Heat Illness and Injury

An estimated 900,000 high school athletes are treated for heat illness per year (Kerr 2013). Athletes participating in football were 10 times more likely to experience heat illness. An estimated 9,237 athletes experience time-loss due to heat illness (Yard 2009). Individuals who exercise in environments that are hot, humid, minimal wind, and high wet bulb are at risk to develop a heat illness (Bergeron 2012). At risk populations are elderly, young children, overweight, untrained, taking certain medications, un-acclimated, high exertion level, or improperly hydrated (Brooks 2005, Hitchcock 2007, Amit 2011). Heat loss is limited in hot humid enviorments and increase heat stress in certain individuals with high cost of oxygen consumption during exercise leading to dehydration and heat stroke (Arsac 2005).

Figure 1: Heat Index

Heat Index uses air temperature and humidity for apparent tempature.

http://www.erh.noaa.gov/rah/heat/

Heat Stroke

The physiological change include endotoxaemia from heat stroke leads to systemic inflammation due to gastrointestinal and immune changes. Gastrointestinal changes are due to the lack of blood flow which is decreased by 80% during exercise. Heat stroke causes a decreased amount of blood flow to the gut leading to hypoxia and ischemia. Immune changes include suppression of immune cells functions, suppression of cell-mediated immunity, translocation of lipopolysaccharide, suppression of anti-lipopolysaccharide antibodies, increased macrophage activity because of damaged muscle, and increased concentration of circulating inflammatory and pyrogenic cytokines. Increased lipopolysaccharides increase risk of sepsis. Natural protection of cells that experience heat stress are heat shock proteins (Chin Leong 2006, Iguchi 2012, Kourtis 2012). When heat stroke occurs heat shock protein benefits are diminished. Heat shock proteins help prevent protein aggregation, boost the immune system, and help transport repair proteins. When norepinephrine is increased due to heat stress prolactin levels and heat shock proteins increase to help combat the negative effects of heat stress (Iguchi 2012). In animal based studies the heat shock protein HSP-16.1 in the Golgi body of a cell helps maintain homeostasis by \( \text{Ca}^{2+} \) and \( \text{Mn}^{2+} \) transportation of...
ATPase and PMR-1 pump to maintain Ca\(^{2+}\) regulation under stress. Other heat shock proteins that help prevent heat stroke include HSP-16.1, HSP-16.41, and DNJ-19. In animals precondition for 30 minutes at 34\(^{\circ}\)C helps limit heat stroke (Kourtis 2012).

Management and treatment of heat stroke include cooling the individual back to a rectal temperature of 28.6\(^{\circ}\)C. Cold water immersion 2\(^{\circ}\)C water for 9 minutes is the idea treatment of an individual with heat stroke. During cold water immersion an individual’s temperature is monitored until it returns to normal, without monitoring and individual can go from hyperthermic to hypothermic (Gagnon 2010). Other methods of cooling include fanning, ice packs placed on major veins and arteries, IV may be done by EMS to cool internally (Brooks 2005, Arsac 2013, Amit 2011).

**Dehydration**

Dehydration, another factor of heat illness, is the loss of fluid from the body. Symptoms include decreased sweat rate, plasma volume, cardiac output, maximal oxygen uptake, work capacity, muscle strength and liver glycogen (Casa 2010, Bardis 2013, Hiderori 2013). Water loss of 5% of body weight is common in exercise and must be replaced or may cause injury. Water loss of 7-10% of body weight symptoms are difficulty salivation and dis-coordination. 15-20% loss of water per body weight symptoms are delirium, dried skin, decreased urine volume, bleeding of the skin. Any greater loss than 20% leads to death (Brooks 2005).

The physiological changes that occur in dehydration is the decrease of blood osmolality, specific gravity of urine increase due to loss of osmolality, sweat increases to cool the body initially and will decrease when they body experiences dehydration (Brooks 2005, Hitchcock 2007, Hindenori 2013). Core temperature increased in a study by Casa, et. al. to 39.49±0.37\(^{\circ}\)C post exercise. When rehydrated temperature decreased to 39.18±0.47\(^{\circ}\)C (Casa 2010). One study found that in a dehydrated state cycling increased core temp, lowered the sweat rate, decrease sweat sensitivity, and increased perceived exertion (Bardis 2013).

Treatment of dehydration includes replacing fluids. Fluid replacement has been suggested to use a carbohydrate sports drink that includes electrolytes (Amit 2011). In extreme cases intravenously saline is given to re-hydrate the body (Brooks 5).

