NUTRITIONAL SUPPLEMENTS

boosting your performance

A SPECIAL REPORT FROM

PEAK PERFORMANCE

The research newsletter on stamina, strength and fitness

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A CIP catalogue record for this book is available from the British Library.

Printed by: Baskerville Press Ltd
6-8 Newton Road, Salisbury, Wiltshire SP2 7QB

Published by Peak Performance Publishing

Peak Performance Publishing is a trading name of Electric Word plc
Registered office: 67-71 Goswell Road, London, EC1V 7EP
Tel: 0845 450 6402
Registered number: 3934419

ISBN: 1-905096-05-4

Publisher Jonathan A. Pye
Editor Isabel Walker
Designer The Flying Fish Studios Ltd

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From the editor

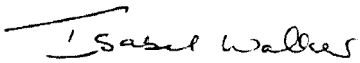
Recently I experienced the chastening – not to mention hugely expensive – consequences of filling my brand new diesel-powered car with petrol. The vehicle struggled on manfully for a few miles before spluttering to an undignified halt. They say I was lucky the engine did not blow up completely.

I am sure you spot the analogy coming: just as an engine is only as good as the fuel pumped into it, so an athlete's performance depends on correct, balanced and adequate nutrition. All the speed, torque and conditioning in the world will not compensate for the wrong diet.

That's why we make no apology for devoting an entire special report to the subject of nutrition – and specifically to the role of nutritional supplements, which often have an undeservedly bad name, partly because of the exaggerated claims made by manufacturers but also, more recently, because of evidence that they can give rise to false positive drug tests.

All the feature material for this special report comes from PP's resident nutrition guru Andrew Hamilton, who charts an expert course through the key areas of interest and debate, from essential fats (good for metabolism) to probiotics (promising for boosting immunity); from iron (deficiency more prevalent than you might think) to antioxidants (potentially damaging in more-than-modest doses); from conjugated linoleic acid (good for boosting lean muscle mass) to chromium (essential for insulin function).

I hope you enjoy reading this fascinating report and that it helps you chart your own course through the complex, confusing and often contentious minefield of nutritional advice on offer to athletes.



Isabel Walker
Editor

...Foods and supplements to build and maintain it

Athletes constantly live on a knife-edge between overreaching and overtraining. That new programme might shed valuable seconds off your PB, but if it proves too much and you come down with a viral infection you stand to lose fitness, not gain it. Building and maintaining immunity should be thought of as a vital part of any athlete's programme, particularly as the existence of post-exercise immunosuppression is now well-established[1,2,3]. But what can athletes do to maximise their immunity and reduce the risk of infection and illness?

The human immune system consists of a complex array of different elements, whose job is to work synergistically to recognise, attack and destroy foreign invaders. In very simple terms, there are two lines of defence. The first is the innate immune system, consisting of barriers designed to prevent foreign agents from infecting the body. These include:

- physical barriers, such as the skin and epithelial tissues of the lungs, nose and intestinal tract;
- chemical barriers, such as the high acidity of the stomach;
- cellular barriers, such as phagocytic cells whose job is to engulf invaders.

If the first line of defence is breached, an infection occurs and then the 'acquired' immune system kicks in to fight the infection and destroy it. The acquired system employs a number of different cell types working in cooperation to help the body recognise and defeat the invaders.

For example, cells known as monocytes or macrophages ingest any foreign material and then present it to other cells, known as lymphocytes. When this material is presented to a B-lymphocyte immune cell, the lymphocyte is signalled to

proliferate and produce antibodies that will specifically bind to the foreign invader. These antibodies attach themselves to the surface of the invader (usually bacteria or a virus-infected cell) and act as 'labels', effectively telling the other cells of the immune system that these invaders are foreign and need to be destroyed. This destruction can be by means of macrophages engulfing the invader, by an attack from other immune cells known as 'natural killer cells', or by immune proteins collectively called 'complement', which can punch holes in the bacterial wall.

The phenomenon of post-exercise immuno-suppression is now well-documented in athletes

If the foreign material is presented to a T-lymphocyte immune cell, it too proliferates; some T-lymphocytes (CD8+) become activated to kill any cells carrying this foreign material, while others (the CD4+ T-cells) secrete biochemical substances (such as interleukins and cytokines) which boost the activity of killer cells.

Wide array of defence mechanisms

The point is that your immune system contains a large number of functionally different cells and a wide array of defence mechanisms. Because of this, it's actually quite difficult to accurately assess the impact of nutrition on immunity, especially as there are no instruments that can predict the cumulative effect of several small positive changes in different immune system parameters[4].

The phenomenon of post-exercise immunosuppression (PEIS), in which certain elements of the immune system become temporarily depressed after heavy bouts of exercise, is now well-documented in athletes, linked with an increased incidence of infection, particularly of the upper respiratory tract[5,6]. In particular, it appears that during recovery from prolonged, intense exercise the number of lymphocytes in the blood is reduced below resting levels and the function of natural killer and B cells is impaired[7,8].

Strenuous exercise also seems to inhibit innate immunity by reducing mucosal protection[9]. And there is evidence that this drop in immunity may worsen the disease outcome when

exercise is performed during the incubation period of an infection[10]. The consensus of scientific opinion is that PEIS occurs mainly as a consequence of the increased secretion of stress hormones, such as adrenaline and cortisol, during vigorous and prolonged exercise, particularly as these types of hormones are known to suppress immune function.

The role of glutamine

In recent years, the role of an amino acid called glutamine has come under intense scrutiny. Several studies have demonstrated a fall in plasma glutamine levels following vigorous exercise, and doctors had long been aware that this also occurs as a consequence of other stressful events, such as trauma and burns, which lead to immunosuppression.

When it was subsequently shown that many immune cells have an unusually high capacity to utilise glutamine[11] and that (unlike most cells in the body) these immune cells are unable to synthesise glutamine *in situ* and therefore require a constant supply from blood plasma[12], glutamine depletion was considered by many to be an obvious trigger for the PEIS commonly observed after acute, exhaustive exercise. This theory was supported by *in-vitro* studies showing that glutamine stimulates the activity of certain immune cells, such as lymphokine-activated killer cells[13].

Given these findings, scientists quickly began to speculate that PEIS might be prevented if extra glutamine could be administered after exercise – hence the proliferation of glutamine supplements. However, more recent research has thrown this idea into doubt. While a study on glutamine-supplemented marathon runners found they experienced only half the rate of respiratory tract infections of unsupplemented controls[14], the same scientists also found that that glutamine supplementation after a marathon did not influence the lymphocyte distribution or the concentration of other immune proteins.

Meanwhile, glutamine-supplemented cyclists who performed 60, 45, and 30 minutes of exercise at 75% of maximal oxygen consumption, separated by two-hour rest periods,

showed no increase in immune activity by comparison with unsupplemented controls[15].

In a nutshell, boosting plasma glutamine concentration did not prevent post-exercise immunodepression, a finding which has since been confirmed by a further study on glutamine-supplemented marathon runners[16]. Scientists now believe this is because the post-exercise drop in plasma glutamine is relatively small, to around 80-90% of resting values, by comparison with the drop in severe burns patients, whose glutamine levels can fall to below 40% of normal.

Although there is a reduction in circulating glutamine after exercise, there still seems to be enough of the stuff for the immune cells to function normally. Moreover, while plasma glutamine levels undoubtedly fall, there is evidence that the intra-cellular levels of glutamine in important immune cells in the blood actually rise[17]. Glutamine, it seems, is not the magic immune bullet that athletes had hoped it would be.

The low-carb diet fashion

Recently, carbohydrate-bashing has become fashionable, and it seems like everyone is now flourishing on a low-carbohydrate diet! But, quite apart from the weight-loss myths pedalled in the press, athletes would seem to have more reason to ignore this fashion than most because there is strong evidence that intense training coupled with a low-carb diet is the perfect recipe for immunosuppression!

The PEIS observed after intense training appears to occur mainly as a result of the secretion of stress hormones into the body, and scientists have proposed that any nutritional manipulation capable of reducing this stress hormone release should limit this immune suppression[18]. The latest research suggests not only that limiting carbohydrate intake induces a greater release of stress hormones during exercise, but also that athletes can manipulate their carbohydrate intake to ameliorate PEIS.

Studies have shown that when athletes train in a glycogen-depleted state after spending several days on low-carbohydrate

diets (less than 10% of dietary intake from carbohydrate), the release of stress hormones, such as adrenaline and cortisol, is exaggerated by comparison with normal or high-carbohydrate dietary conditions[19,20].

Moreover, this enhanced stress hormone release is linked to a decrease in immune function; for example, just one hour's exercise at 75% VO2max in a glycogen-depleted state resulted in a significantly bigger fall in circulating immune lymphocytes than the same amount of exercise on a high-carb diet[21]. The good news for athletes is not just that high-carbohydrate diets can reduce the stress hormone response but also that taking in carbs during exercise reduces stress hormone production which, in turn, seems to ameliorate PEIS.

Anti-viral defences

In a landmark study carried out in 2003, cyclists were fed differing amounts of carbohydrates during 2.5 hours of strenuous cycling[22]. Taking in 30-60g of carbohydrate per hour in the form of a 6% carbohydrate drink was found to prevent the decrease in an important type of immune cell, known as interferon-g-positive T-lymphocytes, experienced by a control group taking placebo. The researchers also discovered that the carbohydrate-supplemented group showed no measurable drop in production of an active chemical (known as interferon-g) that these T-cells secrete when stimulated. Interferon-g production is critical to anti-viral defence, and scientists now believe that suppressed production after strenuous exercise may be an important factor in the increased risk of infection.

These results are supported by another recent study from the US, in which two groups of runners were asked to perform a three-hour treadmill run at 70% VO2max, one ingesting a carbohydrate drink and the other placebo[23]. By comparison with the control condition, carbohydrate ingestion reduced the rise in plasma concentrations of a number of cytokines – very small protein molecules secreted by cells of the immune system, which regulate the intensity and duration of the immune

response. The lower levels of cytokines measured in the carbohydrate-fed runners appeared to indicate reduced 'immune stress'.

However, it remains to be established whether carbohydrate ingestion during training and competition can reduce the incidence of upper respiratory tract infection (URTI). The American researchers mentioned above have noted a beneficial trend in a study of 98 marathon runners, but their results did not reach statistical significance, indicating the need for further, larger studies[24].

Vitamins, minerals and immunity

The role of essential vitamins and minerals in maintaining immunity has long been recognised; deficiencies of the any of the vitamins A, E, folic acid, B6, B12 and C can impair immunity, as can deficiencies of the minerals iron, copper, selenium, zinc, magnesium and manganese[25]. But are there any nutrients that can offer extra immune support when taken in higher quantities than their current UK Reference Nutrient Intake (RNI) values?

