

# CREATINE

cutting through  
the myths

A SPECIAL REPORT FROM



**PEAK**  
**PERFORMANCE**

The research newsletter on  
stamina, strength and fitness



# CREATINE

## cutting through the myths

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**Publisher**

Jonathan A. Pye

**Editor**

Bob Troop

**Production Manager**

Frances Peel Yates

**Designer**

Charlie Thomas

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# From the publisher

**C**reatine is a performance-enhancing supplement that has been in the news for a decade or more. Famous athletes who have used it include Linford Christie and Sally Gunnell. The general consensus of early scientific opinion was that it boosted short-term performance, but more recently researchers have discovered that it aids endurance athletes as well and even helps produce a final kick at the end of a race – both these benefits are discussed in this report. Inevitably, of course, there have been detractors: creatine makes you gain weight, they say, or it dilutes potassium and calcium in the muscles, or it causes cramping.

The purpose of this special report is to sort out the facts from the fiction about creatine. It reviews the scientific research as well as the evidence of practical athletes to tell you what creatine actually does (and doesn't) do, who should try it, how much to take, and how to gain the maximum performance boost. We have assembled quite a battery of experts on your behalf, including one of the world's leading authorities on the subject. After reading this report, you should have a clear idea of the benefits of creatine to your own performance.

Finally, as a bonus, we offer at the end of this report an analysis of three other performance-boosting substances: HMB, caffeine and sodium bicarbonate.

I hope you enjoy this report and find it useful.

A handwritten signature in black ink, reading "J. A. Pye." with a period at the end.

Jonathan A Pye  
Publisher



## **BACKGROUND (1)**

# **Before we get to the nitty-gritty, here's a short history of creatine as an ergogenic supplement**

Creatine, or methyl guanidine-acetic acid, was first discovered in 1832 by a French scientist called Chevreul. However, it was Lieberg's research in 1847, with wild foxes, which confirmed that creatine was a regular constituent of the body. Further research by Heintz and Pettenkofer showed that urine contained a substance known as creatinine, and that it was produced as the result of the metabolic breakdown of creatine stored in muscles. The next step in the unfolding story of creatine came in the early part of the last century. Folin (1912) and Denis (1914) conducted a number of experiments with supplemental creatine and showed that, when extra creatine was ingested, not all of it could be subsequently recovered from the urine, strongly suggesting that supplemental creatine could increase the creatine content of muscle cells. In particular, work with cats showed that up to 70% more creatine could be observed in muscle tissue after creatine ingestion. Around 10 years later, Hahn and Meyer carried out research that estimated the total creatine content of the typical 70kg (11 stone) male to be around 140 grams, and a few years later in 1927, Fiske and Subbarow discovered the existence in muscle tissue of creatine phosphate – creatine and phosphate molecules chemically bound together.

### **Enter Professor Hultman**

During the next 40 years, there was only limited interest in creatine, despite some (unconfirmed) reports that athletes

‘As early as the 1920s and 1930s, people used creatine precursors to improve exercise performance’

from the former Soviet Union had been experimenting with it in training. However, that changed when research into the biochemistry and physiology of creatine began to demonstrate its role in recycling adenosine triphosphate (ATP) within working muscles. Much of this work was carried in Sweden during the 1970s and 1980s by Professor Hultman at the Karolinska Institute, who determined that phospho-creatine (the phosphorylated form of creatine) was strongly related to the development of fatigue during very intense muscle contraction. He wondered if, by raising the muscle creatine stores, it would be possible to improve muscle function and energetics, as, in a similar way, endurance performance could be boosted by increasing the amount of glycogen stored in muscles (research incidentally also pioneered by Hultman). By the end of the late 1980s, it was clear that Hultman's research (and that of others) was providing some positive answers to the questions he had posed and before long the athletic community got wind of the subject.

Although there are scientific papers showing that people as early as the 1920s and 1930s used creatine precursors to try to improve exercise performance, the first well-documented reports of positive experiments using creatine supplements began at around the 1992 Olympics in Barcelona. British athletes who were taking creatine supplements as a result of research in Sweden and the UK were having good results. In August 1992, *The Times* reported that Linford Christie, the 100-metre gold medallist, had trained with creatine before the Barcelona Olympics, while *Bodybuilding Monthly* reported that Sally Gunnell, the 400-metre gold medallist, had also trained with creatine. With further positive research findings emerging from the scientific community, the use of creatine during the past 10 years has now become commonplace, with creatine supplements widely available at a fraction of the original cost. Creatine is one sports supplement that is here to stay!

**Andrew Hamilton**

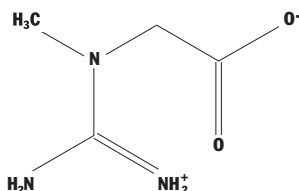




## BACKGROUND (2)

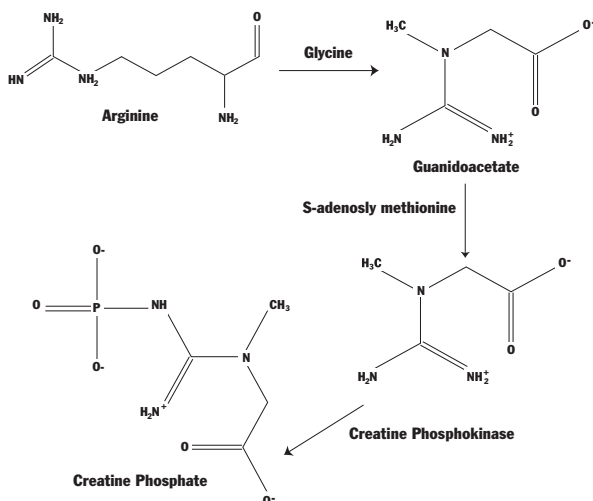
# OK, so exactly what is creatine, and how does it work in the body?

Creatine, or, as we've said, methyl guanidine-acetic acid (to use its chemical nomenclature) is a naturally occurring bio-molecule whose structure is shown below.



**Creatine**

Although creatine is found in both meat and fish, it can also be synthesised in the human body in a two-stage process from the amino acids arginine, glycine, and methionine:



Research has now established that the body of a typical 11-stone adult contains around 120-140 grams of creatine, most of which is stored in the tissues of skeletal muscle. Around two-thirds of this stored creatine is in the phosphorylated, energy-rich or 'active' form known as phospho-creatine or creatine phosphate, with the remaining third as creatine. The turnover of creatine in the body is relatively small; typically only around 2 grams a day from the 'creatine pool' are lost in the urine. This lost creatine can be replenished both by eating creatine-rich foods and by synthesis within the body. The key to understanding creatine's importance in boosting sports performance lies in its pivotal role in the energetics of muscle contraction during high-intensity exercise.

*Although the high-energy content of ATP is used directly to make muscles contract, it is not really used as an energy storage molecule in the muscles* »

### **How exactly creatine works**

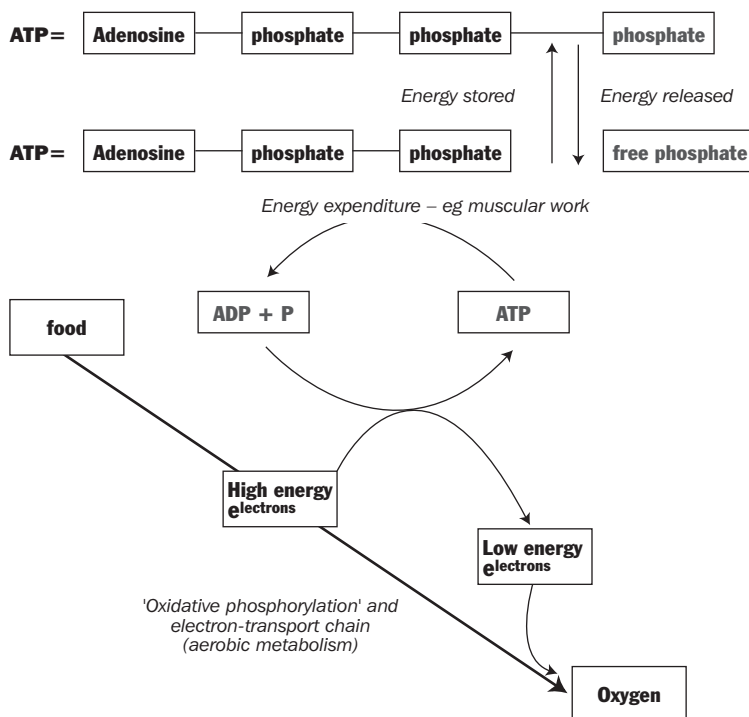
To understand creatine's role as a high-energy source for intense muscular work, you need to know a little bit about the role of ATP in the body (see Understanding ATP, right). Although the high-energy content of ATP is used directly to make muscles contract, it is not really used as an energy storage molecule in the muscles. The reasons for this are to do with the need to maintain a continual and sufficient supply of ATP to working muscles, under a wide range of energy demands. Instead, the ATP required is synthesised according to need. The bulk of the ATP synthesised in the body comes courtesy of the aerobic system. In simple terms, this system passes high-energy electrons from the food we eat to oxygen, with the energy liberated used to create ATP. During exercise, ATP is synthesised in the furnaces of your muscles which are known as mitochondria, then is shuttled over to the contracting myosin fibres, where its high-energy phosphate-phosphate bond is broken, and the energy used to contract muscle fibres. However, while this system works well at low-to-moderate energy requirements, there's a limit to how much oxygen can be transported to the mitochondria in your working muscles (ie your  $\text{VO}_{2\text{max}}$ ) to produce ATP by this route.



## UNDERSTANDING ATP

ATP (adenosine triphosphate) is a very special molecule, being quite literally the 'energy currency of the body'. This is because of its chemical structure, which enables it to supply immediate energy to drive all energy-requiring chemical reactions in the body, including those required to make muscles contract. ATP consists of a molecule of adenosine, on to which are linked three phosphate groups. ATP's special energy-donating ability lies in its phosphate bonds, specifically, the bond between the second and third phosphate groups. This bond contains chemical energy, which can easily be unlocked when the bond is broken and, importantly, readily stored again when the bond is reformed. When the high-energy bond is broken to release energy to do work, the Adenosine triphosphate breaks down into adenosine diphosphate (ADP) and a free phosphate group is released. When ATP is resynthesised, free phosphate is bonded back onto ADP, during which energy is once more locked up into the bond.

Ultimately, all the energy required to synthesise high-energy ATP from ADP and phosphate comes from high-energy electrons from chemical bonds in the food we eat. These electrons pass through metabolic systems (known as oxidative phosphorylation and the electron-transport chain), which harvest their energy by converting ADP back to high-energy ATP. Once their energy has been harvested, these electrons are passed on to oxygen. It's a bit like a waterfall, turning an ATP-producing mill. Under normal aerobic conditions, ATP can be regenerated rapidly enough from ADP and high-energy electrons flowing down the chain, to meet energy demands. When energy demands are very high and can't be met aerobically, ATP can be temporarily generated from other pathways.



