

THERMAL REGULATORY PROBLEMS ASSOCIATED WITH PLAYING SOCCER

By

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ABSTRACT

Playing soccer in a hot environment without drinking fluids is likely to cause dehydration and consequently heat stress related conditions and poor performance. The objective of this study was to determine the amount of total body water loss that soccer players undergo on exposure to various ambient temperatures, *ad libitum*, before, and after exercise. Sixty-one volunteers, mean age 23 ± 3 who play soccer in the Premier Soccer League (PSL) and the First Division Soccer League were recruited into the study. Before the match, they were asked to empty their urinary bladders. They were monitored for changes in body weight and core temperature, albuminuria, urine volume and flow rate, sweat loss, sweating rate and fluid intake. Overall dehydration (total body mass loss) 2.21 ± 0.83 %, sweat loss 1.580 ± 0.71 litres, sweating rate 0.84 ± 0.64 litres/hour, core temperature increase 1.79 ± 0.83 %, water intake 0.28 ± 0.23 litres, urine output $2.4 \times 10^{-2} \pm 1.5 \times 10^{-2}$ litres, urine flow rate $2.8 \times 10^{-4} \pm 1.6 \times 10^{-4}$ litres/minute and albuminuria 1.2 ± 1.4 g/litre. There was a significant correlation between dehydration and water intake ($P < 0.05$) and a strong correlation between dehydration and increase in core temperature and sweat loss ($P < 0.01$). Increase in core temperature had a strong correlation with dehydration, water intake and sweat loss ($P < 0.01$). On predicting dehydration sweat loss, urine output and water intake were significant predictors ($P < 0.05$). On predicting temperature increase, sweat loss was the only predictor ($P < 0.05$). There was a strong positive correlation between the outcome of the game and both the temperature and the extent of dehydration ($P < 0.01$). In conclusion, the results suggest soccer players do not take adequate fluids during the game leading to reduced body mass due to dehydration and consequently poor performance. The increase in the body temperature seemed not to have much bearing on the extent to which dehydration may occur.

DECLARATION

I, Carol Batsirai Maponga, hereby declare that this submission is my own work and that, to the best of my knowledge and belief, it contains no material previously published or written by another person nor material which to a substantial extent has been accepted for the award of any other degree or diploma of the university or other institute of higher learning, except where due acknowledgment has been made in the text.

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STATEMENT REQUIRED UNDER REGULATION 10/1/8**MASTER OF PHILOSOPHY (M.PHIL) DEGREE****Conjoint work with supervisor and other persons**

This thesis describes the work on thermal regulation and its effects on hydration and performance in the field of exercise and performance physiology under the direction of Dr Kaka SMT Mudambo, Department of Physiology, College of Health Sciences, University of Zimbabwe. The work forms an extension of the work in the area of sports medicine and adaptation medicine by Dr Kaka SMT Mudambo. Except for the statistical analysis performed by Ms L. Siziba of the Department of Statistics, Faculty of Science, University of Zimbabwe, all other assessments and analysis of subjects and samples were carried out by Ms C. B. Maponga. However, Ms C.B. Maponga understands the statistical method used to analyse the data.

Dr KSMT Mudambo (Supervisor)

Ms C.B. Maponga (Candidate)

DEDICATION

To mum, dad, Fred, Anisa, my husband Lovemore and son Joseph. You are all the light at the end of the tunnel.

ABBREVIATIONS

Albuminuria (g/litre)	(Alb)
Arginine vasopressin	(AVP)
Atrial natriuretic peptide	(ANP)
Brain natriuretic peptide	(BNP)
Carbohydrate-electrolyte solution	(CE)
Delayed onset muscle soreness	(DOMS)
Heart rate	(HR)
Maximum voluntary isometric contraction	(MVIC)
Percent (%) temperature increase (°C)	(Temp)
Percent (%) dehydration	(Dehy)
Skin temperature	(Tsk)
Stroke volume	(SV)
Sweat rate (litres/min)	(SwR)
Sweat loss (litres)	(SwL)
Urine output (litres)	(UrO)
Urine flow rate (litres/min)	(UrFR)
Water intake (l)	(WI)

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CHAPTER 1: INTRODUCTION

Physical and cognitive performance when playing soccer in hot environments may be affected by dehydration and hyperthermia resulting from excessive sweating without adequate fluid replacement. Thus, the outcome of the soccer game played in hot environments, may be determined by the state of acclimatization and hydration of the two teams. In Zimbabwe, premier soccer league and first division teams come from both hot and cooler regions for example, Hwange Football Club resides in a hot region [range 18° - 42°C] while several teams such as Harare United reside in a much cooler Harare region [range 7° - 32°C]. Over the years, Hwange Football Club has always survived relegation by winning most of its home games normally played during the hot part of the day (from 1500 hours). Most teams have found it difficult to adapt to the high temperature conditions and maintain high levels of performance during these games resulting in poor performance and defeat. However, it is not known how the various environmental conditions encountered in Zimbabwe impact on the performance and hydration status of acclimatized and unacclimatized players coming from a cooler region to play in a hotter region, and how the players from the cooler region adapt when exposed to hotter environmental conditions without prior acclimatization.

LITERATURE REVIEW

1.1 The game of Soccer

Soccer is a 90 minutes endurance sport with varying margins of intensities. There are two halves in the game with a 15 minutes break in between. Due to these differences in intensities during the game, short break and length of game, fatigue tends to affect players especially during the last quarter of the second half of the game. This is likely to impact on the cognitive and physical performance of the players and consequently the outcome of the game. During exercise in the heat, there is a depletion of substrate stores (muscle glycogen) and thermoregulation associated problems including fluid and electrolyte imbalances (Maughan *et al.*, 1994, Mudambo and Reynolds, 1996a, Pandolf *et al.*, 1994). Fluid and electrolyte loss is mainly through high sweat rates especially among the unacclimatized players. Because a significant portion of total body water loss is derived from the vascular space, the resulting reduction in plasma volume and consequently thermoregulatory function (Harrison 1985, Pandolf *et al.*, 1994, Rowell 1974, Wyndham 1976) may contribute to fatigue (Mudambo 1996b). As suggested by Maughan *et al.*, (1994), fatigue occurs due to thermal regulation-associated problems, electrolyte imbalance, a fall in plasma volume and substrate depletion (Mudambo *et al.*, 1996).

1.2 Hydration and Blood Volume adjustments during exercise

When performing any strenuous activity in hot environments, adequate hydration is required. It has been documented that cardiac output can be reduced if adequate fluid intake is maintained when exercising and that, it can be reduced more during high intensity exercise

compared to low intensity exercise (Montain *et al.*, 1998). The decrease in cardiac output may be a result of a decrease in the blood volume ejected by the ventricles of the heart during one heart beat which is known as the stroke volume (SV). According to Ganio *et al.*, (2006); Pandolf *et al.*, (1994) Harrison (1974, 1985) and Wyndham *et al.*, (1969), the progressive decline in SV during prolonged, constant-rate submaximal exercise in a warm environment, is reflective of an increased cardiovascular strain associated with hyperthermia, dehydration, and other changes that occur over time, thereby reducing the amount of oxygen one takes per minute per each kilogram of his body weight (ml.kg.min^{-1}) which is the $\text{VO}_{2\text{ max}}$. Therefore, fluid ingestion during prolonged strenuous exercise improves performance (Mudambo *et al.*, (1996a, 1996b, 1997, 1999); Sawka and Pandolf 1990), in part, by mitigating the decline in SV and its determinants, and preserving $\text{VO}_{2\text{ max}}$.

The reduction in the stroke volume is generally attributed to the decline in central blood volume and cardiac filling which results from increased skin blood flow and volume (Johnson and Rowel, 1975; Rowel 1974, 1986). Gonzalez *et al.*, 2000, in their work with euhydrated subjects, however reported that the reductions in SV was not associated with increases in skin blood flow, but was highly associated with increased heart rate and reduced blood volume

The problems of fatigue and thermal regulation are all due to reduced blood volume as suggested by Gonzalez-Alonso *et al.*, (1999), Mudambo *et al.*, (1996b), Sawka and Pandolf (1990); Pandolf *et al.*, (1994); Rowel (1974); Nadel (1992) and Harrison (1985). Blood is the medium of transport for glucose, the energy source, and electrolytes. According to Gonzalez-Alonso *et al.*, (1999), the effect of dehydration in causing a reduction in exercising muscle

blood flow does not impair either the delivery of glucose and free fatty acids or the removal of lactate during moderately intense prolonged exercise in the heat, they have suggested that dehydration during exercise in the heat causes an elevation of carbohydrate oxidation and lactate production.

The reduced blood volume, besides causing a reduction in central venous blood (stroke volume), also causes reduced muscle blood flow. The body compensates by increasing the heart rate and blood pressure to the central venous circulation and muscle areas mentioned above (haemodynamic response) (Gonzalez-Alonso *et al.*, 1997, 2004). This mechanism is regulated by the Central Command and Exercise Pressor Reflex (Muscle Pump) (Raven *et al.*, 1997). If this system fails to compensate, this leads to a reduction in cutaneous blood flow. These shifts in thermoregulatory adjustments results in the risk of an increase in body temperature and consequently, lethal heat stress related conditions in which the elevated temperatures become destructive to tissue cells like the brain cells. This destruction results in multiple symptoms, like muscle weakness, exhaustion, headache, dizziness, nausea, profuse sweating, confusion, staggering gait, collapse, and unconsciousness and if not treated can result in death. (Guyton and Hall 2000)

In a study by Rico-Sanz *et al.*, (1996) on elite soccer players, it was suggested that additional water intake in heat-acclimated players can increase body water reserves and improved temperature regulation during a soccer match with no significant effect on the decrement in soccer specific performance observed at the end of a soccer match. Associations made by Cleary *et al.*, (2006) have shown a relationship between dehydration and delayed onset

muscle soreness (DOMS), a clinical model of muscle damage consisting of muscular pain and other symptoms experienced 24 hours to 48 hours after novel or intense exercise. The signs and symptoms of DOMS include dull, diffuse pain and tenderness; stiffness; swelling; and decreased strength of the exercised muscle (Fitzgerald *et al.*, 1991, Cleary *et al.*, 2006). The association is based on the idea that when exercising, there is loss of water through sweating to cool the body and to maintain sweating there is an intracellular shift of water and electrolytes to the extracellular space, resulting in cell dehydration and this can adversely affect skeletal muscle function (Sawka 1992). This leads to an interference of the actin-myosin cross-bridge formation (Hargreaves and Febbraio 1998) during excitation contraction coupling resulting in DOMS. Although the association is not conclusive, it has been noted that when the body loses 2.7% of its weight but gets adequate time to rest and rehydrate there is no experience of DOMS (Cleary *et al.*, 2006).

1.3 Core Temperature and thermal adaptation during exercise

The hypothalamic centres in the brain control the body temperature regulatory mechanism. The body transfers heat in four ways; conduction, convection, radiation and evaporation of insensible sweat (unnoticeable sweat) and sensible sweat. To date, no published data exists on the method of measuring core temperature in areas like the brain, but it has been noted by Deklunder *et al.*, (1991) that there is no experimental evidence of a counter current exchange in the vascular bed of the human head that is efficient enough to cool the whole body.

Nielsen *et al.*, (1982) stated that during dynamic exercise, core temperature can rise rapidly and the high internal body temperatures seem to be an independent cause of fatigue during exercise in hot environments. In hot environments, heat acclimation, which is the increase in heat tolerance level while physically working under stress conditions in the natural environment (Armstrong and Maresh, 1991; Nielsen, 1994) can result in an improvement in performance. The physiological adaptations of heat acclimation include improved cardiac output with lowered heart rate (HR), together with increased SV, sweat rate (SwR) and blood plasma volume, decreased core temperature and mean skin temperature (Tsk) (Wyndham *et al.*, 1976), rectal temperature at rest (Buono *et al.*, 1998) and oxygen consumption at a given work rate, earlier onset of sweating during exercise and decreased sodium chloride losses in sweat and urine (Armstrong and Maresh, 1991). A reduction in resting metabolic rate is suggested to be responsible for reducing resting rectal temperature with heat acclimation (Shvartz *et al.*, 1973; Buget *et al.*, 1988). Heat acclimation has also been suggested to significantly reduce rectal temperature by approximately 0.3–0.5°C at rest, and the effect of a decrease in rectal temperature at rest after heat acclimation, would be to reduce rectal temperature at any given point during exercise in the heat, if the same amount of heat was generated (Saat *et al.*, 2005).

