DEHYDRATION IN U/19 RUGBY PLAYERS IN THE HOT CONDITIONS OF THE KAROO

by

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DECLARATION

I, PETRUS VAN DER WALT VERMEULEN (Student No: 1976559086) certify that the script hereby submitted by me for the **M. Sports Medicine** degree at the University of the Free State is my independent effort and had not previously been submitted for a degree at another University or Faculty. I hereby cede copyright of this product in favour of the University of the Free State.

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Dr P V Vermeulen **Date**

DEDICATION

I dedicate this dissertation to my wife, Anne, for her support through the years without ever complaining.

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LIST OF ABBREVIATIONS AND ACRONYMS

ACSM	American College for Sports Medicine
ADH	Antidiuretic Hormone
BMI	Body Mass Index
CNS	Central Nervous System
EAH	Exercise Associated Hyponatremia
EMS	Emergency Medical System
FTO	Fluid Turnover
Hb	Haemoglobin
HCT	Haematocrit
IMMDA	International Marathon Medical Directors Association
IRB	International Rugby Board
NCAA	National Collegiate Athletic Association
Posm	Plasma Osmolalety
PV	Plasma Volume
SBF	Skin Blood Flow
SG	Specific Gravity
SR	Sweat Rate
TBW	Total Body Water
Tre	Rectal Temperature
US	United States
USA	United States of America
USG	Urine Specific Gravity
WBGT	Wet Bulb Globe Temperature

ABSTRACT

Key words: *Youth Rugby, Dehydration, Urine-Specific Gravity (SG)*, Blood *haematocrit (%).*

Objectives: The aim of this study was to determine the dehydration status of u/19 School rugby players during a game of rugby in the Hopetown district in high temperatures.

Methods: This study was a cohort-analytical study on certain variables associated with hydration levels of u/19 rugby players from Hopetown High School during two matches in 2007 and two matches in 2009. The group of rugby players was subjected to a pre-evaluation (15min before the game) followed by a re-evaluation performed 10min after the game. In this way the dehydration status of the players could be determined. Thirty-one rugby players participated. Readings were taken of Urine-Specific Gravity (SG), blood haematocrit, and body mass of every rugby player before and after every rugby match. The student t-test was used to test for significant differences within the group. A significance level of 0.05 was used throughout the study.

Results: The anthropometric characteristics in our study for 2007 and 2009 are very similar as expected, and showed a mean length of 177 ± 7-8 cm, ranging from 165 to 190 cm, a mean body mass of 71.5 ± 13.7kg and a mean body mass index (BMI) of 22.88 ± 3.98kg/m^2. Between 3 (17%) and 10 (67%) of the players were dehydrated post-match according to the decrease in body mass. The pre-exercise urine specific gravity measures were significantly lower ($p < 0.05$) before all 4 matches than after the matches as expected, and most of the players could have been better hydrated at the beginning of the match. 20% - 94% of the players were dehydrated pre-match and almost all the players (93% and 100%) were dehydrated after the match. The pre-match mean haematocrit (HCT) and the post-match mean HCT was in the range of 0.46 - 0.47. However, in two of the matches significant differences ($p < 0.05$) in HCT were recorded.

Conclusions: It was alarming to find that a large number of the players were dehydrated before the match, but more important, almost all of them after the match. Recommendations for fluid and electrolyte replacement must be carefully considered and monitored in rugby players to promote safe hydration and avoid hyponatremia.

DEHYDRATION IN U/19 RUGBY PLAYERS IN THE HOT CONDITIONS OF THE KAROO

CHAPTER 1

INTRODUCTION AND PROBLEM STATEMENT

1.1 INTRODUCTION

Hydration level is a critical factor influencing exercise performance, especially in the heat. Moreover, dehydration due to exercise, heat exposure, diuretics, or a combination of these factors, is common and even intentional in many weight class sports, which often depend largely on anaerobic energy production (Kraft, Green, Bishop, Richardson, Neggers & Leeper, 2012).

It has been also well established that exercise in heat, especially if there is high humidity, can adversely affect performance and may even result in serious heat illness, such as heat cramps, heat exhaustion and heat stroke. However, according to Noakes (2006), a remarkable feature of the human species is our great capacity to lose heat and to regulate our body temperatures when exposed to heat. "***Whenever one's body temperature rises, even for physiological reasons, we enter into danger and anything that interferes with physiological cooling, or adds to the internal heat load exacerbates that danger. The wonder is, not that anyone gets hyperpyrexia, but that so few of us do"*** (Brukner and Khan, 2012). Independent of the outside temperature, sporting exercise lasting several hours leads to an increased body core temperature. Proper hydration behaviours are essential for the well-being of the human body (Sawka, Cheuvront, & Carter, 2005). Whether male, female, old or young, fluid consumption is crucial to the body operating to its fullest potential. It is thus essential that the body needs to prevent hyperthermia, which is extremely dangerous.

Healthy individuals maintain body water balance despite high water needs and exposure to stressors. However, one challenge to body water homeostasis is exercise (Sawka et al*.,* 2005). Schoolboy rugby players do not inherently know the adequate amount of fluid to consume when exercising and playing rugby. Hydration knowledge, as well as exercise intensity and duration may influence the hydration behaviours of players. The level of exercise intensity necessary is unique to each player and his abilities. Understanding what influences hydration practices should help healthcare professionals identify whether a) educational materials or b) a seminar for rugby players about appropriate fluid consumption during a game or c) training is needed, in order to promote adequate fluid intake.

Fluid replacement is an important concern for everyone because dehydration can result in adverse health consequences. Several factors can affect hydration status. The environment in which rugby players live and exercise may have a direct effect on fluid needs. For example, climates with increased heat and humidity result in increased fluid needs.

Vigorous exercise in hot conditions causes the core temperature to rise even higher. At 40^0C, however, a limit is reached above which health may be seriously affected. Nevertheless, rugby games are often scheduled for the hottest part of day during summer. In the hot South African sun, rugby players are often exposed to temperatures that pose risks. According to Yeargin, Casa, Judelson, McDermott, Ganio, Lee, Lopez, Stearns, Anderson, Armstrong, Kraemer, and Maresh (2010) investigators have documented hydration status and related variables in professional and collegiate football players, but few researchers have examined high school athletes or have compared different age groups within adolescence. Many players are unsure of the type and amount of fluid they should consume, and healthcare professionals, coaches, and conditioning coaches can assist players by relaying appropriate hydration information.

Heat-related illnesses have received substantial public attention in the United States after the recent deaths of collegiate and professional athletes in the National Collegiate Athletic Association (NCAA), National Football League, and Major League Baseball from heat stroke (Howe and Boden, 2007). Because exertion heat stroke is entirely preventable, this study focuses on prevention tactics in rugby dehydration that may help reduce the incidence of catastrophic heat-related illness events. These prevention strategies include scientific evidence and detailed advice on acclimatisation to the heat; proper fluid replacement before, during, and after a rugby match; wearing proper rugby jerseys during certain environmental conditions; and early recognition of heat- related illness via direct monitoring of players by other players, coaches and medical staff.

Marshall (2010) stated that the ability to predict a player who is most susceptible to heat related injury and thus offer appropriate early intervention treatments such as specific hydration recommendations would be a major breakthrough in prevention of dehydration. Children may require specific recommendations if, for example the amount of their sweat electrolyte loss is different. Although a base of observational case data, physiological information, and expert opinion exists (Marshall, 2010), the science surrounding this field is devoid of health communication and health behaviour research, and there is a pressing need for analytical studies to evaluate intervention programmes and/or identify new risk factors. There is also a need for ongoing data collection on heat injury incidence and on the knowledge, attitudes and behaviours towards heat injury among youth athletes, their care givers and their coaches. Because sporting activity can occur in hot conditions, such as rugby games in the Hopetown district in South Africa, sports medicine clinicians must be well versed in both prevention and management of heat-associated illness (Brukner and Khan, 2012).

1.2 THE **AIM OF THE STUDY**

The aim of this study was to determine the dehydration status of u/19 school rugby players during a game of rugby in the Hopetown district in high temperatures.

CHAPTER 2
LITERATURE REVIEW

2.1 INTRODUCTION

According to Marshall (2010) heat injury is a potentially lethal condition that is considered to be completely preventable. Fatal heat injury is relatively rare (0.20 per 100 000 player-seasons in US high school football) and there is very limited data on non-fatal incidence. Expert recommendations for prevention include gradual acclimatisation of young athletes to hot conditions, reductions in activity in hot and humid conditions, wearing light and light-coloured clothing, careful monitoring of athletes for signs of heat injury to facilitate immediate detection, having the resources to immediately and rapidly cool affected athletes, and education of athletes, care givers, and coaches about heat injury.

However, rugby is a sports modality that requires a variety of physiological responses from players as result of combined plays, high-intensity repetitive runs and contact frequency. Since each player may play distinct functions, specific physical conditioning and training levels become necessary (Scott, Roe, Coats, and Piepoli, 2003). In rugby, there is a high incidence of collisions, that makes participants to present adequate characteristics of velocity, agility, resistance, strength, flexibility and own abilities. These characteristics in this or in other sportive modalities produce significant increase in body temperature. According to Meir, Brooks, and Shield (2003) during physical activity, low levels of thermal stress may cause discomfort and fatigue, while higher levels may even dramatically decrease the performance. The prolonged thermal stress leads to hypo hydration resulting in decreased blood volume, cardiac yield, blood pressure and finally in the reduction of the sweat process efficiency.

In a rugby game or during training, considerable amounts of liquids and electrolytes are lost in sweat; energy expenditure is also high. The energetic fuel depletion results in muscular fatigue while the disturbances in the hydric and electrolytes balance may lead to more serious complications. The exercise stress is intensified through dehydration that increases body temperature, impairs the physiological responses and physical performance, and brings health risks.

According to Marshall (2010), there are no well-conducted analytical epidemiological studies, such as cohort or case-control studies, that have attempted to identify risk factors for heat injury. The available information on risk factors is entirely based on case series data, clinical observation and data from exercise physiology studies. There are few recent well-controlled exercise physiology studies of heat and exercise in children that are directly applicable to real-world field conditions (Rowland, 2008). Actual observational studies of children exercising under field conditions are typically limited to heat injury precursor conditions, such as mild dehydration or subclinical increases in core body temperature (Bergeron, McLeod, & Coyle, 2007, Rivera-Brown, Ramírez-Marrero, Wilk et al., 2008). Knowledge of risk factors for heat injury is largely dependent on a group of exercise physiology studies, many of which have been limited to adults and were conducted in laboratory settings that may or may not simulate real field conditions (Marshall, 2010). Therefore, in the literature review, all these effects which occur even at light or moderate hydration will be discussed.

2.2 EXERCISE AND THERMAL STRESS

According to Allyson, Howe, Barry and Boden, (2007) at a cellular level in a healthy person, heat stress produces a predictable cascade of events. Peripheral vasodilatation at the skin level will produce heat loss and shunt blood from the central circulation. According to Rowland (2008) the dynamics of heat flux during sustained exercise can be briefly summarised as: heat liberated by contracting muscle fibres is transferred away by its surrounding blood flow, resulting in an increase in core body temperature [estimated as rectal temperature (Tre)]. In response, hypothalamic control centres and peripheral receptors trigger compensatory cooling mechanisms, principally:

1) cutaneous vasodilatation to augment skin blood flow (SBF) for convective heat loss to the surrounding air, and

2) increased rate of sweating (SR) via sympathetic cholinergic stimulation to dissipate heat by evaporation at the skin-air interface (Rowland, 2008).

The magnitude of convective heat loss is governed by the local skin-air temperature gradient as well as adequacy of cutaneous blood flow. This means of heat dispersal is thus most effective in conditions of moderate environmental temperature, and it becomes less so as Tre rises. Heat loss by evaporation is also directly related to both rate of sweat production and the skin-air water vapour pressure gradient. In high Tre, then, body-heat loss is affected primarily through sweating, particularly in conditions of low ambient humidity. Rowland (2008) also stated that many factors influence this basic scheme, including level of aerobic fitness, clothing, energy substrate utilisation, body composition, and wind velocity. Highly critical, however, is the state of body hydration and plasma volume, because increasing levels of dehydration incurred via sweating during exercise are reflected in decreases in cardiac output, decrements in SR, and rise in Tre. In summary, then, thermoregulatory efficacy during exercise is most closely linked to:

1) adequacy of circulatory responses,
2) rate of sweat production, and
3) maintenance of body fluid volume, all in response to exercise intensity

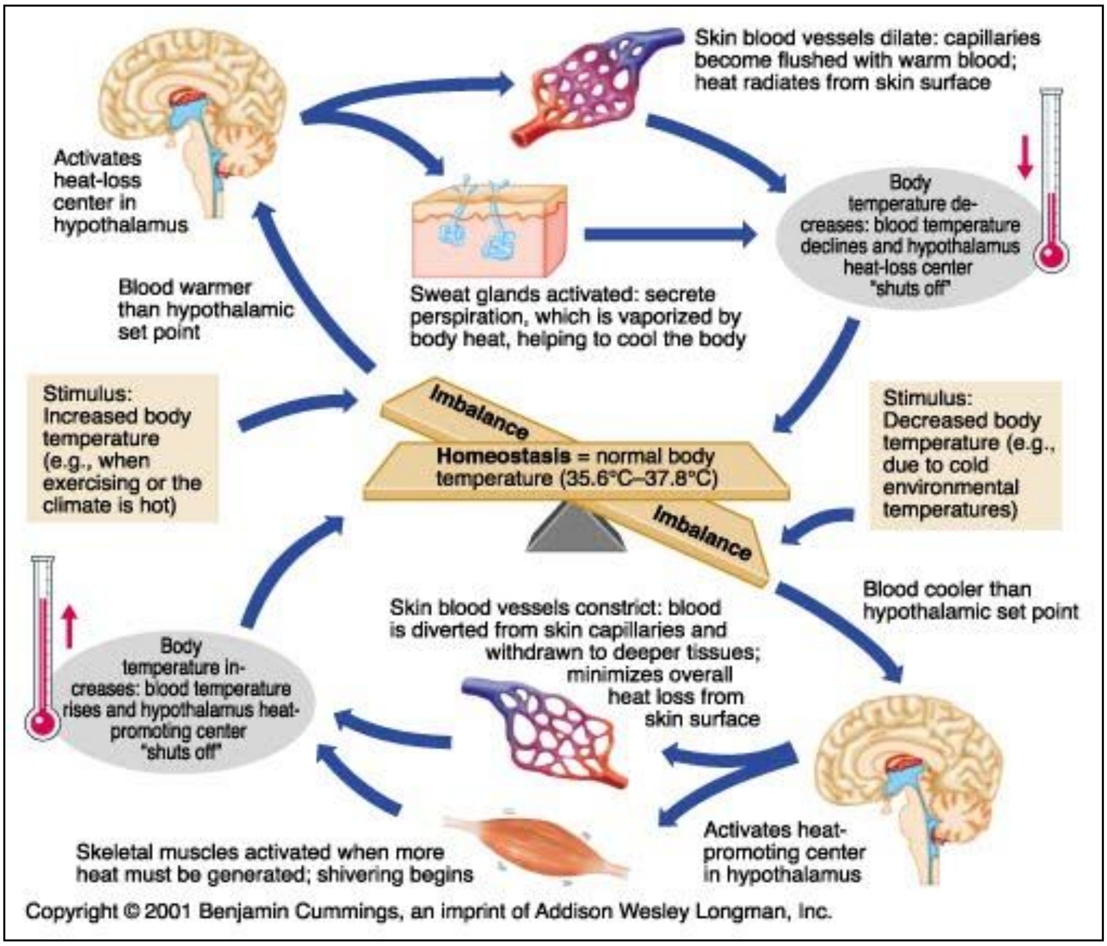
The basis for heat exchange from a human body to the environment occurs in 4 ways - conduction, convection, radiation, and evaporation. All methods are dependent on the presence of a heat gradient. Heat will transfer from a hotter object to a cooler one. Loss of this heat gradient by certain environmental conditions can inhibit appropriate thermoregulation (Rowland, 2008).

2.3 MECHANISMS OF THERMOREGULATION

This study focuses on the problems associated with exercise in the heat and the strategies that rugby players can use to minimise the impact of environmental conditions on performance. The researcher will first provide a basic understanding of human thermoregulation and the mechanisms of thermoregulation.

2.3.1 Thermal Balance

Figures 2.1 and 2.2 show that body temperature, the temperature of the deeper tissues (core), represents a dynamic equilibrium between factors that add and subtract body heat, integration of mechanisms that regulate heat transfer to the periphery (shell) after evaporative cooling and vary the body's heat production to sustain thermal balance. Core temperature rises if heat gain exceeds heat loss, as readily occurs with vigorous exercise in a warm, humid environment; in contrast, core temperature falls in the cold, when heat loss exceeds heat production (McArdle, Katch, and Katch, 2001).



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(McArdle,Katch, and Katch, 2001).

Figure 2.1: Mechanism of thermoregulation

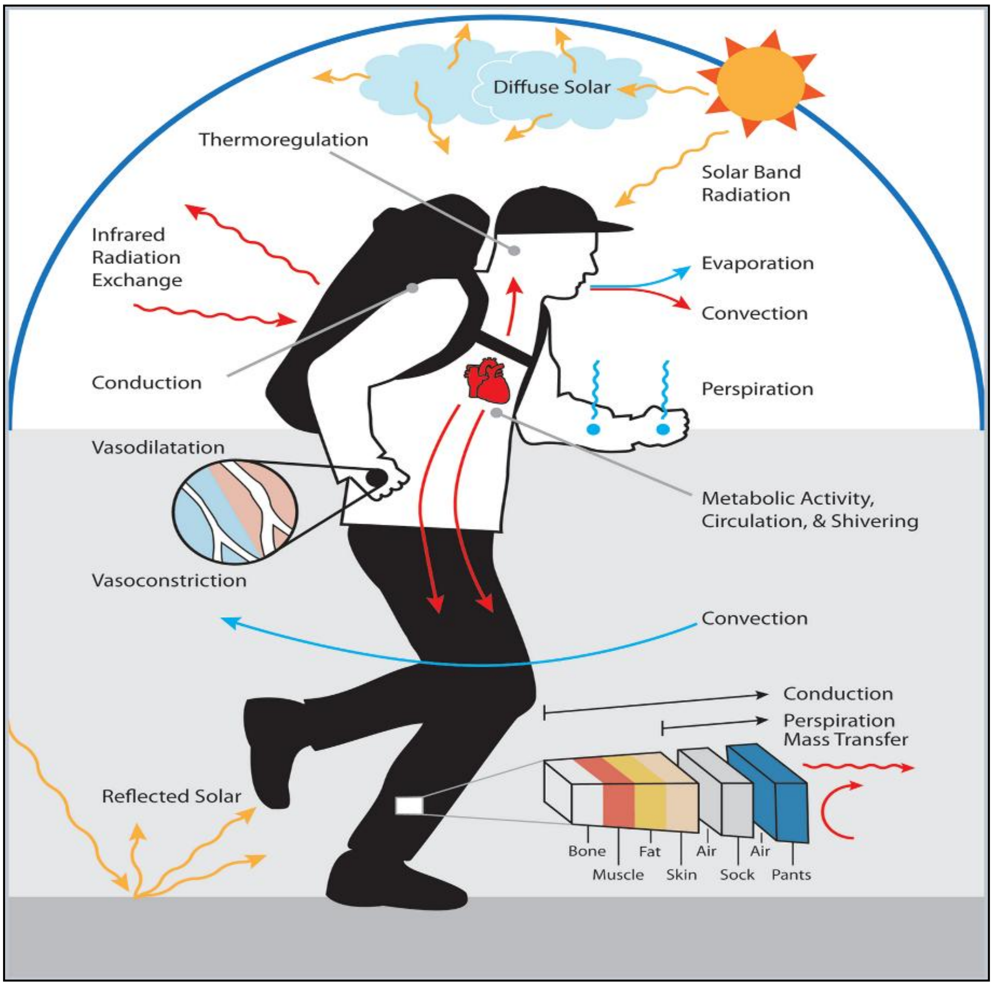

Figure 2.2: Thermoregulation in the heat (McArdle et al., 2001).

Table 2.1 presents thermal data for heat production and heat loss via sweating during rest and maximal exercise (McArdle et al., 2001).

Table 2.1: Thermodynamics during rest and exercise

Condition	**Rest**	**Maximal Exercise**
Body's heat production	-0.25LO$_2$-min^{-1}	-40LO$_2$-min^{-1}
1 LO$_2$ consumption – 4.82kcal	-1.2kcal- min^{-1}	-20.02kcal- min^{-1}
Body's capacity for evaporative cooling		**Maximal sweating**
		Each 1ml sweat evaporate = -30ml-min^{-1} = 18 kcal-min $^{-1}$
		0.6 kcal body heat loss
Core temperature increase	No increase	-1^{o}C every 5 to 7min

(McArdle et al**., 2001)**

According to Wendt, Van Loon & Lichtenbelt (2007) heat leaves the body via the physical mechanisms radiation, conduction and convection, but most importantly by means of insensible water loss and sweating. Insensible water loss comprises the loss of water through ventilation and diffusion, and there are no control mechanisms that govern the rate of insensible water loss for the purpose of temperature regulation. The loss of heat by the evaporation of sweat, on the other hand, can be controlled by regulating the rate of sweating, and sweating rates up to 3.5 l/hour have been reported in trained athletes. For every ml of water that evaporates from the body surface, 2.43kcal heat is lost. At rest in a comfortable environment, about 25% of heat loss is due to evaporation and these percentages change with the onset of exercise, especially when the ambient temperature approaches or is higher than an individual's core temperature (Wendt et al., 2007).

2.3.2 Hypothalamic regulation of temperature

The hypothalamus contains the central coordinating centre for temperature regulation. According to Wendt et al. (2007) several lesion and stimulation studies on the brain have identified the hypothalamus as the neural structure with the highest level of thermoregulatory integration. This group of specialised neurons at the floor of the brain acts as a "thermostat" – usually set and carefully regulated at 37^0C ± 1^0C- that continually makes thermoregulatory adjustments to deviations from a temperature norm.

According to McArdle et al. (2001) t*wo ways activate the body's heat-regulating mechanisms*:

- Thermal receptors in the skin provide input to the central control centre
- Changes in blood temperature perfusing the hypothalamus directly stimulate this area.

