AN ABSTRACT OF THE THESIS OF

Lori-Ann Avedisian for the degree of Doctor of Philosophy in Human Performance presented on May 1, 1995. Title: The Effect of Selected Buffering Agents on Performance in the Competitive 1600 Meter Run.

Abstract approved: Redacted for Privacy
Anthony Wilcox

During performance of activities that emphasize anaerobic glycolysis to generate energy, a reduction in performance is generally associated, in part, with lactacidosis. As lactate levels rise during the performance of the activity, pH and bicarbonate levels decrease. Strategies designed to buffer lactic acid (LA) have demonstrated ergogenic potential for both sodium bicarbonate (NaHCO3) and sodium citrate during activities ranging from 1 to 2 minutes' in duration. The purpose of the present study was to determine if these buffering agents exhibited ergogenic potential during high intensity activity of 4 to 6 minutes duration. Subjects were 7 male and 5 female trained track athletes between the ages of 18 and 33. Each subject participated in a total of 4 competitive 1600 meter races, each scheduled at least 3 days apart. Subjects ingested a treatment (0.4 g/kg NaHCO3 or 0.5 g/kg sodium citrate) or placebo (calcium carbonate) 2 hours prior to three of the races; one race was used as a control. The order in which the races were run was counter-balanced and randomly assigned. Blood

lactate, pH, and bicarbonate levels were measured prior to and immediately following each race. Following the completion of the race, the subjects reported on physical symptoms of gastrointestinal (GI) discomfort they experienced during the run. Means and standard deviations for performance times (in sec) were not different under any condition: HCO3 (319.2, 30.1); Citrate (321.1, 24.1); Placebo (319.9, 27.3); Control (319.8, 27.0). There was an exercise, but no treatment effect on blood lactate, but there was an exercise and treatment effect on pH and bicarbonate. NaHCO3 ingestion resulted in more severe symptoms of GI discomfort than any other condition, and ingestion of sodium citrate led to the greatest number of complaints of GI discomfort. It is concluded that the buffering agents had no effect on racing time and that bicarbonate loading is associated with uncomfortable side effects in many athletes.

Funded in part by the Department of Exercise and Sport Science, Oregon State University.

The Effect of Selected Buffering Agents on Performance in the Competitive 1600 Meter Run

by

Lori-Ann Avedisian

A THESIS

submitted to

Oregon State University

in partial fulfillment of
the requirements for the
degree of

Doctor of Philosophy

Completed May 1, 1995
Commencement June 1995

Doctor of Philosophy thesis of Lori-Ann Avedisian presented on May 1, 1995

APPROVED:

Redacted for Privacy

Major Professor, representing Exercise and Sport Science

Redacted for Privacy

Chair of Department of Exercise and Sport Science

Redacted for Privacy

Dean of Graduate School

I understand that my thesis will become part of the permanent collection of Oregon State University libraries. My signature below authorizes release of my thesis to any reader upon request.

Redacted for Privacy

Lori-Ann Avedisian, Author

Acknowledgment

I wish to express my sincere gratitude to CJ for all the patience, support, love, encouragement, and energy that was invested in this endeavor, I could have never done it without you. I wish to extend my thanks to Anthony Wilcox, Ph.D., for his invaluable time, assistance, and expertise throughout the course of this study. Special thanks is extended to Susan Fox for her assistance in data collection; Nick Cirlincione and his staff for their willingness and technical expertise with regard to the analysis of blood samples; and George Oja for his willingness to help in any way. I wish to thank all of the faculty and staff in the school of Physical Therapy at Pacific University for accommodating my needs whenever possible - this made this adventure go as smoothly as possible. Special thanks go out to Richard Rutt, Ph.D. for everything he did to make my life easier during the past two years. I am indebted to all the subjects who gave freely of their time for the study. I can never thank them enough for their participation. I would also like to thank my family and friends for their support and encouragement, and everyone who directly or indirectly helped me to achieve this goal.

TABLE OF CONTENTS

TABLE OF CONTENTS (Continued)

LIST OF TABLES

LIST OF APPENDICES

CHAPTER I

INTRODUCTION

Tsaneva and Markov (1971) described fatigue as a process and defined this process as a complex phenomenon caused by the working state of a biological system, influenced by many external and internal factors. Grandjean and Kogi (1971) referred to fatigue as a group of phenomena associated with impairment or loss of efficiency in the performance of a skill, as well as the development of anxiety, frustration, or boredom. Bates, Osternig, and James (1977) attempted to define fatigue as a process that affects the organization of movement, while Edwards (1981) defined it as a failure to maintain an expected force output. Schmidt (1968) described fatigue as a performance variable which temporarily decreases the level of proficiency of the performance. All these studies agree that a decrement in performance of the skill occurs with fatigue.

During activities of daily living, fatigue simply represents an irritant. It may necessitate a reduction in or stoppage of the activity in order to recover from the preceding work. However, with the exception of disease states, it does not have prolonged negative consequences. In the realm of competitive athletics however, success is determined by performance outcomes. An individual whose performance is hindered by fatigue may discover that he or she loses an important race, loses a position

on a team, loses some type of award, and, in some instances, it represents economic loss. Under these circumstances, it is not surprising that methods to delay the onset of fatigue during all types of sport are being extensively researched.

The research can be differentiated by the type and intensity of the exercise being performed. Different methods to replenish adenosine triphosphate (ATP) and delay the onset of fatigue are used during exhaustive exercise activities as compared to endurance activities. ATP is the main source of fuel that the body utilizes, and different metabolic pathways exist to generate energy necessary to power the activity. The pathways which are emphasized depend on the functional demands of the situation. Therefore, the mechanism of fuel delivery and fuel supply used during high intensity anaerobic activities differs greatly from that of sub-maximal aerobic activities.

During brief exhaustive activity, the energy supply comes from the breakdown of nutrients stored within the muscle fiber. This is referred to as the ATP-creatine phosphate (CP) system. The configuration of this system permits rapid production of ATP, but it is not capable of supplying the energy required for most activities. Since the CP supply and storage capacities of CP are limited, energy becomes depleted in a matter of a few seconds.

During heavy exercise exceeding a few seconds in duration and lasting up to approximately three minutes in duration, anaerobic glycolytic processes predominate to supply the energy. This involves the incomplete combustion of carbohydrates

obtained via dietary intake. During this breakdown process, lactic acid (LA) is produced. Lactic acid is a strong acid which can accumulate in the muscle. Excessive accumulation of LA has been implicated in the onset of fatigue and reduction in performance in this type of activity. When the rate of LA production exceeds the rate of LA clearance, LA accumulates. LA accumulation in the muscle may interfere with the contractile mechanism of the muscle (Mainwood and Worsley-Brown, 1975; Linderman and Fahey, 1991), it may stimulate pain receptors, and it may slow glycolysis (Goldfinch, McNaughton, and Davies, 1988). All of these effects can lead to a decrease in performance.

During prolonged activity, multiple metabolic pathways can be involved in the production of ATP. Although a considerable amount of ATP can be generated via these pathways, it cannot be resynthesized as quickly. Also in this system, LA does not accumulate, as it is cleared as quickly as it is produced. For activities that are neither purely anaerobic in nature, nor prolonged sub-maximal aerobic work, such as the 1600 meter race, a blending of metabolic pathway activity occurs to supply energy. Performance of this event, which is classified as a sustained speed event, results in LA accumulation. It is the LA accumulation which appears to be a major contributor to the reduction in performance during activities of this type.

Fox and Mathews (1981) suggest that in a race of this distance, anaerobic glycolytic pathways predominate by contributing approximately 55% to the total energy production.

Oxidative pathways contribute approximately 25%, and stored phosphagens the remaining 20%. In an event performed at this level of intensity for a duration of 4-6 minutes, increasing levels of LA in the muscle and blood become a factor in the performance. Since the intensity of the performance easily reaches 100% $\dot{V}O_2$ max during this type of racing situation, LA accumulates (Farrell, Wilmore, Coyle, Billing, and Costill, 1979).

For each of these situations, extensive volumes of research have focused on methods to delay the onset of fatigue. The rationale being that if one is able to postpone the onset of fatigue, the athlete would be able to perform at that higher intensity for a longer portion of the event. This may enable the participant to complete the event before succumbing to fatigue. Methods and techniques designed to enhance performance are referred to as ergogenic aids. This term is broad and may incorporate the use of procedures, use of exogenous substances and nutritional supplements, manipulation of the normal state of the energy systems within the body, or manipulation of the normal physiological response to the activity in order to enhance performance.

Specificity and intensity of training is one method which may serve to enhance performance in activities emphasizing anaerobic glycolysis. Additionally, the body has a number of inherent buffering systems which function to assist in neutralizing the strong acids and postpone the presence of the acidic environment. Of these, the bicarbonate ion (HCO_3) system is the major buffer in the blood (Parry-Billings and MacLaren,

1986). This carbonic acid-bicarbonate system functions effectively in the body to buffer lactic acid. If the hydrogen ion concentration increases in the blood, which is observed when LA accumulates, a corresponding decrease in bicarbonate ion concentration is noted. This is a result of more carbonic acid being formed. This buffering system functions to assist in maintaining the acid-base relationship near an optimal point, which is critical for homeostasis.

In situations such as heavy exercise, activity of these inherent buffering systems alone may not be adequate. Ingestion of substances such as sodium bicarbonate (NaHCO3) or sodium citrate has been utilized by athletes during exercise in an attempt to enhance the buffering of LA. It is thought that by preventing acidosis brought on by the formation of LA, one is able delay the onset of fatigue. Results from several studies suggest that buffering may function effectively as an ergogenic aid during heavy exercise lasting from one to four minutes in duration (Wilkes, Gledhill, and Smyth, 1983; Goldfinch et al., 1988). In these studies of 400 and 800 meter running races, participants were able to demonstrate faster run times following ingestion of a substance which had an alkalizing effect on the blood.

Results from studies involving events of less than 30 seconds duration suggest that in these types of events the amount of LA production had not peaked, and the maximal buffering capacity was not utilized to enhance performance (Inbar et al., 1983). Likewise, results from studies of continuous exercise lasting

longer than 10 minutes in which alkalizing substances were ingested were inconclusive (Johnson and Black, 1953). Plausible explanations for these results include, less reliance on anaerobic glycolysis to generate energy during the longer runs. Since oxidative processes predominate, significant LA accumulation may not have occurred, thus making buffering unnecessary.

The effects of ingestion of sodium bicarbonate, known as "bicarbonate loading" on anaerobic performance have been studied to a greater extent than have the effects of ingestion of citrate or "citrate loading". The results indicate that bicarbonate loading using a dose equal to or greater than 0.3 g/kg body mass may result in improved performance in events that emphasize the LA system to produce ATP (Kindermann, Keul, and Huber, 1977). However, the high doses necessary to enhance performance may also cause symptoms of gastrointestinal (GI) discomfort, such as diarrhea and nausea (Gledhill, 1984; Goldfinch, et al., 1988).

Although not studied extensively, citrate ingestion has also been suggested as an ergogenic aid for performance in high intensity events ranging from 100 to 400 meters (Hewitt and Calloway, 1936). Parry-Billings & MacLaren (1986) found citrate ingestion to be more effective than a comparable dose of bicarbonate in delaying fatigue associated with lactacidosis. Additionally, citrate loading has not resulted in the gastrointestinal distress noted with large doses of bicarbonate (McNaughton, 1990).

Level of training of the subjects, dosage administered, and point in time of ingestion prior to the competition are all factors

in the use of exogenous substances to enhance performance that appear to be crucial to the outcome. Trained individuals who ingest doses of sodium bicarbonate equal to or greater than 0.3 g/kg of body mass 90-120 minutes prior to the event enjoy the greatest potential for enhanced performance if the event is 1-4 minutes in duration (Bouissou, Estrade, Goubel, Guezennec, and Serrurier, 1989; Williams, 1992). In an effort to minimize the potential for GI distress, the sodium bicarbonate is frequently ingested with approximately one liter of water. McNaughton (1990) identified the sodium citrate dose with the greatest ergogenic potential as 0.5 g/kg for similar types of activity.

