School of Public Health

Fluid and Electrolyte Losses Associated with Working and Exercising for Prolonged Periods in Thermally Stressful Environments

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DECLARATION

This thesis contains no material that has been accepted for the award of any other degree or diploma in any university.

To the best of my knowledge this thesis contains no material previously published by any other person except where due acknowledgement has been made.

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ABSTRACT

When working or exercising for prolonged periods in hot and humid environmental conditions, high sweat rates can lead to dehydration and electrolyte imbalance. It is well established that these progressive conditions will impair work and exercise performance in both athletes and manual workers. Consequences of inadequate fluid and electrolyte replacement in both cases can range from uncomfortable heat cramps, to more serious cases of heat illness, which in extreme cases can be fatal.

Fluid loss accompanying work or exercise in the heat has received extensive focus in the literature over the last 20 years, however, the sodium losses associated with large sweat losses have received far less attention. Due to the well-established link between high salt intake and hypertension, the World Health Organisation recommends that on a global scale individuals consume less than 5g salt per day. While these recommendations may be appropriate for the general population, the athlete or manual worker has substantially greater sodium losses and therefore is hypothesised to require a higher sodium intake. One of the principle aims of this thesis is to document sweat sodium losses and provide appropriate guidelines for fluid and sodium replacement in both manual workers and endurance athletes.

Chronic hyponatremia is a condition previously reported to occur in military personnel where over several weeks sodium intake is inadequate to replace large sweat losses. To date no previous study has investigated the incidence of chronic hyponatremia in a group of manual workers. Maintaining adequate fluid and electrolyte balance is essential to this population who are often working for prolonged periods in hot and humid environments. To investigate the incidence of chronic hyponatremia in this population, plasma sodium levels were assessed at the end of both the summer and winter months. Despite the similar dietary sodium intake across the year, the incidence of chronic hyponatremia at the end of the summer period was significantly greater than at the end of the winter period. This was postulated to be due to the higher sweat losses as a result of the more harsh

environmental conditions during the summer months. Dietary sodium intake was found to be inadequate to replace the estimated sodium losses due to the high proportion of rice and legumes consumed with very low sodium content. Increasing the sodium content of fluid and food provided to workers is warranted and may reduce the incidence of work related illness and accidents in this population.

In the athlete population there has been a great deal of focus in the literature on the need to adequately replace fluid losses that occur as a result of heavy sweating. Far less attention has been on the need to replace the sweat sodium losses accompanying this, with few studies documenting sodium losses in an endurance athlete population. There is large inter-individual variation in both sweat rate and sweat sodium loss in athletes with both factors being influenced by exercise intensity and duration, environmental conditions, physical fitness, size and number of individual sweat glands as well as the acclimatisation state of an athlete. It is therefore difficult to establish general recommendations for sodium intake in this population. In some athletes sodium losses can be replaced by normal dietary intake, whereas in other cases increased dietary intake is essential to replace large sweat sodium losses. In order to accurately determine sweat electrolyte losses, comprehensive laboratory based assessment is required, however this is often not practical. Chapter 4 and 5 of this thesis determined the effect of exercise intensity, seasonal heat acclimatisation and physical training status on fluid and electrolyte losses in a group of endurance athletes. The results from both of these studies predicts that endurance athletes with high sweat rates are at risk of both sodium and potassium deficiency, which may impair performance, if dietary intake is not sufficient to replace losses. Simple measures such as pre and post exercise weighing may be a useful tool in determining individual fluid and sodium intake requirements when comprehensive laboratory assessment is not practical.

Based on the findings from these three studies, fluid and electrolyte replacement recommendations have been made for both manual workers and athletes working and exercising in thermally stressful environments.

Key words: Hydration, sodium, potassium, sweat, exercise, hyponatremia, heat acclimatisation

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[1] Oral Presentation

[2] Winner, best oral presentation

LIST OF ABBREVIATIONS

[]	concentration
ALT	alanine Transaminase
AST	Aspartate Aminotransferase
Alk Phos	Alkaline Phosphatase
ANF	atrial natriuretic factor
ANOVA	analysis of variance
Bpm	beats per minute
BM	body mass
BMI	body mass index
CHO	carbohydrate
cAMP	cyclic adenosine mono phosphate
ºC	degrees Celcius
CV	coefficient of variation
ECF	extracellular fluid
ESR	erythrocyte sedimentation rate
FBC	full blood count
g	grams
GGT	Gamma-glutamyl Transpeptidase
h	hour
HCT	hematocrit
HGB	haemoglobin
HR	heart rate
ICF	intracellular fluid
K^+	potassium (ion)
Kg	kilogram

L	litres
LDH	lactate dehydrogenase
LFT	liver function test
M	mean
MCV	mean cell volume
MCH	mean cell hemoglobin
MCHC	mean corpuscular hemoglobin concentration
min	minute
mL	millilitres
mmol	millimole
mmol/L	millimole per litre
mosmol/kg	milliosmol per kilogram
mL/h	mLs per hour
Na$^+$	sodium (ion)
PLT	platelets
RPE	rate of perceived exertion
RH	relative humidity
rpm	revolutions per minute
SD	standard deviation
T Bili	Bilirubin
U&E	urea and electrolytes
$\dot{V}O_2max$	maximal aerobic capacity
W	watts
y	years

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CHAPTER 1: Introduction and literature review

1.1 General Introduction

Fluid and electrolyte disturbances, prompted by high sweat losses, can occur when a person is exposed to hot and humid environmental conditions for prolonged periods (>6h). In the endurance athlete this will have detrimental effects on performance, while also placing them at an increased risk of developing numerous forms of heat illness including heat cramps, and the more serious case of heat exhaustion. Heat exhaustion cases may develop headache, dizziness, fatigue, nausea, vomiting, and shortness of breath or syncope. In the manual labourer this can lead to impaired cognitive ability, dehydration and fatigue and in more serious cases lead to heat stroke. As a consequence of heat illness, productivity is significantly compromised and the risk of work related accidents increased. This research will seek to further explore fluid and electrolyte losses in these two 'at risk' populations in order to provide appropriate recommendations for replacement.

In many countries around the world, workers in the building and construction industry perform long periods of work in thermally stressful environments. Working in hot and humid conditions places an individual at risk of developing potentially severe heat illness due to substantial loss of fluid and electrolytes in sweat. While several studies have investigated the incidence of dehydration in susceptible populations (Bates, Miller, and Joubert 2010b; Bates, Gazey, and Cena 1996), there has been little focus on the need to replace the sodium losses that accompany fluid losses in sweat. In populations such as the Middle East, where the staple diet consists of rice and legumes, sodium consumption is potentially inadequate to replace sweat losses, placing a worker at risk of chronic hyponatremia. The consequences of chronic sodium depletion include fatigue, muscular weakness, nausea and vomiting, muscle cramps and in extremely severe cases may lead to circulatory failure (Hubbard, Szlyk, and Armstrong 1990). Study one (Chapter 2) of this thesis will be the first to document the incidence of hyponatraemia in a population of workers exposed to harsh environmental conditions while consuming a low sodium

starch based diet. As there are over 10 000 workers employed at the workplace studied, providing recommendations for fluid and electrolyte replacement has the potential to significantly impact on productivity, as well as the health of the workers, with wider implications throughout the region.

In athletes, the need to replace water lost through sweat during exercise in the heat is well documented (Rehrer 2001; Coyle 2004); and is important for both physiological function and exercise performance. The sodium loss that accompanies water loss through sweat however, has received much less attention in the literature, leading to a lack of specific sodium replacement guidelines, with many athletes failing to adequately replace sodium losses (Valentine 2007; Montain, Sawka, and Wenger 2001). Currently the World Health Organisation (WHO) recommends that on a global scale salt intake should be less than 5g/day (less than 2g/day sodium) (World Health Organisation 2007). These recommendations may be appropriate for the general population; however, for athletes with large sweat losses, these guidelines are potentially inadequate. In athletes, the consequences of inadequate salt intake are incomplete rehydration, an increase in exertional heat cramps and all forms of heat illness and potentially increase in the risk of exercise associated hyponatremia.

Currently the sodium requirements of athletes are estimated based on the sweat rate of the athlete, which is calculated based on weight loss over an exercise session. The accuracy of this method is questionable due to the lack of information concerning the relationship between sweat rate and sweat sodium concentration in athletes. Study two of this thesis aims to document the relationship between sweat rate and sodium concentration at different exercise intensity levels. This information will aid in providing specific sodium replacement guidelines for athletes training and competing in different events.

Physical training and heat acclimatisation are factors which are known to result in adaptation to both the cardiovascular and thermoregulatory systems. At the level of the sweat gland this results in changes to an individual's sweat rate and sweat sodium concentration. A number of questions still remain as to the effect of heat acclimatisation on the thermo-regulatory adaptations that occur at the level of the sweat gland, particularly in well-trained athletes. No previous study has investigated the effect of seasonal acclimatisation on sweat sodium or potassium loss in a group of endurance-

trained athletes and this was therefore one of the aims of the third study (Chapter 5) presented in this thesis. A further objective of study three (Chapter 5) was to investigate the effect of physical fitness on fluid and electrolyte losses in order to be able to provide more specific guidelines for replacement.

This PhD thesis has the following objectives.

In Manual workers:

1. To investigate the risk of chronic hyponatremia in manual workers in the Middle East by comparing plasma sodium values in the summer and winter months (Chapter 2).
2. To analyse the sodium intake of workers to establish if the sodium intake of workers is adequate to maintain electrolyte balance (Chapter 2).

In endurance trained athletes:

3. To establish whether there is a relationship between sweat rate and sweat sodium concentration in athletes during exercise of varying intensity (Chapter 4).
4. To determine the effects of seasonal heat acclimatisation (summer vs winter) on sweat sodium and potassium concentration and to estimate potential electrolyte losses (Chapter 5).
5. To determine the effects of physical fitness on sweat rates and sweat electrolyte loss during cycling exercise in the heat (Chapter 5).

In both groups:

6. To formulate specific fluid and electrolyte guidelines for athletes and manual workers performing prolonged periods of work in thermally stressful environments (Chapters 2, 4 and 5).

1.2 Background and Literature Review

1.2.1 Heat Balance

Humans are extremely efficient at maintaining thermal homeostasis despite large fluctuations in environmental temperatures. Heat balance is maintained by small but significant changes in the rate of heat loss from the body primarily through adjustments in skin blood flow and sweating rate (Kenny et al. 2010). Metabolic processes within the body result in heat production, which must be dissipated to the environment in order to avoid excessive rises in core body temperature. For heat loss to occur, excess heat is transported through cutaneous vasodilation from the body core to the skin (Kamijo and Nose 2006). Once the metabolic heat has been transferred to the skin it is then able to be dissipated to the environment via conduction, convection, radiation and most effectively through evaporation (Gisolfi, Lamb, and Nadel 1993).

The rate of convective heat transfer is dependent on the difference in temperature between the skin and the surrounding air (Nose et al. 1988). In times when the air temperature is greater than body temperature, heat gain will occur as heat will be transferred from the air to the skin. Radiative heat loss occurs when the surface of the skin is higher in temperature than the surrounding surfaces in the environment. When the body is exposed to sources of radiant heat such as the sun or heavy industrial machinery, heat may be radiated from the environment to the skin surface (Nadel 1984). Conductive heat transfer occurs when the body is in contact with an external object. Heat passes from the warmer to the cooler object in order to equalise the temperatures of the two objects. Most evaporative heat loss takes place through the evaporation of sweat from the skin surface, with sweating being the most effective means of regulating core temperature (Kenefick 2007). There is also some evaporative heat loss from the respiratory lining. The efficiency of this transfer process is dependent on both environmental and physiologic function (Nadel 1984). The environmental temperature and humidity influence the contribution of evaporative and dry heat loss to overall heat loss. In hot and dry conditions, evaporation may account for up to 98% of cooling and must occur at a rate that enables adequate heat dissipation as well as preventing further heat gain (Gisolfi,

Lamb, and Nadel 1993). Physiologically, the efficiency of this process is dependent on the responsiveness of the individual sweat glands to neural signals of thermal load. In order for the sweating process to be effective, water must be evaporated from the skin surface, no heat will be lost if sweat drips or is wiped from the skin surface (Gonzalez and Cena 1985).

1.2.2 Thermoregulation in the Heat

The heat generated from performing external work such as manual labour or physical exercise contributes most significantly to body heat gain. Depending on the type of exercise performed, body metabolism increases up to 15 times in order to support skeletal muscle contraction. Seventy to ninety percent of this energy is released as heat which must be dissipated from the body in order to prevent excessive increases in core body temperature or hyperthermia (Sawka 1992; Sawka and Pandolf 1990).

In sedentary individuals in mild environmental conditions, the heat loss mechanisms of the body are able to dissipate body heat effectively, preventing a rise in core body temperature (Maughan 2010). As ambient temperature increases it is more challenging to maintain body temperature within normal limits, particularly when performing physically demanding tasks. With air or environmental temperatures over 36 degrees, there is insufficient gradient between the skin and the environment resulting in the inability to release body heat (Wendt, Van Loon, and Lichtenbelt 2007). In this case the gradient for heat exchange is reversed and the body may gain heat by radiation. Evaporation of sweat then becomes the only means in which to dissipate body heat. The sweating mechanism is highly efficient, 1L of sweat can remove approximately 580kcal of heat energy (Maughan 2010).

When the relative humidity is also high, sweating becomes less effective as a cooling mechanism (Nadel 1984). Water and electrolytes are continually lost but without the associated reduction in body temperature (Maughan 2010). If large fluid deficits are incurred without adequate replacement, there will be compromise to both the cardiovascular and thermoregulatory systems resulting in a gradual increase in core body temperature or hyperthermia. The level of physical work or exercise in these conditions will be limited with continued work potentiating the development of mild to severe heat illness.

The hypothalamus plays an essential role in monitoring and controlling thermoregulation (Nadel 1984). Thermosensitive neurons within the hypothalamus sense changes in the temperature of blood flowing through the brain centres detecting changes in core body temperature (Wendt, Van Loon, and Lichtenbelt 2007). The hypothalamus is also able to monitor peripheral temperature changes, receiving sensory afferent input from thermoreceptors throughout the body. In this way the hypothalamus is able to assimilate central and peripheral information. The primary effector organs that allow for adjustments in heat flux are vascular smooth muscles, the sweat glands and skeletal muscle (Nadel 1984). The smooth muscle cells allow for changes in vasomotor tone therefore adjusting blood flow from the core to the skin. Activation of eccrine sweat glands allows for fluid to be placed on the skin surface for evaporation and consequential heat dissipation. This process is particularly important at the onset of heavy exercise due to the rapid change in internal body temperature. As the internal body temperature rises, cutaneous vasodilation causes an increase in skin blood flow, while sweating rate also increases (Nadel 1984).

1.2.2.1 Effect of sex on Thermoregulation

Several studies in the literature have investigated differences in thermoregulatory efficiency between males and females (Hazelhurst and Claassen 2006; Kaciuba-Uscilko and Ryszard 2001; Kenny et al. 2010). Even when matched for age, $\dot{V}O_2$max, acclimation and body fat levels, females appear to have lower sweat rates than males in both hot dry and hot and wet climates (McLellan 1998; Mehnert, Brode, and Griefahn 2002; Havenith et al. 2008). Women are however able to maintain their core temperature with similar efficiency to males due to their ability to evaporate sweat from the body surface more effectively (Hazelhurst and Claassen 2006). This has been postulated to be due to the smaller body surface area to mass ratio in males compared to females (Avellini, Kamon, and Krajewski 1980b). As the heat generated during exercise is proportional to body mass, females potentially generate less heat, and due to their larger surface area to mass ratio, are able to dissipate heat more efficiently. A study by Inoue et al (1998) concluded that during passive heat exposure, females rely more on active vasodilation as a method of heat dissipation where males rely more on the sweating mechanism.

A recent study by Medeira et al (2010) investigated the effects of sex and aerobic capacity on sweat gland sensitivity using pilocarpine induced sweating in 44 male and female subjects. The results confirmed the hypothesis that maximal sweating rate is higher in subjects with greater aerobic capacity. There was also shown to be an additional sex related difference in sweat gland sensitivity to pilocarpine independent of $\dot{V}O_2$ max, with males displaying significantly greater sweat losses in response to lower doses of pilocarpine. The authors concluded from this work that variation exists between male and female sweating capacity and is potentially due to variation in gland size, number of receptors and sweat gland output.

Another recent study by Ichinose-Kuwahara (2010) aimed to assess sex differences in the sweat gland response to changes in exercise intensity. A significant difference was noted between sexes, with females displaying a much smaller degree of sweat rate increase when compared to males at a similar exercise intensity level. In addition to this it was noted that in order to reach maximal sweat output, females are required to work at a much higher relative intensity or require a higher body temperature than male subjects.

Conflicting results are often reported in the literature in this area of research. This may be primarily due to differences in the methodology used to induce sweating, with some studies pharmacologically inducing sweating and others using heat and exercise. Pharmacologically induced sweating such as through the use of pilocarpine will assess the sensitivity of the individual sweat glands and prevent local and neural thermoregulatory mechanisms from influencing sweating rate. However, whether the pharmacologically induced sweat response is indicative of the physiological response is unknown and requires further investigation. Further to this, several studies have only examined sweat losses from one region of the body (Havenith et al. 2008; Meyer et al. 1992). It is well known that there are significant variations in sweat losses across different regions of the body (Bates and Miller 2008). Studies using a single sweat site may therefore produce conflicting results to those where whole body sweat losses have been compared (Patterson, Galloway, and Nimmo 2000). In addition to this there are several other compounding factors such as aerobic capacity ($\dot{V}O_2$ max) and surface area to body mass ratio that differ significantly between genders and are not controlled for in all investigations (Madeira et al. 2010).

1.2.2.2 Effect of Age on Thermoregulation

There have been a number of investigations looking at the effect of the aging process on thermoregulation (Armstrong and Kenney 1993). Results of a study by Dufour and Candas (2007) showed a significant decrease in whole body sweat rate with age when comparing young (20-30years), middle aged (40-50 years) and older (60+ years) individuals. The results also showed the older and middle aged groups to have a higher skin and core temperature than the younger subjects. One limitation of this study and a number of others is that they have not matched the older and younger subjects for body composition (muscle and fat mass) or physical fitness level. Because of this it is not clear whether the reduction in heat tolerance is due to a reduced thermoregulatory capacity or another confounding factor.

A recent study by Kenny et al (2010) matched older (mean = 45 years) and younger (mean = 22 years) men for body composition and physical fitness. The subjects performed 90 minutes of recumbent cycling at a steady state of heat production (290W). No difference in the rate of heat loss or heat gain between groups was noted, leading the authors to conclude that physically fit older men have a similar thermoregulatory capacity to younger physically active men.

1.2.2.3 Effect of Dehydration on Thermoregulation

Prolonged work or exercise in the heat can result in significant losses of both fluid and electrolytes through sweat to enable body cooling. Sweat rates of over 1 L per hour are not uncommon when performing prolonged periods of work in the heat (Brake and Bates 2003a; Miller and Bates 2007b), while Armstrong et al (1986) has reported sweat rates as high as 3.1L per hour in the American marathon record holder Alberto Salazar during a heat chamber trial. Without adequate replacement of lost fluid volume, progressive dehydration occurs. Sweat is hypotonic to plasma, therefore prolonged sweating results in a gradual increase in the osmolality of body fluids. This hypertonicity leads to a redistribution of body fluids between the intracellular and extracellular spaces to protect plasma volume (Sawka 1992).

Aerobic exercise can produce a regional change in blood flow distribution (Gisolfi, Lamb, and Nadel 1993). Muscular endurance exercise requires an increased level of

blood flow to the working muscles and an associated decrease in flow to non-exercising muscles (splanchnic area and the skin). As dehydration progressively occurs, there is competition for blood circulation between active muscles for metabolism and the skin for heat dissipation (Gisolfi, Lamb, and Nadel 1993). The associated reduction in skin blood flow, resulting in a reduction in sweat evaporation, causes a gradual increase in core body temperature. A study by Montain and Coyle (1992b) estimated that during endurance exercise in the heat, there is an increase in core body temperature of 0.2 degrees for every 1% of body weight that is lost. This core temperature rise, due to dehydration, is greater during exercise in hot and humid conditions when compared with more temperate climates (Montain and Coyle 1992b).

Along with the decreased ability to thermoregulate, there is an associated increase in cardiovascular strain (Johnson and Kellogg 2010). This is characterised by an increase in heart rate and decreased stroke volume and cardiac output (Gonzalez-Alonso et al. 1997; Montain and Coyle 1992b, 1992a). A well cited study by Montain and Coyle (1992b) demonstrated that the amount of dehydration induced (between 1% and 4% of body weight) was in direct proportion to the increase in heart rate and decrease in stroke volume that was observed. Similarly Sanders et al (1999) reported an increase in heart rate of approximately 10 beats per minute over a 90 minute cycling effort when a person was dehydrated compared to being in the euhydrated state (Sanders, Noakes, and Dennis 1999).

Studies have also reported reduced sweating ability when a person is dehydrated, therefore resulting in a compromised thermoregulatory capacity (Senay 1968). The composition of sweat may also be altered when a person is dehydrated. In a study aimed at investigating whether exercise induced hypohydration affects sweat composition, 8 males exercised for 2 hours in the heat (38ºC, 60% relative humidity) on two separate occasions. Trial one was with no fluid intake (hypohydrated trial) and trial two involved consuming a sodium chloride solution (euhydrated trial). Sweat sodium concentration was significantly higher in the hypohydrated trial and may be due to higher extracellular sodium levels when hypohydrated (Morgan, Patterson, and Nimmo 2004).

1.2.3 Effect of Dehydration on Exercise Performance in the Heat

Despite many professional teams now implementing a fluid replacement plan, several investigations have reported a high incidence of dehydration in athletes prior to regular training practice or competition. A recent study by Hamouti et al (2010) reported that 91% of a group of 43 professional team players awoke in a dehydrated state. Volpe et al (2009) reported the pre-practice hydration status of 138 male and 125 female collegiate athletes with similar findings. Thirteen percent of the sample had urine specific gravity readings indicative of significant dehydration (USG >1.030) while 53% appeared dehydrated (USG1.020-1.029). Only 32% of the samples were euhydrated prior to commencing the exercise session. A further interesting finding from this study was that a greater percentage of males than females were dehydrated (Volpe, Poule, and Bland 2009).

1.2.3.1 Strength Power and High Intensity Exercise Performance

Few studies in the literature have investigated the effects of dehydration on power based or high intensity exercise performance. A review article by Maughan and Shirreffs (2010) discussed the need for clear advice to be given to athletes competing in short duration, high intensity events due to the higher rate of metabolic heat production leading to faster rate of body temperature rise. In events lasting 20 -30 minutes, severe hyperthermia may be far more likely to occur than in longer duration events where thermal equilibrium will be reached and maintained (Sutton 1990).

Diuretic induced dehydration was used to investigate the effects of hypohydration on 50m and 200m sprint running performance (Watson et al. 2005). Administration of Frusemide reduced body mass by 1.7kg (2.2%) but there was no decline in performance over both distances. In a further study fluid and diet restriction combined with forced sweating over a period of 58 hours was used to achieve a 2kg (2.7%) weight loss in male wrestlers and judo players prior to 3 x 30m sprints (Fogelholm et al. 1993). The subjects in this study were well accustomed to fluid restriction. Results showed that fluid and energy restriction had no effect on 30m running performance. These studies indicate that body weight loss of between 2-3% may have no effect on high intensity sprint performance.

Maxwell et al (2009) used 8 untrained males to investigate the effect of dehydration on intermittent sprint exercise performance while also monitoring physiological strain using the physiological strain index (PSI). The physiological strain index (PSI) is a measure designed to evaluate exercise stress and heat strain based on a database of rectal temperatures and heart rate responses obtained from a study of 100 males exercising in the heat (Moran 2000). Three trials were undertaken, the first in a euhydrated state, the second dehydrated by ~2% of body weight and the third dehydrated by ~4% of body weight. No difference was found in the amount of total work or power output over the 3 trials, however there was a significant increase in the physiological strain index with the highest level of dehydration.

Athletes involved in weight class sports such as weight lifting and wrestling often intentionally dehydrate prior to competition. Kraft et al (2010) aimed to investigate the effect of dehydration (3% body weight) on a full body resistance exercise program. Results showed total repetitions were lower for the dehydrated group, while average heart rate and perceived exertion were significantly increased indicating that dehydration magnified the feeling of exhaustion.

In a symposium on 'Performance, exercise and health, hydration, fluids and performance' Shirreffs (2009) discussed the effect of a reduced body mass on the ease of sprinting performance indicating that a reduced body weight may reduce physiological demand promoting improved performance which may in fact counteract any negative effect of dehydration on sprint performance.

1.2.3.2 Endurance Exercise Performance

The effect of dehydration on endurance exercise performance has been studied by either inducing a certain degree of body water loss before exercise, or by allowing dehydration to develop during exercise (Shirreffs 2005). The experimental findings of research are therefore different depending on the approach taken. It is clear from review of the current published literature that dehydration does have a marked effect on endurance performance. A review of the literature conducted in 2003, looked at the effect of dehydration on endurance performance ranging from 1h up to 6h of exercise (Cheuvront, Carter, and Sawka 2003). Conclusions from this review paper were that dehydration significantly hinders endurance performance when fluid loss is

equivalent to 2 – 7% of body weight and this is more significant when exercise is performed in the heat (>30ºC). The authors also conclude that when exercise is less than 90 minutes in more temperate environmental conditions, a fluid loss of <2% of body weight does not affect endurance performance. However, if exercise duration is >90 minutes and in hot and humid conditions, performance is more likely to be negatively affected (Cheuvront, Carter, and Sawka 2003).

Consequences of dehydration in the athlete include reduced training capacity, and sports performance, along with compromised ability to thermoregulate (Horswill 1998). Any level of dehydration will greatly affect events that rely heavily on the cardiovascular system. When body water content is decreased, cardiovascular strain is seen through an increase in heart rate and decrease in stroke volume (Shirreffs 2005). When exercise is performed in a hot and humid environment, dehydration of as little as 2% of body mass has consistently been shown to decrease endurance exercise performance (Below et al. 1995; Barr and Costill 1989; Walsh et al. 1994).

A meta-analysis was performed looking at performance in marathon races. It was determined that the optimal temperature for maximal performance is in the range 10 - 12 degrees (Montain, Cheuvront, and Lukaski 2007). Performing in hotter environments of greater than 35ºC promotes heat gain and reduces the ability to remove body heat resulting in an increase in core body temperature and a subsequent performance decline.

A study performed on endurance cyclists indicated that dehydration resulted in a decreased ability to maintain maximal exertion during a cycling effort (Walsh et al. 1994). In this study the cyclists were to perform a time trial at 90% of $\dot{V}O_2$max for as long as they were able, directly after a 60 minute cycle at 70% of peak $\dot{V}O_2$. It was noted that a 2% reduction in body weight prior to the start of exercise decreased the maximum cycling time at high intensity by approximately 34%, when compared with the hydrated group (Walsh et al. 1994). Furthermore, a review article by Sawka and Pandolf (1990) indicated that small (2% body weight) to moderate (4% body weight) water deficits were shown to significantly decrease exercise performance, primarily due to a significant reduction in maximal oxygen uptake in a hot environment.

The current research indicates that dehydration causes premature fatigue in athletes competing in endurance sport in the heat (Maughan and Shirreffs 2010). This is mainly due to the effects on circulation, which make hyperthermia more difficult to deal with, causing a further increase in core body temperature, heart rate and ratings of perceived exertion. The extent of the performance decline however is highly variable ranging from 7% to 60% (Maughan and Shirreffs 2010). In more temperate conditions the performance decrement is not so clear with evidence suggesting that a 1-2% reduction in body weight has no effect on performance (McConell, Stephens, and Canny 1999). It must also be noted that due to the different protocols used to induce dehydration, the nature of the tests used, as well as the physical characteristics of the subjects, it is difficult to determine an exact relationship between the degree of dehydration and the performance effect. What is clear from the current literature is that dehydration will impair both physical and mental performance to some degree. Severe dehydration will impact more seriously on aerobic performance (Kenefick and Sawka 2007) and also place an athlete at increased risk of developing serious heat illness (Maughan, Shirreffs, and Watson 2007).

In spite of the known negative effects of dehydration on endurance exercise performance, several studies have shown that elite level athletes from a wide range of sports regularly begin training or competition in a dehydrated state (Shirreffs, Sawka, and Stone 2006b; Maughan, Shirreffs, and Watson 2007).

1.2.3.3 Team and Skill Based Sports

Team sports such as basketball, football, hockey, soccer and tennis rely on a high level of aerobic fitness combined with the ability to produce bursts of high intensity efforts. Performance in these types of events also relies on the ability to perform complex skills and cognitive function for decision making.

Dougherty et al (2006) used exercise heat induced 2% dehydration and euhydration with a 6% carbohydrate electrolyte drink to determine the effects of dehydration on the skills of young male, basketball players (Dougherty et al. 2006). Compared with euhydration, 2% dehydration impaired shooting, lateral movement and defensive skills significantly. The drink supplied to the players in the euhydration group was a 6% carbohydrate solution; therefore it cannot be elucidated whether it was the drink

components or the prevention of dehydration which resulted in an improved skill performance. In a further study looking at soccer performance, the authors concluded that the 'no fluid' trial decreased skill performance level by 5% during a 90 minute intermediate exercise protocol (McGregor et al. 1999).

1.2.4 Effect of Dehydration on Work Performance in the Heat

A study by Bates et al (2010a) examined the hydration status of migrant workers across four separate workplaces in the Middle East. A total of 372 workers had urine specific gravity (USG) readings monitored at three time points during the working day for two consecutive days. The average USG reading over the two days was between 1.016-1.020 indicating an adequate level of hydration. However approximately 30% of the workers had USG readings over 1.026 which is a level of dehydration that would place them at increased risk of developing a heat related illness. Hydration status did not change significantly between the three time points analysed. Similar results were found in the Australian mining population where 60% of workers had urine specific gravity readings higher than 1.022 prior to commencing work (Brake and Bates 2003a). There was no change in USG readings over the length of the 12 hour shift indicating that there was no further dehydration occurring during work. This was hypothesized to be due to the strong emphasis on safe work practices and regular drinking in this particular workplace. These results clearly indicate that it is difficult to improve hydration status in the face of high sweat losses when working in challenging environmental conditions. The authors concluded from the results that active interventions in the workplace are required to increase the awareness of the importance of hydration when working in conditions of heat stress.

1.2.4.1 Effect on Physical Performance

Workers in the mining and building sector are often required to perform arduous physical tasks in very hostile environments for up to 12 hours every day. Focus in the literature is mostly commonly placed on athletes competing in the heat while this group is often overlooked. Working in such extreme conditions places high demands on the thermoregulatory mechanisms of the body, which can result in significant fluid losses through sweat on a daily basis which, if not replaced, can result in dehydration and electrolyte imbalance. If this is prolonged or severe the health of the workers may be seriously at risk (Kenefick and Sawka 2007).

A recent study conducted on a subset of Australian workers during the summer months estimated an average sweat loss of 1 L per hour, with an average sweat

sodium concentration of 50mmol/L (Bates and Miller 2008). Over a 12 hour work shift this may equate to a substantial fluid and salt deficit if adequate fluid replacement strategies are not in place. Progressive dehydration may lead to impaired mental and physical performance while posing a serious risk to health. Any level of dehydration in the workplace may be contributing to less than optimal performance and may increase the risk of work related accidents occurring.

An additional challenge for some workers is the need to wear encapsulated protective clothing which has been reported by Bishop et al (1991) to significantly increase sweat rates. The protective clothing has limited vapour permeability therefore increasing metabolic heat production while reducing the ability to dissipate heat to the environment (Barr, Gregson, and Reilly 2010). Further to this, the use of face masks can add additional challenges to maintaining adequate fluid intake (Kenefick and Sawka 2007).

While some studies in the literature have reported the incidence of dehydration in the workplace, very few studies have examined the effect of this on the risk of workplace accidents or productivity. Wasterlund et al (2004) investigated the effect of dehydration on 4 Zimbabwean forest workers engaged in manual harvesting. Workers consumed either 0.17L (dehydration) or 0.6L (Euhydration) every 0.5 hours for a 12 hour working shift. All workers took longer to finish their designated tasks on the low fluid intake regime.

Other studies have indicated that dehydration may increase the risk of workplace accidents due to orthostatic intolerance. Carter et al (2006) showed that when dehydrated by 3% of body weight, subjects had a significant decrease in cerebral blood flow when going from seated to a standing position. A classic study by Adolph (1938) also reported that dehydrated subjects fainted more quickly when undertaking an orthostatic challenge test.

1.2.4.2 Effect on Cognitive Performance

In another study, Gopinathan et al (1988) used cognitive tests to determine the effects of dehydration on mental performance. Eleven soldiers from tropical regions in India were dehydrated by between 1 and 4% of body weight by restricting water

intake while exercising in a climate chamber. Results of the cognitive tests showed significant mental impairments at a level of 2% or more loss of body weight. A further study conducted in the same laboratory found similar results while assessing the effects of dehydration between 1 and 3% on mental performance (Sharma et al. 1986). The additional component to this study was that the behavior testing was conducted in three different environmental conditions; thermoneutral (27°C, 50% RH); hot dry (45°C, 30% RH) and hot humid (39°C, 60% RH). Significant dose related effects were found at each of the levels of hydration and these results were compounded when comparing the thermoneutral to the hot conditions. Lieberman (2007), in a critical review article, noted that while these two studies provide evidence to suggest that dehydration negatively affects cognitive function, they do not identify specifically which behavioral functions are most affected.

There is insufficient information available to elucidate what level of dehydration will produce decrements in cognitive function. However the research that has been conducted in this area clearly shows that dehydration induced by heat, cold, exercise, or through fluid deprivation will have some degree of negative effect on behavior, mood and overall cognitive performance (Cian et al. 2001; Sharma et al. 1986). The dehydration that occurs at the cellular level in the brain as a consequence of dehydration is believed to contribute to the impairment in cognitive function (Lieberman 2007).

1.2.5   Heat Illness

As mentioned previously, when exercising or working in the heat, water loss can occur to such an extent that heat dissipation is impeded, resulting in an elevation of core body temperature and compromise to the metabolic and circulatory processes, resulting in a decrease in work or exercise performance and potentially resulting in heat illness (Jackson and Rosenberg 2010). The active muscles require a constant high rate of blood flow in order to supply oxygen and substrate, while blood flow to the skin is also vital as a means of dissipating body heat. When the ambient temperature is high and an individual has been sweating for some time, there is compromise to the blood flow to both of these tissues. If this situation continues for some time heat illness may ensue (Jackson and Rosenberg 2010).

