and Blancahrd (1998) who found at rest elevated core temperature, lower heat debts and longer tolerance times in the cold during the luteal phase. However, these results were collected during decreasing ambient air temperatures, which may account for the discrepant results. No phase related differences were observed in Tsk during rest or exercise. This is in agreement with previous research during exercise in either neutral or hot environments (Frascarolo et al., 1990). In the cold, Graham (1983), and Glickman-Weiss et al. (2000) found that skin temperature was not changed during the menstrual cycle. In the present study a gradual decrease in T_{sk} was observed during rest in cold and a levelling off during

exercise similar to the neutral session. It appeared that light exercise facilitated maintenance of skin temperature in both phases of the girl's menstrual cycles.

As expected, heat production was higher in the cold than in the neutral temperature during both phases. This provides evidence that the cold stress during exercise places a strain on the thermoregulatory system. According to Horvath and Drinkwater (1982), oxygen uptake increases threefold in the colder environments while Kruk et al. (1990) observed that oxygen consumption increased proportional to the exercise intensity and was higher at 5°C than at 24°C. The young menarcheal girls in the current study responded with similar increases. However, heat production in the cold was not altered by phase for the EM subjects. This is in agreement with Glcikman-Weiss et al. (2000) who reported similar finding for women 18-30 years. Similar results have been reported in the heat (Frascarolo et al., 1990). Further, Stephenson and Kolka (1985) and Stephenson et al. (1998) found heat production in women was consistent between phases during the day. Since our experiments were consistently conducted at the same time during the day, heat production in y9unger girls appears to follow the same pattern during the menstrual cycle as older women

No phase-related variations in heart rate were observed during the cold session. This is in contrast with previous studies reporting higher heart rates in mid-luteal phase during strenuous exercise in the heat Kolka and Stephenson (1997); Stephenson and Kolka, (1985). There is limited published data to compare phase related variations of young girls to women, therefore several factors may have affected heart rates responses including environment, exercise and gynaecological age. Conclusions are difficult to draw with this many variables.

Menarcheal girls were more comfortable in the neutral session during their follicular phase while the same girls were slightly more comfortable in the cold during the luteal phase.

Thermo-sensitivity was also influenced by phase since these girls felt less cold during the luteal phase compared to the follicular, although these differences did not approach statistical significance.

7.3 Effects of body size

The pre-menarcheal girls were significantly different in body surface area-to-mass ratio's from the menarcheal girls and this may have influenced their lower core temperature response during cold exposure. A larger surface-to-mass ratio is a hurdle for temperature regulation. The larger area exposed to the cold with less mass for heat production could be the cause of the poor temperature control responses in the PM girls. However, Smolander et al. (1992) found that boys with a larger surface area-to-mass ratio maintained T_{re} as effectively as men by a greater reduction in T_{sk} and increased metabolic rates. The reduction in T_{sk} did not occur in pre-menarcheal girls. In addition, it has been suggested that during movement situations in women is more influenced metabolic responses than by morphological characteristics (Gallow et al., 1984). Graham et al. (1989) concluded that the different thermoregulatory responses observed in the cold between eumenorrheic and amenorrheic women were physiological in nature because there was no difference in body surface area or body surface per unit of mass. It appears that young pre-menarcheal females demonstrate different thermoregulatory responses in the cold compared to menstruating females mainly due to differences in their hormonal milieu.

There is evidence that the absence of reproductive hormones may have an effect on the thermoregulatory responses of females in the cold (Graham et al., 1989). However, limited data are available concerning thermoregulation in amenorrheic women, and no information has been

reported for pre-menarcheal young girls. Indirectly, the effects of exogenous hormones on temperature control in both younger and older women highlight the differences that an altered menstrual status may have on thermoregulatory responses (Charkoudian and Johnson, 1999; Freedman and Blacker 2002). After the completion of the hormonal analysis, the present data may provide some answer regarding the effect o estrogens and progesterone on thermoregulatory responses in this age group.

7.5 Conclusions

Menstrual status of girls appears to affect their thermoregulatory responses during exercise in the cold. It is concluded that cold stress places extreme pressure on the thermoregulatory system of pre-menarcheal females. With higher heat conductance rates the pre-menarcheal girls could not maintain their core temperature in the cold regardless of their increased rate of metabolic heat production. The control mechanism exhibited by menstruating girls for core temperature regulation in the cold seems to be absent in this population. Excluding size related differences this appears to be facilitated by the lack of menstrual cycle hormones in the PM girls.

There were no menstrual phase related differences in thermoregulatory responses of young girls in either the thermoneutral or cold environments. This data demonstrates that young female adolescents may be different in terms of thermo regulation than older women suggesting that gynaecological age may affect the thermoregulatory responses in young girls.

7.5 Future directions

The major limitation of this research study was the small sample size. For publications purposes the research study will be continued to eradicate this concern. Further, the paucity of information concerning young females and thermoregulation strongly suggests that additional sound research in this area would be beneficial. Specifically, an examination of the responses of menstruating and non-menstruating girls in hot environments may contribute to a more concise conclusion with regard to hormonal status and thermoregulation. A second study of equal importance should examine menstruating girls and women, to determine gynaecological age differences in thermoregulatory responses.

