THE EFFECT OF CREATINE MONOHYDRATE SUPPLEMENTATION ON SPRINT SKATING IN HOCKEY PLAYERS

A Thesis Submitted to the College of

Graduate Studies and Research

In Partial Fulfillment of the Requirements

For the Degree of Master of Science

In the College of Kinesiology

University of Saskatchewan

Saskatoon

By

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Spring 2001

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ABSTRACT

The purpose of this study was to assess the effectiveness of creatine monohydrate supplementation for improving sprint skating performance in competitive male ice hockey players. In a double blind and randomized design, 17 ice hockey players were supplemented with either 0.30 grams of creatine monohydrate (Cr) per kilogram of body weight per day (n = 8; mean ± SE, age = 20.3 ± 0.7 years), or a placebo (Pl) (n = 6; age = 18.4 ± 0.4 years) for 5 days. One day prior to supplementation and one day after supplementation subjects performed repeated sprint skating intervals on the Frappier Acceleration Skating Treadmill. Testing consisted of skating at 16.1 km/hr at a 15% elevation for 10 seconds, resting for 30 seconds and repeating the same procedure until volitional fatigue. Blood lactate concentration (La) was taken at baseline, after each odd numbered skating interval, and again upon volitional fatigue. There was no significant difference in mean total skating time between groups prior to treatment (45.2 ± 12.9 vs. 63.5 ± 12.4 seconds, Pl vs. Cr). Post-test results showed no significant difference in total skating time between groups (67.1 ± 23.3 vs. 84.1 ± 14.0 seconds, Pl vs. Cr). There were no significant differences in La concentration at baseline, after any of the intervals, or fatigue between groups prior to and after supplementation. In conclusion, in this study there was no objective evidence to support the contention that Cr supplementation improves the ability to perform repeated sprint skates in competitive male hockey players.

ACKOWLEDGEMENTS

I wish to extend my sincere thanks and appreciation to my advisor, Dr. Phil Chilibeck and the members of my committee, Dr. Bob Faulkner and Dr. Eric Sprigings. Their time and dedication in helping me reach my goals is greatly treasured. Also, a sincere thanks is extended to Dr. Susan Whiting for her input as an external examiner.

I also wish to thank MuscleTech Research and Development for providing the supplements for this research project.

Thank you to the many individuals who helped to collect data and provided guidance in equipment use and maintenance. Thanks to Carisa Polischuk for monitoring the double blind key and mixing all of the supplements for each subject. Thanks to Heather Whelan and Darren Burke for their assistance in data collection. Without this help the project would not have been possible.

I would also like to extend my sincere thanks to my parents, Gary and Barb, for their continual love and support throughout my years receiving education. Finally, I thank my wife Denise for her continual love and support while I finished obtaining this degree.

DEDICATION

This thesis is dedicated to my family: my wife Denise, my father Gary, my mother Barb, my sister Lori, my brother Jason, my brother Don, and my sister Arla. I feel very blessed to have such a wonderful and loving family who supports me in all that I do. The love that I receive from all of you is what makes me appreciate life to its fullest. This thesis is also dedicated to my Lord and Savior Jesus Christ; without His eternal grace and love none of this would have been possible.

"Do not be deceived, God is not mocked; for whatever a man sows, that he will also reap. For he who sows to his flesh will of the flesh reap corruption, but he who sows to the Spirit will of the Spirit reap everlasting life." Galatians 6:7-8

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CHAPTER 1

SCIENTIFIC FRAMEWORK

1.1 Introduction

In recent years there has been an increased interest in the use of creatine monohydrate as a nutritional ergogenic supplement for athletes in strength and power sports. Creatine monohydrate or creatine (Cr) supplementation has been shown to increase phosphocreatine (PCr) stores within the muscle with a corresponding increase in the ability of muscle to sustain high intensity, anaerobic type exercise (Balsom et al., 1995). Research to date has reported conflicting findings but the majority of the publications indicate that Cr ingestion has improved repeated anaerobic sprint activities.

Mujika & Padilla (1997) define an ergogenic aid as "any means to enhance energy utilization, including energy production, control, and efficiency." Creatine is considered a nutritional ergogenic supplement because it is a substance found commonly in meat. A 1.1 kilogram raw steak contains approximately 5 grams of Cr (Harris et al., 1992). The International Olympic Committee (IOC) has not yet put creatine on the banned list of restricted products. Many amateur and professional athletes are reportedly using Cr as a nutritional supplement to aid their performance (Stanton & Abt, 2000). Some authors believe that Cr supplementation is analogous to carbohydrate loading and that it is a safe and effective means of increasing PCr stores for improved exercise performance (Volek & Kraemer, 1996).

A majority of the research on Cr supplementation has evaluated its effectiveness as an ergogenic aid. A large portion of these studies have been laboratory based and not done in a sport-specific situation. Mujika and Padilla (1997) state that little is known

about the effects Cr supplementation may have on highly trained athletes and the movements that are specific to their sport.

Ice hockey is one sport that relies predominantly on anaerobic energy production (Montgomery, 1988). As such, Cr supplementation would theoretically aid in exercise performance and recovery. Jones, Atter, and Georg (1999) evaluated the effects of Cr supplementation on repeated sprint skating (6 x 80 meters with a work to rest ratio of approximately 1:1) in elite ice-hockey players in Europe. The test involved skating forward for 47 meters, changing direction, and skating backward for the final 33 meters. They found a significant decrease in forward skating time with the creatine group after 10 days and 10 weeks of creatine supplementation (20 grams/day x 5 days + 5 grams/day x 10 weeks). The authors were unclear as to why there was not an overall improvement in sprint skating speed but suggested that the technical factors involved in stopping and changing direction may have confounded the results. This study has shown conflicting results in that creatine supplementation apparently improved forward skating speed but not backward skating speed. A study done to eliminate the confounding factor (i.e. change of direction) would more appropriately measure the effectiveness of creatine supplementation to enhance skating performance.

There are many sports that require a specific movement pattern to be successful. Ice hockey requires athletes to be involved in high intensity intermittent, anaerobic exercise utilizing a specific movement sequence. It is important to determine whether creatine supplementation will have an ergogenic effect on skating performance in these athletes to determine if it is beneficial to this type of athletic performance.

1.2 Review of Literature

1.2.1 Creatine

Approximately 1 gram of creatine is produced endogenously each day and, depending upon one's diet, approximately 1-2 grams is ingested in a mixed diet exogenously (Balsom et al., 1994). The synthesis of creatine endogenously is controlled by the amidinotransferase enzyme activity and mostly occurs in the liver but some is produced in the kidneys and pancreas as well (Walker, 1979). The biochemical process as explained by Kreider (1998) is as follows. Glycine and arginine combine to form guanidinoacetate. Guanidinoacetate and ornithine combine with adenyosylmethionine to produce adenosyhomocysteine and creatine. If an individual does not consume meat as part of their daily diet (i.e. vegans), the endogenous production of Cr must increase to meet daily requirements.

There is between 120-125 mmol/kg dry mass of total creatine (TCr) found normally in human muscle (Greenhaff, 1995; Greenhaff, 1997). Approximately 60-70% of the TCr is stored as PCr and the remaining 30-40% as Cr. There is between 1-2 grams of creatine converted non-enzymatically to creatinine and excreted in urine each day. This loss is maintained by the endogenous production (~ 1 gram/day) and exogenous consumption (~ 1-2 grams/day) of Cr each day. During high intensity anaerobic activity the ATP stored within the muscle is utilized for muscle contraction while PCr is utilized to maintain an adequate ATP concentration. There is approximately three times more PCr that ATP stored within skeletal muscle and during intense anaerobic activity

there is a 80% reduction in PCr concentration while ATP cocnentration remains relatively constant (Miller et al., 1987; Miller et al., 1988; Tesch et al., 1986).

Ingestion of large amounts (20 grams/day or greater) of Cr has been shown to increase the intramuscular stores of Cr and PCr (Harris et al,1993). Typically athletes who ingest a large amount of creatine exogenously will initially go through a loading phase to elevate the level of TCr to its "maximum" level in the muscle. Several researchers suggest this maximum to be between 150-160 mmol/kg dry mass (Harris et al., 1992; Greenhaff et al., 1995; Clark, 1997). Although this increase may occur in some people it may not occur in others. Greenhaff (1994) studied the effects of Cr ingestion on the concentrations of ATP, PCr, Cr, and TCr. He classified his subjects as "responders" and "non-responders". Responders increased their TCr concentration by 20 mmol/kg dry mass or greater and non-responders increased their TCr cocnetration by less than this amount. It is possible that those who eat large amounts of meat may not benefit as much from Cr supplementation because of the elevated level of Cr already present in their bodies. Therefore, it seems the human body is only able to retain a limited amount of Cr and PCr in the skeletal muscles. The production of creatine transport proteins has been shown to decrease with long term ingestion of Cr supplements (Wallimann, 1992); thus, long term, supplemental ingestion may hamper the ability of the body to store and utilize creatine.

Ingesting Cr supplements orally results in the absorption of Cr intact from the intestine (Clark, 1997). Once in the blood stream, 95% of Cr is actively transported into skeletal muscle cells where 60-70% of it is converted to phosphocreatine (PCr) and 30-40% remains as free creatine (Greenhaff, 1997). PCr is used by skeletal muscle for the

resynthesis of adenosine triphosphate (ATP) from adenosine diphosopahte (ADP). ATP is the primary energy source for contracting skeletal muscle. PCr is primarily responsible for ensuring that the concentration of ATP stays relatively constant during anaerobic exercise. It has been shown that during high intensity exercise of short duration, PCr levels are reduced dramatically while ATP levels remain relatively constant (Houston, 1995). Once PCr levels are reduced to nil, then ATP levels would be reduced and exercise intensity could not be sustained because of increased reliance for energy from glycolysis or oxidative phosphorylation.

1.2.2 Possible Mechanisms for Creatine's Ergogenic Effect

Creatine and PCr are used in skeletal muscle in the following reaction:

$$ATP + Cr \overset{CK}{\Leftrightarrow} ADP + PCr + H^{+}$$

As can be seen, creatine kinase (CK) is responsible for the breakdown of PCr which in turn releases the energy necessary to reform ATP. Wallimann et al. (1992) lists several functions of the CK/PCr system. The main function of the CK enzyme reaction is to maintain ATP concentration via its high power buffering activity. Although this system has powerful buffering capabilities, the capacity of the system is low because of the limited supply of PCr. It has been found that supplementing with Cr increases the concentration of Cr and PCr within the muscle (Harris et al., 1992). Greenhaff et al. (1993) has since postulated that the increased concentration of PCr as a result of Cr supplementation may be responsible for maintaining the concentration of ATP to a greater degree during high intensity muscular contraction.

A second function of the CK/PCr system is as an energy transport system where PCr serves as an "energy carrier" for high energy phosphates from the mitochondria to sites of ATP utilization. There are 3 main CK isoenzymes that are involved in this "energy carrier" system. One isoenzyme is associated with actomyosin ATPase and utilizes PCr to regenerate ATP during activation and shortening of the muscle (i.e. actin activated myosin ATPase activity). The second isoenzyme that utilizes PCr is associated with the sodium-potassium ATPase at the muscle plasma membrane, which is responsible for pumping Na^+ ions out and K^+ ions in through the sarcolemma after a muscle membrane depolarization. Finally, there is a CK isoenzyme associated with the sarcoplasmic reticulum Ca^{2+} ATPase isoenzyme that pumps Ca^{2+} ions back into the sarcoplasmic reticulum when the muscle relaxes. All three of these CK isoenzymes utilize PCr to restore the depleted ATP concentration at these sites of ATP utilization. An elevated concentration of PCr within the muscle should theoretically improve the ability of these isoenzymes to function.

A third function of CK/PCr system is to reduce the intracellular ADP cocentration. A build-up of ADP from the breakdown of ATP is associated with a stimulation of glycolysis, increased muscle acidosis, and fatigue. With resynthesis of ATP from ADP and PCr, a reduction in the build up of ADP is accomplished, preventing acidic states and preventing fatigue.

A fourth function of the CK/PCr system is to buffer protons. Hydrogen ions (H^+) are consumed in the conversion of ADP and PCr to ATP and Cr. The use of H^+ in this reaction will buffer the pH level of the working muscles. A delay in the accumulation of acidity in the muscle will subsequently delay the onset of muscular fatigue due to

acidosis. If there is an elevated level of PCr in the muscle, theoretically the reaction should be able to proceed for a longer period of time while keeping the pH of the muscle neutral for longer periods. If ADP levels of the muscle are increased substantially (as during intense exercise), the adenylate kinase reaction will utilize two molecules of ADP to resynthesize one molecule of ATP, producing adenosine monophosphate (AMP) as a by-product. AMP is irreversibly broken down to inosine monophosphate (IMP) and ammonia. Ammonia accepts a proton (H^+) to form an ammonium ion and acts as a buffer in this situation. Indirect evidence that creatine supplementation prevents the build-up of ADP and acts as an intracellular buffer (in the place of ammonia) has been found by some research (Greenhaff, 1993; Birch, 1994).

Creatine has also been theorized to act as an ergogenic aid through one final mechanism. An increased concentration of Cr and PCr in the muscle increases the amount of fluid within the cell. This is theorized to increase the rate of protein synthesis within the muscle which increases the diameter of type II muscle fibers, improving muscular performance and thus training intensity (Volek and Kraemer, 1996).

1.2.3 Creatine and Endurance Exercise

The majority of studies evaluating the effects of creatine supplementation on aerobic exercise tasks report no beneficial ergogenic effect. Creatine has been shown to have no effect on maximal oxygen consumption (VO_2 max) (Nelson et al., 1998; Barnett et al., 1996), time to completion of a 25 kilometre cycling race (Godly and Yates, 1997), or distance cycled in 1 hour (Myburgh et al., 1996).