Acclimatisation to the heat and physical training result in physiological adaptations which improve the ability to thermoregulate in these conditions (Henkin et al 2010). These adaptations include decreased rectal temperature, decrease in sodium concentration in sweat and lowered heart rate during exercise while increasing sweat rate, sweat sensitivity and plasma volume (Armstrong and Maresh 1991; Wendt, Van Loon, and Lichtenbelt 2007). These adaptations will be discussed in more detail later in this literature review.

There are 3 types of heat illness described in the literature, as in Fig 1 – heat cramps, heat exhaustion or heat syncope and heat stroke (Kamijo and Nose 2006). These conditions are characterised by varying degrees of water and salt loss ranging from mild heat cramps to heat stroke with a mortality rate of approximately 80%.

Workers who are performing long periods of manual labour in environmentally challenging conditions are at significant risk of developing heat illness (Jackson and Rosenberg 2010). It is important that workplaces are aware of the need to focus on prevention by educating workers on the need to replenish lost water and electrolytes, particularly sodium lost in sweat.

1.2.5.1 Heat Cramps

Heat cramps are painful spasms of skeletal muscle often associated with working or exercising in hot conditions (Donoghue, Sinclair, and Bates 2000). Most commonly occurring in the arms, legs and trunk, heat cramps can range in severity from very mild to severe and can be an early warning sign of impending heat exhaustion (Coris, Ramirez, and Van Durme 2004). The precise cause of heat cramps has not been elucidated, however, high fluid and electrolyte losses, particularly high sodium losses have been shown in several studies to be a contributor (Bergeron 2003; Stamford 1993; Stofan et al. 2005). Adequate replacement of water and electrolytes, particularly sodium, are believed to aid in prevention. Donoghue, Sinclair and Bates (2000) found a link between heat cramps and dehydration while Horswill (2009) found significantly higher sweat sodium losses in cramping than non-cramping football players. Both of these studies support the notion that fluid and electrolyte balance may be involved in the etiology of heat cramps. Adequate replacement of water and electrolytes, particularly sodium, are believed to aid in prevention. There is also some evidence to suggest that physical training and heat acclimation may decrease the risk of developing heat cramps (Armstrong and Maresh 1991).

1.2.5.2 Heat Exhaustion

Heat exhaustion results from the inability of the circulation to meet both thermoregulatory and circulatory demands (Day and Grimshaw 2005). Outdoor laborers are particularly vulnerable as they are exposed to heat during the extreme conditions in the daytime and can also be required to wear personal protective equipment, increasing heat retention (Kravchenko et al. 2013). Heat exhaustion can occur when an individual performs intense work in the heat as resulting in large fluid and electrolyte losses in sweat with subsequent hypovolemia. If these losses are not replaced the thermoregulatory system becomes overloaded resulting in an elevation in core temperature (Rogers et al. 2007). This is due to a failure of the circulatory system to supply adequate blood flow to the skin, resulting in impaired heat dissipation. The signs and symptoms of heat exhaustion include nausea, weakness, fatigue, thirst, anxiety and fainting (Kamijo and Nose 2006). While the symptoms of

heat exhaustion can be reversed quickly after receiving appropriate treatment, the condition can progress rapidly to heatstroke if fluid and electrolytes are not replaced.

A one year prospective case study of 106 cases of heat exhaustion in a deep underground metalliferous mine found that dehydration, measured by increased urea, creatinine and osmolality, as well as several other biological and haematological markers, was associated with the development of heat exhaustion (Donoghue, Sinclair, and Bates 2000).

A population based case control study was conducted investigating cases of exertional heat illness in male Marine Corps recruits during base training (Gardner et al. 1996). Results of the study showed a strong relationship between greater BMI and longer 1.5 mile run time predicting a higher risk for developing exertional heat illness. Of the 390 cases of heat illness, 47% had a BMI greater than 22kg m^{-2} coupled with a 1.5 mile run time of greater than 12min. There are several proposed reasons for this strong relationship between BMI and development of heat illness. Greater heat production as well as reduced heat dissipation due to a lower surface area to body mass ratio, may explain this finding, however there may be several other metabolic differences in workers carrying excess adipose tissue. Donoghue and Bates (2000) found a similar relationship between higher BMI and greater risk of heat illness.

1.2.5.3 Heat Stroke

Heatstroke results from a complete failure of the thermoregulatory system, resulting in core temperatures in excess of 40ºC and is considered a health care emergency (Rogers et al. 2007). At temperatures greater than 40 ºC, heat denaturation of proteins can cause cells of the nervous system to become necrotic, resulting in brain damage to survivors. Widespread tissue necrosis resulting in death occurs when temperatures continue to rise (Donaldson, Keatinge, and Saunders 2003). Exertional heat stroke has been documented in athletes and military recruits who are performing strenuous physical activity, often for prolonged periods in hot environments (Binkley et al. 2002). Symptoms of exertional heat stroke include hyponatremia, acute renal failure, rhabdomyolysis, prolonged unconsciousness or convulsions and death

(Armstrong et al. 2007). Data from the United States indicate that in 2003 to 2008, 196 deaths were attributed to fatal heat illness (Jackson and Rosenberg 2010).

Carter et al (2005) examined the incidence of heat illness including hospitalisations and death in the US army between 1980-2002. Five thousand two hundred and forty six cases were hospitalised during this time, while 37 died due to heat illness. Over the 22 year period there was a 60% reduction in the number of cases of heat exhaustion indicating that the workplace prevention programs were successful. However, there was shown to be a 14% increase in the number of hospitalised cases of heat stroke. The authors of this paper speculate that this may be due to the increased amount of time spent performing running activities as part of military training, as well as the increased use of nutritional supplements. The other possibility is that the increased focus on hydration has decreased the incidence of heat exhaustion and enabled workers to continue to exercise until more severe heat stroke occurs.

The available evidence to date suggests that the physically trained and heat acclimatised individual is at a decreased risk of developing any form of heat illness (Rowlinson et al. 2013; Tian et al. 2011). In addition to this an individual with a BMI outside the normal range is at greater risk (Donoghue, Sinclair, and Bates 2000). What is also clear is the importance of maintaining fluid and electrolyte balance both prior to and during the period of heat exposure. This may be even more important in the trained and heat acclimatised individual, when sweat losses may be substantially greater.

1.2.5.4 Fluid and Electrolyte Replacement When Working or Exercising in the Heat

The importance of replacing fluid losses during exercise in order to prevent excessive dehydration has been widely acknowledged in the scientific literature. However the recommendations for fluid replacement strategies during exercise continues to be a widely debated topic (Garth and Burke 2013). Since the early 1960's there has been a paradigm shift in the recommendations given to athletes from a message of 'not drink at all' during exercise to 'drink as much as tolerable' (Noakes 2007). Noakes (2012) states that ' there is barely any risk that dehydration can occur in healthy athletes where ample fluid is available'. According to Noakes

(2012), hormonal changes when exercising regulate the conscious sensation of thirst, as this thirst sensation rises performance is impaired. The sensation of thirst ceases when sufficient fluid and electrolytes have been consumed and the plasma osmolality returns to normal. Noakes (2012) believes that this control ensures that humans drink enough fluid but not too much to cause osmolality to fall (Noakes 2007). He argues that the only advice that should be given to athletes is that they should drink according to thirst (Noakes 2007). There are however a number of studies which show that athletes have inappropriate drinking behaviours (Shirreffs, Sawka, and Stone 2006b; Maughan et al. 2007). A classic study by Pitts et al (1944) showed that even when fluid was readily available during long periods of walking in the heat, fluid intake did not match sweat losses and subjects became progressively dehydrated. Similarly in a group of manual workers, Bates et al (2010c) showed that over 30% of the 372 workers tested had USG readings that indicated that the subjects were dehydrated. In a recent review article by Garth and Burke (2013), the importance of considering other factors influencing fluid intake was discussed. Drinking entirely to thirst may be difficult for athletes competing in events that involve continuous activity, where the time taken to consume fluid is included in the race time. It is also difficult for athletes and some workers to drink ad libitum when the opportunity to consume fluids is not self determined. For these athletes an individual "paced" fluid plan may be beneficial rather than relying on thirst alone (Garth and Burke 2013).

The American College of Sports Medicine (ACSM) position stand (1996) and the National Athletic Trainers association position stand (2000) both recommended that during exercise athletes should aim to replace 100% of their sweat losses. "During exercise, athletes should start drinking early and at regular intervals in an attempt to consume fluids at a rate sufficient to replace all the water lost through sweating (i.e., body weight loss), or consume the maximal amount that can be tolerated''(Convertino et al. 1996). However, according to Noakes et al (2005) this overzealous advice lead to an increased number of reported cases of exercise–associated hyponatremia. His concern is also that many athletes as well as fitness enthusiasts still believe that when exercising one can never drink too much fluid which can have life threatening consequences (Beltrami et al 2008). Almond et al (2005) did show that a few participants competing in the Boston marathon event did

drink more fluid than they lost in sweat, which has the potential to lead to hyponatremia with fatal consequences. This most commonly occurs in non-elite competitors who are walking or jogging at the back end of large events as they have more opportunity to overdrink (Maughan and Shirreffs 2010). Recreational athletes may also more commonly be given inappropriate fluid intake advice, it is therefore important that these athletes are aware of the problems relating to excessive fluid intakes (Garth and Burke 2013).

In 2007 the ACSM released an updated position stand which suggested that athletes should aim to maintain body weight to within 2% of pre-exercise weight (American College of Sports Medicine 2007). The major change from the 1996 recommendations was rather than providing a blanket statement for the amount of fluid athletes should consume per hour of exercise, the new statement considered individual sweat rates, exercise intensity and environmental conditions. These guidelines also suggest that athletes should replace 150% of the body weight loss during exercise which according to Noakes (2005) will promote dilution of the plasma increasing the risk of exercise associated hyponatremia. A further concern raised by Beltrami et al (2008) is that many exercise physiology textbooks contain advice regarding fluid replacement strategies which are not regularly updated therefore students are exposed to dated information.

1.2.5.5 Addition of Glucose to an Electrolyte Replacement Drink

It is widely recognised that performance is significantly improved in athletes who consume a carbohydrate-electrolyte fluid as opposed to plain water during exercise (Sawka et al. 2007). The addition of carbohydrate replenishes glycogen depleted muscles, but also aids in water retention (Rehrer 2001). In addition to this, glucose also aids in the rate of intestinal uptake of sodium which in turn increases water retention (Shi and Gisolfi 1998). There are however mixed results in the literature regarding the optimal concentration of carbohydrate to maximise water absorption while still providing an adequate energy source. For endurance events, a study by Tsintzas et al (1995) examined the effect of carbohydrate and electrolyte drinks on marathon running performance. They found that a 5.5% solution improved treadmill-running performance when compared with water. They also reported that numerous

runners experienced gastrointestinal discomfort with the use of the more concentrated 6.9 % solution. Maughan et al (1989) investigated the effect of ingestion of fluids and carbohydrate on cycling time to exhaustion in six healthy young males. Maximal time to exhaustion in the no fluid group was 70mins, 76mins when 100ml of water was given every 10 minutes, 79mins when a concentrated carbohydrate solution was given and 91mins when a dilute carbohydrate solution was administered. These results indicate that the composition of the carbohydrate-electrolyte drink is also important and that hypotonic solutions may produce the greatest performance benefits due to an improved gastric emptying and absorption time.

1.2.5.6 Addition of Sodium to an Electrolyte Replacement Drink

Sodium has been acknowledged as being a vital electrolyte for athletes, particularly those competing in the heat. Current guidelines for sodium replacement are not clear, with the American College of Sports Medicine's recent position stand (2007) stating that many athletes will require sodium in much greater quantity than the currently recommended upper intake level (2.3g/day). The recommendations state that sodium should be added to an electrolyte replacement drink, but no further quantitative recommendations for daily sodium intake are provided.

Far less research has been conducted on the optimal concentration of sodium that should be added to an electrolyte replacement drink. Sweat is hypotonic to plasma and contains approximately 45 mEq/L of sodium. The addition of sodium to a beverage enhances carbohydrate absorption, improves palatability as well as promoting fluid homeostasis. It has been shown that the addition of sodium to a replacement beverage maintains plasma volume more effectively than plain water (Sanders, Noakes, and Dennis 2001; Wemple, Morocco, and Mack 1997). The addition of sodium to an electrolyte replacement drink would therefore be of benefit to an athlete as it would maintain plasma volume while limiting cardiovascular strain (Anastasiou et al. 2009). Merson et al (2008) showed that the cumulative volume of urine produced following rehydration was inversely related to the sodium concentration of the fluid consumed, therefore indicating that a high sodium beverage may enhance rehydration. Post exercise, the administration of high sodium

solutions results in restoration of plasma volume to levels higher than those reported prior to beginning the exercise session (Nose et al. 1988).

Modigliani and Bernier (1971) performed an experiment to determine the optimal concentration of glucose and sodium in order to maximise absorption. They determined that the maximal absorption rate occurred when a 17mEq/L NaCl solution was added to a 133mM glucose solution. At a glucose concentration of 200mM, water absorption fell significantly, at a glucose concentration of 260mM net water secretion occurred. Therefore the optimal glucose/Na ratio in this study was 2:1. A more recent study by Anastasiou et al (2009) reported similar findings. The addition of 19.9mmol/L of sodium to a sports drink was shown to effectively prevent a decrease in plasma sodium concentration during exercise. The authors of this study concluded that replacement of sodium losses should be promoted during prolonged, moderate intensity exercise in order to maintain cardiovascular and thermal stability (Anastasiou et al. 2009).

In a practical setting is it vital that the palatability of the drink is maintained to ensure that large volumes of fluid can easily be consumed. The addition of high sodium concentrations can render a drink unpalatable. It has been well established that palatability is an important factor governing the amount of fluid an athlete will consume (Passe, Horn, and Murray 2000; Maughan and Leiper 1993). Wemple et al (1997) showed that subjects drank significantly greater volumes of a 25mmol/L solution than a 0 or 50mmol/L drink.

1.2.5.7 Addition of Potassium to a Electrolyte Replacement Drink

The concentration of potassium in sweat generally ranges from 1-15mmol/L, however can become more significant during prolonged exercise in the heat (Gisolfi 1990). Potassium depletion (hypokalemia) can cause symptoms such as disorientation and muscle weakness and in more serious cases paralysis and hyporeflexia. Research suggests that potassium should be added to an electrolyte replacement beverage at a concentration of between 5 to 10mEq/L to offset sweat losses. Potassium may also help in replacing lost intracellular fluid volume and is involved in nerve transmission and active transport processes. Far less research has been performed to investigate potassium losses in sweat during exercise.

1.2.6 The Eccrine Sweat Gland

There are an estimated 2.5 million eccrine sweat glands distributed across the human body surface. There is significant individual variation in size and number of sweat glands with a range of between 1.6 to 4 million (Taylor 1986). The density of the glands decreases distally with the greatest number of glands on the head followed by the upper limbs with lowest density on the lower limbs (Shibasaki, Wilson, and Crandall 2006). The primary function of the sweat gland is to maintain body temperature through the evaporation of sweat on the skin surface.

The sweat gland is made up of simple tubular epithelium consisting of a secretary coil located in the lower dermis and a duct which extends through the dermal layer and opens directly onto the skin surface (Werner 1990). The basic structure of the eccrine sweat gland is shown in Figure 1. Sweat glands form on the palms of hands and soles of feet by 16 weeks of gestational age and resemble adult sweat glands by the eight month of gestation (Shibasaki, Wilson, and Crandall 2006). Reabsorption of water and electrolytes occurs throughout the length of the duct allowing vital electrolytes, mainly sodium, to be reabsorbed and therefore conserved, throughout the passage of sweat to the skin (Werner 1990). This reabsorption depends on the active transport of sodium into the interstitial fluid via $Na^+K^+ATPase$ which is localized on the basolateral membrane of the ductal cells. The eccrine sweat glands secrete a fluid which is hypotonic to plasma consisting of variable quantities of sodium, chloride and potassium and very small quantities of lactate, urea, ammonia, proteins and peptides.

There is a considerable difference in the size of individual sweat glands between people with some authors reporting this difference to be fivefold (Sato and Sato 1983). The same authors have also shown that there is a positive correlation between the size of an individual sweat gland and the maximal sweat rate of that gland (Sato and Sato 1983).

Figure 1. Basic structure of an eccrine sweat gland.

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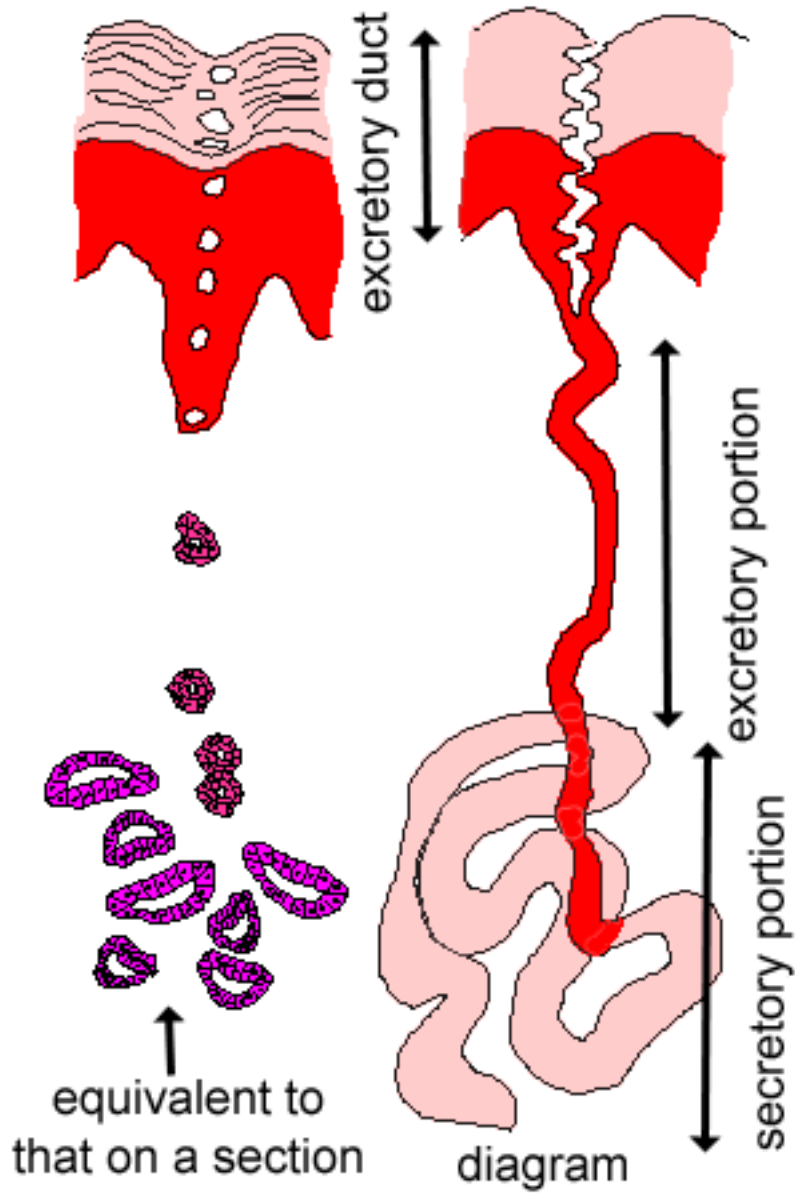
1.2.6.1 Nervous System Control of Sweating

Regulation of internal body temperature is one of the most fundamental functions of the body. The exact neurological pathways responsible for initiating sweating are not entirely understood, however results from animal studies have given some insight into the possible mechanisms (Shibasaki, Wilson, and Crandall 2006). Local heating of the preoptic region of the hypothalamus initiates sweating, vasodilation and panting, while preoptic cooling induces shivering (Smiles, Elizondo, and Barney 1976; Adair 1977). Microelectrode studies have identified two types of thermosensitive preoptic neurons: warm sensitive and cold sensitive. There is an increase in the firing rate of warm sensitive neurons both when the local preoptic temperature increases as well as when there is an increase in skin temperature (Boulant 1981). The set point at which sweat is initiated plays a vital role in temperature regulation and changes with acclimatisation and during exercise or heat exhaustion. During heat exhaustion there is a significant decrease in the sweating rate and in extreme cases sweating may be absent even when core temperatures are very high (Sato et al. 1989).

Efferent signals from the preoptic hypothalamus travel to the intermediolateral cell columns of the spinal cord. Axons of spinal cord neurons from the ventral horn run in the white rami communicantes where they combine with peripheral nerves and travel to the sweat glands. Sympathetic postganglionic nerve terminals are contained in and around the secretory coil, with some extending to the sweat duct (Shibasaki, Wilson, and Crandall 2006). Unlike normal sympathetic stimulation, Acetylcholine is the principal terminal neurotransmitter. Vasoactive peptide and periglandular norepinephine are also stimulators of sweat secretion however only 20-50% as effective as acetylcholine (Sato 1977).

1.2.6.2 Mechanism of Sweat Gland Function

The primary function of the eccrine sweat gland is the secretion of fluid and electrolytes as sweat. The sweat glands are innervated by sympathetic cholinergic nerve fibers, simulation of these fibres results in the secretion of an isotonic precursor fluid similar in consistency to plasma minus the plasma proteins (Wendt,

Van Loon, and Lichtenbelt 2007). The mechanism by which this occurs is not clearly explained, however some evidence suggests that the binding of acetylcholine to the receptor stimulates calcium influx and potassium release while stimulating a phosphatidylinositol - protein kinase cascade resulting in sweat secretion (Sato et al. 1989). The potassium movement out of the cell provides a potential gradient for the transport of sodium into the cell via the co-transporter which is then pumped out across the basolateral membrane in exchange for potassium.

1.2.6.3 Effect of Training on Sweat Gland Function

Physical training is known to modify the sweat rate of an individual by both increasing in the number of activated sweat glands as well as increasing the output per gland (Ichinose-Kuwahara et al. 2010). Several studies have reported significantly higher sweat rates in a trained group compared with a group of untrained subjects (Buono, McKenzie, and Kasch 1991; Ichinose-Kuwahara et al. 2010; Ichinose-Kuwahara et al. 2008).

A recent study by Ichinose-Kuwahara et al (2010) assessed sex differences in the sweat gland response to changes in exercise intensity with respect to subjects' physical training status. They found higher sweat rates in both males and females in the physically trained group. A further interesting finding from this study was that the increase in sweat rate was greater in males than females, indicating that the degree of improvement in sweat gland sensitivity is smaller in females compared to males. The reasons for this are currently not clear, however the authors of this paper conclude that sex differences exist with respect to the change in sweat gland size and/or cholinergic sensitivity with physical training. These findings are in support of the early work of Sato and Sato (1983) who reported that subjects who were judged to be poor sweaters had sweat glands which were smaller in size, and had lower secretion activity than good sweaters. The authors in this study speculated that the increased sweat rate seen as a result of physical training, may be a result of one or all of the following adaptations: increased cholinergic sensitivity of the gland; increased glandular hypertrophy or increased periglandular concentrations of acetylcholine (Sato and Sato 1983).

Buono and Sjoholm (1988) similarly found a positive linear relationship between pilocarpine- induced sweat rate and $\dot{V}O_2$max. The regression equation generated from their results would estimate that an individual with a $\dot{V}O_2$max of 65ml.kg^{-1}.min^{-1} would have a sweat rate that was 210% higher than a subject with a $\dot{V}O_2$max of 42ml.kg^{-1}.min^{-1}.

1.2.6.4 Rate of Sweat Loss

The amount of sweat lost during periods of work or exercise can be calculated from changes in body mass over time. These sweat losses are highly variable between individuals even when a person is performing a similar task in the same environmental conditions (Shirreffs, Sawka, and Stone 2006a). Factors such as metabolic rate, sex, acclimatisation status, fitness level and genetics are believed to account for this large variation (Shirreffs, Sawka, and Stone 2006a). There have been a number of studies which have been conducted during the last 5 years investigating sweat losses associated with different forms of exercise (A table summary of a number of these studies is included in Appendix B). The aim of the majority of these studies is to provide more accurate fluid intake recommendations for athletes competing in different sports. The variation in mean sweat rates across the studies is large. This may in part be due to differences in sex, the methodology used, subject characteristics and training and acclimatisation status.

Several studies conducted in male football players have shown average sweat rates in excess of 2L/h during training and competition (Godek, Peduzzi, et al. 2010; Godek, Bartolozzi, et al. 2010; Kurdak et al. 2010; Shirreffs et al. 2005). Similarly high sweat rates have been shown in male basketballers, volleyballers and soccer players (Hamouti et al. 2010). A recent study conducted in a small group of professional tennis players showed mean sweat rates during match play of 2L/h (Tippet et al. 2011).

Some recent studies have raised the question as to whether the mode of exercise training and the environmental conditions of the training environment can effect sweat gland adaptations and therefore affect the sweat rate of athletes. Henkin et al (2010) conducted a study to determine whether differences exist in the sweating responses of swimmers, runners and nonathletes. Ten endurance swimmers, 10

endurance trained runners and 10 non-athletes completed the study which consisted of 30 minutes of cycling exercise in a climate controlled heat chamber, set to 32 degrees, 40% relative humidity. The interesting finding from this study was that when exercising out of water, swimmers had significantly lower sweat rates than runners (0.9L/h Vs 1.5L/h). This confirmed the author's hypothesis that the condition of the training environment will impact on the training adaptations that occur at the level of the sweat gland. A study by Maughan et al (2009) found similarly low sweat rates (0.3L/h) in a group of swimmers. In this study, half of the subjects gained weight at the end of the 105 minute hard interval training session. The authors concluded that the results from this study indicate that swimming in pool temperatures of approximately 27 degrees results in very little challenge to thermoregulation and body temperature can be well maintained with little fluid intake.

A very different situation occurs in athletes training and competing in high intensity sports, or endurance based events in the heat. Team sports such as football and soccer have additional challenges when ad libitum fluid intake is limited to scheduled breaks in play. Further difficulties are experienced in endurance based sports such as ironman triathlon when athletes can be competing in the heat for multiple hours. In these situations drinking sufficient fluid volume to offset high sweat losses can often be challenging for athletes, particularly the elite level competitor.

It should also be mentioned here that while consuming sufficient fluid during an endurance event is challenging, at the other end of the spectrum, over drinking can place athletes at risk of developing exercise associated hyponatremia (Noakes 2007). Because of this it is vital that athletes are aware of their individual sweat rate and devise a fluid replacement strategy to match. Exercise associated hyponatremia is discussed elsewhere in this literature review.

A number of studies have reported body weight losses at the end of an exercise session in the range of 1.5-3% (Maughan et al. 2005; Godek, Bartolozzi, et al. 2010). While the 2000 National Athletic Trainers Association position statement suggested that fluid losses during exercise should not exceed 2% of pre-exercise body weight (Casa et al. 2000), Coyle (2004) argued that in cold environments these losses may

be tolerated. However, when an athlete begins exercise in a hypohydrated state, or loses greater than 2% of body weight during an exercise session in the heat, performance will be negatively affected and the athlete is at an increased risk of developing heat illness (Maughan et al. 2005).

1.2.6.5 Sweat Electrolyte Composition

1.2.6.5.1   Loss of Sodium in Sweat

As mentioned previously, the primary fluid secreted from the secretory coil of the sweat duct is nearly isotonic to plasma. It is therefore the rate of ductal sodium absorption and the sweat rate of an individual that determines total sodium loss. In the kidney, aldosterone stimulates sodium resorption via genomic and non genomic mechanisms (Lee, Miller, and Buono 2010) is thought to also have an effect on increasing ductal sodium absorption in the sweat gland (Ladell and Shephard 1961), and is proposed to increase with acclimatisation (Nielsen et al. 1993). The level of individual acclimatisation is therefore also a factor influencing the loss of sodium in sweat and will be discussed later in this literature review.

The majority of studies investigating sweat rate and sweat electrolyte composition have focused on highly trained young athletes (Maughan et al. 2009; Kilding et al. 2009; Henkin, Sehl, and Meyer 2010). Few investigations have focused on the industrial population who are at greater risk of heat injury due to lower aerobic fitness, higher body fat and are often older.

A study by Bates and Miller (2008) was the first to quantify likely sodium losses over a workshift during the summer and winter months in a group of untrained workers. 29 male outdoor workers had sweat collected from four sites on the body while cycling in a heat chamber (35 ºC, 50%RH) on two consecutive days in summer and winter. Results of the study showed significantly higher sweat rates combined with lower sodium losses in the summer period (53mmol/L) compared with the winter period (73mmol/L), indicating that workers were seasonally acclimatised to the heat. The authors in this study concluded that sodium losses of between 4.8-6g can be expected during a 12 hour workshift when workers are performing manual work, equivalent to 10-15g salt.

Far more research has been conducted investigating sodium loss in the athletic population. Horswill et al (2009) compared sweat sodium loss in a group of cramp prone footballers versus a reference group. Mean sweat sodium concentration in the cramp prone group (52.6mmol/L) was significantly higher than the reference group

(38.3mmol/L). A further interesting finding from the study was that in the cramp prone group, 3 out of the 6 subjects had plasma sodium values less than 135mmol/L at the end of the 2 hour training session, while the reference group had plasma levels all within the normal range. This provides some evidence to suggest that cramp prone athletes are at greater risk of sodium imbalance, particularly during periods of heavy training in the heat.

Godek at al (2010) reported mean sweat sodium losses of 52mEq/L in a cohort of American football players. These results are similar to sodium losses reported in elite level soccer players (Maughan et al. 2004; Maughan et al. 2005). With reported mean sweat rates in the range of 1.5 -2.5L/h in these athletes, daily sodium losses of 7-12g (17-30g salt) would be expected when these athletes are training in excess of 4 hours per day.

Currently the World Health Organisation (WHO) recommends that daily salt intake should be less than 5g/day (World Health Organisation 2007). In Australia, the National Heart Foundation recommends that the general population reduce sodium intake to less than 2300 mg/day (5.75g salt) (National Heart Foundation of Australia December 2006). These values were selected with the goal of preventing hypertension, one of the primary risk factors for the development of cardiovascular disease. There is little doubt that a large percentage of Australians exceed these government recommendations, with estimates that the average adult consumes upwards of 9g of salt per day (National Health and Medical Research Council 2005). It would be expected that athletes and manual workers would consume a higher number of calories and therefore have greater salt intake than the general population. This however has not been reported in the literature. Hinton et al (2004) showed mean sodium intakes of 2.94 ± 1.3g in a group of National Collegiate Athletic Association Division 1 athletes. Similarly, Gabor et al (2010) showed lower sodium intakes in a group of 309 professional female athletes than those reported in the non-athlete group from the National dietary survey. Athletes are likely to be more 'health conscious' and therefore may consciously restrict salt.

1.2.6.6 Effect of Sweat Rate on Sweat Electrolyte Composition

The variability in sweat rate and sodium loss that accompanies prolonged exercise has been well documented (Bergeron 2003; Godek, Godek, and Bartolozzi 2005; Shirreffs et al. 2005). However, information investigating the effect of exercise intensity on the sweat rate and sweat sodium relationship in the athletic population is lacking. An early study by Cage et al (1965) showed a strong positive linear correlation between sweat rate and sweat sodium concentration, with the authors concluding that with an increase in sweating rate there is a subsequent increase in sodium loss. Accurate measurement of exercise intensity however was not performed as intensity was varied according to perceived exertion rather than any objective measurement of workload. A further limitation of the study design was that sweat samples were only taken from one site on the body. More recent studies have found that sweat composition varies significantly between body sites (Bates and Miller 2008; Sato and Dobson 1970; Kondo et al. 1998). The results of the abovementioned study may therefore lack true accuracy due to limited sweat sampling. Kondo et al (1998) however found conflicting results when looking at the influence of exercise intensity on whole body sweating efficiency in mild thermal conditions, concluding that there is no relationship between exercise intensity and sweat rate. Again there are several methodological limitations to this study that may have affected the results. The sample size of 6 subjects may not have provided sufficient power to detect a difference. There was also no mention of prior testing of the subjects $\dot{V}$ O_2 max, therefore relative exercise intensity was not controlled. The variation in exercise intensity across the three trials was very insignificant and may not have been sufficient to detect a difference.

It is believed that above a certain sweat rate there is insufficient time for sodium reabsorption as fluid passes down the duct, resulting in the potential loss of large amounts of sodium through sweat (Buono, Ball, and Kolkhorst 2007). It has also been shown that at higher exercise intensities, when the heat produced by the working muscles is higher, the sweat rate will also be higher in order to dissipate this heat to the environment (Montain, Latzka, and Sawka 1995). What has not yet been thoroughly investigated is the effect that this increase in exercise intensity has on the rate of sodium loss through sweat. It may be hypothesized in this case that the sodium loss will also be

magnified due to a reduction in time for reabsorption through the sweat duct on the passage of sweat to the skin surface (Shamsuddin et al. 2005).

1.2.7 Physiological Mechanisms Regulating Salt Balance

Body fluid balance is tightly regulated by neuroendocrine control systems. The kidneys have the primary role of regulating fluid and sodium balance through water and salt excretion in the urine, restoring the ECF osmolarity to within normal levels almost instantly (Meneton et al. 2005). Water intake and output is controlled in order to maintain extracellular osmolarity at a set point of 290mosmol/L. The steroid hormone aldosterone, produced in the adrenal glands, enables conservation of sodium by allowing for sodium reabsorption to occur at both the level of the kidney and the sweat gland.

Sodium is by far the most abundant electrolyte in the extracellular fluid and therefore has a key role in regulating the volume of the extracellular fluid (Geerling and Loewy 2008). In response to an acute rise in sodium intake, extracellular fluid (ECF) volume expands leading to an increase in arterial pressure. There is a greater rise in plasma sodium levels in subjects who are hypertension prone due to a defect in the kidneys ability to excrete water and salt in the urine, this defect becomes more apparent with higher levels of sodium intake (De Wardener and Macgregor 1983). The kidney's ability to effectively excrete excess sodium and water in the urine declines exponentially with age, leading to significant increases in blood pressure with only small increases in salt intake in the elderly (Mohan and Campbell 2009).

Extracellular fluid volume however is unable to be either increased or maintained without sodium consumption. Chronic salt deprivation results in a decreased plasma volume and secondary increased plasma potassium, and can result in serious health consequences (Geerling and Loewy 2008). Chronic periods of salt deprivation have been shown in the rat model to cause growth retardation, reproductive problems, reduced bone mineral density as well as reduced muscle mass (Bursey and Watson 1983; Fine, Lestrange, and Levine 1987). In a classic study on experimental human salt deficiency, McCance (1936) showed that a short term sodium restricted diet resulted in nausea, loss of appetite, muscle cramps, fatigue and considerable weight loss. Due to the health consequences associated with salt deficiency, considerable research has focused on the inbuilt behavioral mechanisms in humans to regulate sodium appetite, promoting sodium intake after a period of salt deficiency (Geerling

and Loewy 2008). A classic study by Ricter et al (1936) showed that animals had an innate response to increase salt intake when it became necessary for survival. In this study, animals had their adrenal glands removed and were therefore unable to conserve sodium. Animals who were not given access to salt died within one week, animals who were allowed access to unlimited salt had a considerable increase in salt consumption which therefore allowed for their continued survival.