Investigators recorded a large number of heat-sensitive neurons and about one-third as many cold-sensitive neurons in the preoptic and anterior nuclei of the hypothalamus. These thermo-sensitive neurons effectively monitor the temperature of the blood flow to the brain and can thus detect changes in core temperature. In addition to sensing changes in core temperature, the preoptic-anterior hypothalamus also receives afferent sensory input from thermo receptors throughout the body, including the spinal cord, abdominal viscera, the greater veins and the skin. In this way, the thermo sensitive neurons in the hypothalamus compare and integrate the central and peripheral temperature information. As a result the hypothalamus is able to initiate the thermoregulatory response most appropriate for any given thermal stress. When temperature rises or falls above or below the critical threshold temperature of 37°C, the hypothalamus initiates heat regulation processes to either increase or decrease body temperature accordingly (Wendt*,* et al*.,* 2007).

According to Falk & Dotan, (2008), morphologically, children have a higher body surface area-to-mass ratio - a major factor in "dry" heat dissipation and effective sweat evaporation. Locomotion-wise, children are less economical than adults, producing more heat per unit body mass. Additionally, children need to divert a greater proportion of their cardiac output to the skin under heat stress. Thus, a larger proportion of their cardiac output is shunted away from the body's core and working muscles -- particularly in hot conditions. Finally, under all environmental conditions and allometric comparisons, children's sweating rates are lower than those of adults. The differences appear to suggest thermoregulatory inferiority, but no epidemiological data show higher heat-injury rates in children, even during heat waves. Falk & Dotan (2008) also suggest that children employ a different thermoregulatory strategy. In extreme temperatures, they may indeed be more vulnerable, but under most ambient conditions they are not necessarily inferior to adults. Children rely more on dry heat dissipation by their larger relative skin surface area than on evaporative heat loss. This also enables them to evaporate sweat more efficiently with the added bonus of conserving water better than adults.

2.3.3 Thermoregulation in heat stress and heat loss

Heat is produced by both endogenous sources (muscle activity and metabolism) and exogenous sources (transfer to the body when environmental temperature exceeds body temperature) (Brukner and Khan, 2012). The body’s thermoregulatory mechanisms primarily protect against overheating. Dissipating heat efficiently becomes crucial during exercise in hot weather when inherent competition exists between mechanisms that maintain a large muscle blood flow and thermoregulatory mechanisms. Physical characteristics such as total body mass, lean muscle mass, percentage of body fat, body surface, and surface area-to-mass ratio affect thermoregulation (Godek, Bartolozzi, Burkholder, Sugarman, & Dorshimer, 2006). Aerobic fitness, acclimatisation, clothing and equipment worn, and environmental considerations can contribute to the incidence of heat illness.

Brukner and Khan (2012) also stated that heavier sportspeople are particularly at risk because they produce more heat and have greater difficulty losing that heat adequately than do lighter sportspeople when both exercise at the same velocity in humid conditions. In contrast, because they produce less heat when exercising at the same velocity as heavier sportspeople, small sportspeople are especially advantaged when competing in prolonged events in the heat.

Additionally, dehydration (determined by how quickly fluids are lost via sweating combined with inadequate fluid intake) is considered one of the primary precursors to heat-related disorders (Cheuvront, Carter & Sawka, 2003). The basis for heat exchange from a human body to the environment occurs in 4 ways: conduction, convection, radiation and evaporation (**Table 2.2)**. All methods are dependent on the presence of a heat gradient (Howe & Boden, 2007, Brukner and Khan, 2012).

2.3.3.1 *Heat loss by radiation*

Objects continually emit electromagnetic heat waves. Because our bodies usually remain warmer than the environment, the net exchange of radiant heat energy moves through the air to solid, cooler objects in the environment. Loss of this heat gradient by certain environmental conditions can inhibit appropriate thermoregulation (Howe & Boden, 2007).

Heat radiation involves the mechanism of heat release via radiation through the skin in the infrared wavelength. This form of heat release only works at outside temperatures over 35^{o}C. In this regard, Gaffin and Moran (2001) also stated that this works well if the body temperature exceeds the ambient temperature. In the case of high ambient temperature, the heat gradient does not allow for heat loss from the body to the environment.

2.3.3.2 *Heat loss by conduction*

Heat exchange by conduction occurs in accordance to the law of Physics: it involves direct heat transfer from one molecule to another through a liquid, solid, or gas. The circulation transports most of the body heat to the shell, but a small amount continually moves by conduction directly through the deep tissues to the cooler surface. The rate of conductive heat loss depends on two factors: the temperature gradient between the skin and surrounding surface, and their thermal qualities (McArdle et al., 1994).

2.3.3.3 *Heat loss by convection*

At rest, when environmental temperature is below body temperature, thermal balance is maintained by convection of heat to skin surface and radiation of heat to the environment (Brukner and Khan, 2012). Convection is the cooling of the air around the body by way of cooler air passing over the warmer exposed skin.

This method depends on wind current to bring cooler air to the body or movement of the body through the environment to produce a heat gradient (e.g. with cycling). Lack of wind will reduce heat lost by convection (McArdle et al., 1994).

2.3.3.4 *Heat loss by evaporation*

As an individual starts to exercise and produce more heat, sweating provides compensatory heat loss through evaporation (Brukner and Khan, 2012). Evaporation provides the major defence against overheating. Water vaporising from the respiratory passages and skin surface continually transfers heat to the environment. Each litre of water that vaporises extracts 580 kcal from the body and transfers it to the environment.

Evaporation, of water from the body’s surface provides the major physiologic defence against overheating. Approximately 2 to 4 million sweat glands are distributed throughout the surface of the body. A cooling effect occurs as sweat evaporates. The cooled skin in turn serves to cool the blood that has been shunted from the interior to the surface. In addition to heat loss through sweating, about 350ml of water seep through the skin each day and evaporate to the environment as insensible perspiration (McArdle et al., 1994).

Table 2.2: Mechanisms of heat release during sporting exercise

Forms of heat release	Proportion of heat release (%)
Conduction	10 - 15
Convection	10 - 15
Radiation	10 - 15
Evaporation	60 - 80

(McArdle et al., 1994).

2.4 HEAT-RELATED ILLNESS IN ATHLETES

According to Marshall (2010) heat injury occurs when excessive internal thermal energy is generated or absorbed by the human body. Heat injury covers a wide range of conditions, ranging from swelling, rash and muscle cramps in its mildest forms, through to heat exhaustion and heat stroke in its more severe forms. These events are often referred to as heat ‘‘illness’’; however, since an uncontrolled energy transfer is occurring, heat illness is in fact a type of injury. We are mainly used to injuries that result from kinetic energy (such as a blow to the head causing concussion), but thermal energy is an equally potent source of energy for injury causation.

Heat illness is also generally defined in a dichotomous fashion, either present or absent, reflecting significant symptomatology associated with increased core temperature. In extreme situations, body temperatures can rise too high. Coris, Ramírez, and Durme (2004) also stated that heat illness is currently the third leading cause of death in high school athletes behind head trauma and cardiac disorders; it is also one of the leading causes of death in college athletes.

The American Academy of Pediatrics (2011) define exertional heat illness as a spectrum of clinical conditions that range from muscle (heat) cramps, heat syncope, and heat exhaustion to life-threatening heat stroke incurred as a result of exercise or other physical activity in the heat. They also recognise that appropriate and sufficient regular physical activity plays a significant part in enhancing and maintaining health. However, special consideration, preparation, modifications, and monitoring are essential when children and adolescents are engaging in sports or other vigorous physical activities in warm to hot weather.

According to Marshall (2010) extrinsic and intrinsic factors are the critical risk factors when exercising in the heat. Fundamentally, hot and humid climatic conditions are the single most critical predisposing risk factor. Armstrong, Casa and Millard-Stafford (2007) stated that factors that impede the body's ability to radiate heat, or increase heat absorption, are also considered to be risk factors. These include too high a level of exertion, too much clothing, dark-coloured clothing, insufficient rest breaks and lack of shade (Howe and Boden, 2007). Shea (2007) concludes that global warming has also been proposed as a risk factor.

Marshall (2010) also stated that intrinsic risk factors such as poor acclimatisation to exercise in hot climatic conditions is considered to be an important risk factor, as are poor physical fitness and obesity. It seems that there is considerable intrinsic variation between children in their response to heat and exercise, but the causes and determinants of this variation are unclear (Rowland, 2008). Exertional heat illness, including heat exhaustion and heat stroke, might occur even in a temperate environment, but the risk is highest when children and adolescents are vigorously active outdoors in hot and humid conditions American Academy of Pediatrics (2011). Heat stroke is currently the third leading cause of death in athletes behind cardiac disorders and head and neck trauma (Barrow and Clark, 1998; Kulka and Kenney, 2002).

From 1995 to 2002, heat- and dehydration-related deaths claimed 15 high school football players' lives (Eichelberger, 2003). In 2006, 20 football athlete deaths occurred due to heat and dehydration (Fox News, 2007). Three of the 20 deaths were college athletes, while 13 were middle and high school athletes.

In the USA, the National Center for Catastrophic Sports Injury Research reports that from 1995 to 2008, a total of 39 football players died from heat stroke (29 high schools, seven colleges, two professional and one sandlot). For the high school players, this translates to a death rate of 0.20 per 100 000 player-seasons. Based on the data collected by this centre, it seems that the majority of heat-related deaths in US children and adolescents occur in football, followed by cross-country and other running sports, but it should be noted that this reporting system is influenced by media coverage and volunteerism. Any physical activity in warm or hot conditions has the potential to induce heat injury, including indoor activities. For example, in 1997, three collegiate wrestlers died from heat-related conditions while trying to lose weight in preparation for match weigh-in (Marshal, 2010).

The question arises, why are children and adolescents vulnerable to heat injury? Rowland (2008) stated that children have traditionally been considered to be at increased risk for heat illness (heat stroke, heat exhaustion) during physical activities compared with adults, a supposition based on:

1) their perceived inferior thermoregulatory mechanisms, and

2) a greater incidence of heat stroke in the paediatric age group recorded during times of heat waves.

However, these reports of heat stroke have indicated an augmented risk restricted to infants and small children (<4 yr. old), which has been ascribed largely to dependency factors (such as parent neglect) and pre-existing chronic illness. How this vulnerability might be translated to child athletes or older children playing in the heat is not clear.

Marshall (2010) also stated as with risk factors, there are no controlled studies of interventions, and prevention recommendations have been based on clinical observation and physiological information. Nevertheless, a recent series of systematic reviews, consensus statements and expert opinions have generated a core set of prevention recommendations, and these are summarised in **Table 2.3.** As with risk factors, much of this information comes from studies of adults, and the extrapolation to youth populations is largely untested in empirical terms.

Table 2.3: Expert recommendations for the prevention of heat injury in young athletes

Expert recommendations for the prevention of heat injury in young athletes					
	American Academy of Paediatrics	**American College of Sports Medicine**	**National Athletic Trainers Association**	**National Centre for Catastrophic Sports Injury Research**	**Howe and Boden**
1. Acclimatise athletes gradually. Provide graduated activity sessions for the initial 10-14 days	✓	✓	✓	✓	✓
2. Know both the temperature and the humidity and reduce, modify activity when both are high. Use the wet bulb globe temperature as a guide.	✓	✓	✓	✓	✓
3. Provide frequent rest breaks and actively promote hydration.	✓	✓	✓	✓	✓
4. Educate healthcare providers, coaches, and others to observe for signs of heat illness. Circulate a written emergency plan.	-	✓	✓	✓	✓
5. Be prepared to rapidly lower core body temperature (e.g. large plastic outdoor swimming pool with ice water).	✓	✓	✓	✓	✓
6. Wear light-weight and light-coloured clothing. Wear only one layer.	✓	✓	✓	✓	✓

(Marshall, 2010).

However, there are three main reasons why children and adolescents are considered to be more vulnerable than adults to heat injury, namely physiological vulnerability, exposure vulnerability and social/behavioural reasons (Marshall, 2010).

2.4.1 Physiological vulnerability

According to Marshall (2010) it is widely believed that children are at increased risk of heat injury for physiological reasons. The reasons typically cited include: children’s bodies have a higher surface-area-to-volume ratio than adults, which increases their heat absorption rate under very hot climatic conditions; they have a lower rate of perspiration than adults, and thus a reduced ability to cool through sweating; their metabolic rate, and consequent heat production, per kilogram of body mass is higher than that of adults at the same work rate (Armstrong et al., 2007). Recently, these long-held beliefs have been challenged. It has been argued that these concepts are not well supported by exercise physiology data (Rowland, 2008). According to Rowland (2008) it is also clear that exercise physiology literature lacks definitive well controlled adult–child comparisons, and thus it is difficult to assess the validity of these long-held beliefs.

In summary, results of new research indicate that, contrary to previous thinking, youth do not have less effective thermoregulatory ability, insufficient cardiovascular capacity, or lower physical exertion tolerance compared with adults during exercise in the heat when adequate hydration is maintained. Accordingly, besides poor hydration status, the primary determinants of reduced performance and exertional heat-illness risk in youth during sports and other physical activities in a hot environment include undue physical exertion, insufficient recovery between repeated exercise bouts or closely scheduled same-day training sessions or rounds of sports competition, and inappropriately wearing clothing, uniforms, and protective equipment that play a role in excessive heat retention. Because these known contributing risk factors are modifiable, exertional heat illness is usually preventable. With appropriate preparation, modifications, and monitoring, most healthy children and adolescents can safely participate in outdoor sports and other physical activities through a wide range of challenging warm to hot climatic conditions (American Academy of Pediatrics, 2011).

2.4.2 Exposure vulnerability

Children and adolescents may be at increased risk simply because they are more likely to be exposed to vigorous physical exercise during the warm summer months. For example, each year, August preseason football practices around the US expose 17 times more high school age athletes than collegiate athletes to physical activity in hot conditions. This is because there are 1.1 million high school football players compared with 65 000 collegiate football players nationally. Additionally, children and adolescents tend to attend (and staff) outdoor summer sports camps, while adults seek refuge in indoor environments (Marshall, 2010). However, according to Rowland (2008) it is a fact that cases of serious heat illness in child athletes are conspicuously absent from the medical literature, and informal opinion suggests that such events are rare.

2.4.3 Social, behavioural vulnerability

Primary prevention strategies for heat injury include frequent rest and hydration breaks. However, there is considerable variation between individuals in their thirst response and need for water (Decher, Casa and Yeargin, 2008) and between coaches in their practices regarding provision of water breaks (Luke, Bergeron, and Roberts, 2007). A study of youth in summer sports camps found that personal knowledge about the importance of hydration did not correlate with actual hydration status, possibly indicating the importance of social and structural factors in moderating hydration and rest break behaviour. Children and adolescents who need water or rest, but are being supervised by adult coaches, may be reluctant to interrupt structured exercise drills to take a break. They may also be prone to peer pressure to ‘‘tough the heat out”. Up to 56% of youth in summer sports camps experience significant or severe dehydration (Decher et al., 2008).

Heat illness can present itself in mild or severe cases (Coris et al*.,* 2004). Mild cases of heat illness include heat oedema, heat cramps, heat syncope, and heat exhaustion, while heat stroke is the severest form of heat illness. Heat cramps result primarily from fluid and electrolyte losses. Heat exhaustion results from cardiovascular responses to dehydration. Other forms of heat illness are more related to environment and have less to do with hydration status. Body heat production while exercising is 15 to 20 times greater than at rest. Body temperature will rise one degree Celsius every five minutes without any adjustments being made (Coris et al., 2004). When exercising in the heat, other factors such as humidity, air motion, solar load, and choice of clothing contribute to the amount of sweat lost (Sawka et al., 2005).

The graded continuum of heat illness progresses from very mild to more serious disease to a life-threatening condition known as heat stroke **(Table 2.4)**. There is no evidence that mild heat illness (heat oedema, heat rash, heat cramps, or heat syncope) will progress to severe disease if untreated. However, the development of heat exhaustion is significant. Without treatment, heat exhaustion has the potential to progress to heat stroke (Allyson et al., 2007).

Table 2.4: Criteria for diagnosis of heat illness

CONDITION	CORE TEMPERATURE °F (°C)	ASSOCIATED SYMPTOMS	ASSOCIATED SIGNS
Heat oedema	Normal	None	Mild oedema in dependent areas (ankles, feet, hands)
Heat rash	Normal	Pruritic rash	Papulovesicular skin eruption over clothed areas
Heat syncope	Normal	Dizziness, generalised weakness	Loss of postural control, rapid mental status recovery once supine
Heat cramps	Normal or elevated but <104°F (40°C)	Painful muscle contractions (calf, quadriceps, abdominal)	Affected muscles may feel firm to palpation
Heat exhaustion	98.6°F–104°F (37°C–40°C)	Dizziness, malaise, fatigue, nausea, vomiting, headache	Flushed, profuse sweating, cold clammy skin, normal mental status
Heat stroke	>104°F (40°C)	Possible history of heat exhaustion symptoms before mental status change	Hot skin with or without sweating, CNS disturbance (confusion, ataxia, irritability, coma)

(Allyson et al., 2007)

2.4.4 Heat oedema

According to Allyson et al. (2007) very mild forms of heat illness occur in the form of heat oedema and heat rash (also known as prickly heat or miliaria rubra). Heat oedema appears as dependent soft tissue swelling, usually in the lower extremities, in a person lacking acclimatisation. Peripheral vasodilatation to produce heat loss leads to pooling of interstitial fluid in the distal extremities. This leads to an increase in vascular hydrostatic pressure and resultant third spacing of intravascular fluid into the surrounding soft tissue. The condition is more commonly seen in older adults who enter a tropical climate without proper acclimatisation.

2.4.5 Heat rash

Miliaria rubra (i.e. heat rash or prickly heat) presents as a pinpoint papular erythematous, often intensely pruritic, eruption in areas covered with clothing. It commonly presents in the waist or over highly sweaty areas such as the trunk or groin. Profuse sweating saturates the skin surface and clogs the sweat ducts. Obstruction of the ducts results in leakage of eccrine sweat into the epidermis.

Secondary infection with staphylococcus may produce prolonged symptoms (Allyson et al., 2007).

2.4.6 Heat syncope

Heat syncope occurs with orthostatic hypotension resulting from peripheral vasodilatation (physiologic response to heat production) and venous pooling. Prolonged standing after significant exertion and rapid change in body position after exertion, such as from sitting to standing, may lead to heat syncope (MacKnight and Mistry, 2005). Athletes with heat syncope tend to recover their mental status quickly once supine, as blood flow to the central nervous system returns.

2.4.7 Heat cramps

Brukner and Khan (2012:1138) stated that the popular belief that cramps are caused by severe dehydration and large sodium chloride losses that develop during hot conditions has no scientific basis. According to Stofan (2005) there is no scientific proof that muscle cramps are due to excess loss of sodium, as was believed for many years. Recent studies prove that muscle cramps are more likely because of spinal reflex activity due to Golgi-Tendon end organ exhaustion. Painful muscle cramps most commonly involve the quadriceps, hamstrings, gastrocnemius, and abdominal musculature. Cramps often occur in these active muscle groups when they have been challenged by a prolonged exercise event of more than 2 hours. Likely a result of fluid and sodium depletion, heat cramps are more common in individuals with heavy amounts of salt in their sweat (Stofan, 2005). Tennis players, American football players, steel mill workers, and military members who deploy to hot environments have a high incidence of heat cramps (Ganio, Casa, Armstrong and Maresh, 2007).

The more modern hypothesis proposes that cramps probably result from alterations in spinal neural reflex activity activated by fatigue in susceptible individuals. According to Brukner and Khan (2012) the term “heat cramps” should be abandoned as it clouds understanding of the possible neural nature of this connection.

2.4.8 Heat exhaustion

The American Academy of Pediatrics (Pediatrics, 2011, defines heat exhaustion as moderate heat illness, characterised by the inability to maintain blood pressure and sustain adequate cardiac output, that results from strenuous exercise or other physical activity, environmental heat stress, acute dehydration, and energy depletion. Signs and symptoms include weakness, dizziness, nausea, syncope, and headache; core body temperature is <104°F (40°C).

This condition presents with postural hypotension after completing exercise. This is caused by “blood pooling” in the peripheral dilated veins of the lower extremities because there is no “pump function” from the calf muscles after stopping exercises (Allyson et al., 2007). Dehydration has no contribution to this condition, due to the fact that the athlete does not collapse during exercise, but afterwards. The Barcroft-Edholm reflex may play a role due to lowering right atrial pressure. Athletes with heat exhaustion frequently complain of fatigue, malaise, muscle cramps, nausea, vomiting, and dizziness. The patient should, by definition, be alert and oriented and have normal cognition. There may be evidence of circulatory compromise seen as tachycardia or hypotension. Orthostatic syncope can occur but should be followed by rapid return to normal CNS function. Often the skin is profusely diaphoretic. Identification of heat exhaustion is of utmost importance in order to avoid progression to heat stroke. If there is any question regarding the mental status, it is prudent to treat for heat stroke and continue evaluation for other conditions such as hyponatremia, hypoglycaemia, seizure, or closed head trauma (Allyson et al., 2007).

2.4.9 Heat stroke

Exertional heat stroke is a severe multisystem heat illness, characterised by central nervous system abnormalities such as delirium, convulsions, or coma, endotoxemia, circulatory failure, temperature-control dysregulation, and, potentially, organ and tissue damage, that results from an elevated core body temperature (>104°F [>40°C]) that is induced by strenuous exercise or other physical activity and typically (not always) high environmental heat stress (American Academy of Pediatrics, 2011).

According to Armstrong & Casa (1993) heatstroke is the third leading cause of death in athletics, and an important cause of morbidity and mortality in exercising athletes.

Cerebral dysfunctions (stupor, coma), increase irritability, convulsions and collapsing without loss of consciousness can be a result of rectal temperatures above 41°C.

When running, the metabolic rate is a function of running speed and body mass. Higher rectal temperatures are usually associated with symptoms that include dizziness, weakness, nausea, headache, confusion, disorientation and irrational behaviour, including aggressive combativeness or drowsiness progressing to coma. The presence or absence of sweating does not influence the diagnosis. Examination reveals the patient is hypotensive and has tachycardia (Brukner and Khan, 2012).

The diagnosis of heat stroke is dependent on accurate core body temperature recordings >40°C (104°F) and CNS dysfunction. In situations in which cooling has already begun en route, temperature criteria may not be met. When CNS changes are present but core temperature is below 40°C, it is important to initiate treatment for heat stroke while exploring the differential diagnosis of the patient’s mental status changes. The most reliable measurement of core temperature is obtained via the rectal route. This may be uncomfortable to patients, but it is the standard method to measure core body temperature (Falzon, Grech, Caruana, Magro and Attard-Montalto, 2003).

