THE EFFECT OF PHOSPHATE FEEDING ON LACTIC ACID ACCUMULATION FOLLOWING MAXIMAL WORK

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TABLE OF CONTENTS

LIST OF TABLES

Table | Page

LIST OF FIGURES

NOMENCLATURE

ADP - Adenosine diphosphate

ATP - Adenosine Triphosphate

CO_2 - Carbon dioxide

e^- - electron

FT - Fast-Twitch

H - Hydrogen atom

H^+ - Hydrogen ion

HbC_2 - Hemoglobin

HbO_2 - Oxyhemoglobin

HCO_3 - Bicarbonate ion

HR - Heart Rate

H_2CO_3 - Carbonic acid

H_2O - Water

kcal - kilocalorie

kcal/mm - kilocalories per minute

kg - kilogram

LA - lactic acid

M - Meter

Max VO_2 or VO_2 max - maximal volume of oxygen consumed per minute during exercise.

MET - Metabloic equivalent equal to 3.5 ml of O_2 consumed per kg and per minute.

Min - Minute

Ml - milliliter

mm - millimeter

H_2+ - sodium ion

O_2 - Oxygen

P - Power

PC - Phosphocreatine

Pi - Inorganic phosphate

ST - Slow Twitch

t - time

Vo_2 - Volume of O_2 consumed per minute

CHAPTER I

INTRODUCTION

Through systematic investigations over the past few years, a reasonably comprehensive picture of the operation of the energy sources in muscular exercise has been obtained.

The metabolic processes that supply the energy needs of muscle contraction ordinarily take place in the presence of adequate O_2 to oxidize the carbohydrate sources of energy completely to CO_2 and H_2O. This constitutes aerobic muscle activity which in general is exercise whose intensity is low enough that it can be carried on for at least five minutes or longer. On the other hand, if the intensity of exercise is very high so that exhaustion ensues within one to two minutes, the energy must be supplied largely by anaerobic processes (without O_2) because O_2 cannot permeate the lungs and cardiovascular system rapidly enough to supply such a demand (9).

Energy for short term maximum effort is obtained in various proportions from aerobic and anaerobic metabolic processes. The amount of energy obtained by anaerobic processes cannot be measured directly. It can be approximated by measuring the amount that the metabolism is increased above resting values following exercise (oxygen debt) and by measuring the difference between resting blood lactate and the highest value observed during or after exercise. Oxygen debt and blood lactate concentration provide estimates of different aspects of the energy

obtained from anaerobic sources (1).

Through the research of Hill, Lupton and other early leaders (17), a concept of two separate stages of oxygen debt was established; an alactacid stage and a lactic acid stage. Light to moderate work loads are performed during the alactacid stage of oxygen debt, and a steady state is maintained during this period. The alactacid debt occurs at the initial phase of work with oxygen debts of up to two and one-half liters, while heart rates are not elevated higher than 160 beats per minute. This stage is accompanied by increases of lactic acid in the muscle tissue, but not in the blood stream, and the debt is quickly repaid, generally within three to five minutes. During the lactic acid stage of oxygen debt, there is an accumulation of excess lactic acid in the blood stream, and this is linearly related to the amount of work performed. Heart rates are elevated above 180 beats per minute and the removal of excessive lactic acid during recovery is much slower, taking several hours.

During severe exertion, the respiratory and circulatory adjustments are not adequate, and the oxygen supply to the muscle tissues is insufficient. There will be an accumulation of lactic acid in the muscle, and oxygen debt will be developed.

Karlson (23) stated that when man started to perform dynamic work, chemically bound energy was transformed into mechanical work by the muscles.

In general, four processes all occuring within the muscle cell are concerned with the chemistry of muscle contraction. The first three reactions are reversible. That is, the reactions are such that as some molecules of ATP are being broken down to provide energy for muscle

fiber contraction, other molecules of ADP and P are being regenerated (at a cost of energy provided by the next reaction down, CP → C + P). A balance must obviously be struck between the rate of breakdown and the rate of regeneration or the muscle effort would run out of gas (9).

The glucose molecule first undergoes glycolytic breakdown into two pyruvic acid molecules. The enzyme hexokinase in necessary to add a phosphate (PO_4 or P) radical to make glucose - 6 - phosphate. This process causes the breakdown of one ATP to ADP + P. Each further step of glycolysis is also catalyzed by at least one enzyme specific to that step and one more ATP is broken down to add the second P to form fructose - 1, 6 phosphate (the 1, 6 means that there are two phosphates in the molecule, one at the number 1 carbon atom and one at the number 6 carbon atom) (9).

Fox and Mathews (13) reported that ergogenic aids are thought of only as drugs which may be consumed in order to give the athlete an advantage. They also defined ergogenic aid as something which improved the performance and physical work capacity.

Ergogenic aids affect people differently. Some research studies show a positive influence upon work performance and others, no effect whatsoever.

Consolazio, Nelson, Matoush and Isaak (6) studied the effect of aspartic acid salt (Mg and K) on physical performance of men. They used a double blind study in which the subjects were not informed which group they were in or what medication they were receiving. According to their results, there was no significant difference in metabolic rate and RQ between the treatment A group and treatment B group.

Jokyl (18) studied the effect of dietary supplementation with phosphates upon physical endurance and muscle soreness. He also used a double blind study. A significant difference in favor of the phosphate group was found in each instance concerning the pulse rate.

According to Fox and Mathews (13) during maximal exercise of short duration, large changes occurred in the acid-base balance due primarily to the production of lactic acid. Maximum work (anaerobic) which produces lactic acid will cause the blood and muscle pH to go down. The amount of lactic acid produced depends upon the duration, intensity and muscle mass involved. During rest a blood pH of 7.4 is normal, less then 7.4 indicates acidosis, while a blood pH greater than 7.4 indicates alkalosis.

It was pointed out by Fox and Mathews (13) that during maximum work (anaerobic), blood and muscle pH decrease, while lactic acid concentrations increase. Both lactic acid and the pH have been implicated in the muscular fatigue process. From these facts, Denning, Tablott, Edwards and Dill (8) reasoned that increasing the body's alkali reserve (buffering system) before any heavy muscular exercise (anaerobic) might significantly delay the decrease in pH, and fatigue, and increase exercise performance.

Lundsgaard (26) was able to show that the iodoacetate poisoned excised animal muscle performed lactic acid-free contractions at the same time as energy-rich phosphate compounds were depleted in the muscle.

Lundsgaard (28) demonstrated that the energy supply for muscle contraction could be derived from energy-rich phosphate compounds (16).

The purpose of this study was to examine a commercial product fitting Lundsgaard's description of energy-rich phosphate, and to determine the effect of the use of this product on blood lactate, maximum VO_2, blood pressure, maximum heart rate and fifteen minutes recovery heart rate. The commercial product used in this study was marketed under the name of "Stim O Stam" by the Stam Aids Athletic Product, Inc., of Oklahoma City. One tablet of Stim O Stam contain 200.0 mg dibasic sodium phosphate, 186.7 mg monobasic sodium phosphate, 27.5 mg potassium, 30.0 mg vitamin C (ascorbic acid).

Statement of the Problem

The problem of this study was to determine the effect of phosphate on lactic acid accumulation, max VO_2, heart rate and blood pressure following maximum performance. Most of the past studies have been inconclusive concerning the effect of phosphate on lactic acid accumulation.

Subproblem

To determine if phosphates have an effect on the five, ten, and fifteen minute recoveries in respect to heart rate and blood pressure.

Hypothesis

The null hypothesis was employed in examining the following questions:

1. There will be no significant difference between pre and post lactic acid as a result of phosphate feeding for phosphate group.

2. There will be no significant difference between pre and post maximum VO_2 as a result of phosphate feeding for phosphate group.

3. There will be no significant difference between pre and post maximum heart rate as a result of phosphate feeding for phosphate group.

4. There will be no significant difference between pre and post lactic acid, maximum VO_2 and maximum heart rate as a result of placebo feeding for placebo group.

5. There will be no significant difference in post test lactic acid means between phosphate and placebo group.

6. There will be no significant difference in post test maximum VO_2 means between phosphate and placebo group.

7. There will be significant difference in post test maximal heart rate between phosphate and placebo group.

8. There will be no significant difference in post test fifteen minute recovery heart rate and blood pressure between phosphate and placebo group.

Limitations

1. The population sample was small and composed of volunteers.

2. The author was not able to control the subjects emotional responses, such as nervousness and apprehension which might have affected results.

Delimination

Subjects were volunteers from the men's track team at Oklahoma State University and all were sprinters.

Assumptions

1. The subjects gave maximum effort when they performed the all-out treadmill run.

2. The subjects were equally motivated when they performed the all-out treadmill run.

Significance of the Study

Today, many athletes are given phosphate in hopes of retarding lactic-acid accumulation during all out exercise. It has not been scientifically proven whether this method of increasing performance is useful and appropriate. Lundsgaard (28) was able to show that the iodo-acetate poisoned muscle performed lactic acid-free contractions at the same time as energy-rich phosphate compounds were depleted in the muscle. The need for this study was to provide current data concerning the effect of phosphate on lactic-acid accumulation.

Definition of Terms

Acid: A chemical compound that gives up hydrogen ions (H^+) in solution.

Adenosine Diphosphate (ADP): A complex chemical substance which, when combined with inorganic phosphate (Pi), forms ATP.

Adenosine Triphosphate (ATP): A complex chemical compound formed with the energy released from food and stored in all cells. ATPs are energy producers.

Alactacid Oxygen Debt: That portion of the recovery oxygen used to resynthesize and restore ATP-PC in muscle following exercise.

Alkaline: Pertaining to a base.

Alkali Reserve: The amount of bicarbonate (base) available in the body for buffering.

Alkalosis: Excessive base (bicarbonate-ions) in the extracellular fluids.

Anabolic: Protein building.

Anaerobic: The process of using energy in the absence of oxygen.

Anaerobic Glycolysis: The incomplete chemical breakdown of carbohydrate.

Anaerobic Threshold: That intensity of work load or oxygen consumption in which anaerobic metabolism is accelerated.

Aerobic: The process of using energy in the presence of oxygen.

ATP - PC System: An anaerobic energy system in which ATP is Manufactured when phosphate creatine (PC) is broken down. This system represents the most rapidly available source of ATP for use by muscle. Activities performed at maximum intensity in a period of 10 seconds or less derive energy (ATP) from this system.

Bicarbonate Ion (HCO_3-): A by-product of the dissociation (ionizing) of carbonic acid.

Biopsy: The removal and examination of tissue from the living body.

Blood Pressure: The driving force that moves blood through the circulatory system. Systolic pressure is obtained when blood is ejected into the arteries; diastolic pressure is obtained when the blood drains from the arteries.

Cardiac Output (Q): The amount of blood pumped by the heart in one minute; the product of stroke volume and the heart beat.

Cardiorespiratory Endurance: The ability of the lungs and heart to take in and transport adequate amounts of oxygen to the working muscles.

Double Blind Study: An experimental protocol in which neither the investigators nor the subjects know which group is receiving a placebo and which group the real drug.

Energy: The capacity or ability to perform work.

Enzyme: A protein compound that speeds up chemical reaction.

Ergogenic Aid: Any factor that improves work performance.

Ergometer: An apparatus or device, such as a treadmill or stationary bicycle, used for measuring the physiological effects of exercise.

Fast Twitch Fiber (FT): A muscle fiber characterized by fast contraction time, high anaerobic capacity, and low aerobic capacity, all making the fiber suited for high power output activities.

Fatigue: A state of discomfort and decreased efficiency resulting from prolonged or excessive exertion.

Glucagen: A hormone secreted by the pancreas and that causes increased blood glucose levels.

Glucose: A sugar.

Glycogen: A polymer of glucose; the form in which glucose (sugar) is stored in the body.

Glycogen Loading (Supercompensation): An exercise-diet procedure that elevates muscle glycogen stores to concentrations 2 to 3 times normal.

Glycogenolysis: The breakdown of glycogen to glucose.

Glycolysis: The incomplete chemical breakdown of glycogen. In aerobic glycolysis, the end product is pyruvic acid; in anaerobic glycolysis (lactic acid system), the end product is lactic acid.

Hemoglobin (Hb): A complex molecule found in red blood cells, which contain iron and protein and is capable of combining with oxygen.

High Density Lipoproteins (HDL): A specific kind of cholosteral found in the blood, thought to be protective against coronary heart disease.

Hypoxia: Lack of adequate oxygen due to a reduced oxygen partial pressure.

Ion: An electrically charged particle.

Isokinetic Contraction: Contraction in which the tension developed by the muscle while shortening at constant speed is maximal over the full range of motion.

Isometric (Static) Contraction: Contraction in which tension is developed, but there is no change in the length of the muscle.

Isotonic Contraction: Contraction in which the muscle shortens with varying tension while lifting a constant load. Also referred to as a dynamic or concentric contraction.

Jogging: Slow, continuous running.

Kilogram-Meters (kgm): A unit of work.

Krebs Cycle: A series of chemical reactions occuring in mitochondria, in which carbon dioxide is produced and hydrogen ions and electrons are removed from carbon atoms (Oxidation). Also referred to as the tricarboxylie acid cycle (TCA), or citric acid cycle.

Lactacid Oxygen Debt: The portion of the recovery oxygen used to remove accumulated lactic acid from the blood following exercise.

Lactic Acid (Lactate): A waste product of metabolism resulting from the incomplete breakdown of glucose (sugar).

Lactic Acid System: An anaerobic energy system in which ATP is manufactured when glucose (sugar) is broken down to lactic acid.

Low Density Lipoproteins (LDL): A specific kind of cholesteral found in the blood, thought to cause atherosclerosis.

Maximal Oxygen Consumption (Max VO2): The maximal rate at which oxygen can be consumed per minute.

Metabolic System: A system of biochemical reactions which cause the formation of waste products (metabolites) and the manufacture of ATP; for example, the ATPPC, lactic acid, and oxygen system.

Mole: The grammolecular weight or gram formula weight of a substance.

Muscular Endurance: The ability of a muscle or muscle group to perform repeated contraction against a light load for an extended period of time.

Myoglobin: An oxygen-binding pigment similar to hemoglobin that gives the red muscle fiber its color. It acts as an oxygen store and aids in the diffusion of oxygen.

Net Oxygen Cost: The amount of oxygen, above resting values, required to perform a given amount of work.