Several physiological mechanisms have been proposed to explain the increased salt appetite that occurs with salt deprivation. Dietary salt deprivation is the most potent stimulator of the salt conserving hormone aldosterone, which works to increase renal and sweat gland sodium conservation at the same time as also increasing sodium appetite (Geerling and Loewy 2008). Both of these processes aim to increase the extracellular fluid volume to within normal range (Geerling and Loewy 2008). In addition to this, a recent study has shown that a variety of brain areas are activated after prolonged exposure to a sodium deficient diet which underlines the increased preference for a hypertonic salt solution (Lu et al. 2009). Baroreceptors and the sodium concentration in the cerebrospinal fluid is also believed to influence the sodium appetite (Blackburn, Stricker, and Verbalis 1992).

1.2.8 Aldosterone

Aldosterone targets the epithelia of the kidney, sweat gland and colon to regulate sodium reabsorption and potassium secretion (Booth, Johnson, and Stockand 2002; Williams and Williams 2003). Transcellular transport is dependent on Na^+/K^+-ATPase activity, which acts to establish and maintain and electrochemical gradient to allow luminal entry of sodium and exit of potassium. The limiting step in sodium reabsorption is the number of active luminal sodium channels, while potassium secretion is limited by the number of active potassium channels (Booth, Johnson, and Stockand 2002). One of the primary actions of aldosterone is to increase the total number of ion channels, while stimulating apical ion channels to open (Williams and Williams 2003).

Dietary sodium intake plays a role in the aldosterone response as well as hormone synthesis. Allsopp et al (1998) examined the effect of different levels of dietary sodium intake on plasma aldosterone and sweat sodium concentrations. Subjects

consumed either a low (66mmol/day), moderate (174 mmol/day) or high (384mmol/day) dietary sodium intake for 8 days. Aldosterone concentration was the highest in the low sodium condition and lowest on the high sodium diet. The results of Hargreaves et al (1989) showed similar findings when comparing a low sodium diet (50mmol/day) with a moderate sodium intake (150mmol/day). In addition to this, sweat sodium secretion was lowest in the low sodium conditions in both studies, giving some evidence to suggest that aldosterone is involved in regulating sodium reabsorption at the level of the sweat gland as well as the kidney.

Exercise, heat stress and hypohydration have been shown to stimulate aldosterone release. Francesconi et al (1985) altered the hydration state of subjects from a euhydrated state, minus 3%, minus 5% or minus 7% of baseline body weight. Subjects then performed low intensity exercise (25% $\dot{V}O_2$max) in an environmental chamber (49°C, 20%RH) for 2 hours. Renin and aldosterone concentrations significantly increased with exercise in a 3% hypohydrated state, however high levels of hypohydration did not result in greater hormonal increases. Interestingly, between 5% and 7% hypohydration, there were no further decreases in plasma volume, leading the authors to conclude that aldosterone concentration may be related to the maintenance of plasma volume.

1.2.9 Development of Hyponatremia

Serum sodium levels within the body are tightly regulated between 135-144mmol/L. Hyponatremia is defined clinically as a serum sodium concentration of less than 135mmol/L (Siragy 2006). Hyponatremia is strongly associated with an increased risk of death. Even at mildly reduced sodium levels between 130-134mmol/L there is a 47% increased risk of hospital mortality (Waikar, Mount, and Curham 2009). The reason why this relationship exists is not clear, however, sodium concentration is critical to cellular function, nerve impulse transmission, muscle excitation and the maintenance of transmembrane electrical gradients (Waikar, Curham, and Bruneli 2011).

Over the last 15 years exercise associated hyponatremia (EAH) has been reported to have occurred in numerous endurance sporting events, particularly those lasting in excess of 3- 4 hours (Almond et al. 2005; Speedy et al. 1999; Rothwell and Rosengren 2008; Chorley, Cianca, and Divine 2007; Hew et al. 2003). Hyponatremia has also been reported to occur in the military when soldiers are required to perform extended periods of strenuous work in oppressively hot environments (O'Brien et al. 2001; Garigan and Ristedt 1999). In both cases, the failure to replace lost sweat sodium and/or the consumption of excess water volume, leads to a reduction in plasma sodium levels.

The incidence of exercise-associated hyponatremia (EAH) has been investigated in several marathon-running events around the world. Chorley, Cianca and Divine (2007) performed a prospective observational study of 96 marathon runners competing in the Houston Marathon between 2000−2004. Eighty seven percent of the runners had post-race serum sodium levels lower than their pre-race levels, while 22% of runners had post-race serum sodium levels less than 135mEq/L, which met the criteria for EAH. Interesting findings from this study were that the main risk factors for lower post-race serum sodium levels were larger amounts of fluid ingested during the race, lower pre-race serum sodium levels and less weight loss during the race. The authors also noted that slower runners were more likely to over-consume during the race, as well as also being exposed to more harsh environmental conditions as they are racing into the hotter parts of the day.

Almond et al (2005) found a similar correlation between weight gain during the race and post-race serum sodium levels in 488 runners competing in the 2002 Boston marathon. At the completion of the race 13% had serum sodium levels less than 135mEq/L, while 0.6% had critical hyponatremia with serum levels below 120mEq/L.

A similar study was performed investigating the prevalence of EAH in a group of marathon runners in the 2006 Zurich marathon (Mettler et al. 2008). One hundred and thirty six runners were recruited. Body mass, plasma sodium and osmolality were recorded pre and post-race. Only 3% of runners developed EAH and no symptomatic cases were reported. There was however a direct correlation between fluid intake and post-race serum sodium levels. The environmental conditions were quite mild, approximately 10 degrees with constant light rain and low wind speed throughout the race, which may explain the low incidence of EAH reported. The results of this study suggest the risk of EAH is significantly reduced when the marathon is raced in mild environmental conditions.

Speedy et al (1999) reported pre and post-race serum sodium values in 330 competitors in the 1997 New Zealand ironman triathlon. Eighteen percent of the 330 finishers had serum sodium values below 135mEq/L of these, only 18 (31%) sought medical attention post-race suggesting that in the majority of cases the condition was asymptomatic. Eleven of the 58 competitors with EAH had severe hyponatremia (serum sodium below 130mEq/L). Similar results were found when comparing pre and post weight with post-race serum sodium values. Athletes with the lower serum sodium levels post-race either gained weight or lost small amounts over the course of the ironman event. This again suggests that these athletes had consumed plain water or another hypotonic solution in excess to body needs during the race.

Prior to this, the same authors conducted an investigation in another popular endurance event which consisted of a 67km paddle, 149km cycle and 23.8km run (Speedy et al. 1997). Forty eight athletes had serum sodium levels measured pre and post-race with the average post-race sodium concentration being 139mEq/L. Only one athlete had levels below 135mEq/L. The authors speculate that the reason for these different finding was due to the nature of the event and the limited number of

aid stations providing fluid to athletes. Body weight was reduced on average by 2.5% at the end of the event indicating that athletes had lost a significantly greater percentage of body weight than the previously mentioned studies.

The results from observational studies, case reports and case series have noted that there are three major risk factors for the development of exercise associated hyponatremia: significant weight gain due to water intake during an event; longer finishing times and a BMI of less than 20 (Carter 2008). Other risk factors include excessive sodium losses in sweat and insufficient sodium in food consumed. The consumption of a carbohydrate-electrolyte drink during an endurance event can significantly delay or prevent the occurrence of EAH (Montain, Cheuvront, and Sawka 2006).

Mild chronic hyponatremia is a common electrolyte disturbance with a prevalence of 2-4% in the general population, 7-11% in the elderly and 42% in hospitalized patients (Hawkins 2003). Chronic hyponatremia can develop over several days when body mechanisms fail to maintain sodium homeostasis. This may be from profuse sweating with inadequate solute intake, resulting in a decrease in extracellular fluid volume, compromising the body's ability to thermo regulate (Hamilton, Dickson, and Smith 2006). Cases of hyponatremia have been reported in military and civilian personal working in extreme environmental conditions in southern Iraq (Hamilton, Dickson, and Smith 2006). While the majority of these cases were due to acute water intoxication leading to severe acute hyponatremia, the failure of sodium homeostasis in these extreme temperatures was reported to have been further compounded by poor dietary salt intake during the patrol period (Hamilton, Dickson, and Smith 2006).

Chronic hyponatremia is often thought to be asymptomatic due to the brain adaptation to hypo-osmolality (Renneboog et al. 2006), for this reason few studies have investigated the effects of long term chronic hyponatremia on the central nervous system. A rat study conducted by Miyazaki and colleagues (2010) showed that chronic hyponatremia may impair memory function even in asymptomatic rats. The mechanisms which may have led to memory impairment are believed to be related to both brain swelling and abnormalities in organic osmolytes (Miyazaki,

Ohmoto, and Hirose 2010). The consequences of a long-term decline in plasma sodium have recently been shown to increase the incidence of falls as well as being associated with impaired gait and attention deficits. Renneboog et al (2006) showed that hyponatremia caused more attention deficits than did a blood alcohol reading of 0.6g/L in a group of age and sex matched subjects. In this study the impaired gait and attention deficits occurred with sodium levels between 132-134mmol/L.

There has been some recent evidence to suggest that chronic hyponatremia decreases bone mineral density. Kinsella et al (2010) showed that mild hyponatremia is significantly associated with fracture occurrence independent of bone mineral density. The authors of this paper concluded that hyponatremia is an easily identified and modifiable risk factor for fracture occurrence.

In summary the information from the current literature regarding hyponatremia suggests that:

- Exercise associated hyponatremia is most commonly developed as a result of excessive fluid consumption during an event leading to plasma dilution
- EAH is strongly associated with significant weight gain due to water intake during an event, longer finishing times and a BMI of less than 20.
- Chronic hyponatremia may develop over several days or longer when sodium intake is insufficient to replace losses

Based on this information an athlete or worker is unlikely to develop low plasma sodium (hyponatremia) over the course of their event, or work shift, if an appropriate electrolyte replacement fluid, or salt containing food, is consumed in appropriate amounts to partially replace losses. However, if a high salt sweater performs episodes of prolonged work or exercise in the heat for several days, cumulative sodium losses would potentially be large enough to result in chronic hyponatremia. Athletes or workers with high sweat rates, who are actively restricting sodium intake in line with the current sodium intake recommendations, are particularly at risk.

1.2.10 Heat Acclimatisation

Heat acclimatisation refers to the physiological adaptation of the human body to heat (Rowlinson et al. 2013). It has been well recognised that when a person becomes heat acclimatised by exposure to increased temperatures for several days, there are a number of physiological adaptations that take place enabling them to thermoregulate more efficiently (Wendt, Van Loon, and Lichtenbelt 2007). These adaptations include decreased rectal temperature, decrease in sodium concentration in sweat and lowered heart rate during exercise, while increasing sweat rate, sweat sensitivity and plasma volume (Armstrong and Maresh 1991; Wendt, Van Loon, and Lichtenbelt 2007). This results in a significant decrease in physiological strain, a decrease in perceived exertion, leading to an improved capacity to exercise or work for prolonged periods in the heat, while reducing the risk of heat illness (Tian et al. 2011). There is some disagreement in the literature as to the duration, intensity and climatic conditions required to induce heat acclimatisation. The next section of this review will discuss the key findings of papers investigating the effects of acclimatisation on thermoregulation.

The expansion of plasma volume is one of the key effects of heat acclimatisation, allowing for cardiovascular stability increasing stroke volume and maintaining heart rate (Wendt, Van Loon, and Lichtenbelt 2007). Patterson et al (2004) measured plasma volume, extracellular fluid (ECF) and interstitial fluid volume (ISF) before and after a period of heat acclimation. The authors concluded that either short (8 days) or long term (22 days) acclimation resulted in a generalised expansion of the entire extracellular fluid volume resulting in plasma volume expansion. Following more prolonged heat acclimation, (day 22) the elevation in plasma volume was much greater than the expansion of the ECF and ICF (Patterson, Stocks, and Taylor 2004).

Numerous studies in the literature have shown that following a period of heat acclimatisation there is a significant increase in the secretory capacity of the eccrine sweat gland, as well as morphological changes to the gland itself. Sato et al (1990) in a study using monkeys found in vitro and in vivo methancholine-induced sweating was increased significantly after a period of 9 months of heat acclimation. The key finding from this paper was that the increased sweating capacity was brought about by significant increases in the size of the eccrine sweat glands. The sensitivity to

nervous stimulation was also increased following the acclimation period. In a classic study, Peter and Wyndham (1966) assessed the activity of sweat glands during exercise in a hot and humid environment before and after heat acclimatisation. Six unacclimatised African mine labourers exercised for 4.5 hours by stepping on and off an adjustable stool, in a hot and humid environment (32℃, 90% RH) both before and after a period of heat acclimatisation. Sweat rates were significantly increased mainly due to increased glandular activity, particularly on the back.

More recent studies have found similar increases in sweat rate associated with acclimatisation or acclimation to the heat. Bates and Miller (2008) aimed to quantify sweat and sodium losses during work in the heat in both the summer (when subjects were assumed to be heat acclimatised) and winter (when subjects were assumed to be unacclimatised). Average sweat rates of 0.47L/h were reported in summer compared with 0.4L/h. in winter. Chinevere et al (2008) found after 10 days of heat acclimation, sweat rates in a sample of eight male subjects had increased by 6%. Similar findings have been reported by Cheung and McLellan (1998) and Magalhaes et al (2010).

The effect of sex on thermoregulatory capacity has been previously discussed in this literature review; however, there are additional gender differences at the level of the sweat gland that occur with heat acclimation. Buono et al (2009) showed that humid heat acclimation resulted in a 60-70% increase in pilocarpine induced sweat rate in both men and women. The interesting finding from this study was that whole body sweat rate (WBSR) increased by 20% in the male subjects however there was no significant increase in the women. These results suggest that the lack of a significant increase in WBSR seen consistently in women is not due to an inability to improve sweat gland function with acclimation, but that the peripheral sweat capacity in women during exercise in humid conditions is suppressed. These results are supported by several other studies in the literature (Avellini, Kamon, and Krajewski 1980a; Wyndham, Morrison, and Williams 1965). The physiological mechanism by which females avoid the 'wasteful' increase in sweating in a humid environment is currently unknown. It has been suggested that women have a more sensitive feedback mechanism from the wetted skin surface therefore limiting the dripping of non-evaporated sweat (Avellini, Kamon, and Krajewski 1980a).

Numerous studies in the literature have reported a reduction in sweat sodium following a period of heat acclimatisation (Buono, Ball, and Kolkhorst 2007; Bates and Miller 2008; Daly and Dill 1937; Kirby and Convertino 1986; Smiles and Robinson 1971; Nielsen et al. 1997; Chinevere et al. 2008). This is believed, although not confirmed, to be due to the role of the renin-angiotensin-aldosterone system in conserving Na^+ and Cl^- allowing for the maintenance of extracellular fluid volume.

In a classic study, Kirby et al (1986) found 12% increases in sweat rates following a period of 10 days of heat acclimatisation. Sweat sodium losses were reported to decrease by 59% from 88mmol/l to 42mmol/L. More recently Nielsen et al (1997) found similar results, showing that following eight days of humid heat acclimatisation sweat rates increased by 26%, while sweat sodium concentration decreased from 107mmol/L to 70mmol/L. Buono et al (2007) reported a significant decrease in sweat sodium and sodium osmolarity for any given sweat rate, following a 10 day acclimatisation period in eight healthy male subjects. The slope of the relationship between sweat rate and sodium loss was not affected, however the relationship was shifted to the right with a significantly reduced y-intercept. In the study mentioned previously, Bates and Miller (2008) showed a significant reduction in sweat sodium values in the summer months when the subjects were assumed to be heat acclimatised (63 Vs 42mmol/L).

There are several advantages of an increased sweat rate coupled with the reduced sweat sodium concentration that occurs following heat acclimatisation. A more dilute sweat results in a relative increase in the number of solutes, primarily sodium and chloride remaining within the extracellular space. This increases the osmotic pressure in the ECF, allowing for the redistribution of fluid from the intracellular space, resulting in the ability to maintain plasma volume and therefore allow the body to prevent a rise in core body temperature (Sawka and Montain 2000). In addition to this, high sweat sodium concentration is one of the factors increasing the risk of developing hyponatremia during exercise (Montain, Cheuvront, and Sawka 2006). The reduction in sweat sodium concentration that accompanies heat acclimatisation may therefore be advantageous to athletes competing in endurance events of prolonged duration.

This sodium conservation mechanism is believed in part to be due to the increased level of aldosterone associated with acclimatisation (Nielsen et al. 1993). However, very few studies have investigated the effect of aldosterone at the level of the sweat gland and therefore the role of aldosterone in sweat gland secretion is relatively unknown.

An early study by Ladell et al (1961) used a 10 day heat acclimatisation protocol to investigate the effects of the aldosterone inhibitor spironolactone on sweat sodium concentration. The results showed sweat sodium concentration to be 18mmol/L higher on day eight when spironolactone was administered. These results have been referenced frequently in the literature, however one major limitation of this study was the failure to control for the increase in sweat rate that occurs due to spironolactone administration. Previous results have shown a linear relationship between sweat rate and sweat sodium excretion; these results may therefore be of limited value. Lee et al (2010) investigated the effect of spironolactone on urinary and sweat sodium concentration when sweat rate is controlled. Fifteen subjects performed two 90-minute exercise bouts at varying intensity, once after the administration of spironolactone and once after a placebo. Spironolactone was shown to have an effect at the level of the kidney where urinary sodium excretion was significantly higher with the treatment vs. placebo. There was no effect at the level of the sweat gland. The authors concluded that both genomic and non-genomic mechanisms of aldosterone action are evident in the kidney and the sweat gland. Results from this study suggest that different isoforms of the mineralocorticoid membrane receptor for aldosterone may be present in the sweat gland.

1.2.11 Literature Review Summary

The sweating mechanism is the body's primary means of dissipating heat generated from the active muscles during prolonged work or exercise in the heat. High fluid and electrolyte losses are incurred as a result of this, resulting in dehydration if not adequately replaced. If fluid losses are greater than 2% of body weight, performance decrements, both physical and mental, will result. In the case of athletes this will impair performance and in workers will decrease productivity while increasing the risk of workplace accidents. If fluid losses are prolonged or severe serious heat illness may develop, posing a serious health risk.

The replacement of electrolyte losses in sweat has had far less attention in the literature. Sodium replacement guidelines for athletes and workers are limited and often conflicting. In addition to this, current public health guidelines for sodium restriction in the general population can cause confusion amongst these groups. The adequate replacement of sodium, particularly in heavy salt sweaters is vital in order to prevent fluid and electrolyte disturbances such as heat cramps and hyponatremia.

Heat acclimatisation and physical training are known to improve the ability to tolerate prolonged periods of work or exercise in the heat. Fluid and electrolyte replacement is even more vital in a physically trained and acclimatised individual as sweat losses may be considerably greater.

Therefore the primary aim of this thesis is to further investigate fluid and electrolyte losses in these two at-risk populations. The proposed outcome of this research is an improved understanding of the fluid and electrolyte losses associated with prolonged work in the heat, which will enable more specific guidelines for replacement to be provided.

Chapter 2: **Plasma sodium levels and dietary sodium intake in manual workers in the Middle East**

2.1 Introduction

Dubai is situated on the Arabian Gulf coast in the Northeast of the United Arab Emirates. At the time that this study was undertaken, Dubai was engaged in a building boom, with over 700,000 expatriate workers employed throughout the country in construction and other industries. Situated directly within the Arabian Desert, Dubai experiences an arid subtropical climate with summer temperatures regularly reaching in excess of 45 degrees, some of the harshest in the world. The thousands of expatriate workers are required to perform arduous physical tasks in hostile environments for up to 12 hours per day. Working in such extreme conditions places high demands on the thermoregulatory mechanisms of the body, which can result in dehydration and electrolyte imbalance. If this is prolonged or severe the health of the workers may be seriously at risk.

The Dubai Dry Docks employ approximately 8500 workers in predominately manual roles, from over 30 countries throughout the world. It is hypothesised that as a consequence of the harsh environmental conditions in which they work, sweat rates and sodium losses are extremely high. Previous studies have estimated fluid losses in manual workers in hot environments of up to 10-12L/day with sodium losses of up to 6g over a work shift (Bates and Miller 2008). It is essential that these losses are replaced through fluid and dietary sodium intake in order to prevent serious fluid and electrolyte imbalances that may lead to dehydration, fatigue, heat illness and significantly reduced work rate and overall productivity, as well as increasing the number of work related accidents.

The dockyard workers live in labour camps where all food and fluid is provided at three specific times during the day. All workers are provided with the same base diet and snacking is seldom. As the staple diet is largely made up of pulses, rice and very small quantities of meat, with very low sodium content, it may be inferred that these workers are consuming a diet with insufficient sodium to replace estimated sweat losses

particularly in the summer months when temperatures are extreme. It is therefore postulated that these workers are at risk of developing chronic hyponatremia, a potentially serious abnormality of electrolyte balance where serum sodium drops below 135mmol/L, (Noakes et al. 1990; Speedy et al. 1999). Over the last 15 years this condition has been reported in many athletic populations where athletes are exercising in harsh environmental conditions for prolonged duration (Noakes et al. 1990; Noakes 2002; Speedy et al. 1999). Hyponatremia has also been reported to occur in the military when soldiers are forced to perform extended periods of strenuous work in oppressively hot environments (O'Brien et al. 2001; Garigan and Ristedt 1999). In both cases, the failure to replace lost sweat sodium and/or the consumption of excess water volume leads to a reduction in plasma sodium levels. What has not previously been investigated is the incidence of hyponatraemia in a population of manual workers exposed to similarly harsh environmental conditions for 12 hours/day while eating a predominantly starch based diet. The communal nature of food consumption makes this population a particularly unique group to study, while allowing for accurate unbiased dietary assessment to occur.

The need for adequate fluid intake during work in these conditions has been well documented and workers are generally well aware of the importance of adequate hydration. The sodium loss that accompanies high sweat losses has not been so extensively investigated. The current World Health Organisation (WHO) recommendations state that on a global scale, individuals should aim to reduce salt consumption to below 5g per day. This is a public health measure aimed at preventing hypertension and the development of cardiovascular disease. While these recommendations may be appropriate for the general population, the manual worker has substantially greater sodium losses and therefore is hypothesized to require a higher sodium intake.

The aim of the present study was to investigate the hypothesis that workers consuming a traditional low salt diet and working in hot conditions are at risk of chronic hyponatremia. Firstly plasma sodium levels in a group of manual labourers during both the summer and winter months were investigated for evidence of hyponatraemia. A further aim was to assess the relative adequacy of the current diet provided to workers with focus on the total salt content of the menu. The results of this study will identify

whether this population consumes sufficient dietary sodium to offset the high sweat losses, particularly during the extreme summer months.

The key objectives of this study are:

1. To investigate the risk of chronic hyponatremia in manual workers in the Middle East by comparing plasma sodium values in the summer and winter months.
2. To analyse the sodium intake of workers to determine relative adequacy.

2.2 Methods

The study took place at the DryDocks in Dubai, United Arab Emirates. Part one of the study took place at the end of the summer period (July) while part two took place at the end of the winter period (February). Both trials were completed within the same 12-month period. Plasma sodium levels were tested in the same subjects in the summer and winter trials at the same time of day (between 7-8am) and in a fasted state. The dietary intake data was collected during the winter period.

2.2.1 Subjects

A total of 44 subjects were studied at the end of the summer months and 38 subjects were repeated at the end of winter, the age distribution is shown in Figure 1. The workers who were involved in the study were from Pakistan (13 workers), India (10 workers) and Bangladesh (21 workers). Workers were all employed in various roles (Dockyard workers (10 workers), steel fabricators (17 workers), mechanics (4 workers) and some working in hull treatment (13 workers)) at the DryDocks in Dubai, United Arab Emirates and were randomly selected to take part in the study. Each subject was asked to complete a written informed consent form prior to the study commencement. Permission was sought and granted from the management of the medical centre at the DryDocks and the research protocol explained to all subjects and medical staff involved in the study.

2.2.1.1 Sample Size Consideration

Previous studies have reported average plasma sodium values in a group of subjects to be between 135- 145mmol/L (Renneboog et al. 2006). It is expected that plasma sodium values will be lower in the summer months due to high sweating rates. In order to detect a moderate effect size of 0.5 with 80% power, 34 subjects are required.

2.2.1.2 Inclusion criteria

A full blood profile was taken prior to the start of the study by a trained phlebotomist to screen for any chronic health problems including diabetes and impaired liver and kidney function. Workers using any regular medication were excluded from the study.

Criteria for participation were:

- Aged between 18-50 years.
- Available for testing during both the summer and winter periods.
- No medical conditions requiring medication.

The protocol for testing as outlined below in the summer months was repeated in the winter months. Six out of the 44 subjects who were tested in the summer period were unable to be retested during winter due to illness or relocation of employment. The number in the winter study was therefore reduced to 38. When comparison between summer and winter was made, the data for these six subjects was removed for statistical analysis.

Figure 1. **Age distribution of subjects participating in Study 1 investigating plasma sodium levels and dietary sodium intake in manual workers in the Middle East (n = 44)**
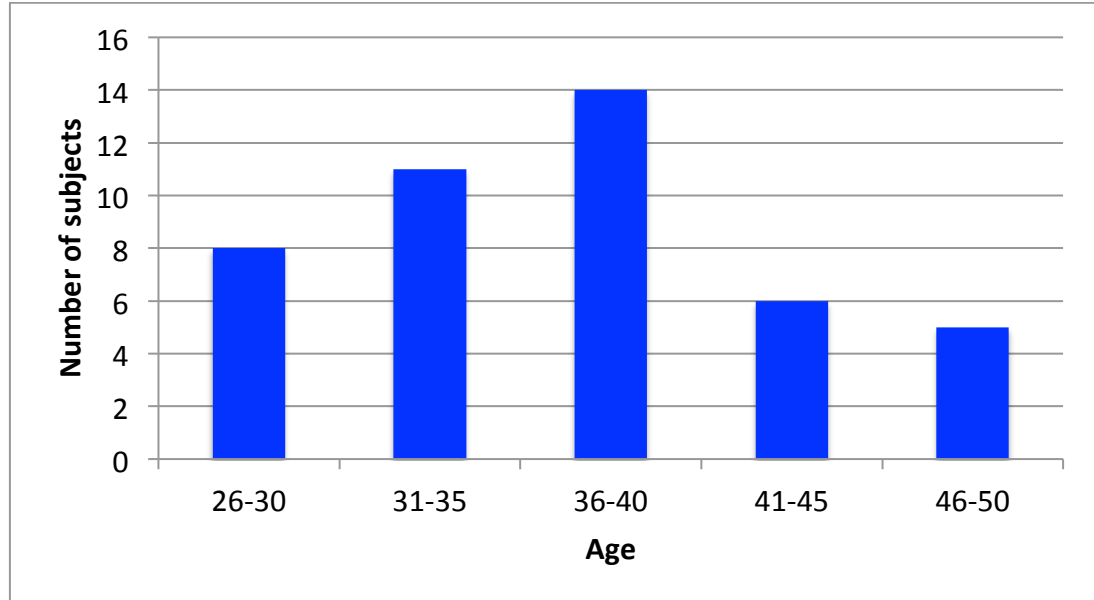
2.2.2 Climatic Conditions at the Study Location

The study was conducted in the summer and winter months within the same 12-month period. Dubai experiences some of the harshest environmental conditions in the world with extreme temperatures and high humidity for most of the day particularly during the summer months. Table 1. below shows the seasonal weather averages for the study location. As shown below average summer temperatures for July range from 29-39ºC with 80% humidity, compared to the month of February when temperatures range from 16-24ºC with 89% humidity.

Table 1.   Average minimum and maximum temperatures (ºC) and average maximum humidity (%) for Dubai, United Arab Emirates.

SOURCE: World Meteorological Organisation

Month	Minimum temperature (ºC)	Maximum temperature (ºC)	Average maximum humidity (Avg %)
January	14	23	90
February	16	24	89
March	18	27	85
April	21	32	83
May	24	36	80
June	27	38	85
July	29	39	80
August	30	39	82
September	27	37	85
October	24	34	87
November	20	29	86
December	16	25	88

2.2.3 Experimental Protocol

2.2.3.1 Measurement of Plasma Sodium Levels

Full blood tests including LFT, FBC, U&E and glucose were taken from all subjects in both the summer (July) and winter (February) months to check general health and compare blood electrolyte profiles (see Table 2. below for complete list of blood variables assessed). Any worker with any medical condition with potential to affect the results was excluded from the study. The venous blood sample was taken in the morning prior to eating (between 0700- 0800 hours) by a trained phlebotomist. An approved accredited pathology laboratory completed all analysis.

Table 2. Blood variables assessed

Full Blood Count (FBC)	Haemoglobin (HGB)
	Haematocrit (HCT)
	Mean Cell Volume (MCV)
	Platelets (PLT)
	Neutrophils (Neutr %)
	Lymphocytes (Lymph %)
	Monocytes (Mono %)
	Eosinophils (Eosin %)
	Basophils (Baso %)
Urea and Electrolytes	Sodium (Na)
	Potassium (K)
	Urea
Liver Function Test (LFT)	Alkaline Phosphatase (ALK Phos)
	Gamma Glutamyl Transpeptidase (GGT)
	Aspartate Transaminase (AST)
	Alanine Transaminase (ALT)
	Bilirubin
	Total Protein
	Albumin
	Lactate Dehydrogenase(LDH)

2.2.3.2 Measurement of Dietary Intake

The candidate, an Accredited Practicing Dietitian conducted an assessment of the diet of the workers in two parts. Part one involved recording and analysing menus and recipes to allow for an estimation of total daily salt intake. Menus and recipes from all food prepared and served in the mess hall were collected and analysed over a 3 day period. Foodworks 2007 nutrient analysis package was then used to estimate the total daily macronutrient intake along with total daily salt intake. Estimation of serve size was conducted through observation of meals served as well as measurement of plated food using household measures. Table salt was provided on a separate table next to the bain-marie. The use of table salt at meals was observed and an estimation of the quantity added was recorded. Part two of the study involved individual interviews with the workers, 38 workers were available for this interview where questions relating to individual dietary practices were asked. Table 3. outlines the questions asked to the workers during the interview. A translator was required for this interview as many of the subjects did not speak adequate English (Figure 2).

Table 3.   Interview questions asked to workers (n = 38) during the dietary

assessment component of Study 1 investigating plasma sodium levels and

dietary sodium intake in manual workers in the Middle East.

Question
1. Do you normally snack outside of meal times (i.e. Breakfast/lunch/dinner)?
2. Do your eating habits change in the summer vs winter period?
3. Do you normally add salt to your food?
4. What do you normally drink during a work shift?

Figure 2.   The dietitian conducting interviews with individual workers participating in Study 1 investigating plasma sodium levels and dietary sodium intake in manual workers in the Middle East (n = 38).

2.3 Statistical Analysis

The mean, 95% confidence interval for the mean, median and standard deviation were calculated for all parameters. Data was assessed for normality and between group comparisons of plasma sodium values and daily sodium intake. Statistical differences were analysed using a two-tailed paired samples t-test to determine differences between plasma sodium values in the summer and winter months. All statistical analysis was done using SPSS for Windows (SPSS Inc., Chicago, IL).

2.4 Results

2.4.1 Physical Characteristics

The physical characteristics, including age, height, weight and BMI of the subject group are shown in Table 4.

Table 4. Physical characteristics of subjects participating in study 1 investigating plasma sodium levels and dietary sodium intake in manual workers in the Middle East.

	Mean (± Std Dev)
Age (years)	37 ± 6.1
Height (cm)	168 ± 6.9
Weight (kg)	67.5 ± 12.5
BMI (kg/m^2)	23.8 ± 3.7

2.4.2 Measurement of plasma sodium levels in the summer and winter months

Results from the blood electrolyte profile in summer and winter showed that a high proportion of subjects (55%) were found to be hyponatraemic in the summer months (Figure 3), all remaining values were in the lower part of the reference range. 5% of subjects had serum sodium levels less than 130mM. Only 8% of subjects had plasma sodium values less than 135mmol/L in the winter months. There was a significant difference (p=0.000) between mean plasma sodium values in the summer and winter months with the mean plasma sodium level being 134.39 in summer compared to 137.09 in winter.

Figure 3.   Comparison of serum sodium values in the summer and winter periods in Study 1 investigating plasma sodium levels and dietary sodium in manual workers in the Middle East.
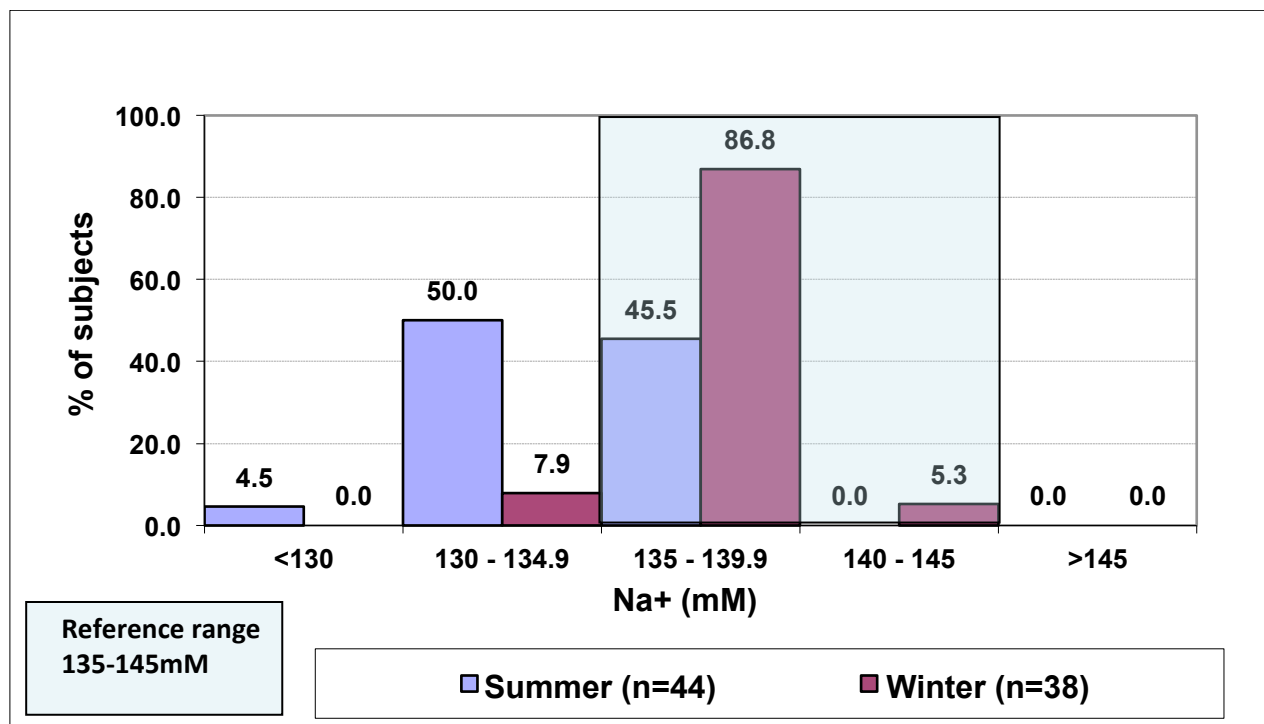
2.4.3 Dietary Assessment

Tables 5,6 and 7 summarise the dietary assessment data collected at the site during the winter period. On average a worker would consume an estimated 2000-3000mg of sodium (5-7.5g salt) in food and fluid per day. The total sodium content of the lunch meal was particularly low averaging approximately 600mg (1.5g salt). In addition to this workers would be provided with a maximum of 1L per day of electrolyte replacement fluid that would provide between 200-400mg sodium (500-1000mg salt). This is only provided in the summer months. Additional table salt is available at all meals: 43% of workers indicated that they added salt to their meals however few workers were observed to do so. Figures 6 and 7 illustrate a typical breakfast, lunch and dinner meal served in the Asian mess daily.

