Fall 1995

Effect of Oral Creatine Monohydrate Supplementation on Body Mass and Composition

Dennis Michael Burke
Old Dominion University

EFFECT OF ORAL CREATINE MONOHYDRATE SUPPLEMENTATION ON BODY MASS AND COMPOSITION

by

Dennis Michael Burke

B.S. May 1993, Old Dominion University

A Thesis submitted to the faculty of
Old Dominion University in Partial Fulfillment of the
Requirement for the Degree of

Master of Science in Education with
Emphasis in Exercise Science and Wellness
Department of Health, Physical Education, and Recreation
Old Dominion University
December, 1995

Approved by:

Melvin H. Williams
(Director)

Elizabeth A. Dowling

Brian C. Leutholtz

Diego R. Redondo

ABSTRACT

Effect Of Oral Creatine Monohydrate Supplementation
On Body Mass And Composition

Dennis M. Burke
Old Dominion University, 1995

Oral creatine monohydrate supplementation has been suggested to be an effective nutritional supplement to increase body mass. Sixteen male college baseball players, participating in an off-season, high-intensity strength training program, underwent body composition tests via hydrostatic weighing, skinfolds, and bioelectrical impedance analysis once-a-week for a total of 5 weeks (Trials). After a baseline trial (Trial 1), in a double-blind, placebo, between-subjects design, 8 subjects orally ingested 30 grams (a loading dose) of creatine monohydrate daily for 7 days (Trial 2) and 10 grams (a maintenance dose) of creatine monohydrate daily for 21 days (Trials 2, 3, 4, and 5). Body mass, percent body fat, and lean body mass (LBM) were analyzed using a 2 (Groups) X 5 (Trials) factorial repeated measures ANOVA. A follow-up test for simple main effects was performed when applicable to identify specific differences. For the creatine group, results revealed significant ($p < 0.05$) increases in body mass in Trials 2, 3, 4, and 5 as compared to Trial 1. Using hydrostatic weighing and bioelectrical impedance analysis, creatine monohydrate

supplementation did not significantly ($p > 0.05$) affect percent body fat or LBM. However, by means of skinfolds, LBM did significantly ($p < 0.05$) increase in Trials 2, 3, 4, and 5 as compared to Trial 1. Therefore, results indicate that oral creatine monohydrate supplementation does increase body mass, but as evaluated by three different body composition measurement techniques does not consistently affect percent body fat or LBM.

ABSTRACT

Effect Of Oral Creatine Monohydrate Supplementation
On Body Mass And Composition

Dennis M. Burke
Old Dominion University, 1995

Oral creatine monohydrate supplementation has been suggested to be an effective nutritional supplement to increase body mass. Sixteen male college baseball players, participating in an off-season, high-intensity strength training program, underwent body composition tests via hydrostatic weighing, skinfolds, and bioelectrical impedance analysis once-a-week for a total of 5 weeks (Trials). After a baseline trial (Trial 1), in a double-blind, placebo, between-subjects design, 8 subjects orally ingested 30 grams (a loading dose) of creatine monohydrate daily for 7 days (Trial 2) and 10 grams (a maintenance dose) of creatine monohydrate daily for 21 days (Trials 2, 3, 4, and 5). Body mass, percent body fat, and lean body mass (LBM) were analyzed using a 2 (Groups) X 5 (Trials) factorial repeated measures ANOVA. A follow-up test for simple main effects was performed when applicable to identify specific differences. For the creatine group, results revealed significant ($p < 0.05$) increases in body mass in Trials 2, 3, 4, and 5 as compared to Trial 1. Using hydrostatic weighing and bioelectrical impedance analysis, creatine monohydrate

supplementation did not significantly ($p > 0.05$) affect percent body fat or LBM. However, by means of skinfolds, LBM did significantly ($p < 0.05$) increase in Trials 2, 3, 4, and 5 as compared to Trial 1. Therefore, results indicate that oral creatine monohydrate supplementation does increase body mass, but as evaluated by three different body composition measurement techniques does not consistently affect percent body fat or LBM.

EFFECT OF ORAL CREATINE MONOHYDRATE SUPPLEMENTATION ON BODY MASS AND COMPOSITION

by

Dennis Michael Burke

B.S. May 1993, Old Dominion University

A Thesis submitted to the faculty of

Old Dominion University in Partial Fulfillment of the

Requirement for the Degree of

Master of Science in Education with

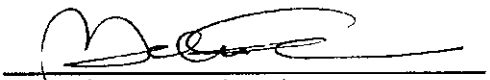
Emphasis in Exercise Science and Wellness

Department of Health, Physical Education, and Recreation

Old Dominion University

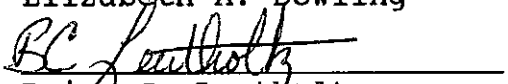
December, 1995

Approved by:

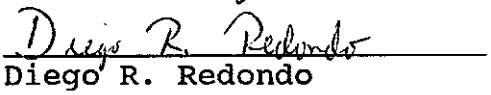


Melvin H. Williams
(Director)

Elizabeth A. Dowling



Brian C. Leutholtz



Diego R. Redondo

DEDICATION

This manuscript is dedicated in loving memory of my father William Henry Burke. In his battle against cancer, he has taught me that whatever the fear may be to stand tall, hold my head up high, and look the fear in the eyes, for every dream is worth pursuing.

ACKNOWLEDGEMENTS

There are many people to thank for their assistance in seeing this research study come to completion. First and foremost, I would like to thank my advisor Dr. Melvin Williams for his wisdom in exercise science. His research etiquette and professionalism has inspired me to develop a keen interest in exercise science research.

Many thanks to my committee members, Dr. Elizabeth Dowling, Dr. Brian Leutholtz, and Dr. Diego Redondo for setting aside time from their busy schedules to provide the guidance needed for me to realize with hard work anything is possible.

My gratitude to coaches Tony Guzzo and Tim Parenton and to the following members of the Old Dominion University Baseball team who made this project possible: Michael H., Jason R., Jesse J., Jason H., Maika S., Thomas S., Ray R., Kevin G., Robert M., Shawn P., Jason K., Robert A., Ryan G., Ron W., Brian F., and Brett W.

Early morning hours and days were volunteered by undergraduate exercise science students. My appreciation is extended to Tonia, Robyn, Kim, Joanne, Jess, Eric, and Lynn who assisted with laboratory preparations and collection of

research data.

Finally, many thanks to Experimental and Applied Science who sponsored this investigation by supplying creatine monohydrate and the placebo.

TABLE OF CONTENTS

LIST OF TABLES

CHAPTER 1

Introduction

An increased body mass may be desirable as a means to enhance performance in certain sports such as American football, competitive bodybuilding and power lifting. Thus, certain athletes have utilized various techniques with training in attempts to gain body weight or lean body mass. One effective method is the use of anabolic steroids, and research has documented that 45 percent (Pope et al., 1988) and 13 percent (Yesalis et al., 1988) of steroid users cited appearance (increase in lean body mass) as the primary reason for using steroids. However, because the use of anabolic steroids is unsafe and illegal in some athletic competitions, athletes have been searching for nutritional means as an effective, yet safe, substitute.

Some research has suggested that creatine monohydrate supplementation may be an effective nutritional ergogenic to increase strength and power in short-term, high-intensity anaerobic exercises (Balsom et al., 1993A; Greenhaff et al., 1993; Harris et al., 1992B; Birch et al., 1994; Earnest et al., 1994; Jacobs et al., 1995). Interestingly, although not a primary dependant variable, several studies also have

reported increases in body weight following creatine monohydrate supplementation (Balsom et al., 1993A; Balsom et al., 1993B; Earnest et al., 1995A; Lemon et al., 1995). Therefore, the use of creatine monohydrate supplementation may be an effective nutritional supplement to increase lean body mass.

Statement of the Problem

At the present time, there is limited data available regarding the effects of oral creatine monohydrate supplementation on body mass and composition. Therefore, the purpose of this study was to determine the effects of oral creatine monohydrate supplementation on body mass and body composition, using a two-component model of percent body fat and lean body mass.

Hypothesis

There will be no significant effect of creatine monohydrate supplementation on body mass, percent body fat or lean body mass.

Significance of the Study

As a safe substitute to anabolic steroids, this study will serve to add further insight on the anabolic effects of oral creatine monohydrate supplementation on body mass, body fat, and lean body mass.

Delimitations

Sixteen male college baseball players, participating in an off-season, high-intensity strength training program,

engaged in one hour of data collection once-a-week for a total of five weeks (trials) in the Human Performance Laboratory. Following a baseline test (Trial 1), each subject, in a double-blinded fashion, orally ingested 30 grams (a loading dose) of either creatine monohydrate (independent variable) or a placebo daily for 7 days (Trial 2) and 10 grams (a maintenance dose), of either creatine monohydrate or a placebo daily for 21 days (Trials 3, 4, and 5). Body mass was measured using a physician scale while percent body fat and lean body mass were determined by hydrostatic weighing, skinfolds, and bioelectrical impedance analysis.

Limitations

For this particular study, limitations include (1) restricting data collection to male college baseball players; (2) predicting residual volume from vital capacity; (3) no control over the subject's class and weight training schedule; and (4) measurement of body composition limited to hydrostatic weighing, skinfolds, and bioelectrical impedance analysis.

Definition of Terms

1. Body Mass. Total body weight measured on a physician scale in air wearing shorts only.

2. Percent Body Fat. Percentage of body composed of essential and storage fat as determined by hydrostatic weighing, skinfolds, and bioelectrical impedance analysis.

4. Lean Body Mass (LBM). Total body mass minus body fat as determined by hydrostatic weighing, skinfolds, bioelectrical impedance analysis.

CHAPTER 2

Review of Literature

The purpose of this chapter is to review the limited literature regarding oral creatine monohydrate supplementation and the key literature regarding body composition determination by hydrostatic weighing, skinfolds, and bioelectrical impedance analysis.

Creatine Literature Review

Most of the creatine of the body (over 95 percent) is found in skeletal muscle and approximately a third exists as free creatine (Balsom et al., 1994). Balsom and associates (1994) summarize the synthesis of creatine beginning extramuscularly in the kidney. While in the kidney, two amino acids, arginine and glycine, undergo transamidination (transfer of the amidine group) to form guanidinoacetic acid and ornithine. In the liver, transmethylation (addition of a methyl group) of guanidinoacetic acid by S-adenosylmethionine forms creatine. The creatine is then transported to the muscle via the bloodstream.

