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Relationship Between Endogenous Creatine Levels and Maximal Upper Body Strength, Short Term Muscle Recovery and Body Fat In Males

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THE RELATIONSHIP BETWEEN ENDOGENOUS CREATINE LEVELS AND
MAXIMAL UPPER BODY STRENGTH, SHORT TERM MUSCLE RECOVERY AND
BODY FAT IN MALES

by

Vincent James Dalbo III

(Under the Direction of Jim McMillan)

ABSTRACT

Numerous studies have found creatine supplementation to positively enhance performance but no research found has examined the effects of endogenous creatine levels on performance. The purpose of this study was twofold. First we examined correlations between endogenous creatine levels and strength, absolute strength, short term muscle recovery and body fat. We also examined the effects of creatine supplementation with a sufficient washout period on plasma creatinine levels. Participants consisted of 24 healthy men who met with the experimenter twice over a 4 day period. Significant positive correlations ($p \leq .05$) were found between creatine and strength, absolute strength, lean body mass and muscle recovery as a function of total weight lifted following 3 and 5 sets of the muscle fatigue protocol.

INDEX WORDS: Endogenous, Creatine, Strength, Muscle Recovery, Body Fat

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by

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CHAPTER 1

INTRODUCTION

Creatine is stored in the liver, brain, kidneys and testes (Persky & Brazeau, 2001) but 95% (Balsom, Soderlund, & Ekblom, 2004; Grande & Graves, 2005) to 98% of creatine is stored in skeletal muscle (Peeters, Lantz, & Mayhew, 1999) as creatine is directly involved in muscular contractions (Grande & Graves, 2005). During creatine supplementation total creatine content in muscle has been found to increase by 10%-20% with 20%-40% of increased intramuscular creatine being stored as phosphocreatine (Kreider, Ferreira, Wilson, Grindstaff, Plisk, Reinardy, et al., 1998). The increased creatine content in muscles enhances the phosphagen system (Almada, Kreider, Ferreira, Wilson, Grindstaff, Plisk, et al., 1997; Bosco, Tihanyi, Pucspk, Kovacs, Gobossy, Colli, et al., 1997; Casey, Constantin-Teodosiu, Howell, Hultman, & Greenhaff, 1996; Earnest, Snell, Rodriguez, Almada, & Mitchell, 1995; Ferreira, Kreider, Wilson, Grindstaff, Plisk, Reinardy, et al., 1997; Grindstaff, Kreider, Bishop, Wilson, Wood, Alexander, et al., 1997; Harris, Viru, Greenhaff, & Hultman, 1993; Kirksey, Warren, Stone, Stone, & Johnson, 1997; Kreider et al., 1998; Kreider, 1998; Prevost, Nelson, & Morris, 1997; Rawson & Volek, 2003; Skare, Skadberg, & Wisnes, 2001) allowing users to maintain higher levels of performance for longer durations of time.

Over 200 studies have been conducted examining the effects of creatine supplementation on athletic performance since 1993 (Rawson & Volek, 2003) and in 2000 The American College of Sports Medicine (ACSM) released a position statement claiming creatine supplementation can improve high intensity, short duration performance especially during repeated exercise bouts (ACMS, 2000; Rawson & Volek;

Terjung, Clarkson, Eichner, Greenhaff, Hespel, Israel, et al., 2000). However, not all creatine supplementation studies have resulted in significant performance improvements as it may be necessary to increase total muscle creatine by close to 20 mmol·kg⁻¹ dry weight (dw) from creatine supplementation to obtain significant performance improvements (Greenhaff, Bodin, Soderlund, & Hultman, 1994). Research has identified three interindividual responses to creatine supplementation: responders, individuals who experience a > 20 mmol·kg⁻¹ dw increase in total intramuscular creatine monohydrate plus phosphorylated creatine (Greenhaff, Bodin, Soderlund, & Hultman; Syrotuik & Bell, 2004); quasi responders, individuals who experience between a 10-20 mmol·kg⁻¹ dw increase and nonresponders, individuals who experience < 10 mmol·kg⁻¹ dw increase (Syrotuik & Bell). It has been established that performance increases are correlated to the amount of creatine that is absorbed during creatine supplementation (Syrotuik & Bell) and this correlation is prominently displayed in vegetarians who have lower endogenous creatine levels than non-vegetarians and have been found to tend to experience greater benefits from creatine supplementation (Rae, Digney, McEwan, & Bates, 2003; Shrier, 2004).

Numerous studies have found creatine supplementation to increase muscular strength (Greenhaff, Casey, Short, Harris, Soderlund, & Hultman, 1993; Balsom, Soderlund, Sjodin, & Ekblom, 1995; Earnest, Snell, Rodriguez, Almada, & Mitchell, 1995; Rawson, Clarkson, Price, & Miles, 2002; Vandenberghe, Goris, Van Hecke, Van Leemputte, Vangerven, & Hespel, 1997; Vandenberghe, Van Hecke, Van Leemputte, Vanstapel, & Hespel, 1999), short term muscle recovery (Almada et al., 1997; Bembien & Lamont, 2005; Bosco et al., 1997; Kreider, 1998) and fat-free mass (Becque, Lochmann,

& Melrose, 1997; Becque, Lochmann, & Melrose, 2000; Earnest, Snell, Rodriguez, Almada, & Mitchell, 1995; Haff, Kirsey, & Stone, 1999; Kreider et al., 1998; Kreider, Grindstaff, Wood, Bullen, Klesges, & Lotz, 1996; Maganaris and Maughan, 1998; Rawson, Clarkson, Price, & Miles, 2002; Vandenberg et al., 1997) but no research found has examined the effects of endogenous creatine levels on performance or body composition even though endogenous creatine levels normally range between 120-140 g dry mass (dm) (Rawson & Volek, 2003; Sarubin-Fragakis), with studies finding endogenous creatine levels ranging from 100-150 mmol/kg dm (Harris, Hultman, & Norjedo, 1974; Steenge, Simpson, & Greenhaff, 2000).

The normal variability in endogenous creatine levels (20 g dm) is equivalent to the proposed amount of creatine that must be absorbed during creatine supplementation to obtain significant performance improvements. Therefore we expect endogenous creatine levels to be positively correlated with strength, absolute strength determined by subtracting body weight from one repetition maximum strength and short term muscle recovery while being negatively correlated with body fat percentage. Additional analyses were conducted to examine if past creatine supplementation followed by an appropriate washout period of 4 months (Rawson, Persky, Price, & Clarkson, 2004) had an effect creatinine levels; as it is unknown if new steady-state relations occur between creatine and creatinine during periods of prolonged creatine supplementation (Culpepper, 1998).

CHAPTER 2

METHODS

Participants

Each participant was required to be a student at Georgia Southern University and had to have at least 1 year of resistance training experience ($M = 4.80$, $SD = 3.03$ years) to reduce the effect of strength differences due to technique. Participants were also required to bench press their body weight and could not have consumed an ergogenic aid over the past 2 months or a creatine supplement in the previous 4 months. Data were collected on 40 male participants but only 23 participants were included in the study. Participants were eliminated because of suspected creatine supplementation (1), blood lysing (2) during the blood draw which negatively affected the accuracy of the creatinine reading. Resistance training during the course of the study (1), failure to meet the strength requirement (2), consuming a creatine supplement (2), consuming an ergogenic aid (4) and (5) failing to return the food log. Each participant was informed of the procedures involved in the study along with the risks and benefits of participation. Informed consent was obtained after approval from the Institutional Review Board.

Procedures

Participants met with the experimenter twice during a four day period. During the first meeting participants read and signed the informed consent (see Appendix C) and were instructed how to record a three day food diary (see Appendix D). Nutritional data were recorded to ensure no significant changes in energy or macronutrient consumption occurred between participants over the duration of the study. Data were analyzed using Diet Analysis Plus (7.0, DA PLUS). Next, participants completed a demographic

questionnaire that contained questions assessing the use of ergogenic aids, current physical activity level and weight training experience (see Appendix E). Then the experimenter demonstrated and described proper bench press technique to each participant using the standards described by the National Strength and Conditioning Association (NSCA) (Baechle & Earle, 2000, see Appendix F). Participants then practiced bench press technique for 2 sets of 5 repetitions with approximately 50% of their bodyweight. Participants were then reminded to refrain from resistance training for the duration of the study and to fast for 8 hours prior to the next meeting with the experimenter.

Three days later, during the second meeting, participants returned their 3 day food log. Next height, weight and body fat percentage (using the 3-site Jackson Pollack skinfold equation) (Jackson & Pollack, 1985, see Appendix G) of each participant were recorded followed by a 10 mL blood draw from the left antecubital vein by a registered nurse. Blood was collected in a Vacutainer Glass Whole Blood Tube with heparin (Mfr. No.: 366535, Fisher Scientific, St. Louis, MO), centrifuged and pipetted to separate the plasma from the blood and the plasma was frozen for later analysis. Participants were then tested for strength using procedures set forth by the NSCA (Baechle & Earle, 2000, see Appendix H). Five minutes following completion of the one repetition maximum test participants performed five bench press sets to failure with 80% of their one repetition maximum with two minute rest periods between sets to determine the correlation between endogenous creatine levels and short term muscle recovery. Participants were verbally encouraged to give maximal effort during each lift.

Creatine Determination

The body surface area of each participant was calculated (see Figure 1), divided by 1.73 m² and multiplied by 120 g giving an estimate of endogenous creatine levels. (Delanghe, De Slypere, De Buyzere, Robbrecht, Wieme, & Vermeulen, 1989).