2.4.4 Further Observations from the Dietary Assessment

- On average each worker would be provided with at least 5 cups of cooked plain white rice per day.
- Lunch and dinner meals are always served with dahl and a small portion of a red meat or chicken based curry.
- There is little variation from day to day in the nutritional composition of the meals provided (0
- Workers are served an identical portion of food at each meal and are not allowed to return for a second helping (Figure 8).
- Observation of mealtime practices revealed that the majority of workers were seen to always finish their entire meal.
- Water, tea and fruit cordial are always served at meal times.
- All meals are prepared onsite with very little use of packaged or processed food (Figure 4).

Photos taken while conducting the dietary assessment component of Study 1

Figure 4.   Preparation of meals in the Asian mess kitchen for 5000 workers

Figure 5.   The Asian dining hall

Figure 6.   A typical breakfast served in the Asian Mess Hall

Figure 7.   A typical lunch/dinner served in the Asian Mess Hall

Figure 8.   Each worker is served an identical type and portion of each meal.

2.4.4.1 Interviews with Workers

93% of the workers stated that they did not consume any other food outside of what was provided in the mess at breakfast, lunch and dinner. This information further confirmed the consistency in dietary intake of the workers. The 3 subjects who did snack between meals stated that they would only consume fruit or fruit juice on occasion; these foods would not contribute significantly to overall daily salt intake. 94% of the workers stated that they do not change anything about their diet between the summer and winter period and that heat did not affect their intake. 43% of the workers stated that they regularly add table salt to their lunch and dinner meals. One or two small pinches of salt was the typical amount added which would provide an estimated 250-500mg of sodium (625- 1250mg salt).

All workers stated that they did consume an electrolyte replacement solution during working hours during the summer months. There is a limit of 1L of electrolyte solution per worker during this period while water consumption is unrestricted. Medical staff indicated that this restriction was placed as a precaution to avoid excessive consumption of sugar and salt.

Table 5.   Dietary analysis including total energy (kJ), protein (g),

carbohydrate (g), fat (g) and sodium (mg) content for day one of the

dietary assessment component of Study 1 conducted during winter.

Menu Day 1	Energy (kJ)	Protein (g)	Carbohydrate (g)	Fat (g)	Sodium (mg)
Breakfast	*4411*	*20.9*	*126.7*	*49.9*	*1359*
3 large sandwich slice bread,white	981	8.0	43.0	2.5	550
3 tsp butter	419	0.1	0.1	11.3	109
3 tsp jam	219	0.0	13.3	0.0	2
5 slices paratha	1751	6.3	42.0	24.6	339
1 cup seera - sweet rice	721	2.6	20.1	9.2	13
1 cup chana masala - chickpea curry	318	3.7	8.0	2.2	316
Lunch	*3937*	*42.0*	*132.1*	*22.8*	*624*
1 cup yellow dahl	493	8.5	17.1	0.8	155
3 cup (cooked) white rice	1746	16.4	79.7	1.2	36
3 Tbls chicken biryani	801	12.5	2.2	14.8	198
1 apple	483	0.6	26.5	0.2	2
1 cup rajma masala	414	3.9	6.5	5.8	233
Dinner	*3415*	*39.2*	*118.3*	*16.9*	*773*
2 cup (cooked) white rice	1164	10.9	53.6	0.8	24
3 slices tandoori roti	1214	8.1	44.6	8.0	420
1 cup yellow dahl	493	8.5	17.1	0.8	155
3 Tbls beef masala	375	10.8	1.5	4.2	92
1 cup green salad	167	0.7	1.8	3.0	82

Table 6.   Dietary Analysis including total energy (kJ), protein (g),

carbohydrate (g), fat (g) and sodium (mg) content for day two.

Menu Day 2	Energy (kJ)	Protein (g)	Carbohydrate (g)	Fat (g)	Sodium (mg)
Breakfast	*4624*	*31.6*	*121.8*	*53.3*	*1235*
3 large sandwich slice bread,white	981	8.0	43.0	2.5	550
3 tsp butter,	419	0.1	0.1	11.3	109
3 tsp jam,	219	0.0	13.3	0.0	2
5 slices paratha	1751	6.3	42.0	24.6	339
1 cup oats	601	3.7	21.2	4.2	7
0.5 cup scrambled egg	651	13.3	2.2	10.6	228
Lunch	*4105*	*44.7*	*134.7*	*23.8*	*464*
1 cup yellow dahl	493	8.5	17.1	0.8	155
3 cup (cooked) white rice	1746	16.4	79.7	1.2	36
3 Tbls chicken kadhi	821	15.2	4.6	12.6	151
1 apple	483	0.6	26.5	0.2	2
4 Tbls cabbage thoran	460	1.9	4.0	8.9	105
1 cup coleslaw salad	99	1.9	2.6	0.1	15
Dinner	*3651*	*34.4*	*118.2*	*25.5*	*1005*
2 cup (cooked) white rice	1164	10.9	53.6	0.8	24
3 slices tandoori roti	1214	8.1	44.6	8.0	420
3 Tbls sausage masala	779	6.8	3.3	15.9	406
1 cup yellow dahl	493	8.5	17.1	0.8	155

Table 7.   Dietary Analysis including total energy (kJ), protein (g),

carbohydrate (g), fat (g) and sodium (mg) content for day 3

Menu Day 3	Energy (kJ)	Protein (g)	Carbohydrate (g)	Fat (g)	Sodium (mg)
Breakfast	*3903*	*19.3*	*117.5*	*37.4*	*1145*
1 cup uppma	631	3.6	14.6	8.1	277
3 slices paratha	1050	3.7	25.2	14.8	200
1 cup cooked oats	601	3.7	21.2	4.2	7
3 large sandwich slice bread,white	981	8.0	43.0	2.5	550
3 tsp butter,	419	0.1	0.1	11.3	109
3 tsp jam,	219	0.0	13.3	0	2
Lunch	*4263*	*42.2*	*151.6*	*22.6*	*654*
3 cup (cooked) white rice	1746	16.4	79.7	1.2	36
1 cup yellow dahl	493	8.5	17.1	0.8	155
2 slices tandoori roti	809	5.4	29.7	5.3	280
3 Tbls aloo baingan	255	0.8	3.4	4.7	132
3 Tbls fish fry	477	10.5	1.1	10.3	49
1 apple	483	0.6	26.5	0.2	2
Dinner	*3428*	*44.2*	*103.8*	*21.2*	*571*
2 cup (cooked) white rice	1164	10.9	53.1	0.8	24
3 Tbls lahori chicken curry	812	16.6	4.3	12.0	132
3 Tbls aloo palak	555	3.4	10.9	4.8	119
1 cup yellow dahl	493	8.5	17.1	0.8	155
1 slice tandoori roti	404	2.7	14.8	2.6	140

Table 8.   Average energy (kJ), fat (g), protein (g), carbohydrate (g) and

sodium (mg) intake for the 3 day analysis period.

Day	Meal	Energy (kJ)	Fat (g)	Protein (g)	Carbohydrate (g)	Sodium (mg)
1	Breakfast	4411	49.9	20.9	126.7	1359
	Lunch	3937	22.8	42.0	132.1	624
	Dinner	3415	16.9	39.2	118.3	773
	Total	*11763*	*89.7*	*102.2*	*377.2*	*2757*
2	Breakfast	4624	53.3	31.6	121.8	1235
	Lunch	4105	23.8	44.7	134.7	464
	Dinner	3651	25.5	34.4	118.2	1005
	Total	*12381*	*102.8*	*110.7*	*374.8*	*2704*
3	Breakfast	3903	41.0	19.3	117.5	1145
	Lunch	4263	22.6	42.2	151.6	654
	Dinner	3428	21.2	44.2	103.1	571
	Total	*11595*	*84.7*	*105.8*	*372*	*2371*
Average	3 Day average	*11913*	*92.5*	*106.2*	*374.6*	*2610*

Note: There was no significant difference ($p < 0.05$) between energy, fat, protein, carbohydrate or sodium values, using one-way ANOVA, between the 3 days of testing.

2.5 Discussion

The initial hypothesis that sodium intake in this population is inadequate to replace estimated sweat losses is supported by the results of this study. During the summer period 55% of workers were found to be clinically hyponatremic with plasma sodium values less than 135mM, compared with only 8% during the winter period. Given that dietary intake is consistent throughout the year, these results indicate that the hyponatremia during summer is most likely due to high sweat sodium losses coupled with inadequate sodium replacement. Nearly 5% of workers were shown to have extremely low plasma sodium levels (below 130mM) posing a serious health risk. At this level it is possible that workers would become symptomatic and be unable to continue work.

Assessment of dietary intake revealed that on average workers consumed between 2000-3000 mg of sodium (5 – 7.5 g salt) per day. This quantity is insufficient, particularly during summer when temperatures are extreme (Exceeding 40ºC on a daily basis) and fluid and electrolyte losses through sweat are assumed to be high due to high sweat rates.

Previous studies conducted in manual labourers in Australia have found average sweat rates of 1.1L/h., equating to over 10L of fluid loss during a work shift (Miller and Bates 2007a; Brake and Bates 2003b). Average sweat sodium losses over a 10 hour work shift in hot conditions have been estimated to be 4.8 – 6 g, equivalent to 10-15g salt (NaCl) (Bates and Miller 2008). Due to the large inter-individual variation in sweat rate and sodium loss these values may be even higher in some individuals, with reported values in excess of 10g of sodium (25g salt) per day (Bates and Miller 2008). The results from this study clearly indicate that salt intake in this population is falling well short of replacing potential sweat losses. Even in a worker with a low sweat rate (500ml/h) with below average sodium losses (20mmol/L), working for a 12-hour work shift could experience over 6L of fluid loss with the loss of nearly 3g of sodium (7g salt). The sodium intake in the workers in the studied workplace would not be sufficient to replace even these losses. In workers at the higher end of fluid and sodium losses, dietary intake is significantly lower than estimated losses, placing workers at risk of developing electrolyte disturbances and heat illness.

There is some evidence to suggest that long term dietary salt restriction may have some potentially adverse health effects (Egan & Stepniakowski, 1997(Cohen, Hailpern, and Alderman 2008). An observational cohort study based on the results from the Third National Health and Nutrition Examination Survey (NHANES 3) found that overzealous restriction of dietary sodium may be associated with increased risk of cardiovascular disease and all-cause mortality (Cohen, Hailpern, and Alderman 2008).

What is also unclear is the effect that this reduction of sodium intake may have on the ability to tolerate prolonged exercise or work in the heat. The effect of the prolonged consumption of a sodium deficient diet in those performing work in the heat has not been thoroughly investigated. Investigations in rats fed a chronic low sodium diet showed a fluid shift towards intracellular compartments resulting in a decreased plasma volume and significantly increased haematocrit (Francesconi, Hubbard, and Mager 1983). It may be argued that this change in hemodynamic state would seriously compromise the body's ability to tolerate further circulatory stress such as that imposed by excessive sweating in the heat.

In a rat study, circulating levels of aldosterone and cortisol have been shown to be significantly increased in sodium depleted mice in order to conserve and maintain circulating sodium levels (Francesconi, Hubbard, and Mager 1983). In salt resistant subjects, salt restriction may lead to activation of the renin-angiotensin system and vasoconstriction which may lead to a rise in blood pressure and potentially adverse metabolic effects in these subjects (Egan, Weder, Petrin, & Hoffman, 1991). In this study a salt resistant subject was an individual who had a mean intraarterial pressure (MAP) during the low NaCl phase greater than or equal to that during the high NaCl phase. A meta-analysis by Graudal et al (1998) found similar results. Subjects with a decreased sodium excretion due to lower intake had 5-6 times higher aldosterone and renin than those with higher excretion. Oliver et al (1975) found similar results in Yanomano Indians who consume habitually a very low sodium diet. The authors found that these people have 3 times higher levels of plasma renin and 10 times higher urinary aldosterone excretion than normal controls. These results indicate that the renin-angiotensin aldosterone system is activated in order to minimise fluctuation

in blood pressure; this may explain why there is a relatively small effect of a decreased sodium intake on blood pressure readings.

There is also some evidence to suggest that chronic salt restriction may result in growth retardation, reproductive problems, as well as reduced muscle mass (Bursey and Watson 1983; Fine, Lestrange, and Levine 1987; Hoorn et al. 2011). While these studies have only been attempted in the rat model, the classic human experimental trial by McCance (McCance 1936) clearly showed that a short term period of salt restriction resulted in nausea, loss of appetite, muscle cramps, fatigue and considerable weight loss. There has also been some recent evidence to suggest an increased risk of fractures in patients with hyponatremia (Hoorn et al. 2011). Hoorn et al (2011) also discuss the effect of hyponatremia on bone architecture. One recent study in a rat model showed that hyponatremia increased the number of osteoclasts in bone while serum osteocalcin was decreased, both changes indicative of osteoporosis (Verbalis 2010). In one study hyponatremia increased the odds of having a fall-related fracture threefold to fourfold (Gankam Kengne et al. 2008). The proposed mechanism responsible for the marked increase in fracture risk is gait instability and attention deficits.

In addition to these effects, a higher sodium diet is known to enhance the thermoregulatory and cardiovascular adaptations that occur during heat acclimatisation (Luetkemeier, Coles, and Askew 1997). A recent study by Miyazaki et al (2010) showed that stable chronic hyponatremia resulted in impaired memory function in rats that was normalised when sodium levels were corrected to within normal range. The effect of chronic hyponatremia on the central nervous system in humans is largely unknown and requires further investigation. A study by Renneboog et al (2006) indicated that patients with mild chronic hyponatremia had an increased incidence of falls due to a global decrease in attention capabilities, posture and gait mechanisms (Renneboog et al. 2006). While the patients in this study were reported to be asymptomatic, the results also identified significant impairment in cognitive function. The mechanisms of these observations are thought to be a result of slowed peripheral and central nerve conduction (Renneboog et al. 2006). For manual workers performing intense physical labour, any impairment in judgment or cognitive capacity will increase vulnerability to workplace accidents and injury and is therefore a major concern.

It is estimated that on a global scale cardiovascular disease alone is killing 17 million people each year and is the single largest risk factor for mortality. High blood pressure is the leading cause of global burden of disease with approximately two-thirds of stroke and one half of ischemic heart disease being attributed to hypertension (Lawes, Vander Hoorn, Law, MacMahon, & Rodgers, 2006). Further to this, a recent study by Gardener et al (2012) showed that a high salt intake was associated with an increased risk of stroke, independent of vascular risk factors. Due to the increasing incidence of cardiovascular disease and high blood pressure on a global scale, dietary salt restriction is being strongly advocated as a public health measure to reduce the incidence of chronic disease worldwide. Currently the World Health Organisation recommends that salt intake should be less than 5g/day (less than 2g/day sodium) (World Health Organisation 2007).

A recent meta-analysis by Graudel et al (2012) investigated the effect of a low vs high sodium diet on blood pressure readings and hormonal responses (renin and aldosterone). A total of 167 studies were included in the meta-analysis. Results indicated that sodium reduction on average resulted in a significant decrease in blood pressure of 1% in normotensives. The authors of this paper concluded that the small size of this effect might be due to the persistent increase in plasma renin and aldosterone that was also found. To date, there has been no randomized clinical trial to determine the effect of a long-term reduction in dietary sodium on cardiovascular health or mortality (Cohen, Hailpern, and Alderman 2008). This has raised considerable debate as to whether a reduction in salt intake on a public health level will be of benefit. A recent Cochane review examining the evidence on the long term effects of reducing salt intake in patients with elevated blood pressure concluded that there is currently insufficient evidence to assess the effects of reduced salt intake on overall health outcomes (Hooper et al. 2009). There is however some evidence to suggest that a reduction in salt intake may help to maintain lower blood pressure in patients who have recently withdrawn from the use of hypertensive medication. The lack of clear evidence is due to the lack of long term randomised controlled trials and the difficulties in controlling salt intake over an extended period of time (Hooper et al. 2009). In addition to this there are a multitude of genetic, ethnic, behavioral and

environmental factors which determine the appropriate level of sodium intake for good health (Alderman 2010). Because of these additional factors it is difficult to indicate a universal level of sodium intake that is appropriate for the population at large. In population groups such as manual labourers or endurance athletes who are sweating profusely for prolonged periods these recommendations are not appropriate.

The results of this study provide some support for the development of sodium intake guidelines specific for these populations. Currently the message to reduce sodium intake, while suitable for the majority of the population, is misleading for these 'at risk' groups. This was highlighted in this study as the medical staff were advising workers to reduce salt intake as a measure to reduce the risk of chronic disease.

In the last five years there have been reports of a number of young labourers presenting to the Intensive Care Unit in hospitals within the Middle East with seriously low plasma sodium levels, placing them at risk of neuromuscular dysfunction. Dr Sadeq Qadri, Chair of the Intensive Care Unit at the Mafraq hospital, in a letter to the Clinical Affairs Director, advised that laborers working in the construction field in a hot environment be advised to consume fluids with added sodium to prevent seriously low plasma sodium levels (Qadri, personal communication, November 2008). This information and the results from this study indicate that there is a need for sodium intake guidelines specific for manual laborers working in the heat to be developed. Providing the workplace with specific guidelines showing estimated sweat sodium losses and practical replacement guidelines for the various working roles will acknowledge that workers in physically demanding outdoor roles will have greater fluid and salt requirements than stationary indoor workers. Interviews with the workers in this study revealed that several individuals had been actively trying to limit salt intake to prevent hypertension. Similar findings have been reported in a study in South African forestry workers where Community Health Workers were advising workers to only consume water and restrict salt intake in an attempt to prevent hypertension (Biggs, Paterson, and Maunder 2011). Given that current health recommendations are aimed at restricting dietary salt intake, educating medical staff and workers on the importance of both fluid and sodium replacement when working in very hot environmental conditions of heat stress is necessary.

The position stand by Sawka et al (2007) supports the need for athletes with high sweat losses to replace sweat electrolyte losses through the addition of salt to meals. The results of this study as well as the results from the study by Biggs et al (2011) support the need for this recommendation to be extended to manual labourers working in extreme environments. In order to increase sodium intake the addition of salt to meals during cooking is indicated. In addition to this, educating workers, particularly those in more physically demanding roles on the benefit of adding table salt to meals is also warranted. When working in severe thermal conditions, replacement of fluid losses with water alone is insufficient, as it does not replace sodium lost in sweat, thus increasing the risk of dilutional hyponatremia and acute water intoxication (Hoorn and Zietse 2008). The provision of an electrolyte replacement fluid that has been designed for prolonged use in an industrial setting is indicated (Nose et al. 1988). The benefit of providing an electrolyte replacement beverage between meals is to promote water uptake and retention, and to offset fatigue by maintaining blood glucose, whilst palatability encourages fluid intake when compared with plain water (Sawka et al. 2007). However, even regular consumption of an electrolyte replacement fluid throughout the work shift would not make up for the dietary inadequacy; the addition of salt to meals is also warranted.

2.6 Summary

The data from this study demonstrate that the diet of some workers in the Middle East has inadequate salt content. High sweat sodium losses due to long periods of manual work in the heat are not being adequately replaced potentially leading to chronic hyponatremia. While workers may be relatively asymptomatic there may be safety implications for workers who are managing heavy machinery or working at height due to an impaired cognitive ability and neuromuscular function and increased susceptibility to fatigue, both of which may increase the risk of workplace accidents. The information collected in this study may apply to hundreds of thousands of workers in the Middle East and other countries where workers are engaged in prolonged manual labour in hot climates while consuming a diet that is predominantly starch based. Increasing the total salt content of both fluid and food consumed by workers may be effective in reducing the incidence of work related illness and accidents in this population.

2.7 Conclusions

- A significant proportion of workers at this site in the UAE were clinically hyponatremic during the summer period. This was not shown during winter indicating that it is primarily due to high sweat sodium losses that are not being replaced.

- The salt content of the diet is insufficient to replace estimated sweat sodium losses during summer.

- Workers are unable to consume food at regular intervals between meals due to logistics of the work environment and will benefit from regular consumption of an electrolyte replacement fluid specifically designed for prolonged industrial use.

- Educating workers and medical staff on the importance of adequate fluid and salt intake to prevent dehydration and electrolyte imbalances is strongly indicated.

- Salt intake guidelines specific for workers engaged in prolonged work in the heat are necessary to decrease the incidence of hyponatremia in this population. Sample guidelines are given in the summary chapter of this thesis.

Heat Chamber Studies

Figure 9. The setup in the environmental chamber where studies 2 and 3 were conducted.

Chapter 3: **Methodology used in Heat Chamber studies**

3.1 Introduction

This chapter will describe and validate the experimental procedures implemented in both of the controlled environmental chamber studies (Chapters 4 and 5). Specific details of the subjects recruited and the experimental protocol will be discussed in detail in subsequent chapters of this thesis.

3.2 Ethical Approval

The Curtin University ethics committee approved both environmental chamber studies reported in this thesis. Testing was carried out in the physiology laboratories at Curtin University. All subjects were informed of the purpose and procedures involved in the study and gave their full written and verbal consent to participate. Subjects were advised that they had the right to withdraw from the study at any time.

3.3 Body height and weight measurement

Nude body weight was measured and recorded immediately after voiding, on an electronic balance scale (Ohaus Corporation, Model CW-11) recorded to the nearest 0.01 kg.

Standing height was measured using a stadiometer fixed to the wall and recorded to the nearest 0.1cm.

3.4 Pre-trial Standardisation

A medical prescreening process was conducted during the recruitment phase of the study either via phone interview or email, in order to ensure that athletes involved in the heat chamber studies were not taking any regular medication.

All subjects strictly followed the following protocol in the 24-hour period prior to testing.

- No alcohol of caffeine to be consumed.
- A maximum of 1 hour of low intensity aerobic exercise.
- 500mL of water or electrolyte replacement fluid was to be consumed before going to bed the night before each testing session.
- On the morning of the testing session all subjects were asked to consume a light breakfast of toast with spread plus consume at least 500mL of water or electrolyte replacement fluid.

3.5 Data Collection

3.5.1 Measurement of Hydration Status

Hydration status was assessed through Urine specific gravity readings (Atago instruments: Hand held urine refractometer) taken prior to subjects entering the environmental chamber. Subjects recording Usg readings >1.015 were asked to consume 500ml of fluid in small aliquots over a 30 minute prior before beginning the testing session.

Dehydration has been shown in numerous studies to reduce the capacity to effectively thermoregulate, while the composition of sweat may also be altered (Senay 1968). Several studies have been performed to assess the validity of Usg as a marker of hydration status. These studies have generally concluded that measurement of hydration status using refractometry is a valid assessment of hydration status. Urine specific gravity measurement has been reported to give a more sensitive indication of hydration status than the use of plasma osmolarity, while also correlating well with urinary osmolarity (Armstrong, Maresh, and Castellani 1994; Armstrong et al. 1998). However, in situations pertaining to dynamic fluid changes such as during post exercise recovery or in acute dehydration, Usg may not be an effective indicator of hydration status as changes in urinary measures may be delayed when compared to changes in plasma osmolarity (Oppliger et al. 2005; Steiner, Nager, and Wang 2007). These large fluid changes are unlikely to occur in the heat chamber studies due to the short duration of testing.

The most recent National Athletic Trainers' Association position statement on fluid replacement (2000) stated that Usg measurement should be used in athletes as a marker of hydration status due to its reliability and feasibility for use in the field

situation. Table 9. outlines the Urine specific gravity values used to determine hydration status.

Table 9.   Indexes of Hydration Status (Casa et al. 2000)

* % Body weight change = [(pre-exercise body weight – post-exercise body weight)/ pre-exercise body weight] x 100

Condition	% Body Weight Change*	Urine Specific Gravity
Well hydrated	+ 1 to -1	<1.010
Minimal dehydration	-1 to -3	1.010 – 1.020
Significant dehydration	-3 to -5	1.021 – 1.030
Clinical dehydration	>5	>1.030

3.5.2 Sweat Collection Method

Sweat collection via the Whole Body Washdown (WBW) technique is considered the criterion method for accurate determination of whole body sweat mineral losses (Baker et al. 2011). This method is considered to be most accurate as all produced sweat is collected and accounted for, while it does not interfere with the normal sweat evaporation process (Baker et al. 2009b). Limitations of this method include the need for testing to be performed in a controlled laboratory setting with cycling being the only means of exercise. There is also the potential for airborne contaminants to enter the sample.

Regional skin surface collection is the most practical method of sweat collection particularly for use in the field setting and has been used frequently in the recent literature. This method involves sampling sweat from various regions of the body and using this information combined with weighing to estimate whole body sweat losses. Baker et al (2009b) investigated the validity of using this method of sweat collection as means of estimating whole body sweat electrolyte losses. Results

showed that all sweat sampling sites (forearm, back, chest, forehead, and thigh) were significantly correlated with the WBW technique for both sodium and potassium concentrations. The regional skin surface collection method did however significantly overestimate both [Na$^+$] and [K$^+$] across all body sites. Results from the thigh and upper arm produced the least variation from WBW results, with the thigh underestimating sodium loss by 7% and the arm overestimating by 20%. The variation in [K$^+$] values was not significantly different. The authors on this paper conclude that regional skin surface sweat collection is a valid method of accurately and reliably predicting sweat [Na$^+$] and [K$^+$] losses. As there was significant [Na+] overestimation reported when using the regional sweat patch method, the authors suggest the use of appropriate regression equations to account for this.

The thigh and upper arm have been chosen as sites for sweat collection in both environmental chamber studies reported in this thesis as they appear to correlate most accurately with the whole body wash down technique, and are the most commonly used sweat collection sites reported in the literature.

3.5.3 Sweat Collection Protocol

Before entering the climate chamber subjects changed into their exercise clothing (shorts and t-shirt) and were fitted with a heart rate monitor (Polar, Model S710i). After the 15-minute warm up period, subjects were required to towel dry and be weighed in minimal clothing (males wore bike shorts and females wore bike shorts and a sports bra) on an electronic balance scale accurate to the nearest 0.01kg. It is recognised that this may introduce some inaccuracy in estimating sweat loss, however the facilities were not suitable for nude weighing. This was minimised by weighing in minimal clothing which would already have been partly saturated with sweat absorbed during the start-up period. Subjects then returned to the climate chamber and were fitted with four sweat collecting devices positioned on the upper arms and upper thighs. The sweat collecting devices were Wescor sweat collection capsules (Hammond, Turcios, and Gibson 1994). The time delay between exercise onset and attachment of devices was to allow sweat onset to be initiated. This avoids any possible concentration changes between "start up sweat" and regular sweat flow. Care was taken to ensure consistent, minimal pressure was applied to the skin. This is to prevent sweat leaking into the collection site. The capsules were positioned on

the lateral aspect of both upper arms and the front of both thighs, approximately midway between the knees and hip. The devices were secured to the limbs after the sites had been shaved and cleaned with alcohol swabs. The high correlation between sodium concentration between the right and left limbs confirms consistency of macroduct placement. It may also suggest that minor variations in placement do not matter as sweat from the whole gross anatomical area is of similar composition.

At the end of the sweat collecting period, sweat collecting devices were removed and placed in individual sealed plastic bags. Subjects were then instructed to shower without wetting their hair, abstain from drinking, eating or urinating and ensure they were completely dry before dressing into the clothes in which they were originally weighed. After re-weighing, the sweat rate (L/hr) was calculated from the weight loss of the subject over time. In calculating the sweating rate, the 'end of sweating' time was taken from when the subject stood under the cool shower, immediately following the exercise session.

3.5.4 Heart Rate Monitoring

Polar heart rate monitors (model S710i) were worn by all subjects while cycling in the chamber. Each subject was given a heart rate goal prior to entering in the environmental chamber. The heart rate receiver was placed on each subject's bike so they were able to monitor their performance. Heart rate was manually recorded at 5 minute intervals throughout the exercise trials. No subject was unable to maintain the desired heart rate level for the entire duration of testing in either study.

Heart rate was used as a guide to estimate relative workload in both of the environmental chamber studies. A percentage of subjects maximal heart rate (%HR max) was used to estimate %$\dot{V}0_2$max in each study according to the target values recommended by the American College of Sports Medicine (American College of Sports Medicine 1991). Swain et al (1994) reported that while this method of estimating relative exercise intensity may have some limitations particularly in untrained individuals, it does provide a relatively accurate measure of workload. In both of the heat chamber studies absolute workloads were not critical and were

simply designed to either produce various sweat rates or ensure a constant workload was maintained across the different trials.

3.5.5 Analysis of Sweat Sodium and Potassium Concentration

The sweat collected was evacuated from the sweat collecting devices with compressed air into small test tubes and immediately frozen. For analysis, test tubes were thawed and sweat was weighed on individual weighing trays. A volume of 0.01 ml was drawn into an automatic pipette by capillary action. Sweat samples were then diluted 1:200 in a volumetric flask (2 ml) for analysis. The sodium and potassium concentration in the samples was determined by an atomic absorption spectrophotometer (Avant E GBC, AA hydride system HG3000).

3.5.6 Calculated Sweat Loss

A change in body weight was calculated from the difference between body weight measurements before and after exercise. The rate of sweat loss (mL) was calculated from the difference in body weight measurement before and after the trials as well as taking into account any fluid volume consumed during cycling. If subjects needed to urinate during the cycling effort, total volume of urine was accounted for by measurement of urine weight.

Chapter 4: Heat Chamber study #1: The effect of exercise intensity on sweat rate and sweat sodium losses in well trained athletes during cycling exercise in the heat

4.1 Introduction

Endurance athletes often train and compete in harsh environmental conditions prompting high sweat losses. During endurance exercise, body metabolism can increase by up to 20 times the resting rate in order to support skeletal muscle contraction (Nadel, Mack, and Takamata 1993). The majority of this energy is released as heat which must be dissipated from the body in order to maintain a stable internal core temperature. The sweating response, whereby water is evaporated from the skin surface, is an important physiological mechanism by which to dissipate this heat to the environment, without which a continual rise in internal body temperature would occur, leading to hyperthermia and severely compromising exercise performance (Hargreaves and Febbraio 1998; Rehrer 2001; Nadel, Mack, and Takamata 1993). Sweat induced dehydration will occur if sufficient and appropriate fluid is not consumed resulting in a substantial decrease in plasma volume.

There is a large body of literature to suggest that a loss of body weight between 2-7% due to dehydration, can significantly reduce exercise performance, particularly when exercise is performed in the heat (i.e. >30 ºC) (Shirreffs 2009). In addition to this, dehydration will increase the physiologic strain associated with exercise and negate the thermoregulatory advantages that are known to result from high aerobic fitness (Coyle 2004). If dehydration is prolonged or severe, the athlete is at risk of more serious medical consequences such as heat stroke, resulting from impaired central nervous system control of thermoregulation.

The sodium loss that accompanies water loss through sweat has had much less attention in the literature. Work in this area has generally been focused on team based sports such as tennis, soccer, football and basketball. Endurance events can often last for in excess of 12-15 hours in hot and humid conditions. Athletes participating in these events therefore represent a group who are at increased risk of fluid and electrolyte disturbances. Guidelines for the adequate replacement of the sodium lost through sweat are limited, often conflicting, and do not differ for athletes training and competing in different events. Lack of specific recommendations for athletes may lead to the inadequate replacement of salt losses in some endurance athletes (Valentine 2007; Montain, Sawka, and Wenger 2001). This is particularly the case for athletes who are training twice daily or for prolonged duration, with large sweat losses. Further research in this unique group is needed to allow for the development of more specific fluid and electrolyte guidelines for athletes competing in these events.

The sweat rate of an athlete can be calculated simply and practically in large athlete groups by measuring weight loss over an exercise session. There are however, practical difficulties in estimating sweat sodium losses, as comprehensive sweat analysis in the laboratory is required for accurate assessment. Several studies in the literature have reported a linear relationship between increases in sweat rate and sweat sodium loss in untrained subjects (Cage and Dobson 1965; Buono, Ball, and Kolkhorst 2007; Buono et al. 2008). This information cannot be directly applied to athletes, due to the sweat gland adaptations that are known to occur with physical training such as sweat gland hypertrophy and an increase in output per gland. Sweat rates have been shown to be higher in athletes resulting in an increase in inter-individual sweat rate variation (Ichinose-Kuwahara et al. 2008).

The aim of this study is to determine whether the linear relationship between sweat rate and sweat sodium concentration previously demonstrated in untrained subjects, is shown in the athlete population. It is hypothesized that there will be an increase in sweat sodium concentration with increases in sweat rate, therefore amplifying sweat sodium losses. With a greater understanding of the effect of sweat rate on sweat sodium loss, more specific sodium replacement guidelines may be developed for athletes competing in endurance events. Athletes with known high sweat losses, who are at an increased risk of heat related illness, need to be identified early so an appropriate fluid and electrolyte replacement strategy can be developed.