Air temperature, humidity, air velocity, radiation and clothing are factors that affect heat stress/heat load. According to Nadel (1977), exercise induced increase in metabolic heat load considerably challenge temperature homeostasis and may ultimately impair physical work performance (Knochel, 1975). This has been disputed by Nielsen *et al.*, (1982) who stated that exercise-induced hyperthermia does not impair ability of muscles to generate force, but the

sustained force production is lowered as a result of a reduced neural drive from the central nervous system. Nybo *et al.*, (2001) attributed the impairment of high intensity exercise performance when there is dehydration and hyperthermia to be mainly related to the effects of hyperthermia on reducing $\text{VO}_{2\text{ max}}$.

The failure of the heat dissipating mechanism results in thermal regulatory conditions such as heat cramps (Mudambo *et al.*, 1996b, 1997), heat exhaustion (Hubbard *et al.*, 1995; Moore *et al.*, 1992), heat stroke and is likely to occur in those in the high risk category such as the obese, unfit, dehydrated, unacclimatized, those who have had a previous history of heat stroke, ill people, children and the elderly Gonzalez-Alonso *et al.*, (1999); Pandolf *et al.*, (1994) noted that more than one-half of the metabolic heat liberated in the contracting leg muscles is dissipated directly to the surrounding environment. The observation by Gonzalez-Alonso and co-workers (1999) indicated that hyperthermia, rather than altered metabolism, is the main factor underlying the early fatigue associated with dehydration during prolonged exercise in the heat. To prevent dehydration from occurring, proper clothing is required, fluid intake must be frequent and there may be the need to cancel the event if the wet bulb thermometer temperature is high. Under these conditions, acclimatization is required and an excuse from training if there is a weight loss greater than 2% in 24 hours. Acclimatization results in a decreased heart rate and core temperature with training and this may need to be done for 1 hour a day for 5 to 10 days. Changes in sweating mechanisms generally take up to 10 days.

1.4 Urine output during exercise and proteinuria.

During exercise, urine output is reduced because of an increased need for blood flow through the exercising muscles, necessitating a decrease in blood flow in the splanchnic and renal circulations. As a result of exercise, the body adjusts its fluid compartments when there is hypervolemia, which is characteristic of acute responses to the exercise stimulus. The stimulus, described as a selective expansion of plasma volume within 24-48 hours after exercise, is a chronic adaptation identified as a general expansion of the extracellular fluid compartment (Gillen *et al.*, 1991; Nagashima *et al.*, 2000; Maw *et al.*, 1996). The expansion of the plasma volume has been attributed to the increase in protein synthesis resulting in the increase in the plasma protein concentration in particular plasma albumin (Gillen *et al.*, 1991; Nagashima *et al.*, 2000). The increase in the plasma albumin can result in the expansion of the extracellular compartment i.e. an increase in the oncotic pressure, which has an affinity for water resulting in an increase in the water retained by the extracellular compartment. This increase in the extracellular compartment increases the pressure exerted by the compartment on the blood vessels, thereby increasing the pressure within the renal capillaries and therefore water, electrolytes, salts and proteins are filtered through. The amount filtered may exceed the transport maximum (T_{max}), i.e. the maximum amount that can be reabsorbed by the nephrons, the functional unit of the kidneys, of which albumin is reabsorbed in the proximal part of the nephron and under normal conditions there may be only very low excretion of polypeptides >750 Da in normal human urine (Norden *et al.*, 2004).

Protein can be found in urine (Poortmans *et al.*, 1988; Norden *et al.*, 2004; Ümit Kemal 2007), of which the albumin makes up the greatest contribution to the increase in exercise-

induced proteinuria (Clerico *et al.*, 1990). The causes of this vary, of which acute increase in blood pressure has been implicated in the magnitude of increase in urinary protein excretion. Cantone and Cerretelli (1960) and De Palo *et al.*, (2003) have attributed the presence of protein in urine during short and exhaustive exercise to haemoconcentration. It has also been shown in a study by Poortmans *et al.*, (1988) and De Palo *et al.*, (2003), that several amino acids inhibit tubular protein reabsorption and their work further demonstrated that post-exercise proteinuria is of mixed type after exhaustive short-term exertion. Proteinuria can be of benign causes such as fever, intense activity or exercise, dehydration, emotional stress and acute illness (Ganong; 2000). Serious causes of proteinuria include glomerulonephritis and multiple myeloma. Exercise-induced proteinuria is a common consequence of physical activity but its mechanism has not been clearly elucidated. Oxidative stress has been proposed as one of different factors involved in post-exercise proteinuria in rat models (Ümit Kemal 2007). According to De Palo *et al.*, (2003) in their study of the effects of acute, heavy-resistance exercise on urinary peptide hormone excretion in humans, physical exercise proteinuria is related to an increased protein filtration and a saturation of the mechanisms responsible for the reabsorption of which it does not appear similar for all peptide hormones. The exercise-induced proteinuria, according to Clerico *et al.*, (1990) is both glomerular and tubular in origin, and is reversible.

1.5 Cognitive Performance and injury during exercise

Cognitive performance is affected by the failure of the heat dissipating mechanism, electrolyte imbalances and neuroglycopenia due to a fall in blood glucose concentration (Mudambo, 1996b). Fatigued individuals are more likely to be tense, angry (combative, aggressive),

confused, vigour, depressed and fatigued during the duration of the game and this may persist during the post-game phase leading to delayed recovery. Visual acuity may also be affected as the photoreceptors are affected by reduced oxygen and glucose supply. In sporting events such as running and cycling, it is easy to demonstrate the effects of the duration of an event, the ambient temperature and humidity on thermoregulation and dehydration and the onset of heat injury. In soccer however, the game is played over 90 minutes of 45-minute halves with a 15-minute break in between. The 90 minutes of exercise at mixed intensities experienced in a game of soccer, is likely to change the thermoregulatory and cognitive capacity of the players. Morris *et al.*, (2005) observed in their work on unacclimatized sportsmen, that the earlier onset of exhaustion during prolonged intermittent shuttle running in the heat is associated with hyperthermia, and that while muscle glycogen utilization may be elevated by heat stress, low whole muscle glycogen concentrations do not seem to be the cause of this earlier exhaustion. In a game of soccer, exhaustion may result from a combination of several factors of which, low muscle glycogen, neuroglycopenia (Marriot 1994; Mudambo *et al.*, 1993, 1996b) hyperthermia, dehydration and this may result in impaired cognitive and visual capacity

Ambient temperatures higher than skin temperature result in a net gain of heat adding to the body's temperature. The only efficient mechanism of heat loss is through sweating (Maughan *et al.*, 1994) which results in dehydration, reduced blood volume and cardiac output. In cup games, if there is no winner at the end of the 90 minutes, an extra 30 minutes of 15-minute halves is played to decide the winner. It is during the 30 minutes extra time that problems of

fatigue, cramps, injuries and decreased cognitive performance and combat behaviour are likely to occur and be high.

1.6 Sweat rates during exercise

Sweating is a mechanism that aids in heat loss from the body and it depends on the climatic conditions, clothing worn and exercise intensity (Shapiro *et al.*; 1982). Athletes exercising vigorously in the heat often have sweat rates of 1.0 -2.0 litres/hour (Costill; 1977). The average sweating rates (SwR) of American footballers wearing equipment and practicing in the heat are consistently over 2.0 litres per hour (Godek *et al.*, 2005). The average SwR for male marathon runners is 1.71 litres per hour (Milliard-Stafford, 1995) under hot environmental conditions. In soccer players, sweat rate is averaged between 1.2 (Broad *et al.*, 1996) and 1.67 (Maughan and Leiper 1994) litres per hour. Broad *et al.*, (1996) reported a sweat rate of 1.6 litres per hour in basketball players competing indoors in ambient temperatures of 20°C, and Pyke and Hahn (1980) reported SwR averaging 1.8 litres per hour in rugby players competing in ambient temperatures of 38°C. During military training in the heat, Mudambo *et al.*, 1996b observed sweat rate of 2 – 4 litres/hour. The rate of sweating may depend on by the following factors: ambient temperature, humidity, air movement, exercise intensity, state of hydration, state of acclimatization, level of fitness, insulating clothing or equipment, and body size.

The exercise intensity of a game may be determined by the team's need to win and the competition/resistance that their opponents may offer. According to Hatch (1963) and Leithead and Lind (1964) sweat loss is one single physiological measurement that could be

used as an index of physiological strain. The higher the intensity, the greater the sweat rate, in particular if there is still adequate cutaneous blood flow. As the body becomes dehydrated, there is hyperosmolality (Mudambo *et al.*, 1997; Ramsay 1989) and hypovolaemia (Harrison 1986; Rowell 1974). This results in the induction of the thirst sensation and vasopressin secretion (Robertson and Athar, 1976), and also suppression of the thermoregulatory responses, including cutaneous vasodilatation and sweating (Nadel *et al.*, 1980; Fortney *et al.*, 1984; Takamata *et al.*, 1997, 1998). Since these responses disappear after recovery of plasma volume and/or plasma osmolality (Montain and Coyle, 1992), baroreflex and osmosensitive mechanisms may be involved. Skin blood flow tends to increase during exercise unlike at rest (Rowell, 1986) because of haemodilution, which may assist in the maintenance of cardiac output and offset any effect of fluid loss through sweating on plasma volume. Yoshi-Ichiro *et al.*, (2005), in their work with non-smoking exercising males, noted that the oropharyngeal stimulation by drinking, released the dehydration-induced suppression of cutaneous vasodilatation and reduced Mean arterial pressure during exercise, and this is accelerated when plasma volume is restored. This observation emphasizes the need for a continuous supply of water to maintain the body homeostasis of the body temperature and reduce the sweat rate, dehydration, hyperosmolality and hypovolaemia. Several workers (Maughan *et al.*, 1994; Mudambo 1996b; Murray 1997 and Naufer *et al.*, 1989) have emphasized the need for regular rehydration and the importance of consuming carbohydrate-electrolyte solutions, which have the capacity to replace water, energy and electrolytes. This decreases the chances of dehydration, energy depletion, and hyperthermia and maintains performance.

Sweat is known to be hypotonic compared to the extracellular fluid (Sawka and Montain 2001, Widmaier 2006) composed mainly of sodium and chloride, and include minor electrolytes such as potassium, calcium and magnesium. Heat acclimation tends to reduce sweat sodium and chloride concentrations (Sawka and Montain 2001). The mechanism by which the sodium-conserving effect of the heat acclimation is dependent on the adrenal cortex and the hormone aldosterone (Sawka *et al.*, 1996). It has been observed that aldosterone concentrations in plasma increase early during acclimation (Francesconi *et al.*, 1983), from dehydration (Francesconi *et al.*, 1985), during high-intensity exercise (Montain *et al.*, 1997) and after a low salt intake (Francesconi *et al.*, 1993). This has the effect of reduced sodium concentration in the sweat and the urine, therefore, this may reduce the urine output and moderate the loss of water through sweating.

1.7 Objective

The objectives of this study were

- To determine the relationship between water intake, urine output and dehydration as a result of playing soccer.
- To assess the effect of ambient temperature on dehydration as a result of playing soccer.
- To relate core body temperature increase to water intake, dehydration as a result of playing soccer.

1.8 Hypothesis

The hypothesis was that exposure of soccer players to high ambient temperatures during the game can affect total body water and associated effects such as hyperthermia when not consuming adequate amounts of water. This is likely to reduce their physical performance and influence the outcome of the game.

CHAPTER 2: MATERIALS AND METHODOLOGY

2.1 Subjects

Subjects were sixty six football players (17- 32 years of age), from three teams of the Zimbabwe Premier Soccer League: Hwange Football Club from a hot region; Buymore Football Club from a cooler Harare region; and Highlanders Football Club from a moderately hot region of Bulawayo. One First Division team Harare United from Harare was included into the study. Sixty-one players completed the study protocol.