The obvious candidate is vitamin C; ever since Dr Linus Pauling carried out his original studies into vitamin C, the notion that it might be beneficial for combating URTIs, such as the common cold, has become widely accepted. But while vitamin C is found in high concentrations in immune cells such as leucocytes and has also been implicated in a number of immune functions, such as the promotion of T-cell proliferation and inhibition of virus replication, the research on athletes and immunity has produced very mixed results.

Two studies carried out in the 1990s initially provided strong support for a protective effect of high doses of vitamin C in athletes. In the first, two groups of ultra-marathon runners were supplemented for three weeks leading up to a 90k race, one group taking 600mg of vitamin C per day (15 times the current RNI of 40mg) and the other taking placebo[26]. In the fortnight after the race, the incidence of URTIs in the supplemented groups was half that of the controls.

A follow-up study carried out three years later supported these results[27]; ultra-marathon runners were split into four groups, one given 500mg per day of vitamin C, the second receiving the same plus 270mg of vitamin E, the third 300mg of vitamin C, 200mg of vitamin E and 18mg of beta-carotene, and the fourth receiving just placebo. After the 90k race, the runners receiving the highest doses of vitamin C showed the lowest incidence of URTIs, regardless of whether they were also receiving the antioxidant nutrients (vitamin E and beta-carotene), clearly pointing to vitamin C as the protective nutrient.

The problem is that other studies have not been able to replicate these findings. For example, no immunity benefits were found in an American study when runners were supplemented with 1,000mg of vitamin C per day for eight days before completing a 2.5-hour run[28]). And in a very recent placebo-controlled study conducted by the same researchers, 1,500mg of vitamin C taken daily for seven days before and during an ultra-marathon did not positively affect any aspect of immune function[29].

A number of herbs are reputed to stimulate immunity, but in recent years it is echinacea purpurea that has become particularly popular among athletes,

Does echinacea work?

A number of herbs are reputed to stimulate immunity, but in recent years it is echinacea purpurea that has become particularly popular among athletes, despite a lack of evidence of its effectiveness against PEIS. There's no doubt that in the laboratory echinacea does demonstrate a significant effect on a number of immune cells, especially on macrophage activity[30], as well as on the activation of some leucocytes and natural killer cells[31]. But how, if at all, does this translate into immune protection for athletes?

The evidence, unfortunately, is rather disappointing. While some small-scale studies have indicated that, in those already infected, the severity and duration of acute URTIs may be modestly reduced with echinacea, three recent double-blind placebo-controlled studies found no evidence of immunostimulation[32,33,34].

Some researchers have suggested that the problem with echinacea studies is that many commercially available echinacea products do not possess enough active constituents to exert a definitive clinical effect. To get around this problem, a very recent double-blind, placebo-controlled study used a formulation prepared from freshly harvested echinacea plants, which contained the suspected active constituents (alkamides, cichoric acid, and polysaccharides) at known and high concentrations[35].

A group of 282 healthy adults with a history of two or more colds in the previous year were randomised to be treated with either echinacea or placebo at the first onset of cold symptoms. During the study period, 128 subjects contracted a common cold (59 on echinacea and 69 on placebo). But the echinacea group reported less troublesome symptoms and responded faster to 'treatment' than the controls. The researchers concluded that their results pointed to the need for more, larger scale studies using standardised extracts.

Probiotics and health

It was almost a century ago that the Nobel prize winner Elie Metchnikoff carried out his research into fermented milk products, such as live yoghurt, and suggested that, far from being inevitably detrimental to health, bacteria could play an important role in maintaining it. Since then, a wealth of research has accumulated, confirming that certain types of bacteria are beneficial to human health when ingested. These so-called 'probiotics' can be defined as 'live microbial feed supplements, which beneficially affect the host by improving its intestinal microbial balance'.

Although probiotics have been shown to produce a number of gastrointestinal health benefits, they've never been perceived as 'sexy' by the athletic community. But that might be about to change! Over the last decade there has been an explosion of research into the immunostimulatory properties of probiotics – and the results are impressive. A recent meta-analysis of relevant studies examined the scientific literature on probiotics

and immunity *in-vitro*, in animals and in humans[36]. Of these, 48 reported positive immunostimulatory effects, 17 *in-vitro*, 21 in animals and 10 in humans.

Positive research findings

At the time of writing there were no published studies on the possible benefits of probiotics for athletes, but things are moving quickly and two studies on this very topic were due for publication later in 2004. In the first, 118 German sports performers were screened for levels of an immune protein called secretory immunoglobulin A (sIgA), with 52 found to have below-average levels[37]. These 52 were then split into three groups for four weeks, the first treated with a probiotic food supplement containing a proprietary blend of beneficial bacteria (Lactobact omni FOS, manufactured by Winclove Bio Industries), the second a zinc and magnesium supplement and the third both together. When their faeces were analysed, only those taking the probiotic were found to have significantly increased levels of sIgA.

The second study, also carried out in Germany, examined the effects of probiotics on post-exercise immunosuppression[38]. A total of 44 endurance athletes were split into two groups, one to receive probiotics, the others to act as controls. After four weeks of supplementation with a probiotic blend (Lactobact omni FOS), the athletes were tested after a 60-minute endurance session. As expected, faecal microflora was improved in the probiotic group, but the researchers also discovered that this group experienced a lesser post-exercise decrease in the level of circulating natural immune killer (NK) cells than the controls, with a faster return to pre-exercise NK cell levels – an indication that probiotics may be able to reduce PEIS. As with all unpublished studies, these results should be interpreted with caution, but if they are confirmed the role of probiotics in protecting the health of athletes looks promising.

In summary, here is the best advice for athletes wishing to maintain maximum immunity:

- *Carbohydrate intake* – The normal diet should provide ample carbohydrate at all times, accounting for 60% or more of total calories. Low-carb diets such as Atkins or Zone should be avoided. For longer (90-plus minutes) or very intense sessions, 500-1,000ml of carbohydrate drink containing 60g of carbohydrate per litre should be ingested every hour;
- *Diet quality* – Immunity can be adversely affected by any number of nutrient deficiencies. Athletes should ensure that their diet is rich in whole unprocessed foods, fruits and vegetables, contains adequate high-quality sources of protein and is low in fatty, sugary, fast or processed food. A broad-spectrum multi-vitamin/mineral supplement may be beneficial in preventing a nutrient shortfall, but large doses of any single nutrient should be avoided as this could create imbalances leading to impaired immunity;
- *Vitamin C* – The evidence is too mixed for a firm recommendation but, given its low toxicity and cost, athletes wishing to take a modest supplement (200-1,000mg per day) have little to lose;
- *Glutamine* – Although beneficial in the clinical setting, there's little hard evidence that it offers immune protection to athletes;
- *Echinacea* – Athletes who contract a URTI may find that taking a standardised echinacea preparation shortens its duration. However, while echinacea did not appear on the IOC's banned substances list for 2004, those subject to drug-testing should be aware that all herbs contain a number of biologically active ingredients which, under certain circumstances, may inadvertently produce a positive result;
- *Probiotics* – Although the early indications are promising, very little data exists on the benefits of probiotics for athletes. Foods like live yoghurt and other fermented products can be included in the diet if desired, especially as they are also rich in other nutrients; indeed, they are recommended after antibiotic treatment. To date there's insufficient evidence of the benefits of supplementing the diet with probiotics, although this may change in the near future;

- *Lifestyle* – Athletes should ensure they get plenty of sleep and relaxation, minimising fatigue and emotional stress where possible. Good hygiene is also important, with regular hand washing recommended to reduce the risk of transferring virus particles to the mucous membranes of the eyes, nose and throat.

Andrew Hamilton

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...The answer to most athletes' prayers – helping to conserve carbohydrate while shedding fat

Nature or nurture? There is an age-old debate about whether performance is primarily 'in your genes' or develops in response to training. The common consensus is somewhere in between: that we inherit a set of genes which determine our potential, but it's our training and nutrition that allow us to reach that potential. However, new evidence suggests this fatalistic approach to our genetic make-up is misplaced; fascinating research is emerging from the world of nutrition to suggest that essential fats in our diet can exert significant control over key metabolic genes in our cells, particularly those involved with fat storage, fat burning and glycogen synthesis.

In plain English this means that, while you might not be born with the ideal genetic make-up for your chosen sport or event, correct fatty acid nutrition could help to 'reprogramme' your genetic code!

There are two principal essential fats: alpha-linolenic acid (sometimes called omega-3) and linoleic acid (omega-6). These two fats are essential because their chemical structure means that they can be used to make hormone-like substances called prostaglandins, which go on to regulate a host of other functions in the body. However, these fats cannot be synthesised by the body, which is why we rely on getting them 'ready-made' from the diet.

The complex structure of the fats also makes them very chemically reactive; put simply, they readily undergo chemical change and 'fall apart' when exposed to heat, light or air. This

means that storing, cooking or processing foods rich in essential fatty acids (EFAs) inevitably leads to a loss in nutritional value. The problem is that we need more of these EFAs per day than any other single nutrient – measured in tablespoons, not milligrams! And the task of obtaining enough of them in unadulterated form in today's world of tinned, dried, frozen, fast and processed food is a major challenge.

Storing, cooking or processing foods rich in essential fatty acids (EFAs) inevitably leads to a loss in nutritional value

The best dietary sources of EFAs are nuts, seeds, fatty fish and unrefined whole grains. However, a glance at the table opposite shows that, while the omega-6 fatty acid is quite abundant, omega-3 is more difficult to obtain. Unless your diet contains significant amounts of seeds and whole grains, it's likely you'll be falling short of your optimum omega-3 intake. And simply using more bottled oils, such as soy, rape and walnut oils, on salads and in cooking, may not be the answer either. Commercial oils are inevitably refined, processed and stored, which means that the essential fatty acid content will be partly degraded.

Fatty fish, such as mackerel, herrings, sardines, trout and salmon, are rich in two different kinds of omega-3 fats – eicosapentanoic acid (EPA) and docosahexaenoic acid (DHA) – which can help to supplement the role of alpha linolenic acid in the body. Notice, though, that olive oil is devoid of omega-3 and very low in omega-6. Contrary to popular belief, olive oil is a very poor source of EFAs!

EFAs and heart disease

The role of EFAs in human nutrition has long been recognised; dietary omega-3 and omega-6 fats are needed for the synthesis of prostaglandins, which help regulate certain aspects of metabolism, such as blood viscosity, inflammatory processes, blood cholesterol and fat levels, and water balance. Additionally, it is now widely accepted that a low ratio of EFAs to saturated fatty acids is associated with an increased risk of coronary heart disease (CHD).