The key objectives of this study are:

In a group of well trained endurance athletes:

1. To investigate the effect of increases in exercise intensity on sweat rates and sweat electrolyte (sodium and potassium) concentrations during cycling exercise in a controlled environmental chamber.
2. To investigate the relationship between sweat rate and sweat sodium concentration.
3. To determine the variability in sweat rates and sweat electrolyte concentrations.
4. To estimate daily sodium and potassium losses based on observed sweat rates to aid in the development of specific fluid and electrolyte replacement guidelines for endurance athletes.

4.2 Method

4.2.1 Subjects

Eighteen male well trained male triathletes (Age 37.9 ± 9.7 years, Weight 83.2 ± 13.6kg, $\dot{V}O_2$max 60.8 ± 9.4ml/kg/min) volunteered to be part of the study (Data shown in Table 10. All trials were conducted in the environmental chamber at Curtin University, Perth, Western Australia. The trials were conducted in January and February during the summer period. The climatic conditions at the study location are shown in Table 11. Subjects were recruited from triathlon and cycling clubs around Western Australia through advertising on the club websites and newsletters. Prior to the start of the investigation all subjects gave their voluntary, written informed consent to participate.

Criteria for participation were:

- Male between the ages of 18-50 years.
- Must have been participating in at least 10 hours per week of moderate intensity cycling and/or running in the three months prior to the trial.
- Have no known medical condition or taking any regular medication.

Each subject was also asked to complete a pre-testing questionnaire determining demographic details and current training volume.

This study was approved by the Curtin University ethics committee.

Table 10. Physical characteristics of subjects involved in Study 2 (n=18).

Note: Values are means ± standard deviation

Characteristic	Value
Age (years)	37.9 ± 9.7
$\dot{V}O_2$max ml/kg/min	60.8 ± 9.4
Training (Hours per week)	16.2 ± 4.9
Weight (kg)	83.2 ± 13.6

4.2.2 Climatic Conditions at the Study Location

The study was conducted during all of January and the first two weeks in February. Table 11. below shows the seasonal weather averages for the study location in the six months prior to the commencement of the study.

Table 11. Mean minimum and maximum temperatures (ºC) and relative

humidity (%) for Perth Western Australia in the four months prior to

testing

SOURCE: Western Australian Bureau of Meteorology

Month	Mean minimum temperature (ºC)	Mean maximum temperature (ºC)	Mean relative humidity (%)
September	8.0	22.1	67
October	9.7	25.1	58
November	14.3	30.3	52
December	15.8	30.5	50
January	19.0	33.7	51
February	20.8	34.9	53

4.2.3 Study Design

Subjects were randomised into one of three groups for the experimental trials. Group 1 began with a low intensity trial (60% max HR), Group 2 moderate intensity (80% max HR) and Group 3 with a maximal effort time trial. Groups were then crossed over so all subjects completed one trial at each of the exercise intensities in order to compare intra-individual differences as well as inter-individual differences (Figure 10). Subjects completed one trial per week over three consecutive weeks.

Before entering the climate chamber, all subjects were required to void their bladder and provide a urine sample for specific gravity analysis to verify that they were adequately hydrated (Usg <1.015).

Figure 10. Study design, Study 2
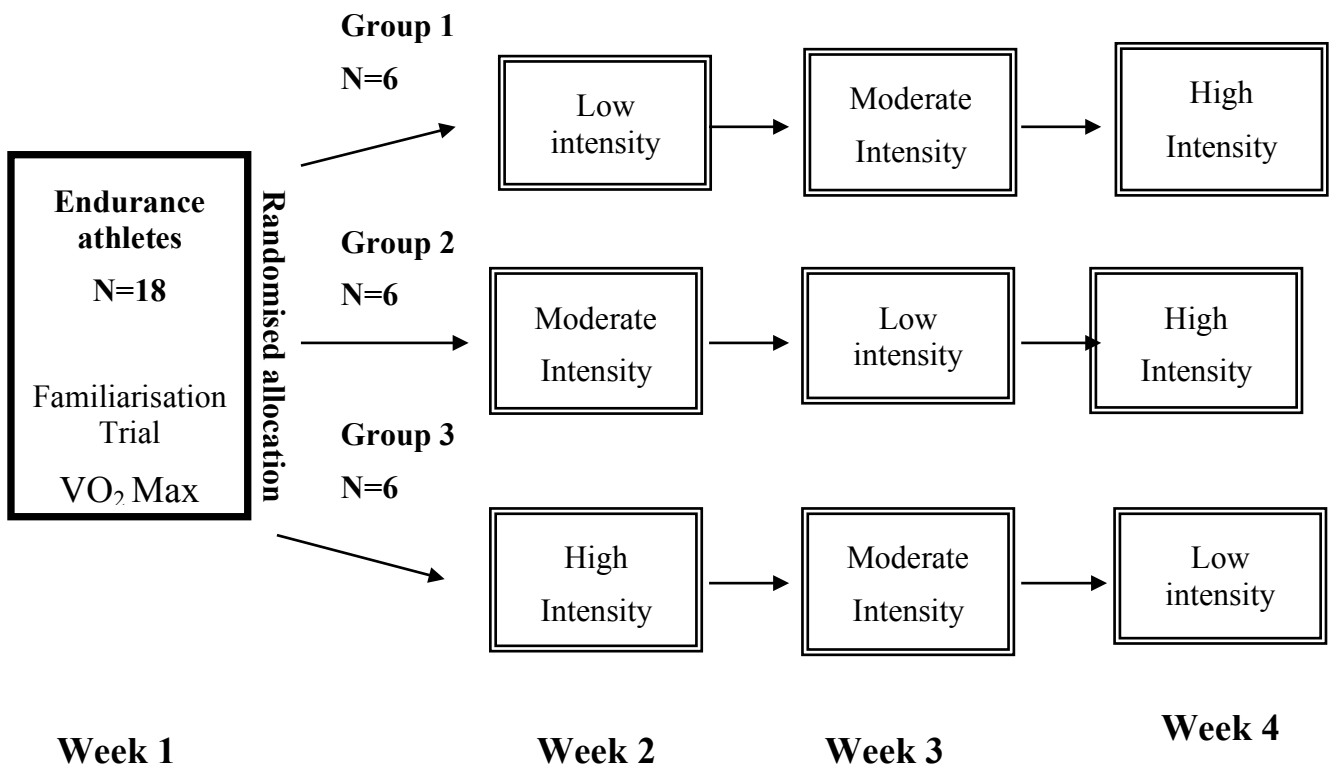
4.2.4 Experimental Protocol

Before the experimental trials all subjects were required to undergo an initial familiarisation trial where $\dot{V}O_2$ max was estimated based on heart rate and work rates using the modified Ấstrand nomogram (Astrand 1960). The aim of the familiarisation trial was to familiarise subjects with the testing equipment and procedures involved. One week following the initial trial subjects performed the first of 3 exercise trials of various intensities. Each of these trials were conducted one week apart. The three trials were performed in a climate chamber set to 35 degrees and 50% relative humidity (RH). These are similar climatic conditions to those encountered in Perth at the height of summer. All trials were conducted in the morning between 0600-0900 hours. The intensity of exercise for each of the trials varied from:

 1) Low intensity (60% max HR)

 2) Moderate intensity (80% max HR)

3) High intensity – maximal intensity time trial.

The trial order varied among subjects to minimise any effect of trial order.

All subjects performed an incremental protocol of exercise using a cycle ergometer in the environmental chamber. All trials consisted of a 15-minute warm up at a workload estimated (from 220 minus age) to be approximately 50% of maximal HR followed by 15 minutes at one of the three intensities. Sweat was collected from the lateral aspects of both upper arms and at the midpoint on both thighs.

The 15-minute sweat collection period was chosen for use in this study as it allows for sufficient time for the sweat collecting coils to be saturated with sweat. There have been few studies investigating the changes that occur in sweat rate and sweat composition over time. The majority of heat chamber studies investigating sweat rate and electrolyte loses have used a short duration protocol of between 15-40 minutes of heat exposure (Meyer et al. 1992; Bates and Miller 2008). A recent long duration study (7h of exercise-heat stress) by Montain et al (2007) aimed to investigate the effect of sustained sweating on sweat mineral composition. The results of this study showed that sweat sodium, potassium and calcium losses during prolonged sweating

can be predicted from initial sweat composition (Montain, Cheuvront, and Lukaski 2007).

4.2.5 Statistical Analysis

Subjects were randomized to one of three exercise intensity groups. Data were summarized as mean (± standard deviation) and tested for normality of distribution. A one-way repeated measures analysis of variance (ANOVA) was then used to detect differences in study outcomes (sweat rate, sweat sodium concentration and sweat potassium concentration) amongst the three exercise intensity levels. Post hoc Bonferroni adjusted t-tests were used for pairwise comparisons. Pearson product-moment correlation was used to assess the relationships between sweat rates and sweat sodium concentration. For all analysis the 0.05 level of significance was used. All statistical analysis was performed using SPSS for Windows (version 18.0, SPSS Inc., Chicago, IL, USA)

4.3 Results

Sweat Rates

Results from the one-way repeated measure ANOVA showed a significant effect of exercise intensity on sweat rate (p<0.001). At the lowest exercise intensity level the mean ± SD sweat rate (L/h) was 1.0 ± 0.3 (95% CI: 0.8, 1.2) compared to 1.48 ± 0.3 (95%CI: 1.3, 1.6) at the moderate intensity level and 1.9 ± 0.3 (95%CI: 1.7, 2.0) at the highest exercise intensity level. This data is presented in Table 12. and Figure 11). There was a large inter-individual variability in sweat rates across the three trials with the coefficient of variation calculated at 27%, 25% and 21% in the low, moderate and high trials, respectively.

Sweat sodium concentration

There was a significant effect of exercise intensity on sweat sodium concentration in both the arm and leg sweat collection sites between the low and high exercise intensity trials (p<0.001). At the lowest exercise intensity the mean ± SD sweat sodium concentration (mmol/L) in the arms and legs respectively was 32.7 ± 15.1 (95% CI: 25.2, 40.1) and 28.5 ± 10.1 (95%CI: 23.6, 33.6) compared to 51.7 ± 27.7

(95%CI: 37.9, 65.5) and 46.8 ± 20.4 (95%CI: 36.6, 56.9) in the arms and legs in the high intensity trial (shown in Figure 13). There was no significant difference in the sodium concentrations between the low and moderate or moderate and high intensity trials.

There was a large individual variation in sweat sodium concentrations across the three trials at varying intensity, with the coefficient of variation ranging from 35%-45%, indicating a high degree of between subject variability.

There was also a significant difference between arm and leg sweat sodium concentration across each of the three trials. Sodium concentration in the arms was consistently higher than the legs, however the difference was statistically significant ($p<0.05$) in the low intensity trial only.

Sweat rate vs Sweat sodium relationship

Within the range of sweat rates studied there was a significant positive linear relationship ($p<0.001$) between sweat rate (L/h) and sweat sodium concentration (mmol/L) with a Pearson's correlation coefficient R =0.479 (shown in Figure 12). Based on the R^2 value of 0.229, approximately 23% of the variability in sweat sodium concentration can be explained by changes in sweat rate.

Sweat potassium concentration

There was no significant difference ($p>0.05$) in sweat potassium concentration from the arms or legs among any of the three exercise intensity trials (Table 12.

Estimated sweat electrolyte (sodium and potassium) losses (mg/h)
The mean ± SD estimated electrolyte losses (mg/h) are displayed in Table 13. There was a significant difference between estimated sodium and potassium losses between the low, moderate and high intensity trials. Mean estimated sodium losses in the high intensity trial were 3 times higher than estimated losses from the low intensity trial.

Mean estimated potassium losses in the high intensity trial were 1.5 times higher than the low intensity trial.

Table 12. Sweating rate, sweat sodium and potassium concentration in each of the exercise trials of varying intensity.

Variable*	Low	Moderate	High
Sweat rate (L/h)^2	$1.0 \pm 0.3^{3, 5}$	1.5 ± 0.3^{4}	1.9 ± 0.4
Sweat [Na$^+$] arms (mmol/L)^1	32.7 ± 15.1^{5}	42.3 ± 18.2	51.7 ± 27.7
Sweat [Na$^+$] legs (mmol/L)^1	28.5 ± 10.1^{5}	39.7 ± 16.7	46.8 ± 20.4
Sweat [Na$^+$] average (mmol/L)^1	30.6 ± 11.4^{5}	41.0 ± 17.1	49.3 ± 22.9
Sweat [K$^+$] arms (mmol/L)	8.4 ± 2.4	7.3 ± 2.0	7.1 ± 2.6
Sweat [K$^+$] legs (mmol/L)	10.3 ± 3.0	9.6 ± 3.8	8.7 ± 3.6
Sweat [K$^+$] average (mmol/L)	9.4 ± 2.1	9.1 ± 2.6	7.9 ± 2.5

*Repeated measures ANOVA analysis completed for sweat rate and sweat electrolyte (sodium and potassium) concentrations for arms and legs and the average of arms and legs for all subjects (n=18).

Values are means ± SD.

Note: [1]Significantly different using repeated measures ANOVA ($p<0.05$); [2]Significantly different using repeated measures ANOVA ($p<0.01$); [3]Significantly different between low and moderate intensity trial using *post hoc* pairwise comparisons ($p<0.01$); [4]Significantly different between moderate and high intensity trial using *post hoc* pairwise comparisons ($p<0.01$); [5]Significantly different between low and high intensity trial using *post hoc* pairwise comparisons ($p<0.01$)

Table 13. Estimated sweat electrolyte losses (mg/h)

		Sodium	Potassium
Estimated sweat losses^ (mg/h)	**Low Intensity**	705.9 ± 333.4^{1}	358.5 ± 94.1^{1}
	Moderate Intensity	1389.2 ± 646.2^{2}	484.9 ± 166.5^{2}
	High Intensity	2196.3 ± 1305.5^{3}	580.6 ± 220.5^{3}

^Estimated mean sweat sodium loss (mg/h) calculated based on the sodium concentration and the sweat rate recorded at each intensity level for each individual.

[1] Low intensity trial is significantly different to the moderate intensity trial [2] Moderate intensity trial is significantly different to the high intensity trial [3] Low intensity trial is significantly different to the high intensity trial

Values are mean ± SD

Figure 11. Mean sweat rate (L/h) in the low (60% maxHR), moderate (80%maxHR) and high intensity (Maximal effort) exercise trials as part of Study 2.

Graph displays mean ± Standard error
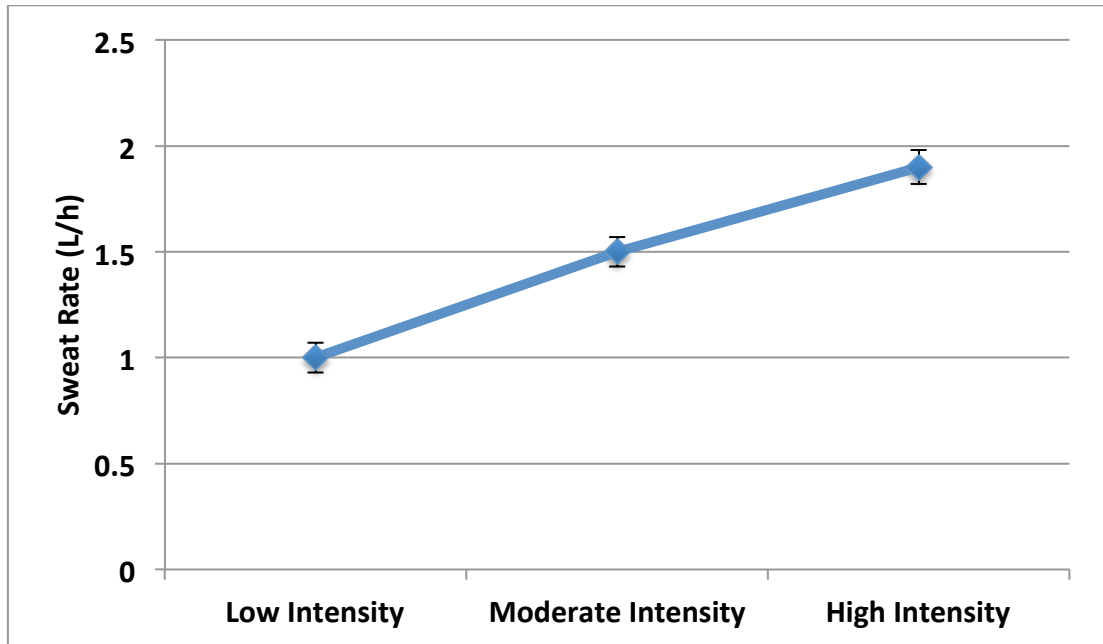
Figure 12. Scatter plot illustrating a statistically significant (p<0.001)

positive linear association between sodium concentration (mmol/L) and

sweat rate (L/h)
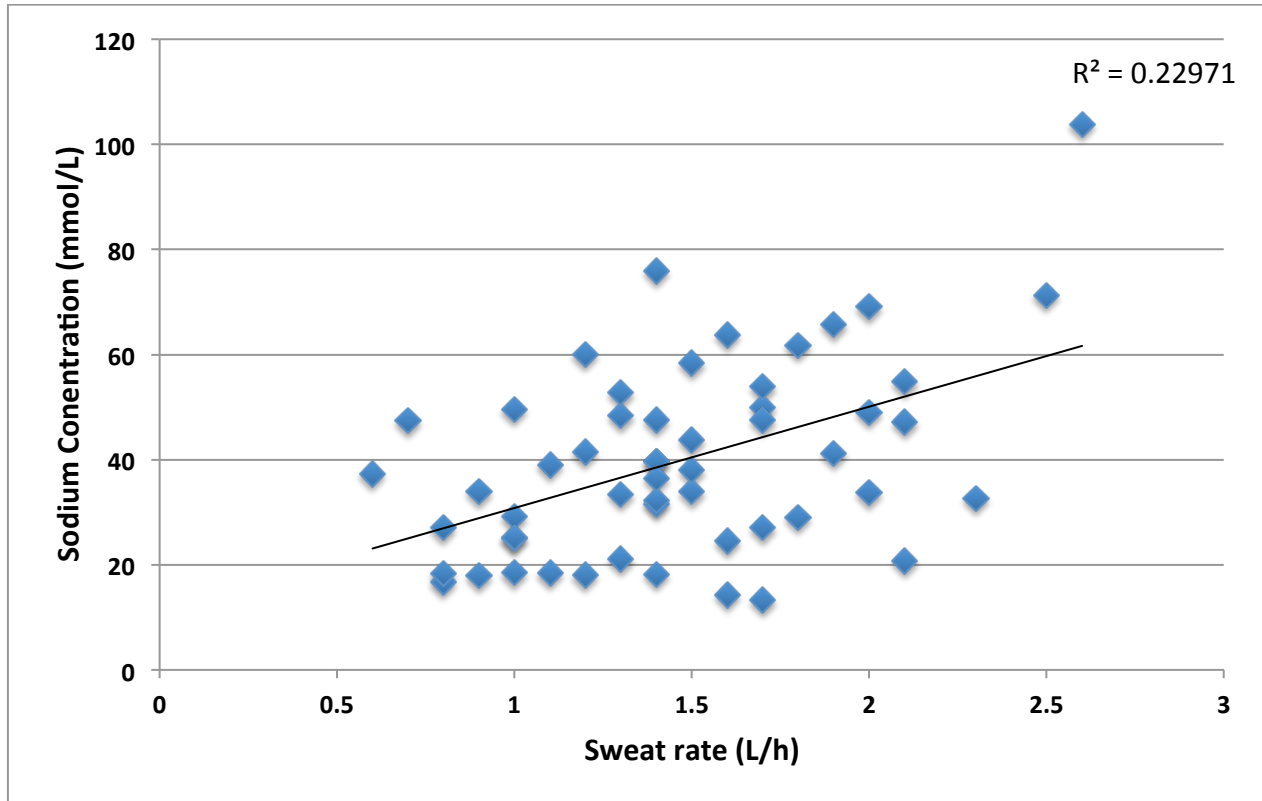
Figure 13. Sweat sodium concentrations (mmol/L) in the low (60%max HR), moderate (80% max HR) and high intensity (Maximal effort) exercise trials.

Graph displays mean ± Standard error
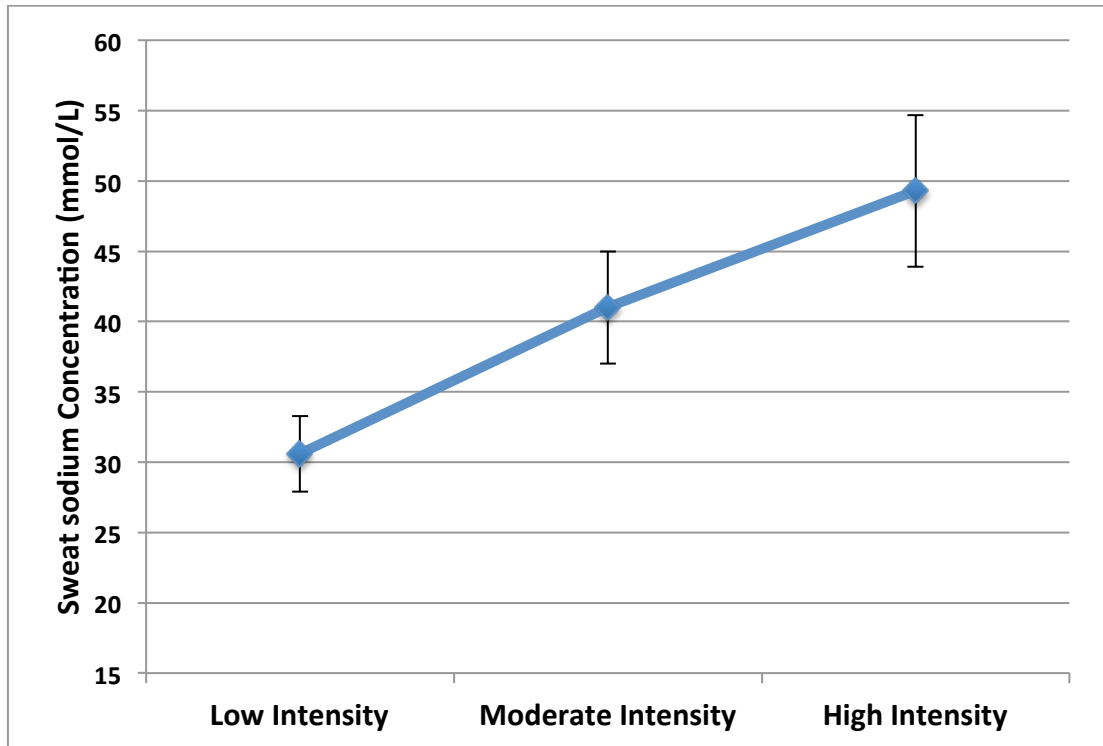
Reliability and Repeatability of the Sweat Collection Method

The sweat collection methodology was tested for reliability and repeatability by comparing sweat sodium and potassium values collected on the left side of the body with those collected on the right.

There was a strong linear relationship between right and left samples in both the arms and legs for both sodium and potassium values. The correlation for right and left sodium samples were consistently stronger than those for potassium with Pearson's correlation coefficient values between 0.83 – 0.95 for sodium, and 0.69-0.85 for potassium (Table 14.

The consistency of the results provides evidence for the reliability and repeatability of the sweat collection methodology.

Table 14. Mean, standard deviation and Pearson product-moment correlation comparing sweat sodium concentration (mmol/L) collected from the arms and legs on the right and left sides of the body of each individual subject.

Intensity		Mean ± SD		Correlation (r value)
		Left	Right	
Low	Arms	32.5±14.5	32.8±13.4	0.94
	Legs	28.9±12.5	26.1±8.9	0.83
Moderate	Arms	40.3±17.0	41.6±19.6	0.95
	Legs	39.9±17.5	38.6±14.7	0.89
High	Arms	52.5±26.4	50.5±28.2	0.92
	Legs	47.3±19.4	46.0±20.7	0.93

Table 15. Mean, standard deviation and Pearson product-moment correlation

comparing sweat potassium concentration (mmol/L) collected from the

arms and legs on both sides of the body of each individual subject.

Intensity		Mean ± SD		Correlation (r value)
		Left	Right	
Low	Arms	8.6±2.4	8.5±2.5	0.85
	Legs	10.2±2.9	10.0±3.6	0.75
Moderate	Arms	7.1±2.4	7.8±1.9	0.69
	Legs	9.4±2.9	9.2±3.2	0.70
High	Arms	6.9±2.6	7.2±2.7	0.81
	Legs	9.1±3.5	8.3±3.2	0.74

4.4 Discussion

This study investigated the effect of increases in exercise intensity on sweat rates and sweat sodium and potassium concentrations. To the best of our knowledge this study is the first to document the sweat rate and sweat sodium relationship in an athlete group with average sweat rates in excess of 1L/h.

The major findings from this study are:

1) A statistically significant (p<0.001) fair degree (R = 0.479), positive linear inter-individual relationship between sweat rate and sweat sodium concentration (mmol/L) was shown.

2) A statistically significant intra-individual increase in sweat sodium concentration (mmol/L) was shown with increases in exercise intensity from the low intensity trial to the maximal effort trial.

3) There was a significant inter-individual variation in both sweat rates (L/h) and sweat sodium and potassium concentrations (mmol/L) at the same relative exercise intensity in the 18 subjects tested.

Relationship between Sweat Rate and Sweat Sodium Concentration

The statistically significant increase in sweat sodium concentration with increasing sweat rates shown in this study supports previous work conducted in untrained subjects (Buono, Ball, and Kolkhorst 2007; Buono et al. 2008; Inoue et al. 1998). Both of these studies, however, have demonstrated a much stronger relationship (R=0.73) than was displayed in the results of the present study (R=0.479). This may be explained by the large inter–individual variation in both sweat rate and sweat sodium concentrations in the 18 athletes tested. Sweat rates ranged from 0.6L/h to 2.6L/h and sweat sodium concentration from 13mmol/L to 103mmol/L, much greater than those previously reported in untrained subjects. These large variations are similar to what has previously been reported by Maughan et al (2005) and Sherriffs et al (2005) in male professional football players.

Several factors may account for the wide variation in both fluid and electrolyte losses including, the state of heat acclimatisation, habitual salt intake and aerobic fitness level (Kirby and Convertino 1986; Allsopp et al. 1998). Physical training has been shown to result in significant increases in the rate of sweat loss due to increases in

both the size and density of sweat glands as well as an increase in the output per gland (Buono and Sjoholm 1988; Ichinose-Kuwahara et al. 2008; Ichinose-Kuwahara et al. 2010). An individual's resting aldosterone concentration, influenced by habitual salt intake also influences sweat sodium concentration (Yoshida et al. 2006). At low levels of sodium intake there is increased aldosterone synthase activity (Williams and Williams 2003). Aldosterone is postulated to be the principal regulator of ductal sodium reabsorption, therefore basal levels are potentially increased in athletes consuming a low sodium diet as a mechanism to conserve plasma sodium (Allsopp et al. 1998; Hargreaves et al. 1989). The only data available to suggest that sweat sodium concentration is reduced in athletes was published in a textbook table which is not cited (Wilmore and Costill 2004). However, a recent study by Hamouti et al (2011) questions this information as exercise intensity level was not given. This is important information as exercise intensity determines sweat rate which in turn affects sweat sodium concentration (Buono et al. 2008). Hamouti et al (2011) investigated sweat sodium concentrations during exercise in the heat in a group of aerobically trained compared to untrained subjects. Results showed that when normalized for sweat rate, high aerobic fitness does not reduce sweat sodium secretion or enhance Na^+ reabsorption.

The physiological mechanism responsible for the increase in sodium concentration with increases in sweat rate is currently unknown. However, there are several possible explanations for these findings. The human eccrine sweat gland consists of two distinct regions involved in both the production and reabsorption of sodium and chloride ions. The secretory coil produces an isoosmotic precursor sweat, while the sweat duct actively reabsorbs sodium from the precursor sweat throughout the passage of sweat to the skin (Shibasaki, Wilson, and Crandall 2006). This reabsorption depends on the active transport of sodium into the interstitial fluid via Na^+ - K^+ ATPase which is localized on the basolateral membrane of the ductal cells. At low levels of sweat production there is ample time for active sodium reabsorption to occur. At higher levels of sweat loss there is insufficient time for complete reabsorption (Taylor 1986). This mechanism is supported by the results of the present study. At higher rates of sweat loss there was a significantly greater concentration of sodium in sweat. The in vivo study by Buono et al (2008) provides further support for this mechanism. This study demonstrated that with higher sweat

rates, the rate of sodium secreted from the secretory coil is greater, while the proportion reabsorbed is decreased, leading to an overall increase in the sodium concentration of the sweat on the skin surface.

Several previous investigations have hypothesized that Na^+ reabsorption would become saturated at high sweat rates, therefore resulting in elevated sweat Na^+ (Shamsuddin et al. 2005; Buono, Ball, and Kolkhorst 2007). The data presented in this study does not support this mechanism as there was a proportional increase in sweat sodium concentration from the low to moderate intensity trial (25% increase) and the moderate to high intensity trial (21% increase). Average sweat rates ranged from 1.0L/h up to nearly 2L/h without a plateau effect being demonstrated. In order to confirm whether a saturation point does in fact exist at higher levels of sweat loss, a study investigating the sodium secretion rates and reabsorption rates in athletes with high sweat rates would need to be performed.

Results of the present study showed on average a 60% increase in sweat sodium concentration (mmol/L) from the low to the high intensity trial. This increase was demonstrated in 14 of the 18 athletes tested. The 4 athletes who did not exhibit this relationship had higher sweat rates (1.7-2.2L/h), lower sweat sodium concentrations (13-33mmol/L) and higher sweat potassium concentrations (10-14mmol/L) than the mean data. It is possible that these 4 athletes had superior thermoregulatory adaptations potentially as a result of acclimatisation to the heat. Heat acclimatisation is known to result in an increased volume of dilute sweat believed to be due to hormonal adaptations involving plasma antidiuretic hormone (ADH) and aldosterone. (Collins and Weiner 1968; Kanikowska et al. 2010). Aldosterone targets the epithelia of the kidney, colon and sweat gland. At the level of the kidney, aldosterone acts on the cortical portion of the renal collecting ducts to increase sodium absorption and potassium excretion. The role of aldosterone at the level of the sweat gland is not clear. However it has been suggested that different isoforms of the mineral corticoid membrane receptor for aldosterone may be present in the kidney and the sweat gland, further work is required to elucidate the aldosterone effect on the sweat gland (Lee, Miller, and Buono 2010).

Another potential explanation for these findings is that these 4 athletes were in negative sodium balance. Allsopp et al (1998) demonstrated that a chronic low

sodium diet potentiates the aldosterone effect associated with heat exposure, further increasing sodium reabsorption. A chronic low sodium diet and acclimatisation to the heat will potentiate aldosterone secretion further increasing sodium reabsorption.

Regional variation in sweat electrolyte concentrations

From a physiological perspective another interesting finding from this study was that there was repeatedly shown to be a lower sweat sodium concentration in the legs compared with the arms. This was statistically significant in the low intensity trial only, however there was a strong trend across all intensity levels. This finding has been reported in previous work by Bates et al (2008). The mechanisms believed responsible for this finding is not entirely understood however, could be explained by the difference in metabolic heat production in the arms and legs during cycling exercise. This relates again to the effect of training on sweat gland adaption. The athletes involved in this study were triathletes with the majority of their training consisting of cycling and running. It is therefore possible that the sweat glands in the legs have adapted to repeated training stress to a greater extent that the relatively inactive arms, resulting in a greater capacity to reabsorb sodium. In this study the volume of sweat from the arms and legs was not collected and weighed. We cannot therefore comment on any sweat rate differences between these two regions. A study by Miyagama (1988) reported that the sweat rate on the thigh was significantly higher than on the arms in athletes who trained the lower limbs repetitively. This is a potential area for future research.

To date there has been no study designed to investigate whether sweat rate and composition varies in athletes who predominantly exercise the upper limbs regularly. Most sweat composition studies in the literature have used cycling as the predominant form of exercise. It would be interesting to determine whether predominantly using the arm muscles would alter sweat composition. This would give further indication as to the mechanism behind the regional sweat rate and composition variation.

Estimation of sodium and potassium losses in sweat

The extent of sweat electrolyte losses is dependent on sweat electrolyte concentration in combination with the volume of sweat lost. Magnitude of sweat sodium losses can vary significantly between individuals due to large variation in these two variables.

Despite this, in the present study there was on average a 70% increase in total sodium loss (mg/h) from the low to the high intensity trial. There was however significant individual variation in estimated sodium losses ranging from 600mg/h up to over 6000mg/h in one athlete in the high intensity trial. This information has practical implications for fluid and sodium intake in athletes. The current recommendations for sodium replacement state the need for adequate replacement, however, do not quantify potential losses in a range of athletes or discuss appropriate methods for replacement. Fluid and sodium losses are obviously higher when performing moderate-high intensity exercise, however, if long duration (greater than 4 hours) exercise is performed at low intensity, losses may be equally high or potentially greater.

The large coefficient of variation observed in this study demonstrates the wide variability that exists between individuals in relation to both sweat rate and sodium concentration. This further highlights the difficulties associated with developing blanket guidelines for fluid and electrolyte intake for all athletes and further supports the need for advice to be specific to the needs of the individual. Although the present study employed a short, 15 minute sweat collection protocol, the results from the long duration study by Montain et al (2007) showed that it is possible to make predictions about the sodium and potassium losses over time from initial sweat electrolyte concentrations. Therefore we are able to predict that over a 4 hour exercise session, an athlete sweating at 0.9L/h with a sodium concentration of 13mmol/L will lose approximately 1.2g of sodium; this quantity will easily be replaced in a normal diet. At the other end of the spectrum, a high salt sweater may lose considerably more than this. For example, in the low intensity trial in this study, completed at a workload that is possible to maintain for a 4 hour period, one subject had a sweat rate of 1.5L/h with a sodium concentration of 33mmol/l. This would equate to a loss of approximately 4.5g sodium and would require an active sodium replacement strategy in order to prevent a severe deficit in total body sodium, particularly if training was repeated daily.

Adequate fluid and sodium replacement is vital to ensure complete rehydration post exercise, this is particularly important in athletes incurring large sweat losses during exercise who will soon return to training or competition. Despite this information, several recent studies in professional level players have shown that athletes regularly

return to training or competition in a state of hypohydration, indicating inadequate post exercise rehydration strategies (Hamouti et al. 2010; Volpe, Poule, and Bland 2009). Ingesting water alone is not sufficient to restore lost sweat volume due to the rapid fall in plasma osmolality leading to an decrease in ADH production, consequently increasing urine output (Shirreffs and Maughan 1998). The addition of sodium to an electrolyte replacement drink restores extracellular fluid volume while maintaining ADH levels, therefore preventing diuresis (Rehrer 2001). Sodium has also been shown to increase thirst and the taste appeal of a drink, therefore significantly increasing voluntary rehydration (Clapp et al. 2000).

