# THE THERMOPHYSIOLOGY OF UNCOMPENSABLE HEAT STRESS: INFLUENCE OF HYDRATION STATUS, FLUID REPLACEMENT, AEROBIC TRAINING, PHYSICAL FITNESS, AND HEAT ACCLIMATION

by

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# Abstract

The thermophysiology of uncompensable heat stress: influence of hydration status, fluid replacement, aerobic training, physical fitness, and heat acclimation

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The present research was developed to investigate the separate and interactive influences of hydration status, fluid replacement, short-term aerobic training, heat acclimation, and aerobic fitness during exercise in the heat. The research focused on an uncompensable heat stress environment, where the capacity for evaporative heat loss is less than the amount required to maintain thermal steady state. This was produced by either light (3.5 $km \cdot h^{-1}$, 0% grade, no wind; Studies 1-3) or heavy (4.8 $km \cdot h^{-1}$, 4% grade, no wind; Study 1) exercise in the heat (40°C, 30% relative humidity) while wearing a multilayered clothing ensemble with a high insulative capacity and a low water vapour permeability. Study 1 investigated the influences of hydration status (euhydration versus hypohydration of 2.5% body mass), fluid replacement, and exercise intensity. Study 2 investigated the influences of short-term aerobic training and hydration status on unfit (LF) individuals. Study 3 investigated the influences of heat acclimation and hydration status on individuals of both LF and high fitness (HF). Hypohydration resulted in

significant increases in cardiovascular and thermal strain and a decrease in exercise tolerance regardless of other factors. Fluid replacement decreased heart rate ($f_c$) and increased tolerance time (TT) during light exercise. In LF, two weeks of aerobic training or heat acclimation increased sweat rate (SR), but did not influence $f_c$, rectal temperature ($T_{re}$), or TT. In HF, two weeks of heat acclimation increased SR, decreased $\overline{T}_{sk}$ and $T_{re}$, but did not influence $f_c$ or TT. Neither training nor heat acclimation in LF was able to increase TT to a level similar to that in HF before undergoing heat acclimation. A greater $\Delta T_{re}$ over the course of the exercise was present in HF, through a combination of both a lower initial, and a higher endpoint, $T_{re}$. The higher endpoint $T_{re}$ in HF was consistent regardless of hydration, training, or acclimation status. It is concluded that even mild hypohydration can significantly impair physiological responses and tolerance to exercise in an uncompensable heat stress environment, and that high aerobic fitness and habitual activity imparts a degree of protection that cannot be replicated by either short-term training or heat acclimation.

*for Debbie*

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Cheung SS, McLellan TM (1998) Influence of hydration status and fluid replacement on heat tolerance while wearing NBC protective clothing. Eur J Appl Physiol 77: 139-148

Cheung SS, McLellan TM (in press) Influence of hydration status and short-term aerobic training on tolerance during uncompensable heat stress. Eur J Appl Physiol

Cheung SS, McLellan TM (in press) Influence of heat acclimation, aerobic fitness, and hydration status on tolerance during uncompensable heat stress. J Appl Physiol

Cheung SS, McLellan TM (submitted) Comparison of short-term aerobic training and high maximal aerobic power on tolerance to uncompensable heat stress. Aviat Space Envt Med

Cheung SS, McLellan TM (submitted) Relative influence of aerobic fitness and individual characteristics on responses to uncompensable heat stress. Eur J Appl Physiol

## List of Abbreviations

| | |
|---|---|
| Acc | Heat acclimation treatment |
| $A_D$ | Dubois body surface area ($m^2$) |
| BV | Blood volume (L) |
| $c_{pa}$ | The heat capacity of dry air (0.28 $W \cdot h \cdot kg^{-1} \cdot {}^{o}C^{-1}$) |
| $c_{pwv}$ | The heat capacity of water vapour (0.52 $W \cdot h \cdot kg^{-1} \cdot {}^{o}C^{-1}$) |
| $\dot{C}$ | Rate of convective heat exchange ($W \cdot m^{-2}$) |
| $\dot{C}_{resp}$ | Rate of respiratory convective heat exchange ($W \cdot m^{-2}$) |
| $(dZ \cdot dt^{-1})_m$ | Maximal rate of change of impedance ($\Omega \cdot s^{-1}$) |
| ECF | Extracellular fluid |
| EU | Euhydrated condition |
| $\dot{E}_{resp}$ | Rate of respiratory evaporative heat exchange ($W \cdot m^{-2}$) |
| $\dot{E}_{sk}$ | Rate of evaporative heat loss from the skin ($W \cdot m^{-2}$) |
| $E_{req}$ | Evaporative heat loss required ($W \cdot m^{-2}$) |
| $E_{max}$ | Maximal evaporative heat loss ($W \cdot m^{-2}$) |
| F | Fluid replacement treatment |
| $f_c$ | Heart rate ($b \cdot min^{-1}$) |
| Hb | Hemoglobin ($mM \cdot L^{-1}$) |
| Hct | Hematocrit value adjusted for blood cell packing and arterial-venous differences (%) |

| | |
|---|---|
| HF-Acc | High fit, heat acclimation treatment |
| HSI | Heat stress index ( $E_{req} \cdot E_{max}^{-1}$ ) |
| HST | Heat stress test |
| HY | Hypohydrated condition |
| ICF | Intracellular fluid |
| ISF | Interstitial fluid |
| $(i_m)$ | Woodcock vapour permeability coefficient |
| $I_T$ | Thermal resistance ( $m^2 \cdot ^\circ C \cdot W^{-1}$ ) |
| $\lambda$ | The latent heat of vapourisation of water (675 $W \cdot h \cdot kg^{-1}$ ) |
| L | Distance between the inner pair of band electrodes (cm) |
| LF-Acc | Low fitness, heat acclimation treatment |
| LF-Control | Low fitness, control treatment |
| LF-Train | Low fitness, training treatment |
| $\dot{M}$ | Rate of metabolic heat production ( $W \cdot m^{-2}$ ) |
| $m_b$ | Body mass (kg) |
| NBC | Nuclear, biological, and chemical |
| PV | Plasma volume (L) |
| $P_A$ | Ambient vapour pressure (kPa) |
| $P_{resp}$ | Respiratory vapour pressure (kPa) |
| $P_{sk}$ | Skin vapour pressure (kPa) |
| $\dot{Q}$ | Cardiac output ( $L \cdot min^{-1}$ ) |

| | |
|---|---|
| $\dot{R}$ | Rate of radiative heat exchange ( $W \cdot m^{-2}$ ) |
| RER | Respiratory exchange ratio |
| RPE | Rating of perceived exertion |
| $\rho$ | Density of air (0.001293 $kg \cdot L^{-1}$ ) |
| rho | blood resistivity ( $\Omega \cdot cm^{-1}$ ) |
| $\dot{S}$ | Rate of heat storage ( $W \cdot m^{-2}$ ) |
| SR | Sweat rate ( $L \cdot h^{-1}$ ) |
| STPD | Standard Temperature Pressure Dry |
| SV | Stroke volume (mL) |
| TBW | Total body water (L) |
| $T_{amb}$ | Ambient temperature (°C) |
| $T_c$ | Core temperature (°C) |
| $T_{re}$ | Rectal temperature (°C) |
| $T_{resp}$ | Respiratory temperature (°C) |
| $\Delta T_{re}$ | Change in rectal temperature (°C) |
| $\overline{T}_{sk}$ | Mean skin temperature (°C) |
| TT | Tolerance time (min) |
| $V$ | Walking speed ( $m \cdot s^{-1}$ ) |
| $\dot{V}CO_2$ | Rate of carbon dioxide production ( $mL \cdot kg^{-1} \cdot min^{-1}$ ) |
| $\dot{V}_E$ | Rate of ventilation ( $L \cdot h^{-1}$ ) |

| | |
|---|---|
| $\Delta V$ | Change in volume or stroke volume (mL) |
| VET | Ventricular ejection time (s) |
| $\dot{V}O_2$ | Oxygen uptake ( $mL \cdot kg^{-1} \cdot min^{-1}$ ) |
| $\dot{V}O_2$ max | Maximal oxygen uptake ( $mL \cdot kg^{-1} \cdot min^{-1}$ ) |
| WBGT | Wet bulb globe temperature (°C) |
| $\dot{W}_k$ | External work ( $W \cdot m^{-2}$ ) |
| $Z_0$ | Basal thoracic impedance ($\Omega$) |

*So Shadrach, Meshach, and Abednego came out of the fire,*
*and the satraps, prefects, governors and royal advisers crowded around them.*
*They saw that the fire had not harmed their bodies,*
*nor was a hair of their heads singed;*
*their robes were not scorched,*
*and there was no smell of fire on them.*

*Daniel 3:26-27*

*It's like a sauna in here!*

*Cosmo Kramer*

# 1. GENERAL INTRODUCTION

Workers in industrial settings and military personnel often must seek protection from the ambient environment through the wearing of protective clothing. For the military, the use of nuclear, biological, and chemical (NBC) protective ensembles was essential for the 1991 conflict in the Persian Gulf. However, increased physiological and psychological strain and a decrease in exercise performance and tolerance are well-documented phenomena with the wearing of protective clothing, either in a thermoneutral or a hot environment (Antunano and Nunneley 1992, Aoyagi 1996, Aoyagi et al. 1994, 1995, Duggan 1988, Faff and Tutak 1989, Fine and Kobrick 1987, Goldman 1963, Gonzalez and Cena 1985, Henane et al. 1979, Holmer 1995, Holmer et al. 1992, Joy and Goldman 1968, McLellan 1993, McLellan and Frim 1994, McLellan et al. 1993b, Montain et al. 1994, Nunneley 1988, 1989, Patton et al. 1995, Smolander et al. 1984, Sullivan and Mekjavic 1992, Teitlebaum and Goldman 1972, 1991, White et al. 1989). Protective clothing increases the physical load on the individual through the extra weight of the clothing and also the hobbling effect from the additional bulk and stiffness of the clothing, leading to an increase in metabolic heat production (Duggan 1988, Patton et al. 1995, Teitlebaum and Goldman 1972). The limited water vapour permeability through the clothing impairs the ability of the body to dissipate heat through evaporation of secreted sweat (McLellan et al. 1996, Nunneley 1988, 1989). As a result, there is a greater increase in body heat storage and rise in body temperature than when the clothing is not worn (McLellan 1993, McLellan et al. 1993a). The rise in core temperature has been hypothesised as being the ultimate determinant of fatigue during exercise-heat exposure (Nielsen 1992, 1994).

During exercise-heat stress while unclothed or wearing clothing with high vapour permeability, it is well documented that fluid replacement and hydration status are important determinants of heat tolerance (for reviews see Coyle and Montain 1992b, Gisolfi 1996, Maughan et al. 1993, Millard-Stafford et al. 1992, Noakes 1993, Sawka 1988, 1992, 1993). The production of sweat for evaporative heat transfer during exercise

results in the loss of fluid from the body, impairing cardiovascular and thermal regulation and exercise tolerance (Adolph 1947, Armstrong et al. 1985, 1997, Brouns 1991, Convertino 1987, Craig and Cummings 1966, Fortney et al. 1981, 1984, Gisolfi and Copping 1974, Gonzalez-Alonso et al. 1995, Gore et al. 1992, Harrison 1986, Harrison et al. 1978, Horstman and Horvath 1972, Montain and Coyle 1992b, Rehrer 1994, Senay and Christensen 1965). Hypohydration, a decrease in total body water prior to exercise-heat stress, impairs exercise performance and increases core temperature and heart rate, while decreasing the sweating response and the core temperature at which subject intolerance terminates exercise (Armstrong et al. 1985, 1997, Burge et al. 1993, Buskirk et al. 1958, Cadarette et al. 1984, Caldwell et al. 1984, Candas et al. 1988, Dengel et al. 1992, Ekblom et al. 1970, Greenleaf and Castle 1971, Jacobs 1980, Montain et al. 1995, Nadel et al. 1980, Saltin 1964, 1984, Sawka et al. 1983b, 1985, 1992, Strydom and Holdsworth 1968, Tankersley et al. 1992, Walsh et al. 1994, Zappe et al. 1993).

Humans are capable of improving their response and tolerance to exercise-heat stress. Classic symptoms of adaptation to heat include an increase in sweating response along with a decrease in core and skin temperature, heart rate, and perceived exertion (Armstrong and Maresh 1991, Wyndham 1973). One strategy for improving exercise-heat tolerance is the maintenance of body fluid volume during exercise through the replacement of fluid lost in sweat production. Rehydration during exercise elicits a significant attenuation of cardiovascular and thermal strain compared to its absence (Barr et al. 1991, Brandenberger et al. 1986, Candas et al. 1986, Castellani et al. 1997, Coyle and Montain 1992a, 1992b, Francis 1979, Gisolfi 1996, Guimaraes and Silamigarcia 1993, Hamilton et al. 1991, Lamb and Brodowicz 1986, Marriott 1993, Maughan 1992, Maughan et al. 1993, Millard-Stafford et al. 1992, Montain and Coyle 1992a, Nielsen et al. 1986). Another strategy is to induce a series of physiological adaptations in the individual through either a period of physical training or heat acclimation from repeated heat exposures. Repeated heat exposures over four days to two weeks have been shown to produce

cardiovascular and thermoregulatory adaptations which result in a decreased physiological strain and an increased tolerance during exercise in the heat (Adams et al. 1960, Allan and Wilson 1971, Aoyagi et al. 1995, Armstrong and Kenney 1993, Armstrong and Maresh 1991, Avellini et al. 1980, Candas et al. 1979, Chen and Elizondo 1974, Finberg and Berlyne 1977, Fox et al. 1967, Francesconi et al. 1983, Garden et al. 1966, Gisolfi and Cohen 1979, Gonzalez et al. 1974, Harrison et al. 1981, Horowitz 1989, Horvath and Shelley 1946, Moseley 1994, Nadel et al. 1974, Nielsen et al. 1993, Pandolf 1997, Pandolf et al. 1988, Roberts et al. 1977, Sawka et al. 1983a, Shapiro et al. 1981, Shvartz and Benor 1971, Shvartz et al. 1972, 1973a, 1973b, 1977, Wenger 1988, Wyndham 1967). Improvement in aerobic fitness, accrued from either strenuous long-term training programs of eight weeks or longer or from habitual exercise, is also known to increase an individual's tolerance to exercise-heat stress (Armstrong and Pandolf 1988, Avellini et al. 1980, Buono et al. 1991, Cadarette et al. 1984, Coyle et al. 1986, Gardner et al. 1996, Gisolfi 1973, Gisolfi and Robinson 1969, Havenith et al. 1995, Havenith and van Middendorp 1990, Henane et al. 1977, Pandolf et al. 1977, Piwonka and Robinson 1967, Shvartz et al. 1977). In some settings, such as military operations, there may not be sufficient time or facilities to undertake a long-term endurance training or a heat acclimation program in preparing for exercise in a hot environment. Short-term aerobic training of two weeks or less has been successful in improving thermoregulatory responses during exercise in a normothermic environment (Green et al. 1991a, Green et al. 1991b). The efficacy of short-term training of two weeks or less in inducing heat adaptations is controversial, with reports demonstrating both the presence (Clausen 1977, Dawson 1994, Houmard et al. 1990, Nadel et al. 1974) and absence (Shvartz et al. 1973a, Strydom and Williams 1969, Strydom et al. 1966) of improvements in physiological responses to exercise-heat stress post-training.

Factors known to modify the dynamics of adaptations to heat stress include the aerobic fitness and hydration status of the individual. The efficacy of a heat acclimation

program may be dependent on the fitness status of the individual. Fit individuals adapt more rapidly to heat exposure, with an inverse relationship reported between maximal aerobic power ($\dot{V}O_2$ max) and the number of days required to reach a heat-acclimated state (Pandolf et al. 1977). However, individuals of low to moderate aerobic fitness, without any prior adaptations to heat from long-term training, may experience a greater relative reduction in heat strain from a period of training or heat acclimation than fit individuals (Havenith et al. 1995), as larger decreases in heart rate and rectal temperature post-acclimation have been observed in subjects with a low, as opposed to a high, $\dot{V}O_2$ max (Cadarette et al. 1984, Shvartz et al. 1977). Hydration status also interacts with fitness and heat acclimation in determining the response to exercise-heat stress. Hypohydration eliminates both the fitness-related differences observed in the thermoregulatory responses prior to heat acclimation and any improvements in thermal responses due to heat acclimation (Cadarette et al. 1984, Sawka et al. 1983b).

While fluid replacement, heat acclimation, and high aerobic fitness are generally accepted as being of benefit during exercise-heat stress while unclothed or wearing clothing with high vapour permeability, the effects of these treatments while wearing protective clothing are less clear. Due to the limited water vapour permeability of the protective ensembles, it is possible that the elevated sweating response in endurance trained or heat acclimated individuals, rather than increasing evaporative heat loss, will increase the dehydration and lead to a greater decrease in blood volume and cutaneous blood flow, increasing cardiovascular and thermoregulatory strain (Rowell and Wyss 1985). In this scenario, aerobic fitness, training, or heat acclimation may be counterproductive in improving exercise-heat tolerance.

In individuals wearing protective clothing in the heat, the high rates of heat storage and the resultant short tolerance times may also limit the benefits that may be derived from physiological interventions. Even at relatively low rates of metabolic heat production, the limited evaporative heat loss through the protective clothing combined with a high ambient

temperature can result in situations of severe uncompensable heat stress, where the evaporative cooling requirements ($E_{req}$) greatly exceed the possible cooling capacity of the environment ($E_{max}$) (Givoni and Goldman 1972). With protective clothing, the kinetics of evaporative heat loss are established by the characteristics of the clothing (Craig and Moffitt 1974, Kenney et al. 1987, McLellan et al. 1996). Thus, the rate of heat storage, and therefore, heat tolerance, may be governed primarily by the rate of heat production, negating the effects of inter-individual variations or physiological interventions on determining the level of physiological strain and tolerance (Craig et al. 1954, McLellan 1993, Shvartz and Benor 1972).

Integrating the results of a series of NBC studies in differing warm and hot (30-40°C, 30-50% relative humidity) environments, McLellan (1993) observed that a hyperbolic relationship existed between metabolic rate and tolerance time, and that at work intensities exceeding 400-500W (~15-20 $mL \cdot kg^{-1} \cdot min^{-1}$ $\dot{V}O_2$), tolerance time converged at approximately 50 minutes. Significant differences in tolerance time among different ambient conditions were only noticeable at lighter metabolic rates, where tolerance time was long enough for some heat transfer to occur through the clothing and for differing ambient conditions to have an effect. A physiological intervention, such as fluid replacement, may be ineffective at high metabolic rates. Ingested fluid must first be emptied from the stomach and absorbed in the intestine before it can enter the body and affect the exercise response. Transit time is dependent on many factors including volume ingested, exercise intensity, hydration status, and fluid and ambient temperatures (Costill and Saltin 1974, Fordtran and Saltin 1967, Houmard et al. 1991, Lambert et al. 1996, Levine et al. 1991, Mitchell et al. 1994, Mitchell and Voss 1991, Murray 1987, Neufer et al. 1989a, 1989b, Rehrer et al. 1990, Vist and Maughan 1994). Therefore, if the tolerance time is less than the transit time for a significant volume of fluid to enter the body, fluid replacement may be of no benefit. During a relatively light exercise, fluid replacement may

be of greater benefit if tolerance time is long enough to allow a significant amount of fluid to enter the body and affect responses before exhaustion occurs from other factors.

In predicting the physiological response or tolerance to exercise with protective clothing, it is difficult to extrapolate from the results of studies performed either while unclothed or wearing clothing which allows a large degree of water vapour permeability (Nunneley 1989). Due to the low water vapour permeability across the clothing layers, a microenvironment is created between the skin and the clothing layers. The fit of the individual within the clothing ensemble determines the volume of the microenvironment, which can be up to 50 L, and the rate of heat transfer (Nielsen et al. 1989, Sullivan and Mekjavic 1992, Sullivan et al. 1987). Fabric material, clothing design, posture, pumping action through body motion, and wind speed are other factors that would affect the size and thermal characteristics of the clothing microenvironment (Holmer 1995). The peripheral stimulus for sweating, and the efficiency of sweating and evaporation, are influenced by the increased skin wettedness and hidromeiosis resulting from the greater sweat drippage within the clothing compared to unclothed conditions (Candas and Hoeft 1995, Candas et al. 1980, Kenney et al. 1987). Furthermore, individual variance in the sweating rate within the clothing could influence the degree of clothing saturation and its insulation value (Craig and Moffitt 1974, Kakitsuba et al. 1988). In calculating heat balance with protective clothing, models designed for calculating direct skin to environment heat transfer typically overestimate evaporative heat loss while wearing protective clothing (Craig and Moffitt 1974, Holmer 1995, Kenney et al. 1987). Calculation of skin vapour pressure from humidity sensors placed at the skin and within the clothing layers demonstrated that evaporative heat loss was overestimated when calculated using changes in dressed weight or when the skin was assumed to be saturated and at 100% humidity from the start of exercise (McLellan et al. 1996). Lastly, the combination of clothing fit and saturation could influence psychological discomfort within the clothing.

The interactions between fitness, heat acclimation, fluid replacement, and hydration status while wearing protective clothing require further investigation. Previous studies with protective clothing have generally been restricted to one of the above factors. A high level of aerobic fitness appears to provide some cardiovascular advantages during exercise in protective clothing (Windle and Davies 1996), but it is unknown whether this protection can be replicated with a short-term training program. Heat acclimation is known to produce a significant improvement in exercise-heat tolerance during uncompensable heat stress resulting from light exercise in the heat with NBC clothing (Aoyagi et al. 1995, McLellan and Aoyagi 1996). In these studies, subjects underwent progressive dehydration with no fluid replacement. Thus, it remains unknown whether benefits from heat acclimation would be present where fluid replacement is provided. Overall, an integrated approach towards research on human interactions with protective clothing is required.

# 2. REVIEW OF LITERATURE

Man is a homeothermic creature who regulates his body temperature within a narrow range (Bligh 1985). When heat is generated by increased metabolic activity, humans are generally successful in maintaining thermal steady state by activating heat-loss mechanisms to dissipate the excess heat. A hot environment, however, imposes a major stress on the human body's ability to maintain physiological stability and performance during exercise. A large body of research has focused on exercise in the heat and physiological treatments which may enhance physiological responses and performance, and this research has been summarised in a number of scientific reviews (Armstrong and Maresh 1991, Armstrong and Pandolf 1988, Brouns et al. 1992, Convertino 1987, Fellman 1992, Francesconi 1988, Frisancho 1981, Gisolfi 1996, Gisolfi and Wenger 1984, Gordon and Heath 1986, Harrison 1985, 1986, Havenith 1985, Holloszy 1973, Horowitz 1989, Johnson 1992, Lamb and Brodowicz 1986, Maughan et al. 1993, Millard-Stafford 1992, Murray 1987, Noakes 1993, Pandolf 1997, Rowell 1993, Rowell and Wyss 1985, Santee and Gonzalez 1988, 1988, 1992, 1993, Sawka and Wenger 1988, Senay 1987, Taylor 1986, Wenger 1988).

In certain circumstances, humans have to perform exercise in environments of extreme heat stress or while wearing protective clothing which impairs heat transfer. In these situations, heat generation exceeds heat dissipation, resulting in continued heat storage within the body and the inability to maintain thermal steady state. This condition is termed uncompensable heat stress (Givoni and Goldman 1972). The focus of the following review will be to summarise the present knowledge on the physiological responses to uncompensable heat stress, focusing on the influences of wearing protective clothing and of alterations in hydration status. In addition, the effects of aerobic fitness, aerobic training, and heat acclimation as treatments to improve exercise-heat tolerance will be discussed. For other reviews on problems associated with exercise in the heat with protective clothing, the reader is referred to the following scientific publications (Aoyagi et al. 1997, Gonzalez 1988, Haslam and Parsons 1987, Holmer 1995, McLellan and Frim

1994, Nunneley 1988, 1989, Parsons 1988, Shitzer and Chato 1985, Shitzer and Eberhart 1985).

## 2.1. Protective Clothing

### *2.1.1. Clothing Characteristics*

When unclothed, the regulation of thermal energy exchanges between humans and the ambient environment can occur directly across the skin. When clothing is worn, however, an air layer is formed directly above the skin surface which forms the initial environmental layer between the body and the environment. The volume of this microenvironment formed between the clothing and skin can be as large as 50 L for industrial protective clothing (Sullivan et al. 1987). Multiple clothing layers form a successive series of microenvironments, each with its own thermal characteristics of temperature and humidity, through which metabolically generated heat must pass before being dissipated to the ambient environment (Holmer 1995, Sullivan and Mekjavic 1992). Therefore, the thermal properties of clothing, such as its insulation, ventilation, and permeability to water vapour, have a significant influence on the rate of heat transfer between the skin and the ambient environment (Gonzalez 1988, Holmer 1995, Shitzer and Chato 1985).

For the purposes of this work, protective clothing is defined as a clothing ensemble which is designed to protect the human body from the ambient environment, which fully encapsulates the body, and which significantly impairs heat transfer because of its high insulation and low permeability to water vapour. The ideal protective clothing would provide protection from the environment while allowing complete permeability to water vapour in order to allow maximal evaporative heat exchange, presently an unobtainable goal. One example of a multilayered clothing ensemble with a high insulative capacity and a low permeability to water vapour is the nuclear, biological, and chemical (NBC) protective clothing worn by the Canadian Forces (CF) and typical of those in use by many

military forces. The CF NBC protective ensemble consists of, in addition to the standard underwear and operational clothing, a respirator, semipermeable hooded overgarment, and impermeable rubber boots and gloves. Total mass of the ensemble is approximately 6.0 kg. In order to allow some sweat evaporation, there is a limited mass penetration of charcoal-filtered air through the fabric. Thermal resistance ($I_T$) and the Woodcock vapour permeability coefficient ($i_m$) of the ensemble determined on a heated and wetted manikin at a wind speed of 1.12 $m \cdot s^{-1}$ were 0.291 $m^2 \cdot °C \cdot W^{-1}$ (1.88 clo) and 0.33, respectively (Gonzalez et al. 1993). Interest in the problems of exercise and heat exhaustion with the NBC clothing intensified during the 1990-1991 conflict in the Persian Gulf, where soldiers of the Allied coalition, faced with the threat of chemical weapons, were forced to wear NBC clothing that was originally intended for use in a temperate climate and not the hot desert environment.

### *2.1.2. Uncompensable Heat Stress*

Protective clothing can influence heat transfer dynamics by altering the rate of production and/or the dissipation of metabolically generated heat. The rate of heat storage, $\dot{S}$, in $W \cdot m^{-2}$, when wearing clothing in the heat, is modified from the basic heat balance equation derived from the First Law of Thermodynamics as follows (McLellan et al. 1996):

$$\dot{S} = \dot{M} - (\pm \dot{W}_k) \pm (\dot{R} \pm \dot{C}) \pm \dot{C}_{resp} \pm \dot{E}_{resp} \pm \dot{E}_{sk} \qquad (W \cdot m^{-2}) \qquad \text{(Eqn. 2.1)}$$

The rate of metabolic heat production, $\dot{M}$ in $W \cdot m^{-2}$, is determined from the measured $\dot{V}O_2$, in $L \cdot min^{-1}$, the respiratory exchange ratio (RER), and the Dubois body surface area ($A_D$), in $m^2$, as (Nishi 1981):

$$\dot{M} = 352 \cdot (0.23 \cdot R + 0.77) \cdot (\dot{V}O_2 \cdot A_D^{-1}) \qquad \text{(Eqn. 2.2)}$$

The external work, $\dot{W}_k$ in $W \cdot m^{-2}$, is calculated using the walking speed, $V$ in $m \cdot s^{-1}$, the grade fraction as a decimal percentage, and the mass, $m$ in kg, as:

$$\dot{W}_k = 9.8 \cdot (grade) \cdot V \cdot m \cdot A_D^{-1} \qquad \text{(Eqn. 2.3)}$$

The radiative and convective heat exchange, $\dot{R}$ and $\dot{C}$ in $W \cdot m^{-2}$, are estimated using the thermal resistance of the NBC clothing ensemble, $I_T$, of 0.291 $m^2 \cdot {}^\circ C \cdot W^{-1}$ (1.88 clo) determined using a heated and dry copper manikin at a wind speed of 4.0 $km \cdot h^{-1}$ (Gonzalez et al. 1993), and the difference between the ambient ($T_{amb}$) and mean skin temperature ($\overline{T}_{sk}$) as (Gonzalez et al. 1993):

$$\dot{R} + \dot{C} = (T_{amb} - \overline{T}_{sk}) \cdot I_T^{-1} \qquad \text{(Eqn. 2.4)}$$

Respiratory evaporative heat exchange, $\dot{E}_{resp}$ in $W \cdot m^{-2}$, and convective heat exchange, $\dot{C}_{resp}$ in $W \cdot m^{-2}$, are calculated using ambient vapour pressure, $P_A$ in kPa, respired vapour pressure, $P_{resp}$ in kPa, the density of air, $\rho$ (=0.001293 $kg \cdot L^{-1}$), the heat capacity of dry air, $c_{pa}$ (=0.28 $W \cdot h \cdot kg^{-1} \cdot {}^\circ C^{-1}$), the heat capacity of water vapour, $c_{pwv}$ (=0.52 $W \cdot h \cdot kg^{-1} \cdot {}^\circ C^{-1}$), the measured $\dot{V}_E$ in $L \cdot h^{-1}$ (STPD), the latent heat of vapourisation, $\lambda$ (=675 $W \cdot h \cdot kg^{-1}$), and the difference in the humidity ratio,

$$W_{resp} - W_A \text{ of } 0.622 \cdot [P_{resp} / (101 - P_{resp}) - P_A / (101 - P_A)] \qquad \text{(Eqn. 2.5)}$$

$$\dot{E}_{resp} = \rho \cdot \lambda \cdot V_E \cdot (W_{resp} - W_A) \cdot A_D^{-1} \qquad \text{(Eqn. 2.6)}$$

$$\dot{C}_{resp} = \rho \cdot V_E \cdot (T_{resp} - T_{amb}) \cdot A_D^{-1} \cdot (c_{pa} + c_{pwv} \cdot W_A) \qquad \text{(Eqn. 2.7)}$$

Evaporative heat loss from the skin, $\dot{E}_{sk}$ in $W \cdot m^{-2}$, is determined using the ambient vapour pressure, $P_A$ in kPa, the skin vapour pressure, $P_{sk}$ in kPa, assuming 100% saturation at the skin temperature, the Lewis relation of 16.5 $^\circ C \cdot kPa^{-1}$, the thermal resistance value, $I_T$ in $m^2 \cdot {}^\circ C \cdot W^{-1}$, and the Woodcock vapour permeability coefficient ($i_m$), as (Gonzalez et al. 1993):

$$\dot{E}_{sk} = 16.5 \cdot (i_m \cdot I_T^{-1}) \cdot (P_{sk} - P_a) \qquad \text{(Eqn. 2.8)}$$

The wearing of protective clothing can result in a 13-18% increase in metabolic rate and internal heat production compared with normal operational clothing during treadmill exercise (Aoyagi et al. 1994, Duggan 1988, Patton et al. 1995, Smolander et al. 1984). The entire NBC ensemble weighs approximately 6 kg, and thus, part of the increase in metabolic rate is due to the added weight. However, other factors, such as

friction between clothing layers and the bulkiness of the clothing, alter gait mechanics and the efficiency of movement. These factors also contribute to the increase in metabolic rate, which has been shown to be significantly higher while wearing protective clothing compared to an equivalent weight worn as a lead-filled belt (Duggan 1988, Teitlebaum and Goldman 1972).

Mechanisms of heat transfer are grouped into two general categories consisting of dry (radiative, conductive, convective) and wet (evaporative) pathways (Sawka and Wenger 1988). Dry heat exchange is dependent on the temperature gradients within the organism (e.g., core to periphery) and also between the organism and the environment. In addition, the rate of cutaneous blood flow to transport heat from the core to the periphery influences the degree of convective heat exchange. Wet heat loss arises from the evaporation of water, typically secreted by the sweat glands within the skin (Taylor 1986). The potential for evaporative heat loss is determined primarily by the water vapour pressure gradient between the body surface and the environment, which in turn may be modified both by the environment and clothing (Craig and Moffitt 1974, Kakitsuba et al. 1988, Kenney et al. 1987, McLellan et al. 1996) as well as physiologically due to alterations in sweat gland activity and output (Alber-Wallerstrom and Holmer 1985, Candas et al. 1979, Kerslake 1972).

When the temperature of the ambient environment is similar to that found at the skin, as is the case during exercise in the heat at approximately 35-40°C while wearing protective clothing (McLellan and Frim 1994), the primary pathway for heat dissipation is from the evaporation of secreted sweat (Wenger 1972). Maximal evaporative heat dissipation from the body occurs when secreted sweat is vapourised at the skin (Nadel 1979). When wearing protective clothing, much of the sweat may become absorbed into the clothing and trapped. The wetting or saturation of clothing by sweat may affect the thermal characteristics of the clothing and influence the rate of heat transfer (Craig and Moffitt 1974, Kakitsuba et al. 1988, Kenney et al. 1987). In addition, as diagrammed in

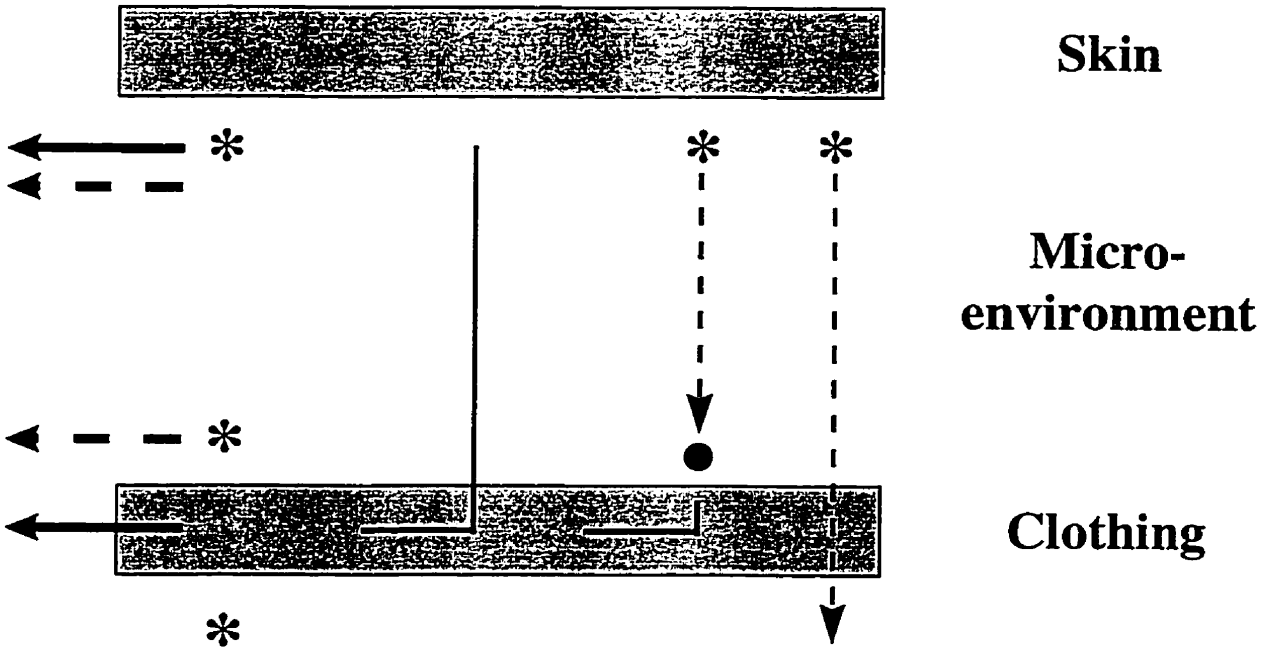
Figure 2.1, the site of phase change may be raised above the skin in a clothing microenvironment, where a portion of the heat energy may come from the environment rather than the body, thus decreasing the efficiency of evaporative heat loss (McLellan et al. 1996, Nunneley 1989).

Due to the elevation in metabolic heat production and a decrease in evaporative efficiency, wearing protective clothing in the heat often results in an inability to dissipate generated heat and a storage of heat in the body. Table 2.1 presents the approximate evaporative heat loss required ($E_{req}$) to maintain a thermal steady state, the maximal evaporative heat loss possible in the environment ($E_{max}$), and a heat stress index ($HSI = E_{req} \cdot E_{max}^{-1}$) values for an individual wearing the Canadian Forces NBC clothing while at rest or working in a warm and a hot environment. $E_{req}$ is taken as the heat storage value ($\dot{S}$) in Equation 2.1 less the value for evaporative heat loss from the skin ($\dot{E}_{sk}$). $E_{max}$ is taken as the $\dot{E}_{sk}$ in Equation 2.8 assuming 100% saturation at the temperature of the skin. As demonstrated in the table, due to the reduced permeability to water vapour and limited capacity for evaporative heat dissipation through the NBC protective clothing, even light exercise in a warm environment or very light exerise in a hot environment can produce a HSI > 1.0, producing a situation of uncompensable heat stress and continued heat storage in the body (Holmer 1995, McLellan 1993).

### *2.1.3. Exercise Tolerance*

Protective clothing has a significant effect on the physiological strain experienced during exercise. Heat tolerance, defined as the exercise time before voluntary exhaustion is reached or at which ethical considerations require the termination of exercise, can be reduced substantially when protective clothing is worn compared with a clothing configuration, such as normal operational clothing, which provides a more effective heat exchange with the environment. Even in a thermoneutral ambient environment, firefighting or gas protective clothing increased heart rate, core and skin temperatures, and rating of

Figure 2.1. Diagram of pathways for secreted sweat when clothing is worn. From Nunneley (1989). Note that evaporation can take place at either the skin or at several sites away from the skin.




* = sites of evaporation, filled circle = condensation, broken lines = water vapour, solid lines = liquid water

Table 2.1: A comparison of the heat stress index (HSI) in different environments (assuming ambient vapour pressure of 1.0 and 2.2 kPa, respectively) for an individual (assuming surface area of 2.0 $m^2$) at metabolic rates representing very light, light, moderate, and heavy metabolic rates for the military (assuming a respiratory exchange ratio of 0.85) while wearing the Canadian Forces nuclear, biological, and chemical protective ensemble (thermal resistance and vapour permeability coefficient of 0.291 $m^2 \cdot °C \cdot W^{-1}$ (1.88 clo) and 0.33, respectively). The equations are detailed in Section 2.1.2. Skin temperature in all conditions was assumed to be constant at 37.0°C. External work and convective and evaporative heat exchange from the respiratory tract was assumed to be nonexistent. The evaporative heat loss required to maintain thermal steady state ($E_{req}$) was divided by the maximal evaporative heat loss possible in the environment ($E_{max}$) to obtain HSI. A HSI<1.0 indicates a compensable heat stress, while a HSI>1.0 indicates a positive rate of heat storage within the body and an uncompensable heat stress.

| Metabolic Rate ($\dot{V}O_2$, $L \cdot min^{-1}$) | | 30°C, 10% RH ($W \cdot m^{-2}$) | HSI | 40°C, 30% RH ($W \cdot m^{-2}$) | HSI |
|---|---|---|---|---|---|
| Very Light | $E_{req}$ | 60 | 0.63 | 95 | 1.36 |
| (0.5) | $E_{max}$ | 95 | | 70 | |
| Light | $E_{req}$ | 145 | 1.53 | 180 | 2.57 |
| (1.0) | $E_{max}$ | 95 | | 70 | |
| Moderate | $E_{req}$ | 230 | 2.42 | 265 | 3.79 |
| (1.5) | $E_{max}$ | 95 | | 70 | |
| Heavy | $E_{req}$ | 315 | 3.32 | 350 | 5.00 |
| (2.0) | $E_{max}$ | 95 | | 70 | |

perceived exertion (RPE) (Duncan et al. 1979, Faff and Tutak 1989, 1991, White et al. 1989). At either light or heavy work intensities, the use of NBC clothing dramatically reduced tolerance time compared with combat clothing alone (McLellan et al. 1993, Montain et al. 1994). Indeed, the impairment of heat transfer while wearing NBC clothing in the heat has been reported to be so severe that body heat storage and an increase in core temperature could exist even under resting conditions (McLellan 1993). The importance of protective clothing in determining exercise-heat tolerance is further demonstrated by the significant attenuation of physiological strain and/or increased tolerance time during partial encapsulation achieved by the removal of components of the NBC ensemble and unzipping the overgarment (Amos and Hansen 1997, McLellan et al. 1993, Montain et al. 1994), or else the removal of the combat clothing layer (McLellan 1996).

The kinetics of evaporative heat loss are largely established by the characteristics of the clothing (Craig and Moffitt 1974, Kenney et al. 1987, McLellan et al. 1996). Under certain conditions, the rate of heat storage and heat tolerance may be governed primarily by the rate of heat production (McLellan 1993). Variations in ambient water vapour pressure, which help to establish the gradient for evaporative heat loss, may have little impact on heat tolerance (McLellan 1993). Craig et al. (1954) and Shvartz and Benor (1972) reported a hyperbolic relationship between voluntary tolerance time and the rate of heat storage. Integrating the results of a series of NBC studies in differing warm and hot environments, McLellan (1993) observed that a hyperbolic relationship existed between metabolic rate and tolerance time, and that at work intensities exceeding 450-500 W (~15-20 $mL \cdot kg^{-1} \cdot min^{-1}$ $\dot{V}O_2$), tolerance times converged at approximately 50 min. Significant differences in tolerance time among different ambient conditions were only noticeable at lighter metabolic rates, where tolerance time was long enough for some heat transfer to occur through the clothing and for differing ambient conditions to have an effect. In a subsequent study, McLellan et al. (1996) reported some variation in tolerance

time at high metabolic rates between low and high ambient humidity, though the variation remained greater at lower exercise intensities.

### *2.1.4. Individual Characteristics*

Individual characteristics can have a significant influence on the response to exercise-heat stress. Gardner et al. (1996) reported that the Marine Corps recruits most at risk for developing exertional heat illness during basic training had a body mass index ($BMI = mass \cdot height^{-2}$) of over 22 $kg \cdot m^{-2}$ and a time for a 1.5 mile run in excess of 12 min at the start of basic training, suggesting an influence of both anthropometric measures and aerobic fitness. Inter-individual variations in fitness, anthropometric measures, and hydration and acclimation status can significantly influence the response to exercise-heat stress when clothing is not worn (Havenith 1985, Havenith et al. 1995, Havenith and van Middendorp 1990, Kenney 1985). In contrast, the influence of individual characteristics on the response to exercise while wearing protective clothing is less clear. Since the clothing limits evaporative heat loss, inter-individual variations in anthropometric measures or aerobic fitness may have only a minimal influence on determining the level of physiological strain and tolerance. Shvartz (1973a) reported that size, surface area, and the surface area-to-mass ratio did not influence the rate of heat storage while wearing vapour barrier clothing in hot environments due to the elimination of a thermal gradient for dry heat transfer and the lack of difference in the rate of evaporative heat dissipation through the clothing.

A high level of aerobic fitness or physiological treatments, such as aerobic training and heat acclimation, may be of limited effectiveness in decreasing physiological strain or prolonging tolerance during exercise-heat stress with protective clothing. Due to the limited evaporative heat loss possible through the clothing, the increased sweat production associated with aerobic fitness, training, or heat acclimation (Nadel et al. 1974, Shvartz et al. 1974) may not result in an increased evaporative heat loss when wearing protective

clothing. Rather, the elevated sweat rate may potentiate the rate of dehydration (Windle and Davies 1996). Smolander et al. (1987) did not observe any difference in physiological responses to exercise-heat stress between trained and untrained subjects while wearing light work clothing. In contrast, while wearing NBC clothing in the heat, fit subjects had a decreased heart rate but no differences in aural or skin temperature (Windle and Davies 1996). Neither an eight-week aerobic training program, which increased $\dot{V}O_2$ max by 16%, nor 6 d of heat acclimation, produced significant improvements in heat tolerance in subjects wearing NBC clothing (Aoyagi et al. 1994). In the latter two studies, however, the metabolic rate used to evaluate the changes in heat tolerance may have produced heat exhaustion before a significant amount of heat transfer could occur through the clothing layers, negating any physiological adaptations due to the treatment programs. Other studies with NBC clothing at a lighter metabolic rate demonstrated an improvement in exercise-heat tolerance with heat acclimation (Aoyagi et al. 1995, McLellan and Aoyagi 1996).

### 2.2. Hydration Status

Total body water (TBW) is generally categorised into either the intracellular (ICF) or extracellular (ECF) fluid compartments, with the latter further divided into the interstitial fluid volume (ISF) and the plasma volume (PV). Total body water content is dependent on individual characteristics, such as fitness, body fatness, and glycogen storage (Neufer et al. 1991, Sawka 1988). Assuming that TBW and ICF represent 60% and 40%, respectively, of the total body mass of a typical male (Sawka 1992), a 75 kg individual would consist of 45 L of water, with an ICF of 30 L and an ECF of 15 L. Blood volume (BV) in healthy young males is approximately 5 L, or 70 $mL \cdot kg^{-1}$, for a 75 kg individual, with about 3 L plasma volume (Sawka et al. 1992b). BV can range up to 100 $mL \cdot kg^{-1}$ blood volume in trained endurance athletes (Dill et al. 1974, Kjellberg et al. 1949). A high correlation has been reported between aerobic fitness and blood volume (Convertino

1991), though Sawka et al. (1992b) proposed that this correlation was due to the covariance between $\dot{V}O_2$ max and lean body mass, and that the latter variable provided a better predictor of blood volume.

Water balance during exercise is determined by a multitude of factors, including the environmental conditions, the nature and intensity of exercise, and the characteristics of the fluid replacement (for review see Adolph 1947, Sawka 1988, 1992). Two terms related to hydration status that are often mistakenly used interchangeably are dehydration and hypohydration. For the present work, dehydration will be defined as the dynamic loss of body water due to sweating over the course of exercise without fluid replacement, or where fluid replacement does not match the rate of fluid loss. In contrast, hypohydration will refer to the state or level of hydration after the loss of a certain amount of body water from the body. As an example of the differences between the two terms, a rower may dehydrate himself through exercise without fluid intake to make a certain weight category, then compete in the actual event in a hypohydrated state.

Sweat rates exceeding 1 $L \cdot h^{-1}$ are typical during moderate exercise, with a high of 3.7 $L \cdot h^{-1}$ having been recorded during the 1984 Olympic Marathon (Armstrong et al. 1986). To a certain extent, mild dehydration during exercise is tolerated by an individual (Armstrong et al. 1985, Broad et al. 1996, Decastro 1992, Engell et al. 1987, Gore et al. 1993, Greenleaf 1992, Hubbard et al. 1984). Even with an adequate fluid supply, the rate of *ad libitum* fluid intake rarely matches the rate at which fluid is lost, and an individual will gradually become dehydrated (Armstrong et al. 1985, Greenleaf 1992). An involuntary dehydration of 2% or more of body weight may occur before a strong drinking response is observed (Adolph 1947, Armstrong et al. 1985). If the fluid lost through sweat production is not adequately replaced, however, even minor levels of hypohydration can impair cardiovascular and thermal regulation and exercise performance (Ekblom et al. 1970).

The following section will discuss the impairment in cardiovascular and thermal regulation and exercise performance brought about by dehydration. The topic of fluid replacement during and/or after exercise covers many areas, and at present, there is controversy about the composition of the fluid and the protocol recommended for the rehydration process. This review will be limited to the factors that impact on the use of water for rehydration.

### *2.2.1. Effects of Dehydration*

During prolonged exercise, the loss of body fluids through sweating without adequate replacement results in dehydration and the impairment of the body's cardiovascular and thermal regulatory abilities (Gonzalez-Alonso et al. 1995, Hamilton et al. 1991, Rowell 1974). Dehydration of 2.9% body weight over the course of 2 h of cycling in a normothermic environment resulted in a significantly greater decrease in blood volume compared with a fluid replacement of 100% of sweat rate (Hamilton et al. 1991). The plasma volume decreased by 9% over the course of exercise, contributing to a decreased stroke volume (SV), an increased heart rate ($f_c$), and an overall decrease in cardiac output ($\dot{Q}$). In addition, rectal temperature ($T_{re}$) was 0.6°C greater without fluid replacement, with most of the cardiovascular and thermoregulatory changes occurring during the second hour of exercise. Exercise in the heat would likely potentiate the effects of dehydration due to greater demands for thermoregulatory blood flow (Sawka 1988). Similar cardiovascular drift and reductions in $\dot{Q}$, along with a continued rise in core temperature ($T_c$), were observed with a dehydration of 4.9% body weight compared with the maintenance of euhydration while cycling in a warm (35°C) environment (Gonzalez-Alonso et al. 1995). Whereas core temperature stabilised with euhydration at ~38.2°C between 60 and 120 min of exercise, the decreased $\dot{Q}$ with dehydration resulted in a progressive reduction in cutaneous and systemic vascular conductance, impairing thermoregulation and resulting in a continued increase in core temperature to nearly

39.5°C following 120 min of exercise (Gonzalez-Alonso et al. 1995). In a warm (32°C) environment, graded levels of dehydration during 2 h of moderate cycling exercise, achieved by varying the amount of fluid replacement, produced graded increases in heart rate and decreases in stroke volume, along with proportionally graded increases in esophageal temperature ($T_{es}$) (Montain and Coyle 1992). In addition, fluid replacement also attenuated the rise in core temperature by increasing skin blood flow and produced a progressive decrease in the ratings of perceived exertion (RPE) (Montain and Coyle 1992).

As fluid for sweat production is drawn from the ICF of the muscle fibres, muscle function may become impaired due to the depletion of muscle water. Maximal strength and muscle force generation do not appear to be significantly affected, with no difference in the maximal power output during 5 s of cycling immediately following dehydration of 1.8% body weight (Walsh et al. 1994). With high intensity exercise of a short duration, progressive dehydration of 1.8% body weight significantly increased RPE and decreased the cycling time to exhaustion from 9.8 to 6.8 min during a subsequent exercise bout at 90% $\dot{V}O_2$ max (Walsh et al. 1994). Whether prolonged high intensity performance is impaired with progressive dehydration is unclear. Despite a 1.9% decrease in body weight over the course of a simulated basketball game, no difference in anaerobic power or jumping height was observed whether subjects received fluid replacement or not, although a non-significant trend towards a progressive decrease in shooting accuracy with continued dehydration might suggest a possible practical impairment in motor performance (Hoffman et al. 1995).

