The physiological effects of ingesting high sodium drinks before, during, and after exercise in the heat

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to the University of Exeter

as a thesis for the degree of Doctor of Philosophy in

Sport and Health Sciences,

May, 2011

I certify that all material in this thesis which is not my own work has be identified and that no material has previously been submitted and approved for award of a degree by this or any other University.

Abstract

This thesis investigated whether highly concentrated sodium solutions ([HS] 126-164 mmol·L^{-1} NaCl) could provide viable strategies before during and after exercise in the heat to improve cardiovascular and thermoregulatory functioning and exercise performance. To do this it also examined the gustatory responses to HS drinks before, during and after exercise. All studies compared HS with a low sodium control ([LS] 10-27 mmol·L^{-1}).

Chapter 4 found that during 3 h recovery from dehydration, ingestion of 120 % body mass losses of HS restored fluid balance to a greater extent (121 vs. 84 %) than LS. Chapter 7 was the first to investigate the effects of ingesting HS during exercise in the heat and in an untrained population. HS attenuated the decline in stroke volume [SV_{Drift}] and increase in heart rate [HR_{Drift}], but did not affect rectal temperature [T_{Rec}], cardiac output, or oxygen uptake during the second of two consecutive 45 min bouts at 55% $\dot{V}O_{2max}$. In Chapters 8 and 9 untrained participants ingested either HS or LS during 30-45 min pre-exercise rest. HS reduced HR_{Drift} and SV_{Drift} but did not affect T_{Rec} during 45-60 min exercise at 10% of the difference between $\dot{V}O_{2max}$ and gas exchange threshold [Δ]. HS also increased both time to exhaustion and exercise tolerance during subsequent exercise bouts at 60-70% Δ.

Chapters 5 and 6 found that taste perceptions act as physiological regulators, in this case, one reflecting the priority to restore hyperosmolality over hypovolemia. Exercise-induced dehydration increased the palatability of water, and decreased the palatability HS, when measured before, immediately after and during 3 h recovery. The changes were highly correlated with physiological indicators of fluid balance.

The ingestion of highly concentrated sodium solutions can be both an efficient and acceptable means to improve hydration, reduce cardiovascular stress, and improve exercise performance in the heat. Whilst highly effective, caution should apply since the unpleasant taste evoked by these solutions persists for at least three hours post exercise.

Acknowledgements

First and foremost I would like to thank my primary Ph.D. Supervisor, Associate Professor Craig Williams. He has been extremely patient and has never wavered in providing endless support, advice and encouragement throughout my studies. I would also like to thank my secondary supervisor Dr. Christopher Byrne who was instrumental during statistical analysis and study design.

I would like to thank the University, and in particular the School of Sport and Health Sciences for their patience and their financial support towards tuition fees. I wish to thank two Laboratory technicians, whose help was essential during the testing process. Jamie Blackwell for coaching me through my phlebotomy training, and sharing his extensive knowledge of analysis techniques, both of which enabled me to complete the measurements undertaken in this thesis. David Childs, for always being available and willing to assist with the technical malfunctions I encountered during my testing.

Lastly, I also wish to express my sincerest thanks to my parents for their emotional and financial support and to my girlfriend Hannah for her enduring encouragement.

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Abbreviations

Generic

1) min = minute(s)
2) h = hour(s)
3) d = day(s)
4) yr = years
5) O_2 = oxygen

Fluid Balance

6) BM = body mass
7) U_{Osm} = Urine osmolality
8) U_{Vol} = Volume of urine excreted
9) P_{Na} = Plasma sodium concentration
10) P_{Osm} = Plasma osmolality
11) P_{Vol} = Plasma volume
12) ECF = Extracellular fluid compartment
13) ICF = intracellular fluid compartment
14) TBW =Total body water
15) Hb = Haemoglobin
16) Hct = Haematocrit
17) AVP = arginine vasopressin

Cardiovascular

18) CV_{Drift} = Cardiovascular drift
19) HR = Heart rate
20) HR_{max} = Maximum heart rate
21) HR_{Drift} = Heart rate drift
22) SV = Stroke volume
23) SV_{Drift} = Stroke Volume drift
24) $\dot{Q}$ = Cardiac output
25) $\dot{Q}_{Drift}$ = Cardiac output drift
26) $\dot{V}O_2$ = oxygen uptake
27) $\dot{V}O_{2max}$ = maximum oxygen uptake
28) GET = Gas exchange threshold
29) EDV = end diastolic volume
30) BF_{Mus} = muscle blood flow
31) BF_{Skn} = skin blood flow
32) T_{Core} = core temperature
33) T_{Skn} = skin temperature
34) T_{Rec} = rectal temperature
35) T_{Esp} = oesophageal temperature
36) CBV = central blood volume

Fluid replacement

37) SA = Sodium Appetite
38) NaCl = Sodium chloride
39) Na = Sodium
40) CH-E = carbohydrate electrolyte drink

Miscellaneous

41) RH = relative humidity
42) EID = Exercise induced dehydration
43) TTE = Time to exhaustion
44) TT = Time trial
45) Δ = Difference between GET and $\dot{V}O_{2max}$
46) VAS = visual analogue scale
47) MVCs = multiple voluntay contractions

Chapter 1. Introduction

This thesis aims to address the question of whether the inclusion of high concentrations of sodium within sports drinks is a viable ergogenic option, capable of replacing current strategies already in common use, before, during and after exercise in the heat. In order to make this assessment it tackles both the ergogenic benefit of high sodium drinks and the negative gustatory response to these solutions. The former, being the obvious evaluation of efficacy within a sports science environment, may in some people's eyes be enough on its own. This may be the case with elite athletes willing to undergo physiological discomfort in order to achieve their goals. However, participation with events, such as marathon or triathlon amongst recreational standard athletes has increased substantially over the last few years. As has the uptake of scientifically designed aids such as sports drinks and supplements. Thus there is a need to assess whether these aids will be acceptable to mass participation athletes, whose interpretation of acceptability differs from elite athletes. If a drink is not acceptable in taste, and will be avoided, it does not serve as an acceptable sports drink for general use.

Many years of research within the field of Sport Science has led to the discovery of numerousergogenic aids capable of improving hydration, cardiovascular and thermoregulatory functioning, and exercise performance, and whilst these may stand up to the rigours of scientific scrutiny, many fall short in offering feasible strategies in both athletic and occupational scenarios. Some are neither authorised by governing bodies, nor practical for use at athletic events or in the workplace. It will be a particular aim of this thesis to propose *practical* ergogenic aids to situations involving the whole spectrum of sporting events, before, during and after exercise. Several

In order to achieve this, the thesis must therefore consider the gustatory responses to high sodium drinks. A drink cannot be entirely practical if it is not *acceptable* to the athlete. If we, as scientists, are to encourage athletes to drink, then we must supply a drink which is acceptable. What is considered acceptable, at least to a recreational athlete or industrial worker, is most likely to be what is most palatable. Drinks containing high concentrations of sodium tend not to be highly palatable, yet as will be explored in this thesis, the palatability of a drink is closely related to the physiological needs of the individual; needs which will be constantly changing

throughout the course of athletic performance. Changes in the perception of salt, or rather of salty drinks, must be taken into consideration when evaluating the efficacy of high sodium drinks.

1.1. Ergogenic benefit

Exercise in the heat provides one of the greatest physiological stresses an athlete can experience. The combination of dehydration and hyperthermia pose significant challenges to the cardiovascular system which can impact on the ability to perform at the highest level. In order to attempt to design strategies to improve exercise performance in the heat, it is important to understand the physiological responses to such exercise and the factors which contribute to the deterioration in performance. These factors will then act as a barometer for the effectiveness of each intervention.

These issues, which may happen both independently, or in conjunction with each other, can occur at many of the major global tournaments which take place in hot environments. The issue of how athletes will cope in Qatar during the forthcoming 2022 Football World Cup, where ambient temperatures may reach 45-50^{o}C, have and will become well published issues, as it was when the 1984 Los Angeles Olympic Marathon took place during the hottest part of the day. Rarely have the fastest times for distance events ranging from 5000 m to 100 km road race been set set in cities in which the average temperature exceeds ~14^{o}C (Marino, 2004).

Whilst the needs of elite athletes are met by experienced physiologists and coaches, exercise in the heat may have a greater detrimental effect for recreational athletes, whose preparations are not so well-informed. Participation in endurance events such as marathons, triathlons and long distance cycling events has grown markedly since the 1990's. The USA Triathlon organisation estimates that between 550,000 and 746,000 people participate in triathlons per year, and the estimated number of marathon participants grew from 260,000 to 435,000 between 1990 and 1999 alone. Most of the events, triathlon especially, take place in the height of summer with temperatures most often between 30-32^{o}C and humidity above 70 %. There is a third population, often disregarded, within the field of Health and Sport Sciences for which dehydration and hyperthermia are also an important consideration - those involved in occupational settings which are exposed to the heat (Brake & Bates 2003). Many industrial

buildings, factories, warehouses, workshops, etc. suffer from poor ventilation and can therefore exposure workers to high temperatures, especially those that require workers to wear heavy protective clothing. For example, wildfire fire-fighters are often required to work for fourteen consecutive days consisting of 12-16 h shifts at temperatures >40^{o}C (Cuddy *et al.,* 2007). Such physiological efforts required mirror those of ultra-endurance marathon (Cuddy *et al.,* 2008).

1.1.1. Exercise in the heat

When exercising total body metabolism increases by 5-15 times the resting rate to support the contraction of skeletal muscles. Humans are relatively inefficient at converting stored energy into kinetic energy, with approximately 70-90 % released as heat (Sawka *et al.,* 1996a; Taylor, 2006). This equates to a rise in the rate of metabolic heat production from approximately 1 $W \cdot kg^{-1}$ at rest to 10 $W \cdot kg^{-1}$ during heavy exercise (Donaldson *et al.,* 2003) and to as much as 16 $W \cdot kg^{-1}$ in elite athletes exercising at a high intensity in the heat (Maughan, 1998).

This alone is capable of raising core body temperature [T_{Core}] substantially, but when combined with exogenous heat from the external environment, exercise performance is almost invariably impaired and at worst, the heat imposes a serious threat to the athlete's health. There have been many cases of athletes suffering ill health when exercising in an environment with a high ambient temperature. The most notable examples include Jim Peters at the Vancouver marathon in 1954, and Gabrielle Andersen-Schiess at the 1984 Los Angeles Olympic marathon, however, athletes in shorter running events and in sports such as rugby, soccer, and American football have also been subject to heat related illnesses during competition.

In order to prevent against rising T_{Core} heat is dissipated from the body to the external environment (Folk *et al.,* 1998). Whilst humans are inefficient at converting stored energy into kinetic energy, the body has evolved to be particularly proficient at dissipating that heat (Donaldson *et al.,* 2003). In response to elevated T_{Core} the body initiates a series of compensatory responses to increase heat transfer from the body. To augment evaporative heat loss, the body redistributes blood to the skin by dilation of superficial veins. The increase in skin blood flow [BF_{Skn}], lengthens the time blood spends in vessels near the surface of the body, thereby increasing the time for heat

transfer (Fortney *et al.*, 1983). BF_{Skn} can increase from 0.2-0.5 $L \cdot min^{-1}$ in thermo-neutral conditions to 7-8 $L \cdot min^{-1}$ under maximal tolerable conditions (Donaldson *et al.*, 2003) and concomitantly sweat rates can exceed > 3 $L \cdot h^{-1}$ during intense exercise in the heat (Costill, 1977; Saltmarsh, 2001; Rehrer, 2001).

1.1.2. Dehydration

Whilst the increase in sweating helps to dissipate heat and reduce T_{Core}, the consequent loss of body fluid may cause dehydration. That is unless fluid losses are compensated for by an increase in fluid intake. However, despite several decades' worth of guidance being available, athletes frequently fail to rehydrate sufficiently during exercise (Hubbard *et al.*, 1984; Saltmarsh, 2001; Sawka *et al.*, 2007). Rates of fluid intake rarely match rates of sweat loss and although individual drinking practices vary markedly, many athletes finish a bout of exercise in a dehydrated state (Noakes & Martin, 2002).

There are three main types of dehydration; hypotonic, hypertonic and isotonic. Hypotonic dehydration is less common and results when a solute loss is greater than fluid loss, such as with cystic fibrosis. It can also occur in situations in which one drinks too much water, particularly following a large loss of electrolytes and this results in dilutional hyponatraemia, a complication which can be life threatening, and is becoming a more frequent occurrence amongst recreational athletes (Almond *et al.*, 2005), who may lack the appropriate knowledge of recovery techniques. Isotonic dehydration occurs when the loss of fluid and electrolytes are equal to that of plasma. It can be induced with prolonged or severe diarrhoea and vomiting, blood withdrawal or via the use diuretics.

Hyperosmotic dehydration often referred to as hyperosmotic hypovolemia, results from excessive sweat loss and as such is the most common form of dehydration which results from exercise (Harrison *et al.*, 1981; Convertino *et al.*, 1980; Brandenberger *et al.*, 1986; Jimenez *et al.*, 1999). Since sweat is hypotonic to plasma, i.e. contains less solutes per unit of solution, sweating results in a greater relative loss of fluid than electrolytes, causing P_{Osm} to rise (hyperosmolality). Proportionate with the decline in total body water [TBW], during exercise is a decrease in P_{Vol}. The fall in P_{Vol} or *hypovolemia* occurs because the fluid lost in sweat is primarily derived from extracellular fluid (Sawka *et al.*, 2001). In fact fluid lost from plasma can be as much as

five times as great as losses from other fluid compartments (Senay, 1979). During 2 h of moderate exercise in the heat (30^{o}C) a loss of 3-5 % of BM reduced P_{Vol} by ~3-5 % (Montain *et al.,* 1992b; Mora-Rodriguez *et al.,* 1996). Thus sweating causes a loss of P_{Vol} which is proportionate to the hypohydration level (Sawka *et al.,* 1996b). A reduction in P_{Vol} is a critical factor in the impairment of cardiovascular functioning and thermoregulation during exercise, associated with a reduction in central blood volume [CBV].

1.1.3. Dehydration and development of hyperthermia

When exercise is conducted in a warm environment, the development of dehydration makes hyperthermia a distinct possibility. A deficit of only 1 % of body mass [BM] elevates T_{Core} during exercise (Ekblom *et al.,* 1970). As the magnitude of water deficit increases, there is a concomitant elevation of T_{Core} when exercising in the heat (Montain & Coyle, 1992a). The T_{Core} elevation in response to dehydration is particularly heightened when exercise is performed at higher ambient temperatures (Coyle & Montain, 1993).

Both the hypovolemic and hyperosmolality aspects of sweat induced dehydration, contribute to the impairment of thermoregulation during exercise. A loss of plasma volume causes a reduction in CBV which increases the T_{Core} threshold at which skin blood flow [BF_{Skn}] is elevated above resting levels (Fortney *et al.,* 1981a; Nadel *et al.,* 1979; Buskirk *et al.,*1958; Ekblom *et al.,* 1970; Greenleaf & Castle, 1971; Lee *et al.,* 1941). Thus a greater rise in T_{Core} occurs before BF_{Skn} and the sweating response is induced, hindering the body's capacity for heat loss. The capacity for blood flow is also impaired by hyperosmolality, which accompanies hypovolemia during exercise-induced dehydration. Hyperosmolality initiates a series of mechanisms designed with conserving fluid loss, a principal avenue of which is sweat loss. Hyperosmolality delays the onset of heat dissipating mechanisms by increasing the threshold at which cutaneous vasodilation occurs, reducing sweat loss and with it the capacity for heat loss (Harrisson *et al.,* 1978; Neilsen *et al.,* 1971; Senay, 1968; Senay, 1979; Fortney *et al.,* 1984; Nielsen *et al.,* 1973; 1974; Takamata *et al.* 1997; 2001).

Hyperthmia alone has been found to impair performance during during prolonged exercise (>60 mins) at intensities ranging from 40 to 80 % $\dot{V}O_{2max}$ (Kay *et al.,* 2001; Nybo *et al.,* 2001b; Tucker *et al.,* 2004; Tucker *et al.,* 2006; Watson *et al.,*

2005), during short-term (<10 min) maximal exercise (Arngrimsson *et al.*, 2003; Saltin *et al.*, 1972; Nybo & Nielsen, 2001a; Gonzales-Alonso & Calbert, 2003), during isometric contractions (Martin *et al.*, 2005; Mortensen *et al.*, 2005; Nybo & Nielsen, 2001a; Thomas *et al.*, 2006) and during repeated sprinting (Drust *et al.*, 2005). Either individually, or coupled with dehydration hyperthermia has a significant negative effect on exercise performance. Prolonged exercise with dehydration and/or hyperthermia is frequently associated with declines in cardiovascular performance, characterised by declines in venous return, stroke volume [SV], cardiac output [$\dot{Q}$] and mean arterial pressure [MAP] and increases in heart rate [HR] and oxygen uptake [$\dot{V}O_2$] (Costill, 1977; Fortney *et al.*, 1981a; Ekelund & Holmgren, 1964; Gonzalez-Alonso *et al.*, 1995; 1999a; Grande *et al.*, 1958; Hopper *et al.*, 1988; Ladell, 1955; Kanstrup & Ekblom, 1982; Krip *et al.*, 1997; Mier *et al.*, 1996; Nadel *et al.*, 1980b; Nose *et al.*, 1994; Nybo *et al.*, 2008; Pearcy *et al.*, 1956; Rowell *et al.*, 1966; Rowell, 1974; Saltin & Stenberg, 1964; Warbuton *et al.*, 1999; Wingo *et al.*, 2005a). Dehydration also significantly impairs thermoregulation. A reduction in CBV is associated with impaired thermoregulation via an increase in the T_{Core} threshold at which BF_{Skn} is elevated above resting levels (Fortney *et al.*, 1981a; Nadel *et al.*, 1979) and a reduction in sweat rates (Buskirk *et al.*, 1958; Ekblom *et al.*, 1970; Greenleaf & Castle, 1971; Lee *et al.*, 1941). Thus a greater rise in T_{Core} occurs before BF_{Skn} and the sweating response are induced. Additionally, along with hypovolemia, hyperosmolality inhibits thermoregulatory responses to heat stress, by encouraging cutaneous vasoconstriction; therefore reducing sweat loss (Takamata *et al.*, 1998).

Athletic competitions performed in the heat, such as the Summer Olympic Games, normally take place in environments favouring evaporative cooling where T_{Skn} remain around 31-32^{o}C, instead of 37-38^{o}C during severe heat stress (Gonzales-Alonso *et al.*, 2008). In these conditions, dehydration rather than heat stress accounts for the majority of the cardiovascular, thermoregulatory and metabolic strain that endurance athletes may experience (Gonzales-Alonso *et al.*, 2008). Nevertheless, strategies designed at improving exercise perfoamnce must focus both on limiting dehydration as well as hyperthermia.

1.1.4. Prevention Strategies

Prevention of dehydration requires endogenous intervention across the whole spectrum of athletic practice; before, during and after exercise. The only practical way to rehydrate during exercise is drinking (Greenleaf & Brock, 1980). The most common approaches have involved the ingestion of large volumes of water or a low-sodium carbohydrate-electrolyte [CH-E] drink during athletic practice. These studies have tended to show an improvement in performance proportionate with the level of rehydration (Adolph & Associates, 1947; Montain & Coyle, 1992a). However, some events and most occupational scenarios, in which dehydration is common, do not permit easy access to fluid and as such the practicality of these *during-exercise* interventions is questioned. In these cases the use of pre-exercise interventions therefore may provide a more practical intervention to prevent dehydration. When exercise is followed by a subsequent bout, for example during training; during occupational scenarios with heat exposure or when hypohydration has been induced to meet weight categories, rapid rehydration may be necessary to prevent an athlete beginning exercise in a dehydrated state.

1.1.4.1. Sodium

Under stressful conditions fluid intake frequently fails to keep up with sweat loss, with fluid replenishment of approximately 50 % being normal (Hubbard *et al.*, 1984; Pitts *et al.*, 1944; Pugh *et al.*, 1967). The failure to drink either occurs because of a mismatch between thirst and physiological need; or because of an unacceptability or unavailability of sufficient fluid. The ingestion of large volumes of fluid is neither desirable nor practical in many sporting or occupational scenarios. When the rate of fluid intake is insufficient to avoid a TBW deficit the artificial acceleration of P_{Vol} restoration may still provide an ergogenic benefit.

A number of studies have found P_{Vol} expansion an effective measure, capable of improving cardiovascular functioning and/or reducing T_{Core} when employed both before (e.g. Fortney *et al.*, 1981a; 1981b) and during (e.g. Dechamps *et al.*, 1989; Nose *et al.*, 1990) exercise. These studies have principally used infusions of either whole blood or artificial P_{Vol} expanders such as albumin, dextran or saline, which are either impractical and/or prohibited by the World Anti Doping Agency (2010). A more feasible method, which accelerates the restoration of P_{Vol}, involves ingesting large volumes of water

mixed with a binding agent such as glycerol or sodium. The use of glycerol has been extensively studied, producing inconsistent results and some detrimental side effects. In contrast, the use of sodium as a method to accelerate P_{Vol} expansion has received relatively little attention.

The employment of high sodium concentrations has received more attention when included in a rehydration solution following exercise-induced dehydration (e.g. Ismail *et al.,* 2007; Merson *et al.,* 2008; Mitchell *et al.,* 1994; 2000; Maughan & Leiper, 1993; Maughan *et al.,* 1994; Shirreffs & Maughan, 1998; Saat *et al*, 2002). The consistent improvements in rehydration with the addition of sodium to a rehydration drink is purported to be a result of increases in fluid retention, or rather, reductions in U_{Vol}. Sodium ingestion promotes P_{Vol} expansion since the volume of extracellular fluid is directly proportional to the total body content of sodium (Verbalis, 2003). If for example, an athlete were to ingest only plain water, the decrease in P_{Osm} would promote diuresis and cause unwanted fluid losses. In contrast, the addition of sodium to the rehydration solution preserves P_{Osm}, sustains the osmotic drive for thirst and minimises diuresis (Merson *et al.,* 2008; Nose *et al.,* 1988c; Shirreffs *et al.,* 1996; Takamata *et al.,* 1994). Put in simple terms, for every unit of fluid ingested, a greater proportion would remain within the vascular space promoting gains in P_{Vol}.

1.1.4.2. Alternative strategies

Whilst the prevention of dehydration represents one strategy to improve performance during exercise in the heat, others have sought to minimise the effects of hyperthermia, thus improving performance both directly, and indirectly through a reduced strain on the dual demands the heart faces in delivering blood to the working muscles and the periphery for cooling. The most common strategy employed to reduce hyperthermia, other than fluid ingestion is pre-cooling. The most commonly tested pre-cooling strategies are cold air exposure (Olschewski & Bruck, 1988), cold water immersion (Booth *et al.,* 1997), or the wearing of ice vests (Arngrimson *et al.,* 2004), none of which represent a consistently practical, comfortable, or effective intervention with widespread utility for athletic or occupational environments (Marino, 2002; Quod *et al.,* 2006). One intervention which does provide a highly practical pre-cooling strategy is the ingestion of cold fluid (0-4^{o}C) and has been shown to reduce T_{Rec} (Lee & Shirreffs, 2007; Mundel *et al.,* 2006), lower heart rate (Mundel *et al.,* 2006), and

improve exercise capacity (Mundel *et al.,* 2006). The ingestion of cold water has also been shown to reduce T_{Rec} when ingested prior to exercise (Lee *et al.,* 2008), and improve performance when combined with the continued ingestion of 500-700 mL of cold fluid during exercise, (Lee *et al.,* 2008). However, it is not known whether exercise performance would improve if fluid was ingested pre-exercise alone.

1.2. Endocrine regulation of fluid and sodium balance

Body fluid balance is meticulously regulated within narrow limits by neuroendocrine control systems (Geering & Loewry, 2008). Following a change in the volume or content of the extracellular fluid ([ECF]; which includes blood plasma), these control systems induce appropriate compensations to firstly conserve and secondly restore fluid and sodium balance. Central to the control of fluid balance is the concentration and volume of ECF (Antunes-Rodriguez *et al.,* 2004). The ratio of fluid within cellular compartments is controlled by the osmotic equilibrium achieved by the relative concentrations of impermeable ions found in each. The concentration of sodium ions within ECF is the principle factor determining osmotic equilibrium and ECF volume (Antunes-Rodriguez *et al.,* 2004; Johnson & Thurnhorst, 1997). Therefore the addition (e.g. through ingestion) or loss (e.g. through perspiration) of either water or sodium affects the osmotic equilibrium thereby changing the relative distribution of fluid between the compartments.

In response to disturbances to homeostasis, such as hypovolemia and hyperosmolality, the body initiates a series of automated and behavioural responses designed at restoring balance as rapidly as possible. Even in benign environments, the major components of body fluids are in constant flux (Johnson, 2007), thus the body has developed reflex mechanisms which respond to small fluctuations in fluid balance. Sympathetic reflex mechanisms occur within seconds of fluid loss, but have limited scope and serve only to maintain cardiovascular functioning, through vasoconstriction or vasodilation (Guyton & Hall, 1996).

The release of the endocrine hormones, Arginine Vasopressin [AVP] and Aldosterone are stimulated within minutes of fluid imbalance (Johnson & Thurnhorst, 1997) and are involved in a complex process to restore fluid balance. In response to hyperosmolality and to a lesser extent hypovolemia, AVP a peptide hormone is released

from the posterior pituitary with the primary purpose to promote antidiuresis (Bourque & Oilet, 1997; Bourque *et al.,* 1997). Elevated AVP concentrations within the bloodstream increase the permeability to water of the distal convoluted tubules in nephrons of the kidneys. This encourages greater water re-absorption, from urine back into the bloodstream.

Since sodium represents less than 1 % of the ECF by weight, the mass of sodium required for volume restoration is quite small relative to the amount of water that must be consumed. Accordingly, antidiuresis and thirst are the primary homeostatic drives stimulated in response to dehydration. However, P_{Osm} is regulated primarily by the ingestion and excretion of water, whereas the volume of extracellular fluid is directly proportional to the total body content of sodium (Verbalis, 2003). Expansion of P_{Vol} is therefore absolutely limited by sodium intake. Water intake alone is inadequate to completely replace volume losses following sweat induced losses of fluid and electrolytes.

Initially, the most critical aspect of sodium regulation is retention by the kidney. Following sodium loss, and in response to both changes in fluid volume and stimulation of osmoreceptors (Andersen *et al.,* 1998; Bie & Sandgaard, 2000), activation of the rennin-angiotensin system [RAS] increases secretion of the sodium retaining hormone, aldosterone (Maughan & Leiper, 1995; Maughan *et al*., 1996; Nose *et al*., 1988a; Takamata *et al*., 1994). Aldosterone, a hormone from the mineralocorticoid family is produced in increased amounts from the adrenal gland. Aldosterone is principally an anti-natriuretic hormone (DiBona 1985), which once released increases the reabsorption of sodium ions and water and the secretion of potassium ions in the collecting ducts and distal convoluted tubule of the kidneys nephrons. It also acts to reduce sodium levels, sweat and saliva (Castenfors, 1967; Thongboonkerd *et al.,* 2003).

The actions of the kidneys can only conserve what is already present in the body; ECF can neither be increased nor reduced without the ingestion of sodium. Sodium appetite (also known as salt appetite) is a motivated behavioural state that arises specifically as a response to sodium deficiency. It drives an animal to seek and ingest foods and fluids that contain sodium. Sodium appetite is a regulatory mechanism which like thirst is essential for complete restoration of ECF.

1.3. Gustatory Responses

Failure to adequately replace fluid losses when allowed to drink *ad libitum* has consistently been reported in the literature (Adolph & Associates, 1947; Carter & Gisolfi, 1989; Nose *et al.,* 1988b). Since fluid intake during and after exercise rarely matches fluid losses during exercise, every effort should be made to provide access to fluid which will encourage its consumption to the greatest degree. It is surprising then that relatively little is known about drinking behaviour following exercise. One of the most important factors in the process of understanding drinking behaviour is palatability (Maughan, 1998; Minehan *et al.,* 2002); a drink that tastes good contributes more to voluntary hydration than a drink which tastes unpleasant. The inclusion of sodium in a rehydration solution has been purported to increase fluid retention and improve rehydration (Maughan *et al.,* 1994; Maughan & Leiper, 1995; Maughan, 1998; Maughan *et al.,* 2004; Merson *et al.,* 2008; Mitchell *et al.,* 1994; Mitchell *et al.*, 2000; Shirreffs *et al.,* 1996; Shirreffs & Maughan, 1998), yet there are clearly palatability issues that influence the formulation.

Providing individuals with a drink that tastes good may enhance rehydration during exercise and recovery. The problem with high sodium concentration is that this may exert a negative effect on taste, resulting in a reduced consumption. Thus when drinking is *ad libitum* the inclusion of high sodium concentrations may be detrimental to rehydration. The perception of salt can be alleviated to a large degree by substituting chloride with other anions such as (sodium) citrate, or by appropriate flavouring. Increasing the sweetness may go some way to masking the unpleasant saltiness, but the addition of both electrolytes and carbohydrates significantly increases the osmolality of the drink, which may impair gastric emptying and intestinal absorption. Cooling may also improve the palatability of some drinks, most probably through the de-sensitisation of the area of the tongue. However, this is not the case with salt. The taste fibres, which respond to salt taste, are very sensitive to changes in temperature (Nakamura & Kurihara, 1988) and the taste intensity of salt has been reported to increase with cooling (Cruz and Green 2000; Talavera *et al.,* 2007).

Assessing palatability is not as simple as it seems since palatability is not a constant phenomena but one that is dependent on the physiological state of the consumer. Changes in taste palatability have been demonstrated to respond to differences within individuals dependent on short-term disturbances to homeostasis

such as hunger (Yeomans, 1998), satiety (Rolls *et al.*, 1980), hyponatraemia (Beauchamp *et al.*, 1990; Huang & Yan, 2008), and following dietary manipulation (Bertino *et al.*, 1982) and exercise (Appleton, 2005; Lesham *et al.*, 1999; Takamata *et al.*, 1994). Therefore a drink which tastes unpleasant before exercise will not necessarily taste unpleasant after exercise. Knowledge of what determines drinking behaviour following exercise may help establish optimum rehydration strategies and help assess the efficacy of high sodium concentrations.

1.4. Thesis Aims and Hypotheses

This thesis will examine different strategies to prevent dehydration, and improve thermoregulation, cardiovascular functioning and exercise performance. Whilst this area has received much research over the last century, too few of the interventions employed provide a practical solution with universal utility in both an athletic and occupational scenario. The studies in this thesis will examine whether the ingestion of high concentrations of sodium represent an effective means to reduce dehydration when employed both during and before exercise and to improve rehydration when employed after exercise. In order that these interventions conform to the goal of producing practical interventions, this thesis will also investigate the impact of ingesting a high sodium concentration on drinking behaviour. The second aspect of this thesis was to examine the perceptual taste responses to ingesting high sodium drinks and the effects of exercise-induced dehydration on these.

The central hypothesis of this thesis was that ingestion of a highly concentrated sodium beverage would form an effective and efficient intervention to improve physiological functioning when employed before, during and after exercise. More specifically it was hypothesised that that ingestion of a highly concentrated sodium sports drink will improve hydration when employed before, during and after exercise via a reduction in U_{Vol} and an increase in P_{Vol} mediated by elevated P_{Na} and P_{Osm}. It is secondly hypothesised that this improved hydration will delay the onset and/or lessen the effects of exercise-induced dehydration, and the negative physiological responses which accompany it, when the highly concentrated sodium drink is ingested both before and during exercise. Thirdly that, as a result of the improved physiological functioning or other means yet unknown, ingestion of the highly concentrated sodium drink will increase exercise capacity and delay the onset of exhaustion. Fourthly, it was

hypothesised that dehydration would induce an increase in the palatability of water and a decrease in the palatability of the sodium drinks. Lastly, it was hypothesised that the magnitude of change in palatability for these drinks would be dependent fluid balance in relation to the physiological responses which accompany dehydration

Chapter 2: Literature Review

2.1. Factors affecting exercise performance

The physiological mechanisms involved with hyperthermia-induced fatigue include a complex interplay of factors, each of which has been subject to much scrutiny and debate. In the main, they can be classified into avenues affecting cardiovascular functioning, muscle metabolism and neuromuscular functioning (central fatigue; Hargreaves & Febraio, 1998; Gonzales-Alonso & Calbert, 2003; Nybo & Secher, 2004; Nybo & Nielsen, 2001a). Though each factor is unique, evidence suggests that they interact together, rather than in isolation, to degrade aerobic exercise performance (Sawka & Coyle, 1999; Cheuvront *et al.*, 2003; Cheuvront *et al.*, 2004). The relative contribution of each factor may differ depending on the specific activity, environmental conditions, heat acclimatization status and fitness status of the athlete.

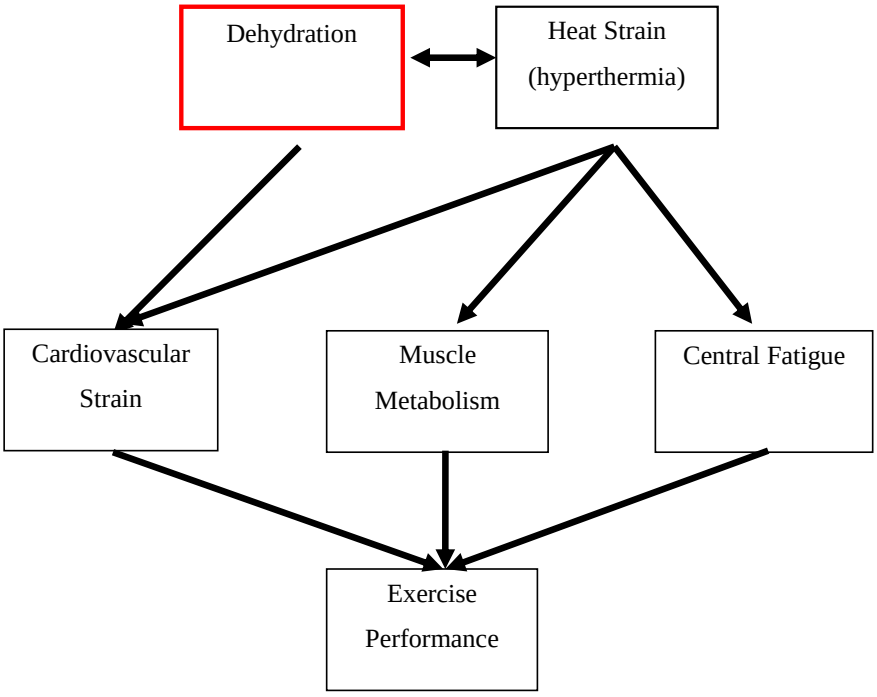

Figure 1.1. Mechanisms by through which dehydration and hyperthermia impact exercise performance.

2.1.1. Cardiovascular function

When prolonged exercise is performed at heavy intensities in a warm-hot environment without fluid replenishment, such that significant dehydration develops, progressive reductions in $\dot{Q}$ and arterial pressure accompany elevations in systemic vascular resistance, cutaneous vascular resistance and plasma noradrenaline levels (Gonzalez-Alonso *et al.* 1995, 1998, 1999b; Gonzales-Alonso & Calbert, 2003). A possible reduction in $\dot{Q}$ of up to 4 $l \cdot min^{-1}$ is accompanied by a reduction in active BF_{Mus}. Failure to deliver the required volume of oxygenated blood to the working muscle would increase the reliance on anaerobic metabolism. This in turn results in higher blood lactate accumulation during exercise as the rate of lactate production exceeding the removal.

Gonzales-Alonso & Calbert (2003) found that the decline in $\dot{V}O_{2max}$ during heat stress was directly related to the fall in $\dot{Q}$ and the concomitant inability to maintain O_2 delivery to exercising muscle. Participants' T_{Core} and T_{Skn} were increased by 1^{o}C and 10^{o}C, respectively when cycling to exhaustion. Heat stress accelerated the decline in $\dot{Q}$, BF_{Mus}, O_2 delivery, and $\dot{V}O_2$. The authors attributed the decline in O_2 delivery to lowering of skeletal muscle O_2 delivery since arterial O_2 content, exercising leg O_2 extraction, and leg vascular conductance were all unaltered. They also reported enhanced muscle lactate accumulation in the exercising leg despite non-depleted muscle energy stores at the end of exercise. More recently, the work of Wingo and colleagues (Wingo *et al.*, 2005a; 2005b; Wingo & Cureton, 2006a; 2006b) has also established a relationship between CV_{Drift} and the decline in $\dot{V}O_{2max}$ which occurred between 15 and 45 min of moderate intensity exercise in the heat (60 % $\dot{V}O_{2max}$; 35^{o}C, 40 % RH). Across all these studies, the greater the increase in HR and reduction in SV, the greater the decline in $\dot{V}O_{2max}$. These findings suggest a significant role of cardiovascular functioning to limiting $\dot{V}O_{2max}$.

The lower SV could therefore be a critical factor in the development of fatigue with dehydration and/or hyperthermia (Montain & Coyle, 1992; Gonzalez-Alonso *et al.* 1997; 1999c; 2000). It appears that cardiovascular factors play a significant role in the development of fatigue during exercise in the heat, at least when performed at high intensities (Nybo, 2008). However, when exercise is performed at a submaximal level,

evidence suggests that reduced BF_{Mus} is not the primary source of fatigue in prolonged moderate exercise in the heat in as much as muscle O_2 delivery is for the most part maintained by a parallel increase in O_2 content with dehydration-induced haemoconcentration and thus active muscle $\dot{V}O_2$ only tends to be depressed at exhaustion (Gonzalez-Alonso *et al.,* 1998).

2.1.2. Muscle Metabolism

When exercise is performed at a submaximal level, oxygen delivery remains adequate, and therefore anaerobic metabolism does not reach levels associated with muscle fatigue. Furthermore, following prolonged exercise in the heat to exhaustion, both Nielsen *et al.* (1993) and Nybo & Nielsen (2001a) observed unchanged force production during brief multiple voluntary contractions [MVCs] for both exercised and "nonexercised" muscle groups. Morrison *et al.* (2004) confirmed these findings during passive heating.

Whilst the ability to produce a given force in a single episode when electrically stimulated is not affected by hyperthermia, the ability to sustain force production over an extended period of time is. The ability to sustain power output for prolonged periods deteriorates and performance during repeated sprinting is also impaired by hyperthermia (Nybo & Nielsen, 2001a). However, the transferability of the results from *in vitro* measurements to *in vivo* situations is not clear (Nybo, 2008). More research is required to examine this relationship in more depth.

2.1.3. Central Fatigue

The nervous system plays a vital role in coordinating physiological responses in the human body. The neuromuscular fatigue model refers to the reduction in the force or power production of a muscle despite increases in perception of effort (Gandevia *et al.,* 2001). The theory proposes that the development of fatigue is associated with the reduction of muscle activation by the central nervous system (Avela *et al.,* 2001). A number of researchers have suggested that a critical T_{Core} exists, at which point performance is terminated to prevent against the possibility of ill health (Gonzalez-Alonso *et al.,* 1999; Neilsen *et al.,* 1993). Evidence for the role of central fatigue

impairing exercise performance during hyperthermia, is the observation that fatigue occurred at the same T_{Core}, just above 40°C, regardless of the initial T_{Core} or rate of heat storage (Gonzalez-Alonso *et al*. 1999a). However, there is some evidence to suggest that high T_{Core} is not a limiting factor during exercise, since final T_{Rec} has been demonstrated between 38°C and 39°C in untrained participants, and a T_{Core} far in excess of 40°C in trained athlete finishing marathon in hot and humid conditions (Byrne *et al.*, 2006).

Technology advances mean that measurement of brain temperature and blood flows are now possible. Through these Nybo and colleagues has shown that exercise in the heat is associated with elevated cerebral $\dot{V}O_2$ and a reduction in cerebral blood flow (Nybo and Nielsen, 2002). During exercise in temperate conditions, cerebral blood flow is markedly increased during exercise (Ide & Secher, 2000), but a decline in blood flow to the brain has been reported during exercise with hyperthermia (Nybo & Nielsen, 2001b). This effect may be mediated, at least in part, by blood flow changes occurring in response to redistribution of cardiac output due to exercise-heat stress (Maughan *et al.*, 2007). A high cerebral temperature is itself associated with reduced alterations in electrical brain activity, and decreased voluntary activation of peripheral nervous pathways and neuromuscular drive (Nagasaka *et al.*, 1998) and consequently, decreased physiological function.

It is thought that a high cerebral temperature may lead to alterations in motor drive that affect the ability to recruit sufficient muscle fibres to meet the demands of exercise (Nybo & Nielsen, 2001a). An association between hyperthermia and fatigue-induced changes in motor unit recruitment and/or discharge rates have also previously been demonstrated (Gonzalez-Alonso *et al.*, 1999), although only when the environmental temperature was uncompensatable and the level of heat gain was severe. In addition, Nielsen *et al.* (2001) found that during hyperthermia, changes in the Alpha to Beta (brain waves) index were correlated with increases in RPE. This suggests that EEG frequency shifts might reflect decreased arousal changing the ability of the brain to sustain motor activity during hyperthermia. Taken together these findings suggest a role for brain activity in the development of fatigue during heat stress, but more evidence is needed to fully evaluate its role.

Recent research examining the association between CNS in fatigue has focused on alterations in brain neurotransmitters and hormones such as serotonine and dopamine (e.g. Davis *et al.*, 2000; Low *et al.*, 2005). Studies supporting the notion that fatigue is

linked to the CNS have observed an increase concentration of serotonin and dopamine in the brain during prolonged exercise, which reduces the level of excitement and recruitment of skeletal muscle (Davis *et al.,* 2000). In addition, levels of dopamine have been suggested to increase during exercise heat stress (Meeusen *et al,* 2006). Furthermore, increase in both serotonin and dopamine has been linked to the reduction in perception of effort, which then reduces exercise performance (Davis *et al.,* 2000). However, due to the complexity of measuring these substances in the human brain, indirect measurements from peripheral blood markers are often used. It is not known what volume of serotonin and dopamine found in peripheral blood orginates from the brain.. It is, likely that various tissues during exercise and hyperthermia release dopamine and serotonin during these times.

The phenomenon of central fatigue, appears only to occur with higher than normal T_{Skn}, normally only seen when exercise is performed in a hot environment in excess of typical ambient temperatures most commonly witnessed during athletic competition such as the Summer Olympic Games for example. Thus it appears, the CNS aspect of hyperthermia-induced fatigue is only relevant during prolonged exercise, where the temperatures of the core and brain may exceed 40°C. In contrast, the impairment in oxygen delivery to the exercising muscles becomes relevant only during high-intensity exercise, where $\dot{Q}$ declines significantly and BF_{Mus} decreases to such extent that increased oxygen extraction cannot compensate for the reduced oxygen delivery (Gonzales-Alonso *et al.,* 1998; Gonzales-Alonso & Calbert, 2005; Nybo, 2008).

2.2. Rehydration

Substantial sweat losses are incurred during prolonged exercise in the heat, where sweating rates may reach 2-3 $L \cdot h^{-1}$ during intense exercise in a warm environment (Maughan *et al.,* 1994; McArdle *et al.,* 1996; Saltmarsh, 2001). Yet, rates of fluid intake during exercise rarely match rates of sweat loss (Adolph & Dill, 1938; Carter & Gisolfi, 1989; Greenleaf 1992; Hubbard *et al.,* 1984; Maughan *et al.,* 2004; Passe, 2001; Pitts *et al.,* 1944; Saltmarsh, 2001), making dehydration a distinct possibility.

There are several explations for the failure of athletes to rehydrate appropriately during exercise. Firstly, not all situations present adequate opportunities to drink. Marathon runners and triathletes, who commonly carry little or no drinks, have access to fluids only at designated places, whilst fell runners have to rely on the position of streams. Additionally those involved in team games such as football have to wait for stoppages in play before they have the opportunity to drink. Many recreational athletes have a poor understanding of their fluid requirements, and when given *ad libitum* access to fluid, people tend to drink at a rate that replaces only around 50 % of their fluid losses (Hubbard *et al.*, 1984; Pitts *et al.*, 1944; Pugh *et al.*, 1967). Lastly, the failure to match fluid intake to sweat losses may also be a consequence of a limited capacity for gastric emptying. During exercise gastric emptying rarely exceeds 1-1.2 $L \cdot h^{-1}$, unless a large volume is maintained in the stomach. Many athletes have a poor tolerance of drinks during competition, experiencing discomfort with high volumes in the stomach, particularly when running is involved. Thus in race situations it is unlikely that athletes could drink more than 2 $L \cdot h^{-1}$, even if they could tolerate it.

Thus although individual drinking practices vary markedly (Noakes & Martin, 2002), many athletes finish a bout of exercise in a dehydrated state (Broad *et al.* 1996; Burke & Hawley, 1997; Maughan *et al.* 2004; Sharp, 2006; Shirreffs *et al.* 2005). The degree of dehydration is increased when exercise is performed in the heat, when greater sweat rates widen the gap between fluid intake and fluid loss. Failure to achieve adequate restoration of fluid balance may result in an athlete beginning a subsequent exercise session in a dehydrated state, which could impair cardiovascular and thermoregulatory function (Montain & Coyle, 1992a; Nadel *et al.*, 1979) to the detriment of exercise capacity. Following a fluid regime that produced only 75 % rehydration, Nielsen *et al.* (1986) reported that sub-maximal exercise elevated HR compared with controls. Similarly, Heaps *et al.* (1994) reported that 65 % rehydration did not restore cardiovascular function as indicated by the persistence of elevated HR and decreased SV. It should be noted that Costill and Sparks (1973) did find that the HR response to exercise was normalised with only 62% replacement.

Nevertheless athletes should be encouraged to rehydrate by 100 % in order to begin subsequent bouts in optimal condition. The first two hours of rehydration have a marked influence on the rate of recovery and performance in subsequent athletic events on the same day (Gisolfi & Duchman, 1992). However, achieving complete restoration within 2 h can be particularly challenging when sweat losses are high or available time

short. This may be common during sporting competitions, where athletes are competing in more than one event per day, during training, or in a workplace environment. Another situation where restoration of a fluid and electrolyte deficit may be of crucial importance is in weight-category sports, where a variety of techniques, including diuretic use and exercise and thermal dehydration, are employed to achieve the desired body mass.

Given the tendency for individuals to fail to match sweat losses during exercise and the relative importance of ensuring the restoration of whole-body fluid balance before the start of a subsequent bout of exercise, considerable research has been devoted to understanding the rehydration process and the restoration of body fluids lost during prior exercise. A variety of rehydration protocols have been reported in the literature many of which have been designed to investigate the influence of electrolyte content and fluid volume on rapid rehydration. There is no general consensus of the optimal rehydration from the reported literature. Discrepancies in these findings are largely related to the variability in rehydration protocols employed, and the drink content and volume.

2.2.1. Volume

In studying rehydration after exercise-induced body fluid loss, investigators have employed three models for rehydration: allow participants to drink fluids *ad libitum* during the rehydration period (Nose *et al.,* 1988b; 1988c; Wemple *et al.,* 1997); prescribe fluid intake to match the fluid lost during the prior exercise (Costill & Sparks, 1973; Gonzalez-Alonso *et al.,* 1992; Ray *et al.,* 1998; Nielsen *et al.,* 1986), and prescribe fluid intake in excess of the fluid lost in the prior exercise (Maughan & Leiper, 1995; Merson *et al.,* 2008; Mitchell *et al.,* 2000; Shirreffs *et al.,* 1996; Shirreffs & Maughan, 1998).

Allowing participants to rehydrate *ad libitum* provides the best representation of actual athletic practice and yet rarely has this method been used to investigate recovery from exercise. This is largely because differences in fluid intake make comparisons between different types of drink difficult. Additionally the volume of *ad libitum* fluid intake is rarely sufficient to restore fluid balance. For example, when six participants were dehydrated by 2.3 % BM loss using thermal and exercise induced fluid loss, *ad libitum* intake of neither tap water + placebo nor tap water + sodium capsules (77

mmol·L^{-1}) induced complete fluid restoration, achieving just 68 and 82 %, respectively (Nose *et al.,* 1988c). Similar studies have also failed to report 100 % fluid replacement with *ad libitum* fluid intake of either plain water or a low sodium (9.2 mmol·L^{-1}) CHO-E drink during 3 h recovery from 1.9-3 % BM loss (Carter & Gisolfi, 1989; Wemple *et al.,* 1997). Thus even with the ingestion of sodium to a rehydration drink, *ad libitum* intake is insufficient to restore fluid balance. For complete restoration a volume of fluid in excess of *ad libitum* intake must be prescribed.

Several studies have examined recovery of body water losses after exercise by providing a volume of fluid equal to the volume of water lost during exercise. Costill and Sparks (1973) dehydrated eight male participants using intermittent exposure to dry heat (70^{o}C) until 4 % of BM loss. Participants ingested a volume of fluid equal to 100 % BM loss of either water or a low sodium CHO-E drink at every 15 min intervals throughout 3 h rehydration period, and yet only 62 % of TBW was restored.

Based on the observations, the majority of studies in recent times have provided fluid in excess of that which was lost during exercise (e.g. Maughan & Leiper, 1995; Merson *et al.,* 2008; Mitchell *et al.,* 1994; Mitchell *et al.,* 2000; Shirreffs *et al.,* 1996; Shirreffs & Maughan, 1998). The rationale for this is, in part, because of the continuation of sweat loss during rehydration and respiratory water losses, but predominantly to compensate for the ongoing urine losses that persist despite individuals being in body-water deficit (Mitchell *et al.,* 1994; Shirreffs *et al.,* 1996). Urine production can be as high as 500 mL·h^{-1} following rehydration (Shirreffs *et al.,* 1996). Therefore, the majority of published studies are in general agreement that the volume of fluid ingested during rehydration should be at least 150 % BM loss (e.g. Mitchell *et al.,* 1994; 2000; Shirreffs *et al.,* 1996).

2.2.2. Drink Content

Merely drinking a large volume of fluid after exercise-induced dehydration is not sufficient to achieve complete rehydration (Shirreffs *et al.,* 1996). Continued ingestion of large volumes of plain water alone, results in a rapid fall in P_{Na} and P_{Osm} (Costill & Sparkes, 1973; Gonzalez-Alonso *et al.,* 1992; Nose *et al.,* 1988a; Nose *et al.,* 1988b; Nose *et al.,* 1988c), stimulating urine production by increasing the permeability

of the distal tubule, and suppresses thirst; therefore delaying the rehydration process (Gonzalez-Alonzo *et al.,* 1992; Shirreffs *et al.,* 1996; Shirreffs & Maughan, 1998).

Whilst P_{Osm} is regulated primarily by the ingestion and excretion of water, the volume of extracellular fluid is directly proportional to the total body content of sodium (Verbalis, 2003). Water intake alone is adequate to replace volume losses only when the sodium concentration is elevated owing to a loss of water greater than the loss of sodium (Geering & Loewry, 2008). In contrast fluid losses involving large amounts of sodium, such as with sweat losses during prolonged exercise, cannot be solely maintained by ingestion water. Thus, the total volume of ECF in the body depends largely upon the amount of sodium present in the extracellular space, around which water input and output are tailored to tightly control osmotic pressure. This regulatory arrangement is the reason that sodium must be excreted to reduce P_{Vol}, and it must be ingested and retained to increase P_{Vol}. In such situations, consuming too much fluid without electrolytes will produce hyponatraemia, which typically inhibits further drinking (Stricker, 1966) and if continued can promote water intoxication and ill health.

2.2.2.1. Sodium

The addition of sodium to water enables the body to retain an isotonic mixture of ingested sodium and water to more effectively restore P_{Vol} (Nose *et al.,* 1988a; Twerenbold *et al.,* 2003). The addition of sodium to the rehydration solution preserves P_{Osm}, sustains AVP secretion and maintains the osmotic drive for thirst and antidiuresis (Merson *et al.,* 2008; Nose *et al.,* 1988c; Shirreffs *et al.,* 1996; Takamata *et al.,* 1994). A reduced diuresis, increased thirst and a greater fluid restoration have consistently been reported when sodium has been added to the rehydration solution (Costill & Sparkes, 1973; Gonzalez-Alonso *et al.,* 1992; Ismail *et al.,* 2007; Merson *et al.,* 2008; Mitchell *et al.,* 1994; 2000; Nielsen *et al.,* 1986; Shirreffs *et al.,* 1996). Perhaps most importantly, it is the reduction in diuresis which is paramount to the improved restoration of TBW with sodium ingestion. This is well illustrated by Figure 2.1. which depicts the decrease in U_{vol} following the ingestion of increasing concentrations of sodium from 0-100 $mmol \cdot L^{-1}$.

