ABSTRACT


Aerobically trained females (n=15) performed 4 supramax exercise bouts 1 hr after orally ingesting either 200 mg/kg body wt. NaHCO₃ or 5.0 g. NaCl, to determine the effects of NaHCO₃ on blood lactate concentration. Using a double blind protocol, each S performed 4 randomized conditions: 1) NaCl-30 sec, 2) NaCl-time to exhaustion (TTE), 3) NaHCO₃-30 sec, 4) NaHCO₃-TTE. The Wingate Anaerobic Test was used to elicit supramax exercise. 4 ml samples of blood were drawn from the antecubital vein 30 min post-ingestion and 5 min post-exercise in each of the 4 conditions. Lactic acid (LA) was determined using the enzymatic method described by Sigma Chemical Co. (1977). Pedal revolutions were recorded for determination of total work (TW). Statistical analysis of the data revealed no sig (p > .05) diff in TW, post-ingestion LA, NaHCO₃ (7 mg%), NaCl (6 mg%), and TTE-LA, NaHCO₃ (88 mg%), NaCl (81 mg%). However, in the 30 sec trial, LA levels were sig (p < .05) higher with NaHCO₃ (73 mg%), than NaCl (62 mg%). This increased LA with NaHCO₃ was attributed to improved efflux of LA across the sarcolemma. The explanation for no sig diff in TTE-LA was unclear, perhaps an error in test protocol. Based on these results, it appears that while NaHCO₃ may improve efflux of LA, it does not improve performance in short-term supramax exercise.
THE EFFECTS OF SODIUM BICARBONATE ON LACTATE LEVELS DURING SUPRAMAXIMAL EXERCISE

A Thesis Presented to The Graduate Faculty University of Wisconsin-La Crosse

In Partial Fulfillment of the Requirements for the Master of Science Degree

Ellen H. Brewster December, 1984
UNIVERSITY OF WISCONSIN–LA CROSSE
College of Health, Physical Education, and Recreation
La Crosse, Wisconsin 54601

Candidate: Ellen H. Brewster

We recommend acceptance of this thesis in partial fulfillment of this
candidate's requirements for the degree: Master of Science, Adult Fitness/
Cardiac Rehabilitation.

The candidate has completed her oral report.

Nancy Kay Butts
Thesis Committee Chairperson

September 13, 1984
Date

Karen Wood
Thesis Committee Member

September 10, 1984
Date

Keith A. Keeney
Thesis Committee Member

September 10, 1984
Date

This thesis is approved for the College of Health, Physical Education,
and Recreation.

John C. Mitchell
Dean, College of Health, Physical Education
and Recreation

Sept. 13, 1984
Date

Deane of Graduate Studies

Sept. 13, 1984
Date
ACKNOWLEDGEMENTS

To be writing acknowledgements seems unbelievable! In June of 1983, completing a thesis seemed like an insurmountable task. I have many people to thank for their contribution in completing this thesis.

First I would like to thank Doug Crowell, my partner in this thesis project. Thanks for getting me interested in NaHCO$_3$!

I would like to express my gratitude to Dr. N.K. Butts for making this thesis a reality. Thanks for your expertise, motivation, and patience, all of which combined to bring out the best in me.

Thanks also to Dr. Kim Wood and Dr. Keith Kensinger. Your scientific and grammatical assistance as well as your flexibility and cooperation in reading my thesis have been appreciated.

I would also like to thank Dixie Talcott and Carol Callahan for their help in drawing and analyzing the blood. Thanks for all your help Carol, without it I would still be testing!

Thanks are in order for all my classmates. You helped make this hectic year exciting and fun, may you all end up in Madison!

Finally, I would like to thank my parents, Mother and Martin, and my family for all their support, financially and emotionally. This thesis would not have been possible without all your support!
# TABLE OF CONTENTS

<table>
<thead>
<tr>
<th>CHAPTER</th>
<th>PAGE</th>
</tr>
</thead>
<tbody>
<tr>
<td>I. INTRODUCTION</td>
<td>1</td>
</tr>
<tr>
<td>Purpose</td>
<td>3</td>
</tr>
<tr>
<td>Hypothesis</td>
<td>3</td>
</tr>
<tr>
<td>Assumptions</td>
<td>3</td>
</tr>
<tr>
<td>Delimitations and Limitations</td>
<td>4</td>
</tr>
<tr>
<td>Definition of Terms</td>
<td>4</td>
</tr>
<tr>
<td>II. RELATED LITERATURE</td>
<td>6</td>
</tr>
<tr>
<td>Anaerobic Metabolism</td>
<td>6</td>
</tr>
<tr>
<td>Lactate Production During Exercise</td>
<td>9</td>
</tr>
<tr>
<td>Measurement of Lactate</td>
<td>11</td>
</tr>
<tr>
<td>Acid-Base Balance During Exercise</td>
<td>12</td>
</tr>
<tr>
<td>Lactate and H⁺ ion Efflux</td>
<td>14</td>
</tr>
<tr>
<td>Training Effects on Lactate</td>
<td>17</td>
</tr>
<tr>
<td>Lactate as a Limiting Factor in Supramaximal Exercise</td>
<td>17</td>
</tr>
<tr>
<td>Effects of Induced Alkalosis and Acidosis</td>
<td>18</td>
</tr>
<tr>
<td>Positive Effects</td>
<td>18</td>
</tr>
<tr>
<td>No Effect</td>
<td>23</td>
</tr>
<tr>
<td>Summary</td>
<td>26</td>
</tr>
<tr>
<td>III. METHODS</td>
<td>28</td>
</tr>
<tr>
<td>Introduction</td>
<td>28</td>
</tr>
<tr>
<td>Subject Selection</td>
<td>28</td>
</tr>
<tr>
<td>Procedures</td>
<td>29</td>
</tr>
<tr>
<td>Practice Sessions</td>
<td>29</td>
</tr>
<tr>
<td>Testing Sessions</td>
<td>30</td>
</tr>
<tr>
<td>Ingestion Procedures</td>
<td>30</td>
</tr>
<tr>
<td>Pre-exercise Blood Draw</td>
<td>30</td>
</tr>
<tr>
<td>Exercise Test</td>
<td>31</td>
</tr>
<tr>
<td>Post-exercise Blood Draw</td>
<td>32</td>
</tr>
<tr>
<td>Determination of Blood Lactate</td>
<td>33</td>
</tr>
<tr>
<td>Statistical Treatment of Data</td>
<td>33</td>
</tr>
<tr>
<td>IV. RESULTS AND DISCUSSION</td>
<td>35</td>
</tr>
<tr>
<td>Introduction</td>
<td>35</td>
</tr>
<tr>
<td>Descriptive Characteristics</td>
<td>35</td>
</tr>
<tr>
<td>Pre-exercise Blood Lactate Concentration</td>
<td>38</td>
</tr>
<tr>
<td>30 Second Blood Lactate Concentration</td>
<td>40</td>
</tr>
<tr>
<td>TTE Blood Lactate Concentration</td>
<td>43</td>
</tr>
<tr>
<td>Lactate Accumulation</td>
<td>45</td>
</tr>
<tr>
<td>Performance</td>
<td>47</td>
</tr>
<tr>
<td>Summary</td>
<td>49</td>
</tr>
<tr>
<td>CHAPTER</td>
<td>PAGE</td>
</tr>
<tr>
<td>---------</td>
<td>------</td>
</tr>
<tr>
<td>V. SUMMARY, CONCLUSIONS AND RECOMMENDATIONS</td>
<td>50</td>
</tr>
<tr>
<td>Summary</td>
<td>50</td>
</tr>
<tr>
<td>Conclusions</td>
<td>51</td>
</tr>
<tr>
<td>Recommendations for future study</td>
<td>53</td>
</tr>
<tr>
<td>REFERENCES CITED</td>
<td>54</td>
</tr>
<tr>
<td>APPENDICES</td>
<td>60</td>
</tr>
<tr>
<td>A. Activity Questionnaire</td>
<td>61</td>
</tr>
<tr>
<td>B. Informed Consent</td>
<td>64</td>
</tr>
<tr>
<td>C. Lactic Acid Determination Procedures</td>
<td>67</td>
</tr>
</tbody>
</table>
LIST OF TABLES

<table>
<thead>
<tr>
<th>TABLE</th>
<th>PAGE</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Means, standard deviations, and ranges of age, height, total body weight, dosage, and resistance of the female subjects</td>
<td>36</td>
</tr>
<tr>
<td>2. Means, standard deviations, and ranges of blood lactate concentrations pre-exercise</td>
<td>38</td>
</tr>
<tr>
<td>3. Mean resting lactate concentrations found in the literature</td>
<td>39</td>
</tr>
<tr>
<td>4. Means, standard deviations, and ranges of blood lactate concentrations after 30 seconds of supramaximal exercise</td>
<td>40</td>
</tr>
<tr>
<td>5. Means, standard deviations, and ranges of blood lactate concentrations after exhaustive supramaximal exercise</td>
<td>43</td>
</tr>
</tbody>
</table>
CHAPTER I

INTRODUCTION

The late Vince Lombardi was once quoted as saying "winning isn't everything - it's the only thing." For the elite athlete, amateur or professional, this statement is true. The elite athlete's livelihood is dependent on winning. Winning ensures money through sponsorships and endorsements. At the international level the difference between winning and losing can be tenths or hundredths of a second. Therefore, any ergogenic aid that can provide that extra advantage is crucial.

The passage of Title IX in 1972 has led to the emergence of a new group of elite athletes...females. Increasing numbers of females are now competing at an international level and are making a living through competitive athletics. Winning is as crucial to female athletes as it is to male athletes. Thus, more and more females are relying on ergogenic aids to enhance their performance.

The emergence of females on the international athletic scene has opened up new areas of study in exercise physiology. It is imperative that research on female athletes keep pace with this growth and development. One area requiring study is the effect of sodium bicarbonate (NaHCO₃) on supramaximal exercise.

Although the mechanisms which limit performance in an endurance event are well established (Astrand & Rodahl, 1977; Fox & Mathews, 1981; Gollnick & Hermansen, 1973), the limiting factor(s) in maximal work of short duration is not as clear cut. Some claim lactic acid, the end product of anaerobic glycolysis, is the limiting factor because it accumulates in the

Several researchers have examined the effect lactate has on short-term supramaximal performance by inducing alkalosis and acidosis to alter acid-base balance (Balberman & Roby, 1983; Costill, Verstappen, Kuipers, Jansson & Fink, 1983; Dennig, Talbot & Dill, 1931; Johnson & Black, 1953; Jones, Sutton, Taylor & Toews, 1975; Jones et al., 1977; Katz, Costill, King, Hargreaves & Fink, 1984; Kindermann et al., 1977; Margaria, Aghemo & Sassi, 1971b, McCartney et al., 1983; Poulus, Doctor & Westra, 1974; Rupp, Bartels, Zuelzer & Fox, 1983; Sutton et al., 1981; Wilkes, Gledhill & Smyth, 1983). The results of these studies are conflicting.

None of the above studies used exclusively female subjects. However, based on the work of Jacobs and Tesch (1981), there is reason to believe that females may react differently than males to sprint exercise. The difference may lie in the reduced function or key skeletal enzymes (Jacobs, Bar-Or, Karlsson, Dotan, Tesch, Kaiser & Inbar, 1982) and in reduced skeletal muscle mass (Fox & Mathews, 1981). These differences may affect the ability of females to buffer lactate.

It is because of the conflicting evidence in the literature and the paucity of data on females that this study was undertaken. There is a need for more research on the effects of induced alkalosis on lactate accumulation during supramaximal exercise. Is NaHCO₃ an effective ergogenic aid? Is it effective on females?
Purpose

This study was conducted to determine the effects of ingestion of sodium bicarbonate (NaHCO₃) or induced alkalosis on lactate levels following supramaximal exercise of short duration.

Hypothesis

The major hypothesis to be tested at the 0.05 level of confidence was: there will be no significance in blood lactate concentration following supramaximal exercise between experimental (NaHCO₃) and control (NaCl) treatments of equivalent duration. It was further hypothesized that blood lactate concentration would not significantly differ following supramaximal exercise between treatments, NaHCO₃ or NaCl, and exercise trials, 30 seconds and time to exhaustion (TTE).