With continued dehydration, fluid losses from the various body fluid compartments occur at different rates. With exercise, the production of a hyposmotic sweat results in a hyperosmotic hypovolemia within the intravascular space, producing a fluid shift from the ICF to the ECF to defend BV (Nose et al. 1988). In general, a larger proportion of the fluid loss is initially drawn from the ECF compared with the ICF (Costill et al. 1976,

Durkot et al. 1986, Nose et al. 1983). With continued dehydration, a greater proportion is derived from the ICF (Costill et al. 1976, Durkot et al. 1986). Beyond a 6% decrease in total body water, Costill et al. (1976) observed a roughly even distribution of fluid loss from the ICF and ECF in humans, while Nose et al. (1983) reported that 41% and 59% of the water deficit was derived from the ICF and ECF, respectively, in rats dehydrated by 10% body weight. In humans, a roughly constant 10% of the total water deficit was derived from the plasma in dehydration of up to 9% of total body water (Costill et al. 1976). Within the ICF, fluid loss is apportioned selectively from the various tissues, likely to prevent crippling losses in vital organs. The majority of the ICF loss in dehydrated rats was from the muscles and the skin (40% and 30%, respectively), with only minimal losses from the brain and liver (Nose et al. 1983). Despite dehydration and the loss of electrolytes, Costill et al. (1976) calculated no significant effect on the resting muscle membrane potential.

Following exercise or heat exposure, the recovery of blood volume appears to be rapid and selectively favoured over other body fluid compartments. Costill and Fink (1974) reported a 7% plasma volume recovery within the initial 30 min following exercise or heat exposure. Following a dehydration of 2.3% body weight with 90-110 min of mild exercise in the heat, plasma volume was decreased by 9.4% (Nose et al. 1988). Despite not receiving any fluid following exercise, plasma volume recovered to within 5.0% and 5.6% of the pre-exercise value within 30 and 60 min, respectively. No changes were observed in the relative size of the ICF versus the ECF during this recovery period, suggesting that the partial recovery of plasma volume was due to fluid from the ISF. Part of the recovery may be from the dermal tissues, with a swollen interstitial compartment due to peripheral vasodilation and sweating (Costill and Fink 1974). With only a partial rehydration consisting of 1% of body weight, a rapid and selective recovery of plasma osmolality and a replenishment of over 96% of the plasma volume lost during an exercise and heat-induced dehydration of 3% body weight occurred within 30 min of the cessation

of exercise for both endurance-trained men and women (Stachenfeld et al. 1996). Plasma volume recovery is likely favoured due to both the increased plasma [$Na^+$] and osmolality along with an increased intravascular oncotic pressure from the post-exercise influx of albumin from the ISF (Convertino et al. 1980b, Gillen et al. 1991, Harrison et al. 1975).

### *2.2.2. Effects of Fluid Replacement*

As discussed above, the maintenance of an euhydrated state during the course of exercise through the replacement of sweat loss produced benefits in exercise performance. Fluid replacement during exercise contributes to the maintenance of plasma volume during exercise, aiding thermal and cardiovascular homeostasis (Candas et al. 1988, Candas et al. 1986). The importance of fluid replacement was illustrated by Candas et al. (1988). During prolonged exercise in the heat, heart rate and $T_{re}$ were significantly higher beyond 2 h in subjects who were initially euhydrated but given no fluid replacement compared with when they were 2% hypohydrated prior to the exercise but given fluid replacement throughout.

The degree of rehydration possible during exercise depends on a wide range of factors, from individual drinking behaviour, environmental parameters, the nature of the activity or the rules or the sport, the exercise intensity, and the nature of the fluid itself (Broad et al. 1996, Gore et al. 1993, Greenleaf 1992). A drink temperature of 15°C had the highest consumption rate in subjects allowed to choose the water temperature (Boulze et al. 1983). Cooling and flavouring drinks had an additive effect on consumption rate, with a 120% greater consumption of 15°C flavoured water compared with 40°C unflavoured water (Hubbard et al. 1984). The difference in consumption rate may be explained either as an affinity for cool and flavoured fluid or else a negative response to warm or unflavoured fluid (Hubbard et al. 1984).

Nose et al. (1990) directly altered blood volume during exercise, and bypassed the time delay required for fluid ingestion and absorption, by infusing subjects with saline.

Without infusion, plasma volume decreased 5% during exercise, and forearm blood flow plateaued once core temperature exceeded 37.7°C. Saline infusion prevented a significant decrease in plasma volume during exercise, elicited a significantly smaller increase in heart rate and esophageal temperature, and increased forearm blood flow throughout the exercise.

Before fluid replacement can have a beneficial effect on the body, it must be emptied from the stomach and absorbed from the intestines. Balanced against ingesting large volumes of fluid to prevent dehydration is the danger of large gastric volumes causing discomfort and impaired performance (Mitchell and Voss 1991). This is especially dangerous if gastric emptying is impaired by either heat exposure or exercise. Gastric emptying is a dynamic function that is affected by many different factors. With an increase in exercise intensity, the decreased splanchnic blood flow due to increased metabolic demands for muscle blood flow has been assumed to inhibit gastric emptying and intestinal absorption (Rowell 1974). Rather than a progressive decrease in the rate of gastric emptying with increasing exercise intensity, Neufer et al. (1989b) observed an increase in gastric emptying when walking or running at < 70% $\dot{V}O_2$ max compared with resting values, possibly due to an increase in stomach motility from the walking motion. Only at exercise intensities > 70% $\dot{V}O_2$ max was the rate of gastric emptying decreased. Fordtran and Saltin (1967) found no impairment in gastric emptying of a glucose solution while exercising at 71% $\dot{V}O_2$ max on the treadmill.

In addition to exercise intensity, several other factors can influence gastric emptying. Thermal strain decreased gastric emptying rate, with a lower rate being observed when exercising at 49°C compared with 18°C (Neufer et al. 1989a). While a period of heat acclimation had no effect on the rate of emptying from the stomach, hypohydration of 5% body weight decreased the rate of gastric emptying (Neufer et al. 1989a). The mode of exercise does not appear to affect gastric emptying rates, as a similar low value compared to that at rest was observed when exercising at 75% $\dot{V}O_2$ max for

either cycling or running (Houmard et al. 1991). The rate of gastric emptying is also influenced by the rate of fluid replacement, and subsequently, the volume of the stomach contents, with a direct relationship being observed between the amount of fluid ingested and the rate of gastric emptying (Mitchell and Voss 1991).

The timing of fluid replacement may also play an important role in exercise responses. Traditionally, athletes have been advised to drink before exercise and also constantly throughout exercise, rather than wait until late in the exercise to begin rehydration. However, the timing of fluid replacement was not found to be significant by Montain and Coyle (1993), who gave a single large bolus of carbohydrate beverage to trained cyclists at either the beginning of exercise or after 40 and 80 minutes. The rise in $T_{es}$ and $f_c$ during exercise was transiently attenuated in each period immediately following fluid ingestion. However, no difference was observed in the final $T_{es}$ or heart rate among the different rehydration schedules, and the magnitude of hypovolemia during exercise was not altered. Interestingly, drinking the same overall amount of fluid continuously during exercise also did not significantly alter $T_{es}$ and $f_c$ at the end of the 140 min of exercise. Unfortunately, this study lacked a control group that did not ingest fluid during exercise, and therefore, it is difficult to comment on the magnitude of improvement brought about by rehydration. A single bolus of water of 50% of the weight lost during prior passive dehydration (2.6% body weight) restored body fluid balance significantly faster than the same volume given in four equal doses 15 min apart during exercise (Melin et al. 1994). Heart rate and $T_{re}$ were significantly lower than with no rehydration throughout exercise at 50% $\dot{V}O_2$ max, and tolerance time was significantly increased. With the single versus dosed rehydration protocols, tolerance times were similar, but heart rate and $T_{re}$ were higher with a dosed rehydration until 50-70 min of exercise.

### *2.2.3. Effects of Hypohydration*

Voluntary loss of body weight and water is often used by athletes to achieve a certain weight category prior to an athletic event. Typically, some time occurs between the weigh-in and competition, in which athletes attempt to restore the lost body weight. The practice of voluntarily dehydrating to reach a weight category should be balanced with the possible decrements in performance. Alternatively, in an industrial setting, inadequate time for rehydration and recovery may be an unintentional or unavoidable cause of hypohydration prior to a subsequent work session.

Conflicting conclusions have been advanced with regards to the influence of hypohydration on anaerobic performance or sports of short duration and relatively high intensity. Webster et al. (1990) observed decreased anaerobic power and anaerobic capacity during a short-duration and supramaximal-intensity Wingate test in a group of wrestlers hypohydrated by 5% body weight, an observation that contradicts the finding of no effect on the Wingate test at either 2, 4, or 5% hypohydration in another group of wrestlers (Jacobs 1980).

The effects of hypohydration on aerobic exercise performance is also equivocal. During a progressive incremental cycling exercise to exhaustion in a thermoneutral environment, Dengel et al. (1992) observed no differences in submaximal values for $\dot{V}_E$, $\dot{V}O_2$, $f_c$, RER, or the lactate threshold in subjects who were euhydrated or hypohydrated by 3.3 and 5.6% body weight. However, Webster (1990) observed a decrease in $\dot{V}O_2$ peak and the running velocity at the lactate threshold at 5% hypohydration. In a simulation of a rowing scenario, Burge et al. (1993) dehydrated subjects by 5% the day before a time trial on a rowing ergometer. Partial rehydration was provided over the recovery period, but the subjects remained hypohydrated prior to the time trial. Work output over every 30 s of the time trial was less with hypohydration and partial rehydration compared with a separate euhydration trial, and the time required to complete

the test was increased significantly. Even though the euhydration trials were not preceded by a session of dehydration and recovery to control for the influence of prior exercise on rowing performance, this was a realistic simulation of hypohydration as used in competition, and serves to illustrate the tradeoff between reaching a particular weight category and performance. Similar increases in 5,000 m and 10,000 m running times with hypohydration were observed in trained distance runners (Armstrong et al. 1985).

With aerobic exercise of lighter intensity and longer duration in the heat, where the limit to exercise tolerance is primarily determined by thermoregulatory considerations, the detrimental effect of hypohydration is clear (Sawka 1988, 1992). In a hot environment or in situations of uncompensable heat stress, the additional stress imposed by hypohydration appears to potentiate the effects of heat stress and severely impairs the ability of individuals to tolerate exercise. A critical hypohydration threshold for exercise impairment does not appear to exist in the heat, where even relatively minor levels of hypohydration are sufficient to produce impairment in the heat. Ekblom et al. (1970) reported an elevated core temperature during exercise with a hypohydration of only 1% body weight. Hypohydration of 5% resulted in an elevation of plasma cortisol and growth hormone (Francesconi et al. 1984). Whereas exercise increased plasma volume when subjects were euhdyrated and had no effect on erythrocyte volume, hypohydration resulted in a lower resting plasma volume and a decrease in both plasma and erythrocyte volume during subsequent treadmill exercise in heat-acclimated individuals (Sawka et al. 1984). However, the degree of hypohydration, the acclimation status, and the exercise employed may affect body fluid responses, as Zappe et al. (1993) observed a similar rate of hemoconcentration in both euhydrated and hypohydrated (2%) unacclimated subjects during cycling exercise. Diuretic-induced hypohydration of 2.7% body weight significantly decreased blood volume, which in turn resulted in a host of circulatory and thermoregulatory impairment during moderate exercise in the heat, including an increased heart rate, a decreased stroke volume and cardiac output, a decreased cutaneous blood

flow, an elevated cutaneous vasodilatory threshold, and an increased $T_{es}$ (Nadel et al. 1980). Cutaneous circulatory impairment was also observed by Tankersley et al. (1992), with a decreased forearm vascular conductance and forearm blood flow. Sawka et al. (1992a) reported that hypohydration of 8% total body water, or about 5% body weight, significantly decreased plasma volume and contributed to a decreased sweating rate and greater heart rate during exercise in a hot and dry environment. Heart rates were significantly higher at exhaustion during the hypohydration condition despite a greatly decreased endurance time to exhaustion (from 120.7 min to 55.3 min), demonstrating a dramatic increase in cardiovascular strain with hypohydration. In addition, the core temperature at which exhaustion occurred was significantly reduced by hypohydration from 39.1 to 38.7 °C, possibly due to a shorter time allowed for heat buildup or else an alteration in the central integration of temperature inputs. An increased $T_{re}$ and $f_c$ during exercise, along with a decreased sweating rate at a given core temperature, occurred when hypohydrated by 5% body weight during exercise in a hot (49°C) and dry (20% relative humidity) environment (Sawka et al. 1983b).

The magnitude of cardiovascular and thermoregulatory impairment due to hypohydration during exercise in the heat appears to be graded in proportion to the severity of hypohydration. This may be partially due to a progressive impairment of evaporative heat loss ability, with increasing levels of hypohydration both increasing the core temperature threshold for the initiation of sweating and also decreasing the sensitivity of the sweating response (Montain et al. 1995, Sawka et al. 1985). Strydom and Holdsworth (1968) observed increased core temperatures in two miners at high (5-8% body weight) versus low (3-5%) levels of hypohydration. Sawka et al. (1985) systematically compared the effects of hypohydration levels of 0, 3, 5, and 7% body weight during exercise in the heat. Plasma volume prior to and during exercise was progressively decreased at 3 and 5% hypohydration compared with euhydration, with an apparent plasma volume defence beyond 5% resulting in no further decrease in plasma

volume at 7% hypohydration. Comparing the four hydration levels, core temperature linearly increased by approximately 0.15°C and heart rate increased 4 beats · $min^{-1}$ with each percentage decrease in body weight. The increased core temperature and heat storage appear to be largely mediated by an impaired heat loss capacity, as the sweating rate at a given core temperature also decreased for each increasing level of hypohydration. As a result of the increased cardiovascular and thermoregulatory strain, subject exhaustion began occurring before the completion of the exercise sessions at 5 and 7% hypohydration.

### *2.2.4. Mechanisms of Action*

In addition to those factors discussed above, several additional factors may influence the physiological effects of dehydration or hypohydration during exercise in the heat. The mode of dehydration produces different responses within the body fluid compartments. Dehydration induced by either exercise or by passive heat exposure resulted in a similar magnitude of decrease in plasma volume but with different time courses (Costill and Fink 1974). A rapid initial drop in plasma volume with the initiation of exercise was followed by a minor decrease over the remaining period. In contrast, passive thermal dehydration featured a minor initial plasma volume decrease and a continued steady decrease throughout the remaining exposure. Decreases in plasma volume during exercise were due to both overall water loss through sweating and changes in the Starling force accompanying the increased perfusion pressure and capillary area (Senay and Pivarnik 1985).

The method used to achieve hypohydration may also have different effects on the body fluid compartments and the physiological responses of the body. Mild hypohydration of 3.5% body weight induced by active exercise did not significantly affect plasma volume, although plasma osmolality increased due to the secretion of hypotonic sweat (Caldwell et al. 1984). In the same study, sauna-induced hypohydration also resulted in a hyperosmotic

hypovolemia, while diuretics produced an isosmotic hypovolemia (Caldwell et al. 1984). All three modes of hypohydration resulted in a decrease in maximal work capacity, though hypohydration via exercise induced less impairment (Caldwell et al. 1984). However, the slight but significantly lower magnitude of weight loss and the longer time allowed for dehydration with exercise may have contributed to the differences in performance.

Either a decrease in blood volume or an increase in osmolality can effect changes in the cardiovascular and thermoregulatory response to exercise. A progressive decrease in plasma volume during exercise in the heat resulted in an increased $f_c$ and $T_{es}$ independent of changes in osmolality (Fortney et al. 1988). Following a drop in blood volume, the competition for blood flow between metabolic and thermoregulatory demands during exercise was increased (Rowell 1974). One compensation to maintain central circulation and cardiac filling pressure is a reduction in cutaneous circulation, and hence, in heat transfer from the core to the periphery. The ability for dry heat exchange appears to be especially sensitive to blood volume changes (Johnson 1987, Johnson and Park 1979, Nadel et al. 1980, Tankersley et al. 1992). The core temperature threshold at which vasodilation and maximal SkBF occurred was decreased following isosmotic hypovolemia induced by diuretics (Nadel et al. 1980). In contrast, hyperosmotic hypovolemia resulted in an impairment in the sensitivity of the SkBF response to increasing core temperature and a lower maximum SkBF compared with hyperosmotic isovolemia (Fortney et al. 1984). In addition to decreasing cutaneous blood flow, a drop in blood volume alone also impaired the sensitivity of the sweating response, though no differences were observed in the core temperature threshold for sweating (Fortney et al. 1981).

Plasma osmolality increases with higher levels of hypohydration, and can rise from a baseline value of approximately 283 $mosm \cdot kg^{-1}$ to in excess of 300 $mosm \cdot kg^{-1}$ with 7% hypohydration (Sawka et al. 1985). Hyperosmolality, either in a state of isovolemia or in conjunction with a decrease in plasma volume, increased the core temperature threshold for the initiation of vasodilation and sweating (Fortney et al. 1984). Some studies report

that a decrease in sweating rates and a rise in core temperature during exercise were primarily related to changes in plasma osmolality, especially due to alterations in [$Na^+$] (Nielsen 1974a, Nielsen 1974b, Senay 1968). However, while it is tempting to attribute physiological responses to a single variable, it is much more likely that changes in blood volume and osmolality produce overlapping effects and a synergistic response (Sawka et al. 1989).

Some of the thermosensitive neurons in the preoptic anterior hypothalamus are also sensitive to changes in osmolality, suggesting that hypohydration may influence thermoregulation centrally (Silva and Boulant 1984). One possible alteration is an adjustment of the thermoregulatory setpoint and the resting core temperature, contributing to observed differences in thermal responses to exercise. For example, even if the rate of heat storage were similar, a higher initial $T_c$ with hypohydration would result in a higher overall $T_c$. Unfortunately, however, the effect of hydration status on resting core temperature has rarely been detailed. Candas et al. (1988) reported no difference in resting $T_{re}$ following 1.8% hypohydration induced by diuretics. No direct mention was made regarding resting $T_{re}$ in a study investigating the effects of 5% hypohydration (Sawka et al. 1992a). However, calculating backwards from the final $T_{re}$ and $\Delta T_{re}$ values given, the resting $T_{re}$ would be 36.9 and 37.1°C for euhydration and hypohydration, respectively. This difference may be sufficient to contribute to overall differences in $T_{re}$ responses or tolerance times.

The effects of hypohydration on muscle energetics and metabolism in the heat are unclear and may contribute to the impaired performance with hypohydration. No difference was observed in the rate of aerobic or anaerobic metabolism during exercise with varying hydration levels, suggesting no effect of hydration status on metabolic heat production (Greenleaf and Castle 1971, Saltin 1964). During hypohydrated exercise in the heat, however, Sawka et al. (1985) reported that the RER decreased progressively during exercise with increasing levels of hypohydration. The mechanism responsible for the

downward shift is not readily apparent, but it may involve a shift in substrate utilisation towards an increased reliance on lipid metabolism (Holloszy 1973). It is possible that the stress of heat exposure may interact with hypohydration to produce the downward shift in RER, as a significant decrease in RER during exercise in a hot compared with a thermoneutral environment was noted by Young et al. (1985). However, a recent study presented conflicting data, observing hyperglycemia and an increased RER during exercise in the heat, brought about by an elevation in plasma cortisol and catecholamines and an increased hepatic glucose release (Hargreaves et al. 1996). During recovery from 5% hypohydration, no difference was evident in the rate at which muscles resynthesised glycogen whether subjects remained hypohydrated or were rehydrated back to normal body weight overnight (Neufer et al. 1991). However, glycogen levels were still significantly below baseline values the following day with either recovery program, such that strenuous exercise the day before can result in a state of impaired energy balance regardless of recovery program. During 60 min of cycling at 51% $\dot{V}O_2$max in a thermoneutral environment, no difference was reported in the RER or the rate of muscle glycogen utilisation (Neufer et al. 1989c).

## 2.3. Physiological Manipulations

Maintenance of a heightened sweating response, a lowered heart rate, and a lowered internal body temperature during exercise in the heat constitute the classic description of an individual who is adapted to hot environments (Nadel et al. 1974, Rowell 1974, Wyndham 1973). Previous exposure to a hot environment is arguably the most specific and direct method of adaptation to exercise in the heat (Wenger 1988). Two terms often used interchangeably to describe the process of adapting an individual to hot environments through prior exposure to heat are heat acclimatisation and heat acclimation. Heat acclimation refers to adaptive changes resulting from exposures in a controlled laboratory setting. In contrast, heat acclimatisation refers to changes produced within the

natural environment; for example, through a change of season or place of residence. The present work will focus primarily on heat acclimation.

A high level of aerobic fitness has also been associated with an improved exercise-heat tolerance, and physical training is another treatment that has been used in an attempt to adapt individuals to work in a hot environment (Armstrong and Pandolf 1988). Controversy exists regarding the specificity of adaptations to exercise in the heat and whether physical training or fitness are adequate substitutes for actual heat exposure. Questions also arise about the effectiveness of physiological treatments in improving exercise-heat tolerance when wearing protective clothing, due to the attenuation of evaporative heat loss imposed by the limited permeability to water vapour of the clothing. The following sections will briefly review the mechanisms behind the various manipulations and summarise their effects on exercise and heat tolerance.

### *2.3.1. Heat Acclimation*

Empirical evidence supporting the efficacy of heat acclimatisation was provided in a long-term clinical study on Marine Corps recruits. Despite a hotter average weather pattern in September, the incidence of exertional heat illnesses was significantly lower than at the start of the hot season in May, when the recruits were unacclimatised to the heat (Kark et al. 1996). Variations in anthropometric characteristics, fitness, and acclimatisation status amongst individuals were calculated to account for a significant portion of the variance in heat storage not explained by differences in metabolic rate and environmental conditions (Havenith et al. 1995, Havenith and van Middendorp 1990). Even a short acclimation period appears to be sufficient for significant reductions in subsequent physiological strain, with major adaptations occurring during the first few days and almost complete adaptation being achieved within 14 days (Armstrong and Maresh 1991, Pandolf et al. 1977, Senay et al. 1976). Heat acclimation may also be a transient phenomenon requiring constant heat exposures to maintain the adaptations, as no

acclimation effects were observed when heat exposures were separated by one week (Barnett and Maughan 1993). Once heat acclimation is achieved, the rate of decay of the adaptations is slow, occurring over several weeks, and the reinduction of heat acclimation can be achieved rapidly (Pandolf et al. 1977).

One adaptation that accompanies heat acclimation is an alteration in the behavioural drinking response. With repeated exercise-heat exposures in subjects given water *ad libitum*, Greenleaf et al. (1983) reported a slight decrease in the magnitude of negative water balance with continued exposures due to an increased total voluntary fluid consumption. This increased consumption was characterised by a decreased time to the first drink, an increased drinking frequency, and an increased mean volume per drink.

Another central adaptation to heat acclimation is an alteration in the basal thermoregulatory setpoint, with a lowering of the resting core temperature reported following heat acclimation programs (Aoyagi et al. 1994, 1995, 1973b, Shvartz et al. 1973c, 1977). If a critical core temperature determines tolerance to exercise-heat stress (Nielsen 1992, 1994), then a lower initial core temperature would permit a greater temperature rise before reaching that threshold. Though the physiological mechanism is unclear, heat acclimation may improve the efficiency of exercise by decreasing the rate of metabolism, and thereby the rate of internal heat production (Houmard et al. 1990, Sawka et al. 1983a).

As discussed previously, hydration status can significantly influence cardiovascular and temperature regulation during exercise in the heat. One common response to a heat acclimation program is an increase in the total blood volume (Bonner et al. 1976, Harrison 1985, Harrison et al. 1981, Senay and Kok 1976, Wyndham et al. 1968) which could contribute to a decrease in cardiovascular and thermoregulatory strain during exercise (Armstrong and Maresh 1991, Harrison 1986). Blood volume expansion may attenuate the competition between metabolic and thermoregulatory demands for blood flow (Rowell 1974). In turn, a decrease in competition would have the effect of increasing cutaneous

blood flow, promoting convective heat transfer and fluid delivery to the sweat glands and contributing to an increased sweating rate (Fox et al. 1963, Nadel et al. 1974). One contributing factor in the expansion of blood volume is an increased translocation of proteins into the intravascular space from the interstitial fluid compartment, resulting in an elevation in the intravascular oncotic pressure and fluid influx from the intestitial space (Bonner et al. 1976, Harrison et al. 1981). Hypervolemia would also be promoted by the increase in fluid-retention hormones, such as aldosterone and vasopressin (Francesconi 1988).

Plasma volume expansion occurs rapidly with the initiation of heat acclimation. Senay et al. (1976) found that the major changes in plasma dynamics occur during the first 6 days of exposure. Over ten days of exercise-heat exposure, an initial rapid increase in plasma volume and a subsequent progressive increase was strongly linked to increases in intravascular protein content (Senay et al. 1976). The magnitude of the increase is highly variable, however, and dependent on the nature of the heat acclimation program (Harrison 1985). The permanence of blood volume expansion with heat acclimation is unclear, however, and it has been suggested that hypervolemia may be a transient effect of heat acclimation, with the plasma volume decaying back to baseline levels and being replaced by other adaptations, such as an increased sweat rate (Wenger 1988).

Heat acclimation produces a series of adaptations in the sweating response that serve to increase the rate of evaporative heat dissipation. An increased sweating rate with heat acclimation is partly mediated at the central thermoregulatory integrators, with a reduction in the core temperature threshold for the initiation of the sweating response (Nadel et al. 1974, Roberts et al. 1977). The nature of the secreted sweat may also change with heat acclimation. A strong inverse correlation was observed by Nose et al. (1988) between the loss of free water and the $[Na^+]$ in sweat. Therefore, one adaptation resulting from heat acclimation is the production of a more dilute sweat (Allan and Wilson 1971).

Heat acclimation may also bring about an increased sweating response through peripheral mechanisms. A high rate of skin wettedness will normally result in a saturation of the sweat gland and suppression of gland secretion through a process termed hidromeiosis (Candas et al. 1980). Repeated heat exposures featuring high sweating rates and skin wettedness may produce adaptations at the level of the sweat glands, minimising hidromeiosis by allowing the glands to maintain high sweat outputs despite high skin wettedness (Candas et al. 1979, Taylor 1986). Sudomotor adaptations may include a widening of the duct orifice or increased secretory pressure (Ogawa et al. 1982), sweat gland hypertrophy (Sato and Sato 1983), or improved efficiency at the sweat gland (Collins et al. 1966). Alternatively, heat acclimation may train an individual to tolerate the psychological discomfort associated with high levels of skin wettedness (Candas et al. 1979). Shvartz and Benor (1971) and Shvartz et al. (1972, 1973c) found that significant increases in sweat rates and heat tolerance in a hot-dry environment can be achieved following heat acclimation using vapour-barrier suits which do not allow evaporative heat transfer; they concluded that the prevention of evaporative heat loss was a strong stimulus for sweat production and a major factor in heat acclimation. The sweating response to local electrical stimulation was significantly increased following whole-body heat acclimation (Chen and Elizondo 1974). This appears to be an integrated whole-body response to repeated heat exposure, as neither local stimulation by itself nor local heating resulted in an increased sweat output during subsequent heat exposure (Chen and Elizondo 1974).

Heat acclimation can be achieved by a variety of heat exposure protocols. In addition to the standard training variables of frequency, duration, and intensity, the environment is also a significant component of a heat acclimation program. The ideal heat acclimation program is likely specific to both the environment that will be encountered as well as the clothing, if any, which will be worn. Shvartz et al. (1973b) acclimated subjects to either unclothed exercise in a hot-dry environment or else a hot-wet environment, using

vapour barrier clothing at a slightly cooler temperature to achieve a similar WBGT. During subsequent hot-dry exercise without clothing, though both groups demonstrated an improvement, the physiological strain was significantly lower in the hot-dry compared to the hot-wet acclimation group.

A lack of transfer of hot-dry heat acclimation adaptations to a hot-wet environment appears to be true also, possibly due to the severe microenvironment and limited heat dissipation limiting the total adaptation possible. Hot-dry heat acclimation did not prolong tolerance for individuals resting in a hot-wet environment (Goldman et al. 1965). Aoyagi et al. (1995) compared the benefits of extending heat acclimation, achieved through unclothed exercise in a hot environment, from 6 to 12 days on subsequent exercise in the heat. Six days of heat acclimation significantly reduced physiological strain while wearing either the combat clothing or the NBC ensemble. However, the 12 days of acclimation was of additional benefit only while wearing combat clothing. Acclimation using NBC clothing appears to be required for optimal adaptation to subsequent work in the NBC ensemble. Both hot-dry or hot-wet acclimation, accomplished by wearing either only combat clothing or the full NBC ensemble, respectively, were able to decrease heart rate and core and skin temperature during subsequent exercise in the heat with combat clothing. However, acclimation with the NBC ensemble produced greater decreases in physiological strain during exercise in the hot-wet microenvironment of the full NBC ensemble compared to acclimation with only the combat clothing (McLellan and Aoyagi 1996). The hot-wet acclimation may have produced a stronger stimulus for sweat production and decreased sweat suppression despite high levels of skin wettedness (Fox et al. 1967).

Individual variations in responses to heat acclimation are evident, with some individuals appearing to be unresponsive to repeated bouts of heat exposure. For example, during occupational screening, some South African mine workers experienced no decrease in physiological strain despite repeated heat exposures and had to be assigned to surface

jobs (Senay and Kok 1976). One major distinguishing characteristic of these heat-intolerant individuals was a significantly lower plasma volume during exercise, likely due to a decreased protein influx into the intravascular space (Senay and Kok 1976). An expansion of plasma volume has been proposed to be the critical event in heat acclimation (Senay et al. 1976). A synergy between fitness and heat acclimation appears to exist, with higher aerobic fitness resulting in an improved and more rapid response to heat acclimation (Armstrong and Maresh 1991, Piwonka and Robinson 1967). Pandolf et al. (1977) reported a significant inverse relationship between an individual's $\dot{V}O_2$ max and the days required to achieve a plateau in physiological response to heat acclimation. In addition, fit subjects had a slower decay and a more rapid reinduction of heat acclimation (Armstrong and Maresh 1991). Heat acclimation status may mediate the effect of other factors on exercise-heat tolerance. Sawka et al. (1983b) examined the effects of hypohydration on exercise in a variety of environments pre- and post-acclimation, achieved through exercise in the heat. In a thermoneutral environment, heat acclimation decreased the thermoregulatory and cardiovascular strain experienced during hypohydration. However, in a hot environment, only cardiovascular strain was reduced.

It should be noted that an improvement in exercise-heat tolerance following heat acclimation is not a universal finding. For example, Piwonka and Robinson (1967) reported no further improvement in exercise-heat tolerance in four highly trained subjects following heat acclimation. However, the small sample size, the relatively low heat stress environment, and the gap of up to two weeks between the last acclimation session and the heat stress test, during which significant decay of heat adaptations may have occurred, could all have been factors contributing against seeing a significant effect. Aoyagi et al. (1994) reported that heat acclimation had no effect when NBC protective clothing was worn. However, as discussed previously, the limited evaporative heat loss and high metabolic rate may have negated the effects of heat acclimation.

### *2.3.2. Aerobic Fitness*

A high level of cardiorespiratory fitness has been associated with an improved exercise-heat tolerance since the initial theoretical connection was made by Robinson et al. (1943) and Bean and Eichna (1943). These suggestions were based largely on anecdotal evidence, but have been largely supported by subsequent studies (Wenger 1988). Piwonka and Robinson (1965) reported that trained distance runners exhibited a decreased physiological strain compared to untrained individuals during exercise-heat stress. Havenith and van Middendorp (1990) reported that inter-individual variations in aerobic fitness and anthropometric measures could account for a significant portion of the variance in heat storage not explained by differences in metabolic rate and environmental conditions. In either temperate, hot-dry (Cadarette et al. 1984, Shvartz et al. 1977), or hot-wet environments (Havenith et al. 1995), $\dot{V}O_2$ max was significantly and inversely correlated with core temperature and heart rate. Henane et al. (1977) reported that trained and fit ( $\dot{V}O_2$ max ~65 $mL \cdot kg^{-1} \cdot min^{-1}$ ) athletes had an improved sweating response and exercise-heat tolerance compared to sedentary and unfit ( $\dot{V}O_2$ max ~40 $mL \cdot kg^{-1} \cdot min^{-1}$ ) individuals. Other studies, however, report only a minimal relationship between $\dot{V}O_2$ max and exercise-heat tolerance (Kielblock 1984, Pandolf et al. 1988).

Aerobic fitness level also appears to mediate the impact of other environmental and individual characteristics. As discussed above, high aerobic fitness potentiates the sensitivity to heat acclimation and also delays the time course of its decay (Pandolf et al. 1977). In contrast, heat acclimation may mimic the thermoregulatory benefits of a higher $\dot{V}O_2$ max, though cardiovascular benefits remain (Cadarette et al. 1984). The magnitude of adaptations to heat acclimation may also be inversely related to the initial level of fitness, with a trend towards greater decreases in heart rate and core temperature in those with a lower initial $\dot{V}O_2$ max (Cadarette et al. 1984). In addition, an increase in aerobic fitness appears to provide some protection against the stress of hypohydration during

exercise in the heat. When the same subjects were exposed to 5% hypohydration, the fitter subjects exhibited a lower heart rate, though no difference was noted in $T_{re}$ (Cadarette et al. 1984).

The benefit of high aerobic fitness during clothed exercise in the heat is unclear. Smolander et al. (1987) reported no difference in cardiovascular or thermoregulatory strain in subjects with a high (60 $mL \cdot kg^{-1} \cdot min^{-1}$), compared to a low (40 $mL \cdot kg^{-1} \cdot min^{-1}$), $\dot{V}O_2$ max while wearing light industrial clothing which permitted evaporative heat transfer. However, all subjects exercised at 30% $\dot{V}O_2$ max, and relative exercise intensity may be the primary determinant of the heart rate and core temperature response (Astrand and Rhyming 1954, Saltin and Hermansen 1966). While wearing NBC protective clothing at a standardised absolute exercise intensity, subjects with a high $\dot{V}O_2$ max relative to lean body mass (75 $mL \cdot kg^{-1} \cdot min^{-1}$ LBM) had a lower heart rate, higher sweating rate, and a trend towards longer tolerance times compared to subjects of moderate (60 $mL \cdot kg^{-1} \cdot min^{-1}$ LBM) fitness (Windle and Davies 1996). No difference was observed in core temperature, however, possibly due to the lack of evaporative heat transfer through the clothing, which resulted in similar rates of heat storage.

Caution must be employed when attributing improvements in exercise-heat tolerance solely to aerobic fitness. An increase in $\dot{V}O_2$ max is an indirect result of the physiological adaptations to training, such as changes in blood volume, cardiac function, and muscle metabolism. Therefore, $\dot{V}O_2$ max by itself may not be a reliable indicator of exercise-heat tolerance (Armstrong and Pandolf 1988, Kielblock 1984, Pandolf et al. 1988). The mode of training used during exercise also appears to influence the responses to exercise-heat stress. While highly trained and fit ($\dot{V}O_2$ max ~65 $mL \cdot kg^{-1} \cdot min^{-1}$) nordic skiers and swimmers both had higher sweat outputs than sedentary and unfit ($\dot{V}O_2$ max = 40 $mL \cdot kg^{-1} \cdot min^{-1}$) individuals, the sweating response and exercise-heat tolerance was significantly higher in the skiers compared to the swimmers (Henane et al.

1977). The authors suggested that the greater exercise-induced hyperthermia in the skiers was responsible for the disparity in the observed adaptations.

### *2.3.3. Physical Training*

Many of the physiological adaptations reported with heat acclimation are also observed following aerobic training or in comparisons between individuals with high versus low cardiorespiratory fitness, leading to the suggestion that physical training may be an adequate replacement for heat acclimation (Armstrong and Pandolf 1988, Pandolf 1979). If true, this would be of great interest in many occupational settings, where a temperate ambient environment or lack of facilities may preclude proper heat acclimation. However, aerobic training may differ from heat acclimation both in the nature and also in the magnitude of the adaptive responses, and the compatibility of these physiological treatments remains an active field of research.

Gisolfi and Robinson (1969) were among the first to report a reduction in physiological strain and an improvement in exercise tolerance in a hot environment following a relatively long-term (6 weeks) interval training program. Four weeks of interval training also produced significant improvements in exercise-heat tolerance, with the improvement reaching a plateau after 8 or more weeks of training of approximately 50% of the adaptive responses brought about by heat acclimation (Gisolfi 1973).

The effects of shorter or less intense training programs on exercise-heat tolerance are less clear. Shvartz et al. (1973b) reported that 6 d of training resulted in no adaptations to exercise-heat stress, whereas 6 d of heat acclimation significantly decreased heart rate and core temperature, suggesting also that physical training is not a substitute for heat acclimation. Supporting this contention are studies by Strydom et al. (1969, 1966) which reported minimal adaptations to exercise-heat stress following two weeks of physical training in a cool environment compared to heat-acclimated subjects. In contrast, two weeks of cycling at 65% $\dot{V}O_2$ max produced a significant though slight increase in

$\dot{V}O_2$ max and was sufficient to elicit an attenuation in cardiovascular and thermal strain during submaximal exercise in a thermoneutral environment (Green et al. 1991). Exercise-heat tolerance was not the focus of this study, and the subjects were not tested in hot environments. Houmard et al. (1990) compared the efficacy of 7 d of training at either 75% $\dot{V}O_2$ max for 30 min or 50% $\dot{V}O_2$ max for 60 min on subsequent exercise-heat stress. Compared with pre-training responses, both training programs produced equivalent and significant decreases in final heart rate and in resting and final core temperature during exercise at 50% $\dot{V}O_2$ max in 40°C.

The effectiveness of a training program in improving exercise-heat tolerance is dependent on many variables, including the intensity, duration, and frequency of the training. Henane et al. (1977) suggested that a 15-20% increase in $\dot{V}O_2$ max is required in order to improve exercise-heat tolerance. Metabolic hyperthermia is the major stimulus for thermoregulatory adaptations (Fox et al. 1963), and the degree and duration of core temperature rise induced by the training may be a determinant of the degree of heat acclimation. A greater sweating response and heat tolerance was noted following land-based, compared to water-based, training on a group of previously untrained subjects, with the disparity being attributed to the higher level of metabolic hyperthermia and sweating rates during land-based exercise (Avellini et al. 1982). Kondo et al. (1996) also reported a higher sweating rate in land-based, compared to water-based, athletes. In addition, individual variables, such as genetics, age, and the starting fitness of the subjects would play a role in the final response to training (Pandolf 1979). Havenith (1995) has proposed that thermoregulatory improvements in relatively unfit subjects would be greater than in fit subjects following an equivalent improvement in absolute aerobic fitness.

### *2.3.4. Mechanisms of Action*

Taylor (1986) reviewed the influence of aerobic training on sweating responses, and concluded that training was capable of stimulating both central and peripheral

adaptations in the sweating responses. Roberts et al. (1977) reported that aerobic training decreased the core temperature threshold for the initiation of sweating, suggesting a central resetting of the sweating response. In addition, both Nadel et al. (1974) and Henane et al. (1977) reported an increase in the sensitivity of the sweating rate to increasing core temperature following ten days of training at 70-80% $\dot{V}O_2$ max for 1 h, suggesting a peripheral adaptation to an increase in thermal strain. Other peripheral adaptations at the level of the sweat glands that may result from training were discussed in Section 2.3.1.

An increase in total blood volume, primarily through an increase in plasma volume, has been proposed to have a beneficial effect on cardiovascular and thermal homeostasis during exercise in the heat (Convertino 1991, Harrison 1985). In theory, by increasing the total circulating volume, cardiac filling pressure is maintained and competition between metabolic and thermoregulatory demands for blood flow are lessened. In addition, skin blood flow is maintained to allow for adequate heat transfer. Maintenance of blood volume also decreases thermoregulatory strain by optimising evaporative heat loss through maintaining adequate blood flow to the sweat glands for sweat production (Convertino 1991, Rowell 1974). Thermoregulatory and cardiovascular adaptations have correlated highly with increases in plasma volume (Senay et al. 1976). Endurance-trained subjects typically exhibit a significantly larger blood volume than untrained subjects (Convertino 1991), though plasma volume expansion alone did not increase $\dot{V}O_2$ max in trained male subjects (Coyle et al. 1986, Mier et al. 1996). A blood volume 20-25% larger than in untrained subjects was observed in both males and females independent of age (Brotherhood et al. 1975, Dill et al. 1974, Kjellberg et al. 1949), and a high correlation existed between aerobic capacity and blood volume (Convertino 1991). However, the correlation disappeared when the vascular volumes were normalised with lean body mass, and may have been due to a high covariance between fitness and lean body mass (Sawka et al. 1992b).

A marked increase in total blood volume is a rapid response to the onset of exercise, and hypervolemia is one of the most consistent adaptations to short-term training (Convertino 1991, Harrison 1985). Plasma volume expansion in the range of 10-20 % was observed following a training program of two weeks (Green et al. 1991), three days (Green et al. 1990, Green et al. 1984), and even after a single extended or supramaximal exercise session (Gillen et al. 1991, Pugh 1969). Training appears to stimulate an iso-osmotic hypervolemia with minimal increases in plasma osmolality due to a translocation of protein into the intravascular space (Convertino et al. 1980a, 1980b). Increases in plasma renin activity and vasopressin concentration during exercise post-training also facilitated retention of sodium and water (Convertino et al. 1980a).

Control of the body fluid compartments appear to be tightly regulated, and periodic exercise needs to be performed in order to maintain hypervolemia. In highly trained subjects, detraining resulted in a reduction in total blood volume within two to four weeks (Coyle et al. 1986). In addition, two months of intensive exercise failed to induce a significant hypervolemic response in fit athletes (Frick et al. 1970), and 11 weeks of aerobic training did not increase either red cell volume or plasma volume in untrained subjects (Shoemaker et al. 1996). This suggests that an initial hypervolemic response may be only a transient response to the onset of exercise, and that the process of a maintained enlarged blood volume in trained subjects may occur by mechanisms different from those seen with short-term training. Convertino (1991) proposes that training-induced hypervolemia consists of two phases. In the first phase, an influx of protein into the intravascular space increases the oncotic pressure, leading to fluid retention and a transient plasma volume expansion. With prolonged training of several weeks or months, changes in neurohormonal and renal mechanisms, specifically an enhancement of sodium reabsorption, bring about an increase in total body water and a chronic enlargement of total blood volume.

### *2.3.5. Interactions*

From the above discussion, it is evident that the various physiological manipulations to improve exercise-heat tolerance overlap each other to some extent in their mechanisms of actions and their ultimate effects on the individual. Similar adaptations occur with exercise training or heat acclimation, making it difficult to isolate the individual effects of training versus thermal stimuli. Furthermore, one manipulation may mask or negate the effects of another. Metabolic heat production and an increase in body heat storage and $T_{re}$ are inherent during aerobic exercise training, further complicating any isolation of factors. The separate roles of thermal versus nonthermal stimuli in the hypervolemic response were investigated by Convertino et al. (1980b). Similar thermal stresses were placed on two groups of subjects using either exercise at room temperature or sedentary heat exposure. Thermal factors were found to account for 40% of the plasma volume expansion, with the remaining hypervolemia due to additional non-thermal factors related to exercise. Green et al. (1984) used three days of intermittent supramaximal exercise in an attempt to maximise exercise stress while minimising the confounding influence of thermal stimuli. Total blood volume and plasma volume both increased significantly following three days of exercise. Surprisingly, red cell volume was found to decrease, a response atypical of short-term training programs (Convertino 1991, Harrison 1985). The authors suggested that this alteration in the blood cell population may be due to the particular characteristics of the training program, which produced a protracted state of metabolic acidosis. Despite the aim of minimising thermal factors, significant thermal stress was still present with this training program, with $T_{re}$ increasing by an average 1.3 °C over the course of each exercise session.

## 2.4. Statement of the Problem

While the human physiological responses to heat exposure are well documented, a smaller body of research has focused on the interactions between individual

characteristics, such as hydration status and aerobic fitness, along with physiological interventions, such as fluid replacement, aerobic training, and heat acclimation. It is difficult to integrate the available research into a coherent whole, due to differences between studies in factors that could influence the responses to exercise-heat stress, including variations in subject populations, test environments, training or acclimation programs, the degree of hypohydration, and the rate and nature of fluid replacement. An integrated research design, involving a series of related experiments with a common methodology, would enable a full analysis of the interactions between these individual characteristics and the various physiological manipulations. There is also little research conducted on the effects of these factors when subjects are wearing protective clothing ensembles with a limited permeability to water vapour. The microenvironment created within protective clothing ensembles may significantly influence the impact of individual characteristics and physiological interventions on responses to exercise-heat stress. Therefore, in order to enhance the protection and performance of workers in an industrial setting or military personnel, a better understanding is required concerning the interactions between fitness, heat acclimation, fluid replacement, and hydration status while wearing protective clothing in the heat.

# 3. OBJECTIVES AND HYPOTHESES

With the preceding review as a background, a series of three experiments was designed with the following objectives:

1. to clarify the influence of hydration status on exercise in an uncompensable heat stress environment. Specifically, the experimental aim was to determine the effect of a minor (2-2.5% body mass) hypohydration on tolerance in an uncompensable heat stress environment during both light and heavy intensity exercise. A second experimental aim was to determine the efficacy of a fluid replacement program during uncompensable heat stress. It was hypothesised that hydration status would have a major impact on heat tolerance while wearing the protective clothing only during light exercise, where longer tolerance times would permit differences in heat dissipation to be expressed. It was further hypothesised that fluid replacement in an uncompensable heat stress environment would be of minimal benefit during heavy intensity exercise, due to the rapid onset of exhaustion from other factors compared with the delay required between the intake of fluid and its absorption into the body (Section 5.1).
2. to investigate the influence of a short-term aerobic training program and hydration status on the response to exercise in an uncompensable heat stress environment. It was hypothesised that training would result in an improved physiological response in both an euhydrated and hypohydrated state, and that the magnitude of improvement would be greater during the hypohydrated exercise. Exercise-heat tolerance was hypothesised to be significantly impaired by minor hypohydration (2-2.5% body mass), regardless of training status (Section 5.2).
3. to determine the influence of a heat acclimation program and hydration status on subsequent exercise in an uncompensable heat stress environment. It was hypothesised that heat acclimation would result in an improved physiological response in both an euhydrated and hypohydrated state, and that the magnitude of improvement would be greater during the hypohydrated exercise. Exercise-heat

tolerance was hypothesised to be significantly impaired by minor hypohydration (2-2.5% body mass), regardless of acclimation status (Section 5.3).

4. to compare the efficacy of a short-term aerobic training program to an equivalent period of heat acclimation on exercise in an uncompensable heat stress environment. It was hypothesised that the heat acclimation would produce a greater adaptation to heat stress, and that exercise-heat tolerance would be improved more following heat acclimation (Section 5.3).
5. to determine the relative influence of aerobic fitness on the response to exercise in an uncompensable heat stress before and after a period of heat acclimation. It was hypothesised that subjects with high aerobic fitness would experience less physiological strain and have a prolonged exercise-heat tolerance in both an euhydrated and hypohydrated state. Due to their relative lack of heat adaptation from prior aerobic training, it was further hypothesised that individuals of low to moderate aerobic fitness would possess a greater potential for adaptation to heat acclimation, and that exercise-heat tolerance would be improved more following heat acclimation in aerobically nonfit subjects (Section 5.3, 6.2).
6. to determine the relative influence of a short-term aerobic training program compared to long-term aerobic fitness achieved through habitual exercise on exercise-heat tolerance. It was hypothesised that aerobic fitness derived from prolonged training and habitual exercise would impart physiological benefits that would not be realised with a short period of aerobic training, and that the more fit subjects would experience less physiological strain and have a prolonged exercise-heat tolerance in both an euhydrated and hypohydrated state compared to nonfit subjects who underwent a short-term aerobic training program (Section 6.1).

Table 3.1 Summary of experimental manipulations

| Manipulations | Chapter Sections | | | | |
|---|---|---|---|---|---|
| | 5.1 | 5.2 | 5.3 | 6.1 | 6.2 |
| Hydration status | X | X | X | X | X |
| Fluid replacement | X | | | | |
| Exercise intensity | X | | | | |
| Short-term aerobic training | | X | | X | |
| Heat acclimation | | | X | | |
| Aerobic fitness | | | X | X | X |

# 4. GENERAL METHODS

## 4.1. Subjects

The subjects for this series of studies consisted of healthy males between the ages of 18 to 40, recruited from the university population or the military community. This group was chosen to be representative of the soldiers in the Canadian Forces who might be expected to wear NBC clothing in an operational theatre. All subjects underwent a medical examination and were informed of all details of the experimental procedures and the associated risks and discomforts before they provided their consent.

## 4.2. Experimental Design

The experimental protocols and instrumentation used in the present series of studies were approved by the Ethics Review Committees of the University of Toronto and the Defence and Civil Institute of Environmental Medicine (DCIEM). To limit initial heat acclimation through casual exposure to high ambient temperatures, testing occurred only from late September to early May. In all subjects, the first session was used as a familiarisation trial and the results were discarded. A minimum of 72 h separated experimental trials, with the large majority of trials separated by at least a week to avoid the effects of accumulated heat acclimation over the course of the study (Barnett and Maughan 1993).

The primary objective of the present series of studies was to investigate, in an integrated manner, the effects of hydration status, physical fitness, and heat acclimation on exercise in an uncompensable heat stress environment. In order to achieve an integrated approach, the studies were designed to complement each other and to have similar methodologies in order to allow for comparisons among the studies. The basic heat stress test (HST) performed throughout all studies consisted of continuous walking on a motorised treadmill in a hot (40°C, 30% relative humidity, no wind) environment while wearing the Canadian Forces Nuclear, Biological and Chemical (NBC) protective clothing

ensemble. The HST was performed while the subjects were either in a euhydrated state (EU) or mildly hypohydrated by approximately 2.5% of their body mass (HY).

