HIGH FLUID INTAKES DO NOT IMPROVE 2 H RUNNING PERFORMANCES IN A 25 C ENVIRONMENT

Thesis submitted in partial fulfilment of the degree of

Master of Science (Exercise Science)

by

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DECLARATION

I, Hayley Natasha Daries, do hereby declare that this dissertation embodies only my original work except where acknowledgement indicates otherwise and that no part of it has been, or is being, submitted for a degree at this or any other university.

This thesis is presented in partial fulfilment of the requirements for the degree of MSc. (Exercise Science).

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Abstract

Purpose: In this study, we examined the effects of greater than ad libitum rates of fluid intake on 2 h running performances. **Methods:** Eight male distance runners performed three runs on a treadmill at 65% of peak oxygen uptake ($VO_{2\ peak}$) for 90 min and then 'as far as possible' in 30 min in an air temperature of 25 °C, a relative humidity of 55% and a wind speed of 13-15 $km.h^{-1}$. During the runs, the subjects drank a 6.9 $g.100ml^{-1}$glucose polymer solution containing 16 mEq $Na^{+}.l^{-1}$ of either ad libitum or in the set volumes of 150 or 350 $ml.70\ kg^{-1}$ body mass (~130 or 300ml) every 17.5 min **Results:** Higher (~0.9 vs. 0.4 $l.h^{-1}$) rates of fluid intake in the 350 $ml.70\ kg^{-1}$trial than in the other trials (which had the same results) had minimal effects on the subjects' urine production (~0.1 $l.h^{-1}$), sweat rates (~1.2 $l.h^{-1}$), declines in plasma volume (~8%) and rises in serum Na^{+} concentrations (~7 $mEq.l^{-1}$) during exercise. A greater (~1.05 vs. 0.45 $g.min^{-1}$) rate of CHO ingestion in the 350 $ml.70\ kg^{-1}$ trial than in the other trials also did not effect plasma concentrations of glucose (5 $mmol.l^{-1}$) and lactate (~3 $mmol.l^{-1}$) during the performance runs. In all three performance runs, plasma lactate concentrations rose from ~1.5 to 3 $mmol.l^{-1}$ with the increases in running speeds from ~14 to 15-16 $km.h^{-1}$,and rises in exercise intensities from ~65% to 75% of $VO_{2\ peak}$ elevated plasma lactate concentrations from ~1.5 to 3 $mmol.l^{-1}$and accelerated CHO oxidation from ~13 to 15 $mmol.min^{-1}$. The only effect of the intake of an additional ~1.0 l of fluid in the 350 $ml.70\ kg^{-1}$trial was to reduce the subjects' loss of body mass when compared to the other trials. But it also

produced such severe gastrointestinal discomfort that two of the eight subjects failed to complete their performance runs. **Conclusion:** Greater than ad libitum rates of fluid ingestion had no measurable effects on plasma volume and osmolality and did not improve 2 h running performances in a 25 °C environment.

Key words: drinking, gastrointestinal discomfort, exercise performance

Chapter One

LITERATURE REVIEW

THE EFFECTS OF DEHYDRATION ON FLUID HOMEOSTASIS, CARDIOVASCULAR FUNCTION AND PERFORMANCE DURING EXERCISE

Introduction

Perhaps the earliest studies of fluid balance in exercise were conducted in the 1920's and 1930's on men working in hot (32 - 49 °C) environments. Talbott et al. (1933) found that although these workmen had large sweat losses, their physiological functions remained within normal limits. They concluded that prolonged physical activity in the heat was not detrimental to an individual's health. Later, Adolf and Dill (1938) found that the average fluid intake of construction workers was about 4 l per day, in high environmental temperatures. However although sweat rates varied from 400 $ml.hr^{-1}$ at rest to 1500 –1700 $ml.hr^{-1}$ during 1-2 h of exercise in the heat, the workmen hardly drank during exercise. Fluid ingestion was greatest immediately after exercise and stopped when the workmen had replaced 50% of their fluid losses. These findings led to the concept of "voluntary dehydration".

The adverse effects of dehydration during exercise were only emphasized in the 1940's. Pitts et al. (1944) found that a complete replacement of sweat losses with fluid intake helps to maintain an individual's thermoregulatory and cardiovascular responses during exercise in the heat. When their subjects ran for 4 h at an ambient temperature of 38 °C and a relative humidity of 35 % in the heat without ingesting fluid, their rectal temperatures and pulse rates were higher and their sweat rates were lower than when they replaced their sweat losses with either 2 % saline or a 3.5 % glucose solution . In 1947, Adolf studied subjects who walked a distance of 33.6 km or until exhaustion in the heat (31 - 34 °C). They found that rectal temperatures, pulse rates and levels of dehydration increased with exercise time, and that subjects became exhausted at dehydration levels of ~ 4-8 % of body weight.

These early experiments demonstrated that, in the absence of adequate fluid replacement, progressive dehydration during prolonged exercise could adversely affect physiological function and exercise performance. However athletes were

unaware of the early industrial and military investigations showing the importance of adequate fluid replacement for prolonged exercise in the heat (Adolf, 1947; Talbott et al.1933). Marathon runners believed that drinking water was not beneficial, and therefore delayed or avoided drinking during a race, as a test of their fitness (Noakes, 1995). The first official reference to fluid replacement during long-distance running can be found in the 1953 Handbook of the International Amateur Athletic Federation (IAAF). The 1953 IAAF rules controlling marathon races (Figure 1) stated that "refreshments shall only be provided by the organisers of a race after 15 km or 10 miles, and thereafter every 5 km or 3 miles. No refreshments may be carried or taken by a competitor other than that provided by the organisers".

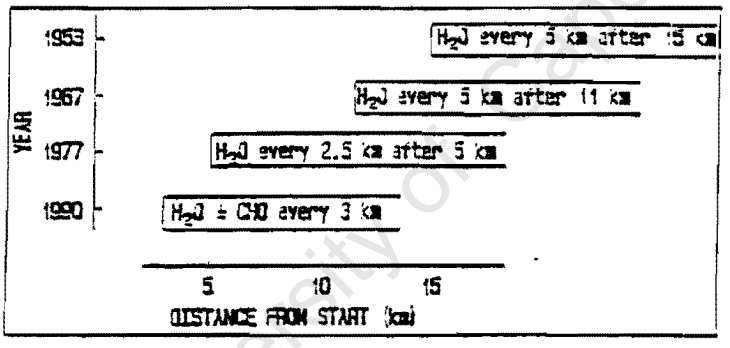

FIGURE 1. The evolution of the International Amateur Athletic Federation rules governing the intake of fluids during long-distance running.

Hawley et al. 1995

It was not until the late 1960's that there was an interest in fluid replacement during marathon running. Pugh et al. (1967) studied competitive marathon runners and found that the fastest runner (race winner) had the highest fluid loss, sweat rate and post-race rectal temperature. These runners only drank ~400 ml of fluid and dehydrated by ~5.9 % of body weight. Later, Wyndham and Strydom

(1969) performed a classic study in which they observed two groups of athletes competing in a 20 mile (32 km) road-race, and found that the runners who became dehydrated during the race had elevated post-race rectal temperatures. Further the most dehydrated athletes had the highest rectal temperatures. This finding led Wyndham and Strydom (1969) to speculate that the athletes' level of dehydration and their elevated post-race temperatures were causally related, and that dehydration alone was the most important factor determining rectal temperature during prolonged exercise (Wyndham, 1977; Wyndham and Strydom, 1969). Largely, as a result of Wyndham and Strydom's study, and a failure to note that rectal temperatures are more a function of metabolic rate than of dehydration (Noakes et al. 1991), the concept that fluid replacement alone was of primary importance for optimising performance during prolonged exercise was promoted. These notions were later reinforced by the study of Costill and Saltin (1974) which showed that during exercise gastric emptying was slowed by a 2.5 % glucose solution relative to water. These workers concluded that "the replacement of water was important during prolonged exercise in the heat and that only when competing in the cold could carbohydrate safely be included in solutions to be ingested during exercise". Accordingly water, in large volumes, was considered to be the optimum fluid replacement for ingestion during prolonged exercise.