The current American College of Sports Medicine (2007) recommendations state the importance of replacing the sodium lost in sweat to ensure euhydration. However, unlike the recommendations for fluid replacement, which are based on body weight, environmental conditions and running speed of an athlete, the sodium guidelines are not specific to the needs of the individual athlete. It is difficult to provide specific sodium intake recommendations because of the large variation in sodium losses due to factors such as genetics, dietary intake, physical fitness and heat acclimatisation state (Sharp 2006). A comprehensive laboratory assessment of electrolyte losses is the ideal scenario in developing a fluid and electrolyte regime, this is obviously not practical for all athletes (Valentine 2007). Therefore it may be useful to advise athletes on ways to estimate their own sweat electrolyte losses and therefore formulate replacement strategies. The results of this study indicate that in an athlete group, predicting sweat sodium concentrations based on the sweat rate of an athlete may be appropriate. More research in a larger athlete group is required to confirm these results.

It has been suggested that athletes may be able to self-assess their sweat composition by subjectively identifying the salty taste of sweat, through the observation of salty stains on clothing or from eye irritation when sweat drips into their eyes (Maughan and Shirreffs 2010; Maughan and Shirreffs 2008). To date no studies have been done to assess the validity of these measures. Combining these indicators with sweat rate data and other subjective information such as the prior history of cramping may be practical for use in a field situation. The development of a multifaceted questionnaire to self-assess sodium losses may be a direction for future research. This may be useful to subjectively identify subjects with high sweat sodium losses and if this tool

is determined to be effective, could potentially be used by coaches, athletes and others who are working in thermally stressful environments.

From a practical viewpoint it is appropriate to recommend increased dietary sodium intake in athletes training or competing in the heat on a daily basis. Given that the public health message is to reduce sodium intake it is important that athletes are aware of their increased needs. In order to meet the high sodium requirements of athletes with high sweat sodium losses, adding salt to meals, consuming an electrolyte replacement drink and consuming high salt snacks is warranted. This information may also be pertinent to other groups such as those in military training or manual labour where endurance exercise or work is undertaken in harsh environmental conditions for prolonged periods.

Limitations of the study

Although this research was carefully prepared there are some limitations that should be addressed. Eighteen subjects were recruited for this study; this number could have been increased to improve the power of the study. It was however difficult to recruit additional subjects who met the selection criteria particularly criteria relating to training volume completed in the three months prior to the study commencement. It is important to note that a small sample size reduces the chance of detecting a true effect as well as potentially overestimating the effect size.

As discussed in the chapter 3 (Methodology used in the Heat Chamber studies) the Whole Body Washdown (WBW) technique is considered to be the criterion method for accurate determination of whole body sweat losses (Baker, Stofan et al, 2011). The laboratory facilities used in this study did not allow for this collection method to be used. Regional skin surface collection, the method used in this study, while often overestimating both $[Na^+]$ and $[K^+]$, has been reported to be a valid method of sweat collection (Baker et al. 2009b).

A further limitation of the present study was that regional sweat rates were not assessed. This is something that has not previously been done in our laboratory, as the macroduct sweat collection method is not suitable for estimation of regional sweat rates as it is not possible to guarantee that all of the sweat secreted by the area under the macroduct, is collected by the collection coil. Other methods for determining regional sweat rates would involve determination of the surface area of

each region or via a washdown technique, both of these methods were not practicable within the scope of this project. This is however a potential area for future research, as it would provide further information on the changes in sweat rate and composition with changes in exercise intensity across various body sites.

As has been discussed, the long duration study by Montain et al (2007) reported that sweat sodium and potassium concentration over a long duration training session could be predicted from initial sweat electrolyte concentrations. We would however have been able to predict sweat electrolyte losses over a long duration exercise session with more accuracy if the duration of sweat testing had been longer.

The estimation of $\dot{V}0_2$max using the Astrand and Rodahl protocol could also be seen as a limitation of the present study. Using a direct method of $\dot{V}0_2$max assessment would have enabled us to modify workload more accurately. The estimation of $\dot{V}0_2$max was deemed appropriate for this study as the absolute workloads were not critical and were designed to produce a range of sweat rates (ranging from low to very high).

While every effort was made to ensure that the sweat collecting devices were placed in the same position on each subject, there was potentially some minor variation, as placement site was not marked up prior to study commencement.

4.5 Conclusions

1. There is a fair positive linear relationship between sweat rate and sweat sodium concentration in athletes. It is therefore appropriate to derive sodium intake requirements from sweat rate if more accurate methods of assessing sweat sodium are not practical.

2. With increases in exercise intensity there is an increase in both sweat rate and sweat sodium concentration in athletes. Fluid and electrolyte requirements may therefore vary depending on the type and intensity of exercise.

3. There is considerable variation in sweat rate and sweat sodium loss in athletes performing exercise of similar intensity and duration. This emphasizes the importance of an individual approach to fluid and electrolyte replacement.

4. Some athletes have the potential to lose large amounts of sweat sodium in training on a daily basis. The current guidelines for sodium intake for the general population (2300mg/day) may not sufficient to replace these losses. Recommendations for replacement in athletes include consuming an electrolyte replacement drink combined with choosing high salt snacks and adding additional salt to meals.

Chapter 5: **Heat chamber study # 2: The effects of physical training and seasonal acclimatisation on fluid and electrolyte losses during cycling exercise in the heat**

5.1 Introduction

Heat acclimation refers to the physiological adaptation of the human body to heat (Rowlinson et al. 2013). It has been well recognised that when a person becomes heat acclimatised by exposure to increased temperatures for several days, there are a number of physiological adaptations that take place enabling them to thermoregulate more efficiently (Wendt, Van Loon, and Lichtenbelt 2007). When working or exercising in thermally stressful environments these adaptations serve to reduce physiological strain and maintain thermal comfort while increasing tolerance during work in the heat (Cheung and McLellan 1998). Adaptations involve both the cardiovascular and thermoregulatory systems where heart rate, core body and skin temperature decrease while blood volume increases (Cheung and McLellan 1998; Buono et al. 2011). Expansion of plasma volume occurs very rapidly with some authors reporting major changes in plasma dynamics within the first 4-6 days of heat exposure (Senay and Kok 1976; Cheung and McLellan 1998).

Heat acclimatisation also produces several adaptations in sweating response that serve to increase the rate of heat dissipation via sweat evaporation. Several studies in untrained individuals have shown that following a period of heat acclimatisation there is a significant increase in the secretory capacity of the eccrine sweat gland as well as morphological changes to the gland itself (Sato et al. 1990; Peter and Wyndham 1966). A reduction in the core temperature threshold at which sweating is initiated, an increase in sweating rate as well as a reduction in the sodium concentration of secreted sweat, has

been observed (Bates and Miller 2008; Taylor 1986; Kirby and Convertino 1986). The physiological mechanism responsible for these changes are not entirely understood, however, the increased sweat rate is potentially due to sweat gland hypertrophy (Sato and Sato 1983) or improved efficiency (Collins, Crockford, and Weiner 1966; Lee et al. 2010).

The electrolyte content of secreted sweat has also been shown to alter after a period of heat acclimatisation. Bates and Miller (2008) investigated the effect of seasonal heat acclimatisation on sweat sodium concentration in 29 untrained subjects. Results showed a significant decrease in sodium concentration (Winter: 63.8mol/L; Summer: 44.7mmol/L) at the end of the summer period. Therefore, one adaptation resulting from heat acclimatisation is thought to be the production of a more dilute sweat (Buono, Ball, and Kolkhorst 2007). This sodium conservation mechanism is believed to be due to the increased reabsorptive capacity of the sweat duct post acclimatisation, possibly via the action of aldosterone(Nielsen et al. 1993; Francesconi, Sawka, and Pandolf 1983). Some authors have reported that the increase in aldosterone concentration is only apparent in those subjects with salt deficiency therefore augmenting the aldosterone response (McCance 1936; Smiles and Robinson 1971). It has been since demonstrated, however, that a salt deficiency is not necessary (Davies et al. 1981). There is also some evidence to suggest that the sweat glands may be more sensitive to aldosterone when a person is heat acclimatised (Kirby and Convertino 1986). Aldosterone concentration has also been shown in two separate studies to be elevated at the end of the summer period. This is believed to be a compensatory mechanism in order to minimise sodium and water loss and therefore prevent electrolyte disturbance and dehydration (Kanikowska et al. 2010; Bain and Jay 2011).

Aldosterone is a steroid hormone that acts directly on the epithelia of the kidney, colon and sweat gland in order to regulate sodium reabsorption and potassium secretion. Few studies have investigated the effect of aldosterone at the level of the sweat gland, therefore the role that aldosterone plays in sweat sodium conservation and potassium excretion is relatively unknown. The aim of part A of this study is to investigate the effect of seasonal heat acclimatisation on sweat sodium and potassium loss in a group of well-trained endurance athletes. Based on the proposed mechanisms relating to aldosterone action it is postulated that at the end of summer when subjects are heat acclimatised, aldosterone concentration will be at its maximum, leading to a decrease in

sweat sodium concentration and increase in potassium concentration. With a better understanding of fluid and electrolyte loss in sweat, accurate advice regarding fluid replacement can be provided to athletes training in the heat.

The current fluid and electrolyte replacement guidelines for athletes do not vary depending on an athlete's acclimatisation state. In addition to this, Maughan and Shirreffs(2010) have recently reported that many athletes are incorrectly informed that their need for fluid consumption decreases as they become accustomed to the heat. The studies that have been performed in untrained subjects in this area have clearly identified the need to increase fluid requirements when heat acclimatised due to the enhanced sweating response. It is also known that if an athlete becomes dehydrated, the improved ability to tolerate heat developed through the heat acclimatisation process will disappear completely (Sawka and Pandolf 1990). Educating athletes on the importance of adequate fluid intake is vital in preventing dehydration and maintaining performance.

The adequate replacement of sweat potassium loss in athletes has received very little focus in the recent literature. The issue as to whether potassium supplementation is necessary in heavy sweaters training in hot weather needs consideration particularly given the potential link between deficiency and skeletal muscle and cardiovascular performance (Knochel, Foley, and Walker 1970). To our knowledge, no previous study has investigated the effect of seasonal heat acclimatisation on sweat potassium loss in endurance trained athletes. The documentation of potassium losses in athletes at different time points within the year will enable more accurate replacement guidelines to be developed.

Highly trained endurance athletes have been shown to display similar thermoregulatory adaptations to those of heat acclimatised individuals (Gisolfi and Robinson 1969). There is some controversy in the literature as to whether physical training or fitness are adequate substitutes for heat exposure. The mechanism response for this is believed to be related to the effect of physical training on sweat gland sensitivity and a decrease in sweating temperature threshold (Roberts et al. 1977). It has also been shown that individuals with low to moderate aerobic fitness may experience a greater benefit from a period of heat acclimatisation than those individuals with a high level of aerobic fitness (Caderette et al. 1984). Therefore, athletes who are highly trained may have less adaptive potential than their untrained or moderately trained counterparts (Garrett et al. 2011).

Few studies have examined differences in sweat composition between trained and untrained individuals. Major limitations of the few studies that have been conducted in this area are; minimal differences between the $\dot{V}O_2$max of the trained and untrained subjects selected, and small sample sizes. Additionally, all previous investigations have used a heat acclimation protocol over 1-2 weeks to induce heat acclimation. To the best of our knowledge no previous study has investigated the separate and combined effects of aerobic fitness and seasonal heat acclimatisation on sweat rate and electrolyte losses.

This study was designed to investigate the following questions:

1) What are the effects of seasonal heat acclimatisation on sweat rates, sweat sodium and potassium loss in endurance trained athletes?

2) Should fluid and electrolyte guidelines for athletes consider the heat acclimatisation status of an athlete?

3) Do subjects with a low level of aerobic fitness demonstrate a greater magnitude of improvement (sweat gland adaptation) with heat acclimatisation compared to subjects with high aerobic fitness?

5.2 Methods

5.2.1 Subjects

Two separate groups were randomly selected for this study. Group one consisted of 26 (15 male, 11 female) well trained endurance triathletes. Group two was made up of 22 (9 male, 13 female) untrained subjects. All trials were conducted in the environmental chamber at Curtin University, Western Australia. Trial one in both groups was conducted at the end of winter (August/September) and trial two at the end of summer (February/March).

Criteria for participation were:

Group One (Endurance athletes):

- Age range between 18-50 years
- Completing at least 10 hours of moderate - high intensity exercise per week
- No expected change in training volume over the 9 months duration of the study
- Have no known medical condition or taking any regular medication

Group Two (Untrained subjects):

- Age range between 18-50 years
- Completing 0 - 5 hours of low - moderate intensity exercise per week
- No expected change in training volume over the 9 months duration of the study
- Have no known medical condition or taking any regular medication

The athletes in Group 1 were recruited from numerous triathlon clubs around Perth. Recruitment was done through advertising on a local triathlon online forum as well as through various coaches around Perth. Subjects in Group two were recruited through a volunteer program run through the Nutrition department at Curtin University. Prior to the start of the investigation all subjects gave their voluntary, written, informed consent to participate.

Each subject was also asked to complete a pre-testing questionnaire determining demographic details of the subjects.

Prior to initiating this investigation, approval was obtained from the Curtin University Ethics Committee.

5.2.2 Experimental Protocol

The 48 subjects (26 trained and 22 untrained) underwent 2 separate trials in the environmental chamber at Curtin University. Trial one was conducted at the end of summer (February/March), trial two was conducted at the end of winter (September/October). Each subject participated in 2 testing sessions on separate days (separated by one week) during both trial periods. Testing session one was a familiarization trial where the testing protocol was explained to the subjects. $\dot{V}O_2$max was also estimated during this session from heart rate and work rate using the modified Åstrand nomogram (Astrand 1960). Testing session two consisted of approximately 40 minutes of moderate intensity cycling at 70% of estimated maximal heart rate in a hot and humid environment (35ºC, 60% relative humidity (RH)) prompting extensive sweat losses. The temperature and humidity of the environmental chamber was continually monitored in order to minimise fluctuations. Athletes were tested at the same time of day for both summer and winter testing. All trials were conducted in the morning between 6am and 11am.

On arrival at the laboratory for testing session two subjects voided their bladders and provided urine samples for analysis. The samples were used immediately to assess the subject's pre-exercise hydration status using urine specific gravity monitoring. Atago hand held clinical refractometers were used (Atago instruments: Hand held urine refractometer). Each subject was weighed using scales (Ohaus Corporation, Model CW-11) accurate to the nearest 0.01kg. Subjects were instructed to wear lightweight shorts and singlets for the weighing. Subjects then re-dressed in cycling shorts and attached a Polar heart rate monitor (model S710i) transmitter belt to the chest before entering the environmental chamber. For each subject a work rate was set to elicit a HR of 70% of the estimated maximal heart rate (220-age). Heart rate was recorded at 5-minute intervals throughout the testing session to ensure that the required work rate was being maintained.

Once in the chamber subjects were reminded of the testing protocol. Subjects then mounted their bikes (Monark cycle ergometer) for the 40-minute session. The seat height of the bikes was individually set in order to produce maximum pedaling efficiency. After the initial 20 minutes of cycling, athletes were asked to stop cycling for two minutes while sweat collecting devices were placed on the arms and legs of all subjects. These were positioned bilaterally on the lateral aspects of the upper arm and on the anterior aspects of the thigh approximately midway down the femur. The collecting devices (Wescor) were secured to the limbs after the areas had been shaved and sterilized with alcohol swabs. Once the devices had been securely attached subjects continued to cycle for a further 20 minutes or until each of the four collecting devices had been filled with sweat. The devices were continually monitored during cycling to ensure that the tension on the straps had not altered and that sweat was being successfully collected in the ducts.

On completion of each exercise session the sweat collecting devices were removed, placed in individually sealed labeled plastic bags. Subjects were then instructed to immediately shower without wetting their hair, towel dry thoroughly and refrain from drinking or urinating. Each subject was then individually weighed in the same clothing as the pre-exercise body weight measurement. Sweat rate (mL/min) was calculated from the weight loss of the subjects over time.

The same protocol described above was conducted in the winter months to investigate differences between fluid and electrolyte losses over the two seasons as well as to investigate the effect of training on these losses.

At the end of the summer period all subjects were assumed to be heat acclimatised as they had been exposed to an Australian summer. The degree of of acclimatisation would depend on the amount of time the subject habitually spent outdoors, this was not controlled for in this study. Similarly at the end of winter all subjects were assumed to be unacclimatised. No subjects left Perth for any substantial length of time during the study period.

5.2.3 Statistical Analysis

Two tailed paired samples t-tests were used to examine the difference in sweat rate and electrolyte losses between summer and winter periods for both trained and untrained groups, respectively. Independent samples t tests were used to detect the difference in sweat rate and electrolyte losses between trained and untrained groups for summer and winter periods respectively. Mixed design repeated measures analysis of variance was used to compare the difference in body weight, $\dot{V}O_2$max, sweat rate and electrolyte losses between summer and winter periods; between trained and untrained subject groups; and to detect possible interaction between seasons and training status. Assumption of normality and homogeneity variances were assessed and if the assumptions had been violated, natural logarithm transformation was used. Box' test and Mauchly's test were used to assess the assumption of equality of covariance matrices and the assumption of sphericity, respectively. When a significant F-ratio was obtained, a Bonferroni post-hoc analysis was performed. For all analysis the 0.05 level of significance was used. All data are expressed at mean ± Standard deviation. All statistical analysis was done using SPSS for Windows (SPSS Inc., Chicago, IL).

5.3 Results

<u>Physical Characteristics</u>

Twenty-eight (15 male, 13 female) well-trained endurance athletes were initially recruited for the study, however due to injury and illness 2 female subjects were unable to take part in the study. Twenty six subjects (15 male, 11 female) completed all of the experimental trials. Twenty three (14 female, 9 male) untrained (less than 5 hours per week of exercise) subjects were also recruited for the study. One of these female subjects was unavailable for the winter testing due to illness; this subject's results were excluded from the data analysis.

The physical characteristics of the subjects are presented in Table 16. There was no statistically significant difference in subject's weight or $\dot{V}O_2$max between the summer and winter. There was a significant difference ($p<0.05$) found in $\dot{V}O_2$max values and pre trial weights between the trained and untrained subject groups for both the summer and winter periods with untrained group having lower $\dot{V}O_2$max values on average for both seasons. There was also a significant difference ($p<0.05$) between the pre trial weight of the male and female subjects in both the trained and untrained subject groups. There was however no statistically significant difference between age or estimated $\dot{V}O_2$max values of the male and female subjects in either the trained or untrained subject groups.

The trained subject group reported to complete an average of 15.1 ± 3.3 hours of training per week while the untrained subject group reported to complete 2.2 ± 1.8 hours per week, given that some subjects were undertaking no exercise on a regular basis.

The pre-trial hydration schedule was successful as all subjects reported to the laboratory in a hydrated state (USG less than 1.015). There was no significant difference ($p>0.05$) between USG readings in the summer and winter trials in either group.

Table 16. Physical characteristics of subjects in the trained and untrained

groups in both the summer and winter testing periods

Group		Age (years)	Weight (kg)		$\dot{V}O_2$max (ml/kg/min)	
			Summer	Winter	Summer	Winter
Trained (n=26)	**Male (n=15)**	28.6 ± 5.7	79.4 ± 7.7^2	80.3 ± 7.0^2	64.6 ± 7.4	62.2 ± 9.3
	Female (n=11)	24.8 ± 5.6	65.3 ± 5.5	65.5 ± 6.0	58.7 ± 8.4	59.2 ± 7.6
	Average (n=26)	26.3 ± 6.0	73.5 ± 10.6^1	74.3 ± 9.9^1	63.3 ± 9.9^1	60.8 ± 9.8^1
Untrained (n=22)	**Male (n=9)**	26.9 ± 6.3	73.7 ± 7.8^2	75.4 ± 8.1^2	39.1 ± 6.0	39.1 ± 6.1
	Female (n=13)	23.5 ± 6.0	55.9 ± 4.9	57.0 ± 5.5	37.1 ± 6.0	39.1 ± 4.6
	Average (n=22)	24.8 ± 6.2	63.2 ± 10.8	64.2 ± 11.2	37.2 ± 6.0	38.1 ± 5.1

Note: Values are mean ± standard deviation; [1]Significantly different from untrained group (p<0.05). [2] Male group is significantly different from female group (p<0.001)

<u>Sweat rates</u>

Based on the mixed design repeated measures ANOVA, there was no significant interaction (p=0.915) between seasons and training status for sweat rates. There was however a significant main effect of training and season on sweat rates (p<0.001). The trained subject group had significantly higher (p<0.001) sweat rate in both the summer and winter periods than the untrained subject group (see Table 17. and Figure 14).

For sweat sodium and sweat potassium outcomes, the mixed design repeated measures ANOVA was performed for observations measured from two body sites (leg and arm), as well as the mean value of the two body sites. Estimated sweat sodium/potassium loss (which was obtained based on actual sweating rate and average sweat sodium/potassium concentration taken from the two body sites) was also calculated.

<u>Sweat Sodium</u>

For arm sweat sodium concentrations, no significant interaction between seasons and training status (p=0.477) and no effect of training status (p=0.221) was found. The main effect of season was significant (p<0.001). Irrespective of training status, sweat sodium concentrations observed from the arms in the winter period were higher by 8.97 mmol/L (95% CI: 4.81, 13,13) compared to the average summer values. A similar trend was found for sweat sodium concentration measured from legs. No significant interaction between seasons and training status was found (p=0.897 for the legs and p = 0.4 for the average concentration). There was also no significant effect of training status (p = 0.301 for the legs and p = 0.212 for the average concentration). The impact of seasons was significant on both the legs (p=0.001) and average concentrations (p<0.001). The sweat sodium concentration observed in the winter period was higher by 8.13 mmol/L (95% CI: 3.72, 12.54) for leg concentrations and 7.94 mmol/L (95%CI: 3.82, 12.05) for average concentrations, than in the summer period.

When looking separately at the trained and untrained subject groups, paired samples t-test results showed that there was a significantly greater sweat sodium concentration in the winter period in the arms and legs than in summer in both groups (p<0.05). In the untrained subject group, sweat sodium concentration in the

arms and legs in the winter period was higher by 10.45 mmol/L (95% CI: 5.16, 15.74) and 9.21 mmol/L (95% CI: 2.55, 15.86) respectively while in the trained subject group sweat sodium values in the arms and legs in winter were higher by 7.48mmol/L (95% CI: 1.03, 13.94) and 7.22 mmol/L (95% CI: 0.93, 13.51) respectively (Table 17.

The mixed design repeated measure ANOVA failed to find a significant interaction (p= 0.165) and main effect of seasons (p= 0.150) on sweat sodium loss. However there was a significant main effect of training found on estimated sweat sodium loss (p<0.001). Estimated sodium loss in summer in the trained group was greater than in winter, while in the untrained group, sodium loss was greater in the winter. The trained subject group lost 2.4 and 1.9 times more sodium than the untrained group in the summer and winter periods respectively (Table 17. and Figure 15).

Sweat Potassium

For sweat potassium observations measured from arms, legs and average concentration, no statistical significant effect of interaction (p = 0.260 for arms, p = 0.299 for legs, and p = 0.184 for average, respectively), no effect of season (p= 0.862 for arms, p = 0.114 for legs, and p = 0.285 for average, respectively) and no effect of training status (p = 0.521 for arms, p = 0.236 for legs, and p = 0.310 for average, respectively) were found.

When looking separately at the effect of season on the trained and untrained subject groups, paired samples t-test analysis showed that there was a significantly greater sweat potassium concentration (p=0.044) in the summer period in the legs in the trained subject group only. Leg sweat potassium concentration in the summer was higher on average by 0.68mmol/L (95%CI: 0.02, 1.35) than in the winter period.

For estimated sweat potassium loss, a significant ordinal interaction between seasons and the training status was found (p<0.001), indicating the effect of seasons on estimated sweat potassium loss was modified by training status. The difference in mean estimated sweat potassium loss between summer and winter for the untrained group was significantly different compared to that for the trained group. In the trained group, the reduction in estimated sweat potassium between the summer and winter periods was 157mg/h compared to 26mg/h in the untrained group.

Table 17. Sweat rates (L/h), sodium and potassium concentration (mmol/L) and estimated daily sodium and potassium loss (mg/h) in both the trained (n= 26) and untrained groups (n=22) in the summer and winter months.

Variable	Average		Trained		Untrained	
	Summer	Winter	Summer	Winter	Summer	Winter
Sweat rate (L/h)	1.3 ± 0.5	1.2 ± 0.5	$1.7 \pm 0.4^{1,3}$	1.5 ± 0.4^{3}	0.9 ± 0.2^{1}	0.8 ± 0.2
$[Na^{+}]$ arms (mmol/L)	46.4 ± 15.5^{1}	55.2 ± 16.4	49.4 ± 16.5^{2}	56.9 ± 18.0	42.8 ± 13.6^{1}	53.3 ± 14.7
$[Na^{+}]$ legs (mmol/L)	40.7 ± 14.5^{1}	48.8 ± 15.1	43.0 ± 16.3^{2}	50.3 ± 14.4	37.9 ± 11.7^{1}	47.1 ± 16.2
$[Na^{+}]$ average (mmol/L)	43.8 ± 15.3^{1}	51.9 ± 15.1	47.0 ± 17.2^{2}	53.3 ± 15.9	40.4 ± 12.3^{1}	50.2 ± 14.5
$[K^{+}]$ arms (mmol/L)	7.9 ± 2.0	7.8 ± 2.7	8.0 ± 2.3	7.4 ± 2.8	7.9 ± 1.7	8.2 ± 2.6
$[K^{+}]$ legs (mmol/L)	8.1 ± 1.7	7.7 ± 2.1	8.0 ± 2.0^{2}	7.3 ± 1.9	8.3 ± 1.3	8.1 ± 2.3
$[K^{+}]$ average (mmol/L)	8.0 ± 1.7	7.7 ± 2.0	8.0 ± 2.0	7.3 ± 2.0	8.1 ± 1.4	8.2 ± 2.0
Estimated $[Na^{+}]$ loss (mg/h)	1507 ± 948	1458 ± 807	2048 ± 947^{3}	1869 ± 800^{3}	869 ± 394	972 ± 490
Estimated $[K^{+}]$ loss (mg/h)	442 ± 198^{1}	344 ± 151	$575 \pm 146^{1, 3}$	418 ± 134^{3}	284 ± 119	258 ± 124

[1]Summer values are significantly different from winter values (p<0.01); [2]Summer values are significantly different from winter values (p<0.05); [3]Trained subject results are significantly different from untrained results (p<0.01); [4]Trained subject results are significantly different from untrained results (p<0.05).

Figure 14. Mean sweat rates (L/h) for both trained (n=26) and untrained (n=22) subjects in both the summer and winter trials.

Graph displays mean ± Standard error
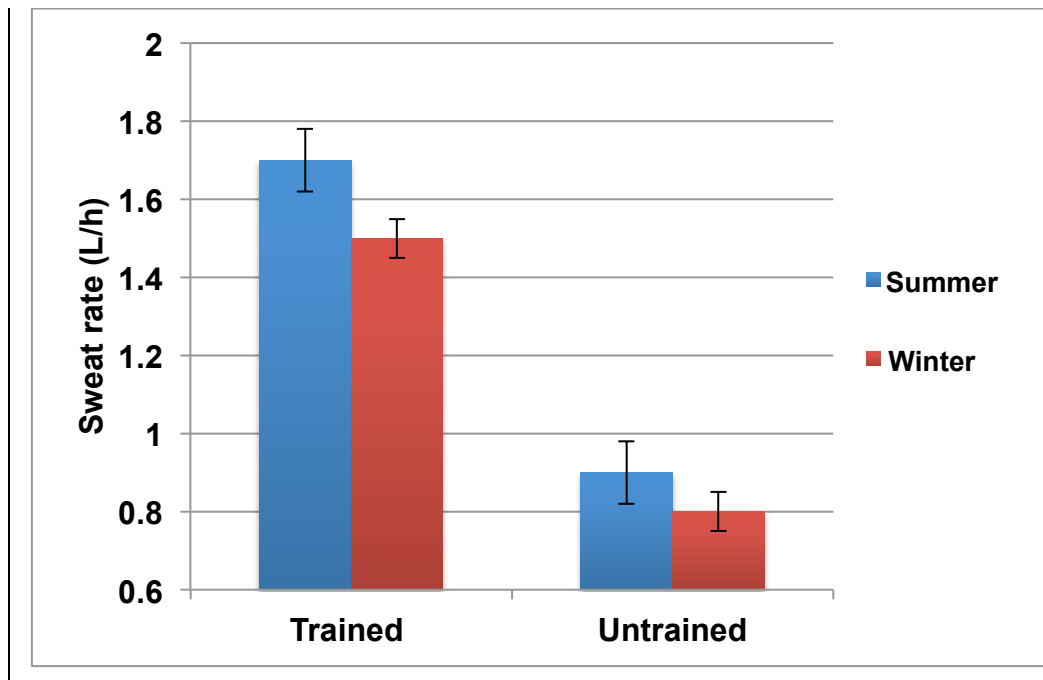
Summer values are significantly different from winter values in both groups (p<0.01)

Trained subject results are significantly different from untrained results (p<0.01);

Figure 15. Estimated sweat sodium loss (calculated based on sweat rate and sweat sodium concentration) in both the trained (n=26) and untrained (n=22) subject groups during both the summer and winter testing periods.

Graph displays Mean ± Standard error
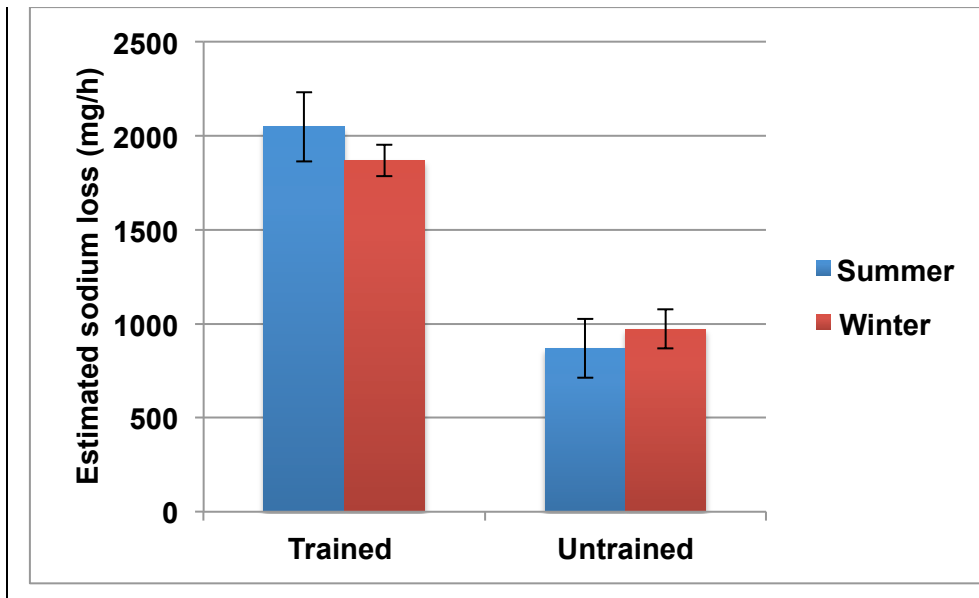
Trained subject results are significantly different from untrained results (p<0.01)

Figure 16. Estimated sweat potassium loss in both the trained (n=26) and untrained (n=22) subject groups during both the summer and winter testing periods.

Graph displays Mean ± Standard error
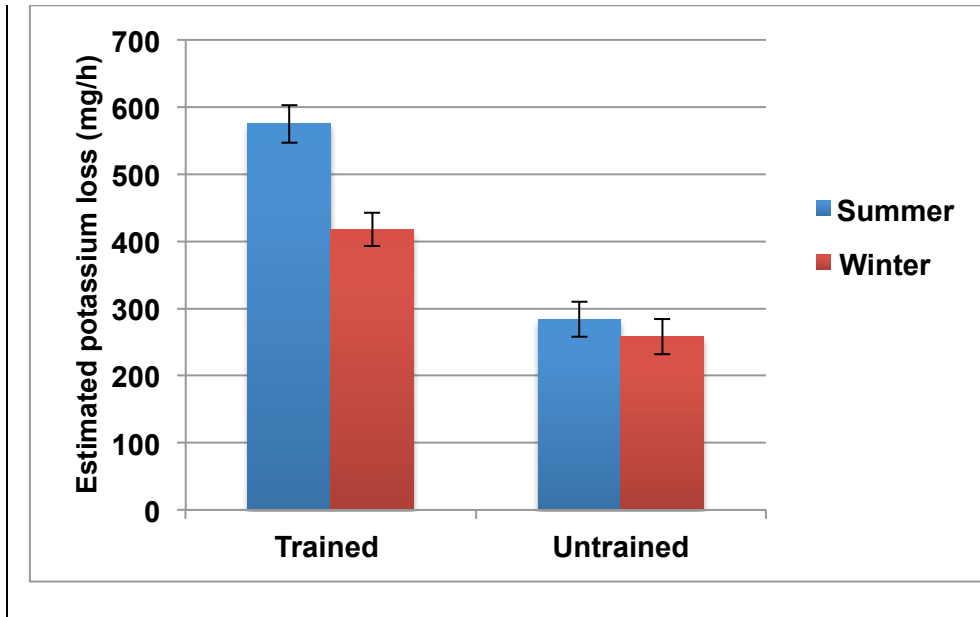
Summer values are significantly different from winter values in the trained group (p<0.01)

Trained subject results are significantly different from untrained results (p<0.01)

5.4 Discussion

5.4.1 Part A: The Effect of Heat Acclimatisation on Sodium and Potassium Losses in Endurance Trained Subjects.

This study is the first of our knowledge to report the effect of heat acclimatisation on sweat sodium and potassium loss in a group of endurance athletes. It is clear from these results that repeated exercise in the heat over a prolonged period of time results in physiological adaptations that improve the ability to tolerate exercise in the heat.

The main findings from this study were significant increases in sweat rate and sweat potassium concentration in the legs and a significant decrease in sweat sodium concentration at the end of the summer period when athletes were assumed to be heat acclimatised. These findings are in support of previous work by Bates and Miller (2008), Nielson and Strange (1997) and Kirby and Convertino (1986) who demonstrated significant increases in sweating rates and decreases in sweat sodium concentration in a group of untrained subjects after a period of heat acclimatisation. To the best of our knowledge this is the first study to report variation in sweat potassium concentrations after heat acclimatisation.

