ANTIOXIDANTS:
train longer, train harder

A SPECIAL REPORT FROM

PEAK PERFORMANCE

The research newsletter on stamina, strength and fitness

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From the editor

Everybody's heard of antioxidants. Most people know that they're good for us, and also that we should be consuming more in our diet. But what exactly are they and how do they work in the body? More importantly, can sportsmen and women benefit from improving their antioxidant nutrition and if so how? And what does the very latest research tell us about the benefits or otherwise of antioxidants? These are some of the questions that this special report aims to address.

The story of antioxidant nutrition in sport to date is a fascinating one, but has also been far from straightforward. Early studies suggested that large doses of antioxidant vitamins and minerals could help performance and protect the body, and the assumption was that more was better. However, later studies cast doubt on these initial findings; indeed, some studies even appeared to indicate that high dose single antioxidant supplementation could be detrimental!

More recently, the pendulum of evidence has swung very much in favour of enhancing antioxidant nutrition in athletes, but the thinking is now far more subtle and complex. The right amounts of the right antioxidants appear to enhance recovery, reduce post-exercise soreness and protect the body at the cellular level. There's even some (very new) evidence of an acute performance boosting effect. However, not all antioxidants are created equal and resorting to supplementation may not be the answer either.

This report is designed to separate the antioxidant facts from the fiction and arm you with the latest knowledge to improve your own antioxidant status for improved short-term sport recovery and long-term sport performance. There's another bonus too; follow even a few of the recommendations here and you'll also be making the best possible nutritional investment in your longevity and long-term health!

Andrew Hamilton BSc Hons MRSC ACSM

Understanding antioxidants

It's a funny old world. Just when you think you've got things sussed, along comes a theory that turns everything upside down. Take exercise for example. Study after study has conclusively demonstrated that aerobic training not only enhances oxygen uptake, transport and utilisation (great for athletes seeking enhanced endurance), but that it also effectively lowers body fat levels, reduces/normalises blood pressure and cholesterol, reduces the risks of strokes and heart disease and may even (through its body fat lowering effect) reduce the risk of certain types of cancers.

At a Glance

- The relationship between oxygen, free radicals and antioxidants is discussed;
- The antioxidant defence systems in the body are outlined;
- The importance of maintaining a healthy antioxidant status f or athletes is explained.

However, more recent research suggests that while efficient oxygen transport and utilisation holds the key to energy availability in sports where endurance plays any kind of role, oxygen is the number one suspect in a range of biological crimes against the body. These range from the aging process in general, to autoimmune diseases, Alzheimer's disease, cancer and numerous other degenerative conditions. Fortunately, Nature has armed us with superb defences against this oxygen-induced havoc – antioxidants!

Antioxidant protection

To understand how antioxidants protect us, we first have to understand a little about oxygen metabolism. Oxygen is amazing stuff. The fact that there's so much of it around (about 20%) in the air we breathe is even more remarkable, because oxygen is such a chemically reactive substance, reacting with virtually every other element in Nature (often explosively).

'Although it's essential for life, oxygen is very much a double-edged sword, playing a major role in our ultimate demise!'

The reason our atmosphere contains such a high concentration of oxygen gas is down to the process of photosynthesis in plants, whereby stored energy in the form of carbohydrates is made by combining water and carbon dioxide in the presence of sunlight with oxygen gas continually produced as a by-product.

Nature has taken advantage of this freely available reactive oxygen by allowing the evolution of organisms that derive their energy for life from aerobic metabolism – ie. using oxygen to produce energy. All animals, including humans, use aerobic metabolism to produce energy to fuel the body by combining highly reactive oxygen gas with fats, proteins or carbohydrates. However the very same reactivity of oxygen that allows us to produce this energy can also, if unchecked, wreak havoc within our cells. Although it's essential for life, oxygen is very much a double-edged sword, playing a major role in our ultimate demise!

Oxygen – the double-edged sword

The key to understanding the 'double-edged sword' nature of oxygen lies buried in its atomic and molecular structure. Without delving into the chemistry too deeply, it's important to appreciate that in most biochemical reactions, electrons in atoms are moved around to form chemical bonds in pairs. That's because when paired, electrons are 'stable'. However when electrons attached to an atom are solitary, they behave extremely reactively and are 'unstable'. Atoms with single electrons are often referred to as free radicals; these free radicals are 'electron hungry' and are desperate to grab an electron from somewhere else in order to pair up and become more stable again.

The problem is that free radicals are so reactive that they'll readily rip away an electron from another electron pair in a nearby chemical bond, which not only destroys that bond, but also creates another unpaired electron (ie a new free radical). Now of course this newly created free radical is desperate to pick up an electron from wherever it can and the process of ripping away an electron from yet another chemical bond to form a pair is repeated, now leading to a 3rd radical being produced.

'The important thing about free radicals is the trail of damage they leave behind in the cell.'

What follows is an extremely quick chain reaction, where this process of bond-breaking and free radical formation happens over and over again resulting in a string of broken and disrupted chemical bonds and potential biological havoc! Each individual free radical in that chain reaction has only a very fleeting existence perhaps lasting for just a billionth of second before snatching back an electron from another chemical bond; for this reason, you could never go and collect a 'bottle of free radicals'!

However, the important thing about free radicals is the trail of damage they leave behind in the cell. If electrons are being ripped out of chemical bonds that hold together things like cell walls or DNA, irreparable damage to the cell and/or its genetic material may be caused. This accumulated damage is now thought to be one of the root causes of degenerative diseases and the ageing process generally.

So where does oxygen figure in all this? Well, the oxygen molecule contains a pair of electrons that consists of two highly reactive electrons that have loosely paired up because chemically speaking, there's 'nothing better on offer'! In fact, it's these electrons that give oxygen its exceptional reactivity. However, given half a chance, these two highly reactive electrons would much rather go and do their own thing by grabbing their own partner electrons from other chemical bonds thus starting a whole sequence of free radical chain reactions.

In other words although they behave as a stable pair when alone in an oxygen molecule, they behave much more like free radicals in the presence of other molecules – for example during aerobic metabolism. This means that the same chemical

properties that give oxygen the reactivity needed to produce energy (when combined with carbohydrates, fats and proteins in our cells) has a massive downside; aerobic metabolism also floods our cells with billions upon billions of potentially destructive oxygen free radicals, which can potentially wreak huge damage if left unchecked.

Free radical defences

'These defence systems are designed to rapidly neutralise or 'quench' oxygen-generated free radicals.'

The good news is that the human body has an array of sophisticated defences to minimise the free radical damage that inevitably results as a consequence of oxygen metabolism (as well as repair enzymes whose job it is to recognise damaged DNA etc. and repair it). These defence systems are designed to rapidly neutralise or 'quench' oxygen-generated free radicals, which then breaks the chain reaction and ensures that any damage caused to cell membranes, DNA etc. is minimised. Because of this reason, they are commonly known as 'antioxidant' defence systems.

There are three main types of antioxidant defence systems in the body; antioxidant enzyme systems, antioxidant nutrients (vitamins and minerals) and antioxidant phytochemicals (see box). All of these systems have one thing in common – they are able to donate a single electron to a free radical, which effectively quenches that radical and renders it harmless *(see figure 1)*.

But surely by doing that, the antioxidant would itself be converted into a free radical I hear you ask? Well, technically that's true. However, antioxidants possess a very special molecular structure, which means they can donate a single

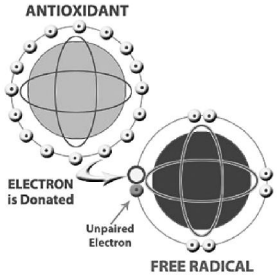

Figure 1: the antioxidant molecule can easily donate an electron to the free radical, so that the free radical's unpaired electron becomes paired. This effectively quenches and deactivates the free radical. Meanwhile, although the antioxidant is now missing an electron, its molecular structure enables it to remain stable; thus the free radical chain reaction that can wreak havoc in the body is stopped in its tracks.

Fantastic defences

Nature has equipped us with fantastic defences against oxidative free radical damage and there are three main types:

Antioxidant enzyme systems – these are enzymes assembled by the body (often containing one or more nutrient) with the ability to rapidly 'quench' or stabilise free radicals, thus breaking the chain reaction of destruction. Examples include glutathione peroxidase (containing selenium) and superoxide dismutase(containing copper and/or manganese). Different enzyme systems target different types of free radicals.

Antioxidant nutrients – these are nutrients required by the body that also act directly as antioxidants by soaking up or quenching a free radical. These include beta-carotene (vitamin A), vitamin C, vitamin E selenium and zinc. As above, different nutrients tend to target different types of free radicals.

Phytochemicals – over recent years, there has been an explosion of research into naturally occurring substances in plants (often responsible for giving the plant its characteristic colours and flavours) called phytochemicals. Many of these display remarkable antioxidant capacities, sometimes tens or even hundreds of times greater than the antioxidant nutrients. The range of compounds is enormous and includes the carotenoid family, the flavenoid family, the tocotrienol family (this includes natural vitamin E) and a number of sulphur containing compounds such as sulphorane in broccoli and allicin in garlic. The antioxidant phytochemicals found in fruits and vegetables are believed to explain why those who consume large amounts of these foods are far less likely to develop degenerative diseases such as cancer. Recent studies with foods rich in these phytochemicals have clearly demonstrated their protective effects against oxidative stress (free radical activity as a result of oxygen metabolism), and there's also evidence that they may actually enhance athletic performance and recovery.

electron and yet remain chemically stable even though they now contain an unpaired electron. This is because the energy of that unpaired electron becomes 'smeared' all over the antioxidant molecule, which effectively dilutes and contains it. Think of taking the hyperactive bull out of the china shop and putting him in a very large well-contained field with padded fences and you'll get the general idea!

Free radical battleground

As stated above, oxygen provides us with the energy for respiration and life. However, your body is battling constantly to contain the damage caused by the inevitable generation of free radicals as a result of oxygen metabolism. Table 1, below, lists some of the key players on both sides of the frontline:

Table 1: the free radical battleground within

The Free Radical Attackers	The Cellular Defenders	
R = fragment of bigger molecule, ” = unpaired electron H = hydrogen atom, O = oxygen atom	Nutrients	Enzymes
The hydroxyl radical – HO”	Vitamin C	
The alkoxyl radical – RO”	Vitamin C	
The peroxyl radical – ROO”	Vitamin E, beta-carotene	Glutathione peroxidase (selenium dependent)
The hydroperoxide molecule – ROOH (falls apart to form RO” and HO”)	Vitamin C	
The alkyl radical – R”	Vitamins A, C and E	
The superoxide radical ion – O_2-”	Vitamin C	Superoxide dismutase (copper manganese and zinc dependent), catalase (iron dependent)
The peroxide ion – O_2^2-	Vitamin E	Glutathione peroxidase
Singlet Oxygen – O_2* (excited state)	Vitamin A (beta-carotene), vitamin C	

How is antioxidant status assessed?

In most early studies, scientists simply looked at the tissue levels of certain antioxidants to see the effect of eating certain foods, exercise or taking antioxidant supplements. While these kinds of measurements are still important, far more useful is to try and see whether those antioxidants in the body are actually providing extra protection against free radical damage and oxidative stress.

To do this, scientists try to look for tell-tale signs or ‘markers’

Table 2: measuring antioxidant status and free radical damage

What they look for	What it tells them
Malondialdehyde (MDA)	How much damage has been done to the lipids in cell walls and to a lesser extent a marker of cell protein damage.
8-hydroxy-2-deoxyguanosine (OH8dG)	The amount of damage done to cell DNA by free radical attack on the nucleotide base deoxyguanosine. The most frequently used marker of oxidative stress.
5-hydroxy-2'-deoxycytidine (5-OH-dCyd)	As above but measures damage to the base cytidine.
Conjugated Dienes (CD)	A measure of damage to unsaturated lipids present in our cells.
Isoprostanes	A marker that seems to not only indicate the background sum of a number of different oxidative stressors, but also directly and adversely affects certain biochemical pathways in the body once it has been formed.
Ascorbic acid/ Dehydroascorbic acid ratio	When vitamin C (ascorbic acid) mops up radicals in the body, it is converted to dehydroascorbic acid. The more the ratio is shifted away from ascorbic acid to the oxidised form dehydroascorbic acid, the greater the degree of oxidative stress.
CoQ10H2/CoQ10 ratio	The enzyme CoEnzyme Q10H2 is easily converted to the oxidised form CoQ10 during oxidative stress. As above, the ratio between the two forms of this enzyme indicates the degree of oxidative stress.

of damage caused to our cells by oxidative stress and also to examine how this changes during exercise or dietary manipulation (see table for details). If giving extra amounts of an antioxidant actually provides extra protection, the amount of free radical damage should drop *ie* a reduced level of markers of damage should result. Some of the most commonly employed tests are shown in table 2, above:

Why do antioxidants matter to athletes?

At this point, you might be wondering why antioxidant nutrition is important for athletes? Let's not mince words here; what makes you different from the ranks of couch potatoes out there is that you train, and probably train hard! And where does all the energy to fuel your training come

from? That's right – by combining dietary carbohydrate and fat with oxygen. In short, the greater the volume and intensity of training, the more oxygen metabolism takes place; an elite endurance athlete can easily consume 4-5 times as much oxygen per day as a sedentary office worker!

Although with training your body's antioxidant defence systems do become more efficient at containing oxygen-induced free radical damage, evidence is accumulating that athletes may benefit from increasing their dietary antioxidant intake in a number of ways (discussed at length in the following articles in this report):

- Reduced post-exercise muscle soreness
- Reduced inflammation and joint stiffness
- Enhanced performance and recovery

'It's a well-established fact that a chronically low antioxidant intake is associated with increased risks of developing a number of degenerative diseases and will do nothing to slow the aging process.'

Then of course, there's the issue of the general protection that antioxidants afford. Although it's true that we need more definitive evidence as to whether enhanced antioxidant intake can improve sport performance in the short term, it's a well-established fact that a chronically low antioxidant intake is associated with increased risks of developing a number of degenerative diseases and will do nothing to slow the aging process.

Most sportsmen and women tend to think a season or two ahead, but don't consider how they'll be performing in ten or fifteen years time. However, in some sports, it may take that long to reach your full potential; the question is could you arrive at that point with less wear and tear and accumulated damage to the body by enhancing your antioxidant intake? The long-term nature of antioxidant protection means that it's too early to give a definitive answer, but much of the evidence to date seems to point in that direction. Beyond that, many of you reading this will no doubt have aspirations to remain fit and active for years and decades to come; all the evidence suggests that enhancing your dietary antioxidant could be the very best way to ensure those aspirations are fulfilled!

Andrew Hamilton

Jargon buster

DNA – Deoxyribonucleic acid; a large double helix molecule found in the nucleus of every cell, which contains the genetic blueprint' of the cell and instructions for cell division and growth

Nucleotide base –the fundamental building blocks of DNA

Electrons – negatively charged sub-atomic particles orbiting atoms and which can form chemical bonds to other atoms to make molecules

Enzymes – large protein molecules that speed up essential biochemical reactions in the body, which would otherwise either occur too slowly to sustain life or not at all

Optimum nutrition for athletes – what can we learn from recent studies?