## What happens when there's no more oxygen?

At higher exercise intensities, the aerobic production of ATP simply can't keep up demand. For example, if your muscles had to rely solely on aerobically produced ATP, stores would be almost entirely depleted of ATP within just one-to-two seconds of maximal exercise. During intense or very intense muscular contractions, the body has to use back-up systems to generate the extra ATP required. These are the glycolysis and the phospho-creatine systems. Glycolysis is a way of partially extracting the energy from the electrons in the chemical bonds of food (stored as muscle glycogen), without the presence of oxygen, and can help to regenerate ATP from ADP and phosphate quite rapidly. However, this pathway produces fatigue-inducing lactic acid as a by-product and takes a few

seconds to become fully active.

The phospho-creatine system, on the other hand, can produce very large amounts of ATP almost instantly by donating the high-energy phosphate bond present in creatine phosphate on to ADP, which regenerates ATP. The phospho-creatine system therefore forms an 'ATP buffer', keeping the concentration of ATP next to the contracting myosin fibres high and allowing around 10 seconds of maximal work to be produced before fatigue sets in. Although we said that ATP is shuttled over from the mitochondria to the contracting myosin fibres, this is not strictly true – much of this high-energy phosphate shuttling is done by creatine phosphate, whose second major role is to act as a spatial buffer. The concentration of creatine in muscle tissue is around five times higher than ATP. This actually makes creatine better at shuttling energy in the form of high-energy creatine phosphate across the cytosol from the mitochondria where ATP is being produced to the myofilaments where the contractions are occurring.

### **To sum up**

During intense exercise, ATP is rapidly depleted to ADP, because aerobic regeneration of ATP can't keep up with demand and there is little ATP storage capacity within muscle tissue. However, creatine phosphate is present in much higher concentrations and able to form an emergency energy reservoir by donating its high-energy phosphate to ADP, which regenerates ATP, prolonging the duration of the high-intensity exercise.

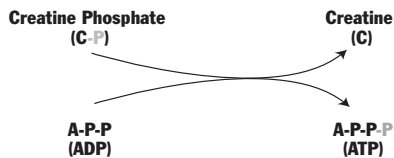
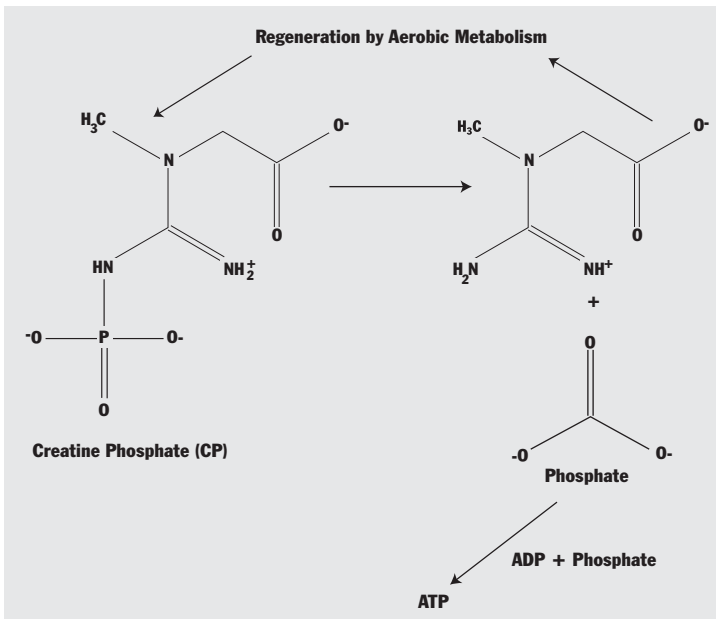
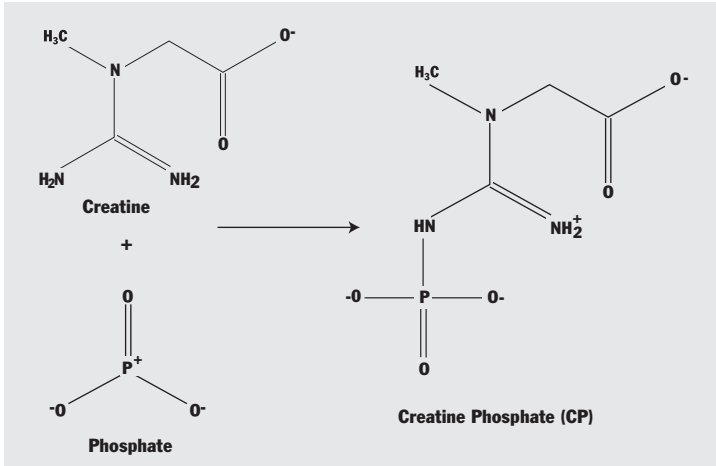
**Andrew Hamilton**

## **GENERATING ATP WITHOUT OXYGEN**

As long as there's enough oxygen present to regenerate ATP from ADP, all the energy required can be met by the aerobic system of electrons tumbling down the 'energy waterfall'. However, when energy demands are very high, for example

during very intense exercise, this system cannot regenerate ATP rapidly enough to sustain the intensity. Despite being a universal 'energy currency', very little energy in the body is stored directly as ATP, the body preferring to make it *in situ*. Because your muscles only store enough ATP for about one-to-two seconds of maximal contraction, other temporary pathways of resynthesising ATP from ADP and phosphate more rapidly need to spring into action. Glycolysis produces ATP by harvesting energy from electrons in the bonds of carbohydrates, in the foods we eat, without passing them on to oxygen. However, this is at a cost of the build up of fatigue-inducing lactic acid. Of more interest here, though, is the 'phospho-creatine' system.

Creatine phosphate consists of a creatine molecule, bonded to a high-energy phosphate. When the levels of ATP in the muscles fall, this high-energy phosphate can be donated to ADP (via the enzyme creatine kinase) to instantly regenerate ATP, without relying on the aerobic or glycolytic pathways. Once the creatine phosphate has donated its high-energy phosphate, the creatine produced can be recycled back to creatine phosphate. This system of phosphate donation from creatine phosphate to ATP and creatine recycling is known as the 'creatine phosphate shuttle' and is a very effective way of moving high volumes of high-energy phosphate from the mitochondria to the myosin (molecules in the muscle that actually shorten during contraction), where it is used in the form of ATP. You can think of the phospho-creatine system as an emergency reservoir of high-energy phosphate, ready to regenerate ATP during very high intensity work. The advantage of the phospho-creatine system over glycolysis is that it can regenerate ATP from ADP virtually instantly, whereas the glycolysis pathway is slower to become fully active. And although the creatine phosphate is exhausted after around 10 seconds of intense work, about 50% is regenerated within 30 seconds during the recovery period and within two to three minutes, it is almost 100% replenished.





## Why does creatine supplementation work when so many other sports supplements don't?

Lies, damn lies and supplements. The history of sporting endeavour is littered with ergogenic supplements that promise the earth yet deliver nothing more than a placebo effect. But creatine is different. Numerous double-blind scientific studies have shown that supplementing creatine produces a significant increase in maximal- or high-intensity exercise performance. The first-ever published study into oral creatine supplementation (Greenhaff *et al*, 1993) confirmed that creatine boosted performance in isokinetic leg extension exercises by up to 6%. Further studies subsequently confirmed the benefits of ingesting extra creatine in high-intensity running, swimming and cycling. Oral creatine supplementation boosts maximal exercise performance because, unlike so many other potentially ergogenic nutrients, ingesting extra creatine orally actually produces a measurable increase in both its concentration and subsequent activity within the body.

Although there are wide variations between individuals, the typical creatine content of dry muscle mass is around 110-140 millimoles per kg. However, given higher doses of creatine, most people can absorb and store more. In 1992, Harris *et al* conclusively showed that ingesting 5 grams of creatine four times a day for several consecutive days, increased muscle concentrations of creatine by around 25 millimoles per kg, of which 30% of the extra was present as the energy-donating creatine phosphate. This ability of the muscles to soak up extra creatine has since been confirmed by other studies. In plain

English, most of us have muscles that are not fully saturated with creatine and taking extra creatine can produce saturation.

But more creatine by itself isn't enough. Can that extra muscle creatine get to work and deliver more phosphate to help regenerate ATP during maximal exercise? The answer is yes. Although there appears to be a wide variation in both the initial concentration of muscle creatine and the ability to "soak up" extra creatine among individuals, Casey *et al* (1996) demonstrated not only that oral creatine boosted maximal performance, but that the greater the increase in creatine uptake, the greater the performance increase.

“Not only does creatine enable you to sustain a high-intensity burst for longer, it speeds recovery”

### **How does it work?**

Remembering that the role of the phospho-creatine system is to donate high-energy phosphate from creatine phosphate to ADP to regenerate ATP, it's easy to understand that if there is a higher concentration of creatine and creatine phosphate in the muscles, there will be more high-energy phosphate available to regenerate ATP from ADP. This explains why athletes supplementing creatine report being able to sustain high-intensity bursts of exercise for longer before fatigue sets in.

However, Greenhaff *et al* (1994) also showed that higher concentrations of muscle creatine helped to hasten the resynthesis of creatine phosphate from creatine and phosphate during the recovery period between bouts of exercise. In other words, not only does creatine enable you to sustain a high-intensity burst for longer, it speeds recovery, enabling you to repeat those bursts sooner. This second effect is intriguing as the production of creatine phosphate from free creatine is linked to the oxidative production of ATP in mitochondria. This implies that extra free-muscle creatine may actually generate an increase in mitochondrial respiration and ATP synthesis, which could have implications for creatine use in endurance athletes (see later in this special report). This hypothesis is supported by *in vitro* research carried out in 1966 (Bessmen *et al*), which showed that creatine increased the respiration rate



in skeletal muscle mitochondria, while similar results were obtained with cardiac muscle fibres (Field *et al*, 1994). New research carried out last year with human quadriceps muscle fibre (Walsh *et al*, *J Physiol* 2001 Dec 15; 537 (Pt 3): 971-8) seems to confirm that increased muscle creatine levels do indeed stimulate mitochondrial ADP-stimulated respiration.

### **Creatine as a lactate buffer?**

During maximal exercise, both the phospho-creatine and the glycolytic energy systems kick in to regenerate ATP. But when glycolysis occurs, lactate is produced and intramuscular acidity increases, causing fatigue. Creatine phosphate has a chemical structure that lends itself to buffering, or soaking up any increase in acidity, and this buffering ability is often cited as another reason for improved maximal exercise performance when supplementing creatine.

However, the evidence for this buffering mechanism is rather unconvincing. In 1986, Katz *et al* showed that fatigue during short-term, exhaustive exercise was related much more closely to a low phospho-creatine concentration than to a high lactate concentration, which suggests that the reduction of available phosphate from creatine phosphate is what causes fatigue, rather than the accumulation of lactate. There have been similar findings in other studies with repeated bouts of high-intensity exercise, where no correlation was found between the decline in work production during each bout of exercise and the muscle lactate concentration. In contrast, studies show that fatigue is positively correlated with both the concentrations and the resynthesis rate of creatine phosphate (Harris *et al*, 1974; Sahlin, 1989; Bogdanis *et al*, 1995).