The total creatine pool is a combination of free creatine and phosphocreatine (Balsom et al., 1994). In the muscle, creatine and phosphate can be joined to form

creatine phosphate (also called phosphocreatine) depending on the amount of energy available (McArdle et al., 1991). Creatine phosphate may replenish adenosine triphosphate (ATP) by adding a phosphate bond to adenosine diphosphate (ADP). Muscle cells have 3 to 5 times as much creatine phosphate as ATP (McArdle et al., 1991). Muscle contraction and relaxation occurs from dephosphorylation of ATP (Greenhaff et al., 1993). Greenhaff and associates (1993) emphasize that skeletal muscle ATP is utilized very rapidly and for muscular work to continue ATP must be resynthesized from its breakdown products. All the energy stored as muscle creatine phosphate is readily available for ATP replenishment needed for muscular contraction (Guyton, 1991), which combined with ATP make the phosphagen energy system. This energy system is responsible for all physical activities lasting approximately 10 seconds (Wardlaw and Insel, 1993).

The turnover rate of creatine is around 2 grams per day for a 70 kg person with a total creatine pool of 120 grams (Balsom et al., 1994). Creatine may be found in the diet in foods such as red meat and fish. Salmon and beef contain 4.5 grams of creatine per kilogram (Balsom et al., 1994). This amount of meat contains excessive amounts of saturated fat and cholesterol; therefore, athletes might rely on creatine supplementation as a more efficacious means to increase creatine intake. Creatine supplementation in humans is

possible by oral ingestion of creatine monohydrate (Balsom et al., 1994). Creatine monohydrate is a white powdered substance soluble in warm water (Balsom et al., 1994). In activities where performance is influenced by the availability of creatine phosphate, creatine supplementation may be useful as an ergogenic aid (Balsom et al., 1994). For example, athletes performing sprint activities in running, swimming, and cycling may benefit from creatine supplementation. There are no apparent side effects associated with short term high dose creatine supplementation (Harris et al., 1992A). However, it is not yet known whether there are any adverse effects caused by long term high dose creatine supplementation (Balsom et al., 1994). Currently, creatine is not listed as a banned substance by the International Olympic Committee (Balsom et al., 1994).

Resting and Strenuous Exercise Muscle Creatine Values

In a study by Harris and colleagues (1992A), resting and strenuous aerobic exercise levels of the total creatine pool (free creatine and creatine phosphate) of the vastus lateralis muscle were shown to significantly increase with creatine supplementation. This was greatest in subjects with low initial creatine content. In this study, 2 subjects received 20 grams of creatine monohydrate per day for 4.5 days; 2 different subjects received 20 grams of creatine monohydrate per day for 7 days; 1 subject received 20 grams

of creatine monohydrate per day for 10 days; 3 subjects received 30 grams per day of creatine monohydrate for 7 days; and 4 subjects received 30 grams of creatine monohydrate on alternating days for 21 days. For the resting values, mean total creatine increased from 126.8 ± 11.7 mmol/kg to 148.6 ± 5.0 mmol/kg with creatine supplementation. The increases in resting total creatine appeared to be less dependent on the duration of supplementation, for example 2 days of creatine supplementation was as affective as 5 days of supplementation in increasing resting total creatine levels. To study the interaction of creatine uptake and exercise, five subjects performed 1 hour of bicycle ergometer exercise each day using one leg. The opposite leg was rested and served as a control. The subjects were administered creatine monohydrate in the following manner: 1 subject received 20 grams per day for 3.5 days, 3 subjects received 30 grams per day for 4 days, and 1 subject ingested 30 grams per day for 7 days. Mean total creatine content increased in the control leg from 118.1 ± 3.0 mmol/kg to 148.5 ± 5.2 mmol/kg and in the exercised leg to 162.2 ± 12.5 mmol/kg. Creatine uptake appeared to be greatest during the first 2 days of supplementation.

Odland and associates (1994) found no effect on resting muscle phosphocreatine levels after ingesting 20 grams of creatine monohydrate per day for 3 days. There were no

significant differences in concentration of ATP, phosphocreatine, or total creatine levels. However, Odland and associates (1994) did see a significant difference in free creatine levels and total creatine to ATP ratio.

In a double-blind study by Lemon and colleagues (1995), 7 males consumed 20 grams of creatine monohydrate per day for 5 days. Muscle creatine levels were measured using P magnetic resonance spectroscopy during 20-30 second maximal ankle extensions (105 degrees). Subjects significantly increased serum creatine at 1 hour post ingestion and increased pre-exercise muscle creatine/ATP ratio by 8 percent.

Effects on Performance

Greenhaff and colleagues (1993) reported creatine supplementation significantly enhanced the ability to produce muscle torque during 5 bouts of 30 maximal voluntary isokinetic contractions interspersed with 60 second recovery periods. Twelve subjects performed maximal voluntary unilateral knee extensions at an angular velocity of 180 degrees per second on a Cybex II isokinetic dynamometer. Each contraction was initiated from a 90 degree knee flexion position. In a double-blind placebo fashion, 6 subjects consumed 20 grams of creatine monohydrate per day for 5 days. On the morning after the final supplementation day subjects performed maximal voluntary knee extensions. The 6 subjects supplementing with creatine were able to sustain

peak isokinetic torque production at a higher level during repeated bouts of maximal voluntary contractions. The investigators speculated that the improvement in exercise performance after creatine supplementation was due to an increased rate of phosphocreatine resynthesis during exercise and/or recovery.

Balsom and associates (1993A) investigated whether creatine supplementation could delay the onset of fatigue during repeated bouts of short duration, high-intensity exercise. In this study, 16 subjects were placed in either a placebo or creatine group and participated in ten 6 second bouts of high-intensity cycling at 130 revolutions per minute and 140 revolutions per minute interspersed with a 30 second recovery period. In a double-blind fashion, 8 subjects received 30 grams of creatine monohydrate per day for 6 days. The main finding was that the creatine group cycling at 140 revolutions per minute could significantly better sustain performance towards the end (last 2 seconds) of each 6-second bout of exercise following supplementation.

Birch and colleagues (1994) investigated the effect of creatine supplementation on performance of repeated bouts of maximal cycling exercises of a longer duration than 6 seconds and to provide a longer duration of recovery. In a double-blind manner, 7 subjects consumed 20 grams of creatine monohydrate per day for 5 days, and 7 other subjects received corresponding glucose dosages. Before

supplementation and the morning following supplementation each subject performed 3 bouts of maximal exercise, each lasting 30 seconds. Each bout was performed at a velocity of 80 revolutions per minute on a cycle ergometer and was separated by 4 minutes of rest. Creatine supplementation significantly increased peak power output in the first exercise bout and mean power output and total work output in the first and second exercise bouts. Creatine supplementation had no effect on any of the parameters in the third exercise bout.

Oral administration of creatine monohydrate in 10 trained middle distance runners indicated a significant reduction in running time (Harris et al., 1992B). In this study 5 subjects consumed 6 x (5 grams of creatine monohydrate + 5 grams of glucose per day for 5 days) and 5 different subjects consumed 6 x 10 grams of glucose per day for 5 days. Prior to and following creatine supplementation, on separate days, subjects performed 4 x 300 meter and 4 x 1000 meter runs with 3 and 4 minute rest intervals. Subjects were encouraged to run at a velocity of 90 to 95 percent of their best. The findings reveal an improvement in running time over the final 300 meter and 1000 meter runs as well as a reduction in running time during the 4 x 1000 meter run.

Earnest and colleagues (1994) studied the effect of oral creatine monohydrate on peak anaerobic power and anaerobic capacity via a 30 second Wingate test. Anaerobic

power was defined as the highest power output in a 5 second period, whereas, anaerobic capacity was defined as total work in a 30 second period. Subjects (n=8 males) were matched in groups (creatine and placebo) according to mean anaerobic capacity. Subjects performed 3 successive Wingate tests interspersed with 5 minutes of rest before and after supplementation. In a double-blind manner, the creatine group ingested 20 grams per day of creatine monohydrate for 14 days or a corresponding placebo (glucose). The results revealed that anaerobic power was not affected in either group. However, post supplementation anaerobic capacity levels were greater for all trials in the creatine monohydrate group.

An investigation by Odland and associates (1994) found no effect on performance during a single short-term maximal cycling task after ingesting 20 grams of creatine monohydrate per day for 3 days. Tests were performed on 9 males that underwent 3 randomly ordered trials (creatine supplementation, placebo, and control). The trials were performed 14 days apart on a Monark cycle ergometer. Pre-exercise muscle biopsies were taken from the vastus lateralis. Subjects cycled maximally for 30 seconds (Wingate protocol). There were no significant differences found between trials for peak power, mean 10 second power, mean 30 second power, percent fatigue, or post-exercise blood lactate concentration.

Redondo and colleagues (1995) found no effect of oral creatine monohydrate supplementation on running velocity. Twenty subjects completed a pre-supplement and post-supplement session 1 week apart wherein they performed 3 trials of 60 meter sprints. Subjects in the treatment group ingested 25 grams of creatine monohydrate per day for 7 days, while the placebo group consumed corresponding dosages of glucose. Velocities of the subjects were videotaped through 3 zones (20 to 30 meter, 40 to 50 meter, and 50 to 60 meter) within the 60 meter sprint and calculated using the Peak 5 system. Results indicated that there were no significant main or interaction effects on velocity between groups.

Burke and associates (1995) studied the effect of creatine monohydrate supplementation on sprint performance in elite swimmers. Swimmers (n=32) were divided into 2 groups matched for sex, stroke/event, and sprint time over a distance of 50 meters. Groups were randomly assigned to 5 days of creatine monohydrate supplementation (20 grams per day) or a placebo (glucose). Tests performed were 25 meter, 50 meter, and 100 meter maximal effort sprints, each with 10 minute recovery periods. Subjects also performed a 10 second maximal cycle ergometery test. Results revealed no significant differences between the group means for sprint times or 10 second maximal cycle ergometry power and work.

Earnest and colleagues (1995A) investigated the effects of creatine monohydrate supplementation on muscular strength and endurance. In this study, 4 weight trained men received 20 grams of creatine monohydrate per day for 28 days and 4 different subjects received a glucose placebo of the same dosage. Prior to and following supplementation each subject was evaluated for 1 RM for the bench press, the number of repetitions at 70 percent of their pre-test 1 RM, and total lifting volume which was defined as the number of complete lifting repetitions times 70 percent of 1 RM. Compared to the placebo group, the creatine monohydrate group showed significant increases after supplementation in 1RM (+8.2 kg), total lifting volume (+441.3 kg), and repetitions at 70% of 1RM (+1.7 kg).

