

Position Statement

Current knowledge on playing football in hot environments

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Preamble

This document focuses on recommended measures and precautions for playing football (soccer) in a hot environment with an emphasis on the prevention of heat illness in competitive male players. The prevalence of heat illness in football is unclear but incidents where footballers from various codes have died with heat-related symptoms have been reported (Mueller, 2008).

Exercising in hot environments

The capacity to exercise in hot environments is markedly reduced relative to that in cooler conditions (Galloway & Maughan, 1997). Mechanisms responsible for this reduction include a variety of homeostatic changes that occur in parallel with the increase in the body core temperature. According to Nybo (2010), these factors can generally be divided into (1) changes in the central nervous system that lead to so-called central fatigue and (2) impairments of cardiovascular function that reduce oxygen delivery and consequently reduce aerobic energy turnover within the exercising muscles and provoke peripheral fatigue. In addition, the extremely high muscle temperatures observed in football (Mohr et al., 2010) may also impair muscle function through both contractile (Van der Poel & Stephenson, 2002) and neural (Racinais et al., 2008) alterations.

In cool conditions, most of the endogenous heat produced by players is released from the skin to the surroundings via convection; however, as environmental temperature increases, evaporation becomes the dominant, and in some instances the only, mechanism for heat dissipation (Armstrong & Maresh, 1998). In football, quadriceps muscle temperature of up to 42 °C have been observed in some individuals (Mohr et al., 2010), while, even in temperate environments, internal body temperature can reach 39–39.5 °C with individual values over 40 °C (Ekblom, 1986; Mohr et al., 2004). The combined effects of this increase in internal body temperature and fatigue induce a reduction in the amount of sprinting, high-intensity running and distance covered in the second half of matches (Mohr et al., 2003). This problem is aggravated in conditions of low environmental wind speed and/or high humidity as the rate of evaporative heat loss is compromised, imposing further physiological strain on the body to release heat. The resulting increase in body temperature can potentially lead to heat illness and heatstroke.

Heat illness and heatstroke

Heatstroke and even heat-related deaths are well documented in high school and collegiate American Football. In 2003, the National Collegiate Athletic Association implemented new regulations for summer conditioning programs: despite this, there were

six recorded heat-related deaths and one sickle cell death in high school and collegiate American Football in 2008 (Mueller, 2008). Exertional heat illnesses have been classified into the three main categories described in Table 1, which summarizes recent reviews (Binkley et al., 2002; Armstrong et al., 2007). In addition Table 1 also provides immediate actions that can be taken in the event of a player experiencing a heat-related illness. However, it is important to note that there are large inter-individual differences in heat tolerance such that some athletes can tolerate internal body temperatures well above 40 °C without medical consequences (Byrne et al., 2006) or evident loss of performance, while others may experience exertional heat stroke.

It has to be acknowledged that the first protective mechanism against exertional heat illness is a reduction in the intensity and duration of the activity. In the laboratory, most volunteers become unwilling to continue exercising, when their core temperature exceeds ~ 40 °C (González-Alonso et al., 1999; Nybo & Nielsen, 2001). However, trained subjects may attain core temperatures as high as 41 °C during competitive running (Pugh et al., 1967; Ely et al., 2009). As described above, the combined effect of an increase in internal body temperature, cardiovascular strain, and central and muscular fatigue in competitive football is to reduce the amount of sprinting, high-intensity running, and distance covered in the second half of matches (Mohr et al., 2003).

Medical staff should be aware of the potential interaction between prescription drugs and heat tolerance and evaluate these before prescribing. For example, antihistamines, commonly found in allergy, cold and sleep medications have been associated with impaired sweating (Department of the Army and Air Force, 2003). Specific attention has to be paid when players are using medication or drugs that can influence thermoregulation, such as methylphenidate (Ritalin) or bupropion that seem to override inhibitory signals from the central nervous system to stop exercising when internal body temperature becomes high (Meeusen & Roelands, 2010).

