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The Effect of Heat Stress on Health and Performance

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Abstract

Objectives: Sport practitioners are required to prepare athletes for competition in hot and humid environments in the effort to preserve health during severe heat stress. For this reason special attention towards developing proactive approaches in the effort to negate symptoms of heat illness and mitigate decrements in performance is becoming a prevalent practice. As such these techniques and approaches warrant further examination in the effort to provide evidence-based recommendations for integrated support staff and athletes. This review discusses the physiological alterations recognized when exercising in the heat, its concomitant potential adverse health outcomes along with examining the effectiveness of popular pre cooling strategies for preserving athletic performance. **Health & Fitness Journal of Canada 2016;9(4):3-17.**

Keywords: Athlete Performance, Heat Stress, Athlete Health, Performance Enhancing

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Introduction

When exercising in a hot and humid environment it has been postulated that the propensity to exercise or to compete diminishes as a result of an increase in core temperature (Brück and Olschewski, 1987; Fink et al., 1975; Nielsen et al., 1990). Often major sporting competitions reside in hot and humid environments forcing athletes to maintain high levels of performance throughout multiple days of competition when experiencing heat stress. To date there is a considerable amount of literature that discusses the influence of hot ambient environments on intermittent (Almudehki et al., 2012; Girard et al., 2013) or continuous (Galloway and Maughan, 1997; González-Alonso et al., 1999b) athletic performance when performed in controlled laboratory settings. Current investigations examining performance and adverse effects in team sport during competition continue to demonstrate similar alterations and limitations in athlete performance (Nassis et al., 2015).

Match play during tournaments and events can often be scheduled according to television priorities and what is most convenient for worldwide spectators. As such athletes are often exposed to environments where experiencing hyperthermia or dehydration is almost unavoidable. These circumstances may be derived from competition occurring at periods of the day where the highest levels of heat are experienced. Consequently, athletes and practitioners must prepare for heat-related illness to help maintain health and performance.

The responsibility of both the athlete and sport practitioner to be well educated in effective hydration strategies prior to entering competition in a hot and humid environment should be a priority when planning future competitions. Understanding the level of adequate fluid to provide in advance and during

competition along with proper utilization of electrolyte pills and cooling techniques preferred should be practiced well in advance in an effort to ensure an effective response to potential signs and symptoms of heat illness.

This narrative review was developed to provide sport practitioners further understanding of the possible physiological limitations to performance, the signs and symptoms to be aware of during competition and how best to be proactive in mitigating the adverse effects of heat stress on athletic performance and health.

The Physiological Effect of Acute Heat Exposure on Athletic Performance

Limited research on soccer team performance in the heat during competition has demonstrated a reduction in total distance covered during match play (Mohr et al., 2012) and total distance covered at high intensity speeds (Özgünen et al., 2010); yet a preservation of top end sprint speed when comparing matches played in a hot and cold environment (Nassis et al., 2015). Research from Australian rules football players also indicates that team sport athletes tend to maintain top end speed yet suffer reductions in the total distance covered at this high velocity (Aughey et al., 2014). This disparity in professional athletic performance when competing in hot and cool environments may shed light on the physiological strain and its concomitant effect on performance where thermoregulation is challenged and hyperthermia and dehydration can develop during match play.

Team sports often involve repeated bouts of high intensity actions interspersed by lower intensity periods allowing for recovery before forthcoming repeating periods of high intensity activities (Gabbett, 2010). The high intensity activities recorded during match play may place a large demand on muscle glycogen storage throughout the match. Furthermore the amount of high intensity activities may be compromised during competition when experiencing heat stress. Previous literature has identified enhanced rates of glycogenolysis and increased blood lactate during exercise in the heat (Fink et al., 1975; Young et al., 1985). A number of theories have been proposed to account for these glycolytic changes and early fatigue when compared to performance in a cool environment. Changes in plasma catecholamines may be partially responsible for alterations in muscle substrate utilization during exercise in heat (King et al., 1985; Kirwan et al., 1987). The response of the adrenal medulla during exercise has revealed to be upregulated with an elevated core temperature possibly explaining the increased presence of plasma catecholamines (Galbo et al., 1979). Hyperglycemia is a common occurrence when exercising in the heat and may be a possible side effect of an enhanced catecholamine response and its resulting effect of increased liver glucose output (Hargreaves et al., 1996). Febbraio and colleagues (1994) demonstrated an enhanced reliance on muscle glycogen and increased catecholamine levels (epinephrine and norepinephrine) during exercise in the heat (Febbraio et al., 1994). They also observed a reduction in glycogen levels in Type 1 muscle fibre with minimal changes in Type II fibres (Febbraio et al., 1994).