References

Baker, F.C. Waner, J.I., Vieira, E.F., Taylor, S.R., Driver., H.S., and Mitchell, D. (2001). Sleep and 24 hour body temperatures: a comparison in young men, naturally cycling women and women taking hormonal contraceptives. Journal of Physiology 530.3: 565-574.

Baker, F.C., Mitchell, D., and Driver, H.S. (2001). Oral contraceptives alter sleep and raise body temperature in young women. Flugers Archives European Journal of Physiology 442: 729-737

Bonen, A., Belcastre, A.N., Ling, W.Y. and Simpson, A.A. (1981). Profiles of selected hormones during menstrual cycles of teenage athletes. Journal of Applied Physiology: Respiration Environment Exercise physiology 50.3: 545-551.

Bonner, Clem, Bowen and Bowen. (1981). Skin tempeature. Journal of Applied Physiology 55: 248-260.

Borg, G. (1970). Percieved exertion s and indicator of somatic stress. Journal of Rehabilitation Medicine 2.2 : 92-98.

Boulant, J.A. (1996) Hypothalamic neurons regulatoring body temperature. In : Handbook of Physiology. Environmental Physiology. Bethesada, MD : Am. Physiol Soc. 4.1 : 105-126

Brooks, E.M., Morgan, A.L., Pierzga, S.L., Wladkowski, O'gorman, J.T., Deer, J.A., and Kenney, W.L. (1997). Chronic hormone replacement therpay alters thermoregulatory and vasomotor function in postmenoausal women. Journal of Applied Physiology 83.2 : 477-484.

Buchanan, L.T. Jr and Britzer et al (1987). Inter-relationship of diet athletic activity, menstrual status, bone density in collegiate females. American Journal of Clinical Nutrition 46: 681-684.

Bunt, J.C. (1990). Metabolic actions of estradiol: significance for acute and chronic exercises responses. Medicine and Science in Sport and Exercise 22: 286-290.

Burrows, M. and Bird, S. (2000). The physiology of the highly trained female endurance runner. Sports Medicine 30: 281-299.

Charkoudian N.C. and Johnson, J.M. (2000) Female reproductive hormones and thermoregulatory control of skin blood flow. Exercise and Sport Science Reviews 28.3:108-112.

Charkoudian N.C. and Johnson, J.M. (1999) Reflex control of cutaneous vasoconstrictor system is reset by exogenous female reproductive hormones. Journal of Applied Physiology 87.1:381-385.

Cunningham et al (1978). Female thermoregulatory responses. Journal of Applied Physiology.

De Souza, M.J., Maguire, M.S., Rubin, K.R., and Maresh, C.M. (1990). Effects of menstrual phase and amenorrhoea on exercise performance of runners. Medicine and Science in Sport and Exercise 22: 575-580.

Doubt, T.J. (1991). Physiology of exercise in the cold. Sport Medicine 11.6:367-381.

DuBois D and DuBois EF.(1916). A formula to estimate the approximate surface area if height and weight be known. Archives of International Medicine 17:863-71

Falk, B, Bar-or, O., Smolander, J., and Frost, G. (1994). Response to rest and exercise in the cold: effects of age and aerobic fitness. Journal of Applied Physiology 76: 72-78.

Fanger, P.O. (1972). Thermal Comfort : Analysis and application in envriomental engineering. New York : McGraw-Hill.

Frank, S. M., Raja, S.M., Bulcao, C.H., & Goldstein D.S. (2000). Age-realated thermoreguloary differences during core cooling in humans. Amercian Journal of Physiology Regulatory Integrative Comparative Physiology, 279.1: R349-R354.

Frascarolo, P., Schutz, Y., and Jequier, E. (1990). Decreased thermal conductance during the luteal phase of the menstrual cycle in women. Journal of Applied Physiology 69: 2029-2033.

Freedman. R.R. and Blacker, C.M. (2002). Estrogen raises the sweating threshold in postmenopausal women with hot flashes. Fertility and Sterility 77.3:487-490.

Frye, A.J., Kamon, E., and Webb, M. (1982) Responses of menstrual women, amenorrheal women and men to exercise in a hot, dry environment. European Journal of Applied Physiology 48: 279-288.

Galliven, E.A., Singh, A., Michelson, S., Bina, P., Gold, and Deuster, P.A. (1997). Hormonal and metabolic responses to exercise across time of day and menstrual phase. Journal of Applied Physiology 83: 1822-1831.

Gallow, D. Graham T.E., S. Pfeiffer. (1984). Comparative thermoregulatory responses to acute cold in women of Asian and European descent. Human Biology 65: 19-34.

Glickman-weiss, E.L., Cheatham, N., Caine, N., Blegen, M., & Marcinkiewicz. (2000) Influence of gender and menstrual cycle on a cold air tolerance test and its relationship to thermosensitivity. Undersear Hyperbaric Medicine 27.2 : 75-81.

Gonzalez, R.R. and Blanchard, L.A. (1998). Thermoregulatory responses to cold transients: effect of menstrual cycle in resting women. Journal of Applied Physiology 85: 543-553.

Gonzalez, R.R., Blanchard, L.A., and Allison, W.F. (1998). Cytokine Interaction with the menstrual cycle during cold stress and exercise. Annuals New York Academy of Sciences 856: 261-265.