**Heat illness prevalence, performance effects, and prevention**

Prevalence—Heat illnesses such as heat stroke and dehydration can occur to any individual who is exercising in hot and humid conditions. Sports such as football which practice in the heat and with equipment tend to experience more dehydration and heat illness (Brooks 5, Hitchcock 2013, Yard 2013). Athletes with a history of concussion have a higher risk for heat stroke (Alosco 2012) Individuals such as distance runners, soccer players, or tennis players who do not hydrate properly also are at risk.

Performance effects decreases with heat stroke and dehydration and can lead to diminished performance, incomplection of event, long term injury, and death (Hillman 2011). Exercise in hot humid enviroments increase oxidative stress put on the body when the body in a dehydrated or hyperthermic state (Cappaert 2008). In football due to the equipment worn in the sport decrease evaporation, which causes greater cardiovascular strain and metabolic cost due to increased heat (Hitchcock 2007). Even mild dehydration can decrease performance due to the increased heat strain and perceived intensity (Bardis 2013).

**Graph 1: Exercise-induced Dehydration and Environmental Heat Stress**
Exercise-induced dehydration with and without environmental heat stress results in increased oxidative stress (Hillman 2013).

Prevention of heat illness includes pre-cooling, acclimatization, hydration, and types of clothing. Pre-cooling is the idea that cooling prior to exercise may lower the body temp allowing for longer exercise in heat. Some studies have shown that cooling garments such as ice vests do not work effectively in cooling the core temperature. Cold water immersion has shown to be the best method of pre-cooling. An alternative to cold water immersion is an ice slurry that lowers core temperature thorough ingestion. Another method of prevention is acclimatization, the process of adjusting to a climate, this normally takes from 1-2 weeks (Lopez 2008, Jones 2012, Siegel 2012, Hausswirth 2012, Chin Leong 2009). Acclimation allows for a decreased blood prolactin levels prior to exercise. Short term increase in training load for endurance athletes may help decrease the chance of heat illness via decreasing plasma lipoposaccaride concentration at rest and 1.5 hours post exercise. Decreased plasma lipoposaccarides reduces the inflammatory response due to exercise and heat stress (Burk 2012). One theory to heat illness prevention is wearing athletic clothing made out of polyester and other synthetic materials. Contrary to company claims moisture wicking synthetic material increases core tempature (Klicklighter 2011) The makeup of synthetic clothing designed for heat or cold weather differs only slightly, in some cases 2%.

**Cold Injuries**

Cold injuries occur with low temperatures, wind chill, prolonged exercise, and improper clothing (Cupp Pett 2012). Individuals at risk for cold injuries, such as hypothermia and frostbite, occur when an individual is exposed to temperatures typically below 0°C, wind chill, water tempature (Bergeron 2012, Cappaert 2008). Individuals at risk for cold injuries have the following characteristics: lean body mass, female, illn ess, exposed skin, and improper clothing (Cupp Pett 2012, Tloughan 2011)
Hypothermia

Hypothermia is the depression of the central nervous system resulting in sleep, inability to shiver, and eventually a coma. Hypothermia occurs in exercise the rate of heat production exceeds the heat of heat loss (Brooks 2005, Cuppett 2012). Hypothermia may be classified in four categories in which the core temperature is below 35°C. Mild is classified as a core temp of 35-37°C, moderate 32-34°C, and severe below 32°C (Cappaert 2008).

The physiological changes of hypothermia includes glycogen depletion from the body due to the body trying to keep itself warm. Depletion of glycogen can lead to hypoglycemia and reduced CNS function (Cappaert 2008, Cuppett 2012). Hypoglycemia decreases shivering which increases hypothermia (Castellani 2006).

Treatment of hypothermia is to remove all wet clothing, passively warm via adding clothing or blankets, room temp 21-24°C, and if hospitalized intravenous fluid (Dhar 2000).

Figure 4: Signs and Symptoms of Hypothermia and Frostbite

(Cappaert 2008)
Frostbite

Frostbite is the result of vasoconstriction in response to cold that results frozen tissue, tends to be the extremities. Long term injury may result in circulator damage or gangrene and result in loss of extremity. Risk factors temperature, wind chill, fatigue, circulatory impairment, improper clothing, high elevation,
alcohol consumption, and tobacco use (Cuppert 2012, Brooks 2005). Frost bite occurs at temperatures from 0°C to -35°C and below (Bergeron 2012). There are four grades of frostbite.

First degree (frost nip) symptoms swelling, numbness, pain (Cuppert 2012). Notice the image below how the right hand is swollen compared to the left.

**Figure 5: First Degree Frostbite**

![First Degree Frostbite](http://www.oilspillsolutions.org/safety.htm)

Second degree symptoms swelling, redness, blister formation (Cuppert 2012).

