# THE EFFECTS OF ACUTE SODIUM BICARBONATE SUPPLEMENTATION ON REPEATED SPRINT ABILITY IN RECREATIONALLY ACTIVE COLLEGE-AGED MALES

By

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A Thesis Submitted in Partial Fulfillment of The Requirements for the Degree of Master of Science in Exercise Science To the office of Graduate and Extended Studies of East Stroudsburg University of Pennsylvania

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## **ABSTRACT**

A Thesis submitted in partial fulfilment of the requirements for the degree of Master of Science in Exercise Science to the office of Graduate and Extended Studies of East Stroudsburg University of Pennsylvania.

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Title: The Effects of Acute Sodium Bicarbonate Supplementation on Repeated Sprint Ability in Recreationally Active College-Aged Males

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## **Abstract**

This study investigated the effects of acute ingestion of sodium bicarbonate on fatigue and power output during a repeated-sprint cycling protocol (10 x 6 second sprints interspersed with 30 seconds of passive recovery) in college-aged males. Nine males participated, but one was dropped. Following the completion of two familiarization sessions, participants were randomly assigned, in a counterbalanced fashion, to begin in the experimental group (NaHCO $_3$ ) or placebo group (NaCl) utilizing a crossover design format, so that all participants engaged in both conditions. Participants supplemented with 0.3 g/kg of sodium bicarbonate and / or 0.1 g/kg of sodium chloride 60 minutes before exercise and were administered in a double-blind format with the ingestion of a 1.5 g/kg fixed carbohydrate meal and 7 ml/kg of fluid to help minimize possible GI upset. There were no significant findings for: Average Mean Power Output (AMPO), Average Peak Power Output (APPO), Average Delta Blood Lactate (ADBL), Average Heart Rate (AHR), Average Rating of Perceived Exertion (ARPE), and fatigue. The mean and standard deviations are listed for the placebo vs experimental conditions: AMPO (907.44 ± 166.86 vs 921.10  $\pm$  162.91 Watts), where (f = 2.062, p = 0.37). APPO (1180.59  $\pm$  242.56 vs 1196.34 ± 239.46 Watts), where (f = 0.667, p = 0.737). ADBL (8.03 ± 1.84 vs 9.13 ± 3.05 mmol/L), where (t = -1.856, p = .113). AHR (161.89  $\pm$  6.94 vs 164.00  $\pm$  8.11 beats per minute), where (t = -1.202, p = 0.268). ARPE (15.25  $\pm$  1.06 vs 14.93  $\pm$  1.15), where (t = 1.075, p = 0.318). Fatigue (10.48  $\pm$  3.17 vs 9.88  $\pm$  4.28 percent), where (t = .780, p = .461). Amount of supplement ingested, and training status likely influenced results. Acidosis may only play a minimal role in fatigue and decline in sport performance during repeated sprint activities in this population. Further research is recommended to investigate the causes of fatigue and the role it plays in repeated sprint ability (RSA).



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#### **CHAPTER I**

### **INTRODUCTION**

Various team and racket sports require athletes to perform repeated sprints interspersed by short recovery periods. The ability to perform a maximal effort sprint and recover before the next sprint occurs is known as repeated-sprint ability (RSA) (Girard, Mendez-Villanueva, & Bishop, 2011). It can be helpful to categorize sprints into two different types of exercises which include intermittent-sprint and repeated-sprint exercises (RSE) (Girard et al., 2011). During intermittent-sprint exercise athletes sprint for 10 seconds or less with longer periods of rest (60-300 seconds) which would allow for an almost complete recovery before the next sprint occurs (Girard et al., 2011). Complete recovery, according to Girard et al. (2011), would mean that the athlete could complete the next sprint with equivalent or nearly as much work output (J) as the previous sprint. In contrast, while RSE also has sprints lasting 10 seconds or less, the recovery periods are remarkably shorter and are defined by rest periods lasting 60 seconds or less (Girard et al., 2011). As can be seen, the major difference between both types of sprints are the length of the recovery periods. As noted above, during intermittent-sprint exercise the recovery periods are long enough for the athlete to be

recovered before the start of the next sprint, while during RSE the recovery periods are so short in duration that the athlete has inadequate time to sufficiently recover, thereby hindering athletic performance (Girard et al., 2011).

When testing work outputs during intermittent and RSE a performance decrement is typically observed during RSE and not during intermittent-sprint exercise. For example, a decrease in performance can be seen during RSE when analyzing work output over the course of 5 sprints, interspersed with 30 seconds recovery (Girard et al. 2011). After each consecutive repeated sprint, work output decreased. On the contrary, when two minutes of recovery were allotted between each sprint during intermittent sprint exercises, work output stayed nearly the same during all five sprints, demonstrating that there was no performance decrement (Girard et al., 2011).

The rate of fatigue is influenced by many factors, including but not limited to the following: exercise mode, running surface, resistive load, repetitions performed, duration of work, duration of rest periods, intensity of exercise, previous activities, sport position, level of fitness, time of day, leg stiffness, environmental conditions, sex, and age (Girard et al., 2011). Fatigue is a multifaceted and complex phenomenon and is brought on by many mechanisms responsible for the decline in athletic performance (Girard et al., 2011). Since many mechanisms are responsible for fatigue it makes it difficult to define what "recovery" means and how this relates to the athlete. For our purposes, recovery, in the context of RSA, can broadly be defined as the athlete's ability to maintain or replicate sprint velocity or power output from sprint to sprint.

We can define fatigue as the loss of power output, such as during cycling activities, or a decrease in speed, such as during running activities (Girard et al., 2011). Fatigue can be assessed via the fatigue index (FI) or percentage decrement score ( $S_{\text{dec}}$ ) during RSE (Girard et al., 2011). The FI considers your best and worst sprint performance and analyzes the decline in power, work, or speed over the course of the RSE, whereas the  $S<sub>dec</sub>$  takes the performance of the athlete and compares that to a suggested optimal performance; assuming that all sprints were performed at maximum effort (Girard et al., 2011).

As mentioned earlier, many mechanisms are responsible for fatigue onset. The primary focus of the present study is to investigate the effects of acidosis on repeatedsprint performance. High intensity exercise causes an increase in intracellular hydrogen ion concentration and this is thought to negatively impact sprint performance by altering contractile elements within the muscles (Girard et al., 2011). The increase in acidity is from the release of protons in the glycolytic pathway resulting from ATP hydrolysis (Robergs, Ghiasvand, & Parker, 2004). ATP is broken down into ADP, Pi and H<sup>+</sup> but only ADP and Pi get recycled leaving H<sup>+</sup> behind to accumulate (Robergs et al., 2004). It should be noted that the increase in acidity is not only due to proton release, but also occurs due to the body's inability to buffer the excess protons (Robergs et al., 2004). Amino acids, proteins, inorganic phosphate, sodium bicarbonate, creatine phosphate hydrolysis and lactate production help to remove these excess protons intracellularly (Robergs et al., 2004). Mitochondrial and sarcolemmal transport as well as a bicarbonate-dependent exchanger also help to buffer these protons (Robergs et al.,

2004). The accumulation of various metabolites during high intensity exercise not only affect muscle function but may also negatively impact force generation (Sale, Saunders, & Harris, 2010). Girard et al. (2011) explains that acidity as a direct cause of fatigue has been questioned. One of the reasons noted by Girard et al. (2011) are because high power outputs have been measured even when acidity was present, however acidity may still induce fatigue and decrease performance by slowing the amount of absolute ATP produced from glycolysis. Glycolytic ATP provision may be compromised due to the acidity inhibiting phosphofructokinase and glycogen phosphorylase activity (Girard et al., 2011).