As well as maintaining osmotic drive for antidiuresis and thirst, the addition of sodium accelerates the restoration of ECF. Since sodium balance is closely related to extracellular balance (Nose *et al.,* 1988b; Wemple *et al.,* 1997), its ingestion is

necessary for restoration of P_{Vol}. In addition, sodium stimulates glucose and water intestinal absorption (Modigliani & Bernier, 1971), and thus may accelerate the rate of rehydration compared with plain water alone (Maughan *et al.,* 1994). The sodium content of rehydration solutions provided after exercise-induced dehydration is therefore a significant factor in the restoration of body water loss (Nose *et al.,* 1988a; Wemple *et al.,* 1997).

Several other electrolytes have been added to rehydration drinks but with fewer reported benefits. Potassium is normally present in commercial sports drinks in concentrations similar to those in plasma (4-6 $mmol \cdot L^{-1}$), although there is no definitive evidence to support its inclusion (Maughan, 1998). Some loss of potassium in sweat in the region of ~3-7 $mmol \cdot L^{-1}$ does occur (Qayyum *et al.,* 1993; Shirreffs & Maughan, 1997), but this is insufficient to affect exercise performance. Maughan *et al.* (1994) found that potassium chloride was as effective as sodium chloride [NaCl] in restoring fluid balance; however, this effect was likely to be due to the chloride contribution and its impact on the ECF compartment. Potassium deficiencies may be prevented by conservation of potassium in urine and an increase in the circulating potassium concentration (Kilding *et al.,* 2009; Sejersted & Sjøgaard, 2000). If an athlete develops potassium deficiency, it is usually caused by drugs, such as diuretics or corticosteroids, or by diarrhoea or repeated vomiting. There is also a lack of evidence to support the inclusion of magnesium in sports drinks. Although magnesium deficiency has been implicated in the development of muscle cramps (Liu *et al.,* 1983), the slight decrease in the plasma magnesium which has been reported during exercise (Consolazio *et al.,* 1963) may be a result of the redistribution of magnesium, not a loss *per se*, since they return to baseline levels within 2 h of terminating exercise (Deuster *et al.,* 1987). Whether or not potassium has an ergogenic effect or not, there is little argument that sodium has the greatest impact on fluid retention (Maughan & Leiper, 1995; Neilsen *et al.,* 1986; Greenleaf *et al.,* 1998; Shirreffs *et al.,* 1996).

Low Sodium Concentrations

A number of studies have compared the effects of ingesting either water or a low sodium carbohydrate drink (< 25 $mmol \cdot L^{-1}$), such as those found in commercially available CHO-E sports drinks. Costill and Sparks (1973) dehydrated eight male participants using intermittent exposure to dry heat (70^{o}C) until 4 % of BM loss.

Participants ingested a volume of fluid equal to 100 % BM loss in equal boluses at 15 min intervals throughout 3 h rehydration period, in a thermo-neutral environment, of either water or a CHO-E drink (22 $mmol \cdot L^{-1}$ Na; 0.5 $mmol \cdot L^{-1}$ glucose). With CH-E, U_{Vol} was significantly reduced (367 vs. 602 mL) and P_{Vol} restoration significantly increased (67 vs. 38 %) compared to water, and yet only 62 % of TBW was restored. Concomitant with the greater fluid retention and P_{Vol} restoration with CHO-E was an elevated serum sodium concentration and osmolality. Gonzalez-Alonso *et al.* (1992) confirmed that a CHO-E solution (60 $g \cdot L^{-1}$ carbohydrate, 20 $mmol \cdot l^{-1}$ Na) was more effective in promoting post-exercise rehydration than either plain water or a low-electrolyte diet cola. In contrast, Ostenberg *et al.* (2010) found that following 90 min exercise in the heat to 2-3 % BM loss, ingestion of 18 $mmol \cdot L^{-1}$ electrolyte drink did not significantly increase fluid retention compared with plain water (72 vs. 66 %), unless a 12 % glucose solution was added to the drink.

Even when a difference in fluid restoration between water and low sodium CHO-E drinks was reported, positive fluid balance was not achieved, even after 2-3 h (Costill & Sparks, 1973; Gonzalez-Alonso *et al.*, 1992). A number of other studies have also failed to report positive fluid balance with low concentrations of sodium (< 25 $mmol \cdot L^{-1}$), even when the volume of fluid ingested was equivalent to 120-200 % of BM loss (Aragón-Vargas & Madriz-Dávila, 2000; Maughan & Leiper, 1995; Maughan *et al.*, 1996; Shirreffs *et al.*, 1996; Shirreffs & Maughan, 1998; Ismail *et al.*, 2007). For example, Mitchell *et al.* (1994) demonstrated only 73 % rehydration with a 14 $mmol \cdot L^{-1}$ solution after 3 h recovery; and after 6 h, Shirreffs *et al.* (1996) reported that fluid balance was not achieved with ingestion of a 23 $mmol \cdot L^{-1}$ solution.

The failure to achieve positive fluid balance with low sodium drinks is attributed to the high urine flow. The loss of large volumes of fluid in the form of urine is the primary obstacle to rapid rehydration; thus the effectiveness of a rehydration solution is assessed by the extent to which it retains ingested fluid (Mitchell & Voll, 1991). It should be noted that a positive fluid balance was achieved in one study with ingestion of 150 % BM losses of a low sodium drink (25 $mmol \cdot L^{-1}$ Na; Mitchell *et al.*, 2000).

Moderate Sodium Concentrations

Maughan & Noakes (1991) suggest that sodium concentrations of 30-50 $mmol \cdot L^{-1}$ are optimal for fluid replacement. This is probably since this figure is close to the sodium concentration of sweat, which ranges from ~20-80 $mmol \cdot L^{-1}$ (Costill, 1977; Costill *et al.,* 1976; Maughan, 1991; Rehrer, 2001; Verde *et al.,* 1982). A number of studies have reported greater fluid retention and rehydration with moderate sodium concentrations compared with low sodium concentrations or water (Maughan & Leiper, 1995; Shirreffs *et al.,* 1996; Shirreffs *et al.,* 2007). In a series of experiments from the same group, positive fluid balance was achieved with moderate concentrations of sodium, following dehydration equivalent to 1.8-2.3 % (Shirreffs *et al.,* 1996; Shirreffs *et al.,* 2007; Maughan & Leiper, 1995). For example, Shirreffs *et al.* (1996) reported greater fluid restoration with a 61 vs. 23 $mmol \cdot L^{-1}$ Na drink following ingestion of three different volumes of fluid ingestion: 100 % BM loss (81 vs. 60 % recovery); 150 % BM (107 vs. 91 %); and 200 % BM (127 vs. 91 %). There was no difference in fluid restoration when 50 % of BM losses were ingested. In all thee studies, U_{vol} was found to be inversely proportionate to the drink sodium concentration.

Maughan and Leiper (1995) investigated the effects of ingesting varied concentrations of sodium (2, 26, 52, 100 $mmol \cdot L^{-1}$) in the rehydration solution (150 % BM loss) following dehydration equivilent to 2 % BM loss, through intermittent cycling in the heat ($32^{o}C$). Fluid was ingested during a 30 min period following dehydration, and then fluid balance was measured over the following 5.5 h. Both low sodium drinks failed to fully restore TBW within 5.5 h (2 $mmol \cdot L^{-1}$; 66 %; 26 $mmol \cdot L^{-1}$: 82 %). In contrast, both the moderate (52 $mmol \cdot L^{-1}$) and high (100 $mmol \cdot L^{-1}$) sodium drinks restored 100 % of BM losses.

High Sodium Concentrations

Systematic evaluations suggest that the amount of fluid retained is inversely related to a drink's sodium concentration between 0-100 $mmol \cdot L^{-1}$ (Maughan & Leiper, 1995; Shirreffs & Maughan, 1998). Figure 2.1. illustrates the effect of sodium concentration on U_{Vol} during recovery from exercise with the ingestion of varying concentrations of sodium between 0-100 $mmol \cdot L^{-1}$. Thus stronger concentrations of sodium may be more beneficial to restoration of fluid balance. Rarely have high sodium concentrations been investigated, largely because they tend to make drinks unpalatable.

To avoid the negative conotations related to the palatability of the solution Nose and colleagues (1988c) had participants ingest sodium capsules. They reported greater restoration of P_{Vol} (178 vs. 74 %) and fluid volume (71 vs. 51 %) with ingestion of a sodium capsule in water (77 $mmol \cdot L^{-1}$) compared with water + placebo (Nose *et al.*, 1988c). However drinking was *ad libitum* and significantly more fluid was consumed in the high sodium trial. Kenefick *et al.* (2000) reported greater restoration of P_{Vol} with ingestion of 0.45 % NaCl (~77 $mmol \cdot L^{-1}$) solution compared with no fluid following exercise-induced dehydration of 4-5 %. The most significant of these studies was conducted by Nielsen *et al*, (1986). This study found that consumption of just 100 % BM losses of a high sodium concentration (127 $mmol \cdot L^{-1}$), resulted in 90 % fluid restoration after just 2 h of recovery from 2.3-4.8 % BM loss. In contrast ingestion plain water resulted in < 50 % restoration.

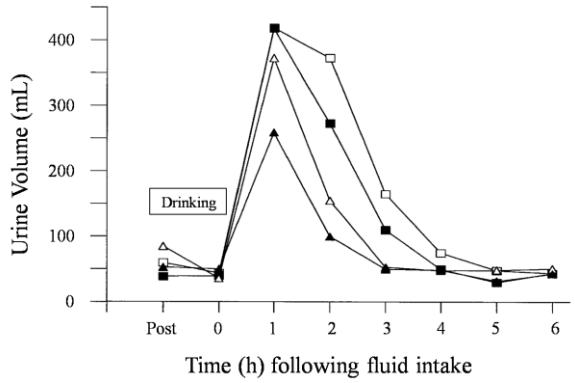

Figure 2.1. The effect of sodium concentration on urine loss. 1.9-2.0 L of drinks containing various sodium concentrations (□ = 0 $mmol \cdot L^{-1}$ ■ = 25 $mmol \cdot L^{-1}$ △ = 50 $mmol \cdot L^{-1}$ ▲ = 100 $mmol \cdot L^{-1}$) were ingested during the 1 h period between exercise and the start of recovery. Urine volume was measured at the end of each hourly period. Note Modified from Shirreffs, S.M., and R.J. Maughan (1998). Volume repletion after exercise-induced volume depletion in humans: replacement of water and sodium losses. Am. J. Physiol. 274: F868-F875.

Plasma Volume

The majority of studies have focussed on whole body restoration, with less attention given to measurement of intra- and extra-cellular spaces (Mitchell *et al.*, 2000). A reduction in P_{Vol} is a critical factor in the impairment of cardiovascular functioning and thermoregulation. Hypovolemia is associated with a reduction in CBV

(Costill, 1977; Fortney *et al.,* 1981a; Gauer *et al.,* 1956; Grande *et al.,* 1958; Ladell, 1955; Pearcy *et al.,* 1956) which compromises venous return (Fortney *et al.,* 1981a; Nadel *et al.,* 1980b; Saltin, 1964) decreasing SV, via the Frank-Starling mechanism (Fortney *et al.,* 1981a; Hopper *et al.,* 1988; Kanstrup & Ekblom, 1982; Krip *et al.,* 1997; Mier *et al.,* 1996; Nadel *et al.,* 1980b; Nose *et al.,* 1994; Saltin & Stenberg, 1964; Warbuton *et al.,* 1999).

The presence of sodium ions in a solution will influence where fluid is distributed among body-fluid compartments, with a preference for extracellular expansion. Accordingly, several studies have reported greater P_{Vol} restoration with sodium drinks compared to water (Costill & Sparks, 1973; Nielsen *et al.,* 1986; Nose *et al.,* 1988c). For example, Costill & Sparks (1973) observed that following thermal dehydration (4 % BM), rehydration with a volume equal to fluid losses of a CHO-E drink (11 % glucose, 22 $mmol \cdot L^{-1}$ Na) restored P_{Vol} faster than the same volume of plain water (-3.5 vs. -7.5 %). This preferential restoration of P_{Vol} is a result of the strong osmotic effect of elevated P_{Na}. However, neither the water nor low sodium drink fully restored P_{Vol} within 3 h (Costill & Sparks, 1973). In contrast, following exercise-induced dehydration (2.3-4.8 %), others have reported complete restoration of P_{Vol} with a low sodium dose (20-25 $mmol \cdot L^{-1}$) within 2 h (Ismail *et al.,* 2007; Nielsen *et al.,* 1986). The failure to achieve full P_{Vol} restoration in the study by Costill & Sparks (1973) is possibly because participants completed four 5 min treadmill runs throughout recovery. Posture is an important determinant of P_{Vol}, and moving from a seated to a standing position is known to decrease P_{Vol} (Carter & Gisolfi, 1989), this requirement may have prevented P_{Vol} returning to baseline levels.

Restoration of P_{Vol} appears to be proportionate to the sodium concentration of the solution. Mitchell *et al.* (2000) reported a main effect for sodium concentration in P_{Vol} restoration, and a trend for greater restoration with a 50 $mmol \cdot L^{-1}$ compared with a 25 $mmol \cdot L^{-1}$ solution. In fact, with the 50 $mmol \cdot L^{-1}$ solution, P_{Vol} actually reached positive levels during rehydration from ~7.5 % P_{Vol} loss (2.9 % BM loss).

2.2.2.2. Milk

In addition to sodium, there are a number of other drinks which may provide a rehydrative advantage during recovery from exercise. Milk is a one candidate with

potential as an effective post-exercise solution, given its naturally high electrolyte content and a presence of carbohydrate in a concentration similar to many commercially available sports drinks. Shirreffs *et al.* (2007) found that cumulative U_{Vol} during 5 h recovery from 1.8 % BM loss, was significantly reduced following the ingestion of milk (39 $mmol \cdot L^{-1}$; 611 mL urine) and milk with added electrolytes (50 $mmol \cdot L^{-1}$; 550mL urine) compared with rehydrating with either water (0.3 $mmol \cdot L^{-1}$; 1184 mL urine) or a regular CHO-E drink (23 $mmol \cdot L^{-1}$; 1205 mL urine) with a volume equal to 150 % BM loss. As a result participants remained in net positive fluid balance throughout recovery in the milk trials but returned to net negative fluid balance 1 h after drinking the other drinks.

Milk, however, may not be highly desirable after exercise. Firstly, sports drinks are a much more acceptable rehydration drink in terms of social association. People are just as highly likely to choose a drink which conforms to social norms and/or which is associated with sport. In addition, athletes may also be discouraged from ingesting milk because it evokes enhanced feelings of stomach discomfort, compared with a sports drink for example. Milk has a higher energy density than both water and the sports drink largely due to the presence of protein and fat in addition to the carbohydrate. Energy density is one of the most important variables that regulate the rate at which ingested solutions empty from the stomach (Vist & Maughan, 1995; Calbet & MacLean, 1997). It is therefore likely that milk will be emptied from the stomach at a slower rate than a regular sports drink or water, which may have implications for gastric comfort. Indeed Shirreffs *et al.* (2007) reported from a subjective feelings questionnaire that participants felt greater sensations of stomach fullness after ingesting milk than at the same time-points after ingesting a CHO-E drink.

2.2.2.3. Coconut Water

Coconut water has been commonly used as an oral rehydration solution in patients with diarrhoea (Chavalittamrong *et al.*, 1982). However, just one study has investigated the potential for coconut water to form an effective post-exercise rehydration solution. Saat *et al.* (2002) found that coconut water (5 $mmol \cdot L^{1}$ Na, 53 $mmol \cdot L^{-1}$ K) was as effective as both water and a low sodium CHO-E drink (19 $mmol \cdot L^{-1}$) in restoring fluid balance following exercise-induced dehydration of 2.8 %. The authors claimed that the coconut water "caused less nausea, fullness and no stomach

upset and was also easier to consume in large volumes" that plain water or the CHO-E drink (Saat *et al.*, 2002; p93). Despite these findings research is limited regarding the effectiveness of coconut water as a practical post-exercise rehydration solution.

2.2.2.4. Food

Many of the studies investigating rehydration from exercise have lasted between 3-6 h. In real life situations it would be expected that an athlete would look to consume a solid meal meal in this time. Maughan *et al*. (1996) compared the effects of ingesting fluid alone and fluid plus a meal following exercise induced dehydration equivalent to 2.1 % BM loss. Over a 60 min period, beginning 30 min after exercise, participants ingested a CHO-E drink (21 $mmol \cdot L^{-1}$ Na) or a standard meal (63 $Kj \cdot Kg^{-1}$ BM; ~42 $mmol \cdot L^{-1}$ Na; plus low sodium drink (1 $mmol \cdot L^{-1}$ Na). Fluid intake was equal to 150 % BM loss. Cumulative U_{Vol}, measured during a recovery period lasting 6 h, was significantly lower following food+fluid ingestion (665 mL, compared with following each of two CHO-E trials (934 & 954 mL). As a consequence, 6 h after exercise, participants in the food+fluid trial had a net fluid balance similar to their pre-exercise euhydration state, whilst those participants in the two CHO-E trials had a negative net fluid balance of 373 & 337 mL. However, the sodium content of the two interventions was unequal. The reduced U_{vol} can be attributed to the higher sodium content ingested in the food+fluid trials compared with the CHO-E trials (63 vs. 42 $mmol \cdot L^{-1}$).

From a practical perspective these findings suggest that the addition of electrolytes to the ingested fluid, a factor known to detract from the taste of drinks when included at high concentrations, is not necessary if solid food with an appropriate sodium content is consumed together with plain water. However, in situations which may cause gastrointestinal discomfort, such as when when limited time is available before the next event/session, rehydrating with food plus fluid may not be a viable option.

2.2.3. Recovery Protocol

Some discrepancies between studies regarding the optimal rehydration solution and volume occur because of differences in methodological approaches to rehydration from exercise. The majority of research examining the influence of sodium content of

rehydration has been conducted by the same group of researchers; Maughan, Shirreffs and colleagues (Maughan & Leiper, 1995; Maughan *et al.*, 1994; 1996; Shirreffs *et al.*, 1996; Merson *et al.*, 2008). These studies employ a recovery protocol which involves the ingestion of large volumes of fluid (2-3 litres) in a relatively short (30-60 min) period of time, followed by a period of 5-6 h of observation. Typically in these studies, positive fluid balance is induced immediately following the ingestion period, as a result of U_{Vol} < 50 mL with all drinks. These small urine losses are surprising given the large volume of fluid ingested. It is likely that there was a time-delay between ingestion of fluid, U_{Vol} and BM measurement that did not allow for accurate attainment of fluid balance, with the possibility of some of the fluid ingestion remaining within the stomach. During the subsequent hours of observation net fluid balance retracts as urine production increases by as much as 1.2 L over a 6 h period (Shirreffs & Maughan, 1998). The longer the observation period persists the greater the decreases in fluid balance.

The advantage of employing this type of rehydration strategy is that it provides a complete picture of the rehydration process over a number of hours. However, relatively little differences exist in U_{Vol} or fluid balance have occurred between different rehydration drinks during the last 2-3 h of these protocols. Further, it is unlikely that an individual would not seek to ingest food within 5-6 h of exercise. Indeed the same authors have reported this to be advisable, compared with ingestion of fluid alone (Maughan *et al.*, 1996). Thus this length of protocol may not be necessary following exercise-induced dehydration. Furthermore, the one-off ingestion of a large volume of fluid does not represent typical ingestion patterns following exercise. In addition, the ingestion of large volumes of fluid, even in dehydrated participants, rapidly decreases AVP before P_{Vol} and P_{Osm} are restored (Figaro & Mack, 1997); thus inducing an increase in urinary output.

Instead, the ingestion of fluid in serial feedings represents a more practical rehydration scenario and may avoid the possibility of stomach discomfort as a result of extreme fullness. Several other studies (e.g. Mitchell *et al.*, 1994; 2000; Ismail *et al.*, 2007) have employed such recovery protocols in which participants ingest smaller volume of fluid in serial feedings over a period typically lasting1-3 h. It is also possible that this method of rehydrating is advantageous over the one-off drinking routines. Mitchell *et al.* (2000) was able to demonstrate positive fluid balance within 3 h of dehydration following the consumption of 150 % fluid losses of a 25 $mmol \cdot L^{-1}$ solution

in 6 feedings, once every 30 min. In contrast, ingestion of the same percentage of fluid with the same sodium concentration (23-25 $mmol \cdot L^{-1}$) using the one-off drinking protocol did not induce positive fluid balance 3 h post ingestion (Shirreffs & Maughan, 1998; Maughan & Leiper, 1995; Maughan *et al.*, 1996).

2.2.4. Summary

There is no clear consensus as to the optimum sodium concentration for a rehydration solution. However, the available evidence suggests a positive relationship between sodium concentration and the rate of fluid rehydration following exercise-induced dehydration [EID]. As yet only once has a high sodium concentration been measured and therefore more research is needed in this area. The most practical strategy for fluid replacement following exercise-induced dehydration involves periodic ingestion of small volumes of fluid at regular intervals. This strategy promotes a steady decrease in negative fluid balance, which can be eradicated within 3 h of exercise, if the sodium concentration is sufficient. It avoids the stomach discomfort and volume-related diuresis which often accompany one-off drinking strategies.

Whilst several researchers have concluded that the optimal volume to be ingested is at least 150 % of body fluid losses, much of this research has been based on one-off drinking strategies, and has rarely employed high sodium concentrations. This thesis does acknowledge that a volume in excess of 100 % body fluid losses is necessary to promote positive fluid balance, because of continued urinary losses during recovery. Given the strong negative relationship between drink sodium concentration and urinary losses, it is feasible that a higher sodium concentration would reduce the volume of ingested fluid necessary to achieve positive fluid balance. It is not entirely surprising that drinks containing a high sodium concentration have not received extended investigation since the inclusion of such a sodium content will render rehydration solution less palatable. This is an important consideration when designing a sports drink and will be the focus of the next section of this literature review.

2.3. Drinking Behaviour

Since fluid intake during and after exercise rarely matches fluid losses during exercise, every effort should be made to provide access to fluid which will encourage its consumption to the greatest degree. As rigid drinking formulas are not representative of

common practice following exercise, it is somewhat surprising that relatively little is known about drinking behaviour at this time. The inclusion of sodium in a rehydration solution has been purported to increase fluid retention and improve rehydration (Shirreffs & Maughan, 1996; Mitchell *et al.,* 2001), yet there are clearly palatability issues that influence the formulation. Given that a drink that tastes good contributes more to voluntary hydration than a drink which tastes unpleasant, palatability is an important factor in the process of understanding drinking behaviour (Maughan, 1998; Minehan *et al.,* 2002).

It would be a simple test to find which drinks are preferable after exercise. However, what is more interesting is discovering whether there is a change in the relative palatability of a drink after exercise compared with before exercise. i.e. would a drink be more or less palatable after exercise than before? This would give us a better understanding of the role exercise plays in changing drinking behaviour. Surely the length of exercise, or more likely the level of dehydration incurred will be a significant variable affecting palatability. Understanding this may help determine the optimum drinking strategy following various athletic events.

2.3.1. Physiological Usefulness Theory

The sense of taste acts as a vigilant gatekeeper to the body (Cowart, 2005), providing protection from what is harmful and encouraging the consumption of food/fluid items which satisfy physiological need (Cabanac, 1971). It is a transitory system which is determined not only by the physiological and chemical properties of a food/fluid, but also by the physiological characteristics of the individual consuming it (Rogers, 1999; Yeomans, 1998). Changes in taste palatability have been demonstrated to respond to differences between individuals, dependent on physiological characteristics such as age, weight and body status (de Graaf & Zandstra, 1999; Drewnowski *et al.,* 1985; Tepper & Selder, 1999); and within individuals dependent on short-term disturbances to homeostasis such as hunger (Yeomans, 1998), satiety (Rolls *et al.*, 1980), hyponatraemia (Beauchamp *et al.,* 1990; Huang & Yan, 2008), and following dietary manipulation (Bertino *et al.,* 1982) and exercise (Appleton, 2005; Bacon *et al.,* 1962; Lesham *et al.,* 1999; Rolls *et al.,* 1981; Takamata *et al.,* 1994).

Related to short-term physiological situation, it has previously been suggested that the perceived pleasantness of a food or fluid item may be directly related to the

'physiological usefulness' of that item to the consumer (Cabanac, 1971; 1992). According to Cabanac (1971), "a stimulus can feel pleasant or unpleasant depending upon its usefulness as determined by internal signals" (p. 1103). This theory of physiological usefulness suggests that any food/fluid which the body deems physiologically useful in a specific physiological situation will be perceived as more palatable (Appleton, 2005). The theory also holds that once that situation no longer exists, the food/fluid is rendered less palatable (Cabanac, 1992). In a physiological situation of salt deficiency, for example, salty foods are expected to be perceived as more palatable than in a situation of no salt requirement. Similarly, in a physiological situation of high fluid requirement, fluids are expected to be perceived as highly palatable; once fluid requirements, however, are satisfied, the perceived palatability of fluids would reduce.

The theory is supported by a finding that the changes in pleasantness of food items is specifically related to nature of the physiological disturbance, for example, when sodium deprived, humans display a specific appetite for sodium (Bertino *et al.,* 1982), not for other salts, such as potassium, even if the cation is coupled with the same anion. Further evidence is the interesting finding that the hedonic values of other, normally rewarding stimuli, such as sugar, appear to decrease following sodium loss, in concert with the increasing appeal of sodium, (McCance, 1936; Grippo *et al.,* 2006). In fact, the normal preference for sugar over salt reverses during sodium deficiency, so that rats will ingest more saline than glucose or other sugary solutions (Nozaki *et al.* 2002). Further, Conover *et al.* (1994) observed that sodium-deprived rats choose the taste of salt over moderate intensities of directly rewarding brain stimulation.

2.3.2. Exercise-induced dehydration

Exercise-induced dehydration resulting from profuse sweating is one such situation which has been demonstrated to influence taste perceptions (Appleton *et al.,* 2006; Lesham *et al.,* 1999; Takamata *et al.,* 1994). The shifts in fluid balance as a result of dehydration induce a change in the body's physiological needs and therefore a different combination of nutrients carry a greater hedonic reward.

Sweat losses during exercise induce, not just a loss of fluid but significant losses of salt as well. This results in a state of both fluid *and* sodium deficiency, both of which need to be addressed following exercise. When fluid and sodium losses are severe, as

commonly observed during intense exercise in the heat without adequate fluid replacement, sweat losses promotes a specific type of dehydration known as hyperosmotic hypovolemia. Since plasma provides the majority of the fluid for sweating, hypovolemia ensues. Since sweat is hypotonic to plasma, it causes a greater relative loss of fluid than electrolytes, resulting in hyperosmolality. Thus the regulation of fluid balance is not simply a function of restoring fluid balance but restoring both of P_{Vol} and a start of osmotic equilibrium.

2.3.2.1. Automated Responses to dehydration

In response to disturbances to homeostasis, such as hypovolemia and hyperosmolality, the body initiates a series of automated and behavioural responses designed at restoring balance as rapidly as possible. Even in benign environments, the major components of body fluids are in constant flux (Johnson, 2007), thus the body has developed reflex mechanisms which respond to small fluctuations in fluid balance. Sympathetic reflex mechanisms occur within seconds of fluid loss, but have limited scope and serve only to maintain cardiovascular functioning, through vasoconstriction or vasodilation (Guyton & Hall, 1996).

The release of the endocrine hormones, AVP and aldosterone are stimulated within minutes of fluid imbalance (Johnson & Thurnhorst, 1997) and are involved in a complex process to restore fluid balance. In response to elevated P_{Osm} and hypovolemia, AVP is secreted from the posterior pituitary into the bloodstream in increased amounts (Daniels & Fluharty, 2008; Moses & Miller, 1971). The primary purpose of AVP is to promote antidiuresis (Bourque & Oilet, 1997; Bourque *et al.*, 1997), by increasing the permeability to water of the distal convoluted tubules in nephrons of the kidneys. This encourages greater water re-absorption, from urine back into the bloodstream.

The amount of sodium in ECF compartment must be kept within narrow limits despite a constant exchange with the environment (Conteras & Frank, 1979). Following sodium loss, and in response to both changes in fluid volume and stimulation of osmoreceptors (Andersen *et al.*, 1998; Bie & Sandgaard, 2000), activation of the RAS increases secretion of the sodium retaining hormone, aldosterone (Maughan & Leiper, 1995; Maughan *et al*., 1996; Nose *et al*., 1988a; Takamata *et al*., 1994). Aldosterone, principally an anti-natriuretic hormone (DiBona 1985), reduces sodium levels in urine, sweat and saliva (Castenfors, 1967; Thongboonkerd *et al.*, 2003).

2.3.2.2. Behavioural Responses

Renal mechanisms act to conserve fluid and electrolyte losses from the body, but are limited (Fitzsimons, 1998; Hew-Butler *et al.*, 2006) and complete restoration of fluid balance requires the behavioural mechanisms; thirst and sodium appetite ([SA] Johnson & Thurnhorst, 1997; Johnson, 2007).

Thirst

Thirst is a motivational state characterised by a deep-seated desire for water (Robertson, 1984; Stricker & Verbalis, 1988, Fitzsimons, 1998). Although thirst is associated with oral sensations such as dryness and irritation (Rolls *et al.*, 1980; Phillips *et al.*, 1985), a dry mouth is not normally the primary stimulus for thirst. This explains why wetting the mouth does not by itself reduce thirst (Bellows, 1939). Instead thirst results from either intracellular (due to increased P_{Osm}) or extracellular (due to hypovolemia), dehydration mediated by a complex pattern of neural activity in the hypothalamus and higher regions of the cortex (Grossman, 1979).

Hyperosmotic-Thirst

The most potent stimulus of thirst is an elevation in the P_{Osm}, and consequent cellular dehydration, (Antunes-Rodriguez *et al.*, 2004). In response to hyperosmolality, AVP secretion stimulates thirst (Szczepanska-Sadowski, 1991), which works together with antidiuresis to reduced plasma hyperosmolality. The stimulation of thirst and antidiuresis requires the same afferent input, but is initiated at different latencies; the threshold for antidiuresis (~280 to 295 $mOsm \cdot kg^{-1}$) normally occurs 5 to 10 $mOsm \cdot kg^{-1}$ below the threshold for thirst (~290 to 295 $mOsm \cdot kg^{-1}$; Robertson, 1984). This evolutionary design prevents individuals from constantly seeking water. Thirst is only stimulated once antidiuresis reaches its maximum capabilities (Hew-Butler *et al.*, 2006), *or* at least a significant point (Rolls *et al.*, 1980).

Hypovolemic-Thirst

Changes in P_{Osm} are insufficient to explain all the phenomena of thirst alone. Hypovolemia is capable of inducing thirst without changes in P_{Osm} (Saltmarsh, 2001; Fitzsimons, 1998) detected by stretch receptors in the walls of the heart and vasculature (Gauer & Henry, 1963), and possibly by other receptors as well (Fitzsimons & Moore-Gillon, 1980; Johnson & Thurnshorst, 1997). For example, Sagawa *et al.* (1992) demonstrated that dehydration-induced drinking and thirst sensation were significantly attenuated during water immersion, suggesting that central blood volume has a significant effect on the thirst.

One study perfectly depicts the changing causes of thirst during recovery from exercise. Following 7 h intermittent exercise in the heat (35^{o}C), which induced a BM loss of 2.7 %, a P_{Vol} loss of ~6 % and a P_{Osm} increase of ~4 mOsm·kg^{-1}, Takamata *et al.* (1994) reported increased ratings of thirst. During the first few hours following exercise, thirst was associated with hyperosmolality, but not hypovolemia, since *ad libitum* ingestion of plain water, reduced P_{Na} and P_{Osm}, and alleviated thirst, whilst P_{Vol} remained at negative 5-6 %. Rehydration with plain water continued *ad libitum* for 23 h following exercise. Between 3-17 h, thirst remained at baseline levels whilst P_{Na} and P_{Osm} steadily fell. After 17 h, an increase in thirst was associated, not with hyperosmolality but with hypovolemia, since P_{Osm} was reduced from baseline and P_{Vol} was still negative 5-6 %.

Osmotic-thirst appears to occur long before volemic-thirst. This is due to the far narrower range within which P_{Osm} is maintained; an increase in P_{Osm} of as little as 1-2 % is a potent stimulus for thirst (McKinley & Johnson, 2004), whilst changes in blood volume of 8-10 % is necessary before thirst is stimulated by this method alone (Saltmarsh, 2001; Stricker *et al.*, 1992; Antunes-Rodrigues *et al.*, 2005; Fitzsimons, 1998; Kimura *et al.*, 1976). The considerably greater hypovolemic than osmotic threshold is because the range of blood flow rates for different activities is so extended and the scope for change in $\dot{Q}$ so great (Fitzsimons, 1998) that it is necessary to avoid the controls of P_{Vol} being triggered by small changes. An ample reserve of fluid in the interstitial fluid compartment acts as a buffer for P_{Vol}, and can be mobilised should the need arise. Therefore, while extracellular volume depletion stimulates thirst (Ramsey *et al.*, 1977a; 1977b) hyperosmolality appears to be a more potent stimulus (Moriomoto *et al.*, 1981; Nose *et al.*, 1985; 1986).

This pattern of drinking behaviour following exercise is an important consideration when assessing palatability. Reductions in elevated P_{Osm} are restored most rapidly by an influx of low osmolality fluid (Fitzsimons, 1998), and this requirement is reflected by the endocrine and behavioural responses. Several studies have reported preference for low osmolality fluids over high osmolality fluid immediately after exercise (Appleton, 2005; King *et al.,* 1999; Takamata *et al.,* 1994). It is thought that low osmolality fluids are rated more palatable since they satisfy the sensations of thirst to a greater extent, than high calorie/salty drink.

Whilst it is significant that low osmolality fluids are rated more preferable following exercise, than high osmolality fluids, it is perhaps more important to consider the change that occurs as a result of exercise, i.e. why is low osmolality fluid more palatable after exercise than before. An increase in palatability for plain water but not for ten higher concentrations of sodium in water was demonstrated immediately after dehydration ~2.7 % BM loss, induced by 7 hr intermittent exercise in the heat (35^{o}C; Takamata *et al.,* 1994). Further, when thirst declined during the first hour after exercise, the palatability rating of water also declined. Additionally, following dehydration of just ~0.7 % BM loss, an increased palatability for three hypotonic sports drinks (22-230 $mOsm{\cdot}kg^{-1}$), but not two hypertonic sports drinks (418, 489 $mOsm{\cdot}kg^{-1}$) was demonstrated following 50 min of gym exercise (Appleton, 2005). These findings are perhaps more significant since sports drinks were used. Thus the demonstration of an increased preference for hypotonic drinks occurred despite a greater concentration of both artificial and natural sweeteners. However, the study was poorly controlled: as it was conducted in a public gymnasium, without pre-tests standardization and with no control of drink taste intensity. Furthermore, the change in palatability, after dehydration equivalent to just 0.7 % BM loss (Appleton, 2005) was surprising given that osmotic thirst is not normally stimulated until a sweat loss of ~1.7 % to 3.5 % body water (Robertson *et al.,* 1984). It is possible that the 'regular gym users' employed in that study were more sensitive to fluid losses than a trained athletic population. The need for a more robust investigation of the influence of osmolality on palatability is warranted.

Sodium Appetite

In addition to restoring fluid, EID is also associated with a loss of sodium in sweat which also needs replacing. The desire to replenish this imbalance is known as sodium appetite, and is characterised by an increased demand, or decreased aversion for salty foods (Conteras & Frank, 1979; Stricker *et al.*, 1979). Whilst thirst is preceded by antidiuresis, sodium appetite is itself preceded by the conservation of sodium from urine known as antinatriuresis. In the same way the antidiuresis acts only to conserve fluid losses, antinatriuresis is also limited as it can only adapt to retain what is already present in the body. Complete restoration of ECF volume expansion requires the consumption of sodium. Sodium appetite is a motivated behavioural state that arises in a number of species specifically as a response to sodium deficiency. It drives an animal to seek and ingest foods and fluids that contain sodium. It acts as a regulatory mechanism and, like thirst, it is vital for restoring extracellular fluid.

In times of sodium need taste plays a fundamental role in identifying cations[1] to resolve the ionic deficit. Humans exhibit an exaggerated preference for sodium following both chronic and acute states of fluid and/or sodium loss (Beauchamp *et al.*, 1990; Blackburn *et al.*, 1993; Farleigh *et al.*, 1987; Lesham & Rudoy, 1997; Lesham *et al.*, 1999; McCance, 1936; Richter, 1936; Shepherd *et al.*, 1986; 1987; Takamata *et al.*, 1994). It is typified by decreased sensitivity to sodium containing foods and/or an ability to ingest higher concentrations normally not tolerated. Interestingly, and specific to salt, humans do not display an increased preference for salt *per se*, not in the same way that we do for water, when thirsty, but for foods containing salt.

With sweat sodium concentrations on average between 20-80 $mmol \cdot L^{-1}$ (Costill, 1977; Costill *et al.*, 1976; Maughan, 1991; Rehrer, 2001; Verde *et al.*, 1982), exercise-induced sweating is capable of inducing sodium depletion at rates of about 120-180 $mmol \cdot h^{-1}$. Sodium losses are primarily from the extracellular fluid compartment leading to a loss of plasma and blood volume. Thus sodium ingestion plays a fundamental role in the restoration of P_{Vol}. To date, only two studies have investigated the effects of exercise on sodium appetite (Lesham *et al.*, 1999; Takamata *et al.*, 1994).

[1] Cations, are positively charged ions such as sodium, whilst anions, are negatively charged ions such as chloride.

Takamata *et al.* (1994) investigated the long-term effects of EID on fluid balance and taste preference during a 24 h period following fluid losses of 2.7 % BM loss and sodium losses of ~230 mmol, induced over 7 h intermittent exercise in the heat (35^{o}C). During rehydration, participants drank deionised water *ad libitum* and ate a salt free diet. Whilst an increase in thirst was demonstrated immediately following exercise, an increase in palatability of sodium was not demonstrated until 3 h post exercise. Interestingly, this occurred at the same point at which elevated P_{Osm} (289 mOsm·kg^{-1}) which occurred during exercise had returned to baseline (286 mOsm·kg^{-1}). The failure to demonstrate a SA occurred despite hypovolemia of -6 % during the first 3 h following exercise. Following 3 h post-exercise, P_{Na} and P_{Osm} continued to decrease with ingestion of plain water below baseline levels. This was associated with a reduction in plasma AVP, and thirst and an increase in U_{Vol}, and palatability of increasingly higher concentrations of sodium. Preference for the highest concentrations of sodium peaked between 17-23 h following exercise, at which point, P_{Na} and P_{Osm} were lowest.

A delayed onset of SA is a consistent theme that has emerged from SA studies (Takamata *et al.*, 1994; Sugimoyto, 1987; Yawata *et al.*, 1987). Whilst thirst is stimulated within minutes (Antunes-Rodrigues *et al.*, 2005; Johnson & Thurnhorst, 1997) typically neither anti-natriuresis nor SA occur until several hours after sodium loss (Ferreyra & Chiaraviglio, 1977). This delay is certainly robust; even after Furosemide[2] treatment SA does not occur immediately (Fitts *et al.*, 2007). The delay is considered a result of the body protecting P_{Osm} over P_{Vol} regulation (Hew-Butler *et al.*, 2006). Not until P_{Osm} is stabilised should P_{Vol} restoration begin (Johnson, 2007). Even if P_{Vol} is restored, following fluid loss, if P_{Osm} remains elevated, overall fluid balance of both the intracellular fluid compartment [ICF] and ECF will remain unstable (Johnson, 2007). For example, a 2-3 % dilution of elevated P_{Osm} will eradicate water intake in rats even with 35 % P_{Vol} deficits (Stricker, 1966). In contrast, P_{Vol} expansion does not inhibit drinking response to osmoregulatory thirst (Fitzsimons, 1961). Thus immediately after exercise, restoration of P_{Osm}, not P_{Vol} is the primary objective (Fitzsimons, 1998; Geerling & Loewry, 2008; Johnson, 2007).

[2] ; Furosemide, a diuretic commonly used in studies examining drinking behaviour, which produces high levels of RAS activity within a few minutes.

The level of sodium depletion induced in the study of Takamata *et al.* (1994) was not representative of a normal exercise sodium loss. The protocol, conducted in the heat, experimentally accelerated sodium loss with the ingestion of plain water. It is not known whether the same responses would be evident with typical sodium losses during exercise. The second study which has investigated SA following exercise was conducted with just 1 h of exercise in a thermo-neutral environment, which the authors claimed sodium loss was 'mild' (Lesham *et al.,* 1999). Interestingly sodium appetite, as measured by the amount of salt participants added to soup, was demonstrated within 30 min of exercise ceasing. Salt intake increased by 50 %, compared to baseline, whilst there was no change in the control group or in preference for sugar in tea. The demonstration of SA within 30 min of mild sodium loss suggests the level of sodium loss is critical in the timing of sodium appetite. Unfortunately no measurements of P_{Na} or P_{Osm} were collected. Further, the method for assessing sodium appetite was unreliable and open to confounding factors such as social stigma. Clearly, there is a need for a more robust investigation of sodium appetite following exercise-induced dehydration and mild sodium loss.

2.3.3. Summary

Following sustained sweat losses the body initiates a series of automated and behavioural measures to restore fluid balance. Antidiuresis and antinatriuresis act to conserve fluid and sodium losses, respectively. However, they are limited in their capacity and complete restoration of fluid balance requires the behavioural responses, thirst and SA. These responses are initiated at different latencies in order to maintain optimal regulation of fluid balance. For example, immediately after exercise, the primary goal appears to be the restoration of P_{Osm}, over P_{Vol}. Evidence of this is found from both the automated and behavioural responses to the situation, wherein we find an increase in the antidiuretic response, and an increase in thirst, and a delayed onset of antinatriuresis and SA. The specificity of these regulatory behaviours is such they must be considered the optimal solution to restoring fluid balance. Knowledge of gustatory behaviours following exercise can give an accurate interpretation of what is 'physiologically useful' and as such could be used as a guide to ingestive practices.

2.4. Rehydration interventions during exercise

A number of strategies have been employed to reduce thermoregulatory and cardiovascular strain and enhance performance during exercise in the heat. These have tended to involve interventions being applied during exercise such as rehydrating with either water or a carbohydrate-electrolyte sports drink. A wealth of research, over the past 75 years has been devoted to examining the benefits of rehydrating during exercise, with the purported benefits being an improvement in thermoregulation and cardiovascular functioning. In some cases an improvement in performance has also been demonstrated.

A classic study by Pitts *et al.* (1944) was one of the first to demonstrate an inverse relationship between fluid intake and T_{Rec} during exercise. Since then more accomplished studies have found that when exercising in the heat (>30^{o}C), increases in both T_{Core} and HR and reductions in SV and $\dot{Q}$ are directly related to the magnitude of fluid deficit (Hamilton *et al.,* 1991; Montain & Coyle, 1992a; Sawka *et al.,* 1979). One of the most cited of these studies was conducted by Montain & Coyle (1992a). They demonstrated a proportionate decrease in HR and T_{Rec} and increase in SV and $\dot{Q}$ with increasing fluid restoration (0, 20, 48, 81 % BM losses) during 2 h moderate intensity exercise in the heat (60 % $\dot{V}O_{2max}$; 33^{o}C, 50 % RH). Studies like this have led fluid replacement guidelines to ascertain that athletes should be encouraged to drink 80-100 % fluid losses during exercise (Coyle, 2004).

Whilst the ergogenic benefits of fluid ingestion during exercise have been well documented, many athletic and occupational situations, however, make it unfeasible to ingest 80-100 % of BM losses during exercise (Convertino *et al.,* 1996) due to issues such as availability, and the circumstances of competition. For example, fire-fighters have limited provision for water whilst engaging fires; or footballers and cricketers having access only during specific stoppages in play. Moreover, it is not often possible for athletes to exercise with the large gastric volume needed to promote the high rates of gastrointestinal fluid absorption that are required to prevent cellular dehydration during exercise. Trying to offset dehydration, in athletes with high sweat rates may require exercising with a gastric volume of in excess of 0.6–1.0 litre of fluid. The associated discomfort of that added volume and weight, in runners in particular, will not reduce physiological stress if it remains in the gut towards the end of the event and some

individuals may not benefit by drinking the extra volume that ensures no body weight reduction at the end of exercise (Coyle, 2004).

2.4.1. Drink Composition

The composition of the drink has been the focus of much debate regarding the optimal fluid strategy during exercise. The primary goal of drinking during exercise is to prevent excessive dehydration (2 % BM loss) and changes in electrolyte balance (Sawka *et al.*, 2007).

2.4.1.1. Water

The ingestion of water during exercise has been demonstrated to attenuate or prevent progressive increases in HR, T_{Core}, and perceived effort and progressive declines in SV, and $\dot{Q}$ (Gonzalez-Alonso *et al.*, 1995; 1997), and improve endurance performance (Below *et al.*, 1995; Fallowfield *et al.*, 1996; McConell *et al.*, 1997; Pitts *et al.*, 1944), compared with no fluid ingestion.

Whilst there is a need to replace volume losses during exercise, restoration of P_{Vol} and P_{Osm} cannot be achieved without the ingestion of sodium. During constant exercise the most concentrated electrolyte lost through sweating is sodium (60-80 $mmol \cdot L^{-1}$). This corresponds to approximately 1.5 g of sodium for every litre of sweat. This loss in sodium concentration can have a severe impact on the human body, with one of the main problems being a reduction in P_{Vol}. The continued ingestion of large volumes of plain water alone results in a rapid fall in P_{Na} and P_{Osm} (Costill & Sparkes, 1973; Gonzalez-Alonso *et al.*, 1992; Nose *et al.*, 1988a; 1988b; 1988c); stimulating U_{Vol} reducing compartmental shifts from intracellular to extracellular fluid compartments, and delaying the rehydration process (Casa *et al.*, 1999; Gonzalez-Alonzo *et al*, 1992; Shirreffs & Maughan, 1998; Nose *et al.*, 1988a). Whilst the replacement of fluid losses, is important, for optimal restoration of fluid balance, the addition of sodium to a rehydration solution is paramount. The ingestion of sodium reduces the fall in P_{Osm} during exercise; maintaining fluid shifts from intracellular to extracellular fluid compartments, and hence sustaining the antidiuretic response to exercise.

If excessive the dilution of P_{Na} and P_{Osm} can promote dilutional hyponatraemia, a potential life threatening condition which is a common occurrence when "too much" plain water is ingested. In fact the ingestion of excessive water has occurred in almost every incident involving hyponatraemia (Hew-Butler *et al.,* 2005). Hyponatraemia does not just occur with athletes who grossly overdrink, but also with athletes who drink moderately more than they need but retain the excess that they would otherwise readily excrete at rest.

Normally excess water ingestion is not problematic, because the excess water is very rapidly excreted by the kidneys. However, kidneys are limited in their capacity to excrete fluid. Normal kidneys can excrete about 0.8 to 1.0 liters of water per hour in urine at rest (Noakes, 2001). When fluid ingestion exceeds the ability of the kidneys to excrete fluid, the retained water may dilute P_{Na} and P_{Osm} sufficiently that excessive fluid ingestion causes cells to swell. This is particularly problematic in the brain because the skull which allows very little room for expansion. Consequently the symptoms and illness of hyponatremia are those of brain dysfunction (hyponatremia encephalopathy): change in mental status, sensory distortion, confusion, incoordination, bizarre behavior; and ultimately seizures, coma and death. If salt is consumed in addition to water, however, the body can retain an isotonic mixture of ingested sodium and water to more effectively restore blood volume (Nose *et al.* 1988b; Twerenbold *et al.* 2003).

2.4.1.2. Carbohydrate-Electrolyte drinks

Carbohydrate-electrolyte drinks with similar concentrations as commercially available sports drinks (6-8 % carbohydrates; 9-25 $mmol \cdot L^{-1}$) have been demonstrated to enhance P_{Vol} restoration, reduce cardiovascular and thermoregulatory strain and improve exercise performance, compared with water alone (e.g. Barr *et al.,* 1991; Candas *et al.,* 1986; Carter & Gisolfi, 1989; Montain & Coyle, 1992b). During prolonged moderate intensity exercise in the heat (2 h; 60 % $\dot{V}O_{2max}$; 33°C) Montain & Coyle (1992b) ingestion of 100 % BM losses of a CHO-E drink restored TBW but not blood volume, attenuated the fall in SV, increased BF_{Skn} and reduced T_{Core}.

These findings do not elucidate whether the greater restoration of P_{Vol} was a result of electrolyte or carbohydrate content. Both electrolytes, and to a lesser extent carbohydrates can individually have an osmotic effect on fluid balance. However, Ryan

et al. (1989) reported greater P_{Vol} restoration with a 5 % CHO-E drink than a 5 % carbohydrate solution alone. Further, when sodium content was held constant, the addition of carbohydrates (100 $g \cdot L^{-1}$) had no effect on P_{Vol} over a 4 h period following 4 % BM loss (Lambert *et al.*, 1992), thus suggesting that sodium concentration as opposed to carbohydrate concentration is the critical factor in promoting P_{Vol} restoration during exercise with ingestion of a CHO-E drink.

Despite greater P_{Vol}, the ingestion of low sodium CHO-E drinks does not consistently improve either cardiovascular functioning or thermoregulation during exercise, compared with water alone. For example, Ishijima *et al.* (2009) found no differences in $\dot{V}O_2$ drift or HR_{Drift} with ingestion of either mineral water or a low sodium CHO-E solution in six untrained participants at the end of 90 min of cycling at 55 % $\dot{V}O_{2max}$ in a warm environment (28°C; 50 % RH). In just 5 untrained participants, Candas *et al.* (1986) reported similar HR with ingestion of 82 % BM losses of a CHO-E drink compared with water during 4 h intermittent exercise (50 % HR_{max}; 4 x 25 min cycling 5 min rest; 4 x 20 min cycling 10 min rest) at 34°C. CHO-E actually resulted in delayed onset of sweating, as a result of an elevated P_{Osm}. It should be noted that in both these studies a small and untrained sample was employed which may have contributed to the failure to achieve significant differences in HR between drinks. Untrained participants are more prone to interindividual variability. In seven participants, Carter & Gisolfi (1989) also reported no differences in T_{Rec} nor HR during 3 h exercise in the heat (60 % $\dot{V}O_{2max}$; 30°C), with CHO-E compared with water, although ingestion was *ad libitum*, and there was a trend for greater water intake. However, further studies employing larger populations (≥8) have also failed to find noticeable differences in HR between water and low sodium CHO-E drinks. For example, Costill & Sparks (1973) reported that replacement of 62 % BM loss of both water and a CHO-E drink resulted in similar HR and T_{Rec}. Barr *et al.* (1991) had 8 participants perform 6 hr exercise at 55% $\dot{V}O_{2max}$ in a heat (30°C), during which they ingested either no fluid, water, or sodium (25 $mmol \cdot L^{-1}$). Compared with water, the ingestion of sodium did not affect HR or T_{Core}. Further, Fritzsche *et al.* (2000) actually reported an increase in HR with ingestion of a CHO-E drink compared with water during prolonged exercise. Despite greater P_{Vol} restoration with sodium compared to water, the ingestion of low sodium concentrations (<25 $mmol \cdot L^{-1}$) does not result in complete attenuation in the fall in P_{Vol}, even with the ingestion of a volume equivalent to >80 % of BM losses (Barr *et al.*, 1991; Candas *et*

al., 1988; Costill & Sparks, 1973; Candas *et al.*, 1986; Owen *et al.*, 1986). Demonstration of significant differences in cardiovascular functioning and thermoregulation may require greater differences in P_{Vol} restoration.