At this time the IAAF, obviously became aware of the "dangers" of dehydration during long-distance events and changed their rules (Figure 1) so that competitors during a marathon could now have "refreshments provided by the organisers of the race after approximately 5 km or 3 miles". In addition, rule number 165, clause 4, stated that the "organisers shall provide sponging points where water only shall be supplied midway between (two) refreshment stations". (IAAF Handbook, page 98, 1977).

Fluid balance during prolonged exercise

Rates of fluid and electrolyte losses

Fluid loss during exercise is determined by the sweat rate, which is proportional to the athlete's metabolic rate (Costill, 1977; Davies, 1979; Davies et al. 1976; Greenhaff, 1989; Greenhaff et al.; Noakes et al. 1991; Wyndham et al. 1970). Typical sweat rates in runners during longer distance races are ~1.2 l. hr^{-1} (Noakes et al. 1993). Higher than 1.2 $l.hr^{-1}$ sweat rates are usually recorded only when the environmental temperatures are greater than 25°C, especially when the humidity is high (Armstrong et al. 1986; Costill et al. 1976).

Compared to electrolyte concentrations in cellular and extracellular fluids, sweat is hypotonic. The principal ions lost in sweat are sodium and chloride ions from the extracellular compartments (Costill, 1977), and their concentrations depend on an individual's level of fitness and state of heat acclimation (Allan and Wilson, 1971; Kirby and Convertino, 1986). Despite a 12 % increase in overall sweat rate *after* heat acclimation, sodium losses in sweat decrease by almost 60% (Kirby and Convertino, 1986). Thus for a given sweat rate, the heat acclimated individual loses less solute from the plasma.

Rates of fluid ingestion

Sweat rates invariably exceed rates of fluid intake during competitive exercise. Noakes (1993) reviewed voluntary rates of fluid intake and exercise-induced weight changes in runners, cyclists, and triathletes competing over a wide range of distances. That review suggests that the rates of fluid intake during exercise are seldom more than ~0.5 $l.hr^{-1}$ and are invariably less than sweat rates. During exercise lasting less than 6 h, athletes typically experience a weight loss of 2 or 3 kg and this loss of weight appears to be independent of either the type or duration of the activity (Noakes et al. 1993).

One explanation for the failure of both runners (Costill et al. 1970) and cyclists (Mitchell and Voss, 1991) to meet their fluid requirements is that they develop symptoms of abdominal "fullness" when they attempt to drink fluid at rates of between 1.2 and 1.7 $l.hr^{-1}$. The discomfort of drinking large volumes of fluid repetitively is especially marked during running (Brouns et al. 1987; 1991). Brouns et al. (1991) showed that the rate of fluid ingestion of subjects encouraged to drink as much as possible during a simulated triathlon was 2 to 3 times higher during the cycle leg (0.6 to 0.8 $l.hr^{-1}$) than during the running leg (0.1 to 0.3 $l.hr^{-1}$), suggesting that running reduces the desire to drink more than cycling. Feelings of abdominal fullness with high rates of fluid ingestion may also be due to limited rates of small bowel fluid absorption. Rates of water absorption may be less than rates of ingestion. Gisolfi et al. (1991) have calculated that only 37 % of fluid infused into the duodenum and jejenum at rates of 0.9 $l.hr^{-1}$ was absorbed, while others have reported that the maximum rate of water absorption from a 100 $mmol.l^{-1}$ isotonic sodium chloride solution was only 0.8 $l.hr^{-1}$ (Davis et al. 1980). Although Robinson et al.(1995) did not measure rates of fluid absorption, most of their subjects felt uncomfortably bloated, and was indicated by ratings of abdominal fullness during the 1 h performance ride, which suggested that some of the ingested fluid may have remained in the intestine. It has been suggested that maximal fluid absorption may be considerably less than 0.8 $l.hr^{-1}$, especially at high exercise intensity (Fordtran and Saltin, 1967). Several groups have suggested that rates of fluid absorption may be limited during exercise (Barr et al. 1991 and Williams et al. 1976). Thus, the maximum rates of fluid absorption by the small bowel during exercise could be less than the high rates of fluid loss incurred by athletes during intensive exercise.

Sodium chloride in sweat decreases during exercise as sodium is conserved with training. As sodium chloride sweat concentration reduces more, the sodium concentration in the plasma rises more which helps retain water. Nose et al. (1988 a;b;c) have shown that human drinking behaviour is regulated by changes in both serum osmolality and plasma volume. Hence dipsogenic drive in

dehydrated humans ceases prematurely when serum osmolality is returned to isotonicity by the ingestion of plain water before either fluid or sodium losses are replaced. Ingestion of solutions containing sodium chloride also terminates drinking prematurely by restoring plasma. But changes in osmolality and plasma volume during exercise-induced dehydration in humans are not dependent on each other, the rising serum osmolality with dehydration acts to maintain plasma volume and to reduce the volume-dependent drive for fluid replacement (Nose et al. 1988). The result is that whether dehydrated humans drink plain water or sodium chloride solutions, they tend to stop drinking before they are fully rehydrated.

Pitts et al (1944) reported that when working in the dessert heat, men do not voluntarily replace all the water losses incurred due to sweating. Adolf and coworkers (1947) called this phenomenon voluntary dehydration.

The rapid alleviation of symptoms which initiate drinking, such as dryness of the mouth, may cause premature cessation of drinking before full rehydration has occurred (Hubbard et al. 1990). Involuntary dehydration may also depend on a hereditary predisposition to be a "heavy" or "reluctant" drinker. Szlyk et al. (1989) reported that 13 of 33 men were classified as "reluctant" drinkers during exercise in the heat because they did not maintain body weight loss below 2% despite the provision of water ad libitum. Similarly, Itoh (1953) observed an average fluid intake of only 258 ml in 18 Oriental laborers after a mean exercise-induced sweat loss of 2.2 l. Thus, thirst does not appear to be a sufficient stimulus for maintaining body water during exercise. (Greenleaf, 1992).

The physiological effects of progressive dehydration during prolonged exercise

The physiological responses of hypohydration (fluid loss) induced by diuretics, saunas, and/or fluid restriction before exercise, are more marked than those resulting from exercise-induced dehydration (Coyle and Hamilton, 1990; Coyle and Montain, 1992; Sawka, 1991) and are not considered in this thesis. Instead, attention is focused on studies of the physiological effects of dehydration during exercise.

Plasma volume

Depending on the type and intensity of exercise and the posture adopted, plasma volume falls to varying extents within the first 10 min of exercise (Coyle and Hamilton, 1990). Thereafter, further falls in plasma volume are determined by the amount of fluid ingested during exercise. Below et al. (1995) found that increases in fluid intake from 0.2 to 1.3 l during 50 min rides at 80% of peak oxygen consumption (VO_{2peak}) helped maintain plasma volume when carbohydrate was present in the drink. Schroeder et al. (1997) also showed that increases in water consumption from 0.36 l to 0.52 l and 1.2 l progressively eliminated an 11 % fall in plasma volume during 1 h rides at 50% of VO2peak. Maughan et al. (1996) also reported that drinking 100 ml of a hypotonic glucose-electrolyte solution every 10 min during a cycle ride to exhaustion at 70% of peak VO_2 reduced the decline in plasma volume from ~ 7% in a no fluid trial to ~0.5% in a fluid trial. Montain and Coyle (1992b) found that increasing (0, ~0.3, ~0.7 and ~1.2 $l.hr^{-1}$) rates of fluid ingestion progressively reduced calculated falls in plasma volume from ~9% to ~6% during 2 h rides at ~65% of VO_{2peak} in a 33 ° C environment. However, others have observed that drinking does not measurably attenuate the estimated declines in plasma volume in the first 1-2 h of exercise. With or without fluid replacement, plasma volumes fell after the first 10 minutes of exercise by 8-12%

at 55-70% of VO_{2peak} (Barr et al. 1991; Montain and Coyle, 1992a), and by about 15% at ~85% of VO_{2peak} (Powers et al, 1990; Robinson et al. 1995).