Due to the potential for undesirable symptoms of gastrointestinal distress when using certain buffering agents, it is important to determine if there is an ergogenic agent which is equally effective in this role without yielding the undesirable side-effects. The 1600 meter racing distance is a highly competitive event with performance outcomes carrying enormous consequences for some participants. With outcomes being determined by mere seconds, there is a strong desire and need to identify all the factors which can be influenced to lead to improved performance. These factors may include the determination of a balance between desirable improvements and undesirable side-effects. If one substance can demonstrate a decided advantage over others to accomplish this, it would be the ergogenic aid of choice.

Statement of Problem

The purpose of the present study was to determine the effects on performance in a competitive 1600 meter run following ingestion of selected buffering agents in trained track athletes. Previous studies suggest that in this event lactacidosis plays a major role and influences performance in part via fatigue. By minimizing the acidic environment, performance may be enhanced in this event.

The selected biochemical agents included:

1. sodium bicarbonate
2. sodium citrate
3. placebo (calcium carbonate)

The parameters selected for analysis included:

1. run time
2. blood lactate level
3. blood pH level
4. blood bicarbonate level
5. subjective symptom scale

Hypotheses

1. following ingestion of the buffering agents, performance will be significantly enhanced as measured by faster run times compared to the placebo and control conditions.

2. at the completion of the run, following ingestion of the buffering agents, blood pH levels will be significantly higher compared to the placebo and control conditions.

3. at the completion of the run, following ingestion of the buffering agents, blood lactate levels will be significantly higher compared to the other conditions.

4. at the completion of the run, following ingestion of the buffering agents, blood bicarbonate levels will be significantly higher compared to the placebo and control conditions.

5. ingestion of sodium citrate will result in significantly fewer gastrointestinal side-effect symptoms compared to sodium bicarbonate ingestion.

6. ingestion of sodium citrate will result in significantly faster race times than all other conditions.

Delimitations

The population chosen for analysis consisted of competitive male and female track athletes, ages 18 to 33. All male runners were able to complete a 1600 meter race in less than 5 minutes. Females were capable of completing this distance in less than 6 minutes.

Limitations

1. subjects were obtained via a sample of convenience and may not be representative of the track running population at large.
2. subjects may have made assumptions as to the substance ingested, which may have influenced performance.
3. subjects were not required to race during inclement weather conditions.
4. subjects were encouraged to maintain their normal routine concerning exercise, sleep, and eating habits in the 24 hours previous to data collection in attempt to control for fatigue and/or motivational level.

Definition of terms

Fatigue. Fatigue is defined in this study as an increase in the performance time of the 1600 meter run.

Alkalosis. Alkalosis is defined in this study as a venous pH blood value exceeding 7.35.

Buffering. Buffering is defined in this study as the chemical reactions that occur in an effort to minimize changes in the concentration of hydrogen ions.

Loading. Loading is defined in this study as ingestion of a substance which will alter the normal physiological response to exercise in a manner which may enhance performance.

$\dot{V}O_2max$ In this study, $\dot{V}O_2$ max is defined as the point at which oxygen consumption plateaus or decreases despite increasing workloads.

CHAPTER II

REVIEW OF LITERATURE

Metabolism

The performance of an activity requires the expenditure of energy. In order to accomplish this, an energy supply must be available. Adenosine triphosphate (ATP) is the fuel that humans utilize to power the muscular activity. There are several methods available within the body, capable of generating ATP. A relationship exists between the intensity and the duration of the exercise which dictates how the energy will be made available. During brief intense activity, the energy is supplied via stored nutrients found within the muscle fiber. These nutrients, ATP and another high energy phosphate, creatine phosphate (CP), are able to be used immediately as a source of energy. Because the nutrients are stored in the muscle fiber and only one biochemical step is required, this mechanism permits rapid ATP generation. Despite the speed of ATP production yielded from this process, the CP supply and storage capacities are limited. Activities of all out effort can only be sustained for approximately 5-6 seconds, as these CP stores become rapidly depleted (Williams, 1992).

Once the CP stores become depleted, a switch to other sources for the generation of energy must occur to supplement

the energy supply, or else the activity needs to terminate. Glycolytic pathways for energy production become active to supplement and allow for continuation of the activity. Anaerobic glycolysis involves the incomplete combustion of carbohydrates obtained via dietary intake. This allows for rapid production of ATP, but deleterious metabolic end products are formed. Increases in the concentration of the end product lactic acid (LA) result in increases in hydrogen ion (H+) concentrations (Kindermann et al., 1977; Goldfinch et al., 1988; Linderman & Fahey, 1991; Williams, 1992). During this breakdown process, the normal acid-base balance of the body is disrupted, resulting in acidosis. At rest, arterial blood tends to be in a neutral or slightly alkalotic state, with a pH of approximately 7.4 (Williams, 1992). At rest, venous blood is approximately 7.35, and muscle pH is approximately 6.9 (Linderman & Fahey, 1991). During heavy activity, they all become more acidic, and pH decreases.

Lactic acid is a strong acid which, under certain conditions, may accumulate in the muscle and blood. Along with the increased LA concentration, decreases occur in pH and bicarbonate (HCO3) concentration, attributable to the LA formed from the preceding exercise (Hermansen and Osnes, 1972). Excessive accumulation of LA has been implicated in the onset of fatigue and reduction in performance (Sutton, Jones, and Toews, 1981). Brooks (1985) indicates that when the rate of LA production exceeds the LA clearance rate, this metabolite accumulates. Numerous researchers (Asmussen, 1979; Linderman

& Fahey, 1991; McKenna, 1992; and Sahlin, 1992) indicate that with LA accumulation comes acidosis and a decrease in pH, which may stimulate pain receptors and interfere with muscle contractile protein function.

Reduction in excitation-contraction coupling secondary to decreased calcium (Ca++) activation has been suggested as a contributing factor to fatigue. Cross-bridge formation may be impaired, resulting in a decrease in the amount of tension produced. Fuchs, Reddy, and Briggs (1970) indicate that elevated H+ levels may compete with Ca++ for binding sites on the troponin molecule. Other researchers (Nakamaru and Schwartz, 1972; Fabiato and Fabiato, 1978) indicate that elevated H+ may alter the normal conformational structure of the tropomyosin molecule. They also suggest that this may decrease the number of actin and myosin cross-bridge interactions leading to a reduction in the amount of tension which can be developed. Goldfinch et al. (1988) report that this may slow glycolysis by inhibiting key enzyme activity. Activity of the rate limiting enzyme phosphofructokinase (PFK) has been demonstrated to decrease as H+ levels rise. This slows the velocity of ATP production. All these factors lead to a decrease in performance.

Sjogaard (1986) analyzed individuals during the performance of maximal and sub-maximal activities. The individuals participated in various activities involving the lower extremities, and specific ionic and water levels were evaluated for change from the pre-test to post-test condition. Blood was collected from the femoral vein. During the maximal condition in

all activities, a decrease in muscle pH and accumulation of LA was noted, and a slowing of glycolysis occurred. Homeostasis was disrupted, causing the force-generating capacity of the muscle to become temporarily inadequate. When this happens, activity must either decrease in intensity or terminate.

Natural buffering systems exist within the body to help neutralize the strong acids produced during metabolism. Buffering refers to the chemical reactions which occur to minimize increases in hydrogen ion concentration. A buffering system consists of a weak acid and the salt of that acid. During heavy exercise, LA accumulates in the muscle, creating an acidic environment. Although there are some mechanisms within the muscle to assist in buffering H+, most of the buffering occurs within the extracellular fluid component of the body.

Extracellular buffering serves to alkalize the bodily fluid and enhance the lactate and H+ efflux from the muscle into the blood (Goldfinch et al., 1988). As this happens, the blood bicarbonate (HCO3) levels decrease proportional to the increasing H+ levels. When H+ combines with HCO3 during the buffering process, carbonic acid (H2CO3) is formed. Carbonic acid breaks down into the end products of carbon dioxide (CO2) and water (H2O) in the pulmonary capillaries. Although this additional amount of CO2 is eliminated into the atmosphere by the lungs, there comes a point at which this system is unable to fend off the developing acidosis (Barr, Himwich and Green, 1923). The hydrogen ion level increases to a level which is greater than what the bicarbonate stores can buffer.

During these instances, the activity of the natural systems is not adequate to effect the alkalotic changes needed to maintain homeostasis. The bicarbonate reservoir may not be adequate to buffer all of the LA formed. Athletes frequently utilize a technique called "bicarbonate loading" to supplement this process. This technique requires ingestion of buffering substances designed to increase blood bicarbonate. This enhances the efflux of H+ and lactate from the muscle to the blood. Since the muscle membrane is relatively impermeable to sodium bicarbonate, buffering primarily occurs in the extracellular fluid of the body (Robin, 1961; Goldfinch et al., 1988). Ingestion of sodium bicarbonate creates a larger pH gradient across the membrane, which facilitates the H+ efflux. By increasing the bicarbonate reservoir prior to exercise, blood buffering potential is increased (Mainwood & Worsley-Brown, 1975). By increasing blood pH and postponing the development of the acidic environment, the disruption in contractile activity, slowing of glycolysis, and generation of pain can be delayed. This translates into increased endurance, increased power, greater work rates accomplished, and delaying of fatigue (Costill, Verstappen, Kuipers, Janssen, and Fink, 1984).

During the performance of a competitive 1600 meter running race, energy is supplied via a combination of anaerobic and oxidative processes (Fox & Mathews, 1981). Since metabolism via anaerobic glycolysis contributes approximately 55% of the energy generated, LA accumulation can be a factor in the performance. During the performance of this event, runners

approach their VO2max and easily exceed the point at which LA begins to accumulate (Farrell et al., 1979). When this happens, it contributes to the onset of fatigue and decrease the performance ability (Haverty, Kenney, and Hodgson, 1988).

Fatigue

Fatigue represents a reduction in performance secondary to the preceding work performed. It reflects a decrease in the energy supply necessary to meet the metabolic demands of the activity, an increase in metabolic end products, activation of central processes such as frustration and boredom, staleness, or decreased motivation, or a combination of all these factors. The central parameters involve a subjective component which may impact the outcome of the performance. Examples of these subjective parameters include: mood states, perceived exertion, and pain threshold. Asmussen (1979) reports that central fatigue results in lowered arousal and a reduction in the amount of voluntary effort output. Kirkendall (1990) relates it to a change in the level of motivation. In the 1600 meter run it appears that a combination of increases in LA and the individual's pain tolerance are factors which may limit the performance.

Ergogenic aids

For many years scientists have been studying methods to manipulate the normal physiological response to work in order to delay the onset of fatigue and subsequently enhance performance. Substances or procedures which enhance exercise performance are referred to as "ergogenic aids". For decades, strategies designed to accomplish this goal have been utilized in virtually all activities. Strategies geared at making desired changes in the energy utilization activity of the LA system have focused on increasing plasma and muscle pH values. By minimizing the occurrence of acidosis, the normal physiological and chemical interactions can proceed optimally. The use of buffering substances have facilitated the attainment of this desired pH level.

As early as 1923, Hill & Lupton proposed that using agents designed to buffer H+ could counteract the fatiguing effects of intense short-term exercise. During this type of activity, as the LA level rises, pH drops in the muscle and blood. In this acidic environment, glycolysis is slowed (Goldfinch et al., 1988), the muscle contractile mechanism is temporarily impaired (Mainwood & Worsley-Brown, 1975), and performance deteriorates.

Ingestion of buffering substances serves to create an alkalinizing effect within the blood. Since the muscle membrane is impermeable to the buffering substances, the majority of buffering occurs in the extracellular environment. By increasing

the bicarbonate concentration in the blood, the efflux of H+ and LA from the muscle is enhanced (Robin, 1961; Mainwood & Worsley-Brown, 1975). The disruptions associated with the formation of lactic acid become less pronounced. Sodium bicarbonate and sodium citrate have been cited as buffering agents and have demonstrated some promising ergogenic potential.

Sodium bicarbonate

As stated previously, increasing the alkalinity of the blood assists in neutralizing the strong metabolic acids produced during intense exercise. Sodium bicarbonate (NaHCO3) has been demonstrated to effectively achieve this during intense anaerobic exercise lasting greater than 30 seconds in duration. Results from many studies suggest that this may result in increased performance. Dennig, Talbott, Edwards, and Dill (1931), in a treadmill running study, determined that subject performance did improve when exercise commenced following alkalosis induced by ingestion of NaHCO3. In the study, the subject ingested substances designed to produce either an alkalotic or acidotic internal environment. Ten grams of sodium bicarbonate was administered in a single dose to facilitate alkalosis, and one dose of 15 grams of ammonium chloride (NH4Cl) was used to attain the acidotic state.

