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Creatine Supplementation: It's Association with Muscle Injury in Young Rugby Players.

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A research report submitted to the Faculty of Health Sciences, University of the Witwatersrand, Johannesburg in partial fulfilment of the requirements for the degree of Master of Science in Physiotherapy

Johannesburg

2005

Declaration

I, Lindsay Harris, hereby declare that this research report is my own unaided work, except to the extent indicated in the reference citations and acknowledgements. It is being submitted in partial fulfilment of the requirements for the degree of MSc (Physiotherapy) at the University of the Witwatersrand. It has not been submitted before for any other degree or examination at this or any other University.

DATE: **SIGNED:**

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Abstract

Coaches and athletes are continually searching for ways to gain the "competitive edge" and improve athletic performance. Ergogenic substances and procedures are used routinely at almost all competitive levels. Creatine has become one of the most popular nutritional supplements among athletes in recent times. There is evidence suggesting that there are side effects of creatine supplementation. These include renal stress/failure, muscle injury and cramping. While creatine supplementation has the potential to be a safe and effective nutritional aid, these potential side effects may lead to long term problems in athletes.

The aim of the study was to determine whether creatine supplementation is associated with injury defined as cramping and muscle strains.

This was done using a questionnaire. A questionnaire was developed to consist of three sections determining the player's training program, incidence of injury and use of creatine supplementation, if any. A pilot study was conducted to establish the validity and reliability of the questionnaire, estimate the time for data collection and identify any unanticipated problems. First team rugby players between the ages of 16 and 19 selected from six competitively recognized rugby schools within the Gauteng Province were included in the study.

Consent was obtained from the headmasters of the relevant schools, coaches, and parents/guardians. Questionnaires were completed with the researcher present to explain any part of the questionnaire, which the players did not understand.

Data were analyzed using the odds ratio from a logistic regression.

The results reveal that no association exists between creatine supplementation and muscle injury in the form of cramping and muscle strains. The subjects were not aware of creatine supplementation recommendations and as a result it was being taken inconsistently and haphazardly.

Glossary of terms

Aerobic	:	In the presence of oxygen. Aerobic training is performed to increase aerobic capacity or fitness. The aerobic capacity of an individual is the maximum ability to utilize the body's glycogen stores via the aerobic metabolic pathway. It is measured by determination of the oxygen consumption. The VO_2 max is defined as the maximum amount of oxygen an individual is able to utilize in one minute per kilogram of body weight.
Anaerobic	:	In the absence of oxygen. Anaerobic exercise utilizes anaerobic (oxygen independent) metabolism of glucose to produce energy.
Cramp	:	Muscle cramps are painful, involuntary muscle contractions that occur suddenly and can be temporarily debilitating.
Endogenous	:	Originating internally.
Endurance	:	The time limit of the individual's ability to maintain either a specific isometric force or a specific power level involving combinations of concentric or eccentric muscular contractions.
Ergogenic aids	:	Substances or procedures that are thought to improve physical work capacity or athletic performance.
Exogenous	:	Having an external origin due to external causes.
Performance	:	Performance or fitness is determined by the individual's capacity for energy output (aerobic and anaerobic processes and oxygen transport), neuromuscular function (muscle strength, co-ordination and technique), joint mobility and psychological factors (example motivation and tactics).

pH	:	Expression of a solution's acidity. It is the negative logarithm to the base 10 of H^+ concentration. pH decreases as acidity increases.
Power	:	The rate of performing work; the derivative of work with respect to time; the product of force and velocity (unit: watt).
Strain	:	Muscles are strained or torn when some or all of the fibres fail to cope with the demands placed upon them.
Torque	:	Effectiveness of a force to produce axial rotation (unit: Newton meter).
Work	:	Force expressed through a distance but with no limitation on time (unit: joule or kilojoule).

List of Abbreviations

1RM	One repetition maximum
ADP	Adenosine diphosphate
ALT	Alanine amino transferase
AMP	Adenosine monophosphate
AST	Aspartate amino transferase
ATP	Adenosine triphosphate
CK	Creatine kinase
CM	Creatine monohydrate
cm	Centimetre
CR	Contract relax
Cr	Creatine
d	Day
dm	Decimetre
DOMS	Delayed onset muscle soreness
F	Female
FCr	Free creatine
FFM	Fat free mass
FTF	Fast twitch fibres
G	Gender
g	Gram
h	Hour
Kg	Kilogram
Km	Kilometre
L	Litre

LDH	Lactate dehydrogenase
M	Male
m	Meter
min	Minute
ml	Millilitre
mm	Millimetre
mmol	Millimol
MTU	Musculotendinous unit
N	Newton
No.	Number
P	Phosphate
PCr	Phosphocreatine or creatine phosphate
Pi	Inorganic phosphate
PL	Placebo
PNF	Proprioceptive neuromuscular facilitation
ROM	Range of movement
s	Second
STF	Soft twitch fibres
TCr	Total creatine
Wk	Week
µmol	Micromole

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Chapter 1

1.0 Introduction

Creatine is an amino acid which is synthesized endogenously but which can also be obtained exogenously from the diet (Kreider, 1998b; Balsom et al, 1994). It is stored primarily in the muscle as free creatine (FCr) and phosphocreatine (PCr) (Balsom et al, 1994; Engelhardt et al, 1998; Kreider, 1998b).

During brief explosive type exercises the rate at which energy is supplied to rephosphorylate adenosine diphosphate (ADP) to adenosine triphosphate (ATP) is determined largely by the amount of phosphocreatine (PCr) stored in the muscle (Kreider, 1998b). As phosphocreatine stores become depleted, performance is likely to rapidly deteriorate, due to the inability to resynthesise ATP at the rate required (Kreider, 1998b). Since the availability of phosphocreatine stores in the muscle may significantly influence the amount of energy generated during brief periods of high intensity exercise, it has been hypothesized that increasing muscle creatine content via creatine supplementation may increase the availability of phosphocreatine and allow for an accelerated rate of resynthesis of ATP during and after high intensity, short duration exercises (Balsom et al, 1994; Greenhaff et al, 1993; Harris et al, 1992).

Studies have been performed indicating that creatine supplementation increases muscle creatine content (Bosco et al, 1997; Brannon et al, 1997; Balsom et al, 1995; Casey et al, 1996; Engelhardt et al, 1998; Harris et al, 1992; Hultman et al, 1996; Kraemer and Volek, 1999; Kreider, 1998b; Lemon et al, 1995; Odland et al, 1997; Robinson et al, 1999; Smith et al, 1999; Snow et al, 1998; Vandenberghe et al, 1999 and 1997; Volek et al, 1999), improves anaerobic sprint performance (Bosco et al, 1997; Terrilion et al, 1997), and promotes greater gains in strength (Lemon et al, 1995; Kreider et al, 1998b; Volek et al, 1999) and fat-free mass (Volek et al, 1999) during training.

Coaches and athletes are continually searching for ways to gain the "competitive edge" and improve athletic performance. It is not surprising therefore, that a variety of ergogenic substances and procedures are used routinely at almost all competitive levels. Consequently creatine has become one of the most popular ergogenic aids among athletes in recent times, largely because as a nutritional supplement its use is not considered to be illegal.

There is limited evidence suggesting the side effects of creatine supplementation. These include renal dysfunction, muscle injury and cramping (Kreider, 1998b). Reports in lay articles, advertisements and on the Internet suggest that athletes taking creatine may experience a greater degree of cramping when training in hot or humid conditions. The reason for this may be that creatine promotes water retention (i.e. a shift into the muscle), thus altering muscle homeostasis and potentiating cramping. However no definitive scientific evidence exists to support this hypothesis. The cause of cramping is largely unknown. To date studies (Kreider et al, 1998b) have reported no disproportionate changes in body water, resulting in excessive intracellular fluid retention, plasma volume shifts, and/or electrolyte alterations in response to creatine supplementation. Also, studies have reported no cramping related to creatine supplementation taking into account that most studies are carried out on highly trained athletes during heavy training periods (Kreider, 1998b).

It has also been suggested that creatine promotes a greater incidence of muscle strains due to increases in weight and/or strength. The theory for this is that the gains in strength and body mass may place additional stress on bone, joints and ligaments. Yet, again, there is no study which documents an increased rate of injury following creatine supplementation (Kreider, 1998b)

Creatine may have the potential to be a safe and effective nutritional aid however these alleged side effects may cause long term problems in athletes.

It is clear that athletes and sports people at all levels of participation are continually striving to be the best, and in order to gain this “edge” legal ergogenic aids such as creatine have

become more popular. However with such supplements being used more frequently we need to be sure what the associated side effects might be.

Thus the aim of this study was to determine whether creatine supplementation is associated with muscle injury and/or cramping.

Chapter 2

2.0 Literature Review

2.1 Introduction

This chapter aims to describe what creatine is and the ergogenic benefits of creatine supplementation. The theoretical ergogenic effects including: phosphocreatine availability; increased phosphocreatine resynthesis; reduced muscle acidity; oxidative metabolism; enhanced training and increased body mass will briefly be described. Recommendations on how to supplement with creatine will be introduced followed by the storage of creatine in the body after supplementation. A detailed description of the effects which creatine has on performance will be tabulated as well as its effects on body composition. A brief description of all side effects will be given focusing in depth on muscle cramping and injury. Due to the subjects chosen for this study, an indication of the physical demands of rugby will be presented, namely; physical characteristics and strength requirements of the players.

2.2 Creatine

Creatine is an amino acid (Kreider, 1998b). Three amino acids are involved in the synthesis of creatine namely glycine, arginine and methionine (Brannon et al, 1997; Balsom et al, 1994; Harris et al, 1992; Kreider, 1998b). In humans the enzymes involved in the synthesis of creatine are located in the liver, pancreas and kidney, therefore creatine is produced outside of the muscle and transported into the muscle via the bloodstream (Balsom et al, 1994; Brannon et al, 1997; Engelhardt et al, 1998; Walker, 1979). The normal concentration of creatine in plasma is 50-100 μmol/L (Balsom et al, 1994) while the normal creatine concentration in muscle ranges between 100 – 140mmol/kg dry mass in the human population studied (Kraemer and Volek, 1999). In the absence of exogenous creatine, the rate of turnover of creatine to creatinine is approximately 1.6% per day. Therefore with a body weight of 70kg and a total creatine pool of 120g; the turnover is approximately 2g/d (Balsom et al, 1994;

Ekblom, 1996; Harris et al, 1992; Kreider, 1998b; Kraemer and Volek, 1999). The creatinine is filtered in the kidney by simple diffusion and excreted in the urine. Any excess creatine is also excreted in the urine (Kraemer and Volek, 1999). Based on measurements of renal excretion of creatinine, the daily requirement for creatine supplied though the diet and from endogenous synthesis in a 70kg man approximates 2g/d (Walker, 1979).

Creatine is replaced through endogenous and exogenous sources. The endogenous creatine synthesis is believed to be at least partly regulated by exogenous intake most likely by a feedback mechanism (Balsom et al, 1994). Creatine is found mostly in meat, fish and other animal products with only trace elements in some plants (Balsom et al, 1994; Ekblom, 1996; Engelhardt et al, 1998; Kraemer and Volek, 1999; Kreider, 1998b; Walker.1979). For example, about one gram of creatine is found in 250g raw red meat (Kreider, 1998b). The average intake of creatine from a mixed diet is estimated to be one gram per day. While at least part of the daily creatine requirement can be attained from the diet, this needs to be complemented by endogenous synthesis. On a creatine free diet, as is the case with vegetarians, the daily needs are met exclusively by means of endogenous synthesis (Balsom et al, 1994; Engelhardt et al, 1998; Harris et al, 1992). The presence of all the enzymes needed for the biosynthesis of creatine in our bodies, excludes dietary creatine as an essential nutrient in our diet (Kraemer and Volek, 1999).

Of the total creatine pool, 95% in humans is found in skeletal muscle therefore skeletal muscle is the target tissue for loading with creatine supplements (Balsom et al, 1994; Kreider, 1998b; Kraemer and Volek, 1999). Of the remaining 5%, the highest levels are found in the heart, brain and testes (Balsom et al, 1994; Kreider, 1998b). In skeletal muscle, phosphocreatine accounts for about two thirds (66%) of the total creatine pool (Balsom et al, 1994; Engelhardt et al, 1998; Kreider, 1998b) and is unable to pass through membranes, thus trapping creatine in cells (Greenhaff, 1997). The remaining 30-40% remains as free creatine. However, creatine possibly binds to intracellular components, which may further facilitate muscle creatine retention (Walker, 1979). Creatine is an osmotically active substance; thus an increase in intracellular creatine concentration induces the influx of water into cells (Volek et al, 1997).

2.3 Ergogenic Benefits of Creatine Supplementation

2.3.1 An Energy Substrate for Muscle Contraction

The immediate energy source for skeletal muscle contraction is ATP (Balsom et al, 1994; Bosco et al, 1997; Engelhardt et al, 1998; Kamber et al, 1999; Mujika et al, 1996). During muscle contraction ATP is hydrolysed to ADP and must continuously be replenished to maintain usable energy levels (Balsom et al, 1994; Brannon et al, 1997; Casey et al, 1996; Ekblom, 1996; Kraemer and Volek, 1999; Mujika et al, 1996).

(Exercise) ATP − APTase → ADP + Pi

During intense exercise, the rate of ATP hydrolysis is extremely high i.e. the reaction above shifts to the right (Kraemer and Volek, 1999).

With rapid increases in energy demand, the phosphocreatine is degraded and phosphate is donated to ADP to regenerate ATP. Energy derived from the degradation of PCr allows the ATP pool to be turned over several dozen times during all-out maximal-effort exercise. This reaction is catalysed by the enzyme creatine kinase and leads to the accumulation of free creatine (FCr) in active muscles.

(Exercise) PCr (Cr phos) + ADP − (Creatine kinase) → FCr + ATP

Thus phosphocreatine functions as a "temporal energy buffer" during periods of rapid ATP turnover as occurs at the onset and during intense exercise when ATP consumption exceeds synthesis (Balsom et al, 1994; Bosco et al, 1997; Brannon et al, 1997; Casey et al, 1996; Engelhardt et al, 1998; Kraemer and Volek, 1999; Odland et al, 1997; Greenhaff et al, 1993). Although there is about three to four times more PCr in the muscle than there is ATP, its supply is also limited and needs to be replenished in order to maintain very high intensity exercise.

During recovery from exercise, the creatine kinase reaction is reversed and PCr is replenished (Balsom et al, 1994; Bosco et al, 1997; Brannon et al, 1997; Casey et al, 1996; Engelhardt et al, 1998; Kraemer and Volek, 1999; Odland et al, 1997).

(Recovery) Cr + ATP − (Creatine Kinase) → PCr + ADP

Phosphocreatine resynthesis is rapid with a half-life of approximately 30 seconds. Approximately 95% of PCr is resynthesised after only three or four minutes (Kraemer and Volek, 1999).

Phosphocreatine also functions in a "phosphocreatine energy shuttle" therefore PCr acts as an energy carrier transporting energy from mitochondria to different ATPase sites in the cytosol (Balsom et al, 1994).

The advantage of using PCr as an energy buffer is that it is a high power energy system (i.e. it produces a large amount of ATP per unit of time), however, as the storage capacity is relatively small, phosphocreatine is depleted quickly during maximal activity (10-20 seconds) (Ekblom, 1996; Kraemer and Volek, 1999). Therefore the availability of phosphocreatine for muscular contraction has been considered a limiting factor for short-term supramaximal exercise performance. An increase in the total muscle PCr content would limit the rate of depletion of the PCr stores during intense muscular exercise, and the rate of decline in the ATP resynthesis. Enhanced rephosphorylation of ADP can be achieved by increasing muscle PCr content with supplementary Cr ingestion (Kraemer and Volek, 1999; Mujika et al, 1996).

PCr + ADP + H^+ → ATP + Cr

Phosphocreatine may help buffer H^+. By increasing the cell's buffering capacity, the reaction above may serve to attenuate the decline in pH levels during intense exercise and may delay fatigue (Walker, 1979).

2.4 Theoretical Ergogenic benefits

2.4.1 Phosphocreatine availability

During a brief bout of high intensity exercise the ATP demand in the working muscles can increase several hundred fold higher than at rest (Balsom et al, 1994). It is estimated that with high intensity exercise PCr stores can be totally depleted within ten seconds (Balsom et al, 1994).

Fast twitch fibres (FTF) have a higher total creatine content than slow twitch fibres (STF) and the rate of degradation is greater in type II (fast) muscle fibres, compared to type I (slow) muscle fibres (Engelhardt et al, 1998; Kraemer and Volek, 1999; Casey et al, 1996). A study by Casey et al (1996) reports that the resting PCr concentration in type II muscle fibres is 12% greater than that of type I muscle fibres and the rate of PCr degradation during 30 seconds of maximal intensity dynamic exercise is 10-25% greater in type II muscle fibres. They report that the decline in muscle force during contraction is because of a fall in energy provision from PCr in type II muscle fibres. Thus the availability of PCr as an energy substrate in type II muscle fibres is considered to be a possible limiting factor for maintaining muscle forces during high intensity exercise (Balsom et al, 1994.).

2.4.2 Increased Phosphocreatine Resynthesis

Bogdanis et al (1995) reported that PCr resynthesis during recovery from high-intensity exercise appears to be a determining factor in restoration of energy for a subsequent high-intensity exercise task. Greenhaff (1995) indicated that the availability of FCr has been ascribed a central role in the control of PCr resynthesis, and he also noted (1997) that the acceleration of post exercise PCr resynthesis would be expected to increase muscle contractile capability by maintaining ATP turnover during exercise.