Graham, T.E. (1983). Alcohol ingestion and sex differences on the thermal responses to mild exercise in a cold environment. Human Biology 55.2: 463-475.

Graham, T.E., Viswanathan, M., Van Dijk, J.P., Bonen, A., and George, J.C. (1989). Thermal and metabolic responses to cold by men and by eumenorrheic and amenorrheic women. Journal of Applied Physiology 67: 282-290.

Graham, T.E. (1988). Thermal, metabolic, and cardiovascular changes in men and women during cold stress. Medicine and Science in Sport and Exercise 20: S185-S192.

Graham, T.E., & Lougheed, M.D. (1985) Thermal responses to exercise in the cold: Influence of sex differences and alcohol. Human biology, 57 (4), p. 687-698.

Grucza, R., Pekkarinen, H., Timonen, K.,Titove, E., Kononoff, A., and Hanninen, O. (1997). Physiological Responses to cold in relation to the phase of the menstrual cycle and oral contraceptives. Annuals New York Academy of Sciences 813: 697-702.

Grucza, R., Pekkarinen, H., Titove, E., Kononoff, A., and Hanninen, O. (1993). Influence of the menstrual cycle and oral contraceptives on thermoregulatory responses to exercise in young women. European Journal of Applied Physiology 67: 279-285.

Hammel, H.T. (1968) Regulation of internal body temperature. Annual Reviews of Physiology 30: 641-710.

Harber, V.J., Petersen, S.R., and Chilibeck, P.D. (1998) Thyroid hormone concentrations and muscle metabolism in amenorrheic and eumenorrheic athletes. Canadian Journal of Applied Physiology 23(3), p. 293-306.

Hessemer, V. and Bruck, K. (1985a). Influence of menstrual cycle on thermoregulatory, metabolic, and heart rate responses to exercise at night. Journal of Applied Physiology 59: 1911-1917.

Hessemer, V. and Bruck, K. (1985b) Influence on shivering, skin blood flow, and sweating responses measured at night. Journal of Applied Physiology 59: 1902-1910.

Hirata, K., Nagasaka, T., Hirai, A., Hirashita, M., Takahata, T., & Nunomura. (1986) effect of human menstrual cycle on thermoregulatory vasodilatation during exercise. European journal of Physiology, 54, p. 559-565

Hong, S.L., and Nadel, E.R. (1979). Thermogenic control during exercise in a cold environment. Journal of Applied Physiology 47: 1084.

Horvath S.M. (1958). Journal of Applied Physiology.

Horvath. S.M., Radcliffe, C.E., Hutt, B.K., and Spurr, G.B. (1955). Metabolic responses of older people to cold environments. Journal of Applied Physiology 8: 145-148.

Horvath, S.M. and Drinkwater, B.L. (1982) Thermoregulation and the Menstrual cycle. Aviation Space and Environmental Medicine 53: 790-794.

Horvath, S.M. (1980) Exercise in a cold environment. Institute of environmental stress :221-241.

Jurkowski, J.E., Jones, N.L., Walker, W.C., Younglai, E.V., and Sutton, J.R (1978)Ovarian hormonal responses to exercise. Journal of Applied Physiology 44: 109-114.

Kim, H.E. and Tokura, T. (1995). Effects of the menstrual cycle on dressing behaviour in the cold. Physiology and Behaviour 58: 699-703.

Kolka, M.A., and Stephenson, L.A. (1997). Effect of luteal phase elevation in core temperature on forearm blood during exercise. Journal of Applied Physiology 82: 1079-1083.

Kurk, B., Pekkarinen, H., Manninen, K., and Hanninen, O. (1990). Comparison in men of physiological responses to exercise of increasing intensity at low and moderate ambient temperatures. European Journal of Applied Physiology 62: 353-357.

Loucks. A.B. (1990). Effects of exercise training on the menstrual cycle: existence and mechanisms. Medicine and Science in Sports and Exercise 22(3): 275-280.

Loucks, A.B., & Callister, R. (1993) Induction and prevention of low-T3 syndrome in exercising women. American Journal of Physiology 264: R924-R930.

Loucks, A.B., Laughlin, G.A., Mortola, J.F., Girton, L., Nelson, J.C. & Yen, S.S.C. (1992). Hypothalamic-pituitary-thyroidal function in eumenorrheic and amenorrheic athletes. Journal of Clinical endocrinology and Metabolism 75: 517-518.

Masters, W.H., Johnson, V.E., and Kolodny (1995). Human Sexuality (5th ed.). College New York, NY: Harper Collins.

Matsuo, T., Saitoh, S., and Suzuki, M. (1999). Effects of the menstrual cycle on excess post-exercise Oxygen consumption in healthy young women. Metabolism: Clinical and Experimental 48: 275-277.

Melanson, K.J., Saltzman, E., Russell, R., and Roberts, S.B. (1996). Postabsorptive and postprandial energy expenditure substrate oxidation do not change during the menstrual cycle in young women. Human and Clinical Nutrition :2531-2538.

Montagnani, C.F., Arena, B., and Maffulli, N. (1992) Estradiol and progesterone during exercise in healthy untrained women. Medical Science in Sport and Exercise 24: 21-25.

Morris, F.L., and Wark, J.D. (2001) An effective, economic way of monitoring menstrual cycle hormones in at risk female athletes. Medicine and Science in Sport and Exercise 33: 9-14.