However, more recent research on EFA nutrition has yielded some intriguing new findings. One of these is that increased intakes of these essential fats appear to reduce tissue levels of

Dietary sources of EFAs		
Food	**Omega-3 (grams per 100g)**	**Omega-6 (grams per 100g)**
Flax	20.3	4.9
Hemp seeds	7.0	21.0
Pumpkin seeds	3.2	23.4
Salmon	3.2	0.7
Walnuts	3.0	30.6
Rape seed	2.1	9.0
Herring	2.0	0.4
Soybeans	1.2	8.6
Butter	1.2	1.8
Olive oil	0.6	7.9
Wheat germ	0.5	5.5
Sunflower seeds	0	30.7
Almonds	0	9.2
Olives	0	1.6

triglycerides (stored fats), which improves the sensitivity of insulin (the hormone that drives amino acids and glucose into muscle cells), so reducing the risk of obesity and CHD[(1)]. Initially, these beneficial effects of EFAs were thought to be due to changes in the fatty acid composition of the cell membranes, leading to subsequent alterations in hormonal signalling. However, when researchers dug a little deeper it became apparent that something else was going on.

They discovered that these fats, particularly those of the omega-3 family, play essential roles in the maintenance of energy balance and glucose metabolism. In particular, they observed a phenomenon known as 'fuel partitioning', whereby dietary EFAs were able to direct glucose (from digested carbohydrates) towards glycogen storage while at the same time directing other fatty acids in the body away from triglyceride synthesis (*ie* fat storage) and towards fatty acid oxidation!

The fuel partitioning effect

In addition, these studies suggested that omega-3 fatty acids have the unique ability to enhance thermogenesis (the burning of excess fat to produce heat), thereby reducing the efficiency of body fat deposition[2-7]. In simple terms, this fuel partitioning phenomenon appears to conserve carbohydrate while simultaneously shedding fat – exactly what most athletes would give their right arm for!

Further study of this fuel partitioning effect led to the discovery that the EFAs were somehow boosting the production of enzymes involved with fatty acid oxidation (such as carnitine palmitoyltransferase, which helps transport fatty acids into the mitochondria of the cells for burning) while at the same time down-regulating the production of enzymes involved in fat synthesis, such as fatty acid synthase[8-12].

At first it was assumed that this 'up-regulation' of fat burning/glycogen synthesising enzymes and 'down-regulation' of fat storage enzymes occurred through hormonal signalling; in other words that the EFAs were somehow altering the cell membranes, causing a change in chemistry and leading to altered enzyme production by the genes responsible. However, these changes in gene transcription occur too quickly to be explained in this way; there seemed to be a much more direct effect. And eventually researchers discovered, to their amazement, that these EFAs were able to control gene expression directly via a steroid-like substance called PPARa[13,14].

PPARa is known as a 'lipid-activated transcription factor'. This means it switches on key genes by binding to DNA, but only when it has been activated itself by binding to lipids such as EFAs. And it turns out that the genes it switches on are precisely those which code for enzymes involved in fat burning! Not only was this a remarkable discovery in itself, it was also the first time science had clearly demonstrated that nutritional components of the diet can exert direct control over the function of genes.

Although PPARa was believed to act as a 'master switch', helping to switch on genes involved in fatty acid oxidation and

switch off those involved in fat storage, more recent research has demonstrated that the down-regulation of fat storage enzymes occurs because EFAs impair the release of another group of steroid-like substances called 'sterol response element binding proteins', or SREBPs for short![15,16]. One of these (SREBP-1) helps to switch on the gene that codes for a fat synthesis enzyme called fatty acid synthase. However, a different type (SREBP-2) is a regulator of genes coding for proteins involved in cholesterol synthesis, which probably explains why healthy intakes of the EFAs reduce cholesterol[17,18].

EFAs and performance

The thermogenesis effect of omega-3 fats mentioned earlier is now believed to occur as a result of their ability to activate a gene that codes for a protein called 'uncoupling protein-3'[13]; this protein allows the energy derived from the oxidation of fatty acids to be dissipated as heat, rather than coupled to the metabolic processes in order to do work.

The role of EFAs in modifying gene expression and stimulating the phenomenon of fuel partitioning now appears to be scientifically beyond doubt. But how does this translate into athletic performance? Can athletes expect to benefit from metabolic changes brought about by higher intakes of EFAs? Anecdotal reports of increased human performance on high EFA diets abound, but this is a relatively new area of research and hard scientific evidence is thin on the ground.

Subjective improvements in endurance

In 2001 Dr Udo Erasmus (considered by many to be a crusader for the health benefits of EFAs) carried out a study with 61 Danish athletes. After eight weeks of supplementation with a 2:1 blend of omega-3/omega-6 oil, the athletes (selected from a wide variety of sports) showed a significant increase in HDL (healthy) cholesterol levels, a more favourable ratio of HDL to LDL (unhealthy) cholesterol and lower levels of fasting triglycerides. A large percentage of the group also reported subjective improvements in endurance and recovery.

However, subjective measurements are notoriously prone to the placebo effect, which means that the results should be interpreted with caution.

Meanwhile, a well-controlled study carried out on football players in 1997 showed no increase in VO2max or anaerobic threshold when diets were supplemented with 2.5 grams per day of omega-3 from fish oils[19]. However, the dose of omega-3 used was very small, and the fuel partitioning effects of EFAs described above could only be expected to improve endurance and reduce body fat – parameters which were not assessed in this study.

Turn to animal and *in vitro* studies, though, and things begin to look much more promising. In a study carried out last year, scientists studied the effects of omega-3 fat supplementation on swimming performance in rats[20]. By comparison with a control group of unsupplemented rats, there was a 300% rise in the 'swimming muscle' levels of FABP, a protein that binds fatty acids and transports them to the mitochondria for oxidation, but no increase in muscle triglycerides. The researchers concluded that this effect was probably due to an up-regulation of the fatty acid metabolism genes via the PPARa mechanism discussed earlier.

Effects on muscle function

In a study on rat muscle fibres, high omega-3 and omega-6 diets produced 16-21% more muscle tension and up to 32% greater endurance during high frequency stimulation[21]. Moreover, when these rats resumed their standard diets for a period of six weeks, their muscle function returned to the level of un-supplemented rats.

Rat studies on EFAs and body composition also look promising. In a Japanese study, very young rats were fed for four months on a diet containing one of the following[22]:

- 12% perilla oil (very rich in omega-3);
- safflower oil (very rich in omega-6);
- olive oil (rich in mono-unsaturates);
- beef fat (rich in saturated fats).

The amount of food consumed and the weight gained was the same in all four groups, but the amount of fat stored, the number of fat cells and fat cell volume were all significantly lower in the omega-3 and -6 groups. Furthermore, the genes involved in fat cell differentiation were significantly down-regulated in the omega-3 group by comparison with the olive oil and beef fat groups!

Intriguingly, some human research points to a synergistic effect between endurance training and EFA metabolism. In 2003, scientists studying the phenomenon of 'uncoupling' in human muscle mitochondria found (as expected) that the genes coding for uncoupling proteins (the ones that stimulate thermogenesis via uncoupled respiration) were activated by omega-3 fats. What surprised them, however, was that after endurance training the stimulating effect of omega-3 fats was even stronger. In other words, omega-3 oils seem to stimulate thermogenesis most effectively in muscles that are endurance-trained!

The simple fact is that there is very little consensus among nutritionists about how much omega-3 and omega-6 oils are needed in total for optimum health

So where does all this leave athletes? Although there's a dearth of well-controlled double-blind studies on the interaction of EFA and genes in humans, there's no doubting the weight of evidence accumulating from animal and *in-vitro* studies. Numerous studies have demonstrated that western diets containing significant amounts of processed foods and saturated or chemically-altered fats are very low in EFAs, particularly omega-3 fats, creating an unbalanced ratio of dietary omega-6:omega-3[23]. Typically, this ratio in modern diets is between 10:1 and 25:1, although the World Health Organisation recommends a ratio of between 5 and 10:1. Some nutritional researchers recommend an even higher proportion of omega-3, with as much as a third of total EFA intake from omega-3.

Conservative dietary recommendations

Current UK dietary recommendations are for around 6% of calorie intake to come from polyunsaturated (essential) fats, with around 0.2g per day of omega-3 fats[24]. However, this figure seems extremely conservative; assuming a total calorie intake

of around 2,000 per day, it would equate to 13g of omega-6, giving an omega-6:omega-3 ratio of over 60:1!

The simple fact is that there is very little consensus among nutritionists about how much omega-3 and omega-6 oils are needed in total for optimum health, and about the ideal ratio between the two. Pioneers in the field of fatty acid nutrition, such as Dr Erasmus, recommend around 9g per day of omega-6 and 6g per day of omega-3 oils for general health (1.5:1 omega-6:omega-3). This sits well with recommendations from the US National Cholesterol Education Program Diet and American Heart Association that no more than 30% of total calorie intake should come from fat, of which polyunsaturates (omega-3 and 6) should constitute 10% – *ie* around 20g per day in total.

On the available evidence, this would seem a very good place to start. For the fuel-partitioning effects mentioned earlier, higher intakes of EFA might be required; the animal studies demonstrating this effect supplied EFAs at between 10 and 20% of total calorie intake (22-44g per day in a 2,000 calorie/day diet). The studies on Danish athletes carried out by Dr Erasmus supplemented around 20g of omega-3 and 10g of omega-6 per day.

It's all too easy to fall short of even the minimum intakes of EFAs required to maintain optimum health, let alone to produce any of the potential benefits discussed here. And this is not just down to the popularity of processed and refined foods. In their efforts to follow a healthy 'low-fat' lifestyle, many people, including athletes, have thrown out the 'EFA baby' with the bathwater!

Below are some dietary tips which can help to boost your EFA intake.

- Use fresh seeds sprinkled on salads, especially hemp, pumpkin and sunflower;
- Use nuts in salads or mixed with raisins as snacks, especially walnuts, pecans and hazelnuts;
- Switch to wholemeal bread – the wheatgerm in whole wheat is a good source of EFAs;

- Eat whole grain breakfast cereals, such as Shredded Wheat, Weetabix and oat flakes, rather than refined cereal, such as cornflakes;
- Use brown rice and wholemeal pasta instead of white varieties;
- Use a cold-pressed seed oil in salad dressings, but make sure that it is fresh and has been packaged in an oxygen-free container that is also opaque to light;
- Eat fatty fish at least once a week. If you can get fresh mackerel, herring or unfarmed salmon and trout, so much the better;
- Don't rely too heavily on low fat/diet foods and shakes for your calories – these are nearly all devoid of EFAs;
- Choose free range chicken and wild meats where possible – these generally contain higher amounts of EFAs than their intensively-reared counterparts;
- Choose organic free-range eggs if you can get them. Free foraging hens fed on natural foods lay eggs containing up to 30% of the fat as EFAs.