The mechanism responsible for the increase in sweating rate remains unclear and the glandular mechanisms have not been addressed in the present study. It has been proposed that increases in sweat rate after a period of heat acclimatisation are due to an increase in the size of the eccrine sweat gland (Sato et al. 1990), an increase in the number of activated sweat glands or an increase in output per gland (Peter and Wyndham 1966) reflecting adaptive changes in thermoregulation. Another potential mechanism is that blood volume expansion may attenuate the competition between metabolic and thermoregulatory demands for blood flow (Rowell 1974). This in turn will increase cutaneous blood flow, promote convective heat transfer and fluid delivery to the sweat glands contributing to an increased sweat rate (Nadel 1984)

Kanikowska et al (2010) recently investigated seasonal changes in hormonal and thermoregulatory responses at four time points throughout the year. Aldosterone (ALD) and antidiuretic hormone (ADH) concentrations were shown to be significantly higher at the end of the summer period. Sweat rate was also reported to be significantly higher in the summer months in a group of 8 untrained students. The authors of this paper concluded that elevated hormone levels may be a compensatory

mechanism to avoid dehydration during the summer period through both water and sodium conservation in the kidney and the sweat gland (Kanikowska et al. 2010). As this study was performed in untrained subjects, a study designed to investigate hormonal responses in trained athletes would be an interesting comparison. Given that athletes are known to have higher average sweat rates, greater increases in both ALD and ADH may be expected as a mechanism to further prevent dehydration.

Numerous studies in the literature have reported a reduction in sweat sodium concentration following a period of heat acclimatisation (Bates and Miller 2008; Chinevere et al. 2008; Kirby and Convertino 1986; Nielsen et al. 1997). Following 8 days of humid heat acclimatisation, Nielsen et al (1997) reported sweat rates increasing by 26% while sweat sodium concentrations decreased from 107mmol/L to 70mmol/L. Kirby et al (1986) reported a 12% increase in sweat rate following a period of 10 days of heat acclimatisation while sweat sodium concentrations decreased by 59% from 88mmol/L to 42mmol/L. Bates and Miller (2008) reported significant seasonal variation in sweat rates and sweat sodium concentrations in a group of untrained subjects. Sweat rates were 13% higher at the end of summer while sweat sodium concentration had decreased from 64 mmol/L to 45mmol/L. Similar results are reported in the present study with a 12% increase in sweat rates at the end of the summer period and a 12% decrease in sweat sodium concentration from 53mmol/L to 47mmol/L in the trained subjects. The variation in sweat sodium concentration between studies may reflect methodological differences as well as variation in the level of acclimatisation.

Buono et al (2007) were the first to report significant decreases in sweat sodium concentration after a period of heat acclimation across a range of sweat rates. The decline in sodium concentration was attributed to an increased sodium absorptive capacity of the sweat gland possibly via the action of aldosterone. Some authors believe that plasma aldosterone concentrations rise following a period of heat acclimatisation (Nielsen et al. 1993) while others report an increased sensitivity to aldosterone when in the acclimatised state (Kirby and Convertino 1986). As aldosterone concentrations were not measured in the present study we cannot be sure of the mechanism involved. Our results however provide evidence of sodium conservation occurring at the level of the sweat gland after heat acclimatisation.

A further interesting finding from this study was a significant difference between potassium concentrations in the summer (8.0mmol/L) and winter (7.3mmol/L) in the leg sweat of the athletes. These potassium concentrations are similar to the levels reported in previous studies (Baker et al. 2009b; Maughan et al. 2005; Meyer et al. 1992). While there was a trend towards a higher potassium concentration in summer for both the arms and legs, there was a significant increase in the legs only. However, in 19 of the 26 subjects, average sweat potassium concentration across all body sites in summer were greater than those reported in winter. At the level of the kidney, aldosterone is known to stimulate sodium conservation as well as potassium secretion. This mechanism may therefore explain why there was an increase in sweat potassium concentration in the summer period when sweat sodium was shown to be significantly lower across all collection sites. It is not clear as to why the legs were shown to have greater losses than the other collection sites. Sodium losses in the legs at the end of summer also demonstrated greater adaptation than the arms. As aldosterone is a systemic hormone, its effects would be expected to be widespread across all body sweat glands. It is possible that the increased metabolic activity in the legs may have increased the sweat gland sensitivity to aldosterone. It is also possible that, as the athletes in this study were triathletes who were regularly cycling and running, both the size and/or density of sweat glands are increased in the highly trained legs. The early work of Sato and Sato (1983) clearly identified that functionally active sweat glands are characterised by greater gland size and density, as well as increased cholinergic sensitivity. Future studies should aim to address whether the increased metabolic heat generated by the active leg muscles during the cycling and running exercise is responsible for greater sweat gland adaptation.

Little is known about the effects of aldosterone at the level of the sweat gland therefore, the role it plays in sweat sodium conservation and potassium excretion is relatively unknown. The few studies that have been conducted have produced conflicting results. The early study by Ladell and Shephard(1961) showed that administration of spironolactone, an aldosterone antagonist, significantly increased both urinary and sweat sodium excretion as previously noted. The major limitation of this study was that they did not control for the differences that occur in sweat sodium concentration with changes in sweat rate. Buono et al (2007) has previously reported that a linear relationship exists between sweat rate and sweat sodium concentration in

untrained subjects and, therefore, the results of Ladell and Shephard(1961) are questioned. The more recent study by Lee et al (2010) examined the effect of spironolactone on sweat sodium secretion, this time accounting for sweat rate differences. The results of this study did not support the conclusions made by Ladell and Shephard(1961) as spironolactone administration resulted in significant natriuresis, however there was no change in sweat sodium excretion. From these results the authors concluded that the non-genomic effects of aldosterone on the sweat gland are different to the actions on the kidney. The most likely explanation for these findings is due to different isoforms of the aldosterone membrane receptor being present in the kidney and sweat gland. Further research is needed to determine if aldosterone receptors in the kidney are distinct from those in the sweat gland.

The results of the present study identify a need for fluid and electrolyte replacement guidelines to consider an athlete's state of acclimatisation. Significant increases in sweat rates have been noted consistently in both trained and untrained individuals (Bates and Miller 2008; Nielsen et al. 1997; Kirby and Convertino 1986). While sweat sodium concentration declined in the summer period in our study, estimated sweat sodium losses were not different between seasons indicating that, due to increased total sweat losses, sodium requirements do not decrease when acclimatised.

Maughan and Shirreffs (2010) have reported that many athletes are incorrectly informed as to their increased fluid requirements when heat acclimatised. Educating athletes and coaches on the need for increased fluid intake both during training and in the recovery phase is vital to maintain fluid balance.

The loss and replacement of potassium in sweat has received very little focus in the literature; however, over the course of an endurance event, losses may be quite considerable. The variation in sweat potassium losses between subjects in this study was large; with the lowest reported concentration being 4.4mmol/L and the highest 13.3mmol/L. This variation indicates that while some athletes may be able to easily replace potassium losses with a normal diet, athletes with losses at the higher end of the spectrum may require additional supplementation. Based on median population intake, the current adequate intake guidelines for potassium intake in Australia are set at 2800mg for women and 3800mg for men (National Health and Medical

Research Council 2005). These levels may not be adequate for an athlete with high sweat losses. For example, an athlete with an average sweat rate of 1.2L/h and a high potassium concentration of 10mmol/L would lose approximately 4.5g of potassium over the course of a 10 hour endurance event. If losses of this magnitude occur regularly there may be associated neuromuscular disturbances and alteration to the acid base balance of cells. The extracellular fluid compartment (ECF) is the initial source of the potassium lost in sweat. The intracellular fluid (ICF), being high in potassium concentration, is then able to replace the lost ECF levels. At low levels of potassium loss this is able to occur without compromise to the cellular acid base balance, however, in cases of high sweat potassium loss, acid base disturbances may occur due to the influx of hydrogen ions into the ICF compartment in order to maintain the electrochemical gradient between the ICF and ECF compartments. Symptoms of depleted intracellular potassium levels include cardiac rhythm abnormalities, muscular weakness and impaired nerve conduction (Hubbard, Szlyk, and Armstrong 1990). Defective storage and synthesis of glycogen has also been noted as a consequence of muscle potassium deficiency therefore significantly limiting endurance performance (Blachley, Knochel, and Long 1974).

For athletes with known high sweat potassium losses competing in endurance events it may be necessary to ensure dietary potassium intake is high as well as regularly consuming an electrolyte replacement beverage containing potassium. A study by Knochel (1977) demonstrated significant potassium depletion in a group of healthy men undergoing 5 weeks of intensive physical training in hot weather. Although subjects were not hypokalemic at the end of the 5 weeks, there was a significant reduction in serum potassium levels. This occurred despite the provision of adequate nutrition to meet their needs and what is considered to be a normal potassium intake of 106mEq/day (4100mg/day). As the potassium losses that occur as a result of continuous sweating has received little focus in the literature, future studies should aim to determine whether potassium supplementation is required, particularly for those athletes competing in ultra-endurance events or those seeking optimal performance. In recreational athletes with relatively low sweat losses, the potassium contained in a normal diet would easily replace these losses.

Conclusion

Seasonal heat acclimatisation in athletes was shown to significantly increase total sweat losses, decrease sweat sodium concentration and increase potassium losses from the legs. The hypothesized increase in aldosterone concentration after a period of heat acclimatisation is the most likely explanation for these findings. As aldosterone concentrations were not measured in this study it is difficult to draw direct conclusions; this would be an interesting follow up study. From a practical viewpoint, educating athletes, particularly those training and competing in endurance events, on the need for their fluid and electrolyte intake to vary depending on their acclimatisation state is vital.

5.4.2 Part B: The Effect of Training Status and Heat Acclimatisation on Sweat Rate and Electrolyte Losses

The aim of part B of this study was to compare the effect of seasonal heat acclimatisation in trained and untrained individuals in order to determine the separate and combined effects of physical training and heat acclimatisation. There is a large body of literature addressing the issue of sweat rates and training. Few studies have examined differences in sweat rates and composition between trained and untrained individuals. A major limitation of each of the previous studies is the small difference in $\dot{V}O_2$max between the trained and untrained groups. In the recent study by Ichinose-Kuwahara (2010) the untrained group had a mean $\dot{V}O_2$max of 43 ml kg^{-1} min^{-1}compared to 53 ml kg^{-1} min^{-1}in the trained subject group. In the present study there was a 26 ml kg^{-1} min^{-1}difference (37 ml kg^{-1} min^{-1}vs 63 ml kg^{-1} min^{-1})in $\dot{V}$O$_2$max between the two groups. The trained group was competitive endurance athletes while the untrained group was participating in less than 5 hours per week of low intensity physical activity.

It has been previously suggested that trained athletes behave physiologically as if they have undergone a period of heat acclimatisation (Taylor and Cotter 2006). Typical adaptations include a lowered resting core temperature and a lower core temperature at the onset of sweating. When heating is coupled with exercise, these adaptations are further pronounced (Taylor and Cotter 2006). We therefore expected the magnitude of any physiological changes to be lower in the trained group in comparison to the untrained subject group.

The hypothesis that sweat glands need to actively produce sweat in order to fully acclimatise has been explored in the literature using pilocarpine to artificially induce sweating. Buono et al (2009) used intra-dermal injections of BOTOX in order to prevent neural stimulation and sweat production during heat acclimatisation. Prior to heat acclimatisation the sweat rate in the BOTOX treated arm and the control arm were equal. Following a period of heat acclimatisation the pilocarpine induced sweat rate in the control arm was 18% higher while the BOTOX forearm had a decrease of 52%. The authors concluded that in order for complete acclimatisation to occur the sweat glands must be active if they are to effectively adapt and increase their sweating capacity.

Several studies however have alluded to the fact that even in the absence of physical activity, partial heat adaptation is evident after repeated exposure to heat (Fox et al. 1967). Both the trained and untrained groups in this study demonstrated an increase in sweat rate at the end of the summer period when they were assumed to be heat acclimatised. This may be due to a centrally mediated decrease in the temperature threshold for vasodilation and sweating onset, known to occur with acclimatisation (Nadel 1984). Our results also demonstrated a slightly greater relative change in both sweat rates and sodium concentration in the untrained group compared to the trained group between seasons. Sweat rates were 14% greater in the summer months compared with the winter in the untrained group compared with a 12% increase in the trained subjects. Sweat sodium concentrations showed greater variation with a 20% decrease in sodium concentration in the summer compared to the winter months in the untrained group compared with a 12% decrease in the trained subjects. This may suggest that the athletes, who were regularly exposed to thermal stress during training, had already experienced some exercise-induced adaptation and therefore the effects of seasonal acclimatisation were not as great when compared to the untrained group. However, as mentioned by Taylor and Cotter (2006) endurance training without regular heat exposure does not provide adequate stimulus for complete heat adaptation. Our results support this as additional thermoregulatory adaptations (increased sweat rate and decreased sweat sodium concentration) were seen in the trained group when seasonally heat acclimatised. Therefore for the endurance athlete it is essential that training in the heat forms a component of their preparation for competition in the heat (Taylor and Cotter 2006).

The environmental conditions at the study location during the summer period are hot and moderately humid, so likely to be sufficient to elicit some acclimatisation response without the need for intense physical training. Very low intensity physical exercise, even incidental exercise, may be sufficient to stimulate active sweating in these conditions. It should also be noted that modern behavioral responses to hot conditions such as living and working in air conditioning and the tendency towards a more sedentary lifestyle could minimise summer induced heat acclimatisation. This was not investigated in the present study however is a potential area for future research.

The other notable finding from Part B of this study was that the untrained subject group had significantly lower ($p<0.05$) mean sweat rates in both the summer (0.90L/h) and winter (0.78L/h) periods compared to the trained group (summer 1.73L/h, winter 1.53L/h. There are several possible explanations for this finding:

In order for the trained subjects to reach the 50% $\dot{V}O_2$max they are required to perform at a greater metabolic work rate, therefore producing greater metabolic heat than the less aerobically trained subjects. Therefore in order to maintain heat balance and dissipate this heat, the physically trained group requires a higher sweat rate.

The untrained subject group also had a greater number of females (13 vs 11) than the trained subject group. It has previously been reported in that females display lower mean sweat rates than males at the same workload possibly due to their larger surface area to mass ratio and improved thermoregulatory efficiency (Inoue et al. 1998; Ichinose-Kuwahara et al. 2010). There is also some evidence to suggest that testosterone is involved in enhancing the sweating response while estradiol may inhibit it (Kawahata 1960). The increase in testosterone as a result of physical training is also significantly greater in males than females (Keizer et al. 1989).

A further possible explanation is due to increased thermoregulatory efficiency that is gained as a result of physical training therefore resulting in improved heat loss mechanisms. This is believed to be due to a reduction in the temperature threshold at which sweating is initiated (Nadel 1984), an increase in the size and number of activated sweat glands as well as increased cholinergic sensitivity (Sato et al. 1990; Buono and Sjoholm 1988). Buono and Sjoholm(1988) have reported that sweat rate is significantly greater in the trained individual compared to untrained due to a greater number of activated sweat glands (ASG) and sweat output per gland (SGO).

The recent study by Henkin et al (2010) provided some evidence to suggest that the thermal adaptations seen in trained subjects come about as a result of constant exposure to heat rather than as a direct result of physical fitness. In this study sweat rates and electrolyte concentrations in swimmers, runners and nonathletes were compared. The sweat rates in swimmers (0.9L/h) was significantly lower than the runners (1.5L/h) and similar to the nonathletes (0.6L/h). Similar results were shown when analysing sweat sodium concentrations. The swimmers and nonathletes had sodium concentrations of approximately 65mmol/L while significantly lower average

concentrations were shown in the runners (45mmol/L). The authors concluded from this study that the degree of thermal load experienced regularly during training is the primary factor influencing the adaptations to the heat. While swimmers have a large aerobic capacity, their training environment allows for their core and skin temperature to remain largely unchanged therefore resulting in low levels of thermal stress. The athletes in the present study were triathletes who were spending the majority of their training either cycling or running therefore experiencing conditions of high thermal load regularly. The mean sweat rates of 1.5-1.7L/h in this group are similar to those reported in the runners in the above-mentioned study.

In order to accurately determine the effect of physical training on sweat rate, a comparison between trained and untrained male and female subjects, exercising at the same absolute workload would need to be made. It is possible to make an indirect comparison using the data collected in both of the heat chamber studies reported in this thesis (Study 2 and 3). The trained group of subjects involved in Study 2 of this thesis, investigating the effect of exercise intensity on sweat rates and sodium concentration, had an average $\dot{V}O_2$max of 60mL/kg/min. At the lowest intensity trial where the subjects worked at 60% of age adjusted maximal heart rate (220-age), equivalent to approximately 40% $\dot{V}O_2$max (24mL/kg/min), the average sweat rate recorded was 1.0L/hr. This data was collected at the end of the summer period when the subjects were assumed to be heat acclimatised. Similarly, in Study 3, the heat acclimatisation trial, the untrained subjects in the summer period had an average $\dot{V}O_2$max of 40mL/kg/min and were required to exercise at a workload of 70% of age adjusted maximal heart rate (220-age), equivalent to approximately 50% $\dot{V}O_2$max (20mL/kg/min). At this workload average sweat rates of 0.9L/hr were reported, a similar value to that of the trained subjects at a similar workload. This information suggests that the greater sweat rates reported in the trained subject group may largely be due to the increased metabolic work rate required to reach the set workload. Future studies should aim to address the effect of physical training on sweat rates by matching absolute workload of the two groups.

The other significant finding from part B of this study was that there was a significant decrease in sweat sodium concentration at the end of the summer period across all body sites in both the trained and untrained subject groups. There was slightly greater sodium conservation observed in the untrained group at the end of the

summer period when acclimatised. On average the summer sodium concentration was 80% of the winter concentration in the untrained group compared to approximately 85% in the trained group. This could be explained by the fact that the trained group are constantly exposed to a heat load through exercise and therefore are partially heat acclimatised. In comparison the untrained group, without any prior exposure, responds to the effect of seasonal change more strongly.

There was no significant difference in average sodium concentration (mmol/L) between the trained and untrained groups in either the summer or winter period. Roberts et al (1977) states that since training and heat acclimatisation share similar adaptations, it is possible that trained athletes could have lower than average sweat sodium concentrations. Few studies have investigated whether differences in sweat sodium concentration exist between trained and untrained individuals. The studies that have been conducted have produced conflicting results. There is information discussed in a textbook by Wilmore and Costill (2004) which suggests that sweat sodium concentration in trained individuals is lower than untrained subjects even prior to heat exposure. This information has been criticised however due to the lack of recording of exercise intensity. It has been previously shown that exercise intensity determines sweat rate which in turn affects sweat sodium concentration (Buono et al. 2008). A recent study by Hamouti et al (2011) did not support the information reported by Wilmore and Costill (2004), showing that aerobic fitness level does not reduce sweat sodium concentration when normalised for sweat rate.

A further significant finding from the present study was that there was a significant difference between estimated sweat sodium loss (mg/h), which also considers sweat rate, between the two groups in both summer and winter. Total sodium loss in the trained group was 2048mg/h in summer compared with 869mg/h in the untrained group. At the end of the winter period total sodium loss in the trained group was 1869mg/h compared to 972mg/h in the untrained group when exercising at the same relative intensity. This information is relevant to the area of sports nutrition as aerobic fitness level will potentially influence the degree of sodium loss and therefore determine sodium intake requirements. Recreational athletes with low to moderate sodium losses coupled with low training outputs will easily be able to meet their sodium intake requirements following a normal moderate sodium diet as recommended by the Heart Foundation. Well trained endurance athletes however

with higher sodium losses and extreme training outputs will often have much higher requirements than the current recommendations. In addition to this, many endurance athletes are also health conscious and may be actively restricting dietary salt intake through low intake of processed foods without being aware of their increased needs.

The importance of exposure to thermal load in order to maximize adaptation to the heat may be of relevance to athletes who are travelling to compete in a hot climate. Prior exposure to a thermal stimulus will ensure that athletes optimise performance while minimising the risk of heat related illness. The length of time required to maximize these effects is not entirely clear however for most individuals between 7-14 days of heat exposure is sufficient to develop full adaptation (Wendt, Van Loon, and Lichtenbelt 2007).

Limitations of the study

It is important to note the methodological limitations of this research project. Athletes involved in the study were assumed to be heat acclimatised leading into the summer testing as they had been regularly training outdoors in the hot and humid conditions typical of a Perth summer. The exact details relating to time spent training outdoors in the months leading into the study was not known and therefore may have influenced the acclimatisation state of the athletes involved.

Another limitation that should be addressed is that the menstrual cycle was not accounted for when testing the female athletes. Core temperature has previously been shown to vary depending on the phase of the menstrual cycle (Janse et al. 2012). There has also been some evidence to suggest that sweating rates increase in women during the luteal phase of the menstrual cycle (Garcia et al. 2006).

While every effort was made to ensure that the sweat collecting devices were placed in the same position on each subject, there was potentially some minor variation, as placement site was not marked up prior to study commencement. The strong correlation between right and left limbs suggests that this variation is minor.

Conclusion

Sweat rates were shown to be significantly greater in the trained subject group compared with the untrained group. There are several possible explanations for this finding, one of them being due to the sweat gland adaptations that occur as a result of

physical training. Future studies should address the effect of physical training on sweat rates when athletes are working at the same absolute work rate. There was no significant difference between sweat sodium concentration noted between the trained and untrained subject groups, however due to significantly higher sweat rates in the trained subjects, estimated sweat sodium loss was significantly greater. From a practical viewpoint, athletes with high aerobic fitness, who are training in endurance based events, should be aware of their increased fluid and sodium needs particularly if training or competing in hot and humid environmental conditions.

Chapter 6: **General Conclusions and Recommendations for Fluid and Electrolyte Replacement**

6.1 Overview

Over the last decade there has been a number of position statements published on the need for adequate fluid replacement particularly in those individuals who are exposed to a hot environment for prolonged periods of time (Sawka et al. 2007; Convertino et al. 1996; Casa et al. 2000). There has been far less published work investigating the electrolyte losses, particularly sodium and potassium, accompanying large sweat losses. In both athletes and manual workers it is known that large sweat losses can result in significant losses of fluid and other sweat constituents mainly sodium and potassium. It has been documented previously that large sodium losses can have detrimental effects on both the physical and cognitive performance of an individual, while losses in large quantity can result in a significant risk to health. The loss of sodium in sweat has been linked to hyponatremia (Montain, Cheuvront, and Sawka 2006), muscle cramping (Horswill et al. 2009; Donoghue, Sinclair, and Bates 2000) and alterations in fluid balance (Sawka 1992). Current sodium replacement recommendations for the general population place emphasis on the need to reduce sodium intake in order to prevent hypertension and the link with coronary heart disease. These recommendations are potentially set too low for athletes and manual workers with high sweat rates who are working or exercising for prolonged duration. There is a need for separate sodium replacement guidelines for these populations.

The aim of this thesis was to address the following questions:

1) Are manual workers who are performing prolonged periods of work in the heat plus consuming a potentially low sodium diet, at risk of hyponatremia? **(Chapter 2)**

2) What are the current levels of dietary sodium intake in a population of manual workers from the Middle East? **(Chapter 2)**

3) Is there a relationship between sweat rate and sweat sodium concentration in well trained endurance athletes? **(Chapter 4)**

4) What is the effect of seasonal heat acclimatisation on sweat rates and electrolyte (sodium and potassium) concentrations? **(Chapter 5)**

5) Does physical fitness change the response of an individual to seasonal heat acclimatisation? **(Chapter 5)**

6) Are there individuals or specific situations that may require an increased sodium and potassium intake? **(Chapters 2, 4 and 5)**

6.2 Fluid intake recommendations for manual workers

Several recent studies have shown that manual workers regularly begin work in a hypohydrated state (Bates and Schneider 2008; Miller and Bates 2007b; Bates, Miller, and Joubert 2010c). This finding has been consistent across a range of cultural, ethic and economic backgrounds indicating that this population is challenged by hydration issues (Miller and Bates 2009). Working in a dehydrated state places workers at a significantly increased risk of developing all forms of heat illness. Dehydration in the workplace can also affect productivity, morale and may increase the risk of workplace accidents. The risk is substantially greater when working for prolonged periods in thermally stressful environments prompting high sweat losses. As mentioned previously in the literature review of this thesis, it is not uncommon for workers to lose up to or exceeding 1L/h (Brake and Bates 2003a; Miller and Bates 2007b). Over the course of a 12 hour work shift this can equate to over 12L of fluid loss. This becomes a significant issue when work is repeated for 6 or even 7 days of the week, which is common practice in many workplaces.

Educating workers on the need to consume adequate quantities of fluid both before and during work is an important strategy in order to address this issue. An

interventional study conducted in the Middle East by Bates and Schneider (2008) showed that educating workers with simple fluid intake strategies significantly increased fluid consumption while reducing physiological strain associated with work in the heat.

Discussion with medical staff and workers at the workplace studied in Chapter 2 highlighted the misinformation surrounding adequate fluid and electrolyte replacement. There is need for education as to the most appropriate fluid for prolonged work in the heat (Bates and Miller 2008). Many of the commercially available sports drinks are hypertonic in relation to plasma and therefore delay both gastric emptying from the gut and absorption from the small intestine (Valentine 2007). Over the course of a 12 hour working day, regular consumption of these fluids would greatly increase sugar and total energy (kj) intake to unadvisable levels. Conversely, the consumption of plain water may result in a dilution of the plasma further increasing the risk of hyponatremia (Noakes 2002). The most appropriate beverage is one which is hypotonic to plasma, containing glucose and sodium in levels which help to maintain plasma volume and blood glucose levels, while maximizing absorption from the gut. Sodium is required in a beverage in order to promote fluid consumption and help to increase glucose absorption (Valentine 2007).

6.3 Electrolyte Replacement Recommendations for manual workers

The results of study 1 (Chapter 2) of this thesis have highlighted the need for sodium replacement guidelines appropriate for manual labourers to be developed. This is particularly important in countries such as the Middle East where the typical diet of workers consists primarily of rice and legumes with very low total sodium content. Educating workers on the need for adequate salt intake is vital given the public health message is strongly advocating a global reduction in salt intake. A number of the medical staff and workers interviewed as part of this study were misinformed as to the need for increased salt intake when working in conditions of thermal stress.

The following recommendations were made to the medical staff at the workplace studied. These guidelines were based on previous work investigating the fluid needs of manual workers (Brake and Bates 2003a). In addition to the recommendations for

fluid consumption, the present study supports recommendations for electrolyte replacement in the form of fluid or regular food intake. These guidelines could be adapted to suit the needs of any occupational setting where workers are performing physical labour in the heat coupled with a diet low in salt or processed foods.

Table 18. Fluid and electrolyte recommendations for workers performing

manual work in thermally stressful environments while consuming a

predominantly starch based diet.

Role in the workplace	Recommendations
For all workers	• Workers are to carry their own personal water bottle with them to their work area so they are able to assess their individual fluid intake. • Urine specific gravity readings prior to the start of a work shift should be <1.015 • Educating both medical staff and workers on the need for adequate fluid and electrolyte consumption is vital. Workers particularly need to be aware of their increased needs during the summer period when sweat losses are substantially increased. It is recommended that the work management strategy for hot workplaces includes ongoing training in the importance of hydration and electrolyte replacement. • An appropriate electrolyte replacement formula developed for use in the industrial setting should be made available to workers in unlimited quantity particularly during the summer period. If workers are able to snack every couple of hours the need to consume an electrolyte replacement drink is reduced. ○ It is important to ensure that this fluid is kept cool and therefore increase palatability

For workers in physically demanding roles	• Approximately 1 litre of electrolyte replacement formula per hour of working time is advised • If possible, the consumption of salty snacks during meal breaks is advised • Informing these workers on the need to supplement their meals with additional salt is warranted. When meals are predominately rice and legume based, 1-2 pinches of salt (~2g salt or 800mg sodium) per meal is advised or alternatively salt could be added to food during cooking.
For machinery operators and workers in sedentary roles	• 400 – 600ml of electrolyte replacement formula per hour of working time is advised • Informing workers of the need to supplement rice and legume based meals with additional salt during the summer months is warranted.

6.4 Fluid intake recommendations for athletes

The importance of replacing both fluid and electrolytes lost in sweat continues to be a topic that is debated by many sports scientists (Beltrami et al 2008). There has also been debate over the real-word practical application of some of the developed hydration guidelines for athletes (Garth and Burke 2013). As mentioned in the literature review, there has been a paradigm shift in fluid intake recommendations over the last 50 years, which have ranged from 'not drinking at all' to 'drink as much as tolerable' (Noakes 2007). There is also debate over whether the current guidelines are practical in a real world setting where there are many other factors influencing fluid consumption (Garth and Burke 2013).

Noakes (2008) argues that thirst is the best physiological indicator of fluid needs during exercise and that athletes should drink only when thirsty. The basic premise of Noakes' ideas is that humans are able to subconsciously self regulate fluid intake when under duress. Several authors in the literature however have stated that thirst is an inadequate indicator of the fluid needs of athletes (Convertino et al 1996; Casa et al 2000). A recent article by Garth and Burke (2013) discusses the complex array of factors, which influence an athlete's opportunity to drink, many of which are out of the athlete's control, therefore making it difficult for fluid intake to be truly 'ad libitum'. Factors such as the availability of fluids, the rules and regulations of the sport regarding when is appropriate for drinking to take place, as well as in the elite competitor, the need to maintain speed and technique, which can all influence the volume of fluid consumed (Garth and Burke 2013). Noakes (2002) raises concern over the current fluid intake guidelines which he believes promote overdrinking which can have fatal consequences in an endurance event. There are reports in the literature of exercise-associated hyponatremia occurring over the course of an endurance event, potentially due to excessive fluid intake (Almond 2005). This situation however is more common in slow paced participants as they take more frequent breaks with a larger fluid volume consumed than do faster paced participants (Speedy et al. 2001). It is important that recreational competitors are aware of the consequences of overdrinking during long duration events (Garth and Burke 2013).

This situation, which allows for overconsumption as mentioned above, is not the case across all sporting events. Athletes who are competing in multiple events per day or training twice daily may commence an exercise session with a fluid deficit due to failure to replace large fluid losses from previous sessions. In these athletes an individual, paced fluid plan may be advantageous (Garth and Burke 2013). Casa et al (2000) have reported that if an athlete has in excess of 24 hours between exercise sessions, adequate fluid restoration can occur from normal food and fluid intake, however, when training twice daily and the fluid deficit is large, athletes may fail to adequately replace losses. Silva et al (2011) showed that in a group of youth soccer players training twice daily, a substantial proportion of the players were hypohydrated prior to training and continued to voluntarily dehydrate during the training session. Soccer is an example of a sport in which there are limited opportunities for fluid intake. The authors on this paper concluded that, as sweat losses can be substantial in these athletes, educating athletes on their individual fluid losses may be more appropriate than relying on thirst alone (Silva et 2011).

The information collected in many previous studies as well as in both of the heat chamber studies in this thesis has shown that there is a wide variation in sweat rates in athletes. Individual fluid needs are therefore also widely variable depending on factors such as the environmental conditions, state of heat acclimatisation, physical fitness and activity intensity and duration (Baker et al. 2011). The results of chapter 4 of this thesis showed that sweat rates varied from 700ml/h to 2.6L/h in one athlete. A similar range in sweat rates has been shown in previous studies (Maughan et al. 2005; Shirreffs et al. 2005). It is impractical and dangerous for athletes to aim to replace all fluid losses incurred as a result of prolonged exercise in the heat. Current fluid replacement guidelines encourage athletes to drink enough to maintain body weight to within 2% to prevent performance impairment and reduce heat illness (Sawka et al. 2007). In an ACSM roundtable discussion on hydration and physical activity, Casa, Clarkson and Roberts (2005) identified a need for individualised fluid replacement strategies to be devised particularly for athletes with large sweat losses (>1L/h). It was also advised that athletes should learn to estimate sweat rate to optimize hydration strategies for long distance events (Casa, Clarkson, and Roberts 2005)

Because of the multitude of factors influencing sweat rate and the many sports specific differences in the factors influencing fluid intake during exercise, it is inappropriate to prescribe a fluid and electrolyte regime that meets the needs of all athletes. Athletes need to be educated on the importance of recognizing their individual needs based on their estimated sweat losses and to develop strategies to overcome practical issues in maintaining fluid intake during exercise (Casa, Clarkson, and Roberts 2005).

What is clear from the current literature in this area is that further studies investigating hydration practices of athletes in a field setting are required. As the needs, and factors influencing fluid consumption in recreational and elite competitors vary considerably, there may be a need for different hydration guidelines for each group. As there is currently large numbers of recreational athletes participating in endurance and ultra endurance events, there is a public health need for data to be collected on the hydration practices of these athletes who may be at risk of hyponatremia due to excessive hypotonic fluid intakes (Garth and Burke 2013).

6.5 Electrolyte replacement recommendations for athletes

In both studies in the separate athlete groups there was also shown to be a wide variation in mean sweat sodium losses with the following factors shown to influence sodium losses. In terms of developing guidelines for sodium intake in athletes, this information indicates that again recommendations need to be based on the needs of the individual athlete rather than providing a blanket guideline for all. What was clearly shown from the data collected in both athlete studies was that a large number of athletes require sodium in greater quantity than what is currently recommended for the average person. Currently the Australian Heart Foundation recommends an intake of less than 6g salt per day (2400mg sodium). Data from these studies showed that over a modest 2 hour exercise session an athlete could lose between 600 – 6000mg sodium. Clearly an athlete at the higher end of this scale would require significantly greater quantities of sodium than these recommendations, while an athlete at the lower end could easily meet their requirements from a normal diet. There is a known link between high dietary salt intake and blood pressure it is therefore not appropriate to recommend that all athletes consume a high salt diet or

consume an electrolyte replacement beverage during exercise, the needs of the individual athlete must be considered.

Based on the results of the studies conducted as part of this thesis the following factors need to be considered in forming sodium guidelines for athletes:

- **_Sweat Rate_** – The results from heat chamber study 1 (Chapter 4) showed a fair positive linear relationship between an athlete's sweat rate and sweat sodium concentration. This information suggests that athletes with higher than average sweat rates are therefore more at risk of sodium depletion due to increased sodium concentration of sweat as well as large total sodium losses. Calculating sweat rate from body weight loss over an exercise session is a relatively simple procedure and is practical for use in large athlete groups (Cheuvront, Haymes, and Sawka 2002). Further research is required in a range of athletes competing in different sports to determine whether sweat rate can be accurately used as a measure from which to predict sweat sodium concentration.

  Early identification of athletes with high sweat losses is an important strategy in order to avert potential heat related illness as well providing appropriate fluid and electrolyte replacement strategies to maintain performance.