There are however two studies indicating that creatine supplementation may be ergogenic in nature for aerobic type activity. Engelhardt et al. (1998) developed a cycling test comprising aerobic work bouts interspersed with interval work for 12 national caliber triathletes. The design was a single group repeated-measures with no subject blinding. The triathletes supplemented with 6 grams of creatine per day for 5 days. The results indicated that after creatine supplementation the subjects increased the amount of intervals performed from 5.7 to 8.3. The authors concluded that creatine supplementation improved intense interval work incorporated into aerobic exercise. They also made it clear that this study needs replication in a randomized double blind placebo control design to have more validity and applicability. Smith et al. (1998) studied 15 active males and females. Subjects were randomized into a placebo or creatine (20 g/day) group for 5 days. A cycling task was performed before and after supplementation and involved pedaling at 4 predetermined workloads until exhaustion. The authors found a 7.2 % improvement in time to exhaustion at 3.7 W/kg in the creatine group but not the placebo group. They concluded that creatine supplementation is beneficial to high intensity, short duration aerobic exercise.

There have also been various studies evaluating the effects of creatine supplementation on other types of laboratory and field exercise tasks. Balsom et al. (1993) found no significant differences in a running treadmill test to exhaustion or a 6 kilometer terrain run in 18 active well trained male subjects who consumed creatine (20 grams for 6 days) or placebo supplement for 6 days. Also, Thompson et al. (1996) found no improvement in swim time over 400 meters in 10 female competitive swimmers after 2 grams of creatine supplementation for 56 days. Furthermore, Stroud

et al. (1994) reported no measurable influence on substrate utilization on a graded treadmill running exercise test at workloads between 50 – 90 % of maximal oxygen uptake before or after creatine (20 grams for 5 days) and placebo supplementation.

Contrary to the above results, McNaughton et al. (1998) found a 6.6% improvement in work performed after creatine supplementation (20 grams for 5 days) versus placebo in a 300 second kayak ergometer test in 16 elite kayakers. Also, Rossiter et al. (1996) supplemented 38 male and female competitive rowers with either placebo or creatine (0.25 grams/kilogram of body mass for 5 days). They measured time to complete a simulated 1000-meter rowing task before and after supplementation. The authors reported a significant 2.3 second decrease in time to complete the task in the creatine supplemented group.

It is evident that creatine supplementation may be beneficial for high intensity, short duration aerobic exercise tasks most likely because these exercise tasks rely heavily on anaerobic energy production as well. It is unlikely that creatine ingestion provides an ergogenic advantage to those involved in long-term aerobic type activity that relies most predominantly on oxidative phosphorylation.

1.2.4 Creatine and High Intensity Continuous Exercise

Numerous researchers have studied the effects of creatine supplementation on anaerobic sprint exercise performance. There are five studies that indicate Cr supplementation has no effect on exercise performance in a single anaerobic sprint situation. Snow et al. (1998), used a double-blind crossover design to evaluate whether or not Cr supplementation (30 grams/day x 5 days) would improve a 20 second sprint

performance in 8 active untrained men on a air braked bicycle ergometer. Although muscle biopsies indicated an increase in muscle total Cr content, 20-second exercise performance was not significantly different after Cr ingestion.

In a similar study Odland et al. (1997) tested 9 men for power output during a Wingate bicycle ergometer test as well as muscle concentration of ATP, PCr, and total creatine (TCr). Each subject was tested under three randomly ordered conditions that included creatine supplementation, placebo, and control with 14 days separating each test. The Cr supplementation protocol used consisted of 20 grams/day x 3 days. No significant difference were found in 30-second bicycle sprint performance or muscle concentration of ATP, PCr or TCr. The results did indicate a higher TCr/ATP ratio in the Cr condition as opposed to the placebo and control conditions.

Chetlin et al. (1998) assessed two different amounts of Cr supplementation on exercise performance in the Wingate test. The study had thirty-three resistance trained males randomly assigned to one of three groups: 20 g creatine monohydrate x 10 days, 10 g creatine monohydrate plus dextrose x 10 days, or a placebo. Neither amount of Cr supplementation increased Wingate exercise performance.

Ruden et al. (1996) evaluated the effects of Cr supplementation on exercise performance in a Wingate test with a single-group repeated-measures design. The subjects ingested 20 g/day x 3 days of Cr monohydrate and placebo with treatments being counterbalanced. Cr supplementation did not affect power output or total work completed. In this study the washout period was 14 days which is likely too short a time period to be considered a valid study. It has been shown that creatine should not

be ingested for at least 30 days to be completely cleared from the body (Hultman et al., 1996).

A cycling ergometer study done by Jacobs et al. (1997) examined if Cr supplementation (5 grams 4 times/day x 5 days) would increase anaerobic capacity and maximum accumulated oxygen deficit more so than a placebo supplement. Subjects cycled to exhaustion at 125% of VO_2 max. before any supplementation, after 5 days of supplementation and again 7 days after cessation of supplementation. Cr ingestion increased maximal accumulated oxygen deficit from 4.04 ± 0.31 to 4.41 ± 0.34 L ($p < 0.001$) and it remained elevated 7 days after cessation of creatine supplementation (4.31 ± 0.33 L, $p < 0.001$). Time to exhaustion also increased in the Cr supplemented group from 130 ± 7 to 141 ± 7 seconds ($p < 0.01$) and remained elevated 7 days after supplementation (139 ± 8 seconds, $p < 0.01$). These data demonstrate that Cr ingestion has an ergogenic effect on cycling performance that remains for at least 7 days after cessation of supplementation. These results indicate that anaerobic capacity is improved by Cr supplementation.

Finally, a study done by Dawson et al. (1995) evaluated the ergogenic benefit of Cr in a single 10-second cycle sprint. A double blind, randomized assignment was used with 9 subjects in the Cr group and 9 in the placebo group. The subjects ingested 5 grams of creatine or placebo four times per day for five days. The amount of work completed at 2, 4, 6, 8, and 10 seconds as well as peak power was higher after supplementation versus baseline in both groups. Post-test results revealed no significant difference between groups in their performance scores. Their results suggest that short term Cr supplementation is not beneficial to single anaerobic sprint activity. This

makes sense intuitively because supplementing with Cr for a short duration will not improve the power of the anaerobic energy system. It may improve the capacity of the system and also allow the energy system to recover more quickly between sets of exercise.

1.2.5 Creatine and High Intensity Intermittent Exercise

Anaerobic power can be defined as the maximum rate of energy output during a specified time period of maximal effort exercise. The ability to sustain maximal effort exercise is dependent upon the stores of Cr and PCr in the muscle. By increasing the levels of PCr intracellularly, there is a corresponding improvement in the ability of the anaerobic energy system to produce ATP. Subjects who use Cr as a supplement have shown improved anaerobic exercise performance as compared to pre-dose performance.

Cycle ergometer anaerobic power testing is used frequently to test the ergogenic effects of Cr. Some research has focused on single effort cycling performance of high intensity and short duration to evaluate creatines ergogenic properties. Other studies have used repetitive cycle sprints to evaluate the ability of Cr to reduce the effects of fatigue.

A number of researchers have concluded that supplementation with Cr enhances exercise performance in repeated Wingate cycling tests. Birch et al. (1994), Earnest et al. (1995) and Theodoru et al. (1998) used similar protocols (3 sets of Wingate tests with 4-6 minutes rest between sets) to evaluate the ergogenic potential of creatine supplementation. These three studies utilized the following doses respectively: 20 grams/day for 5 days, 20 grams/day for 14 days, and 25 grams/day for 4 days. All these

results demonstrate that Cr ingestion enhances anaerobic performance indicators. Ledford and Branch (1999) performed a similar study as the above 3 mentioned studies but found no significant difference in peak power production or work capacity in 9 female subjects. The authors concluded that power production is unaffected by short term Cr supplementation in women. These results are in contrast to the other 3 studies and this may suggest that females do not respond to Cr supplementation as well as males do.

Three other studies (Earnest et al., 1998; Greenhaff et al., 1994; Casey et al., 1996) have evaluated the effects of Cr supplementation on 2 Wingate tests with 4-5 minutes rest between sets. These results also indicate that Cr supplementation improves power output, increases anaerobic work capacity, and improves peak power. From the above results it seems evident that creatine supplementation improves the ability of the anaerobic energy system to maintain or improve power production in human muscle.

Creatine supplementation has also been shown to reduce fatigue during high intensity intermittent cycling. Smith et al. (1998) reported an improvement in the ability to maintain power in a constant power cycling task. Subjects completed 4 work intervals on an air braked cycle ergometer task designed to elicit fatigue between 90 and 600 seconds with at least 2 hours of rest between intervals. The subjects were randomly assigned to either a creatine or placebo group and ingested either 20 grams of Cr/day or 24 grams of glucose/day. The Cr group increased the mean amount of time they were able to pedal from 93 s to 103 s in work bout 4 and from 236 s to 253 s in work bout 3 (an 11% and 7% increase respectively). The placebo group decreased the mean time to exhaustion by 6% and 10% in work bout 4 and 3 respectively.

There are numerous studies which have evaluated the effects of creatine on repeated short term (15 seconds or less) exercise performance with rest between sets. Schneider et al. (1997) evaluated the effect of Cr and placebo supplementation on work performed in 5 x 15 second cycling bouts with 60 seconds rest between sets. The nine untrained males ingested the placebo for 7 days, repeated the testing protocol and then ingested Cr for 7 days in this single blind study. The researchers found a significant increase in the work performed in each of the 15-second exercise bouts after Cr supplementation. The study also evaluated the subjects on 5 x 60-second maximal cycling exercise bouts with 5:00 minutes of rest between sets. In this procedure they found no significant ($p>0.05$) difference between the placebo and Cr exercise performance. The authors suggested that Cr supplementation may enhance the ability of the body to resynthesize ATP from PCr during maximal short-duration exercise only.

Prevost et al. (1997) studied the effects of Cr (18.75 grams/day for 5 days and 2.25 grams/day of testing) and placebo supplementation on intermittent cycling at a predetermined workload. In this study subjects pedaled at 150% peak oxygen uptake under 4 conditions: nonstop, intermittently for 60 seconds of work 120 seconds rest, intermittently for 20 seconds work 40 seconds of rest, and intermittently for 10 seconds of work and 20 seconds of rest. Each work bout was done until volitional fatigue. Significant increases were found ($p<0.01$) in all 4-work bouts in the Cr supplemented group. There was a >100% increase in work performed in the 10 second protocol, a 61.9% increase in the 20 second protocol, a 61.0% increase in the 60 second protocol, and a 23.5% improvement in the nonstop effort. The conclusion was that Cr

supplementation significantly improved the ability to maintain a specific power output during intermittent exercise performance, especially during shorter interval work.

Balsom et al. (1995) demonstrated that 7 male subjects were better able to maintain power production in a cycling test after 6 days of Cr supplementation (20 grams/day) versus no supplementation. The exercise protocol consisted of 5 x 6 second cycle sprints at a fixed intensity with 30 seconds of passive rest between each set and a final sprint of 10 seconds after a 40 second rest to determine the percent decline in power production. After Cr supplementation subjects were better able to maintain power production in the last 10-second sprint. The authors also performed muscle biopsies to determine the concentration of ATP, PCr, Cr, lactate, and TCr before and after Cr supplementation. These results indicated that PCr concentration was higher and muscle lactate concentration was lower after versus before supplementation. This suggests that the subjects were better able to recover between exercise bouts because a larger concentration of Cr was present for a quicker resynthesis of PCr. Another cycle ergometer study done by Febbraio et al. (1995) showed that the concentrations of PCr and TCr were higher after Cr supplementation but that exercise performance was not affected. They utilized a different exercise protocol than the one utilized by Balsom et al. (1995). In their protocol subjects performed 4 x 1 minute bouts of exercise at a workload corresponding to between 115-125% VO_2 max with 1 minute of passive recovery between sets and a final set to fatigue. The conclusion of this study was that the creatine kinase system was predominantly involved during this type of exercise; therefore, supplementation with creatine had no effect on exercise performance.

Kreider et al. (1998) tested 25 male collegiate football players on a cycling test to evaluate the effects of 28 days of Cr supplementation (15.75 grams/day). The test involved 12 sets of 6-second cycle ergometer sprints with 30 seconds of rest between each sprint. More work was done in the first 5 sprint bouts in the Cr group versus the placebo group but that there was no significant difference in total work done over all 12 sets when comparing the placebo group to the Cr group.

Balsom et al. (1993) studied the effects of creatine ingestion on 16 active, well-trained male subjects in a randomized double blind study. They utilized a repeated bicycle ergometer exercise protocol where subjects did 10 sets of 6 second sprints at 130 rev/min and 10 sets of 6 second sprints at 140 rev/min with 30 seconds rest between sprints. The subjects either ingested creatine monohydrate (25 g/day x 6 days) or a placebo. The results showed that the placebo group declined in performance during the first three sprints while the creatine group increased performance. The differences between performance in the two groups became increasingly noticeable with repeated sets and by the seventh set the differences became statistically significant.

Dawson et al. (1995) also studied the effects of creatine supplementation on 6 sets of 6-second cycle sprint performance with 24 seconds of passive rest between sets in 18 healthy, active males. The results showed increases of 4.6% in power output and 4.6% in total work completed after creatine ingestion (20 grams/day for 5 days).

Kamber et al. (1999) studied how placebo and creatine ingestion (20 g/day x 5 days) effected power indices in 10 well-trained sports students in a crossover design with a counterbalanced order and mean washout time of 61 days. The exercise protocol evaluated cycling performance in 10 x 6-second sprints with 30-second rest intervals.

There was a 3.5% increase in revolutions/minute during creatine supplementation while no increase was apparent during placebo supplementation. The ergogenic effect was most prevalent in the last two seconds of the last 6 sprint sets. They concluded that creatine ingestion may be more ergogenic during the later seconds of high intensity exercise.

Thirty-six male and female track and field athletes were randomly assigned to a placebo or creatine supplementation group (0.3 g/kg body mass per day x 6 weeks) and evaluated by cycle ergometer by performing 5 x 10 second sprints with 60 seconds recovery between sprints (Kirksey et al. 1997). The authors noted a significant group-by-trial interaction in mean peak power throughout all 5 sprints. There was a 13% improvement in mean peak power for the creatine group and a 5% improvement in mean peak power for the placebo group. The conclusion was that creatine supplementation provided an ergogenic effect for male and female track and field athletes in cycle ergometer sprint power.