Oxygen Debt: The amount of oxygen taken up in excess of the resting value during recovery period.

Oxygen Deficit: The time period during exercise in which the level of oxygen consumption is below that necessary to supply all the ATP required for the exercise; the time period during which an oxygen debt is contracted.

Oxygen System: An aerobic energy system in which ATP is manufactured when food (principally sugar and fat) is broken down.

Oxyhemoglobin (HbO_2): Hemoglobin chemically combined with oxygen.

pH: The power of the hydrogen ion; the negative logarithm of the hydrogen ion concentration.

Phosphogen: A group of compounds; collectively refers to ATP and PC.

Phosphocreatine (PC): A chemical compound stored in muscle, which when broken down aids in manufacturing ATP.

Placebo: An inert substance having the identical physical characteristics of a real drug.

Plasma: The liquid portion of the blood.

Power: Performance of work expressed per unit of time.

Protein: A compound containing amino acids. One of the basic food stuffs.

Pyruvic Acid (Pyruvate): The end product of aerobic glycolysis.

Slow Twitch Fiber (ST): A muscle fiber characterized by slow contraction time, low anaerobic capacity, and high aerobic capacity.

Strength: The force that muscle or muscle group can exert against a resistance in one maximal effort.

Type I Muscle Fiber: Slow twitch fibers.

Type II Muscle Fiber: Fast twitch fibers.

Vitamin: An organic material in the presence of which important chemical (metabolic) reactions occur.

Work: Application of a force through a distance. For example, application of one pound through one foot equals one footpound work.

Description of Instruments

Quinton Motorized Treadmill: An apparatus with continuously moving

belts which can be adjusted to run at varying speeds and can be adjusted to varying inclines (model 24072, Seattle, Washington).

Electrode: Two disc-shaped devices attached to the skin surface on either side of the heart. These electrodes transduce the electrical impulses of the heart into electrical signals.

Biotelemetry Transmitter: A battery powered device which sends electrical signals from the electrodes by telemetry to a biotelemetry receiver (model FM 1100-E2; NARCO Instrument Co., Inc., Houston, Texas).

Telemetry: A method by which a signal can be sent by radio waves from a battery powered transmitter to a receiver.

Biotelemetry Receiver: An instrument which receives electrical signals from the biotelemetry transmitter by telemetry and sends them to the physiograph machine (model FM 1100-7, NARCO Instrument Co., Inc., Houston, Texas).

Narco Physiograph Machine: A multi channel instrument which records and monitors electrical signals from the biotelemetry receiver (type PMR-4Z, six channels; NARCO Instrument Co., Inc., Houston, Texas).

Sphygmomanometer: A device used to indirectly monitor blood pressure (Trimline by PyMatt Corporation, Sommerville, New Jersey).

Stethoscope: An instrument used to manually monitor heart sounds. (Exercise Model, Quinton Instrument, Seattle, Washington.)

Collins Tissot Tank: An instrument used to measure large volumes of gas. (Chain-compensated Gasometer, 120 liter capacity, Collins, Inc., Boston, Braintree, Massachusetts.)

Beckman Oxygen Analyzer: An instrument used to measure the content of oxygen in atmospheric air, or expired air from a subject.

Godart Pulmo Analyzer: An instrument used to measure oxygen and carbon dioxide content of a gas sample.

CHAPTER II

REVIEW OF RELATED LITERATURE

Scheele (40) described the isolation of lactic acid from sour milk. It was soon evident that lactic acid could be isolated from other material. Berzelius (5) demonstrated the presence of lactic acid in skeletal muscle. It was not until about 100 years later that definite evidence of the relationship between muscle activity and production of lactic acid in the muscle could be provided (Fletcher and Hopkins, 12). Meyerhaf and Hill (33) first demonstrated lactic acid production as a final product in glycolysis. Until the 1930's, when Lundsgard (27 and 28) and Lohman (25) demonstrated the significance of creating phosphates and adenosine triphosphate, respectively, as a source of energy in muscle contraction, lactic acid formation was assumed to be necessary in conjunction with the conversion of the chemically bound energy to mechanical energy in muscle contraction.

In the normal resting individual, the supply of O_2 to the tissues is sufficient; therefore, a complete breakdown of glycogen occurs, which results in the formation of CO_2 and H_2O without accumulation of any lactic-acid. Whenever the supply of O_2 is not sufficient to meet demands, an individual is said to contract an oxygen debt.

O_2 debt is used to describe the post-exercise elevation of VO_2 above the pre-exercise level. The possible causes and the magnitude of O_2 debt have been extensively examined following both submaximal and

maximal exercise. O_2 debt and blood lactate concentration provide estimates of different aspects of the energy obtained from anaerobic sources. Under overload conditions (work load greater than aerobic capacity) it had been established that for every liter of O_2 debt, the lactic acid level increased by seven grams. However, the classic work of Margaria, Edwards, and Dill (31) has shown that for the first 2.5 liters of O_2 debt no increase in lactic acid accumulation occurred.

Through the research of Hill, Margaria, and other early leaders (1923), a concept of two separate stages of oxygen debt was established; an alactacid stage and lactic acid stage.

1. Alactacid: Light to moderate work loads are performed during this stage of oxygen debt, and steady state is maintained during this period. The alactacid debt occurs at the initial phase of work with oxygen debts of up to two and one-half liters, while heart rates are not elevated higher than 160 beats per minute. This stage is accompanied by increases of lactic acid in the muscle tissue, but not in the blood stream, and the debt is quickly repaid, generally within three to five minutes.

2. Lactic acid: During the lactic acid stage of oxygen debt, there is an accumulation of excessive lactic acid in the blood stream, and this is linearly related to the amount of work performed. Heart rates are elevated to above 180 beats per minute, and the removal of excess lactic acid during recovery is much slower, taking from 30 minutes to several hours.

In the work by Orskov (34), the conditions necessary for lactic acid formation in the working muscle were studied, considering both local blood supply, exercise and carbohydrate-rich diet. The author

showed that certain amounts of lactate (2-8 mmoles x kg^{-1} weight) were present in the muscles of the resting animal, supposedly well supplied with oxygen. The concentrations rose strikingly during exercise, and values about 20 - 30 mmoles x kg^{-1} net weight were obtained in the group of working muscle of intact animals (19).

The mechanical and thermal energy released during muscle contraction is, in the last analysis, derived from oxidation and from carbohydrate oxidation in particular. Nevertheless, an isolated muscle is capable of performing a considerable amount of work even in the absence of oxygen. Thus the fundamental chemical process involved in muscle contraction is not an oxidative, but rather an anaerobic, exothermic one. The exothermic transformation of glycogen into lactic acid was the first to be recognized as a process of this type (26).

When exercise is raised to a strenuous level at which energy can no longer be provided in sufficient quantity through oxidation (because of the limit on the rate of delivery of oxygenated blood to the tissues), the muscles begin to supplement the energy supply by means of glycolysis. When glycogen is resynthesized from lactic acid, the process seems to be quite slow, taking about fifteen minutes to reach half its maximum rate. Therefore, since the resynthesis is so slow, it can be virtually disregarded, and the concentration of lactic acid in the blood can be taken as a good indicator of the energy contribution of glycolysis beyond that which is supplied by oxidation (14).

Experiments have proven these premises to be true. Measurements of lactic acid concentrations in the blood, after strenuous exercise on a treadmill, indicated that the amount of glycolysis for a given work load increased linearly with time. These measurements confirmed that in

steady exercise the body meets the energy needs exclusively by means of oxidation up to a certain level of energy requirement, and they showed that ordinarily the maximum provided by oxidation is about 220 calories per minute per kilogram of body weight. The production of lactic acid usually began when the energy requirement passed that level after decrease in phosphogan stores. Furthermore, the amount of lactic acid produced in relation to the energy needs indicated that the energy yield from glycolysis is about 230 calories per gram of lactic acid produced (22).

The evidence thus suggests that the disposition of lactate generated by exercise is one limit of physical fitness. This hypothesis has three corollaries, which were investigated by Mann and Garrett (30).

1. The rate of removal of lactate from blood can be augmented by physical training.

2. The capacity to remove lactate from blood can be augmented by feeding either L-(+)-lactate or a carbohydrate diet.

3. The level of physical fitness is correlated with the rate of lactate clearance from the blood, and both fitness and lactate clearance will be augmented by training or by diets that induce the enzymes necessary for lactate clearance through gluconeogenesis. The evidence supported the hypothesis and implied that dietary measures may be used to augment physical fitness (30).

Diamont, Karlson, and Saltin (10) have shown that the lactic concentration in human skeletal muscle was higher than in the blood immediately after exercise of maximal intensity of approximately three minutes duration. However, shortly after the cessation of work, at the time when the peak blood lactate value was reached, the muscle and blood

lactate values were approximately the same (10).

Karlson (23) demonstrated that with an increasing work load, phosphagen stores in the muscle display certain depletion before lactate starts to increase beyond values at rest. The existence of lactate accumulation in the working muscle, despite incomplete phosphagen depletion, suggests that there was energy yield from anaerobic glycolysis at the same time as there was phosphagen depletion. Anaerobic energy yield and the process by which it is produced may be summarized as follows: (1) by phosphagen depletion with low work intensity (less than 50% of max. oxygen uptake), (2) partly by phosphagen depletion and partly by anaerobic glycolysis with moderate work loads (greater than 50% but less than 90% of max. oxygen uptake), and (3) by maximal phosphagen depletion and anaerobic glycolysis, resulting in very high muscle lactate concentrations with heavy and exhaustive work loads (greater than 90% of max. O_2 uptake) (19).

Karlson and Saltin (22), studied lactate, ATP and CP in working muscles during exhaustive exercise in man. The dynamic of lactate accumulation in working muscle was studied in three subjects performing maximal bicycle exercise of 2, 6, and 16 min. duration. Biopsy specimens from the quadriceps femoris were obtained immediately after the work was terminated for determination of ATP, CP glycogen, G-6-P, and lactate. Blood lactate was also determined. The breakdown of the phosphagen (ATP and CP) was already maximal after 2 min. of work in all experiments and averaged 2.7 and 3.6 mmole kg^{-1} wet muscle, respectively. The accumulation of lactate in the muscle and in the blood increased continuously until exhaustion and averaged in the muscle tissue both at the highest and medium loads 16.1 but was only 12.0 mmole

kg^{-1} wet muscle at the lowest load. It was concluded that low ATP and CP stores in these experiments was not the reason for muscular fatigue. Further, if the muscle tissue lactate concentration was the reason for exhaustion on the two heaviest work loads, another factor must be present to explain the exhaustion in the 16 min. experiment (22).

Essen and Haggmark (11) studied the lactate concentration in Type I and II muscle fibers during muscular contraction in man. Five healthy subjects participated in their study. Three subjects performed maximal bicycle exercise. Biopsies were taken from each subject at two different situations in separate experiments. Samples were obtained at rest and after 10, 20, and 40 seconds after the start of the exercise and at exhaustion (3-4 min). One subject performed work of maximal intensity with a pressure cuff placed around one leg (300 mmHg). Muscle samples were obtained from both legs at exhaustion (80s). Two subjects performed isometric contraction at 50% of MVC. Biopsies were taken in separate experiments after 20s and at exhaustion (1 min) subject one and five is the same person. The muscle biopsies were taken from vastus lateralis and frozen in liquid nitrogen within 2 - 4 sec.

No significant difference was seen in lactate concentration between the two extraction procedures. After 20 sec. of isometric contraction, lactate concentration in both fiber types was similar but at exhaustion Type II fibers had 10 mmol/kg wet weight higher lactate concentration than Type I fibers in both subjects. The fact that Type II fibers have a greater capacity for glycolysis may be related to this finding (11).

Robinson and Harmon (38) studied the lactic acid mechanism and certain properties of the blood in relation to training. Their study was designed to determine the effects of training upon the lactic acid

mechanism in work and its relation to certain properties and constituents of the blood. The alkaline reserve has received particular attention because of the above mentioned variance in results. As subjects, nine nonathletic college students, age 18 to 22, were chosen from 40 applicants who went through preliminary testing on the treadmill. To insure cooperation they were paid for their time. The training was continued for a period of 6 months and consisted of a carefully supervised running program with four workouts on the track each week.

Observations in the laboratory were made on the men before training started and at regular intervals during the training period. The work tests in the laboratory were performed on a motor driven treadmill and consisted of: (1) a standard 15 minute walk at 5.6 km per hour on an 8.6 per cent grade with finger blood being drawn for lactate and sugar after 10 minutes of walking when the subjects had attained a steady state (2) a 10 minute run on the level at a moderate pace which was 12.9 km an hour for seven of the men and 14 km for the two best runners; blood for analysis was drawn from an arm vein five minutes after the end of the run, and (3) a run severe enough to exhaust the men in three to five minutes.

The results were as follows:

1. Strenuous athletic training for six months did not affect the basal H_6O_2 capacity, plasma protein, blood lactic acid, blood sugar, alkaline reserve or alveolar CO_2 tension in nine men.

2. The ability of the men to accumulate lactic acid during anaerobic work increased with training.

3. During grade walking blood sugar and lactic acid declined slightly with training.

4. In submaximal running the blood lactate and sugar declined significantly with training (38).

Jorfeldt, Dennfelt and Karlson (21) studied lactate release in relation to tissue lactate in human skeletal muscle during exercise in four healthy volunteers. Muscle lactate concentration and the release of lactate from the leg were determined at rest and at 4 and 12 minutes of bicycle exercise at four intensities. The muscle biopsies were obtained by needle biopsy technique from the vastus lateralis muscle. Both leg blood flow and leg oxygen consumption increased linearly with work intensity. The release of lactate rose approximately linearly with the muscle lactate concentration up to about 4 - 5 mmol/min, but then the relationship revealed a clear leveling off.

Poortmans, Delescaille-Vanden, and Leclercq (37) studied lactate uptake by the inactive forearm during progressive leg exercise. Eleven male subjects were studied during graded leg exercises from 60 to 270 watts. Arterial and venous lactate concentrations were measured from the resting forearm during the exercise and recovery periods. Lactate concentration rose regularly during the work and declined slowly to basal levels after the exercise. The arteriovenous difference rapidly became positive during the exercise, indicating a net uptake of lactate by the nonexercising muscles. The uptake of lactate by the muscle correlated directly with the arterial concentration. After the fifth minute of recovery, there was no longer any significant difference between arterial and venous lactate concentrations. Their conclusions were: (1) nonexercising muscles play a small role in the removal of lactate during exercise, and (2) significant removal of lactate from the blood by nonexercising muscles stops soon after the cessation of exercise.