**Figure 6: Second Degree Frostbite**

https://www.youtube.com/watch?v=E6Jv9vDk8t8

http://www.oilspillsolutions.org/safety.htm

Third degree symptoms full thickness destruction of skin, blisters and hardened wax like skin (Cuppett 2012).

Figure 7: Third Degree Frostbite
Fourth degree symptoms are the loss of entire extremity (bone, muscle, tendon) redness, edema, ashy skin, numbness tingling, burning (Cuppett 2012).

Figure 8: Fourth Degree Frostbite
Physiological changes of frostbite includes vasoconstriction to bring the body’s blood supply to the trunk (Brooks 2005, Cuppett 2012). Vasoconstriction occurs when the skin’s temperature is less than 35°C and
31°C in water (Castellani 2006). The limited blood flow to extremities allows for the cells to freeze. Cellular damage is due to electrolyte and water concentration freezing (Tloughan 2011).

The treatment for frostbite is to remove the individual from cold exposure, passively rewarm tissue, rehydration, and restore circulation (Cuppett 2012).

**Cold injury prevalence, performance effects, and prevention**

Prevalence of hypothermia and frostbite occur mainly in outdoor winter sports, occasionally hypothermia and frostbite are seen in outdoor fall sports like soccer, football, cross country, and lacrosse. 3% to 5% of all injuries in mountaineers and 20% of all injuries in Nordic skiers reported were due to hypothermia or frostbite (Sallis 1999). The CDC found on average 689 deaths per year are due to hypothermia (CDC 2013). One study found in 2005 83% of mountaineers got 1st degree frostbite, hands (26.4%) and feet (24.1%) involvement were most common (Harirchi 2005).

Hypothermia and frostbite are serious cold injuries, they can prevent individuals from competing, loss of extremities, and death. Hypothermic conditions slows recovery time of potential contractile properties of muscles (Drinkwater 2007).

Prevention of cold injuries such as hypothermia and frost bite include different types of clothing and acclimatization. Clothing that keeps the individual warm and acts as a wind breaker are suggested. One study in 2012 by Burtscher et al. found that walking 1 hour at 0°C and a wind speed of 10 the more clothed an individual is the greater prevention of hypothermia there is. Moderate exercise in cold environments helps individuals feel warmer and relieves pain after 90 minutes of immobility briefly (Muller 2011). Cold induced vasodilatation is a hypothesized protective mechanism. Cold induced vasodilatation occurs in the toes during exercise and increases core temp (Dobnikar 2009).

**Figure 9: Clothing Combinations for Insulation**

<table>
<thead>
<tr>
<th>Clothing Combination</th>
<th>Clothing Insulation Factor (clo)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Shirt, lightweight trousers, socks, shoes, underwear briefs</td>
<td>0.6</td>
</tr>
<tr>
<td>Shirt, trousers, jacket, socks, shoes, underwear briefs</td>
<td>1</td>
</tr>
<tr>
<td>Wind-proof, waterproof jogging suit (jacket and pants), T-shirt, briefs, running shorts, athletic socks, athletic shoes</td>
<td>1.03</td>
</tr>
<tr>
<td>Fleece long-sleeved shirt, fleece pants, briefs, athletic socks, athletic shoes</td>
<td>1.19</td>
</tr>
<tr>
<td>Lightweight jacket, thermal long underwear top and bottoms, briefs, shell pants, athletic socks, athletic shoes</td>
<td>1.24</td>
</tr>
<tr>
<td>Lightweight jacket, long-sleeved fleece shirt, fleece pants, briefs, shell pants, athletic socks, athletic shoes</td>
<td>1.67</td>
</tr>
<tr>
<td>Ski jacket with detachable fiberfill liner, thermal long underwear bottoms, knit turtleneck, sweater, fiberfill ski pants, knit hat, goggles, mitten shell with fleece glove inserts, thin knee-length ski socks, insulated waterproof boots</td>
<td>2.3</td>
</tr>
<tr>
<td>Extreme cold-weather down-filled parka with hood, shell pants, fiberfill pants liner, thermal long underwear top and bottoms, sweat shirt, mitten shell with inner fleece gloves, thick socks, insulated waterproof boots</td>
<td>3.28</td>
</tr>
<tr>
<td>Extreme cold-weather expedition suit with hood (down-filled, 1 piece), thermal long underwear top and bottoms, sweat shirt, mitten with fleece liners, thick socks, insulated waterproof boots</td>
<td>3.67</td>
</tr>
</tbody>
</table>

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(Harirchi 2005)