Assumptions

The following assumptions were made for this study: (1) the subjects would perform the supramaximal exercise tests to the best of their ability; (2) the subjects would not alter their daily living habits (particularly their dietary habits) in any way that would significantly alter the results; (3) the dosage of sodium bicarbonate or sodium chloride would be eliminated from the subject's body before further testing (no sooner than 72 hours); (4) the Monark bicycle ergometer and pedal revolution counter would function accurately and consistently during each exercise test; (5) the analysis of blood lactate concentration was performed accurately and consistently; (6) the dosages of sodium bicarbonate and sodium chloride would taste similar enough that the subjects would not be able to
differentiate between the two; and, (7) the subjects were considered aerobically trained if they exceeded the minimal recommendations set forth by the American College of Sports Medicine for quantity and quality of exercise for developing and maintaining fitness (American College of Sports Medicine, 1978).

**Delimitations and Limitations**

The delimitations and limitations in this study were: (1) the subjects were female volunteers, and not randomly selected; (2) different motivational levels of the subjects on each of the four exercise tests could have influenced the results; (3) human error may have existed in the measurement of lactate in the blood; and, (4) the subjects were aerobically trained.

**Definition of Terms**

*Acidosis* - altering the acid-base balance of the blood plasma so that pre-exercise pH values are decreased below normal.

*Alkalosis* - altering the acid-base balance of blood plasma so that pre-exercise pH values are elevated above normal through ingestion of sodium bicarbonate.

*Anaerobic work* - "any metabolic process by which energy is delivered to the contractile apparatus of skeletal muscle that does not depend upon an immediate source of oxygen," (Gollnick & Hermansen, 1978, p. 2).

*Blood lactate concentration* - the amount of lactic acid (mg/100 ml) in venous blood taken from the antecubital vein in the arm five minutes post-exercise.

*Kilopond meter (kpm)* - a unit of work, as described by force times
distance. On the Monark, it is the kilograms of resistance times six meters times the revolutions per minute.

Monark bicycle ergometer - a stationary bicycle that gives varying resistances through placement of weights on a pan, thus tightening a belt which applies friction to the wheel. Increasing the amount of weight on the pan increases the amount of work required to move the wheel.

Supramaximal exercise - an individualized workload based on kilogram body weight. This workload was derived from the Wingate Anaerobic Test (Kraemer & Fleck, 1982), a test used to evaluate capacity in short-term, exhaustive work.

Total work - the total number of pedal revolutions achieved within 30 seconds or time to exhaustion workloads.
CHAPTER II
RELATED LITERATURE

Introduction

In order to better understand the significance of sodium bicarbonate (NaHCO₃) as a possible ergogenic aid, a review of the literature was necessary. This review discusses basic principles of: (1) anaerobic metabolism; (2) lactate production; (3) measurement of lactate; (4) acid-base balance during exercise; (5) lactate and H⁺ ion efflux; (6) training effects on lactate; and, (7) lactate as a limiting factor in supramaximal exercise. In addition, studies previously performed which induced alkalosis and acidosis have been reviewed.

Anaerobic Metabolism

In order for persons to perform work, chemically bound energy, adenosine triphosphate (ATP), must be converted into mechanical work by the muscles (Karlsson, 1971). Energy released by the breakdown of ATP "represents the immediate source of energy that can be used by the muscle cell to perform its work" (Fox & Mathews, 1981, p. 12). There are three pathways available for the conversion of ATP to mechanical work. Two pathways, the ATP-PC and anaerobic glycolysis systems, operate anaerobically or without oxygen. The third pathway, oxidative phosphorylation, operates aerobically thus requiring oxygen. It is the availability of oxygen to the muscle cell that determines which pathway will be utilized (Astrand & Rodahl, 1977). In maximal work of short duration availability of oxygen is limited, therefore the anaerobic pathways, of ATP-PC (phosphagen system)
and anaerobic glycolysis (lactic acid system) must supply the energy (Fox & Mathews, 1981). While utilization of the anaerobic pathways provides an immediate source of ATP, the end product of anaerobic glycolysis, lactic acid, is often considered the limiting factor in maximal exercise of short duration.

The first 45 - 90 seconds of most exercise is anaerobic, due to the rapid adjustments in cardiac output required to go from rest to exercise and the unavailability of oxygen (Astrand & Rodahl, 1977). The ATP-PC system provides the most readily available supply of ATP for use by skeletal muscle (Fox & Mathews, 1981). This is due to the storage of ATP and PC directly in muscle tissue. Estimates of ATP and PC concentration in skeletal muscle range from 4 - 6 to 5 - 20 millimoles (mM)/kg wet muscle (Karlsson, 1980; Kraemer & Fleck, 1982).

At the onset of muscular contraction, the ATP levels fall, stimulating the oxidation of creatine phosphate (CP). When oxidized, the CP molecule liberates energy and phosphate. This energy and phosphate then combines with adenosine diphosphate (ADP) to form ATP and creatine (C) (Luciano, Vander & Sherman, 1978). The reaction is reversible, and forces the rapid formation of ATP from CP and ADP only when ATP levels are falling.

The utilization of ATP and CP stores during exercise differs. While CP stores are virtually depleted following exercise, ATP stores are depleted only to 40% of their resting value (Gollnick & Hermansen, 1973). This suggests that while the CP molecule's main function is to resynthesize ATP for energy, not all ATP stored in skeletal muscle is available for muscular contraction.

The phosphagen energy system, while capable of providing quick energy, is incapable of providing large amounts of energy over time on its own.
However, the ATP-PC system has a large power capacity, in that it can provide a large amount of energy per unit time (Kraemer & Fleck, 1982). According to Fox and Mathews (1981), the total energy yield from the ATP-PC system constitutes 5.7 – 6.9 kcals, enough energy for approximately 10 seconds of all-out exercise. Thus, we rely on the other anaerobic pathway, anaerobic glycolysis, to provide additional energy for supramaximal work of short duration.

Anaerobic glycolysis extends the amount of time one can perform supramaximal exercise without oxygen. The energy provided by this system is not stored but derived through the process of anaerobic glycolysis.

Anaerobic glycolysis refers to the breakdown of glucose or glycogen to pyruvic acid. The site of glycolysis and lactic acid production is the sarcoplasm of the muscle cell. An important reaction in the pathway involves the transfer of two hydrogen atoms to a molecule called nicotinamide adenine dinucleotide (NAD) to form NADH₂. The NADH₂ must then transfer the H⁺ atoms to another molecule to re-form NAD, which is necessary for the breakdown of glucose, enabling glycolysis to continue in the muscle cell. In the presence of oxygen, NADH₂ transfers the H₂ to molecular oxygen, and the end product of glycolysis becomes pyruvic acid. However, when exercise is intense and an insufficient amount of oxygen is available for the transfer of hydrogen atoms, NADH₂ transfers its hydrogen to pyruvic acid, forming lactic acid. Thus, lactic acid becomes the end product of anaerobic glycolysis because of its ability to accept hydrogen atoms and regenerate NAD from NADH₂ (Luciano et al., 1978).

The anaerobic glycolytic pathway utilizes exclusively carbohydrates to generate energy. Glucose circulating in blood and glycogen contained
in muscle are the primary substrates. The amount of energy derived from anaerobic glycolysis depends in part on the amount of glycogen stored in skeletal muscle (Jacobs, 1981). Normally between 13 – 15 grams of glycogen are stored in every kg of muscle (Fox & Mathews, 1981), however, glycogen stores are affected by the diet and training.

Anaerobic glycolysis is the preferred energy pathway both at the onset of exercise and during intense exercise. Although the energy yield is smaller, it operates at a much higher rate. Therefore, while anaerobic glycolysis requires more glucose to produce ATP, it produces the ATP almost twice as fast (Luciano et al., 1978). The advantage of the anaerobic pathway is speed, however the metabolic cost of this speed is lactic acid build-up in the muscle and blood. Excessive accumulation of lactate eventually reduces or stops the activity (Kraemer & Fleck, 1982).

**Lactic Acid Production during Exercise**

When anaerobic metabolism is relied upon to supply energy, lactic acid is produced in the muscle cell. Lactic acid, which dissociates into lactate and $\text{H}^+$, accumulates in the muscle and then diffuses into the blood (Gollnick & Hermansen, 1973). Due to the inability of aerobic oxidation to "catch-up" with the energy demand, the lactate accumulation is large in heavy or supramaximal exercise (Astrand & Rodahl, 1977).

There is a small but constant amount of lactate in the blood at all times. At rest, it is estimated to be 3 – 12 mg% (Fox & Mathews, 1981; Gollnick & Hermansen, 1973; Karlsson & Saltin, 1970; Sigma Chemical Company, 1977). It is assumed that in the resting state lactic acid is re-synthesized back to glycogen, mainly in the liver and kidneys.
During exercise of moderate intensity (50 - 85% \( \bar{VO}_2 \) max) accumulation of lactate in the blood occurs rapidly in the first five to ten minutes. If the exercise is sustained for over 10 minutes, the accumulation of lactate in the blood levels off or decreases due to oxidation of lactate back to glycogen. However, constant levels of lactate obtained with moderate exercise may be maintained for 30 minutes or more (Gollnick & Hermansen, 1973). In high intensity exercise (90% or more of \( \bar{VO}_2 \) max), the increase in blood lactate concentration is continuous until "the subjective feeling of exhaustion or fatigue is incompatible with continuation of work" (Gollnick & Hermansen, 1973, p. 15).

Lactic acid production is cumulative and the highest levels are found following intermittent exercise with supramaximal bursts of effort (Karlsson, 1971; Osnes & Hermansen, 1972). The amount of lactate produced depends not only on the intensity and duration of exercise (Karlsson, 1971; Margaria et al., 1971b), but on the amount of muscle mass available to produce energy anaerobically. Typical peak blood lactate values during supramaximal exercise are 200 mg% or greater in males (Fox & Mathews, 1981; Gollnick & Hermansen, 1973; Karlsson, 1980).

Due to the fact that females tend to have a smaller muscle mass than their male counterparts, "females tend to have lower levels of lactic acid in their blood following maximal exercise than do males" (Fox & Mathews, 1981, p. 355). Komi and Karlsson (1978) also related lower peak lactate levels in women to the decreased glycolytic and contractile properties of their skeletal muscle. In more recent studies, the reduced glycolytic capacity of female skeletal muscle has been attributed to reduced function of key enzymes such as phosphorylase (Komi & Karlsson, 1979) and phosphofructokinase (Nygaard, 1981).
The fact that females accumulate less lactate than males during equivalent work is well documented (Jacobs et al., 1982; Jacobs, Tesch, Bar-Or, Karlsson & Dotan, 1983; Karlsson & Jacobs, 1981; Komi & Karlsson, 1978). Komi and Karlsson (1978) found that females accumulated 72.7% the lactate males accumulate for similar exercise. Typical peak blood lactate values for females during supramaximal exercise range from 90 – 120 mg% (Astrand, 1960; Berg & Keul, 1981; Hagerman, Fox, Connors & Pompei, 1974).

Measurement of Lactate

To determine the extent of anaerobic metabolism, measurement of lactate concentration is performed either directly from the muscle or indirectly in venous blood. Direct measurement of lactate concentration requires a muscle biopsy. An indirect and less obtrusive method of determining lactate concentration is to draw a sample of venous blood since it has been shown that a lactate concentration gradient exists from muscle to blood (Hubbard, 1973; Hultman & Sahlin, 1980; Jones, 1980; Jorfeldt, Juhlin-Dannfeldt & Karlsson, 1978; Karlsson, 1971). Some controversy has arisen, however, when blood lactate concentrations have been extrapolated to identify lactate levels.

