Training for

MASTER ATHLETES

A SPECIAL REPORT FROM

PEAK PERFORMANCE

The research newsletter on stamina, strength and fitness

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From the publisher

After reading this special report, I feel quite invigorated. Or is it depressed? There I was telling myself I was far too old, say, to ever win Wimbledon or to become world butterfly champion over 200 metres and yet now I read that almost anything is possible at any age. All you need is the right training, the right diet and loads upon loads of willpower. Pardon me while I lie down. (I wonder how old the UK tiddliewinks champion is?)

This special report on the problems and potential of older athletes has been prepared by the Peak Performance team of experts, including physiologists, nutritionists and masters athletes themselves. It covers a vast range of subjects with one principal aim in mind: how you can overcome the ravages of getting older and still produce winning performances, while staying healthy and – most importantly – continuing to enjoy yourself. If you're in any doubt on that last point, read Bruce Tulloh's article on page 21. It positively vibrates with good humour. (Maybe, on second thoughts, I'll reconsider that point about winning Wimbledon. After all, look at Navratilova.)

I hope you enjoy this report and find it useful.

Jonathan A Pye
Publisher

A top sports scientist asks: 'Is the ageing process inevitable – or simply the result of "detraining"?'

A leading question to ask yourself if you're past your first youth, is: 'how old would you think you were if you didn't know how old you are?' I would predict that the disparity between theory and fact in the answer would give an indication of your exercise level. No one can escape their genetic programme, but most people can do a great deal, barring injury and illness, to minimise some of the physiological aspects of ageing by modest exercise programmes which embrace conditioning in strength and flexibility as well as the more commonly-prescribed aerobic exercise.

It is worth noting that, from the 100m to the Marathon, men and women of 80 take only up to 55% and 110% respectively longer than they did at 40, with a virtual plateau in performance between 20 and 35-40 (see Table 1).

This article will focus on the achievements of older distance runners, including veteran and masters athletes, together with changes in some of the physiological parameters relating especially to distance running in older people.

In 1900 people were considered old in their 40s; just 40 years ago people seemed old in their 60s; but today many do not feel old in their 80's – a phenomenon described as 'youth creep'. Yet there is no common experience in the ageing process, for while an orchestra conductor might see himself as a mere stripling at 50, a 20-year-old rhythmic gymnast can feel hopelessly middle-aged.

The oldest Olympic medallist (silver) was Swedish rifleman Oscar Swahn, at 72 in 1920. He was also picked for the 1924 Paris Games, but had to drop out due to injury, and had previously won gold in 1912. The oldest British competitor was dressage expert Hilda Johnstone, aged 70 at the Munich Olympics. At least six Olympians have spanned 40 years, three yachtsmen, two fencers and one equestrian.

Turning to running, in 1994 41-year-old Eamon Coughlan ran the mile in 3:58:15, while 42-year-old Yekaterina Podkopayeva just beat 38-year-old Mary Decker in the 1997 world indoor 1500m final, and later ran 3:59:10. In the marathon, Carlos Lopez won the Los Angeles event in an Olympic record of 2:09:21 at age 38, having broken the world 10,000m record at 37, running 27:17:48. A 90-year-old man has run 4:25.27 and an 80-year-old woman 5:10:04.

At 80, men and women can run 100m in 14.35 and 18.0 respectively and 10k in under 45 and 59 minutes; and in all distances, from 100m to 10,000m, the general decline with age in trained men and women is linear and very gradual until 80, when times become less consistent [1,2]. Until then, the rate of slowing-up is about 1-2% per year, about the same as the loss of maximum heart rate over the same period.

What are the key physiological changes impacting on performance with age? Body fat composition, which at 20 stands at about 12-16% in men and 23-28% in women, rises steadily to some 19-26% and 28-38% respectively by age 60-70, while lean body mass declines. Height tends to decrease by about 1cm per decade after age 40.

The 'rete pegs', which anchor the outer layer of skin – the epidermis – to the dermis beneath, shorten with age, making older runners increasingly susceptible to blister formation and skin trauma. Melanocytes, the skin cells producing melanin, diminish at a rate of some 2% annually in the 40s, and the cutaneous inflammatory response diminishes.

Hence older athletes (especially novices) are more susceptible to sunburn than the young but do not show its acute effects as promptly.

The ageing heart and lungs

As far as the cardiorespiratory system is concerned, forced vital capacity decreases by about 250ml per decade. The elasticity of the pulmonary support structures diminishes and the size of the alveoli (air sacs) increases, which can boost the work of breathing from 10% of the energy cost of exercise in the 20s to up to 20% in the 60s. The number of pulmonary capillaries declines, as does perfusion quality, and the respiratory centre becomes increasingly sensitive to blood levels of carbon dioxide. Thus ventilation increases disproportionately to oxygen intake in older subjects (much as it does for younger women in pregnancy).

Maximum heart rate declines by some 40 beats per minute between 20 and 60, while an age-related increase in the size of the myocardium (heart muscle) leads to a diminishing stroke volume by decreasing the size of the ventricular chamber size. These changes contribute to a decline in VO_2max of about 5ml.kg-1. min-1 per decade in untrained men and women[3]. For those who remain in training, VO_2max may remain consistently higher than in the untrained, eg by 20ml.kg-1.min-1 at age 40 and 10ml.kg-1.min-1 at 70.

Skeletal muscle capillarisation has been found to be as high in older runners as in younger ones of equivalent performance[4]. Ageing muscle has been shown by some workers to contain higher percentages of Type 1 fibres, which would appear potentially beneficial to elderly marathon runners[5]. With age, muscle protein diminishes, as does the size and number of mitochondria (the site of the cell's energy production), although this is less true if relative habitual activity is held constant. In part, such decreases may be due to disuse atrophy – or 'detraining'.

Muscle power and strength

Between the ages of 65 and 90, muscle power is lost more rapidly than strength – 3.5% per year for the former compared with about 1.8% for the latter[6]. Also, concentric force development is lost more rapidly than the eccentric variety. In

both sexes there is little fall-off in strength until about the mid-40s, after which it drops by approximately 25% by age 65. In women, there may be an accelerated post-menopausal fall-off in power and strength. With aging in general there is progressive muscle atrophy (wasting).

Nevertheless, to some extent, these force parameter losses appear to be reversible [7]; for example, eight weeks' strength training in 56-70-year-old-men has produced marked improvement in local muscle endurance [8]; and strength training, even of nonagenarians, may produce a doubling of force development by quadriceps [9].

It is noteworthy that regular exercise appears to be

Table 1 USA single-age marathon records

Age	Male	Female	Age	Male	Female
4	6:03	–	28	2:10:40	2:21
5	5:25	4:56:30	29	2:10	2:31
6	4:07	4:00:30	30	2:10	2:28
7	4:04	3:52	31	2:09:30	2:30
8	3:37	3:13:30	32	2:11:30	2:30
9	3:07	3:11	33	2:11:30	2:28
10	3:02	3:07	34	2:13:12	2:30
11	2:47	3:04	35	2:12	2:32
12	2:46:30	2:58	36	2:15:30	2:36
13	2:43	2:53	37	2:12:42	2:28
14	2:41:30	2:51	38	2:17:18	2:27
15	2:29	2:46:30	39	2:14:18	2:31
16	2:23	2:34	40	2:17	2:40:30
17	2:24	2:47	41	2:19:18	2:38
18	2:17	2:42	42	2:20	2:42
19	2:15	2:34:42	43	2:23	2:40
20	2:13	2:30:18	44	2:25	2:35
23	2:08	2:30:10	45	2:26	2:45
25	2:09:18	2:26:00	46	2:26	2:53
26	2:11	2:29	47	2:27	2:52
27	2:08:40	2:28	48	2:31	2:51

accompanied by a slowing in the rate of decline of movement/reaction times in the elderly, which could help minimise falls or stumbling during the later stages of endurance runs or races. Studies have shown that 'old active' subjects have faster response times than both their inactive counterparts and 'young non-active' people [10].

Well-used tendon retains more of its elastic properties. And collagen turnover is increased with exercise, which would allow for greater elastic energy storage in tendon and ligament, thus improving running economy.

Regular weight-bearing exercise has been shown to decrease the rate of bone demineralisation leading to osteoporosis,

Age	Male	Female	Age	Male	Female
49	2:33	2:51	70	3:01	4:09
50	2:25:42	2:50	71	3:01	4:25
51	2:29	2:52	72	3:09	4:38
52	2:25	3:01	73	3:13	4:57
53	2:31	3:00	74	3:37	4:48
54	2:32	2:52	75	3:18	4:32
55	2:34	2:52	76	3:35	4:31
56	2:39	3:07	77	3:33	5:13
57	2:38	3:13	78	3:37	5:01
58	2:38	3:21	79	3:49	5:04
59	2:48	3:24	80	4:28	5:10
60	2:47	3:15	81	4:50	5:51
61	2:43	3:28	82	4:38	6:14
62	2:49	3:31	83	5:20	6:33
63	2:48	3:30	84	4:18	6:02
64	2:43	3:39	85	5:22	6:54
65	2:51	3:37	86	5:40	7:57
66	2:43	3:49	87	6:49	7:09
67	2:55	4:02	88	6:52	8:03
68	2:52	4:03	89	6:36	8:09
69	3:01	4:01	90	7:53	10:13

although this effect is reasonably specific to the body segments exercised.

In sedentary people thermal control in the cold diminishes with age on account of a combination of factors, including reduced body mass, lowered basal metabolic rate and diminished shiver response by muscles. But exercise has also been shown to improve thermal control, helping older people to resist hypothermia in the cold and overheating while exercising in hot weather. There is increasing evidence that moderate exercise, perhaps particularly in the elderly, may enhance some immune responses and lead to a lowered incidence of illness – eg respiratory disease.

In very approximate terms, one might estimate that 20-40% (or more) of the physiological deterioration associated with ageing is not inevitable but is due to a detraining effect of decreased exercise, often coupled with an increase in body fat. The athletes' motto 'If you don't use it – you lose it' applies equally to the ageing population. And the extraordinary marathon performances of elderly runners set out in the accompanying table confirm it.

Craig Sharp

Adapted from 'Aging and the marathon', a paper in Marathon Medicine, RSM Press Ltd, RSM Press Ltd, £19.95, 2001, www.rsmpress.co.uk/bktunstall.htm.

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'I am now running, at 59, the sort of times I was running as a 19-year-old National Serviceman'

(Ten years ago, the celebrated coach and European 5000m champion, Bruce Tulloh, wrote this article for Peak Performance. We thought it eminently worth reprinting for this special report)

I can remember the time when a boxer was considered old at 28, over the top at 30. The prejudices against grown men taking sport seriously take a long time to disappear. In America sport stops when you leave college – or it did until people voted with their feet and the running boom started. These attitudes are in the nature of self-fulfiling prophecies. We say that we are too old for sport, so we stop taking exercise, so we cannot perform as well as we used to – that's hardly surprising.