Figure 1. Calculation of body surface area (BSA)

$$\text{BSA} = ((\text{body weight} \times \text{height}) / 3125)^{1/2}$$

Body weight in pounds

Height in inches

BSA is calculated in square meters

Creatinine Determination

Blood samples were centrifuged (MARATHON 21 K/R, Fisher Scientific, St. Louis, MO) at 850 x g for 10 minutes at 4 degrees Celsius. Then plasma was pipetted from the Vacutainer into 2 separate vials and immediately frozen at -80 degrees Celsius for later analysis using a creatinine kit (500701 from Cayman Chemical Company, Ann Arbor, MI). For creatinine determination plasma samples were thawed at room temperature (~74 degrees Celsius) and 8 standards were prepared by mixing the creatinine standard with HPLC-grade water. Next plasma samples were placed on a Vortex mixer and 15 µl of the creatinine sample were pipetted into three wells using a multichannel adjustable volume micropipette (Finnpipett, Thermo electron Corporation,

Waltham, MA). A new pipette tip was used for each sample during each step of the procedure. Then the reaction was started by adding 150 μl of an alkaline picrate solution and the plate containing the samples was covered with a plate cover and mixed on an orbital shaker (Maxi Mix II, Type 37600 Mixer, Barnstead/ThermoLynce, Dubuque, IA) for 10 minutes at a low speed. The plate cover was removed and the samples were gently blown on to pop excess bubbles. Initial absorbance values were read at 492 nm on a plate reader (Spectromax 340, SOFTmaxPro 2.6.1, Sunnyvale, CA). The plate was removed from the plate reader and 5 μl of acid solution was added to each well. The plate was covered with the plate cover and mixed on an orbital shaker for 20 minutes at a low speed. The plate cover was removed and the samples were gently blown on to pop excess bubbles. Final absorbance values were read at 492 nm on a plate reader. For the extended creatinine analysis see Appendix I.

CHAPTER 3

RESULTS

Plasma creatinine levels were determined in triplicate ($M = 1.152$, $SD = .267$ mg/dl) with an intraclass correlation coefficient of $r = .947$. Endogenous creatine levels were found to be significantly ($p \leq .05$) positively correlated with strength ($r = .797$, $p < 0.001$) and absolute strength ($r = .456$, $p = 0.029$). Endogenous creatine levels were not found to be correlated with short term muscle recovery when examining total repetitions after three ($r = -.144$, $p = 0.512$) or five sets ($r = -.124$, $p = 0.574$); however, when examining the correlation between endogenous creatine levels and total weight lifted (weight lifted x repetitions) as a measure of short term muscle recovery significant positive correlations were found after three sets ($r = .503$, $p = 0.014$) and five sets ($r = .522$, $p = 0.011$). Endogenous creatine levels were not found to be correlated with body fat percentage ($r = .292$, $p = 0.177$) but were significantly positively correlated with lean body mass ($r = .524$, $p = 0.010$). Additional significant correlations were found between creatine and height ($r = .876$, $p < 0.001$), weight ($r = .989$, $p < 0.001$), years of resistance training ($r = .701$, $p < 0.001$) and current amount of repetitions performed while resistance training ($r = -.683$, $p = 0.003$).

Results from independent t-tests revealed participants who previously used a creatine supplement to have significantly ($p = 0.005$) higher endogenous creatine levels than participants who never used a creatine supplement. Additionally those who had used a creatine supplement had significantly more resistance training experience ($p = 0.002$) weighed more ($p = 0.004$) and had a higher body fat percentage ($p = 0.001$) than participants who have never used a creatine supplement. Moreover no difference in

creatinine levels were found between participants who had and had not consumed a creatine supplement ($p = .941$). For descriptive statistics see Table 1.

Table 1

Participant Descriptive Statistics

Descriptive Statistics	M	SD
Age	20.91	3.04 years
Height	68.61	2.59 inches
Body Weight (BW)	172.57	20.30 pounds
Body Fat Percentage	13.14	5.75 %
Lean Body Mass	152.57	12.15 pounds
1 Repetition Maximum (RM)	222.61	40.73 pounds
Absolute Strength (1RM – BW)	50.04	27.13 pounds
Repetitions Set 1 (80% 1 RM)	6.43	1.80 reps
Repetitions Set 2 (80% 1 RM)	4.35	1.07 reps
Repetitions Set 3 (80% 1 RM)	3.13	1.01 reps
Repetitions Set 4 (80% 1 RM)	2.43	0.66 reps
Repetitions Set 5 (80% 1 RM)	2.22	0.60 reps
Total Repetitions (Three Sets)	13.91	3.07 reps
Total Repetitions (Five Sets)	18.57	3.92 reps
Calorie Consumption	2737.82	913.08 calories
Protein Consumption	114.75	43.22 grams

CHAPTER 4

DISCUSSION

The majority of research has found creatine supplementation to increase strength, muscle recovery and fat free mass with the most recent research finding a positive relationship between the amount of creatine absorbed from creatine supplementation and performance improvements (Greenhaff, Bodin, Soderlund, & Hultman, 1994, Syrotuik & Bell, 2004). Interindividual differences in endogenous creatine levels have been found to vary as much as 50 g so it is reasonable to believe endogenous creatine levels could effect strength, short term muscle recovery and fat free mass as much as creatine supplementation.

Endogenous Creatine and Strength

Bench press was used to assess strength because the majority of creatine supplementation studies have measured strength using bench press or leg press performance. Endogenous creatine levels were found to be significantly correlated with strength and absolute strength. The correlation between endogenous creatine levels and absolute strength is particularly important because strength has been found to be related to body weight and body weight has been found to be correlated with creatine (Mosteller, 1987). Absolute strength negated the effects of body weight on strength by subtracting each participants body weight from the amount of weight lifted. Arciero, Hannibal, Nindl, Gentile, Hamed and Vukovich (2001) found 40% of increases in strength during creatine supplementation occur independent of exercise and the results from this study suggest that 63.5% of variance in upper body strength can be accounted for by endogenous creatine levels.

Endogenous Creatine and Short Term Muscle Recovery

The primary effect of creatine supplementation is to enhance the phosphagen system (Almada et al., 1997; Bosco et al., 1997; Casey, Constantin-Teodosiu, Howell, Hultman, & Greenhaff, 1996; Earnest, Snell, Rodriguez, Almada, & Mitchell, 1995; Ferreira et al., 1997; Grindstaff et al., 1997; Harris, Viru, Greenhaff, & Hultman, 1993; Kirksey, Warren, Stone, Stone, & Johnson, 1997; Kreider et al., 1998; Kreider, 1998; Prevost, Nelson, & Morris, 1997; Rawson & Volek; Skare, Skadberg, & Wisnes, 2001) by increasing total creatine content in muscles (Kreider et al., 1998) allowing users to train harder for longer durations of time (Rawson & Volek, 2003). This study mimicked a muscle fatigue protocol used by Kelly and Jenkins (1998). When using total repetitions performed after three sets and five sets no significant correlations were found between endogenous creatine and short term muscle recovery, most likely a function of participants with higher creatine levels lifting significantly heavier weights. When examining short term muscle recovery as a function of the total amount of weight lifted a statistically significant correlation was found between creatine and short term muscle recovery because participants with higher endogenous creatine levels were lifting greater amounts of weight for the same amount of repetitions.

Endogenous Creatine and Fat Free Mass

The majority of research has found creatine supplementation to positively influence body mass as studies have found creatine supplementation to increase fat free mass (Becque, Lochmann, & Melrose, 1997; Becque, Lochmann, & Melrose, 2000; Earnest, Snell, Rodriguez, Almada, & Mitchell, 1995; Haff, Kirsey, & Stone, 1999; Kreider et al., 1998; Kreider, Grindstaff, Wood, Bullen, Klesges, & Lotz, 1996;

Maganaris & Maughan, 1998; Rawson, Clarkson, Price, & Miles, 2002; Vandenberg et al., 1997) and lean body mass (Flisinska-Bojanowska, 1996; Haff, Kirksey, & Stone; Kirksey, Stone, Warren, Johnson, Stone, et al., 1999; Maganaris & Maughan, 1998; Rawson, Clarkson, Price, & Miles; Noonan, Berg, Latin, Wagner, & Reimers, 1998; Vandenberg et al., 1997) but a few studies have not found creatine supplementation to have a statistically significant effect on body fat percentage (Kirksey et al. 1997; Noonan, Berg, Latin, Wagner, & Reimers). Results from this study were equivocal in regard to the effects of endogenous creatine levels on body mass as no statistically significant correlation was found between endogenous creatine levels and body fat percentage, but endogenous creatine levels were found to be significantly positively correlated with lean body mass.

Creatine and Creatinine

A concern surrounding long term creatine supplementation is that a new steady state relation between creatine and creatinine may develop (Culpepper, 1998), however results from this study were consistent with prior research (Culpepper; Hultman, Soderlund, Timmons, Cederblad, & Greenhaff, 1996; Poortmans, Auquier, Renault, Durussel, Saugy, & Brisson, 1997) which have suggested plasma creatinine levels to be unaffected by short term creatine supplementation. Future research should focus on the effects of long term creatine supplementation on creatinine levels as the possibility of new steady state relations forming between creatine and creatinine following long term creatine supplementation appears to be the last known disproved danger of long term creatine supplementation. Researchers could also examine the effects of endogenous creatine levels on aerobic performance as creatine supplementation has been found to

enhance some forms of aerobic exercise (Birch, Nobel, & Greenhaff, 1994; Peyrebrune, Nevill, & Donaldson, 1998) or the effects of caffeine on endogenous creatine levels as caffeine appears to negate the positive effects of creatine supplementation (Vandenbergh, Gillis, Van Leemputte, Van Hecke, Vanstaple, & Hespel, 1996). A replication of the current study is also noteworthy as the sample size was small and with a larger number of participants stronger correlations can be expected.

Additional Analysis

Strong significant positive correlations were found between creatine and height and weight which is consistent with the work of Mosteller (1987) who found endogenous creatine levels to be significantly correlated with body surface area. Endogenous creatine levels were also found to be positively correlated with years of resistance training which is expected as creatine has been found to be correlated with muscle mass (Culpepper, 1998). Endogenous creatine was also found to be negatively correlated with the amount of repetitions performed while resistance training.

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APPENDIX A: RESEARCH HYPOTHESES AND STUDY LIMITATIONS

Research Hypotheses

Endogenous creatine levels will be significantly positively correlated with upper body strength ($\alpha \leq .05$), absolute strength ($\alpha \leq .05$) and muscle recovery ($\alpha \leq .05$) while being negatively correlated to body fat percentage ($\alpha \leq .05$). Participants with the highest endogenous creatine levels will have the highest one repetition maximum bench press lifts relative to body mass, will recover quicker between sets and have low body fat percentages than participants with lower endogenous creatine levels.