The controversy evolves around the observation that two to three times more lactate has been found in working muscles as compared to blood immediately following brief maximum exercise (Karlsson, 1971). However, when exercise is prolonged, the muscle to blood gradient equilibrates. Gollnick and Hermansen (1973) noted that lactate is small and easily diffuses into most water compartments of the body. Thus, equilibrium between muscle and blood levels may take up to 5 – 10 minutes. Fujitsuka, Yamamoto, Ohkuwa, Saito and Miyamura (1982) found that in maximal exercise of one minute or
less, accurate measurements of peak lactate concentration were found in venous blood 6 - 9 minutes after the cessation of exercise. However, many researchers feel that a venous blood draw between 3 - 6 minutes post-exercise gives the most accurate concentration of blood lactate (Astrand, 1960; Drinkwater, Horvath & Wells, 1975; Komi & Karlsson, 1978; McCartney et al., 1983; Sahlin et al., 1976; Tesch, 1978; Wilkes et al., 1983).

**Acid-Base Balance During Exercise**

Supramaximal exercise and its end-products, excess carbon dioxide and lactate, have a profound effect upon the acid-base balance of the body. When the glycolytic pathway is utilized to generate energy, equimolar amounts of lactate and $H^+$ ions are produced (Hultman & Sahlin, 1980). A strong inverse relationship exists between lactate production and pH (Gollnick & Hermansen, 1973). Hultman and Sahlin (1980) indicated that lactate accumulation during anaerobic exercise is the main factor determining the lowering of pH. Sahlin et al. (1976) found that the lactate and pyruvate content of muscles following exercise is linearly related to the fall in pH.

In order for the body to function normally and/or optimally, correct $H^+$ ion concentration, thus pH, must be maintained. Normal resting values for pH are approximately 7.0 for muscle and 7.4 - 7.7 for blood. During supramaximal exercise, pH values decrease to 6.4 - 6.6 in muscle and 6.8 - 7.0 in blood (Fox & Mathews, 1981; Osnes & Hermansen, 1972). Since the body cannot tolerate drastic changes in pH, it relies on its internal buffering system to remove excess $H^+$ ions and increase pH to more normal values.

If the excess $H^+$ ions produced as a result of supramaximal exercise were not buffered, the pH of skeletal muscle could conceivably decrease
to a pH as low as 1.5 (Hultman & Sahlin, 1980), a level incompatible with life. However, the body's buffering systems do not allow such imbalances and they work to maintain acid-base equilibrium.

The two main buffering systems are referred to as physico-chemical and metabolic by Hultman and Sahlin (1980). About 61% of the $H^+$ ion uptake in muscle is a result of physico-chemical buffering, while 39% is due to the metabolic processes.

The physico-chemical buffering system depends on the uptake of $H^+$ ions by weak bases. These $H^+$ ions are removed by phosphate compounds, bicarbonate ($HCO_3^-$), and amino acids. Gollnick and Hermansen (1973) report that "when blood lactate concentration increases there is a nearly equivalent decrease in the plasma bicarbonate concentration" (p. 33). These same researchers claim that if the increase in lactate concentration in plasma is representative of the increases in whole blood, "almost all lactate which enters the blood is buffered by the $CO_2/HCO_3^-$ system" (Gollnick & Hermansen, 1973, p. 33). When blood lactate concentration further increases to maximal levels, however, other buffering systems increase in importance. Hultman and Sahlin (1980) contend that amino acids are the major physico-chemical buffers within muscle tissue. This observation is bolstered by the large amino acid content in muscle available to buffer $H^+$ ions.

The metabolic buffering processes involve $H^+$ ion absorption through various metabolic changes. These changes include lactate and pyruvate sensitivity, utilization of creatine phosphate and the formation of ionsine-monophosphate (IMP) (Hultman & Sahlin, 1980).
The largest metabolic buffering process involves lactate and pyruvate (Hultman & Sahlin, 1980). As lactate and pyruvate content increase, due to supramaximal exercise, intramuscular pH decreases. Certain enzymes in the glycolytic pathway, PFK in particular, are sensitive to the lowered pH and shutdown glycolysis when pH levels get too low (Sahlin et al., 1976). The slowing of glycolysis decreases the amount of $H^+$ ions released.

Creatine phosphate stored in the muscle cell is also involved in the uptake of excess $H^+$ ions. According to the investigations by Hultman and Sahlin (1980), "utilization of creatine phosphate causes absorption of $H^+$ ions and synthesis of creatine phosphate liberates $H^+$ ions" (p. 78). The contribution of creatine phosphate buffering differs between individuals. The higher the content of creatine phosphate in muscles, the higher the buffering capacity of the muscles.

Following exhaustive exercise, an increase in IMP has been observed (Hultman & Sahlin, 1980). The IMP is formed through the deamination of adenosine monophosphate (AMP). This metabolic reaction results in the uptake of $H^+$ ions. The contribution IMP makes toward $H^+$ ion removal and pH balance is small, due to the small amounts of IMP formed. In addition to buffering as a means to maintain proper acid-base balance, intramuscular $H^+$ ions and lactate are excreted or effluxed extracellularly into blood plasma.

**Lactate and $H^+$ ion Efflux**

The efflux of excess lactate from working muscle to blood plasma insures normal muscle function and protects against an acid-base imbalance. Several investigators have observed that when lactate levels increase, $HCO_3^-$ levels in the plasma decrease (Gollnick & Hermansen, 1973; Hultman & Sahlin,
1980; Sahlin, Alvestrand, Brandt & Hultman, 1978b). This observation has led to the theory that lactate and H\(^+\) ion leave the muscle together. However, different phases of the exercise cycle may alter the simultaneous efflux of lactate and H\(^+\) ion. Jones (1980) claimed that in some situations lactate efflux is greater than H\(^+\) ion efflux due to intramuscular buffering of the H\(^+\) ion. Others say that lactate and H\(^+\) ion are simultaneously transported across the sarcolemma during exercise and that only for a short time, during early recovery from maximum exercise, a difference in efflux exists, with H\(^+\) ion exceeding lactate efflux (Hultman & Sahlin, 1980; Sahlin et al., 1978b).

The mechanism of lactate efflux is controversial. The controversy involves whether the lactate is actively transported in its dissociated form as lactate ion or if it is diffused in its undissociated form as lactic acid. The difference between the two is energy, with active transport requiring energy while diffusion does not.

Supporters of the active transport theory point out that following supramaximal exercise lactate concentration in the muscle is 2 - 3 times higher than the lactate concentration of perfusing blood. Increased muscle concentration coupled with the fact that lactic acid is a strong acid with a low physiological pH indicates that the permeating form is lactate ion, requiring active transport (Karlsson, 1971, Sahlin et al., 1976).

Supporters of the diffusion theory point out the close relationship between lactate efflux and plasma HCO\(_3^-\) concentration. A study by Sutton et al. (1981) showed that as plasma HCO\(_3^-\) concentration decreased, plasma lactate for a given level of exercise decreased and muscle lactate increased, indicating decreased lactate efflux. According to Jones (1980), Mainwood and Lucier, and Roos, theorized that lactic acid crosses the
sarcolemma in the undissociated form and dissociates to lactate only when it reaches the higher pH environment of circulating blood. This theory asserts that the efflux of lactic acid occurs because of a transmembrane pH gradient, requiring diffusion rather than active transport.

Regardless of whether lactate is actively transported across the cell membrane or diffused down a transmembrane pH gradient, there appears to be a saturation point beyond which efflux is inhibited. In their study of translocation of lactate, Jorfeldt et al. (1978) found that at high intracellular lactate concentrations efflux of lactate leveled off. They found that the maximum rate of lactate efflux was between 4 – 5 mM/min (Jorfeldt et al., 1978). However, altering extracellular pH appears to affect submaximal lactate efflux rates.

Animal studies on muscle metabolism performed by Hirche, Hombach, Longohr, Wacker and Busse (1975) and Mainwood and Worsley-Brown (1975) have shown that extracellular pH influences the efflux of lactate from muscle. In a state of metabolic alkalosis (pH of 7.5 or higher), lactate was transported at a rate 2 – 3 times the rate during metabolic acidosis (pH of 7.0 or lower). The fluctuation in pH was found to be dependent on HCO$_3^-$ concentration. Therefore if the plasma had an increased HCO$_3^-$ concentration, thus elevated pH, the rate of efflux from the muscle to plasma increased (Hultman & Sahlin, 1980).
Training Effects on Lactate

The traditional theory on how training affects lactate production and clearance has been questioned of late. This theory states that a trained individual will have decreased lactate production at any given submaximal workload compared to an untrained individual and that at supramaximal workloads the trained individual will have increased lactate production compared to an untrained individual (Astrand & Rodahl, 1977; Fox & Mathews, 1981; Gollnick & Hermansen, 1973). However, a recently published article by Donovan and Brooks (1983) challenges the traditionally held view. They claim that the effect of endurance training is improved lactate clearance from blood, not reduced production. According to these researchers, "training results in enhanced lactate clearance and in greater lactate carbon conservation due to greater gluconeogenesis from lactate and reduced oxidation of lactate during submaximal exercise" (p. E83). However, whether one supports the traditional theory or the new theory of Donovan and Brooks (1983), the outcome of this study is unaffected.

Active recovery promotes circulation of blood to the muscles, enhancing oxygen supply and waste removal. The speed at which lactate is cleared during recovery from exercise is thought to be improved with active recovery at approximately 50 – 65% of \( \dot{V}O_2 \) max (Astrand & Rodahl, 1977, Gollnick & Hermansen, 1973; Weltman et al., 1979).

**Lactate as a Limiting Factor in Maximal Exercise**

The very nature of anaerobic metabolism and its limited supply of energy have caused much inquiry into the limiting factor of supramaximal
exercise of short duration. The accumulation of lactate and other metabolites causing increased $H^+$ ion concentration and decreased pH have been suggested as a possible limiting factor in that a low pH reduces glycolytic potential and lactate removal from the muscle (Buono & Roby, 1982; Costill, Barnett, Sharp, Fink & Katz, 1983; Gollnick & Hermansen, 1973; Havel & Skranc, 1971; Hermansen & Osnes, 1972; Jones, Sutton, Lin, Ward, Richardson & Toews, 1976; Jones et al., 1977; Karlsson, 1980; Sahlin et al., 1978b; Sahlin et al., 1976; Weltman et al., 1979).

During short-term supramaximal exercise lactate accumulates and pH decreases creating a state of metabolic acidosis in the muscle cell. Metabolic acidosis disrupts enzymes and glycolytic intermediaries responsible for the intricate metabolic reactions in the glycolytic pathway, inhibiting glycolysis and decreasing muscle function. Thus, an alkalotic state would be desirable to provide intracellular buffering and to promote transmembrane efflux of lactate and $H^+$ ions. Several investigators have explored the hypothesis of improved performance with induced alkalosis by increasing extracellular $HCO_3^-$ concentration prior to exercising.

Effects of induced Alkalosis and Acidosis

Positive Effects

As early as 1931, studies of performance after induced alkalosis and acidosis appeared in the literature. Dennig and associates (1931) found that runners who ingested sodium bicarbonate prior to exercise had increased performance capacity and higher maximum blood lactate contents than runners who did not ingest sodium bicarbonate. According to Hultman and Sahlin (1980), Dorow, Galuba, Hellwig and Becker-Freyseng repeated the study by Dennig and co-workers (1931) using swimmers and reached the same conclusion.
However, more recent studies have both confirmed (Costill et al., 1983; Jones et al., 1975; Jones et al., 1977; Rupp et al., 1983; Sutton et al., 1981; Wilkes et al., 1983) and refuted (Balberman & Roby, 1983; Johnson & Black, 1953; Kinderman et al., 1977; Margaria, Aghemo & Sassi, 1971a; McCartney et al., 1983; Poulus et al., 1974) the early work of Dennig and associates.

In 1975, Jones and co-workers studied the effects of induced alkalosis and acidosis on blood lactate concentration and performance in five male subjects on three different occasions. Sodium bicarbonate (NaHCO₃) and ammonium chloride (NH₄Cl) were administered orally to induce alkalosis and acidosis, respectively, while calcium carbonate (CaCO₃) was given as a control. The dosage was not disclosed. The exercise test was performed on a bicycle ergometer and was continuous at 30% VO₂ max for 20 minutes, then 70% VO₂ max for 20 minutes, and then 90% VO₂ max to exhaustion. Endurance times at 90% VO₂ max for alkalosis, acidosis, and control were 7.3, 1.67, and 4.5 minutes, respectively. There was no mention of blood lactate values or significance.