In each condition, the exercise began once the desired internal environment was achieved. The researchers evaluated

the duration of the run and the speed of the run under the conditions of acidosis, alkalosis, and control. They determined that the subjects' performance improved following alkalosis induced by ingestion of NaHCO3, as demonstrated by longer run times as compared to the control or acidotic condition. In the acidotic condition, subjects demonstrated a reduction of approximately 25% in duration of the performance of the running activity compared to normal, and a reduced capacity for oxygen debt. Performance terminated following 15 minutes of activity during the acidotic condition, while run times ranging from 18-20 minutes were noted during the other conditions. In this study, it appeared that the alkalosis condition may have neutralized the increase in acidity which occurred during the exercise, while the acidosis condition resulted in reduced LA buffering capacity.

Simmons and Hardt (1973) studied the effects of the ingestion of alkalizing substances on 5 highly trained sprint and 3 distance swimmers. The swimming distance selected for the sprinters was 100 meters, and for the distance swimmers it was 400 meters. Swim times for all subjects were assessed at a pre-test and 4 swim tests, all which were carried out at one-week intervals. The swimmers were assigned into either an experimental or a control group. The alkali substances used in the study included 0.715 grams of sodium citrate, 0.5 grams of sodium bicarbonate, and 0.215 grams of potassium citrate mixed into non-carbonated soft drinks. The control condition used 1.43 grams of sucrose mixed into the non-carbonated soft drink liquid.

Results indicated that the buffering conditions led to significantly better performances in sprint swimming than those performances of the control condition for all subjects. This was evidenced by significantly faster swim times for the 100 meter distance. However, the extent of improvement may have been limited in the trained subjects by the fact that these individuals already had an increased buffer capacity, secondary to their specific training regimen. No comparisons were made concerning the distance swimmers, as illness forced one of the three subjects to leave the study.

Wilkes et al. (1983) studied 6 trained male middle-distance runners performing in an 800 meter treadmill race following ingestion of 0.3 g/kg body weight of NaHCO3, ingestion of 0.3 g/kg of a placebo (calcium carbonate), or in a control condition. A large volume of water was consumed following the ingestion conditions. Racing times significantly improved following ingestion of NaHCO3 compared to those of the control and placebo conditions. Performance time improved in the experimental condition compared to the control condition by an average of 2.9 seconds. An average improvement of 2.2 seconds was noted in the experimental condition compared to the placebo condition. LA, pH, and bicarbonate concentrations were all higher in the experimental group compared to the control and placebo groups at the completion of the race. These results were consistent with those of Dennig and his colleagues.

In a similar study by Goldfinch et al. (1988), 6 highly trained male 400 meter runners served as subjects. All subjects

ingested 0.4 g/kg of NaHCO3 during the experimental condition of the study. Performance was enhanced during the experimental phase as measured by significantly faster run times following induced alkalosis compared to either the placebo or control condition. Subjects posted a mean run time that was 1.52 seconds faster in the experimental condition compared to either of the other conditions. Post-exercise bicarbonate and pH levels were significantly higher during this condition compared to the control and placebo condition.

Sutton et al. (1981), in a study of 5 male subjects performing exercise on a bicycle ergometer, reported similar findings. Each subject participated in a total of three data collection sessions, in which they received one of three substances per session. The substances which were randomly assigned included: NaHCO3 to facilitate the alkalosis condition; CaCO3 for the control condition; and NH4Cl for the acidosis condition. Substances were administered in a dose of 0.3 g/kg body mass and ingested over a 3 hour period prior to exercise.

Subjects performed continuous 20 minute exercise bouts on the cycle ergometer at 33% and 66% $\dot{V}O_2$ max. The subjects then performed until exhaustion at 95% $\dot{V}O_2$ max. Results indicated that endurance time until exhaustion at the 95% intensity was significantly longer in the induced alkalosis condition compared to both other conditions. Subjects were able to pedal for an average of 48 and 131 seconds longer following NaHCO3 ingestion compared to the control and acidosis conditions, respectively.

Horswill et al. (1988), in a study of 9 endurance trained male cyclists performing 2 minute sprint tests on an isokinetic bicycle ergometer, found no improvement in performance after ingestion of NaHCO3. Subjects performed one 2-minute sprint test on 4 separate occasions spaced a minimum of 5 days apart, and the total work performed was measured. One hour prior to each test, subjects consumed one of 4 solutions administered in random fashion. The solutions included: a placebo consisting of 400 ml of a flavored drink; and flavored drink solutions containing either 0.1, 0.15, or 0.2 g/kg body weight of NaHCO3.

Results indicate that post-exercise bicarbonate levels were significantly higher after consuming NaHCO3 compared to placebo, and was proportional to the dose administered. However, performance was not enhanced, as the total work performed was not significantly different in any condition. It is possible that the lack of improvement in performance was due to the fact that the duration of the event was not long enough to maximize the buffering effects associated with manipulation of the anaerobic glycolytic system, or the doses administered were inadequate. The researchers selected the doses used in the study in an attempt to minimize the potential for GI discomfort reported in other studies using higher doses. The results of this study support previous research by Wilkes et al. (1983), which indicate that ingestion of NaHCO3 in doses equal to or greater than 0.3 g/kg body mass is necessary to realize ergogenic potential.

Approximately 50% of the subjects participating in the Wilkes et al. study experienced GI symptoms secondary to

bicarbonate ingestion. Symptoms such as diarrhea, GI discomfort, and nausea were reported hours after ingestion of the substance. Similar GI symptoms were reported by several subjects in the Goldfinch et al. (1988) study following the completion of a 400 meter run. It appears that the ergogenic effects are evident in activities ranging from 1-4 minutes in duration with ingestion of this much bicarbonate, but the side-effects may render this strategy impractical for some individuals.

Not all the results have been as promising. Johnson & Black (1953) studied highly trained cross-country runners performing a 1.5 mile run. Results indicated no improvement in performance following bicarbonate ingestion. The subjects were administered a single dose of 3.5 grams of bicarbonate prior to the event. It is plausible that one dose of this quantity was not adequate in increasing the buffer reserve, and/or the aerobic nature of this event eliminated the need for buffering. If oxidative metabolic processes predominated, then factors other than buffering of LA may have determined race performance.

Sodium citrate

Sodium citrate has also been used as an ergogenic aid during anaerobic exercise in an attempt to facilitate an alkalotic state in the body. This leads to a reduction in the H+ concentration, improved buffering of LA, and increases in HCO3 and pH similar to that of NaHCO3 (Parry-Billings & MacLaren, 1986; Kowalchuk, Maltais, Yamaji, and Hughson, 1989). McNaughton and Cedaro

(1992) suggest that sodium citrate ingestion has an ergogenic benefit during cycling activities lasting from 2 to 4 minutes. Their research involved investigating the ergogenic benefit of sodium citrate ingestion in ten healthy college-aged students who performed cycling tasks that were of 10, 30, 120, and 240 seconds in duration. Blood bicarbonate, base excess, and pH levels were among the parameters assessed in all subjects. Three conditions were used, including an experimental, placebo, and control.

Total work performed and peak power were also assessed. Results suggest that 0.5 g/kg body mass of sodium citrate was more effective than the placebo, which consisted of 0.3 g/kg body weight of CaCO3 plus 0.5 g sodium chloride (NaCL), or the control in achieving this potential during the 120 and 240 second exercise periods. Peak power increased by an average of 150 Watts (W) in the experimental condition compared to both the control and placebo conditions for the 120 second exercise period. An approximate 100 W increase was also noted during the 240 second period for the experimental condition. Total work performed significantly increased in the experimental condition for the 120 and 240 second tasks. At 120 seconds, a 10 kilojoule (kj) increase was noted compared to the control condition. This increased to approximately a 20 kj improvement over control during the 240 second trial.

The researchers indicated that it was the ingestion of this alkalizing substance that caused the improvements in performance. The ingestion of the sodium citrate succeeded in

inducing alkalosis by creating a greater buffering gradient. Blood bicarbonate and blood lactate levels were significantly increased following citrate ingestion compared to the control and placebo conditions. Total work and peak power increased in the experimental condition when the duration of the cycling exceeded 2 minutes.

McNaughton (1990), in a cycling study involving 11 male subjects, obtained results that were in support of previous citrate research. He also determined that 0.5 g/kg of body mass was the most effective dose to achieve enhanced performance. He studied the effects of five different doses of sodium citrate ingested by the subjects prior to performing maximally on a bicycle ergometer for 1-minute exercise bouts. The purpose was to determine the most effective dose in terms of enhancing performance. Doses selected for study were 0.1 g/kg body mass, 0.2 g/kg, 0.3 g/kg, 0.4 g/kg, and 0.5 g/kg. Subjects ingested the substance approximately 1.5 hours before performance of the task. Subjects performed the maximal task for one minute at one particular dose on a given day. The control test was always first, and was followed by the 5 different doses. The order was randomly assigned, and each test occurred at least 2 days, but not longer than 5 days, following the previous one.

Base excess, bicarbonate concentration, and lactate concentration were among the parameters analyzed. Total work and peak power were the performance variables measured. The results indicated that, in this task, no ergogenic benefit was realized until the dose ingested reached 0.3 g/kg body mass. At

this point the total amount of work performed (40.62 kj) and peak power (1,197 W) significantly increased compared to the control condition (35.26 kj, 1,081 W). The 0.5 g/kg dose demonstrated significant increases in total work performed (44.63 kj) and peak power (1,306 W) compared to any other dose, the placebo condition, and the control condition. The blood bicarbonate concentration increased significantly in a linear fashion with each increasing dose. Base excess values significantly increased compared to the control and placebo conditions, and greater improvements were noted with increasing amounts ingested.

The results support the use of sodium citrate as an ergogenic aid to maximal anaerobic performance of 1-minute duration. Although the mechanism is not completely understood, it appears that ingestion increases the blood bicarbonate level similar to NaHCO3 ingestion. This creates a larger concentration gradient, which leads to enhanced extracellular buffering. Another finding from this study suggested that use of sodium citrate did not result in reports of symptoms of GI distress. This may represent an advantage over NaHCO3. Provided performance outcomes are similar, and GI discomfort avoided, sodium citrate ingestion in this type of activity may be warranted over sodium bicarbonate ingestion.

Direct comparison of acid-base results between studies utilizing buffering agents in attempt to enhance performance is difficult, secondary to the use of different blood sampling sites. Linderman et al. (1990) compared acid-base measurements

obtained via arterial, arterialized venous, and venous blood samples from 10 trained male cyclists performing a maximal graded exercise test on a cycle ergometer. Results determined that at the end of the exercise period, pH, lactate, and HCO3 values were similar between arterialized venous blood and venous blood samples. Both were significantly different than the values obtained using arterial blood.

At the conclusion of the exercise period, arterial lactate levels were higher than either arterialized venous or venous blood. Arterial pH and HCO3 levels at the conclusion of exercise were lower than venous values. They concluded that the use of arterialized venous or venous blood may not accurately reflect values found in arterial blood secondary to local tissue metabolism, and are not an appropriate substitute. These results support previous research by Doll, Keul, and Maiwald (1968). However, arterial sampling requires invasive procedures, is frequently painful to the subject, and requires the presence of trained medical personnel. As it represents a greater risk to the subject, the use of arterialized venous or venous blood samples are frequently used.

Arterialized venous blood is obtained by warming the surface surrounding the sampling site. By increasing the temperature of the skin in this manner, approximately 75% of the arterial blood is shunted away from the capillary beds and directed to the venous system. Often in a laboratory setting, the body part is heated during the performance of the activity using a hair dryer, heat lamp, or hot pack device. While appropriate for the

laboratory, this procedure is not practical for studies that involve field testing.