According to Brukner and Khan (2012), if, or during exercise, a previously healthy athlete shows marked changes in mental functioning (e.g. collapse with unconsciousness or a reduced level of consciousness (stupor, coma) or mental stimulation (irritability, convulsions) in association with a rectal temperature above 41°C), the diagnosis of heat stroke is confirmed and warrants immediate initiation of cooling.

Athletes with heat stroke have often progressed through heat exhaustion without recognition of the condition. Their team mates or coaches may have observed vomiting, fatigue, or loss of athletic ability that progressed to confusion, ataxia, or agitation. Although in the setting of classic heat stroke a person’s skin may be identified as dry and hot (anhidrosis), this is often not the case with exertional heat stroke. Recognition of profuse sweating should not eliminate the diagnosis of heat stroke (Allyson et al., 2007).

Factors that predispose to heatstroke are those that disturb the equilibrium between the rate of heat production and heat loss. The rate of heat production is determined by the athlete's mass and running speed. The rate of heat loss is controlled by air temperature, humidity and the rate of wind movement across the athlete's body. One factor that determines the rate at which the athlete loses heat is his clothing, because the more clothing he wears, the less heat he will lose by means of convection and sweating. The athlete's state of heat acclimatisation is another factor, because heat acclimatisation increases both the athlete's ability to lose heat by sweating and his resistance to an elevated body temperature and his state of hydration, because dehydration impairs the ability to lose heat by sweating. Finally, it is clear that only certain individuals are prone to heatstroke for unknown reasons. It seems likely that they have a hereditary abnormality of muscle cell metabolism (Allyson et al., 2007).

2.4.10 Hyponatremia

According to Ganio et al. (2007) it is especially important to mention that the differential diagnosis of exertional heat stroke is exertional hyponatremia. Defined by serum sodium levels <130 mmol/L, this type of hyponatremia can present with a clinical appearance similar to heat stroke, with mental status changes and an altered level of consciousness. Exertional hyponatremia is distinguished from heat illness by a normal core body temperature (Ganio et al., 2007; Seto, 2005). However, the first international consensus statement on exercise-associated hyponatremia (EAH) has concluded that the role of sodium loss in the development of exercise-associated hyponatremia has yet to be established (Brukner and Khan, 2012).

Noakes, Sharwood, Speedy et al. (2005) identify three factors that can explain why the range of serum sodium concentrations after exercise is so variable even when the weight change is the same. Thus, to develop EAH, subjects must:

1. Over-drink, usually by drinking in excess of 750ml per hour for at least four hours during exercise,
2. Fail adequately to suppress the inappropriate secretion of the anti-diuretic hormone (ADH) arginine/vasopressin, and
3. Either inappropriately osmotically inactivate circulating serum ionized sodium or fail to mobilise osmotically inactive sodium to maintain a normal serum sodium concentration in an expanded total body water.

The conclusion of these findings is that the avoidance of over-drinking is the sole factor required to prevent exercise-associated hyponatremia (Brukner and Khan, 2012).

Risk factors for hyponatremia also differ somewhat from those for heat stroke (**see Table 2.5**). These athletes are typically female, have slower race times, lower body weights, and have a high availability of fluids. Severe hyponatremia (serum sodium <120 mmol/L) can precipitate seizures, coma, and death. Treatment of the condition is beyond the scope of our article, but often begins with oral sodium solutions if mild and progresses to intravenous hypertonic saline for severe cases (Ganio et al., 2007; Seto, 2005).

Table 2.5: Summary of risk factors for heat illness

Internal Factors	**External Factors**
Age (<15 years or >65 years)	Activity level
Alcohol consumption	Excessive clothing wear
Comorbid medical conditions—respiratory, cardiovascular, hematologic	Lack of water or sufficient shade
Dehydration	Temperature (ambient)
History of heat-related illness	Humidity
Lack of air conditioning	Wet bulb globe temperature
Lack of appropriate sleep	
Medications or supplements	
Obesity	
Over motivation	
Poor acclimatisation	
Poor cardiovascular fitness	
Recent febrile illness	
Sickle cell trait	
Skin condition—eczema, psoriasis, burns, etc	
Social isolation	
Sunburn	
Use of psychiatric medications	

(Allyson et al**., 2007)**

According to Beltrami, Hew-Butler and Noakes (2008) drinking policies during exercise have changed substantially throughout history. Since the mid-1990s, however, there has been an increase in the number of organisations that encourage over-drinking by athletes. This kind of advice is subsequently printed in many exercise physiology textbooks and remains current at the present time. The scientific community, however, is slowly moving away from ‘‘blanket range’’ advice for drinking during exercise and moving towards more modest and individualised hydration guidelines in which thirst is being recognised as the best physiological indicator of a subject’s fluid needs during exercise (Noakes, 2007).

2.5 PREVENTION MEASURES FOR HEAT ILLNESS

As with risk factors, there are no controlled studies of interventions, and prevention recommendations have been based on clinical observation and physiological information. Nevertheless, a recent series of systematic reviews, consensus statements and expert opinions have generated core set of prevention recommendations (Howe and Boden, 2007; Armstrong et al., 2007; Mueller and Colgate, 2009; Casa and Csillan, 2009; Brukner and Khan, 2012).

As with risk factors, much of this information comes from studies of adults, and the extrapolation to youth populations is largely untested in empirical terms. Logically, frequent rest breaks, reductions in exercise in very hot conditions and adequate hydration are expected to reduce risk in adolescents (Casa and Csillan, 2009; Bergeron, McKeag and Casa, 2005). Some studies have found that children will voluntarily drink more fluid during exercise in warm or hot environments when provided with a ‘‘sports drink’’ (e.g. Gatorade) than when plain water is given (Rivera-Brown, Gutie´rrez and Gutie´rrez et al., 1999; Bergeron , Waller and Marinik, 2006) but other studies have not replicated this finding (Rivera-Brown et al., 2008). This variation likely reflects social factors and individual differences in behavioural patterns of voluntary water consumption. Irrespective of whether they increase consumption, electrolyte-based drinks are preferred, since overconsumption of water can disrupt the body’s electrolyte balance, resulting in hyponatremia. As indicated above, both heat and humidity play an important role in the onset of heat injury. Wet bulb globe temperature is an index that combines ambient temperature and ambient humidity data into one overall index. Published guidelines for exertion at various levels of wet bulb globe temperature are available (Howe and Boden, 2007; Armstrong et al., 2007).

However, results of new research indicate that, contrary to previous thinking, youth do not have less effective thermoregulatory ability, insufficient cardiovascular capacity, or lower physical exertion tolerance compared with adults during exercise in the heat when adequate hydration is maintained. Accordingly, besides poor hydration status, the primary determinants of reduced performance and exertional heat-illness risk in youth during sports and other physical activities in a hot environment include undue physical exertion, insufficient recovery between repeated exercise bouts or closely scheduled same-day training sessions or rounds of sports competition, and inappropriately wearing clothing, uniforms, and protective equipment that play a role in excessive heat retention. Because these known contributing risk factors are modifiable, exertional heat illness is usually preventable. With appropriate preparation, modifications and monitoring, most healthy children and adolescents can safely participate in outdoor sports and other physical activities through a wide range of challenging warm to hot climatic conditions (Pediatrics 2011).

Brukner and Khan, (2012) provided these guidelines for the prevention of heat illness, and according to them most cases of heat illness can be prevented if the following guidelines are followed: (see full Guidelines in Brukner and Khan, 2012).

1. Perform adequate conditioning.
2. Undergo acclimatisation if competing in unaccustomed heat or humidity.
3. Avoid adverse conditions.
4. Alter training times.
5. Wear appropriate clothing.
6. Drink appropriate amounts of fluids before the event.
7. There is no evidence that fluid ingestion during exercise can prevent heat stroke in those predisposed to develop the condition.
8. To minimise the uncomfortable sensations of thirst and so to optimise performance during exercise, sportspeople can be assured that they need drink only according to the dictates of their thirst.
9. Ensure sportspeople and officials are well educated.
10. Provide proficient medical support.

2.6 TREATMENT OF HEAT ILLNESS

The American Academy of Pediatrics (Pediatrics, 2011), defines heat injury as the profound damage and dysfunction to the brain, heart, liver, kidneys, intestine, spleen, or muscle induced by excessive sustained core body temperature associated with incurring exertional heat stroke, especially for those victims in whom signs and/or symptoms are not promptly recognised and are not treated effectively (rapidly cooled) in a timely manner.

Treatment protocols for heat illness follow a critical common theme—lower the core body temperature to an acceptable level (37.5–38°C) as quickly as possible. A major determinant of outcome in heat stroke is the duration of hyperthermia. The human critical thermal maximum is 41.6°C to 42°C for 45 minutes to 8 hours. Beyond this time frame, lethal or near-lethal injury occurs and is irreversible *(*Bouchama and Knochel, 2002).

Treatment of all heat illness should begin with an assessment of airway, breathing, and circulation (ABCs), and transfer of the patient to a cooler environment. With exertional heat illness, this may be as simple as taking a player off the field to sit still on the bench or bringing the athlete to a shaded area. Most beneficial, of course, would be to move the patient to an air-conditioned building if available at the time of evaluation. These treatments should be universally employed in the setting of heat illness.

2.6.1 Treatment of heat oedema

Oedema of the hands and feet should be mild and improve with elevation and relative rest. Compressive stockings may be helpful in cases that are slow to resolve. Ensuring that the athlete is well hydrated and has adequate salt intake is important as these conditions may delay resolution. Diuretics are not helpful as they further reduce intravascular volume and can exacerbate the condition. Generally, this condition improves in 7 to 14 days as acclimatisation occurs or sooner if the athlete returns to his or her home climate (Allyson et al., 2007).

2.6.2 Treatment of heat rash

According to Seto (2005) cooling the area and reducing clothing coverage where possible will help resolution. The rash is benign but often takes a week or more to resolve completely. Habif (2004) stated that the application of a mild anti-inflammatory lotion such as Desonide may relieve symptoms and shorten the duration of the rash.

2.6.3 Treatment of heat syncope

The treatment of heat syncope involves safely moving the patient into a supine position in a cool location. Often this alone will resolve the condition as cerebral blood flow is restored. Elevating the patient's legs will aid in venous return of blood flow. Intravenous fluids may be necessary to correct volume depletion that likely contributed to the syncopal event (Seto, 2005).

2.6.4 Treatment of heat cramps

Stretching of affected muscles, cooling with ice, massage of cramped muscles, and removal from activity are generally effective (Brukner and Khan, 2012). According to Seto (2005) oral replenishment of fluid and electrolytes must be initiated for prevention of subsequent cramping but are generally not effective acutely for treatment. In severe cases or when the symptoms continue to rebound, intravenous hydration with 0.9% normal saline is indicated. This is often rapidly curative.

2.6.5 Treatment of heat exhaustion

According to Allyson et al. (2007) an athlete with heat exhaustion often presents with several concomitant symptoms. It is important to consider heat illness in the athlete who complains of nausea, vomiting, headache, or dizziness. Left untreated, this condition can progress to heat stroke. Core temperature readings, ideally with a rectal thermometer, are necessary to accurately identify athletes at risk of permanent injury and need for higher levels of care. If an athlete has mild illness and normal vital signs, cooling the athlete with removal from the heat and oral rehydration with cool salt-containing fluids (i.e. sports drinks) will often be sufficient to lower temperature effectively. If there are more serious symptoms present such as abnormal vital signs, vomiting, or failure to improve with the above conservative techniques, intravenous fluids are indicated. Ice bags applied to the axilla and groin can also produce rapid lowering of body temperature and are recommended when repeat monitoring of core body temperature is available.

2.6.6 Treatment of heat stroke

According to Brukner and Khan (2012) it appears that there is an individual susceptibility to heat stroke. Heat stroke demands an aggressive approach to lowering body temperature. Direct correlation between duration of elevated temperature and morbidity/mortality of a patient has been established (Lugo-Amador, Rothenhaus and Moyer, 2004). The more rapidly the rectal temperature is reduced to 38 degrees Celsius the better the prognosis. The patient should be placed in a bath of ice water for 5 – 10 minutes. The body temperature should decrease to 38 degrees Celsius within this time (Brukner and Khan, 2012). Another report suggested improved survival if cooling to the same level occurs within 30 minutes (Lugo-Amador et al., 2004). As previously stated, all treatment begins with an assessment of the ABCs, movement to a cooler location, and removal of clothing. This is unlikely to be effective alone in heat stroke, which requires more aggressive treatment.

There are many documented heat cooling techniques, but the level of effectiveness is controversial. In a comprehensive review of cooling techniques, it was demonstrated that immersion in ice water is the most effective method to produce total body cooling (Smith, 2005). There are obvious problems and limitations to this method. Unless heat illness is anticipated, ice water immersion baths and the personnel required to monitor a patient in a bath are not readily available. If a patient is severely ill, immersion may limit the ability to monitor cardiovascular status and can be dangerous in the setting of reduced consciousness. Treatment with intravenous fluids can be difficult when the patient’s body, other than the arm with the intravenous access, is immersed in the ice bath. Concern regarding peripheral vasoconstriction and slower cooling rates in patients immersed has not been proven experimentally.

2.6.7 Field treatment of heat stroke

Operationally, paediatricians, coaches, and administrators need to make appropriate recommendations and “on-the-field” decisions to improve safety and minimise exertional heat-illness risk for a team or event as a whole. However, given individual variations in health status, conditioning, or other circumstances, some participants might not require the same heightened concern as other young athletes who might need implementation of additional exertional heat-illness prevention measures and closer monitoring in the same or a less challenging environment (American Academy of Pediatrics, 2011).

On-field treatment of heat stroke requires common sense and use of available resources. The initial step in heat illness treatment is to recognise an athlete in trouble. Often team mates or coaches are made aware of athletes who are not feeling well before the medical staff is apprised. Early symptoms such as dizziness, nausea, malaise, and fatigue may not be reported to the medical staff by the athlete. The coaches and athletes should be taught the signs and symptoms of heat illness and be instructed to notify the medical staff if any exist. Treatment for mild heat illness should be initiated as rapidly as possible to avoid progression to severe heat stroke (**see Table 2.6)**. Evaluation of the ABCs is the first critical step. In addition to moving the patient and removing equipment and clothing, ice packs to the axilla, groin and neck are often the most available resource and should be used. If the athlete is able to drink fluids, cool sports drinks or water should be encouraged. When available, a rectal temperature should be taken. Periodic questioning of the athlete to assess mental status changes will help alert the staff of a worsening condition. If the temperature is >104°F or if the mental status is unstable, elevation of the level of medical care is critical. Accessing the emergency medical system rapidly will allow for faster implementation of advanced care techniques, such as cooling with fans or using intravenous therapy.

Table 2.6: On-Field Treatment of Heat Stroke

Emergency Medical System (EMS)
Recognize there is an athlete with signs or symptoms of heat illness.
Initiate cooling methods: move to cool area, ice bags to groin/axilla/neck, ice water tub immersion, and fan-sprayed mist.
Assess for mental status changes.
Assess need for rectal temperature (and repeat during cooling every 3–5 min).
—Encourage liberal oral fluid intake with cool sports drinks or water if able to tolerate.
—Check blood glucose and sodium levels if available.
Access EMS immediately if the athlete has any of the following:
Altered mental status
Temperature elevated >104°F
Persistent vomiting (unable to rehydrate orally)

(Allyson et al**., 2007)**

2.6.8 Treatment of Hyponatremia

According to Brukner and Khan (2012) hyponatremia (EAH exercise–associated hyponatremia) is perhaps the most important differential diagnosis in sportspeople who seek medical attention at an event undertaken in the heat, particularly in endurance events lasting four or more hours. Under no circumstances should any hypotonic or isotonic fluids be given to unconscious or semiconscious sportspeople with EAH. They recommended some or all of the following interventions:

- fluid restriction
- diuretics
- intravenous hypertonic (3-5%) saline at rates of about 100ml/hr.

In summary it is essential (Brukner and Khan, 2012) that physicians caring for sportspeople with EAH are aware of the correct management of this condition. The current management includes:

- bladder catheterisation to monitor the rate of urine production during recovery - spontaneous recovery will occur if adequate amounts of urine (>500ml/hr) are passed
- no fluids by mouth - salt tablets and sodium containing foods can be given

- high sodium (3%-5%) solutions given intravenously provided they are infused slowly (50-100ml/hr)
- use of diuretics.

2.6.9 Return to play after heat illness

For mild forms of heat illness, proper hydration will allow an athlete to return to play within a 24-hour period. In the case of exertional heat stroke, further monitoring is warranted before returning to competition. A physician should evaluate any athlete with exertional heat stroke. Risk factors for heat stroke should be thoroughly addressed. Before returning to play, an athlete must be asymptomatic and all laboratory tests and vital signs should have normalised (Mercer and, Densmore, 2005). It is also prudent to monitor body weight until normalisation. After treatment of the acute heat stroke event, it has been suggested that an athlete wait at least 1 week to return to play. The task force recommends a gradual and monitored return to exercise including progressive exposure to heat and level of sports equipment. Waiting for a period of 48 to 72 hours until return to duty has been used in a military setting when the heat stroke was relatively mild (i.e. rapid CNS recovery and normal laboratory testing). Each exertional heat stroke case must be considered independently. Overall, the severity of heat stroke illness should dictate the delay in return to play for an individual athlete.

However, according to American Academy of Pediatrics, (2011) the field evidence is not currently sufficient to optimally guide paediatricians, coaches, administrators, and youth sport governing bodies in making the most appropriate and advantageous modifications to play and practice specific to heat safety or deciding when to cancel activities altogether if necessary. Accordingly, parents, teachers, coaches, athletic trainers, and paediatricians as well as youth sports governing bodies and administrators should always emphasise and use suitable prevention strategies, to the best of their ability, to improve safety and appropriately minimise the risk of exertional heat illness for all children and adolescents during exercise, sports participation, and other physical activities in warm to hot weather. To this end, the American Academy of Pediatrics recommends the following:

1. Community and team/school physicians as well as athletic directors, community parks and recreation programmes and youth sport governing bodies should emphasise comprehensive awareness, education, and

implementation of effective exertional heat-illness risk-reduction strategies to coaches and their staff, athletic trainers, teachers, administrators, and others who oversee or assist with exercising children and adolescents and youth sports, especially for those involved with youth and preseason high school American football.

2. Trained personnel and facilities capable of effectively treating all forms of heat illness, especially exertional heat stroke by rapidly lowering core body temperature, should be readily available on site during all youth athletic activities and community programmes that involve vigorous physical activity and are held in the heat.

3. Children and adolescents should be regularly educated on the merits of proper preparation, ample hydration, honest reporting, and effectively managing other factors under their control, such as recovery and rest, which will directly affect exercise-heat tolerance and safety.

4. Each child and adolescent should be given the opportunity to gradually and safely adapt to preseason practice and conditioning, sport participation, or other physical activity in the heat by appropriate and progressive acclimatisation. This process includes graduated exposure (typically over a 10- to 14-day period) to the environment, intensity, duration, and volume of physical activity and to the insulating and metabolic effects of wearing various uniform and protective-equipment configurations. Specific guidelines for American youth football are available and can be used as a basis for developing other youth sports-acclimatisation and practice-modification/monitoring strategies.

5. Sufficient, sanitary and appropriate fluid should be readily accessible and consumed at regular intervals before, during, and after all sports participation and other physical activities to offset sweat loss and maintain adequate hydration while avoiding over-drinking. Generally, 100 to 250 ml (approximately 3–8 oz) every 20 minutes for 9- to 12-year-olds and up to 1.0 to 1.5 L (approximately 34–50 oz) per hour for adolescent boys and girls is enough to sufficiently minimise sweating-induced body-water deficits during exercise and other physical activity as long as their pre-activity hydration status is good. Pre-activity to post-

activity body-weight changes can provide more specific insight to a person's hydration status and rehydration needs. Although water is often sufficient to maintain adequate hydration, long-duration (e.g. ≥1-hour) or repeated same-day sessions of strenuous exercise, sport participation, or other physical activity might warrant including electrolyte-supplemented beverages that emphasise sodium to more effectively optimise rehydration. This is especially justified in warm- to hot-weather conditions, when sweat loss is extensive.

6. Exercise, sport participation, and other physical activity should be modified for safety in relation to the degree of environmental heat stress: air temperature, humidity, and solar radiation, as indicated by the heat index or wet-bulb globe temperature (WBGT), for those with access to such a device. Effective modifications include lowering the intensity and/or shortening the activity duration and increasing the frequency and duration of breaks, which would preferably be in the shade. Individual medical conditions and other risk factors identified by a pre-participation physical examination or as indicated by a more recent change in health status that could lower tolerance for exercise in the heat and increase risk for exertional heat illness should also prompt these and additional modifications. Any child or adolescent should avoid or limit exercise, sport participation, or other physical activity in the heat if he or she is currently ill or is recovering from an illness, especially those involving gastrointestinal distress (e.g. vomiting, diarrhoea) and/or fever.

7. Supervisory staff such as coaches, athletic trainers, physical education teachers, and playground aides should receive appropriate training and closely monitor all children and adolescents at all times during sports and other physical activity in the heat for signs and symptoms of developing heat illness. Any significant deterioration in performance with notable signs of struggling, negative changes in personality or mental status, or other concerning clinical markers of well-being, including pallor, bright-red flushing, dizziness, headache, excessive fatigue, vomiting, or complaints of feeling cold or extremely hot, should be sufficient reason to immediately stop participation and seek appropriate medical attention for those affected. First aid for evolving heat illness should not be delayed. Anyone experiencing exertional heat

illness should not return to practice or competition, recreational play, or other physical activity for the remainder of the current session, game/match, or play/activity period.

8. An emergency action plan with clearly defined written protocols should be developed and in place ahead of time. Emergency medical services (EMS) communication should be activated immediately for any child or adolescent who collapses or exhibits moderate or severe central nervous system dysfunction or encephalopathy during or after practice, competition, or other physical activity in the heat, especially if the child or adolescent is wearing a uniform and/or protective equipment that is potentially contributing to additional heat storage. Although treatment should not be delayed pending core body-temperature verification, when feasible, rectal temperature should be promptly checked by trained personnel and, if indicated (rectal temperature > 40°C [104°F]), on-site whole-body rapid cooling using proven techniques should be initiated without delay. This process includes promptly moving the victim to the shade, immediately removing protective equipment and clothing, and cooling by cold- or ice-water immersion (preferred, most effective method) or by applying ice packs to the neck, axillae, and groin and rotating ice-water–soaked towels to all other areas of the body until rectal temperature reaches just under 38°C (approximately 102°F) or the victim shows clinical improvement. If rectal temperature cannot be assessed in a child or adolescent with clinical signs or symptoms suggestive of moderate or severe heat stress, appropriate treatment should not be delayed. Prompt rapid cooling for 10 to 15 minutes and, if the child or adolescent is alert enough to ingest fluid, hydration should be initiated by attending staff while awaiting the arrival of medical assistance.

