EFFECTS OF ACUTE SODIUM INGESTION ON WATER CONSUMPTION AND RETENTION, DEHYDRATION, AND EXERCISE PERFORMANCE IN THE HEAT

A Thesis
by
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Abstract

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Hyperhydration prior to exercise (HPE) in the heat has been shown to improve endurance exercise performance. Co-consumption of sodium and water is one method of hyperhydrating. While this strategy is effective in controlled environments, promoting fluid consumption in unsupervised, non-thirsty individuals is difficult. Thirst can be induced with sodium ingestion via the vasopressin response. **PURPOSE:** We sought to determine the effects of sodium ingestion on ad-libitum, pre-exercise fluid consumption and retention, rates of dehydration, and subsequent exercise performance in the heat. **METHODS:** Nine male subjects (27 ± 4 yrs, 73 ± 9 kg) underwent three experimental procedures in which they consumed 60 mg/kg bm NaCl, (NA), an equal volume of placebo (PL), or no treatment (NT) followed by a 2-hr hydration period in which they consumed water ad-libitum (HP). Fluid consumption and urinary output were measured during HP. Immediately following HP, subjects began a 1-hr, moderate-intensity exercise session (1 hr ride), followed by a 200 kJ time trial

(TT), both performed on a cycle ergometer at an ambient temperature of 30° C. Nude body mass was measured to determine hydration status. Elapsed time to complete TT was used as a performance measure. Blood samples were analyzed for plasma volume (PV). Heart Rate (HR) was obtained at minutes 5 and 50 of the 1-hr ride. Data were analyzed using two-way repeated measures ANOVA.

RESULTS: Subjects consumed more water during HP in NA (1380 ± 581 mL) compared to PL (815 ± 483 mL, P = 0.05) and NT (782 ± 454 mL, P = 0.02). Fluid retained at the end of HP was greater in NA (821 ± 367 mL) compared to PL (244 ± 402 mL, P = 0.004) and NT (148 ± 289 mL, P < 0.001). NA induced a significantly greater rate of HPE (1.1 ± 0.5%) than did PL (0.3 ± 0.4%, P = 0.03) and NT (-0.01 ± 0.6%, P = 0.003). Dehydration rate following 1-hr ride was lower in NA (0.7 ± 0.6%) compared to PL (1.3 ± 0.7%, P = 0.045) and NT (1.6 ± 0.4%. P = 0.004). The TT was completed faster in NA (773 ± 158 s) compared to PL (851 ± 156 s, P = 0.004) and NT (872 ± 190 s, P = 0.006). No significant differences were detected between PL and NT for any of these variables. No significant effects due to treatment were observed in PV or HR.

CONCLUSION: The results suggest that acute sodium ingestion can enhance voluntary fluid consumption leading to hyperhydration and improved exercise performance in the heat. However, the increase in body water with sodium did not significantly affect plasma volume or exercising heart rates.

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Chapter 1

Introduction

Dehydration is marked by a decrease in body water levels and is quantified by a percent loss in body mass from normal basal levels. During exercise in the heat the primary thermoregulation mechanism is sweating. The fluid that constitutes sweat primarily comes from plasma. Thus, profuse sweating without concomitant fluid consumption can lead to dehydration and reductions in plasma volume. Low plasma volume can lead to reduction in blood flow to the skin which can compromise heat dissipation and thermoregulation (Sawka, 1992). Maintaining normal hydration status is important to exercise performance, as a dehydration rate of only one percent can be detrimental to work output (Convertino et al., 1996). During vigorous exercise, core body temperature rises, which can be detrimental to exercise performance and can lead to permanent disability and death.

Sweat rates during high intensity exercise in hot environments have been measured in excess of three liters per hour (Rehrer, 2001). This rate of fluid loss far exceeds the maximum rate of fluid emptying from the stomach. Thus, maintaining this inequality between fluid loss and fluid replacement will ultimately result in dehydration and an accompanying drop in exercise performance.

To combat dehydration, some individuals practice pre-hyperhydration. This strategy involves consuming large amounts of fluid prior to exercise in the heat in an attempt to raise body water levels. Techniques have been proposed to pre-hyper hydrate athletes prior to endurance exercise in the heat in order to maintain plasma volume and ultimately exercise performance. Three approaches to pre-hyper hydration are the over-consumption of water, consumption of glycerol with water, and consumption of sodium with water. By increasing body water levels, high sweat rates can be maintained for longer periods of time before dehydration occurs and work performance suffers. In fact, numerous investigations have demonstrated ergogenic effects from pre-hyperhydration prior to exercise in the heat.

Despite the efficacy of pre-hyperhydration in laboratory settings, this strategy may not be optimally used in the field. Thirst is the only stimulus humans possess to encourage fluid consumption. This reflex is propagated by the hormones angiotensin II and vasopressin, both of which are generally only secreted when an individual is dehydrated (Guyton, 1986). Thus, it can be difficult to achieve hyperhydration in non-dehydrated free-living individuals unless they are actively encouraged to drink, especially in individuals such as athletes, soldiers, and firefighters who are often preoccupied with preparing for their upcoming tasks. As a result, these individuals may not consume adequate amounts of fluid to achieve pre-hyperhydration. The rapid losses in body fluid pose great concern because the rate of gastric empting is only 1.0 L/h (Naghii, 2000). This is compounded by the fact that while exercising in the heat individuals have been found to seldom drink more than 0.5 L/h (Sanders, Noakes, & Dennis, 1999), while individuals exercising in the heat can reach sweat rates of 3.0 L/h (Rehrer, 2001).

The sole consumption of copious amounts of water does increase total body water. However, consuming only water causes dilution of the blood leading to excess renal excretion of fluids. As a result, body fluid quickly drops back down to basal levels. To attain hyperhydration, two strategies can be employed: co-consumption of water with glycerol, and co-consumption of water with sodium. In regards to glycerol and water consumption, studies have shown some benefit attaining hyperhydration in normal and hot environments. Glycerol is very hydrophilic, meaning it likes water. When consumed with water, it functions to hold onto water and expand plasma volume. The consumption of glycerol and water has been shown to not only significantly expand plasma volume prior to exercise, but also yield performing enhancing results (Hitchins, et al., 1999). However, glycerol has not consistently provided greater water retention than water alone and it is not known whether glycerol fully sustains hyper hydration, especially in hot conditions (Coutts, Reaburn, Mummery, & Holmes, 2002; Latzka, et al., 1997). Furthermore, glycerol consumption does not stimulate thirst and, therefore, its consumption may not be accompanied by adequate amounts of water consumption in free-living individuals.

Co-consumption of sodium with water prior to exercise has been investigated and results suggest that this technique successfully increases plasma volume resulting in increases in exercise performance. When sodium levels increase in the blood stream, there is an increase in the release of vasopressin. Vasopressin acts to help retain water at the level of the kidney and ultimately increase plasma volume (Guyton, 1986). In addition to its actions at the kidney, vasopressin also elicits the thirst reflex, which in theory would be beneficial to increasing and maintaining hydration status. If sodium

induces thirst reflex and is consumed with water, individuals would theoretically consume more water leading to greater increases in plasma volume and exercise performance.

To our knowledge, there is no literature that attests to the increase in thirst reflex and voluntary fluid consumption with sodium intake, and a subsequent exercise performance in the heat. With an ingestion of an acute sodium load, followed by ad libitum water consumption prior to exercise, it is believed that increased fluid consumption, decreased fluid excretion, and pre-hyperhydration will be achieved. Furthermore, these actions will lead to a blunting in the rate of dehydration during exercise in the heat and an improvement in exercise performance.

Statement of Problem

Despite the research done thus far on acute sodium loading and exercise performance, the direct relationships between acute sodium ingestion and voluntary fluid consumption, hyperhydration, and exercise performance in the heat have not been measured. The purpose of this study is to examine the effects of sodium ingestion on ad libitum water consumption, hydration status, and exercise performance in the heat.

Hypotheses

1. Sodium consumption will increase ad libitum fluid consumption and retention prior to exercise.

2. Sodium consumption and ad libitum water consumption will cause pre-hyperhydration.

3. Ingesting an acute sodium load followed by ad libitum water consumption will significantly increase plasma volume.

4. Sodium consumption combined with ad-libitum water consumption will result in a reduction in the rates of dehydration during exercise in the heat.

5. Improved hydration levels will result in lower exercising tympanic temperature.

6. Sodium induced hyperhydration will improve exercise performance in the heat.

7. Improved hydration will decrease heart rate response during submaximal exercise in the heat.

Significance of Findings

Individuals who partake in physical work in hot environments will have an improved way to maintain hydration status leading to enhanced exercise performance.

Chapter 2

Review of Literature

Hydration and Exercise Performance

Proper maintenance of hydration is essential to exercise performance. Many investigations have reported the effects of hydration status on endurance, sprint, power, and strength performance trials. It has been consistently found that endurance exercise performance is sustained better when individuals reduce rates of dehydration through adequate consumption of fluids. This relationship between hydration status and exercise performance is especially prevalent during exercise in hot environments. A study conducted by Casa et al. (2010) observed the effects of hydration status on exercise performance. Seventeen subjects took part in four separate 12 km trail runs consisting of a hydrated race trial, dehydrated race trial, hydrated submaximal trial, and a dehydrated submaximal trial. For the dehydrated trials, subjects began restricting fluid consumption 22 hours prior to their start time and were not given fluid during the trial. For the hydrated trials, subjects were allowed to drink water ad libitum until their start time and were given water during the trial. Results indicated that when properly hydrated, subjects were able to complete the 12 km trail race in the heat five percent faster than when dehydrated. A similar study conducted by Stearns et al. (2009) observed trail running race times and pacing. It was found that hydrated individuals were able to complete a 12 km (three 4 km loops) trail run in the heat faster than dehydrated individuals (3,191

seconds for hydrated compared to 3,339 seconds for dehydrated), and were able to maintain more consistent paces over the course of the exercise challenge with the greatest pacing discrepancy occurring during the final loop. The hydrated individuals had an average difference of 54 seconds between fastest and slowest loops, while the dehydrated individuals had an average difference of 111 seconds between fastest and slowest loops.