Although marked increases in blood glucose and muscular glucose oxidation rates have been observed, muscle glycogen depletion is thought not to be

the main cause of fatigue when exercising in the heat as storage levels after exercise remain relatively unaffected (Parkin et al., 1999). A potential explanation for the conflicting evidence regarding muscle glycogen depletion and its effect on exercise performance when experiencing heat stress may be the various methodologies in previous investigations. Single bouts of exercise lasting 10 to 30 min may lack the intensity and duration required to compromise muscle glycogen stores. Short bouts of exercise may not provide as great of a stimulus as seen during 70-90 min intermittent team sport events (Jentjens and Jeukendrup, 2003). Furthermore, potential issues may arise regarding muscle recovery and the re-synthesis of glycogen during repeated competition as seen within tournament schedules. Further research investigating muscle glycogen depletion using repeated bouts of exercise in the heat that mimic the demands of intermittent sport are warranted in an effort to provide greater insight for practitioners and team dietitians when developing proper nutritional and exercise recovery strategies.

The development of a critical level of core temperature (expressed as 40^{o}C) has often related to impaired performance in the heat (González-Alonso et al., 1999b; Nielsen et al., 1993; Nybo and Nielsen, 2001a, 2001b). At this critical temperature, a series of responses occur to help negate excessive hyperthermia induced through exercise (Nielsen and Nybo, 2003; Nybo, 2007). The development of severe hyperthermia (≥40^{o}C) may have an effect on the central nervous system (CNS) and its ability to recruit motor neurons for both health and performance (Hargreaves, 2008). Heat production is attenuated with lowered levels of metabolism (as observed when approaching this critical core temperature during exercise), which may be due to a protective negative feedback response as expressed through reductions in motor drive (Hargreaves, 2008). Evidence for exercise-induced hyperthermia inhibiting sustained maximum muscle force production has been attributed to the CNS and temperature-related contractile properties of the muscle (Todd et al., 2005). However, only prolonged muscle contractions demonstrated a diminished force production when compared to brief contractions that remained unaffected (Todd et al., 2005).

Limited investigations examining muscle fatigue due to heat stress have suggested that hyperthermia significantly affects sustained voluntary muscle contractions (Todd et al., 2005). Reductions in cycling performance in the heat (when core temperature was equal to that recorded in a cool environment) showed reduced power outputs during self-paced exercise (Kay et al., 2001). It is possible that the development of heat storage is regulated through afferent neural input from thermoreceptors of the blood and skin helping regulate metabolic rate in an effort to attenuate exaggerated heat production when exercising in hot environments (Tucker et al., 2006). The development of hyperprolactinemia, a physiological state identified by elevated levels of prolactin, can be observed during exercise in the heat and is thought to be an indirect indication of the possible involvement of CNS serotonergic activity and its effect on exercise fatigue in the heat (Pitsiladis et al., 2002). Further literature examining CNS limitations during exercise in the heat have demonstrated improved exercise

performance when noradrenaline and dopamine re-uptake inhibitors were administered beforehand (Watson et al., 2005). Additional research directed towards examining the effects of hyperthermia on CNS performance during exercise and its subsequent alterations in perceptions of fatigue can help shed light on a potentially large piece of the heat-induced fatigue model during athletic performance.