Plasma Na^+ concentrations and osmolality

If no fluid is ingested during prolonged exercise, water and electrolyte losses in sweat increase plasma Na^+ concentration and osmolality (Cade et al. 1972; Greenleaf et al. 1980; Montain and Coyle, 1992b; Powers et al. 1990). Plasma osmolality rises because the concentration of sodium in the sweat is lower than that of plasma. To facilitate the lower rise in plasma sodium during exercise, a compartental shift occurs and water moves from the intracellular fluid compartment to the extracellular fluid compartment (Kozlowski and Saltin, 1964; Nose et al. 1988a; Sawka et al. 1984). Since rises in plasma osmolality with dehydration reduce skin blood flow (Montain and Coyle, 1992a; Nose et al. 1990), it is important for athletes to drink enough fluid to maintain their plasma osmolality in events where dehydration and thermoregulation are of primary concern (Coyle and Hamilton, 1990; Nadel et al. 1990; Noakes, 1993). A complete fluid replacement with either water or commercial carbohydrate-electrolyte solutions maintains plasma volume and osmolality (Barr et al. 1991; Cade et al. 1972; Greenleaf et al. 1980; McConnel et al. 1997; Powers et al. 1990) and extends the time to exhaustion in prolonged exercise, especially in the heat (Barr et al. 1991; Fallowfield et al. 1996; Maughan et al. 1989; McConnell et al. 1997; Millard-Stafford et al. 1992; Montain and Coyle, 1992 a;b). Although plasma volume responses to prolonged exercise in the heat are variable (Costill and Miller, 1980), plasma volume declines about 3-5% with the transition from rest to exercise and when prolonged exercise (i.e. cycling at 50% of VO_{2peak} in the heat) is performed "without fluid replacement" plasma volume continues to decline to levels 9% below the resting state.

Furthermore skin and muscle competes for blood flow and their combined needs can easily exceed the pumping capacity of the heart. Cutaneous vasodilation

displaces blood volume into cutaneous veins, and lowers cardiac filling pressure and stroke volume. The cardiac pumping capacity may be reduced at a time when the demands of oxygen transport to the skin are increased, which may exceed the heart's function. Hence, reduced/cessation of work output results from failure to maintain adequate muscle blood flow, and hyperthermia results from failure to maintain adequate cutaneous blood flow.

Sweat rates and skin blood flow

Dill's (1938) original proposal that a prevention of a rise in plasma osmolality may help to maintain sweat rates, however remains contentious. Some studies have shown that the sweat rates falls with increasing levels of dehydration (Ekblom et al.1970; Greenleaf and Castle, 1971; Ladell, 1955; Strydom et al. 1975) whereas others have failed to show this effect (Barr et al. 1991; Candas et al. 1988; Gisolfi and Copping, 1974; Hamilton et al. 1991; Montain and Coyle, 1992 a;b). These discrepancies may be due to the considerable individual variability in the effects of dehydration on sweat rates during exercise (Strydom and Holdsworth, 1968) or varying levels of dehydration in different experiments which may have a threshold rather than a graded response, or individual differences in sweat rates .

It is also debatable whether fluid ingestion increases sweat rates during exercise. Some investigators have reported that athletes sweat more with fluid ingestion than with no fluid ingestion (Ekblom et al. 1970; Greenleaf and Castle, 1971; Libert et al. 1986; Moroff and Bass, 1965) but other have found no effect of fluid ingestion on sweat rates. In the latter studies cyclists rode at 62-70% VO_{2peak} in ambient temperatures of either 22 °C (Hamilton et al. 1991) or 32-33 °C (Montain and Coyle 1992 a;b). Under both moderate and hot conditions, the cyclists' 1.2. - 1.4 $l.hr^{-1}$ sweat losses were unaffected by up to 1.2 $l.hr^{-1}$ rates of fluid intake.

Instead, fluid ingestion may attenuate the development of hyperthermia during exercise by maintaining skin blood flow (Montain and Coyle, 1992a; Nose et al. 1990). Fluid ingestion slows the reduction in forearm blood flow with increasing dehydration during exercise (Montain and Coyle, 1992 a;b).

Rectal or esophageal temperatures

Fluid ingestion also attenuates the rises in body core temperature after 60-80 min of exercise in the heat (Adolf, 1947; Barr et al. 1991; Cade et al. 1972; Costill et al. 1970; Gisolfi and Copping, 1974; Greenleaf and Castle, 1971; Hamilton et al. 1991; Montain and Coyle, 1992a,b; Strydom et al. 1975; Strydom and Holdsworth, 1968). Eventual rises in esophageal temperatures are linearly related to levels of dehydration (Cade et al. 1972; Montain and Coyle, 1992 a;b; Wyndham and Strydom, 1969). Sawka et al. (1985) reported that each percent decrease in body weight increased core temperature by 0.15 °C during exercise in the heat. Similarly Greenleaf and Castle (1971) found that core temperature was elevated by 0.10 °C for each percent decrease in body weight during exercise at about 50% of VO_{2peak}. Gisolfi and Copping (1974) reported that core temperature was elevated by 0.4 °C for each percent decrease in body mass of greater than 2% at an exercise intensity of 74% of VO_{2peak}.

Since the magnitude of the effect of fluid ingestion on rises in rectal temperature is relatively small, its physiological relevance may be questioned (Noakes et al. 1988). Most studies indicate that levels of dehydration of up to 5% (equivalent to a weight loss of 2 to 4 kg) usually increase rectal temperature by less than 1°C (Barr et al. 1991; Gisolfi and Copping, 1974; Hamilton et al. 1991; Montain and Coyle, 1992 a;b; Noakes et al. 1988).

Heart rate and stroke volume

Prolonged exercise causes "cardiovascular drift", characterised by a progressive increase in heart rate and a decrease in stroke volume throughout exercise. The drift increases with increasing environmental temperature and relative exercise intensity. This suggests that the major factor associated with cardiovascular drift is a shift of the circulating blood volume into the circulation of the skin to dissipate heat (Raven and Stevens, 1988).

Fluid deficits that develop during exercise also proportionately increase heart rates (Adolf, 1947; Cade et al. 1972; Candas et al. 1988; Greenleaf and Castle, 1971; Montain and Coyle, 1992b; Maughan et al. 1987; Strydom and Holdsworth, 1968) by reducing venous return and stroke volumes. Falls in stroke volumes and cardiac output are prevented when the rates of fluid ingestion are sufficient to maintain euhydration (Hamilton et al. 1991). However, heart rate is elevated even when dehydration is prevented by adequate fluid ingestion and infusion during exercise (Montain and Coyle, 1992a), suggesting that dehydration is not the sole cause of the progressive increase in heart rate during prolonged exercise (Hamilton et al. 1991). Since the rise in heart rate was prevented by a glucose infusion, Hamilton et al. (1991) have proposed that one component of cardiovascular drift during prolonged exercise may result from the rise in serum catecholamine concentrations that is reduced by glucose infusion.

Perception of effort

In the early studies of the effects of prolonged exercise-induced dehydration on physical work capacity of military personnel it was generally found that fewer subjects completed an exercise task when they did not ingest fluid (Adolf, 1947; Bean and Eichna, 1943; Eichna et al. 1945; Ladell, 1955; Pitts et al. 1944). Furthermore, it was observed that fluid ingestion had more obvious effects on the psyche than on the soma. Eichna at al. (1945) reported that dehydrated subjects

were "reduced to apathetic, listless, plodding men straining to finish the same task" they had completed "energetically and cheerfully" when fully hydrated. More modern studies have shown that the perception of effort during exercise is proportional to the fluid deficit (Montain and Coyle, 1992b). Even partial fluid replacement has a significant effect on the perception of effort during exercise of high intensity (Walsh and Noakes, 1992). Interestingly, fluid ingestion reduces the rise in the ratings of perceived exertion more than fluid infusion (Montain and Coyle, 1992a).