Grindstaff and associates (1995) investigated the effect of a supplement containing creatine monohydrate on isokinetic performance. In this study, 18 subjects were given either 190 grams per day for 7 days of maltodextrin (placebo) or a mixture containing 64 grams per day of carbohydrate, 67 grams per day of protein, 5 grams per day of fat, and 20 grams per day of creatine monohydrate, yeast derived RNA, and taurine. Subjects completed a pre and post supplementation trial. Each trial consisted of 2 tests. During the first test, subjects performed 5 sets of 15 maximal effort repetitions on the Arial computerized bench press with an ascending bar velocity of 0.25 meters per

second with 60 second rest intervals. Subjects rested for 5 minutes then performed 5 sets of 15 maximal effort repetitions on the upright squat machine with an ascending bar velocity of 0.41 meters per second with 60 second rest intervals. Results indicated that bench press absolute peak power significantly increased in the treatment group (11 ± 4 joules) versus the placebo group (-3 ± 4 joules). Increase in total work was greater but not significantly so in the treatment group (183 ± 52 joules) compared to the placebo group (5 ± 69 joules). Relative peak power values were greater in the treatment group. There were no significant differences observed in absolute or relative bench press peak force, average force, and average power values or in upright squat peak force, average force, total work, peak power, or average power values. These results indicate that 7 days of a supplement containing 20 grams per day of creatine monohydrate has limited effects on muscle endurance during intense, slow velocity, concentric bench press and squat performance with minimal rest intervals.

As a continuation of the previous study (Grindstaff et al., 1995), Almada and colleagues (1995) investigated the effects of a supplement containing creatine monohydrate on multijoint isokinetic performance for 28 days. Results revealed significant increases in absolute and relative bench press and upright squat peak force, total work, and peak power in both the placebo and treatment groups during

the training period. Average power was not significantly affected in either group. There were no significant group or interaction differences between groups. These results indicate that 28 days of a supplement mixture containing 20 grams per day of creatine monohydrate does not affect muscular endurance during intense, slow velocity, concentric bench press and squat performance with minimal rest intervals.

Hall and colleagues (1995) hypothesized that creatine supplementation would increase total work, but would not affect critical power. In this double-blind study, 7 females and 4 males completed 4 bouts of exercise on a cycle ergometer at work rates determine to elicit fatigue in 1 to 10 minutes. After ingesting 20 grams of creatine monohydrate per day for 5 days or corresponding placebo, subjects performed 4 bouts of exercise at the same previous work rates. Results indicated that oral creatine monohydrate increases work without affecting critical power.

Cooke and associates (1995) studied the effect of oral creatine supplementation on exercise performance during high-intensity short duration bicycle sprinting. Subjects (n=6) received 20 grams per day for 5 days of creatine monohydrate and performed 2 maximal sprints against a constant load for 15 seconds on a cycle ergometer. The 2 trials were separated by 20 minutes of rest. Measurements were taken before and the morning after supplementation.

Mean values for peak power, time to peak power, total work, and fatigue index were measured. This study found no significant differences within or between groups before or after creatine supplementation with regards to peak power, time to peak power, total work, and fatigue index.

Earnest and associates (1995B) investigated the effects of creatine monohydrate ingestion on intermediate length anaerobic (80 to 110 seconds) treadmill running to exhaustion. Six male subject ingested 20 grams of creatine monohydrate per day for 4 days and 10 grams of creatine monohydrate per day for 6 days. Five other subjects ingested corresponding dosages of a glucose placebo. All of the subjects practiced exhaustive treadmill running at a standard speed of 8 miles per hour at 10, 15, and 20 percent gradients for 2 weeks prior to testing. After this practice session, the subjects performed an exhaustive treadmill run at each percent grade. This was used to determine an appropriate individual grade that would elicit exhaustion within 90 seconds. Subjects then performed 2 pre-test runs to exhaustion, separated by an 8 minute recovery. Results indicated no significant differences between the creatine and placebo group. Also, there were no significant differences in run times and blood lactate levels in the creatine and placebo group. These researchers concluded that creatine supplementation may not improve intermediate length anaerobic work tasks and that its efficacy may be most

prevalent during shorter anaerobic work efforts.

However, Jacobs and associates (1995) conclude that there is an ergogenic effect on anaerobic exercise capacity as determined by maximal accumulated oxygen deficit after ingestion of creatine monohydrate. In a double-blind study, subjects ingested 20 grams of creatine monohydrate for 5 days or a corresponding placebo. Before treatment, maximal oxygen output was determined using a cycle ergometer. Before supplementation and immediately following 5 days of supplementation subjects exercised to exhaustion at 125 percent of maximal oxygen consumption. The maximal accumulated oxygen deficit was calculated as the difference between the oxygen demand of work and the cumulative maximal oxygen uptake measured during exercise. Time to exhaustion significantly increased from 131 $\pm$ 22 seconds to 143 $\pm$ 21 seconds in the creatine group. The maximal accumulated oxygen deficit significantly increased from 4.05 $\pm$ 1.33 liters to 4.36 $\pm$ 1.42 liters. Interestingly, this significant increase in maximal accumulated oxygen deficit in the creatine group remained significantly elevated for another 7 days (4.26 $\pm$ 1.39 liters) after the last day of supplementation.

The effects of creatine monohydrate supplementation on endurance type exercises have also been reported (Balsom et al., 1993B). In a double-blind fashion 9 well-trained male subjects received 20 grams of creatine monohydrate per day

for 6 days and 9 different subjects received corresponding dosages of glucose. After completing a maximal oxygen test, subjects returned on 2 consecutive days to perform a treadmill run to exhaustion at 120 percent of maximal oxygen consumption and a 6 kilometer terrain run on a forest track. This study revealed no significant improvement in the treadmill run or in the time taken to complete a 6 km undulating terrain run. In fact, run time was significantly greater following creatine supplementation in the creatine group. Balsom and associates speculate that the decrease in performance may be associated with an increase in body mass. There were no changes seen in the placebo group. These results fail to show any improvement in endurance type performance following creatine supplementation suggesting, with theoretical reasoning, effects of creatine supplementation are limited to short duration high-intensity exercise (Balsom et al., 1993B).

Effects on Body Mass

Creatine monohydrate supplementation has been accompanied by increases in body weight (Balsom et al., 1993A; Balsom et al., 1993B; and Earnest et al., 1995A). Balsom and colleagues (1993A) reported a significant increase in body mass of 1.1 kg (range 0.3 to 2.5 kg) after participants consumed 20 grams of creatine monohydrate per day for 6 days. The largest increase (2.5 kg) was found in a vegetarian. Balsom and associates (1993A and 1993B) suggest

that the increase in body mass may be two-fold; possible increase in protein synthesis and an increase in total body water content.

In agreement with the previous study (Baslom et al., 1993A), body mass increased significantly from 73.5 ± 2.3 to 74.4 ± 2.3 kilograms after consuming 20 grams of creatine monohydrate for 6 days (Balsom et al., 1993B). This increase in body mass may be a likely explanation for impairment in endurance type exercise noted above (Balsom et al., 1993B). Balsom and associates (1994) note any isolated short term benefits of creatine supplementation in sports such as football, basketball, ice hockey, and tennis where game time often exceeds 1 hour, may be counteracted by an increase in body mass. Earnest and colleagues (1995A) report a significant 1.7 kg increase in body weight after creatine monohydrate supplementation (20 grams per day for 28 days) and no significant changes in percentage of fat mass via hydrostatic weighing in either creatine or placebo group. Lemon and associates (1995) report favorable findings of significant increases in body mass (+1.3 kg) after consuming 20 grams of creatine monohydrate per day for 5 days.

Body Composition Literature Review

Measurements of body composition are becoming popular among fitness professionals and provide valuable information for athletes interested in competitive preparation and enhanced performance (Jackson, 1984; Jackson and Pollock,

1985; Thorland et al., 1984). Body composition is a term referring to the components of the human body (Adams, 1994). The American College of Sports Medicine Resource Manual (1993) defines body composition as relative amounts of muscle, bone, and fat in the body, often taken as the relative amounts of fat and LBM.

Measuring body density allows a more accurate method of determining fat weight and LBM rather than using height/weight tables (American College of Sports Medicine, 1993). There are many ways of determining body composition. However, many methods are limited because of lack of sufficient research to determine reliability and validity, considerable time, equipment and personnel expense, and complexity of methods (American College of Sports Medicine, 1993; Wilmore and Behnke, 1969). Hydrostatic weighing and skinfolds are the most common methods for determining body density (American College of Sports Medicine, 1993). However, all methods involve assumptions and several types of errors (Dugdale and Griffiths, 1979; Katch, 1984).

Hydrostatic Weighing

Hydrostatic weighing is based on the application of Archimedes's Principle. This principle states that an object submerged or floating in water is buoyed up by a counterforce that equals the weight of the water displaced. This buoyant force supports the submerged body against the downward pull of gravity (McArdle et al., 1991). Bone and

muscle tissue are more dense than water. Therefore, a person with more lean body mass will weigh more in water and have a higher body density. Whereas, fat tissue is less dense than water, thus, a person will weigh less under water and have a higher percentage of body fat (American College of Sports Medicine, 1991).

Hydrostatic weighing is considered to be the "gold standard", but body density measured by hydrostatic weighing contains some sources of error: measurement of residual volume, air in the intestines, and assumptions of fat and muscle densities (American College of Sports Medicine, 1993; Jackson, 1984; Pollock and Jackson, 1984). Technique has an important influence on body density scores measured by means of hydrostatic weighing (Katch and Katch, 1980). McArdle and colleagues (1991) suggest subjects repeat hydrostatic weighing 8 to 12 times. This many trials are needed because the subjects undergo a "learning effect" to expel more air from their lungs to achieve a "true" underwater score (McArdle et al., 1991). Katch and Katch (1980) recommend averaging the last few trials to provide a reliable score for subjects, especially when residual volume is measured separately from hydrostatic weighing. Reported in the American College of Sports Medicine Resource Manual (1993) the standard error of estimate of body fat is $\pm$ 2.5% of the "true" value.

McArdle and colleagues (1991) define residual volume as the amount of air remaining in the lungs after a deep exhalation. Residual volume averages between 1.2 and 1.4 liters for men and tends to increase with age (McArdle et al., 1991). The volume of gas in the gastrointestinal tract is approximately 100 millimeters (Wilmore, 1969).