In 1977, Jones and associates repeated their study on the effects of induced alkalosis and acidosis on blood lactate concentration, and exercise performance time. The researchers tested five male subjects on three different occasions. They administered 300 mg/kg body wt. of NaHCO₃, NH₄Cl, or CaCO₃ orally, in capsule form, over a period of three hours. The dosage significantly increased blood pH in alkalosis (7.43 ± 0.025) and decreased the blood pH in acidosis (7.21 ± 0.033), while the control group remained normal (7.38 ± 0.015). The exercise was performed on a bicycle ergometer and was continuous at 33% VO₂ max for 20 minutes, 66% VO₂...
max for 20 minutes, and 95% \( \dot{V}O_2 \) max to exhaustion. Endurance time at 95% \( \dot{V}O_2 \) max was significantly greater in alkalosis (436 ± 120 sec.) than in control (270 ± 13 sec.) or acidosis (160 ± 22 sec.). Blood lactate concentration was highest in alkalosis and lowest in acidosis at each power output, 33%, 66%, and 95% of \( \dot{V}O_2 \) max. Significance was reported at the 0.01 level of confidence.

Sutton et al. (1981) repeated the same study. Again, five male subjects came into the laboratory on three different occasions and were given 300 mg/kg body wt. dosages of NaHCO\(_3\), NH\(_4\)Cl, and CaCO\(_3\) in capsule form over three hours. The exercise test was exactly the same as Jones et al., (1977) and the results were also similar. Endurance time was again significantly greater in alkalosis (5.44 ± 1.05 min.) than in control (4.56 ± 1.31 min.) or acidosis (3.13 ± 0.97 min.). Blood lactate concentration at exhaustion was higher in alkalosis (7.9 ± 0.9 mM/l) than in control (7.10 ± 0.8 mM/l) or acidosis (4.0 ± 0.5 mM/l). Significance was reported at the 0.001 level of confidence.

In these three experiments the researchers attributed the decreased plasma lactate concentration during acidosis as compared to alkalosis to an inhibition of muscle glycolysis combined with a reduction in lactate efflux from the muscle. The inhibition of muscle glycolysis may occur in acidosis because an increased H\(^+\) ion concentration in blood may increase H\(^+\) ion concentration in the muscle cell. As discussed previously, an increase in intracellular H\(^+\) ions decreases pH, which in turn inhibits the metabolic reactions of the glycolytic pathway.

Reduced efflux of lactate across the sarcolemma during acidosis would be expected based on the studies of Mainwood and Worsley-Brown (1975) and
Hirche et al. (1975). Both of these studies demonstrated that lactate efllux is facilitated by a high extracellular pH and is retarded by a low extracellular pH.

In 1983, Rupp and associates studied the effect of alkalosis on blood lactate concentration and exercise performance with similar results. Their four male subjects went to the laboratory on two separate occasions and orally ingested either 300 mg/kg body wt. NaHCO$_3$ or lactose (control) in capsule form over three hours. The exercise test was again performed on a bicycle ergometer and was continuous at 66% $\dot{V}O_2$ max for 20 minutes, followed by 95% $\dot{V}O_2$ max until exhaustion. Endurance times were significantly longer in alkalosis (287 ± 62 sec.) than for lactose (214 ± 47 sec.). Blood lactate concentrations were higher in the alkalotic condition than in the control condition.

Costill and co-workers (1983) conducted a study to assess the effect of induced alkalosis on blood lactate levels and performance on repeated bouts of short-term exercise. Ten males and one female participated in five one-minute supramaximal cycling bouts at 125% of their $\dot{V}O_2$ max. The fifth bout was performed to exhaustion. The cycling took place one hour after the oral ingestion of 200 mg/kg body wt. of NaHCO$_3$ or a placebo, table salt (NaCl). Performance times in the fifth exercise bout were significantly longer in the NaHCO$_3$ trials. Performance times averaged (160.8 ± 19.1 sec.) and (113.5 ± 12.4 sec.) for NaHCO$_3$ and NaCl, respectively. This was significant at the 0.01 level of confidence. The $H^+$ ion/lactic acid ratios were measured between exercise bouts and recovery. The NaHCO$_3$ trials had consistently lower $H^+$ ion/lactic acid ratios. The authors suggested that improved performance time during NaHCO$_3$ was a result of greater buffering of lactate and $H^+$ ion in the blood.
Wilkes and co-workers (1983) also studied the effect of induced alkalosis on six male varsity track athletes. On three separate occasions the athletes ingested 300 mg/kg body wt. of NaHCO$_3$, or CaCO$_3$ (placebo) over a two hour period. There was also a control group that received no drug. Water was consumed ad libitum. Following the ingestion period, the athletes performed their usual pre-race warm-ups for thirty minutes and then raced 800 meters. The results were similar to the previously mentioned studies (Costill et al., 1983; Jones et al., 1975; Jones et al., 1977; Rupp et al., 1983; Sutton et al., 1981). Blood lactate levels measured five minutes post-800 meters were significantly higher ($p < .05$) in alkalosis (14.29 ± 0.05 mM/l) than for placebo (13.32 ± 0.15 mM/l) or control (12.62 ± 0.13 mM/l) trials. Race times were also significantly faster following the NaHCO$_3$ trial (2:02.9 ± 1.9 sec.) than in placebo (2:05.1 ± 2.0 sec.) or in control (2:05.8 ± 2.2 sec.) trials.

The above studies have shown that inducing alkalosis improves performance of anaerobic work following extended warm-up or performance in repeated bouts of supramaximal work. Furthermore, the blood lactate concentrations were always higher in the alkalosis trials. In the studies by Rupp et al. (1983) and Wilkes et al. (1983), HCO$_3^-$ in plasma was also measured. They found the greatest decrease in plasma HCO$_3^-$ levels after exercise with NaHCO$_3$.

The improved performance times observed by the above researchers were attributed to improved extracellular buffering, due to increased plasma HCO$_3^-$ concentration, that augmented transmembrane efflux of lactate and H$^+$ ions. The increased plasma lactate concentration and decreased plasma HCO$_3^-$ concentration in alkalosis serve as evidence of improved efflux. Because
lactate and $H^+$ ions are drawn out of the muscle cell, the time required for intracellular pH to reach critically low values, inhibiting glycolysis, was prolonged. Therefore, anaerobic glycolysis production of energy is extended, and fatigue is delayed allowing performance to improve (Wilkes et al., 1983). However, other studies in the literature fail to show improved performance following sodium bicarbonate ingestion.

No Effect

In 1953, Johnson and Black studied eleven male high school cross country runners to determine the effect of various ergogenic aids. Four hours prior to two races (1.5 miles) the runners consumed 3.5 grams of NaHCO$_3$. The results showed no improvement in race time following NaHCO$_3$ ingestion. There was no mention of performance time or blood lactate levels in the study.

Margaria and associates (1971a) looked at the effects of induced alkalosis on twelve male subjects of different fitness levels (4 trained athletes, 4 fairly active, and 4 not active). The subjects ingested 3.24 grams of an alkali solution containing 0.81 grams of NaHCO$_3$. One half hour after ingestion the subjects performed a treadmill run at a speed and elevation which required an energy production of 80 ml.kg.min$^{-1}$, supra-maximal for all subjects. The researchers reported that performance time on the treadmill was not significantly altered following ingestion of the alkali solution. In further testing, the subjects ingested 12 grams of NaHCO$_3$, maximum performance on the treadmill did increase up to 5.8%, however this increase was not considered statistically significant.

Poulos and co-workers (1974) studied the effect of alkalosis on subjective feelings of fatigue. Six trained male subjects participated in
four exercise tests to exhaustion on a bicycle ergometer. The workload on the ergometer was increased by 10 watts every minute until the point of exhaustion. The participants rated their subjective feelings of fatigue by means of a rating scale. Two of the exercise tests served as controls while in the other two 0.9% NaCl or 8% NaHCO₃ were infused through a small catheter inserted into a superficial vein. The infusion occurred during the test to study the effect of correction of acidosis on subjective ratings of fatigue. The results showed no significant difference in maximum performance time between trials. There was, however, significantly more H⁺ ion efflux after NaHCO₃ infusion. Thus, infusion of NaHCO₃ corrected the acidemia caused by physical activity. However, infusion of NaHCO₃ did not influence subjective ratings of fatigue. Thus, based on their study, Poulus and co-workers (1974) felt that the acid-base balance of arterial blood is not a factor influencing feelings of fatigue.

In 1977, Kindermann et al. studied the effect of induced alkalosis on performance time in a 400 meter run. Ten active males participated in two 400 meter runs. Prior to the run the subjects had 8.4% NaHCO₃ or tris buffer infused into a vein. The infusions were completed in 100 minutes or when resting pH reached 7.5. An average of 190 mM of NaHCO₃ and 130 mM of tris buffer were infused. Following a fifteen minute warm-up the subjects ran the 400 meters. The results showed no significant difference in running time between alkalosis (62.6 ± 4.9 sec.) and control (62.4 ± 4.1 sec.) or in maximum blood lactate concentration between alkalosis (18.11 ± 1.98 mM/l) and control (18.14 ± 1.98 mM/l). This surprised the researchers because extracellular pH and HCO₃⁻ concentrations were significantly higher in alkalosis compared to control, thus improving the
plasma buffering capacity in alkalosis. Kindermann and co-workers (1977) concluded that although alkalosis improved buffering capacity and reduced metabolic acidosis during the run, it did not improve performance time. The authors also concluded that inducing alkalosis was not an effective ergogenic aid.

In a 1983 study, McCartney et al. studied the effect of acid-base alterations on short-term dynamic exercise. Six males participated in four exercise tests performed with altered acid-base balance. For a period of three hours prior to the exercise test, subjects consumed capsules containing a dosage equivalent to 300 mg/kg body wt. of NaHCO₃ (metabolic alkalosis), NH₄Cl (metabolic acidosis) or CaCO₃ (placebo). The fourth trial, respiratory acidosis required inhalation of 5% CO₂ gas. The exercise test required acidosis subjects to exert maximal force on the pedal of a constant-velocity bicycle ergometer set at 100 rpm for 30 seconds. The results showed no significant difference in total work between trials. There was a significant difference in plasma lactate measured immediately post-exercise, with metabolic acidosis (2.8 ± 1.6 mM/l) and respiratory acidosis (1.5 ± 0.8 mM/l) significantly lower than placebo (5.9 ± 3.3 mM/l) and metabolic alkalosis (7.8 ± 4.2 mM/l) conditions. The authors concluded that although increased plasma lactate following alkalosis augmented efflux of lactate and H⁺ ions, blood pH changes are associated with small and insignificant reductions in total work performance in 30 seconds of maximal exercise.

Balberman and Roby (1983) studied the effects of induced alkalosis and acidosis on ten male subjects. A dosage of 300 mg/kg body wt. of NaHCO₃, NH₄Cl, and CaCO₃ was used to produce acid-base changes. The
exercise test required maximum work on a Cybex II dynamometer for one minute. The results showed no significant difference in total work or blood lactate concentration between alkalosis, acidosis, and placebo trials. This led the researchers to conclude that sodium bicarbonate is ineffective as an ergogenic aid in short-term intense exercise.

The most recent study conducted to determine the effects of induced alkalosis on maximal exercise tolerance was done by Katz et al. (1984). These researchers studied the effects of induced alkalosis on eight male subjects. The subjects performed two bicycle rides to exhaustion at a work load corresponding to 125% of $\tilde{V}O_2$ max, one hour after orally ingesting either 200 mg/kg body wt. of NaHCO$_3$ or 1.0 gram of NaCl. Blood pH, HCO$_3^-$, base excess and lactic acid were measured pre and post exercise. The results showed no significant difference in cycling time to exhaustion between NaHCO$_3$ (100.6 ± 6.1 sec.) and NaCl (98.6 ± 5.7 sec.) treatments. In addition, there were no significant differences in blood pH, HCO$_3^-$, base excess or peak lactate acid between treatments until the ninth minute of recovery. After nine minutes of recovery blood pH, HCO$_3^-$, base excess, and lactic acid values were higher in the NaHCO$_3$ trial than in the NaCl trial. These results led the researchers to conclude that NaHCO$_3$ ingestion prior to short-term intense exercise of 1 to 2 minute duration does not improve one's ability to maintain a high work rate or performance.