The resynthesis of PCr in human skeletal muscle is an oxygen-dependent process with a fast and a slow component (Balsom et al, 1994). Following high intensity exercise approximately half of the pre-exercise PCr content is restored within one minute of recovery. Total resynthesis of PCr was complete after approximately five minutes in studies done by Soderlund et al, (1991) as cited by Balsom et al, (1994).

Creatine supplementation is reported to enhance the rate of ATP and PCr resynthesis following intense exercise. The increased total Cr and PCr concentrations maintain ATP concentration during high intensity exercise as well as enhance PCr resynthesis (Balsom et al, 1994; Bosco et al, 1997; Brannon et al, 1997; Casey et al, 1996; Engelhardt et al, 1998; Kraemer and Volek, 1999; Kreider, 1998b; Mujika et al, 1996; Smith et al, 1999; Snow et al, 1998; Volek et al, 1999).

An increase in the rate of PCr resynthesis during recovery between bouts of exercise, and thus higher PCr levels at the start of the subsequent exercise bout, is believed to be the primary mechanism explaining the ergogenic effects of creatine supplementation during intense intermittent protocols (Kraemer and Volek, 1999). It is thus the increased amount of Cr, which promotes the resynthesis of PCr if adequate ATP exists (Bosco et al, 1997).

However, not all studies show that Cr supplementation enhances ATP and PCr resynthesis (Casey et al, 1996; Cooke et al, 1995; Snow et al, 1998; Vandenberghe et al, 1999). These authors suggest that Cr supplementation does cause a small but significant increase in TCr content. This increase does not however result in an improved sprint exercise performance or any alterations in markers of muscle anaerobic energy metabolism during and on recovery from sprint exercise. The likely explanation for this is that the increase in TCr content after Cr supplementation is insufficient to induce an enhanced sprint performance and to allow an improved rate of PCr resynthesis after exercise.

2.4.3 Reduced Muscle Acidity

Glycolysis may be modulated by PCr (Walker, 1979). Phosphofructokinase, the rate-limiting enzyme in glycloysis, is partially inhibited by PCr working in concert with inhibitory ATP. During strenuous muscular activity, this inhibition would limit the H^+ production, which occurs during glycolysis. The increased hydrogen ion concentration is a possible contributor to muscle fatigue.

Phosphocreatine acts as the principal metabolic buffer in muscle, accounting for around 30% of the total muscle buffer capacity (Hultman and Sahlin, 1980). ATP resynthesis from ADP and PCr consumes a hydrogen ion in the process, so utilisation of PCr will contribute to the buffering of hydrogen (Harris et al, 1992). Rossiter et al (1996) has suggested that one benefit of an elevated buffer level is that it should allow the muscle to buffer more lactic acid before reaching a limiting muscle pH, and thus allow more high intensity exercise to be performed.

Although some researchers report lower post exercise lactic acid levels following creatine supplementation, even in spite of a higher workload (Soderlund et al, 1994), most studies reveal no effect of creatine supplementation on blood lactate (Greenhaff et al, 1993; Mujika et al, 1996). This observation would depend on the sensitivity of blood lactate as a reflection of muscle lactate levels.

2.4.4 Oxidative Metabolism

Although PCr breakdown to provide ATP is an anaerobic process, several investigators speculate that creatine supplementation, possibly by increasing PCr, may modify substrate utilisation and possibly improve performance during prolonged submaximal exercise (Stroud et al, 1994). Creatine supplementation, in combination with training, increases the level of citrate synthase activity, a marker of oxidative capacity, in both fast- and slow-twitch muscle (Brannon et al, 1997).

Engelhardt et al (1998), noted that aerobic endurance athletes have to use anaerobic work only to a limited extent but that this type of work may influence their performance in certain situations, such as anaerobic energy expenditure during intermittent and finishing spurts.

However to date no evidence exists that creatine supplementation influences endurance performance (Kreider et al, 1998b and Engelhardt et al, 1998).

2.4.5 Enhanced Training

Several investigators have suggested that creatine supplementation could benefit athletes over the long term by enabling a higher training load, improving repetitive-interval sprint capacity, reducing training fatigue, and possibly accelerating muscle hypertrophy (Vandenberghe et al, 1997; Volek et al, 1999). This is demonstrated in more detail in 2.7 “Effects on Performance”.

2.4.6 Increased Body Mass

An increased fat-free body weight, or muscle mass, might be advantageous in sports requiring a high absolute power output to resist the inertia of another body or to overcome an external object (Volek et al, 1999). Discussed in further detail in 2.8 “Body Composition”.

2.5 Supplement Recommendations

Creatine supplementation protocols involve a loading and a maintenance phase. A typical loading regime to increase muscle creatine concentrations is to ingest a total of 20-30g of Cr, usually creatine monohydrate (CM), in four equal doses of 5-7g dissolved in about 250ml of fluid, over the course of the day (Harris et al, 1992). This loading procedure is repeated for three to six days. Based on body weight, the recommended loading dose is 0.3g/kg body mass per day for a period of three to six days (Kreider, 1998b; Kraemer and Volek, 1999). Hultman et al (1996) recommended a lower dose of 3g/d over a longer time frame of about one-month as an alternative loading protocol.

A maintenance phase follows the loading phase where the dosages are considerably lower. It is recommended that the athlete consume approximately two to five grams of creatine per day or 0.03g/kg body mass per day (Kreider, 1998b; Kraemer and Volek, 1999).

There is evidence that taking glucose with creatine increases the uptake of Cr into the muscle (Green et al, 1996; Robinson et al, 1999). Kreider et al (1998b) adds that the addition of creatine to the glucose/taurine/electrolyte supplement promotes greater gains in fat/bone free mass.

However the supplement recommendations depend on the desired outcome i.e. for performance enhancement, ingesting 15 to 25g/d (0.3g/kg/d) with glucose for five to seven days and further ingesting five grams per day with glucose to maintain the creatine stores are recommended. If the athlete wishes to enhance performance and also promote gains in fat-free mass, ingesting 15 to 25g/d (0.3g/kg/d) with glucose or carbohydrate/protein until goal weight/body composition is achieved and then continue with a maintenance dose (5g/d) is recommended (Kreider, 1998b). The exact maintenance dose required to retain muscle creatine stores for various populations is not known, but a daily dose of approximately 0.03g/kg should be adequate to replace the normal breakdown of creatine and maintain elevated creatine stores in most cases (Kraemer and Volek, 1999).

There are several types of creatine available. The most common type of creatine examined is CM. Whether other types of creatine, for example creatine citrate and creatine phosphate, have ergogenic aids is unknown (Kraemer and Volek, 1999).

Creatine is available in powder or capsules and other forms including liquid, bars, gels, candy and gum and these may be combined with other ingredients like liquid protein and carbohydrates. Since glucose, sodium, and taurine may enhance creatine uptake by the muscle cell, commercial products blend CM with other substances.

2.6 Storage following Creatine Supplementation

Creatine supplementation in humans is possible by oral administration of CM. On ingestion of five grams CM the plasma level of Cr has been shown to rise within one hour after administration (Harris et al, 1992). The Cr is then transported into the muscle from the

bloodstream. The exact mechanism in which Cr enters human skeletal muscle is not clear (Balsom et al, 1994).

2.6.1 Total Creatine Pool

By following the most common creatine loading protocol of 20-30g of CM daily for five to six days, significant increases in TCr, FCr and PCr occur (Hultman et al, 1996; Vandenberghe et al, 1997: Vandenberghe et al, 1999: Volek et al, 1999).

A study by Harris et al (1992) showed that Cr supplementation increases the TCr of skeletal muscle by up to 30% and that of PCr by up to approximately 20%. No increase in ATP content in the muscle was associated with any increase in PCr content. Muscle ATP content was unaffected by the changes in TCr content. They add that the favourable effect of Cr ingestion on metabolism and performance during exercise and recovery may be dependent on the magnitude of the increase in muscle TCr concentration during supplementation.

Casey et al (1996) demonstrated increases in muscle TCr concentration of about 18% and PCr concentrations of about 10% after supplementation with 20g-CM for five days. Once again the changes in both peak and total work production were related to the magnitude of increase in muscle TCr concentration that was shown to determine improvements in exercise performance. Odland et al (1997) noted that although muscle TCr was higher following creatine supplementation there was no change in the PCr content.

The administration of Cr doses in the study by Harris et al (1992) ranged from 70g given over 3, 5 days to 330g over 21 days. However they reported that the greatest uptake occurred within the first two days of supplementation. Therefore three days were shown as sufficient to increase TCr stores but not PCr stores. Other studies investigating the effects of oral Cr supplementation reveal an increase in both FCr and PCr concentrations (Bosco et al, 1997; Brannon et al, 1997; Balsom et al, 1995; Engelhardt et al, 1998; Hultman et al, 1996; Kraemer

and Volek, 1999; Kreider, 1998b; Lemon et al, 1995; Robinson et al, 1999; Smith et al, 1999; Snow et al, 1998; Vandenberghe et al, 1999 and 1997; Volek et al, 1999).

Studies done by Volek et al (1999); Robinson et al (1999) and Vandenberghe et al (1999) show that the increase in TCr content and PCr content is accompanied by an unchanged ATP concentration. This, however, conflicted with studies performed by Smith et al (1999); Snow et al (1998) and Casey et al (1996) in which the increase in TCr and PCr contents was accompanied by a marked decrease in ATP concentration. The authors explain that despite an increase in TCr after Cr supplementation, it is insufficient to induce an enhanced performance and allow an improved rate of PCr resynthesis after exercise.

The preceding studies show that creatine supplementation is effective in increasing muscle TCr, PCr or both. These studies were performed over five to seven days with subjects ingesting between 100 and 150 grams of Cr over five days (5X20g or 5X30g) Kreider et al (1998b). The creatine supplementation does not appear to alter pre-exercise concentrations of ATP but elevated phosphocreatine concentrations serve to maintain the ATP concentration to a greater degree during maximal effort performance (Kreider, 1998b).

The only study that did not show an expected increase in resting PCr levels post oral creatine supplementation was that performed by Cooke et al (1995). These authors did not do anything differently but propose that the ergogenic potential of Cr supplementation is related to an increased capacity for PCr resynthesis during recovery rather than to a pre-exercise elevation of intramuscular PCr stores

The amount of exogenous uptake is influenced by a number of factors. The increases are subject to large individual variances and the uptake of creatine is related to the initial TCr levels. It is reported that vegetarians have a greater uptake because of the lower initial values of TCr associated with non-vegetarians (Balsom et al, 1994). Harris et al (1992) reported that the effect of Cr supplementation was greatest in subjects with a low initial TCr content and that the effect was to raise the content in these subjects closer to the upper limit of the normal range; in some cases the increase was as much as 50%. This was confirmed by Ekblom (1996)

who added that those subjects who begin with low Cr levels benefit more from supplementation than those who start with higher Cr levels. The Cr levels of the latter subjects may not increase at all (Ekblom, 1996; Odland et al, 1997; Vandenberghe et al, 1999). Studies done by Ekblom in 1996 and Odland et al in 1997 report that the greatest uptake is in the first few days of supplementation i.e. greater uptake will occur on day one compared to day six or seven with a plateau occurring around day four. It appears that there is an upper limit to the amount of Cr which can be stored in muscle. For most participants it seems to be approximately 160mmol/ (kg.dm) (Balsom et al, 1994; Kraemer and Volek, 1999).

2.6.1.1. Creatine supplementation with carbohydrates

Creatine supplementation with carbohydrates, such as glucose will increase Cr transport into muscle even in subjects with high levels of muscle Cr (Green et al, 1996). The latter study used a solution consisting of five grams of Cr and approximately 90 grams of carbohydrate taken four times per day. Increases in TCr and PCr concentrations were noted in both the Cr and the creatine-carbohydrate groups but the increases in the latter group were much larger. Kreider et al (1998b) adds that the addition of glucose/taurine/electrolyte to the creatine supplement promotes greater gains in fat/bone free mass and sprint performance during intense resistance/agility training. It is thus recommended that athletes consume creatine with carbohydrate to further enhance the effects of creatine (Robinson et al, 1999). It is suggested that the effects of the creatine-carbohydrate combination can be attributed to an insulin-mediated effect. It has also been reported that creatine uptake is greater when administered with insulin (Kraemer and Volek, 1999).

2.6.1.2. Creatine Supplementation with exercise

It has been hypothesised that the amount of Cr uptake is greater in an exercised leg as against a non-exercised control leg. Robinson et al in 1999, investigating the difference in the total creatine concentration between the exercised and non-exercised limb five days after creatine supplementation, confirmed this hypothesis. Their results revealed that a single bout of

exhaustive exercise prior to Cr supplementation promotes skeletal muscle creatine accumulation, with this response being restricted to the exercised muscle. It is feasible that the exercise may enhance the Cr accumulation by increasing the maximal rate of Cr transport in the exercised limb.

Harris et al (1992), confirm in a creatine-supplement study using a one-legged protocol that one hour of hard exercise per day increased the TCr content of the exercised leg, but had less additional effect on the contralateral leg. On average the TCr content went from 118 mmol/kg dm at baseline to 148.5 in the control leg and 162.2 in the exercised leg.

2.6.2 Effects on Biosynthesis

It is believed that Cr supplementation suppresses the body's natural Cr synthesis (Balsom et al, 1994; Kreider, 1998b; Kraemer and Volek, 1999; Walker, 1979). It is important to know whether this decline in synthesis is reversed once supplementation is stopped. There is currently no evidence available from human studies to answer this question but results from an animal study show that processes suppressed during creatine feeding return to normal following removal of exogenous creatine (Balsom et al, 1994).

2.7 Effects on Performance

Short term Cr loading ensures that muscle Cr increases by taking a dose of five grams given four times daily for between five and seven days i.e. 20g to 30g/d for five to seven days (Kreider, 1998b; Kraemer and Volek, 1999). Long-term creatine loading refers to a loading period of 20 to 25g/d for between five to seven days followed by a period of maintenance in which the athlete consumes a reduced or similar amount for a period of up to three months. Thus a typical short-term study lasts five to seven days while a long-term study can be between seven to 84 days (Kreider, 1998b).

Table 2.1: High Intensity, Short duration (≤30 sec) Single or Repetitive Isometric (IM) Resistive Exercise Tasks involving the ATP-PCr Energy System

Investigator	Year	Population	N	G	Design	Initial CM dose gXd=dose(g)	Mode	Ergogenic effect as %change	Comments
Bermon et al	1998	Elderly	32	M/F	RDBPC	20X5=100 3X47=141	Leg extension/ Chest press	0	
Lemon et al	1995	Physically active	7	M	RDBPCX	20X5=100	Ankle extension (20X30 s max)	11%	Increase in total and maximal force production

RDBPC = Randomised, double blind placebo control

RDBPCX = Randomised, double blind, placebo control, crossover

The table above therefore shows mixed results in two studies using different study designs and populations.

Bermon et al (1998) had a larger population of elderly men and women and concluded that oral creatine supplementation does not provide additional benefits for maximal dynamic strength and dynamic and isometric endurance of healthy elderly subjects, whether or not it is associated with effective strength training. This suggests that creatine supplementation does not enhance one repetition maximum type performance in this study. Lemon et al (1995) using only seven physically active men, performing 20 maximal isometric ankle extensions, show that Cr supplementation enhances performance by increasing total and maximal force production. The available literature on isometric performance following Cr supplementation is somewhat equivocal. However, the majority of studies report an effect following Cr supplementation. Although isometric force production is a convenient laboratory based methodology, perhaps a contributing factor to the paucity of studies is the limited application of isometric force production to actual sport performance (Kreider et al 1998a).

On the other hand, studies have found that work performed during low intensity muscle contractions, as well as work performed during high intensity muscle contractions, is not enhanced by creatine supplementation (Kurosawa et al, 1997 as cited by Kreider, 1998b; Thompson et al, 1996 as cited by Kreider, 1998b, respectively).

Table 2.2: High Intensity, Short duration (≤ 30 sec) Single or Repetitive Isotonic (IT) Resistive Exercise Performance Tasks Involving the ATP-PCr Energy System

Investigator	Year	Population	N	G	Design	Initial CM dose gXd=dose(g)	Mode	Ergogenic effect as %change	Comments
Kreider et al	1998b	Football players	25	M	RDBPC	15.75X28= 441	1RM bench press, squat, power clean	40%	Increase in total lift volume was greater for CM than PL group.
Volek et al	1999	Resistance trained	19	M	RDBPC	25X7=175 5X77=385	1RM bench press and squat	24% 32%	Bench press and squat increased after 12 wk: bench increased by 5% after 7d.
Bermon et al	1998	Elderly	32	M/F	RDBPC	20X5=100 3X47=141	Chest press, leg press, leg extension	0	

RDBPC = Randomised, double blind placebo control

Both Kreider et al (1998b) and Volek et al (1999) were able to demonstrate using similar populations (i.e. male NCAA 1A football players and resistance trained men) and the same study design (RDBPC) that creatine monohydrate supplementation (varied dosages) positively enhances isotonic resistive exercise performance. Kreider et al (1998b) shows an increased isotonic lifting volume from maximal effort repetition tests on the bench press, squat and power clean, similarly Volek et al (1999) display that creatine subjects' IRM squat increase slightly after one week (4%) and significantly after 12 weeks (32%). They also displayed that the 1RM bench press increased by 5% after one week and 24% after 12 weeks.