If you choose to supplement EFAs additionally, the best way to do this is with a bottle of proprietary seed oil blend. These tend to contain around two thirds omega-3 to one third omega-6. This is the reverse of the recommended dietary ratio, the idea being that you need extra omega-3 because it's harder to obtain from normal dietary sources than omega-6. Flax seed oil is also an excellent source of omega-3 but contains very little omega-6. Whatever you choose, it should be fresh and packaged in an oxygen-free container that is also opaque to light. Ideally, the oil should have been kept refrigerated since production and should be stored in your fridge and used within four weeks of opening.

Remember that EFAs (especially omega-3) are chemically very fragile and spoil rapidly if not stored correctly. For this reason you would be wise to avoid seed oil or fish oil capsules, which will almost certainly have been processed and stored at room temperatures for long periods of time!

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... Much more prevalent than you imagined and posing particular risks for athletes

It is one of life's paradoxes that many of the most familiar features turn out on closer inspection to be the most complex. And so it is with nutrition. Take, for example, iron, one of the most familiar and researched yet, arguably, least 'sexy' nutrients. Most athletes know that iron is a mineral required for the formation of the red blood cells used to transport oxygen to hard-working muscles, and that insufficiency of iron causes anaemia, characterised by fatigue, listlessness and a general lack of energy. Because of this, they also know that maintaining iron status and checking red blood cell or haemoglobin (Hb) levels is vital for performance.

However, most athletes are far less aware of the fact that iron is one of the most difficult minerals to absorb, and that they are especially vulnerable to iron depletion through training-induced losses, especially if their event involves endurance training. To make matters worse, the latest ways of measuring iron indicate that that it is perfectly possible to have a healthy blood Hb count while simultaneously suffering from depleted levels of tissue iron. And, if that weren't enough, recent research has demonstrated that this tissue iron depletion impairs the ability of the body to adapt to endurance training.

To better appreciate the complexities of iron nutrition, it helps to understand a little about how iron functions in the body. Most of us are aware of its role in transporting oxygen molecules around the bloodstream to the working muscles; the red colour of oxy-haemoglobin in our red blood cells is visible

evidence of iron in action. When buried deep in the haemoglobin molecule, an iron atom has the perfect atomic structure to bind strongly enough with an oxygen molecule to be transported around the bloodstream (in the form of oxy-haemoglobin) but, crucially, loosely enough to give up the bound oxygen to a muscle needing it.

Classic signs of anaemia

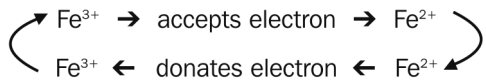
If your iron status becomes severely depleted (through inadequate intake, poor absorption or iron losses), your blood haemoglobin levels will drop, leading to a reduction in your oxygen-carrying capacity. The result is fatigue and breathlessness, even after gentle exertion – the classic signs of anaemia. Most doctors test for blood haemoglobin levels when they test for iron anaemia, although there are other tests, as we'll see later.

However, iron is also crucial for a number of energy-releasing processes because it activates enzymes called catalases, among others. In this role, iron functions as an 'electron shuttle', passing electrons to and accepting electrons from other molecules, thereby helping to make and break chemical bonds in biochemical reactions that would otherwise not occur.

Although as a plain metal iron is very stable and inert, excellent for making cars etc, it is no good for humans in that form

Although as a plain metal iron is very stable and inert, excellent for making cars *etc*, it is no good for humans in that form. Biological systems need iron in its 'ionic' form. Strip away two negatively charged electrons from an iron atom and you generate an iron ion, carrying two positive charges (abbreviated as Fe2+); remove a third electron and you get an iron ion carrying three positive charges (Fe3+). The energy levels of the Fe2+ and Fe3+ ions are quite close, which means that these two ions can easily inter-convert by donating and accepting (ie shuttling) electrons. If an Fe3+ ion accepts an electron from a molecule in a biochemical reaction, it gains a negative charge and becomes Fe2+. If this Fe2+ ion then passes that electron on to a different molecule, it returns to its original Fe3+ state *(see figure 1, opposite top):*

Figure 1: Schematic diagram of iron's 'electron shuttling' role in the body



So far, so good; but the positive charges carried by these iron ions means that they are easily attracted to negatively charged molecules, or parts of molecules, to which they can often 'lock on' and bind. This is particularly the case with the very strongly positively charged Fe3+ ions, which are attracted to and bind especially strongly with molecules containing negatively charged oxygen atoms. A good example of this strong binding is with carbohydrates, which are built from molecules with lots of oxygen-containing fragments. While many carbohydrate foods contain iron, the iron ions are sometimes bound so strongly that the process of digestion is not able to pluck them away. The iron stays joined to these carbohydrates as they pass through the digestive tract, and passes out largely unabsorbed.

If the iron is in the more positively charged Fe3+ state, this binding is even stronger than with the Fe2+ state because there is more attraction between the negative oxygens and the more positively charged Fe3+. This accounts for the poor iron bioavailability of many iron-rich foods: the iron is there but can't be easily prised away for absorption. Even in foods whose iron is readily available, uptake can be considerably reduced by the simultaneous consumption of other food or drinks containing 'iron blockers'. The classic example is tea, containing tannic acid, which readily forms complexes with iron, rendering it far less available to the body. Whatever the health benefits of tea, drinking it to wash down your meal is bad for your iron status!

Barrier to iron absorption

Another barrier to iron absorption arises from the fact that the cell walls of the digestive tract are electrically neutral while iron ions are strongly positively charged, making it hard to transport

them across the gut wall into your body. However, iron that is chemically bonded to protein molecules (eg haem-iron found in meat) carries no overall charge and is much more easily absorbed.

For all these reasons, iron nutrition presents a challenge. It is not just a case of consuming enough iron but of consuming it in a way that makes it fully available to your body.

Then there is the problem of iron loss, which is potentially greater than for many other trace minerals. In menstruating women, for example, monthly losses amount to an average 28mg – easily doubled if periods are heavy or if intrauterine contraceptive devices are used. More importantly for athletes, there is a growing body of evidence that heavy training, particularly of the endurance variety, is a major cause of iron loss.

Iron status in cyclists

A recent study examined the effects of a six-week high-intensity interval training programme, followed by two weeks' recovery, on iron status in trained cyclists[1]. Dietary intake was monitored to ensure that iron intake remained consistent throughout the study, but by the end of week three, haemoglobin, haematocrit and red blood cell count (three different markers of iron status) were all depressed. Meanwhile, serum ferritin (a blood protein involved with iron storage) decreased significantly by week five and remained depressed even in the recovery phase. Total iron binding capacity (TIBC – a measure of a blood protein that transports iron from the gut to the cells that use it) was significantly increased after three weeks, suggesting low iron stores. And the researchers suggested that this reduction could be sufficient over time to have an adverse effect on aerobic cycling performance.

Iron loss as a result of endurance exercise has been confirmed in other studies. For example, a large and comprehensive study examined the effects of different types of exercise on the iron status of 747 athletes divided into three groups (power, mixed and endurance sports) compared with untrained controls[2]. The researchers found that the endurance athletes had reduced levels of haemoglobin and haematocrit

which was mainly attributable to exercise-induced plasma volume expansion: in other words, the same amount of iron carrying compounds were present, but diluted in a larger volume of plasma.

However, they also found that physical activity of increasing volume and duration led to decreased ferritin (an iron storage protein) levels, which were particularly pronounced in runners. This was probably a result of haemolysis – the breakdown and destruction of red blood cells caused by the physical pounding action of running, leading to the release and loss of iron.

This effect of endurance training on iron status has been demonstrated even in very young athletes. An eight-month study examined elite swimmers in the 10-12 age bracket and compared them with inactive controls[3]. Although swimming is regarded as a 'non-traumatic' activity, during the competition phase the elite swimmers suffered significant decreases in serum ferritin and iron stores by comparison with the controls.

Although swimming is regarded as a "non-traumatic" activity, during the competition phase the elite swimmers suffered significant decreases in iron stores

At the same time, the swimmers showed significantly higher levels of a new and highly sensitive indicator of tissue iron status known as 'serum transferrin receptor concentration' (STFR). When cells require more iron, they signal this need by increasing the number of transferrin receptors on their surface; a small proportion of these receptors actually come off the cell surface and are carried into the bloodstream, where they can be measured. A high serum transferrin receptor concentration is, therefore, related to iron deficiency at a truly fundamental level – within the cells or tissues.

The haemoglobin test

Given that iron availability in foods is frequently poor, that iron is difficult to absorb and that training (especially endurance training) can deplete iron stores, it is hardly surprising that iron status in athletes has come under scrutiny. In the past, the age-old haemoglobin test was thought to be sufficient to determine an athlete's iron status, the 'normal' range being 12-16 g/dl (grams per decilitre), with anything under 12g/dl signifying iron anaemia.

However, more recent research has indicated that you can be quite iron deficient without being diagnosed as anaemic. This is because reduced blood haemoglobin is one of the very final stages in iron deficiency, and a lot of iron-dependent systems can suffer before this final stage is detectable. For example, a Canadian study found that although 39% of Ontario women had depleted iron when assessed by the more sensitive serum ferritin test, less than one tenth of these were identified as anaemic by the conventional haemoglobin test[4]!

Moreover, research increasingly shows that a low iron status without a corresponding low blood haemoglobin level still impairs physical performance. Another study found that women athletes who were not conventionally anaemic but had a mild iron depletion as demonstrated by the serum ferritin test had significantly lower VO2max values than those with no iron depletion[5]. The researchers concluded that this reduction in VO2max was due to lower stored iron rather than reduced blood haemoglobin. They also demonstrated that when these women were given iron supplements their serum ferritin values and performances improved without any apparent changes in blood haemoglobin.

Young elite athletes

Another study examined 40 young elite athletes with normal haemoglobin levels but below-average serum ferritin[6]. The athletes were spilt into two groups and randomly assigned to a 12-week treatment with either iron supplements or placebo. Before and after the treatment, aerobic and anaerobic capacity was measured in both groups by means of treadmill tests. At the end of the study period, the iron-supplemented athletes recorded significant increases in VO2max and oxygen consumption by comparison with those on placebo, despite the fact that there were no significant changes in haematological measures.

Such findings are not restricted to endurance activities. A recent six-week study examined the effects of tissue iron depletion on dynamic knee extensions in young women[7]. The

participants, who all had low serum ferritin but normal haemoglobin levels, were treated with either iron or placebo. In the iron-supplemented group, the number of maximal voluntary contractions performed in a subsequent test was significantly higher than in the placebo group. These improvements did not seem to be related to measured changes in iron-status indices or tissue iron stores. Interestingly, though, serum transferrin receptor concentrations increased significantly in the placebo group, suggesting that they were suffering further iron depletion!

It has long been recognised that iron deficiency serious enough to lead to reduced blood haemoglobin also impairs aerobic performance and reduces VO2max; the function of haemoglobin is, after all, to transport oxygen to the working muscles. But how do more marginal iron deficiencies that are not accompanied by anaemia affect performance? Although this type of iron deficiency is known to be commonplace in Western societies[8], there has until recently been a poor understanding of how it impacts on physical performance.