Yet we have and have had for many years hosts of examples which give the lie to these attitudes. A 70-year-old weight-lifter is stronger than the average 30-year-old. The ballet dancer of 70 is more flexible than the average young adult, while the 70-year-old marathon runner will easily outrun the majority of 30-year-olds.

Such people owe their achievements not to the fact that they were outstanding when they were younger but to the fact that they have continued to practise the activity that they enjoy. They are often fortunate, in that society allows them dispensation because of their former achievements, but they are also determined. The will-power that made them great when they were young gives them the ability to continue into old age, when

lesser men have been shamed out of it. If you want examples, take Jean Borotra and Kitty Godfrey, playing tennis into their 90s, Edward Weston, who walked across the USA – and back – in his mid-70s, Ron Taylor, who could run 100 metres in 11.3 seconds at the age of 60, or Cliff Young, who won the first Sydney to Melbourne race at the age of 61.

The questions to ask

When we are young we feel immortal and in a sense we are, because our cells renew themselves constantly. As we get older, the rate of cell division slows down and some tissues begin to perform less efficiently. There is a loss of elasticity, both in the skin and in ligaments. There is a decline in the maximum heart rate and in maximum power output. The questions we need to look at are: how early do these changes set in? Is there anything we can do to reverse the process? What performance should we expect at a certain age?

Athletics has the advantage of being completely measurable, so that we can see just what is happening. First, we see that it is possible to remain at the very highest level until at least 35 in the sprints (see Linford Christie, Carl Lewis and Merlene Ottey) and until the late-30s in the longer events; Carlos Lopes won the world cross-country title and the Olympic marathon at the age of 37. With continued training it is possible to remain at international level to the age of 40 – as witness Eamonn Coghlan's sub-four-minute miles, Al Oerter's record in the discus and the victories of the 42-year-old Podkopayeva at 1500 metres in 1994. In events involving pure endurance, the limits can be extended for longer. The world's best for a marathon at the age of 50 stands at 2:11, a time that would win many international marathons.

Second, when decline does set in, it is very gradual – of the order of 0.5% a year for many years. The reason we cannot be more definite is that, until recently, few athletes have been able to defy social convention and continue their sporting career into their 60s or 70s. Now that master sport is becoming more accepted, data is accumulating.

My own rate of decline

A look at my own training diary gives an idea of the rate of decline. I ran the equivalent of 13:45 for 5k in 1960 and continued at that level until 1967. All that time I was training very hard as opposed to merely keeping fit and putting in the occasional hard session, which has been my routine since 1969. In 1971, aged 35, I ran 14:28. Twenty years later, aged 55, I ran 16:19 – a decline of 109 seconds. In the Hyde Park Fun Run, four kilometres, I slowed from 12:20 to 13:40, a decline of 80 seconds over 15 years. Over 10k I have declined from being a 29:00 man in 1967 to running 32:30 in 1985 (210 seconds over 18 years) and 35:00 in 1994; this is a decline of 360 seconds over 27 years and 150 in the last nine years.

Comparing the three distances, we get a decline rate of

Hyde Park (4k on grass)	**1.3 secs/km/year**
5000m (track)	**1.1 secs/km/year**
10km (road)	**1.3 secs/km/year**

Rounding things up, we can reckon a steady rate of decline, after the age of 35, of about two seconds per mile per year (0.55%). This means that over five years, other things being equal, you will be a half-minute slower over 6 miles/10k and two minutes slower in a half-marathon.

How motivated are you?

However, other things are not equal. The most important variable is how hard you train, which is related to your psychological drive.

When someone takes up running for the first time or comes back to it after a long break, he or she will improve for another year or two, whatever their age, because they are getting fitter. The body is gradually being converted into that of a runner. An improvement of 10 seconds a mile in the first year is not unusual. On the other hand, a person who has been competing since the age of 12 is not going to change very much.

The only exceptions to this are when one moves to a new

event or when one changes the training dramatically. This applies particularly to endurance events – marathon and beyond – because it doesn't matter how much speed you have, if you haven't got the endurance you won't be able to keep running. Thus the 30-year-old middle-distance runner may not be able to break three hours for a marathon, just because of lack of endurance, whereas a 60-year-old who has done the training would manage it easily.

There is no doubt that the average person (that is to say, an unfit person) can reverse most of the effects of ageing by using the right exercise programmes (as other articles in this special report suggest). The person who has kept fit will always be younger, in the physiological sense, than his real age. I am now running, at 59, the sort of times I was running as a 19-year-old National Serviceman. On the other hand, my flexibility and my muscle strength, apart from the leg muscles, have declined considerably because I have not worked specifically on them.

The three lessons

What are the implications of this for the average sportsman? First, there is no reason to stop purely because of age, because the decline is very gradual and a bit of hard training and some good conditions can sometimes enable you to improve on last year's times.

Second, one cannot train with the intensity of a young athlete because the rate of regeneration is not as fast. The reason for this may well be hormonal, therefore a change in the way of life or a move to another event – taking up triathlon, for example – can provide the stimulus for further improvement. You have to find the intensity and the frequency of hard training that you can tolerate.

Third, it will probably become necessary, if you seriously want to stay as fit and healthy as possible, to work on your weak points. Muscular strength declines unless your muscles are exercised. Flexibility and mobility, too, cannot be taken for granted. It is necessary for the specialist sportsman to become less of a specialist in order to maintain the all-round fitness that

he took for granted in his teens or his 20s. The squash player, for example, would need to run more regularly to maintain his oxygen intake and his endurance. The runner should spend more time on upper body training and flexibility exercises, and the swimmer should spend more time walking or running so that his supporting muscles do not weaken.

Bruce Tulloh

These are the best foods and supplements to protect your joints from age-related degeneration

There's good news and bad news for older athletes. The good news is that, if training intensity can be maintained, age-related performance decrements are actually quite minimal; The bad news is that recovery from hard training sessions takes longer, while the cumulative effects of normal 'wear and tear' and previous injuries are increasingly evident. As time goes by, joints tend to become less flexible, full-range movement more difficult and pain and stiffness ever more apparent. It is these mechanical limitations, more than anything else, which can scupper the best-laid plans of even the most determined older athletes!

However, there are a number of nutritional strategies that can help to offset the inexorable decline in mobility and even accelerate recovery from injury.

Before we get down to nutritional nuts and bolts, it's worth outlining some of the causes of joint degeneration and immobility. Basically, a joint exists whenever two bones meet. Many joints are freely moveable, allowing large ranges of movement; for example, the knees, ankles, hips, elbows and fingers. Others, such as those between the spinal vertebrae, allow for only partial movement. There are even some fixed joints (eg, in the skull, although maintaining range of motion is obviously not an issue here!)

The freely moveable joints are generally 'synovial'-type joints, where the two ends of the bones that meet are covered

with 'articular cartilage' – a sort of 'low friction' coating. Although the two bones are in fairly close proximity, they don't actually touch, being separated by a joint cavity, which is like a fluid-filled sac. The walls of this cavity are lined with synovial membrane, which secretes synovial fluid into the cavity to lubricate the movement between the two cartilage surfaces. Meanwhile, the whole joint structure is stabilised by ligaments – tough fibrous tissues connecting and anchoring the two bones.

The joints between the vertebrae of the spine are slightly different in that the movement between any two adjacent vertebrae is quite restricted. However, the cumulative effect of several small intervertebral movements allows for a large degree of global movement of the spinal structure as a whole. Another difference is that the main joints between the vertebral bodies are 'cartilaginous', containing an intervertebral disc. These discs are like soft pads, which allow relative movement between adjacent vertebrae and can accommodate the different curvature requirements of the spine at different points along its length.

There are a large number of possible causes of joint pain and stiffness, and the diagnosis of a particular problem can be a very complex process – just ask any physiotherapist! In general terms, however, there are a number of well-recognised causal factors.

1. **Acute injuries** come on suddenly and are usually associated with some kind of trauma. Common examples include:

- ligaments torn or damaged by unusual or excessive movement of the joint;
- impact injuries, where one or more of the joint structures is damaged by an external blow;
- protruding/prolapsed intervertebral disc, where unusual intervertebral forces lead to the deformation of the disc, allowing it to come into close proximity with nerves.

2. **Chronic injuries** tend to come on quite gradually, thus making them trickier to diagnose and harder to overcome. Common examples include:

● overuse injuries, where the long-term training volume exceeds the capacity of the joints involved to undergo adequate repair and recovery;
● muscle imbalance injuries, where the joint fails to operate through its correct range of movement because of unequal or unbalanced muscular forces acting on the joint, or (particularly in the case of the spine) inadequate stabilisation of the joint(s) by the deep postural muscles.

3. Degenerative conditions are associated with longer-term, less easily reversible functioning of the joints and are much more common in mature athletes.

While previous acute or chronic injuries are known to increase the risk of long-term degeneration, simple ageing factors also come into play. These conditions frequently include:
● arthritic-type wear and tear, where the articular cartilage becomes worn, leading to narrowed joint spaces, sometimes referred to as osteoarthritis;
● rheumatoid arthritis, an inflammatory condition of the joints caused by an auto-immune reaction;
● low synovial fluid secretions, leading to reduced lubrication in the joint capsule.

Common to all these causal factors is the process of inflammation which, although part of the normal healing process, can actually impede this process when it becomes chronic.

The role of nutrition in combating degenerative or inflammatory joint conditions has traditionally been regarded with scepticism, but in recent years research has indicated that good nutritional practice can play a significant role, both in promoting recovery from acute and chronic injuries and in ameliorating some of the effects of the degenerative conditions described above.

The latest thinking

We'll begin by taking a look at the latest thinking on optimum

dietary practice, then move on to examine the claims of some of the more esoteric 'joint health' nutrients on offer.

As with all athletes, it is important for older athletes looking to maximise joint health to consume a whole, natural and unprocessed diet, rich in fruit, vegetables, complex carbohydrates (such as whole grains, starchy vegetables, beans, peas and lentils) and high quality, low-fat sources of protein, keeping processed, refined, fatty and sugary foods to a minimum. However, there are a number of nutrients that are particularly important for older athletes, which should be well supplied in their diet. These and their effects are described below.

Vitamin C

Among other roles in the body, vitamin C is vital for the formation of collagen, which is a protein forming the basis for connective tissue, such as tendons and intervertebral discs. Vitamin C activates the enzymes that convert proline and lysine into hydroxyproline and hydroxylysine respectively, both of which are needed to give collagen its correct 3D structure.