When performing in the heat the physiological demands of exercise drive blood flow to multiple locations of the human body such as skeletal muscles to maintain energy production, the cutaneous layer for thermoregulation, and to the CNS for optimal functioning (Cheuvront et al., 2010). With a continued rise in core temperature as a result of metabolic heat production, a high level of hyperthermia may arise, resulting in a substantial redirection of blood flow to the cutaneous layer for thermoregulatory purposes. This large redistribution in blood flow elicits a reduction in oxygen delivery to working muscles and CNS (González-Alonso et al., 2004; Nybo and Nielsen, 2001a; Rasmussen et al., 2004). As such, when an athlete is determined and highly motivated during maximal exercise, efferent neural activity relaying heat sensory information to central command can be neglected. When this state is achieved the body has demonstrated to succumb to the exercising muscle demands whereby maintaining blood pressure and muscle blood flow at the expense of the cutaneous layer (González-Alonso et al., 2008). Empirical evidence has demonstrated a linear relationship between core temperature and skin blood flow; augmented levels of cutaneous blood flow permit enhanced heat dissipation through convection; thus promoting a reduction in core temperature as whole body circulation is maintained (Ely et al., 2009; González-Alonso et al., 1999b; Maron et al., 1977; Pandolf and Goldman, 1977). The redistribution of blood flow to the cutaneous layer and its resulting effect on exercise performance was further investigated by Sawka and colleagues (2001) who observed elevated skin temperatures due to clothing or extreme heat leading to exercise termination or the collapsing of participants before core temperatures climbed above 38.5^{o}C (Sawka et al., 2001). These findings provide further evidence that redistribution of blood flow to the skin during mild exercise elicits a large cardiovascular strain that can diminish oxygen delivery to the working muscles whereby increasing the potential for premature fatigue before severe hyperthermia is observed. The main contributing factor to diminished aerobic performance (VO_2max) in the heat is thought to be the result of the redistribution of blood flow to the skin and the consequential reduction in both peripheral supply, diastolic preload, stroke volume and the resulting reduction in cardiac output (Arngrímsson et al., 2003; González-Alonso and Calbet, 2003; MAcDougall et al., 1974; Sawka et al., 1985; Wingo and Cureton, 2006). The fundamental limitations to aerobic performance in the heat are believed to be a result of a decrease in VO_{2max} and cardiac output, each being a consequence of a reduction in the cardiovascular reserve (Bassett and Howley, 2000; Di Prampero, 2003). A continuous reduction in VO_2max can be observed with elevated ambient temperatures that elicit large increases in skin temperature with

minimal changes in core temperature, this physiological response further supports the correlation between aerobic performance and the redistribution of muscle blood flow to the cutaneous layer for thermoregulatory purposes (Arngrímsson et al., 2003).

It has been observed that a reduction of >2% body mass due to dehydration can diminish aerobic performance and VO_2max (Sawka, 1992; Sawka et al., 2007; Sawka and Noakes, 2007). This reduction in total body water includes the vascular compartment, specifically plasma volume; such a loss in plasma has a concomitant effect on limiting diastolic filling and stroke volume (Gonzalez-Alonso et al., 1995; Gonzalez-Alonso et al., 2000; González-Alonso et al., 1999a). Reductions in the vascular volume have demonstrated to limit skeletal muscle blood flow and alter muscle metabolism during exercise (Febbraio, 2000; González-Alonso et al., 1998). This change in metabolism may be a consequence of greater reliance towards carbohydrate utilization in the working tissue as a result of an altered anaerobic threshold for a given speed or power output. Together both dehydration, as represented as plasma volume loss, and hyperthermia have been observed to account for at least 50% of the reduction in cardiac performance, anaerobic threshold and VO_{2max} (Gonzalez-Alonso, 1998). Competition for blood flow between the cardiovascular system to support aerobic performance and the cutaneous layer for supporting thermoregulation during exercise in the heat demonstrates a balanced relationship which regulates both desired performance and overall health. Developing an understanding of the relationship between a rising core temperature and the resulting redirection of blood flow to the cutaneous may allow practitioners to develop strategies to help preserve muscle blood flow prior to competing in the heat in the effort to sustain optimal performance. Strategies to enhance both the pre and intra competition hydration levels may also help favour performance whereby possibly preventing or delaying the body's decision to compromise performance for the maintenance of health (Hamilton et al., 1991). Practitioners must recognize that extreme environmental conditions that elicit rapid elevations in skin temperature or whereby heat dissipation is dramatically attenuated due to increased ambient levels of vapour pressure may produce reductions in performance and health that may be dramatically compromised before core temperature rise to significant levels. For this reason when monitoring core temperature during competition and training it is recommended to examine additional risk factors such as relative humidity and heat stress (WBGT) in combination with monitoring core temperature.