### **Cell volumising and glycogen resynthesis**

When researchers studied the effects of oral creatine loading (ie ingesting enough creatine to saturate the muscles over a period of a few days), they discovered that the urinary volume decreased and this decrease exactly paralleled the associated increase in body mass, strongly suggesting that the

supplemental creatine was stimulating muscle cells to soak up water and become 'volumised' or hydrated (Hultman *et al*, 1996). This finding stimulated a great deal of speculation, because we know that a similar process happens during the process of carbohydrate loading. In addition, both animal and muscle cell preparation studies have been carried out which show that an increase in muscle cell volume can stimulate carbohydrate synthesis. If the same process occurs in humans, creatine could help athletes not only to perform better during single and repeated bouts of intense exercise, but also by playing a role in helping the resynthesis of muscle glycogen in between training sessions. However, while there is some evidence for this effect from preliminary animal studies, the scientific jury remains out until more research into this aspect of creatine supplementation has been completed.

**Andrew Hamilton**





# Factors modifying creatine accumulation in human skeletal muscle

The ingestion of 5g of creatine (Cr) in solution will raise the plasma Cr concentration from 40  $\mu\text{mol/l}$  to 600-800  $\mu\text{mol/l}$  within one hour and plasma levels then decrease to close to basal over the subsequent five hours (Harris *et al*, 1992, Green *et al*, 1996a). Repeating this procedure on four evenly spaced occasions each day for five days can increase the muscle total Cr (TCr) store by up to 40%. This increase is comprised of changes in both free Cr and phospho-creatine (PCr), with the magnitude of increase in the former being the largest (Harris *et al*, 1992, Greenhaff *et al*, 1994). The variation between individuals in the magnitude of muscle TCr increase is marked, with the extent of uptake being inversely related to the initial muscle TCr content (Harris *et al*, 1992, Greenhaff *et al*, 1994). The reasons for the large variation between subjects in the magnitude of Cr accumulation during supplementation are unknown and require further investigation.

The majority of muscle Cr accumulation occurs within the initial two days of loading and muscle Cr accumulation is saturated following five days of supplementation with four doses of 5g (Harris *et al*, 1992, Hultman *et al*, 1996). If Cr ingestion is stopped following loading, muscle Cr stores decline gradually and basal levels are reached after about four weeks (Febbraio *et al*, 1995, Hultman *et al*, 1996). Ingesting Cr at a rate of 3g per day will increase muscle Cr content but the time-course of change is slower, ie it takes 30 days to reach muscle TCr values similar to those observed after five days of 20g/day Cr ingestion (Hultman *et al*, 1996). Following loading, elevated

muscle Cr stores can be maintained for at least one month by ingesting 2g of Cr per day in a single dose (Hultman *et al*, 1996). This maintains muscle Cr delivery at slightly above the rate of muscle Cr degradation to creatinine. Urinary creatinine output increases by about 20% which parallels the increase in muscle Cr content (Hultman).

Sub-maximal exercise performed prior to Cr ingestion can augment muscle Cr accumulation by about 10%, but again there is a marked variation in response between individuals (Harris).

### **Creatine plus carbohydrates**

Muscle Cr accumulation can be substantially augmented by ingesting Cr in combination with large quantities of simple carbohydrates (Green *et al*, 1996b). This reduces the variation in responses between individuals and also outweighs any stimulatory effect exercise has on muscle Cr accumulation (Green *et al*, 1996a, 1996b). Muscle Cr accumulation is thought to be augmented as a result of insulin-stimulating muscle sodium pump activity, and thereby sodium-dependent Cr transport. Recent evidence has demonstrated that it will require in the region of 100g of simple carbohydrates to be ingested to achieve an insulin-mediated stimulation of muscle Cr transport (Steenge *et al*, 1998). In practical terms, this will be difficult to achieve as the ingestion of such a large quantity of carbohydrate is at the limit of palatability. There is no data currently available to demonstrate the time-course of muscle Cr accumulation when Cr is ingested in the presence of large quantities of carbohydrate. This may be of importance.

A muscle Cr transport protein has recently been identified and it has been shown that its expression is down-regulated in rat skeletal muscle following six months of supra-physiological amounts of Cr supplementation (Guerrero-Ontiveros and Wallimann, 1998). Whether a similar response occurs in humans is unknown, as are the consequences of chronic Cr ingestion on the muscle Cr transport mechanism, ie, why does the muscle become desensitised to Cr as a result of chronic ingestion?

**Paul Greenhaff**







# WHO BENEFITS?

## First it was power athletes and sprinters. Now creatine seems to work for endurance athletes as well

There's no doubt that boosting the ability of the phospho-creatine system to deliver more energy to hard-working muscles during intense bursts exercise is of great benefit to power and strength athletes, such as sprinters and throwers, and this has been confirmed beyond doubt by numerous scientific studies. But once maximal exercise duration extends over about two minutes, very little of the ATP generated to contract muscle fibres comes from the phospho-creatine system. This explains the lack of initial enthusiasm for creatine supplementation from endurance athletes, whose events (save for a final kick to the finishing line – but see later) rely almost exclusively on aerobic power. This reluctance was compounded when reports of weight gain during the creatine-loading phase began to circulate amongst the athletic community. But that is all changing thanks to recent research showing that endurance athletes really can benefit from creatine supplementation. The reasons are many, but include the following:

**Interval training** – Almost all endurance athletes carry out interval training, both to increase their basic speed and lactate threshold, and to help them provide the necessary kick at the end of a tight race. Research previously reported in *Peak Performance* (Hot topics special, issue 162) and elsewhere has demonstrated that, for endurance athletes such as kayakers and distance runners, the quality of interval training, even with longer intervals of over three minutes' duration, was increased

significantly after creatine supplementation. Higher-quality interval sessions in training can produce higher speeds in competition, while the ability to out-kick rivals for the line during tactical or close races can mean the difference between winning and finishing nowhere!

**Increased strength** – Most endurance athletes need a certain degree of strength training to perform well in their event, help withstand the rigours of training and to stave off injury. Since high-intensity strength or resistance training is powered almost exclusively by the phospho-creatine system, it's easy to understand how creatine supplementation can facilitate more intense, higher-quality workouts, leading to greater strength gains per unit of time invested, in turn freeing up more time for endurance training.

**Anti-catabolic action** – At high aerobic training volumes, so much energy is expended that some muscle mass loss through catabolism is virtually unavoidable. While excess weight harms the performance of most endurance athletes, excessive weight loss is equally problematical, leading to loss of power and an increased injury risk. But new research carried out in Ontario, Canada (Parise et al, *J Appl Physiol*, 2001 Sep; 91(3): 1041-7) indicates that, in men at least, creatine supplementation produces an anti-catabolic effect, by reducing by one-fifth the amount of leucine (a crucial and abundant amino acid in muscle protein) subsequently oxidised. Although more work needs to be carried out, the initial implications for endurance athletes are positive.

**Glycogen resynthesis** – A major challenge for endurance athletes is to maintain stores of muscle glycogen during periods of heavy training and competition. As we've seen, the scientific jury is still out on the benefits of creatine supplements and increased muscle glycogen synthesis, but new research trickling in is certainly encouraging. A study last year conducted at the Louisiana State University (Nelson *et al*, *Med Sci Sports Exerc*

*‘The scientific jury is still out on the benefits of creatine supplements and increased muscle glycogen synthesis, but new research is certainly encouraging’*

2001 Jul; 33(7): 1096-100) used 12 men to show that a five-day creatine-loading regime increased the subsequent glycogen-loading ability of the vastus lateralis muscle of the quadriceps by around 20% compared to no creatine supplementation. The authors concluded that the glycogen-loading capacity of a muscle is influenced both by its initial levels of creatine and the accompanying alterations in cell volume.

**Creatine as an anti-oxidant** – Endurance athletes training and competing at high percentages of  $\text{VO}_{2\text{max}}$  encounter increased oxidative stress as a result of the increased production of Reactive Oxygen Species (ROS) via normal aerobic metabolism. Scientists now believe that oxidative stress and free radical damage is linked to long-term cellular degeneration, ageing and degenerative disease, and that boosting the body's anti-oxidant defenses may confer some protection against this process. In addition, oxidants such as free radicals can affect muscle fatigue and protein turnover. Although creatine is not classed as an anti-oxidant nutrient, it seems it may offer anti-oxidant protection as a bonus. In an *in-vitro* study earlier this year (Lawler *et al*, *Biochem Biophys Res Commun* 2002 Jan 11; 290 (1): 47-52), researchers found that creatine displayed a powerful anti-oxidant scavenging ability, particularly against the superoxide anion.

**Andrew Hamilton**



# Endurance cyclists improve their performance after taking creatine

Much of the research work on creatine supplementation has focused on short-term exercise, since creatine is linked to the phospho-creatine energy pathway. A paper by academics at West Sussex Institute examined the effect of oral creatine supplementation in longer-term exercise, this time on the bike.

Sixteen subjects were split into two groups, who had been previously assessed. There was no difference in 10-mile performance between the two groups before the study commenced. One group was given the creatine supplement with a glucose polymer for four days while the other group was given the glucose polymer only. Apart from that, subjects were asked to stick to their normal dietary regime.

After the four days the subjects were re-tested on the bike. Both groups saw an improvement in 10-mile performance but this improvement was only statistically significant in the creatine supplement group. Such work indicates that there are possible ergogenic effects of such a supplement for long-term exercise and certainly warrants further research before there is a move for another substance to be added to the banned list!



## Low doses of creatine can help endurance athletes when they need to surge or

Creatine, as most readers of this special report will know, is manufactured routinely by the liver and kidneys and may be found in significant quantities in muscle, nerve and sperm cells. Within the muscles, it is used in the form of creatine phosphate, a high octane chemical which assists in supplying the energy indirectly needed to perform muscle contractions. This process of muscles being powered is performed by adenosine triphosphate (ATP), which is created inside muscle cells from carbohydrates, fats and proteins in the presence of oxygen, being broken down to adenosine diphosphate (ADP) during exercise. However, since ATP is generated slowly, creatine phosphate acts as a reserve source of immediately available energy by giving its phosphate to ADP, creating ATP quickly in order to make the muscles work.

Numerous studies, some of them noted in this special report, show that oral creatine monohydrate supplementation can produce performance-enhancing effects during anaerobic bouts of repeated exercise. This, of course, is good news for sprinters and weight lifters, but what about endurance athletes, who surge during a race to break the field or kick for the line at the end – can creatine supplementation help them? Recently, Dr Martin Engelhardt from the Orthopoadische Universitatsklinik in Frankfurt formulated a cycle ergometer experiment comparing endurance/ power endurance exercise in athletes without creatine supplementation against those with low-dose supplementation.

## **What the experiment involved**

Twelve regional class triathletes aged between 22 and 27 were selected to take part in the test. Each athlete underwent a preliminary incremental cycle ergometer test to determine their 3mmol.L<sup>-1</sup> blood lactate intensity. Once established, this intensity was used as the aerobic exercise intensity for the experiment.