- **_Intensity of Exercise_** – The results from heat chamber study 1 (Chapter 4) showed on average a 60% increase in sweat sodium concentration and a 50% increase in sweat rates from the low to the high intensity exercise trials. This increase was reported in 14 of the 18 subjects tested. This data is similar to that reported previously by Buono et al (2007) and Yoshida et al (2006). It is important to note that fluid and sodium requirements will vary significantly depending on the duration and intensity of exercise.

- **_Physical Training Status_** – The results from heat chamber study 2 (Chapter 5) showed a significant difference between mean sweat rates in the trained and untrained subjects groups in both the summer and winter months. Although there appeared to be no effect of training status on mean sweat sodium concentration in either season, the untrained group demonstrated a

slightly greater relative decrease in sweat sodium concentration at the end of the summer period. These results are similar to the recent findings of Hamouti et al (2011) who reported no significant differences between sweat sodium concentrations in trained and untrained subjects exercising at the same relative intensity. Due to the significantly higher sweat rates in the trained subject group in both seasons, estimated total sodium loss (mg/h) was significantly higher. Dietary sodium requirements are therefore also higher in the trained group in order to replace these losses.

It is often expected that well trained athletes will consume greater than average dietary sodium intakes simply due to their high energy requirements. This has been shown in several studies to not be the case, with athletes reported to be consuming sodium in similar quantities to that of untrained individuals (Hinton et al. 2004). There are several explanations for this including the possibility that energy requirements in endurance athletes are often over estimated with athletes regularly consume significantly lower intakes than estimated. Health conscious athletes may also be actively restricting sodium intake through limiting processed food intake and avoiding adding additional salt when cooking.

This information has practical implications for the development of sodium intake guidelines. Athletes at risk of sodium deficiency due to high sweat losses coupled with the consumption of a relatively low sodium diet, need to be educated on the need for both fluid and sodium replacement particularly when training twice daily or for prolonged duration.

- *__State of Acclimatisation__* – A period of seasonal heat acclimatisation was shown in heat chamber study 2 (Chapter 5) to significantly increase sweat rates, decrease the sodium concentration in sweat in both the trained and untrained subject groups, and increase the potassium concentration in the legs samples only in the trained subject group. Sweat sodium concentrations in the trained group decreased by an average of 6 mmol/L at the end of the summer months when the athletes were assumed to be heat acclimatised. In the untrained subject group, seasonal acclimatisation resulted in an average decrease of 10 mmol/L at the end of summer. In both groups average sweat

rates were also significantly greater at the end of the summer period indicative of seasonal heat acclimatisation. Total sodium losses calculated based on sweat rate and sweat sodium concentration was therefore not significantly different between seasons in either the trained or untrained subject group. Based on this information, dietary sodium intake requirements are not necessarily lower in the summer period due to significant increases in sweating rate. For this reason focus on maintaining adequate fluid intake during summer should perhaps be the main focus for athletes whilst being aware of their high salt needs year round.

Results from this study also showed a significantly greater sweat potassium concentration from the leg sweat glands at the end of summer in the trained athletes. The loss and replacement of potassium in sweat has received little focus in the recent literature and this is the first study of our knowledge to report this finding. Estimated sweat potassium losses in the trained athletes in this study during summer averaged nearly 600mg/h. Endurance athletes, particularly in those with high sweat losses, should be educated on the need to maintain adequate dietary potassium intake.

Based on this information the following practical recommendations for fluid, sodium and potassium intake are made for endurance athletes with high sweat losses who are training and competing in the heat:

- A comprehensive hydration protocol should be established for each individual athlete which considers the duration and intensity of exercise, frequency of training sessions, heat acclimatisation status, environmental conditions as well as practical limitations associated with carrying fluid.
- Athletes should be advised to weigh themselves before and after training sessions of various duration and intensity to assess sweat losses. Athletes with losses greater than 1-2% body weight per session should be advised that their fluid intake is insufficient. Any athlete who has gained weight over a training session should be advised that they may need to consume less fluid to prevent over hydration.

- Athletes should be educated on the effect of heat acclimatisation on fluid and electrolyte losses and be particularly aware of the increased fluid needs in the summer period.
- During exercise of greater than 60-90 minutes duration the consumption of an electrolyte replacement beverage is indicated.
- For athletes with known high sweat losses or those with a history of heat cramping, the addition of salt with meals or consuming salty snacks is also indicated. The use of salt tablets is rarely warranted.
    - Adding salt to everyday foods is an inexpensive and effective method of increasing salt intake. Table 19. shows examples of high salt snack foods suitable for inclusion in the diet of athletes with high salt requirements
    - Athletes may be advised to consume an electrolyte beverage containing sodium at regular intervals throughout the day, particularly if training twice daily or for prolonged durations.
- Post exercise there is considerable evidence to support the replacement of both fluid and sodium losses to aid in the restoration of fluid balance.
- Educating athletes with known high sweat losses on their need for additional sodium while advising some health conscious athletes against choosing low salt options may be indicated.

Table 19. High salt snack examples suitable for inclusion in the diet of

athletes with high salt requirements

Food product	Sodium (mg/100g)	Sodium (mg/typical serve)
White bread	509	325
Deli sliced ham	1580	790
Rice crackers	575	143
Corn flakes	1081	270
Cheddar cheese	655	262
Baked beans	400	548
Flavoured tuna	438	810
Potato chips	640	320
Salted peanuts	340	340
Pretzels	1980	990
Pickled onions	790	632

Chapter 7: **References**

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Chapter 8: **Appendices**

8.1 **Appendix A: Published Paper**

1. Holmes NA, Miller VS, Schneider J, Hasan O, Bates GP.

   Plasma sodium levels and dietary sodium intake in manual workers in the
   Middle East.

   Annals of Occupational Hygiene 2011; 55: 397-402.

Plasma sodium levels and dietary sodium intake in manual workers in the Middle East

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Introduction

Manual labourers working in the building and construction industry in some parts of the world perform physically demanding tasks in the heat for 12 hours per day, 6 days per week. High sweat rates over the workshift can lead to progressive dehydration placing an individual at significant risk of heat related illness and injury (Donoghue and Bates 2000; Mirabelli et al. 2010). While several studies have investigated the incidence of dehydration in susceptible populations (Bates, Miller, and Joubert 2010b; Bates, Gazey, and Cena 1996), there has been little focus on the need to replace electrolyte losses associated with prolonged sweating in the occupational setting. The majority of research has focused on the electrolyte needs of endurance athletes due to their known high fluid and electrolyte losses (Baker et al. 2009a; Palmer, Logan, and Spriet 2010b; Maughan et al. 2005). The industrial population form a unique group, performing longer periods of work at a lower metabolic rate and repeating the process daily. Requirements for salt intake in this population need further consideration.

In developed countries such as the United States and Australia an estimated 75% of salt intake comes from processed foods (Dyer et al. 1997). Because of the high intake of these foods the dietary salt intake is seldom inadequate and often excessive. In countries where the traditional diet consists primarily of rice and legumes with very little processed food, the salt content is comparatively low. It was therefore hypothesised that workers consuming such a diet and working in hot environments would be at risk of chronic hyponatraemia due to inadequate replacement of sweat sodium losses. Expatriate manual workers in the Middle East form such a group, many thousands of these workers in the UAE are housed in labour camps, whereeach worker is served an identical type and quantity of food at each meal. The communal nature of food preparation and consumption leads to a uniformity of dietary intake not present in other populations. The consistency in dietary intake makes this population a unique group to study, while allowing for accurate dietary assessment.

Previous studies conducted in manual labourers in Australia have found average sweat rates of 1.1L/h, equating to over 10L of fluid loss during a work shift (Miller and Bates 2007a; Brake and Bates 2003b). Average sweat sodium losses over a 10 hour work shift have been estimated to be 4.8 – 6 g, equivalent to 10-15g salt (NaCl)

(Bates and Miller 2008). Due to the large inter-individual variation in sweat rate and sodium loss these values may be even higher in some individuals, with reported values in excess of 10g of sodium (25g salt) per day (Bates and Miller 2008). Regular consumption of food and fluid containing adequate salt content is therefore essential to replace these sweat losses to avoid the development of chronic hyponatremia, potentially compromising the health and safety of the worker.

While chronic hyponatremia is often throught to be asymptomatic the consequences of a long term decline in plasma sodium may put the health and safety of a worker at risk. The clinical symptoms of hyponatremia depend largely on the extent of plasma sodium decline and the rapidity of onset (Freda, Davidson, and Hall 2004) and can range from nausea, headache, vomiting, confusion, coma, convulsion and ultimately death (Kumar and Beryl 1998). Chronic hyponatremia can develop over several days when body mechanisms fail to maintain sodium homeostasis. This may be from profuse sweating with inadequate solute intake, resulting in a decrease in extracellular fluid volume, compromising the body's ability to thermoregulate (Hamilton, Dickson, and Smith 2006). Cases of hyponatremia have been reported in military and civilian personal working in extreme environmental conditions in southern Iraq (Hamilton, Dickson, and Smith 2006). While the majority of these cases were due to acute water intoxication leading to severe acute hyponatremia, the failure of sodium homeostasis in these extreme temperatures was reported to have been further compounded by poor dietary salt intake during the patrol period (Hamilton, Dickson, and Smith 2006).

The aim of the present study was to investigate the hypothesis that workers consuming a traditional low salt diet and working in hot conditions are at risk of chronic hyponatremia. Firstly plasma sodium levels in a group of manual labourers during both the summer and winter months were investigated for evidence of hyponatraemia. A further aim was to assess the relative adequacy of the current diet provided to workers with focus on the total salt content of the menu. The results of this study will identify whether this population consumes sufficient dietary sodium to offset the high sweat losses, particularly during the extreme summer months.

Methods

This study was carried out at a ship building and construction site in Dubai, United Arab Emirates, during summer (July) and winter (February).

All participants were volunteers who gave their written and informed consent to participate in the study. The study was supported and authorised by management and ethical approval was obtained from the Al-Ain Medical District Human Research Ethics Committee.

A total of 44 subjects were studied at the end of the summer months and 38 subjects were repeated at the end of winter. The same subjects were used in the summer and winter trials. The subjects were male dockyard workers (various trades) from India, Bangladesh and Pakistan, aged between 18 and 50 years.

Biochemistry

Full blood tests including LFT, FBC, U&E and glucose were taken from all subjects in both the summer (July) and winter (February) months to check general health and compare blood electrolyte profiles. Any worker with any known medical condition was excluded from the study. The venous blood sample was taken in the morning prior to eating (0800 hours) by a trained phlebotomist. An approved accredited pathology laboratory completed all analysis.

Dietary Assessment

An assessment of the diet of the workers was conducted in two parts by an Accredited Practising Dietitian. Part one involved recording and analysing menus and recipes to allow for an estimation of total daily salt intake. Menus and recipes from all food prepared and served in the mess hall was collected and analysed over a 3 day period. Foodworks 2007 nutrient analysis package was then used to estimate the total daily macronutrient intake along with total daily salt intake. Estimation of serve size was conducted through observation of meals served as well as measurement of plated food. Table salt was provided on a separate table next to the bain-marie. The use of table salt at meals was observed and an estimation of the quantity added was recorded. Part two of the study involved individual interviews with the workers, 38 workers were available for this interview where questions

relating to individual dietary practices were asked. Table 1 outlines the questions asked to the workers during the interview.

Results

Table 2 summarises the physical characteristics of the subject group.

Biochemistry

Results from the blood electrolyte profile in summer and winter showed that a high proportion of subjects (55%) were found to be hyponatraemic in the summer months (Graph 1), all remaining values were in the lower part of the reference range. 5% of subjects had serum sodium levels less than 130mM. In the winter months only 8% of subjects were found to be hyponatremic.

Dietary Assessment

Part One: Menu and Recipe Analysis

Table 3 summarises the dietary assessment data collected at the site during the summer period. On average a worker would consume an estimated 2000-3000mg of sodium (5-7.5g salt) in food and fluid per day. The total sodium content of the lunch meal was particularly low averaging 600mg (1.5g salt). In addition to this workers would be provided with a maximum of 1L per day of electrolyte replacement fluid which would provide between 200-400mg sodium (500-1000mg salt). This is only provided in the summer months. Additional table salt is available at all meals, however only 43% of workers indicated that they added salt to their meals.

Part Two: Interviews with workers

93% of the workers stated that they did not consume any other food outside of what was provided in the mess at breakfast, lunch and dinner. This information further confirmed the consistency in dietary intake of the workers. The three subjects who did snack between meals stated that they would only consume fruit or fruit juice on occasion; these foods would not contribute significantly to overall daily salt intake.

94% of the workers stated that they do not change anything about their diet between the summer and winter period and that heat did not affect their intake.

43% of the workers stated that they regularly add table salt to their lunch and dinner meals. One or two small pinches of salt was the typical amount added which would provide an estimated 250-500mg of sodium (625- 1250mg salt). Observation of

table salt use did support these self-reports as it was noted that few workers chose to add salt to their meals and if they did it was in small quantities.

All workers stated that they did consume an electrolyte replacement solution during working hours during the summer months. There is a limit of 1L of electrolyte solution per worker during this period while water consumption is unrestricted. Medical staff indicated that this restriction was placed as a precaution to avoid excessive consumption of sugar and salt.

Discussion

The initial hypothesis that sodium intake in this population is inadequate to replace sweat losses is supported by the results of this study. During the summer period 55% of workers were found to be clinically hyponatremic with plasma sodium values less than 135mM, compared with only 8% during the winter period. Given that dietary intake is consistent throughout the year, these results indicate that the hyponatremia during summer is most likely due to high sweat sodium losses coupled with inadequate sodium replacement. Assessment of dietary intake revealed that on average workers consume between 2000-3000 mg of sodium (5 – 7.5 g salt) per day. This quantity is insufficient, particularly during summer when temperatures are extreme and fluid and electrolyte losses through sweat are assumed to be high due to high sweat rates.

Due to the increasing prevalence of hypertension and cardiovascular disease on a global scale, dietary salt restriction is being strongly advocated as a public health measure to reduce the incidence of chronic disease worldwide. Currently the World Health Organisation recommends that salt intake should be less than 5g/day (less than 2g/day sodium) (World Health Organisation 2007). These recommendations are not appropriate for populations engaging in heavy manual labour in the heat as sweat sodium losses far exceed this level of intake.

The long term health effects of a chronic sodium restricted diet have not been well investigated. The results from animal studies have shown a significantly decreased plasma volume and increased haematocrit in sodium depleted mice (Francesconi, Hubbard, and Mager 1983). It is postulated that this change in hemodynamic state would seriously compromise the body's ability to tolerate further circulatory stress such as that imposed by excessive sweating. In addition to this, a higher sodium diet is known to enhance the thermoregulatory and cardiovascular adaptations that occur during heat acclimatisation (Luetkemeier, Coles, and Askew 1997). A recent study by Miyazaki et al (2010) showed that stable chronic hyponatremia resulted in impaired memory function in rats that was normalised when sodium levels were corrected to within normal range. The effect of chronic hyponatremia on the Central Nervous System in humans is largely unknown and requires further investigation. A study by Renneboog et al (2006) indicated that patients with mild chronic

hyponatremia had an increased incidence of falls due to a global decrease in attentional capabilities, posture and gait mechanisms (Renneboog et al. 2006). While the patients in this study were reported to be asymptomatic, the results also identified significant impairment in cognitive function. The mechanisms of these observations are throught to be a result of slowed peripheral and central nerve conduction (Renneboog et al. 2006). For manual workers performing intense physical labour, any impairment in judgment or cognitive capacity will increase vulnerability to workplace accidents and injury and is therefore a major concern.

Since conducting this research there have been reports of a number of young laborers presenting to the Intensive Care Unit in hospitals within the Middle East with seriously low plasma sodium levels, placing them at risk of neuromuscular dysfunction. This information and the results from this study indicate that there is a need for sodium intake guidelines specific for manual laborers working in the heat to be developed. Providing workers with specific guidelines showing estimated sweat sodium losses and practical replacement guidelines for the various working roles will acknowledge that workers in physically demanding outdoor roles will have greater fluid and salt requirements than stationary indoor workers. Interviews with the workers in this study revealed that several individuals had been actively trying to limit salt intake to prevent hypertension. Given that current health recommendations are aimed at restricting dietary salt intake, educating medical staff and workers on the importance of both fluid and sodium replacement when working in conditions of heat stress is necessary.

In order to increase sodium intake the addition of salt to meals during cooking is indicated. In addition to this, educating workers, particularly those in more physically demanding roles on the benefit of adding table salt to meals is also warranted. When working in severe thermal conditions, replacement of fluid losses with water alone is insufficient, as it does not replace sodium lost in sweat, thus increasing the risk of dilutional hyponatremia and acute water intoxication (Hoorn and Zietse 2008) The provision of an electrolyte replacement fluid that has been designed for prolonged use in an industrial setting is indicated. The benefit of providing an electrolyte replacement beverage between meals is to promote water uptake and retention, and to offset fatigue by maintaining blood glucose, whilst palatability encourages fluid

intake (Sawka et al. 2007). However even regular consumption of an electrolyte replacement fluid throughout the work shift would not make up for the dietary inadequacy; the addition of salt to meals is also needed.

The data from this study demonstrate that the diet of some workers in the Middle East is not adequate in salt content. High sweat sodium losses due to long periods of manual work in the heat are not being replaced leading to the chronic hyponatremia shown. While workers are relatively asymptomatic there may be safety implications for workers who are managing heavy machinery or working at height due to an impaired cognitive ability and increased susceptibility to fatigue, both of which may increase the risk of workplace accidents. The information collected in this study may apply to hundreds of thousands of workers in the Middle East as well as anywhere where workers are engaged in prolonged manual labour in hot climates while consuming a diet that is predominantly starch based. Increasing the total salt content of both fluid and food consumed by workers may be effective in reducing the incidence of work related illness and accidents in this population.

Conclusions

- A significant proportion of workers at this site in the UAE were clinically hyponatremic during the summer period. This was not shown during winter indicating that it is primarily due to high sweat sodium losses that are not being replaced.

- The salt content of the diet is insufficient to replace estimated sweat sodium losses during summer

- Workers are unable to consume food at regular intervals due to logistics of the work environment and will benefit from regular consumption of an electrolyte replacement fluid specifically designed for prolonged industrial use.

- Educating workers and medical staff on the importance of adequate fluid and salt intake to prevent dehydration and electrolyte imbalances is strongly indicated.

- Salt intake guidelines specific for workers engaged in prolonged work in the heat are necessary to decrease the incidence of hyponatremia in this population

TABLES AND FIGURES

Table 1: Interview questions asked to workers

Question
5. Do you normally snack outside of meal times (i.e. Breakfast/lunch/dinner)?
6. Do your eating habits change in summer compared with winter?
7. Do you normally add salt to your lunch and dinner meals?
8. Do you consume an electrolyte replacement drink during working hours?

Table 2:Physical Characteristics of subjects

	Mean (± Std Dev)
Age (years)	37 (6.1)
Height (m)	168 (6.9)
Weight (kg)	67.5 (12.5)
BMI (kg/m^2)	23.8 (3.7)

Graph 1: Comparison of serum sodium values in summer and winter
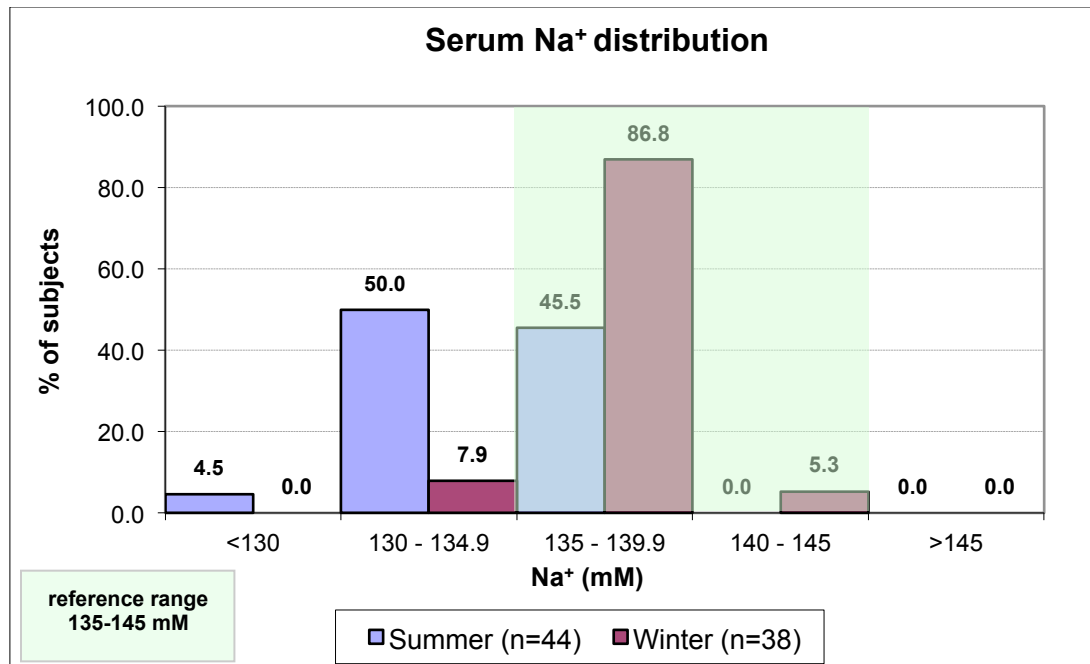
Table 3: Analysis of energy and sodium content in the 3 day menu

Day	Meal	Energy (kJ)	Sodium (mg)
1	Breakfast	4411	1359
	Lunch	3937	624
	Dinner	3415	773
	Total	**11763**	**2757**
2	Breakfast	4624	1235
	Lunch	4105	464
	Dinner	3651	1005
	Total	**12381**	**2704**
3	Breakfast	3903	1145
	Lunch	4263	654
	Dinner	3428	571
	Total	**11595**	**2371**
Average	3 Day average	**11913**	**2610**

8.2 Appendix B: Sweat rates and sodium loss across a range of sports

Reference	Sport	Conditions	Sweat collection method	Testing	Sweat rate (L/h)	Sodium concentration (mmol/L)	Potassium concentration (mmol/L)
Bates and Miller (2008)	Work in the heat	35 degrees 50%RH chamber study	Wescor coils (Right and left upper arm and thigh)	29 manual workers, untrained, 2 consecutive days testing in summer and winter	Summer = 0.47L/h Winter = 0.41L/h	Summer = 53mmol/L Winter = 73mmol/L	N/A
Neville et al (2009)	Competitive sailing	32 degrees 52%RH field study in summer	Sweat patches (chest, scapular, forearm, thigh)	32 elite males 100 minutes of racing	1.4L/h (0.44-2.1L/h)	27mmol/L (12-43.5mmol/L)	N/A

Baker et al (2009b)	Triathletes	30degrees, 44% RH Heat chamber study	Sweat patches (Forearm, back, chest, forehead and thigh) and whole body wash down	10m/10f athletes cycled in heat chamber for 90 minutes	0.8L/h (0.6-1.2L/h)	41 mmol/L (21-127mmol/L)	4.4mmol/L (3.1 – 5.8mmol/L)
Maughan et al (2005)	Professional football players	5 degrees 88% RH Field study	Sweat patches (chest, forearm, back and thigh)	17 males, 1h 40min training session	1.69 ± 0.45L/h (1.06-2.65L/h)	73 ± 31mmol/L (29-121mmol/L)	7.1 ± 2.8mmol/L (3.4-14.8mmol/L)
Shirreffs et al (2005)	Football players	32 degrees, 20%RH Field study	Sweat patches (chest, arm, back and thigh)	26 males, 90 mins training session	2.2 ± 0.4L/h (1.67-3.14L/h)	67 ± 37mmol/L (26-129mmol/L)	8 ± 2.0mmol/L (5-12mmol/L)

Godek et al (2010)	Professional football players	25.9ºC Field study	Sweat patches (right forearm)	18 backs and receivers (BK) 12 linebacks (LB), 14linemen (LM), 2 hour practice	BK = 1.41L/h LB = 1.98L/h LM = 2.16L/h	50 ± 16 mmol/L (15-99 mmol/L)	N/A
Maughan et al (2009)	Swimmers	36ºC – air temp 27ºC – pool temp Field study	Sweat patches (forearm, back, chest and thigh)	9 male 8 female, 105 min training session	0.31 ± 0.1L/h	43 ± 14mmol/L	4.0 ± 1.0mmol/L
Horswill et al (2009)	Professional American Football players	29-32ºC Field study	Sweat patches (forearm only)	14 males, 2.2h training session	Crampers: 2.9 ± 1.6L/h Non-crampers: 2.3 ± 0.4L/h	Crampers: 52.6mmol/L Non-crampers: 38.3mmol/L	N/A

Kilding et al (2009)	internation al female soccer players	14.1 ± 0.7ºC	Sweat patches	13 players 2 x 90 min soccer specific training sessions. Sweat patches used	0.5 ± 0.27L/h	46 ± 14mmol/L	6.1± 3.9mmol/L
Pahnke et al (2010)	Ironman triathletes	Hawaii Ironman® 2003 26.4℃, 65% RH	Sweat patches (right forearm and right scapula)	46 male athletes	Females: 1.1 ± 0.38L/h Males: 1.53 ± 0.36 L/h	Females: 39.95 ± 12.6 mmol/L Males: 44.97 ± 16.4 mmol/L	N/A
Palmer et al (2010a)	Club level Ice hockey players	11.4℃, 52% RH	Sweat patch (forehead)	14 males, 90 minute training session	1.5L/h (0.7-2.1L/h)	71.6 mmol/L	N/A

Kurdak et al (2010)	Club level football players	34℃, 65% RH	Sweat patches (forearm, chest, back and thigh)	22 males, 90 min game	2.0L/h	43mmol/L± 11 (27-59)	3.5mmol/L ± 0.5 (2.9-4.0)
Hamouti et al (2010)	Elite team players (volleyball, basketball, handball and soccer)	21℃, 32% RH	Sweat patch (forearm)	43 males, 70 min practice game	1.4 ± 0.3L/h	49 ± 10mmol/L	4.3 ± 0.3mmol/L
Bergeron (2003)	Tennis players (cramp prone)	31.9 ± 0.5ºC	Sweat patches	Game (duration N/A)	2.6 ± 0.1L/h	44.5 ± 13.5mmol/L	N/A

8.3 Appendix C: Consent Forms

PROJECT TITLE

"Fluid and electrolyte losses associated with working/exercising for prolonged periods in thermally stressful environments"

NAME OF CHIEF INVESTIGATORS

Miss Nicola Holmes, Dr Jill Sherriff, Dr Veronica Miller, Dr Graham Bates

Thank you for expressing interest in our study.

The work that we are doing will increase our knowledge of the risks associated with working or exercising in hot environments, and enable us to make recommendations which will help to protect your health and safety in hot conditions.

The study involves:

- Answering a few questions about your health and habits, this gives us background information to assist in interpreting your data.

- Recording of your fluid intake during the shift and answering questions about your fluid intake when off duty.

- Having your blood collected in the morning of your shift after an overnight fast. This will be performed by a trained phlebotomist.

- Having your diet assessed by a trained dietitian who will analyse the nutritional content of the food you consumed throughout your shift

POSSIBLE RISKS, INCONVENIENCE AND DISCOMFORTS

There are no substantial risks with this study

I have been asked to participate in the above research study and give my consent by signing this form on the understanding that:

1) The research study will be carried out in a manner conforming to the principles set out by the National Health and Medical Research Council.

2) I comprehend the general purposes, methods, demands and possible risks, inconvenience or discomforts of the study

3) In giving my consent I acknowledge that my participation in this research study is voluntary and that I may withdraw at any time and if I do withdraw there will be no adverse consequences.

4) I agree that research data gathered for the study may be published provided my name is not used and if I request I may have a copy of my own results.

5) Any questions concerning the project can be directed to Nicola Holmes contact number 0414 765 906. Email nicola.holmes@postgrad.curtin.edu.au

6) This study has been approved by the Curtin University Human Research Ethics Committee. If needed, verification of approval can be obtained either by writing to the Curtin University Human Research Ethics Committee, c/- Office of Research and Development, Curtin University of Technology, GPO Box U1987, Perth, 6845 or by telephoning 9266 2784 or by emailing hec@curtin.edu.au.

Participant _____   Date _____

Investigator_____
        Date_____

FORM OF DISCLOSURE AND INFORMED CONSENT: STUDY #2/3

PROJECT TITLE

"Fluid and electrolyte losses associated with working/exercising for prolonged periods in thermally stressful environments"

NAME OF CHIEF INVESTIGATORS

Miss Nicola Holmes, Dr Jill Sherriff, Dr Veronica Miller, Dr Graham Bates

Thank you for expressing interest in our study.

The work that we are doing will increase our knowledge in the effects of sweat rate on sweat sodium loss. This information will enable us to make recommendations which will help to ensure that your hydration level is best maintained during exercise.

The study involves:

- Answering a few questions about your health and training habits. This gives us background information to assist in interpreting your data

- Measurement of your hydration status at the start of the exercise trial. This test simply requires a small urine sample which will be discarded after testing

- Determination of your sweat rate by weighing you at the start and end of the shift and recording all inputs and outputs over the shift

- Exercising on a stationary bicycle at a set workload for a period of approximately 30 minutes. 15 minutes of this will be done as a warm- up at a low intensity level. The next 15 minutes will consist of exercise at low- high intensity The conditions in the chamber will be hot and humid.

- Having samples of your sweat collected in small collection coils secured by Velcro straps to shaved areas of skin.

POSSIBLE RISKS, INCONVENIENCE AND DISCOMFORTS

There are no substantial risks with this study

I have been asked to participate in the above research study and give my consent by signing this form on the understanding that:

1) The research study will be carried out in a manner conforming to the principles set out by the National Health and Medical Research Council.

2) I comprehend the general purposes, methods, demands and possible risks, inconvenience or discomforts of the study

3) In giving my consent I acknowledge that my participation in this research study is voluntary and that I may withdraw at any time and if I do withdraw there will be no adverse consequences.

4) I agree that research data gathered for the study may be published provided my name is not used and if I request I may have a copy of my own results.

5) Any questions concerning the project can be directed to Nicola Holmes contact number 0414 765 906. Email nicola.holmes@postgrad.curtin.edu.au

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Participant _____   Date _____

Investigator_____
        Date_____

8.4 Appendix D: Information to volunteers

Dear athletes,

Thank you for your interest and for volunteering your time to be part of this research.

Please read through this information prior to your designated testing day. If you have any further questions please don't hesitate to contact me at nicola.holmes@postgrad.curtin.edu.au

Information to read prior to your testing days

Hydration

It is vitally important that you are adequately hydrated prior to your sessions. We will be measuring your hydration level before you commence cycling (be ready to give a urine sample on arrival!)

Hydration Protocol – 24 hours prior to test day

- Drink at least 1.5L of water or electrolyte replacement fluid in the 24 hours prior to your test day.
    - o A good way to tell whether you are well hydrated is by your urine colour. When you are adequately hydrated your urine will be a straw yellow colour.
- Avoid alcohol
- Consume at least 500ml of fluid and avoid all caffeinated beverages on the morning of the test

Where to go on the day

All testing will be conducted in the heat chamber at Curtin University located in the Public Health building (building 400). Go to the link below to see a detailed map of Curtin

http://properties.curtin.edu.au/maps/

Building 400 is close to the hockey stadium. As you will be coming to Curtin outside of normal working hours you are able to park in any parking bay.

Please meet outside the glass sliding doors at the entrance of the building, someone will meet you and direct you to the heat chamber..

What to bring

- o Normal cycling clothing (light weight clothing is best as it doesn't absorb as much sweat)
- o bike shoes or sneakers
- o Heart rate monitor if you own one
- o Towel
- o Water bottle

There are stationary bikes set up in the chamber which we will be using for the testing.

There are shower facilities that you are able to use after your session.

If you cannot find the building on the morning of testing my contact number is 0414765906, please call me and I will point you in the right direction.

I look forward to meeting you.

Kind Regards

Nicola

This study has been approved by the Curtin University Human Research Ethics Committee. If needed, verification of approval can be obtained either by writing to the Curtin University Human Research Ethics Committee, c/- Office of Research and Development, Curtin University of Technology, GPO Box U1987, Perth, 6845 or by telephoning 08 9266 2784.

8.5 Appendix E: Pre-testing Questionnaires: Study #2/3

Heat Chamber study #1: The effect of exercise intensity on sweat rate and sweat sodium loss in well trained athletes during cycling exercise in the heat

Name_____   **Age**_____

Contact Number:

(Home)_____**(Mobile)**_____

Email: _____

Anthopometry measurements

Height: _____ Weight: _____

Estimated Maximal Heart rate (BPM) _____

1) Are you currently training for any particular event?

_____

_____

_____

2) Do you ever suffer from cramps during training or competition?

_____

_____

_____

3) Do you currently use an electrolyte replacement fluid during training/competition?
If yes what type?

_____

_____

_____

_____

_____

Please estimate your current approximate training hours per week.

      a) Swimming _____ hs

      b) Cycling _____ hs

      c) Running _____ hs

      d) Other _____hs

Heat Chamber study #2: The effects of physical training and seasonal acclimatisation on fluid and electrolyte losses during cycling exercise in the heat

Name_____   **Age**_____

Subject ID Number _____

Contact Number: _____

Email: _____

Anthopometry measurements

Height: _____

Pre weight:_____

Post Weight:_____

Sweat rate:_____

Estimated Maximal Heart rate (BPM) _____

Skinfolds

Bicep_____

Tricep_____

Subscapular_____

Abdominal_____

Supra spinale_____

Iliac Crest _____

Mid Thigh_____

Calf_____

% Body Fat _____

Please outline below the amount/type/intensity of physical activity you have been doing over the last 2 months on a weekly basis

_____

_____

_____

8.6 Appendix F: Dietary intake questionnaires: Study #1

Plasma sodium levels and dietary sodium intake in manual workers in the Middle East

Department _____   **Date**
_____
Nationality_____

1. Where do you normally eat your meals:

    Home ☐   Asian Mess ☐

    Filipino Mess ☐   Guest House ☐

    Other ☐

2. Do you normally snack outside of meal times:

    No ☐

    Yes ☐

    If 'Yes' what types of snacks do you normally eat?
    _____

3. Do your eating habits change in the summer period Vs winter period?

    No ☐

    Yes ☐

    If 'yes' how do your eating habits change?
    _____

4. <u>Do you normally add salt to your food?</u>

No ☐

Yes ☐

If 'yes' approximately how much salt do you add? (i.e. 1 teaspoon per meal)

5. <u>What do you normally drink during a work shift?</u>

Water ☐

Electrolyte solution ☐

Other ☐

Nothing ☐