Inclusion criteria: Subjects were included if they played the full 90 minutes of the game.

Exclusion criteria: Subjects who were substituted during the game or came on as substitutes were excluded from the study. All of the 11 players of the Hwange team, and 10 each from the rest of the participating teams volunteered to take part in the study. Measurements were taken during league games between the participating teams. To evaluate the effect of playing soccer, the subjects did not get verbal or visual feedback from the researchers during the game so that the usual pacing strategies during the games are maintained if they can be highly reproducible when the testing conditions are identical each time when the experiment is repeated on all the subjects (Tucker *et al.*, 2004; Schabert *et al.*, 1998).

2.1.1. Ethics

The study proposal was approved by the ZIFA Sports Medicine Committee. On approval, a written request was made to the teams' management on the importance and benefits of the study and its relevance to performance. The clubs and players were informed in writing and verbally on what parameters would be measured, what samples were to be collected on the

day of the game and what possible parameters in the samples would be measured. All the subjects were informed on the purpose, aims, benefits and risks of the study. The subjects were also informed that they were free to withdraw from the study without any adverse consequences and that they were free to refuse to give samples if they wished in accordance with the Helsinki Declaration of 1975. All the subjects gave their informed consent to the ZIFA Sports Medicine Committee before the study was approved.

2.1.2 Location

The subjects who volunteered to take part in the study were drawn from Bulawayo, Harare and Hwange based teams.

2.2. Methodology

2.2.1. Measurements

2.2.1.1 Body Mass Changes

Body weight, with minimum clothing was measured before and after the game upon emptying their bladder in order to determine possible water loss due to sweating and insensible loss. The subjects were asked not to drink water after the game before being weighed and the monitoring of this activity was carried out by the team doctor and the assistant researcher. A digital scale accurate to ± 0.05 kg (UC-300 kg, A.N.D., Toshima-ku, Tokyo, Japan) was used to measure the body weight according to Mudambo 1996b. The difference between the pre-game weight change and the post-game weight was used to estimate the percentage dehydration. Dehydration was calculated according to Saat *et al.*, 2005:

$$\text{Percent Dehydration (\%)} = \frac{[\text{pre-exercise BW} - \text{post-exercise BW (kg)}]}{(\text{Pre-exercise BW})} \times 100$$

(BW=body weight)

2.2.1.2 Body Temperature Changes

Core temperature was measured in the changing rooms 10 minutes before the game (resting body temperature) and immediately after the game within 5-10 minutes of leaving the soccer pitch. Temperature was measured using digital thermometers (Microlife SE 0.1°C). The temperatures were used to calculate percent temperature changes [ΔT (%)]:

$$\Delta T (\%) = \frac{[\text{pre-exercise BT} - \text{post-exercise BT (kg)}]}{(\text{pre-exercise BT})} \times 100$$

ΔT (%) = percent temperature change

(BT = body temperature)

2.2.1.3 Water intake.

Water intake was estimated from every drink the players took throughout the game and at half time *ad libitum*. Water bottles in ice supplied by the team management were placed at the team bench and all around the touchline of the soccer field. The players were informed of the water points and that they could take the water at any time during the game. Each time a player took water, the research assistants placed strategically around the football ground recorded the number of times and volumes consumed. Each mouthful of water was estimated

to be 0.15 litres. At the end of the game, each research assistant provided the total amount of water consumed by each player. This was reconciled by a questionnaire given to the players at the end of the game on which they stated the number of times they took water during the game. Information on water including any other sports drink taken 1 hour before the game was also obtained from the questionnaire. The FIFA regulations now allow and recommend players to take drinks during the course of the game.

2.2.1.4 Urine collection and calculation of urine output and flow rate.

Before the game, each subject was asked to empty his urinary bladder and the volume recorded. If a player required to empty their bladders during the game, they were instructed to use the urine containers supplied and the time they had urinated was recorded (very rarely do players empty their bladders during the game). At the end of the game a urine sample was collected and the volume recorded. All urine samples were put on ice and transported back to the laboratory at the University of Zimbabwe Physiology Laboratory where each subject's urine volume was accurately measured, verified, and analysed. The urine samples were used to calculate the urine output, urine flow rate (Godek *et al.*, 2004, 2005 Physiology Practical Schedule 2002), urine colour (Mudambo 1996b) and to assess the presence of protein in the urine sample using dip sticks for the duration of the game.

2.2.1.5 Sweat Rate.

The change in body weight, water intake and urine output was used to calculate the sweat loss (SwL) and sweat rate (SwR) using the following formulae (Casa *et al.*, 2002; Godek *et al.*, 2005):

$$\text{SwL} = (\text{pre-exercise BW} - \text{post-exercise BW} + \text{fluid intake} - \text{urine volume})$$

$$\text{SwR} = \frac{(\text{pre-exercise BW} - \text{post-exercise BW} + \text{fluid intake} - \text{urine volume})}{\text{Exercise time in hours}}$$

(SwL = Sweat loss SwR= Sweat Rate)

This calculation does not take into account glycogen and associated water loss during the game.

2.2.1.6 Ambient temperature and humidity.

All games started at 3 pm which is the standard time used by all the major leagues in Zimbabwe for games to begin in the afternoon unless otherwise stated. Ambient temperature and humidity were obtained through communication with the Harare National Meteorological centre as carried out before by Mudambo *et al.*, (1996a, 1996b, 1997). No rain or showers were recorded during any of the games.

2.2.1.7 Estimated water intake.

Estimated fluid replacements were calculated as 130% of the fluid lost through sweating during the game. This was based on current research and fluid replacement recommendations and guidelines, which suggest the replacement of 25–50% more than sweat losses to account for obligatory losses in urine, respiration, and sweating (Godek *et al.*, 2005, Casa *et al.*, 2002, Shirrefs *et al.*, 1996). Fluid replacement after the game was calculated as (Godek *et al.*, 2005):

$$1.30\% \times \text{sweat loss during exercise (litres)} - \text{fluid consumed during game (litres)}.$$

2.2.2 Measurements during Game

Two groups of research assistants composed of 3 members each were assigned to each team to support collection of samples and taking measurements during the game. There was an average of one substitution per team when playing in cooler conditions of Harare and an average of 2 substitutes when playing in the heat of Hwange.

2.2.3 Statistical Analysis.

SPSS statistical package was used for statistical analysis. All data were presented as means $\pm$ Standard deviation and Pearsons' correlation was conducted to derive any relationship between variables ($P < 0.05$ and $P < 0.01$). To determine the significance of differences between the environments where the games were played, dehydration, core temperature change, sweat rate, sweat loss and water intake during the game, one way analysis of variance was used. A regression was conducted to evaluate the significance of each variable in determining dehydration and also in determining core temperature increase ($P < 0.05$). All values were

reported as Mean $\pm$ SD [range] unless otherwise stated. Statistical significance was pegged at the 0.05 level or 0.01 level.

CHAPTER 3: RESULTS

A total of 63 football players, mean age 23 ± 3 [19-26] years out of a possible 66 were measured during the study. 61 were included in the study as substitutions took place during the games. All (11) of the regular first team players in the Hwange team were measured on all three occasions. Highlanders had a total of 10 subjects who participated, Harare United had 10 and Buymore United 10 subjects. Table 1. shows the group mean values of the parameters measured and the outcomes seem to show the following:

Table 1. Mean values of measured variables

N	Dehy	Temp	WI	Pr-UrO	UrO	UrFR	Alb	SwL	SwR
61	2.21	1.79	0.28.	0.210	2.4×10^{-2}	2.8×10^{-4}	1.2	1.58	0.84
	± 0.83	± 1.12	± 0.23	± 0.029	$\pm 1.5 \times 10^{-2}$	$\pm 1.6 \times 10^{-4}$	± 1.4	± 0.71	± 0.64

Results are mean $\pm$ SD: Temp- % Temperature increase ($^{\circ}$ C), Dehy- % Dehydration, WI- Water intake litres , PrUrO- pre-game Urine output (litres), UrO- urine output (litres), UrFR- Urine flow rate (litres/min), Alb- Albuminuria (g/litre), SwL- Sweat loss (litres), SwR- Sweat Rate (litres/hour).

3.1 Dehydration.

The pregame mean body weight of the players was 71.3 ± 6.1 kg and post game mean body weight was 69.9 ± 6.1 kg. The mean loss in body weight, which was used to determine the dehydration status, was $2.21 \pm 0.83\%$.

There were significant correlations between dehydration and water intake (0.321) at $P < 0.05$, percentage temperature increase (0.381) and sweat loss (0.934) at $P < 0.01$

3.2 Thermal responses.

Calculations on Core temperature showed significant increases within groups/teams during a game ($P<0.05$). The regression model showed that mean core temperature increase was insignificant in the environments in which the matches were played. The mean increase in core temperature was $1.79 \pm 1.2\%$. The temperature changes ranged from 0.6% (a player from Harare in a game played in Harare) to 5.8% in Hwange (a Hwange player). The percentage increases in body temperatures were characteristically similar irrespective of the location or climatic region they played in. In relation to the sweat loss and water intake, temperature change had a significant correlation ($P<0.01$).

3.3 Sweat loss and sweat rate.

The mean sweat loss was 1.58 ± 0.71 litres and the sweating rate was 0.84 ± 0.64 litres per hour. There was a very significant correlation between sweat loss and dehydration, water intake, temperature increase ($P<0.01$). Albuminuria showed a weak correlation. On fitting the regression model, sweat loss was a significant predictor of dehydration and temperature ($P<0.05$).

3.4 Fluid intake.

The mean water intake was 0.298 ± 0.224 litres [range: 0 to 0.675 litres]. The 0.675 litres intake was recorded during a game in Hwange. 15 out of 61 subjects did not drink any water. There was a significant correlation between water intake and dehydration ($P<0.05$); and water intake and albuminuria ($P<0.05$) and sweat loss and temperature increase ($P<0.01$). On fitting the regression model, water intake was significant ($P<0.05$) indicating that it was a good determinant factor of dehydration. However, the results further suggested that water intake

was an insignificant determinant factor of temperature increase. Table 2 shows the mean water intake compared to the mean percentage temperature and mean percentage body mass change of each team.

Table 2. Mean water intake per team, percentage temperature increase and percentage body mass change

Team	Average water intake (litres)	%Temp increase	%Dehydration
A (Hwange)	0.150	1.4	1.98
B (Hwange)	0	1	1.7
C (Buymore)	0.500	2.7	3.5
D(Harare UTD)	0.214	1.2	1.43
E (Hwange)	0.539	2.97	3
F (Highlanders)	0.325	1.45	1.97

3.5 Urine output and flow rate.

The urine flow rate and urine output of the players were low $2.8 \times 10^{-4} \pm 1.6 \times 10^{-4}$ litres/min and $2.4 \times 10^{-2} \pm 1.5 \times 10^{-2}$ litres respectively. 7% failed to produce any urine sample. There was an insignificant negative correlation between urine output and dehydration, percentage temperature increase, albuminuria and sweat loss ($P < 0.05$). In the regression model, the urine output was a significant predictor of dehydration ($P < 0.05$) but it was not significant in the prediction of increase in core temperature. Visual inspection of the urine samples showed presence of haematuria and albuminuria, sediments such as salts which had settled at the bottom of urine containers and a deep coffee colour (heavily concentrated) urine. Observation

of urine colour showed that footballers from Hwange, who reside in the hot region, produced less concentrated urine compared to players from Bulawayo, moderate to hot region, and Harare, a cooler region suggesting that Hwange players were acclimatized to hot conditions. The urine colour intensities on visual inspection were graded from deep coffee, dark brown to light yellow respectively for Harare, Bulawayo and Hwange players. No subject passed urine during the 90 minutes of the game and in the post-game period, very few players produced a urine sample.

3.6 Albuminuria.

The average albuminuria of the players was 1.2 ± 1.4 g/litre. There was a significant correlation between albuminuria and water intake ($P < 0.05$). However, insignificant negative correlations between albuminuria, and urine output (-0.113) and dehydration (-0.065) were observed. In the regression model, albuminuria was an insignificant predictor of dehydration/increase in core temperature.