Nakayama T, and Suzuki M (1975) Action of progesterone on prethermosentive neurones. Nature 258: 34-38.

Nagashima, K., Yoda, T., Yagishita, T., Taniguchi, A., Hosono, T., and Kanosue, K. (2002). Thermal regulation and comfort during a mild-cold exposure in young Japanese women complaining of unusual coldness. Journal of Applied Physiology 92: 1029-1035.

Paolone, V.J., and Paolone, A.M. (1995). Thermogenesis during rest and exercise in cold air. Canadian journal of Physiology and Pharmacology 73: 1149-1153.

Pivarnik, J.M., Marichal, C.J., Spillman, T., and Morrow, J.R. (1992). Menstrual cycle phase affects temperature regulation during endurance exercise. Journal of Applied Physiology 72: 543-548.

Pettit, S.E., Marchand, I., and Graham T. (1999) Gender differences in cardiovascular and catecholamine responses to cold-air exposure at rest. Canadian Journal of Applied Physiology 24: 131-147.

Rogers, S.M., and Baker, M.A. (1997). Thermoregulation during exercise in women who are taking oral contraceptives. European Journal of Applied Physiology 75: 34-38.

Sato, H., Yamasaki, K., Yasukouchi, A., Watanuki, S., & Iwanaga, K.(1988). Sex differences in human thermoregulatory response to cold. Journal of Human Ergo logy 17: 57-65.

Silami-Garcia, E., and Haymes, E.M. (1989). Effects of repeated short-term cold exposures on cold induced thermogenesis of women. International Journal of Biometeorology 33: 222-226.

Sloan, R.E.G., and Keatinge, W.R., (1973). Cooling rates of young people swimming in cold water. Journal of Applied Physiology 33.3: 371-375.

Smolander, J., Bar-Or, O., Korhonen, O., and Ilmarinen, J., (1992). Thermoregulation during rest and exercise in the cold in pre- and early pubescent boys and in young men. Journal of Applied Physiology 72.4: 1589-1594.

Stachenfeld, N.S., Silva, C., and Keefe, D.L.(2000). Estrogen modifies the temperature effects of progesterone. Journal of Applied Physiology 88: 1643-1649

Stephenson, L.A and Kolka, M.A., (1985) Menstrual cycle phase and time of day alter references signal controlling arm blood blow and sweating. American Journal of Physiology 249: R 186-R191.

Stephenson, L.A., Kolka, M .A., and Wilkerson, J.E.(1982). Metabolic and thermoregulatory responses to exercise during the human menstrual cycle. Medicine and Science in Sport and Exercise 14: 270-275.

Stevens, G.H.J., Graham, T.E., and Wilson, B.A. (1987). Gender differences in cardiovascular and metabolic responses to cold and exercise. Canadian Journal Physiology and Pharmacology 65: 165-171.

Tenaglia, S.A., McLellan. T.M., and Klentrou, P.P. (1999). Influence of menstrual cycle and oral contraceptives on tolerance to uncompensable heat stress. European Journal of Applied Physiology 80: 76-83.

Vander, A., Sherman, J., and Luciano, D. (2000) Human Physiology: The Mechanism of Body Function. Boston, MA: McGrawHill.

Viswanathan, M., Van Dijk, J.P. Graham, T.E., Bonen, A., and George, J.C. (1987). Exercise and cold induced changes in plasma B-endorphin and B-Lipotropin in men and women. Journal of Applied Physiology 62.2: 622-627.

Vuorento, T., and Huhtaniemi, I. (1992). Daily levels of salivary progesterone during menstrual cycle in adolescents girls. Fertility and Sterility 58: 685-690.

Wagner, J.A., and Horvath, S.M. (1985). Influences of age and gender on human thermoregulatory responses to cold exposure. Journal of Applied Physiology 58: 180-186.

Wagner, J.A., Robinson, S., and Marino, R.P. (1974). Age and temperature regulation of humans in neutral and cold environments. Journal of Applied Physiology 37: 562.

Walsh, C.A. and Graham, T.E. (1986). Male-female responses in various body temperatures during the following exercise in cold air. Aviation-Space, and Environmental Medicine 57: 966-973.

Webb, P. (1986). 24-hour energy expenditure and the menstrual cycle. American Journal of Clinical Nutrition 44: 614-619.

Yakimenko, M.A.(1991). Thermoregulation in man during cold adaptation. Genetics and Human Adaptation supplement: 534-536.

APPENDICES

Appendix 1: Menstrual Status Questionnaire

1. Have you ever had or have your period? Yes No

If NO, you are finished with this questionnaire.

2. How old were you when you had your first period?

3. Would you say that your menstrual cycle is regular(i.e., is there approximately an equal amount of time between each one)?