Limitations

The study was limited by the following:

- Not a random sample.

Delimitations

The study was delimited to the following:

- All participants were male students from Georgia Southern University.
- The only exercise used to assess strength and muscle recovery was the bench press.
- All participants had at least one year weight training experience.

Assumptions of the Study

The conduct of this study was based upon the following assumptions:

- Sample was representative of the population.

- Observations were drawn from normally distributed populations in regards to endogenous creatine levels.
- Observations represented random samples from other populations.
- Participants had at least one year resistance training experience.
- Participants gave maximal effort during study.
- Measures of each participant's creatine levels were accurate.
- Participants did not consume an ergogenic aid within two months of the study.
- Participants did not consume a creatine supplement within four months of the study.
- Received an accurate measure of each participant's one repetition maximum on bench press.
- The experimenter was able to consistently identify improper technique.
- Participants properly prepared themselves for testing, getting appropriate amounts of sleep and eating properly before the test.
- Participants accurately recorded their dietary intake.

Definition of Terms

- Absolute Strength- Maximal one repetition maximum strength minus body weight.
- Failure- The inability to perform a repetition with proper bench press technique as defined by the NSCA.
- Short term muscle recovery- Will be calculated as the total number of

repetitions performed after five sets of a muscle fatigue protocol similar to that used by Kelly and Jenkins (1998).

APPENDIX B REVIEW OF LITERATURE

Review of Literature

Effects of Hormones on Endogenous Creatine Levels

In humans endogenous creatine (Cr) levels achieve homeostasis at approximately 125 g (range 120 g -140 g) (Sarubin-Fragakis, 2000) which is significantly less Cr than the body is capable of storing as Cr levels have been found to reach 195.1 mmol/kg dry mass following 5 days of Cr/carbohydrate supplementation at a dose of 20 g per day (Green, Hultman, Macdonald, Sewell, & Greenhaff, 1996). Endogenous Cr levels may be maintained at significantly lower levels than maximal storage because of the importance of arginine, glycine and methionine in the production of proteins and other cell constituents such as keratin, ATP, DNA, RNA, GTP, methionine, histones, carnosine and norepinephrine (Walker, 1979). Additionally the body appears to have developed separate organs for the biosynthesis and utilization of Cr, as tissues responsible for Cr biosynthesis do not significantly phosphorylate Cr and the organs responsible for Cr biosynthesis do not contain significant amounts of creatine kinase (CK) which is used to break down Cr into a usable form of energy. Furthermore mitochondria have been found to contain either CK or amidinotransferase which is used in Cr production (Walker). The separation of Cr production and utilization has several advantages. First the liver contains the enzymes necessary to process dietary components that can be used in endogenous Cr production before releasing the nutrients into the blood. Moreover the liver can produce Cr from compounds released from muscle and nervous tissues and can slow its metabolism through hormonal signals which is beneficial during periods of nutritional constraints (Walker).

Cr production is most efficient with optimal blood concentrations of insulin, somatotropin, thyroid hormone and testosterone (Walker, 1979). Insulin and glucocorticoids specifically effect Cr retention and excretion as insulin has been shown to enhance Cr uptake in muscle while glucocorticoids have been found to increase plasma Cr. Glucocorticoids are particularly important during dietary deficiencies and muscle wasting diseases which increase the breakdown of muscle proteins resulting in increased levels of amino acids in the liver. Increased levels of arginine and glycine in conjunction with decreased protein synthesis promote Cr biosynthesis (Walker).

Creatine and Phosphocreatine

During high-intensity, short-duration bouts of exercise lasting approximately 1-14 seconds the phosphagen system is the primary energy supplier allowing the body to function. Cr plays an essential role in the formation of ATP in the phosphagen system (Powers & Howley, 2004) and ATP facilitates the phosphorylation of Cr (Walker, 1979). As stored ATP begins to be synthesized for movement, phosphocreatine (PCr) initiates the process of producing ATP in an attempt to keep ATP levels high. PCr is catalyzed by CK to yield Cr, P_i (inorganic phosphate) and energy (ATP) (Powers & Howley). The energy from this endergonic reaction is used in an exergonic reaction to combine ADP with P_i to yield ATP (Haff, Kirksey, & Stone, 1999; Powers & Howley; Volek & Kraemer, 1996). For movement ATP must be broken down by CK (ATPase) to produce ADP, P_i and energy (Powers & Howley). Since the phosphagen system relies on PCr for the production of ATP, as PCr levels decline performance decreases because of a decreased amount of ATP being supplied to working muscles (Powers & Howley).

During strenuous exercise the phosphocreatine energy shuttle acts in contracting muscles to regenerate ATP through the return of high-energy phosphate bonds from the PCr reserve to produce ATP (Culpepper, 1998). To keep the energy pathway (CK reaction) running in a cyclic manner Cr is resynthesized into PCr by other energy pathways, currently believed to be oxidative metabolism in the mitochondria (Haff, Kirksey, & Stone, 1999). When the oxidative production of ATP exceeds energy needs the high-energy phosphate bond of ATP is transferred to Cr to produce PCr in the mitochondrial membrane.

The phosphocreatine energy shuttle can be described in three stages: the peripheral terminus, the intervening space and the energy-generating terminus. The phosphocreatine energy shuttle begins at the peripheral terminus which is the site of energy utilization and is where isoenzymes of CK rephosphorylate ADP produced via the CK reaction allowing muscular contractions. Cr that is produced from the CK reaction travels across sites of energy production and utilization known as the intervening space. Then Cr transverses in the intervening space and arrives in the energy-generating terminus where Cr interacts with isoenzymes of CK to yield PCr from mitochondrial ATP. Next regenerated PCr is shuttled back to sites of energy utilization and the process of energy production continues.

ATP is the primary energy source for the body but PCr has a phosphate group transfer potential approximately 3 kcal/mol higher than ATP (Walker, 1979) and PCr/ATP ratios have been found to vary from 3:1 to 4:1 while PCr/Cr ratios have been reported to range from 2:1 to 2.5:1 (Culpepper; 1998; Greenhaff, 1995; Hultman, Soderlund, Timmons, Cederblad, & Greenhaff, 1996). Greater quantities of PCr and Cr

are stored in favor of ATP because Cr and PCr have a lighter mass and are less negatively charged than ATP and ADP which enables greater amounts to be stored in cells without disrupting ionic and osmotic balances (Grande & Graves, 2005). Cr and PCr also act as energy messengers making each less vulnerable to enzymatic diversion than ATP (Walker). ATP and ADP are substrates to hundreds of enzymes while Cr and PCr are substrates to only CK which emphasizes the importance of PCr and Cr for providing the body with a constant energy source for smooth skeletal muscle contractions which allows the heart to pump (Walker) and serves as an energy source for the brain (Rae, Dingney, McEway, & Bates, 2003; Wallimann, Wyss, Brdiczka, Nicolay, & Eppenberger, 1992; Norwood, Ingwall, Norwood, & Fossel, 1983).

Cr was first used commercially to promote growth and increase the energy of chickens (Walker, 1979) and until the 1960s Cr had to be extracted from raw meat (Grande & Graves, 2005). The synthetic production of Cr made the substance widely available to the general population and in a matter of a few decades Cr became the most popular athletic supplement (Grande & Graves) with sales nearing 100 million dollars in 1998 (Haff, Kirksey, & Stone, 1999) and exceeding 400 million dollars in 2004 (Grande & Graves).

Positive Benefits of Cr Supplementation

In addition to being used as an ergogenic aid Cr supplementation has been found to have positive effects on general health and has been used in the treatment of muscle wasting diseases (Tarnopolsky, Roy, & MacDonald, 1997). Cr has been used in the treatment of gyrate atrophy of the choroid retina (Sipila, Rapola, Simell, & Vannas, 1981) and has been found to enhance muscle functional capacity along with aerobic and

anaerobic performance in people with various forms of neuromuscular diseases (Tarnopolsky & Martin, 1999; Walter, Lochmuller, Reilich, Klopstock, Huber, Hartard, et al., 2000) and McArdle's disease (Vorgerd, Zange, Kley, Grehl, Husing, Jager, et al., 2000). Incidentally many muscle wasting diseases are associated with irregularities in Cr metabolism (Haff, Kirksey, & Stone). However Cr supplementation is not an effective technique in the "treatment" of all muscle wasting diseases as Cr supplementation was unable to improve the functional capacity in people inflicted with chronic progressive external ophthalmoplegia or mitochondrial myopathy (Hespel, Eijnde, Van Leemputte, Urso, Greenhaff, Labarque, et al., 2001; Klopstock, Querner, Schmidt, Gekeler, Walker, Hartard, et al., 2000).

Research has also found Cr supplementation to enhance muscle size and strength following disuse muscle atrophy leading Hespel et al. (2001) to suggest Cr can be effective at preventing and or reversing neuromuscular degeneration in humans. In a study examining the role of PCr and Cr in muscle metabolism Cr depleted rats experienced atrophy in fast twitch-glycolytic muscle fibers (Meyer, Brown, Krilowicz, & Kushmerick, 1986) followed by a study which found Cr supplementation to significantly enhance protein content in rat muscle as a result of increases in myofibrillar proteins (Flisinska-Bojanowska, 1996). These results suggest the consumption of Cr to promote an increased rate of myofibular synthesis contributing to increases in lean body mass (Haff, Kirksey, & Stone, 1999). Other studies have found Cr supplementation in conjunction with resistance training to result in increases in lean body mass (Haff, Kirksey, & Stone). An additional study examining *in vitro* supplementation of Cr in rats found an increased synthesis rate of myosin heavy chains and actin when they are formed

in vitro and *in vivo* (Haff, Kirksey, & Stone). Gyrate atrophy patients were also found to have increased fast twitch-glycolytic muscle fiber diameter after consuming 1.5 g of Cr per day for 1 year (Haff, Kirksey, & Stone; Sipila, Rapola, Simell, & Vannas, 1981).