9. To improve athlete safety and performance, youth sports governing bodies, tournament directors, and other event administrators should provide adequate rest and recovery periods of 2 hours or *more* between same-day contests in warm to hot weather to allow sufficient recovery and rehydration.

10. In conditions of extreme heat or humidity when children or adolescents can no longer maintain thermal balance, safety should be the priority, and outdoor contests and practice sessions should be cancelled or rescheduled to cooler times, even if it means playing or practicing very early in the day or later in the evening (American Academy of Pediatrics, 2011).

2.7 HYDRATION

According to Sawka et al. (2005) proper hydration is important for all body functions. Water is the main constituent of the human body; for example, in an average adult male, body water is 50% to 70% of body weight. The effect of body water balance on aspects of exercise performance has been extensively researched and, in recent years, reviewed comprehensively. The routes of water loss from the body are the urinary system, the skin, the gastrointestinal tract, and the respiratory surfaces. The primary avenues for restoration of water balance are fluid and food ingestion, with water from oxidation making a minor contribution (Howe & Boden, 2007). The volumes of water that individuals obtain from food and drinks are highly variable, although it is generally reported that the majority comes from liquids, with a smaller, although still significant, proportion from solid foods.

Body water loss in humans results in fluid losses from both the intracellular and extracellular fluid compartments (Costill, Cote, & Fink, 1976). The fluid losses, however, can cause very different effects on the remaining body water pools depending on the type of water loss that occurs. Hypotonic water loss, as can occur with sweating, results in an increase in body fluid tonicity, while isotonic loss causes a net fluid loss but no increase or decrease in body fluid tonicity. Hypertonic fluid losses, as can occur with the production of concentrated urine, cause a reduction in body fluid tonicity. Adequate hydration, being in water balance, is considered euhydration (Shirreffs, 2003). In order to maintain balance, water must be provided to the body. Water is obtained primarily through food and drink. Around 80% of fluid is consumed through beverages while 20% is consumed through food (Sawka et al., 2005).

2.7.1 Definitions of euhydration, hyper hydration and dehydration

Euhydration is the state or situation of being in water balance. However, although the dictionary definition is an easy one, establishing the physiological definition is not so simple (Shirreffs, 2003). According to Armstrong (2007) the term "euhydration" is synonymous with the phrase "normal body water content". Euhydration is not a specific point, but rather is best represented by a sinusoidal wave that oscillates around an average. Body mass is commonly used to represent acute changes of body water. For example, body mass fluctuates with a group coefficient of variation of 0.66 _ 0.24% for repeated days.

The term **hyper hydration** refers to the state that exists when ingested fluid temporarily increases total body water above the average basal level prior to its removal by the kidneys. **Hydration,** therefore, involves the point at which the body presently resides, among states of euhydration, hyper hydration, and dehydration (Armstrong, 2007). Hyper hydration is therefore a state of being in positive water balance (a water excess) and hypohydration the state of being in negative water balance (a water deficit). Dehydration is the process of losing water from the body and rehydration the process of gaining body water. Fluid loss causes changes in body functions (Armstrong, 2007).

Although no consensus exists regarding a definition for the term **dehydration**, it refers to the process of uncompensated water loss via urine, sweat, faeces, and respiratory vapour; this process reduces total body water below the average basal value. Lack of consensus exists, in part, because physiologists use different techniques to evaluate dehydration (e.g. plasma osmolality, urine-specific gravity, or body weight) (Armstrong, 2007). Dehydration is signalled by thirst, flushed skin, cramps, apathy, dizziness, vomiting, nausea, chills, and dyspnea (Binkley, Beckett, Casa, Kleiner, & Plummer, 2002).

2.7.2 Markers of hydration status

Armstrong (2007) stated in his article "**Assessing Hydration Status: The Elusive Gold Standard"** that some authorities claim that a Total Body Water (TBW) value, in combination with a plasma osmolality (Posm) measurement, provide the "gold standard" for hydration assessment (i.e., provides superior accuracy, precision, and reliability). The claim regarding TBW is widely accepted; that is, the isotope dilution and neutron activation analysis techniques are considered to be the standards for measurements of TBW and body fluid spaces. This claim of a gold standard apparently refers to balance (i.e., arginine vasopressin) and is distinct from the regulation of tonicity (i.e., aldosterone). Thus, all hydration assessment techniques are best viewed as singular measures of a complex and dynamic fluid matrix, containing interconnected compartments.

Water is the medium of circulatory function, biochemical reactions, metabolism, substrate transport across cellular membranes, temperature regulation, and numerous other physiological processes. Fluid-electrolyte turnover and whole-body water balance change constantly because water is lost from the lungs, skin, and kidneys, and because water is gained in food and fluids. Therefore, accurate and precise laboratory and field techniques are needed to evaluate human hydration status (Armstrong, 2007). However, in order to monitor hydration in the field, techniques are required that involve little technical expertise and sophisticated instruments. According to Armstrong (2007) future research and development efforts should focus on novel hydration assessment techniques that:

a) measure fluid volume and concentration in real time;
b) have excellent precision, accuracy and reliability;
c) are non-invasive;
d) are interpreted in concert with other hydration indices; and
e) are portable, inexpensive, safe, and simple to use.

2.7.2.1 *Body mass changes*

Acute changes in body mass over a short time period can frequently be assumed to be due to body water loss or gain; 1ml of water has a mass of 1g and therefore changes in body mass can be used to quantify water gain or loss (Armstrong, 2007). Over a short time period, no other body component will be lost at such a rate, making this assumption possible. Throughout the exercise literature, changes in body mass over a period of exercise have been used as the main method of quantifying body water losses or gains due to sweating and drinking. Indeed, this method is frequently used as the method to which other methods are compared. However, one study showed that a loss of up to seven percent of body mass can be tolerated before adverse effects are experienced, and that the upper body muscles are affected much more than the lower body muscles (Shirreffs, 2005). Respiratory water loss and water exchange due to substrate oxidation are sometimes calculated and used to correct the sweat loss values, but this is not always done (Mitchell, Nadal and Stolwijk, 1972).

Baker, Lang, & Kenney (2009) conclude that the change in body mass during exercise is “an accurate and reliable method to assess” exercise-related changes in total body water. Nolte and Noakes (2010) have major concerns with the manner in which the data were analysed by Baker et al. (2009). However, their analysis shows that the conclusion (Baker et al., 2009) that the change in body mass accurately predicts the change in TBW during exercise is an artefact of the manner in which they analysed their data and is probably confounded by the unusual manner in which they used different biological samples (urine or serum) to measure changes in TBW in the same individuals.

2.7.2.2 *Haematocrit*

Measurement of haemoglobin concentration and haematocrit has the potential to be used as a marker of hydration status or change in hydration status, provided a reliable baseline can be established. In this regard, standardisation of posture for a time prior to blood collection is necessary to distinguish between postural changes in blood volume, and therefore in haemoglobin concentration and haematocrit, which occur (Harrison, 1985) and change due to water loss or gain. Plasma or serum sodium concentration and osmolality will increase when the water loss inducing dehydration is hypotonic with respect to plasma. An increase in these concentrations would be expected, therefore, in many cases of hypo hydration, including water loss by sweat secretion, urine production or diarrhoea. However, in subjects studied by Francesconi, Hubbard, Szlyk, Schnakenberg, Carlson, Leva, Sils, Hubbard, Pease, & Young, (1987), who lost more than 3% of their body mass mainly through sweating, no change in haematocrit or serum osmolality was found, although as described below certain urine parameters did show changes. Similar findings to this were reported by Armstrong, Maresh, Castellani, Bergeron, Kenefick, LaGasse, and Riebe (1994). This perhaps suggests that plasma volume is defended in an attempt to maintain cardiovascular stability, and so plasma variables will not be affected by hypo hydration until a certain degree of body water loss has occurred.

2.7.2.3 *Urine-specific gravity*

Measurement of urine osmolality has recently been an extensively studied parameter as a possible hydration status marker. In studies of fluid restriction, urine osmolality has increased to values greater than 900 mOsm/kg for the first urine of the day passed in individuals dehydrated by 1.9% of their body mass, as determined by body mass changes. Armstrong et al. (1994) have determined that measures of urine osmolality can be used interchangeably with urine-specific gravity, opening this as another potential marker. It seems fair to conclude that urinary measures are more sensitive than the other methods, but they may have a time lag over the short term. Euhydration (specific gravity of 1.013 – 1.029 Gcm^3). The validity of USG measures is questionable after an acute change in body mass, as would occur following exercise-induced sweat loss (Popowski et al., 2001).

2.7.3 Errors in the estimation of hydration changes in body mass

There is, however, no general agreement on the best way to assess an individual's hydration status at any given instant (Marshall, 2010). This makes for difficulties in establishing appropriate hydration strategies. Many different markers for the assessment of hydration status have been proposed and normal ranges have been suggested for some of the parameters measured. Hydration status is not easily measured, but acute changes in hydration status are often estimated from body mass change. Changes in body mass are also often used as a proxy measure for sweat losses. There are, however, several sources of error that may give rise to misleading results, and our aim in this paper is to quantify these potential errors. Respiratory water losses can be substantial during hard work in dry environments. Mass loss also results from substrate oxidation, but this generates water of oxidation which is added to the body water pool, thus dissociating changes in body mass and hydration status: fat oxidation actually results in a net gain in body mass as the mass of carbon dioxide generated is less than the mass of oxygen consumed. Water stored with muscle glycogen is presumed to be made available as endogenous carbohydrate stores are oxidised.

Fluid ingestion and sweat loss complicate the picture by altering body water distribution. Loss of hypotonic sweat results in increased osmolality of body fluids. Urine and faecal losses can be measured easily, but changes in the water content of the bladder and the gastrointestinal tract cannot. Body mass change is not always a reliable measure of changes in hydration status and substantial loss of mass may occur without an effective net negative fluid balance. The inevitable conclusion is that body mass loss is the only realistic proxy measure of hypo hydration for the athlete and the field-based practitioner (Coyle, 2004).

For the laboratory physiologist, there is no single solution, as the changes in the osmolality of body fluids may be as important as any change in volume, and this cannot be quantified without direct measurement. There is not a simple relationship between the change in body mass during prolonged exercise and the change in body hydration status. A significant loss of body mass – perhaps of the order of 1 – 3% of pre-exercise mass – may occur without resulting in hypo hydration (Maughan, Shirreffs and Leiper, 2007).

2.8 DEHYDRATION IN SPORT

According to Maughan & Shirreffs, (2004) exercise in the heat poses a formidable challenge to the body's ability to control its internal environment due to the high rates of metabolic heat production and heat gain by physical transfer from the environment. In an attempt to restrict the rise in core temperature, an increased rate of sweat secretion onto the skin is invoked. This may limit the rise in core temperature, and can prolong the time before a limiting temperature is attained, but it does so at the cost of a loss of body water and electrolytes. The effects of the diminished blood volume are offset to some extent by cardiovascular adaptations, including an increased heart rate and an increased peripheral resistance, but these are insufficient to maintain functional capacity when blood volume is reduced. Prior dehydration will impair performance in both prolonged exercise and short-term high-intensity exercise. Athletes living and training in the heat may experience chronic hypo hydration due to inadequate replacement of fluid losses. The negative consequences of exercise in the heat are attenuated to some extent by a period of adaptation, and by the ingestion of water or other appropriate fluids. Optimum fluid replacement strategies will depend on the exercise task, the environmental conditions and the individual physiological characteristics of the athlete. Manipulation of pre-exercise body temperature can also influence exercise performance and may be a strategy that can be used by athletes competing in stressful environments (Maughan & Shirrefs, 2004).

However, Noakes, (2007) stated, “Early human ancestors evolved hunting in the midday heat on the dry African savannah and developed favourable biological adaptations that permit prolonged running in the heat. These physiological adaptations must have included the capacity to sweat profusely to maintain a low body temperature when running for 4-6 hours in dry heat, an absence of adverse consequences from developing mild to moderate fluid deficits caused by sweat losses during the hunt, a serum osmolality based thirst mechanism and the ability to 'outrun their thirst' (to resist the deleterious psychological and other effects of severe thirst)”. Noakes (2007) argues that humans are designed to drink just enough to maintain plasma osmolality, not necessarily bodyweight, both at rest and during exercise. Drinking to maintain bodyweight may impair exercise performance by inducing a weight penalty and may increase the probability of exercise-associated hyponatremia in slow marathon runners. Understanding that humans evolved as hot weather runners who had limited access to fluid during exercise and that serum osmolality-regulated thirst mechanisms are protective, should encourage a critical re-evaluation of the current guidelines for fluid ingestion during marathon running. The author concluded that the time is now right for the development of novel guidelines that are solely evidence-based.

2.8.1 Fluid guidelines for sport

According to Beltrami, Hew-Butler and Noakes (2008), scientists have sought the perfect strategy to optimally replace the correct amounts of water and electrolytes lost through sweating. From these investigations, ‘‘dehydration’’ defined as ‘‘the loss of ’body water’’’ has been considered the greatest threat to exercise in the heat. It is further believed that the physiological sensation of thirst is an inadequate indicator of the hydration needs of exercising athletes, so that athletes drinking to thirst will lose weight and hence will be ‘‘dehydrated’’ and therefore supposedly be at increased risk of several dangers such as heat illness, heat cramps and heat stroke.

Athletes in training and competition are at risk of hypo hydration, as fluid intake seldom matches fluid loss (Burke and Hawley, 1997). Over hydration, or hyper hydration, is relatively uncommon, but can also cause adverse health consequences. This condition usually occurs if a person becomes overly concerned with adequate hydration. Over hydration ("Excessive Fluid") occurs when fluid intake far exceeds fluid loss. The increased body water reduces the concentration of sodium in the blood. The decrease in serum sodium level can become dangerous if below 130 mmol/L (Binkley et al., 2002), resulting in a condition called exertional hyponatremia. Where drinks with low sodium content are consumed in very large volumes, there is a risk of a dilutional hyponatremia, with potentially serious consequences (Noakes, 1992).

The inter-individual variation between players in the same team taking part in the same training session or match is so great that players must be treated as individuals with regard to their water and electrolyte needs. This approach seems to work for every other creature on planet Earth. When athletes drink according to thirst, the risk that they will over-drink and so develop exercise-associated hyponatremia is minimised and there is no evidence that they are at any significant disadvantage from the 3-5% level of “dehydration” that they develop as a result.

Understanding that humans evolved as hot weather runners who had limited access to fluid during exercise and that serum osmolality-regulated thirst mechanisms are protective, should encourage a critical re-evaluation of the current guidelines for fluid ingestion during marathon running. The time is now right for the development of novel guidelines that are solely evidence-based (Noakes, 2007). General guidelines have an important role to play in allowing sports people to exercise safely and optimally, but there are clearly situations when an individualisation of the advice given is preferable or indeed necessary.

A comprehensive hydration strategy involves ensuring good hydration before training/competition maintaining it during exercise and then replacing any shortfall as soon as possible afterwards. Water-fluid loss via urine and, especially, sweating involves the loss of electrolyte minerals – calcium, magnesium, sodium, potassium and chloride. The key found to have emerged from those studies is that there is no standard answer to the following questions:

- What is the best drink?
- How much of it should I consume?

Some top football players put away almost two litres of fluid in a 90-minute training session while others drank almost nothing. American College of Sports Medicine (ACSM, 1996), for example, has published guidelines for endurance athletes recommending that “During exercise, athletes should start drinking early and at regular intervals in an attempt to consume fluids at a rate sufficient to replace all the water lost through sweating (*i.e.,* body weight loss), or consume the maximal amount that can be tolerated". ACSM (1996) also advises: "During intense exercise lasting longer than 1 hour, it is recommended that carbohydrates be ingested at a rate of 30-60 (grams per hour) to maintain oxidation of carbohydrates and delay fatigue. This rate of carbohydrate intake can be achieved without compromising fluid delivery by drinking 600-1,200 (ml per hour) of solutions containing 4-8% carbohydrates (*i.e.* 40 – 80g per 100ml).

The International Marathon Medical Directors Association (IMMDA) has recommended a fluid intake of something between 400 and 800ml per hour, with the higher rates being appropriate for faster or heavier runners and the lower rates for slower runners and walkers. The problem with both sets of recommendations is that they are too inflexible. The ACSM (1996) guidelines can be interpreted as encouraging runners to drink as much as they can: this may be more than is necessary and can lead to problems of water overload and even hyponatremia. As a rule of thumb, during an endurance event one should aim to drink just enough to be sure one loses no more than 1-3% of one's prerace weight. Fluid is vital for adequate recovery – not just to replace water lost in sweat but also to help replenish glycogen.

The American Academy of Pediatrics Committee on Sports Medicine and Fitness (2000) and Barrow and Clark (1998) stated that dehydrated athletes are more likely to suffer heat illness. Mild dehydration (<2% body weight loss) occurs commonly in athletics and may be unavoidable. Dehydration levels can be approximated by weighing athletes before and after practices and competition. An athlete should be able to compete with a weight loss less than 3% of pre-exertion body weight. For weight loss greater than 3%, athletes should be restricted until body weight recovers with hydration. In athletes with weight loss greater than 3% dehydration, muscular strength and endurance decreases, plasma and blood volume decreases, cardiac output is compromised, thermoregulation is impaired, kidney blood flow and filtration decreases, liver glycogen stores decrease, and electrolytes are lost (NCAA 2006-2007 Sports Medicine Handbook). In contrast, body weight gains greater than 3% may predispose athletes to exertional hyponatremia from excessive water intake.

However, Beltrami et al. (2008) stated that accumulating evidence suggests that the human body does not regulate body weight during exercise, but instead activates mechanisms to control the rise in plasma osmolality caused by hypotonic sweat losses. According to Beltrami et al. (2008) the physiological sensation of thirst is stimulated by an increase in plasma osmolality or when plasma volume decreases by 10%. Thus, whilst it has always been believed that the thirst mechanism is physiologically ‘‘sufficient’’ at rest, current interpretation implies that thirst becomes an inadequate guide for fluid replacement during prolonged physical activity.

Prevention of dehydration is a reasonable goal for all who monitor athletes as well as the athletes themselves. Unfortunately, many athletes do not realise they are becoming dehydrated. Perhaps this is due to a lack of education or because of their intense concentration on the sport they are playing. In either case, a coach, athletic trainer, or parent may need to intervene to ensure that adequate hydration occurs. Ideally hydration starts before a practice session or game competition. Different exercise science textbooks offer specific guidelines as to the amount of fluid needed (**see Table 2.7**).

Table 2.7: Drinking guidelines in various exercise science textbooks

Author Edition Guidelines		
Shephard	1974	No guideline presented
DeVries	1980 ,90	180ml every 10–15 minutes
Brooks & Fahey	1984, 1987	Forced pattern (more than thirst)
Wilmore & Costill	1988	200–300ml every 15–20 minutes
Sharkey	1990	No specific guideline
Bloomfield, Fricker & Fitch	1992	More than thirst
Pollock & Wilmore	1993	Abundant amounts before, during and after
Harries, Williams *et al*.,	1994	Start drinking long before feeling thirsty
Brooks, Fahey & White	1996	According to weight loss
Wilmore & Costill	1994, 1999	100–150ml every 10–15 minutes
Weineck	1999	According to weight changes
Robergs & Roberts	2000	7–15ml per kg per hour
International Sports Medicine Directory	2001	Equal to weight loss or as much as tolerable
Brooks, Fahey & Baldwin	2005	Drink to match (or almost) sweat rate
Powers & Howley	2007	150–300ml every 10–15 minutes and 150% of weight loss after exercise

(Beltrami et al**., 2008).**

The ACSM now recognises that ‘‘because there is a considerable variability in sweating rates and sweat electrolyte content between individuals, customized fluid programs are not recommended’’. The ACSM also concluded that ‘‘the goal of drinking during exercise is to prevent excessive (2% body weight loss from water deficit) dehydration'' (Beltrami et al., 2008).

Teaching athletes to monitor their urine colour and output may be prudent to assist in the process of hydration. The goal for the athlete is copious output of clear to light yellow urine.

2.8.2 Children versus adults fluid needs during exercise

According to Bergeron et al. (2005) as with adult athletes, maintaining fluid balance can be difficult for young football players, especially in hot and humid conditions. Intensity and duration of practice, scheduling of fluid breaks, uniform configurations, and number of sessions per day are also key factors in tempering or exacerbating this challenge. Unfortunately, specific data and insight regarding fluid loss and intake patterns in young football players during practice or games are very limited.

Stover et al. in Begeron et al. (2005) observed moderate rates of sweating (1l per hour) and small body weight deficits (about 1%) in high school players during preseason practice. These measures were slightly lower than losses described in collegiate players training in similar moderate (wet bulb globe temperature [WBGT] 25°C) environmental conditions (Stofan et al., 2003). In another recent on-field examination of high school players during two successive days of preseason football training in much hotter and more humid conditions (33°C, 56% relative humidity), Bergeron et al. (unpublished findings) noted similar pre- to post-practice body weight deficits of nearly 1%, despite each player consuming about 2 L of water during the daily 2-h practice sessions. Moreover, greater sweat fluid losses led to greater body weight deficits. This is not surprising, as athletes often do not match sweat loss with fluid intake during exercise in the heat (Bergeron et al., 2005).

Bergeron et al. (2005) also noted that the 10 players presented with elevated urine specific gravities on day 1, suggesting that they were not well-hydrated at the start of practice. Notably, the same players had even higher urine specific gravities at the start of practice on day 2, suggesting that their recovery fluid intake to restore sweat fluid losses from the previous day was insufficient and that they were more dehydrated than on day 1.

Stover et al., as cited in Bergeron et al. (2005) also examined day-to-day changes in body weight and pre-practice hydration status across 5 days of the two-a-day training sessions. The players' body weights remained steady, after an initial decrease (0.5kg) after the first day, and urine specific gravities from pre-practice samples remained high (yet unchanged), suggesting that these players were not well-hydrated as well before the start of each practice. The above observations suggest that young football players tend to *begin* practice measurably dehydrated and this continues on successive days of practice, especially in the heat, even when the athletes have ample time and opportunity to rehydrate overnight. Large sweat losses, insufficient fluid intake, and consequent fluid deficits could likely impair performance and may increase the risk of hyperthermia and heat injury (Stofan et al., 2003).