4. How often is your period?

5. Approximately how many days does your period last?

6.What was the first day of your last three periods? (dates DD/MM/YY)

7. Have you ever experienced bleeding between periods?

8. IF YES, how often?

Appendix 2: Thermal Comfort Scale

+4 very hot

+3 somewhat hot

+2 warm

+1 somewhat warm

0 neither warm nor cold

-1 somewhat cool

-2 cool

-3 somewhat cold

-4 being very cold

Appendix 3:Thermal Sensitivity Scale

4 very uncomfortable

3 uncomfortable

2 somewhat uncomfortable

1 slightly uncomfortable

0 comfortable

Appendix 4: Core temperature in cold environment (°C)

Time Interval	EM-Follicular (mean ± SE)	EM-Luteal (mean ± SE)	PM (mean ± SE)
Cold 0 min (rest)	37.20 ± 0.11	37.08 ± 0.06	37.24 ± 0.81
Cold 10 min (rest)	37.28 ± 0.08	37.22 ± 0.08	37.22 ± 0.13
Cold 20min (rest)	37.40 ± 0.11	37.32 ± 0.12	37.2 ± 0.14
Cold 30 (exercise)	37.48 ± 0.13	37.42 ± 0.15	36.62 ± 0.50
Cold 40 (exercise)	37.53 ± 0.14	37.38 ± 0.23	36.36 ± 0.56
Cold 50 min (exercise)	37.53 ± 0.13	37.22 ± 0.35	36.02 ± 0.51
Cold 60 min (exercise)	37.5 ± .15*	37.26 ± 0.48	35.78 ± 0.45*

[x] Subject AM04 core temperature decreased rapidly between the 30^{th}-35^{th} min. in chamber she was removed due to health concerns because of cold stress.

Appendix 5: Skin temperature in cold environment (°C)

Time Interval	EM-Follicular (mean ± SE)	EM-Luteal (mean ± SE)	PM (mean ± SE)
Cold 10 min (rest)	25.34 ± 1.22	24.98 ± 0.45	25.49 ± .97
Cold 20min (rest)	23.24 ± 1.22	23.19 ± 0.34	23.79 ± 1.09
Cold 30 (exercise)	21.96 ± 1.60	22.20 ± 0.53	22.81 ± 1.10
Cold 40 (exercise)	21.49 ± 1.76	21.81 ± 0.32	22.66 ± 1.10
Cold 50 min (exercise)	21.1 ± 1.60	21.44 ± 0.21	22.39 ± 1.06
Cold 60 min (exercise)	21.02±1.56	21.20 ± 0.18	22.48 ± 0.87

Appendix 6: Oxygen consumption cold environment:

Time Interval	EM-Follicular (mean ± SE) w/kg	EM-Luteal (mean ± SE) w/kg	PM (mean ± SE) w/kg
Cold (rest)	3.22 ± .32	2.46 ± .23	3.4 ± .38
Cold (rest)	2.6 ± .24	2.11 ± .07	3.0 ± .16
Cold (exercise)	6.3 ± .33	5.32 ± .22	6.4 ± .56
Cold (exercise)	5.5 ± .37	4.96 ± .27	6.47 ± .38*
Cold (exercise)	5.5 ± .46	5.1 ± .35	7.0 ± .28**
Cold (exercise)	5.5 ± .35	5.22 ± .45	7.2 ± .28**

Appendix 7: SPSS data sheets

Codes

Id- study identification
Status-menstrual status 1=EM, 2=NM

Vo2- $\dot{V}O_2$ max ml kg^{-1} lean body mass min^{-1}

Age-age in years
Hgt-height cm
Wgt- weight kg
Bodyfat-skinfold percent body fat
Bia- bioelectrical impedance
Cond-condition, 1=EM-follicular, 2=EM-luteal, 3=NM
n-neutral
c-cold
pr-pre chamber
pt-post chamber
hr-heart rate (bpm)
cr-core temperature
sk-skin temperature
thecom-thermal comfort
thesen-thermal sensitivity
code number- time

	id	status	vo2	age	hgt	wgt	bodyfat
1	2	1	45.98	15	162.20	54.90	16.5
2	3	1	49.55	16	176.60	55.75	17.0
3	4	1	49.02	16	170.50	62.30	16.5
4	5	1	43.10	13	168.80	60.10	17.0
5	2	1	45.98	15	162.20	54.90	16.5
6	3	1	49.55	16	176.60	55.75	17.0
7	4	1	49.02	16	170.50	62.30	16.5
8	5	1	43.10	13	168.80	60.10	17.0
9	1	2	47.57	13	157.20	41.80	14.0
10	2	2	54.26	15	159.00	47.10	12.0
11	3	2	43.03	12	153.80	38.10	14.0
12	4	2	53.55	13	143.40	34.50	18.0
13	6	2	42.80	12	149.10	37.00	14.5

	bia	cond	no2r1	no2e2	no2e3	nhrpr	nhr0
1	20.0	1	.261	.654	.655	.	.
2	19.0	1	.	.	.	.	.
3	24.0	1	.345	1.060	1.010	65.00	63.00
4	22.0	1	.307	.733	.676	93.00	93.00
5	20.0	2	.262	.622	.627	97.00	95.00
6	19.0	2	.229	.825	.691	64.00	68.00
7	24.0	2	.262	.780	.806	72.00	82.00
8	22.0	2	.323	.700	.635	84.00	72.00
9	18.0	3	.283	.537	.553	105.00	102.00
10	10.0	3	.235	.675	.670	73.00	78.00
11	13.0	3	.216	.622	.635	76.00	83.00
12	11.0	3	.229	.617	.617	101.00	94.00
13	16.0	3	.218	.421	.396	98.00	94.00