Ask sportsmen and women what they think are the key issues in sports nutrition and the importance of optimising carbohydrate intake, using protein to aid lean tissue growth, or specific ergogenic aids like creatine will probably feature in their responses. But, according to Andrew Hamilton, there's evidence that many athletes may be neglecting the dietary basics – bad news for maintaining a healthy antioxidant status...

At a Glance

- Evidence is presented that some athletes may be neglecting dietary basics;
- An increased knowledge of basic nutrition does not always guarantee improved dietary habits;
- Athletes who seek nutritional assessments to improve basic diet are offered guidance on how to select a nutritionist;
- The most common nutrient deficiencies and strategies to avoid them are presented, together with food recommendations to avoid them.

Punch 'sports nutrition' into any internet search engine and you'll get a deluge of results from sports supplement companies and retailers, all offering products claiming to enhance sport performance. Likewise, the shelves of most sports and health food retailers are crammed to overflowing with 'sports nutrition' products – an Aladdin's cave of pills, powders and potions!

But while there have been great technological advances in sports nutrition products, these types of products do not

automatically guarantee optimum nutrition for athletes in hard training. Indeed, a reliance on products such as carbohydrate and protein powders, fluid replacement drinks and ergogenic aids may help to foster a 'performance from a bottle' mentality – an assumption that today's high-tech sports nutrition formulations can guarantee optimum performance.

'A reliance on products such as carbohydrate and protein powders, fluid replacement drinks and ergogenic aids may help to foster a 'performance from a bottle' mentality.'

However, this assumption is flawed. Relying heavily on sports nutrition products may actually lead to a poorer basic diet quality, because many athletes simply assume that they no longer need to worry about eating high quality natural foods, leading to a reduced intake of key nutrients. A poorer, low-nutrient diet is undesirable for a number of reasons, but particularly because such diets are associated with lowered immunity and a generally reduced resilience of the body to withstand the day-in, day-out rigours and cumulative stresses of training.

Supplementation is no universal panacea either. Even the most advanced nutrient supplement in the world will contain only a minute fraction of the vast number of naturally occurring beneficial substances that are continually being discovered in food – particularly antioxidant substances that may help to keep athletes healthier, thereby minimising time lost through illness and injury.

Athletes often lack nutritional knowledge

It's a generally accepted theory of sports nutrition that athletes don't need to supplement nutrients because their increased energy expenditure means they eat more food to fuel activity. Provided this extra food also contains nutrients, the net result of a higher calorie intake should be a greater overall nutrient intake.

For example, suppose a male sedentary office worker weighing 70kg consumes about 1,800kcals per day and the food that provides those calories supplies around the **RNI** of 1.4mg of vitamin B6. Now suppose he begins a training programme, averaging 10km of running per day. Assuming the energy cost of running is about 1 kilocalorie per kilogram of body mass per kilometre, our newly active office worker

will need to consume around 700kcals extra per day (*ie* 2,500kcals in total) to fuel this activity. If he continued to eat exactly the same foods, but simply increased portion sizes to meet this increased energy demand, we would expect a proportional rise in his vitamin B6 intake from 1.4 to 1.95mg per day (25/18 x 1.4mg) – more than enough to meet any increased metabolic need.

However, while this theory is broadly true, there are three implicit assumptions:

1. The basic quality of the diet is nutritionally balanced, supplying sufficient amounts of key nutrients *ie* vitamins, minerals and naturally occurring antioxidants (from fresh fruits and vegetables);
2. The 'extra' calories consumed to fuel training are also derived from nutritious foods and not from 'empty calories' (*eg* sugary foods such as confectionary, junk food and fizzy drinks);
3. An athlete has no special needs – *eg* female athletes undertaking high-impact sports such as running and who are now thought to have significantly higher iron requirements.

While these assumptions seem perfectly reasonable, it's surprising how many athletes appear to lack the knowledge required to help them make healthy food choices. For example, a study carried out in the early 1990s evaluated the nutrition knowledge and dietary practices of four groups of women [(1)]:

- 18 postmenopausal women;
- 14 college-aged dancers;
- 13 members of a college track team;
- 14 non-athletic college women.

All the subjects completed a personal information questionnaire, a 24-hour food recall, a food frequency questionnaire and a nutrition knowledge test. Although the track athletes scored significantly higher in the knowledge test than the dancers (26.5 vs 22.2), they scored less well than

Challenges of assessing nutritional status

Assessing nutritional status accurately and drawing firm conclusions is no easy task. For example, studies that rely on athletes 'self-reporting' their dietary intake are beset with potential problems; it's a well-known phenomenon that what we eat and what we *think* we eat may not be quite the same! There may also be occasions where an athlete deliberately over-or underreports the intake of a particular food or food group, for example to keep the coach happy, or perhaps as a result of an eating disorder.

These difficulties can be minimised if any assessment is also supplemented by biochemical testing for nutrient status; *ie* as well as examining dietary patterns to see whether there are likely to be nutrient shortfalls, you then measure nutrient levels in your subjects to see if these are actually borne out in practice. If a nutrient shortfall is detected by both criteria, you can be pretty sure that an athlete really is short of that nutrient. However, studies that also include biochemical testing (as opposed to simply dietary screening) are time-consuming and expensive, and are therefore less numerous in the scientific literature than simple dietary screening studies.

Another complicating factor is the fact that many studies assess groups of athletes who train together, *eg* from a squad, team or club. These athletes will almost certainly be comparing notes, swapping experiences and taking advice not only from the coach, but also from each other. This inevitably means that there'll be some degree of sharing of nutritional approaches and practices, which in turn means that the results of any study carried out on a group may be only particularly relevant to that group and not more generally. For example, a study on a group of swimmers drawn from a squad may reveal a dietary shortfall of calcium, but it could be that a couple of the stronger personalities in that squad had persuaded the other squad members that avoiding dairy produce (a rich source of calcium) would help performance. Given this scenario, it would obviously be a nonsense to draw the conclusion that swimmers generally are at risk of a calcium deficiency.

the postmenopausal women (28.5) and the non-athletic college women (29.7).

A much more recent study compared the nutrition knowledge and dietary composition of Italian athletes and non-athletes [2]; 60 athletic and 59 non-athletic adolescent

females completed 3-day food recall and nutrition knowledge questionnaires. Although the athletes reported higher carbohydrate, iron and fibre intakes and consumed lower fat, the intakes of calcium, iron, and zinc were still less than the recommended dietary allowance for these nutrients. Furthermore, while the athletes gave a slightly higher rate of correct answers on the nutrition knowledge questionnaire than non-athletes, the difference was quite small (77.6% vs 71.6%).

Another recent study examined the dietary practices, attitudes and physiological status of collegiate freshman football players in Atlanta and reported that over half the group believed that protein supplements were necessary for muscle growth and development, and that protein was the primary source of energy for muscle [3].

'Many athletes are still confused about what constitutes a healthy and nutritious diet required to support training.'

It's clear from these and other studies that while the age, type of sport and cultural/social background of athletes plays an important role in determining the quality of diet, many athletes are still confused about what constitutes a healthy and nutritious diet required to support training. There's also evidence that even when athletes are more knowledgeable about and have more positive attitudes towards, nutrition, they still fail to follow it up with improved dietary practice [4].

Evidence for sub-optimum nutrition in athletes

Do the poor nutritional knowledge and practices observed in some studies translate into sub-optimum dietary intakes in practising athletes? This is a complicated issue and the answer depends very much on which studies and which groups of athletes you look at (*see box, overleaf*).

As you might expect, many studies on elite athletes (who often have access to specialist dietary advice and even a dedicated dietician) have not demonstrated wholesale dietary deficiencies. For example, a US study carried out on 19 elite female heptathletes (average age 26 years) combined data on body composition, dietary nutrient intake, dietary practices and biochemical indices of iron status during training [5]. Apart from vitamin E, average nutrient intakes were over 67% of the

Common dietary pitfalls

Although different dietary patterns produce a huge amount of inter-individual variability in nutritional status, a few common pitfalls are frequently observed. The table below shows some of the most common nutrient deficiencies observed in athletes, their possible causes and good food sources that can help improve the status of that nutrient.

Nutrient	Important function(s) for athletes	Causes of/risk factors for shortfall	Good food sources
Calcium	Bone growth/maintenance; muscular contraction; nervous system and hormonal signalling	Calorie restriction (dieting); diets low in dairy produce (the richest source of calcium)	Milk, cheese, yoghurt, tinned fish (with bones), green leafy vegetables, nuts and seeds
Iron	Haemoglobinproduction for oxygen-carrying red cells; enzymes involved in energy metabolism; immunity	Low-meat or vegetarian/ vegan diets; large intakes of tea with meals (which contains iron-blocking tannins); pounding sports such as distance running; monthly blood losses in pre-menopausal women	All red meats such as beef, liver, kidney; shellfish, sardines, eggs, prunes, sunflower seeds
Zinc	Protein turnover and muscle growth; immunity; metabolism during 'stress'	Low-protein diets; stress; excessive alcohol consumption; refined/ unk diets	Beef, eggs, herrings, pork, oysters, almonds, brazil nuts, pumpkin seeds, walnuts, whole-grain/wholemeal breads and cereals
Magnesium	Energy production via ATP; carbohydrate metabolism; nervous system and muscular function	Refined diets or those low in wholegrain cereals, green vegetables, pulses and uts and seeds; excessive alcohol consumption;	Almonds ,brazil nuts, dark green vegetables, buckwheat flour, peanuts, chickpeas, sesame seeds, beans and lentils, wholegrain cereals
Vitamin C	Immune function; connective tissue (ligaments, tendons) integrity; aid to iron absorption; long-term protective effect (protection against oxidative stress)	Low fruit and vegetable ntake; stressors such as illness, injury, or psychological stress	Blackberries, broccoli, Brussels sprouts, kiwi fruit, cabbage, limes, lemons, peppers, new potatoes, oranges, grapefruits, tomatoes, strawberries, watercress
Essential fats	Hormonal synthesis; energy regulation (via insulin); possible role in fat metabolism and weight control	Low-fat diets, high saturated or processed fat diets, excessive alcohol intake; contraceptive pill	Fatty fish (trout, salmon, sardines, mackerel, pilchards, herrings), some nuts (eg walnuts), sunflower and pumpkin seeds, wheatgerm and wholegrains, some margarines

Nutrient	Important function(s) for athletes	Causes of/risk factors for shortfall	Good food sources
Folic acid	Regulation of the formation of red blood cells; helps iron to function properly in the body; cell growth and turnover	Low-calorie diets, or diets rich in fast/junk food; low vegetable intake; regular use of antibiotics or anti-inflammatory medication	Spinach, dark leafy greens, asparagus, turnip, Brussels sprouts, beans, liver, brewer's yeast, root vegetables, wholegrain breads and cereals, wheatgerm, oysters, salmon, orange juice, avocado and milk

recommended intake and most **nutrient densities** were higher than the recommended densities for women in this age group. However, there are plenty of other studies suggesting that athletes from a wide range of sporting disciplines and levels may be at risk from sub-optimum nutrient intakes as a result of either a poor understanding or inadequate execution of the basic dietary principles.

For example, a study examined the nutritional status of eight Brazilian male elite swimmers by means of a 4-day food record, a fasting blood sample and body composition measurements [6]. Although the dietary assessment showed a generally adequate intake of calories, vitamins and minerals, only half the group were consuming the recommended daily intake of calcium. Moreover, carbohydrate intakes were insufficient, resulting in high levels of an **enzyme** called creatine-kinase – a sure sign of increased muscle degradation.

More evidence is available from other studies. A study of female judo athletes examined the nutritional status, iron-deficiency-related indices and immunity of female athletes [7]. The subjects' 3-day food records were evaluated for a range of nutrient intakes and compared to a group of controls. Although intakes of energy, protein, phosphate, vitamin B1 and niacin were higher in the judo athletes than the controls, intakes of the minerals iron and calcium still failed to meet the recommended daily allowance. In addition, levels of immunoglobulin (Ig)G (a blood protein that can be used as a marker of immunity – higher levels indicate better immunity)

indicated slight immunosuppression in the athletes – those with the lowest dietary intakes of iron, B1 and niacin exhibiting the lowest (Ig)G levels.

Meanwhile, a Spanish study looked at the magnesium, zinc and copper status of 78 women involved in karate, handball, basketball and running[8]. A 7-day food intake was gathered with all subjects weighing food portions to improve the accuracy of the data and the results compared with those of a control group of 65 sedentary women. The results were not encouraging; although better than the controls, no group of female athletes reached the then recommended daily intake for magnesium (280mg) or zinc (12mg), intakes that have since been revised upwards. Moreover, the handball athletes also failed to meet their daily needs for copper.

'The current consensus among nutritionists and health promotion agencies is that no more than 10% of calorie intake should come from refined sugars, not least because these sources of carbohydrate tend to be very low in vital nutrients.'

A US study on elite figure skaters used 3-day food records to look at the energy and **macronutrient** intakes of 80 males and 81 females taking part in a series of training camps held in Colorado between 1988 and 1995[9]. Of particular interest was what proportion of their carbohydrate intake was consumed as complex, unrefined (nutrient-rich) carbohydrate, and what proportion as refined simple carbohydrate (*ie* sugars). Worryingly, the skaters derived in excess of 25% of their daily energy intake from sugars (typically around 100-142g of sugars per day). The current consensus among nutritionists and health promotion agencies is that no more than 10% of calorie intake should come from refined sugars, not least because these sources of carbohydrate tend to be very low in vital nutrients.

Another study in the late 1980s at the University of Alabama studied eight highly trained female cyclists and also found their diets wanting[10]. Each cyclist kept a 3-day weighed food record and diets were analysed for nutrient content, while blood tests were also carried out. The results were far from ideal; not only were the cyclists' diets low in carbohydrate, mean daily dietary intakes were also well below the RNIs for folic acid (76% RNI), magnesium (81% RNI), iron (59% RNI), and zinc (48% RNI). In addition, more than one-third

of the cyclists failed to consume even two thirds of the RNI for vitamins B6, B12, E and the minerals magnesium, iron, and zinc. The researchers noted that foods such as meats, poultry, fish, beans, peas and nuts were low or virtually absent from many of these cyclists' diets.

These findings are pretty dismal, but surely the explosion in nutrition research and education over the past 15 years would make it unlikely that 21st century athletes could commit such basic nutritional sins? Unfortunately the evidence suggests otherwise. A 2002 US study on 23 nationally ranked female volleyballers provides more evidence that some athletes may still be struggling to fulfil even basic nutritional needs [(11)]. Nutrient and energy intakes and energy expenditure were determined by 3-day weighed food records and activity logs, while blood tests were taken to measure nutrient status. Mean intakes for folic acid, iron, calcium, magnesium and zinc were all less than the respective RNIs for these nutrients, while 50% of the athletes were consuming less than the RNIs for the B-complex vitamins and vitamin C. To make matters worse, both carbohydrate and protein intakes were found to be inadequate for athletes of this activity level, while three athletes presented with gross iron deficiency anaemia (blood haemoglobin less than 12mg/dL) and a marginal vitamin B12 and C status were found in one and four athletes, respectively.