Harmansen (14) studied the production and removal of lactate during exercise in man. Blood lactate concentration was measured in four female and three male well-trained subjects before and during 30 minutes of continuous treadmill running at four different speeds, demanding about 30, 60, 70, and 80 percent of the individuals maximal oxygen uptake (VO_2 max). The same subjects also performed in another series of experiments where maximal intermittent exercise preceded 30 minutes of running at the same four speeds, or resting in a chair. During continuous running, starting from resting conditions, the blood LA increased only slightly up to a critical level (60-80%) of Max. VO_2. From then on, a pronounced lactate production may occur. The lactate removal rate was higher during exercise than during rest, and increased with increasing work load up to the same critical level (60-80% of VO_2 max), beyond which a reduction was observed. The highest removal rate was 8 mg lactate/100m/x min at 63% of VO_2 max (average values). These results indicate that human skeletal muscle possesses a pronounced capacity to oxidize lactate. Therefore, a production of lactate is possible even with no increase in the blood LA. These results also indicate that skeletal muscle, rather than the liver, may be regarded as the main site for lactate removal during exercise.

Belcastro and Bonen (4) studied lactic acid removal rates during controlled and uncontrolled recovery exercise. Seven male physical education students participated in this study. All exercise sessions were performed on a bicycle ergometer and prior to the investigation each subject completed two load-incremented (180 kpm/2 min) maximal oxygen uptake tests. After a standardized 6 minute bicylce ergometer exercise (89% max. VO_2) lactic acid removal rates were compared during

recovery at rest and exercise at 29.7, 45.3, 61.8, and 80.8% VO_2 max., and twice while the subjects regulated their own recovery exercise. Blood samples were taken after the standardized exercise and every five minutes during the 30 minute recovery periods. During the controlled recovery periods lactic acid removal rates were dependent on the intensity of the recovery. Optimal removal was predicted to occur at 32% VO_2 max. Removal rates during the self-regulated recoveries were not different ($P < 0.05$), but these removal rates were faster than during recovery at rest and exercise at 61.8 and 80.8% VO_2 max. ($P < 0.01$). Removal rates during the self-regulated recovery and recovery at 29.7 and 45.3% VO_2 max. were not different ($P < 0.05$). The subjects were therefore able to remove lactic acid effectively when selecting their own recovery exercise.

Stamford, Moffatt, Weltman, Maldonando, and Curtis (41) studied blood lactate disappearance after supramaximal one-legged exercise. The authors of this study used ten male subjects and performed supramaximal one minute cycling exercise with the right leg followed immediately by six separate 24 minute recovery treatments. Three one-legged recovery treatments were performed at 50 Watt, and represented approximately 40% of one-legged maximal oxygen consumption (VO_2 max). The three recovery treatments involved pedaling with the right leg (RL), pedaling with the right leg while the subjects breathed 100% oxygen, and pedaling with the left leg (LL). A two legged (2L) recovery was also performed at 50 Watt and represented approximately 30% of two legged VO_2 max. A right-legged recovery at 25 Watts (30% of one legged VO_2 max.) and a resting control (C) recovery were also performed. Each recovery treatment was performed separately. Treatments were ranked as follows with respect to degree of

lactate disappearance during the 24 minute recovery period: 2L, RL25, RLO_2, C, LL, and RL. Treatment 2L did not differ significantly from RL25. Treatment 2L and RL25 differed significantly ($P < 0.05$) from control (C), LL, and RL. There was no significant difference from any other treatment. Lactate disappearance was inversely related to the degree of base-line blood lactate accumulation and closely related to percentage of VO_2 max. imposed during recovery. One hundred percent oxygen breathing and exercising fatigued versus nonfatigued muscles were not influential factors during recovery.

Prampero, Peeters, and Margaria (36) studied alactic O_2 debt and lactic acid production after exhausting exercise in man. Their experiments were performed on five healthy subjects (age 22-31 years and weight 65-80 kg). The exercise consisted of running on a treadmill at a speed of 18km/hr, and inclines of 10, 15, and 20% leading to exhaustion in about 50, 25, 15 seconds respectively. The kinetics of O_2 uptake was measured on a breath-by-breath basis during and after short bursts of exhausting exercise. The increase of blood lactic acid concentration as a result of the exercise was also measured. The net alactic O_2 debt measured at the end of the exercise amounted to 32 ml/kg, instead of 16-20 ml/kg as previously observed after maximal aerobic exercise. This corresponds to about 26 mmoles phosphagen (ATP + CP) split per kg of active muscle, a value of the same order as the total phosphagen content of the resting muscle. After short strenuous exercise part of the split phosphagen is resynthesized by delayed lactic acid production, thus shifting a fraction of the O_2 debt from the alactic to the lacticid mechanism. Delayed lactic acid production does not take place if exhaustion is reached in a time long enough, 50 seconds or more, to lead

to full exploitation of the lactacid energy source in the muscles, in this case phosphagen resynthesis is entirely dependent on oxidation.

Phosphate Studies

Pederson (35), investigated the source of phosphate for the glycolytic intermediates in muscular contraction. The two sources of phosphate described are orthophosphate for the initial phosphorolysis of glycogen, and ATP (adenosine triphosphate) for conversion of F-6-P (fructose - 6 - phosphate) to FDP (fructose diphosphate). Experiments employing tracer phosphate were performed on cat gastrocnemiums muscle. ATP, FDP and other phosphate compounds of interest were isolated both in resting and contracted muscles one to two hours after injection of tracer phosphate. The basic requirement for the experiments to give valid results was found to be satisfied. This was a differential incorporation of the isotope into FDP and ATP in the resting muscle. An equilibration of the isotope between carbon-1 of FDP and the terminal P of ATP would, therefore, have been expected with a sufficiently long contraction, if ATP were the source of phosphate. The results obtained, however, showed no equilibration of the isotope between carbon-1 of FDP and the terminal P of ATP, suggesting that ATP does not serve as a source of phosphate for the glycolytic intermediates during muscular contraction. Moreover, the data suggested that intracellular phosphate is the most probable source of phosphate. An unanticipated result indicated that G-6-P formation in the resting muscle occurred by reaction with plasma phosphate rather than with ATP.

Mackler, Olsmsted and Simpson (29), studied the hydrolysis of phosphocreatine and lactic acid production in frog's muscle. Their

hypothesis was "if the hydrolysis of phosphocreatine is a mechanism for the neutralization of lactic acid should not there be a direct relation between the amount of phosphocreatine hydrolyzed and the amount of lactic acid produced." To test their hypothesis they performed the following experiment. Large frogs weighing 300 to 500 grams were quickly killed by cutting off their heads, their spinal cords were destroyed, and the sciatic nerves cut so that the gastrocnemii should be in as nearly as state of rest as possible. One-third of each excised muscle was split off with sharp scissors, quickly thrown into liquid air, and ground to a powder. One-half of this powder was treated with ice cold 8 percent trichloracetic acid and kept at 0 degrees to obtain a protein free filtrate for estimation of phosphocreatine, the other half was used for estimation of lactic acid by the Friedman, Cotonio, Shaffer method.

These results showed: (1) that production of lactic acid from a cut surface of frog muscle causes little or in some cases no decrease in labile phosphorus, and (2) although labile phosphorus disappears during muscular contraction the amount hydrolyzed by no means keeps pace with the lactic acid produced. There appears, therefore, to be no direct quantitive relation between hydrolysis of phosphocreatine and production of lactic acid (28).

Karlson (23) studied lactate and phosphagen concentration in working muscle of man. He used 49 subjects for his study. Fifteen of these subjects were studied immediately after induction into military service, only a few subjects were nationally prominent competitors in endurance events. The physical work of the exercise test was performed on bicycle ergometer. Oxygen uptake was determined with the Open Circuit method. The individual max. VO_2 was predicted according to the leveling off

criterion. Blood lactate concentration was determined using an enzymatic method on 0.1 - 0.2 ml of arterial fingertip blood taken from a prewarmed hand. The LDH activity of biopsy specimens of man was determined from tissue homogenates. It was demonstrated that phosphagen concentration and the accumulation of lactate were the same at identical, relative, submaximal work loads even if great differences in absolute load existed. With test loads greater than 90-100% of individual maximal oxygen uptake, phosphagen store seemed to be maximally reduced as early as 2-3 minutes after the start of exercise, there being no further decrease over the next 10-15 minutes. The highest muscle lactate concentrations were observed at exhaustion. It was demonstrated that the observed increase in blood pyruvate concentration with exercise did not reflect a corresponding increase in muscle pyruvate concentration. Phosphagen depletion as well as muscle lactate concentration were closely related to the calculated O_2 deficit. An estimate of the total breakdown of ATP and CP and the amount of ATP resynthesis by the glycolysis corresponding to the lactate content in muscle and blood agrees in magnitude with the O_2 deficit expressed in ATP equivalents. With standardized work loads performed after a prolonged heavy exercise period, blood lactate concentrations were lower than in a control study after prolonged rest. On the other hand, muscle lactate concentrations were unchanged. The most plausible explanation for these findings is that the preceding prolonged heavy exercise induced on increased utilization of blood-bone lactate. The lowered blood lactate concentrations might enhance lactate flux from the working muscle to the blood (23).

Harris, Sahlin and Hultman (15), studied phosphagen and lactate contents of the quadriceps femoris muscle of man after exercise. Muscle

biopsies were taken from the quadriceps femoris of man immediately after termination of dynamic and isometric exercise. These were analyzed for Adenosine triphosphate (ATP), adenosine 5' - diphosphate (ADP), Adenosine 5" - Phosphate (AMP), phosphorylcreatine (CP), Creatine, Pyruvate, and Lactate. Regardless of type, intensity, and duration of the preceding exercise, a general pattern of the relation between high-energy phosphates and lactate content could be observed. PC showed a non-linear relationship to the muscle lactate content. The ratio of ATP to ADP appeared to decrease linearly when lactate content increased. The relationships are believed to be the consequence of a steady-state condition where muscle pH is one of the major determining factors.

Bacchus, Gamble, Haddy and Scott (2) studied the "Role of Inorganic Phosphate in Active Hyperemia in Skeletal Muscle". For their experiment they used mongrel dogs of either sex weighing 18-20 kg as subjects. The subjects were anesthetized with sodium pentobarbital (30 mg/kg), and artificially ventilated with room air by a positive pressure respirator. Supplemental doses of anesthetic were given as required throughout the experiment. The animals were hydrated by infusions of isotonic sodium chloride solution (50 ml/kg) at the beginning of the surgical procedure. In order to assess the vasoactivity of exogenous inorganic phosphate, an aqueous solution of sodium monobasic phosphate buffered with sodium bicarbonate at pH 6.9 was infused intra-arterially into the hindlimb preparation described above. Infusions were made over a 30 mm. period with step changes in rate so as to increase the venous blood final concentration of Pi (inorganic phosphate) up to 12 times normal. A sodium chloride solution was infused in the same manner to serve as a control. One lymph sample was collected during the sodium chloride

infusion, and another during the phosphate infusion. Venous blood samples were obtained at the beginning and at 10 minute intervals in each infusion period. Arterial inflow and arterial perfusion pressure were continuously monitored. Lymph and venous plasma were analyzed for inorganic phosphate. Also determination of pH, hemotocrit and osmolality were done on all venous blood samples. All data were analyzed with the students t-test modified for paired observations. The results showed that there was no increase in venous plasma Pi with muscle contraction, only a small increase in lymph Pi, and that the phosphate ion is very vasolonactive. They concluded that the inorganic phosphate ion may play only a minor role, if any, in active hyperemia in skeletal muscle.

Fatigue Studies

There is such a thing as physical fatigue. It is true that psychological factors will affect one's response to physical effort and make it difficult to determine if a person is "mechanically tired" or if he is likely to complain of sensations of fatigue. A distasteful physical task leads to early fatigue. Simple muscular fatigue from physical effort is a direct result of using up the glycogen (compacted glucose molecules) in the muscles available for energy. This causes a buildup of lactic acid (usually from inadequate delivery of oxygen by circulation) and a general depletion of energy compounds in the muscle, combined with an accumulation of metabolic products. The muscle may even be damaged as a result of overwork and cause a release of enzymes that can be measured in the blood. Severe overwork of the muscle fibers may lead to swelling and even necrosis of muscles. The muscle may

remain swollen and sore for several days after excess physical fatigue (23).

It is generally believed that at the onset of heavy muscular activities, adenosine triphosphate (ATP) and creatine phosphate (CP) stored in the muscle exclusively will cover the energy demand. Theoretically, however, these immediate energy sources can be depleted in man within 10 seconds. Such short periods of maximal exercise concomitantly result in the formation of lactate. Simultaneously, signs of muscle fatigue or impairment can be recognized as indicated by a decrease in mechanical output, within this time limit (13).

Tesch (42) studied muscle fatigue in man, also with special reference to lactate accumulation during short term intense exercise. A total of 64 subjects was used. Age, height and weight averaged 24 years, 180 cm, and 72 kg. All the subjects were physically fit. Among this group 14 individuals participated in more regular physical training programs and competed on the national elite level in cycling, long-distance running, orienteering and downhill skiing. Tesch used students "t" tests in order to determine the difference between the means. In order to examine the early onset of muscle fatigue, force (torque) development during repeated knee extensions was recorded using isokinetic equipment. Maximal oxygen uptake was determined using leg exercise on a Monark cycle ergometer by stepwise increasing the work load according to the levelling off criterion. Expired air was collected in Douglas bags and the volume measured in a spirometer.

To make comparisons possible with previous muscle fatigue studies, short term maximal cycle exercise was applied. This took place without any warm-up periods. An exhaustive cycling test was performed on a

Monark cycle ergometer with a work load corresponding to 120% of VO_2 max. To study the effect of short term heavy muscle exercise in sport events, subjects performed submaximal and maximal skiing of approximately one minute duration on a slope adjusted for competition. To evaluate changes in electromyographic activity during the muscle fatigue experiments, EMG was registered from the vastus lateralis muscle. Surface electrode of 10 mm in diameter were placed over the belly of the muscle as close as possible to the site of the muscle biopsy insertion. Muscle tissue specimens (approximately 30-50mg) were taken from the vastus lateralis using the percutaneous needle biopsy technique.