Exercise performance

Since the early studies of exercise-induced dehydration in subjects working at relatively low work rates for many hours in the dry heat, several groups have shown that fluid ingestion also extends endurance in moderate to high intensity exercise (55% -69.2 % VO_{2peak}) (Barr et al. 1991; Fallowfield et al. 1996; Maughan et al. 1989; McConnell et al. 1997; Millard-Stafford et al. 1992; Montain and Coyle, 1992 a;b). Even low (<2% of body mass) levels of dehydration may impair high-intensity exercise performance in 31-32 °C ambient temperatures. Walsh et al. (1994) showed that when cyclists fully replaced their ~1.1 l fluid losses during 1 h rides at 70% of peak oxygen consumption (VO_{2peak}) in the heat, they were able to cycle for 34% longer in a subsequent exercise bout at 90% of VO_{2peak}. Below et al. (1995) reported that subjects who drank 1.3 l of fluid during a 50 min ride at 80% of VO_{2peak} in the heat (31.2 ± 0.1 °C, 54 ± 3% relative humidity, 3.5.m.s^{-1} wind speed) were able to complete a subsequent (~10 min) work bout 6.5% faster than without fluid ingestion. In contrast, Robinson et al. (1995) found that water ingestion did not improve 1 h cycling performances in a moderate ambient temperature of 20 °C. Under that condition, an ingestion of 1.5 l of water had no measurable physiological benefit and only produced an uncomfortable abdominal fullness and reduced the distance covered in the 1 h ride by 2%, from 43.1 to 42.3 km ($P<0.05$). They concluded that trying to replace

more than 1.0 l.hr $^{-1}$ sweat losses during high intensity, short duration exercise in moderate environments may impair exercise performance.

The latter finding raises a question of whether it is advisable or practical for athletes to try and replace their fluid losses during competitions in thermoneutral conditions. Most endurance athletes voluntarily drink less than 0.5 l of fluid.h^{-1} and lose up to 1.0-1.5 l of sweat.h^{-1} in distance races conducted in 20-25 °C environments (Noakes, 1993). When athletes attempt to drink fluid at rates closer to their 1.2 l.h^{-1} and above sweat rates, they generally experience gastrointestinal discomfort, especially during running (Costill et al. 1970; Mitchell and Voss, 1991). Therefore, the American College of Sports Medicine position stand on exercise and fluid replacement states that "*during prolonged exercise, frequent (every 15-20 min) consumption of moderate (150 ml) to large (350 ml) volumes is possible*" but recommends that "*individuals learn their tolerance limits for maintaining a high gastric volume for various exercise intensities*" (ACSM, 1996). In this study, we examined whether 0.4 and 0.9 l.h^{-1} rates of fluid intake confer any advantage over ad-libitum fluid ingestion on 2 h running performances in a thermoneutral environment.

Chapter Two

METHODS

Eight male endurance-trained runners participated in this study. All of the subjects ran regularly more than 60 km per week and had completed marathon races in under 2 h: 50 min. The study was approved by the Research and Ethics Committee of the Faculty of Medicine of the University of Cape Town and each subject signed an informed consent.

Preliminary testing

The means and standard deviations of the subjects' ages, body masses, heights, peak O_2 uptakes (VO_{2peak}) and peak treadmill speeds (PTS) were 31 ± 4 years, 61 ± 9 kg, 171 ± 9 cm, 68 ± 3 $ml.min^{-1}.kg$ body $mass^{-1}$ and 20 ± 1 $km.h^{-1}$, respectively. Body masses and heights were determined with a Model 770 Seca precision balance and stadiometer (Seca, Bonn, Germany). VO_{2peak} and PTS were measured during a progressive exercise test to exhaustion on a Powerjog EG 30 treadmill (Sports Engineering Ltd, Birmingham, U.K.). Runs were started at a treadmill speed of 13 $km.h^{-1}$ and increased by 0.5 $km.h^{-1}$ every 30 s until the subject could no longer maintain the pace, as described by Scrimgeour et al (1986).

During the exercise test, subjects wore a nose-clip and breathed through a mouthpiece connected to an Oxycon Alpha automated gas analyser (Mijnhardt, Netherlands). Before each test, the analyser was calibrated with a Hans Rudolph 3 l syringe (Vacumed, Ventura, USA), and the oxygen and carbon dioxide analysers were calibrated with room air and a 5% CO_2:95% N_2 gas mixture.

Pneumotach and gas analyser outputs were processed by a computer which calculated breath by breath ventilation, oxygen consumption (VO_2) and carbon dioxide production (VCO_2) l min-1. VO_{2peak} values were the average of the highest VO_2 values measured over the final 60 s.

Following the measurements of VO_{2peak}, the subjects rested for about 5 min and then performed a familiarisation run on the treadmill. At first, they ran at ~65% of VO_{2peak} for 45 min and then they ran "as far as possible" in 15 min on the treadmill by adjusting their own running speed. These performance runs were conducted in our environmental chamber (Scientific Technology, Cape Town, South Africa) at an ambient temperature of 25 °C, a relative humidity of 55% and a wind velocity equal to the 13-15 $km.h^{-1}$ treadmill running speeds of the subjects in the first 60 min of the experimental trials. In this practice run, there were no measurements and the subjects drank as and what they wished. The only purpose of the run was to familiarise the subjects with the conditions to be used in the experimental trails and to minimise any learning effect.

Experimental trials

At weekly intervals after the practice run, the subjects returned to the laboratory at the same time of day and repeated, in random order, the three experimental performance runs. Over that period, the subjects continued their usual training and consumed similar diets, but refrained from strenuous physical activity on the

day before each trial. Training and dietary records were kept in order to aid the subjects' compliance with the requests.

On the day of the trial, the subjects ate a standardised breakfast and arrived in the laboratory, 2 h after drinking 500 ml.70 kg^{-1} body mass of a commercial 6.9 g.100 ml^{-1} glucose polymer solution containing 16 mEq $Na^{+}.l^{-1}$. The consumption of 380-580 ml of fluid 2 h before the trials was designed to ensure equal hydration at the start of exercise. Euhydration was later confirmed by similar pre-trial body masses, haematocrits, haemoglobin concentrations and serum osmolalities (Table 1).

Table 1 Evidence of equal hydration before exercise

Trial	Ad libitum	150 ml·70 kg^{-1}	350 ml·70 kg^{-1}
Body mass (kg)	60.5 ± 9.1	60.5 ± 9.2	60.0 ± 9.5
Heamoglobin (g·100 ml^{-1})	15.3 ± 0.9	15.3 ± 1.2	15.6 ± 1.3
Haematocrit (%)	41.7 ± 2.0	42.2 ± 2.0	42.8 ± 2.3
Osmolality (mosmol·l^{-1})	281 ± 6	278 ± 3	281 ± 6

Results are means ± SD

At the laboratory, the subjects urinated and weighed themselves in the nude. An 18-gauge cannula was then inserted into an antecubital vein and attached to a three-way stopcock for the withdrawal of blood. Venous blood samples (10 ml) were collected (a) after the subject had stood on the treadmill for 10-15 min, (b) at

15 min intervals during the 90 min run at ~65% of VO_{2peak} and (c) at the end of a subsequent 30 min performance run. Following the collection of each blood sample, the venous cannula was flushed with 1-2 ml of sterile saline containing heparin (5 $IU.l^{-1}$) to prevent coagulation.

Once a venous blood sample had been drawn at rest, the subjects began running at `65% of VO_{2peak} for 90 min. At the end of this run, there was a 2 min rest interval before the 30 min performance run. In that interval and at the end of the performance run, the subjects towelled dry, urinated and re-weighed themselves in the nude for later calculations of body water loss. Sweat rates were calculated from the decreases in body mass plus the volume of fluid ingested minus any urine excreted. No corrections were made for the approximately 150 g of carbon lost in the measured production of CO_2 during the trials.

During the runs, the subjects drank the commercial 6.9 $g.100\ ml^{-1}$ glucose polymer solution containing 16 mEq $Na^+.l^{-1}$ either ad libitum or in set volumes of either 150 or 350 $ml.70kg^{-1}$ body mass every 17.5 min. The set volumes of 150 ml and 350ml are equal to ingestion rates of ~0.5 - 1.2 $l.hr^{-1}$ respectively, based on a 70kg person and was adjusted accordingly for greater or lesser body weight. The amount administered was adjusted accordingly for greater or lesser body weight. Before each drink, the subjects were asked to indicate their ratings of perceived exertion using the 20-point Borg scale (Borg, 1975) and to rank their stomach fullness on a scale of 1 (empty) to 5 (uncomfortably bloated). Between drinks,

VO_2 and VCO_2 were measured over 6 min intervals of which the timing was standardised, and used to calculate rates of carbohydrate (CHO) and fat oxidation using the formulae of Frayn et al. (1983), assuming a non-protein respiratory exchange ratio. Rates of CHO oxidation in $g.min^{-1}$ were converted to $mmol.min^{-1}$ by dividing the values by the 180 $mg.mmol^{-1}$ molecular weight of glucose.