Environmental measurements

A combination of exercise and hot and humid environmental conditions is the main cause of the heat-related conditions described in Table 1. However, even at rest, hot environmental temperatures can threaten the health of coaches, technical staff, and spectators. For instance, a 2-week heat wave across Western Europe during August 2003 was associated with an excess mortality of nearly 14000 deaths in France alone (Rey et al., 2007), which suggests that excessive passive heat can predispose to mortality. The sick and the elderly are most

Table 1. Warning signs and symptoms of heat illness and injury

Condition	Sign or Symptom	Immediate Actions
Heat Syncope	Dehydration	Remove from training or competition Allow player to rest in shade, fan, and spray with water Loosen or remove clothing Provide sips of water Monitor temperature and check for mental confusion
	Fatigue	
	Tunnel vision	
	Pale or sweaty skin	
	Decreased heart rate	
	Dizziness	
	Light-headedness	
Fainting	Monitor temperature and check for mental confusion	
Exercise (heat exhaustion (Rectal temperature between 37 and 40°C)	Normal or elevated body temperature	Immediately call for ambulance or emergency services while doing the following Lay the player down in the shade with feet elevated Undress the player as much as possible Pour cool water over the player and fan Cool by best means possible, such as water immersion or ice packs If the patient is still conscious give sips of water Monitor airway and breathing
	Dehydration	
	Dizziness	
	Light-headedness	
	Syncope	
	Headache	
	Diarrhea	
	Decreased urine output	
	Persistent muscle cramps	
	Pallor	
	Profuse sweating	
	Chills	
	Cool, clammy skin	
	Intestinal cramps	
Urge to defecate		
Weakness		
Hyperventilation		
Exertional heat stroke (Rectal temperature >40°C)	Body temperature (< 40°C)	Immediately call for ambulance or emergency services while doing the following Lay the player down in the shade with feet elevated Undress the player as much as possible Pour cool water over the player and fan Cool by best means possible, such as water immersion or ice packs If the patient is still conscious give sips of water Monitor airway and breathing
	Central nervous system changes	
	Dizziness	
	Drowsiness	
	Behavioral changes	
	Confusion/ disorientation	
	Unsteady walk	
	Seizures	
	Loss of consciousness	
	Coma	
	Dehydration	
	Weakness	
	Hot and wet or dry skin	
	Hypotension	
Hyperventilation		
Vomiting		
Diarrhea		

susceptible, but young apparently healthy individuals can also be affected (Kovats & Hajat, 2008). Since the 1970s, organizers of fun runs have taken specific measures (i.e. running the event in cooler conditions and using spotters to identify exhausted runners before they collapse) to reduce the incidence (Richards et al., 1979a) and improve the management (Richards et al., 1979b) of heat-related casualties. When the environmental conditions are such that the capacity to dissipate heat through sweating is reduced, then internal body temperature can increase to levels where performance and health are compromised (Brotherhood,

2008). Defining such environmental conditions is the first step in the prevention of heat illness.

The Wet Bulb Globe Temperature (WBGT) is an empirical index of environmental heat stress that has been validated and given ISO (ISO/DIS 7933 1984) certification and, therefore, possibly represents the best current measure available, particularly during hot and humid days. The difficulty lies in defining the different cut-off values from which decisions can be made about the likely impact on player health. The American College of Sports Medicine (Sawka et al., 2007) recommends a WBGT cut-off for exercise of $>30.1^{\circ}\text{C}$ in non-acclimatized, unfit, and high-risk individuals, and $>32.3^{\circ}\text{C}$ in acclimatized, fit, and low-risk individuals. However, it has been proposed recently that marathon races in northern latitudes ($>40^{\circ}$ latitude) should be cancelled if the start WBGT is $>21^{\circ}\text{C}$ (Roberts, 2010). The wide difference in these recommendations comes from marathon running inducing a continuous heat production for several hours without interruption rather than other guidelines that have recommended reduced work–rest ratios before exercise cancellation. A recent study examining the energy cost of football (Osgnach et al., 2010) revealed that outfield players covered 2.8 km at a high intensity in Italian Serie A football matches. However, 45 min of laboratory-based soccer-specific intermittent exercise has been shown to induce the same physiological strain as steady-state exercise performed at the same average speed (Drust et al., 2000b). In the Italian Serie A outfield players, the average running speed was just over 7 km/h, which is considerably lower than the average speed of most marathon participants or the 12 km/h average speed used by Drust et al. (2000b). To date, there are no specific recommendations for playing football in hot environments. Therefore, in this interim period, based on the American College of Sports Medicine (Armstrong et al., 2007) recommendations, Table 2 is proposed as a guideline for acclimatized footballers playing in different environmental conditions.