Wilmore (1969) remarked that the determination of residual volume is time consuming. Therefore, he investigated the potential error in using a predicted value of residual volume as opposed to actual measurement of residual volume when determining body density. A fraction of the vital capacity was used to predict residual volume. Vital capacity was multiplied by 0.24 for males to estimate residual volume. Interestingly, there were no significant differences between the means for density, percent fat, or LBM using actual residual volume and the means from predicting residual volume. However, Wilmore (1969) explains there was a large difference in values of the subjects to question the use of predicting residual volume. Expiring underwater to the point of residual volume is sometimes an impossible technique (Weltman and Katch, 1981). Therefore, for research purposes, accurate measurement of residual volume is essential for observing changes in body composition (Wilmore, 1969).

Skinfolds

The use of calipers to measure subcutaneous skinfolds has become increasingly popular in the fitness industry (Adams, 1994). Skinfolds predict body density more accurately than height/weight or circumferences (Jackson and Pollock, 1985). Skinfold measurements rely on the observation that approximately one half of the total body fat is subcutaneous fat depending on age, sex, and measurement technique used (American College of Sports Medicine, 1993; McArdle, 1991).

A linear regression equation provides accurate predictions for those with middle values and a larger error of predictions at the extremes (Jackson and Pollock, 1985; Adams, 1994). For example, linear equations underestimate body density for lean individuals and overestimates it in obese individuals. Therefore, Jackson and Pollock (1985) developed nonlinear or quadratic equations which will provide consistent measurements of body density throughout body fat ranges.

Jackson and Pollock (1985) discuss population-specific and generalized equations. Population-specific equations are based on homogenous samples and applications are limited to that subsample. However, generalized equations are based on heterogenous groups using nonlinear regression models. The primary advantage of using generalized equations is the validity over a wide range of subjects without losing

prediction accuracy by accounting for fatness and age differences and the nonlinear relationship between body density and subcutaneous fat (Jackson and Pollock, 1985; Pollock and Jackson, 1984). Generalized equations use the sum of three or seven skinfolds with the age of the subject. Jackson (1984) reports the sum of skinfolds provides a reliable estimate of total body fat. Recommended by Jackson and Pollock (1985) is the use of the sum of three skinfolds which is highly correlated ($r = 0.97$) with the sum of seven skinfolds.

To increase validity, the technician should use the exact skinfold site and technique to derive the equations (American College of Sports Medicine, 1993). Skinfold sites should be measured in succession and then the cycle repeated two to three times, using the average of the scores at each site as the final score (Katch and Katch, 1980). This technique will help reduce tester bias. According to the American College of Sports Medicine Resource Manual (1993), skinfold measurement has an error of approximately $\pm$ 3.7%.

Bioelectrical Impedance Analysis

Bioelectrical impedance analysis is a relatively new method for measuring body composition and has become increasingly popular (Eckerson et al., 1992; Jackson et al., 1988). Bioelectrical impedance analysis is based on the principle that fat is a very poor electrical conductor while lean body tissues offer a very low resistance to electrical

current. The low level electrical signal operating at 50 kHz penetrates the deep tissues, providing a baseline resistance measurement that directly correlates to the volume and density of the biological systems. The higher the body water content (found in lean body tissue) the lower the impedance. This impedance is converted to lean body mass (American College of Sports Medicine, 1993; McArdle et al., 1991; Valhalla Scientific, Inc., 1988).

According to the American College of Sports Medicine (1993) Resource Manual, bioelectrical impedance has been found to be reliable, but not valid. Compared to hydrostatic weighing, bioelectrical impedance was shown to underestimate low body fat in the obese and overestimate body fat in the lean (Kaminsky and Whaley, 1993; American College of Sports Medicine, 1993). Factors such as hydration level, alcohol intake, medications, skin resistance, and previous exercise may alter the readings of bioelectrical impedance analysis (American College of Sports Medicine, 1993; McArdle et al., 1991).

Jackson and associates (1988) investigated the reliability and validity of determining body composition by means of bioelectrical impedance analysis. Results indicated that bioelectrical impedance was reliable ($r = 0.957$) with a standard error of estimate of 1.4% fat. However, cross-validation correlation ($r = 0.71$) for bioelectrical impedance determinations of percent fat appeared to be

significantly lower than seven site skinfolds equation (r = 0.92). In this study, the standard error for estimating percent fat from bioelectrical impedance analysis was 5 to 6 percent.

Summary

This literature review presented current research investigating the effects of oral creatine monohydrate supplementation on performance and body mass. In this review, creatine monohydrate supplementation was suggested to increase muscle content of creatine and enhance performance (Balsom et al., 1993A; Birch et al., 1994; Earnest et al., 1994; Greenhaff et al., 1993; Harris et al., 1992; and Harris et al., 1993); increase muscular strength (Earnest et al., 1995A); and increase body mass (Balsom et al., 1993A; and Balsom et al., 1993B; and Earnest et al., 1995A). Also, the literature review presented some key research findings on body composition determination by hydrostatic weighing, skinfolds, and bioelectrical impedance analysis.

CHAPTER 3

Methodology

This chapter characterizes subjects, treatment, instrumentation, and procedures used to investigate the effects of creatine monohydrate supplementation on body mass and composition, for example body fat and lean body mass (LBM).

Subjects

Sixteen male varsity college baseball players gave their written consent to participate in a 5 week study to assess the effects of oral creatine monohydrate supplementation on body mass, percent body fat, and LBM. The mean (range) age and height were 19.69 (18 to 21) years and 179.6 (166.4 to 196.9) centimeters, respectively. Subjects were matched by LBM after a control trial and assigned to a placebo (n=8) or a creatine monohydrate (n=8) group. Under the supervision of the university strength coach, subjects followed a high-intensity strength-training program designed to build overall strength. Appendix A details this high-intensity strength training program. The study was performed as a double-blind, placebo between-subjects design. Body mass and composition were measured following each week of

treatment.

Creatine Monohydrate or Placebo Administration

After a baseline trial (Trial 1), subjects in the creatine monohydrate group (CM) ingested in successive order, approximately 5 grams (1 heaping teaspoon) of creatine monohydrate 6 times a day for 7 days (Trial 2), approximately 5 grams of creatine monohydrate 2 times a day for 14 days (Trials 3 and 4), followed by approximately 5 grams of glucose 2 times a day for 7 days (Trial 5). The placebo group (P) ingested corresponding dosages of glucose. The subjects were instructed to consume each dose at least 2 hours apart and instructed on the proper dose. A weekly written compliance sheet was collected at the beginning of each trial to check that each subject conformed to these procedures.

Instrumentation

Body mass and height were measured using a physician scale and stadiometer. Percent body fat and LBM were determined using the following methods: hydrostatic weighing, skinfolds, and bioelectrical impedance analysis.

Body Mass and Height

Body mass was measured to the nearest 0.1 kg using a Continental 159.0 kg capacity scale. The subjects were weighed in tight shorts worn for hydrostatic weighing. Each subject was instructed to wear the same shorts for each trial. The scale was set to zero and calibrated with a 4.0

kg known weight before each use. Height was measured without shoes to the nearest 0.5 cm using a 198.0 cm stadiometer.

Hydrostatic Weighing

A Chatillon 15.0 kg capacity scale was used for the measurement of underwater weight. The scale was calibrated prior to testing each subject by hanging a 4.0 kg known weight. A basic pool thermometer from Lifegard was used to measure water temperature. Water temperature was maintained between 32-40 degrees celsius. Tare weight consisted of the chair, linkage, swim suit, and weight belt if needed. Tare weight was calculated prior to testing each subject. During Trial 1, a Sensor Medics Spirometer connected to an IBM 433 DX/D computer was used to measure vital capacity of each subject. Subjects replicated the position during underwater weighing and performed 3 vital capacity trials. The best of the 3 trials was used to predict residual volume. Residual volume (RV) was predicted by the following formula:

RV (ml) = 0.24 X vital capacity (ml) (Wilmore, 1969)

The predicted residual volume during Trial 1 was used to calculate percent body fat and LBM via hydrostatic weighing for Trials 2, 3, 4, and 5. Body density (Db) and body fat percentage (%BF) were calculated from the following formulas:

$Db = W_{air} / (W_{air} - W_{water} / Dw) - RV + 100$ (American College of Sports Medicine, 1991)

%BF = (495 / Db) - 450 (Siri, 1961)

Where: W_{air} = body weight in air measured in grams (g)

W_{water} = net weight in water (body weight in water minus tare weight) measured in grams (g)

Dw = density of water at a given temperature

RV = subject's residual volume measured in milliliters (ml)

100 = Estimated Intestinal Volume (Wilmore, 1969)

Each subject performed 10 trials of hydrostatic weighing to obtain three similar readings to the nearest 20 grams. The average of the 3 scores was used as the "true" value.

Skinfolds

Skinfolds were measured using a Lange caliper (10 g · mm^{-2} constant pressure). All sites were measured on the right side of the body to the nearest 0.5 mm. Jackson and Pollock's (1978) three-site skinfold measurement was performed to determine body density. The technician rotated through all sites before taking a second and third measurement. The average of the 3 measurements was used for each site.

Skinfold points were marked with a tape measure and marker. The chest skinfold was measured on a diagonal fold midway between the anterior axillary line and the nipple. The abdomen skinfold was taken as a vertical fold measured 2 cm from the umbilicus. The thigh skinfold was a vertical fold measured on the anterior midline of the thigh halfway between the inguinal crest and proximal border of the

patella (Jackson and Pollock, 1978). Body density (Db) was determined by the following formula:

$$Db = 1.1093800 - 0.0008267\ (X1) + 0.0000016\ (X1)^2 - 0.0002574\ (X2)$$

Where: X1 = sum of chest, abdomen, and thigh (mm)

X2 = age (years)

Percent body fat was calculated using the Siri (1961) formula.

$$\%BF = (495 / Db) - 450$$

Bioelectrical Impedance Analysis

Bioelectrical impedance analysis was measured using a Valhalla Scientific 1990B bio-resistance body composition analyzer. The machine was calibrated prior to testing each subject. Since bioelectrical impedance analysis has been found to be reliable (American College of Sports Medicine, 1993), 1 trial was performed on each subject.

With the subject in a supine position, on a non-conductive surface, the right foot and hand were exposed. The subject remained motionless with arms bent at the elbow and palms down. The first electrode was placed on the line bisecting the styloid processes of the ulna and radius. The second electrode was placed on the index finger distal metacarpal located 4-5 cm away from the first electrode. A third electrode was placed on the line bisecting the medial and lateral malleoli. Lastly, a fourth electrode was placed on the first digit metatarsal located 4-5 cm away from the

third electrode (Valhalla Scientific, Inc., 1988).