Whilst ingestion of low-sodium CHO-E drinks does not completely restore P_{Vol} and does not always result in cardiovascular or thermoregulatory benefits, the ingestion of CHO-E drinks has often resulted in an improvement in exercise performance, compared with water (e.g. Coyle *et al.*, 1986; Khanna & Manna, 2005; Fritzsche *et al.*, 2000; McConnell *et al.*, 1999). This suggests that the mechanisms for the improvements in performance may possibly be by some means other than changes in cardiovascular or thermoregulatory functioning, such as by preventing hypoglycemia (Coggan & Coyle, 1988; Coyle *et al.*, 1986; 1983) or by another factor which remains unclear (Below *et al.*, 1995; Ivy *et al.*, 1979; Montain *et al.*, 1998). In these cases, the use of CHO-E solutions makes it difficult to interpret the separate effects of electrolyte replacement and substrate provision, since carbohydrate ingestion alone is capable of improving performance independently (Campbell *et al.*, 2008). However, the availability of glycogen appears only to be a critical factor in the development of fatigue during events lasting >1-2 h (Coyle & Montain, 1992) when inadequate supply of blood glucose is a possible cause of fatigue. Thus in these cases, the osmotic influence of carbohydrates, and predominantly electrolytes on P_{Vol} expansion is likely to result in a more significant impact on cardiovascular function than for example, the prevention of hypoglycemia.

2.4.1.3. High sodium ingestion

Complete restoration of P_{Vol} may require the ingestion of a greater electrolyte concentration. For example, with a possible sweat sodium concentration of 50 $mmol \cdot L^{-1}$ (average 20-80 $mmol \cdot L^{-1}$; Costill, 1977; Costill *et al.*, 1976; Maughan, 1991; Rehrer, 2001; Verde *et al.*, 1982), with ingestion of 80 % fluid intake, would require a drink sodium concentration of approximately 75 $mmol \cdot L^{-1}$ to maintain sodium balance during exercise. This represents a sodium concentration roughly 4-5 times as high as that found currently in most commercial sports drinks. Only a handful of studies have employed sodium concentrations as high as this, most likely because of the unpleasant taste of the drink.

Sanders *et al.* (2001) investigated the effects of ingesting three concentrations of sodium during 4 hr intermittent rest/cycling in a neutral environment (55% of $\dot{V}O_{2max}$; 20^{o}C). Participants replaced ~100 % of fluid losses (~3.85 L) of an 8 % CHO-E drink containing 5, 50, or 100 mmol·L^{-1} of sodium. Ingestion of the high sodium solution reduced total fluid lost during exercise in comparison to the other drinks, largely as a result of reduced diuresis. Calculation of water compartment changes revealed a significant loss of fluid from P_{Vol} (-6 %) in the low sodium trial; no change in P_{Vol} in the moderate sodium trial; and expansion of P_{Vol} (5 %) in the high sodium trial (Sanders *et al.*, 2001).

Despite elevated TBW and P_{Vol}, Sanders *et al.* (2001) reported no noticeable differences in cardiovascular or thermoregulatory variables between trials. Similar increases in both HR (~130-140 b·min^{-1}) and T_{Rec} (~37.8-38.4^{o}C) were reported in all three conditions. The authors suggest that the failure to demonstrate noticeable differences in HR between trials was because HR was not entirely due to changes in P_{Vol} during exercise.

The mechanism underlying CV_{Drift} have been open to much discussion, as yet with no discerning view prevailing (Coyle & Gonzalez-Alonso, 2001). The traditional hypothesis is that CV_{Drift} is caused by peripheral displacement of blood volume to augment evaporative heat loss, which results in the progressive fall in CBV, SV, and arterial pressure (Ekelund & Homgren, 1964; Johnson & Rowell, 1975; Rowell, 1986). Whilst this made good theoretical sence, more recent thinking is that cardiovascular functioning is not compromised by an increase in BF_{skn} since CV_{Drift} can occur independently of reductions in CBV (Montain & Coyle, 1992b; Saltin & Stenberg, 1964).. An alternatiove hypothesis is that an elevated blood temperature detected by the sinoatrial node (Gorman & Proppe, 1984; Johnson & Proppe, 1996) increases sympathetic nervous system activity, raising HR and thus decreasing ventricular filling time, end-diastolic volume, and SV (Coyle & Gonzalez-Alonso, 2001). This notion is supported by the findings of Fritzsche *et al.* (1999) which reported a strong correlation (r^2 = 0.95) between increases in T_{Core} and HR during CV_{Drift}. Similarly, Wingo and colleagues demonstrated substantial CV_{Drift} when exercise was performed at 35^{o}C but failed to demonstrate a significant change at 22^{o}C (Wingo *et al.*, 2005a; Wingo *et al.*, 2005b). Likewise both Wingo & Cureton (2006b) and Sharraff & Adams (1984) found that body cooling with fan airflow was successful in attenuating CV_{Drift}. Thus, the

failure to demonstrate any noticeable differences in HR between the control and high sodium condition may be because the exercise and environmental conditions were such that thermoregulation and therefore HR were not significantly impaired.

To date only two studies have investigated the effects of ingesting a high sodium concentration on performance. Sims *et al.* (2007a; 2007b) found that ingestion of a greater sodium concentration (156 $mmol \cdot L^{-1}$) induced P_{Vol} expansion of 4.3-4.5 % when ingested over an hour long period beginning 105-80 min prior to exercise. In contrast to the finding of Sanders *et al.* (2001), HR and T_{Rec} were significantly reduced with the ingestion of the high sodium concentration. Furthermore, time to exhaustion significantly increased by 21-25 %.

The discrepancy between the findings of Sanders *et al.* (2001) and that of Sims *et al.* (2007a; 2007b) presents an interesting problem which requires further investigation to indentify if high sodium concentration could be used during exercise to improve performance. A direct comparison between the studies is difficult given that one employed a during-exercise protocol (Sanders *et al.*, 2001) and the others had participants ingest the drinks over a protracted pre-exercise period (Sims *et al.*, 2007a; 2007b). However the premise for improvements in cardiovascular functioning in both cases was a result of P_{Vol} expansion. Given that both studies demonstrated an elevated P_{Vol}, it begs the question as to what factor or factors produced these conflicting findings. It is possible that pre-eercise interventions are more capable of improving exercise capacity than during exercise interventions. Alternatively we must also consider the differences in exercise conditions; the intenisity, environemnet and duration emkployed by Sanders *et al.* (2001) may have been insufficient to expose the detrements in physiological function in the control condition that are observed when exercise is of a hgher intensity, performed without stoppages nd n the heat, as with the studies of Sims and colleagues (Sims *et al.*, 2007a; 2007b).

2.4.2. Volume

Replacement of 80-100 % fluid losses during exercise has been suggested as necessary to prevent the deleterious effects of dehydration on cardiovascular and thermoregulatory functioning (Coyle, 2004; Montain & Coyle, 1992a). However, despite considerable efforts within the past decade to educate endurance athletes about the importance of maintaining adequate hydration during exercise (Sawka *et al.*, 2007),

recent research shows that athletes still routinely dehydrate by more than 2 % BM during prolonged exercise (Dugas *et al.,* 2006).

Failure to adequately rehydrate during exercise can be a consequence of either a self-conscious unwillingness to drink and/or because of the practical difficulties faced by athletes. For example, many athletic and occupational situations make it unfeasible to ingest 100 % of BM losses during exercise (Convertino *et al.,* 1996) due to issues such as availability, and the circumstances of competition. Much debate has focused on why, when given *ad libitum* access to fluids, athletes tend to drink at a rate that replaces approximately 50 % of their fluid losses (Hubbard *et al.,* 1984; Pitts & Consolazio, 1944; Pugh *et al.,* 1967).

It may on some occasions, be advisable to actually drink less than needed for full fluid replacement. As already noted, it is not often possible for athletes, especially runners, to exercise with the large gastric volume needed to promote the high rates of gastrointestinal fluid absorption that are required to prevent cellular dehydration during exercise. When sweating rate exceeds 1 $L \cdot h^{-1}$, it becomes progressively more difficult to offset cellular dehydration in many individuals because this requires exercising with a gastric volume of approximately 0.6-1.0 L of fluid (Coyle, 2004). The discomfort of that added volume and weight will not reduce physiological stress if it remains in the gut towards the end of the event and some individuals may not benefit by drinking the extra volume that ensures no body weight reduction at the end of exercise. Therefore, from performance perspective athletes may finish with up to a 2 % reduction in BM provided that the drinking schedule is designed to minimise gut fluid volume towards the end of exercise.

2.4.3. Summary

The ingestion of fluid is the only feasible method to rehydrate during exercise. A number of studies have demonstrated ergogenic benefits of ingesting water alone, but more significant findings have been found when electrolytes are included in the rehydration. This is because of the osmotic influence they exert, maintaining elevated P_{Osm}, promoting fluid conservation and P_{Vol} expansion.

Commonly available sports drinks tend to include approximately 10-25 $mmol \cdot L^{-1}$ of sodium, yet in conditions where free access to fluid is not available, the inclusion of

a higher concentration of sodium has been purported to improve cardiovascular functioning and exercise performance. This has been the case when drinks were ingested prior to exercise (Sims *et al.,* 2007a; 2007b) but as yet not when high sodium drink was ingested during exercise (Sanders *et al.,* 2001). Further research is required to examine the possible influence of high sodium drinks ingested during exercise, particularly exercise performed in an intense environment likely to stress the cardiovascular system to its limits.

2.5. Pre-exercise strategies to improve fluid balance

Pre-exercise interventions offer alternative strategies which are particularly useful when during-exercise ingestion of fluids is impractical and/or unfeasible. Since it appears difficult for athletes to drink sufficiently during exercise to maintain exercise endurance performance, starting a prolonged exercise while hyperhydrated, as opposed to only euhydrated, could contribute in improving performance as a result of delaying dehydration. Thus pre-exercise preparation becomes vital to maintaining hydration and offers a practical alternative to drinking during exercise. Methods designed to optimize pre-exercise hydration have become the focus of a large body of current research (Wingo *et al.,* 2004).

2.5.1. Infusion

A number of studies have employed techniques involving the artificial infusion of fluids into the body (e.g. Coyle *et al.,* 1986; 1990; Deschamps *et al.,* 1989; Fortney *et al.,* 1981a; 1981b; 1983; 1984; Grant *et al.,* 1997; Hopper *et al.,* 1988; Krip *et al.,* 1997; Montain & Coyle, 1992b). This technique, whilst highly successful is both impractical in either an athletic or occupational scenario, and illegal in competitive events. However, the studies employing this technique to promote P_{Vol} do provide the bulk of the literature regarding what is known of the impact P_{Vol} has on cardiovascular and thermoregulatory functioning during both short and long term exercise in both thermo-neutral and warm environments. Thus it would be remiss not to consider these studies in this review.

The early methods employed to promote pre-exercise hypervolemia involved the (re-) infusion of whole blood (Fortney *et al.,* 1981; Robinson *et al.,* 1966) or packed erythrocytes (Ekblom *et al.,* 1972). An advantage of using whole-blood is that it does not dilute the haemoglobin concentration in the same way the P_{Vol} expansion can. The illegality and the health risks associated with this technique drove researchers to develop alternative strategies to promote hypervolemia; mostly employing techniques normally used to treat clinical cases of hypovolemia, including the infusion of dextran, albumin or saline (Deschamps *et al.,* 1989; Fortney *et al.,* 1988a; Hopper *et al.,* 1988). Dextran is a potent osmotic agent that is used medicinally to treat hypovolemia. A normal dextran solution contains approximately 6 % dextran in 0.9 % saline and requires a lengthy infusion time (30-90 min). Serum albumin the most abundant plasma protein in humans has been linked to P_{Vol} expansion, during 3-8 d training studies (Gillen *et al.,* 1994; Convertino *et al.,* 1980). Due to its high osmotic pressure it increases circulating P_{Vol} by drawing on the tissue fluids (Janeaway *et al.,* 1944), but whilst decreasing the pool of fluid available for sweating (Hubbard *et al.,* 1984). Saline is the general term referring to solutions of NaCl in water. Since sodium is the major electrolyte of extracellular fluid, accounting for 95 % of variation in P_{Osm} (Saltmarsh, 2001) it acts as a powerful osmotic stimulus conserving fluid losses from kidney and promoting fluid fluxes from intracellular fluid compartment. A normal saline solution contains approximately 0.9 % NaCl i.e. 9 g of NaCl dissolved in 1 L of water (154 $mmol \cdot L^{-1}$) and has an osmolality of approximately 300 $mOsm \cdot L^{-1}$.

2.5.1.1. Cardiovascular adaptations

Reductions in HR have consistently been demonstrated following pre-exercise P_{Vol} expansion in a range of protocols. Fortney and colleagues demonstrated a reduction in HR during 30 min exercise (60-70 % $\dot{V}O_{2max}$; 30-35^{o}C; 40 % RH) when pre-exercise P_{Vol} expansion was induced with infusion of whole blood (410 mL; Fortney *et al.,* 1981a) or albumin (7.8 %; Fortney *et al.,* 1983). Reductions in HR of 13 $b \cdot min^{-1}$ have also been demonstrated during prolonged mild exercise in extreme heat (90 min; 45 % $\dot{V}O_{2max}$; 45^{o}C, 20 % RH), following pre-exercise P_{Vol} expansion of 13 % (50 g albumin; Sawka *et al.,* 1983). It should be noted that not all studies have reported a reduction in HR following pre-exercise P_{Vol} expansion during exercise in the heat. Watt *et al.* (2000)

also reported no change in HR during 40 min exercise at 64 % $\dot{V}O_{2max}$ at 35°C following 12 % P_{Vol} expansion.

When exercise has been conducted in a thermo-neutral environment, disparate findings have been reported. For example, Hopper *et al.* (1988) found no change in HR from control when pre-exercise P_{Vol} was expanded by either 11 or 22 % in untrained men, or by 11 % in trained men, before two short bouts of exercise (2 x 16 min, 56 % $\dot{V}O_{2max}$). During exercise lasting 5-7 min, performed in a thermo-neutral environment, Berger *et al.* (2006) also demonstrated no change in HR when exercise was performed at 70 % ΔGET-$\dot{V}O_{2max}$ [3], following P_{Vol} expansion of 14 % and Kanstrup *et al.* (1992) actually reported a 12 $b \cdot min^{-1}$ increase in HR when exercise was performed at 50 W following 15 % expansion of blood volume. When exercise is performed in a thermo-neutral environment but is prolonged, others have found an improvement in cardiovascular function (Grant *et al.,* 1997; Roy *et al.,* 2000). Roy *et al.* (2000) demonstrated a reduction in HR in trained men. However in these studies, the level of pre-exercise P_{Vol} expansion was greater (14-21 %) than when commensurate reductions in HR were demonstrated during exercise in the heat (Fortney *et al.,* 1981; 1983).

Studies inducing P_{Vol} expansion of 12-20 % have consistently demonstrated increased SV of 9-20 % during exercise in the heat (Coyle *et al.,* 1986; Fortney *et al.,* 1981a; Fortney *et al.,* 1983; Montain & Coyle, 1992b) and in a thermo-neutral environment (Grant *et al.,* 1997; Hopper *et al.,* 1988; Kanstrup & Ekblom, 1982). Coyle *et al.* (1990) also demonstrated an increase in SV of 15 % following P_{Vol} expansion of just (7 %). The increases in SV with P_{Vol} expansion would presumably result from an increase in venous filling (Hopper *et al.,* 1988; Horwitz & Lindenfield, 1985; Krip *et al.,* 1997; Mier *et al.,* 1996; Robinson *et al.,* 1966; Warburton *et al.,* 1999), resulting in a greater end diastolic volume [EDV] and increased SV.

Interestingly, in trained athletes Coyle *et al.* (1986) and Hopper *et al.* (1988) failed to demonstrate an increase in SV following 12-15 % increase in P_{Vol}. It is possible that trained athletes, who already possess an elevated P_{Vol} in respect of their untrained counterparts, may be limited to further increase their capacity. Indeed, Hopper

[3] GET-VO_{2max}. Difference in power output measured at gas exchange threshold and maximum oxygen uptake.

et al. (1988) and Grant *et al.* (1997) both demonstrated that P_{Vol} expansion of 21 % added no further cardiovascular advantage than P_{Vol} expansion of 12-14 %.

Coyle *et al.* (1986) demonstrated a 5 % increase in $\dot{Q}$, as a result of a 12 % increase in SV and only 6 % decrease in HR, when P_{Vol} was expanded by 28 % in untrained participants prior to exercising at 50-60 % $\dot{V}O_{2max}$. Similarly, Fortney and colleagues (Fortney *et al.*, 1981a; 1983) demonstrated a 1.0-1.4 $L \cdot min^{-1}$ increase in $\dot{Q}$ as a result of a 10-15 $mL \cdot min^{-1}$ increase in SV, and just a 6-7 $b \cdot min^{-1}$ decrease in HR, during 30 min exercise in the heat (30-35^{o}C; 30 min, 60-70 % $\dot{V}O_{2max}$), following P_{Vol} expansion of 10-12 % (whole blood, and albumin). Following blood volume expansion of 398 mL, which increased P_{Vol} by 2.5 % during 10-110 min period of prolonged exercise in the heat, Montain & Coyle (1992b) demonstrated an attenuated decline in $\dot{Q}$ from 11 to 0 %, in respect of no fluid ingestion, in which P_{Vol} decreased by 3 % over the same period. In contrast, despite an 11 % increase in SV and just a 3-4 $b \cdot min^{-1}$ (non-significant) decrease in HR, Hopper *et al.* (1988) reported just a trend for an increase in $\dot{Q}$ (1.3-1.4 $L \cdot min^{-1}$) during 2 x 16 min bouts at 56 % $\dot{V}O_{2max}$ in a thermo-neutral environment, following P_{Vol} expansion of 12 & 21 %. Grant *et al.* (1997) demonstrated similar findings during 2 h exercise in a thermo-neutral environment at 46 % $\dot{V}O_{2max}$ following P_{Vol} expansion of both 14 and 21 %. It is likely that differences it $\dot{Q}$ are only demonstrated when the exercise and or environmental stress are sufficient to induce reductions in muscle blood flow [BF_{Mus}].

2.5.1.2. Thermoregulatory adaptations

In a series of experiments consisting of 30 min of moderate intensity exercise in the heat (60-70 % $\dot{V}O_{2max}$; 30-35^{o}C; 30 % RH), Fortney and colleagues (Fortney *et al.*, 1981a; 1981b; 1983) examined whether pre-exercise hypervolemia (7.6-7.9 %) induced by blood or albumin infusion improved thermoregulation. In just one study (Fortney *et al.*, 1981b) did hypervolemia reduce T_{Esp} significantly and then only by 0.11^{o}C. BF_{Skn} was not significantly increased in any study. Similarly Sawka *et al.* (1983) demonstrated no change in T_{Core} or sweat rates during mild exercise in severe heat (90 min walk, 45 % $\dot{V}O_{2max}$, 45^{o}C, 20 % RH) with pre-exercise P_{Vol} expansion of 13 %. In addition, during prolonged exercise (90-120 min) in a thermo-neutral environment

Leutkemeier and Thomas (1994) and Grant *et al.* (1997) failed to demonstrate a change in T_{Core} following P_{Vol} expansion of 9-21 %. Watt *et al.* (2000) also failed to demonstrate an increase in BF_{Skn} when P_{Vol} was expanded by 12 % before cycling for 40 min in a thermoneutral environment (72 % $\dot{V}O_{2max}$; 22^{o}C, 40 % RH), although there were also no differences in cardiovascular variables between conditions. Calver *et al.* (1992) did demonstrate an increase in BF_{Skn} when P_{Vol} was expanded by 7 %, although during rest, when the competition between BF_{Skn} and BF_{Mus} is substantially reduced.

The failure to demonstrate a change in thermoregulation after pre-exercise hypervolemia is surprising given the robust detrimental effect of pre-exercise hypovolemia on BF_{Skn}, sweat rates and T_{Core} (Fortney *et al.,* 1981a; Nadel *et al.,* 1979). The reduction in BF_{Skn} reflects the body's attempt to maintain $\dot{Q}$ in the face of diminishing blood volume. In response to a reduction in CBV detected by cardiopulmonary low pressure baroreceptors (Johnson *et al.,* 1974b; Kellogg *et al.,* 1990; Mack *et al.,* 1988; Zoller *et al.,* 1972) and/or a reduction in MAP detected by arterial high-pressure baroreceptors (Rowell *et al.,* 1972; Zoller *et al.,* 1972), the cardiovascular system increases systemic vascular resistance through vasoconstriction of the skin, splanchnic area and inactive muscle (Gonzalez-Alonso *et al.,* 1997; Johnson *et al.,* 1974a; Rowell *et al.,* 1972). Thus it is surprising that despite a greater CBV with pre-exercise hypervolemia, no increases in $BF_{Skn,}$ sweat rates, and/or decreases in T_{Core} have consistently been demonstrated. An increase in BF_{Skn} has also been demonstrated when P_{Vol} expansion occurs during exercise (Fortney *et al.,* 1988; Deschamps *et al.,* 1989; Nose *et al.,* 1990). It is possible that a ceiling effect occurs in which expansion of P_{Vol} above resting levels donot increase BF_{Skn} further, which is not the case when P_{Vol} decline is preventing during exercise.

Montain & Coyle (1992b) found that P_{Vol} expansion during exercise (2 h; 62-67 % $\dot{V}O_{2max}$; 33^{o}C, 50 % RH), which induced dehydration of (3-4 % BM loss) failed to prevent reductions in BF_{Skn} or to attenuate hyperthermia, even though it prevented hypovolemia and partially restored SV (-17 vs. -27 %). Replacement of 100 % BM losses (~2.4 L) with a 6 % CHO-E drink, however, increased BF_{Skn} and reduced T_{Esp} and T_{Rec} in respect of both no fluid and blood volume expansion conditions. These findings suggest that fluid replacement attenuates the rise in T_{Core} by some means other than blood volume, possibly as a result of reduced P_{Osm} and greater BF_{Skn}. Hyperosmolality has been shown to induce a rise in the thermoregulatory threshold or

vasodilation, which delays the onset of BF_{Skn} redistribution, lowers sweat rates and conserves fluid losses (Nielsen, 1974; Greenleaf & Castle, 1971; Takamata *et al.,* 1997) in attempt to minimise hyperosmolality. Pre-exercise hyperosmolality has consistently been reported to affect thermoregulation (Nielsen *et al.,* 1974; Takamata *et al.,* 2001).

2.5.1.3. Performance

There is strong evidence to suggest that the delivery of oxygen to the working muscles is the principal determinant for $\dot{V}O_{2max}$ attained by large muscle groups (Gonzalez-Alonso & Calbert, 2003). Thus a P_{Vol} expansion mediated increase in $\dot{Q}$ and presumably an increase in BF_{Mus} should theoretically enhance delivery of oxygen and extend performance. Conversely, acute P_{Vol} expansion reduces the Hb concentration and thus reduces arterial oxygen content and may compromise $\dot{V}O_{2max}$. For example, when haemodilution has been experimentally induced, with no change in P_{Vol} and the related adaptations, by hypoxia (Stenberg *et al.,* 1966) and carbon monoxide (Ekblom *et al.,* 1975) a decrease in arterial oxygen content and reduced aerobic power during maximal exercise have been demonstrated. Haemodilution is also associated with a reduced hydrogen buffering capacity (Gledhill *et al.,* 1982), yet it increases SV with P_{Vol} expansion (Hopper *et al.,* 1988; Krip *et al.,* 1997; Kanstrup & Ekblom, 1982; Mier *et al.* 1996; Warbuton *et al.,* 1999) and may offset the reduction in Hb and preserve or even enhance muscle oxygen delivery (Gledhill *et al.,* 1994). Given this conflict, it is understandable that the literature regarding P_{Vol} expansion and maximal aerobic performance has produced disparate findings, with reports of decreases (Kanstrup & Ekblom, 1992), increases (Berger *et al.,* 2006; Coyle *et al.,* 1990; Krip *et al.,* 1997) or no changes (1984; Mier *et al.* 1996; Warbuton *et al.,* 1999) in $\dot{V}O_{2max}$.

Whether or not P_{Vol} expansion improves $\dot{V}O_{2max}$ depends on the extent to which increases in SV and $\dot{Q}$ compensate for decreases in Hb, or *vice versa.* For example, Coyle *et al.* (1986) demonstrated a 3 % increase in $\dot{V}O_{2max}$ following 28 % P_{Vol} expansion despite a 14 % decrease in Hb in untrained participants. In trained participants P_{Vol} expansion of 15 % did not significantly affect $\dot{V}O_{2max}$ despite a 9 % decrease in Hb. The increase and maintenance of $\dot{V}O_{2max}$ with untrained and trained participants can be attributable to 5 % and 4 % (non significant) increase in $\dot{Q}$,

respectively. In contrast, an 8 % increase in $\dot{Q}$ with blood volume expansion of 690 mL did not affect $\dot{V}O_{2max}$ probably as a result of the 11 % decrease in Hb (Kanstrup & Ekblom, 1982). Coyle *et al.* (1990) demonstrated a 4 % increase in $\dot{V}O_{2max}$ following 7 % P_{Vol} expansion, but no change $\dot{V}O_{2max}$ following 17 % P_{Vol} expansion. Hb was 4 and 14 % reduced with the low and high P_{Vol} expansion, respectively. The increase in $\dot{V}O_{2max}$ with low P_{Vol} expansion can be attributed to a 15 % increase in SV. SV was not measured with high P_{Vol} expansion, although Hopper *et al.* (1988) demonstrated that P_{Vol} expansion of 706 mL (21 %) did not increase SV to a greater extent than P_{Vol} expansion of 400 mL (12 %). These findings suggest that P_{Vol} expansion below 400 mL increases or maintains $\dot{V}O_{2max}$ because an increase in SV and $\dot{Q}$ compensate for a reduction in Hb. However, P_{Vol} expansion above 400 mL tends not to increases SV or $\dot{Q}$ to a greater extent and as such the greater haemodilution impairs $\dot{V}O_{2max}$.

Whilst $\dot{V}O_{2max}$ provides a good indicator of performance, it is not a true performance test. Unfortunately, changes performance; either time to exhaustion or time trial, have rarely been measured following acute P_{Vol} expansion and of those that have, findings have been inconsistent. Berger *et al.* (2006) demonstrated a 16 % (365 vs. 424 sec) increase in time to exhaustion whilst cycling at 70 % GET-$\dot{V}O_{2max}$, following pre-exercise P_{Vol} expansion of 14 %. Leutkemeier and Thomas (1994) demonstrated a 10 % decrease in time trial performance at a moderate intensity following 8.7 % P_{Vol} expansion. Coyle *et al.* (1990) reported a significant increase in time to exhaustion following 7 % (282 mL) but not 17 % (624 mL) P_{Vol} expansion.

In contrast, despite a reduction in T_{Esp} and HR, Deschamps *et al.* (1989) did not demonstrate any significant differences in time to exhaustion when 0.9 % saline (mean vol. 1280 mL) was infused at the onset of exercise. However, exercise was brief and conducted in a thermo-neutral environment (84 % $\dot{V}O_{2max}$; ~20 min; 24^{o}C). Kanstrup and Ekblom (1982) and Coyle *et al.* (1986) reported a decline in time to exhaustion in moderately trained and endurance trained men, respectively, despite little change in $\dot{V}O_{2max.}$ Coyle *et al.* (1986) also reported a decline in time to exhaustion in detrained participants despite a 3 % increase in $\dot{V}O_{2max}$. The variations in environmental temperatures, exercise intensities and infusate may explain the disparate findings. Clearly further research is needed.

2.5.1.4. Summary

There is a consistent cardiovascular benefit of pre-exercise P_{Vol} expansion, when exercise is performed in the heat and/or is prolonged. However, although blood volume is an important determinant of BF_{Skn}, when hypovolemic, expansion of P_{Vol} does not appear to enhance BF_{Skn} or lower T_{Core}. This is possibly because of a ceiling effect whereby further increases in blood volume make little difference to blood distribution. More likely is that the concomitant elevations in P_{Na} and/or P_{Osm} with P_{Vol} expansion impair the sweating response. The effect of pre-exercise P_{Vol} expansion on performance is unclear, with a need for more ecologically valid measures of performance. There appears to be little difference in the infusates employed to promote P_{Vol} expansion. The ideal volume of P_{Vol} expansion appears to be approximately 200-400 mL (5-12 %) with further expansion having no additional cardiovascular benefit whilst further impairing haemoglobin concentration.

2.5.2. Ingestion

Whilst infusion of whole blood, saline or a naturally occurring P_{Vol} expander may be highly effective in improving cardiovascular functioning, their employment is impractical and lacks transferability into real life sporting or occupational scenarios. The ingestion of fluid represents a more practical strategy to improve pre-exercise hydration and/or increase P_{Vol}, which is easily adaptable to real life sporting situations.

Hyperhydration, often referred to as ‘overdrinking’, involves the ingestion of fluid in excess of immediate bodily requirements. Simple overdrinking will usually stimulate urine production (Institute of Medicine, 2005) with the possible risk of dilutional hyponatraemia (Zambraski *et al.,* 2005). Alternatively overdrinking can be combined with an agent that "binds" water within the body (Freund *et al.,* 1995; Greenleaf *et al.,* 1998). These binding agents include glycerol and other hypertonic drinks such as those high in sodium that can induce hyperhydration for varied durations. Over consumption of fluids with most hyperhydration binding agents will still elevate U_{Vol} well above normal levels, although to a far lesser extent than water.

Many hyperhydration studies were performed on athletes who had been deliberately dehydrated first. The advantage of beginning an exercise hyperhydrated is

that it will delay or eliminate the onset of dehydration if an athlete fails to completely replace their sweat losses during exercise. Given that athletes routinely finish an event in a dehydrated state (Broad *et al.* 1996; Burke & Hawley, 1997; Maughan *et al.* 2004; Sharp, 2006; Shirreffs *et al.* 2005), it would certainly be advantageous for them to start exercising in a hyperhydrated state. Although many pre-exercise hyperhydration protocols are available the most common methods employ either the ingestion of large quantities of water alone or water mixed with an agent that binds water within the body such as glycerol or sodium.

2.5.2.1. Water

Findings are equivocal as to whether pre-exercise hyperhydration with water provides a physiological advantage. Some studies have demonstrated a reduction in cardiovascular and thermoregulatory strain (Moroff & Bass, 1965; Gruczka *et al.,* 1987; Nielsen *et al.,* 1971; Gisolfi & Copping, 1974; Lyons *et al.,* 1990) and an improvement in performance (Blyth & Burt, 1961; Greenleaf *et al.,* 1997; Latzka *et al.,* 1998), compared with no fluid ingestion. However, others have also failed to demonstrate noticeable changes in thermoregulation (Gisolfi & Copping, 1974; Candas *et al.,* 1988; Montner *et al.,* 1996; Greenleaf & Castle, 1971; Latzka *et al.,* 1997; 1998; Nadel *et al.,* 1980) or cardiovascular efficiency (Latzka *et al.,* 1998). The discrepancy in results relates largely to the timing of ingestion or the length of exercise protocol. The timing is important since simple overdrinking of plain water stimulates urine production (Institute of Medicine, 2005), as a result of P_{Osm} dilution and the suppression of antidiuretic hormones (Casa *et al.,* 1999; Gonzalez-Alonzo *et al,* 1992; Shirreffs & Maughan, 1998; Nose *et al.,* 1988a). Thus, increases in TBW and blood volume are typically only transient (Koenigsberg *et al.,* 1995; Freund *et al.,* 1995; Murray *et al.,* 1991). The overdrinking of hypotonic fluids and loss of total body sodium is one of the main factors contributing to exercise-associated hyponatraemia (Montain *et al.,* 2006; Zambraski, 2005). Hyponatraemia occurs when P_{Na} rapidly drops below ~130 $mmol \cdot L^{-1}$ (Sawka *et al.*, 2007) and has been reported in a number of participants from a variety of occupational and recreational activities (Ayus *et al.,* 2000; Levine & Thompson, 2005).

2.5.2.2. Glycerol

The addition of glycerol to the ingested fluid is purported to delay the excretion of the excess fluid and thereby prolongs the hyperhydration state compared with the equivalent volume of water ingestion (Anderson *et al.,* 2001; Coutts *et al.,* 2002; Freund *et al.,* 1995; Goulet *et al.,* 2006; Goulet *et al.,* 2008; Hitchens *et al.,* 1999; Koeningsberg *et al.,* 1995; Lyons *et al.,* 1990; Magal *et al.,* 2003; Montner *et al.,* 1996; Riedesel *et al.,* 1987; Wingo *et al.,* 2004). Glycerol is a three-carbon alcohol metabolite that the human body produces naturally. It is a safe, clear, syrupy, and exceptionally sweet liquid classified as legal by the International Olympic Committee. It increases P_{Osm} and, when accompanied by copious amounts of water (20-35 $mL \cdot kgBM^{-1}$) and provides an osmotic drive that augments retention of large quantities of water otherwise eliminated by the kidneys (Robergs & Griffin, 1998).

When compared with no fluid, an improvement in exercise performance with glycerol hyperhydration has been reported, although without changes in either cardiovascular functioning or thermoregulation. For example, Latzka *et al.* (1998) measured endurance time whilst partcipants exercised to exhaustion (or a critical T_{Core} of 40^{o}C) at 55 % $\dot{V}O_{2max}$ in the heat (35^{o}C), 1 h after hyperhydrating with (29 $mL \cdot kg^{-1}$ BM) of either glycerol [+ water]; water alone; or no fluid. No significant differences in T_{Core}, whole body sweating rate, or $\dot{Q}$ were demonstrated between trials. Yet glycerol hyperhydration induced a 15 % increase in endurance time, compared with no fluid. Kavouras *et al.* (2006) demonstrated a 74 % increase (19 vs. 33 min) in exercise capacity following glycerol hyperhydration vs. no fluid during exercise to exhaustion in an uncompensable ambient temperature (74 % $\dot{V}O_{2max}$; 37^{o}C, 48 % RH), but also failed to demonstrate any significant cardiovascular of thermoregulatory benefits. Goulet *et al.* (2008) also demonstrated an increase in exercise capacity (11.9 vs. 13.6 min) and a reduction in HR with glycerol hyperhydration vs. no fluid, but no differences in T_{Core} during 2 h of prolonged moderate intensity exercise, although exercise was performed in a warm environment (26-27^{o}C).

Several groups have shown positive physiologic effects after glycerol vs. water hyperhydration. Glycerol hyperhydration has been demonstrated to increase sweat rate (Lyons *et al.,* 1990) and SV (Montner *et al.,* 1996 1999) and reduce T_{Rec} (Anderson *et al.,* 2001) and HR (Anderson *et al.,* 2001; Goulet *et al.* 2008; Montner *et al.,* 1996; 1999); and improve exercise performance in both a thermo-neutral (Montner *et al.,*

1996) and hot (Anderson *et al.,* 2001; Hitchins *et al.,* 1999; Kavouras *et al.,* 2006) environment. However, others have failed to demonstrate differences in either cardiovascular functioning, thermoregulation or performance with glycerol hyperhydration compared to water hypehydration (Coutts *et al.,* 2002; Hitchins *et al.,* 1999; Latzka *et al.,* 1997; 1998; Marino *et al.,* 2003; Montner *et al.,* 1996; Freund *et al.,* 1995; Goulet *et al.,* 2008; Montner *et al.,* 1996; Inder *et al.,* 1998; Magal *et al.,* 2003; Marino *et al.,* 2003; Wingo *et al.,* 2004).

The failure to consistently demonstrate either a decrease in thermoregulatory or cardiovascular strain, means researchers are unable to identify the mechanism by which glycerol hyperhydration improves exercise capacity. Though it cannot be completely ruled out that glycerol improved exercise capacity because of a yet-to-be-identified mechanism, it is important to mention that there are characteristics of each of these studies which have demonstrated an increase in exercise performance, which have flaws in the methodologically which may have confounded the results. For example, no studies reported that the distinctive taste of glycerol was efficiently/totally masked. Some studies did not control for or standardise pre-exercise hydration, diet, and levels of activity (Couts *et al.,* 2002; Goulet *et al,* 2006). The majority of studies have investigated the effects of glycerol hyperhydration vs. water hyperhydration on euhydrated participants, whilst others pre-dehydrated their participants (Kavouras *et al.,* 1998). Some studies allowed fluid ingestion during exercise (Magal *et al.,* 2003; Anderson *et al.,* 2001; Goulet *et al.,* 2008), whilst others have not. The majority of studies have added glycerol to a plain water solution, whilst some have used sports drinks (Anderson *et al.,* 2001; Coutts *et al.,* 2002). Some of the studies employed a small sample size with mixed gender participants (Goulet *et al.,* 2006; Goulet *et al.,* 2008). Lastly, one of the studies has shown that the increase in exercise capacity with glycerol was mainly due to 2 athletes out of 11 who increased their exercise capacities by about 60 minutes, compared to about 6 minutes for the rest of the group (Coutts *et al.,* 2002). A closer examination of the data reveals that two subjects actually showed a decrease in exercise capacity. Therefore, a lack of reliability in the exercise tests and not glycerol's effect *per se*, could well have accounted for the increase in exercise capacity in that study.

There are a number of negative side effects which occur with glycerol hyperhydration such as nausea, vomiting, gastrointestinal distress, blurred vision, headaches and light-headedness. Additionally, women who are pregnant and individuals

with diabetes, kidney, liver and cardiovascular disorders must consult their family physician before using it. Further, tissue shrinkage is a well-recognized consequence of the administration of large amounts of glycerol (Maughan, 1998). The elevation of the osmolality of the extracellular space may result in water movements from the intracellular space, and cell dehydration. To date, no research has evaluated the long-term effects of glycerol ingestion. The molecular weight of glycerol is substantial, inducing pronounced increases in osmolality of all fluid compartments. In contrast with water hyperhydration, glycerol hyperhydration increases P_{Osm} by more than 10 $mOsmol \cdot kg^{-1}$. For instance, Murray *et al.* (1991) demonstrated significant increases in P_{Osm}, maxing at 313 $mmol \cdot L^{-1}$ following glycerol hyperhydration, whilst water hyperhydration maintained P_{Osm} at baseline levels throughout exercise. The increase in P_{Osm} of this magnitude may have a negative effective on thermoregulation. Hyperosmolality has been found to delay the onset of sweating, attenuate BF_{Skn}, and reduce sweat rate (Nielsen, 1974; Greenleaf & Castle, 1971; Takamata *et al.*, 1997).

Surprisingly, the large increases in TBW with glycerol hyperhydration, compared to water hyperhydration, appear to have little impact on P_{Vol}. This is because glycerol is distributed evenly throughout the body water, and results in both intracellular and extracellular compartment gains. For example, a 750 mL increase in TBW evenly distributed among body water compartments would induce just a ~50-60 mL increase in P_{Vol} (Montner *et al.*, 1999; Freund *et al.*, 1995), equivalent to just ~1.7-2 % for (based on a resting P_{Vol} of trained male: 2900 mL). Such small changes in P_{Vol} would be difficult to detect given the relatively small numbers of participants used in most past research (e.g. Nadel *et al.*, 1980; Montner *et al.*, 1996; 1999; Freund *et al.*, 1995; Lyons *et al.*, 1990).

2.5.2.3. Sodium

Greater P_{Vol} expansion is demonstrated with sodium hyperhydration since sodium primarily results in extracellular gains. Thus ingestion of sodium represents an effective strategy to increase P_{Vol}. Greenleaf & Brock (1980) investigated the effects of NaCl ingestion on P_{Vol} distribution at rest and exercise (40-47 % $\dot{V}O_{2max}$), in cool ($27^{o}C$) and hot ($39^{o}C$) environments. During the pre-exercise rest period, participants drank 16-17 $mL \cdot kg \cdot BM^{-1}$ (~1.2 L) of either hypertonic (250 $mmol \cdot L^{-1}$; 493 $mOsm \cdot kg^{-1}$) or isotonic (155 $mmol \cdot L^{-1}$; 315 $mOsm \cdot kg^{-1}$) NaCl or a hypertonic calcium glutamate

solution (Control). Compared with controls, both sodium drinks increased P_{Vol} during rest and maintained P_{Vol} at an elevated level throughout exercise, in both environments. There was no difference in P_{Vol} expansion between sodium drinks in either cool or hot environments during rest, or in the cool environment during exercise. However, during exercise in the heat, P_{Vol} was elevated to a greater extent with isotonic sodium at the end of 60 min exercise. This was likely to be due to a faster rate of absorption from the gastrointestinal tract with isotonic sodium, compared to hypertonic sodium. Osmolality exerts a significant influence on intestinal water absorption (Coombes & Hamilton, 2000). A negative correlation exists between the osmolality of luminal contents and water absorption (Farthing, 1988; Wapnir & Lifshitz, 1985). Whilst hypotonic and isotonic solutions promote intestinal water absorption (Hunt *et al.,* 1985; Leiper & Maughan, 1988), solutions hypertonic to human plasma (> 290 $mOsm \cdot kg^{-1}$) stimulate less water absorption and more secretion into the gastrointestinal lumen (Maughan & Noakes, 1991). Thus an optimal sodium concentration would not exceed ~300 $mOsm \cdot kg^{-1}$.

Harrison *et al.* (1978) reported greater blood volume with ingestion of 1 % saline (~170 $mmol \cdot L^{-1}$ Na) compared with water, following thermal dehydration (~ 5 % BM, -8 % blood volume). Saline restored blood volume to baseline levels, whilst ingestion of water restored blood volume to only -6 %, at the start of exercise. During a subsequent bout of moderate intensity cycling exercise, lasting 45 min, HR was significantly reduced but T_{Core} was significantly elevated, with saline compared with water. However, this study investigated saline ingestion following thermal dehydration, not in a euhydrated state.

Sims and colleagues demonstrated a 4.3-4.5 % increase in P_{Vol}, a reduction in HR and T_{Rec} and a 21-25 % increase in exercise performance in endurance trained female cyclists (Sims *et al*., 2007a) and male runners (Sims *et al*., 2007b) following ingestion of 10 $mL \cdot kgBM^{-1}$ (~ 630 & 757 mL) of a solution containing a high vs. low sodium concentration (164 vs. 10 $mmol \cdot L^{-1}$). One of the problems with hyperhydrating with water or glycerol is that participants are required to ingest large amounts of fluid 20-35 $mL \cdot kgBM^{-1}$, which will greatly increase the risk of having to void during competition (Freund *et al.,* 1995; O'Brien *et al.,* 2005) and may be unrealistically consumable in field settings (Ganio *et al.,* 2006). It is doubtful whether a large part of the athletic population could easily tolerate the necessary volume of fluid to be taken before key competitions when stress and anxiety are at their peak. An advantage of

sodium hyperhydration, is that it requires participants to consume far less quantities of fluid (757 mL; Sims *et al.*, 2007a) compared to previous hyperhydration studies (>2 $L \cdot h^{-1}$).

During the studies of Sims, ingestion of two drinks occurred over a 1 h rest period starting 105 min and 80 before exercise, in males and females, respectively. Measurement of P_{Vol} at -5 and -25 min, respectively revealed P_{Vol} expansion of 4.3-4.5 %, largely as a result of a reduction in U_{Vol}. However P_{Vol} expansion was only transient and retracted back to below baseline at the point of the next measurement; at 10 min and the start or exercise, respectively. It is probable that most of the extra fluid was shifted to the interstitial compartment after its absorption. Thus, although the hypervolemic effect was transient, the absolute increase in fluid volume was sustained.

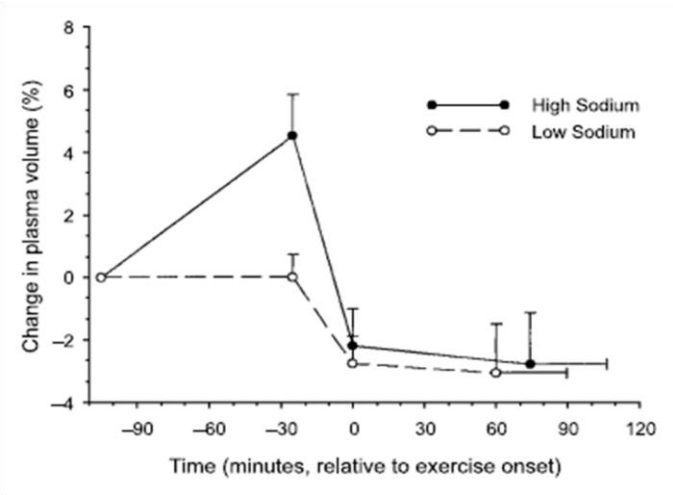

Figure 2.2. Changes in plasma volume as a percentage from baseline. *Note.* Modified from Sims ST, van Vliet L, Cotter J, and Rehrer N (2007b). Sodium Loading Aids Fluid Balance and Reduces Physiological Strain of Trained Men Exercising in the Heat. *Med Sci Sports Exerc* 39 pp123-130. Values are means ± SD.

2.5.3. Summary

To date these are the only studies which have used sodium hyperhydration to promote pre-exercise P_{Vol} and measure the effect on performance. The decrease in HR, despite a relatively small P_{Vol} expansion, in respect of the infusion studies is an interesting finding. In light of these studies, it is somewhat surprising to witness a reduction in T_{Rec}. Unlike the former studies, Sims *et al.* (2007a; 2007b) reported baseline levels of P_{Osm} at the start of exercise. It is possible that in other P_{Vol} studies

thermoregulation was inhibited by plasma hyperosmolality and thus advantage that could have been gained from P_{Vol} expansion was neutralised. Unfortunately the study did not measure SV or $\dot{Q}$, thus it is not known whether the change in cardiovascular performance was responsible for the increase in time to exhaustion.

Despite the apparent benefits of ingesting a highly concentrated sodium solution, the two studies by Sims and colleagues (Sims *et al.*, 2007a; 2007b) are currently the only studies to investigate this strategy as a means to reduce CV_{Drift} in exercising athletes. Furthermore, these studies only examined the effect of ingesting high sodium concentrations prior to exercise. It is not known, what effect, if any, the additional ingestion of fluid during exercise might have on exercise performance.

2.6. Conclusions and Rationale for Thesis

In each of the exercise settings discussed in this literature review; before, during and after exercise, there are already in place a number of well developed strategies to minimise dehydration and restore fluid balance. These are often impractical, such as P_{Vol} expansion by infusion of saline; rehydrating with large volume of fluid within 1 h; or attempting to ingest 100 % of BM losses during exercise. In all cases the ingestion of a high sodium concentration (>150 $mmol \cdot L^{-1}$) offers an alternative and often more practical solution. To date there is relatively few studies which have sought to employ such high concentrations, in part because of the unpleasant taste sodium evokes. It would be highly impractical to suggest the consumption of unpalatable solutions. Thus in order to assess the efficacy of the high sodium concentration it is also necessary to consider the role of taste to these solutions. By doing so it is important to consider the changing taste perceptions for sodium in times of physiological need. It is hoped that this thesis will provide a preliminary assessment of the efficacy of ingesting high sodium concentrations before, during and after exercise.

The aim of this thesis is to investigate:

1) Whether the ingestion of a high sodium concentration accelerates recovery from exercise-induced dehydration, when compared with a regular sports drink commonly employed by recreational athletes.

2) Whether exercise-induced dehydration effect the taste perceptions for three drinks of different sodium concentrations.

3) The relationship exists between body fluid balance and changes in taste perceptions during 3 h recovery from exercise-induced dehydration and the accompanying mild sodium loss.

4) The effects of ingestion a high sodium concentration, compared with a regular sports drink, on cardiovascular and thermoregulatory functioning duing high intensity exercise in the heat.

5) The effects of pre-exercise ingestion of a high sodium concentration on physiological functioning and exercise capacity during high intensity exercise inthe heat.

6) The effects of the combination of pre-exercise ingestion of a high sodium concentration and the supplementary ingestion of fluid during exercise on physiological functioning and exercise capacity during high intensity exercise inthe heat.

In addition to these core aims the thesis also addressed some other important issues. Firstly, the effect of pre-cooling with cold-fluid ingestion, and secondly the combination of pre-cooling and the pre-exercise ingestion of a high sodium concentration on physiological functioning and exercise capacity during high intensity exercise in the heat.

Chapter 3. Common Methodologies

All experimental protocols conformed to the standards set by the Declaration of Helsinki and were approved by the appropriate ethical review board at University of Exeter. For each study, participants provided written informed consent and completed health screening questionnaires. Testing took place in a controlled laboratory environment at the University of Exeter, Sport & Health Sciences. In addition to exclusion criteria outlined by the PAR-Q health questionnaire, all participants were non-smokers, and were screened for known taste defects, renal and heat related abnormalities. All studies employed repeated measures designs. Chapters 8 and 9 utilised a counterbalanced trial order. All other chapters employed a randomly assigned trial order. Participants were not informed of the objective of the study until after its completion and were blinded to each drink.

3.1. Participants

Participants were recruited from local cycle and tri-athlete clubs and from the University of Exeter, and were given no financial remuneration. Table 3.1 provides a summary of the participant characteristics for each study. Sample sizes were calculated based on previous findings from a related paper, using the following equation:

$$N = (2 \times SD^2 \times (Z_\alpha + Z_ß)^2) / \Delta^2$$

Vincent, 1999 p 142

The number of participants (N) employed was determined for a given power (= .80) and level of significance (P = 0.05). Using the table of normal distribution (Vincent, 1999); $Z_ß$ = .84 which corresponds to Z_α = 1.96.

3.2. Standardisation

Equal hydration before each trial was obtained by asking the participants to maintain the same diet and fluid intake in the 24 h period before the test, and to refrain from ingesting any alcohol or caffeine. Participants were also required to abstain from

strenuous exercise 48 h before exercise. Equal hydration was confirmed by the pre-trial measurement of BM, U_{Osm}, P_{Osm} and P_{Na}, subject to the availability of equipment (see table 3.2). To ensure against a learning effect, studies involving a performance element employed a familiarisation session; conducted with the same exercise intensity and environmental conditions as for experimental trials (Currell & Jeukendrup, 2008).

Table 3.1. Summary of participant characteristics

	Sample Size	Age (yr)	Body Mass (kg)	$\dot{V}O_{2max}$ ($mL \cdot kg \cdot min^{-1}$)	Training Status
1	8	27 ± 6	74 ± 7	59 ± 7	Regularly Active
2	10	29 ± 9	76 ± 6	63 ± 5	Endurance Trained
3	8	27 ± 6	74 ± 7	59 ± 7	Regularly Active
4	8	24 ± 4	72 ± 5	53 ± 6	Regularly Active
5	8	24 ± 4	77 ± 6	54 ± 5	Regularly Active
6	8	26 ± 7	77 ± 6	49 ± 6	Regularly Active

Endurance trained cyclists, defined as those engaging in cycle training >2 $h \cdot d^{-1}$, 3-5 $d \cdot wk^{-1}$; Regularly active, defined as those regularly engaging in non-cycle exercise training (>1 $h \cdot d^{-1}$, 2-4 $d \cdot wk^{-1}$).

3.3. Determination of exercise intensity

In each study, the intensity of exercise for experimental trials was determined by completing an incremental maximal cycle exercise test. $\dot{V}O_{2max}$ was ascertained by completing a continuous incremental ramp test on an electrically braked cycle ergometer (Lode Excalibur Sport V2, Lode BV, Groningen, The Netherlands). Participants completed a five min steady state cycling at 50 W, and then continued to volitional exhaustion at a self-selected cycle cadence as the intensity of exercise increased 25-30 $W \cdot min^{-1}$. Ventilation, oxygen uptake ($\dot{V}O_2$) and carbon dioxide production ($\dot{V}CO_2$) were recorded continuously by an on-line gas analyzer (Cortex Metalyzer 2R, Cortex, Leipzig, Germany). Gas analysers were calibrated using gases whose concentrations had previously been determined by chemical analysis.

On the basis of the $\dot{V}O_2$-work rate relationship the power output was calculated for experimental tests. Appropriate exercise intensities (Table 3.2.) were determined from pilot tests; ensuring the balance between an intensity and duration of exercise

which would facilitate a rate of sweat loss equivalent to >2 % BM loss, whilst allowing the completion of exercise.

3.3.1. Gas Exchange Threshold

Chapters 5-8 employed the more common method of assigning exercise intensities as a percentage of $\dot{V}O_{2max}$. To advance research methods, Chapters 8 and 9 employed exercise intensities as a percentage of the difference between GET and $\dot{V}O_{2max}$; a method more likely to normalize a given intensity than the use of a percentage of $\dot{V}O_{2max}$ alone (Whipp & Rossiter, 2005). Since lactate threshold occurs at a range between 60-80 % $\dot{V}O_{2max}$, whilst exercising at 65 % $\dot{V}O_{2max}$, some partcipants may be below lactate threshold whilst others above. Similarly, whilst working at 85 % $\dot{V}O_{2max}$ some below may be below maximum lactate steady state, whilst others above. GET, synonymous with ventilation or lactate threshold, separates moderate and heavy domains more reliably.