Blood analyses

Venous blood samples (10 ml), collected during the trials, were divided into three aliquots and stored on ice until the end of the trial. One aliquot (1 ml) was placed into a tube containing lithium heparin for determinations of haematocrit and haemoglobin concentration, immediately after the trial. Another aliquot (5 ml) was allowed to clot (in a tube containing SST gel and clot activator), spun at 2500 x g for 12 min in a Sigma 302K centrifuge (Laborzentrifugen, Germany) at 4 °C and the supernatent was stored at -20 °C for determinations of serum sodium and potassium concentrations and osmolality. The remaining blood (4 ml) was placed into a tube containing sodium fluoride and potassium oxalate, centrifuged at 2500 x g for 12 min at 4 °C and the supernatent was stored at -20 °C for later measurements of plasma glucose and lactate concentrations.

Haematocrits were measured in triplicate by micro-centrifugation. Haemoglobin concentrations were determined in triplicate with the cyanomethemoglobin method of Hainline (Hainline, 1958) using a Beckman DU 60 spectrophotometer

(Beckman Instruments, Fullerton, Calif. USA). Changes in haematocrit and haemoglobin concentration were used to estimate the decreases in plasma volume during exercise, as described by Dill and Costill (1974).

Serum osmolalities were determined from freezing point depressions in an Osmette A automatic osmometer (Precision Systems, Newton, Mass., U.S.A.). Serum Na^+ and K^+ concentrations were assayed with ion selective electrodes (KNA 1 Radiometer, Copenhagen, Denmark). Plasma glucose concentrations were measured with an automated LM 3 glucose analyser (Analox Instruments, London, UK). Plasma lactate concentrations were determined with a spectrophotometric enzymatic assay using a commercial kit (Lactate Pap, Bio Merieux, Marcy-L Etoile, France).

Statistical analyses

All results are expressed as means ± standard deviations (SD). Statistical analyses were performed with a two-way analysis of variance (ANOVA) for repeated measures and the Scheff's post-hoc test. Differences in ratings of perceived exertion and stomach fullness were tested with a non-parametric Kruskal-Wallis ANOVA. A value of $P < 0.05$ was regarded as significant for all determinations.

Chapter Three

RESULTS

Before each of the 90 min runs at 65% of VO2 peak and subsequent 30 min performance runs, the subjects weighed themselves and a blood sample was taken at rest. Similar body masses, haemoglobin concentrations, haematocrits and serum osmolalities indicated later that each subject was equally hydrated at the start of the three trials (Table 1).

Fluid balance

During the trials, the subjects drank the 6.9 g.100 ml-1 glucose polymer solution containing 16 mEq $Na^{+}.l^{-1}$ either ad libitum or at rates of 150 or 350 ml.70 kg-1 body mass every 15-20 min. Total fluid intakes in the ad libitum, 150 ml.70 kg-1 and 350 ml.70 kg-1 trials were 0.76 ± 0.35 l, 0.78 ± 0.12 l and 1.83 ± 0.28 l, respectively (Fig. 2). The intake of an extra ~1 l of fluid in the 350 ml.70 kg^{-1} trials did not significantly increase the excretion of urine immediately after exercise above that in the other two trials (Fig. 2). Compared to the 0.19 ± 0.24 l and 0.11 ± 0.21 l urine volumes in the ad libitum and 150 ml.70 kg^{-1} trials, the 0.36 ± 0.44 l urine volume in the 350 ml.70 kg^{-1} trials was only increased by 0.1-0.2 l ($P = 0.15$).

Differences in fluid intakes also had little effect on the estimated sweat rates during the 90 and 30 min runs (Fig. 2). When the mass of the retained, ingested fluid was added to the loss of body mass during each run, the calculated sweat rates were found to be relatively constant over time and similar from trial to trial. Average sweat rates in the ad libitum, 150 ml.70 kg^{-1} and 350 ml.70 kg^{-1} trials

were 1.29 ± 0.41, 1.15 ± 0.44 and 1.21 ± 0.33 $l.h^{-1}$, respectively. The only effect of the additional fluid intake was to reduce the subjects' loss of body mass from 1.179 ± 0.375 % and 0.954 ± 0.444 % in the ad libitum and 150 $ml.70\ kg^{-1}$ trials to 0.45 ± 0.266 % in the 350 $ml.70\ kg^{-1}$ trials (Fig. 2).

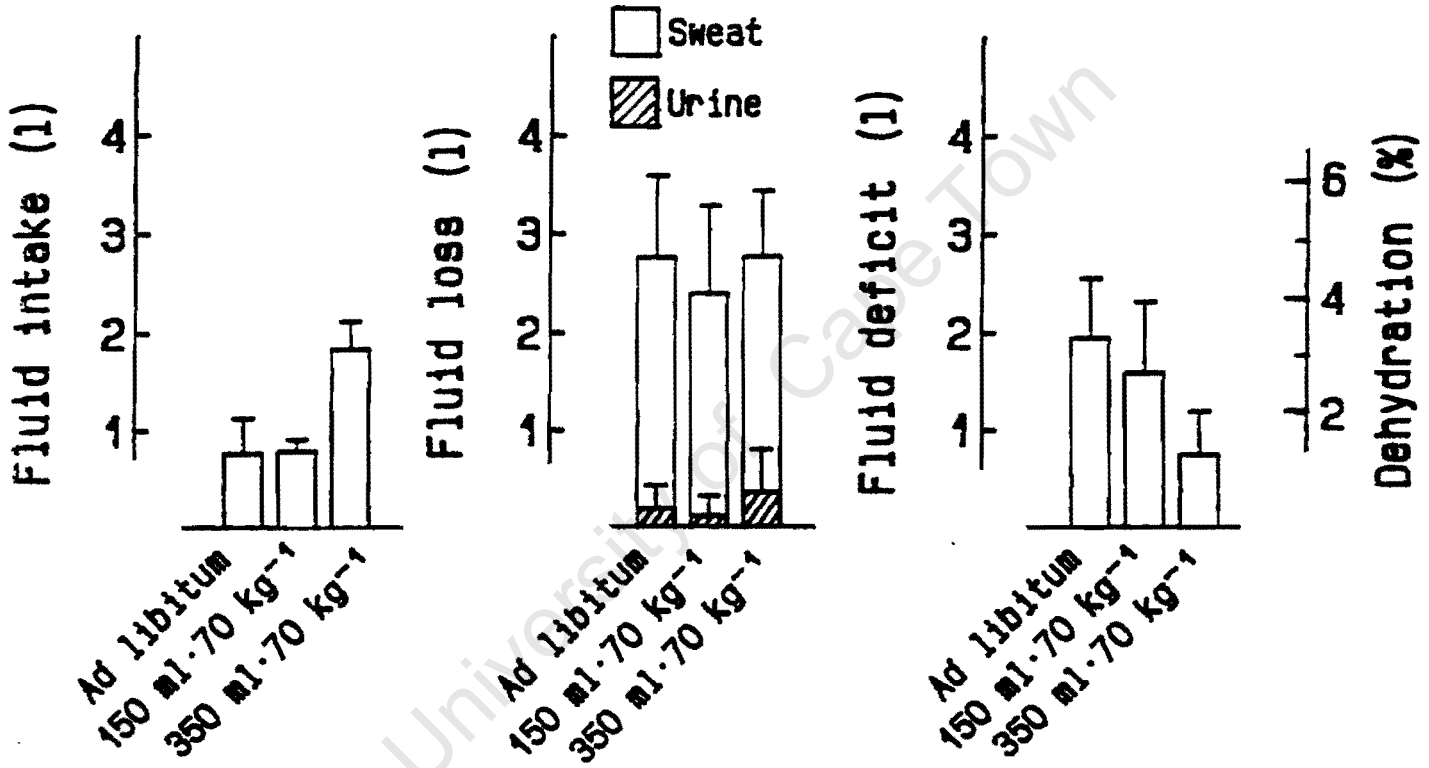

Fig. 2 Effects of ingested fluid volume on fluid balance during exercise.