Animal studies have indicated that endurance capacity and the effects of endurance training are diminished when a mild iron deficiency without anaemia exists, and that this probably occurs as a result of diminished concentrations of iron-dependent muscle enzymes and respiratory proteins involved in the biochemical pathways of aerobic metabolism[9,10].

However, although many previous human studies have found suggestive relationships between mild iron deficiency without anaemia and reduced aerobic performance, many of these findings have failed to reach statistical significance – *ie* the results were not sufficiently clear cut to draw reliable conclusions and were probably clouded by the inclusion of subjects with both normal and deficient tissue-iron status.

The problem has been that until recently there has been no definitive test for a real 'tissue iron deficiency'. While measures like serum ferritin, total iron binding capacity (TIBC) and transferrin saturation do give a much clearer picture of an athlete's iron status than a simple blood haemoglobin test, they

still don't tell the whole story – only whether an athlete is within certain 'normal' ranges.

Definitive test for deficiency

They say that every cloud has a silver lining, and it seems that a really definitive test has emerged from the battle to detect erythropoietin (EPO) abuse in athletes. The use of EPO to artificially enhance the red blood cell count (and therefore the blood's oxygen-carrying capacity) in endurance athletes is believed to have become widespread during the mid-to-late 80s; and in the search to come up with a reliable test for possible EPO abuse, a new marker of iron status was identified – serum transferrin receptor concentration (STFR). As we've already seen, STFR is an excellent indicator of tissue iron status because it actually shows how 'hungry' the cells are for iron.

STFR is an excellent indicator of tissue iron status because it actually shows how "hungry" the cells are for iron

The use of STFR as a marker of iron status is at the centre of some very new US research, which suggests that tissue iron deficiency without anaemia can not only impair aerobic performance but also blunt the adaptations that occur following aerobic training. In the first study, 41 untrained iron-depleted but non-anaemic women were randomly assigned to receive either a twice-daily iron supplement or placebo for six weeks[11]. From week three of the study, all the subjects trained on cycle ergometers five days a week.

As expected, iron supplementation significantly improved several markers of iron status, including serum ferritin, transferrin saturation and serum transferrin receptor (STFR) concentrations, yet this occurred without affecting blood haemoglobin concentrations or haematocrit. And, while the average VO2max and maximal respiratory exchange ratio (a measure of how efficiently oxygen is used in aerobic metabolism) improved in both groups after training, the iron group experienced significantly greater improvements in VO2max.

When the researchers analysed the results for relationships between the iron status markers and the measured improvements, it became apparent that it was the STFR concentrations that held the key. In the women whose STFR

levels had been greater than 8mg per litre, taking extra iron produced a significant increase in VO2max above and beyond that produced by training alone; (remember, higher STFR levels indicate that the cells are signalling they need to take up more iron). Conversely, in women with STFR levels below 8mg per litre there were no significant benefits associated with iron supplementation.

The same researchers followed up with another study designed to investigate the role of tissue iron status in the impairment of endurance adaptation, using STFR as the main marker of tissue iron deficiency[12]. Using a very similar testing protocol, 51 iron-depleted but non-anaemic women were selected and randomly assigned to supplementation with either iron or placebo, undergoing five days a week of training on the cycle ergometer (between 75 and 85% of max heart rate) from week three of the six-week supplementation period. At the end of the study, all of the women completed three consecutive 5k time trials with only a short rest between trials. STFR measurements were taken at the beginning, middle and end of the study.

Differences between women

The researchers were particularly interested to see what differences emerged between women with raised levels of STFR and those without, and also how the former were affected by iron supplementation. The results showed that it was the raised STFR group who benefited from iron supplementation, working at a significantly lower percentage of their maximum work capacity during the first and second 5k bouts (indicating improved aerobic efficiency) and showing the largest overall improvement as a result of the training regime, especially by comparison with raised STFR subjects on placebo.

This placebo group reduced their time trial times by an average of only 36 seconds, compared with 3mins 24secs for the raised STFR/iron supplemented group. Moreover, the raised STFR/placebo group had to work at a higher percentage of their VO2max than the iron group for their relatively negligible

improvement! Given that all the women in this study were assessed as iron depleted but non-anaemic, the researchers came to two main conclusions:

1. Iron depletion as measured by serum ferritin was not a reliable indicator of how the women adapted to training. All the women in the placebo group had depleted serum ferritin, but only those with raised STFR suffered an impaired training response. Moreover, in the iron group extra iron only helped those with raised STFR levels. While iron raised serum ferritin levels, it did not produce any significant performance increase in women whose STFR was already below the 8mg per litre baseline. It appears, therefore, that STFR is a far more reliable measure of a truly 'functional' tissue iron deficiency;
2. iron tissue deficiency not only reduces VO2max but also impairs the body's ability to adapt to an aerobic training load (probably due to a decrease in the iron-containing proteins involved in aerobic energy production), with serious implications for athletes!

Maintaining optimum iron status

In the light of the latest research, maintaining an optimum iron status could be far more important for athletes than has previously been realised, especially given that even a mild shortfall appears not only to reduce maximum oxygen uptake capacity and aerobic efficiency but also to impair the body's response to aerobic training. The fact that iron is more difficult to absorb than most other nutrients and that vigorous aerobic training appears to readily deplete tissue iron only serves to underline the extent of the potential problem, especially for young female athletes.

Testing for iron status is also far from straightforward. A low blood haemoglobin (Hb) measurement only appears in the very advanced stages of iron deficiency. It is perfectly possible to have a normal blood Hb level while suffering severe effects from a tissue deficiency. Some athletes and coaches seeking a more reliable method of monitoring iron status have been using

a combination of tests on iron storage/transport compounds in the body as shown in this table.

Table 1: Current tests for iron status

METHOD	VALUES		
	Normal	**Depleted**	**Anaemic**
Haemoglobin	12-16 g/dl		<12 g/dl
Serum ferritin	40-160 mcg/l	20 mcg/l	<12 mcg/l
Total iron binding capacity (TIBC)	300-360 mcg/dl	360 mcg/dl	410 mcg/dl
Transferrin saturation	30-50%	<30%	<10%
Haemocrit	37-47%		<37%
Serum transferrin receptor (STFR)*	<8mgs/l	>8mgs/l	

**A new test, which will require further research to determine the ideal values for athletes. Provisional ranges used in scientific studies are shown*

However, the latest research suggests that, although better then Hb alone, even these tests are insufficient to assess the real need for iron at the cellular level. For example, a reduced serum ferritin concentration generally indicates depletion of the iron stores; but, as the studies mentioned above showed, a reduced serum ferritin does not necessarily mean that performance will suffer because tissue iron stores may not actually be depleted.

Serum ferritin is also what's known as an 'acute phase protein', which means that concentrations are raised during inflammatory conditions. Thus, serum ferritin may be normal (or even raised) in an athlete with such a condition even if he or she is genuinely iron deficient. To determine the real need for iron, a serum transferrin receptor test is the best on offer, although it is relatively new and may not be readily available from your GP.

At this point athletes may be wondering why, given the complexities of iron nutrition, they can't just swallow iron supplements willy-nilly? There are three main reasons:

1. Excess iron is not easily excreted. Self dosage on high-strength iron supplements for long periods of time can induce toxicity;
2. Iron competes for uptake with several minerals in the body, especially copper and zinc; large doses of iron can therefore reduce the uptake of other important minerals, creating imbalances;
3. At high doses, iron is known to function as a 'pro-oxidant', helping to promote the generation of cell-damaging free radicals.

A sensible way forward for athletes is to consume a diet that is naturally rich in iron and to assess their risk for iron deficiency, as shown opposite. Those whose diets are not iron- rich should consider having their iron status tested, using the STFR test if possible. Those who assess their iron deficiency risk as being significant should seek a test for iron status, regardless of diet quality. Routine use of iron supplementation is not recommended until iron status has been properly assessed.

Ways to boost your dietary iron intake

- If you're not vegetarian, try to include some lean cuts of red meat in your diet once or twice each week;
- If you are vegetarian, aim to consume more beans (especially lima beans), lentils, dark green leafy vegetables, eggs and nuts;
- Increase your intake of vitamin C-rich foods (including citrus fruits, berries, new potatoes, broccoli, sprouts, tomatoes, peppers and kiwis). Vitamin C helps convert $Fe3+$ in the body to $Fe2+$, making it up to four times more absorbable!
- Don't drink tea and coffee with meals as the tannins present strongly bind to any iron in food, making it less available to the body;
- Go easy on your consumption of pure bran as it is very high in phytates, which also bind iron. If you want more fibre in your diet, go for whole grain breads and cereals;
- Use stainless steel cookware, which can add useful amounts of iron to cooked foods.

Are you iron-deficient?

All the factors listed below may increase the risk of iron deficiency, particularly those marked with an asterisk:

- My sport involves significant volumes of running or other forms of endurance exercise*;
- I am female;
- I have regular periods*;
- I have had children;
- There is a history of anaemia in my family;
- I am vegetarian;
- I am vegan*;
- I drink tea and coffee with my meals*;
- I use bran products (eg All-Bran);
- I only eat white meat and fish (not red meat);
- I give blood regularly*;
- I cook using aluminium or enamel cookware (not stainless steel or iron);
- I frequently take antibiotics, aspirin or antacids (indigestion remedies).

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…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.

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.

Without getting into the chemical fine detail, a free radical is simply a molecule that contains an unpaired electron. Why is that important? Well, the laws of physics dictate that electrons are only really 'happy' and stable when paired up with a partner, which explains why stable, or non-reactive, chemical molecules nearly always have chemical bonds containing a pair of shared electrons.

An atom or molecule containing a single, or unpaired, electron is distinctly 'unhappy'; it has a lot of energy, is very unstable and is highly reactive, eager to snatch an electron from somewhere else in order to form a stable electron pair. This is what free radicals are: molecules or molecule fragments containing unpaired electrons, desperate to snatch electrons from other chemical bonds in order to form a stable electron pair.

But by doing this, and stealing a single electron from a molecule already containing an electron pair, a second radical is created, which can itself go on to snatch an electron from elsewhere. When free radicals are generated in the body, a

chain reaction is set up in which thousands of molecules are robbed of an electron and then obliged to pinch one from somewhere else!

A chain reaction causing chaos

Remember what it was like at school when the teacher gave out 29 textbooks to 30 classmates? If you were the unlucky one without, you nicked a book from someone else; when he found out, he nicked one from someone else, and so on. Although the class was only ever one book short, the result was often a chain reaction of thefts, causing utter chaos in the classroom!