Omega-3 oils

Prostaglandins (PGs) are short-lived hormone-like chemicals synthesised from dietary fatty acids to regulate cellular activities. There are three families of prostaglandins – series 1, 2 and 3. Series 1 PGs play a number of roles in the body, including exerting an anti-inflammatory effect. By contrast, series 2 PGs exert an *inflammatory* effect (remember that inflammation can be a good thing when it is required!).

While series 1 and 2 PGs are synthesised from the omega-6 essential fatty acid, 'linoleic acid', series 3 PGs are synthesised from the other essential fatty acid, omega-3 alpha-linolenic acid. One of the intermediate steps during the conversion of alpha-linolenic acid to series 3 PGs involves the formation of eicosapentaenoic Acid (EPA). EPA acts to inhibit the excessive formation of the inflammatory series 2 PGs, and this explains why omega-3 oils exert an anti-inflammatory effect in the body,

and why fish oils (which contain 'ready-made' EPA) have been shown to have the same effect.

Sulphur-containing amino acids (SAAs)

Sulphur has long been recognised as an essential nutrient for human health. In the diet, sulphur is found in a number of forms, but mainly as the sulphur-containing amino acids methionine, cysteine and taurine. Dietary sulphur is also present as inorganic free sulphate and loosely bonded sulphates. Because these forms are present in much smaller amounts, they have been considered relatively unimportant. However, recent research has shown that inorganic sulphates in the diet can be used not only to synthesise cysteine and taurine, but also to synthesise the chondroitin matrix of joint cartilage[1]. (Chondroitin helps to promote water retention and elasticity in joint cartilage and inhibit enzymes that break down cartilage.)

In the body, sulphur is present in a number of compounds critical for joint function and health (in addition to the SAA amino acids). Glutathione is a powerful antioxidant, which can be depleted during heavy training. If intakes of the SAAs methionine and cysteine are sub-optimal, cysteine can be preferentially incorporated into body proteins, producing a pro-inflammatory response[2].

Chondroitin sulphate is a sulphur-containing polysaccharide essential for joint cartilage health, while glucosamine is an amino-acid-containing monosaccharide, concentrated in joint cartilage, which is used to synthesise cartilage glycosaminoglycan (GAG for short) GAGs are large molecules comprising long-branched chains of sugars and smaller nitrogen-containing molecules known as amino-sugars.

Methlysulphonylmethane (more commonly known as MSM) is another sulphur-containing compound found in some foods, which is also present in the body. Although the biochemistry of MSM is poorly understood, it appears to be able to donate some of its sulphur for the formation of connective tissue and may also have an anti-inflammatory

effect. Meanwhile S-adenosylmethionine (SAMe) is another sulphur-containing compound in the body (produced from the metabolism of methionine), which also appears to exert an anti-inflammatory effect. We'll revisit these last four compounds later in this article.

Bioflavanoids

These are naturally occurring compounds found mainly in fruit and vegetables, which appear to possess anti-inflammatory properties in addition to their antioxidant effects. Animal studies on two such compounds, rutin and quercetin, have demonstrated significant anti-inflammatory effects in both acute and chronic inflammation[3]. Furthermore, there is also evidence that these compounds improve local circulation and promote a strong collagen matrix in joints[4].

Antioxidants

When free radical damage occurs in joint linings, inflammation can be increased. There are a number of antioxidant nutrients that afford protection from free radical damage in the body, but selenium and vitamin E appear to be especially important. Vitamin E has been shown to help combat the effects of exercise-induced oxidative stress (which increases free radical production), while selenium is an essential component of the critically important antioxidant enzyme called glutathione peroxidase, as well as being involved in the production of the prostaglandins and substances known as leukotrienes that are also involved in regulating inflammatory processes[5].

Zinc and copper

Zinc is an important mineral, activating numerous enzyme systems in the body. These include enzymes that process amino acids in the body (including the SAAs) – a process known as transamination. Zinc also functions as an antioxidant and is able to protect sulphur-containing bio-molecules from oxidation. Additionally, sub-optimum intakes of zinc are known to impede the formation of collagen[6]. Like zinc, copper is

needed for important antioxidant enzymes (eg superoxide dismutase) and is also required for collagen formation.

Key sources of these nutrients are listed in Table 1, below.

Table 1: Dietary sources of key nutrients

Nutrient	Good sources in the diet
Vitamin C	Grapefruit, lemons, oranges, kiwis, strawberries, raspberries, blackberries, blackcurrants, pineapple, papaya, peppers, tomatoes, cabbage, broccoli, Brussels sprouts, new potatoes
Omega-3 oils	Walnuts, pumpkin seeds, flax and flax-seed oil, herring, trout, mackerel, salmon, sardines, pilchards, wheat germ
Bioflavanoids	All fruit and vegetables, especially citrus fruit (particularly the pith), apricots, cherries, grapes, green peppers, tomatoes, broccoli. Buckwheat (a cereal) is also a good source
SAAs	Broccoli, cabbage, onion, garlic, eggs, meat, poultry, fish, milk and cheese, oats, corn, sunflower seeds
Vitamin E	Almonds, sunflower seeds, spinach, wheat germ, whole grain breads and cereals, cold-pressed seed oils, egg yolk
Selenium	Brazil nuts (extremely good source), tuna, whole grain breads and cereals, swordfish, herring
Zinc	Oysters, lean beef, pumpkin seeds, lamb, peanuts, crab meat, pork, sunflower seeds, wholemeal flour and bread, turkey
Copper	Beef liver, oysters, lobster, sunflower seeds, hazelnuts, crab, baked beans, chickpeas, lentils, wholemeal bread and whole grain cereals

In addition to ensuring a good supply of the above nutrients, it is important to avoid excessive intakes of saturated fats from red meats, full fat dairy produce, etc, as these tend to be rich in arachidonic acid, which is a precursor to the inflammatory series 2 PGs. Likewise, too much omega-6 and insufficient omega-3 oils (a common imbalance in Western diets) enhances the production of series 2 PGs.

The importance of the sulphur amino acids is worth

emphasising. The US committee on recommended daily amounts suggests a combined SAA intake of around 1 gram per day for a typical adult. Other authorities believe this figure is too low and should be closer to 2g per day[7,8]. Given that the stress of heavy training can deplete blood glutathione, which is an important peptide and reservoir for the SAA cysteine, athletes need to take more care than most[9,10]. This is especially true for those on low protein or strict vegetarian diets, which tend to supply lower levels of SAAs per calorie consumed. Vegetarians may wish to note that corn, sunflower seeds, oats, chocolate, cashew nuts, walnuts and almonds are all good very sources of methionine, while oats and corn are high in cysteine too.

Assuming that your diet is optimal, are there any food supplements that can further improve joint health, both in terms of helping to overcome acute and chronic injuries and in combating the long-term degeneration that is an inevitable part of the ageing process? Those which might be useful are described below.

Glucosamine sulphate

Glucosamine is used in the manufacture of very large molecules found in cartilage, called proteoglycans. These are large linear chains of repeating polysaccharide units (GAGs), which radiate out from a protein core like the bristles of a bottlebrush and can attract and hold water like a sponge. When compressed, this bound water helps to absorb force and distribute it equally, which explains the ability of cartilage to protect the joints under load and during movement.

In the body, these GAG chains are synthesised from glucose, the amino acid glutamine, and sulphate, but there's plenty of evidence that additional supplementation not only increases GAG significantly but can also relieve the pain and inflammation associated with osteoarthritis[11].

Recently, researchers carried out an exhaustive meta-analysis of all the randomised, placebo-controlled clinical trials (RCTs) on the efficacy of oral glucosamine that were published or performed between January 1980 and March 2002[12]. They

concluded that the supplement was not just highly effective in reducing pain and increasing mobility but also reduced the joint space narrowing that typically occurs in degenerative conditions.

The long-term benefits of oral glucosamine sulphate appear to be supported by a three-year study in which 200 patients with osteoarthritis of the knee were randomised to receive either oral glucosamine sulphate (1500mg daily) or placebo. By the end of the study, average joint spaces had reduced by more than 5% in the placebo group, while the glucosamine group showed no narrowing at all! Moreover, pain and stiffness was significantly reduced in the glucosamine group by comparison with the controls[13].

The pain relief afforded by glucosamine is also significant. In a mini meta-analysis of two double-blind RCTs, oral glucosamine sulphate (1.5g/day) was compared with ibuprofen (1.2g/day) for the relief of joint pain in osteoarthritis[13] and was shown to be equally effective. Even more persuasive is the fact that many non-steroidal anti-inflammatory drugs (NSAIDS), including ibuprofen, have been shown to inhibit the repair and even accelerate the destruction of cartilage[14]. In fact, the only drawback of using glucosamine is that the benefits take a while to accrue, with most users finding it takes a good six weeks-or-so before the full effects are felt.

Chondroitin sulphate

This is another of the GAG polysaccharides found in cartilage. But whereas glucosamine appears to promote the formation and repair of cartilage, chondroitin seems to promote cartilage water retention and elasticity. Initially it was believed that, as a big molecule, chondroitin couldn't be absorbed intact. But subsequent research has shown not only that up to 15% is absorbed whole but also that, once in the body, chondroitin makes a beeline for GAG-rich tissues such as the joints and lumbar discs[15].

Although fewer studies have been carried out on chondroitin than on glucosamine, the evidence points very strongly towards its efficacy. In the large meta-analysis described above[12], the

outcomes of supplementing chondroitin were also observed and it was found to be effective in reducing pain and increasing mobility. Moreover, chondroitin also appears to offer long-term benefits in arthritic conditions. In a one-year Swiss RCT 42 patients with knee pain, those taking 800mg of chondroitin per day showed significantly reduced pain and increased overall mobility compared with those on placebo [16]. In addition, the metabolism of bone and joint, assessed by various biochemical markers, stabilised in the chondroitin group but remained abnormal in the placebo group.

Methylsulphonylmethane (MSM)

This micronutrient is extremely rich in sulphur (containing 34% elemental sulphur by weight) and is found in small amounts in fruit, alfalfa sprouts, tomatoes, tea and coffee. Despite its rapidly growing popularity as a supplement, its metabolism in the human body remains poorly understood. One study found that 97% of orally ingested MSM is converted into other metabolites [17], while its very high sulphur content has led researchers to speculate that it could act as a sulphur donor in the synthesis of sulphur amino acids. However, studies on guinea pigs, using radio-labelled MSM, showed that only 1% of the sulphur is actually incorporated, so this seems unlikely [18].

By comparison with glucosamine and chondroitin, scientific studies of MSM supplementation for joint health are thin on the ground. In a preliminary study carried out on 16 patients with degenerative arthritis, one group received 2250mgs per day of MSN and the other a placebo [19]. After six weeks, eight out of 10 patients in the MSM group experienced significant pain relief compared with just one who experienced minimal pain relief in the placebo group.