Fatigue occurring secondary to acidosis may potentially be mitigated by ingestion of substances that act as buffers, which includes the nutritional supplement sodium bicarbonate. Sodium bicarbonate is an extracellular blood buffer (Ducker et al., 2013). Supplementing with an acute dose of sodium bicarbonate has been shown to raise blood pH which may slow the decline in pH observed during high intensity exercise, again potentially enhancing RSA through diminishing and / or delaying the onset of acidosis and fatigue (Ducker et al., 2013). When sodium bicarbonate is increased extracellularly, it raises the pH gradient and encourages the transport of hydrogen ions across the cell membrane (Sale, Saunders, Hudson, Wise, Harris, & Sunderland, 2011). By purposely inducing an alkaline state within the body by supplementing with sodium bicarbonate, Sale et al. (2011), explain that this may increase high intensity exercise performance by increasing work output while simultaneously delaying fatigue.

One recent study had eleven participants perform 10 x 6 second sprints interspersed with 60 seconds recovery (Miller, Robinson, Sparks, Bridge, Bentley, & McNaughton, 2016). The study demonstrated that the experimental group ingesting sodium bicarbonate performed greater total work over the duration of the protocol than either the control or placebo groups. The study concluded that improved buffering capabilities may be at play (Miller et al., 2016). Although this study is similar to the present study, current literature on sodium bicarbonate has yet to clarify whether acidosis is the main cause of fatigue during repeated-sprint activities. There could be other mechanisms responsible for the decline in performance. The objective of this study is to investigate whether the acute supplementation of sodium bicarbonate allows for better maintenance of RSA.

## *Purpose*

The aim of this study was to investigate the effects of the acute ingestion of sodium bicarbonate on fatigue and power output during a repeated-sprint cycling protocol (10 x 6 second sprints interspersed with 30 seconds of passive recovery) in recreationally active college-aged males.

## *Null Hypotheses*

- 1. There will be no difference in fatigue scores (percent decrement) between groups.
- 2. There will be no difference in average mean power output between groups.
- 3. There will be no difference in average peak power output between groups.
- 4. There will be no difference in average delta blood lactate between groups.
- 5. There will be no difference in average rating of perceived exertion.
- 6. There will be no difference in average heart rate between groups.

## *Limitations*

- 1. The participants may or may not have gave their maximal effort during the repeated sprint protocol.
- 2. There is the possibility of participant drop-out due to injury, illness or for other unanticipated events.
- 3. The measurement of pH was not directly measured, but rather we used blood lactate as an indicator of pH.
- 4. There was high variability in data which was impacted by the low number of participants who completed the study protocol.

## *Delimitations*

- 1. Recreationally trained males between the ages of 18-30 participated.
- 2. Participants were apparently healthy and free of any musculoskeletal injuries and / or surgeries 6 months prior to testing.
- 3. Participants were free of any gastrointestinal issues such as: Irritable Bowel Syndrome (IBS) or Inflammatory Bowel Disease (IBD) including Crohn's and Colitis.

4. Participants were not to ingest any ergogenic supplements 30 days prior to study participation.

## *Operational definitions*

- 1. Repeated-sprint ability (RSA) The ability to perform a maximal effort sprint and recover so as to be able to replicate the previous power output during the next sprint (Girard et al., 2011)
- 2. Repeated-sprint protocol 10 sets of a 6 second sprint on a cycle ergometer, separated by 30 seconds passive recovery between each set.
- 3. Average mean power output The mean power output from each 6 second sprint averaged across the protocol.
- 4. Average peak power output The highest power output from each 6 second sprint averaged across the protocol.
- 5. Recreationally active Healthy adults should participate in ≥30 minutes of physical activity daily, ≥5 days or week totaling ≥150 minutes weekly (Garber et al., 2011).
- 6. Fatigue Calculated using the percent decrement method; ((fatigue =  $100 -$ [(total power output/ideal power output) x 100] where: total power output = sum of MPO values from all sprints; ideal power output = the number of sprints x MPOmax)) (Glaister, Stone, Stewart, Hughes, & Moir, 2004).

### **CHAPTER II**

## **LITERATURE REVIEW**

## *Energy Systems and Repeated Sprints*

The phosphagen system (ATP-PC system) is the fastest and most efficient way the body can generate Adenosine Triphosphate (ATP) needed for exercise (Powers & Howley, 2012). The body can generate ATP through a simple reaction which requires phosphocreatine (PC) to donate a phosphate group to ADP to form ATP (Powers & Howley, 2012). Creatine kinase is the enzyme responsible for catalyzing the reaction. ATP can be resynthesized quickly from the PCr reaction. It should be noted that there is a limited supply of phosphocreatine in skeletal muscle. As these levels drop, this may cause a decrease in athletic performance because the rate of ATP resynthesis is compromised (Powers & Howley, 2012).

As mentioned above, the most rapid way to rephosphorylate ATP during high intensity exercise is through the phosphocreatine system. Muscular ATP stores are approximately 20-25 mm/kg dm, and after just one second; the ATP turnover rate is 15 mm/kg (Spencer, Bishop, Dawson, & Goodman, 2005). Spencer et al., also state that

after 1 set of a maximal 6 second cycling sprint, an 8-16 percent reduction of ATP was recorded however, when 5 repetitions of a 6 second cycling sprint were performed with 24-30 seconds recovery a 4-24 percent decrease in muscular ATP was recorded.

One kilogram of muscle can house approximately 80 mmol of phosphocreatine however, after just one second; phosphocreatine turnover rate is 9 mmol/kg (Girard et al., 2011). Girard et al., also states that after a 6 second sprint phosphocreatine stores can be depleted 35-55 percent from original resting values. In one study, 10 x 6 second sprints were performed with 30 seconds recovery and after the first sprint a 57% reduction in PCr was recorded and after the tenth sprint it had decreased to 16% (Spencer et al., 2005). Since the complete resynthesis of phosphocreatine can take up to five minutes, and given the fact that recovery times are usually less than 60 seconds; a decline in performance is seen following each consecutive repeated sprint due to the incomplete recovery of the phosphocreatine system (Girard et al., 2011).