3.7 Ambient Temperature and relative humidity.

Table 3. below shows the different ambient temperatures and relative humidity of the locations in which the teams played. The ambient temperatures and relative humidity were insignificant predictors of dehydration and core temperature increase ($P < 0.05$).

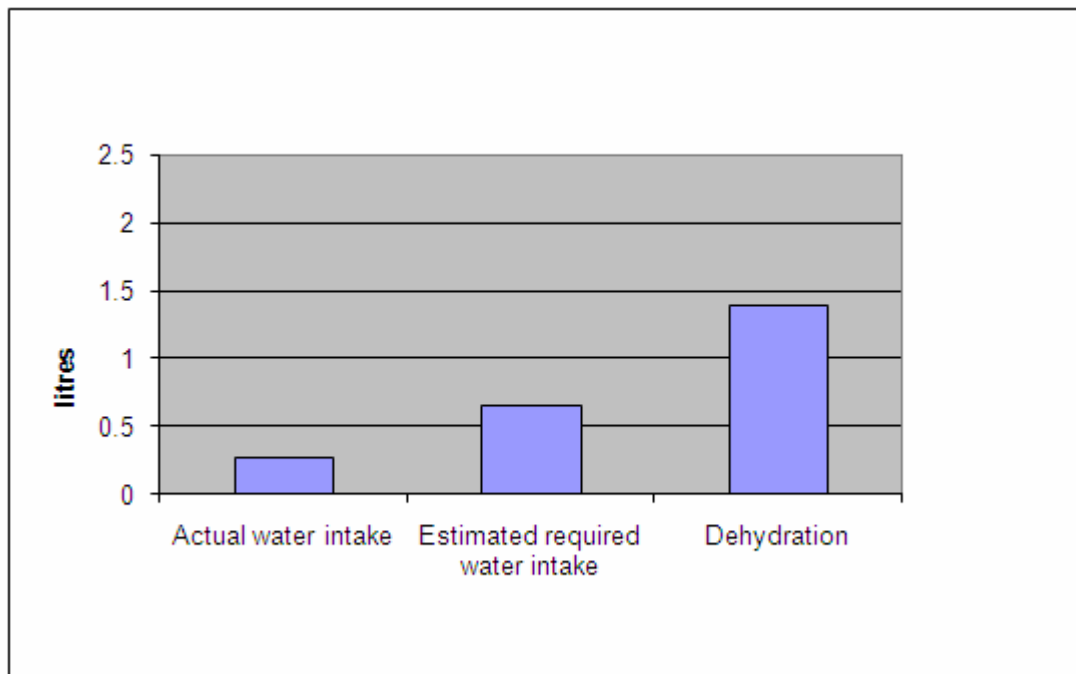
Table 3. Ambient heat stress components during each teams's game in cool, hot and humid environment

Team	Location	Ambient Temperature	Relative humidity
A (Hwange)	Hwange	27	63%
B (Hwange)	Harare	21.8	65%
C (Buymore)	Harare	21.8	65%
D (Harare UTD)	Harare	22.3	56%
E (Hwange)	Hwange	31.4	60%
F (Highlanders)	Hwange	31.4	60%

3.8 Estimated fluid replacement.

Fig 1. shows the actual water consumed, estimated required water intake and the level of dehydration that had occurred. The estimated required water intake required to return to normal hydration status was calculated to be approximately 0.654 ± 0.5 litres.

Figure 1: Comparison of average actual water intake, average estimated required water intake and the average extent of dehydration



3.9 Substitution and Outcomes of game

Hwange FC played three games during which a total of 32 players were measured. During the two games that Hwange played, they did not use any substitutes at all, while the other teams (Black Rhinos, Buymore, Highlanders, and Harare United) used an average of two substitutes per game.

Table 4 shows the outcomes of games played by the teams relative to temperature change and body mass changes.

Table 4. Game outcomes, mean percentage temperature increase and mean dehydration per team

Team	Team	Home/	%Temp	%	Outcome	Scores
	Played	Away	increase	Dehydration	of game	
A (Hwange)	Black Rhinos	Home	1.4	1.98	Loss	0-2
B (Hwange)	Buymore	Away	1	1.7	Win	1-0
C (Buymore)	Hwange	Home	2.7	3.5	Loss	0-1
D(Harare UTD)	Kambuzuma UTD	Away	1.2	1.43	Win	1-0
E (Hwange)	Highlanders	Home	2.97	3	Loss	0-3
F (Highlanders)	Hwange	Away	1.45	1.97	Win	3-0

There was a strong positive correlation between a negative outcome of the game and the percentage body temperature increase (0.75) at $P < 0.01$ and the negative outcome of the game and the extent of dehydration (0.76) at $P < 0.01$. Only one red card was recorded in all the games and no yellow cards were recorded in all the games.

CHAPTER 4: DISCUSSION

From available information, it appears that this investigation is probably the first to examine sweat loss, sweat rate, body weight loss, water intake and urine output and compare changes in these parameters with the game's outcome, dehydration, physical and cognitive performance and acclimatization when playing soccer in both cool and hot conditions in Zimbabwe. An important finding of the present study is the significant correlations between dehydration and water intake ($P < 0.05$), sweat loss and core temperature increase ($P < 0.01$) suggesting that the players were dehydrated. The dehydration observed during the study is further confirmed by the failure of 7% of the subjects to produce a urine sample at the end of the game. These results therefore, indicated that the players do not take adequate water before, during and after the game and therefore, they stay with some degree of dehydration. Dehydration impacts on performance and work output as suggested before by (Mudambo 1996b, Mudambo et.al 1997 and Mudambo, 1999) in soldiers doing physical training in the heat. Thus, playing football in the heat when dehydrated could influence the game outcome in situations where one team is euhydrated and the other dehydrated, and also endangers the health of the players who are likely to suffer from heat cramps, heat injury and sometimes death (Knochel, 1975 and Mudambo, 1996b).

The mean group dehydration due to sweat loss was 2.21% of body weight which may have contributed to decrements in performance during the second half of the game for teams visiting Hwange. This observation is supported because the teams visiting Hwange used an

average of 2 substitutes but Hwange did not use any substitutes at all. Thus, the Hwange players acclimatized to hot conditions were able to finish the 90 minutes with no substitution. The results further showed sweat loss, urine output and water intake were significant predictors of dehydration. On predicting temperature increase, sweat loss was the only predictor.

The study showed the following significant results:

4.1 Core temperature increase.

The mean increase in core temperature was calculated to be 1.79 ± 1.2 % [range 0.6% to 5.8%]. The highest percentage temperature change (5.8 %) was observed in Hwange players when the game was played in Hwange where the ambient temperature was 31.4 °C and in this game Hwange Football Club lost to the team from a moderately cool climate. This result could be explained by the ability of Hwange players to tolerate higher temperature increases, have lower dehydration and temperature increases and still continue to play suggesting the importance of acclimatization. The Hwange players could therefore play at a higher intensity while players from the cooler environment would be severely affected by the heat, tire out and get substituted due to decrements in performance. Although Watson *et al.*, (2005), suggested that when bupropion, a dual dopaminergic and noradrenergic reuptake inhibitor was introduced, subjects cycled faster in a warm environment which showed an increase in performance, we did not measure dopamine and norepinephrine in the current study. Dopamine and norepinephrine have been implicated in increased stimulation and performance and an increase in core temperature. In the match in which percentage temperature increase

was highest, Hwange lost the game probably due to the psychological disadvantage that they were playing against Highlanders who were performing much better than most teams and would lift the league cup if they won this encounter. Under the prevailing conditions, Hwange would have been expected to have lower temperature changes than the observed, this however, could have been influenced by the motivational factor which influenced the temperature outcome. Another factor that could have influenced the outcome was that Hwange was highly motivated to get a result since they were playing at home and fighting to maintain the third place on the Premier Soccer League log. There was a significant correlation between core temperature increase and dehydration, sweat loss and water intake, although on fitting the regression, temperature was insignificant as a predictor of dehydration which reflects that temperature change may not be the only cause of dehydration but it may be an outcome of dehydration considering that there is a strong correlation between both variables (McMorris *et al.*, 2008). The ability of individuals to tolerate heat strain might be affected by their prior state of acclimatization, fitness level and hydration state, the sudden onset of extreme fatigue is a frequent occurrence even in well-trained athletes (Cade *et al.*, 1992, Wyndham and Strydom 1969) and psychological impact of the progression of the game. Cheung and McLellan, (1998) concluded in their study on heat acclimatization that exercise-heat tolerance in an uncompensable heat-stress environment is not influenced by short-term heat acclimatization but is significantly improved by long-term aerobic fitness. Thus, the short-term exposure of teams from cooler regions to the hot environment of Hwange is inadequate in imparting the benefits of acclimatization. This was also observed by Saat *et al.*, (2005) in their investigation on the effects of a short term aerobic training program in a hot environment on thermoregulation, blood parameters, sweat secretion and composition in

tropic-dwellers who have been exposed to passive heat. These workers suggested that tropic-dwelling subjects although exposed to prolonged passive heat exposure, were not fully heat acclimatized. While this may apply to sedentary individuals, subjects engaged in prolonged strenuous exercise become acclimatized because of improved thermoregulation mechanisms.

The teams with low temperature changes had a favourable outcome and there was a strong positive correlation between the game outcome and the temperature change, meaning that preventing hyperthermia in dehydrated subjects could restore $\text{VO}_{2\text{ max}}$ and performance time by 65 and 50% respectively (Nybo *et al.*, 2001). However, this depends on the level of physical fitness which is likely to produce the desired or favoured outcome. Teams with higher levels of physical fitness are therefore better able to perform on exposure to hot conditions. This however, is further enhanced by taking water at regular intervals during the game. Nybo *et al.*, (2001) observed in their study with cyclists that the level of thermal stress experienced was high as core temperature was elevated by 1°C throughout exercise, the subjects in the current study had a mean elevation of 0.66 °C. This shows that physical fitness is important to produce maximum performance required to get a desired or favourable outcome. From the observation made on the soccer players in the current study, the highest body temperature changes were observed in the players from teams that lost compared to those that won the game and these were not as great as those observed by Nybo *et al.*, (2001). Although Hwange players originate from a hot environment, they did have a high temperature (2.97°C) change in their home game against Highlanders. Highlanders' players who come from a moderately hot environment, had a temperature change of 1.45° C and seemed to be more physically fit than the Hwange team. Thus, higher physical fitness levels may minimize

the degree of heat stress likely to affect the players. The higher heat change observed in the Hwange players suggested that the players are able to tolerate higher temperature levels because they stay in this hot region hence acclimatized. From water intake records, the Hwange players did not take adequate water before and during the game and this apart from their ability to tolerate higher temperatures as a result of acclimatization, may have influenced these increases. It has however, been observed that if water balance is not maintained, some of the advantages of acclimatization may be lost (Senay 1975, Sawka *et al.*, 1983) and this could be true of the Hwange players when they played a much physically fit Highlanders team. Work by Hasegawa *et al.*, (2006) when they looked at the combined effects of pre-cooling and water ingestion on thermoregulatory responses and exercise capacity at 32 °C and 80% relative humidity in untrained athletes, suggested that the combination of pre-cooling and water ingestion increases exercise endurance in a hot environment through enhanced heat storage and decreased thermoregulatory stress and consequently less cardiovascular strain. In contrast however, the Highlanders footballers drank 0.300 to 0.500 litres of water 30 minutes before the game at Hwange. Thus, the Highlanders players were hydrated pre-game and therefore, better able to provide sufficiently cutaneous blood flow during the game for evaporative sweat loss in the heat. Highlanders players also continued to drink water during the game, this would have helped maintain fluid volume levels and evaporative cooling water resulting in low temperature change. This observation further confirms suggestions by other workers (Costil and Saltin 1974, Coyle and Montain 1992, Mudambo *et al.*, 1997, Sawka 1983) that pre-event hydration and during event hydration helps to maintain performance and may delay the onset of fatigue. Continuous fluid intake has also been observed by Maughan *et al.*, (1994b) and Mudambo *et al.*, (1996a) to decrease the need for post-exercise re-hydration

and therefore a quicker recovery in subjects taking fluid compared with those taking little or no fluids before and during exercise.