Blood samples were obtained from the finger tip and analysed for lactate concentration according to the Barker and Summerson method (3).

The results of the study were as follows:

1. Muscle fiber type distribution in the vastus lateralis muscle in 64 healthy young men averaged 49 (range 10-79%) FT fibers.

2. The absolute as well as relative torque decline was taken as criterion for muscle fatigue and was found to be more pronounced in individuals with muscles rich in FT and/or FTb muscle fibers.

3. After various types of exercise (25 isokinetic contractions, supramaximal cycling exercise, downhill skiing) lactate concentration was related to %FT fibers in the activated muscle. Lactate concentration was also related to LDH_{tot} and M-LDH activity. This seems to indicate a higher rate of lactate production in FT fibers as compared to ST fibers.

4. After 25 maximal repeated contractions, a relationship between force decline and FT/ST lactate ratio was deomnstrated. A similar relationship was also demonstrated for LDH_{tot} and M-LDH ratio, indica-

ting a close relationship between the metabolic profile of the different fiber type, muscle metabolism and muscle function.

5. Lactate accumulation and associated metabolic changes interfering with the contractile mechanism was suggested to be responsible for muscle fatigue in the situations described here (42).

Ergogenic Aids - Studies

Fox and Mathews (3) reported that, frequently ergogenic aids are thought of only as drugs which may be consumed in order to give the athlete an advantage. They defined the ergogenic aids as something which improves or is thought to improve performance; not only athletic performance, but all physical work as well.

Those ergogenic aids affect different people differently. For some, research studies show a positive influence upon work performance and for others, no effect whatsoever.

Fox and Mathews (13) say, drugs, music, warm-up, oxygen, vitamins and other nutritional substances such as carbohydrates and water, and even selected psychological phenomena; for example, hypnosis, mental practice, and suggestions may affect human performance. In this understanding one could call anything an ergogenic aid which can be directly related to enhancing performance.

According to Fox and Mathews (13), during maximal exercise of short duration, large changes occur in the acid-base balance due primarily to the production of lactic acid. Maximum work (anaerobic) which produces lactic acid will cause the blood and muscle pH of less than 7.4 indicating acidosis, while a blood pH greater than 7.4 indicates alkalosis.

It was pointed out by Fox and Mathews (13) that during anaerobic exercise, blood and muscle pH decrease, while lactic acid concentrations increase. Both lactic acid and the pH have been implicated in the muscular fatigue process.

Dennis, Talbott, Edwards, and Dill (8) reasoned that increasing the body's alkali reserve (Buffering system) prior to heavy exercise might significantly retard the decrease in pH, and delaying fatigue and increasing exercise performance. They studied the Alkalosis and the capacity for work of one subject who was a runner. They concluded their study by saying, "Thus, a runner in an alkaline state ran six minutes, and four seconds to exhaustion in comparison with five minutes, 22 seconds starting from a normal state". The oxygen debt was about 20 percent greater in the first case, although the lactic acid concentration in blood from the femoral vein three and one-half minutes after work stopped was 40 percent greater and the change in alkaline reserve of blood was also 40 percent greater. During the first five minutes of recovery after running to complete exhaustion, on the other hand, samples of blood from the femoral vein show little decrease in the level of lactic acid. During this period, little lactic acid leaves the muscle, for samples of blood from an artery have about the same lactic acid content a simultaneous sample from the femoral vein. Notwithstanding these facts the oxygen debt is nearly one-half paid during the first five minutes of recovery. The removal of lactic acid then proceeds at a slow and decreasing rate, requiring from 60 to 90 minutes for completion. An initial alkaline state increases the capacity for neutralizing lactic acid but may have no effect on phosphocreatine breakdown. After this finding, they concluded their study with this statement "Thus if

the quantity of lactic acid formed is increased one-half, the oxygen debt may be increased one-fourth or less" (8).

Consolazio, Nelson and Matoush studied the Effect of Aspartic Acid Salts (Mg and K) on physical performance of men. The subjects were twelve healthy men ranging in age from 20 to 48 years. The authors divided the total group in to two groups of six, treatment A group and treatment B group. Treatment A group took placebo and treatment B group took aspartate, however, the subjects were not informed which group they were in or what medication they were receiving. The study continued for nine weeks, including: a) a two week preliminary control period designed for training, b) five weeks of therapy, either with aspertate or placebo, and c) two final weeks of control or recovery. There were no significant differences in metabolic rate and RQ between the treatment A group and treatment B group. Differences in other factors, such as maximum breathing capacity, vital capacity, breath holding time, were also nonsignificant (6).

Jokyl (18) studied the effect of dietary supplementation with phosphates upon physical endurance and muscle soreness. Fourteen college basketball players were tested over a period of 25 days, each subject took two capsules (equivalent to 2½ tablets) of phosphate daily, while a like number used as control were given an "inert" substance. A significant difference in favor of the phosphate group was found in each instance in respect to pulse rate.

Jones, Sutton, Taylor and Toews (19) studied the effect of pH on cardiorespiratory and metabolic responses to exercise. Five male subjects performed exercise at 33, 66, and 95% of their maximum power output on three occasions in random order. Each study was preceded by a

three hour period in which capsules were taken by mouth, containing either C_2CO_3 (control) NH_4Cl (acidosis), or $NaHCO_3$ (alkalosis) in a dose of 0.3 g/kg body weight. Exercise was continuous and maintained for 20 minutes at the two lower power outputs and for as long as possible at the highest. During maximal exercise to exhaustion, endurance performance was increased from 4.5 minutes (270 seconds) under normal control conditions to 7.3 minutes (438 seconds) after consuming the following ingestion of ammonium chloride (making the blood acidic), endurance time was reduced to only 2.6 minutes (160 seconds). No differences were observed for central cardiovascular changes in exercise. The respiratory changes expected from changes in blood pH were observed, with a higher alveolar ventilation in acidosis. At all power outputs arterialized venous lactate was lowest in acidosis and highest in alkalosis. Plasma glycerol and free fatty acid were lowest in acidosis. Changes in blood (HCO_3-) bicarbonate and pH were shown to have major effects on metabolism in exercise which presumably were responsible for impared endurance.

Stim - O - Stam Studies

Wood (44) at Abeline Christan (1978) studied the effect of Stim - O - Stam (phosphate) on blood lactate, urinary phosphate, blood phosphate, systolic blood pressure, diastolic blood pressure, respiratory rate, and heart rate. They used nine male volunteers for their study, the age of the subjects was not reported. They used laboratory procedures to determine blood lactate concentration, urine phosphate concentrations, and blood phosphate concentrations. Besides the three above parameters, systolic and diastolic blood pressures, respiratory rate,

and heart rate were examined. The nine subjects were required to run on a treadmill, set at a speed equivalent to an eight minute mile run. Upon reporting to the laboratory, each subject had blood samples drawn and the various tests were then performed on that sample. One hour later, each person ran on the treadmill for eight minutes (one mile). The second day testing was conducted same as the first day except for a few changes. The major change was immediately following the drawing of the first blood sample, four Stim-O-Stam tablets were taken. After one hour rest the subjects were put on the treadmill for eight minutes (one mile). After the treadmill run, blood was again drawn to provide the data for phosphate-exercise conditions. The third and final day of testing involved finding values for normal-rest, phosphate-rest, and phosphate-exercise. After the first blood sample, each subject again took four Stim-O-Stam tablets and was allowed to rest for exactly one hour. After this period another blood sample was taken to obtain a phosphate-resting value. Immediately after this sample was taken, the individual was put on the treadmill for another eight minute mile. After running each individual had a third blood sample drawn to ascertain the phosphate-exercise value. The blood samples were drawn from the venous blood. The statistics applied to the raw data showed direct effect on blood lactate reduction ($p<.01$), elevation of diastolic blood pressure ($p<.05$), respiratory rate reduction ($p<.01$), and a decrease in heart rate ($p<.001$) with the use of Stim-O-Stam.

Ulrich (43) studied the effect of Stim-O-Stam on oxygen intake and maximum work-output of 20 athletes. During the first test, athletes were tested until fatigued on an arm erpometor for a five minute period, while oxygen intake and maximum work output were recorded. The average

of the maximum work for the first 20 tests was about 4300 meter-kilograms (x6.8 to convert to foot-lbs. work), while the average oxygen intake was about 20.6 ml/kg/min. After the first test phosphates were given for the next 21 days. At the end of the first eight days the second tests were given, the maximum work average jumped to 4600 meter kilograms. After the next seven days, another test was given, the maximum work average jumped to 5200 meter kilograms. At the end of the 21 days another test was given, maximum work average jumped to 5400 meter kilograms and the oxygen intake average was 20.5 ml/kg/mm. For the eight days (no phosphate given) the author tested all the subjects. For the first three days the maximum work remained at the high 5400 meter kilograms. At the end of the eighth day, the work output was 4300 meter kilograms, and the oxygen intake remained at a constant average as before. The author concluded his study with this statement, "With constant oxygen intake, the maximum work increased from 4300 meter kilograms to 5400 meter kilograms, a 26% increase in work efficiency due to 21 days phosphate (Stim-O-Stam) feeding" (43, p. 12).

Davenport (7) studied the effect of the commercial product Stim-O-Stam on ten college men. He used ten male volunteers from Oklahoma State University students with ages ranging from twenty to twenty-five years old. These ten volunteers were taken to the varsity track and clocked in a twelve minutes endurance run (pre-tested). After the pre-test the author divided the group (10) into two groups of five, experimental group and control group. The experimental group took Stim-O-Stam and the control group took a placebo for fourteen days. After this period, the groups were post-tested by the same procedures used in the pre-test. There was no significant difference between pre

and post-test twelve minute run for control group. There was a significant difference between pre and post-test twelve minute run for the experimental group ($<.05$) (7).

McFadden (32) studied the effect of phosphate feeding on the mile run time of college students. The author used 36 subjects from Oklahoma State University, who volunteered from a weight training class. The subjects were males between the ages of 19-23 years old. During the pretest all 36 subjects ran one mile for time to assess their fitness level. After the first time trial the subjects were divided into three matched groups, according to their times. Two weeks before the post-test two of the groups were given tablets that they were told was a food supplement designed to increase endurance. One group received the placebo, and another group received Stim-O-Stam. The third group was the control group and did not receive any tablets. The experiment was a double-blind study. After 14 days all the subjects repeated the one mile run under the same conditions as the pre-test. There were no significant differences in the mean times recorded by the three groups.

Rosson (39) studied the effect of phosphate feeding on blood lactate concentration after wrist flexion to exhaustion. Subjects were eight male and six female Oklahoma State University physiology of exercise class members who volunteered for his study. Subjects participated in a wrist flexion test to exhaustion and gave a sample of blood three minutes later. The subjects were then paired based on their pre-test lactic acid results. The author labeled one group as experimental and the other as the control group. The experimental group took the phosphate and the control group took the placebo for seventeen days. The author used a double blind study in administering the pills. After the

seventeen day period, a post-test was given to check for lactic acid concentration between the two groups. There was no significant difference between experimental group lactic acid content and control group lactic acid content.

Summary

The literature reveals that lactic acid content increases linearly with exercise and decreases with training.

The lactate removal rate after maximum exercise was higher during light (low intensity) exercise than during rest.

According to the studies, it was generally believed that at the onset of heavy muscular activities, ATP, CP stored in the muscle exclusively will cover the energy demand.

An estimate of the total breakdown of ATP and CP and the amount of ATP resynthesis by the glycolysis corresponded to the lactate content in muscle. Therefore, the ratio of ATP to ADP appeared to decrease linearly when lactate content increased.

Most of the studies have shown that with the presence of phosphates the endurance of muscular activity was greater. Some studies did not show any significant difference in muscular endurance due to an excess of phosphate present in the body.

From the literature it was also concluded that Stim-O-Stam (phosphate) tended to decrease the lactic acid, maximum heart rate, and increased max VO_2, and maximum work compacity (7, 27, 28, 39, 43, and 44).

Finally, the benefit of the ergogenic aids may be associated in part with psychological changes within the users.

CHAPTER III

METHOD AND PROCEDURE

The purpose of this study was to measure the effect of phosphate on lactic acid accumulation, maximum VO_2, 15 minute recovery heart rate and blood pressure. This chapter outlines the methodology and procedures used for assessing the effect of phosphate on lactic acid accumulation during the all out treadmill run.

Selection of Subjects

Varsity track men at Oklahoma State University were used as subjects for this study. Eighteen track men between the ages of eighteen and twenty-two participated on a volunteer basis in the pre and post-test. The subjects were randomly assigned to two matched groups on the basis of their lactic-acid results on the pre-test, to form two matched groups, then randomly assigned to the phospate group and the placebo group.

Personal Data

Upon arrival at the Physiology of Exercise Laboratory, each athlete was asked to fill out a form, inlcuding the following information: name, age, and phone number.

Testing Procedures

All the subjects reported to the Physiology of Exercise Laboratory

in September, 1980, wearing activity clothes, running shoes, shorts, and tee-shirt. They were instructed in how to use the treadmill and how to use the breathing valve during the run.

Telemetry equipment was attached to the subject's chest, (one electrode over the sternum and the other electrode just below and to the left of the left nipple). Heart rate was monitored every minute during the run and every five minutes during the fifteen minute recovery.

Each subject ran on the treadmill using the following steps:

1. All subjects started at 5 mph at 5% elevation for five minutes (warm-up period).
2. Treadmill speed was increased to seven mph at 7% elevation for three minutes.
3. Elevation was increased to 10% where the subject ran until he became exhausted.
4. The subjects gave a signal when they felt that they could go only one more minute. After the signal, the subject's expired air was collected in the tissot tank for thirty seconds. At this point the subject's heart rate was 180 beats per minute or higher.
5. Subjects were helped off the treadmill and seated on a chair. At the end of three minutes of recovery, blood samples were drawn from the subject's right index finger by a laboratory technician. (The three minute recovery was recommended by Dr. William Haskell, Ph.D. and Bruno Balke, M.D.) The drawn blood was collected inside capillary tubes. These blood samples were taken to the microbiology laboratory for determination of the lactic acid content. A microbiology laboratory

assistant and Ph.D. candidate measured the lactic acid content using manometric technique patterned after Barker and Summerson (3).

6. The monitored recovery period was for fifteen minutes. At the end of the fifth, tenth and fifteenth minute the heart rate and blood pressure were measured.