Increases in fluid intake from 150 to 350 $ml{\cdot}70\ kg^{-1}$ body mass every 17.5 min during the 2 h runs reduced the cumulative mean ± SD of the fluid deficit ($P < 0.001$)

Plasma volume

The calculated decreases in plasma volume (Fig. 3) was not attenuated by less (1.2% vs. 2.7 - 3.4%) dehydration in the 350 ml.70 kg^{-1} trials than in the other trials. In all three trials, plasma volumes fell by ~6% in the first 15 min of exercise ($P < 0.01$) and then declined by a further 2% in the remainder of the trial.

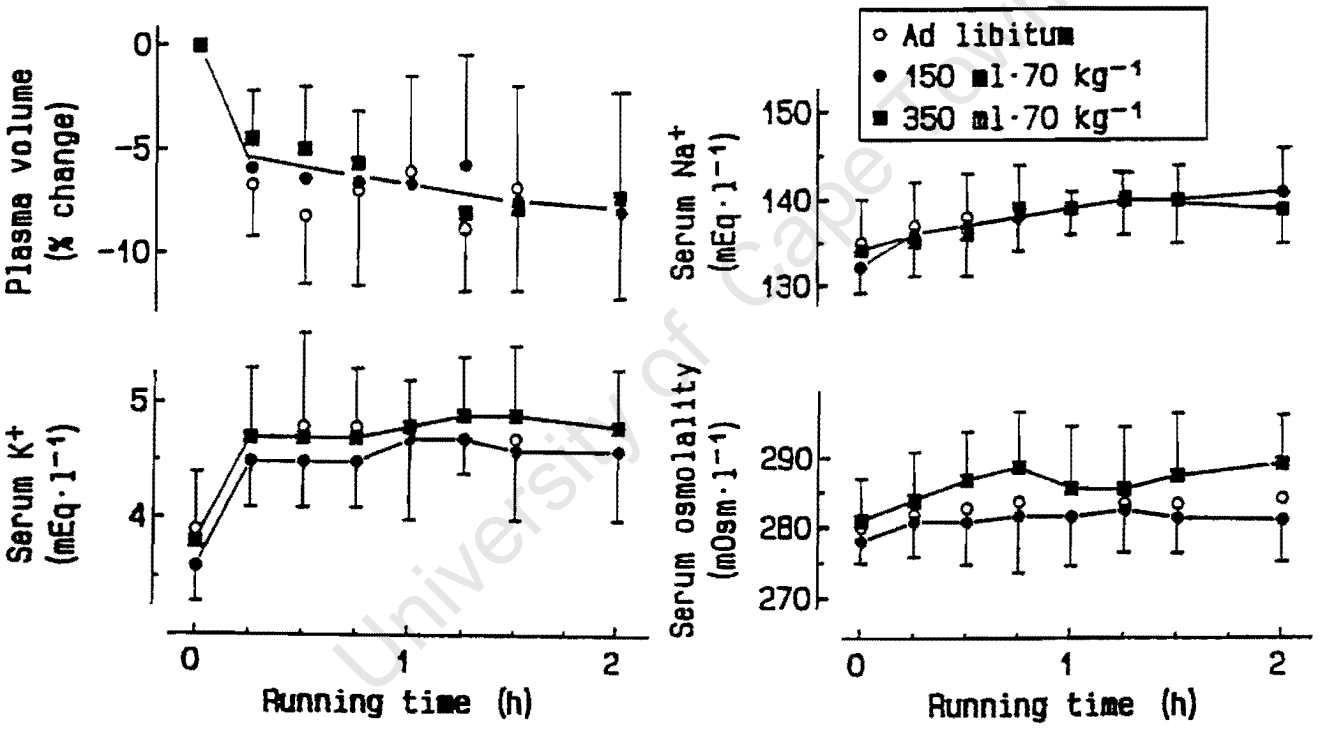

Fig. 3 Effects of ingested fluid volume on plasma volume, serum K^+ and Na^+ concentrations and serum osmolality during exercise.

Means ± SD were similar in the three trials and, after 15 min of exercise, did not change significantly over time.

Serum K^+ and Na^+ concentrations and osmolality

The fluid ingestion regimen also had little influence on serum K^+ and Na^+ concentrations and osmolality (Fig. 3). Irrespective of whether the average drinking rate was 0.39 ± 0.06 $l.h^{-1}$ or 0.91 ± 0.14 $l.h^{-1}$, circulating K^+ concentrations rose from 3.6 - 3.9 $mEq.l^{-1}$ to 4.6 - 4.8 $mEq.l^{-1}$in the first 15 min of exercise ($P < 0.01$) and then remained constant for the rest of the trial. In contrast, the ~7 $mEq.l^{-1}$ and ~5 $mOsm.l^{-1}$ increases in serum Na^+ concentrations and osmolalities in the three trials were more gradual and not statistically significant.

Plasma glucose and lactate concentrations

There was also no effect of the higher (1.05 ± 0.16 vs. 0.45 ± 0.07 $g.min^{-1}$) rates of CHO ingestion on $^{-1}$trials (Fig. 4). In all three runs, plasma glucose concentrations remained between 5.5 and 6.5 $mmol.l^{-1}$. Plasma lactate concentrations were also similar in the ad libitum, 150 $ml.70\ kg^{-1}$ and 350 $ml.70\ kg^{-1}$ trials (Fig. 4). At the end of the 90 min runs at ~65% of VO_{2peak}, plasma lactate concentrations were 1.2 ± 0.2, 1.3 ± 0.6 and 1.5 ± 0.6 $mmol.l^{-1}$and, at the end of the 30 min performance runs, they were increased to 2.9 ± 1.8, 2.9 ± 1.8 and 3.2 ± 2.1 $mmol.l^{-1}$, respectively ($P < 0.01$).

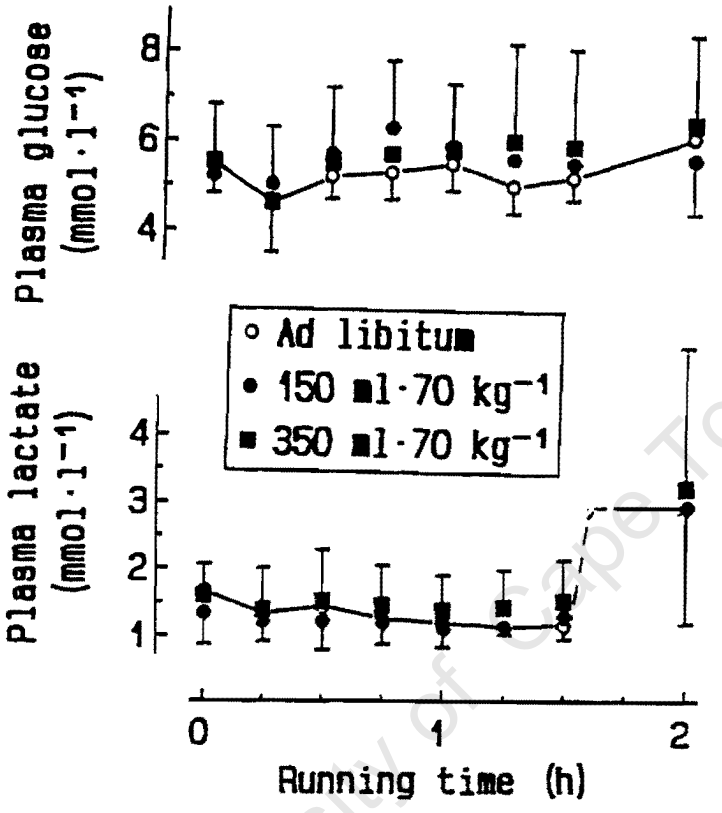

Fig. 4 Effects of ingested fluid volume on plasma and lactate concentrations during exercise

Higher average (1.05 vs. 0.45 g.min $^{-1}$) rates of CHO ingestion in the 350 ml.70kg^{-1} trials than in the 150 ml.70kg^{-1} trials had no effect on mean ± SD plasma glucose and lactate concentrations during exercise.