Horswill et al. (1988), in their study of 9 endurance-trained cyclists performing 2-minute sprint tests on a cycle ergometer, used arterialized venous blood obtained from a pre-heated forearm vein to analyze for lactic acid, pH, and bicarbonate. Goldfinch et al. (1988), in a study of 6 trained male 400 meter runners used arterialized venous blood obtained from an indwelling catheter placed in a dorsal vein of the hand. The blood was analyzed for pH, bicarbonate, and blood gas levels. The Wilkes et al. (1983) study of male 800 meter runners used a procedure similar to Goldfinch.

The use of venous blood for analysis is used often as a procedure during field studies. Costill et al. (1984), in a sprint cycling study, obtained 5 ml blood samples from a forearm vein of each subject. The blood samples were obtained pre-ingestion, pre-race, and post-race, and included analysis for pH, and bicarbonate level. Although, the acid-base values were not identical to those obtained via other sampling techniques, they were similar and consistent. The acid-base measurements obtained provide information concerning the relative change in pH, lactate, and HCO3 from blood draw to blood draw. Even though the values obtained are different than that obtained via other methods, the change in values of these variables render this procedure appropriate in many instances. Direct comparison between studies using different sites and sampling techniques should be avoided as the results may be inaccurate.

In summary, at the 1600 meter distance, the anaerobic glycolytic pathways make the most significant contribution to the total energy produced. Increases in LA concentration along with other factors, such as pain and impaired muscle contractile activity, appear to lead to deterioration of performance in this event. If both agents selected to buffer the increase in H+ demonstrate ergogenic potential as compared to the control and placebo substances, the subsequent question would be to determine if one has a decided advantage over the other in terms of performance outcomes. The desired performance enhancing benefits must be weighed against the undesirable side-effects of ingesting each substance. If one substance demonstrates equivalent or better ergogenic potential, and it has fewer side-effects, it is reasonable to consider it to be the ergogenic aid of choice.

CHAPTER III

METHODS

This study was conducted at Oregon State University, Corvallis, Oregon and Pacific University, Forest Grove, Oregon. The purpose of the study was to determine if ingestion of either sodium bicarbonate or sodium citrate prior to a competitive 1600 meter race enhanced performance in this high intensity activity. Previous research has demonstrated that buffering improves performance during anaerobic activities lasting approximately 1-2 minutes in duration. This study attempted to add to the knowledge base and to determine if either of the buffering agents selected have a decided advantage over the other in outcome.

Subjects

Seven male and five female competitive track athletes served as subjects for the study and completed an Informed Consent Form and medical questionnaire prior to participation. The subjects ranged from 18 to 33 years of age and were considered to be competitive runners as they were in training for and competing in races at the time of this investigation. Male runners were capable of completing this distance in a time faster than 5:00 minutes, while female subjects were required to be capable of completing this distance in a time better than

6:00 minutes. All subjects were apparently healthy with no known history of respiratory, cardiac, or metabolic disease, and had not sustained any injury which forced them to miss more than 3 scheduled training days in the month prior to data collection.

Instrumentation

Maximal oxygen consumption to determine each participant's aerobic capacity was assessed using a Sensormedics 2900 metabolic cart (Yorba Linda, CA). This test for aerobic capacity was conducted on a motorized treadmill (Sensormedics, MAX-1) which was calibrated for speed and grade prior to testing. Assessment of body composition was obtained via the hydrostatic weighing technique in a specially designed indoor tank with the subjects seated on a chair which was suspended in the tank. The tank utilized a custom made suspension type of scale (Toledo load cell). A GO-MI 5000 lung function testing apparatus was used to measure residual lung volumes.

The four 1600 meter races each subject participated in were conducted on a 400 meter synthetic surface track utilized for competitions. The subjects were not subjected to inclement weather conditions during the races. The performances were measured as the elapsed time to complete the distance using accepted track racing standards, with recorded accuracy to within one tenth of a second. Blood samples of 50 uL were obtained via a standard finger puncture from each participant at

specific intervals of the testing sessions to measure blood lactate levels. Blood lactate levels were assessed using a (Yellow Springs Incorporated Sport 1500 model, Yellow Springs, OH) lactate analyzer. Blood samples of 5 ml were obtained via venipuncture using the median cubital vein of each participant to assess bicarbonate level and pH. Bicarbonate levels and pH were assessed using an AVL model 995 (AVL Inc., Roswell, GA) blood gas analyzer. The bicarbonate concentrations, pH, and lactate levels were determined prior to beginning the race, and immediately upon completion of the race.

Subjective symptoms were reported via a Subjective Symptom Scale (see Appendix B) designed by the researcher. The interval scale was administered immediately following the post-race blood draw. It consisted of identifying the presence of any symptoms of GI discomfort that occurred at specific intervals during the performance of the race. Symptoms were rated in the following manner: not present, mild, moderate, or severe.

Substances ingested during the treatment sessions included: 0.4 g/kg of body mass of NaHCO3, 0.5 g/kg of sodium citrate, and, as the placebo, an equal number of gelatin capsules filled with calcium carbonate as was filled with NaHCO3. Only one treatment was administered at each data collection session. The substances were administered in gelatin capsule form and taken with approximately one liter of water. The order of administration of the substances was via a counter-balanced double-blind assignment.

Procedures

Each subject was required to produce evidence ensuring that he or she met the criteria for inclusion prior to participation in the study. All subjects participated in an aerobic capacity assessment, a hydrostatic weighing assessment, and four competitive 1600 meter races. The races were scheduled at least 3 days apart to allow for adequate recovery between each test. The four race conditions were assigned in a counter-balanced double-blind manner. Conditions selected for analysis included: control, placebo (calcium carbonate), sodium bicarbonate, and sodium citrate. Following explanation of the procedures and completion of a medical questionnaire for screening, informed consent was obtained (see Appendix A).

Laboratory Testing

On the initial visit, aerobic capacity was determined via a maximal oxygen consumption test conducted on the motorized treadmill. For male subjects, the treadmill was initially set at a 6% grade and a speed of 6 miles per hour (mph). Progression was as follows: 0.5 mph increases per minute until the subject reached a velocity of 9 mph. Following this point the grade was increased by 1% per minute until the subject became exhausted. For female subjects, the treadmill was initially set at a 6% grade and a speed of 5 mph. Speed was increased by 0.5 mph each minute until reaching 8 mph. After this point the treadmill

elevation was increased by 1% per minute. The test continued in this manner until the subject became too fatigued to continue. The test was designed to range from 8 to 12 minutes in duration.

During the test, the subject breathed room air through a two-way breathing valve, with the exhaled air conducted through low resistance tubing to the metabolic cart. This was to permit quantitative analysis of expiratory volumes and concentrations of oxygen and carbon dioxide. From this analysis, the volume of oxygen consumed can be determined. During the procedure, continuous electrocardiographic (ECG) data was collected. Present during all data collection sessions were individuals certified in cardiopulmonary resuscitation (CPR).

During the same session, all subjects underwent a hydrostatic weighing procedure to determine body composition. The indoor tank contained water approximating body temperature (35-37° C). The tank featured a suspension scale and, following maximal exhalation, the subject submerged him or herself for a period of 2-3 seconds until the underwater weight could be recorded. The protocol of taking the average of 3 highest trials which were within 0.1 kg, as outlined by Bonge and Donnelly (1989), was utilized to estimate the underwater weight. Residual volume (RV) was estimated using the neon dilution method. Body density could then be calculated by using Archimedes' principle as outlined by Goldberg & Buskirk, (1961). Once body density was determined, percent body fat was calculated by using the Siri Equation. Except for one subject, the hydrostatic weighing procedure was performed prior to the test

for aerobic capacity. This was to minimize the effect of fatigue, and ensure the subjects could achieve optimal residual volume measurements. At subsequent sessions, prior to the 1600 meter race, the body weight of each subject was obtained using a medical quality scale.

1600 Meter Field Tests

Approximately 120 minutes prior to the race on buffering and placebo condition days, each subject ingested the assigned substance. The pre-determined dose of the prescribed substance was encapsulated in gelatin capsules and ingested with one liter of water to minimize any symptoms of gastrointestinal distress. Subjects were instructed to avoid strenuous activity until the race. Approximately 2 hours after ingestion, a 50 uL blood sample was obtained from each participant via the standard finger puncture method in order to measure blood lactate levels. Bicarbonate concentration and pH level was determined via analysis of a 5 ml sample of blood obtained via venipuncture using the median cubital vein. This procedure was repeated immediately following completion of the race.

Following the initial blood draw, participants proceeded to the track where they performed warm-up exercises similar to those of their usual routine. Following a 30 minute warm-up period, a trained starter called the runners to the starting line and began the race in accordance with track racing standards. As subjects completed the race, their times were recorded, and they

were led back to the blood draw area where the final blood draw was completed. Blood samples for bicarbonate and pH were unable to be obtained during any trial for one subject (Subject #10). For this subject, blood samples were obtained at each trial via finger punctures, and those samples used to analyze lactate values. The subjects were instructed to complete a subjective symptom scale in which they reported on the presence and severity of symptoms of GI discomfort they experienced during specific intervals in the race. The intervals identified by the scale were at 400, 800, 1200, and 1600 meters; subjects were also instructed to list all symptoms experienced and indicate whether they were mild, moderate, or severe in nature (see Appendix B).

In the period of time between testing sessions, subjects were strongly encouraged to maintain a consistent pattern of training, eating, and sleeping. They were encouraged to refrain from a hard training session on the day prior to data collection for each trial. They also were asked to note the presence of injuries or other factors they perceived to potentially influence their performance that session.

Data Analysis

The StatView SE+ Graphics computer program (Abacus Concepts Inc., Berkeley, CA) was used for statistical analysis of the data. The analysis consisted of the calculation of means and standard deviations for pH, lactate, and bicarbonate levels at

each measurement interval for each substance ingested. The significance of the difference in the means of each was determined using a repeated measures analysis of variance (ANOVA). If a significant difference was noted, Newman-Keul's multiple range post-hoc test was utilized to isolate the source of the difference. A one-factor ANOVA for overall performance time versus substance ingested and a one-factor ANOVA for symptoms versus substance ingested were performed. Significance was accepted at the alpha=.05 level.

CHAPTER IV

RESULTS AND DISCUSSION

Results

Demographic data, percent body fat as assessed via hydrostatic weighing, and aerobic capacity ($\dot{V}O_2$ max) data for all subjects appear in Table 1. All subjects were considered to be in a well-trained state as evidenced by $\dot{V}O_2$ max values that exceeded 60 ml/kg/min for males, and 50 ml/kg/min for female subjects. All subjects had percent body fat values that were lower than average for their age and sex. The subjects performed the runs grouped into three smaller pools secondary to scheduling constraints, and therefore, the entire group did not compete at the same times. The first group consisted of five subjects (2 male, 3 female); the second consisted of 3 males and 1 female subject; and the third was composed of 2 males and 1 female subject.

Data collection for Group 1 occurred during the months of July and August 1994. Group 2 data collection occurred during October 1994, and Group 3 participated during November and December of that year. Details including means and standard deviations regarding environmental conditions can be found in Appendix C. Average dry temperature recorded in degrees Fahrenheit during the data collection period for Group I was 79.5°, wet bulb was 68.1°, and conditions were sunny and breezy.

Table 1. Demographic and descriptive subject data

Subject	Sex	Age (yrs)	Hgt (in)	Wgt (kg)	Body Fat (%)	$\dot{V}O_{2\,max}$ (ml/kg/min)
1	M	31	70.0	70.0	9.8	61.38
2	M	22	73.0	77.0	8.6	67.63
3	M	18	68.0	63.0	3.4	66.88
4	M	23	73.0	75.0	7.8	65.25
5	M	26	73.0	71.0	12.7	65.89
6	M	31	68.0	70.0	6.5*	60.81
7	M	25	66.0	65.0	7.9*	67.40
Mean		25	70.1	70.1	8.1	65.00
8	F	27	66.0	50.0	9.9	63.51
9	F	33	67.0	51.0	14.9	58.05
10	F	31	65.0	51.5	12.7	50.61
11	F	25	66.0	58.0	6.0	61.67
12	F	19	63.5	71.0	19.3*	50.90
Mean		27	65.5	56.3	12.6	56.90

* Obtained using Lange Calipers and the Sum of Seven Skinfold Technique (Jackson and Pollock, 1977)

Conditions for Group 2 included: average dry temperature of 55°, average wet bulb was 53.25°, with overcast skies and windy conditions. Group 3 participated under an average dry temperature of 46.25°, mean wet bulb temperature of 44.25°, and cool and windy conditions.