Given that not all facilities have the equipment required to measure WBGT, Table 3 provides an indication of WBGT taken from shaded dry bulb temperature and humidity. This table is compiled from an approximate formula that relies on tempera-

ture and humidity (ACSM, 1984). However, the formula used in this table is valid only for full sunshine and a light wind, and it does not take into consideration the intensity of solar radiation or wind speed. This approximation may lead to incorrect estimates of thermal stress, particularly in cloudy and windy conditions, where the approximation is likely to overestimate the thermal stress. The approximation will also overestimate nighttime and early-morning conditions when the sun is low or below the horizon.

It is appreciated that there is a difference between “at risk environmental conditions” and “at risk individuals.” In addition, internal body temperature is influenced by metabolic heat production as well as by the environmental conditions. Therefore, environmental conditions alone do not predict physiological strain and consequently, as discussed below, accurate and non-invasive indicators of physiological strain are needed (Moran et al., 1998).

Individual measurements

Internal body temperature provides the best indicator of the thermal strain being experienced by individuals (González-Alonso et al., 1999). However, while internal body or core temperature is easily recorded in the laboratory and field experimental settings, it is difficult to obtain accurate measures during competitive play. Other physiological indices such as heart rate are not correlated to internal body temperature during intermittent exercise (Morante & Brotherhood, 2007). While exhaled temperature and infrared scans possibly provide interesting alternatives to direct measurements of internal body temperature, neither technique has been validated. Given that these measures require an interruption to play, there is interest in using telemetric systems that give recordings of intestinal temperature, but the costs of such systems do not make this solution practical beyond those participating at the top level. There are also limitations to this method as the thermistor must be at a point in the gastrointestinal tract at which food or fluid ingestion will not compromise accuracy (Casa et al., 2007). Therefore, the best initiative at the moment is to perform pre-participation screening before competing in the heat. This can be used to identify at-risk players to whom extra attention should be paid during competition, and should consist of determining pre-existing cardiac myopathies, arteriosclerotic vascular disease (Binkley et al., 2002), sickle cell trait (Connes et al., 2008), details of previous heat-related illness, and relevant family history.

Hydration

Players exposed to thermal stress must consider their hydration status and electrolyte balance before,

Table 2. Warning signs and symptoms of heat illness and injury

	WBGT ($^{\circ}\text{C}$)	Comments
Unrestricted	<22	Normal activity
Low risk	22–28	Monitor fluid intake
High risk	28–30	Observe signs and symptoms Watch at-risk individuals
Very high risk	30–32	Additional fluid breaks added Medical coverage mandatory
Stop play	>32	

Table 3. Estimated Wet Bulb Globe Temperature from Temperature and Humidity

		Temperature (°C)																														
		20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38	39	40	41	42	43	44	45	46	47	48	49	50
Relative Humidity (%)	0	15	16	16	17	18	18	19	19	20	20	21	22	22	23	23	24	24	25	25	26	27	27	28	28	29	29	30	31	31	32	32
	5	16	16	17	18	18	19	19	20	21	21	22	22	23	24	24	25	26	26	27	27	28	29	29	30	31	31	32	33	33	34	35
	10	16	17	17	18	19	19	20	21	21	22	23	23	24	25	25	26	27	27	28	29	30	30	31	32	32	33	34	35	36	36	37
	15	17	17	18	19	19	20	21	21	22	23	23	24	25	26	26	27	28	29	29	30	31	32	33	33	34	35	36	37	38	39	
	20	17	18	18	19	20	21	21	22	23	24	24	25	26	27	27	28	29	30	31	32	32	33	34	35	36	37	38	39			
	25	18	18	19	20	21	22	23	24	24	25	26	27	28	28	29	30	31	32	33	34	35	36	37	38	39						
	30	18	19	20	20	21	22	23	23	24	25	26	27	28	29	29	30	31	32	33	34	35	36	37	39							
	35	18	19	20	21	22	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38	39								
	40	19	20	21	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38	39									
	45	19	20	21	22	23	24	25	26	27	27	28	29	30	32	33	34	35	36	37	38											
	50	20	21	22	23	23	24	25	26	27	28	29	30	31	33	34	35	36	37	38												
	55	20	21	22	23	24	25	26	27	28	29	30	31	32	34	35	36	37	38													
	60	21	22	23	24	25	26	27	28	29	30	31	32	33	35	36	37	38														
	65	21	22	23	24	25	26	27	28	29	31	32	33	34	36	37	38															
	70	22	23	24	25	26	27	28	29	30	31	33	34	35	36	38	39															
	75	22	23	24	25	26	27	29	30	31	32	33	35	36	37	39																
80	23	24	25	26	27	28	29	30	32	33	34	36	37	38																		
85	23	24	25	26	28	29	30	31	32	34	35	37	38	39																		
90	24	25	26	27	28	29	31	32	33	35	36	37	39																			
95	24	25	26	27	29	30	31	33	34	35	37	38																				
100	24	26	27	28	29	31	32	33	35	36	38	39																				