Avoiding the pitfalls

It's clear from these and other studies that while many athletes may be more knowledgeable about nutritional basics than their sedentary counterparts, and more motivated to translate this knowledge into action, some are still unwittingly neglecting the dietary fundamentals. This being the case, you might at this point be wondering whether your own diet is up to scratch, or whether there are certain areas that could be improved.

As we've already hinted, building a detailed and accurate picture of nutritional status is a time-consuming process, which needs to be carried out by a suitably qualified professional. If you suspect that your diet falls short of your

nutritional requirements and wish to have a proper assessment, you need to ensure that you consult someone who is appropriately skilled and qualified to carry out what is a potentially complex task. In the UK for example, this means someone who is on the British Association for Nutritional Therapy (BANT) register. A degree background in biological/biochemical/ chemical sciences or nutrition/sports nutrition is also desirable.

'Beware of 'consultants' who claim to offer wacky methods of analysis; hair tests for mineral status are of very limited value, while assessments based on blood groups, pendulums, crystal healing etc are no better than guesswork.'

A proper nutritional assessment should consist of at least two elements:

- A detailed food diary containing details of all foods/portion sizes consumed for at least three days and preferably longer;
- A questionnaire about general dietary habits.

In a more comprehensive assessment, this information is often supplemented by one or more biochemical tests carried out on blood, urine and maybe even sweat, but of course these add to the costs. Beware of 'consultants' who claim to offer wacky methods of analysis; hair tests for mineral status are of very limited value, while assessments based on blood groups, pendulums, crystal healing etc are no better than guesswork.

However, you don't necessarily need a full nutritional assessment in order to increase the basic quality of your diet. So long as you remember that a) dietary basics *are* important and b) that the majority of your diet should be comprised of whole and unprocessed foods such as wholegrain breads and cereals, fresh fruits and vegetables (particularly important for antioxidant intake), high quality proteins such as lean meats, fish and low-fat dairy produce, beans and lentils, and nuts and seeds, you'll be on the right track.

Andrew Hamilton

Jargon buster

Ergogenic – a substance that enhances sports performance either directly, or indirectly via improved response to training

RNI –Reference Nutrient Intake; the daily amount of a nutrient required to produce health (*NB.* many countries still use the term RDA, or Recommended Daily Amount)

Nutrient density – the nutrient content of a food per calorie consumed; nutrient dense foods contain high levels of nutrients for each calorie of energy they provide

Enzymes – large protein molecules that speed up essential biochemical reactions in the body, which would otherwise either occur too slowly to sustain life or not at all

Macronutrients – nutrients needed in comparatively large amounts by the body (ie protein, fat, carbohydrate, fibre and water)

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Can antioxidants help reduce post-exercise soreness?

The unfolding story of antioxidant nutrition for athletes is full of twists and turns. Just a couple of decades ago, the word 'antioxidant' was almost unheard of by athletes and coaches. But as nutritional science began to unravel the role of these compounds in protecting and enhancing cell performance and longevity, it soon became apparent that athletes might have increased antioxidant requirements because of their increased throughput of oxygen during training and competition.

At a Glance

- The theories and causes of muscle damage and soreness are discussed ;
- The possible role of antioxidants in ameleorating post-exercise muscle damage is outlined;
- Research on the potential benefits or otherwise on post-exercise muscle soreness of increasing antioxidant intake is discussed and conclusions drawn.

The prospect of athletes being able to protect themselves from damaging 'free radicals' (by-products of oxygen metabolism) by supplementing their diet with extra antioxidants was too attractive to ignore. And before long many sportsmen and woman were taking high doses of antioxidant nutrients as a matter of routine.

However, more recent research has muddied the waters considerably, as evidence has emerged that large single doses

of antioxidants can actually increase the markers of oxidative damage and slow athletes down! When it comes to antioxidants, more is not always better!

But there is a further twist to the story, because other recent research has suggested that antioxidant supplementation may have some short-term beneficial effects on recovery from exercise – particularly on post-exercise muscle soreness and dysfunction following 'unfamiliar' exercise. This is a very different proposition from the long-term protective benefits that

Antioxidants and athletes

Chemically speaking, oxygen is amazing stuff. Its special reactivity provides us with the energy required to sustain life, including the ability to power movements and muscular contraction. This explains why oxygen – and the ability to absorb, transport and use it – is so important to athletes, who need lots of the stuff to sustain maximum power and work outputs.

However, the oxygen molecule is a double-edged sword, because this same chemical reactivity can also wreak havoc in the cells by means of the potentially destructive molecules called free radicals, which are produced unavoidably as a consequence of harnessing the chemical energy of oxygen within the body. Free radical damage is believed to be a major factor in ageing, degeneration and many diseases.

Fortunately, the human body comes equipped with a number of systems capable of deactivating the free radicals produced as a result of oxygen metabolism, and dissipating their energy harmlessly. Collectively known as the 'antioxidant defence system', these systems use both antioxidant enzymes (large protein molecules manufactured in the body) and antioxidant nutrients (consumed in the diet) to 'soak up' the energy of free radicals, thereby minimising damage to the body.

In recent years, there has been much speculation that athletes might be at increased risk of free radical damage, or 'oxidative stress'. Athletes don't just process a larger volume of oxygen than their sedentary counterparts – they also process it at a higher rate; during training, the rate of oxygen processing by the mitochondria (the energy producing furnaces in the cells) can rise by a factor of 20, placing exceptionally high demands on the antioxidant defence system. The fact that free radical generation does increase during vigorous exercise is no longer in doubt.

have so far underpinned the use of antioxidant supplements by athletes *(see box opposite)*.

'Evidence has emerged that large single doses of antioxidants can actually increase the markers of oxidative damage and slow athletes down.'

Muscle fibres are not perfectly resilient, and when placed under load some damage can and does occur. However, this damage can be seen as a normal and healthy response to training; during the recovery period after training, any damaged tissue is broken down and removed, then replaced with new and healthy tissue. Post-exercise muscle damage is believed to be the main cause of the muscle soreness that occurs after particularly strenuous workouts, or those containing unfamiliar movement patterns.

Studies using a technique known as electron microscopy indicate that this damage occurs principally to the 'Z-line' regions of muscle fibres, which can be thought of as the boundaries between contractile units. Type II (fast-twitch) muscle fibres are known to suffer more damage than their type I (slow-twitch) counterparts, possibly because they have wider Z-lines and thicker connective tissue surrounding the fibre.

It has been shown that eccentric exercise (where the muscle fibres lengthen under load) produces more damage than concentric exercise (where fibres shorten under load). However, it is also known that eccentric training leads to a reduction in exercise-induced muscle damage during subsequent exercise, and this may be because of the replacement of weak fibres by stronger ones.

How is antioxidant nutrition linked with exercise-induced muscle damage? Take a muscle biopsy immediately after a strenuous training session and you'll certainly detect muscle damage. However, take a muscle biopsy from the same area 2-3 days later and you'll detect even more damage! Although the initial damage that occurs is believed to be caused by mechanical strain, the delayed and additional damage is now known to occur as a result of an inflammatory response, aggravated by free radicals.

When a muscle cell is damaged, it becomes more 'leaky' to calcium, which then accumulates within the cell. This acts as a signal, attracting a range of immune cells, such as macrophages

and monocytes, to the damaged area. These immune cells release toxins, including free radicals, to further break down the damaged areas and mop up tissue debris. In other words, the destructive power of free radicals is harnessed positively to help break down damaged tissue.

At this point, you might be feeling a bit confused. If cells containing free radicals are required to break down damaged tissues and initiate repair, surely antioxidants (which deactivate free radicals) would hinder this process?

There are two reasons why this does not happen:

1. Studies suggest that an improved antioxidant status enhances the adaptive response to exercise- induced muscle damage by increasing the concentration of the immune cells charged with initiating breakdown and repair[1];
2. While the free radicals released by these cells help to break down the damaged tissue, if unchecked they can also attack adjacent healthy tissue. It's a bit like using a sledgehammer to crack a nut: yes, it will get the job done, but it may take out your coffee table in the process! An optimally functioning antioxidant defence system, however, appears to minimise this collateral damage[2].

So much for the theory. But how much evidence is there from human studies that improved antioxidant status can reduce post-exercise muscle soreness in athletes undergoing vigorous training?

Although there have been anecdotal reports from athletes that vitamin C (a powerful antioxidant) seems to help recovery and reduce delayed onset muscle soreness (DOMS), scientific evidence for this effect has been thin on the ground. But in 2001 a study was carried out to examine the effects of two weeks of vitamin C supplementation on recovery from an 'unaccustomed' bout of exercise[3].

Sixteen male subjects were randomly assigned to either vitamin C (consuming 200mg of ascorbic acid twice a day for two weeks) or placebo. All the subjects then performed a 90-minute intermittent shuttle-running test, a protocol chosen to be

significantly different from their normal exercise patterns. Most of the post-exercise blood tests showed no significant differences between the groups. However, the researchers discovered that vitamin C supplementation had modest beneficial effects on muscle soreness, muscle function and plasma concentrations of malondialdehyde (a marker for free radical damage). Furthermore, the rise in blood levels of interleukin-6 (a marker of inflammation) was considerably lower in the vitamin C group than in the controls. The researchers concluded that prolonged vitamin C supplementation did offer some modest beneficial effects on recovery from unaccustomed exercise.

However, interleukin-6 levels can also change in response to metabolic demands, and it was unclear in this study whether the lower rise in this inflammatory marker in the vitamin C group resulted from a reduced response to muscle damage or some form of interaction with the metabolic demands of the activity.

'The possible benefits of vitamin C supplementation may be also related to the timing and duration of the supplementation period.'

Effects of vitamin C supplementation

To clarify this question, a study carried out last year investigated the effects of vitamin C supplementation on a bout of exercise that initiated similar muscle damage but had a lower metabolic cost[4]. As in the previous study, male subjects were split into vitamin C and control groups, the former taking 200mg of ascorbic acid twice a day for two weeks and the latter taking placebo. At the end of this period, the subjects completed 30 minutes of downhill running at a gradient of -18% (providing lots of eccentric muscle contractions at low energy cost) and recovery was monitored for up to three days after the exercise.

This time, both placebo and vitamin C groups reported similar degrees of muscle soreness, and although downhill running increased plasma interleukin-6, there was no significant reduction in the magnitude of this rise in the vitamin C group, suggesting that supplementation did not offer significant benefits following eccentric exercise with a low metabolic component.

The possible benefits of vitamin C supplementation may be also related to the timing and duration of the supplementation period. In another study on vitamin C, nine habitually active

men completed two training sessions consisting of a 90-minute intermittent shuttle-running test, exactly as in the first study above[5]. Before one test, they consumed a 1,000mg dose of ascorbic acid and before the other, an identical-looking placebo.

Although the vitamin C supplementation produced a peak of plasma vitamin C immediately after the shuttle run test, muscle soreness and markers of muscle damage and free radical activity were elevated to an equal extent after exercise in both trial conditions. The researchers speculated that such short-term supplementation might have been ineffective because it occurred at an inappropriate time; in other words, if vitamin C supplementation does protect against high metabolic post-exercise muscle soreness and damage, longer-term supplementation may be more effective than the 'pop a pill before a workout' approach!

'If vitamin C supplementation does protect against high metabolic post-exercise muscle soreness and damage, longer-term supplementation may be more effective than the 'pop a pill before a workout' approach!'

Long-term studies on vitamins C and E have drawn a blank. A study of 15 experienced male distance runners looked at whether four weeks of daily supplementation with vitamin C or vitamin E could reduce markers of muscle damage following a 21k run[6]. The runners were divided into two groups (vitamin or placebo) and were supplemented for four weeks before completing the first 21k run in as fast a time as possible.

After a four-week 'washout period', the vitamin-treated subjects crossed over and received the alternate supplement for the next four weeks. They then completed a second 21k run. No significant differences were found between the vitamin and placebo groups for a range of markers tested.

More encouraging findings

More encouraging results were obtained from another study examining the effects of antioxidant supplementation on 'neutrophil oxidative burst' (the capacity of the immune cells to release free radicals to help break down damaged muscle)[7]. Twelve healthy endurance runners took either a placebo or an antioxidant vitamin supplement (containing 18mg beta-carotene, 900mg vitamin C and 90mg vitamin E) for seven days before a two-hour treadmill run at 65% VO2max. Blood

samples were drawn before and immediately after exercise and analysed for neutrophil oxidative burst activity.

Neutrophil oxidative burst in the antioxidant group was significantly higher than in the placebo group after exercise, and the researchers concluded that antioxidant supplementation may be of benefit to endurance athletes for the maintenance of this particular function of the immune system.

The evidence suggests that how accustomed an athlete is to a particular mode of exercise is an important factor in determining whether extra antioxidants can reduce post-exercise muscle damage and soreness. But new evidence suggests that age and gender are also relevant.

In a recent study on the impact of antioxidants on exercise-induced DNA damage, scientists studied 10 female and 11 male runners during a 50k ultra-marathon who had been randomly assigned to supplementation with either an antioxidant mix (1,000mg vitamin C and 400IU vitamin E) or placebo[8]. Tests were conducted to assess DNA damage in blood cells known as leukocytes at a number of different times: pre-race, mid-race, two hours post-race and daily for six days afterwards.

Overall, DNA damage increased at mid-race but returned to baseline by two hours post-race, indicating that the exercise had induced transient DNA damage. There was, however, a marked difference between the men and women. Women taking the antioxidant cocktail had 62% less DNA damage at one day post-race than those on placebo. By contrast, there were no significant differences between the men at any time point, indicating that the antioxidant mix enhanced recovery in women alone. The implication is that female endurance athletes may have more to gain than men from using antioxidants to enhance post-exercise recovery.

'Female endurance athletes may have more to gain than men from using antioxidants to enhance post-exercise recovery.'

As far as older athletes are concerned, there is evidence that exercise-induced muscle damage is more extensive than in younger ones for any given intensity/duration of exercise, but also that older athletes may have most to gain from antioxidant supplementation.

One study compared the structural muscle damage produced

'As with so many areas of sports nutrition research, the evidence that antioxidant supplementation can reduced post-exercise muscle damage and soreness is far from clear-cut.'

by 45 minutes of high-intensity eccentric exercise in young (20-30) and old (59-63) men and found that the same intensity and duration of exercise produced significantly more muscle damage in older men[9].

Although older athletes appear to be more susceptible to exercise-induced muscle damage, they also seem to respond better to antioxidant supplementation[10]. Twenty-one male volunteers were split into two age groups (22-29 and 55-74 years). The members of each group were then randomised to 48 days of supplementation with either vitamin E (800IU per day) or placebo before a downhill running session designed to accentuate damaging eccentric muscular contractions.

After the run, all subjects were monitored for 12 days for changes in responses to muscle damage. As might be expected, young subjects on placebo mounted a stronger attempt to repair damaged tissue than the older ones. However, in the vitamin E supplemented groups, the neutrophil oxidative burst response of the older group was elevated to such an extent that the differences between the two age groups effectively disappeared. The researchers concluded that vitamin E supplementation may affect the rate of repair of skeletal muscle after muscle damage and that these effects may be more pronounced in older subjects.