Another RCT was conducted on athletes with acute injuries, who were undergoing routine chiropractic manipulation, ultrasound and muscle stimulation [20]. On average, those taking MSM were discharged from care after just 3.25 visits and experienced a 58.3% reduction in symptom severity, while those on placebo needed 5.25 visits and experienced a

reduction in symptom severity of just 33.3%. While these results are encouraging, both of these studies were sponsored by suppliers of MSM and further, independent peer-reviewed trials are needed before firm conclusions about the efficacy of MSM can be drawn.

S-adenosylmethionine (SAMe)

This is produced in the body by the metabolism of the SAA methionine and, like methionine, SAMe is used in a number of metabolic processes that require sulphur. Normally the body can synthesise all it needs, but low intakes of methionine, or of other co-factors needed (choline, folic acid) or an inherited defect in the ability to carry out a biochemical process known as methylation, are all thought to reduce the body's ability to make SAMe.

Orally supplemented SAMe has been shown to stimulate the synthesis of cartilage proteoglycans and to be as effective as commonly prescribed NSAIDS (eg ibuprofen) for pain relief[21]. A large meta-analysis of RCTs on SAMe found SAMe as effective as NSAIDs in relieving pain and improving functional limitation in patients with osteoarthritis, without the adverse effects often associated with NSAIDS[22]. SAMe therapy for joint pain may also offer an advantage over glucosamine in that pain relief appears to occur relatively rapidly – within two weeks[23].

In summary, despite the fact that older athletes are more vulnerable to chronic joint pain and stiffness, you are not powerless to act. While it is obviously vital to get your training right, and to incorporate any other rehab/injury prevention techniques deemed necessary by your coach/trainer/physiotherapist, there is also a place for nutrition.

Your number one priority should be to follow the dietary recommendations outlined earlier, paying special attention to the key joint health nutrients. Only then should you consider supplementation. On the available evidence, glucosamine and chondroitin, supplemented at around a gram per day, both offer effective pain and stiffness reduction, and even appear to

be able to slow down the process of cartilage degeneration itself. Their regenerative mode of action means they need to be supplemented on a long-term basis (ie for six weeks or longer), but for those prone to chronic joint stiffness and pain, there's no reason not to take them indefinitely, especially as they are relatively inexpensive.

One word of caution: virtually all the studies on glucosamine have been carried out using glucosamine sulphate and this is the recommended form to use. The evidence in favour of MSM is much less convincing. True, there are promising signs, but there is simply too little peer-reviewed scientific evidence in the literature to recommend its use unreservedly. Nevertheless, it is also one of the least toxic substances known in biology, so if you want to try it and see for yourself, there's little to worry about[24]. Although probably less familiar to most readers, and less extensively researched, SAMe (supplemented at 0.5-1g/day) looks promising for helping to combat joint degeneration. However, drawbacks include its high cost and its chemically fragile nature, which means it needs to be stored in a cool, dry, dark environment from the point of manufacture to consumption. SAMe is also being studied as an alternative and more 'natural' anti-depressant; it appears to exhibit significant anti-depressive activity in some people by increasing the levels of two brain neurochemicals, serotonin and dopamine. However, if you are currently taking any anti-depressant medication or receiving any other psychiatric treatment, you should not experiment with SAMe without first consulting your doctor.

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The bad news is that speed declines with age; the good news is that you can arrest and even reverse this process

Of all the physiological variables, speed seems to get written off most quickly as we get older. Football pundits make jokes about outfield players being 'a few yards slower' and goal-keepers diving in 'instalments' as soon as the former hit 30 and the latter become David Seaman. But England's Rugby World Cup winning pack averaged well over 30, and despite being called 'Dad's Army' still fathered a victory: the likes of Neil Back and Martin Johnson were certainly very speedy around the field. In track, Carl Lewis, Frankie Fredericks, Linford Christie and Merlene Ottey are – or were – still winning titles well into their 30s and, in the case of Ottey, beyond. But can masters athletes still put in speedy sprinting performances in their 40s, 50s, 60s – and beyond?

First, let's take a look at why we slow with age? One significant factor is a decline in muscle mass and muscle fibre (sarcopenia). We will all experience a 10% decline in muscle mass between the ages of 25 and 50 and a further 45% shrinkage by our eighth decade – if we do nothing about it. To illustrate this decline by example, the biceps muscle of a newborn baby has around 500,000 fibres, while that of an 80-year-old has a mere 300,000. As we age, we also produce less growth hormone, which leads to reduced levels of protein synthesis and, again, muscle atrophy. This is not the kind of acceleration needed by the older athlete in search of speed

because decreased muscle equates to reduced strength and power and less oomph for sprinting.

To make matters worse

Unfortunately, the bad news keeps on coming! Fast-twitch muscle fibre, that most precious of commodities for speed and power, displays a much more marked decline than slow-twitch fibre as we age. Speedsters, it appears, are not as blessed as endurance athletes in the ageing-and-performance stakes. The latter can expect to maintain their slow-twitch fibres and even increase them – by as much as 20% with the right training – as they ripen. They can also hold on to nearly all their aerobic capacity until late into their fifth decade at least. If only it were so for their sprinting counterparts, whose fast-twitch fibres can decline by as much as 30% between the ages of 20 and 80.

To add another blow, creatine phosphate, that premium ingredient for short-term physical activity, also declines with age. With less quick-release energy in our muscles, we're theoretically less able to tackle high intensity sprint-type workouts.

Flexibility, another important physiological variable for sprinting (and injury prevention), also declines with age as our soft tissue hardens and our joints stiffen (see later in this special report).

What are the known effects on performance of these various reductions in capacity? It gets worse! Numerous studies have indicated that stride length declines considerably with age. Korhonen analysed the performances of 70 finalists (males 40-88, females 35-87) in the 100m event at the European Veterans Athletics Championships in Jyvaskyla, Finland, in 2000, using high-speed cameras with a panning video technique to measure velocity, stride length, stride rate, ground contact time and flight time[1]. Unsurprisingly, his research team discovered a general decline in sprint performance with age, which was particularly marked for those aged 65-70. Velocity during the different phases of the run declined, on average, between 5-6% per decade in men and 5-7% in women. Key to this decline was an accelerating reduction in stride length and an increase in

contact time, with stride rate remaining largely unaffected until the oldest age groups in both genders.

Twice as many steps

Hamilton compared 35-39-year-old runners with 90-year-olds and found that stride length declined by as much as 40%, from 4.72 metres per stride (2.36 metres per step) to 2.84 metres per stride (just 1.42 metres per step). The implication is that the oldest sprinters may need to take almost twice as many steps in the 100m than their younger counterparts. More positively, though, this research group also found that stride frequency did not decline significantly with age [(2)].

If you take a look at table 1, below, you'll read some much better news. Take note of the phenomenal times recorded by

Table 1: Masters world age records

Age group	Time (secs)	Athlete	Age when record set	Country
40	10.84	Erik Oostweegel	40	NED
45	10.96	Neville Hodge	45	US
50	10.95	William Collins	50	US
55	11.57	Ron Taylor	57	GB
60	11.70	Ron Taylor	61	GB
65	12.62	Malcom Pirie	65	AUS
70	12.91	Patton Jordan	74	US
75	13.40	Patton Jordan	75	US
80	14.35	Patton Jordan	80	US
85	16.16	Suda Giichi	85	JPN
90	18.08	Kozo Haraguchi	90	JPN
95	24.01	Erwin Jaskulski	96	AUT
100	43.00	Everett Hosak	100	US

Source: World Masters Athletics Association as at 24/09/02

master 100m sprinters. These indicate that it is possible to maintain a significant amount of speed with age. So now let's take a look at what we have to do to achieve that goal.

Hill training for stride length

As we've seen, two crucial factors affecting speed decline in the older sprinter are a reduction in stride length and an increase in ground contact time. Hill sprinting can reverse these negatives; the gradient will emphasis dorsiflexion (a greater toe-up foot position) on foot strike, which will, in turn, generate more work for the calf muscles on push off, enhancing stride length and reducing contact time on the level. Lower limb and ankle strength and power are crucial for the sprinters of all ages, although they can be overlooked by coaches and athletes in favour of conditioning the quadriceps and glutes.

One of the key factors contributing to the age-related decline in stride length is the action of the free leg as it leaves the running surface and the foot travels a curvilinear path beneath the body to a forward position in preparation for the subsequent foot strike. An older runner's 'return phase' is much less dynamic than that of his or her younger counterparts. For optimising speed transference into the next running stride, the lower leg needs to 'fold up' towards the butt and be pulled through quickly and powerfully as a short lever. This action relies on hip, glute and hamstring strength.

Returning to Hamilton's work, she and her co-workers discovered that range of motion at the knees during running decreased by 33% – from 123 degrees to just 95 – between ages 35 and 90. For the oldest runners in the study, this meant that the lower part of the leg attained a right angle with the thigh at the point of maximum flexion, dramtically slowing free leg transition into the next stride.

Hill sprints can play a key role in combating this lower leg lethargy; by creating a greater leg drive, they can increase the speed of the free leg through reaction to the ground and condition a much more effective and speedy biomechanical sprinting action.

Weights for fast-twitch maintenance

Weight training is crucial for mature sprinters determined to hang on to as much zip as possible, particularly after 50, when muscle mass begins to decline more steeply. Training with weights set around 75% of one rep maximum will offset fast-twitch fibre shrinkage significantly. Unfortunately, though, it has no impact on muscle fibre reduction, which is governed by an age-related decline in motor cells in the spinal cord.

Weight training, by strengthening soft tissue, will also go someway towards protecting older speed merchants from injury.

Plyometrics for stretch/reflex

Plyometric exercises condition the stretch/reflex in our muscles and, as well as boosting speed and power, can stimulate the fast-twitch fibres of older sprinters into further action. As mentioned above, stride length declines significantly with age and plyometrics, like hill training, offer another significant training option for offsetting this decline. Bounding and hopping are two very effective exercises for enhancing stride length.

Intense exercise for GH release

Exercise is known to stimulate growth hormone (GH) release, which is crucial for speed maintenance in later life [3]. Growth hormone helps us hold on to more lean muscle mass, retain more energy and offset some of the general effects of ageing. The positive release of GH begins almost immediately after we start to exercise, and it seem that the higher the intensity of the exercise, the more GH will be released.

Stokes and co-workers compared the effects of maximal and less intense cycle ergometer sprinting in a group of 10 male cyclists, who completed 2 x 30s sprints separated by one hour's passive recovery on two occasions [4]. The first effort was completed against a resistance equal to 7.5% of body mass and the second to 10% of body mass. Blood samples were taken at rest, between the two sprints and one hour post exercise.