When Study 1 was originally designed, the level of aerobic fitness was not a selection criterion for subjects, and it was anticipated that the subjects would represent a range of fitness levels. However, by random selection, all subjects for Study 1 were extremely fit and active, resulting in a fairly homogenous subject group. This homogeneity directly affected the designs of Studies 2 and 3. To investigate the influence of long-term fitness and high maximal aerobic power ($\dot{V}O_2$ max), strict fitness criteria for both $\dot{V}O_2$ max and activity patterns were developed in Studies 2 and 3 to create a clear separation. Subjects were grouped into two general categories of either low (LF) or high (HF) fitness based on an interview of their exercise habits and the results of a treadmill test of $\dot{V}O_2$ max. For the purpose of these studies, LF subjects were either inactive at the time of the study or engaged in physical activity only on an irregular basis, and had a $\dot{V}O_2$ max between 40 and 50 $mL \cdot kg^{-1} \cdot min^{-1}$. All LF subjects agreed to abstain from regular aerobic activities for the duration of the experiment. HF subjects were defined as those engaged in a regular program of physical activity and having a $\dot{V}O_2$ max in excess of 55 $mL \cdot kg^{-1} \cdot min^{-1}$. To establish a clear separation in aerobic fitness between the HF and LF groups, volunteers with an initial $\dot{V}O_2$ max between 50-55 $mL \cdot kg^{-1} \cdot min^{-1}$, or those with $\dot{V}O_2$ max > 55 $mL \cdot kg^{-1} \cdot min^{-1}$ but not engaged in regular physical activity, were declined. The separate groups were labeled based on their fitness and the treatment employed as follows: 1) LF-Control: low fit, control treatment; 2) LF-Train: low fit, training treatment; 3) LF-Acc: low fit, heat acclimation treatment; 4) HF-Acc: high fit, heat acclimation treatment.

### 4.3. Methodology

Except where noted, the following methodology was common among Studies 1-3:

### *4.3.1. Maximal Aerobic Power ($\dot{V}O_2$ max)*

$\dot{V}O_2$ max was determined on a motorised treadmill using open-circuit spirometry before the series of HST's in the climatic chamber. For Studies 2 and 3, $\dot{V}O_2$ max was also determined following the training, control, or heat acclimation program. Following 3 min of running at a self-selected pace, the treadmill grade was increased 1% each minute until a grade of 10% was attained. Thereafter, increases in treadmill speed and grade of 0.22 $m \cdot s^{-1}$ (0.8 $km \cdot h^{-1}$) or 2%, respectively, alternated each minute until the subject could no longer continue. Subjects were given verbal encouragement throughout the test. $\dot{V}O_2$ max was defined as the highest 30 s oxygen consumption ($\dot{V}O_2$) observed during the incremental test. The achievement of $\dot{V}O_2$ max was confirmed by supplementary variables, including a heart rate approaching the age-predicted maximum of 220 - age, a respiratory exchange ratio (RER) > 1.15, and a plateau in $\dot{V}O_2$. Heart rate ($f_c$) was monitored throughout the incremental test from a telemetry unit (Polar Vantage XL). The value recorded at the end of the exercise test was considered to be the individual's maximum heart rate.

### *4.3.2. Anthropometry*

Height and body mass were measured on each subject. The body surface area ($A_D$) was calculated from height and body mass using the DuBois equation, and the surface area-to-mass ($A_D$:mass) ratio calculated. Body fatness was estimated from skinfold measurements using a gender-specific regression equation developed from hydrostatic measurements of body density (Forsyth et al. 1984). In Section 6.2, lean body mass (LBM) was calculated by subtracting the calculated mass of body fat from the total body mass, and $\dot{V}O_2$ max was normalised to LBM.

### *4.3.3. NBC Protective Clothing*

The Canadian Forces NBC protective clothing ensemble worn in all trials consisted of shorts, T-shirt, socks, combat shirt and trousers, running shoes, semipermeable NBC

overgarment, impermeable gas mask and cannister, and impermeable rubber gloves and overboots. Total mass of the ensemble was approximately 8.0 kg. In order to allow some sweat evaporation, a limited mass penetration of charcoal-filtered air occurs through the fabric. Thermal resistance and the Woodcock vapour permeability coefficient of the ensemble determined on a heated and wetted manikin at a wind speed of 1.12 $m \cdot s^{-1}$ were 0.291 $m^2 \cdot ^\circ C \cdot W^{-1}$ (1.88 clo) and 0.33, respectively (Gonzalez et al. 1993).

$E_{req}$ with the protective clothing in the experimental environment was determined taking into account the metabolic rate, radiative and conductive heat losses, and respiratory heat loss using the equations of McLellan et al. (1996) and Gonzalez et al. (1993). $E_{max}$ while wearing the protective clothing was determined using the equations of Gonzalez et al. (1993). The equations used are outlined in Chapter 2.1.2. HSI ($E_{req} \cdot E_{max}^{-1}$) was calculated to be approximately 2.5 for the light exercise used during Studies 1-3, and about 3.5 for the heavy exercise employed during Study 1, producing a situation of extreme uncompensable heat stress.

#### *4.3.4. Dehydration and Overnight Protocol*

For each study, identical dehydration and overnight protocols were employed. On the afternoons before each HST session, the subjects reported to the laboratory at ~1330 h and exercised in the heat until they dehydrated by 2.5% of their body mass. The timing of the dehydration allowed approximately 15 h for body fluid compartments to stabilise between the completion of the dehydration and the HST. Dehydration sessions took place in the same environmental chamber (40 °C, 30% relative humidity) as used for the HST. Both nude and dressed mass (shorts, socks, shoes) were recorded prior to entry into the chamber. Subjects walked on a motorized treadmill at an exercise intensity (5-6.5 $km \cdot h^{-1}$, 5-8% grade) that induced sweat loss at a rate of 0.8-1.5 $L \cdot h^{-1}$. Rectal temperature ($T_{re}$) and body mass were monitored throughout the dehydration, and subjects were removed from the chamber upon losing 2.5% of their baseline body mass.

In the evening following the dehydration, the nutritional intake was controlled by providing the subjects with a set meal plan consisting of PowerBar® meal replacement bars (940 $kJ \cdot bar^{-1}$). Gatorade® (1100 $kJ \cdot L^{-1}$, 18.4 $mmol \cdot L^{-1}$ $Na^+$) was used for fluid replacement following the dehydration session. For subjects undergoing EU trials, sufficient Gatorade® was provided immediately following the dehydration session to replace the amount of mass lost during the session. The [$Na^+$] of Gatorade® is at the lower range of [$Na^+$] in sweat, and replacement of 100% of the body mass lost following 2% dehydration using a drink of similar [$Na^+$] resulted in a net fluid balance of -528 mL 6 h post-exercise (Shirreffs et al. 1996). The subjects were therefore instructed to drink a minimum of 600 $mL \cdot h^{-1}$ of Gatorade® or juices that evening prior to going to bed, and at least 600 mL in the morning prior to reporting to the laboratory, for an additional rehydration volume of approximately 3 L. This recovery program was generally sufficient to return the subject to basal body mass the next morning prior to the HST. In the rare (2-3) cases where the body mass did not return to within 1% of the baseline overnight, the morning trial was aborted and rescheduled. Subjects undergoing the HY trials were given a total ration of 800 mL of Gatorade®, based on expected basal body mass losses over a 15 h period. In the rare (2-3) cases where the subject were not hypohydrated by at least 1.5% the following morning, the trial was aborted and rescheduled. However, in 3 additional cases where the subjects were not hypohydrated by at least 1.5% body mass, but were unable to repeat the hypohydration procedure, the HST was performed. These subjects were hypohydrated by at least 0.9% body mass.

#### *4.3.5. Dressing and Weighing Procedure*

Upon arrival at the laboratory, the subjects first inserted a rectal thermistor approximately 0.15 m beyond the anal sphincter. Prior to the dressing procedure, the subjects remained in an upright posture for 10 min, whereupon a 5 mL blood sample was obtained within 90 s of lying down to obtain samples representative of upright exercise

(Lundvall and Bjerkhoel 1994). They were then weighed nude on an electronic scale sensitive to the nearest 0.005 kg (Setra Super Count). For $f_c$ measurements, the telemetry unit was clipped to ECG spot electrodes on the chest; the receiver (Polar Vantage XL) was taped to the outside of the NBC clothing and displayed continuously for the duration of each HST. Bioelectrical impedance band electrodes were placed around the neck and torso for the determination of stroke volume and cardiac output, and thermistors for the determination of the mean skin temperature ( $\overline{T}_{sk}$ ) were applied. Subjects then dressed in their NBC ensemble, and a full dressed mass was obtained before entry into the climatic chamber.

Upon entering the chamber, the subject's skin and rectal thermistor monitoring cables were connected to a computerised data acquisition system and the exercise began. Mean values over 1-min periods for $T_{re}$ and $\overline{T}_{sk}$ were calculated, recorded, and printed by the data acquisition system. $f_c$ was recorded every 5 min from the Polar Vantage XL unit. After the completion of each trial, dressed mass was recorded within 1 min after exit from the chamber; nude mass was recorded following a 5-min undressing procedure.

Differences in nude and dressed mass before and after each trial were corrected for respiratory and metabolic weight losses (see below). The amount of sweat produced was calculated as pre-trial minus post-trial nude mass (corrected) plus water given. Evaporative sweat loss from the clothing was calculated as pre-trial minus post-trial dressed mass (corrected) plus water given. Inaccuracy in measurement due to sweat drippage through the mask and mouthpiece was assumed to be minor, with $< 10$ g collected through the mouthpiece in pilot trials. Unfortunately, the calculation of whole body sweating rates provides no information about the pattern of sweating throughout a given trial, nor about the threshold and sensitivity of the sweating response.

### *4.3.6. Blood Analyses*

Plasma osmolality was determined in duplicate by freezing point depression (Osmette A, Fisher Scientific) in Study 1 and by calculation from [$Na^+$], [Glucose], and [blood urea nitrogen] in Studies 2 and 3 (Novastat, Nova Biochemical). Hemoglobin concentrations were determined in duplicate by photometry (HemoCue, Hemocue AB, Helsingborg, Sweden) and hematocrit in triplicate by microcentrifugation. Hematocrit (Hct) values were adjusted for blood cell packing and for arterial-venous difference by correction factors of 0.96 and 0.92, respectively (Harrison 1986). In Study 1, plasma volume changes relative to the euhydration trial with fluid replacement were calculated from hematocrit and hemoglobin values using the equations of Dill and Costill (1974). In Studies 2 and 3, changes in plasma volume were determined relative to the pre-treatment euhydration trial.

### *4.3.7. Tolerance Time*

Tolerance Time (TT) for all trials was defined as the time until rectal temperature reached 39.3 °C, heart rate remained at or above 95% of maximum for 3 min, dizziness or nausea precluded further exercise, either the subject or the experimenter terminated the experiment, or 4 h had elapsed. Except for differences in the time limit, the criteria used to define TT were identical to those used previously in heat stress studies with NBC clothing conducted at DCIEM (McLellan and Frim 1994).

### *4.3.8. Core Temperature*

Core temperature was measured at the rectum using a flexible vinyl-covered probe (Pharmaseal 400, Baxter Healthcare Corp.) inserted approximately 0.15 m beyond the anal sphincter. The rectal site was preferred over esophageal temperature measurements due to the difficulty and discomfort associated with an esophageal probe while wearing the NBC clothing and the respirator. Due to the slower response time of the rectal temperature site, there is a possiblity that $T_{re}$ would underestimate the "real" core temperature rise during

heat exposure (Saltin and Hermansen 1966). However, under conditions of uncompensable heat stress, Kraning and Gonzalez (1991) observed nearly identical rectal and esophageal temperatures after approximately 30 minutes of exercise. The tolerance time for all trials was anticipated to be at least 45 minutes, so it is likely that rectal temperature was a valid indicator of overall core temperature in the present series of studies.

#### *4.3.9. Skin Temperature*

Skin temperature was measured using thermistors (Thermistor Bead 4404, Yellow Springs Instrument). An overall $\overline{T}_{sk}$ value that provided a general indication of skin temperature throughout the body was calculated from the weighted averages over 12 sites, using the weightings presented by Vallerand (1989). The sites and weighting factors were as follows: forehead (0.07), chest (0.085), abdomen (0.085), upper back (0.09), lower back (0.09), forearm (0.14), wrist (0.05), front (0.095) and rear thigh (0.095), front (0.065) and rear calf (0.065), and foot (0.07).

#### *4.3.10. Gas Exchange Analyses*

During each trial, open-circuit spirometry was used to determine expired minute ventilation, $\dot{V}O_2$, and carbon dioxide production from a 2-min average obtained every 15 min. An adaptor was attached to the respirator to collect expired air. Expired gases were directed into a 5 L mixing box and through a turbine (Alpha Technologies VMM 110 series ventilation module) for determination of minute ventilation ($\dot{V}_E$). A sampling line directed dried gases from the mixing box to $O_2$ (S-3A Applied Electrochemistry) and $CO_2$ (CD-3A Applied Electrochemistry) analysers. The gas analysers were calibrated before each test with a precision analysed gas cylinder with known $O_2$ and $CO_2$ composition, while the turbine was calibrated with a 3 L syringe. After conversion of the analogue voltage outputs from the ventilation module and the gas analysers into digital signals (Hewlett-Packard 59313 A/D converter), $\dot{V}_E$, carbon dioxide output ($\dot{V}CO_2$), and $\dot{V}O_2$

were calculated and printed on-line every 60 s using appropriate software on a microcomputer. Respiratory water loss was calculated using the measured $\dot{V}O_2$, in $L \cdot min^{-1}$, using the equation presented by Mitchell et al. (1972). Metabolic body mass loss was calculated from the $\dot{V}O_2$, in $L \cdot min^{-1}$, and the RER using the equation described by Snellen (1966).

$$\text{Respiratory water loss } (g \cdot min^{-1}) = 0.445 \cdot \dot{V}O_2 \qquad \text{(Eqn. 4.1)}$$

$$\text{Metabolic weight loss } (g \cdot min^{-1}) = \dot{V}O_2 \cdot (1.9769 \cdot RR - 1.42904) \qquad \text{(Eqn. 4.2)}$$

### *4.3.11. Cardiac Output*

Stroke volume and cardiac output were obtained by impedance cardiography, using the methods first described by Kubicek (Kubicek et al. 1966). A good overview of the theory and methodology behind impedance cardiography was provided by Denniston et al. (1976). One advantage of impedance cardiography over the $CO_2$ rebreathing technique in this situation was that the complete gas mask and respirator could be worn, allowing a more realistic simulation of the breathing resistance and discomfort of the NBC environment. Two aluminised mylar band electrodes (IFM Cardiographic Tape, Bionetics, St. Laurent, Quebec) each were applied around the neck and around the chest. ECG electrodes were also applied. Every 15 min during the HST, 6-8 s of cardiac impedance waveforms were obtained. In order to minimise the motion and respiratory artifacts, the subjects straddled the treadmill and quickly performed an end-expiratory breath hold immediately prior to sampling.

Analyses of the cardiac impedance waveforms were performed on a customised program which allowed the digitisation of individual waveforms. For each timepoint, the waveforms from 5 cardiac cycles were digitised and averaged. Blood resistivity (rho) for each trial was calculated using the adjusted $T_{re}$ and hematocrit values (Mohapatra and Hill 1975). Stroke volumes were calculated using the equations derived by Kubicek (Kubicek et al. 1966) and verified by Denniston et al. (1976) as follows:

$$\Delta V = rho \cdot L^2 \cdot Zo^{-2} \cdot (dZ \cdot dt^{-1})_m \cdot VET \quad \text{(Eqn. 4.1)}$$

$$rho = (6.272Hct + 75.176) - (0.104Hct + 1.467) \cdot T_{re} \quad \text{(Eqn. 4.2)}$$

| | |
|---|---|
| $\Delta V$ | *change in volume or stroke volume (mL)* |
| *rho* | *blood resistivity* ($\Omega \cdot cm^{-1}$) |
| *L* | *distance between the inner pair of band electrodes (cm)* |
| $Z_o$ | *basal thoracic impedance* ($\Omega$) |
| $(dZ \cdot dt^{-1})_m$ | *maximal rate of change of impedance* ($\Omega \cdot s^{-1}$) |
| *VET* | *ventricular ejection time (s)* |
| *Hct* | *corrected hematocrit value* |
| $T_{re}$ | *rectal temperature* ($^oC$) |

### *4.3.12. Heat Gain*

The body heat gain (HG in kJ) in Sections 5.1 and 6.2 during the heat exposure was calculated using the thermometric method of McLellan and Ducharme (1996). For each subject, the body heat content at thermoneutrality before the trial ($HC_N$ in kJ) was subtracted from the body heat content at the end of the trial after the heat exposure ($HC_H$ in kJ) as follows:

$$HG = HC_H - HC_N \quad \text{(Eqn. 4.3)}$$

$$HC_H = (0.90T_{re(f)} + 0.10\overline{T}_{sk(f)}) \cdot m_{b(f)} \cdot 3.47 \quad \text{(Eqn. 4.4)}$$

$$HC_N = (0.79T_{re(i)} + 0.21\overline{T}_{sk(i)}) \cdot m_{b(i)} \cdot 3.47 \quad \text{(Eqn. 4.5)}$$

where the initial and final mean body temperatures for the heat exposure were estimated as $0.79T_{re(i)} + 0.21\overline{T}_{sk(i)}$ and $0.90T_{re(f)} + 0.10\overline{T}_{sk(f)}$, respectively. $m_{b(i)}$ and $m_{b(f)}$ represent the initial and final nude body masses, respectively, and 3.47 is the average heat capacity of the body tissues (in $kJ \cdot kg^{-1} \cdot {}^oC^{-1}$).

# 5. THE EXPERIMENTS

## 5.1. Influence of hydration status and fluid replacement on exercise-heat tolerance while wearing NBC protective clothing

### *5.1.1. Introduction*

The purpose of the first study in this series was to investigate the influence of exercise intensity, hypohydration, and fluid replacement on heat and exercise tolerance in an environment of uncompensable heat stress due to the wearing of protective clothing. The subjects wore the NBC protective clothing ensemble in a hot environment and exercised at light and heavy intensities producing a corresponding HSI ($E_{req} \cdot E_{max}^{-1}$) of 2.5 and 3.5, respectively. The subjects performed exercise in either a euhydrated or a mildly hypohydrated (~2.5 %) state, with the hypohydration level chosen to simulate a level typical of voluntary dehydration (Greenleaf 1992). To investigate the effects of fluid replacement during exercise in an uncompensable heat stress environment, the subjects in the euhydrated state either underwent a fluid replacement program or refrained from drinking. It was hypothesised that hypohydration and fluid replacement will have a major impact on heat tolerance while wearing the protective clothing only during light exercise, where tolerance times are expected to approach 2 hours under normal hydration conditions.

### *5.1.2. Methods*

The experimental protocol was as shown in Chapter 4.

#### 5.1.2.1. Experimental Design

This study examined the responses to exercise in an uncompensable heat stress environment while manipulating the exercise intensity, the initial hydration status, and the availability of fluid replacement during exercise. The responses to the HST were evaluated during light (3.5 $km \cdot h^{-1}$, 0% grade) and heavy (4.8 $km \cdot h^{-1}$, 4% grade) exercise in a euhydrated (EU) and a hypohydrated (-2.5% body mass; HY) state. Classifications of light and heavy exercise were according to U.S. Army guidelines for work rates below 325 W

and above 500 W, respectively (Gonzalez et al. 1993). During EU sessions, the effects of fluid replacement during exercise in the NBC clothing were investigated by the presence (200 or 250 mL each 15 min during light and heavy exercise, respectively; F) or absence (NF) of water rehydration. The subjects undergoing HY trials were tested only in the F condition (i.e., given 200 or 250 mL of water each 15 min) to minimise further losses in body mass. The order in which the different conditions were presented was randomised to minimise any order effects and the effects of partial heat acclimation. For all three studies, plain water was chosen over a commercial replacement fluid to simulate an operational scenario, and water temperature was maintained close to 37°C to minimise effects on body temperature through acting as a heat sink.

#### 5.1.2.2. Data Analyses

Data are presented as mean (± standard deviation). The responses to light and heavy exercise were analysed separately. A two-factor (hydration status x time) repeated-measures analysis of variance (ANOVA) was used to compare the changes in rectal and skin temperature, heart rate, gas exchange responses, stroke volume, and cardiac output. A one-factor (hydration) ANOVA was used to detect differences in tolerance time, body mass changes, sweat rate, evaporative efficiency, heat gain, and plasma osmolality. When a significant F-ratio (corrected for the repeated measures factor) was obtained, a Newman-Keuls post-hoc analysis was performed to isolate differences among treatment means. For all statistical analyses, the 0.05 level of significance was used.

### *5.1.3. Results*

Eight males volunteered to participate in the study. Mean values and S.D. for age, height, body mass, peak aerobic power, body fatness estimated from skinfolds, and Dubois body surface area were 29.3±6.4 y, 1.78±0.07 m, 75.6±9.7 kg, 56.5±4.4 $ml \cdot kg^{-1} \cdot min^{-1}$, 12.4±2.8%, and 1.94±0.15 $m^2$. In addition to the relatively high $\dot{V}O_2 max$, all of the subjects engaged in regular aerobic activities.

The hydration schedule following the dehydration was successful in either reinstating euhydration or in maintaining hypohydration overnight (Table 5.1). In the EU trials, the body mass returned to baseline levels overnight, and was similar for all EU trials. The morning body mass of the subjects during the HY trials was 2.2% lower than during the EU trials for both the light and heavy intensity trials, and was similar to their body mass following the dehydration protocol. For both the light and the heavy exercise trials, plasma osmolality during the HY trials was significantly higher than during the EU trials, while plasma volume was decreased by 5% for light exercise, indicating a hypohydrated state prior to the HY condition.

Compared with the light exercise, the increased rate of heat production during heavy exercise led to higher sweat rates (Table 5.2). However, the low water vapour permeability of the NBC clothing did not permit an increase in evaporative heat loss to the environment, with evaporation rates being similar at approximately 0.30 $L \cdot h^{-1}$ across exercise intensities. Thus, the time required for a 1.0°C increase in $T_{re}$ was less during the heavy trials, indicating a greater rate of heat buildup within the body. During both the light and heavy trials, $T_{re}$ was greater during the HY/F conditions compared with EU/F (Figure 5.1). This was primarily due to an elevated initial $T_{re}$ with HY/F trials, although the rate of $T_{re}$ increase was also slightly greater with HY/F light exercise trials. During heavy exercise, there was a slight, but significant, increase in evaporation rate with EU/F compared with HY/F.

Tolerance to the HST was determined by time, ethically imposed physiological endpoints, or subject exhaustion. None of the trials approached the 4 h time limit. Most of the subjects were very familiar with the NBC clothing and the HST, with five of eight subjects having served as subjects in previous heat experiments in this laboratory. The initial familiarisation session served to accustom all subjects to the protocol and any associated discomforts prior to the actual test. All subjects were highly motivated, and little doubt existed about the presence of exhaustion and imminent collapse. Of the 48 test

Table 5.1 Nude body mass, relative body mass changes, plasma volume changes, and serum osmolality prior to exercise (n=8). EU=euhydration; HY=hypohydration; F=fluid replacement during exercise; NF=no fluid replacement during exercise.

| | Light Exercise | | | Heavy Exercise | | |
|---|---|---|---|---|---|---|
| | EU/F | EU/NF | HY/F | EU/F | EU/NF | HY/F |
| Body Mass (kg) | 75.2* (9.0) | 75.1* (9.2) | 73.6 (9.3) | 75.4* (9.2) | 75.3* (9.5) | 73.8 (9.2) |
| % Body Mass Loss | 0.0* (0.0) | -0.2* (0.6) | -2.2 (1.0) | 0.0* (0.0) | -0.2* (0.7) | -2.2 (0.9) |
| Plasma Volume Change (%) | 0.0* (0.0) | -0.8* (4.9) | -5.8 (5.4) | 0.0 (0.0) | 0.3 (8.8) | -3.1 (7.1) |
| Osmolality (mosm·kg $H_2O^{-1}$) | 285.1* (2.5) | 285.3* (2.8) | 293.6 (5.2) | 283.9* (2.0) | 283.6* (2.6) | 291.9 (3.4) |

Values are means (S.D.). * Significantly different from HY/F.

Table 5.2 Average metabolic rate, sweat rate, and evaporation rate. EU=euhydration; HY=hypohydration; F=fluid replacement during exercise; NF=no fluid replacement during exercise. n=8.

| | Light Exercise | | | Heavy Exercise | | |
|---|---|---|---|---|---|---|
| | EU/F | EU/NF | HY/F | EU/F | EU/NF | HY/F |
| Average Metabolic Rate ($mL \cdot kg^{-1} \cdot min^{-1}$) ++ | 11.2 (0.6) | 11.6 (0.9) | 11.6 (1.0) | 17.9 (0.7) | 17.8 (1.3) | 18.3 (1.0) |
| Average Metabolic Rate ($W \cdot m^{-2}$) ++ | 164 (8) | 166 (12) | 162 (11) | 262 (9) | 260 (14) | 264 (14) |
| Sweat Rate ($L \cdot h^{-1}$) ++ | 1.26 (0.33) | 1.17 (0.29) | 1.27 (0.28) | 1.55 (0.35) | 1.53 (0.37) | 1.41 (0.38) |
| Evaporation Rate ($L \cdot h^{-1}$) | 0.33 (0.06) | 0.30 (0.05) | 0.31 (0.06) | 0.34* (0.04) | 0.33 (0.04) | 0.32 (0.05) |

Values are means (S.D.). * Significantly different from HY/F. ++ Light exercise trials significantly different from heavy exercise trials.

trials, 30 were terminated due to exhaustion. Of the remainder, 15 were terminated due to the $T_{re}$ reaching 39.3°C, and 3 due to the $f_c$ reaching 95% of maximum. In all these cases, the subjects reported that they were very near the point of collapse.

With light exercise, tolerance time for EU/F was significantly longer than either EU/NF or HY/F (Table 5.3), demonstrating an impairment in exercise tolerance due to hypohydration and also a beneficial effect due to fluid replacement during exercise. No difference was observed between the EU/NF and the HY/F trials. For the heavy exercise trials, fluid replacement during exercise produced no additional benefit for tolerance time. A decreased tolerance time was observed during HY/F compared with either EU trial, indicating a slight impairment due to hypohydration even at higher workrates with their shorter tolerance times. No differences were observed in the endpoint $T_{re}$ at which the exercise sessions were terminated across either exercise intensity or hydration conditions.

For both the light and heavy exercise, the body heat gain was significantly less during EU/NF than during either EU/F or HY/F (Table 5.3). During light exercise, heat gain during EU/F was also greater than HY/F. Fluid replacement may benefit heat tolerance by maintaining body mass and providing a greater mass for heat storage (see Equation 4.3-4.5). When heat gain was recalculated by discounting the fluid consumed (the amount of fluid given was subtracted from $m_{b(f)}$) during exercise, no significant differences were observed among the hydration conditions.

The rectal temperature responses over the course of the heat stress tests are presented in Figures 5.1-5.2. During light exercise, $T_{re}$ was significantly higher when hypohydrated than during EU/F. There was a non-significant trend towards an elevated initial $T_{re}$ with HY/F during both the light and heavy exercise, with the resting rectal temperature being elevated by approximately 0.10-0.15°C. The rate of rectal temperature rise was also slightly greater in HY/F compared with EU/F during the light exercise.

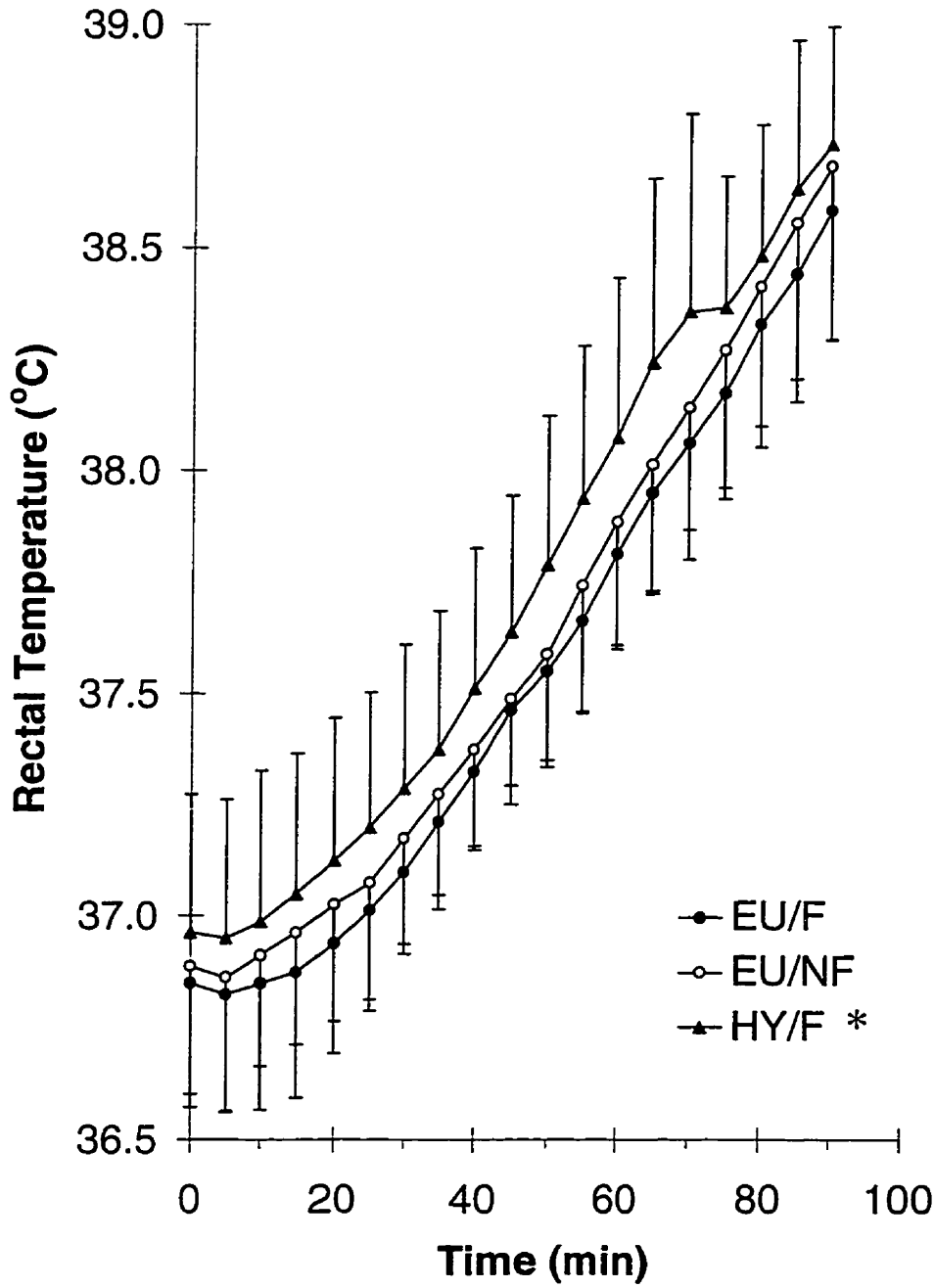
Figures 5.3-5.4 presents the changes in mean skin temperature throughout the exercise sessions. The subjects in all conditions exhibited similar pattern of a rapid

Table 5.3 Tolerance time, time required for a 1.0 °C increase in rectal temperature ($\Delta T_{re}$), starting and ending $T_{re}$, and total heat storage in the body during exercise with and without accounting for the fluid consumed. EU=euhydration; HY=hypohydration; F=fluid replacement during exercise; NF=no fluid replacement during exercise. n=8.

| | Light Exercise | | | Heavy Exercise | | |
|---|---|---|---|---|---|---|
| | EU/F | EU/NF | HY/F | EU/F | EU/NF | HY/F |
| Tolerance Time (min) ++ | 106.5* α (22.1) | 93.1 (20.8) | 87.1 (14.2) | 59.7* (9.5) | 58.3* (11.1) | 53.3 (8.9) |
| Time (min) for 1.0 °C increase in $T_{re}$ ++ | 62.5* (14.3) | 63.6* (12.2) | 54.9 (14.7) | 42.0 (8.3) | 37.1 (4.9) | 37.1 (7.8) |
| $T_{re}$ Start (°C) | 36.85 (0.28) | 36.89 (0.29) | 36.96 (0.31) | 36.94 (0.27) | 36.88 (0.21) | 37.01 (0.25) |
| $T_{re}$ at Endpoint (°C) | 38.90 (0.40) | 38.74 (0.68) | 38.76 (0.46) | 38.69 (0.62) | 38.71 (0.43) | 38.74 (0.47) |
| Heat Storage (kJ) | 570.8* α (132.8) | 413.3 (98.5) | 508.2 α (109.0) | 554.9 α (142.2) | 464.1 (104.7) | 546.8 α (103.8) |
| Heat Storage Discounting Fluid (kJ) | 407.1 (124.0) | 413.3 (98.5) | 378.0 (89.1) | 432.8 (121.8) | 464.1 (104.7) | 433.5 (97.3) |

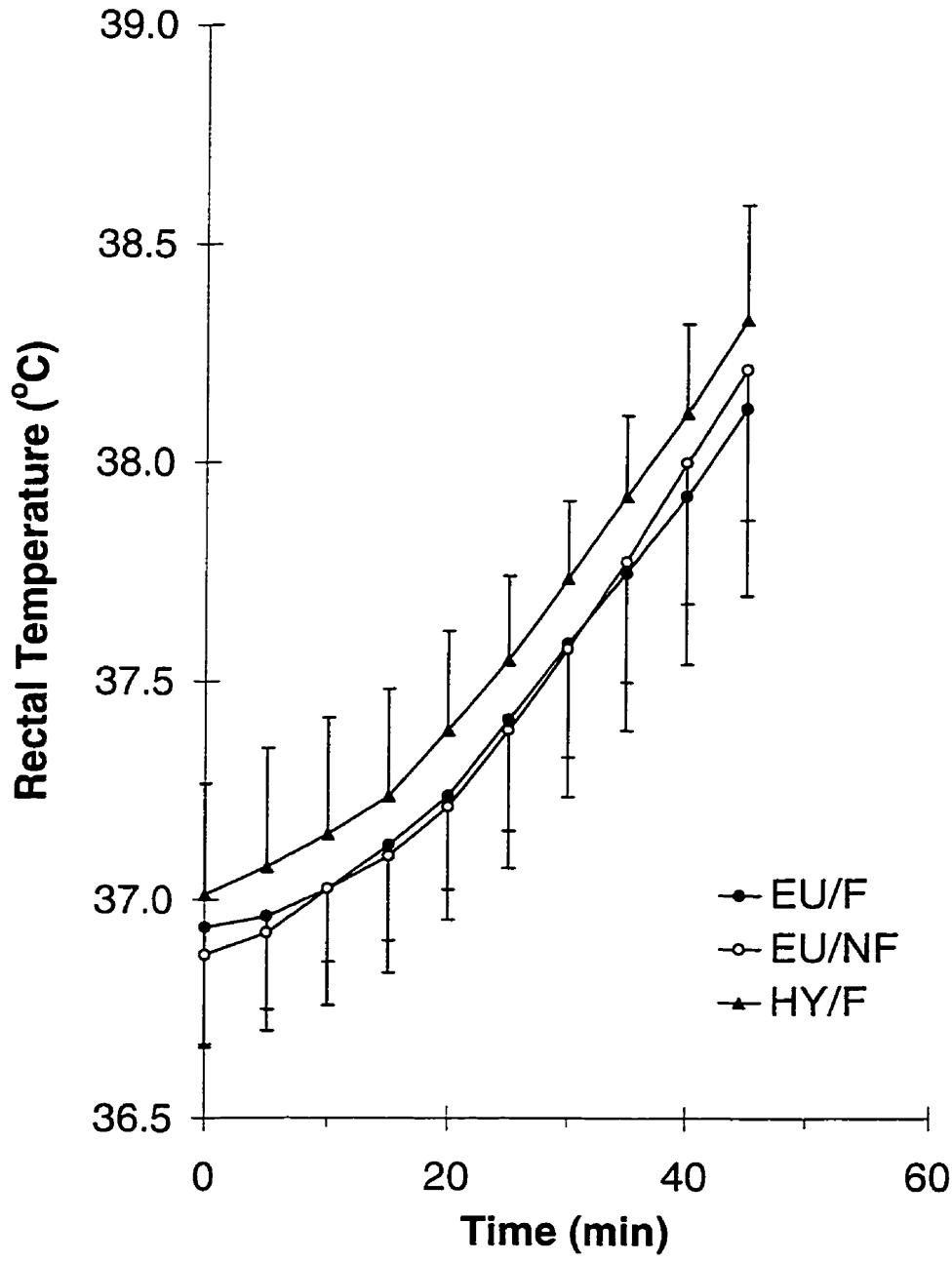
Values are means (S.D.). * Significantly different from HY/F. α Significantly different from EU/NF conditions. ++ Light exercise trials significantly different from heavy exercise trials.

Figure 5.1 Rectal temperature ($T_{re}$) responses to heat stress tests during light (n = 8 to 50 min, n = 7 to 70 min, n = 6 to 90 min) exercise. EU=euhydration; HY=hypohydration; F=fluid replacement; NF=no fluid replacement.



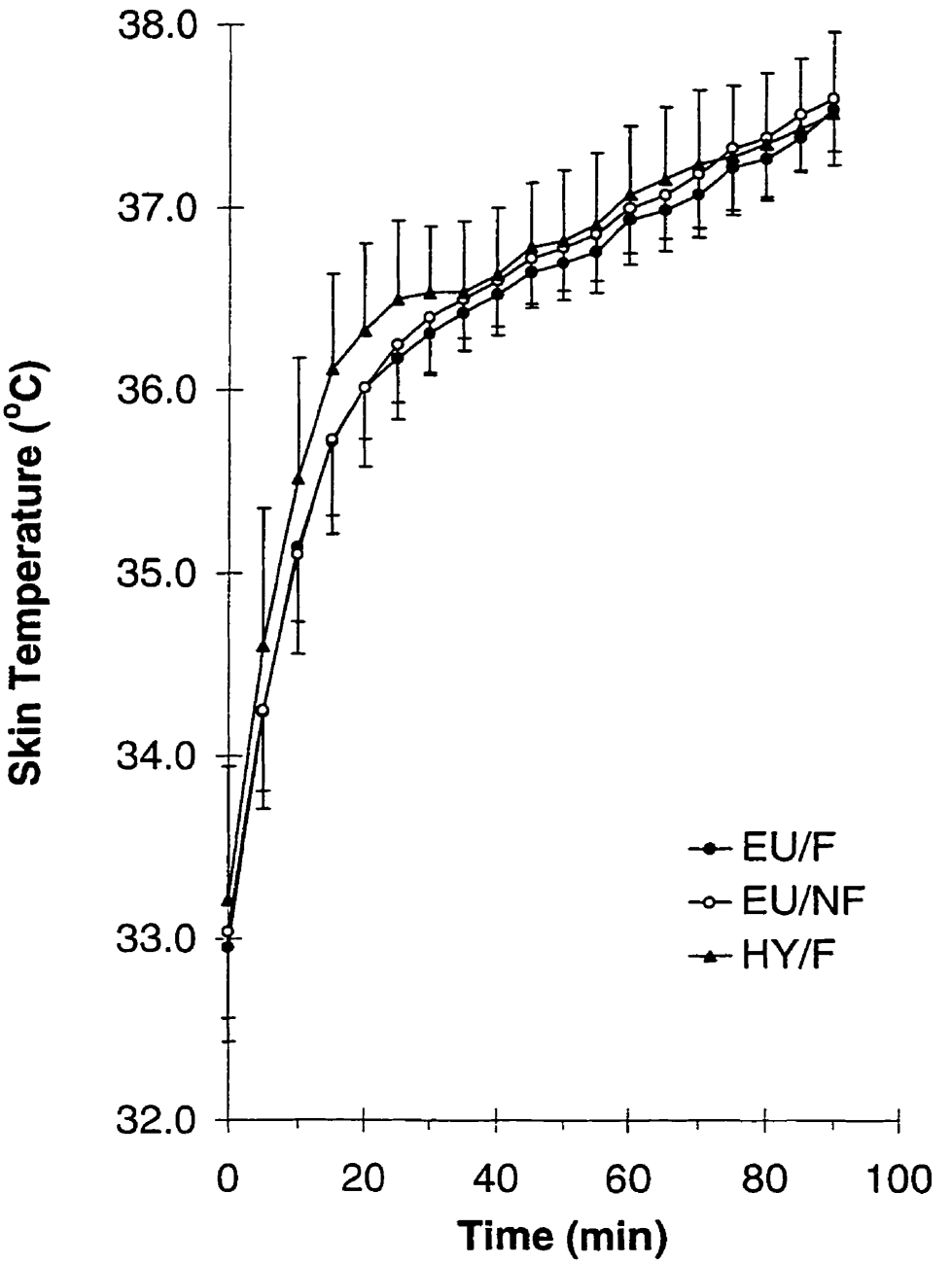

Values are means ± S.D. * Significant main effect of HY/F compared with EU/F.

Figure 5.2 Rectal temperature ($T_{re}$) responses to heat stress tests during heavy (n = 8 to 35 min, n = 7 to 45 min) exercise. EU=euhydration; HY=hypohydration; F=fluid replacement; NF=no fluid replacement.



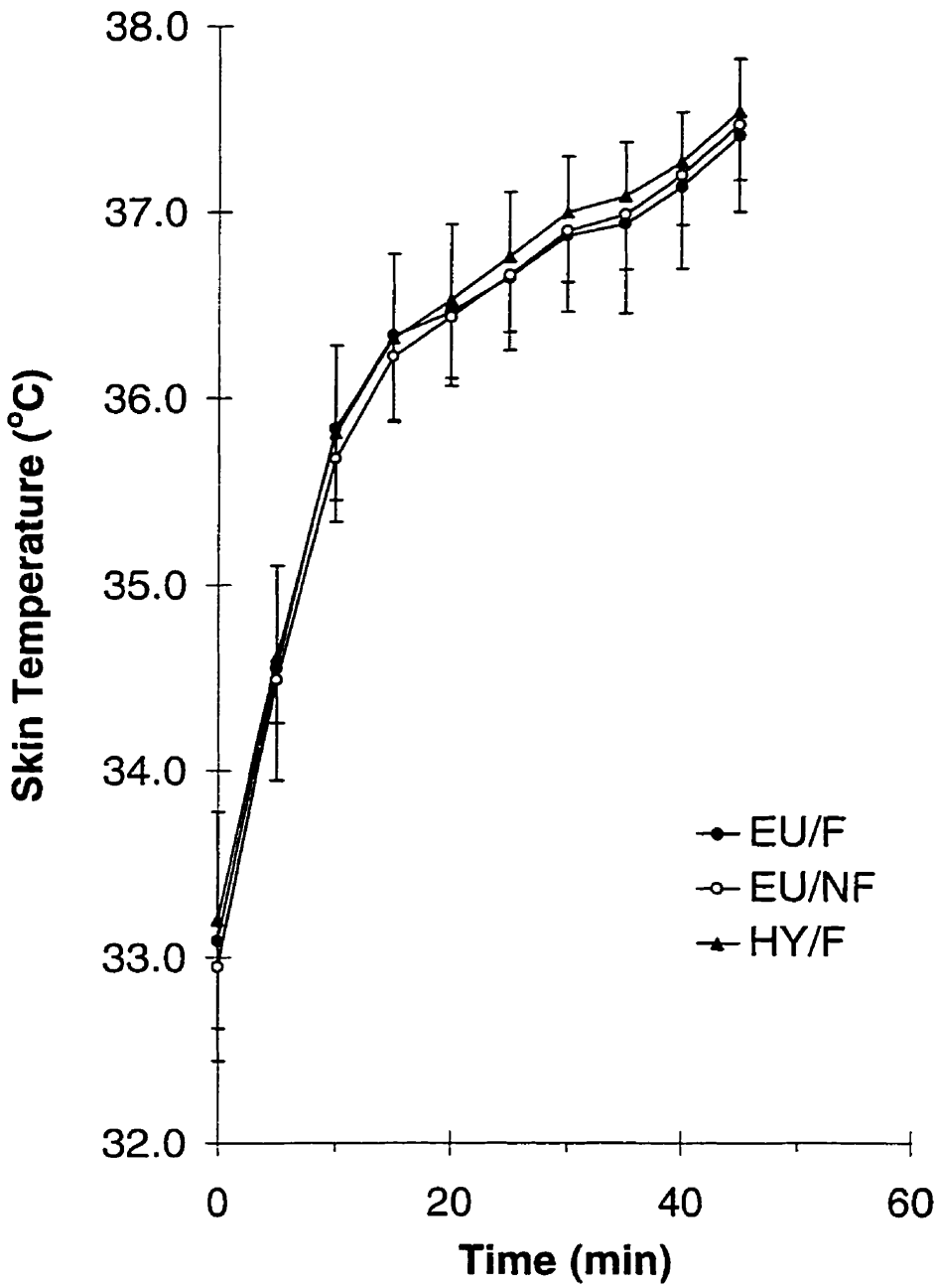

Values are means ± S.D.

Figure 5.3 Mean skin temperature ( $\overline{T}_{sk}$ ) responses to heat stress tests during light (n = 8 to 50 min, n = 7 to 70 min, n = 6 to 90 min) exercise. EU=euhydration; HY=hypohydration; F=fluid replacement; NF=no fluid replacement.




Values are means ± S.D.

Figure 5.4 Mean skin temperature ( $\overline{T}_{sk}$ ) responses to heat stress tests during heavy (n = 8 to 35 min, n = 7 to 45 min) exercise. EU=euhydration; HY=hypohydration; F=fluid replacement; NF=no fluid replacement.




Values are means ± S.D.

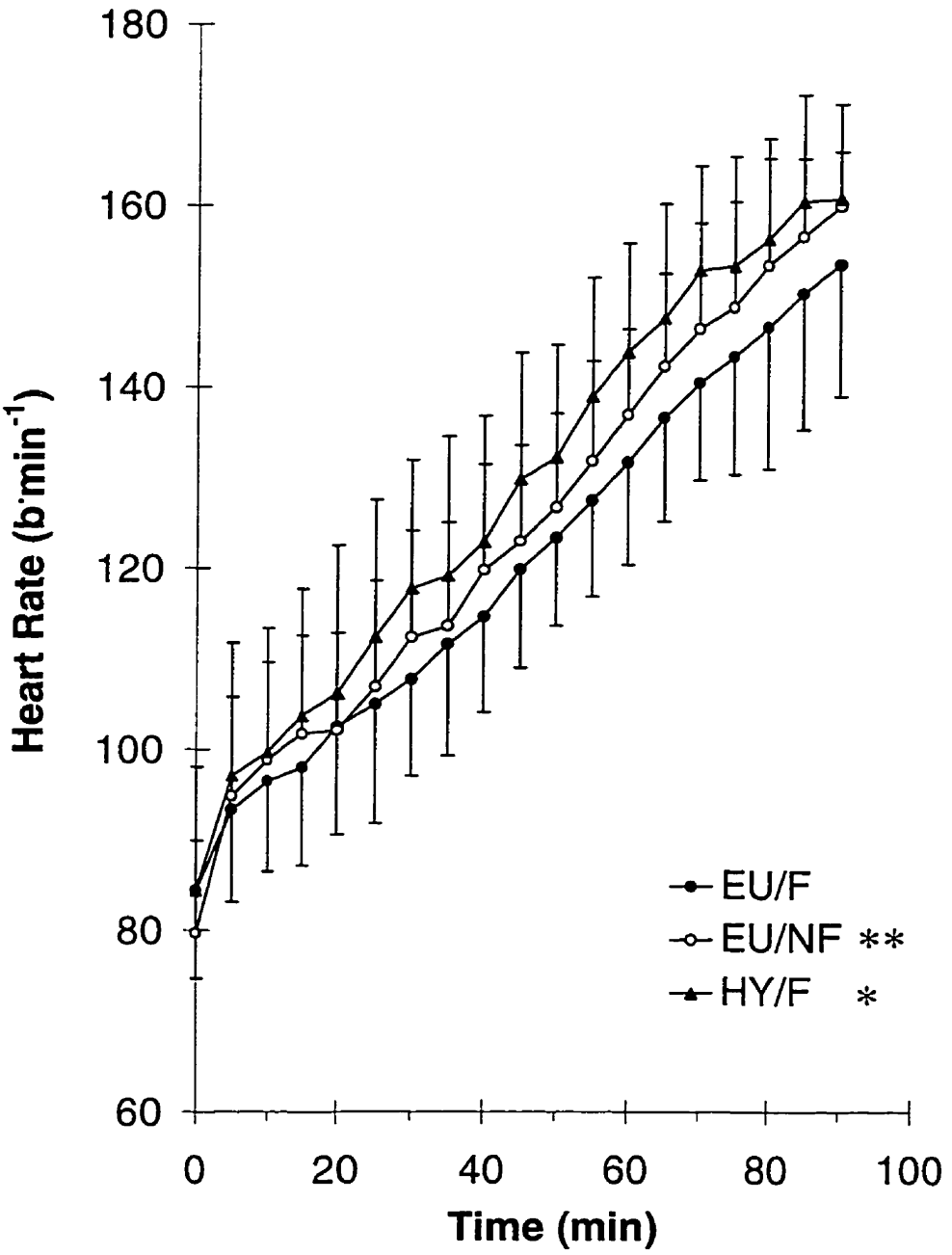
increases in $\overline{T}_{sk}$ during the initial 15 minutes of exercise, followed by a much slower rate of increase. Neither the hydration status nor restricting fluid replacement had an impact on the increase in $\overline{T}_{sk}$ during either the light or heavy exercise.

The changes in heart rate throughout HST for the light and heavy exercise are presented in Figures 5.5-5.6. During light exercise, $f_c$ was significantly higher for HY/F compared with the EU/F trials after 25 minutes and remained elevated for the remainder of the exercise. Fluid replacement elicited a significantly lower $f_c$ in the EU trials after 60 minutes of exercise. During heavy exercise, higher heart rates were found after 10 minutes of walking for HY/F compared with either EU condition. After 30 minutes of walking, fluid during exercise produced an attenuation in the heart rate rise, with a significantly higher heart rate for EU/NF compared with EU/F.

Stroke volume changes throughout the HST are presented in Figures 5.7-5.8. Stroke volume steadily decreased over the course of the HST for all hydration conditions and exercise intensities. During light exercise, there was a main effect of hydration conditions on stroke volume, with a lower stroke volume during HY/F (average of 55.0 mL) compared with both EU/F and EU/NF (averages of 64.2 and 63.0 mL, respectively). During heavy exercise, the EU/F stroke volumes (average of 72.7 mL) were higher than both EU/NF and HY/F (64.9 and 60.6 mL, respectively).