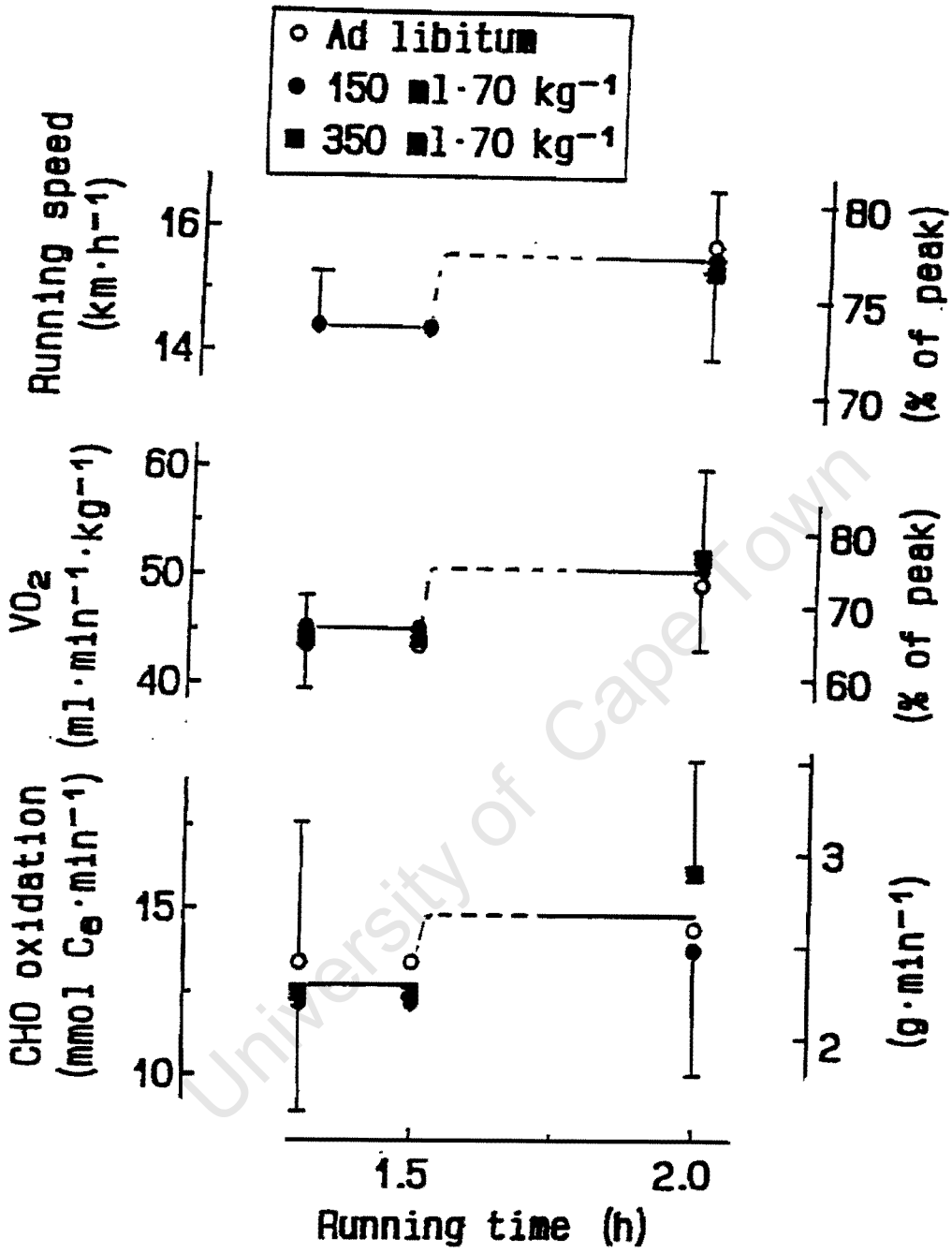

Fig. 5 Effects of ingested fluid volume on increase in running speed, VO_2 and CHO oxidation during the 30 min performance runs.

Increases in mean ± SD treadmill speeds, VO_2 values and rates of CHO oxidation in the 30 min performance runs were similar in the three trials. The first data points are from the steady state exercise.

Running performances

Rises in plasma lactate concentrations in the 30 min performance runs were associated with increases in treadmill running speeds (Fig. 5). Following the 90 min runs at ~65% of VO_{2peak}, the subjects accelerated from 14.3 ± 0.9 $km.h^{-1}$ to 15.8 ± 0.9, 15.6 ± 1.1 and 15.4 ± 1.4 $km.h^{-1}$ in the ad libitum, 150 $ml.70\ kg^{-1}$ and 350 ml.70 kg-1 trials. Increases in running speeds during the time trial raised $VO_2{}^{-1}.kg^{-1}$, or from about 65% to about 73%, 75% and 76% of VO_{2peak}, respectively (Fig. 5). Less dehydration in the 350 $ml.70\ kg^{-1}$ trials than in other trials had no effect on VO_2 uptake at given running speeds. Oxygen costs per kilometer in the ad libitum, 150 $ml.70\ kg^{-1}$ and 350 $ml.70\ kg^{-1}$ treadmill performance runs were 192 ± 26, 196 ± 23 and 203 ± 27 $ml.\ kg^{-1}.\ km^{-1}$, respectively. Fig. 5 illustrates that there was no performance enhancing effect in any of the three trails.

Rates of CHO oxidation were also unaffected by the volumes of fluid consumed (Fig. 5). In all three trials, mean rates of CHO oxidation decreased steadily from 17 ± 4 to 13 ± 3 $mmol.min^{-1}$ during the 90 min runs and then increased to 15 ± 6 $mmol.min^{-1}$ with the rise in exercise intensity from ~65% to 75% of VO_{2peak} in the 30 min performance runs (both $P < 0.05$). However, the acceleration of CHO oxidation in the performance runs did not increase the percent contribution to energy production from CHO oxidation. Respiratory exchange ratios indicated that the percent contribution to energy production from CHO oxidation fell from 87

5% to 65 ± 4% during the 90 min runs and then remained at 65 ± 5% during the 30 min performance runs (data not shown).

Ratings of perceived exertion and stomach fullness

Ratings of perceived exertion (RPE) were also similar in the three trials (Fig. 6). During each trial, RPE rose from ~9 to 15 units. In contrast, ratings of stomach fullness (RSF) were significantly greater in the latter half of the 350 ml.70 kg^{-1} trial than in the other trials (Fig. 6). Two of the eight subjects experienced such severe gastrointestinal discomfort in the 350 ml.70 kg^{-1} trial that they failed to complete their 30 min performance runs. Those subjects stopped running after 15 and 18 min.

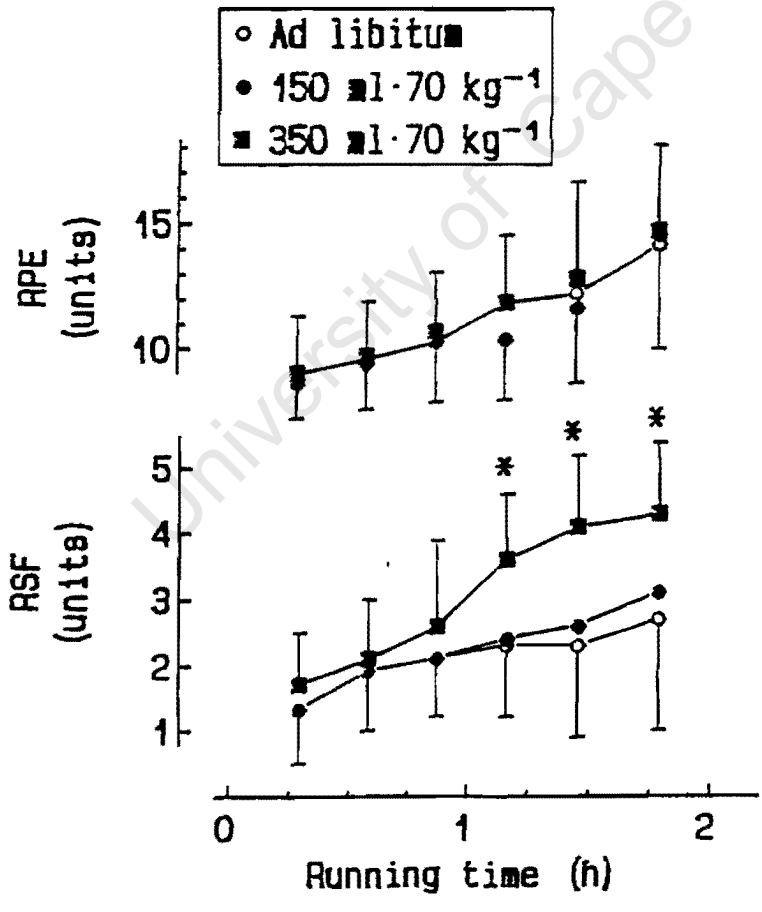

Fig. 6 Effects of ingested fluid volume on ratings of perceived exertion and stomach fullness during exercise.