Free radical chain reactions are very fast. One free radical can easily produce a chain of a hundred billion reactions in the time it takes to blink. Each individual free radical in that chain has only a very fleeting existence, perhaps lasting for just one hundred millionth 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.

But 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 structures like cell walls or DNA, irreparable damage to the cell and/or its genetic material may be the end result. And this damage is now thought to be one of the root causes of degenerative diseases, inflammation and the ageing process in general.

The good news, though, is that human body comes equipped with a number of systems capable of deactivating the free radicals produced as a result of aerobic 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 mop up unwanted free radical activity, 'soak up' the energy of these unpaired electrons and break the chain of free radical reactions, thereby minimising damage to 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.

Athletes don't just process a larger volume of oxygen than their sedentary counterparts – they also process it at a higher rate

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:

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?

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 than 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.

Relying on a single marker to measure oxidative stress in humans is fraught with difficulties

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.

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.

Adaptation to oxidative stres

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.

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% VO2max) than a control group[19].

Beneficial effects of vitamin E

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.

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].

Protection from vitamin C

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 (International Units) 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 which persisted for 24 hours[22].

There is some evidence that vitamin C offers antioxidant protection, particularly when given in combination with vitamin E

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 low-density lipoprotein diene conjugation (a measure of oxidative stress) in the bloodstream[24].

A fascinating 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]. The

researchers also collected detailed dietary information from each of the 184 subjects in the study. They 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).

The case for supplements

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 new 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 daily supplementation regimes, as follows:

- placebo;
- 1,000mg of vitamin C;
- 400IU of vitamin;
- 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!

Risk of cellular damage

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', whereby 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.

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 counter-productive 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].

Recent studies seem to indicate that high doses of antioxidant nutrients may actually harm performance

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].

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 even impair performance.

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!

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...Just how ergogenic is this trace element – and how much is too much?

Sports nutrition can be infuriating. Just when you've think you got things sussed, along comes some new research to turn your ideas upside down. Take chromium, for example: this trace element has long been seen as vital to the integrity of carbohydrate metabolism in the body, particularly for the proper functioning of the insulin system. More recently, chromium has also found favour with athletes and coaches for its apparent ability to enhance lean muscle mass, especially when taken as a supplement in the form of chromium picolinate. However, trawl the scientific data about the benefits of chromium for athletes and things become about as clear as mud!

There's still plenty of controversy about the precise efficacy of this mineral; and, to add to the confusion, the UK Food Standards Agency's recently published report on Safe Upper Limits (SULs) in vitamin and mineral supplements proposed a ban on chromium picolinate in all chromium supplements. So what is the real story? Does chromium really work as suggested? And why has chromium picolinate been singled out as potentially harmful?

In its elemental form, pure chromium is a shiny silvery metal that was first chemically isolated from a Siberian mineral in 1797 by a French chemist called Vauquelin. Although it wasn't realised at the time, chromium is actually widely distributed in nature, occurring in soils, water, biological materials and (to a lesser extent) even in air! However, although metallic chromium is produced on a large scale for a number of industrial purposes (for example as an additive to iron to make stainless steel), naturally occurring

chromium exists almost exclusively in its ionic form as the Cr3+ ion, often known as 'trivalent chromium'.

Chromium and insulin function

It was in the 1950s that nutritionists began to suspect that trivalent chromium was required by the body in order for insulin to work efficiently. However, it wasn't until the mid-70s that chromium was firmly established as essential for human health. A study published in the American Journal of Clinical Nutrition described a female patient who had been on total parenteral nutrition (given by infusion) for over five years[1]. As well as suffering unexplained weight loss, she also developed glucose intolerance (an inability to utilise glucose for energy), neuropathy (problems with the peripheral nerves), high levels of blood fats and abnormalities of nitrogen metabolism. The administration of insulin to improve her condition was ineffective, but when 250 micrograms per day of chromium were added to her parenteral nutrition infusate, she made a dramatic recovery. Further research confirmed these findings, and shortly afterwards the US Food and Nutrition Board designated chromium as 'essential for human health'.

Chromium is widely distributed in wholegrain breads and cereals, which typically contain around 130-140mcg of chromium per kilo. However, the refined (white) versions of these food groups contain much lower concentrations. Meats, beans and pulses, some cheeses and spices are also quite good sources of this mineral. Fruits and vegetables are unreliable sources of chromium, while refined foods, especially those containing sugar, are very poor sources. Cooking or preparing foods using stainless steel utensils may add significant amounts of chromium to the diet. The table opposite shows chromium contents of a number of different foods, but chromium content in different batches of the same food can vary significantly, making accurate calculations of daily chromium intake difficult to achieve.

Maintaining an optimum chromium intake can be quite difficult because there is no one exceptionally rich food source of chromium. And, to complicate matters further,

Chromium content of various foods		
Food	**Portion size**	**Chromium content* (micrograms)**
Beef	3.5oz	57
Calf liver	3.5oz	55
Wholemeal bread	3.5oz	42
Whole rye bread	3.5oz	30
Cheddar cheese	3.5oz	30
Apple peel	3.5oz	27
Oysters	3.5oz	26
Potatoes (with skins)	3.5oz	21
Green pepper	3.5oz	19
Eggs	3.5oz	16
Chicken	3.5oz	15
Cornflakes	3.5oz	14
Lamb	3.5oz	12
Broccoli	1/2 cup	11.0
Spinach	3.5oz	10
Grape juice	8 fl oz	7.5
Oranges	1 medium	5
Orange juice	8 fl oz	2.2
Apple (peeled)	1 medium	1.4
Green beans	1/2 cup	1.1
Banana	1 medium	1.0
Sugar	3.5oz	0
**Figures supplied by Murray (1996)*		

some foods, particularly those rich in quick-releasing carbohydrates, can act to 'wash out' chromium from body stores (of which more later).

Chromium has been established relatively recently as an essential nutrient, so there is currently no UK Reference

Nutrient Intake (UKRNI). However, the UK Food Standards Agency's panel of scientific experts recently suggested a daily intake figure 'in excess of' 25mcg per day. Meanwhile, the US National Research Council has estimated a safe and adequate daily dietary intake at between 50 and 200mcg for adults, which is worrying since many refined Western diets typically contain significantly less than 50mcg of dietary chromium per day.

Tiny amounts are needed

The human body requires chromium in comparatively tiny amounts, with recommended daily intakes measured in millionths of a gram or micrograms, as opposed to the milligrams (a thousand times larger) required for most other minerals. This has made it very difficult to discover exactly how chromium is absorbed and utilised in the body. However, it seems that dietary trivalent chromium we eat in foods is passively absorbed in the gut and then binds to blood plasma proteins such as transferrin for transport around the body.

Once in circulation, the main role of chromium is to enhance the action of insulin, which regulates blood sugar and is one of the body's most anabolic hormones

Once in circulation, the main role of chromium is to enhance the action of insulin, which regulates blood sugar and is one of the body's most anabolic hormones. Insulin acts by binding to cells and then signalling to them that the level of circulating glucose in the blood (derived mainly from digested carbohydrates) is high, thereby promoting the uptake of blood glucose by these cells. Insulin also promotes the uptake of other sugars and amino acids into cells. The overall effect of these actions is to increase the rate of glycogen and protein synthesis – hence the anabolic label.

However, simply secreting insulin into the blood isn't enough to stimulate proper uptake of sugars and amino acids by hungry cells! Insulin, it seems, needs a little help in order to bind tightly and communicate efficiently with cells. Nutritionists have long been aware of an insulin-potentiating agent known as Glucose Tolerance Factor (GTF), which is able to help insulin to carry out its signalling role more efficiently, and recent research has identified that agent as a small peptide called chromodulin[2].

The role of chromodulin

Chromodulin sits on the surface of a cell – eg a muscle cell – that is waiting to be stimulated by insulin. At this point it consists solely of amino acids, is inactive and is known as apochromodulin. But once insulin is secreted it binds circulating trivalent chromium to form a chromodulin-chromium complex, an active peptide, which stimulates the insulin receptor, enabling insulin to bind tightly and communicate with the cell. Once the insulin is properly bound to the cell, the chromodulin-chromium complex is released back into the bloodstream and is eventually excreted via the urine.

If insufficient trivalent chromium is available in the circulating blood plasma, the formation of the chromodulin-chromium complex become more difficult, which in turns reduces the ability of insulin to bind and communicate properly.

When chromium is scarce in the diet, or body stores are insufficient, the benefits of increasing chromium intake are irrefutable, particularly for people with diabetes or glucose intolerance. A number of studies with such patients have demonstrated that boosting chromium intake increases insulin sensitivity, especially where body stores of the mineral are known to be low. For many people with Type II (adult-onset) diabetes, the benefits of improved chromium status, as one of a number of dietary modifications, are enough for them to dispense with insulin administration completely!

But it's not just diabetics who seem to benefit from improved chromium status. Both human[3] and animal[4] studies have shown that chromium supplementation can produce beneficial changes in blood chemistry, thereby lowering the risk of coronary heart disease (CHD). These include lower blood lipids, lower total cholesterol and increased HDL cholesterol (the protective type). Given that refined Western diets are frequently borderline or insufficient in dietary chromium, many nutritionists believe that improving the chromium status of the general population could make an important contribution to the continuing fight against CHD.

So far so good, but where's the evidence that extra chromium can benefit hard-training athletes, especially as exercise is

known to increase chromium excretion? Can chromium really help to increase lean body mass and reduce body fat, as is often claimed by supplement manufacturers? A search of the recent literature suggests that, unless an athlete has a low or borderline chromium status, extra chromium (even when given as the popular supplement chromium picolinate) may have little or no effect on body composition.

Research shows small effects

Earlier this year, a large-scale analysis of all the randomised, double-blind, placebo-controlled trials with supplemental chromium picolinate for weight loss was published[5]. The results show only a very small statistical effect – with most of the evidence for this derived from a single study.

Meanwhile, recent studies on chromium for strength gain are also less than convincing, with most finding little or no effect with chromium supplementation and with a lot of inconsistency, even within studies. Some researchers believe this is because chromium supplementation only benefits people whose existing chromium status is low. In other words, if you're already getting enough chromium to meet your needs, there's no advantage in taking more!

The situation is further complicated by the fact that assessing precise chromium status is very difficult, with little correlation between chromium supply and blood plasma concentrations. Indeed, recent research suggests that impaired insulin function is associated with increased rather than reduced plasma chromium!

Various forms of trivalent chromium are used in supplements, but by far the most popular, especially among athletes, is chromium picolinate. This compound consists of a chromium atom loosely bonded to three molecules of an organic substance called picolinic acid. Studies have demonstrated that chromium picolinate is not only much better absorbed than other forms of chromium, such as chromium chloride, but also more 'lipophilic', which means it can accumulate more easily in cellular tissue.