A study conducted by Moquin and Mazzeo (2000) looked at exercise performance in seven women in a euhydrated and dehydrated state. These subjects performed two separate graded tests to exhaustion, one in a dehydrated state and one in a hydrated state. Dehydration was achieved via a 45-minute submaximal exercise bout the evening before testing followed by 12 hours of fluid restriction. Results indicated a significant decrease in time to exhaustion during the dehydrated trial compared to the hydrated trial, 16.3 +/- 0.7 min and 17.3 +/- 0.7 min respectively. A study conducted by Cheuvront, Carter, Castellani, and Sawka (2005) also looked at exercise performance in euhydrated and dehydrated states. Subjects sat for three hours in a hot environment (45° C) either with fluid replacement (euhydrated) or without fluid replacement (3 % dehydrated). Subjects then performed a 30-minute cycling session at 50% VO_2 max followed by a performance trial. Results indicated that when dehydrated, subjects' performance trial resulted in a significant decrease in time to exhaustion.

Another study conducted by Merry, Ainslie, and Cotter (2010) looked at the effects of euhydration and dehydration in six trained and six untrained males. Subjects partook in two separate trials both consisting of cycling for 40 minutes @ 70 % of VO_2 max immediately followed by a 40-minute work performance trial. The evening before each exercise trial subjects underwent 50 minutes of exercise plus heat stress followed by

complete or partial overnight rehydration. During the euhydration ride, 100 % of mass loss was replaced at 10-min intervals throughout exercise by ingestion of a solution containing NaCl (2.9 $g \cdot L^{-1}$) and artificial sweetener, while during the dehydration trial only 20 % was replaced during constant load exercise followed by ad libitum intake during the performance ride. They found that when subjects were 1.5-2.0 % dehydrated compared to euhydrated, performance decreased by 12 % in the untrained group and seven percent in the trained group.

Slater et al. (2005) conducted a study measuring the effects of dehydration on sprint rowing trials. Four total trials were performed, two dehydrated and two euhydrated. Dehydration of four percent was accomplished with a 24-hour fluid restriction period that did not take place prior to the euhydrated trials. During the dehydration trials rowers showed a significant increase in time to complete the 2000 m performance trial.

In regards to strength and power, a study conducted by Judelson et al. (2007) measured the effects of dehydration on resistance exercise performance. Subjects completed three identical exercise trials under different states of hydration. Subjects either were euhydrated, 2.5 % dehydrated, or five percent dehydrated. Dehydration was achieved by heat-strain exercise (36-37° C) prior to trial and fluid restriction leading up to trial. The exercise protocol measured vertical jump height, peak lower body power, peak lower body strength, and central drive. In addition, subjects also attempted to complete a six-set back squat protocol with ten repetitions per set at 80 % of one repetition-maximum, which was determined during baseline measurements. If a subject was unable to complete all ten repetitions they simply went to exhaustion, rested and then

attempted to finish the remaining sets. There was no significant difference in vertical jump height, lower body peak power, or lower body strength between the three trials. However, in regards to the squat protocol when subjects were 2.5 % dehydrated, the percentage of total work performed compared to baseline significantly decreased in sets two and three and when five percent dehydrated, and percentage of total work performed compared to baseline was significantly decreased in sets two, three, four, and five.

More recently, Hayes and Morse (2010) reported performance decrements during a graded dehydration protocol with respect to strength and power. Subjects partook in one baseline exercise bout and five additional bouts with an increasing state of dehydration. Dehydration was induced via jogging in a hot environment (48° C) for 20 minutes. From baseline weight, exercise bouts were done at 1.0 +/- 0.5, 1.9 +/- 0.7, 2.6 +/- 0.8, 3.3 +/- 0.9 and 3.9 +/- 1.0 % dehydrated, in successive order. Results revealed no significant differences in vertical jump height, electromyography, or isokinetic knee extension peak torque at $120° \cdot s^{-1}$ with progressive dehydration. However, isometric leg extension peak torque was significantly reduced after the first and succeeding dehydration trials and isokinetic knee extension peak torque at $30° \cdot s^{-1}$ was significantly reduced after the third and succeeding dehydration trials.

Dehydration appears to have a greater negative effect on work performance during exercise in hot temperatures as opposed to cooler temperatures. When the temperature of the air is cooler than core temperature, the body is able to diffuse heat away from itself into the atmosphere, thus regulating its temperature. It has been previously reported by Nielson, Sjøgaard, Ugelvig, Knudsen, and Dohlmann (1986) that exercising at 30° C for two hours at 50 % of VO_2 max can decrease plasma volume by 16

% when in a hot environment. The earlier-mentioned study conducted by Casa et al. (2010) concluded that performance during the 12-km trail run significantly decreased due to dehydration while running in a high wet bulb globe temperature (WBGT) of 26.5° C. WBGT is a composite temperature used to estimate the effect of temperature, humidity, wind speed/wind chill, and visible infrared radiation/sunlight on humans, and is a far more accurate measurement of heat strain. In comparison, dehydration does not seem to result in the same magnitude of performance decrements during exercise in the cold.

The earlier-mentioned study by Cheuveront et al. (2005) concluded that three percent dehydration did not decrease exercise performance in colder weather (2° C) while it did significantly decrease exercise performance in hot weather (45° C). The previously-mentioned study conducted by Slater et al. (2005) not only observed the effects of hydration status on exercise performance, but also observed the effect of temperature. Two of the trials were conducted in a neutral temperature environment, 21.1 +/- 0.7° C and two of the trials were conducted in a hot temperature environment, 32.4 +/- 0.4° C. Time to complete the 2000 m performance time trial was significantly increased in both four percent dehydration trials, but was significantly greater in the hotter environment compared to the cooler neutral environment.

Based on the literature to date, it is clear that proper hydration techniques need to be applied in order to combat dehydration and the performance decrements that come with it, especially in a hot environment.

Physiological Causes and Responses to Dehydration

During exercise, heat production increases in skeletal muscle. Krustrup, González-Alonso, Quistorff, and Bangsbo (2001) found increases from 37.0° C up to 38.2° C in skeletal muscle with strenuous bouts of exercise. Gleeson (1998) found that during strenuous exercise the body's rate of heat production may exceed 1000 W. When increases in body temperature arise, they are sensed by proper central and skin thermoreceptors, which cause the hypothalamus to act out appropriate responses (Gleeson, 1998). In an attempt to maintain proper core temperature, the body utilizes a number of mechanisms to dissipate excess heat. One way the body is able to regulate core temperature is through conduction. Conduction is the transfer of heat by diffusion via a temperature gradient. The heat will move from the higher temperature body to the lower temperature body. As long as core/body temperature is higher than that of the external environment, conduction will allow an individual to adequately regulate core temperature (Gleeson, 1998). Another primary mechanism for heat dissipation is the evaporation of sweat. When core temperature does not exceed environmental temperature the body must find another way to dissipate its heat, sweating (Sawka, 1992). Sweat is compromised largely of water that is drawn from the plasma. Fluid must then be consumed to maintain proper plasma levels. If fluid consumption does not adequately replace fluid lost from the plasma volume due to sweating, dehydration will result. As dehydration progresses, plasma volume can deplete to the point that blood flow to working muscle is compromised and exercise performance is reduced (Sawka, 1992).

Exercise in the heat poses greater concern for dehydration and places an enhanced burden on the body when attempting to dissipate heat. It becomes more difficult to

conduct, or diffuse heat due to a temperature gradient away from the body due to the high ambient temperatures. This causes the main method for dissipation to be sweating. According to Sawka (1992), during exercise in the heat, sweat output tends to exceed water intake leading to dehydration. This state of dehydration leads to hypertonic-hypovolemia of the blood, causing hypernatremia, which can lead to CNS dysfunction. When individuals are dehydrated, sweat rate will diminish, leading to a compromised capacity to dissipate heat, greater heat storage, and a further decrease in ability to tolerate heat strain. Core temperature will increase even more, and with blood attempting to get to the skin to conduct heat away from the body, the overall central venous pressure drops and cardiac output is insufficient to maintain the metabolic and thermoregulatory demands of the hot environment. Sawka, Young, Francesconi, Muza, and Pandolf (1985) researched the effects of three, five, and seven percent dehydration on thermoregulatory and blood response in eight male subjects and determined that three to five percent dehydration level primarily reduced plasma volume with little effect on plasma osmolality and while the highest level of dehydration did not further decrease plasma volume, it did increase plasma osmolality. They concluded, that with exercise in the heat, core temperature and heart rate increased with severity of dehydration, sweating responses for a given rectal temperature were decreased with increased dehydration, and that reduced sweating was also strongly tied to plasma hyperosmolality.

Rates of Sweating/Dehydration

It has been previously stated that exercising in an environment that triggers sweating can ultimately lead to dehydration and the threat of dehydration increases with increases in exercise duration. An early study by Buskirk and Beetham (1960) measured

total water loss in response to endurance exercise. They took measurements on net body water loss due to a marathon (26.2 miles) and an 18-mile road race run in what were considered moderate to cool temperatures (specific ambient temps unknown). The marathon yielded a 6.0 +/- 0.8% dehydration rate, a calculated rate of 1.52 $L \cdot h^{-1}$, while the 18-mile road race resulted in a 4.1 +/- 0.8 % loss in fluid. An additional study conducted by Pugh, Corbett, and Johnson (1967) observed a mean dehydration rate of 5.2 +/- 1.2 % in 56 marathon runners.