Procedures

Before the initial visit to the laboratory, all subjects were familiarized with specific guidelines of the protocol. This consisted of proper clothing, compliance forms, and being instructed not to eat a large meal, participate in heavy exercise, consume caffeine, and/or ingest large amounts of fluids within 6 hours upon arrival to the Human Performance Laboratory. All subjects signed-up to be tested at the same time and day for each trial.

Subjects initially visited the laboratory to become familiar with the type of tests to be performed in the study (Trial 1). During this visit, initial height was measured as well as the subject's vital capacity to predict residual volume. The subject's body mass was measured and percent body fat and LBM were estimated by hydrostatic weighing, skinfolds, and bioelectrical impedance analysis.

One week prior to Trial 2, subjects picked-up their appropriate treatment for the week along with a compliance form and a dietary log. No testing was performed on the subjects during this visit. During Trials 2, 3, 4, and 5, the subject's body mass and body composition were measured. Compliance forms and treatments were issued to all subjects for each week. Subjects were debriefed on the last visit (Trial 5).

Statistical Analysis

A 2 X 5 factorial repeated measures ANOVA was performed for the statistical analysis. An alpha level of 0.05 was established for statistical significance. In addition, a follow-up test for simple main effects was performed when applicable.

CHAPTER 4

RESULTS

The purpose of this study was to investigate the effects of oral creatine monohydrate supplementation on body mass and body composition, using a two-component model of percent body fat and lean body mass (LBM) measured weekly over the course of a 5 week double-blind, placebo, between-subjects study. Following a baseline trial (Trial 1), eight subjects (CM) received creatine monohydrate supplements for Trials 2, 3, 4, and a placebo for Trial 5. Eight other subjects (P) received a placebo for Trials 2, 3, 4, and 5.

Body Mass

In the CM group, means and standard deviations of body mass in Trials 1, 2, 3, 4, and 5 were 82.35 $\pm$ 10.80, 84.40 $\pm$ 11.53, 84.70 $\pm$ 11.65, 84.53 $\pm$ 11.42, and 84.22 $\pm$ 11.44 kilograms respectively. In the P group, means and standard deviations of body mass in Trials 1, 2, 3, 4, and 5 were 81.23 $\pm$ 8.48, 82.04 $\pm$ 9.01, 81.58 $\pm$ 8.82, 82.16 $\pm$ 9.24, and 81.49 $\pm$ 9.35 kilograms respectively (Table 1). Table 2 illustrates no significant differences in body mass for groups ($F = 0.2096$, $p > 0.05$). However, there was a significant difference in trials ($F = 8.3517$, $p < 0.05$) and

a significant interaction ($F = 2.9792$, $p < 0.01$). This significant interaction effect necessitates a test for simple main effects, which revealed significant differences ($p < 0.01$) in the CM group between Trial 1 and Trials 2, 3, 4, and 5.

Table 1. Means and (Standard Deviations) for Body Mass in Kilograms

	Trial 1	Trial 2	Trial 3	Trial 4	Trial 5
CM Group					
Body Mass	82.35	*84.40	*84.70	*84.53	*84.22
	(10.80)	(11.53)	(11.65)	(11.42)	(11.44)
	Trial 1	Trial 2	Trial 3	Trial 4	Trial 5
P Group					
Body Mass	81.23	82.04	81.58	82.16	81.49
	(8.48)	(9.01)	(8.82)	(9.24)	(9.35)

* In the CM Group, a test for simple main effects revealed mean body mass in Trial 1 was significantly different than Trials 2,3,4, and 5 ($p < 0.01$). However, Trials 2,3,4, and 5 were not significantly different from each other.

However, Trials 2, 3, 4, and 5 were not significantly different from each other. In the P group, a test for simple main effects found no significant differences ($p > 0.05$) between the trials. In the CM group, body mass increased significantly from Trial 1 (82.35 ± 10.80 kg) to Trial 2

(84.40 ± 11.53 kg) and this significant increase was maintained through the supplement period relative to Trial 1. Interestingly, this significant increase of body mass in the CM group was maintained even after going off the treatment in Trial 5. Overall, body mass increased as much as 2.35 kg in the CM group, while in the P group body mass increased only 0.93 kg which was not significant, and the increase was not maintained through the trials. Individual measures of body mass are located in Table B-1 (p. 57).

Table 2. Summary of Repeated Measures ANOVA for Significance in Body Mass

Source	Degrees of Freedom	Mean Square Variance	F	P
Between Subj.	15			
Groups	1	109.3950	0.2096	0.6541
Error	14	522.0156		
Within Subj.	64			
Trials	4	6.3740	8.3517	0.0001
Interaction	4	2.2737	2.9792	0.0266
Error	56	0.7632		

Percent Body Fat Measured by Hydrostatic Weighing

In the CM group, means and standard deviations of percent body fat measured by hydrostatic weighing in Trials 1, 2, 3, 4, and 5 were 15.89 ± 7.83, 16.80 ± 7.22, 15.40 ± 8.19, 15.66 ± 6.61, and 16.21 ± 5.95 percent respectively. In the P group, means and standard deviations of percent body fat in Trials 1, 2, 3, 4, and 5 were 17.01 ± 9.53, 16.35 ± 8.47, 15.94 ± 8.00, 15.39 ± 6.72, and 15.10 ± 5.86 percent respectively (Table 3). Table 4 illustrates no significant group differences in percent body fat ($F = 0.001$, $p > 0.05$) as measured by hydrostatic weighing. There was no significant difference in trials ($F = 0.591$, $p > 0.05$) and no significant interaction ($F = 0.460$, $p > 0.05$). Individual body fat percentages measured by hydrostatic weighing for both groups are reported in Table B-2 (p. 58).

Table 3. Means and (Standard Deviations) of Percent Body Fat Measured by Hydrostatic Weighing, Skinfolds, and Bioelectrical Impedance Analysis (BIA)

Method	Trial 1	Trial 2	Trial 3	Trial 4	Trial 5
CM Group					
Hydro	15.89	16.80	15.40	15.66	16.21
	(7.83)	(7.22)	(8.19)	(6.61)	(5.95)
Skinfold	11.96	11.00	10.98	10.40	11.90
	(4.81)	(4.20)	(4.52)	(3.63)	(4.15)
BIA	14.13	12.65	13.68	15.01	12.69
	(3.17)	(2.52)	(3.05)	(4.98)	(3.51)
	Trial 1	Trial 2	Trial 3	Trial 4	Trial 5
P Group					
Hydro	17.01	16.35	15.94	15.39	15.10
	(9.53)	(8.47)	(8.00)	(6.72)	(5.86)
Skinfold	12.66	12.68	12.40	11.98	11.85
	(4.19)	(4.11)	(4.09)	(5.27)	(4.09)
BIA	11.70	12.19	11.31	12.64	13.35
	(3.06)	(2.72)	(2.79)	(2.60)	(2.86)

Table 4. Summary of Repeated Measures ANOVA for Significance in Percent Body Fat via Hydrostatic Weighing

Source	Degrees of Freedom	Mean Square Variance	F	P
Between Subj.	15			
Groups	1	0.024	0.001	0.9923
Error	14	256.178		
Within Subj.	64			
Trials	4	3.934	0.591	0.6708
Interaction	4	3.064	0.460	0.7647
Error	56	6.660		

Percent Body Fat Measured by Skinfolds

In the CM group, means and standard deviations of percent body fat measured by skinfolds in Trials 1, 2, 3, 4, and 5 were 11.96 $\pm$ 4.81, 11.00 $\pm$ 4.20, 10.98 $\pm$ 4.52, 10.40 $\pm$ 3.63, and 11.90 $\pm$ 4.15 percent respectively. In the P group, means and standard deviations of percent body fat in Trials 1, 2, 3, 4, and 5 were 12.66 $\pm$ 4.19, 12.68 $\pm$ 4.11, 12.40 $\pm$ 4.09, 11.98 $\pm$ 5.27, and 11.85 $\pm$ 4.09 percent respectively (Table 3).

Table 5 illustrates no significant group differences in percent body fat ($F = 0.255$, $p > 0.05$) as measured by skinfolds. There was no significant difference in trials ($F = 2.166$, $p > 0.05$) and no significant interaction ($F = 1.766$, $p > 0.05$). Table B-3 (p. 59) illustrates individual

values measured by skinfolds for both groups.

Table 5. Summary of Repeated Measures ANOVA for Significance in Percent Body Fat via Skinfolds

Source	Degrees of Freedom	Mean Square Variance	F	P
Between Subj.	15			
Groups	1	22.685	0.255	0.6211
Error	14	88.804		
Within Subj.	64			
Trials	4	2.622	2.166	0.0847
Interaction	4	2.138	1.766	0.1486
Error	56	1.211		

Percent Body Fat Measured by Bioelectrical Impedance

In the CM group, means and standard deviations of percent body fat measured by bioelectrical impedance analysis in Trials 1, 2, 3, 4, and 5 were 14.13 $\pm$ 3.17, 12.65 $\pm$ 2.52, 13.68 $\pm$ 3.05, 15.01 $\pm$ 4.98, 12.69 $\pm$ 3.51 percent respectively. In the P group, means and standard deviations of percent body fat in Trials 1, 2, 3, 4, and 5 were 11.70 $\pm$ 3.06, 12.19 $\pm$ 2.72, 11.31 $\pm$ 2.79, 12.64 $\pm$ 2.60, 13.35 $\pm$ 2.86 percent respectively (Table 3).

Table 6 illustrates no significant group difference in percent body fat ($F = 1.158$, $p > 0.05$), no significant difference in trials ($F = 1.148$, $p > 0.05$), and no

significant interaction ($F = 1.848$, $p > 0.05$) as measured by bioelectrical impedance analysis. Individual measurements measured by bioelectrical impedance analysis for the CM group and P group are located in Table B-4 (p. 60).

Table 6. Summary of Repeated Measures ANOVA for Significance in Percent Body Fat via Bioelectrical Impedance Analysis

Source	Degrees of Freedom	Mean Square Variance	F	P
Between Subj.	15			
Groups	1	38.920	1.158	0.3001
Error	14	33.610		
Within Subj.	64			
Trials	4	5.021	1.148	0.3437
Interaction	4	8.085	1.848	0.1325
Error	56	4.375		

Lean Body Mass Measured by Hydrostatic Weighing

In the CM group, means and standard deviations of LBM measured by hydrostatic weighing in Trials 1, 2, 3, 4, and 5 were 68.82 $\pm$ 7.47, 69.82 $\pm$ 6.47, 71.13 $\pm$ 7.82, 70.90 $\pm$ 7.73, 70.24 $\pm$ 8.29 kilograms respectively. In the P group, means and standard deviations of LBM in Trials 1, 2, 3, 4, and 5 were 67.28 $\pm$ 9.40, 68.75 $\pm$ 10.52, 68.50 $\pm$ 9.55, 69.49 $\pm$ 9.35, and 69.06 $\pm$ 8.32 kilograms respectively (Table 7).