Anaerobic glycolysis contributes to about 40% of the energy requirement during a 6 second sprint, but this contribution declines after successive sprints are performed (Girard et al., 2011). Lastly, oxidative phosphorylation supplies up to 10% of the energy requirement, but as more sprints are performed the contribution of ATP from this pathway is increased up to 40% (Girard et al., 2011). Since  $VO<sub>2max</sub>$  is usually reached during this type of activity, the ability to perform sprints may be limited by  $VO_{2max}$ . This limitation is hypothesized to decrease RSA and therefore, increasing one's  $VO<sub>2max</sub>$  could help in the final several sprints ultimately enhancing sprint performance and delaying fatigue onset (Girard et al., 2011). Research has suggested that increasing one's VO2

max or lactate threshold through endurance or high intensity interval training has demonstrated an increased rate of phosphocreatine resynthesis (Bishop et al., 2011). Therefore, enhancing an athlete's aerobic metabolic pathways may maximize their ability for resynthesis of phosphocreatine; ultimately yielding more ATP to be used as energy during high intensity activity (Bishop et al., 2011).

### *Muscle Fatigue*

As mentioned in chapter 1, many factors contribute to fatigue that may cause a decrease in sport performance. One such potential mechanism for fatigue could be ionic disturbances at the cellular level arising from intense exercise. Intense exercise will negatively impact cell membrane excitability hindering muscular force production however, more research needs to be conducted whether this adds to fatigue during RSE (Girard et al., 2011).

Inorganic phosphate accumulation may also play a role in fatigue development. It is suggested that disrupted excitation contraction coupling may decrease force production due to the impaired functioning of contractile machinery. *In vitro* studies show that elevated inorganic phosphate is present and can alter calcium release, calcium sensitivity of myofibrils and consequently can decrease the number of cross-bridge formations (Girard et al., 2011). Further research is needed to see if this theory is present during repeated sprint activities (Girard et al., 2011).

A decrease in action potential firing rate also known as neural drive might also contribute to fatigue development. During high levels of fatigue, the body may fail to

recruit the needed musculature to perform the given task (Girard et al., 2011). When fatigue is low, research shows that neural activation on an EMG is stable and fixed however, when fatigue is high, EMG signals show that neural activation is suppressed (Girard et al., 2011). Therefore, it is apparent that during high levels of fatigue the body fails to activate the needed machinery to perform the task and consequently may add to fatigue during RSA. A potential reason for this failure could be due to arterial  $O<sub>2</sub>$ desaturation and is associated with a decline in work output (Girard et al., 2011). Spinal factors, specifically certain motoneurons, may exhibit reduced excitability that may also contribute to fatigue during RSA (Girard et al., 2011). Furthermore, supraspinal factors, relating to electrical brain activity and associated neurotransmitters may also be the cause however, it is still not well understood (Girard et al., 2011).

Our primary focus for this study was to investigate how the ingestion of sodium bicarbonate may help to prevent fatigue due to acidosis brought on by high intensity exercise. As mentioned earlier, the increases in acidity are from the release of protons in the glycolytic pathway resulting from ATP hydrolysis (Robergs et al., 2004). As ATP is broken down, H + is left behind and accumulates (Robergs et al., 2004). This accumulation of excess hydrogen ions may negatively impact contractile components within the muscle and may also undesirably impact glycolysis by negatively affecting the ATP derived from this pathway (Girard et al., 2011). Glycolysis may be affected by adverse consequences to phosphofructokinase and glycogen phosphorylase (Girard et al., 2011). The increasing number of metabolites brought on by exercise such as hydrogen, ADP, and Pi not only affect muscle function, but may also negatively impact

force generation (Sale et al., 2010). One way the body deals with excess accumulation of H<sup>+</sup> are through the body's various membrane transport systems (Bishop et al., 2011). A major membrane transporter within the muscle is known as the monocarboxylate transporters (MCTs) (Bishop et al., 2011). This membrane transporter is responsible for pH regulation and is at the forefront of pH regulation both during and after exercise (Bishop et al., 2011). Other membrane transporters include: sodium-bicarbonate cotransporter and the sodium-hydrogen exchanger (Bishop et al., 2011). As stated earlier, amino acids, proteins, inorganic phosphate, sodium bicarbonate, creatine phosphate hydrolysis and lactate production also help to remove these excess protons intracellularly (Robergs et al., 2004).

## *Co-supplementation of beta alanine and sodium bicarbonate on RSA*

Co-supplementation of both beta alanine and sodium bicarbonate have shown varying results as it relates to sport performance, and whether the additive effects of both supplements enhance performance are not clear (Hobson, Harris, Martin, Smith, Macklin, Gualano, & Sale, 2013). In one study that examined the combined effects of beta alanine and sodium bicarbonate on a maximal 4-minute cycling bout concluded that the additive effects of both supplements were minor (Bellinger, Howe, Shing, & Fell, 2012). In another study examining the combined effects of both supplements, participants completed a cycling capacity test to determine time to exhaustion (Sale et al., 2011). The results of this study concluded that there was no additive benefit from co-supplementation (Sale et al., 2011).

Another study examining the combined effects of beta alanine and sodium bicarbonate instructed their participants to ingest 4.8 grams beta alanine or placebo for a total of 4 weeks, and then the following two weeks they supplemented with 6.4 grams beta alanine. Sodium bicarbonate was ingested acutely before test administration. The participants were to perform a RSA protocol which consisted of 5 sets of a 6 second cycling sprint. A cycling capacity test was also performed and was completed at a workload of 110% of their VO<sub>2</sub> peak. The results of the study conclude that carnosine levels were increased due to beta alanine supplementation and blood bicarbonate levels were also increased due to ingestion of sodium bicarbonate. Performance was only increased during the cycling capacity test in the beta alanine group, with no improvement from co-supplementation of sodium bicarbonate. There were no significant results relating to the RSA test between groups (Danaher, Gerber, Wellard, & Stathis 2014).

## *Sodium bicarbonate supplementation on RSA*

As discussed previously, there are many mechanisms responsible for the onset of fatigue. Recent research suggest that low pH can hinder athletic performance during sprints. It is hypothesized that performance is decreased due to the interactions of low pH and its antagonistic effects it has on contractile elements within the muscle (Miller et al., 2016). The decrease in performance could also be due diminished ATP regeneration (Miller et al., 2016). As exercise commences, the muscle simultaneously begins to produce protons. These protons are then released from muscle cells and make their

way into the extracellular fluid surrounding the cell where they contact various buffers, such as bicarbonate (Miller et al., 2016). Ingestion of sodium bicarbonate essentially makes the hydrogen gradient from inside to outside the cell much larger (by increased extracellular buffering) thereby enhancing the rate at which hydrogen and lactate is shuttled out of the cell (Miller et al., 2016). By improving buffering, we can improve the amount of work muscles can generate (Miller et al., 2016).