Potential Adverse Health Outcomes as a Result of Exercising in Hot Environments

The development of heat illness can be categorized by the monitoring of two risk factors; external factors such as type of clothing and equipment, ambient temperature and relative humidity, and internal factors such as medical conditions of an athlete, drug use, and dehydration or sunburn (Barrow and Clark, 1998; Howe and Boden, 2007). Dehydration resulting from poor hydration prior to and during competition have shown to be a decisive factor in the

onset of heat illness (Howe and Boden, 2007). Integrative support staff often follow a linear continuum in the development of heat illness which evolves from a mild to a more serious life threatening situation. Heat edema is recognized as the mildest form of heat illness; it can proceed to heat rash, heat syncope, heat exhaustion and eventually the most life threatening situation called heat stroke (Howe and Boden, 2007). Practitioners must recognize that the development of a mild form of heat illness does not necessarily elicit the development of a more serious form of heat illness if the athlete is left untreated (Howe and Boden, 2007). Perhaps the most important aspect in monitoring athletes competing in the heat is the proper diagnosis of symptoms being experienced by the athlete, this can allow for proper procedures and protocols to be followed in order to maintain the safety and health of the individual.

Heat edema can occur in the presence of a normal core temperature and can generally lack significant signs and symptoms (Coris et al., 2004; Howe and Boden, 2007). Swelling of the interstitial compartment due to increased fluid buildup as a consequence of enhanced peripheral vasodilation for thermoregulatory mechanism often results in heat edema (Coris et al., 2004; Howe and Boden, 2007). This enhanced vascular volume can lead to increased hydrostatic forces causing increased capillary filtration rates and a resulting fluid buildup in the interstitial compartment (Haskell et al., 1997; Howe and Boden, 2007). Elevation of the limbs accompanied with compression garments and proper rehydration of fluids and electrolytes may help improve immediate symptoms (Coris et al., 2004; Howe and Boden, 2007). Heat rash is another form of heat illness that has been observed in athletes with a normal core temperature (Coris et al., 2004; Howe and Boden, 2007). Onset of this form of heat illness is typically from the blockage of eccrine sweat glands from athletic equipment or clothing that results in the leakage from the sweat gland into the dermis layer often developing a prickling sensation (Habib, 2004). Successful treatment for reducing symptoms of heat rash can include immediate cooling of the affected area, reducing any clothing around the rash, and the use of a mild topical corticosteroid anti-inflammatory (Habib, 2004). Heat syncope is the third form of heat illness that can occur in the presence of a normal core temperature and is often developed from extreme exertion or quick postural changes during enhanced venous pooling and peripheral vasodilation (Seto et al., 2005). A sudden loss of balance or coordination during a momentary transition period during competition may lead to injury if an athlete is unable to support themselves or lose control of their movement (Lugo-Amador et al., 2004). Typical treatment for heat syncope involves the immediate transition to a supine position and the elevation of peripheral limbs in an effort to stimulate venous return and the gradual loss of syncope (Chen et al., 2006; Seto et al., 2005).