	nhr5	nhr10	nhr15	nhr20	nhr25	nhr30	nhrpt
1	70.00	82.00	105.00	101.00	106.00	107.00	.
2	.	.	.	.	.	.	.
3	62.00	65.00	92.00	100.00	97.00	95.00	60.00
4	87.00	91.00	111.00	113.00	106.00	104.00	.
5	78.00	92.00	110.00	120.00	116.00	113.00	87.00
6	68.00	69.00	90.00	99.00	102.00	104.00	77.00
7	75.00	86.00	104.00	104.00	104.00	103.00	76.00
8	80.00	83.00	97.00	102.00	104.00	108.00	81.00
9	107.00	97.00	113.00	114.00	115.00	111.00	92.00
10	66.00	64.00	92.00	104.00	99.00	103.00	83.00
11	82.00	90.00	108.00	114.00	120.00	124.00	80.00
12	102.00	105.00	119.00	115.00	123.00	123.00	91.00
13	101.00	98.00	113.00	110.00	112.00	114.00	86.00

	ncrpr	ncr0	ncr5	ncr10	ncr15	ncr20	ncr25
1	.	37.30	37.36	37.30	37.40	37.40	37.50
2	.	.	.	.	.	.	.
3	37.10	37.20	37.20	37.20	37.20	37.20	37.40
4	37.70	37.70	37.70	37.80	37.80	37.80	37.70
5	37.20	37.20	37.20	37.20	37.20	37.20	37.30
6	37.10	37.20	37.20	37.20	37.20	37.20	37.30
7	37.40	37.40	37.40	37.40	37.40	37.40	37.50
8	37.20	37.20	37.20	37.30	37.30	37.40	37.50
9	37.20	37.50	37.30	37.20	37.20	37.00	36.90
10	37.40	37.40	37.40	37.50	37.50	37.50	37.50
11	37.50	37.50	37.40	37.40	37.20	37.00	36.80
12	37.60	37.50	37.70	37.60	37.70	37.70	37.80
13	37.40	37.40	37.40	37.50	37.50	37.50	37.50

	ncr30	ncrpt	nskpr	nsk0	nsk5	nsk10	nsk15
1	37.50	.	.	32.41	32.24	32.12	31.50
2	.	.	.	.	.	.	.
3	37.30	37.40	30.89	30.41	30.24	28.81	29.14
4	37.50	37.50	32.26	31.89	31.51	31.13	30.78
5	37.30	37.30	32.67	31.98	31.43	31.25	30.94
6	37.40	37.40	32.10	31.53	30.79	30.66	30.05
7	37.60	37.60	32.98	32.21	31.67	31.39	30.90
8	37.50	37.50	33.43	32.52	31.80	30.56	30.56
9	36.90	37.00	34.06	32.79	32.21	32.30	31.86
10	37.60	37.60	31.73	30.94	29.45	29.88	29.91
11	36.60	36.60	30.52	.	28.72	28.48	28.27
12	37.70	37.70	33.31	32.37	31.17	30.55	29.77
13	37.50	37.50	33.78	32.94	31.64	31.34	30.94

	nsk20	nsk25	nsk30	nskpt	nthcompr	nthcom0	nthcom5
1	31.54	31.65	31.67	.	.	.	.
2	.	.	.	.	.	.	.
3	28.80	28.90	28.88	29.12	1	2	2
4	30.48	30.41	30.67	30.84	2	2	2
5	30.89	30.72	30.82	31.30	1	1	1
6	30.04	29.82	29.79	30.49	1	1	1
7	31.03	31.06	31.31	32.14	1	1	1
8	30.41	30.32	30.37	30.59	1	2	2
9	31.62	31.76	31.64	32.41	.	.	1
10	29.88	30.11	30.07	30.83	1	1	2
11	28.27	.	.	.	1	1	1
12	30.05	29.94	29.91	30.90	1	1	1
13	31.09	30.99	30.85	31.72	1	1	1

	nthcom10	nthcom15	nthcom20	nthcom25	nthcom30	nthcompt	nthsenpr
1	2	2	2	1	1	.	.
2	.	.	.	.	.	.	.
3	2	1	1	1	1	1	0
4	2	2	2	2	2	1	2
5	1	1	1	1	1	1	0
6	1	1	1	1	1	1	2
7	1	1	1	1	1	1	2
8	2	2	2	1	1	1	0
9	1	2	2	3	3	2	.
10	2	2	2	1	2	1	2
11	1	1	1	1	1	1	2
12	1	1	1	1	1	1	0
13	1	1	1	1	1	1	0

	nthsen0	nthsen5	nthsen10	nthsen15	nthsen20	nthsen25	nthsen30
1	.	0	0	0	0	1	1
2	.	.	.	.	.	.	.
3	-1	-1	-1	0	0	0	0
4	2	1	-1	0	1	1	1
5	0	-1	-1	0	0	0	1
6	0	1	1	1	2	2	2
7	-1	-1	-1	0	0	0	0
8	-1	-1	-1	-1	-1	0	0
9	.	0	0	0	0	0	0
10	1	0	-2	1	1	1	1
11	1	0	2	2	2	2	2
12	0	0	0	0	0	0	0
13	-1	-1	0	0	0	0	1