Rate of sweating and dehydration becomes even greater during exercise in a hot environment and should be closely monitored. According to Sawka and Montain (2000) sweating rates of 1.0 - 2.5 $L \cdot h^{-1}$ are common with high intensity exercise in the heat, where rates in a cooler environment range from 0.25 - 2.0 $L \cdot h^{-1}$. Kurdak et al (2010) conducted a study which observed hydration status, body mass changes, sweat loss, and drinking behaviors of 22 football players. The study took place during a game set in a hot environment, 34.3 +/- 0.6° C. Players lost an average of 3.1 +/- 0.6 L of sweat over the 90-minute game. In another study conducted by Shirreffs et al. (2005) the sweat rate and total body mass loss due to water was observed in 26 male soccer players. The duration of the training session was 90 minutes in 32 +/- 3° C. After the training session, athletes were 1.59 +/- 0.61 % dehydrated. The average sweat loss was 2193 +/- 365 ml, equaling 1.46 +/- .24 $L \cdot h^{-1}$. In this study, soccer players only managed to replace 62 % of total fluid lost, indicating that athletes frequently due not adequately replace water and maintain hydration status, which can be problematic when athletes have multiple games in a day or even in subsequent days in hot conditions.

A study conducted by Fowkes Godek et al. (2010) looked at the sweat rates of 44 NFL players broken into three groups; small(wide receivers/defensive backs, 93 +/- 6 kg), midsize (linebackers/quarterbacks, 110 +/- 5 kg), and large (linemen: tight ends/defensive ends/interior linemen, 135.6 +/- 17 kg). Data for sweat rates was collected between days six and 14 on two consecutive years of training camps held at the same facility. Average temperature during the collection period was 25.9 +/- 1.9° C. Sweat rates were found to be significantly lower for the small group (1.42 ± 0.45 L $\cdot$ h^{-1}) compared to the midsize (1.98 ± 0.49 L $\cdot$ h^{-1}) and large (2.16 ± 0.75 L $\cdot$ h^{-1}) groups. However, sweat rates were not significantly different between midsize and large groups. When compared relative to body size the sweat rates were approximately 0.0153 L $\cdot$ kg^{-1} $\cdot$ h^{-1}, 0.0179 L $\cdot$ kg^{-1} $\cdot$ h^{-1}, and 0.0159 L $\cdot$ kg^{-1} $\cdot$ h^{-1} for the small, midsize, and large groups, respectively. Even though the temperatures in this study were relatively warm, they were cooler than the previous studies mentioned and yielded lower sweat rates.

Furthermore, a study by Morante and Brotherhood (2007) concluded significantly lower sweat rates with lower ambient temperatures in tennis players. In this study, tennis players were observed on 86 occasions in 43 tennis singles matches. During the observations temperatures ranged from 14.5 - 38.4° C. Results indicated an average sweat rate of 0.5 L $\cdot$ h^{-1} at the lowest ambient temperatures (approximately 15° C) up to sweat rates of 1.5 L $\cdot$ h^{-1} in the highest ambient temperatures (approximately 38° C). Maintaining body mass during exercise, especially in hot environments is challenging because sweat rates of up to 3.0 L $\cdot$ h^{-1} (Rehrer, 2001) will exceed maximum rates of gastric emptying of about 1.0 L $\cdot$ h^{-1} (Naghii, 2000).

Hyperhydration Techniques and Performance Benefits

Because rates of fluid loss due to sweating can easily exceed maximum rates of gastric emptying for water, dehydration may be inevitable during prolonged exercise in the heat. To combat the inevitable development, a strategy known as pre-hyperhydration can be employed. Pre-hyperhydration is a condition where body water levels prior to exercise are expanded to higher than normal levels. By inducing this state, individuals provide themselves with an expanded reserve of body water that can be used to form sweat. In doing so, pre-hyperhydrated individuals can lose greater volumes of body water to sweat before becoming dehydrated and experiencing the accompanying decrease in exercise performance. Simply consuming large amounts of fluid prior to exercise has been shown to be ineffective in achieving hyperhydration. When water is consumed alone, the renal system increases urine production in an attempt to maintain proper blood osmolarity and pressure. Thus, the excess water consumed is excreted and hyperhydration is not achieved.

A study conducted by Vrijens and Rehrer (1999) determined that attempting to replace sweat losses solely with pure water is less successful and decreases maintenance of plasma sodium levels compared to consuming equal amounts of Gatorade. In this study ten subjects performed a baseline one-hour pretrial followed by two, three-hour experimental trials on a cycle ergometer at 55 % VO_2 max. On trial days subjects consumed their last meal (of their choice) two hours prior to testing and were also instructed to drink one liter of Gatorade the night before the testing trial. On the day of the testing, subjects were allowed to consume water ad libitum leading up to coming to the lab. During the three-hour trial subjects were given either plain water or Gatorade

every 15 minutes at a rate equal to their fluid loss, which was determined in the pretrial. Plasma sodium decreases were greater with water than with Gatorade during the exercise bout. It was concluded that consuming sodium-free beverages during rigorous exercise does not adequately replace the sweat loss that occurs compared to a sodium-containing beverage.

Consumption of glycerol with water has been used to promote pre-hyperhydration. Glycerol is a three-carbon molecule that has hydrophilic characteristics. Because it is hydrophilic, glycerol will tend to hold onto water and reduce free water clearance. When glycerol and water are consumed together, more of the consumed water is retained allowing for maintenance of body water levels (Goulet, Rousseau, Lamboley, Plante, & Dionne, 2008; Hitchins, et al., 1999; Anderson, Cotter, Garnham, Casley, & Febbraio, 2001). A study conducted by Goulet et al. (2008) observed the effects of glycerol and pre-exercise hyperhydration during two hours of cycling at 65 % of VO_2 max with five, two-minute intervals performed at 80 % VO_2 max in 26-27° C. Following the two hours of cycling, subjects performed a cycling test to exhaustion. It was determined that when subjects were pre-exercise hyperhydrated (glycerol), total body water was 21.7 +/- 2.10 ml · kg body mass $^{-1}$ greater than when subjects were pre-exercise euhydrated. Body mass loss at the end of exercise was significantly different between groups with 1.7 +/- 0.3 % dehydration for pre-exercise hyperhydration and 3.3 +/- 0.4 % dehydration for pre-exercise euhydration. During the two hours of cycling, pre-exercise hyperhydration significantly decreased heart rate and perceived thirst, but rectal temperature, sweat rate, perceived exertion and perceived heat-stress did

not differ between conditions. Pre-exercise hyperhydration significantly increased time to exhaustion and peak power output, compared with pre-exercise euhydration.

An additional study conducted by Hitchins et al. (1999) looked at glycerol hyperhydration and its effects in a hot environment. Eight male cyclists underwent two separate 60-minute cycle ergometer time trials in 32° C. Each time trial was split into two phases, a 30-minute fixed workload phase and a 30-minute variable workload phase. Subjects either ingested a glycerol (1 g $\cdot$kg $^{-1}$ BM in a diluted CHO-electrolyte drink) drink equaling 22 ml $\cdot$ kg body mass $^{-1}$, or an equal amount of placebo (diluted carbohydrate-electrolyte drink). Glycerol ingestion expanded body water 600 ml greater than the placebo and in the 60-minute time trials at 32° C, the glycerol ingestion significantly improved performance by five percent compared to the placebo.

Coutts et al. (2002) measured the effects of glycerol ingestion on hyperhydration in Olympic distance triathletes in hot environments of 30.5 +/- 0.5° C and 25.4 +/- 0.2° C, days one and two respectively. The glycerol drink was a 0.5 M glycerol solution containing 1.2 g $\cdot$ kg $^{-1}$ body mass of glycerol combined with 25 ml $\cdot$ kg $^{-1}$ body mass of diluted carbohydrate-electrolyte solution. The placebo drink consumed consisted of equal amounts of the diluted carbohydrate-electrolyte solution along with water plus 1.0 ml $\cdot$ kg $^{-1}$ body mass of saccharine. Both drinks were consumed over a 60-minute period, two hours prior to exercise. Plasma volume during the loading period on the hot day was significantly greater when glycerol ingestion attenuated performance decrements in the heat. Following the glycerol loading, the increase in time to finish the triathlon between hot and warm conditions was significantly less compared to placebo trial, with a majority of the improvement occurring in the final 10 km run on the hot day. Despite performance

increases, sweat loss and overall fluid retention were not found to be significantly different between glycerol and placebo trials.

However, a study conducted by Anderson et al. (2001) concluded that when glycerol (1 g) was ingested with a bolus of water (20 ml · kg body mass $^{-1}$ low-calorie lemon-orange sweetener) two hours prior to 90 minutes of steady state exercise, overall fluid retention did increase as indicated by urine output (measured in pre-exercise urine volume: 1119 +/- 97 ml urine output, Glycerol vs. 1503 +/- 146 ml urine output, Placebo) compared to a placebo (20 ml · kg body mass $^{-1}$ low-calorie lemon-orange sweetener), leading to a five percent increase in exercise performance.

A study conducted by Latzka et al. (1997) further investigated the effects of glycerol on hyperhydration and thermoregulation with heat stress. Eight heat-acclimated subjects performed five trials (euhydration alone and glycerol hyperhydration, and water hyperhydration both with and without rehydration) on a treadmill in 35° C at approximately 45 % of VO_2 max. The glycerol solutions contained 1.2 g glycerol · kg lean body mass $^{-1}$ and total water drank during the hyperdydration period was 29.1 ml · kg lean body mass $^{-1}$. Results indicated no significant differences in increasing total body water between hyperhydration trials. In addition, the hyperhydration trials did not significantly differ from euhydration in regards to core temperature or sweating rate. Furthermore, no significant differences were found in either of these measures between water and glycerol hyperhydration trials. The previous research in regards to glycerol and its ability to aid in hyperhydration, total body water increases, retentions, and exercise performance appears to be inconclusive and may require further investigation.