Summary

Due to conflicting reports in the literature, it is apparent that more research is needed on the effects of sodium bicarbonate on blood lactate levels and performance in short-term supramaximal exercise. Since there is so little data on females, this study used females and attempted to fill in gaps in the literature on their response to induced alkalosis.
In the previous studies mentioned, there are discrepancies in experimental design that may explain some of the conflicting results. One area of discrepancy is dosage. How much bicarbonate is necessary to effectively alkalinate the blood? According to Costill et al. (1983) a dosage of 200 mg/kg body wt. is sufficient to alkalinate blood plasma and avoid gastrointestinal discomfort. The dosages used by Johnson and Black (1953) and Margaria et al. (1971a) were no where near this amount. Perhaps they did not sufficiently alkalinate the blood of their subjects. Another area of discrepancy was the duration of the exercise. In studies by Jones et al. (1975, 1977), Sutton et al. (1981), and Wilkes et al. (1983) where duration of the maximal exercise exceeded two minutes, NaHCO₃ enhanced performance. However, in studies by Katz et al. (1984), Kindermann et al. (1977), McCartney et al. (1983), and Balberman & Roby (1983) where duration of maximal exercise was less than two minutes NaHCO₃ had no significant effect. Are the ergogenic effects of NaHCO₃ negated when exercise duration is less than two minutes? The final area of discrepancy involves the fitness level and sex of the subjects. It is well-established that aerobic training effects the metabolic profile of muscle, improving glycolytic potential (Astrand & Rodahl, 1977; Fox & Mathews, 1981; Karlsson, 1971). It is also well-known that males generally have a larger total muscle mass and greater glycolytic potential than females (Astrand, 1960; Fox & Mathews, 1981; Komi & Karlsson, 1978). Most of the studies in the literature investigated the effects of NaHCO₃ on male subjects of varying fitness levels. No studies using exclusively female subjects were found in the literature. Do females respond differently? Does aerobic training affect response to NaHCO₃?
CHAPTER III

METHODS

Introduction

This study was conducted to determine the effects of sodium bicarbonate (NaHCO₃) on lactate levels during supramaximal exercise. Following ingestion of either NaHCO₃ or sodium chloride (NaCl), the fifteen female subjects participated in four supramaximal exercise trials: NaHCO₃-30 seconds, NaHCO₃-time to exhaustion (TTE), NaCl-30 seconds, and NaCl-TTE. The order of the exercise trials was randomized and a double blind existed so that neither experimenter or subject knew which substance, NaHCO₃ or NaCl, had been ingested. Blood was drawn pre-exercise and post-exercise in each of the four work tests and analyzed for lactate concentration.

Subject Selection

Fifteen female graduate and undergraduate students at the University of Wisconsin-LaCrosse volunteered to participate in the study. The background of the subjects was predominantly physical education. All of the subjects were considered aerobically trained, based on their responses to a self-administered activity questionnaire (see Appendix A). The criteria for "trained" status in this study were the minimal recommendations set forth by the American College of Sports Medicine for quantity and quality of exercise for developing and maintaining fitness (American College of Sports Medicine, 1978).

Preceding participation in any part of the experiment, testing procedures were thoroughly explained to each subject, both verbally and in
writing. Each subject was required to read and sign an informed consent form (see Appendix B) indicating that she understood the experimental requirements and had no known medical limitations for which exercise of the nature required would be contraindicated.

**Procedures**

**Practice Sessions**

Prior to actual testing, the subjects were required to participate in two practice sessions. At the first practice session, the subjects read and signed the informed consent and completed the activity questionnaire. The subjects were told at this time that the purpose of the study was to determine whether NaHCO₃ improved performance in supramaximal work of short duration. They were encouraged to give "true" supramaximal efforts on each of the four tests. They were also told that a double blind would exist and neither experimenter or subject would know which treatment, NaHCO₃ or NaCl, the subject had received.

The subject was then weighed and the exercise test workload was calculated at .075 kg/kg body weight. At this time proper seat height was determined and recorded. The seat height that allowed almost complete extension of the leg in the down position and the most comfort was determined the proper seat height. The subject's feet were strapped onto the pedals with velcro straps. The subject was then instructed to pedal "as fast as you can, for as long as you can" while remaining seated on the bicycle seat. She then sprinted against a practice workload, which was less than the actual test workload of .075 kg/kg body wt., for a short period of time (less than 30 seconds). The first practice session ended with the subject making an appointment for a second practice session, usually the following day.
The second practice session consisted of an identical bout of sprinting on the bicycle ergometer. Again, the workload was less than the actual test workload and the duration of the sprint less than 30 seconds. Appointments for the actual testing were made at the second practice session. The subjects were reminded that they were to be tested in a post-absorptive state (fasting at least 8 hours).

Testing Session

Ingestion procedures. On the day of each test, the subjects arrived one hour prior to the bicycle test. The subjects were asked to ingest a drink containing 18 ounces of cold tap water mixed with either 200 mg/kg body wt. of NaHCO₃ or 5 grams of NaCl. This dosage of NaHCO₃ has been shown to change blood parameters enough to place the subjects in an alkalotic condition and avoid gastro-intestinal discomfort (Costill et al., 1983). The NaCl was chosen as the placebo because of its solubility in water and similarity in taste to NaHCO₃. Five grams of NaCl was determined by the experimenters to sufficiently mimic the taste of varying amounts of NaHCO₃. The measuring, mixing, and administration of the drink was done by a laboratory assistant. The subjects were given five minutes to finish the drink. Upon finishing the drink a period of one hour elapsed before the exercise test began. The subjects were instructed to sit and relax in the test area until it was time for the exercise test. They were allowed to drink water and use the bathroom ad libitum.

Pre-exercise blood draw. Thirty minutes after ingesting the dosage, the first blood sample was drawn from the antecubital vein in the arm. Ingestion of NaHCO₃ and NaCl, while lowering blood pH, does not significantly alter blood lactate levels (Jones et al., 1977; Kindermann et al.,
1977; McCartney et al., 1983; Sutton et al., 1981; Wilkes et al., 1983), therefore resting lactate was determined from this pre-exercise draw. The four ml sample was drawn by either a trained phlebotomist or a registered nurse. After the draw the subjects returned to the test area and relaxed until it was time for the exercise test.

**Exercise test.** The exercise test administered was a modification of the Wingate Anaerobic Test (Ayalon, Inbar & Bar-Or, 1975), which has been used to evaluate capacity for short-term, exhaustive exercise. Reliability and usage of the Wingate Anaerobic Test has been reported in several studies (Ayalon et al., 1975; Inbar, Bar-Or & Dotan, 1976; Jacobs, 1980; Kaiser, Rossner & Karlsson, 1981; Rothstein, Bar-Or & Dlin, 1982; Jacobs et al., 1982; Jacobs et al., 1983).

A mechanically braked Monark bicycle ergometer was used. The workload was calculated for each individual subject on the basis that one pedal revolution resulted in .075 kg/kg body wt. resistance. A resistance of .075 kg/kg body wt. has been shown to reliably elicit anaerobic work (Inbar, Kaiser & Tesch, 1981; Bosco, Luhtanen & Komi, 1983). The work test involved maximal voluntary pedalling for 30 seconds or to exhaustion (TTE). A pedal revolution counter recorded the number of revolutions achieved during the test for calculation of total power output.

Each subject participated in four exercise trials. Two trials required a supramaximal work test of 30 second duration one hour after ingesting NaHCO₃ or NaCl. The other two trials required a supramaximal work test to exhaustion (TTE) one hour after ingesting NaHCO₃ or NaCl. The duration of the trials were randomly assigned. The tests were conducted with at least 72 hours between tests. This was to ensure that the NaHCO₃ and NaCl ingested was removed prior to additional testing.
One hour after ingesting the dosage, the subject was readied for the test. The seat was adjusted to the proper height and the subject's feet were securely strapped onto the pedals. The subject then warmed-up for three minutes at 0.5 kp, at approximately 10 mph. After three minutes of warm-up the subject stopped pedalling. Before the test began, the subject was reminded to pedal as fast and as hard as possible until instructed to stop (30 second test) or until she could not pedal any longer (TTE). The point of exhaustion was subjectively determined by the experimenters when they observed the subject standing-up on the bicycle or using her arms to pull-up on the handle bars thereby involving other muscle groups. To start the test, the subject was instructed to steadily increase her revolutions per minute against a free wheel for five seconds at which point the pre-determined workload (0.075 kg/kg body wt.) was placed on the wheel and the test began. Subjects were encouraged and motivated throughout the test.

Upon termination of the test, the workload was immediately removed and the subject warmed-down for three minutes at 0.5 kp at the speed of her choice.

Post-exercise blood draw. After the three minute warm-down the subject got off the bicycle, walked across the room to the blood laboratory and had her second blood sample drawn as close to five minutes post-exercise as possible. Blood lactate concentration has been shown to peak between three and six minutes post-exercise (Astrand, 1960; Drinkwater et al., 1975; Jones et al., 1977; Komi & Karlsson, 1978; McCartney et al., 1983; Sahlin et al., 1976; Sutton et al., 1981; Tesch, 1978; Wilkes et al., 1983), thus, the second blood sample was drawn five minutes post-exercise.
After the second blood draw the subject was encouraged to walk around and stretch for a few minutes before leaving the laboratory.

**Determination of Blood Lactate**

Lactic acid concentration was determined by analyzing the two blood samples drawn during each of the four exercise tests. Immediately after drawing the blood from the antecubital vein, using a vacutainer, cold 8% perchloric acid was added to the sample to inhibit further glycolysis and lactate production. The blood and perchloric acid were centrifuged for ten minutes. The clear supernatent was then removed and refrigerated until lactate concentration analysis took place.

Lactate concentration was determined using the enzymatic method described by Sigma Chemical Company (1977) (see Appendix C). Standard lactate solutions at 0, 20, 40, and 60 mg% were used to develop the calibration curve against which the samples were compared. Commercial controls of lactic acid were used to verify analysis procedures. Two determinations of each sample were performed and the mean value reported.

Two samples were drawn during each visit for a total of eight samples per subject. As stated previously, the pre-exercise sample, drawn 30 minutes post-ingestion, served as the resting lactate value. The post-exercise sample, drawn as close to five minutes post-exercise as possible, served as the peak blood lactate value.

**Statistical Treatment of Data**

The standard descriptive characteristics of the subjects were analyzed by finding the mean age, height, and weight. A one-way ANOVA was used to analyze the pre-exercise lactate levels. Significance was accepted at the .05 level of confidence.
A dependent t-test was used to compare differences in lactate accumulation in the experimental (NaHCO$_3$) and placebo (NaCl) tests of 30 second duration. Significance was accepted at the .05 level of confidence.

A two by two ANOVA with repeated measures was used to analyze the lactate levels for treatment (NaHCO$_3$ and NaCl) and trial (30 seconds and TTE). Significance was accepted at the .05 level of confidence. When necessary a post hoc test was calculated using the Sheffé test.
CHAPTER IV
RESULTS AND DISCUSSION

Introduction

The purpose of this study was to elucidate what, if any, changes in lactate occur with NaHCO₃ versus NaCl ingestion after supramaximal work of 30 seconds and work to exhaustion. In addition, variables such as descriptive characteristics and total work were analyzed. In this chapter the statistical analysis of these data have been discussed.

Descriptive Characteristics

The fifteen female subjects that volunteered for the study were all considered aerobically trained based on their responses to an activity questionnaire (see Appendix B). Twelve of the fifteen subjects had a background in physical education, with remaining possessing backgrounds in physical therapy and health. The subjects exercised aerobically an average of 4.7 times per week, at greater than 60% of their maximum heart rate for an average of 43.5 minutes per session. According to the American College of Sports Medicine (1980), these values exceed the minimum recommended quantity and quality of aerobic exercise necessary for developing and maintaining cardiorespiratory fitness and body composition.

The standard descriptive characteristics of the subjects are presented in Table 1. The descriptive characteristics of these female subjects are similar to those reported in the literature for non-endurance trained yet active subjects with a background in physical education.