Table 7. Means and (Standard Deviations) for Lean Body Mass in Kilograms

Method	Trial 1	Trial 2	Trial 3	Trial 4	Trial 5
CM Group					
Hydro	68.82	69.82	71.13	70.90	70.24
	(7.47)	(6.47)	(7.82)	(7.73)	(8.29)
Skinfold	72.24	*74.92	*75.21	*75.58	*73.96
	(8.05)	(9.10)	(9.40)	(9.14)	(8.77)
BIA	70.52	73.56	73.01	71.61	71.67
	(7.90)	(8.93)	(9.50)	(8.90)	(6.00)
	Trial 1	Trial 2	Trial 3	Trial 4	Trial 5
P Group					
Hydro	67.28	68.75	68.50	69.49	69.06
	(9.40)	(10.52)	(9.55)	(9.35)	(8.32)
Skinfold	70.80	71.49	71.39	72.09	71.66
	(6.50)	(6.90)	(7.70)	(7.07)	(7.27)
BIA	71.55	71.88	72.20	71.66	70.48
	(5.87)	(6.62)	(6.80)	(7.11)	(7.26)

*In the CM Group, a test for simple main effects revealed mean LBM in week 1 was significantly different than weeks 2,3,4, and 5 ($p < 0.01$). However, weeks 2,3,4, and 5 were not significantly different from each other.

As noted in Table 8, for LBM measured by hydrostatic weighing there was no significant group difference ($F = 0.1411$, $p > 0.05$), no significant difference in trials ($F = 2.2056$, $p > 0.05$), and no significant interaction ($F = 0.3154$, $p > 0.05$). Overall, LBM increased as much as 2.31 kg in the CM group and 2.21 kg in the P group, but this increase was not statistically significant. Individual measures of LBM from hydrostatic weighing for the CM group and the P group are located in Table B-5 (p. 61).

Table 8. Summary of Repeated Measures ANOVA for Significance in Lean Body Mass via Hydrostatic Weighing

Source	Degrees of Freedom	Mean Square Variance	F	P
Between Subj.	15			
Groups	1	49.0002	0.1411	0.7129
Error	14	347.3505		
Within Subj.	64			
Trials	4	10.7675	2.2056	0.0800
Interaction	4	1.5399	0.3154	0.8665
Error	56	4.8818		

Lean Body Mass Measured by Skinfolds

In the CM group, means and standard deviations of LBM measured by skinfolds in Trials 1, 2, 3, 4, and 5 were 72.24 ± 8.05, 74.92 ± 9.10, 75.21 ± 9.40, 75.58 ± 9.14, and 73.96 ± 8.77 kilograms respectively. In the P group, means and standard deviations of LBM in Trials 1, 2, 3, 4, and 5 were 70.80 ± 6.50, 71.49 ± 6.90, 71.39 ± 7.70, 72.09 ± 7.07, and 71.66 ± 7.27 kilograms respectively (Table 7). There was a significant interaction ($F = 2.6628$, $p < 0.05$) in mean LBM in the CM group as measured by skinfolds (Table 9). A test for simple main effects showed a significant difference ($p < 0.01$) between Trial 1 and Trials 2, 3, 4, and 5. However, Trials 2, 3, 4, and 5 were not significantly different from each other. The P group reported no significant differences ($p > 0.05$) in LBM measured by skinfolds. In the CM group, LBM significantly increased from Trial 1 (72.24 ± 8.05 kg) to Trial 2 (74.92 ± 9.10 kg) and continued to significantly increase through the supplement period relative to Trial 1. Interestingly, there was a 1.62 kg decrease of LBM in the CM group after going off the treatment in Trial 5, but this decrease was not statistically significant. Overall, LBM significantly increased as much as 3.34 kg in the CM group. Individual LBM values for the CM group and P group are reported in Table B-6 (p. 62).

Table 9. Summary of Repeated Measures ANOVA for Significance in Lean Body Mass via Skinfolds

Source	Degrees of Freedom	Mean Square Variance	F	P
Between Subj.	15			
Groups	1	167.7363	0.5270	0.4798
Error	14	318.2660		
Within Subj.	64			
Trials	4	12.1539	8.1120	0.0001
Interaction	4	3.9895	2.6628	0.0418
Error	56	1.4983		

Lean Body Mass Measured by Bioelectrical Impedance

In the CM group, means and standard deviations of LBM measured by bioelectrical impedance analysis in Trials 1, 2, 3, 4, and 5 were 70.52 ± 7.90, 73.56 ± 8.93, 73.01 ± 9.50, 71.61 ± 8.90, and 71.67 ± 6.00 kilograms respectively. In the P group, means and standard deviations in Trials 1, 2, 3, 4, and 5 were 71.55 ± 5.87, 71.88 ± 6.62, 72.20 ± 6.80, 71.66 ± 7.11, and 70.48 ± 7.26 kilograms respectively (Table 7).

Referring to Table 10, determination of LBM by bioelectrical impedance analysis did not reveal significant group differences ($F = 0.0203$, $p > 0.05$). There was no significant difference in trials ($F = 1.8347$, $p > 0.05$) and no significant interaction ($F= 0.0811$, $p >0.05$). Individual

values of LBM determined by bioelectrical impedance analysis for both groups are located in Table B-7 (p. 63).

Table 10. Summary of Repeated Measures ANOVA for Significance in Lean Body Mass via Bioelectrical Impedance Analysis

Source	Degrees of Freedom	Mean Square Variance	F	P
Between Subj.	15			
Groups	1	5.3769	0.0203	0.8887
Error	14	264.6475		
Within Subj.	64			
Trials	4	10.5418	1.8347	0.1350
Interaction	4	4.6027	0.0811	0.5297
Error	56	5.7458		

Chapter 5

Discussion

A 2 X 5 factorial repeated measures ANOVA with a follow-up test for simple main effects ($p < 0.05$) of all data indicated creatine monohydrate supplementation did affect body mass. However, as evaluated by three different body composition measurement techniques, creatine monohydrate supplementation did not consistently affect percent body fat or lean body mass (LBM) in college baseball players. The following discussion presents a comparison of this research with other studies as to the effects of creatine monohydrate supplementation on body mass and body composition.

Body Mass Responses

It has been previously observed that 20 to 25 grams per day of creatine monohydrate supplementation for 5 to 28 days induces an increase in body mass of around 1.0 to 1.7 kg (Balsom et al., 1993A; Balsom et al., 1993B; Earnest et al. 1995A; and Lemon et al., 1995). This is in agreement with the findings of the current study, as significant increases ($p < 0.05$) in body mass from 82.35 $\pm$ 10.80 kg (Trial 1) to 84.40 $\pm$ 11.53 kg (Trial 2), 84.70 $\pm$ 11.65 kg (Trial 3),

84.53 ± 11.42 kg (Trial 4), and 84.22 ± 11.44 kg (Trial 5) were recorded in the creatine monohydrate (CM) group, suggesting creatine monohydrate supplementation does increase and maintain body mass relative to Trial 1. There was a significant interaction ($F = 2.9792$, $p = 0.0266$) which necessitated a test for simple main effects, which revealed significant differences ($p < 0.01$) in the CM group between Trial 1 and Trials 2, 3, 4, and 5. However, Trials 2, 3, 4, and 5 were not significantly different from each other. There were no significant differences ($p > 0.05$) found between the trials in the placebo group. Therefore, the hypothesis that there will be no effect of creatine monohydrate supplementation on body mass is rejected.

The most likely explanation of the increase in body mass following creatine monohydrate supplementation may be due to an increase in total body water content and water retention (Balsom et al., 1993A; and Balsom et al., 1994). The significant 2.35 kg increase in body mass found in the creatine monohydrate group is most likely water retention found intercellularly. There is a possibility that creatine monohydrate supplementation stimulates protein synthesis causing a structural change in skeletal muscle resulting in an increase in overall body mass and is currently under investigation (Balsom et al., 1993A; Balsom et al., 1993B; and Balsom et al., 1994).

Percent Body Fat Responses

With regard to percent body fat responses measured by hydrostatic weighing, skinfolds, and bioelectrical impedance analysis, results of this research appeared to show no significant effect of creatine monohydrate supplementation over a period of 5 trials. These data coincide with the findings of Earnest and associates (1995A) who noted no significant changes in percent body fat via hydrostatic weighing after ingesting 20 grams of creatine monohydrate per day for 28 days.

There were no significant differences in percent body fat for groups ($F = 0.001$, $p = 0.9923$), no significant differences in trials ($F = 0.591$, $p = 0.6708$), and no significant interaction ($F = 0.460$, $p = 0.7647$) as measured by hydrostatic weighing. Individual measures of percent body fat measured via hydrostatic weighing (Table B-2) appeared to show great variation between trials suggesting some source of error, possibly due to the subjects not exhaling completely.

There were no significant differences in percent body fat for gropus ($F = 0.255$, $p = 0.6211$), no significant differences in trials ($F = 2.166$, $p = 0.0847$), and no significant interaction ($F = 1.766$, $p = 0.1486$) as measured by skinfolds. Also, there were no significant group differences ($F = 1.158$, $p = 0.3001$), no significant difference in trials ($F = 1.148$, $p = 0.3437$), and no

significant interaction ($F = 1.848$, $p = 0.1325$) in percent body fat via bioelectrical impedance analysis. Therefore, the hypothesis that there will be no effect of oral creatine monohydrate supplementation on percent body fat is accepted.

Lean Body Mass Responses

Research is nonexistent with regard to the effect of oral creatine monohydrate supplementation on LBM. In the CM group, LBM did not significantly increase over the 5 trials using hydrostatic weighing and bioelectrical impedance analysis as a means of determination. Interestingly, there was a significant interaction ($F = 2.6628$, $p = 0.0418$) found in the CM group using skinfolds as a means of determination of LBM necessitating a follow-up test for simple main effects. Relative to Trial 1 (72.24 ± 8.05 kg), skinfold measurements did show a significant increase ($p < 0.01$) in LBM in Trial 2 (74.92 ± 9.10 kg), Trial 3 (75.21 ± 9.40 kg), Trial 4 (75.58 ± 9.14 kg), and Trial 5 (73.96 ± 8.77 kg) in the CM group. The increase in LBM can be explained by water retention in skeletal muscle. This increase in water retention will increase body density causing an increase in LBM.