After the pre-test, subjects were arranged in order (high to low) according to the amount of lactic acid found in their blood. Dr. A. B. Harrison and Dr. Betty Edgley randomly assigned the subjects based on these lactic acid measurement to two groups of nine each (phosphate and placebo groups). They used the following procedures:

1. Subjects were arranged in order (high to low) according to the lactic acid measurements, #1 and #2 were paired, a coin was tossed to determine into which group each fell. The same procedures were followed for each pair.
2. After determining two equal groups, a coin was tossed to determine which group would be the phosphate group and which group would be the placebo group.

A local drugstore supplied phosphate capsules and placebo capsules which hid the taste and appearance of the pills. A 16 day supply of capsules (three per day) was packaged in numbered pill bottles.

After the phosphate and placebo groups had been determined, the phosphate group took the phosphate pill (Stim-O-Stam) for 16 days. Each athlete took 3 to 4 pills 30 minutes prior to his daily workout (mostly interval type running such as 2 x 400 and 2 x 300 etc.). The placebo group took color-coded pills for 16 days. This method of administering capsules is known as the double blind method because neither the author

nor the subjects knew who was getting the color coded placebo and who was getting the phosphate. All the subjects did the same kind of exercise during the 16 day period.

At the end of 16 days the subjects were given a post test following procedures the same as the pre-test. For the post test each subject stayed on the treadmill as long as the pre-test time. The purpose of this was to give each subject the same work-load in both the pre-test and the post-test.

The pre and post-test means for both groups were compared by a paired t-test to determine the effect of phosphate on lactic acid accumulation, maximum VO_2, maximum heart rate, fifteen minute recovery heart rate and blood pressure. Also the post test means were compared by means of a paried t-test to determine if there was a difference between phosphate and placebo groups (matched pairs).

The .05 confidence level was used to test for the significance of difference.

CHAPTER IV

RESULTS AND DISCUSSION

The author has attempted to determine if phosphate had an effect on the lactic acid accumulation, maximum VO_2, maximum heart rate, fifteen minutes recovery heart rate and fifteen minutes recovery blood pressure.

Eighteen male track athletes participated in the pre and posttest all-out treadmill run. A total of seven variables were measured and recorded from both the pre and posttest. After the pre-test, the group was divided into two groups, phosphate group and placebo group. Paired t-tests were calculated on all variables. The .05 was chosen as level of significance. Calculations were done on the phosphate group pre and posttest results for all the variables, placebo group pre and posttest results for all the variables, phosphate group and placebo group post-test means for all the variables. A summary of the pre and posttest raw data for phosphate and placebo groups appears in Appendix A, Tables VI through IX.

Means, standard deviation and standard error of the mean for the entire group's pre and posttest, and the differences of the pre and posttest means are displayed in Appendix B, Tables X, XI and XII. Pre and posttest means, standard deviation and standard error group are presented in Appendix B, Tables XIII and XIV, and the differences of the pre and post-test means are presented in Appendix B, Table XV. Pre and posttest means, standard deviation and standard error of the mean for the phosphate group are presented in Appendix B, Tables XVI and XVII,

and the differences between pre and posttest means are presented in Appendix B, Table XVIII.

During the pre-test all the subjects averaged 14.44 minutes on the treadmill and all had approximately the same fitness level.

During the posttest the phosphate group showed better progress on their maximum Vo_2, maximum heart rate, maximum lactic acid, fifteen minutes recovery heart rate and fifteen minutes recovery blood pressure.

Significant Differences

The t-test for the phosphate group's pre and posttest differences showed significant change on lactic acid, maximum heart rate, second maximum Vo_2. The t-test for the placebo group's pre and posttest differences revealed no significant changes on any of the variables. The t-test for differences between posttest means showed significant differences on all of the variables except on the lactic acid accumulation, maximum VO_2 and fifteen minute recovery blood pressure.

Phosphate Group

The phosphate group showed a general improvement in most of the variables. The pre-test mean for lactic acid was 10.495 mg lactic acid/10 ml plasma, and the posttest mean was 7.00 mg lactic acid/10ml plasma, and the differences was 3.495 mg lactic acid/10ml plasma. Standard deviation for pre-test was 0.873 and for the posttest it was 2.023. t value was 4.56 which made it significant at the .01 level of confidence (Appendix B, Table XIX). This means that the phosphate group showed better improvement in the lactic acid content by using Stim-O-Stam prior to all-out performance. This also showed that Stim-O-Stam was good ergogenic aid for the athletes during the all-out treadmill run.

Pre-test mean for maximum Vo_2 was 47.37 ml/kg/min and the posttest mean was 57.20 ml/kg/minutes. The mean difference was 10.17 ml/kg/minutes. Standard deviation for the pre-test was 10.218 and for the posttest it was 4.563. The t value was 3.65 which made it significant at the .01 level of confidence (Appendix B, Table XX). It is generally accepted that the source of energy in the human body comes from ATP (Adenosine Triphosphate) and CP (Creatine phosphate). Since the body cannot store the excess phosphate in the body for extra energy, if the body gets extra phosphate (Stim-O-Stam) prior to the all-out exercise, the body will produce more energy (VO_2), as shown by the phosphate group. The change that occurred in maximum VO_2 was not a major issue of this study because at the beginning of this study the groups were equally matched according to their lactic acid, not maximum VO_2.

Pre-test maximum heart rate was 187.33 beats per minute and the posttest rate was 180 beats per minute. The mean difference was 7.33 beats per minute. The standard deviation for the pre-test was 5.000 and for the posttest 4.243. The t value was 5.50 which made it significant at the .01 level of confidence (Appendix B, Table XXI and Figure 1). This means that with the same duration and intensity a lower heart indicates a greater cardiac output (Table I).

Placebo Group

The placebo group did not show significant improvement in any of the variables.

Pre-test lactic acid mean was 10.67 mgLA/10ml plasma, and the posttest mean was 9.04 mgLA/10ml plasma. This produced a mean difference of 1.63 mg lactic acid/10ml/plasma. The standard deviation for pre-test

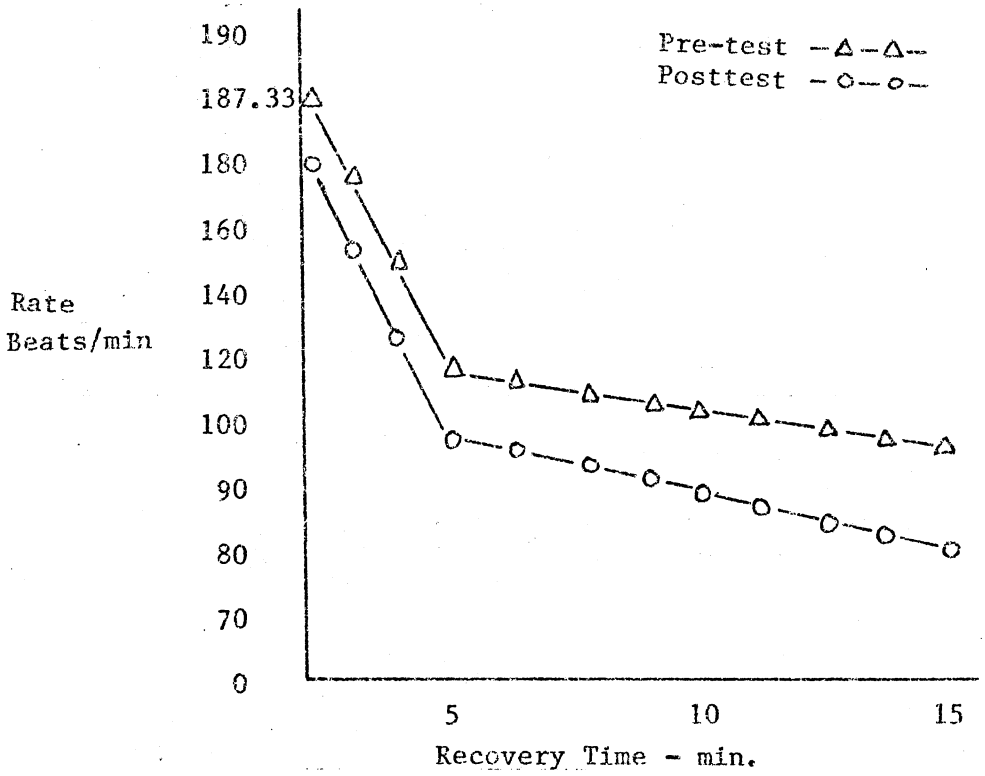


Figure 1. Pre and Post Recovery Heart Rate for Phosphate Group

was 0.937 and for the posttest was 1.528. The t value of 2.23 was not significant at the .05 level of confidence (Appendix B, Table XXII).

TABLE I

t-VALUES FOR PHOSPHATE GROUP BETWEEN PRE AND POSTTEST MEANS

Variables	t-value	Significance
Lactic acid	4.56*	.01
Maximum VO_2	3.65*	.01
Maximum Heart Rate	5.50*	.01

Pre-test maximum Vo_2 mean was 57.54 ml/kg/mm., and the posttest mean was 63.22 ml/kg/mm. This produced a mean difference of 5.68 ml/kg/minute. The standard deviation for the pre-test was 9.936, and for the post-test was 6.919. The t value of 1.49 was not significant at the .05 level of confidence (Table II and Appendix B, Table XXIII).

Pre-test maximum heart rate mean was 186.66 beat per minute and the post-test mean was 186 beat per minute. The standard deviation for the pre-test was 4.690 and for the post test was 5.196. The t value of .43 was not significant at the .05 level of confidence (Appendix B, Table XXIV and Figure 3).

TABLE II

t-VALUES FOR PLACEBO GROUP BETWEEN PRE AND POSTTEST MEANS

Variables	t-value	Significance
Lactic acid	2.23	NS
Maximum VO_2	1.49*	NS
Maximum Heart Rate	.43	NS

Posttest Differences Between Phosphate Group and Placebo Group Means

There was a significant difference between the phosphate and placebo groups posttest means, except in lactic acid, maximum Vo_2 and fifteen minute recovery blood pressure.

Posttest lactic acid mean for the phosphate group was 7.03 mg /10ml plasma, and the posttest lactic acid mean for placebo group was 9.05 mg lactic acid/10ml plasma. This revealed a mean difference of 2.02 mg lactic acid/10ml plasma. The standard deviation for the phosphate group was 2.023 and for the placebo group was 1.528. The t value of 2.28 was not significant at .05 level of confidence (Table III and Appendix B, Table XXV).

During the posttest the phosphate group showed lower lactic acid than the placebo group. Both groups had similar lactic acid content during the pre-test. The phosphate group showed some improvement by producing less lactic acid. This difference was not significant at the .05 level, but this improvment would be significant at the .10 level.

TABLE III

t-VALUES BETWEEN PHSOPHATE AND PLACEBO GROUPS: POSTTEST MEANS

Variables	t-value	Significance
Lactic acid	2.28	NS
Maximum VO_2	1.26	NS
Maximum Heart Rate	3.46*	.01

Posttest Vo_2 gain for the phosphate group was -5.6956 ml/kg/min, and the posttest Vo_2 gain for the placebo group was -9.8267 ml/kg/min. This revealed a difference of 4.1311 ml/kg/min. The standard deviation for the phosphate group was 11.378 and for the placebo group was 8.075. The t value of 1.26 was not significant at the .05 level of confidence (Appendix B, Table XXVI). The reason that the author compared the percent of gain between the two groups was the large difference of VO_2 at the beginning of the study.

Posttest maximum heart rate for phosphate group was 180 beats per minute and for the placebo group was 186 beats per minute. This revealed a difference of six beats per minute. The standard deviation for the phosphate group was 4.243 and for the placebo group was 5.196. The t value of 3.46 was significant at the .01 level of confidence (Table III and Appendix B, Table XXVII). These results show that the phosphate group produced lower heart rates than the placebo group even though the workload was the same for both groups. However, the only difference was that the phosphate group took Stim-O-Stam prior to exercise.