The testing began with each athlete performing their aerobic exercise on the cycle for 30 minutes, followed by two bouts of high-intensity interval training, which involved cycling hard for 15 secs and then 45 secs at their aerobic exercise intensity, 10 times before two minutes' rest and systematically repeating the interval session. After this, the subjects were 'rewarded' with another 30 minutes of cycling at their specific aerobic exercise intensity (a total of one hour and 20 minutes cycling!). The test results were then kept from the athletes, and the following day they were administered six grams of creatine daily (to be taken in two 3g doses) for a period of five days before returning to repeat the test. You've probably noticed that the creatine loading dose only amounted to 30g over the five days, whereas in many other studies of creatine supplementation the normal dose is 100g (four doses of 5g daily for five days).

Blood samples were taken throughout the testing to monitor creatine, creatinine (the degradation product of creatine), glucose and lactate levels, while urine samples (for measurement of creatine and creatinine urine levels), heart rate and oxygen uptake were measured both before and after the aerobic exercises.

## **And the results?**

As you would expect, after creatine supplementation analysis of the blood and urine samples showed significant increases in the serum creatine concentration in the blood and serum creatinine levels in the urine. 50% of the athletes in the first test couldn't complete the entire second 30-minute bout of endurance exercise, whereas after taking creatine each of the athletes who had struggled in the first test managed to increase



their ride time by four minutes, although this increase didn't reach any statistical significance. However, after taking creatine supplements, the total number of interval reps increased significantly and 75% of the athletes improved their performance (nobody did the reverse). Lactate concentrations showed no changes in the endurance test and interval training following supplementation.

After analysing the results, the researchers concluded that, at lower doses, ie, 6g daily for five days, creatine supplementation appears to have an effective impact on endurance athletes if they are required to sprint during an aerobic bout (as may happen when, for instance, you find yourself up against a couple of Kenyans during a 10k race). This conclusion is supported by the 18% increase in interval performance after supplementation that was found in the study.

The problem is that the test wasn't performed as a double-blind trial, so both the athletes and the testers knew exactly when each athlete was taking creatine. Thus, sceptics could argue that it was the psychological or placebo effect that enabled the athletes to perform much better during the second test because they believed the creatine was going to help them. That objection apart, it is interesting that such a comparatively low dose of creatine compared to normal practice seems to have had such a significant effect. One possible explanation is that an individual's creatine 'pool' is positively related to lean body mass; the more muscle, the larger the natural creatine stores. Thus an athlete with a slight frame would need less creatine supplementation than a bulkier athlete.

*‘After taking creatine supplements, the total number of interval reps increased significantly and 75% of the athletes improved their performance’*

**Jamie McLoughlin**

## **Reference**

Engelhardt, M. et al (1998). 'Creatine supplementation in endurance sports'. *Medicine & Science in Sports & Exercise*, 30 (7): pp 1123-1129



# Are we to believe the recent scare stories about creatine?

Despite the enormous weight of evidence that creatine supplementation ‘does what it says on the bottle’, there have been a few claims disputing its efficacy, while some have raised concerns about possible side effects. Let’s examine these more closely.

### **Creatine doesn’t work**

Some studies have reported little or no benefit when supplemental creatine is ingested, but many of these negative results can be explained in terms of the experimental design. For example, some studies used insufficient creatine to saturate muscles, while others did not check to ensure that the extra creatine given has actually been taken up into muscle stores. Some studies on repeated bouts of exercise used recovery intervals that were simply too long to demonstrate the increased ability of the phospho-creatine system to deliver higher power intensities. One study even used a predominantly aerobic exercise task of 10-15 minutes duration to confirm the absence of any effect of creatine supplementation – not surprising really!

What is known, however, is that there is considerable individual variability in both the transport of creatine into muscle cells and its subsequent storage. Studies show that while many people supplementing creatine seem to retain it in large quantities, a few others don’t retain any at all. These individuals may have an initial total creatine concentration that is already relatively high. In these circumstances, neither an appreciable uptake of creatine nor an effect on phospho-creatine resynthesis or performance has been found after creatine supplementation (Harris, 1991; Greenhaff, 1994).

## **Creatine causes weight gain**

During the loading phase of supplementation, many users report a short-term weight gain in the region of 3-5lbs, most of which appears to be due to increased hydration within muscle cells. However, it's worth pointing out that this increase is approximately the same as that observed after carbohydrate loading, and any negative effects associated with an increase in body mass, for example in weight-bearing events, are offset by the increase in exercise capacity that creatine supplementation produces. Moreover, recent research (*Running Research News*, 1998) reported in *Peak Performance* indicates that a longer, lower-dose loading phase of around 3g per day, taken in half-gram amounts, produces only around a 1lb gain in weight, with lower spillage (ie waste) into the urine. Longer term, weight gains are likely to be associated with lean muscle-mass increase. This is because creatine supplementation can enhance the quality of high-intensity workouts, which in turn stimulates muscle synthesis.

‘There is considerable individual variability in both the transport of creatine into muscle cells and its subsequent storage’

## **Creatine causes muscle cramping**

There have been a few anecdotal reports of muscle cramping during periods of creatine loading and speculation that this may occur as a result of the dilution of muscle electrolytes. However, although there has been no systematic investigation of this phenomenon, no studies have ever reported cramping as a side effect of creatine supplementation.

**Creatine causes liver or kidney problems** – Used in the quantities required to load and sustain muscle creatine saturation, there is no evidence that creatine supplementation produces any discomfort or harmful side effects. Greenhaff *et al* (1993) found that when full haematologic and clinical chemistry screening was carried out before and after creatine supplementation of 20 grams per day for five days, no alteration in markers of liver or kidney function were reported. This finding has subsequently been confirmed in several other studies (Almada, 1996; Earnest, 1996). However, the long-term

effects of creatine supplementation, over several months, for example, are not known, so athletes shouldn't feel they have carte blanche to swallow large quantities of creatine indiscriminately.

**Andrew Hamilton**



# Could caffeine take the buzz out of your creatine?

Creatine continues to ride high on a wave of positive research results. As readers of this special report will know, taking creatine supplements has consistently been shown to improve performance that involves repeated intensive exercise – eg, a series of sprints.

Once inside muscle cells, a proportion of creatine is converted into creatine-phosphate. High levels of creatine-phosphate are thought to increase muscles' capacity for work in three ways:

- 1) providing an instant source of energy
- 2) mopping up some of the fatigue-causing acid that builds up during high-intensity exercise
- 3) directly stimulating muscle proteins to contract.

Much of the creatine research consists of carefully controlled lab studies. But what about real-life situations, where other factors may have an effect? The overall composition of the diet may be relevant – for example, there have been suggestions that creatine supplementation has an optimal effect in the context of a high-carb diet. This is because carbohydrate stimulates insulin release, which in turn encourages the uptake of creatine into cells.

Another common dietary component is caffeine. Caffeine is known to boost the activity of the transport system that shuttles creatine from the bloodstream across into the muscle cells. It also increases the amount of adrenaline coursing through the blood, which should also encourage uptake of creatine. A team of Belgian researchers therefore recently investigated the effect

of taking creatine supplements with caffeine. They anticipated that caffeine would lend a helping hand to creatine. They were surprised and disappointed to find the opposite – caffeine actually counteracted creatine's positive effects!<sup>1</sup>

### **What they did**

The researchers recruited nine men who did regular recreational exercise, but were not at a high level of training. Each man then undertook a series of three regimes, allocated in a random order, with a three-week break in between. At no stage did the men know whether they were taking caffeine, creatine or placebo. The timetables were as follows:

- 1) creatine loading for six days, taking 0.5g of creatine monohydrate per kg of body weight per day
- 2) placebo pills for six days
- 3) creatine loading as in A, plus taking a dose of caffeine at breakfast in the last three days of loading. Caffeine dose was 5mg caffeine per kg of body weight (for a 70kg man, the equivalent of three strong cups of ground coffee, or seven cups of tea).

Muscle creatine-phosphate levels were measured before and after each regime. In addition, a series of tests to analyse muscular force were carried out on the subjects by making measurements during a standardised sequence of knee extension exercises.

Both creatine and creatine-plus-caffeine led to higher levels of muscle phospho-creatine compared with placebo. But the addition of caffeine didn't boost the phospho-creatine to a higher level than that achieved by creatine alone.

This may be explained by a phenomenon which has been observed in other studies – basically, you can only stuff a muscle cell with so much creatine. Once it's reached a certain level, any extra just gets shunted to the kidneys and excreted. So the caffeine may have helped the muscles to fill up more quickly with creatine, but once saturation point was reached, there would have been no additional effect.

*‘They were surprised and disappointed to find caffeine actually counteracted creatine’s positive effects!’*



When creatine supplements were given, knee muscle force was boosted significantly, as would be expected given the raised muscle levels of creatine phosphate. But, big surprise: although caffeine-plus-creatine also raised muscle phospho-creatine levels above placebo, the boost to muscle performance seen with creatine alone was completely missing! The researchers were at a loss to explain this unexpected result. Maybe creatine has its positive effect on muscles via another route than merely raising the creatine phosphate pool inside the muscle cells.

### **Resynthesising**

Other research has suggested that it is the ability to rapidly resynthesise creatine phosphate between bouts of activity that is critical, rather than having elevated levels to begin with. It's possible that caffeine interferes in some way with this process once exercise has started, but it's by no means clear how.

We need to wait for more work in this area to see if caffeine's knock-out effect on creatine is found consistently. The way this study was set up was not ideal – from other reports in the literature, it appears that muscles maintain an elevated level of creatine for six-eight weeks after a period of creatine loading. In this case, only three weeks were allowed as a 'wash-out' period between the regimes. This may well have confused the results.

Meanwhile, what's the practical take-home message for the way? If caffeine really does downgrade creatine's effect, what should you do? It all depends on what type of exercise you're involved in. So far, creatine supplementation has only been proved to be of benefit for repeated, intensive exercise. By a lucky coincidence, this type of exercise doesn't seem to be boosted by caffeine. In contrast, there's evidence that caffeine can improve endurance exercise performance – and creatine doesn't seem to have any positive effect on this type of activity (though see earlier articles in this special report).

So, if you're in a situation where you think creatine loading might help you, you may want to avoid caffeine during the loading period, and for four days before you start taking the

creatine supplements. If you're involved in endurance exercise, creatine probably won't help you anyway, so you can gulp down caffeine with no worries.

**Janet Stansfeld**

### **Reference**

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# HOW MUCH TO TAKE

## **If you want to improve performance, get the dosage right, and maximise creatine uptake. Combine it with carbo!**

It's important to realise that once any ingested creatine has been taken up by muscles, it is effectively 'locked up' within the muscle cells and is only turned over and degraded at the rate of around 2 grams per day. So to produce a net gain in stored muscle creatine and promote creatine loading, you need to ingest a daily amount that is in reasonable excess of 2 grams. It is also important to appreciate that once muscle creatine saturation has been achieved, you need only consume enough creatine to replace your daily losses in order to maintain this state of saturation.