During one study, 25 male rugby players ingested 0.3 grams/kg of sodium bicarbonate or placebo (Cameron, McLay-Cooke, Brown, Gray, & Fairbairn, 2010). Sixtyfive minutes post ingestion of sodium bicarbonate, the athletes performed a sport specific warm-up followed by a high intensity workout. The nine-minute workout consisted of running, sprinting, sidestepping, passing and tackling and was designed to simulate gameplay. Afterwards, time motion analysis was used to measure the Rugby Specific Repeated Sprint Test (RSRST) in which the athletes performed 10 x 40-meter sprints interspersed with 30 seconds rest (Cameron et al., 2010). The results of the study demonstrated that there was an increase in pH and sodium bicarbonate blood concentrations after the RSRST in the sodium bicarbonate group. Blood lactate was higher in the sodium bicarbonate group versus the placebo group. There were no differences in performance between experimental and placebo groups. It should be noted that GI upset was elevated in the experimental group ingesting sodium bicarbonate. The GI issues associated with sodium bicarbonate ingestion could affect gameplay, thus it would be wise to use caution when ingesting this supplement (Cameron et al., 2010).

### *Sodium Bicarbonate Supplementation*

Research shows that 0.3 g/kg of sodium bicarbonate raises the concentration of bicarbonate in the body to about 5 or 6 mmol/L<sup>-1</sup> (Siegler, Marshall, Bray & Towlson, 2012). This precise amount has been deemed an optimal quantity to yield a "bloodbuffering pool" to combat the additional protons released during high intensity exercise (Siegler et al., 2012). It has been shown that 0.3 g/kg sodium bicarbonate increases exercise performance (McNaughton, Gough, Sanjoy, Bentley, & Sparks, 2016). To reap the benefits of the ingestion of sodium bicarbonate it is essential to reach peak alkalosis, or the time after ingestion when bicarbonate levels are at their highest (McNaughton et al., 2016). Current research remains unclear as to the appropriate timing of sodium bicarbonate ingestion when bicarbonate levels are peaking (McNaughton et al., 2016). Research demonstrates a link between ingestion of sodium bicarbonate and gastrointestinal upset (GI upset) however, there is limited research on the optimal dosing strategy to limit GI discomfort while also promoting increases in exercise performance (McNaughton et al., 2016). It is suggested to consume 1.5 g/kg body weight of a carbohydrate meal and 7 ml/kg body weight of fluid to minimize GI upset (Carr, Slater, Gore, Dawson, Burke, 2011). During the present study, 0.3 g/kg of sodium bicarbonate were consumed 60 minutes prior to exercise (Gough, Rimmer, Osler & Higgins, 2017).

### **CHAPTER III**

## **METHODOLOGY**

## *Subjects*

This study was approved by the Institutional Review Board of East Stroudsburg University. All testing took place in the Human Performance Laboratory at East Stroudsburg University. A total of nine males volunteered for the study between the ages of 18 and 30. One participant could not complete all four sessions due to GI upset from sodium bicarbonate ingestion and, consequently, was not included in data analysis. During their first session, the protocols and any possible risks and benefits were explained to the participants and any questions were answered. The participants signed informed consent (Appendix B) and then completed a PAR-Q (Appendix E) before they participated in the study. Participants were also instructed to fill out an Activity Questionnaire (Appendix C) to determine their current activity status. The data collection sheet can be found in (Appendix D). Participants engaged in two familiarization sessions and two experimental trials. During both familiarization sessions the entire cycling protocol was performed. Data on their mean and peak power outputs were collected, however the familiarization data was

not analyzed. The same procedures were conducted during the experimental trials. After gathering the data from both experimental trials, the percent decrement formula was used to determine fatigue scores. It should be noted that all participants were tested under both conditions in a cross-over research design. Participants ingested both supplements throughout the course of the study. Half the participants were randomly assigned to first be tested with placebo (sodium chloride), while the other half of the participants were first tested with sodium bicarbonate. After they had consumed their first supplement, during their next visit they were to consume the opposite supplement. A total of 48 hours was the minimum time allotted between testing sessions. Supplements were administered in a double-blind format. The participants were instructed to not consume any additional ergogenic aids during the duration of testing. Participants were asked to refrain from the ingestion of caffeine and alcohol 24 hours prior to testing days. Participants were encouraged to maintain their current level of physical activity according to ACSM guidelines throughout the study, but were asked to not exercise 24 hours prior to testing. Participants were asked to eat a similar diet 24 hours prior to testing days. Participants were encouraged to use *MyFitnessPal™* to log and track their food intake 24 hours prior to all testing. The *MyFitnessPal™* app was used so participants could go back and review what they had previously eaten to assist participants in replication of the 24-hour pretest diet. This was implemented to ensure they would be consuming a similar diet prior to each experimental trial

however, it should be noted that this was not requirement. Participants needed to be 60 minutes post absorptive prior to testing.

## *Supplementation*

Participants ingested 0.3 grams per/kg body weight of sodium bicarbonate or 0.1 grams per/kg placebo (NaCl) 60 minutes before testing (Gough et al., 2017). Sodium bicarbonate or placebo was mixed with 7 ml/kg body weight of liquid (Crystal Light<sup>™</sup> Lemonade Kraft Heinz Foods Company, Chicago, IL) to disguise the supplements so the participant would not know which supplement they were consuming (Carr et al., 2011). In addition, 1.5g CHO/kg body weight of a carbohydrate meal (Thomas Bagels, Bimbo Bakeries USA™) was consumed with their supplement to help minimize the possibility for gastrointestinal upset (Carr et al., 2011). If any adverse side effects from the supplement occurred, the participants were informed to let the researcher know immediately so the situation could be handled safely and appropriately.

### *Procedures*

The current study used a double-blind placebo-controlled crossover design to investigate the effects of sodium bicarbonate on repeated sprint ability. A total of four sessions were needed for the completion of the study. Students volunteered for the study by signing their name and listing their contact information on a sheet of paper. The investigator later contacted them and told them instructions for their first visit. The participants were previously informed to wear athletic clothing (shorts and t-shirt),

closed toed shoes for their first visit, and to arrive 60 minutes post-absorptive. Participants were also instructed not to perform any exercise 24 hours prior to testing. They were also told to avoid alcohol and caffeine 24 hours prior to testing.

During their first visit (familiarization session one), the delimitations, cycling protocol and supplementation was explained. The participant had the opportunity to ask any questions. They were informed that they could drop out of the study at any time. The informed consent, PAR-Q, and activity assessment were distributed, read and signed by participants. The participants were asked to eat a similar diet before all trials. As mentioned earlier, participants were instructed in the use of *MyFitnessPal™* to help them remember what they had eaten on previous testing days however, the use of the app was encouraged but not required. The participants were instructed to maintain their current level of physical activity during the duration of the study. During their first visit, participant demographic data, including age (years), height (m), body mass (kg), and body composition (% body fat) were collected. A stadiometer was used to measure the height of each participant while standing barefoot, and a handheld device (Omron HBF-306, Bannockburn, Illinois) measuring body mass and body composition was used.