In the same study as discussed in Table 2.1 Bermon et al (1998) suggest that creatine supplementation does not enhance 1RM type performance in a group of 32 elderly men and women who were randomly assigned into four groups and put into a strength training programme of three sets of eight repetitions at 80% of 1RM for leg press, leg extension and chest press, three days a week.

Table 2.3: High Intensity, Short Duration (≤30 sec) Single or Repetitive Isokinetic (IK) Resistive Exercise Performance Tasks involving the ATP-PCr Energy System

Investigator	Year	Population	N	G	Design	Initial CM dose gXd=dose(g)	Mode	Ergogenic effect as %change	Comments
Greenhaff et al	1993	Physically active	12	M/F	RDBPC	20X5=100	30 reps X 5 sets	6.8%	Greater absolute leg torque and attenuated decline in torque after CM supplementation
Vandenberghe et al	1997	Healthy, sedentary	19	F	RDBPC	20X4=80 5X70= 350	Elbow flexion power (70% 1-RM 5 X30 reps)	25%	Arm flexion torque was increased in 5th set. CM + resistance training increased
Vandenberghe et al	1999	Healthy volunteers	9	M	RDBPC	25X5=125	5 X30 max voluntary contractions with 2 min rest	5 – 13%	Increased performance similar at both 2 days and 5 days of supplementation
Vandenberghe et al	1997	Healthy, sedentary	19	F	RDBPC	20X4=80	Elbow flexion power at 70% 1-RM: 5 X 30	0	

RDBPC = Randomised, double blind placebo control

Three out of four studies support the ergogenic effect of creatine supplementation on isokinetic resistive exercise performance tasks. Greenhaff et al (1993); Vandenberghe et al (1999) and Vandenberghe et al (1997) show significant improvements in torque during repetitive exercise following short term (≤ 5 days) and chronic (Vandenberghe et al, 1997) supplementation. Vandenberghe et al (1997) found that the results, despite showing a slight improvement in torque, did not produce a significant increase in torque after the acute phase of supplementation, and only after the chronic supplementation period of 70 days, was a significant improvement noted.

Table 2.4: High Intensity, Short Duration (≤30 sec) Single or Repetitive Cycle Ergometre (CE) Performance Tasks involving the ATP-PCr Energy System.

Investigator	Year	Population	N	G	Design	Initial CM dose gXd=dose(g)	Mode	Ergogenic effect as %change	Comments
Balsom et al	1995	Physically active	7	M	SGRM	20X6=120	5 X 6 s 1 X10 s	5%	Increased power during 10 s trial
Casey et al	1996	Healthy	9	M	SGRM	20X5=100	2 X 30 s isokinetic cycling	4%	Increase in peak power and total work for bout 2
Kamber et al	1999	Well trained sports students	10	M	RDBPCX	20X5=100	10 X 6 s maximal sprints (rev/min) with 30 s rest	3.5%	Increase in mean performance for all sprints following creatine supplementation compared to placebo
Kreider et al	1998b	Football players	25	M	RDBPC	15.75X28=441	12 X 6 s sprints	N/A	Total sprint work was increased following CM
Kreider et al	1998a	Untrained/trained	50	M/F	RDBPC	16.5X14=231 15.75X14=220.5	6 X 6 s sprints	N/A	Comparison following CM and Phophogain HP. Both improved average work for each sprint, with no difference between treatments.
Cooke et al	1995	Untrained	12	M	RDBPC	20X5=100	2 X 15 s	0	
Odland et al	1997	Physically active	9	M	SGRM	20X3=60	Wingate	0	
Snow et al	1999	Football players	8	M	RDBPCX	20X35=700 8X35=280	15 X 5 s sprints with 1 min rest	0	

SGRM = Single group, repeated measures

RDBPC = Randomised, double blind placebo control

RDBPCX = Randomised, double blind, placebo control, crossover

Five studies using short term, high intensity repetitive cycle ergometer protocols, with time frames ranging from six to 30 seconds were able to show that Cr supplementation enhanced performance.

Balsom et al (1995) showed that his seven physically active subjects were able to maintain power output, shown by an attenuated rate of decline in pedal frequency at the end of the ten second bout. Using nine healthy males Casey et al (1996) found significant increases (4%) in total work production.

Kamber et al (1999) found that mean sprint performance (rev/min) was increased by 3.5% with this ergogenic effect being most apparent in the latter seconds of repetitive, high intensity, short duration, sprint cycle ergometry. Kreider et al (1998 a and b) echoed the ergogenic effect of Cr supplementation using male football players and both trained and untrained male and female subjects showing that Cr supplementation increases total work repetitive cycle ergometre performance tasks.

In contrast Cooke et al (1995) were unable to report any significant effect on peak power, time to peak power, total work, and an index of fatigue.

Odland et al (1997) investigated the effect of Cr supplementation on the Wingate test. No significant findings were found which were thought to be associated with the insignificant difference in [PCr]/[ATP] ratio despite a significantly higher [TCr]/[ATP] ratio in the muscle obtained via muscle biopsy. Snow et al (1998) also found an increase in [TCr] but none in the [PCr] and concluded that Cr supplementation did not improve sprint performance.

Table 2.5: High Intensity Short Duration (≤30 sec) Single or Repetitve Jumping Tasks involving the ATP-PCr Energy System

Investigator	Year	Population	N	G	Design	Initial CM dose gXd=dose(g)	Mode	Ergogenic effect as %change	Comments
Bosco et al	1997	Sprinters and jumpers	14	M	RDBPC	20X5=100	45 s continuous jump test	12%	Increased work capacity during the first 15 s of the test (7%) with 12% increase from 15 to 30 s

RDBPC = Randomised, double blind placebo control

The efficacy of Cr supplementation to improve jumping performance remains uncertain. This was clearly demonstrated in the study above. It is thought that gains in body mass may be counter productive and may impair vertical jumping ability.

Table 2.6: Intensity Short Duration High (≤30 sec) Single or Repetitive Running Performance Tasks involving the ATP-PCr Energy System

Investigator	Year	Population	N	G	Design	Initial CM dose gXd=dose(g)	Mode	Ergogenic effect as %change	Comments
Stout et al	1999	Football players	24	M	RDBPC	21X5=105	91.4 m (time)	N/A	Decrease in 91.4m time after Phosphagen HP

RDBPC = Randomised, double blind placebo control

Stout et al, (1999) report contrasting results when looking at the ability of creatine (in different forms) to enhance sprint performance. Following Phosphagen HP supplementation, the 100 yard (91.4m) dash time decreased significantly when compared to the placebo. In contrast the subjects using creatine plus glucose did not show a significant improvement in sprint time compared to the placebo. More studies need to be done to show whether or not creatine does significantly improve sprint running performance. It may be that the gain in body mass, which occurs with creatine supplementation, may have an effect on enhancing sprint performance.

Table 2.7: High Intensity Short Duration (≤30 sec) Single or Repetitive Swimming related Performance Tasks involving the ATP-PCr Energy System

Investigator	Year	Population	N	G	Design	Initial CM dose gXd=dose(g)	Mode	Ergogenic Effect as %change	Comments
Grindstaff et al	1997	Junior competitive swimmers	18	M/F	RDBPC	21X9=189	3 X 20 s max swim bench	0	
Mujika et al	1996	Swimmers	20	M/F	RDBPCX	20X5=100	25m	0	

RDBPC = Randomised, double blind placebo control

RDBPCX = Randomised, double blind, placebo control, crossover

The authors discuss a greater change in work in the first sprint for the creatine group compared to the placebo group. There were however large standard deviations around these mean changes in work, which may mean an inter individual response exists with regard to repeated trials.

Table 2.8: High Intensity Prolonged duration (> 30 to ≤ 2.5min) Single or Repetitive Isometric (IM), Isotonic (IT), and Isokinetic (IK) Resistance Exercise Tasks involving the Anaerobic Glycolysis Energy System

Investigator	Year	Population	N	G	Design	Initial CM dose gXd=dose(g)	Mode	Ergogenic Effect as %change	Comments
Smith et al	1998b	Young versus. middle aged	9	M	SGPC	20X5=100	IT Leg exhaustion exercise (X3); bouts 1-2, 2min: bout 3 to exhaustion	30%	Resting and recovery [PCr] increased by 15% (young) and 30% (middle aged). Bout 3 time to exhaustion increased by 30%, 118 s (pre-) vs. 154 s (post-).

SGPC = Single group, placebo control

Smith et al (1998) found that creatine ingestion significantly increased resting and recovery [PCr] in both young (15%) and middle aged (30%) subjects. For all subjects, time to exhaustion in the third set, increased by 30% after creatine supplementation. The authors concluded that creatine supplementation had a greater effect on [PCr] availability and resynthesis in middle aged subjects. This is an unique and interesting finding in light of the fact that the population studied in the vast majority of the creatine supplementation literature consists of young (≤30 years) physically active males.

Table 2.9: High Intensity Prolonged Duration (> 30 to ≤ 2.5min) Single or Repetitive Running Tasks involving the Anaerobic Glycolysis Energy System

Investigator	Year	Population	N	G	Design	Initial CM dose gXd=dose(g)	Mode	Ergogenic effect as %change	Comments
Bosco et al	1997	Sprinters/ Jumpers	14	M	RDBPC	20X5=100	Treadmill(20km/hr at 5%grade) (~60s)	13.2%	Increase in time to exhaustion
Terrilion et al	1997	Runners	12	M	RDBPC	20X5=100	700m run	0	

RDBPC = Randomised, double blind placebo control

Bosco et al (1997) reported a 13% increase in treadmill running (20Km/hr at 5% incline) time to exhaustion (60 sec) following creatine supplementation. However, not all studies performed found that creatine enhanced exercise performance. A study done by Terrilion et al (1997) investigated the effect of creatine supplementation (5g of creatine and one gram of

sucrose or 6g of sucrose 4 times/d) on two repeated maximal running bouts lasting approximately two minutes. Each trial consisted of two 700-meter runs with a 60-minute recovery between the runs. Results reveal that there was no significant difference in running times between the two treatment groups. So at this stage more research is needed to support the ergogenic effect of creatine on high intensity, prolonged, running performance.

Table 2.10: High Intensity Prolonged duration (> 30 to ≤ 2.5min) Single or Repetitive Swimming Tasks involving the Anaerobic Glycolysis Energy System

Investigator	Year	Population	N	G	Design	Initial CM dose gXd=dose(g)	Mode	Ergogenic effect as %change	Comments
Grindstaff et al	1997	Junior competitive swimmers	18	M/F	RDBPC	21X9=189	3 X 100m freestyle cumulative time: 3 X 50m freestyle cumulative style	N	
Mujika et al	1996	Swimmers	20	M/F	RDBPC	20X5=100	50m time 100m time	N	

RDBPC = Randomised, double blind placebo control

Once again, more research is needed to show the ergogenic effect of creatine on high intensity, longer duration, swimming performance as presently studies indicate that creatine supplementation is ineffective in improving swim time for distances from 50 to 100 metres.

Table 2.11: Miscellaneous High Intensity Prolonged Duration (> 30 to ≤ 2.5min) Single or Repetitive Tasks involving the Anaerobic Glycolysis Energy System

Investigator	Year	Population	N	G	Design	Initial CM dose gXd=dose(g)	Mode	Ergogenic effect as %change	Comments
Bosco et al	1997	Sprinters/jumpers	14	M	RDBPC	20X5=100	45s maximal continuous jumping	0	

RDBPC = Randomised, double blind placebo control

Bosco et al (1997) supports other findings that creatine supplementation is not likely to improve high intensity prolonged duration tasks of any nature.

Table 2.12: High Intensity Prolonged Duration (>2.5min) Cycle Ergometre Tasks involving the Oxidative Phosphorylation Energy System

Investigator	Year	Population	N	G	Design	Initial CM dose gXd=dose(g)	Mode	Ergogenic effect as %change	Comments
Engelhardt et al	1998	Triathletes	12	M	SGRM	6X5=30	262 W for 30min followed by 10 X 15 s at 7.5W/kg	18%	Increase in interval power performance. Possible benefit of CM on interval work incorporated into aerobic exercise training

SGRM = Singe group, repeated measures

A report by Engelhardt et al (1998) confirms that creatine supplementation does not enhance exercise lasting longer than 60 minutes. In their study a special exercise test was created for tri-athletes combining endurance and interval performance. After performing a pre-treatment exercise test, the 12 subjects ingested six grams of creatine twice daily for five days and on day six the other exercise test was performed. The results showed that the creatine significantly increased the interval power performance by 18%, but endurance performance was not influenced at all. More studies are needed to show whether or not creatine positively enhances high intensity prolonged duration cycle ergometer tasks.

Table 2.13: High Intensity Prolonged Duration (>2.5min) Running Tasks involving the Oxidative Phosphorylation Energy System

Investigator	Year	Population	N	G	Design	Initial CM dose gXd=dose(g)	Mode	Ergogenic effect as %change	Comments
Balsom et al	1993	Well trained	18	M	RDBPC	20X6=120	6km terrain run time: treadmill run to exhaustion at 125% VO2max	0	
Stroud et al	1994	Physically active	8	M	SGRM	20X5=100	50 – 90% VO2max steady state	0	

RDBPC = Randomised, double blind placebo control

SGRM = Singe group, repeated measures

Both studies show that creatine does not enhance metabolic responses or endurance time in aerobic running performance.

Table 2.14: Miscellaneous High Intensity Prolonged Duration (>2.5min) Tasks involving the Oxidative Phosphorylation Energy System

Investigator	Year	Population	N	G	Design	Initial CM dose gXd=dose(g)	Mode	Ergogenic effect as %change	Comments
Rossiter et al	1996	Rowers	38	M/F	RDBPC	20X5=100	1,000m time	1.1%	2.3 s decrease in rowing time

RDBPC = Randomised, double blind, placebo control

Rossiter et al (1996) show a significant (1.1%) decrease in rowing time, showing that creatine can enhance high intensity prolonged duration tasks, however more research would be beneficial.

There are a number of factors influencing whether or not creatine supplementation is an effective ergogenic aid. Creatine fails as an enhancing ergogenic aid when supplementation regimens are less than 20g/d for 5d resulting in inadequate muscle creatine accumulation in subjects (Kraemer and Volek, 1999), and it also fails to enhance performance when the supplementation regimens involve low-dose supplementation (2 to 3g/d) without any initial higher dose loading period. The increase in muscle total creatine content is therefore insufficient to induce an enhanced performance and allow an improved rate of phosphocreatine resynthesis after exercise (Snow et al, 1998).

Other factors include studies, which used relatively small sample sizes (e.g. less than six subjects per group) or employed crossover experimental designs with less than a five-week washout period between trials (Kreider, 1998b). Creatine supplementation may also be less ergogenic depending on the amount of work and rest ratios performed. Thus, several studies report that creatine supplementation does not affect performance in sprints lasting six to 60 seconds when prolonged recovery periods (5 to 25- min) are observed between sprint trials

(Kreider, 1998b). The use of single versus repeated exercise bouts influences the effect of creatine as an ergogenic aid together with whether the exercise protocols place greater emphasis on aerobic or anaerobic metabolism (Kraemer and Volek, 1999).

2.8 Body Composition

Body mass represents the sum total of body materials, while body weight represents the measurement of body mass, as acted upon by the gravitational force. Of major interest to scientists in exercise and sport science are the four major components of total body mass: Total body fat, fat free mass (FFM), bone mineral content, and body water. Fat free mass represents the body mass devoid of all extractable fat and consisting primarily of muscle, bone, skin, organs, and water.

Creatine is an osmotically active substance; thus an increase in intracellular total creatine concentration as free creatine and phosphocreatine, may induce the influx of water into the cell, increasing intracellular water and, therefore, body mass (Volek et al, 1999). Hultman et al (1996) reported that creatine ingestion largely reduced urinary volume by 0.6L during the initial days of supplementation, therefore suggesting that the increased body mass was likely attributable to body water retention. Volek and Kraemer (1996), also report that the increase in body mass was most likely the result of water retention, as indicated by a decreased urine volume. They added that the impact of creatine supplementation on body composition seems to be mediated more by the long-term use of higher intensities in weight training, rather than the acute effects of short-term water influx, to maintain proper solute to hydration relationships in the muscle cell. Also increased cellular hydration and/or increased PCr may stimulate protein synthesis or decrease protein degradation, possibly increasing FFM (Kreider et al, 1998b; Vandenberghe et al, 1997; Volek and Kraemer, 1996; Volek et al, 1997) although whether this is as a result of creatine use, per se, is undetermined.

Several studies have been carried out in order to determine the short and long term effects of creatine supplementation on body composition. The changes seen are an increase in total body mass (Balsom et al, 1995) an increase in fat free mass (FFM) (Vandenberghe et al, 1997) with

no gain in fat mass (Grindstaff et al, 1997). Most studies indicate that short-term creatine supplementation (20g - 25g/d for 5-7d) increases the total body mass by approximately 0,7kg to 1,6kg (Kreider et al, 1998a; Kreider, 1998b). With longer use, gains are up to 0,8kg to 3kg more than in the matched control groups. These gains depend on the length and amount of supplementation (Kreider et al, 1998a).

2.8.1 Short Term Studies

Short-term studies have been performed on sedentary, physically active individuals and recreational athletes, trained athletes and resistance trained athletes.