Numerous studies have found Cr supplementation to positively influence the health of people with heart problems (Andrews, Greenhaff, Curtis, Perry, & Cowley, 1998; Fagbemi, Kane, & Parratt, 1982; Gordon, Hultman, Kaijser, Kristjansson, Roll, Nyquist, et al., 1995; Haff, Kirksey, & Stone, 1999; Hearse, Tanaka, Crome, & Manning, 1986; Robinson, Braimbridge, & Hearse, 1984; Pauletto & Stumia, 1996) and others undergoing cardiac surgery (Pauletto & Stumia). Gordon et al. (1995) have found 1 week of Cr supplementation to benefit chronic heart failure patients by improving muscular strength and endurance. Another study involving chronic heart failure patients found PCr to significantly improve left ventricular systolic and diastolic function (Pauletto & Stumia) while other studies have found PCr to exert antiarrhythmic properties (Fagbemi, Kane, & Parratt; Haff, Kirksy, & Stone; Hearse, Tanaka, Crome, & Manning, 1986). PCr has been administered to people during cardiac surgery and because of PCr's ability to provide myocardial protection from acute ischemic injury and preoperative ischemic damage as a treatment for acute myocardial infarction (Pauletto & Stumia). Some researchers have even suggested that PCr be used in the treatment of chronic heart failure patients (Andrews, Greenhaff, Curtis, Perry, & Cowley, 1998; Gordon et al., 1995; Haff, Kirksey, & Stone). Cr also improves heart health indirectly as 56 days of Cr supplementation was shown to improve blood lipid profiles by decreasing total cholesterol and triglycerides in middle-aged males and females diagnosed as hypertriglyceridemic (Earnest, Almada, & Mitchell, 1996). Additionally Kreider et al.

(1998) found high density lipoproteins (HDL-C) to increase by 13% while very low density lipoproteins (VLDL-C) to decrease by 13% in response to 28 days of Cr supplementation.

Cr supplementation may also improve brain function as Norwood, Ingwall, Norwood and Fossel (1983) found the CK/PCr system to be a rapidly available source of ATP synthesis in the brain, suggesting CK to be a significant enzyme for energy metabolism in the brain (Wallimann, Wyss, Brdiczka, Nicolay, & Eppenberger, 1992). Moreover Cr supplementation at a dose of 5 g per day for 6 weeks has been shown to enhance brain function in vegetarians by significantly improving intelligence test scores requiring speed of processing such as the Raven's Advanced Progressive Matrices and working memory performance using a backward digit span test (Rae, Digney, McEwan, & Bates, 2003).

Cr has also been shown to control the rate of actin and myosin biosynthesis, a determinant of muscle hypertrophy (Walker, 1979). In 2003 Rawson and Volek conducted a meta-analysis which cited a large variability in 1RM (repetition maximum) bench press performance with improvements ranging from 3% to 45% among users of Cr. Improvements in muscular endurance ranged from 16% to 43% and a negative correlation was found between training status and strength increases aided by Cr supplementation. A 45% increase in 1RM bench press performance was found in 19 untrained males (Vandenbergh, Goris, Van Hecke, Van Leemputte, Vangerven, & Hespel, 1997) while a 30% increase was found in 14 males with approximately 6 years of resistance training experience and a 6% increase was reported in 8 males with approximately 11 years of resistance training experience (Rawson & Volek; Volek,

Kramer, Bush, Boetes, Incledon, Clark, & Lynch, 1997). The negative correlation found between Cr supplementation and strength increases is most likely a function of people with more training experience being closer to their genetic potential, thus making it more difficult to improve performance (Rawson & Volek). Another mechanism that may explain the greater increase in weightlifting performance in untrained participants is that most increases in strength during the first few weeks of weight training are due to neuromuscular adaptations to exercise (Baechle & Earle, 2000). The possibility exists that untrained individuals learned how to use the muscle they possessed more efficiently rather than improving performance primarily through Cr consumption.

Another study compared 1RM strength gains in participants consuming Cr for a duration of 4 weeks. The Cr plus training group improved bench press performance by 18% and leg press performance by 42%. While the Cr without training group improved bench press performance by 8% and leg press performance by 16%, suggesting 40% of the improvements in weightlifting performance were due to consuming a Cr supplement independent of training (Arciero, Hannibal, Nindl, Gentile, Hamed, & Vukovich, 2001; Rawson & Volek, 2003). Rawson and Volek (2003) suggest the 60% of unexplained variance is most likely attributed to participants being able to train at higher workloads. Although the mechanisms for performance enhancement are unknown, it has been established that raising baseline Cr levels improves weightlifting performance (Rawson & Volek).

Proposed Mechanisms of Improved Performance

Several hypotheses have been formulated to explain how Cr improves resistance training performance. Possible mechanisms include an increase in lean body mass (Volek

et al., 1999), enhanced anticatabolic protection (Parise, Mihic, MacLennan, Yarasheski, & Tarnopolsky, 2001), an increase in myosin heavy chain mRNA and protein expression (Willoughby & Rosene, 2001), an alteration in the expression of myogenic transcription factors (Hespel et al., 2001), increased protein synthesis (Rawson & Volek, 2003) an increase in satellite cell mitotic activity (Dangott, Schultz, & Mozdziak, 2000), and or increased intensity in individual workouts due to a greater supply of ATP (Casey, Constantin-Teodosiu, Howell, Hultman, & Greenhaff, 1996; Rawson & Volek).

Parise, Mihic, MacLennan, Yarasheski and Tarnopolsky (2001) conducted a study examining the effects of Cr supplementation leucine kinetics and mixed muscle-protein synthesis in trained individuals. Male and female participants were randomly placed into a control group who were supplemented with Cr at an initial dose of 20 grams of Cr per day for five days followed by maintenance dose of 5 grams for 3-4 days or placebo group who consumed an equivalent amount of a glucose polymer. Cr supplementation was found increase muscle PCr and Cr levels in males and females but only reduced leucine oxidation (19.6%) and plasma leucine rate of appearance (7.5%) in males; suggesting short term Cr supplementation may provide anticatabolic protection for lean muscle tissue in males and that tissues responding to the Cr supplementation or the signaling response(s) to Cr supplementation may be gender specific. No significant differences were found in whole body or mixed muscle protein synthesis (Praise, Mihic, MacLennan, Yarasheski, & Tarnopolsky, 2001).

In 2001 Willoughby and Rosene conducted a 12 week study composed of 3 groups (control, placebo and Cr) to examine the effects of Cr supplementation and heavy resistance training on muscle strength, myosin heavy chain (MHC) isoform mRNA and

MHC protein expression. MHC mRNA expression was found to be significantly greater in Type I, IIa and IIx muscle fibers in the Cr supplementation group than the placebo and control groups. Moreover MHC protein expression was significantly greater in Type I and IIx muscle fibers than the placebo and control groups; no significant difference was found between the Cr and placebo groups in IIa fibers but each was significantly greater than the control group (Willoughby and Rosene). Past research has found heavy resistance training to enhance myofibrillar protein synthesis (Welle, Bhatt, & Thornton, 1999) and Cr supplementation appears to increase MHC and myofibrillar activity enhancing the positive effects from heavy resistance training (Willoughby and Rosene).

The expression of myogenic transcription factors have been associated in the regulation of muscle fiber adaptations that occur during muscle hypertrophy. Which lead Hespel et al. (2001) to examine the effects of Cr supplementation on functional and structural adaptations of skeletal muscle and the expression of myogenic transcription factors during 2 weeks of leg immobilization, resulting in disuse atrophy, followed by an exercise rehabilitation program in a double-blind trial involving 22 participants. Leg immobilization resulted in a decrease in quadriceps muscle cross sectional area (CSA) by approximately 10% and maximal knee extension power by approximately 25% in both groups. During the 10 week recovery period the Cr supplementation group recovered at a significantly faster rate than the placebo group in terms of CSA and maximal knee extension power. The immobilization procedure did not result in myogenic factor protein expression in either group; however, in the placebo group myogenin protein expression was significantly increased while myogenic regulatory factor (MRF) MRF4 protein expression was significantly increased in the Cr group. The increase in MRF4 was also

positively correlated with the change in mean muscle fiber diameter. These results suggest Cr supplementation promotes muscle hypertrophy during rehabilitative strength training and the effect may be caused by a Cr induced increase in MRF4 and myogenin expression (Hespel et al., 2001).

Satellite cell mitotic activity is vital in postnatal muscle growth and Cr supplementation is the first oral nutritional supplement to result in enhanced satellite cell mitotic activity (Dangott, Schultz, & Mozdziak, 1999). As muscles grow myofiber hypertrophy occurs and increases in muscle fiber diameter result in increases in myofiber nuclei. Myonuclei do not divide and satellite cells provide the new DNA for enlarging myofibers by proliferating and when appropriate donate their nuclei to the enlarging myofibers (Moss & Leblond, 1971; see also Dangott, Schultz, & Mozdziak). The importance of satellite cells has been demonstrated in a prior study suggesting a decrease in satellite cells contribution of nuclei to enlarging muscle fibers resulted in reduced muscle growth (Mozdziak, Schultz, & Cassens, 1997). Other research has suggested increased satellite cell mitotic activity is necessary to maintain a constant volume of cytoplasm surrounding each myonucleus during an increase in muscle size (McCall et al., 1998; Rosenblatt & Parry, 1993).

Dangott, Schultz, and Mozdziak (1999) conducted a study examining the effects of Cr supplementation on compensatory hypertrophy and satellite cell mitotic activity in rats. Results from this study suggest Cr supplementation in conjunction with increased functional load results in significant increases satellite cell mitotic activity in overloaded muscles. However, there was no significant increase in satellite cell mitotic activity in the non-overloaded muscles. No significant differences were found in myofiber diameter in

overloaded and non-overloaded plantaris muscles between the Cr supplemented and control groups. This study suggests that Cr supplementation increases satellite cell mitotic activity during compensatory hypertrophy in which Cr supplementation in conjunction with increased functional capacity increases satellite cell mitotic activity (Dangott, Schultz, & Mozdziak). Brannon, Adams, Coniff and Baldwin (1997) have conducted a study suggesting that increased functional capacity of a muscle is necessary for Cr supplementation to elicit a hypertrophic response in rats.