After the necessary documents were signed and demographic data were collected, familiarization session one began, and was the first of two familiarization sessions to be completed before experimental trials commenced. The familiarization session consisted of the subject performing the entire cycling protocol (10 x 6 second sprints separated with 30 seconds passive recovery) on an electromagnetically braked cycle ergometer (Lode Excalibur Sport, Groningen, The Netherlands). A heart rate

monitor (Polar Accurex Plus: Polar Electro Oy, Kempele, Finland) was fitted before the cycling protocol began. Seat height was also adjusted so that a 5◦ knee flexion occurred when the hip was in full extension. Handle bars were adjusted according to participant preference. Toe clips were used to prevent foot slippage during the cycling protocol. Before testing began an initial 4 minute warm up was performed on the cycle ergometer at 120 watts followed by one minute of passive recovery. Next, three maximal accelerations lasting one second each interspersed with 30 seconds passive recovery occurred. After the third and final maximal acceleration three minutes of seated passive recovery took place. At minute one of recovery, resting blood lactate was taken with a handheld blood analyzer (Lactate Pro; Arkray, Inc., Kyoto, Japan). All sprints began with the dominant leg's crank arm in a 45◦ position in the forward direction of the vertical axis. During the recovery periods in the protocol, participants were given a 5 second verbal countdown followed by the command "go" to initiate the next 6 second sprint. A set resistance of 0.70 NM/kg $^{-1}$  was utilized for the cycling protocol. Participants were instructed to remain seated throughout the entirety of the protocol. Participants were verbally encouraged to pedal as fast as possible during each sprint, and a rating of perceived exertion (RPE) using the 6-20 Borg Scale (Borg, 1970) was administered immediately after sprints 2, 4, 6, 8, and 10. Peak and mean power outputs were recorded with a computer interface for each 6 second sprint performed on the cycle ergometer. Blood lactate was drawn immediately after the cycling protocol ended. Familiarization session two was identical to familiarization session one.

Experimental trial one consisted of the participant consuming either sodium bicarbonate or placebo. After the ingestion of their supplement and a fixed carbohydrate meal, the timer was set for one hour. After one hour, the participant then went through the warmup and the protocol (10 x 6 second sprints interspersed with 30 seconds recovery). Experimental trial two was the same as experimental trial one except the opposite supplement was ingested. At least 48 hours was allotted between experimental trials one and two. The maximum amount of time that passed between trials one and two was 10 days. For each of the 6 second sprints that occurred, APPO and AMPO were recorded via computer interface. Heart rate, rating of perceived exertion, and blood lactate were collected. Fatigue was also calculated using the percent decrement score. *Figure 1* depicts the process in which participants progressed through the study.

### *Figure 1. Flow Chart*

Familiarization Session 1: Entire cycling protocol is performed

Familarization Session 2: Identical to Familiarization Session 1

Experimental ingestion of NaHCO<sub>2</sub> or NaCl

Experimental ingestion of opposite supplement not consumed during Experimental Trial 1

### *Data Reduction*

Mean and peak power outputs were recorded for each sprint via the computer interface for each participant under each condition. These outputs were manually

entered in an *Excel™* spreadsheet for both placebo and experimental conditions where the percent decrement formula was used to calculate fatigue scores. AMPO was calculated by taking the MPO from each sprint and averaging that across the experimental trial for each participant. Afterwards, a grand mean of all participants was calculated. APPO was calculated the same way AMPO was calculated. This process was done for both placebo and experimental conditions. ADBL was first calculated by finding the difference between post and pre blood lactate values before averaging all participants across a condition. This process was done for both placebo and experimental conditions. AHR and ARPE were calculated by taking either variable and averaging that across a condition for each participant before averaging all participants across a condition.

## *Data Analysis*

All statistics were performed using the SPSS software. Descriptive data (means, standard deviations) were calculated for all demographic (age, height, weight) data. In addition, descriptive data was calculated for all dependent variables (AMPO, APPO, ADBL, AHR, ARPE, and fatigue scores). A One-way ANOVA and a paired sample t-test was used to evaluate differences in dependent variables across placebo and experimental conditions. An alpha of 0.05 was used for all analyses.

## **CHAPTER IV**

## **RESULTS**

The objective of this study was to investigate the acute effects of sodium bicarbonate ingestion during a repeated-sprint cycling protocol in college-aged males. This chapter presents data on the following: Average Mean Power Output (AMPO), Average Peak Power Output (APPO), Average Delta Blood Lactate (ADBL), Average Heart Rate (AHR), Average Rating of Perceived Exertion (ARPE), and fatigue. Continue to next page to view *Table 1.*

Below, you will find the results of the dependent variables in *Table 1*.



## *Table 1 Results of dependent variables*

*Note. No statistical significance was found at the p < 0.05 level.*

The one-way ANOVA revealed no significant difference for both AMPO and APPO. The mean and standard deviation for AMPO in the placebo vs experimental conditions were 907.44  $\pm$  166.86 and 921.10  $\pm$  162.91, respectively (f = 2.062, p = 0.37). The mean and standard deviation for APPO in the placebo vs experimental conditions were 1180.59  $\pm$  242.56 and 1196.34  $\pm$  239.46, respectively (f = 0.667, p = 0.737).

The paired sample t-test showed no significant difference for both ADBL and fatigue. The mean and standard deviation values for ADBL in the placebo vs. experimental conditions were 8.03  $\pm$  1.84 and 9.13  $\pm$  3.05 mmol/L, respectively (t = -1.856, p = .113). The mean and standard deviation for fatigue (*Figure 2*) in the placebo vs. experimental conditions was  $10.48 \pm 3.17$  and  $9.88 \pm 4.28$  percent, respectively (t =

.780, p = .461). *Table 2* presents pre and post measurement of lactate data.

# *Table 2 Results of Pre and Post Lactate Values*



*Note. Actual blood lactate measurements before and after exercise for both conditions.*

The paired sample t-test revealed no significant difference for both AHR and ARPE. The mean and standard deviation for AHR in placebo vs experimental conditions was  $161.89 \pm 6.94$  and  $164.00 \pm 8.11$  beats per minute, respectively (t = -1.202, p = 0.268). The mean and standard deviation for ARPE in the placebo vs experimental conditions was 15.25 ± 1.06 and 14.93 ± 1.15, respectively. (t = 1.075, p = 0.318). *Figure 2* shows a visual representation of the relationship between fatigue and percent decrement.



*Figure 2.* Fatigue and Percent Decrement. The relationship of placebo and experimental conditions to percent decrement.

### **CHAPTER V**

### **DISCUSSION, FUTURE RECOMMENDATIONS & CONCLUSION**

### *Discussion*

The current study investigated the effects of acute ingestion of sodium bicarbonate on repeated-sprint ability in recreationally active, college-aged males. This was assessed by examining average mean power output (AMPO), average peak power output (APPO), average delta blood lactate concentration (ADBL), average heart rate (AHR), average rating of perceived Exertion (ARPE), and fatigue scores between groups. The one-way ANOVA showed no significant difference for AMPO, APPO and fatigue scores during the present study.

