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A COMPARISON OF ENDOGENOUS VERSUS EXOGENOUS HEATING ON THE SUBSEQUENT DEVELOPMENT OF HYPOTHERMIA

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Thesis submitted to the
Faculty of Graduate and Postdoctoral Studies
in partial fulfillment of the degree of Master of Arts in Human Kinetics,
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September 2000

Abstract

This study investigated the effect of endogenous and exogenous heating on body core cooling rate, afterdrop and re-warming rate in humans during subsequent cold-water immersion. On three separate days, following 30 min baseline resting at an ambient temperature of 25°C, seven subjects were either: 1) seated upright for 15 min rest (Control); 2) exercised 15 min on a cycle ergometer at 70% VO_2 max (Exercise); or, 3) immersed 16.8 ± 6.2 min in warm water (40°C) (Warm-water immersion) to an esophageal temperature (T_{ES}) similar to that of end of exercise. Subjects were then cooled in water (7°C) to a T_{ES} of 34.5°C and rewarmed by spontaneous shivering (shivering thermogenesis) in air. There was a 1.3-fold increase in the overall body core cooling rate during Exercise as compared to Control ($P<0.01$). Warm-water immersion demonstrated the largest difference in overall cooling rate above Exercise and Control with a 2.5- and 3.3-fold increase, respectively ($P<0.01$). The greatest difference between trials occurred within the initial 15 min (i.e., a 2.0- and 4.9-fold increase above Control for Exercise and Warm-water immersion, respectively). There was no significant difference in the T_{ES} afterdrop (~0.5°C) nor afterdrop duration (~15 min). Similarly, rewarming rates were almost identical under all conditions (~3.1°C·h^{-1}). These data show that pre-warming can have a detrimental effect on survival time during a subsequent cold-water immersion.

Key words: hypothermia, thermoregulation, cooling rate, afterdrop, rewarming, exercise, warm-water immersion

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List of Abbreviations

T_{CO} = Core Body Temperature

T_{ES} = Esophageal Temperature

T_{AMB} = Ambient Temperature

RH = Relative Humidity

EF = Effector Mechanism

POA = Pre-optic Area of the Anterior Hypothalamus

AH = Anterior Hypothalamus

PH = Posterior Hypothalamus

VO_2 = Oxygen Consumption

VO_2 max = Maximal Aerobic Capacity (Maximum Oxygen Consumption)

VE = Ventilation

RER = Respiratory Exchange Ratio

OCR = Overall Cooling Rate

CR_I-15 = Cooling Rate during the Initial 15-minutes of the Cooling Period

CR_F-15 = Cooling Rate during the Final 15-minutes of the Cooling Period

ADCR = Afterdrop Cooling Rate

ΔCR = Change in Cooling Rate Between CR_F-15 and ADCR

AD = Afterdrop

ADL = Length of the Afterdrop

ORR = Overall Rewarming Rate

RR_F-15 = Rewarming Rate during the Final 15-minutes of the Rewarming Period

Acknowledgements

The following acknowledgements are to identify those people who assisted with my graduate studies and research:

- My advising professor Dr. Kenny, for his assistance with all aspects of my research experience.
- Dwayne N. Jackson, for his assistance with my data collection.
- Dr. Reardon and Dr. Robertson, for their technical assistance throughout my graduate studies.

This research was supported by the Natural Science and Engineering Research Council (Canada) and the University of Ottawa Grants Committee.

1.0 INTRODUCTION

The maintenance of core temperature (T_{CO}) is achieved by balancing heat production against heat loss. Cellular metabolism is the primary means of heat production and thus is important in the maintenance of core temperature. Exercise and warm exposure can significantly increase the body's cellular metabolism, thus increasing heat production. As a result heat loss mechanisms (i.e., cutaneous vasodilation and sweating) are stimulated in an attempt to transfer this excess heat from the body core to the superficial shell, where it is removed by means of conduction, convection and evaporation. It has been demonstrated that stimulation of these heat loss mechanisms by exercise and warm exposure prior to exposure to a cold environment (i.e., cold water immersion) could result in an increase in cooling and thus have a detrimental effect on survival in cold conditions (McDonald et al., 1984; Windle et al., 1994; Castellani et al., 1999).

Recent studies investigating the effects of prior warming (exercise or warm exposure) on the cooling rates during the subsequent cold-water immersion have reported inconsistent results and are therefore deemed inconclusive. According to McDonald et al. (1984) vigorous pre-immersion exercise may shorten survival time in cold water due to a significant increase in core-cooling rate. These conclusions were supported by Castellani et al. (1999) when active and passive heating preceded cold air exposure. In contrast, Windle et al. (1994) compared the effects of warming by active (exercise) and passive (warm-water immersion) means on the subsequent responses to cold water immersion and concluded that a raised deep body temperature

due to active or passive warming does not alter short- or long-term survival prospects in cold water.

The inconsistent results reported in the previous studies may have been the result of differences in their experimental methods. According to Castellani et al. (1999) and Windle et al. (1994) it is important to equilibrate pre-immersion esophageal temperatures for an accurate comparison of cooling rates. Kenny et al. (1996) have demonstrated that the increased esophageal temperature following warm-water immersion may return to its pre-immersion baseline value within ten minutes of exiting the warm-water bath. Thus, if it is important to equilibrate the temperature gradients to compare the experimental trails, immediate cold exposure is necessary following the warm-water heating condition. However, this was not the case in the study conducted by Castellani et al. (1999), where following an exercise and a warm-water heating treatment, subjects were exposed to a ~20 minute transition period in an ambient temperature of 22.8°C prior to the cold air exposure (4°C). Thus it is possible that the initial period of cooling in the warm-water condition of Castellani et al. (1999) was missed, which may explain the differences between their results and that of Windle et al. (1994). In the study by McDonald et al. (1984) a warm-water immersion treatment to equilibrate the pre-immersion esophageal temperatures was not utilized, thus providing inconclusive results.

Furthermore, all of the previous studies failed to evaluate core-cooling rates in severely hypothermic individuals (i.e., final core temperature of 34.5°C). They were limited to a core temperature decrease of ~0.8°C or an exit temperature of 36.3°C which is considered normothermic and at best, slightly hypothermic.

There are a few possible physiological explanations for an increased cooling rate when cold-water immersion follows exercise or warm-water immersion, including a greater temperature gradient between the skin surface and the cold-water bath. Second, a reduction in the insulative properties of the skin and muscle as a result of increased blood flow. Finally, subcutaneous fat plays an important role in thermal insulation. It would seem likely that perfusion through the subcutaneous fat would further decrease the total body insulative capacity, thus increasing heat loss by means of convection.

Extensive research has been conducted on the post-exercise thermal responses involved in the removal of heat, however, little has been completed on how these responses effect the rate of heat loss during subsequent cold exposure. Further, no research has been conducted on the afterdrop and re-warming rates following heat stress prior to cold-water immersion. This research could be important in estimating the survival time of hypothermic individuals who experience prior heating, such as pilots, sportsmen, and watercraft enthusiasts.

Thus, because of the present inconsistencies in the understanding of prior heating (exercise or warm exposure) on the core-cooling rates during cold water immersion and because of the lack of knowledge on the afterdrop and rewarming rates when exercise precedes cold water immersion, further investigation is warranted.

1.1 REVIEW OF LITERATURE

1.1.1 General Thermoregulatory Control Mechanisms

The internal body temperature is maintained by balancing heat production against heat loss. There are two classes of thermoregulatory responses that function to maintain this balance. There are physiological responses, which are the secondary functions of organ systems, and behavioral responses. The physiological responses modify rates of heat transfer from the core to the body surface, or modify the level of heat generation within the tissues (Hammel, 1968). The behavioral responses are the coordinated activity of the whole animal in selecting or creating a microenvironment in which optimal internal temperature may be achieved without the assistance of the physiological mechanisms (Hammel, 1968).

The physiological effector mechanisms which operate to maintain thermal homeostasis when one is subjected to a hypothermic medium (i.e., cold water immersion) are cutaneous vasoconstriction, shivering thermogenesis, and non-shivering thermogenesis (Banet & Hensel, 1977; Boulant & Dean, 1986; Cabanac, 1975; Hammel, 1968; Hynson et al., 1993; Kanosue et al., 1994; Jansky, 1973; Marques et al., 1981; Thornhill & Halvorson, 1994a; Thornhill & Halvorson, 1994b). As well, extensive study has been conducted on the heat loss mechanisms; mainly cutaneous vasodilation and sweating, which function to maintain body core temperature (T_{CO}) in a hyperthermic environment (Boulant & Dean, 1986; Cabanac, 1975; Hammel, 1968; Gisolfi & Wenger, 1984; Johnson, 1992). These effector mechanisms that function to increase heat production or heat loss are stimulated by

sensory information from the preoptic area (POA), anterior hypothalamus (AH), and the posterior hypothalamus (PH), which with other central and peripheral thermoreceptor information, are involved in the generation of this effector stimulus. Research indicates that increasing the POA temperature above its normal level evokes heat-loss responses (i.e., cutaneous vasodilation and sweating); as well, cooling the POA below resting levels evokes heat production responses (i.e., cutaneous vasoconstriction, shivering and non-shivering thermogenesis) (Boulant & Dean, 1986). Hammel (1968) states that no other part of the central nervous system may be substituted to evoke a response similar to that of the POA/AH. Thus it would seem evident that the POA/AH serves as the thermostatic body temperature control center (Boulant, 1981; Boulant & Dean, 1986; Cabanac, 1975; Guyton & Hall, 1996; Hammel, 1968). Operating in conjunction with the thermoregulatory control center is the posterior hypothalamus, which has been suggested to act in the integration of both the central and peripheral thermoreceptive information and the neural information from the POA/AH (Hardy, 1973; Guyton & Hall, 1996; Puschmann & Jessen, 1978; Kushakov, 1980). Furthermore, it would seem likely that the integrative property of the PH would be important in the propagation of the central effector signal (Bell et al., 1981; Hardy, 1973). The thermosensitivity of the PH reacts in a fashion opposite to the POA and thus constitutes a positive feedback loop, which counteracts the activity of the negative feedback loop of the control center. In effect if the POA/AH were cooled producing a heat production response (i.e., shivering), then cooling the PH would inhibit the shivering and decrease that heat production (Puschmann &

Jessen, 1978). Thus, this regulatory action of the PH would seem to be important in maintaining the thermal homeostasis of the body.

The central deep tissue thermoreceptors located in the spinal cord, abdominal viscera, and great veins, and the peripheral skin thermoreceptors provide information on the T_{CO} and skin temperature, respectively. These thermoreceptors are responsible for both warm and cold thermal information, which is sent via the afferent sympathetic neurons to the POA/AH and PH. It is in the hypothalamus that this information is compared to a reference temperature (Gisolfi & Wenger, 1984; Hammel, 1968). This reference is often referred to as the hypothalamic temperature or set point temperature. The difference between the combined central and peripheral neural temperature information and the hypothalamic reference temperature yields an error signal (Hammel, 1968). This error signal, depending on its magnitude and direction, stimulates a specific effector mechanism(s) to correct for this difference (Cabanac, 1975).