There are several studies reporting that creatine supplementation has no ergogenic effect on repeated cycle ergometer sprints. A study done by Cooke and Barnes (1997) evaluated the effect of Cr supplementation on the ability of male subjects to reproduce and maintain a peak power output during 2 sprints on a bicycle ergometer. They utilized 4 different recovery times of 30, 60, 90, and 120 seconds. Subjects were randomly assigned into Cr and Placebo groups and ingested 20 g/day of Cr or 24 g/day of glucose (placebo) for 5 days. There were no statistically significant group interactions and the authors concluded that Cr supplementation had no effect on the ability of men to sustain a high peak power. Another study done by Cooke et al. (1995)

found no significant differences between creatine supplementation (20 g/day x 5 days) or placebo ingestion in 2 sets of 15 second maximal cycle sprinting on a ergometer. The results indicated that peak power, time to peak power, total work, and fatigue index were all unaffected by creatine supplementation.

A study by Barnett et al. (1996) showed Cr supplementation provided no significant improvement in peak power, mean power, end power or percent power decline in 17 subjects randomly assigned to a Cr group (20 grams/day for 4 days) or a placebo. The subjects all completed 7 x 10 second bicycle ergometer sprints with 30 seconds of rest between each set except the fifth and sixth interval which were separated by a 5 minute rest.

Burke et al. (1996) assessed the effectiveness of creatine supplementation on 2 sets of 10-second cycle sprints with 10-minute rest between sets in 32 elite male and female swimmers. The subjects were randomly assigned to a creatine ingestion group (20 g/day x 5 days) or a placebo group. The results showed no differences between groups or trials in work done, peak power, or time to peak power.

Finally, Stone et al. (1999) studied 42 American football players on their ability to perform 15 x 5 second cycle sprints on a cycling ergometer before and after randomization to the following four conditions: placebo, high dose creatine (0.22 g/kg body mass/day x 5 weeks), low dose creatine (0.09 g/kg body mass/day x 5 weeks), and calcium pyruvate. The results indicated no significant differences between or within any of the groups in terms of peak power, average peak power, or total work across the 15 sets.

This summary of studies indicates that the majority of research involving repeated intervals of anaerobic work benefit from Cr supplementation, most likely due to an increase in PCr stores and therefore anaerobic capacity. The minority of studies that do not demonstrate a effect may be due to individual responsiveness to Cr supplementation. That is, those with a high content of Cr in their regular diets may not respond to supplementation as well as others.

1.2.6 Creatine and Resistance Exercise

Oopik et al. (1998) studied the effect of Cr supplementation on isokinetic muscle performance after a loss of body mass of 3.0-4.3% in karate athletes. This study was used to determine if Cr supplementation could enhance performance in athletes that are required to "cut weight" in order to qualify for a lower weight class (eg. karate or wrestling). The protocol evaluated both sub-maximal and maximal isokinetic strength, measuring the peak torque and work at peak torque for the knee extensor muscles. The study utilized a crossover design where 6 subjects completed the exercise test before any supplementation or weight loss and again after Cr or placebo supplementation and weight loss. The results showed no significant difference in maximal peak torque or work at peak torque between the Cr or placebo group before or after supplementation and loss of weight. Sub-maximal results showed that in comparison to the placebo group the Cr group's performance scores were less. These results confounded the authors, but they suggested that creatine uptake may be impaired during rapid body mass loss, which could have accounted for the performance detriment.

Greenhaff et al. (1993) studied the effects of placebo or creatine supplementation (20 g/day for 5 days) on the ability of 12 active, but untrained subjects, to produce and maintain power output during isokinetic knee extension. There was a significant improvement in peak torque production in the creatine compared to the placebo group after supplementation. The authors concluded that creatine supplementation resulted in an increased phosphocreatine resynthesis and in turn enhanced the ability of the muscle to maintain a greater rate of ATP for muscle contraction.

Vandenberghe et al. (1996) utilized a crossover design to determine the effects of 40 grams of Cr for 6 days or placebo on isokinetic torque in 9 healthy males. Subjects performed maximal voluntary contractions of 3 sets x 30, 4 sets x 20, and 5 sets x 10 contractions separated by 2 minutes of rest. The PCr/ATP ratio increased in the Cr group as assessed by P-MRS. The authors found a 10-23% increase isokinetic torque production after Cr supplementation. This study supports the hypothesis that Cr supplementation improves the resynthesis of ATP post-exercise due to the increased level of PCr available.

Vandenberghe et al. (1997) assessed the effectiveness of long term Cr supplementation on muscle strength and body composition in 19 young females. The study involved supplementing with either creatine for 4 days (20 grams/day) and then 10 weeks (5 grams/day) or a placebo for the entire duration of the study. The subjects also participated in a resistance-training program for 3 hours/week. The strength training program involved 7 different exercises, which included, leg press, bench press, leg curl, leg extension, squat, shoulder press, and sit-ups. The subjects performed 5 sets of 12 repetitions of each of the exercises at 70% of their 1 repetition maximum. Their

one repetition maximum was determined before any supplementation, after 5 weeks of training, and after 10 weeks of training. The exercise test used in this study involved 5 sets of 30 repetitions of maximal right arm flexion on a isokinetic dynamometer with 2 minute rest intervals between sets. Compared to the placebo group, the Cr supplemented group had a 20-25% increase in maximum strength of the muscle groups trained, a 10-15% increase in maximal intermittent exercise capacity, and a 60% increase in fat free mass. The authors concluded that long term creatine supplementation is beneficial to muscle strength during resistance training exercise in sedentary females.

The majority of studies found creatine monohydrate supplementation resulted in an improvement in isotonic strength. 1RM bench press, squat, and/or power clean were improved in several randomized, double blind, placebo control studies (Earnest et al., 1995; Kreider et al., 1998; Noonan et al., 1998b; Peeters et al., 1999; Stone et al., 1999; Stout et al., 1999b; Volek et al., 1999). The subjects in these studies were competitive athletes (football, soccer) or resistance trained individuals. Earnest et al. (1995) found a significant increase in the absolute amount lifted for a 1RM bench press but no significant difference in relative terms once body mass was accounted for. The studies utilized appropriate supplementation techniques to ensure Cr and PCr concentrations would be increased intramuscularly (15-25 grams/day for 3-42 days with a maintenance dose of between 5 to 10 grams/day following the loading phase). One study showed no ergogenic effect of creatine supplementation (20 grams/day for 5 days and then 3 grams/day for 48 days) on 1RM or 12 RM strength measures (Bermon et al. 1998). This study is unique from other studies because subjects used in the study were from the

elderly population. The authors reported that the uptake of creatine into the muscle, measured via urinary analysis, was lower than that reported by previous studies on younger subjects. This may result in decreased PCr resynthesis and thus no improvement in exercise performance.

Brenner et al. (2000) studied the effects of Cr supplementation combined with 5 weeks of pre-season strength training in 16 female NCAA Division I lacrosse players. The subjects were randomized into either a Cr group (20 grams/day x 7 days + 2 grams/day x 28 days) or a placebo group (N=9). One repetition maximum (1RM) strength testing was done for bench press and leg extension. The results showed that Cr supplementation significantly improved 1RM strength in bench press but not in the leg extension. The authors suggested that their subjects may be genetically "more close" to their 1RM for leg extension than for bench press. They also stated that the shorter period of strength training (5 weeks) might be responsible for a lower strength gain. This reasoning is questionable because improvements were seen in bench press with 5 weeks of resistance training.

Syrotuik et al. (2000) assessed the ability of creatine supplementation to improve absolute strength (one repetition maximum), total lifting volume (80% of 1RM to fatigue), and strength to mass ratio in 21 male recreational weight lifters. Subjects were randomly assigned to 3 groups: an acute creatine supplementation and placebo group (5 days x 0.30 grams/kg/day + 32 days of placebo), an acute creatine supplementation and maintenance group (5 days x 0.30 grams/kg/day + 32 days x 0.03 grams/kg/day), or a placebo group. Each group completed the same periodized strength-training program with the volume of training kept constant for each individual. The results indicated no

statistically significant difference between groups in 1RM, total lifting volume or strength to mass ratio except for the acute load only group who demonstrated an increased lifting volume for bench press after the acute creatine loading phase.

In summary, the majority of data from well-controlled research thus far indicates an improvement in maximum strength when creatine supplementation is combined with resistance training versus placebo supplementation and resistance training.

1.2.7 Creatine and Biochemical Markers

The effectiveness of creatine to modify biochemical markers of metabolic processes has been studied by some researchers. In theory, creatine supplementation is able to buffer the build up of hydrogen ions during anaerobic exercise, thus delaying fatigue during heavy anaerobic exercise. That is, the muscle is able to rely on the ATP/PCr system for a longer period of time without having to rely predominantly on anaerobic glycolysis for ATP production. Also, creatine supplementation is theorized to decrease the rate of production of ammonia after intense anaerobic exercise. It does so by buffering the build up of protons thus decreasing the need for the body to rely on the adenylate deaminase reaction and preserving the adenine nucleotide pool.

Blood lactate concentrations have been evaluated before, during and after exercise in creatine supplemented subjects by a number of researchers. Most of the results indicate no significant change or an increase in blood lactate comcentration after creatine supplementation (Birch et al. 1994, Dawson et al. 1995, Odland et al. 1997, and Snow et al. 1995). There are three studies indicating a lower concentration of blood lactate following exercise. Andrews et al. (1998) found that creatine supplementation

decreased lactate concentration, decreased ammonia cocnentration, as well as improved grip strength in congestive heart failure patients. Balsom et al. (1995) and Kamber et al. (1999) both found that blood lactate cocnentration was lower following creatine supplementation in tests involving repetitive cycle ergometer sprints. Furthermore, Birch et al. (1994) and Greenhaff et al. (1993) found lower ammonia concentrations following creatine supplementation in their healthy subjects. It is evident that there is not a clear consensus on the effect of creatine supplementation on biochemical markers of metabolic function. More research is needed to clarify this issue.

1.2.8 Creatine and Changes in Body Mass

One of the possible benefits or side effects associated with Cr ingestion is an increase in body mass. An increase in body mass may be considered beneficial for some sport participants (such as football or hockey) but could be considered detrimental if there was not a corresponding increase in lean body mass to move the body efficiently over the playing surface. Grindstaff et al. (1997) showed no significant difference in body mass, fat free mass, fat mass, total body water and percent body fat in competitive swimmers before and after 9 days of Cr supplementation. Green et al. (1996) found an increase in body weight in two of their four groups of subjects. There was an increase in weight of a Cr supplementation group and a Cr and carbohydrate ingestion group. There was no increase in body mass in a Cr, carbohydrate and 1 hour of aerobic exercise group or a control group. This study found an increase of 0.6 ± 0.2 kg and 2.1 ± 0.5 kg in body mass of the Cr and Cr/Carbohydrate groups respectively. This suggests that carbohydrate ingestion along with Cr may enhance the uptake of Cr

into the muscle with a corresponding increase in the amount of water taken up into the skeletal muscle thus, increasing body weight. The majority of studies show an increase in body mass of between 0.7 to 2.8 kilograms after 5-7 days of creatine supplementation (Balsom et al., 1995, 1993a, 1993b; Cooke and Barnes, 1997; Green et al., 1996a, 1996b; Kelly and Jenkins, 1998; Magnaris and Maughan, 1998; McNaughton et al., 1998; Mujika et al., 1996; Noonan et al., 1998; Peeters et al., 1999; Snow et al., 1998;Vandenberghe et al., 1997; Viru et al., 1994; Volek et al., 1997; Vukovich and Michaelis, 1999). There are three studies which did not show an increase in body mass after creatine supplementation (Barnett et al., 1996; Bermon et al., 1998;Grindstaff et al., 1997). In the study by Barnett et al. (1996) they utilized a 4 day protocol to load with creatine (20 grams per day for 4 days); this may not be sufficient to produce changes in body mass. The study done by Bermon et al. (1998) utilized an elderly population as their subjects. The protocol they used for creatine supplementation was appropriate (20 grams per day for 5 days) but the uptake of creatine into the muscle may be slower in the older population because of a decrease in fast twitch muscle fiber composition. Balsom et al. (1994) have stated that PCr is higher in type II muscle fibers as opposed to type I muscle fibers. Elderly people have a lower proportion of type II muscle fibers and thus, it may take a longer time to "load" their muscles with creatine.

The increase in body mass associated with Cr supplementation has been attributed to two possible mechanisms. First, some authors believe it is due to an increase in water within the muscle cell (Volek et al., 1997). As Cr is actively transported into the muscle cell, water is transported along with it. Thus, body mass is increased because skeletal muscle is retaining more water. Furthermore, the increase in cellular water

associated with Cr ingestion is theorized to act as a anabolic proliferative signal. This signal is hypothesized to initiate an increase in protein synthesis within the muscle cell thus, increasing the amount of muscle present in an individual.

1.2.9 Creatine Supplementation Protocols

The loading phase typically involves taking 20-30 grams of Cr throughout the day for between 5 and 7 days. Hultman et al. (1996) found that a quicker way to increase muscle TCr cocentration was to supplement based on body mass. They state that 0.3 grams of Cr per kilogram of body mass is desirable. Depending upon your body mass this corresponds to the recommended 20-30 grams of Cr per day. Once the loading phase is complete, a maintenance phase is necessary to keep the concentration of TCr at its elevated level. Most investigations use between 2-5 grams of Cr/day for the maintenance dose. Hultman et al. (1996) suggest that 0.03 grams of Cr/kilogram of body mass is sufficient to maintain elevated levels of TCr. The majority of research prescribes to this methodology but there are a few studies which do not use enough creatine to obtain the ergogenic effect and there are those that use too much to obtain the desired effect.