Table 1

Means, standard deviation, and ranges of age, height, total body weight, dosage, and resistance of the female subjects (n=15)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean</th>
<th>S.D.</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yrs.)</td>
<td>24.1</td>
<td>3.76</td>
<td>20 - 32</td>
</tr>
<tr>
<td>Height (cm.)</td>
<td>166.8</td>
<td>5.85</td>
<td>155.6 - 180.9</td>
</tr>
<tr>
<td>Weight (kg.)</td>
<td>61.3</td>
<td>4.99</td>
<td>51.8 - 69.5</td>
</tr>
<tr>
<td>Dosage NaHCO₃ (gms.)</td>
<td>12.3</td>
<td>0.99</td>
<td>10.4 - 13.9</td>
</tr>
<tr>
<td>Resistance (kp.)</td>
<td>4.6</td>
<td>0.37</td>
<td>3.9 - 5.2</td>
</tr>
</tbody>
</table>

In 1981, Nygaard studied 54 females. Twenty of the subjects were called active, possessing a physical education background. This active group had mean values of 25 years for age, 169 cm. for height, and 61 kg. for total body weight. In 1982, Jacobs et al. studied nine female physical education students and reported mean values of 21.1 years, 62.6 kg., and 166 cm. for age, weight, and height, respectively. Similar results were reported by Jacobs et al. (1983) when they studied seven female physical education students and reported mean values of 59 kg. for total body weight and 166.6 cm. for height.

Descriptive characteristics of the untrained female population differ slightly from those reported in this study. Bassett-Frey, Doerr, Laubach, Mann and Glueck (1982) studied sixteen healthy, sedentary females. They found mean values of 22.4 years, 57.3 kg., and 164.4 cm., for age, weight,
and height, respectively. In a study by Dale, Detlef, Gerlach, Martin and Alexander (1979), sedentary females had mean values of 28.1 years, 59.0 kg., and 166.6 cm., for age, weight, and height, respectively. Lesmes, Fox, Stevens and Otto (1978) also studied untrained female volunteers. The descriptive characteristics of the females closest in age to the females used in the present study reveal values of 22.4 years for age, 58.8 kg. for weight, and 162.7 cm. for height. Therefore, the main difference between trained and untrained females seems to be in total body weight. In general, trained females appear to weigh more than untrained females of similar height.

The descriptive characteristics of the subjects in the present study are most similar to those described in other studies using active females with physical education backgrounds (Jacobs et al., 1982; Jacobs et al., 1983; Nygaard, 1981). Based on the guidelines set forth by the American College of Sports Medicine (1980), all of the subjects in the present study could be considered aerobically trained. Trained females have a larger percent lean body mass and smaller percent body fat than sedentary females (Fox & Mathews, 1981). Therefore, while the total body weight of trained females is higher than the total body weight of untrained females, it can be assumed that the contribution of lean body mass to total body weight is greater in the trained female. Thus, the trained female has less body fat and more lean muscle tissue than an untrained female of similar total body weight. The increased muscle mass the trained female possesses improves her glycolytic potential, making her more efficient anaerobically than her sedentary counterpart.
Pre-Exercise Blood Lactate Concentration

The pre-exercise blood lactate concentrations for the subjects are presented in Table 2. Statistical analysis revealed no significant (p > .05) difference in the pre-exercise blood lactate values drawn on each subject prior to each of the four trials.

Table 2

Means, standard deviations, and ranges of blood lactate concentrations pre-exercise (n=15)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean</th>
<th>S.D.</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-exercise NaCl mg%</td>
<td>6</td>
<td>1.5</td>
<td>3 - 10</td>
</tr>
<tr>
<td>Pre-exercise NaHCO₃ mg%</td>
<td>7</td>
<td>4.0</td>
<td>4 - 25</td>
</tr>
</tbody>
</table>

The pre-exercise lactate levels of 6 mg% for NaCl and 7 mg% for NaHCO₃ found in this study are compared to normal resting lactate values in the literature (see Table 3). The pre-exercise blood lactate concentrations found in the present study were well within the normal range for resting lactate, 3 - 12 mg% (Sigma Chemical Company, 1977). They are somewhat lower than pre-exercise levels found in other studies. However, the study by Drinkwater and associates (1975) is the only other study to report resting lactate values for females. The other studies either averaged their male and female values or reported only male resting lactate values. All of the studies reported lactate levels in the normal range.
### Table 3
Mean resting lactate concentrations found in the literature

<table>
<thead>
<tr>
<th>Reference</th>
<th>Subject Sex</th>
<th>Blood Lactate Concentration (mg%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>NH₄Cl 8.4</td>
</tr>
<tr>
<td></td>
<td></td>
<td>NaHCO₃ 12.5</td>
</tr>
<tr>
<td>Drinkwater et al., (1975)</td>
<td>F</td>
<td>11.7*</td>
</tr>
<tr>
<td>Fox &amp; Mathews, (1981)</td>
<td>M/F</td>
<td>10</td>
</tr>
<tr>
<td>Gollnick &amp; Hermansen, (1973)</td>
<td>M/F</td>
<td>10</td>
</tr>
<tr>
<td>Havel &amp; Skranc, (1971)</td>
<td>M</td>
<td>10.8*</td>
</tr>
<tr>
<td>Wilkes et al., (1983)</td>
<td>M</td>
<td>control 10.4*</td>
</tr>
<tr>
<td></td>
<td></td>
<td>placebo 11.2*</td>
</tr>
<tr>
<td></td>
<td></td>
<td>NaHCO₃ 11.8*</td>
</tr>
</tbody>
</table>

*converted from mM · l⁻¹ to mg%

Ingestion of the predetermined dosage of NaHCO₃, while increasing blood pH, does not significantly alter resting blood lactate levels (Balberman & Roby, 1983; Jones et al., 1977; Katz et al., 1984; Kindermann et al., 1977; McCartney et al., 1983; Wilkes et al., 1983). Therefore, the pre-exercise blood lactate levels served as resting blood lactate levels in this study.
30 Second Blood Lactate Concentration

The mean blood lactate concentrations after 30 seconds of supramaximal exercise following ingestion of NaHCO₃ or NaCl are presented in Table 4. Statistical analysis revealed that significantly (p < .05) more lactate accumulated in the blood following NaHCO₃ ingestion compared to NaCl ingestion during the 30 second supramaximal work tests.

Table 4

Means, standard deviations, and ranges of blood lactate concentrations after 30 seconds of supramaximal exercise (n=15)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean</th>
<th>S.D.</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>NaHCO₃ trial (mg%)</td>
<td>73*</td>
<td>18</td>
<td>42 - 108</td>
</tr>
<tr>
<td>NaCl trial (mg%)</td>
<td>62</td>
<td>18</td>
<td>30 - 91</td>
</tr>
</tbody>
</table>

* p < .05

The blood lactate levels found in this study following 30 seconds of supramaximal exercise are comparable to lactate values for similar exercise reported in the literature. As discussed in Chapter II, muscle lactate concentrations immediately post-exercise are comparable to blood lactate concentrations 3 - 6 minutes post-exercise (Astrand, 1960, Drinkwater et al., 1975; Komi & Karlsson, 1978; McCartney et al., 1983; Sahlin, Alvestrand, Brandt & Hultman, 1978a; Tesch, 1978; Wilkes et al., 1983). Thus, the lactate values discussed below lend themselves to comparison.

Jacobs and Tesch (1981) studied female (n=14) performance on the Wingate Anaerobic Test for 30 seconds. They found a mean lactate level
of 53.6* mM · kg\(^{-1}\) dry muscle (* converted form wet muscle weight to dry muscle weight using 4.36 as the conversion factor, Jacobs et al., 1982). In another study by Jacobs and co-workers (1982), changes in muscle metabolites during a 30 second Wingate Anaerobic Test were studied in females (n=9). The intramuscular lactate concentration reported was 60.5 mM · kg\(^{-1}\) dry muscle. Jacobs and associates (1983) studied the response of human skeletal muscle to supramaximal exercise of 10 and 30 second duration. A Wingate Anaerobic Test was used on male (n=15) and female (n=7) subjects to observe changes. The mean lactate concentration reported for the female subjects on the 30 second test was 47.4 mM · kg\(^{-1}\) dry muscle. All these values are comparable to the blood lactate levels reported in the present study for the placebo (NaCl) trial, 62 ± 18 mg%.

McCartney and co-workers (1983) studied the effects of alkalosis and acidosis on a maximal bicycle ergometer test of 30 second duration in six males. The blood lactate concentrations reported were extrapolated from a table in the article (fig. 3., p. 227). After induced alkalosis blood lactate concentration was greater than 117 mg%, after induced acidosis it was greater than 81 mg%, after respiratory acidosis it was greater than 80 mg%, and after a placebo it was greater than 90 mg%. Statistical analysis revealed significantly (p ≤ .05) more lactate in the blood following induced alkalosis than any other trial. The lactate levels reported in the present study reveal the same trend identified in the McCartney et al. (1983) study. There was significantly more lactate in the blood after NaHCO\(_3\) ingestion than after NaCl ingestion.

Augmented efflux of lactate is the most widely accepted hypothesis that explains the higher levels of lactate found in blood following 30
seconds of supramaximal exercise after induced alkalosis. The improved efflux of lactate and $H^+$ ions appears to be related to increased plasma $HCO_3^-$ concentration (Hirche et al., 1975; Mainwood & Worsley-Brown, 1975). In their work on lactate efflux, Hirche et al. (1975) and Mainwood and Worsley-Brown (1975) found that increasing the plasma $HCO_3^-$ concentration increased the rate of efflux, whereas decreasing plasma $HCO_3^-$ concentration decreased the rate of efflux. The mechanism for increased efflux is not clearly understood. Some investigators claim that the process involves active transport of lactic acid and $H^+$ ions across the sarcolemma (Karlsson, 1971; McCartney et al., 1983; Sahlin et al., 1978b), while others assert that the improved efflux is due to passive diffusion down a transmembrane pH gradient (Hultman & Sahlin, 1980; Jones, 1980).

Regardless of the mechanism involved, it has been hypothesized that increased rate of efflux delays the inhibition of glycolysis that occurs when excess lactate and $H^+$ ion accumulate intramuscularly, lowering pH, and disrupting glycolytic enzymes (Costill et al., 1983; Jones et al., 1975; Jones et al., 1977; Rupp et al., 1983; Sutton et al., 1981; Wilkes et al., 1983). Therefore, by delaying the inhibition of glycolysis, more energy would be provided via anaerobic glycolysis, resulting in improved performance. However, neither study, the one by McCartney et al. (1983) or the present investigation, reported improved performance. Statistical analysis revealed no difference ($p > .05$) in total work completed under conditions of alkalosis in either study.

One possible explanation for the lack of improvement under conditions of alkalosis is the duration of the supramaximal exercise. In a work test requiring 30 seconds of supramaximal effort a large proportion of the ATP
utilized is regenerated by the creatine phosphate reaction (Astrand & Rodahl, 1977). The creatine phosphate reaction absorbs protons, thereby decreasing the fall in intramuscular pH (McCartney et al., 1983). In supramaximal exercise of longer than 30 second duration when anaerobic glycolysis provides more ATP than the creatine phosphate reaction, the fall in intramuscular pH may be greater (McCartney et al. 1983). Thus, an increased plasma $\text{HCO}_3^-$ concentration, promoting lactate and $H^+$ ion efflux, may be of greater value in supramaximal exercise lasting longer than 30 seconds, where the energy contribution of anaerobic glycolysis is greater than the creatine phosphate system. Therefore, in supramaximal exercise of 30 second duration, alkalosis improved efflux of lactate and $H^+$ ion, but did not improve performance.

**TTE Blood Lactate Concentration**

The mean blood lactate concentrations for the two time to exhaustion (TTE) supramaximal work tests are presented in Table 5. Statistical analysis revealed no difference ($p > .05$) in blood lactate concentrations between $\text{NaHCO}_3$ and $\text{NaCl}$ in the TTE work tests.

