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Effects of creatine supplementation on muscular strength and power development in well-trained football players

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EFFECTS OF CREATINE SUPPLEMENTATION ON MUSCULAR STRENGTH AND POWER DEVELOPMENT IN WELL-TRAINED FOOTBALL PLAYERS

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To the Graduate Faculty:

I am submitting herewith a thesis written by Josef D. McQuain entitled "Effects of Creatine Supplementation on Muscular Strength and Power Development in Well-Trained Football Players." I recommend that it be accepted for six semester hours of credit in partial fulfillment of the requirements for the degree of Master of Science, with emphasis in Exercise Physiology.

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TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

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Abstract

EFFECTS OF CREATINE SUPPLEMENTATION ON MUSCULAR STRENGTH AND POWER DEVELOPMENT IN WELL-TRAINED FOOTBALL PLAYERS.

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The purpose of this study was to determine the effects of creatine supplementation on strength and power performance in well-trained football players. Twenty-one subjects were randomly assigned to three treatment groups: 1)creatine monohydrate, 2)creatine monohydrate+dextrose, or 3)placebo utilizing a double-blind design. Prior to assignment, subjects were matched according to lean body mass. After a five day loading dose, a maintenance dose was administered for 12 days. Test protocols included three anaerobic work bouts: 1)two 30 s Wingate bike tests interspersed with five minutes recovery were used to ascertain relative and absolute power, 2)two 100 yard sprints with five minutes rest between each bout were used to determine times for 40, 60, and 100 yards sprints, 3)one repetition maximum (1 RM) and 70% 1 RM for the bench press were determinants for muscular strength and endurance. Over the 17 day study, an intense program which combined sprint and resistance training was employed. A two way ANOVA for repeated measures revealed non-significance for the effects of creatine on performance parameters at a 0.05 level of confidence. Within subjects training effects were significant for percent body fat ($p<.01$), 1RM ($p<.001$), peak anaerobic power ($p<.01$), and 100 yard sprint ($p<.001$). These findings suggest that creatine supplementation does not positively affect performance in previously trained football players.

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Chapter I

INTRODUCTION

Our society has placed increasing amounts of pressure on athletes to win. Many athletes have responded by striving to become better conditioned through the utilization of trainers, conditioning coaches, and the latest trends in exercise science. As athletes continue to become better conditioned, however, they also approach human physiological limitations for athletic performance. For example, the world record in the 100 m dash has changed very little since 1968, progressing from 9.95 to 9.85 s. Also, in the 33 years since Jesse Owens' 1935 long jump of 26 ft 8.25 in, the world record has increased by only 8.5 in. Then, in 1968 Bob Beamon surpassed the 28 ft barrier; ironically, it was not until the 1980 Olympics that another athlete jumped over 28 feet (Wallechinsky, 1996). These two examples are not isolated incidents. World records in almost every sporting event continue to be improved. Unlike Bob Beamon's performance however, the records are surpassed only by very small increments.

Athletes at any level (high school, recreational, college, or professional) often utilize performance enhancing substances to gain an advantage over competitors or to surpass current records. Although many of these substances cause non-significant physiological results and are no more than just marketing schemes, one substance that is currently yielding

promising results by some researchers is creatine monohydrate (CM).

HISTORICAL BACKGROUND

As early as the 1800s, scientists were aware of creatine (Cr) and its importance to muscle function. In 1835, Chevreul, a French scientist, first identified Cr in meat extract (Balsom, Soderlund, & Ekblom, 1994). However, due to problems with the method to identify Cr, it was not until 1847 that Lieburg effectively demonstrated that Cr could be extracted from the muscle tissue of several mammals. At this time Lieburg found that the muscles of wild foxes killed in the chase contained approximately 10 times the amount of Cr as captive foxes; therefore, he concluded that muscle work involved the accumulation of Cr (Balsom et al., 1994). Around the same time of Lieburg's discovery, Heintz and Pattenkofer (Balsom et al., 1994) discovered a substance that was excreted in the urine, which was confirmed to be creatinine. It was then concluded that creatinine was related to muscle mass and it was further speculated that the creatinine excreted in urine was directly derived from the Cr stored in the muscle.

Early in this century several studies involving Cr feeding were undertaken. The Cr utilized in these studies was extracted from either fresh meat, its most productive source, or urine.

Due to its cost effectiveness, using Cr from urine was deemed more favorable (Balsom et al., 1994). It was observed that not all of the Cr ingested by animals and humans was recovered in the urine; some was retained in the body. Folin (1912) and Denis (1914) partly explained the fate of this exogenous Cr by determining that the creatine content of cats increased by up to 70% after Cr ingestion (Balsom et al., 1994). In 1923 Hahn and Meyer, estimated the total Cr content of a 70 kg male to be approximately 140 gram, which correlates to the figure used today (Balsom et al., 1994).

Between 1927 and 1929, a "labile phosphorus" was discovered in resting muscle of cats which was termed phospho-creatine (PCr) by Fiske and Subbarow (Balsom et al., 1994). It was demonstrated that during electrical stimulation of the muscle, PCr diminished, only to reappear during a recovery period. Therefore, it follows that PCr is a renewable energy source by its phosphorylation and dephosphorylation.

Even though most studies to this point focused on supplemental Cr, Cr is also endogenously synthesized in the liver and pancreas of the human body (Walker, 1979). Approximately 95% of the total amount of Cr in the human body is stored within skeletal muscle, away from the synthesization site (Walker, 1979). Oral ingestion of Cr depresses biosynthesis, which is reversible when supplementation ceases (Greenhaff, 1995).

Thus, it is difficult to predict how much creatine will be retained since Cr ingestion influences biosynthesis.

Despite several decades of studies focusing on Cr metabolism, it was only recently that the influence of supplemental Cr upon exercise performance has been examined. Research by Harris, Soderlund, and Hultman (1992), showed that total Cr content could be increased by 20% following Cr supplementation.

Energy is created anaerobically from the breakdown of high energy phosphates adenosine triphosphate (ATP) and PCr. During short, high intensity work, cellular ATP concentrations begin to fall. If ATP levels are excessively diminished, fatigue occurs resulting in decreased performance (Brooks, Fahey, & White, 1996).

As intracellular ATP continues to decline, regeneration of ATP is essential if fatigue is to be delayed. Therefore, as quickly as ATP is metabolized during a muscle contraction, it is continuously reformed from adenosine diphosphate (ADP) and an inorganic phosphate (Pi) by the energy liberated from the breakdown of stored PCr. These reactions are represented as follows:

PCr ---> Pi + Cr + Energy

Energy + ADP + Pi ---> ATP

This suggests that the onset and development of short-term muscular fatigue is linearly related to the decreasing levels of PCr. Therefore, through oral Cr supplementation, intramuscular total Cr levels can be increased which may reduce the depletion of PCr stores resulting in delayed fatigue and improved performance.

STATEMENT OF THE PROBLEM

The purpose of this study was to examine the effects of Cr supplementation on strength and power performance in well-trained football players. Comparisons were made between groups of subjects receiving creatine monohydrate (CM), creatine + dextrose (CM+D), and a placebo (P) group. Also, body composition variables assessed were lean body mass (LBM) and fat mass (FM).

NULL HYPOTHESIS

There will be no significant difference in the performance variables of strength and power between the athletes receiving either type of Cr and the athletes receiving the placebo. Also, no significant changes will be found when comparing body composition variables between groups.

IMPORTANCE OF THE STUDY

In the current literature, several studies have demonstrated the effects of Cr supplementation on performance (Balsom, Soderlund, Sjodin, & Ekblom, 1995; Birch, Noble, & Greenhaff, 1994; Cooke, Grandjean, & Barnes, 1995; Geenhaff, Casey, Short, Harris, Soderlund, & Hultman, 1993). However, only one study to the author's knowledge has used Cr supplementation with strength trained athletes (Earnest, Snell, Rodriguez, Almada, & Mitchell, 1995). Well-trained subjects are closer to their physiological limitations of strength and power. It has not been established that highly trained subjects will benefit from creatine supplementation, especially in view of the fact that they are already closer to their genetic potential for strength and power.

BASIC ASSUMPTIONS

1. The subjects participating in this study exhibited high levels of strength and conditioning which resulted from chronic resistance training.
2. All subjects performed at maximum levels of effort during testing of the performance variables.
3. Environmental conditions did not significantly influence performance.
4. The subjects did not significantly change their nutritional behaviors during the study other than the consumption of CM.

LIMITATIONS

1. Only twenty-one athletes were involved in this study.
2. Subjects performed very difficult and uncomfortable tests in the pre-testing which caused subjects to dropout of the study or decrease their effort on post-testing.
3. Subjects in the placebo group reported that they did not consume Cr. However, these reports could not be confirmed.

Chapter II

REVIEW OF LITERATURE

This review of literature focuses on the metabolic fate, as well as the function of Cr for energy production. Secondly, the effects of Cr supplementation upon fatigue and performance are presented. Thirdly, differences in the results of the current literature involving Cr supplementation, and its effects on muscular Cr content and performance, as well as relevant side effects will be discussed.

CREATINE

Cr is a nitrogenous organic compound obtained predominantly from the ingestion of meat, fish, and other animal products. The average intake of Cr from a mixed diet has been estimated to be one gram per day (Hoogwerf, Laine, & Greene, 1986). Also, Cr is synthesized from glycine, arginine, and methionine by enzymes located in the liver, pancreas, and kidneys (Walker, 1979). However, between 95 (Balsom et al., 1994) and 98% (Heymsfield, Arteaga, McManus, Smith, & Moffit, 1983) of the total Cr is contained in skeletal muscle. Cr must therefore be transported from its site of synthesis to its primary site of storage via the bloodstream, where active uptake by skeletal muscle against a concentration gradient occurs. The normal concentration of Cr in

plasma is 50 to 100 mmol/L (Harris et al., 1992). Whereas, the concentration of Cr in skeletal muscle is approximately 125 mmol/kg of dry muscle (Harris, Hultman, & Nordesjo, 1974).

The mechanism responsible for the movement of Cr from the bloodstream into the muscle has recently been discovered. A sodium-dependent neurotransmitter related to the taurine and GABA/betaine transporters has demonstrated an effective role in the transportation of Cr into a number of cell types (Bennett, Bevington, & Walls, 1994; Guimbal & Kilimann, 1993; Scholoss, Mayser, & Betz, 1994).

The presence of insulin (Haugland & Chang, 1975; Koszalka & Andrew, 1972) and triiodothyronine (Odoom, Kemp, & Radda, 1993) may enhance the uptake of Cr, while the presence of digoxin or lack of vitamin E may depress it (Gerber, Gerber, Koszalka, & Emmel, 1962). Although the maintenance of normal Cr levels can be attained endogenously, such as in vegetarians, individuals who consume foods high in Cr content tend to have higher basal Cr levels (Delangh, De Slypere, De Buyzere, Robbrecht, & Vermeulen, 1989; Greenhaff, 1995; Harris et al., 1992). In the absence of exogenous Cr, Cr and PCr are degraded into creatinine through a nonenzymatic, irreversible reaction that is estimated to occur at a rate of approximately 1.6% per day (Crim, Calloway, & Margon, 1975; Crim, Calloway, & Margon, 1976). Through diffusion, creatinine is eventually excreted in the urine.

FUNCTION OF CREATINE AND PHOSPHOCREATINE

Temporal Energy Buffer

The major role of PCr during periods of high energy demand is the donation of phosphate for energy production (Brannon, Adams, Conniff, & Baldwin, 1997). As energy demands increase, intracellular ATP is utilized for energy. However, as the ATP is broken down it is quickly resynthesized from ADP and Pi by the energy liberated from the breakdown of stored PCr. The enzymatic reaction involving Cr and PCr is the reversible Cr kinase reaction, which is controlled by Cr kinase (CPK):

$$PCr + ADP + H^{+} \leftarrow\!-\!-(CPK)\!-\!-\!\rightarrow Cr + ATP$$

Spatial Energy Buffer

Since Bessman (1954) first postulated the existence of the PCr energy shuttle, data has continued to accumulate supporting its presence in muscle (Bessman & Savabi, 1990). In the shuttle, Cr and PCr are reciprocally diffused between the mitochondrial production sites and sites of utilization (myofibrils) in order for energy production to continue. There are three parts to the phosphocreatine energy shuttle:

1. A peripheral terminus located at the site of utilization (myosin);
2. An intervening space between utilization and production;

3. An energy-generating terminus located within the mitochondria.

At the peripheral terminus, PCr is utilized by CPK to rephosphorylate ADP, producing ATP and liberating free Cr. The free Cr enters the intervening space, where Cr and PCr diffuse in opposite directions. The free Cr arrives at the mitochondrial site, where it interacts with CPK producing PCr through mitochondrial ATP utilization. The PCr is then shuttled back to the site of utilization and the process begins again.

Proton Buffer

During the hydrolysis of ATP, PCr acts to buffer protons (H+) (Brannon et al., 1997). When conditions are favorable, H+ are utilized in the Cr kinase reaction to produce ATP, thereby contributing to the prevention of acidification and maintaining pH. It has been postulated that any improvements in anaerobic athletic performance may be a result of creatine's buffering capacity (Hultman, Spriet, & Soderlund, 1987).