Some research has found that glycerol does not hold an ergogenic effect and may actually cause more harm than good. A study conducted by Murray, Eddy, Paul, Seifert, and Halaby (1991) observed the physiological effects of four different drinks (10 % glycerol solution (G), 6 % carbohydrate-electrolyte beverage (CE), 6 % carbohydrate-electrolyte beverage plus 4 % glycerol (CEG), and water placebo (WP)) during a 90-minute cycle ergometer exercise bout at 50 % of VO_2 max. The testing was done at 30° Celsius. Though increases in plasma osmolality and attenuation of the decrease in plasma volume were found to be significantly greater with ingestion of the two glycerol solutions compared to placebo, data revealed no significant differences between treatments for heart rate, esophageal temperature, sweat rate, rating of perceived exertion, or levels of cortisol and aldosterone. In addition, gastrointestinal distress was found to be the greater in G compared with the other treatments.

Other concerns with the consumption of glycerol are the potential for hyponatremia, which is defined as blood sodium concentrations below 135 mmol, and CNS dysfunction. With the hydrophilic tendencies of glycerol and the compartmentalization of fluids that occurs with exercise and sweating, it is possible that the blood could become diluted with water and cause a drop in sodium concentration of the plasma. Glycerol can also cause issues in the brain because of its hydrophilic tendencies. Glycerol has been used to reduce intracranial pressure (ICP) via osmotic brain dehydration (Tourtellotte, Reinglass, & Newkirk, 1972). Glycerol also decreases cerebrospinal fluid (CSF) levels and increases cerebral blood flow to ischemic brain tissue (Tourtellotte et al., 1972). Other adverse effects of orally-administered glycerol include confusion, dizziness, nausea, and vomiting.

Sodium and Water Retention

Co-consumption of sodium and water has also been used to induce pre-hyperhydration. Sodium is an important electrolyte in the maintenance of hydration status. Its presence in the renal interstitium is vital to reabsorption of fluid that would otherwise be excreted following the filtration of the blood. Sodium consumption also helps to maintain proper plasma osmolality, which regulates vasopressin secretion from the pituitary gland. Circulating levels of vasopressin are essential for reabsorption of water from the filtrate in the proximal and distal tubules and the loop of henle of the nephron (Guyton, 1986). When plasma becomes dilute, for example after consumption of large amounts of pure water, vasopressin secretion ceases, urine production increases and fluid is excreted (Guyton, 1986). In addition to its role in promoting fluid retention, vasopressin also elicits the thirst reflex, which increases the desire to consume fluid. These two physiological responses to sodium support that its consumption may promote hyperhydration.

Inducing hyperhydration in free living, euhydrated individuals can be difficult because these individuals lack the thirst stimulus and thus do not readily consume water. While in hot environments, dehydration must be combated by replacing any fluid that is lost due to sweat. This can be tough to accomplish because thirst is not a good indicator of fluid replacement requirements. Inducing voluntary hyperhydration in free living individuals can be difficult because thirst is typically not perceived until an individual has reached a dehydration rate of two percent (Sawka & Montain, 2000), meaning that if a soldier is one percent dehydrated prior to deployment to combat, he/she will not feel the need to consume any fluids and will likely not voluntarily hyperhydrate. This evidence

suggests that sodium consumption can increase plasma osmolality and induce the thirst reflex even in euhydrated individuals via the vasopressin response.

Greenleaf et al. (1998) had euhydrated subjects undergo six different hyperhydrating treatments, drinking varying ionic and constituent compositions of fluid amounting to 10 ml/kg body weight (768 ml) during a 90 minute sitting period [(P1: 55 mEq · L^{-1} Na^+, 5 mEq · L^{-1} K^+, 365 mOsm · kg^{-1} H_2O, 2.0 mg · dL^{-1} glycerol, 2049 mg · dL^{-1} glucose, 416 mg · dL^{-1} citrate), (P2: 97 mEq · L^{-1} Na^+, 10 mEq · L^{-1} K^+, 791 mOsm · kg^{-1} H_2O, 4.0 mg · dL^{-1} glycerol, 3579 mg · dL^{-1} glucose, 753 mg · dL^{-1} citrate), (P2G: 113 mEq · L^{-1} Na^+, 11 mEq · L^{-1} K^+, 1382 mOsm · kg^{-1} H_2O, 2916 mg · dL^{-1} glycerol, 3543 mg · dL^{-1} glucose, 731 mg · dL^{-1} citrate), (AA: 164 mEq · L^{-1} Na^+, < 0.1 mEq · L^{-1} K^+, 253 mOsm · kg^{-1} H_2O, 1.0 mg · dL^{-1} glycerol, < 0.5 mg · dL^{-1} glucose, 854 mg · dL^{-1} citrate), and 01 and 02 (no drinking)] and an exercise drink also amounting to 10 ml · kg body weight $^{-1}$ of P1 for a 70 minute submaximal exercise bout (P1 for all treatments except 02 (no drinking)). Results indicated that P1 and AA significantly increased plasma volume during the sitting period 4.7 % and 7.9 %, respectively. The increases in plasma volume achieved at rest for AA, the lowest Osmolarity beverage (253 mOsm · kg^{-1}), maintained plasma volumes during exercise, whereas drinks containing lower amounts or no sodium at all and higher Osm (365-1382 mOsm · kg^{-1}) did not.

Another study conducted by Greenleaf et al. (1997) tested the effects of pre-exercise hyperhydration and cycle ergometer exercise. In this study time to exhaustion was measured during an exercise bout at 87-91 % VO_2 max. The five subjects in this study consumed either no drink (N), or one of two fluid formulations equaling 10 ml · kg

$^{-1}$ (743 +/- 161 ml). One drink being a multi-ionic carbohydrate drink, P1 (55 mEq · 1 $^{-1}$ Na^+, 4.16 g · 1 $^{-1}$ citrate, 20.49 g · 1 $^{-1}$ glucose and 365 mOsm · kg $^{-1}$ H_2O) and the other being a sodium chloride- sodium citrate drink, AA (164 mEq · 1 $^{-1}$ Na^+, 8.54 g · 1 $^{-1}$ citrate, < 5mg · 1 $^{-1}$ glucose, and 253 mOsm · kg $^{-1}$ H_2O). Percent changes in plasma volume from 105 minutes post consumption to the start of exercise were -1.5 +/- 3.2 % in the no drink trial, 0.2 +/- 2.2 % with P1, and 4.8 +/- 3.0 % with AA ($P < 0.050$ AA vs. P1 and no drink, P1 vs. no drink). In response to the different changes in plasma volume, exercise times to exhaustion were 24.68 +/- 1.50 min, 24.55 +/- 1.09 min, and 30.50 +/- 3.44 min for N, P1, and AA, respectively. Researchers concluded that the attenuated exercise performance for N and P1 could not be attributed differences in exercise metabolism from the carbohydrate or citrate. It was determined that the higher level of resting plasma volume for AA (greatest Na^+ concentration), along with greater acid buffering and potential increased energy substrate from citrate, may have caused the increased exercise performance.

The effects of immediate pre-exercise sodium loading have also been observed. A study conducted by Coles and Luetkemeier (2005) measured the effects of an immediate pre-exercise sodium load on plasma volume, endurance performance, and thermoregulation performance. Subjects exercised at 70 % VO_2 max for 45 minutes in 21.0 - 23.3° C immediately following ingestion of either a sodium load (IPOSL), 164 mEq Na^+, 255 mOsm · kg $^{-1}$ (10 ml · kg $^{-1}$ Shaklee U.S.), or a no sodium placebo (10 ml · kg $^{-1}$ doubly concentrated lemon flavored Crystal Light), beverage, each divided into three equal portions taken at 15 minute intervals. The submaximal ride was followed by a 15-minute performance time trial. Results indicated changes in resting plasma volume

prior to exercise were significantly different between treatments, with IPOSL having greater plasma volume. In addition it was determined that IPOSL lead to greater maintenance of plasma volume at both 15 and 30 minutes during the submaximal ride compared to placebo. Furthermore, there was a significant 7.8 % improvement in time trial performance with IPOSL compared to placebo.

A study conducted by Sims, Rehrer, Bell, and Cotter (2007) investigated whether a concentrated sodium beverage would increase plasma volume and reduce physiological strain leading to an increase in endurance cycling performance in women. Subjects either ingested a high Na^+ beverage (164 mmol $Na^+ \cdot L^{-1}$) or a low Na^+ beverage (10 mmol $Na^+ \cdot L^{-1}$) with 10 ml $\cdot$ kg $^{-1}$ body mass of H_2O, prior to exercising to exhaustion at 70 % of VO_2 max at 32° Celsius. The beverages were consumed in seven portions over a 60-minute period starting 105 minutes prior to the submaximal exercise. Results demonstrated that the high Na^+ beverage increased plasma volume 4.4 +/- 1.1 % prior to exercise compared to the low Na^+ beverage which only increased plasma volume 1.9 +/- 1.3 % prior to exercise, ($P < 0.001$). Furthermore, time to exhaustion increased significantly in the high Na^+ compared to the low Na^+ trial, 98.8 +/- 25.6 min vs. 78.7 +/- 24.5 min. Lastly, the core temperature rose much more quickly in low Na^+, 1.6 +/- 0.2° C $\cdot$ h $^{-1}$ vs. 1.2 +/- 0.2° C $\cdot$ h $^{-1}$ in high Na^+.