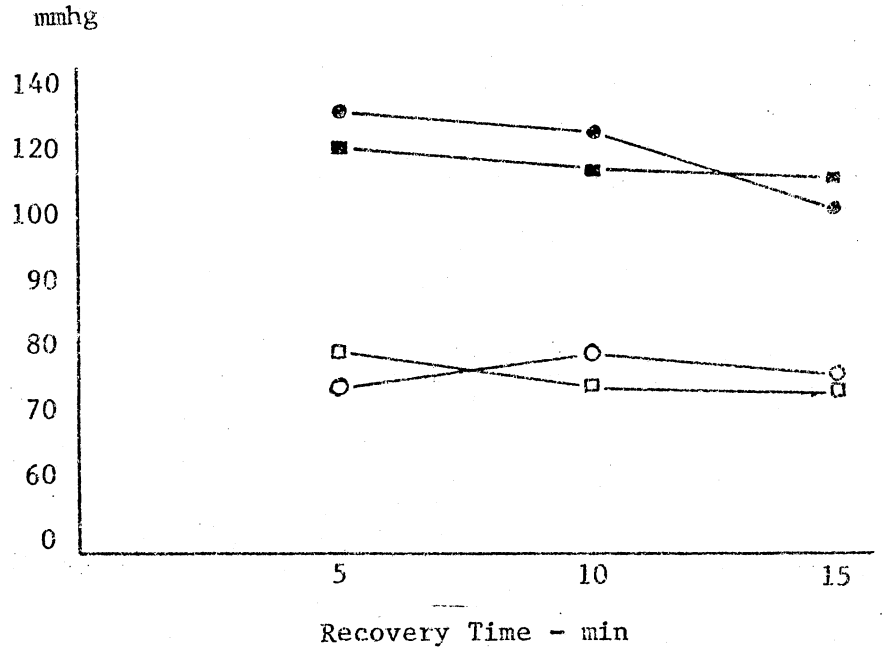


Figure 2. Pre and Posttest Recovery Blood Pressure for Phosphate Group

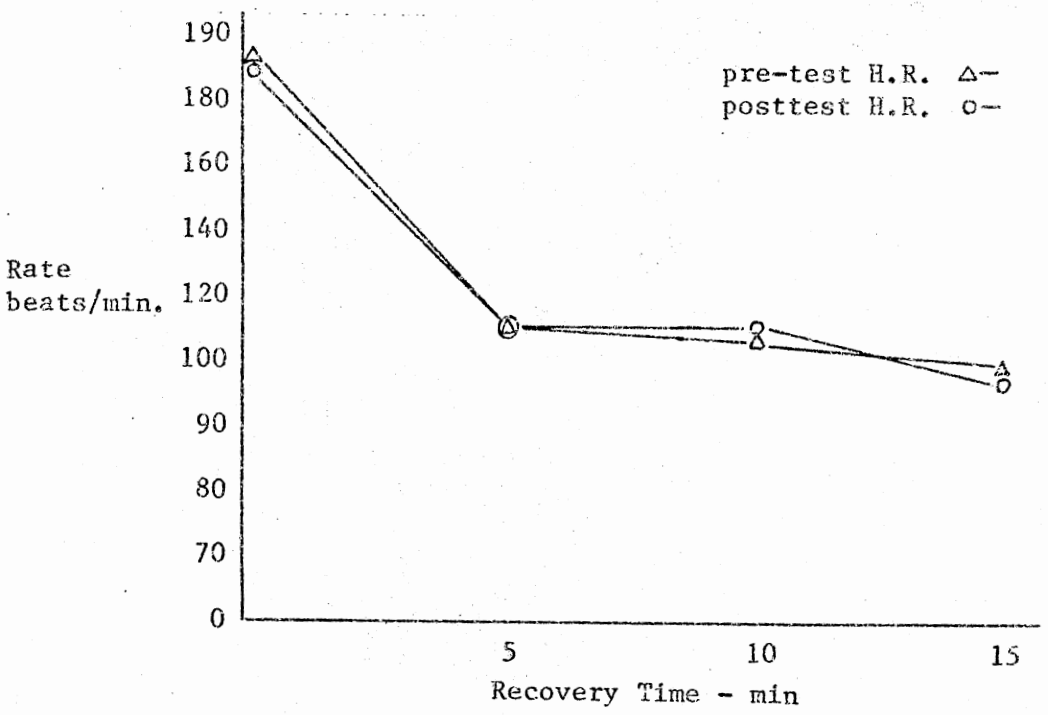


Figure 3. Pre and Posttest Recovery Heart Rate for Placebo Group

Recovery

Five minute recovery heart rate for the phosphate group was 97.33 beats per minute, and for the placebo was 110.66 beats per minute. This revealed a difference of 13.33 beats per minute. The t value of 2.37 was significant at .05 level of confidence (Table IV and Appendix B, Table XXVIII). This means that during the first five minute recovery the phosphate group showed faster recovery heart rate than the placebo group.

TABLE IV

t-VALUES BETWEEN PHOSPHATE AND PLACEBO GROUPS: POSTTEST RECOVERY HEART RATE

Variables	t-value	Significance
5 minute recovery H.R.	2.37	.05
10 minute recovery H.R.	2.29	NS
15 minute recovery H.R.	2.87	.05

Ten minutes recovery heart rate for the phosphate group was 90 beast per minute and for the placebo group was 103 beats per minute. This revealed a mean difference of 13 beats per minute. The t value of 2.29 was not significant at .05 level of confidence (Appendix B, Table XXIX). This means during the ten minute recovery both groups showed no significant change from the pre-test rates which means that both groups recovered at almost the same rate.

Fifteen minutes recovery heart rate for the phosphate group was 82.66 beats per minute, and for the placebo group was 98 beats per minute. This revealed a mean difference of 15.34 beats per minute. The t value of 2.87 was significant at .01 level of confidence (Appendix B, Table XXX and Figure 5). This indicates a better heart rate recovery for the phosphate group at fifteen minute recovery.

There was no significant difference between the phosphate group and placebo group fifteen minute recovery blood pressure means (Figures 2 and 4). The t values of the 5, 10, and 15 minute recoveries for systolic were t_5= 1.91, t_{10}= .85, t_{15}= .16 respectively (Table V and Appendix B, Tables XXXI, XXXII, and XXXIII). For the diastolic they were t_5= 1.32, t= .5, t_{15}= .32 respectively (Appendix B, Tables XXXIV, XXXV, and XXXVI).

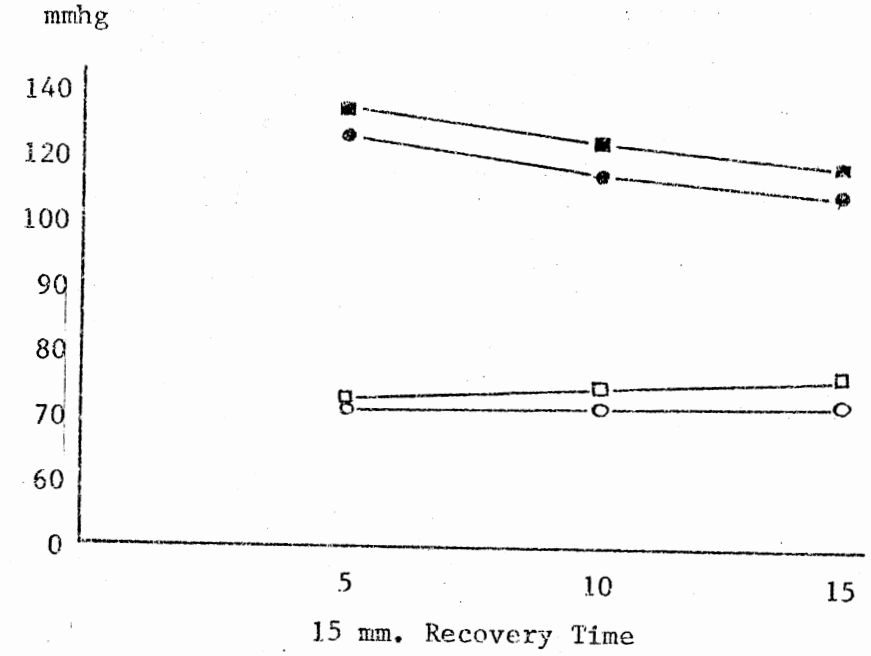
TABLE V

t-VALUES BETWEEN PHOSPHATE AND PLACEBO GROUPS: POSTTEST RECOVERY BLOOD PRESSURE

Variables	t-value	Significance
5 minute recovery B.P.	1.91	NS
10 minute recovery B.P.	.85	NS
15 minute recovery B.P.	.16	NS

Summary and Discussion

The purpose of this study was to measure the effect of phosphate on



pre systolic BP ●–
pre diastolic BP o–
post systolic BP ■–
post diastolic BP □–

Figure 4. Pre and Posttest Recovery Blood Pressure for Placebo Group

lactic acid accumulation, maximum Vo_2, maximum heart rate, 15 minutes recovery heart rate and fifteen minutes recovery blood pressure following maximal treadmill run. The second purpose was to compare the posttest phosphate group results with the posttest placebo group results.

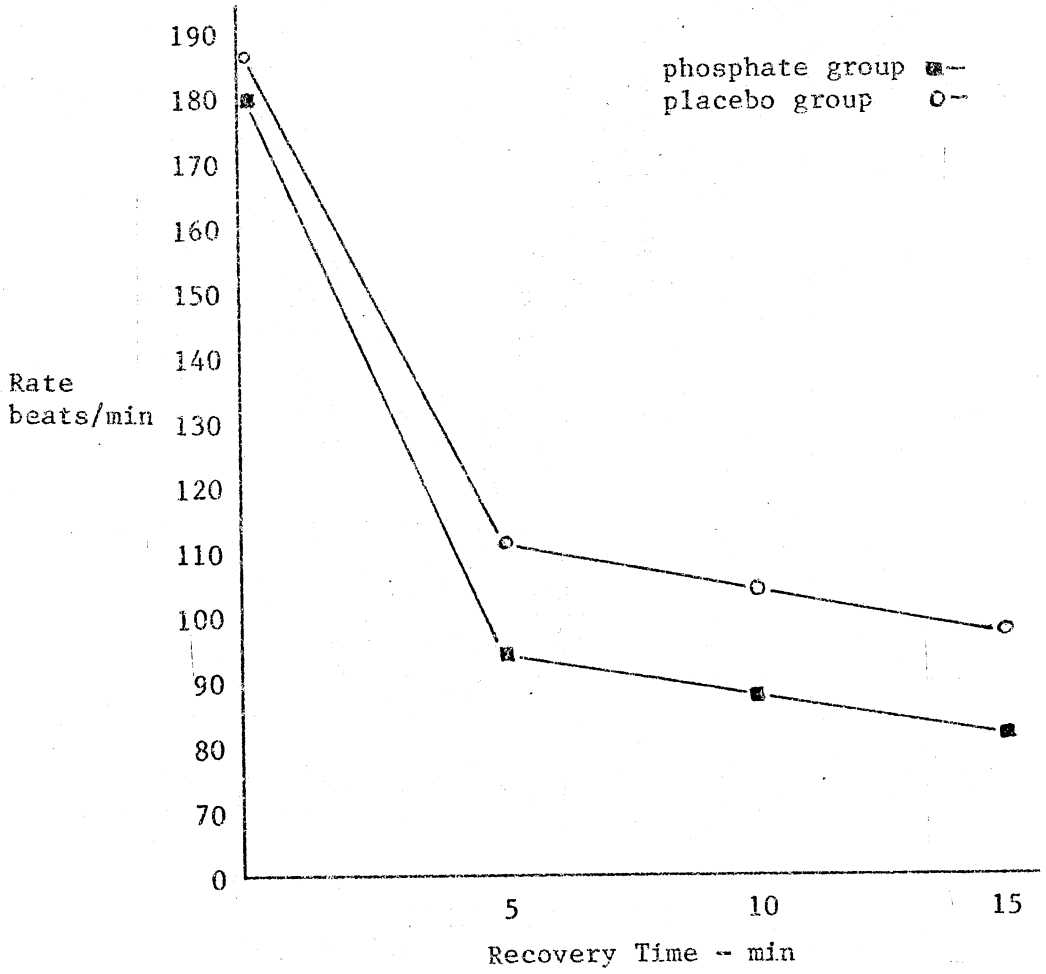


Figure 5. Posttest Recovery Heart Rate for Phosphate and Placebo Groups

The phosphate group showed a significant increase in maximum heart rate ($P<.05$), maximum Vo_2 ($P<.05$), and significant decrease in maximum lactic acid ($P<.05$), and fifteen minutes recovery heart rate ($P<.05$). There was no significant change in the recovery blood pressure. It was expected that the phosphate group would recover faster than those who did not take phosphate (placebo group) prior to their work-out. It was expected that those who participated in the sixteen day interval training and used phosphate prior to the activity would have more maximum Vo_2, less lactic acid, less maximum heart rate and faster recovery than those who did exercise but did not take phosphate. However, those who did not take phosphate (placebo group) had some improvement due to exercise, but this improvement was not significant at .05 level.

The difference between the posttest means for the phosphate group and placebo group showed a significant difference in maximum heart rate and fifteen minutes recovery heart rate. It was expected that those who took phosphate would recover faster. There was no significant difference between the posttest maximum lactic acid and maximum Vo_2 means for the phosphate and placebo group. The lactic acid improved in the phosphate group but was a little short of significance about (.03). It was the author's opinion that the significant improvment of the phosphate group was due to Stim-O-Stam feeding prior to all-out treadmill run.

Finally the group which took phosphate (Stim-O-Stam) did show lower lactic acid, maximum heart rate, and recovered faster than those who did not take phosphate (placebo group) as indicated in the result of this study. The results of this study showed a similar effect on the performance of the subjects as did the previous phosphate studies. Some of the previous studies were outdated and some were unpublished reports. These results were new and more updated.

CHAPTER V

CONCLUSIONS AND RECOMMENDATIONS

There have been many coaches who believe that their athletes will increase their athletic performance if they take some kind of ergogenic aid prior to the competition.

Lundsgard (52) was able to show that the iodoecetate poisoned muscle performed lactic acid free contractions at the same time as energy-rich phosphate (ergogenic aid) compounds were injected in the muscle.

With the current interest in ergogenic aid, most athletes follow the mainstream of ergogenic aid users and take some kind of vitamin or food supplement for extra energy.

The unique aspect of this study was to determine whether or not energy-rich phosphate taken as a food suplement had any effect on the athlete's potential for performance.

Eighteen male track athletes from Oklahoma State University were pre-tested, using the all out treadmill run. The results of the pretest were used to divide the entire group (18 people) into two equal groups: phosphate group and placebo group. The author used a doubleblind study for this experiment. Over the prescribed sixteen days, the subjects performed a similar activity. During the sixteen day period the phosphate group took the phosphate tablet and the placebo group took the color-coded placebo tablet. The author was not able to distinguish who

was taking phosphate and who was taking placeboes. At the end of the posttest, however the author was able to find out who was in the phosphate group and who was in the placebo group.

A paired t-test was used to analyze the changes between pre-test and posttest scores for both groups. Also paired t-test was used to analyze the difference between the posttest results for both groups. Mean raw data differences were tested for significance of differences at the .05 level of confidence.

Summary of Finding

1. The differences between pre and post test lactic acid means for the phosphate group was significant at the .05 level of confidence; therefore, the null hypothesis was rejected (Appendix B, Table XIX).

2. The differences between pre and posttest maximum Vo_2 means for the phosphate group was significant at the .05 level of confidence; therefore, the null hypothesis was rejected (Appendix B, Table XX).

3. The differences between pre and posttest maximum heart rate means for the phosphate group was significant at the .05 level of confidence, therefore the null hypothesis was rejected (Appendix B, Table XII).

4. The differences between pre and posttest lactic acid means for placebo group was not significant at the .05 level of confidence, therefore the null hypothesis was accepted (Appendix B, Table XXI).

5. The differences between pre and posttest maximum Vo_2 means for the placebo group was not significant at the .05 level of confidence, therefore the null hypothesis was accepted (Appendix B, Table XXIII).

6. The differences between pre and posttest maximum heart rate means for the placebo group was not significant at the .05 level of confidence; therefore, the null hypothesis was accepted (Appendix B, Table XXIV).

7. The differences between posttest lactic acid means for the phosphate and the placebo groups was not significant at the .05 level of confidence; therefore, the null hypothesis was accepted (Appendix B, Table XXV).

8. The differences between posttest maximum Vo_2 means for the phosphate and the placebo groups was not significant at the .05 level of confidence; therefore, the null hypothesis was accepted (Appendix B, Table XXVI).