Modulator of Glycolysis

Cr may also play a role in modulating glycolysis. As muscular activity progresses in intensity, the rate of glycolysis must increase greatly. Also, in order to meet the demand for ATP, the concentration of PCr declines via the Cr kinase

reaction. Research has suggested that the process of glycolysis may be stimulated by the decline of PCr levels. It is theorized that phosphofructokinase (PFK), the rate limiting enzyme for glycolysis, is partially inhibited by PCr concentrations and ATP/ADP ratio. During strenuous activity, when PCr levels decline and the ATP/ADP ratio drops, PFK is disinhibited resulting in an increased rate of glycolysis (Storey & Hochachka, 1974). This increase in glycolysis permits the production of more ATP, allowing for muscular work to continue.

ENERGY DEMANDS OF HIGH-INTENSITY EXERCISE THROUGH PHOSPHOCREATINE DEGRADATION AND ANAEROBIC GLYCOLYSIS

During high-intensity exercise, the demand for the phosphorylation of ADP to ATP is primarily supplied through degradation of PCr and anaerobic glycolysis. At rest, muscle fibers are capable of increasing PCr concentration to levels approximately four times that of ATP (Brooks et al., 1996; Vander, Sherman, & Luciano, 1990). Upon the onset of muscle contraction, when the concentration of ATP begins to decline and ADP concentration rises due to the increased rate of ATP utilization, mass action favors the formation of ATP from PCr. This transfer of energy from PCr to ATP is so rapid that the concentration of ATP in a muscle fiber changes very little at the onset of muscle contraction, whereas the concentration of PCr

declines dramatically (Balsom et al., 1995; Gaitanos, Williams, Boobis, & Brooks, 1993; Spriet, Soderlund, Bergstrom, & Hultman, 1987).

Although the formation of ATP from PCr is very rapid, the amount of ATP produced by this process is limited by the PCr concentration. Therefore, if contractile processes continue for more than a few seconds, the muscle must be able to produce ATP from a source other than PCr. Since PCr depletion occurs faster than glycogen depletion, the continued ATP demands are met by anaerobic glycolysis (Boobis, 1987; Hirvonen, Rehunen, Rusko, & Harkonen, 1987; McCartney, Spriet, Heigenhauser, Kowalchuk, Sutton, & Jones, 1986; Spriet et al., 1987). In fact, Boobis (1987) estimated that the ATP from PCr declined more than 3 times as fast as the rate of glycolysis.

IDENTIFYING FATIGUE DURING HIGH-INTENSITY EXERCISE

Prior to determining the cause and site of fatigue, it must first be defined. Fatigue, in athletic competition, means an inability to maintain a given exercise intensity or power output. Due to the broad nature of research on fatigue, this review will focus only on those variables associated with fatigue during high-intensity exercise, such as PCr depletion, pH alteration, and lactic acid accumulation.

The Relationship of Phosphocreatine and Fatigue

Research has indicated that the concentration of PCr may contribute to force production and fatigue. Studies utilizing muscle biopsies of human quadriceps muscle during cycling exercise demonstrate that PCr levels drop rapidly, which appears to be related to the relative work intensity for the subject. Therefore, as the work load increases so does the depletion of PCr. In fact, it can be estimated that PCr levels become totally depleted within 10 s of high-intensity exercise (Gaitanos et al., 1993; Spriet et al., 1987).

Hirvonen et al. (1987), observed that PCr stores were depleted in subjects after 5-7 s following sprints of 40, 60, 80, and 100 m. Also, running speed was found to decline after 5 s of exercise; therefore, Hirvonen and associates concluded that the decline in energy production was related to the lack of energy supplied from PCr.

In a study by Katz, Sahlin, & Henriksson (1986), which focused on the maintenance of isometric maximal voluntary contraction force of the knee extensor muscles, they concluded that fatigue was more closely associated with low PCr levels than with high lactate content (anaerobic glycolysis). They reached this conclusion because the range of muscle lactate values at the point of fatigue were very broad, whereas the PCr levels were nearly depleted (86-99% of resting values).

Several studies have demonstrated higher basal PCr levels as well as greater depletion of PCr and glycogen in fast-twitch (FT) muscle fibers compared to slow-twitch (ST)(Edstrom, Hultman, Sahlin, & Sjoholm, 1982; Essen, 1978; Tesch, Thorsson, & Fujitsuka, 1989). Also, PCr content appears to be lower in FT than ST fibers after recovery. Due to the greater loss and slower resynthesis of PCr and glycogen in FT fibers following high-intensity exercise, an assumption can be made that FT fibers contribute to force reduction, as these fibers are predominantly recruited during exercise of high-intensity (Hultman & Greenhaff, 1991; McCardle, Katch, & Katch, 1994).

Phosphocreatine Resynthesis

The process of PCr resynthesis in human skeletal muscle is oxygen dependent, with a slow and fast component (Harris, Edwards, Hultman, Nordesjo, Nylind, & Sahlin, 1976; Sahlin, Harris, & Hultman, 1979). Following high-intensity exercise, approximately half of the pre-exercise PCr content is restored within one to three minutes of recovery (21-22 s for the fast component; 170 s for the slow component) (Soderlund & Hultman, 1991). Whereas, total resynthesis of PCr occurred in approximately five minutes of recovery (Bogdanis, Nevill, Lakomy, & Boobis, 1993; Soderlund & Hultman, 1991).

To maintain constant force production, the resynthesis of PCr must occur at a rate equal to the depletion of PCr. During high-intensity exercise, energy demands are great requiring the rate of PCr resynthesis to increase proportionally in order to maintain power output. When inadequate recovery periods are given, several studies have demonstrated that PCr resynthesis is not as efficient, resulting in impaired performance (Balsom, Seger, Sjodin, & Ekblom, 1992a; Balsom, Seger, Sjodin, & Ekblom, 1992b; Bogdanis et al., 1993).

Lactic Acid Accumulation

The production of lactic acid during high-intensity exercise may result in the decline of power production. As the production of lactic acid exceeds its removal, lactic acid accumulates in the muscle. The accumulated lactic acid dissociates into a lactate anion and a hydrogen cation (H+). It is the H+ that causes the pH to decrease.

The H+ accumulation resulting from lactic acid production can have several adverse effects. Within the muscle, elevated hydrogen concentration and reduced pH inhibit two key enzymes of glycolysis, phosphorylase and PFK (Brooks et al., 1996). The H+ may have a direct effect on the contractile process by displacing calcium from troponin (Brooks et al., 1996).

Furthermore, pain receptors may be stimulated by the drop in pH (Brooks et al., 1996). Finally, acidosis may affect the equilibrium reactions such as CPK reaction, resulting in further PCr depletion (Sahlin, 1983). Due to the numerous effects of H+ and lactate accumulation, a decline in muscle force production can be postulated.

CREATINE SUPPLEMENTATION

Since the beginning of the twentieth century studies have demonstrated that a fraction of exogenous creatine has been retained within the body. These studies were primarily based on the amount of creatine ingested versus the amount of creatinine excreted. Folin (1912) and Denis (1914), however, found increases up to 70% in the creatine content of muscle in cats and rabbits following creatine feeding (Balsom et al., 1994). A recent study by Harris and associates (1992) confirmed that the size of the total Cr pool can be increased through Cr supplementation in humans.

Cr feeding in humans is possible by oral adminstration of Cr monohydrate, a white power which is soluble in warm water. Upon ingestion of five grams of Cr monohydrate, the plasma level of Cr has been shown to rise from between 50 to 100 micro moles/L to over 500 micro moles/L one hour after ingestion (Harris et al., 1992).

Changes in Total Muscle Creatine Content

In a study by Harris and associates (1992), the content of total Cr (free Cr + PCr) within the muscle was shown to increase in a group of healthy participants following a regimen of Cr feeding. Cr supplementation consisted of five grams of Cr administered four to six times daily for two or more days. The mean total Cr content prior to Cr feeding for all subjects was 126.8 (SD 11.7) mmol/kg of dry muscle. With the exception of two subjects, whose initial total Cr content was above 145 mmol/kg of dry muscle, all other subjects demonstrated an increase in total Cr content. The mean total Cr content of all subjects after Cr supplementation was 148.6 (SD 5.0) mmol/kg of dry muscle. The increase in total Cr content appeared to be less dependent upon the duration of supplementation and dosage rate, than the initial total Cr content. The increase in total Cr was a result of an increase in both Cr and PCr, of which PCr increased from 20-40%.

No ill effects or blood profile changes were noted during Cr supplementation. Harris and associates (1992) did suggest that an upper limit for total Cr existed (155 mmol/kg of dry muscle), at least when consuming four to six grams of Cr daily.

Several recent studies confirmed the observations by Harris and associates (1992), that total Cr content was increased above resting values after Cr supplementation. In one study, a group of cardiac patients consumed 20 grams of Cr daily for 10 days.

While no changes were seen in the placebo from baseline, the Cr supplementation group demonstrated an increase in total Cr by 17 $\pm$ 4% ($p<.05$) and PCr 12 $\pm$ 4% ($p<.05$) (Gordan, Hultman, Kaijser, Kristjansson, Rolf, Nyquist, & Sylven, 1995).

Greenhaff, Bodin, Soderlund, and Hultman (1994) obtained biopsy samples from the vastus lateralis muscle of eight subjects after 0, 20, 60, and 120 s of recovery from intense electrically evoked isometric contraction. The samples were taken prior to and after their subjects ingested 20 gram of Cr a day for five days. In five of the eight subjects, Cr supplementation substantially increased muscle total Cr by a mean of 29 $\pm$ 3 mmol/kg of dry muscle. In the remaining three subjects, Cr supplementation had little effect on total muscle Cr content, producing increases of 8-9 mmol/kg of dry muscle.

Another study in which seven male subjects performed repeated bouts of high-intensity exercise on a bicycle ergometer, before and after six days of Cr supplementation at 20 grams/day, demonstrated that total muscle Cr content at rest increased from 128.7 $\pm$ 4.3 to 151.5 $\pm$ 5.5 mmol/kg dry muscle ($p<.05$) (Balsom et al., 1995). Again, these results were similar to Harris et al. (1992).

In contrast to Harris et al. (1992) and others, research by Odland, MacDougall, Tarnopolsky, Elorriaga, and Borgmann (1997) concluded that no difference existed between Cr, placebo or

control groups for ATP, PCr, or total Cr content. However, the total Cr content/ATP ratio was greater in the Cr group ($p<.05$). It was unclear as to why this occurred; Odland et al. (1997) offered no explanation.

Nine males in the latter study underwent a series of randomly ordered tests following the ingestion of Cr, placebo, and control. The dosage regimen for Cr supplementation was 20 grams/day for three days, which was dissolved in a flavored drink. The placebo consisted of the drink only. Tests were performed 14 days apart on a Monarch ergometer. Needle biopsies were obtained from the vastus lateralis at the end of each treatment period and prior to each exercise test.

Phosphocreatine Resynthesis

To investigate the effects of Cr supplementation upon the PCr resynthesis, subjects underwent 20 intense, electrically evoked isometric muscle contractions of the quadriceps lasting 1.6 s interspersed with 1.6 s rest periods. During the testing each subject was supplemented with 20 grams of Cr for five days (Greenhaff, Bodin, Harris, Hultman, Jones, McIntyre, Soderlund, & Turner, 1993). After two minutes of recovery, the Cr supplemented group had PCr values 20% higher than those of the control group. Greenhaff, Bodin et al. (1993) concluded that Cr supplementation had increased the rate of PCr resynthesis.

Another study confirmed the findings of Greenhaff, Bodin et al. (1993). Greenhaff, Bodin, Soderlund, and Hultman (1994) determined the PCr level in the vastus lateralis was depleted to approximately 8 mmol/kg of dry muscle via electrical stimulation. Then the rate of PCr resynthesis over the first two minutes of recovery was compared before and after Cr supplementation (20 grams/day over five days). This study revealed that PCr resynthesis was similar during the first minute of recovery when comparing subjects before and after Cr supplementation. However, during the second minute of recovery the mean rate of PCr resynthesis after Cr ingestion increased by approximately 42% (Greenhaff et al., 1994). Despite this trend, the mean PCr resynthesis was not significantly different at the end of recovery when comparing treatments. Again, the subjects with the lowest initial total Cr content demonstrated greater gains in total Cr, as well as an increased rate of PCr resynthesis after Cr ingestion.

Performance During High-Intensity Exercise

The importance of delaying fatigue in order to maintain force production for longer periods of time seems to be highly applicable for athletic performance. Many athletes utilize the phosphagen energy system during participation in their sport, sprinters, power lifters, body builders, football, basketball,

hockey, and volleyball players. All of these individuals may benefit from increased PCr stores. Several studies have indicated that with Cr supplementation (20 grams a day for 5-6 days), improvements in performance have been demonstrated when compared to placebo or control groups (Balsom, Ekblom, Soderlund, Sjodin, & Hultman, 1993; Birch et al., 1994; Earnest et al., 1995; Greenhaff, Casey et al., 1993; Harris, Viru, Greenhaff, & Hultman, 1993; Soderlund, Balsom, & Ekblom, 1994). However, several studies with similar methodologies demonstrated non-significant improvements in performance (Balsom et al., 1995; Cooke et al., 1995; Mujika, Chatard, Lacoste, Barale, & Geyssant, 1996; Odland et al., 1997; Rossiter, Cannel, & Jakeman, 1995).