The proportion of water in the body decreases with age. According to (Friis-Hansen, 1961), approximately 80% of the body mass at birth comprises of water. This drops to about 61% and 63% in the first and third year of life, respectively. In male adults, body water ranges from 50 – 70% of body mass and is lower in females.

A unique aspect of fluid replacement in young children, compared to adults is that they need more fluid daily as a percentage of body mass. Respectively, daily water turnover at one and six years is 13% and 11% of the total body mass, while in adults it is 6% (Hickman and Yasuda, 1986). Rivera-Brown et al. (1999) also stated that several studies have demonstrated that children are less-effective thermo regulators than are adults in extreme environmental conditions. Children sweat less than adults do when exercising in a hot environment, which limits their reliance on evaporative heat loss. Additionally, children, similar to adults, do not drink enough to replace sweat losses and exhibit hypo hydration when water is provided ad libitum during exercise in the heat. Thus, children who participate in sports programmes in tropical regions may be in a state of chronic hypo hydration if they train and compete frequently without replenishing their fluid losses completely.

However, Rowland (2008) in his article "**Thermoregulation during exercise in the heat in children: old concepts revisited"** disagreed and challenged these traditional old concepts. Rowland (2008) stated "*Children possess certain physiological and anatomic characteristics that have traditionally been considered to impair thermoregulatory responses to exercise in the heat: low exercise economy, high ratio of body surface area to mass, diminished sweating capacity, and less* *cardiac output at the same work load compared with adults. Consequently, children have been regarded as an at-risk group for not only decrements of physical performance but also heat injury during physical activities performed in conditions of high ambient temperature. Recent investigations that have directly compared thermoregulatory responses to exercise in the heat in children and adults have challenged these traditional concepts. Such studies have failed to indicate group differences in heat dispersal when adult-child comparisons are appropriately considered in respect to relative exercise intensity. These findings imply that no maturational differences exist in thermal balance or endurance performance during exercise in the heat, nor that child athletes are more vulnerable to heat injury*".

2.8.3 Electrolyte disturbances

Besides containing water, sweat contains electrolytes that are lost. If not appropriately replaced, water and electrolytes imbalances (dehydration and hyponatremia) can develop and adversely impact on the individuals.

The main routes of electrolyte loss from the human body are the urine, faeces, and sweat, although vomiting also results in significant losses. Large amounts of electrolytes are also temporarily secreted from the body into the gastrointestinal tract, but these are largely reabsorbed later in the system. It is sweat electrolyte losses that have the potential to set exercisers apart from non-exercisers and will therefore be the focus for consideration here. A wide range of values for all of the major sweat electrolytes has been reported in the literature, reflecting variations between individuals, differences due to the experimental conditions, and differences due to the collection methods.

The main electrolyte lost in sweat is sodium, with chloride being present in slightly smaller amounts. The other electrolytes present (e.g. potassium, calcium, magnesium) are at vastly lower concentrations (Maughan & Shirreffs, 2004; Lentner, 1997). During matches and training sessions, some players will lose considerable quantities of electrolytes – particularly sodium – and may need to replace these during the match or training session. If sodium-containing beverages do not suffice, athletes may want to consume small amounts of salted snacks between periods to replace salt losses and stimulate drinking.

Prolonged heavy sweating can lead to significant mineral losses (particularly of sodium). Drinking pure water dilutes the concentration of electrolyte minerals in the blood, which can impair a number of normal physiological processes. An extreme example of such an impairment is “hyponatremia”, when low plasma sodium levels can be literally life threatening.

2.8.4 Water versus sport drinks

Nicholas et al. (2005) stated that water and sports drinks provide benefits for the physically active person. While sedentary individuals need to consume water, a moderate to extremely active person may receive necessary electrolyte replacements with sports drinks. Currently, water and carbonated beverages represent most of the beverage consumption during active thirst occasions. In 2005, 64% of active people consumed bottled water during activity (Shirreffs, Aragon-Vargas, Keil, Love, & Phillips, 2007).

Sports drinks may have a slight advantage over water in fluid balance and palatability. The addition of either potassium or sodium to a rehydration beverage can be effective (Shirreffs et al., 2007). A sports drink can affect the rate of water assimilation and replace depleted muscle reserves. To enhance water absorption, glucose is recommended at concentrations of 3% to 5%. Any concentration above five percent is not recommended. Enhanced water absorption and increased glucose oxidation can aid in performance. The amount of fluid retained is related to the sodium content of the drink (Shirreffs et al., 2007). The sodium content in sports drinks is around 10 to 30 mmol/L.

Rosenbloom, Jonnalagadda, and Skinner (2002) determined that only three percent of college football players agreed with drinking sports drinks during an event. Consequently, only 22% of the subjects in that study perceived sports drinks as a better choice than water. Numerous studies have been conducted on the effectiveness of sports drinks and water consumption. Shirreffs et al. (2007) discovered that subjects who hydrated intermittently with Gatorade were at fluid balance after exercise. However, subjects who consumed Evian water and other commercially available products had a lowered hydration status, with the difference in fluid balance between the Gatorade and Evian of 328 millilitres or 0.5% body mass. In contrast, Byrne, Lim, Chew, and Ming (2005) concluded that sports drinks consisting of carbohydrate and electrolytes provide no added benefits. In the same study, the sports drink did elevate blood glucose levels. This suggests that glucose was available for oxidation. When rehydration is essential, sports drink selection may be advised (Shirreffs et al., 2006).

2.8.5 Sweat rate and fluid turnover in hot and humid / dry environment

According to Godek, Godek and Barttolozzi (2005) individual sweat rates vary and depend on factors such as ambient temperature, humidity, air movement, exercise intensity, insulating clothing or equipment, and body size. Sweat rates and fluid turnover have been reported in runners, cyclists, and athletes participating in team sports such as basketball, netball, soccer, and Australian Rules football. Godek et al. (2005) reported sweat rates during practice sessions and competitions were 0.985 and 1.209 l/h for soccer, and 1.371 and 1.601 l/h for basketball players. The average sweat rate of 1.71 l/h in male runners found during a simulated 40 km run compares with the sweat rate range of 1.0–2.5 l/h common in runners exercising intensely in hot and humid environments. At least these studies assessed fluid turnover during a single bout of exercise, but the vast majority of information related to heat stress and hydration in athletes involves aerobically trained subjects where rugby is primarily an anaerobic sport.

Important factors that affect sweat production during exercise are physical conditioning (aerobic and anaerobic), acclimatisation, hydration status, exercise intensity, physical size, and amount of clothing or equipment worn (Godek et al., 2005).

Athletes in training and competition are at risk of hypo hydration, as fluid intake seldom matches fluid loss. Many published reports have described sweat losses and drinking behaviours of various groups of athletes in training and in competition. For several sports, there are data for athletes of different competition levels, and measurements have been made under varying environmental conditions. Broad, Burke and Cox (1996) have reported sweat rates of soccer players training in summer (985 ± 320 ml/hr: mean ± SD) and winter (746 ± 249 ml/hr), with intakes being much lower for each (429 ± 312 ml/hr in summer and 311 ± 257 ml/hr in winter).

This combination of loss and intake would result in a negative fluid balance of about 500 ml/hr. It is generally accepted that the loss of exercise capacity is apparent when the fluid deficit exceeds about 1% of body mass and that the performance decrement is proportional to the fluid deficit.

Although the normal response to exercise is to incur a fluid deficit, some individuals, in situations where sweat losses may not be high and where there are ample opportunities for drinking, may consume volumes of fluid far in excess of losses. Where drinks with low sodium content are consumed in very large volumes, there is a risk of a dilatational hyponatremia, with potentially serious consequences. In a sport such as rugby, this situation may be more likely to arise in training, where frequent breaks are allowed and drinks are readily obtainable, than in competition, where opportunities for drinking are limited.

When sweat rates are high, significant losses of electrolytes, especially sodium will occur, but both sweating rate and sweat electrolyte content vary greatly between individuals. Exercise-induced muscle cramps are often thought to be linked to electrolyte disturbances, especially losses of sodium, but evidence is not strong. Fluid replacement during both training and competition is recommended to provide carbohydrate, water, and electrolytes, but the requirements of individuals for these three different components can vary greatly. Although the composition varies from person to person (partly as a function of acclimatisation) a litre of sweat typically contains the following;

- Calcium – 0.02g
- Magnesium – 0.05g
- Sodium – 1.15g
- Potassium – 0.23 g
- Chloride – 1.48g

Football players, while they trained in the preseason, exhibited mean Fluid Turnover (FTO) rates of 10 L/d. It was estimated that about 70% of the FTO could be accounted for by sweat loss during training and that this high rate of turnover might have made it difficult for the players to maintain hydration during training (Godek et al., 2005).

Mashiko, Umeda, Nakaji, and Sugawara (2004) suggest that, in rugby players attending a 20-day camp, exercise training resulted in muscular damage, loss of electrolytes due to sweating, and changes in immune function. Backs exhibited a higher rate of fat metabolism and loss of electrolytes than forwards, possibly because they did more running during the camp. In contrast, forwards experienced more physical contact, performed more physically strenuous exercise, and exhibited higher levels of muscular damage and tissue protein degradation.

2.8.6 Dehydration and physical performance

According to Kraft, Green, Bishop, Richardson, Neggers & Leeper (2012) the hydration level is a critical factor influencing exercise performance, especially in the heat. Dehydration has profound negative effects on endurance performance, (Cheuvront et al., 2003, Sawka et al., 2007). These decreases in aerobic performance may be attributed to decrease in plasma volume and stroke volume and subsequent reductions in cardiac output as well as changes in muscle blood flow (Gonzalez-Alonso, Crandall, & Johnson, 2008).

Conversely, studies examining the impact of dehydration on anaerobic performance (short duration bouts primarily dependent on oxygen independent metabolic pathways) are limited. Judelson, Maresh, Anderson, Armstrong, Casa, Kraemer & Volek, (2007) stated that this has made it difficult to generalise the impact of dehydration on anaerobic performance, which may be influenced, at least in part, by different mechanisms than aerobic performance. According to Kraft et al. (2012) one of the problems in describing the effects of dehydration relative to anaerobic performance is exercise mode variations. All the different exercise modes are dominated by anaerobic pathways, but inherent differences exist (*i.e.*, duration, active muscle volume, specific joint actions and skill requirements). Therefore, comparisons between existing studies are difficult because a variety of modes of physical testing have been used (e.g. vertical jump, 10s maximal cycle sprints, Wingate anaerobic test, Margaria power test, Isokinetic force production). Furthermore, comparisons among studies are complicated by the variant levels of dehydration tested (ranging from 1%-5.7%) and other confounders (inclusion of heat exposure, influence of exercise) potentially associated with differing modes used to elicit dehydration.

Yoshida, Takanishi, Nakai, Yorimoto, & Morimoto, (2002) suggested that a critical level of water deficit for anaerobic performance (~ 3-4%; mode dependent) does exist, but this deficit appears to be greater for anaerobic performance than aerobic performance (~2%).

However, exercise performance is impaired when dehydration exceeds about 2% of the pre-exercise body mass, especially in warm environments. These statements are based on the assumption that 1kg of mass loss is equal to 1 litre of sweat loss. Clearly, any fluid intake and urine or faecal loss needs to be accounted for and appropriate corrections made. There are limited data on the effects of a body water deficit on performance in elite players. From the limited information available, it would appear that there is little likelihood that any negative effects of dehydration on soccer performance would be different from those in other intermittent-activity sports or endurance sports, and indeed there is no reason why this should be so. That is, it is unlikely that any negative effects would be seen until hypo hydration reaches a level equivalent to a 2% loss of body mass and perhaps even greater losses could be tolerated in cool environments.

The only direct child-adult comparison of hydration status during exercise without fluid replacement is that of Drinkwater, Kuppart, Denton, Crist and Horvath (1977). The low SR of children during exercise in the heat might be expected to beneficially limit body fluid losses. However, there exists no evidence that their levels of dehydration during such exercise are any different from that of adults (Rowland, 2008). Drinkwater et al. (1977) found that percent weight loss, rate of weight loss, and change in plasma volume during walking in 28°C, 35°C, and 48°C conditions were similar in premenarcheal girls and young adult women.

The degree of dehydration during exercise is dictated by fluid intake as well as SR. No experimental data are available regarding maturational differences in thirst drive relative to dehydration thresholds. Limited information suggests, however, that voluntary drinking and dehydration during exercise in the heat is similar in children and adults. The eight boys and eight men studied by Rowland et al. (2008) consumed an average of 5.1 and 5.3 ml/kg, respectively, when drinking cool water ad libitum during cycling in 31°C and 50% relative humidity for 30 min. Rowland et al. (2008) also noted that comparisons with studies in adults were difficult because of different climatic conditions, exercise protocols, and type of ingested fluid. The low SR of children during exercise in the heat might be expected to beneficially limit body fluid losses. However, there exists no evidence that their levels of dehydration during such exercise are any different from that of adults.

2.9 HEAT ACCLIMATISATION

2.9.1 Heat acclimatisation

American Academy of Pediatrics (2011) recognises that appropriate and sufficient regular physical activity plays a significant part in enhancing and maintaining health. However, they recommended that special consideration, preparation, modifications, and monitoring are essential when children and adolescents are engaging in sports or other vigorous physical activities in warm to hot weather. Exertional heat illness, including heat exhaustion and heat stroke, might occur even in a temperate environment, but the risk is highest when children and adolescents are vigorously active outdoors in hot and humid conditions. Severe exertional heat injury or heat stroke is associated with significant morbidity and mortality, especially if diagnosis is delayed and appropriate medical management is not initiated promptly (American Academy of Pediatrics, 2011).

Sportspeople are able to cope much better with hot or humid conditions if they are acclimatised (Sparling, 1997). According to Brukner and Khan (2012) the human body adjusts to exposure in hot conditions by increasing blood volume and venous tone and, particularly, by alterations of the sweating mechanism. The main ways according to these authors in which the sweating mechanisms are affected are by:

- earlier onset of sweating
- increased amount of sweating
- increased dilution of the sweat (lower sodium concentration)

These sets of changes result in increased heat loss for a given set of environmental conditions and a smaller rise in body temperature.

It has been well established that regular exposure to hot environments results in a number of physiological adaptations that reduce the negative effects associated with exercise in the heat. These adaptations include a decreased core temperature at rest, decreased heart rate during exercise, increased sweat rate and sweat sensitivity, decreased sodium losses in sweat and urine, and an expanded plasma volume (Wendt et al., 2007).

Two primary determining factors for adaptation to hot conditions are:

1) the rise in body temperature that occurs and
2) the sweating response that is induced.

Adaptation, therefore, depends largely on the intensity and duration of exercise and on the environmental conditions, and there is an optimum set of conditions for the most effective acclimatisation. Adaptation is more or less complete for most individuals within about 7-14 days. There are two ways of acclimatising for competitions in the heat. One is to live and train in a climate similar to that expected at the competition venue, referred to as acclimatisation. The other approach is to live at home and adapt by training in an artificial climate: referred to as acclimation. The ideal may be an increasing level of heat exposure at home during the last 1-2 weeks before travelling. The ideal temperature and humidity for acclimatisation are not well established, but a temperature of at least 30°C, but not more than 35°C is preferable.

2.9.2 Practical recommendations for heat acclimatisation

Knowledge of the signs and symptoms of heat illness is important for athletes, parents, and coaches as well as medical staff. Early recognition of a problem and simple treatments initiated at the onset of symptoms may be lifesaving measures.

According to Bergeron et al. (2005) from 1995 to 2001, 21 young football players reportedly died from heat stroke in the United States. Since that time, the media has highlighted a number of similar incidents, as well as other heat-related problems with young players on the football field, such as exertional collapse. Despite the recognised benefits of sufficient fluid intake and precautionary measures to optimize performance and reduce the risk of heat illness, heat- and dehydration-related problems persist on the football field—particularly in preseason practice.

Bergeron et al. (2005) also stated that when planning the preseason practices and schedules, coaches and league organisers should consider that many athletes in these age groups will report with minimal, if any, conditioning and without sufficient acclimatisation to the heat stress challenges of on-field football practice. Therefore, to minimise heat strain and allow a safe transition to full-intensity practice in full gear, gradual and increasing exposure to practice intensity and duration, and gradual introduction of the different uniform configurations that considers the insulating properties of the equipment are critical. Most of the early-season football heat stroke deaths have occurred in the first 4 days of practice (with days 1 and 2 having the highest risk). These acclimatisation recommendations (s**ee Table 2.8)** are intended to reduce the incidence of exertional heat stroke, as well as the incidence of general injury, during the highest-risk days of the preseason.

Table 2.8. Acclimatisation guidelines for football

NCAA Guidelines:[a]	**ACSM Guidelines:**[b]
5-day acclimatisation period at the beginning of the season - restricted to no more than 1 practice session a day lasting <3 hours Helmet wear only for days 1, 2 Helmet plus shoulder pads only days 3, 4 Full equipment on day 5 After day 5, multi-practice days are allowed with specific guidelines —the total practice time per day must be <5 hours —a single practice may not last longer than 3 hours —at least 3 hours of rest between practices must occur —the multi-practice days must not occur on consecutive days	6-day acclimatization period at the beginning of the season—no more than 1 practice lasting <3 hours during this time —Days 1, 2: helmet only —Days 3-5: helmet and shoulder pads only —Day 6-7: full equipment —No contact drills during acclimatization period —Limit consecutive practice days to 6 —Day 8: multiple practice sessions with same restrictions as above
a NCAA: National Collegiate Athletic Association; ACSM: American College of Sports Medicine.	
b Differ slightly from NCAA Guidelines, for high school athletes.	

(Bergeron et al**., 2005)**

Wendt et al. (2007) stated that the process of acclimatisation to exercise begins within a few days, and full adaptation takes 7–14 days for most individuals. It is clear from **Table 2.9** that the systems of the human body adapt at varying rates to successive days of heat exposure. The early adaptations during heat acclimatisation primarily include an improved control of cardiovascular function through an expansion of Plasma Volume (PV) and a reduction in heart rate. An increase in sweat rate cutaneous vasodilatation is seen during the later stages of heat acclimatisation.

Table 2.9 Range of days required for different adaptations to occur during heat acclimatisation

Adaptation	Days of heat acclimatization
Decrease in heart rate during exercise	3–6
Plasma volume expansion	3–6
Decrease in sweat Na+ and Cl–concentrations	5–10
Increase in sweat rate and sweat sensitivity	7–14
Increase in cutaneous vasodilatation	7–14

(Wendt et al., 2007).

The American College of Sports Medicine (ACSM) has modified these guidelines slightly for adolescent athletes given the fact that adolescents are more susceptible to heat illness. Adolescents tend to begin their practice sessions under-hydrated, ingest insufficient fluids during exertion, and take longer to acclimatise to hot conditions. In addition, they have a greater surface body area-to-body-mass ratio than adults. This leads to greater heat gain from the environment on a hot day. Children and adolescents have a lower sweating capacity than adults and produce more metabolic heat per unit of mass during physical activities (walking or running). The acclimatisation modifications for adolescents are described in Table 2.9. It should be noted that these guidelines have not yet been validated to be effective at reducing the number of athletes with heat illness.

However, the idea that children are more intolerant to exercise in the heat compared with adults stems principally from the study of Drinkwater et al. (1977).

According to Rowland (2007), morphologically, children have a higher body surface area-to-mass ratio -- a major factor in "dry" heat dissipation and effective sweat evaporation. Locomotion-wise, children are less economical than adults, producing more heat per unit body mass. Additionally, children need to divert a greater proportion of their cardiac output to the skin under heat stress. Thus, a larger proportion of their cardiac output is shunted away from the body's core and working muscles -- particularly in hot conditions. Finally, under all environmental conditions and allometric comparisons, children's sweating rates are lower than those of adults. The differences appear to suggest thermoregulatory inferiority, but no epidemiological data show higher heat-injury rates in children, even during heat waves. Rowland (2007) suggests that children employ a different thermoregulatory strategy. In extreme temperatures, they may indeed be more vulnerable, but under most ambient conditions they are not necessarily inferior to adults. Children rely more on dry heat dissipation by their larger relative skin surface area than on evaporative heat loss. This also enables them to evaporate sweat more efficiently with the added bonus of conserving water better than adults.

2.10 IMPORTANT HEAT STRESS INFORMATION

Results of new research (Council on Sports Medicine and Fitness and Council on School Health, American Academy of Pediatrics, 2011) indicate that, contrary to previous thinking, youth do not have less effective thermoregulatory ability, insufficient cardiovascular capacity, or lower physical exertion tolerance compared with adults during exercise in the heat when adequate hydration is maintained. Accordingly, besides poor hydration status, the primary determinants of reduced performance and exertional heat-illness risk in youth during sports and other physical activities in a hot environment include undue physical exertion, insufficient recovery between repeated exercise bouts or closely scheduled same-day training sessions or rounds of sports competition, and inappropriately wearing clothing, uniforms, and protective equipment that play a role in excessive heat retention. Because these known contributing risk factors are modifiable, exertional heat illness is usually preventable.

2.10.1 Strategies for exercise in the heat

Bergeron et al. (2009) also stated that during outdoor youth sports tournaments in the heat, young athletes typically do not have the opportunity to promptly get out of the heat and completely rehydrate and cool down between games or matches. “Accordingly, our findings support previous laboratory and field studies on adults to the extent that there is a potential for the residual or carryover effects from a recent prior bout of strenuous exercise and heat exposure to decrease performance and increase clinical risk during a subsequent same-day exercise bout in the heat, even when hydration is maintained and core body temperature returns to baseline between bouts. Therefore, on the basis of our findings and those from other related studies cited in this report, youth sports governing bodies, tournament directors, and other event administrators should recognise and appreciate that providing longer rest and recovery periods between contests during hot weather events may improve athlete performance and safety. Moreover, organisers of tournaments and multiple same-day practice sessions for youth in the heat also should consider the potential effects of exercise and heat exposure from the previous day, even if the current day appears “safe,” to minimise exertional heat injury risk” (Bergeron et al., 2009).

2.11 CONCLUSION

Perhaps the most tragic fact surrounding heat stroke deaths in players is that the condition is entirely preventable. At the same time, the preventable nature of heat stroke offers the opportunity to prepare for these events and decrease the incidence. Heat illness occurs along a spectrum that begins with relatively mild disease and can progress to life-threatening heat stroke. Recognition of heat illness and initiation of early treatment may prevent progression to heat stroke. Heat stroke in players occurs as a result of intrinsic body heat production and impaired heat dissipation. It is commonly seen in hot and humid weather but has occurred in the setting of mild weather conditions. Diagnosis of heat stroke includes elevated core body temperature (>104°F or >40°C) and CNS dysfunction. Any player with CNS dysfunction during or after exertion should be evaluated for heat stroke even in the setting of core body temperature <104°F as cooling may have already begun. Paramount to the treatment of heat stroke is rapid temperature cooling and access to higher levels of care via the emergency medical system.