### **Loading phase**

Most studies have used creatine supplementation at a dosage of 20g per day (divided in four doses of 5g) for a period of five-to-six days. Some studies have used even higher doses for shorter periods, but it is also possible to saturate muscles fully at a much lower dose. For example, taking 3g per day for four weeks is equally effective at producing saturation – it just takes longer to get there. However, patience could be a virtue, since studies show that lower loading doses appear to be better tolerated and more efficiently absorbed by the body (*International Journal of Sports Nutrition and Exercise Metabolism* Sept, 2000). Whichever route you take, once saturation has been achieved, there's no point in continuing on a high-dose regime, since any extra is simply excreted in the urine.

## Maintenance phase

Two grams per day seems to be sufficient to maintain most of this muscle creatine saturation for periods of up to 28 days. The efficacy of the maintenance dose in sustaining muscle creatine concentration beyond this period is unknown.

## Creatine type

Virtually all the scientific studies on creatine have used creatine monohydrate (a creatine molecule chemically bonded to one water molecule). Despite the appearance of a number of 'newer' and more expensive versions of creatine such as creatine phosphate, creatine citrate and creatine serum, there is absolutely no peer-reviewed, double-blind scientific research to substantiate the superiority of these new forms over regular creatine monohydrate in humans, despite marketing claims to the contrary.

In fact, it is probably more important to worry about the purity of the creatine you use than its type. A recent consumer survey found that although the overall purity of each product averaged about 90%, there were dramatic differences in the amount of several potentially toxic impurities present. During the industrial production of creatine monohydrate from sarcosine and cyanamide, variable amounts of contaminants such as dicyandiamide, dihydrotriazines and creatinine are generated (Benzi and Ceci, *Sports Med Phys Fitness* 2001). Creatine users should only purchase it from reputable manufacturers who are able to provide a Certificate of Analysis. The purity recommendations include:

**Appearance** – should be white to pale cream

**Assay** – should be at least 95% via HPLC or HPCE

**Moisture content** – should be less than or equal to 12.5%

**Microbial/pathogenic contamination** – should be negative for *E. coli*, *S. aureus*, and *Salmonella*

**Yeasts and moulds** – should be less than 50 per gram

**Poisons/heavy metals** – should be less than 10ppm for lead and mercury

**Other contaminants** – should be less than 3ppm for arsenic, 30ppm for dicyandiamide and non-detectable for dihydrotriazine

## What about creatine in the diet?

The best sources of creatine in the diet are meat and fish. However, a quick glance at the creatine content of some of the better sources (below) shows that while dietary creatine may make a significant contribution to a 2-3g per day maintenance phase, it would be virtually impossible to achieve the multi-gram daily intake required in the loading phase from dietary creatine alone, making supplementation unavoidable.

<b>Prawns</b>	trace
<b>Herring</b>	3-4g/lb
<b>Beef</b>	2.0g/lb
<b>Cod</b>	1.4g/lb
<b>Salmon</b>	2.0g/lb
<b>Tuna</b>	1.8g/lb
<b>Milk</b>	0.05g/lb

## How to maximise creatine uptake

The research shows unequivocally that the greater the creatine uptake into muscles and the greater the saturation that is subsequently achieved, the greater the increase in performance. However, a complicating factor is that some individuals appear to soak up creatine and achieve saturation far more easily than others, and this has led researchers to discover whether there are strategies for maximising the efficiency of creatine uptake into muscles and which allow the widest range of individuals to achieve their apparent upper limit.

## Creatine and exercise

Initial studies carried out by Harris *et al* (1992) showed that creatine uptake was increased by around a third into muscles simply by exercising those muscles sub-maximally for an hour a day. However, this approach still produces large variations in the response to creatine uptake between individuals.

## Creatine and carbohydrate

A much more effective approach is to combine creatine supplementation with carbohydrate intake. In a landmark study (Green *et al*, 1996) demonstrated that during the loading phase (5g of creatine four times per day for three days), taking 500ml of 18.5% sugar solution with each 5g dose, almost doubled creatine retention compared to simple creatine loading. This carbohydrate regime was also superior to creatine plus exercise. Although the creatine retention figures were similar for the first day, by the third day (as the point of muscle saturation was approached), the creatine/carbohydrate regime produced creatine retention figures some 50% higher than the creatine/exercise combination. The creatine/carbohydrate combo also produced a much more uniform response among individuals, indicating that this combination may be particularly suitable for those who have difficulty achieving muscle-creatine saturation.

Adding exercise to the creatine/carbohydrate regime produced no extra benefits whatsoever, suggesting that the mechanism behind the creatine/carbohydrate combination was what really counted. Although this mechanism has yet to be conclusively identified, it seems highly likely that ingesting simple carbohydrate with creatine produces an insulin response, which in turn helps to 'drive' the circulating creatine into muscle cells. The study above used around 90g of simple sugars following each 5g dose of creatine – a ratio of 18g of carbohydrate per gram of creatine (a total of 360g of simple sugars per day!). If, however, you're someone whose blood sugar is sensitive to high intakes of simple sugars, you can take

“Creatine uptake was increased by around a third into muscles simply by exercising them sub-maximally for an hour a



comfort from the fact that other creatine users have reported good results using a more modest carbohydrate ratio of around 6g of carbohydrate per gram of creatine (around 30g per 5g dose of creatine).

**Andrew Hamilton**



## HMB does wonders for chickens and pigs, but does it really work for human athletes?

The aim of this review is to provide you with the latest information currently available about HMB and look at results of studies that have evaluated its effectiveness, so that you can answer questions about its effectiveness for yourselves.

To give it its full name, HMB is beta-hydroxy beta-methylbutyrate, which is formed in a metabolic pathway in which leucine, an important branched-chain amino acid, is converted to HMB; thus HMB is often known simply as a metabolite of leucine. We rely largely on our normal diet for sufficient intake of protein containing leucine, since up to 5% of this amino acid can be converted into HMB (Gatnau *et al*, 1995). Small amounts can be found in citrus fruits and catfish.

### How was it discovered?

You may first have heard of HMB during the 1996 Olympic Games when American swimmers were quoted as saying that they had used HMB during training to enhance performance. As with many sports nutrition supplements now on the market, HMB was first discovered during animal tests in 1988. Recognising the importance of leucine in muscle building, Dr Steven Nissen, then a researcher at Vanderbilt University, supplemented cattle's diets with leucine with the aim of producing a leaner herd. However, though effects were positive, and have been replicated with chickens, goats,

hamsters, pigs and sheep, the amounts of leucine required were found to be phenomenal. Dr Nissen then analysed the by-products of leucine breakdown and pinpointed the metabolite HMB as being responsible for the hypothetical muscle-building effects in cattle.

So what we want to know is, can HMB's positive benefits be transferred to humans and, more importantly, will it be an effective supplement in sports performance? Certainly Owen Anderson thought so, in his 1996 article in *Peak Performance*, reckoning HMB would be of value to both strength and endurance athletes. Before considering what later research has said, let's first examine why HMB is supposed to work.

### What HMB does

The main theory for its effectiveness appears to be that it is 'anti-catabolic'. Catabolic means the breaking down of tissue, as opposed to anabolic, which means building it up. So in effect HMB spares muscle-tissue breakdown. When you train, you stress the body's systems, causing a breakdown of specific tissues. The body then regenerates this tissue to a greater extent than pre-training levels. So it follows that if you break down less of the muscle tissue with the same training response, the recuperation or anabolic phase begins from a higher starting point, leading ultimately to more defined increases in muscle mass.

Some of you might think that it is the breakdown of tissue that triggers the rebuilding, so with reduced levels, less rebuilding will take place. However, if that was the case, then when a muscle injury was sustained, the muscle would rebuild to a greater level, and this clearly does not happen. Briefly, HMB is thought to increase muscle mass by preventing protein breakdown caused by resistance training, and enhancing the repair process (Nissen *et al*, 1996).

### What recent studies suggest

If the latest scientific thinking is to be believed, all athletes would benefit from HMB! Here's a rundown of the current

“HMB is thought to increase muscle mass by preventing protein breakdown caused by resistance training, and enhancing the repair process”

research, some published in sport science journals, some of it unpublished work taken from the web:

### *Strength athletes and those wishing to build muscle*

Nissen *et al* (1996a) revealed similar results when testing both trained and untrained athletes and concluded that HMB supplementation does increase muscle mass beyond that of training alone. This research suggests that HMB can increase muscle mass by 1.2kg over three weeks when taken as a daily 3g supplement while undertaking resistance training.

However, some scepticism exists because much of the early research was promoted by the major manufacturers of the supplement. Further work by Nissen *et al* (1996b) substantiates the claim that HMB may, indeed, increase muscle mass. Other studies have shown that the ratio of muscle gained to muscle lost improves with HMB supplementation, and, in order to quantify this improvement, analysis of research findings shows that almost 44% less protein catabolism (protein breakdown) occurs with HMB supplementation. In addition, there is less evidence of muscle damage following training.

### *Women athletes*

Again, Nissen *et al* (1997) reported that 'enhanced strength, lean gains and increased fat loss occurred following HMB supplementation' in women undertaking an exercise programme.

### *Older athletes*

To date, two pieces of research have been carried out in this area, the idea being that, in contrast to emphasising the strength gains shown in the research already mentioned, older athletes lose muscle mass in a catabolic fashion and thus HMB might decelerate this process. In the first study, by the ubiquitous Nissen *et al* (1996), it was shown that leg strength increased by 7.1% with HMB over training alone, lean mass increased by 2.1%, and fat mass reduced by 4.4%. Although recent research by Panton *et al* (1998) showed no significant difference in leg

and chest strength with older individuals on a course of HMB, functional ability did improve which probably, above 70 years of age, is more important than strength gains.

### *Endurance athletes*

A study by Vukovich and Adams (1997) showed that when endurance-trained cyclists supplemented with HMB, time to reach VO<sub>2</sub>max and lactate threshold was significantly higher. However, maximum lactic acid levels remained unchanged, indicating no alteration in aerobic and anaerobic metabolism.

Some of the studies mentioned above have shown that HMB may reduce body fat by up to 4.4%. The theory behind this may be that increased muscle mass allows accelerated oxidation of fuel (so that you burn more calories) and is supported by research suggesting that HMB may lower cholesterol levels.

So how much HMB should you take and how much does it cost? The existing research has centred around the use of 3g a day in three 1g doses. The current cost of HMB is around £40 for 120 capsules, each contained 250mg. That amounts to four capsules three times a day, and means a cost of £40 for a 10-day supply. Most of the studies of strength and muscle-mass gain have been over six-eight weeks, which would cost about £160-200.

How much muscle could you expect to gain? It is suggested that you will, at best, double your usual muscle-mass gains, which for a normal athlete would be about 1kg. So for an outlay of £160-200 you could expect to gain an extra 1kg of muscle mass in roughly two months!

### **How should you train while taking HMB?**

In order for the gains to take place, Nissen and Baier (1997), supported by an HMB manufacturer, devised a programme which helped to intensify training (essential for maximum effect). The full programme can be found on the internet at <http://mettechnic.com/research.html>. In brief, the programme centres around a split routine of compound exercises for three sets of six-eight repetitions (reps) to failure (the split being

chest, back, shoulders, abdominals, and legs and arms). Chest, etc, trained on day one, legs and arms day three, chest, etc, day five, and reversing the order the following week. Days two and four involve 30 minutes of cardiovascular work, and days six and seven are rest days.