Greenhaff, Casey et al. (1993) had 12 subjects, non-highly trained, who performed five bouts of 30 maximal voluntary isokinetic contractions, interspersed with one minute rest periods. These tests were completed before and after five days of placebo or Cr supplementation. Peak torque, plasma ammonia, and blood lactate accumulation were analyzed. Results revealed that the Cr group was able to significantly reduce the rate of peak torque decline during bouts two, three, and four. Also, plasma ammonia accumulation was lower during and after exercise for the Cr group. However, no differences were seen in lactate production between groups. The greater torque production generated by the Cr group was attributed to the increased

availability of PCr, which allows ATP regeneration to occur at a rate closer to the energy demand.

The finding that Cr supplementation could enhance performance of short duration, high-intensity exercise was observed in a double-blind study in which approximately 16, physically active to well-trained, subjects were randomly assigned to a placebo or Cr group (Balsom, Ekblom et al., 1993). Each subject was to perform ten 6 s bouts of high intensity cycling at 140 revolutions per minute, with recovery periods of 30 s. The exercise protocol was performed before and after treatments. Variables measured were pedal frequency, blood lactate, hypoxanthine, and oxygen uptake. The study revealed that the Cr group was better able to maintain pedal frequency during the second half of each exercise bout. Also, plasma hypoxanthine accumulation was reduced after Cr supplementation, as was blood lactate. Decreased hypoxanthine levels are indicative of reduction in nucleotide degradation, which is theorized to contribute to the buffering of ATP during exercise (Volek & Kraemer, 1996).

Balsom, Ekblom et al. (1993) suggested that the increased power output was due to an acceleration of PCr resynthesis during recovery periods, which allowed PCr concentration to be greater prior to each bout of exercise. They also postulated that the lower lactate and hypoxanthine accumulation was related to the

greater content of Cr within the muscle, thereby altering the production of energy.

In a follow up study using a similar exercise model and the same Cr regimen, 8 male subjects performed five 6 s bouts of exercise with 30 s recovery periods. After 40 s rest, one ten second bout of exercise followed, where the ability to maintain power output was evaluated. Results revealed that after Cr ingestion the ability to sustain high power output improved significantly (Soderlund et al., 1994). Total Cr concentration was higher at rest. Also, higher PCr concentration and lower lactate accumulation were measured following the five 6 s bouts of exercise. They attributed their results to higher pre-exercise PCr concentrations following Cr supplementation, which may have led to less dependence upon anaerobic glycolysis for the resynthesis of ATP (i.e., lower lactate accumulation).

Balsom et al. (1995) duplicated the study by Soderlund et al. (1994), adding a series of counter movement jumps and squat jumps before and after the administration period. In agreement with Soderlund and associates (1994), the results revealed an increase in total Cr, higher PCr concentration and lower lactate accumulation after Cr ingestion. Subjects were also better able to maintain power output during a ten second exercise period. However, no change in jump performance was noted as a result of Cr supplementation ($p<.05$). The findings that jump performance

was not enhanced suggests that short-term Cr ingestion does not influence peak power. This seems plausible, due to the fact that during one jump (counter movement or squat) ATP is utilized to produce energy, not PCr.

Birch and associates (1994) examined the effects of Cr supplementation on performance during three 30 s bouts of maximal isokinetic cycling interspersed with four minutes recovery. Fourteen healthy men were used as subjects. Peak and mean power, total work, plasma ammonia, and lactate were investigated. Placebo ingestion reveal no differences in all variables. However, following Cr supplementation peak power output was approximately 8% higher in bout one, mean power was approximately 6% greater during bouts one and two. Total work was also higher in bouts one and two. Peak plasma ammonia was lower following Cr ingestion (160 $\pm$ 18 to 129 $\pm$ 22 micro moles/L). No differences were noted in lactate accumulation before or after Cr supplementation, which was in agreement with Greenhaff, Casey, and colleagues (1993) but in contrast to Balsom, Ekblom et al. (1993) and Soderlund et al. (1994).

Cooke and colleagues (1995) examined the effect of Cr supplementation on power output and fatigue in 12 untrained males. The exercise protocol consisted of two consecutive 15 s maximal sprints on a cycle ergometer interspersed with 20 minutes of rest. Each trial was performed before and after, either Cr or

placebo ingestion as used by Harris and associates (1992). Variables of interest were peak power, time to peak power, total work, and fatigue index (percent power decline). Results revealed no significant differences within or between groups before or after Cr supplementation for any mechanical variables measured ($p<.05$).

In a similar study, Odland et al. (1997) proposed to determine the effect of Cr supplementation on power output during a 30 s maximal Wingate test. Nine healthy males participated in three randomly ordered exercise tests following ingestion of Cr, placebo, and control. Cr supplementation consisted of 20 grams a day for three days. Tests were performed 14 days apart. Needle biopsies from the vastus lateralis were taken at the end of each treatment period and before each test. Results demonstrated no differences between peak, mean 10 s, and mean 30 s power output, percent power decline, or post-exercise lactate concentration. Also, no difference was observed for ATP, PCr, or total Cr. However, the total Cr/ATP ratio was higher in the Cr group compared to the placebo and control conditions ($p<.05$). The total Cr contents were normalized relative to ATP content in order to control for possible differences that may have been a result of varying quantities of blood or connective tissue in the different biopsy samples (Odland et al., 1997).

These findings suggest that three days of Cr supplementation does not increase the resting muscle PCr and has no effect upon performance during a single short term maximal cycling exercise bout.

Earnest and colleagues (1995) supplemented four strength-trained males with 20 grams of Cr a day for 28 days while four subjects received a placebo. After 14 days of Cr supplementation, total anaerobic work for the Wingate was significantly higher for each of the 3 trials ($p<.05$). No changes were noted in the placebo. Following 28 days of Cr supplementation, the 1RM for the bench press increased 6% ($p<.05$). Total lifting volume (the number of repetitions at 70% 1RM) was significantly higher as well, increasing 26% ($p<.05$). Conclusion was made that Cr supplementation may increase muscle strength and endurance.

Balsom, Harridge et al. (1993) investigated the effects of Cr supplementation on prolonged continuous exercise at intensities exceeding maximal oxygen consumption. Eighteen habitually active to well-trained subjects were divided into a Cr or placebo group. After completion of a maximal oxygen consumption test, each subject returned on two consecutive days to perform a supra maximal (120% maximal oxygen consumption) treadmill run till exhaustion and a 6 km terrain run on a forest track. The two runs were repeated following Cr (five grams four

times daily) or placebo supplementation. Results for the treadmill run revealed no significant changes in performance (time to run 6 km), oxygen consumption, or peak heart rate. Lactate accumulation was significantly greater with the treadmill run following Cr supplementation. Balsom, Harridge et al. (1993) suspected that this increase in lactate accumulation was related to the increased body mass experienced by their subjects. During the terrain run a significant increase in run time was noted for the Cr group (6 km time slower post-ingestion compared to pre-ingestion). However, no changes in mean heart rate, lactate accumulation, or plasma hypoxanthine concentration were observed in the terrain run. Balsom, Harridge, and associates (1993) concluded that Cr supplementation does not enhance performance or increase peak oxygen uptake during prolonged continuous exercise.

Ten trained middle distance runners were divided into two groups Cr or placebo. The Cr group received five grams of Cr plus five grams of glucose six times daily, whereas the placebo group ingested 10 grams of glucose a day. Subjects were unaware of the treatment allocations though this was known by their supervising coach. On separate days, prior to and following Cr supplementation, subjects performed 4 x 300 m and 4 x 1000 m runs maximally with four and three minute rest intervals between repetitions. Results revealed a reduction in run time over the final 300 m and 1000 m runs significantly greater in the Cr

group, as was the reduction in the 4 x 1000 m running time (Harris et al., 1993). While, the best 300 m and 1000 m times decreased significantly with Cr supplementation, they were unchanged in the placebo group.

Mujika and associates (1996) examined the effects of Cr supplementation of sprint performance in competitive swimmers. Twenty highly trained swimmers (9 female, 11 male) were tested for blood ammonia and lactate after 25, 50 and 100 m performance on two separate occasions seven days apart. After the first trial, subjects were randomly assigned to a Cr (five grams four times a day for five days) or placebo group. Results demonstrated no significant difference in performance times or lactate accumulation between trials. Post-exercise ammonia levels decreased in the 50 and 100 m trials in the Cr group and in the 50 m trial in the placebo. It was concluded that Cr supplementation cannot be considered an ergogenic aid for sprint performance in highly trained swimmers.

In a study by Rossiter and colleagues (1995), two matched groups of 19 competitive rowers followed five days of Cr or placebo supplementation. The Cr group received 0.25 grams of Cr per kg of body weight. After supplementation with placebo, no changes in the 1000 m rowing performance were noted. In contrast, 16 of 19 subjects in the Cr group improved their performance times. The mean improvement in rowing performance

for the Cr group was 2.3 s (211 ± 21.5 s versus 208.7 ± 21.8 s) ($p<.001$), an overall improvement of 1% for the Cr group. Rossiter and associates concluded that for competitive rowers, Cr provided a positive, though statistically non-significant, ($r = 0.426$) relationship with 1000 m rowing.

Gordan et al. (1995) supplemented 17 chronic heart failure patients with 20 grams of Cr daily for 10 days with a double-blind placebo-controlled design. Before and on the last day of supplementation, left ventricular ejection fraction (LVEF) was measured, as was one-legged and two-legged knee extensor performance on a cycle ergometer. No changes were revealed in the placebo group compared to baseline. In contrast, increases were observed for total Cr and PCr, one-legged (21% $p<.05$) and two-legged (10% $p<.05$) performance, and peak torque (5% $p<.05$) for the Cr group. The increments in one-legged, two-legged, and peak torque were significant when compared to the placebo group ($p<.05$). However, LVEF was unaffected.

Based on these studies there appears to be some conflicting evidence as to the effectiveness of Cr supplementation on performance. Athletic performance usually consists of several parameters, which tend to be difficult to measure, while increases in specific variables, such as isometric contractions, demonstrate more discernable improvements. Further research should focus on well-trained subjects and the application of

small performance gains through Cr supplementation to their particular sport.

Side Effects

Studies examining Cr feeding date back approximately 100 years. The only documented side effect associated with Cr supplementation is an increase in body mass. However, in those studies which reported significant weight gain, the consumption of Cr was much greater than that obtained in a normal mixed diet (Balsom, Ekblom et al., 1993; Balsom, Harridge et al., 1993; Earnest et al., 1995; Greenhaff et al., 1994; Soderlund et al., 1994). Also, most of the published studies consisted of a relatively short duration in Cr supplementation, less than one week. In a long term study, Cr supplementation was maintained over one year, at a dose similar to that which is consumed in a normal mixed diet (approximately one gram) (Sipila, Rapola, Simell, & Vannas, 1981). There is little data concerning side effects of long term Cr supplementation.

In further support of the association between Cr supplementation and increased body mass, Earnest et al. (1995) supplemented four weight trained male subjects with 20 grams of Cr a day for 28 days. Results revealed an increase in body mass from 86.5 $\pm$ 13.7 to 88.2 $\pm$ 14.1 kg for the Cr group. Other studies revealed increased body mass associated with short term

Cr ingestion (Balsom, Ekblom et al., 1993; Balsom, Harridge et al., 1993; Greenhaff et al., 1994; Soderlund et al., 1994). In these studies Cr supplementation consisted of 20-30 grams per day for 5-7 days. Body mass increases were very similar to those obtained by Earnest et al. (1995).

Interestingly, the composition of the weight gain in most of these studies was not determined, with the exception of Earnest et al. (1995). Through hydrostatic weighing, no differences were noted in lean body mass or percent body fat. Therefore, questions arise concerning body composition related to the increases in body mass for the other studies, as well as the mechanism behind the increased body mass.

Balsom, Ekblom et al. (1993) postulated that the increased weight gain may be related to an increased diameter of FT muscle fibers, based on the work by Sipila et al. (1981). Sipila and colleagues demonstrated that long term, low dose Cr supplementation increased the muscle diameter of FT muscle fibers in patients with gyrate atrophy.

Another theory by Balsom, Ekblom et al. (1993) is that Cr increases the rate of protein synthesis. The involvement of Cr with protein synthesis is supported by the work of Ingwall (1976), who suggested Cr may be the chemical signal coupling increased muscular activity to increased contractile protein synthesis in hypertrophy.

Bessman and Savabi (1990) proposed that exercise acts to stimulate protein synthesis by the increased contractile activity which causes more transport of PCr. During muscle contraction Cr is liberated at utilization sites and returns to the mitochondria to be rephosphorylated to PCr, at the same time stimulating oxidative phosphorylation. Also, Cr can diffuse to ATP producing sites of glycolysis, thus stimulating the anaerobic production of ATP. The PCr created in these pathways can be utilized for all endergonic functions such as contraction, protein synthesis, and ion transport.