Another study conducted by Sims, van Vliet, Cotter, and Rehrer (2007) further enforced the performance benefit of sodium consumption and its effects it has in the heat. The methods in this study essentially matched that of the first study with the subjects now being male and the exercise now being running as opposed to cycling. Subjects ingested either a high Na^+ beverage (164 mmol $Na^+ \cdot L^{-1}$) or a low Na^+ beverage (10 mmol $Na^+ \cdot$

L $^{-1}$) with 10 ml · kg $^{-1}$ body mass of H_2O, prior to exercising to exhaustion at 70 % of VO_2 max in 32° Celsius. The beverages were consumed in seven portions over a 60-minute period starting 105 minutes prior to the submaximal running exercise bout. Results in this study revealed that the high Na^+ beverage increased plasma volume 4.5 +/- 3.7 % compared to the low Na^+ beverage which did not increase plasma volume, 0.0 +/- 0.5 %, ($P < 0.040$). Time to exhaustion increased significantly in the high Na^+ compared to the low Na^+ trial. In this study some of the subjects were forced to stop due to an ethical end point (core temperature = 39.5° C), stop times were 57.9 +/- 6 min (High Na^+) vs. 46.4 +/- 4 min (Low Na^+) ($P = 0.040$) and when subjects were exhausted times were 96.1 +/- 22 min vs. 75.3 +/- 21 min ($P = 0.030$) in high Na^+ and low Na^+, respectively. This study also revealed a significantly lower core temperature in the high Na^+ (38.9° C) trial compared to the low Na^+ trial (39.9° C).

The evidence in these studies gives strong indication that sodium consumption prior to exercise in the heat can help to maintain hydration status. However, thus far these studies did not allow for ad libitum water consumption and are not measuring the effects of Na^+ consumption on thirst and ad libitum water consumption levels.

Sodium and Thirst Reflex

If vasopressin elicits the thirst reflex just as it helps to retain water at the level of the kidney, then with pre-exercise sodium loading, individuals may be induced to consume significantly higher amounts of water and ultimately elevate plasma volume when compared to placebo or no treatment. However, despite the knowledge of vasopressin and its effects on thirst reflex, only one study tested the effects of sodium

loading and ad libitum water consumption. Johannsen, Lind, King, and Sharp (2009) had 10 males and 10 females exercise at 58 +/- 4 % of VO_2 max for 90 minutes on a cycle ergometer, 45 minutes after ingestion either 355 mL of chicken noodle soup (167 mmol $Na^+ \cdot L^{-1}$), a carbohydrate-electrolyte beverage (16 mmol $Na^+ \cdot L^{-1}$), or plain water. Following the 90 submaximal exercise bout, subjects completed a physical performance task, which was the total calculated work that would be completed in 30 minutes of exercise at 60 % of VO_2 max. The important factor in this study is that water consumption was ad libitum in all exercise trials. Fluid balance was found to be improved in the SOUP trial compared to the water trial, with subjects losing 251 +/- 418 g in the SOUP trial compared to a loss of 657 +/- 593 g in the water trial ($P = 0.002$). This was due to greater water intake throughout the SOUP trial compared to the water trial and an increase of water retention. Though the study did not show differences in performance between trials, it did reveal that when water consumption is allowed to be ad libitum, sodium consumption increases the amount of water consumed.

It has been demonstrated that sodium consumption helps to maintain hydration status and elevate plasma volume in both normal and hot environments via its influence on the hormone vasopressin. Because of the ability to maintain and/or elevate plasma volume, the consumption of sodium has also been shown to increase exercise performance in both normal and hot environments. Furthermore, in the single study to date that observed the effects of sodium and the thirst reflex it was determined that, with sodium consumption, water intake is increased. It now needs to be determined whether or not an acute sodium load will significantly increase the level of ad libitum water consumption prior to exercise and, if so observed, will result in pre-hyperhydration.

Furthermore, if an acute sodium load does increase water consumption, and pre-hyperhydration, will plasma volume, hydration level, and maximal exercise performance in the heat also increase when compared to trials that are not preceded by sodium consumption? If this is the case, this gap in current literature will not only be applicable to recreational or elite athletes, but also to people in the professional work world such as farmers, or even our country's military force. For example, when soldiers are overseas in the field they may not be consciously thinking to drink water due to all the external stress at hand, and may become dehydrated, causing decrease in personal performance. If they are given an acute sodium load, evidence suggests that soldiers would increase their water consumption and better maintain hydration status. By investigating this gap in literature, this study will produce evidence as to whether or not increases in water consumption increase plasma volume and ultimately provide further help for soldiers to perform maximally while in the field of duty.

Chapter 3

Methodology

Male subjects (n = 9) were recruited from a group of well-trained cyclists and triathletes. All subjects read and signed an informed consent form approved by the Institutional Review Board of Appalachian State University. Subjects also underwent a health screening to ensure that they qualified as low-risk individuals. During the first visit to the lab, subjects performed a graded exercise test to exhaustion (GET) on a Lode Excalibur electronically braked cycle ergometer (EBE) to determine their maximal oxygen consumption and power output at maximum oxygen consumption (PPO). The GET began with the subject pedaling at a Wattage equal to three times their mass in kilograms. The work rate progressed by 0.3 Watts $\cdot$ kg $^{-1}$ each minute until volitional exhaustion of the subject was achieved. Criteria for a maximal effort was: plateau in maximum oxygen consumption with an increase in work rate, volitional exhaustion of subject, respiratory exchange ratio > 1.15, and a heart rate near estimated maximum (220-age). Following completion of the GET, subjects rested for 10-15 minutes before undergoing a familiarization trial of a 12.2 km performance test ride (PT) that was used to assess performance of subjects during their subsequent lab visits. The objective of the PT was to complete 12.2 km as quickly as possible. During the PT, subjects were allowed to adjust the work rate of the cycle ergometer as desired, but were blinded to the actual work rate. The subjects were able to see their elapsed distance throughout the trial.

After the initial testing and familiarization visit, the subjects reported to the lab on three additional occasions, each following an overnight fast. Upon arrival, subjects first evacuated their bladder and had nude body mass and tympanic temperature measured. Subjects also underwent a blood draw via finger prick to measure hematocrit and hemoglobin levels using a Hemopoint H2 Analyzer. Urine specific gravity (USG) was measured using a portion of the initial bladder evacuation. Subjects then consumed a pre-event meal consisting of roughly 360 kcals accompanied by either 60 mg NaCl · kg · bm^{-1}, an equal volume of placebo (non-caloric sweetener Splenda), both distributed in capsule form, or no treatment. The subjects then rested quietly for two hours at an ambient temperature of 30-32° C while having unlimited access to water. Subjects were previously encouraged to drink if thirsty. Ad-libitum water consumption and urinary output was measured during the two-hour period to nearest ml. Following the two-hour hydration period, nude body mass, tympanic temperature, a blood draw for hematocrit and hemoglobin levels and bladder evacuation were repeated. Subjects then underwent a 60-min dehydration ride at an ambient temperature of 30-32° C and work rate of 50 % of their PPO. Heart rate was taken at the five and 50-minute marker during the dehydration ride. Following dehydration ride, blood samples for hematocrit and hemoglobin levels were drawn, tympanic temperature was measured, and total body wipe down, and a bladder void took place prior to nude body mass measurement. Immediately following assessments subjects returned to EBE to perform PT (as described above). Following the termination of the PT, the subjects evacuated their bladder, underwent total body wipe down prior to nude body mass measurement, had tympanic temperature measured, and had a blood sample drawn to assess hematocrit and hemoglobin levels. Subjects

refrained from water consumption from the termination of the hydration period until the completion of the PT. All exercise was performed on the EBE. Blood samples were analyzed for blood volume using the method of Dill and Costill (Dill, 1974). At least two days, but no more than seven days, separated each visit. Diet and fluid consumption were controlled for two days prior to each visit. Subjects were instructed to maintain their normal water consumption to maintain euhydration and were instructed to drink one liter of water the night before testing. Subjects were also instructed to keep a low sodium diet in the two days leading up to testing, and refrained from processed foods such as deli meats and cheeses. Blood variables, body mass, fluid consumption and retention, urinary output, and core temperature were analyzed using separate two-way, repeated measures ANOVA utilizing Sidak's post-hoc analyses where appropriate. Time to complete the time trial was compared using a one-way repeated measures ANOVA with Sidak's post-hoc. Level of significance was set at $P \leq 0.050$ and all results are presented as mean +/- *SE*.

Chapter 4

Results

Subjects

Age, mass, and VO_2 max measurements were taken for nine male subjects (mean +/- *SD*). All nine of the subjects completed the two-hour hydration period and dehydration ride. Two subjects, who were not experienced cyclists or triathletes, did not perform the performance trials due to the possibility of a learning effect in the performance trial. The mean age of the male cyclists (n = 9) was 27 +/- 4 years. The mean mass (n = 9) was 73.8 +/- 9.6 kg. The GET yielded a mean VO_2 max of 55.9 +/- 9.3 ml $\cdot$ kg $^{-1}$ $\cdot$ min $^{-1}$.

Water Consumption & Retention

Water consumption and retention measurements during the hydration period were taken on nine subjects. The amounts of water consumed during the two hour hydration period were 782 +/- 151 mL, 815 +/- 161 mL, and 1,380 +/- 194 mL for the no treatment (NT), placebo (Pl), and sodium (Na) trials, respectively. The amount of water consumed during the Na trial was significantly greater compared to both the NT (P = 0.024) and Pl (P = 0.050). Water consumption amounts were not significantly different between the NT and Pl trials (P = 0.959). Overall water amounts retained during the hydration period were 148 +/- 96 mL, 244 +/- 134 mL, and 821 +/- 122 mL for NT, Pl, and Na

respectively. Water retention for the Na trial was significantly greater compared to both the NT ($P < 0.001$) and Pl ($P = 0.004$) trials. Water retention for the Pl trial was not significantly different than the NT trial ($P = 0.724$). Water consumption and retention results are presented in Figure 1.