Analysis of blood samples showed that the first effort elicited a much more significant serum GH response than the second. Note that, although both sprints generated the same peak and mean power outputs, the first resulted in higher and mean RPM scores – ie, to pedal faster.

Despite the apparent attenuation of GH release in the second effort, since speed is maintained and enhanced by regular anaerobic training, older sprinters should benefit from regular and above-normal GH release from their training.

Creatine for muscle power

Intense speed and power training can also combat the normal age-related decline in creatine phosphate. Research has shown that anaerobic (and aerobic) training increases the production of creatine phosphate. Research by Moller and co-workers showed that six weeks of cycle ergometer training increased the creatine phosphate levels of 61-80 year olds to levels similar to those of younger adults[5]. The regular anaerobic workouts of sprint training will maintain and increase the ability of our muscles to replenish high-energy phosphates, regardless of age.

But since there's nothing wrong with giving Mother Nature a legal helping hand, the older sprinter should take supplementary creatine. Numerous studies have shown that creatine supplementation can increase muscle power and power maintenance over a series of anaerobic repetitions and will contribute to the maintenance of lean muscle mass.

One interesting piece of research that specifically addressed sprinting threw up some encouraging – and other slightly less encouraging – information for master sprinters supplementing with creatine. Schedel et al looked at whether the improvement in maximal sprinting speed after creatine supplementation could be attributed to an increase in stride frequency, stride length, or both[6]. Seven sprinters completed four consecutive sprints after one week of placebo or creatine supplementation. By comparison with the placebo condition, creatine-fed sprinters increased their running speed (+1.4%) and stride frequency (+1.5%), but not their stride length.

This research also substantiated the use of creatine for sustaining power output, as decline in performance of subsequent sprints was partially prevented after creatine supplementation. The researchers concluded that their findings could be related to the recent discovery that creatine supplementation can produce a shortening in muscular relaxation time, thus promoting increased sprint times.

Train smart for all-round benefits

Finally, the older sprinter needs to make use of the wiser head on his or her shoulders. Training needs to be intense to minimise the age-related decline in sprint speed, but it also needs to take account of the fact that older bodies may be less able to sustain daily, flat-out power-oriented work. Rest, proper nutrition and supplementation, and a commonsense approach that involves 'listening to the body' need to be key features of the training routine of any veteran sprinter intent on maintaining speed.

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A sports physiotherapist shows why flexibility is so important to keep ageing at bay, and explains the science behind it

Achieving a certain degree of flexibility is absolutely critical for anyone involved in sports; otherwise there will be at some stage a breakdown in body tissues leading to an injury. Don't kid yourself if you never stretch: it is only a matter of when you get injured, not if. In addition, if you are too tight in certain parts of your body, you are functioning below your real potential – remember that performance enhancement is the second very important reason to stretch: flexible muscles perform a lot better than tight muscles.

In order to improve flexibility, it's important to first understand some of the science underpinning the principles of stretching. This is also critical in order to avoid direct injury from trying new stretches that you are unfamiliar with.

Most coaches, athletes and sports medicine personnel use stretching methods as part of the training routine for athletes. Many would agree that it forms an integral part of training and preparation. However, most of the theoretical and practical factors in stretching are often incorrectly applied. The purpose of this article is primarily to provide an overview on the theoretical basis of stretching routines.

What is flexibility? De Vries defines it as the range of motion available in a joint, such as the hip, or series of joints such as the spine. This encompassing definition takes into account a number of important aspects about flexibility. That is, it deals

with a joint or series of joints used to produce a particular movement, and it considers that flexibility is both static and dynamic in nature.

It is important to highlight some points regarding flexibility.

First, flexibility is joint specific. That is, you cannot say someone is flexible just because they can touch their toes. The same person may not even be able to reach around and scratch the small of his/her back because their shoulder has poor flexibility.

Second, flexibility is sport specific. You would not expect a front row rugby forward to have the same flexibility as an Olympic gymnast, because it is not required for his sport. In fact, in a contact sport like rugby, being that flexible would be detrimental to his body.

Components of flexibility

Flexibility has two important components: static and dynamic flexibility.

1) Static flexibility describes range of motion without a consideration for speed of movement. This is the maximum range a muscle can achieve with an external force such as gravity or manual assistance. For example, holding a hamstring stretch at an end-of-range position.

2) Dynamic flexibility describes the use of the desired range of motion at a desired velocity (usually quickly). Dynamic flexibility is the range athletes can produce themselves. For example, a javelin thrower or baseball pitcher needs a lot of shoulder rotational flexibility, but they also need to be able to produce it at rapid speeds of movement.

Here are some useful points:

(a) Good static flexibility is a necessary prerequisite for good dynamic flexibility; however, having good static flexibility does not in itself ensure good dynamic flexibility.

(b) Dynamic flexibility is vitally important in those high velocity movement sports such as sprinting, kicking and gymnastics.

(c) Dynamic flexibility is limited by the ability of the tissues to lengthen quickly, and the inhibition of what is called the 'stretch

reflex', which if present would act to limit the range of motion (more about this later).

Why is flexibility important?

Good flexibility allows the joints to improve their range of motion. For example, flexibility in the shoulder musculature allows a swimmer to 'glide' the arm through the water using shoulder elevation. This allows the joints to easily accommodate the desired joint angles without undue stress on the tissues around them. It therefore is essential for injury prevention.

Stretching also forms an integral part of rehabilitation programmes following injury. For example, it is accepted that a muscle tear will heal with scar tissue. This scar tissue tends to be functionally shorter and have more resistance to stretch than normal healthy muscle tissue. Therefore stretching is used at an appropriate time in the healing process to assist in lengthening this contracted scar tissue.

Good flexibility improves posture and ergonomics. Our bodies have a tendency to allow certain muscles to tighten up which will affect our posture. Vladimir Janda, a Czech rehabilitation specialist, describes a group of muscles in the body that universally show a tendency towards tightness and also being overactive in movements. Some of these include the hamstrings, rectus femoris, TFL, piriformis, adductors, gastrocnemius and quadratus lumborum. These muscles are often implicated in postural syndromes causing musculoskeletal pain.

Flexibility, because it allows good range of motion, may improve motor performance and skill execution. Think of a sprinter who needs flexibility in the hip flexors to allow good hip extension at toe off, and good hip extensor flexibility to allow necessary knee drive in the leg recovery phase of sprinting. Skill execution and reduced risk of injury will be greatly enhanced if the body has the flexibility necessary for that particular sport.

There is also an argument that stretching may reduce post exercise muscle soreness, or DOMS, by reducing muscle spasm associated with exercise.

Relative flexibility

Shirley Sahrmann, an American physiotherapist, uses the term 'relative flexibility' to describe how the body achieves a particular movement using the relative flexibility available at a series of joints. She believes that in order for the body to achieve a particular range of motion, it will move through the point of least resistance, or area of greatest relative flexibility.

A good example is to think of a rower at the bottom of the catch position. In this position the rower must have his hands (and the oar) past his feet in order to generate the drive necessary to transfer force from his body to the oar. If for some reason the rower has excessively tight hips and can't bend up (or flex) the hips (usually due to gluteal tightness), his body will find somewhere else to move to compensate for that lack of hip flexibility. More often than not, this rower will flex the lumbar and thoracic spines to make up for the lack of hip flexion. That is, the back has more 'relative flexibility' and therefore contributes to the overall range of motion. In this case however, the back will exhibit movement that is more than ideal, possibly leading to lumbar and thoracic dysfunction and pain.

The concept of relative flexibility is vital when understanding movement dysfunction in athletes. It is imperative that joint movements are not looked at in isolation, for other more distant joints will influence that movement. Try this simple test to highlight this point. Sit on a chair with your upper back slumped (that is, assume a poor posture). Now, maintaining this position, try to elevate both arms above your head. Now straighten yourself up (assume a good posture) and try it again. Unless you have gross shoulder dysfunction, you will be able to elevate more with a straight back than a curved one. By assuming a slumped position, you prevent the upper back (thoracic spine) from extending. This extension of the upper back is necessary for full range elevation. Without extension, it is difficult for the shoulder to fully elevate. If you do this for long enough (months to years) eventually the lack of movement will attempt to be taken up elsewhere (such as the lower back, or the shoulder itself). This may eventually lead to breakdown of these joints

due to the excessive movement they may eventually demonstrate.

What factors limit flexibility?

Flexibility can be limited by what are called 'active' or 'contractile' and 'passive' or 'non-contractile' restraints.

Muscle contraction is one of these 'active/contractile' restraints. Flexibility can be limited by the voluntary and reflex control that a muscle exhibits while undergoing a stretch, in particular a rapid stretch that activates the 'stretch reflex'. As a muscle is rapidly stretched, a receptor known as a 'spindle' causes the muscle to reflexively contract to prevent any further stretch. If left unchecked, the stretch reflex would work to prevent elongation while the muscle was being stretched. A benefit of ballistic or fast stretching is that the nervous system learns to accommodate by delaying the stretch reflex until closer to end of range of movement (more on this later).

Furthermore, a resting muscle does not always mean that it is 'resting'. Muscles usually exist with a certain degree of muscle 'tone'. An increase in tone will increase the inherent stiffness in muscles. If you are scientifically minded, this describes the way actin and myosin remain bound and thus resist passive stretching of the muscle. The actin and myosin stay bound because of a constant low-level discharge in the nerves supplying that muscle. With actin and myosin unbound, a muscle should in theory be able to stretch to 150% of its original length (in theory of course).

'Passive/non-contractile' restraints in the form of connective tissues will also limit flexibility. The passive restraints include the connective tissues within and around muscle tissue (epimysium, perimysium and endomysium), tendons and fascial sheaths (deep and superficial fascia). The important microscopic structure to consider in passive tissues is collagen. The way collagen behaves with stretching will be discussed shortly.

Other passive restraints include the alignment of joint surfaces. An example of this is the olecranon of the elbow in the

olecranon fossa that will limit full extension (straightening) of the elbow. Other joint constraints include capsules and ligaments. The joint capsule/ligament complex of the hip joint is important in limiting rotation of the hip.

The nerves passing through the limbs can also limit flexibility. As a limb is taken through a full movement, the ropey nerve tracts also become elongated and become compressed. The nerve endings and receptors in the nerves trigger a reflex response that causes the muscle to increase its resistance to stretch.

Other points to note

In addition, a number of other factors influence flexibility.

1. An older muscle has more inherent stiffness due to the morphological changes in the muscle and collagen in the connective tissues.

2. A muscle that has been immobilised with a cast will demonstrate increase in stiffness over time (longer than four weeks).