As with so many areas of sports nutrition research, the evidence that antioxidant supplementation can reduced post-exercise muscle damage and soreness is far from clear-cut. However, it is possible to interpret these findings with a view to making useful recommendations for athletes and coaches.

An improved antioxidant intake doesn't appear to prevent the mechanical damage induced by exercise, but in some circumstances it may be able to reduce the amount of post-exercise damage that occurs as part of the repair and regeneration process.

The evidence to date suggests that for younger male athletes this beneficial effect is felt mainly when the exercise is 'unaccustomed', vigorous in nature and including a significant amount of eccentric work.

Female athletes, on the other hand, may have more to gain

from antioxidant supplementation, even during regular training. For older athletes, who are more at risk than young ones from exercise-induced muscle damage, the case for antioxidant supplementation appears rather more clear-cut, and fortifying the diet with these nutrients seems a reasonable thing to do.

For those thinking of taking supplements, bear in mind that most studies showing a positive effect have used longer-term supplementation, over weeks rather than days. Remember, too, that the antioxidant nutrients work synergistically; so it is probably better to take a combination of vitamins A (retinol or beta carotene), C, E and the mineral selenium than a single large dose of any one nutrient.

Finally, don't overlook the contribution of diet. Some of the most powerful dietary antioxidants out there come in the form of the brightly coloured phytochemicals found in a wide range of fresh fruits and vegetables. As a rule of thumb, the more colourful the fruit or vegetable, the higher its phytochemical content – one more reason to eat up those greens!

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Antioxidant vitamins: can they do athletes more harm than good?

Oxygen is amazing stuff. Thanks to its special chemical reactivity, it provides us with the energy required to sustain life, including the ability to power movements and muscular contraction. This explains why oxygen – and the ability to absorb, transport and use it – is so important to endurance athletes, who need lots of the stuff to sustain maximum power and work outputs.

At a Glance

- Methods of assessing free radical activity during exercise are outlined;
- Research into the use efficacy of antioxidant supplements to combat oxidative stress during exercise is presented;
- The significance of conflicting evidence and the potential pitfalls of single nutrient supplementation is discussed;
- Conclusions are drawn and recommendations made.

However, the oxygen molecule is a double-edged sword. For this same chemical reactivity can also wreak cellular havoc by means of the transient, highly reactive and potentially extremely destructive molecular species called *free radicals*, which are produced unavoidably as a consequence of harnessing the chemical energy of oxygen within the body.

In recent years, there has been much speculation that athletes, who not only consume more oxygen than others to fuel their training but also frequently train at or near their maximum oxygen uptakes, might be at increased risk of free radical-induced damage, or 'oxidative stress'. Athletes don't

just process a larger volume of oxygen than their sedentary counterparts – they also process it at a higher rate; during training, the rate of oxygen processing by the mitochondria (the energy-producing furnaces in the cells) can rise by a factor of 20, placing exceptionally high demands on antioxidant defence systems. The fact that free radical generation does increase during vigorous exercise is no longer in doubt[1-5]. However, considerable confusion remains about the implications of this increased free radical generation. There are three key questions:

'When oxygen-free radicals attack the lipid membranes around cells, molecules called peroxides are formed.'

1. Does this increased oxidative stress actually lead to significant biological damage within the cells of athletes?
2. Can the body of an athlete adapt to this increased oxidative stress by manufacturing higher levels of the antioxidant enzymes?
3. Can an athlete's antioxidant defences be fortified by ingesting increased dietary amounts of the antioxidant nutrients, including beta carotene, vitamin C, vitamin E and the mineral selenium?

Free radical activity during exercise

The answer to the first question is not yet clear. Two powerful techniques, known as 'electron spin resonance' and 'paramagnetic resonance spectrometry' now enable scientists to directly measure the concentration of free radicals during exercise and can be used to detect the 'superoxide' radical, one of the most reactive and damaging radical species. However, most of these studies have been carried out in animals rather than humans; moreover, it is not possible to prove conclusively that the increased production of superoxide radicals automatically leads to free radical damage.

An alternative approach is to look for signs of free radical damage, rather for the presence of the free radicals themselves. One of the commonest current methods is to measure how much *lipid peroxidation* has occurred. When oxygen-free radicals attack the lipid membranes around cells, molecules called peroxides are formed. These peroxides

are not produced in other metabolic pathways, so an increase in peroxide concentration is a sure sign that more oxidative stress has occurred. Other techniques look for signs or fragments of oxygen radical-damaged DNA, such as 8-hydroxyguanine.

However, it is important to realise that in humans these tests are subject to error. Many of these oxidative stress markers are very fragile and readily degrade before analysis, while other substances can interfere with the testing reagents, producing false positive readings. Relying on a single marker to measure oxidative stress in humans is, therefore, fraught with difficulties and probably explains some of the conflicting results that have emerged from clinical trials.

Conflicting results on oxidative stress

For example, increases in blood levels of a molecule called malondialdehyde (MDA), which is formed in the body when lipids are damaged by oxygen radicals, have been found after:

- an 80k race [6];
- a 30-minute treadmill test at 60% and 90% of maximal oxygen uptake [7];
- downhill running [8];
- incremental cycling tests to exhaustion in sedentary and moderately trained men [9,10].

By contrast, no increases in MDA were found after:

- a half-marathon [11];
- 60 minutes of bench-stepping exercise [12];
- maximal cycle ergometry exercise [13];
- incremental cycle ergometry exercise in elite athletes [14].

The implication of these conflicting results is that tests for oxidative stress and damage in humans need be interpreted with caution, especially when a single marker is used.

The human body can adapt to many environmental and metabolic stressors, so can it adapt to oxidative stress? On balance, the evidence suggests that it can. A number of

studies have compared the antioxidant defence systems of athletes before and after a period of increased training intensity or duration and have found that both increased volume and intensity of training stimulate the production of antioxidant enzymes in the body, including glutathione peroxidase and superoxide dismutase[15,16,17]. Moreover, some studies have also shown that this increase in antioxidant enzymes can reduce the levels of oxidative stress markers in the blood after training, so apparently offering protection against oxidative damage[18].

However, these results still need to be interpreted with caution because many of the studies have used different markers of antioxidant status and different training levels of subjects. More importantly, it is highly debatable whether the increased production of antioxidant enzymes observed is sufficient to combat the increased oxidative stress of heavy training loads, which has led to suggestions that athletes should take further steps to boost their defences by supplementing their diet with antioxidant nutrients.

'Some studies have demonstrated that certain antioxidant nutrients can reduce apparent oxidative stress when supplemented at higher levels than would ordinarily be found in the diet.'

This is where the story begins to get really tangled. Some studies have demonstrated that certain antioxidant nutrients can reduce apparent oxidative stress when supplemented at higher levels than would ordinarily be found in the diet. For example, a selenium-supplemented group of healthy males produced significantly higher levels of glutathione peroxidase (one of the body's main antioxidant enzymes) in response to a mixture of treadmill running and cycling at different intensities (65-100% VO_2max) than a control group[19].

Similar beneficial effects have been observed for vitamin E. In a long-term study on endurance racing cyclists, a group supplemented with vitamin E at 10 times the normal rate showed a smaller increase in blood MDA after strenuous exercise than a placebo group[20]. The supplemented cyclists also had lower levels of blood creatine kinase (a protein normally found in muscle, which can leak into the blood after membrane damage), suggesting a protective effect of vitamin E on muscle damage induced by oxidative stress.

Benefits of vitamins C and E

Other studies have also indicated that vitamin E supplementation may help reduce oxidative damage during exercise. When cyclists were supplemented with vitamin E at 40 times the RDA, the amount of pentane they breathed out from their lungs (pentane is a gas produced by lipid peroxidation) dropped significantly[21]. There is also some evidence, albeit rather less convincing, that vitamin C offers antioxidant protection, particularly when given in combination with vitamin E. For example, 400IU of vitamin E and 200mg of vitamin C taken for four weeks before a marathon run resulted in reduced levels of blood MDA immediately after the event and persisted for 24 hours[22].

However, other well-conducted studies have cast doubt on the efficacy of antioxidant nutrient supplementation. Athletes ingesting either 2,000mg per day of vitamin C or a carbohydrate placebo were asked to run 27k, after which their blood levels of dienes (a marker of lipid peroxidation) was measured. No differences were observed between the groups[23]. Another study comparing athletes supplemented with a combination of antioxidant nutrients (294mg vitamin E, 1,000mg vitamin C and 60mg of coenzyme Q10) and placebo before a 31k run found that the blood antioxidant potential (a measure of total antioxidant activity) was raised substantially in the supplemented group; however, there was no corresponding reduction in the amount of LDL diene conjugation (a measure of oxidative stress inflicted on molecules called low-density lipoproteins, which circulate in the bloodstream)[24].

A recent American study examined the effects of supplemental vitamin C (500mg per day) and vitamin E (400IU per day) for two months on oxidative damage to DNA by measuring the levels of a marker substance called 8-hydroxy-2'-deoxyguanosine (8-OHdG) excreted in the urine[25]. They also collected detailed dietary information from each of the 184 subjects in the study. The researchers found that, by comparison with placebo, neither vitamin

reduced the level of excreted 8-OHdG, suggesting no effect on oxidative damage to DNA.

Intriguingly, however, the researchers found that higher intakes of fruit and vegetables *did* reduce the amount of excreted 8-OHdG. They also found that the greater the level of exercise, the lower the level of damaged DNA marker, supporting the hypothesis that the body can upregulate its antioxidant defence systems in response to increased oxidative stress.

Although the increased intake of fruit and vegetables correlated with an increase in dietary vitamin C intake (fruit and vegetables being particularly rich in this vitamin), the researchers did not believe that these higher vitamin levels were responsible for the reduction in DNA damage (otherwise this same reduction should have been seen in the supplemented group, which it wasn't). Rather they concluded that there there must be other biologically active substances in fruit and vegetables responsible for this protective effect (something we'll return to later).

Staying on the safe side

Given current uncertainties about the effectiveness of antioxidant nutrient supplementation, wouldn't it be wisest for athletes to take a supplement containing a mixture of the antioxidant nutrients 'just to be on the safe side'? Perhaps not, because a recent study suggests that, far from being synergistic, some antioxidant nutrients may actually work against each other[26]! Seven trained male cyclists were treated with four different supplementation regimes, as follows:

- placebo;
- 1,000mg of vitamin C per day;
- 400IU of vitamin E per day;
- 1,000mg of vitamin C *plus* 400IU of vitamin E.

After completing a steady-state ride and performance ride on the ergometer, blood samples were drawn and analysed for MDA (a lipid peroxidation marker). As expected, there were

no differences in terms of performance benefits between the different supplementation regimes. In line with other studies, it was also found that the combination of vitamins C and E reduced blood levels of MDA. However, the researchers were surprised to discover that vitamin E supplementation *alone* reduced pre-exercise blood MDA levels far more than the combined supplement – by around 40% – and also substantially reduced post-exercise MDA levels!

More worrying, though, was the finding that, by comparison with placebo, vitamin C supplementation alone actually *elevated* MDA levels; in other words, it acted as a *pro*-oxidant rather than an antioxidant. The researchers concluded that, while 400IU daily of vitamin E did offer protection, 1,000mg daily of vitamin C appeared to promote cellular damage. This is certainly a plausible theory because, taken in excess, vitamin C is known to exhibit a phenomenon known as 'Fenton chemistry', where it acts as a catalyst to stimulate the production of the highly damaging hydroxyl radical from minerals (such as iron) and naturally occurring substances (such as hydrogen peroxide) in the body.

'Some scientists have even proposed that excessive antioxidant supplementation may be counter-productive because oxidative stress and some degree of free radical damage may actually be an essential part of the adaptation process within muscles.'

Oxidative stress may be essential

Although appropriate levels of antioxidant supplementation may offer some long-term protection to athletes, and although there is some limited evidence that vitamin C may help reduce post-exercise muscle damage, there is no real evidence to date that antioxidant nutrients can boost short-term performance in athletes. Indeed, some scientists have even proposed that excessive antioxidant supplementation may be counterproductive because oxidative stress and some degree of free radical damage may actually be an essential part of the adaptation process within muscles.

Additionally, recent animal studies lend support to the notion that 'more isn't always better'. In one of these, greyhounds were treated with three different supplementation regimes, as follows [27]:

- placebo;

- 1,000mg vitamin C daily with food;
- 1,000mg administered orally one hour before racing on race days and with food on non-race days.

The results demonstrated that, regardless of when the vitamin C was administered, supplemented dogs ran 0.2 seconds slower over 500m than their non-supplemented counterparts – a small but statistically significant difference. These results appear to support those from an earlier study, which showed that, while a modest daily dose of 100IU of vitamin E didn't affect running performance, a higher dose of 1,000IU caused greyhounds to run more slowly [28].

Other recent studies seem to indicate that high doses of antioxidant nutrients may actually harm performance. For example, rats fed high doses of vitamin E were not able to produce as much muscle force as their unsupplemented counterparts during low frequency stimulation[29]; and in a human study, vitamin C and N-acetyl cysteine (another antioxidant) given during the acute phase inflammatory response to an eccentric arm injury increased the amount of oxidatively damaged lipids, resulting in transiently increased tissue damage [30].

The best advice for athletes

Faced with this bewildering array of information, what's the best advice for athletes seeking maximum performance today and optimal protection for tomorrow? First, the evidence is that on balance, while not improving short-term performance, modest doses of antioxidant nutrients do appear to offer some protection. However, more is not necessarily better and higher doses may actually increase oxidative damage and could even lead to reduced performances.

Secondly, because antioxidant nutrients work together synergistically, both with each other and with the antioxidant enzymes of the body, any supplementation should be in the form of a complex (for example containing beta-carotene,

vitamin C, vitamin E and selenium) rather than single nutrients. Although it is difficult to make hard and fast recommendations, the evidence suggests that total daily vitamin C intake should not exceed 500mg per day, with 300-400mg per day the upper supplementation limit for most people.

Although there is less evidence for detrimental effects of high vitamin E supplementation, many studies suggesting a protective effect have used around 400IU per day, and it seems prudent not to exceed this figure. The UK Foods Standards Agency suggests a safe upper limit of 350mcg per day for selenium supplementation, but in the absence of a proven deficiency most studies have shown little or no benefit to exceeding 200mcgs per day. The safe upper limit for beta-carotene is set at 7mg per day.

Finally – and perhaps most importantly of all – don't forget about fruit and vegetables. In recent years, there has been an explosion of research into naturally-occurring substances in plants (often responsible for giving the plant its characteristic colours and flavours) called phytochemicals. Many of these compounds display remarkable antioxidant capacities, sometimes tens or even *hundreds* of times greater than the antioxidant nutrients. Example include the carotenoid family found in red and green fruits and vegetables, the flavenoid family found in citrus fruits, the tocotrienol family found in nuts, seeds and wheat-germ, and a number of sulphur-containing compounds, such as sulphorane, found in broccoli, and allicin found in garlic.

As a rule of thumb, the more colourful the fruit or vegetable, the higher its phytochemical content will be. It was almost certainly the higher phytochemical intake of those fruit and vegetable lovers in the study on DNA damage[25] that afforded them the real protection So if you're serious about obtaining maximum protection, make sure you're getting at least the recommended levels of those fruit and veg portions a day – if not more!