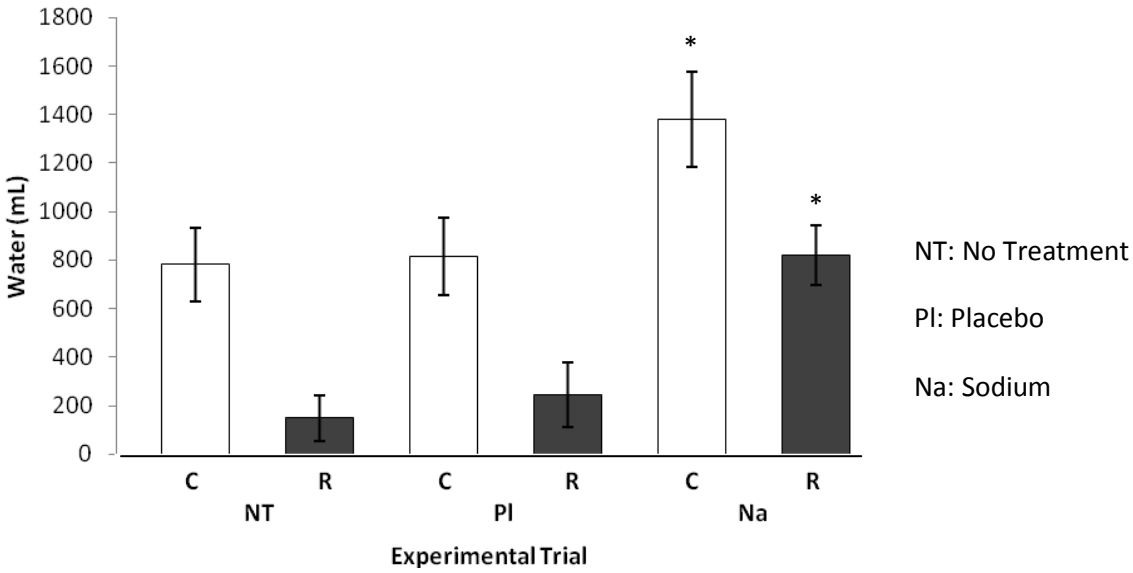

Figure 1. Water consumed (C) and retained (R) during the hydration period (mean +/- *SE*) for the three different treatments. * Indicates a significant difference compared to other trials ($P \leq 0.050$).

Performance Test

Average times to complete the PT were 872 +/- 72 seconds for NT, 851 +/- 59 seconds for Pl, and 773 +/- 60 seconds for Na. PT time was significantly improved during the Na trial compared to the NT ($P = 0.006$) and the Pl ($P = 0.004$) trials. No statistically significant differences were observed in completion times between the NT and Pl trials ($P = 0.614$). PT results are presented in Figure 2.

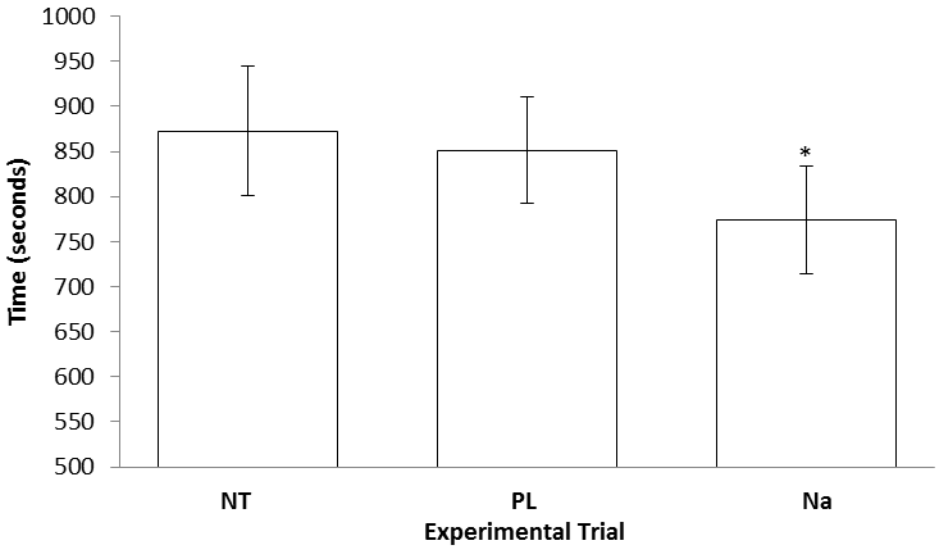

Figure 2. Performance trial times (mean +/- *SE*) for the three different treatments. * Indicates a significant difference compared to other trials (P ≤ 0.050).

Dehydration

Dehydration rates following the dehydration ride were measured in eight of nine subjects. One subject's data were not used because there was a clear error made during body mass measurements. After the dehydration ride, subjects were 1.6 +/- 0.1 % dehydrated in NT, 1.3 +/- 0.2 % dehydrated in Pl, and 0.7 +/- 0.2 % dehydrated in Na. Dehydration rate during the Na trial was significantly lower compared to both the NT ($P = 0.004$) and the Pl ($P = 0.045$) trials. Dehydration rate during the NT and Pl trials were not significantly different ($P = 0.444$). Dehydration results are presented in Figure 3.

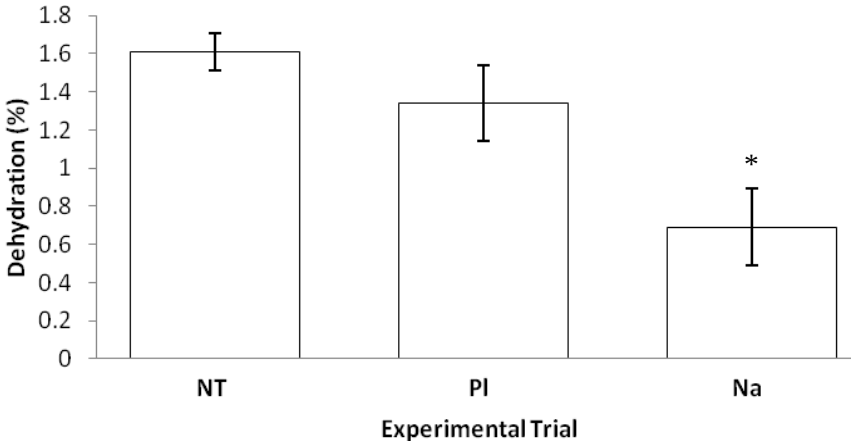

Figure 3. Percent Dehydration between trials (mean +/- *SE*). * Indicates a significant difference compared to other trials ($P \leq 0.050$).

Tympanic Temperature

Tympanic temperature (TT) measurements were taken and analyzed before consumption of the pre-exercise meal, before the beginning of the dehydration ride, before the beginning of the PT, and after completion of the PT. From pre-meal consumption to the beginning of the dehydration ride there was a 0.87 +/- 0.25 % increase in NT, a 1.2 +/- 0.41 % increase in Pl, and a 0.78 +/- 0.47 % increase in Na. Change in TT from pre-meal consumption to beginning of dehydration trial was not found to be significantly different between any trials (NT vs. Pl, $P = 0.740$; NT vs. Na, $P = 0.987$; Na vs. Pl, $P = 0.689$). Pre-meal consumption to the end of the dehydration ride yielded a 2.3 +/- 0.49 % increase, a 2.4 +/- 0.59 % increase, and a 1.6 +/- 0.34 % increase in the NT, Pl, and Na trials, respectively. Changes in TT from pre-meal consumption to the end of the dehydration trial were not found to be significantly different between any trials (NT vs. Pl, $P = 0.950$; NT vs. Na, $P = 0.124$; Na vs. Pl, $P = 0.052$). Increases in TT from pre PT to post PT were 1.6 +/- 0.29 %, 0.96 +/- 0.24 %, and 1.8 +/- 0.36 % in NT, Pl, and Na

trials, respectively. Change in TT from pre to post PT were found to be significantly different between Na and Pl trials ($P = 0.043$), but not Na and NT ($P = 0.955$) or NT and Pl ($P = 0.638$) trials. Increases in TT from pre-meal consumption to post PT were 3.6 +/- 0.55 %, 3.5 +/- 0.87 %, and 3.4 +/- 0.72 % in NT, Pl, and Na trials respectively. No significant differences were found between trials (NT vs. Pl, $P = 0.997$; NT vs. Na, $P = 0.983$; Na vs. Pl, $P = 0.991$). Actual temperatures (mean +/- *SD*) are presented in Table 1.

Table 1.

Average Tympanic Temperature Throughout Trials.

Treatment	**Pre-meal**	**Pre-dehydration ride**	**Post-dehydration ride**	**Post-PT**
NT	36.3 +/- 0.3	36.6 +/- 0.2	37.1 +/- 0.6	37.6 +/- 0.7
Pl	36.0 +/- 0.5	36.5 +/- 0.3	36.9 +/- 0.5	37.3 +/- 0.5
Na	36.2 +/- 0.5	36.5 +/- 0.3	36.8 +/- 0.5	37.4 +/- 0.5

*All values are expressed in degrees Celsius

Plasma Volume

Plasma volume (PV) measurements were analyzed for nine subjects pre-meal, post hydration period, and after the dehydration ride. Changes in PV after the hydration period were -1.1 +/- 3.2 %, -4.4 +/- 3.1 %, and 1.3 +/- 1.7 % in the NT, Pl, and Na trials, respectively. PV after the hydration period was not significantly different in the Na trial

compared to the NT (P = 0.908) and Pl (P = 0.507) trials or in the Pl compared to the NT trials (P = 0.842). Changes in PV after the dehydration ride compared to pre hydration period were -8.7 +/- 3.4 %, -10.1 +/- 2.1 %, and -7.0 +/- 2.8 % in the NT, Pl, and Na trials, respectively. No significant differences were found between Na and NT (P = 0.980), Na and Pl (P = 0.467), or Pl and NT (P = 0.928).

Table 2.

Plasma Volume Percent Change During Trials.