3. Excessive training causes more crosslinking to occur between collagen fibres and therefore increases stiffness.

4. Excessive repeated muscle contractions cause high volumes of neural discharge. A muscle can remain in a state of high resting tone following training sessions.

5. Increase in temperature causes a decrease in muscle stiffness. This can be environmental temperature or temperature increases induced by friction of muscle contraction. We therefore tend to be less stiff around 2.00 in the afternoon.

6. Finally, an increase in intramuscular fluid (fluid in the muscle cell) can increase stiffness due to a splinting effect. This is the suggested reason why use of creatine monohydrate tends to make muscles feel stiffer.

More about collagen

It has been mentioned above that the connective tissues in and around muscle are considered to be 'passive' or 'non-contractile'. The principal structure in these tissues that we

need to consider is collagen. A key term used in physics and biomechanics to describe the way collagen behaves is 'viscoelasticity'.

Viscoelastic tissues are made up of viscous and elastic properties. A viscous tissue will deform and stay deformed permanently – if you pull on a piece of play dough, for instance, it will keep that shape. An elastic tissue will return to its original length when the force is removed. For example, pulling on a rubber band and letting go – the band snaps back to its original length. Viscoelasticity describes a property of tissues (collagen being one of them) whereby deformation/lengthening of a tissue is sustained and the recovery is slow and imperfect when the deforming force has been removed. That is, it will stretch, stay stretched for a while before slowly returning to its original length.

Viscoelasticity tells us a number of practical things about stretching the connective tissues in muscle:

1. Studies on the cyclic loading of tissues suggest that most deformation occurs in the first stretch and after four stretches there is little change in ultimate length. Thus there is no extra benefit from stretching a muscle 10 times in one session.

2. It takes 12-18 seconds to reach stress relaxation, so there is no need to hold a stretch for longer than 20 seconds.

3. Greater peak tensions and more energy are absorbed the faster the rate of stretch. This means that a tissue will generate greater tension if the rate of stretch is faster and thus not achieve the same length as a tissue undergoing a slow stretch. That is, do passive stretches *slowly*.

4. Once elongated, length changes are not rapidly reversible due to the viscous nature of the tissue. However, deformations are not permanent since the elastic properties will eventually bring the tissue back to its original length. Lasting changes come from adaptive remodelling of the connective tissues, not mechanical deformation. One study in South Africa showed that stretching every four hours was the most effective way to achieve elongation in a muscle. This may suggest that the temporary change in length following a stretch may start to

regress after four hours (Grace Hughes, unpublished study).

How stretching happens

A number of physical properties of viscoelastic tissues help describe how these tissues elongate with stretching. These are creep, load relaxation and hysteresis.

Creep describes the ability of a tissue to elongate over time when a constant load is applied to it. For example, if we applied 10kg of force to our leg in order to stretch our hamstring, we may initially get our leg to 90 degrees before our tissues prevented further movement. If we sustained that load, we would find that our leg would gradually 'creep' a few degrees over a period of time.

Load relaxation describes how less force is required to maintain a tissue at a set length over time. Using the above example again, if we applied 10 kg of force to get our leg to 90 degrees, we would find that less force would be needed (9,8,7kg etc) to keep it at 90 degrees.

Hysteresis describes the amount of lengthening a tissue will maintain after a cycle of stretching (deformation) and then relaxation. Again, let's assume that if we gained an extra 10 degrees of range in hamstrings after the stretches described above, we would maintain that range for some time after the load was removed.

Neuromuscular considerations

Certain neuromuscular mechanisms acting on muscles influence 'tension' and have important implications for the value of stretching. These mechanisms include the stretch reflex, autogenic inhibition and reciprocal inhibition.

1. The stretch reflex is governed by a long thin receptor in muscles called a 'muscle spindle'. The spindle's role is to let our feedback systems know about muscle length and the rate of muscle lengthening. When a muscle is rapidly stretched, the spindle (via a loop of nerves) triggers a reflex contraction of the muscle being stretched. A high-speed stretch will therefore trigger the spindle and a reflex contraction of the muscle will

limit its ability to stretch.

2. The spindle is also responsible for the phenomenon known as reciprocal inhibition. What happens here is that if a muscle contracts, the opposite or antagonistic muscle will relax to allow the movement to occur without resistance. For example, if the quadriceps are contracted, the hamstrings should relax to allow the knee to straighten.

3. The Golgi Tendon Organ (GTO) is the important receptor to consider in 'autogenic inhibition'. The role of the GTO is to provide information on tension increases in muscles. This tension can come from contraction or stretch. The GTO connects with a small nerve cell in the spinal cord that inhibits or relaxes the muscle where the GTO is found. The GTO will trigger if a stretch is sustained (for longer than six seconds) or if the muscle contracts forcefully.

The way these mechanisms are utilised are discussed below under the heading of proprioceptive neuromuscular facilitation-type stretching.

The theory behind different stretching types

STATIC

Held static stretches are done so that the joints are placed in the outer limits of the available range and then subjected to a continuous passive stretch (gravity, weights, manual). One obvious benefit is that the chance of injury is minimal. This type of stretching is ideal to stretch the connective tissue/non-contractile elements since it makes use of the viscoelastic properties to cause elongation of the tissue. Furthermore, it makes use of autogenic inhibition to trigger a relaxation in the muscle (remember the six- second rule).

DYNAMIC

1. Dynamic range of motion: this describes a type of stretch whereby a muscle is taken through a full, slow and large amplitude movement. The opposing muscles are used to produce the force in this type of stretching. This type of stretching is done under control and is not jerky in nature.

2. Ballistic: the type that is done fast and rapidly and through large ranges of motion. An example is leg swings to stretch the hamstrings. The benefit of this type of stretching is that it is sport- specific to ballistic sports and it allows integration of the 'stretch reflex' if done quite often over a period of time. As the neuromuscular system adapts to this stretching, the stretch reflex will minimise its contribution to limiting muscle range.
3. Bouncing: similar to ballistic, but it is performed in small oscillations at the end of range.

The dangers of (2) and (3) are that they can lead to significant muscle soreness caused by the rapid lengthening of the muscle. This in itself initiates the stretch reflex and increases muscle tension. Furthermore, it fails to provide adequate time for the tissues to adapt to the stretch.

PNF (PROPRIOCEPTIVE NEUROMUSCULAR FACILITATION)

PNF uses the concept that muscle relaxation is fundamental to elongation of muscle tissue. In theory, it is performed in a way that uses the proprioceptive abilities of the GTO and muscle spindle to relax or inhibit the muscle in order to gain a more effective stretch. It does so using autogenic inhibition and reciprocal inhibition.

PNF stretching exists in a number of different forms, but the only ones discussed here will be the contract relax (CR), hold-relax (HR) and contract relax and antagonist contraction (CRAC) methods.

a) Contract relax (CR)

The muscle to be stretched is passively taken to end of range. Maximum contraction of the muscle to be stretched is performed against resistance (usually another person). With this form of contraction, the muscle is allowed to shorten during an isotonic contraction. This is continued for at least six seconds (which allows autogenic inhibition to occur). The muscle is then relaxed and taken to a new range and held for about 20 seconds. This can be repeated 3-4 times.

b) Hold relax (HR)
Very similar to contract relax as above, but the contraction type is static/isometric. The muscle to be stretched is passively taken to end of range. Maximum contraction of the muscle to be stretched is performed against resistance (usually another person). With this form of contraction, the muscle does not shorten during its isometric contraction. This is continued for at least six seconds (allowing autogenic inhibition to occur). The muscle is then relaxed and taken to a new range and held for about 20 seconds. This can be repeated 3-4 times.

c) Contract relax antagonist contraction (CRAC)
The first part of this stretch is similar to the CR method above; however, when the muscle to be stretched is relaxed after its six-second contraction, the *opposite or antagonist* muscle is contracted for at least six seconds (allowing reciprocal inhibition to occur). The antagonist is then relaxed and the stretched muscle is taken to a new range.

Final thought

So there you have it! In the last few thousand words I have attempted to give a Readers Digest version of the background to the theory of stretching. Some of the theory is obviously difficult to grasp, and may challenge your existing preconceived ideas of stretching. But I promise you that, if you're an older athlete, these ideas will keep you flexible and able to perform better and longer.

Chris Mallac

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

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

What is ALC

Some readers may be familiar with the amino acid carnitine, which carries fatty acids into the mitochondria (the cellular furnaces) where they are 'oxidised' for energy. As its name suggests, acetyl L-Carnitine (ALC) is very similar, consisting of the same basic amino acid structure, with an acetyl group attached. 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 to cross cell membranes in general more easily. In plain English, ALC reaches parts of the body that L-Carnitine just can't reach! In addition to its role in mitochondrial activity, ALC is involved in the production of the key brain neurotransmitter acetylcholine and is also able to donate its acetyl group in a number of other biochemical reactions.

What is ALA?

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

The initial excitement about ALC/ALA supplementation began when a team of researchers in California in 2002 fed elderly rats both nutrients for a period of seven weeks and then compared them with young rats[1]. They were testing the theory

that mitochondrial decline is caused by free radical damage (see panel below). There was already evidence that supplementation with ALC could reverse the age-related decline in mitochondrial activity in rats, increase fatty acid oxidation and boost general metabolic activity[2]. However the down side to this increased mitochondrial function was that more oxidative damage occurred[3], so the researchers 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

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.

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!

The implications for human health

These studies on rats caused a huge stir within the scientific community. Here was evidence that some of the processes of ageing could be slowed or even reversed, and the implications for human health and performance were enormous. In the months that followed, a number of human studies were started, many of which are still under way.

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

One of the earliest studies examining the effect of ALC and ALA in humans was carried out at San Francisco State University in 2001. In a double-blind, placebo-controlled study lasting 17 weeks, 18 healthy sedentary men aged 60-71 were randomised to one of two treatment egimes: a placebo tablet twice a day or 1000mgs of ALC and 400mgs of ALA in two divided doses. Both groups were then asked to perform a demanding sequence of exercises, after which blood was drawn and analysed for signs of exercise-induced oxidative stress (a measure of how efficiently and cleanly the mitochondria produce energy). 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 registered more positive results with the ALC/ALA combination than with placebo.

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. However, the same research group is currently carrying out a much larger clinical trial in Boston, the results of which are due to be published this year.

Other human studies are also currently under way, but so far there are no published human studies available, although positive studies in animals continue to proliferate. 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 a number of other studies on mice and rats have shown that ALC/ALA supplementation reduces oxidative stress and improves mitochondrial function in a number of tissues, including brain, muscle and heart.