In contrast to the current study's findings, a study investigating the acute cosupplementation of creatine and sodium bicarbonate found an increase in mean and peak power outputs (Barber, McDermott, McGaughey, Olmstead, & Hagobian, 2013). After a designated warmup, participants completed six Wingate sprints each lasting 10 seconds. 60 seconds of active recovery was allotted between sprints where participants pedaled at 50 watts. There were three groups involved in the study: a placebo group, a

creatine group, and a creatine + sodium bicarbonate group. The supplementation period lasted two days. For the creatine + sodium bicarbonate group, 20 grams of creatine, and 0.5 g/kg of sodium bicarbonate were consumed (Barber et al., 2013). After analyzing differences in data between creatine and placebo groups, the investigators noticed a 4% increase in peak power over placebo when creatine was consumed. Even more surprising, a 7% increase in peak power was seen when both creatine and sodium bicarbonate were ingested together compared to placebo (Barber et al., 2013). The study also showed that peak power declined more slowly over the repeated sprint protocol when these supplements were ingested together. It could be argued that the additional increase in performance was caused by sodium bicarbonate's unique buffering capabilities. Sodium bicarbonate seems to have more of an advantageous effect when hydrogen ion concentration is at its peak (Barber et al., 2013). The ingestion of sodium bicarbonate increases its intracellular concentration essentially forcing these ions out of the muscle helping to decrease fatigue brought on by exercise (Barber et al., 2013).

An explanation as to why the participants in the current study showed no difference in performance could be due to the amount of sodium bicarbonate ingested. Barber et al. (2013), mentioned that during their creatine + sodium bicarbonate study, the participants supplemented sodium bicarbonate over a 48-hour time period with 0.5 g/kg sodium bicarbonate being ingested during each day via ingestion of 4 equal doses. On testing day, no supplements were consumed (Barber et al., 2013). As mention before, research demonstrates that 0.3 g/kg increases the concentration of bicarbonate

in the body to about 5 or 6 mmol/L<sup>-1</sup> (Siegler et al., 2012). This specific amount has been deemed an optimal quantity to yield a "blood-buffering pool" to combat the additional protons released during high intensity exercise (Siegler et al., 2012). During the present study, 0.3 g/kg were ingested versus the 1.0 total grams ingested (Barber et al., 2013). Perhaps a larger amount of sodium bicarbonate is still needed to have a significant effect on performance. It is plausible that sodium bicarbonate did not induce a level of alkalinity necessary to improve performance in this specific population. This would seem unlikely since 0.3 g/kg of sodium bicarbonate has been shown to be an optimal quantity to consume (Siegler et al., 2012), but this still cannot be ruled out since neither plasma pH nor plasma bicarbonate levels were measured in the present study.

One might argue that the time over which sodium bicarbonate was ingested could be a potential explanation for their performances. Barber et al., (2013) had their participants essentially "load" over the course of 48 hours by ingesting eight total doses. During the present study, only 60 minutes was allotted between ingestion and time of exercise. It could be that these differences in dose timing may have impacted exercise performance. One study investigating dose timing of sodium bicarbonate determined that dose timing might not impact performance (Siegler et al., 2012). Eight males engaged in three sprinting trials of which they ingested 0.3 g/kg sodium bicarbonate at 60, 120, and 180 minutes before exercise. Ten sprints lasting ten seconds each were performed on a nonmotorized treadmill. Their study concluded that supplement timing does not affect sport performance (Siegler et al., 2012). From that investigation, we can obtain more clarity that supplement timing might impact sport performance after all.

An acute dose taken just hours before exercise may not facilitate increases in sport performance however, "loading" sodium bicarbonate over the course of several days could show positive results relating to repeated sprint ability. Perhaps two days of loading would have a greater effect on performance than would a dose taken 60-180 minutes before exercise.

Cameron et al., (2010) had an interesting study in which some outcomes were very similar to the current investigation. As mentioned previously, 25 male rugby players ingested 0.3 g/kg sodium bicarbonate or placebo 65 minutes before their warmup (Cameron et al., 2010). Afterwards, a Rugby Specific Repeated Sprint Test (RSRST) was conducted in which the athletes performed 10 x 40-meter sprints interspersed with 30 seconds rest (Cameron et al., 2010). During their study, the investigators noticed no increase in performance. The investigators hypothesized that there was no increase in performance due to the overwhelming number of participants who had gastrointestinal upset. The stomach upset included but was not limited to diarrhea and vomiting. During the present study, only two of the nine participants had GI upset. One of those two individuals who had GI upset could no longer participate due to significant stomach discomfort and vomiting. Therefore, GI upset was not a major limiting factor relating to performance during the present study however, GI discomfort was still observed. It is important to note that these undesirable GI symptoms could affect performance during an actual sporting event, along with their mental preparation and recovery (Cameron et al., 2010).

The study conducted by Cameron et al., (2010) had other results similar to the present study. Cameron et al., (2010) stated that the athletes in their study were highly trained individuals. The investigators postulated that since the participants were highly sprint trained, they more than likely already had advanced buffering capabilities and therefore, supplementing with sodium bicarbonate would have little to no effect (Cameron et al., 2010). The rugby players are more likely to fatigue from other variables other than acidosis as a direct cause of a decrease in sport performance (Cameron et al., 2010). Interestingly, Cameron et al., (2010) helped to clarify what might be taking place during the present study. Perhaps a similar effect took place in the present study as well, where there may be a relationship between training status and performance. The possibility exists, particularly related to the training status, that the participants in the present study may have possessed well-developed buffering capabilities, thereby potentially nullifying the ergogenic effect of sodium bicarbonate. Surprisingly, it should be noted that the creatine + sodium bicarbonate study used trained males as well, yet their performances had increased (Barber et al., 2013). These athletes partook in greater than 5 hours of weekly training and greater than two hours per week of high intensity exercise (Barber et al., 2013), and therefore were well trained just as the elite rugby players were. This does add some conflicting views because the trained rugby athletes had not increased in their performances, but the athletes in the creatine + sodium bicarbonate study had improved in their performances. However, the ingestion of creatine was still consumed simultaneously with sodium bicarbonate and the creatine may have influenced the outcomes of their study in some way. Also, much more

sodium bicarbonate was consumed during the creatine + sodium bicarbonate study than was consumed during the rugby study, or during the present study. This further enhances the fact that the amount of sodium bicarbonate consumed may not have been enough to yield ergogenic effects during the present study.