However, new research suggests that heavy and prolonged

supplementation with chromium picolinate may have potentially harmful side effects, including the production of damaging 'free radicals'. But, before you begin to panic, it's important to stress that this effect has so far been observed only in test tube studies and at very high concentrations. To date there have been no animal or human studies to support these findings, so the risk remains theoretical.

Harmful side effects have so far been observed only in test tube studies and at very high concentrations

Additional warnings about chromium picolinate have been issued based on individual case reports. In one of these, a 33-year-old woman, taking 1,200-2,400mcg of chromium picolinate daily for 4-5 months in an effort to lose weight, developed renal failure[7]. In another, a 49-year-old woman who took 600mcg daily for six weeks was also diagnosed with chronic renal failure[8].

An extremely popular supplement

These cases should be viewed in the context of more than a decade of chromium picolinate studies, in which no ill effects have been noted, and bearing in mind that this is an extremely popular supplement with the population in general, not just athletes. Nevertheless, the UK's Food Standards Agency has called for more research on chromium picolinate and hinted at a possible future ban on over-the-counter sales meanwhile.

If you're a regular or occasional user of chromium, or are thinking of using it as part of your nutritional strategy, where do you go from here? First, it's important to stress that, while chromium is not a miracle muscle-enhancing or weight-loss substance, it is essential for the proper functioning of the insulin system, making it vital for any athlete to maintain an optimum chromium status. However, once this status has been attained, more is not better.

In an ideal world, we would meet all our chromium requirements from our diet. However, because of the known variability in the chromium content of foods, the increased excretion of chromium due to exercise and the difficulty in accurately determining chromium status, some routine supplementation may be the best way to maintain optimum status. Here, then, are a few guidelines for doing just that:

- Eat a wide variety of unrefined foods and ensure that your bread, cereals and pasta are of the wholemeal or wholegrain variety, which contain significantly higher amounts of dietary chromium;
- Try to minimise your consumption of quick-releasing sugary carbohydrates. Not only are these generally low in chromium, but they trigger a much stronger insulin response than complex carbohydrates, leading to greater urinary excretion of chromium via the chromodulin-chromium complex mentioned earlier;
- Regular supplementation to maintain optimum chromium status can be a useful strategy but, unless you have been clinically diagnosed with a low chromium status, you should not exceed 200mcg per day of supplemental chromium for long periods of time;
- Although the evidence that chromium picolinate is harmful to humans at normal levels is far from solid, it may well be prudent to opt for other forms of trivalent chromium until more research data are gathered. Chromium chloride is cheap and quite well tolerated, while chromium polynicotinate (a complex of chromium and vitamin B3 molecules) is well absorbed and biologically active, albeit at a higher price!

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…Is it really a magic route to fat loss and a healthy old age?

Lies, damn lies and supplements! At least, that's the way it can seem in the world of performance-enhancing sports supplements. All too often, the mere mention of a theoretical and untested benefit of a metabolic intermediate or obscure nutritional component in a scientific paper leads to a torrent of expensive products on the shelves, supported by quasi-scientific marketing aimed at the gullible consumer. When subjected to rigorous scientific scrutiny, many of these products don't generate enough hard evidence to substantiate their claims. In plain English, they simply don't work!

However, it's not always like that – witness the proven benefits of creatine – and now evidence is beginning to accumulate about the potential benefits of a derivative of linoleic acid. Reports suggest that supplementation with this derivative, known as conjugated linoleic acid, or CLA, may help promote fat loss in both animals and humans and, further, that CLA may also act as an antioxidant, protecting the cells of the body against the free radical damage that is thought to be at the heart of ageing and degenerative disease.

Does this emerging evidence stand up to scrutiny and, if so, how can athletes manipulate their diet and supplementation regime to reap the benefits of CLA?

CLA has been known about for some time, but interest in it became much more widespread when Pariza and co-workers identified it as a constituent of beef that appeared to possess anti-carcinogenic properties[1]. CLA actually covers a whole group of closely related biologically active compounds called isomers, all of them derivatives of linoleic

acid, which is one of the essential fatty acids and a common component in the diet.

However, unlike linoleic acid, CLA is found in significant quantities only in animal products such as cheese, milk and meat. This is because it is generated from linoleic acid in significant amounts in a process called 'enzymatic isomerisation', which occurs during the metabolism of linoleic acid by rumen bacteria, found in the gut of ruminant animals, such as cows and sheep.

So far at least eight CLA isomers of linoleic acid have been identified, although only two are thought to possess significant biological activity: c9, t11 CLA, which is the most common natural form, and the t10, c12 isomer.

CLA in animal foods

CLA occurs in a number of animal foods, especially full-fat dairy produce, lamb and beef, although the actual amounts of CLA present can be quite variable. Studies have shown that when cattle are fed diets rich in linoleic acid (such as sunflower and soyabean oils), the CLA content of the milk they produce also increases[2]. In addition, cows grazing pasture produce CLA-enriched milk, especially when the grass is at an early growth stage. As a rule of thumb, however, most full-fat beef and dairy produce will contain between 3 and 7mg of CLA per gram of total fat content, 85-95% of which is present as the c9, t11 isomer.

There were early reports that the CLA content of these foods could be increased by heat processing, pasteurisation and pan-frying. However, later studies suggested that CLA content is increased by water loss rather than cooking *per se* and that the actual ratio of CLA to total fat grams remains constant[3]. The table opposite shows typical CLA contents of various foods (expressed as mg of CLA per gram of total fat) and the percentage present as the c9, t11 isomer. Remember, though, that while some low-fat foods, such as yoghurt, have a favourable CLA/total fat ratio, their low fat content means that the absolute amount of CLA per portion will also be low.

Table 1: CLA content of common foods		
FOOD	**Typical mg of CLA per gram of total fat**	**% of CLA present as c9, t11 isomer**
Lamb	5.6	92
Homogenised cow's milk	5.5	92
Low-fat yoghurt	4.8	84
Butter	4.7	88
Cottage cheese	4.5	83
Fresh ground beef	4.3	85
Sharp cheddar cheese	3.6	93
Chicken	0.9	84
Pork	0.6	82

Typical daily intakes of CLA from food sources are very hard to estimate. Not only do CLA contents vary, even among different samples of the same food, but CLA intake will also depend very much on the lamb, beef and dairy content of an individual's diet. Estimates range from just 102mg per day[4] up to 500-1,500mg per day[5]!

Much of the interest in CLA has centred on its apparent ability to affect body composition favourably by increasing lean muscle mass and reducing body fat. Most of the early studies on CLA showed promising results; specifically, adding supplemental CLA to the diet appeared to improve the ratio of lean body mass to body fat. Although the mechanisms of possible action were (and still are) very poorly understood, it seemed that extra CLA facilitated the accumulation of more lean tissue and less fat during periods of growth.

In a study carried out at the University of Wisconsin in 1997, mice fed a regular diet and supplemented with CLA during a six-week period gained 65-73% less body fat than unsupplemented controls[6]. The dose used was 2.5mg of CLA per calorie of food intake, which translates to about 6g per day

for someone on a 2,500-calorie diet. Although the animals in the treatment group reduced their voluntary food intake by 9-13%, they actually maintained a higher level of energy expenditure than the control mice.

CLA and fat metabolism

In a second study carried out that same year, Pariza looked at the effect of CLA on fat metabolism and found evidence that CLA reduces fat deposition in fat cells while boosting fat breakdown in muscle cells[7].

But despite the excitement surrounding these studies, and their substantiation by other research, there was still a big fly in the ointment: virtually all the studies were carried out on animals, with very little data on humans available. The joke in the scientific community was that CLA was a great weight loss supplement – for mice!

Research on humans

In the intervening years, CLA research on humans has gathered pace, but the results of studies are much more mixed. In one major trial, researchers from Norway studied a group of healthy but overweight men and women, who were given 1.7g, 3.4g, 5.1g, or 6.8g of CLA per day[8]. All groups showed a significant reduction in body fat and increase in lean muscle, but this effect was most significant for the two latter groups. It was also observed that all groups showed a reduction in blood fat and cholesterol.

Subsequently, Swedish researchers studied 25 obese men aged 39-64 for four weeks and found that those taking 4.2g of CLA per day showed an average loss in waist circumference of 1.4cm[9].

However, a large number of more recent studies have failed to demonstrate significant benefits of supplementation with CLA, particularly in respect of body fat reduction. For example, a study of experienced resistance trainers taking 6g per day of supplemental CLA carried out in 2002 failed to find any significant beneficial changes in total body mass, fat-free

mass, fat mass, % body fat, bone mass, strength, serum substrates or general markers of catabolism and immunity during training[10].

Subsequently, in 2003, a three-month double-blind placebo-controlled study failed to find any improvement in body weight maintenance after a period of weight loss[11]. Interestingly, however, CLA seemed to aid the preferential regain of fat-free mass at both experimental doses of 1.8 and 3.6g per day.

Studies in humans have found more evidence for a gain in fat-free mass than for a straightforward loss in body fat

Other studies in humans have also found somewhat more evidence for a gain in fat-free mass than for a straightforward loss in body fat. And this distinction may help, in part, to explain the discrepancy between animal and human studies, since many of the animal subjects used have much faster metabolisms than humans, with tissue growth and development forming a major part of the metabolic processes.

The scientific jury is still out

On the whole, then, while there is some promising evidence, the scientific jury's still out over CLA and body composition. Meanwhile, scientists are currently researching a number of other possible benefits of CLA, including its antioxidant properties (which would make it a valuable weapon against both cancer and heart disease), its ability to improve insulin function (with positive implications for diabetes) and its role as an immune stimulant.

So, even if more research on body composition and CLA is needed, isn't there enough evidence of other benefits to justify using it anyway? Unfortunately, not all the changes noted with CLA supplementation have been beneficial. Some of the animal studies have suggested a reduced sensitivity to insulin. More worryingly, this impairment of insulin sensitivity has also been observed in humans[12].

Why does this matter to athletes? Well, a reduced sensitivity to insulin makes it harder for the body to absorb glucose and amino acids into muscle tissue (exactly what athletes don't want) and is also associated with an increased risk of diabetes and heart disease.

Another problem centres on the t10, c12 isomer – the variety less prevalent in food. Recent animal studies appear to show that this is the isomer responsible for reducing body fat, and some commercially available CLA preparations have started to appear with increased proportions of this isomer. However, in a large human study on obese men carried out in 2002, t10, c12 supplementation increased markers of both oxidative stress (suggesting increased oxidative damage to cell membranes) and inflammation[13]. Inflammation is frequently a response to tissue injury, and some of the inflammatory markers measured in this study are highly correlated with an increased risk of heart disease.