Proper education of rugby coaches and players, identification of high-risk players, concentration on preventative hydration and acclimatisation techniques, and appropriate monitoring of players for heat-related events are important ways to prevent heat stroke. There is a need for survey data on the knowledge, attitudes and reported behaviours of children, parents and coaches towards heat injury. Ideally, these surveys should be nationally representative and ongoing (Bergeron et al., 2005; Rowland, 2007; Wendt et al., 2007; American Academy of Pediatrics, 2011).

CHAPTER 3
METHOD OF RESEARCH

3.1 INTRODUCTION

This chapter describes the protocol that was designed to investigate the objectives stated in chapter one. A description of the instruments that were used as well as the technique for every measurement will be discussed. In preparation for this thesis, literature was collected from electronic databases such as Kovsiekat, Pubmed, EbscoHost (Academic Search Elite and Medline), academic journals, and textbooks.

3.2 STUDY DESIGN

This study was a cohort-analytical study on certain variables associated with hydration levels of u/19 rugby players from Hopetown High School during two matches in 2007 and two matches in 2009. The group of rugby players was subjected to a pre-evaluation (15min before the game) followed by a re-evaluation performed 10min after the game. In this way the dehydration status of the players could be determined.

3.3 STUDY PARTICIPANTS

Before the study commenced and before the subjects were recruited, the study was approved by the Ethics Committee of the University of the Free State. A meeting with the Principle and Rugby Coach was arranged in order to get full permission for execution of the investigation as well as to explain the procedures that followed **(See Appendix C)**.

All u/19 rugby players (age 16 - 19 years) from Hopetown High School participating in two matches during March 2007 and two during March 2009 were approached to participate in the study. In total 33 healthy male high school football players volunteered to participate in this study. Fifteen players were tested who participated in the two matches in 2007 and 18 players were tested who played in two matches in 2009. The sample size goal was to include a maximum of u/19 school rugby players. Positions were not represented in large enough numbers to explore possible differences in variables among positions. The relatively small sample size is due to the fact that the Hopetown High School has only one u/19 rugby team.

Furthermore, the inclusion criteria for testing were that the temperature at the time of the match had to exceed 30˚C; therefore only four matches in this period met the inclusion criteria for the study.

3.4 MEASUREMENT

The rugby games began at 15:00 and lasted 120 minutes including 10-15 minutes of a warm-up and stretching before the actual start of the game. The players were all dressed in shorts, rugby boots, socks, and a rugby jersey. No cool down was performed after the match.

Readings were taken of *Urine-Specific Gravity (SG)*, blood *haematocrit*, length and mass of every rugby player before and after every rugby match. Research assistants (two qualified nurses) were used to assist the researcher in gathering the data. The research assistants were trained by the Researcher to assist with the data collection. All measurements were recorded on the measurement form **(Appendix B)**.

Fifteen to eighteen players were evaluated during each match. The data were captured on an Excel spread sheet. No recommendations in terms of fluid and/or food intake before or after the match were made to players. Length was measured with a *stadiometer*. The heels of the player were placed against the base of the *stadiometer*, feet together, upright with the hands next to the sides while looking straight ahead.

Body mass was measured with a calibrated *Secca-Alpha electronic* scale 15-20 minutes before the match and 10 minutes after the match. Body mass was rounded off to the nearest tenth of a kilogram.

Length and mass were used to determine the body mass index (mass divided by length squared), which indicated the size of the individual. Larger individuals should lose more fluid than smaller individuals under similar circumstances (temperature, humidity, conduction, convection, radiation and evaporation) (Godek et al., 2006). One gram change in body mass represents one millilitre change in body fluid (Armstrong, 2007). In order to evaluate the sweat rate (SR), the players were weighed 15-20 minutes before the beginning of the match (BW) and 10 minutes after the match (AW).

Urine-SG was used as measurement for hydration status (Armstrong et al., 1994). Fluid loss/increase was determined by the testing/reading of *urine-SG* by means of Multistix™ urine strips.

Change in plasma volume is another means by which to determine the hydration status (Harrison, 1985). For this the *haematocrit (HCT)* (ratio of volume solid blood parts i.e. red blood corpuscles, white blood corpuscles and blood platelets to plasma volume) was used. Higher HCT means dehydration due to water loss. Players were requested to empty their bladders two hours prior to the collection of the first samples. Urine was collected 10 - 20 minutes before the start of each match, as well as 10 minutes after the end of the match. *Urine-SG* was done on these samples by means of multistix-10 SG urine strips from Bayer as well as with a *refractometer*. *Haematocrit* was determined on blood samples which were collected from each participant 15 - 20 minutes before and after each match.

An environmental temperature of a minimum of 30 degrees centigrade had to be present to qualify for measuring. Humidity was not taken into account.

3.5 METHODOLOGICAL AND MEASUREMENT ERRORS

Errors were minimised by training the assistants and utilising the same assistant for taking each of the readings. In order to minimise errors each participant was weighed with the same calibrated scale. Blood was drawn from the *antecubital vein* of every participant by means of a no.21G needle and *vacutainer blood vials.* A new needle was used for every blood sample. SG of each participant was determined from urine which was collected individually in clean glass beakers with a screw-top lid. Immediately after each match all blood samples were sent simultaneously to the Path Care Laboratory in Kimberley.

3.6 PILOT STUDY

A pilot study with five players was conducted nine months prior to the intervention regimen. It consisted of testing the hydration levels of the player before and after the game to determine the effectiveness of the proposed data sheets, equipment, and protocols. Additionally, duration of testing per player as well as the continuity of testing was evaluated in order to properly plan testing schedules for the study. Data sheets, equipment, and protocols were found to be effective in testing the proposed objectives.

3.7 ANALYSIS OF THE DATA

Data was captured electronically by the researcher in Microsoft Excel (Microsoft Office 2007). Any further analysis was done by a biostatistician using SAS Version 9.1.3. Descriptive statistics namely means, standard deviations, minimum and maximum values were calculated for all variables. The student t-test was used to test for significant differences within the group. A significance level of $p < 0.05$ was used throughout the study.

3.8 ETHICS

Before the study commenced and the subjects were recruited, the study was approved by the Ethics Committee of the University of the Free State (ECUFS 201/6). Informed consent forms approved by the Ethics Committee of the University of the Free State were handed out and had to be signed by the individuals' legal guardians, because the partaking individuals were minors **(See Appendix D and E)**. The form contained all the necessary information and basic elements as specified by the *Research Quarterly for Exercise and Sport*, by Thomas in 1983 as advised to use by Thomas, Nelson & Silverman (2011).

3.9 LIMITATIONS OF THE STUDY

This study has three limitations. The first limitation is that water ingestion was not measured before and during the games. The second limitation is that we did not perform a dietary survey before the games. Furthermore, we did not measure the intensity of the game, which may also influence the hydration levels of the players.

CHAPTER 4
RESULTS

4.1 SAMPLE CHARACTERISTICS

Thirty-three subjects completed the study. The demographic information is presented in Table 4.1.

Table 4.1 Demographic and anthropometric information

	Match 1 & 2 (n = 15)				Match 3 & 4 (n = 18)			
	Age (years)	Length (cm)	Mass (kg)		Age (years)	Length (cm)	Mass (kg)	
			M_1	M_2			M_3	M_4
1	16	170	73.9	74.5	17	181	66.1	65.5
2	16	171	60.7	60.9	18	183	61.2	61.5
3	17	174	68.4	70.1	17	190	75.8	74.9
4	16	176	72.6	72.0	16	175	103.8	104.4
5	17	181	69.3	69.0	16	174	73.1	73.3
6	18	171	85.9	86.6	17	186	86.0	85.7
7	18	184	70.0	72.3	15	168	53.4	54.1
8	17	182	66.3	66.4	16	165	51.1	50.5
9	17	182	60.7	61.2	18	184	70.3	69.4
10	17	191	75.3	75.3	18	171	85.7	86.5
11	16	178	105.5	105.7	17	181	69.1	68.7
12	16	176	73.4	75.0	16	176	72.6	72.4
13	16	186	86.0	87.4	17	174	69.1	68.7
14	15	169	53.6	53.2	16	170	61.0	60.5
15	16	165	51.0	53.6	16	170	73.8	74.1
16					18	181	67.8	66.9
17					18	165	61.0	60.5
18					17	186	85.3	85
$\overline{x}$ ± sd	17±1	177±7	71.5±13.7	72.2±13.6	17±1	177±8	71.5±12.8	71.3±13.0

***M_1 = Match1, M_2 = Match 2, M_3 = Match 3 and M_4 = Match 4. M_1 and M_2 were played in the 2007 season. M_3 and M_4 were played in the 2009 season**.

From **Table 4.1** it can be seen that the participants in 2007 and 2009 had a mean (± SD) age of 17 ± 1 years, ranging from 16 to 18 years. The anthropometric characteristics for 2007 and 2009 are also very similar, and showed a mean length of 177 ± 7-8 centimetres, ranging from 165 to 191 centimetres.

4.2 MEASURABLE ENVIRONMENTAL FACTORS

Thermal regulation in heat stress (heat loss) depends on the principle of heat exchange from the human body to the environment by ways of conduction, convection, radiation and evaporation. Therefore thermal regulatory markers like humidity, temperature and wind speed were taken into account **(See Table 4.2).**

Table 4.2 Measurable environmental factors

	Match 1	**Match 2**	**Match 3**	**Match 4**
Temperature (°C)	33.2	30.8	30.2	31.8
Humidity (%)	11	43	22	21
Wind speed (km.h^{-1})	1.86	2.14	7.20	18.36

***M_1 and M_2 were played on the 22nd and 28th of March 2007 respectively. M_3 and M_4 were played on the 23rd and 27th of March 2009 respectively. All matches were played at 15:00 in the afternoon.**

The average on-field temperatures (°C), relative humidity (%) and wind speed (km.h^{-1}) during the games were Match 1: 33.2 ˚C, 11%, and 1.86 km.h^{-1}, Match 2: 30.8˚C, a relatively high percentage humidity of 43%, and a wind speed of 2.14 km.h^{-1}.

In 2009 during Match 3: the average temperature was 30.2 ˚C, humidity 22% and a wind speed of 7.2 km.h^{-1}. During the last rugby match, Match 4: the average temperature was 31.8 ˚C, humidity 21 %, and the wind speed 18.36 km.h^{-1} respectively.

Only the humidity during Match 2 (43%) together with wind speed (18.36 km.h^{-1}) during Match 4 differed significantly between the four matches.

4.3 PRE- AND POST-MATCH RESULTS OF THE RUGBY PLAYERS

The hydration related measures and calculated values for all four rugby matches are shown in **Figure 4.1** to **Figure 4.3**. Player positions were not represented in large enough numbers to explore possible differences in variables among positions.

4.3.1 Pre- and Post-Match Results: Body mass (kg)

The pre- and post-match body mass (kg), mean, median, and range of the four matches were:

Match 1:	Pre-match body mass	Mean: 71.5 ± 13.7kg Median: 70.0 kg Range: 51.0 to 105.5kg
	Post-match body mass	Mean: 71.0 + 13.6kg Median: 69.3kg Range: 51.2 to 105.0kg
	Pre-post match difference:	$p = 0.003$
Match 2:	Pre-match body mass	Mean: 72.2 ± 13.6kg Median: 72.0kg Range: 53.2 to 105.7kg
	Post-match body mass	Mean: 71.0 + 13.4kg Median: 71.0kg Range: 52.6 to 104.1kg
	Pre-post match difference:	$p = 0.001$
Match 3:	Pre-match body mass	Mean: 71.5 ± 12.8kg Median: 69.7kg Range: 51.5 to 103.8kg
	Post-match body mass	Mean: 71.9 + 12.7kg Median: 69.2kg Range: 51.3 to 102.9kg
	Pre-post match difference:	$p = 0.001$
Match 4:	Pre-match body mass	Mean: 71.3 ± 13.0kg Median: 69.1kg Range: 50.5 to 104.4kg
	Post-match body mass	Mean: 71.0 + 12.9kg Median: 68.9kg Range: 50.8 to 103.8kg
	Pre-post match difference:	$p = 0.029$

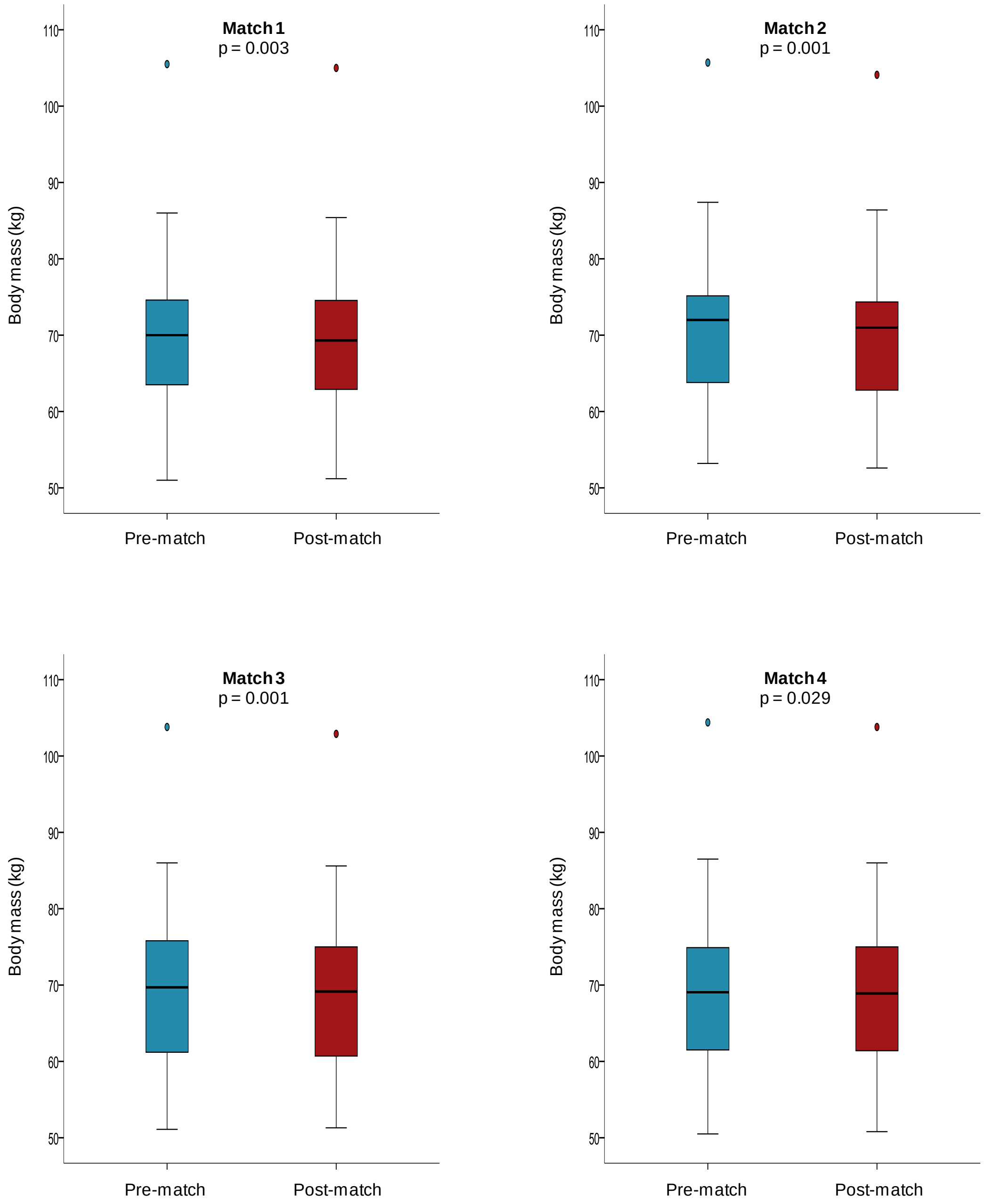

Figure 4.1: Pre-match and post-match body mass (kg) for Match 1-4.

Table 4.3: Players dehydrated according to decrease in body mass

	Decrease in body mass (kg) (mean ± sd)	Players dehydrated post-match n (%)
M_1 (n = 15)	-0.50 ± 0.49	5 (33)
M_2 (n = 15)	-1.18 ± 0.72	10 (67)
M_3 (n = 18)	-0.52 ± 0.49	6 (33)
M_4 (n = 18)	-0.24 ± 0.44	3 (17)

***M_1 = Match1, M_2 = Match 2, M_3 = Match 3 and M_4 = Match 4. M_1 and M_2 were played in the 2007 season. M_3 and M_4 were played in the 2009 season. Dehydration defined as a decrease in body mass in excess of 1%.**

As shown in **Table 4.3 and Figure 4.1** on the first day of data collection (Match 1-March 2007) the players had a pre-match mean body mass of 71.5kg ± 13.7kg and a mean body mass index (BMI) of 22.88 ± 3.98 kg/m^2. On the post-match data collection the mean body mass decreases to 71.0kg ± 13.6kg. A significant difference in post-match body mass (p = 0.0003) was recorded during Match 1. It is interesting to note (see Table 4.3) 5 of the players (33%) were dehydrated post-match. During the second match the pre-match body mass was 72.2kg ± 13.6kg and the BMI 22.82 ± 4.00 kg/m^2 ;the body mass also decreased significantly (p = 0.0325) to 71.0kg ± 13.6kg. It is also important to notice that 10 payers (67%) were dehydrated post-match and that the humidity was the highest (43%) during match two.

In 2009 on the first day of data collection (Match 3) the players had an average mean body mass of 71.5kg ± 12.8kg and a mean body mass index (BMI) of 22.88 ± 3.98kg/m^2. Interestingly, during the post-test the mean body mass increased significantly (p = 0.001) to 71.9kg ± 12.7kg. However, 6 players (33%) were dehydrated post-match. During the last match (Match 4) the average pre-match body mass was 71.3kg ± 13.0kg and BMI 22.82 ± 4.00kg/m^2, and as in Match 1 and Match 2 the body mass also decreased significantly (p = 0.029) to 71.0kg ±12.9kg). Only 3 players (17%) were dehydrated post-match.

4.3.2 Pre- and Post-Match Results: *Urine specific gravity (*SG)

Table 4.4 and **Figure 4.2** showed the mean pre-match and post-match urine specific gravity of all four matches.

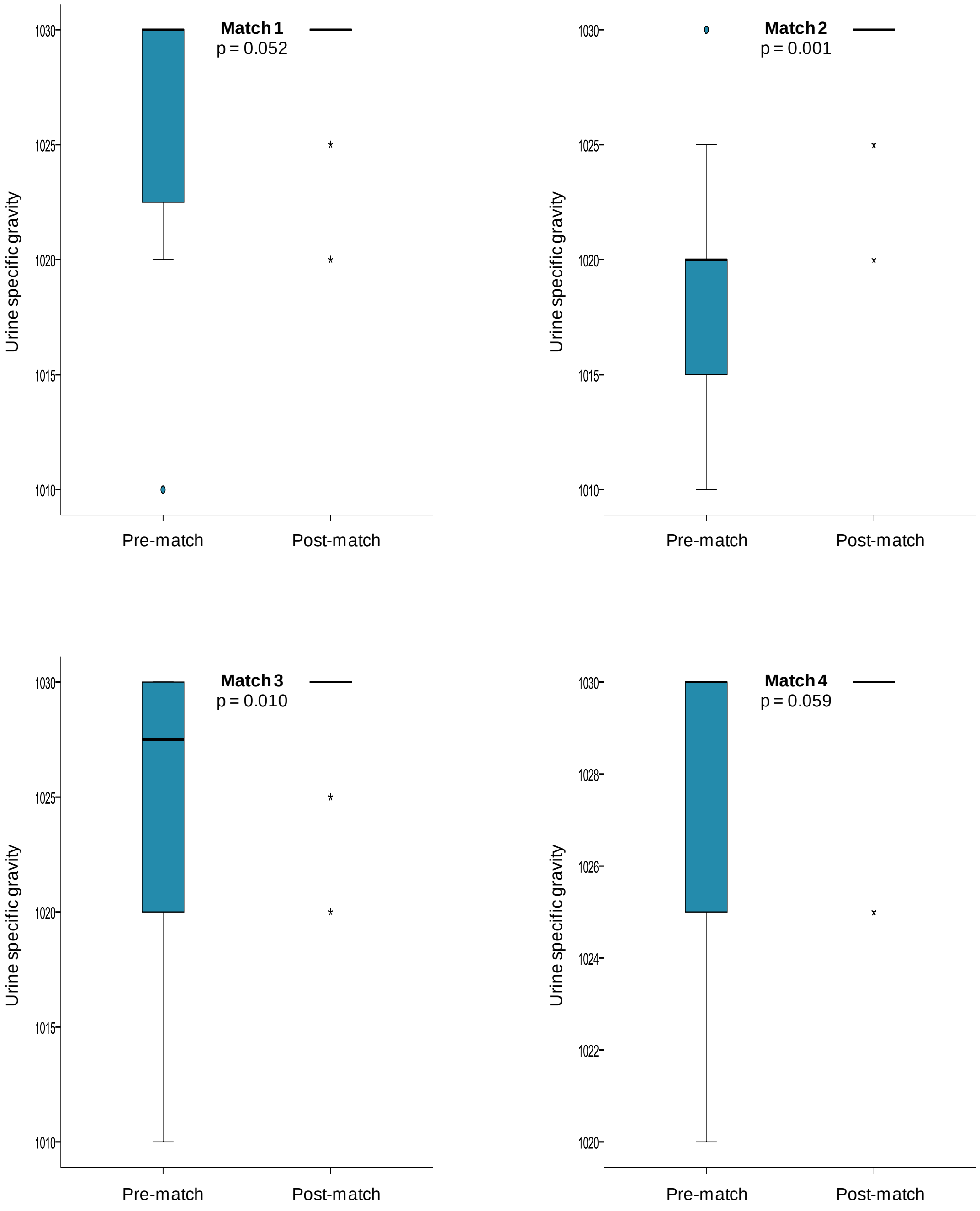

Figure 4.2: Pre-match and post-match urine specific gravity for Match 1-4.