Sedentary subjects. Although data is limited, studies do show that individuals do not have to be physically active in order to increase their body weight after creatine supplementation. In a study using 22 young healthy men Green et al (1996) demonstrated that creatine loading increased body mass by 0.6kg, but when the creatine was consumed with 370g of simple carbohydrate, 2.1kg was gained. The placebo group experienced no gain in body mass. In this study one group of subjects exercised for one hour at an intensity of 70% $V0_2max$, in addition to consuming the creatine-carbohydrate mixture. The group that consumed the creatine carbohydrate mixture and exercised for one hour at an intensity of 70% $V0_2max$ did not experience an increase in body mass.

Physically active individuals and recreational athletes. In a study by Balsom et al (1995) body mass increased significantly from 78.3 to 79.4kg following creatine supplementation of 20g/d for 6 days. The control group showed no significant increase in body mass. Stroud et al (1994) performed a study on eight physically active males and demonstrated a 1.0kg increase in body mass after loading with 20g/d for five days. The study did not involve a control group. Lastly, in a double blind, crossover study on active, untrained men, Snow et al (1998), report that after supplementing with 30g/d for five days, body mass increased by one kilogram as compared to the control trial.

Trained athletes. Mujika et al (1996) report an increase in body weight in the creatine group of twenty highly trained competitive sprint swimmers after the supplementation period of five days (5g x 4/d for 5 d) (from 71.7 ± 10.1 to 72.4 ± 9.8kg) with no significant changes in the placebo group (from 70.4 ± 11.1 to 70.1 ± 10.6kg). Even on a lower dose of creatine (6g/d for 5 days) Engelhardt et al (1998) noted an increased body mass of 0.6kg in regional class athletes. No control group was used and the authors failed to indicate whether the increase in body mass was significant when compared to the presupplement measurement.

However, not all studies show significant gains in body mass. For example a study by Terrilion et al (1997) report no significant changes in body weight in well-trained, competitive male runners after creatine supplementation of 20g/d for five days. Grindstaff et al (1997) showed that creatine supplementation (21g/d for 9 days) in seven male and 11 female regional and/or national amateur, competitive swimmers had no significant effect on total body mass, FFM, fat mass, percent body fat, or total body water.

Resistance-trained individuals. A study by Volek et al (1999) performed on 19 healthy resistance trained men over a 12 week period (25g/d for 1 wk and 5g/d as maintenance for 11 wks) revealed no significant difference in the percent body fat and fat mass over the 12 week training period in either the placebo or creatine subjects. The creatine subjects, however, showed an increased body mass of 1.7kg after one week and 5.2kg after 12 weeks. Their fat free mass increased by 1.5kg after one week and 4.3kg after 12 weeks. The delta change in FFM from zero to one week and zero to 12 weeks was significantly greater in the creatine subjects.

Another study by Volek et al (1997), on 13 healthy resistance trained men, reported a 1.3kg increase in body mass after supplementing with creatine at 25g/d for 7 days. In this study the effect of creatine supplementation on seven-site skin fold thickness was evaluated. No effect of creatine supplementation on the sum of skin fold thickness was noted although a mean increase of 4.3mm was found. This suggests that the increase in body mass was accounted for by an increased FFM.

It can, therefore, be seen in short term studies across a diverse range of subjects (sedentary, physically active, trained and resistance trained individuals), that in most of the studies Cr supplementation causes an increase in body mass. In some studies, despite recording weight gain in subjects, this was not significant.

2.8.2 Long-term studies

Long-term studies normally involve a loading phase followed by a longer maintenance phase. Studies lasting 14 days or more are considered long term.

Kreider et al (1998b) performed a 28-day creatine supplementation program on 25 NCAA division 1A football players during resistance/agility training. Two groups were randomly chosen; one took a phosphagen HP placebo (P) containing glucose, taurine and disodium phosphate, the other phosphagen HP containing pure creatine monohydrate (HP). Results revealed that creatine supplementation with glucose, taurine, and electrolytes promoted significantly greater gain in total body mass (P 0.85 ± 2.2kg; HP 2.42 ± 1.4kg), scanned body mass (P 0.77 ± 1.8kg; HP 2.2 ± 1.5kg) and fat/bone-free mass (P 1.33 ± l.lkg; HP 2.43 ± 1.4kg) in comparison with ingestion of the glucose, taurine, electrolyte supplement alone. These increases in body mass could not be explained by disproportionate increases in total body water content, as determined by bioelectrical impedance analysis, thus suggesting that creatine supplementation may promote lean tissue growth during resistance/agility training and that ingestion of creatine with glucose, taurine, and electrolytes may promote greater gains in fat/bone free mass (2.43 ± 1.4kg) than with creatine supplementation alone, (i.e. 1.5kg).

Stout et al (1997) found that eight weeks of creatine supplementation (21g/d for 5 d and 10.5g/d for 5 Id) during the off-season football resistance/agility training, did not significantly increase fat-free mass (2.6 ± 2.0kg) in comparison to the glucose placebo (-0.01 ± 2.6kg). However, addition of glucose, taurine and electrolytes to the creatine supplement, promoted significant increases in fat-free mass (2.9 ± 1.5kg) in comparison with those in the glucose placebo.

Vandenberghe et al (1997) investigated the effects of creatine supplementation in 19 young female volunteers during 10 weeks of resistance training (3h/wk). Four days of high dose (20g/d) creatine supplementation was followed by 10 weeks of low dose (5g/d) creatine supplementation. The placebo group gained an average of 1.0kg, while the creatine group gained 1.8kg, therefore demonstrating no significant differences in body mass between the two groups. The gains in lean body mass in the creatine group (2.6kg) were however significantly greater than those in the placebo group (1.6kg). When compared to the initial values, percentage gains in FFM increased significantly more in the creatine group than the placebo group after both 5 and 10 weeks (creatine group 4.5-5.8%; placebo group 2.5-3.7% respectively).

Nineteen healthy resistance-trained men were matched and randomly assigned in a double blind protocol to either a creatine or placebo group in a study performed by Volek et al (1999). Periodized heavy resistance training was performed for 12 weeks while subjects consumed either a creatine or placebo capsule of 25g/d for one week followed by a maintenance dose of 5g/d for the remainder of the training. After one week body mass (82.1 to 83.8kg) and FFM (68.7 to 70.2kg) were significantly increased in the creatine group but not the placebo group, while after 12 weeks both groups significantly increased body mass (creatine, 82.1 to 87.3kg; placebo 82.9 to 85.9kg) and FFM (creatine 68.7 to 73.0kg; placebo 68.6 to 70.7kg), but the increases were significantly greater in the creatine subjects.

There are however studies which demonstrate no beneficial effects. Bermon et al (1998), report no significant effect of creatine supplementation of 20g/d for five days and 3g/d for 47 days on body mass, body fat, and lower limb muscular volume in sedentary elderly men, or women who either remained sedentary or engaged in eight weeks of resistance training.

However, additional research is necessary to evaluate the effects of creatine supplementation on body composition, fluid retention/total body water content. Additional research should also evaluate the potential additive and/or synergistic effects that creatine, glucose, taurine, sodium phosphate and potassium phosphate may have on lean tissue growth during training.

2.9 Side Effects

To date, fears and cautions about creatine supplementation have many times been mediated by the lack of controlled experimental data and ease of making alleged observations from uncontrolled field environments (Kraemer and Volek, 1999). It is therefore important that comments related to side effects of creatine supplementation be factual evidence and not simply speculation. Although few studies investigating side effects of creatine supplementation exist, discussion about possible side effects is warranted. (Kreider, 1998b).

Researchers have investigated the effects of creatine and phosphocreatine supplementation on various markers of clinical status. These reports may provide valuable information on the medical safety of creatine supplementation.

2.9.1 Suppressed creatine synthesis

It is believed that creatine supplementation suppresses the body's natural creatine synthesis (Balsom et al, 1994; Kreider, 1998b; Kraemer and Volek, 1999). It is important to know whether this decline in synthesis is over, once supplementation is stopped. There is currently no evidence available from human studies to answer this question but results from an animal study show that processes suppressed during creatine feeding return to normal following removal of exogenous creatine (Balsom et al, 1994). It is however known that it takes about four to five weeks after cessation of creatine supplementation for TCr and PCr levels to return to normal, suggesting that the suppression of the endogenous creatine synthesis is reversible (Hultman et al, 1996; Lemon et al, 1995 and Vandenberghe et al, 1997). The potential lag in the reversibility of the suppression of endogenous creatine production after prolonged administration, remains to be studied (Juhn and Tarnopolsky, 1998). Hultman et al (1996) stated that the rate of creatine formation is indirectly proportional to the muscle creatine concentration and indicates that the endogenous production of creatine may not be inhibited after creatine ingestion.

2.9.2 Renal Function

Also of concern is the effect of creatine supplementation on kidney function. High protein diets (>3g/kg.d) have been reported to increase renal stress in renal failure patients and thus there are concerns as to whether creatine may cause renal stress (Kreider, 1998b). Ingesting 15 to 25g per day of creatine increases the protein intake by 0,1 to 0,2g/kg.d but there is no evidence that adding the equivalent of less than one ounce of protein per day (0,1 to 0,2g/kg.d) to the diet, promotes renal stress/failure in healthy subjects (Kreider, 1998b). Although creatinine levels have been reported to be mildly elevated following creatine supplementation (e.g. 1, 2 - 1, 4 mmol/1), these values are within the normal ranges for athletes undergoing intense training (Engelhardt et al, 1998; Kreider et al, 1998b;Vandenberghe et al, 1997). The increased creatinine is likely due to increased release and cycling of intramuscular creatine as a consequence of enhanced muscle protein turnover and/or greater training volume in response to creatine supplementation and not of a pathologic nature (Balsom et al, 1994; Harris et al, 1992; Kreider et al, 1998b; Vandenberghe et al, 1997). The body is able to dispose of the excess creatine without any problem (Poortmans and Francaux, 1999). Recent studies indicate that oral creatine supplements do not induce detrimental effects on the kidneys of healthy individuals, whether they are taken over a short, medium or long term (Poortmans and Francaux, 1999; Robinson et al, 1999). Poortmans and Francaux (1999) state that healthy individuals are not confronted with health risks when consuming reasonable amounts of oral creatine monohydrate. They, however, urge consumers to be tested regularly for excess albumin excretion (>20/xg/min) in urine collected under resting conditions. Juhn and Tarnopolsky (1998) add that individuals with pre-existing renal disease, and those with a potential for renal dysfunction, should carefully be screened and their suitability for creatine supplementation established.

2.9.3 Elevated muscle and liver enzymes

Elevations in muscle and liver enzymes are used as indicators of muscle, heart and liver stress. However, pathologically they may be elevated in response to degenerative muscle disease, myocardial infarction, or liver disease. Intense exercise may also increase muscle and liver

enzyme efflux. It is for this reason that muscle enzyme efflux may be used as an indicator of exercise intensity and training stress. Robinson et al (1999) supply evidence that there are no adverse effects of acute or chronic creatine supplementation on any indices of muscle function measured, and suggest no obvious risk to health when ingesting creatine at recommended doses. Engelhardt et al (1998), show that creatine supplementation does not affect creatine kinase (CK), lactate dehydrogenase (LDH), aspartate amino transferase (AST), alamine amino transferase (ALT), and gamma-glutamine transferase (γGT) levels. A study by Kreider et al (1998b), however, noted increased levels CK(159 vs. 70%), LDH(24 vs. 11%), and ALT(17 vs. -7,3%) in comparison with the placebo group, in a group of football players after ingesting creatine (15.75g/d for 28days) during an intense resistance agility-training program. The authors suggest that this increase may reflect an increased ability to train more intensely after supplementation.

2.9.4 Electrolyte status and blood volume

Since muscle creatine uptake is sodium dependent, it has been queried whether creatine supplementation may influence electrolyte status, blood volume, or both, and therefore promote dehydration, muscle cramping, or both. However, studies indicate that short or long term creatine supplementation does not affect serum electrolyte status or blood volume (Harris et al, 1992; Kreider et al, 1998b).

In studies of preoperative and postoperative patients, untrained subjects and elite athletes with dosages of 1, 5 to 25g per day, the only side effects have been weight gain (Balsom et al, 1994; Kreider et al, 1998c; Sewell et al, 1998).

2.9.5 Gasto-intestinal distress

Diarrhoea and gastro-intestinal pain have been reported anecdotally. However, several studies of performance noted that none of the subjects experienced gastro-intestinal symptoms of any kind (Grindstaff et al, 1997; Kreider et al, 1998b; Vandenberghe et al, 1997). However, the sample sizes in these studies were relatively small (<12 in the experimental group). Because

the average person consumes one to two g/d, it may be reasonable to assume that a loading dose of 20g/d is excessive for the digestive systems of some subjects (Juhn and Tarnopolsky, 1998). However, Poortmans and Francaux (2000) note that even if there are anecdotal reports of gastro-intestinal distress among consumers of oral creatine, these assertions are not supported by real evidence. There is no reason to believe that oral creatine supplementation has any detrimental effect on the gastro-intestinal tract.

2.9.6 Muscle cramping

There have been some reports in lay articles, advertisements, and on the Internet suggesting that athletes taking creatine may experience a greater evidence of cramping when training in hot or humid conditions. It is possible that since creatine may allow an athlete to train more intensely, this may predispose one to dehydration and/or heat injury. Another reason for this may be that creatine promotes water retention (i.e. a fluid shift into the muscle) (Hultman et al, 1996; Vandenberghe et al, 1997), which will alter the electrolyte status, promote dehydration and/or increase thermal stress. Theoretically it is conceivable that a decrease in intracellular water, as mentioned above, may disturb the normal intracellular electrolyte balance, causing muscle cramps. However, supplementation does not lead to dehydration, but actually greater body water content. Yet, the cause of cramping is unknown.

No studies have reported disproportionate increases in total body water, excessive intracellular fluid retention, plasma volume shifts, and/or electrolyte alterations in response to creatine supplementation. Also, no studies have reported cramping related to creatine supplementation, taking into account that most studies are carried out on highly trained athletes during heavy training periods (Kreider, 1998b). According to Kreider (1998b), cramping while training in the heat (e.g. during a two-a-day football practice in autumn) is related to muscle fatigue and dehydration while exercising.

Greenwood et al (2000a) examined the effects of creatine supplementation on cramping/injury rates during a camp in which the subjects trained three times a day. Seventy-two division 1A football players participated in this study. Thirty-eight of the 72 ingested 20-30g/d of creatine

for five to seven days, followed by five to ten g/d of creatine for the 12 day training period. Subjects practised three times per week in environmental conditions ranging from 30-37°C. Results revealed that the incidence of cramping (39%), heat/dehydration (33%), muscle tightness (27%), muscle pulls/strains (32%), non-contact joint injuries (21%), contact joint injuries (36%), illness (50%), missed practice due to injury (26%), players lost for the season (0%), and total injuries/missed practices (33%) were generally lower than the creatine use rate among players. The findings indicate that creatine supplementation does not increase the incidence of injury/cramping during a college football three-a-day training camp. They performed a similar study during a 24-day training period (Greenwood et al, 2000b). Thirty-nine division one-baseball players took part in the study. Twenty-one ingested 15 to 25g/d of creatine for five days, followed by five g/d for the 24-day training period. Results revealed that the incidence of cramping (0%), heat/dehydration (0%), muscle tightness (33%), muscle pulls/strains (40%), non-contact joint injuries (38%), contact joint injuries (33%), illness (67%), missed practice due to injury (33%), players lost for the season (0%), and total injuries/missed practices (42%) were generally lower/proportional to the creatine use rate among players. Once again these findings indicate that creatine supplementation does not increase the incidence of injury/cramping during a baseball 24 day training period.

Hunt et al (1999) examined the effects of creatine supplementation on the incidence of injury during preseason football training in relation to the percent of creatine use among players. Of the 77 division 1A football players that took part in the study, 34 consumed 15.75g/d of creatine for five days followed by 5.25g/d for 20 days; 34 ingested a carbohydrate and protein supplement containing 8.3g/d of Cr; and the remainder were supplied with a carbohydrate/protein supplement containing no Cr. Results revealed that the overall injury incidence in Cr users was significantly lower than the Cr use rate. They observed an interaction where the injuries were significantly lower (36±7%) in the Cr group in the pre-camp training, and 34±5% lower during the camp. Therefore creatine supplementation during intense training may reduce the incidence of injury, particularly during the pre-camp training phase.

Kreider et al (1998b) examined the effects of creatine supplementation on cramping/injury rates during two phases of preseason college football training. Thirty-four of 77 athletes consumed 15.75g/d of creatine for five days followed by 5.25g/d for 20 days. Thirty-four of 100 subjects ingested a carbohydrate and protein supplement containing 8.3g/d of creatine. The remaining subjects, who were either former creatine users or non-users, were provided with a carbohydrate/protein supplement containing no creatine. Pre-camp training consisted of four to five d/wk of resistance training indoors (28±1°C) and sprint agility training outdoors (32±0.9 °C). Subjects practiced two to three times per day at temperatures ranging between 29 to 37°C. Results revealed that the incidence of cramping (0%/35%), heat/dehydration (0%/38%), muscle tightness (33%/42%), muscle pulls/strains (0%/14%), non-contact joint injuries (0%/32%), contact joint injuries (0%/38%), missed practice due to injury (0%/38%), and total injuries/missed practices (13%/34%) were generally lower/proportional to the creatine use rate among players during pre-camp conditioning (44%) and fall camp (34%). Once again these findings indicate that creatine supplementation during pre-season college football training in hot/humid conditions does not increase the incidence of injury/cramping.