It must be noted that Cr supplemented rats had a higher increase in body mass and had higher increases in muscle mass, myofiber diameter, and index of satellite cell mitotic activity than the control group and the small sample size of 12 rats (6 in each group) may account for the reason more significant differences were not found between the groups. More significant differences may have been found but muscle fiber type changes were introduced to the plantaris muscle of the rats in which myofibers may have lost their fast phenotype and developed a slower phenotype during the study (Ianuzzo & Chen, 1979; Noble, Tang, & Taylor, 1984; Roy, Talmadge, Fox, Lee, Ishihara, & Edgerton, 1997) and other studies have found fast twitch muscle fibers to be more responsive to Cr supplementation than slow twitch muscle fibers (Brannon, Adams, Coniff, & Baldwin, 1997; Sipila, Rapolao, Simell, & Vannas, 1981; Syrotuik & Bell, 2004).

As of 1999 it was unknown how Cr supplementation was related to increased satellite cell mitotic activity, but a link does exist. Cr supplementation may increase Cr levels within myofibers (Brannon, Adams, Coniff, & Baldwin, 1997; Casey, Constantin-Teodosiu, Howell, Hulman, & Greenhaff, 1996; Dangott, Schultz, & Mozdziak, 1999)

which increases osmotic pressure on the myofibers (Dangott, Schultz, & Mozdziak) and the increased osmotic pressure may lead to increases in muscle size (Brannon, Adams, Coniff, & Baldwin). Increased osmotic pressure may indirectly initiate satellite cells to proliferate and fuse with myofibers undergoing hypertrophy (Dangott, Schultz, & Mozdziak). Dangott, Schultz and Mozdziak (1997) also suggest Cr supplementation enhances muscle hypertrophy via a myofiber mechanism because satellite cell mitotic activity is controlled by myofibers (Mozdziak, Truong, Macius, & Schulz, 1998).

Many studies suggest an increase in body mass from Cr supplementation, long term studies ranging from 8 to 140 days suggest Cr can increase total body mass and fat-free mass (Haff, Kirksey, & Stone, 1999). Increases in body mass due to short-term Cr supplementation are most likely explained by water retention, but Balsom, Soderlund, Sjodin and Ekblom (1995) suggest that increased body mass resulting from long-term Cr supplementation and a resistance training regime may result from increases in fast twitch-glycolytic muscle fiber diameter, as long-term Cr supplementation stimulates an increased myofibular protein synthesis rate. In support of Balsom et al's. (1995) theory numerous studies have suggested that Cr plays a critical role in protein synthesis, in which Cr may be the chemical signal linking muscular activity and increased contractile protein synthesis during hypertrophy (Haff, Kirksey, & Stone).

The majority of research has suggested Cr supplementation to improve performance in high intensity, short duration bouts of exercise including sprint cycling (Balsom, Ekblom, Soderlund, Sjodinm, & Hultman, 1993; Birch, Noble, & Greenhaff, 1994; Casey, Constantin-Teodosiu, Howell, Hultman, & Greenhaff, 1996; Prevost, Nelson, & Morris, 1997; Syrotuik & Bell, 2004) and resistance training (Greenhaff,

Bodin, Soderlund, & Hultman, 1994; Greenhaff, Casey, Short, Harris, Soderlund, Hultman, 1993; Rawson & Volek, 2003; Volek et al., 1997) but other studies with similar protocols have failed to yield significant performance improvements (Barnett, C., Hinds, M., & Greenhaff, 1996; Cooke & Barnes, 1997; Febbraio, Flanagan, Snow, Zhio, & Carey, 1995; Kilduff et al., 2002; Stevenson & Dudley, 2001; Syrotuik & Bell). Methodological, procedural and or experimental design differences may account for the equivocal results but Greenhaff, Bodin, Soderlund and Hultman (1994) have suggested the effectiveness of Cr supplementation is dependent upon the extent of elevation experienced in intracellular muscle Cr that occurs in each individual. Studies support this claim by finding people with lower endogenous Cr levels (vegetarians) to experience greater improvements in performance than people with higher endogenous Cr levels prior to Cr supplementation (Burke, Chilibeck, Parise, Candow, Mahoney, & Tarnopolsky, 2003; Syrotuik & Bell).

There are three proposed interindividual responses to Cr supplementation: responders (R), individuals who experience a > 20 mmol·kg⁻¹ dw increase in total intramuscular Cr and PCr (Greenhaff, Bodin, Soderlund, & Hultman, 1994; Syrotuik & Bell); quasi responders (QR), individuals who experience between a 10-20 mmol·kg⁻¹ dw increase (Syrotuik & Bell) and nonresponders (NR), individuals who experience < 10 mmol·kg⁻¹ dw increase (Greenhaff, Bodin, Soderlund, & Hultman; Syrotuik & Bell).

Greenhaff, Bodin, Soderlund and Hultman (1994) suggest it may be necessary to increase total muscle Cr by close to 20 mmol·kg⁻¹ dry weight (dw) from Cr supplementation to obtain significant performance improvements. Kilduff et al. (2002) support Greenhaff et al's. (1994) claim by noting significant performance improvements

were not obtained in peak force or total work output during repeated isometric contractions on bench press in their study as a result of having 4 nonresponders to Cr supplementation in the subject pool. The nonresponders experienced increased Cr levels of ≤ 21 mmol·kg⁻¹ dw which may have obscured the overall group changes of the 32 participants in their study (Syrotuik & Bell, 2004).

People respond differently to stimuli such as resistance training programs and ergogenic aids (Bouchard & Malina, 1986; Syrotuik & Bell, 2004; Willams, 1998) furthermore genetic research supports the idea of having responders and nonresponders to treatments (Syrotuik & Bell). Greenhaff, Bodin, Soderlund and Hultman (1994) suggest approximately 20%-30% of individuals fall into the category of NR following 5 days of Cr supplementation at a dose of 20 grams per which may account for the lack of significant performance improvements in some studies particularly with small sample sizes (Syrotuik & Bell).

Syrotuik and Bell (2004) conducted a study to determine the physiological profile of R and NR to Cr supplementation. Participants consisted of 11 males who supplemented with Cr for 5 days at a dosage of 0.3 g·kg⁻¹·d⁻¹. Of the 11 participants 3 were R, 5 were QR, and 3 were NR. There was a descending trend regarding muscle fiber composition between the three groups. R had the highest percentage of type II fibers (63.1%), followed by QR (45.5%) and NR (39.5%). R and QR had a greater muscle cross-sectional area than NR pre and post Cr supplementation. R and QR had mean cross-sectional areas for type I (1,509 and 1,270 μm^2), type IIa (1,807 and 2,238 μm^2) and type IIb (1,695 and 1,740 μm^2) compared to NR with type I (900 μm^2), type IIa (1,377 μm^2), and type IIb (1,213 μm^2). R had greater mean muscle fiber increases than NR, type I (320

verse $60 \mu\text{m}^2$), type IIa (971 verse $46 \mu\text{m}^2$), and type IIb (840 verse $78 \mu\text{m}^2$). A descending trend was also found in body mass and fat free mass between the groups. R had the greatest increase in total body mass (2.4 kg) and fat free mass (2.0 kg), followed by QR (2.1 kg and 1.9 kg) and NR (1.9 kg and 1.7 kg). R improved maximal leg press performance by 25.8 kg followed by QR (2.5 kg) and NR (2.0 kg). The best determining factors determining the effectiveness of Cr supplementation were muscle fiber distribution and muscle CSA (Syrotuik & Bell).

Creatine Supplementation and Nutritional Choices

Nutritional choices also play a role in the effectiveness of Cr supplementation as caffeine may negate some of the positive performance benefits received from Cr supplementation (Vandenberghe, Gillis, Van Leemputte, Van Hecke, Vanstaple, & Hespel, 1996). A study conducted by Vandenberghe et al. (1996) was composed of three groups: a placebo group, a Cr group and a Cr plus caffeine group. The Cr and Cr plus caffeine groups significantly increased intramuscular PCr concentrations, however only the Cr group experienced significant improvements in dynamic torque production. In 1998 another study was published examining the effects of caffeine on Cr supplementation and was composed of the same three groups: a placebo group, a Cr group and a Cr plus caffeine group (Vanakoski, Kosunen, & Meririnne, 1998). As in the Vandenberghe et al. (1996) study researchers found that caffeine had no effect on Cr absorption, however researchers did not find any significant performance improvements in any of the groups in anaerobic or aerobic performance. Anaerobic performance was measured by maximal pedaling speed (rpm), maintenance of maximal speed (rpm) (or total work output (kJ)) during 1 minute cycling bouts. Aerobic measurements were made

by 45 minutes of cycling at a constant speed. No statistical differences were found between the groups for heart rate or blood lactate during aerobic or anaerobic exercise. Sample sizes in each study were small, 9 and 7 participants respectively.

Cr absorption rates are not affected by the consumption of caffeine so theoretically performance improvements gained from Cr should not be affected by caffeine, however no evidence exists to support this claim. In both studies Cr supplementation in the caffeine plus Cr group failed to significantly improve performance. Clearly more research needs to be conducted on this topic before a stance can be taken of the effectiveness of Cr supplementation when consumed in conjunction with caffeine.

Protein and carbohydrate consumption in conjunction with Cr supplementation have been found to significantly affect Cr absorption rates, due to insulin release (Steenge, Simpson, & Greenhaff, 2000). Numerous studies and books have reported insulin release due to carbohydrate consumption (Green, Hultman, Macdonald, Sewell, & Greenhaff, 1996; Steenge, Lambourne, Casey, Macdonald, & Greenhaff, 1998; Ivy & Portman, 2004; Powers & Howely, 2004; Steenge, Simpson, & Greenhaff) and insulin appears to aid Cr retention in response to Cr supplementation. Studies have shown carbohydrate or a combination of carbohydrate and protein consumption in conjunction with Cr supplementation to significantly enhance muscle Cr retention (Steenge, Simpson, & Greenhaff) as insulin release at concentrations of approximately 100 mU/l or greater can enhance Cr retention in human skeletal muscle (Steenge, Lambourne, Casey, Macdonald, & Greenhaff, 1998). Cr retention rates have been shown to be enhanced by approximately 25% (Steenge, Simpson, & Greenhaff) to 60% when a creatine

supplement is consumed with an insulin releasing stimulus (Green, Hultman, Macdonald, Sewell, & Greenhaff; Green, Simpson, Littlewood, Macdonald, & Greenhaff, 1996; Steenge, Simpson, & Greenhaff). The wide variability in enhanced retention rates may be due to functional differences between the studies in which urinary Cr analyses were conducted at 40 and 24 hours respectively following consumption of a Cr supplement (Steenge, Simpson, & Greenhaff).