To many strength athletes, this programme will be nothing new, but here we would like to add our own knowledge of strength training in the form of ‘negatives’ (sometimes known as ‘eccentrics’). For those of you unaware of this concept, the assistance of a training partner is required, who lifts the weight when you have reached failure, allowing you to lower it slowly, for a maximum of one-two further reps. This is a very intense way of training and has been shown to produce maximum strength gains because of the overload it creates on the muscle and the DOMS felt over the next 72 hours (McArdle, Katch and Katch, 1994).

**Note:** This type of training should only be performed by experienced strength athletes. If the main theory of how HMB works proves to be accurate, then it makes sense to use negatives to promote maximum strength and muscle-mass gains. Remember, however, that this is an untested theory but one which, by definition, could work.

## **Is HMB safe?**

Very little research has been conducted here and we must rely on animal studies. An amount equal to 50 times the recommended dosage of 3g a day has been fed to pigs and ‘no adverse side effects were revealed’. Nevertheless, can we really assume that HMB won’t prove harmful to humans?

Of the little research that has been done, one recent study, Kreider *et al* (1998), again supported by a manufacturer of HMB, reported no difference in blood and urinary samples between those who took HMB and those who didn’t, but found raised levels of HMB in both samples following 28 days of supplementation. What does this mean? Basically, that none of the indicators of any damage was altered and that excess

HMB can be broken down and excreted from the body.

It is impossible to draw any concrete conclusions about safety from this one piece of research alone. Although positive benefits have been revealed and supported by accomplished researchers, neither the immediate nor long-term effects of taking HMB have yet been definitively established. We need to wait for further research before that happens.

If, however, you want to try it for yourself, HMB is available in many health food stores, weight training centres, by mail order through body-building magazines or on the internet. HMB is licensed to Metabolic Technologies Inc. in the USA, and only by buying it from companies supplying it under licence can you be sure that you are buying genuine HMB.

**Mistrelle Airstone and Peter Baker**

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# TWO MORE ERGOGENIC SUPPLEMENTS

## These three short reports look at two other performance boosters – caffeine and sodium bicarbonate

### *Caffeine (1)*

In recent research carried out at McMaster University in Ontario, researchers gave either a placebo or about 600 milligrams of caffeine (approximately the amount in four cups of brewed coffee) to five endurance athletes prior to prolonged rides to exhaustion on exercise bicycles. The study was carried out in a randomised, double-blind fashion (athletes who initially ingested caffeine tried the placebo at a later date and vice versa; neither athletes nor researchers were initially aware of who was actually taking in caffeine).

After the exhaustive rides, the scientists used an electrical device to stimulate muscles in the lower portions of the athletes' legs. When the electrical stimulation was greatest, the 'caffeine-loaded' muscles proved to be about 25% stronger (they contracted about 25% more forcefully with caffeine than with placebo).

### **Why caffeine works**

How did caffeine reduce fatigue and enhance muscle strength following an exhaustive workout? Theories about caffeine's actual effects abound, with some scientists suggesting that the drug acts primarily on the nervous system and others believing that caffeine is purely a muscle stimulant. Since the electrical stimulation of leg muscles permitted the Canadian researchers

*“Caffeine may free up the sarcoplasmic reticulum to keep the muscles working at a high level”*

to bypass the athletes' nervous systems, it allowed them to show that caffeine can indeed act directly on the muscles.

Specifically, caffeine may enhance the release of calcium from the 'sarcoplasmic reticulum', a network of tubules inside muscle cells which acts as a holding tank for calcium. The setting free of calcium from the sarcoplasmic reticulum is the actual stimulus for muscle contraction, but this calcium liberation tends to become lethargic in the late stages of prolonged exercise. Caffeine may free up the sarcoplasmic reticulum to keep the muscles working at a high level.

In related research at the University of Guelph in Canada, scientists asked four well-trained runners to run to exhaustion at an intensity of 85%  $\dot{V}O_{2\max}$  (about 91% of maximal heart rate, MHR) one hour after ingesting caffeine tablets, placebo (sugar) tablets, decaffeinated coffee, decaffeinated coffee plus caffeine, or regular coffee.

### **What was the result?**

The Guelph researchers discovered two key things: (1) caffeine-associated diuresis was minimal, amounting to no more than about half a cup of extra urine; (2) caffeine tablets proved to be far more ergogenic than regular coffee or decaffeinated coffee plus caffeine. The athletes cruised at 91% MHR for just 26-28 minutes after the latter beverages but buzzed along for a full 41 minutes after swallowing the tablets, even though the actual dosage of caffeine (4.5 mg per kg of body weight) was the same in all three cases.

Following the ingestion of placebo tablets, the runners came to a stop after just 22 minutes, but – strangely enough – they fared just as well after drinking pure decaffeinated coffee as they did after taking in real coffee or coffee plus caffeine (perhaps they were aware that caffeine might boost performance and thought they were taking the real thing, thus getting a mental, if not physiological, boost). Still, ingesting pure caffeine tablets was by far the best strategy.

Although runners love to swill coffee before marathons, the Guelph investigation is the first to link caffeine with improved

running performances at a distance greater than 1500 metres. However, it's not clear why the caffeine tablets were better than real coffee, and the small number of subjects involved makes the study a preliminary one.

('Caffeine and Neuromuscular Fatigue in Endurance Athletes', *Canadian Journal of Applied Physiology*, vol. 19, Supplement, p. 41P, 1994, and 'Comparison of Coffee and Caffeine as an Ergogenic Aid', *Canadian Journal of Applied Physiology*, vol. 19, Supplement, p. 19P, 1994)

## *Caffeine (2)*

Another study, however, suggests that caffeine doesn't boost short-term performance – but it increases the heart rate.

As we've already seen, two questions that have intrigued sports scientists for 25 years are: does caffeine ingestion prior to exercise cause an improved performance, and if it does, what mechanisms are responsible for that improvement? Early research indicated that caffeine ingestion increased time to exhaustion in endurance exercise because it caused a metabolic response in humans. The reason for this was thought to be that the presence of caffeine in the blood appeared to stimulate the release of the catecholamines adrenaline and noradrenaline, which in turn brought about an increase in the availability of fat as fuel for the working muscles; the effect of this was thought to be a sparing of muscle glycogen, or carbohydrate stores.

Other possible explanations for caffeine's action – for instance, that it may result in more forceful muscular contractions by affecting the action of calcium in the muscle – have been discounted because of the unfeasibly large (probably toxic) amount of caffeine needed to promote such actions. This leaves the stimulation of catecholamine release as the most likely explanation for caffeine's ergogenic effect, and, for this reason, most studies investigating caffeine's effect on performance have concentrated on endurance exercise. There would appear to be little point in studying its effect on short-term (ie, less than five minutes), intense (90-100%  $\text{VO}_2\text{max}$ )

exercise, where the provision of glycogen is not a limiting factor.

Nevertheless, some research has shown that caffeine does improve short-term performance, although the results have not always been statistically significant. In such exercise, it is believed that caffeine must act directly on the muscle or on the central nervous system if it is to alter performance.

## **Withdrawal symptoms**

In a recent study at the University of Brighton, a group of undergraduate sports scientists took part in an experiment which was designed to test whether caffeine does affect short-term performance. The subjects were given a gelatine capsule containing either a placebo or caffeine (5mg/kg body mass) one hour prior to a 1500m time-trial performed on a friction-braked cycle. Each subject chose his own strategy to cover the 1500m as quickly as possible. Subjects were asked to refrain from caffeine ingestion for two weeks prior to the first test and until after the second test had been completed. The second test was carried out by all subjects one week after the first trial at the same time of day.

The potency of caffeine as a drug was initially illustrated by the fact that many of the students experienced quite powerful withdrawal symptoms. When the placebo and caffeine capsules were given out, 81% of the subjects correctly identified that they had taken caffeine and 94% correctly identified that they had taken the placebo. Caffeine ingestion did not cause any significant changes in heart rates during the warm-up, or after a recovery period, and it did not alter the time taken to reach half-distance. It also did not significantly change the time taken to complete the 1500m, or mean  $\text{VO}_2$  (oxygen uptake). It did, however, result in a significantly increased mean and peak exercise heart rate.

The conclusion from all this? Caffeine did not cause an improvement in this type of performance but it did cause a significant increase in exercise heart rate. That being said, the fact that the mean time to complete the trial was 1.2 seconds quicker during the caffeine trial than during the placebo initially

looked exciting. This is because the level of improvement required by an athlete may be smaller than the level of scientific significance. Does this therefore suggest that caffeine ingestion would be a worthwhile tactic before competing in events of this nature? No, for the following reason.

### **The learning effect**

Although no significant differences were found between the performance times when expressed by trial order, mean performance during the second trial was 1.3 seconds quicker. Similarly, mean time taken to perform the last 750m was 2.6 seconds quicker during the second trial. This implies that there was a learning effect, or, more simply, the subjects got better at performing the task once they got used to it.

The results from this experiment suggest that the most likely cause of caffeine's ergogenic effect in endurance events is that it does stimulate catecholamine release. This was borne out by the significant increase in the exercise heart rate found in this experiment, because catecholamines accelerate the depolarisation of the sinus node and cause the heart to beat faster. The increase in the warm-up and recovery heart rates in this experiment were not significant, though they were clearly elevated during the caffeine trial. The greater increase in heart rates during the exercise may have been caused by additional catecholamine release stimulated by physical performance.

This experiment suggests that there is no benefit in using caffeine as an ergogenic aid in short-term, high-intensity exercise. In addition, the changes in heart rates during the experiment, together with some of the comments from the students about how they actually felt (ranging from 'profoundly sick' to 'weird') did show that caffeine is a very potent drug even when only a moderate dose (5mg/kg) is taken. This dosage would result in urinary caffeine levels below the limit set by the International Olympic Committee for competition. The unpleasant side-effects might cause even endurance athletes to have second thoughts about using caffeine.

**Lee Oliver**

## *Sodium bicarbonate*

This supplement has been researched by sports scientists for some time, producing inconsistent results, though some studies have suggested it has great potential for enhancing anaerobic performance. Perhaps the one major confounding factor is the relatively common side-effect of stomach problems. I'll come back to this later, but, first, what is the rationale behind the use of bicarb by athletes?

### **Underlying theory**

Energy production via anaerobic glycolysis, which is particularly important for events lasting between 30 seconds and 15 minutes, increases the acidity inside the muscle cells, and very soon after does the same to the blood. It is this increase in acidity within the muscle cells that is a major factor in producing fatigue in such events. If there was some way to reduce the acidity within the muscle cells, one could theoretically delay fatigue and thus continue exercising at a very high intensity for longer. Sodium bicarbonate is an alkalising agent and therefore reduces the acidity of the blood (known as a buffering action), but cannot enter the muscle cells to reduce the acidity there. However, by buffering acidity in the blood, bicarbonate may be able to draw more of the acid produced within the muscle cells out into the blood and thus reduce the level of acidity within the muscle cells themselves. This could delay the onset of fatigue.