Depending on the internal and environmental stresses placed on the body, the "normal" T_{CO} can deviate $\pm$ 0.5°C from the hypothalamic temperature (Brengelmann, 1973). This hypothalamic reference temperature is more of a mean temperature of all of these fluctuations within this range with specified thresholds for the various effector responses. A possible reason for the normal fluctuations in T_{CO} can be described by thermoregulatory time lag (Sawka & Wenger, 1988). Thermoregulatory time lags can come from various sources. First, the time needed for sensory information to reach the hypothalamus. Secondly, the delay in the hypothalamus in interpreting all of the thermal and non-thermal information. Lastly, the time needed

for action potentials from the hypothalamus to reach the effector (Sawka and Wenger 1988). The combination of these thermoregulatory time lags may account, at least in part, for the variations in T_{CO}, as well as the time required for T_{CO} to reach the thresholds for the different effector responses.

It is the intent of this review to summarize the relevant literature and thus provide an understanding on general temperature regulation, thermoregulatory heat producing mechanisms, and the effects of exercise on these mechanisms.

1.1.2 Hypothalamic Regulation of Body Temperature

It has been generally accepted by most physiologists that the hypothalamus, more specifically the preoptic area and the anterior hypothalamus play important roles in the regulation of the body temperature. It has also been proposed that the posterior hypothalamus is important in the integration of the afferent neural signals as well as in the generation of the central effector signal. The POA/AH and PH are involved in central temperature detection and the stimulation and inhibition of appropriate effector responses, which together operate to regulate internal body temperature. Hammel (1968) states the ability of the hypothalamus to transduce its own temperature into neural impulses, and then to activate an effector mechanism(s), depending upon the sign and magnitude of its temperature displacement, indicates its importance in the regulation of body temperature. Refinetti and Carlisle (1986) and Halvorson and Thornhill (1993) indicate that through anterior and posterior hypothalamic heating and cooling in rats, metabolic and behavioral responses can be

stimulated. Thus supporting the involvement of both the anterior and posterior hypothalamus in temperature regulation.

1.1.2.1 Role of the Anterior Hypothalamus – Preoptic Area

The anterior hypothalamus (AH) and preoptic area (POA) have been shown to respond to both warm and cold thermal stimulation. Warming or cooling the POA/AH beyond a set temperature or threshold activates the appropriate effector response, thus counteracting the thermal stimulus (Berner & Heller, 1998; Boulant, 1981; Boulant & Dean, 1986; Cabanac, 1975; Hammel, 1968; Refinetti & Carlisle, 1986). This is accomplished through the many thermosensitive neurons that exist in the POA/AH. These neurons are able to sense changes in internal temperature and then initiate the appropriate effector response (Boulant, 1981).

The POA/AH contains warm and cold thermosensitive neurons as well as temperature insensitive ones (Boulant, 1981; Boulant & Dean, 1986). The proportions of these neuron types in the hypothalamus are approximately 30% warm sensitive, and 10% cold sensitive with the remaining 60% insensitive to thermal stimulus (Boulant & Dean, 1986). Boulant (1981) states that the neural firing rate and the range of optimal thermosensitivity of the POA warm-sensitive neurons is determined by the amount of afferent input that they receive. Thus warm-sensitive neurons that receive little afferent input have low firing rates and those that receive the greatest amount of afferent input have the highest firing rates. As well, Boulant & Dean (1986) suggest that warm-sensitive neurons that have low firing rates, which tend to be sensitive to temperatures above thermoneutral, quite possibly control heat

loss responses. Warm-sensitive neurons with moderate firing rates that tend to be sensitive to temperatures both above and below thermoneutral suggest possible heat-retention responses, such as changes in behavior and skin blood flow. Finally, warm sensitive neurons that receive the greatest amount of afferent input and thus have the highest firing rates are primarily sensitive to temperatures below thermoneutral, suggesting possible control of the heat production responses (Boulant & Dean, 1986). It would seem logical that the thermosensitive neurons that received the greatest amount of afferent input would be responsible for the heat production responses due to the fact that the majority of the deep tissue and skin thermoreceptors detect mainly cold sensations (Guyton & Hall, 1996).

The cold-sensitive neurons receive afferent information from the central and peripheral thermoreceptors and from the high firing warm-sensitive neurons (Boulant, 1981). However the response of the cold-sensitive neuron to the skin and spinal temperatures is generally opposite to that of the warm-sensitive neurons (Boulant, 1981). Skin warming not only decreased the neuron's firing rate, but also decreased its thermosensitivity (Boulant & Hardy, 1974). Thus indicating that the cold-sensitivity of these neurons may actually be due to inhibitory synaptic inputs from nearby warm-sensitive neurons. Therefore POA cooling would decrease the firing rate in the warm-sensitive neurons resulting in less inhibition of the cold sensitive neuron, resulting in increased heat production (Boulant & Dean, 1986). The combination of the decreased inhibition from warm-sensitive neurons and the increased excitatory stimulus from the central and peripheral thermoreceptors evokes an increased firing rate and thermosensitivity of the cold-sensitive neurons (Boulant

& Dean, 1986). It would appear that it is the combination of the warm- and cold-sensitive neuron's ability to sense changes in temperature and provide a stimulus for an appropriate effector response, which enables the POA/AH to have central thermoregulatory control.

1.1.2.2 Posterior Hypothalamic Controls

The posterior hypothalamus has been shown to be involved in the heat production responses during exposure to cold (Keller & Hare, 1932; Bell et al., 1981). Through the removal of the posterior hypothalamus in dogs, Keller and Hare (1932) indicated the prepared animals had little, if any, ability to maintain body temperature near thermoneutral when exposed to cold. The animal could however, retain a slightly impaired regulation against hyperthermia. Bell et al. (1981) found that T_{CO} decreased when rats with posterior hypothalamic lesions were exposed to cold, whereas the control group of rats was able to maintain their T_{CO}. Hardy (1973) and others (Bell et al., 1981; Guyton & Hall, 1993; Puschmann & Jessen, 1978; Kushakov, 1980) state that it seems likely that the caudal posterior hypothalamus is the area where integration of the various neural sensory signals is completed and the central effector signal is developed.

The PH has been shown to react oppositely to that of the anterior hypothalamus, in that, warming the PH stimulates increased levels of heat production and cooling inhibits the heat production responses (Puschmann & Jessen, 1978). Puschmann and Jessen (1978) studied the effects of temperature displacements in the anterior and posterior hypothalamus on heat production in goats and found that the

warming of the posterior hypothalamus increased the stimulation of heat production through the cooling of the anterior hypothalamus or through peripheral cooling. Further, heat production was inhibited when the PH was cooled and when the cold stimulus was removed from the PH, heat production immediately returned to its previous level. Therefore the PH acts as a positive feedback loop, counteracting the negative feedback loop of the control center.

Halvorson and Thornhill (1993) showed that electrical stimulation of the posterior hypothalamus (PH) of anesthetized rats maintained at 37°C evokes shivering thermogenesis. As well, rats exposed to cold prior to the PH electrical stimulus required less stimulation to induce the shivering response. They also have shown that PH stimulation of animals already shivering due to cold exposure augmented the activity of the shivering thermogenesis (Halvorson & Thornhill, 1993). Others propose that the posterior hypothalamus serves an integrative function with respect to behavioral thermoregulatory responses, but is not able to elicit the autonomic thermoregulatory responses when stimulated by increases or decreases in hypothalamic temperature (Refinetti & Carlisle, 1986). This difference in the literature may be explained by the characteristics of the thermosensitive neurons of the posterior hypothalamus shown by Kushakov (1980). Kushakov (1980) found that due to the low percentage of neurons capable of central temperature detection in the PH, thermoregulatory responses might not be induced through temperature stimulation. Another reason for the differences may be due to the placement of the thermodes within the posterior hypothalamus.

In light of the research on the posterior hypothalamus it is reasonable to support the view proposed by Hardy (1973) in that the posterior hypothalamus has a important role in the regulation of body temperature through the integration of both the central and peripheral thermoreceptive information and the neural information from the POA/AH. Further it would seem likely that the integrative property of the PH would be important in the propagation of the central effector signal.

1.1.3 Mechanisms for Increasing Body Temperature

Metabolism, which is the primary means of heat production, is the sum of all chemical reactions in the body (Vander et al., 1994). Thousands of these chemical reactions occur each instant throughout the body, of which the principle by-product is heat. The rate of heat production is influenced by the metabolic rate of all of the cells in the body. Factors that cause increases in metabolic rate include muscle activity (including muscle contraction during shivering), thyroxine, growth hormone and testosterone, as well epinephrine, norepinephrine, and sympathetic stimulation, and increased chemical activity in the cells themselves, especially when the cell temperature increases (Guyton & Hall, 1996).

The temperature increasing mechanisms of the body can be divided into two categories, heat production mechanisms and heat conservation mechanisms. The mechanisms that function to increase heat production, which include the increased rates of metabolism described above, are shivering and non-shivering thermogenesis. The heat conservation mechanisms are vasoconstriction, the insulative properties of tissue, and piloerection. Vasoconstriction, shivering and non-shivering

thermogenesis, and the insulation properties of tissue will be described in detail in the following sections. However because piloerection is a less important heat conservation mechanism in humans, only a brief description will follow.

Piloerection, meaning hairs "standing on end", is accomplished through sympathetic stimulation causing the arrector pili muscles attached to the hair follicles to contract (Guyton & Hall, 1996). This upright projection of the hairs allows animals to entrap a thicker layer of air next to their skin. This air is then heated through conduction providing an insulative effect, thus greatly depressing the rate of transfer of heat from the skin to the environment.

1.1.3.1 Skin Vasoconstriction

The reduction of blood flow to the skin decreases conductive and evaporative heat loss from the body to the environment, thus acting to maintain internal body temperature. Body cooling causes cutaneous vasoconstriction via a generalized increase of adrenergic vasoconstrictor activity (Oberle et al., 1988). When hypothalamic temperature or peripheral skin temperature decreases, an effector signal is stimulated from the POA/AH sympathetic centers inhibiting of the positive feedback of the posterior hypothalamus. The effector signal then travels via the efferent neural pathway to the smooth muscles surrounding the arterioles, metarterioles, and precapillary sphincters, thus stimulating vasoconstriction (Cabanac, 1975; Guyton & Hall, 1996; Hammel, 1968; Oberle et al., 1988). Smooth musculature is not only stimulated by sympathetic nervous activity, but also has been shown to contain a large degree of spontaneous activity stimulated mainly through

hormone interaction (Vander et al., 1994). The temperature at which the effector response is stimulated is the threshold for vasoconstriction. The stimulation of this effector response depends on both skin and core temperature and can be increased or decreased through various factors (Cheng et al., 1995; Kurz et al., 1995; Oberle et al., 1988). The contraction of these smooth muscles decreases the vessel's radius, thus decreasing local blood flow.

The mechanisms that control skin vasoconstriction in response to cold include the sympathetic nervous system and hormone interaction (Boulant & Gonzalez, 1977; Cabanac, 1975; Guyton & Hall, 1996; Hammel, 1968; Oberle et al., 1988; Vander et al., 1994). The sympathetic nervous system is continually active. The basal rates of this activity are known as sympathetic tone. The value of sympathetic tone is that it allows a single nervous system to increase or decrease the activity of a stimulated organ or tissue (Guyton & Hall, 1996). Systemic arterioles are a good example of this, in that sympathetic tone usually keeps almost all of the systemic arterioles constricted to about one half their maximum diameter (Vander et al., 1994). By increasing the degree of sympathetic stimulation, these vessels can be constricted even more and by inhibiting normal tone, they can be dilated (Guyton & Hall, 1996; Vander et al., 1994). Most arterioles receive numerous sympathetic postganglionic nerve fibers, which release norepinephrine. This neurotransmitter diffuses to the vascular smooth muscle, where it combines with the alpha-adrenergic receptors causing vasoconstriction (Guyton & Hall, 1996; Vander et al., 1994).