9. The differences between posttest maximum heart rate means for the phosphate and the placebo groups was significant at the .05 level of confidence; therefore, the null hypothesis was rejected (Appendix B, Table XXVII).

10. The differences between posttest five minutes recovery heart rate means for the phosphate and placebo groups was significant at the .05 level of confidence; therefore, the null hypothesis was rejected (Appendix B, Table XXIII).

11. The differences between posttest ten minutes recovery heart rate means for the phosphate and placebo groups was not significant at the .05 level of confidence; therefore, the null hypothesis was accepted (Appendix B, Table XXIX).

12. The differences between posttest fifteen minutes recovery heart rate means for the phosphate and placebo groups was significant at the .05 level of confidence; therefore, the null hypothesis was rejected (Appendix B, Table XXX).

13. The differences between posttest five, ten and fifteen minutes recovery blood pressure means for the phosphate and placebo groups was not significant at the .05 level of confidence; therefore, the null hypothesis was accepted (Appendix B, Tables XXXI-XXXVI).

Based on the hypothesis stated and the limits of this study the following conclusion was drawn: the phosphate (Stim-O-Stam) feeding thirty minutes prior to all-out treadmill run showed a significant change which improved the subjects performance concerning the maximum lactic acid, maximum heart rate and fifteen minute recovery heart rate.

Recommendations

There is no question that more studies are needed to determine the effects of the energy-rich phosphate on human performance. When investigating athletes, it would be advantageous in the future to use a homogeneous group of athletes such as sprinters, middle distance runners, and swimmers, etc. This will aid the investigator in dividing the group into equal or matched groups.

It is also recommended that more subjects be used. This should reduce the problems of variability associated with a small group of less than thirty people.

In future studies the investigator should compare or correlate the amount of lactic-acid with the sprint running time, such as 200 or 400 meters. During the recovery the blood lactic should be tested every minute up to ten to fifteen minutes and correlate with the recovery heart rate. Also, the investigator should give the subjects Stim-O-Stam after the maximal work to see if the recovery heart rate and lactic acid content will decrease.

The investigator should measure lactic acid level and pH of the blood at rest, pre-exercise and at various time intervals after taking Stim-O-Stam.

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APPENDIXES

APPENDIX A

RAW DATA FOR PLACEBO GROUP AND PHOSPHATE GROUP

TABLES VI THROUGH IX

TABLE VI

FIVE, TEN AND FIFTEEN MINUTE RECOVERING HEART RATE AND RECOVERING BLOOD PRESSURE FOR PLACEBO GROUP

Subject	Age	Height	5 min. Recovering Heart Rate		10 min. Recovering Heart Rate		15 min. Recovering Heart Rate		5 min. Recovering Blood Pressure		10 min. Recovering Blood Pressure		15 min. Recovering Blood Pressure	
			Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post
1	19	6'4"	114	114	108	102	102	102	140/70	145/65	120/75	130/70	120/75	130/75
2	20	5'10"	108	108	102	96	102	96	140/70	140/70	120/70	120/75	115/70	115/70
3	18	5'9"	96	96	126	90	120	74	130/70	120/70	110/70	110/70	105/70	105/70
4	20	5'7"	108	132	102	126	102	120	140/70	135/70	115/70	125/75	115/75	115/75
5	18	5'6"	108	102	74	96	74	96	130/75	130/75	115/75	120/75	110/75	110/75
6	19	6'4"	108	114	108	108	90	92	135/70	140/65	115/70	130/70	110/70	120/70
7	19	6'2"	96	102	90	96	90	96	130/70	130/75	115/70	125/75	110/70	115/75
8	20	5'10"	120	120	120	114	114	108	120/75	130/75	115/70	120/75	110/70	115/75
9	19	6'2"	114	108	96	102	90	102	120/75	120/75	110/70	115/75	105/70	115/75
	171	52'5"	996	996	926	930	884	886	1175/645	1190/640	1035/640	1095/660	1000/645	1040/660
M	19	5'0¼"	110.66	110.66	102.88	103	98.22	98	130.55/71.66	132.20/71.11	115/71.11	121.66/73.33	111.11/71.66	115.55/73.33

TABLE VII

RESTING HEART BEATS, MAX HEART RATE, MAX VO_2 AND LACTIC ACID FOR PLACEBO GROUP

Subject	Age	Height	Weight		Resting Heart Beats		Maximum Heart Rate		Maximum VO_2		Lactic Acid	
			Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post
1	19	6'4"	180	182	62	60	186	186	48.17	59.40	9.78	12.04
2	20	5'10"	715	176	64	62	186	186	64.00	70.70	9.28	9.66
3	18	5'9"	157	156	60	60	180	180	73.10	67.56	11.38	7.23
4	20	5'7"	152	163	66	64	180	180	69.22	68.27	11.63	7.20
5	18	5'6"	150	146	68	66	192	192	42.00	66.30	9.78	9.92
6	18	6'4"	190	188	70	68	192	192	55.41	47.27	11.05	9.87
7	19	6'2"	173	176	72	70	192	180	58.85	62.37	12.20	7.85
8	20	5'10"	150	152	68	68	186	186	54.35	65.54	11.41	9.28
9	19	6'2"	169	169	68	64	186	192	52.79	69.90	10.64	7.89
	171	53'5"	1496	1508	598	582	1680	1674	517.94	569.00	96.10	81.40
M	19	5'9½"	166.22	167.55	66.4	64.6	186.66	186	57.54	63.22	10.67	9.04
SD							4.690	5.196	9.936	6.914	0.937	1.528
SEM							1.563	1.732	3.312	2.306	0.312	0.509

TABLE VIII

FIVE, TEN AND FIFTEEN MINUTE RECOVERING HEART RATE AND RECOVERING BLOOD PRESSURE FOR PHOSPHATE GROUP

Subject	Age	Height	5 min. Recovering Heart Rate		10 min. Recovering Heart Rate		15 min. Recovering Heart Rate		5 min. Recovering Blood Pressure		10 min. Recovering Blood Pressure		15 min. Recovering Blood Pressure	
			Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post
1	20	5'10"	120	108	102	96	102	90	140/65	125/65	120/70	115/65	110/70	110/70
2	18	5'8"	108	96	96	90	96	84	150/75	135/80	130/80	130/80	115/80	120/80
3	19	5'8"	108	84	102	78	96	72	135/70	130/75	115/75	120/80	105/75	120/70
4	20	6'2"	126	108	126	102	114	90	130/30	130/80	125/80	120/85	125/80	120/80
5	20	5'10"	120	102	108	96	108	78	130/75	125/75	125/80	120/75	110/80	120/75
6	19	6'3"	102	96	96	84	90	78	130/60	125/70	115/75	120/70	110/65	100/70
7	18	5'9"	102	84	96	78	90	78	120/70	115/70	112/70	110/70	105/70	105/70
8	19	6'2"	108	102	102	96	96	90	130/75	120/75	115/75	115/75	105/70	110/70
9	19	5'8"	114	96	96	90	96	84	140/70	130/70	135/70	120/70	130/70	120/70
	171	53'6"	1008	876	924	810	888	744	1205/640	1135.559	1087/675	1070/670	1025/660	1045/665
M	19	5'9½"	112	97.33	102.66	90	98.66	82.66	133.85 / 71.11	126.11 / 73.33	120.77 / 75	118.88 / 74.44	113.88 / 73.33	116.11 / 72.77

TABLE IX

RESTING HEART BEATS, MAX HEART RATE, MAX VO_2 AND LACTIC ACID FOR PHOSPHATE GROUP

Subject	Age	Height	Weight		Resting Heart Beats		Maximum Heart Rate		Maximum VO_2		Lactic Acid	
			Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post
1	20	5'10"	147	152	68	60	192	180	36.27	57.27	10.18	9.34
2	18	5'8"	147	147	72	70	192	180	33.80	45.79	10.82	5.87
3	19	5'8"	148	148	68	62	186	180	63.63	59.70	11.43	6.44
4	20	6'2"	185	184	64	60	186	186	47.42	57.68	10.96	9.37
5	20	5'10"	165	166	68	62	186	180	40.50	57.70	11.10	7.13
6	19	6'3"	181	179	60	60	180	174	45.93	59.73	9.62	4.90
7	18	5'9"	142	146	58	56	180	174	46.46	50.75	11.50	4.12
8	19	6'2"	184	179	72	64	192	186	61.57	62.31	9.98	9.19
9	19	5'8"	152	157	78	70	192	180	50.00	61.09	9.37	7.43
	171	53'6"	1451	1458	608	564	1686	1620	426.38	514.82	94.46	63.29
M	19	5'9½"	161.22	162	67.5	62.6	187.33	180	47.37	57.20	10.49	7.00
SD							5.000	4.243	10.218	4.563	0.873	2.023
SEM							1.667	1.414	3.406	1.54	0.291	0.174

APPENDIX B

TABLES X THROUGH XXXVI

TABLE X

ENTIRE GROUP PRE-TEST

Variable	N	Mean	Standard Deviation	Standard Error of The Mean
Resting Heart rate	18	66.95	--	--
Max. Heart rate	18	186.99	4.845	1.615
Max. Lactic Acid (mg LA/10 ml plasma)	18	10.58	.905	.3015
VO_2 max. (ml/kg/min)	18	52.45	10.077	3.359
5 min. Recovery Heart rate	18	111.33	--	--
5 min. Recovery Blood Pressure	18	132.2/71.38	--	--
10 min. Recovery Heart Rate	18	102.77	--	--
10 min. Recovery Blood Pressure	18	117.88/73	--	--
15 min. Recovery Heart Rate	18	98.44	--	--
15 min. Recovery Blood Pressure	18	112.49/72.49	--	--

TABLE XI

ENTIRE GROUP POST TEST

Variable	N	Mean	Standard Deviation	Standard Error of The Mean
Resting Heart rate	18	63.6	--	--
Max. Heart rate	18	183	4.719	1.573
Max VO_2	18	60.21	5.741	1.015
Max. Lactic Acid	18	8.02	1.775	.591
5 min. Recovery Heart rate	18	103.79	--	--
5 min. Recovery Blood Pressure	18	129.16/72.22	--	--
10 min. Recovery Heart Rate	18	96.5	--	--
10 min. Recovery Blood Pressure	18	120.71/74.05	--	--
15 min. Recovery Heart Rate	18	90.33	--	--
15 min. Recovery Blood Pressure	18	115.83/73.05	--	--

TABLE XII

DIFFERENCES BETWEEN PRE AND POST TEST FOR ENTIRE GROUP

Variable	N	Difference	Standard Deviation	Standard Error of The Mean
Resting Heart rate	18	3.35	--	--
Max. Heart rate	18	3.99	.126	.042
Max. Lactic Acid (mg LA/10 ml plasma)	18	2.56	.87	.289
VO_2 max. (ml/kg/min)	18	-7.76	4.336	2.344
5 min. Recovery Heart rate	18	7.34	--	--
5 min. Recovery Blood Pressure	18	3.04/-.84	--	--
10 min. Recovery Heart Rate	18	6.27	--	--
10 min. Recovery Blood Pressure	18	-2.83/-1.05	--	--
15 min. Recovery Heart Rate	18	8.11	--	--
15 min. Recovery Blood Pressure	18	-3.34/-.56	--	--

TABLE XIII

PHOSPHATE GROUP PRE-TEST

Variable	N	Mean	Standard Deviation	Standard Error of The Mean
Resting Heart rate	9	67.05	--	--
Max. Heart rate	9	187.33	5.000	1.667
Max. Lactic Acid (mg LA/10 ml plasma)	9	10.49	0.873	0.291
VO_2 max. (ml/kg/min)	9	47.37	10.218	3.406
5 min. Recovery Heart rate	9	112	--	--
5 min. Recovery Blood Pressure	9	133.88/71.11	--	--
10 min. Recovery Heart Rate	9	102	--	--
10 min. Recovery Blood Pressure	9	120.77/75	--	--
15 min. Recovery Heart Rate	9	98	--	--
15 min. Recovery Blood Pressure	9	113.88/73.33	--	--

TABLE XIV

PHOSPHATE GROUP POST TEST

Variable	N	Mean	Standard Deviation	Standard Error of The Mean
Resting Heart rate	9	62.6	--	--
Max. Heart rate	9	180	4.243	1.414
Max. Lactic Acid (mg LA/10 ml plasma)	9	7.00	2.023	0.674
VO_2 max. (ml/kg/min)	9	57.20	4.563	1.521
5 min. Recovery Heart rate	9	97.33	--	--
5 min. Recovery Blood Pressure	9	126.11/73.33	--	--
10 min. Recovery Heart Rate	9	90	--	--
10 min. Recovery Blood Pressure	9	118.88/74.44	--	--
15 min. Recovery Heart Rate	9	82.66	--	--
15 min. Recovery Blood Pressure	9	116.11/72.77	--	--

TABLE XV

DIFFERENCES BETWEEN PRE AND POST TEST FOR PHOSPHATE GROUP

Variable	N	Mean	Standard Deviation	Standard Error of The Mean
Resting Heart rate	9	4.9	--	--
Max. Heart rate	9	7.33	0.757	0.253
Max. Lactic Acid (mg LA/10 ml plasma)	9	3.49	-1.15	-.383
VO_2 max. (ml/kg/min)	9	-9.83	5.655	1.885
5 min. Recovery Heart rate	9	14.67	--	--
5 min. Recovery Blood Pressure	9	7.79/-2.22	--	--
10 min. Recovery Heart Rate	9	12	--	--
10 min. Recovery Blood Pressure	9	1.89/.56	--	--
15 min. Recovery Heart Rate	9	15.34	--	--
15 min. Recovery Blood Pressure	9	-2.23/.56	--	--

TABLE XVI

PLACEBO GROUP PRE-TEST

Variable	N	Mean	Standard Deviation	Standard Error of The Mean
Resting Heart rate	9	66.4	--	--
Max. Heart rate	9	186.66	4.690	1.563
Max. Lactic Acid (mg LA/10 ml plasma)	9	10.67	.937	.312
VO_2 max. (ml/kg/min)	9	57.54	9.936	3.312
5 min. Recovery Heart rate	9	110.66	--	--
5 min. Recovery Blood Pressure	9	130.55/71.66	--	--
10 min. Recovery Heart Rate	9	102.88	--	--
10 min. Recovery Blood Pressure	9	115./71.11	--	--
15 min. Recovery Heart Rate	9	98.22	--	--
15 min. Recovery Blood Pressure	9	111.11/71.66	--	--