	nthsenpt	nrpe15	nrpe20	nrpe25	nrpe30	co2r1	co2r2
1	.	12	12	13	13	.603	.390
2	.	.	.	.	.	.	.
3	1	6	6	6	6	.624	.577
4	0	6	6	6	6	.466	.424
5	0	11	10	10	10	.486	.356
6	0	9	9	10	10	.336	.320
7	0	6	6	6	6	.396	.387
8	1	6	6	6	6	.481	.406
9	0	9	9	9	9	.573	.413
10	1	7	7	7	7	.456	.470
11	2	8	8	9	9	.420	.374
12	0	7	8	9	9	.238	.261
13	1	9	9	11	11	.343	.321

	co2e3	co2e4	co2e5	co2e6	chrpr	chr0	chr5
1	1.070	.796	.835	.846	72	68	89
2	.	.	.	.	.	.	.
3	1.150	1.120	1.150	1.110	62	62	76
4	.976	.900	.839	.862	89	86	102
5	.878	.758	.817	.884	91	82	94
6	.897	.916	.978	1.000	61	64	.
7	.833	.852	.821	.751	70	70	95
8	.940	.778	.800	.830	83	95	102
9	.688	.717	.793	.800	95	122	125
10	1.000	.887	.991	.977	74	87	86
11	.754	.	.	.	86	127	104
12	.763	.735	.769	.811	94	88	97
13	.496	.556	.654	.715	88	95	101

	chr10	chr15	chr20	chr25	chr30	chr35	chr40
1	76	78	97	94	90	90	89
2	.	.	.	.	.	.	.
3	90	70	80	96	93	96	98
4	100	96	87	113	108	110	106
5	88	82	89	105	101	102	105
6	69	68	76	86	85	82	86
7	85	93	97	108	101	109	102
8	89	103	95	112	107	102	106
9	108	113	107	107	111	117	118
10	90	70	97	96	104	93	91
11	101	90	115	115	125	.	.
12	85	96	99	115	112	109	108
13	103	92	121	105	104	110	118

	chr45	chr50	chr55	chr60	chrpt	ccrpr	ccr0
1	93	96	95	96	85	37.20	37.20
2	.	.	.	.	.	.	.
3	93	93	95	93	57	37.20	37.10
4	106	110	110	113	83	37.50	37.50
5	106	104	106	102	72	37.00	37.00
6	86	88	85	87	64	37.20	37.10
7	105	96	108	105	81	37.20	37.20
8	100	111	104	108	83	37.20	37.20
9	119	104	108	83	.	37.10	37.20
10	104	93	98	104	57	37.40	37.30
11	.	.	.	.	.	37.00	37.00
12	114	118	106	111	75	37.50	37.50
13	122	128	121	110	72	37.20	37.20

	ccr5	ccr10	ccr15	ccr20	ccr25	ccr30	ccr35
1	37.10	37.20	37.30	37.40	37.40	37.50	37.50
2	.	.	.	.	.	.	.
3	37.20	37.20	37.30	37.30	37.30	37.40	37.40
4	37.50	37.50	37.60	37.70	37.80	37.80	37.90
5	37.00	37.10	37.20	37.20	37.20	37.30	37.00
6	37.10	37.20	37.20	37.20	37.20	37.30	37.20
7	37.30	37.40	37.50	37.60	37.70	37.80	37.90
8	37.30	37.40	37.50	37.60	37.70	37.70	37.80
9	37.20	37.10	37.10	37.00	36.40	35.90	35.40
10	37.30	37.30	37.30	37.30	37.40	37.20	37.20
11	36.80	36.80	36.90	36.80	36.40	35.00	34.80
12	37.60	37.60	37.60	37.60	37.60	37.60	37.60
13	37.20	37.30	37.30	37.30	37.40	37.40	37.40

	ccr40	ccr45	ccr50	ccr55	ccr60	ccrpt	cskpr
1	37.50	37.50	37.60	37.50	37.50	37.30	32.62
2	.	.	.	.	.	.	.
3	37.50	37.50	37.50	37.50	37.60	37.60	30.68
4	37.90	37.90	37.80	37.80	37.80	37.80	30.75
5	37.30	37.30	37.30	37.30	37.20	37.20	31.99
6	36.80	36.00	36.00	35.90	35.90	35.70	31.48
7	38.00	38.00	38.00	38.10	38.10	38.10	33.00
8	37.80	37.80	37.80	37.70	37.70	37.70	31.78
9	35.20	35.10	35.10	35.10	35.10	.	33.71
10	37.00	36.60	35.80	35.80	35.50	35.80	31.62
11	.	.	.	.	.	35.30	.
12	37.50	37.50	37.40	37.40	37.30	37.20	32.14
13	37.30	37.20	37.00	36.80	36.20	36.10	33.10

	csk0	csk5	csk10	csk15	csk20	csk25	csk30
1	30.26	28.15	27.72	27.67	25.56	25.56	25.09
2	.	.	.	.	.	.	.
3	29.06	24.86	23.65	22.44	21.40	20.09	19.77
4	29.74	26.04	24.65	23.72	22.76	21.64	21.03
5	29.90	25.94	23.88	23.01	22.30	21.85	21.43
6	28.01	26.06	24.88	24.14	23.27	22.06	21.39
7	31.29	27.84	26.09	25.29	23.98	23.92	23.65
8	29.94	26.70	25.08	24.34	23.22	22.33	22.31
9	32.12	29.28	28.09	27.31	26.86	25.30	24.76
10	28.51	24.26	23.43	23.07	22.79	23.18	23.68
11	.	.	.	.	.	.	.
12	29.19	24.68	25.07	22.92	21.83	20.70	19.68
13	30.20	26.90	25.38	24.35	23.69	23.73	23.10