Andrew Hamilton

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Antioxidant protection for athletes: is it time to ditch the pills?

During the last decade, research into antioxidant nutrition and athletic performance has been one of the most rapidly evolving areas of sports nutrition. But while many athletes take antioxidant supplements, the most recent research suggests that there may be more effective approaches to protecting the athletic body.

At a Glance

- Evidence is presented on the potential benefits for athletes of antioxidant nutrient supplementation as well as conflicting evidence for this approach;
- More recent research suggesting greater benefits by increasing naturally occurring dietary phytochemicals from fruits and vegetables is oulined;
- Dietary strategies on how to boost phytochemical intakes are given.

Although there's plenty of evidence for the theory of antioxidant protection from free radical damage (*see box, overleaf*) to cells generally, the link between antioxidant supplements and protection during athletic performance is poorly understood. Some studies appear to show a benefit[1-4], others have shown little benefit[5-8] and some animal studies have even suggested that large doses of antioxidant vitamins may be detrimental[8-11] (for a full discussion see *Antioxidant vitamins: can they do athletes more harm than good?*" elsewhere in this issue). Thus the antioxidant story has been characterised by changing scientific consensus and confusion:

do athletes really need extra antioxidant protection, and if so what kind and how much?

Nutrients as antioxidants

Much of the research into antioxidants and athletic protection/ performance has centred on nutrients such as vitamins A, C and E and the mineral selenium. Not only are these essential for other functions in the body, they also activate some of the key antioxidant enzymes in the body, which help to defend cells against free radical damage.

Most studies into antioxidants and athletes have involved athletes taking large doses of one or more of these antioxidant nutrients and then observing the effect on a subsequent bout of exercise. In particular, researchers have been keen a) to investigate whether the administration of antioxidant nutrients reduces the amount of oxidative

What is free radical damage?

Free radical damage describes the damage that occurs within cells (for example cell membranes and **DNA**) at a molecular level as a result of 'free radicals'. These free radicals are transient but extremely reactive chemical species that unavoidably occur during oxygen metabolism when fats, proteins and carbohydrates are combined with oxygen in the body to produce energy (aerobic metabolism). For this reason they are sometime called 'reactive oxygen species' (ROS) or 'oxygen free radicals' (see *Understanding antioxidants* elsewhere in this report).

Although our cells have very efficient antioxidant defence systems to quench and neutralise harmful free radicals, these systems are not 100% efficient, and over time biochemical damage gradually accumulates, leading to a reduction in cellular function. Most scientists now believe that accumulated cellular free radical damage lies at the heart of the ageing process and many degenerative diseases such as cancer, autoimmune diseases and Alzheimer's disease. Athletes process and use larger volumes of oxygen and at higher rates than the majority of the population; this explains why many scientists believe that they may benefit from higher intakes of antioxidant nutrients to bolster defences.

damage caused by exercise and b) to see whether antioxidants actually enhance performance.

The answer to the first part of this question is that there does seem to be evidence that extra antioxidant nutrients can reduce the markers of free radical damage during subsequent exercise, but, as mentioned above, this is by no means clear-cut. In terms of performance gain, there's little evidence to date that antioxidant nutrients can enhance actual physical performance but there may be other benefits associated with taking them.

Muscle soreness

One potential benefit is the reduction in post-exercise muscle soreness, (see *Can antioxidants help reduce post-exercise soreness?* elsewhere in this report). To recap briefly, we now know that the destructive power of oxygen free radicals can be harnessed positively by immune cells to help break down exercise-damaged muscle tissue as part of the process of tissue repair. We also know that this immune-cell-mediated free radical damage appears to peak roughly 24 hours after exercise, which explains why muscle soreness also peaks then.

However, an optimally functioning antioxidant defence system appears to minimise extraneous free radical damage to otherwise healthy tissue, and may therefore help to minimise the degree of post-exercise muscle soreness; studies have shown that mice fed a compound called PEG-SOD (an extremely powerful free radical deactivator) performing prolonged eccentric exercise exhibited much less delayed-onset free radical damage to otherwise healthy muscle tissue than controls[12].

In our previous review of this subject we reported that, while some studies on supplementing antioxidant nutrients had produced inconclusive results[13,14], others had reported positive results including:

- reduced muscle soreness after shuttle running when taking vitamin C [15];

- reduced exercise-induced DNA damage in immune cells in women when taking vitamins C and E[16];
- enhanced muscle damage repair in older runners running downhill when taking vitamin E[17].

But what does the more recent research say on this subject? In an American study on vitamin C supplementation and delayed onset muscle soreness (DOMS), 18 healthy men were randomly assigned to one of two groups[18]; the vitamin C group took 3g per day (1g morning, noon and night) of vitamin C for two weeks prior to a heavy exercise session and for four days afterwards, while the control group took placebo (dummy) pills. Both groups then performed 70 eccentric action repetitions of elbow flexion to stress the triceps of the rear arm in such a way as to produce maximum post-exercise soreness. Blood samples were also taken in order to measure creatine kinase (an indicator of muscle breakdown) and oxidised glutathione/glutathione ratio (a measure of free radical damage due to oxidative stress). Among the key findings were the following:

- At all times during the 96-hour period following the exercise, muscle soreness levels were significantly reduced in the vitamin C group compared to the control group;
- The increase in creatine kinase in the vitamin C group was significantly less at 48+ hours after exercise than the control (indicating reduced muscle breakdown);
- The oxidised glutathione/glutathione ratio was lower in the vitamin C group at four and 24 hours post-exercise than the control group (indicating less oxidative damage).

Meanwhile, a study published in 2006 looked at the effects of giving an antioxidant supplemented carbohydrate /protein drink to cyclists riding to exhaustion at 70% VO_2max and then again 24 hours later at 80% VO_2max[19]. Compared to an isocaloric carbohydrate drink without added antioxidants, the antioxidant supplement drink reduced post-exercise muscle soreness and markers of muscle damage, even

though the cyclists consumed less carbohydrate on this regime than when taking the carbohydrate-only drink.

However, not all the latest research on supplementing antioxidant nutrients is positive. A study published in the New Year on 22 runners during and after a 50km ultramarathon showed that, compared to a placebo, taking 1,000mgs of vitamin C and 300mgs of vitamin E did not reduce markers of post-exercise muscle damage or the contractile ability of the quadriceps and hamstrings[20].

'Many nutritionists now believe that dietary phyto-chemicals are at least as important as the antioxidant nutrients (if not more so) in protecting cells from free radical damage.'

The fruit and vegetable connection

The weight of evidence for supplementing antioxidant nutrients is on balance more favourable than not, but still far from clear-cut. One possible reason for the mixed results in these studies is that, until recently, researchers have focused on supplementing antioxidant nutrients but have paid scant attention to a huge range of naturally occurring compounds in plant foods called phytochemicals (*see box, overleaf*).

Many nutritionists now believe that dietary phyto-chemicals are at least as important as the antioxidant nutrients (if not more so) in protecting cells from free radical damage. There's good evidence for this in a US study that looked at the effects of supplemental vitamin C (500mg per day) and vitamin E (400IUs per day) for two months on oxidative damage to DNA by measuring the levels of a marker substance called 8-hydroxy-2-deoxyguanosine (8-OHdG) excreted in the urine in 184 subjects[21].

Compared to placebo, neither vitamin reduced the level of markers of oxidative DNA damage. However, a closer analysis of the subjects' diets showed that higher intakes of fruit and vegetables *did* reduce the amount of DNA damage, regardless of whether they were taking vitamins or placebo – persuasive evidence that the phytochemical content of the diet exerted more of a protective effect than supplemented antioxidant nutrients.

Further evidence comes from a recent study this year on oxidative stress during exercise (30-minute run at 80% of

Phytochemicals

Phytochemicals are often responsible for giving a plant its characteristic colours and flavours and many of these compounds display remarkable antioxidant capacities, sometimes tens or even *hundreds* of times greater than those of antioxidant nutrients. Examples include the carotenoid family (found in red and green fruits and vegetables), the flavenoid family (found in citrus fruits), the tocotrienol family (found in nuts, seeds and wheat-germ), and a number of sulphur-containing compounds, such as sulphorane (found in broccoli) and allicin (found in garlic). As a very rough rule of thumb, the deeper and more vivid the colour of the fruit or vegetable, the higher the phytochemical content and therefore its antioxidant activity.

VO_2max), which compared the protective effects of daily supplemented antioxidant nutrients (400IUs of vitamin E and 1,000mgs of C) with a mixed fruit and vegetable juice powder concentrate containing 108IUs of vitamin E and 276mgs of vitamin C[22]. The results showed that while only the vitamin supplements raised blood vitamin levels, both treatments reduced the amount of a marker of oxidative stress called protein carbonyl and by similar amounts. Compared to the vitamin supplements, the fruit/vegetable powder contained less than a quarter of the vitamins C and E, which suggests that additional antioxidant activity in the fruit/vegetable extract (*ie* phytochemicals) may have been important.

Fruit and vegetable research

Unsurprisingly, some researchers have begun to investigate whether diets or fruit/vegetable extracts containing high levels of phytochemicals offer superior antioxidant protection to athletes compared to conventional supplements, and the results so far look promising.

For example, a Spanish study in 2005 tested the effects of an antioxidant-rich beverage containing black grape (81 grams per litre [g/L]), raspberry (93g/L) and redcurrant

(39g/L) concentrates on exercise-induced oxidative stress in 26 cyclists[23]. Half the group were randomly allocated to receive the antioxidant beverage 15 minutes pre-exercise and during a 90-minute bicycle ergometer test at 70% VO_2max, while the other half received placebo. Measured protein carbonyl levels were 29% less in the fruit juice concentrate group. Moreover, 8-OHdG increased by 21% in the placebo group, but did not increase in the juice concentrate group.

More evidence for the benefits of brightly coloured fruits and vegetables comes from a Polish study on rowers carried out at the end of 2005, which investigated the effect of consuming an increased intake of phytochemicals called anthocyanins (contained in chokeberry juice) on the measures of oxidative stress (free radical damage at a molecular level) in rowers performing intense workouts during a one-month training camp[24].

The rowers were randomly assigned to receive either 150mls of chokeberry juice daily (containing around 34mgs of active anthocyanins) or a placebo. Before and after the supplementation period, the subjects performed a 20-minute incremental rowing exercise test starting at 40% and increasing to 90% of VO_2max. Compared to the placebo, taking the chokeberry juice produced a significant drop in the measures of free radical damage induced by the strenuous exercise, and this was confirmed by lower levels of activity of an enzyme called glutathione peroxidase, which fights oxygen free radical species in the body.

Although relatively few studies have been conducted into the protective effects of enhanced fruit and vegetable intake in athletes, those that have appear to have produced far more positive results than those using single antioxidant nutrients. But do fruit and vegetables and their extract/juices actually enhance performance?

As we reported in a recent 'What The Papers Say' (*PP* 235), US researchers have looked at the effects of drinking cherry juice on post-exercise muscle damage and soreness[25].

'Although relatively few studies have been conducted into the protective effects of enhanced fruit and vegetable intake in athletes, those that have appear to have produced far more positive results than those using single antioxidant nutrients.'

Volunteers drank 12 fl oz of either the cherry juice blend (equivalent to 120 cherries) or a placebo drink twice a day for eight consecutive days and on the fourth day they performed a bout of 2 x 20 maximum eccentric elbow flexion contractions, designed to induce muscle damage and soreness. After the exercises, strength losses averaged 22% with the placebo but only 4% with the cherry juice. Moreover, pain in the elbow flexors peaked at 24 hours with the cherry juice trial whereas it continued to increase in the placebo trial to peak at 48 hours, indicating reduced levels of oxidative damage in the cherry group.

A fascinating study meanwhile examined the relationship between reduced levels of dietary antioxidants and levels of free fatty acids in the blood (a major fuel source for humans at rest and during moderate intensity exercise)[26]. Seventeen trained athletes followed a restricted antioxidant diet (containing about a third of the antioxidant content of a 'high-antioxidant' diet) for two weeks then underwent submaximal and incremental exercise testing to exhaustion. These results were compared to an initial test conducted while they were consuming their habitual high antioxidant diet.

Although the same types and amounts of fats were consumed during both diets, the results showed that circulating blood levels of the fatty acids omega-3 and omega-6 were significantly reduced on the low antioxidant diet and, while the exercise time to exhaustion was the same for both diets, athletes reported a higher perceived rate of exertion during submaximal exercise on the low antioxidant diet.

Practical advice

How can the athlete make best use of the current antioxidant knowledge to maximise protection during training and competition? The first thing to say is that the evidence that taking single doses of antioxidant nutrients such as vitamin C or vitamin E is beneficial is rather patchy; some studies show that single nutrient supplementation can reduce levels of muscle damage and a couple of studies have indicated that

ORAC units for various fruits and vegetables

Food	ORAC units per 100g*
70% cocoa solid dark chocolate	13,500
Pomegranate	10,500
Dried prunes	5,770
Raisins	2,830
Kale	2,410
Blueberries	2,400
Garlic	2,320
Blackberries	2,040
Spinach	1,700
Brussels sprouts	1,580
Strawberries	1,540
Alfalfa sprouts	1,450
Broccoli flowers	1,290
Raspberries	1,220
Beets	1,170
Plums	949
Red pepper	810
Oranges	750
Corn	720
Cherries	670
Onion	560
Aubergine	510
Cauliflower	510
Cabbage	480
Potato	460
Sweet potato	430
Leaf lettuce	410
String bean	390
Carrot	340
Yellow squash	280
Iceberg lettuce	230
Tomatoes	195
Celery	130
Cucumber	110

*Sources: US Dept of Agriculture; Brunswick Laboratories; Journal of American Chemical Society

vitamin C may help reduce post-exercise muscle soreness. However, plenty of other studies have produced inconclusive results. Supplementing combinations of antioxidant nutrients (*eg* vitamins A, C, E and selenium) may be more beneficial as antioxidant nutrients do not work in isolation in the body but synergistically; a multi-antioxidant nutrient supplement probably makes more sense.

However, athletes should take note of the rapidly growing body of evidence pointing to the protective benefits of phytochemical-rich foods, such as brightly coloured fruits and vegetables. These not only contain antioxidant nutrients but hundreds of other naturally occurring powerful antioxidant compounds.

While the strength and depth of colour gives a very rough rule-of-thumb guide to the antioxidant activity of plant

foods, a more scientific approach has been developed that measures the Oxygen Radical Absorbance Capacity (ORAC) of foods. The higher the ORAC score, the higher the potential capacity of a food to 'quench' oxygen free radicals and render them harmless. Natural fruits typically score between 500 and 900 ORAC units per 100 grams and the US Food and Drug Administration (FDA) has recently suggested that a daily consumption of around 7,000 ORAC units may provide optimum antioxidant protection (that's around 5-10 servings of fruit and vegetables per day!).

'While ORAC scores give a better indication of antioxidant capacity of foods in vitro than mere colour, it's important to realise that the relationship between ORAC scores and antioxidant activity in the body is still poorly understood.'