GET was determined noninvasively by the V-slope method as the point at which carbon dioxide production [$\dot{V}CO_2$] began to rise at a more rapid rate than $\dot{V}O_2$. Respiratory compensation point was recorded as the time at which ventilation began to rise more rapidly than $\dot{V}CO_2$ and was determined subjectively from a plot of ventilation dependent on $\dot{V}CO_2$, with data smoothed by use of a 10 s moving average. $\dot{V}$-slope was ascertained by systematically dissecting the $\dot{V}CO_2/\dot{V}O_2$ data and plotting linear regression lines using all data from either side of that point. The $\dot{V}$-slope was recorded as the point at which the ratio of the largest standard error of the two lines and the distance from the intersection of the two lines to a single regression line drawn through the data set was minimised. To facilitate optimal detection of the GET and reduce the opportunity for premature fatigue, the work rate was increased by 30, rather than 25 $W \cdot min^{-1}$ during Chapters 8 and 9, in order that exhaustion was reached within 10-12 min (Buchfuhrer *et al.*, 1983).

3.4. Standard Measurements

The coefficient of variation [COV] for key measurement equipment are presented in table 3.3. Nude BM was measured before during and after exercise using scales sensitive to ± 10 g (Hampel, XWM-150K Electronic Scales, Hampel Electronics Co. Taiwan). Participants were provided with a towel to clear residual perspiration. Rectal temperature was measured by self-inserting a rectal thermistor (YSI temperature probe, Henleys Medical, Hertforshire) 10-12 cm past the anal sphincter. The thermistor probe was connected to a thermometer (± 0.01^{o}C; YSI Precision 4000, Henleys Medical, Hertforshire) which logged T_{Rec} throughout (rest and) exercise. Participants were also fitted with a HR monitor (Polar Electro, Finland), which logged HR during the same periods. Environmental data was recorded at intervals throughout the whole protocol.

Table 3.2. Summary of experimental designs

	Temperature	Relative Humidity	Exercise Intensity	Exercise Duration	Key Measurements
1	33 ± 0.2^{o}C	50 ± 3 %	60 % $\dot{V}O_{2max}$	60 min	BM, P_{Vol}, P_{Osm}, P_{Na}, U_{Osm}
2	32 ± 0.3^{o}C	60 ± 2 %	53 % $\dot{V}O_{2max}$	2 x 45 min	BM, U_{Osm}
3	33 ± 0.2^{o}C	50 ± 3 %	60 % $\dot{V}O_{2max}$	60 min	BM, P_{Vol}, P_{Osm}, P_{Na}, U_{Osm}
4	33 ± 0.2^{o}C,	50 ± 3 %	55 % $\dot{V}O_{2max}$	60 min	HR, SV, $\dot{Q}$, $\dot{V}O_{2}$, T_{Rec}, BM, P_{Vol}, P_{Na}, U_{Osm}
5	34 ± 0.2^{o}C	55 ± 3 %	10 % Δ 60 % Δ	45 min	HR, T_{Rec}, BM, P_{Vol}, P_{Osm}, P_{Na}, U_{Osm}, TTE
6	34 ± 0.3^{o}C	55 ± 4 %	10 % Δ 70 % Δ	60 min	HR, SV, $\dot{Q}$, $\dot{V}O_{2}$, T_{Rec}, BM, P_{Vol}, P_{Osm}, P_{Na}, U_{Osm}, TT

Signal Morphology-based Impedance Cardiography (PhysioFlow, Neumedx, Bristol) was used to measure HR, SV, and $\dot{Q}$, during Chapters 7 and 9. Six electrodes (Physio Flow, Manatec) were attached to each participant at rib, chest, neck, back. These sites were sterilized and shaved for optimal conductance. Duplicate measurements of resting blood pressure (Pro 100V2 dinamap) were taken prior to

exercising whilst seated on a cycle ergometer. $\dot{V}O_2$ was also measured during these studies using an on-line gas analyser (Cortex Metalyzer 2R, Cortex, Leipzig, Germany).

3.5. Drink Preparation

All drinks were blinded to the participant and served in concealed containers. Drinks were prepared in a laboratory environment using digital scales sensitive to 10 mg (Ohaus CS-200, Ohaus, New Jersey). For Chapters 8 and 9, sodium drinks were prepared in a powdered format by SIS Ltd. (Science in Sport, Ashwood Research Labs, Blackburn, UK), then converted into liquid 2 h prior to the start of the experimental session. Drinks were stored in a thermostatically controlled water bath (GD100, Grant Instruments Ltd, Cambridge) or refrigerated at 4^{o}C.

3.6. Blood and Urine Measurement and Analysis

A handheld digital refractometer (Pocket Osmocheck, Vitech Scientific, UK) was used to measure samples of urine. Accurate measurement of Hct and Hb was ensured by maintaining a standardised posture, arm position, and T_{Skn} (Sawka *et al.*, 1999). Hb microvettes were analyzed using a B-Hemoglobin photometer (Hemocue Ltd, Angelholm, Sweden). To determine Hct samples of whole blood were drawn into capillary tubes and centrifuged for 10 min and read using a microhematocrit tube reader (Hawksley & Sons, England). During Chapter 5-7 P_{Na} was ascertained by capillary blood samples drawn into epindorph tubes (20 μL) containing lithium heparin. For Chapters 8 and 9, venous blood samples taken from a suitable forearm vein were drawn into a vacutainer (4 mL) tube containing lithium heparin and immediately centrifuged for 10 min at 8^{o}C and 3000 rpm (IEC CEntra-4R centrifuge, UK). Plasma was separated from whole blood and frozen (-72^{o}C) for later analysis of P_{Osm} and P_{Na}. P_{Osm} was measured using the freezing point depression technique (Osmomat 030-D, Ganotec Gmbh, Berlin, Germany) and P_{Na} using an electrolyte analyzer employing the ion selective electrode principle (9180 Electrolyte Analyzer, Roche Diagnostics, Mannheim, Germany).

3.7. Performance

Chapters 8 and 9 included performance based measures. Taking physiological measurements, such as $\dot{Q}$, RPE or blood, during performance trials may interfere with the performance (Currell & Jeukendrup, 2008); thus the performance trials were undertaken in a separate bout of exercise subsequent to one in which physiological measurements were taken. The only measurements made during the performance test were HR and T_{Rec}, stipulated for health and safety. The preliminary bouts lasted 45-60 min, since this is a duration during which considerable CV_{Drift} occurs (Ekelund & Holmgren, 1964) and which is typical of aerobic exercise used for conditioning. It is a period of time which is replicable to similar studies e.g. (Wingo *et al.*, 2004; 2005; 2006a; 2006b).

3.8. Calculations

Whole body fluid loss was calculated from the reduction in BM during the trial. Sweat loss was estimated from the reduction in BM minus U_{Vol}, fluid intake and blood sample volume. Reductions in mass as a result of respiratory water, substrate oxidation and metabolic water gain, were assumed to be negligible and similar from trial to trial (Pugh *et al.*, 1967) and therefore were excluded from the calculations. Changes in P_{Vol} from baseline were estimated from changes in Hct and Hb using the following equation:

$$\text{Percentage change in } P_{Vol} = 100 \times [(Hb_b / Hb_t) \times ((1 - Hct_t) / (1 - Hct_b))] - 100\%)$$

(Dill & Costill, 1974)

where, *b* and *t* denote measurements at baseline and subsequent time intervals. Hb is in g·dL and Hct is a fraction. Hct corrected for trapped plasma and the venous-to-whole blood Hct excess, respectively (Dill & Costill, 1974). Whole body fluid loss was calculated from the reduction in BM during the trial. Sweat loss was estimated from the reduction in BM minus U_{Vol}, and fluid intake. For ease of comparison with prior investigation, U_{Osm} was estimated from urine specific gravity according to the regression equation:

$$U_{Osm} = 34248U_{SG} - 34282$$

Vitech Scientific, UK

The change in stroke volume [SV_{Drift}] and heart rate [HR_{Drift}] were calculated by subtracting the measurement of SV (or HR) upon termination of exercise, or the exercise bout, from the measurement of SV (or HR) made at 10 min into the exercise bout.

$$SV_{Drfit} = SV_{Final} - SV_{10\ min}$$

3.9. Statistical Anlyses

For each study data were analysed with SPSS version 13.0 with a significance level of $P < 0.05$ accepted for all statistical tests. Data are presented as mean ± SD with the exception of figure 8.1 and figure 9.1. which are presented as mean ± SE for clarity of presentation. Measures of fluid balance (sweat rate, U_{Vol}, U_{Osm} and BM); blood parameters (P_{Vol}, P_{Na} and P_{Osm}); perceptual ratings of taste and thirst; physiological measures (heart rate [HR], HR_{Drift}, SV, SV_{Drift}, $\dot{Q}$ and cardiac output drift [$\dot{Q}_{Drift}$], oxygen uptake [$\dot{V}O_2$]); ratings of perceived exertion [RPE]; and exercise performance (exercise capacity and time to exhaustion [TTE]) were analysed using analysis of variance [ANOVA] with repeated measures to test the significance of mean differences between fluid conditions, time, and their interaction. After a significant *F*-ratio, pairwise differences were identified by using Tukey's honest's significant difference *post hoc* procedure. Assumptions of sphrecity and homogeneity were tested using Maunchly's test and Levene's test, respectively.

Where appropriate, Pearson product-moment correlations (*r*) were used to analyse relationships between selected variables. For example, between thirst and taste ratings; RPE and HR; between changes in P_{Vol} [ΔP_{Vol}] and T_{Rec} [ΔT_{Rec}] and HR, and between P_{Na}, P_{Osm} and P_{Vol}.

Table. 3.3. Coefficient of Variation of key measurements

Measure	Tool	COV
Haematocrit	Microhematocrit tube reader (Hawksley & Sons, England)	1.2
Haemoglobin	Hemoglobin photometer (Hemocue Ltd, Angelholm, Sweden)	2.5
Plasma Osmolality	Osmomat 030-D (Ganotec Gmbh, Berlin, Germany)	1.4
Plasma Sodium Concentration	9180 Electrolyte Analyzer (Roche Diagnostics, Mannheim, Germany)	4.6
Urine Osmolality (Specific Gravity)	Pocket Osmocheck (Vitech Scientific, UK)	2.9
Heart Rate	Polar Heart Rate monitor (Polar Electro, Finland)	1.9

COV = Coefficient of Variation

Where appropriate multiple regression analysis was used to examine the strength of the predictors. In Chapter 5, it was used to examine the strength of predictors of palatability and in Chapter 8, it was used to examine the strength of predictors of time to exhaustion. All variables were transformed to standardized *z* scores to remove the variation between participants. Standardized *z* scores were calculated for each participant and each variable by using mean values. Prior to conducting each multiple regression analysis, the assumptions of normality and homoscedasticity of the dependent and predictor variables were confirmed. The possibility of multicollinearity between independent variables was examined with the VIF collinearity diagnostic. In all cases, VIF was ≈1.0, indicating that collinearity was not evident among the independent variables. The relative contribution of independent variables to the explained variance in TTE was determined from their standardized regression coefficient. The relative

contribution of independent variables to the explained variance in palatability was determined from their standardized regression coefficient [β]: Independent variable contribution to R^2 = (β for independent variable / $\sum \beta$ coefficients in equation) x R^2, where R^2 represents the adjusted R^2.

Chapter 4: Study 1

Inclusion of a very high sodium concentration within a sports drink accelerated recovery from exercise-induced dehydration.

4.1. Introduction

Since many athletes finish a race in a hypohydrated state (Noakes *et al.,* 2002), the restoration of fluid and electrolyte loss forms an essential part of recovery from exercise. The addition of sodium has been established as a prerequisite for effective rehydration (Shirreffs *et al.,* 1996), with the aim of minimising diuresis by preserving elevated P_{Na} and P_{Osm}. However there is no clear understanding of which concentration of sodium optimises rehydration, possibly because of differences in experimental protocols and fluid volumes employed.

A number of authors have reported reduced U_{Vol} and greater fluid restoration with increasing sodium concentration between 0 and 100 $mmol \cdot L^{-1}$ (Maughan & Leiper, 1995; Shirreffs *et al.,* 1996; Shirreffs & Maughan, 1998; Merson *et al.* 2008; Gonzalez-Alonso *et al.,* 1992). In these studies, positive fluid balance was only achieved when a volume of fluid equal to 150 % of fluid losses was ingested and then only with concentrations of sodium > 50 $mmol \cdot L^{-1}$. It has therefore become generally accepted that replacing 150 % of BM losses is paramount to achieve positive fluid balance (Mitchell *et al.,* 2000; Shirreffs *et al.,* 1996).

However, when allowed to drink *ad libitum* the rate of fluid intake adopted during recovery rarely reaches such volumes (Carter & Gisolfi, 1989; Adolph & Dill, 1938). Thus ingestion of 150 % of BM losses is not representative of athletic practice. Given that the U_{Vol} is inversely proportionate to drink sodium concentration (e.g. Merson *et al.,* 2008), the consumption of a high sodium concentration may reduce the volume of fluid required. Previously when a very high sodium concentration (127 $mmol \cdot L^{-1}$) was ingested, consumption of 100 % of fluid losses resulted in 90 % of fluid replacement within 2 h (Nielsen *et al.,* 1986). Thus complete fluid retention may be a possibility with a volume of fluid less than the commonly agreed volume equivalent to 150 % BM losses.

4.1.1. Aims & Hypotheses

The aim of this study was to investigate the effect of ingesting a very high sodium concentration, compared with a concentration similar to a regular sport drink (136 vs.15 $mmol \cdot L^{-1}$) on recovery from exercise-induced dehydration. Participants replaced 120 % BM losses of either drink in periodic feedings at 30 min intervals. It was hypothesised that ingestion of the high sodium solution will reduce U_{Vol} and accelerate restoration of fluid balance, and P_{Vol} in comparison with the regular sports drink. Secondly, that ingestion of the high sodium concentration will induce positive fluid balance within 3 h of recovery.

4.2. Materials and Methods

A single-blind, randomly controlled repeated measures design was employed. Eight male cyclists with mean ± SD, age: 27 ± 6 yr, body mass: 73.7 ± 7.0 kg and $\dot{V}O_{2max}$ 59 ± 7 $ml \cdot kg^{-1} \cdot min^{-1}$, volunteered to participate in the study. Participants initially completed an incremental maximal cycle exercise test [$\dot{V}O_{2max}$] for determination of exercise intensity (See 3.3.1). On the basis of the $\dot{V}O_2$-work rate relationship, the power output equivalent to 60 % $\dot{V}O_{2max}$ was calculated and assigned to subsequent tests. Subsequently, participants performed two experimental trials separated by > 3 d. The first trial was randomly allocated to the first participant and then alternated thereafter. Equal hydration was confirmed by similar pre-trial BM, U_{Osm} and P_{Na}.

4.2.1. Depletion Period

On arrival at the laboratory, each participant urinated and weighed themselves in the nude (± 10 g; Hampel, XWM-150K Electronic Scales, Hampel Electronics Co. Taiwan). Participants entered the environmental chamber that was set at (33 ± 0.2^{o}C, 50 ± 3 % relative humidity, $V_{\alpha} \approx 2.5$ $m \cdot s^{-1}$), mounted the cycle ergometer (Lode Excalibur Sport V2, Lode BV, Groningen, Netherlands) and remained seated for 15 min to ensure steady state P_{Vol} and constituents. A capillary blood sample was taken for determination of Hct, Hb and P_{Na}. Thirst was then self-assessed by marking a visual analogue scale. Subsequently participants completed 1 h cycling a power output of 185 ± 35 W, which

initially elicited 60 % $\dot{V}O_{2max}$. From pilot investigation it was known that this length and intensity of protocol would induce at least 2 % BM loss.

Immediately after exercise, participants showered and rested in a seated position at room temperature (22°C, 50 % relative humidity) for 30 min to allow the reversal of exercise-induced P_{Vol} shifts (Nose *et al.*, 1988a). At the end of this transition period, a capillary blood sample was taken and thirst was assessed before each participant urinated and weighed themselves in the nude. Total dehydration was calculated at this point; thus dehydration was based on the difference between the initial nude BM and the BM at the end of the transition period.

4.2.2. Rehydration Period

Immediately after the transition period (0 h), participants rehydrated by ingesting the first of six boluses of fluid each of a volume equivalent to 20 % of BM lost (345 ± 66 mL) during exercise (=120 % BM loss). Participants ingested either a regular low sodium sports drink ([LS] Lucozade Sport, GlaxoSmithKline) or high sodium sports drink ([HS] Lucozade Sport, plus 4.5 $g \cdot L^{-1}$ sodium-chloride and 7.7 $g \cdot L^{-1}$ sodium-citrate) drink. The relative osmolality, sodium and carbohydrate content of the drinks are listed in Table 4.1. Drinks were prepared in a laboratory environment using digital scales (± 10 mg; Ohaus CS-200, Ohaus, New Jersey) and refrigerated until 2 h before the test, then kept at room temperature. The mean drink temperature at the start of each session was 18 ± 0.3^{o}C. Drinks were supplied in concealed containers. At the end of each hour period, capillary blood sample was taken and ratings of thirst assessed before each participant urinated and weighed themselves in the nude. Following this the next drink was supplied. Fluid replacement was calculated by the measurements made at the end of each hour period.

4.2.3. Subjective ratings

Participants' subjective ratings of thirst were assessed by self-completed visual analogue scale ([VAS] Wewers & Lowe, 1990). The scale consisted of a horizontal line, 100 mm in length, anchored by word descriptors at each (e.g. Not thirsty, Very Thirsty). Participant marked on the line the point that they felt represented their perception of

their *current* state. The VAS score was determined by measuring in millimetres from the left hand end of the line to the participant's mark.

4.2.4. Blood and Urine analysis

All samples were measured in triplicate. Blood samples were drawn into epindorph tubes (20 µL) containing lithium heparin and centrifuged for 10 min at 3000 rpm (Micro Haematocrit, Analax Instruments, Hawksley & Sons, England). Plasma was separated from whole blood for analysis of P_{Na}. P_{Na} was measured using an electrolyte analyzer employing the ion selective electrode principle (9180 Electrolyte Analyzer, Roche Diagnostics, Mannheim, Germany). Hb microvettes were analyzed using a B-Hemoglobin photometer (Hemocue Ltd, Angelholm, Sweden). To determine Hct samples of whole blood were drawn into capillary tubes and centrifuged for 10 min and read using a microhematocrit tube reader (Hawksley & Sons, England). U_{Osm} was analyzed using a handheld refractometer (Pocket Osmocheck, Vitech Scientific, UK). U_{Vol} voided during exercise and rehydration was recorded on digital scales.

Table 4.1. Drink properties

	LS	HS
Osmolality ($mOsm \cdot kg^{-1}$)	290	393
Electrolyte Concentration ($mmol \cdot L^{-1}$)	15	136
Sodium Chloride ($g \cdot L^{-1}$)	0.6	4.5
Sodium Citrate ($g \cdot L^{-1}$)	-	7.7
Energy content ($kJ \cdot L^{-1}$)	410	410

4.3. Results

Pre-exercise BM (73.7 ± 7.0 kg; P = 0.99); U_{Osm} (237 ± 91 $mOsm \cdot kg^{1}$, P = 0.72); and P_{Na} (137.7 ± 0.7 $mmol \cdot L^{-1}$, P = 0.55) were similar between trials. There was a significant decrease in BM (1.7 ± 0.5 kg; P = 0.007) and increase in U_{Osm} (444 ± 123 $mOsm \cdot kg^{-1}$; P = 0.005) and P_{Na} (3.6 ± 0.5 $mmol \cdot L^{-1}$; P = 0.01) during exercise, but there

were no differences between trials ($P > 0.59$). Exercise induced a decrease in P_{Vol} of similar magnitude with both drinks ($P = 0.78$), resulting in a P_{Vol} of -4.4 ± 1.3 %. U_{Vol} was not significantly different between trials at 0 h (132 ± 42 mL, $P = 0.89$).

4.3.1. Plasma Sodium Concentration and Volume

Figure 4.1.A illustrates the changes in P_{Na}, at baseline, and during recovery. A two way ANOVA revealed a significant main effect for drink ($F_{(1, 7)} = 42.13$, $P < 0.001$), time ($F_{(4, 28)} = 88.32$, $P < 0.001$) and the interaction ($F_{(4, 28)} = 4.30$, $P = 0.01$) for P_{Na}. With HS, P_{Na} remained elevated from baseline throughout recovery ($P < 0.03$), whereas with LS, P_{Na} returned to baseline within 1 h (139.6 ± 0.6 $mmol \cdot L^{-1}$, $P = 0.08$), where it remained for the next two hours ($P > 0.05$).

Figure 4.1.B. illustrates the changes in P_{Vol}, at baseline, and during recovery. A two way ANOVA revealed a significant main effect for drink ($F_{(1, 7)} = 17.57$, $P = 0.004$), time ($F_{(4, 28)} = 72.65$, $P < 0.001$) and the interaction ($F_{(4, 28)} = 14.61$, $P < 0.001$) for percentage change in P_{Vol} from baseline. With HS P_{Vol} returned to baseline levels within 1 h and continued to increase above baseline during the next 2 h. With LS, P_{Vol} was inferior to baseline levels until 3 h into recovery. Thus there was a significant difference in P_{Vol} between drinks throughout recovery ($P < 0.02$).

Table 4.2. Relationship (r) between plasma sodium concentration and both urine osmolality and urine volume, during recovery.

	0 h	1 h	2 h	3 h	Mean
U_{Osm}	0.52^{a}	0.78^{b}	0.67^{b}	0.55^{a}	0.87^{b}
U_{Vol}	-0.67^{a}	-0.57^{a}	-0.62^{a}	$-0.81^{\mathbf{b}}$	-0.77^{b}

Significant relationships denotes by ^{a} ($P < 0.05$), ^{b} ($P < 0.01$).

4.3.2. Urine Osmolality and Volume

Figure 4.1.C illustrates the changes in U_{Osm}, at baseline, and during recovery. A two way ANOVA revealed a significant main effect for drink ($F_{(1, 7)} = 114.21$, $P < 0.001$), time ($F_{(4, 28)} = 57.96$, $P < 0.001$) and the interaction ($F_{(4, 28)} = 56.34$, $P < 0.001$)

for U_{Osm}. Whilst U_{Osm} remained elevated from baseline throughout recovery with HS ($P < 0.004$), it returned to baseline with 2 h with LS ($P = 0.10$). There was a significant difference in U_{Osm} between drinks throughout recovery ($P < 0.001$).

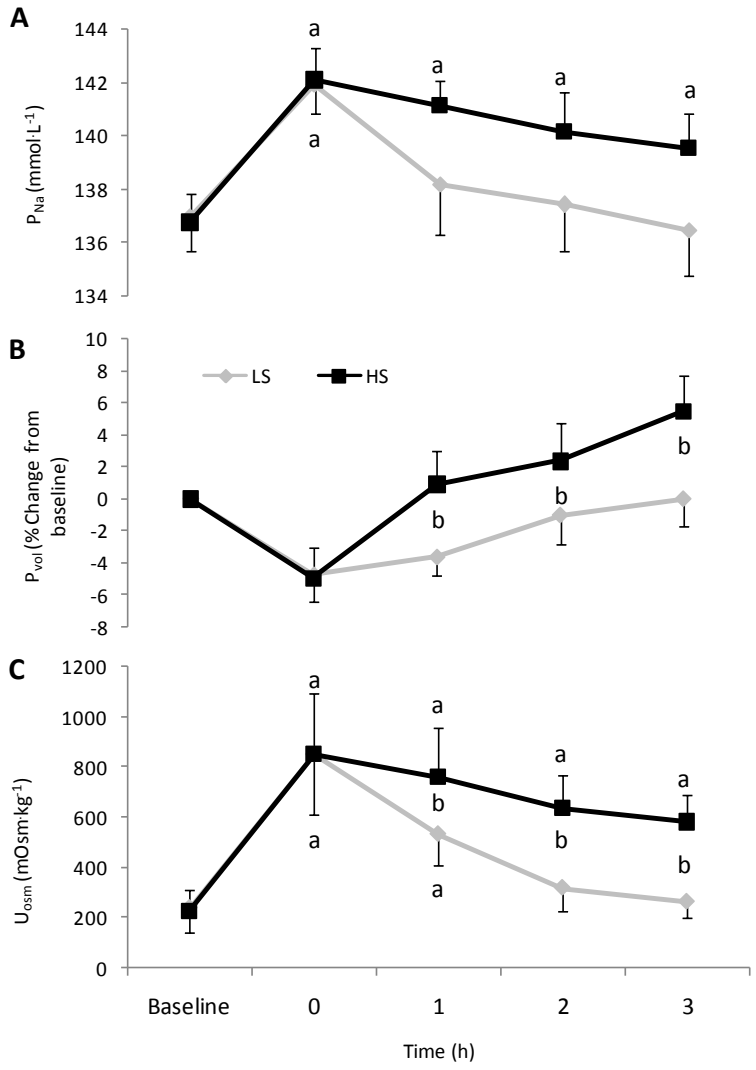

Figure 4.1. Plasma sodium concentration (A), plasma volume (B), and urine osmolality (C). Each variable was measured at baseline, and at the start and every hour during recovery.. [a] denotes significant difference from baseline, [b] denotes significant difference between drinks ($P \leq 0.05$).

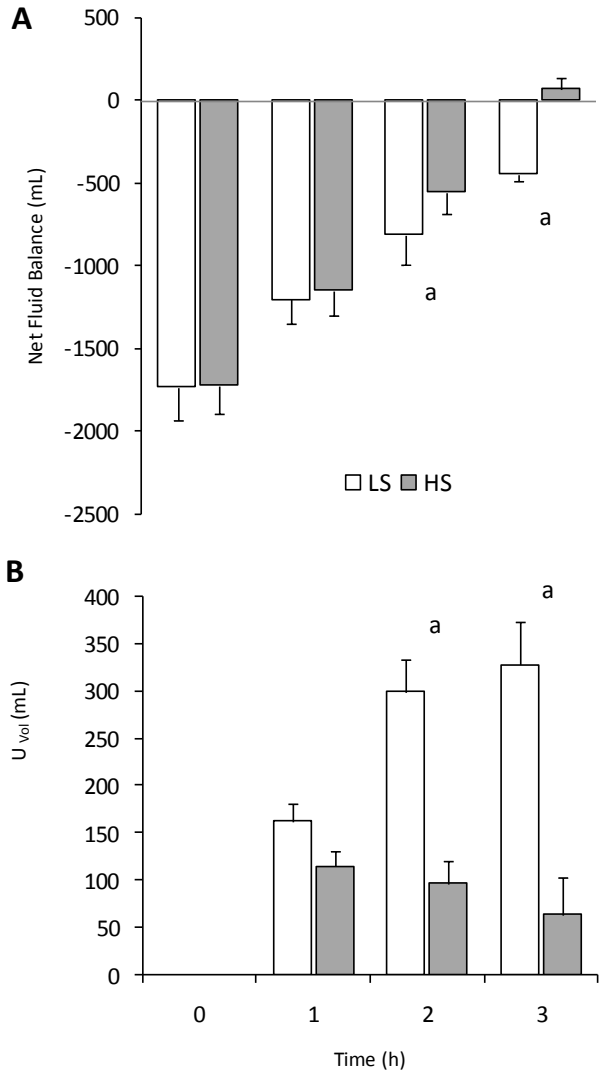

Figure 4.2. Net fluid balance (A) and urine volume (B). Measured at the start (0 h) and every hour during recovery. [a] denotes significant difference between drinks ($P < 0.05$).

Total U_{Vol} produced during recovery was significantly reduced with HS compared to the LS (453 ± 46 mL vs. 1106 ± 117 mL, $P < 0.001$). Analysis of U_{Vol} at hourly time intervals using a two way ANOVA revealed a significant main effect for drink ($F_{(1, 7)} = 114.21$, $P < 0.001$), time ($F_{(3, 21)} = 57.96$, $P < 0.001$) and the interaction ($F_{(3, 21)} = 56.34$, $P < 0.001$) for U_{Vol}. The U_{Vol} during recovery is illustrated in figure 4.2.B. U_{Vol} was similar between drinks at 1 h (154 ± 36, $P = 0.19$), but was significantly

reduced with HS at 2 h 96 ± 42 vs. 360 ± 53 mL, $P < 0.001$) and 3 h (82 ± 31 vs. 363 ± 58 mL, $P < 0.001$). Table 4.2. illustrates the relationships between P_{Na} and U_{Osm} and U_{Vol}, during recovery. P_{Na} was positively associated with U_{Osm} at 1, 2 and 3 h (weakest: $r = 0.52$, $P < 0.01$) and negatively U_{Vol} at 1, 2 and 3 h (weakest $r = 0.57$, $P < 0.006$) during recovery.

4.3.3. Fluid restoration

Figure 4.2.A. illustrates the changes in fluid restoration during recovery. A two way ANOVA revealed a significant main effect for drink ($F_{(1, 7)} = 54.11$, $P < 0.001$), time ($F_{(3, 21} = 112.82$, $P < 0.001$) and the interaction ($F_{(3, 21)} = 14.28$, $P = 0.01$) for fluid restoration. Ingestion of HS resulted in significantly greater restoration of fluid balance than LS throughout recovery ($P < 0.001$).

4.4. Discussion

The main findings of this study are that ingestion of a sports drink containing a high sodium concentration accelerated restoration of fluid balance and P_{Vol} compared with a regular low sodium sports drink. Secondly, and importantly, that the ingestion of a volume equivalent to just 120 % BM losses resulted in positive fluid balance within 3 h. Comparable studies have failed to demonstrate positive fluid balance in this time frame unless a volume equal to 150 % of BM losses was ingested (Mitchell *et al.,* 2000; Merson *et al.,* 2008; Saat *et al.,* 2002; Shirreffs *et al.,* 2007). The negative fluid balance demonstrated with the ingestion of the low sodium concentration supports prior investigation; even when 120-150 % of fluid losses are replaced low-moderate concentrations of sodium (3-60 $mmol \cdot L^{-1}$) frequently result in negative fluid balance after 3 h of recovery (Ismail *et al.,* 2007; Maughan *et al.,* 1995; Merson *et al.,* 2008; Saat *et al.,* 2002; Shirreffs *et al.,* 2007).

When left to their own devices, athletes rarely choose to ingest large volumes of fluid (Carter & Gisolfi, 1989; Adolph & Dill, 1938). Thus the ingestion of 150 % BM loss (2.5-3 L) may be an unrealistic and impractical strategy, especially when consumed within a short space of time. The demonstration of positive fluid balance with a volume equivalent to just 120 % BM loss is therefore highly significant and thus the strategy used in the present study represents a more practical post-exercise recovery than those

used previously (Mitchell *et al.,* 2000; Merson *et al*., 2008; Saat *et al.,* 2002; Shirreffs *et al.,* 2007). The possibility that this could be achived was formed from the findings of Nielsen *et al.* (1986) who demonstrated that 90 % fluid restoration was possible with the ingestion of just 100 % BM losses if the rehydration solution is highly concentrated with sodium (127 $mmol \cdot L^{-1}$).

4.4.1. Urine Output

The greater fluid restoration with HS compared with LS is attributed to the reduction in total U_{Vol} during recovery from 790 to 277 mL. Given a fluid intake of ~2.1 L, this equates to an increase in fluid retention from 62 % with LS, to 87 % with HS. The role of sodium in conserving fluid is well established (Maughan & Leiper, 1995; Merson *et al.,* 2008; Mitchell *et al.,* 2000; Shirreffs & Maughan, 1998; Shirreffs *et al.,* 1996; Wemple *et al.,* 1997), with systematic evaluations suggesting that the volume of fluid retained is inversely related to the sodium concentration of the drink. For example, during 6 h rehydration from a similar degree of dehydration as the present study, Shirreffs & Maughan (1998) reported a fluid retention of 60 % and 72 % with a sodium concentration of 50 and 100 $mmol \cdot L^{-1}$, respectively. The present study found a greater fluid retention (87 %) following the ingestion of a higher sodium concentration (136 $mmol \cdot L^{-1}$ Na). This finding confirms this inversely proportionate relationship and helps explain why this study and not others was able to achieve complete fluid retention within 3 h of recovery from exercise.

The regulation of urine production is controlled by the activation of the fluid-regulating hormone, AVP, in response to the elevated P_{Osm} which occurs following profuse sweat loss. The release of AVP stimulates antidiuresis, reabsorbing fluid back into the bloodstream, increasing the concentration of urine released. The ingestion of hypotonic fluids, such as LS, is known to reduce P_{Osm} and blunt the release of AVP (Takamata *et al.,* 1994), reducing the antidiuretic response and promoting fluid loss. In contrast the ingestion of high sodium concentrations, such as with HS, maintains an elevated P_{Osm} stimulating AVP release and sustaining the antidiuretic response (Maughan *et al.,* 1996). In the absence of measurements of AVP or P_{Osm}, in the present study P_{Na} and the commensurate changes in U_{Osm} provide a reliable surrogate for these responses. Despite differences in P_{Na} between drinks at each time point during recovery, statistically significant differences in U_{Vol} and U_{Osm} and net fluid balance were only

found at 2 h and 3 h. This delayed response may be a result of a lag in the time required for changes in P_{Na} to elicit the stimulation / suppression of AVP release and affect renal responses (Shirreffs *et al.*, 1998).

4.4.2. Drinking Strategy

Previous studies investigating the effect of sodium concentration on recovery from exercise have tended to employ one of two drinking strategies. The majority, undertaken by the same groups of authors, involves the ingestion of large volumes of fluid (100-150 % BM loss) within a relatively short space of time (0.5-1 h), followed by a period of monitoring lasting several hours (Maughan & Leiper, 1995; Merson, *et al.*, 2008; Shirreffs *et al.*, 1996; Shirreffs & Maughan, 1998). Whilst these strategies allow extended periods of observation, they do not represent practical applications for an athletic or occupational setting. Further, the consumption of large volumes of fluid in a short space of time, can blunt the release of AVP, and thus promote diuresis, even when hypohydrated (Shirreffs *et al.*, 2007). The other strategy, and that employed in the present study involves the ingestion of smaller volumes of fluid periodically during recovery, and as such represents a more realistic drinking regime for athletes. Furthermore, it is possible that periodic feeding promotes better fluid restoration than one-off drinking strategies, since Mitchell *et al.* (2000) demonstrated ~105 % fluid restoration following ingestion of 150 % fluid losses of two moderate sodium concentrations (25 and 50 $mmol \cdot L^{-1}$). Within the same time frame and with commensurate sodium concentrations, no study employing a one-off drinking strategy has demonstrated positive fluid balance. Of the studies employing periodic feedings, the presents study is the first to report positive fluid balance with ingestion of a volume of fluid less than 150 % BM losses.

4.5. Conclusion

The addition of a high sodium concentration to a sports drink (136 $mmol \cdot L^{-1}$) significantly accelerated the rate of rehydration of both fluid balance compared with the regular low sodium sports drink (15 $mmol \cdot L^{-1}$). The substantial reduction in total U_{Vol} from 790 mL to 276 mL with HS, compared to LS was critical in accelerated the rate of recovery achiving fluid restoration of 104 vs. 77% within 3 h of terminating exercise-

induced dehydration. The greater fluid retention with HS compared to LS confirms the inversely proportionate relationship between sodium content and diuresis previously reported (Maughan & Leiper, 1995; Merson *et al.,* 2008; Mitchell *et al.,* 2000; Shirreffs & Maughan, 1998; Shirreffs *et al.,* 1996; Wemple *et al.,* 1997). The most significant finding of the present study and that which separates it from previous investigation is that the complete restoration of fluid balance can be achieved with a volume of fluid equal to just 120 % of BM losses. These findings therefore offer an alternative strategy to the well-acknowledged belief that positive fluid balance can only be achieved with a volume of fluid ≥ 150 % BM losses. The study employed a practical drinking strategy involving periodic feedings with a smaller volume of fluid to what has previously been used to restore fluid balance. The drinking strategy therefore represents a highly efficient and practical procedure.

Chapter 5: Study 2

Changes in taste perceptions following exercise-induced dehydration

5.1 Introduction

The taste system acts as a "vigilant gatekeeper" to the body (Cowart, 2005), providing protection from what is harmful and encouraging the consumption of food/fluid items which satisfy physiological need. This transitory system responds to disturbances to homeostasis such as hunger (Yeomans, 1998), hyponatraemia (Beauchamp *et al.*, 1990; Huang & Yan, 2008) and dehydration (Appleton, 2005; Takamata *et al.,* 1994) by increasing the palatability of the food/drink specifically related to each disturbance. For example, in a state of hunger, energy rich food will be perceived most palatable and in a state of sodium loss, salty food will be perceived more palatable.

Exercise-induced dehydration, resulting from profuse sweating, stimulates transient shifts in fluid balance, which promotes hyperosmotic hypovolemia (Brandenberger *et al.*, 1986; Saltmarsh, 2001; Sawka *et al.*, 1984). This creates a physiological requirement for both low osmolality fluids, to restore P_{Osm}, and water and sodium to restore P_{Vol}. Following exercise-induced dehydration there is therefore a potential for a change in taste perceptions for both low osmolality fluids and sodium containing foods/fluids. In situations whereby there may be a physiological requirement for more than one substance, Cabanac (1971) suggests 'the food/fluid which satisfies the requirement to the greatest extent will be perceived most palatable'. Research shows that immediately after exercise, preservation of P_{Osm} takes priority over restoration of P_{Vol} (Hew-Butler *et al.*, 2006). Thus one would anticipate the low osmolality fluid, not highly salted food/fluid will be perceived more palatable.

5.1.1 Aims and Hypotheses

The aim of this study was to investigate the potential changes in taste perceptions for three drinks of different sodium concentrations, before and after two levels of exercise-induced dehydration (1.7% [Deh-45] / 3.2% [Deh-90]). It was

hypothesised that dehydration would induce an increase in the palatability of plain water and a decrease in the palatability of the isotonic and hypertonic drinks. It was also hypothesised that the magnitude of change in palatability for these drinks would be dependent upon the degree of dehydration.

5.2. Materials and Methods

Ten healthy, male cyclists with mean ± SD $\dot{V}O_{2max}$: 63 ± 5 $mL \cdot kg^{-1} \cdot min^{-1}$, age 29 ± 9 y and BM: 76 ± 6 kg, completed this single blind randomly controlled repeated measures design. Participants were non-smokers, and were screened for known taste defects. Participants initially completed an incremental maximal cycle exercise test for determination of exercise intensity (See 3.3.1.). On the basis of the $\dot{V}O_2$-work rate relationship, the power output equivalent to 53 % $\dot{V}O_{2max}$ was calculated and assigned to the experimental test.

5.2.1. Experimental Protocol

Each participant performed one experimental trial. Participants completed the same pre-exercise routine as for Chapter 4. For more details see section 4.3.1. Participants then entered the environmental chamber (32 ± 0.3°C, 60 ± 2 % relative humidity, $V_{\alpha} \approx 2.5\ m \cdot s^{-1}$), mounted the cycle ergometer (Lode Excalibur Sport V2, Lode BV, Groningen, Netherlands). Whilst seated on the ergometer, the first taste test session was conducted. Participants then completed two 50 min bouts of cycle exercise, which included a 5 min warm-up at 50 W, followed by 45 min at a mean power output (183 ± 21 W) that initially elicited 53 % $\dot{V}O_{2max}$. Bouts were separated by a 10 minute rest interval. Immediately following each bout participants completed a taste test whilst seated on the ergometer, then exited the chamber, urinated, towelled dry and weighed themselves nude. U_{Vol} was recorded and a sample analysed for U_{Osm}.

5.2.2. Taste Tests

Taste tests were carried out in an identical fashion at baseline and immediately after each 45 min bout (Deh-45 and Deh-90). Three orange-flavoured drinks were

tested: water ([W] 0 mmol·L^{-1} Na), low sodium ([LS] 27 mmol·L^{-1} Na) and high sodium ([HS] 126 mmol·L^{-1} Na). The sodium and energy content and osmolality, of the drinks are listed in Table 5.1. Drinks were prepared in a laboratory environment using digital scales (±10 mg; Ohaus CS-200, Ohaus, New Jersey), then refrigerated until 2 h before the test, when they were stored at room temperature. The mean drink temperature at the start of each session was 18 ± 0.4^{o}C. Drinks samples (50 mL) were supplied in concealed containers.

In a randomly assigned order, participants tasted three drinks using the 'sip, swish and spit' technique. Between each drink, participants' rinsed their mouths with deionized water. One taste questionnaire was completed for each of the three drink samples. The taste questionnaires measured; thirst, taste palatability, sweetness, saltiness and strength of drink on a nine-point scale anchored at each end (e.g. not palatable, very palatable) and sweetness, saltiness and strength on a three-point scale (e.g. too sweet, just right, not sweet enough).

Table 5.1. Drink Properties

	Sodium Content (mmol·L^{-1})	Energy (kJ·L^{-1})	Osmolality (mOsm·kg^{-1})
W	0	60	56
LS	27	410	290
HS	126	410	435

W =water, LS= Low sodium, HS = High sodium.

5.3. Results

Exercise induced a decrease in BM of 1.7 ± 0.3 % (P < 0.001) and 3.2 ± 0.3 % (P < 0.001), at Deh-45 and Deh-90, respectively. Table 5.2. illustrates the fluid losses incurred during exercise in more detail. U_{Osm} increased from 320 ± 95 mOsm·kg^{-1} to 596 ± 165 mOsm·kg^{-1} at Deh-45 (P < 0.001) and 802 ± 160 mOsm·kg^{-1} at Deh-90 (P < 0.001). The level of BM loss and U_{Osm} increase was significantly greater at Deh-90 than Deh-45 (P < 0.001). These findings show the exercise undertaken was sufficient to increase fluid requirements after both periods of exercise.

Table 5.2. Changes in body mass during exercise.

	Baseline (kg)	Loss (mL)	% Change
Baseline	76.0 ± 5.4		
Deh-45	74.7 ± 5.4	1307 ± 334	1.7 ± 0.3
Deh-90	73.6 ± 5.2	1112 ± 438	1.5 ± 0.4
Total		2419 ± 305	3.2 ± 0.4

5.3.1. Palatability

Figure 5.1. illustrates ratings of palatability at all time points. A two way ANOVA revealed a significant main effect for drink ($F_{(2,20)}$ = 11.11, P < 0.001), time ($F_{(2,20)}$ = 9.54, P < 0.001) and the interaction ($F_{(4,40)}$ = 5.68, P < 0.001) for ratings of palatability. LS was rated significantly more pleasant than either W (P = 0.003) or HS (P < 0.001) at baseline.

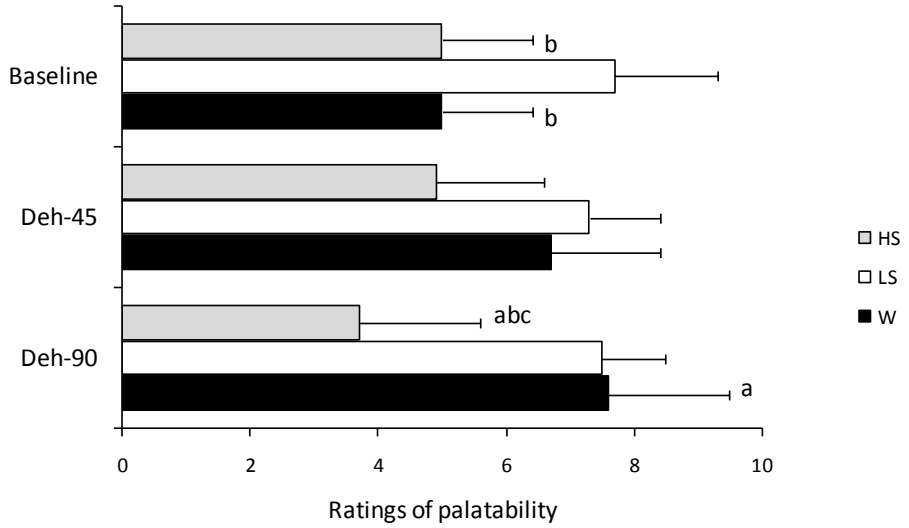

Figure 5.1. Changes in ratings of palatability during exercise. Rating measured for each drink (W = water, LS = low sodium, HS = high sodium) pre-exercise and at Deh-45 and Deh-90. [a] denotes significant difference from baseline value, [b] denotes significant difference from LS, [C] denotes significant difference W ($P \leq 0.05$).

There were no significant changes in palatability for either drink at Deh-45 ($P > 0.05$), however, at Deh-90, there was a significant increase in the palatability for W (7.6 ± 1.4, $P = 0.002$), and a significant decreased in the palatability HS (3.7 ± 1.9, $P < 0.004$), but no significant change with the LS (7.5 ± 1.0, $P = 0.73$).

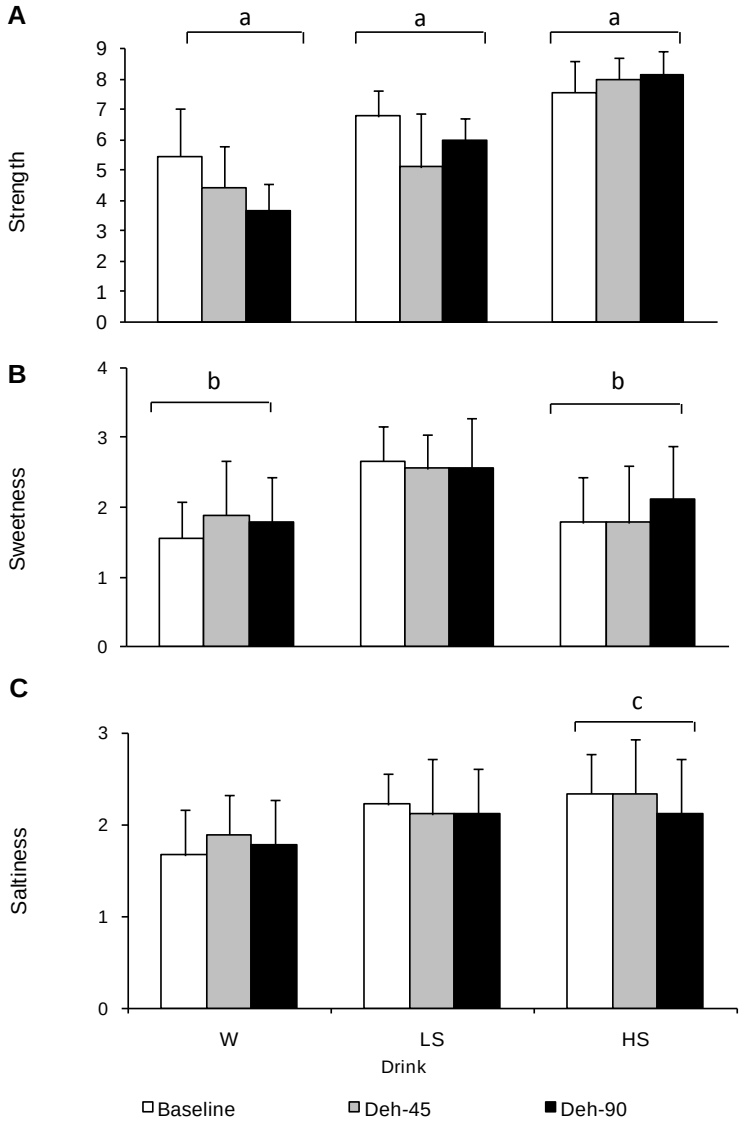

Figure 5.2. Ratings of strength (A), sweetness (B) and saltiness (C). Ratings measured for each drink (W =water, LS= low sodium, HS = high sodium), pre–exercise and at Deh-45 and Deh-90. [a] denotes significant difference between all drinks across all time points. [b] denotes significant difference from LS across all time points. [c] denotes significant difference from W across all time points ($P \leq 0.05$).

5.3.2. Other taste perceptions

Across all time points, LS (2.3 ± 1.4) was significantly sweeter than W (1.7 ± 1.4, $P = 0.007$) and HS (1.85 ± 1.4, $P = 0.05$). There were no significant differences in ratings of sweetness between HS and W ($P = 0.98$). Ratings of saltiness were significantly influenced by drink ($F_{(2,24)} = 3.89$, $P = 0.03$), but not time ($F_{(2,48)} = 0.51$, $P = 0.60$) or the interaction ($F_{(4,48)} = 0.62$, $P = 0.65$). Across all time points HS (2.3 ± 0.4) was rated significantly saltier than the W (1.8 ± 0.2, $P = 0.04$) but not LS (2.1 ± 0.4, $P = 0.95$). There was no significant difference between the W and LS drinks ($P = 0.15$).

Table 5.3. Summary of the regression analysis for the prediction of palatability at baseline, Deh-45 and Deh-90.

Time	Independent Variable	Constant	B	SE B	ß	R^2
Baseline	Saltiness		-1.26	0.31	-0.53	
	Sweetness	6.45	0.92	0.30	0.42	0.59#
Deh-45	Strength		-0.44	0.09	-0.58	
	Sweet	7.07	0.86	0.22	0.46	0.66*
Deh-90	Strength		-0.58	0.17	-0.61	.
	Saltiness	11.29	-0.27	0.46	-0.68	0.53*

Significant F-change, * $p < 0.001$, # $P \leq 0.05$.

Multiple regression analysis (Table 5.3, Figure 5.3) revealed that a two-component model incorporating saltiness and sweetness together explained 59 % of the variance in palatability at baseline ($P < 0.001$). At Deh-45, drink strength and sweetness together explained 66 % of the variance in palatability ($P = 0.005$). At Deh-90, drink strength and saltiness together explained 53 % of the variance in palatability ($P < 0.001$).

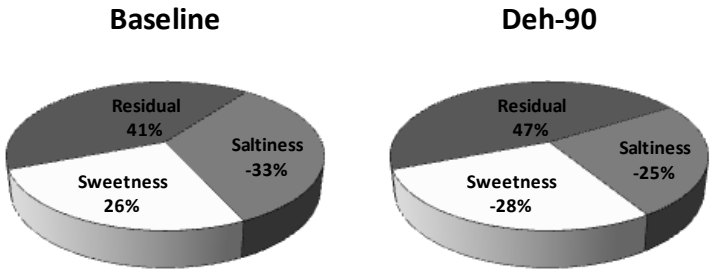

Figure 5.3. Relative contribution of the predicters of palatability.

Comment [j1]: Not new

5.4. Discussion

The aim of the study was to investigate potential changes in taste perceptions, following exercise-induced dehydration. The main findings of the study support our hypotheses that compared to baseline measures, dehydration equivalent to 3.2 % BM loss increased palatability of W by 52 % and decreased palatability of HS by 26 %, but rejects the hypothesis that there would be a decrease in the palatability of LS. The findings indicate that the level of dehydration is a critical determinant of changes in taste perceptions, with only minimal and non-significant changes found at Deh-45. In this sence the findings also support the hypothesis that the magnitude of change in palatability for these drinks would be dependent upon the degree of dehydration. This study also demonstrated a change in the relative importance of sweetness, saltiness and drinks strength to palatability. At baseline, saltiness and sweetness together explained 59 % of the variance in palatability, whilst at Deh-90, saltiness and drink strength together explained 53 % of the variance in palatability.

It is well established that disturbances to homeostais both long-term and short-term result in changes to the perception of taste and flavour and of ingestive behaviour (Beauchamp *et al.*, 1990; Huang & Yan, 2008; Yeomans, 1998). Less is known regarding the effect of exercise-induced dehydration on taste behaviour with just a handful of published studies, some of which fall short of the upmost rigours of scientific scuitany (Appleton, 2005; Lesham *et al.*, 1999; King *et al.*, 1999; Takamata *et al.*, 1994). Thus the main findings of the present study add to the limited knowledge we have regarding ingestive behaviour following exercise and provide support those which have previously found a relationship between physiological need and taste perceptions.