The temperature difference among the Hwange players was higher when they played at home, probably due to the higher environmental temperature, or because they were losing the game which was becoming more intense and therefore, they were forced to play harder than they would when not losing. Pressure from the home fans also motivated them to increase the playing intensity of the game which could have resulted in a surge of catecholamine (dopamine and norepinephrine). This, over years has always been the case that visiting teams suffered heat stress compared to Hwange players who were acclimatized to the hot environment. Over the years, Hwange has also had good technical players and on the average good teams. Another factor that was observed when we were carrying out the study was that, the team had lost good players to better paying teams (Hwange coal mine was not performing well and hence team sponsorship had decreased), hence the need to change the overall game strategy by increasing the intensity of the game in the hope that the visitors would tire from heat stress. Tucker *et al.*, 2004 noted that impaired exercise performance in the heat was not a result of a limiting core temperature, but was a result of part of the central regulation of skeletal muscle recruitment, which controls the rate of heat storage, thereby preventing the development of thermoregulatory derangement during self-paced exercise in the heat. This could also explain why the temperature difference is higher in one of their games in which they lost to Black Rhinos, another Harare based team. Morrison *et al.*, (2006) observed, in their work with healthy males that, at high body temperatures of varying physical fitness, maximum force production and voluntary activation were impaired to an equal level

regardless of training status. This observation suggests that the Hwange players were acclimatized and could tolerate higher increases in core temperature than would the players from the cooler region. Despite this observation, the data obtained showed that the increase in core temperature may not be an important variable in predicting dehydration, however as suggested by Gonzalez-Alonso *et al.*, (1999) fatigue is associated with core temperature limiting exercise performance. Therefore, a footballer's performance during the game is likely to be affected by the link between dehydration, fatigue and other associated factors including core temperature increase. Considering that the mean end of game temperature was 37 °C, which was lower than that of the study by Galloway and Morgan (1997); and Nybo and Nielsen (2001) in studies on cyclists and runners. The current ambient temperature may not have been high enough to elicit higher core temperature in players who live and work in the heat. Nybo and Nielsen (2001) also observed that exercise in a hot environment (40 °C) raised the body temperature to 40 °C whilst exercise in temperate environments (18 °C) raised the body temperature to 38 °C only, which is the threshold of heat stress. These observations suggested that there is a threshold limit at which the core temperature can be increased at which point the players performance decrease and fatigue sets in. In the present study, when the maximum ambient temperature was 31.8 °C, the post-game core temperature was 37.3 °C and when the minimum ambient temperature was 21.8 °C, the post-game core temperature was 37.05 °C. This suggests that the increase in core temperature may not be a reliable predictor of dehydration since dehydration is likely to occur long before notable increases in core temperature are observed.

There was a negative correlation, although not significant, between urine output and increase in core temperature. This suggested that there was negative feedback to reduce the loss of body water through sweating, an efficient method of cooling. Selective brain cooling (SBC) may explain why their body temperature change(s) was/were lower when they played in Hwange. In work conducted by Irmaka *et al.*, (2004), it was observed that selective brain cooling seems to be a mechanism leading to adaptive craniofacial diversity observed in different geographical regions. This amplifies the importance of SBC for long-term biological adaptation, not necessarily for protecting the brain from acute thermal damage. From the conclusion by Irmaka *et al.*, (2004) on SBC, this may play a role in the player's perception of the intensity of the game, and also a mechanism for protection from heat injury during a game. The craniofacial diversity may again explain the differences in the changes of core temperature between the players from Hwange and all the other subjects who took part in the study. This is likely to have been influenced by the differences in the degree of acclimatization. To support the idea of SBC, when a comparison was done to assess the environmental factors, i.e. ambient temperature and relative humidity, it was noted statistically that environmental conditions were an insignificant factor.

Exercising subjects who have a higher post exercise core temperature increase and consume little or no water could reduce their ability to do work and consequently their ability to play a game of football due to severe decrements in performance. But, observations from the current study are that the more the water consumption the higher the dehydration and core temperature increase. This phenomenon comes to be because these players were better hydrated and were doing more work compared to the less hydrated, who are likely to slow

down by decreasing the pace. It has also been observed by Mudambo (1996c), Mudambo and Reynolds (1996a) and Sawka and Montain (2001) that water alone may not provide adequate hydration. On the other hand, the volumes of water taken during these games were very low and inadequate to prevent dehydration and temperature increases. The result would be a surge in the core temperature, severe dehydration due to loss of total body water and a decrease in the body fluid volume leading to performance decrement as observed by (Harrison *et. al*, 1981, Francesconi 1985, Mudambo, 1996b and Edwards *et.al*, 2007). Results from the present study also showed that when Hwange played in a cooler environment (21.7° C) players who did not consume any water during the duration of the game, had a high sweat loss [1.2 ±0.42 litres] compared to those who took water. It was also observed that in this particular group of individuals, they had the lowest urine output, core temperature increases and dehydration level compared to players who had drunk water during the game. The low urine output confirms that sweat rates were high and consequently, the players had a high degree of dehydration resulting from non-replacement of fluids.

An elevated core temperature reduces the maximum voluntary isometric contraction (MVIC) (Morrison *et al.*, 2004). MVIC is a measure of muscle strength. The footballers from teams that lost the game had a greater core temperature change and this can affect the cognitive performance and the ability of the player to judge properly on what next move to make. Under these conditions the players are likely to make serious mistakes that could lead to a goal being scored against them and hence, loss of the game and reckless challenges leading to injuries and warnings, bookings (yellow card) or sending off (red card) by the referee. This provides an explanation of the failure of the heat dissipating mechanism, electrolyte imbalances and

neuroglycopenia due to a fall in blood glucose concentration (Mudambo 1996). In the present game, blood glucose concentrations were not measured because there was no ethical clearance applied for the test. Thus, the players became fatigued and more likely to be tense, vigor, angry (combative, aggressive), confused, depressed and resorting to reckless challenges during the duration of the game as suggested by Mudambo (1996b) among soldiers exercising in the heat. This state of affairs is likely to persist during the post-game phase leading to delayed recovery. Observations showed that post-game arguments with the referee or opponents are common and these sometimes lead to scuffles. Gonzalez-Alonso *et al.*, (1999), Nielsen *et al.*, (1990), Nielsen *et al.*, (1993), Savard *et al.*, (1998) have argued that fatigue in the heat may not necessarily be caused by reductions in cardiac output, exercising muscle blood flow, impaired substrate availability or utilization, or by the accumulation of potassium from damaged tissues or metabolic products like lactate in the plasma, but simply a result of an increase in body temperature during prolonged exercise in hot environments. In a study by Morrison *et al.*, (2006) that looked at the influence of aerobic fitness on central neuromuscular activation and maximal voluntary contractile force during hyperthermia, it was observed that at high body temperatures, maximum force production and voluntary activation were impaired to an equal level regardless of training status.

4.2 Hydration.

In the current study the mean water consumption was less than half of what was reported by Broad and colleagues (1996) who reported that male soccer players consumed approximately 0.516 litres/hr of water. This strongly suggests that the players in Zimbabwe do not consume fluids during the game in spite of high levels of dehydration. On interviewing twenty-four of

the players during one of their training sessions, only 37.5% said they do drink water before the game. As discussed before, observations from the current study showed a significant correlation of water intake with dehydration and albuminuria, and sweat loss with temperature increase with water intake being a significant predictor of dehydration, but an insignificant predictor of core temperature increase.

Despite a temperature change of 1.45%, Highlanders footballers performed well when they played in Hwange because they were physically fitter and adequately hydrated having consumed approximately 0.325 litres of water during the game. Highlanders players' frequency of water intake was highest compared to the other teams who consumed water once or did not take water at all. This higher water intake by Highlanders may be attributed to the fact that their team medic underwent the FIFA and International Olympic Sports Medicine Courses which is not the case with medics of the other teams. A group of footballers who consumed more water than Highlanders had the highest amount of dehydration and sweat loss and the highest core temperature increase, but did not have the most urine output. The expectation is that by consuming more water, one would decrease the degree of dehydration and produce more urine at the end of exercise, however, recommendations by Maughan and Mudambo (1996a) and Nose (1988) were that carbohydrate electrolyte (CE) drinks provide better rehydration than water since they provide water, energy and electrolytes compared to water. This observation shows that an increase in the water intake does not necessarily result in a lower probability of dehydration occurring.

It is critical to point out here that although the mean amount of water (0.325 litres) consumed during the game may appear higher than that of players who did not consume water at all, the water intake was not adequate to match the sweat rates hence the dehydration and failure to produce urine at the end of the game. Many studies and reviews have been carried out to determine the need for fluid replacement during and after exercise in the heat (Cade *et al.*, 1972 and 1992, Coggan and Coyle 1989, Maughan *et al.*, 1994b, Edwards *et al.* 2007, Edwards and Noake 2009) however, most of these, were carried out under controlled laboratory conditions and very few of them have observed responses during training or competition under field conditions. Mudambo (1996b) showed that during field training in the heat soldiers who drank water compared to carbohydrate electrolyte (CE) drinks at the end of training had slower recovery rates, while those who took CE (0.200 litres) 15 minutes before training and at regular intervals (0.400 litres/20 minutes) during exercise had faster recovery rates. In the same study however, Mudambo observed that some soldiers who took water during exercise developed abdominal problems while those who took CE did not.

In the current study, no abdominal problems were observed probably because the players did not take adequate water. A high water intake has been associated with the burden of the water on the gastrointestinal tract thereby increasing the effort needed to do more work and therefore the more metabolic work needed to do this work. Most football players and soldiers believe that water intake during exercise makes them heavy hence the reluctance in taking water even though it was made available (Mudambo 1996a). In the work conducted by van Nieuwenhoven *et al.*, (1999), it was observed that the lactulose/rhamnose ratio and intestinal glucose absorption were significantly decreased in the cycling trial, meaning that

gastrointestinal function is affected as exercise duration is prolonged (van Nieuwenhoven *et al.*, 2000; van Nieuwenhoven *et al.*, 2004). This means that if the players are not adequately hydrated prior to a game and at regular intervals during the game by drinking small amounts of water, they may not be adequately hydrated to withstand the effects of hyperthermia and hypovolemia as suggested by Mudambo *et al.*, 1996c that a 3 hour walk-run exercise in the heat decreases their gastric emptying times. If the current results are compared to those of Broad *et al.*, (1996) [0.516 litres/hr of water] then, the Zimbabwe football players are not drinking enough fluids during the game. Thus, the culture of water drinking is not within these players. Edwards *et al.*, (2007) observed in their study with soccer players that there was a significant increase in core temperature when subjects did not consume fluids and the post-match performance of a sport-specific test was greatly impaired in subjects who had not consumed fluid. This report emphasises the importance of water intake which in the current study, was insufficient to meet the dehydration levels.

An important observations from the current study was that the amount of water drank per game is very low as confirmed by calculations made on the estimated amount of fluid required to maintain body fluid balance which was 0.654 ± 0.5 litres compared to the 0.279 ± 0.23 litres that the footballers drank. It is of importance to note that sweat is hypotonic compared to the extracellular fluid (Sawka and Montain 2001) and heat acclimatization seems to reduce the sweat sodium and chloride concentration. In the current study the composition of sweat was not measured, but it is important to note that sweat is composed of between 20 to 80 mmol/Litre sodium (Guyton and Hall 2000) and when fluid without sufficient

electrolytes is ingested during the game, the extracellular fluid can become hypotonic, therefore less water may be easily retained in the body, or less water will be absorbed from the gastrointestinal tract into the circulatory system. From the observations made in this study, there is a strong correlation between water intake and sweat loss. The suggestions of Sawka and Montain's (2001) offer a probable explanation of why the footballers from the hot regions seem to have a higher sweat rate and consumed more water for example Hwange consumed 0.600 litres of water and had the highest sweat rate (1.87 litre/hour) and the lowest urine output (1.8×10^{-2} litres). This is again observed when they consumed no water, their sweat rate was lower (0.81 litres/hour) and their urine output (1.8×10^{-2} litres) was the same as when they consumed 0.600 litres of water, suggesting they were acclimatized.