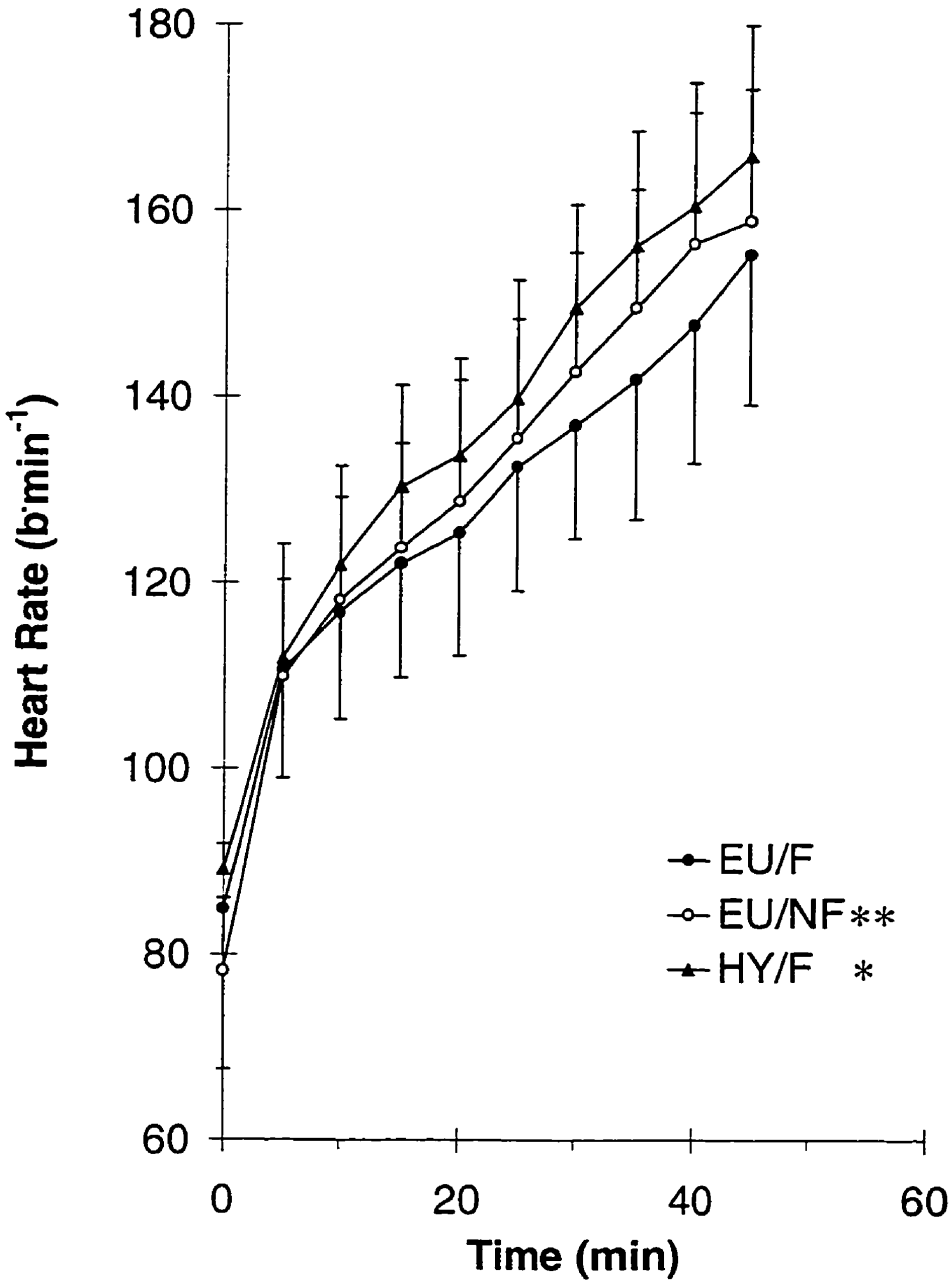
Figures 5.9-5.10 presents the changes in cardiac output throughout the exercise sessions. The decrease in stroke volume during exercise was more than compensated by the increase in heart rate, and cardiac output increased over the course of the HST concomitant with the increase in $\dot{V}O_2$. During both the light and the heavy exercise, no significant differences were observed in the cardiac output for any of the hydration conditions.

Figure 5.5 Heart rate responses to heat stress tests during light (n = 8 to 50 min, n = 7 to 70 min, n = 6 to 90 min) exercise. EU=euhydration; HY=hypohydration; F=fluid replacement; NF=no fluid replacement.



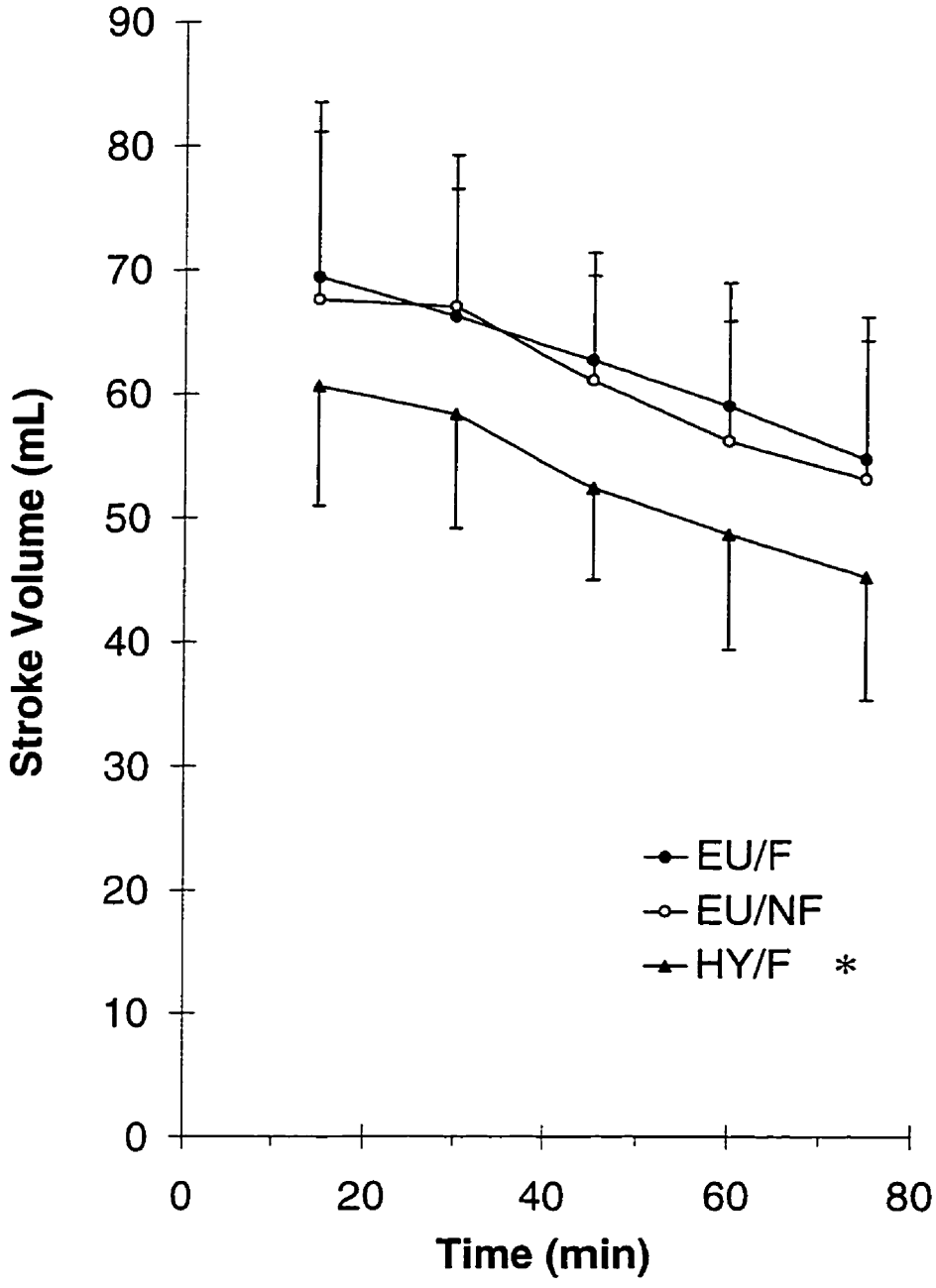

Values are means ± S.D. * HY/F significantly different from EU/F to the end of the session. ** EU/NF significantly different from EU/F to the end of the session.

Figure 5.6 Heart rate responses to heat stress tests during heavy (n = 8 to 35 min, n = 7 to 45 min) exercise. EU=euhydration; HY=hypohydration; F=fluid replacement; NF=no fluid replacement.



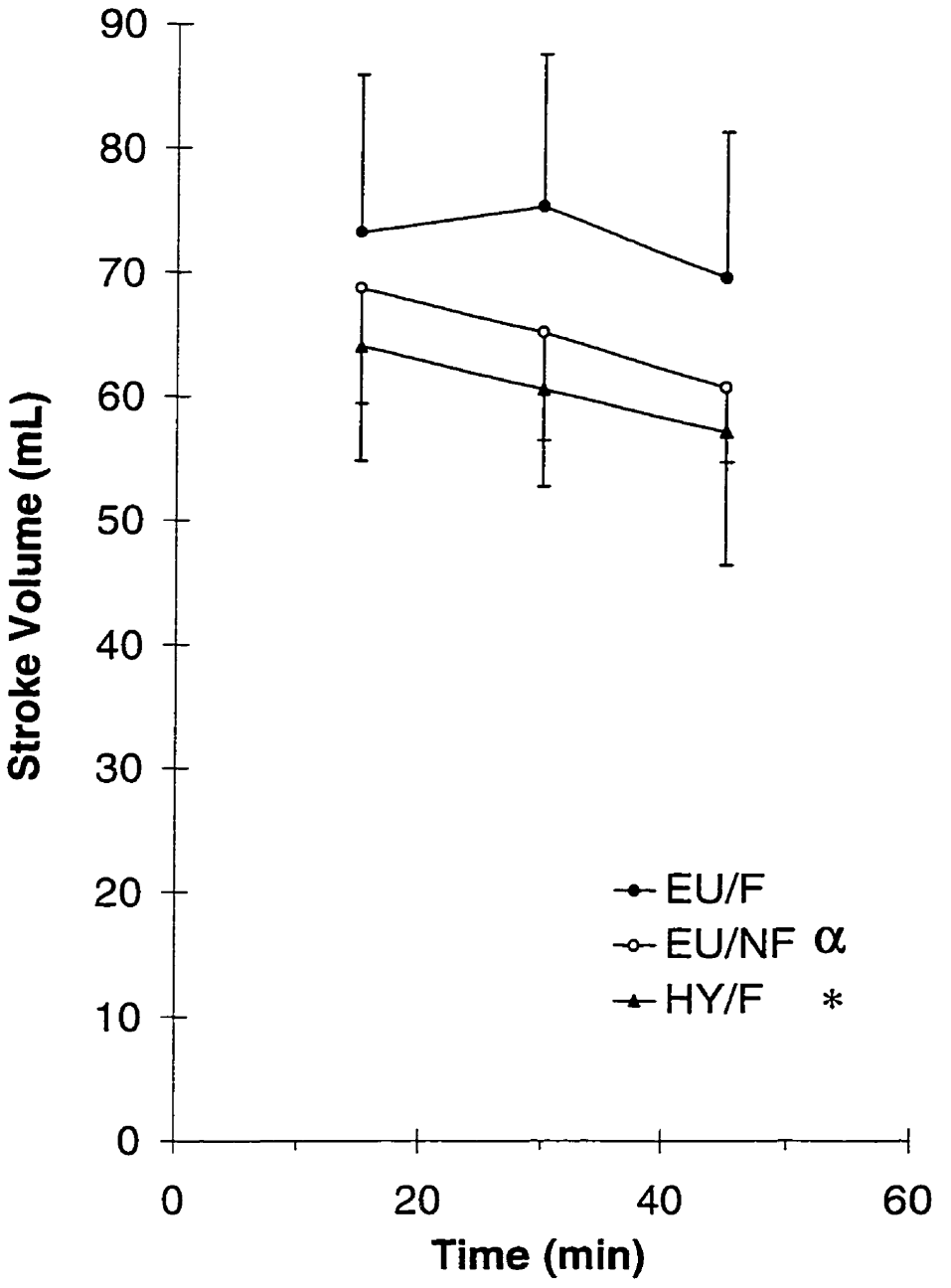

Values are means ± S.D. * HY/F significantly different from EU/F to the end of the session. ** EU/NF significantly different from EU/F to the end of the session.

Figure 5.7 Stroke volume responses to heat stress tests during light (n = 8 to 45 min, n = 7 to 75 min) exercise. EU=euhydration; HY=hypohydration; F=fluid replacement; NF=no fluid replacement.




Values are means ± S.D. * Significant main effect of HY/F compared with EU/F.

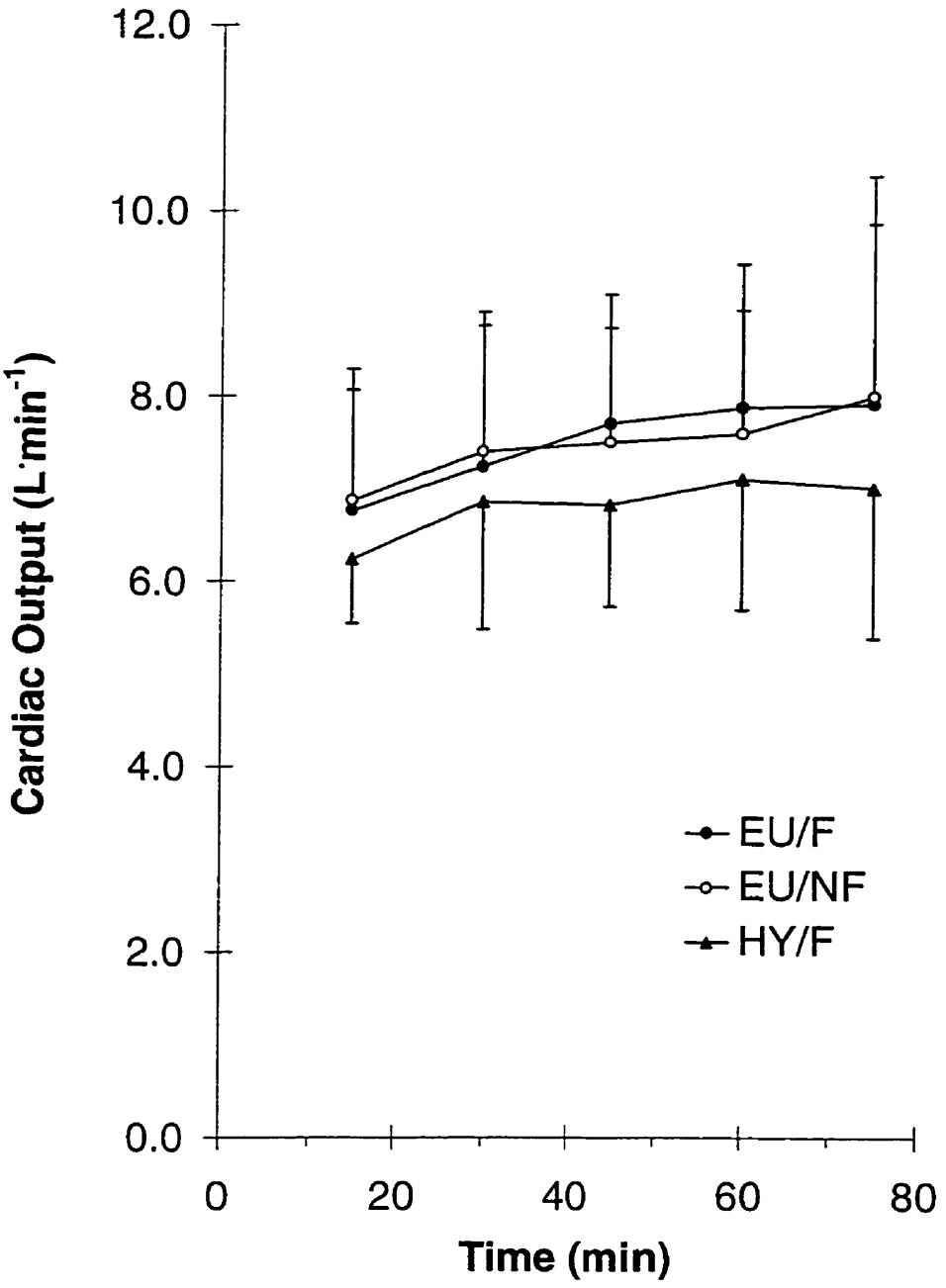
Figure 5.8 Stroke volume responses to heat stress tests during heavy (n = 8 to 30 min, n = 7 to 45 min) exercise. EU=euhydration; HY=hypohydration; F=fluid replacement; NF=no fluid replacement.




Values are means ± S.D. * Significant main effect of HY/F compared with EU/F.

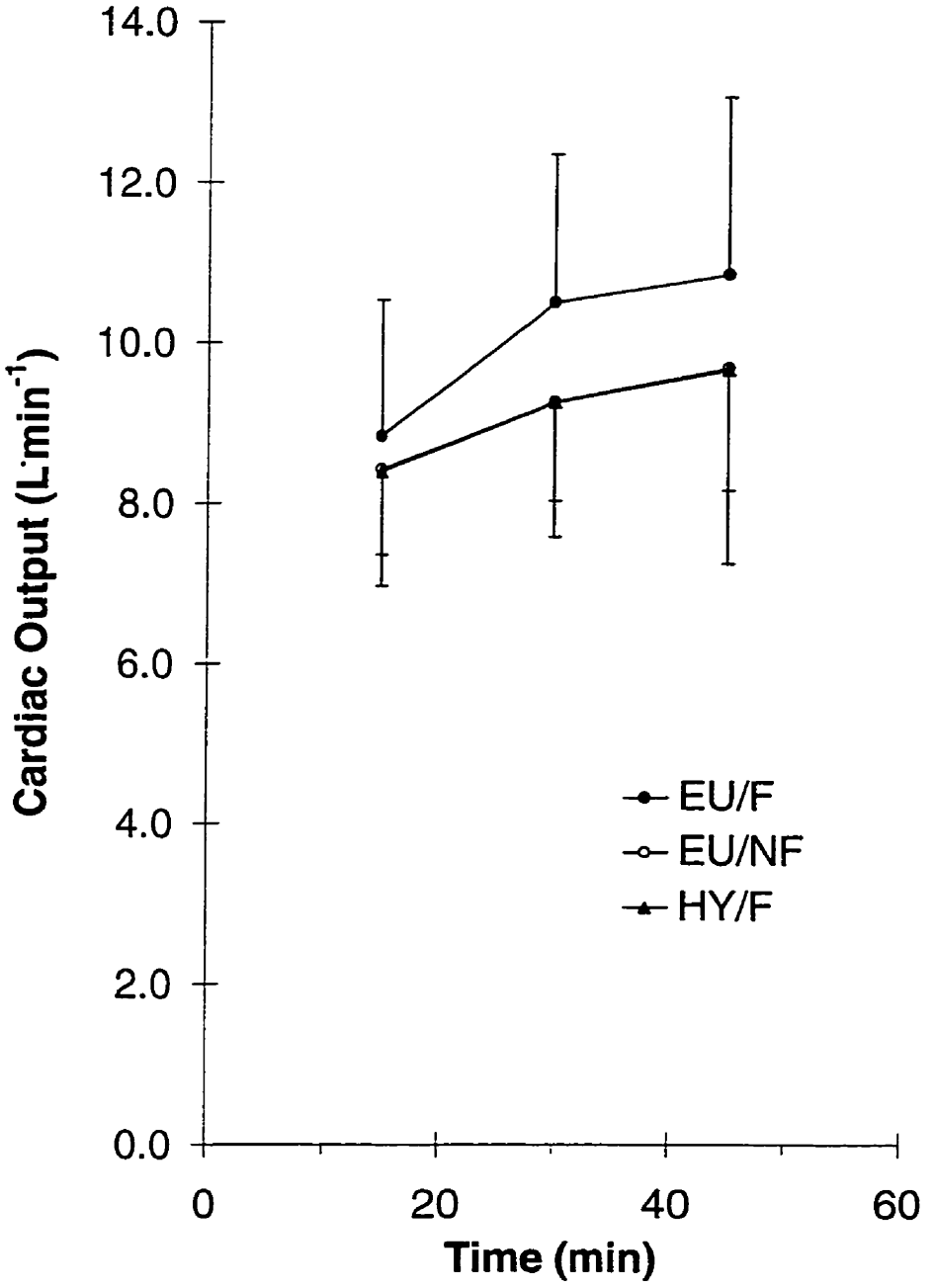
α Significant main effect of EU/NF compared with EU/F.

Figure 5.9 Cardiac output responses to heat stress tests during light (n = 8 to 45 min, n = 7 to 75 min) exercise. EU=euhydration; HY=hypohydration; F=fluid replacement; NF=no fluid replacement.




Values are means ± S.D.

Figure 5.10 Cardiac output responses to heat stress tests during heavy (n = 8 to 30 min, n = 7 to 45 min) exercise. EU=euhydration; HY=hypohydration; F=fluid replacement; NF=no fluid replacement.




Values are means ± S.D.

### *5.1.4. Discussion*

The wearing of protective clothing during exercise in the heat is known to cause a significant increase in energy expenditure and physiological strain to the individual (Candas and Hoeft 1995, Duggan 1988, Holmer 1995, Patton et al. 1995, Smolander et al. 1984). The combination of the limited vapour permeability of the protective clothing, the treadmill exercise, and the hot environment resulted in an environment of uncompensable heat stress, where the required evaporative heat loss greatly exceeded the capacity, as defined by the vapour pressure of the environment and the characteristics of the clothing (Givoni and Goldman 1972). For the light and heavy exercise intensities in the present study, the HSI values were 2.5 and 3.5, respectively. This was a more severe environment than those of previous studies that have examined the influence of hydration status on uncompensable heat stress, where the HSI approached a maximum of about 1.0 (Sawka et al. 1985, Sawka et al. 1992). In addition, the present study was unique in investigating the separate effects of hypohydration and fluid replacement during exercise in the heat specifically while wearing protective clothing. The results of the present study have indicated that, under these conditions of uncompensable heat stress, tolerance time was significantly affected by the hydration status of the subjects prior to beginning the exercise bout. The rate of $T_{re}$ increase during light exercise was significantly higher for HY/F compared with EU/F, indicating a beneficial thermoregulatory effect of maintaining euhydration prior to exercise. However, no significant difference between the EU/NF and the HY/F condition was present. This suggests that, in an environment of uncompensable heat stress, fluid replacement during light exercise is of equal importance to euhydration in maintaining performance. During heavy exercise, there was a small, but significant, reduction in tolerance time during HY/F compared with EU/F or EU/NF, indicating a minor beneficial effect from maintaining an euhydrated state prior to exercise.

Decreased sweating has been suggested as the main contributor to the excessive rise in core temperature during hypohydration (Greenleaf and Castle 1971). Hydration status may affect thermoregulatory or cardiovascular responses to exercise via alterations of body fluid volumes or tonicity (Nielsen 1974) or through the direct impairment of sweat gland function (Taylor 1986). Increasing levels of hypohydration result in a graded increase in the threshold temperature for the onset of sweating and a graded decrease in the sensitivity of the sweating response during exercise in a warm environment (Montain et al. 1995). In the present study, 2.2% hypohydration significantly raised serum osmolality prior to both exercise conditions (Table 5.1). No differences were observed in whole-body sweat rates among hydration conditions (Table 5.2). However, during light exercise, $T_{re}$ was elevated during the HY/F compared with EU/F (Figure 5.1), implying that the relationship between core temperature and sweat rate was altered, though our method of estimating whole-body sweat rates from changes in nude body mass could not isolate the nature of the change.

Hypohydration resulted in a non-significant trend towards a higher resting core temperature compared with either euhydrated condition prior to both light or heavy exercise (Table 5.3). It would therefore appear that another possible contributing factor to the greater thermal strain observed with hypohydration is an elevation of the resting rectal temperature, suggesting a resetting of the central thermoregulatory setpoint (Bligh 1985). A higher initial $T_{re}$ would result in an elevated $T_{re}$ even if the rate of heat storage was not increased by hypohydration. However, during the light exercise, the rate of $T_{re}$ rise was also greater in HY/F compared with EU/F (Table 5.3), resulting in further thermal strain. The lack of difference in the rate of $T_{re}$ increase over time during the heavy exercise may have been due to the short tolerance time caused by the high metabolic rate and heat storage. If a critical core temperature existed at which heat exhaustion occurred, a higher initial $T_{re}$ would decrease the overall $\Delta T_{re}$ over the course of the exercise, thereby contributing to a decreased tolerance time. This central response to hypohydration may

have been mediated by the elevated serum osmolality, as some of the thermosensitive neurons in the preoptic anterior hypothalamus are also sensitive to changes in osmolality (Silva and Boulant 1984).

Hypohydration placed an additional strain on the cardiovascular system during exercise in the NBC clothing. During both the light and heavy exercise, $f_c$ was significantly increased while hypohydrated compared to euhydrated with fluid replacement over the course of the HST (Figures 5.5-5.6. Progressive increases in plasma osmolality and decreases in plasma volume were observed with increasing levels of hypohydration by Sawka et al. (1985), resulting in progressive elevations in heart rate and core temperature, with core temperature increasing 0.15°C and heart rate 4 $b \cdot min^{-1}$ for each percentage increase in hypohydration. In the present study, heart rate was increased during hypohydration in order to compensate for the decreased stroke volume which reflected, presumably, a decreased blood volume and end-diastolic ventricular volume (Gonzalez-Alonso et al. 1995). In the present experiment, stroke volume was significantly decreased by hypohydration during both the light and heavy exercise (Figures 5.7-5.8). However, the elevated $f_c$ response was successful in maintaining a similar cardiac output during both the EU/F and HY/F conditions (Figures 5.9-5.10).

With the light exercise in the present study, the increased tolerance time during EU/F compared with EU/NF (Table 5.3) was likely due to the beneficial effects of fluid replacement on maintaining adequate plasma and blood volume and central venous return. Tolerance times during the light exercise were of sufficient length to allow the ingested fluids to be emptied from the stomach and absorbed from the intestines to produce a benefit in plasma or blood volume. Heat stress and hypohydration were both observed to reduce the rate of gastric emptying in fit subjects during treadmill running at 50% $\dot{V}O_2$ max (Neufer et al. 1989a). However, the rate of fluid consumption in the present study during the light trials was lower than the gastric emptying rate observed by Neufer et al. (1989a), so it is unlikely that gastric emptying was significantly slowed in the present

study. Fluid replacement during exercise may also have delayed or prevented significant fluid loss from the intracellular fluid compartment. The heart rate for EU/NF was increased at 60 min (n=7) compared with EU/F (Figure 5.5). Fluid replacement during exercise also afforded the subjects a greater tolerance to heat buildup, as heat storage within the body was significantly greater for EU/F than with EU/NF at the point of exhaustion (Table 5.3).

It is possible that the majority of the ingested fluids remained in the digestive tract at the time of exhaustion. Indeed, ingesting fluids during the heavy exercise may have increased subject discomfort, due to an increased stomach volume and gastric residue (Mitchell and Voss 1991). This may have been a particular problem in the HY/F trial, where the subjects were already discomforted by the hypohydration. However, though fluid absorption rates were not measured, the rate of fluid replacement was likely sufficient for the majority of the fluid ingested to empty completely from the digestive tract, as the rate of fluid replacement during heavy exercise corresponded with the gastric emptying rate observed during hypohydrated exercise in the heat (Neufer et al. 1989a). It would appear that, during the heavy exercise, even if the ingested fluid was emptied and absorbed from the digestive tract fast enough to counteract body fluid shifts and decreases in plasma volume due to exercise and sweat loss, other factors limited exercise tolerance.

Similar to previous studies in this laboratory investigating exercise tolerance while wearing the full NBC ensemble in a hot environment, the "heavy" workrates of approximately 18 $ml \cdot kg^{-1} \cdot min^{-1}$ or 500 W resulted in tolerance times of approximately 50-60 min (Table 5.3), with a greater variation in tolerance times evident only during the lighter exercise (McLellan 1993). Because of the increased rate of heat production with the heavy exercise, few subjects in the present study were able to exercise more than 65-70 minutes regardless of hydration status prior to exercise or the presence of fluid during exercise. Thus, it appears that physiological manipulations, in this case fluid replacement,

are ineffective for improving heat tolerance while wearing the protective clothing at higher exercise intensities.

Interestingly, despite the differences in sweat rates, rates of $T_{re}$ increase, and tolerance times between the light and heavy workrates, no differences were observed in the $T_{re}$ at which subjects terminated the trials (Table 5.3). The majority of sessions was terminated by the subjects due to exhaustion. In the trials that were terminated due to the subject reaching the ethically imposed upper limit for $T_{re}$ or $f_c$, the subjects reported that they were very near the point of voluntary termination. The similarity in the endpoint $T_{re}$ was in contrast to studies which observed a significantly lower $T_{re}$ at exhaustion with hypohydration (Sawka et al. 1985, Sawka et al. 1992). However, the reduced tolerance times and lower $T_{re}$ endpoints were only observed with significantly greater (5-7%) levels of hypohydration than in the present study, with no differences being observed at 3% hypohydration (Sawka et al. 1985). It would appear that the mild hypohydration induced in the present study does not necessarily reduce the core temperature that may be tolerated before exhaustion occurs in the NBC clothing. Despite a slower response time, $T_{re}$ was likely to be representative of overall core temperature, as Kraning and Gonzalez (1991) reported nearly identical rectal and esophageal temperatures after ~30 min of exercise during similar uncompensable heat stress conditions.

From the present study, it was concluded that, at both light and heavy exercise intensities, minor levels of hypohydration significantly impaired exercise tolerance in a severely uncompensable heat stress environment. Hypohydration decreased plasma volume and increased plasma osmolality, which may have inhibited peripheral blood flow and the sweating response, resulting in an increased rate of heat storage. Fluid ingestion during exercise was successful in prolonging exercise tolerance only at light exercise intensities. At high exercise intensities, fluid replacement did not have an effect on the body fluid compartments before exhaustion occurred from other factors.

## 5.2. Influence of short-term aerobic training and hydration status on exercise-heat tolerance while wearing NBC protective clothing

### *5.2.1. Introduction*

Section 5.1 manipulated the intensity of the exercise, hydration status, and the availability of fluid replacement. Tolerance times converged at approximately 50-60 min for the heavy exercise regardless of the physiological manipulations, as found previously by McLellan (1993) for a range of ambient conditions. Therefore, in order to maximise the impact of the physiological manipulations, the remaining studies in this series were performed only at the light exercise intensity. The subjects in Section 5.1 were fairly homogenous, with both a high $\dot{V}O_2$ max and an active lifestyle. The purpose of the present study was to determine, in a group of relatively nonfit and inactive subjects, the separate and combined effects of hypohydration and a short-term aerobic training program on exercise-heat tolerance. The subjects of low to moderate fitness were tested while euhydrated and also while hypohydrated by ~2.5% of body mass. Tests were conducted before and after either a two week program of daily aerobic training or a two week control period, with the training program based on a short-term training model which produced an acute and short-term increase in fitness and improvement in physiological response to exercise in previously untrained subjects (Green et al. 1991). The training program was hypothesised to result in an improved physiological response in both an euhydrated and hypohydrated state, with a decrease in the magnitude of impairment while hypohydrated following the training program. It was hypothesised that minor hypohydration would significantly impair the physiological response to exercise in the heat both before and after short-term aerobic training.

### *5.2.2. Methods*

The experimental protocol was detailed in Chapter 4.

### 5.2.2.1. Experimental Design

As one of the purposes was to examine the efficacy of a short-term training program, the subjects with a low initial level of aerobic fitness (LF) were selected in order to maximise the effects of training. Fifteen subjects with a $\dot{V}O_2 max < 50$ $mL \cdot kg^{-1} \cdot min^{-1}$ participated and were randomly assigned to one of two groups. The training group (LF-Train, n=8) underwent a two-week (6 days per week) aerobic fitness program consisting of daily 1 h treadmill sessions at 60-65% $\dot{V}O_2 max$. The control group (LF-Control, n=7) maintained their regular daily routines, which did not include aerobic exercise, during the two weeks. The subjects underwent a HST in both the EU and HY conditions prior to and following the training or control period, exercising at the light intensity (3.5 $km \cdot h^{-1}$, 0% grade) employed in Section 5.1. In between the two post-training HSTs, the LF-Train subjects performed 3-4 training sessions to maintain their fitness levels. The order in which the hydration conditions were presented was randomised among subjects. To counterbalance any order effects, the order of the hydration trials for each subject was reversed following the training or control periods. Fluid replacement at a rate of 200 mL each 15 min was provided in all trials.

### 5.2.2.2. Data Analyses

Data are presented as mean values (± standard deviation). A three-factor (period x hydration x time) repeated measures ANOVA was used to compare the rectal and skin temperature, heart rate, stroke volume, and cardiac output of the subjects undergoing the training or control manipulations. A two-factor (period x hydration) repeated measures ANOVA was used to compare the responses of tolerance time, body mass changes, sweat rate, evaporative efficiency, plasma osmolality, metabolic rate, and respiratory exchange ratio. Following separate data analyses within either the LF-Train or LF-Control group, a comparison was performed across the two groups to detect differences in response to exercise-heat tolerance as a result of the training manipulation. When a significant F-ratio

(corrected for the repeated measures factor) was obtained, a Newman-Keuls post-hoc analysis was performed to isolate the differences among treatment means. For all statistical analyses, the 0.05 level of significance was used.

### *5.2.3. Results*

The physical characteristics of the subjects are presented in Table 5.4. The training and control subjects were similar in age, height, body mass, body fat content, surface area, and surface area-to-mass ratio. The effects of the training and control regimes on the physiological responses during the first and the final day of the manipulations are summarised in Tables 5.4 and 5.5. The short-term training program was effective in significantly increasing $\dot{V}O_2$ max 6.5% from 43 to 46 $mL \cdot kg^{-1} \cdot min^{-1}$ (Table 5.4). In contrast, the two-week control period had no effect on aerobic capacity. Despite the significant increase in aerobic capacity following training, no differences were observed in $\dot{V}O_2$ max between groups either before or following the manipulation. The training program was responsible for a decrease in the cardiovascular and thermal strain induced by the training exercise, as evidenced by a significantly lower heart rate and $T_{re}$ rise at the end of the hour of exercise at the end of the training period (Table 5.5). The training program did not result in an increased sweating response during the training exercise.

Tolerance to the HST was determined by time, ethically imposed physiological endpoints, or subject exhaustion. None of the trials approached the 4 h time limit. The large majority of the HSTs, 58 out of 60, were terminated due to exhaustion, as determined by the subject or the experimenter. The remaining 2 were terminated due to the subject reaching the ethically imposed core temperature limit. No differences in the endpoint $T_{re}$ were observed with any condition in either group.

The hydration schedule following the dehydration protocol was successful in either restoring a euhydrated state or in maintaining hypohydration overnight (Table 5.6). In the EU trials, body masses returned to baseline levels overnight, and were similar for all the

Table 5.4 Physical characteristics of the subjects in the training and control groups. Pre=before and Post=after the training or control period.

| Group | Age (y) | Height (m) | Body Mass (kg) | Body Fat Content (%) | Surface Area ($m^2$) | Surface Area-to-Mass ($m^2 \cdot kg^{-1} \cdot 10^2$) | $\dot{V}O_2$ max ($mL \cdot kg^{-1} \cdot min^{-1}$) | |
|---|---|---|---|---|---|---|---|---|
| | | | | | | | Pre | Post |
| Training (n=8) | 30.3 (6.7) | 1.77 (0.05) | 84.7 (12.6) | 19.2 (3.9) | 2.02 (0.14) | 2.41 (0.20) | 43.2 (2.7) | 46.0 + (2.5) |
| Control (n=7) | 30.6 (6.2) | 1.79 (0.05) | 80.4 (8.3) | 16.3 (3.1) | 1.99 (0.09) | 2.50 (0.18) | 45.3 (2.8) | 45.3 (4.9) |

Values are means (S.D.). + Significantly different from pre-training values

Table 5.5 Sweat rate (SR), change in rectal temperature ($\Delta T_{re}$) and final rectal temperature ($T_{re}$), final heart rate (HR), oxygen uptake ( $\dot{V}O_2$ ) at 45 min, and respiratory exchange ratio (RER) at the start and end of the training (n=8) and control (n=7) periods.

| | Training | | Control | |
|---|---|---|---|---|
| | Day 1 | Day 12 | Day 1 | Day 12 |
| SR ( $L \cdot h^{-1}$ ) | 0.73 (0.18) | 0.78 (0.22) | 0.73 (0.09) | 0.66 (0.07) |
| SR ( $L \cdot {}^{o}C^{-1}$ ) | 0.64 (0.30) | 0.74 (0.19) | 0.64 (0.16) | 0.59 (0.08) |
| $\Delta T_{re}$ (°C) | 1.26 (0.44) | 1.09+ (0.31) | 1.17 (0.17) | 1.11 (0.09) |
| $T_{re}$ end (°C) | 38.38 (0.25) | 38.21 (0.26) | 38.34 (0.13) | 38.43 (0.26) |
| HR end ( $b \cdot min^{-1}$ ) | 146.9 (12.9) | 137.3+ (5.8) | 145.0 (14.8) | 148.1 (16.3) |
| $\dot{V}O_2$ ( $mL \cdot kg^{-1} \cdot min^{-1}$ ) | 28.9 (3.4) | 29.1 (2.4) | 27.7 (2.4) | 27.5 (2.2) |
| RER | 0.89 (0.02) | 0.87 (0.03) | 0.91 (0.04) | 0.90 (0.03) |

Values are means (SD). + Significantly different from Day 1.

Table 5.6 Absolute and relative (to Pre-EU) body mass, change in plasma volume (relative to Pre-EU), and serum osmolality prior to the heat stress tests before and after the training (n=8) or control (n=7) period. EU=euhydration; HY=hypohydration.

| | Training | | | | Control | | | |
|---|---|---|---|---|---|---|---|---|
| | Pre-Training | | Post-Training | | Pre-Control | | Post-Control | |
| | EU | HY | EU | HY | EU | HY | EU | HY |
| Body Mass (kg) | 84.15 (12.69) | 82.15* (12.37) | 84.00 (12.80) | 82.09* (12.41) | 80.13 (8.13) | 78.22* (8.09) | 79.57 (8.16) | 77.91* (8.46) |
| % Body Mass Loss | 0.00 (0.00) | -2.00* (0.41) | -0.15 (0.51) | -2.06* (0.53) | 0.00 (0.00) | -1.92* (0.57) | -0.56 (0.85) | -2.22* (0.83) |
| ΔPlasma Volume (%) | 0.00 (0.00) | -4.34* (7.67) | -3.63 (8.55) | -7.00* (5.64) | 0.00 (0.00) | -4.13* (2.40) | -0.61 (4.92) | -5.87* (6.15) |
| Osmolality (mosm kg $H_2O^{-1}$) | 287.3 (5.6) | 293.2* (3.4) | 286.2 (2.5) | 291.8* (3.3) | 287.4 (2.3) | 291.7* (3.3) | 287.7 (2.2) | 292.4* (3.8) |

Values are means (SD). * Significant main effect of hydration

EU trials. The average morning body mass of the training and control subjects during the HY trials ranged from 1.9-2.2% less than during the Pre-EU trial, with no significant differences in relative hypohydration within or among groups. A main effect of a decreased plasma volume with hypohydration was observed in both the LF-Train and LF-Control groups. Compared with the euhydrated trials, serum osmolality was elevated prior to the hypohydrated trials for both the LF-Train and LF-Control groups. These results all indicate that the subjects were significantly hypohydrated prior to the HY trials.

Table 5.7 summarises some of the physiological responses to the HST. The metabolic rate, and therefore, the rate of heat production, was similar for all trials for both the control and training groups. As expected, the responses to the HST were similar before and following the two-week control period. In trained subjects, though no change in sweating response occurred while training in a cool environment, the more extreme heat environment of the HST was sufficient to expose a difference in the drive for heat dissipation due to training. One major change as a result of the two-week aerobic training program was an increased sweat rate during the HST . However, due to the difficulty in water vapour transfer through the NBC ensemble, these adaptations did not result in significant changes in evaporation rate. Overall, no effect of the training or control period was observed on tolerance time.

Hydration status had a significant effect on the responses to exercise-heat tolerance. Hypohydration did not affect the rate of heat production or the drive for heat dissipation, with similar metabolic rates and sweating and evaporation rates for both the LF-Train and LF-Control groups. Before and after the training or control period, hypohydration resulted in a significantly shorter tolerance time, with the higher initial $T_{re}$ a likely contributing factor. Interestingly, the respiratory exchange ratio was significantly lower during hypohydration trials in both the LF-Train and LF-Control groups. No significant differences were observed in $\dot{V}O_2$, indicating that the difference was due to a decreased $\dot{V}CO_2$.

Table 5.7 Sweating and evaporation rate, average metabolic rate, respiratory exchange ratio (RER), rates of ventilation ($\dot{V}_E$), oxygen uptake ($\dot{V}O_2$), and carbon dioxide production ($\dot{V}CO_2$), tolerance time, time required for rectal temperature ($T_{re}$) to increase by 1.0, and initial and final $T_{re}$ during heat stress tests before and after the training (n=8) or control (n=7) period. EU=euhydration; HY=hypohydration. Values are means (SD). * Significant main effect of hydration. + Significant main effect of training. ++ Significant difference between the training and control groups.

| | Training | | | | Control | | | |
|---|---|---|---|---|---|---|---|---|
| | Pre-Training | | Post-Training | | Pre-Control | | Post-Control | |
| | EU | HY | EU | HY | EU | HY | EU | HY |
| Sweat Rate ($L \cdot h^{-1}$) | 0.87 (0.27) | 0.92 (0.26) | 1.00+ (0.29) | 1.03+ (0.25) | 0.85 (0.16) | 0.90 (0.19) | 0.83++ (0.18) | 0.88++ (0.18) |
| Evaporation Rate ($L \cdot h^{-1}$) | 0.31 (0.07) | 0.26 (0.06) | 0.29 (0.05) | 0.29 (0.04) | 0.27 (0.04) | 0.24 (0.06) | 0.27 (0.03) | 0.27 (0.05) |
| Average Metabolic Rate ($W \cdot m^{-2}$) | 173.9 (8.3) | 175.0 (8.3) | 171.4 (12.8) | 168.8 (12.2) | 170.9 (9.8) | 163.9 (8.3) | 166.5 (9.0) | 165.7 (8.2) |
| RER | 0.87 (0.03) | 0.84* (0.02) | 0.88 (0.04) | 0.83* (0.02) | 0.86 (0.04) | 0.83* (0.02) | 0.87 (0.05) | 0.83* (0.02) |
| $\dot{V}_E$ ($L \cdot min^{-1}$) | 21.8 (2.4) | 22.0 (2.7) | 22.0 (2.7) | 21.0 (3.1) | 22.8 (2.1) | 22.2 (2.8) | 22.8 (1.9) | 22.8 (3.8) |
| $\dot{V}O_2$ ($mL \cdot kg^{-1} \cdot min^{-1}$) | 11.00 (1.15) | 11.32 (1.05) | 10.85 (0.98) | 10.72 (1.45) | 11.05 (0.70) | 10.85 (0.38) | 10.82 (0.44) | 11.00 (0.55) |
| $\dot{V}CO_2$ ($L \cdot min^{-1}$) | 0.868 (0.059) | 0.848* (0.060) | 0.860 (0.083) | 0.823* (0.064) | 0.838 (0.064) | 0.774* (0.048) | 0.822 (0.077) | 0.788* (0.068) |
| Tolerance Time (min) | 93.1 (18.9) | 75.8* (14.4) | 94.0 (16.2) | 80.3* (11.7) | 85.3 (10.2) | 74.6* (10.1) | 90.9 (11.9) | 79.6* (10.3) |
| $\Delta T_{re} = 1$ °C (min) | 64.3 (8.1) | 57.4 (11.1) | 65.1 (9.6) | 62.5 (9.3) | 65.3 (10.1) | 63.3 (6.2) | 66.4 (7.6) | 64.7 (5.4) |
| Initial $T_{re}$ (°C) | 37.08 (0.24) | 37.18* (0.34) | 36.93 (0.34) | 37.20* (0.35) | 36.93 (0.22) | 37.04* (0.22) | 36.99 (0.19) | 37.19* (0.13) |
| Endpoint $T_{re}$ (°C) | 38.70 (0.37) | 38.63 (0.35) | 38.61 (0.25) | 38.60 (0.42) | 38.46 (0.36) | 38.49 (0.28) | 38.60 (0.34) | 38.56 (0.39) |

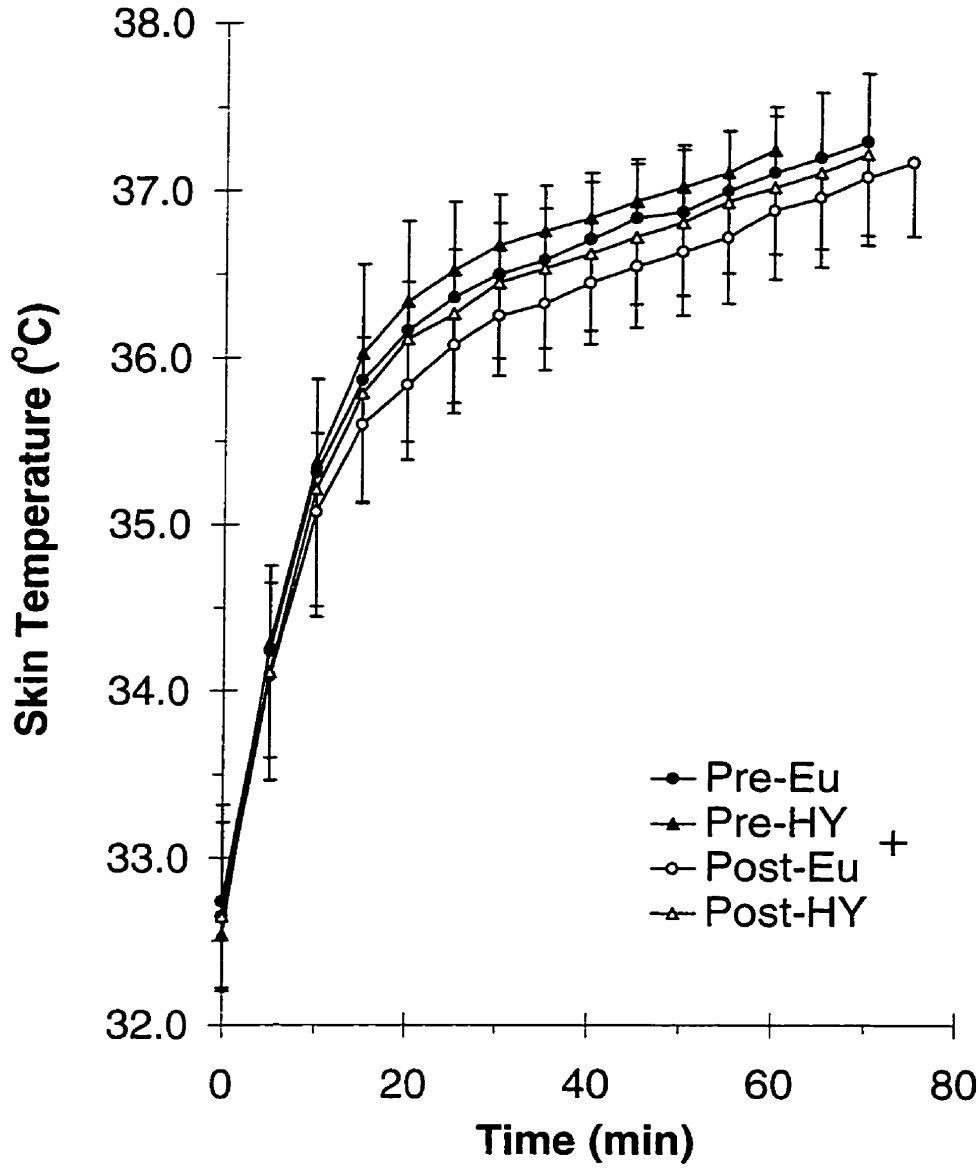
Comparing the responses to the HSTs between the LF-Train and LF-Control groups, no significant between-group differences were observed in tolerance time, metabolic rate, RER, evaporation rate, initial and endpoint $T_{re}$, and time required for $T_{re}$ to increase 1.0°C. While the sweating rates were similar prior to the training or control period, the LF-Train group had a significantly higher sweat rate compared with the LF-Control group following the two weeks of treatment.

The $\overline{T}_{sk}$, $T_{re}$, and $f_c$ responses to the HST are presented in Figures 5.11 to 5.16. As expected, the two-week control period did not influence the responses of the three variables to the HST. The primary effect of the two-week training program was observed in the $\overline{T}_{sk}$ response, in which training led to a significantly lower $\overline{T}_{sk}$ (Figure 5.12), which may have been brought about by the increased sweating and skin wettedness following training. Training did not significantly alter the $T_{re}$ or $f_c$ response to the HST.

Hydration status had a significant effect on thermal and cardiovascular responses to the HST. In both the LF-Train and LF-Control groups, hypohydration resulted in a significantly higher $T_{re}$ throughout the HST, with the primary factor being an elevated initial $T_{re}$ (Figures 5.13-5.14). The $f_c$ response to the HST was also influenced by hydration status (Figures 5.15-5.16). Hypohydration elicited a significantly higher rise in heart rate after 5 and 15 minutes in the LF-Train and LF-Control groups, respectively, contributing to a higher overall heart rate with hypohydration in both groups. In contrast, $\overline{T}_{sk}$ was not influenced by hydration status in either the LF-Train and LF-Control group (Figures 5.11-5.12). No overall differences in $T_{re}$, $\overline{T}_{sk}$, or $f_c$ response were observed between the LF-Train and LF-Control groups.