**Table 5**

Means, standard deviations, and ranges of blood lactate concentrations after exhaustive supramaximal exercise (n=6)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean</th>
<th>S.D.</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\text{NaHCO}_3$ trial (mg%)</td>
<td>88</td>
<td>37</td>
<td>56 - 157</td>
</tr>
<tr>
<td>$\text{NaCl}$ trial (mg%)</td>
<td>81</td>
<td>22</td>
<td>48 - 109</td>
</tr>
</tbody>
</table>
Although the blood lactate values were slightly higher following the NaHCO₃ trial, this difference was not significant (p > .05). These results are similar to those reported in the literature for sprint exercise of similar duration.

In 1977, Kindermann and associates studied the effects of induced alkalosis on 400 meter performance time in ten males. Their results showed no significant difference in maximum blood lactate concentrations between alkalosis (163 mg%) and control (163 mg%) conditions.

In 1983, Balberman and Roby studied the effects of induced alkalosis and acidosis on total work output. In the study, ten college males exercised maximally on a Cybex II dynamometer for one minute. The results showed no significant difference in post-exercise blood lactate concentration between alkalosis (97 mg%), acidosis (79 mg%), and placebo (95 mg%) trials.

In 1984, Katz and associates studied the effects of induced alkalosis on cycling time to exhaustion at a supramaximal workload. The results showed no significant difference in time to exhaustion between NaHCO₃ and NaCl or in peak blood lactate, 7 - 9 minutes post-exercise, between NaHCO₃ (130 mg%) and NaCl (120 mg%).

The studies by Kindermann et al. (1977) and Balberman and Roby (1983) used male subjects and subsequently reported higher lactate concentrations than the present study. However, regardless of absolute values, statistical analysis revealed no difference in blood lactate concentration between alkalosis, acidosis, and placebo trials in the studies by Katz et al. (1984), Kindermann et al. (1977), Balberman and Roby (1983), or in the present study.
In addition, statistical analysis revealed no significant (p > .05) difference in total work between alkalosis and placebo trials in the present study. These results support the work of Katz et al. (1984), Kindermann et al. (1977), and Balberman and Roby (1983). None of these studies reported a difference in performance between alkalotic and acidotic conditions.

It appears from the data presented that in supramaximal exercise greater than 30 but less than 70 seconds, induced alkalosis has no effect on lactate accumulation or performance. While the explanation for no difference in lactate accumulation may be methodological, the explanation for no difference in performance may be physiological.

**Lactate Accumulation**

The results of the present study, which showed no significant difference in peak blood lactate concentration in the TTE trials are supported by the work of Katz et al. (1984). Katz and co-workers also reported no significant difference in peak blood lactate concentration following supramaximal, exhaustive exercise. However, Katz and co-workers (1984) did report significantly higher blood lactate concentrations in late recovery (9 - 20 minutes post-exercise) in the NaHCO₃ trial compared to the NaCl trial.

In the present study the reason there was no difference in lactate accumulation may have been methodological. The Wingate Anaerobic Test (Ayalon et al., 1975) is a work test of 30 seconds duration. Reliability and usage of the test has been well documented in males (Inbar et al., 1976; Kaiser et al., 1981; Rothstein et al., 1982) and females (Jacobs et al., 1982; Jacobs et al., 1983; Jacobs & Tesch, 1981). However, its reliability and usage in a time to exhaustion work test has not been documented.
In a 1977 study, Katch, Weltman, Martin and Gray attempted to identify optimal test characteristics for maximal anaerobic work on a bicycle ergometer. Their results showed that for a bicycle test to utilize predominantly anaerobic energy systems the test duration needed to be approximately 40 seconds and the work load 5.0 to 6.0 kp. These test characteristics were identified using male subjects.

In a more recent study, Katch and Weltman (1979) claimed anaerobic capacity was reliably elicited with a test of 120 seconds duration. However, they acknowledge that:

Any anaerobic work test will necessarily involve considerable energy production via aerobic means if the test duration exceeds a few seconds. Nevertheless, a short duration performance test would still be considered anaerobic in nature so long as the work is above the level of anaerobiosis and the major percentage of the total energy production is achieved via anaerobic means (p. 326).

In a 1981 study by Evans and Quinney, different resistance settings were studied to identify the optimal resistance for an anaerobic power test. The subjects again were males. Their results showed that an optimal combination of resistance setting and pedalling speed seemed necessary to elicit "true maximal power" in a maximal anaerobic bicycle ergometer test. The Wingate Anaerobic Test relies only on a resistance setting of .075 kg/kg body wt. to elicit anaerobic work. Evans and Quinney (1981) reported that their maximal power output test significantly (p < .05) exceeded the power output obtained in the Wingate Anaerobic Test.

In the present study usage of the Wingate Anaerobic Test to elicit anaerobic supply of energy to the muscles, in the TTE workload, appeared to be unreliable. Only six of fifteen subjects in the present study performed the TTE work tests correctly: an all out sprint on the bicycle to the point of exhaustion. These six subjects averaged 51 seconds with a
range of 34 - 69 seconds for the two TTE tests. The remaining nine subjects may have misunderstood the purpose of the TTE work test. Instead of sprinting all out to the point of exhaustion, these subjects appeared to slow their pedal cadence and adapt to the work load. These nine subjects averaged 124.5 seconds with a range of 50 - 277 seconds for the two TTE trials. According to the work of Katch et al. (1977) and Katch and Weltman (1979), a work test of greater than 40 seconds may exceed the optimal duration of an anaerobic test. Therefore in this study the TTE trials may have resulted in the utilization of both aerobic and anaerobic energy systems instead of the anaerobic systems exclusively.

The contribution of the aerobic system to total energy may have been the reason that while induced alkalosis enhanced lactate and H⁺ ion efflux during supramaximal exercise of 30 second duration it did not enhance efflux for similar exercise of exhaustive duration. The fact that the aerobic system contributed to total energy output may have been due to methodological error in using the Wingate Anaerobic Test to exhaustion.

Performance

The reason alkalosis did not improve performance in supramaximal exercise lasting between 30 - 70 seconds in this study was probably physiological, not methodological. All the studies that have demonstrated improved performance with induced alkalosis have occurred during exercise lasting over 120 seconds (Costill et al., 1983; Jones et al., 1975; Jones et al., 1977; Rupp et al., 1983; Sutton et al., 1981; Wilkes et al., 1983). In these studies, the maximal work test was performed after at least 20 minutes of lower intensity exercise of around 66% VO₂ max (Jones et al., 1975; Jones et al., 1977; Rupp et al., 1983; Sutton et al., 1981; Wilkes
et al., 1983) or after repeated bouts of supramaximal exercise each followed by rest (Costill et al., 1975). During these "warm-up" periods both aerobic and anaerobic energy systems were utilized to provide ATP. According to McCartney et al. (1983), this may result in a lower than normal intramuscular pH prior to the start of the maximal work test. The subsequent maximal work test resulted in a further decrease in intramuscular pH due to increased lactate and H⁺ ion accumulation. Thus, critically low pH intramuscularly would eventually lead to the inhibition of glycolysis. Therefore in the previous studies, alkalosis and enhanced lactate and H⁺ ion efflux may have improved performance because intramuscular pH was abnormally low before the maximum work test had begun. This implies that alkalosis is useful only when intramuscular pH is abnormally low prior to the maximum work test.

In this study, the supramaximal work tests of 30 second and TTE duration were performed after a three minute warm-up at approximately 10 mph with a resistance of 0.5 kp. It was assumed that the energy for the warm-up was provided aerobically, thus the exercise tests started with normal intramuscular pH values. Two possible reasons why alkalosis had no effect on performance in the 30 second and TTE supramaximal work tests are: (1) intramuscular pH was not lowered prior to the supramaximal exercise test by an extended strenuous warm-up, and (2) the significant contribution of the ATP-PC energy system in maximal work of less than one minute duration. As stated previously, the creatine phosphate reaction absorbs protons, thereby decreasing the fall in intramuscular pH (McCartney et al., 1983). Therefore, when the ATP-PC system is not relied upon to provide energy, the fall in intramuscular pH may be greater.
Thus, in the present study performance may not have been enhanced by alkalosis for a number of reasons. One possibility may be that intramuscular pH may not have reached critically low values because prior to the work tests intramuscular pH levels were probably normal. A second possibility was that the duration of exercise allowed the ATP-PC energy system to make a large contribution towards energy production resulting in uptake of protons and prevention of falls in intramuscular pH. Either one or a combination of these possibilities may have been responsible for the lack of significant alteration in performance with NaHCO₃.

**Summary**

The results of this study on fifteen aerobically trained females showed that in two supramaximal work tests of 30 second duration, blood lactate concentration was significantly \( p < .05 \) higher under conditions of alkalosis (NaHCO₃) than under placebo conditions (NaCl). The greater blood lactate concentration in the alkalosis trial was attributed to enhanced efflux of lactate and \( \text{H}^+ \) ion out of the muscle cell.

In the two supramaximal work tests of exhaustive duration, there was no significant \( p > .05 \) difference in blood lactate concentration between the NaHCO₃ and NaCl trials. The reason that efflux of lactate and \( \text{H}^+ \) ion were not enhanced may have been methodological error.

Total work achieved was not improved under alkalotic conditions in either the 30 second or TTE work tests.
CHAPTER V
SUMMARY, CONCLUSIONS AND RECOMMENDATIONS

Summary

This investigation was carried out to determine what, if any, effect NaHCO₃ ingestion would have on lactate levels after supramaximal work of 30 seconds and exhaustion. Fifteen aerobically trained females volunteered to participate in four supramaximal exercise bouts. Female subjects were studied because of the paucity of data on the response of females to this type of exercise. The lactate levels found in this study were comparable to levels reported in the literature.

Following two practice sessions designed to familiarize the subjects with test equipment and protocol, the subjects participated in the actual testing. One hour prior to each of the four tests, the subjects were required to ingest either 200 mg/kg body wt. of NaHCO₃ (alkalosis) or 5 g. of NaCl (placebo) mixed with 16 ounces of cold tap water. After relaxing for thirty minutes, a pre-exercise (resting) blood sample was drawn for determination of blood lactate content. After thirty more minutes of relaxing, the work tests began. The subjects performed both 30 second and exhaustive tests after ingesting NaHCO₃ and after ingesting NaCl. Five minutes post-exercise the second blood sample was drawn for determination of peak blood lactate content. A double blind testing situation existed so that neither subject or experimenter would know which treatment, NaHCO₃ or NaCl, had been ingested. In addition, the duration of the exercise trials were randomized to help avoid pacing.
The pre-exercise lactate concentrations were analyzed using a one-way ANOVA with repeated measures. The 30 second blood lactate concentrations were analyzed using a dependent t-test. The lactate levels for treatment (NaHCO$_3$ and NaCl) and trial (30 seconds and TTE) were analyzed using a two by two ANOVA with repeated measures. Significance was accepted at the .05 level of confidence.

Conclusions

The following conclusions were deducted from this study taking into account the scope of its limitations and delimitations.

The fifteen female subjects who volunteered to participate in the study were aerobically trained and had backgrounds in physical education. Their physical characteristics were similar to those of trained females with physical education backgrounds reported in the literature. Due to training, the subjects may have possessed a larger muscle mass than non-trained females, augmenting the glycolytic potential of their skeletal muscle.

The mean pre-exercise lactate levels found in this study were 6 mg% for NaCl, and 7 mg% for NaHCO$_3$. Resting lactate levels reported in the literature for males and females were slightly higher. However, in the present study and those in the literature, the resting lactate levels were within the normal range. Since only the Drinkwater et al. (1975) study used female subjects, this study contributes to the knowledge base on female exercise physiology.

The mean blood lactate levels found after the 30 second work tests were 62 mg% for NaCl, and 73 mg% for NaHCO$_3$. There was significantly more lactate in the blood following the NaHCO$_3$ trial. The lactate level found
after the NaCl (placebo) trial was similar to levels reported in the literature for similar exercise with female subjects. The elevated lactate level found after the NaHCO₃ trial was also in agreement with levels reported in the literature following alkalosis. The increased blood lactate concentration found under conditions of alkalosis was attributed to improved efflux of lactate and H⁺ ion.

The mean lactate levels found after the TTE work tests were 81 mg% for NaCl, and 88 mg% for NaHCO₃. There was no significant difference between these values. These values represent the only values for females during a Wingate Test of exhaustive duration. The reason alkalosis did not enhance efflux in the TTE work test is unclear, perhaps it was a methodological error as a result of extending the Wingate Test to exhaustion.