Haussinger and Lang (1991) and Haussinger, Roth, Lang, and Gerok (1993) suggested that Cr may act indirectly by increasing the hydration status of the cell. They concluded that due to the osmotically active nature of Cr, an increase of intracellular Cr may induce swelling. Haussinger postulated that this may be an anabolic proliferation signal which may translate into increased lean body mass over time in healthy, resistance trained individuals.

It is clear that several mechanisms are functioning as body mass increases with Cr supplementation. However, additional research to obtain the exact means responsible for body mass increases should be completed. In the future, studies must determine the composition of the observed weight gain in order to compile more meaningful data.

Ergogenic Aid

The beneficial effects associated with Cr supplementation appear to be an increase of total Cr and PCr content, and an enhanced ability to resynthesize PCr during periods of recovery. These effects are supported by Harris et al. (1992), which were then confirmed by several other studies (Gordan et al., 1995; Greenhaff, Boden et al., 1994; Balsom et al., 1995). In contrast to these studies, Odland et al. (1997) concluded that no difference existed between Cr, placebo, or control groups for variables of ATP, total Cr, or PCr. At this time only two studies have focused on PCr resynthesis (Greenhaff, Bodin et al., 1993; Greenhaff et al., 1994). Each revealed that after two minutes of recovery, PCr resynthesis had been increased between 20-42%.

The athletes most likely to gain from Cr supplementation are those who participate in anaerobic sports, of high-intensity, short duration (Balsom et al., 1993; Birch et al., 1994; Earnest et al., 1995; Greenhaff, Casey et al., 1993; Harris et al., 1993; Soderlund et al., 1994). Again, studies of similar methods demonstrated non-significant effects of Cr supplementation on performance (Balsom et al., 1995; Cooke et al., 1995; Mujika et al., 1996; Odland et al., 1997; Rossiter et al., 1995).

Another factor that may influence the effect of Cr upon athletic performance is the basal levels of muscle Cr. Greenhaff (1995) demonstrated that individuals with lower basal muscle Cr content are more likely to increase performance than those with already high concentrations of muscle Cr.

Again, any athlete involved in a sport which utilizes PCr as an energy source, especially those of intermittent nature, may benefit from elevated Cr stores. Cr supplementation may also enhance the quality of the athlete's training session, which could transfer to in-sport performance enhancements. Finally, the increase in body mass related to Cr supplementation may be beneficial to those athletes requiring strength and power.

CHAPTER III

METHODS

Prior to the collection of data, approval from the Marshall University Institutional Review Board for Human Investigation was obtained. An informed consent was obtained from each subject. Statements within the informed consent form described the benefits and risks associated with this study. All subjects were informed that they have the right to terminate participation at any time during the study. To indicate understanding of the contents within the informed consent each subject was asked to sign the form as required by the Institutional Review Board.

SUBJECTS

Subjects were selected from area high school and college football programs. Selection was limited to those players who had been participating in a strength and conditioning program prior to the study. Furthermore, any players who had been consuming creatine or any derivatives of creatine within one month of the study were refused.

Initially, 33 well-trained football players, aged 15-24 served as subjects. Each subject reported to the Marshall University Human Performance Laboratory where they were

hydrostatically weighed. A double-blind design was employed, at which time 33 subjects were each randomly assigned to one of three treatment groups according to lean body mass (LBM). No significant difference existed between the LBM among the treatment groups. However, due to injury and general attrition rate, a total of 21 subjects completed the required training and/or testing.

PROCEDURES AND INSTRUMENTATIONS

Prior to creatine monohydrate supplementation, each subject reported to the Marshall University P-SET laboratory for pre-treatment data collection.

Test Protocols

Before testing began each subject was allowed to familiarize himself with the testing protocols. The investigator clearly explained each phase of the testing prior to data collection. The subjects were reminded that they could stop the test at any time. Each test was performed before and after the treatment period of 17 days.

Wingate. To test anaerobic power, two consecutive 30 s Wingate bike tests (utilizing the SMI Opto-Sensor by Sports Medicine Industries) were completed interspersed with five minutes rest (Bar-Or, 1987). Peak anaerobic power was defined as the greatest number of revolutions (R) per 5 s interval. Total anaerobic work was defined as the greatest number of revolutions over a 30 s time period for a constant kilopond force setting. Mean anaerobic power was calculated by averaging all one second power values over the entire 30 s test. Initial workload setting was determined by multiplying the total body weight by .075. Also, percent power decline was calculated as:

(highest # of R - lowest # of R) / highest # of R * 100

Sprint Test. Each athlete performed two sets of sprints over a distance of 100 yards (times for 40, 40-100 split, and 100 yards were measured) with five minutes rest between each set. Tests were completed at the Marshall University football stadium (artificial surface), Marshall University track, and the Fairland High School track. Sprint time were measured using a timing device called the "Speed Trap II"(Brower Timing Systems).

Body Composition. Body density was measured through a hydrostatic weighing technique utilizing a Chatillon scale. Percent body fat was calculated using the Siri equation (Siri, 1961).

Muscular Strength and Endurance. To test muscular strength, one repetition maximum (1RM) for the bench press was used. The subjects were asked to approximate their previously determined 1RM and that weight was placed on the bar. If that weight was lifted more than once, a formula to predict their 1RM was utilized (Hoeger, Barette, Hale, Hopkins, 1987). To determine muscular endurance, a technique from Earnest and colleagues (1995) was used, in which 70% of the 1RM was lifted until fatigue occurred. Lifting cadence was paced through the use of a metronome. The metronome was set at 1 s intervals, allowing 1 s for both concentric and eccentric contractions. Fatigue was defined as the inability to complete a repetition or the inability to maintain the lifting pace. Total lifting volume was calculated by multiplying 70% 1RM by the total number of repetitions completed by the distance measured from manubrium of the sternum to full extension of the bench press.

Treatments

Subjects received creatine monohydrate + dextrose (CM+D), creatine monohydrate (CM), or a placebo (P) over a 17 day period. A dosage of 43 grams of CM+D (5 grams Cr + 35 grams dextrose + 3 grams taurine), 16 grams of CM (10 grams Cr + 6 grams glucose), or 35 grams of P (glucose was used as a placebo) was administered four times each day for five days. After five days of supplementation, the initial dosage was reduced to a maintenance dose consisting of one dose of either CM+D, CM, or P per day for the remainder of the study. Subjects reported to the head athletic trainer who randomly assigned each athlete to a treatment using a chart of randomization. Also, the athletic trainer supervised the administration of CM+D, CM, and P.

In addition, subjects participated in a Marshall University football weight training program on Monday, Wednesday, and Friday. On Tuesday and Thursday a sprint training program for Marshall University football program was administered to all the subjects. Recovery periods for the subjects fell on Saturday and Sunday.

DESIGN AND ANALYSIS

A two-way analysis of variance (ANOVA) for repeated measures was employed to determine the differences between the means for the treatments of CM+D, CM, and P (Keppel, 1982). A Crossover technique was not used since the rate of recovery and effect of creatine supplementation is not known. For demographic data a one-way ANOVA was utilized to determine if significant differences were present between groups, especially for LBM (Rothstein, 1985). A student's t-test for unequal population was employed to determine differences between groups after matching for LBM was eliminated and the two Cr groups were combined (Rothstein, 1985).

CHAPTER IV

RESULTS

As previously stated, all subjects were hydrostatically weighed and matched according to LBM prior to random assignment in treatment groups. An ANOVA was employed for the demographic data (age, height, and weight, as well as LBM). No significant differences were noted between treatment groups (Table 1).

Several modes of exercise were utilized in this study which mimic the components of performance as applied to football. As a result of a number of subjects failing to complete the required training and/or testing, differences in population size exist for each exercise mode (Table 2).

A two-way ANOVA for repeated measures was employed for the variables of the Wingate cycle ergometer test, sprint test, muscular strength and endurance test, and hydrostatic weighing. Cr supplementation was found to have no significant ($p<.05$) effect upon any of these variables.

Wingate

No significant ($p<.05$) differences were observed in performance variables of trial one or two for the Wingate cycle ergometer tests between treatment groups. However, the effects

TABLE 1

DEMOGRAPHIC DATA

	CM	*CM + D*	*P*	*F RATIO*
	mean (SD)	mean (SD)	mean (SD)	mean (SD)
AGE	18.86 (2.47)	18.86 (2.47)	18.28 (2.65)	0.0061781
HEIGHT (cm)	179.57 (7.25)	179.26 (12.69)	185.43 (3.18)	0.0076492
WEIGHT (kg)	89.21 (20.36)	90.77 (18.56)	92.74 (21.16)	0.0068353
LBM (kg)	74.06 (11.65)	74.94 (11.51)	75.00 (11.03)	0.0009767

no significant differences between groups

TABLE 2

TOTAL POPULATION DIFFERENCES

FOR EACH EXERCISE MODE

PERFORMANCE TEST	*N*	*VARIABLES*
WINGATE	12	PEAK ANAEROBIC POWER TOTAL ANEROBIC WORK MEAN ANEROBIC POWER %DECLINE
SPRINT	15	40 YARD 100 YARD 40-100 SPLIT
MUSCULAR STRENGTH AND ENDURANCE	18	1 R M TOTAL LIFTING VOLUME
BODY COMPOSITION	21	LBM BODY MASS % FAT

of training were found to be significant for peak anaerobic power (trial one), total anaerobic work (trials one and two), and mean anaerobic power (trial two). The means and associated F ratios are presented in Tables 3-6. Also, comparisons of means for these variables are illustrated in Figures 1-4.

Sprint Test

The effects of training were significant for trial one of the 100 yard dash and 40-100 yard split. Tables 7 and 8 provide the means and F ratios for these variables.

Body Composition

The variables of LBM, body mass, and percent fat were all significantly affected through training. Means and F ratios are presented in Tables 9-11, while comparisons are made for each variable in Figures 5-7.

Muscular Strength and Endurance

Both 1RM and total lifting volume were significantly affected by training (Tables 12 and 13). However, after determining that no significant difference in LBM existed between treatment groups, matching accordingly was eliminated. Data from several subjects had to be excluded because a match in another treatment group had failed to complete the required testing.

TABLE 3

PEAK ANAEROBIC POWER-TRIAL 1

	SS	*DF*	*MS*	*F*
TREATMENT	5.2225	2	2.61125	1.523828822
ERROR	15.4225	9	1.713611111	
TRAINING	3.08166667	1	3.08166667	15.0121786*
TRAINING TREATMENT INTERACTION	0.45083333	2	0.225416665	1.09810554
ERROR	1.8475	9	0.205277778	
TOTAL	26.025			

*Significant at $p < .01$

TABLE 4

TOTAL ANAEROBIC WORK-TRIAL 1

	SS	*DF*	*MS*	*F*
TREATMENT	34378.0833	2	17189.0417	0.437544258
ERROR	353567.375	9	39285.2639	
TRAINING	33975.375	1	33975.375	27.6001286*
TRAINING TREATMENT INTERACTION	3908.25	2	1954.125	1.58744683
ERROR	11078.875	9	1230.98611	
TOTAL	436907.958			

*Significant at $p < .001$

TABLE 5

TOTAL ANAEROBIC WORK-TRIAL 2

	SS	*DF*	*MS*	*F*
TREATMENT	44042.3333	2	22021.1667	0.732354135
ERROR	270621.125	9	30069.0139	
TRAINING	16485.0417	1	16485.0417	5.5076355*
TRAINING TREATMENT INTERACTION	83.3333333	2	41.6666667	0.013920791
ERROR	26938.125	9	2993.125	
TOTAL	358168.958			

*Significant at $p < .05$

TABLE 6

MEAN ANEROBIC POWER-TRIAL 2

	SS	*DF*	*MS*	*F*
TREATMENT	21895.0833	2	10947.5417	1.256534761
ERROR	78412.375	9	8712.48611	
TRAINING	41583.375	1	41583.375	7.23624354*
TRAINING TREATMENT INTERACTION	7803.25	2	3901.625	0.678951833
ERROR	51718.875	9	5746.54167	
TOTAL	201412.958			