However, some athletes with a high volume of training may struggle to include such large amounts of fruit and vegetables in their diet. This is because these foods are bulky and tend to contain relatively large amounts of water but low amounts of carbohydrate and very small quantities of protein. A large intake of fruits and vegetables increases satiety and could displace carbohydrate and protein-rich foods from the diet, making the task of muscle glycogen replenishment and recovery more difficult. The key then is to emphasise foods that are especially rich in antioxidant activity – *ie* with high ORAC scores. Some examples of ORAC scores are shown in the table on the previous page.

But while ORAC scores give a better indication of antioxidant capacity of foods in vitro than mere colour, it's important to realise that the relationship between ORAC scores and antioxidant activity *in the body* is still poorly understood; for this reason, it's important not to sacrifice variety by consuming just one or two high ORAC foods in order to boost ORAC unit intake. Many lower scoring foods may offer particular benefits and work synergistically with other foods. Also beware of relying on some of the very high ORAC food extracts now coming onto the market claiming 20,000 ORAC units or more per 100g. It's not yet known whether such values are accurate or if such concentrated antioxidants can be absorbed by the human body as effectively as those found in natural foods.

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Jargon buster

DNA – deoxyribonucleic acid; a double helix-shaped molecule in the nucleus of the cell, which contains the genes instructing that cell how to operate

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Could ALA and ALC combine to form the Elixir of Life?

It's a bit like a scene from a movie: the elderly scientist, working late in the lab, takes a sip of potion from a bubbling flask and undergoes a miraculous transformation as his body regains its youth and vigour. Pure fantasy? Maybe not – because that's pretty much what happened to elderly laboratory rats when they were fed two dietary supplements in a recent landmark study. According to the professor in charge of the study, 'the old rats became so full of energy, they got up and did the Macarena'!

At a Glance

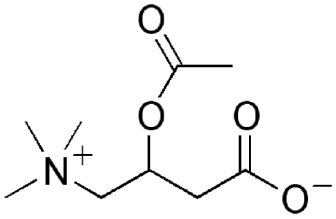
- The structure and function of ALA and ALC are outlined;
- The role of these nutrients as powerful antioxidants and enhancers of mitochondrial function is explained, as well as the relevance to athletes;
- Evidence for the benefits of ALA/ALC supplementation is presented;
- Conclusions are drawn and recommendations given.

Over the last 3 years, scientific interest in Alpha Lipoic Acid (ALA) and Acetyl L-Carnitine (ALC), the two supplements used in the studies, has exploded and a large number of studies are now underway with humans. Initial results look encouraging, but what are the implications for athletes – and can these nutrients be harnessed to improve performance?

What is ALC

Some readers may be familiar with the amino acid Carnitine, which carries fatty acids into the mitochondria (the cellular furnaces) where they are 'oxidised' for energy. As its name suggests, acetyl L-Carnitine (ALC) is very similar, consisting of the same basic amino acid structure, with an acetyl group attached *(see figure 1)*. In the body Acetyl L-Carnitine is synthesised from L-Carnitine by the enzyme carnitine acetyltransferase. Although levels tend to decrease after the age of 40, ALC is not normally considered an 'essential nutrient' because the body can manufacture all it needs.

Figure 1: chemical structure of ALC



One of the main roles of ALC is to carry fatty acids from the cytosol (the main body of the cell) into the mitochondria (the energy-producing furnaces within cells) so that these fats can be oxidised for energy. Although L-Carnitine carries out this role too, ALC also provides acetyl groups, from which acetyl-Coenzyme A (a key metabolic intermediate) can be regenerated, thereby facilitating the transport of metabolic energy and boosting mitochondrial activity.

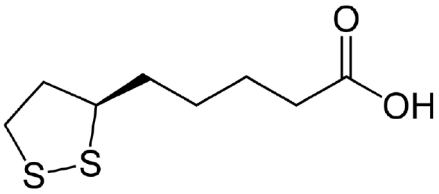
The addition of the acetyl group also endows ALC with a greater solubility in water, which enables it not only to diffuse easily across the inner wall of the mitochondria and into the cell cytosol, but also cross cell membranes in general more easily. In plain English, ALC reaches parts of the body that L-Carnitine just can't reach! In addition to its role in mitochondrial activity, ALC is involved in the production of the key brain neurotransmitter acetylcholine

and is also able to donate its acetyl group in a number of other biochemical reactions.

What is ALA?

Alpha Lipoic Acid (ALA) is a sulphur-containing antioxidant *(see figure 2)*, which occurs naturally, in small amounts, in such foods as spinach, broccoli, beef, yeast, kidney, and heart. ALA is readily soluble in water and fat, enabling it to exert an antioxidant effect in almost any part of the body, including the brain. In the mitochondria, ALA can act as both an antioxidant, capable of recycling other antioxidant nutrients such as vitamin C and vitamin E, and as a coenzyme for key metabolic enzymes involved in energy production. In addition to its role as an antioxidant, ALA also raises the levels within cells of a substance called glutathione, which is critical for neural function and aids in glycolysis, the first stages of breaking down carbohydrates for energy.

Figure 2: chemical structure of ALA



The initial excitement about ALC/ALA supplementation began when a team of researchers in California fed elderly rats both nutrients for a period of seven weeks and then compared them with young rats [1]. They were testing the theory that mitochondrial decline is caused by free radical damage (see panel below). There was already evidence that supplementation with ALC could reverse the age-related decline in mitochondrial activity in rats, increase fatty acid oxidation and boost general metabolic activity [2]. However the down side to this increased mitochondrial function was that more oxidative damage occurred [3], so the researchers

The theory of mitochondrial decline and ageing

The free radical theory of ageing is based on the idea that our cells and DNA (the latter containing the code for proper cell division and replication) eventually become irreversibly damaged by the onslaught of highly-reactive chemical species called 'free radicals'. These transient species are generated unavoidably as a by-product of aerobic (oxygen) metabolism. In other words, while oxygen provides us with the energy for life, it's also responsible for generating highly damaging chemical species that cause biochemical havoc within the cells of our bodies. The mitochondrial decline theory of ageing takes this process one step further. Mitochondria are the energy-producing furnaces in the body, whose job is to make adenosine triphosphate (ATP), the energy currency of life, by burning fuel in the presence of oxygen. But this process inevitably leaves the mitochondria themselves subject to very high levels of damaging free radical attack by reactive oxygen species. Mitochondria lack many of the defence systems found in other parts of the body, so they decline in number and efficiency with age, leading to a corresponding decline in ATP production. Reduced ATP means less energy to fuel the vital life-sustaining processes of the body, which can result in the onset of a number of disease states and processes.

'After a month on the supplements, elderly (24-month-old) and lethargic rats had more energy and did better on memory tests, while their mitochondria worked better. The researchers likened this result to a group of 80-year-old humans throwing away their walking sticks and starting to act 35 years younger!'

decided to add the powerful mitochondrial antioxidant ALA to the mix to see if they could get the best of both worlds: increased mitochondrial energy output, with reduced mitochondrial damage.

This two-pronged 'punch' to ageing cells seemed to work, with the two supplements together producing better results than either one alone. After a month on the supplements, elderly (24-month-old) and lethargic rats had more energy and did better on memory tests, while their mitochondria worked better. The decline in overall activity typical of aged rats was reversed to the level of young-to-middle-aged adult rats, aged 7-10 months. The researchers likened this result to a group of 80-year-old humans throwing away their walking sticks and starting to act 35 years younger!

These studies on rats caused a huge stir within the scientific community. Here was evidence that some of the

processes of ageing could be slowed or even reversed, and the implications for human health and performance were enormous. In the months that followed, a number of human studies were started, many of which are still under way.

However, the question of whether the benefits observed in rats might also apply to humans will not be easy to determine. For one thing, the ageing process in humans is much slower than in rats, so the seven-week supplementation period used in the rat studies described above would equate to around five years of supplementation in humans! Secondly, the amounts of ALC/ALA used in the rat studies were very high – equivalent to 50g per day of ALC and 5g of ALA for an 11-stone adult. That's around 50 times more than is typically available in ALC/ALA supplements found on the shelves of most health food stores!

One of the earliest studies examining the effect of ALC and ALA in humans was carried out at San Francisco State University in 2001. In a double-blind, placebo-controlled study lasting 17 weeks, 18 healthy sedentary men aged 60-71 were randomised to one of two treatment regimes: a placebo tablet twice a day or 1000mgs of ALC and 400mgs of ALA in two divided doses. Both groups were then asked to perform a demanding sequence of exercises, after which blood was drawn and analysed for signs of exercise-induced oxidative stress (the more efficiently and cleanly the mitochondria produce energy, the less potentially damaging oxidative stress occurs). To measure this oxidative stress, the study evaluated nine different biomarkers: ammonia, beta-carotene, glutamine, glutathione, malondialdehyde, total antioxidant status (TAS), vitamin C, vitamin E-alpha tocopherol, and vitamin E-gamma tocopherol. For eight of these nine biomarkers, a majority of subjects recorded values indicating that levels of oxidative stress had fallen when the ALC/ALA combination was taken. This was in contrast to the placebo, where no such benefits were reported.

If an ALC/ALA combination can reduce exercise-induced oxidative stress, that would be good news for athletes, who

'American researchers have demonstrated that ALA supplementation in older racehorses reduced the oxidative stress burden even under light training loads.'

are particularly vulnerable. However, because the small scale of this study made it difficult to reach statistically significant conclusions, the results were not submitted for scientific publication, which means they should be interpreted with caution.

Other human studies are also currently under way, but so far there are no published human studies available, although positive studies in animals continue to proliferate. For example, American researchers have demonstrated that ALA supplementation in older racehorses reduced the oxidative stress burden even under light training loads [4], while a number of other studies on mice and rats have shown that ALC/ALA supplementation reduces oxidative stress and improves mitochondrial function in a number of tissues, including brain, muscle and heart.

In one of these studies, researchers examined the effects of ALC/ALA therapy on ageing and hearing in rats, and found that it reduced the normal age-associated deterioration in auditory sensitivity and improved inner ear function [5]. They concluded that these improvements were related to the ALC/ALA combination's ability to protect and repair age-induced mitochondrial DNA damage, thereby boosting mitochondrial function and improving energy turnover. However, while the initial evidence from animal studies looks extremely promising, the jury is still out as far as humans are concerned.

Nevertheless, the signs are that these nutrients have a great deal to offer to athletes, as follows:

- ALC and human growth hormone (GH). A study carried out in 2001 suggests that 500mgs of ACL combined with 25-100mg of the amino acid L-ornithine, taken at bedtime after a 3-4-hour fast, can boost nocturnal growth hormone release [6]. The reasons for this are unclear, but it seems that the body's normal hypothalamic GH release includes a 'whole body' mitochondrial 'feedback loop', which is controlled by systemic ALC levels. An increase in naturally-released growth hormone could enhance the

recovery and repair processes that occur during sleep and which are vital to hard-training athletes;

- ALA and acute altitude sickness. In a study carried out on mountaineers, researchers investigated whether free radical damage to the blood-brain barrier could be implicated in the condition of acute altitude sickness [7]. Eighteen mountaineers were randomised into two groups, with one group taking a combination of ALA, vitamin C and vitamin E for three weeks before and during the ascent to a base camp at 5,180m, and the other group taking a placebo preparation. The ALA group not only experienced fewer symptoms of acute altitude sickness, but also demonstrated significantly higher resting arterial oxygen saturation levels, suggesting that ALA supplementation could be a useful aid for high-altitude endurance athletes;
- ALA and age-related inflammation. Studies carried out in vitro showed that ALA reduced the markers of chronic age-related inflammation typically seen in human cells [8];
- ALC and mood. Motivation and positive mood are crucial for athletes training and competing at the highest level. In a study carried out on mildly-depressed patients, 12 weeks of supplemental ALC not only improved scores on the Hamilton Depression Rating Scale, but also showed positive chemical changes (detected by MRI scans) in the frontal portions of the brain, suggesting that further studies are warranted [9].

For athletes in hard training, the prospect of preventing or even reversing some of the age-related decline in physical performance is enticing, holding out the promise of longer careers, including more sustained levels of peak performance. However, as is so often the case with new and unfolding nutritional research, it is difficult to make hard and fast recommendations about the benefits supplementation.

The first thing to point out is that dietary manipulation to boost these nutrients is not an option. Although ALA and

ALC are present in some foods, the amounts are very small by comparison with those used in human studies. To boost these nutrients, therefore, it is necessary to take supplements.

Secondly, it's important to realise that even if the ALA/ALC combination is eventually proven to slow down or reverse mitochondrial decline, the evidence suggests this will not lead to sudden and dramatic improvements in performance. Like the antioxidant phytochemicals in fruit and vegetables and the antioxidant vitamins and minerals, ALA/ALC is most likely to offer a long-term investment for your health.

'Don't confuse Acetyl L-Carnitine (ALC) with L-Carnitine. While it is considerably cheaper, L-Carnitine does not have the same bioavailability as ALC and has not been used in studies on mitochondrial decline.'

If you are tempted to 'jump the scientific gun' and supplement these nutrients anyway, the good news is that they appear to be relatively non-toxic, even at very high doses. The only caveat is that ALA in high doses is known to enhance sensitivity to insulin, which could lead to a drop in blood sugar. For this reason, it should be taken with food.

The bad news is that ALA and ALC not particularly cheap, and athletes need to ask themselves whether that expenditure could be more effectively allocated to improving the basic quality of their diet. As yet, there is no clear guidance on what the optimum or most cost-effective intake of ALA/ALC might be. The altitude sickness study [7] used 600mgs of ALA per day, while studies showing that ALC improves brain function in Alzheimer's patients [10] have used between 1,500 and 3,000mgs per day. However, the human study carried out in San Francisco, which used 400mgs of ALA and 1,000mgs of ALC per day was overseen by the same team that carried out the initial rat studies, so that might be a good place to start.

One final point: don't confuse Acetyl L-Carnitine (ALC) with L-Carnitine. While it is considerably cheaper, L-Carnitine does not have the same bioavailability as ALC and has not been used in studies on mitochondrial decline. And while L-Carnitine has often been promoted as a popular 'fat burning' and endurance supplement, there is actually very little evidence for this in the scientific literature. But that's another story!

Andrew Hamilton

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ALA/ALC update: why the latest research is still getting scientists excited

When scientists gave elderly rats a combination of two powerful antioxidant nutrients to try and reverse the process of mitochondrial decline, they were astonished to discover that the rats soon started to look and behave half their age! That was back in 2002, and the results prompted a flurry of research into these nutrients. But are the latest research findings on acetyl L-carnitine and alpha lipoic acid still getting scientists excited, and what are the implications for human health and performance?

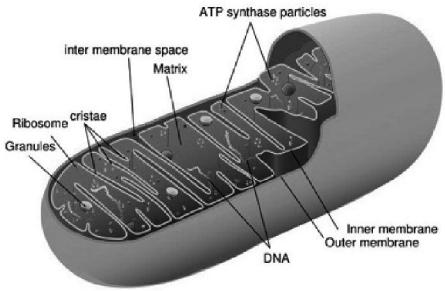
At a Glance

- The latest research on the benefits of ALA/ALC supplementation is presented;
- Ongoing recommendations to athletes are made.