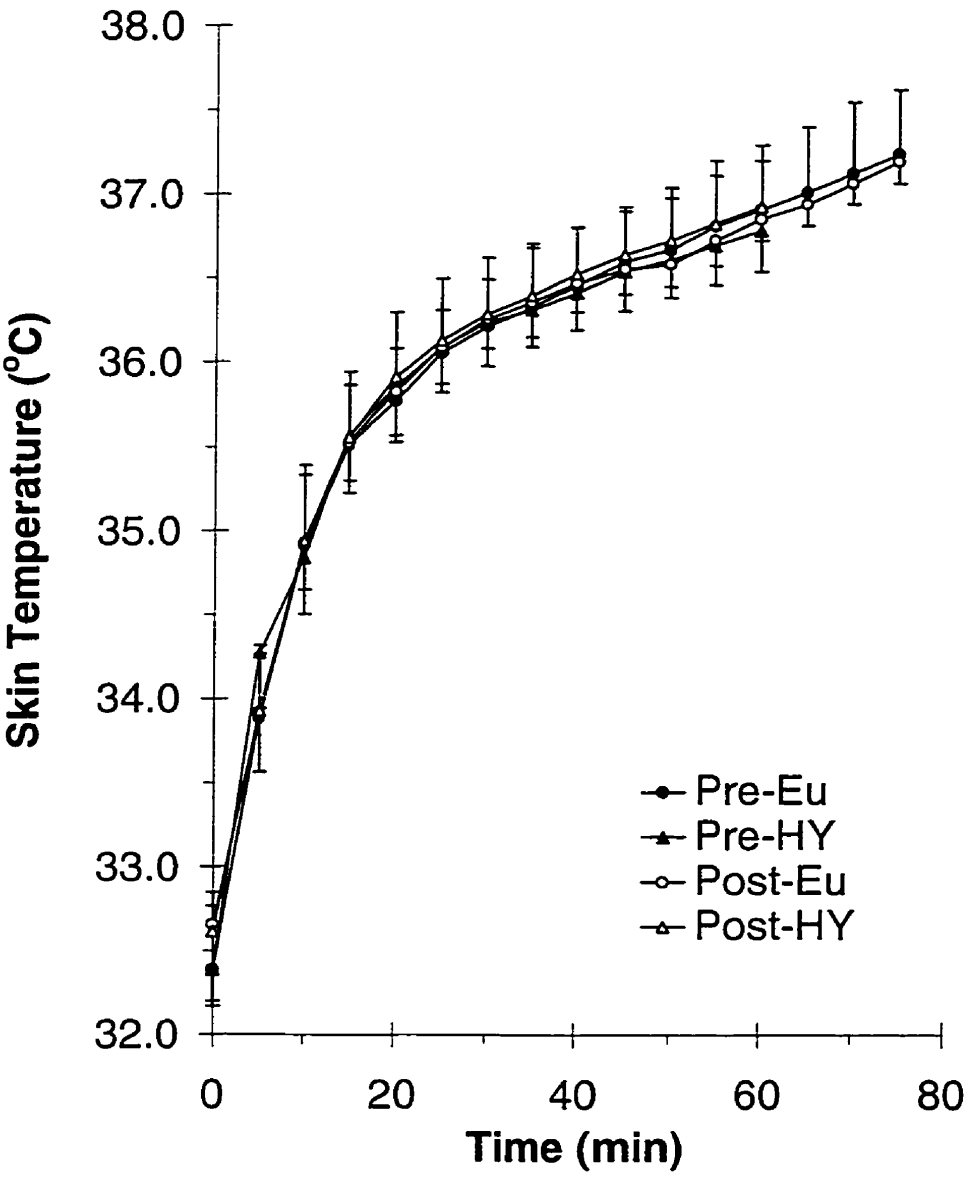
The stroke volume and cardiac output responses during the HST are presented in Figures 5.17-5.20. The increased aerobic capacity brought about by the training program did not influence stroke volume or cardiac output during the HST. However, the hydration status had a minor influence on stroke volume in the LF-Train subjects, with a slight, but non-significant ($p < 0.06$), decrease in stroke volume being observed when

Figure 5.11 Mean skin temperature ( $\overline{T}_{sk}$ ) response to the heat stress test while either euhydrated (EU, circles) or hypohydrated (HY, triangles), before (filled symbols) and after (open symbols) two weeks of training (n = 8).



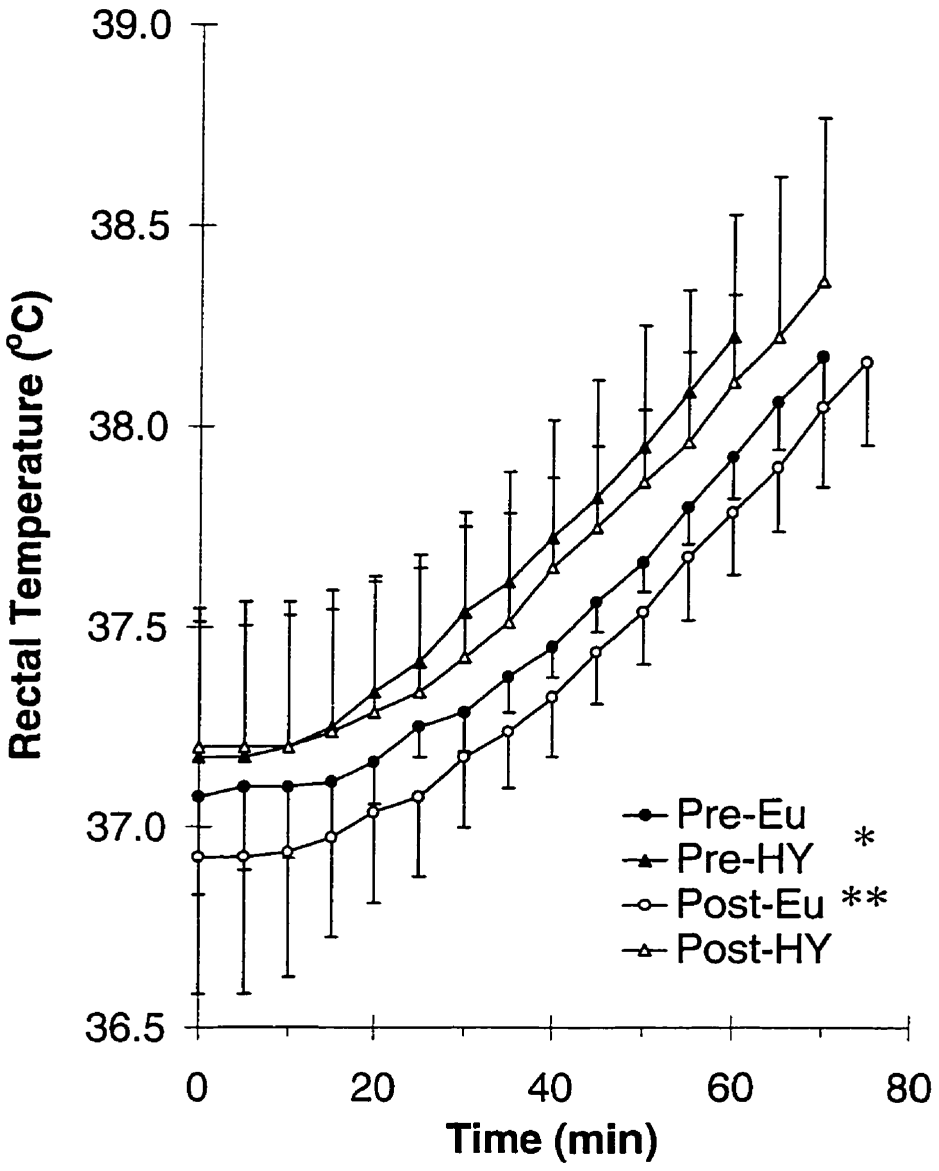

Values are mean ± S.D. + Significant main effect of training.

Figure 5.12 Mean skin temperature ( $\overline{T}_{sk}$ ) response to the heat stress test while either euhydrated (EU, circles) or hypohydrated (HY, triangles), before (filled symbols) and after (open symbols) two weeks of control (n = 7).



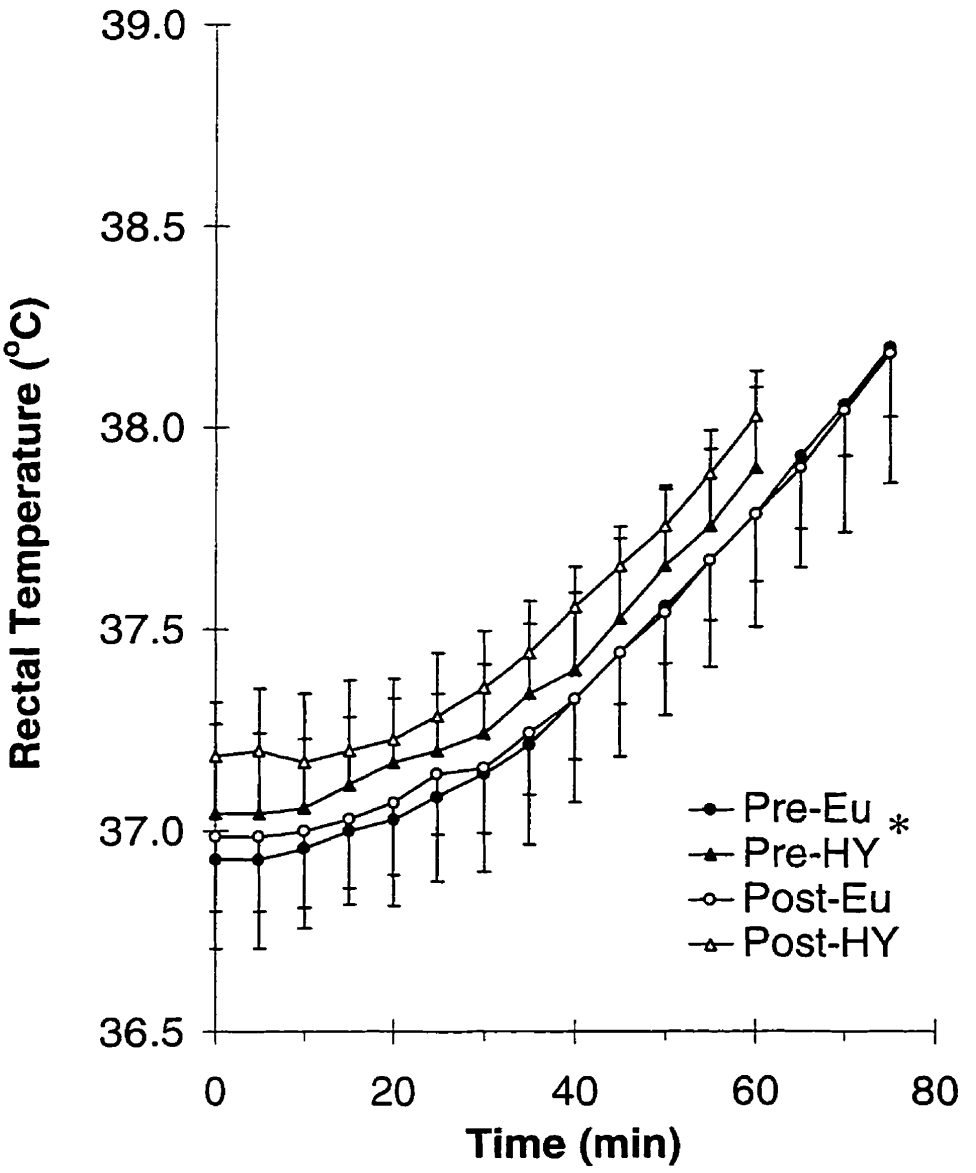

Values are mean ± S.D.

Figure 5.13 Rectal temperature ($T_{re}$) response to the heat stress test while either euhydrated (EU, circles) or hypohydrated (HY, triangles), before (filled symbols) and after (open symbols) two weeks of training (n = 8).



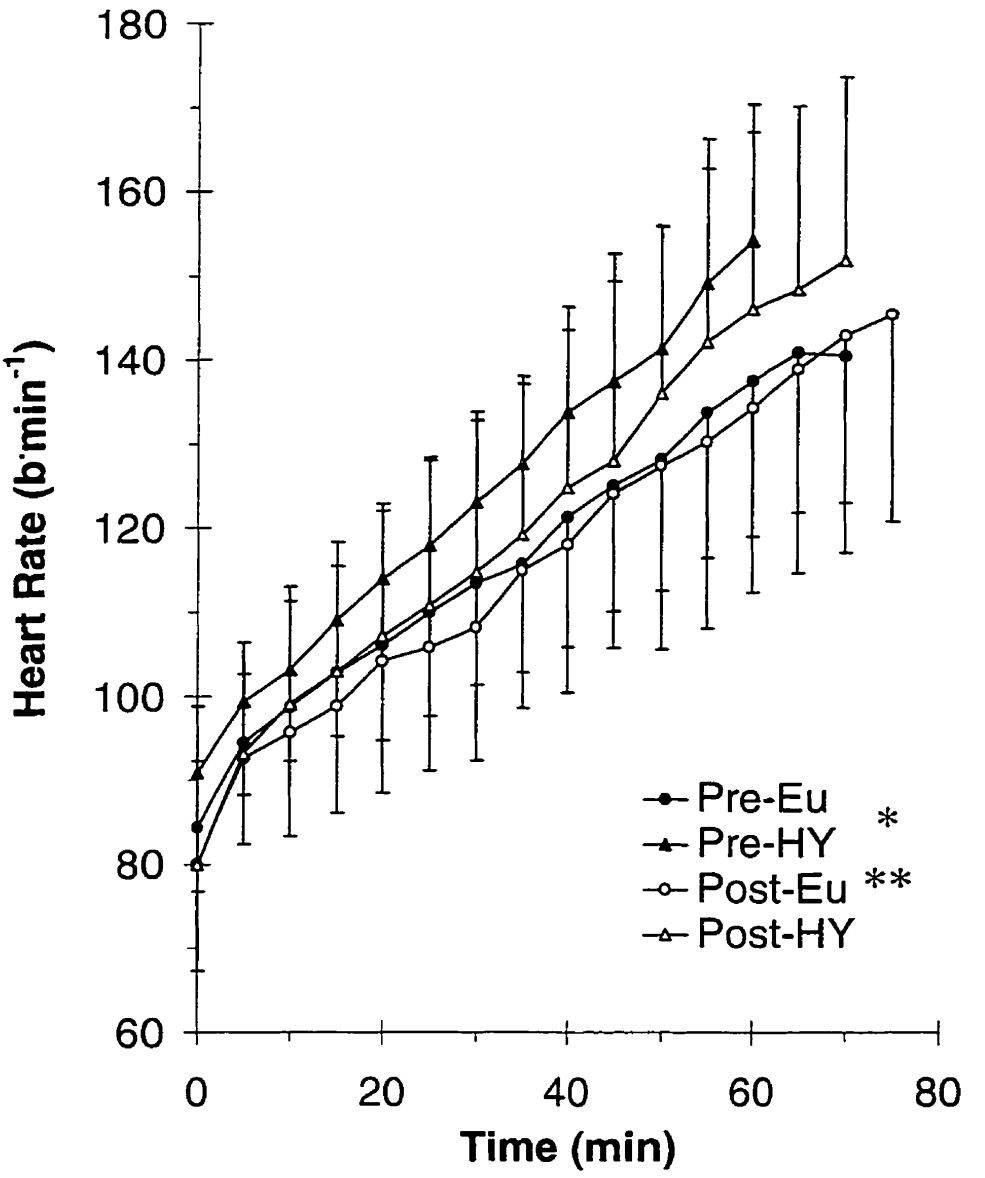

Values are mean ± S.D. * Significant main effect of hydration. ** Significant hydration x time interaction.

Figure 5.14 Rectal temperature ($T_{re}$) response to the heat stress test while either euhydrated (EU, circles) or hypohydrated (HY, triangles), before (filled symbols) and after (open symbols) two weeks of control (n = 7).



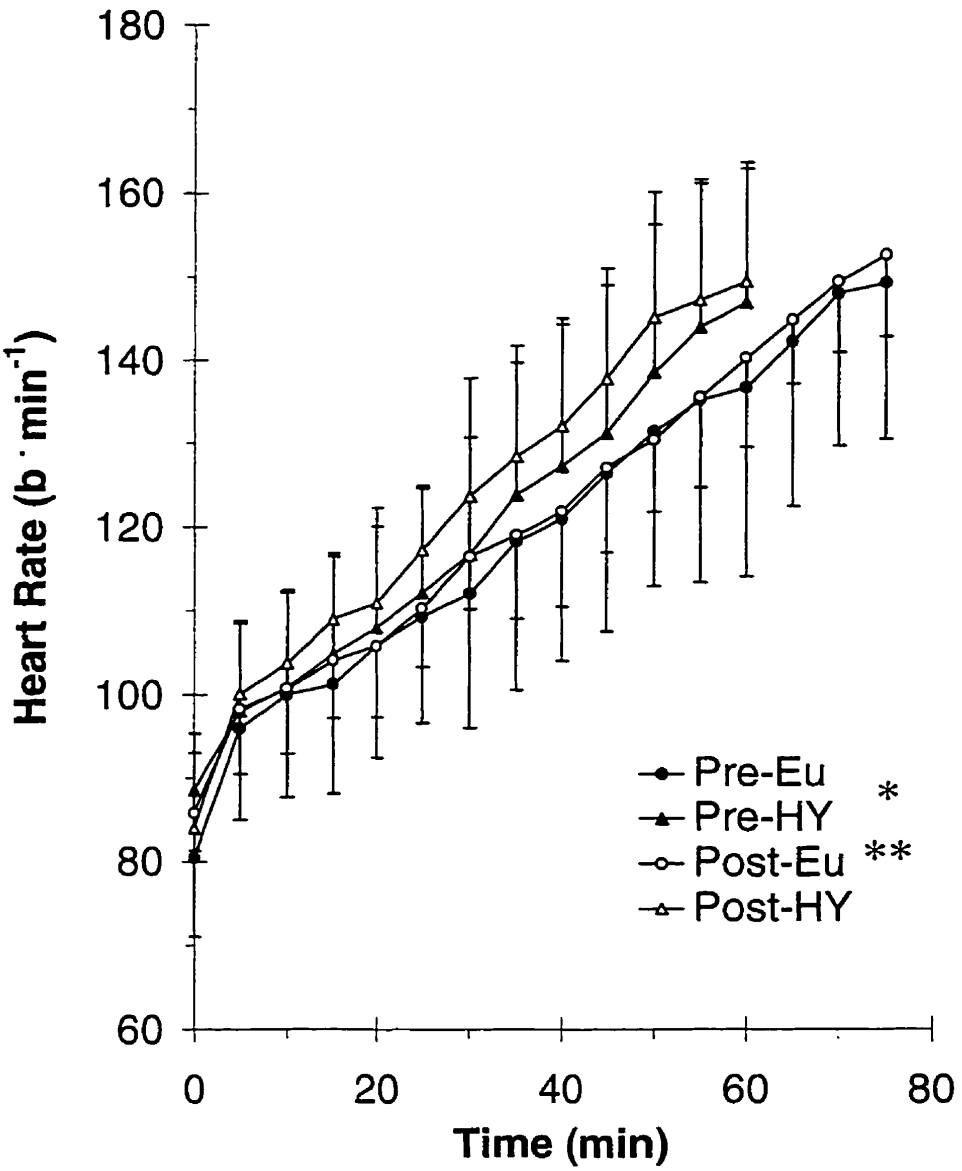

Values are mean ± S.D. * Significant main effect of hydration.

Figure 5.15 Heart rate response to the heat stress test while either euhydrated (EU, circles) or hypohydrated (HY, triangles), before (filled symbols) and after (open symbols) two weeks of training (n = 8).



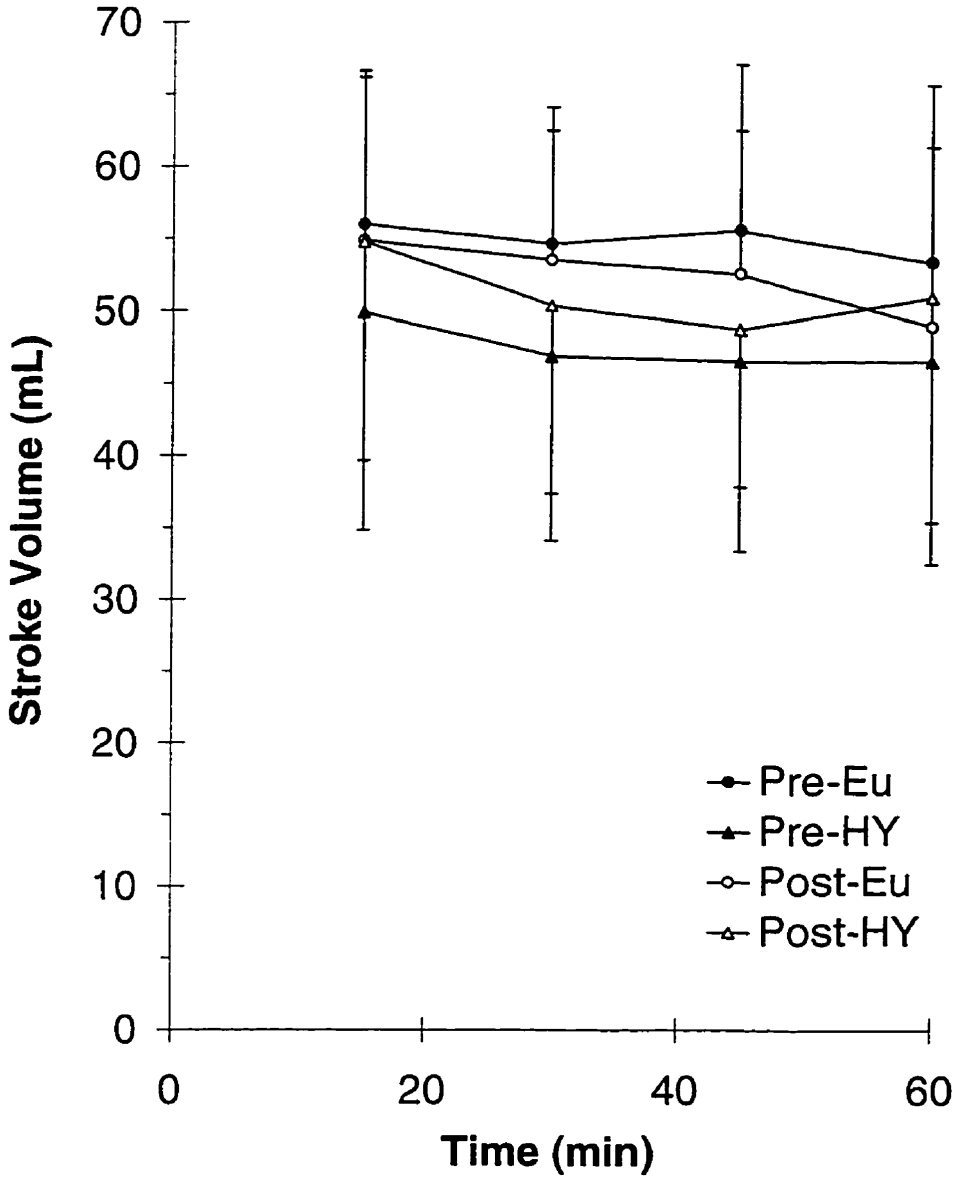

Values are mean ± S.D. * Significant main effect of hydration. ** Significant hydration x time interaction.

Figure 5.16 Heart rate response to the heat stress test while either euhydrated (EU, circles) or hypohydrated (HY, triangles), before (filled symbols) and after (open symbols) two weeks of control (n = 7).



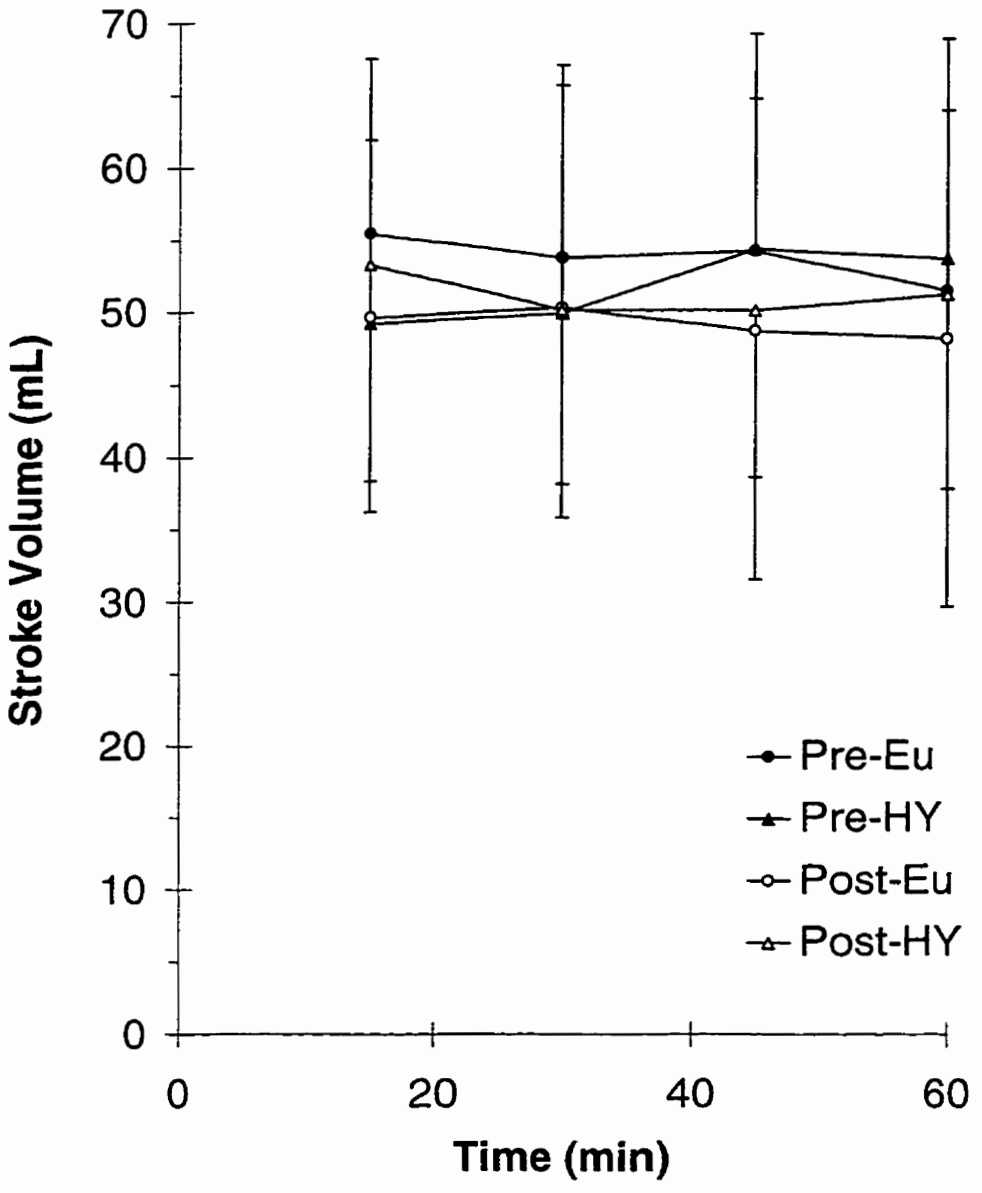

Values are mean ± S.D. * Significant main effect of hydration. ** Significant hydration x time interaction.

Figure 5.17 Stroke volume response to the heat stress test while either euhydrated (EU, circles) or hypohydrated (HY, triangles), before (filled symbols) and after (open symbols) two weeks of training (n = 8).



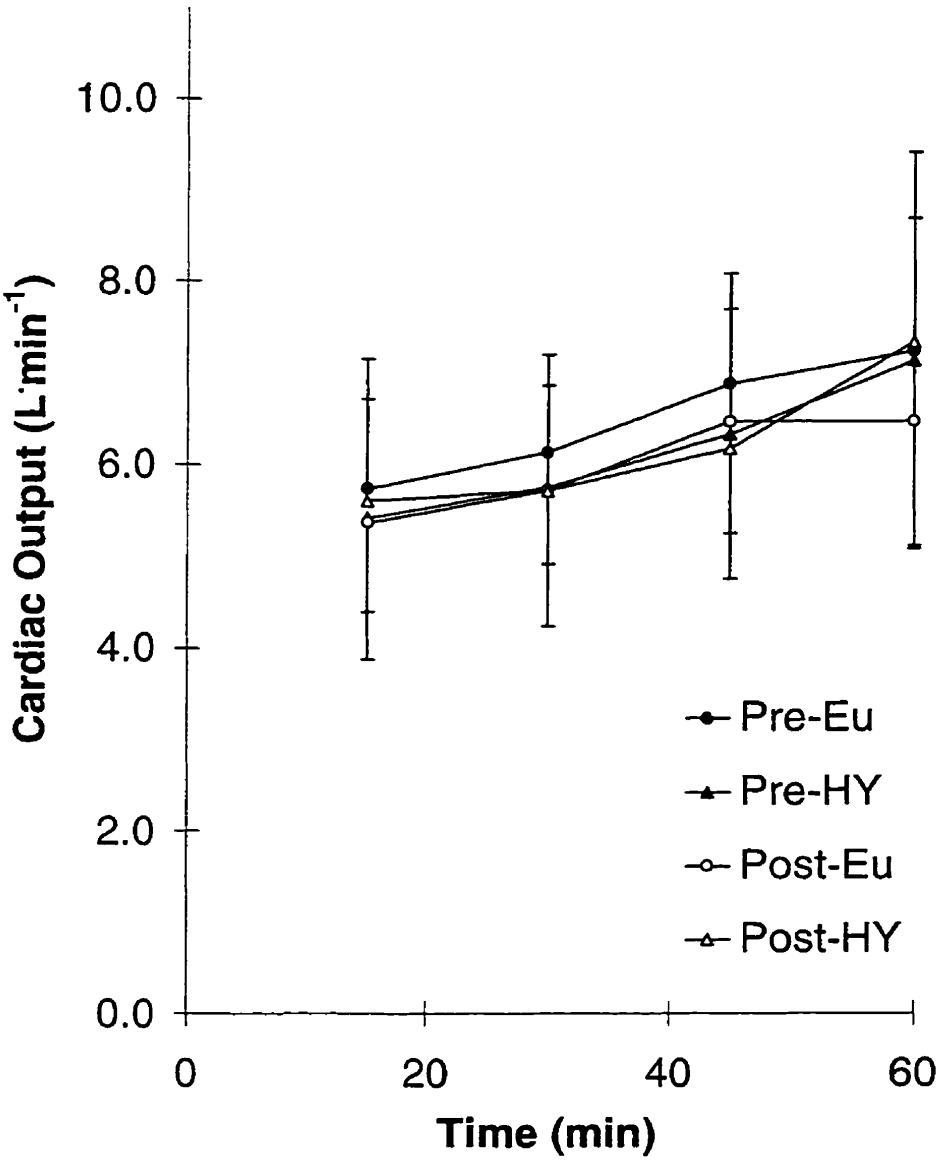

Values are mean ± S.D.

Figure 5.18 Stroke volume response to the heat stress test while either euhydrated (EU, circles) or hypohydrated (HY, triangles), before (filled symbols) and after (open symbols) two weeks of control (n = 7).



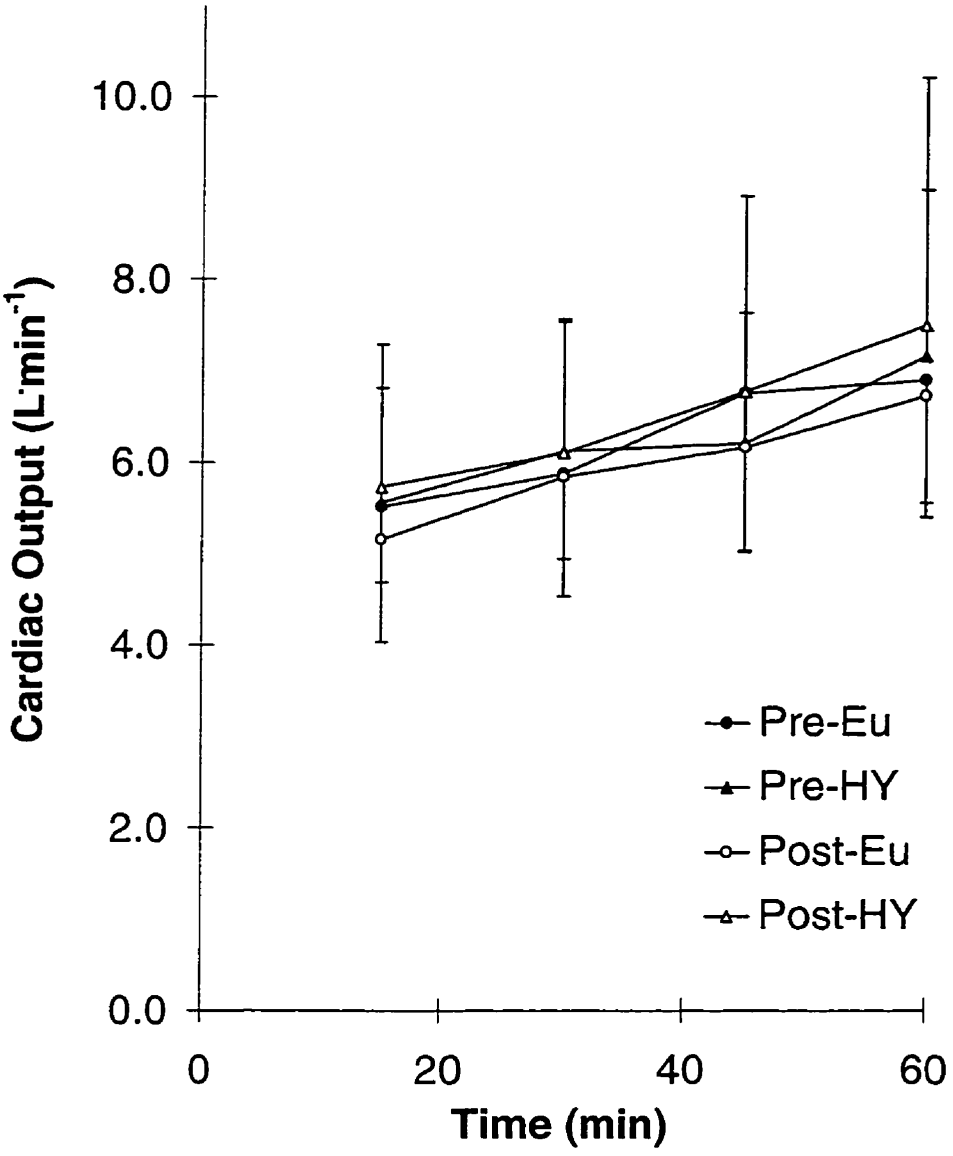

Values are mean ± S.D.

Figure 5.19 Cardiac output response to the heat stress test while either euhydrated (EU, circles) or hypohydrated (HY, triangles), before (filled symbols) and after (open symbols) two weeks of training (n = 8).




Values are mean ± S.D.

Figure 5.20 Cardiac output response to the heat stress test while either euhydrated (EU, circles) or hypohydrated (HY, triangles), before (filled symbols) and after (open symbols) two weeks of control (n = 7).




Values are mean ± S.D.

hypohydrated. The decreased stroke volume in the LF-Train group when hypohydrated was compensated by an elevation in heart rate, resulting in no overall effect of hydration status on cardiac output in the LF-Train group. No effect of hypohydration on stroke volume was observed in the LF-Control group, and the relative instability and variability in stroke volume may have been responsible for the lack of an overall difference in cardiac output despite the increased heart rate.

### *5.2.4. Discussion*

A period of physical training elicits adaptations in the body that are similar to those produced by a period of repeated heat exposures, including increases in blood volume (Green et al. 1991, Harrison 1986), a lower resting core temperature (Shvartz et al. 1974), and improvements in the sweating responses (Henane et al. 1977, Nadel et al. 1974). Long-term fitness has also been associated with an increased tolerance to exercise in the heat in both training (Avellini et al. 1982, Gisolfi and Robinson 1969) studies and in comparison across fitness groups (Piwonka and Robinson 1967). For these reasons, improvements in aerobic fitness have been associated with a reduction in physiological strain and an increased performance time during exercise in the heat, and as a result, physical training programs have been used as a method of heat acclimation (Armstrong and Pandolf 1988).

The design of the short-term training model employed in the present study attempted to simulate a military scenario in which individuals could be adapted relatively quickly in a temperate environment prior to deployment to a hot environment. Treadmill exercise was chosen for its lack of requirement for specialised equipment and as an unskilled task to control for any learning component. Training also had to be of sufficient intensity to elicit adaptation, yet be light enough that the untrained and relatively non-fit subjects could perform the entire protocol. The tolerance time for untrained and non-fit subjects cycling at 70-75% $\dot{V}O_2$ max has been reported to be approximately 50 min

(McLellan and Skinner 1985), while Green et al. (1991) have stated that not all previously untrained subjects could complete 2 h of continuous cycling at 59% $\dot{V}O_2$ max for the initial few days of a 10-12 day training program. Therefore, we chose an exercise intensity of approximately 65% $\dot{V}O_2$ max in order to allow all subjects to exercise continuously for at least 1 h daily throughout the two-week period.

The experimental design incorporated a control group which maintained their inactivity over a two week period. The subjects in the LF-Control group were identical to the LF-Train group in all anthropometric measures and in aerobic fitness prior to the study (Table 5.4). As expected, the control period had no effect on $\dot{V}O_2$ max, and no differences were observed during exercise at 65% $\dot{V}O_2$ max in a thermoneutral environment (Tables 5.4-5.5). The responses to either the euhydrated or hypohydrated HST before or following the control period were identical for all measured variables. Therefore, we are confident that any adaptations in the training group can be attributed to the training manipulation, as opposed to either a learning effect or partial acclimation from the periodic exposures to heat over the course of the study.

Short-term training in the present study resulted in a series of adaptations (Table 5.5) during exercise in a thermoneutral environment, including a decrease in $f_c$ and a decrease in $\Delta T_{re}$ as seen with other training programs (Armstrong and Pandolf 1988, Shvartz et al. 1974). The training program did not result in a significant improvement in the overall sweating response as seen with a higher-intensity program (Henane et al. 1977). However, while the total sweating rate was similar, the decrease in $\Delta T_{re}$ with training implies an effect on the relationship between sweat rate and core temperature (Nadel et al. 1974). Despite a degree of adaptation during exercise in a thermoneutral environment, the only significant alleviation of physiological strain during the exposure to the post-training HST was an increased sweat rate (Table 5.7) and a decreased $\overline{T}_{sk}$ (Figure 5.11), with no improvement in tolerance time (Table 5.7) or in heart rate (Figure 5.13) or rectal temperature (Figure 5.15) response.

The efficacy of a physical training program for improving heat tolerance is not a universal finding (Shvartz et al. 1973). Factors such as the design of the training program and the environment to be encountered must also be considered. The intensity of the training stimulus is a major determinant of the magnitude of adaptation following training (Armstrong and Pandolf 1988), and the training program employed in the present study may have been of insufficient intensity to produce significant heat adaptations. Henane et al. (1977) suggest that training must increase $\dot{V}O_2$ max by approximately 15% to induce a level of physiological strain that would result in significant adaptations to heat. One stimulus to heat adaptation may be metabolic hyperthermia activating thermoregulatory responses, such as changes in blood flow distribution (Rowell 1974), body fluid shifts (Harrison 1986), and hormonal mechanisms (Francesconi 1988). The degree of heat acclimation may therefore depend on both the magnitude and duration of hyperthermia in the body (Avellini et al. 1982, Fox et al. 1963). The subjects in the present study did increase their $T_{re}$ approximately 1.2°C over the hour of exercise (Table 5.5), though with a relatively short time at a maintained state of hyperthermia. Despite these objections, the decrease in $f_c$ and $\Delta T_{re}$ during the final exercise session (Table 5.5), and the significant increase in sweating rate (Table 5.7) and decrease in skin temperature (Figure 5.11) in the HST post-training, demonstrate that a moderate degree of heat adaptation was achieved.

An alternate explanation for the lack of a significant post-training effect on exercise-heat tolerance is the uncompensable hot-wet microenvironment created within the NBC clothing during exercise in the heat. The wearing of protective clothing impairs the dissipation of metabolic heat (Holmer 1995), resulting in a significant impairment of cardiorespiratory and thermoregulatory responses (Smolander et al. 1984) which may overwhelm any physiological manipulations. Comparing three methods of acclimating to subsequent exposure to dry heat, Shvartz et al. (1973) observed no improvement in exercise-heat tolerance following aerobic training, whereas either hot-dry or hot-wet heat acclimation produced substantial improvements in response. In hot-wet environments,

Strydom et al. (1966) and Strydom and Williams (1969) found only partial adaptation through physical conditioning compared with heat acclimation. In a group of untrained and non-fit individuals, Aoyagi et al. (1994) increased $\dot{V}O_2$ max by 16% following 8 weeks of aerobic training, yet did not observe any significant improvements in exercise-heat tolerance while wearing NBC clothing in the heat. However, the high rate of metabolic production used resulted in tolerance times of only about 50-60 min, with only the rare trial exceeding 70 min. McLellan (1993) has proposed that tolerance times in the severe uncompensable heat stress environment of NBC clothing are primarily independent of ambient conditions or any physiological manipulations at these rates of heat production, due to an insufficient amount of time for significant heat transfer through the clothing before the onset of exhaustion from other factors. The present study suggests that, while wearing NBC clothing, the adaptations with moderate endurance training may be insufficient to improve exercise-heat tolerance even at low metabolic rates.

It is well known that the decrease in plasma volume with hypohydration is associated with an increase in $f_c$, $T_{re}$, and ratings of perceived exertion (Candas et al. 1988, Sawka 1988). It was originally hypothesised that this short-term training model may have offered some protection during the hypohydrated trials because of an increase in plasma volume, such that exercise-heat tolerance would be improved. Our estimates of plasma volume revealed no change following the 10 days of training (Table 5.6), in contrast to a 12% increase reported by Green et al. (1991) with 10-12 d of training at 59% $\dot{V}O_2$ max. Given the lack of significant improvement during the euhydrated trials post-training, it is not surprising that hypohydration resulted in a severe impairment of exercise-heat tolerance regardless of training status.

Comparing the results of Section 5.1 with the present study, the magnitude of impairment with hypohydration was similar across the two fitness groups, with an approximate 18% decrease in tolerance time with hypohydration (Tables 5.3, 5.7). In addition, a non-significant trend of an approximate 10 min increase in tolerance time

during either euhydrated or hypohydrated trials existed in the more fit group of Study 1 compared with the low fit individuals in the present study, supporting the contention of Gisolfi and Robinson (1969) and Piwonka et al (1967) of a benefit from long-term fitness on responses to exercise in the heat.

One interesting and unexpected observation in the present study was that hydration status appeared to affect energy metabolism during exercise in the heat. The decrease in the RER during the hypohydrated trials was consistent in both the LF-Train and LF-Control groups pre- and post-manipulation (Table 5.7). Similar findings have been presented by Sawka et al. (1985), who observed a progressive decrease in RER with increasing severity of hypohydration during exercise in the heat but did not elaborate on any possible mechanisms. The finding of a decreased RER during hypohydration in the heat is different from that observed in thermoneutral environments, where hypohydration did not elicit any differences in $\dot{V}O_2$ or RER during submaximal exercise (Dengel et al. 1992, Neufer et al. 1989b). It is possible that the stress of heat exposure may interact with hypohydration to produce the downward shift in RER, as a significant decrease in RER during exercise in a hot, compared with a thermoneutral, environment was noted by Young et al. (1985). However, a recent study presented conflicting data, observing hyperglycemia and an increased RER during exercise in the heat, brought about by an elevation in plasma cortisol and catecholamines and an increased hepatic glucose release (Hargreaves et al. 1996).

The finding of a decrease in RER with hypohydration in the heat is surprising, and the underlying mechanism is not readily apparent. In the present study, no significant differences were observed in $\dot{V}O_2$ or $\dot{V}_E$ (Table 5.7). In contrast, $\dot{V}CO_2$ was significantly decreased during HY, suggesting that the decrease in RER was due to a shift in substrate utilisation toward an increased reliance on lipid metabolism (Holloszy 1973). The combination of a constant $\dot{V}_E$ despite a decreased $\dot{V}CO_2$ indicates that a slight hyperventilation is present relative to the rate of $CO_2$ production. This could be due to

either a corresponding and minor decrease in the arteriolar $PCO_2$ or else an increase in the ratio between the volume of respiratory dead space and tidal volume. Substrate availability does not appear to be impaired by hypohydration, as no differences have been observed in either the rate of glycogen resynthesis overnight (Neufer et al. 1991) or in the rate of muscle glycogen use during subsequent exercise (Neufer et al. 1989b) relative to a euhydrated state. Diet was controlled in the present study during the period following the dehydration protocol until the HST the next morning.

In summary, a two-week program of aerobic exercise produced a moderate, but significant, training effect and improvements in physiological strain during exercise in a thermoneutral environment in a group of untrained and relatively non-fit subjects. The short-term training program elicited an increase in maximal aerobic capacity, along with a significant decrease in heart rate and the rate of rise of rectal temperature. These adaptations were moderately successful in alleviating the physiological strain during light exercise in an uncompensable heat stress environment, with an increase in sweat rate and a decrease in the skin temperature, suggesting an increased sweating response and drive for evaporative heat loss. However, the adaptations due to training were ultimately unsuccessful in significantly prolonging exercise-heat tolerance in either a euhydrated or mildly hypohydrated state in an uncompensable heat stress environment.

## 5.3. Influence of heat acclimation, aerobic fitness, and hydration status on exercise-heat tolerance while wearing NBC protective clothing

### *5.3.1. Introduction*

The purpose of the present study was to determine the separate and combined effects of heat acclimation, aerobic fitness, and hydration status on exercise-heat tolerance during uncompensable heat stress. Heat acclimation, along with physical training, are two manipulations commonly used to prepare individuals for exercise-heat stress. Having found minimal benefits on exercise-heat tolerance from a longitudinal aerobic training program in Section 5.2, the focus of the present study was shifted to a cross-sectional analysis of the influence of initial aerobic fitness and activity levels. The original design of Section 5.1 did not call for subject selection criteria based on aerobic fitness or activity level, and the subjects were generally of moderate fitness. In the present study, the subjects were separated into either a low fit (LF) or high fit (HF) group based on their aerobic fitness and exercised in a hot environment wearing the NBC clothing while euhydrated and also while hypohydrated by ~2.5% of body mass. In particular, the following questions were investigated: 1) Do subjects with a high level of aerobic fitness derived from long-term training and habitual exercise have an improved tolerance during light exercise compared with individuals of moderate fitness and sedentary lifestyle? 2) When fluid replacement is provided, will heat acclimation produce similar benefits as observed previously without fluid replacement? 3) Is the magnitude of improvement with heat acclimation greater in subjects of moderate fitness? 4) Does mild hypohydration impair exercise-heat tolerance regardless of fitness or heat acclimation status?

### *5.3.2. Methods*

The experimental protocol was detailed in Chapter 4.

### 5.3.2.1. Experimental Design

Fifteen subjects participated and were assigned to either an aerobically high fit (HF-Acc, n=8) or an aerobically low fit (LF-Acc, n=7) group as defined in Chapter 4. The subjects in both groups performed a HST in both the EU and HY conditions prior to and following a heat acclimation (Acc) program, exercising at the light intensity (3.5 $km \cdot h^{-1}$, 0% grade) employed in Section 5.1. The Acc regimen consisted of two weeks (5 days per week) of daily 1 h treadmill sessions in a hot (40°C, 30% relative humidity) environment at an intensity that induced a $\Delta T_{re} \geq 1.5$°C during the initial session. The subjects wore the complete NBC ensemble except for the gas mask and canister, rubber gloves, and overboots. The overgarment was worn with the hood covering the head and the front completely zippered. The subjects were allowed the *ad libitum* consumption of one canteen (~1 L) of water at a temperature near 37°C. The order in which the hydration conditions were presented was randomised among subjects. To counterbalance order effects, the order of the hydration trials for each subject was reversed following the heat acclimation period. Fluid replacement at a rate of 200 mL each 15 min was provided in all trials.

### 5.3.2.2. Data Analyses

Data are presented as means (± standard deviation). A three-factor (period x hydration x time) repeated measures ANOVA was used to compare the rectal and skin temperature, heart rate, stroke volume, and cardiac output of the HF and LF subjects undergoing the short-term heat acclimation. A two-factor (period x hydration) repeated measures ANOVA was used to compare the responses of tolerance time, body mass changes, sweat rate, evaporative efficiency, plasma osmolality, metabolic rate, and respiratory exchange ratio. Following separate data analyses within either the HF or the LF group, a comparison was performed across the two groups to detect differences in response to exercise-heat tolerance as a result of long-term fitness. When a significant F-

ratio (corrected for the repeated measures factor) was obtained, a Newman-Keuls post-hoc analysis was performed to isolate differences among treatment means. For all statistical analyses, the 0.05 level of significance was used.

### *5.3.3. Results*

The physical characteristics of the subjects are presented in Table 5.8. The HF and LF subjects were similar in age and height. As expected, the HF subjects were distinguished from the LF subjects by their much higher $\dot{V}O_2$ max and a lower body fat content. Furthermore, the HF subjects were generally smaller than the LF group, with a lower body mass, a smaller surface area, and a larger surface area-to-mass ratio. The effects of the heat acclimation regimes on the physiological responses during the first and the final day of exposure are summarised in Tables 5.8 and 5.9. Acclimation did not induce an increase in aerobic capacity, with no changes in $\dot{V}O_2$ max following acclimation in either the HF or LF group (Table 5.8). LF subjects experienced a significantly lower heart rate during the final day of acclimation, along with a higher sweat rate (Table 5.9). Adaptation also occurred in HF individuals, with an increased sweat rate along with a decreased $T_{re}$ and heart rate in response to the heat acclimation exercise on day 10.

Tolerance to the HST was determined by time, ethically imposed physiological endpoints, or subject exhaustion. The reasons for trial termination are presented in Table 5.10. None of the trials approached the 4 h time limit. For the LF subjects, the large majority of the experimental trials was terminated due to exhaustion, as determined by the subject or the experimenter. Only 4 trials, all by the same subject, were terminated due to the subject reaching the $f_c$ ethical limit. In contrast, the HF subjects generally terminated trials due to reaching the ethically-imposed core temperature limit of 39.3°C. While voluntary exhaustion was often coincident with reaching the $T_{re}$ limit, the subjects in many cases felt that they were not completely exhausted and could have continued further.