Exercise-induced dehydration, resulting from profuse sweating, stimulates transient shifts in fluid balance, which promotes hyperosmotic hypovolemia (Brandenberger *et al.*, 1986; Saltmarsh, 2001; Sawka *et al.*, 1984). This creates a physiological requirement for both low osmolality fluids, to restore P_{Osm}, and water and sodium to restore P_{Vol}. Following exercise-induced dehydration there is therefore a potential for a change in taste perceptions for both low osmolality fluids and sodium containing foods/fluids. In situations whereby there may be a physiological requirement for more than one substance, Cabanac (1971) suggests 'the food/fluid which satisfies the requirement to the greatest extent will be perceived most palatable'. Research shows that immediately after exercise, preservation of P_{Osm} takes priority over restoration of P_{Vol} (Hew-Butler *et al.*, 2006). The changes in taste perceptions demonstrated in the present study would suggest the restoration of P_{Osm} has been prioritised here.. The specific increase in palatability for the water, and decrease in palatability of HS, is supportive of the need to replace fluid over electrolytes immediately after exercise. On the basis that it would satisfy the need to reduce elevated P_{Osm} to the greatest extent, water was perceived more pleasant than in a situation of fluid balance, prior to exercise. Since water was perceived more pleasant because of an increase in its need, it is reasonable to assume that the decrease in palatability of HS was a result of a reduction in its requirement.

An increase in palatability of plain water has previously been demonstrated after exercise (Takamata *et al.*, 1994; Appleton *et al.*, 2005; King *et al.*, 1999; Farleigh *et al.*, 1987). For example, Takamata *et al.* (1994) demonstrated an increase in palatability of plain water, but not of 10 different sodium concentrations, immediately after 7 h intermittent exercise in the heat. However, it is not known whether the increase in palatability of W, and the decrease in palatability for HS was a result of the sodium content, osmolality or both. After approximately 45 min running at 70 % $\dot{V}O_{2max}$, King *et al.* (1999) reported a ~30 % increase in palatability of plain water, but did not find a significant change in the palatability of two sweetened drinks, despite the same sodium concentration (trace). This suggests that drink osmolality over drink sodium content has the greatest influence on perceptions of palatability following exercise. These findings are confirmed by Appleton (2005) who demonstrated an increase in palatability of three low sodium (< 18.5 $mmol \cdot L^{1}$)/ hypotonic drinks (<121 $mOsm \cdot kg^{-1}$) following 1 h gym exercise. There was no change in palatability of a hypertonic drink (418 $mOsm \cdot kg^{-1}$) despite a similar low sodium concentration (9 $mmol \cdot L^{-1}$). As yet no previous study has

demonstrated a decrease in palatability following dehydration. Appleton (2005) found no change in palatability for two hypertonic drinks (418 and 489 $mOsm \cdot kg^{-1}$) after 50 min mild exercise, whilst after 7 h intermittent exercise in the heat, Takamata *et al.* (1994) only reported a tendency for decreased palatability and only in the two highest of ten different sodium concentrations. Nevertheless, our findings support the relationship between physiological requirement and palatability.

The present findings showed that a level of dehydration between 1.7 and 3.2 % is necessary to induce significant changes in palatability. In contrast, Appleton reported an increase in palatability of three hypotonic drinks after a sweat loss of just 0.4 %. These findings are surprising given that osmotic thirst is not normally associated with such a small sweat loss (Robertson, 1984). A change in palatability is unlikely to occur whilst thirst has yet to be stimulated. The contrasting findings might be explained by the training status of participants in each study. The present study was the first to investigate palatability following exercise-induced dehydration in endurance-trained cyclists who are likely more physiologically adept at coping with sweat loss than the untrained participants employed in the previous studies (Appleton, 2005; Takamata *et al.*, 1994). Those athletes regularly subjected to situations eliciting dehydration may have a suppressed sensitivity to fluid balance disturbances or have renal systems more proficient at conserving fluid loss before fluid intake behaviours are necessary. The findings of the present study thus provide a more practical application to trained athletes, than previous studies which recruited untrained participants.

An interesting finding of the present study was the change in the relative importance of sweetness, saltiness and drink strength to palatability. Drink strength was considered a measure of drink osmolality. Drink palatability has a strong positive association with sweetness (deGraaf & Zandstra, 1999), whilst a high salt intake is generally regarded as unpleasant. In support of this association, saltiness and sweetness were equally strong predictors of palatability at baseline, together explaining 59 % of the variance. However, following 3.2 % dehydration, sweetness ceased to be a significant predictor of palatability. Instead drink strength, along with saltiness together explained 53 % of the variance in palatability. This study is the first to acknowledge the importance of drink strength and osmolality, relative to other taste properties, following dehydration. Previously, Appleton (2005), demonstrated increases in palatability which were synonymous with increased ratings of sweetness, thus it is unclear whether the changes in palatability were a function of osmolality or sweetness. It is important to

note that the present study does not suggest sweetness is an unimportant characteristic of palatability following exercise, but in this instance, highlights the greater importance of saltiness and osmolality. The change in predictors of palatability as a function of the level of dehydration is further indication of the relationship between physiological requirement and palatability.

A limitation of the study is that only subjective measures of palatability were assessed. From a practical viewpoint, behavioural measures are also valid (Wilmore *et al.*, 1998*;* Szlyk *et al.*, 1989). Since behavioural and subjective measures of palatability are closely related, and because behaviour can be influenced by other variables, such as mouth-feel (Brunstrom, 2002) or gastrointestinal discomfort (Lepkovsky *et al.*, 1957), it was not included as a measured variable. A second limitation of the study is that change in BM was the measure of disturbances to homeostasis. Although it is not the most precise surrogate for body fluid balance (Hew-Butrler *et al.,* 2006) it is the most common measure within the field of hydration (Coyle, 2004) and is a practical method utilized by coaches and athletes.

5.5. Conclusion

In conclusion, exercise-induced dehydration equivalent to 3.2 %, but not 1.7 % induced an increase in palatability of the W and a decrease in palatability of HS. Furthermore, dehydration increased the relative importance of drink strength as a predictor of palatability. These findings support the relationship between physiological requirement and palatability. In the physiological situation of a requirement to restore fluid balance, only the fluid which satisfied the need to the greatest extent increased in palatability. The fluid which hindered the restoration of fluid balance was rated less palatable. These findings further the understanding of the contribution of osmolality to palatability following dehydration, and may assist the determination of fluid intake strategies required to optimize recovery from dehydration.

Chapter 6 – Study 3

The relationship between body fluid balance and taste perceptions during recovery from exercise-induced dehydration

6.1. Introduction

When exercise in the heat is prolonged, substantial sweat secretion results in a significant loss of water and electrolytes, promoting a state of hyperosmotic hypovolemia. The magnitude of these responses is minimised by the conservation of fluid and sodium, in urine and sweat. However complete restoration requires the ingestion of fluid and electrolytes. Whilst these physiological responses to exercise-induced dehydration are generally well acknowledged, less is known about the effect these physiological needs have on taste perceptions.

A change in the palatability of various tastants has been demonstrated following exercise. For example, Horio and Kawamura (1998) demonstrated an increase in the palatability of a range of intensities of sweet and sour, but not salty, bitter, or umami-flavoured solutions, following 30 min of exercise at 50 % $\dot{V}O_{2max}$. King *et al.* (1999) demonstrated an almost 30 % increase in pleasantness ratings of water, but just a ~10 % increase in pleasantness of two sweetened solutions (no significant main effect for sweetness). Similarly, Appleton (2005) demonstrated an increase in the pleasantness for water and two other lower osmolality drinks after exercise.

Only one study has looked at the changes in taste perceptions over a continued period of time following exercise (Takamata *et al.,* 1994). Following 7 h intermittent exercise in the heat, Takamata *et al.* (1994) demonstrated an immediate increase in the palatability of water, but no change in the rating of palatability for 10 sodium solutions. Three hours into recovery with *ad libitum* water intake, ratings of palatability for water normalised, whilst ratings of palatability for several intensities of sodium in water increased. This occurred at the same point at which complete restoration of P_{Osm} and P_{Na} had been achieved. In contrast, Lesham *et al.* (1999) demonstrated an increase in salt added to soup, but not sugar added to tea, within 30 min of terminating 1 h of mild exercise. The level of sodium loss appears to be a critical factor in the timescale differences for changes in sodium palatability, and may explain the discrepancy in

findings between these two studies. However, as with many studies investigating taste perceptions, Lesham *et al.* (1999) failed to report any measures of fluid balance.

6.1.1. Aims and Hypotheses

The aim of the present study was to investigate the relationship between body fluid balance and changes in taste perceptions during 3 h recovery from a mild sodium loss induced by exercising for 1 h at 60 % $\dot{V}O_{2max}$. Fluid balance was manipulated by rehydrating with two concentrations of sodium (reh-A: 15 mmol $Na^{+} \cdot L^{-1}$; reh-B: 136 mmol $Na^{+} \cdot L^{-1}$). It was hypothesised that exercise-induced dehydration would result in an increase in palatability of water and a decrease in the palatability of two sodium solutions. It was also hypothesised that once fluid balance was achieved an increase in the palatability for the two sodium solutions would be demonstrated.

6.2. Materials and Methods

Eight males (age: 27 ± 6 yr, body mass: 74 ± 7 kg and $\dot{V}O_{2max}$: 59 ± 7 $ml \cdot kg^{-1} \cdot min^{-1}$), completed this single-blind, randomly controlled repeated measures design. Participants initially completed an incremental maximal cycle exercise test ($\dot{V}O_{2max}$) for determination of exercise intensity (see 3.1. General Methods). On the basis of the $\dot{V}O_2$-work rate relationship, the power output equivalent to 60 % $\dot{V}O_{2max}$ was calculated and assigned to subsequent tests.

6.2.1. Experimental Trials

Each participant performed two experimental trials separated by > 3 d. The first trial was randomly allocated to the first participant and then alternated thereafter. Equal hydration was confirmed by similar pre-trial BM, U_{Osm} and P_{Na}.

6.2.2. Depletion Period

On arrival at the laboratory, each participant urinated and weighed themselves in the nude (± 10 g; Hampel, XWM-150K Electronic Scales, Hampel Electronics Co.

Taiwan). Participants entered the environmental chamber that was set at (33 ± 0.2^{o}C, 50 ± 1 % relative humidity, $V_{\alpha} \approx 2.5$ m·s^{-1}), mounted the cycle ergometer (Lode Excalibur Sport V2, Lode BV, Groningen, Netherlands) and remained seated for 15 min to ensure steady state P_{Vol} and constituents. A capillary blood sample was taken for determination of Hct, Hb and P_{Na}. A taste-test session was then conducted. Subsequently participants completed 1 h cycling a power output of 185 ± 35 W, which initially elicited 60 % $\dot{V}O_{2max}$. From pilot investigation it was known that this length and intensity of protocol would induce at least 2 % BM loss.

Immediately after exercise, participants showered and then rested in a seated position at room temperature environment (22°C, 50 % relative humidity) for 30 min to allow the reversal of the exercise-induced P_{Vol} shifts (Nose *et al.*, 1988a). At the end of this transition period, capillary blood sample was then taken; a taste-test session conducted and each participant urinated and weighed themselves in the nude. Total dehydration was calculated at this point; thus dehydration was based on the difference between the initial nude BM and the BM at the end of the transition period.

6.2.3. Rehydration Period

Immediately after the transition period (0 h), participants rehydrated by drinking one of two rehydration solutions of a volume equal to 120 % of BM losses (2076 ± 95 mL) in six equal boluses (345 ± 45 mL) one every 30 min. Participants ingested either a low sodium ([reh-A] Lucozade Sport, GlaxoSmithKline) or high sodium ([reh-B] Lucozade Sport, plus 4.5 g·L^{-1} sodium-chloride and 7.7 g·L^{-1} sodium-citrate) drink. The relative osmolality, sodium and carbohydrate content of the drinks are listed in Table 6.1. Drinks were prepared in a laboratory environment using digital scales (± 10 mg; Ohaus CS-200, Ohaus, New Jersey) and refrigerated until 2 h before the test, then kept at room temperature. The mean drink temperature at the start of each session was 18 ± 0.3^{o}C. Drinks were supplied in concealed containers. At the end of each hour period, capillary blood sample was then taken; a taste test session conducted and each participant urinated and weighed themselves in the nude. Following this the next drink was supplied. This process was repeated six times until 3 h post transition period.

6.2.4. Subjective ratings

Participants' subjective ratings of thirst and palatability for water [W], LS and HS were assessed by self-completed VAS questionnaire (Wewers & Lowe, 1990). The scale consisted of a horizontal line, 100 mm in length, anchored by word descriptors at each (e.g. Not thirsty, Very Thirsty). Participant marked on the line the point that they felt represented their perception of their *current* state. The VAS score was determined by measuring in millimetres from the left hand end of the line to the participant's mark.

Table 6.1. Drink properties

	W	LS / reh-A	HS / reh-B
Osmolality ($mOsm \cdot kg^{-1}$)	48	290	393
Electrolyte Conc. ($mmol \cdot L^{-1}$)	0	15	136
Sodium Chloride ($g \cdot L^{-1}$)	-	0.6	4.5
Sodium Citrate ($g \cdot L^{-1}$)	-	-	7.7
Energy content ($kJ \cdot L^{-1}$)	-	410	410

LS / reh-A= Low sodium, HS / reh-B = High sodium.

6.2.5. Blood and Urine analysis

Blood and urine were analysed using the same methods as outlined in section 4.2.4.

6.3. Results

Pre-exercise BM (73.7 ± 7.0 kg; P = 0.99); U_{Osm} (237 ± 91 $mOsm \cdot kg^{1}$, P = 0.72); and P_{Na} (137.7 ± 0.7 $mmol \cdot L^{-1}$, P = 0.55) were similar between trials. There was a significant decrease in BM (1.7 ± 0.5 kg; P = 0.007) and increase in U_{Osm} (444 ± 123 $mOsm \cdot kg^{-1}$; P = 0.005) and P_{Na} (3.6 ± 0.5 $mmol \cdot L^{-1}$; P = 0.01) during exercise, but there were no differences between trials (P > 0.59). Exercise induced a decrease in P_{Vol} of similar magnitude with both drinks (P = 0.78), resulting in a P_{Vol} of -4.4 ± 1.3 %. U_{Vol} was not significantly different between trials at 0 h (132 ± 42 mL, P = 0.89).

6.3.1. Plasma volume and constitutes

Figure 6.1A illustrates the changes in P_{Na}, at baseline, and during recovery. A two way ANOVA revealed a significant main effect for drink ($F_{(1, 7)} = 42.13$, $P < 0.001$), time ($F_{(4, 28)} = 88.32$, $P < 0.001$) and the interaction ($F_{(4, 28)} = 4.30$, $P = 0.01$) for P_{Na}. With HS, P_{Na} remained elevated from baseline throughout recovery ($P < 0.03$), whereas with LS, P_{Na} returned to baseline within 1 h (139.6 ± 0.6 mmol·L^{-1}, $P = 0.08$), where it remained for the next two hours ($P > 0.05$).

Figure 6.1B. illustrates the changes in P_{Vol}, at baseline, and during recovery. A two way ANOVA revealed a significant main effect for drink ($F_{(1, 7)} = 17.57$, $P = 0.004$), time ($F_{(4, 28)} = 72.65$, $P < 0.001$) and the interaction ($F_{(4, 28)} = 14.61$, $P < 0.001$) for percentage change in P_{Vol} from baseline. With HS P_{Vol} returned to baseline levels within 1 h and continued to increase above baseline during the next 2 h. With LS, P_{Vol} was inferior to baseline levels until 3 h into recovery. Thus there was a significant difference in P_{Vol} between drinks throughout recovery ($P < 0.02$).

Table 6.2. Relationship (r) between plasma sodium concentration and both urine osmolality and urine volume, during recovery.

	0 h	1 h	2 h	3 h	Mean
U_{Osm}	0.52^a	0.78^b	0.67^b	0.55^a	0.87^b
U_{Vol}	-0.67^a	-0.57^a	-0.62^a	-0.81^b	-0.77^b

Significant relationships denotes by ^{a} ($P < 0.05$), ^{b} ($P < 0.01$).

6.3.2. Urine Osmolality and Volume

Figure 6.1C. illustrates the changes in U_{Osm}, at baseline, and during recovery. A two way ANOVA revealed a significant main effect for drink ($F_{(1, 7)} = 114.21$, $P < 0.001$), time ($F_{(4, 28)} = 57.96$, $P < 0.001$) and the interaction ($F_{(4, 28)} = 56.34$, $P < 0.001$) for U_{Osm}. Whilst U_{Osm} remained elevated from baseline throughout recovery with HS ($P < 0.004$), it returned to baseline with 2 h with LS ($P = 0.10$). There was a significant difference in U_{Osm} between drinks throughout recovery ($P < 0.001$).

Total U_{Vol} produced during recovery was significantly reduced with HS compared to the LS (453 ± 46 mL vs. 1106 ± 117 mL, $P < 0.001$). Analysis of U_{Vol} at hourly time intervals using a two way ANOVA revealed a significant main effect for drink ($F_{(1, 7)} = 114.21$, $P < 0.001$), time ($F_{(3, 21)} = 57.96$, $P < 0.001$) and the interaction ($F_{(3, 21)} = 56.34$, $P < 0.001$) for U_{Vol}. U_{Vol} was similar between drinks at 1 h (154 ± 36, $P = 0.19$), but was

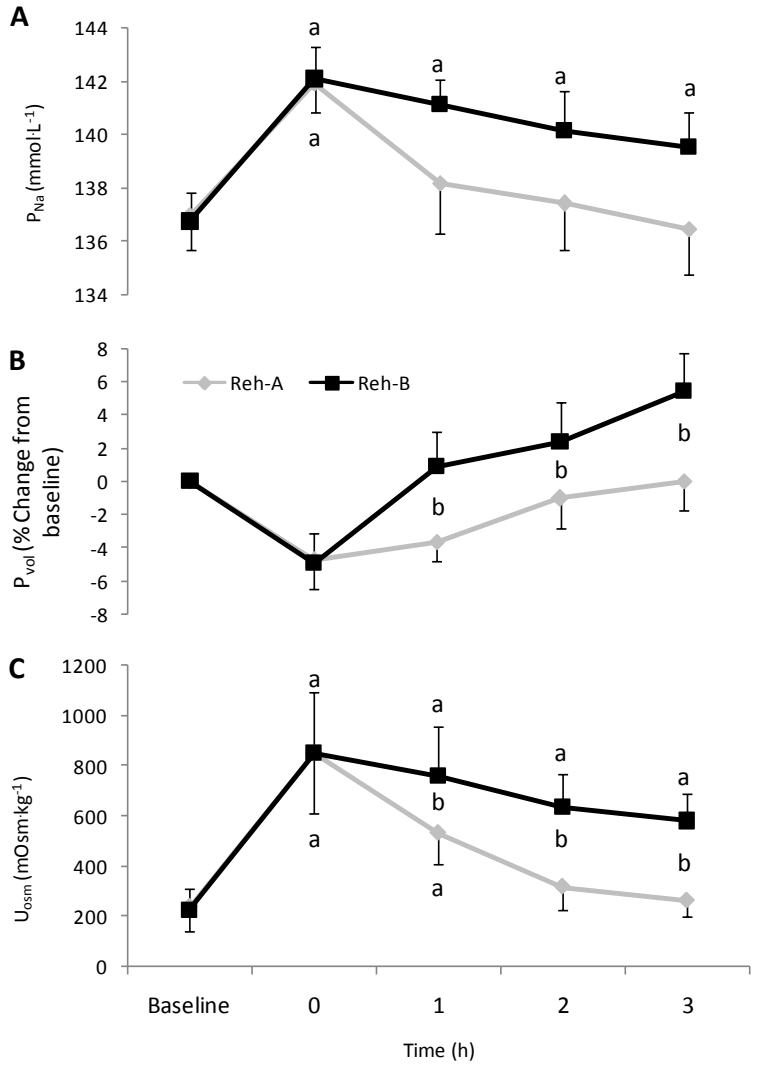

Figure 6.1. Plasma sodium concentration (A), plasma volume (B), and urine osmolality (C). Each variable was measured at baseline, and at the start and every hour during recovery.. [a] denotes significant difference from baseline, [b] denotes significant difference between drinks ($P \leq 0.05$).

significantly reduced with HS at 2 h 96 ± 42 vs. 360 ± 53 mL, $P < 0.001$) and 3 h (82 ± 31 vs. 363 ± 58 mL, $P < 0.001$). Table 6.2. illustrates the relationships between P_{Na} and U_{Osm} and U_{Vol}, during recovery. P_{Na} was positively associated with U_{Osm} at 1, 2 and 3 h (weakest: $r = 0.52$, $P < 0.01$) and negatively U_{Vol} at 1, 2 and 3 h (weakest $r = 0.57$, $P < 0.006$) during recovery.

6.3.3. Thirst

Figure 6.2 shows subjective ratings of thirst. A two way ANOVA revealed a significant main effect for drink ($F_{(1, 7)} = 39.53$, $P < 0.001$), time ($F_{(4, 28)} = 43.20$, $P < 0.001$) and the interaction ($F_{(4, 28)} = 3.30$, $P = 0.02$) for ratings of thirst. Ratings of thirst between rehydration conditions were similar at baseline (37 ± 10 mm, $P = 0.72$). Thirst ratings increased markedly during the depletion period in both conditions (81 ± 9 mm, $P = 0.87$). With reh-A, ratings of thirst remained elevated from baseline at 1 h ($P < 0.001$) returning to baseline within 2 h ($P = 0.24$). With reh-B, thirst also remained elevated from baseline at 1 h ($P = 0.02$).

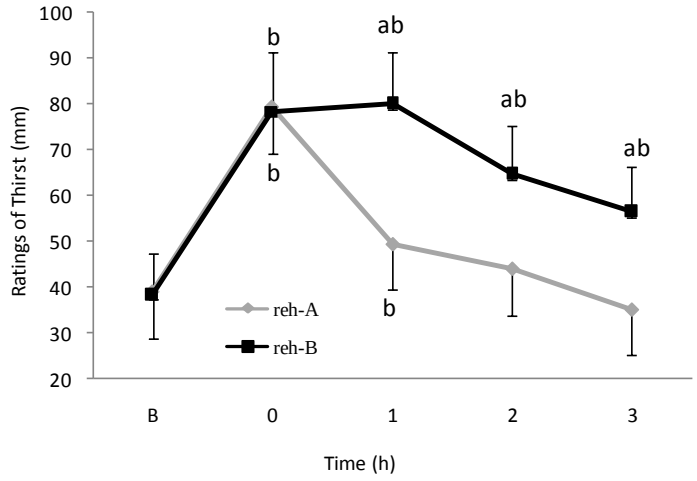

Figure 6.2. Ratings of thirst. Ratings measured during each rehydration condition (reh-A and reh-B). [a] denotes a significant difference between conditions. [b] denotes a signifcant difference from baseline ($P \leq 0.05$).

6.3.4. Palatability

Figure 6.3. shows the palatability ratings for three sampled drinks during both rehydration conditions, before exercise and during rehydration. For analysis of drink palatability, a three-way ANOVA revealed a significant main effect for rehydration condition ($F_{(1, 7)} = 12.68$, $P = 0.03$), sample drink ($F_{(2, 14)} = 479.94$, $P < 0.001$), time ($F_{(4, 28)} = 14.45$, $P < 0.001$) and the interactions of rehydration condition x sample drink ($F_{(2, 14)} = 57.65$, $P < 0.001$), rehydration condition x time ($F_{(4, 28)} = 4.91$, $P = 0.004$), sample drink x time ($F_{(8, 56)} = 35.61$, $P < 0.001$), and rehydration condition x sample drink x time ($F_{(8, 56)} = 37.12$, $P < 0.001$).

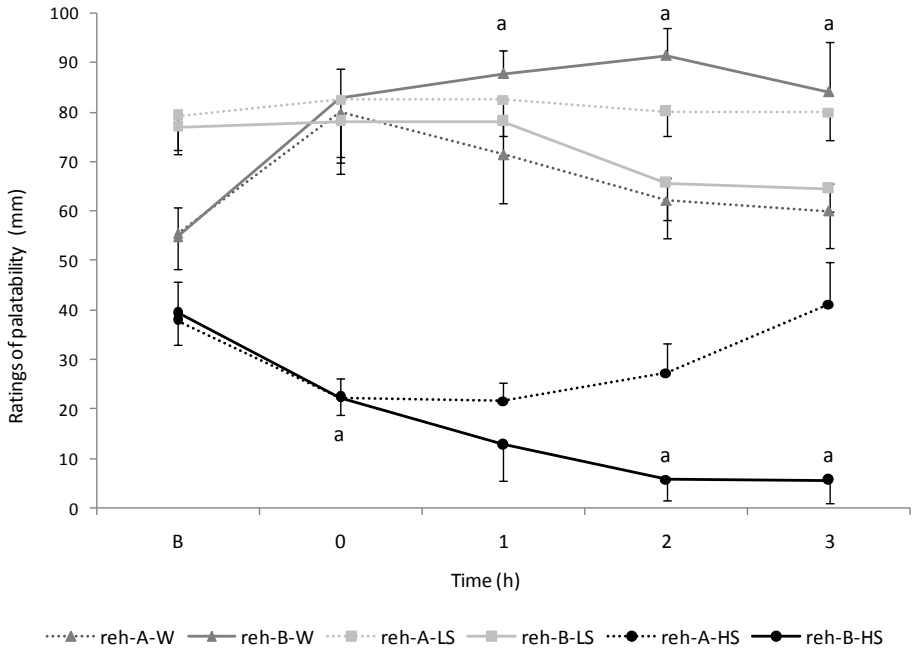

Figure 6.3. Ratings of palatability . During both rehydration conditions (reh-A vs. reh-B), for three samples of drink (W, LS, HS), at baseline (B) and during rehydration (0-3 h). [a] denotes a significant difference between rehydration conditions (reh-A vs. reh-B) for a given drink sample ($P \leq 0.05$). For clarity of presentation, significant differences from baseline are not illustrated, but described in text.

At baseline ratings of palatability were equal between reh-A and reh-B with all drink samples (W, LS, and HS; $P > 0.20$). Palatability ratings for W were essentially neutral before exercise (55 ± 7 mm). The W was rated significantly less pleasant than LS (76 ± 7 mm, $P = 0.02$), and significantly more pleasant than HS (37 ± 8 mm, $P =$

0.01). With both reh-A and reh-B, palatability ratings significantly increased for W ($P < 0.001$) and significantly decreased for HS ($P < 0.02$) but were unchanged with LS ($P = 0.89$). At 0 h, there were no differences in ratings of palatability for any sample between rehydration conditions ($P > 0.12$).

With reh-A, the palatability ratings for W gradually decreased during recovery, and returned to baseline levels within 2 h ($P = 0.37$). Palatability for HS remained at sub baseline levels until 3 h ($P = 0.65$). Palatability for LS was unchanged ($P = 0.78$). With reh-B, palatability for W gradually increased during rehydration, peaking at 2 h and remaining significantly elevated from baseline throughout ($P < 0.001$). Palatability of LS remained at baseline levels for the first hour ($P = 0.34$) then decreased below baseline levels for the remainder of recovery ($P = 0.01$). Palatability of HS continued to decrease during rehydration and remained significantly reduced from baseline throughout the period ($P < 0.001$). Ratings of palatability were significantly correlated with P_{Na} during each rehydration condition.

Table 6.3. Relationship (*r*) between palatability and plasma sodium concentration .

		Baseline	0 h	1 h	2 h	3 h	mean
	W	.56	.88[b]	.90[b]	.83[a]	.79[a]	.71[b]
Reh-A	LS	.80[a]	.78[a]	.78[a]	.81[a]	.78[a]	.75[b]
	HS	-.71	-.74[a]	-.76[a]	-.81[a]	-.88[b]	-.80[b]
	W	.37	.73[a]	.81[a]	.70[a]	.89[b]	.75[b]
Reh-B	LS	-.67	-.76[a]	-.74	-.81[a]	-.76[a]	-..75[b]
	HS	-.64	-.89[b]	-.68[a]	-.61	-.70[a]	-.74[b]

Measured before and after exercise and during recovery. N=8 [a] denotes significant correlation ($P < 0.05$), [b] denotes significant correlation ($P < 0.01$)

6.3.5. Ratings of Saltiness

Figure 6.4. illustrates the saltiness ratings for three sampled drinks during both rehydration conditions, before exercise and during rehydration. For analysis of saltiness, a three-way ANOVA revealed a significant main effect for rehydration condition ($F_{(1, 7)}$

= 8.83, $P < 0.0001$), sample drink ($F_{(2, 14)} = 330.87$, $P < 0.001$), time ($F_{(4, 28)} = 23.43$, $P < 0.001$) and the interactions of rehydration condition x sample drink ($F_{(2, 14)} = 8.90$, $P = 0.003$), rehydration condition x time ($F_{(4, 28)} = 45.15$, $P < 0.001$), sample drink x time ($F_{(8, 56)} = 6.51$, $P < 0.001$), and rehydration condition x sample drink x time ($F_{(8, 56)} = 8.92$, $P < 0.001$).

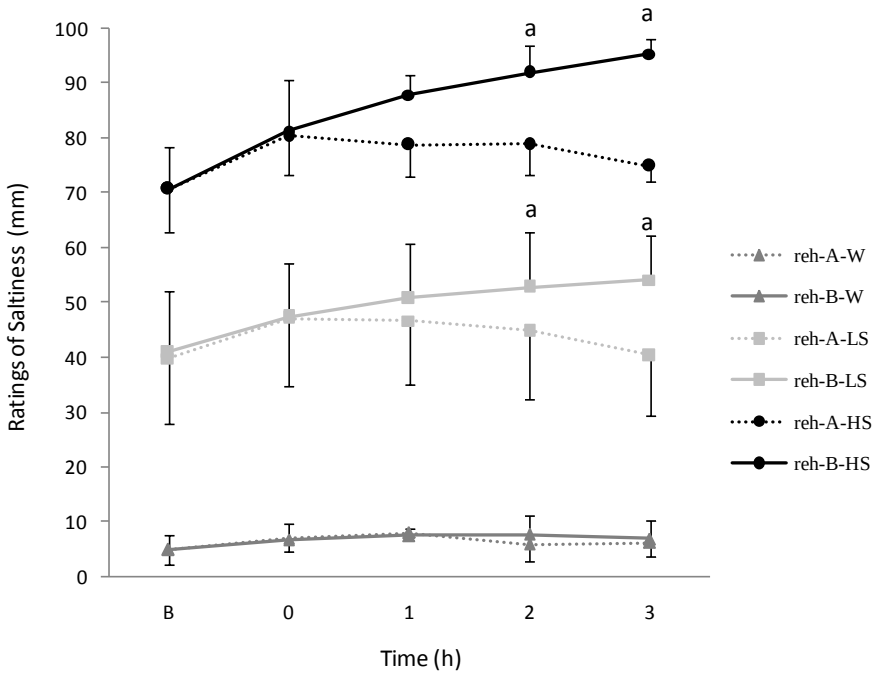

Figure 6.4. Ratings of saltiness. During both rehydration conditions (reh-A vs. reh-B), for three samples of drink (W, LS, HS), at baseline (B) and during rehydration (0-3 h). [a] denotes a significant difference between rehydration conditions (reh-A vs. reh-B) for a given drink sample ($P \leq 0.05$). For clarity of presentation, significant differences from baseline are not illustrated, but described in text.

At baseline ratings of saltiness were equal between reh-A and reh-B with all drink samples (W, LS, and HS; $P > 0.45$). W was rated significantly less salty than either LS or HS ($P < 0.001$) and HS was rated significantly saltier than LS ($P < 0.001$). Except for W, there was a significant increase saltiness rating of both LS and HS following exercise with both reh-A and reh-B ($P < 0.01$). The magnitude of change was not significantly different between reh-A and reh-B ($P = 0.56$). During rehydration,

there was no change in ratings of saltiness for W with either rehydration condition. With reh-A, ratings of saltiness for both LS and HS gradually declined during recovery, and returned to baseline by 3 h ($P > 0.12$). In contrast, with reh-B, ratings of saltiness for both LS and HS continued to increase during recovery and remained elevated from baseline throughout ($P < 0.001$).

6.3.6. Ratings of Strength

For drink strength, a three way ANOVA revealed a significant main effect for sample drink ($F_{(2, 14)} = 319.00$, $P < 0.001$) and time ($F_{(4, 28)} = 2.81$, $P = 0.04$) and the interaction of rehydration condition and sample drink ($F_{(2, 14)} = 22.45$, $P < 0.001$), but not for rehydration condition ($F_{(1, 7)} = 4.99$, $P = 0.06$), or the interactions of rehydration condition x time ($F_{(4, 28)} = 1.66$, $P = 0.19$), sample drink x time ($F_{(8, 56)} = 2.13$, $P = 0.06$) or rehydration condition x sample drink x time ($F_{(8, 56)} = 1.06$, $P = 0.40$). Across all time points and both rehydration conditions, HS (68 ± 12 mm) was rated significantly stronger than both LS (38 ± 8 mm, $P < 0.001$) and both drinks were rated significantly stronger than W (17 ± 4 mm, $P < 0.001$).

Table 6.4. Ratings of drink strength before and during rehydration

		Baseline	0 h	1 h	2 h	3 h	Mean
	W	17 ± 6	17 ± 4	16 ± 4	18 ± 4	18 ± 4	17 ± 4
Reh-A	LS	37 ± 10	40 ± 10	39 ± 9	40 ± 5	37 ± 8	38 ± 8[b]
	HS	62 ± 7	62 ± 10	65 ± 9	67 ± 5	63 ± 7	64 ± 9[c]
	W	18 ± 5	17 ± 4	16 ± 4	16 ± 5	17 ± 4	17 ± 4
Reh-B	LS	36 ± 10	37 ± 11	36 ± 6	34 ± 9	39 ± 6	36 ± 8[b]
	HS	61 ± 7	67 ± 15	74 ± 15	79 ± 14	80 ± 11	72 ± 10[ac]

[a] denotes significant difference between rehydration conditions (reh-A vs. reh-B). [b] denotes significant difference from W, [c] denotes a significant difference from W and LS ($P \leq 0.05$).

6.3.7. Other perceptual ratings

Ratings of sweetness, were significantly influenced by sample drink ($F_{2, 14)}$ = 218.07, $P < 0.001$) but not for rehydration condition ($F_{(1, 7)} = 0.81$, $P = 0.40$), time ($F_{(4, 28)} = 0.71$, $P = 0.56$) or the interactions ($P > 0.54$). Across both rehydration conditions and time, LS (64 ± 9 mm) was rated significantly sweeter than both W (38 ± 8 mm, $P < 0.001$) and HS (42 ± 9 mm, $P < 0.001$). Ratings of sourness, were significantly influenced by sample drink ($F_{2, 14)} = 55.12$, $P < 0.01$) but not for rehydration condition ($F_{(1, 7)} = 1.01$, $P = 0.34$), time ($F_{(4, 28)} = 0.91$, $P = 0.81$) or the interactions ($P > 0.34$). Across both rehydration conditions and time, LS (45 ± 12 mm) was rated significantly sweeter than both W (2 ± 4 mm, $P < 0.001$) and HS (22 ± 14 mm, $P < 0.001$). There were no significant differences in sourness ratings between HS and W ($P > 0.23$). Ratings of bitterness, were not significantly influenced by sample drink ($F_{2, 14)} = 6.02$, $P = 0.21$), the rehydration condition ($F_{(1, 7)} = 0.21$, $P = 0.734$), time ($F_{(4, 28)} = 0.41$, $P = 0.99$) or the interactions ($P > 0.74$).

6.4. Discussion

The present study demonstrated significant changes in ratings of palatability for water and sodium solutions following exercise-induced dehydration and during recovery. Synonymous with antidiuresis and an increase in thirst, ratings of palatability for water substantially increased, and ratings of palatability for a high-sodium drink significantly decreased. Another major finding of this study is the demonstration of an increase in palatability of the high-sodium drink 3 h into recovery with ingestion of 120 % BM losses of a low sodium rehydration solution. These adaptations were strongly correlated with changes in P_{Na} and suggest a physiological regulation of body fluid balance. The changes in palatability appear to be selective to the restoration of hyperosmolality before hypovolemia.

6.4.1. Thirst

The primary stimuli for thirst, following exercise are cellular dehydration and extracellular contraction (Fitzsimons, 1998; Johnson, 2007). Decreased P_{Vol}, detected by baroreceptors throughout the vasculature, is also known to stimulate thirst (Stricker *et al.*, 1992), but only after a loss of 8-10 % (Fitzsimons, 1998; Kimura *et al.*, 1976).

Elevations in P_{Osm} of 1-2 % have previously been associated with increased sensations of thirst (McKinley & Johnson, 2004; Johnson, 2007). The earlier onset of hyperosmotic thirst reflects the greater importance of maintaining P_{Osm} over P_{Vol}. Evidence of this prioritisation in the present study is the thirst responses to changes in P_{Na} during recovery. When reh-A was ingested the gradual fall in P_{Na} was synonymous with a fall in ratings of thirst. When reh-B was ingested the maintenance of elevated P_{Na} was associated with high ratings of thirst throughout recovery. These changes occurred despite negative and positive P_{Vol} restoration with reh-A and reh-B, respectively. Previously, Takamata *et al.* (1994) reported elevated ratings of thirst following exercise, when levels P_{Na} and P_{Osm} were increased, which decreased to baseline levels, when P_{Na} and P_{Osm} normalised.

In response to hyperosmolality the stimulation of thirst occurs with the specific aim to increase fluid consumption and restore P_{Osm} to normality. It is reasonable to assume that the fluid which will alleviate the sensations of thirst to the greatest extent is the one which will restore P_{Osm} most rapidly, i.e. plain water. These results reinforce previous findings that the palatability of water becomes more attractive to humans as bodily fluid levels diminish (Rolls *et al.*, 1980; Takamata *et al.*, 1994). In conjunction with thirst, this study demonstrated an increase in the palatability of W and LS at the start of recovery which decreased simultaneously with P_{Na} and thirst during recovery with reh-A. With reh-B, palatability of W remained superior to baseline throughout recovery. However, there was a progressive decrease in the palatability of LS, which dipped below baseline levels by 2 h. It is likely that as P_{Na} continued to be elevated, even small sodium concentrations would be disadvantageous to the regulation of P_{Osm}, and as such were perceived less pleasant. Under this condition W was deemed more pleasant than LS. Once P_{Na} had returned to baseline levels with reh-A, W was deemed less pleasant than LS, presumably to avoid a hypo-osmotic state.

6.4.2. Sodium Appetite

Whilst the practices governing thirst have been well researched, less is known about the control of sodium intake following exercise. Sodium appetite, an increase in the natural preference for sodium, is a regulative response designed with restoring P_{Vol}. The present study failed to demonstrate an increase in palatability of HS above that of baseline levels, but did find a small but significant increase throughout recovery and a

change in the ratings of saltiness. Our results therefore reinforce previous findings that the taste of salt becomes more attractive to humans as bodily salt levels diminish (Beauchamp *et al*., 1990; Leshem & Rudoy, 1997; Takamata *et al*., 1994), and suggest a physiological regulation of sodium levels in humans.

It is unlikely that the failure to demonstrate changes in palatability of HS at the start of recovery was not a result of insufficient hypovolemia. The activation of sodium conserving hormones can increase salt intake even in the absence of considerable sodium deficit (Johnson & Thunhorst, 1997; Sakai *et al*., 1987). The activation of these hormones is commonplace following exercise of similar intensity and duration (Maughan & Leiper, 1995; Maughan *et al*., 1996). Instead it is proposed that the failure to demonstrate an immediate increase in SA following exercise, despite a P_{Vol} deficit, is a consequence of the need to restore elevated P_{Osm} to baseline levels. This priority is evidenced by the strong relationship between P_{Na} and palatability for HS. Only once P_{Na} had returned to baseline levels 3 h into recovery with reh-A, was there a slight decrease in ratings of saltiness and slight increase in the palatability of HS. When reh-B was ingested, and P_{Na} failed to normalise, ratings of saltiness increased, and ratings of palatability for HS decreased substantially. The changes in ingestive behaviour demonstrated in this study are indicative of the protection of osmoregulation over volume regulation. It suggests the volume-dependent response was overridden by osmoregulatory controls; not until P_{Na} returns to baseline does an increase in sodium palatability occur.

Similar findings have been demonstrated previously when humans were subjected to 7 h intermittent exercise in the heat ($35^{o}C$) with only plain water ingestion (Takamata *et al.*, 1994). Following an exaggerated sodium loss, the authors reported an increase in the palatability of 10 different sodium concentrations starting 3 h after exercise, once P_{Na} and P_{Osm} had returned to baseline. A delayed onset of SA has also been reported in rats following sodium deprivation (Yawata *et al.*, 1988; Sugiomoto, 1988) and in humans following dialysis (Lesham & Roduy, 1997). In contrast, Lesham *et al.* (1999) demonstrated SA within 30 minutes of terminating exercise. Compared with baseline, participants increased their *ad libitum* addition of salt to soup, but not sugar to tea following 1 h of exercise in a thermo-neutral environment. Unfortunately no measures of fluid balance were taken, nor was the intensity of exercise recorded, thus the level of sodium and/or fluid deficits are unknown and difficult to predict. The authors' brief description of exercise does support their claim that the sodium loss was

'mild' and it is reasonable to assume inferior to that evoked in the present study and that by Takamata *et al.* (1994). It is possible that since sodium and fluid losses were mild, the elevated P_{Osm} which normally suppresses the expression of a SA had returned to baseline levels prior to the palatability test. It is also possible, however that the measure of assessing SA was unreliable.

6.4.3. Limitations

Previous investigation has employed isolated solutions of NaCl in water (e.g. Takamata *et al.*, 1994). From a practical perspective humans would not readily choose to ingest such solutions, and therefore in order to increase the ecological validity of this study, adaptations of commercially available products were employed. It is noted that increasing the number of tastants with a solution raises the possibility that changes in palatability were a function of other variables others than saltiness. However, ratings of saltiness were negatively correlated with ratings of palatability for HS and LS, and there were no significant changes in ratings of sweetness, sourness or bitterness throughout recovery. It is not known the extent to which osmolality as opposed to saltiness affected palatability. Several studies have previously demonstrated an increase in preference for low osmolality fluids following exercise (Appleton, 2005; King *et al.*, 1999; Rolls *et al.*, 1981; Takamata *et al.*, 1994). The strong relationship between ratings of saltiness and palatability in the present study suggest sodium content to be a significant determinant.

A limitation of the study is that due to the nature of the protocol only subjective measures of taste palatability were assessed. From a practical viewpoint, behavioural measures are also valid (Wilmore *et al.*, 1998; Szlyk *et al.*, 1989). It is not known whether an increase in the palatability of a high sodium drink will affect consumption of sodium following exercise. However, subjective and behavioural measures of palatability are intimately related (Yeomans *et al.*, 2000).

6.5. Conclusions

The present study demonstrated significant changes in ratings of palatability for water and sodium solutions following exercise-induced dehydration and during recovery. Synonymous with antidiuresis and an increase in thirst, ratings of palatability for water substantially increased, and ratings of palatability for a high-sodium drink

significantly decreased. These changes were strongly related to the level of fluid balance i.e. P_{Na} and the automated responses to this disturbance, U_{Osm} and thirst. Herein, the changes in taste perception appear to act as a physiological regulator, and moreover, this regulation prioritises restoration of hyperosmolality over hypovolemia. Our findings support previous investigation in which an increase in palatability for sodium was delayed until P_{Na} and P_{Osm} normalised.

Chapter 7 – Study 4

The physiological effects of ingesting a high vs. low sodium drink during exercise in the heat

7.1. Introduction

When exercise is performed at a high intensity in the heat, sweat losses are elevated to augment evaporative heat loss. If fluid losses are not adequately replaced with the consumption of fluid during exercise, dehydration develops. Sweat loss induced dehydratyion is characterised by an increase in P_{Osm} and a decrease in $P_{Vol,}$ which is known as hyperosmotic hypervolemia. Both hyperosmoality and hypovolemia impact on cardiovascular and thermoregulatory functioning (Montain & Coyle, 1992a; Nose *et al.,* 1990), to the detriment of exercise performance.

The ingestion of low sodium (<25 $mmol \cdot L^{-1}$) carbohydrate-electrolyte sports drinks have resulted in small reductions in P_{Osm} (Barr *et al.,* 1991; McConnell *et al.,* 1997), but have not consistently restored P_{Vol}, when compared with ingestion of plain water, or no ingestion (Barr *et al.,* 1991; Cade *et al.,* 1972; Greenleaf & Brock, 1980; McConnell *et al.,* 1997; Powers *et al.,* 1990). In contrast, the infusion of high sodium concentrations during exercise has consistently been shown to reduce the decline in P_{Vol} during exercise in the heat and at a thermo-neutral temperature, as well as increase sweat rates, and reduce T_{Core} and HR_{Drift} (Deschamps *et al.,* 1989; Fortney *et al.,* 1988; Montain & Coyle, 1992b; Nose *et al.,* 1990). However this method is impractical in an occupational environment and prohibited in athletic events. Ingesting high sodium concentrations represent a more practical intervention yet only once has the effect of this strategy been investigated *during* exercise (Sanders *et al.,* 2001).

Sanders *et al.* (2001) found that the replacement of 100 % of fluid losses during 4 h exercise, of a high sodium solution (100 $mmol \cdot L^{-1}$) attenuated the decline in P_{Vol} compared with low sodium (5 $mmol \cdot L^{-1}$) and moderate sodium (50 $mmol \cdot L^{-1}$) solutions. Despite this, neither HR nor T_{Core} were statistically different between conditions. The partcipants in this experiment were trained cyclists and exercise in a thermo-neutral environment ($20^{o}C$). Previous investigation tells us that more pronounced changes in cardiovascular variables occur in untrained, compared with trained participants

following pre-exercise P_{Vol} expansion (Coyle *et al.,* 1986; Hopper *et al.,* 1988) and that P_{Vol} expansion has a more consistent effect, than when exercise is performed in the heat. Whilst a decrease in HR is a common occurrence following P_{Vol} expansion in the heat (Fortney *et al.,* 1981a; Fortney *et al.,* 1983; Montain & Coyle, 1992b; Nose *et al.,* 1990; Sawka *et al.,* 1983), when exercise is performed in a thermo-neutral environment others have failed to report an ergogenic effect (Berger *et al.,* 2006; Hopper *et al.,* 1988; Watt *et al.,* 2000) and Kanstrup *et al.* (1992) actually reported a 12 $b \cdot min^{-1}$ increase in HRIndeed Nose *et al.* (1990) directly compared the influence of environemnetal temperature on the effects of P_{Vol} expansion. Nose *et al.* (1990) demonstrated a 5 % decrease in HR_{Drift} whilst untrained participants exercised for 50 min at 60 % $\dot{V}O_{2max}$ at $30^{o}C$, but no change in HR_{Drift} when participants completed the same exercise protocol at 22^{o}CAs yet, it is not known what effect P_{vol} expansion with a high sodium concentration has during exercise in the heat in untrained partcipants.

7.1.1. Aims and Hypotheses

The purpose of this investigation was to examine the cardiovascular and thermoregulatory effects of ingesting a low (15 $mmol \cdot L^{-1}$) and high (151 $mmol \cdot L^{-1}$) sodium solution during 90 min moderate intensity exercise in untrained participants exercising in the heat ($33^{o}C$). It was hypothesised that ingestion of a high sodium drink would significantly attenuate the decline in P_{Vol}, reduce CV_{Drift} and the rate of rise in T_{Core} compared to a low sodium drink.

7.2. Materials and Methods

Eight untrained males (age: 24 ± 4 yrs; mass: (71.6 ± 2.0 kg; $\dot{V}O_{2max}$: 53 ± 6 $L \cdot kg^{-1} \cdot min^{-1}$) completed this double-blind, randomly controlled repeated measures design. Participants initially completed an incremental maximal cycle exercise test ($\dot{V}O_{2max}$) for determination of exercise intensity (see 3.1. General Methods). On the basis of the $\dot{V}O_{2}$-work rate relationship, the power output equivalent to 60 % $\dot{V}O_{2max}$ was calculated and assigned to subsequent tests. Each participant performed two experimental trials separated by > 3 d. The first trial was randomly allocated to the first

participant and then alternated thereafter. Equal hydration was confirmed by similar pre-trial BM, U_{Osm} and P_{Na}.

7.2.1. Experimental Trials

Participants completed the same pre-exercise routine as with previous chapters. For more details see section 4.3.1. In addition, Signal morphology-based impedance cardiography (PhysioFlow, Neumedx, Bristol) was used to record HR, SV and $\dot{Q}$. Six electrodes (Physio Flow, Manatec) were attached to each participant at rib, chest, neck, back. These sites were sterilized and shaved for optimal conductance. Duplicate measurements of resting blood pressure (Pro 100V2 dinamap) were taken prior to exercising whilst seated on a cycle ergometer. $\dot{V}O_2$ was measured using an on-line gas analyser (Cortex Metalyzer 2R, Cortex, Leipzig, Germany) for ten-minute intervals between the time periods of 10-20 minutes and 30-40 minutes of each bout.

Participants entered the environmental chamber that was set at 33 ± 0.2°C, 50 ± 0.6 % relative humidity, $V_{\alpha} \approx 2.5$ m·s^{-1}, mounted the cycle ergometer (Lode Excalibur Sport V2, Lode BV, Groningen, Netherlands) and remained seated for 15 min to ensure steady state P_{Vol} and constituents. A capillary blood sample was taken for determination Hct, Hb and P_{Na}. Participants then completed 5 min cycling at 50 W, followed by 2 x 45 min (separated by a ten minute rest interval) of cycling a power output of 201 ± 26 W, which initially elicited 55 % $\dot{V}O_{2max}$.

Table 7.1. Drink Properties

	Sodium Chloride (g·L^{-1})	Sodium Citrate (g·L^{-1})	Sodium Content (mmol·L^{-1})	Carbohydrate (g·L^{-1})	Osmolality (mOsm·kg^{-1})
LS	-	-	15	20	130
HS	4.5	7.72	151	20	435

LS = Low sodium, HS = High sodium.

During each bout, the participants drank a volume of fluid equivalent to 12 $mL \cdot kg^{-1}$ BM of one of two fluids; either a low sodium-carbohydrate-electrolyte drink, or a high sodium-carbohydrate-electrolyte drink. Drink properties are listed in table 7.1. The volume of fluid was chosen to equal 100 % of BM losses, determined from pilot testing. Drinks were served in concealed containers at 22 ± 1°C at the start of each bout. Following each bout, a capillary and venous blood sample was taken whilst the participant remained seated on the cycle ergometer, then participants urinated, towelled themselves dry and weighed themselves in the nude. U_{Vol} voided before and during exercise was recorded and analyzed for U_{Osm}.

7.2.2. Blood and urine analysis

Blood and urine were analysed using the same methods as outlined in section 4.2.4.

7.2.3. Calculations

HR_{Drift} was calculated by subtracting the mean HR for the 45th minute from the mean HR for the 10th minute. The same calculation was performed for SV_{Drift}. For example:

$$SV_{Drift} = SV_{45} - SV_{10}$$

$\dot{V}O_2$ was calculated by averaging the values for the previous 5 min period prior to each time point.

7.3. Results

7.3.1. Hydration Status

There were no differences in pre-exercise BM (71.6 ± 2.0 kg, $P = 0.90$), U_{Osm} (157 ± 86 $mOsm \cdot kg^{-1}$, $P = 0.46$) or P_{Na} (136 ± 4, $P = 0.832$) between trials. Table 7.2. illustrates the changes in fluid balance during the protocol. A two way ANOVA revealed a significant main effect for drink ($F_{(1, 7)} = 5.45$, $P = 0.05$) for sweat loss responses during both bouts. Across both time points, sweat loss was greater with LS

than HS (2.00 ± 0.17 vs. 1.69 ± 0.25 L). There was no main effect for time ($F_{(2, 14)}$ = 3.05, P = 0.12) or the interaction ($F_{(2, 14)}$ = 1.51, P = 0.26). U_{Vol} voided following bout 1, was significantly greater with LS, compared to HS ($F_{(1, 14)}$ = 4.49, P =0.05). Due to the greater sweat and U_{Vo} with LS, compared with HS, ingestion of fluid replaced a significantly smaller proportion of BM losses with LS compared to HS (72 ± 10 vs. 83 ± 9 %, P = 0.01).

Table 7.2. Fluid balance during exercise.

	LS	HS
Baseline BM (kg)	71.6 ± 2.1	71.6 ± 1.9
Sweat Loss (L)	1.96 ± 0.24	1.81 ± 0.22[a]
Fluid Intake (L)	1.72 ± 0.05	1.72 ± 0.05
U_{Vol} (L)	0.45 ± 0.16	0.32 ± 0.10 [a]
End BM (kg)	70.9 ± 2.2	71.6 ± 2.0 [a]
Fluid Replacement %	72 ± 10 %	83 ± 9 % [a]
Net BM Loss (%)	1.0 ± 0.5	0.5 ± 0.4 [a]

LS = Low sodium, HS = high sodium. [a] denotes a significant difference between treatments ($P \leq 0.05$).