Another study by Kreider et al (1998b), evaluated the incidence of side effects in subjects who participated in one of five placebo controlled, double blind studies, investigating the effects of creatine containing supplements. One hundred and sixty four subjects completed a post-study questionnaire describing the positive and negative aspects of the supplement they ingested. Results reveal that no reports of cramping/muscle strains or pulls in subjects taking creatine or the placebos occurred, therefore indicating that creatine supplementation during various exercise training conditions does not increase the incidence of cramping and muscle strains and pulls.

Poortmans and Franscaux (2000), in a recent unpublished study, investigated 12 young, healthy physically active males who received three grams of creatine per day over a period of 28days. Five athletes experienced at least one cramp during sporting activities over the supplementation period (there was no history of cramp for any of the athletes before supplementation). There is however no proof that these cramps are directly related to the creatine supplementation.

Studies that primarily investigated performance, noted that none of the subjects experienced cramping or strains during this study (Greenstaff et al, 1997; Kreider et al, 1998b; Vandenberghe et al, 1997). However, the largest sample size of the creatine group in these studies was 11 subjects. Studies with greater subject groups are needed but to date no evidence of the causal relationship between creatine use and muscle dysfunction exists (Juhn and Tarnopolsky, 1998).

Anecdotal reports of muscle cramping might be due to intensity of exercise rather than creatine supplementation. Staying well hydrated could reduce the risk. Furthermore, psychological stimulation could cause an individual to exercise above his/her optimal intensity (Poortmans and Francaux, 2000).

Although anecdotal reports suggest that creatine supplementation during intense training may promote dehydration and increase the incidence of muscle cramping, available scientific evidence does not support these contentions (Kreider et al, 1998c)

2.9.7 Muscle injury/strains

It has also been suggested that creatine promotes a greater incidence of muscle strains or pulls due to rapid increases in weight and/or strength. Gains in strength and body mass may place additional stress on bone, joints and ligaments. Yet there is no study which documents an increased rate of injury following creatine supplementation (Kreider, 1998b).

Many athletes have been taking creatine for up to 10 years, yet no follow-up studies investigating the effects of creatine have been carried out for more than a year (Kreider, 1998b). Thus, there is much concern about the long-term effects of creatine supplementation. Schilling et al (2001) conducted a retrospective study to examine markers related to health, the incidence of reported side effects and the perceived training benefits in athletes supplementing with creatine monohydrate. Various clinical examinations were performed and a questionnaire completed regarding dietary habits, creatine supplementation, medical, and training history, and perceived effects of supplementation. Subjects were grouped by

supplementation length or no use: Group 1 (control) = no use; Group 2 = 0.8 – 1.0 years and group 3 = 1+years. The results reveal that creatine supplementation ranged from 0.8 to four years. They conclude that the means of all variables within each group fell within normal clinical ranges. There were no differences in the reported incidence of muscle injury, cramps, or other side effects. They conclude that the data suggest that long-term creatine supplementation does not result in adverse health effects. Additional research on the safety of creatine supplementation is needed because of the ongoing dispute on side effects resulting from prolonged use of creatine.

Scientific data concerning the potential clinical effects of creatine supplementation over long periods of time are insufficient and are just starting to be reported in patient populations undergoing clinical intervention (e.g. a myotrophic lateral sclerosis and Huntington's disease) (Kraemer and Volek, 1999). Creatine and phosphocreatine are reported to be used medically to reduce muscle wasting after surgery and to improve heart function and exercise capacity in people with ischaemic heart disease (Pauletto and Strumia, 1996 as cited by Kreider, 1998b; Gordon et al, 1995 as cited by Kreider, 1998b). It may even improve blood lipids in hypercholesteraemic individuals and thus reduce the risk of heart disease (Ernest, Almada & Mitchell, 1996 cited by Kraemer and Volek, 1999 and Kreider et al, 1998b).

In addition, a study by Metzl et al (2001) which aimed to determine the frequency, risk factors, and demographics of creatine use among middle and high school students, via a survey collecting information regarding school grade and gender specific sport participation. They found that despite current recommendations against use in adolescents less than 18 years old, creatine is being used by middle and high school athletes at all grade levels. The prevalence in grades 11 and 12, approaches levels reported among collegiate athletes. They add that until the safety of creatine can be established in adolescents, the use of this product should be discouraged. Ray et al (2001) carried out a study using a questionnaire to assess the awareness and use of creatine supplementation. This questionnaire was completed by 674 athletes from 11 high schools. They conclude that the use of creatine by adolescent athletes is significant but does not conform to recommendation for optimal dosing. They admit the need

for physicians, athletic trainers, and coaches to disseminate proper information and advise these adolescent athletes on creatine use.

Another study using an anonymous questionnaire on creatine use, determined the prevalence, frequency and patterns of creatine use among a local population of high school athletes. Smith and Dahm (2000) observed that male and female high school athletes as young as 14 years use creatine. Of the high school athletes participating in their study, 8.2% reported creatine use. The creatine users seem to believe that creatine improves their performance, but may lack sufficient information to make informed decisions regarding creatine use. They, however, feel that larger scale studies are warranted.

Determining whether creatine supplementation has any short or long-term side effects is an area receiving much additional research attention. If there are side effects from long-term creatine supplementation, it is important to consider the liability of coaches, trainers, universities and athletic governing bodies who provide the athletes with creatine. Thus the athlete taking creatine must be made aware that the possible long-term effects of creatine have not as yet been established.

2.10 The Game of Rugby

2.10.1 The physical demands of rugby

Rugby Union has 15 players a side, seven backs (ball carriers) and eight forwards (ball winners). Three of these forwards are known as loose forwards because of their more agile positioning and involvement in the game. Rugby Union is characterized by rucks, mauls, scrums and line-outs. Open running and tackling are also prominent factors.

A host of factors determine the load on the individual players. Each positional role in rugby has unique demands. The type and frequency of training varies markedly with the level of play, especially in the amateur game. Performance in the game relies on tactical considerations, interplay of the individuals in tactical moves, proficiency of players in the skills of catching, passing, kicking, tackling and those specific to playing positions. The game

requires a mixture of fast reactions, speed, agility, muscular strength, and anaerobic and aerobic power.

The duration of a game of Rugby Union is 80 minutes of play. The time for which the ball is actually in play is consistently measured as less than 30 minutes. Time is spent preparing scrums and line-outs for play to commence. Penalty takers also take time for kicks and time is also needed for players to reform after rucks and mauls have broken down. According to Williams (1976) as cited by Reilly (1990) a typical game would be made up of a 140 sequences of actions, the activity/rest periods being about 20 to 40 seconds.

Estimates of distance covered in a rugby game range from 4.8 to 9.6km (Reid and Willliams, 1974 as cited by Reilly, 1990). According to Docherty (1988), as cited by Reilly (1990), players spend 47% of the total time in low intensity activity (walking and jogging), 6% in intense activity (running and sprinting), 9% in non-running intense activity (tackling and competing for the ball) and 38% standing.

The activity is intermittent but movement of the ball is generally carried out at speed. This is most pronounced in the backs, although forwards are frequently called upon to run all-out. It has been estimated that the forwards in international matches may have to cover one third of the total distance at top speed (Williams, 1976 as cited by Reilly, 1990). However, the fastest runs are expected of the wings, whether in attacking moves or covering in defence.

Although emphasis is placed on anaerobic metabolism during these fast moves, there is ample time to recover during the periods of low intensity activity. Players need a sufficient level of aerobic fitness to be able to sustain activity to the end of the game. All players need muscular power to change speed and direction quickly, when necessary, to slip tackles and support colleagues. The need for muscular strength is most pronounced in the forwards, on whom the team depends for winning possession in scrums, rucks and mauls. Isometric strength in leg, trunk and shoulder muscles is clearly important in scrummaging. Power in these muscle groups is a factor in co-ordinating and controlling the shove against the opposing scrum. Power in the legs is important in jumping to gain possession in the line-outs.

2.10.2 Physical Characteristics

As mentioned above, there are a wide variety of physical characteristics among rugby players. The diversity depends on the positional role, the level of play, and the range of skills required by the game.

The most noticeable difference between Rugby Union backs and forwards is in terms of body size, the latter being on average 20-cm taller (Rigg and Reilly (1998) as sighted by Reilly, 1990). Average values disguise the differences within the forwards and backs: second-row forwards (and lock and no. 8) for example, are taller than the remaining players in the unit and their height rather than their anaerobic power gives them the advantage in jumping in the line-outs. The hooker tends to be the smallest of the forward players, thus the reason the hooker is very often used to throw the ball into the line-out where he would have little chance of acquiring possession.

Body mass is an important factor in scrummaging because it is difficult for forwards to push a heavy pack of opponents backwards. It is preferable that this weight is lean body mass rather than fat, which would constitute an extra energy demand when the forward has to move around the field of play. The heaviest of the top Rugby Union club players examined by Rigg and Reilly (1988) as cited by Reilly, 1990 were the second-row forwards (101±7kg); the lightest were the half-backs with 24kg lean body weight. The forwards are on average 18.3kg heavier than the backs. The body mass of the rugby player falls between 67 and 130kgs. It is important to note that the above statistics are not based on South African data, as this was not available.

2.10.3 Strength Demands

Upper body strength is particularly important for tackling in rugby and it is possible that this function is better developed in the professional Rugby League game whose training regimens are more systematised. It is also important for scrummaging. Performance of press-ups has been found to be superior in first class compared to second class Rugby Union club players,

and among the positional roles was best in front-row players and poorest in second-row forwards (Rigg and Reilly, 1988 as cited by Reilly, 1990).

Jumping ability is important for catching the ball in the line-out and fielding high kicks. Although players at the top level are better jumpers than players at a lower level, the difference between playing positions is not very marked (Rigg and Reilly, 1988 as cited by Reilly, 1990). It would seem that the forwards gain advantage from their height more than from their leg muscle power in jumping for ball possession in the line-out play.

2.10.4 Training

Training for games can be evolved from an analysis of the demands of the game. Players must be prepared for intermittent sprinting with recovery periods that vary markedly between all out spurts. They also need the type of endurance training that will enable them to sustain 80 minutes of play, leaving them capable of all-out effort towards the end of the game.

Rugby is a contact sport and thus there is a recognized need to develop muscle strength in both upper and lower limbs. A variety of muscle groups are used in the different skills of the game such as kicking, tackling and breaking tackles. Weight training can be employed for improving muscle strength; it can also be incorporated into a circuit of exercises for aerobic fitness training. Muscle strength, specific to game skills for forward players, can be developed on a scrum machine. Power in the leg muscles needs to be developed for jumping, accelerating and changing direction of movement rapidly. Agility running drills are best practiced on the playing field.

2.10.5 Decreased Musculotendinous Flexibility

Flexibility is defined as the range of motion (ROM) available in a joint or a group of joints that is influenced by muscles, tendons, ligaments and bones (Anderson and Burke, 1991). In contrast, stretching refers to the process of elongating connection tissue, muscles and other tissues.

Decreased musculotendinous flexibility is commonly regarded as a cause of soft tissue injury. As in the case of warm-up though, there have been no human studies conducted which confirm this. Some reports have claimed a strong association between injuries and hypermobile joints (Rupp et al, 1995), while others claim an association between hypomobility and injuries (Jonhagen et al, 1994; Maffulli et al, 1994). The effects of flexibility on injury prevention are therefore not clear. It is possible that different flexibility is needed in different joints, and for different sports.

Despite the controversies, musculotendinous stretching techniques are widely used and advocated as a means of increasing musculotendinous flexibility in order to prevent injury. Thorough and correct flexibility training is therefore advisable.

The studies which have been conducted indicate that the reasons that decreases in flexibility may predispose to injury are three-fold (Garrett. 1996; Taylor et al, 1990).

1 Inflexible musculotendinous units reach failure at a shorter length than their flexible counterparts when subjected to a tensile/shearing force.
2 Inflexible musculotendinous units are not able to absorb high contraction forces and tissue disruption therefore occurs at a lower contraction force.
3 Incorrectly treated previous injury, which results in scar tissue formation and therefore inflexibility, also predisposes the athlete to further injury.

Stretching techniques have also been shown to increase muscle power and thereby enhance athletic performance (Norris, 1993; Wilson et al, 1992; Worrell et al, 1991). The reasons for this increase in performance are related to the effects of increased elastic strain, which is utilised to reduce stiffness in the muscle (Cameron et al, 1994; Wilson et al, 1992). It is suggested that the muscle, when more flexible, enables greater forces to be absorbed during the eccentric contraction, and consequently more forces are then generated during the concentric contraction (Worrell et al, 1991). The injured athlete may therefore benefit from the increased flexibility as this would lead to greater force absorption and thereby decrease the load on the injured tissues (Worrell et al, 1991).

Young and Behm (2003) compare the effect of running, static stretching of the leg extensors and practice jumps on explosive force production and jumping performance. They found that submaximum running and practice jumps had a positive effect while static stretching had a negative influence on explosive force and jumping performance. They suggest an alternative for static stretching to be considered in warm-ups prior to power activities.

Other reported benefits include both decreased, and alleviation of post exercise muscle soreness (DOMS) (Anderson and Burke, 1991; Agre, 1985), improvement of overall muscle function, such as skill and relaxation, and regaining loss of range of motion during rehabilitation (Millar, 1976).

A decrease in musculotendinous unit ROM can have a number of underlying causes. These include: nerve root tension (Gajdosik, 1991), past or present injury to that particular MTU (Chandler et al, 1990; Worrell et al, 1991), increased age (Booth et al, 1994; Norris, 1993), obesity (Norris, 1993), decreased MTU temperature (Norris, 1993; Rigby, 1964; Sapega et al, 1981), and prolonged usage in a limited ROM (Herbert, 1988; Starring et al,1988). Usage of the MTU in a limited ROM may cause imbalances at joints, postural misalignment and joint dysfunction (Bullock-Saxton and Bullock, 1994; Cahill and Griffith, 1978; Chandler et al, 1990; Cureton, 1941; Herbert, 1988; Starring et al, 1988; Toppenburg and Bullock, 1990; Travell and Simons, 1983; Weber and Kraus, 1949).

Considering the data available, and despite its ambivalence regarding flexibility training, it is still regarded as a vital component of injury prevention. In order to obtain the most significant increases in flexibility, the following scientifically developed guidelines should be prescribed (Norris, 1993).

1. Each musculotendinous structure should be statically stretched for 30-60 seconds. This should be repeated 3-4 times at each stretching session.
2. Stretching should be performed three times daily, for at least three weeks.
3. Stretching should be performed in the non-weight bearing position.
4. Ideally, these stretching sessions should be carried out after a warm-up session.

5. If PNF stretching is used (CR or CRAC); a ten second isometric contraction is performed between the static stretches.

2.11 Conclusion

The literature review above has described creatine and the mechanism in which it works, it has highlighted the ergogenic and ergolytic effects that it has on performance, illustrated the way in which it influences body composition, and shown from the existing literature the adverse affects which this ergogenic aid may have. It has also highlighted the characteristics of the game of rugby union as well as the physical demands of the rugby player. It has illustrated that creatine may be beneficial as an ergogenic aid to a rugby union player as it enhances performance tasks that are characteristic of the game of rugby and facilitates the physical demands of the player. The specific recommendations within the literature which show how to maximise the benefits of creatine are presented, and adverse effects, if the dosages are not adhered to, are shown.

Chapter 3

3.0 Methodology

3.1 Introduction

This chapter describes the method in which the study was carried out. It includes the development of the questionnaire and its contents, the study design, the inclusion and exclusion criteria of the subjects chosen to participate in the study, the procedure in which the study was carried out, and finally the means in which the data were analyzed.

3.2 Development of Questionnaire

The study was carried out using a questionnaire, as the data collection tool.

The first part of the procedure consisted of the compilation of the questionnaire. The questionnaire was divided into three sections.

The first part of the first section evaluated the training program. This included the duration of training, description of typical training sessions obtaining an estimation of the proportion of training per week spent on skills and fitness training respectively, as well as non running fitness training including for example skipping and stair climbing. It also determined other training methods, which the subject carried out. For example, whether or not the subject did any weight training and if so the frequency and duration of the weight training. It determined which body areas were trained, the number of sets and repetitions per exercise. Should any other gym equipment have been used the questionnaire determined the frequency and duration of its use.

The second part of this section established whether the individual stretched, when they stretched and which method of stretching was used. Once again a description of which body areas were stretched was obtained. This included the number of stretches per area in total, how many of each stretch was carried out and the duration of each stretch.

The third part of this section determined whether the subject played any other sport, the age at which he commenced playing the sport, the duration of the season for that sport and the highest level of participation.

The second section determined the incidence of injury and thus the occurrence of muscle strains or "pulls" and cramping. It established where the injury occurred, when it occurred and its possible cause. In an attempt to rule out the incidence of a previous injury at one body part causing a recurrent weakness or vulnerability and therefore injury at this body part, injury of all body parts prior to playing at first team level was determined. Should any injury have occurred both prior to playing at first team level and at first team level in a common body area, these were eliminated from the data. Once the presence of injury and muscle strain had been obtained, detail of the frequency (once, twice or more per season); period (pre-season, pre-match, in season, post season) and possible cause (over/under training, over/under stretching, bad tackle or other) of the injury was determined. The frequency (per practice, game or at rest) and possible cause of cramping was also obtained.

The third section established whether or not the subject used creatine or any other performance enhancer. If a performance enhancer other than creatine was used, these subjects were not included as this was an exclusion criterion for the study. How long the subject had been using creatine, whether the subject was currently using it, at what intervals (yearly, monthly, daily), the dosage (both maintenance and loading), the number of times per day it was taken, the form of creatine that was used (gum, powder, bar, other) and whether something was taken with it (apple/grape juice, water or other) was established.