Van Nieuwenhoven *et al.*, (2000), have noted that participants in endurance sports frequently suffer from gastrointestinal (GI) symptoms such as abdominal pain, urge to defecate, diarrhoea, heartburn, nausea, and vomiting. This suggests that exercise can influence GI function. Most of the soccer players did not take water (voluntarily) during the course of their matches. The belief by soccer players that taking water makes them too heavy to run or that they will get stomach cramps can be solved by taking smaller volumes at regular intervals rather than a high volume at a time (Mudambo, 1999). It is very difficult to measure GI blood flow during exercise, but it has been reported by Clausen (1977) that at maximal exercise intensity, the flow may be reduced to 20% of the resting value in both trained and untrained people. This is because during exercise, blood to the splanchnic region is decreased due to the demand to supply exercising muscles and cooling the brain (Wyndham *et.al* 1976). Sympathetic output plays an important role in redistributing blood flow during exercise. As a

result, blood flow may be decreased tremendously during maximal sympathetic stimulation, affecting GI motility, intestinal absorption, and mucosal integrity. This may be a cause for exercise-induced GI symptoms (Mudambo 1996b, Van Nieuwenhoven *et al.*, 2000, Van Nieuwenhoven *et al.*, 2004). Training status, exercise intensity, hydration state, and nutrition seem to play an important role. GI symptoms during strenuous physical activity occur more frequently in untrained people compared with trained people and more in women than in men (Van Nieuwenhoven *et al.*, 2000). This brings to question the training and physical fitness level of the Zimbabwean football players because fully fit players should not complain of GI symptoms like abdominal pain when they consume water on the soccer field. Studies done by Van Nieuwenhoven *et al.*, (2000) on the use of sports drinks, which have had caffeine added, have been seen to reduce the GI symptoms, and according to Wald *et al.*, (1976) in their study on jejunal secretions, the secretions may increase small bowel transit time giving rise to lower GI symptoms. Use of caffeine and caffeine based products in athletes is prohibited and is monitored by the Anti Doping Agency. In this study, no investigation was done on the teams total caffeine (from beverages like coca cola, tea and coffee) intake prior to the game. Of importance here is that according to Gisolfi *et al.*, (1991): (1) exercise has no effect on water or solute absorption in the duodenojejunum, (2) fluid absorption occurs significantly faster from a carbohydrate-electrolyte solution (CE) than from water, and (3) fluid absorption is increased six fold by addition of carbohydrate to an electrolyte solution. From their observation it is important that the soccer players are adequately hydrated before, during and after a game, with frequent intake of water but not big gulps. The recommended hydration fluid should be a CE solution which ensures replacement of both water and electrolytes (Maughan and Leiper 1994, Mudambo and Reynolds 1996, Mudambo 1999, Mudambo *et al.*,

1997). The increase in hydration may assist in maintaining the splanchnic circulation, hence maintaining the GI function (intestinal absorption rate), and continuous rehydration, supply of carbohydrate and electrolytes, during a game.

Mudambo (1996b) observed that subjects who exercised without fluids perceived exercise as being very hard after 2 hours of walking running exercise. In the current study there were 15 who did not drink any water during the games. According to Mudambo (1996b), after 2 hours of exercising in the heat, blood glucose would have fallen significantly below pre-exercise concentration and as exercise progressed subjects would complain of increased thirst and fatigue resulting in them walking instead of running and the probability of rehydration and glycogen synthesis, which requires potassium that is lost as part of sweat (Knochel, 1993). In the study by Mudambo (1996b), symptoms of neuroglycopenia developed at 2.75 hours at which stage blood glucose concentration had fallen to between 2.2 and 2.8 mmol.l⁻¹ which suggested that hypoglycemia contributes to fatigue (Coggan and Coyle 1987, Levine *et al.*, 1924). The reduction in plasma glucose (Levine *et al.*, 1924), dehydration (Wyndham and Strydom, 1968), a reduction of plasma volume (Nose *et al.*, 1988) and hyperthermia (Sawka *et al.*, 1992b) have been implicated as the cause of fatigue. In the same study by Mudambo (1996b), combative behaviour of tired soldiers would start to show when the blood glucose concentrations were lower than 2.8 mmol.l⁻¹.

According to Bass and Henschel (1956), Harrison *et al.*, (1981) Gillen *et al.*, (1991); Nagashima *et al.*, (2000); Maw *et al.*, (1996) and Pandolf *et al.*, (1994), during acclimatization, the plasma volume expands. Observations by Mudambo (1996b) showed that

there is an increase in the plasma osmolality and Arginine Vasopressin (AVP) and decreases in Total Body Water, body mass, plasma volume and Atrial Natriuretic Peptide (ANP) at rest after acclimatization without the significant increase in Brain Natriuretic Peptide (BNP). Exercise and heat-stress affect plasma volume in two ways. *Firstly*, they cause fluid movement between compartments (Mudambo 1996b) which is determined by the magnitude and direction of movement of fluids as a result of the exercise type and intensity, state of heat acclimatization, hydration status and temperature (Pandolf *et al.*, 1994) of which the overall effect may be either to increase or decrease plasma volume (Pandolf *et al.*, 1994) and *Secondly*, sweating is the main cause of fluid losses during exercise in the heat. Leiper *et al.*, (2001) in their study noted that changes in Total Body water over long periods, the water turnover rates are faster in individuals undertaking prolonged exercise than in sedentary men, and that the difference was due to the almost three times greater non-renal water losses that the exercising group incurred, suggesting that exercise-induced increases in respiratory water loss and sweat rate are major factors in water loss even in cool environments. As observed in the current study, the changes in weight, water intake, urine flow and sweat loss did not numerically take into consideration loss of respiratory water loss and weight loss due to energy deficit. The observation by Mudambo (1996b) of soldiers in the field suggested that the energy and water requirements of men during prolonged physical activity in hot dry environments are greater than when doing normal work. Therefore, the total energy utilised in the game may also have been high resulting in the observation of 2.21% dehydration and fatigue.

In general, as noted on calculating the estimated required water intake, the football players did not drink adequate amounts of water. An effective post-exercise rehydration will depend on

adequate provision and ingestion of fluids containing electrolytes (especially sodium, chloride and potassium), which are likely to replace deficits from the extracellular and intracellular fluid compartments (Maughan *et al.*, 1994b, Nose *et al.*, 1988). According to Mudambo (1996b) subjects that ingested oral rehydration solution maintained serum osmolality and sodium above pre-exercise concentration, in doing so, maintaining the drive to drink more fluids (Nose *et al.*, 1988). It must also be noted that the loss of body weight may also have been attributed to the partial loss of glycogen stores and its associated water. According to Godek *et al.*, 2005, when they did a comparison of American Football players and cross country runners, they found that the football players lost more weight compared to runners, which they explained to be a result of the high percentage of fast twitch fibres that they have (Zopiec and Taylor 1979). In the present study, in general, the body frame of the football players was much smaller compared to the American Football players. Nevertheless, it must be taken into consideration that the game of football is strenuous, and probably not as strenuous as American football.

4.3 Urine output and albuminuria.

The mean urine flow rate of $2.8 \times 10^{-4} \pm 1.6 \times 10^{-4}$ litres/min and mean total urine output of $2.4 \times 10^{-2} \pm 1.5 \times 10^{-2}$ litres was low as it could impact on renal function. The presence of protein and other sediments in the urine observed in the present study suggested that there may have been clearance of plasma proteins as a result of an increased glomerular permeability and a partial inhibition of tubular reabsorption, indicating that exercise decreases the glomerular electrostatic barrier and facilitates transfer of macromolecules (Clerico 1990, Miyai and Ogata 1990, Suzuki and Ikawa 1991). The observation here shows a good correlation between water

intake and albuminuria and correlation, although weak, with the increase in core temperature. Albuminuria was greater in players consuming more water, suggesting that there may be an overload in the glomerular filtration rate leading to some proteins escaping. According to De Palo *et al.*, (2003) the presence of protein in urine in physical exercise may be related to an increase in protein filtration and the saturation of the mechanism responsible for the reabsorption of the proteins. Considering that the amount of albuminuria was in a negative correlation with urine output, the conclusion by De Palo *et al.*, (2003) may be justified by the observation in the present study that the lower the urine output the higher was the concentration of albumin in the urine because of the reduction in the reabsorption of protein in the proximal convoluted tubule resulting from saturation of the transporters, i.e. T_{max} would have been reached, even though the reabsorption of water is maintained by other systems that facilitate the uptake of water in the renal system with the assistance of the hormones renin, angiotensin, vasopressin, atrial natriuretic peptide (ANP), brain natriuretic peptide (BNP) and aldosterone. Although there is only one area for the reabsorption of the filtered proteins because T_{max} has been reached, haemoconcentration of the blood (Cantone and Cerretelli 1960) still occurs due to the loss of water in sweat and respiration. As a result of the haemoconcentration there is an increase in the amount of albuminuria as the glomerular filtration increases due to the decrease in renal plasma flow or renal blood flow into the renal arteries thereby increasing the filtration fraction (fraction of renal plasma flow that is filtered by the glomerulus) accompanied by a decrease in the glomerular filtration rate which may not be exceeded by decline in renal plasma flow. Yaguchi *et al.*, (1998), acknowledged in their observation of triathletes, that it was reasonable to presume that glomerular damage may occur and persist in some subjects who do heavy exercise, which is what they observed in one

of their subjects in which proteinuria continued till the next day. This could be what may be happening to the soccer players. Another factor that was not looked at is the dietary intake, which could play a factor in the amount of proteinuria. It has been documented that the presence of amino acids, infused or not infused, can increase the protein load that is filtered by the glomerulus, and some of these amino acids like l-arginine and lysine can inhibit the reabsorption of proteins by the renal tubules (Poortmans *et al.*, 1988; Tack *et al.*, 1996; Bello *et al.*, 1999). Of importance is the investigation by Edes *et al.*, (1990) on the effect of prolonged exercise on glomerular permeability and proteinuria that the subsequent decline in albuminuria and albumin-creatinine ratio, despite continued exercise, which was unexpected, indicates a decrease in glomerular permeability.

In the current study there were 7% of the subjects failed to produce a urine sample and subjects playing in teams not from Hwange the produced dark coloured urine (haemoglobulinuria). This was also observed by Mudambo (1996b). Mudambo (1996b) who attributed several factors which were likely to result in dark urine: (1) when blood myoglobin concentration rises to a range of 300ng/ml-2µg/ml, renal threshold is reached and myoglobin spills into urine thereby producing a range of light “iced tea” to darker “Coca Cola” (Clarkson 1993); (2) the breakdown of red blood cells as well as bleeding of the bladder tissue, will also result in dark urine (Clarkson 1993). Observations by Schmidt *et al.*, (1988, 1989) who investigated the effects of cycle exercise on red cell mass and found that the daily destruction of red cells amounted to 2% of the total cell mass in untrained subjects of which during long term training increased red cell production overcompensates for this destruction (Schmidt *et al.*, 1989). The observation by Sawka *et al.*, (1996), on the effects of aldosterone on urine

sodium concentration, explains here, how the reduction on urinary sodium output reduces urine water output thus, making it more concentrated with waste products such as urea, bile pigments of which the dominant is bilirubin a product of red blood cell breakdown, creatinine (Widmaier *et al.*, 2006, Ganong 2005, Guyton and Hall 2000), and unreabsorbed myoglobin (Guyton and Hall 2000). Bilirubin is known to give urine the characteristic yellow colour.