Hormones are able to stimulate the contraction of the smooth muscle fibers when the muscle cell membrane contains hormone-gated excitatory receptors for the

respective hormone (Guyton & Hall, 1996). Some of the more important blood-borne hormones that stimulate vasoconstriction are norepinephrine, epinephrine, angiotensin, and vasopressin. The sympathetic nervous system is stimulated in generally all parts of the body during stress and exercise. The stimulation of the sympathetic nerves to the adrenal medullae releases large amounts of norepinephrine and epinephrine, which then circulates to all of the tissues of the body via the blood (Guyton and Hall, 1996; Vander et al., 1994). Both the epinephrine and norepinephrine circulating in the blood will have virtually the same effects on circulation as direct sympathetic stimulation, except that the effects will last five to ten times longer due to the slow removal of these hormones from the blood (Guyton & Hall, 1996). Thus circulating norepinephrine causes constriction of essentially all of the blood vessels in the body. As well, the norepinephrine will cause increased activity of the heart, thus increasing arterial pressure. The circulating epinephrine, through greater excitation of the heart, increases arterial pressure as well, but causes a weaker constriction of the blood vessels compared to norepinephrine (Guyton & Hall, 1996).

Angiotensin has also been shown to constrict most of the arterioles and also increases the activity of the sympathetic nervous system. Angiotensin is one of the most powerful vasoconstrictor substances, thus blood flow to any given area can be greatly depressed through the powerful constriction of the small arterioles (Vander et al., 1994). The main action of angiotensin is that it acts simultaneously on all arterioles of the body to increase total peripheral resistance, thereby increasing arteriole pressure (Guyton & Hall, 1996).

Vasopressin or antidiuretic hormone is formed in the hypothalamus and is transported down through the center of the nerve axons to the posterior pituitary gland, where it is secreted to the blood (Guyton & Hall, 1996). Although vasopressin is the body's most powerful constrictor substance, only minute amounts are secreted and thus play little role in vascular control (Guyton & Hall, 1996).

1.1.3.2 Shivering Thermogenesis

The initial response of skeletal muscle to the cold is a gradual and general increase in muscle contraction (Tipton, 1989). Further cold stimulus initiates shivering which is the rhythmical oscillation of skeletal muscle, causing tremors. These tremors, occurring at a rate of about 10 to 20 per second, can generate a four to five fold increase in heat production, thus operating to maintain or increase body temperature when exposed to the cold (Guyton & Hall, 1996; Hammel, 1968; Martin & Cooper, 1981; Vander et al., 1994). The increase in heat production from shivering thermogenesis is the result of the extra metabolic rate that is associated with an increase in muscular activity.

Hammel (1968) indicates that the shivering tremor in response to body cooling seems to require an intact posterior hypothalamus, activated in turn by POA tissue. It has been proposed that a primary motor center for shivering exists in the dorsomedial portion of the posterior hypothalamus near the wall of the third ventricle (Guyton & Hall, 1996). As well, it is thought that the PH is normally inhibited by the heat center in the POA/AH and is stimulated by cold signals from the skin and spinal cord (Guyton & Hall, 1996; Hammel, 1968; Mekjavic & Morrison, 1985).

When an effector signal is stimulated it travels through the bilateral ducts down to the brain stem, into the lateral columns of the spinal cord, and, finally, to the anterior motor neurons (Guyton & Hall, 1996). The anterior motor neurons, once stimulated, increase the tone within the skeletal muscles. When the tone rises above some critical level shivering begins (Guyton & Hall, 1996).

1.1.3.3 Non-Shivering Thermogenesis

Non-shivering thermogenesis can be defined as an increase in metabolic rate, and thus heat production, through the secretion of epinephrine, and norepinephrine, with some contribution to thyroid hormone. Either sympathetic stimulation or circulating epinephrine and norepinephrine in the blood can cause an immediate increase in the rate of cellular metabolism. It has been shown that metabolic rate can increase by as much as 100% due to the secretion of epinephrine by the adrenal medullae (Guyton & Hall, 1996). This increased level of these hormones has the ability to uncouple oxidative phosphorylation, which means that excess foodstuffs are oxidized and thereby release energy in the form of heat instead of causing adenosine triphosphate to be formed (Gale, 1973; Jansky, 1973). The degree of non-shivering thermogenesis seems almost directly proportional to the amounts of brown fat in the tissues of the animal (Hammel, 1968). This type of fat contains large numbers of special mitochondria, which is where the uncoupled oxidation occurs (Guyton & Hall, 1996).

It has been shown that rats exposed to a cold environment for several weeks can exhibit a 100% to 500% increase in heat production as a result of non-shivering

thermogenesis, whereas an acutely exposed rat may increase its heat production by one third that value (Jansky, 1973; Gale, 1973). However in adult humans, because there is almost no brown fat, non-shivering thermogenesis may account for only a 10% to 15% increase in heat production (Guyton & Hall, 1996). Whereas in infants, who do have larger amounts of brown fat, can increase their heat production by up to 100% (Guyton & Hall, 1996).

Long-term increases in heat production can be accomplished through an increase in the output of thyroxine. Studies indicate that cooling the POA/AH increases the production of thyrotropin-releasing hormone by the hypothalamus (Marques et al., 1981). This hormone reaches the anterior pituitary gland through the hypothalamic portal veins, where it stimulates the release of thyroid-stimulating hormone (Guyton & Hall, 1996). The thyroid-stimulating hormone in turn stimulates the thyroid gland to release thyroxine, which can increase the metabolic activity throughout the body (Vander et al., 1994). This, however, takes several weeks because the thyroid gland must hypertrophy in order for it to reach a new level of thyroxine secretion (Jansky, 1973). Animals exposed to a cold environment for several weeks can increase the size of their thyroid gland by as much as 40% (Jansky, 1973; Gale, 1973). Little human research exists on the influence of the thyroid methods of cold adaptation, thus the importance is not known. However, there is evidence of increased metabolic rate due to increased levels of thyroxine in the Inuit people who live in colder climates year round (Guyton & Hall, 1996).

1.1.3.4 Insulative Properties Involved in Heat Maintenance

The exposed surface area relative to the total mass of the individual influences heat transfer from the body to the environment (Glickman-Weiss et al., 1991, Strong et al., 1985). So those who have a small surface-area to body-mass ratio and those with more fat are less susceptible to hypothermia.

The body's insulation consists of the skin, subcutaneous tissues, the subcutaneous fat of the tissues and muscle (Veicsteinas, 1982). Body insulation is an important aspect of heat conservation due to its reduced ability to conduct heat (Hayward & Keatinge, 1981). Veicsteinas et al. (1982) has proposed that although subcutaneous fat is important in heat conservation, it accounts for only about 10-15% of the insulative properties of the tissue during cold-water immersion. The remainder of the insulative ability is most likely due to poorly perfused muscle (Veicsteinas et al., 1982). During complete vasoconstriction the insulative properties of the tissues in an average male is equal to approximately three quarters the insulating properties of a normal suit of clothes and is still better in women (Guyton & Hall, 1996). This insulation allows for the maintenance of T_{CO} even with skin temperatures approaching the temperature of the surroundings (Hayward & Keatinge, 1981).

1.1.4 Thermal Heat Balance

A large portion of the energy that is generated within the various systems of the body is degraded to heat. The majority of this heat is produced in the deep tissues, especially in the liver, brain, heart, and the skeletal muscles during exercise. Thus all of these metabolically active tissues are important in maintaining thermal

balance through the production of heat. As well, when the temperature of the surroundings is greater than that of the body, heat will be transferred to the body from these surroundings. If the internal and environmental heat gains exceed the heat loss, then T_{CO} will begin to rise. Thus in order for thermal homeostasis to be maintained there must be mechanisms that are responsible for liberating the body of this heat. These heat transfer mechanisms are radiation, conduction and evaporation. The rates of this heat transfer via conduction and evaporation can be increased further by means of convection.

1.1.4.1 Physical Mechanisms of Heat Transfer

Radiation is the transfer of heat between objects through electromagnetic waves. The human body radiates heat in all directions and at normal room temperature will lose about 60% of the excess body heat through radiation at rest (Guyton & Hall, 1996). As well, all objects that are not at absolute zero radiate infrared heat waves. Thus if the temperature of surrounding objects is greater than T_{CO}, the body will receive radiational heat.

Another heat transfer mechanism is conduction, which is the transfer of heat energy between objects or tissues of different temperatures in direct contact with one another. Therefore heat that is produced in the deep tissues of the body can be conducted through adjacent tissue until it reaches the body surface (Vander et al., 1994). Conduction will also occur between the body surface and any object or environment that is in direct contact with it. The temperature of the object or

environment determines the degree to which heat is gained or lost, however heat loss by conduction to an object represents only about 3% of the total body heat loss.

When water evaporates from the skin, 0.58 kcal of heat is lost for each gram of water that evaporates (Guyton & Hall, 1996). Heat is continually lost through the evaporation of water from the skin and respiratory tract. This constant evaporation removes about 10% of the total basal metabolic heat that is produced, and is known as insensible heat loss. Insensible heat loss is an insufficient heat loss mechanism when the T_{CO} begins to rise, as it does in an extremely hot environment or during exercise. When the T_{CO} reaches its threshold temperature sweating begins, increasing the availability of water for evaporation. The onset of sweating due to exercise can account for as much as four times the heat loss compared to rest.

The transfer of heat via conduction and evaporation can be increased further by means of convection. Convection is the transfer of heat from one place to another by the motion of a heated substance. Air molecules adjacent to the skin are heated or humidified by conduction or evaporation, respectively. These molecules are then carried away by convection allowing for new unheated, dry molecules to take their place. Thus conduction and evaporation become a continual process of heating and humidifying adjacent molecules. In air, the combination of conduction and convection represents about 15% of the total body heat loss at rest (Guyton & Hall, 1996). However, because the heat conductivity of water is greater than air and water has a specific heat several thousand times greater than that of air, the dissipation of heat is nearly 26 times greater in water when exposed to a similar air temperature (Guyton & Hall, 1996).

1.1.5 Exercise and Thermoregulatory Control

Exercise imposes an internal heat load on the body, which produces an imbalance between the rates of heat loss and heat production and, therefore increases body temperature in almost any environment. This increase in heat production is due mainly, to the increased metabolism caused by muscle activity, by the effect of epinephrine and norepinephrine, and sympathetic stimulation on the cells, and the extra metabolism caused by the increases in chemical activity in the cells (Guyton & Hall, 1996).

Gisolfi and Wenger (1984) describe the path of the heat produced in the muscle during exercise and its effect on heat loss. A small amount of the heat produced in working muscle is conducted passively to the overlying skin; the majority of the remaining heat is transported by the cardiovascular system to the body core. Early in exercise heat production will exceed heat loss and the difference will be stored in the core and the exercising muscle, elevating their temperatures. The increase in these temperatures will be detected by thermoreceptors and a signal will be sent to the POA/AH where effector responses can be stimulated.

Thus it is easy to see that exercise is by far the most significant means of increasing the metabolic rate in the body. Short burst of maximal muscle contraction in any single muscle can liberate as much as 100 times its normal resting amount of heat for a few seconds at a time (Guyton & Hall, 1996). Since survival depends upon keeping nearly all tissue below 45-50°C, and exercise produces much larger than normal amounts of heat, the body must dissipate this excess heat from the tissues to

the environment. The heat loss is accomplished through cutaneous vasodilation and the sweating mechanisms.