*Significant at $p < .05$

FIGURE 1

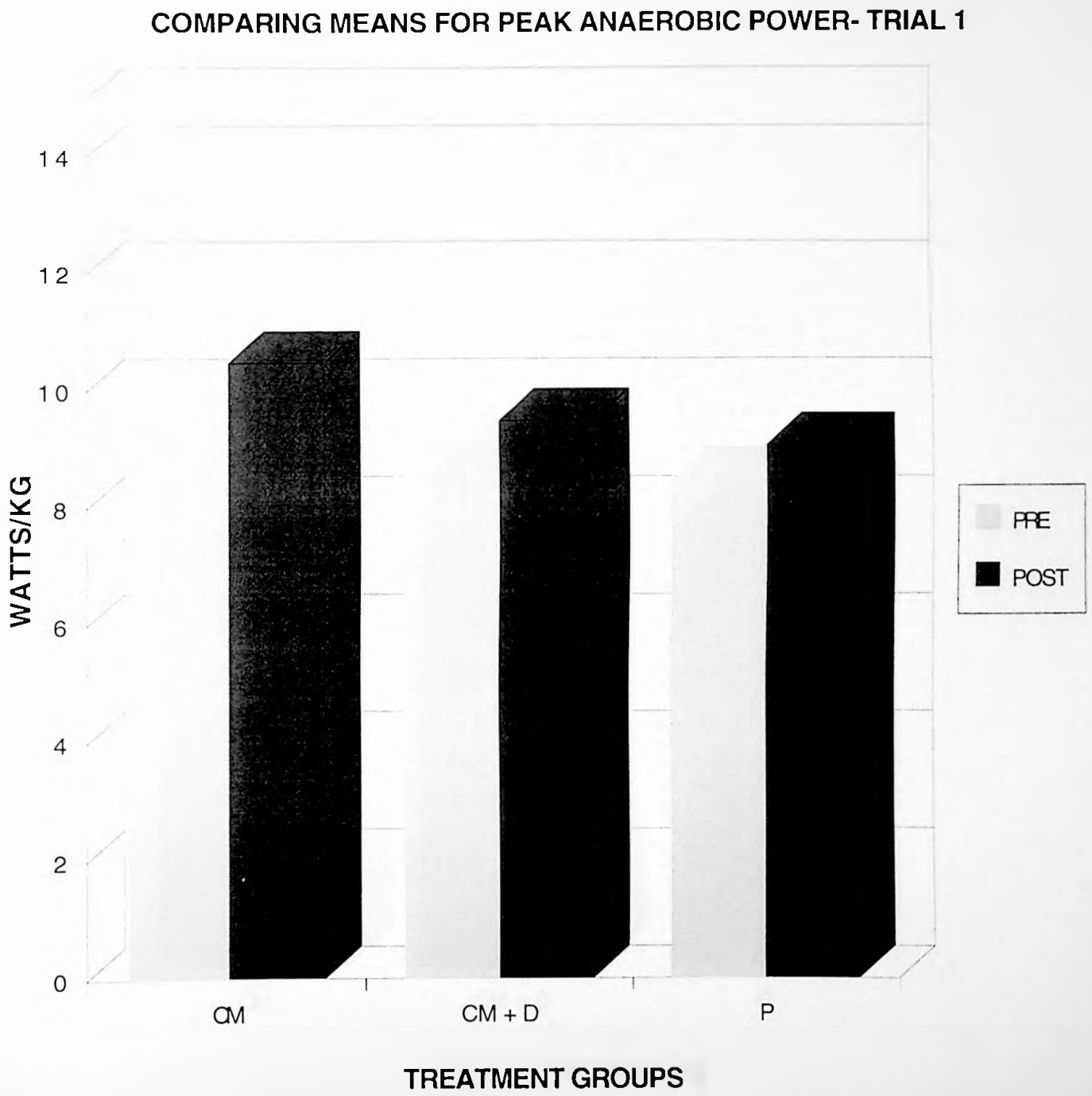

COMPARING MEANS FOR PEAK ANAEROBIC POWER- TRIAL 1
14
12
10
8
6
4
2
0
WATTS/KG
CM
CM + D
P
PRE
POST
TREATMENT GROUPS

FIGURE 2

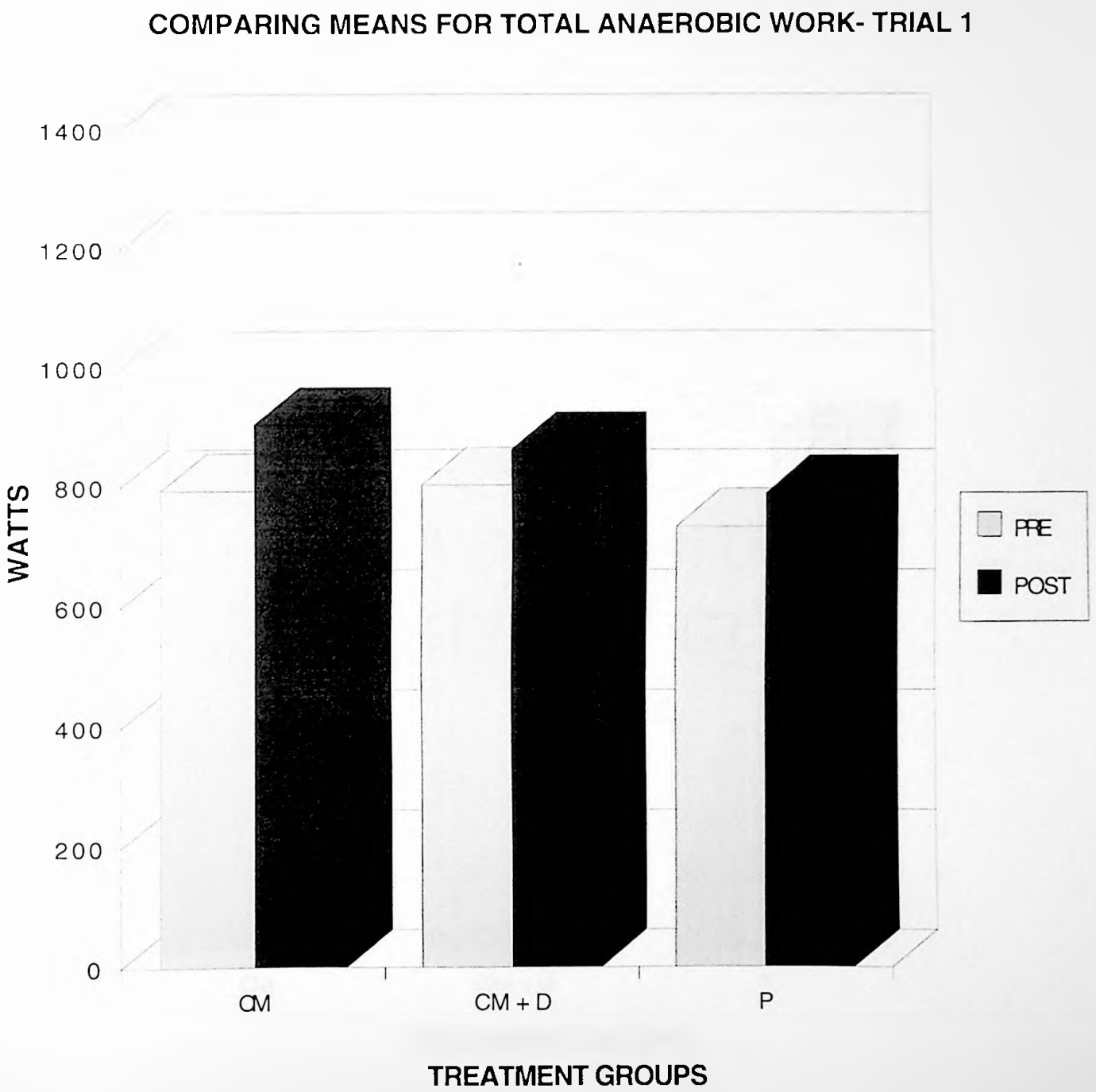

COMPARING MEANS FOR TOTAL ANAEROBIC WORK- TRIAL 1
1400
1200
1000
800
600
400
200
0
WATTS
CM
CM + D
P
PRE
POST
TREATMENT GROUPS