Table 5.8 Physical characteristics of the subjects in the low fit (LF) and high fit (HF) groups. Pre=before and Post=after heat acclimation period.

| Group | Age (y) | Height (m) | Body Mass (kg) | Body Fat Content (%) | Surface Area ($m^2$) | Surface Area-to-Mass ($m^2 \cdot kg^{-1} \cdot 10^2$) | $\dot{V}O_2$ max ($mL \cdot kg^{-1} \cdot min^{-1}$) Pre | $\dot{V}O_2$ max ($mL \cdot kg^{-1} \cdot min^{-1}$) Post |
|---|---|---|---|---|---|---|---|---|
| LF (n=7) | 27.3 (6.9) | 1.78 (0.08) | 92.9++ (5.0) | 20.9++ (4.0) | 2.11++ (0.11) | 2.28++ (0.05) | 46.1++ (2.9) | 46.0++ (2.9) |
| HF (n=8) | 27.9 (6.5) | 1.77 (0.03) | 76.8 (4.3) | 11.5 (2.9) | 1.94 (0.14) | 2.56 (0.08) | 59.8 (2.8) | 59.5 (4.1) |

Values are means (S.D.). ++ Significantly different from the HF group

Table 5.9 Sweat rate (SR), change in rectal temperature ($\Delta T_{re}$) and final rectal temperature ($T_{re}$), final heart rate (HR), oxygen uptake ($\dot{V}O_2$) at 45 min, and respiratory exchange ratio (RER) in low (n=7) and high fit (n=8) subjects at the start and end of the heat acclimation period.

| | Low Fit | | High Fit | |
|---|---|---|---|---|
| | Day 1 | Day 10 | Day 1 | Day 10 |
| SR ($L \cdot h^{-1}$) | 1.35 (0.21) | 1.54+ (0.21) | 1.67 (0.37) | 1.92+ (0.48) |
| SR ($L \cdot °C^{-1}$) | 0.95 (0.24) | 1.02 (0.21) | 1.03 (0.28) | 1.28+ (0.42) |
| $\Delta T_{re}$ (°C) | 1.54 (0.34) | 1.54 (0.21) | 1.66 (0.23) | 1.56 (0.31) |
| Final $T_{re}$ (°C) | 38.70 (0.24) | 38.53 (0.15) | 38.75 (0.23) | 38.54+ (0.20) |
| Final HR ($b \cdot min^{-1}$) | 167.3 (15.1) | 159.9+ (19.0) | 156.1 (13.6) | 146.9+ (15.3) |
| $\dot{V}O_2$ ($mL \cdot kg^{-1} \cdot min^{-1}$) | 21.17 (1.32) | 20.96 (2.14) | 19.14 (2.46) | 18.92 (2.38) |
| RER | 0.90 (0.05) | 0.89 (0.05) | 0.88 (0.03) | 0.89 (0.03) |

Values are means (S.D.). + Significantly different from Day 1.

Table 5.10 Reasons for the termination of heat stress tests in low fit (LF, n=7) and high fit (HF, n=8) subjects.

| Group | Time | Core Temperature | Heart Rate | Volition |
|---|---|---|---|---|
| LF (n=7) | 0 | 0 | 4 | 24 |
| HF (n=8) | 0 | 24 | 0 | 8 |

The hydration schedule following the dehydration protocol was successful in either reinstating euhydration or in maintaining hypohydration overnight (Table 5.11). In the EU trials, body masses returned to baseline levels overnight, and were similar for all the EU trials. The hypohydration protocol resulted in a relative decrease in mass from 1.9 to 2.8% in the HF and LF groups compared with the Pre-EU trial. Hypohydration also increased serum osmolality and decreased plasma volume in both fitness groups. While no significant difference in the degree of hypohydration existed within or between groups, there was a trend towards a higher degree of hypohydration in the LF group.

Table 5.12 summarises some of the physiological responses to the HST. The metabolic rate, and therefore, the rate of internal heat production, was similar for all trials for both the HF and LF groups. In both groups, one major adaptation to the acclimation program was an increased sweat rate during the HST. However, due to the difficulty in water vapour transfer through the NBC ensemble, no significant changes in evaporation rate were observed. Heat acclimation did not significantly prolong tolerance times in either fitness group.

While heat acclimation had no effect on exercise-heat tolerance, hypohydration resulted in a significant degree of impairment regardless of acclimation status. While the endpoint $T_{re}$ was unaffected by hydration status within either group, the initial $T_{re}$ for LF was significantly higher prior to the hypohydrated trials. The RER was significantly lower during hypohydration trials in both the HF and LF groups. No significant differences were observed in $\dot{V}O_2$ max, indicating that the change in RER was due to a decreased $\dot{V}CO_2$. Overall, in both the HF and LF groups, hypohydration resulted in a significantly shorter tolerance time regardless of acclimation status.

The skin temperature, rectal temperature, and heart rate responses to the HST are presented in Figures 5.21 to 5.26, respectively. In the LF group, despite the cardiovascular and sweating adaptations observed over the course of the acclimation program, no differences in the $\overline{T}_{sk}$, $T_{re}$, and $f_c$ responses were evident following the two-week

Table 5.11 Absolute and relative (to Pre-EU) nude body mass, relative plasma volume (to Pre-EU) and serum osmolality in low fit (LF, n=7) and high fit(HF, n=8) groups prior to the heat stress tests. EU = euhydration, HY = hypohydration.

| | Low Fit | | | | High Fit | | | |
|---|---|---|---|---|---|---|---|---|
| | Pre-Acclimation | | Post-Acclimation | | Pre-Acclimation | | Post-Acclimation | |
| | EU | HY | EU | HY | EU | HY | EU | HY |
| Body Mass (kg) ++ | 93.74 (5.52) | 90.97* (5.60) | 93.61 (5.90) | 91.10* (5.47) | 76.80 (9.80) | 74.76* (9.81) | 76.79 (9.95) | 75.36* (10.05) |
| % Body Mass Loss | 0.00 (0.00) | -2.78* (0.92) | -0.14 (1.15) | -2.64* (0.61) | 0.00 (0.00) | -2.04* (0.52) | 0.00 (0.71) | -1.87* (0.75) |
| ΔPlasma Volume (%) | 0.00 (0.00) | -5.51* (3.36) | -3.54 (4.79) | -4.63* (5.37) | 0.00 (0.00) | -6.53* (5.22) | -1.65 (6.28) | -6.02* (6.00) |
| Osmolality (mosm·kg $H_2O^{-1}$) | 286.9 (3.8) | 292.7* (4.0) | 286.6 (3.7) | 292.6* (3.9) | 286.2 (2.6) | 292.5* (3.6) | 285.4 (1.8) | 291.4* (3.5) |

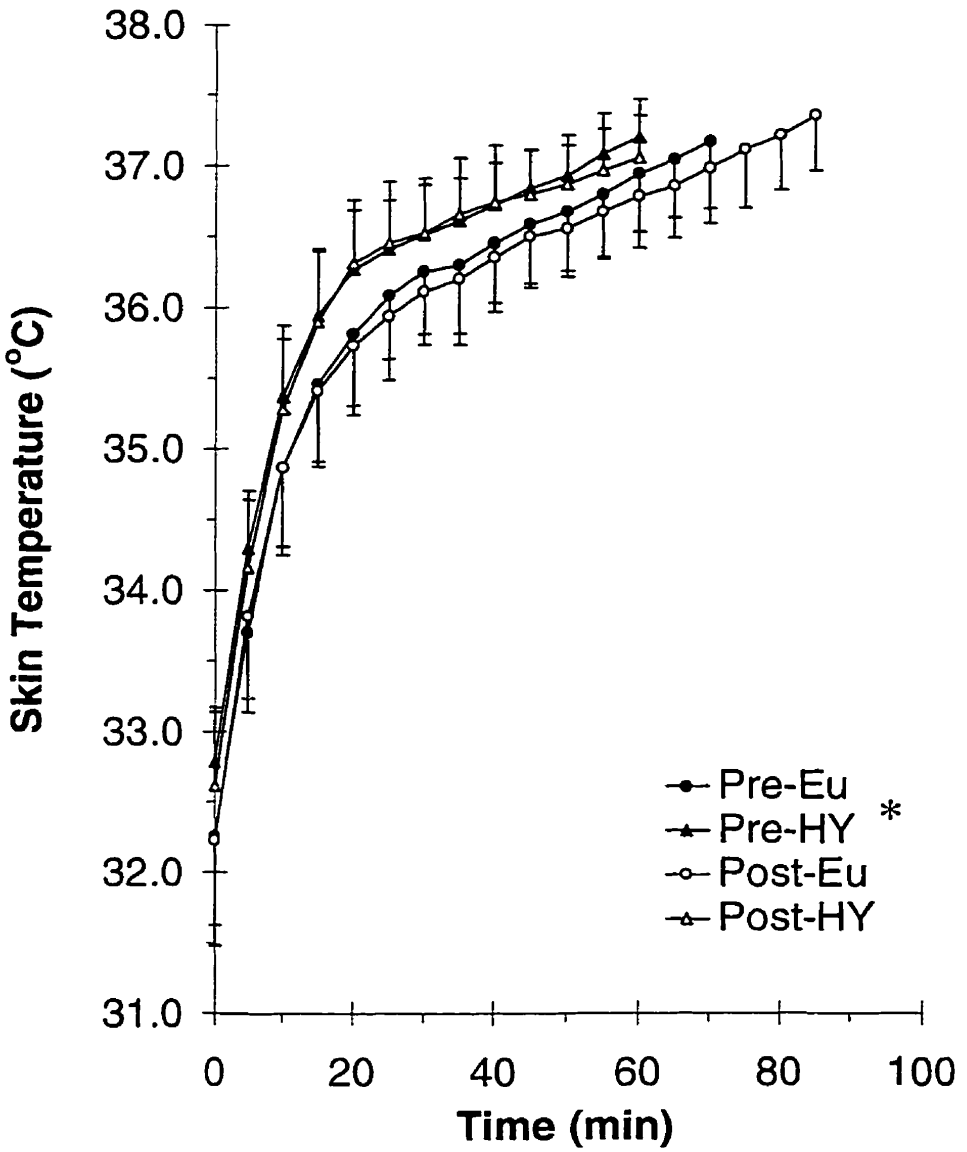
Values are means (S.D.). * Significant main effect of hydration. ++ Significant difference between LF and HF groups

Table 5.12 Sweating and evaporation rate, average metabolic rate, respiratory exchange ratio (RER), rates of ventilation ($\dot{V}_E$), oxygen uptake ($\dot{V}O_2$), and carbon dioxide production ($\dot{V}CO_2$), tolerance time, time required for rectal temperature ($T_{re}$) to increase by 1.0 (based on the $T_{re}$ from t=30-60 min), and initial and final $T_{re}$ in low fit (LF, n=7) and high fit (HF, n=8) fit groups. EU = euhydration, HY = hypohydration.

| | Low Fit | | | | High Fit | | | |
|---|---|---|---|---|---|---|---|---|
| | Pre-Acclimation | | Post-Acclimation | | Pre-Acclimation | | Post-Acclimation | |
| | EU | HY | EU | HY | EU | HY | EU | HY |
| Sweat Rate ($L \cdot h^{-1}$) | 1.04 (0.26) | 1.08 (0.19) | 1.33+ (0.40) | 1.27+ (0.27) | 1.30 (0.34) | 1.28 (0.35) | 1.43+ (0.39) | 1.42+ (0.35) |
| Evaporation Rate ($L \cdot h^{-1}$) | 0.33 (0.04) | 0.30 (0.04) | 0.34 (0.04) | 0.31 (0.06) | 0.38 (0.13) | 0.36 (0.08) | 0.40 (0.14) | 0.35 (0.08) |
| Average Metabolic Rate ($W \cdot m^{-2}$) | 181.6 (11.9) | 185.6 (13.2) | 185.1 (14.8) | 182.9 (12.2) | 175.1 (23.8) | 178.0 (24.0) | 175.8 (15.8) | 174.6 (21.2) |
| RER | 0.88 (0.06) | 0.82* (0.04) | 0.85 (0.04) | 0.82* (0.04) | 0.88 (0.05) | 0.82* (0.03) | 0.88 (0.04) | 0.84* (0.04) |
| $\dot{V}_E$ ($L \cdot min^{-1}$) | 24.4 (5.0) | 23.3 (4.0) | 24.1 (4.5) | 23.0 (4.5) | 21.2 (3.4) | 21.5 (2.8) | 21.6 (2.3) | 21.1 (2.3) |
| $\dot{V}O_2$ ($mL \cdot kg^{-1} \cdot min^{-1}$) | 11.07 (0.90) | 11.36 (0.82) | 11.36 (0.74) | 11.52 (0.87) | 11.30 (1.13) | 11.87 (1.07) | 11.16 (0.62) | 11.42 (0.79) |
| $\dot{V}CO_2$ ($L \cdot min^{-1}$) | 0.96 (0.11) | 0.91* (0.05) | 0.95 (0.11) | 0.91* (0.13) | 0.85 (0.11) | 0.81* (0.14) | 0.85 (0.08) | 0.81* (0.11) |
| Tolerance Time (min) ++ | 96.6 (19.6) | 78.3* (16.9) | 101.4 (11.4) | 80.6* (18.0) | 114.5 (27.4) | 100.9* (20.4) | 115.6 (18.4) | 110.5* (29.7) |
| $\Delta T_{re}$ = 1.0 °C (min) ++ | 44.2 (10.3) | 44.2 (8.7) | 49.0 (8.7) | 46.2 (9.0) | 41.6 (6.5) | 39.2 (8.8) | 45.5 (7.6) | 38.7 (9.9) |
| Initial $T_{re}$ (°C) ++ | 36.93 (0.27) | 37.26* (0.27) | 36.96 (0.28) | 37.24* (0.32) | 36.85 (0.22) | 36.99 (0.22) | 36.74 (0.19) | 36.83 (0.22) |
| Endpoint $T_{re}$ (°C) ++ | 38.77 (0.27) | 38.69 (0.30) | 38.79 (0.31) | 38.63 (0.31) | 39.15 (0.18) | 39.20 (0.08) | 39.14 (0.21) | 39.21 (0.06) |

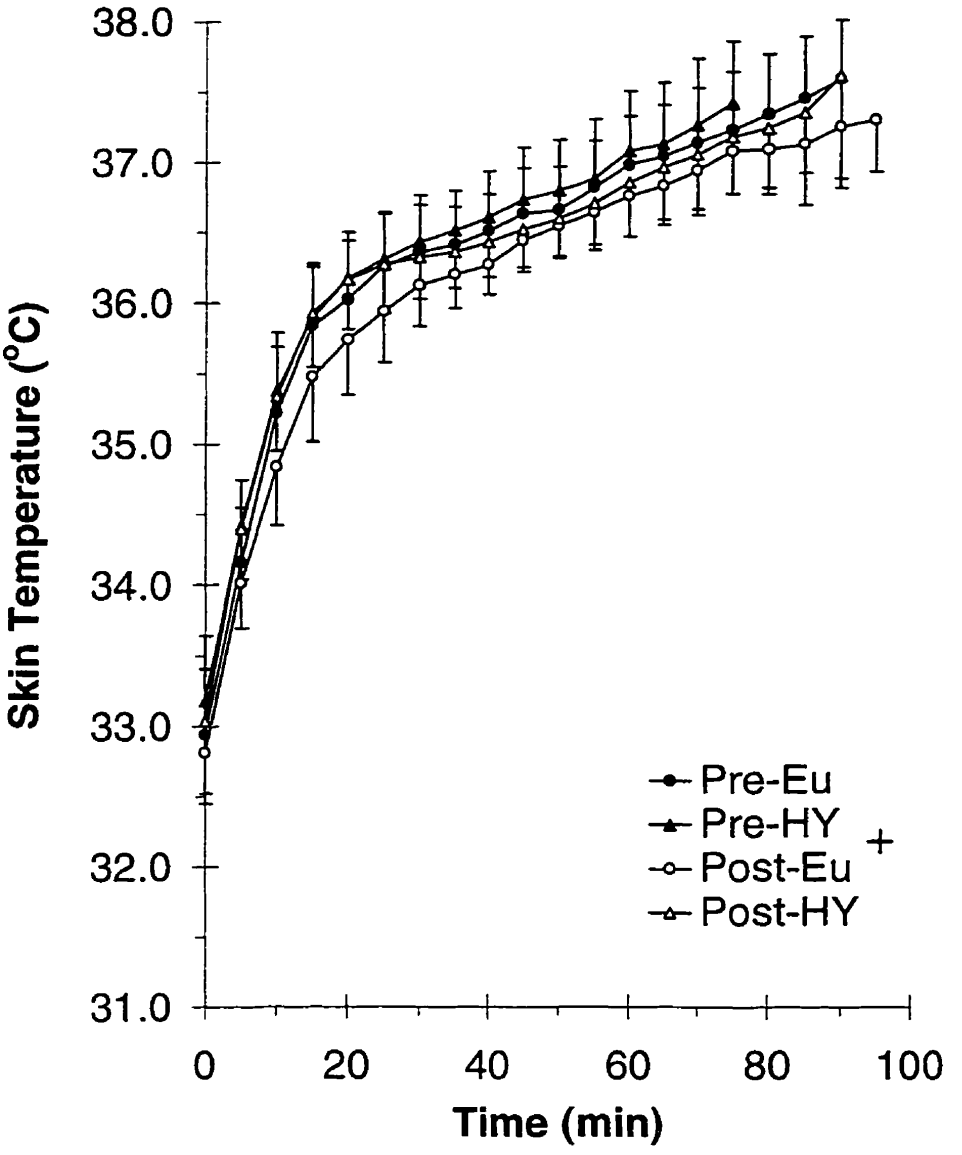
Values are means (SD). * Significant main effect of hydration. + Significant main effect of acclimation. ++ Significant difference between LF and HF groups

Figure 5.21 Mean skin temperature ( $\overline{T}_{sk}$ ) response to the heat stress test while either euhydrated (EU, circles) or hypohydrated (HY, triangles), before (filled symbols) and after (open symbols) two weeks of heat acclimation in low fit (n = 7) subjects.



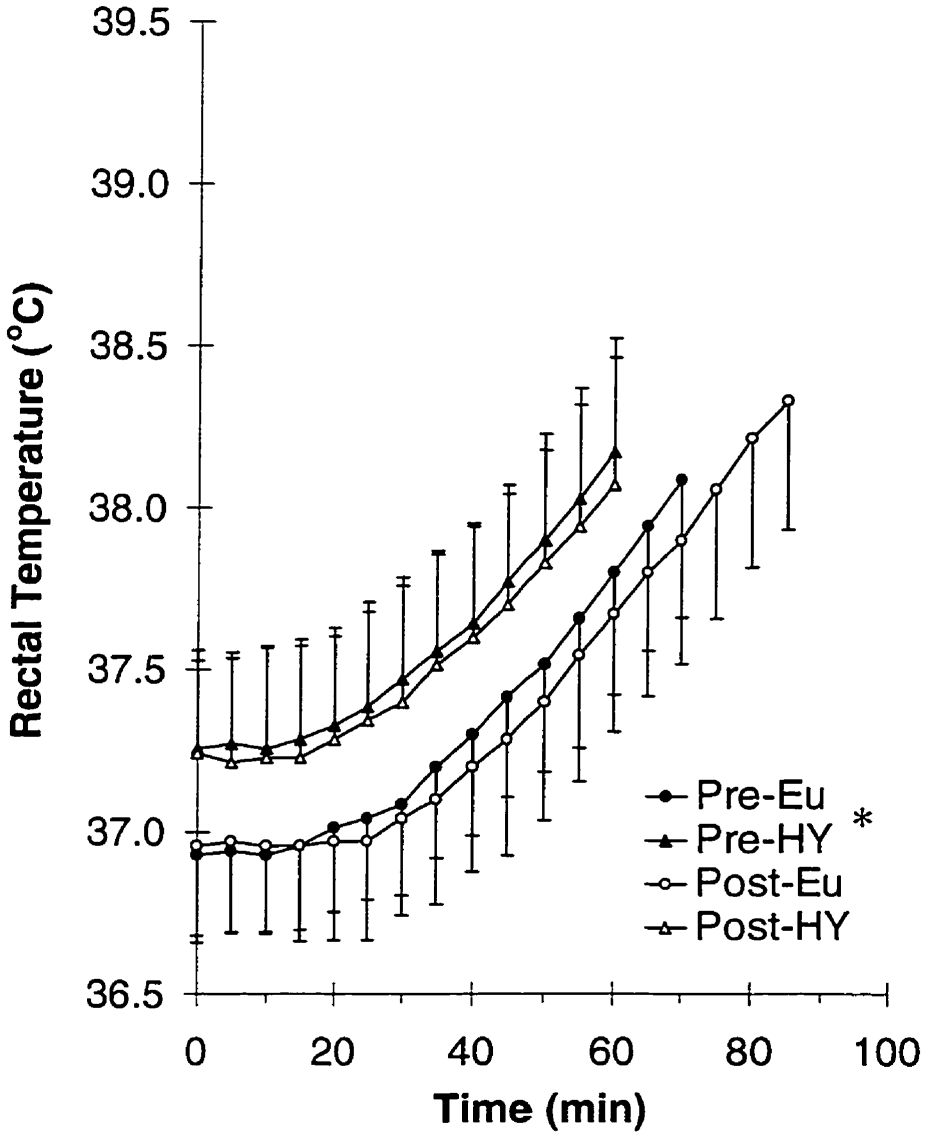

Values are mean ± S.D. * Significant main effect of hydration.

Figure 5.22 Mean skin temperature ( $\overline{T}_{sk}$ ) response to the heat stress test while either euhydrated (EU, circles) or hypohydrated (HY, triangles), before (filled symbols) and after (open symbols) two weeks of heat acclimation high fit (n = 8) subjects.




Values are mean ± S.D. + Significant main effect of heat acclimation.

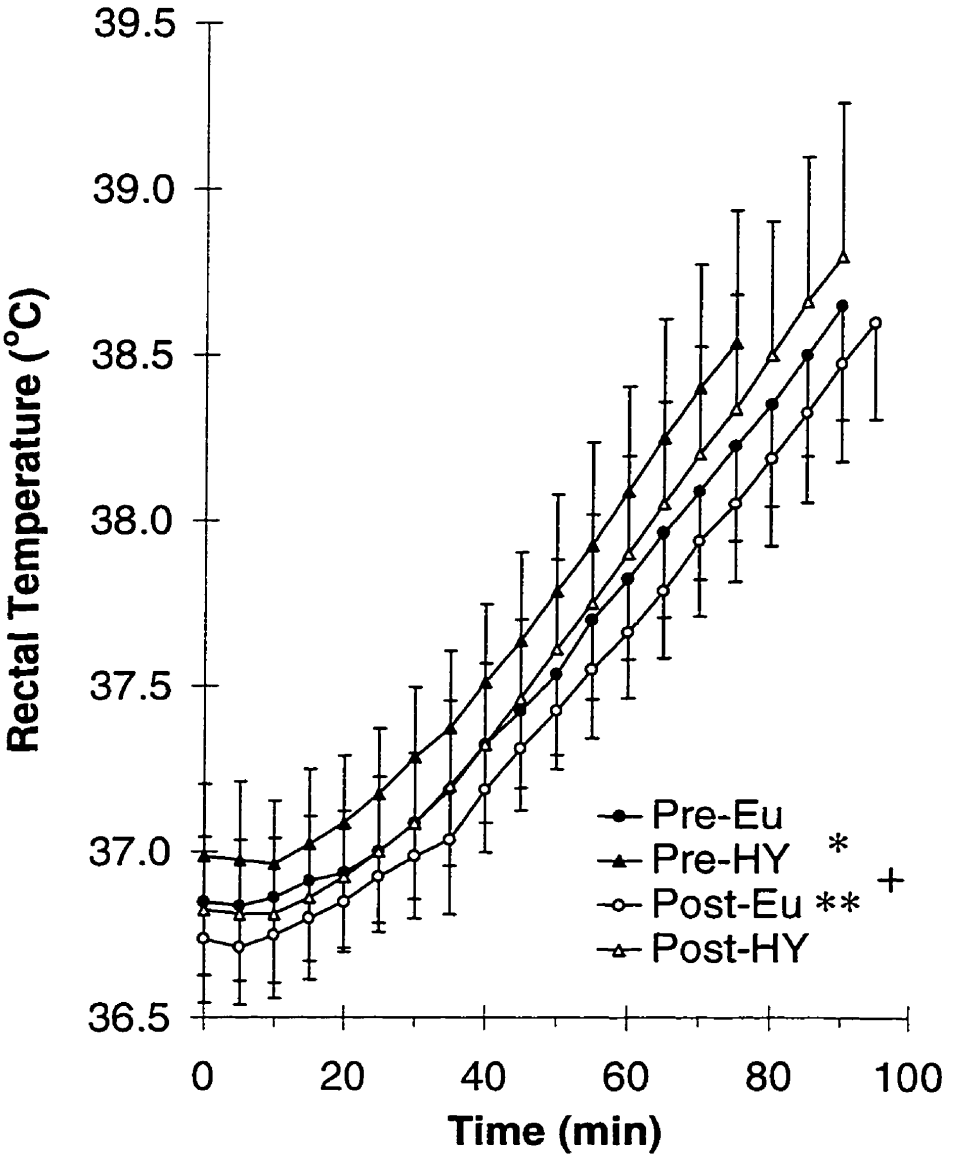
Figure 5.23 Rectal temperature ($T_{re}$) response to the heat stress test while either euhydrated (EU, circles) or hypohydrated (HY, triangles), before (filled symbols) and after (open symbols) two weeks of heat acclimation in low fit (n = 7) subjects.




Values are mean ± S.D. * Significant main effect of hydration.

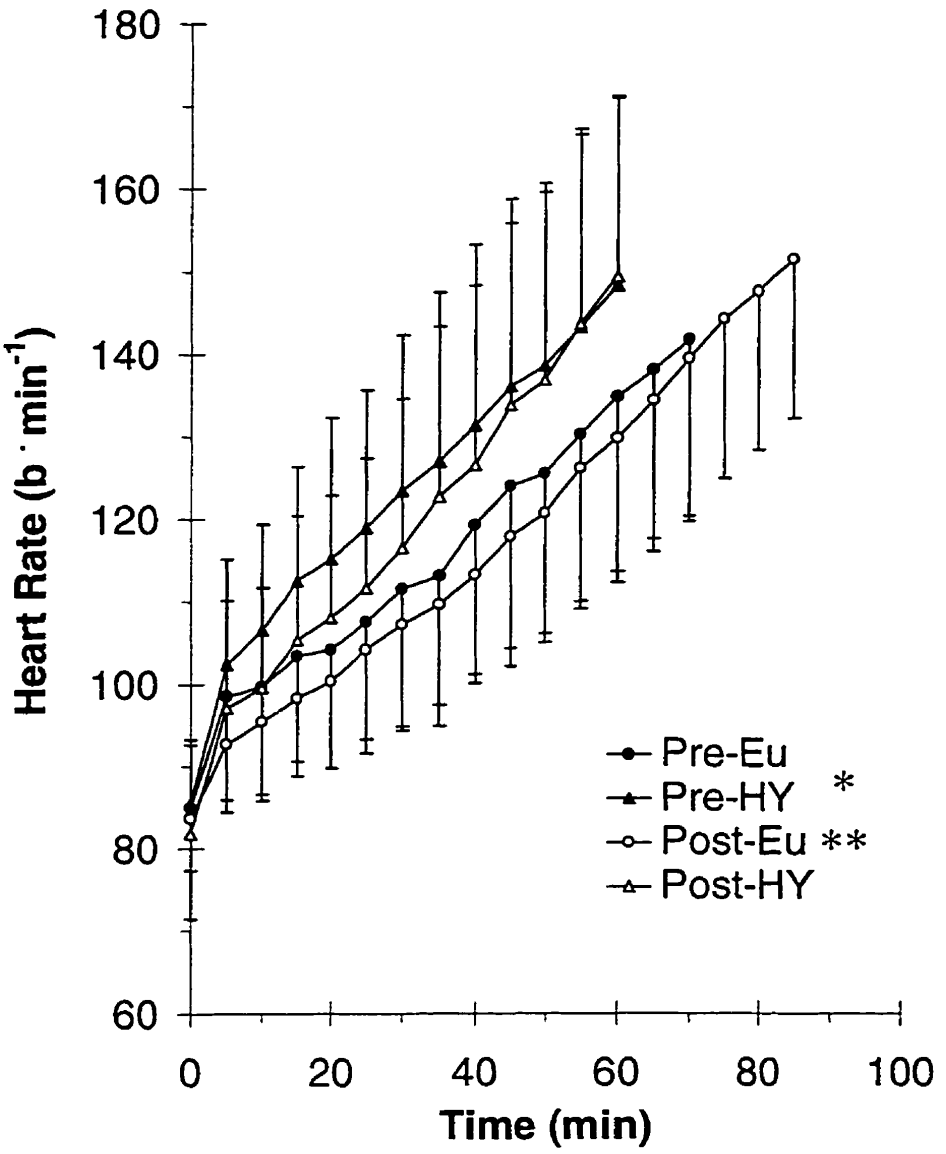
Figure 5.24 Rectal temperature ($T_{re}$) response to the heat stress test while either euhydrated (EU, circles) or hypohydrated (HY, triangles), before (filled symbols) and after (open symbols) two weeks of heat acclimation in high fit (n = 8) subjects.

+



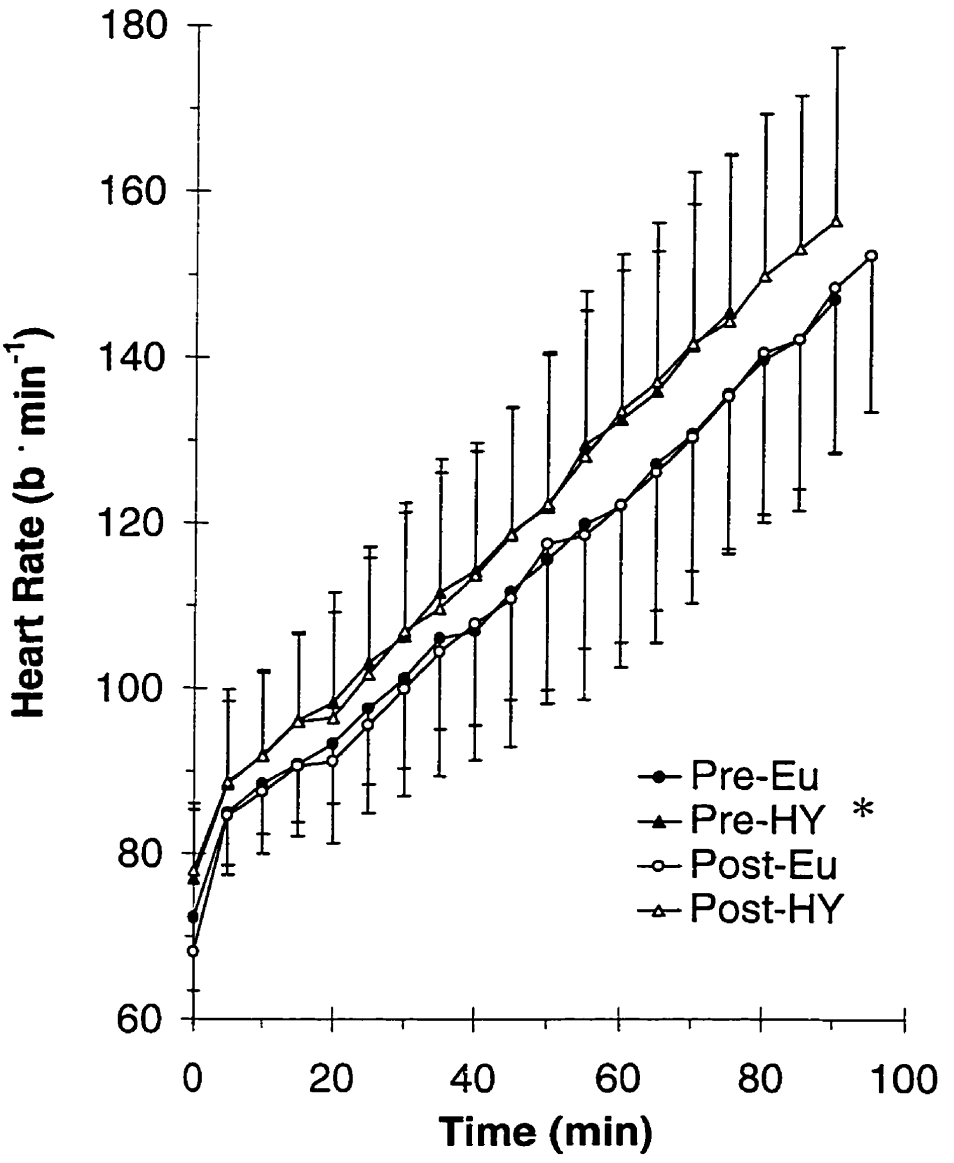

Values are mean ± S.D. * Significant main effect of hydration. ** Significant hydration x time interaction. + Significant main effect of heat acclimation.

Figure 5.25 Heart rate response to the heat stress test while either euhydrated (EU, circles) or hypohydrated (HY, triangles), before (filled symbols) and after (open symbols) two weeks of heat acclimation in low fit (n = 7) subjects.




Values are mean ± S.D. * Significant main effect of hydration. ** Significant hydration x time interaction.

Figure 5.26 Heart rate response to the heat stress test while either euhydrated (EU, circles) or hypohydrated (HY, triangles), before (filled symbols) and after (open symbols) two weeks of heat acclimation in high fit (n = 8) subjects.




Values are mean ± S.D. * Significant main effect of hydration.

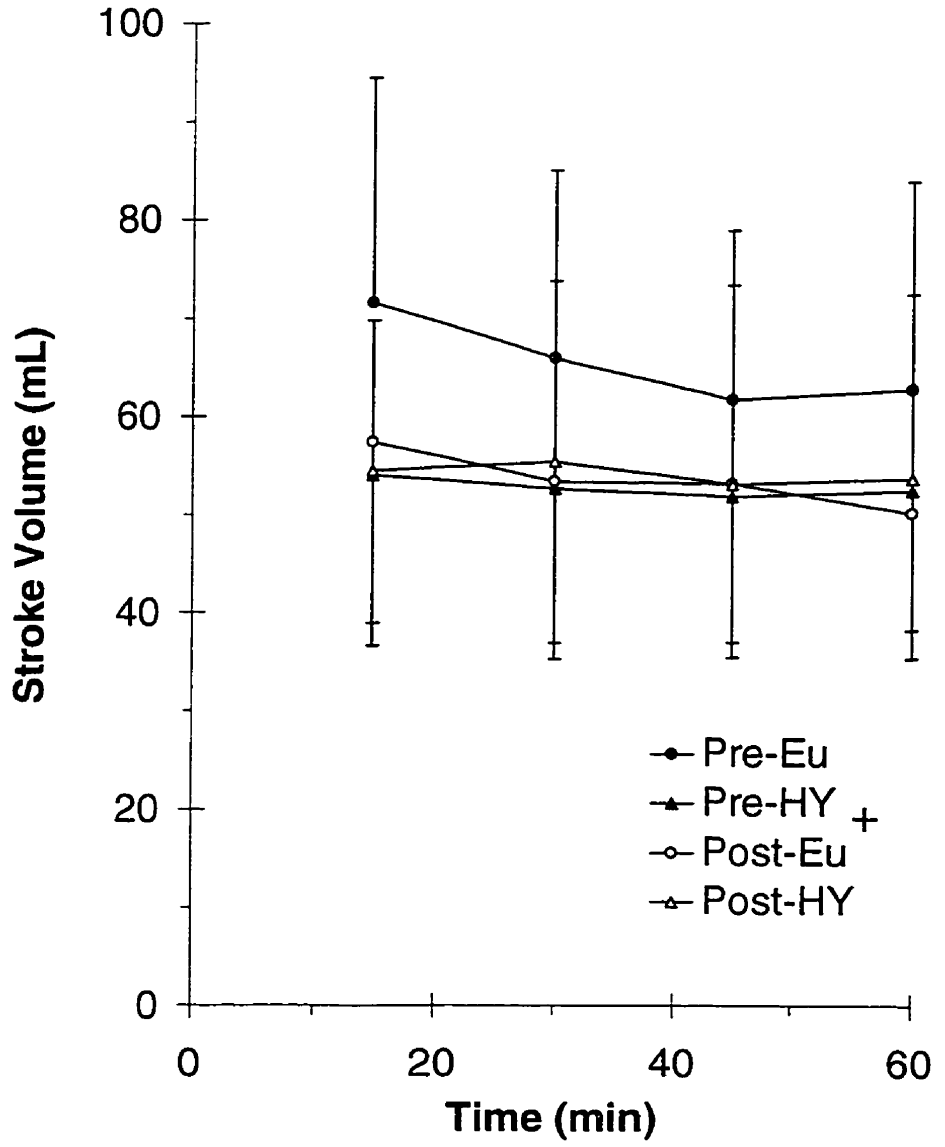
acclimation period. Interestingly, the acclimation period had a greater impact on the thermoregulatory responses to the HST in the subjects who were already highly fit aerobically, in that the acclimation program was successful in decreasing the thermoregulatory strain during the HST, with a main effect found for a lower $\overline{T}_{sk}$ (Figure 5.22) and $T_{re}$ (Figure 5.24) post-acclimation.

The hypohydration significantly affected the thermal and cardiovascular responses to the HST in both fitness groups. In both the LF and the HF groups, hypohydration resulted in a significantly higher $T_{re}$ (Figures 5.23-5.24). The heart rate response to the HST was influenced by hydration status in both fitness groups (Figures 5.25-5.26), with a significant elevation in heart rate in both the HF and LF subjects during HY. In the LF subjects, HY also elicited a hydration by time interaction for $f_c$. In the LF subjects, $\overline{T}_{sk}$ was significantly elevated during HY. In the fit subjects, hypohydration did not affect the overall $\overline{T}_{sk}$, but the initial increase in $\overline{T}_{sk}$ over the first 25 min was significantly slower during Post-EU compared with all other conditions.

Comparing the results between the HF and LF groups, the HF subjects had a greater $\Delta T_{re}$, as a result of both a significantly lower initial $T_{re}$ and a higher final $T_{re}$. The greater $\Delta T_{re}$ was the major contributing factor to an overall increased tolerance time in the fit subjects, as the rate of rise in $T_{re}$, calculated over the linear portion of the $T_{re}$ increase from 30-60 min, was greater in the HF than in the LF subjects. The rate of rise in $\overline{T}_{sk}$ was significantly lower overall in those with increased aerobic fitness. No significant between-group differences were observed in cardiovascular response, sweat rate, evaporation rate, metabolic rate, and RER during the HST.

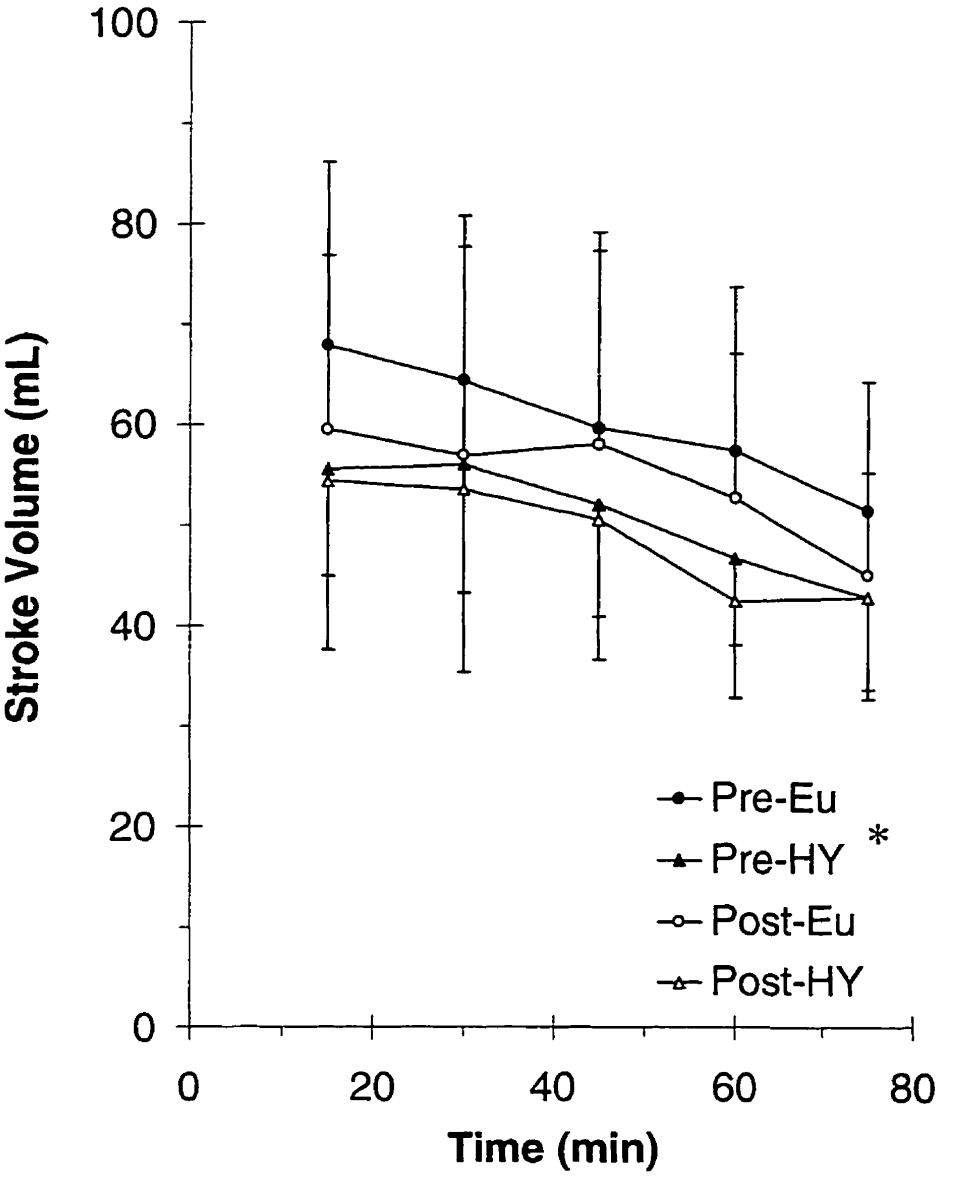
The stroke volume and cardiac output of the LF and HF groups during the HST are presented in Figures 5.27-5.30. In the LF subjects, stroke volume was relatively stable throughout the HST, but progressively decreased over the course of the HST in the HF subjects. Hypohydration resulted in a significant main effect of a lower overall stroke volume in the HF subjects. This decrease in stroke volume was compensated by an

Figure 5.27 Stroke volume response to the heat stress test while either euhydrated (EU, circles) or hypohydrated (HY, triangles), before (filled symbols) and after (open symbols) two weeks of heat acclimation in low fit (n = 7) subjects.



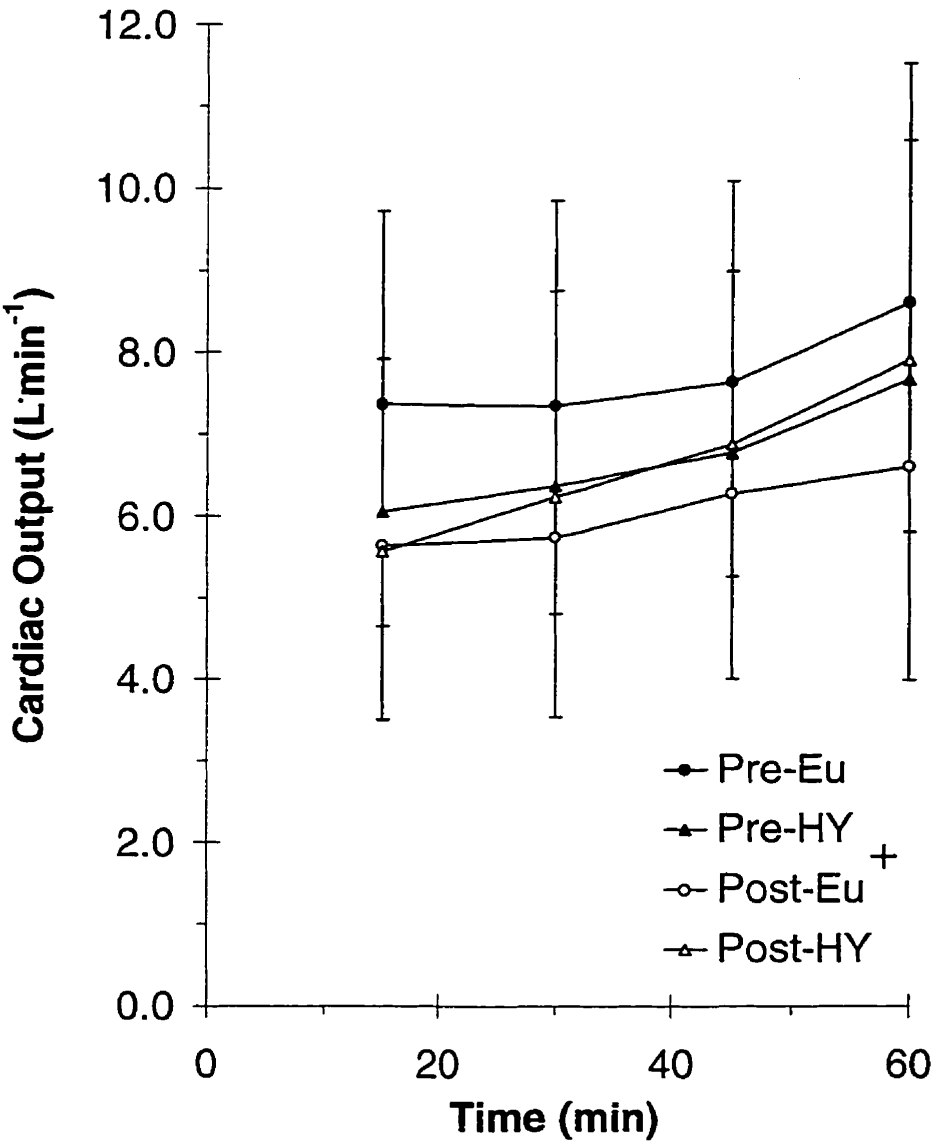

Values are mean ± S.D. + Significant main effect of the heat acclimation.

Figure 5.28 Stroke volume response to the heat stress test while either euhydrated (EU, circles) or hypohydrated (HY, triangles), before (filled symbols) and after (open symbols) two weeks of heat acclimation in high fit (n = 8) subjects.



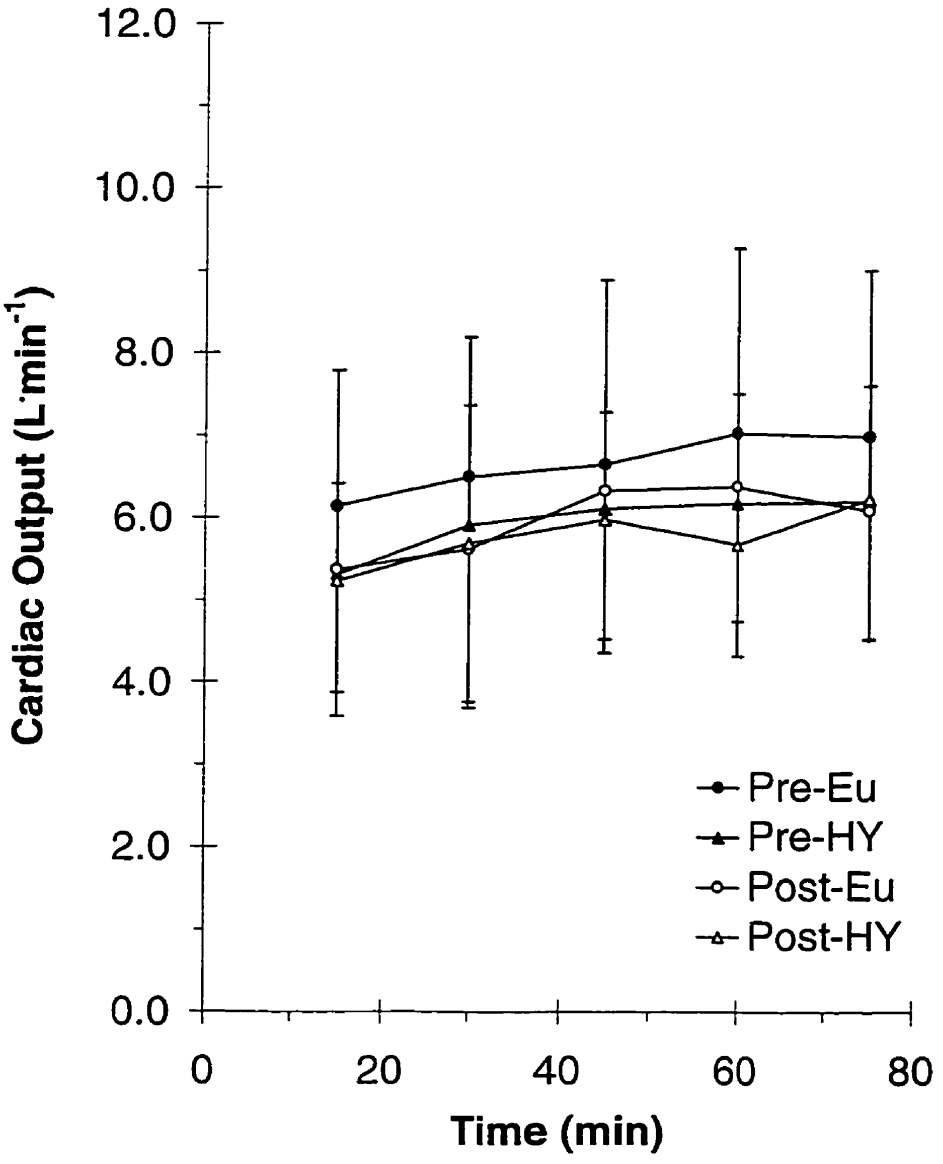

Values are mean ± S.D. * Significant main effect of hydration.

Figure 5.29 Cardiac output response to the heat stress test while either euhydrated (EU, circles) or hypohydrated (HY, triangles), before (filled symbols) and after (open symbols) two weeks of heat acclimation in low fit (n = 7) subjects.




Values are mean ± S.D. + Significant main effect of the heat acclimation.

Figure 5.30 Cardiac output response to the heat stress test while either euhydrated (EU, circles) or hypohydrated (HY, triangles), before (filled symbols) and after (open symbols) two weeks of heat acclimation in high fit (n = 8) subjects.




Values are mean ± S.D.

elevation in heart rate while hypohydrated, resulting in no overall effect of hydration status on cardiac output in the HF group. In the LF group, there was a non-significant trend towards an elevated stroke volume during the Pre-EU compared with the other three conditions, which in turn was responsible for a significantly higher overall stroke volume and cardiac output before the acclimation period.

### *5.3.4. Discussion*

The purpose of the present study was to determine the separate and combined effects of heat acclimation, aerobic fitness, and hydration status on tolerance during uncompensable heat stress. Each of these factors has been demonstrated to influence exercise-heat tolerance in an environment of compensable heat stress, where the $E_{max}$ exceeds or matches the $E_{req}$, resulting in a HSI of 1.0 or less (Armstrong and Pandolf 1988, Sawka 1988, Wenger 1988). However, even very light exercise in the heat while wearing clothing with limited permeability to water vapour will result in the inability to maintain thermal steady state (Givoni and Goldman 1972). The light exercise employed in the present study was sufficient to produce an uncompensable heat stress environment, with a HSI of approximately 2.5. Under these conditions, heat acclimation produced a moderate attenuation of thermal strain in individuals with high aerobic fitness but had no influence on tolerance during uncompensable heat stress. In contrast, individuals of low to moderate fitness experienced neither physiological benefits nor increased tolerance following heat acclimation. Long-term aerobic fitness resulted in a significant improvement in exercise-heat tolerance during uncompensable heat stress during each of the EU and HY trials both before and following heat acclimation.