1.1.5.1 Control of Skin Blood Flow and the Sweating Response during Exercise

Heat is transported from the body core to the skin via conduction to dissipate the excess heat produced by exercise. The physical laws of conduction indicate that there is a transfer of heat between objects or tissues of different temperatures in direct contact with one another. Thus heat produced in the deep tissues of the body can be transported through adjacent tissues until they reach the skin surface. This method of heat loss is not sufficient in maintaining the internal body temperature; thus a more effective method is utilized.

An increase in blood flow through the cutaneous and muscle vascular beds increases the rate of heat transfer between the skin and the core; the direction of transfer depending on which is at the greater temperature (Hammel, 1968). Control of blood flow to the periphery can readily regulate the flow of heat from the core to the skin, and is the only controllable way to distribute internal heat to the skin (Hammel, 1968). This is accomplished by blood vessels that penetrate the fatty subcutaneous tissue and are distributed profusely immediately beneath the skin (Vander et al, 1994). Important in heat dissipation are the capillaries and the venous plexus, which lie directly below the skin surface. Blood is supplied to the venous plexus by the capillaries and by small arteries through highly muscular arteriovenous anastomoses (Guyton & Hall, 1996). The rate of blood flow into the venous plexus

can range from near zero to as much as 30% of the total cardiac output, thus demonstrating an eight fold increase in the conductance of heat between the fully vasoconstricted state and the fully vasodilated state (Guyton & Hall, 1996). Thus skin blood flow is an effective controlled heat radiator system and flow of blood to the skin is an effective mechanism of heat transfer from the body core to the skin (Gisolfi & Wenger, 1984).

If the skin and core temperatures continue to rise, signals are sent from the POA/AH through the autonomic pathways to the spinal cord and then through the sympathetic outflow to the skin everywhere in the body, which act to initiate the sweating response (Gisolfi & Wenger, 1984). Once sweating occurs the main action of the skin blood flow is to deliver heat to the skin necessary to evaporate the sweat (Hammel, 1968). The sweat gland is made up of two parts: a deep subdermal coiled portion, that secrets the sweat and a duct portion, which passes outward through the dermis and epidermis of the skin (Guyton & Hall, 1996). The cholinergic sympathetic nerve fibers secret acetylcholine that stimulates the glandular cells to secret the sweat (Vander et al., 1994). The sweat then travels through the duct where its concentrations and constituents are modified, before its release to the skin surface (Vander et al., 1994). In addition to cholinergic stimulation, epinephrine and norepinephrine circulating in the blood may also stimulate the sweat glands (Guyton & Hall, 1996). These hormones are released from the adrenal medullae during exercise and thus act to increase sweat secretion.

1.1.5.2 Thermoregulatory Control of Exercise Recovery

Exercise increases body core temperature through increased metabolic rate and as a result skin blood flow, mean skin temperature, oxygen consumption, heart rate and ventilation also increase. Post-exercise elevated values immediately begin to drop off upon the completion of exercise. The various systems of the body work together in an attempt to remain homeostatic. Thus after 10-15 minutes of recovery these values return to their pre-exercise levels, with the exception of the skin temperatures at the sites directly over the exercised muscles, and the esophageal temperature which plateaus at an elevated level of as much as 0.5°C for up to 65 minutes of recovery (Thoden et al., 1994). The mechanisms for this elevated esophageal temperature post-exercise have been proposed to be the result of some exercise-related response which has thermal effects, such as an endocrine response and metabolic by-products, changes in compartmental osmolarity, and baroreflex activity; or the effect of a significant elevation in heat load and whole-body heat content (Kenny et al., 1997b). The mechanism of an increased whole body heat content was explored through comparing the increased esophageal temperature during exercise (endogenous heating) to warm-water immersion (exogenous heating). Kenny et al. (1996) found that within a 10 minute period of exiting the warm-water, the subject's T_{ES} return to its pre-immersion value, thus indicating that the post-exercise increase in T_{ES} does not seem to be the result of increased whole-body heat content alone (Kenny et al., 1996).

Further research in the area of post-exercise thermal homeostasis has revealed that exercise increases the thresholds for both warm and cold effector responses

(Kenny et al., 1997a, 1998). Kenny et al. (1997a, 1998) suggests that the increase in post-exercise thresholds may be due to enhanced vasoconstrictor activity, attenuation of vasodilator activity, or a combination of both of these. The sympathetic active vasoconstrictor and vasodilator systems affect skin blood flow, however it is not clear how skin blood flow is controlled during recovery from exercise. It has been shown that the cutaneous vasodilator system is under baroreceptor control and that exercise causes post-exercise hypotension (Kenny et al., 1998). Kenny et al. (1998) suggests that a decrease in skin blood flow (i.e., vasoconstriction) would help maintain adequate filling pressure in response to the decrease in systemic vascular resistance. Thus this non-thermal factor may influence the elevated threshold for vasoconstriction during exercise recovery (Kenny et al., 1998).

Kenny et al. (1997a) have also demonstrated an increase in the post-exercise threshold for sweating. The parallel increase in both warm and cold effector thresholds is proposed to be the result of either an increase in set-point control with a similar tolerance for core temperature displacement before heat loss or heat gain responses are initiated, or separate but parallel increases in each individual response threshold (Kenny et al., 1998).

1.1.5.3 Hypothermia and Subsequent Exercise

Exercise, due to its ability to increase metabolic rate, has been examined as a means of rewarming a hypothermic individual (Bristow et al., 1994; Giesbrecht & Bristow, 1992, 1998; Romet, 1988). Research indicates that exercise immediately following cold-water immersion increases the post-cooling afterdrop (Giesbrecht &

Bristow, 1998). The afterdrop is associated with a sharp decline in core temperature in the initial stages of warming and is the result of convective heat loss (Romet, 1988; Giesbrecht & Bristow, 1992; Bristow et al., 1994). Romet (1988) states that the afterdrop in T_{CO} is due to peripheral vasodilation leading to the return of cold blood from the peripheral tissue to the central circulation.

Exercise during the initial phases of rewarming has been shown to increase the post-immersion afterdrop by up to five times (Giesbrecht & Bristow, 1998). The increased afterdrop is most likely explained by an exercise-induced increase in peripheral blood flow and thus increased heat loss (Giesbrecht & Bristow, 1998). As well, Giesbrecht and Bristow (1998) indicate that exercise following the post-cooling afterdrop increases the rate of re-warming significantly. Therefore, to gain the benefits of both a decreased afterdrop through shivering thermogenesis, and an increased re-warming rate with exercise, Giesbrecht and Bristow (1998) have suggested allowing the body to re-warm until the afterdrop has passed and then increase the re-warming rate with the onset of exercise.

1.1.5.4 Exercise During Hypothermic Conditions

The heat production from exercise has been shown to offset the heat loss experienced during cold water exposure at various temperatures, thus maintaining thermal homeostasis (McArdle et al., 1984; Craig & Dvorak, 1966). In contrast Veicsteinas et al. (1982) has shown that below a certain level of intensity, exercise may actually increase overall heat loss in cold water due to an increased heat conductance. Others state that the increased cooling rate evident during exercise in

cold water is the result of the poorly insulated, highly perfused active limbs creating an increased heat loss as a result of increased convection (McArdle et al., 1992; Ferretti et al., 1989). Thus it is obvious that the differences in literature indicate that the intensity and duration of the exercise bout, and the temperature of the water influence the core-cooling rate of humans when exercise is introduced during the immersion in a cold-water bath. McArdle et al. (1992) studied 16 volunteers to determine the effect of exercise at various intensities on thermal responses of men and women during cold-water immersion. The subjects were asked to exercise at three different intensities of VO_2, 0.70 l/min, 1.25 l/min, and 1.70 l/min, for one hour immersed in both 28°C and 20°C water. Results for the male subjects immersed in 28°C water indicate that exercising at an intensity of 0.70 l/min compared to rest, showed no significant difference in core cooling rate. A decrease in T_{CO} of 0.79°C occurred in both conditions despite the fact that energy expenditure was essentially doubled during the exercise trial. As well, at an exercise intensity of 1.25 l/min a decrease in T_{CO} of 0.54°C also occurred. However during the final exercise intensity (1.70 l/min), T_{CO} remained essentially unchanged from minute 20 through minute 60, averaging 0.06°C above the pre-immersion value at the completion of the one-hour exercise bout. When immersed in 20°C, T_{CO} began to fall upon immersion under all conditions with no significant difference observed in the final T_{CO} between conditions.

The women subjects, when immersed in 28°C water showed similar effects. At rest T_{CO} decreased by 0.8°C after the 60 minute period. During the exercise intensity of 0.70 l/min the T_{CO} decreased by 0.44°C. In contrast, the exercise

intensities of 1.25 and 1.70 l/min showed no significant decrease in T_{CO} for the one-hour immersion. During the water temperature of 20°C, the women exhibited no significant difference in the change in T_{CO} for the 60 minute immersion period and showed an average decrease of 1.35°C. However, a significantly higher final T_{CO} was noted with exercise at both 1.25 and 1.70 l/min compared to rest and the lowest exercise intensity. Thus these results, as well as previous research (Craig & Dvorak, 1966, 1968; Veicsteinas et al., 1982; McArdle et al., 1984), indicate that as water temperature decreases, a greater level of exercise intensity is needed to achieve the desired effect of decreased cooling; otherwise exercise can actually be detrimental (McArdle et al., 1992).

1.1.5.5 The Influence of Exercise Prior to Hypothermia

The responses of the thermoregulatory system to exercise have been studied extensively (Hammel, 1968; Gisolfi & Wenger, 1984; Johnson, 1992). However the responses of this system to cold water immersion following exercise has not. McDonald et al. (1984) studied five male subjects immersed at two different times in a whole body calorimeter for 60 minutes. The subjects would lay supine immersed to neck level in water at 19°C after either a 30 minute sedentary period or following a 30 minute period of running at 80% of the subject's heart rate maximum. A cooling rate during the exercise trial, of 0.031°C · min^{-1} (~1.86°C · h^{-1}) compared to the non-exercise control trial (0.019°C · min^{-1}; ~1.14°C · h^{-1}) was reported between 10 and 45 minutes of the immersion. Thus the difference between the trials was 0.012°C · min^{-1} (0.72°C · h^{-1}). This indicates a significant increase ($P<0.01$) in the core-cooling rate

when cold-water immersion is followed by an intense dynamic exercise. McDonald et al. (1984) propose that this increased cooling rate may have occurred because of a larger temperature gradient between the body and the water or because of some physiological alteration in the thermoregulatory system. The increased cooling rates found by McDonald et al. (1984) were supported when Castellani et al. (1999) showed that exercise before cold air exposure caused an increased cooling rate compared to that of rest. Ten male subjects participated in 60 minutes of semirecumbent cycling exercise immersed to the neck in 35°C water at an intensity of approximately 55% VO_2 max, or were immersed in 38.2°C water until their rectal temperature matched the rectal temperature at the completion of exercise. These manipulations were spaced one week apart and both were followed by a 120 minute cold air exposure. Cooling rates from minute 10 to the end of the cold air exposure were 0.64°C · h^{-1} for the exercise trial compared to 0.57°C · h^{-1} for the control trail. The differences in cooling rates were shown to be significant ($P<0.05$) thus indicating that prior exercise impairs the ability to maintain thermal balance during whole-body cold-air exposure (Castellani et al., 1999). Castellani et al. (1999) and others (McDonald et al., 1984) suggest that exercise before cold exposure may lead to a greater fall in T_{CO} due to reduced insulation and a redistribution of heat from the core to the periphery. Further, Castellani et al. (1999) states that the data suggests the greater fall in T_{CO} was the result of an exercise-related factor (i.e. heat redistribution) and not the exercise-induced rise in T_{CO}.