The pre- and post-match urine specific gravity, mean, median and range of the four matches were:

Match 1:	Pre-match Urine SG	Mean: 1026 ± 6 Median: 1030 Range: 1010 to 1030
	Post-match urine SG	Mean: 1029 ± 3 Median: 1030 Range: 1020 to 1030
	Pre-post match difference:	p = 0.052
Match 2:	Pre-match Urine SG	Mean: 1019 ± 6 Median: 1020 Range: 1010 to 1030
	Post-match urine SG	Mean: 1029 ± 3 Median: 1030 Range: 1020 to 1030
	Pre-post match difference:	p = 0.001
Match 3:	Pre-match Urine SG	Mean: 1025 ± 6 Median: 1028 Range: 1010 to 1030
	Post-match urine SG	Mean: 1029 ± 3 Median: 1030 Range: 1020 to 1030
	Pre-post match difference:	p = 0.010
Match 4:	Pre-match Urine SG	Mean: 1028 ± 3 Median: 1030 Range: 1020 to 1030
	Post-match urine SG	Mean: 1029 ± 2 Median: 1030 Range: 1025 to 1030
	Pre-post match difference:	p = 0.059

Table 4.4: Players dehydrated according to urine specific gravity measurements (SG)

	Players dehydrated pre-match n (%)	**Players dehydrated post-match n (%)**
M_1 (n = 15)	11 (73)	14 (93)
M_2 (n = 15)	3 (20)	14 (93)
M_3 (n = 18)	12 (67)	17 (94)
M_4 (n = 18)	17 (94)	18 (100)

***M_1 = Match1, M_2 = Match 2, M_3 = Match 3 and M_4 = Match 4. M_1 and M_2 were played in the 2007 season. M_3 and M_4 were played in the 2009 season. Dehydration defined by urine specific gravity (SG) greater then1030.**

Figure 4.2 and **Table 4.4** show that the urine specific gravity (SG) pre-match was 1026 ± 6 and post-match SG was 1029 ± 3 during match 1. This difference was not significant (p = 0.053). It is important to note (see Table 4.4) that 11 (73%) of the players were already dehydrated pre-match and 14 (93%) were dehydrated after the match.

During Match 2 a mean pre-match SG of 1019 ± 6 and post-match SG of 1029 ± 3 was recorded, which was highly significant (p = 0.001). Only 3 players (20%) were dehydrated before the match, but disturbingly 14 players (93%) after the match. A significant difference (p = 0.01) was also found between the pre-match urine SG (1025 ± 6) and post-match urine SG (1029 ± 3) in Match 3. Again, the same tendency was found: 12 (67%) of the players were dehydrated before the match and 17 (94%) after the match. Lastly, as in Match 1 the pre-match urine SG (1028± 3) and post-match urine SG (1029 ± 2) do not differ significantly (p = 0.059). However 17 players (94%) were dehydrated before the match, and all (100%) the players were dehydrated after the match.

4.3.3 Pre- and Post-Match Results: *Haematocrit (%)*

The pre- and post-match *haematocrit (%)*, mean, median and range of the four matches were:

Match 1:	Pre-match urine HCT	Mean: 0.46 ± 0.03 Median: 0.47 Range: 0.40 to 0.50
	Post-match urine HCT	Mean: 0.46 ± 0.03 Median: 0.46 Range: 0.38 to 0.49
	Pre-post match difference:	p = 0.102
Match 2:	Pre-match HCT	Mean: 0.45 ± 0.04 Median: 0.45 Range: 0.34 to 0.48
	Post-match urine HCT	Mean: 0.45 ± 0.03 Median: 0.47 Range: 0.37 to 0.49
	Pre-post match difference:	p = 0.026
Match 3:	Pre-match urine HCT	Mean: 0.47 ± 0.03 Median: 0.48 Range: 0.41 to 0.50
	Post-match urine HCT	Mean: 0.46 ± 0.03 Median: 0.47 Range: 0.39 to 0.49
	Pre-post match difference:	p = 0.041
Match 4:	Pre-match urine HCT	Mean: 0.47 ± 0.03 Median: 0.48 Range: 0.42 to 0.50
	Post-match urine HCT	Mean: 0.47 ± 0.03 Median: 0.48 Range: 0.42 to 0.50
	Pre-post match difference:	p = 0.564

Figure 4.3 illustrates the mean pre-match and post-match haematocrit (%) of all four matches.

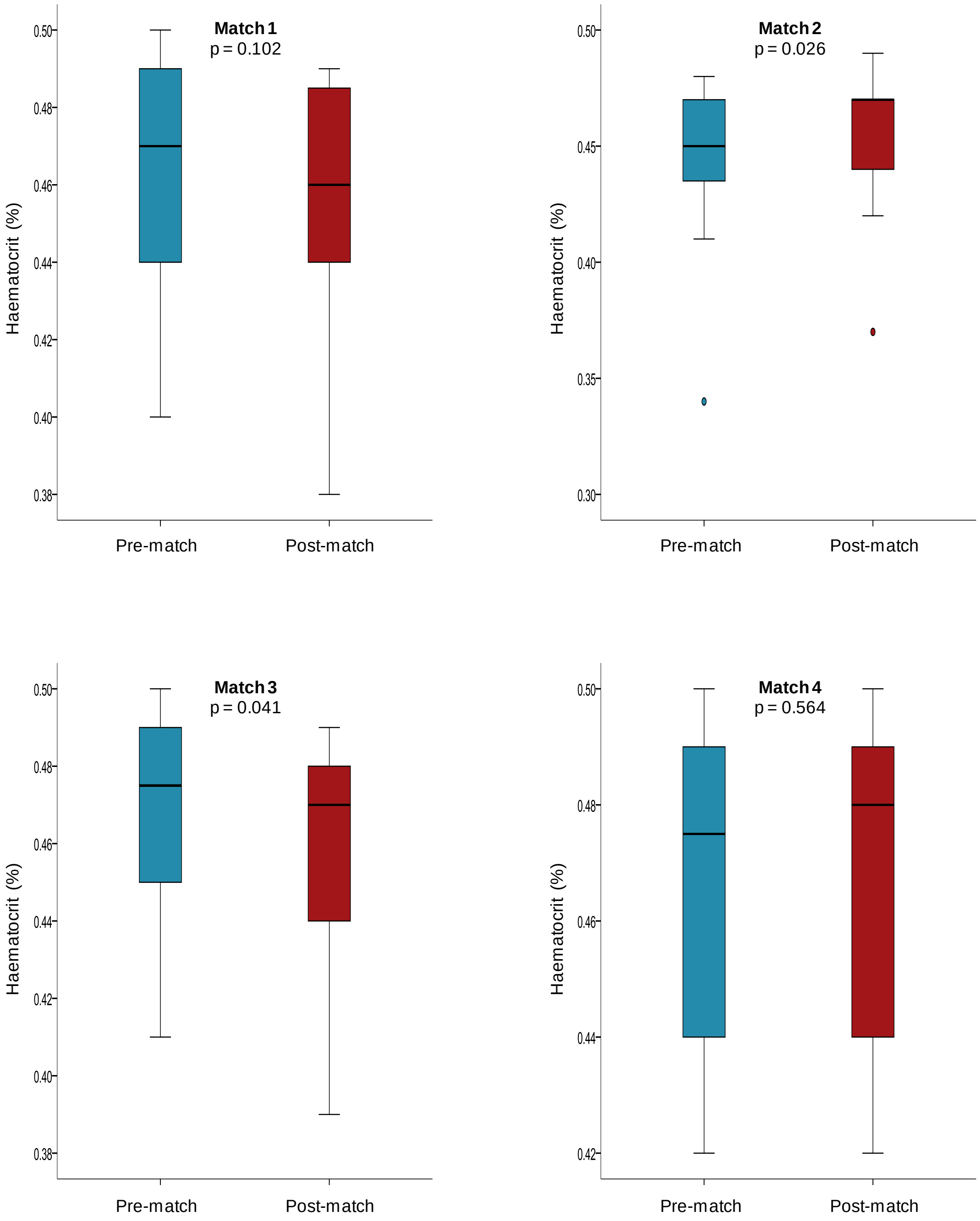

Figure 4.3: Pre-match and post-match haematocrit (%) for Match 1-4.

As shown in **Figure 4.3**, in Match 1 the players had a pre-match mean HCT of 0.46 ± 0.03 and on the post-match data collection the mean HCT was also 0.46 ± 0.03kg. No significant difference in HCT (p = 0.102) was recorded during Match 1.

During the second match the pre-match HCT mean was 0.45 ± 0.04 and the post-match HCT also 0.45 ± 0.04. Interestingly the difference was significant ($p = 0.026$). In Match 3, the average mean HCT was 0.47 ± 0.03 and during the post-test the mean HCT was 0.46 ± 0.03 which differed significantly ($p = 0.041$). During the last match (Match 4) no significant difference in HTC was found as in Match 1 with the average HCT pre-match: 0.47 ± 0.03 and the post-match HCT of: 0.47 ± 0.03.

CHAPTER 5
DISCUSSION OF RESULTS

5.1 INTRODUCTION

Only a limited amount of information is available on school rugby players, especially in the rural areas like the Karoo region. For this reason, we initiated this study to determine the exact situation regarding a lack of knowledge about hydration status, as well as fluid requirements, and fluid guidelines for u/19 schoolboy rugby players in the Karoo. Problems with obtaining accurate data were experienced to a certain extent during the study. This could be due to the reluctance of managers to allow anything to distract players that might thus influence the outcome of the match. There are also considerable practical difficulties in obtaining valid data from players in a competitive match situation, and many of the published reports lack detail on procedures. Bergeron et al*.* (2005) stated in this regard "Unfortunately, specific data and insight regarding fluid loss and intake patterns in young football players during practice or games are very limited".

5.2 DEMOGRAPHIC INFORMATION AND ANTHROPOMETRIC CHARACTERISTICS

Sample characteristics. Thirty-three players completed the study. The sample characteristics are presented in **Tables 4.1 to 4.3**, including the results of the readings taken of *Urine-Specific Gravity (SG)*, blood *haematocrit*, and body mass of every rugby player before and after every rugby match. The players' profiles (anthropometric characteristics) consist of age, height, body mass, and BMI (body mass index). With regard to the anthropometrical evaluation, according to Duthie, Pyne & Hooper (2003), the body weight of rugby players ranges according to game position: forwards 68.9 ± 6.6kg and backs 60.8 ± 5.7kg. The weight difference of forward players is lower in higher competitive levels. As seen in **Table 4.1**, the anthropometric characteristics in our study for 2007 and 2009 is very similar as expected, and showed a mean length of 177 ± 7-8 cm, ranging from 165 to 190 cm, a mean body mass of 71.5 ± 13.7kg and a mean body mass index (BMI) of 22.88 ± 3.98kg/m^2. In terms of fitness, physical activities and dietary habits, players were rather similar. This could be due to the fact that all the players participated in athletics, tug-of-war and rugby practices. Players had also been exposed to similar environmental conditions for several weeks before the game, and they also followed the same conditioning programme under the supervision of the same coach, and all players being resident in the same geographical region.

5.3 PRE- AND POST-MATCH RESULTS OF THE RUGBY PLAYERS: Players dehydrated according to decrease in body mass (kg)

The hydration related measures and calculated values for all the rugby matches are shown in **Tables 4.1 to 4.3** and **Figure 4.1 – 4.3**. There is, however, no general agreement on the best way to assess an individual's hydration status at any given instant (Marshall, 2010). This makes for difficulties in establishing appropriate hydration strategies. Many different markers for the assessment of hydration status have been proposed and normal ranges have been suggested for some of the parameters measured. Hydration status is not easily measured, but acute changes in hydration status are often estimated from body mass change. Changes in body mass are also often used as a proxy measure for sweat losses. There are, however, several sources of error that may give rise to misleading results (Marshall, 2010). Player positions were not represented in large enough numbers to explore possible differences in variables among positions.

As shown in Table 4.3 and **Figure 4.1** on the first day of data collection the players had a pre-match mean body mass of 71.5kg ± 13.7kg and a mean body mass index (BMI) of 22.88 ± 3.98kg/m^2. On the post-match data collection the mean body mass decreased significantly ($p < 0.003$) to 71.0kg ± 13.6kg. Similar reductions in body mass were recorded in Match 2 and Match 4. It is also important to note (see **Table 4.3**) that between 3 (17%) and 10 (67%) of the players were dehydrated post-match according to the decrease in body mass.

Interestingly, only in 2009 during Match 3, the players' average mean body mass of 71.5kg ± 12.8kg increased significantly ($p = 0.001$) to 71.9kg ± 12.7kg. All players drank water and/or carbohydrate-electrolyte drinks during the game. There was a large inter-individual variability in fluid intake. It is clear that the fluid intake of some of the players was excessive, leading to a gain in body mass. It must also be noted that the range in body mass was 50.5kg – 105.7kg which also must have an influence. Some studies have also found that children will voluntarily drink more fluid during exercise in warm or hot environments when provided with a ''sports drink'' (e.g. Gatorade) than when plain water is given (Rivera-Brown et al., 1999; Bergeron et al*.,* 2006) but other studies have not replicated this finding (Rivera-

Brown et al*.,* 2008). However, 6 players (33%) were still dehydrated after Match 3 according to body mass.

The reduction on the body weight as indicative of fluid loss of athletes, according to Fleck & Figueira Júnior (1997) is one of the best evaluations. However, this evaluation does not occur linearly during exercise, and the comparison between initial weight (before physical activity) and final weight (after the end of physical activity) could aid in the fluid replacement during the rest period, once it is associated to the thirst symptoms. According to Rehere and Burke (1996) male rugby players at a temperature ranging from 18 to 23^{o}C may present a sweat rate of 26.6 to 36.7 ml/min. Stover et al. in Begeron et al. (2005) observed moderate rates of sweating (1l per hour) and small body weight deficits (about 1%) in high school players during preseason practice. These measures were slightly lower than losses described in collegiate players training in similar moderate (wet bulb globe temperature [WBGT] 25°C) environmental conditions (Stofan et al., 2003). Pyke and Hahn (1980) reported SR averaging 1.8 litres/h in rugby players competing in ambient temperatures of 38˚C.

In another recent on-field examination of high school players during two successive days of preseason football training in much hotter and more humid conditions (33°C, 56% relative humidity), Bergeron et al. (unpublished findings) noted similar pre- to post-practice body weight deficits of nearly 1%, despite each player consuming about 2 L of water during the daily 2-hour practice session. Moreover, greater sweat fluid losses led to greater body weight deficits. This is not surprising, as athletes often do not match sweat loss with fluid intake during exercise in the heat (Bergeron et al., 2005). These studies support the findings in this study (see Table 4.3 and **Figure 4.2**).

Baker et al. (2009) conclude that the change in body mass during exercise is “an accurate and reliable method to assess” exercise-related changes in total body water. Nolte and Noakes (2010) have major concerns with the manner in which the data were analysed by Baker et al. (2009). However, their analysis shows that the conclusion (Baker et al., 2009) that the change in body mass accurately predicts the change in TBW during exercise is an artefact of the manner in which they analysed their data and is probably confounded by the unusual manner in which they used different biological samples (urine or serum) to measure changes in TBW in the same individuals.

In conclusion, important factors that affect sweat production during exercise are physical conditioning (aerobic and anaerobic), acclimatisation, hydration status, exercise intensity, physical size, and amount of clothing or equipment worn (Godek et al., 2005). Mathews, Fox & Tanzi, (1969) showed significantly higher weight loss in subjects wearing full football equipment than in those wearing only shorts but carrying weight equal to that of the equipment. Wearing heavy protective clothing has been shown by others to decrease the time of onset of sweating and to increase SR (Armstrong et al., 1992).

5.4 PRE- AND POST-MATCH RESULTS OF THE RUGBY PLAYERS: Players dehydrated according to Urine-Specific Gravity (SG)

When considering changes in fluid balance occurring during the matches, the responses of the players from the 4 matches were remarkably similar, with players from both years (2007 & 2009) showing wide individual variability in all parameters. It must be remembered that the body mass range was between 50.4kg and 105.7kg.

The pre-exercise urine specific gravity measures were significantly lower ($p < 0.05$) before all 4 matches than after the matches as expected, and most of the players could have been better hydrated (**see Table 4.4**) at the beginning of the match, which has been shown to reduce physiological strain and enhance heat tolerance in children and adolescents (Bar-Or, Dotan, Inbar, Rotshtein, and Zonder, 1980; Dougherty et al., 2006; Rivera-Brown et al., 2006; Bergeron et al., 2007). In two of the four matches the SG was significantly higher, namely in Match 2: Pre-post-match difference: $p = 0.001$ and Match 3: Pre-post-match difference: $p = 0.010$, the other pre-and post-match difference was almost significant (Match 1: Pre-post-match difference: $p = 0.052$; Match 4: Pre-post-match difference: $p = 0.059$).

Urine specific gravity generally provides a valid indication of hydration status (Armstrong et al., 1992), although urine osmolality is also used and sometimes preferred, especially when plasma osmolality is not available. However, recent fluid (volume and type) and food intake, exercise, and heat exposure can alter renal hemodynamics and thus the accuracy in interpreting these measures to reflect acute total body water gains or losses due to associated changes in plasma osmolality body water distribution, and volume fluctuations in fluid compartments. A limitation in our study was that we did not perform a dietary survey before the games.

Moreover, variations in urine specific gravity and osmolality lag behind rapid changes in sweat losses or fluid intake (Plank, Hipp, and Mahon, 2005).

Measurement of urine osmolality has recently been an extensively studied parameter as a possible hydration status marker. In studies of fluid restriction, urine osmolality has increased to values greater than 900 mOsm/kg for the first urine of the day passed in individuals dehydrated by 1.9% of their body mass, as determined by body mass changes. Armstrong et al. (1994) have determined that measures of urine osmolality can be used interchangeably with urine-specific gravity, opening this as another potential marker. It seems fair to conclude that urinary measures are more sensitive than the other methods, but they may have a time lag over the short term. Euhydration can be defines as a specific gravity of 1.013 – 1.029 units. However, the validity of USG measures is questionable after an acute change in body mass, as would occur following exercise-induced sweat loss (Popowski et al., 2001).

It seems clear that not all players were well hydrated at the beginning of the matches. When looking at the pre-match hydration status of the players, between 20% and 94% were dehydrated pre-match. The results of this study confirm the findings of other researchers that a substantial proportion of the players were likely dehydrated before kick-off, in spite of the games being played late in the day and affording ample opportunities for fluid intake in the hours beforehand (Maughan et al., 2007). The reason for this discrepancy is not perfectly clear, but we could argue that a lack of knowledge of the players, their parents, as well as the coaching staff, could play a role.

Godek et al*.* (2005) found that SR in footballers performing intermittent exercise during practice was higher than that of athletes running continuously in the same environmental conditions. Although making direct comparisons is difficult because of the different exercise intensities inherent in different sports, it is important to discuss SR reported in other groups of athletes. Sweat losses during a 90-minute football-training session in warm climates, were up to 3,5L in some individuals (Maughan et al., 2007).

Body-water content of some of the players in this study at the end of the game has been substantially less than optimal. When analysing the players' post-match hydration status it is alarming to find that almost all the players (93% and 100%)

were dehydrated. In a match situation, the rate of gastric emptying of ingested fluids, and therefore the availability of ingested fluid and carbohydrate, is likely to be reduced, increasing the risk of gastrointestinal discomfort and limiting the benefits of drinking. Most of the studies showing adverse performance effects of hypo hydration on exercise performance, skill, and cognitive function have been conducted in warm or hot environments, and notwithstanding also the theoretical energetic advantage of a reduced body mass, there might be disadvantages to pre-match hypo hydration. Sweat rate, sweat composition, and drinking behaviour varied greatly between individuals (Maughan et al., 2007).

According to Bergeron et al. (2005), as with adult athletes, maintaining fluid balance can be difficult for young football players, especially in hot and humid conditions. Intensity and duration of practice, scheduling of fluid breaks, uniform configurations, and number of sessions per day are also key factors in tempering or exacerbating this challenge. Unfortunately, specific data and insight regarding fluid loss and intake patterns in young football players during practice or games are very limited. These data confirm and extend observations made on elite players in training and suggest that generalised guidelines on fluid intake might be of little use to individual players. Given the large variability that was observed in hydration status, and drinking behaviours, individualised assessment of fluid and electrolyte needs might be recommended to optimise fluid-replacement strategies in both training and competition. If a value for urine SG of 1.029 is accepted as a cut-off point for indication of adequate hydration status, 9 (60%) of the 15 players in Match 1 were not well hydrated when they arrived at the match grounds. This is potentially a concern, because of the possible negative effects of prior dehydration on performance and skilled performance. Therefore, education is necessary to encourage players to increase their fluid intake. Inadvertently, the coach did encourage the intake of more fluids, and only 3 (20%) of the 15 players were dehydrated prior to Match 2.

The large difference in post-match body mass loss, between Match 1 and Match 2 (600gm=600ml), suggests that, as one would expect, the sweat loss of the players during Match 1 was much less than that of Match 2, because of dehydration status of 73.3% of subjects before Match 1 as seen by urine SG values (> 1,029), as well as humidity of 43% during Match 2, in contrast to 11% at Match 1. It is important to recognise that the body mass changes reflect only those attributable to changes in total body water. Fluid replacement strategies designed to prevent symptomatic hyponatremia should target both fluid and electrolyte intake. Athletes should be encouraged to develop customised fluid replacement programmes, through trial and error. Opportunities for fluid intake during the game are limited, and the ability to empty ingested fluid from the stomach and to absorb it in the small intestine may be compromised, so it would seem appropriate for players to ensure that they are fully hydrated before beginning either training or match play.

Bergeron et al. (2005) also noted that the 10 players presented with elevated urine specific gravities on day 1, suggesting that they were not well-hydrated at the start of practice. Notably, the same players had even higher urine specific gravities at the start of practice on day 2, suggesting that their recovery fluid intake to restore sweat fluid losses from the previous day was insufficient and that they were more dehydrated than on day 1. Stover et al., as sited in Bergeron et al. (2005) also examined day-to-day changes in body weight and pre-practice hydration status across 5 days of the two-a-day training sessions. The players' body weights remained steady, after an initial decrease (0.5kg) after the first day, and urine specific gravities from pre-practice samples remained high (yet unchanged), suggesting that these players were not well-hydrated as well before the start of each practice. The above observations suggest that young football players tend to *begin* practice measurably dehydrated and this continues on successive days of practice, especially in the heat, even when the athletes have ample time and opportunity to rehydrate overnight. Large sweat losses, insufficient fluid intake, and consequent fluid deficits could likely impair performance and may increase the risk of hyperthermia and heat injury (Stofan et al., 2003). However, Rowland (2008) in his article "**Thermoregulation during exercise in the heat in children: old concepts revisited"** disagrees and challenges these traditional old concepts. Rowland (2008) stated **"***Children possess certain physiological and anatomic characteristics that have traditionally been considered to impair thermoregulatory responses to exercise in the heat: low exercise economy, high ratio of body surface area to mass, diminished sweating capacity, and less cardiac output at the same work load compared with adults. Consequently, children have been regarded as an at-risk group for not only decrements of physical performance but also heat injury during physical activities performed in conditions of high ambient temperature. Recent investigations that have directly compared thermoregulatory responses to exercise in the heat in children and adults have challenged these traditional concepts. Such studies have failed to indicate group differences in heat dispersal when adult-child comparisons are appropriately considered in respect to relative exercise intensity.*

These findings imply that no maturational differences exist in thermal balance or endurance performance during exercise in the heat, nor that child athletes are more vulnerable to heat injury".