Means and standard deviations for overall performance times appear in Table 2. Results from a one-factor Analysis of Variance (ANOVA) indicate that there was no significant difference between overall performance time and substance ingested for the group as a whole ($p= 0.99$). Bicarbonate ingestion resulted in the overall fastest mean performance time with a mean of 319.2 seconds, and citrate ingestion led to the slowest mean performance time of 321.1 seconds.

Four of the twelve subjects (33.3%) achieved their best performances after ingestion of bicarbonate. However, five of the subjects (41.67%) ran their fastest times during the control trial in which they did not ingest any substance. One subject (8.3%) performed the fastest trial after ingesting citrate, while another subject ran the fastest trial following ingestion of the placebo, calcium carbonate. Four of the subjects (33.3%) ran their slowest times following ingestion of bicarbonate, compared to 25.0% each for citrate and control, and 16.67% for placebo.

Gender comparison via a one-factor ANOVA indicated that there was no significant difference in overall performance time

Table 2. Performance times for the 1600 meter run

1600 meter Subject	Time (sec)			
	Control	Placebo	Citrate	HCO_3
1	301.8	293.0	300.2	294.7
2	308.4	310.3	310.8	306.2
3	306.1	310.8	307.3	318.1
4	290.1	296.6	293.3	292.2
5	321.2	307.0	307.4	294.0
6	286.3	284.4	304.1	283.5
7	288.0	296.2	298.5	288.7
8	344.8	342.7	335.9	335.5
9	360.9	355.6	354.3	362.6
10	358.2	366.3	366.2	364.3
11	331.8	328.0	333.1	328.5
12	340.1	347.7	342.0	352.0
Mean	319.8	319.9	321.1	319.2
SD	27.0	27.3	24.1	30.1

p=.9986

across any condition for male subjects (p= .73). The HCO3 condition resulted in the fastest performance time with a mean time of 296.8 seconds, while citrate ingestion resulted in the slowest overall time, with a mean of 303.1 seconds. For female subjects, there was no significant difference in performance time for any trial (p= .96). Ingestion of citrate resulted in the fastest performance time with a mean of 346.3 seconds, while the HCO3 condition yielded the slowest overall time, with a mean of 350.6 seconds.

Results from a one-factor ANOVA demonstrated no significant differences between substance ingested and severity of symptoms of gastrointestinal (GI) discomfort experienced for the entire subject group (p=.06). Means and standard deviations are presented in Table 3. A score of 0 was given if no symptoms were experienced, a score of 1 indicated mild GI symptoms, a score of 2 represented moderate symptoms, and a score of 3 indicated severe symptoms were experienced. Bicarbonate ingestion produced the most severe symptoms, with one individual (8.3%) reporting severe symptoms including diarrhea and gas; three reporting moderate symptoms of bloating, gas, and an overall sensation of fullness; and one reporting mild symptoms. Citrate ingestion resulted in the greatest number of individuals reporting symptoms (50.0%). Three of the six subjects who reported symptoms, indicated they experienced moderate symptoms such as gas and bloating, while the remaining three indicated that they experienced mild gas and bloating. Twenty five percent of the subjects reported mild

Table 3. Severity of GI symptoms experienced

0= no GI symptoms
1= mild GI symptoms
2= moderate GI symptoms
3= severe GI symptoms

Subject	Severity of GI symptoms			
	Control	Placebo	Citrate	HCO_3
1	0	0	0	0
2	0	1	0	0
3	0	0	1	0
4	0	0	2	1
5	0	0	2	3
6	0	0	2	0
7	0	0	0	0
8	0	1	1	2
9	1	1	0	2
10	0	0	2	0
11	0	0	0	0
12	0	1	0	0
Mean	.083	.333	.75	.833
SD	.289	.492	.866	1.115

p=.0649

symptoms of GI discomfort following ingestion of placebo, while 8.33% experienced mild symptoms during the control condition. Further analysis, based on gender, of severity of GI symptoms reported following ingestion of the substances, indicated that no significant difference existed between substance ingested and severity of symptoms for the male subjects (p= .09). For female subjects, the one-factor ANOVA also did not identify any significant differences (p=.17).

Pre-race acid-base values were within normal ranges in all conditions for pH and HCO3, and slightly elevated in all conditions for lactate. A one-factor ANOVA for pre-race values compared to substance ingested determined that there was no difference in pre-race mean lactate values in any condition. Bicarbonate levels indicate that ingestion of certain substances led to significantly higher pre-race HCO3 values (p=.0006). Post-hoc analysis using the Newman-Keuls multiple range test to isolate the source of the difference determined that both the bicarbonate and the citrate ingestion led to significantly higher HCO3 levels compared to placebo and control conditions at the beginning of the race.

Pre-race pH values indicated that a significant treatment effect existed (p=.0399). Post-hoc analysis using the Newman-Keul procedure failed to isolate the source of the difference. However, ingestion of citrate resulted in the highest pre-race pH value at 7.381. This was closely followed by ingestion of HCO3 at a value of 7.379. The control condition exhibited the lowest pre-race value of 7.318. Based on the operational definitions used in

this study, alkalosis was achieved in both of the buffering conditions. Results of pre-race blood values versus substance ingested are presented in Table 4.

Repeated measures ANOVA indicated that there was a significant exercise (p=.0001) but no treatment effect for lactate level (p=.945). The performance of the exercise directly influenced lactate values and caused them to increase from the pre-race to post-race condition. The nature of the activity suggests that LA accumulated in the muscle and blood. There were significant exercise (p=.0001) and treatment effects for pH (p=.0423). During the performance of the 1600 meter run, the blood became more acidic as pH declined secondary to LA accumulation. Ingestion of HCO3 led to the highest post-race pH value at 7.173, followed by citrate at 7.159; placebo at 7.095; and control at 7.09. Newman-Keul's multiple range post-hoc analysis was unable to isolate the source of the difference (Table 4).

Repeated measures ANOVA indicated a significant exercise (p=.0001) and treatment effect for bicarbonate (p=.0005). Reductions in HCO3 values were noted from pre-race to post-race in all conditions. Post-hoc analysis using the Newman-Keuls multiple range test indicated the significant difference was between the control and citrate ingestion condition (see Table 4).

Table 4. Acid-base results for the 1600 meter run (Mean ± SD)

# indicates exercise effect (p<.05)
% indicates treatment effect (p<.05)
† indicates exercise and treatment effect (p<.05)

Variable	Condition	Measurement time	
		Pre-race	Post-race
pH	control	7.318 (.038)	7.090 (.087)#
	citrate	7.381 (.064)	7.159 (.077)#
	HCO3	7.379 (.055)	7.173 (.087)#
	placebo	7.348 (.064)	7.095 (.130)#
lactate (mmol/l)	control	1.801 (1.324)	13.478 (3.404)#
	citrate	1.753 (0.715)	14.331 (3.752)#
	HCO3	1.813 (1.005)	13.897 (4.793)#
	placebo	1.826 (0.808)	14.620 (5.154)#
HCO3 (mmol/l)	control	28.255 (1.576)	14.355 (3.551)#
	citrate	31.709 (0.932)%	17.891 (3.222)†
	HCO3	32.082 (3.263)%	17.427 (3.519)#
	placebo	29.682 (2.404)	15.336 (2.341)#

Discussion

Although physiological values found post-race in the present study concerning pH, lactate levels, and bicarbonate levels were similar to those cited in previous research, there was no improvement in overall performance time noted in this study. Previous research (Wilkes et al., 1983; Goldfinch et al., 1988) found significant improvements in performance time and buffering capacity following ingestion of NaHCO3 at the 800 meter and 400 meter distance, respectively. In the Wilkes et al. study (1983) of trained 800 meter runners, subjects ran an average of 2.9 seconds faster following ingestion of NaHCO3 compared to the control condition, and 2.2 seconds faster than compared to the placebo condition. The dose used in the present study was equivalent to the one used by Wilkes et al.

A similar study by Goldfinch et al. (1988) of 6 trained male 400 meter runners determined that ingestion of NaHCO3 resulted in an improvement of performance time by an average of 1.52 seconds compared to either the control or placebo condition. The dose used in their study, 0.4 g/kg, was equivalent to that used in the present study. In the present study, there was an average improvement in performance time of 0.6 seconds following ingestion of NaHCO3, and an average reduction in performance of 1.3 seconds following ingestion of citrate compared to the control condition. One can speculate that since the 1600 meter distance does not emphasize anaerobic glycolysis as a means to generate energy to the extent that races of 400 and 800 meter

distances do, maximal ergogenic potential is not realized in the 1600 event. Oxidative processes contribute a greater amount to the total energy production as the duration of the exercise period is extended beyond 4 minutes.

The lack of significant improvement in overall performance time is consistent with the results found by Johnson & Black (1953). Their study consisted of trained cross country runners performing a 1.5 mile run either during the control condition or after ingestion of a single 3.5 g dose of NaHCO3. In the Johnson & Black study, it is probable that the lack of improvement was attributable to the inadequate size of the dose administered and/or the distance of the race. In the present study, the race was of a longer distance than those studied by either Wilkes or Goldfinch. Previous research (Kindermann et al., 1977) suggests that ingestion of at least 0.3 g/kg is necessary to enhance performance. In the current study, a dose of 0.4 g/kg was used.

In the present study, only one subject (Subject #5) demonstrated a substantial improvement in time after ingestion of NaHCO3. He improved by 11.0 seconds over his next fastest time following ingestion of a total of 28.2 grams. However, this same subject reported that he had severe symptoms of diarrhea during the warm-up period and just prior to the run.

Of the four subjects who ran their slowest times following ingestion of NaHCO3, the decrement in performance compared to their next slowest time exceeded 4 seconds. In three out of the four, it exceeded 6 seconds and, for all practical purposes, took them out of contention in the race. Three of the four reported no

symptoms of GI discomfort, while the fourth indicated she had moderate symptoms of intestinal cramping during the race after she ingested 20 grams of NaHCO3. None of the 400 meter subjects involved in the Goldfinch et al. (1988) study, and only one of the six runners in the Wilkes et al. (1983) 800 meter study demonstrated a reduction in performance following ingestion of NaHCO3 compared to the control condition. The decrease noted in performance of the subject in the Wilkes study was only 0.1 second. The results of the present study clearly do not support those of previous research concerning ingestion of NaHCO3.

Three individuals ran their slowest times after ingesting citrate. Previous research involving ingestion of citrate as an ergogenic aid has utilized total work and peak power as the measured variables. Therefore, those results do not apply to the present study.

Five of the 12 subjects (41.67%) experienced symptoms of GI discomfort following ingestion of HCO3. Of those reporting symptoms, 80% rated severity as being moderate or severe. The presence of temporary acute discomfort following ingestion of HCO3 is consistent with previous work by several researchers (Wilkes et al., 1983; Gledhill, 1984; Goldfinch et al., 1988; Linderman & Fahey, 1991). In the Wilkes study, 50% of the subjects reported symptoms. Linderman and Gosselink (1994) indicate that these GI symptoms are secondary to the large amount of sodium that needs to be ingested in order to realize an ergogenic potential. The ingestion of large quantities of sodium

requires additional fluid to be drawn into the intestine, resulting in the presence of the symptoms in some subjects.

Though a greater number of subjects reported symptoms of GI discomfort following citrate ingestion (50%) compared to bicarbonate ingestion, the symptoms were not as severe. Previous studies indicate that citrate is normally well tolerated by the subjects (McNaughton, 1990). In the present study, half of the participants reported symptoms after citrate ingestion, while 25% of the subjects also reported symptoms after ingestion of the placebo, and 8% in the control condition. Perhaps anxiety concerning the performance of a competitive event led to a greater than expected stress response in these subjects. This may explain why such a large percentage of the subjects reported symptoms following citrate ingestion, as the results differ from those of previous research.