‘By buffering acidity in the blood, bicarbonate may be able to draw more of the acid produced within the muscle cells out into the blood and thus reduce the level of acidity within the muscle cells themselves’

### **Who might benefit?**

The specific athletes who might stand to benefit from bicarb supplementation will typically compete in events that last between one and seven minutes, ie, 400m-1500m running, 100m-400m swimming, most rowing competitions, and many team sports with their repeated nature of high-intensity exercise which stresses the anaerobic glycolysis system significantly and produces a lot of acidity.

### **Recent research saying it works...**

Bird and colleagues (*Journal of Sports Sciences*, 1995, vol. 13,



no.5, pp 399-403) persuaded 12 middle- and long-distance runners to compete in a total of six 1500m races. The three different conditions were: after bicarbonate ingestion, after placebo ingestion, and after ingestion of neither of these. The bicarbonate ingestion trial produced race times (about 4:14 mins) mainly between three and five seconds faster than the other two conditions.

Hausswirth and colleagues (*European Journal of Applied Physiology and Occupational Physiology*, 1995, vol. 71, no. 4, pp 362-368) found that eight subjects were able to improve local muscle endurance of the quadriceps during a sustained contraction at 35% of maximal force after ingestion of sodium citrate (sodium citrate raises blood bicarbonate by a similar amount as sodium bicarb itself).

Callier and colleagues (*Cinesilogie*, 1994, vol. 33, pp. 45-50) had 12 male subjects perform five one-minute bouts of cycling with two-minute rest intervals at an intensity equivalent to 100%  $\text{VO}_{2\text{max}}$  after placebo or citrate ingestion. The fifth bout of cycling was in fact longer than one minute and continued until exhaustion. Citrate ingestion delayed fatigue in the fifth exercise period, adding an average of 20 seconds to exercise capacity that was determined largely by anaerobic function.

### **...and that it doesn't**

Cox and Jenkins (*Journal of Sports Sciences*, 1994, vol. 12, pp 469-475) used eight moderately-active male subjects to evaluate the effects of sodium citrate ingestion on repeated 60-second sprints on a cycle ergometer. Despite changes in blood bicarbonate and lactate measures which suggested that the supplementation was working correctly, performance (work done cycling) was no different between supplementation and placebo trials.

Kozak-Collins and colleagues (*Medicine and Science in Sports and Exercise*, 1994, vol. 26, no. 12, pp. 1510-1515) also found no significant improvement in performance, although their raw data did suggest some improvement which may have been significant if a larger sample size had been used. After

ingestion of sodium bicarbonate or a placebo, seven competitive female cyclists performed intermittent exercise of one minute at 95%  $\dot{V}O_{2\max}$ , the next minute at just 60 watts, until exhaustion after an average of nine bouts at each intensity. Once again, blood measures of bicarbonate and 'buffering capacity' had increased but had not been reflected sufficiently in the all-important performance measure.

### **Stomach irritation**

One possible reason why there has been such conflicting research both recently and earlier is the fact that many subjects suffer short-term stomach complaints after ingesting sodium bicarb. These may take the form of pain, cramping, diarrhoea or a feeling of being bloated. So it's hardly surprising that individuals who feel nauseous do not go out and perform better than they normally do. Thus, some potential benefits of supplementation may be neutralised by the effects of nausea in some subjects, and when the effects are averaged in the scientific trials, ergogenic effects are hidden.

### **A practical approach**

Before using either bicarbonate or citrate supplements, it's wise to check with the governing body of your sport that the substance is not contrary to doping regulations.

The most important practical point is the need to experiment with the supplement during training. Typically, an 800m runner, for example, may perform a time-trial (this should really be with competition to ensure maximum effort) on a particular day after a couple of days of light training. A further couple of days later, after only more light training, he/she can repeat the time-trial in a similar environment after bicarbonate supplementation.

The exact protocol would be to ingest 0.3g of sodium bicarbonate per kg body weight approximately one-to-two hours before the time-trial. That is, for a 66kg runner, consume 20g of sodium bicarbonate (about four teaspoons) – and, yes, the commonly found bicarb of soda is exactly the substance

needed. This experimenting, if repeated several times, should reveal whether bicarb supplementation is likely to produce any benefit and whether the athlete concerned is susceptible to stomach upsets.

It's likely that large individual differences do exist as far as response to supplementation is concerned. It has been suggested that the more highly trained athletes are less likely to benefit from it because their body's natural buffering systems are already so well developed, but so far this is just speculation. It has also been shown that sprinters build up more acidity within their muscles than endurance runners in response to the same exercise, and so may be more likely to benefit from the buffering effect. From the scientific research, it appears that the size of the dose is quite important, and that taking only 0.2g per kg is less likely to be beneficial than 0.3g per kg, although no evidence exists suggesting that an even greater dose is better still.

### **Counteracting the side-effects**

As for the side-effects, the athlete who suffers must try to eliminate them. Drinking up to a litre of water with the dose is often effective and should be carried out as standard. Breaking up the bicarbonate dose into, say, four equal portions taken over the course of an hour may also help. Finally, some researchers have reported that using citrate instead of bicarbonate reduces the incidence of stomach irritation, although the report referred to earlier by Cox and Jenkins unfortunately observed that nausea was experienced by seven out of the eight subjects following citrate consumption, and that five of those seven subjects vomited during exercise. Only one subject vomited during exercise after taking the placebo.

### **Buffering at altitude?**

From the scientific evidence available, it appears that bicarb or citrate supplementation does improve buffering capacity, and thus clearly has the potential to enhance anaerobic performance. However, the responses are likely to vary

between individuals, as will the susceptibility to side-effects. For a competitive athlete in an appropriate sport, experimenting at a personal level should establish whether the supplement is beneficial. As for the scientists, they need to establish whether supplementation is particularly suited to certain types of events more than others. In addition, the potential of regular bicarb supplementation to increase training load as well as one-off performance capability deserves some research attention, as does its potential for athletes who train or compete at altitude, where natural buffering capacity is reduced.

**Alun Williams**





# WHAT THE PAPERS SAY

## **Creatine works best for men...**

Despite the extensive use of creatine monohydrate for performance enhancement, few studies have examined the potential side effects of supplementation and none has directly compared the response to supplementation in men and women.

This gap in knowledge has now been filled by a Canadian study of young, healthy, physically active men and women, which showed no adverse effects of short-term treatment but greater body-building benefits for men than for women.

Fifteen men and 15 women were randomly assigned to five days' supplementation with 20g per day of either creatine monohydrate (CrM) or a similar-looking inactive substance (placebo) after extensive pre-trial checks, including measurement of body composition, blood pressure and maximal strength. On day six they returned to the lab for retesting.

Blood pressure was unaffected by supplementation, and blood tests suggested there were no effects on kidney function. Plasma levels of the muscle enzyme creatine kinase, which is thought to have potentially damaging effects at increased levels, was unaffected by treatment.

As far as body composition was concerned, there was no effect of treatment on percentage body fat but clear increases in both total body mass (TBM) and fat-free mass (FFM). However, these changes were much smaller for women than for men: men increased both TBM and FFM by 2%, while for women the respective increases were only 0.8 and 1.0%. CrM treatment had no effect on grip strength during forearm tests and no significant effect on resting and post-exercise blood lactate levels.

The implication of these findings is that creatine monohydrate may be less useful as a performance-enhancing aid for women than for men. 'It was not anticipated *a priori* that such large sex differences would exist in response to CrM loading, given that the subjects were matched for age and training status,' explain the researchers. They point out that it is possible the women had higher muscle concentrations of total

creatine before supplementation, which might have reduced its effectiveness.

In terms of safety, CrM gets the thumbs-up although the potential side effects of long-term supplementation have yet to be examined. In addition to its benefits for athletes, CrM could also be used to treat people with muscle atrophy and other wasting conditions.

*Med Sci Sports Exerc* 2000 Feb 32 (2) 291-6

**Isabel Walker**

## **...But half of it goes straight down the toilet**

Creatine is one of the most widely-used ergogenic aids, but how do you work out exactly how much creatine you need in order to elicit those reported performance-enhancing properties? Could you be flushing money down the toilet? If the findings of a study carried out in Canada are anything to go by, then the answer is yes – literally!

The dosage required to maximise intramuscular creatine is poorly understood. For years manufacturers have recommended dosages based on limited scientific support and studies of non-athletic, average populations. One popular dosage regime involves a loading phase of 25g per day for the first five-to-seven days followed by a maintenance phase of 5g per day for the remaining supplementation period. The Canadian researchers aimed to find out whether this loading phase was really necessary or simply a clever ploy to shift more product.

A group of 40 sportsmen (20 football players and 20 hockey players, aged 18-25) were assigned to either creatine supplementation or placebo after calculation of their body density, percent body fat and lean body mass. The supplementation group then took creatine monohydrate (0.1g.kg<sup>-1</sup> of lean body mass) dissolved in 500ml of warm grape juice for seven days, while the placebo group took the same amount of grape juice minus the creatine. Urine was collected before and after the supplementation period, during which subjects followed a structured resistance training programme.

So what did the men and women in white coats see when they looked at all that urine? In a nutshell, they found that approximately



half of the supplemented creatine was being excreted in the urine – that's half your money straight down the toilet!

It is well documented that creatine has the greatest effect on subjects with low initial total levels of creatine. And with this in mind, the researchers suggested that the poor retention could in part be due to high levels of stored creatine in the subjects, inhibiting further uptake. However, the research shows that even a meagre dosage of about 7g per day (compared to the 20-25g per day recommended during a loading phase) results in a loss of around 50%.

Clearly these results support the use of smaller dosages and suggest that athletes supplementing their diet with creatine should base their individual servings on their lean body mass, ensuring that each dose does not exceed 0.1g.kg<sup>-1</sup>. Obviously more research is needed and, as always, *Peak Performance* will bring you the latest news.

*Journal Of Strength and Conditioning Research*, vol 15 (1): 59-62

**Nick Grantham**

*“Poor retention could be due to high levels of stored creatine”*

## **Creatine works on inactive muscles as well**

The effects of creatine supplementation on performance during competition and on strength gains during training have been well documented, not least in *Peak Performance*. Creatine delays fatigue and allows higher power outputs to be achieved. Results from a new study led by B. Op't Eijnde at the August Krogh Institute in Denmark show that creatine may even benefit inactive muscles.

Twenty-two subjects volunteered to have their right leg put in a cast for two weeks, during which time half of them took creatine (group C) and the other half a placebo (group P). Before and after immobilisation, maximal knee extension strength, muscle cross-sectional area and muscle creatine-phosphate concentrations were measured.

Both groups showed similar decreases in muscle strength and size over the two weeks of immobilisation. However, during a subsequent 10-week rehabilitation programme, group C showed a greater increase in muscle size and strength returning to pre-immobilisation levels earlier than group P. This was attributed to a decrease in muscle

creatine phosphate concentrations within the muscle fibres for group P whereas group C maintained their levels.