4.4 Sweat loss and Sweat Rate.

There was a very significant correlation between sweat loss and dehydration, water intake, temperature increase. The 1.58 litres sweat loss would lead to a fall in body fluid/blood volume leading to depletion of body energy stores and consequently severe decrements in physical performance. As discussed above, Sweat loss was a significant predictor of dehydration and core temperature increase. The average sweat rate of the subjects was slightly lower than the expected ranges of 1.2 (Broad *et al.*, 1996) and 1.67 (Maughan and Leiper 1994) litres/hour. Work by Godak *et al.*, 2005, showed that the average sweat rate for runners was 1.77 litres/hour which was nearly identical with the 1.71 litres/hour reported in male runners during a 40 km run in similar environmental conditions by Millard-Stafford *et al.*, (1995). Godak *et al.*, (2005) also reported a sweat rate of 2.1 litres/hour [range 1.1–3.6 litres/hour] in American football players. The sweat rate in basketball players competing indoors in ambient temperatures of 20°C was 1.6 litres/hour, whilst Pyke and Hahn (1980) reported a sweat rate averaging 1.8 litres/hour in rugby players competing in ambient temperatures of 38°C. The following factors favour a high sweat production during exercise: physical conditioning (aerobic and anaerobic), acclimatization, hydration status, exercise intensity, physical size, and amount of clothing or equipment worn. The footballers who were

from Hwange and played in a very hot environment (31.4 °C, 60% relative humidity), a game which was a must win for them to get closer to 3rd position on the log and at home (intense exercise and stress), of which they eventually lost had a sweating rate of 1.8 litres/hour, a value which was greater than that observed by Broad *et al.*, (1996); and Maughan and Leiper (1994). Comparing the results of the teams there seems to be a relationship between urine output, albuminuria, weight changes, core changes in temperature, ambient temperature and sweat loss. The higher the sweat rate the higher the albuminuria, temperature and weight change. The correlation between sweat rate and increase in core temperature and dehydration were very strong whilst that of albuminuria was weak, hence not significant. It was also observed that the higher the sweat rate, the lower was the urine output, but the correlation was weak and insignificant. These observations re-emphasize the need for adequate hydration during the course of the game because exercise intensity exceed water intake. Another factor that could explain the higher sweat rate is the number of sweat glands. The more the number of sweat glands that are recruited to open, the more is the sweat produced. The footballers from hot regions had higher sweat rate confirming that adaptation to the hot environment probably due the ability to recruit more sweat glands leading to more efficient body cooling had occurred over long periods of exposure to exercise in the heat. The current study further observed that the higher the pre game body weight the higher the sweat rate, which brings in the issue of the body size/surface area. It is expected that the greater the surface area the greater the sweating rate. The Highlanders players from a moderately high temperature region had the second highest sweat rate of 1.03 litres/hour. The observation here shows that the link may not necessarily be the environmental temperature at the time of the game but the previous exposure to high temperatures which has resulted in the players recruiting more sweat glands

so as to favour heat loss from the body. This is in agreement with Irmak *et al.*, (2004) who suggested that environmental pressures produce noticeable differences among people. Although Irmak *et al.*, (2004) expressed the variations in the selective brain cooling mechanism to the issue of ethnic groups, this may still be applied to regional variations, as is the case in this study. The strong correlation between sweat rate and water intake shows that the higher the water intake the higher the sweat rate.

Sodium and chloride are the main solutes in sweat and a large fraction of water lost in sweat will be lost at the expense of extracellular fluid including plasma (Pandolf *et al.*, 1994, Mudambo 1996b). The mean sweat loss per match as observed here was 1.58 ± 0.93 litres (range 0.5-4 litres) and therefore, possibility of acute dehydration can occur if the football players are not encourage to drink water frequently during the game and chronic dehydration resulting from the weekly practice sessions that teams undergo. Sodium and chloride concentrations need to be replaced too in order to maintain relative rehydration. However, if dietary intake of sodium meets the daily requirement, replacement of the electrolytes lost during sweating may not be necessary. Again, it is important to mention here that, electrolyte loss very much depends on the duration and intensity of exercise and the environmental conditions in which it is played. Thus, a 90 minute soccer game with a 15 minute break at half time (45 minutes) may not elicit adequate changes in electrolyte balance.

According to Knochel (1994) exercise in the heat results in loss of potassium through urine, as a result of the aldosterone effect, and sweat. This is said to be a result of muscle damage because of exercising in the heat (Knochel 1994). The loss of potassium from the damaged

muscle affects glycogen metabolism. Ingested carbohydrates travel through the blood stream to the liver where it is converted to fat, stored as glycogen or transported to muscle for storage as muscle glycogen. Potassium is a pre-requisite for glycogen synthesis, therefore, the muscles are not able to anabolise glycogen resulting in decreased storage of this primary source of energy for the muscles energy stores (Knochel 1994). During strenuous exercise, muscle glycogen becomes depleted and if not replenished, fatigue follows (Mudambo 1996b, Costill and Haggreaves 1992, Coyle 1988, Coyle and Montain 1992) remembering that the loss of body weight may be attributed to the partial loss of glycogen stores and its associated water. As noted above, potassium is also an important ion that needs to be replaced during and after exercise.

Although fatigue cannot necessarily be prevented by carbohydrate feeding, it can be delayed, especially during the final stages of a match when glycogen stores are low and the subjects rely on the blood glucose for energy (Bergstrom and Hultman 1967a, Bergstrom *et al.*, 1967b, Costill *et al.*, 1971, Coyle *et al.*, 1983) by carbohydrate feeding during the exercise (Coyle *et al.*, 1983, Coyle and Montain 1992).

4.5 Substitution and Outcome of game

The differences in the number of substitutes between Hwange, which did not use any substitutes, and the other teams suggested that Hwange players were more acclimatized and therefore suffered less fatigue in the heat. Comparing the extent of dehydration during the game and percentage temperature increase, Buymore had a greater percentage dehydration of 3.5% vs. 1.7% of Hwange and percentage temperature increase of 2.7% vs. 1% of Hwange. It

was also evident during the last quarter of the game that the Buymore players were tired as shown by the differences in the dehydration and temperature increase and the 1-0 goal scored during the last 15 minutes of the game. The incidents prior to the goal were a result of reckless tackles which resulted in unnecessary free kicks near the 18 metre area and the resultant yellow cards ushered to Buymore players. These reckless tackles were indications of fatigue and the development of combative behaviour associated with fatigue and dehydration when exercising in the heat (Mudambo 1996b). According to Mohr *et al.*, (2003) most of the incidents of recklessness occurred towards the end of the matches and this could provide some explanation on the impact of cognitive impairment on the player's judgement which in the case of Buymore was costly as it resulted in them losing the game.

Although there was a strong positive correlation between outcomes and body temperature change and extent of dehydration, the use of this may or may not be of value in assessing the cognitive performance of the team. A comparison of the percentage temperature increase and percentage dehydration of Buymore (1.4 % and 1.98% respectively) and Highlanders (1.45% and 1.97% respectively), the readings were similar but the outcomes were different. This makes the use of the relationship very subjective. There is further work required in this area to get more conclusive results that would show that cognitive impairment in addition to temperature changes and extent of dehydration may play a role in some games outcome.

According to Nybo and Nielsen (2001b) hyperthermia increases the perceived exertion. This has some limited explanation because the neurobiological mechanisms have not been clearly defined. According to Hasegawa *et al.*, (2000) dopamine, a hormone secreted by the

substantia nigra and neurons located adjacent to the *substantia nigra*, which when secreted increases self-stimulation, is implicated in the control of body temperature and in tolerance or exercise in the heat (Bridge *et al.*, 2003). According to Nestler *et al.*, 2001, a combination of Dopamine and Norepinephrine, which are both catecholamines and synthesised as a result of hydrolysis of tyrosine to Dopa, decarboxylation of Dopa gives dopamine then hydroxylation of Dopamine producing norepinephrine, have both been implicated in the arousal, motivation, reinforcement and reward, the control of motor behaviour and mechanisms of addiction. The depletion of the two is linked to central nervous system fatigue (Ganong 2005, Guyton 2005). The combination of the effect of the two catecholamines may impact on the final temperature at the end of the game as a result of the increase in the motivation as a result of the goals and possible outcome of the game, thereby affecting the perceived exertion. Cognitive performance still needs further work and cannot be truly reflective of the outcome of the game. McMorris *et al.*, (1999) also observed in soccer players that exercise had no significant effect on accuracy during maximal exercise but the speed of decision making was significantly affected by exercise. This does show that exercise intensity is a determining factor of decision making but this may not be influential in the ability to physically perform during a game.

If the outcomes of games can help determine the possible impact of the game on temperature change and extent of dehydration this may help in explaining the reason behind the number of yellow cards and especially red cards. In all matches played there was only 1 red card recorded which was to a player who had just been substituted in the 74th minute. The yellow cards may not be a true reflection because players may intentionally get a yellow card as they

may be defending their position (zone defence) but a red card is not always the conscious intention which could be explained by the depletion of dopamine and epinephrine.

CHAPTER 5 CONCLUSION AND RECOMMENDATIONS

5.1 Conclusion

Based on results of fluid intake, urine output, weight and temperature changes, the observations of the present study seem to suggest that the players do not take adequate fluids leading to losses in body weight during the game, and consequently dehydration and very low urine output at the end of the game. Thus, the Zimbabwe players seem to stay in a dehydrated state/chronic dehydration. The presence of albuminuria suggested that the players experienced constant renal stress resulting from inadequate water intake. The increase in the body temperature seems not to have much bearing on the extent at which dehydration may occur, but may be a reflection of the function of the central nervous system as reflected by the outcome of the games. While there are no conclusive results, the reckless tackles and a display of combative behaviour when playing in the heat which resulted in fouls leading to goals, would point towards some cognitive impairment which would affect the judgement of some players. This however, requires further investigation.

5.2 Recommendations

1. Further work is needed in the area to ascertain the possible extent of dehydration.
2. There is also need to look into the possible sodium, chloride and calcium loss in sweat in the 90 minutes that a game is played although the time that game is played is may not long enough to show significant change.
3. There is need to compare the extent of dehydration when carbohydrate-electrolyte fluids are compared to water during the training sessions and competition.
4. There is need to look at the time it will take for rehydration to occur in the football players at different levels of dehydration, gastric emptying rates and rehydration rates.
5. Assessment of dehydration relative to position played may need to be done to determine who is most vulnerable to dehydration.
6. The observed mean sweat loss was low (1.58 L) and water intake was 0.298 litres, which is lower than the sweat loss. It is recommended that players should take 0.350-0.400 litres per 15 minutes during the game, of which by the end of the game should have consumed 2.1-2.4 litres of water in small amounts at regular intervals in order to avoid dehydration.

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APPENDICES

APPENDIX A: LIST OF DEFINITIONS

Acclimation and Acclimatization these terms are often used interchangeably to refer to any adaptive changes which occur when an individual undergoes prolonged or repeated exposure to stressful environment, and which reduce the physiological strain produced by such an environment (Pandolf *et al.*, 1994, Mudambo 1996b). In the current study, such changes will be called: Acclimation if change is produced in a controlled laboratory setting and acclimatization if changes are produced in a natural environment. (Mudambo 1996b)

Dehydration is referred to as the process of reducing total body water. (Sawka and Montain 2001)

Delayed Onset Muscle soreness (DOMS), a clinical model of muscle damage consisting of muscular pain and other symptoms experienced 24 hours to 48 hours after novel or intense exercise. The signs and symptoms of DOMS include dull, diffuse pain and tenderness; stiffness; swelling; and decreased strength of the exercised muscle (Fitzgerald *et al.*, 1991, Cleary *et al.*, 2006).

Filtration fraction the fraction of renal plasma flow that is filtered by the glomerulus

Glomerular filtration rate, the rate at which substances are filtered through the glomerulus.

Heat Cramps painful migratory skeletal muscle spasms attributed to salt depletion and resultant hyponatremia. (Gardener *et al.*, 1994, Mudambo 1996b)

Heat exhaustion is a “functional” illness usually due to the combination of increased circulatory load due to thermoregulatory and muscular demand and reduced “effective” plasma volume and venous return due to vasodilation in skin and muscle and sweating induced depletion of salt and water. (Gardener *et al.*, 1994, Mudambo 1996b)

Heat stroke a life threatening progressive multi-system disorder reflecting collapse of the thermoregulatory system with severe neurological symptoms including obtundation or coma. (Gardener *et al.*, 1994, Leithead and Lind 1964, Mudambo 1996b)

Haemoconcentration the progressive increasing concentration of intravascular constituents arising from a continuous loss of fluid (plasma water) from the intravascular space. (Harrison 1985, Mudambo 1996b)

Haemodilution the progressive decreasing concentration of intravascular constituents arising from a continuous gain of (interstitial) fluid by intravascular space. (Harrison 1985, Mudambo 1996b)

Haemodynamic response the body's compensatory response to a reduction in blood volume by increasing the heart rate and blood pressure to the central venous circulation and muscle areas mentioned above. (Gonzalez-Alonso *et al.*, 1997, 2004).