Results from the acid-base variables indicate that, as expected, blood lactate levels demonstrated dramatic increases following completion of the event compared to prior to the run. Likewise, post-race pH values were significantly lower than pre-race values. Since this racing distance necessitates a significant contribution from anaerobic glycolysis to generate the energy necessary to power the activity, lactic acid accumulates in the muscle and blood, and the environment becomes more acidic. These results are consistent with previous research (McNaughton, 1990), that suggests this is due to the anaerobic nature of the activity, and implicates it as the major cause of fatigue in this type of event.

Repeated measures ANOVA indicated a significant exercise ($p=.0001$) and treatment effect for bicarbonate level ($p=.0005$). Reductions in HCO3 values were noted from pre-race to post-race in all conditions. Post-hoc analysis indicated that the significant difference was between the control and citrate condition. Analysis of the pre-race blood sample for HCO3 indicated that both the citrate and bicarbonate conditions were significantly higher than the other conditions. These buffering conditions served to increase the extracellular buffer reserve prior to commencement of the exercise. The results from this study are in agreement with previous research (Wilkes et al., 1983; Gledhill, 1984; Goldfinch et al., 1988).

It is an expected response for bicarbonate levels to decrease from pre-race to post-race as a result of the heavy exercise performed in a competitive race such as the 1600 meter run. Hermansen & Osnes (1972) suggest that this is due to the LA formed during the exercise period. In this type of event, energy is supplied via a combination of anaerobic and oxidative processes, with anaerobic glycolysis contributing approximately 55% of the energy generated (Fox & Mathews, 1981). Due to this, the accumulation of LA in the blood and muscle is noted. The results of this study support the previous research (Gledhill, 1984). The ingestion of a buffering agent may function to maintain bicarbonate levels higher for a longer period of time. This indicates better ability to buffer as an efflux of H+ ions is noted. Hydrogen ions are more readily buffered in the extracellular fluid, allowing greater work to be performed before exhaustion.

Direct comparison of acid-base variables between the present study and similar previous studies demonstrated consistent and similar values for pH found post-race (Kindermann et al, 1977; Wilkes et al., 1983; and Goldfinch et al., 1988). In the present study, post-race pH in the control condition was 7.090, in the NaHCO3 condition it was 7.173, and following ingestion of citrate it was 7.159. The Wilkes et al. (1983) study of 800 meter runners by comparison found values to be 7.07 for the control condition, and 7.18 in the alkalosis condition. Goldfinch et al. (1988) found similar pH values post-race in a study of 400 meter runners.

In the Wilkes et al. study, post-race HCO3 and lactate levels were slightly lower than in the present study. In the present study, lactate values for the control and NaHCO3 conditions were 13.478 mmol/l, and 13.897 mmol/l, respectively. Bicarbonate levels for the control and NaHCO3 conditions in the present study were 14.355 mmol/l and 17.427 mmol/l, respectively. In the Wilkes study, lactate level for the control and NaHCO3 conditions were 12.62 mmol/l and 14.29 mmol/l. Bicarbonate levels found post-race in the Wilkes study were 9.9 mmol/l in the control condition and 14.3 for the alkalotic condition. In that study, the use of a slightly smaller dose of NaHCO3 compared to the present study may explain the differences in values between the studies. Similar results were noted in the Goldfinch study.

Based on the similar acid-base results obtained from studies that were of similar nature, but of shorter duration, one can speculate as to why no improvement in performance occurred in

the present study. A plausible explanation is that there was a greater reliance on the aerobic system in the present study, and buffering was not utilized to the same degree. Factors other than LA accumulation may have been responsible for the onset of fatigue. Possibly a reduction in the rate of oxidative enzymatic activity, or decreases in glycogen levels occurred. Also, it is possible that the symptoms of GI discomfort experienced by many subjects decreased their willingness to perform at as high a level of intensity as they may have been capable of.

Although the subjects were not required to race in inclement weather, there was quite a bit of variability in conditions within the three groups of subjects. There was an approximate 30 degree difference in mean dry temperature between Group 1 and Group 3. However, conditions were similar across trials within each group, and each subject had been regularly training outdoors in similar environmental conditions and was acclimatized to them.

CHAPTER V

SUMMARY, CONCLUSIONS, AND RECOMMENDATIONS FOR FUTURE STUDY

Summary

This study was performed to determine if ingestion of sodium bicarbonate or sodium citrate resulted in faster performance time during a competitive 1600 meter run. Seven male and 5 female trained track athletes served as subjects. Ages ranged from 19 to 33 years. All subjects participated in a total of 4 competitive 1600 meter races. During each racing session, one of the following substances was administered via a counter-balanced design: sodium bicarbonate; sodium citrate; calcium carbonate as the placebo; and one session served as the control. Acid-base data were recorded prior to and immediately following each race. Overall performance time and severity of symptoms of GI discomfort were recorded at the completion of the run.

Means and standard deviations for overall performance time and severity of symptoms were calculated. Ingestion of NaHCO3 resulted in the fastest overall performance time with a mean of 319.2 seconds, and citrate ingestion resulted in the slowest overall time with a mean of 321.1 seconds. Ingestion of sodium citrate resulted in 50% of the participants reporting symptoms of GI discomfort. Fewer subjects (41.6%) reported symptoms following ingestion of NaHCO3, however, symptoms were classified as more severe in nature. There were no significant

differences noted in overall performance time (p=.99) or severity of GI symptoms (p.06) experienced for any condition. In this study, the ingestion of selected buffering agents did not enhance performance in the 1600 meter run.

A one-factor analysis of variance was used to determine pre-race acid-base values for each substance. A significant treatment effect was noted for pH (p=.039). Compared to the control or placebo conditions, ingestion of either buffering agent resulted in achievement of an alkalotic state, as pH exceeded 7.35. Post-hoc analysis using the Newman Keul multiple range test failed to isolate the source of the difference. Significant increases in bicarbonate values were found pre-race for both HCO3 ingestion and citrate ingestion compared to the placebo and control conditions (p=.0006).

Repeated measures analysis of variance was used to determine the significance of the difference among the means of each blood variable from pre-race to post-race. A significant difference in bicarbonate level was found post-race between the control and the citrate condition (p=.0005), with ingestion of citrate resulting in a bicarbonate level of 17.891 mmol/l.

Conclusions

1. Mean performance time was not significantly different between the buffering conditions and the control or placebo conditions. This finding does not support the first hypothesis.

2. Blood pH levels were significantly higher in the buffering conditions compared to the placebo and control conditions following the completion of the run. However, post-hoc analysis failed to isolate the source of the difference. This finding supports the hypothesis, and therefore, the hypothesis is accepted.

3. Blood lactate concentrations were not significantly different across the conditions at the completion of the run. The third hypothesis is rejected based on the fact that the findings do not support the hypothesis.

4. The blood bicarbonate level was significantly higher in the citrate condition compared to all other conditions following the completion of the run. This finding only partially supports the fourth hypothesis, as ingestion of one of the two buffering agents led to significantly higher post-race values.

5. Sodium citrate ingestion did not lead to significantly fewer symptoms of GI discomfort compared to bicarbonate ingestion. The fifth hypothesis is therefore rejected.

6. There was no significant difference in race time following ingestion of sodium citrate compared to ingestion of the other substances. This finding does not support the sixth hypothesis.

Recommendations for future study

To the knowledge of this investigator, there have been only a limited number of quantitative studies conducted in which the use of selected buffering agents were used in high intensity running activities exceeding 2 minutes' duration. The results from these few studies have yielded informative if not conflicting results. The need for additional research is evident. Suggestions for further study include:

1. Repeat the same investigation controlling more stringently for other behaviors such as diet, sleep, and exercise; and compare the results obtained with the results of this investigation. Although subjects in the present study were encouraged to refrain from difficult training on the day prior to data collection, no effort was made to force them to change their normal behaviors.

2. Repeat the present study running all the male subjects together in one group, and all the female subjects together in one group. This may foster a more competitive racing scenario.

3. Repeat the same study using a population of subjects considered to be more elite runners.

4. Repeat the present investigation utilizing a larger number of subjects, and utilizing different populations of runners. A

power analysis revealed a low power value, secondary to the small sample size.

5. Repeat the present investigation using shorter distances such as the 1,000 and 1,200 meter race. The contribution to energy production from anaerobic glycolysis would be greater in races of this distance compared to the present study, and perhaps greater ergogenic potential would be realized.

BIBLIOGRAPHY

Asmussen, E. (1979). Muscle fatigue. Medicine and Science in Sports and Exercise, 11(4), 313-321.

Barr, D. P., Himwich, H. E., & Green, R. P. (1923). Studies in the physiology of muscular exercise. Changes in acid-base equilibrium following short periods of vigorous muscular exercise. Journal of Biological Chemistry, 55, 495-523.

Bates, B., Osternig, L., & James, S. (1977). Fatigue effects in running. Journal of Motor Behavior, 9(3), 203-207.

Bouissou, P., Defer, G., Guezennec, C., Estrade, P., & Serrurier, B. (1988). Metabolic and blood catecholamine responses to exercise during alkalosis. Medicine and Science in Sports and Exercise, 20, 228-232.

Brooks, G. (1985). Anaerobic threshold: review of the concept and directions for future research. Medicine and Science in Sports and Exercise, 17(1), 22-31.

Costill, D. L., Verstappen, F., Kuipers, H., Janssen, E., & Fink, W. (1984). Acid-base balance during repeated bouts of exercise: influence of HCO3. International Journal of Sports Medicine, 5, 228-231.

Dennig, H., Talbott, J. H., Edwards, H. T., & Dill, D. B. (1931). Effects of acidosis and alkalosis upon the capacity for work. Journal Clinical Investigation, 9, 601-613.

Doll, E., Keul, J., & Maiwald, C. (1968). Oxygen tension and acid-base equilibria in venous blood of working muscle. American Journal of Physiology, 215, 23-29.

Edwards, R. (1981). Human muscle function and fatigue. London: Pitman.

Fabiato, A., & Fabiato, F. (1978). Effects of pH on the myofilaments and the sarcoplasmic reticulum of skinned cells from cardiac and skeletal muscles. Journal Physiology London, 276, 233-255.

Farrell, P. A., Wilmore, J. H., Coyle, E. F., Billing, J. E., & Costill, D. L. (1979). Plasma lactate accumulation and distance running performance. Medicine and Science in Sports and Exercise, 11(4), 338-344.

Fox, E. & Mathews, D. (1981). The Physiological Basis of Physical Education in Athletics. Philadelphia: Saunders College Publishers.

Fuchs, F., Reddy, Y., & Briggs, F. N. (1970). The interaction of cations with the calcium-binding site of troponin. Biochemistry Biophysiology Acta, 221, 407-409.

Gledhill, N. (1984). Bicarbonate ingestion and anaerobic performance. Sports Medicine, 1, 177-180.

Goldfinch, J., McNaughton, L., & Davies, P. (1988). Induced alkalosis and its effects on 400-m racing time. European Journal of Applied Physiology, 57, 45-48.

Grandjean, E. & Kogi, K. (1971). Proceedings of Symposium on Methodology in Human Fatigue Assessment. London: Taylor & Francis Ltd.

Haverty, M., Kenney, W. L., & Hodgson, J. L. (1988). Lactate and gas exchange responses to incremental and steady state running. British Journal of Sports Medicine, 22(2), 51-54.

Hermansen, L. & Osnes, J-B. (1972). Blood and muscle pH after maximal exercise in man. Journal of Applied Physiology, 32(3), 304-308.

Hewitt, J. E. & Calloway, E. C. (1936). Alkali reserves of blood in relation to swimming performance. Research Quarterly, 7, 83-93.

Horswill, C. A., Costill, D. L., Fink, W. J., Flynn, M. G., Kirwan, J. P., Mitchell, J. B., & Houmard, J. A. (1988). Influence of sodium bicarbonate on sprint performance: relationship to dosage. Medicine and Science in Sports and Exercise, 20(6), 566-569.

Inbar, O., Rotstein, A., Jacobs, I., Kaiser, P., Dlin, R. & Dotan, R. (1983). The effects of alkaline treatment on short-term maximal exercise. Journal of Sports Science, 1, 95-104.

Johnson, W. R. & Black, D. H. (1953). Comparison of effects of certain blood alkalinizer and glucose upon competitive endurance. Journal of Applied Physiology, 5, 577-578.

Kindermann, W., Keul, J., & Huber, G. (1977). Physical exercise after induced alkalosis (bicarbonate or tris-buffer. European Journal of Applied Physiology, 37, 197-204.