A two factor ANOVA (drink x time) revealed a significant main effect for drink ($F_{(1, 7)}$ =9.14, P = 0.02), time ($F_{(2, 14)}$ = 77.05, P < 0.001) and the interaction ($F_{(2, 14)}$ = 4.75, P = 0.03) in the U_{Osm} response to exercise. U_{Osm} was significantly greater at the end of both bouts, compared with LS (P < 0.02). A two factor ANOVA also revealed a significant main effect for drink ($F_{(1, 7)}$ = 4.87, P = 0.05), time ($F_{(2, 14)}$ = 223.60, P < 0.001) and the interaction ($F_{(2, 14)}$ 73.26, P < 0.001) in the P_{Na} response to exercise. P_{Na} was significantly greater at the end of both bouts with HS, compared to LS (P < 0.001). P_{Na} increased significantly during bout 1 with both drinks, and continued to increase significantly during bout 2, with HS only (P < 0.001). LS attenuated the increase in P_{Na} during bout 2 (P = 0.10). Whilst LS attenuated the increase in P_{Na} from baseline (P = 0.70), ingestion of HS significantly increased P_{Na} during bout 1 (P = 0.01). During the

second bout, when no fluid was ingested, P_{Na} increased to a similar extent with both HS and LS (P = 0.26).

Table 7.3. Plasma Sodium Concentration and Urine Osmolality responses to exercise

	LS	HS
P_{Na} ($mmol \cdot L^{-1}$)		
Baseline	136 ± 3	136 ± 4
Post Bout 1	139 ± 5	144 ± 5[ab]
Post Bout 2	144 ± 5[c]	148 ± 5[c]
U_{Osm} ($mOsm \cdot kg^{-1}$)		
Baseline	147 ± 85	139 ± 79
Post Bout 1	455 ± 164[b]	823 ± 125[ab]
Post Bout 2	553 ± 163[b]	915 ± 155[ac]

P_{Na} = Plasma sodium concentration, U_{Osm} = Urine osmolality. [a] denotes a significant difference between drinks; [b] denotes a significant difference from baseline; [c] denotes a significant difference from baseline and 'post bout 1'.

7.3.2. Plasma Volume

Changes in P_{Vol} during rest and exercise are illustrated in Figure 7.1. Ingestion of both fluids during bout 1 maintained P_{Vol} at near baseline level. Analysis of changes in P_{Vol} during exercise using a two factor ANOVA revealed a significant main effect for drink ($F_{1, 7)}$ = 14.96, P = 0.006), time ($F_{(2, 14)}$ = 81.43, P < 0.001), and the interaction ($F_{(2, 14)}$ = 20.79, P < 0.001). Following bout 1, there were no significant differences in P_{Vol} between treatments (-5.1 ± 1.9 %, P = 0.14). Following bout 2, there was no significant change in P_{Vol} with LS (-6.3 ± 2.1 %, P = 0.56), but P_{Vol} increased significantly with HS to -1.9 ± 1.0 % (P = 0.02). Changes in P_{Vol} were positively correlated with changes in ΔP_{Na}-B1(r = 0.72, P = 0.01) and ΔP_{Na}-B2 (r = 0.87, P = 0.01).

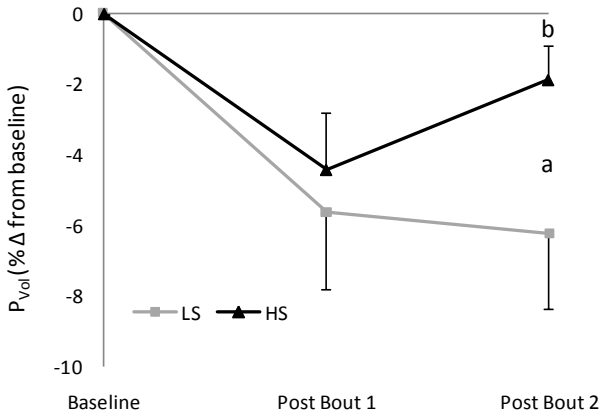

Figure 7.1. Changes in plasma volume during rest and exercise. P_{Vol} as a percentage from baseline was measured, at the end of bout 1, and bout 2, for both treatments (HS = High Sodium, LS = Low Sodium). [a] denotes significant difference between drinks, [b] denotes significant difference from end of bout 1 ($P < 0.05$).

7.3.3. Cardiovascular Responses

Resting HR (65 ± 4 $b \cdot min^{-1}$), SV (71 ± 4 $mL \cdot b^{-1}$), and $\dot{Q}$ (5.0 ± 0.4 $L \cdot min^{-1}$) were similar in all trials ($P > 0.05$). Two factor ANOVA's revealed no significant effects for drink for changes in HR, SV or $\dot{Q}$ throughout bout 1. During bout 2, a two factor ANOVA revealed a main effect for drink ($F_{(1, 7)} = 7.47$, $P = 0.03$), time ($F_{(2, 14)} = 162.00$, $P < 0.001$) and the interaction ($F_{2, 14}$) = 9.80, $P = 0.02$), for HR responses to exercise. There was no significant difference in HR between drinks at 55 min ($P = 0.35$). With both drinks, HR increased significantly during exercise ($P < 0.001$), although HR was significantly reduced with HS, compared to LS at 90 min ($P = 0.02$).

A two factor ANOVA revealed a main effect for drink ($F_{(1, 7)} = 22.06$, $P = 0.002$), time ($F_{(2, 14)} = 67.41$, $P < 0.001$) and the interaction ($F_{2, 14}$) = 27.27, $P = 0.01$), for SV responses to exercise. SV progressively decreased with both fluid from 55-90 min ($P < 0.001$), although the decline in SV was significantly attenuated with HS, such that at 90 min, SV with LS was significantly greater than with HS ($P = 0.001$). A two factor ANOVA revealed a significant main effect for time ($F_{(2, 14)} = 13.95$, $P = 0.007$),

but not for drink ($F_{(1, 7)} = 0.14$, $P = 0.72$) or the interaction ($F_{2, 14)} = 0.35$, $P = 0.57$) for $\dot{Q}$ responses during exercise.

Table 7.4. Cardiovascular, perceptual and thermoregulatory responses to exercise

	Bout 1			Bout 2		
	LS		HS	LS		HS
HR ($b \cdot min^{-1}$)						
10 min	131 ± 7		130 ± 8	136 ± 7		135 ± 7
45 min	140 ± 8	c	138± 6	150 ± 7[b]		145 ± 8[ab]
SV ($ml \cdot b^{-1}$)						
10 min	137 ± 5		138 ± 5	133 ± 6		134 ± 7
45 min	124 ± 5	c	128 ± 5	117 ± 7[b]		124 ± 6[ab]
$\dot{Q}$ ($L \cdot min^{-1}$)						
10 min	18.0 ± 1.2		18.2 ± 1.0	18.1 ± 0.9		18.3 ± 1.1
45 min	17.6 ± 1.1	c	17.8 ± 0.9	17.3 ± 1.3	c	17.7 ± 1.1
$\dot{V}O_2$ ($L \cdot min^{-1}$)						
10 min	2.44 ± 0.4		2.43 ± 0.4	2.58 ± 0.5		2.52 ± 0.4
45 min	2.63 ± 0.4	c	2.66 ± 0.3	2.54 ± 0.3	c	2.64 ± 0.3
RPE						
10 min	12.0 ± 0.8		12.1 ± 0.6	12.0 ± 0.8		12.0 ± 0.8
45 min	13.5 ± 0.5	c	13.1 ± 0.6	14.8 ± 0.5[b]		14.3 ± 0.6[ab]
T_{Rec} (^oC)						
10 min	37.1 ± 0.2		37.1 ± 0.3	37.5 ± 0.4		37.6 ± 0.3
45 min	38.0 ± 0.3	c	38.1 ± 0.2	38.5 ± 0.3	c	38.7 ± 0.2

Values are in mean ± SD. HR= heart rate; SV = stroke volume; $\dot{Q}$ = cardiac output; $\dot{V}O_2$ = oxygen uptake; RPE = ratings of perceived exertion. [a] denotes significant difference between drinks at the same time point; [b] denotes a significant change between 10 and 45 min; [C] denotes a significant main effect for time ($P \leq 0.05$).

7.3.4. Ratings of Perceived Exertion

During bout 1, there a significant main effect for time ($F_{(2, 14)} = 82.89$, $P < 0.001$). With both drinks RPE increased during exercise. There was no main effect for drink ($F_{(1, 7)} = 0.37$, $P = 0.56$) or the interaction ($F_{2, 14)} = 0.00$, $P = 1.00$). During bout 2,

a two factor ANOVA revealed a main effect for drink ($F_{1, 7)} = 7.00$ $P < 0.03$), time ($F_{(2, 14)} = 87.50$, $P < 0.001$) and the interaction ($F_{(2, 14)} = 7.00$, P = 0.03) for the RPE. At 55 min, RPE was identical between drinks ($P = 1.0$). RPE increased during exercise with both drinks ($P < 0.001$), although to a greater extent with LS ($P = 0.03$).

7.3.5. Oxygen Uptake

During both bouts, a two factor ANOVA revealed a main effect for time ($P < 0.01$) for the $\dot{V}O_2$ response. For both drinks, $\dot{V}O_2$ increased significantly between 10-45 min and between 55-90 min. There was no main effect for drink or the interaction ($P > 0.05$).

7.3.6. Thermoregulatory Responses

During bout 1, a two factor ANOVA revealed a significant main effect for time ($F_{(1, 7)} = 131.43$, $P < 0.001$) and the interaction ($F_{(1, 7)} = 7.41$, $P = 0.03$) for the T_{Rec} responses to exercise. As expected there was a significant increase in T_{Rec} during exercise. However, there was no main effect for drink ($F_{(1, 7)} = 2.48$, $P = 0.16$). During bout 2, a two factor ANOVA revealed a significant main effect for time ($F_{(2, 14)} = 338.01$, $P < 0.001$), but no main effect for drink ($F_{(1, 7)} = 0.98$, $P = 0.55$) or the interaction ($F_{(2, 14)} = 3.98$, $P = 0.32$). T_{Rec} increased significantly with both drinks ($P < 0.001$) during exercise.

7.4. Discussion

The purpose of this study was to determine the effect of ingesting a high sodium sports drink on cardiovascular variables during exercise in the heat. The novel aspect of this study was that exercise was performed in the heat and with an untrained population. The main findings of this study are that the ingestion of a high sodium concentration (151 $mmol \cdot L^{-1}$ Na) attenuated the decline in P_{Vol} by 5.4 %, and conserved fluid losses by 340 ± 260 mL, compared with the ingestion of the same volume of a low sodium sports drink (15 $mmol \cdot L^{-1}$ Na). This resulted in an attenuation of HR_{Drift} by 4 ± 1 $b \cdot min^{-1}$, SV_{Drift} by 5 ± 4 $mL \cdot b^{-1}$ and ΔRPE, during the second bout of exercise . This was the first study to show that the pre-exercise ingestion of a high sodium concentration is effective in attenuating the fall in SV, normally evident during intense exercise in the heat.

Sodium concentration had no effect on T_{Rec}, $\dot{Q}$ or, $\dot{V}O_2$; or on HR or SV during the first 45 min of exercise.

7.4.1. Fluid Balance

By reducing the fall in P_{Na} during exercise, and maintaining fluid shifts from intracellular to extracellular fluid compartments and the antidiuretic response to exercise, the present study demonstrated greater restoration of P_{Vol} with the high sodium compared with the low sodium drink. Adding sodium to a drink has previously been shown to improve fluid retention, before (Sims *et al.,* 2007a; 2007b), during (Barr *et al.,* 1991; Candas *et al.,* 1986; Carter & Gisolfi, 1989; Sanders *et al.,* 2001) and after exercise (Costill & Sparkes, 1973; Maughan & Leiper, 1995; Shirreffs *et al.,* 1996). With HS, the large difference in intake and estimated losses in sodium drove increases in P_{Na}, relative to the low sodium drink at 45 min and at 90 min. An elevated P_{Na}, or rather P_{Osm} maintains antidiuretic hormone release and reduces diuresis. This study found a major effect of sodium concentration and water and electrolyte excretion. High sodium intake reduced U_{Vol} from 452 to 317 mL and increased U_{Osm} from 455 to 823 $mOsm \cdot kg^{-1}$. Plasma hyperosmolality also promotes fluid retention in the form of reduced sweat rates (Harrison *et al.,* 1978; Nielsen *et al.,* 1971; Senay, 1979; Fortney *et al.,* 1984; Nielsen *et al.,* 1974; Takamata *et al.* 1998; 2001). The greater P_{Vol} in the HS trial, in the present study, was also a result of a reduced sweat loss with HS conserving 171 ± 94 mL. The presence of sodium ions in a drink favours the distribution of conserved fluid within the ECF compartments, over other fluid compartments (Costill & Sparks, 1973; Nielsen *et al.,* 1986; Nose *et al.,* 1988c). The elevated P_{Na} with HS, attenuated decline in P_{Vol}, by driving fluid shifts from intracellular to extracellular space.

7.4.2. Cardiovascular Responses

When a high vs. low-sodium sports drink was ingested during the first 45 min of a 90 min exercise period, the present study demonstrated a 4 $b \cdot min^{-1}$ attenuation of HR_{Drift}, a 6 $mL \cdot b^{-1}$ attenuation of SV_{Drift} but no significant difference in $\dot{Q}_{Drift}$ during the second 45 min of bout. No differences in cardiovascular variables were demonstrated

during the first 45 min of exercise. This was not unduly surprising since no notable differences in P_{Vol} and TBW between drink conditions were demonstrated.

The increases in SV with P_{Vol} expansion would presumably result from an increase in venous filling (Hopper *et al.*, 1988; Horwitz & Lindenfield, 1985; Krip *et al.*, 1997; Mier *et al.*, 1996; Robinson *et al.*, 1966; Warburton *et al.*, 1999), resulting in a greater EDV and increased SV. In support of this, several studies have demonstrated improvements in cardiovascular function during exercise when P_{Vol} was maintained at baseline levels with intravenous infusion of a P_{Vol} expander during exercise (Deschamps *et al.*, 1989; Fortney *et al.*, 1988; Montain & Coyle, 1992b; Nose *et al.*, 1990). Whilst the majority of these studies have found the infusion of a P_{Vol} expander during exercise to be highly effective, the use of intravenous infusions is neither practical nor feasible in both an athletic and occupational scenario, thus alternative P_{Vol} expansion strategies must be found.

Past research has shown that despite greater P_{Vol}, the ingestion of low sodium drinks does not consistently improve either cardiovascular functioning or thermoregulation during exercise (Barr *et al.*, 1991; Candas *et al.*, 1986; Carter & Gisolfi, 1989; Costill & Sparks, 1973). Complete restoration of P_{Vol} may require the ingestion of a greater electrolyte concentration. Sanders *et al.* (2001) investigated the effects of ingesting 100 % of BM loss of three different sodium concentrations (5, 50, 100 $mmol \cdot L^{-1}$ Na) during 4 h intermittent moderate intensity exercise. Ingestion of the low sodium drink decreased P_{Vol} by 6 % of a 15 min exercise value. Ingestion of the moderate sodium drink maintained P_{Vol} at this 15 min value throughout exercise, and ingestion of the high sodium drink, increased P_{Vol} by 5 %. Despite the differences in P_{Vol} between low and high sodium drinks, larger than that induced in the present study, no significant differences in T_{Rec} or HR were demonstrated between conditions. No measurements of SV, $\dot{Q}$, $\dot{V}O_2$ or RPE were made.

The disparate findings between the present study and that of Sanders *et al.* (2001) can be explained by the methodological differences; training status and environmental conditions. Previous studies promoting P_{Vol} expansion during exercise with infusions of saline, albumin or dextran, have also found differences in findings dependent on training status. For example, Coyle *et al.* (1986) and Hopper *et al.* (1988) both failed to demonstrate an increase in SV following 12-15 % increase in P_{Vol} in highly trained athletes. More consistent improvements in cardiovascular functioning

have been demonstrated in untrained participants (e.g. Coyle *et al.*, 1986; Deschamps *et al.*, 1989; Fortney *et al.*, 1981a; 1981b; 1983). Untrained men may have a greater capacity to dilate the left ventricle and increase SV in response to conditions which increase venous filling, i.e. P_{Vol} expansion. The endurance trained cyclists employed in the study of Sanders *et al.* (2001) may have been functioning at near maximum diastolic capacity, which would prevent significant increases in SV, with P_{Vol} expansion. It is possible that trained athletes, who already possess an elevated P_{Vol} in respect of their untrained counterparts, may be limited in their capacity to increase further. Indeed, Hopper *et al.* (1988) and Grant *et al.* (1997) both demonstrated that P_{Vol} expansion of 21 % added no further cardiovascular advantage than P_{Vol} expansion of 12-14 %.

Secondly the environmental conditions in which exercise took place may have had a significant effect on cardiovascular variables, since environmental temperature is known to have a significant influence on CV_{Drift} (e.g. Gonzalez-Alonso *et al.*, 2000; Wingo *et al.*, 2005). Nose *et al.* (1990) found that when particpants exercise at 60 % $\dot{V}O_{2max}$ at 22^{o}C, intravenous infusion of 340 mL of isotonic saline during 20-50 min of exercise had no effect on HR_{Drift}. However, when the same exercise took place at 30^{o}C the same level of P_{Vol} expansion resulted in a 5 b·min^{-1} drop in HR_{Drift}. The warm conditions employed in the present study are characterised by a greater rate of sweat loss, and increased cardiovascular and thermoregulatory strain, and therefore create a situation more likely to facilitate differences in these variables that those used during the study of Sanders *et al.* (2001).

The decrements in cardiovascular functioning as a result of exercise-induced dehydration can have a significant impact on exercise performance. Wingo and colleagues demonstrated a causal link between the effect of CV_{Drift} on $\dot{V}O_{2max}$ by measuring $\dot{V}O_{2max}$ immediately following a bout of submaximal exercise lasting 45 min in which CV_{Drifft} was induced (Ganio *et al.*, 2006; Wingo & Cureton, 2006a; Wingo & Cureton 2006b; Wingo *et al.*, 2005). These studies manipulated the degree of CV_{Drift} during prolonged exercise by lowering ambient temperature (Wingo *et al.*, 2005a), cooling with air flow (Wingo & Cureton 2006b) and reducing the exercise intensity (Wingo & Cureton 2006a). A relationship between CV_{Drift} and $\dot{V}O_{2max}$ was also demonstrated by Ganio *et al.* (2006) when CV_{Drift} was manipulated in a similar way to the present study by altering the level of dehydration with fluid ingestion. Ganio *et al.* (2006) found that in the control experiment, 2 h of cycling in a warm environment

without fluid ingestion resulted in a decrease in SV of 14 % and a decline in $\dot{V}O_{2max}$ of 9 %. When dehydration was prevented with fluid ingestion, the decline in SV and $\dot{V}O_{2max}$ were both eliminated.

Despite a reduction in SV_{Drift} with high sodium intake, no significant differences in $\dot{V}O_2$ between conditions were demonstrated in the presents study. This is not sureprising given that no notable differences in $\dot{Q}$ were found between conditions. Whether $\dot{Q}$ is affected during exercise depends on the extent to which the decrease in SV, as a result of fluid loss, can be counterbalanced by an increase in HR. From previous investigations it is known that this is dependent upon the environmental conditions, participant characteristics and the intensity of exercise (Gonzalez-Alonso *et al.,* 2008; Nybo *et al.,* 2008). Normally, when exercise is performed in the heat, the reduction in SV which occurs as a result of the combination of both the peripheral displacement of blood volume and the loss of TBW with increased sweat rates will outweigh the capacity for increased HR to compensate for the loss of $\dot{Q}$. This study was undertaken in a warm environment, at a temperature which has previously been shown to compromise cardiovascular and thermoegulatory capacity and limit exercise performance (Wingo Cureton, 2006a). Yet, the significant replacement of fluid losses during the first bout of exercise may have reduced TBW losses sufficiently as to limit SV_{Drift}. Therefore, the increase in HR was sufficient to compensate for a loss of SV during exercise, which explains why $\dot{Q}$ was not significantly compromised during exercise. Given that $\dot{Q}$ was not dissimilar between conditions; one would have expected a similar $\dot{V}O_2$.

7.4.3. Thermoregulation

Despite a reduction in sweat rates with HS, compared to LS, there were no noticeable differences in T_{Rec} during exercise. Previous studies promoting P_{Vol} either before or during exercise have produced equivocal findings regarding thermoregulation. Some studies have reported a reduction in T_{Rec} (Deschamps *et al.,* 1989; Fortney *et al.,* 1981b; 1988b; Nose *et al.,* 1990; Sanders *et al.,* 2001; Sims *et al.,* 2007a; 2007b), whilst others have reported no effect (Fortney *et al.,* 1981a; Grant *et al.,* 1997; Leutkemeier & Thomas 1994; Montain & Coyle, 1992b; Sanders *et al.,* 2001; Sawka *et al.,* 1983a; Watt

et al., 2000). The similar mean T_{Rec} between conditions in the present study should not necessarily be seen as a 'failure' of HS drinks, since the promotion of plasma hyperosmolality which accompanies the ingestion of high sodium drinks promotes the opposite respone; an increase in T_{Core} (Harrison *et al.*, 1978; Nielsen *et al.*, 1971; Senay, 1968; Senay, 1979; Fortney *et al.*, 1984; Nielsen *et al.*, 1973; 1974; Takamata *et al.* 1997; 2001). Thus it is possible that plasma hyperosmolality countered any possible positive thermoregulatory advantage of P_{Vol} expansion, or *vice versa.*

The similar T_{Rec} responses between conditions are important when considering the mechanisms responsible for the change in HR and SV with high sodium ingestion. It is acknowledged that using T_{Rec} as the only measure of body temperature is not ideal to reflect changes in temperature during exercise. However, ignoring the study's minor shortcomings these findings suggest that differences in CV_{Drift} in this study at least appear independent of body temperature. Much debate in the literature has focussed on the cause of CV_{Drift} during exercise in the heat (Coyle & Gonzalez-Alonso, 2001). The traditional hypothesis is that CV_{Drift} is caused by peripheral displacement of blood volume to augment evaporative heat loss, which results in the progressive fall in CBV, SV, and arterial pressure (Ekelund & Homgren, 1964; Johnson & Rowell, 1975; Rowell, 1986). Whilst this made good theoretical sence, others have found that cardiovascular functioning is not compromised by an increase in BF_{skn}, since CV_{Drift} can occur independently of reductions in CBV (Montain & Coyle, 1992b; Saltin & Stenberg, 1964). An alternatiove hypothesis is that an elevated blood temperature detected by the sinoatrial node (Gorman & Proppe, 1984; Johnson & Proppe, 1996) increases sympathetic nervous system activity, raising HR and thus decreasing ventricular filling time, end-diastolic volume, and SV (Coyle & Gonzalez-Alonso, 2001). This notion is supported by the findings of Fritzsche *et al.* (1999) which reported a strong correlation (r^2 = 0.95) between increases in T_{Core} and HR during CV_{Drift}. The findings of this study provide no support for the latter argument. This study found no relationship between T_{Rec} and SV_{Drift}, HR_{Drift} or $\dot{Q}_{Drift}$. Instead the changes in cardiovascular function that occurred following ingestion of the high sodium drink were a result of the attenuated decline in P_{Vol} with HS, compared to LS.

7.5. Conclusions

The consumption of a highly concentrated sodium sports drink increased total body water and attenuated the decline in P_{Vol} and resulted in a decrease in HR_{Drift} and SV_{Drift} during the second of two 45 minute bouts of exercise in the heat. High sodium ingestion not affect T_{Rec}, $\dot{Q}$, or $\dot{V}O_2$. This was the first study to investigate the effects of high sodium concentrations during exercise in the heat in untrained participants. The result raises the possibility that high sodium drinks could be used as a performance enhancing aid during high intensity exercise in the heat. However, more research is required to identify the optimal conditions for which high sodium drinks and the subsequent P_{Vol} expansion may affect performance. Although $\dot{V}O_2$ was measured, it does not provide an accurate measure of performance, which is necessary to properly assess the efficacy of high sodium drinks as an ergogenic aid when used during exercise.

Chapter 8 – Study 5

Pre-exercise ingestion of a high sodium and cold drink increases exercise capacity in the heat

8.1. Introduction

During competitions in a hot environment athletes can experience significant cardiovascular and thermoregulatory stress, that impacts on their ability to perform effectively (Gonzalez-Alonso *et al.,* 2008; Nybo, 2008). Strategies to improve athletic performance in the heat are therefore widespread and have tended to involve the ingestion of fluids during exercise (e.g. Ganio *et al.,* 2006; Montain & Coyle, 1992a). Unfortunately not all competitions or workplace environments permit easy access to fluids during an event; in which case pre-exercise strategies represent more practical and effective methods to improve performance.

8.1.1. Pre-cooling

Pre-cooling represents an intervention whereby pre-exercise lowering of T_{Core} or T_{Skn} increases the reserve for heat storage (Olschewski & Bruck, 1988) lowers exercise T_{Core} and has been demonstrated to improve cardiovascular efficiency and exercise capacity (Arngrimson *et al.,* 2004; Booth *et al.,* 1997; Lee *et al.,* 2008; Olschewski & Bruck, 1988). Previously employed strategies include cold air exposure (Olschewski & Bruck, 1988), cold water immersion (Booth *et al.,* 1997), or ice vests (Arngrimson *et al.,* 2004); none of which represent a consistently practical, comfortable, or effective intervention with widespread utility for athletic or occupational environments (Marino, 2002; Quod *et al.,* 2006). Furthermore, methods such as these used to cool the body from the outside can induce vasoconstriction (Park *et al.,* 1999) which can potentially impair muscle function (Peiffer *et al.,* 2008).

However, endogenous cooling methods such as ingestion of either crushed ice or cold fluid (0-4^{o}C) represent highly practical pre-cooling strategies, particularly since pre-exercise fluid intake is a recommended and well-practiced strategy to ensure euhydration prior to exercise in heat (Sawka *et al.,* 2007). In fact, combining cooling and fluid replacement is more effective for improving subsequent exercise performance than either cooling or rehydration alone (Hasegawa *et al.* 2006). The ingestion of cold

water during prolonged exercise has been shown to reduce T_{Rec} (Gisolfi & Copping, 1974; Lee & Shirreffs, 2007; Mundel *et al.*, 2006; Wimer *et al.,* 1997), lower HR (Mundel *et al.*, 2006), and improve exercise capacity (Mundel *et al.*, 2006). It should be noted that Gisolfi & Copping (1974) performed no statistical analysis, and that in the study of Mundel *et al.* (2006) drinking was *ad libitum* with a preference for the colder fluid. Further, ingestion of cold water has been shown to reduce T_{Rec} when ingested prior to exercise (Lee *et al.*, 2008), and when combined with the continued ingestion of 500-700 mL of cold fluid during exercise, improve performance (Lee *et al.*, 2008). However, it is not known whether exercise performance would improve if fluid was ingested pre-exercise alone.

8.1.2. Plasma Volume Expansion

Pre-exercise P_{Vol} expansion is another intervention capable of improving cardiovascular function, thermoregulation and exercise capacity (Berger *et al.*, 2006; Coyle *et al.*, 1986; Kanstrup *et al.*, 1992; Sims *et al.*, 2007a; 2007b). Strategies employed to increase P_{Vol} include intravenous infusion of saline or a P_{Vol} expander such as dextran or albumin, which are both impracticable and prohibited in athletic events (WADA, 1999). Pre-exercise ingestion of fluid with a high sodium concentration (157-164 $mmol \cdot L^{-1}$) represents a more efficient method of promoting hypervolemia (Greenleaf *et al.*, 1997; 1998; Sims *et al.*, 2007a; 2007b). Sims *et al*. (2007a; 2007b) demonstrated P_{Vol} expansion of 4.5 and 4.3 %, in males and females, respectively following ingestion of 10 $ml \cdot kg^{-1}$ of a high sodium (164 $mmol \cdot L^{-1}$) drink. The interventions lowered HR and T_{Rec} whilst running at 70 % $\dot{V}O_{2max}$ and increased time to exhaustion (or a critical T_{Rec} of 39.5°C) by 21 and 25 %, respectively. In these studies, drinking commenced 105 and 80 min before exercise and ceased 45 and 20 min before exercise, after which elevated P_{Vol} returned to baseline. This length of drinking strategy is unfeasible in many athletic and occupational situations and should be shortened to increase the viability of the intervention. Further it is not known whether greater physiological and performance benefits would be found if P_{Vol} remained elevated at the start of exercise.

8.1.3. Aims & Hypotheses

The object of the present study was to investigate the effects on physiological functioning and exercise capacity in the heat, of three pre-exercise interventions: i) pre-cooling with cold fluid ingestion ii) P_{Vol} expansion with a high sodium drink; iii) the combination of both treatments. The protocol will employ a pre-exercise strategy in the same environmental temperature as the exercise period. This will represent a more ecologically valid scenario, than when drinking and exercise take place in a thermo-neutral and warm environment, respectively (Lee *et al.*, 2008; Sims *et al.*, 2007a; 2007b). This study will also employ exercise intensities calculated as percentages of the difference between gas exchange threshold and $\dot{V}O_{2max}$, which are more likely to normalize a given intensity than the use of a percentage of $\dot{V}O_{2max}$ alone (Whipp & Rossiter, 2005). It is hypothesised that compared with a control, both cold and high sodium drink ingestion would significantly increase exercise capacity and that the combination intervention would induce an additive and significant benefit over either intervention employed in isolation.

8.2. Materials and Methods

Eight males with mean ± SD, age: 24 ± 4 yr, body mass: 77 ± 6 kg and $\dot{V}O_{2max}$ 54 ± 5 $ml \cdot kg^{-1} \cdot min^{-1}$, completed this single-blind, counter-balanced controlled repeated measures design. To ensure participants were not heat acclimatized, testing took place during the winter months of the northern hemisphere (Oct-Dec; average outdoor temperature of 5-8^{o}C). Each participant performed four experimental trials separated by > 3 d. Participants initially completed an incremental maximal cycle exercise test [$\dot{V}O_{2max}$] for determination of exercise intensity. On the basis of the $\dot{V}O_2$-work rate relationship, the power output equivalent to 10 and 60 % of the difference between GET and $\dot{V}O_{2max}$ was calculated and assigned to subsequent tests.

8.2.1. Experimental Trials

During the experimental sessions, participants wore shorts, socks and cycling shoes and were provided with a towel to clear residual perspiration. Participants entered the environmental chamber that was set at (33 ± 0.2^{o}C, 50 ± 0.6 % relative humidity, V_{α}

≈ 2.5 $m \cdot s^{-1}$), mounted the cycle ergometer (Lode Excalibur Sport V2, Lode BV, Groningen, Netherlands) and remained seated for 15 min to ensure steady state P_{Vol} and Constituents. A capillary blood sample was taken for determination Hct, Hb and a venous blood sample was taken for determination of P_{Osm} and P_{Na}.

8.2.2. Rest Phase

Participants remained seated for a further 45 min in the environmental chamber. Half way through this period, participants were required to walk for 1 min to prevent venous pooling (Sims *et al.,* 2007a; 2007b). During rest, participants ingested 10 $mL \cdot kg^{-1}$ BM of one of four lemon-flavored drinks: control (26°C, 10 mmol $Na^{+} \cdot L^{-1}$); high sodium (HS; 26°C, 157 mmol $Na^{+} \cdot L^{-1}$); cold (PC; 4°C, 10 mmol $Na^{+} \cdot L^{-1}$); or combined cold and high sodium (PC-HS; 4°C, 157 mmol $Na^{+} \cdot L^{-1}$). Drink properties are listed in table 8.1. Drinks were prepared in a powdered format by SIS Ltd. (Science in Sport, Ashwood Research Labs, Blackburn, UK), then converted into liquid 2-4 hours in advance and either kept 26°C in a thermostatically controlled water bath (GD100, Grant Instruments Ltd, Cambridge) or refrigerated at 4°C. All drinks were blinded to the participant and allocated in a square lattice design. Drinks were served in concealed containers, in four 2.5 $mL \cdot kg^{-1}$ BM (~190 ± 25 mL) boluses at 45, 32, 19 and 6 min prior to the start of exercise. After 45 min rest, participants voided their bladder, had their nude BM measured, and a capillary and venous blood sample was taken.

Table 8.1. Drink Properties

	Control	PC	HS	PC-HS
Temperature (°C)	26	4	26	4
Sodium Chloride ($g \cdot L^{-1}$)	0.2	0.2	7.7	7.7
Sodium Citrate ($g \cdot L^{-1}$)	nil	nil	4.5	4.5
Sodium Conc. ($mmol \cdot L^{-1}$)	10	10	157	157
Carbohydrate ($g \cdot L^{-1}$)	2	2	2	2
Sugars	0.1	0.1	0.1	0.1
Osmolality ($mOsm \cdot kg^{-1}$)	40	40	275	275

8.2.3. Exercise Phase

Following rest phase, participants mounted the same cycle ergometer as used for the preliminary tests and then completed 5 min cycling at 50 W, followed by 45 min of cycling a power output of 211 ± 26 W, which initially elicited 10 % Δ (bout 1). Immediately following this, participants continued cycling to exhaustion at 60 % Δ. Ratings of perceived exertion (Borg, 1974) were recorded at the start and every 5 min during heavy exercise and at exhaustion. No motivation was given to the participant during either exercise bout, although participants were aware of elapsed time during the first bout via measurement of RPE at 5 min intervals. Exhaustion was defined as the point at which the participant was unable to maintain a cycle cadence above 50 rpm. Upon termination of exercise, participants cycled for 5 min at 50 W. Venous and capillary blood samples were then taken whilst the participant remained seated on the ergometer. All instrumentation was then removed and the participant left the environmental chamber, whence a post-exercise urine sample was obtained and nude BM recorded. Unevaporated sweat was removed with a towel before nude BM measurement. U_{Vol} voided during rest or exercise was also recorded.

8.2.4. Calculations

HR_{Drift} was calculated by subtracting the mean HR for the 45th minute from the mean HR for the 10th minute. For example:

$$HR_{Drift} = HR_{45} - HR_{10}$$

8.3. Results

Table 8.2 illustrates fluid balance over the whole protocol. Pre-rest and pre-exercise BM was similar in all trials ($P > 0.05$). Ingestion of the high sodium drink during rest significantly reduced U_{Vol} when measured just before the start of exercise ($P < 0.02$). There was no significant difference in U_{Vol} between HS and PC-HS ($P = 0.34$) or between control and PC ($P = 0.21$). During exercise, sweat losses were significantly lower in the pre-cooling conditions compared with control and HS ($P < 0.01$).

8.3.1. Plasma Volume & Constituents

Pre-rest P_{Na} (140.4 ± 2.9 mmol·L^{-1}, P = 0.99) and P_{Osm} (284 ± 4 mOsmol·kg^{-1}; P = 0.91) were similar during the four trials. The changes in P_{Na}, P_{Osm} and P_{Vol} during rest are illustrated in Table 8.3. Compared with baseline there was no change in P_{Na}, P_{Osm} or P_{Vol} with the control or PC, whereas there was a significant increase in both variables with HS and PC-HS (P < 0.05).

Table 8.2. Fluid Balance during rest and exercise

		Control	PC	HS	PC-HS
Baseline	BM (kg)	77.5 ± 5.7	77.4 ± 5.7	77.4 ± 5.7	77.6 ± 5.6
	Fluid Intake	774 ± 56			
Rest	Sweat Loss	223 ± 78	199 ± 89	285 ± 88	210 ± 64
	Urine Output	305 ± 90[b]	321 ± 96[b]	164 ± 55[a]	179 ± 61[a]
Post-Rest BM (kg)		77.7 ± 5.6	77.7 ± 5.6	77.7 ± 5.7	77.9 ± 5.7
Exercise	Sweat Loss (L)	1.42 ± 0.41[b]	1.23 ± 0.32[a]	1.54 ± 0.33[b]	1.22 ± 0.42[a]
Post-Exercise BM (kg)		76.3 ± 5.9[b]	76.4 ± 5.8[a]	76.2 ± 5.9[b]	76.8 ± 5.1[a]
Net BM loss (%)		-1.5 ± 0.3	-1.3 ± 0.2	-1.5 ± 0.2	-1.1 ± 0.2

Fluid balance during rest and exercise. Units are in litres [mL] unless stated. n=8 unless stated. BM = body mass. [a] denotes significant difference from control, [b] denotes significant difference from PC-HS (P < 0.05).

8.3.2. Thermoregulation

Baseline T_{Rec} was similar between trials (37.1 ± 0.2°C; P = 0.41). Figure 8.1 illustrates the T_{Rec} response during both rest and exercise. Ingestion of both PC and PC-HS lead to an equal and significant decrease in T_{Rec} during rest of -0.4 ± 0.2°C (P < 0.001). In comparison neither the control nor HS had an effect on T_{Rec} (P = 0.67). With the control (37.7 ± 0.3°C), mean T_{Rec} during heavy exercise was significantly reduced with PC (0.3 ± 0.2°C; P < 0.04) and PC-HS (0.4 ± 0.3°C; P < 0.006), but was not significantly different with HS (P = 0.57). Yet at the end of heavy exercise, there was only a tendency for a reduced T_{Rec} with PC (-0.32 ± 0.36°C, P = 0.06) and PC- HS (-

0.37 ± 0.44°C, P = 0.07) compared with control. T_{Rec} was not significantly different from control with HS (P = 0.36). At exhaustion, T_{Rec} reached a similar temperature of 38.7 ± 0.5°C in all trials (P = 0.36).

Table 8.3. Change in Plasma osmolality, sodium concentration and volume during rest.

	P_{Na}	P_{Osm}	P_{Vol}
Control	0.1 ± 1.6	-0.7 ± 2.2	0.1 ± 0.9
PC	0.3 ± 1.6	0.6 ± 2.0	0.2 ± 2.1
HS	4.5 ± 2.2[a]	4.1 ± 3.5[a]	4.8 ± 1.4[a]
PC- HS	5.0 ± 2.5[b]	4.2 ± 3.9[b]	5.1 ± 1.2[b]

Changes in P_{Na} (mmol·L^{-1}), P_{Osm} (mOsm·kg^{-1}), and P_{Vol} (%) were measured pre- and post-rest period. Significant differences from control are denoted by [a] P < 0.01, [b] P < 0.05. Significant changes in P_{Na} and P_{Osm} frm baseline are discussed n parenthesis.

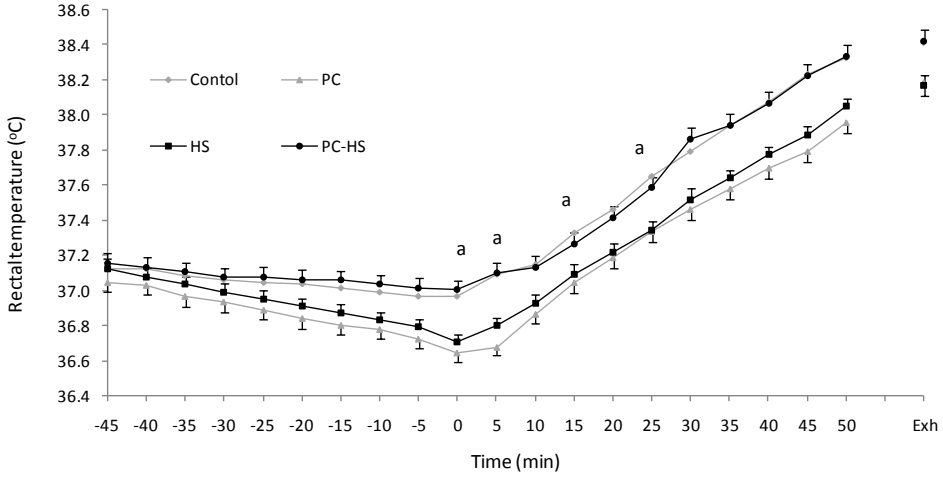

Figure 8.1. Mean ± SE rectal temperature. T_{Rec} measured over the whole experimental protocol: rest (-45-0 min); warm-up (0-5 min); heavy exercise (5-50 min); and at exhaustion (Exh). Significant differences from control denoted by [a] for both PC and PC-HS at 10 min time points (P < 0.05). Mean T_{Rec} was also significantly reduced from control with PC and PC-HS.

8.3.3. Heart rate

Baseline HR (67 ± 5 b·min^{-1}, P = 0.58) and pre-exercise HR (77 ± 5 b·min^{-1}, P = 0.18) were similar in all trials. The HR responses during exercise are illustrated in figure 8.2. HR_{Drift} (change in HR from 10 to 50 min) was significantly attenuated with PC (8 ± 5 b·min^{-1}, P < 0.004), HS (9 ± 4 b·min^{-1}, P < 0.001) and PC-HS (17 ± 5 b·min^{-1}, P < 0.002). PC-HS had an additive effect over both PC (P < 0.04) and HS (P = 0.002). Mean HR was also significantly reduced, by 4, 5 and 8 b·min^{-1}, respectively with PC, HS and PC-HS (P < 0.03). HR_{Drift} was negatively correlated with the change ΔT_{Rec} during rest (r = 0.87, P < 0.001) and the ΔP_{Vol} and during rest (r = 0.71, P < 0.001).

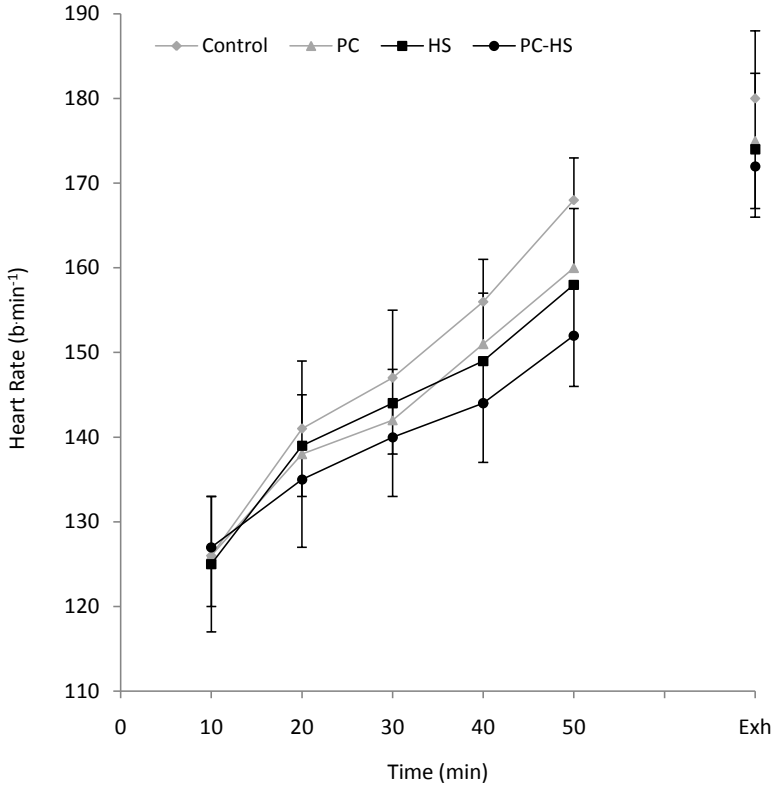

Figure 8.2. Mean ± SD heart rate. HR (b·min^{-1}) measured during exercise (5-50 min) and at exhaustion (Exh). [a] denotes significant differences from control (P < 0.05).

8.3.4. RPE

With control, mean RPE during heavy exercise was (13 ± 1). There was a non-significant trend for a lower RPE with PC (12 ±1; P = 0.08), but no significant difference in RPE between control and HS (13 ± 1; P = 0.22). With PC-HS RPE was significantly lower than the three other interventions (12 ± 1, P < 0.02).

8.3.5. Exercise Capacity

There was no trial order effect on exercise capacity (P = 0.89). Figure 8.3 illustrates the time to exhaustion during the performance test. Participants cycled 13, 15 and 24 % longer than control with PC, HS and PC- HS, respectively (P < 0.01). Performance time was significantly greater than both PC and HS with PC-HS (P < 0.05). Exercise capacity was positively correlated with ΔT_{Rec} during rest (r = 0.65, P < 0.001) and ΔP_{Vol} during rest (r < 0.61, P < 0.001) but not with T_{Rec} at the start of the performance test (r = 0.25, P = 0.16), or P_{Vol} post exercise (r = 0.12, P = 0.50).

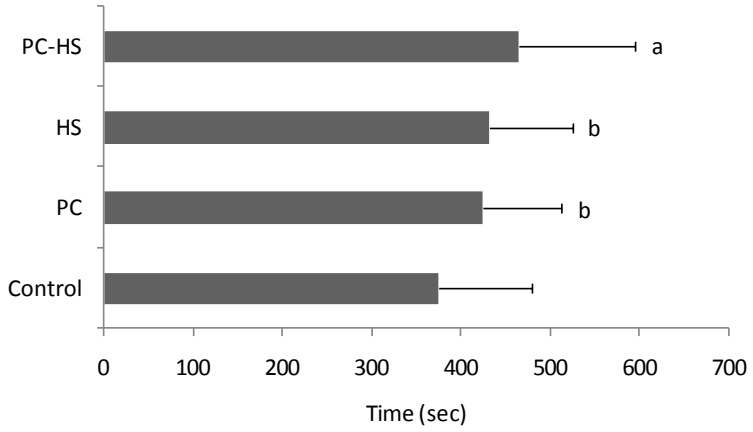

Figure 8.3. Time to exhaustion. Time (sec) measured during time to exhaustion protocol subsequent to a heavy exercise bout. [a] denotes a significant difference from control, [b] denotes a significant difference from control and PC-HS ($P \leq 0.05$).

8.3.6. Regression Analysis

Regression analysis tested the strength of the association between TTE as a dependent variable and changes in P_{Vol} [ΔP_{Vol}] and T_{Rec} [ΔT_{Rec}] during rest and exercise; mean $T_{Rec,}$ final T_{Rec}, final ΔT_{Rec}, mean HR, HR_{Drift}, final HR, mean RPE, and final RPE during heavy exercise, as independent variables. Analysis revealed that a

three-component model incorporating HR_{Drift} during heavy exercise, and ΔP_{Vol} and ΔT_{Rec} during rest explained 78 % of the variance in TTE (see Table 8.4).

Table 8.4. Summary of multiple regression analysis for the prediction of time to exhaustion.

Independent Variable	Constant	B	SE B	β	R^2
Step 1					
HR_{Drift}	847.66	-23.72	3.946	-0.74	-0.53[b]
Step 2					
HR_{Drift}	823.27	-19.98	3.11	-0.62	
ΔP_{Vol} rest		2.64	42.64	0.46	0.73[b]
Step 3					
HR_{Drift}	647.05	-14.59	3.39	-0.46	
ΔP_{Vol} rest		19.55	3.92	0.44	
ΔT_{Rec} rest		176.56	62.80	0.30	0.78[a]

Significant F-change, [a] $P < 0.05$, [b] $P < 0.001$.

8.4. Discussion

The main findings of this study are, firstly that pre-exercise ingestion of both a cold and a high sodium drink increases exercise capacity in the heat; and that the combination of both interventions have an additive effect, enhancing performance to a greater extent than either used in isolation. The findings provide supporting evidence for cold drinks, high sodium, and their combination as practical intervention strategies to improve physiological function and exercise capacity in the heat.

8.4.1. Cold Fluid Ingestion

Ingestion of 774 ± 56 mL of cold (4^{o}C) vs. room temperature (26^{o}C) fluid reduced pre-exercise T_{Rec} by 0.4^{o}C with both PC and PC-HS. Commensurate reductions in T_{Core} have previously been demonstrated following ingestion of 700-900 mL of cold (4^{o}C) vs. body temperature (38^{o}C) water during pre-exercise rest (Lee *et al.,* 2008). When an additional 500-600 mL of the cold fluid was ingested during exercise to exhaustion at a moderate intensity in the heat (66 % $\dot{V}O_{2max}$; 35^{o}C; 60 % RH), Lee *et al.* (2008) reported reductions in mean T_{Rec}, HR and RPE, and an increase in endurance performance. Given that lower T_{Core}, lower HR's and improvements in performance have also been demonstrated when cold fluid was ingested during exercise (Lee & Shirreffs, 2007; Mundel *et al.,* 2006), the extent of improvements attributable to interventions prior to exercise in the study by Lee *et al.,* (2008) is unknown. Furthermore, since the ingestion of refrigerated fluid is uncommon during athletic events, the discovery that the same thermoregulatory, cardiovascular and performance benefits can be procured with prior ingestion alone, is highly significant.

Recently, the pre-exercise ingestion of crushed ice has also been found to be effective in lowering T_{Core} (Ihsan *et al.,* 2010; Ross *et al.,* 2010; Siegel *et al.,* 2010; Stanley *et al.,* 2010) and in the most part improving exercise performance (Ihsan *et al.,* 2010; Ross *et al.,* 2010; Siegel *et al.,* 2010). However, the ingestion of larger volumes of crushed ice may be unpleasant and as such may not represent an ideal pre-exercise routine for athletes. Furthermore, providing crushed ice may not be logistically possible as cold fluid just prior to exercise.

The lower T_{Core} and hence greater capacity for heat storage after pre-cooling permits a greater scope for metabolic heat production lessening the strain on the thermoregulatory system compared with control conditions during exercise (Booth *et al.,* 1997; Lee *et al.,* 2008; Wilson *et al.,* 2002). The creation of a heat deficit increases the time, and therefore the work which can be achieved before the onset of heat dissipating mechanisms (Hasegawa *et al.,* 2006; Wilson *et al.,* 2002). Thus the present study and others before (Booth *et al.,* 1997; Hasegawa *et al.,* 2006; Lee *et al.,* 2008; Olshewski & Bruck, 1988; Wilson *et al.,* 2002) have observed a reduced sweat rate following pre-cooling compared with control conditions.

The present study demonstrated an attenuation of HR_{Drift} and a reduction in mean HR with both cold drinks, compared with the control. The lower HR with PC

during exercise persisted for just 25 min, which in line with previous studies in which temporary reductions in HR lasted a similar length of time (15-35 min; Booth *et al.*, 1997; Lee *et al.*, 2008). Concomitant with the temporary reduction in HR, differences in T_{Rec} between the cold drinks and control lasted for just 20 min. This pattern of responses is reflective of the strong relationship found between mean HR and mean T_{Rec} during exercise in the present study (r = 0.87). The increase in HR with increasing T_{Core} is a proposed consequence of increasing autonomic nervous system activity, in response to elevated blood temperature detected by the sinoatrial node (Johnson & Proppe, 1996). Evidence of a reduced cardiovascular strain was the reduced RPE during exercise with both PC and PC-HS compared with control. The reduced RPE in this study appear to be more a function of thermoregulatory, rather than cardiovascular strain; despite a reduced HR_{Drift} and mean HR, RPE was not significantly different from control, with HS.

Pre-cooling with cold fluid ingestion significantly increased time to exhaustion by 13, and 24 % with PC and PC- HS, respectively. An increase in exercise capacity has been reported by several other studies following pre-cooling (Olschewski & Bruck, 1988; Booth *et al.*, 1997; Lee *et al.*, 2008; Arngrimson *et al.*, 2004), yet the precise mechanism by which performance improves is unknown. A number of researchers have suggested the existence of a critical T_{Core}, at which point performance is terminated to protect the CNS (Gonzalez-Alonso *et al.*, 2008; Gonzalez-Alonso *et al.*, 1999; Nielsen *et al.* 1993; Parkin *et al.*, 1999). Evidence supporting this concept is the observation that following pre-cooling participants fatigued at the same T_{Core} (40^{o}C) despite varying durations of exercise and rates of heat storage (Gonzalez-Alonso *et al.*, 1999; Booth *et al.*, 1997; Kay *et al.*, 1999; Lee & Haymes, 1995; Schmidt & Bruck, 1981).