Hyperthermia temperature elevation, which results from the inability of the thermoregulatory systems to maintain normal core temperature due to heat generation in excess of heat dissipation. (Gardener *et al.*, 1994, Mudambo 1996b)

Hypervolemia a blood volume greater than normal. (Harrison 1985, Mudambo 1996b)

Hyponatremia serum sodium below 130 mmol/litre, which can be caused by drinking an excessive amount of water (Sawka and Montain 2001)

Hypovolemia a blood volume less than the normal (Harrison 1985, Mudambo 1996b)

Insensible sweat is unnoticeable sweat.

Physical Fitness refers to the strength at which the body is able to function e.g. aerobic fitness, muscle fitness.

Rehydration is referred to as the processes of replacing the lost total body water (Sawka and Montain 2001)

Sensible sweat is noticeable sweat.

Stroke volume the blood volume ejected by the ventricles of the heart during one heart beat.

(Widmaier *et al.*, 2006, Ganong 2005, Guyton and Hall 2000).

The transport maximum (Tmax), the maximum amount that can be reabsorbed at a given time.

VO_{2 max} is the amount of oxygen one takes per minute per each kilogram of his body weight (ml.kg.min⁻¹) (Guyton and Hall 2000)

APPENDIX B: DATA COLLECTION FORM

Name_____

Age_____

Body weight before game_____ Body weight after game_____

Core body temperature before game_____

Core body temperature after game_____

Urine sample given after game: Yes/No

Water Intake during the game

No. of times water drank	Estimated no. of sips	Estimated vol drank

Did you drink water before the game? Yes/ No

APPENDIX C: CONSENT FORM

Physiology Department
University of Zimbabwe
P.O. Box MP167
Mt Pleasant
Harare

303211 ext 1057

Dear Candidate

You have been selected to participate in a study being conducted by Carol B. Maponga of the Physiology Department, University of Zimbabwe for duration of 10 months. The aim of the study is to determine the thermal regulation of football players from a hot region of the country and those from a cool region of the country on exposure to cold and high temperatures respectively before, during and after exercise. In general we will be looking at the response of football players to change in environmental temperature when exposed to temperatures which are traditionally not the usual environmental temperatures they are used to playing. The differing temperatures can affect how one performs during the game. This will be done during the football season when two teams play return matches with each other; namely Hwange Football Club and Buymore Football Club. We anticipate that the results of this study could help improve the understanding of the performance of sports persons in Zimbabwe.

As mentioned above two teams will be participating in the study. The two teams will be followed when they play both at home and away – during return matches with each other in the league matches at their respective home grounds. You have to be between 18-32 years of age. You will have to be from a selected team from either one of the two categories below:

1. the hot region group
2. the cooler region groups
3. the moderate region

We will collect the following biological specimens:

- Urine samples will be collected on the same day to assess water loss and electrolyte loss during training and during the return matches.

This is the only tests that will involve biological specimens.

Other measures to be conducted include

1. Anthropometrical measures (height, weight),
2. Water intake assessment during the game
3. Body temperature changes

There will be no form of discomfort that will be encountered during the exercise.

All records collected will be strictly confidential. You will be given a candidate number, which will be your only form of identification. No names will be used or anything that may make a subject identifiable in the report that will be produced. The registration to this study is voluntary and you are free to deregister from the study without any repercussions. We advise that you first consult your health provider before filling in the consent form.

If there are any queries please feel free to approach Carol Maponga so that they can be addressed at the above address.

Yours Sincere

Carol Maponga

APPENDIX D: ABSTRACTS RELATED TO THESIS – OCTOBER 2007

UNIVERSITY OF ZIMBABWE

ANNUAL MEDICAL RESEARCH DAY ABSTRACT FORM

Type only within the borders:

Dehydration, sweat rate and fluid turnover in African Footballers playing in

Zimbabwe. RD/01/07

Maponga CB, Mudambo Kaka SMT, Sithole-Niang I, and Tafirenyika A

Introduction. Playing soccer without drinking fluids is likely to cause dehydration and consequently heat stress related conditions and poor performance.

Objective. The objective of the study was to determine the level of dehydration that football players undergo during a game of football, and the outcomes of different factors involved in maintaining the body temperature and the hydration levels. **Methodology.** Sixty-one volunteers who play in the Premier Soccer League (PSL) and the First Division Soccer League were recruited into the study. Before the match they were asked to empty their bladders. They were monitored for changes in body weight and core temperature, albuminuria, urine volume and the flow rate, sweat loss, sweat rate and fluid intake measured. **Results.** Overall dehydration (total body weight loss), sweat loss, sweating rate, core temperature increase, water intake, urine output, urine flow rate and albuminuria were 2.21 (0.83)% 1.58 (0.71) litres, 0.84 (0.64) litres/hour, 1.79 (0.83) %, 0.28 (0.23) litres , 2.4×10^{-2} (1.5×10^{-2}) litres, 2.8×10^{-2} (1.6×10^{-4}) litres/minute and 1.2 (1.4) g/litre respectively.

There was a significant correlation between dehydration and water intake ($P<0.05$) and a strong correlation between dehydration and increase in core temperature and sweat loss ($P<0.01$). Increase in core temperature had a strong correlation with dehydration, water intake and sweat loss ($P<0.01$). On predicting dehydration sweat loss, urine output and water intake were significant predictors ($P<0.05$). On predicting temperature increase, sweat loss was the only predictor ($P<0.05$). **Conclusion.** The results, based on fluid intake, urine output, weight and temperature changes seem to suggest that the players do not take adequate fluids leading to reduced body weight during the game, dehydration and consequently poor performance. The increase in the body temperature seems not to have much bearing on the extent at which dehydration may occur.

References

1. Mudambo, K.S.M.T. and Reynolds N. (1996a) Body fluid shifts after field exercise in dehydrating conditions: effects of water and electrolyte solution on rehydration. Dehydration, Rehydration and exercise in the heat. *J. Sport Sci* 14 (4): 359
2. Godek S F, A R Bartolozzi, J J Godek. Sweat rate and fluid turnover in American football players compared with runners in a hot and humid environment. *Br J Sports Med* 2005; 39:205–211.

Abstracts should be presented with the following headings: Introduction (study justification), Objectives, Materials & Methods (participants, setting and study design, how the study was carried out, interventions and outcome measures used), Results and Conclusions, Not more than 2 References.

APPENDIX E: RESULTS DATA

Table 5: Fitting of the regression model with dehydration as the dependent variable

Tests of Between-Subjects					
Dependent Variable: Percent Dehydration					
Source	Type III of	df	Mean	F	Sig.
Corrected	71.21 ^a	5	14.24	184.07	.000
Intercep	.073	1	.073	.945	.335
Water intake (ml)	3.694	1	3.694	47.74	.000
Urine output	.439	1	.439	5.669	.021
Sweat loss	57.42	1	57.42	742.12	.000
Albuminuria	.159	1	.159	2.057	.157
Ambient temperature	.006	1	.006	.079	.780
Error	4.255	55	.077		
Total	321.23	61			
Corrected	75.46	60			

a. R Squared = .944 (Adjusted R Squared = .938)

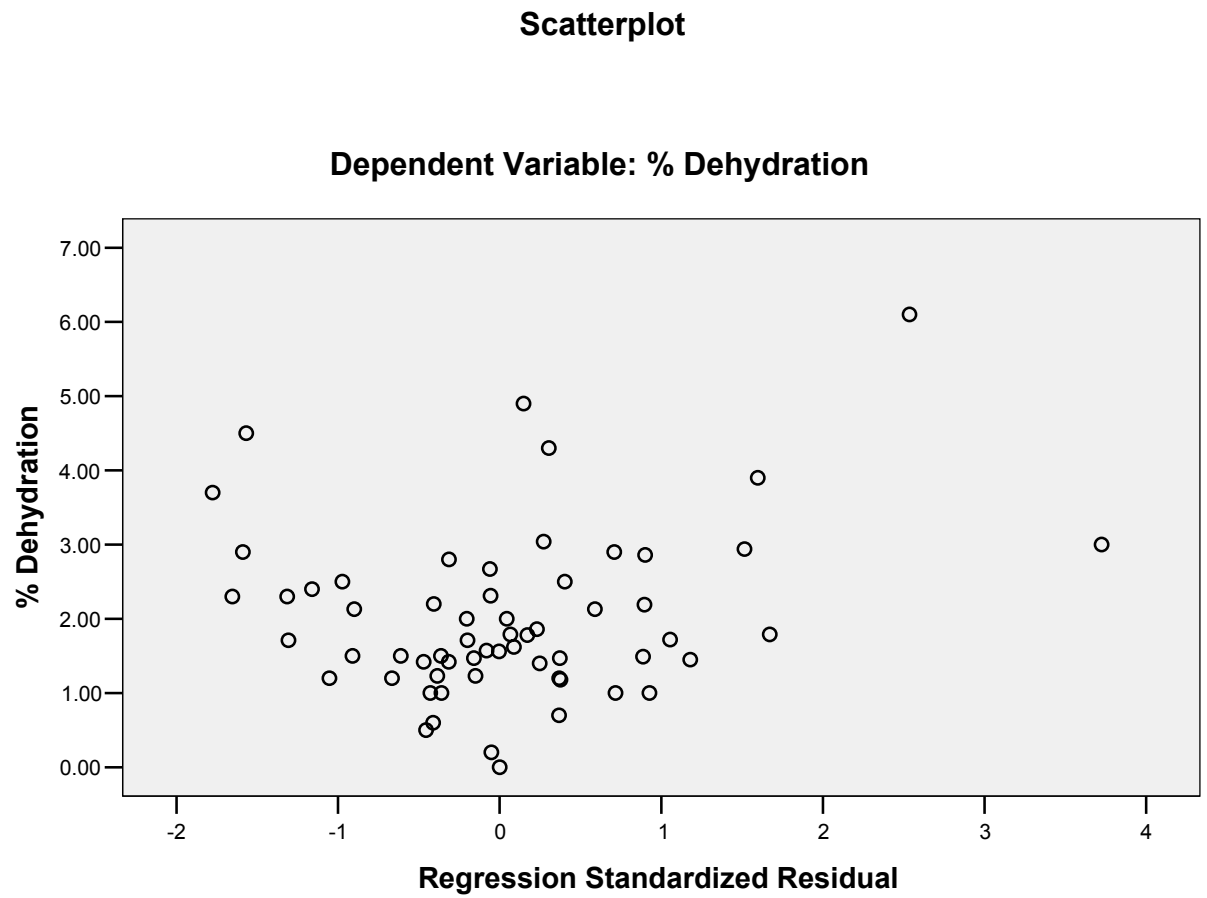
Table 7: Relationship of percentage temperature change within a team.

ANOVA						
		Sum Square	df	Mean	F	Sig.
Percent Dehydration	Between	7.006	1	7.006	6.037	.017
	Within	68.46	59	1.160		
	Total	75.46	60			
Percent temperature increase	Between	9.650	1	9.650	8.007	.006
	Within	71.11	59	1.205		
	Total	80.76	60			

Table 8: Fitting of the regression model with temperature as the dependent variable.

Tests of Between-Subjects					
Dependent Variable: Percentage temperature increase					
Source	Type III of	df	Mean	F	Sig.
Corrected	26.11 ^a	5	5.224	5.258	.001
Intercep	6.196	1	6.196	6.237	.016
Water intake (mls)	1.796	1	1.796	1.808	.184
Urine output	1.176	1	1.176	1.184	.281
Sweat loss	4.483	1	4.483	4.512	.038
Albuminuria	.902	1	.902	.908	.345
Ambient temperature	.533	1	.533	.536	.467
Error	54.64	55	.994		
Total	253.13	61			
Corrected	80.76	60			

a. R Squared = .323 (Adjusted R Squared = .262)

Normal Scatterplot showing standardized residual verses %Dehydration

Normal P-P Plot of Regression Standardized Residual