Cr retention following supplementation varies among participants from no retention to a recorded 40 mmol/kg dm increase (Green, Hultman, Macdonald, Sewell, & Greenhaff, 1996; Greenhaff, 1996; Steenge, Simpson, & Greenhaff, 2000; See also Greenhaff, Casey, Short, Harris, Souderlund, & Hultman, 1994; Harris, Soderlund, & Hultman, 1992). Variables such as muscle fiber type (Syrotuik & Bell, 2004), age (Rawson, Clarkson, Price, & Miles, 2002; Rawson & Clarkson, 1999), endogenous Cr levels (Shrier, 2004) and insulin release affect absorption rates (Steenge, Simpson and Greenhaff, 2000; Walker, 1979). Moreover individuals with similar pre-supplementation Cr concentrations have been found to vary in Cr retention as much as six-fold (Steenge, Simpson, & Greenhaff), however the consumption of carbohydrates in conjunction with Cr supplementation has been found to reduce interindividual variability in the amount of muscle Cr retention during supplementation. In one study all 12 participants experienced an increase in muscle total creatine (TCr) of ≥ 20 mmol/kg dm which is equivalent to responders of Cr supplementation (Syrotuik & Bell, 2004). The Steenge, Simpson and Greenhaff study suggests a stimulatory effect of carbohydrates on muscle Cr retention due to insulin release stimulating sodium-potassium pump activity (See also Haugland, &

Chang, 1975). This research is consistent with animal based research that suggested insulin to increase Cr absorption (Walker, 1979).

Steenge, Simpson and Greenhaff (2000) also found that consuming 50 g of protein in conjunction with 47 g of carbohydrates is as effective as consuming 96 g of carbohydrates at increasing Cr retention following Cr supplementation and is significantly more effective at increasing Cr retention while consuming a Cr supplement in conjunction with 5 g or 50 g of carbohydrates. The mechanism explaining this phenomena is that even though glucose is the primary regulator of pancreatic insulin release several proteins have been found to stimulate insulin secretion (Spiller, Jensen, Pattison, Chuck, Whittam, & Scala, 1987; Steenge, Simpson, & Greenhaff) and the consumption of proteins in conjunction with carbohydrates can result in greater increases in serum insulin concentrations than would be expected from summing individual serum insulin responses from the consumption of protein or carbohydrates independent of each other (Steenge, Simpson, & Greenhaff). The results of the Steenge Simpson and Greenhaff study suggest the consumption of protein in conjunction with carbohydrates has an insulin-potentiating and blood glucose-moderating effect (Steenge, Simpson, & Greenhaff), as insulin promotes the entrance of P_i into muscle tissue as a result of insulin release (Green, Hultman, Macdonald, Sewell, & Greenhaff, 1996).

The effects of Cr retention from insulin release are reduced within 24 hours of the initial consumption of a Cr supplement thus the longer a Cr supplement is consumed the less effect insulin has on Cr retention (Steenge, Simpson, & Greenhaff, 2000). As a whole Cr retention may diminish over time as the body nears maximal Cr storage capacity (Rawson & Volek, 2003). However research has found the consumption of 5 g

of Cr in conjunction with 94 g of carbohydrates four times a day over a period of 5 days to increase total muscle Cr accumulation in participants by 62.7% over the amount of Cr retained in participants who consumed a Cr supplement without carbohydrates (Green, Hultman, Macdonald, Sewell, & Greenhaff, 1996).

Insulin, insulin-like growth factor I, triiodothyronine and amylin have also been shown to stimulate sodium-potassium ATPase pump activity enhancing Cr transport in muscle cells (Persky & Brazeau, 2001; Steenge, Simpson, & Greenhaff, 2000). Cr primarily enters muscles by binding to a specific transporter protein and is transported into muscles against a high-concentration gradient (Guerrero-Onriveros & Wallimann, 1998; Steenge, Simpson, & Greenhaff). However the amount of Cr transporter protein may be reduced when the body is exposed to prolonged periods of high plasma Cr concentrations, as such transporter proteins may serve as a regulator of muscle Cr content (Steenge, Simpson, & Greenhaff). Studies have also suggested that a slowing of muscle Cr accumulation and or transport occurs in comparable amounts with an increase in muscle Cr stores (Harris, Soderlund, & Hultman, 1992; Loike, Zalutsky, Kaback, Miranda, & Silverstein, 1988). Additionally sodium-potassium pump inhibition has been found to occur during prolonged periods of high plasma Cr concentrations resulting in inhibition of cellular Cr transport into muscle tissue, an expected response because Cr transport into muscle is sodium dependent (Steenge, Simpson, & Greenhaff).

Population Differences Effect Creatine Supplementation

Cr absorption in response to supplementation is also affected by population characteristics. Vegetarians have lower endogenous Cr levels and have been found to benefit more from Cr supplementation than individuals who regularly consume meat

(Shrier, 2004). The elderly population (> 68 years) have also been found to possess lower endogenous Cr levels than the younger population (Rawson, Clarkson, Price, & Miles, 2002; Rawson & Clarkson, 2000). The elderly may have lower endogenous Cr levels because of lower levels of dietary Cr consumption.

Differences in Cr absorption rates have also been observed between young and old subjects (> 60 years) in which younger populations are better able to absorb Cr than older populations (Rawson & Clarkson, 1999; Rawson, Clarkson, Price, & Miles 2002). Older populations may also become less efficient at storing Cr because of decreased Cr absorption in the gut, less proficient Cr transport in the blood and or less effective muscle Cr uptake (Rawson, Clarkson, Price, & Miles). Therefore older individuals may require longer supplementation periods or may need to consume higher doses of Cr to reach saturation levels in muscles (Rawson & Clarkson). Cr supplementation has been found effective at increasing performance in the elderly but results are not as impressive as what has been found in younger populations (Rawson & Clarkson).

Concerns of Consuming a Supplement

In accordance with the Dietary Supplement Health and Education Act (DSHEA) of 1994 the Food and Drug Administration (FDA) is no longer responsible for regulating the effectiveness or safety of dietary supplements (Congeni & Miller, 2002). A dietary supplement is defined as a product taken by mouth that contains a “dietary ingredient” intended to supplement the diet. Dietary ingredients can include vitamins, minerals, herbs, other botanicals, amino acids, and substrates such as enzymes, organ tissues, glandulars and metabolites. Under the DSHEA bill the law does not require manufacturers and/or distributors of supplements to investigate or forward reports they

receive of injuries or illnesses that may be related to the use of their products to the FDA. Moreover the FDA cannot remove a supplement from the market until they provide evidence suggesting the supplement is unsafe (U.S. Food and Drug Administration, 2001).

Under current regulations the FDA does not ensure supplements contain the ingredients manufacturers place on the label and as of 2001 the FDA had not established a minimum standard of practice for supplement manufacturers (U.S. Food and Drug Administration, 2001). Manufacturers must only provide evidence to the FDA suggesting a supplement is reasonably safe before introducing the product to the market. Additionally no evidence must be presented to the FDA suggesting the safety of a supplement if the dietary ingredient(s) is found in foods or the ingredient(s) was marketed before October 15, 1994 (U.S. Food and Drug Administration, 2001); Cr falls under each of these categories. It is also important to note the FDA does not regulate serving size or amount of a nutrient in a supplement (U.S. Food and Drug Administration, 2001). As a result most companies suggest a loading phase of consuming 20-25 grams of Cr a day for a period of 4 to 5 days followed by a maintenance dosage of 5 grams a day (Juhn, 1999). A loading phase of 10 grams a day for 4 days has been shown to sufficiently load the body with Cr and a maintenance phase of 2 g or more specifically 0.03 g/kg of body weight per day is enough to maintain maximal muscle Cr concentration (Juhn, O’Kane, & Vinci, 1999). Research has also found the loading phase of Cr supplementation to be unnecessary as supplementation with 0.3 g*kg⁻¹*d⁻¹ for a 5 day period has been found to elevate muscle total Cr and PCr by 10-25% (Syrotuik & Bell, 2004).

Possible Negative Effects of Creatine Supplementation

As of 1999 the FDA officially logged 32 complaints regarding Cr supplementation including seizure, cardiac arrhythmia, cardiomyopathy, deep venous thrombosis, rhabdomyolysis and death. However, no conclusions have been made linking the complaints in these reports to Cr supplementation (Juhn, O'Kane, & Vinci, 1999), moreover most research conducted on Cr supplementation has no reported adverse side effects except weight gain viewed as a negative (Juan, 1999). In 1999 Haff, Kirksey and Stone reported that 80 research articles in peer-reviewed journals and approximately 70 papers presented at professional conferences only one demonstrated the occurrence of side effects from Cr supplementation.

Despite accumulating evidence suggesting the safety of short term Cr supplementation the media has reported anecdotal claims suggesting Cr supplementation can increase the occurrence of muscle cramps, muscle strains, increase renal stress and cause liver damage. An apparent fallacy of Cr supplementation is that individuals who consume Cr and train in hot and humid climates experience an increased likelihood of muscle cramps. A study by Williams and Brach (1998) suggests that Cr supplementation results in a fluid balance shift where more water enters muscle cells which could theoretically alter electrolyte balance, promote dehydration and possibly increase thermal stress. However a study by Kilduff et al. (2004) provides evidence contradictory to this hypothesis. Participants consisted of 21 endurance trained males who performed two tests to exhaustion at 63 +/- 5% of VO_2 max. During the first trial participants were not given any supplements. For 7 days before the second trial participants consumed either 20 g a day of a Cr supplement or a placebo and exercised in a hot environment (30.3 +/- 0.5 C).