Significant physiological adaptations occurred in both the HF and LF groups over the 10 d of heat exposure (Table 5.8), notably an increased sweat rate which was also evident during the full encapsulation conditions of the HST (Table 5.12). The increase in sweat rate in both fitness groups could be due to a centrally-mediated decrease in the

threshold temperature for vasodilation and sweating onset with acclimation (Nadel et al. 1974), an increased sensitivity of the sweating response through peripheral modifications (Taylor 1986), or an improved sweat gland resistance to skin wettedness and hidromeiosis (Candas et al. 1980). However, the increased sweat production did not result in an elevation in evaporative heat loss or a slowing of the rate of $T_{re}$ increase (Table 5.12), due to the limited permeability to water vapour of the NBC clothing. In uncompensable heat stress, therefore, the higher sweat rate from heat acclimation is a negative adaptation which, rather than enhancing evaporative heat loss and attenuating thermal strain, increases the rate of dehydration and physiological strain.

In the HF group, heat acclimation slightly but significantly decreased the initial core temperature by approximately 0.1°C regardless of hydration status (Table 5.12), resulting in a lower overall $T_{re}$ post-acclimation (Figure 5.24). This lowering of the initial $T_{re}$ would indicate a central mechanism of heat acclimation brought about by an alteration of the central thermoregulatory setpoint (Nadel et al. 1974), and may help to offset the increased rate of sweating and dehydration post-acclimation. Despite the reduction in thermal strain, tolerance times were unaffected by heat acclimation, with similar values pre- and post-acclimation (Table 5.12). The magnitude of the shift in $T_{re}$ setpoint was likely too small to affect tolerance times significantly. Given the roughly linear increase in $T_{re}$ after 30 min of the HST in the fit group of approximately 1.0°C every 40 min (Table 5.12), the slight but significant decrease in initial $T_{re}$ of 0.1°C, with no change in endpoint $T_{re}$, would result in a predicted minor increase in tolerance time of only 4 min. Nevertheless, the presence of a heat acclimation effect in the post-acclimation $T_{re}$ response of the HF group (Figure 5.24) supports the general consensus that even high levels of aerobic fitness produce only a partial heat adaptation (Shvartz et al. 1973a, Strydom et al. 1966).

The greater heat acclimation effect in the HF subjects is opposite to what has been observed in situations of compensable heat stress, where the HSI is ~1 or less (Cadarette

et al. 1984). Based on these reports, we initially hypothesised a greater response in the LF subjects due to a lower initial level of adaptation, and therefore, possibly a greater potential for heat acclimation. Several possibilities may account for the lack of improvement in the LF subjects in response to the HST, despite the two-week heat acclimation period. One explanation is that the LF subjects may not have been fully acclimated by our program. Highly fit individuals are predisposed to a more rapid acclimation to a severe heat stress (Piwonka and Robinson 1967), and a significant relationship between the level of aerobic fitness ($\dot{V}O_2$ max) and the number of days that are required for a plateau of response to acclimation has been reported in hot-dry environments (Pandolf et al. 1977). Using the regression equation presented in the latter study, the LF and HF subjects in the present study should have reached a plateau in response by 7 and 4.5 d, respectively. However, hot-wet acclimation may produce a slower plateau rate than observed under hot-dry conditions. In addition, full heat acclimation in a hot-humid environment required >1 h of exposure per day (Garden et al. 1966). Counteracting these arguments are the reports of significant adaptations while wearing clothing with limited water vapour permeability following 4-6 (Shvartz et al. 1973b) or 12 (McLellan and Aoyagi 1996) days. In the present study, a comparison of the responses to the heat acclimation on days 9 and 10 revealed a plateau in $f_c$, $T_{re}$, and SR in both the HF and LF subjects (unpublished data). In addition, as an increased sweating rate is one of the physiological adaptations with the slowest time course (Horvath and Shelley 1946), the elevated sweat rate in both groups (Table 5.9) suggest near-maximal adaptations in other physiological systems. We are therefore confident that the LF subjects achieved a near-complete state of heat acclimation. Another possible reason for the lack of heat acclimation effects on the LF subjects was that their significantly shorter tolerance time did not allow sufficient time for differences to be evident. However, at the metabolic rate used in this study, the average tolerance times of 95 (EU) and 80 (HY) min for the LF

subjects (Table 5.12) have been demonstrated to be long enough for physiological or environmental differences to be expressed (McLellan 1993).

Any benefits accruing from the heat acclimation in an uncompensable heat stress environment appear to be minor compared with the influences of fluid replacement and hydration status on exercise-heat tolerance. In the present study, the finding of no heat acclimation effects on exercise-heat tolerance in both fitness groups is in contrast to two previous studies in our laboratory, which demonstrated a decrease in physiological strain and an increase in tolerance time during exercise in the heat with NBC clothing following heat acclimation with (McLellan and Aoyagi 1996) or without (Aoyagi et al. 1995) NBC clothing. The disparity in the effects of acclimation may be due to the fact that, while subjects in the above studies were not provided any fluid replacement during the HST, water was provided to subjects in the present study at regular intervals. Fluid replacement has been demonstrated to reduce physiological strain during exercise in the heat (Candas et al. 1986), and specifically to decrease cardiovascular strain and increase tolerance time while exercising in the heat with NBC clothing (Section 5.1). As such, the fluid replacement may have extended the exercise-heat tolerance during the pre-acclimation HST to near the maximum possible given the uncompensable heat stress environment, thereby limiting the amount of additional improvement that could be observed with subsequent heat acclimation. If this is the case, it would underline the importance of fluid replacement in an uncompensable heat stress environment regardless of fitness or acclimation status.

The deleterious influence of hypohydration on exercise performance in thermoneutral and hot environments has been reviewed in detail elsewhere, and it is well known that the decrease in plasma volume with hypohydration is associated with an increase in $f_c$, $T_{re}$, and RPE (Candas et al. 1988, Sawka 1988). In a euhydrated state, heat acclimation significantly decreased final $T_{re}$ in environments with a HSI of approximately 1.0 (Sawka et al. 1983). However, in the same study, hypohydration significantly

increased thermal strain regardless of acclimation status, with similar final rectal temperatures both before and following the heat acclimation. The present study extends these finding to a more severe uncompensable heat stress environment. Compared with euhydration, hypohydration resulted in a decreased tolerance time regardless of fitness or acclimation status, with an elevated resting $T_{re}$ in both fitness groups and an increased rate of rise in $T_{re}$ in fit subjects. Therefore, not only fluid replacement during exercise, but also hydration status prior to exercise, appears to take precedence over acclimation status in determining physiological strain during uncompensable heat stress.

The present study used both the $\dot{V}O_2$ max and the level of regular physical activity to define subject inclusion into two distinct fitness groups. The inclusion of activity level as a selection criterion was prompted by the observation that $\dot{V}O_2$ max by itself was only moderately correlated with heat tolerance, and that the amount of regular physical activity may be a better indicator of the presence of training-induced adaptations to heat exposure (Avellini et al. 1982, Kielblock 1984, Pandolf et al. 1988). The LF subjects led a sedentary lifestyle and had a $\dot{V}O_2$ max under 50 $mL \cdot kg^{-1} \cdot min^{-1}$, while the HF subjects had a $\dot{V}O_2$ max in excess of 55 $mL \cdot kg^{-1} \cdot min^{-1}$ and engaged in a regular program of physical activity of approximately 7 h per week for the past year prior to participation in the study (Table 5.8).

Exercise-heat tolerance was improved by fitness regardless of hydration or heat acclimation status, with an average combined tolerance time of 110 and 88 min in the HF and LF, respectively (Table 5.12). These observations support the general consensus that an association exists between the level of cardiorespiratory fitness and improvements in the physiological responses to exercise in a hot environment (Armstrong and Pandolf 1988). At a higher exercise intensity in the heat while wearing NBC clothing, the trained subjects exhibited a trend towards an increase in tolerance time compared with the untrained subjects (Windle and Davies 1996). In the present study, the HF group had both a significantly lower initial and a higher endpoint $T_{re}$, resulting in a $\Delta T_{re}$ over the course of

the HST of 2.3°C in the HF group, which was significantly higher than the 1.6°C $\Delta T_{re}$ in the LF group (Table 5.12). Extrapolating an increase of the $\Delta T_{re}$ in the non-fit group by 0.7°C, and given their linear rate of $T_{re}$ increase of approximately 1.0°C per 45 min, the LF tolerance time would increase by 31.5 min. In contrast, decreasing the $\Delta T_{re}$ of the fit group by 0.7°C, the linear rate of $T_{re}$ increase of approximately 1.0°C per 40 min would decrease their tolerance times by 28 min. With an actual observed difference in overall tolerance time of approximately 22 min between the LF and HF groups (Table 5.12), it is evident that the difference in tolerance times between the two fitness groups could be largely accounted for by the difference in $\Delta T_{re}$.

The large majority of the HST with the HF subjects were terminated due to the individual reaching the ethically-imposed $T_{re}$ limit of 39.3°C, whereas LF subjects generally reached voluntary exhaustion at a $T_{re}$ well below 39.3°C (Table 5.10). All subjects underwent an initial familiarisation trial involving the complete experimental protocol, and no order or training effects were observed on tolerance time or endpoint $T_{re}$. HF individuals may be capable of subjectively tolerating a higher level of discomfort, skin wettedness, or physiological strain due to their regular program of physical activity. Alternatively, a given combination of $T_{re}$ and $\overline{T}_{sk}$ may have produced a greater degree of subjective discomfort in the LF subjects. While the thermal convergence of core and skin temperature may not be a reliable determinant of tolerance in an uncompensable heat stress environment (Nunneley et al. 1992), a particular skin-core temperature difference or the increased rate of $\overline{T}_{sk}$ increase could result in greater discomfort in the LF subjects. Fit subjects may also be better able to tolerate high levels of skin wettedness and the effects of hidromeiosis (Candas et al. 1980).

In summary, this study leads to the following observations regarding exercise-heat tolerance in an uncompensable heat stress environment: 1) High aerobic fitness from long-term training and habitual exercise is of significant benefit. 2) When fluid replacement is provided, heat acclimation does not provide significant benefit regardless of fitness status.

Fluid replacement may therefore be an effective substitute for a heat acclimation program. 3) The magnitude of the improvements in physiological strain with heat acclimation are greater in those with high aerobic fitness, but are still insufficient to improve exercise-heat tolerance. 4) A mild hypohydration of 2-3% of body mass results in a significant impairment regardless of fitness or heat acclimation status.

# 6. SUPPLEMENTARY ANALYSES

## 6.1. Comparison of short-term aerobic training and high maximal aerobic power on tolerance to uncompensable heat stress

### *6.1.1. Introduction*

In occupational settings, where workers may be required to work in hot environments with minimal preparation time or facilities to perform heat acclimation through heat exposures, the ability of short-term training programs to mimic the benefits of high aerobic fitness on exercise-heat tolerance is of interest. The purpose of the present analysis was to determine whether the aerobic training performed by the LF-Train group in Section 5.2 could improve the physiological responses to exercise-heat stress to a level comparable to those of the HF subjects in Section 5.3 prior to their undergoing heat acclimation. For the purposes of simplicity, the LF-Train group will be renamed LF-Pre and LF-Post for pre- and post-training, respectively, while the HF-Acc group will be renamed HF. Comparisons between LF-Pre and LF-Post were previously detailed in Section 5.2. and are included here only for completeness. It is hypothesised that, regardless of training or hydration status, the HF group will experience less thermoregulatory and cardiovascular strain and have prolonged tolerance compared to either the LF-Pre or LF-Post group.

### *6.1.2. Methods*

The experimental protocol was detailed in Chapter 4. The results of the individual experiments have been detailed in Sections 5.2 and 5.3.

### *6.1.3. Data Analyses*

Data are presented as means (± standard deviation). The EU and HY conditions were analysed separately. The physiological responses and tolerance to the HST of the HF group was compared with the LF-Pre and LF-Post groups using a between-group analysis of variance (ANOVA). When a significant F-ratio (corrected for the repeated measures factor) was obtained, a Newman-Keuls post-hoc analysis was performed to isolate

differences among treatment means. For all statistical analyses, the 0.05 level of significance was used.

### *6.1.4. Results*

#### 6.1.4.1. Subjects

Table 6.1 presents the physical characteristics of the test populations. The selection process was successful in producing two subject groups with a strong separation in maximal aerobic power. Short-term aerobic training was effective in significantly increasing $\dot{V}O_2$ max 6.5%, from 43 to 46 $ml \cdot kg^{-1} \cdot min^{-1}$. However, the $\dot{V}O_2$ max in the LF-post subjects remained significantly lower than that of the HF subjects. The LF subjects were also significantly heavier and had a higher body fat content than the HF subjects.

#### 6.1.4.2. Physiological Response of LF to the Training Program

The results of the short-term aerobic training program on the LF group are presented in Table 6.2. In addition to the increase in $\dot{V}O_2$ max, the two weeks of daily training resulted in an attenuation in the cardiovascular and thermal strain induced by the training exercise, as evidenced by a significantly lower heart rate and $\Delta T_{re}$ at the end of the hour of exercise. The training program did not result in an increased sweating response or a decrease in the respiratory exchange ratio during the exercise.

#### 6.1.4.3. Physiological Response to the HST

The recovery program following the dehydration protocol was successful in either restoring a euhydrated state or in maintaining hypohydration overnight for all groups (Table 6.3). In the EU trials, body mass returned to baseline levels overnight. The average morning body mass during the HY trials was about 2.0% less than during the EU trial, with no significant differences in relative hypohydration among groups. Compared to the

Table 6.1 Anthropometric measures of the subjects in the low fit (LF) and high fit (HF) groups, with age, height, body mass and fat content, surface area and surface area-to-mass ratio, and maximal aerobic power ($\dot{V}O_2$ max) before (Pre) and following (Post) the training period.

| Group | Age (y) | Height (m) | Body Mass (kg) | Body Fat Content (%) | Surface Area ($m^2$) | Surface Area-to-Mass ($m^2 \cdot kg^{-1} \cdot 10^2$) | $\dot{V}O_2$ max ($mL \cdot kg^{-1} \cdot min^{-1}$) Pre | Post |
|---|---|---|---|---|---|---|---|---|
| LF (n=8) | 30.3 (6.7) | 1.77 (0.05) | 84.7 (12.6) | 19.2$^{++}$ (3.9) | 2.02 (0.14) | 2.41 (0.20) | 43.2$^{++}$ (2.7) | 46.0$^{++}$ (2.5)$^{+}$ |
| HF (n=8) | 27.9 (6.5) | 1.77 (0.03) | 76.8 (4.3) | 11.5 (2.9) | 1.94 (0.14) | 2.56 (0.08) | 59.8 (7.4) | — |

Values are means (SD)

+ Significant effect of aerobic training

++ Significantly different from the HF group

Table 6.2 Sweat rate (SR), change in rectal temperature ($\Delta T_{re}$) and final rectal temperature ($T_{re}$), final heart rate (HR), oxygen uptake ($\dot{V}O_2$), and respiratory exchange ratio (RER) in the low fit group (LF, n=8) at the start and end of the training periods.

| | Low Fit | |
|---|---|---|
| | Day 1 | Day 12 |
| SR (L) | 0.73<br>(0.18) | 0.78<br>(0.22) |
| SR ($L \cdot °C^{-1}$) | 0.64<br>(0.30) | 0.74<br>(0.19) |
| $\Delta T_{re}$ (°C) | 1.26<br>(0.44) | 1.09+<br>(0.31) |
| $T_{re}$ end (°C) | 38.38<br>(0.25) | 38.21<br>(0.26) |
| HR end ($b \cdot min^{-1}$) | 146.9<br>(12.9) | 137.3+<br>(5.8) |
| $\dot{V}O_2$ ($mL \cdot kg^{-1} \cdot min^{-1}$) | 28.9<br>(3.4) | 29.1<br>(2.4) |
| RER | 0.89<br>(0.02) | 0.87<br>(0.03) |

Values are means (SD)

+ Significantly different from Day 1

Table 6.3 Absolute and relative (to euhydration trial) nude body mass and serum osmolality prior to the heat stress tests in the low fit (LF, n=8) and high fit (HF, n=8) groups. EU=euhydration, HY=hypohydration.

| | Low Fit | | | | High Fit | |
|---|---|---|---|---|---|---|
| | Pre-Train | | Post-Train | | | |
| | EU | HY | EU | HY | EU | HY |
| Body Mass (kg) | 84.15 (12.69)++ | 82.15* (12.37)++ | 84.00 (12.80)++ | 82.09* (12.41)++ | 76.80 (9.80) | 74.76* (9.81) |
| % Body Mass Loss | 0.00 (0.00) | -2.00* (0.41) | -0.15 (0.51) | -2.06* (0.53) | 0.00 (0.00) | -2.04* (0.52) |
| Osmolality (mosm·kg $H_2O^{-1}$) | 287.3 (5.6) | 293.2* (3.4) | 286.2 (2.5) | 291.8* (3.3) | 286.2 (2.6) | 292.5* (3.6) |

Values are means (SD)

* Significant main effect of hydration

++ Significant difference from HF

EU trials, serum osmolality was elevated prior to HY trials for all groups. These results all indicate that the subjects were significantly hypohydrated prior to HY trials.

Table 6.4 summarises some of the physiological responses to the HST. The metabolic rate, and therefore, the rate of heat production, was similar for all trials for the LF-Pre, LF-Post, and HF groups. Though no change in sweating response occurred in the LF while training in a thermoneutral environment, the more extreme heat environment of the HST was sufficient to expose a difference in the drive for heat dissipation due to training. One major change as a result of a two-week aerobic training program was an increased sweat rate during the HST . However, due to the limited water vapour transfer through the NBC ensemble, these adaptations did not result in a significant change in evaporation rate. Overall, the HF subjects maintained a significantly higher SR than the LF subjects either before or after training, though this did not translate into a higher evaporation rate.

High levels of aerobic fitness appear to impart some degree of protection during exercise in an uncompensable heat stress environment, with significantly lower levels of physiological strain in the HF compared to the LF-Pre subjects. Prior to undergoing the training program, in either an euhydrated or hypohydrated state, $T_{re}$, $f_c$, and $\overline{T}_{sk}$ were all higher in the LF-Pre than in the HF individuals (Figures 6.1-6). In the LF subjects, the primary effect of training was a significantly lower $\overline{T}_{sk}$, which may have been brought about by the increased sweating and skin wettedness following training. Training resulted in a slight, but non-significant, decrease in $T_{re}$ during either EU or HY, and also a non-significant decrease in $f_c$. Though minor, these changes in physiological responses following training were sufficient to eliminate the significant differences observed between the LF-Pre and HF subjects during the HST. The $T_{re}$, $f_c$, and $\overline{T}_{sk}$ in either the EU or HY trials were not significantly different between the LF-Post and HF groups.

Table 6.4 Sweating and evaporation rate, average metabolic rate, respiratory exchange ratio (RER), rates of ventilation ($\dot{V}_E$), oxygen uptake ($\dot{V}O_2$), and carbon dioxide production ($\dot{V}CO_2$), tolerance time, time required for rectal temperature ($T_{re}$) to increase by 1.0 (based on the $T_{re}$ from t=30 - 60 min), and initial and final $T_{re}$ for the low fit (LF, n=8) and high fit (HF, n=8) groups. EU=euhydration, HY=hypohydration.

| | Low Fit | | | | High Fit | |
|---|---|---|---|---|---|---|
| | Pre-Train | | Post-Train | | | |
| | EU | HY | EU | HY | EU | HY |
| Sweat Rate ($L \cdot h^{-1}$) | 0.87 (0.27)++ | 0.92 (0.26)++ | 1.00+ (0.29)++ | 1.03+ (0.25)++ | 1.30 (0.34) | 1.28 (0.35) |
| Evaporation Rate ($L \cdot h^{-1}$) | 0.31 (0.07) | 0.26 (0.06) | 0.29 (0.05) | 0.29 (0.04) | 0.38 (0.13) | 0.36 (0.08) |
| Average Metabolic Rate ($W \cdot m^{-2}$) | 173.9 (8.3) | 175.0 (8.3) | 171.4 (12.8) | 168.8 (12.2) | 175.1 (23.8) | 178.0 (24.0) |
| $\Delta T_{re}$ = 1 °C (min) | 47.7++ (6.1) | 46.7++ (12.5) | 50.4++ (8.3) | 45.5++ (10.2) | 41.6 (6.5) | 39.2 (8.8) |
| Initial $T_{re}$ (°C) | 37.08 (0.24)++ | 37.18* (0.34)++ | 36.93 (0.34) | 37.20* (0.35)++ | 36.85 (0.22) | 36.99 (0.22) |
| Endpoint Tre (°C) | 38.70 (0.37)++ | 38.63 (0.35)++ | 38.61 (0.25)++ | 38.60 (0.42)++ | 39.15 (0.18) | 39.20 (0.08) |
| Tolerance Time (min) | 93.1 (18.9)++ | 75.8 (14.4)++ | 94.0 (16.2)++ | 80.3 (11.7)++ | 117.6 (26.1) | 99.8 (19.8) |

Values are means (SD)

* Significant main effect of hydration

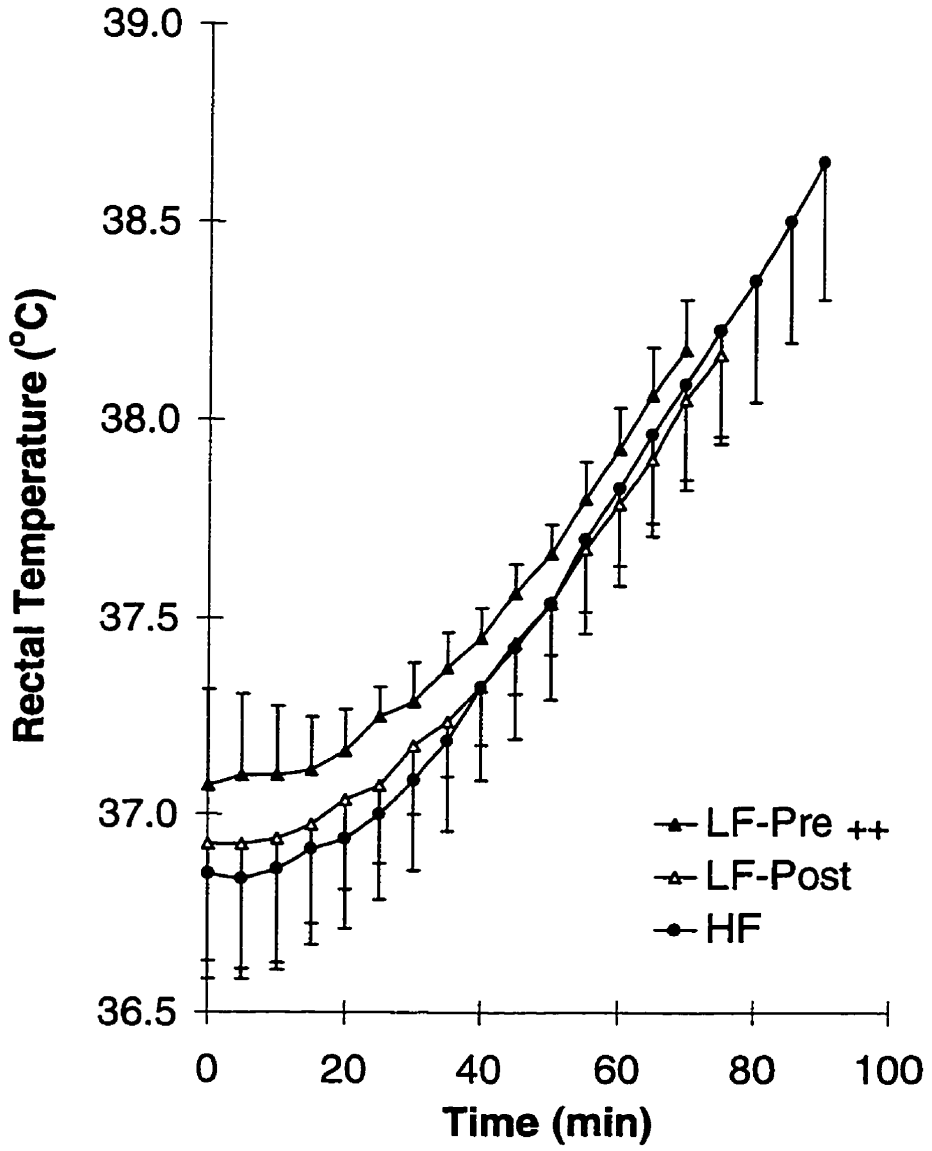
+ Significant main effect of training

++ Significant difference between LF and HF groups

Table 6.5 Reasons for the termination of heat stress tests in low fit (LF, n=8) and high fit (HF, n=8)) subjects.

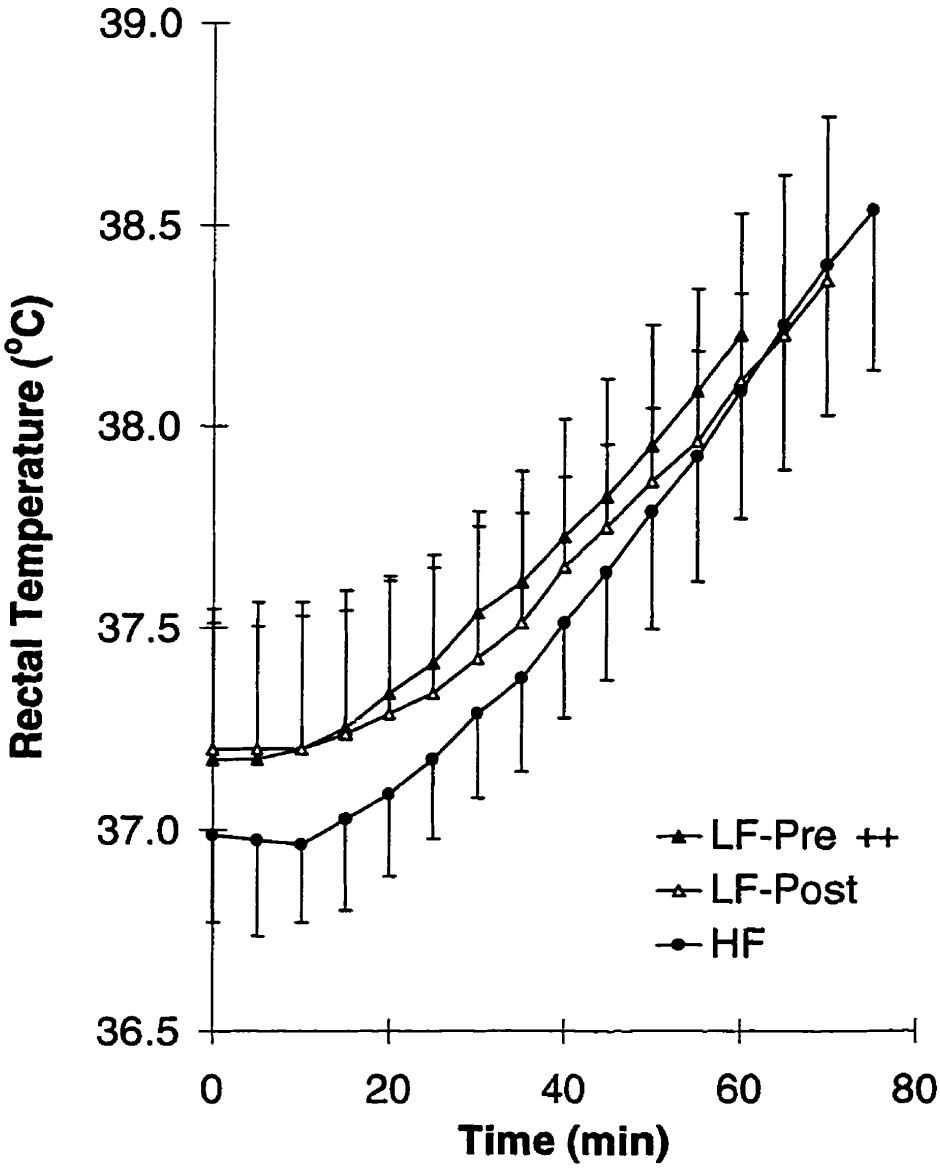
| Group | Time | $T_{re}$ | Heart Rate | Volition |
|---|---|---|---|---|
| LF (n=8) | 0 | 2 | 0 | 30 |
| HF (n=8) | 0 | 12 | 0 | 4 |

Figure 6.1 Rectal temperature responses of low fit subjects (n=8) pre- (LF-Pre) and post-training (LF-Post), and high fit (HF, n=8) subjects during the euhydrated heat stress tests.



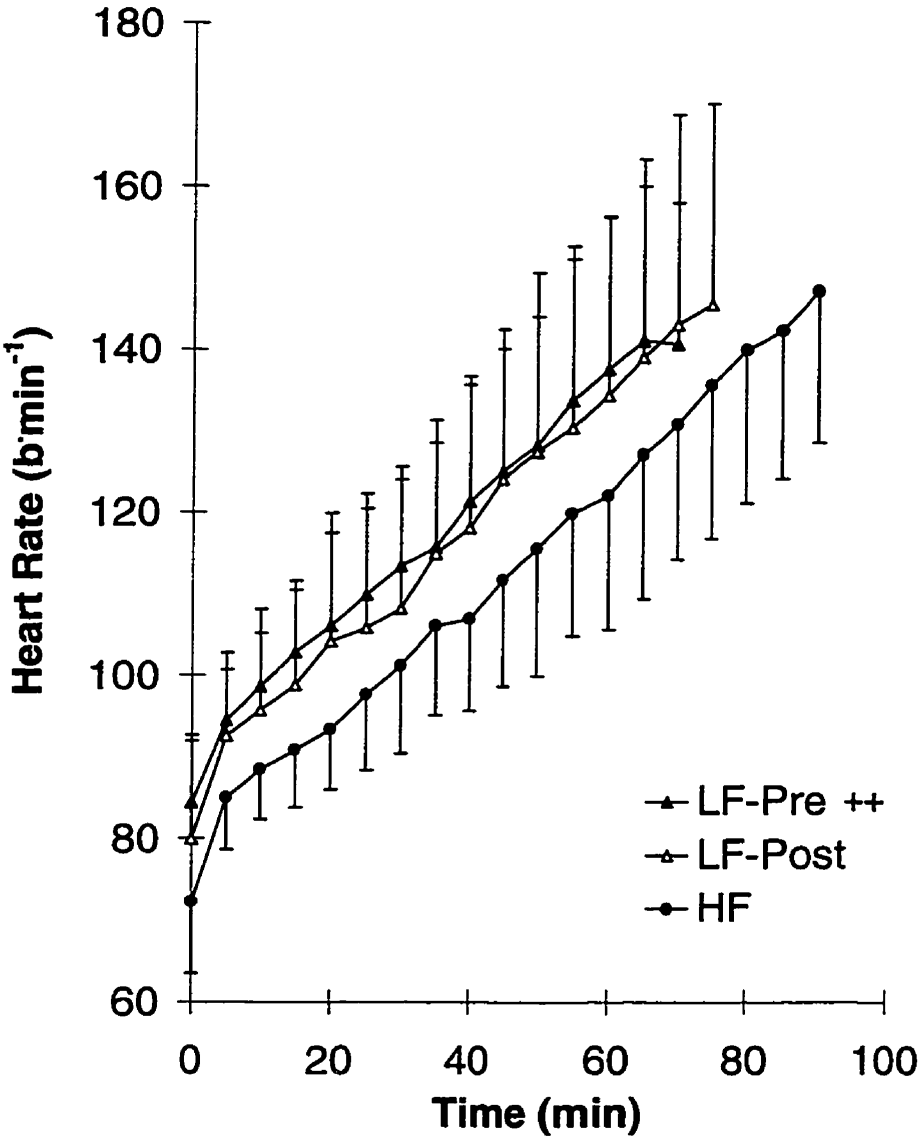

Values are means ± S.D. ++ Significantly different from HF

Figure 6.2 Rectal temperature responses of low fit subjects (n=8) pre- (LF-Pre) and post-training (LF-Post), and high fit (HF, n=8) subjects during the hypohydrated heat stress tests.



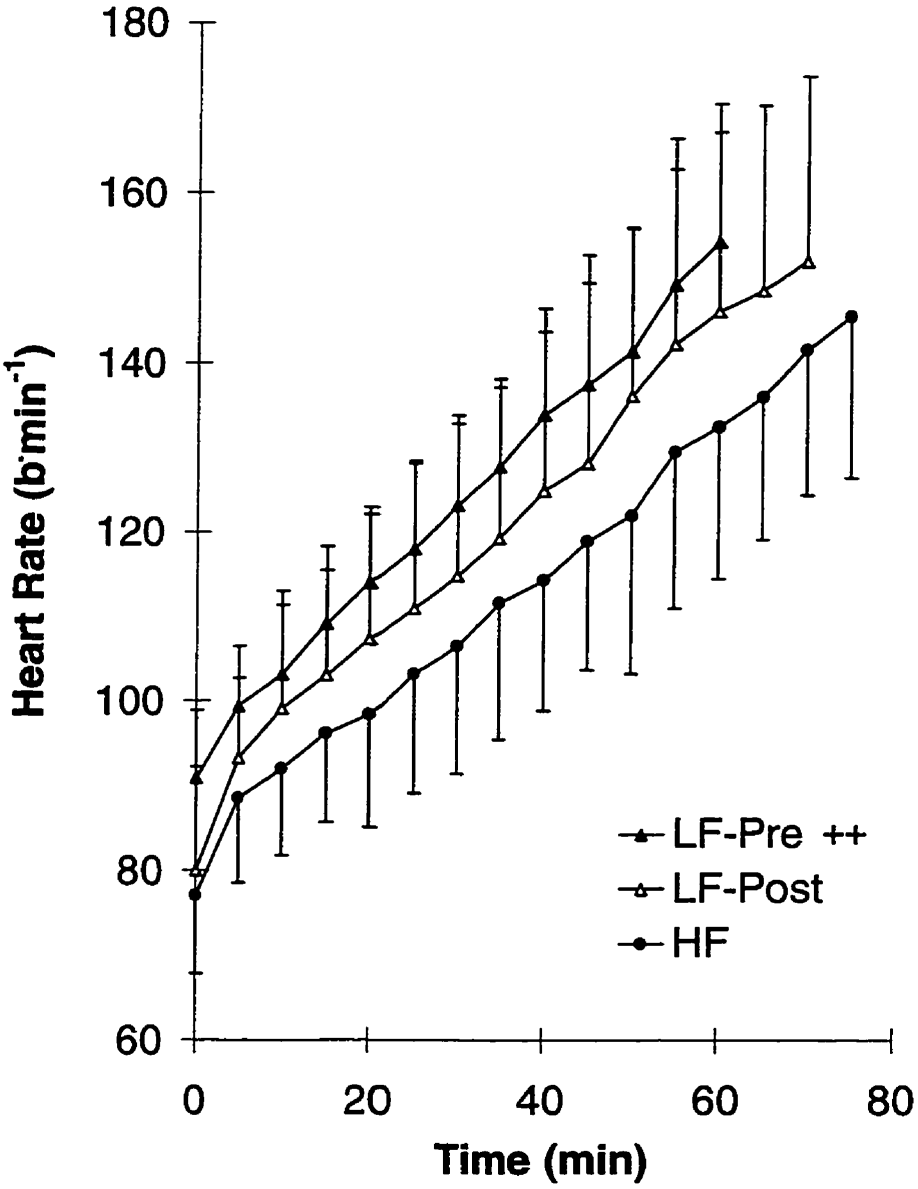

Values are means ± S.D. ++ Significantly different from HF

Figure 6.3 Heart rate responses of low fit subjects (n=8) pre- (LF-Pre) and post-training (LF-Post), and high fit (HF, n=8) subjects during the euhydrated heat stress tests.



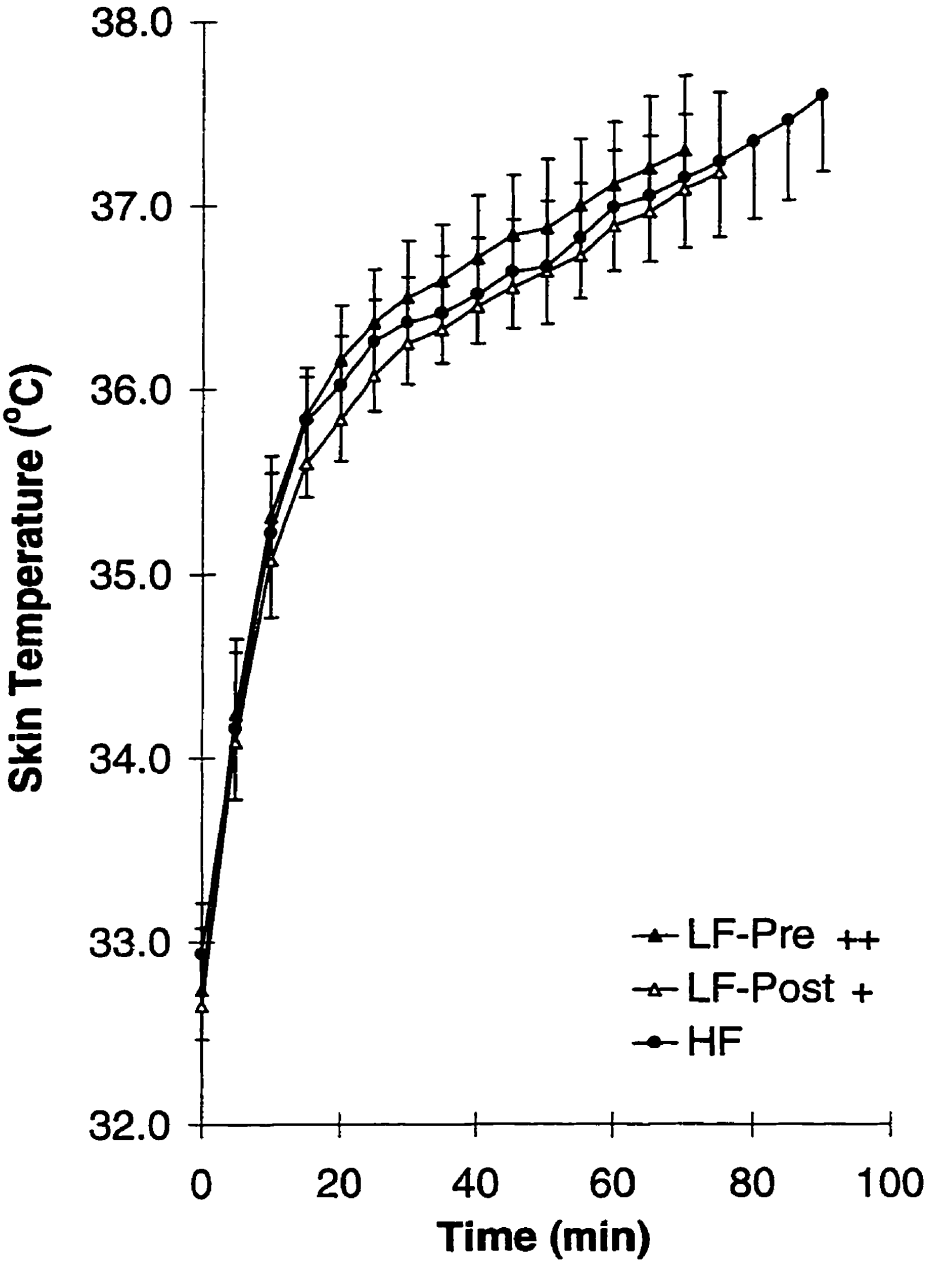

Values are means ± S.D. ++ Significantly different from HF

Figure 6.4 Heart rate responses of low fit subjects (n=8) pre- (LF-Pre) and post-training (LF-Post), and high fit (HF, n=8) subjects during the hypohydrated heat stress tests.



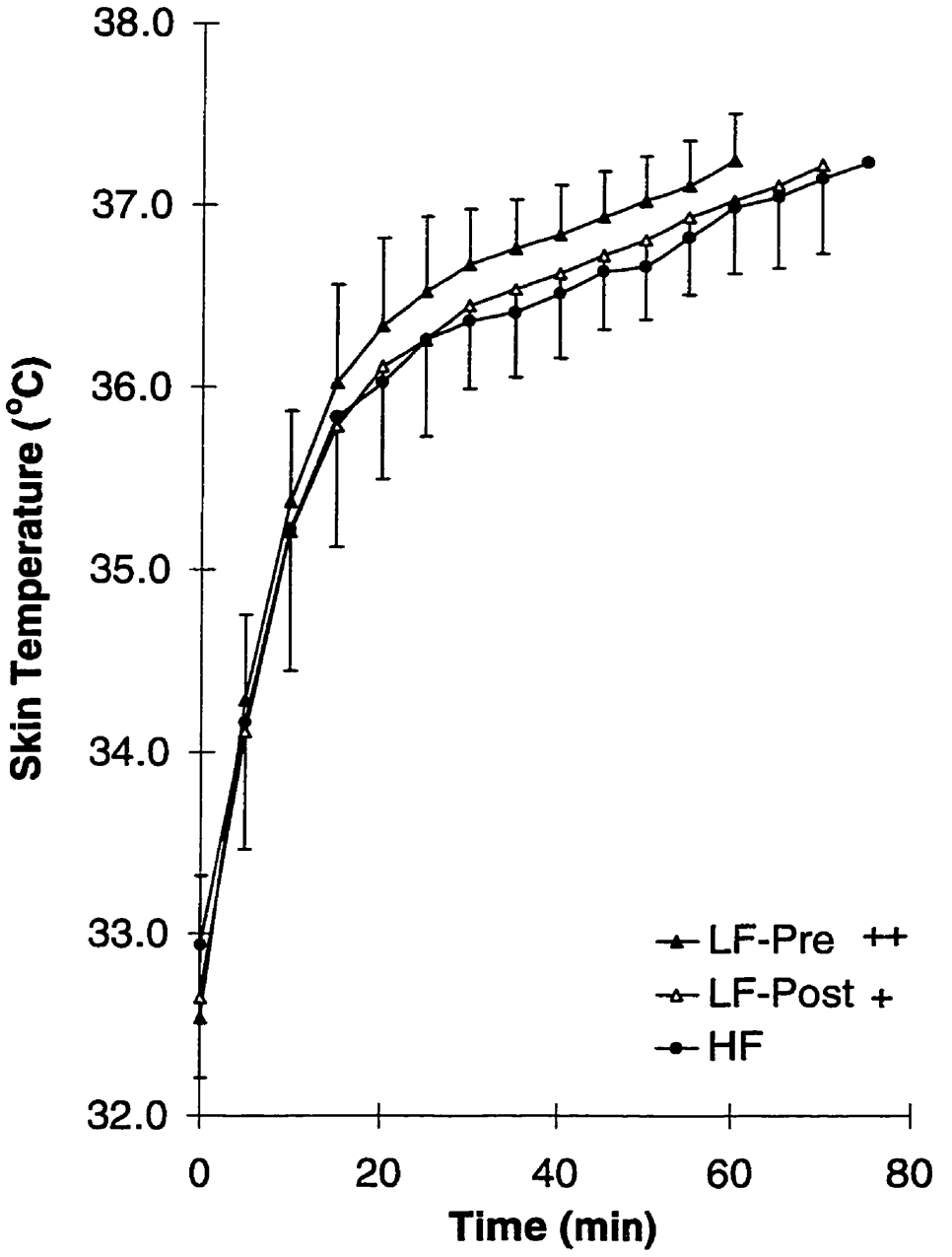

Values are means ± S.D. ++ Significantly different from HF

Figure 6.5 Mean skin temperature responses of low fit subjects (n=8) pre- (LF-Pre) and post-training (LF-Post), and high fit (HF, n=8) subjects during the euhydrated heat stress tests.




Values are means ± S.D. + Significant main effect of training; ++ Significantly different from HF

Figure 6.6 Mean skin temperature responses of low fit subjects (n=8) pre- (LF-Pre) and post-training (LF-Post), and high fit (HF, n=8) subjects during the euhydrated heat stress tests.




Values are means ± S.D. + Significant main effect of training; ++ Significantly different from HF

#### 6.1.4.4.Tolerance to the HST

Tolerance to the HST was determined by time, ethically imposed physiological endpoints, or subject exhaustion. None of the trials approached the 4 h time limit. In the LF individuals, the training did not result in an increased tolerance during uncompensable heat stress in either a EU or HY state (Table 6.4). In addition, the HF subjects had a significantly higher tolerance time of approximately 15-20 min compared to the LF-Pre and LF-Post subjects during either the EU or HY trials. In all groups, hypohydration resulted in a significantly shorter tolerance time, with the higher initial $T_{re}$ a likely contributing factor.

A significant disparity in the reasons for termination of the HST was evident between the LF and HF groups (Table 6.5). In the LF subjects, the large majority of the HST were terminated due to voluntary exhaustion before reaching the ethically-imposed core temperature limit of 39.3°C. In contrast, most of the HST with the HF subjects were terminated due to $T_{re}$ reaching 39.3°C. While voluntary exhaustion was sometimes coincident with this limit, the HF subjects often felt that they could have continued further. As a result, the $T_{re\ end}$ was likely underestimated in this group, but was still significantly higher than in LF subjects either pre- or post-training.

### *6.1.5. Discussion*

Since the initial studies by Piwonka et al (1965), a general consensus has arisen that an elevated level of aerobic fitness is linked to an improvement in physiological response to exercise in the heat (Armstrong and Pandolf 1988). A short-term period of physical training can produce an increased blood volume (Green et al. 1991, Harrison 1986), a lower resting core temperature (Shvartz et al. 1974), and improvements in the sweating responses (Henane et al. 1977, Nadel et al. 1974). In occupational settings, it would be of interest to examine whether a short-term period of training in previously untrained subjects can simulate the improved level of heat tolerance seen in subjects with

high aerobic fitness. In the present study, the subjects with a low aerobic fitness and sedentary lifestyles (LF) were compared, before and after a two-week aerobic training program, with subjects already possessing a high aerobic fitness brought about by months or years of habitual activity. The subjects were tested while either euhydrated or mildly (~2% body mass) hypohydrated to investigate whether training benefits are more apparent with hypohydration. In both an euhydrated and hypohydrated state, short-term training in the LF subjects resulted in an intermediate level of thermoregulatory and cardiovascular strain that was not significantly different from that found either before training or in highly fit subjects. However, the $\Delta T_{re}$, $T_{re\ end}$, and tolerance times remained significantly higher in the HF subjects, demonstrating that short-term training was ineffective in extending exercise-heat tolerance in an uncompensable heat stress environment.

The short-term training had to be of sufficient intensity to elicit adaptation, yet be light enough that the untrained and relatively unfit subjects could perform the entire protocol. Tolerance time for untrained and non-fit subjects cycling at 70-75% $\dot{V}O_2$ max has been reported to be approximately 50 min (McLellan and Skinner 1985), while Green et al. (1991) have stated that not all previously untrained subjects could complete 2 h of continuous cycling at 59% $\dot{V}O_2$ max for the initial few days of a 10-12 day training program. Therefore, we chose treadmill exercise at an exercise intensity of approximately 65% $\dot{V}O_2$ max in order to allow all subjects to exercise continuously for at least 1 h daily throughout the two-week period.

Before undergoing the training program, $T_{re}$, $f_c$, and $\overline{T}_{sk}$ were significantly higher in the LF-Pre subjects than in the HF subjects during both the EU and HY trials (Figures 6.1-6.6). The training period elicited a core temperature and heart rate response to exercise-heat stress that was intermediate between the LF-Pre and HF during both the EU and HY trials, resulting in a lack of a significant difference in the LF-Post subjects compared to either the LF-Pre or HF responses (Figures 6.1-6.6). Through the first 60 minutes of the HST, no significant differences were observed in $T_{re}$ and $f_c$ between the LF-

Post and HF, though a trend towards a higher $T_{re}$ and $f_c$ in the LF-Post remained. However, the HF subjects had both a significantly lower initial and a higher endpoint $T_{re}$ than the LF-Pre subjects, and a higher endpoint $T_{re}$ than the LF-Post subjects (Table 6.4). This resulted in a $\Delta T_{re}$ over the course of the HST of 2.3°C in the HF group, significantly higher than the 1.6°C $\Delta T_{re}$ in either the LF-Pre or LF-Post group. The elevated $T_{re\ end}$ in the HF subjects suggests that high aerobic fitness and/or habitual activity trains individuals to tolerate a higher levels of core temperature and discomfort, as the lower $\overline{T}_{sk}$ (Figures 6.5-6.6) and trend towards a decreased $f_c$ (Figures 6.3-6.4) in the LF-Post compared to the LF-Pre argues against either factor being the limiting factor to tolerance.

In an occupational setting involving work with protective clothing, a major question of interest is often the length of time that work can be performed before the subjects reach the point of voluntary exhaustion. With the light exercise intensity of the present analysis, the HF subjects exhibited a longer tolerance time than the LF-Pre subjects (Table 6.4), supporting prior comparisons between fitness groups using protective clothing in the heat (see Section 5.3), with heavy exercise (Windle and Davies 1996), and the general consensus of an improved exercise-heat tolerance with increased fitness (Armstrong and Pandolf 1988). Short-term training was unsuccessful in prolonging the tolerance time for the LF subjects in either a euhydrated or hypohydrated state (Table 5.12). Furthermore, despite the lack of any significant difference between the HF and LF-Post subjects in $T_{re}$, $\overline{T}_{sk}$, or $f_c$ during either the euhydrated or hypohydrated exercise (Figures 6.1-6.6), tolerance time remained significantly higher in the HF subjects compared to the LF-Post subjects (Table 6.4). On the basis of tolerance time, therefore, short-term training was of no benefit to subjects of low aerobic fitness.

The increased tolerance times in the HF subjects appear to be primarily due to a greater overall range of core temperature which could be tolerated during the HST. The difference in overall core temperature range between the HF and either the LF-Pre of LF-Post subjects was approximately 0.7°C (calculated from initial and endpoint $T_{re}$ in Table

6.4). Extrapolating an increase of $\Delta T_{re}$ in the LF-Pre group by 0.7°C, and given the linear rate of $T_{re}$ increase of approximately 1.0°C per 45 min (Table 6.4), the LF-Pre tolerance time would increase by 31.5 min. In contrast, decreasing the $\Delta T_{re}$ of the HF group by 0.7°C, the linear rate of $T_{re}$ increase of approximately 1.0°C per 40 min would decrease their tolerance times by 28 min. With an actual observed difference in overall tolerance time of approximately 22 min between the LF-Pre and HF groups, it is evident that the difference in tolerance times between the two fitness groups could be largely accounted for by the difference in $\Delta T_{re}$. Following training, the initial $T_{re}$ in the LF-Post subjects was slightly, but non-significantly, reduced from the LF-Pre during the EU trials. However, the $T_{re\ end}$ remained significantly lower than in the HF subjects, indicating that the training did not adapt the LF individuals towards tolerating a higher $T_{re}$ or $\Delta T_{re}$ before reaching voluntary exhaustion.