As previously mentioned, there could be a plausible link between sport performance and training status and whether sodium bicarbonate would be an effective ergogenic aid. In a different study, moderately trained males underwent two, 30 minute intermittent cycling tests where the participants engaged in 10 x 3-minute bouts of exercise pedaling at various intensities (Price, Moss, & Rance, 2003). The amount of 0.3 g/kg sodium bicarbonate or placebo was ingested 60 minutes before testing. The results concluded that participants were better able to replicate their sprint performances even during the latter stages of the test (Price et al., 2003). The investigators mentioned that these individuals were moderately trained and engaged in three bouts of aerobic exercise weekly (Price et al., 2003). The previous study mentioned contained trained males (Barber et al., 2013) Correspondingly, the previous study that recruited rugby players were also highly trained, and exercised eight hours weekly (Cameron et al., 2010). Four of those eight hours were high intensity exercise (Cameron et al., 2010). Yet, the trained individuals from both studies had not increased their sport performances. This further enhances the argument that training status may have been a determining factor in whether sodium bicarbonate was useful during repeated sprints. From the previous studies mentioned a few conclusions can be made. Perhaps, moderately trained individuals with less efficient buffering systems benefit

most from 0.3 g/kg of sodium bicarbonate ingestion. As individuals become more highly trained with advanced buffering capabilities the possibility that 0.3 g/kg not promoting the level of alkalinity necessary to see distinguishable increases in sport performance could exist.

Different mechanisms are thought to contribute to fatigue that would explain the decline in force production during repeated sprint activities (Miller et al., 2016). Other potential possibilities other than acidosis being the direct cause of fatigue could be due to many different factors such as, but not limited to, a decline in energy supply, ionic disturbances, and the increase of inorganic phosphate (Miller et al., 2016). These other potential causes may help to explain the reason that the athletes and the participants in the current study had shown no increases in exercise performance regarding repeated-sprint ability.

It should be noted that AHR, ADBL, and ARPE also did not show any significant differences between conditions during the present study. This supports the fact that sodium bicarbonate did not have an ergogenic effect as one would expect to find a decrease in DBL with sodium bicarbonate supplementation, however this was not the case. It can also be inferred that since AHR and ARPE were unchanged during the present study, it seems likely that participants were giving equal effort during both conditions. This further supports the fact that sodium bicarbonate did not have any ergogenic effects.

### *Future Recommendations*

If the current study were to be conducted again, the following recommendations are suggested:

- 1. A larger sample size is needed to increase the statistical power.
- 2. Untrained males should be recruited to remove the possibility that training status may have affected results. Perhaps untrained males would benefit more from this supplement rather than trained males.
- 3. A test to determine time to exhaustion through Function Electrical Stimulation (FES) equipment may be utilized as an alternative test variable of total work produced throughout each sprint.
- 4. During the ingestion of either sodium bicarbonate or placebo some participants felt as if the disguising agent was not strong enough. The participants felt as if they could use the process of elimination to figure out what they had consumed during their experimental trials. For future studies, perhaps more of the disguising agent or a different disguising agent should be used.

## *Conclusion*

The ingestion of 0.3 g/kg sodium bicarbonate demonstrated no significant difference for AMPO, APPO, ADBL, AHR, ARPE, and fatigue compared to placebo. Many variables contributing to the outcome of the study may have influenced the results. Amount of supplement ingested, and training status of participants appear to be likely to explain the lack of effect of the sodium bicarbonate ingestion. It could be that

acidosis as a direct cause of fatigue may only play a minimal role in fatigue and diminishment of sport performance during repeated sprint activities in this population. Further research is suggested.

#### **REFERENCES**

Carr, A., Slater, G., Gore, C., Dawson, B., Burke, L. (2011). Effect of Sodium Bicarbonate on [HCO3-], pH, and Gastrointestinal symptoms. *International Journal of Sport Nutrition and Exercise Metabolism*, 21(3), 189-194.

Barber, J., McDermott, A., McGaughey, K., Olmstead, J., & Hagobian, T. (2013). Effects of Combined Creatine and Sodium Bicarbonate Supplementation on Repeated Sprint Performance in Trained Men. *The Journal of Strength & Conditioning Research*, 27(1), 252-258.

- Bellinger, P., Howe, S., Shing, C., Fell, J. (2012). Effect of Combined Beta Alanine and Sodium Bicarbonate Supplementation on Cycling Performance. *Medicine & Science in Sports & Exercise,* 44, 1545-1551.
- Bishop, D., Girard, O., Mendez-Villanueva, A. (2011). Repeated-Sprint Ability Part II: Recommendations for Training. *Sports Med,* 41(9), 741-756.
- Cameron, S., McLay-Cooke, R., Brown, R., Gray, A., Fairbairn K. (2010). Increased Blood pH but Not Performance With Sodium Bicarbonate Supplementation in Elite Rugby Union Players. *International Journal of Sport Nutrition and Exercise Metabolism*, 20(4), 307-321.

Danaher, J., Gerber, T., Wellard, M., Stathis, C. (2014). The Effect of Beta-alanine and NaHCO<sub>3</sub> co-ingestion on buffering capacity and exercise performance with highintensity exercise in healthy males. *European Journal of Applied Physiology*, 114(8), 1715-1724.

- Ducker, K., Dawson, B., Wallman, K. (2013). Effect of Beta Alanine and Sodium Bicarbonate supplementation on Repeated-Sprint performance. *The Journal of Strength and Conditioning Research*, 27(12), 3450-3460.
- Garber, C., Blissmer, B., Deschenes, M., Franklin, B., Lamonte, M., Lee, I., … Swain, D. (2011). Quantity and Quality of Exercise for Developing and Maintaining Cardiorespiratory, Musculoskeletal, and Neuromotor Fitness in Apparently Healthy Adults: Guidance for Prescribing Exercise. *Medicine & Science in Sports & Exercise,* 43(7), 1334-1359.
- Girard, O., Mendez-Villanueva, A., and Bishop, D. (2011). Repeated-Sprint Ability Part I: Factors Contributing to Fatigue. *Sports Med*, 41(8), 673-694.
- Glaister, M., Stone, M., Stewart, A., Hughes, M., & Moir, G. (2004). The relationship and validity of fatigue measures during short duration maximal-intensity intermittent cycling. *Journal of Strength and Conditioning*, 18, 459-462.
- Gough, L., Rimmer, S., Osler, C., Higgins, M. (2017). Ingestion of Sodium Bicarbonate (NaHCO3) Following a Fatiguing Bout of Exercise Accelerates Postexercise Acid-Base Balance Recovery and Improves Subsequent High-Intensity Cycling Time to Exhaustion. *International Journal of Sport Nutrition and Exercise Metabolism*, 27(5), 429-438.
- Hobson, R., Harris, R., Martin, D., Smith, P., Macklin, B., Gualano, B., Sale, C. (2013). Effect of Beta-Alanine With and Without Sodium Bicarbonate on 2,000-m Rowing Performance. *International Journal of Sport Nutrition and Exercise Metabolism,* 23, 480-487.

McNaughton, L., Gough, L., Sanjoy, D., Bentley, D., Sparks, A. (2016). Recent Developments in the Use of Sodium Bicarbonate as an Ergogenic Aid. *American College of Sports Medicine*, 15(4), 233-244.