Although ratings of perceived exertion (RPE) were similar in the three trials, ratings of stomach fullness (RSF) were significantly greater in the latter half of the 350 ml·70 kg^{-1} trial than in the other trials ($P < 0.05$)

Chapter Four

Discussion

In this investigation, eight, equally hydrated (Table 1) male endurance runners ran on a treadmill at 65% of VO_{2peak} for 90 min and then "as far as possible" in 30 min in a 25 °C environment. During the runs, the subjects drank a commercial 6.9 g.100 ml^{-1} glucose polymer solution containing 16 mEq $Na^+.l^{-1}$ either ad libitum or in the set volumes of 150 or 350 ml.70 kg^{-1} body mass every 15-20 min intervals. Increases in fluid intake from ~0.4 $l.h^{-1}$ in the ad libitum and 150 ml.70 kg^{-1} trials to ~0.9 $l.h^{-1}$ in the 350 ml.70 kg^{-1} trial had little effect on the subjects' 0.05-0.20 $l.h^{-1}$ urine production during exercise and no effect on their ~1.2 $l.h^{-1}$ sweat rates (Fig. 2). Others have also found that fluid ingestion does not significantly increase urine production or sweat rates during moderate intensity exercise. In those studies, cyclists rode at 62-70% of VO_{2peak} in ambient temperatures of either 22 °C (Hamilton et al. 1991) or 32-33°C (Montain and Coyle, 1992 a;b). Under both moderate and hot conditions, the cyclists' 1.2-1.4 $l.h^{-1}$ sweat losses were unaffected by up to1.2 $l.h^{-1}$ rates of fluid intake.

In contrast, Montain and Coyle (1992b) found that increasing (0, ~0.3, ~0.7 and ~1.2 $l.h^{-1}$) rates of fluid ingestion progressively reduced estimated falls in plasma volume from ~9% to ~6 % and reduced rises in serum Na+ concentration from ~6 to ~2 mEq.l^{-1} during 2 h rides at ~65% of VO_{2peak} in a 33°C environment. However, others have observed that drinking does not measurably attenuate declines in plasma volume in the first 1-2 h of exercise. With or without fluid replacement, plasma volumes fell by 8-12% at 55-70% of VO_{2peak} (Barr et al, 1991; Montain and Coyle, 1992a), and by about 15% at ~85% of VO_{2peak} (Powers et al. 1990; Robinson et al. 1995). In the present study, the intake of ~1.0 l of additional fluid in the 350 ml.70 kg^{-1} trial also did not reduce the estimated declines in plasma volume (Fig. 3). Irrespective of the amount of fluid ingested, plasma volumes decreased by ~8%, circulating K^+ and Na^+ concentrations

increased by ~1 and ~7 mEq.l^{-1} and serum osmolality rose by ~5 mOsm.l^{-1}. The observation that serum Na^+ concentrations did not fall when fluid was ingested at the highest rates suggests a benefit of consuming electrolyte-containing drinks during prolonged exercise.

A greater (~1.05 vs. 0.45 g.min^{-1}) rate of CHO ingestion in the 350 ml.70 kg^{-1} trial than in the other trials also did not effect plasma 5 mmol.l^{-1} glucose and ~3 mmol.l^{-1} lactate concentrations during the 30 min performance runs (Fig. 4). In all three performance runs, lactate concentrations rose from ~1.5 to 3 mmol.l^{-1} with the increases in running speeds from ~14 to 15-16 km.h^{-1}, the rises in exercise intensities from ~65% to 75% of VO_{2peak} and the accelerations of CHO oxidation from ~13 to 15 mmol.min^{-1} (Fig. 5). However, the acceleration of CHO oxidation in the performance runs did not increase its percent contribution to energy production. In all three trials, the percent contribution to energy production from CHO oxidation fell from about 87% to 65% during the 90 min sub-maximal runs and then remained at around 65% during the 30 min performance runs (data not shown).

A failure of the rise in exercise intensity to influence patterns of fuel utilisation is contrary to the prediction of the "crossover" concept (Brooks and Mercier, 1994) and may be due to the ingestion of CHO during exercise. Rauch et al. (1995) found that CHO ingestion eliminated 50% vs. 65% differences in final contributions to energy production from CHO oxidation in cyclists' drinking water

during 3 h rides at 55% or 70% of VO_{2peak}. Van Zyl et al. (1996) also showed that the contribution to energy production from ingested and endogenous CHO oxidation remained at ~65% during a simulated 40 km time-trial at nearly 80% of VO_{2peak} after a 2 h ride at 60% of VO_{2peak}, therefore CHO oxidation is maximised, and increased exercise intensity does not benefit to increase fuel utilization.

Only the ratings of stomach fullness were different in the three trials (Fig. 6). Most of the eight subjects felt uncomfortably bloated in the 350 ml.70 kg^{-1} trial and two of the subjects experienced such severe gastrointestinal discomfort that they failed to complete their performance runs. Those subjects did not have particularly low sweat rates and were not reluctant drinkers in the ad libitum trial. More gastrointestinal discomfort with greater than ad libitum rates of fluid ingestion may have been due to either the athletes unfamiliarity with running with a full stomach or to delays in gastric emptying at high exercise intensities (Moses, 1990). Part of the uncomfortable abdominal fullness could also have been caused by limits to the rates of fluid absorption from the small intestine. Although reported maximum rates of intestinal fluid absorption are about 0.8 $l.h^{-1}$ at rest (Davis et al. 1980), they may be less during exercise. Some believe that intestinal absorptive capacity is unaffected by exercise intensities that can be sustained for 30 min (Gisolfi et al. 1991; Schedl et al. 1994) and other data supports that high exercise intensities decrease rates of intestinal fluid absorption (Fordtran and Saltin, 1967; Williams et al. 1976). It is also possible

that fluid absorption was slowed by the 6.9% CHO content of the drink. Ryan et al (1984) have shown that an increase in the CHO content of fluid ingested during exercise from 6 to 8% slows the absorption of water by 50%. Whatever the mechanism, these data suggest that it may not be possible for athletes to absorb sufficient fluid to maintain hydration during competitive running exercise, which may be limited by either gastric emptying or intestinal absorption. The additional ingestion of about 1.0 l of fluid in the 350 ml.70 kg^{-1} trial had no measurable effects on plasma volume and osmolality and did not improve 2 h running performances in a 25 °C environment. Robinson et al. (1995) also found that fluid ingestion did not influence plasma volume or improve 1 h cycling performances in a moderate 20 °C environment. In that study, an ingestion of 1.5 l of water only produced an uncomfortable abdominal fullness and reduced the 'distances covered' in the simulated rides by 2%.

One reason why greater than ad libitum rates of fluid ingestion did not improve running speeds may be that self-paced treadmill runs are too variable to detect subtle changes in exercise performance. Schabort et al. (1998) found that a 1.8 - 4.0% 95% confidence interval in the performances of eight subjects who ran 'as far as possible' on a treadmill in 1 h on three occasions. Alternatively, the effect of fluid replacement on performance may be more noticeable in exercise conducted over longer durations or in hot environments where adequate hydration and thermoregulation are of greater concern. Our findings only apply to 2h of exercise. In more prolonged exercise leading to greater levels of

dehydration, athletes probably should follow the ACSM recommendation and "*attempt to consume fluid at a rate sufficient to replace all the water lost through sweating or consume the maximal amount that can be tolerated*" (ACSM, 1996). Most studies have shown that drinking delays fatigue in prolonged exercise (Barr et al. 1991; Fallowfield et al. 1996; Maughan et al. 1989; McConnell et al. 1997; Millard-Stafford et al. 1992; Montain and Coyle, 1992 a;b); and improves exercise performance in the heat (Below et al. 1995; Walsh et al. 1994).

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