As we've established in the previous article, acetyl L-carnitine (ALC) and alpha lipoic acid (ALA) are nutrients that offer (theoretically at least) huge benefits to the mitochondria in our cells. Just to recap, the mitochondria are the energy-producing furnaces in the body, whose job is to make adenosine triphosphate (ATP), the energy currency of life, by burning fuel in the presence of oxygen *(see figure 1)*. Optimal mitochondrial function therefore is vital for all the other process of life, which necessarily depend on energy.

However, as we saw in the introductory article, the process of using oxygen to produce energy results in the generation of oxygen free radicals, which inevitably leaves the mitochondria

Figure 1: schematic representation of mitochondria structure



themselves subject to very high levels of damaging free radical attack by reactive oxygen species.

Unfortunately, mitochondria lack many of the antioxidant defence systems found in other parts of the body, so over time and as a result of the continual free radical onslaught, they decline in number and efficiency. This can lead to a corresponding decline in ATP production, which in turn means less energy to fuel the vital life-sustaining processes of the body. Many scientists now believe that this general 'mitochondrial decline' plays a major role in the weakening of the body and the onset of a number of disease states and processes. The good news is that an ALC/ALA combination could provide the perfect 'antioxidant double-whammy' to help counter mitochondrial decline *(see box, opposite)*.

'Many scientists now believe that this general 'mitochondrial decline' plays a major role in the weakening of the body and the onset of a number of disease states and processes.'

What does the latest research say?

When the original studies on the effects of ALC/ALA on rats were carried out1, the results created immense interest in the scientific community and a number of other studies followed, some of which we reported on in the previous article. It's often the case in nutrition that an area of research becomes temporarily fashionable, only to fade away as less convincing results roll in from subsequent studies. The interest in ALA and particularly ALC however has most definitely not been a 'flash in the pan'; very recent research seems to indicate that these nutrients can benefit human health and performance in a

How ALC and ALA may help boost mitochondrial function

The reason why ALC and ALA are under such intense scrutiny for their potentially beneficial effects on mitochondria is because they appear to work synergistically in the following manner:

ALC – enhances mitochondrial energy production. This is because (unlike normal L-carnitine), the acetyl group confers ALC with two valuable properties:

- It makes ALC much more water soluble, which means it can easily cross the mitochondrial membranes that separate the mitochondria from the rest of the cell and reach the matrix, where all the biochemical action is taking place. This extra solubility is very important because it means ALC can more easily carry fats from the main cell body into the mitochondria, where they can be oxidised for energy.
- ALC also provides acetyl groups, from which a key metabolic intermediate in aerobic metabolism (acetyl-Coenzyme A) can be regenerated. The overall effect is to boost energy output.

ALA – protects mitochondria against free radical damage. This is because it's an extremely powerful antioxidant and being both water and fat-soluble, ALA is perfectly suited to the environment of the mitochondria. In addition, ALA also helps to recycle other nutrients and enzymes involved in protecting the mitochondria against free radical damage – important if mitochondrial energy output is being enhanced by ALC!

surprisingly large number of ways. Here then is a brief summary of some of the very latest studies to be conducted in this area:

- **ALC/ALA and learning** – Canadian researchers studied the learning abilities and performance in memory tasks of Beagle dogs administered a twice-daily supplement of ALA and ALC over approximately 2 months and found significant improvements compared to dogs given a placebo2. The researchers commented on their results that 'the long-term maintenance on ALA and ALC may be effective in attenuating age-associated cognitive decline by slowing the rate of mitochondrial decay and cellular aging'.
- **ALC and fibromyalgia** – Fibromyalgia (FMS) is a chronic syndrome characterised by widespread pain, troubled sleep, disturbed mood, and fatigue. Although

antidepressants are considered the treatment of choice in most patients, it has been recently suggested that FMS may be associated with metabolic alterations including a deficit of carnitine, and a recent Italian study seems to confirm this3. 102 fibromyalgia patients were received 1000mg of ALC daily and a 500mg muscular injection each week for 2 weeks followed by 8 more weeks of 1500mg of ALC by oral supplementation. Compared to controls who received inert placebos, 10 weeks of ALC treatment reduced depression scores, muscular fatigue and the number of 'tender points', which often characterise this condition.

- **ALC/ALA and vascular function** – In an American double-blind crossover study, the scientists examined the effects of combined ALA/ALC for 8 weeks on artery vasodilator function and blood pressure in 36 subjects with coronary artery disease4. In short, arteries are thought to become less elastic with and therefore less able to relax fully (leading to higher blood pressure) partly because of free radical damage by oxygen species (generated in the mitochondria). The researchers discovered that the ALA/ALC treatment increased the brachial artery diameter by 2.3% (ie facilitated relaxation), leading to a significant fall in blood pressure, lending support to the notion of the protective effects of ALA/ALC on the mitochondria.
- **ALA/ALC and neuronal protection** – In another US study, scientists looked at the protective effects of ALA/ALC when administered to brain cells exposed to a substance known as 4-Hydroxy-2-nonenal (HNE)5. HNE is highly reactive compound that is produced when lipids in the cells are damaged by oxygen free radical in a process called 'lipid peroxidation'. HNE in turn depletes a key enzyme known as GSH, which plays a major role in protective against oxidative damage. HNE accumulation and GSH depletion is now thought to play a major role in the development of Alzheimer's disease. The researchers discovered that treating cells with an ALA/ALC combination helped protect brain cells against the toxic effects of HNE, and

reduced a number of markers of oxidative stress.

- **ALC, high altitude and oxidative stress** – It's known that in humans, inadequate oxygen availability at high altitude can increase oxidative stress and the generation of reactive oxygen species, which may lead to memory impairment. In particular, the hippocampus area of the brain (which plays a key role in the learning and memory processes), is especially vulnerable to hypoxic (low oxygen pressure) damage. In a study using rats scientists investigated the effects of ALC supplementation on working memory before and after a 3-day exposure to a simulated altitude of 6100m6. Prior to exposure, the rats were trained in Morris Water Maze for eight days and performing the same test after the altitude simulation, the ALC treated rats showed considerably less impairment in their working memory compared to non-ALC-treated rats. Moreover, subsequent tests revealed that the ALC treated rats had lower levels of markers of oxidative stress (*eg* reduced lipid peroxidation) and better antioxidant status.
- **ALC and radiation induced oxidative stress** – A study on rats looked at the protective effects of ALC gamma radiation-induced oxidative damage in liver and lung tissue after total body irradiation7. Compared to non-ALC treated rats, those receiving ALC had increased levels of two key antioxidant enzymes (superoxide dismutase and glutathione peroxidase) before the radiation was administered. After the gamma radiation, the researchers discovered that the ALC-treated rats had lower levels of markers of free radical damage in both lung and liver tissues than the non-treated rats. The scientists also discovered that the ALC treated rats had a significant decrease in blood triglycerides, low-density lipoprotein-cholesterol (LDL) and total cholesterol, all of which indicate improved blood chemistry in respect of cardiovascular health. The researchers concluded that 'ALC could increase the endogenous antioxidant defence mechanisms in rats and thereby protect the animals from

'The ALC treated rats showed considerably less impairment in their working memory compared to non-ALC-treated rats. Moreover, subsequent tests revealed that the ALC treated rats had lower levels of markers of oxidative stress.'

radiation-induced organ toxicity'.

Conclusions

The research into ALC and ALA as powerful antioxidants is still in its infancy, but already the evidence points to a number of protective effects, both for mitochondrial energy production and against oxidative stress generally. From the athlete's point of view, all of the effects observed so far in human and animal studies indicate that in terms of long-term protection, ALA/ALC supplementation certainly deserves consideration. What's missing however are the in-depth studies to probe the potentially beneficial effects of ALA/ALC undergoing hard training and/or competition. However, based on what we know so far, the initial indications look promising. This is one of the most exciting areas of antioxidant research, and those interested in maintaining an optimum status should watch this space!

Andrew Hamilton

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New kid on the block – does quercetin have any value for athletes?

Athletes are always looking for a competitive edge and optimising nutrition is one area where significant gains can be had. Ron Maughan looks at emerging evidence for the possible benefits of a naturally occurring and powerful antioxidant called quercitin, which is attracting attention from researchers in the field of sports nutrition…

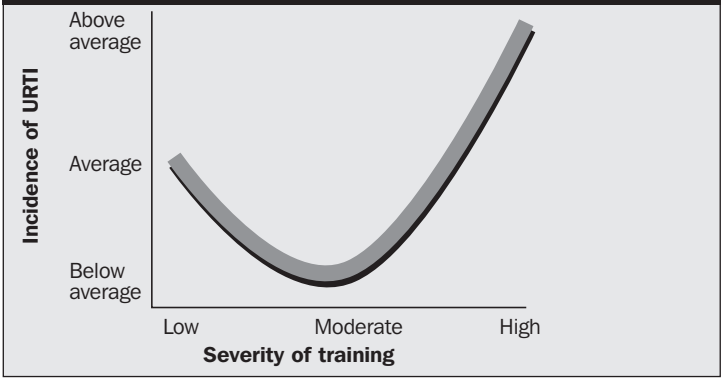
At a Glance

- The structure and function of a little known dietary antioxidant called quercitin is discussed;
- New research on the immune-enhancing benefits of quercitin supplementation for athletes is presented;
- Recommendations for athletes are given.

A recent press release on February 14, 2007 drew attention to research that has recently been conducted by Dr David Nieman and his colleagues at Appalachian State University in the USA. David Nieman is well known around the world, especially for his work on the effects of exercise and stress on the immune system. He has conducted many important studies in this area, including many field studies on marathon and ultra-marathon runners.

David Nieman was largely responsible for the first description of the so-called "J-shape" relationship between exercise and the risk of illness and infection *(figure 1)*. This is important, because it seems that the ability of our immune system to fight off

Figure 1: 'J-shaped' model of URTI incidence and training load



infection is enhanced with moderate levels of exercise, which is good news. With very high levels of exercise stress, however, athletes seem to be more susceptible to minor illness and infection. These usually amount to little more than a few sniffles, but they may be enough to interrupt training. Two or three such interruptions over the course of a season may have serious effects, especially if one of them coincides with a major race.

For many years, Nieman – along with many other research teams around the world - has been investigating the effects of a number of different nutritional interventions on the ability of the immune system to fight off infections. Most of the earlier work has focused on the traditional interventions, and from this we know that athletes who want to stay healthy should be sure to eat enough food to match their energy demands, to get enough carbohydrate and protein, and to make sure they select a wide range of foods that will provide essential vitamins and minerals in the amounts that are necessary.

More recently however, attention has turned to the effects of a range of herbal extracts and botanical compounds on the immune system. There are many such products on the market, and Echinacea has for many years been one of the top selling supplements aided at promoting a healthy immune system, but it is only one of an enormous number of such products. Many of

these herbals form an important part of the traditional medicine culture in different parts of the world, but western medicine has largely ignored them in favour of antibiotics and other more powerful pharmaceuticals.

David Nieman has gathered evidence within the last 18 months or so that Quercetin (see box), a natural antioxidant that is found in quite high levels in some plants, is able to reduce illness and maintain mental performance in physically stressed test subjects, and it is the public release of this information that led to the press announcement noted above. According to a quote from David Nieman in the press release, "These are ground-breaking results, because this is the first clinical, double-blind, randomised, placebo-controlled study that has found a natural plant compound to prevent viral illness".

'Herbals form an important part of the traditional medicine culture in different parts of the world, but western medicine has largely ignored them in favour of antibiotics and other more powerful pharmaceuticals.'

The US Army has certainly taken this seriously as they paid for the research to the tune of $1.1 million, in the hope that it will come up with something that can help to maintain the immune systems of troops who are undergoing the physical and cognitive stresses of combat. In some ways, the athlete training hard and stressed by competition faces the same problems as the soldier (though the stresses of sport really are considerably less than the life or death stresses of soldiers in Iraq, Afghanistan and elsewhere!).

Quercetin is a naturally occurring chemical found in many different fruits and vegetables that most of us eat on regular basis, including red grapes (and therefore also red wine) red apples, red onions, green tea, broccoli. Along with a whole range of other compounds, including perhaps most notably Vitamin C, it can be shown to have anti-oxidant properties.

This is important for the fruits and vegetables that produce it – it stops them from being attacked by the oxygen in the air. An apple will keep for a long time, largely because the skin acts as a barrier, protecting it from the oxygen in the air, but also from the bugs that would grow on its flesh. Cut it in half, and the exposed flesh will turn brown rather quickly as the oxygen in the air causes oxidation of various chemical components: this is soon followed by growth of yeasts and other infectious organisms. If you spread some lemon juice on the flesh of the apple as soon as

it is cut, the flesh is protected from the chemical attack but the antioxidant action of the Vitamin C in the lemon juice.

David Nieman has made the first public presentation of his latest findings at a regional chapter meeting of the American College of Sports Medicine that was held on February 9th 2007 in North Carolina. Full details were not presented, but he gave 20 cyclists a daily dose of 1 gram of quercetin for five weeks, while another 20 acted as a placebo group and were given dummy tablets. It is important to note that Vitamins C and B1 were also given.

'Even though quercetin is relatively stable during cooking, fresh fruits and vegetable are generally better sources of quercetin'

Three weeks into the study, the athletes rode a bicycle three hours a day for three days to the point of exhaustion. Monitoring of the subjects showed that 9 of the cyclists who took a placebo reported illness following the extreme exercise, while only 1 in the quercetin group reported any days of sickness. "That's a highly significant difference," Nieman said. There was also some evidence that quercetin helped maintain mental alertness and reaction time of the cyclists in a fashion similar to that of caffeine.

There have been previous reports, some of them from very reputable laboratories, that Quercetin may have some important biological actions, and that it may help protect against some cancers and against Alzheimer's diesease and other neurodegenerative disorders.

The average adult eating a normal, healthy diet consisting of Western foods consumes about 25 to 50 milligrams of quercetin a day, as well as varying amounts of other flavonoids and related compounds. People with a high energy intake (that includes most athletes training hard), and more especially those with a high intake of fruits and vegetables, will get far more than this. The supplement used in David Nieman's study contained 1 gram (1000 milligrams) –an amount that could not reasonably be obtained from the diet. You could, for example, get this from about 700 grams of onion leaves or 1 kg of bird chillies. However, no sensible person would eat these foods in such amounts!

Even though quercetin is relatively stable during cooking, fresh fruits and vegetable are generally better sources of quercetin (as well as the myriad of other biologically active

compounds that are present) than cooked or processed products because the compound is mainly concentrated in the skin of many of these rather than in the flesh. Products such as apple juice and apple sauce generally do not contain significant amounts of skin Red apples tend to have more of the antioxidant than green or yellow ones, although any apple variety is a good source of quercetin.

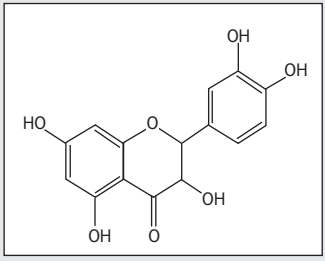
There are many other good sources for those who don't like apples. Other foods containing high levels of quercetin include onions, which have some of the highest levels of quercetin among vegetables, as well as berries, particularly blueberries and cranberries. We don't yet know if the other flavonoids found in these foods will have the same effects as quercetin, but it seems very likely that similar effects might be seen.