- Miller, P., Robinson, A., Sparks, A., Bridge, C., Bentley, D., McNaughton, L. (2016). The Effects of Novel Ingestion of Sodium Bicarbonate on Repeated Sprint Ability. *Journal of Strength and Conditioning,* 30(2), 561-568.
- Powers, S. K. & Howley, E. T. (2012). *Exercise Physiology: Theory and Application to Fitness and Performance*. New York, NY. McGraw Hill Companies.
- Price, M., Moss, P., Rance, S. (2003). Effect of Sodium Bicarbonate Exercise on Prolonged Intermittent Exercise. *American College of Sports Medicine,* 35(8), 1303-1308.
- Robergs, R., Ghiasvand, F., & Parker, D. (2004). Biochemistry of Exercise-Induced Metabolic Acidosis. *American Journal of Physiology Regulatory Integrative and Comparative Physiology,* 287, 502-516.
- Sale, C., Saunders, B., Harris, R. (2010). Effect of beta-alanine supplementation on muscle carnosine concentrations and exercise performance. *Amino Acids,* 39(2), 321-333.
- Sale, C., Saunders, B., Hudson, S., Wise, J., Harris, R., Sunderland, C. (2011). Effect of Beta Alanine and Sodium Bicarbonate on High-Intensity Cycling Capacity. *Medicine & Science in Sports & Exercise,* 43(10), 1972-1978.

Siegler, J., Marshall, P., Bray, J., Towlson, C. (2012). Sodium Bicarbonate

Supplementation And Ingestion Timing: Does It Matter?. *Journal of Strength and Conditioning*, 26(7), 1953-1958.

Spencer, M., Bishop, D., Dawson, Brian., Goodman, C. (2005). Physiological and Metabolic Responses of Repeated Sprint Activities: Specific to Field-Based Team Sports. *Sports Med,* 35(12), 1025-1044.

**APPENDICES** 

#### **APPENDIX A**

#### **IRB APPROVAL PAGE**



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## **APPENDIX B**

## **INFORMED CONSENT**

# **Title: Acute Sodium Bicarbonate Supplementation on Fatigue and Power Output During a Repeated-Sprint Ability Cycling Protocol**

- 1. Matt Kelchner, a graduate student in the Department of Exercise Science, has requested my participation in a research study at East Stroudsburg University. The title of the study called: *Acute Sodium Bicarbonate Supplementation on Fatigue and Power Output During a Repeated-Sprint Ability Cycling Protocol.*
- 2. I (the participant) have met the requirements for participation in this study for age, sex, and activity and health statuses. I been informed that the purpose of the study is to investigate the effects on supplementation that sodium bicarbonate has on muscular fatigue and power outputs during a repeated sprint cycling protocol (10 x 6 second sprints interspersed with 30 seconds recovery) using a cycle ergometer.
- 3. My participation in this study will involve the oral acute ingestion of sodium bicarbonate 60 minutes before the final testing trial begins. Prior to supplementation, I will first participate in two familiarization sessions so I can get comfortable with the cycling protocol and procedures. During these familiarization sessions, the data gathered from them will be used to calculate fatigue scores using the percent decrement method. After the two familiarization trials are completed, the  $3<sup>rd</sup>$  session (also known as experimental trial 1) will be used to gather baseline data. Following baseline testing, I will then participate in the  $4<sup>th</sup>$  and final session (experimental trial 2). The last session is essentially a post-testing session to evaluate post supplementation data. Prior to any of the 4 testing days I should be 60 minutes post absorptive. I must eat similar diets on testing days. Download *MyFitnessPal* to log previous meals to eat similar meals on testing days. If I choose to participate, I will sign the consent form and have full understanding of my responsibilities.
- 4. I understand there are no more than minimal risks or discomforts associated with physical activity if I agree to participate.
- 5. Benefits of my participation helps myself to get a better inside look at graduate thesis work, and helps the research conduct an important study on sodium bicarbonate to investigate whether or not it improves athletic performance.
- 6. I understand there are no feasible alternative procedures for this study.
- 7. I understand that the results of the research study may be published but my name and my identity will not be revealed. In order to maintain confidentiality of my records, Matt Kelchner and his committee chair are the only people with access to the records.
- 8. I have been informed that I will not be compensated for my participation and I may withdraw at any time without penalty or loss of benefit.
- 9. If I have questions regarding the research project or my participation in it, I can contact Matt Kelchner at (484) 619-4802. If I have concerns or I feel I was placed at risk, feel free to contact Dr. Chad Witmer at (570) 422-3362.

I have read the above information. I was given as much time as I needed to read this consent form to gain full understanding of the study. The nature, demands, risks, and benefits of the project have been explained to me. I knowingly assume the risks involved and understand in signing this consent form I am not waiving any legal claims, rights or remedies. A copy of this consent form will be given to me. This consent form was reviewed and signed in the presence of Matt Kelchner, the research investigator.

Participant's Signature: Department of the state of t

Date: \_

Investigator's Signature: \_\_\_\_\_\_

Date: \_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_

## **APPENDIX C**

### **ACTIVITY AND DEMOGRAPHIC QUESTIONNAIRE**

**Participant Name:** 

**Directions: Check the appropriate box as it relates to you. If "YES" check "YES". If** 

**"NO" check "NO".**

**YES NO**

 $\Box$  am a recreationally trained male between the ages of 18 and 30.

(Recreationally trained is identified as an individual who participates in moderate to vigorous intensity exercise 3 to 5 days a week for 75-150 minutes weekly).

**YES NO**

 $\Box$  I am apparently healthy, free of any musculoskeletal injuries, and have not had any major surgery within the previous 6 months prior to testing.

## **YES NO**

 $\Box$   $\Box$  Participants must be free of any gastrointestinal issues such as: Irritable Bowel Syndrome (IBS) or Inflammatory Bowel Disease (IBD) including Crohn's and Colitis due to supplement ingestion which may be unhealthy for the participant with these conditions.

## **YES NO**

 $\Box$   $\Box$  I have not ingested any ergogenic supplements 30 days prior to study participation. (Reminder: Any additional ergogenic supplementation ingestion should be withheld throughout the duration of the study).

Participant's Signature:

Date: \_\_\_\_\_\_

Investigator's Signature: 1988

Date:

### **APPENDIX D**

## **DATA COLLECTION SHEET**

**Participant Name:\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_ Age:\_\_\_\_\_ Height (m):\_\_\_\_\_ Weight (kg):\_\_\_\_\_ BMI:\_\_\_\_\_**









**Notes:\_**

**\_**

**\_**

### **APPENDIX E**

Physical Activity Readiness Questionnaire - PAR-Q<br>(revised 2002)



#### (A Questionnaire for People Aged 15 to 69)

Regular physical activity is fun and healthy, and increasingly more people are starting to become more active every day. Being more active is very safe for most people. However, some people should check with their doctor before they start becoming much more physically active.

If you are planning to become much more physically active than you are now, start by answering the seven questions in the box below. If you are between the ages of 15 and 69, the PAR-Q will tell you if you should check with your doctor before you start. If you are over 69 years of age, and you are not used to being very active, check with your doctor.

Common sense is your best guide when you answer these questions. Please read the questions carefully and answer each one honestly: check YES or NO.



or GUARDUN (for participants under the age of majority)

GSB<sup>9</sup>



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