There were no significant group differences ($F = 0.1411$, $p = 0.7129$), no significant difference in trials ($F = 2.2056$, $p = 0.0800$), and no significant interaction ($F = 0.3154$, $p = 0.8655$) in LBM using hydrostatic weighing as a means of determination. Also, there were no significant

group differences ($F = 0.0203$, $p = 0.8887$), no significant differences in trials ($F = 1.8347$, $p = 0.1350$), and no significant interaction ($F = 0.0811$, $p = 0.5297$) in LBM using bioelectrical impedance analysis as a means of determination. Therefore, using hydrostatic weighing as the "gold standard", the hypothesis that there will be no effect of oral creatine monohydrate supplementation on LBM is accepted, although it should be noted there were large intraindividual variations in underwater weighing scores in this study.

Summary

The results of this research in agreement with others (Balsom et al., 1993A; Balsom et al., 1993B; Earnest et al., 1995A; Lemon et al., 1995) suggest oral creatine monohydrate supplementation increases body mass. There were no consistent affects of oral creatine monohydrate supplementation on percent body fat and LBM. Interestingly, skinfold measurements did show a significant increase in LBM.

Conclusions

The following conclusions may be drawn from the results of this investigation.

1. Oral creatine monohydrate supplementation does significantly increase overall body mass.
2. Oral creatine monohydrate supplementation does not affect percent body fat as measured by hydrostatic weighing,

skinfolds, and bioelectrical impedance analysis.

3. Oral creatine monohydrate supplementation does not consistently affect LBM as measured by 3 different body composition tests.

Recommendations

The design of this study focused upon a representative segment of competitive athletes interested in increasing body mass and LBM. Considered as the "gold standard", hydrostatic weighing was available for determining body composition. Also, skinfolds and bioelectrical impedance analysis were used to gather quick and easy assessments of body composition. Based on this information and the results of the statistical analysis the following recommendations are proposed:

1. Increase the length of the investigation to evaluate long-term effects of oral creatine monohydrate supplementation on body mass and composition.

2. Follow-up to determine if the effects of oral creatine monohydrate supplementation on body mass are maintained or increased with repeated doses.

3. Increase the number of subjects.

4. Measure residual volume directly during hydrostatic weighing.

5. Include techniques such as dual x-ray absorptiometery (DEXA) in evaluating body composition.

6. Recommend oral creatine monohydrate supplementation to athletes such as wrestlers as a means of quickly increasing body mass to "make weight" and to bodybuilders for a more "bulky" appearance prior to competition.

APPENDIX A

High-Intensity Strength-Training Program

The purpose of this high-intensity program was to build overall body strength. One session consisted of 17 to 20 exercises of the entire body with 1 maximum set of 10 repetitions to exhaustion (10 RM) for each exercise. Subjects participated in 3 sessions per week. The body was broken down into 5 groups:

1) Neck (trapezius)
2) Hips/Legs (gluteuls, quadriceps, hamstrings)
3) Midsection (abdominal, lower back)
4) Torso (deltoids, upper-back, chest)
5) Arms (biceps, triceps, forearm)

Free-weights, nautilus, and universal machines were used to perform the exercises in the university conditioning room. Example exercises for the neck include, but are not limited to dumbbell shrugs and barbell shrugs. Squats, leg presses, leg extensions, and calf raises focused on the legs. Exercises for the midsection included crunch sit-ups with and without weight, leg raises, nautilus abdominal machine, and barbell deadlift.

Barbell press, universal press, and lateral raises focused specifically on the deltoids. The upper-back or the trapezius muscle was worked by lat pulldown with a medium and close grip, seated cable rows, and barbell and dumbbell bent rows. Exercises for the chest included flat bench press, incline bench press, decline bench press, dumbbell fly, dumbbell presses, and parallel bar dips. Arms were focused on by using lat pushdowns, dumbbell extensions, lying barbell extensions, barbell curls, and dumbbell concentration curls.

APPENDIX B

Table B-1. Individual Measures of Body Mass in Kilograms for Creatine Monohydrate (CM) and Placebo (P) Groups

Subject	Trial 1	Trial 2	Trial 3	Trial 4	Trial 5
CM Group					
1	73.64	75.00	76.36	75.00	75.68
2	97.27	102.27	103.64	102.73	102.27
3	65.68	67.73	68.18	68.18	67.73
4	82.27	85.00	83.64	83.98	83.18
5	76.14	76.82	77.05	77.05	76.14
6	88.18	90.00	90.00	90.23	90.23
7	95.13	96.59	97.05	96.02	96.02
8	80.45	81.82	81.70	83.07	82.50
P Group					
9	86.36	87.50	88.41	88.41	88.41
10	87.73	87.27	85.23	86.82	86.68
11	64.55	64.09	64.77	64.09	64.55
12	89.60	92.95	92.75	93.64	94.20
13	86.14	86.36	85.45	86.82	85.91
14	83.63	83.86	83.64	83.64	82.50
15	76.41	76.59	75.80	76.59	74.32
16	75.45	77.73	76.59	77.27	76.36

Table B-2. Individual Measures of Percent Body Fat by Hydrostatic Weighing for Creatine Monohydrate (CM) and Placebo (P) Groups

Subject	Trial 1	Trial 2	Trial 3	Trial 4	Trial 5
CM Group					
1	10.9	10.9	8.9	11.6	13.7
2	18.7	24.4	17.4	18.2	17.3
3	14.8	14.5	9.4	11.4	15.6
4	6.2	9.5	9.6	10.0	10.4
5	13.0	15.1	17.3	17.3	14.6
6	18.4	19.1	17.7	17.3	17.1
7	32.5	29.9	33.1	29.6	29.6
8	12.6	11.0	9.8	9.9	11.4
P Group					
9	8.0	9.6	8.0	9.4	9.4
10	26.6	16.5	17.7	16.8	16.3
11	20.7	21.2	15.4	17.2	13.3
12	17.4	11.1	15.8	15.1	16.8
13	33.9	33.9	33.9	29.5	26.6
14	12.2	17.9	11.3	10.6	11.6
15	7.9	7.0	9.8	8.2	8.4
16	9.4	13.6	15.6	16.3	18.4

Table B-3. Individual Measures of Percent Body Fat by Skinfolds for Creatine Monohydrate (CM) and Placebo (P) Groups

Subject	Trial 1	Trial 2	Trial 3	Trial 4	Trial 5
CM Group					
1	9.9	10.3	10.5	10.9	10.9
2	14.4	13.3	12.5	11.5	14.5
3	6.8	6.8	7.1	5.3	8.1
4	7.9	5.1	4.7	4.9	6.1
5	13.6	14.0	13.4	13.4	14.7
6	21.6	17.5	19.2	13.9	18.4
7	13.0	13.1	12.6	13.9	13.9
8	8.5	7.9	7.8	9.4	8.6
P Group					
9	8.4	8.6	7.2	6.5	9.5
10	16.9	18.0	16.2	16.4	16.1
11	11.5	10.3	12.6	8.9	9.4
12	14.6	14.6	13.6	16.1	12.7
13	18.4	17.3	17.6	19.5	18.2
14	15.2	15.5	15.7	14.7	14.1
15	9.1	9.0	8.4	7.7	8.3
16	7.2	8.1	7.9	6.0	6.5

Table B-4. Individual Measures of Percent Body Fat by Bioelectrical Impedance Analysis for Creatine Monohydrate (CM) and Placebo (P) Groups

Subject	Trial 1	Trial 2	Trial 3	Trial 4	Trial 5
CM Group					
1	10.5	11.7	11.7	11.3	12.2
2	14.9	13.7	14.7	13.8	14.1
3	9.1	8.7	12.2	6.5	7.3
4	19.1	9.2	9.0	22.4	9.1
5	14.0	13.9	18.5	18.5	13.3
6	16.1	14.6	14.8	15.4	12.5
7	15.7	15.5	16.6	19.0	19.0
8	13.6	13.9	12.0	13.2	14.0
P Group					
9	10.5	11.4	10.3	11.4	12.1
10	13.5	14.0	11.6	15.4	16.4
11	6.4	8.4	7.8	9.1	8.6
12	12.9	13.4	12.0	12.6	13.1
13	15.5	15.6	16.0	15.7	16.7
14	14.8	15.0	14.4	15.4	13.8
15	10.8	10.7	9.4	11.6	10.6
16	9.2	9.0	9.0	9.9	15.5

Table B-5. Individual Measures of Lean Body Mass in Kilograms by Hydrostatic Weighing for Creatine Monohydrate (CM) and Placebo (P) Groups

Subject	Trial 1	Trial 2	Trial 3	Trial 4	Trial 5
CM Group					
1	65.61	66.83	69.56	66.30	65.31
2	79.08	77.31	85.61	84.03	84.58
3	55.98	57.91	61.77	60.41	57.16
4	77.17	76.93	75.61	75.58	74.53
5	66.24	66.25	63.72	63.72	65.02
6	71.95	72.82	74.11	74.67	74.61
7	64.21	67.71	64.93	67.60	67.60
8	70.31	72.82	73.69	74.85	73.10
P Group					
9	79.50	79.10	81.34	80.09	80.09
10	64.39	72.90	70.13	72.26	71.69
11	51.19	51.10	54.81	53.07	55.98
12	74.06	82.64	78.06	79.52	78.36
13	56.94	57.05	56.48	61.18	63.02
14	73.43	68.86	74.20	74.80	72.93
15	70.37	71.22	68.35	70.29	68.07
16	68.36	67.16	64.63	64.67	62.63

Table B-6. Individual Measures of Lean Body Mass in Kilograms by Skinfolds for Creatine Monohydrate (CM) and Placebo (P) Groups

Subject	Trial 1	Trial 2	Trial 3	Trial 4	Trial 5
CM Group					
1	66.35	67.28	68.34	66.83	67.43
2	83.26	88.67	90.69	90.92	87.44
3	61.23	63.12	63.34	64.57	62.24
4	75.77	80.67	79.71	79.86	78.10
5	65.78	66.09	66.74	66.74	64.92
6	69.13	74.22	72.73	77.71	73.45
7	82.76	83.94	84.82	82.67	82.67
8	73.61	75.36	75.33	75.34	75.41
P Group					
9	79.10	79.98	82.04	82.66	80.01
10	72.90	71.56	71.45	72.54	71.90
11	57.13	57.50	56.60	58.37	58.48
12	76.57	79.35	80.15	78.58	82.23
13	70.29	71.46	70.37	69.85	70.28
14	70.92	70.85	70.50	71.38	70.86
15	69.46	69.71	69.46	70.66	68.17
16	70.02	71.47	70.56	72.66	71.37

Table B-7. Individual Measures of Lean Body Mass in Kilograms by Bioelectrical Impedance Analysis for Creatine Monohydrate (CM) and Placebo (P) Groups

Subject	Trial 1	Trial 2	Trial 3	Trial 4	Trial 5
CM Group					
1	65.91	66.23	67.41	66.50	66.50
2	82.80	88.27	88.41	88.55	75.03
3	59.72	61.84	59.87	63.73	62.77
4	66.56	77.18	76.09	65.14	75.64
5	65.48	66.18	62.82	62.82	66.00
6	73.98	76.82	76.73	76.32	78.73
7	80.19	81.50	80.91	77.73	77.73
8	69.51	70.45	71.87	72.05	70.95
P Group					
9	77.30	77.53	79.32	78.33	77.77
10	75.89	75.10	75.32	73.50	71.64
11	60.42	58.68	59.69	58.27	59.00
12	78.09	80.55	81.55	81.86	81.82
13	72.79	72.87	71.77	73.18	71.55
14	71.25	71.23	71.59	70.73	71.14
15	68.16	68.36	68.64	67.73	66.45
16	68.51	70.73	69.73	69.64	64.50

REFERENCES

Adams, G.M. Exercise Physiology Laboratory Manual Dubuque, Iowa: Wm. C. Brown Communications, Inc., 1994.