1.2.10 Creatine and Side Effects

Ingesting creatine in high doses as a nutritional supplement has led some to speculate that creatine ingestion may have undesirable side effects. The side effect most substantiated in research to date is an increase in body mass (Volek and Kraemer, 1996). This may be detrimental to some athletes in mass-dependant activities but may

be beneficial to some athletes in strength/power sports such as football. Anecdotal evidence suggests that gastrointestinal distress, muscle cramping, and dehydration may be found in some individuals who ingest creatine monohydrate (Juhn et al., 1999). In a retrospective study performed by Schilling et al. (2001) 19 subjects, who ingested creatine for a period between 0.8 and 4 years, were asked if they experienced any side effects while supplementing with creatine. Three of the nineteen subjects questioned stated they experienced occasional muscle cramping but also felt that creatine supplementation was not responsible for the cramps. Also, three of the nineteen subjects stated they experienced gastrointestinal upset during the loading phase of creatine supplementation. Sixteen of the nineteen subjects in this study reported that creatine supplementation was ergogenic. Schilling et al. (2001) found no statistically significant differences between the control group and the creatine-supplemented group for serum enzymes, blood chemistries, hormone concentrations, or lipid profiles and all the variables measured were within normal clinical ranges. Vogel et al. (2000) evaluated the effects of creatine supplementation (20 grams/day x 5 days) or placebo on performance of 5, 5-second maximal cycle ergometer sprints in 16 men enclosed in a temperature and humidity controlled chamber. During the experimental trial, the subjects completed 5, 5-second sprints followed by a 75-minute exercise session designed to facilitate body water loss, then 5, 5-second sprints followed by another 75-minute exercise session. The authors reported that skeletal muscle tightness and/or cramping were reported in both groups. The authors concluded that creatine supplementation did not increase the incidence of muscle cramping or tightness and does not appear to negatively affect hydration status. There is no evidence to date that a

causal relationship exists between creatine supplementation and muscle dysfunction or gastrointestinal symptoms (Juhn and Tarnopolsky, 1998b). Furthermore, there is no evidence that the exogenous feeding of large amounts of creatine inhibits the endogenous production of creatine (Vandenberghe et al., 1997). These authors found that after 4 weeks of creatine supplement cessation that endogenous production returned to a normal level. The long-term effects of creatine supplementation on endogenous creatine production warrants further investigation.

There is concern over the effects of creatine supplementation and other organ systems besides skeletal muscle. Some concerns exist over the long-term effects of creatine supplementation on the cardiovascular system. In their review on the side effects associated with creatine supplementation, Juhn and Tarnopolsky (1999b) state there are no known adverse affects on cardiac ejection or blood pressure over the short-term (10 days) but that more long term research on humans is needed. Also, anecdotal evidence suggests creatine supplementation may have neurological effects on the brain, especially seizure activity, but no causal link has been found yet (Juhn and Tarnopolsky, 1998b). In their review Juhn and Tarnopolsky (1999b) suggest more research should be done to determine the effects creatine supplementation may have on the structure or function of other organ systems such as the brain, heart, and testes, since CK activity is high in these tissues.

Creatine is converted non-enzymatically to creatinine and eliminated in the urine each day. When creatine is ingested in supplemental form, some authors speculate that it may increase the chance of straining the kidney (Poortmans et al., 1997). Poortmans et al. (1997) suggest "that a nitrogen-rich diet might itself induce chronic renal

hyperfiltration and hyperfusion and thereby contribute to the functional and structural deterioration of the kidney". Poortmans et al. (1997) performed a short-term study that evaluated if creatine supplementation (20 grams/day x 5 days) increased arterial or urinary output of creatine or creatinine. The results showed that arterial creatine concentration was increased by 3.7 fold, and creatinine clearance in the urine was increased 27 fold after creatine supplementation. The arterial creatinine concentration, urinary creatinine excretion rate, and the estimated glomerular filtration rate were not increased by creatine supplementation. Furthermore, Poortmans and Francaux (1999) performed a long-term study evaluating the same parameters as above. The creatine-supplemented subjects in the long-term study had supplemented with creatine for between 10 months and 5 years ingesting between 2 and 30 grams per day. The results showed no significant differences between the creatine group and the control group for plasma contents of creatine, creatinine, urea, and albumin. Again the excretion rate of creatine in urine was increased substantially in the creatine-supplemented group but urine excretion rates for creatinine, urea, and albumin were all within a normal range. The authors in the short and long-term studies concluded that Cr supplementation does not significantly alter the function of the kidney in healthy humans. One study using rats with kidney disease has shown that the rate of disease development is much faster in rats supplemented with creatine as opposed to rats not supplementing with creatine (Edmunds et al., 2001). This animal model may lead one to speculate that humans with kidney disease or renal dysfunction may develop the disease at a much faster rate as well. Juhn and Tarnopolsky (1998b) advise that individuals with renal dysfunction or diabetes should not use supplemental creatine.

In summary, the short term effects of creatine supplementation indicate that adverse effects are not present in human studies. More research should be done to evaluate if there are short-term and long-term effects of creatine supplementation on the human body. These studies should be designed in a randomized controlled fashion, which attempt to evaluate effects as completely as possible.

1.2.11 Review of Literature Summary

Creatine monohydrate is recognized as a nutritional ergogenic aid used to enhance sport performance. It is theorized to work through three commonly accepted mechanisms. The first mechanism is through an enhanced buffering capacity for ATP with the increased concentration of Cr and PCr associated with supplementation. The second mechanism theorizes that the increased concentration of PCr within the cell will aid the system in buffering protons thus, reducing the build up of lactic acid and delaying the need for anaerobic glycolysis. Finally, creatine supplementation is proposed to be ergogenic by increasing the amount of fluid within the cell, which stimulates an increase in the rate of protein synthesis thus, improving muscular performance and training intensity.

Creatine supplementation has been studied under a variety of exercise conditions. Creatine supplementation is not ergogenic for aerobic type activities. During single anaerobic sprint activities the results of creatine supplementation have been debatable in producing an ergogenic effect. The majority of research with intermittent repeated sprints of high intensity have shown that creatine is ergogenic. Also, the majority of research evaluating muscular strength and muscular endurance has shown creatine to be

ergogenic. Studies evaluating the effect of creatine monohydrate supplementation on biochemical indicators of muscle energetics have found contestable results. Finally, most well-controlled studies have shown that there is an increase in body mass associated with creatine ingestion, especially during the loading phase.

Only one study has been done on the effect of creatine supplementation in ice hockey players (Jones et al., 1999). This study has contestable results indicating creatine supplementation aided forward repeated sprint skating speed but not backward repeated sprint skating speed. Also, no biochemical markers were evaluated in this study which could give an indication as to the predominate energy source being utilized by the muscle. Furthermore, the sprint skating was performed on ice, which would not allow the researchers to control the intensity of the subjects' performance.

1.3 Statement of the Problem and Hypotheses

1.3.1 Statement of the Problem

Only one study has investigated the effects of creatine monohydrate supplementation on ice hockey skating (Jones, Atter, & Georg, 1999). The study showed inconsistent findings (i.e. an improvement in forward skating speed but not backward skating speed), which suggests further study needs to be done. A possible confounding factor with the above study is that the amount of work done by each subject may have varied depending on the skill level of each player. One way to improve this would be to utilize a standard speed and grade on a treadmill. Another confounding factor in the above study was that dietary analysis was not done for the subjects. To improve upon this, dietary analysis should be performed on each subject.

The purpose of this study was to evaluate the effects of creatine monohydrate supplementation on repeated skating intervals on a skating treadmill in hockey players. This was accomplished by evaluating the effect of Cr supplementation on time to exhaustion and blood lactate response to intervals of treadmill skating. A second purpose was to evaluate the effect of Cr supplementation on isokinetic muscular endurance. Dietary analysis and Cr intake before testing were evaluated as possible confounding variables in this study.

1.3.2 Hypotheses

The major hypothesis was that:

1. There would be an increase in skating time to exhaustion in the creatine trial as compared to the placebo trial.

The secondary hypotheses were that:

1. Blood lactate levels would take longer (i.e. increased time to exhaustion) to reach a maximum level in the creatine trial as compared to the placebo trial.
2. Blood lactate concentrations will be lower, before volitional fatigue, during the creatine trial as compared to the placebo trial.
3. Peak torque and average power assessed isokinetically would remain higher in the creatine trial versus the placebo trial.

1.3.3 Limitations

1. Exercise performance was assessed by time skated to voluntary exhaustion, and thus is dependent on the motivation of each subject. A subject may have been more or less highly motivated during subsequent testing sessions, which may have affected the results of the study.
2. The dietary habits of each subject could not be completely controlled during this study. The food ingested by each subject is an estimate based upon portion size, as well as accuracy in filling out every detail of the three-day food record.

1.3.4 Delimitations

1. The results of the study apply to people in the same age range, training status, and creatine intake as those involved in the study, since these variables may influence the results of the study.
2. The testing was done in a laboratory location, therefore it is difficult to determine whether or not creatine supplementation would have a similar effect in a field environment.

CHAPTER 2

METHODS

2.1 Research Design

This study utilized a double blind repeated measures procedure where subjects were randomized into either a creatine group or a placebo group. The subjects were required to come to the laboratory on five occasions, once for treadmill familiarization, twice for treadmill data collection, and twice for isokinetic muscular testing of the knee joint. The dependent variables in this study were (1) time to exhaustion, (2) blood lactate concentration, (3) peak muscle torque, (4) average muscle power, and (5) body mass. Also, actual dietary intake prior to testing was assessed as a possible confounding factor.

2.2 Participants

Seventeen competitive male ice hockey players were recruited as subjects for the study. They were randomized into either a placebo condition (N=8) or a creatine condition (N=9). Their physical characteristics are described in Table 3.1. The subjects were recruited from competitive intercollegiate hockey at the University of Saskatchewan as well as a competitive junior "B" hockey team. Some subjects were not included in the statistical analyses because they did not attend their scheduled testing sessions. For the dietary analysis variables, 6 subjects were included in the placebo group and 9 subjects included in the creatine group before supplementation. During supplementation the number of subjects in the placebo group for dietary analysis

was reduced to 5 and in the creatine group reduced to 6. Skating time to exhaustion included 6 subjects in the placebo group and 8 subjects in the creatine group at both testing sessions. The number of subjects in the blood lactate data also varied due to the fact that not all blood samples were obtained.

Before supplementation, 6 and 8 subjects were included in the placebo and creatine group respectively at baseline and fatigue. After the first skating interval, before supplementation, 5 subjects were included in the placebo group and 3 subjects in the creatine group. After the third skating interval, before supplementation, 3 subjects were included in the placebo group and 7 subjects in the creatine group.

After supplementation, 6 and 8 subjects were included in the placebo and creatine group respectively at baseline and fatigue. After the first skating interval, after supplementation, 6 subjects were included in the placebo group and 8 subjects in the creatine group. After the third skating interval, after supplementation, 4 subjects were included in the placebo group and 7 subjects in the creatine group.

The isokinetic data and the data on body mass included 6 subjects in the placebo group and 9 subjects in the creatine group before and after supplementation.

Before any testing, a calculation of the number of subjects necessary to find a significant difference with 90% power was done (Glasnapp & Poggio, 1985). The formula used is as follows: $n = S.D.^2 (z_{1-\alpha} + z_{\beta})^2 / d^2$, where n = the number of subjects required, d = the difference you expect to see between the means of placebo and creatine supplementation (difference in means was taken from Prevost et al., 1997 which was 60 seconds), and S.D. = the expected standard deviation for the means (also taken from Prevost et al., 1997 which was 55 seconds). The calculation was based on

previous research which used time to exhaustion as the dependent variable in a study assessing the efficacy of creatine supplementation in high intensity intermittent cycling (Prevost et al., 1997). The results of the formula used to estimate sample size for this research indicated seven subjects per group was adequate. There were eight subjects in the creatine group and six subjects in the placebo group. The possibility exists that statistical power was too low as a result of the limited number of subjects who completed the study. If this is true, then calculating a statistically significant difference between the creatine and placebo groups could not be accomplished due to limited statistical power.

2.3 Procedures

2.3.1 Test Protocol

Subjects were recruited by attending team practices, explaining the procedures of the study, and asking for volunteers who would be interested in participating. The participants who signed up were explained the essence of the study as well as the time commitment it would require on their part. Written consent was obtained from each participant (Appendix A). Each subject was given two three-day food diaries (Appendix B) which were to be completed three days prior to the first testing day and three days prior to the experimental testing date.

Each participant was required to attend the laboratory five times during the study. The first session was to familiarize the participants with the skating treadmill and the proper technique required to skate effectively on the machine. The second and fifth

session required the subjects to complete a test of isokinetic muscular endurance of the right knee using the Biodex isokinetic dynamometer (Biodex System 3, Biodex Medical Systems Inc., Shirley, NY). These trials were before and after creatine or placebo supplementation. The third and fourth sessions were the repeated skating sprints before and after either creatine or placebo supplementation. The muscular endurance testing was done in the Exercise Physiology Laboratory in the College of Kinesiology at the University of Saskatchewan. The skating treadmill testing was done in the Frappier Acceleration Centre at the Jemini Ice Rinks in Saskatoon. There were 6 days between the skating treadmill tests and the isokinetic muscular endurance tests. A time line for the testing is shown in Figure 2.1.

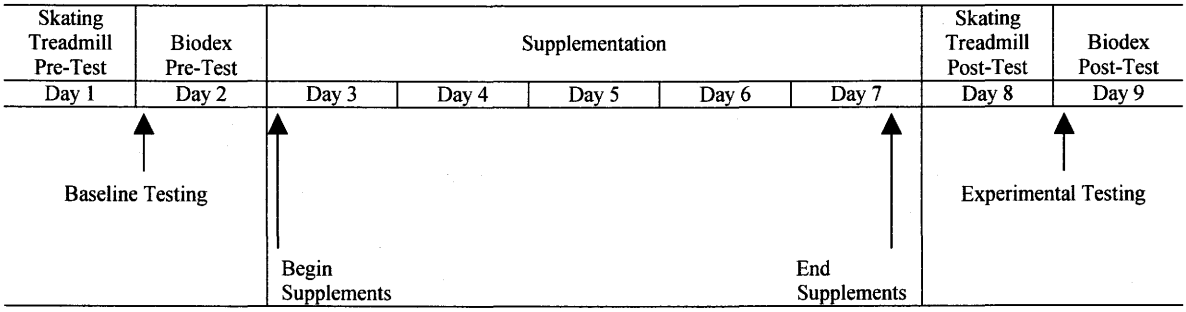

Figure 2.1. Supplementation time line

The study was approved by the University of Saskatchewan's Advisory Committee on Ethics in Human Experimentation. The cost of the creatine supplementation was covered by Musceltech Research and Development. Each subject in the study was given an equivalent amount of creatine or placebo for the 5 days of supplementation.