This was disputed by Windle et al. (1994) when a cross-sectional methodology compared the effects of warming by active and passive means on the

subsequent responses to cold-water immersion. Twelve healthy volunteers participated in the first experiment, where a 45 minute cold-water immersion (15°C) in a semi-recumbent position followed either 20 minutes of cycle ergometer exercise, which increased deep body temperature by 1°C (or to 38°C), or a control trial in which they rested for an equivalent amount of time seated on the cycle ergometer. The second experiment, which involved 16 different participants, consisted of a warm-water and a thermoneutral immersion. The warm-water immersion (40°C) elevated their deep body temperature by 1°C (or to 38°C) and lasted approximately 20 minutes. The thermoneutral immersion (35°C) followed an equivalent time line. In experiment 1, the rate of fall in rectal temperature during the final 15 minutes of cold-water immersion was significantly ($P<0.01$) faster when the immersion followed the active warming ($2.46°C \cdot h^{-1}$) compared with the control condition ($1.68°C \cdot h^{-1}$) (Windle et al., 1994). However, when comparing the cooling rates of the passive warming and thermoneutral conditions, no significant difference was found in the final 15 minute period. Through this cross-sectional comparison Windle et al. (1994) concluded that a raised deep body temperature due to active or passive warming does not alter short- or long-term survival prospects in cold water. These conflicting results warrant further investigation as to the effects of prior heating (endogenous or exogenous) on the core-cooling rates.

1.2 RESEARCH HYPOTHESES

- This study investigated the hypothesis that owerall-cooling rates during cold-water immersion would be greater following exercise and warm-water immersion compared to control.

- I also hypothesized that the afterdrop and rewarming periods would be unaffected by the cold water immersion following the heating treatments.

1.3 ASSUMPTIONS

- All subjects followed pre-experimental guidelines outlined in the preliminary session including abstinence from stimulants and alcohol, 8-h of sleep and a minimum of 0.25 L of water during each waking hour.

- Subjects were careful to avoid major thermal stimuli or substantial increase of metabolic rate between awakening and the start of the experiment.

- Anthropometric measures remained the same for all experimental trials.

- Room temperature was constant throughout the study.

- Esophageal temperature is an accurate measure of core temperature.

- Increases in oxygen consumption during the cold-water immersion reflect shivering thermogenesis.

1.4 DELIMITATION'S

- All subjects were males, between the ages of 18-35 years and were not engaged in any prolonged or rigorous exercise program.

2.0 EXPERIMENTAL METHODOLOGY

2.1 SUBJECTS

With approval from our Faculty Human Ethics Committee, 7 healthy male volunteers between the ages of 18–35 years with no history of cardiovascular or respiratory disease participated after providing written, informed consent (anthropometric characteristics see Table 1 - appendix 1). The subjects were physically active, although none engaged in daily or intensive training programs. Mean values (± SD) of the subjects age, height, body mass, body surface area, body fat content and maximal oxygen consumption (VO_2 max) were: 24 (6) years, 1.8 (0.1) m, 78.5 (11.5) kg, 2.0 (0.2) m^2, 16.1 (5.5) % and 3.38 (0.77) $l \cdot min^{-1}$.

2.2 INSTRUMENTATION

The esophageal temperature (T_{ES}) was measured as an indication of core temperature. This has been shown to respond rapidly to changes in central blood temperature (Shiraki et al., 1986). T_{ES} was measured with a esophageal thermocouple (Mon-a-therm, Mallinckrodt Medical) inserted through a nostril and swallowed to a depth equal to one-quarter of body height at the level of the left atrium and greater vessels (Mekjavic & Rempel, 1990). T_{ES} from this location resembles the temperature of the blood returning to the heart and is accepted as an accurate measure of core body temperature.

Skin temperature was monitored at 12 sites by waterproofed heat flow sensors (Concept Engineering, Old Saybrook, CT). These were fixed to the skin with surgical tape, and the area-weighted mean was calculated by assigning the following regional

percentages: center of forehead 6%, upper chest 9.5%, upper back 9.5%, biceps 10%, anterior forearm 8%, finger 2%, lower back 9.5%, abdominal 9.5%, anterior thigh 10%, posterior thigh 10%, anterior calf 9% and posterior calf 7%. Both esophageal and skin temperature measurements were collected and digitized (Hewlett Packard data acquisition module, model 3497A) at 10 second intervals, displayed graphically on a computer monitor, and recorded in spreadsheet format on a hard disk (Hewlett Packard, model PC-312, 9000).

Oxygen consumption (VO_2), ventilation (V_E) and respiratory exchange ratio (RER) were collected and analyzed using an automated metabolic cart (Med-Graphics CPX-D, model 790600-001). The automated metabolic cart sampled in 30 second averages for the duration of the testing. Heart rate was measured using a Polar heart rate monitor (Vantage NV). This device consisted of a transmitter chest strap and a receiver, which recorded the data. The data was collected continuously throughout the experiment and was recorded as beats per minute.

2.3 EXPERIMENTAL PROTOCOL

The subjects performed one incremental VO_2 max test on a cycle ergometer and all anthropometric measures were taken on the first day. These data were used to select the workload for the submaximal experimental exercise trial (endogenous heating trial). Subjects were then required to participate in three experimental trials separated by a minimum of 48 hours. The experimental trials were conducted in the morning following a 24-h period without heavy or prolonged physical activity, the last 12-h of which included abstinence from stimulants and alcohol, 8-h of sleep and a

minimum of 0.25 L of water during each waking hour. On each study day care was taken to avoid major thermal stimuli or substantial increase of metabolic rate between awakening and the start of the experiment.

Upon arrival at the laboratory, subjects were instrumented appropriately and clothed in shorts. Baseline data were collected over 30 min at an ambient temperature (T_{AMB}) of 25°C and relative humidity (RH) of 31% with the subject in an upright seated position (see Figure 1). The subjects then received one of three treatments: 1) 15 min upright seated resting (Control); 2) 15 min cycle ergometry at 70% of VO_2max (Exercise); or, 3) 16.8 ± 6.2 min warm-water immersion in a 40°C water bath to the level of the clavicles while in an upright seated position (Warm-Water Immersion).

Following the treatment condition the hands and feet were insulated in thinsulate-insulated socks and mitts, and the subject was immediately immersed up to the clavicles while seated in a semi-recumbent position in a circulated tub of 7°C water until T_{ES} decreased to 34.5°C (~40.2 min). Once T_{ES} reached 34.5°C, subjects were removed from the water, towel dried and seated in an upright position (similar to baseline resting). Subjects were then rewarmed by spontaneous rewarming. Once the subjects T_{ES} returned to near baseline value (~42.1 min), all equipment was disconnected and the subjects were then immersed in a warm water bath (40°C) to ensure that the subject was normothermic.

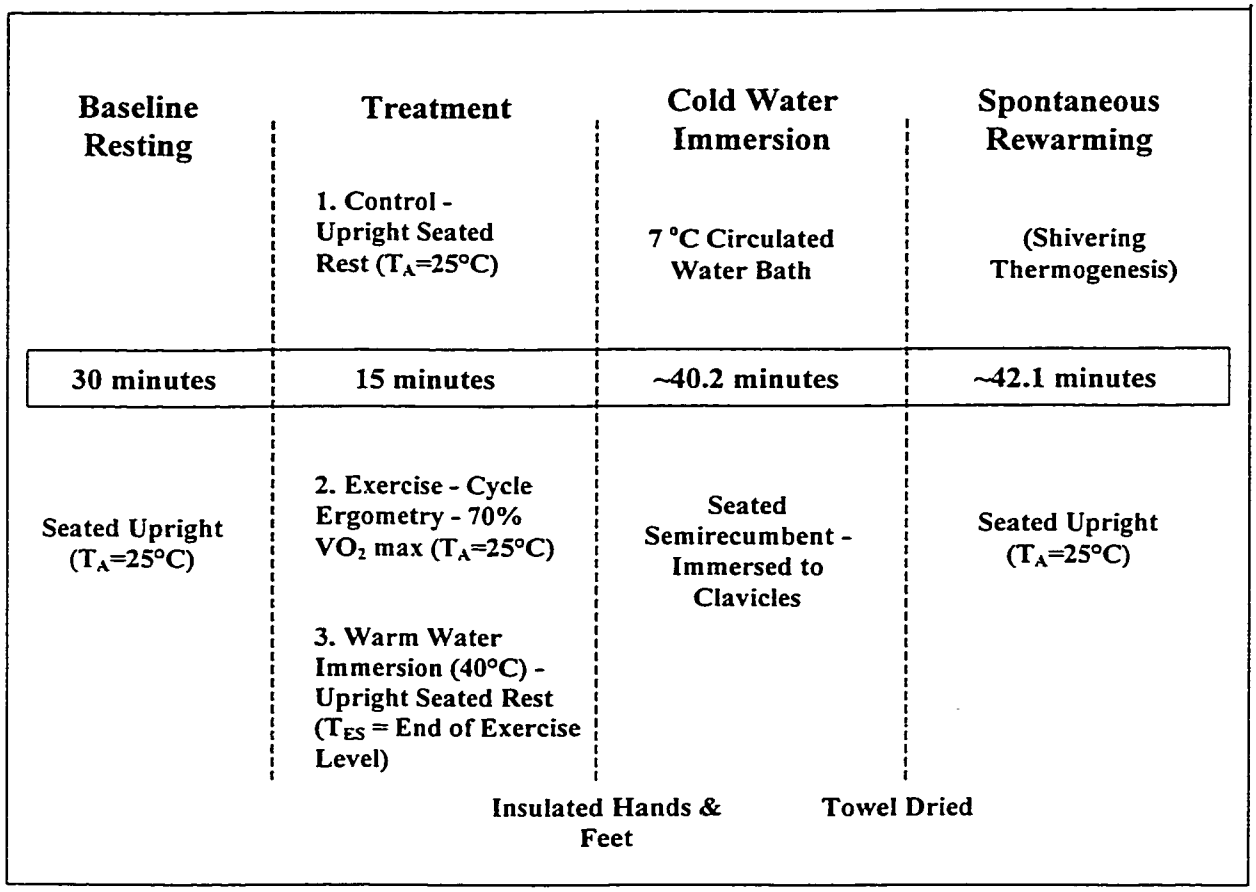

Figure 1: *Experimental Timeline*

2.4 ANALYSIS OF RESULTS

Statistical analyses were performed for the cold-water immersion and subsequent rewarming periods (i.e., from exit of water bath to end of experiment). Using the format described by Giesbrecht et al. (1998), we analysed the cooling rate in five minute intervals during the initial 15 min and final 15 min periods. This methodology enables the researcher to obtain a more detailed thermal response associated with the cold water exposure that is normally not possible when cooling

rates are measured as an overall average calculated over the full period of immersion. The initial 15 min period describes the immediate response, under normothermic conditions, associated with sudden cold exposure (i.e., cold water immersion at 7°C). The final 15 minutes provides information regarding the cold-induced adaptive response, under hypothermic conditions, over the prolonged immersion period.