Kirkendall, D. T. (1990). Mechanisms of peripheral fatigue. Medicine and Science in Sports and Exercise, 22(4), 444-449.

Kowalchuk, J. M., Maltais, S. A., Yamaji, K., & Hughson, R. L. (1989). The effect of citrate loading on exercise performance, acid-base balance and metabolism. European Journal of Applied Physiology, 58, 858-864.

Linderman, J. & Fahey, T. D. (1991). Sodium bicarbonate ingestion and exercise performance. Sports Medicine, 11(2), 71-77.

Linderman, J., Fahey, T.D., Lauten, G., Brooker, A.S., Bird, D. (1990). A comparison of blood gases and acid-base measurements in arterial, arterialized venous, and venous blood during short-term maximal exercise. European Journal of Applied Physiology, 61(4), 294-301.

Linderman, J. & Gosselink, K. (1994). The effects of sodium bicarbonate ingestion on exercise performance. Sports Medicine, 18(2), 75-80.

Mainwood, G. W. & Worsley-Brown, P. (1975). The effects of extracellular pH and buffer concentration on the efflux of lactate from frog sartorius muscle. Journal of Physiology, 250, 1-22.

McKenna, M. (1992). The roles of ionic processes in muscular fatigue during intense exercise. Sports Medicine, 13(2), 134-145.

McNaughton, L. (1990). Sodium citrate and anaerobic performance: implications of dosage. European Journal of Applied Physiology, 61, 392-397.

McNaughton, L. & Cedaro, R. (1992). Sodium citrate ingestion and its effects on maximal anaerobic exercise of different durations. European Journal of Applied Physiology, 64, 36-41.

Nakamaru, Y., & Schwartz, A. (1972). The influence of hydrogen ion concentration on calcium binding by skeletal muscle sarcoplasmic reticulum. Journal of General Physiology, 59, 22-32.

Parry-Billings, M. & MacLaren, D. P. M. (1986). The effect of sodium bicarbonate and sodium citrate ingestion on anaerobic power during intermittent exercise. European Journal of Applied Physiology, 55, 524-529.

Robin, E. D. (1961). Of men and mitochondria: intracellular and subcellular acid base relations. New England Journal of Medicine, 265, 780-785.

Rupp, J. C., Bartels, R. C., Zuelzer, W., Fox, E. L., & Clark, R. N. (1983). Effect of sodium bicarbonate ingestion on blood and muscle pH and exercise performance. Medicine and Science in Sports and Exercise, 15(2), 115.

Sahlin, K. (1992). Metabolic factors in fatigue. Sports Medicine, 13(2), 99-107.

Schmidt, R. (1968). Performance and learning a gross motor skill under conditions of artificially-induced fatigue. Research Quarterly, 40(1), 185-190.

Simmons, R. W. F. & Hardt, A. B. (1973). The effect of alkali ingestion on the performance of trained swimmers. Journal of Sports Medicine, 13, 159-163.

Sjogaard, G. (1986). Water and electrolyte shifts during exercise and their relation to muscular fatigue. Acta Physiologica Scandanavica, 128(Suppl. 556), 129-136.

Sutton, J. R., Jones, N. L., & Toews, C. J. (1981). Effect of pH on muscle glycolysis during exercise. Clinical Science, 61, 331-338.

Tsaneva, N. & Markov, S. (1971). A model of fatigue. London: Taylor & Francis Ltd.

Wilkes, D., Gledhill, N., & Smyth, R. (1983). Effect of acute induced metabolic alkalosis on 800-m racing time. Medicine and Science in Sports and Exercise, 15(4), 277-280.

Williams, M. (1992). Bicarbonate loading. Sports Science Exchange, 4(36).

APPENDICES

APPENDIX A

INFORMED CONSENT FORM

The Effect of Selected Buffering Agents on Performance in the Competitive 1600 Meter Run

Investigators: Lori Avedisian, M.S., R.P.T.; Arthur Guerra; and Anthony Wilcox, Ph.D.

Purpose: The purpose of this investigation is to study the effect of sodium bicarbonate ($NaHCO_3$) ingestion and sodium citrate ingestion by trained track athletes on their performance in a competitive 1600 meter race.

I have received an oral explanation of the study procedures and understand they entail:

1. Tests of maximal oxygen consumption ($\dot{V}O_2$ max)
At the beginning of my participation in the study, I will undergo a test of my aerobic capacity. The test will be conducted on a motorized treadmill, starting at a slow speed and progressing with gradual increases in speed and treadmill elevation until I become too fatigued to continue. The test will take approximately 8 to 12 minutes, with only the final few minutes being at a high intensity.

During the test, I will breathe room air through a mouthpiece so that the amount of oxygen I am using can be determined. My heart rate will be continuously monitored electrocardiographically. Trained laboratory personnel, certified in CPR, will administer the exercise tests.

2. Body composition test using hydrostatic weighing.
My body composition (percent body fat) will be determined using an underwater weighing procedure in a specially designed indoor tank. Water in the tank will be near body temperature (35-37ºC). Sitting on a chair that is suspended from a scale, I will submerse myself following a maximal exhalation, and remain underwater for 3-5 seconds while the scale is read. The procedure will be repeated 5-6 times. I will perform a test to determine my residual volume (volume of air in the lungs following maximal exhalation), which requires me to breathe into a spirometer filled with oxygen for a period of 30-60 seconds. Two or three such trials will be performed.

3. Sixteen hundred meter race tests.
In order to study the effects of ingesting sodium bicarbonate or sodium citrate on 1600 meter running performance, I will run 4 such races against other subjects in the study. One of the races will be after ingesting $NaHCO_3$, one will be following sodium citrate, one after ingestion of $CaCO_3$, and one will be without any treatment. These races will be on a 400 meter synthetic surface track designed for track competition, and they will be timed using accepted track racing procedures. The races will be scheduled at least five days apart to allow for complete recovery between each. Participants will not run during inclement weather. I agree to wear standard racing attire, including shoes for these race tests.

4. Sodium bicarbonate (NaHCO3) treatment, sodium citrate treatment and placebo (CaCO3) procedures

I understand that I will ingest NaHCO3 in a quantity equivalent to 400 mg/kg body weight prior to a 1600 meter race. This quantity of NaHCO3 will be encapsulated in gelatin capsules, and taken with approximately one liter of water 90-120 minutes before the race. I also understand that prior to a separate 1600 meter race, I will ingest 500 mg/kg body weight of sodium citrate. The sodium citrate will be encapsulated in gelatin capsules as above. I also understand that prior to a separate 1600 meter race, I will ingest CaCO3 in a manner similar to that of NaHCO3 ingestion. I will also run another 1600 meter race without prior ingestion of any substance. The order of the four races (treatment A, treatment B, placebo, and control) used as tests for this study will be randomly assigned and administered in a double-blind fashion so that I will not know which substance I am ingesting.

5. Blood Samples

Blood samples will be taken at three times at each of the four racing sessions. The first interval will be approximately two hours prior to the race, the second will be immediately prior to the race, and the final interval will be immediately following completion of the race. A standard hygienic finger-puncture method using a sterile lancet will be used to collect blood samples that will be used to determine blood pH and blood lactate levels. A standard venipuncture using the antecubital vein will be performed using sterile equipment, to collect blood samples that will be used to determine blood bicarbonate concentrations.

6. Subjective Symptom Scale

Immediately after completing each race, I will be asked to complete a brief survey and describe how I physically felt at specific intervals during the race.

7. Risks

There is a remote risk of death associated with the test of maximal oxygen consumption. In large, varied populations, this risk is one death per 10,000 tests. Since I am from a low risk segment of the population (young and healthy), and will be screened to exclude individuals with known symptoms of heart disease, the risk is considerably less. Furthermore, trained personnel will be administering the test and monitoring me for signs of exercise intolerance. There will be a remote risk of injury or death associated with running a one mile race, but this is a level of exertion that I am familiar with, since I am an experienced track athlete and regularly perform this type of activity.

Ingestion of sodium bicarbonate may cause temporary gastrointestinal discomfort (gas, diarrhea, bloating). The chance of experiencing gastrointestinal symptoms is lessened when it is ingested with large quantities of water, which is why I will be drinking one liter when I take the dose. The chronic ingestion of sodium has been shown to increase blood pressure in some individuals. The dose of NaHCO3 I will take will contain 4-6 grams of sodium, an amount similar to the average intake of sodium in the average American diet. Since I do not have high blood pressure and since this study involves a one-time only consumption of NaHCO3, this dose will not pose a health risk to me.

8. Benefits

I will benefit from my participation by contributing to the understanding of the effects of selected buffering agent ingestion on performance during a one mile race, which may have implications for competitive performance. I will also gain information about my aerobic capacity and percent body fat, which may be useful to me in planning my training program.

Participation in this study will involve participating in one laboratory session requiring 45-60 minutes, and four races, each requiring a time commitment of approximately two and one-half hours scheduled about five days apart. The initial laboratory session will consist of determination of maximal aerobic capacity and a test of body composition. On the four racing days, I will report two hours before the race so that blood sampling and ingestion of treatment or placebo can occur. I will remain for a short period of time following completion of the race to allow for collection of blood samples.

My anonymity will be ensured by assigning me a code number upon entry into the study. All data will be recorded using the code number. The list containing the names of the subjects and their code numbers will only be available to the researchers in the study. I will not be identified in any way in the presentation or publication of the results of the study.

Persons who have ever had hepatitis B or C, who have tested positive for HIV or any AIDS virus, or persons having AIDS should not donate body fluids. Persons at risk for getting and spreading any AIDS virus should not participate in this investigation. You are at risk is:

* you are a man who had sex with another man since 1977, even one time.
* you have shared a needle, even one time, to inject drugs or medication.
* you have taken clotting factor concentrates for a bleeding disorder such as hemophilia.
* you have ever had a positive test for any AIDS virus or hepatitis B or C or any AIDS antibody.
* you have had sex with any person described above.
* you have had sex with a male or female prostitute since 1977.

Questions about the research or any aspects of my participation in it should be directed to Dr. Anthony Wilcox (737-5922). I understand that the University does not provide a research subject with compensation or medical treatment in the event the subject is injured as a result of participation in the research project.

I have been completely informed and understand the nature and purpose of the research project. The researchers have offered to answer any further questions that I may have. I understand that my participation in this study is completely voluntary and I may withdraw from the study at any time without prejudice or loss of benefits to which my participation entitles me.

I have read the foregoing and agree to participate in this study.

_____ _____
Subject's Signature **Date**

_____
Subject's Address

_____
Subject's Phone Number

_____ _____
Investigator's Signature **Date**

APPENDIX B

SUBJECTIVE SYMPTOM SCALE

Subject identification number _____

Please indicate the presence and severity of any symptoms of gastrointestinal (GI) discomfort that you experienced during the 1600 meter run today. Please check the appropriate response, identify all symptoms you experienced, and identify the severity of each symptom by indicating if it was: mild, moderate, or severe.

1. I did not experience any symptoms of GI discomfort. _____

2. I did experience symptoms of GI discomfort during the run. _____

3. If you did experience symptoms, continue to number 4.

4. List each of the GI symptoms you experienced, and indicate the severity of each using the choices (mild, moderate, or severe).

5. Try to remember back to the actual run, and describe <u>how</u> you felt physically at the following points in the race:

400 meters-

800 meters-

1200 meters-

1600 meters-

The space below has been provided for you to make any additional comments concerning the run.