2.3.2 Dietary Assessment

Every participant was required to complete 2 - three day food diaries. The diaries were completed with two weekdays and one weekend day each time. One was completed before the first skating treadmill test and the other was completed before the final skating treadmill test. The dietary data derived from the three-day food diaries was entered into a nutritional analysis software program (FUEL, Logiform International Inc., Sainte-Foy, Quebec). The major dietary substrates analyzed included: fat, protein, and carbohydrate, which were analyzed to determine if the subjects' diets were similar in substrate concentration and total amount of kilocalories before and after the Cr supplementation. The average amount of grams ingested in the form of fat, carbohydrate, and protein was also assessed. A diet too low or too high in carbohydrate may affect the results of the study because it has been shown that carbohydrate feeding along with creatine enhances the uptake of Cr into the muscle (Green et al., 1996). These authors suggested that creatine uptake was higher in a carbohydrate and creatine fed group, which they suggest was mediated by a glucose stimulated release of insulin. The food diaries were also completed to examine the possible effects that a low or high dietary creatine ingestion may have on exercise performance in creatine supplemented individuals.

2.3.3 Supplement Administration

Subjects were supplemented with creatine or placebo for five days. The subjects were randomized into either group by drawing numbers from a hat where odd numbers received placebo and even numbers received creatine treatment. An individual not

involved in the study completed this procedure. They were required to ingest their supplement 3 times per day with approximately 4 hours between each ingestion. The subjects were told to ingest the supplement with fruit juice since it has been demonstrated that ingestion of creatine with carbohydrate enhances the uptake of creatine into the muscle cell (Green et al., 1996). The subjects who were randomized to the creatine group received 0.30 grams of creatine per kilogram of body weight. This amount has been shown to produce an optimal increase in the total concentration of creatine within the muscle (Harris et al., 1992). The subjects randomized to the placebo group received an equal amount of flour and sugar as a placebo. The subjects received a total of 15 packets of their supplement with the appropriate dose for the five-day period of supplementation.

2.3.4 Skating Treadmill Familiarization

The purpose of the first visit to the Frappier Acceleration Centre was to allow the subjects to become accustomed to skating on the treadmill at the speed and elevation to be used during the test. All participants read what the testing protocol would involve and signed the informed consent form (Appendix A). Each subject was given verbal instructions on the safety aspects of the treadmill and how to get on and off the treadmill. The speed and elevation were kept at levels where the subjects felt comfortable skating. Each subject spent between 45 and 60 minutes becoming accustomed to the treadmill. Towards the end of the session subjects completed a "practice" session on the treadmill at the speed and elevation that would be required

during the test. During this accustomization trial the subjects were not required to have blood taken for lactate analysis.

2.3.5 Skating Treadmill Test

The third and fourth testing trials were done on the skating treadmill. When the subjects arrived at the laboratory, they were given a standardized warm-up which consisted of 5 minutes of cycling and 5 minutes of static stretching of the leg and hip muscles. Once this warm up was completed, each subject put on their skates and a safety harness before starting the standardized warm-up on the skating treadmill. Each subject was required to give a baseline sample of blood at this point for lactate analysis. After the blood sample was taken, each subject completed the standardized warm-up seen in Table 2.1 before starting the testing protocol.

Table 2.1

Skating Treadmill Warm-up Procedure

Speed (km/h)	Elevation (% grade)	Duration (seconds)
11.9	5	45
12.9	10	10
12.9	15	10
12.9	15	10

After the standardized warm-up subjects were given a rest of between 2 – 3 minutes. During this time, testing instructions were given to the subjects so that they understood the procedure to be followed. Subjects were required to skate at a speed of 16.1 km/h and a elevation of 15 % grade for a period of 10 seconds. Once this sprint was completed, the treadmill was turned off and they rested for 30 seconds. With 10 seconds left in their rest the treadmill was started to allow it to reach full speed, and with 5 seconds left in the rest subjects were given a verbal cue of "ready, set, go" when their rest period had expired. The subjects then completed another 10-second skating sprint followed by another 30-second rest interval and repeated this procedure until volitional fatigue. If the subject stopped skating any time during an interval this was considered volitional fatigue and the time in seconds for that interval was recorded. After each odd numbered skating sprint, the subjects were required to give a blood sample for lactate analysis. Once the subject had reached volitional fatigue, he was also required to give one last blood sample. Upon reaching volitional fatigue, the subject's total time to exhaustion was calculated by summing the total number of seconds skated.

Blood lactate was measured throughout the skating test using the Accusport Portable Lactate Analyzer (Sports Resource Group, Inc.). Blood samples were taken from a finger prick on the subject's choice of hand. The subject's finger was pricked using a hand held lancet and the blood was gathered using a heparinized capillary tube. The blood was then immediately pipetted onto the Accusport Lactate Test Strips (Sports Resource Group, Inc.) and the lactate concentration of the blood was measured by the Accusport Portable Lactate Analyzer after 60 seconds. This system uses enzymatic determination and reflectance photometry (wavelength 660 nm) of lactate in a sample of

whole blood. The system reads lactate levels by straining out the red blood cells and analyzing the lactate concentration in the plasma portion of whole blood. The analyzer was calibrated with the control solutions before any testing was done with the instrument. The Accusport Portable Lactate Anaylzer has been validated against various blood lactate analyzers and correlation coefficients reported have been between 0.934 - 0.998 (Gambke et al., 1997; Fell et al., 1998; and Slomovitz et al., 1998). Depending upon how many skating intervals the subjects completed, more than 1 and usually between 2 – 4 finger pricks were required to obtain the blood samples.

2.3.6 Isokinetic Testing Trials

The second and fifth testing trials were done using the isokinetic dynamometer (Biodex System 3, Biodex Medical Systems Inc., Shirely NY) both before and after either creatine or placebo supplementation.

After the subjects' height and weight were recorded they completed a standardized warm-up which consisted of cycling for 5 minutes and 5 minutes of static stretching of the quadriceps and hamstring muscles. Each subject was then seated in the isokinetic machine and the seat settings were recorded. Range of motion consisted of movement from 90 to 10 degrees of knee flexion. Subjects sat against a back support, producing an angle of 80 degrees of hip flexion. Stabilizing belts were placed over the lap, across the chest, and across the distal one-third of the thigh of the tested leg. The rotational axis of the dynamometer was positioned to be coaxial with the knee axis (lateral epicondyle) during testing. Torque measures were corrected for the effects of gravity on the lower leg and the dynamometers resistance pad. The torque output on the dynamometer was

checked with a calibration weight on a weekly basis throughout the study duration. The subjects were then instructed as to how the machine worked and were given opportunity to practice knee extension and flexion with what they considered a 50% effort level.

The exercise test consisted of 3 sets of 10 repetitions at a maximal effort knee extension and flexion with 60 seconds of rest between sets. The speed of the dynamometer was set at 60 degrees/second. Subjects were given verbal encouragement throughout the testing procedure.

Data from the test was recorded on the computer interfaced with the Biodex machine and it was printed out immediately after the test. The peak torque and average power generated by each subject were the two indices utilized for statistical analyses. Peak torque is defined as the highest muscular force output at any moment during a repetition and is indicative of a muscle's strength capabilities. Average power is defined as the total amount of work divided by time and is indicative of the product of torque and angular velocity.

2.4 Statistical Analyses

All data was entered into a statistical analysis program (SPSS, version 7.5.1, SPSS Inc., Chicago, IL). All averages were obtained for each of the variables at baseline and at the end of both testing trials.

The values for skating treadmill time were determined by summing all of the skating sprints and calling the value the total time to exhaustion. Measurements for average power and peak torque were determined by averaging the values of all 3 sets both before and after supplementation.

The data was analyzed using a repeated measures, 2 x 2 factor, analysis of variance (ANOVA – treatment x time) with placebo or creatine as the two treatment conditions and pre- and post- time as the repeated measure. The Bonferonni adjustment was made to correct the alpha level for multiple tests. Thus, statistical significance was set at $p \leq 0.01$ for all tests. Analysis of covariance was used in time to exhaustion with baseline time to exhaustion, dietary intake (kilocalories), and dietary creatine intake as covariates. Each of the preceding covariates was entered individually for analysis in SPSS. Also, body weight before supplementation was included as a covariate when analyzing the isokinetic data.

CHAPTER 3

RESULTS AND DISCUSSION

3.1 Results

The purpose of this study was to assess the effectiveness of creatine monohydrate supplementation for improving sprint skating performance in competitive male ice hockey players. The primary hypothesis of this study was that creatine supplementation would increase skating time to exhaustion. The secondary hypotheses were that (1) blood lactate levels would take longer to reach "maximum" values in the creatine trial, (2) isokinetic power would be higher in the creatine trial, (3) isokinetic torque would be higher in the creatine trial, and (4) body mass would be higher in the creatine trial.

To determine if any significant differences were evident between the creatine and placebo trial, a repeated measures ANOVA was used to assess the dependent variables (time to exhaustion, blood lactate, average power, peak torque, and body mass).

3.1.1 Participant Characteristics

The characteristics of the subjects are displayed in Table 3.1. No significant differences were present between the two groups during baseline testing.

Table 3.1

Subject Descriptive Characteristics (mean ± standard error)

Characteristic	Creatine (n = 9)	Placebo (n = 8)
Age (years)	20.3 ± 0.7	18.4 ± 0.4
Height (cm)	178.0 ± 3.6	177.8 ± 1.2
Weight (kg)	77.8 ± 3.1	81.8 ± 2.8

3.1.2 Dietary Analysis

Dietary intake was analyzed in this study to determine if diet would be a confounding factor. Three-day food diaries were given to each subject before supplementation and during supplementation. The results of the dietary analyses are shown in Table 3.2. Four subjects failed to fill in a three-day food diary during supplementation; therefore, their results were not included in the analyses. The results of repeated measures ANOVA showed that the amount of calories ingested did not differ significantly between the placebo and creatine group ($F(1, 9) = 1.223$, $p = 0.297$). There were no differences between groups or over time for creatine intake from food sources. The results of creatine intake in food are presented in Table 3.3.

Table 3.2

Dietary intake (mean ± SE) before and during supplementation

Dietary Intake	**Placebo (N = 6)**	**Creatine (N = 9)**
Before Supplementation		
Kilocalories	3060 ± 369	2870 ± 363
% Carbohydrate	51 ± 4	49 ± 2
% Fat	32 ± 3	35 ± 1
% Protein	17 ± 2	16 ± 1
Grams Carbohydrate	398 ± 66	345 ± 42
Grams Fat	109 ± 16	113 ± 16
Grams Protein	123 ± 10	109 ± 12
Dietary Intake	**Placebo (N = 5)**	**Creatine (N = 6)**
After Supplementation		
Kilocalories	2650 ± 434	2370 ± 242
% Carbohydrate	46 ± 5	49 ± 3
% Fat	37 ± 4	34 ± 2
% Protein	17 ± 2	17 ± 2
Grams Carbohydrate	326± 78	293 ± 36
Grams Fat	104 ± 12	88 ± 9
Grams Protein	112 ± 18	95 ± 12

Note. No significant differences between trials.

Table 3.3

Dietary intake of creatine in grams (mean ± SE) before and during supplementation

Dietary Intake	Placebo (N = 6)	Creatine (N = 9)
Before Supplementation	1.1 ± 0.1	0.8 ± 0.1
	Placebo (N = 5)	**Creatine (N = 6)**
During Supplementation	1.1 ± 0.3	0.7 ± 0.2

Note. No significant differences between trials.

3.1.3 Time to Exhaustion

Time to exhaustion was evaluated by a repeated measures ANOVA and results showed a significant time main effect, $F(1, 12) = 11.075$, $p = 0.006$, but no significant interaction difference (time x treatment) between groups, $F(1, 12) = 0.011$, $p = 0.920$. Data for three of the participants (two in the placebo group and one in the creatine group) was not collected for time to exhaustion because the subjects failed to show up for the final testing. The data for time to exhaustion are presented in Table 3.4.

Table 3.4

Time to exhaustion in seconds (mean ± SE) during baseline and experimental testing

Time to Exhaustion	Placebo (n = 6)	Creatine (n = 8)
Baseline	45.2 ± 12.9	63.5 ± 12.4
Experimental	67.1 ± 23.3	84.1 ± 14.0

Note. No significant differences between trials.

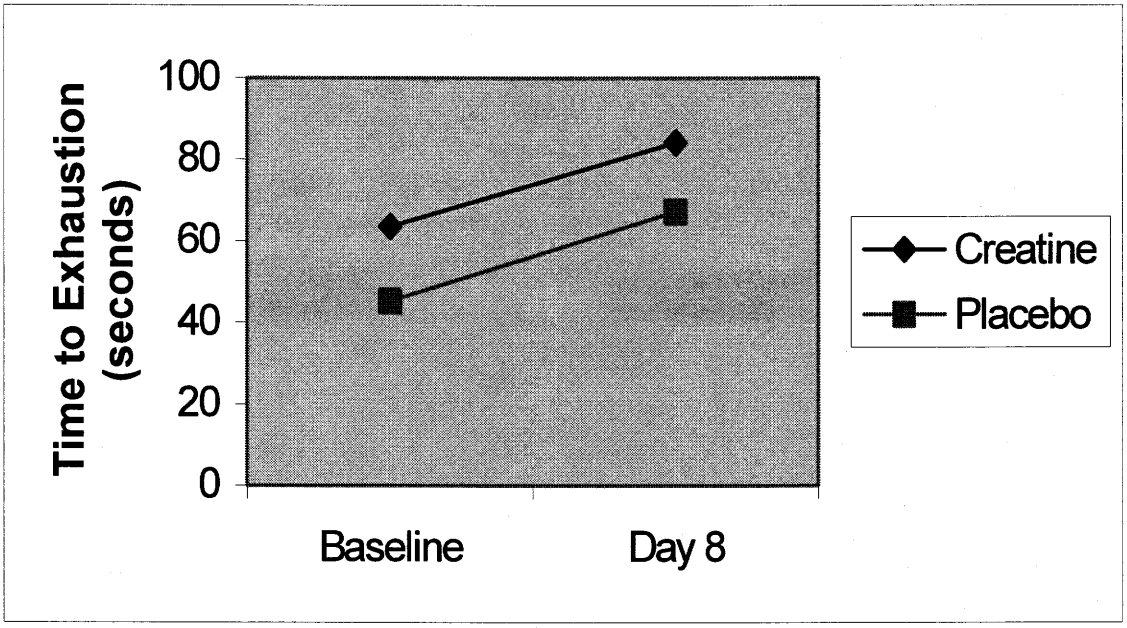

Figure 3.1. Skating time to exhaustion before and after creatine supplementation.