Finally, it determined details of creatine dosage and the length of time which creatine had been taken if an injury did occur whilst the subject was supplementing with creatine.

The content validity was established by gaining the coaches', various sports physiotherapists', players' and study supervisors' opinion of the questionnaire. The questionnaire described above was drawn up and then handed to five rugby coaches, five sports physiotherapists, and twenty players (not taking part in the study) who were willing to review the content of the questionnaire. Positive and negative points, which they felt existed within the questionnaire, were noted and appropriate changes made.

A pilot study was performed to establish the test retest reliability of the questionnaire, to estimate the time taken for the data collection process and to identify any unanticipated problems. Once the questionnaire was completed, a school that was not going to take part in the study was approached, to carry out the pilot study. It was explained to them that they were going to be used as the pilot study group and would need to complete the questionnaire twice, having a one week interval in-between. The full procedure of the data collection as described below was carried out and all problems identified were noted and corrected before proceeding with the study. There were no differences, when looking at the results of both sets of questionnaires, which were completed by the same group of subjects, one week apart.

3.3 Study Design

A questionnaire based study, using a sample of convenience.

3.4 Subjects

First team school rugby players between the ages of 16 and 19 years were included in this study. These school rugby players were selected from the top six competitively recognized school rugby squads in the Gauteng Province. Each squad consisted of approximately 20 players. Rugby players using performance enhancers other than creatine were excluded from the study. In addition, incomplete questionnaires were withdrawn from the study.

The ideal subject for this research would have been a professional rugby player who used creatine as his only ergogenic aid. Unfortunately, at this level of play, creatine is not the only

aid used to enhance performance. Had this been the case, the sample group would have been too small and accurate results not easily obtained.

To increase the sample size, rugby players at school level were selected. At this level either no ergogenic aid or creatine, as the only legally accepted aid, were used. The negative factors regarding these subjects were that rugby was not the only sport played. Back to back winter and summer sports were encouraged which may have influenced injury incidence.

Table 3.1: List of Inclusion and Exclusion Criteria

Inclusion Criteria	**Exclusion Criteria**
Scholar	Use of performance enhancer other than creatine
Rugby player	Incomplete questionnaires
First team level	
16 to 19 years of age	

Ethical clearance was applied for from the committee for Research on Human Subjects (Medical) of the University of the Witwatersrand. The protocol number for this study is M01-05-09. Informed consent was obtained as described in the procedure below. Confidentiality was ensured by each subject being assigned a number and no names were used. No prejudice was involved for those not willing to participate in the study. No physical or psychological harm was involved.

3.5 Procedure

The first part of the procedure consisted of the compilation of the questionnaire, as described above.

The second part of the procedure consisted of the data collection.

A letter introducing myself and describing the aim and nature of the study was delivered to both the headmasters and sports administrators of each school. One week after the delivery of the letters a meeting was set up telephonically to meet with a representative of each school. Permission was gained from both the headmasters and/or sports administrators of each school for their first team rugby players to participate in the study. Once this was done a date was co-ordinated to meet with the squad (with the coach/sports administrator present) prior to a rugby practice. One week before the date arranged, parent/guardian consent forms were delivered to the school to be handed out to each player. These completed consent forms were then collected on the date arranged to meet the squad. Failure to complete these forms led to exclusion of that player from the study.

On arrival at each rugby practice a brief description of each study was given to the squad. At this stage an opportunity was given to ask any questions regarding the study. Once all questions were answered, assent was obtained from each player to participate in the study. The questionnaires were then handed out to each player. The researcher then proceeded to guide the players through the questionnaire and answer any questions which may have arisen regarding uncertainties within the questionnaire. Each questionnaire took approximately twenty minutes to complete. The questionnaires were completed and handed in prior to rugby practice. Data collection took approximately three weeks, with two schools being visited per week.

3.6 Data Analysis

Of primary interest was to assess whether or not more injuries occured when exposed to the use of creatine. Summary statistics included mainly percentages and cross tabulations. When of interest, associations between variables were assessed using Fisher's Exact Test or Pearson's Chi-square Test. Analysis was performed at the 0.05 level of significance.

Chapter 4

4.0 Results

4.1 Introduction

In this chapter the results of the data analysis will be displayed and described. This will include an observation of the demographic sample, detail of creatine supplementation, and the association of creatine with muscle injury and cramping.

4.2 Demographic Sample

Table 4.1: Position Played by School Rugby Players

Position	N=108	Percentage of sample
1 – Full Back	11	10.19
2 – Centre	8	7.41
3 - Wing	17	15.74
4 – Fly Half	14	12.96
5 – Scrum Half	8	7.41
6 – Eighth Man	9	8.33
7 – Lock	8	7.41
8 – Flank	15	13.89
9 – Hooker	12	11.11
10 – Prop	6	5.56
- Forwards	50	46.30
- Backs	58	53.70

The above shows that there was no difference in the distribution of forward and back positions within the sample. Forward positions included eighth man, lock, flank, hooker and prop, whilst the back positions included full back, centre, wing, fly half and scrum half.

Table 4.2: Age at which Started Playing First Team Rugby

Age Started	N=108	Percentage of sample
18	11	10.19
17	45	41.67
16	45	41.67
15	7	6.48

It can be seen that most first team school rugby players in this sample commenced this level of play at the ages of 16 and 17 years with the remainder being younger (15) and older (18) age groups.

Table 4.3: Age of First Team Rugby Player this Season

Age Now	N=108	Percentage of sample
19	5	4.63
18	42	38.89
17	37	34.26
16	24	22.22

Most first team rugby players in this sample were 17 and 18 years of age with the remainder being from the younger age group of 16 years and very few from the older age group of 19 years.

Table 4.4: Highest Level of Current Rugby Participation

Highest Level	N=108	Percentage of sample
National	0	0
Provincial	13	12.04
First team	95	87.97

It is obvious that the majority of subjects in this sample played at school first team level only, with few playing at a provincial level and none representing the country nationally.

Table 4.5: Number of Seasons Played at Highest Level of Rugby

Number of Seasons	N=108	Percentage of sample
Three	6	5.56
Two	35	32.40
One	67	62.04

Most school rugby players in this sample played at their highest level for only one season, with approximately one third of all subjects playing at this level for two seasons and the remainder for three seasons.

4.3 Creatine Use/Supplementation

Table 4.6: Use of Steroids

Steroids	N=108	Percentage of sample
Yes	1	0.93
No	107	99.07

At this level of participation it was observed that only one subject in this sample made use of steroids and was thus excluded from the study.

Table 4.7: Whether Creatine has ever been taken

Creatine Usage	N=108	Percentage of sample
Yes	50	46.30
No	58	53.70

Out of 108 subjects just less than half (46.30%) had taken creatine at any one stage whilst playing first team school rugby, and slightly more than half (53.70%) had not ever ingested creatine.

Table 4.8: Length of Time Creatine had been used

Length of Dosage		N=50	Percentage of sample
Less 1 season	Yes	26	52
	No	24	48
1 season	Yes	8	16
	No	42	84
2 seasons	Yes	13	26
	No	37	74
3 seasons	Yes	2	4
	No	48	96
More 3 seasons	Yes	0	0
	No	50	100

The above table illustrates that most subjects who did supplement with creatine took it for less than one season (52%), with fewer subjects supplementing for one season (16%), two seasons (26%), and three seasons (4%). None of the subjects supplementing with creatine ever used it for more than three seasons consecutively.

Table 4.9: Currently taking Creatine (at the time of completing the questionnaire)

Creatine	N=50	Percentage of sample
On	2	4
Off	48	96

It was observed that at the time of completing the questionnaire, only two subjects were using creatine and the remaining (96%) was not.

Table 4.10: Intervals at which Creatine was taken

Interval		N=50	Percentage of sample
Years on		0	0
Years off		0	0
Months on	≥3 months	8	16
	<3 months	25	50
Months off	≥3 months	6	12
	<3 months	27	54
Days on	≥ 15 days	2	4
	< 15 days	4	8
Days off	≥ 15 days	0	0
	< 15 days	6	12

It appears that most subjects in this sample had used creatine for less than three months and were off creatine for less than three months.

Table 4.11: Loading Dosage of Creatine used by Creatine Sample (n=50)

Loading Dose gXd (g)	**Frequency**	**Percentage of sample**
0	18	36%
15	1	2%
20	1	2%
25	3	6%
30	2	4%
35	1	2%
42	1	2%
50	2	4%
56	1	2%
60	3	6%
70	1	2%
80	1	2%
100	2	4%
125	**1**	**2%**
140	1	2%
210	1	2%
280	2	4%
400	1	2%
900	3	6%
1,050	1	2%
1,350	1	2%

The recommended dosage for the loading phase is 20 – 30g/d for 5 – 7days or 0.3g per kg of body mass. Thus for an average body weight of 70kg, 21g of creatine will be taken for approximately 6 days. The mean dosage for this phase is thus 126g. From the above table it can be seen that only one subject completed the loading phase as recommended.

Subjects make use of the measuring spoon supplied in the container to estimate the dose of Cr taken. The dosage is therefore as accurate as their recall.

Table 4.12 Maintenance Dosage of Creatine used by Creatine Sample (n=50)

Maintenance Dose gXd (g)	Frequency	Percentage of sample
0	22	44%
7	1	2%
7.5	1	2%
10	3	6%
15	1	2%
20	1	2%
25	2	4%
28	1	2%
40	1	2%
50	1	2%
70	**1**	**2%**
75	1	2%
105	1	2%
275	1	2%
320	1	2%
420	1	2%
450	1	2%
500	1	2%
600	2	4%
700	1	2%
830	1	2%
1,800	1	2%
4,500	1	2%

The recommended maintenance dose is calculated as 0.03g per kilogram of body weight. This phase may last between four to six weeks. For an average body weight of 70kg the daily dosage is 2g (Kreider et al, 1998b). The mean maintenance dosage is thus 70 grams. Once again the table illustrates that from the sample group only one subject carried out the maintenance phase correctly.

Table 4.13 Frequency of dosage taken per day

Frequency Dosage	N=50	Percentage of sample
Once/day	10	20
Twice/day	18	36
Three/day	10	20
Four/day	7	14

This table displays that the number of times that both the loading and maintenance loads of creatine were taken per day was variable, but with the greatest frequency being twice/day (36%) and the least frequent being four/day (14%).

Table 4.14 Form of Creatine taken

Form		N=50	Percentage of sample
Powder:	Yes	47	94
	No	0	0
Gum:	Yes	1	2
	No	46	92
Bar:	Yes	0	0
	No	47	94
Other:	Yes	1	2
	No	46	92

From the above table it is observed that most subjects used creatine in powder form, with only one subject using it in bar form, and one as a gum.

Table 4.15 Fluid taken when Supplementing with Creatine

Fluid		N=50	Percentage of sample
Apple juice:	Yes	40	22
	No	36	72
Grape juice:	Yes	29	58
	No	18	36
Water:	Yes	17	34
	No	30	6
Other:	Yes	10	2
	No	36	72

It can be seen that the majority of subjects using creatine used it with a carbohydrate containing drink (apple and/or grape juice) (58%), whilst the remainder used water (34%). Only two percent used another form being either milk or coffee.

4.4 Injuries and Creatine Association

Table 4:16 Incidence of Injury at First Team Rugby Level and Creatine Supplementation

Body Area	Subject on Creatine (n=50)		Subject off Creatine (n=58)		P Values
	No.	Percent %	No.	Percent %	
Neck	4	50.00	4	50.00	1.00
Shoulder	8	61.54	5	38.46	0.36
Elbow	2	66.67	1	33.33	0.60
Hand	5	71.43	2	28.57	0.24
Chest	0	0	0	0	0
Abdominals	0	0	1	100.00	1.00
Back	4	80.00	1	20.00	0.16
Hip	1	50.00	1	50.00	1.00
Knee	2	28.57	5	71.43	0.46
Foot	15	57.69	11	42.31	0.36

The table above illustrates that there was no association between the incidence of injury and creatine supplementation in any particular body area in this sample. The association between injury at each body area and creatine supplementation was estimated using the Fisher's Exact Test.

Table 4:17 Incidence of Cramping and Creatine Supplementation in First Team Rugby School Boys

Body Area	Subject on Creatine (n=50)		Subject off Creatine (n=58)		P Values
	No.	Percent %	No.	Percent %	
Neck	0	0	0	0	0
Shoulder	2	66.67	1	33.33	0.61
Upper arm	1	50.00	1	50.00	1.00
Forearm	0	0	1	100.00	1.00
Chest	1	25.00	3	75.00	0.63
Abdominals	4	66.67	2	33.33	0.40
Back	3	60.00	2	40.00	0.65
Groin	2	25.00	6	75.00	0.50
Buttocks	3	30.00	7	70.00	0.51
Hamstring	11	34.38	21	65.63	0.20
Quadriceps	10	55.56	8	44.44	0.27
Calf	21	47.73	23	52.27	0.83

This table shows that in this sample of subjects there is no association between creatine supplementation and cramping in any body areas. The Fisher's Exact Test was used to determine the association between cramping at all body areas and creatine supplementation.

Table 4:18 Incidence of Muscle Strains and Creatine Supplementation in First Team Rugby School Boys

Body Area	Subject on Creatine (n=50)		Subject off Creatine (n=58)		P Values
	No.	Percent %	No.	Percent %	
Neck	0	0	0	0	0
Shoulder	8	66.67	4	33.33	0.21
Upper arm	2	66.67	1	33.33	0.60
Forearm	1	100.00	0	0	0.47
Chest	1	100.00	0	0	0.45
Abdominals	3	75.00	1	25.00	0.32
Back	5	41.67	7	58.33	1.00
Groin	10	43.48	13	56.52	1.00
Hamstring	9	40.91	13	59.09	0.80
Quadriceps	6	28.57	15	71.43	0.11
Calf	7	53.85	6	46.15	1.00

Table 4.18 illustrates that there is no association between muscle strains and creatine supplementation in any body areas investigated within this sample. This was analyzed using the Fisher's Exact Test.

5.0 Discussion

5.1 Introduction

In this chapter the association between creatine supplementation and muscle injury, referring to both cramping and muscle strains, will be discussed. Comment will be made on the way in which the subjects within this sample supplemented with creatine, together with a brief discussion on the training programs and injury incidence. The subject selection and questionnaire will be assessed, together with the limitations of the study and possible recommendations for future studies.

5.2 Association between Creatine Supplementation and Muscle Injury

From the results we are able to see that there is no association between muscle injury, including both cramping and muscle strains, and creatine supplementation in this sample of young rugby players. There may be several reasons why this study demonstrates that there is no association present.

Firstly, several subjects were eliminated from the study if they had had an injury in one body part and then experienced an injury in the same body part whilst playing at first team level. The current injury may well have been due to the creatine supplementation but these subjects could not have been included as the previous injury may have resulted in a recurrence of the same injury at that particular body part. In future studies a more accurate investigation of the injury and creatine presence via muscle biopsy could be carried out. This was not pursued in this study due to the ethical limitation of getting parental consent in order to carry out these investigations on adolescents.

Only subjects having an injury at this level of competition and supplementing with creatine were included in the sample group. These current injuries may however have been due to other factors including training methods, participation in strength training programmes with little or no knowledge, advice or supervision, and not carrying out a routine stretch

programme. These subjects were young athletes whose growing tissues were far more vulnerable to developing injuries, especially during the adult growth spurt (Gerrard, 1993). He states that the immature musculoskeletal system has less chance than an adult's of coping with repetitive stress.

Whilst competing at this level, many schools insist that both a summer and winter sport be played, thus the subjects are training back to back, and not focusing on the demands of one sport specifically. Insufficient strengthening, stretching, and game technique, due to poor coaching, may also influence the incidence of injury at all levels of competition. It is recommended that subjects at a higher level of play be investigated in future studies where all training is dedicated to one sport, thus back to back summer and winter sports are not played. Very often however, when playing at a more professional level, the season is longer, which may also influence injury incidence.

It could clearly be seen in the results of this study, that the subjects within the sample who were supplementing with creatine, were not taking it as recommended, but instead using it haphazardly and inconsistently. Very often only one of the phases was carried out and within the phase the correct dosage was not adhered to. These features may result in a false negative being present, when investigating the association between creatine supplementation and injury, particularly if the doses taken were below the recommended dose. In future studies perhaps the means of supplementation needs to be supervised in order to ensure that the recommended dosages are taken.

The presence of muscle strains and cramping may very often be overlooked by subjects as they are not aware of the discomfort experienced. Muscle strains and cramps exist to different degrees and, if not severe, are often played through. Thus these may have been excluded when completing the questionnaire as they were not severe enough to limit function or result in absence from activity. Despite both being defined in detail by the researcher whilst completing the questionnaire, in future studies the researcher should be present throughout the season to assess and grade any injuries that may occur. Thus any injury will be accurately diagnosed and if an association between creatine is found, it will be direct.

However, two groups exist: A control group (n=58) and an experimental group (n=50). The only difference between the control and experimental groups was creatine utilization. A direct comparison between these two groups was made and we believe that we are able to nullify to a large extent these possible sources of error. When looking within the creatine supplementation group no satisfactory control exists and we concede that inappropriate use of CM may have displayed that creatine has no influence on the incidents of injury.