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

Signs of hope for athletes

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

✻ **ALC and human growth hormone (GH)**. A study carried out

in 2001 suggests that 500mgs of ACL combined with 25-100mg of the amino acid L-ornithine, taken at bedtime after a 3-4 hour fast, can boost nocturnal growth hormone release[6]. The reasons for this are unclear, but it seems that the body's normal hypothalamic GH release includes a 'whole body' mitochondrial 'feedback loop', which is controlled by systemic ALC levels. An increase in naturally-released growth hormone could enhance the recovery and repair processes that occur during sleep and which are vital to hard-training athletes;

✻ **ALA and acute altitude sickness**. In a study carried out on mountaineers, researchers investigated whether free radical damage to the blood-brain barrier could be implicated in the condition of acute altitude sickness[7]. Eighteen mountaineers were randomised into two groups, with one group taking a combination of ALA, vitamin C and vitamin E for three weeks before and during the ascent to a base camp at 5,180m, and the other group taking a placebo preparation. The ALA group not only experienced fewer symptoms of acute altitude sickness, but also demonstrated significantly higher resting arterial oxygen saturation levels, suggesting that ALA supplementation could be a useful aid for high-altitude endurance athletes;

✻ **ALA and age-related inflammation**. Studies carried out in vitro in 2002 showed that ALA reduced the markers of chronic age-related inflammation typically seen in human cells[8];

✻ **ALC and mood**. Motivation and positive mood are crucial for athletes training and competing at the highest level. In a study carried out on mildly-depressed patients, 12 weeks of supplemental ALC not only improved scores on the Hamilton Depression Rating Scale, but also showed positive chemical changes (detected by MRI scans) in the frontal portions of the brain, suggesting that further studies are warranted[9].

For athletes in hard training, the prospect of preventing or even reversing some of the age-related decline in physical performance is enticing, holding out the promise of longer

careers, including more sustained levels of peak performance. However, as is so often the case with new and unfolding nutritional research, it is difficult to make hard and fast recommendations about the benefits 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.

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 600mgs of ALA per day, while studies showing that ALC improves brain function in Alzheimer's patients[10] have used between 1,500 and 3,000mgs per day. However, the human study carried out in San Francisco, which used 400mgs of ALA and 1,000mgs of ALC per day was overseen by the same team that carried out the initial rat studies, so that might be a good place to start.

One final point: don't confuse Acetyl L-Carnitine (ALC) with L-Carnitine. While it is considerably cheaper, L-Carnitine does not have the same bioavailability as ALC and has not been

used in studies on mitochondrial decline. And while L-Carnitine has often been promoted as a popular 'fat burning' and endurance supplement, there is actually very little evidence for this in the scientific literature.

Andrew Hamilton

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WHAT THE SCIENTISTS SAY

This roundup of recent research from the scientific, medical and sports journals looks at the problems and possibilities of older athletes

How fitness protects the ageing brain...

An important US study has provided the first empirical proof that aerobic fitness protects the brain as well as the body from the degenerative effects of ageing.

The human brain gradually loses tissue from the third decade onwards, with concomitant declines in cognitive (intellectual) performance. And given the projected rapid growth in aged populations and the staggering costs associated with geriatric care, identifying mechanisms that may reduce or reverse brain deterioration is rapidly emerging as an important public health goal.

Previous research has demonstrated that aerobic fitness training improves cognitive function in older adults and can enhance brain health in aging laboratory animals. But no previous study has demonstrated a direct protective effect of fitness on aging brain tissue.

The participants in the current study were 55 right-handed, high-functioning, community-dwelling over-50s, recruited locally by researchers based at the University of Illinois. All had their brains scanned by high-resolution magnetic resonance imaging (MRI), with the resultant images scanned for systematic variation in tissue density as a function of age, aerobic fitness (as assessed by a one-mile walk protocol) and other health markers.

Consistent with previous studies, the researchers observed substantial age-related deterioration in tissue densities in the frontal, parietal and temporal cortices of the brain. More importantly, though, they found that losses in these areas were substantially reduced as a function of cardiovascular fitness, quite independently of other 'moderator variables', including alcohol and

caffeine consumption, HRT, education and hypertension.

The researchers state that: 'the role of cardiovascular fitness as protector and enhancer of cognitive function and [central nervous system] integrity in older adults appears to have a solid biological basis'. The results also 'suggest a rather simple and inexpensive mechanism to ward off the effects of senescence on human brain tissue'.

Most importantly, the regions and tissue that show the greatest sparing with aerobic fitness are the ones that play central roles in successful everyday functioning, while declines are associated with a variety of clinical syndromes, including schizophrenia and Alzheimer's disease.

'Future research examining the benefits of aerobic fitness on the functional and structural integrity of the CNS in clinical populations seems both promising and highly desirable', the researchers conclude.

(J Gerontol A Biol Sci Med Sci 2003 Feb;58(2):176-180)

... and improves memory in mid-life

Recent British research has linked physical activity in the mid-30s with a significantly slower rate of memory decline in middle age. But it also suggests that this protective effect is lost in those who do not maintain their fitness.

Participants in this study, carried out by researchers from University College, London, were the study population of the Medical Research Council National Survey of Health and Development, also known as the British 1946 birth cohort. This initially consisted of 5,362 children born during one week in March 1946. The cohort has been studied on 21 occasions for a variety of reasons between birth and age 53 and has been shown to be a representative sample in most respects of the UK population born in the immediate post-war era.

For the current study, the researchers tested the association between two kinds of activity at 36 years – physical exercise and spare-time activity – with verbal memory at age 43 and 53 years

in a total of 1,919 of the surviving members of the cohort.

Physical activity was assessed by a questionnaire asking about engagement in a choice of 25 sports and recreational activities in the previous month, with those who engaged in 'any' activity being compared with those who did 'none'. Participants were also asked about current engagement in seven spare-time activities (eg chess, church, the arts, voluntary work etc), with a total spare-time score obtained by adding activities together.

Both kinds of activity were significantly and positively associated with memory performance at age 43, independently of such other variables as sex, education, occupational social class, IQ and physical and mental health. Furthermore, physical exercise at 36 (but not spare time activity) was associated with a significantly slower rate of memory decline from 43 to 53 years. There was also evidence that continuing exercise after age 36 was important for protection, since those who gave up exercise after 36 years did not show the same benefit as those who were still exercising at age 43 or had taken it up for the first time.

The researchers comment: 'Not only does this strengthen the suggestion of a causal link between physical activity and protection of memory in mid-life, but it also implies that the cognitive benefit of physical exercise is enhanced by persistent or more recent activity. Conversely ... these findings suggest that this benefit is lost if activity is not maintained. Our study suggests that uptake of physical exercise in young to middle adulthood benefits memory, an aspect of cognitive function likely to be important for conduct of activities of daily living during ageing. Furthermore, sustained physical activity appears to reinforce this benefit, whereas abandonment of this activity appears to result in its loss'.
(Social Science & Medicine 56(2003):785-792)

Power v endurance: what goes first in the ageing stakes?

A fascinating US study based on world record statistics has made it clear that ageing diminishes muscle power considerably sooner and more dramatically than endurance in both men and women.

The researchers compared age-related changes in athletic performance, as reflected in world records for stationary rowing and power-lifting, to test the theory that ageing affects activities requiring short bursts of muscle power more than those based on endurance.

World-record performance data were plotted for each event across the age groups, from under-12 to 80-89 for one-hour stationary rowing, and from 20-35 to 80-plus for Olympic power-lifting. Heavyweight and lightweight-class world records were plotted for men and women in each age group.

Key findings were as follows:

* In the heavyweight-class men's rowing event, performance rises rapidly and peaks in the third decade. From age 25-85 performance decreases by 29%, with a gradual decline of just 4% from 25-55 and a more rapid decline thereafter of 0.83% per year. These values indicate a strong relation between performance and age for the entire age range, with the curve for lightweight-class men's records following a similar trend but at 0-6% lower performance;
* Data for women in the same age groups show similar trends but at a lower level. Notably, however, whereas men's performance peaks in the third decade, women's peaks in the fourth decade. From age 35-55 there is a gradual (5%) decline in rowing performance, after which it declines more rapidly by 0.80% per year;
* For power-lifting, men's records show a performance peak in the third decade, rapidly decreasing by 3% per year until age 37 and then steadily declining by 0.9% per year from age 37.5 to age 85;
* Women's power-lifting performance also peaks in the third decade, rapidly decreasing by 3.4% per year until age 37.5 and then steadily decreasing by 1.2% per year until age 52.

'Our findings suggest', say the researchers, 'that a statistically significant difference exists in the effect of aging on the ability to engage in activities requiring explosive movements over short time intervals versus activities requiring greater endurance capacity. Moreover, rates of men's and women's age-related changes in these activities are similar.'

The findings also indicate, they add, that there is 'an inherent

loss of ability to produce powerful muscle contractions with increasing age, despite persistent training and otherwise good health. In other words, the effectiveness of training for development and maintenance of muscle strength decreases progressively with age'.

Although the biological mechanisms underlying this effect remain to be clarified, the researchers speculate that preferential loss of fast-twitch muscle fibre function with age – as reported in previous studies – may play a role.

From their particular viewpoint as orthopaedic surgeons and rehabilitation specialists, they suggest that some form of regular resistance training should be advocated for patients over 30 in an attempt to offset age-associated strength loss (see also John Shepherd's article on page 41 of this special report).
(Am J Orthop 2002 Feb;31(2):93-98)

Bone maintenance in older runners

Bone density can be maintained by running in older active men. That is the main conclusion of a US research team which followed a group of 54 male master (veteran) athletes ranging in age from 40 to 80 over a period of 5-7 years.

The subjects had already been recruited to a 20-year longitudinal study which began in 1986. The purpose of the authors of the current study was to relate changes in training volume (miles run per week and days per week of exercise) and VO_2 peak to changes in whole body, spine and hip bone mineral density (BMD) over a 4-5 year period.

All the subjects were actively training and competing in their respective athletic events at the time of baseline assessment of body composition, fitness and bone mineral density (as measured by dual-energy x-ray absorptiometry). The measurements were repeated 4-5 years later and the results compared in the light of subjects' self-reported training and performance data.

Analysis of these data showed the runners decreasing both mileage and the number of days per week they trained with age,

accompanied by a significant decline in 5k and 10k times – although not in marathon times.

Predictably, in the light of the decline in training, the subjects significantly increased body weight (1.4%), body fat (1%) and body mass index (2.6%), with significant decreases in VO_2 peak.

Between the two sets of tests, whole body BMD and bone mineral content (BMC) declined slightly but not significantly. Over the same period, however, a significant increase in spine BMC and BMD and an increase in hip BMC occurred.

'The fact that the spine and hip BMD are maintained while the (whole body) BMD declines suggests a site-specific influence of distance running', conclude the authors.