The following variables were calculated for each trial: 1) the overall cooling rate – the average of T_{ES} data at five minute intervals was calculated by linear regression during the entire cold water immersion period. Overall cooling rate was represented by the average cooling rate calculated for each five minute period; 2) initial cooling rate – calculated in the same way as overall cooling rate except the time period was limited to the initial 15 min of immersion; 3) final cooling rate - calculated in the same way as overall cooling rate except the time period was limited to the final 15 min of immersion; 4) afterdrop – the afterdrop is calculated as the difference between T_{ES} on exit from cold water and its nadir; 5) afterdrop length - represents the time between exit from cold water until T_{ES} returned to original water bath exit T_{ES} (usually 34.5°C); 6) afterdrop nadir – the afterdrop nadir was calculated as the lowest point in T_{ES} during the afterdrop period); 7) afterdrop cooling rate - calculated by linear regression using the T_{ES} data from exit to the nadir; 8) final rewarming rate - calculated by linear regression from T_{ES} for the final fifteen minutes of the rewarming period; 9) overall rewarming rate - calculated by linear regression using T_{ES} measured at five minute intervals during the linear increase following the T_{ES} nadir.

Oxygen consumption was used as an indicator of metabolic heat production above baseline resting. An elevation in oxygen consumption during both cold-water immersion and post-immersion rewarming was attributed to shivering thermogenesis (Bristow et al., 1988). Data for the three trials were compared using an ANOVA for repeated measures with Tukey's post hoc test used to identify significant differences. Results are reported as means ± SD, with the actual α level indicated when $P<0.05$.

3.0 RESULTS

All subjects completed 30 minutes of upright-seated rest in an ambient temperature of 25°C and a relative humidity of 31%, to obtain steady state resting baseline values. Thus pre-treatment esophageal temperatures (T_{ES}) for all conditions were similar and stable prior to the treatment condition (Table 2). Both exercise and warm water heating resulted in a significant elevation of core temperature of 0.7 and 0.8°C, respectively, compared to the resting control (P=0.0001). Core temperature at pre-immersion was similar for both the Exercise and Warm-water immersion conditions (37.8°C) while the pre-immersion T_{ES} for Control was 37.0°C.

The mean cold-water immersion time for each of the treatment conditions were 50.3, 48.6, and 21.8 min for Control, Exercise and Warm-water immersion, respectively. Warm-water immersion prior to cold-water exposure resulted in an overall cooling rate (OCR, Table 3) that was 2.5 and 3.3 times greater than Exercise (P=0.008) and Control (P=0.004), respectively. In contrast, only a 1.3-fold increase in overall cooling rate was measured between Exercise and Control (P=0.009) (see figure 2).

The largest difference in cooling rate occurred during the initial 15 minutes of the cold-water immersion (CR_I-15, Table 3) for Exercise and Warm-water immersion compared to Control. Exercise and Warm-water immersion prior to cold-water immersion resulted in an initial cooling rate 2 and 4.9 times greater than Control (P=0.0001). Furthermore, Warm-water immersion was 2.4 times greater than Exercise (P=0.002).

With respect to the cooling rate in the final fifteen minutes of cold-water exposure (CR_F-15, Table 3), no significant difference was measured between Exercise and Control (see Table 3). However, the final cooling rate for Warm-water immersion remained 2.2- and 2.4-fold greater than Exercise and Control, respectively (P=0.04).

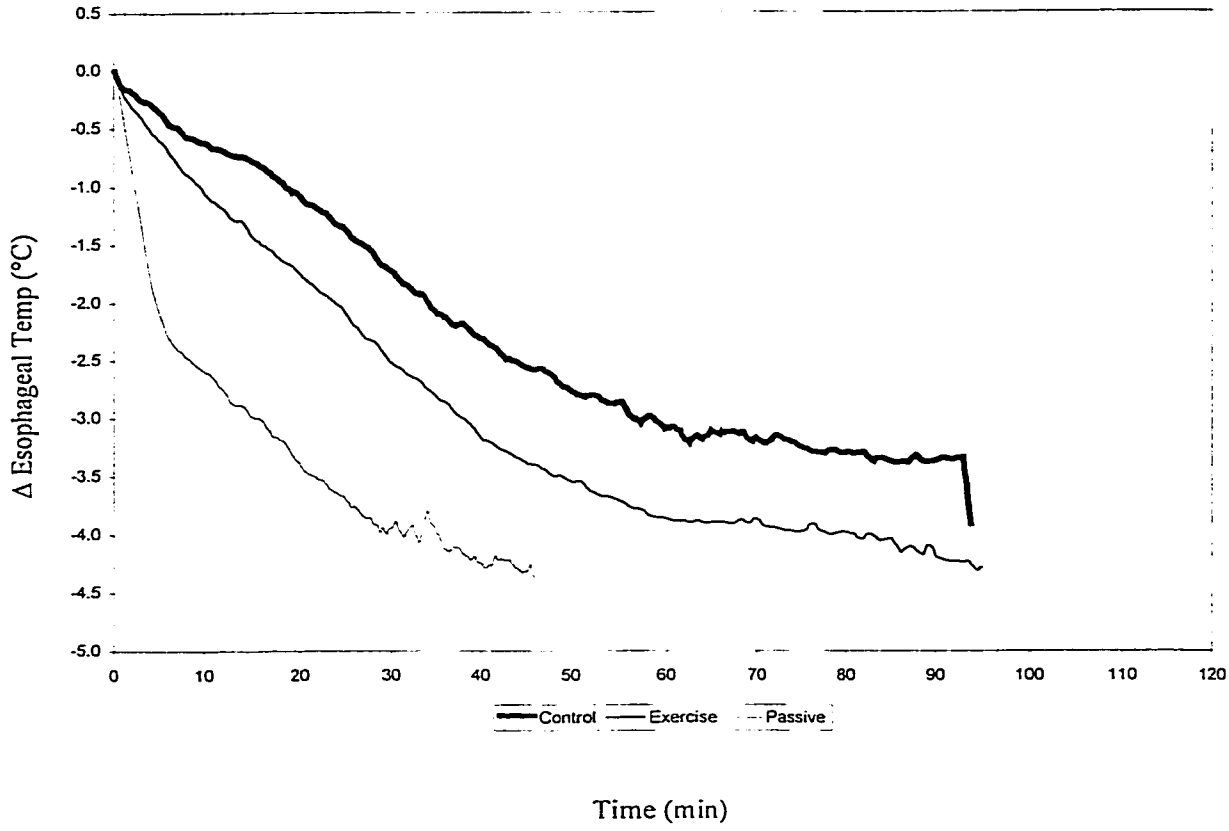

Figure 2: Mean change in esophageal temperature (Δ Esophageal Temp °C) during the entire cooling period for three treatment conditions (n=7). Time and T_{ES} = 0 are the beginning of cooling period.

Upon removal from the cold-water bath, T_{ES} continued to fall. The Control condition resulted in the largest afterdrop-cooling rate (ADCR, Table 3) followed by Exercise. Warm-water immersion demonstrated the lowest afterdrop-cooling rate of all conditions. Although there was a tendency for the afterdrop-cooling rate to increase immediately upon removal from the cold, the differences between treatments were not significant (Table 3). A similar response pattern to that of the afterdrop-cooling rate was observed in the difference between final cooling rate and afterdrop cooling rate (ΔCR) (Table 3). However, the ΔCR for Control (P=0.047) and Exercise were 2.4- and 1.6-fold greater than Warm-water immersion condition.

Treatment	Total Immersion Time (min)	Baseline T_{ES} (°C)	Pre-Immersion T_{ES} (°C)	Difference T_{ES} (°C)	Exit T_{ES} (°C)
Control	**50.3**	**37.1**	**37.0**	**-0.1**	**34.6**
	(25.6)	(0.3)	(0.3)	(0.1)	(0.2)
Exercise	**48.6**	**37.1**	**37.8**	**0.7♣**	**34.5**
	(24.5)	(0.2)	(0.2)	(0.2)	(0.0)
Warm-Water Immersion	**21.8♠**	**37.0**	**37.8**	**0.8♣**	**34.5**
	(12.7)	(0.2)	(0.1)	(0.1)	(0.0)

TABLE 2: MEAN (± SD) ESOPHAGEAL TEMPERATURE DYNAMICS DURING COOLING FOR THREE TREATMENTS (N = 7).
Treatments: Resting control; Cycling exercise initiated immediately following resting baseline; Immersion in warm water immediately following resting baseline. ♣ Significantly different from Control. ♠ Significantly different from Exercise and Control.

The afterdrop (AD), T_{ES} nadir and afterdrop length (ADL) in all conditions were similar, showing no significant difference (Table 4). The total mean rewarming time for each of the conditions were 43.5, 41.4 and 41.4 min for Control, Exercise

and Warm-water immersion, respectively. As well, the overall-rewarming rate (ORR) and final rewarming rate (RR_F-15) were almost identical for all conditions.

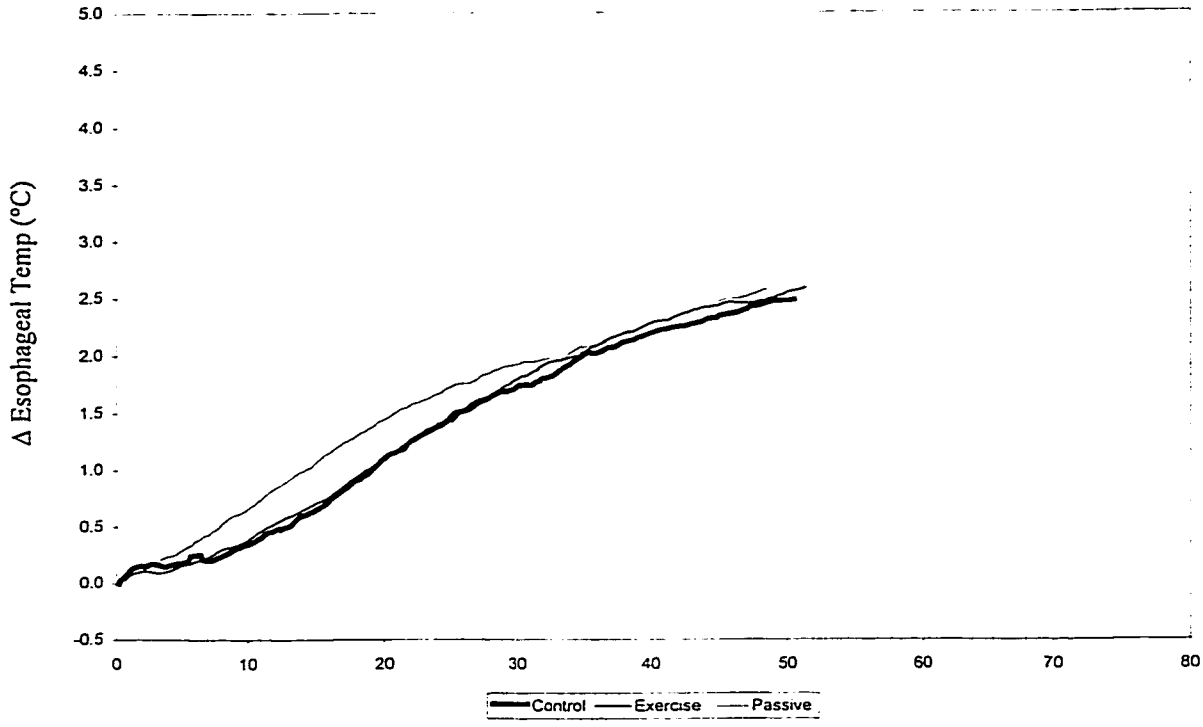

Figure 3: Mean change in esophageal temperature (Δ Esophageal Temp °C) during the entire rewarming period for three treatment conditions (n=7). Time and T_{ES} = 0 are the beginning of rewarming period.