As a whole Cr supplementation did not significantly improve performance, but did increase intracellular water and reduced thermoregulatory and cardiovascular responses by decreasing heart rate, rectal temperature and sweat rate compared to the placebo group. Additionally participants identified as responders significantly improved time to exhaustion.

Haff, Kirksey and Stone (1999) found no data from scientific literature suggesting that muscle cramping occurs with Cr supplementation. In a study examining the effects of Cr supplementation during training and the incidence of muscle cramping, injuries, and gastrointestinal distress on 164 athletes of which 80 consumed a Cr supplement. No injuries or muscle cramping were reported during the study (Kreider, Rasmussen, Ransom, & Almada, 1998). Moreover in a 3.5 year retrospective study on the long term health effects of Cr supplementation there were no reported incidence of muscle cramping from any participants (Schilling, Fry, Kearney, Smith, O'Bryant, Utter, et al., 1998). The current literature on Cr supplementation does not support the claim that Cr supplementation increases the likelihood of muscle cramps (Haff, Kirksey, & Stone).

Another concern is that Cr supplementation may increase renal stress and possibly damage the liver as high protein diets have been shown to impair kidney function. The ingestion of a high protein meal or a solution of amino acids can increase renal blood flow and the glomerular filtration rate (GFR) by up to 20%, placing added stress on the kidneys (Culpepper, 1998). Animal models of renal injury suggest that high protein diets increase proteinuria which slows the decline in the GFR (Culpepper). As of 1998 it was unknown if Cr supplementation as a result of amino acid metabolism could produce

similar effects (Culpepper) but the only link of Cr supplementation and renal stress has been reported in a male with pre-existing liver failure (Pritchard & Kalra, 1998).

In 1998, the *Lancet* published a case study of a 25 year old male with focal segmental glomerulosclerosis and frequently relapsing steroid-responsive nephritic syndrome who experienced an adverse side effect to Cr supplementation. The patient had been consuming a Cr supplement at the suggested dose for 7 weeks and during this period doctors diagnosed him with renal dysfunction. Upon advice from his doctors the male stopped using the Cr supplement and his kidneys began to function properly. Results from this study provide researchers with strong evidence that Cr supplementation was responsible for renal dysfunction in this case (Pritchard & Kalra, 1998). However, Haff, Kirksey and Stone (1999) identified several clinical studies (Alamada, Mitchell, & Earnest, 1996; Earnest, Alamada, & Mitchell, 1996; Poortmans, Auquier, Renaut, Durussel, Saugy, & Brisson, 1997; Poortmans & Francaux, 1998) that have not shown Cr to negatively affect liver or kidney function. Additionally Poortmans and Francaux (1998) found Cr supplementation to have no effect on markers of renal stress. In a 3.5 year retrospective study on Cr supplementation researchers reported no increases in markers of renal stress (Schilling et al., 1998). The current body of literature suggests there is no significant increase in renal stress when a Cr supplement is consumed at the suggested dosage for up to 3.5 years.

The last concern from the media surrounding Cr supplementation involves the possible long term side effects. Research is not widely available on the long term effects of Cr supplementation but Cr has been prescribed as a long term treatment for medical conditions in which no side effects have been reported (Sipila, Rapola, Simell, & Vannas,

1981; See also Haff, Kirksey, & Stone; 1999). Cr has been administered to participants with gyrate atrophy of the choroids and retina daily at a dose of 1.5 g per day for 1 year (Sipila, Rapola, Simell, & Vannas). Cr has also been sold as a sport supplement since the 1980's and no apparent long term side effects have surfaced, but more research is needed before scientists can proclaim the long term use of Cr supplements as safe.

There was a concern in the scientific community involving the effects of Cr supplementation on blood pressure, because Cr is an osmotically active substance it raises the amount of water present in muscle cells which increases intracellular water and blood volume which may result in increased blood pressure. Peeters, Lantz and Mayhew (1999) examined the effects of Cr supplementation on blood pressure over a 6 week period in 34 strength trained males (20 Cr supplementation: 11 CrM, 9 CrP and 14 placebo) and found no significant changes in blood pressure.

A valid concern surrounding Cr supplementation is that endogenous Cr production is suppressed during the course of supplementation. However, in two studies conducted by Walker endogenous Cr production resumed following cessation of Cr supplementation (Walker, 1960; Walker, 1979; See also Haff, Kirksey, & Stone; 1999). More research needs to be conducted involving the effects of Cr supplementation on the biosynthesis of endogenous Cr production particularly in the long term with an emphasis placed on new steady-state relations that may occur between creatinine and Cr during periods of prolonged supplementation (Culpepper, 1998).

Cr appears to be safe when cycled with periods of consumption and non-consumption. The most common short term side effects of Cr supplementation that have been reported are diarrhea, muscle cramps, dehydration and weight gain viewed as a

negative effect. In one study involving 52 male collegiate athletes (28 football and 24 baseball players) side effects were self reported as follows: 16 diarrhea, 13 muscle cramps, although only two athletes reported experiencing a sprain or muscle tear, 7 experienced weight gain viewed as a negative, 7 reported dehydration, 12 reported other effects and 14 reported no negative side effects. It must be noted 39 participants exceeded the recommended maintenance dose of 2 g to 5 g per day with the most common maintenance dose being 6 g to 8 g per day; while 18 participants consumed 9 g or more per day and 18 participants consumed 17 g to 20 g per day as a maintenance dose. Despite the reported side effects, 40 athletes (77%) reported they plan to continue using Cr or plan on using Cr in the future (Juhn, O'Kane, & Vinci, 1999).

In a similar study on the perceived effects of Cr supplementation on division I collegiate athletes similar results were found. Of the 219 athletes surveyed 90 athletes (41%) reported using a Cr supplement and 80 athletes (89%) reported perceived positive effects while 34 athletes (38%) reported perceived negative side effects and 10 athletes (11%) reported no effects. It is important to note every athlete who reported a perceived negative effect of Cr supplementation also reported a positive effect. In each group there was a trend to use a lower than recommended loading phase dose and a higher than recommended maintenance phase dose suggesting there is a need to education athletes on the proper use of Cr supplementation (Greenwood, Farris, Kreider, Greenwood, & Byars, 2000).

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APPENDIX C: INFORMED CONSENT

COLLEGE OF COLLEGE OF HEALTH AND HUMAN SCIENCES

DEPARTMENT OF JIANN-PING HSU SCHOOL OF PUBLIC HEALTH

INFORMED CONSENT

I understand that study I am about to participate in part of a research project entitled The Relationship between Endogenous Creatine Levels, Maximal Upper Body Strength, Body Fat and Short-Term Muscle Recovery in Males, conducted by Vinny Dalbo (912-871-1991), a graduate student at Georgia Southern University. The purpose of this study is to examine if endogenous (naturally occurring) creatine levels present in the human body can influence indices of athletic performance and general health.

Participation in this research study requires two meetings with the experimenter over a four day period. Participation will include completion of a three day food diary and a demographic questionnaire during the first meeting. The second meeting requires a blood test of less than 10 mL of blood, an eight hour fast, measurement of height, weight, and body fat percentage (using the Jackson-Pollack 3-site skin fold assessment). Participants will also be asked to complete a one repetition maximum bench press test, and five repeated maximal effort bench press lifts with 80% of the participant's one repetition maximum, with two minute rest intervals between sets.

It is not believed nor are the three day food diary or demographic questionnaire intended to cause any psychological discomfort to any participant. Minimal amounts of physical discomfort may be experienced during the blood

test, which will be conducted by a licensed phlebotomist and the exercise portion of the test. Participants will be asked to exert maximal effort during the one repetition maximum test and the muscle recovery portion of the test. Discomfort experienced from lifts should be minimal as participants should have at least one year weight training experience and should be accustomed to weight training. “I understand that medical care is available in the event of injury resulting from research but that neither financial compensation nor free medical treatment is provided. I also understand that I am not waiving any rights that I may have against the University for injury resulting from negligence of the University or investigators.” For those who wish to seek assistance following completion of the study may contact Vinny Dalbo (912-871-1991), the Counseling Center (912-681-5541) and or Health Services.

I understand that any relationship between myself and the information I contribute to this study will be kept confidential. I understand that I may stop answering any question at any time without prejudice to myself, course grade, employment status or any other personal matter. For students in which extra credit is offered for completion of the study, I understand that by answering all of the questions in the assessment and completing the physical aspect of the test, I may be able to receive extra credit to my final numerical grade. I understand that if I do terminate the study, I will not receive extra credit, but will be allowed to complete the compensatory extra credit assignment.

As a participant you have the right to ask questions and have those questions answered. If you have questions about this study, please contact Vinny Dalbo

(912-871-1991) or Dr. Jim McMillan (912-871-1926). For questions concerning your rights as a research participant, contact Georgia Southern University Office of Research Services and Sponsored Programs (912-486-7758).

No monetary compensation is offered for participation in this study and participation is voluntary. You the participant are in no way required to participate in this study and may end your participation at any time by telling the person in charge or not returning the questionnaires. You also reserve the right to not answer any question(s) you do not want to answer. You may chose to not participate, stop participation, and or not answer any question(s) without penalty.

You must be 18 years of age or older to consent to participate in this research study. If you consent to participate in this research study and to the terms above, please sign your name and indicate the date below. You will be given a copy of this consent form to keep for your records.

Title of Project: The Relationship between Endogenous Creatine Levels, Maximal Upper Body Strength, Body Fat, and Short Term Muscle Recovery in Males
 Principal Investigator: Vinny Dalbo, 2218 Hanner Field House, telephone: 912-871-1991, vincent_j_dalbo@GeorgiaSouthern.edu
 Faculty Advisor: Dr. Jim McMillan, Hollis Building, 912-871-1926, jmcmillan@GeorgiaSouthern.edu

 Participant Signature

 Date

I, the undersigned, verify that the above informed consent procedure has been followed.

 Investigator Signature

 Date

APPENDIX D: THREE DAY FOOD DIARY

Name:

Day 1 (Use the back of the sheet if needed.)