≥ WBGT 40°C

Adapted from the Australian Bureau of Meteorology: http://www.bom.gov.au/info/thermal_stress

during and after exercise. Prior dehydration of about 1.5–2% of body mass can reduce performance even during short duration exercise (e.g. 1500 m track events; Armstrong et al, 1985) or after 30 min of repeated sprints (Maxwell et al., 2009). Sweat rates and sweat composition depend on the ambient temperature, humidity, and the exercise intensity, but they also vary greatly among individuals (Shirreffs et al., 2006). Some groups with pre-existing conditions, such as those with sickle cell trait, should be screened and pay greater attention to hydration during training and game play because they could be at a greater risk for exercise heat illnesses (Connes et al., 2008). A dehydration-induced loss in body mass of 1–2% can exacerbate thermal and cardiovascular strain (Hoffman et al, 1994), which impairs athletic performance (Cheuvront et al., 2003; Sawka & Noakes, 2007). Therefore, the most recent Position Stand from the American College of Sports Medicine (Sawka et al., 2007) suggested that fluid intake should be sufficient to restrict body mass loss to <2% of loss of the pre-exercise mass. Players should avoid drinking so much that there is a gain in body mass during exercise. This latter caution may, however, not hold true if an athlete begins exercise in a severely dehydrated state. No single recommendation is best for all individuals in every situation, and development of an individualized hydration strategy is essential for the protection of health and preservation of performance (Maughan & Shirreffs, 2008).

Developing a long history of standardized morning body weight measurements of players is important to obtain an indication of each player’s stable body mass. A fall in body mass of more than 2% may be indicative of hypohydration. Urine osmolality could possibly provide a reasonable alternative for identification of dehydrated players. A urine osmolality ≤ 700 mOsm/kg or a urine specific gravity of <1.020 g/mL can be

used as an index of euhydration (Sawka et al., 2007). It is strongly recommended that this index is used as a pre-participation requirement when the calculated WBGT (or the estimated value from Table 3) is >29.5 °C (i.e. high to very high risk). Urine color and frequency can be used when high precision is not needed (Armstrong, 2000).

Electrolyte balance should not be a concern for most players during a 90–120-min game. Salt balance can be maintained by habitually maintaining salt intake in the diet. However, medical staff and coaches should look for salt stains on players’ shirts to get an indication of those with a predisposition for high sodium losses and make adjustments accordingly in hydration and salt intake (Maughan, 2010; Shirreffs, 2010). Where unusually high sweat losses are anticipated, it may be important to identify and counsel any individuals who consciously restrict salt intake due to a family history of hypertension or for other reasons.

Pre-cooling/half-time heat removal

Cold drinks do not seem to influence the rate of internal body temperature rise but potentially sustain performance by lowering the core temperature (Lee et al., 2008; Siegel et al., 2009). Whole body pre-cooling has been observed to increase the time to fatigue (González-Alonso et al., 1999) or the work done (Vaile et al., 2008) during cycling in the heat by decreasing the initial temperature. When playing football in a hot environment (WBGT 32.1 °C), five minutes of leg cooling during halftime had a limited effect on specific skills but was effective in improving sprint performance in the second half (Yasumatsu et al., 2008). Pre-cooling does not affect the physio-

logical responses to a football-specific intermittent exercise when performed in temperate environmental conditions (Drust et al., 2000a).