Indeed, the difference in $\Delta T_{re}$, $T_{re\ end}$, and tolerance times between the HF and LF groups (Table 6.4) was likely underestimated in the present study due to our definition of tolerance. The test sessions were terminated when subjects either reached the point of voluntary exhaustion and collapse or else a $T_{re}$ of 39.3°C was reached. However, the termination of the HST was primarily due to voluntary exhaustion, at a $T_{re}$ below 39.3°C, in the LF subjects. In contrast, the HF subjects typically terminated the HST due to $T_{re}$ reaching 39.3°C (Table 6.5). While voluntary exhaustion was sometimes coincident with $T_{re}$ reaching the ethical limit, the HF subjects subjectively felt that they could continue further in most cases, such that the time required to reach voluntary exhaustion was likely underestimated.

Hypohydration has been previously demonstrated to impair thermoregulatory abilities and tolerance in a hot environment in subjects with heterogenous fitness characteristics (Candas et al. 1988, Sawka et al. 1985, Sawka et al. 1992). Compared with fit individuals, however, the adaptations brought about by a short-term training program are likely to be greater in a group of subjects with low to moderate fitness (Havenith et al.

1995). Therefore, it was originally hypothesised that short-term training might increase the exercise-heat tolerance of the LF-Post to match that of the HF subjects even in a hypohydrated state. In a pattern identical to that observed during the euhydrated trials, the $T_{re}$, $\overline{T}_{sk}$, and $f_c$ were similar between the LF-Post and HF subjects (Figures 6.1-6.6), but the $T_{re\ end}$, $\Delta T_{re}$, and tolerance times remained higher in the HF subjects (Table 6.4). Therefore, the short-term training was ineffective in improving exercise-heat tolerance in the LF-Post compared to the HF subjects in either an euhydrated or hypohydrated state.

Due to the constraints imposed by the cross-sectional experimental design, care must be taken to avoid automatically assuming that fitness was the primary or only factor responsible for the differences observed during exercise-heat stress between the LF and HF groups. The comparison of fitness levels was made between two subject groups separated based on aerobic fitness and activity levels. In addition to significant differences in $\dot{V}O_2$ max, the HF subjects also had a lower body mass and body fat content. Each of these factors, along with several other factors which were not measured, could also contribute to the observed differences. In defence of the primacy of aerobic fitness, the lower body fat content and body mass was likely an indirect result of increased activity and $\dot{V}O_2$ max. Furthermore, groups with higher $\dot{V}O_2$ max were previously reported to demonstrate improved physiological strain and tolerance during exercise-heat stress in both a hot-dry (Shvartz et al. 1977) environment and while wearing protective clothing in the heat (Windle and Davies 1996), and aerobic fitness was found to be significantly correlated with physiological strain (Havenith et al. 1995, Havenith and van Middendorp 1990).

Another caveat to the interpretation of the results is to avoid an overemphasis on the dependent measure of tolerance time as the primary determinant of heat strain. While tolerance time is often the measure of most practical or occupational interest, its reliability and repeatability during submaximal exercise in a thermoneutral (McLellan et al. 1995) or an uncompensable heat stress (Montain et al. 1994) environment has been criticised. In the

present study, the claims of a decreased heat strain and an increased exercise-heat tolerance in the HF compared with either the LF-Pre or LF-Post subjects are supported by an increased $\Delta T_{re}$ and $T_{re\ end}$ in addition to the increased tolerance times.

In summary, the present study attempted, in a group of subjects with relatively low aerobic fitness, to replicate the decreased physiological strain and increased tolerance typically seen with high $\dot{V}O_2$ max and habitual exercise by using a short-term aerobic training program consisting of two weeks of daily exercise. Prior to the training period, inactive individuals of low aerobic fitness experienced significantly greater physiological strain and decreased tolerance during both euhydrated or hypohydrated exercise compared to active individuals with high aerobic activity. Following training, cardiovascular and thermoregulatory strain were similar in individuals of low fitness compared with those of high fitness during both the euhydrated and hypohydrated trials. However, the range of core temperature that could be tolerated during the heat exposure remained significantly lower, as did the final $T_{re}$ before the onset of voluntary exhaustion and overall tolerance time. It was concluded that, in preparation for exercise in an uncompensable heat stress environment, short-term aerobic training is not an adequate substitute for a high level of aerobic fitness resulting from habitual exercise and training.

## 6.2. Relative influence of aerobic fitness and individual characteristics on responses to uncompensable heat stress

### *6.2.1. Introduction*

The analyses detailed in Chapter 5 and Section 6.1 have concluded that, while wearing protective clothing with limited water vapour permeability, both aerobic fitness and hydration status are significant determinants of physiological responses and tolerance to exercise-heat stress. In contrast, despite minor improvements in physiological responses, exercise-heat tolerance was unaffected by either short-term aerobic training in previously non-fit individuals or a period of heat acclimation regardless of fitness. The purpose of the present study was to analyse the relative influences of inter-individual variations in aerobic fitness, hydration status, and anthropometric measures on the physiological responses and tolerance to uncompensable heat stress.

### *6.2.2. Methods*

#### 6.2.2.1. Experimental Design

The subject pool of 34 subjects were derived from the Fit subjects in Section 5.1 (n=5), the LF-Train (n=8) and LF-Control (n=6) subjects of Section 5.2, and the LF-Acc (n=7) and HF-Acc (n=8) subjects of Section 5.3. Subjects who did not achieve a hypohydration of at least 1.5% body mass were not included. The trials that were included in the present analysis were: the EU/F and HY/F conditions for Fit subjects during light exercise and the Pre-EU and Pre-HY trials for the LF-Train, LF-Control, LF-Acc, and HF-Acc groups. The exercise-heat stress was therefore standardised at an absolute intensity of 3.5 $km \cdot h^{-1}$ and 0% grade in an environment of 40°C, 30% relative humidity, and a wind speed $< 0.1$ $m \cdot s^{-1}$.

#### 6.2.2.2. Data Analyses

For the hypohydrated trials, the level of hypohydration (%HY) was calculated as the difference in nude body mass between HY and EU, expressed as a percentage of the EU nude body mass. The relative intensity (% $\dot{V}O_2$ max) of each HST was calculated by dividing the average oxygen uptake ( $\dot{V}O_2$ ) over the course of the HST by the subject's $\dot{V}O_2$ max.

For the present analysis, the values for $T_{re}$, $\overline{T}_{sk}$, $f_c$, and HG at t=60 min were used for comparison, as this was the longest common tolerance time for which all subjects completed the HST. the $T_{re\ end}$ was taken as the core temperature at which the subjects were removed from the HST.

Data are presented as the means and the standard deviations of the entire subject pool, along with the minimum and maximum response. Each independent variable was separately correlated with the physiological responses to the HST. Based on these correlations, a stepwise multiple linear regression analysis was performed, with the order of the steps determined by the investigator. The measure of fitness ( $\dot{V}O_2$ max in $L \cdot min^{-1}$, $mL \cdot kg^{-1} \cdot min^{-1}$, or $mL \cdot kg\ LBM^{-1} \cdot min^{-1}$ ) or relative exercise intensity (% $\dot{V}O_2$ max) with the highest correlation was taken as the first step in the regression analysis. Upon introduction of this variable into the equation, the anthropometric parameters related to body composition (body mass, LBM, BF, $A_D$, or $A_D$:mass) were included in the regression equation if they correlated significantly with the remaining variance. The $r^2_{adj}$ was calculated to account for the number of predictors used in the regression.

### *6.2.3. Results*

#### 6.2.3.1. Subjects

The data for the means and ranges of the individual characteristics for the 34 subjects are presented in Table 6.6. The subjects represented a heterogeneous spectrum of anthropometric measures. However, due to the requirement for a distinct separation in

Table 6.6 Physical characteristics of the subjects (n=34), with mean, minimum, and maximum values for age, aerobic capacity ($\dot{V}O_2$ max), body mass, percent body fat, lean body mass (LBM), Dubois surface area ($A_D$).

| | Mean | SD | Minimum | Maximum |
|---|---|---|---|---|
| Age (y) | 29.2 | 6.4 | 19 | 40 |
| $\dot{V}O_2$ max ( $L \cdot min^{-1}$ ) | 4.09 | 0.64 | 3.08 | 5.55 |
| $\dot{V}O_2$ max ( $mL \cdot kg^{-1} \cdot min^{-1}$ ) | 50.2 | 7.7 | 38.9 | 66.1 |
| $\dot{V}O_2$ max LBM ( $mL \cdot kg^{-1} \cdot min^{-1}$ ) | 59.7 | 7.0 | 48.1 | 76.5 |
| Body Mass (kg) | 82.1 | 11.4 | 64.0 | 104.5 |
| Body Fat (%) | 16.1 | 5.1 | 8.4 | 26.1 |
| LBM (kg) | 68.5 | 7.6 | 54.5 | 80.1 |
| $A_D$ ($m^2$) | 2.00 | 0.14 | 1.77 | 2.29 |
| $A_D$:mass ( $m^2 \cdot kg^{-1} \cdot 10^2$ ) | 2.46 | 0.20 | 2.18 | 2.85 |

aerobic fitness in designing Sections 5.2-5.3, and the generally high $\dot{V}O_2$ max of subjects in Section 5.1, individuals with a maximal aerobic power between 50-55 $mL \cdot kg^{-1} \cdot min^{-1}$ were largely excluded from the subject population.

### 6.2.3.2. Physiological Responses

The heat stress index ($HSI = E_{req} \cdot E_{max}^{-1}$) with the metabolic rate and climatic conditions used in the HST was approximately 2.5, such that heat storage continued throughout the HST and the subjects were unable to achieve a plateau in either $T_{re}$, $\overline{T}_{sk}$, or $f_c$. The mean and ranges of physiological responses to the euhydrated and hypohydrated HST are presented in Tables 6.7 and 6.8, respectively. The subjects demonstrated a wide variation in physiological responses. The values for $T_{re}$, $\overline{T}_{sk}$, $f_c$, and S were compared at 60 min, as this was the maximum exercise time completed by all subjects in either the EU or HY conditions. As outlined in Table 6.9, a distinct disparity was evident in the reasons for termination of the HST. The subjects in the three LF groups generally reached the point of voluntary exhaustion before their core temperature reached the ethically-imposed limit of 39.3°C. In contrast, while voluntary exhaustion was sometimes coincident with a $T_{re}$ of 39.3°C in the HF subjects, many of the HF could likely have continued further.

The first order correlations of the individual parameters with the physiological responses to the euhydrated and hypohydrated HST are presented in Tables 6.10 and 6.11, respectively. Summaries of the stepwise multiple regression analyses for the euhydrated and hypohydrated HST are presented in Tables 6.12 and 6.13, respectively.

### 6.2.3.3. Rectal Temperature

A prediction equation for $T_{re}$ after 60 min during both EU and HY trials could not be constructed, as none of the measured parameters reached a significant correlation.

Rectal temperature at the termination of either HST showed a strong correlation with several indices of aerobic fitness and individual characteristics for both EU and HY. For both EU and HY, the strongest regression equation was produced with relative

Table 6.7 Physiological responses of the subjects (n=34) to the euhydrated heat stress test, with mean, minimum, and maximum values for relative work intensity (% $\dot{V}O_2$ max), level of hypohydration (% body mass), rectal temperature ($T_{re}$), heart rate (HR), and skin temperature ($\overline{T}_{sk}$) after 60 minutes, sweat rate (SR), evaporation rate, change in $T_{re}$ ($\Delta T_{re}$), heat gain (HG), and tolerance time (TT) calculated over the entire HST.

| | Mean | SD | Minimum | Maximum |
|---|---|---|---|---|
| (% $\dot{V}O_2$ max) | 22.93 | 3.63 | 16.70 | 29.62 |
| $T_{re}$ (°C) | 37.83 | 0.25 | 37.4 | 38.5 |
| HR ($b \cdot min^{-1}$) | 131.6 | 18.4 | 102 | 179 |
| $\overline{T}_{sk}$ (°C) | 36.99 | 0.34 | 36.4 | 37.7 |
| SR ($L \cdot h^{-1}$) | 1.06 | 0.33 | 0.45 | 1.89 |
| Evaporation rate ($L \cdot h^{-1}$) | 0.33 | 0.09 | 0.19 | 0.69 |
| $\Delta T_{re}$ (°C) | 1.90 | 0.49 | 0.8 | 2.7 |
| $T_{re}$ end (°C) | 38.83 | 0.39 | 38.0 | 39.3 |
| HG (kJ) | 386.7 | 108.4 | 172.2 | 596.2 |
| TT (min) | 101.1 | 22.7 | 70 | 175 |

Table 6.8 Physiological responses of the subjects (n=34) to the hypohydrated heat stress test (HST), with mean, minimum, and maximum values for relative work intensity (% $\dot{V}O_2$ max), level of hypohydration (% body weight), rectal temperature ($T_{re}$), heart rate (HR), and skin temperature ($\overline{T}_{sk}$) after 60 minutes, sweat rate (SR), evaporation rate, change in $T_{re}$ ($\Delta T_{re}$), heat storage (S), and tolerance time (TT) calculated over the entire HST.

| | Mean | SD | Minimum | Maximum |
|---|---|---|---|---|
| (% $\dot{V}O_2$ max) | 23.60 | 3.28 | 17.84 | 29.67 |
| % Hypohydration | 2.71 | 0.63 | 1.71 | 4.66 |
| $T_{re}$ (°C) | 38.13 | 0.28 | 37.6 | 38.8 |
| HR ($b \cdot min^{-1}$) | 145.3 | 18.7 | 110 | 183 |
| $\overline{T}_{sk}$ (°C) | 37.13 | 0.34 | 36.5 | 37.9 |
| SR ($L \cdot h^{-1}$) | 1.09 | 0.31 | 0.42 | 1.72 |
| Evaporation rate ($L \cdot h^{-1}$) | 0.30 | 0.07 | 0.11 | 0.53 |
| $\Delta T_{re}$ (°C) | 1.66 | 0.52 | 0.7 | 2.6 |
| $T_{re}$ end (°C) | 38.79 | 0.40 | 37.9 | 39.3 |
| HG (kJ) | 422.2 | 114.5 | 158.5 | 638.4 |
| TT (min) | 84.3 | 18.3 | 60 | 141 |

Table 6.9 Reasons for the termination of heat stress tests in the four groups of subjects in the three studies, with subjects defined as low fit (LF), fit, (Fit), or high fit (HF).

| Group | Time | Core Temperature | Heart Rate | Volition |
|---|---|---|---|---|
| Section 5.1 (Fit) (n=5) | 0 | 10 | 0 | 0 |
| Section 5.2 (LF) (n=14) | 0 | 2 | 0 | 26 |
| Section 5.3 (LF) (n=7) | 0 | 0 | 2 | 12 |
| Section 5.3 (HF) (n=8) | 0 | 12 | 0 | 4 |

Table 6.10 First order correlations of the individual parameters of the subjects (n=34) with physiological responses to the heat stress test while euhydrated, either measured overall or over the first 60 minutes of heat exposure. Correlation coefficients with absolute values above 0.34 (bold print) are significant to $p < 0.05$.

| | $T_{re}$ | HR | $\overline{T}_{sk}$ | SR | Evap | $\Delta T_{re}$ | $T_{re}$ end | HG | TT |
|---|---|---|---|---|---|---|---|---|---|
| Age | -0.20 | -0.30 | -0.27 | 0.11 | -0.09 | -0.14 | -0.11 | -0.07 | 0.13 |
| $\dot{V}O_2$ max | -0.14 | **-0.44** | -0.22 | **0.71** | **0.66** | **0.54** | **0.49** | -0.25 | **0.41** |
| $\dot{V}O_2$ max (kg) | -0.04 | -0.30 | -0.05 | **0.74** | **0.52** | **0.73** | **0.70** | **-0.72** | **0.51** |
| $\dot{V}O_2$ max (LBM) | -0.01 | -0.31 | -0.06 | **0.76** | **0.53** | **0.67** | **0.67** | **-0.60** | **0.45** |
| % $\dot{V}O_2$ max | 0.30 | **0.50** | 0.36 | **-0.64** | **-0.63** | **-0.78** | **-0.72** | **0.62** | **-0.73** |
| Mass | -0.10 | -0.16 | -0.20 | -0.01 | 0.15 | -0.21 | -0.20 | **0.49** | -0.09 |
| Fat (%) | 0.08 | 0.16 | 0.04 | **-0.38** | -0.27 | **-0.53** | **-0.45** | **0.65** | **-0.40** |
| LBM | -0.17 | -0.27 | -0.28 | 0.19 | 0.34 | 0.03 | 0.00 | 0.25 | 0.11 |
| $A_D$ | -0.11 | -0.16 | -0.27 | 0.02 | 0.18 | -0.15 | -0.21 | **0.47** | -0.08 |
| $A_D$:mass | 0.11 | 0.14 | 0.14 | 0.04 | -0.13 | 0.25 | 0.19 | **-0.43** | 0.08 |

Table 6.11 First order correlations of individual parameters of the subjects (n=34) with physiological responses to the heat stress test while hypohydrated, either measured overall or over the first 60 minutes of heat exposure. Correlation coefficients with absolute values above 0.34 (bold print) are significant to $p < 0.05$.

| | $T_{re}$ | HR | $\overline{T}_{sk}$ | SR | Evap | $\Delta T_{re}$ | $T_{re}$ end | HG | TT |
|---|---|---|---|---|---|---|---|---|---|
| Age | -0.17 | -0.15 | -0.21 | 0.09 | -0.08 | -0.24 | -0.19 | -0.11 | 0.01 |
| $\dot{V}O_2$ max | -0.01 | **-0.51** | 0.15 | **0.58** | **0.45** | **0.35** | **0.45** | -0.10 | **0.38** |
| $\dot{V}O_2$ max (kg) | -0.02 | **-0.41** | 0.01 | **0.68** | **0.65** | **0.75** | **0.78** | **-0.62** | **0.62** |
| $\dot{V}O_2$ max (LBM) | 0.05 | **-0.44** | 0.11 | **0.67** | **0.61** | **0.65** | **0.73** | **-0.45** | **0.54** |
| % $\dot{V}O_2$ max | 0.31 | **0.57** | 0.21 | **-0.50** | **-0.61** | **-0.66** | **-0.67** | **0.53** | **-0.78** |
| % HY | 0.19 | -0.01 | -0.18 | 0.28 | 0.26 | 0.02 | 0.19 | -0.30 | 0.04 |
| Mass | 0.03 | -0.12 | 0.17 | -0.09 | -0.20 | **-0.49** | **-0.38** | **0.56** | -0.28 |
| Fat (%) | 0.13 | 0.19 | 0.20 | **-0.40** | **-0.45** | **-0.64** | **-0.56** | **0.67** | **-0.50** |
| LBM | -0.05 | -0.24 | 0.11 | 0.09 | -0.03 | -0.26 | -0.16 | 0.32 | -0.07 |
| $A_D$ | 0.00 | -0.17 | 0.05 | -0.07 | -0.18 | **-0.44** | **-0.36** | **0.54** | -0.25 |
| $A_D$:mass | -0.03 | 0.06 | -0.27 | 0.11 | 0.22 | **0.49** | **0.35** | **-0.48** | 0.27 |

Table 6.12 Regression coefficients for the physiological responses to the heat stress test with the individual parameters of the subjects (n=34) while euhydrated.

| Variable | Constant | $\dot{V}O_2$ max | $\dot{V}O_2$ max (kg) | $\dot{V}O_2$ max (LBM) | % $\dot{V}O_2$ max | Age | $A_D$ | $r^2_{adj}$ |
|---|---|---|---|---|---|---|---|---|
| $T_{re}$ | | | | | | | | |
| HR | 73.4 | | | | 2.54 | | | 0.23 |
| $\overline{T}_{sk}$ | 36.23 | | | | 0.033 | | | 0.10 |
| SR | -0.752 | | | 0.019 | | 0.006 | | 0.61 |
| Evap | 0.001 | 0.040 | | | | | | 0.41 |
| $\Delta T_{re}$ | 4.31 | | | | -0.11 | | | 0.60 |
| $T_{re}$ end | 40.60 | | | | -0.08 | | | 0.51 |
| HG | 425.5 | | -9.01 | | | | 206.6 | 0.56 |
| TT | 205.6 | | | | -4.56 | | | 0.52 |

Table 6.13 Regression coefficients for the physiological responses to the heat stress test with the individual parameters of the subjects (n=34) while hypohydrated.

| Variable | Constant | $\dot{V}O_2$ max | $\dot{V}O_2$ max (kg) | $\dot{V}O_2$ max (LBM) | % $\dot{V}O_2$ max | % HY | Age | $A_D$ | $r^2_{adj}$ |
|---|---|---|---|---|---|---|---|---|---|
| $T_{re}$ HR | 68.6 | | | | 3.25 | | | | 0.30 |
| $\overline{T}_{sk}$ SR | -0.146 | | 0.014 | | | | | | 0.44 |
| Evap | -0.015 | | 0.003 | | | | | | 0.40 |
| $\Delta T_{re}$ | -0.89 | | 0.05 | | | | | | 0.55 |
| $T_{re}$ end | 36.72 | | 0.04 | | | | | | 0.60 |
| HG | 155.9 | | -7.38 | | | | | 318.3 | 0.49 |
| TT | 186.9 | | | | -4.35 | | | | 0.59 |

$\dot{V}O_2$ max, expressed in $mL \cdot kg^{-1} \cdot min^{-1}$, with no other individual characteristic being significantly correlated with the remaining variance.

The range of $T_{re}$ an individual could tolerate over the course of the HST ($\Delta T_{re}$) was primarily influenced by the level of aerobic fitness. Relative exercise intensity (% $\dot{V}O_2$ max) provided the best regression with $\Delta T_{re}$ during EU, with no other significant contributing factors being found. Relative $\dot{V}O_2$ max (in $mL \cdot kg^{-1} \cdot min^{-1}$) provided the best prediction for $\Delta T_{re}$ during HY.

#### 6.2.3.4. Heart Rate

Heart rate at the end of 60 min during either EU or HY correlated significantly only with measures of aerobic fitness. The best regression equation was based on relative exercise intensity (% $\dot{V}O_2$ max), with no other individual characteristic providing a significant correlation after this first step. The amount of variance explained from the prediction equations were low for both EU and HY.

#### 6.2.3.5. Skin Temperature

Skin temperature after 60 min had minimal correlation with any measured variable. During EU, $\overline{T}_{sk}$ was significantly correlated with % $\dot{V}O_2$ max, though it explained only 10% of the observed variance. None of the measured parameters was significantly correlated with $\overline{T}_{sk}$ during HY.

#### 6.2.3.6. Sweating and Evaporation

During EU, the rate of sweating was primarily predicted by an individual's $\dot{V}O_2$ max relative to lean body mass ($mL \cdot kg\,LBM^{-1} \cdot min^{-1}$), with the subsequent incorporation of age producing a small, but significant, increase in $r^2_{adj}$. The absolute $\dot{V}O_2$ max provided the strongest prediction of the rate of sweat evaporation when euhydrated. During HY, both the rate of sweat production and evaporation exhibited a strong correlation with an individual's relative $\dot{V}O_2$ max.

#### 6.2.3.7. Heat Storage

The rate of heat storage during the HST had strong correlations with indices of aerobic fitness as well as with anthropometric measures of mass, body fat, and surface area. Of the fitness indices, relative $\dot{V}O_2$ max (in $mL \cdot kg^{-1} \cdot min^{-1}$) provided the regression equations with the highest $r^2_{adj}$ for both EU and HY. Following incorporation of relative $\dot{V}O_2$ max into the prediction equations for both EU and HY, heat storage remained significantly correlated with absolute surface area.

#### 6.2.3.8. Tolerance Times

Of the anthropometric measures related to body composition, only body fat content was initially significantly correlated with tolerance times for both EU and HY. However, for both EU and HY, the relative exercise intensity (% $\dot{V}O_2$ max) provided the strongest correlation to tolerance times, explaining 50-60% of the inter-individual variance.

### *6.2.4. Discussion*

The wearing of protective clothing alters the interaction between the individual and the environment, and presents a number of additional challenges in predicting the physiological response to exercise in the heat. Metabolic rate, environmental conditions, and the degree of encapsulation within the protective clothing are important determinants of physiological response and tolerance during exercise in the heat (Montain et al. 1994). In addition, clothing-specific variables can dramatically affect the ability to thermoregulate and maintain heat balance (Nunneley 1989). For example, the bulk and weight of the clothing increases the metabolic cost of activity and the rate of heat production (Duggan 1988, Patton et al. 1995), while the fit of the clothing and its permeability to water vapour affects the rate of heat dissipation (Holmer 1995).

Inter-individual variations in fitness, anthropometric measures, and hydration status, can also significantly influence the response to exercise-heat stress (Havenith 1985,

Havenith et al. 1995, Havenith and van Middendorp 1990, Kenney 1985). Despite the additional variability brought about by clothing, differences in individual characteristics, notably aerobic fitness, remained a significant determinant of physiological responses to exercise in an uncompensable heat stress environment. Aerobic fitness was represented in either absolute or relative terms or by expressing the fixed work load as a percentage of $\dot{V}O_2$ max, and was found to be significantly correlated with all measured variables in either an euhydrated or a hypohydrated state. Compared with other individual characteristics, aerobic fitness formed the strongest correlation with all physiological responses to exercise-heat stress and with exercise-heat tolerance, with the lone exception of body fat content, which provided a slightly higher first order correlation with heat storage while hypohydrated. Therefore, a measure of aerobic fitness was used as the first step in the design of regression equations estimating the physiological responses.

While variations in fitness and anthropometric measures were found to contribute significantly to inter-individual responses during exercise-heat stress in the present analysis, the relative predictive values of the calculated equations, while significant, were not overly high. In a hot-humid environment while wearing only shorts, Havenith (1995) reported $r^2_{adj}$ values of 0.6-0.8 for many of his variables. In contrast, the HST in the uncompensable heat stress environment produced by the wearing of protective clothing with limited water vapour permeability resulted in $r^2_{adj}$ values ranging from non-significant to a high of only approximately 0.60 (Table 6.12-6.13). Several reasons may help to explain the lower proportion of variance explained by aerobic fitness and individual characteristics in the present analysis. Firstly, a high degree of unexplained variance could be brought about by a subject population insufficiently heterogeneous in it physical characteristics and fitness. However, the original design of the study resulted in a strong selection criteria for subjects with a wide range of aerobic fitness. Subjects with a $\dot{V}O_2$ max of 50-55 $mL \cdot kg^{-1} \cdot min^{-1}$ were deliberately excluded in the experimental design of Sections 5.2-5.3, resulting in a selection favouring a wide distribution of aerobic fitness.

The subjects also exhibited a high degree of heterogeneity in all other anthropometric measures (Table 6.6).

An alternative explanation is that the addition of the NBC clothing introduced a significant proportion of variance into the system. A microenvironment of as much as 50 L can be created in the air volume between the skin surface and the suit when wearing protective clothing (Sullivan et al. 1987). The multiple layers of clothing significantly impede the rate of heat dissipation, with each layer forming a separate microenvironment with different insulative properties and water vapour permeability (Sullivan and Mekjavic 1992). In addition, the efficiency of evaporative heat loss is reduced, as the sweat can be evaporated in the air layer above the skin layer and also because some of the heat of vapourisation may come from the ambient environment (Nunneley 1989). Because of these reasons, the use of dressed weight losses in either calorimetric or thermometric calculations significantly overestimated the actual evaporative heat loss from the skin and underestimated the heat storage within the body (McLellan et al. 1996). Therefore, clothing characteristics must be factored into heat strain predictive models (Holmer 1995). However, even when the same clothing ensemble is worn, individual variance can remain high. The fit of the clothing to the individual will influence the relative bulkiness of the clothing. This may alter gait mechanics and walking efficiency, and therefore, influence the rate of metabolism and heat production (Patton et al. 1995, Teitlebaum and Goldman 1972). Clothing fit also determines the volume of the microenvironment, which would impact on evaporative heat loss from the skin and the amount of heat stored within the body versus within the microenvironment (Sullivan et al. 1987). Furthermore, individual variance in the sweating rate within the clothing would influence the degree of clothing saturation and its insulation value (Candas and Hoeft 1995, Craig and Moffitt 1974). Lastly, the combination of clothing fit and saturation could affect psychological discomfort within the clothing. As the heat gain calculation in the present analysis does not account

for the heat stored within the microenvironment, the total heat storage within the clothing ensemble is likely underestimated.

One potential benefit from habitual exercise and high aerobic fitness is an increased rate of sweat production (Nadel et al. 1974, Taylor 1986). In a thermoneutral environment, the higher sweat rate increases the evaporative heat loss and decreases the heat storage within the body. When wearing protective clothing, however, the limited water vapour permeability may result in increased sweating, thereby promoting a faster rate of dehydration, rather than increasing the evaporative heat loss. In the present analysis, both the sweating and evaporation rate were found to be significantly related to a measure of maximal aerobic power. Therefore, an increased aerobic fitness appears to benefit heat tolerance by increasing the rate of heat dissipation to the environment even during uncompensable heat stress. As discussed above, however, caution should be taken to avoid directly linking elevated evaporation rates with decreases in heat storage when wearing protective clothing, due to the overestimation of evaporative heat loss from the skin and the variance in interactions between the individual and the microenvironment within the clothing.

A greater tolerance time with an increased level of aerobic fitness appears to be due primarily to the higher core temperatures which could be tolerated during the HST, with a significant relationship between a measure of aerobic fitness and both $T_{re\ end}$ and $\Delta T_{re}$ (Tables 6.10-6.11). A significant disparity in the reasons for termination of the HST was evident between the subjects with low, versus those with high, aerobic fitness (Table 6.9). In the original studies, subjects were categorized as being low fit (LF) and high fit (HF) based on their $\dot{V}O_2$ max and activity pattern (Sections 5.2-5.3). LF subjects primarily terminated the HST due to voluntary exhaustion before reaching the ethically-imposed core temperature limit of 39.3°C. In contrast, most of the HST with the Fit and HF subjects were terminated due to $T_{re}$ reaching 39.3°C. While voluntary exhaustion was sometimes coincident with this limit, the subjects often felt that they could have continued

further. As a result, the $T_{re\ end}$ was likely underestimated in those individuals with a high $\dot{V}O_2$ max. The higher $T_{re\ end}$ with aerobic fitness would increase the $\Delta T_{re}$, which in turn would increase tolerance time given a similar rate of $T_{re}$ increase.

Therefore, our definition of tolerance may have distorted the nature of the relationships between the individual characteristics and the physiological responses to exercise-heat stress and exercise-heat tolerance. Due to the ethical requirement to terminate the HST at 39.3°C, an artificial ceiling was imposed on the maximum $T_{re\ end}$ possible. Nearly half of the HST, almost all of which involved subjects in the two groups with a high $\dot{V}O_2$ max, were terminated upon reaching the $T_{re}$ limit rather than because subjects reached the point of voluntary exhaustion (Table 6.9). Therefore, both the true $T_{re}$ and the tolerance time at which voluntary exhaustion could be expected were likely underestimated in those with high $\dot{V}O_2$ max values. This would have the effect of increasing the inaccuracy of the regression and the remaining variance. Therefore, due to ethical considerations, the true nature and strength of the relationship between aerobic fitness and either $T_{re\ end}$ or tolerance time could not be determined confidently with our protocol.

In summary, inter-individual variations in aerobic fitness and anthropometric characteristics were found to have a significant influence on the physiological responses to exercise in an uncompensable heat stress environment while either euhydrated or hypohydrated. It therefore remains necessary to incorporate individual characteristics into models designed to estimate responses when protective clothing is worn. However, the proportion of variance accounted for by individual characteristics was less than that reported for a hot-humid environment where clothing was not a consideration. This may have been due to an increased amount of variance brought about by the addition of protective clothing with low water vapour permeability.

# 7. GENERAL DISCUSSION AND SUMMARY

## 7.1. General Discussion

The present research was developed to investigate the separate and interactive influences of hydration status, fluid replacement, short-term aerobic training, heat acclimation, and aerobic fitness during exercise in the heat. To our knowledge, this research program is unique in designing a series of experiments with consistent methodology in order to study the interactions between these factors. The research focused on an uncompensable heat stress environment, where the capacity for evaporative heat loss is less than the amount required to maintain thermal equilibrium. This condition was produced by exercising in a hot environment while wearing the NBC protective clothing, a multilayered clothing ensemble with a high insulative capacity and a low water vapour permeability worn by the Canadian Forces. It is anticipated that the findings of this research would be applicable to other occupational settings that feature the wearing of protective clothing in the heat, including firefighting and nuclear or chemical waste disposal.

In a compensable heat stress environment, or where clothing with significant water vapour permeability is worn, it is generally accepted that hypohydration prior to exercise produces a significant impairment in the physiological responses and tolerance to exercise. The present research extends this finding to an uncompensable heat stress environment. Regardless of an individual's training, fitness, or acclimation status, minor (2-2.5% body mass) levels of hypohydration prior to exercise significantly increased cardiovascular and thermal strain and decreased exercise-heat tolerance in all subject groups in each of the three experimental studies, demonstrating the relative importance of hydration status compared with physiological treatments or individual characteristics (Chapter 5). The relative importance of hydration status was also highlighted by the observation of a 10% decrease in tolerance times while exercising at a heavy intensity during the HY/F trials compared with the EU/F trials (Section 5.1). In contrast, a previous study in our

laboratory with NBC clothing in the heat, which used a similar metabolic rate, reported that neither aerobic training nor heat acclimation were successful in increasing exercise-heat tolerance (Aoyagi et al. 1994). However, the 10% decrement in performance translated into an absolute decrease in tolerance time of only 6 min, which is not of practical concern for the military.

The subsequent two studies employed only the light exercise intensity employed in the initial study. This light metabolic rate was chosen in order to maximise the potential differences in tolerance times observed with aerobic training or heat acclimation (Aoyagi et al. 1994, McLellan 1993). In the present research, following a two-week period of either daily aerobic training or heat acclimation, only minor improvements in the physiological responses to exercise were reported in either an euhydrated or hypohydrated state. However, tolerance times were not improved following either intervention (Sections 5.2-5.3). Similarly, in a group of individuals with high aerobic fitness and activity levels, thermal strain was mildly attenuated following heat acclimation, but no differences were observed in tolerance times (Section 5.3). The finding that heat acclimation had no effect on exercise-heat tolerance in both the low and high fitness groups is in contrast to two previous studies in our laboratory, which demonstrated significant decreases in physiological strain and increases in tolerance time during exercise in the heat with NBC clothing following heat acclimation (Aoyagi et al. 1995, McLellan and Aoyagi 1996).

A possible explanation for the disparity in the results between the present and previous research lies in the interaction between fluid replacement and training or heat acclimation. During these previous studies, no fluid replacement was provided (Aoyagi et al. 1995, McLellan and Aoyagi 1996). However, fluid replacement and the prevention of dehydration during exercise is another intervention that had a significant impact on exercise in an uncompensable heat stress environment. Compared with the maintenance of an euhydrated state during exercise, achieved by providing water at regular intervals, progressive dehydration resulted in an increase in cardiovascular strain at both light and

heavy exercise intensities and a decrease in tolerance time during light exercise (Section 5.1). During light exercise, physiological strain and tolerance times were similar between the EU/NF and HY/F trials, indicating that fluid replacement during exercise was as important as hydration status prior to exercise (Section 5.1). Fluid replacement may have extended exercise-heat tolerance during the pre-training or pre-acclimation HST to near the maximum possible given the uncompensable heat stress environment, thereby limiting the amount of additional improvement that could be achieved with training or heat acclimation. The findings of the present research, therefore, underline the importance of maintaining euhydration both before and during exercise as an intervention towards maximising exercise-heat tolerance in an uncompensable heat stress environment.

While aerobic training and heat acclimation did not influence exercise-heat tolerance, one of the consistent findings from the present research was the improved physiological response and tolerance time to exercise-heat stress in individuals with high aerobic fitness and a pattern of habitual physical activity compared with inactive individuals with low aerobic fitness (Section 5.3). Improved exercise-heat tolerance with high fitness was observed in both an euhydrated or hypohydrated state, either before or after a period of heat acclimation. In addition, though short-term aerobic training in a group of unfit and previously untrained individuals was able to improve the physiological response to a level similar to that of highly fit and active individuals, tolerance time still remained significantly lower after training (Section 6.1). While the experimental design does not permit the direct interpretation of an improvement in exercise-heat tolerance due to high aerobic fitness and habitual activity, it is evident that the observed improvement could not be replicated by either short-term aerobic training or heat acclimation programs.

The mechanisms or factors responsible for the improved exercise-heat tolerance in highly fit and active individuals remains unclear but the present work offers some clues. If fitness is the primary factor, then it is reasonable to assume that, at some point with continued aerobic training, tolerance times would increase to the level of those who are

highly fit. At what point does this occur, and what is the ideal training program? Though a wide range (40-65 $mL \cdot kg^{-1} \cdot min^{-1}$) of $\dot{V}O_2$ max was included in the present research, fitness accounted for less than 50% of the inter-individual variations in tolerance times (Section 6.2). Therefore, it is likely that a combination of factors determine exercise-heat tolerance. The lower body fat content of the highly fit group may have influenced the rate and capacity of heat storage, but it did not achieve significance once fitness was entered into the prediction equations (Section 6.2). Other possible explanations are that habitual exercise accustoms an individual to tolerating higher levels of skin wettedness or discomfort.

One of the primary distinguishing characteristics between the low and high fitness groups was the greater overall range of core temperature which could be tolerated during the heat exposure by highly fit individuals, regardless of hydration, training, or acclimation status (Section 5.3, 6.1). Both a lower initial and a higher endpoint $T_{re}$ contributed to the increased $\Delta T_{re}$ of approximately 0.7°C in highly fit individuals. Given a similar rate of rise in core temperature, this difference in $\Delta T_{re}$ would account for much of the differences in tolerance times between fitness groups. The difference in $\Delta T_{re}$ and tolerance times between the fitness groups may have even been underestimated due to the ethical $T_{re}$ limit of 39.3°C. The majority of the low fitness subjects terminated the trials due to voluntary exhaustion at a $T_{re}$ below 39.3°C. In contrast, the majority of the highly fit subjects terminated the trials due to reaching a $T_{re}$ of 39.3°C, and most reported that they could likely have voluntarily continued further. The physiological or psychological mechanisms by which the highly fit individuals were able to tolerate a higher core temperature remain unclear and an interesting recommendation for future research.

## 7.2. Conclusions

In response to the initial objectives outlined in Chapter 3, the following conclusions may be drawn from this research:

1. Hydration status is a critical determinant of exercise-heat tolerance. Minor (2-2.5% body mass) levels of hypohydration prior to exercise significantly increased physiological strain, with an elevated core temperature and heart rate response. Hypohydration decreased tolerance time compared to a euhydrated state at both a light and a heavy exercise intensity. Fluid replacement during exercise significantly increased tolerance during light intensity exercise, but was of no benefit during the heavy exercise intensity. It is speculated that this was due to the rapid onset of exhaustion from other factors compared with the delay required between the intake of fluid and its absorption into the body. The improvements during fluid replacement during light exercise may have minimised any benefits in exercise-heat tolerance that were expected from either the heat acclimation or short-term aerobic training program.
2. In a group of unfit and previously untrained individuals, short-term aerobic training was ineffective in reducing the physiological strain or prolonging tolerance time during exercise in an uncompensable heat stress environment in either a euhydrated or hypohydrated state. Minor hypohydration (2-2.5% body mass) significantly impaired exercise-heat tolerance regardless of training status.
3. Heat acclimation mildly attenuated physiological strain during exercise in an uncompensable heat stress environment in either an euhydrated or hypohydrated state, but did not significantly prolong exercise tolerance. Minor hypohydration (2-2.5% body mass) significantly impaired exercise-heat tolerance regardless of acclimation status.
4. In unfit and previously untrained individuals, neither heat acclimation nor short-term aerobic training significantly improved exercise tolerance in an uncompensable heat stress environment.
5. Aerobic fitness was a significant determinant of exercise-heat tolerance in an uncompensable heat stress environment. Compared to individuals who were unfit

and untrained, individuals with high levels of aerobic fitness and habitual activity experienced decreased levels of thermal strain and had significantly longer tolerance times in either an euhydrated or hypohydrated state. The primary distinguishing characteristic was a higher $\Delta T_{re}$ in fit individuals, due to a combination of a lower initial $T_{re}$ and a higher $T_{re}$ at endpoint. Fit individuals also experienced greater adaptation to heat following a heat acclimation program than did unfit individuals.

6. Physiological responses to exercise-heat stress were similar in unfit and untrained individuals following a short-term aerobic training program compared to highly fit and active individuals. However, tolerance time remained significantly higher in the fit individuals, primarily due to a lower initial $T_{re}$ and a higher $T_{re}$ at the endpoint, which resulted in a greater $\Delta T_{re}$.

In addition, the following issues, which were not original research objectives, were raised during the course of this research:

7. The limit to exercise-heat tolerance, as defined by the endpoint $T_{re}$, is not influenced by hydration, training, or heat acclimation status. The similarity in the endpoint $T_{re}$ was in contrast to studies which observed a significantly lower $T_{re}$ at exhaustion with hypohydration (Sawka et al. 1985, Sawka et al. 1992). However, the reduced tolerance times and lower $T_{re}$ endpoints were only observed with significantly greater (5-7%) levels of hypohydration than in the present study, with no differences observed at 3% hypohydration (Sawka et al. 1985).
8. Minor (2-2.5% body mass) hypohydration resulted in a decreased respiratory exchange ratio, primarily due to a decreased $\dot{V}CO_2$. The finding of a decreased RER during hypohydration in the heat is different from that observed in thermoneutral environments, where hypohydration did not elicit any differences in $\dot{V}O_2$ or RER during submaximal exercise (Dengel et al. 1992, Neufer et al. 1989).

9. Inter-individual variations in aerobic fitness and anthropometric characteristics were found to be significant predictors of physiological responses to both euhydrated and hypohydrated exercise in an uncompensable heat stress environment. The proportion of variance accounted for by individual characteristics, ranging from 0.4-0.6, was less than that reported for a hot-humid environment where clothing was not a consideration, which ranged up to 0.8 (Havenith et al. 1995). Therefore, it remains necessary to incorporate individual characteristics into models designed to estimate responses when protective clothing is worn.

### 7.3. Limitations of the Research

In addition to the caveats discussed in Chapters 5-6, the following limitations may arise in the interpretation and/or application of this research:

1. During an operational scenario in the field, psychological pressures and anxiety, inadequate nutrition, and sleep deprivation form additional stresses that may magnify the effects of any differences in physiology or hydration, or else overwhelm the effects resulting from any physiological treatment. Each of these factors are also capable of affecting performance, and are difficult to replicate in a laboratory setting.
2. As discussed in Section 5.2, the short-term training program may have been of insufficient intensity to provoke heat adaptations. However, it is unlikely that a higher intensity training program would have been successful in improving exercise-heat tolerance in this uncompensable heat stress environment, as a period of heat acclimation, which induced a greater degree of adaptation (see Section 5.3), did not result in longer tolerance times.
3. Water was the sole fluid replacement beverage studied. In a 24 h simulated NBC situation in a temperate climate, subjects receiving only water suffered a 15% and 20% decrement in physical and mental performance, respectively, compared to

subjects given an isotonic carbohydrate drink (van Dokkum et al. 1996). The biggest physical decrement was observed in tests measuring aerobic performance, with subjective ratings of exertion also higher amongst subjects given only water. Furthermore, 25% of the water-only subjects had to withdraw before the end of the 24 h due to physical exhaustion (van Dokkum et al. 1996). It is, therefore, unknown whether carbohydrate and/or electrolyte replacement during the shorter exercise times in the present research would be of benefit.

4. The timing and amount of fluid replacement was regulated in the present research. Altering the frequency and amount of fluid replacement may produce different effects on the physiological responses to exercise-heat stress. However, a previous study reported no differences in the final responses to exercise-heat stress in subjects given a single large bolus of fluid at various time points compared with the same amount of fluid at regular intervals (Montain and Coyle 1993).

5. A difference in energy intake between the EU and HY trials existed during the overnight recovery from the dehydration protocol, which may have influenced the degree of recovery and the responses to the HST. Diet and rehydration were controlled in the present study during the period following the dehydration protocol until the HST the next morning using PowerBars® and Gatorade®. The total energy intake during the HY trials was similar to the amount provided in a previous study that reported no difference in the rate of glycogen resynthesis while either euhydrated or hypohydrated (Neufer et al. 1991), so it is likely that muscle glycogen was adequately restored. However, with an average consumption of 4 L of Gatorade® or 4400 kJ overnight during the EU trials, compared with 0.8 L or 880 kJ during the HY trials, a difference of 3520 kJ existed between the EU and HY trials, which could influence the ability of the body to recover from the exercise of dehydration and also the responses to the subsequent exercise.

6. The present research has focused on fairly short duration, continuous exercise in the heat. The effects of manipulations which would alter the dynamics of heat exchange were not incorporated into the study design. Examples of these manipulations include rest breaks, which would decrease the average rate of metabolic heat production but which may stimulate thermoregulatory responses differently than continuous exercise, and partial encapsulation, which would increase the rate of heat dissipation.

### 7.4. Suggestions for Further Research

The present research has raised the following avenues for further investigations:

1. What are the physiological and psychological factors determining heat exhaustion? Individuals with a high $\dot{V}O_2$ max had a significantly higher endpoint $T_{re}$ than did non-fit individuals. What are the mechanisms behind these differences in endpoint temperature and $\dot{V}O_2$ max?
2. By what mechanism does hypohydration induce a higher resting core temperature?
3. What are the metabolic or hormonal mechanisms by which hypohydration resulted in a decreased carbon dioxide production and respiratory exchange ratio?
4. What are the effects of carbohydrate and/or electrolyte replacement during exercise in an uncompensable heat stress environment? Will the relatively short tolerance times in the NBC ensemble preclude any additional benefit from rehydration using a beverage besides water?

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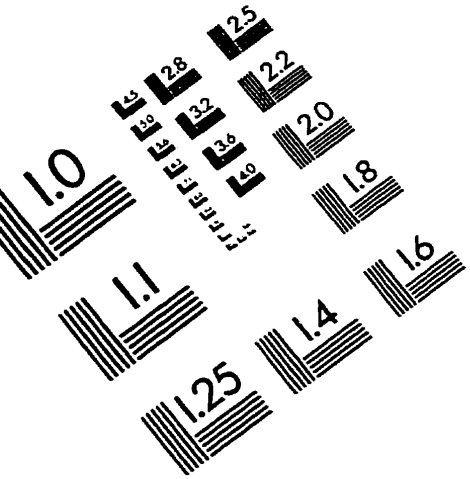
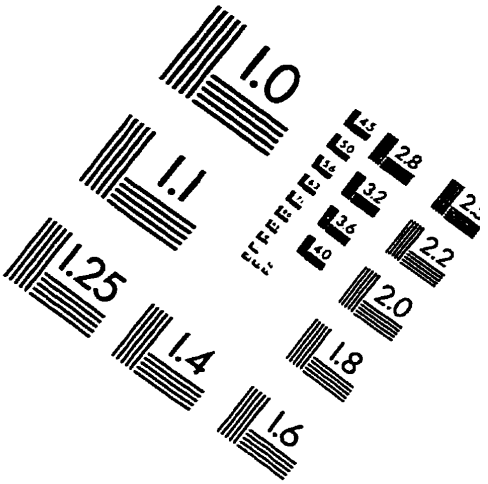
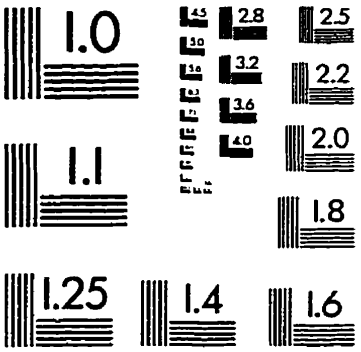
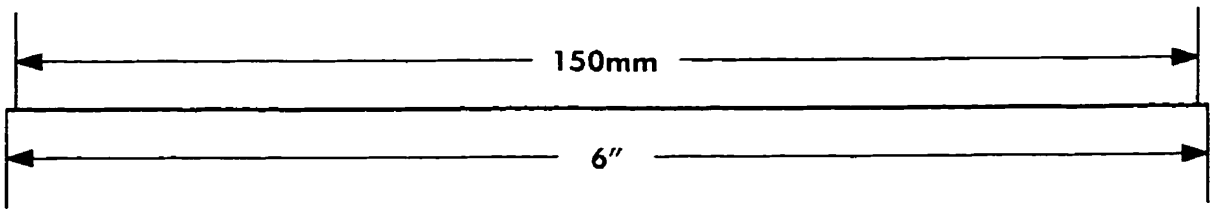
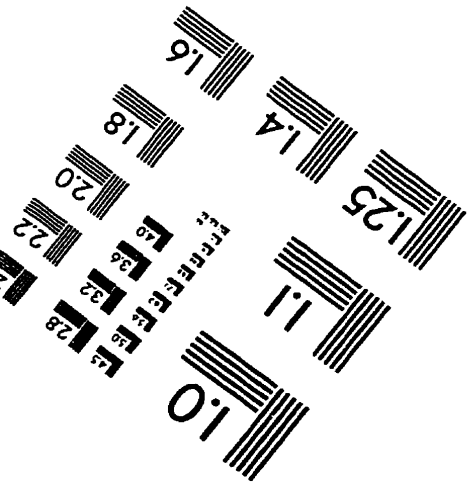
# IMAGE EVALUATION
# TEST TARGET (QA-3)

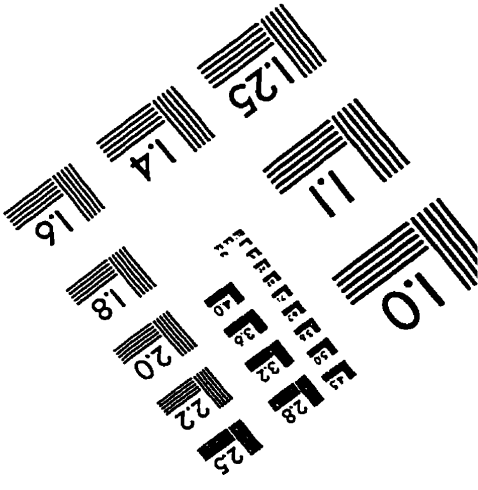
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