However, T_{Rec} and T_{Eso} > 40°C have been observed in distance runners and were apparently well tolerated for extended periods of time (Byrne *et al.*, 2006; Christensen & Ruhling; Ely *et al.*, 2009; Robinson, 1963). In the present study, mean T_{Rec} at exhaustion in was just 38.7^{o}C, thus it is unlikely a critical T_{Core} influenced performance here. Instead it is suggested the reduced cardiovascular strain experienced during bout one with PC and PC-HS may have increased, or be reflective of an increase in the capacity for subsequent anaerobic performance. Regression analysis supports this claim, revealing HR_{Drift} as a significant predictor of exercise capacity. A common feature of these studies (Byrne *et al.*, 2006; Christensen & Ruhling; Ely *et al.*, 2009; Robinson, 1963) and that of the present is that environmental temperature was either cool (<15^{o}C)

or warm (25-33^{o}C) but not hot (>35^{o}C). Under these environmental conditions the reliance on BF_{Skn} to transfer body heat from the body core to the periphery is relatively low due to modest skin temperatures and large core-to skin temperature gradients (Ely *et al.,* 2009). Consequently a larger CBV might maximise cardiovascular efficiency (Gonzalez-Alonso *et al.,* 2000; Kenefick *et al.,* 2004; Nybo & Nielsen, 2001b; Rowell *et al.,* 1969) and would sustain a higher level of exercise performance. During those studies undertaken in a hot environment the higher skin temperatures (e.g. Gonzalez-Alonso *et al.,* 1999; Nielsen *et al.,* 1993; Nielsen *et al.,* 2001), will increase BF_{Skn}, reduce CBV and impair cardiovascular performance.

Cardiovascular factors may also play a significant role in the development of fatigue during exercise in the heat when performed at high intensities (Nybo, 2008), such as the present study. This notion is supported by the work of Wingo and colleagues (Wingo *et al.,* 2005a; 2005b; Wingo & Cureton, 2006a; 2006b) which sought to established a relationship between CV_{Drift} and the decline in exercise capacity $\dot{V}O_{2max}$which occurred between 15 and 45 min of moderate intensity exercise in conditions similar to the present study (60 % $\dot{V}O_{2max}$; 35^{o}C, 40 % RH). Across all these studies; the greater the increase in HR (and reduction in SV), the greater the decline in performance. Furthermore, Wingo and colleagues (Wingo *et al.,* 2005a; Wingo *et al.,* 2006b) also observed that the decline in performance was significantly reduced when CV_{Drift} was attenuated by reducing T_{Core}.

8.4.2. High Sodium Fluid Ingestion

Pre-exercise ingestion of a high sodium concentration (157 mmol·L^{-1}) increased P_{Vol} by 4.8 and 5.1 % with HS and PC-HS, respectively. The magnitude of change is commensurate with previous findings following the ingestion of the same volume of a high vs. low sodium drink (157-164 mmol·L^{-1}) prior to exercise (Greenleaf *et al.,* 1997; 1998; Sims *et al.,* 2007a; 2007b). The ingestion of sodium elevates P_{Na} and P_{Osm} which stimulates the release of antidiuretic hormones that promote fluid retention (Johnson & Thurnhorst, 1997). This is evidenced by the small but significant reduction in U_{Vol} with HS and PC-HS, compared with control. The reduced U_{Vol} in the present study in respect of past studies (Sims *et al.,* 2007a; 2007b; Johanssen *et al.,* 2009) is a consequence of the timing of ingestion. Previous studies have either employed drinking regimes that have been impractically short (Greenleaf *et al.,* 1997; 1998), or preceded exercise by a

long duration (i.e. 20 or 45 min) such that P_{Vol} expansion returns to baseline before exercise begins (Sims *et al.,* 2007a; 2007b*).* The current study represents an advance on previous approaches since it employed a more time efficient yet equally effective means of achieving hypervolemia.

P_{Vol} expansion had no effect on thermoregulation during exercise; neither sweat rates nor T_{Rec} were significantly different between high and low sodium conditions. The effects of P_{Vol} expansion on thermoregulation has produced equivocal findings with some demonstrating reduced T_{Core} (Deschamps *et al.,* 1989; Nose *et al.,* 1990; Sims *et al.,* 2007a; 2007b) and others reporting no effect (Fortney *et al,* 1981a; Grant *et al.,* 1997; Watt *et al.,* 2000). The failure to demonstrate changes in thermoregulation in the present study could be attributable to the elevated P_{Osm} at the start of exercise with HS and PC-HS. Hyperosmolality has previously been shown to reduce skin blood flow, impairing sweating and lowering the capacity for heat dissipation (Nielsen, 1973). Thus any potential increase in sweating as a result of P_{Vol} expansion may have been compromised by hyperosmolality-induced reductions in sweat rate. In contrast to our findings Sims and colleagues (2007a; 2007b) demonstrated a reduced rate of rise in T_{Rec} following the ingestion of sodium. In that study, the retracted drinking strategy allowed P_{Osm} to return to baseline levels by the start of exercise. However, contrary to this explanation, there were no significant differences in sweat rates between PC and PC-HS despite a similar elevation in P_{Osm} between HS and PC-HS at the start of exercise, suggesting that, at least with the cold drinks, hyperosmolality did not impair thermoregulation.

The observation of a reduction in HR_{Drift} following P_{Vol} expansion is consistent with a number of previous studies (Montain & Coyle, 1992b; Sims *et al.,* 2007a; 2007b; Deschamps *et al.,* 1989; Fortney *et al.,* 1981a; Sawka *et al.,* 1983; Grant *et al.,* 1997). In those studies in which pre-exercise P_{Vol} did not influence HR, exercise was brief (Berger *et al.,* 2006; Kanstrup *et al.,* 1992) or conducted in a thermo-neutral environment (Coyle *et al.,* 1990; Hopper *et al.,* 1988). The decrements in cardiovascular functioning as a result of exercise-induced dehydration in the control condition have a significant impact on exercise performance. As already discussed in the previous chapter (Section 7.5.2.) a series of studies by Wingo and colleagues have been able to demonstrate a causal link between CV_{Drift} and $\dot{V}O_{2max}$ (Ganio *et al.,* 2006; Wingo *et al.,* 2005; Wingo & Cureton, 2006a; Wingo & Cureton 2006b). These studies manipulated

the degree of CV_{Drift} during prolonged exercise by lowering ambient temperature (Wingo *et al.*, 2005a), cooling with air flow (Wingo & Cureton, 2006b) and reducing the exercise intensity (Wingo & Cureton, 2006a). Similarly, by altering the level of dehydration with fluid ingestion, Ganio *et al.* (2006) found further support for the relationship between CV_{Drift} and $\dot{V}O_{2max}$. Following 2 h of cycling in a warm environment without fluid ingestion in which SV had decreased 14% $\dot{V}O_{2max}$ was reduced by 9 %. In contrast, when dehydration was prevented with fluid ingestion, the decline in SV and $\dot{V}O_{2max}$ were both eliminated. Thus, reducing dehydration improves performance in prolonged exercise, in part, by mitigating the decline in SV and $\dot{V}O_{2max}$. Accordingly the present study found an improvement in exercise capacity with pre-exercise high sodium ingestion, likely by attenuating the decline in SV. Regression analysis supports this notion, revealing ΔP_{Vol} during rest as a significant predictor of exercise capacity. Thus in the present study the performance differential with PC, HS and PC-HS appears to be a consequence of the degree of HR_{Drift} during the preceding 45 min bout.

The observed increase in exercise capacity with the high sodium drink compared with control, is consistent with the majority of studies which induced pre-exercise P_{Vol} expansion (Berger *et al.*, 2006; Sims *et al.*, 2007a; 2007b; Coyle *et al.*, 1990; Kanstrup & Ekblom, 1982; Luetkemeier *et al.*, 1994). Those studies which have reported no change (Coyle *et al.*, 1986; Deschamps *et al.*, 1989; Watt *et al.*, 2000) or a decrease in performance (Coyle *et al.*, 1986; Kanstrup *et al.*, 1992) employed an exercise protocol which was either too brief (Kanstrup *et al.*, 1992), or conducted in a thermo-neutral environment (Coyle *et al.*, 1986; Deschamps *et al.*, 1989; Kanstrup *et al.*, 1992).

It should be acknowledged that factors other than P_{Vol} expansion may have contributed to the increased exercise capacity demonstrated with HS. For example, the ingestion of sodium citrate, one of the constituents within our drinks has previously been found to enhance the buffering capacity of hydrogen ions and increase exercise performance (Oopik *et al.*, 2003). However, the volume of sodium citrate of 0.3 - 0.5 $g \cdot kg^{-1}$ (~22-37 g) ingested was three to four times greater than in the present study, thus it is unlikely the increase in exercise capacity in this study resulted from enhanced buffering capacity.

8.4.3. Combined Cold and High Sodium Fluid Ingestion

The combined ingestion of a cold and high sodium drink had a powerful additive effect on HR_{Drift} and exercise performance, but not T_{Rec}, sweat rates, or RPE. The additive effects represent the amalgamation of the individual effects of both interventions. Cold high sodium drinks have been previously been demonstrated to improve performance. For example, the drinks served in the study of Sims and colleagues (2007a; 2007b) were chilled at 4^{o}C until just before serving. Additionally, in a poorly controlled study, Dill *et al.* (1973) had a range of participants, including himself (aged 81) ingest water and salt served at 15^{o}C in equal portions to that lost during 84-120 min walking at 6 km·h^{-1} in variable heat (37-47^{o}C). However, neither study design allowed elucidation of the separate effects of sodium ingestion and drink cooling. Thus these findings represent an advancement of the previous results in the literature.

8.5. Conclusion

The ingestion of cold and high sodium fluid were effective in reducing pre-exercise T_{Rec} and increasing pre-exercise P_{Vol}, respectively and were associated with reduced HR_{Drift} and increased exercise capacity. The most important finding is that the combination of pre-cooling and P_{Vol} expansion had an additive effect, reducing HR_{Drift} and increasing exercise capacity to a greater extent than the individual intervention alone. Since fluid ingestion is not always possible during athletic and occupational scenarios, the demonstration of three highly efficient and practical pre-exercise strategies to improve exercise performance in the heat is highly significant. Future research is recommended to examine whether further benefits would be procured if fluid was also ingested during exercise.

Chapter 9 – Study 6

Pre-exercise sodium ingestion increases stroke volume and time trial performance in untrained males exercising in the heat

9.1. Introduction

During competitions in a hot environment athletes can experience significant cardiovascular stress that impacts on their ability to perform effectively (Coyle *et al.*, 1986). Fundamental to cardiovascular performance is blood volume (Montain & Coyle, 1992; Sawka *et al.*, 1984), as demonstrated by a reduction in SV with hypovolemia of 10-12 % (Coyle *et al.*, 1986; Fortney *et al.*, 1983); and an increase in SV with P_{Vol} expansion of 7-12 % (Coyle *et al.*, 1986; 1990; Fortney *et al.*, 1981a; 1983; Grant *et al.*, 1997; Hopper *et al.*, 1988; Kanstrup & Ekblom, 1982; Krip *et al.*, 1997; Montain & Coyle, 1992b).

Given the importance of blood volume, it is not surprising that strategies have sought to increase P_{Vol} both before and during exercise. Such interventions represent highly effective means of improving cardiovascular function, thermoregulation and exercise capacity (Berger *et al.*, 2006; Coyle *et al.*, 1986; Kanstrup *et al.*, 1992; Sims *et al.*, 2007a; 2007b). Frequently, pre-exercise P_{Vol} expansion strategies have involved the intravenous infusion of saline, dextran or albumin, which are both impracticable and prohibited in athletic events (WADA, 1999).

One pre-exercise strategy which represents a more efficient method of promoting hypervolemia is the ingestion of fluid with a high sodium concentration (157-164 $mmol \cdot L^{-1}$) (Greenleaf *et al.*, 1997; 1998; Sims *et al.*, 2007a; 2007b). The ingestion of a high sodium concentration promotes greater fluid retention than if water alone is ingested. Such is the potential of sodium, that Sims *et al*. (2007a; 2007b) demonstrated P_{Vol} expansion of 4.5 and 4.3 %, in males and females, respectively following ingestion of 10 $ml \cdot kg^{-1}$ of a high sodium (164 $mmol \cdot L^{-1}$) drink. These interventions lowered HR and T_{Rec} whilst running at 70 % $\dot{V}O_{2max}$ and increased time to exhaustion (or a critical T_{Rec} of 39.5°C) by 21 and 25 %, respectively.

Along with chapter 8, these are the only studies which have employed such a high sodium concentration prior to exercise. Whilst the initial findings seem positive,

further research is necessary to fully explore the capabilities of such drinks. One area in particular which needs addressing is the impact of high sodium intake on SV and $\dot{Q}$ during exercise. The few previous studies have only examined the effects of pre-exercise sodium ingestion (Greenleaf *et al.,* 1997; 1998; Sims *et al.,* 2006a; 2006b), which is an unlikely scenario in athletic events. Whilst it is not always possible to drink fluid during exercise at a rate of 80-100 % BM losses, commonly recommended by guidelines (e.g. Coyle, 2001), some fluid ingestion is expected and thus it is worthy to investigate whether the continued ingestion of fluid during exercise might induce the same physiological adaptations. Furthermore, there is a need for a more reliable measurement of exercise performance than used previously. Time to exhaustion protocols (e.g. Sims *et al.,* 2007a; 2007b; Study 5) are not replicable to 'real-life' situation; rarely are athletes required to reach maximal effort during events. Instead time trial protocols allow simulation of variable intensities (Palmer *et al.,* 1994) normally witnessed during athletic events. Accordingly, these achieve similar physiological responses to actual events than when the same amount of work is performed at a set intensity (Currell & Jeukendrup, 2008). As such they have been shown to correlate strongly with actual performance during cycling (Russel *et al.,* 2004), whilst time to exhaustion protocols do not (Laursen *et al.,* 2002).

9.1.1. Aims and Hypotheses

The primary aim of this study was to determine whether pre-exercise ingestion of a high sodium drink affects physiological responses and time trial performance during exercise in the heat. Secondly, to examine the additive effect of ingesting a low-sodium sports drink during exercise. It was hypothesised that the ingestion of the high sodium drink would attenuate the rise in HR and decreases in SV and $\dot{Q}$ during exercise and improve time trial performance, when compared with pre-exercise ingestion of the low sodium concentration. Secondly it was also hypothesised that the additional ingestion of fluid during exercise would enhance the differential when compared to the pre-exercise condition alone.

9.2. Materials and Methods

Eight untrained males with mean ± SD, age: 26 ± 7 yr, body mass: 76 ± 6 kg $\dot{V}O_{2max}$: 49 ± 6 $mL \cdot kg^{-1} \cdot min^{-1}$ completed this single-blind, counter-balanced controlled repeated measures design. Participants completed an incremental maximal cycle exercise test for determination of exercise intensity. On the basis of the $\dot{V}O_2$-work rate relationship, the power output equivalent to 10 and 70 % of the difference between gas exchange threshold [GET] and $\dot{V}O_{2max}$ was calculated and assigned to subsequent tests.

9.2.1. Experimental Design

Each participant performed a familiarisation session and four experimental trials separated by > 2 d. One of four drinking strategies was allocated in a square lattice design to each experimental trial and are illustrated in Table 9.1. Rest and exercise were conducted in an environmental chamber (32 ± 0.2°C, 50 ± 0.6 % relative humidity, fan speed ≈ 1.5 $m \cdot s^{-1}$). This would represent a more valid scenario than when rest and exercise were performed in a thermo-neutral and warm environment respectively (e.g. Sims *et al.*, 2007a; 2007b).

Table 9.1. Drinking Protocols

	Pre-exercise	During-exercise
LS-N	Low-Sodium	No fluid
HS-N	High-Sodium	No fluid
LS-F	Low-Sodium	Fluid
HS-F	High-Sodium	Fluid

Participants completed the same pre-exercise routine as with previous chapters. For more details see section 4.2.1. During the experimental sessions, participants wore shorts, socks and cycling shoes and were provided with a towel to clear residual perspiration. Participants entered the environmental chamber that was set at (32 ± 0.2°C, 50 ± 0.6 % relative humidity, V_α ≈ 2.5 $m \cdot s^{-1}$), mounted the cycle ergometer (Lode

Excalibur Sport V2, Lode BV, Groningen, Netherlands) and remained seated for 15 min to ensure steady state P_{Vol} and Constituents. A capillary blood sample was taken for determination Hct, Hb and a venous blood sample was taken for determination of P_{Osm} and P_{Na}.

Participants remained seated for a further 45 min in the environmental chamber but for walking for 1 min, after 22 min to reduce the likelihood of venous pooling (Sims *et al.*, 2007a; 2007b). During rest participants ingested either 10 $mL \cdot kgBM^{-1}$ (776 ± 56 mL) of either a low or high sodium drink in three equal servings at 0, 15 and 30 min. Participants then mounted the same cycle ergometer with identical settings as used for the preliminary tests. Signal morphology-based impedance cardiography (PhysioFlow, Neumedx, Bristol) was used to record HR, SV and $\dot{Q}$. Six electrodes (Physio Flow, Manatec) were attached to each participant at rib, chest, neck, back. These sites were sterilised and shaved for optimal conductance. Duplicate measurements of resting blood pressure (Pro 100V2 dinamap) were taken just prior to exercising whilst seated on a cycle ergometer.

Participants then exercised at a steady state for 5 min at 50 W, followed by 1 h at a power output equivalent to 10 % Δ. RPE and thermal sensation were recorded at the start and every 15 min during exercise and at exhaustion. A thermometer (± 0.01°C; YSI Precision 4000, Henleys Medical, Hertforshire) logged T_{Rec} throughout exercise. Immediately following steady state exercise, participants performed a time trial. The total amount of work (J) was calculated from the work rate equivalent to 70 % Δ multiplied by 900 s. The ergometer was set to linear mode using the equation:

$$W = L\ (rpm)^2$$

where *L* is the linear factor and rpm is the pedaling rate. The factor was chosen to represent a pedal rate of 90 rpm at 70 % Δ. Power output, cadence and work completed was recorded online. Participants were asked to perform the time trial as fast as possible. The only information the participant received was the percentage of work completed relative to target work at 10 % intervals. No verbal encouragement was given, no music was played and no physiological measurements were made during the

performance trial. Performance results were only provided to the participant at the end of their involvement in the study. Following exercise, participants cycled for 5 min at 50 W, then voided their bladder and had their nude BM measured. Participants then left the environmental chamber and remained seated for 15 min before venous and capillary blood samples were taken.

9.2.2. Drink Preparation

During rest, participants ingested 10 $mL \cdot kg^{-1}$ BM of one of two lemon-flavored drinks: high sodium ([HS]157 mmol $Na^{+} \cdot L^{-1}$) or low sodium ([LS] 10 mmol $Na^{+} \cdot L^{-1}$). Drink properties are listed in table 9.2. Drinks were prepared in a powdered format by SIS Ltd. (Science in Sport, Ashwood Research Labs, Blackburn, UK), then converted into liquid 2 h in advance. During exercise, participants ingested either no fluid, or a volume of fluid equal to ~40 % of sweat loss during a preliminary test of a low-sodium regular sports drink in three equal boluses at 0, 15 and 30 min. All drinks were served at 37 ± 0.5^{o}C with the temperature maintained in a thermostatically controlled water bath (GD100, Grant Instruments Ltd, Cambridge). All drinks were blinded to the participant and served in concealed thermally insulated containers.

Table 9.2. Drink Properties

	LS	HS
Sodium Chloride ($g \cdot L^{-1}$)	0.2	7.7
Sodium Citrate ($g \cdot L^{-1}$)	nil	4.5
Sodium Conc. ($mmol \cdot L^{-1}$)	10	157
Carbohydrate ($g \cdot L^{-1}$)	2	2
Sugars	0.1	0.1
Osmolality ($mOsm \cdot kg^{-1}$)	40	307

9.2.3. Calculations

HR_{Drift} was calculated by subtracting the mean HR for the 60^{th} minute from the mean HR for the 10^{th} minute. The same calculation was performed for SV_{Drift}. For example:

$$SV_{Drift} = SV_{60} - SV_{10}$$

9.3. Results

Pre-exercise indicators of hydration: body mass, haemoglobin concentration, U_{Osm}, P_{Na} and P_{Osm} were not significantly different between trials ($P > 0.05$). During rest, fluid intake (745 ± 68 mL, $P = 0.78$) and sweat rate (185 ± 34 mL, $P = 0.56$) were similar between trials however U_{Vol} was significantly greater when both low-sodium drinks were ingested compared with both high sodium drinks ($P < 0.04$).

9.3.1. Plasma Volume and Constituents

A two way ANOVA revealed a significant main effect for drink ($F_{(1, 7)} = 42.43$, $P < 0.001$), time ($F_{(4, 28)} = 92.32$, $P < 0.001$) and the interaction ($F_{(4, 28)} = 14.30$, $P = 0.01$) for P_{Osm} responses during the whole protocol. Similar main effects were observed for the P_{Na} response to both rest and exercise. The changes in P_{Na} and P_{Osm} during both rest and exercise are illustrated in Table 9.3. Whilst ingestion of the low sodium drinks had little effect on either P_{Na} or P_{Osm} ($P > 0.05$), ingestion of both high sodium drinks led to a significant increase in the concentration of both plasma variables ($P < 0.001$), to the extent that there was a significant difference in P_{Na} and P_{Osm} between low and high sodium drinks Post-Rest ($P < 0.01$) *i.e.* the start of exercise. During exercise, P_{Na} and P_{Osm} increased significantly when no fluid was ingested ($P < 0.02$). The increase in P_{Na} and P_{Osm} was partially attenuated with the ingestion of fluid; when comparing LS-N with LS-F, the elevations in P_{Na} (5 ± 4 vs. 3 ± 3 $mmol \cdot L^{-1}$) and P_{Osm} (6 ± 3 vs. 3 ± 3 $mOsm \cdot kg^{-1}$) during exercise were significantly reduced when fluid was ingested. Similarly, when comparing HS-N with HS-F the rises in P_{Na} and P_{Osm} during exercise were also attenuated with fluid intake. There were no differences in P_{Na} or P_{Osm} changes during exercise as a result of the pre-exercise drink selection ($P > 0.05$). Thus the differences between LS-N and HS-N and between LS-F and HS-F were similar Post-TT as they were Post-Rest ($P > 0.05$).

Table 9.3. Fluid balance during rest and exercise.

		LS-N	HS-N	LS-F	HS-F
Baseline BM (kg)		75.8 ± 3.7	75.7 ± 3.8	75.8 ± 3.8	75.8 ± 3.9
	Fluid intake	758 ± 37	757 ± 39	758 ± 38	758 ± 38
Rest	Sweat Loss	182 ± 70	162 ± 76	190 ± 81	172 ± 63
	Urine Loss	318 ± 93[ab]	164 ± 94	327 ± 76[ab]	157 ± 78
Post-Rest BM (kg)		76.1± 3.8[ab]	76.2 ± 3.8	76.0 ± 4.1[ab]	76.3 ± 3.9
	Fluid intake	-	-	532 ± 109	539 ± 95
Exercise	Sweat loss (L)	1.4 ± 0.2	1.2 ± 0.2	1.5 ± 0.2	1.3 ± 0.1
	Urine Loss	0.0	0.0	155 (n=1)	0.0
Post-TT BM (kg)		74.7 ± 5.5	75.0 ± 5.6	75.2 ± 5.4[a]	75.5 ± 5.2[a]
BM loss* (%)		-1.8 ± 0.4[ab]	-1.6 ± 0.3	-1.1 ± 0.5[ab]	-1.0 ± 0.4

Fluid balance during rest and exercise. Units are in litres [mL] unless stated. n=8 unless stated. BM = body mass. *Percentage change in BM corrected for differences in fluid intake between drinking protocols. Respiratory fluid losses are assumed negligible and ignored. [a] denotes significant difference from HS-N; [b] denotes significant difference from HS-F ($P < 0.01$).

Table 9.4. Plasma osmolality and sodium concentration during the whole protocol.

	P_{Osm}			P_{Na}		
	Baseline	Post-Rest	Post-TT	Baseline	Post-Rest	Post-TT
LS-N	283 ± 4	283 ± 3	288 ± 4[ab]	139 ± 3	140 ± 3	145 ± 3[ab]
HS-N	284 ± 4	288 ± 3[a]	295 ± 3[ab]	139 ± 3	145 ± 3[a]	149 ± 4[ab]
LS-F	284 ± 3	284 ± 4	285 ± 3[b]	138 ± 3	138 ± 3	140 ± 3
HS-F	284 ± 3	289 ± 4[a]	291 ± 2[a]	139 ± 4	144 ± 3[a]	146 ± 3[a]

Responses P_{Osm} ($mOsm \cdot kg^{-1}$) and P_{Na} ($mmol \cdot L^{-1}$) to rest and exercise. Significant differences from: [a] baseline; [b] post-rest ($P < 0.05$). Differences between drinks are described in text below.

Percentage changes in P_{Vol} during rest and exercise are illustrated in table 9.4. Analysis of changes in P_{Vol} during the whole protocol using a two factor ANOVA revealed a significant main effect for drink ($F_{1, 7)} = 14.96$, $P = 0.006$), time ($F_{(2, 14)} = 81.43$, $P < 0.001$), and the interaction ($F_{(2, 14)} = 20.79$, $P < 0.001$). Pre-exercise high-sodium ingestion (HS-N & HS-F) increased, pre-exercise P_{Vol} by 4.4-4.5 %, more than the low-sodium drink ($P = 0.002$) As a result of pre-exercise routine, P_{Vol} also remained elevated at the end of exercise with HS-N compared with LS-N ($P < 0.03$), and to a lesser extent with HS-F, compared with LS-F ($P = 0.08$). Exercise induced a substantial decline in P_{Vol} in all conditions, which was slightly reduced when fluid was ingested. Compared to LS-N, the change in P_{Vol} relative to baseline was greater with LS-N than with LS-F ($P = 0.08$), and with HS-N compared with HS-F ($P = 0.09$). The higher variability in readings post exercise had a considerable effect on the failure to find significant differences between drink conditions.

Table 9.5. Percentage change in plasma volume during rest and exercise

	Post-Rest	Post-Exercise
LS-N	0.3 ± 0.7 %;	-11.7 ± 2.3 %
HS-N	4.6 ± 0.8 %[a]	-8.7 ± 2.1 %[a]
LS-F	0.2 ± 1.0 %	-8.8 ± 3.7 %
HS-F	4.7 ± 1.0 %[a]	-5.4 ± 4.2 %

. [a] denotes significant differences from LS-N ($P < 0.05$).

9.3.2. Cardiovascular responses

Resting HR (62 $b \cdot min^{-1}$), SV (77 $mL \cdot b^{-1}$), and $\dot{Q}$ (4.8 $L \cdot min^{-1}$) were similar in all trials ($P > 0.05$). Cardiovascular responses to exercise are illustrated in table 9.6. Two way ANOVAs revealed a significant main effect for drink, time and the interaction for HR, SV and $\dot{Q}$ response during exercise. Ten min into exercise, there were no differences in HR (137 ± 8 $b \cdot min^{-1}$; $P = 0.31$) between trials but there was a trend for greater, SV ($P = 0.09$) and $\dot{Q}$ ($P = 0.08$) with pre-exercise ingestion of the high sodium drinks.

With pre-exercise ingestion of the low sodium drink, steady state exercise (10-60 min) was characterised by a progressive increase in HR ($P < 0.003$) and decline in SV ($P < 0.005$) and a small but significant decrease in $\dot{Q}$ ($P < 0.05$). However, with pre-exercise ingestion of the high sodium drink, HR_{Drfit} ($P < 0.03$) and SV_{Drift} ($P < 0.01$) were partially attenuated, and $\dot{Q}$ was significantly increased ($P < 0.04$). Ingestion of fluid during exercise produced an insignificant decrease in both SV_{Drift} ($P < 0.10$) and HR_{Drift} ($P < 0.08$), in the high-sodium pre-exercise, but had no noticeable effect in the low-sodium pre-exercise condition or on $\dot{Q}$.

9.3.3. Perceptual Responses

A two-way ANOVA revealed a significant main effect for drink ($F_{(1, 7)} = 32.11$, $P = 0.001$), time ($F_{(2, 14)} = 75.51$, $P < 0.001$), and the interaction ($F_{(2, 14)} = 7.91$, $P = 0.04$) for RPE response during exercise. After 10 min of exercise RPE was similar between conditions (12 ± 1; $P = 0.66$).

Table 9.6. Cardiovascular reponses to exercise

	LS-N	HS-N	LS-F	HS-F
HR ($b \cdot min^{-1}$)				
10 min	137 ± 7	136 ± 8	137 ± 7	137 ± 7
60 min	164 ± 8	155 ± 6*	159 ± 7	155 ± 8*
SV ($ml \cdot b^{-1}$)				
10 min	143 ± 5	142 ± 5	144 ± 6	144 ± 7
60 min	114 ± 5	125 ± 5*	119 ± 7	128 ± 6*
$\dot{Q}$ ($L \cdot min^{-1}$)				
10 min	19.6	19.3	19.7	19.7
60 min	18.7	19.4*	18.9	19.8*

Values are in mean ± SD. HR= heart rate; SV = stroke volume; $\dot{Q}$ = cardiac output; * denotes significant difference from LS-N ($P < 0.05$).

During the next 50 min RPE increased steadily in all conditions, but to a lesser extent in the high sodium conditions (14 ± 1 vs. 15 ± 1, $P < 0.01$). Ingestion of fluid during exercise did little to affect RPE ($P = 0.37$). There was no difference in RPE

between conditions at the end of the time trial (18 ± 1, $P = 0.21$). A two-way ANOVA revealed a significant main effect for time ($F_{(2, 14)} = 52.81$, $P < 0.001$), but not drink ($F_{(1, 7)} = 2.81$, $P = 0.20$), or the interaction ($F_{(2, 14)} = 0.81$, $P = 0.45$) for thermal sensation.

9.3.4. Thermoregulation

There was no significant difference in baseline T_{Rec} between conditions (37.0 ± 0.3°C; $P = 0.81$). Similarly, there was no significant difference in T_{Rec} between conditions following rest (37.3 ± 0.4°C; $P = 0.63$). Figure 9.1 illustrates the T_{Rec} response during exercise. A two way ANOVA revealed a significant main effect for time ($F_{(4, 28)} = 72.71$, $P < 0.001$) but not drink ($F_{(1, 7)} = 0.43$, $P = 0.45$), or the interaction ($F_{(4, 28)} = 0.30$, $P = 0.64$) for T_{Rec}.

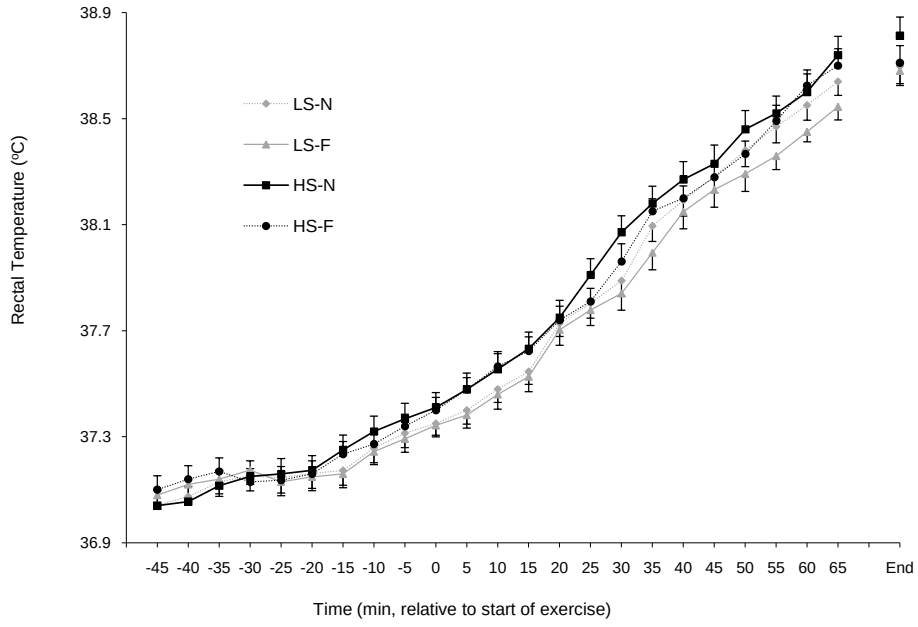

Figure 9.1. Mean ± SE rectal temperature. T_{Rec} measured over the whole experimental protocol: rest (-45-0 min); warm-up (0-5 min); steady state exercise (5-65 min); and post time trial (End). Significant differences are discussed in text.

Across all drinks, T_{Rec} was significantly elevated from baseline Post-rest ($P < 0.01$), and significantly elevated from the start of steady state exercise at each time point from +15 min ($P < 0.01$) and at exhaustion ($P < 0.001$). Sweat loss during exercise is

illustrated in Table 9.3. There was a trend for reduced sweat loss during exercise when high-sodium fluid was ingested during rest (1.31 ± 0.21 vs. 1.46 ± 0.24 L; $P = 0.07$) and also a trend for increased sweat loss with fluid intake during exercise (1.45 ± 0.33 vs. 1.34 ± 0.34 L; $P = 0.10$).

9.3.5. Exercise Performance

There was no trial order effect on exercise capacity ($P = 0.95$). Figure 9.2 illustrates average power output (A) and performance time (B) during the time trial. A one-way ANOVA revealed a significant main effect for drink ($F_{(1, 7)} = 32.11$, $P = 0.001$), time ($F_{(2, 14)} = 75.51$, $P < 0.001$), and the interaction ($F_{(2, 14)} = 7.91$, $P = 0.04$) for time trial time. Similar main effects were demonstrated with time trial power output. Pre-exercise high-sodium ingestion significantly reduced the duration of time trial by ~42 ± 31 sec ($P < 0.01$) and increased average power output by ~13 ± 12 W ($P < 0.008$). Ingestion of additional fluid during exercise had no significant effect on exercise performance in either pre-exercise condition ($P > 0.05$).

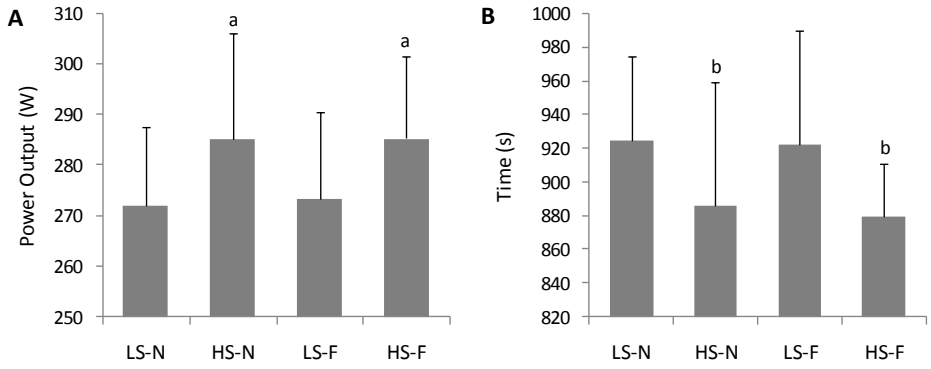

Figure 9.2. Time Trial Performance. Mean ± SD Average power output (A) and performance time (B) during 2800 kJ time trial. [a] denotes significant difference from HS-F, [b] denotes significant difference from LS-F and LS-N ($P < 0.05$).

9.4. Discussion

The main findings of this study are firstly, that pre-exercise ingestion of ~776 mL of a highly concentrated sodium drink reduced HR_{Drift} and SV_{Drift} and attenuated the fall in $\dot{Q}$ and improved time trial performance, compared with the ingestion of the same

volume of a regular low-sodium sports drink. These findings therefore support the use of sodium ingestion as a practical and efficient pre-exercise strategy to improve performance in the heat. This is the first study to report an increase in SV and $\dot{Q}$ following high sodium intake, and to therefore provide better understanding regarding the mechanisms which explain the changes in exercise performance as a result of this highly effective pre-exercise routine. Secondly, this study sought to investigate whether the additional ingestion of 530 mL of a regular sports drink during the first 30 min of exercise provided any additional physiological benefits. The study demonstrated a *non-significant trend* for a reduced T_{Rec} and SV_{Drift}, but no effect on HR, $\dot{Q}$, or time trial performance'. Thus, these findings reject the requirement for further ingestion of fluid during short-term severe intensity exercise.

9.4.1. Fluid Balance

Pre-exercise ingestion of a high sodium concentration (157 $mmol \cdot L^{-1}$) increased P_{Vol} by 4.4-4.5 %. The magnitude of change is commensurate with previous findings following the pre-exercise ingestion of the same relative volume of a high vs. low sodium drink (157-164 $mmol \cdot L^{-1}$; Greenleaf *et al.,* 1997; 1998; Sims *et al.,* 2007a; 2007b). The ingestion of sodium elevated P_{Osm} by 4-5 $mOsm \cdot kg^{-1}$ and P_{Na} by 5-6 $mmol \cdot L^{-1}$, whilst the ingestion of the low-sodium drink had little if no effect on either variable. A rise in P_{Osm} is known to stimulate the release of antidiuretic hormones that promote fluid retention (Johnson & Thurnhorst, 1997). The significant reduction in U_{Vol} just prior to the start of exercise in the high, compared to low-sodium trials is indicative of this antidiuretic response. The reduced U_{Vol} in the present study in respect of past studies (Sims *et al.,* 2007a; 20007b) is a consequence of the timing of ingestion. Previous studies have either employed drinking regimes that have been impractically short (Greenleaf *et al.,* 1997; 1998), or preceded exercise by a long duration (i.e. 20 or 45 min) such that P_{Vol} expansion returns to baseline before exercise begins (Sims *et al.,* 2007a; 2007b). The current study represents an advance on previous approaches since the protocol employed a more time efficient yet equally effective means of achieving hypervolemia.

9.4.2. Cardiovascular Responses

The present study demonstrated a reduction in HR_{Drift} (8-9 $b \cdot min^{-1}$); SV_{Drift} (11-12 $mL \cdot b^{-1}$) and $\dot{Q}_{Drift}$ of 0.8 $L \cdot min^{-1}$ following the pre-exercise ingestion of a high-

sodium compared with a low-sodium sports drink. A reduction in HR has been demonstrated previously following sodium ingestion-induced P_{Vol} expansion of 4.3-5.1 % (Chapter 8; Sims *et al.,* 2007a; 2007b), however a reduced HR_{Drift} alone is insufficient to explain improvements in performance, and a comprehensive investigation also requires analysis of changes in SV and $\dot{Q}$. A number of prior investigations have demonstrated a consistent attenuation of the increase in HR and the declines in SV and $\dot{Q}$ during exercise with pre-exercise P_{Vol} expansion, presumably via the Frank Starling mechanism (Coyle *et al.,* 1986; Fortney *et al.,* 1981a; Grant *et al.,* 1997; Hopper *et al.,* 1988; Kanstrup & Ekblom, 1982; Kanstrup *et al.,* 1992). Whilst the principle has been verified, the present study is the first to investigate the effect of P_{Vol} expansion on SV and $\dot{Q}$ during exercise induced by pre-exercise high sodium ingestion. Many of these prior studies have employed techniques which are not comparable to the present study and that have induced P_{Vol} expansion far greater in magnitude than the present (7-12 %; Coyle *et al.,* 1986; Fortney *et al.,* 1981a; Grant *et al.,* 1997; Hopper *et al.,* 1988; Kanstrup & Ekblom, 1982; Kanstrup *et al.,* 1992). To our knowledge, elevated SV and $\dot{Q}$ have not previously been reported following pre-exercise P_{Vol} expansion of less than 7 %. Thus the present study not only represents an advancement of previous investigation, but presents a more practical strategy to improve $\dot{Q}$ during high intensity exercise in the heat.

Under room temperature conditions the decline in SV can be compensated for by an increase in HR which maintains $\dot{Q}$ during exercise. However, when exercise is performed in the heat, the dual demands for blood flow between muscles and the periphery compete to the extent in which one, or the other, is compromised. Thus when exercising in the heat SV_{Drift} is more pronounced and $\dot{Q}$ can be impaired (Coyle & Montain, 1993; Rowell *et al.,* 1986). In the present study, when participants ingested just the low-sodium regular sports drink, before exercise $\dot{Q}$ fell during exercise, as the rate of SV_{Drift} exceeded the rate of $HR_{Drift.}$ However, when participants ingested the high sodium drink during rest, a small but significant increase in $\dot{Q}$ was observed at the end of steady state exercise. This increase in $\dot{Q}$ is significant in explaining the mechanism by which improvements in performance are possible. A greater $\dot{Q}$ increases BF_{Mus}, increasing oxygen available for energy provision.

There are now several studies, including one described in this thesis, that have investigated the effects of pre-exercise ingestion of a high sodium concentration (Sims *et al.*, 2007a; 2007b). However, none of these have sought to identify if any additional benefits can be derived from the ingestion of further fluid during exercise. In that respect they are not representative of athletic practice where it has become commonplace for athletes to ingest during exercise. In advancement of previous studies which have investigated pre-exercise high sodium ingestion, this study sought to examine whether further ingestion of fluid during exercise would produce further physiological gains. Participants ingested either no fluid or a volume of fluid equal to ~40 % of sweat loss during a preliminary test of a low-sodium regular sports drink. Ingested over the first 30 min of exercise this equated to an ingestion rate of ~1 $L \cdot h^{-1}$, any greater may have induced stomach discomfort and been detrimental to exercise performance. Fluid intake of less than this magnitude had previously been shown to have a positive impact on exercise performance when exercise began in a state of hydration (e.g. Montain & Coyle, 1992a).

Ingestion of a further 530-540 mL of low-sodium fluid during exercise had little or no effect on CV_{Drift} in either pre-exercise condition. In the low-sodium pre-exercise condition there was a slight but significant reduction in SV_{Drift} and a non-significant trend for a reduction in HR_{Drift} when fluid was ingested, but there were no significant differences in either HR or SV in the high-sodium pre-exercise condition. It is somewhat surprising that fluid intake produced no significant cardiovascular differences during exercise, given that the ingestion of water during exercise has often been demonstrated to attenuate or prevent progressive increases in HR, and progressive declines in SV, and $\dot{Q}$ (Gonzalez-Alonso *et al.*, 1995; 1997), and improve endurance performance (Below *et al.*, 1995; Fallowfield *et al.*, 1996; McConell *et al.*, 1997; Pitts *et al.*, 1944), compared with no fluid ingestion. However, not all studies demonstrate noticeable differences in cardiovascular functioning as a result of fluid intake during exercise. In a similar protocol to the present study, Ganio *et al.* (2006) reported no significant differences in cardiovascular, or performance variables at 1 h of exercise (Ganio *et al.*, 2006). Similarly, Wingo *et al.* (2005b) found no differences in HR, SV, $\dot{Q}$ or $\dot{V}O_2$, when participants replaced near 100 % of fluid losses (-2.5 and -0.3 %) during 45 min moderate intensity exercise in the heat (60 % $\dot{V}O_{2max}$; 35°C, 40 % RH). It is possible that fluid ingestion only provides an ergogenic benefit when exercise

duration is more prolonged, and sweat losses are more severe. The volume of fluid ingested, or rather the percentage of fluid replaced appears a critical factor to potential improvements in cardiovascular functioning and performance (Montain & Coyle, 1992a). If Wingo *et al.* (2005b) failed to demonstrate an ergogenic effect with 100 % BM replacement it is hardly surprising that the present study did not find an effect with just 40-50 % replacement, which produced only a partial attenuation of the decline in P_{Vol}. Furthermore, in the present study, participants started exercise in hyperhydrated state following pre-exercise ingestion. The failure to demonstrate a significant difference in CV_{Drift} is most likely a result of the pre-exercise drinking; with the capacity for cardiovascular differences diminished.

9.4.3. Thermoregulatory Responses

Pre-exercise ingestion of the high sodium drink and the subsequent P_{Vol} expansion did not affect thermoregulation during exercise. Neither sweat rates nor T_{Rec} were significantly different following pre-exercise ingestion of high compared with the low sodium drink. These findings confirm those of Chapter 8. A number of other studies investigating P_{Vol} expansion have also reported no effect on thermoregulation during moderate-high intensity exercise, conducted either in the heat or in a thermo-neutral environment (Fortney *et al.*, 1981a; 1981b; Grant *et al.*, 1997; Montain & Coyle, 1992b; Sawka *et al.*, 1983). Whilst the impaired thermoregulation with hypovolemia results from a decrease in BF_{Skn} (Fortney *et al.*, 1981a; 1981b), an increase in BF_{Skn} does not accompany the equivalent volume expansion (Fortney *et al.*, 1981a; 1981b). A possible explanation for the failure to report changes in thermoregulation with P_{Vol} expansion is that expansion is often in simultaneous occurrence with elevated P_{Osm}. Hyperosmolality has previously been shown to reduce BF_{Skn}, impairing sweating and lowering the capacity for heat dissipation (Takamata *et al.*, 2001; Nielsen, 1973). Thus any potential increase in sweating as a result of P_{Vol} expansion may have been compromised by hyperosmolality-induced reductions in sweat rate.

Previous studies investigating P_{Vol} expansion, have however, produced equivocal findings with others reporting an improvement in thermoregulation (Deschamps *et al.*, 1989; Fortney *et al.*, 1983; 1988; Nose *et al.*, 1990; Sims *et al.*, 2007a; 2007b). Of those that did report a lower T_{Core} or slower rate of rise in T_{Core}, most of studies induced P_{Vol} expansion which occurred either during, or at the onset of

exercise (Deschamps *et al.,* 1989; Fortney *et al.,* 1983; 1988; Nose *et al.,* 1990) and are as such not comparable to the present study's finding. However, following sodium loading induced P_{Vol} expansion, Sims *et al.* (2007a; 2007b) demonstrated a reduction in the rate of rise in T_{Rec} following pre-exercise P_{Vol} expansion with ingestion of a high vs. low sodium drink. Whilst, in the present study, the ingestion of the high sodium sports drink substantially elevated P_{Na} and P_{Osm} at the start of exercise, Sims *et al.,* (2007a; 2007b) employed a protracted drinking strategy which enabled elevated P_{Na} and P_{Osm} to return to baseline by the start of exercise in their studies. These findings suggest a significant role for osmolality in the impairment in thermoregulation during exercise in the heat, during the present study. The improved thermoregulation demonstrated by Sims *et al.* (2007a; 2007b) likely resulted from a slower absolute rise in P_{Osm} during exercise.

Whilst the additional ingestion of fluid had little effect on cardiovascular performance, it produced a non-significant trend for a reduction in T_{Core}. This can be attributed to the reduction of osmotic inhibitory input to the thermoregulatory system. Given the negative influence of P_{Osm} on sweat rates during exercise in the heat, the present study sought to investigate whether ingestion of a low-sodium sports drink during exercise might improve thermoregulation in a state of pre-exercise hyperosmolality. In the hyperosmotic state induced by high sodium ingestion, ingestion of low-sodium fluid produced a non-significant trend for reduction in the rate of rise in T_{Rec} and a increase in sweat rates' and a significant increase in sweat rates, which were concomitant with a reduced rise in both P_{Na} and P_{Osm}. In a state of hyperosmolality, induced by high sodium ingestion and confounded by fluid free exercise, P_{Na} and P_{Osm} rose at an accelerated rate during exercise compared to the other conditions. Under situations of high P_{Osm} the body initiates a series of mechanisms designed with curbing fluid losses such as antidiuresis and reduced sweat secretion. The trend for an increase in sweat rates with the continued ingestion of fluid during exercise, suggests a possible role for the combination of treatments in improving thermoregulation. It is plausible that a greater volume of fluid ingested, and hence greater decreases in P_{Osm}, may promote more marked improvements in thermoregulation.

9.4.4. Exercise Performance

In advancement of previous studies which employed time to exhaustion protocols (e.g. Sims *et al.*, 2007a; 2007b; Chapter 8), this study employed a time trial protocol to measure athletic performance. As athletes are rarely required to reach maximal effort during events, time to exhaustion protocols is not replicable to 'real-life' situation. Instead time trial protocols allow simulation of variable intensities (Palmer *et al.*, 1994) normally witnessed during competition. Accordingly, time trials achieve similar physiological responses to actual events when the same amount of work is performed at a set intensity (Currell & Jeukendrup, 2008). As such they have been shown to correlate strongly with actual performance during cycling (Russel *et al.*, 2004), whilst time to exhaustion protocols do not (Laursen *et al.*, 2002). Therefore the employment of time trials in this study improves the ecological validity of the findings.

Pre-exercise high-sodium ingestion significantly reduced the duration of time trial by 4.6 % and increased average power output by 8 %. The influence of P_{Vol} expansion on performance is equivocal, with previous studies reporting either no change (Kanstrup & Ekblom, 1982; Warburton *et al.*, 1999) or enhancements (Chapter 8; Berger *et al.*, 2006; Coyle *et al.*, 1986; Coyle *et al.*, 1990; Krip *et al.*, 1997; Greenleaf *et al.*, 1998; Sims *et al.*, 2007a; 2007b) in $\dot{V}O_{2max}$ and or performance.

P_{Vol} expansion of 4.5-5.1 % induced by pre-exercise ingestion of a high sodium drink has consistently induced an increase in exercise capacity (Chapter 8; Greenleaf *et al.*, 1998; Sims *et al.*, 2007a; 2007b). However, the mechanism by which it is achieved as yet remains unclear. Sims *et al.* (2007b) also reported that pre-exercise sodium ingestion increased exercise capacity, although in 6 out of 8 participants ethical restrictions on T_{Rec} prevented a definitive determination of exercise capacity. At termination of exercise they found that exercise duration was modestly related to the extent of attenuation in both T_{Rec} (r = 0.92) and HR (r = 0.85). In the present study, exercise capacity had no statistically significant relationship with T_{Rec}, and maximum T_{Rec} at exhaustion was just 39.1^{o}C. Furthermore, the additional ingestion of fluid during exercise, which reduced T_{Rec}, but did not significantly affect CV_{Drift} had no significant effect on exercise performance. Thus, it is unlikely a critical T_{Core} influenced performance here.

In chapter 8 the increase in exercise capacity, following pre-exercise sodium ingestion was attributed to a reduction in cardiovascular strain, mediated by the change

in pre-exercise P_{Vol}. The more comprehensive measurement of cardiovascular variables in the present study provides more rigorous evidence in support of the relationship between pre-exercise P_{Vol} expansion and exercise performance. Pre-exercise P_{Vol} induced significant attenuation $\dot{Q}_{Drift}$ during exercise, resulting in a greater potential of BF_{Mus} and O_2 delivery, extending the point at which participants are exercising at their maximum potential. In support of this conclusion, Ganio *et al.* (2006) demonstrated a reduction in the decline in performance of the $\dot{V}O_{2max}$ test when CV_{Drift} was attenuated by reducing the decline in P_{Vol} during exercise.

9.5. Conclusion

The pre-exercise ingestion of a high sodium drink was effective in increasing pre-exercise P_{Vol}, reducing CV_{Drift} and increasing subsequent exercise capacity, but did not affect thermoregulation, in comparison with a regular low-sodium sports drink. These findings confirm results of several previous studies that have utilised high sodium drinks as highly efficient and practical pre-exercise means to improve exercise performance in the heat. This is the first study to report an increase in SV and $\dot{Q}$ following high sodium intake, and to my knowledge the first to demonstrate these effects with P_{Vol} expansion of less than 7 %.

There is a potential for reduced T_{Core} with the additional ingestion of fluid during exercise. However, no further cardiovascular or exercise performance benefits were demonstrated on top of those induced by the pre-exercise sodium intake. Thus given that T_{Rec} was not a critical factor in determining exercise capacity in the present study, and the additional weight of ingested fluid, and possible gastrointestinal discomforts, under these experimental conditions, the additional replacement of 40 % fluid losses during exercise may not be necessary. Given that ingesting fluid is widely advocated during exercise, future research should explore whether the additional intake of fluid during exercise may be advantageous during different exercise conditions and/or with different volumes of fluid.

10. General Discussion and Conclusions

10.1. Main findings

Many athletic and occupational situations make it unfeasible to ingest 100 % of BM losses during exercise (Convertino *et al.*, 1996) due to issues with availability, and the circumstances of competition. For example, with access to drinks only avalible during stoppages in play, fluid intake during the 2022 FIFA World Cup in Qatar will likely become a critical issue. Avaliability is also an issue for those involved in industrial jobs in which high temperatures are frequent or for firefighters, who regularly surround themselves with critically high temperatures without fluid replacement for upwards of 45 minutes. In addition, athletes may *choose* not to ingest 100 % BM losses because of either perceived or actual discomfort as a result of consuming large volumes of fluid whilst exercising. Therefore in these situations, where it is impractical to replace 100 % of fluid losses, alternative strategies might be necessary to prepare athletes and workers for peak performance.