5.3 Creatine Supplementation

The literature clearly suggests the manner in which creatine should be taken during both the loading and maintenance phases. As is evident from the results, very few subjects supplementing with creatine were following the recommended dosage, as can be seen from the quantity taken in each phase, the duration of the phase, the frequency at which it was taken daily, the form most commonly used, and the fluid with which it was taken. The majority of subjects were either above or below the recommended loading and maintenance doses.

It appears that due to the level of competition (first team) at which they were playing; considerable pressure is put onto school boys in first rugby teams to perform maximally throughout the season. This pressure may come from the school (coach and team mates) and in many cases from home (parents). The boys thus may be encouraged to take any legal ergogenic substance which may possibly enhance their performance.

These expensive substances were being taken by the boys and possibly provided or encouraged by the parents. Little time is spent educating the boys on how to take them in order to gain maximal egogenic benefits. It is likely that the coaches and parents are as ignorant as the subjects on optimal supplementation in order to maximize performance enhancement, thus only professionals in the field would be able to advise the players appropriately. It seems as though the idea of “more is better” is practiced and very few are actually aware of the short and long term effects which may arise if proper dosages are not adhered to.

Due to this lack of knowledge it is evident that very few subjects were aware that loading and

maintenance phases existed and that a loading phase is essential to maximize the creatine plasma levels within the body. Once this is completed after approximately five days, a maintenance phase is commenced to maintain peak creatine plasma levels. It is also doubtful that any of the subjects were aware that CP depletion before commencing the loading dose will enhance supplementation. These procedures would maximize the benefits of this ergogenic aid.

Although the subjects used creatine monohydrate in a powder form, few were aware that it existed in other forms including liquid, gum and bars. Those who were making use of these other varieties were not aware of the amount of creatine within each unit. This alone may have resulted in poor adherence to proper supplementation and inconsistent haphazard use. Few were aware that combining creatine with a simple carbohydrate such as glucose would increase creatine transport into muscles and thus aid those who were less responsive to creatine supplementation. It is common knowledge that the response to creatine supplementation differs from person to person.

Apart from the lack of knowledge of optimal dosage, limitation may exist in the complexity of the dosage regime (Morris and Schultz, 1993). Keeping to a dosage regime requires dedication and the following difficulties should be noted:-

- There are two phases of supplementation, namely maintenance and loading, and the following points are common to both:-
    - The duration of each phase differs.
    - The dosage frequency is high and differs in each phase.
    - The dosage itself differs in each phase.
- The maintenance phase (in g) is determined by finding the product of body weight (Kg) and 0.03. This in itself is complicated and requires that subjects be aware of their body weight in order to determine the amount of creatine that they should be taking daily throughout the maintenance phase.

From the results it can be seen that out of 108 subjects only 50 used creatine as an ergogenic aid. Despite the pressures placed on the boys as mentioned above, and the existing high expectations, it may be that many parents do not believe in the use of ergogenic aids in any form at this adolescent age and that its use is strictly forbidden. The expense involved in using creatine may also influence the parents' decision not to support its use, especially since it needs to be taken for prolonged periods during the maintenance phase. Also impacting negatively may be both the parents' and boys' lack of knowledge and commitment to a complicated regime, as discussed above.

5.4 Questionnaire

The main advantage of using a questionnaire is that you are able to design and customize it for the purpose of a study. Another advantage is that the questionnaire does not necessarily have to be administered by the researcher and thus large samples of people can be included in a study.

Questionnaires however also have limitations. This questionnaire was lengthy and despite the researcher being present throughout the completion of the questionnaire in order to guide the subjects and give examples to eliminate any confusion, subjects of this age are easily distracted and may have found this questionnaire complex. The study also relied on subjects' memory which may have influenced some of the results.

5.5 Training Methods

Training methods were felt to be beyond the scope of this study. The physical demands of a rugby player are largely dependent on the position played. It is expected that due to these differences, the training programmes of the forward and back rows would differ, but it was noted that all players participate in similar training regimes. Gabbett (2000) found that the physiological and anthropometric characteristics of amateur Rugby Union players are poorly developed. His findings suggest that position specific training does not occur in amateur Rugby Union. The poor fitness of non elite players may be due to low playing intensity,

infrequent matches of short duration, and/or inappropriate training stimulus.

In future studies it is recommended that training methods in adolescents playing at this level be thoroughly investigated.

5.6 Commenting on Injury in Rugby Union

The physical demands on the rugby player due to the nature of the game dictates that injury incidents are high. Numerous factors may cause an increase in the incidence of injuries in both professional and amateur rugby players. The introduction of professionalism to Rugby Union (Garraway et al, 2000), increase in the level of rugby (Targett, 1998; Lee and Garraway, 1996); and playing summer league may increase injury incidence. Since the introduction of a Summer League, with players involved in back to back summer and winter rugby seasons, the change in ground conditions and playing season have contributed to injury incidence (Phillips et al, 1998).

In order to decrease the incidence of injuries, attention needs to be focused on specific aspects of the game i.e. the tackle, where many injuries occur. Garraway et al (2000) feel that the International Rugby Board should place a moratorium on the use of protective equipment in competitive matches until its contribution to player morbidity has been fully assessed. The frequency and severity of injuries can be decreased by adequate preseason training and conditioning, proper tackling and falling techniques, strengthening of the neck muscles and allowing only the experienced, fit athlete to play in the front row (Dietzen and Topping, 1999). It is essential that coaches should be experienced and attend clinics or courses on the medical emergencies and safety techniques of the game.

From clinical experience physiotherapists are aware that should an injury occur in any one part of the body, unless it is fully rehabilitated, and very often despite being fully rehabilitated this site is often vulnerable, thus causing further injury. It is for this reason that subjects with the occurrence of a previous injury at one body area and another injury occurring in the same area were eliminated from the study.

An in-depth analysis of injury occurrence is not part of this study but could be considered in a future study.

5.7 Conclusion

The discussion has highlighted that creatine supplementation was not associated with muscle injury. Poor dosage regimens were followed and subjects had little knowledge of correct dosage regimens and were thus taking it haphazardly. Incorrect dosage regimens made it difficult to assess the true association between injury and creatine supplementation. The presence of previous injuries due to numerous other factors leads to a smaller sample group. In future studies rugby players of a higher level should be used as their training is more rugby specific and their coaches are more advanced regarding specific strength and flexibility programmes. A summer sport is seldom played, but their seasons are longer and very often creatine is not the only ergogenic aid used.

Chapter 6

6.0 Conclusion

The main findings of this study were:

In this study, no evidence of an association between creatine supplementation and muscle injury, defined as cramping and muscle strains, was found.

The subjects in this study used creatine haphazardly and supplement recommendations were not followed. If the recommended dosage regimen is not adhered to, creatine is not effective as an ergogenic aid and thus will not enhance performance, at times resulting in adverse effects.

Chapter 7

7.0 References

1. Agre J C, 1985. Hamstring injuries: Proposed Etiological Factors. Prevention and Treatment. Sports Medicine 2: 21 – 23.

2. Anderson B, Burke E R, 1991. Scientfic, Medical and Practical aspects of Stretching. Clinical Sports Medicine 2: 21 – 23.

3. Astrand, Rodahl, 1970 Textbook of work physiology: Physiological Basis of Exercise, McGraw - Hill Inc, 1: 6

4. Balsom P D, Soderlund K, Sjodin B, Ekblom B, 1995. Skeletal muscle metabolism during short duration high intensity exercise: influence of creatine supplementation. Acta Physiolologica Scandinavica 154: 303 - 310.

5. Balsom P D, Soderlund K, Ekblom B, 1994. Creatine in humans with special reference to creatine supplementation. Sports Medicine 18(4): 268-280.

6. Balsom P D, Harridge S D R, Soderlund K, Sjodin B, Ekblom B, 1993. Creatine supplementation per se does not enhance exercise performance. Acta Physiolologica Scandinavica 149: 521 - 523.

7. Bermon S, Venebre P, Sachet C, Valour S, Dolisi C, 1998. Effects of creatine monohydrate ingestion in sedentary and weight-trained older adults. Acta Physiolologica Scandinavica 164:147-155.

8. Bogdanis G C, Nevill M E, Boobis L H, Lakomy H K, Nevill A M, 1995. Recovery of power output and muscle metabolites following 30s of maximal sprint cycling in man. Journal of Physiology 482: 467 - 480.

9. Booth F W, Weeden S H, Tseng B S, 1994. Effect of Aging on Human Skeletal Muscle and Motor Function. Medicine and Science in Sports 26(5): 556 – 560.

10. Bosco C, Tihanyi J, Pucspk J, Kovacs I, Gabossy A, Colli R, Puivirenti G, Tranquilli C, Foti C, Viru M, Viru A, 1997. Effect of oral creatine supplementation on jumping and running performance. Journal of Sports Medicine 18: 369 - 372.

11. Brannon T A, Adams G R, Conniff C L, Baldwin K M, 1997. Effects of creatine loading and training on running performance and biochemical properties on rat skeletal muscle. Medicine and Science in Sports and Exercise 29(4): 489-495.

12. Bullock-Saxton J E, Bullock M I, 1994. Repeatability of Muscle Length Measures around the Hip. Physiotherapy Canada 46(2): 105 – 109.

13. Cahill B R, Griffith E H, 1978. Effect of Preseason Conditioning on the Incidence and Severity of High School Football Knee Injuries. American Journal of Sports Medicine 6(4): 180 – 184.

14. Cameron D M, Bohannon R W, Owen S V, 1994. Influence of Hip Position on the measurements of the Straight Leg Raise Test. Journal of Orthopaedic and Sports Physical Therapy 19(3): 168 – 172.

15. Casey A, Constantin-Teodosiu D, Howell S, Hultman E, Greenhaff P L, 1996. Creatine ingestion favourably affects performance and muscle metabolism during maximal exercise in humans. American Journal of Physiology 271 (Endocrinology Metabolism 34): E 31-E37.

16. Chandler T J, Kibler B, Wooten B, 1990. Flexibility Comparisons of Junior Elite Tennis Players to other Athletes. American Journal of Sports Medicine 18(2): 136 – 136.

17. Collier P F, 1997 Colliers Dictionary, Halsey W D, P F Collier Inc, London and New York, A -K: 337-359.

18. Cooke W H, Grandjean P W, Bames W S, 1995. Effective of oral creatine supplementation on power output and fatigue during bicycle ergometry. Journal of Applied Physiology 78 (2): 670 - 673.

19. Cureton T K, 1941. Flexibility as an Aspect of Physical Fitness. Research Quarterly for Exercise and Sport 381 – 390.

20. Dietzen C J, Topping B R, 1999. Rugby football. Physical Medicine and Rehabilitation Clinics of North America 10(1): 159 – 175.

21. Ekblom B, 1996. Effects of creatine supplementation on performance. American Journal of Sports Medicine 24(6): S38 - S39.

22. Engelhardt M, Neuwmann G, Berbalk A, Renter I, 1998. Creatine supplementation in endurance sports. Medicine and Science in Sports and Exercise 30(7): 1123 - 1129.

23. Gabbett T J, 2000. Physiological and anthropometric characteristics of amateur rugby league players. British Journal of Sports Medicine 34(4): 303 – 307.

24. Gadjosik R L, 1991. Effects of Static Stretching on the Maximal Length on the Standing Position and Flexion Range of Motion of the Pelvic Angle, Lumbar Angle, and Thoracic Angle. Journal of Orthopaedic and Sports Physical Therapy 20(4): 213 – 219.

25. Garraway W M, Lee A J, Hutton S J, Russell E B, Macleod D A, 2000. Impact of professionalism on injuries in rugby union. British Journal of Sports Medicine 34(5): 348 – 351.

26. Garrett W E, 1996. Muscle Strain Injuries. American Journal of Sports Medicine 24(6): S9 –S13.

27. Gerrard D F, 1993. Overuse Injury and Growing Bones: the young athlete at risk. British Journal of Sports Medicine 27(1): 14 – 18.

28. Green A, Simpson E, Littlewood J, MacDonald I, Greenhaff P, 1996. Carbohydrate ingestion augments creatine retention during creatine feedings in humans. Acta Physiolologica Scandinavica 158: 195-202.

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

30. Greenhaff PL, 1997. The nutritional biochemistry of creatine. Journal of Nutritional Biochemistry 11: 610-618.

31. Greenhaff P L, 1995. Creatine and its application as an ergogenic aid. International Journal of Sports Nutrition 5: S100 - Sl10.

32. Greenwood M, Kreider R, Greenwood L D, Byars A, 2000a. Effects of Creatine Supplementation on the Incidence of Cramping/Injury during college Football three a day training. Medicine and Science in Sports and Medicine 32 (S61): S136.

33. Greenwood L D, Greenwood M, Kreider R, Byars A, 2000b. Effects of Creatine Supplementation on the Incidence of Cramping/Injury during collegiate fall baseball. Medicine and Science in Sports and Medicine 32 (S61): S136.

34. Grindstaff P, Kreider R, Bishop R, Wilson M, Wood L, Alexander C et al, 1997. Effects of creatine supplementation on repetitive sprint performance and body composition in competitive swimmers. International Journal of Sports Nutrition 7:330-346.

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

36. Herbert R, 1988. The Passive mechanical Properties of Muscle and their Adaptations to Altered Patterns of Use. Australian Journal of Physical Therapy 34(3): 141 – 149.

37. Hultman E, Sahlin K, 1980. Acid-base balance during exercise. Exercise and Sports Science Reviews 8: 41-128.

38. Hultman E, Soderlund K, Timmons J A, Cederblad G, Greenhaff P L, 1996. Muscle creatine loading in men. Journal of Applied Physiology 81(1): 232-237.

39. Hunt J, Kreider R, Melton C, Ransom J, Rasmussen C, Stroud T, Cantler E, Milnor P, 1999. Creatine does not increase incidence of cramping or injury during pre-season college football training II. Medicine and Science in Sports and Exercise 31(5): 121 – 127.

40. Jonhagen S, Nemeth G, Eriksson E, 1994. Hamstring Injuries in Sprinters – The Role of Concentric and Eccentric Muscle Strength and Flexibility. American Journal of Sports Medicine 22(2): 262 – 266.

41. Juhn M S, and Tamopolsky M, 1998. Potential side effects of oral creatine supplementation: A critical review. Clinical Journal of Sport Medicine 8: 298 - 304.

42. Kamber M, Koster M, Kreis R, Walker G, Boesch C, Hoppeler H, 1999. Creatine supplementation part 1: performance clinical chemistry and muscle volume. Medicine and Science in Sports and Exercise 31 (12): 1763 - 1769.

43. Kraemer W J, Volek J S, 1999. Creatine supplementation - Its Role in Human Performance. Clinics in Sports Medicine 18(3): 651 -666.

44. Kreider R B**,** 1998a. Creatine supplementation: analysis of ergogenic value, medical safety, and concerns. Internet Society for sport Science. http://www.css.edu/users/tboone2/asep/jan3.htm.

45. Kreider R B, 1998b. Creatine, the next ergogenic supplement? Internet Society for Sport Science, http://www.sportsci.org/traintech/creatine/rbk.html.

46. Kreider R B, Ferreira M, Wilson M, Grindstaff P, Plisk S, Reinardy J, Cantler E, Almanda A L, 1998b. Effect of creatine supplementation on body composition strength and sprint performance. Medicine and Science in Sports and Exercise 30(1): 73 - 82.

47. Kreider R, Rasmussen C, Ransom J, Almada A, 1998c. Effects of creatine supplementation during training on the incidence of muscle cramping, injuries and GI distress. Journal of Strength and Conditioning Research 12: 275.

48. Lee A J, Garraway W M, 1996. Epidemiological comparison of injuries in school and senior club rugby. British Journal of Sports Medicine 30(3): 213 – 217.

49. Lemon P, FACSM, Boska M, Bredle D, Rogers M, Ziegenfuss T, Newcomer B, 1995. Effect of oral creatine supplementation on energetics during repeated maximal muscle contraction. Medicine and Science in Sports and Exercise 27: S204.

50. Maffuli N, King J B, Helms P, 1994. Training in elite young athletes (the training of young athletes study (TOYA)): injuries, flexibility and isometric strength. British Journal of Sports medicine 28(2): 123 – 136.

51. McArdle W D, Katch F I, Katch V L, 1981 Exercise Physiology: Energy, Nutrition and Human Performance, Lea & Febiger, Philadelphia 22: 305-318.

52. Millar A P, 1976. An Early Stretching Routine for Calf Muscle Strains. Medicine and Science in Sports 8: 39 – 42.

53. Morris L S, Shultz R M, 1993. Medication compliance: The patient's perspective. Clinical Therapy 15(3): 593 – 606.

54. Mujika J, Chatard J, Lacoste L, Barale F, Geyssant A, 1996. Creatine supplementation does not improve sprint performance in competitive swimmers. Medicine and Science in Sports and Exercise 28(11): 1435 – 1441

55. Norris C M, 1993. Sports Injuries and management for Physiotherapists. 1st Edition. Oxford. Butterworth-Heinemann Ltd. 1993

56. Odland M L, MacDougall D J, Tarnopolsky M A, Elorriaga A, Borgmann A, 1997. Effect of oral creatine supplementation on muscle (PCr) and short-term maximum power output. Medicine and Science in Sports and Exercise 29(2): 216-219.

57. Phillips L H, Standen P J, Batt M E, 1998. Effects of seasonal change in rugby league on the incidence of injury. British Journal of Sports Medicine 32(2): 144 – 148.