The authors concluded that oral creatine supplementation reduces the functional deterioration of muscles during disuse. Thus creatine could be used as a therapeutic measure to maintain muscle strength for people whose muscles weaken for reasons such as breaking a limb or getting old.

## **Tennis players don't benefit**

The increasing power of the male tennis game, as evidenced in Wimbledon champion Goran Ivanisevic's thundering serves, is unlikely to be explained by creatine loading, if the results of a new study from Belgium – the home of women's runner-up Justine Henin – are to be believed.

The study, involving eight well-trained young male tennis players, was designed to investigate the effect of acute creatine supplementation on stroke quality during simulated match play.

Over the last decade creatine has become one of the most popular nutritional supplements in sports, including tennis. Short-term supplementation has been shown to improve power output during various modes of short all-out sprint exercise, repeated jumping and various types of resistance exercises. Furthermore, some findings suggest that the performance benefits are greatest during short maximal intermittent exercise bouts, of the type which feature in tennis.

Thus, hypothesised the researchers, creatine supplementation might improve stroke performance in tennis by acting directly on arm force and power and/or by enhancing intermittent sprint performance, allowing for better positioning at the time of stroke execution.

In the double-blind study, the subjects were assigned in random order to two six-day experimental protocols, separated by a five-week 'wash-out period'. For the first five days of each protocol the subjects were supplemented either with creatine (20g of creatine monohydrate powder per day) or a similar-tasting placebo (inactive substance). On day six they performed the Leuven Tennis Performance test, which aims to evaluate stroke quality in competition tennis players in match-like

conditions, featuring four games of 10 rallies each.

When the results of the two sets of tests were analysed and compared, the researchers found that creatine loading did not significantly impact on either the power or precision of the subjects' strokes. 'Thus,' they conclude, 'the current data clearly demonstrate that there is no performance benefit of acute high-dose creatine supplementation in elite tennis players.'

However, they do not rule out the possibility that long-term creatine intake, with its stimulatory effect on muscle growth, might still be a factor in enhancing stroke quality and sprint power in the game.

*Int J Sports Med* 2001 Jan 22(1) p76-80

**Isabel Walker**

## **Low dosages may work as well**

Supplementation with creatine has been shown to increase muscle levels of phospho-creatine – the compound used by the body to replenish energy during high-intensity explosive exercise. A large number of studies have investigated the relationship between creatine supplementation and subsequent exercise performance, and many of these have demonstrated a positive impact on performance in events requiring explosive power, such as sprinting.

Boosting muscle levels of phospho-creatine involves an initial loading phase of 20g of creatine monohydrate per day for five days. This is followed by a 'maintenance' phase, when a dosage of 5-10g per day is taken to maintain muscle phospho-creatine at the new levels. However, the problem with supplementation at these dosages is that it can cause side effects such as muscle cramps, diarrhoea and other gastrointestinal problems in some people.

Is it possible that continued low-dose supplementation might produce similar performance benefits with fewer side effects? A new study investigating this hypothesis has produced interesting results. A group of 47 male university athletes were treated with average doses of 7.7g of creatine over a 20-day period in combination with a resistance training programme, and the researchers were able to demonstrate greater increases in peak power, peak force and lean

body mass than those observed in a control group treated with placebo.

Although there is a clear need for further research into different loading regimens, these encouraging results suggest that it may be possible to gain significant improvements in performance on lower dosages than are currently recommended. We will continue to report on further developments, so watch this space!

*International Journal of Sports Nutrition and Exercise Metabolism*  
2000 Sep 10 (3):235-244

**Ian Carlton**

## **Creatine works for trained sprinters**

Much of the research on creatine has been carried out on physically active subjects, but there has been relatively little information about the effects of supplementation on highly trained athletes.

Now a group of Norwegian researchers has moved to correct the balance with a study of 18 well-trained male sprinters at local competition level, whose performances were observed to improve significantly after five days of supplementation with high-dose creatine.

During the previous two years a substantial part of these athletes' training had consisted of a series of maximal sprints with short rest periods to improve their fatigue resistance. For the study they were split into two groups of nine, one group taking 20g of creatine daily and the other a placebo preparation for a period of five days. Before and after supplementation they completed one 100m sprint and a series of six intermittent 60m sprints starting every 50secs.

### **Key results were as follows:**

- Only one of the sprinters was able to identify correctly whether he had received creatine or placebo;
- Although no significant body weight changes were seen in the placebo group, a significant increase of 0.6kg was seen in the creatine group;
- Blood creatine levels were unchanged in the placebo group but increased significantly in the creatine group;

*‘It may be possible to gain significant improvements in performance on lower dosages’*

- The 100m sprint times and 60m sprint times were not statistically different after supplementation with placebo. But creatine supplementation resulted in significant improvements, reducing the 100m sprint time from 11.68 to 11.59secs and reducing the total time of the six intermittent 60m sprints from 45.63 to 45.12secs.

'An increased performance could be a result of the increased amount of PCr [creatine phosphate] available in skeletal muscle, since the amount of PCr is one of the most likely limitations to muscle performance during brief, high power exercise,' suggest the researchers. 'Increased PCr in skeletal muscle may delay the depletion of PCr stores and maintain ATP turnover rate, suggesting increased energy availability during heavy exercise.'

The benefit of an improvement in 100m sprint time is self-evident, as the researchers point out, but the value of improved intermittent six times 60m sprint times is less obvious. 'It may suggest that each training component can be performed at a higher quality level and thus Cr supplementation may improve the quality of training, leading to greater gains in sprint performance.'

From the sprinter's perspective, the news about creatine looks good. However, the researchers take a cautious view that, although there have been no reports of serious side effects with creatine supplementation, further investigation of the effects of long-term use should now be carried out.

*Scand J Med Sci Sports* 2001 Apr 11(2), pp96-102

**Isabel Walker**

*‘From the sprinter’s perspective, the news about creatine looks good’*

## **Now school kids are creatine users**

A disturbing trend towards creatine use by schoolchildren has been uncovered by a major US study of middle and high school athletes. Of 1,103 athletes aged 10-18 taking part in a confidential survey, 62 (5.6%) admitted taking creatine. Use was reported among all age groups, although it was greatest among athletes in the highest grade

(grade 12), of whom 44% reported using creatine.

Creatine use was significantly more common among boys (8.8%) than girls (1.8%). And, although it was taken by participants in every sport – including tennis and cheerleading – its use was significantly more common among football players, wrestlers, hockey players, gymnasts and lacrosse players.

The most common reasons cited for taking creatine were enhanced performance (74.2% of users) and improved appearance (61.3%), while the most common reason for not taking creatine was safety (45.7% of non-users). Of the five schools sampled, creatine use was most prevalent in the private high school, suggesting a connection with family income.

Should we be concerned by this trend? Undoubtedly, say the paediatricians who organised the study, for the following reasons:

- the safety of creatine use in under-18s has not been demonstrated and the short and long-term health risks in adolescents and pre-adolescents are unknown;
- use of performance-enhancing nutritional supplements could set children and young adults on a path leading to use of dangerous and banned substances, like anabolic steroids.

So why are children as young as 10 using creatine? First, say the authors, creatine, in common with other nutritional supplements, is touted as a safe and natural method of improving performance, which is widely marketed and readily available; secondly, teenagers may be taking ergogenic aids to imitate famous athletes; finally, young athletes are under increased pressure from parents, coaches and peers to succeed in athletics, and the win-at-all-costs mentality has emerged in youth sports.

They conclude: 'Open communication is needed among a team of physicians, nurses, coaches and athletic trainers, all of whom are responsible for the health and safety of young athletes. Until more information is available about safety in children and adolescents, health care providers should actively discourage use of creatine in their young patients.'

*Paediatrics* 2001 Aug 108(2), pp421-425

**Isabel Walker**

## Can creatine work for younger performers?

Creatine is extremely popular with adult athletes, many of whom believe it gives them a performance-enhancing boost. But does creatine offer any ergogenic benefits to young performers? A group of sports scientists based at the University of San Francisco have examined all the available research in a bid to establish a rationale for creatine supplementation in child and adolescent athletes.

The main argument for the use of creatine in this age group is that children struggle to use and reproduce creatine phosphate and ATP effectively, so limiting their ability to regenerate high-energy phosphates during exercise. Creatine supplementation, it is suggested, could help children improve their performances in high-intensity exercise. However, there is a lack of compelling evidence to support this theory and a number of arguments against it. Here are the main ones:

- children are not mini adults and have a greater reliance on aerobic rather than anaerobic metabolism. If the goal of creatine supplementation is to enhance anaerobic metabolism, it would therefore have a limited effect;
- adolescents appear able to regenerate high-energy phosphates during high-intensity exercise and improve performance in short-term high-intensity exercise through training, therefore reducing the need for supplementation;
- performance during growth tends to be limited by mechanical factors rather than by the relative contribution of the aerobic and anaerobic energy systems;
- the long-term safety and efficacy of creatine supplementation has not been established in children and adolescents.

However, the arguments for and against creatine supplementation in children and adolescents are derived from an extremely limited number of studies. A significant amount of research is needed to enable us to fully understand the metabolic changes that accompany growth before we can even start to consider the efficacy and safety of creatine supplementation. With this in mind the research team concluded that there was insufficient evidence to support the use of creatine by

children and adolescents.

*The Journal of Strength and Conditioning Research*, vol 15, no 4, 524-528

**Nick Grantham**

## **Sedentary elderly gain most from creatine supplementation**

Can creatine stave off the reduction in muscle performance that accompanies ageing? That is the question a team of French researchers set out to answer with a study examining the effects of short-term doses of oral creatine on healthy elderly men.

Forty-two healthy volunteers were divided into three groups, as follows:

1. 14 elderly sedentary men with a mean age of 70.1 years;
2. 14 elderly trained cyclists (mean age 66.4 years);
3. 14 young sedentary men (mean age 26).

Half the people in each group were treated with creatine (three 5g doses per day) and the other half with placebo for five days. Before and after the study period all the subjects performed five all-out 10-second sprints on a cycle ergometer, separated by 60-second intervals of passive recovery. Power output, work performance and heart rate data were recorded during each sprint.

Those treated with creatine in both sedentary groups showed significant improvements in maximal power and work performed in the subsequent tests compared with those given placebo. But no significant change in pedalling performances was seen in the trained elderly subjects. However, power outputs were always greater in the trained than the sedentary groups, confirming the difference in fitness between them.

‘Our study suggests,’ conclude the authors, ‘that creatine given by mouth increases the anaerobic power and work capacity of sedentary people of different ages during maximal pedalling tasks. However, the level of physical activity seems to be a determinant of the ergogenic effect of creatine in older subjects.’

These results accord with those of a number of studies on younger



adults, with an ergogenic effect generally reported in untrained subjects not always replicated in highly trained or élite subjects.

*Eur J Appl Physiol* 2001 Jun 84(6), pp533-9

**Isabel Walker**

## **Notes**

## **Notes**

## **Notes**

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# PEAK PERFORMANCE

The research newsletter on  
stamina, strength and fitness

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