Initially upon immersion, oxygen consumption decreased slightly during the initial 5 minutes (n.s.) followed a progressive sustained increase above baseline resting for the duration of the cold-water immersion (P=0.0001). A similar response was demonstrated for all treatment conditions. The mean oxygen consumption, as

measured over the full period of immersion were similar (i.e., 0.9, 0.9 and 0.8 $L \cdot min^{-1}$ for the Control, Exercise and Warm-water immersion, respectively).

Treatment	OCR ($°C \cdot h^{-1}$)	CR_I-15 ($°C \cdot h^{-1}$)	CR_F-15 ($°C \cdot h^{-1}$)	ADCR ($°C \cdot h^{-1}$)	Δ CR ($°C \cdot h^{-1}$)
Control	3.8	2.7	4.2	12.1	7.9
	(2.2)	(2.5)	(3.3)	(14.2)	(16.4)
Exercise	5.0*	5.4*	4.7	10.0	5.3
	(2.6)	(2.4)	(2.8)	(8.7)	(9.3)
Warm-Water Immersion	12.4*▲	13.2*▲	10.2*▲	8.7	3.3*▲
	(6.8)	(5.9)	(9.6)	(5.0)	(12.4)

TABLE 3. MEAN (± SD) ESOPHAGEAL TEMPERATURE COOLING RATES FOR THREE TREATMENTS (N = 7).
Abbreviations: OCR, cooling rate for the entire cold immersion period; CR_I-15, cooling rate for the initial 15-minutes of the cold water immersion; CR_F-15, cooling rate for the final 15-minutes of cold water immersion; ADCR, cooling rate from the exit of the cold to reaching T_{ES} nadir; Δ CR, change in cooling rate (ADCR – CR_F-15). * Significantly different from Control. ▲ Significantly different from Exercise and Control.

Treatment	Total Rewarming Time (min)	Exit T_{ES} (°C)	T_{ES} (°C) Nadir	AD (°C)	ADL (min)	ORR ($°C \cdot h^{-1}$)	RR_F-15 ($°C \cdot h^{-1}$)
Control	43.5	34.6	34.1	0.5	15.6	3.0	2.7
	(4.8)	(0.2)	(0.2)	(0.2)	(6.2)	(0.7)	(0.9)
Exercise	41.4	34.5	34.0	0.4	14.8	3.1	2.8
	(6.8)	(0.0)	(0.2)	(0.2)	(5.5)	(0.8)	(1.3)
Warm-Water Immersion	41.4	34.5	33.9	0.6	13.9	3.3	2.3
	(7.3)	(0.0)	(0.4)	(0.4)	(6.1)	(0.5)	(0.6)

TABLE 4. MEAN (± SD) ESOPHAGEAL TEMPERATURE DYNAMICS DURING AFTERDROP & REWARMING FOR THREE TREATMENTS (N = 7).
Abbreviations: AD, afterdrop; ADL, length of afterdrop period; ORR, rate of rewarming for the entire rewarming period; RR_F-15, rate of rewarming for the final 15-minute period.

4.0 DISCUSSION

This is the first study to investigate how an increase in core temperature induced by exercise and warm water immersion prior to cold-water exposure affects core cooling rate, afterdrop and subsequent rewarming in humans. Exercise (P=0.008) and Warm-water immersion (P=0.004) increased the overall core-cooling rate, and therefore the onset of hypothermia, by as much as 1.3- and 3.3-times, respectively, as compared to Control. Our Exercise results are comparable to the findings of McDonald et al. (1984) of a 1.6-fold increase. However, McDonald et al. (1984) did not include a warm-water immersion condition, thus a comparison cannot be made. Other studies conducted by Windle et al. (1994) and Castellani et al. (1999) either did not provide overall cooling rates or did not include a resting control. Furthermore, the following study is the only known research conducted that has evaluated core cooling rates in severely hypothermic individuals (i.e., final core temperature of 34.5°C and mean decrease in core temperature of 2.6°C below baseline resting). Previous studies were limited to a core temperature decrease of ~0.8°C or an exit temperature of 36.3°C which is considered normothermic and at best, slightly hypothermic.

To provide a more detailed analysis, this study evaluated the cooling rates in five minute intervals throughout the entire cooling period. These results indicated that among the three conditions, the largest difference in cooling rate occurred in the initial fifteen minutes of the immersion. Similar patterns in the cooling rates were discussed by Windle et al. (1994) that demonstrated the greatest cooling rate occurred in the initial fifteen minutes, with warm-water immersion (water temperature of

40°C) demonstrating the greatest rate of cooling. This was comparable to our findings, however, the cold-water immersion period was limited to an exit core temperature of ~36.1°C in the study by Windle et al. (1994), making actual comparisons difficult. In fact, a core temperature of 36.1°C is still considered normothermic. Furthermore, Windle et al. (1994) hypothesized that if cooling continued the cooling rates of both the exercise and warm water treatment conditions would converge and then fall at a similar rate to that of the resting control. However, our study contradicts his findings as our cooling rates following Warm-water immersion remained significantly elevated above both the Exercise and Control conditions for the entire cooling period (P=0.04).

To our knowledge, no previous studies have examined the subsequent afterdrop and rewarming periods following cold-water immersion with prior exercise and warm-water heating treatments. In all conditions, no significant difference was measured in the afterdrop in T_{ES} upon removal from the cold water. The mean afterdrop for each of the conditions was 0.5, 0.4 and 0.6°C for the Control, Exercise and Warm-water immersion treatment conditions, respectively. T_{ES} to nadir in each of the conditions followed an inverse pattern to that of the overall cooling rates in that the higher the overall cooling rate the lower the T_{ES} nadir. Although T_{ES} nadir exhibited this pattern, no significant difference was measured between the treatment conditions. As well, a pattern emerged between the afterdrop length and the T_{ES} nadir, in that the lower the T_{ES} nadir, the shorter the duration of the afterdrop. However, this also showed no significant difference between the conditions. Thus, our data demonstrated that prior heating by means of Exercise and Warm-water

immersion do not increase rewarming rates above Control values in severely hypothermic individuals.

Oxygen consumption values remained similar between all treatment conditions. McDonald et al. (1984), Windle et al. (1994) and Castellani et al. (1999) support these results in that no significant difference in oxygen consumption was measured between any of the treatment conditions.

4.1 POSSIBLE MECHANISMS FOR RESULTS

It is likely that the increased overall core-cooling rate was caused, in part, by the larger temperature gradient between body core and shell, and the shell and water, thus increasing heat loss via conduction and convection. The Newtonian cooling equation predicts that objects cool at a rate that is proportional to the temperature difference between the object and its surroundings (McDonald et al, 1984). However, if the increased core-cooling rate were caused entirely by the increased temperature gradient, cooling rates for both the Exercise and Warm-water treatments would be very similar. The data provide further evidence to demonstrate a possible alteration in the heat conservation mechanisms of the thermoregulatory system (Kenny et al., 1998).

To examine the importance of the temperature gradient, it is necessary to equilibrate the temperature gradients when comparing the experimental trials. To duplicate the change in T_{ES} between baseline and the T_{ES} at the end of exercise, immersion in warm water (40°C) was utilized. In the study conducted by Castellani et al. (1999), following the exercise and the warm-water heating treatments, subjects

were exposed to a ~20 minute transition period in an ambient temperature of 22.8°C prior to the cold air exposure (4°C). Kenny et al. (1996) have demonstrated that the increased esophageal temperature following warm-water immersion may return to its pre-immersion baseline value within ten minutes of exiting the warm-water bath. Thus, if it is important to equilibrate the temperature gradients to compare to experimental trails, immediate cold exposure is necessary following the warm-water heating condition. This would account for the significant differences in pre-immersion T_{ES} between the exercise and the warm-water treatment condition in the study by Castellani et al (1999). Therefore it is probable that the initial period of cooling in the warm-water condition of Castellani et al. (1999) was missed. This may explain why their cooling rate results for the warm-water treatment condition differ from those of this study and Windle et al. (1994).

Cooling rate could increase if the prior heating blunted the drive for vasoconstriction normally elicited in response to cold, as suggested by Castellani et al. (1999). However, cold-induced vasoconstriction is mediated sympathetically and locally by norepinephrine secretion in response to a peripheral cold stimulus. The sympathetic system responds to the cold stimulus almost instantaneously at the level of the skin. Thus, if a delay in the vasoconstriction response did occur, it is not likely that it could be responsible for the large amounts of heat loss associated with these increases in cooling rate. Further, although plasma norepinephrine concentrations were not measured in this particular study, Castellani et al. (1999) indicates that no measurable differences in norepinephrine concentrations were observed between exercise and warm-water heating. Thus, it is unlikely that a change in the

vasoconstrictor activity in response to the cold stimulus (i.e. cold water immersion) could account for the differences in cooling rates between the three conditions.

It is known that a redistribution of blood flow occurs as a result of exercise and warm water immersion. The exercise-induced hyperemia, which results in up to a fifteen-fold increase in skeletal muscle blood flow, would have a significant effect on the total body insulation (Fox et al., 1988). It has been suggested that 70-90% of total body insulation is provided by poorly perfused muscle in resting individuals immersed in cold water (Veicsteinas et al., 1982). Thus, the exercise-induced hyperemia may significantly reduce the insulative effect of the muscle, thereby increasing the peripheral heat loss from the skin by mass flow (Windle et al., 1994). This may continue into the cooling period, which would result in an increased cooling via convective heat transfer from the core to the periphery overlying active musculature, explaining the increased overall cooling rates between the Exercise and Control condition. As well, warm-water immersion increases blood flow largely to the cutaneous vasculature. The relatively large skin versus muscle blood flow following warm-water immersion may distribute heat above the insulation of the subcutaneous fat to the superficial shell. This allows for greater heat loss at the skin surface via convection, possibly explaining the greater cooling rates of the Warm-water immersion treatment compared to Exercise and Control conditions.

Shivering thermogenesis is an important mechanism in maintaining or increasing body temperature when exposed to extreme cold. It is capable of increasing heat production by up to four to five times above basal metabolic rate. Therefore, it is plausible that the increase in cooling rate is a result of a decreased

effectiveness of the shivering thermogenic response (i.e., spontaneous rewarming utilized in this study). However, results suggest that shivering intensity was similar regardless of the prior treatment based on similarities in oxygen consumption values. McDonald et al. (1984), Windle et al. (1994) and Castellani et al. (1999) support these results in that shivering intensity, measured by oxygen consumption was virtually unchanged between all treatment conditions.

5.0 CONCLUSIONS

Therefore based on the results obtained the increased core-cooling rate that was evident when exercise and warm-water heating precede cold-water immersion was the result of both an increased temperature gradient and blood flow/heat redistribution. We conclude that the increased rates of cooling caused by prior heating will alter survival time in subjects exposed to cold-water immersion due to the significantly increased cooling rates following both exercise and warm-water immersion.