Heat cramps are typically experienced in large muscles during prolonged activity (>2hrs) and are often observed during normal to <40^{o}C core temperatures (Glazer, 2005; Wexler, 2002). Classic symptoms distinguishing heat cramps from exertional cramps is the often wide spread of affected musculature and its influence to cause similar symptoms to adjacent muscles that have

yet to experience any symptoms (Bergeron, 2007). This type of heat illness is often observed with individuals who possess large Na^+ concentrations in their sweat output, possible leading to exaggerated extra and intra cellular water and Na^+ loss (Stofan et al., 2005). A potential consequence of Na^+ reduction in the interstitial compartment may lead to nerve axon terminals of nearby motor neurons becoming mechanically deformed with a concomitant increase in surrounding neurotransmitters and ions concentration (Layzer, 1994). As a result, a portion of the developed hyper excitable axon terminals can discharge spontaneously eliciting new action potentials in the affected musculature (Layzer, 1994). Often the progression of heat cramps from being hardly noticeable by the athlete to the state of unbearable pain can take place within 20 to 30 minutes after the first mild twitch is felt (Bergeron, 2007). A combination of Na^+ (1150mg) with water or a sports drink can be an effective response for controlling muscle cramps in athletes who demonstrate a high sweat Na^+ concentration (Bergeron, 2007). Individuals who experience reoccurring heat cramps are often advised to ingest large Na^+ concentrations prior to or during competition (Bergeron, 2003), however special attention to potential gastro interstitial disturbance and bloating is advised as these are common side effects when consuming such large amounts of Na^+ and may develop into an independent factor for the cessation of exercise (Zietsman et al., 1989). Massage, static stretching or the cooling of affected muscles can also help reduce immediate discomfort and spasms (Seto et al., 2005).

Heat exhaustion has shown to be the most common form of heat illness in athletes (Armstrong and Maresh, 1993). This stage of heat illness is accompanied with a rise in core temperature ≤40.5^{o}C and prevents the athlete from further competing in competition (Armstrong and Maresh, 1993; Barrow and Clark, 1998). The onset of heat exhaustion can be sudden and quick, potentially developing before major signs or symptoms are recognized (Armstrong et al., 1996). The inability of the cardiovascular system to simultaneously meet the exercising metabolic and thermoregulatory demands when experiencing severe dehydration is thought to be responsible for the development of heat exhaustion (Lee-Chiong Jr and Stitt, 1995; Wexler, 2002). Symptoms that typically accompany this stage of heat illness are nausea, intense sweating, muscle weakness, chills, vomiting and vertigo (Lee-Chiong Jr and Stitt, 1995; Wexler, 2002). Alterations in CNS functioning can involve dizziness, disparity and the onset of a strong headache (Howe and Boden, 2007).When an athlete is conscious and vomiting and diarrhea is not present, immediate removal from the heat and rapid cooling using water immersion, along with oral consumption of fluids are appropriate for reducing core temperature (Armstrong et al., 1996). When an athlete is unable to safely consume fluids utilization of intravenous fluids ranging from 5% dextran in 0.45% to 0.9% saline may help facilitate rehydration and reduce hyperthermia especially if oral fluid consumption is problamatic (Coris et al., 2004; Howe and Boden, 2007).

Heat stroke is the most severe form of heat illness and is usually accompanied with elevated core temperatures that lead to the destruction of cellular tissues and impaired organ function (Bouchama and

Knochel, 2002). Heat stroke can be observed with core temperatures above 40.5°C in the absence of sweating and with large impairment in mental status (Coris et al., 2004; Howe and Boden, 2007). Tissue injury, cardiac arrest, organ failure and mortality have been observed to be closely correlated to the length of time that core temperature has been elevated before cooling begins (Armstrong et al., 1989). Current literature suggests that enhanced survival rates are seen when core temperature is reduced below 38.5°C within 30 minutes (Dematte et al., 1998; Lugo-Amador et al., 2004) to 60 minutes (Lugo-Amador et al., 2004; Vicario et al., 1986) and that the use of ice water immersion is most effective in reducing core temperature during this period (Smith, 2005). A time difference between 30 and 60 minutes may be significant when experiencing such severe physiological impairment. Enhanced alterations in CNS functioning at this stage can be observed and typically involve ataxia, confusion, increased irritability and potentially the development of a coma state (Howe and Boden, 2007). Further research investigating the immediate response is warranted in the effort to provide medical practitioners effective and efficient protocols for both on field and in house occurrences of heat stroke.