5.5 PRE- AND POST-MATCH RESULTS OF THE RUGBY PLAYERS: Players dehydrated according to blood *haematocrit*

According to Mashiko et al. (2004) Hb and HCT levels are used to assess hydration status and increases in blood concentration of various substances resulting from high sweat rates after exercise.

As shown in **Figure 4.3**, the pre-match mean HCT and the post-match mean HCT was in the range of 0.46 - 0.47. However, in two of the matches significant differences ($p < 0.05$) were recorded. Physical activity in a hot environment accelerates sweat loss and consequent dehydration, resulting in increases in blood Hb and HCT levels (Harrison, 1985). Measurement of Hb-concentration and HCT has the potential to be used as a marker of hydration status or change in hydration status, provided a reliable baseline can be established. In this regard, standardisation of posture for a time prior to blood collection is necessary to distinguish between postural changes in blood volume, and therefore in haemoglobin concentration and HCT, which occur (Harrison, 1985) and change due to water loss or gain.

It should also be indicated that there is no correlation between blood (HCT) and urine-SG values in our study. It seems fair to conclude that urinary measures are more sensitive than the other methods, but they may have a time lag over the short term. Mashiko et al. (2004) find also that although their summer training camp with 25 college rugby players took place in a hot environment, Hb and HCT levels did not change significantly after the camp. We speculate that the athletes ingested sufficient water during and after daily exercise to compensate for water lost through sweating.

CHAPTER 6
CONCLUSION

6.1 INTRODUCTION

Rugby games are often scheduled for the hottest part of day during the summer. In the hot South African sun, rugby players are often exposed to temperatures that pose risks. According to Yeargin et al. (2010) many rugby players are unsure of the type and amount of fluid they should consume, and healthcare professionals, coaches, and conditioning coaches can assist players by relaying appropriate hydration information.

Howe and Boden (2007) stated that heat-related illnesses have received substantial public attention in the United States after the recent deaths of collegiate and professional athletes in the National Collegiate Athletic Association (NCAA), National Football League, and Major League Baseball from heat stroke, but to the best knowledge of the researcher, this is not the case in South-Africa. Because exertion heat stroke is entirely preventable, this study has focussed on prevention tactics in rugby dehydration that may help reduce the incidence of catastrophic heat-related illness events in South-Africa, especially in rural areas. These prevention strategies include scientific evidence and detailed advice on acclimatisation to the heat; proper fluid replacement before, during, and after a rugby match; wearing proper rugby jerseys during certain environmental conditions; and early recognition of heat-related illness via direct monitoring of players by other players, coaches and medical staff.

Marshall (2010) also stated that the ability to predict a player who is most susceptible to heat related injury and thus offer appropriate early intervention treatments such as specific hydration recommendations would be a major breakthrough in prevention of dehydration. Children may require specific recommendations if, for example, the amount of their sweat electrolyte losses is different. Although a base of observational case data, physiological information, and expert opinion exists (Marshall, 2010), the science surrounding this field is devoid of health communication and health behaviour research, and there is a pressing need for analytical studies to evaluate intervention programmes and/or identify new risk factors. There is also a need for ongoing data collection on heat injury incidence and on the knowledge, attitudes and behaviours towards heat injury among youth athletes, their care givers and their coaches. Because sporting activity can occur in hot conditions, such as rugby games in the Hopetown district in South Africa, sports medicine clinicians must be well versed in both prevention and management of heat-associated illness.

6.2 RECOMMENDATIONS

The aim of this study was to determine the dehydration status of u/19 school rugby players during a game of rugby in the Hopetown district in high temperatures. In conclusion, the researcher highlights some of the key findings and then gives specific recommendations.

6.2.1 Key findings

1. It is important to note (see **Table 4.3**) that between 3 (17%) and 10 (67%) of the players were dehydrated post-match according to the decrease in body mass.

2. There was a large inter-individual variability in fluid intake. It is clear that the fluid intake of some of the players was excessive, leading to a gain in body mass. It must also be noted that the range in body mass was 50.5kg – 105.7kg which also must have an influence on fluid intake.

3. The pre-exercise urine specific gravity measures were significantly lower ($p < 0.05$) before all 4 matches than after the matches as expected, and most of the players could have been better hydrated (see **Table** 4.4) at the beginning of the match.

4. In two of the four matches the SG was significantly higher - Match 2: Pre-post match difference: $p = 0.001$ and Match 3: Pre-post match difference: $p = 0.010$. The other pre-and post-match difference was almost significant (Match 1: Pre-post match difference: $p = 0.052$; Match 4: Pre-post match difference: $p = 0.059$).

5. When analysing the pre-match hydration status of the players between 20% and 94% of the players were dehydrated pre-match. Therefore, the body-water content of some of the rugby players at the end of the game

has been substantially less than optimal. When analysing the players' post-match hydration status it was alarming to find that almost all the players (93% and 100%) were dehydrated.

6. As shown in **Figure 4.3**, the pre-match mean HCT and the post-match mean HCT was in the range of 0.46 - 0.47. However, in two of the matches significant differences ($p < o.o5$) were recorded. Physical activity in a hot environment accelerates sweat loss and consequent dehydration, resulting in increases in blood Hb and HCT levels.

7. Measurement of haemoglobin concentration and HCT has the potential to be used as a marker of hydration status or change in hydration status, provided a reliable baseline can be established.

6.2.2 Prevention measures for dehydration in rugby players

As with risk factors, there are no controlled studies of interventions, and prevention recommendations have been based on clinical observation and physiological information. Nevertheless, a recent series of systematic reviews, consensus statements and expert opinions has generated a core set of prevention recommendations (Howe and Boden, 2007; Armstrong et al., 2007; Mueller and Colgate, 2009; Casa and Csillan, 2009; Brukner and Khan, 2012). As with risk factors, much of this information comes from studies of adults, and the extrapolation to youth populations is largely untested in empirical terms.

However, results of new research (Pediatrics 2011) indicate that, contrary to previous thinking, youth do not have less effective thermoregulatory ability, insufficient cardiovascular capacity, or lower physical exertion tolerance compared with adults during exercise in the heat when adequate hydration is maintained. Accordingly, besides poor hydration status, the primary determinants of reduced performance and exertional heat-illness risk in youth during sports and other physical activities in a hot environment include undue physical exertion, insufficient recovery between repeated exercise bouts or closely scheduled same-day training sessions or rounds of sports competition, and inappropriately wearing clothing, uniforms, and protective equipment that play a role in excessive heat retention. Because these known contributing risk factors are modifiable, exertional heat illness is usually preventable. With appropriate preparation, modifications, and monitoring, most healthy children and adolescents can safely participate in outdoor sports and other physical activities through a wide range of challenging warm to hot climatic conditions (*Pediatrics* 2011).

Taking the available literature and findings from this study into consideration, the following guidelines are suggested to minimise the risk of heat illness:

- Perform adequate conditioning, especially in the pre-season.
- Undergo acclimatisation by training in the heat, if competing in unaccustomed heat or humidity.
- Avoid adverse conditions.
- Alter training and game times.
- Logically, frequent rest breaks, reductions in exercise in very hot conditions and adequate hydration are expected to reduce risk.
- Drink **appropriate amounts** of fluids before the game.
- Some studies have found that children will voluntarily drink more fluid during exercise in warm or hot environments when provided with a ‘‘sports drink’’.
- Wear appropriate clothing.
- Ensure sportspeople and officials are well educated.
- Provide proficient medical support.

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APPENDIX A

IRB HEAT GUIDELINES

APPENDIX A

INTERNATIONAL RUGBY BOARD
Putting players first

IRB Heat Guideline

Introduction

Exercising in extreme environments is known to be associated with medical complications. The American College of Sports Medicine has developed guidelines for exercising in a hot environment. These guidelines were developed to provide advice during endurance events, in particular, distance foot races. The ACSM recommended using, an on-site Wet Bulb Globe Temperature (WBGT) reading and recommended that consideration should be given to cancelling events when the WBGT was above 28.

Rugby is a team sport played by athletes of varying stature where the game is of an intermittent nature and limited to two 40 minute halves. The intermittent nature of the sport probably allows for greater access to fluid intake during competition when compared with endurance events.

Considering the significant differences between endurance foot racing and Rugby, a review of other sports Heat Guidelines, more closely aligned to Rugby was undertaken. This investigation revealed that the National Rugby League (NRL) in Australia had developed Heat Guidelines during 2000 following research undertaken by Dr John Brotherhood.

The outcome of this research revealed that "relying on the WBGT was limited as it only took into account ambient temperature, globe temperature (radiant heat) and humidity and 70% of this reading was dependent on humidity". In addition it was identified that "the WBGT was not recorded by weather bureaus and a figure had to be estimated from the Wet Bulb Temperature".

Dr. Brotherhood placed more weight on the Belding Hatch Stress Index (BHSI) than the WBGT. BHSI is calculated by dividing the Evaporative requirement of the player by the Maximum Evaporative capacity of the environment x 100. A figure of 100 represents an equilibrium between heat loss and heat gain.

In 2001 the NRL adopted guidelines based on the Heat Stress Index measured using a Whirling Hygrometer to assess environmental conditions. Since the introduction of these guidelines there has not been a reported incidence of heat illness during a competition game.

The recommended guidelines are based on utilizing the Heat Stress Index as measured by the Whirling Hygrometer at the site of the game.

- The following combination of factors is often reported in cases of heat stroke.
  - (a) Lack of acclimatization. Acclimatization is a gradual exposure to increasing heat loads and work volumes and is an important factor in reducing a potential heat stroke incident.
  - (b) Impaired individual temperature regulation on that day eg viral illness, commencing activity dehydrated, use of "adverse" medication eg stimulants.
  - (c) Extra effort on that day eg to make the team, impress the coach or achieve a goal.

Research has not identified a specific temperature and / or humidity when exercise is not recommended.

Recommended strategies to reduce heat stress

Critical steps

The following are the critical steps in minimizing heat illness during competition and training

1. Education
2. Appropriate scheduling of training and playing
3. Acclimatization
4. Assessment of extreme conditions
5. Implementing Interventions.
6. Crises Management.

Education

Players should be advised to

- (a) Report to medical staff previous episodes of heat illness.
- (b) Report to coaching and medical staff, any current viral infection especially if associated with a temperature – this should be re-emphasized to players regularly.
- (c) Avoid using stimulants eg pseudoephedrine or caffeine prior to training
- (d) Always start a training session well hydrated
- (e) Always drink fluids during a training and playing session
- (f) Report early the signs of heat stress - cramps, headaches, nausea, vomiting – this should be re-emphasized to players regularly.

Coaching, management and medical staff should

- (a) Be aware of the early signs of heat stress – cramps, headaches, nausea, vomiting, reduced performance, poor coordination, "abnormal" behaviour.
- (b) Implement processes that encourage the reporting of current viral infections.
- (c) Implement strategies that encourage all players to commence a playing and/or training session fully hydrated eg pre and post exercise weigh in, pre-exercise urine specific gravity assessments.
- (d) Recognise and accept the potential seriousness of a severe heat illness, that is, heat stroke.

Scheduling training and playing

If practical, training and playing should be scheduled when ambient temperatures, radiant heat (direct sunlight) and humidity are expected to be at acceptable levels. Utilising the Heat Stress Index, a guide to acceptable levels would be –

a) temperature ≤ 30 degrees Celsius

b) humidity ≤ 60%

There is no evidence to suggest training or playing at higher temperature and humidity levels will result in a heat illness.

Historical data should be obtained from the local Bureau of Meteorology to identify times throughout a day and month when these conditions are most likely to prevail. This information can then assist with scheduling training to minimize risk.

Acclimatisation

Allowing athletes to acclimatize should also be a component of managing potential heat illness. Activity in hot humid conditions should be introduced gradually to allow athletes to acclimatize to these difficult conditions.

Acclimatisation is reported to occur following 7 – 10 days of exposure to the appropriate environment.

Assessment of extreme conditions

In extreme weather conditions an objective assessment of the environment may be required to assist in determining the safety of the prevailing conditions.

Research and experience has confirmed that the "Heat Stress Index" measured using a "Whirling Hygrometer" is both practical and reliable and it is recommended that each Rugby Ground have access to a Whirling Hygrometer to measure the weather conditions. This Index (see attached chart) takes into account Air Temperatures at various Relative Humidity. Prior studies have confirmed that if the Heat Stress Index % is below 150, the risk to players should be minimal. Experience suggests that players are able to cope with an Index as high as 250 but it is recommended that all of the Heat Illness Prevention Interventions listed below are applied if the Index is above 150.

From a practical perspective, the hygrometer needs to be whirled for 20 seconds to obtain readings. Three measurements should be undertaken and averaged.

The Whirling Hygrometer (≈ $A 175) can be obtained from Arthur Bailey Surgico Pty Ltd 55 Lilyfield Road Rozelle 2039 NSW. Ph – (02) 9555 1588.
www.abailey.com.au

Heat illness prevention interventions

Whilst the Heat Stress Index has been successfully utilized in Australian Rugby League the IRB recognizes that Rugby players are potentially at a higher risk of a heat illness than Rugby League players. The reasons for this opinion are listed below and have been taken into account when formulating the IRB Prevention Interventions:

1. Rugby athletes are generally bigger athletes
2. League players are able to access interchange during their games
3. Aerobic fitness of League players is higher.
4. Less "hugging" in League
5. Rugby athletes from Northern Hemisphere less acclimatized.

The following Game Day Interventions should be implemented when the Heat Stress Index is above 150:

Game day interventions

1. Provision of dressing room fans if air conditioning not available
2. Provision of side line shade if game played during the day when radiant heat (direct sunlight) is a contributing factor.
3. Strategic positioning of towels immersed in ICE water around the ground – behind goal posts and at junction of each quarter line and side line.
4. 2 minute break at the 20 minute mark of each half. The focus of this break should be threefold, a medical assessment of each athlete for signs of heat stress, cooling of athletes and re-hydration. It is suggested that cooling would be best achieved by immediately removing jersey and shoulder pads, application of ICE water to head ± body. Utilising sideline fans and shade (if game during day) would also be ideal. It should be noted that a 2 minute break has been recommended (as opposed to a 1 minute break) because the focus during this break is primarily medical assessment and cooling.

It should be noted that increasing access of water carriers to the field has not been recommended as it is felt that there is adequate breaks in the course of a game to allow water carrier access and player re-hydration.

Training guidelines

The recommendations re education, scheduling and acclimatization should also apply to training sessions.

Training sessions are more easily manipulated and the following is recommended during periods of significant heat stress (Index > 150):

(a) Plan training at the most appropriate time of the day – check Bureau of Meteorology statistics.
(b) Plan training to allow players to acclimatize – gradually increase exposure times and training volumes and intensity.
(c) Identify players who may have a viral infection or are volume depleted.
(d) Be aware of the early signs of heat stress
(e) Schedule fluid breaks every 10 – 15 minutes.
(f) Each 40 minute session should be followed by a 15 minute rest period where players are rested, **cooled**, re-hydrated and protected from radiant heat (direct sunlight).

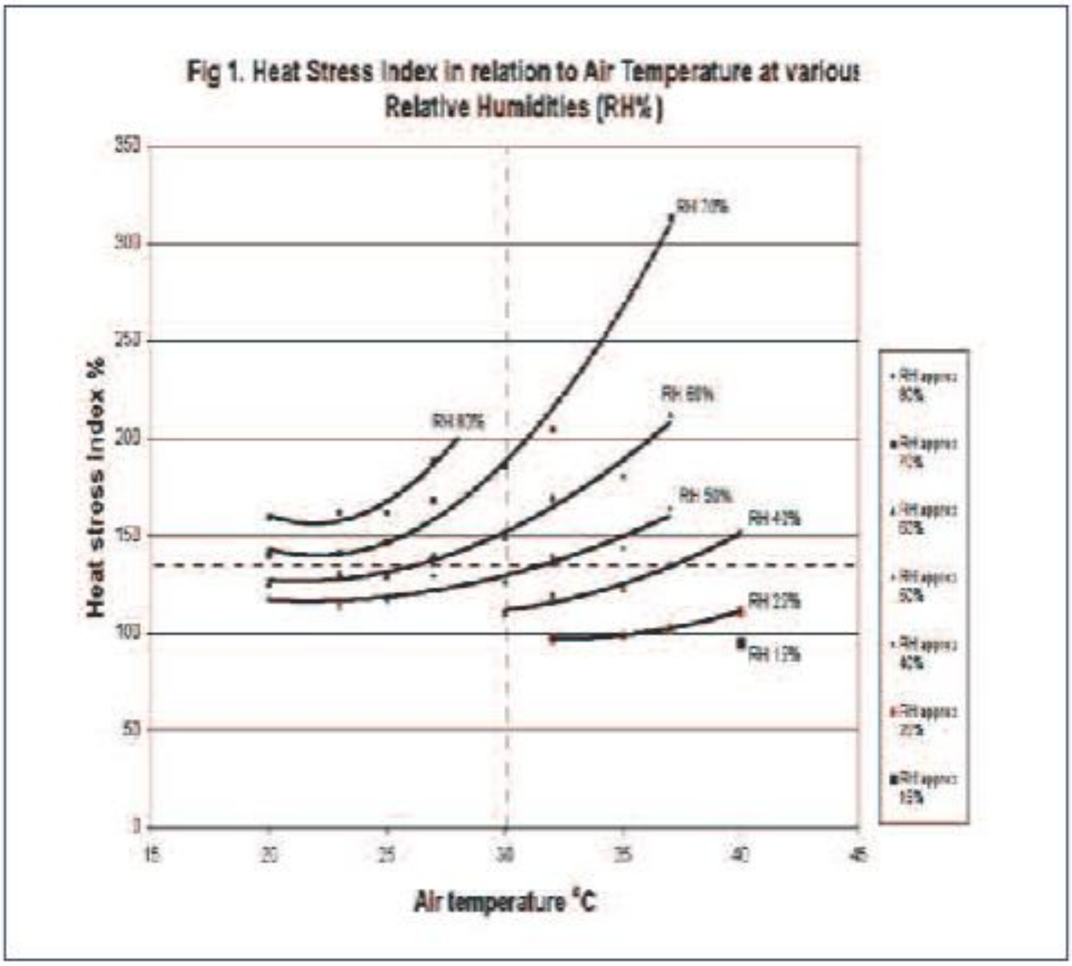
In addition the following are also recommended:

(a) training apparel should be light weight, loose fitting and allow evaporation of sweat
(b) adequate quantities of ICE should always be available
(c) drinks should be provided at a temperature which are known to assist with rapid absorption - less than 15 degrees Celsius (ICE fluids)
(d) shade should be utilized during any break in training

Crisis management

Each training and playing venue should have in place a crises management plan. This plan should reflect that prompt recognition and immediate total body cooling will resolve or mitigate the problems of hyperthermia.

Figure 1: Heat Stress Index in relation to air temperature at various relative humidities (RH%)



Conclusion

These Heat Guidelines are intended to minimize the risk of the onset of heat illness and provide a framework for each team and venue to operate safely during periods of climatic extreme.

APPENDIX B

MEASUREMENT FORM

MEASURING FORM

APPENDIX B				BEFORE MATCH			AFTER MATCH			FOR OFFICE USE
No.	Sex	Age	Length	Mass	Urine SG	Blood Haematocrit	Mass	Urine SG	Blood Haematocrit	

APPENDIX C

PERMISSION LETTER FROM PRINCIPAL OF HOPETOWN HIGH SCHOOL

Date ………../…../ 2007

I, MC Smit, as principal of Hopetown High School, do hereby give permission to Dr PvdW Vermeulen (local medical practitioner in Hopetown) to conduct his research study at Hopetown High School on the u/19 rugby players on condition that it is done with the informed written permission of both the parents and the children.

Signed at Hopetown on this ………..day of ……….2007.

……………………
MC SMIT

…………………
Witness (1)

………………..
Witness (2)

APPENDIX D

INFORMED CONSENT FROM PARENTS

Date: ………./…2007

Dear parent

I hereby wish to inform you that I am busy with a Masters Degree in Sports Medicine at the University of the Free State.

After the research protocol has been approved by the Ethics Committee of the University of the Free State, I want to take the liberty of requesting your permission to conduct research on your child/rugby player of the u/19 rugby team of Hopetown High School. This comprises taking urine and blood samples +/- 10 minutes before and after the match. The mass of your child will also be taken 10 minutes before and after the match.

The costs of the laboratory and all other costs will not be for your account. I undertake to provide free medical service to all prospective players on and off the rugby field during and after the match. This study will be conducted during the beginning of the rugby season (+/- February/March 2007 / 2009) and only at 4 matches. All parents who are satisfied with the above can please sign the informed letter of consent below.

I, …………………………………parent of ………………………. …., hereby give permission to Dr PvdW Vermeulen to conduct his research study as set out above.

……………………….. **…..…………….**
Parent **Witness (1)**

……………….
Witness (2)

APPENDIX E

INFORMED CONSENT FROM PLAYERS

Date: ………./…2007

Dear U/19 rugby player

I hereby wish to inform you that I am busy with a Masters Degree in Sports Medicine at the University of the Free State.

After the research protocol has been approved by the Ethics Committee of the University of the Free State, I want to take the liberty of requesting your permission to conduct research on yourself as rugby player of the u/19 rugby team of Hopetown High School. This comprises taking urine and blood samples +/- 10 minutes before and after the match. Your mass will also be taken 10 minutes before and after the match.

The costs of the laboratory and all other costs will not be for your account. I undertake to provide free medical service to all prospective players on and off the rugby field during and after the match. This study will be conducted during the beginning of the rugby season (+/- February/March 2007) and only at one match. If you are satisfied with the above please sign the informed letter of consent below.

I, ……………………… …., hereby give permission to Dr PvdW Vermeulen to conduct his research study as set out above.

……………………….. **…..……………**
U/19 Rugby player **Witness (1)**

……………….
Witness (2)