The findings of this thesis support the central hypothesis, demonstrating that highly concentrated sodium drinks are both efficient and acceptable means to improve hydration, reduce the cardiovascular stress during exercise in the heat, and improve exercise performance. However, whilst the ingestion of high sodium concentrations might be highly effective, caution should be made when athletes are allowed to drink *ad libitum* since the unpleasant taste evoked by these solutions persists until P_{Osm} and P_{Na} has returned to baseline, which may take at least two hours

10.2. Ergogenic benefit of high sodium drinks.

More specifically this thesis has addressed: a) fluid balance, b) cardiovascular functioning c) thermoregulatory and d) performance. In order to fully access its capabilities these parameters have been examined across the whole spectrum of sporting scenarios; before, during exercise and after exercise.

10.2.1. Fluid balance

The results presented in this thesis present strong evidence that the ingestion of a high sodium drink accelerates restoration of fluid balance and promotes an expansion of P_{Vol} with an ergogenic potential. During Chapters 8 & 9, the strong osmotic potential of sodium promoted significant and consistent increases in P_{Na} (4-6 mmol·L^{-1}) and P_{Osm} (4-5 mOsm·kg^{-1}) and during Chapter 4, P_{Na} remained approximately 4 mmol·L^{-1} higher during rehydration with the high sodium drink compared with rehydration with the low sodium drink.

Changes in P_{Na} and P_{Osm} have significant consequences for regulation of fluid balance. Urine production is controlled by the activation of the fluid-regulating hormone, AVP, in response to the elevated tonicity of ECF following profuse sweat loss. The release of AVP stimulates antidiuresis, reabsorbing fluid back into the bloodstream, increasing the concentration of urine released. A secondary response to AVP release is the increased sensations of thirst, designed to increase fluid intake. In the present studies the contemporaneous changes in U_{Osm}, U_{Vol} and ratings of thirst with changes in P_{Na} and P_{Osm} illustrate this relationship. The initial increase in both P_{Na} and P_{Osm} and the maintenance of elevated P_{Na} during exercise and recovery had significant effects on P_{Vol} and fluid balance.

Chapter 4 compared the rate of fluid restoration of a regular sports drink with a regular sports drink and a rehydration solution containing a sodium concentration of 136 mmol·L^{-1}; a concentration greater than any which had been employed in previous investigations. The negative fluid balance demonstrated with the ingestion of the low sodium concentration supports prior investigation; even when 120-150 % of fluid losses are replaced low-moderate concentrations of sodium (3-60 mmol·L^{-1}) frequently result in negative fluid balance after 3 h (Ismail *et al.,* 2007; Maughan *et al.,* 1995; Merson *et al.,* 2008; Saat *et al.,* 2002; Shirreffs *et al.,* 2007).

The high sodium drink significantly accelerated the rate of rehydration of both fluid balance and P_{Vol} compared with a regular sports drink, by reducing U_{Vol} by 59 %. The greater fluid retention in the present study confirms the inversely proportionate relationship between sodium content and diuresis, reported previously (Maughan & Leiper, 1995; Merson *et al.,* 2008; Mitchell *et al.,* 2000; Shirreffs & Maughan, 1998; Shirreffs *et al.,* 1996; Wemple *et al.,* 1997).

The high potential for fluid conservation of sodium was used to promote pre-exercise P_{Vol} expansion in Chapters 8 & 9. Pre-exercise ingestion of the high sodium concentration increased P_{Vol} by 4.5-5.1 % with the high sodium drinks, whereas ingestion of the low sodium drinks had little or no effect on P_{Vol}. This magnitude of change is commensurate with previous findings following the ingestion of the same volume of a high sodium drink (157-164 $mmol \cdot L^{-1}$) prior to exercise (Greenleaf *et al.*, 1997; 1998; Sims *et al.*, 2007a; 2007b).

As with previous studies (e.g. Sims *et al.*, 2007a; 2007b) the expansion of P_{Vol} is attributed to the reduction in pre-exercise U_{Vol} from 305-164 mL and from 331-160 mL in Chapter 8 & 9, respectively. This equates to an improvement in fluid retention from 57-60 % up to 78-79 %. Compared to those in the rehydration trials, these figures show a similar, if not greater level of retention. This seems paradoxical given that during the rehydration trials the body was in a state of fluid depletion; one would expect a greater fluid retention than when TBW is positive. However, it must be considered that the level of retention in the pre-exercise studies may be inflated, since at the point at which U_{Vol} voided was measured, not all fluid ingested would have passed through the digestive system. Therefore, we cannot be sure of the actual figures of fluid retention, but we can still acknowledge the large difference in fluid retention between the low and the high sodium drinks, which is the likely cause of P_{Vol} expansion.

Chapter 8 & 9 employed a high sodium drink as a pre-exercise method to promote P_{Vol} expansion. A more common method to induce pre-exercise hypervolemia is the intravenous infusion of saline or an artificial P_{Vol} expander such as dextran or albumin. However, infusions are both impracticable and prohibited in athletic events (WADA, 1999) and as such do not represent a likely occurrence in either an athletic or occupational scenario. The drinking of fluid prior to exercise is a strategy already common place in sport and represents a more efficient means to promote P_{Vol} expansion. An alternative drinking strategy involves hyperhydrating with either water or water and glycerol (e.g. Anderson *et al.*, 2001; Coutts *et al.*, 2002; Freund *et al.*, 1995). Whilst these drinks are a more practical solution than infusions, they do require a large volume of fluid to be ingested, far greater than with sodium. A similar level of hyperhydration can be reached with the ingestion of just ~750-800 mL of a high sodium solution (Chapter 8 & 9; Sims *et al.*, 2007a; 2007b), whereas hyperhydrating with plain water or glycerol requires the ingestion of >2 L (e.g. Anderson *et al.*, 2001; Coutts *et al.*, 2002; Freund *et al.*, 1995). It is doubtful that a large part of the athletic population

could easily tolerate the necessary volume of fluid to be taken before key competitions when stress and anxiety are at their peak. Thus the pre-exercise ingestion of a highly concentrated sodium solution represents a more pragmatic approach to preparation for competition.

10.2.2. Cardiovascular Responses

The studies in this thesis have provided strong evidence that, the ingestion of highly concentrated sodium drinks have attenuated CV_{Drift} during exercise in the heat. The studies in this thesis employed protocols which previous studies (Shaffrath & Adams, 1984; Wingo *et al.,* 2005b; Nassis & Geladas, 2002; Ekelund & Holmgren, 1964) have shown should cause considerable CV_{Drift}. In accordance with our expectation, considerable CV_{Drift} occurred in the control conditions. Chapter 7 investigated whether the ingestion of a high sodium drink would affect cardiovascular performance during 90 min exercise in the heat. Ingestion of the high vs. low sodium drink during the first 45 min of exercise attenuated the decline in HR_{Drift} (4 $b \cdot min^{-1}$) and SV_{Drift} (5 $mL \cdot b^{-1}$) but did not affect $\dot{Q}$ during the second 45 min of exercise. The failure to demonstrate significant differences in $\dot{Q}$ is likely to be a result of the intensity of exercise being insufficient to significantly strain the cardiovascular system. In Chapter 8, high sodium intake attenuated the HR drift by 9 $b \cdot min^{-1}$ compared to the low sodium drink. Similarly, in Chapter 9, pre-exercise ingestion of the high sodium concentration induced an 8-9 $b \cdot min^{-1}$ attenuation of HR_{Drift}, a 11-12 $mL \cdot b^{-1}$ attenuation of SV_{Drift}, and a small but significant increase in $\dot{Q}$ of 0.8 $L \cdot min^{-1}$ compared with the regular low sodium sports drink.

Much debate over the last few decades has concerned the causes of the decline in SV consistently witnessed during exercise in the heat. One theory proposes that the reduction in CBV, as a result of both the redistribution of blood flow to the periphery and the resulting sweat-induced fluid loss, will cause a decline in central venous pressure and EDV (Coyle & Montain, 1993; Rowell *et al.,* 1986) and consequently reduce SV. A more recent proposal suggests a direct result of the rise in T_{Core} on SV, via an increase in HR, and reduction in end-diastolic filling time (Fritsche *et al.,* 1999; Gonzalez-Alonso *et al.,* 2008). The findings presented in Chapters 7 & 9 support the former argument. Whilst high sodium ingestion was associated with a reduction in HR,

and an increase in SV and $\dot{Q}$, it had no significant effect on thermoregulatory responses during either study. The breakdown of a relationship between T_{Rec} and HR suggests the latter argument does not apply in this case. Instead the attenuated decline in SV in the high sodium trials appears a direct response to the pre-exercise increase in P_{Vol}, as evidenced by an elevated EDV. The HR response in these studies therefore appears a reaction to the changes in SV, not *vice versa*. Following blood volume expansion, Kanstrup *et al.* (1992), reported similar findings; SV was significantly elevated following blood volume expansion as a result of an increase in left ventricle EDV, but not left ventricle end systolic volume or ejection fraction. Thus the improvements in cardiovascular functioning witnesses in Chapter 7-9 appear a direct function of elevated P_{Vol}.

A number of prior investigations have demonstrated a consistent attenuation of the increase in HR and the declines in SV and $\dot{Q}$ during exercise with pre-exercise P_{Vol} expansion (Coyle *et al.*, 1986; Fortney *et al.*, 1981a; Grant *et al.*, 1997; Hopper *et al.*, 1988; Kanstrup & Ekblom, 1982; Kanstrup *et al.*, 1992). Whilst the principle has been verified, Chapter 9 is the first to investigate the effect of pre-exercise high sodium ingestion on SV and $\dot{Q}$ during exercise. Many of these previous studies have employed techniques which are not comparable to Chapter 9 and have induced P_{Vol} expansion far greater in magnitude (7-12 %; Coyle *et al.*, 1986; Fortney *et al.*, 1981a; Grant *et al.*, 1997; Hopper *et al.*, 1988; Kanstrup & Ekblom, 1982; Kanstrup *et al.*, 1992). To our knowledge, elevated SV and $\dot{Q}$ have not previously been reported following pre-exercise P_{Vol} expansion of less than 7 %. Thus this thesis represents an advancement of previous investigation, and delivers a more practical strategy to improve $\dot{Q}$ during high intensity exercise in the heat.

10.2.2.1. Modifying Factors

These findings support the consensus that fatigue from heat strain is a multifaceted, integrated phenomenon that is highly contingent on circumstances (Cheung & Sleivert, 2004; Cheuvront & Sawka, 2001; Ely *et al.*, 2009; Gonzalez-Alonso *et al.*, 2008; Nybo, 2008). Whilst these findings are impressive given the small magnitude of P_{Vol} expansion, it should be acknowledged that testing a different population or in a different environment may not yield similar findings.

Training Status

An untrained population was chosen as participants in chapters 7, 8, and 9; partly to aid participant recruitment, but predominantly because previous studies investigating high sodium drinks had tended to employ trained athletes (e.g. Sims *et al.,* 2007a; 2007b; Sanders *et al.,* 2001). Thus the findings in this study represent an advancement in the research. Previously, pre-exercise P_{Vol} has produced contrasting results in untrained vs. trained populations. For example Hopper *et al.* (1988) demonstrated that acute P_{Vol} expansion increased SV by 11 % during upright exercise in untrained males (45 $mL \cdot kg^{-1} \cdot min^{-1}$), but not in endurance trained males (62 $mL \cdot kg^{-1} \cdot min^{-1}$), whose P_{Vol} was 12 % above untrained men at rest. It is possible that the capacity to increase SV is limited and that in athletes that already have an expanded P_{Vol} may not benefit from acute P_{Vol} expansion to the same extent as untrained athletes. However, improvements in exercise performance have been demonstrated in endurance trained participants following high sodium intake. Sims *et al.* (2007a; 2007b) reported an increase in exercise capacity of 26-28 %. This suggests that high sodium ingestion is an effective means of promoting P_{Vol} expansion and improvements in exercise performance in both a trained and untrained population. Unfortunately Sims and colleagues (Sims *et al.,* 2007a; 2007b) only measured HR, and failed to measure SV or $\dot{Q}$, so it is difficult to decree that in their study the improvements in exercise performance were a direct result of improved cardiovascular functioning.

Environmental Conditions

The majority of studies investigating high sodium intake have used a warm environment (Chapter 7-9; Sims *et al.* 2007a; 2007b). It is likely that these conditions are characterised by a greater rate of sweat loss, and increased cardiovascular and thermoregulatory strain, and therefore creating a situation more likely to facilitate differences in these variables. Only once has the effect of ingesting high sodium concentrations during exercise been investigated (Sanders *et al.,* 2001). During prolonged exercise (4 h) in a thermo-neutral environment ($20^{o}C$), ingestion of 100 % of

fluid losses of a high (100 $mmol \cdot L^{-1}$) sodium solution attenuated the decline in P_{Vol} compared with low (5 $mmol \cdot L^{-1}$) and moderate (50 $mmol \cdot L^{-1}$) sodium solutions. Despite this, no differences in HR or T_{Core} were found. It is possible that the effects of P_{Vol} restoration might be more modest when exercise is performed in a thermo-neutral environment.

From studies using artificial P_{Vol} expansion techniques, it is known that the environmental conditions are critical to whether P_{Vol} evokes an improvement in cardiovascular efficiency. For example, Nose *et al.* (1990) demonstrated a 5 % decrease in HR_{Drift} whilst untrained participants exercised for 50 min at 60 % $\dot{V}O_{2max}$ at 30°C, but no change in HR_{Drift} when participants completed the same exercise protocol at 22°C. Further, whilst a decrease in HR is a common occurrence following P_{Vol} expansion in the heat (Fortney *et al.,* 1981a; Fortney *et al.,* 1983; Montain & Coyle, 1992b; Nose *et al.,* 1990; Sawka *et al.,* 1983), when exercise is performed in a thermo-neutral environment others have failed to report an ergogenic effect (Berger *et al.,* 2006; Hopper *et al.,* 1988; Watt *et al.,* 2000) and Kanstrup *et al.* (1992) actually reported a 12 $b \cdot min^{-1}$ increase in HR. In these studies exercise was of relatively short duration (5-16 min; Berger *et al.,* 2006; Hopper *et al.,* 1988) or at a low intensity (50 W; Kanstrup *et al.,* 1992). However, when exercise is prolonged (>2 h) a reduction in HR_{Drift} has been demonstrated in trained men (Roy *et al.,* 2000). These findings suggest that unless exercise is prolonged, when performed in a thermo-neutral environment the likelihood of P_{Vol} having an ergogenic effect in diminished.

10.2.3. Thermoregulatory Responses

The findings of this thesis suggest that the ingestion of a high sodium drink does not affect thermoregulation during exercise either when ingested during exercise or during pre-exercise rest period. Chapter 7 found no main effect for T_{Rec} for drink when a high vs. low sodium drink was ingested during exercise. Similarly when fluid was ingested pre-exercise (Chapter 8 & 9) there was no significant difference in T_{Rec} or sweat rates between the high and low sodium drinks, when served at room temperature (22°C). There was a trend for a lower T_{Rec} with high sodium compared with low sodium when drinks were served at 4°C, but at no point were the differences statistically significant. The failure to demonstrate changes in thermoregulation in this thesis could be attributable to the elevated P_{Osm} and P_{Na} with high vs. low sodium drinks.

Hyperosmolality has previously been shown to reduce skin blood flow, impairing sweating and lowering the capacity for heat dissipation (Nielsen, 1973). Thus any potential increase in sweating as a result of P_{Vol} expansion may have been compromised by hyperosmolality-induced reductions in sweat rate. Indeed, in contrast to our findings Sims and colleagues (2007a; 2007b) demonstrated a reduced rate of rise in T_{Rec} following the re-exercise ingestion of a high sodium drink. In that study, the retracted drinking strategy allowed P_{Osm} to return to baseline levels by the start of exercise.

Overall, the effects of P_{Vol} expansion on thermoregulation has produced equivocal findings with some demonstrating reduced T_{Core} (Deschamps *et al.,* 1989; Nose *et al.,* 1990; Sims *et al.,* 2007a; 2007b) and others reporting no effect (Fortney *et al,* 1981a; Grant *et al.,* 1997; Watt *et al.,* 2000). The failure to report differences in T_{Rec} despite significant differences in fluid balance, P_{Vol} and cardiovascular responses has been reported previously by others (*see section 2.4.1.2;* Fortney *et al.,* 1981a; Fortney *et al.,* 1983; Grant *et al.,* 1997; Leutkemeier *et al.,* 1994; Sawka *et al.,* 1983; Watt *et al.,* 2000). Fortney *et al.* (1981b) did report a significant reduction in T_{Esp} following pre-exercise hypervolemia, although only by 0.1^{o}C. Furthermore, when P_{Vol} expansion is attempted during exercise, Montain & Coyle (1992b) reported similar failure to demonstrate changes in T_{Rec}. They found that P_{Vol} expansion during exercise (2 h; 62-67 % $\dot{V}O_{2max}$; 33^{o}C, 50 % RH), which induced dehydration of (3-4 % BM loss) failed to prevent reductions in BF_{Skn} or to attenuate hyperthermia, even though it prevented hypovolemia and partially restored SV (-17 vs. -27 %). In the same study (Montain & Coyle, 1992b) replacement of 100 % BM losses (~2.4 L) with a 6 % CHO-E drink, however, increased BF_{Skn} and reduced T_{Esp} and T_{Rec} in respect of both no fluid and blood volume expansion conditions. These findings suggest that fluid replacement attenuates the rise in T_{Core} by some means other than blood volume expansion, i.e. hyperosmolality.

10.2.4. Performance

The findings of this thesis showed that pre-exercise ingestion of high-sodium drinks provides an efficient means to increase exercise capacity when exercise is performed in the heat. Chapter 8 demonstrated an increase in time to exhaustion of 15 % when drinks were served at room temperature with pre-exercise ingestion of a high vs. low sodium drink. Similarly, in Chapter 9 pre-exercise high-sodium ingestion

significantly reduced the duration of time trial by 4.6 % and increased average power output by 8 %. P_{Vol} expansion of 4.5-5.1 % induced by pre-exercise ingestion of a high sodium drink has consistently induced an increase in exercise capacity (Chapter 8 & 9; Greenleaf *et al.,* 1998; Sims *et al.,* 2007a; 2007b).

In contrast, in studies using infusion of saline or an artificial P_{Vol} expander, P_{Vol} expansion has produced equivocal findings, with previous studies reporting either no change (Kanstrup & Ekblom, 1982; Warburton *et al.,* 1999) or enhancements (Chapter 8 & 9; Berger *et al.,* 2006; Coyle *et al.,* 1986; Coyle *et al.,* 1990; Krip *et al.,* 1997; Greenleaf *et al.,* 1998; Sims *et al.,* 2007a; 2007b) in $\dot{V}O_{2max}$ and/or performance. The reason why the high sodium studies have been consistent and the reason for the conflicting results with other techniques might be explained by two factors; the magnitude of the P_{Vol} expansion and the training status of the participants (Berger *et al.,* 2006; Coyle *et al.,* 1986; Coyle *et al.,* 1990; Krip *et al.,* 1997; Warburton *et al.,* 1999). Trained athletes already possess an inflated P_{Vol} in respect of normal levels (Convertino, 1991; Coyle *et al.,* 1990), and therefore experience a reduced increase in SV with P_{Vol} expansion, making them more likely to experience a reduction in $\dot{V}O_{2max}$, as a result of hemodilution, if P_{Vol} undergoes substantial "additional" expansion using artificial means (Coyle *et al.,* 1990; Hopper *et al.,* 1988; Warburton *et al.,* 1999). For example, Coyle *et al.* (1986) observed that a 13 % expansion of P_{Vol} in endurance-trained participants failed to increase SV during sub-maximal exercise and actually resulted in a non-significant decline in $\dot{V}O_{2max}$. In the same study, the same volume of expansion in untrained participants induced a 12 % increase in SV and a 3 % increase in $\dot{V}O_{2max}$. Clearly the effects of P_{Vol} expansion on $\dot{V}O_{2max}$ and exercise capacity will depend on the extent to which SV increases. It appears logical that untrained participants, such as those used in the present study, would stand to benefit more from larger P_{Vol} expansion than would better trained participants in whom the P_{Vol} is already elevated by approximately 200 mL (Convertino, 1991; Coyle *et al.,* 1990).

Secondly, it is possible that a particularly large reduction in Hb caused by a substantial expansion of the P_{Vol} greater than 14 % might exceed the potential for compensatory alterations in SV and $\dot{Q}$ to maintain muscle O_2 delivery, such that $\dot{V}O_{2max}$ is reduced. For example, Coyle *et al.* (1990) observed in untrained men that 7 % of P_{Vol} expansion increased SV by 10-15 %, and only a small (4 %) amount of

hemodilution, and as a result, $\dot{V}O_{2max}$ and performance during high intensity running were slightly improved. However, a 17 % expansion of P_{Vol} expansion resulted in an excessive hemodilution (11 %), but no further increase in SV, resulting in a subsequent decline in $\dot{V}O_{2max}$ and performance to normal levels. Clearly, the potential for P_{Vol} expansion to enhance P_{Vol} depends on striking a delicate balance between the potentially negative effects of a hemodilution and the potentially positive effects of an increased SV. There appears to be an optimal P_{Vol} expansion level for eliciting improvements in exercise performance. Previous studies indicate that the infusion of fluid volumes of 4-14 % might positively impact on $\dot{V}O_{2max}$ and exercise performance in untrained participants (Berger *et al.*, 2006; Coyle *et al.*, 1986; Coyle *et al.*, 1990; Krip *et al.*, 1997; Greenleaf *et al.*, 1998; Sims *et al.*, 2007a; 2007b), whereas P_{Vol} expansion greater than volumes of greater than 14 % might have a detrimental effect (Coyle *et al.*, 1990; Kanstrup & Ekblom, 1982).

Whilst P_{Vol} expansion with high sodium ingestion has consistently improved exercise performance, the mechanism by which it is achieved as yet remains unclear. Sims *et al.* (2007b) also reported that pre-exercise sodium ingestion increased exercise capacity, although ethical restrictions on T_{Rec} prevented a definitive determination of exercise capacity. In Chapters 8-9, exercise capacity had no significant relationship with T_{Rec}, and mean T_{Rec} at exhaustion was just 38.7-39.1^{o}C. Furthermore, the additional ingestion of fluid during exercise in Chapter 9, which reduced T_{Rec}, but did not significantly affect $CV_{Drift,}$ and had no significant effect on exercise performance. Thus it is unlikely that critical T_{Core} influenced performance here.

In Chapter 8, the increase in exercise capacity was attributed to a reduction in cardiovascular strain, mediated by the change in pre-exercise P_{Vol}. The more comprehensive measurement of cardiovascular variables in Chapter 9 provides more rigorous evidence in support of the relationship between pre-exercise P_{Vol} expansion and exercise performance. Pre-exercise P_{Vol} induced significant attenuation $\dot{Q}_{Drift}$ during exercise, resulting in a greater potential of BF_{Mus} and O_2 delivery, extending the point at which participants are exercising at their maximum potential.

10.3. Other important findings

In addition to the main aims of this thesis, during the course of the chapters other hypotheses were tested, which will be discussed in the next section.

10.3.1. Pre-cooling with cold fluid ingestion

In Chapter 8, the pre-exercise ingestion of high sodium fluid was combined with a cold fluid ingestion in order to test the individual and combined effects of both pre-cooling and P_{Vol} expansion. Both interventions, when employed in isolation resulted in significant and equal reductions in HR_{Drift}

With cold fluid ingestion the reduction in mean HR appears in response to the reduction in T_{Rec}. The present study demonstrated an attenuation of HR_{Drift} and a reduction in mean HR with both cold drinks, compared with the control. The lower HR with PC during exercise persisted for just 25 min. Concomitant with the temporary reduction in HR, differences in T_{Rec} between the cold drinks and control lasted for just 20 min. This pattern of responses is reflective of the strong relationship found between mean HR and mean T_{Rec} during exercise in the present study (r = 0.87). The increase in HR with increasing T_{Core} is a proposed consequence of increasing autonomic nervous system activity, in response to elevated blood temperature detected by the sinoatrial node (Johnson & Proppe, 1996). Evidence of a reduced cardiovascular strain was the reduced RPE during exercise with both PC and PC-HS compared with control. In contrast the reduction in mean HR with highs sodium intake appears a consequence of increased pre-exercise P_{Vol}. Since there was no difference in T_{Rec} between high and low-sodium conditions, the reduction in mean HR must have resulted from some other means, such as increased P_{Vol}. Interestingly the additive HR response following the combined pre-exercise ingestion of both a high sodium and cold drink must have resulted from both a thermoregulatory and a P_{Vol} based effect.

Both cold and high sodium drink interventions resulted in an increase in exercise capacity of 13 and 15 %, respectively. Since, termination of exercise occurred at a mean T_{Rec} of just 38.7^{o}C, it is unlikely that exercise capacity was determined by a thermoregulatory mechanism, but rather as a result of the reduction in HR and potential greater capacity for cardiovascular work. Interestingly, when combined, the interventions had an additive effect, increasing exercise capacity by 24 % compared

with the control. The additive ergogenic benefit found with PC-HS in Chapter 8, show that drink cooling would be a useful addition to pre-exercise high sodium drinking strategy.

10.3.2. Additional drinking during exercise

There are now several studies, including Chapter 8, that have investigated the effects of pre-exercise ingestion of a high sodium concentration (Greenleaf *et al.*, 1998; Sims *et al.*, 2007a; 2007b). However, none of these have sought to identify if any additional benefits can be derived from the ingestion of further fluid during exercise. To improve the ecological validity of the study and provide a strategy more representative of athletic practice, in Chapter 9, participants who had previously hyperhydrated with either low or high sodium drink ingested either no fluid or an additional ~530 mL during the first half of 60 min of exercise.

There was a trend for a reduced T_{Core} and increased sweat rtes with the addition of fluid during exercise. Whilst these findings suggest a possible thermoregulatory benefit of continued ingestion of fluid during exercise, since T_{Rec} was not a critical factor affecting exercise tolerance in any of Chapters 7-9, thus the potential overall gain from this practice, under these conditions, is limited.

The ingestion of fluid during exercise has produced equivocal findings with some reporting reduced cardiovascular strain (Gonzalez-Alonso *et al.*, 1995; 1997), and improved endurance performance (Below *et al.*, 1995; Fallowfield *et al.*, 1996; McConell *et al.*, 1997; Pitts *et al.*, 1944), but with others demonstrating no difference in cardiovascular variables or exercise performance (Ganio *et al.*, 2006; Wingo *et al.*, 2005b). It is possible that fluid ingestion only provides an ergogenic benefit when exercise duration is more prolonged, and/ or sweat losses are more severe. Additionally the volume of fluid ingested, or rather the percentage of fluid replaced also appears a critical factor to potential improvements in cardiovascular functioning and performance (Montain & Coyle, 1992a). Furthermore, in the present study, participants started exercise in hyperhydrated state following pre-exercise ingestion, possibly diminishing the potential for ergogenic cardiovascular responses.

10.4. Drinking behaviour following exercise-induced dehydration

Whilst the ingestion of high sodium concentrations might be highly effective, caution should be made when athletes are allowed to drink *ad libitum* since the unpleasant taste evoked by these solutions persists until P_{Osm} and P_{Na} has returned to baseline, which may take at least of two hours. This thesis found no evidence to support the findings of Lesham *et al.* (1999), which showed an increase in preference for salty foods immediately after exercise. These findings do support those of Takamata *et al.* (1994) who also reported an increase in palatability of salt was delayed until P_{Osm} returned to baseline. What is interesting is that the current thesis tested a scenario more unaccustomed to athletes; whilst Takamata *et al.* (1994) tested sodium solutions in water, the current study examined drinks which were adapted from commercially available products. Whilst Takamata *et al.* (1994) experimentally manipulated fluid balance by having participants ingest hypotonic fluid during prolonged walking exercise; the current thesis presented an exercise routine similar to a routine athletic training session. Thus, these findings provide more ecologically valid support for those first presented by Takamata and colleagues (Tkamata *et al.*, 1994).

The primary focus of Chapter 5 & 6 was to determine the impact of hydration status on drink palatability and the perceptions of saltiness throughout the process of rehydration. Previous research had provided evidence that the taste system is a transitory system which responds to disturbances to homeostasis such as hunger (Yeomans, 1998), hyponatraemia (Beauchamp *et al.*, 1990; Huang & Yan, 2008) and dehydration (Appleton, 2005; Takamata *et al.*, 1994) by increasing the palatability of the food/drink specifically related to each disturbance. Based on the theory of *physiological requirement*, whereby 'the food/fluid which satisfies the requirement to the greatest extent will be perceived most palatable' (Cabanac, 1971) it was predicted that exercise-induced dehydration would have a significant impact on taste perceptions for salty drinks. Given that the salt content of the drinks used in this investigation was the primary factor which may contribute to an *unpleasant* taste, it was considered important to investigate whether our pre-conceived attitude to salty drinks, based entirely on hydrated experiences may be changed in a state of dehydration.

The initial study (Chapter 5) demonstrated that exercise-induced dehydration is capable of inducing significant changes in the palatability. Dehydration, equivalent to 3.3 % BM loss induced an increase in palatability of the flavoured water, and a decrease

in palatability of a flavoured high sodium sports drink. Furthermore, dehydration increased the relative importance of drink strength as a predictor of palatability. These findings support the relationship between physiological requirement and palatability; in the physiological situation of a requirement to restore fluid balance, only the fluid which satisfied the need to the greatest extent increased in palatability. The fluid which hindered the restoration of fluid balance was rated less palatable.

In a similar investigation by Appleton (2005), in which exercise induced a change in palatability of various drinks, osmolality was predicted to be the determining factor. Theoretically a drink with a low osmolality, e.g. water, would be more acceptable than before exercise, since immediately following exercise there is a specific need to reduce P_{Osm} within the extracellular fluid compartment. Equally a high osmolality drink, such as the high sodium drink used in this study, which would hinder the restoration of P_{Osm} to baseline levels, would be less acceptable. This study provided a more scientific support for the theory of physiological requirement than that of Appleton (2005), which was plagued by lack of standardisation and controls; this study also failed to measure P_{Osm}, or P_{Na}. Whilst it is well acknowledged that dehydration induced by sweat loss is accompanied by an increase in P_{Osm}, measurements of this variable are not always consistent with the level of BM loss. The next study (Chapter 6) was undertaken in an effort to examine the problem with a more systematic approach, and to provide a more extensive look at the whole rehydration period.

Chapter 6 confirmed the findings of Chapter 5, demonstrating a significant change in the palatability of three solutions following 1 h cycling in the heat. Cyclists completed two trials, and their combined BM decreased by 1.7 ± 0.5 kg. In addition to the measures made in Chapter 5, the study observed a decrease in P_{Vol} (-4.4 ± 1.3 %), and an increase in U_{Osm} (444 ± 123 $mOsm \cdot kg^{-1}$), P_{Na} (3.6 ± 0.5 $mmol \cdot L^{-1}$) and ratings of thirst (44 ± 9 mm). Accompanying these responses there was an increase in palatability of water and to a lesser extent the low sodium drink and a decrease in the palatability of the high sodium drink. These findings support the conclusions drawn in the previous study and provide more scientific evidence of the relationship between P_{Osm} (P_{Na}) and the change in taste perceptions for drinks of different sodium contents and osmolalities.

This study also sought to investigate how these taste perceptions respond during recovery from exercise. Recovery was manipulated by rehydrating with either one of two solutions. Firstly, acting as a control, participants ingested a low sodium regular

sports drink akin to those commercially available, and secondly participants ingested a high sodium concentration. Measurements and ratings of taste were then made at regular intervals for a three hour period. During recovery, the changes in ratings of palatability for water and both low and high sodium solutions were strongly associated with the level of hydration, more specifically with P_{Na}. Synonymous with antidiuresis and an increase in thirst, ratings of palatability for water substantially increased, and ratings of palatability for a high-sodium drink significantly decreased. These findings support the notion that the changes in taste perception appear to act as a physiological regulator, and moreover, this regulation prioritises restoration of hyperosmolality over hypovolemia. Our findings support previous investigation in which an increase in palatability for sodium was delayed until P_{Na} and P_{Osm} normalised (Takamata *et al.*, 1997). Another major finding of this study is the demonstration of an increase in palatability of the high-sodium drink 3 h into recovery with ingestion of 120 % BM losses of a low sodium rehydration solution. These adaptations were strongly correlated with changes in P_{Na} and suggest a physiological regulation of body fluid balance. The changes in palatability appear to be selective to the restoration of hyperosmolality before hypovolemia.

10.5 Other Considerations

Both epidemiological and experimental studies have established the strong positive relationship between sodium intake and blood pressure, and cardiovascular morbidity and mortality (Alderman, 2000; He *et al.*, 2000). Participants taking part in the studies in this thesis would have ingested up to 5.6 $g \cdot L^{-1}$ of sodium, far in excess of the recommended intake of sodium in both the UK and USA, of 2.4 $g \cdot d^{-1}$. It should be noted that the rate of sodium loss, during high intensity exercise in the heat greatly exceeds that of general life for which these recommendations are aimed. The ingestion of large volumes of sodium, in excess of the daily requirement is not a recommendation of this thesis. The use of high sodium drinks should be used as a one-off strategy to improve rehydration and/or performance, and not as a sports drink suitable for everyday consumption or training aid. The unpleasant taste associated with high sodium drinks both before and after exercise, demonstrated in Chapters 5 & 6, make it unlikely that these drinks will be easily adopted by as a regular routine, even in the most enthusiastic athletes. There is as yet no evidence to suggest that exceeding the daily recommended

consumption of sodium on one-off occasions has significant long term implications. Indeed the body has evolved to be particularly efficient at excreting unnecessary sodium on a daily basis (Hall, 1986). That said, caution would certainly have to be applied when advocating the use of high sodium drinks in populations such as older subjects or hypertensive patients where the effect of a high salt diet on rates of stroke, coronary heart, or coronary vascular disease appears to be more substantial (Alderman, 2000).

10.6. Methodological Issues

10.6.1. Performance tests

A number of recent studies measuring the effectiveness of rehydration have employed a subsequent performance test to assess rehydration in real terms (e.g. Merson *et al.*, 2008; Mitchell *et al.*, 2000). It is unfortunate that in Chapter 4 no measurement of performance was made; however, this failure does not detract from the findings of this study. No previous studies measured the effects of ingesting such a high concentration of sodium, let alone demonstrating it as such an effective means of restoring TBW. A study in which a performance measure was perhaps more important was Chapter 7. Ratings of perceived exertion and $\dot{V}O_2$ provided indicators of performance, yet they are not true measures of performance. This study would certainly have benefitted from having a performance test such as those used in Chapters 8 and 9.

Of those chapters which did employ a performance measure, Chapter 8 used a TTE test which requires a participant to exercise to exhaustion at a constant intensity, expressed as $\%\dot{V}O_{2max}$. Whilst this is the most common method of performance test (Hopkins *et al.*, 2000) TTE protocols are not replicable to 'real-life' situation; rarely are athletes required to reach maximal effort during events. TT protocols, which require a participant to complete a set amount of work or a set distance as quickly as possible, allow simulation of variable intensities (Palmer *et al.*, 1994) normally witnessed during athletic events. Accordingly, TT's achieve similar physiological responses to actual events when the same amount of work is performed at a set intensity (Currell & Jeukendrup, 2008). As such TT's have been shown to correlate strongly with actual performance during running (Palmer *et al.*, 1996) and cycling (Russel *et al.*, 2004), whilst (Laursen *et al.*, 2002) found no relationship between TTE. The TTE test was

initially used in study 8 to replicate those tests employed in previous literature (e.g. Sims *et al.,* 2007a; 2007b). Nevertheless, it is acknowledged that TT's make for a stronger experimental paradigm. In order to improve the ecological validity, Chapter 9 employed a time trial protocol.

In addition to increasing the ecological validity of the tests, TT's also improve the reliability of measurements; typically TT's have a coefficient of variation [COV] < 5 % whilst TTE's COV often exceeds > 20 % and can rise to as much as 50 % (Currell & Jeukendrup, 2008). It should be noted that the variation in TTE times is reduced when exercise is performed at a high intensity (Currell & Jeukendrup, 2008) such as with the present study. To improve the reliability of this thesis a number of steps were taken to control all factors during the performance trials. Firstly, both Chapter 8 & 9 used a performance test subsequent to an earlier bout of exercise. This is in contrast to previous studies testing high sodium drinks (Sims *et al.,* 2007a; 2007b). Taking physiological measures during performance trials may interfere with the performance of the participant, with the possible mechanism linked to concentration (Jeukendrup *et al.,* 1996). However, a steady-state preload can be added to a protocol to measure physiological variables without affecting performance, such as that used by Jentjens & Jeukendrup (2003). Using this method did not prevent this thesis from answering the mechanistic questions with physiological measures. Secondly, a familiarisation session was used to reduce the likelihood of a learning effect (Currell & Jeukendrup, 2008). When the trials have been included in a reliability analysis the coefficient of variation between the first two trials is 1.3-fold greater than between following trials (Hopkins *et al.,* 2001). This may be particularly important in untrained athletes such as those tested in this thesis. Furthermore the COV is increased when performing a TTE trial compared with a TT (Currell & Jeukendrup, 2008) making familiarisation trials particularly pertinent. Thirdly, no feedback, in terms of encouragement of performance cues were given to the participant either during or at the end of the trials

10.6.2. Selection of exercise intensity

In each study, the intensity of exercise for experimental trials was determined by completing an incremental maximal cycle exercise test. Chapters 4-7 employed the common method of assigning exercise intensities as a percentage of $\dot{V}O_{2max}$. The selection of exercise intensity from an individual's $\%\dot{V}O_{2max}$ may be flawed as there are

inter-individual differences with the ventilatory parameters. For example lactate threshold occurs at a range between 60-80 % $\dot{V}O_{2max}$; working at 65 % $\dot{V}O_{2max}$, some participants may be below lactate threshold whilst others above. Working at 85 % $\dot{V}O_{2max}$ some may be below critical power (maximum lactate steady state), whilst others above. In an advancement of methods, Chapters 8 and 9 employed exercise intensities as a percentage of the difference between GET and $\dot{V}O_{2max}$; a method more likely to normalize a given intensity than the use of a percentage of $\dot{V}O_{2max}$ alone (Whipp & Rossiter, 2005). GET, synonymous with ventilation or lactate threshold, separates moderate and heavy domains. Such exercise settings enables participants own landmarks to be set at a corresponding wattage on any loaded cycle and therefore acted to improve the validity of these studies.

10.6.3. Measurement of body temperature

Measurement of T_{Rec} is the most commonly used measurement of temperature during thermoregulatory investigations (Sawka & Wenger, 1988) and has gained wide acceptance due to its relative ease of use, its stability during steady-state conditions (Lee *et al.,* 2000) and its independence from environmental temperature (Gerbrandy *et al.,* 1954). However, the slow response time of T_{Rec} is well documented (Byrne & Lim, 2007; Eichna *et al.,* 1951; Kolka *et al.,* 1993; Rowell, 1986) and inconsistency in T_{Rec} readings can also result from changes in leg blood flow (Saltin & Hermansen, 1966) or from hot and cold areas along the rectum (Folk *et al.,* 1998).

However, whilst other sites for measuring T_{Core}, such as the oesophagus, tympanic artery and the gastrointestinal tract present their own challenges. The oesophagus is the physiologically preferred site (Sawka & Wenger, 1988) because of the deep body location, the close proximity to the left ventricle (Rowell, 1986), aorta (Cooper & Kenyon, 1957) and direct blood flow to the central thermoreceptors in the hypothalamus (Gerbrandy *et al.,* 1954), and its rapid response to changes in heat storage (Gerbrandy *et al.,* 1954). However, its measurement is a highly invasive procedure with difficulty of insertion of the thermistor (vomiting), irritation to nasal passages and/or throat, and general participant discomfort (El-Radhi & Barry, 2006; Sawka & Wenger, 1988; Stitt *et al.,* 1993). Among the non-invasive sites, tympanic temperature [T_{Tymp}] probably has the strongest association with T_{Core} (Lim *et al.,* 2008). However, the

accuracy of T_{Tymp} measurement is highly dependent on the skill of the technician (Cattaneo *et al.,* 2000; Farnell *et al.,* 2005) and readings can be influenced by ambient temperature (Nadel & Horvath, 1970). The biggest disadvantage, and the reason it could not be used in this thesis is that T_{Tymp} cannot be measured continuously. A relatively new technique for the estimation of T_{Core} is the measurement of intestinal temperature. Whilst the telemetric pill avoids the same participant discomfort felt with T_{Rec}, the telemetric sensor is expensive in comparison with measurement of T_{Rec}; and temperature can be influenced by fluid intake, making it inappropriate for the studies in this thesis. Thus whilst measurement of T_{Core} from the rectum may not be the most accurate measure, it was the most appropriate for this thesis.

A number of conclusions were drawn from these studies regarding the effect on thermoregulation. It was to the detriment of this study that T_{Skn} was not measured. With a number of invasive measures it was deemed an acceptable omission to ensure participant comfort, increasing compliance.

10.6.4. Measurement of hydration

Whilst isotope dilution techniques continue to need 3-5 h for internal isotope equilibration and analysis (Armstrong, 2007), there currently is no general consensus of a 'gold standard' assessment of hydration status during exercise (Cheuvront & Sawka, 2005; Institute of Medicine, 2005; Kavouras, 2002). Several methods o measure hydration were employed in the studies of this thesis. These ranged in their applicability due to methodological limitations such as simplicity, reliability, sensitivity and accuracy. These include BM, U_{Osm}, P_{Vol}, P_{Na} and P_{Osm}.

Other than isotope dilution analysis, the measurement of P_{Osm} is generally considered to provide the most valid and precise assessment of body hydration status (Institute of Medicine, 2005). This thesis employed the most common method of determining P_{Osm}; the freezing point depression technique. Analyses of P_{Osm} using a freezing point depression osmometer provide excellent measurement accuracy (Armstrong, 2007). Alternative techniques for measuring P_{Osm}, such as the use of vapour pressure osmometry, have more consistently lead to erroneous results (Armstrong, 2007). The measurement of P_{Osm} was only available for Chapters 8 and 9. In its place, P_{Na} provided an alternative tool, since changes in osmolality are primarily a factor of changes in sodium concentration (Costill, 1977). It should be noted, however,

that the relationship between hydration and sodium concentration is more variable than the relationship between hydration and osmolality (Bartok *et al.*, 2004; Senay, 1979).

Decreases in P_{Vol} occur proportionately with dehydration (Cheuvront & Sawka, 2005), making it an obvious method for assessing hydration status. Direct measurement of P_{Vol} is made by administering Evans blue dye (e.g. Hopper *et al.* 1988; Fortney *et al.*, 1981a). However, the Evans blue dye is an expensive and invasive method and is largely unreliable at detecting small changes in P_{Vol}. An alternative method, common to many investigations, including those undertaken as part of this thesis, involves the estimation of changes in P_{Vol} from changes in Hct and Hb (Dill & Costill, 1974).

Body mass, the most commonly used method to represent acute changes of body water (Kavouras, 2002) is by far the most simplistic method of assessing hydration status. This method also makes the incorrect assumption that sweat loss is the only source of fluid loss from the body. This assumption leads to erroneous assessment of hydration; with other sources of fluid loss including respiratory water loss or substrate utilisation (Maughan *et al.*, 2007; Mitchell *et al.*, 1972). When sweat rates are high, such as during exercise in the heat, these factors are usually discounted as being negligible (Maughan *et al.*, 2007). Thus if proper controls are made, BM changes can provide a sensitive estimate of acute TBW changes to access hydration changes during exercise. Urine concentration (specific gravity or osmolality) is a relatively inexpensive, easy to use and reliable assessment technique (Bartok *et al.*, 2004; Shirreffs & Maughan, 1998). However, urine concentration is affected by fluid intake; large volumes of dilute fluid induce a large U_{Vol} before euhydration is achieved (Shirreffs *et al.*, 1996). When used in isolation, measurement of urine concentration has its limitations, but used together with other measures can provide valuable insight.

10.7. Future Directions

By the very nature of this thesis, it should be viewed as a starting point. So rarely have high sodium concentrations been used in sports drinks that there is little research to date in the areas discussed in this thesis. The studies offer a broad range of scenarios where a high sodium drink can be effective but further research is essential to decide the full capabilities of the drink.

10.7.1. Rehydration from exercise

A number of recent studies measuring the effectiveness of rehydration have employed a subsequent performance test to assess rehydration in real terms (e.g. Merson *et al.,* 2008; Mitchell *et al.,* 2000). With the ergogenic benefit from pre-exercise ingestion of a high sodium drink, found in this thesis, it would be logical to extend on the work of Chapter 4 which demonstrated rapid improvements in rehydration and employing a measure of exercise performance. This would both improve the ecological validity of the intervention and provide a more comprehensive evaluation of the potential effectiveness of high sodium ingestion.

10.7.2. Pre-exercise ingestion of a high-sodium drink

All of the studies examining pre-exercise high sodium ingestion (Chapter 8-9; Sims *et al.,* 2007a; 2007b) have tested the effects in a warm environment. The environmental temperature appears critical for determining whether P_{Vol} expansion is effective in reducing cardiovascular strain during exercise, when P_{Vol} expansion is induced by artificial infusions (Nose *et al.,* 1990). Thus a logical next step would be to measure the effects of pre-exercise high sodium ingestion in a thermo-neutral environment.

The additive ergogenic benefit found with PC-HS in Chapter 8, show that drink cooling would be a useful addition to pre-exercise high sodium drinking strategy. Future research is needed to examine the possible extent of this additional benefit. In addition to sodium other important nutrients such as carbohydrates could be combined with drink cooling to enhance exercise performance.

Chapters 8-9, employed a 45 min drinking strategy lasting right up until the point of exercise. In contrast, Sims *et al.* (2007a; 2007b) employed a protracted pre-exercise drinking strategy where drinking commenced 105 and 80 min before exercise and ceased 45 and 20 min before exercise. This length of drinking strategy is unfeasible in many athletic and occupational situations and should be shortened to increase the viability of the intervention. However, this method enabled elevated P_{Na} and P_{Osm} to return to baseline by the start of exercise, which may have been a critical factor and a reason why an improvement in thermoregulation was demonstrated with participants in the study of Sims & colleagues (Sims *et al.,* 2007a; 2007b) but not the studies in this

thesis (Chapter 8-9). Future research should examine the optimal timing of intake for hyperhydration purposes.

Chapter 9 investigated the effects of additional ingestion of fluid during exercise in participants following pre-exercise ingestion of a high-sodium drink. The addition of fluid during exercise had a positive effect on thermoregulatory responses; with sweat rates significantly greater and T_{Rec} significantly reduced, but did not affect cardiovascular responses or exercise performance. Studies investigating possible ergogenic benefit of fluid ingestion during exercise have produced equivocal findings, particularly when the length of exercise protocol is short (< 1 h; Ganio *et al.*, 2006; Wingo *et al.*, 2005b). Additionally the volume of fluid ingested, or rather the percentage of fluid replaced also appears a critical factor to potential improvements in cardiovascular functioning and performance (Montain & Coyle, 1992a). In this light, further research is advocated to establish whether under different exercise conditions, or with different fluid volumes, the additional intake of fluid would enhance cardiovascular performance. This is particularly relevant since drinking during exercise is common practice amongst athletes and non-athletes alike.

10.8. Overall Summary & Conclusions

This thesis has addressed two key areas; firstly the ergogenic benefit of high sodium drinks, and secondly the effects of ingesting highly concentrated sodium drinks on palatability, before and after exercise.. It has shown that the inclusion of high sodium concentrations within sports drinks can be both an efficient and acceptable means to improve hydration, reduce the cardiovascular stress of exercise in the heat, and improve exercise performance. However, whilst the ingestion of high sodium concentrations might be highly effective, caution should be made when athletes are allowed to drink *ad libitum* since the unpleasant taste evoked by these solutions persists until P_{Osm} and P_{Na} has returned to baseline, which may take upwards of two hours

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Sodium ingestion and exercise performance

Current Health Status Questionnaire for male up to 45 years of age and females up to age 55 years

	Yes	No
Have you suffered from a viral illness in the last two weeks?		
Have you eaten within the last hour?		
Have you consumed alcohol within the last 24 hours?		
Have you performed exhaustive exercise within the last 48 hours?		
Do you have a cold, sore throat or sinus infection?		
Is there anything to your knowledge that may prevent you from successfully completing the tests that have been outlined for you?		

I have completed the questionnaire to the best of my knowledge and any questions that I have raised have been answered to my full satisfaction.

Signed: ……

Date: ……

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SSHS General Health Questionnaire

Name: ……
Date of Birth:……
Male/Female (please circle)
Height:……Weight:..……
Address:……..
……..
……
Phone No……..

Please Read the following carefully and answer as accurately as possible. The questions are designed solely to determine whether the proposed exercise is appropriate to you. Your answers will be treated strictly as confidential. If you have any doubts of difficulties with any of the questions please contact the person responsible for the study.

		Yes	No
1.	Have you seen a doctor in the last 6 months?	……	……
2.	Are you currently taking any prescriptive medications?	……	……
3.	Has a doctor ever said you have heart trouble?	……	……
4.	Do you suffer frequently from chest pains?	……	……
5.	Do you often feel faint or have spells of dizziness?	……	……
6.	Has a doctor ever said you have epilepsy?	……	……
7.	Has your doctor ever said you have kidney abnormalities?	……	……
8.	Has a doctor ever said you have high blood pressure?	……	……
9.	Has a doctor ever said you have diabetes?	……	……
10.	Has a doctor ever said you have asthma?	……	……
11.	Do you have a bone, joint or muscular problem which		

may be aggravated by exercise? …… ……

12. Do you have any form of injury? …… ……

13. Has your doctor ever said you have high cholesterol? …… ……

14. Have you suffered from a viral illness in the last 2 weeks? …… ……

15. Do you smoke, or have quit smoking in the last 6 months? ……. ……

16. Do you have a close blood relative who had a heart attack or heart surgery before age 55 (father or brother) or age 65 (mother or sister)? ……. ……

17. Has your doctor ever said you suffer from any taste abnormalities? ……. …….

18. Has your doctor ever said you suffer from heat related abnormalities ……. …….

I have completed the questionnaire to the best of my knowledge and any questions that I have raised have been answered to my full satisfaction.

Signed: ……

Date: ……

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Sodium ingestion and exercise performance

Consent Form for Participants

I agree to participate in the research project concerned with the assessment of cycling performance.

I have read the Information Sheet concerning this project and understand what it is about. All my questions have been answered to my satisfaction. I understand that I am free to request further information at any stage.

The study has been explained to me by the Researchers and I understand that I will have my height, nude weight, heart rate, temperature and aerobic fitness examined on four separate occasions, three of which will be in hot conditions. I understand that I will have urine and venous and capillary blood samples taken. I understand that I will be asked to ingest drinks during exercise. I understand the risks involved and if at any point, I am unhappy with the procedure or wish to stop I am able to.

- The information I give will only be used for the completion of a research student's investigation at the School of Sport and Health Sciences, University of Exeter, and I am aware and give my consent that this data may be published, but that my anonymity is maintained.
- The results will be stored on computer in coded form and password protected. Any raw data will be stored under 'lock and key' in a filling cabinet on the premises of the School of Sport and Health Sciences. In both circumstances it will only be available to the researcher. Data will be held until May 2014, whence it will be destroyed.
- I have the right to see the results and the completed study.
- I have the right to withdrawal from the study at any point.

Signed: …… [name]

Print Name: …….

Date: ……

This project has been reviewed and approved by the Ethics Committee of the of Sport and Health Sciences, University of Exeter

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Sodium ingestion and exercise performance

Pre-test Instructions

Thank you for agreeing to take part in the study. To help you successfully complete the test we would like you to complete the following instructions:

1. Avoid tiring exercise 24 hours before the tests. If you are involved in some sports training, try to miss some of the hardest exercises during your session.
2. Avoid caffeine and alcohol 24 hours before the test.
3. Otherwise, ensure you follow your normal diet.
4. On the day of the test, ensure you eat a normal balanced meal (e.g. breakfast: a bowl of cereal and milk or several slices of toast with jam should be enough).
5. Do not eat anything 1 hour before you test.
6. Avoid carbonated fizzy drinks.
7. Bring your sports kit (shorts, T-shirt and trainers)
8. It may also be useful to bring spare kit, because clothing can become saturated with sweat. For this reason it may also be useful to bring a towel with you.
9. Drink 500 mL of water (about a pint) 2 hours before the test.