3.1.4 Blood Lactate

The blood lactate data was analyzed using repeated measures ANOVA and revealed no statistically significant differences between groups over time on any of the measurements taken. The test statistics were: $F(1, 12) = 0.265$, $p = 0.616$ at baseline, $F(1, 6) = 2.182$, $p = 0.190$ after the first set, $F(1, 5) = 1.571$, $p = 0.265$ after the third set, and $F(1, 12) = 0.788$, $p = 0.392$ at exhaustion. In the cells where there were 3 or more subjects per group (baseline, after sprint 1, after sprint 3, and at exhaustion) there were no significant differences between groups before or after supplementation for blood lactate. For the remaining sprints, means were also similar between groups, but there were insufficient numbers of subjects completing these sprints for statistical analyses. The blood lactate data are presented in Table 3.5.

Table 3.5

Blood Lactate Values (mean ± SE) During Sprint Skating

Blood Lactate Sample	Placebo Group (mmol/L)	Creatine Group (mmol/L)	n = Pl/Cr
Before Supplementation			
Baseline	4.3 ± 0.7	4.0 ± 0.4	6/8
After Sprint 1	10.0 ± 1.0	8.2 ± 1.0	5/3
After Sprint 3	13.7 ± 3.1	13.5 ± 2.2	3/7
After Sprint 5	15.6 ± 0.0	13.1 ± 1.4	1/5
After Sprint 7	15.5 ± 1.8	12.9 ± 0.0	2/1
After Sprint 9	16.3 ± 0.0	13.6 ± 3.8	1/2
Fatigue	19.4 ± 2.0	21.6 ± 1.9	6/8
After Supplementation			
Baseline	4.3 ± 0.6	3.5 ± 0.3	6/8
After Sprint 1	9.3 ± 1.6	9.7 ± 1.1	6/8
After Sprint 3	12.5 ± 4.5	10.8 ± 1.7	4/7
After Sprint 5	13.3 ± 0.0	17.3 ± 3.3	1/6
After Sprint 7	17.0 ± 2.5	15.0 ± 4.4	2/2
After Sprint 9	16.6 ± 0.0	18.7 ± 2.7	1/4
Fatigue	21.2 ± 2.6	19.9 ± 1.8	6/8

Note: No significant differences between the trials or between groups.

3.1.5 Average Power

ANOVA results showed no significant differences between the placebo and creatine trial in terms of isokinetic power output for knee extension averaged over the three sets, $F(1, 13) = 2.329, p = 0.151$. There was a significant time main effect for average power in the first set of knee extension ($F(1, 13) = 10.773, p = 0.006$) and knee flexion ($F(1, 13) = 8.681, p = 0.011$). The results indicated no significant group x time interaction for average power output for knee extension during each individual set (See Table 3.6 for complete results). There was no significant difference between the placebo and creatine group in isokinetic power output for knee flexion averaged over the three sets, $F(1, 13) = 0.03, p = 0.866$. Also, there was no significant difference in average power output for knee flexion between the placebo and experimental group in each individual set (See Table 3.7 for complete results). Two subjects in the placebo group were not included in these data analyses because they failed to show up for the testing sessions.

Table 3.6

Isokinetic average power (watts) for knee extension (mean ± SE) at 60°/sec during baseline and experimental testing

Average Power	Placebo (n = 6)	Creatine (n = 9)
Before Supplementation		
Set 1	191.3 ± 18.3	209.8 ± 17.3
Set 2	183.1 ± 15.3	195.6 ± 16.5
Set 3	151.4 ± 17.0	180.5 ± 17.9
Average of 3 Sets	175.3 ± 16.2	195.3 ± 16.6
After Supplementation		
Set 1	216.7 ± 13.7	218.1 ± 13.6
Set 2	203.5 ± 12.2	195.8 ± 11.2
Set 3	176.9 ± 10.0	179.4 ± 12.5
Average of 3 Sets	199.0 ± 11.9	197.8 ± 11.6

Note. No significant difference between the trials or the groups.

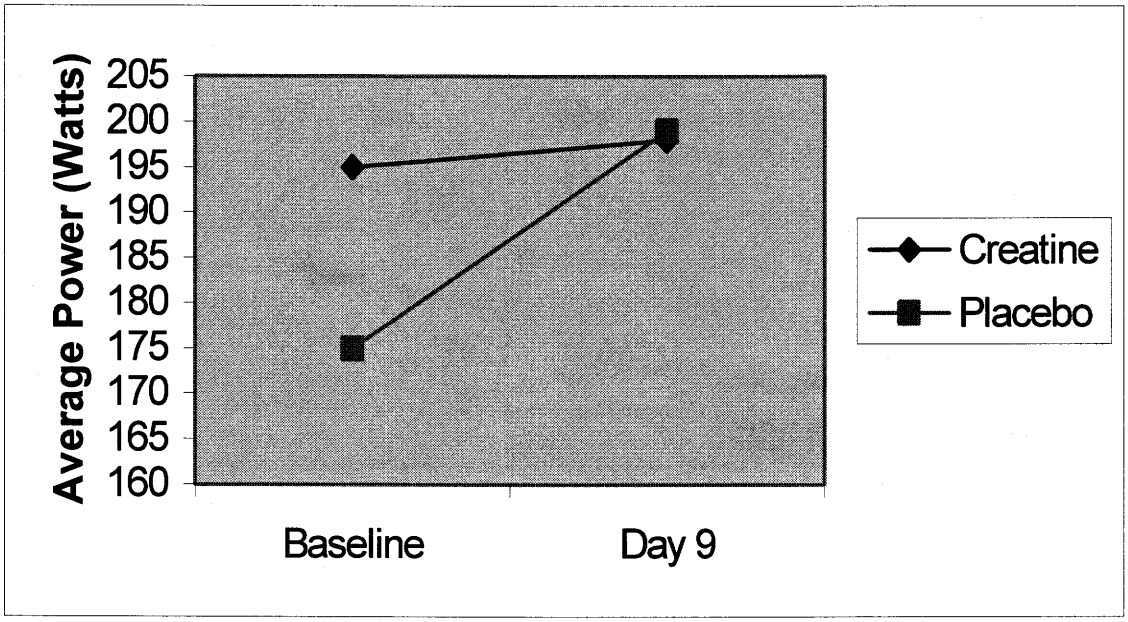

Figure 3.2. Isokinetic average power for knee extension before and after creatine supplementation.

Table 3.7

Isokinetic average power (watts) for knee flexion (mean ± SE) at 60°/sec during baseline and experimental testing

Average Power	Placebo (n = 6)	Creatine (n = 9)
Before Supplementation		
Set 1	107.5 ± 11.2	112.6 ± 12.1
Set 2	103.3 ± 8.7	105.3 ± 11.3
Set 3	86.0 ± 7.7	92.2 ± 10.6
Average of 3 Sets	98.9 ± 8.9	103.4 ± 11.2
After Supplementation		
Set 1	116.6 ± 8.7	121.9 ± 9.8
Set 2	104.0 ± 7.4	105.6 ± 8.1
Set 3	89.0 ± 7.0	91.8 ± 8.5
Average of 3 Sets	103.2 ± 7.5	106.4 ± 8.6

Note. No significant difference between the trials or the groups.

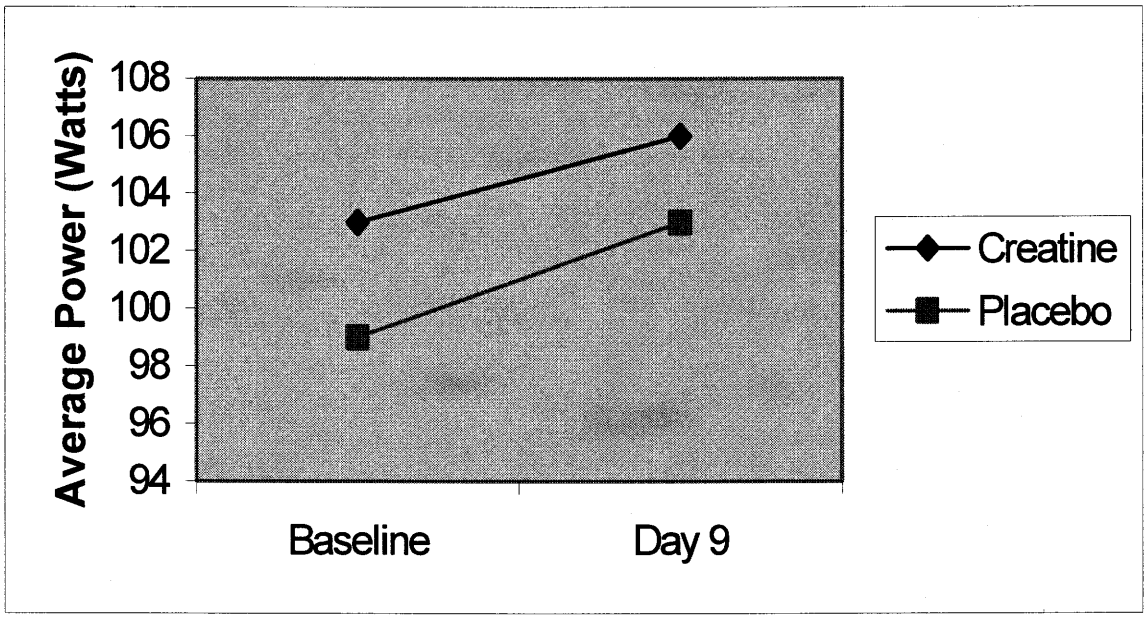

Figure 3.3. Isokinetic average power for knee flexion before and after creatine supplementation.

3.1.6 Peak Torque

The results of a repeated measures ANOVA showed no significant differences between the placebo or creatine trials for knee extension peak torque averaged over the three sets, F (1, 13) = 0.208, $p = 0.656$. Also, ANOVA showed no significant differences in knee flexion peak torque averaged over the three sets, F (1, 13) = 0.083, $p = 0.778$. See Table 3.9 for complete results.

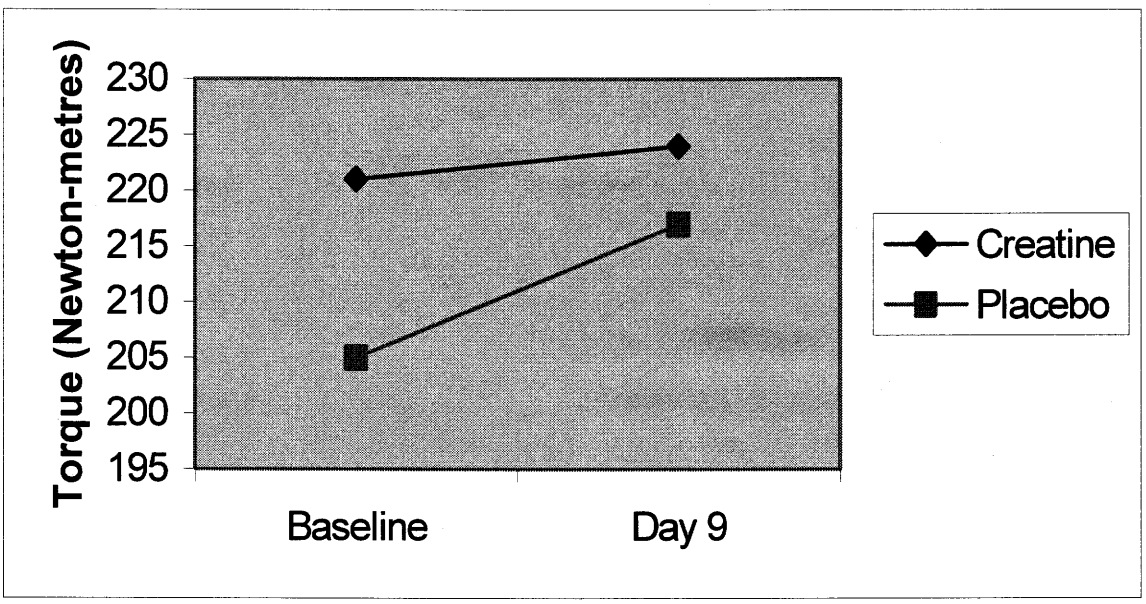

Figure 3.4. Isokinetic peak torque for knee extension both before and after creatine supplementation.

Table 3.8

Isokinetic peak torque (N•m) for knee extension (mean ± SE) at 60°/sec during baseline and experimental testing

Average Power	Placebo (n = 6)	Creatine (n = 9)
Before Supplementation		
Set 1	223.8 ± 19.4	240.0 ± 15.6
Set 2	214.6 ± 18.7	221.0 ± 17.5
Set 3	175.5 ± 19.4	201.4 ± 18.5
Average of 3 Sets	204.6 ± 18.6	220.8 ± 16.2
After Supplementation		
Set 1	247.1 ± 13.9	246.1 ± 13.9
Set 2	213.1 ± 21.4	225.6 ± 11.6
Set 3	190.0 ± 20.3	201.1 ± 11.1
Average of 3 Sets	216.7 ± 16.1	224.3 ± 11.3

Note. No significant difference between the trials or the groups.

Table 3.9

Isokinetic peak torque (N•m) for knee flexion (mean ± SE) at 60°/sec during baseline and experimental testing

Average Power	Placebo (n = 6)	Creatine (n = 9)
Before Supplementation		
Set 1	118.3 ± 10.3	118.9 ± 10.3
Set 2	117.7 ± 10.4	117.5 ± 11.7
Set 3	108.7 ± 9.8	105.3 ± 11.0
Average of 3 Sets	114.9 ± 10.0	113.9 ± 10.8
After Supplementation		
Set 1	126.1 ± 9.9	127.1 ± 9.8
Set 2	121.0 ± 8.6	116.5 ± 9.2
Set 3	108.4 ± 9.0	103.4 ± 8.9
Average of 3 Sets	118.5 ± 9.0	115.7 ± 9.1

Note. No significant difference between the trials or the groups.