TABLE XVII

PLACEBO GROUP POST TEST

Variable	N	Mean	Standard Deviation	Standard Error of The Mean
Resting Heart rate	9	64.6	--	--
Max. Heart rate	9	186	5.196	1.732
Max. Lactic Acid (mg LA/10 ml plasma)	9	9.04	1.528	.509
VO_2 max. (ml/kg/min)	9	63.22	6.919	2.306
5 min. Recovery Heart rate	9	110.66	--	--
5 min. Recovery Blood Pressure	9	132.22/11.11	--	--
10 min. Recovery Heart Rate	9	103	--	--
10 min. Recovery Blood Pressure	9	121.66/73.33	--	--
15 min. Recovery Heart Rate	9	98	--	--
15 min. Recovery Blood Pressure	9	115.55/73.3	--	--

TABLE XVIII

DIFFERENCES BETWEEN PRE- AND POSTTEST FOR PLACEBO GROUP

Variable	N	Mean	Standard Deviation	Standard Error of The Mean
Resting Heart rate	9	-1.8	--	--
Max. Heart rate	9	-- .66	.506	.169
Max. Lactic Acid	9	-1.63	.591	.197
VO_2 max. (ml/kg/min)	9	5.68	-3.017	-1.006
5 min. Recovery Heart rate	9	0	--	--
5 min. Recovery Blood Pressure	9	1.67/-.55	--	--
10 min. Recovery Heart Rate	9	.12	--	--
10 min. Recovery Blood Pressure	9	6.66/2.22	--	--
15 min. Recovery Heart Rate	9	- .22	--	--
15 min. Recovery Blood Pressure	9	4.44/1.67	--	--

TABLE XIX

COMPARISON OF LACTIC ACID MEANS BETWEEN THE PRE-TEST AND POSTTEST FOR PHOSATE GROUP

	Pre-test	Posttest	Difference	
No.	X_1	X_2	X	X_2
1	11.50	4.12	7.38	54.44
2	11.43	6.44	4.99	24.90
3	11.10	7.13	3.97	15.76
4	10.96	9.37	1.59	2.52
5	10.82	5.87	4.95	24.50
6	10.18	9.34	.84	.70
7	9.98	9.19	.79	.624
8	9.12	4.40	4.72	22.278
9	9.37	7.43	1.94	3.76

$X_1 = 94.46$ $X_2 = 63.29$ $X = 31.17$ $X^2 = 149.48$

$X_1 = 10.495$ $X_2 = 7.00$

$$t^2 = \frac{(X)^2 (X_1 - 1)}{NEX^2 - (X)^2} = \frac{(31.17)^2 (8)}{9(149.48) - (31.17)^2} = \frac{7772}{1345.32 - 971.568}$$

$$= \frac{7772}{373.752} = 20.794 = t^2$$

$t^2 = 20.794$
$t = 4.56$

Probability at the .05 level of confidence is 2.31
Probability at the .02 level of confidence is 2.90
Probability at the .01 level of confidence is 3.36

TABLE XX

COMPARISON OF MAX VO_2 MEANS BETWEEN THE PRE-TEST AND POSTTEST FOR PHOSPHATE GROUP

Variable	Number	Mean Ml-kg-min	Mean Difference	Standard Deviation	S. Error of Mean	T Value
X_1	9	47.375	-9.826	10.218	3.406	-3.65*
X_2		57.202		4.563	1.521	

*$p < .01$

TABLE XXI

COMPARISON OF MAX. HEART RATE MEANS BETWEEN THE PRE-TEST AND POSTTEST FOR PHOSPHATE GROUP

Variable	Number	Mean Ml-kg-min	Mean Difference	Standard Deviation	S. Error of Mean	T Value
X_1	9	187.333	7.333	5.000	1.667	5.50*
X_2		180.000		4.243	1.414	

*$p < .01$

TABLE XXII

COMPARISON OF LACTIC ACID MEAN BETWEEN THE PRE-TEST AND POSTTEST FOR PLACEBO GROUP

Variable	Number	Mean Ml-kg-min	Mean Difference	Standard Deviation	S. Error of Mean	T Value
X_1	9	10.677	1.633	0.937	0.312	2.23
X_2		9.044		1.528	0.509	

* N.S.

TABLE XXIII

COMPARISON OF MAX VO_2 MEANS BETWEEN THE PRE-TEST AND POSTTEST FOR PLACEBO GROUPS

Variable	Number	Mean Ml-kg-min	Mean Difference	Standard Deviation	S. Error of Mean	T Value
X_1	9	57.548	-5.673	9.936	3.312	-1.49*
X_2		63.222		6.919	2.306	

*N.S.

TABLE XXIV

COMPARISON OF MAX. HEART RATE MEAN BETWEEN THE PRE AND POSTTEST FOR PLACEBO GROUPS

Variable	Number	Mean Ml-kg-min	Mean Difference	Standard Deviation	S. Error of Mean	T Value
X_1	9	186.666	0.666	4.690	1.563	0.43*
X_2		186.000		5.196	1.732	

*N.S.

TABLE XXV

COMPARISON OF POSTTEST LACTIC ACID MEANS BETWEEN THE PHOSPHATE AND PLACEBO GROUPS

Variable	Number	Mean Ml-kg-min	Mean Difference	Standard Deviation	S. Error of Mean	T Value
X_1	9	7.032	-2.016	2.023	0.674	-2.28*
X_2		9.048		1.529	0.510	

*N.S.

TABLE XXVI

COMPARISON OF POSTTEST % OF VO_2 GAIN BETWEEN THE PHOSPHATE AND PLACEBO GROUPS

Variable	Number	Mean Ml-kg-min	Mean Difference ml-kg-min	Standard Deviation	S. Error of Mean	T Value
X_1	9	62.444	-5.6956	11.378	3.793	1.26*
X_2		57.202	-9.8267	8.075	2.692	

* N.S.

TABLE XXVII

COMPARISON OF POSTTEST MAX. HEART RATE MEANS BETWEEN THE PHOSPHATE AND PLACEBO GROUPS

Variable	Number	Mean Ml-kg-min	Mean Difference	Standard Deviation	S. Error of Mean	T Value
X_1	9	180	-6.000	4.243	1.414	-3.46*
X_2		186		5.196	1.732	

*$p < .05$

TABLE XXVIII

COMPARISON OF POSTTEST FIVE MINUTE RECOVERY HEART RATE MEANS BETWEEN THE PHOSPHATE AND PLACEBO GROUPS

Variable	Number	Mean Ml-kg-min	Mean Difference	Standard Deviation	S. Error of Mean	T Value
X_1	9	110.666	13.333	10.863	3.621	2.37*
X_2		97.33		8.888	2.963	

* $p < .05$

TABLE XXIX

COMPARISON OF POSTTEST TEN MINUTE RECOVERY HEART RATE MEANS BETWEEN THE PHOSPHATE AND PLACEBO GROUPS

Variable	Number	Mean Ml-kg-min	Mean Difference	Standard Deviation	S. Error of Mean	T Value
X_1	9	103.333	13.33	11.136	3.712	2.29*
X_2		90.000		8.485	2.828	

*N.S.

TABLE XXX

COMPARISON OF POSTTEST FIFTEEN MINUTE RECOVERY HEART RATE MEANS BETWEEN THE PHOSPHATE AND PLACEBO GROUPS

Variable	Number	Mean Ml-kg-min	Mean Difference	Standard Deviation	S. Error of Mean	T Value
X_1	9	98.444	15.777	12.441	4.147	2.87*
X_2		82.666		6.557	2.186	

*$p < .05$

TABLE XXXI

COMPARISON OF POSTTEST FIVE MINUTE RECOVERY SYSTOLIC BLOOD PRESSURE MEANS BETWEEN THE PHOSPHATE AND PLACEBO GROUPS

Variable	Number	Mean Ml-kg-min	Mean Difference	Standard Deviation	S. Error of Mean	T Value
X_1	9	132.222	6.111	8.700	2.900	1.91
X_2		126.111		6.009	2.003	

*N.S.

TABLE XXXII

COMPARISON OF POSTTEST TEN MINUTE RECOVERY DIASTOLIC BLOOD PRESSURE MEANS BETWEEN THE PHOSPATE AND PLACEBO GROUPS

Variable	Number	Mean Ml-kg-min	Mean Difference	Standard Deviation	S. Error of Mean	T Value
X_1	9	121.666	2.777	6.614	2.205	0.85*
X_2		118.888		5.465	1.822	

*N.S.

TABLE XXXIII

COMPARISON OF POSTTEST FIFTEEN MINUTE DIASTOLIC BLOOD PRESSURE MEANS BETWEEN THE PHOSPHATE AND PLACEBO GROUPS

Variable	Number	Mean Ml-kg-min	Mean Difference	Standard Deviation	S. Error of Mean	T Value
X_1	9	115.555	-0.555	6.821	2.274	-0.16*
X_2		116.111		6.009	2.003	

*N.S.

TABLE XXXIV

COMPARISON OF POSTTEST FIVE MINUTE RECOVERY DIASTOLIC BLOOD PRESSURE MEANS BETWEEN THE PHOSPHATE AND PLACEBO GROUPS

Variable	Number	Mean Ml-kg-min	Mean Difference	Standard Deviation	S. Error of Mean	T Value
X_1	9	71.111	-2.222	4.167	1.389	-1.32*
X_2		73.333		5.000	1.667	

*N.S.

TABLE XXXV

COMPARISON OF POSTTEST TEN MINUTE DIASTOLIC BLOOD PRESSURE MEANS BETWEEN THE PHOSPHATE AND PLACEBO GROUPS

Variable	Number	Mean Ml-kg-min	Mean Difference	Standard Deviation	S. Error of Mean	T Value
X_1	9	73.333	-1.111	2.5000	0.833	-0.55*
X_2		74.444		6.346	2.115	

*N.S.

TABLE XXXVI

COMPARISON OF POSTTEST FIFTEEN MINUTE DIASTOLIC BLOOD PRESSURE MEANS BETWEEN THE PHOSPHATE AND PLACEBO GROUPS

Variable	Number	Mean Ml-kg-min	Mean Difference	Standard Deviation	S. Error of Mean	T Value
X_1	9	73.333	0.555	2.500	0.833	0.32*
X_2		72.777		4.410	1.470	

*N.S.

Name: Hayrettin Daylan Date of Degree: May, 1982

Institution: Oklahoma State University Location: Stillwater, Oklahoma

Title of Study: THE EFFECT OF PHOSPHATE FEEDING ON LACTIC ACID ACCUMULATION FOLLOWING MAXIMAL WORK

Page in Study: 92 Candidate for Degree of Doctor of Education

Major Field: Higher Education

Minor Field: Health, Physical Education, and Recreation

Scope and Method of Study: The purpose of this study was to measure the effect of phosphate on lactic acid accumulation following maximal work. 18 volunteers from the Oklahoma State University varsity track team were selected as subjects. The subjects were men between the ages of 18 and 22. The subjects were tested in the Oklahoma State University Physiology of Exercise Laboratory for lactic acid, maximum Vo_2, maximum heart rate, 15 minutes recovery heart rate and blood pressure following all-out treadmill run. The subjects were pre-tested at the beginning of the study then the subjects were randomly assigned to two groups on the basis of their lactic-acid results on the pre-test to form two matched groups, then randomly assigned to the phosphate group and the placebo group. After 16 days of phosphate and placebo feeding, the subjects were retested (posttest) to check for the differences in lactic acid, maximum Vo_2, maximum heart rate, 15 minutes recovery heart rate and blood pressure. This study was a double blind study.

Findings and Conclusions: The pre-test results and posttest means of each variable were evaluated with a t-ratio to test for significance of differences between means. Values for "t" between the pre and posttest lactic acid, maximum VO_2 and maximum heart rate within the phosphate group were 4.56*, 3.65*, and 5.50* respectively. Since a t-value of 2.31 is necessary for significance at the .05 level of confidence, the null hypothesis was rejected. Values for "t" between pre and posttest lactic acid, maximum VO_2 and maximum heart rate within the placebo group were 2.23, 1.49, and .43 respectively. Since the t-values are under 2.31, the null hypothesis was accepted. The value for t on posttest lactic acid between phosphate and placebo groups was 2.28. This was not significant at the .05 level of confidence. The value for t on posttest maximum heart rate between phosphate and placebo groups was 3.46*. This was significant at the .05 level of confidence. The value for t on posttest 15 minute heart rate between groups was 2.87*. This was significant at the .05 level of confidence. The value for t on the posttest 15 minute recovery blood pressure between groups was .16. This was not significant at the .05 level of confidence, therefore the null hypothesis was accepted. Overall, the 16 day phosphate feeding had a significant effect on the phosphate group.

ADVISER'S APPROVAL A. B. Harrison

VITA 2

Hayrettin Daylan

Candidate for the Degree of

Doctor of Education

Thesis: THE EFFECT OF PHOSPHATE FEEDING ON LACTIC ACID ACCUMULATION FOLLOWING MAXIMAL WORK

Major Field: Higher Education

Minor Field: Health, Physical Education, and Recreation

Biographical:

Personal Data: Born in Nusaybin-Mardin, Turkey, January 1, 1953, the son of Mr. and Mrs. Daylan.

Education: Graduated from Hasan Oglan Teacher Training School, Ankara, in 1971; received the Bachelor of Science in Secondary Education degree from Oklahoma State University with a major in Physical Education in 1977; received the Master of Science degree from Oklahoma State University in 1979; completed the requirements for Doctor of Education degree at Oklahoma State University in May, 1982.

Professional Experience: Taught second and third grades for one year, then went to Physical Education of Anlara for three semesters as an undergraduate student. After that won a nationwide scholarship to come to Oklahoma State University. Walked on the varsity track team, and coached intramural volleyball. Coached volleyball and soccer at private club in California; also managed the speed soccer stadium at California for one semester in 1978. Worked at the Physiology Exercise Laboratory as a laboratory technician for two years (1977-1978, 1978-1979). Presently teaching body massage at Oklahoma State University and assisting Oklahoma State University varsity track coach, Ralph Tate, since 1978.

Publications: Published an article in the Athletic Journal, 1980 "How to Discover and Train Born Sprinters", and ready to be published Track and Field chapter for Oklahoma State University HPELS department; and ready to publish "How to Cure Tension Headache and Body Aches with Turkish Massage".