Strategies for Optimizing Athletic Performance in the Heat and Mitigating its Adverse Effects

The responsibility of both the athlete and sport practitioner to be well educated in effective hydration strategies prior to entering competition in a hot and humid environment should be a priority when planning future competitions. Understanding the level of adequate fluid to provide in advance and during competition along with proper utilization of electrolyte pills and any cooling techniques preferred should be practiced well in advance in an effort to ensure an effective response to potential signs and symptoms of heat illness.

Adequate ingestion of fluids prior to the start of competition have demonstrated to be an effective method for the prevention of cardiovascular and thermoregulatory impairment (Hamilton et al., 1991). The immediate consumption of fluid during competition is typically used to restore plasma volume, the resulting improvement in cardiovascular and thermoregulatory benefits has been observed to be proportional to the amount of fluid ingested by the athlete in the absence of excessive intake beyond that of their sweat rate (Montain and Coyle, 1992). Ingestion of electrolyte sport drinks containing carbohydrate and adequate Na^+ levels 60 minutes prior to competition where the duration is greater than 60 minutes has shown to be advantageous during match play (Coyle, 2004; Montain and Coyle, 1993). Empirical evidence suggests that consumption of fluid ad libitum can maintain or improve aerobic performance during heat stress and that the drive to consume this amount should be developed through the proper education of the athlete and the immediate environmental conditions experienced during competition (Noakes, 2007).

Utilization of external precooling strategies defined as medium, cold air or fluid exposure to reduce core temperatures in the effort to optimize performance have shown to be successful in athletes during major competition in the heat (Grantham et al., 2010). Cold air exposure ranging from 0° to 5°C for

periods of 15 minutes have demonstrated a reduction in both skin and core temperatures (Hessemer et al., 1984; Olschewski and Bruck, 1988). This transient exposure to extreme cold is thought to initiate vasoconstriction and a reduction in heat transfer from the core to the periphery via conduction (Charkoudian, 2010). This rapid shunting of blood from the periphery can promote larger temperature gradients between core and shell prior to or during exercise allowing for greater convective heat transfer at the cutaneous layer (Hessemer et al., 1984; Ross et al., 2013). Enhanced thermal comfort and aerobic performance in cycling and running have been observed with cold air exposure, however research regarding its effectiveness within intermittent team sport remains limited (Olschewski and Bruck, 1988; Ross et al., 2013). Although this technique has demonstrated promising results for athletes competing in individual sporting events, the practicality of a chamber able to accommodate multiple athletes simultaneously may not be possible for most team sports.

Cold tubs have been viewed as common pieces of recovery equipment in most intermittent team sport environments and may provide an alternative option to cold air chambers. Heat loss through water has demonstrated to be two to four times greater than through the use of cold air, suggesting enhanced effectiveness and practicality when working with multiple athletes who require exposure immediately prior to or during competition (Molnar, 1946; Smith and Hanna, 1975). Literature examining cold water immersion (17^{o}C) for performance enhancement have shown improvements up to 2.7% in power output and a 4% increase in aerobic power demonstrated in running time trials (Booth et al., 1997; Marsh and Sleivert, 1999). Previous investigations examining repeated sprint ability has shown to be promising as reductions in rate of decay in speed have been observed when cold water immersion is implemented between bouts of sprinting (Vaile et al., 2008). Current literature suggests cold-water immersion is a practical and efficient technique for cooling core temperature in intermittent team sport and may be an effective method to limit performance decrements when experiencing heat stress.