	csk35	csk40	csk45	csk50	csk55	csk60	cskpt
1	25.00	24.96	24.60	24.25	24.33	24.08	.
2	.	.	.	.	.	.	.
3	19.41	19.23	19.23	19.02	19.09	18.97	22.35
4	20.41	20.27	20.23	20.03	20.16	20.01	23.02
5	21.66	21.29	21.17	21.11	20.95	20.74	23.85
6	21.38	21.33	21.52	21.57	21.30	21.18	24.41
7	23.06	22.66	22.48	21.99	21.19	21.63	24.99
8	21.99	21.96	21.21	21.09	20.92	21.24	24.00
9	24.42	24.33	23.90	23.81	23.70	23.87	.
10	23.40	23.51	23.31	23.27	23.28	23.30	25.99
11	.	.	.	.	.	.	.
12	19.42	19.42	19.35	19.24	19.93	19.94	23.26
13	23.21	23.36	22.99	23.25	22.91	22.80	27.12

	cthcompr	cthcom0	cthcom5	cthcom10	cthcom15	cthcom20	cthcom25
1	1	3	4	4	4	4	4
2	.	.	.	.	.	.	.
3	1	4	4	4	4	4	4
4	1	4	4	4	4	4	4
5	1	3	3	4	4	3	3
6	1	3	3	3	3	2	2
7	1	3	3	4	3	3	3
8	1	4	4	4	4	4	4
9	1	3	4	3	3	3	3
10	1	4	4	4	4	4	4
11	1	3	3	4	3	4	3
12	1	3	4	4	4	4	4
13	1	2	3	4	4	4	4

	cthcom30	cthcom35	cthcom40	cthcom45	cthcom50	cthcom55	cthcom60
1	4	3	3	3	3	3	3
2	.	.	.	.	.	.	.
3	4	4	4	4	4	4	4
4	4	4	4	4	4	4	4
5	3	3	3	3	3	2	3
6	2	2	2	2	2	2	2
7	3	3	3	2	2	2	2
8	4	4	4	4	4	4	4
9	2	2	3	2	3	3	3
10	4	4	4	4	4	4	4
11	4	.	.	.	.	.	.
12	4	4	4	4	4	4	4
13	4	4	4	4	4	4	3

	cthcompt	cthsenpr	cthsen0	cthsen5	thsen10	thsen15	thsen20
1	1	0	-3	-4	-3	-3	-3
2	.	.	.	.	.	.	.
3	1	0	-3	-4	-4	-4	-4
4	1	0	-4	-4	-4	-4	-4
5	2	0	-2	-3	-4	-4	-3
6	1	0	-3	-3	-4	-3	-3
7	1	0	-3	-3	-4	-3	-4
8	1	0	-4	-4	-4	-4	-4
9	.	0	-4	-4	-3	-2	-3
10	1	2	-4	-4	-4	-4	-4
11	2	2	-4	-4	-4	-4	-4
12	2	0	-3	-4	-4	-4	-4
13	2	0	-3	-3	-4	-4	-4

	thsen25	thsen30	thsen35	thsen40	thsen45	thsen50	thsen55
1	-3	-3	-3	-3	-3	-3	-3
2	.	.	.	.	.	.	.
3	-4	-4	-4	-4	-4	-4	-4
4	-4	-4	-4	-4	-4	-4	-4
5	-3	-3	-3	-3	-3	-3	-3
6	-2	-2	-1	-1	-1	-1	-1
7	-3	-3	-3	-3	-3	-2	-2
8	-4	-4	-4	-4	-4	-4	-4
9	-3	-2	-2	-2	-2	-3	-4
10	-4	-4	-4	-4	-4	-4	-4
11	-4	-4	.	.	.	.	.
12	-4	-4	-4	-4	-4	-4	-4
13	-4	-4	-4	-4	-4	-4	-4

	thsen60	thesenpt	rpe25	rpe30	rpe35	rpe40	rpe45
1	-3	0	12	11	9	9	9
2	.	.	.	.	.	.	.
3	0	6	6	6	6	6	6
4	-4	0	6	6	6	6	6
5	-3	-2	11	9	9	9	9
6	-1	1	9	9	8	8	7
7	-2	0	6	6	6	6	6
8	-4	2	6	6	6	6	6
9	-3	.	13	11	11	11	11
10	-4	2	7	7	7	7	7
11	.	2	7	7	.	.	.
12	-4	2	9	9	9	9	9
13	-4	-2	.	.	.	.	.

	rpe50	rpe55	rpe60	hrvo2max
1	8	8	7	203.00
2	.	.	.	195.00
3	6	6	6	185.00
4	6	6	6	191.00
5	8	8	7	203.00
6	7	6	6	195.00
7	6	6	6	185.00
8	6	6	6	191.00
9	13	13	13	206.00
10	7	7	7	200.00
11	.	.	.	194.00
12	9	9	9	196.00
13	.	.	.	.