Almada, A., R. Kreider, L. Weiss, A. Fry, L. Wood, D. Bullen, M. Miyaji, P. Grindstaff, L. Ramsey, and Y. Li. Effects of Ingesting a Supplement Containing Creatine Monohydrate for 28 Days on Isokinetic Performance. Medicine and Science in Sports and Exercise 27: S146, 1995.

American College of Sports Medicine. Guidelines For Exercise And Testing Philadelphia: Lea and Febiger, 1991.

American College of Sports Medicine. Resource Manual for Guidelines for Exercise testing and Prescription Philadelphia: Lea and Febiger, 1993.

Balsom, P.D., K. Soderlund, B. Sjodin, and E. Hultman. Creatine supplementation and dynamic high-intensity intermittent exercise. Scandinavian Journal of Medicine and Science in Sports 3: 143-149, 1993A.

Balsom, P.D., S.D.R. Harridge, K. Soderlund, B. Sjodin, and B. Ekblom. Creatine supplementation *per se* does not enhance endurance exercise performance. Acta Physiological Scandinavia 149: 521-523, 1993B.

Balsom, P.D., K. Soderlund, and B. Ekblom. Creatine in Humans with Special Reference to Creatine Supplementation. Sports Medicine 18: 268-280, 1994.

Behnke, A.R., B.G. Feen, and W.C. Welham. The specific gravity of healthy men: Body weight/volume as an index of obesity. Journal of American Medical Association 118: 495-498, 1942.

Birch, R., D. Nobel, and P.L. Greenhaff. The influence of dietary creatine supplementation on performance during repeated bouts of maximal isokinetic cycling in man. European Journal of Applied Physiology 69: 268-270, 1994.

Burke, L.M., D.B. Pyne, and R.D. Telford. Oral Creatine Supplementation Does Not Improve Sprint Performance In Elite Swimmers. Medicine and Science in Sports and Exercise 27: S146, 1995.

Cooke, W.H., P.W. Grandjean, and W.S. Barnes. Effect of oral creatine supplementation on power output and fatigue during bicycle ergometery. Journal of Applied Physiology 78: 670-673, 1995.

Dugdale, A.E., and M. Griffiths. Estimating fat body mass from anthropometric data. American Journal of Clinical Nutrition 32: 2400-2403, 1979.

Earnest, C.P., P.G. Snell, T.L. Mitchell, R. Rodriguez, and A.L. Almada. Effect of Creatine Monohydrate Supplementation on Peak Anaerobic Power, Capacity, and Fatigue Index. Medicine and Science in Sports and Exercise 26: S39, 1994.

Earnest, C.P., P.G. Snell, R. Rodriguez, A.L. Almada, and T.L. Mitchell. The Effect of Creatine Monohydrate Ingestion on Anaerobic Power Indicies, Muscular Strength and Body Composition. Acta Physiologica Scandanavica 153: 207-209, 1995A.

Earnest, C.P., J. Rash, P.G. Snell, A.L. Almada, T.L. Mitchell. Effect of Creatine Monohydrate Ingestion on Intermediate Length Anaerobic Treadmill Running to Exhaustion. Medicine and Science in Sports and Exercise 27: S14, 1995B.

Eckerson, J.M., T.J. Housh, and G.O. Johnson. Validity of bioelectrical impedance equations for estimating fat-free weight in lean males. Medicine and Science in Sports and Exercise 24: 1298-1302, 1992.

Greenhaff, P.L., A. Casey, A.H. Short, R. Harris, K. Soderlund, and E. Hultman. Influence of oral creatine supplementation of muscle torque during repeated bouts of maximal voluntary exercise in man. Clinical Science 84: 565-571, 1993.

Grindstaff, P., R. Kreider, L. Weiss, A. Fry, L. Wood, D. Bullen, M. Miyaji, L. Ramsey, Y. li, and A. Almada. Effects of Ingesting a Supplement Containing Creatine Monohydrate for 7 Days on Isokinetic Performance. Medicine and Science in Sports and Exercise 27: S146, 1995.

Guyton, A.C. Textbook of Medical Physiology Philadelphia: W.B. Saunders Company, 1991.

Hall, E.L., J.C. Smith, D.P. Stephens, P.G. Snell, and C.P. Earnest. Effect of Oral Ingestion of Creatine Monohydrate on Parameters of the Work-Time Relationship. Medicine and Science in Sports and Exercise 27: S15, 1995.

Harris, R.C., K. Soderlund, and E. Hultman. Elevation of creatine in resting and exercised muscle of normal subjects by creatine supplementation. Clinical Science 83: 367-374, 1992A.

Harris, R.C., M. Viru, P.L. Greenhaff, and E. Hultman. The effect of oral creatine supplementation on running performance during maximal short term exercise. Queen Mary and Westfield College Meeting 17-18 December C64, 1992B.

Jackson, A.S. Research design and analysis of data procedures for predicting body density. Medicine and Science in Sports and Exercise 16: 616-620, 1984.

Jackson A.S., and M.L. Pollock. Generalized equations for predicting body density of men. British Journal of Nutrition 40: 497-504, 1978.

Jackson, A.S., and Pollock, M.L. Practical Assessment of Body Composition. Physician and Sports Medicine 13: 76-89, 1985.

Jackson, A.S., M.L. Pollock, J.E. Graves, and M.T. Mahar. Reliability and validity of bioelectrical impedance in determining body composition. Journal of Applied Physiology 64: 529-534, 1988.

Jacobs, I., S. Bleue, and J. Goodman. Creatine Ingestion Increases Maximal Accumulated Oxygen Deficit and Anaerobic Exercise Capacity. Medicine and Science in Sports and Exercise 27: S204, 1995.

Kaminsky L.A., and M.H. Whaley. Differences in estimates of percent body fat using bioelectrical impedance. Journal of Sports Medicine and Physical Fitness 33: 172-177, 1993.

Katch, F.I., and V.L. Katch. Measurement and Prediction Errors in Body Composition Assessment and the Search for the Perfect Prediction Equation. Research Quarterly for Exercise and Sport 51: 249-260, 1980.

Katch, V.L. A reaction to laboratory methodology. Medicine and Science in Sports and Exercise 16: 604-605, 1984.

Lemon, P., M. Boska, D. Bredle, M. Rogers, T. Ziegenfuss, and B. Newcomer. Effect of Oral Creatine Supplementation on Energetics During Repeated Maximal Muscle Contraction. Medicine and Science in Sports and Exercise 27: S204, 1995.

Lohman, T.G. Skinfolds and Body density and Their Relation to Body Fatness: A Review. Human Biology 53: 181-225, 1981.

Lohman, T.G. Research progress in validation of laboratory methods of assessing body composition. Medicine and Science in Sports and Exercise 16: 596-603, 1984.

McArdle, W.D., F.I. Katch, and V.L. Katch. Exercise Physiology Energy, Nutrition, and Human Performance Philadelphia: Lea and Febiger, 1991.

Odland, L.M., J.D. MacDougall, M. Tarnopolsky, A. Elorriaga, A. Borgmann, and S. Atkinson. The Effect of Oral Cr Supplementation on Muscle [PCr] and Power Output During a Short-Term Maximal Cycling Task. Medicine and Science in Sports and Exercise 26: S23, 1994.

Pollock, M.L., and A.S. Jackson. Research progress in validation of clinical methods of assessing body composition. Medicine and Science in Sports and Exercise 16: 606-613, 1984.

Pope, H.G., D.L. Katz, and R. Champoux. Anabolic-androgenic Steroid Use Among 1,010 College Men. Physician and SportsMedicine 16: 75-81, 1988.

Redondo, D., M. Williams, E. Dowling, B. Graham, S. Jones, and A. Almada. The Effect of Oral Creatine Monohydrate Supplementation on Running Velocity. Medicine and Science in Sports and Exercise 27: S146, 1995.

Siri, W.E. Body Composition from fluid spaces and density: Analysis of methods in techniques for measuring body composition. National Academy of Science National Research Council Washington D.C.: 223-244, 1961.

Thorland, W.G., G.O. Johnson, G.D. Tharp, T.G. Fagot, and R.W. Hammer. Validity of anthropometric equations for the estimation of body density in adolescent athletes. Medicine and Science in Sports and Exercise 16: 77-81, 1984.

Valhalla Scientific, Inc. Model 1990B Bio-resistance Body Composition Analyzer Operation Manual San Diego: Valhalla Scientific, 1988.

Wardlaw, G.M., and P.M. Insel. Perspectives In Nutrition St.Louis: Mosby, 1993.

Weltman, A., and V. Katch. Comparison of hydrostatic weighing at residual volume and total lung capacity. Medicine and Science in Sports and Exercise 13: 210-213, 1981.

Wilmore, J.H. The use of actual, predicted and constant residual volumes in the assessment of body composition by underwater weighing. Medicine and Science in Sports and Exercise 1: 87-90, 1969.

Wilmore, J.H., and A.R. Behnke. An anthropometric estimation of body density and lean body weight in young men. Journal of Applied Physiology 27: 25-31, 1969.

Yesalis, C.E., R.T. Herrich, W.E. Buckley, K.E. Friedl, D. Brannon, and J.E. Wright. Self-Reported Use of Anabolic-Adrogenic Steroids by Elite Power Lifters. Physician and SportsMedicine 16: 91-98, 1988.