Another practical option for team sport athletes may be the usage of cold garments. Cold garments are easily transported to competition locations and allow for easy access within a change room or a team bench setting. Ice vests containing either gel or ice are a popular cold garment option and have demonstrated to successfully reduce core temperature during competition (Arngrïmsson et al., 2004; Smith et al., 1997). Ice vests can be utilized during individual sporting events or prior to competition and during half time during intermittent team sport. Often financial restraints commonly seen within team sport can limit sport science equipment, this outcome is typically the consequence of having to simultaneously cool multiple athletes at once and the demand for equipment can become very costly. A potential advantage for practitioners working within team sport is the opportunity to use garments that are already possessed by athletes such as sweaters, long sleeve t-shirt or fleece jackets and cooling them using water. Cold garment utilization during a 15 minute warm up period has demonstrated reductions in skin

temperature and an overall improvement in thermal comfort of the athlete (Arngrïmsson et al., 2004). Current literature supports the use of cold garments as being effective at reducing core temperature during warm up or physical activity when core temperature is elevated (Bogerd et al., 2010). To date the majority of research supports this form of temperature regulation as improved aerobic performance when running or cycling has been commonly observed when implemented (Arngrïmsson et al., 2004; Smith et al., 1997). Limited research identifying enhanced aerobic performance for use in intermittent sport suggests future research is necessary before implementing in a team sport setting.

External cooling techniques that can reduce internal core temperature have shown promising results for improving aerobic performance(Arngrïmsson et al., 2004; Smith et al., 1997). However the potential for decreased muscular performance due to exposure of a cold garment or water immersion may elicit negative outcomes. Therefore, internal cooling methods may be more advantageous through minimizing decrements in muscular performance (Ihsan et al., 2010). Ice slurries made from liquid and crushed ice is a convenient and affordable method for initiating reductions in core temperature when working with multiple athletes who require cooling simultaneously. Siegel et al. were able to demonstrate improved running speeds at aerobic threshold using ice slurries with a concomitant reduction in core temperature of 0.41^{o}C (Siegel et al., 2010). Current literature suggests any potential benefits associated with the ingestion of cold beverages or ice slurries are dependent on the temperature of the beverage rather than the amount consumed (Ross et al., 2013). A beverage temperature of 4^{o}C has shown to be optimal for reducing both core (-0.5^{o}C) and skin temperature (-0.7^{o}C) while aiding cycling performance in the heat (Burdon et al., 2010). Lee and company were able to further demonstrate enhanced performance with the use of a 4^{o}C beverage as compared with a beverage at room temperature or 10^{o}C (Lee et al., 2008). The option for developing a customized cold beverage with a range of flavours and electrolyte balances may be a promising option for sport practitioners when managing multiple athletes requiring simultaneous cooling. Sport practitioners must continue to experiment with cooling techniques to help identify effective and efficient cooling strategies that are both evidence based and practical for their athlete population.

Conclusions & Practical Application

Current evidence suggests that a gradual rise in core temperature to a critical level (≥40^{o}C) may elicit a reduction in motor unit recruitment in the effort to reduce metabolic heat and negate further rises in temperature and cellular necrosis. Additionally the redistribution of blood flow to the cutaneous layer in the presence of an elevated core temperature or due to rapid increases in skin temperature can elicit cardiovascular strain whereby causing cessation of exercise.

It is recommended that practitioners and athletes identify common heat illnesses frequently observed in their given sport. Recent evidence suggests the signs and symptoms of each level of heat stress may be influenced through clothing, sporting environmental

conditions, and the availability of rehydration/nutritional intake. As such, it may be advantageous to consume ice slurries when possible and to utilize authorized pre cooling garments during competition or intermission.

Competing in the heat is often an unavoidably circumstance for most athletes. Consequently it is recommended that athletes and practitioners develop a thorough understanding of the physiological effects when competing in the heat along with effective, proactive techniques and health response actions to maintain performance and health.

Authors' Qualifications

The author's qualifications are as follows: Andrew S. Perrotta PhD (ABD), MKin, BSc (HON), CEP, CSCS, USSF "C"; Nicholas J. Held MHK, BSc (HON), CEP, CSCS; Darren E.R. Warburton PhD, MSc, HFFC-CEP.

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