Warm-Up

Performing an active warm-up is believed by athletes and coaches to improve performance and reduce the risk of injury (Shellock & Prentice, 1985). The active warm-up effects can be temperature dependent (i.e. decreased resistance of muscles and joints, greater off-loading of oxygen from hemoglobin and myoglobin, speeding of metabolic reactions, increased nerve conduction rate) as well as non-temperature dependent (i.e. increased blood flow to muscles, elevation of baseline oxygen consumption, resting cross-bridge breakdown, post-activation potentiation) (Bishop, 2003). It has also been hypothesized that warm-up may have a number of positive psychological effects (e.g. increased preparedness).

The difficulty with an active warm-up is to obtain the beneficial responses (e.g. increase in muscle temperature) without the associated negative consequences (e.g. substrate depletion, elevation of core temperature leading to early fatigue). Before sprinting or jumping in a temperate environment, the active warm-up intensity should be within 40% and 60% of the maximum oxygen consumption with a duration between 5 and 20 min and followed by a 5-min recovery (Sargeant & Dolan, 1987). Footballers generally perform longer and more intense active warm-up than this and there is no published guideline for a complex activity involving various skills such as football. However, it has been shown that performing a low-intensity exercise during halftime preserved muscle temperature and maintained sprint performance at the onset of the second half (Mohr et al., 2004).

When considering exercising in hot environments, performing an active warm-up failed to increase muscle maximal power (Bishop & Maxwell, 2009) but increased the core temperature and reduced the time to exhaustion at a fixed intensity (Gregson et al., 2002) and the ability to perform several repeated sprints (Bishop & Maxwell, 2009). From this perspective, active warm-up should be minimized before playing football in a hot environment to prevent a potentially detrimental increase in internal body temperature. We suggest that warm-up before playing football in hot environment should be limited to 10 min, including practice of specific skills.

Travelling to games to be played in hot environments

Many football players travel outside their country of residence to play games in a hot environment. This

requires them to acclimatize to the environmental conditions at their destination and often also to a new time zone. Near-complete exercise-heat acclimation occurs within 7–10 days of exposure to both hot-dry and hot-humid environments, with approximately 66–75% of the physiological adjustments and improvements in performance seen within 4–6 days (Nielsen, 1998; Pandolf, 1998; Voltaire et al., 2002). However, full-performance acclimation is impossible as even in tropical island natives, performance is impaired in hot and humid conditions relative to that in a temperate environment (Voltaire et al., 2003). We suggest a minimum of 1 week and an optimum of 2 weeks of acclimatization before playing in hot environment. However, it is of practical interest to note that exercising in a hot room while living in a temperate environment can increase resting plasma volume and reduce exercising core temperature and sodium sweat concentration (Kirby & Convertino, 1986), that may subsequently reduce the period required to acclimate to a hot environment.

Jet lag will occur when more than four time zones are crossed by air travel. Symptoms are due to an internal desynchronization of the different circadian rhythms as they adapt to the local time zone at different rates (Waterhouse et al., 2007). Adaptation requires approximately 1 day per time zone crossed and is easier when flying westwards than eastwards (Waterhouse et al., 2007). When travelling from west to east, we recommend promoting phase advance through training and light exposure in the morning. When travelling east to west, we recommend facilitating phase delay by exposure to bright light in the afternoon or evening (Waterhouse et al., 2007). Avoiding daytime naps and providing darkness facilitates nocturnal sleep.

Summary

Facts

- The capacity to exercise is markedly reduced in hot environments.
- High humidity imposes further physiological strain on the body.
- The combination of exercise and hot environments increases the risk of heat illness and heatstroke.

Recommendations for players

- Fluid intake should be sufficient to restrict body mass loss to <2% of the pre-exercise mass.
- A minimum of 1–2 weeks of acclimatization should be undertaken before playing in a hot environment.

Recommendations for organizers

- Perform pre-participation screenings before competing in the heat.
- Watch at-risk individual (e.g. sickle cell trait, vascular disease) and observe signs and symptoms if WBGT > 28 °C.
- Add additional fluid breaks and foresee medical coverage if WBGT > 30 °C.
- Reconsider event if WBGT > 32 °C.

Future research should investigate

- The optimal cooling strategies prior and during sporting activities, i.e., halftime in football games.

- The influence of acclimatization on football performance.
- The impact of heat on decision making in players and match officials.
- The impact of heat on spectators from cooler regions.

Key words: heat illness, soccer, exercise.

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