Treatment	**Post-hydration period**	**Post- dehydration ride**
NT	-1.1 +/- 3.2 %	-8.7 +/- 3.4 %
Pl	-4.4 +/- 3.1 %	-10.1 +/- 2.1 %
Na	1.3 +/- 1.7 %	-7.0 +/- 2.8 %

Heart Rate

Heart rates (HR) were recorded at minutes five and 50 of the dehydration ride. HR at minute five was 136 +/- 4 bpm, 135 +/- 3 bpm, and 136 +/- 4 bpm for the NT, Pl, and Na trials, respectively. No significant differences were found between Na and NT (P = 0.999), Na and Pl (P = 0.864), or Pl and NT (P = 0.885). HR at minute 50 were 155 +/- 4 bpm, 151 +/- 4 bpm, and 151 +/- 4 bpm for NT, Pl, and Na trials, respectively. No significant difference was found between Na and NT (P = 0.416), Na and Pl (P = 1.000), or Pl and NT (P = 0.221). Change in HR between the five and 50-minute marker was 19 +/- 1 bpm, 16 +/- 3 bpm, and 15 +/- 3 bpm for NT, Pl, and Na, respectively. No

significant difference in heart rate change was found between Na and NT ($P = 0.481$), Na and Pl ($P = 0.868$), or Pl and NT ($P = 0.729$). There was a significant time effect on HR. All treatments saw a significant increase in HR over the dehydration ride, NT ($P < 0.001$), Pl ($P < 0.001$), and Na ($P < 0.001$).

Table 3.

Heart Rates During Each Trial at Five Minutes, 50 Minutes, and Change from Five to 50 Minutes.

Trial	HR @ 5 min	HR @ 50 min	Change in HR
NT	136 +/- 4	155 +/- 4	19 +/- 1
Pl	135 +/- 3	151 +/- 4	16 +/- 3
Na	136 +/- 4	151 +/- 4	15 +/- 3

* All values are expressed in beats per minute (BPM)

Tests of Hypotheses

- We accept the hypothesis: Sodium consumption will increase ad libitum fluid consumption and retention prior to exercise ($P < 0.050$).
- We accept the hypothesis: Sodium consumption and ad libitum water consumption will cause pre-hyperhydration ($P < 0.050$).
- We reject the hypothesis: Ingesting an acute sodium load followed by ad libitum water consumption will significantly increase plasma volume ($P > 0.050$).

- We accept the hypothesis: Sodium consumption combined with ad-libitum water consumption will result in a reduction in the rates of dehydration during exercise in the heat ($P < 0.050$).
- We reject the hypothesis: Improved hydration levels will result in lower exercising tympanic temperature ($P > 0.050$).
- We accept the hypothesis: Sodium induced hyperhydration will improve exercise performance in the heat ($P < 0.050$).
- We reject the hypothesis: Improved hydration will decrease heart rate response during submaximal exercise in the heat ($P > 0.050$).

Chapter 5

Discussion

The four main findings with this research were that consumption of 60 mg NaCl · kg · bm^{-1} stimulated a significant increase ad libitum water consumption, increased water retention, decreased rate of dehydration and improved PT time. Subjects consumed nearly 0.5 L more water during a two-hour period following ingestion of sodium vs. placebo or no treatment. This particular finding is important because the research on the effect of sodium ingestion on ad libitum water consumption is limited. The only other study conducted on sodium consumption in regards to thirst reflex did reveal an increase in ad libitum water consumption (Johannsen et al, 2009). However, the ad libitum fluid consumption was during exercise and did not measure a direct effect on sodium consumption in regards to pre-exercise hyperhydration. In addition to increasing the amount of fluid consumed, when subjects ingested sodium they also retained a significantly greater amount of fluid compared to the other trials. These two findings supported our first hypothesis that sodium consumption will increase ad libitum fluid consumption and retention prior to exercise. Though we did not actually test for levels of vasopressin, we assume that with its relationship to increased sodium levels, vasopressin levels did increase and ultimately played a large role in both stimulating water consumption in our subjects as well as helping them to retain greater amounts of total body fluid.

The increase in fluid consumption and retention with sodium consumption also led to the support of our second hypothesis that sodium consumption followed by ad libitum water consumption will cause pre-hyperhydration. The rate of hyperhydration prior to exercise was 1.1 +/- 0.5 % with sodium, which was significantly greater than the 0.3 +/- 0.4 % with Pl and the -0.01 +/- 0.6 % with NT. This finding is in support of previous research on varying sodium concentrated beverages inducing pre-hyperhydration, in which the beverages with the highest concentration of sodium induced the greatest state of hyderhydration (Greenleaf et al., 1997; Greenleaf et al., 1998).

Despite the increases in fluid retention and rate of hyperhydration with sodium consumption, it is not clear as to where all of the fluid was housed. With an increase in fluid consumption and retention and an increase in the rate of hyperhydration we also expected that ingesting an acute sodium load followed by ad libitum water consumption would significantly increase plasma volume. However, this was not the case. Plasma volume levels with sodium consumption did slightly increase 1.3 +/- 1.7 % prior to exercise compared to a 1.1 +/- 3.2 % decrease in NT, and a 4.4 +/- 3.1 % decrease in Pl. Changes in plasma volume with sodium consumption were not found to be significantly different compared to NT or Pl trials before or after the dehydration ride. Our findings are refuted by several studies in which sodium induced significant expansion of plasma volume prior to exercise. Coles and Luetkemeier (2005) found a 7.8 % difference in plasma volume with sodium compared to placebo prior to exercise, with sodium increasing plasma volume 3.1 %. Greenleaf et al. (1998) found a 7.9 % increase in plasma volume prior to exercise and Sims, van Vliet, Cotter, and Rehrer (2007) found a 4.5 % increase in plasma volume with sodium. Though sodium did slightly increase

plasma volume prior to exercise in our study, it was not to the extent found in previous research nor was it found to be significantly greater compared to other treatments as in previous research. The lack of significance could in part be due to a small sample size and high variability, causing the statistical power to be low (0.115). A lack of significance could mean that a large portion of the fluid consumed during the two-hour hydration period moved and remained in the interstitial fluid. Alternatively, perhaps the water was absorbed into other cells and structures throughout the body. We do suspect, however, that it did not end up as plasma. The Hemopoint H2 Analyzer, the machine used to determine plasma volume, is factory calibrated and has a total precision coefficient of variation of less than one percent. In addition, in order to further minimize variability of readings we tested four blood samples at each collection time. We took the samples and ran coefficient of variation tests on each four-sample set. The coefficients of variation for these series of samples ranged from zero to 0.143.

Dehydration rate measured from the pre-hydration period time point to the post-dehydration time point was significantly lower in Na (0.7 +/- 0.2 %) compared to NT (1.6 +/- 0.1 %) and Pl (1.3 +/- 0.2 %). Plasma volume was not different between Na, PL, and NT at any time during the protocols. Though plasma volume did not expand, the subjects did consume significantly greater amounts of water with Na during the hydration period leading to an increase in total body water prior to exercise and thus a decrease in the rate of dehydration. Our finding that sodium helped to maintain hydration status with exercise in the heat is similar to previous research findings. Coles and Luetkemeier (2005) determined that subjects were able to better maintain hydration status with sodium consumption compared to placebo during submaximal exercise. Greenleaf et al. (1998)

also found that higher sodium-concentration drinks led to greater maintenance of hydration status during submaximal exercise. The reduced rate of dehydration found is important because, as was discussed previously, dehydration levels of as little as one percent can result in decreased exercise performance (Convertino et al., 1996). This evidence suggests that the dehydration rates we observed of one percent or greater in the NT and Pl trials would lead to a decrease in subjects' performance.

Tympanic temperature was another variable that we suspected would be significantly altered with sodium consumption. Given the increase in prehyperhydration with sodium consumption, we suspected subjects would be able to better thermoregulate themselves, leading to a significant decrease in TT change throughout the trials compared to Pl and NT trials. However, in most cases this did not occur. Our findings in regard to sodium's effects on TT differ from those of Sims, van Vliet, Cotter, and Rehrer (2007) wherein sodium ingestion caused reduced thermoregulatory strain in exercise to exhaustion at 70 % VO_2 max. This reduction in thermoregulatory strain was evidenced by a significantly lower core temperature at exhaustion in the sodium trials. Sims van Vliet, Cotter, and Rehrer (2007) may have seen significant decreases in thermoregulation due to the fact that their exercise protocol required approximately one hour to complete, whereas in the current investigation, the average exercise duration was approximately 13 minutes.

We saw no difference in heart response to exercise due to treatment. In our original hypothesis, we expected to see a decrease in heart rate response in Na because of the expected expansion of plasma volume. However, we did not see any significant differences in either plasma volume or heart rate due to treatment. This is congruent with

the results of Sims, van Vliet, Cotter, and Rehrer (2007), who found no changes in plasma volume during exercise or heart rate response during exercise as a result of sodium-induced hyperhydration.

With a significant increase in rate of hyperhydration prior to exercise and a significant decrease in dehydration rate in Na it is not surprising that sodium consumption led to a significant improvement in performance during the PT. During Na, subjects completed the PT one-and-a-half to two minutes faster on average compared to NT and Pl, likely due to a reduced rate of dehydration. Our findings support previous research conducted on the effects of sodium and exercise performance in both normal and hot environments. Coles and Luetkemeier (2005) determined that with an acute sodium load subjects performed significantly more work during the 15 minute performance trial in a mild environment (21.0 - 23.3° C). Likewise Sims, van Vliet, Cotter, and Rehrer (2007) concluded that exercise time to exhaustion was significantly increased with sodium consumption in a hot environment (32° C). Our findings, along with the findings of Sims, van Vliet, Cotter, and Rehrer (2007), have implications for both elite and recreational athletes, soldiers, and individuals who work in hot environments for prolonged periods of time.