FIGURE 3

COMPARING MEANS FOR TOTAL ANAEROBIC WORK- TRIAL 2

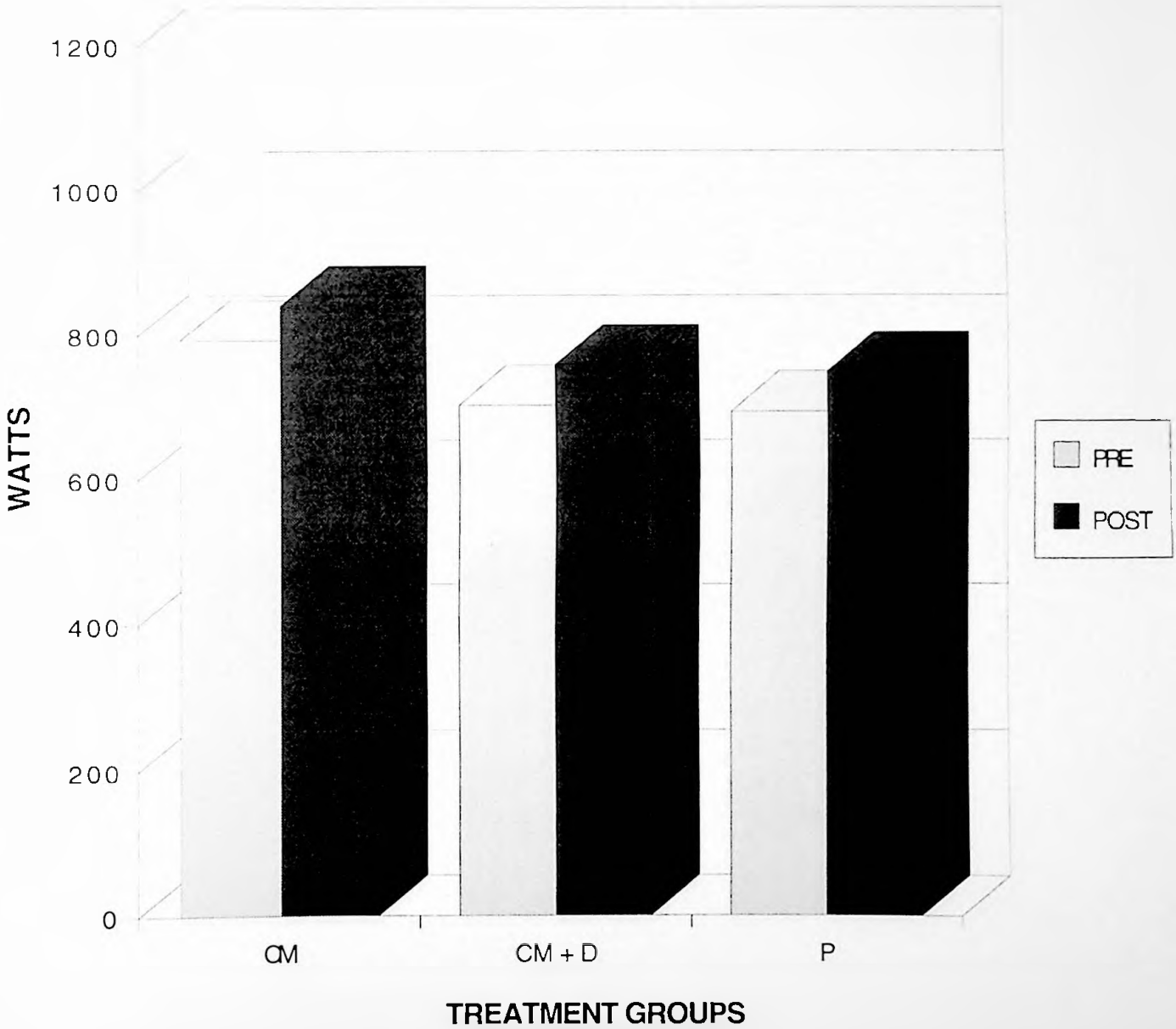


FIGURE 4

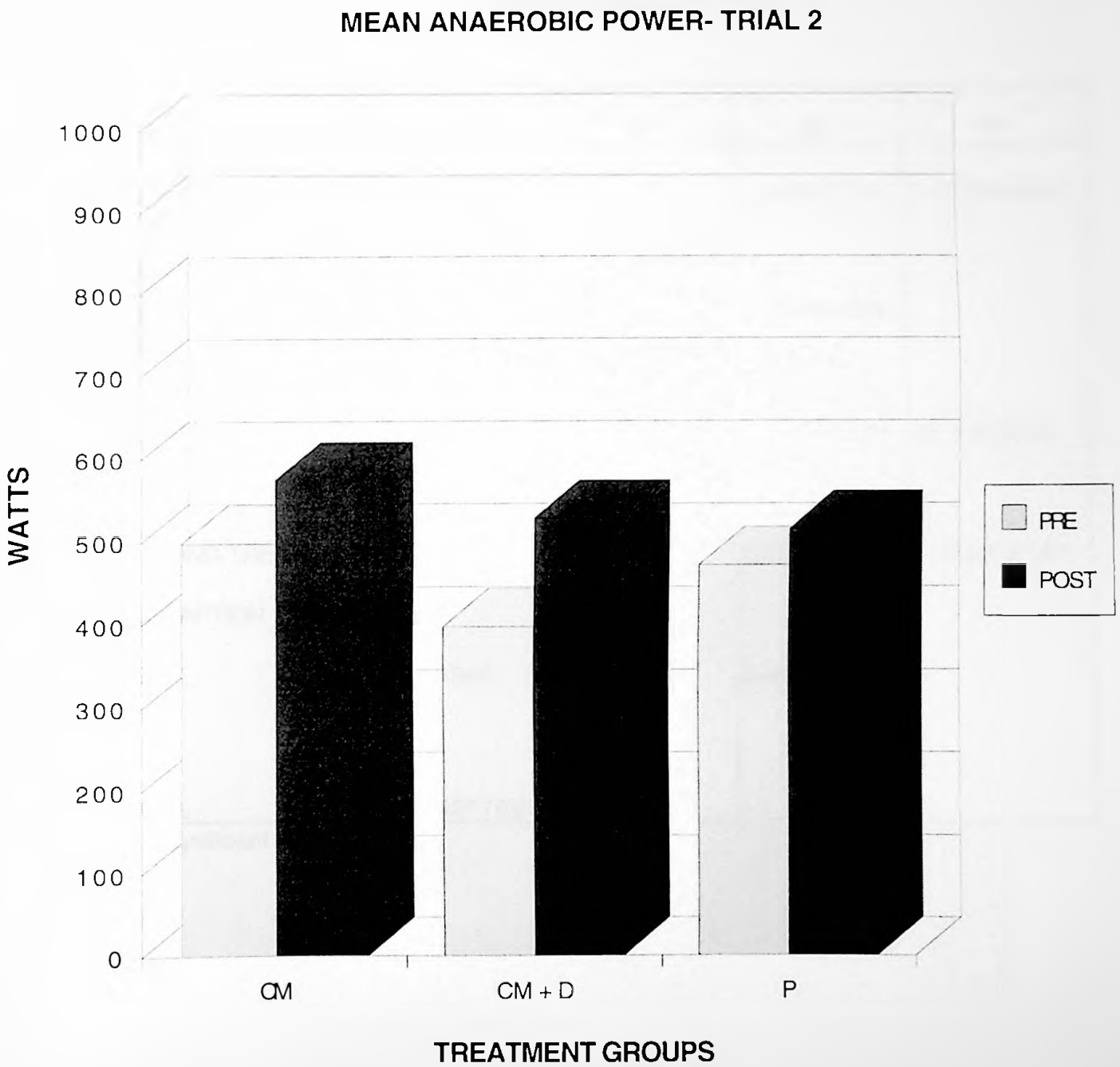

MEAN ANAEROBIC POWER- TRIAL 2
1000
900
800
700
600
500
400
300
200
100
0
WATTS
CM
CM + D
P
TREATMENT GROUPS
PRE
POST

TABLE 7

100 YARD SPRINT-TRIAL 1

	SS	*DF*	*MS*	*F*
TREATMENT	2.68000667	2	1.34000334	1.222242324
ERROR	13.15618	12	1.09634833	
TRAINING	1.03045333	1	1.03045333	22.3420663*
TRAINING TREATMENT INTERACTION	0.10168667	2	0.05084334	1.102374191
ERROR	0.55346	12	0.04612167	
TOTAL	17.5217867			

*Significant at p < . 001

TABLE 8

40-100 SPLIT-TRIAL 1

	SS	*DF*	*MS*	*F*
TREATMENT	1.15862	2	0.57931	1.254877468
ERROR	5.53976	12	0.46164667	
TRAINING	0.79056333	1	0.79056333	35.3930757*
TRAINING TREATMENT INTERACTION	0.04284667	2	0.02142334	0.959110655
ERROR	0.26804	12	0.02233667	
TOTAL	7.79983			

*Significant at p < . 001

TABLE 9

LEAN BODY MASS

	SS	*DF*	*MS*	*F*
TREATMENT	16.9328571	2	8.46642855	0.005697976
ERROR	26745.5886	18	1485.86603	
TRAINING	293.885952	1	293.885952	70.3584078*
TRAINING TREATMENT INTERACTION	9.08333333	2	4.54166667	1.087307619
ERROR	75.1857143	18	4.17698413	
TOTAL	7.79983			

*Significant at p < . 001

TABLE 10

BODY MASS

	SS	*DF*	*MS*	*F*
TREATMENT	72.8842857	2	36.4421429	0.038921135
ERROR	16853.5314	18	936.3073	
TRAINING	26.0859524	1	26.0859524	50.0583308*
TRAINING TREATMENT INTERACTION	2.02904762	2	1.01452381	1.946847397
ERROR	9.38	18	0.52111111	
TOTAL	16963.9107			

*Significant at p < . 001

TABLE 11

PERCENT FAT

	SS	*DF*	*MS*	*F*
TREATMENT	24.6539476	2	12.3269738	0.099155264
ERROR	2237.75844	18	124.319913	
TRAINING	11.0572024	1	11.0572024	9.87518225*
TRAINING TREATMENT INTERACTION	6.57091905	2	3.28545953	2.934242354
ERROR	20.1545286	18	1.11969603	
TOTAL	2300.19504			

*Significant at p < . 01

FIGURE 5

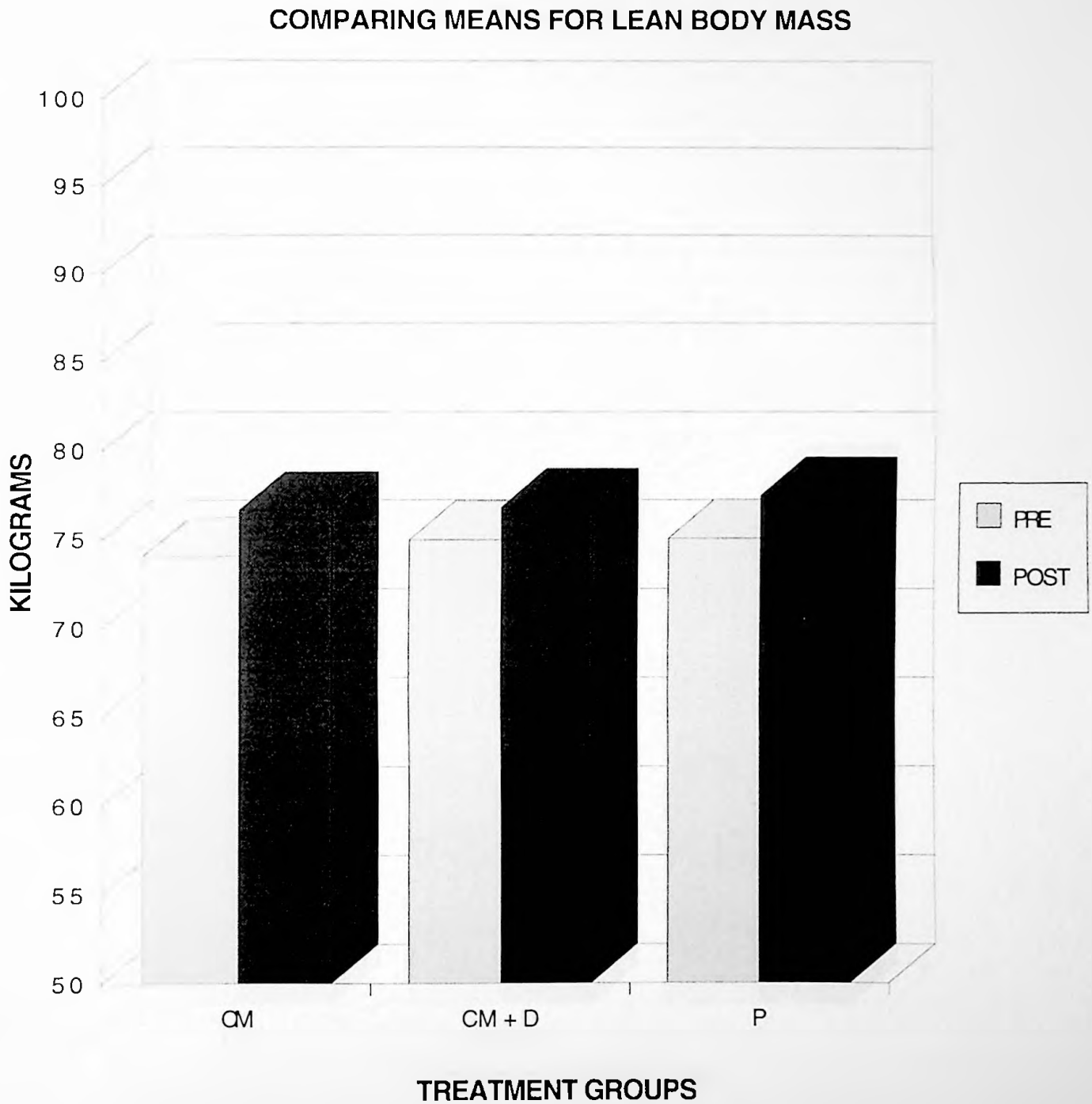

COMPARING MEANS FOR LEAN BODY MASS
100
95
90
85
80
75
70
65
60
55
50
KILOGRAMS
CM
CM + D
P
TREATMENT GROUPS
PRE
POST

FIGURE 6

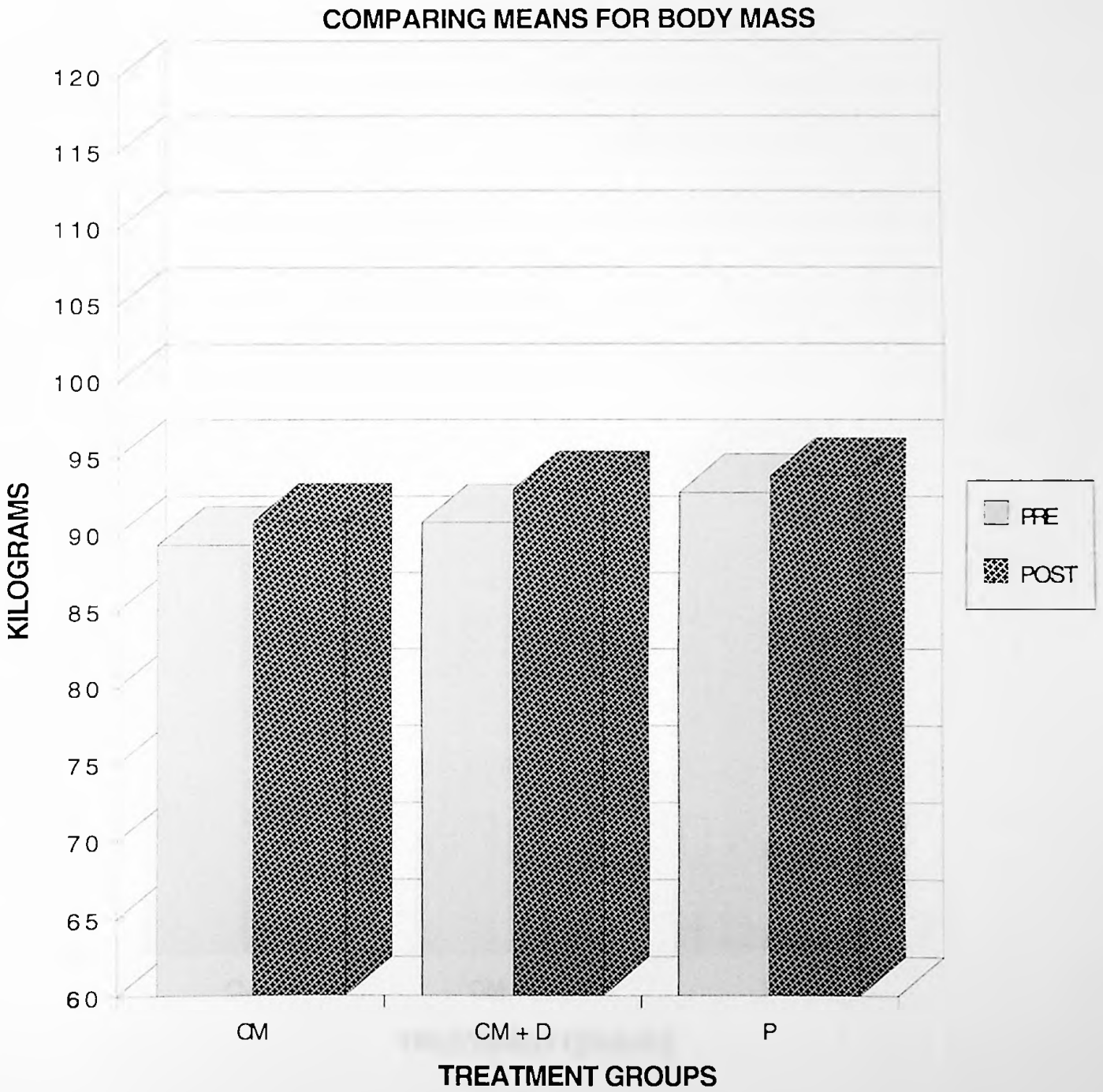

COMPARING MEANS FOR BODY MASS
120
115
110
105
100
95
90
85
80
75
70
65
60
KILOGRAMS
CM
CM + D
P
TREATMENT GROUPS
PRE
POST