'These data suggest that BMD can be maintained as a result of continuous training in male runners. They also suggest that changes in training patterns (either increases or decreases in days per week of training or miles per week of running) have little influence on the ability to maintain skeletal mass.'
(J Gerontol A Biol Sci Med Sci 2002 Apr 57(4) M203-8)

Older athletes actually reduce the risk factors for heart disease

The various risk factors for coronary-artery disease usually increase as people get older, but senior athletes are definitely bucking that trend – even as they reach their 80s and 90s. In fact, in a study carried out at the University of Florida, a group of 21 ageing track athletes actually improved their cardiovascular profiles over a 20-year period.

The 21 athletes, who were race walkers and runners, reached an average age of 70 at the study's conclusion, but actual ages varied from 60 to 92. All of the subjects maintained regular aerobic training throughout the two decade period. Each athlete was assigned to one of three groups (high, medium, or low) based on the intensity of his training.

The athletes remained lean through the 20-year period, averaging only 17% body fat at the end. Mean body weight stayed exactly the same – 154 pounds – over the extended period, and

total blood cholesterol levels actually declined over the course of the study. Eleven of the subjects had cholesterol readings over 200 at the beginning of the research, while only four had such lofty levels at the end. Exercise wasn't the sole factor producing this change, however; as the subjects continued training, they also improved their diets and ate less saturated fat.

In addition, there was not an increased frequency of high blood pressure over the 20-year period, and average systolic and diastolic blood-pressure readings remained exactly the same. Likewise, none of the athletes developed diabetes mellitus or became obese (had greater than 25% body fat).

Only one real negative showed up: the number of athletes with total cholesterol/HDL cholesterol ratios greater than five increased from zero to six, mainly because HDL cholesterol ('good cholesterol') tends to decline as a result of ageing.

While the risk factors for cardiovascular disease normally increase between the ages of 50 and 70, athletes who stay active through this period seem to be able to prevent most negative changes from occurring. This is happy news for ageing exercisers, although it does *not* mean that it's okay to skip regular check-ups with your doctor.

(Changes in Coronary Artery Disease Risk Factors in 60 to 92-Year-Old Male Athletes at 20-Year Follow-Up, Medicine and Science in Sports and Exercise, vol.25(5), p. S74, 1993)

This exercise regime will boost bone density and lean muscle mass

For endurance athletes, two of the key problems associated with getting older are a gradual decline in muscle mass and a potential loss of bone density. The missing muscle makes it more difficult to run, cycle or swim powerfully, and the shrinking bones increase the likelihood of injury and osteoporosis.

Of course, running and cycling preserve leg muscles tissue, and studies have shown that running about 20 miles per week enhances bone density. Unfortunately, running has little impact on

upper-body muscle mass, and scientific studies have shown that runners who log 40-75 miles per week actually may have decreased bone densities in their upper spines, shoulders and ribs, compared to sedentary individuals.

Since ageing leads to bone and muscle loss, and since running offers an incomplete protective effect (and a potentially negative effect for the upper part of the body), many exercise experts recommend that older athletes include resistance training along with their regular aerobic workouts. After all, weight training is an almost foolproof way to build bone and muscle mass.

To see which plan – aerobic activity alone or aerobic activity plus strength training – is better for overall skeletal and muscular health, scientists at East Tennessee State University tested 43 healthy individuals who were all 55 years of age or older. Twenty-three of the subjects worked out three times per week for 30 minutes per session. Actual exercise consisted of walking vigorously on a treadmill, stair climbing or cycling, with heart rates at 65-85% of maximum during all of the workouts.

The other 20 exercisers performed aerobic activities (walking, stair climbing, cycling) for only 15 minutes per day and spent the rest of their workout time strength-training all of their major muscle groups using weight machines. Resistance was always set at 50-65% of one repetition maximum – the greatest amount of weight which could be lifted successfully one time.

After four months, bone density (averaged over the whole body) and lean muscle mass increased significantly in the group which combined aerobic activity with weight lifting but didn't improve for the athletes who only engaged in aerobic exercise. In addition, the density of the 'femoral neck' – a part of the femur which links the straight shaft of the femur with the actual hip-joint socket – advanced for strength-trained athletes but stayed constant in the aerobic group. This is particularly important for older individuals, since the femoral neck is a frequent site of fractures.

Neither group was able to lift the density of the lumbar vertebrae, and each group improved the ability to do sit-ups and press-ups by similar amounts. Although weight training is sometimes viewed as 'risky' for older athletes, none of the weight

trainers was injured during the four-month study.

Overall, a programme of aerobic activity plus strength training was better than aerobic exercise alone in terms of improving the integrity of the skeletal and muscular systems. As the researchers put it, 'We recommend that healthy people over the age of 55 years enrol in a combination of aerobic and weight lifting exercises'.
(Are Aerobic Exercises as Beneficial on the Musculoskeletal System as Weight-Lifting Exercises in Subjects 55 Years of Age and Older? Journal of Aging and Physical Activity, vol. 1 (1), October 1993)

Why the muscles shrink with age – and what to do about it

Most peoples' muscles reach their maximum size during their 25th year of life, grow smaller by about 10% between the ages of 25 and 50, and then shrink by 45% over the next three decades. Why does so much muscle tissue disappear and why does the degeneration accelerate after a half-century?

This research from Sweden has the answer. The primary reason for the sinew reduction is that the total number of cells in any particular muscle stays pretty constant until the age of 30 but then begins a steady decline. The fall-off is slow at first but increases dramatically after the age of 50. For example, if one of your muscles consisted of 100 cells (fibres) when you were 30, the muscle would probably still contain 90-95 fibres 20 years later, but the fibre count would plummet to only 50-55 when you became an octogenarian.

Individual muscle cells in your body can be either type 1 (slow-twitch) fibres, which contract slowly but have great endurance potential, or type 2 (fast-twitch) fibres, which contract quickly and powerfully but have little endurance. A decrease in the size of type 2 fibres plays a role in the muscle-shrinking process, with individual fast-twitchers shrivelling by about 25-30% between the ages of 20 and 80. However, this loss is somewhat compensated for by the steadfastness of type 1 (slow-twitch) muscle cells, which either remain unchanged in size or can expand by up to 20% in individuals who remain very physically active as they get older.

What causes the fairly dramatic loss in muscle-cell numbers?

Over time – and especially after the age of 50 – 'motor nerve cells' in the spinal cord begin to deteriorate at a steady rate. By means of their long arms, which spread outward from the spinal cord like the tentacles of an octopus, the motor nerve cells are normally in close contact with muscle cells. The motor nerves' key function is to 'tell' muscle fibres when to contract during physical activity, but the connection between motor nerves and their associated muscle cells is also necessary to keep the muscle fibres alive. As motor nerve cells die, the muscle cells to which they are attached also bite the dust.

Fortunately, there's a positive side to the story. People who participate in resistance training don't necessarily halt the fibre-death process, but they can stop and even reverse the tendencies of their type 2 cells to grow smaller. Although the number of muscle cells declines, type 2 (and sometimes even type 1) fibres may get larger as a result of strength training, leading to a potential advancement – instead of a loss – of total muscle tissue in the body. As a fringe benefit, resistance training in older individuals seems to increase the number of small blood vessels around muscles by up to 15%, potentially increasing endurance capacity.

Since the overall process of muscle atrophy picks up steam after the age of 50, strength training for people over 50 is especially critical. Fortunately, it's never too late. Research demonstrates that even individuals over the age of 80 can fortify their muscles by participating in regular strength-training workouts. *(Ageing and Human Muscle: Observations from Sweden, Canadian Journal of Applied Physiology, vol. 18(1), pp 2-18, 1993)*

Do the young respond more effectively to aerobic training? Don't you believe it

We hear it all the time: old people can't respond to training as young people do. After all, muscles somehow lose their ability to adapt. In fact, after the age of 60, they're struggling just to stay alive and keep their connections with the nervous system intact.

The general lack of fitness in the elderly population seems to support these assertions, but there's just one problem: it's all wrong. Athletes over the age of 65 can respond to training just as effectively as 30 year-olds. There's just no excuse for sitting by the fireside anymore!

Older people's responsiveness to training was determined in a study carried out at the University of Florida. There 10 sedentary, old (aged 67) males and females and 11 sedentary, young (aged 30) males and females completed a 16-week exercise programme.

All subjects worked out three times per week on a treadmill and/or stair-climbing machine. Over the 16-week period, workout duration increased from 20 to 40 minutes and training intensity advanced from about 60-80% of maximal heart rate.

At the end of 16 weeks, the young athletes had increased their maximal aerobic capacity (VO_2max) by 12%. The oldies? You guessed it: they had bolstered VO_2max by 14% (the difference wasn't statistically significant).

Since the old people improved just as much as the youngsters, the researchers wisely concluded that when the training stimulus is similar, older adults can increase their aerobic capacities to the same extent as young people.

(Aerobic Exercise Training Responses in Young and Elderly Men and Women, Medicine and Science in Sports and Exercise, vol. 25(5), p. S79, 1993)

And finally, here's a group of elderly hour-a-day exercisers who are aerobically 30 years younger

Maximal heart rate, VO_2max, muscular strength and respiratory capacity all tend to decline as a function of age, causing some exercise scientists to speculate that individuals over the age of 70 may not be able to improve their exercise capacities very much in response to training. However, some research carried out at Appalachian State University in North Carolina suggests that 70-plus individuals can still achieve dramatic improvements in aerobic

capacity, provided their training intensity is high enough.

At Appalachian State, 30 sedentary elderly women (average age 74) were divided into two groups. One group began a 12-week walking programme consisting of five 30 to 40 minute walks per week, each at an intensity of about 60% VO_2max. Average heart rate during the walks was around 120 beats per minute (about 78% of maximal heart rate). The second group engaged in 'mild musculoskeletal exercises' consisting of callisthenics and stretching routines designed to improve range of motion and flexibility. Heart rates averaged only 86 beats per minute during this second group's training sessions – just seven beats above resting heart rates.

After 12 weeks, VO_2max increased by almost 13% for the walkers, versus only 2% for the callisthenics participants. In addition, gains in VO_2max weren't limited by age: 85-year-old walkers achieved the same gains in VO_2max as their more youthful (67-year-old) counterparts

The last word? It's never too late. Even 85-year-olds can dramatically boost their aerobic capacities. Physical capacities do tend to go downhill as people age, but it's always possible to reverse the process through regular exercise. In fact, for people over the age of 65, as the foregoing report also shows, a 12-16-week programme of aerobic exercise can produce 10-20% improvements in aerobic capacity, as long as heart rates go above 75% of maximal during some of the workouts.

The North Carolina researchers uncovered one other pleasing result. When they tested a different group of 12 highly conditioned older women (average age 73) who exercised about one hour a day, they found that these fit women had aerobic capacities comparable to women 30 years younger!

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