APPENDIX C

SUBSTANCE INGESTED

			substance ingested during			
subj.	sex	design	race 1	race 2	race 3	race 4
1	M	(C)	HCO3	citrate	placebo	control
2	M	(A)	control	placebo	citrate	HCO3
3	M	(D)	citrate	control	placebo	HCO3
4	M	(B)	placebo	HCO3	control	citrate
5	M	(A)	control	placebo	citrate	HCO3
6	M	(C)	HCO3	citrate	placebo	control
7	M	(A)	control	placebo	citrate	HCO3
8	F	(A)	control	placebo	citrate	HCO3
9	F	(D)	citrate	control	placebo	HCO3
10	F	(C)	HCO3	citrate	placebo	control
11	F	(B)	placebo	HCO3	control	citrate
12	F	(D)	citrate	control	placebo	HCO3

Design
A 1,2,4,3
B 2,3,1,4
C 3,4,2,1
D 4,1,2,3
A 1,2,4,3

1=control
2=placebo (CaCO3)
3=HCO3
4=citrate

APPENDIX D

SUBSTANCE DOSE

Subject	wt. (kg)	# placebo capsules	HCO3 dose (g) (# capsules)	citrate dose (g) (# capsules)
1	69.0	18	27.6 (16)	29.0 (16)
2	78.0	20	31.2 (17)	39.0 (24)
3	61.0	16	24.4 (14)	30.5 (18)
4	73.0	19	29.2 (17)	36.5 (22)
5	70.5	16	28.2 (14)	32.3 (21)
6	68.0	18	27.2 (16)	34.0 (21)
7	66.0	15	26.4 (14)	33.0 (17)
8	50.0	12	20.0 (12)	25.0 (16)
9	50.0	12	20.0 (11)	25.0 (14)
10	51.0	15	20.4 (15)	25.5 (16)
11	58.0	15	23.2 (14)	29.0 (16)
12	63.5	14	25.4 (13)	31.8 (16)

APPENDIX E

ACID-BASE RESULTS

HCO3 units=mmol/l
lactate units=mmol/l

Subj.	Race	Blood Values	Pre-Race (A)	Post-Race (B)
1	1	pH	7.431	7.191
		HCO3	33.9	17.2
		lactate	3.6	17.16
	2	pH	7.489	7.165
		HCO3	32.6	20.7
		lactate	1.92	15.63
	3	pH	7.357	7.113
		HCO3	28.6	10.7
		lactate	1.11	20.61
	4	pH	7.345	7.127
		HCO3	29.5	20.2
		lactate	1.35	13.44
2	1	pH	7.405	7.143
		HCO3	30.9	18.9
		lactate	5.31	14.91
	2	pH	7.347	7.088
		HCO3	26.1	16.2
		lactate	1.56	15.18
	3	pH	7.405	7.143
		HCO3	33.3	19.7
		lactate	1.92	17.52
	4	pH	7.337	7.153
		HCO3	33.8	21.9
		lactate	3.21	16.11

ACID-BASE RESULTS

HCO3 units=mmol/l
lactate units=mmol/l

Subj.	Race	Blood Values	Pre-Race (A)	Post-Race (B)
3	1	pH	7.393	7.064
		HCO3	31.0	10.8
		lactate	2.52	18.21
	2	pH	7.324	7.058
		HCO3	28.2	8.6
		lactate	1.20	19.98
	3	pH	7.359	7.021
		HCO3	34.1	12.6
		lactate	1.50	21.69
	4	pH	7.335	7.041
		HCO3	28.4	13.0
		lactate	0.99	12.694
4	1	pH	7.322	7.028
		HCO3	27.4	13.4
		lactate	-----	-----
	2	pH	7.415	7.171
		HCO3	35.7	15.7
		lactate	-----	16.70
	3	pH	7.326	7.091
		HCO3	27.9	12.3
		lactate	0.90	11.99
	4	pH	7.363	7.143
		HCO3	31.6	16.1
		lactate	0.65	19.20
5	1	pH	7.352	7.156
		HCO3	27.2	11.6
		lactate	-----	-----
	2	pH	7.380	7.026
		HCO3	31.80	14.80
		lactate	-----	-----
	3	pH	7.392	7.157
		HCO3	29.9	15.7
		lactate	1.74	18.16
	4	pH	7.445	7.140
		HCO3	31.5	11.7
		lactate	1.10	22.56

ACID-BASE RESULTS

HCO3 units=mmol/l
lactate units=mmol/L

Subj.	Race	Blood Values	Pre-race (A)	Post-race (B)
6	1	pH	7.381	7.021
		HCO3	36.5	17.4
		lactate	1.08	20.34
	2	pH	7.319	7.111
		HCO3	32.1	19.2
		lactate	1.71	11.94
	3	pH	7.319	6.881
		HCO3	31.9	15.9
		lactate	1.83	20.13
	4	pH	7.293	6.919
		HCO3	29.4	14.8
		lactate	1.26	16.5
7	1	pH	7.2	7.013
		HCO3	30.2	11.8
		lactate	3.09	16.77
	2	pH	7.244	7.051
		HCO3	31.3	15.0
		lactate	2.58	16.41
	3	pH	7.272	7.074
		HCO3	32.3	17.0
		lactate	3.27	16.65
	4	pH	7.271	7.182
		HCO3	31.9	23.0
		lactate	3.21	12.60
8	1	pH	7.324	7.071
		HCO3	27.6	18.2
		lactate	1.03	11.17
	2	pH	7.393	7.226
		HCO3	27.3	17.7
		lactate	0.85	7.71
	3	pH	7.470	7.313
		HCO3	30.9	21.5
		lactate	1.11	9.87
	4	pH	7.459	7.277
		HCO3	31.5	19.9
		lactate	1.17	8.96

ACID-BASE RESULTS

HCO3 units=mmol/l
lactate units=mmol/l

Subj.	Race	Blood Values	Pre-race (A)	Post-race (B)
9	1	pH	7.407	7.283
		HCO3	31.8	18.9
		lactate	0.90	10.05
	2	pH	7.360	7.267
		HCO3	25.8	14.1
		lactate	0.70	7.80
	3	pH	7.481	7.394
		HCO3	29.2	17.2
		lactate	0.93	9.8
	4	pH	7.366	7.308
		HCO3	26.0	15.1
		lactate	1.088	6.94
10	1	pH	-----	-----
		HCO3	-----	-----
		lactate	1.55	7.94
	2	pH	-----	-----
		HCO3	-----	-----
		lactate	1.49	8.18
	3	pH	-----	-----
		HCO3	-----	-----
		lactate	2.80	8.18
	4	pH	-----	-----
		HCO3	-----	-----
		lactate	1.64	10.26
11	1	pH	7.362	7.120
		HCO3	30.1	16.8
		lactate	3.21	14.49
	2	pH	7.371	7.240
		HCO3	34.7	19.8
		lactate	1.32	12.78
	3	pH	7.322	7.094
		HCO3	27.6	15.1
		lactate	1.77	13.11
	4	pH	7.356	7.172
		HCO3	32.1	21.5
		lactate	1.59	12.96

ACID-BASE RESULTS

HCO3 units=mmol/l
lactate units=mmol/l

Subj.	Race	Blood Values	Pre-Race (A)	Post-Race (B)
12	1	pH	7.324	7.122
		HCO3	31.2	15.7
		lactate	2.22	13.6
	2	pH	7.271	7.063
		HCO3	26.5	12.3
		lactate	1.56	12.33
	3	pH	7.262	7.096
		HCO3	28.7	18.4
		lactate	1.89	12.00
	4	pH	7.360	7.180
		HCO3	29.0	17.0
		lactate	1.62	14.79

APPENDIX F

PERFORMANCE TIME RESULTS

Subj.	Race	Performance Time (min:sec) at 400(m)	800(m)	1200(m)	1600(m)
1	1	1:09.9	2:27.4	3:44.7	4:54.7
	2	1:10.0	2:29.0	3:48.0	5:00.2
	3	1:10.1	2:25.0	3:40.9	4:53.0
	4	1:13.8	2:30.2	3:46.4	5:01.8
2	1	1:13.3	2:31.0	3:52.0	5:08.4
	2	1:10.7	2:26.7	3:48.9	5:10.3
	3	1:14.5	2:31.3	3:50.4	5:10.8
	4	1:11.5	2:28.1	3:47.3	5:06.2
3	1	1:11.0	2:31.4	3:50.5	5:07.3
	2	1:14.9	2:31.9	3:51.2	5:06.1
	3	1:14.5	2:31.3	3:50.4	5:10.8
	4	1:12.3	2:31.0	3:52.4	5:18.1
4	1	1:08.2	2:27.8	3:43.8	4:56.6
	2	1:09.8	2:24.6	3:40.9	4:52.2
	3	1:11.3	2:25.8	3:40.7	4:50.1
	4	1:13.7	2:28.7	3:43.6	4:53.3
5	1	1:08.4	2:25.4	3:55.8	5:21.2
	2	1:12.1	2:27.5	3:47.7	5:07.0
	3	1:12.2	2:30.2	3:51.1	5:07.4
	4	1:13.4	2:28.9	3:43.8	4:54.0
6	1	1:08.3	2:19.8	3:33.4	4:43.5
	2	1:09.5	2:26.1	3:46.9	5:04.1
	3	1:07.9	2:18.6	3:32.1	4:44.4
	4	109.5	2:22.6	3:35.8	4:46.3

PERFORMANCE TIME RESULTS

Subj.	Race	Performance time (min:sec) at 400 (m)	800 (m)	1200 (m)	1600 (m)
7	1	1:08.5	2:20.4	3:34.0	4:48.0
	2	1:09.9	2:24.9	3:42.5	4:56.2
	3	1:08.7	2:22.3	3:40.4	4:58.5
	4	1:10.2	2:23.4	3:37.9	4:48.7
8	1	1:25.2	2:50.3	4:16.0	5:44.8
	2	1:23.5	2:49.5	4:17.0	5:42.7
	3	1:25.8	2:51.2	4:15.3	5:35.9
	4	1:22.7	2:47.0	4:11.5	5:35.5
9	1	1:25.6	2:54.3	4:26.0	5:54.3
	2	1:23.8	2:53.5	4:27.6	6:00.9
	3	1:27.6	2:58.7	4:29.1	5:55.6
	4	1:28.3	2:59.8	4:31.2	6:02.6
10	1	1:26.3	2:58.0	4:34.2	6:04.3
	2	1:25.2	2:59.8	4:36.4	6:06.2
	3	1:26.6	2:57.9	4:31.7	6:06.3
	4	1:28.3	2:59.8	4:31.2	5:58.2
11	1	1:19.1	2:43.5	4:06.3	5:28.0
	2	1:18.8	2:44.7	4:12.3	5:38.5
	3	1:19.9	2:43.5	4:08.6	5:31.8
	4	1:25.1	2:50.0	4:11.8	5:33.1
12	1	1:18.3	2:47.0	4:16.5	5:42.0
	2	1:15.6	2:43.2	4:14.7	5:40.1
	3	1:18.2	2:50.5	4:22.7	5:47.7
	4	1:22.2	2:53.3	4:26.7	5:52.0

APPENDIX G

HYDROSTATIC WEIGHING RESULTS

Subj.	Dry wt. (lbs)	Ht. (in)	UW wt. (lbs)	RV (l)	Temp (° C)	Body Fat (%)
1	154.0	69.0	3.72	1.60	34	9.8
2	173.0	73.0	4.02	2.22	36	8.6
3	139.0	68.0	3.94	1.43	36	3.4
4	164.0	73.0	4.40	1.64	36	7.8
5	157.0	72.5	3.18	1.85	36	12.7
6	153.5	70.0				6.5*
7	144.0	69.0				7.9*
8	110.0	66.0	2.50	1.29	36	9.9
9	113.0	67.0	1.82	1.52	37	14.9
10	113.5	65.5	6.16	1.49	37	12.7
11	128.0	66.5	2.98	1.89	36	6.0
12	157.0	68.0				19.3*

* Obtained via Lange calipers using the Sum of Seven Skinfold Technique (Jackson & Pollock, 1977).

APPENDIX H

ENVIRONMENTAL CONDITIONS

Group	Trial	Dry Temp (°F)	Wet bulb (°F)	Conditions
	1	81.0	67.5	Sun, breezy
1	2	77.0	67.0	Sun, breezy
	3	85.5	71.5	Sun
	4	74.5	66.5	Sun, breezy
Mean		79.5	68.1	
SD		3.8	1.5	
	1	54.0	57.0	Windy
2	2	48.0	50.0	Windy, overcast
	3	57.0	53.0	Windy, overcast
	4	61.0	53.0	Clear, breezy
Mean		55.0	53.3	
SD		4.0	1.9	
	1	52.0	49.0	Clear, sunny
3	2	43.0	39.0	Cool, windy
	3	42.0	39.0	Cool, windy
	4	48.0	50.0	Drizzle, mild
Mean		46.3	44.3	
		3.8	5.3	