FIGURE 7

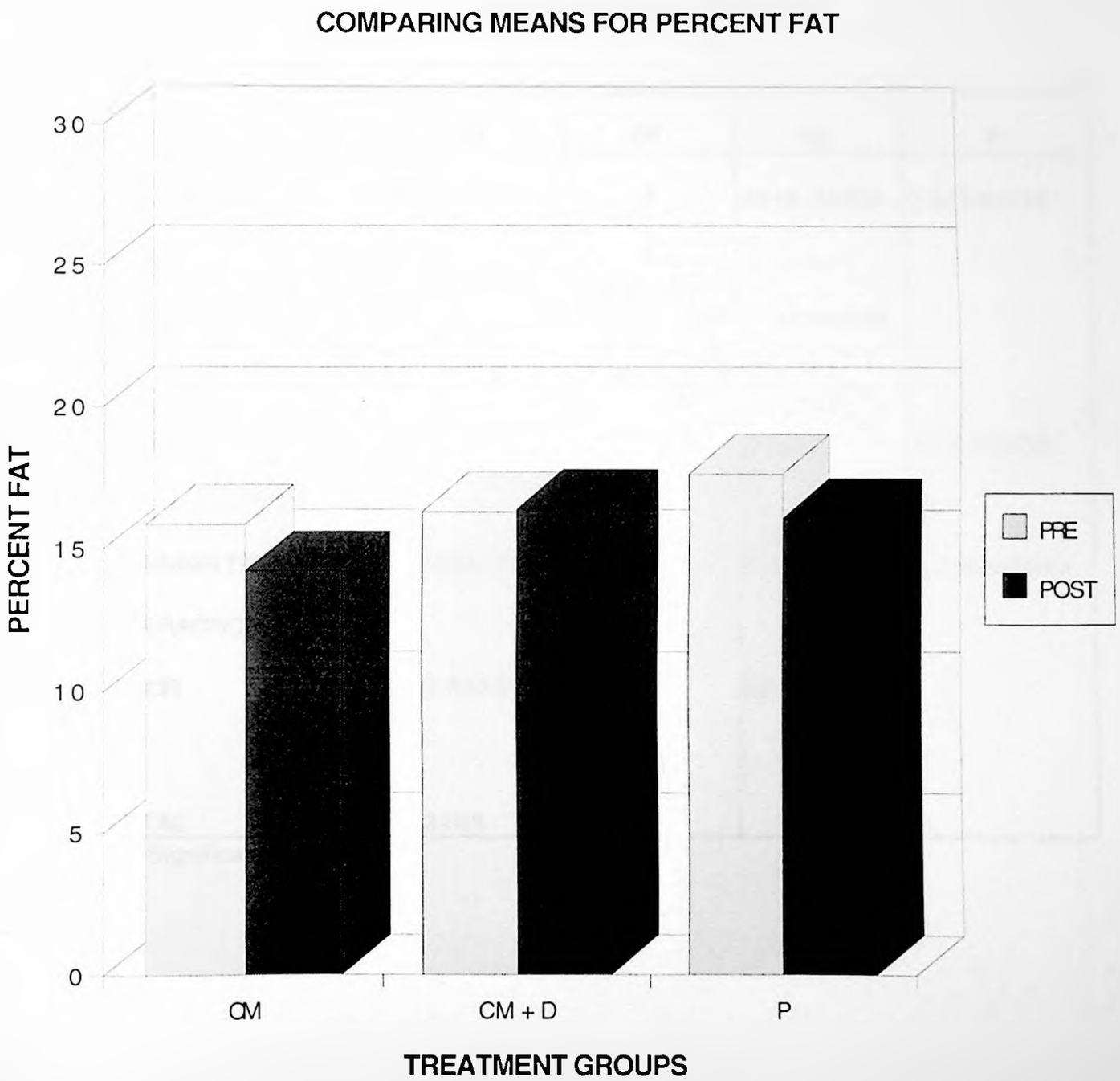

COMPARING MEANS FOR PERCENT FAT
30
25
20
15
10
5
0
PERCENT FAT
PRE
POST
CM
CM + D
P
TREATMENT GROUPS

TABLE 12

ONE-REPETITION MAXIMUM

	SS	*DF*	*MS*	*F*
TREATMENT	9837.16667	2	4918.58334	0.42882198
ERROR	172049.833	15	11469.9889	
TRAINING	1111.11111	1	1111.11111	42.2119038*
TRAINING TREATMENT INTERACTION	15.0555556	2	7.5277778	0.285985649
ERROR	394.833333	15	26.3222222	
TOTAL	183408			

*Significant at $p < .001$

TABLE 13

TOTAL LIFTING VOLUME

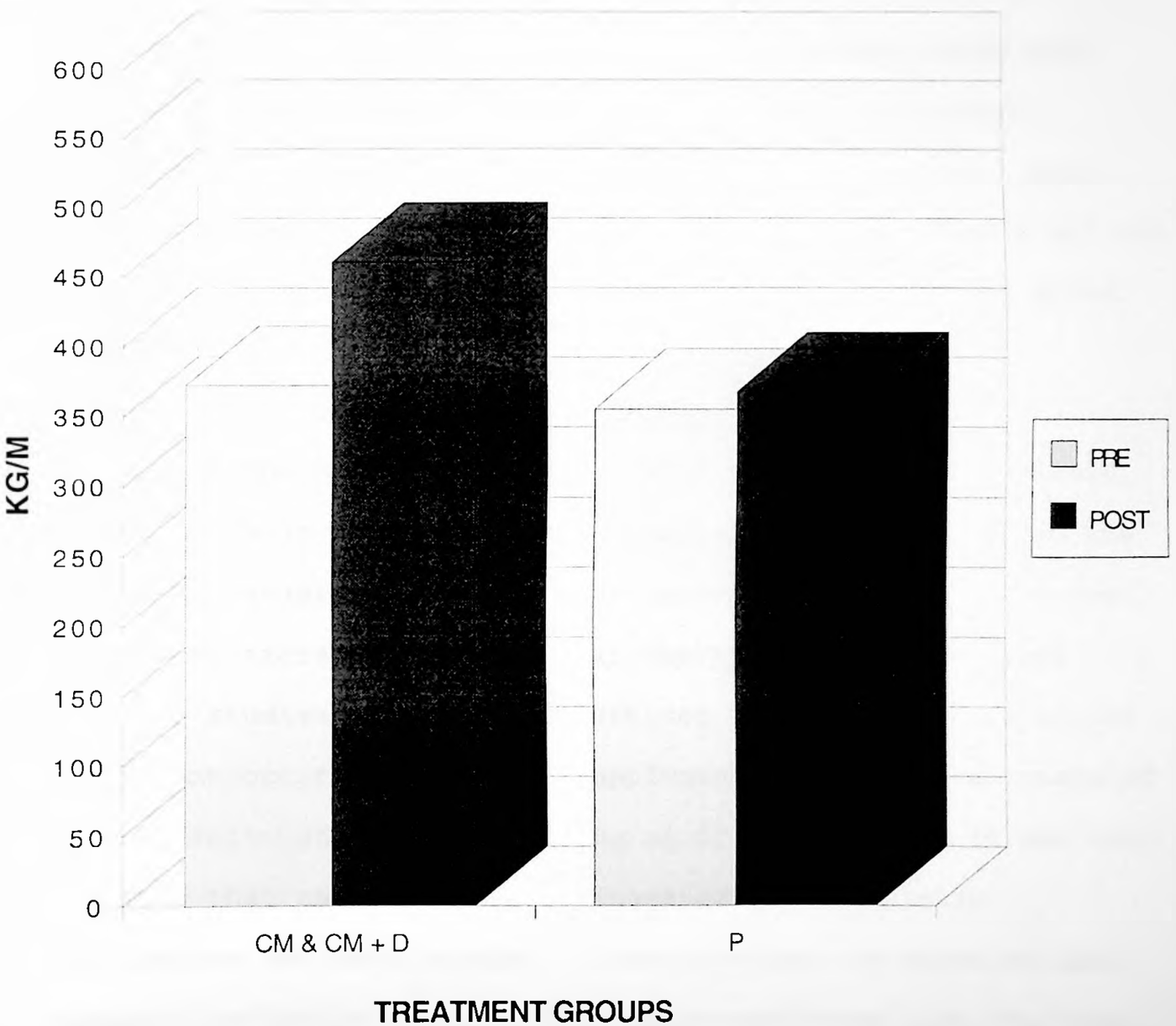
	SS	*DF*	*MS*	*F*
TREATMENT	45418.1667	2	22709.0834	1.156110655
ERROR	294639.833	15	19642.6555	
TRAINING	25921	1	25921	6.20982919*
TRAINING TREATMENT INTERACTION	12882.1667	2	6441.08335	1.543074241
ERROR	62612.8333	15	4174.18889	
TOTAL	441474			

*Significant at $p < .025$

After inclusion of these data, the two Cr groups were combined resulting in one Cr group consisting of 15 subjects and a placebo with 8 subjects. The purpose was to increase the total population of the study which also enhanced the statistical power. At this point, a student's t-test for unequal populations was employed for several performance variables. For one variable, total lifting volume, the effects of Cr significantly ($p<.05$) enhanced performance. Figure 8 illustrates differences in the means with standard deviations.

FIGURE 8

COMPARING MEANS FOR TOTAL LIFTING VOLUME



Chapter V

DISCUSSION, CONCLUSION, AND RECOMMENDATIONS

This chapter discusses results of the present study and relates them to the current literature. Upon contrasting outcomes, inferences are made as to why these occurred. Also, conclusions about the effectiveness of Cr as an ergogenic aid are presented. Finally, suggestions for future research are given.

DISCUSSION

Since Chevreul (1835) first identified Cr in meat extract, many studies on Cr have followed (Balsom et al., 1994). In the beginning, studies focused on the amount of Cr consumed versus the amount excreted. However, at the turn of the twentieth century, studies on animals determined that increases in muscle Cr content occurred following supplementation. Several years of studies followed, mainly focusing on Cr metabolism. It was only recently that the influence of ingested Cr on athletic performance has been studied. These studies are based on the research by Harris et al. (1992) which confirmed that the total content of Cr could be increased within skeletal muscle.

Currently, the majority of studies reporting performance enhancing effects of Cr supplementation have been completed on untrained subjects. A plausible explanation is that untrained individuals have not reached their physiological limitations for strength or power, whereas well-trained individuals are much closer to their genetic ceiling. Therefore, improvements in performance with untrained subjects may be attributable to the ergogenic effect, since they have not reached their genetic ceiling. Studies in which well-trained subjects were used have revealed mixed results. Balsom, Ekblom et al. (1993), Harris et al. (1993), and Earnest et al. (1995) demonstrated improvements in performance during cycle ergometry, short to middle distance running, and weight lifting. In contrast, Balsom, Harridge et al. (1993) and Mujika et al. (1996), revealed that no significant changes occurred after Cr supplementation during prolonged supra maximal running and swimming sprints.

In the present study, it was postulated that if performance improvements were detected in well-trained subjects, then the ergogenic effect is strong and can be generalized to the competitive athletic populations. Therefore, the present study was designed to determine if Cr supplementation could enhance strength and power performance, as measured by the Wingate cycle ergometer test, 40 and 100 yard sprints, and bench press, in well-trained football players.

Results from the present study indicate that 17 days (5 loading and 12 maintenance) of Cr supplementation had no significant effect ($p<.05$) on the performance variables as defined in this study. However, 17 days of training significantly affected several of these variables.

Wingate

In the present study, no significant change occurred as a result of Cr supplementation for total anaerobic work, peak anaerobic power, and mean anaerobic power during both trials of the Wingate cycle ergometer test. In contrast, Earnest et al. (1995) demonstrated significantly ($p<.05$) higher increases in total anaerobic work for trials one, two, and three of the Cr group. On the other hand, Birch et al. (1994) observed improvements in total anaerobic work for trials one and two, as well as improvements in peak anaerobic power for trial one, and mean anaerobic power in trials one and two.

A possibility as to why the results of the present study contradict those of Earnest et al. (1995) and Birch et al. (1994) may be related to the Wingate protocol. Initially, three Wingate cycle ergometer tests were to be used in the present study. However, during several pilot tests, the subjects could not complete three trials due to nausea, vomiting, lightheadedness, and extreme fatigue. As a result of these problems, two trials

of Wingate tests with 5 minutes recovery were selected for the present study. Since there were no reports of nausea and vomiting in the Earnest et al. (1995) study over three trials, we question their Wingate protocol. Our subjects were instructed to give an all-out effort for all trials. When maximal effort is given, it is common to experience nausea and vomiting after a single trial of the Wingate. This precludes multiple trials for approximately 30% of the subjects. Subsequently, it is difficult to achieve maximal work output over two or three trials. To complete multiple trials, subjects would have to pace themselves across trials. This procedure, if followed by Earnest et al. (1995), would explain the conflict in the results.

In similar studies, Cooke et al. (1995) and Odland et al. (1997) supported our findings by demonstrating no significant differences in peak anaerobic power, total anaerobic work, and percent decline during Cr supplementation. However, these studies were less similar than Earnest et al. (1995) when compared to the present study. Cooke et al. (1995) performed two consecutive 15 s maximal sprints on a cycle ergometer interspersed with 20 minutes rest, while Odland et al. (1997) utilized only one 30 s Wingate test.