	Food Type (Include Drinks) Include brand and names (eg. Kellogg's Oatbran cereal, KFC chicken wings Ocean Spray Cranberry Juice)	Food Preparation (Ingredient list, type of preparation: baked or fried, etc)	Serving Size/number of servings (include ounces, fluid ounces, milli – liters, cups, teaspoons, etc)
Breakfast			
Lunch			
Dinner			
Snacks			

Maintain a 3-day food record on three consecutive days. Be sure to include extras (i.e. – sugar in tea, cream in coffee, butter on popcorn, etc.) when recording your intake. Estimate the amount to the nearest common household measure such as teaspoon, cup or ounces. Please ensure your records are accurate as possible.

Name:

Day 2 (Use the back of the sheet if needed.)

	Food Type (Include Drinks) Include brand and names (eg. Kellogg's Oatbran cereal, KFC chicken wings Ocean Spray Cranberry Juice)	Food Preparation (Ingredient list, type of preparation: baked or fried, etc)	Serving Size/number of servings (include ounces, fluid ounces, milli – liters, cups, teaspoons, etc)
Breakfast			
Lunch			
Dinner			
Snacks			

Maintain a 3-day food record on three consecutive days. Be sure to include extras (i.e. – sugar in tea, cream in coffee, butter on popcorn, etc.) when recording your intake. Estimate the amount to the nearest common household measure such as teaspoon, cup or ounces. Please ensure your records are accurate as possible.

Name:

Day 3 (Use the back of the sheet if needed.)

	Food Type (Include Drinks) Include brand and names (eg. Kellogg's Oatbran cereal, KFC chicken wings Ocean Spray Cranberry Juice)	Food Preparation (Ingredient list, type of preparation: baked or fried, etc)	Serving Size/number of servings (include ounces, fluid ounces, milli – liters, cups, teaspoons, etc)
Breakfast			
Lunch			
Dinner			
Snacks			

Maintain a 3-day food record on three consecutive days. Be sure to include extras (i.e. – sugar in tea, cream in coffee, butter on popcorn, etc.) when recording your intake. Estimate the amount to the nearest common household measure such as teaspoon, cup or ounces. Please ensure your records are accurate as possible.

APPENDIX E: DEMOGRAPHIC QUESTIONNAIRE

Demographic Questionnaire

ID# _____

Section I

1. Age (in years): _____
2. Gender - Male
3. How many years have you been weight training: _____
4. Are you currently on a weight training regimen? Yes No

If yes answer questions 5, 6, 7, 8

If no answer question 9

5. What type of rep ranges are you training with most frequently (circle the response that most applies)?

High weight low reps	Moderate weight and reps	Low weight high reps
(1-8 reps)	(8-12 reps)	(12 or more reps)

6. What is the average break (rest period) you take between sets in minutes? _____

7. How many days per week do you currently lift weights?

1 2 3 4 5 6 7

8. Have you consistently been lifting weights 3 or more days per week for the last 30 days?

Yes No

9. How many months have passed since you consistently lifted weights 3 or more days per week for 30 days? _____

Section II

1. Have you ever consumed a sport supplement? Yes No

2. Have you ever consumed a supplement containing creatine? Yes No

3. Have you consumed a sport supplement in the past 2 months? Yes No

4. If yes what supplement(s)?

5. Have you consumed a supplement containing creatine in the past 4 months? Yes No

If yes what supplement(s)?

6. When was the last time you consumed a sport supplement (answer in months)?

7. What was the last sport supplement you consumed?

8. On average how many days per week do you weight train?

1 2 3 4 5 6 7

9. On average how many days per week do you exercise?

1 2 3 4 5 6 7

10. Are you a hemophiliac? (trouble stopping bleeding) Yes No

11. Do you have diabetes? Yes No

12. Do you have any liver problems? Yes No

13. If yes, what problems do you have:

14. Do you have any kidney problems? Yes No

15. If yes, what problems do you have:

16. Do you have any blood born illnesses? Yes No

DATA SHEET

ID # _____

Height _____

Weight _____

Skin fold measurements

	First	Second	Third	Average
Chest				
Abdomen				
Thigh				

1RM _____

80% 1RM _____

Set #	# of Reps
1	
2	
3	
4	
5	

APPENDIX F: NSCA BENCH PRESS STANDARDS

Proper Bench Press and Spotting Technique

Beginning Position: Athlete

Starting position: Assume supine position on a bench in a five-point body contact position. Place body on bench so that eyes are below the edge of the supports. Grasp the bar with a closed pronated grip. Grip should be slightly wider than shoulder-width. Signal the spotter for assistance in moving the bar off the supports. Place the bar over the chest with elbows fully extended. All subsequent repetitions begin from this position.

Downward Movement Phase: Athlete

Lower the bar to touch the chest at approximately nipple level. Keep the wrists ridged and directly above the elbows. Maintain the five-point body contact position.

Upward Movement Phase: Athlete

Push the bar upward until the elbows are fully extended. Keep wrists ridged and directly above the elbows. Maintain five-point body contact position. Do not arch back or raise chest to meet the bar. After the set is completed, signal the spotter for assistance in racking the bar. Keep a grip on the bar until it is racked.

Beginning Position: Spotter

Stand erect and very close to the head of the bench. Place feet should-width apart with knees slightly flexed. Grasp the bar with a closed, alternated grip inside the athlete's hands. At athlete's signal, assist in moving the bar off the supports. Guide the bar to a position over the athlete's chest. Release the bar smoothly.

Downward Movement Phase: Spotter

Keep hands in the alternated grip position close to-but not touching-the bar as it descends. Slightly flex the knees, hips, and torso and keep the back flat when following the bar.

Upward Movement Phase: Spotter

Keep the hands in the alternated grip position close to-but not touching-the bar as it ascends. Slightly extend the knees, hips, and torso and keep the back flat when following the bar. At the athlete's signal after the set is completed, grasp the bar with an alternated grip inside the athlete's hands. Guide the bar back onto the supports. Keep a grip on the bar until it is racked.

Note. From *Essential of strength training and conditioning* (2nd ed.) (p.359), by T. R. Baechle and R. W. Earle, 2000, Champaign: National Strength and Conditioning Association.

APPENDIX G: JACKSON POLLACK 3-SITE SKINFOLD EQUATION

Jackson Pollock 3-Site Skin Fold Equation

Male 3-Site Equation (SUM3 = chest, abdomen, thigh)

$$D_b = 1.10938 - (0.0008267 * \text{SUM3}) + (0.0000016 * \text{SUM3}^2) - (0.000257 * \text{AGE})$$

$$\%BF = [(4.57 / D_b) - 4.142] * 100$$

Body Density

$$\text{Black Male Age 19-45} = (4.86) / D_b - 4.39$$

$$\text{White Male Age 17-19} = (4.99) / D_b - 4.55$$

$$\text{White Male Age 20-80} = (4.95) / D_b - 4.50$$

Jackson, A.S., & M.L. Pollack. Practical Assessment of body composition. *Physician Sport Medicine*, 13:76-90, 1985. (As published in: Nieman DC: The Sports Medicine Fitness Course. Bull Publishers. Palo Alto, 1986, pp. 101.

APPENDIX H: NSCA ONE REPETITION MAXIMUM BENCH PRESS PROTOCOL

NSCA One Repetition Maximum Bench Press Protocol

Equipment

- Olympic-style weightlifting set with enough total weight to accommodate the maximum lift of the strongest athlete and a variety of plate sizes to allow for 5-lb gradations in weight.
- A sturdy bench-press bench with integral bar rack of adjustable height.

Procedure

1. Instruct the athlete to warm-up with a light resistance that easily allows 5-10 repetitions.
2. Provide a 1-minute rest period.
3. Estimate a warm-up load that will allow the athlete to complete 3-5 repetitions by adding
 - 10-20 lb or 5-10%
4. Provide a 2-minute rest period.
5. Estimate a conservative, near-maximum load that will allow the athlete to complete 2-3 repetitions by adding
 - 10-20 lb or 5-10%
6. Provide a 2 to 4-minute rest period.
7. Make a load increase
 - 10-20 lb or 5-10%
8. Instruct the athlete to attempt a one repetition maximum lift.

9. If the athlete was successful, provide a 2 to 4-minute rest period and go back to step 7.

If the athlete failed, provide a 2 to 4-minute rest period, decrease the load by subtracting

- 5-10 lb or 2.5-5%

AND then go back to step 8.

Continue increasing or decreasing the load until the athlete can complete one repetition with proper exercise technique. Ideally, the athlete's one repetition maximum will be measured within five testing sets.

Note. From Essential of strength training and conditioning (2nd ed.) (p.409), by T. R. Baechle and R. W. Earle, 2000, Champaign: National Strength and Conditioning Association.

APPENDIX I: EXTENDED CREATININE ANALYSIS

Extended Creatinine Analysis

The first step of data analysis required making the 8 standards by combining progressively greater amounts of the creatine standard with progressively fewer amounts of HPLC-grade water in each of the 8 standard test tubes. Each of the 8 standards was placed pipetted into 2 empty wells on a 96 well plate along with the creatinine samples. Initial and final absorbance values were recorded at 492 nm on a plate reader. The final absorbance was subtracted from the initial absorbance on each of the 8 standards yielding the adjusted absorbance. The adjusted absorbance values for each standard was plotted and fitted with a trend line yielding the creatinine standard curve (see Figure 2)

The next step was to subtract the final absorbance values from the initial absorbance values to determine the adjusted absorbance value for each creatinine sample. The adjusted absorbance values for each creatinine sample were plugged into the formula seen in Figure 3 yielding the creatine values in mg/dl for each participant.

Figure 11. Creatinine standard curve

$$y = 0.0406x - 0.0016$$

$$r^2 = 0.9993$$

Figure 12. Creatinine determination formula

$$\text{Creatinine (mg/dl)} = ((\text{sample absorbance} - \text{y intercept}) / \text{slope})$$

ADDITIONAL REVIEW OF LITERATURE REFERENCES

Baechle, T. R., & Earle, R. W. (2000). *Essential of strength training and conditioning*

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