Performance was not improved under alkalosis in either the 30 second or TTE work tests. This may have been due to the fact that intramuscular pH prior to the actual test was not below normal and the duration of the tests favored a significant energy contribution by the ATP-PC system. The ATP-PC system removes excess protons thereby preventing drastic falls in intramuscular pH. Thus in this study, the duration of supramaximal exercise may have been too short to see the ergogenic effects of NaHCO₃.

Based on the results of this study, NaHCO₃ is not an effective ergogenic aid for short-term supramaximal work. Although NaHCO₃ enhanced efflux of lactate out of the muscle cell in the 30 second trial, performance was not improved in either trial. However, generalizing these results to elite athletes may prove erroneous. Elite athletes engage in extended warm-ups, and often have to perform several heats within a few hours. Much of the literature shows that with extended warm-ups or with
repeated bouts of maximal work, NaHCO₃ does benefit supramaximal performance. Therefore, while the present investigation showed no improvement in performance of short-term supramaximal work with NaHCO₃, the applicability of these results to elite athletes is questionable.

**Recommendations for Future Study**

It is recommended that in future studies on induced alkalosis or acidosis that blood and intramuscular pH be measured. The most accepted theory explaining the efficacy of inducing alkalosis to improve performance depends on pH, therefore it should be measured.

It is recommended that sprint trained females take part in future studies that require supramaximal exercise of short duration. The information gleaned from such studies is only applicable to sprint athletes, therefore sprinters should serve as subjects. In addition, sprinters may have altered the glycolytic capacity of their skeletal muscle enough that comparing them to endurance trained athletes would be invalid.

It is recommended that in bicycle ergometer tests intended to elicit anaerobic work of about one minute duration, that a test other than the Wingate Anaerobic Test be used.
REFERENCES CITED


Bassett-Frey, M., Doerr, B.M., Laubach, L.L., Mann, B.L., & Glueck, C.J. Exercise does not change high-density lipoprotein cholesterol in women after ten weeks of training. Metabolism, 1982, 31(11), 1142-1146.


Inbar, O., Bar-Or, O., & Dotan, R. Aerobic and anaerobic components of a thirty second supramaximal cycling task. *Medicine and Science in Sports*, 1976, 8, 51. (Abstract)


Komi, P.V., & Karlsson, J. Physical performance, skeletal muscle enzyme activities and fiber types in monzygous and dizyogous twins of both sexes. *Acta Physiologica Scandinavica*, 1979, 462(suppl.)


Margaria, R., Aghemo, P., & Sassi, G. Lactic acid production in supramaximal exercise. Pfluegers Archives, 1971, 326, 152-161. (b)


Sahlin, K., Harris, R.C., Nylin, B., & Hultman, E. Lactate content and pH in muscle samples obtained after dynamic exercise. Pfluegers Archives, 1976, 367, 143-149.


APPENDIX A

ACTIVITY QUESTIONNAIRE
ACTIVITY QUESTIONNAIRE

Name_________________________
Date_________________________
Height_________________________
Weight_________________________
Age___________________________
Telephone_______________________

1. Are you presently engaged in some form of regular exercise or activity? (3 times/week) _______
2. If yes, have you engaged in it regularly for the last 3 weeks? _______
3. Were you active in the last three months? ______ (prior to the last 3 weeks)
4. How frequently and how intensely do you engage in the following activities?

Intensity (max H.R. = 220 - age)

Strenuous = 60% of max H.R.
Moderate = 50 - 60% of max H.R.
Light = 0 - 50% of max H.R.

<table>
<thead>
<tr>
<th>Activity</th>
<th>Days/Wk</th>
<th>Min/Session</th>
<th>Intensity</th>
</tr>
</thead>
<tbody>
<tr>
<td>aerobic dance</td>
<td>_______</td>
<td>_______</td>
<td>_______</td>
</tr>
<tr>
<td>basketball</td>
<td>_______</td>
<td>_______</td>
<td>_______</td>
</tr>
<tr>
<td>bicycling (outdoor)</td>
<td>_______</td>
<td>_______</td>
<td>_______</td>
</tr>
<tr>
<td>bicycling (stationary)</td>
<td>_______</td>
<td>_______</td>
<td>_______</td>
</tr>
<tr>
<td>bowling</td>
<td>_______</td>
<td>_______</td>
<td>_______</td>
</tr>
<tr>
<td>calisthenics</td>
<td>_______</td>
<td>_______</td>
<td>_______</td>
</tr>
<tr>
<td>cross-country skiing</td>
<td>_______</td>
<td>_______</td>
<td>_______</td>
</tr>
<tr>
<td>down-hill skiing</td>
<td>_______</td>
<td>_______</td>
<td>_______</td>
</tr>
<tr>
<td>golf</td>
<td>_______</td>
<td>_______</td>
<td>_______</td>
</tr>
<tr>
<td>gymnastics</td>
<td>_______</td>
<td>_______</td>
<td>_______</td>
</tr>
<tr>
<td>handball</td>
<td>_______</td>
<td>_______</td>
<td>_______</td>
</tr>
<tr>
<td>hiking</td>
<td>_______</td>
<td>_______</td>
<td>_______</td>
</tr>
<tr>
<td>ice skating</td>
<td>_______</td>
<td>_______</td>
<td>_______</td>
</tr>
<tr>
<td>jogging</td>
<td>_______</td>
<td>_______</td>
<td>_______</td>
</tr>
<tr>
<td>Activity</td>
<td>Days/Wk</td>
<td>Min/Session</td>
<td>Intensity</td>
</tr>
<tr>
<td>-----------------------</td>
<td>---------</td>
<td>-------------</td>
<td>-----------</td>
</tr>
<tr>
<td>racketball</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>rebounding (mini-tramp)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>rollerskating</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>rowing</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>running</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>snowshoeing</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>softball</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>swimming</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>squash</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>tennis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>volleyball</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>walking</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>other</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

5. Have you had a max VO₂ test?  
   when? ________ treadmill ________ bike ________  
   what was your value ________ ml/kg/min⁻¹

6. Are you presently lifting weights? ________  
   how often? ________ (days/wk)  
   how long? ________ (wks, mths)
APPENDIX B

INFORMED CONSENT
INFORMED CONSENT

THE EFFECT OF SODIUM BICARBONATE ON LACTATE LEVELS DURING SHORT-TERM SUPRAMAXIMAL EXERCISE

I, ________________________, being of sound mind and ___ years of age, volunteer to be a subject in a study to determine the effects of sodium bicarbonate on work output and lactate levels during short-term exhaustive exercise. I understand participation in this project will require that I ingest either sodium bicarbonate (baking soda), or sodium chloride (table salt) prior to riding on a bicycle ergometer for a specified period of time.

I will be asked to participate in four trials on a Monark bicycle ergometer. Two trials will involve a work test of 30 seconds following ingestion of either sodium bicarbonate or sodium chloride. Two trials will involve a work test to exhaustion following ingestion of either sodium bicarbonate or sodium chloride. The workload on the bicycle will be determined based on my body weight. I will then pedal at a maximal rate for the required time.

One hour prior to each ride, I will be required to consume either 200 mg/kg body weight of sodium bicarbonate or 5 grams of sodium chloride dissolved in a drink with 18 ounces of water. I will not be informed which drink I have consumed. I will then exercise maximally at my individually calculated workload. Prior to this exercise I agree to abstain from food, drink, and aspirin at least 8 hours and will attempt to keep my diet the same for the 72 hours between tests.

I also understand that preceding and following each bicycling test I will have 4 ml of blood drawn from the antecubital vein in my arm. The area will be sterilized with alcohol preceding the draw and the blood will be drawn by a trained phlebotomist. Since there are 4 bicycle tests, a total of 8 draws will be taken. This blood will be analyzed for lactate content.

As with any exercise, there exists the possibility of adverse changes occurring (staggering, difficulty in breathing, dizziness, etc.) during the tests. In addition, I will feel tired at the end of the exercises. If any abnormal observations are noted the test will be immediately terminated. Also, the dosage of sodium bicarbonate has been determined to be well within safe levels, however there may exist the possibility of some slight gastric discomfort immediately following the exercise.

The tests will be conducted by Doug Crowell and Ellen Brewster. These individuals are graduate students in the Adult Fitness/Cardiac Rehabilitation graduate program at UW-LaCrosse, and will be under the supervision of N.K. Butts, PhD., Professor of Physical Education.
I consider myself to be in good health and to my knowledge am not infected with a contagious disease or have any limiting physical condition or disability, especially with respect to my heart, that would preclude my participation in the tests described.

I have read and I understand the above document and understand the procedure. Any questions which may have occurred to me have been answered to my satisfaction. I have been fully advised of the nature and the possible risks and complications involved in it, all of which risks and complications I hereby assume voluntarily.

I hereby acknowledge that no representations, warranties, guarantees, or assurances of any kind pertaining to this project have been made to me by the University of Wisconsin-LaCrosse, the officers, administration employees or by anyone on behalf of any of them.

I understand that I may withdraw from the program at any time. Signed at ________________ this __________ day of ________________, 19__, in the presence of the witnesses whose signatures appear opposite my signature.

Signed: ___________________________  Witness: __________________________

Witness: __________________________  Witness: __________________________
APPENDIX C

LACTIC ACID DETERMINATION
SIGMA CHEMICAL COMPANY (1977)
LACTIC ACID DETERMINATION

A. Blood Drawing and Preparation of Lactate Samples

1. draw 4.0 ml of blood from the antecubital vein into a vacutainer

2. immediately pipette 1.0 ml of blood out of vacutainer into a test tube containing 2.0 ml of 8% perchloric acid

3. mix on vortex

4. cover with parafilm and refrigerate for 5 minutes

5. centrifuge blood and perchloric acid mixture for 10 minutes

6. pipette clear supernatent off top and store the lactate sample in a freezer until the time of analysis

B. Lactate Analysis Procedure

1. turn on spectrophotometer, set at 340 nm light absorbance and place cuvettes in water bath

2. make control lactate solution
   -place 1.0 ml Lactic Acid Standard #826-10 and 2.0 ml 8% perchloric acid in a test tube
   -mix on vortex
   -refrigerate for 5 minutes
   -centrifuge for 10 minutes
   -pipette clear supernatent off top and store for use in lactate analysis

3. make standard lactate solution at 0, 20, 40, and 60 mg% for curve
   -in 4 test tubes marked 0, 20, 40, and 60, add the following:
     0- 200 ul of 70% perchloric acid solution
     400 ul of deionized water
     20- 200 ul of 70% perchloric acid
     300 ul of deionized water
     100 ul of lactic acid standard #826-10
     40- 200 ul of 70% perchloric acid
     200 ul of deionized water
     200 ul of lactic acid standard #826-10
     60- 200 ul of 70% perchloric acid
     100 ul of deionized water
     300 ul of lactic acid standard #826-10
4. make NAD solution
   - to 5 NAD Preweighed Vials #260-110 add:
     2.0 ml glycine buffer #826-3
     4.0 ml water
     0.1 ml lactic dehydrogenase #826-6
   - mix well, then pour into a beaker
   - pipette 1.4 ml NAD solution into the cuvettes marked: 0, 20, 40,
     60, control, control, and 6 lactate samples in duplicate (two
     cuvettes for each sample)

5. take initial reading of cuvettes with NAD solution on spectrophotometer

6. - to cuvettes marked 0, 20, 40, 60 add 0.1 ml of the standard lactate
    solution marked 0, 20, 40, 60 mg%, respectively
   - to cuvettes marked control, add 0.1 ml of control lactate solution
   - to cuvettes labeled pre-exercise lactate samples, add 0.1 ml of
     the lactate sample
   - to cuvettes labeled post-exercise lactate samples, add 0.1 ml of
     a 1:2 diluted sample (1 part sample, 2 parts water)

7. wait 30 minutes

8. take final readings of each cuvette on spectrophotometer until two
    readings are within .002 nm of each other

9. follow Sigma Chemical Company (1977) hand-book for development of
    the standard curve and the calculations to arrive at lactate con-
    centrations in mg%