If you want to boost your CLA intake to the levels used in scientific studies, you will have to take supplements

In summary

Faced with such a mixed bag of evidence, it's hard to make firm recommendations, but here goes:

1. There is no point in trying to increase your dietary CLA because, even if all the theoretical benefits of CLA were confirmed, you'd have to consume at least 500g of (mainly saturated) fat each day to get meaningful amounts (3-plus grams); that's over 4,500 fat calories each day – hardly conducive to fat loss! If you want to boost your CLA intake to the levels used in scientific studies, you will have to take supplements;
2. Since the research indicates that CLA may be more effective at increasing lean muscle mass than simply reducing body fat, you would probably do better to use supplemental CLA in a strength or mass-building phase rather than continuously. Since there's very little data on long-term supplementation with CLA, this would probably be prudent anyway;
3. In terms of the actual CLA supplement you use, it's probably wise to stick to those whose isomer content is similar to naturally-occurring CLA – *ie* consisting predominantly of c9, t11 CLA. While the t10, c12 isomer may exert a much more potent fat-reducing effect, there are simply too many unanswered questions over its possible health risks to recommend using large amounts at present;

4. Finally, because CLA is known to share metabolic pathways with linoleic and alpha-linolenic acid (the essential fats, or EFAs), if you supplement with CLA it is worth ensuring that your dietary intake of these oils is up to scratch. Good sources of EFAs include seeds, such as sunflower and pumpkin, nuts, such as walnuts and hazelnuts, wheatgerm, cold-pressed seed oils and most unrefined whole grain cereals.

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...Could they 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'!

Over the last 18 months, 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 under way with humans. Initial results look encouraging, but what are the implications for athletes – and can these nutrients be harnessed to improve performance?

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. 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.

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.

Effects of ALA

Alpha lipoic acid (ALA) is a sulphur-containing antioxidant, 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 both as 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 plain English, ALC reaches parts of the body that L-carnitine just can't reach!

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.

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 opposite)*.

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 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

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.

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. 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 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!

Research on oxidative stress

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 1,000mg of ALC and 400mg 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 (a potentially damaging by-product of energy production).

To measure 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 in the treatment group recorded values indicating that levels of oxidative stress had fallen. By contrast, no such benefits were reported in the placebo group.

If an ALC/ALA combination can reduce exercise-induced oxidative stress, that would be good news for athletes, who are particularly vulnerable to such stress. 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.

Positive findings in animals

Other human studies are also currently under way, but so far there are no published results available, although positive studies in animals continue to proliferate. In 2002, for example, American researchers demonstrated that ALA supplementation in older racehorses reduced the oxidative stress burden even under light training loads[4], while other animal studies 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.

Benefits for athletes

Nevertheless, the following findings suggest that these nutrients have a great deal to offer to athletes:

- **ALC and human growth hormone (GH)**. A study carried out in 2001 suggests that 500mg 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* last year showed that ALA reduced the markers of chronic age-related inflammation typically seen in human cells[8];
- **ALC and mood.** 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].

An enticing prospect

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 of 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.

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.

ALA/ALC is most likely to offer a long-term investment for your health

The bad news is that ALA and ALC are 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 600mg of ALA per day, while studies showing that ALC improves brain function in Alzheimer's patients[10] have used between 1,500 and 3,000mg per day. However, the human study carried out in San Francisco, which used 400mg of ALA and 1,000mg 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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WHAT THE SCIENTISTS SAY

Reports on recent nutrition-related research by Isabel Walker and Raphael Brandon

Why power trainers need extra protein

There are many protein and amino acid supplements on the market, but how do you know if you need them in addition to your normal diet? New research suggests that boosting protein intake could be beneficial to athletes.

In a recent study, competitive sprinters and jumpers completed two sets of two testing sessions, interspersed by a five-week training period, while the researchers measured the effects of the activity on a number of blood variables.

The first test was a short run session, comprising three sets of 4x60m sprints, two minutes between reps and six minutes between sets; the second test – performed the next day – was a long run session comprising short 20-second treadmill sprints to exhaustion. The speed of the treadmill was increased slightly between each rep, separated by 100 seconds of rest.

The athletes' blood levels of amino acids, testosterone, growth hormone and cortisol were measured on the morning of the tests (fasting level), then 10 minutes before and 10 minutes after the running workouts. They then completed five weeks of typical off-season training, comprising sprints, speed endurance, plyometrics and free weights sessions, before repeating both testing sessions.

Throughout the study period, the athletes consumed their normal self-selected diets, which were made up, on average, of 53% carbohydrates, 32% fat and 15% protein.

Analysis of the athletes' blood tests showed that fasting levels of amino acids were about 20% lower after the five-week training period than they had been before. By contrast, resting testosterone had increased by about 25%. And while the short run session led to reduced levels of certain amino acids – asparagine, valine and taurine – the long run session produced no significant changes.

The reduction in fasting levels of amino acids after the training period suggests that the athletes' protein intake was inadequate to cope with the demands of power training, when the body increases its rate of protein synthesis by plundering its pool of free amino acids. The rise in testosterone and relatively low levels of cortisol after the training period showed that the athletes were in a healthy anabolic state – *ie* their bodies were ready to recover from training and build muscle.

However, the implication is that they might have reaped greater benefits from their training programme if they had increased the level of 'building materials' (amino acids) in their diets and that a dietary intake of 15% protein (or 1.25g per kg of body weight) is too low for power athletes performing quality training.

The researchers suggest that 1.7g/kg might be more appropriate for maximising training benefits. Ideally, power athletes should focus on low-fat protein foods, such as cottage cheese, turkey, egg whites, soya products, beans and pulses. This research also supports the use of protein supplements during exercise sessions; a practical method would be to mix them with carbohydrate and electrolytes in a drink.

Journal of Strength and Conditioning Research, 16(2), 390-398

Re-think on zinc

The theory that athletes need to take zinc supplements to protect their immune systems during periods of heavy training has been cast into doubt by a new study from Australia and New Zealand.

Zinc is vital to healthy immune function, and previous studies have shown that people with severe zinc deficiency are prone to a range of disorders linked with compromised immunity, including an increased incidence of infection. It has been suggested that athletes may have inadequate dietary intakes of zinc (found more in protein than in carbohydrate foods) and that regular intense exercise may reduce blood zinc concentrations further, so posing a threat to health.

The researchers put this hypothesis to the test in a study of 10 male runners, who increased their normal training volume of long

slow distance running by 16% over four weeks, then reduced it to recovery levels for a further two weeks. Blood samples taken from the athletes before and after the four weeks' intensified training, and again after the two weeks' recovery, were analysed for plasma zinc concentration and immune system markers and compared with samples taken from a group of seven sedentary male 'controls'.

Key findings were as follows:

- The athletes generally had lower plasma zinc concentrations than the non-athletes both before and during the four weeks of intensified training, but these remained within the clinically normal range and were unchanged in response to the moderate increase in training volume;
- Although cell counts for some lymphocytes (white blood cells involved in immunity) were lower in the athletes than the controls, they were unaffected by training and lymphocyte responsiveness was similar or higher in the athletes than the controls. Thus plasma zinc concentrations generally correlated poorly with the immune parameters under investigation;
- Five out of 10 athletes experienced varying degrees of illness during the study, compared with two out of seven controls.

The researchers conclude that their results do not support the use of zinc supplements among well-trained athletes during a period of moderately increased training volume.
Int J Sports Med 2003;24:212-216

GACIK – is it even better than creatine?

A promising rival to creatine has been put forward in the shape of GACIK – a dietary supplement containing a mixture of amino acids and ketoacids.

A pair of US researchers set out to study the effects of GAKIC supplementation on repeated bouts of anaerobic cycling performance, following an earlier study that showed the supplement

significantly increased work output and delayed muscle fatigue during exhaustive isokinetic knee extensions.

Ten male college students, all of whom engaged in planned high-intensity exercise on at least three days per week, each performed two sessions of five supramaximal 10-second cycle ergometer sprints, one after supplementation with GACIK and the other after supplementation with placebo. Both of these supplements were administered in three doses 45, 30 and 10 minutes before exercise. The two exercise sessions were separated by an average of seven days, and the five sprints were separated by rest intervals of one minute.

Analysis of the results revealed a significant difference between placebo and GACIK in the pattern of change in mean power output over the five sprints, with the decrease in mean power output between sprints 1 and 2 being significantly less with GACIK than with placebo.

'The results of the present study indicate that GACIK ingestion significantly attenuates the drop in mean power output associated with repeated sprints of anaerobic cycling,' state the researchers.

However, they are unable to explain precisely how GACIK exerts this ergogenic effect, especially as a similarity in post-exercise lactate concentrations after both GACIK and placebo appeared to rule out an effect on glucose kinetics.

They conclude, in the light of their own research and the previous findings mentioned earlier, that GACIK can serve as an ergogenic aid to improve muscle performance in repeated high-intensity exercise.

'These findings reinforce the notion that GAKIC supplementation may be a useful aid for strength-power athletes, similar to creatine supplementation,' they say. And they point out further: 'Unlike creatine, which requires approximately 5 [days] of loading to produce improvements in high-intensity work, GACIK appears to impart an ergogenic effect within minutes of consumption. This presents an obvious advantage for GAKIC supplementation.'

However, further studies are needed to corroborate GACIK's effectiveness and to determine whether its effects might be enhanced by being given in combination with creatine.

Med Sci Sports Exerc, vol 36, no 4, pp583-587

Sodium bicarbonate and sprint performance

The positive impact of sodium bicarbonate on sprint performance has been confirmed by a study comparing its effects with those of two other potential 'buffering' agents.

When continuous exercise is performed at high intensity for up to five minutes, energy production by anaerobic glycolysis is accompanied by a rise in intramuscular acidity that contributes to fatigue. Extracellular bicarbonate helps to buffer this acidity, thereby potentially delaying fatigue. And, indeed, ingestion of sodium bicarbonate has been shown to improve high-intensity performance substantially in previous studies.

The researchers (from the Netherlands, New Zealand and the UK) in the current study were keen to compare the effectiveness of sodium bicarbonate with sodium citrate and sodium lactate, which are known to produce indirect buffering effects.

In a double blind randomised crossover trial, 15 competitive male endurance runners, accustomed to brief intense efforts as part of their interval training, performed a run to exhaustion 90 minutes after ingestion of each of the agents and a placebo (sodium chloride) on separate days.

The mean run times to exhaustion were:

- bicarbonate 82.3 seconds;
- lactate 80.2s;
- citrate 78.2s;
- chloride 77.4s.

There were no substantial differences in gut discomfort between the buffer treatments.

The researchers conclude they have found 'more evidence that ingestion of sodium bicarbonate is an effective strategy to enhance sprint performance. Sodium lactate is also likely to be effective, although possibly not as effective as sodium bicarbonate. Sodium citrate is probably not as effective as sodium bicarbonate.

'We therefore recommend ingestion of sodium bicarbonate to enhance sprint performance.'

Med Sci Sports Exerc, vol 36, no 7, 1239-1243

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