Sprint Test

Cr supplementation had no significant effects ($p<.05$) upon repeated sprint performances over distances of 40, 40-100 yard split, and 100 yards. Normally, without Cr supplementation, PCr stores totally resynthesize after 5 minutes of recovery Bogdanis et al., 1993; Soderlund & Hultman, 1991). Theoretically, through Cr supplementation, levels of total Cr and PCr should be increased (Gordan et al., 1995; Greenhaff et al., 1994; Harris et al., 1992; Balsom et al., 1995). If these two scenarios occurred, then the 5 minutes of recovery given between trials for both, sprinting and the Wingate, should have more than adequately restored total Cr as well as PCr content. This would have allowed for quicker recovery and greater utilization of PCr, which should have delayed fatigue in the Cr groups, especially for the second trials of the 100 yard sprint and Wingate test. In support of the present study, Odland et al. (1997) demonstrated that through Cr supplementation of 20 grams a day for three days no differences existed between Cr, placebo, or control for PCr or total Cr, which translated into no improvements in performance for one trial of high-intensity exercise.

The use of 100 yard sprints was intended to be specific for the sport of football. The data clearly show that Cr supplementation had no significant effect for the 100 yard

sprint; however, in another study sprints of longer duration (300 m and 1000 m) were improved via Cr supplementation (Harris et al., 1993). One explanation for this is that the distance of 100 yards is too short. PCr does not become totally depleted until approximately 10 s; the time range for completion of the 100 yard sprint for all groups combined in the present study was 11.9-13.0 s. For some well-trained athletes, anaerobic conditioning may allow longer periods of exercise before the depletion of PCr. Therefore, sprints of 300 to 1000 m may tax the rate of PCr decline to a greater extent. Because of the duration of these sprints, added total Cr and PCr through supplementation may be beneficial to performance. Again, it should be noted that the 100 yard sprint consisted of multiple trials of all-out effort. This presents the same methodological problems associated with effort dependent tests of maximal effort across trials.

Body Composition

Lean body mass (LBM), body mass, and percent fat were not significantly affected ($p<.05$) by Cr supplementation. This finding is in contrast to the studies of Balsom, Ekblom et al. (1993), Balsom, Harridge et al. (1995), Balsom, Soderlund, Sjordin et al. (1993), Earnest et al. (1995), Greenhaff et al. (1994), Mujika et al. (1996), and Soderlund et al. (1994).

All of these studies demonstrated significant increases in body mass; however, these studies were of relatively short duration (consisting of approximately one week). Only Earnest et al. (1995) determined the composition of the weight gain over Cr supplementations periods greater than one week (28 days). In support of the present study, Earnest et al. (1995) demonstrated no significant differences in LBM and percent fat in Cr versus the placebo groups. Also, the majority of studies in which increases in body mass were apparent after Cr supplementation also demonstrated increases in total Cr and PCr resynthesis. Mujika et al. (1996) did not measure muscle Cr and PCr content, but an increase in body mass was observed in the Cr group, at which time they concluded that through Cr supplementation the desired "Cr loading" had been achieved. Therefore, if total Cr and PCr levels are not increased, then the added body mass may also not increase.

Muscular Strength and Endurance

Prior to the elimination of matching for LBM, significant differences ($p<.05$) in the 1RM and total lifting volume were not apparent. However, when data from several excluded subjects were added and both Cr groups were combined, significant differences were observed in the Cr group for total lifting volume, versus the placebo (Figure 8, Chapter IV). This finding is supported by

Earnest et al. (1995), in which the total lifting volume increased 26% ($p<.05$) for the Cr group. In contrast to the present study, Earnest et al. (1995) also observed increases of 6% ($p<.05$) in 1RM for the bench press, while no differences were noted in either variables for the placebo group.

The demonstrated increase in total lifting volume, while increases in 1RM were not apparent, may also be supported in a study completed by Greenhaff, Casey et al. (1993), in which five trials of 30 maximal isometric contraction were used as variables. Results revealed that after Cr supplementation, the rate of peak torque decline was reduced during trials two, three, and four. Both studies, Earnest et al. (1995) and Greenhaff, Casey et al. (1993), concluded that a regimen of Cr supplementation intended to increase the availability of Cr, resulted in a greater ability to perform repeated trials of high-intensity exercise.

Finally, two factors which may have contributed to the results of the present study are the effort-dependent nature of the Wingate test and the 100 yard sprint, and levels of total Cr and PCr prior to Cr supplementation. During the pre-test Wingates, subjects experienced nausea, vomiting, lightheadedness, and extreme fatigue. Therefore, if subjects gave maximal effort and experienced uncomfortable symptoms, then efforts during the post-test may have been compromised to prevent experiencing the

symptoms that were common during pre-testing. Similarly, the pre-test 100 yard sprint induced muscle tightness and soreness, as well as overall fatigue. It can therefore be concluded that subjects may have given less effort during the post-test 100 yard sprint due to these factors. In contrast, the bench press tests are not particularly grueling and because of the emphasis placed on the 1RM value, subjects probably did not alter their effort between trials for this test.

Nutritional evaluations for subjects were not completed during the present study. Therefore, the types of food consumed by the subjects prior to and during the study is unknown. Theoretically, if the levels of total Cr and PCr were already high in the subjects due to diet or genetics, then the effects of supplemental Cr on total Cr, PCr, and performance could have been reduced. It has been demonstrated that subjects with lower basal levels of Cr are more likely to increase total Cr and PCr stores (Harris et al., 1992) as well as performance (Greenhaff, 1995) when compared to subjects with already high concentrations of muscle Cr.

CONCLUSION

The present study was designed to determine the effects of Cr supplementation on muscular strength and power development in well-trained football players. Initially, 33 area high school and college football players were selected as subjects and matched according to LBM. A double-blind design was employed, at which time the subjects were randomly assigned to one of the three treatment groups: CM; CM + D; and P. However, due to injury and general attrition rate only 21 subjects completed the required training and/or testing (see Table 2 for population size pertaining to each exercise mode). A five day loading dose, followed by a 12 day maintenance dose was ingested. Test protocols included three anaerobic work bouts: 1) Two 30 s Wingate cycle ergometer tests interspersed with 5 minutes recovery were used to determine relative and absolute power; 2) Two 100 yard sprints with 5 minutes rest between trials were used to ascertain times for 40, 40-100 split, and 100 yards; 3) 1RM and total lifting volume for the bench press were determinants of muscular strength and endurance. During the 17 days between pre and post-testing, all subjects participated in an intense program combining sprint and resistance training.

A two-way ANOVA for repeated measures revealed that Cr supplementation did not significantly ($p<.05$) improve variables for the Wingate test, sprint test, body composition, or muscular strength and endurance. However, the effects of training were significant ($p<.05$) for several variables (Tables 3-12, Chapter IV). After the elimination of matching, a student's t-test for unequal populations demonstrated that Cr significantly ($p<.05$) increased total lifting volume for the bench press. After examining the numerous variables, Cr supplementation did not significantly enhance performance as measured by the present study. Therefore, the null hypothesis that there will be no significant difference in the performance variables of strength and power between athletes receiving either type of Cr and athletes receiving placebo is not rejected. Also, investigators do not reject the null hypothesis that no significant changes will be found when comparing body composition variables between groups.

RECOMMENDATIONS

The use of Cr or any other nutritional supplement to enhance performance inevitably raises ethical issues. These substances are banned by governing bodies of sport for one of two reasons. They may pose a threat to the individuals health, or they create an unfair advantage for the individual. Although there is no

evidence to suggest that short-term Cr supplementation with high doses has any drastic health effects, studies examining the effects of long-term Cr supplementation are minimal at the present time. Also, due to the upper limit of Cr content and the presence of Cr in the diet, problems with detection and enforcement of certain Cr levels arise. Therefore, "policing" Cr supplementation in athletes will be very difficult.

In future studies, comparing dietary recall to initial muscle total Cr content may be beneficial, especially in specific populations (high dietary meat versus vegetarians). Also, to maximize the performance enhancing qualities of Cr, more studies need to use well-trained athletes. Increases in performance for this population can be highly attributable to Cr, since the physiological limitations for performance are more closely met in highly-trained subjects. Additional studies should focus on the exact mechanism by which body mass increases, as well as determining the composition of the increased body mass. Finally, the long-term effects of Cr supplementation and "Cr loading", similar to carbohydrate loading, may be of utmost importance for disease rehabilitation and performance enhancement.

In the present study, it may have been beneficial to select more area high school athletes. The majority of the college athletes were not highly motivated to participate in this study, which may be attributable to their lack of concern for factors

outside the guidelines of their scholarship. The high school athletes, on the other hand tended to be highly motivated to participate in both the testing and training. Again, effort-dependent tests were utilized for this study. If these types of tests are to be used, the subjects in the study must be highly motivated, either through self motivation or by the investigators, to participate (especially for post-testing). To enhance the effectiveness of the present study, a dietary recall could have been utilized in order to determine the amount of meat consumed in the diet, as well as if any dietary changes occurred in the course of the study. Due to these circumstances it remains unclear as to the effectiveness of Cr supplementation on muscular strength and power development in well-trained football players.

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APPENDIX A

Informed consent to participate in a research study on the effects of creatine supplementation on muscular strength and power development in post-pubescent football players

I understand that I am giving my consent to participate in this research study. I also understand that the purpose of this study is to determine the effects of creatine supplementation combined with a strength and sprint training program on muscular strength and power development of post-pubescent football players.

1. Procedures:

I realize that in order to determine the effects on performance, I must participate at a maximal level in four testing sessions. Two sessions will be held at the Marshall University Stadium, while the other two will be completed in Gullickson Hall. To be more specific, I will participate in the following procedures:

To determine anaerobic power, I will perform two consecutive Wingate bike tests at maximal effort. Each test will last for thirty seconds and I will receive five minutes recovery between tests.

To further examine anaerobic power, I will complete three sets of sprinting over distances of 40 and 100 yards interspersed with five minutes rest. I understand that the length of the tests will be dependent upon how long it takes me to complete each sprint.

To determine muscular strength, I will perform a one repetition maximum (1RM) for the bench press. To determine muscular endurance and work output, I will lift 70% of my 1RM for the bench press until I can no longer complete each repetition or until I cannot maintain lifting cadence. I understand that lifting cadence will be set through the use of a metronome, which will allow one second for concentric and one second for eccentric contractions. I understand that proper technique will be maintained throughout this test as determined by the strength coach and researchers.

_____ _____

Subject's Signature Date

_____ _____

Parent's or Guardian's Signature Date

_____ _____

Subject's Address Phone

To examine body composition, I understand that I must sit in a tank filled with water placing my whole body underwater as I exhale as much air as possible. I will then hold my breath as long as I can. I understand that the length of this test will be determined on the amount of air and the consistency by which I exhale.

I will be required to consume creatine monohydrate +dextrose, creatine monohydrate, or glucose. I understand that for five days I must consume a substance four times daily. However, for twelve days I will take a substance only once a day. Also, I understand that creatine is produced within the human body and is classified as a normal food supplement which is not considered a drug.

2. Risks and Benefits:

I may find myself experiencing symptoms of muscle fatigue such as burning, twitching, and tightness as I complete each test to my maximal ability. I also understand that these sensations will be very similar to those that I experience during training at high intensity.

I understand that injury could occur from falling or stumbling during the sprint tests. However, I understand that every precaution will be taken to prevent this from occurring.

I also understand that nausea, muscular fatigue, or sensations of passing out may result from performing the Wingate bike tests. I understand that if any of the previous do occur I will be treated promptly and effectively by trained personnel.

I may experience some discomfort as I hold my breath during hydrostatic weighing. Also, some breathlessness may occur. I understand that proper technique will be used to ensure my safety.

I understand that the consumption of these substances may cause gastrointestinal distress, which includes diarrhea and indigestion. As a result, I will be required to eat at approximately the same time that I consume one of these substances.

I understand that in the event of illness or injury as a result of participation in this study no compensation, financial or otherwise, will be provided by Marshall University, Experimental and Applied Science, or the investigators.

_____ _____

Subject's Signature Date

_____ _____

Parent's or Guardian's Signature Date

3. Benefits:

By participating in this study I will receive information regarding my muscular strength and power, as well as my percent body fat. In addition, this information may help my training sessions, therefore, improving my performance.

4. Confidentiality:

I understand that confidentiality of my records will be maintained and that my identity on research forms and in publishing articles will not be revealed. I understand that the records may be seen by the Marshall University IRB, the Food and Drug Administration or other Federal or state government agencies (if appropriate) for inspection.

I have been advised that if I have any questions regarding this study I may contact Dr. Terry Shepherd at 696-2925 or Joe McQuain at 733-6413. If I have any questions regarding my rights as a research participant in a research study I may call Dr. Nancy Scher at 696-7320.

Participation in this study is strictly voluntary. I understand that I may withdraw from this study at any time without bias. In addition, I understand that during any test or during any portion of the testing procedures if I feel that I can no longer continue and wish to stop I may do so freely.

Prior to participating in this study I will be given a copy of this consent form. I understand the procedures involved and hereby agree to participate in the previously described study.

_____ _____

Subject's Signature Date

_____ _____

Parent's or Guardian's Signature Date

_____ _____

Witness Date

_____ _____

Principal Investigator Date