58. Poortmans J R, Francaux M, 2000. Adverse effects of creatine supplementation: Fact or fiction? 30 (3): 155 – 170

59. Poortmans R J, Francaux M, 1999. Long-term oral creatine supplementation does not impair renal function in healthy athletes. Medicine and Science in Sports and Exercise 31(8): 1108-1110.

60. Ray T R, Eck J C, Covington L A, Murphy R B, Williams R, Knudtson J, 2001. Use of oral creatine as an ergogenic aid for increased sports performance: perceptions of adolescent athletes. South Medical Journal 94(6): 608 – 612.

61. Reilly T, Secher N, Snell P, Williams C, 1990. Physiology of Sports, E and F N Spon, London, New York, Tokyo, Melbourne and Madras 13: 411 – 418.

62. Rigby B J, 1964. The Effect of the Mechanical Extension upon the Thermal Stability of Collagen. Biochemica et Biophysica Acta 79: 634 – 636.

63. Robinson T, Sewell D, Hultmann E, GreenhaffP, 1999. Roll of submaximal exercise in promoting creatine and glycogen accumulation in human skeletal muscle. Journal of Applied Physiology 87(2): 598-604.

64. Rossiter H B, Cannell E R, Jakeman P M, 1996. The effect of oral creatine supplementation on the 1000-m performance of competitive rowers. Journal of Sports Sciences 14: 175-179.

65. Rupp S, Berninger K, Hopf T, 1995. Shoulder problems in high level swimmers – impingement, anterior instability, muscular imbalance? International Journal of Sports Medicine 16: 557 – 562.

66. Sapega A A, Quendenfeld T C, Moyer R A, 1981. Biophysical Factors in Range-of-Motion exercise. Physician and Sports Medicine 9(12): 57 – 65.

67. Schilling B K, Stone M H, Utter A, Kearney J T, Johnson M, Coglianse R, Smith L, O'Bryant H S, Fry A C, Startks M, Keith R, Stone M E, 2001. Creatine supplementation and health variables: a retrospective study. Medicine and Science in Sports and Exercise 33(2): 183 – 188.

68. Sewell D A, Robinson T M, Casey A, GreenhaffP L, 1998. The effect of acute dietary creatine supplementation upon indices of renal, hepatic and haematological function in human subjects. Proceedings of the Nutrition Society. 59: 17A.

69. Smith J, Dahm D L, 2000. Creatine use among a select population of high school athletes. Mayo Clinic Proceedings 75(12): 1257 – 1263.

70. Smith S A, Montain S J, Matott R P, Zientara G P, Jolesz F A, Fielding R A, 1999. Effects of creatine supplementation on the energy cost of muscle contraction: 3 IP - MRS Study. Journal of Applied Physiology 87(1): 116-123.

71. Smith S A, Montain S J, Matott R P, Zientara G P, Jolesz F A, Fielding R A, 1998. Creatine Supplementation and age influence muscle metabolism during exercise. Journal of Applied Physiology 85: 1349 – 1356.

72. Snow R, McKenna M, Selig S, Kemp J, Stathis C, Zhao S, 1998. Effect of creatine supplementation on sprint exercise performance and muscle metabolism. Journal of Applied Physiology 84(5): 1667-1673.

73. Soderlund K, Balsom P D, Ekblom B, 1994. Creatine supplementation and high intensity exercise: Influence on performance and muscle metabolism. Clinical Science 87(Suppl.): 120-121.

74. Starring D T, Gossman M R, Nicholson G G, 1988. Comparison of Cyclic and Sustained Passive Stretching Using a Mechanical Device to Increase resting Length of Hamstring Muscles. Physical Therapy 68(3): 314 – 320.

75. Stout J R, Echerson J, Noonan D, Moore G, Cullen D, 1999. Effects of creatine supplementation on exercise performance and fat free weight in football players during training. Nutrition Research 19: 217 – 225.

76. Stout J, Eckerson J, Noonan D, Moore G, Cullen D, 1997. The effects of a supplement designed to augment creatine uptake on exercise performance and fat-free mass in football players. Medicine and Science in Sports and Exercise 29: S 251.

77. Stroud M A, Holliman D, Bell D, Green A L, Macdonald I, GreenhaffP L, 1994. Effect of oral creatine supplementation on respiratory gas exchange and blood lactate accumulation during steady state incremental treadmill exercise and recovery in man. Clinical Science 87: 707 - 710.

78. Targett S G, 1998. Injuries in professional rugby union. Clinical Journal of Sports Medicine 8(4): 280 – 285.

79. Taylor D C, Dalton J D, Seabar A V, 1990. Viscoelastic Properties of Muscle Tendon Units. American Journal of Sports Medicine 18(3): 300 – 309.

80. Terrilion K, Kolkhorst F, Dolgener F, Joslyn S, 1997. The effects of creatine supplementation on two 700-m maximal running bouts. International Journal of Sport Nutrition 7: 138-143.

81. Toppenberg R, Bullock M, 1990. Normal Lumbo-Pelvic Muscle Length and their Interrelationships in Adolescent Females. Australian Journal of Physiotherapy 36(2): 105 – 109.

82. Travell J G, Simons D G, 1983. Myofascial Pain and Dysfunction – The Trigger Point Manual. 1st Ed. Baltimore. Williams and Wilkins.

83. Vanderberghe K, Van Hecke P, Van Leemputte M, Vanstapel F, Hespel P, 1999. Phosphocreatine resynthesis is not affected by creatine loading. Medicine and Science in Sports and Exercise 31(2): 236 - 242.

84. Vandenberghe K, Goris M, Van Hecke P, Van Leemputtee M, Vangerven L, Hespel P, 1997. Long-term creatine intake is beneficial to muscle performance during resistance training. Journal of Applied Physiology 83(6): 2055-2063

85. Volek J, Duncan N, Mazzetti S, Staron R, Putukian M, Gomez A, Pearson D, Fink W, Kraemer W J, 1999. Performance and muscle fibre adaptations to creatine supplementation and heavy resistance training. Medicine and Science in Sports and Exercise 31(8): 1147-1156.

86. Volek J, Boetes M, Bush J, Putukian M, Sebastianelli W, Kraemer W, 1997. Response of testosterone and cortisol concentrations to high intensity resistance exercise following creatine supplementation. Journal of Strength and Conditioning Research 11: 182 -187.

87. Volek J S, Kraemer W J, 1996. Creatine Supplementation: Its effects on human muscular performance and body composition. Journal of Strength and Conditioning Research 10: 200-210.

88. Walker J, 1979. Biosynthesis, regulation, and function. Advances in Enzymology 50: 177-242.

89. Weber S, Kraus H, 1949. Passive and Active Stretching of Muscles. Physical Therapy Review 29(9): 407 – 411.

90. Wilson G J, Elliott B C, Wood G A, 1992. Stretch Shorten Cycle Performance Enhancement through Flexibility Training. American Journal of Sports Medicine 24(1): 116 – 123.

91. Worrell T W, Perrin D H, Gansneder B M, 1991. Comparison of Isokinetic Strength and Flexibility measures between Hamstring Injured and Noninjured Athletes. Journal of Orthopaedic and Sports Physical Therapy 13(3): 118 – 125.

92. Young W B, Behm D G, 2003. Effects of running, static stretching and practice jumps on explosive force production in jumping performance. Journal of Sports Medicine and Physical Fitness. 43(1): 21-27. .

Clearance Certificate

Serial No: ……

QUESTIONNAIRE

Position:

Full Back
Centre
Wing
Fly Half
Scrum Half
Eighth Man
Lock
Flank
Hooker
Prop

Age at which you started playing first team rugby: …….

Age this season: ……...

Highest level at which you are currently playing:

First Team
Provincial
National

Number of seasons played at this level: ……...

TRAINING PROGRAMME

1. DURATION OF TRAINING:

Months per year: ……..

(Sept - Nov) Days per week: …….

Hours per day: ……..

(Dec – Feb) Days per week: ……

Hours per day: …….

(March – May) Days per week: ……

Hours per day: …….

(June – Aug) Days per week: ……

Hours per day: …….

2. DESCRIPTION OF TYPICAL TRAINING SESSION:

Estimate the proportion of **training per week** spent on:

Skills Training per week: ¼ ½ ¾ All

Fitness Training per week: ¼ ½ ¾ All

Training for:	Sprint	Less than 50m	**Yes**	**No**
		50 – 100m	Yes	No
		150 – 200m	Yes	No
		200 – 400m	Yes	No
	Middle distance	400 – 800m	Yes	No
		800 – 1500m	Yes	No
		1500 – 3000m	Yes	No
	Long distance	more than 3000m	Yes	No

Non running fitness training e.g. skipping or stair climbing: Yes No

Sets per week …….

Repetitions per week ……..

3. OTHER TRAINING METHODS:

a) <u>Gym</u>

Weight Training: | **Yes** | | **No** |

Age at commencement: ……

<u>Frequency of training</u>: Months per year: ……..

(Sept - Nov) Days per week: ……..

Hours per day: ……

(Dec – Feb) Days per week: ……

Hours per day: ……

(March – May) Days per week: ……

Hours per day: ……

(June – Aug) Days per week: ……

Hours per day: ……

<u>Name of exercises</u>:

Shoulder exercises: | Yes | | No |

Number of exercises: …….

Number of sets (total): ……

Number of repetitions (total): ……

Upper arm exercises: Yes No

Number of exercises: …….

Number of sets (total): ……

Number of repetitions (total): ……

Forearm exercises: Yes No

Number of exercises: …….

Number of sets (total): ……

Number of repetitions (total): ……

Chest exercises: Yes No

Number of exercises: …….

Number of sets (total): ……

Number of repetitions (total): ……

Back exercises: Yes No

Number of exercises: …….

Number of sets (total): ……

Number of repetitions (total): ……

Abdominal exercises: Yes No

Number of exercises: …….

Number of sets (total): ……

Number of repetitions (total): ……

Groin and Buttock: Yes No

Number of exercises: …….

Number of sets (total): ……

Number of repetitions (total): ……

Hamstring exercises: Yes No

Number of exercises: …….

Number of sets (total): ……

Number of repetitions (total): ……

Quadriceps exercises: Yes No

Number of exercises: …….

Number of sets (total): ……

Number of repetitions (total): ……

Calf exercises: Yes No

Number of exercises: …….

Number of sets (total): ……

Number of repetitions (total): ……

Shin exercises: Yes No

Number of exercises: …….

Number of sets (total): ……

Number of repetitions (total): ……

Other:

Name of exercise: …….

Name of exercise: …….

Other gym apparatus:

Name of apparatus: ……

…….

Age at commencement: …….

Frequency of training: Months per year: ……..

(Sept - Nov) Days per week: ……..

Hours per day: ……

(Dec – Feb) Days per week: ……

Hours per day: ……

(March – May) Days per week: ……

Hours per day: ……

(June – Aug) Days per week: ……

Hours per day: ……

b) Stretching:

Do you stretch: Yes No

If not, why not: ……

……

When do you stretch:

Before training: Yes No

Before a game: Yes No

After training: Yes No

After a game: Yes No

Before gym: Yes No

After gym: Yes No

Method of stretching:

Prolonged (holding): Yes No

Ballistic (bouncing): Yes No

Assisted (buddy): Yes No

Description of stretches:

Neck: Yes No

Number of stretches per area (total): ……

How many of each stretch: ……..

Duration of stretch: 0-10s ☐

10-30s ☐

30-60s ☐

Other: ☐

Shoulder: ☐ Yes ☐ No

Number of stretches per area (total): ……

How many of each stretch: ……..

Duration of stretch: 0-10s ☐

10-30s ☐

30-60s ☐

Other: ☐

Upper arm: ☐ Yes ☐ No

Number of stretches per area (total): ……

How many of each stretch: ……..

Duration of stretch: 0-10s ☐

10-30s ☐

30-60s ☐

Other: ☐

Forearm: ☐ Yes ☐ No

Number of stretches per area (total): ……

How many of each stretch: ……..

Duration of stretch: 0-10s ☐

10-30s ☐

30-60s ☐

Other: ☐

Back: ☐ Yes ☐ No

Number of stretches per area (total): ……

How many of each stretch: ……..

Duration of stretch: 0-10s ☐

10-30s ☐

30-60s ☐

Other: ☐

Groin and buttocks: ☐ Yes ☐ No

Number of stretches per area (total): ……

How many of each stretch: ……..

Duration of stretch: 0-10s ☐

10-30s ☐

30-60s ☐

Other: ☐

Hamstring muscle: ☐ Yes ☐ No

Number of stretches per area (total): ……

How many of each stretch: ……..

Duration of stretch:

0-10s	☐
10-30s	☐
30-60s	☐
Other:	☐

Quadriceps muscle: ☐ Yes ☐ No

Number of stretches per area (total): ……

How many of each stretch: ……..

Duration of stretch:

0-10s	☐
10-30s	☐
30-60s	☐
Other:	☐

Calf muscle: ☐ Yes ☐ No

Number of stretches per area (total): ……

How many of each stretch: ……..

Duration of stretch:

0-10s	☐
10-30s	☐
30-60s	☐
Other:	☐

Shin muscle: Yes No

Number of stretches per area (total): ……

How many of each stretch: ……..

Duration of stretch: 0-10s ☐

10-30s ☐

30-60s ☐

Other: ☐

c) <u>Other sports played</u>:

Name of sport	Age at commencement	Duration of season (months)	Level of participation
1.			
2.			
3.			
4.			
5.			

<u>INJURIES</u>:

Have you ever, before playing at 1st team level, had an injury to your:

	Yes	**No**
Neck:	Yes	No
Shoulder		
Elbow	Yes	No
Hand	Yes	No
Chest	Yes	No
Abdominals	Yes	No
Back	Yes	No
Hip	Yes	No
Knee	Yes	No
Foot	Yes	No
Other	Yes	No

If other, please state: …….

If yes, what injury occurred to this area e.g. dislocation, fracture, etc ……..

……

Have you ever, at 1st team level, had an injury to your:

	Yes	**No**
Neck:	Yes	No
Shoulder		
Elbow	Yes	No
Hand	Yes	No
Chest	Yes	No
Abdominals	Yes	No
Back	Yes	No
Hip	Yes	No
Knee	Yes	No
Foot	Yes	No
Other	Yes	No

If other, please state: …….

If yes, what injury occurred to this area e.g. dislocation, fracture, etc ……..

……

Have you ever strained or "pulled" your;

Shoulder	Yes	No
Upper arm	Yes	No
Forearm	Yes	No
Chest	Yes	No
Abdominals	Yes	No
Back	Yes	No
Groin	Yes	No
Hamstring	Yes	No
Quadricep	Yes	No
Calf	Yes	No
Other	Yes	No

If other, please state: ……..

If yes, how often have these injuries occurred:

1 per season	Yes	No
2 per season	Yes	No
Other	Yes	No

If other, please state: …….

When did your injury occur:	Preseason	Summer	Yes	No
		Pre-Match	Yes	No
		In season	Yes	No
		Post season	Yes	No

What caused the injury:	Over	Training	Yes	No
	Under	Training	Yes	No
	Over	Stretching	Yes	No
	Under	Stretching	Yes	No
		Bad tackle	Yes	No
		Other	Yes	No

If other, please state: …….

Do you ever experience muscle cramping:	Yes	No

If yes, in which muscle group:	Shoulder	Yes	No
	Upper arm	Yes	No
	Forearm	Yes	No
	Chest	Yes	No
	Abdominals	Yes	No
	Back	Yes	No
	Groin	Yes	No
	Buttocks	Yes	No
	Hamstring	Yes	No
	Quadricep	Yes	No
	Calf	Yes	No
	Other	Yes	No

If other, please state: …….

How often do you experience muscle cramping:		1/practice	Yes	No
	More than	1/practice	Yes	No
		1/game	Yes	No
	More than	1/game	Yes	No
	When not	playing	Yes	No

What is the cause of your muscle cramping: ……

SUPPLEMENTATION

Have you ever or are you currently taking a performance enhancing substance :

Steroids	Yes	No
Creatine	Yes	No
Other	Yes	No

If creatine, please continue:

How long have you been taking creatine:	Less than	1 season	Yes	No
		1 season	Yes	No
		2 seasons	Yes	No
		3 seasons	Yes	No
	More than	3 seasons	Yes	No

Are you currently taking creatine:	Yes	No

At what intervals e.g. 3 months (on) 4 months (off)		On	Off
Years		
Months		
Days		

What dosage of creatine:		G/day	Days
Loading	Dose		
Maintenance	Dose		

How many times a day e.g. 5grams 4 times a day	1
	2
	3
	4

What form of creatine do you use:	Powder	Yes	No
	Gum	Yes	No
	Bar	Yes	No
	Other	Yes	No

With what do you drink or eat it:	Apple juice	Yes	No
	Grape juice	Yes	No
	Water	Yes	No
	Other	Yes	No

Please continue on the following page

If you have had an injury while taking creatine, please continue:

In which phase of supplementation did the injury occur:			
	Loading	Yes	No
	Maintenance	Yes	No
	Both	Yes	No
	Interval	Yes	No
	Other	Yes	No

If other, please state: ……..

What was the dosage of creatine at the time of injury:

		G/day	Days
Loading	Dose		
Maintenance	Dose		

How often has the injury occurred since you have been taking creatine:

	1/season	Yes	No
	2/season	Yes	No
More than	2/season	Yes	No
	Other	Yes	No

If other please state: ……..

How long did the injury keep you from playing: …….

Do you think the cause of the injury could be creatine supplementation:

Yes	No

Thank you for taking part in this questionnaire.