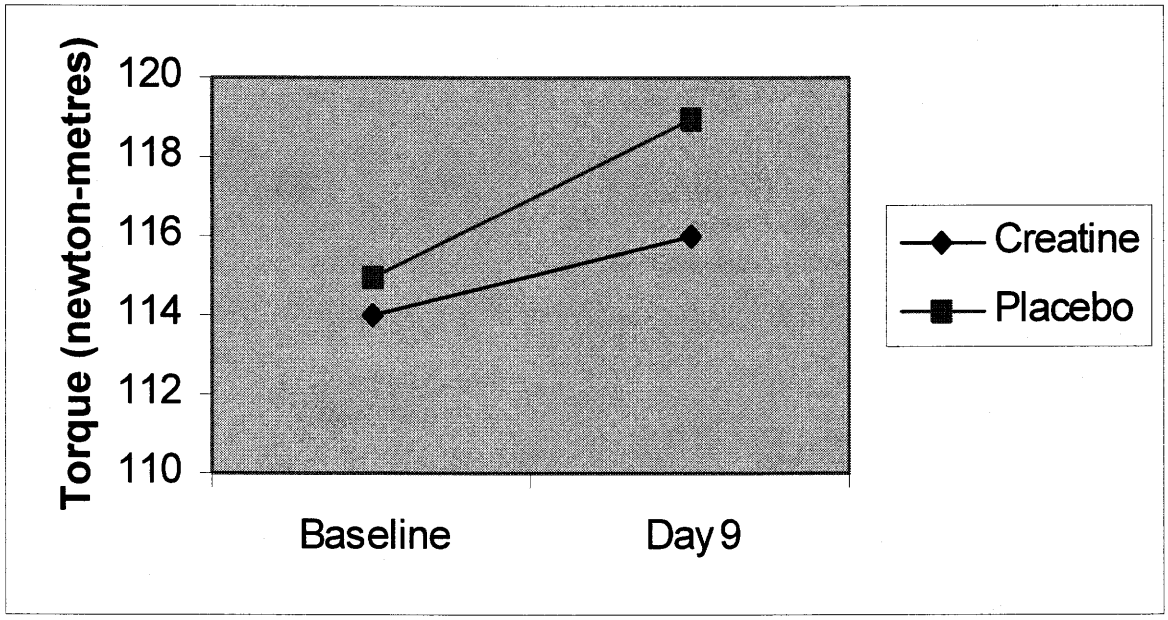

Figure 3.5. Isokinetic peak torque for knee flexion before and after creatine supplementation.

3.1.7 Body Mass

The results of an analysis of variance for body mass indicated no significant difference between the creatine and placebo groups after supplementation, $F(1, 13) = 1.239$, $p = 0.286$. Two subjects in the placebo group were not included in this analysis because they did not show up for the final testing session.

Table 3.10

Body mass in kilograms (mean ± SE) at baseline and experimental trials

Body Mass	Placebo (N = 6)	Creatine (N = 9)
Before Supplementation	82.7 ± 3.3	77.8 ± 3.1
After Supplementation	82.9 ± 3.0	78.6 ± 3.2

Note. No significant differences between trials.

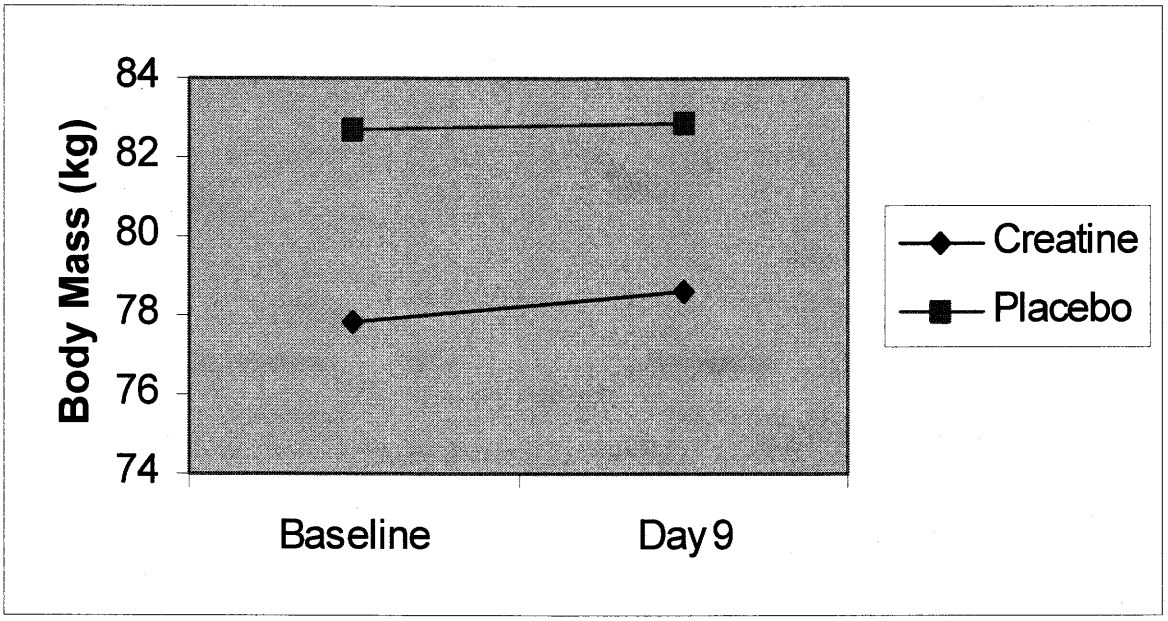

Figure 3.6. Body mass both before and after creatine supplementation.

3.2 Discussion

The purpose of this study was to evaluate the efficacy of creatine monohydrate supplementation in providing an ergogenic effect to repeated sprint skates, muscle power, muscle torque, and blood lactate in male ice hockey players. Subjects were randomized to either a creatine group or a placebo group. All subjects performed one baseline trial and one experimental trial after either placebo or creatine ingestion. The participants were required to complete 10-second skating sprints with 30-second rest periods on a skating treadmill until volitional exhaustion in both trials. Also, subjects were required to perform an isokinetic dynamometer test for repeated knee extension and flexion both before and after supplementation.

The main hypothesis of this study was that creatine supplementation would increase skating time to exhaustion. This hypothesis was based upon the theory that creatine supplementation has the potential to be ergogenic through three possible mechanisms. First, creatine supplementation has been shown to increase PCr stores which should buffer the concentration of ATP for a longer time period during intense activity (Greenhaff, 1997). Secondly, creatine supplementation should accelerate the recovery of PCr between exercise sets (Greenhaff et al., 1994; Smith et al., 1998) and finally, creatine supplementation is theorized to decrease the accumulation of lactic acid during intense activity (Balsom et al, 1994; Kamber et al, 1999). The results of this study indicated that creatine supplementation did not improve skating time to exhaustion or decrease blood lactate values. Also, there was no evidence of an improvement in muscle torque or average power as assessed by isokinetic knee extension and flexion. The results are important to note because a large number of athletes use creatine

supplements in an attempt to improve performance. These results indicate that creatine supplementation did not provide an ergogenic benefit to skating time to exhaustion.

The results of this study do not support the proposed mechanisms by which creatine supplementation may act as an ergogenic aid. A study performed by Odland et al. (1997) indicated that there was an increase in Cr storage within the muscle with creatine supplementation but not an increase in PCr stores. Although not measured directly, the results of the current study suggest that PCr stores were not increased with the supplementation protocol used. If PCr stores were increased, there should have been an increased buffering of ATP and longer exercise performance compared to the control group. Another proposed mechanism for creatine supplementation's ergogenic effect is that it increases the rate of PCr recovery during rest intervals of intermittent activity. Vandenberghe et al. (1999) found that the rate of PCr recovery following isometric gastrocnemius contractions was unaffected by creatine supplementation. This may suggest that creatine supplementation does not improve PCr recovery during rest intervals and thus, the proposed ergogenic mechanism may not be responsible for increases in exercise performance. The results of Vandenberghe's (1999) study may suggest a reason as to why supplementation with creatine did not increase exercise performance in this study.

Research in the past on creatine supplementation has produced conflicting results. The majority of studies report that creatine supplementation seems to improve the work capacity in repeated anaerobic cycling intervals (Balsom et al.1993a; Birch et al. 1994; Casey et al. 1996; Dawson et al. 1995; Earnest et al. 1995; Kamber et al. 1999; Kreider et al. 1998; Schneider et al. 1997). Also, Jones, Atter, and Georg (1999) evaluated the

effect of creatine supplementation on repeated sprint skates in 16 elite ice-hockey players and found a significant improvement in forward skating speed after 10 days of supplementation. The current study utilized time to exhaustion on a skating task as the main dependent variable but found no improvement in skating time to exhaustion after supplementation. Jones et al. (1999) utilized a 10-day supplementation period whereas this study only used a 5-day period. Although it was in accordance to current scientific theory, the shorter supplementation period may not have been long enough to increase the concentrations of Cr and PCr within the muscle to elicit an ergogenic effect. Also, Jones et al. (1999) used hockey players of a different skill level (i.e. professional European league) than the subjects in this study. The athletes in the Jones et al. (1999) study may be considered more elite that the subjects utilized in this study and may have provided a more consistent effort between trials. The testing protocol in the Jones et al. (1999) study was also different from the current study. They used six on-ice repeated sprint skates of 80 meters with a change of direction at 47 meters (from forward to backward). Their results showed an improvement in forward skating speed with supplementation but not backward skating speed. Also the work to rest ratio in the Jones et al. (1999) study approximated 1:1. In the current study the work to rest ratio was 1:3. Paterson (1979) indicated a work to rest ratio approximating 1:3 in a time analysis study of ice hockey play; therefore, we used this work to rest ratio in our study to make the results more specific to ice hockey. The majority of published research indicates that creatine is ergogenic thus, the hypothesis of this study was that creatine would again prove to be ergogenic. The results of this study did not support the proposed hypothesis. The differences between the Jones et al. (1999) and the current

study (i.e. skill level, duration of supplementation, work to rest interval) may suggest a possible reason as to why creatine supplementation was not effective in providing an ergogenic effect in the subjects of this study.

One hypothesis of this study was that blood lactate concentration would be lower during the skating task where the placebo and creatine groups could be compared before fatigue. This hypothesis was based on the theory that an increased concentration of PCr will allow increased capacity of the high-energy phosphate system and a delay in the onset of glycolysis within the skeletal muscle. Blood lactate concentrations have been evaluated before, during and after exercise in creatine supplemented subjects by a number of researchers. Most of the results indicate no significant change or an increase in blood [lactate] after creatine supplementation (Birch et al. 1994; Dawson et al. 1995; Odland et al. 1997; and Snow et al. 1995). There are three studies indicating a lower concentration of blood lactate following exercise. Andrews et al. (1998) found that creatine supplementation decreased lactate concentration, decreased ammonia concentration, as well as improved grip strength in congestive heart failure patients. Balsom et al. (1995) and Kamber et al. (1999) both found that muscle and blood lactate concentration respectively, was lower following creatine supplementation in tests involving repetitive cycle ergometer sprints. This study did not show any statistically significant differences in blood lactate concentration between the creatine and placebo trials after supplementation on any of the sprint skates. This suggests that the Cr supplementation protocol may not have elevated the concentration of Cr and PCr within the muscle to increase the capacity of the PCr system.

Although not significant, there was a mean increase in body mass of 0.8 kilograms in the creatine group as compared to a mean increase of 0.2 kilograms in the placebo group (see Table 3.10). This is consistent with findings of other research which shows increases of 0.7 to 2.8 kilograms after 5-7 days of creatine supplementation (Balsom et al., 1995, 1993a, 1993b; Cooke and Barnes, 1997; Green et al, 1996a, 1996b; Kelly and Jenkins, 1998; Magnaris and Maughan, 1998; McNaughton et al., 1998; Mujika et al., 1996; Noonan et al., 1998; Peeters et al., 1999; Snow et al., 1998;Vandenberghe et al., 1997; Viru et al., 1994; Volek et al., 1997; Vukovich and Michaelis, 1999). An increase in body mass may be responsible for decreased performance in most athletes involved in weight bearing activities. Montgomery (1988), in his review of ice hockey physiology, states that added body mass increases the energy expenditure required to skate at any particular velocity so that the energy systems are taxed to a maximum at a slower velocity. He also states that increased body mass will shorten the duration that an athlete can maintain their pace. With this background, it may be that increased body mass negated the effects of creatine's ergogenic potential in the current study. The subjects in the creatine trial had to try and maintain the same pace as in the baseline trial but with added body mass which would have made the intensity of the skating task more difficult for this group during the experimental trial. Thus, an increase in body mass may suggest a reason as to why creatine was not found to be ergogenic in this experiment.

Creatine has been shown to be absorbed more readily into skeletal muscle of individuals who consume a diet that is low in creatine (Greenhaff et al., 1994). A calculation of the amount of creatine ingested by the subjects before creatine

supplementation was done to assess the amount ingested exogenously each day (See Table 3.9). Normally, 1-2 grams of creatine is ingested per day (Juhn and Tarnoppolsky, 1998). The subjects in the creatine group ingested an average of 0.8 grams of creatine/day before baseline testing. Ingesting this amount per day suggests that their diets may have been low in creatine and that they would respond favorably to creatine ingestion. Although the supplementation protocols followed in this study prescribed to current scientific recommendations, the analysis of creatine, phosphocreatine, or total creatine content via muscle biopsy or ^{31}P-NMR was not assessed in this study. Therefore, conclusions about the effectiveness that the supplementation protocol had on increasing skeletal muscle creatine content cannot be drawn. It may be that the subjects in the creatine group were not responsive to creatine supplementation and thus, creatine supplementation was not ergogenic for these individuals.

A possible limitation of this study is there may have been a learning effect. The results of the analysis of variance indicate a significant time effect for time to exhaustion during the skating test and for average power and peak torque in the first set of knee extension and flexion. The skating treadmill familiarization trial was designed to accustom the subjects to the test procedure thus limiting the chance of a learning effect. The results indicated that eleven of the fourteen subjects increased the skating time to exhaustion from the baseline trial to the experimental trial. Five of the six subjects in the placebo group increased their time to exhaustion. Six of the eight subjects in the creatine group increased their time to exhaustion. This may indicate that

learning had a greater effect on the results than expected, which would suggest that more familiarization trials before testing would be necessary.