Conclusion

We demonstrated that consumption of sodium induced fluid consumption and retention and led to hyperhydration prior to exercise in the heat. Though consumption of sodium did not lead to a significant expansion of plasma volume, reduced submaximal heart rate changes during exercise in the heat, or consistent lowering of temperature

during trials, sodium consumption did significantly improve the total amount of water retained by subjects, leading to a decrease in dehydration rate and ultimately an improvement in exercise performance in the heat. These findings suggest that sodium consumption can be beneficial for improving hydration status for individuals exposed to a hot environment, whether it be athletic, recreational, or work related. Further research needs to be conducted on sodium's effects on heart rate, tympanic temperature, and plasma volume expansion.

References

Anderson, M., Cotter, J., Garnham, A., Casley, D., & Febbraio, M. (2001). Effect of glycerol-induced hyperhydration on thermoregulation and metabolism during exercise in the heat. *International Journal of Sport Nutrition and Exercise Metabolism, 11* (3), 315-333.

Buskirk, E. B., & Beetham, W. P. (1960). Dehydration and body temperature as a result of marathon running. *Medicina Sportiva (Roma), 14* (9), 493-506.

Casa, D., Stearns, R., Lopez, R., Ganio, M., McDermott, B., Walker, Y. S.,…, Maresh, C M. (2010). Influence of hydration on physiological function and performance during trail running in the heat. *Journal of Athletic Training, 45* (2), 147-156.

Cheuveront, S. N., Carter, R. I., Castellani, J. W., & Sawka, M. (2005). Hypohydration impairs endurance exercise performance in temperate but not cold air. *Journal of Applied Physiology, 99,* 1972-1976.

Coles, M., & Luetkemeier, M. (2005). Sodium-facilitated hypervolemia, endurance performance, and thermoregulation. *International Journal of Sports Medicine, 26* (3), 182-187.

Convertino, V., Armstrong, L., Coyle, E., Mack, G., Sawka, M., Senay, L. J., & Sherman W. M. (1996). American College of Sports Medicine position stand. Exercise and fluid replacement. *Medicine and Science in Sports and Exercercise, 28* (1), i-vii.

Coutts, A., Reaburn, P., Mummery, K., & Holmes, M. (2002). The effect of glycerol hyperhydration on Olympic distance triathlon performance in high ambient temperatures. *International Journal of Sport Nutrition and Exercise Metabolism, 12* (1), 105-119.

Dill, D. C. & Costill D. L. (1974). Calculation of percentage changes in volumes of blood, plasma, and red cells in dehydration. *Journal of Applied Physiology, 37* (2), 247-248.

Fowkes Godek, S. P., Peduzzi, C., Burkholder, R., Condon, S., Dorshimer, G., Bartolozzi, A. R. (2010). Sweat rates, sweat sodium concentrations, and sodium losses in 3 groups of professional football players. *Journal of Athletic Training, 45* (4), 364-371.

Gleeson, M. (1998). Temperature regulation during exercise. *International Journal of Sports Medicine,* 19, S96-S99.

Goulet, E., Rousseau, S., Lamboley, C., Plante, G., & Dionne, I. (2008). Pre-exercise hyperhydration delays dehydration and improves endurance capacity during 2 h of cycling in a temperate climate. *Journal of Physiological Anthropology, 27* (5), 263-271.

Greenleaf, J., Looft-Wilson, R., Wisherd, J., Jackson, C., Fung, P., Ertl, A.,…, Whittam, J. H. (1998). Hypervolemia in men from fluid ingestion at rest and during exercise. *Aviation Space, and Environmental Medicine, 69* (4), 374-386.

Greenleaf, J., Looft-Wilson, R., Wisherd, J., McKenzie, M., Jensen, C., & Whittam, J. (1997). Pre-exercise hypervolemia and cycle ergometer endurance in men. *Biology of Sport/ Institute of Sport, 14* (2), 103-114.

Guyton, A. (1986). *Textbook of medical physiology.* Philadelphia: W.B. Saunders Company.

Hayes, L., & Morse, C. (2010). The effects of progressive dehydration on strength and power: is there a dose response? *European Journal of Applied Physiology, 108* (4), 701-707.

Hitchins, S., Martin, D., Burke, L., Yates, K., Fallon, K., Hahn, A., & Dobson, G. P. (1999). Glycerol hyperhydration improves cycle time trial performance in hot humid conditions. *European Journal of Applied Physiology and Occupational Physiology, 80* (5), 494-501.

Johannsen, N. M., Lind, E., King, D. S., & Sharp, R. L. (2009). Effect of preexercise electrolyte ingestion on fluid balance in men and women. *Medicine & Science in Sports & Exercise, 41* (11), 2017-2025.

Judelson, D., Maresh, C., Farrell, M., Yamamoto, L., Armstrong, L., Kraemer, W.,…, Anderson, J.M. (2007). Effect of hydration state on strength, power, and resistance exercise performance. *Medicine & Science in Sports & Exercise, 39* (10), 1817-1824.

Krustrup, P., González-Alonso J., Quistorff, B., Bangsbo, J. (2001). Muscle heat production and anaerobic energy turnover during repeated intense dynamic exercise in humans. *The Journal of Physiology, 536* (3), 947-956.

Kurdak, S., Shirreffs, S., Maughan, R., Ozgünen, K., Zeren, C., Korkmaz, S.,…, Dvorak, J. (2010). Hydration and sweating responses to hot-weather football competition. *Scandinavian Journal of Medicine and Science in Sports*, 133-139.

Latzka, W. A., Sawka, M.N., Montain, S. J., Skrinar, G.S., Fielding, R.A., Matott, R. P., & Pandolf, K. B. (1997). Hyperhydration: thermoregulatory effects during compensable exercise-heat stress. *Journal of Applied Physiology, 83*, 860-866.

Merry, T., Ainslie, P., & Cotter, J. (2010). Effects of aerobic fitness on hypohydration-induced physiological strain and exercise impairment. *Acta Physiologica (Oxf), 198* (2), 179-190.

Moquin, A., & Mazzeo, R.S. (2000). Effect of mild dehydration on the lactate threshold in women. *Medicine & Science in Sports & Exercise, 32* (2), 396-402.

Morante, S. M., & Brotherhood J. R. (2007). Air temperature and physiological and subjective responses during competitive singles tennis. *British Journal of Sports Medicine, 41* (11), 773-778.

Murray, R., Eddy, D.E., Paul, G.L., Seifert, J.G., & Halaby, G.A. (1991). Physiological responses to glycerol ingestion during exercise. *Journal of Applied Physiology, 71* (1), 144-149.

Naghii, M. (2000). The significance of water in sport and weight control. *Nutrition and Health, 14* (2), 127-132.

Nielsen, B., Sjøgaard, G., Ugelvig, J., Knudsen, B., & Dohlmann, B. (1986). Fluid balance in exercise dehydration and rehydration with different glucose-electroyte drinks. *European Journal of Applied Physiology and Occupational Physiology, 55* (3), 318-325.

Pugh, L. G., Corbett, J. L., & Johnson, R. H. (1967). Rectal temperatures, weight losses, and sweat rates in marathon running. *Journal of Applied Physiology, 23* (3), 347-352.

Rehrer, N. (2001). Fluid and electrolyte balance in ultra-endurance sport. *Sports Medicine, 31* (10), 701-715.

Sanders, B., Noakes, T., & Dennis, S. (1999). Water and electrolyte shifts with partial fluid replacement during exercise. *European Journal of Applied Physiology, 80* (4), 318-323.

Sawka, M. (1992). Physiological consequences of hypohydration: exercise performance and thermoregulation. *Medicine & Science in Sports & Exercise, 24* (6), 657-670.

Sawka, M. N., & Montain, S.J. (2000). Fluid and electrolyte supplementation for exercise heat stress. *The American Journal of Clinical Nutrition, 72*, 564S-572S.

Sawka, M., Young, A., Francesconi, R., Muza, S., & Pandolf, K. (1985). Thermoregulatory and blood responses during exercise at graded hypohydration levels. *Journal of Applied Physiology, 59* (5), 1394-1401.

Shirreffs, S., Aragon-Vargas, L., Chamorro, M., Maughan, R., Serratosa, L., & Zachwieja, J. (2005). The sweating response of elite professional soccer players to training in the heat. *International Journal of Sports Medicine, 26* (2), 90-95.

Sims, S., Rehrer, N., Bell, M., & Cotter, J. (2007). Preexercise sodium loading aids fluid balance and endurance for women exercising in the heat. *Journal of Applied Physiology, 103* (2), 534-541.

Sims, S., van Vliet, L., Cotter, J., & Rehrer, N. (2007). Sodium loading aids fluid balance and reduces physiological strain of trained men exercising in the heat. *Medicine & Science in Sports & Exercise, 39* (1), 123-130.

Slater, G., Rice, A., Sharpe, K., Tanner, R., Jenkins, D., Gore, C., & Hahn, A. G. (2005). Impact of acute weight loss and/or thermal stress on rowing ergometer performance. *Medicine & Science in Sports & Exercise, 37* (8), 1387-1394.

Stearns, R., Casa, D., Lopez, R., McDermott, B., Ganio, M., Decher, N.,…, Maresh, C. M. (2009). Influence of hydration status on pacing during trail running in the heat. *The Journal pf Strength and Conditioning Research, 23* (9), 2533-2541.

Tourtellotte, W. W., Reinglass, J.L., & Newkirk, T.A. (1972). Cerebral dehydration action of glycerol. I. Historical aspects with emphasis on the toxicity and intravenous administration. *Clinical Pharmacology and Therapeutics, 13* (2), 159-171.

Vrijens, D., & Rehrer, N. (1999). Sodium-free fluid ingestion decreases plasma sodium during exercise in the heat. *Journal of Applied Physiology, 86* (6), 1847-1851.

Vita

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