It is perhaps too early to know if Quercetin supplements really will benefit all athletes, as this study was a rather artificial one – no athlete normally does the sort of exercise that these volunteers were subjected to. What is clear though is that this information adds to the already convincing evidence that everyone should be sure to include lots of fruits and vegetables in their diet. Further research will tell us if supplements are helpful, or even necessary, for athletes training hard.

Ron Maughan

What is quercetin?

Quercetin is not a nutrient, but belongs to a large group of compounds known as flavonoids. It is a large molecule with a complex structure as shown below. All those hydroxyl (-OH) groups as well as the carbonyl (C=O) group mean there are lots of possibilities for chemical interactions with other ompounds. Other potentially important compounds related to Quercetin are Resveratrol, Curcumin, Turmeric and Rutin.



Magnesium – why it could be even more important for athletes than we thought

The mineral magnesium is something of a 'Cinderella' nutrient. Most sportsmen and women know that it's required for health, but few really appreciate its importance for sport performance. And now new research suggests that an optimum magnesium intake could be even more vital than we previously believed…

At a Glance

- A recap of the vital importance of magnesium in energy production and exercise performance is given;
- New research is presented indicating that magnesium may influence lactate production during intense exercise and also play a role in antioxidant protection;
- The implications of maintaining optimum magnesium intake are outlined for athletes, and dietary tips given on how to achieve this.

Think of the 'big hitters' in minerals for sports nutrition and the chances are you'll come up with iron, calcium and perhaps zinc. Yet despite magnesium's pivotal role in energy production, many coaches and athletes remain unaware of its critical importance in maintaining health and performance. To make matters worse, magnesium is a mineral that is often poorly supplied in the diet; dietary intakes of magnesium in the West have declined to less than a half of those recorded at the end of the 19th century and are still falling[1]. Moreover, many nutritionists believe that the amount of magnesium required for optimum health has been underestimated in the past, and recent research has suggested suggests that even small shortfalls in magnesium intake can

seriously impair athletic performance. This evidence includes the following *(for a full account, see PP issue 187)*:

- A study on women put on a magnesium restricted diet, which showed for a given cycling workload, peak oxygen uptake, total and cumulative net oxygen utilization and heart rate all increased significantly during the period of magnesium restriction, with the amount of the increase directly correlated with the extent of magnesium depletion (ie the magnesium deficiency reduced metabolic efficiency, increasing the oxygen consumption and heart rate required to perform a given workload)[2];
- A study of male athletes supplemented with 390mgs of magnesium per day for 25 days, which resulted in an increased peak oxygen uptake and total work output during work capacity tests[3];
- A sub-maximal work study, which showed that magnesium supplementation reduced heart rate, ventilation rate, oxygen uptake and carbon dioxide production for a given workload[4];
- A study on physically active students, which showed that supplementing with 8mgs of magnesium per kilo of body weight per day produced significant increases in endurance performance and decreased oxygen consumption during sub-maximal exercise[5].

The likely explanation for these findings lies in the fact that that magnesium is required for the activation of crucial enzymes known as ATPases, which are required for the generation of ATP, the body's 'energy currency' used for all muscular contraction (see box 'what is magnesium'). A magnesium shortfall also appears to reduce the efficiency of muscle relaxation, which accounts for an important fraction of total energy needs during exercise.

Magnesium and lactate

Since we last reported on magnesium and sports performance, very recent research has indicated that magnesium supplementation could enhance performance in a hitherto

What is magnesium and why does it matter?

Pure magnesium is the second most abundant mineral in cells after potassium, but the two ounces or so found in the typical human body is present not as metal but as magnesium ions (positively charged magnesium atoms found either in solution or complexed with other tissues such as bone). Roughly one quarter of this magnesium is found in muscle tissue and three-fifths in bone; but less than 1% of it is found in blood serum, although blood magnesium is used as the commonest indicator of magnesium status. This blood serum magnesium can further be subdivided into free ionic, complex-bound and protein-bound portions, but it's the ionic portion that's considered most important in measuring magnesium status, because it is physiologically active.

Magnesium is required for more than 300 biological reactions in the body, including those involved in the synthesis of fat, protein, and nucleic acids, neurological activity, muscular contraction and relaxation, cardiac activity and bone metabolism. Even more important for athletes is magnesium's pivotal role in both anaerobic and aerobic energy production, particularly in the metabolism of adenosine triphosphate (ATP), the 'energy currency' of the body. The synthesis of ATP requires magnesium-dependent enzymes called 'ATPases'. These enzymes have to work extremely hard; the average human can store no more than about 3oz of ATP, yet during strenuous exercise the rate of turnover of ATP is phenomenal, with as much as 15kgs of ATP per hour being continually broken down and reformed!

unrecognised way – by reducing the accumulation of fatiguing lactic acid during intense exercise.

A Turkish study carried out last year looked at the effects of supplementing 10 milligrams of magnesium per kilo of bodyweight per day in 30 subjects undertaking a 4-week jumping training program6. The subjects were separated into 3 groups:

- **Group 1**: sedentary taking magnesium supplementation only;
- **Group 2**: magnesium supplemented plus 90-120 training minutes, 5 days a week;
- **Group 3**: training-only 90-120 min 5 days a week.

Lactate levels of all groups were measured four times; at rest and exhaustion at the beginning of the study and after the end of the study. Although both the training groups had reduced lactate

levels after the training period (as would be expected – training improves lactate metabolism), the magnesium-supplemented group recorded a significantly greater drop in post-exercise lactate levels compared to the no-magnesium group. The researchers concluded that 'magnesium supplement may positively affect performance of sportsmen by decreasing their lactate levels'.

A study carried out using rats earlier this year provides further evidence of the magnesium/lactate connection[7]. In the study, Taiwanese researchers investigated the effects of administering pre-exercise magnesium (17mgs per kg of body weight) on rats forced to swim for 15 minutes. In particular, they wanted to observe the effect of the supplemented magnesium on blood lactate, glucose and pyruvate (an important intermediate compound at the 'crossroads' of aerobic metabolism).

'The researchers concluded that not only did supplemental magnesium help suppress lactate production, but that it also somehow increase glucose availability and metabolism in the brain during exercise.'

Prior to exercise, the blood levels of lactate, glucose and pyruvate were no different in magnesium-supplemented rats when compared with rats given no magnesium (control group). However, following the forced swimming, the lactate levels in the magnesium-supplemented rats rose to only 130% above pre-exercise levels compared with a 160% rise in the control group. Moreover, swimming caused brain glucose and pyruvate levels in the control group to decrease to 50-60% of the pre-exercise level; in the magnesium-supplemented rats, brain glucose levels increased to 140% of the pre-exercise level, and increased pyruvate levels to 150% of the basal level during forced swimming!

The researchers concluded that not only did supplemental magnesium help suppress lactate production, but that it also somehow increase glucose availability and metabolism in the brain during exercise. This is important because scientists now believe that the brain and central nervous system play a large role in determining the degree of muscular fatigue we feel[8]; higher brain glucose availability could in theory translate into lower levels of perceived fatigue.

An antioxidant role for magnesium?

Until recently, magnesium has had something of a Cinderella status among sports nutritionists, many of whom have not

appreciated just how important optimum magnesium status is for athletic performance. However, it now seems that magnesium also has another surprise up its sleeve, as new research indicates it may play a vital role as antioxidant, helping to protect the body from the potential ravages caused by oxidative stress (cellular damage occurring as a result of oxygen generated free radicals within the body – *see box*).

What is free radical damage?

Free radical damage describes the damage that occurs within cells (for example cell membranes and DNA) at a molecular level as a result of 'free radicals'. These free radicals are transient but extremely reactive chemical species that unavoidably occur during oxygen metabolism when fats, proteins and carbohydrates are combined with oxygen in the body to produce energy (aerobic metabolism). For this reason they are sometime called 'reactive oxygen species' (ROS) or 'oxygen radicals'.

Although our cells have very efficient antioxidant defence systems to quench and neutralise harmful free radicals, these systems are not 100% efficient, and over time biochemical damage gradually accumulates, leading to a reduction in cellular function. Most scientists now believe that accumulated cellular free radical damage lies at the heart of the ageing process and many degenerative diseases such as cancer, autoimmune diseases and Alzheimer's disease. Athletes process and use larger volumes of oxygen and at higher rates than the majority of the population; this explains why many scientists believe that they may benefit from higher intakes of antioxidant nutrients to bolster defences.

Although other minerals such as copper, zinc and selenium are known to be involved in activating enzymes that deactivate free radicals and thus protect the body, the possible role of magnesium as an antioxidant nutrient is extremely surprising to say the least. That's because unlike other antioxidants, magnesium is not 'chemically speaking' considered adept at accepting and passing on electrons (something that characterises all other antioxidant molecules). However, despite this fact, a growing body of recent evidence suggests that adequate dietary magnesium is essential for the control of oxidative stress.

One the earliest studies to indicate a possible connection between magnesium and oxidative stress, was conducted at the Military Medical Academy in Belgrade involving young military recruits exposed to chronic stress[9]. The researchers monitored markers of oxidative stress such as increased superoxide anion (free radical) concentration and malondialdehyde (a marker of cell lipid damage) in each subject and as well as magnesium status. They discovered that a low magnesium status was correlated with increased levels of oxidative stress and that the poorer the magnesium status, the higher the recorded oxidative stress.

Correlation of course isn't the same as cause, but further evidence of a link between magnesium and oxidative stress surfaced three years later in an Indian study carried out on rats that were given an injection to make them diabetic[10]. Compared to non-treated rats (controls), the diabetic rats showed a significant decrease in blood magnesium levels and an increased urinary excretion of magnesium. In addition, there was a marked increase in markers of cell damage and a corresponding decrease in the antioxidant vitamins C and E, and other protective compounds called thiols.

Interestingly however, giving the diabetic rats magnesium supplementation for four weeks restored blood magnesium levels to near normal levels and reduced markers of cell damage. Moreover, supplementing magnesium also boosted vitamin C and thiols, and increased antioxidant enzyme activity generally, suggesting a strong causal link.

Another very recent animal study examined the effect of a magnesium deficiency on free radical damage in cultured cells from chick embryos[11]. In particular the researchers wanted to investigate whether magnesium deficiency enhanced the oxidative damage caused by a naturally produced pro-oxidant (a substance that enhances oxidative stress) in animal cells called hydrogen peroxide. They found that incubating the cells in a magnesium deficient environment doubled the amount of hydrogen peroxide produced and significantly enhanced cell damage caused by this compound. This effect was probably

because the magnesium deficiency reduced the activity of an enzyme called catalase, which helps to break down and render harmless any hydrogen peroxide produced in the body. Other recent animal studies have also confirmed that low magnesium intake is strongly correlated with increased oxidative stress[12-15].

Antioxidant and anti-inflammatory activity of magnesium in humans

Animal studies are all very well, but can optimising magnesium status help protect the human body? Very few studies have been conducted in this area so far, but the evidence so far suggests this is quite likely. There's certainly a growing body of evidence that low magnesium intakes are correlated with increased inflammation, which is itself strongly associated with oxidative stress.

For example, an Italian study carried out last year of over 1,600 adults showed that low intakes of dietary magnesium were correlated to increased levels of an inflammatory marker known as C-reactive protein[16]; although this study looked at middle-aged sedentary adults, an increased tendency towards inflammation is undesirable in all populations, especially athletes, where it is generally associated with increased post-exercise muscle soreness and joint stiffness.

'The researchers showed that the higher the intake of dietary magnesium, the more positive the lung function (indicating healthier more elastic lung tissue).'

Another study looked at lung function and in particular whether dietary antioxidants might protect lung tissue against reactive oxygen species-induced injury, adverse respiratory effects and reduced pulmonary function[17]. Healthy, non-smoking freshmen students who were lifetime residents in the Los Angeles or the San Francisco Bay areas of California completed comprehensive residential history, health history and food frequency questionnaires. Blood samples were also collected and forced expiratory volume (lung power) measurements were obtained. Using a statistical technique called multivariable regression, the researchers showed that the higher the intake of dietary magnesium, the more positive the lung function (indicating healthier more elastic lung tissue).

A third study published very revcently examined the effect of

magnesium supplementation on inflammatory markers in patients with chronic heart disease[18]. The study, conducted by Israeli researchers, compared the levels of the inflammatory marker C-reactive protein in patients given 300mgs a day of magnesium citrate with a control group given no magnesium. The result showed unequivocally that the extra magnesium produced a significant drop in C-reactive protein levels, indicating reduced inflammation, so much so that the researchers commented that 'targeting the inflammatory cascade by Mg administration might prove a useful tool for improving the prognosis in conditions of heart failure'.

Implications for athletes

What does this all mean for athletes? The simple message is that a growing body of evidence suggests that maintaining an optimum magnesium status is probably even more important then we've previously realised (see box on optimising intake). Quite apart from acute performance benefits, it appears that an optimal magnesium intake is also essential for antioxidant protection and for the correct regulation of inflammation, both of which are desirable for athletes, young and old. Although more research is needed to discover the underlying mechanism behind these effects, the take home message is that you should ignore the importance of magnesium at your peril!

Andrew Hamilton

Glossary

- ***Free radicals*** – highly unstable atoms, molecules or molecular fragments containing an unpaired electron, which makes them extremely reactive and with the capacity (if unchecked) to damage cells at the molecular level
- ***Enzymes*** – large protein molecules that speed up essential biochemical reactions in the body, which would otherwise either occur too slowly to sustain life or not at all

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Optimising dietary magnesium intake

Magnesium is well supplied in unrefined whole grains, such as wholemeal bread and whole grain cereals, and also in green leafy vegetables, nuts and seeds, peas, beans and lentils (see table). Fruit, meat and fish supply poor levels, as do refined/sugary foods. Contrary to common belief, milk and dairy products are not particularly rich sources of magnesium. Magnesium is a fairly soluble mineral, which is why boiling vegetables can result in significant losses; in cereals and grains, it tends to be concentrated in the germ and bran, which explains why white refined grains contain relatively little magnesium by comparison with their unrefined counterparts.

The UK recommended intake for magnesium is set at 300mgs for men and 270mgs for women[19]. The US has recently revised its figures upwards and now recommends an intake of 400mgs per day for men aged 19-30 and 420 for those over 30; the figures for women under and over 30 are 300 and 310mgs per day respectively[20]. However, some investigators believe that even these levels are too conservative and that they should be set even higher at 450-500mg/day for all adults[21].

Table 1: the magnesium content of some common foods
(source USDA Nutrient Database)

Food	Magnesium content (milligrams per 100g)
Pumpkin seeds (roasted)	532
Almonds	300
Brazil nuts	225
Sesame seeds	200
Peanuts (roasted, salted)	183
Walnuts	158
Rice (whole grain brown)	110
Wholemeal bread	85
Spinach	80
Cooked beans	40
Broccoli	30
Banana	29
Potato (baked)	25
White bread	20
Yoghurt (plain, low fat)	17
Milk	10
Rice (white)	6
Cornflakes ('Frosties' or 'Honeynut')	6
Apple	4
Honey	0.6

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