Although less likely, the time main effects may be indicative of a placebo effect. That is, the subjects in the placebo group believed they were ingesting creatine, knew of its ergogenic claims, and thus, performed the skating task with more effort and the isokinetic exercise task with more torque and power after supplementation. It was hypothesized that creatine supplementation would maintain power output and peak torque more so than placebo over the three sets of isokinetic testing. The results showed no interaction effect between treatment and time for any of the three sets. This demonstrates that creatine supplementation was unable to produce an ergogenic effect for muscle power and torque in these subjects. If the subjects in the placebo group believed they were ingesting creatine, they may have tried harder in the experimental testing and negated any ergogenic effect that may have been present in the creatine group.

Another possible limitation of the study is dietary intake (kilocalories) during supplementation. ANCOVA results revealed a significant interaction between time and the covariate when kilocalorie's during supplementation was entered as the covariate. This suggests that dietary intake acted as a confounding variable. When looking at the data closely, both the placebo and the creatine group had a decrease in the amount of kilocalories they ingested during the supplementation period. Also, subject compliance was low in filling in the 3-day food diaries (i.e. only 11 subjects completed the diary during supplementation as compared to 15 subjects who completed it before

supplementation). Thus, although dietary intake was a statistical confounding variable, it may not have been a meaningful confounding variable in this study.

A conceivable limitation of this study is that it is not known whether or not Cr supplementation increased the skeletal muscle concentration of Cr or PCr. To control for this limitation, muscle biopsies or nuclear magnetic resonance spectroscopy would need to be done to confirm that Cr supplementation increased the muscle concentration of Cr and PCr. Furthermore, it is quite possible that creatine is not an ergogenic supplement, and that supplementing with creatine provides no additional improvements to exercise or sport performance.

A strength associated with this study is that the isokinetic dynamometer testing was done in addition to the skating treadmill test. This gives the study more credibility because the results indicated no improvement in exercise performance with either exercise test. The isokinetic dynamometer test is a relatively simple motor skill to master and thus, it is doubtful that a learning effect was present in this exercise task. The inclusion of this test in the study helps to draw a more convincing conclusion as to the ergogenic claims of creatine supplementation.

Another strength in this study is the fact that blood lactate concentrations did not differ between the placebo and creatine groups at the same time points during the experimental trial. These results are indicative that creatine supplementation did not provide an ergogenic advantage to the creatine supplemented group. Evaluating blood lactate concentration during the skating test lends credibility to the fact that creatine did not prove to be ergogenic through one of the possible three mechanisms proposed in previous research.

A limitation associated with this study may have been the training status of the individual athletes. The competitive season was just beginning for both teams that participated in the study and thus, all the participants may have not been in the same state of physical conditioning. It is doubtful that the athletes were all prescribing to the same off-season training regime. If the subjects were all at similar fitness levels they may have been able to produce more consistent results. Highly trained athletes would also be more likely to provide a consistent effort between testing trials. If they provided more consistent efforts between trials the creatine supplementation may have had an ergogenic effect.

Another possible confounding variable is that the subjects may have been ingesting creatine before the initiation of the study. Subjects were excluded from the study if it was known they had ingested creatine supplements for up to 6 weeks before the initiation of the study. Research has shown that creatine supplementation will increase the amount of creatine in skeletal muscle and keep it elevated for up to 4-5 weeks after cessation of supplementation (Hultman et al., 1996). If subjects in this study did not disclose that they were ingesting creatine supplements before the study then the results of the study would be inapplicable because of the lingering effects of creatine supplementation before the study.

In conclusion, supplementing a diet with creatine monohydrate did not improve skating time to exhaustion during high intensity intermittent exercise. There is no evidence that creatine was ergogenic for the subjects in this study. An analysis of creatine ingestion in food was done to evaluate whether or not the subjects in the creatine trial already had an elevated level of creatine in their bodies. These results also

indicated no significant differences between the creatine and placebo trials. The results of this study are limited due to the possibility of a learning or placebo effect. Creatine did not improve skating time to exhaustion in ice hockey players performing a repeated sprint skating task on a treadmill.

CHAPTER 4

SUMMARY AND CONCLUSIONS

4.1 Summary

Sports are an integral part of society and have become more and more promoted in recent years. Creatine monohydrate supplementation has become very popular among professional, elite, amateur, and recreational athletes to enhance sport performance. There is a vast amount of research studying the effectiveness of creatine supplementation to enhance sport performance. When only tenths or hundredths of seconds separate the top competitors, there seems to be an increasing amount of research aimed at providing an ergogenic aid to the athletes. Creatine supplementation has been shown to enhance high intensity intermittent exercise and improve strength but, in mass dependent activities, such as running and swimming, and single effort sprints, results are discrepant (Juhn and Tarnopolsky, 1998). Swimming could be considered a mass dependent activity because an individual with more fat free mass will have a higher body density thus, making the individual sink faster in water with the opposite being true for the person with higher fat mass. Creatine supplementation does not improve endurance exercise (Juhn and Tarnopolsky, 1998).

This research was done to evaluate the effects creatine monohydrate supplementation had on high intensity intermittent skating performance in relation to skating time to exhaustion and blood lactate. Also, the effects of creatine supplementation on isokinetic intermittent exercise were evaluated. It was hypothesized that creatine monohydrate supplementation would increase skating time to

exhaustion, decrease blood lactate, increase isokinetic power, and increase isokinetic torque.

In a double-blind randomized order, seventeen competitive male ice hockey players either ingested placebo (n = 8) or creatine supplements (n = 9) and completed a high intensity intermittent skating task, as well as isokinetic strength testing, both before and after five days of supplementation. Creatine ingestion in food and food intake was assessed for a three day period before supplementation and during supplementation. For five days preceding the experimental trial, subjects either ingested creatine 0.30 grams/kilogram body weight or placebo (sucrose). The skating task had subjects skate at 16.1 km/h at 15% incline for 10 seconds, rest for 30 seconds, and repeat the same procedure until volitional fatigue. Blood lactate was measured at baseline and after every odd numbered skating interval. The isokinetic exercise task required subjects to perform 3 sets of 10 repetitions at a maximal effort knee extension and flexion with 60 seconds of rest between sets. The speed of the dynamometer was set at 60 degrees/second. Muscle torque and power was measured throughout this testing session by the computer interfaced with the dynamometer.

Repeated measures ANOVA indicated no significant differences between the creatine and placebo groups for skating time to exhaustion, blood lactate, isokinetic power, or isokinetic strength. The results do not support the hypotheses of this study.

4.2 Conclusions

The results of this study indicate the following conclusions can be drawn:

1. Creatine supplementation did not significantly increase time to exhaustion.

2. Creatine supplementation did not significantly decrease blood lactate concentrations.
3. Creatine supplementation did not significantly increase isokinetic torque.
4. Creatine supplementation did not significantly increase isokinetic power.
5. Creatine ingestion in food and dietary intake did not explain the variability in the results.

4.3 Recommendations for Future Research

This study monitored normal dietary creatine ingestion in participants for three days preceding both testing trials. This factor may influence variability in performance after creatine supplementation (Greenhaff et al., 1994). Although this mediating variable was assessed it was unable to explain the differences in response to creatine supplementation in these subjects. To decrease error, subject characteristics should be more controlled in future research. The results of this research suggest the following recommendations for future research:

1. Evaluate athletes within their competitive season when their level of physical conditioning should be similar instead of during the pre-competitive season when training status may not have been similar.
2. Evaluate fitness levels before testing to ensure a more homogeneous group.
3. Have the subjects perform more familiarization trials to ensure that improvements due to learning are not involved.
4. Include a true control group to evaluate whether or not a placebo effect exists.

5. Evaluate skeletal muscle creatine content via muscle biopsy or ^{31}P-NMR spectroscopy to ensure creatine concentration was increased in the muscle cell.

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APPENDICES

Appendix A Consent Form

CONSENT FORM

Title of Study: The Effect of Creatine Supplementation on Repeated Skating Treadmill Sprint Skates in Male Hockey Players.

Names of Researchers:

Stephen M. Cornish
College of Kinesiology at the University of Saskatchewan
Phone: 306-966-2604

Philip D. Chilibeck
College of Kinesiology at the University of Saskatchewan
Phone: 306-966-6469

The purpose of the study is to examine the possible beneficial effects of creatine ingestion on repeated high intensity skating exercise.

The possible benefits of the study include: an increased high intensity exercise performance, decreased recovery time between bouts of exercise, and increased muscle mass. These benefits are not guaranteed.

The procedures of the study are as follows:

You will be randomly assigned to either a creatine or placebo group. You will be given a preparation of creatine monohydrate and glucose (0.30 g/kg of body weight creatine and 5 g glucose) or a placebo of glucose (0.30 g/kg of body weight). You will take this dose for a period of five days. You will be tested on a number of measurements **before** any supplementation and again on the day **after** supplementation. The first measurement involves repeated high-intensity skating on the skate treadmill. You will be required to skate at a speed of 10 mph and a grade of 15% for a period of 10 seconds. Once this skating interval is complete you will stop and rest for 30 seconds. After the rest interval, you will repeat the same procedure again for as many repeats as possible (until fatigue occurs). A finger tip blood sample will be drawn (1-2 mL) periodically throughout the skating interval's. An estimate of percent body fat will be taken with skinfold calipers both before and after treatment. Lastly, a test of maximal muscular endurance will be done using a Biodex Isokinetic Strength measurement machine.

Potential risks of the study include: muscle cramping, diarrhea, nausea from exercise, and muscle cell water retention.

Your are free to withdraw from the study at anytime and this withdrawal will not affect your academic status or access to services.

All data received and recorded will be kept completely confidential and stored in locked filing cabinets at the University of Saskatchewan. Data will be used to complete a thesis

project for a Master's of Science degree and may be published in a journal article. In either case, your identity will remain confidential.

If you have any questions in regard to the study they should be directed to Steve Cornish at 306- 966-2604 or Philip Chilibeck at 306-966-6469.

You will be advised of any changes in procedures that will have a bearing on your decision to continue in the study.

You will be given feedback on your results at the end of the study.

I acknowledge that the study and contents of the consent have been explained to me, that I understand the contents, and that I have received a copy of the consent for my own records.

Subject's Name (printed): _____

Subject's Signature:_____

Witness:_____

Date:_____

Researcher's Signature:_____

Appendix B Three-Day Food Record

INTRODUCTION

This booklet is used to record you detailed daily food intake. It is meant to give researchers some idea of your *usual* dietary intake. Therefore, it is very important that you do not alter your eating habits while taking part in this study. In other words, do not let the fact that you are writing down what you eat influence your choice of foods. The names of the participants in this study will be kept confidential.

The usefulness of the results of this study depends on the accuracy with which you record your daily food intake. Please write down full details on all the food and drink that you consume each day.

INSTRUCTIONS

1. The purpose of this diary is to record *all* the food (including drinks) which you eat for a three day period.

2. Two pages are provided for each day of the three day period.

3. After each meal or snack that you eat, please write down in detail each separate food item you consumed – including the type of food (e.g. processed cheese) and the amount of food in household measures (e.g. 1 cup of cooked spaghetti). A meal will have to be listed by its separate parts (e.g. fried steak – 8 oz., french fries – 1 cup, coleslaw – 3 tbsp.).

4. The best way to record the information is by carrying this diary around with you wherever you go. Before going to sleep, you should look over the diary and check that you have not missed anything. Remember to include snacks!

5. If you eat fast food, you can just list the type of food you ate (e.g. 1 Big Mac, 1 large fries, 1 chocolate milkshake).

6. The following pages explain the use of household measures, and the description of foods. A sample day's diet sheet is given. Please take the time to read these pages as it will help you to make your diet record more accurate.

RECORDING IN THE DIARY

1. Please use household measures. For example:

   Cup: vegetables, cereal, fruit, milk, beverages
   Tablespoon: sauces, fats
   Teaspoon: sugar, honey, drink mix
   Slices: bread, bacon
   Fractions: 1/6 pie

2. State the type of food eaten. For example:

   Milk: homo, 2%, 1%, skim, goat's
   Cheese: processed, swiss, spread
   Bread: enriched white, 60% whole wheat, sweet cinnamon bun, bran muffin
   Cereal: Sugar Pops, Miniwheats, granola, oatmeal
   Meat: hamburger, fried chicken, scrambled eggs, cod fillets
   Others: strawberry jam, Becel margarine, caesar dressing, oatmeal cookies

3. State the <u>amount</u> of food eaten. For example:

   Cheese: 1" cube cheddar
   3 tbsp lite cream cheese
   ¼ cup 2% creamed cottage cheese
   Fruit: ½ cup canned peaches (in heavy syrup)
   12 grapes
   1 medium banana
   Bread: 2 slices 100% whole wheat
   1 large kaiser
   Cereal: ¾ cup corn flakes
   1 shredded wheat biscuit
   Meat: 1 cup baked beans with pork
   2 cups tuna casserole (tuna, cream of mushroom soup, noodles, peas)
   4 thin slices roast beef
   Vegetables: 2 slices cucumber
   ½ cup boiled cabbage

4. Include manner of cooking: fried, boiled, raw

5. Remember all alcoholic drinks

Here is a sample: Date: Monday March 1, 1999

Time	Food Description	Amount	Code
9:30 a.m.	Waffles – white flour	3, 8" x 4"	
	Syrup – Aunt Jemima	½ cup	
	Yogurt - peach	125 mL	
	Coffee, tsp sugar	1 cup	
	Milk 2%	¼ cup	
10:30 a.m.	Chocolate chip cookies	3	
	Coffee, 1tsp sugar	1 cup	
	Milk (half and half – 10%)	¼ cup	
12:30 p.m.	Sandwich		
	- 2 slices shole wheat bread	2 slices	
	- mozarella cheese (3"x1/4"x2")	2 slices	
	- salami	4 slices	
	- lettuce	1 leaf	
	- butter	1 tsp	
	- mayonnaise	1 tsp	
5:30 p.m.	Spaghetti	1 cup	
	Meat sauce	½ cup	
	Garlic toast	2 slices	
	Milk 1%	2 cups	
8:30 p.m.	Sandwich		
	- 2 slices 60% whole wheat bread	2 slices	
	- peanut butter	1 tbsp	
	- honey	1 tbsp	
	Milk 2%	2 cups	
	*** continue on next page if you need**		
	*** leave CODE column blank**		

Date:

Time	Food Description	Amount	Code