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APPENDIX 1

Anthropometric Characteristics of Subjects

Subject	Age (yr.)	Height (m)	Weight (kg)	Body Surface Area (m^2)	Sum of 7 Skinfolds (mm)$^{\nabla}$	Body Fat (%) - Skinfolds$^{\#}$	Body Fat (%) – Hydrostatic Weighing^{0}	VO_2 Max. ($l \cdot min^{-1}$)
1	24	1.78	83.0	2.04	85.1	12.1	14.7	4.43
2	25	1.74	83.0	2.02	96.3	13.6	15.3	4.20
3	20	1.88	81.5	2.07	62.3	8.4	9.3	3.47
4	34	1.75	97.8	2.20	196.6	27.0	26.2	3.60
5	20	1.76	65.6	1.80	57.6	7.8	12.7	2.60
6	19	1.86	73.0	1.94	75.2	10.2	14.0	3.01
7	29	1.75	65.5	1.79	123.0	17.6	20.2	2.37
Mean	**24.4**	**1.79**	**78.5**	**1.98**	**99.4**	**13.8**	**16.1**	**3.38**
SD	**5.5**	**0.06**	**11.5**	**0.15**	**48.2**	**6.7**	**5.5**	**0.77**

TABLE 1. ANTHROPOMETRIC CHARACTERISTICS OF SUBJECTS.

Calculated according to Gehan and George (1970).

$^{\nabla}$ 7 skin fold sites: chest, axilla, triceps, subscapular, abdominal, suprailiac and front thigh.

$^{\#}$ Calculated according to the Jackson and Pollock Method.

^{0} Calculated according to the Siri equation.

APPENDIX 2

Subject Rates Tables

Control-Averages

Time (min)	AP0601	CS0329	DS0108	GK0413	MS0528	MT0107	PS0726
Cooling 0-5 min	-1.662	-5.640	-5.958	-1.680	-2.322	-6.372	-0.882
Cooling 5-10 min	-1.896	-4.254	-10.542	-0.018	-0.888	-2.808	-2.280
Cooling 10-15 min	-0.606	-0.174	-7.128	-0.030	-0.180	-2.826	-3.918
Cooling 15-20 min	-1.872	-3.420		-4.578	-3.546	-3.096	-6.186
Cooling 20-25 min	-2.094	-3.090		-0.570	-4.536	-3.606	-6.438
Cooling 25-30 min	-2.472	-2.694		-2.424	-4.296	-6.540	-7.086
Cooling 30-35 min	-2.202	-2.232		-4.428	-3.750	-4.236	-6.696
Cooling 35-40 min	-0.618	-2.388		-2.760	-1.878	-6.234	
Cooling 40-45 min	-2.370	-3.048		-2.796	-4.038		
Cooling 45-50 min	-2.178	-2.532		-2.628	-3.246		
Cooling 50-55 min	-0.714	-1.188		-1.926			
Cooling 55-60 min	-1.236	-1.122		-3.660			
Cooling 60-65 min	-1.194	-0.222					
Cooling 65-70 min	-0.792						
Cooling 70-75 min	-0.342						
Cooling 75-80 min	-0.318						
Cooling 80-85 min	-1.056						
Cooling 85-90 min	0.150						
Cooling 90-95 min	-5.748						
Final 15 min Average	**-0.312**	**-1.884**	**-10.026**	**-2.664**	**-3.048**	**-5.172**	**-6.534**
Exit to Nadir	**-41.802**	**-18.156**	**-8.772**	**-7.368**	**-4.422**	**-1.29**	**-2.928**
Overall Average	**-1.538**	**-2.462**	**-7.876**	**-2.292**	**-2.868**	**-4.465**	**-4.784**
Cooling Rate (In-Out)	***-1.228***	***-2.517***	***-9.766***	***-2.551***	***-2.842***	***-4.454***	***-4.922***
Rewarming 0-5 min	0.750	3.582	2.010	0.270	0.312	2.022	1.572
Rewarming 5-10 min	-0.702	2.010	2.850	-1.548	2.106	3.456	3.342
Rewarming 10-15 min	1.104	3.438	3.852	2.022	5.628	4.410	4.476
Rewarming 15-20 min	6.504	3.810	4.620	3.492	6.300	6.096	6.840
Rewarming 20-25 min	4.314	3.282	4.512	3.360	5.802	3.786	5.124
Rewarming 25-30 min	2.238	0.936	2.130	1.944	5.172	3.972	5.244
Rewarming 30-35 min	6.810	3.684	1.938	1.938	3.090	2.742	3.984
Rewarming 35-40 min	4.920	1.596	1.740	2.748	1.356	1.806	2.508
Rewarming 40-45 min		1.854	1.110	1.914		1.062	2.088
Rewarming 45-50 min		1.362		1.584			
Rewarming 50-55 min							
Rewarming 55-60 min							
Rewarming 60-65 min							
Final 15 min Average	**3.894**	**1.854**	**2.244**	**1.932**	**3.798**	**1.884**	**3.132**
Overall Average	**3.242**	**2.555**	**2.751**	**1.772**	**3.721**	**3.261**	**3.909**
Rewarming Rate (Out)	***3.467***	***2.447***	***3.061***	***2.050***	***4.082***	***3.332***	***3.877***

Exercise-Averages

Time (min)	AP0526	CS0318	DS0118	GK0513	MS0517	MT0116	PS0804
Cooling 0-5 min	-6.294	-8.448	-10.374	-1.392	-5.676	-8.700	-4.416
Cooling 5-10 min	-4.002	-5.796	-11.598	-2.106	-4.938	-6.744	-5.724
Cooling 10-15 min	-1.830	-3.306	-6.570	-3.480	-3.774	-4.800	-5.742
Cooling 15-20 min	-3.378	-2.724	-10.326	-1.404	-2.010	-3.444	-7.008
Cooling 20-25 min	-3.408	-3.102		-3.066	-3.312	-4.674	-6.270
Cooling 25-30 min	-3.516	-3.042		-3.714	-5.748	-4.260	-8.706
Cooling 30-35 min	-2.160	0.204		-4.386	-4.434	-4.998	-8.226
Cooling 35-40 min	-4.524	-1.938		-2.772	-5.910	-4.866	
Cooling 40-45 min	-0.912	-3.216		-1.986	-6.186	-9.096	
Cooling 45-50 min	-3.072	-2.838		-0.216			
Cooling 50-55 min	-2.838	-2.730		-1.266			
Cooling 55-60 min	-3.276	-7.650		-0.936			
Cooling 60-65 min				-0.270			
Cooling 65-70 min				0.270			
Cooling 70-75 min				-0.954			
Cooling 75-80 min				-0.732			
Cooling 80-85 min				-0.618			
Cooling 85-90 min				-0.690			
Cooling 90-95 min				-1.116			
Final 15 min Average	-2.550	-2.976	-8.844	-1.242	-5.286	-4.698	-7.614
Exit to Nadir	-25.482	-1.242	-17.466	-11.292	-5.424	-3.69	-5.592
Overall Average	-3.268	-3.716	-9.717	-1.623	-4.665	-5.731	-6.585
Cooling Rate (In-Out)	*-3.383*	*-3.611*	*-10.053*	*-1.743*	*-5.049*	*-5.092*	*-6.571*
Rewarming 0-5 min	3.822	0.762	2.880	0.252	0.522	0.846	0.102
Rewarming 5-10 min	2.358	3.216	0.924	1.962	3.906	3.534	3.744
Rewarming 10-15 min	3.942	2.994	2.490	2.106	5.436	3.930	4.440
Rewarming 15-20 min	4.098	2.892	5.310	3.828	6.228	6.438	5.646
Rewarming 20-25 min	3.396	2.436	3.762	4.176	4.998	5.478	4.938
Rewarming 25-30 min	5.316	1.914	4.044	2.886	4.818	4.560	3.912
Rewarming 30-35 min	4.896	0.174	3.102	1.644	2.598	1.884	3.126
Rewarming 35-40 min			2.712	2.166	3.222	1.824	3.984
Rewarming 40-45 min				1.056		2.046	2.802
Rewarming 45-50 min				1.002			1.062
Rewarming 50-55 min							1.644
Rewarming 55-60 min							
Rewarming 60-65 min							
Final 15 min Average	4.140	1.842	4.104	1.296	4.212	1.770	2.112
Overall Average	3.975	2.055	3.153	2.108	3.966	3.393	3.218
Rewarming Rate (Out)	*3.718*	*2.470*	*3.409*	*2.242*	*4.326*	*3.490*	*3.365*

Passive-Averages

Time (min)	AP0406	CS0323	DS0208	GK0322	MS0307	MT0209	PS0212
Cooling 0-5 min	-30.822	-28.398	-27.378	-32.712	-11.754	-28.554	-28.230
Cooling 5-10 min	0.804	-9.966	-22.236	-2.262	-6.246	-6.414	-6.636
Cooling 10-15 min	-3.864	-8.268		-1.182	-4.578	-3.594	-10.362
Cooling 15-20 min	-3.342			-2.730	-7.572	-6.960	
Cooling 20-25 min	-1.602			-4.014	-4.380		
Cooling 25-30 min	-1.908				-5.088		
Cooling 30-35 min	0.516						
Cooling 35-40 min	-2.388						
Cooling 40-45 min	-0.678						
Cooling 45-50 min	-4.902						
Cooling 50-55 min							
Cooling 55-60 min							
Cooling 60-65 min							
Cooling 65-70 min							
Cooling 70-75 min							
Cooling 75-80 min							
Cooling 80-85 min							
Cooling 85-90 min							
Final 15 min Average	**-1.698**	**-14.124**	**-29.082**	**-2.322**	**-6.468**	**-4.740**	**-12.852**
Exit to Nadir	**-17.328**	**-9.786**	**-3.198**	**-5.694**	**-3.588**	**-11.118**	**-9.906**
Overall Average	**-4.819**	**-15.544**	**-24.807**	**-8.580**	**-6.603**	**-11.381**	**-15.076**
Cooling Rate (In-Out)	***-4.241***	***-14.887***	***-28.113***	***-8.450***	***-7.158***	***-10.484***	***-14.610***
Rewarming 0-5 min	0.654	2.928	6.036	4.584	1.878	3.246	5.946
Rewarming 5-10 min	5.568	4.110	4.242	5.442	2.946	5.370	4.506
Rewarming 10-15 min	4.158	4.152	3.822	4.524	4.254	6.870	4.818
Rewarming 15-20 min	5.916	2.094	3.318	3.288	3.894	6.420	6.078
Rewarming 20-25 min	3.150	3.600	0.936	2.406	5.046	3.312	4.224
Rewarming 25-30 min	3.642	2.310	1.134	0.924	4.200	1.056	3.558
Rewarming 30-35 min	2.388	0.762		1.866	4.440	-1.704	2.322
Rewarming 35-40 min	2.694	2.514		0.582	3.486	2.736	1.638
Rewarming 40-45 min	1.818	1.968			4.272	1.266	1.698
Rewarming 45-50 min		0.888				2.136	
Rewarming 50-55 min							
Rewarming 55-60 min							
Rewarming 60-65 min							
Rewarming 65-70 min							
Final 15 min Average	**2.316**	**1.812**	**2.586**	**1.398**	**3.408**	**2.088**	**2.208**
Overall Average	**3.332**	**2.533**	**3.248**	**2.952**	**3.824**	**3.071**	**3.865**
Rewarming Rate (Out)	***3.431***	***2.359***	***3.599***	***3.125***	***3.723***	***3.331***	***4.094***